June 20, 2018

The Honorable Bob Latta
Chairman
Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection
United States House of Representatives
2125 Rayburn House Office Building
Washington, D.C. 20515

AMERICAN QUARTER HORSE ASSOCIATION

Dear Chairman Latta and Ranking Member Schakowsky:

Thank you for the opportunity to submit this important information for the record to the Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection for consideration at your June 22, 2018 hearing on H.R. 2651, the Horseracing Integrity Act of 2017. I am writing to you on behalf of the American Quarter Horse Association.

In short, our Association is the official breed registry for the American Quarter Horse, and along with its 250,000 members, boasts the largest single registry of horses in the world. While the American Quarter Horse Association strongly supports uniformity in the horse racing industry, it is unable to support this proposed legislation.

Our opposition is multifaceted. Of particular concern regarding this proposal is the elimination of all race-day medications, including Lasix. Lasix has been endorsed by several equine groups and the American Association of Equine Practitioners to help mitigate the occurrence of exercise induced pulmonary hemorrhage in racehorses. We maintain that this legislation, if enacted, could actually *create* an animal welfare concern by leaving our equine athletes vulnerable to a potentially deadly health risk.

Funding sources for the program is also an area of concern regarding the legislation proposed. Most racing jurisdictions operate on thin margins, and we feel the economic impact of the burden this legislation places on our industry will be detrimental.

AQHA is committed to the welfare of the racehorse and continues to work with international, national and state racing organizations and commissions to evaluate protocols for uniform medication rules and deterrents of performance-enhancing drugs. In this endeavor, we work closely with the Association of Racing Commissioners International, and maintain they are making great strides in guiding each state's own racing regulation. Note that in their pursuit of uniformity amongst jurisdictions, 94% of pari-mutual handle is now regulated under the ARCI's Controlled Therapeutic Substance List, and 31 of 32 laboratories are RMTC accredited labs. Additionally, the ARCI has incorporated breed specific regulation within their model rules. As a result, the specific needs of each type of horse racing can be addressed individually. There is no mention of breed specific concerns in HR 2651. For years, key industry stockholders have participated in establishing these regulations, and we are very concerned about an added layer of bureaucracy undoing such efforts.

Thank you for the opportunity to address this important topic. I welcome your inquiry should you have additional questions or comments.

Sincerely,

Janet VanBebber

Janet VanBebber
Chief Racing Officer
American Quarter Horse Association

June 20, 2018

The Honorable Jan Schakowsky
Ranking Member
Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection
United States House of Representatives
2322A Rayburn House Office Building
Washington, D.C. 20515

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Janet VanBebber

Janet VanBebber
Chief Racing Officer
American Quarter Horse Association

AMERICAN
Racing & Entertainment · LLC

June 20, 2018

Chairman Bob Latta
Ranking Member Jan Schakowsky Committee on Energy & Commerce
Unites States House of Representatives
Washington, DC 20515

Dear Chairman Latta:

I appreciate the opportunity to share my views on the Horseracing Integrity act of 2017, H.R. 2651, and its importance to the Thoroughbred, Quarter Horse, and, my passion, Standardbred racing industries. I have been a fan of horse racing for more than 50 years. When I was young I used to sneak away to Roosevelt Raceway on Long Island, and my enthusiasm for horse racing has never diminished. Today, I am a Standardbred owner and breeder, and I operate three harness racetracks: Meadowlands Racetrack in New Jersey and Tioga Downs and Vernon Downs in New York. I have operated Tioga and Vernon since 2005 and Meadowlands since 2013.

One of my core beliefs in horse racing is that everyone should compete on a level playing field with integrity and credibility. I have gone to great lengths to get performance-enhancing drugs out of the sport, including the implementation of "house rules," which include strict medication and testing enforcement policies, at all three of my tracks. I also make everyone who races at my tracks sign an agreement that enables my investigator to perform out-of-competition testing, a fundamental part of any anti-doping regulation. My investigator can show up at any time at the tracks or on a farm, including Sunday if that's when they are least likely to expect it. The out-of-competition testing and surveillance are at the expense of my tracks.

Unfortunately, not all racetrack operators are as diligent, and modern horse racing suffers from a patchwork system of medication rules set by 38 different state regulatory commissions. This causes confusion about the rules for owners and trainers racing in different jurisdictions and enables unscrupulous trainers to change tactics and jurisdictions to avoid detection. Our sport needs uniformity and oversight so that we all compete on a level playing field.

H.R. 2651 will provide that uniformity and oversight through the Horseracing Anti-Doping and Medication Control Authority (HADA). HADA would be private, non-profit, non-governmental, and independent, and it would develop and administer a nationwide anti-doping program, including extensive out-of-competition testing.

As an owner and breeder, racetrack operator, and lifelong fan, the future of this sport is incredibly important to me. To ensure the welfare of our athletes and to maintain the integrity of our sport, I wholeheartedly support passage of the Horseracing Integrity Act.

Regards,

Jeffrey Gural

Tioga Downs Casino • 2384 West River Road • Nichols NY 13812
1-888-WIN-TIOGA • www.TiogaDowns.com
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1-877-88VERNON • www.VernonDowns.com

June 20, 2018

Energy & Commerce Subcommittee on Digital Commerce and Consumer Protection

Re: HR 2651

Dear Members of the Committee,

My name is Chris McCarron and I am writing in support of the Horseracing Integrity Act, H.R. 2651. I was a professional Thoroughbred jockey for 28 years, from 1974 through 2002. During my career I won 7,141 races, and when I retired I was the leading money earner in the history of the sport. I won six Triple Crown races and nine Breeders' Cup race, five in the Classic. I was inducted into the National Racing Hall of Fame in 1989 during my first year of eligibility.

I support H.R. 2651 because horse racing, as a sport, has been in a serious decline for years and something has to be done to curb this trend. I believe one of the reasons for this decline is an extreme lack of public confidence in our product. Over the past few decades, medication violations (horses testing positive for banned and non-banned substances) have escalated to the point of being unacceptable by our patrons and participants alike.

When I began my career in Maryland in 1974, the average number of *annual* starts was approximately 14. Today the average number of *career* starts is 11. This stat alone has caused many of our horse owners to leave the sport and many still to be disinterested in becoming an owner. Racing simply cannot sustain this serious decline. Why would anyone, knowing these stats, want to become involved in racehorse ownership?

There are far too many horses becoming injured to the point where their careers come to an early end. To borrow and slightly change an old adage, "medications don't kill horses, trainer discretion kills horses," i.e., racing horses that would be better served by getting some much-needed rest. Instead of giving the animal the rest it needs, a trainer relies on his/her veterinarian to administer a medication to mask pain by reducing inflammation caused by an injury. Horses' careers would last much longer if this practice was less prevalent.

Additionally, I have a personal interest in getting this bill passed. Far too many of my brethren, the active jockeys and exercise riders plying their trade on a daily basis, are being injured due to horses breaking down while racing or training. Several years ago, the *Journal of American Medical Association* conducted a study that revealed that jockeys experience 35 accidents per week. Needless to say, this is a very dangerous occupation. More to the point, on average, two jockeys die each year due to training or racing accidents and two more are left paralyzed. The vast majority of these tragedies occur due

to a horse's breaking down. Very often these horses are racing with pre-existing conditions that have been masked by medication.

The status quo with 38 different states governing one industry with 38 different sets of rules and penalties and numerous different laboratories doing the testing using different standards simply cannot go on if Thoroughbred racing is to continue to be a viable industry.

I'm sure you are aware of the hundreds of thousands of jobs and the billions of dollars of revenue our industry provides. Something has to be done to stem the tide and stabilize an industry that is so vital to communities all over our country.

I hope you give H.R. 2651 the thumbs-up and help the Thoroughbred racing industry right this ship that is sinking.

Yours truly,

Chris McCarron
HOF 1989

Equine Health and Welfare Alliance, Inc. 501(c)3

100 S. Main St.

Versailles, KY 40383

(859) 227-0879

President: Frank D. Marcum, DVM

The Honorable Bob Latta, Chairman
Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection
United States House of Representatives
2125 Rayburn House Office Building
Washington, D.C. 20515

The Honorable Jan Schakowsky, Ranking Member
Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection
United States House of Representatives
2322A Rayburn House Office Building
Washington, D.C. 20515

Re: Equine Health and Welfare Alliance in **Opposition** to H.R. 2651

Our organization has been dedicated to the proper care and maintenance of the horse and all equines since its inception in 2010. Our mission is to advocate on behalf of all horses, and in that role, we reach out to your committee to request that H.R. 2651 be tabled indefinitely. The proposed bill seeks to impose medication uniformity on an industry that already enjoys considerable uniformity. The best example of this uniformity in horse racing medication regulation is the universal acceptance of furosemide, administered under strict regulatory guidelines, and transparency to public. H.R. 2651 seeks to set aside the great progress that has been made in alignment of racing jurisdictions and completely remove the most uniform of all medication regulations that exists in this country…the administration of furosemide as a pre-race medication.

Horses rely on their caretakers to do the best thing for them. They do not have a voice in their own care. This places a great responsibility on the horsemen and women that care for them. This responsibility includes protection from injury and illness.

The scientific literature is abundant and clear. Furosemide provides protection from injury in the lungs of racing horses. As administered currently in all racing jurisdictions across the country, it cannot interfere with drug testing, or act as a masking agent. Other criticisms have been that it is performance enhancing because it causes weight loss. This is an assertion that has never been confirmed in any research. The use of furosemide to help protect horses from lung injury is simply the ethical and humane thing to do.

The current framework for establishing regulations in horse racing has been in place for decades and has been getting closer to achieving uniformity every year. The current framework permits all industry stakeholders to attend LOCAL meetings and provide input into the formation of regulations. It permits the careful and systematic review of new regulations at each level in every jurisdiction, ultimately providing opportunity for crafting of the most beneficial regulations. Where the regulations are the best thing for the horse and the industry, they are adopted rapidly and completely across all jurisdictions. The administration of furosemide on raceday is the best example of how good and appropriate regulations become installed nationwide. We continue to advocate for the best version of medication regulations across the country, and we feel that current system provides a framework to allow us and other stakeholders to influence that process, a framework unlikely to be available with a centralized authority.

Respectfully Submitted and Sincerely;

Frank D. Marcum, DVM

AQHA®

AMERICAN QUARTER HORSE RACING

AQHA OPPOSES THE NEWLY INTRODUCED VERSION OF THE HORSERACING INTEGRITY ACT OF 2017.

June 8, 2017

While AQHA strongly supports uniformity in the horse racing industry, it is unable to support the latest version of the newly introduced legislation.

On May 25, Congressman Andy Barr (R-KY) introduced the Horseracing Integrity Act of 2017 to the House of Representatives. In summary, the bill requires "a uniform anti-doping and medication control program to be developed and enforced by an independent Horseracing Anti-Doping and Medication Control Authority."

While the American Quarter Horse Association strongly supports uniformity in the horse racing industry, it is unable to support the latest version of the newly introduced legislation.

"Of particular concern regarding this proposal is the elimination of all race-day medications, including Lasix, the use of which has been endorsed by several equine groups and the American Association of Equine Practitioners to help mitigate the occurrence of exercise induced pulmonary hemorrhage in racehorses," said Craig Huffhines, AQHA executive vice president. "American Quarter Horse representation on the Authority and funding sources for the program are also among other areas of concern that we have regarding the legislation as currently proposed."

AQHA is committed to the welfare of the racehorse and continues to work with international, national and state racing organizations and commissions to evaluate protocols to allow for uniform medication rules and deterrents of performance-enhancing drugs. In addition, the use of Lasix in AQHA shows is currently under review by the AQHA Animal Welfare Commission by request of the Executive Committee.

In recent months, AQHA worked with the Association of Racing Commissioners International to separate American Quarter Horse flat racing in its medication violation model rules to help eliminate the use of illegal performance-enhancing medications. The Association has also supported recent industry movements that include out-of-competition testing and hair testing.

For more information on American Quarter Horse racing, visit www.aqha.com/racing.

AAEP STATEMENT ON THE HORSERACING INTEGRITY ACT, H.R. 2651

Statement by American Association of Equine Practitioners 2017 President R. Reynolds Cowles, DVM:

"While the American Association of Equine Practitioners supports the uniformity of medication rules in U.S. horse racing, which is the one of the chief goals of the Horseracing Integrity Act, our association opposes the newly introduced version of the legislation.
"The AAEP's current policy on race-day medication administration endorses the use of furosemide to help mitigate the occurrence of exercise-induced pulmonary hemorrhage (EIPH) in the racehorse. This policy is based on the overwhelming body of international scientific and clinical evidence.

"H.R. 2651 seeks to end the administration of furosemide on race day, which conflicts with the AAEP's long-held position. While we are optimistic that current research projects will yield an alternative treatment for EIPH which does not require race-day administration, as doctors of veterinary medicine we cannot abandon our current policy until science provides an efficacious option for protecting the health and welfare of the horse.

"The ability of USADA to regulate a sport which has far more participants than any sport they currently oversee remains a concern for the AAEP, but we are pleased with the change to the legislation's proposed structure which allows for the inclusion of a veterinarian as part of the governing body. We also are pleased with the expansion of the bill's language to clearly delineate the role of therapeutic medication and a formal anti-doping program.

"We appreciate the opportunity provided to us previously by Rep. Barr to offer input on the legislation in the areas of governance and veterinary involvement, although our suggestions were not incorporated into this version of the bill. The AAEP wishes to continue to serve as a resource to Rep. Barr and Rep. Tonko as issues affecting the health and the welfare of the racehorse are considered."

OP/ED: YES, THE AAEP IS PROTECTING THE HORSE

by Jeff A. Blea, DVM & AAEP Racing Committee Chair

The American Association of Equine Practitioners (AAEP)'s recent decision to oppose the Horseracing Integrity Act of 2017 has been met with both support and criticism, depending on one's perspective within the industry. As chair of the AAEP Racing Committee, I'd like to address why we believe our position best represents the health and welfare of the racehorse.

First, I respect all the industry stakeholders who have invested an incredible amount of time and resources to ensure horse racing's sustainability. We have the same goals, I believe, even though we may differ on specific aspects of the proposed federal legislation.

The AAEP's decision to oppose the Horseracing Integrity Act was principally based on our long-standing policy in support of the race-day administration of furosemide to help mitigate the occurrence of exercise-induced pulmonary hemorrhage (EIPH). This policy is based on a vast body of scientific and clinical evidence and on what we, as equine veterinarians, believe is in the best interest of the health and welfare of the horse.

The scientific community recognizes that EIPH is a disease that affects equine athletes, in addition to human athletes (Diwakar, Amit, and Gregory A. Schmidt. "Exercise-Induced Pulmonary Hemorrhage in a Nonathlete: Case Report and Review of Physiology." Lung 192.2 (2014): 329-331). Currently, without debate, the only scientifically proven medication to ameliorate the effects of EIPH in the racehorse is furosemide (Lasix). It is in fact used in training in most countries around the world with few exceptions (personal communication).

In 2015, the AAEP developed a long- range 10-Point Plan for horse racing which included the goal of pursuing research to investigate alternative strategies for managing EIPH that did not require race-day Lasix administration. Promising research projects are currently underway, but it is too soon to know if any will yield an alternative. While there are elements of the Horseracing Integrity Act we certainly support, the AAEP was not able to abandon our EIPH efforts and our long-held position for political expediency.

The AAEP has members who are staunch advocates on both sides of the Lasix debate. In fact, AAEP strongly advocated for the third-party administration of race-day Lasix in order to negate any premise that our support of race-day Lasix was in any way based upon racetrack veterinarians' financial interests. Suggestions from some industry stakeholders that the AAEP's support of race-day Lasix is a dollars and cents issue for veterinarians is incorrect, inaccurate, and is directly refuted by our endorsement of third-party administration.

The race-day administration of Lasix is without doubt one of the most polarizing issues in horse racing. We respect the fact that other jurisdictions around the globe compete without the use of race-day Lasix. The racing business model is complex and arguably a justification of why other countries can exist successfully without race-day Lasix. In order for the U.S. racing industry to compete in similar fashion to other global jurisdictions, a cultural shift in U.S. racing must first occur.

The AAEP understands that the development of alternative effective treatments to mitigate race-day EIPH, without affecting performance, will require resources, commitment and patience, and most importantly, time. It's a lofty goal that may be in vain. However, we are committed to doing what is best for the horse, while ensuring the integrity and sustainability of the racing industry for the future, without an emphasis on financial gains.

Association of Racing Commissioners International

August 10, 2017

Rep. Ralph Abraham
U.S. House of Representatives
417 Cannon House Office Building
Washington, DC 20515

Dear Rep. Abraham:

We write concerning H.R. 2651, the so-called Horse Racing Integrity Act of 2017.

The Association of Racing Commissioners International (ARCI) is the umbrella organization of the independent entities designated by statute in the United States, Canada, Mexico, and parts of the Caribbean to regulate and police professional horse racing. The ARCI sets regulatory standards, encourages uniformity, and develops best practices to ensure the integrity and safety of the sport.

We challenge the necessity for H.R. 2651 and have deep reservations about the provisions contained therein, especially those that redundantly create integrity programs modeled after those already in place.

Performance enhancing drugs are not allowed or tolerated in professional horse racing. There is total uniformity on this issue. Horse racing's standards are stronger than those deployed in human sport because there is no provision to permit athletes to compete under the influence of a prohibited substance if the anti-doping agency grants an exemption. This is routinely done in human sport unbeknownst to the other competitors and fans.

Horse racing regulators have long been involved with working to create and implement uniform policies to safeguard against doping and protect horses. We strongly support the existing process where policies are proposed by the Racing Medication and Testing Consortium and vetted by the ARCI before being included in the Model Rules of Racing.

While we do acknowledge the few instances where a state may have not have uniformly adopted an ARCI standard, we note that in these rare instances the differences in actual policy are minor and limited to how long prior to a race a legal and appropriate therapeutic treatment should be stopped.

Horse racing regulators in the United States collectively operate an anti-doping and testing program that is over thirty (30) times the size of the program that Congress funds at the U.S. Anti-Doping Agency and larger than the entire testing program performed worldwide for the World Anti-Doping Agency.

H.R. 2651 is a highly controversial proposal that is opposed by organizations representing the largest constituent segments in the sport. These include the: American Association of Equine

Practitioners, National Association of Racetrack Veterinarians, American Quarter Horse Association, Thoroughbred Horseman's Association, National Horseman's Protective and Benevolent Association, Thoroughbred Owners of California, New York Thoroughbred Horseman's Association, Harness Horsemen International, Standardbred Breeders and Owners Association, the Jockeys Guild and countless other local and regional groups.

We strongly urge you not to support this legislation.

We also believe it is not in the welfare interest of the race horse to place medication and anti-doping policy in the hands of an entity with no veterinary expertise, as H.R. 2651 proposes. We additionally do not believe it good public policy to permit an organization to determine its own budget with authority to impose new and unlimited assessments on the States or taxes on racing industry participants as is being proposed.

Finally, this proposal also raises ethical and moral issues in that it outlaws an otherwise legal medication that has been scientifically proven to help horses withstand vigorous exercise and competition. H.R. 2651 proposes to repeal a thirty-year equine welfare policy to mitigate or safeguard against a condition recently elevated in its degree of seriousness by the American College of Veterinary Internal Medicine. This policy was instrumental in stopping the abusive practice of denying horses water beginning the night before the day they race.

When one considers the World Anti-Doping Agency's therapeutic medication policy it is clear that human athletes are treated with far more consideration than the supporters of this bill would have race horses treated. We ask: "Is not the health and wellbeing of our equine athletes as important?" We hope you agree that it is and resist those who urge you to support this proposal.

Finally, the ARCI has long served as a resource to legislators interested in horse racing regulatory matters. We continue to work with all aspects of the racing industry to develop and implement policies to protect horses, those who ride or drive, as well as the integrity of each contest.

We appreciate the interest of any Member of Congress in the regulation of this sport, and welcome the opportunity to brief you or your staff on these matters. We are always open to exploring ways the federal government may be of meaningful assistance to the states in this area. If we can be of any assistance on these matters, please do not hesitate to contact us.

Sincerely,

Jeff Colliton

Jeff Colliton, Chair

Edward J. Martin, President

United States Trotting Association Opposes Horse Racing Integrity Act

Sept. 5, 2017

Following is a statement from the U.S. Trotting Association expressing its opposition to the Horse Racing Integrity Act of 2017:

While the United States Trotting Association (USTA) strongly supports breed-specific, uniform medication rules for horse racing, the USTA, which has had no input into the preparation of the bill, opposes the Horse Racing Integrity Act of 2017 (H.R.2651) for a number of reasons.

Two of the primary objections to the proposed legislation are the elimination of race-day medications, specifically furosemide (Lasix), and the lack of separate, uniform regulations governing the use of therapeutic medications for the different breeds.

In March 2012, the USTA announced its official position on furosemide stating, "The U.S. Trotting Association believes that the most humane way to address this problem (Exercised-Induced Pulmonary Hemorrhage) is through the continued approval of the race-day administration of furosemide under controlled conditions and by a licensed veterinarian."

"After a year of considering all the issues concerning the race-day administration of furosemide, commonly known as Salix or Lasix, the U.S. Trotting Association believes the determining factor should be the welfare of the horse," said then USTA President Phil Langley in making the announcement at that time.

The American Association of Equine Practitioners also endorses the use of race-day Lasix "based on the overwhelming body of international scientific and clinical evidence."

The USTA has long been an advocate for separate rules for the different breeds in the use of therapeutic medications.

"As the Association of Racing Commissioners International has recently agreed and the USTA has advocated all along, the differences in the racing breeds and their business models, particularly the frequency that the horses race, requires there to be separate rules for each breed in the use of therapeutic medications," said USTA President Russell Williams. "A 'one-size-fits-all' approach, which is what H.R.2651 appears to advocate, isn't right, isn't fair, doesn't promote equine health, and won't work."

Further, the USTA has concerns about the makeup of the proposed federal board of the Horseracing Anti-Doping and Medication Control Authority (HAMCA) created by the legislation.

"The proposed board members will have no experience with or understanding of the horse racing industry or the welfare of the horses," said Williams. "It seeks to replace the current state regulatory system where uniformity largely exists and is made up of regulators with extensive experience and knowledge of horse racing.

"Also, it is a significant concern to the USTA that this legislation would designate the Federal Trade Commission as the ultimate regulatory authority, bypassing agencies like the Department of Agriculture and the Food and Drug Administration that have experience with animal welfare issues."

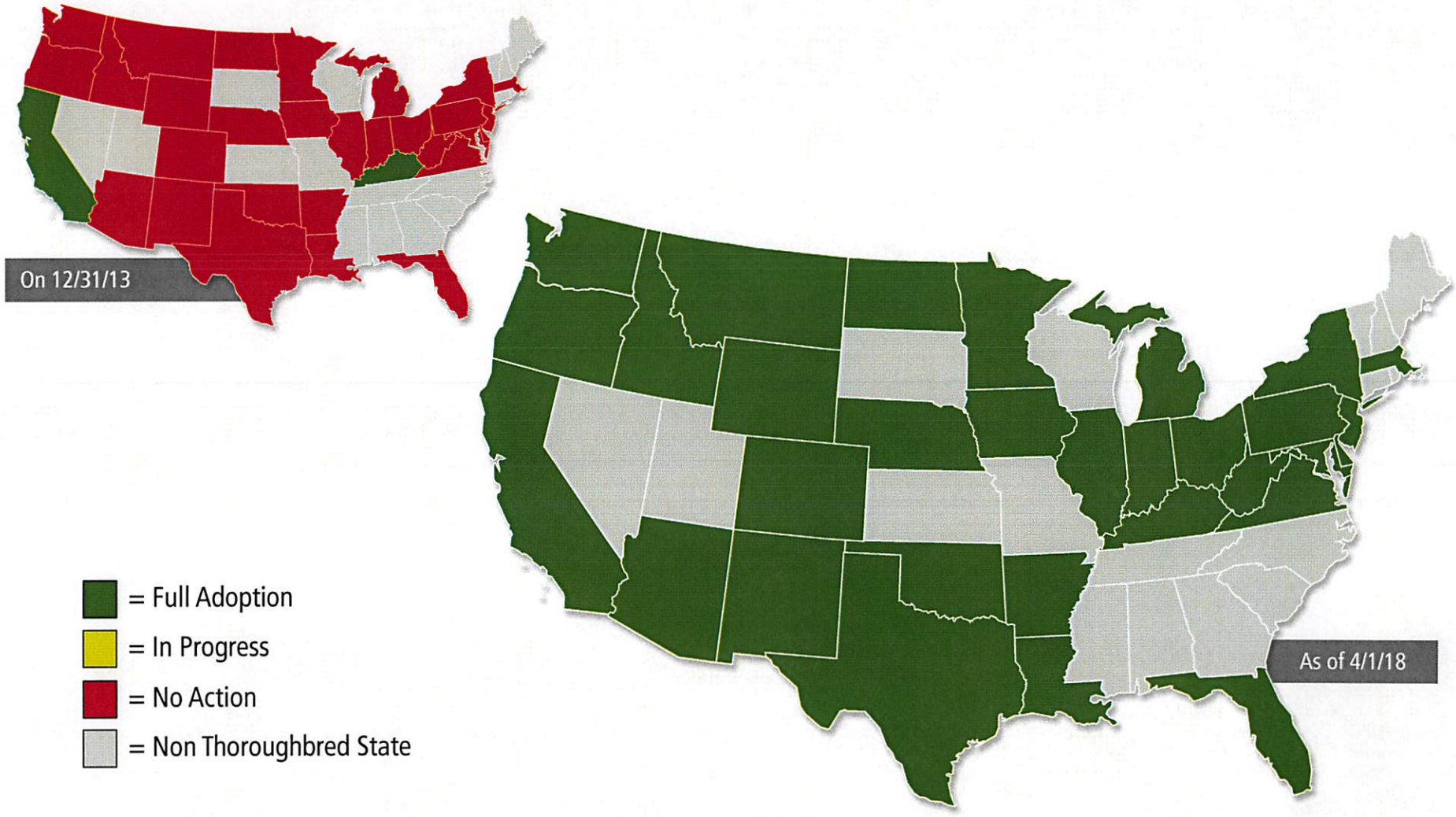
In addition, the proposed legislation would create a regulatory commission that could mandate significant additional expenses to the horse racing industry.

"There is no stipulation for federal funding in the legislation as there is for the United States Anti-Doping Agency in its testing of human athletes, which would give HAMCA a blank check to impose new costs to racetracks and horsemen with minimal oversight or accountability," added Williams.

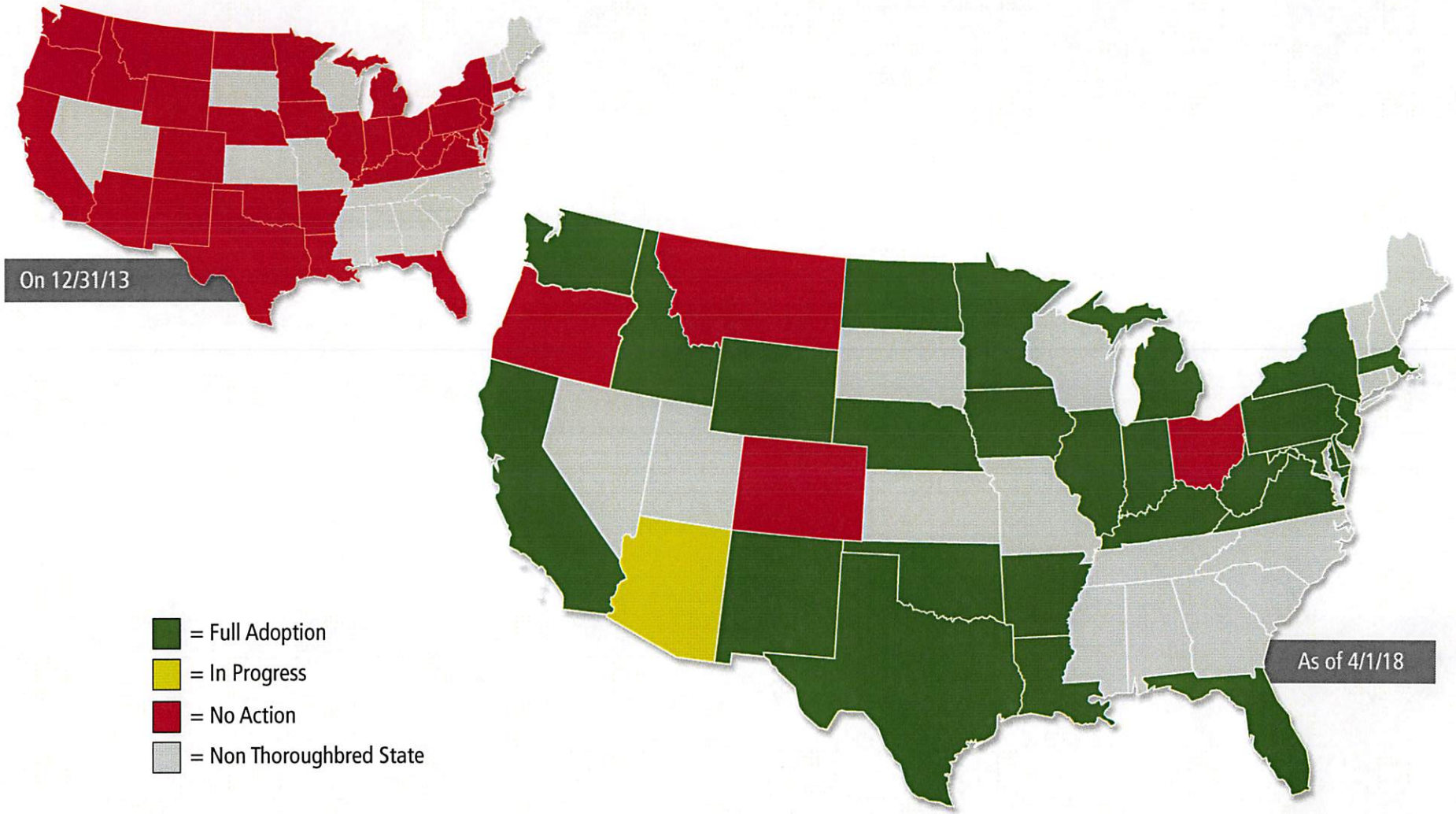
The USTA joins the Horsemen's Benevolent and Protective Association and Thoroughbred Horsemen's Association (the two major, national organizations representing Thoroughbred owners, breeders and trainers); Harness Horsemen International (the international organization that represents Standardbred owners, breeders and trainers in the U.S. and Canada); Association of Racing Commissioners International (the national organization representing independent state racing commissions); the American Association of Equine Practitioners and North American Association of Racetrack Veterinarians (the two principal organizations representing the equine veterinary community); and the American Quarter Horse Association as well as numerous other racing and breeding organizations in opposing the proposed Horse Racing Integrity Act of 2017 (H.R. 2651).

For further information about the U.S. Trotting Association, visit www.ustrotting.com or call 877/ 800-8782.

Thoroughbred States Where NUMP Has Been Adopted Or Is In Progress

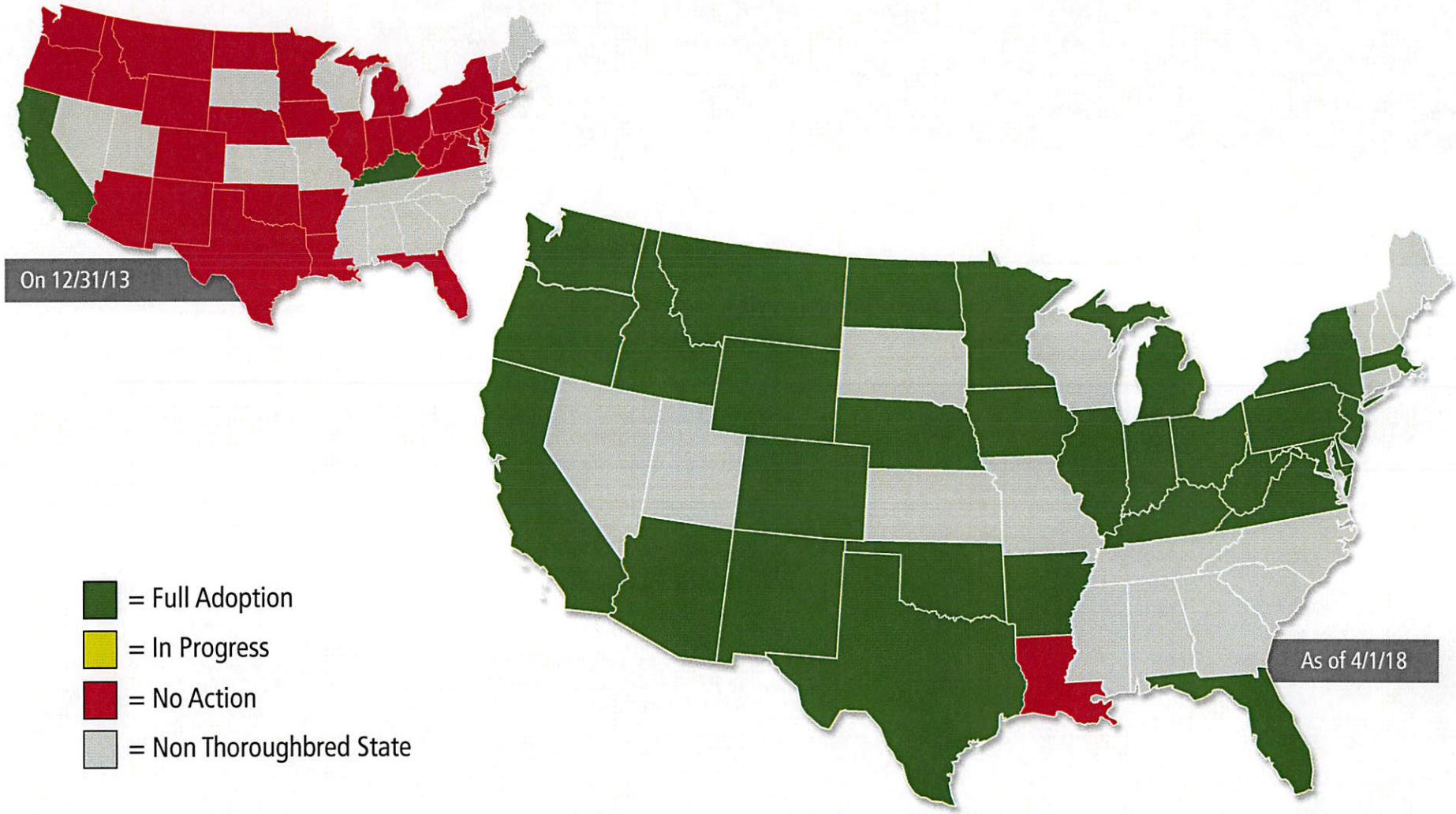


Controlled Therapeutic Medication List Adoption Schedule By Thoroughbred States



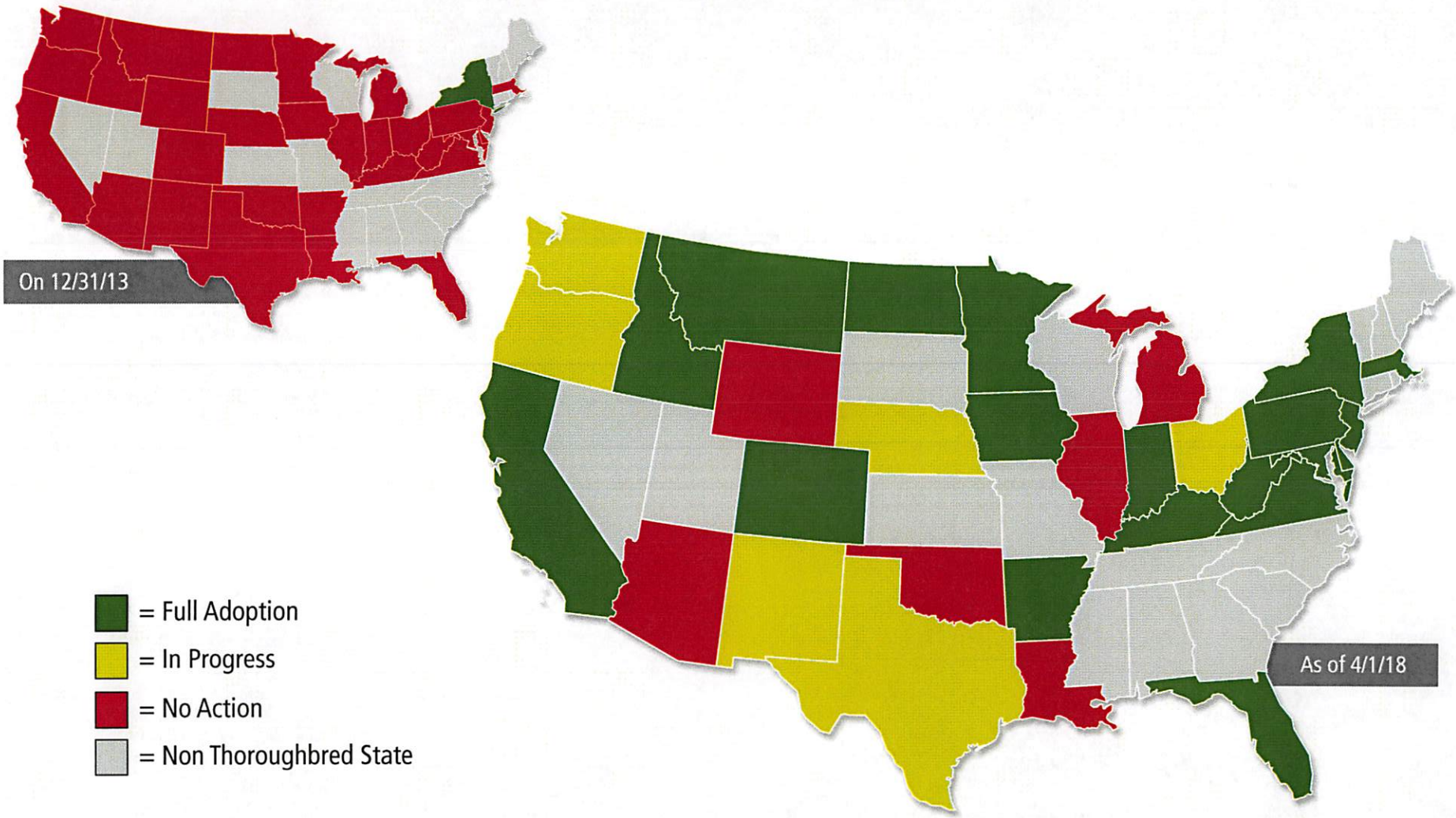
The Controlled Therapeutic Medication Schedule has been implemented in thoroughbred states representing 97% of the U.S. pari-mutuel handle.

RMTC Code Laboratory Accreditation By Thoroughbred States



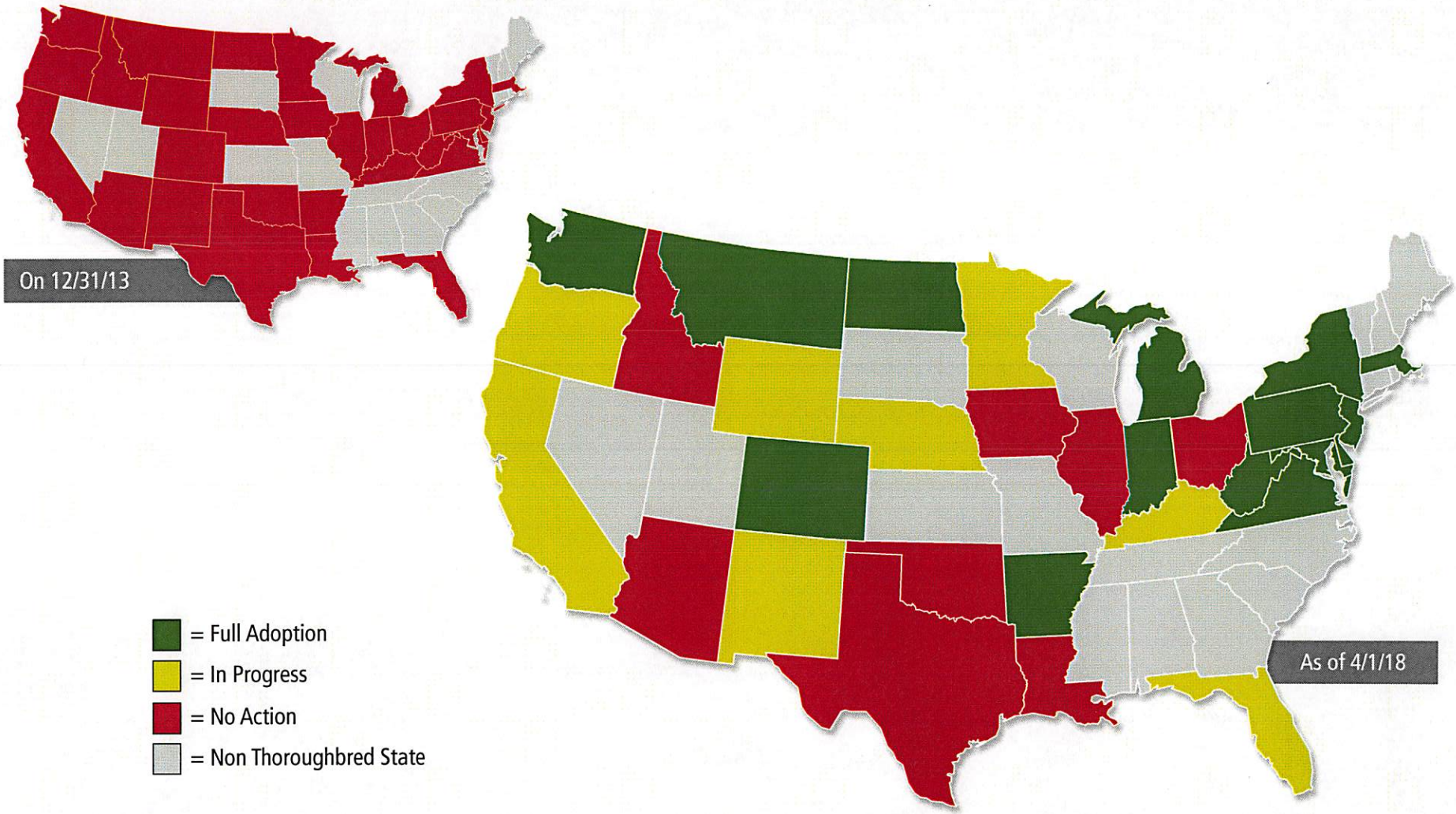
States representing more than 96% of the U.S. pari-mutuel handle on thoroughbred races are conducting testing at a lab accredited by the Racing Medication and Testing Consortium.

Third Party Lasix Administration By Thoroughbred States



Third-party Lasix administration procedures have been implemented in thoroughbred states representing 87% of the U.S. pari-mutuel handle.

Multiple Medication Violation Penalty System By Thoroughbred States



Thoroughbred states representing 40% of the U.S. pari-mutuel handle and 39% of thoroughbred races have implemented the Multiple Medication Violation Penalty System.

Congressional Research Service
Informing the legislative debate since 1914

MEMORANDUM October 27, 2015

To: Representative Joe Pitts
[redacted]

Subject: **Analysis of Potential Constitutional Challenges to Delegations Made to a Private Entity Under H.R. 3084**

This memorandum responds to your request for a legal analysis of H.R. 3084, the "Thoroughbred Horseracing Integrity Act of 2015." Specifically, you asked whether the bill's delegation of power to a private regulatory body, known as the Thoroughbred Horseracing Anti-Doping Authority (THADA or the Authority), is consistent with the U.S. Constitution.[1] At your request, a duplicate copy of this memorandum with identical language has been provided to Senator Udall.

The permissibility of federal delegations of authority to private entities is relatively unsettled. However, there would appear to be a consensus that delegations of regulatory and enforcement powers to private entities generally raise constitutional concerns, particularly when federal involvement and supervision in the exercise of those powers are absent. Based upon our review of existing law, it would appear that a strong argument can be made that H.R. 3084 delegates to THADA, a private entity, the kinds of regulatory and enforcement powers that would implicate these concerns.

The Thoroughbred Horseracing Anti-Doping Authority

H.R. 3084 would create the Thoroughbred Horseracing Anti-Doping Authority, "an independent organization with responsibility for developing and administering an anti-doping program for covered

[1] This memorandum will not address other potential constitutional concerns, including whether H.R. 3084 infringes on executive power by failing to provide the President with the authority to either appoint or remove THADA board members. The Constitution requires that any official exercising "significant authority pursuant to the laws of the United States" be appointed in conformance with the Appointments Clause, U.S. Const. art. II, § 2, i.e. by the President with the advice and consent of the Senate, or, in the case of inferior officers, by the President, the head of a department, or the courts. *See* Buckley v. Valeo, 424 U.S. 1 (1976). With respect to removal, the Supreme Court held in *Free Enterprise Fund v. Public Company Accounting Oversight Board*, 561 U.S. 477 (2010), that the President must retain "general administrative control of those executing the laws." *Id.* at 492-93. That control, the Court reasoned, is exercised primarily via the power of removal. *See also* CRS Report R43708, *The Take Care Clause and Executive Discretion in the Enforcement of Law*, by Todd Garvey. The application of these two principles to scenarios in which federal law empowers a private individual, as opposed to a government official, has not been considered by the Supreme Court. Finally, this memorandum also will not address any Tenth Amendment concerns that may arise as a result of provisions in H.R. 3084 that would appear to require state racing commissions to collect fees set by a private body pursuant to an authorization received from Congress.

horses, covered persons, and covered horseraces."[2] THADA would be structured as a non-profit corporation[3] and "shall not be considered nor construed to be an agent of, or an actor on behalf of, the United States Government...."[4] Under the bill, THADA would be given "exclusive jurisdiction" over anti-doping matters for all covered horses, covered persons, and covered horseraces beginning on January 1, 2017.[5] The legislation defines certain elements that must be included in the anti-doping program to be established and implemented by THADA, including lists of permitted and prohibited substances and sanctions for violations.[6] THADA would be empowered to impose sanctions "up to and including a lifetime ban from horseracing" for covered persons and/or covered horses.[7]

Covered persons and covered horses would be required to submit themselves to the jurisdiction of THADA and agree to comply with THADA's anti-doping program in order to participate in covered horseraces.[8] Additionally, THADA's "jurisdiction and authority" would also be imposed as a condition upon the ability to "accept, receive or transmit wagers on covered horseraces and to participate in such races."[9]

Under the bill, THADA would be funded "entirely by the Thoroughbred horseracing industry."[10] Funds necessary for THADA's initial establishment would be obtained through loans and private donations. Subsequent ongoing expenses would be funded by a "per racing start" fee set each year by THADA that, based on the prior year's budget, is estimated to be adequate to cover THADA's expenses for the coming year.[11] State racing commissions would then be required to "remit" to THADA, on a monthly basis, "an amount equal to the applicable fee per racing start multiplied by the number of racing starts in the State in the previous month."[12] Each state racing commission would have discretion, subject to applicable state laws, to determine "the method by which the requisite amount shall be allocated, assessed, and collected."[13] However, because the bill mandates that the "establishment and administration" of the anti-doping program "shall be paid entirely by the Thoroughbred horseracing industry," it would appear that the state racing commissions may only assess, allocate, and collect funds from members of that undefined group.[14] The bill also expressly states that the federal government is not required to "provide funding for or to guarantee the debts of the Authority."[15]

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[2] H.R. 3084, § 5(a). "Covered persons" is defined as "all trainers, owners, veterinarians, and the agents and employees of such persons and other horse support personnel who are engaged in the care, training, or racing of covered horses." *Id.* at § 3(4). "Covered horse" is defined as "any Thoroughbred horse, beginning on the date of the Thoroughbred horse's first timed and reported workout at a race track that participates in races that are the subject of interstate off-track wagers or a licensed Thoroughbred training facility until the Authority receives written notice that the horse has been retired." *Id.* at § 3(3). "Covered horserace" is defined as "any horserace that involves only Thoroughbreds and that is the subject of interstate off-track wagers." *Id.* at § 3(2).

[3] *Id.* at § 5(b).

[4] *Id.* at § 10.

[5] *Id.* at § 4(a).

[6] *Id.* at §§ 6(a), 7(b)

[7] H.R. 3084, § 7(f).

[8] *Id.* at §§ 4(c), 6(b).

[9] *Id.* at § 4(b).

[10] *Id.* at § 12.

[11] *Id.* at § 12(2).

[12] *Id.* at § 12(3).

[13] H.R. 3084, § 12(4).

[14] H.R. 3084 does not expressly define "Thoroughbred horseracing industry." However, the bill does define "Thoroughbred constituencies" to include "owners and breeders, trainers, horse racing associations, veterinarians, State racing commissions and jockeys." *Id.* at § 3(17). Section 5(b) of the bill, which relates to the appointment of THADA board members, may provide further insight. That provision states that "[t]he United States Anti-Doping Agency shall solicit lists of two candidates each from (continued...)

As a threshold matter, it is important to establish that THADA would be a wholly private entity. H.R. 3084 characterizes THADA as an "independent" and "non-profit corporation" that "shall not be considered...to be an agency." Nevertheless, it is "not for Congress to make the final determination" of THADA's status as a government entity.[16] However, when expressed congressional intent is combined with a nearly complete absence of federal supervision,[17] control, funding, and involvement in day-to-day operations, it becomes apparent that THADA would not be considered a government entity, but rather an "autonomous private enterprise."[18]

Delegation of Legislative Authority to a Private Entity

The Constitution's vesting of "all legislative powers" in "a Congress of the United States" has traditionally been interpreted as limiting Congress's authority to delegate "legislative power" to the other branches of government.[19] This "nondelegation doctrine" is based in the separation of powers and exists primarily to prevent Congress from abdicating the core legislative function assigned to it by Article I of the Constitution.[20] By restricting Congress's ability to give away its power, in many respects the nondelegation doctrine "protects Congress from itself."[21]

Although the Supreme Court has declared categorically that "the legislative power of Congress cannot be delegated,"[22] the standard adopted for determining whether Congress has in fact delegated "legislative authority" is a lenient one—as evidenced by the fact that the Court has used the test to invalidate federal laws only twice.[23] In order for a delegation to survive scrutiny, Congress need only establish an "intelligible principle" to govern the exercise of the delegated power.[24] Although allowing Congress to make broad delegations, the "intelligible principle" test ensures that Congress, not the delegee, renders the underlying policy decision by delineating reasonable legal standards for the exercise of the provided authority.[25] When a delegation is accompanied by an "intelligible principle," Congress is clearly

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a cross-section of thoroughbred industry representatives, the members of which include owners and breeders, trainers, veterinarians, racing associations, State racing commissions and jockeys."

[15] *Id.* at § 12.

[16] *See* Lebron v. Nat'l R.R. Passenger Corp., 513 U.S. 374, 392 (1995) ("But it is not for Congress to make the final determination of Amtrak's status as a government entity for purposes of determining the constitutional rights of citizens affected by its actions.").

[17] The federal government's role under the bill appears to be limited to a periodic report that the Comptroller General must provide to Congress that "analyzes the Authority's operations" and reviews "the Authority's effectiveness as an anti-doping organization and the efficiency of such anti-doping program." H.R. 3084 § 5(f).

[18] *See* Dep't of Transp. v. Ass'n of Am. R.R., 575 U.S. __, 135 S. Ct. 1225, 1232 (2015) [hereinafter *American Railroads*].

[19] U.S. CONST. art. I, § 1.

[20] Mistretta v. United States, 488 U.S. 361, 371 (1989) ("The nondelegation doctrine is rooted in the principle of separation of powers that underlies our tripartite system of Government. The Constitution provides that '[a]ll legislative Powers herein granted shall be vested in a Congress of the United States,' and we long have insisted that 'the integrity and maintenance of the system of government ordained by the Constitution' mandate that Congress generally cannot delegate its legislative power to another Branch.") (internal citations omitted).

[21] *See* Neil Kinkopf, *Of Devolution, Privatization, and Globalization: Separation of Powers Limits on Congressional Authority to Assign Federal Power to Non-Federal Actors*, 50 RUTGERS L. REV. 331, 358 (1998).

[22] *See, e.g.*, United States v. Shreveport Grain & Elevator Co., 287 U.S. 77, 85 (1932).

[23] *See* Panama Refining v. Ryan, 293 U.S. 388 (1935); A.L.A. Schechter Poultry Corp. v. United States, 295 U.S. 495 (1935).

[24] J.W. Hampton, Jr. & Co. v. United States, 276 U.S. 394, 409 (1928) ("If Congress shall lay down by legislative act an intelligible principle to which the person or body authorized [] is directed to conform, such legislative action is not a forbidden delegation of legislative power.").

[25] *See, e.g.*, *Panama Refining Co.*, 293 U.S. at 421 ("The Constitution has never been regarded as denying to the Congress the (continued...)

transferring some degree of authority, but by confining the delegee's discretion in the exercise of that authority, the delegation is not of a "legislative" nature such that it would offend the separation of powers.

Some commentators have asserted that a congressional delegation should be treated the same whether it empowers a *private* or *public* entity.[26] Regardless of what entity ultimately exercises the delegated authority, under this line of reasoning, the standard for evaluating its permissibility is the same: a court need only determine whether Congress has provided an "intelligible principle" to guide the entity's exercise of the delegated power. If a reviewing court were to adopt this position, any delegation that does not provide a private entity with essentially unbridled discretion in carrying out its powers would likely be deemed valid for the purposes of the nondelegation doctrine.[27] Under this theory, H.R. 3084 would likely pass constitutional muster. Sections 6 and 7, which lay out THADA's powers and establish an "outline" of the anti-doping program, would appear to adequately confine THADA's discretion by providing an "intelligible principle" to guide THADA in its implementation of the required anti-doping program.

There is substantial evidence, however, to suggest that a reviewing court may employ a different analytical framework in evaluating the type of private delegation envisioned by H.R. 3084. Rather than applying the "intelligible principle" test, some judicial decisions—including Supreme Court opinions—appear to have instead adopted a different approach to evaluating congressional delegations to private entities.[28] This line of reasoning is sometimes referred to as the "private delegation doctrine" and is typically triggered when the federal government allows a private party to "make the law and force it upon a minority."[29] Although these private delegation cases are relatively rare, the reasoning applied generally finds its genesis in the 1936 Supreme Court case of *Carter v. Carter Coal Co.*[30]

In *Carter Coal*, the Supreme Court invalidated the Bituminous Coal Conservation Act of 1935, which provided a majority of coal producers and miners in a given region the authority to impose maximum hour and minimum wage standards on all other miners and producers in the region. The Court reasoned that by conferring on a majority of private individuals the authority to regulate "the affairs of an unwilling minority," the law was "legislative delegation in its most obnoxious form; for it is not even delegation to an official or an official body, presumptively disinterested, but to private persons whose interests may be

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necessary resources of flexibility and practicality, which will enable it to perform its function in laying down policies and establishing standards, while leaving to selected instrumentalities the making of subordinate rules within prescribed limits and the determination of facts to which the policy as declared by the legislature is to apply.").

[26] *See, e.g.*, Alexander Volokh, *The New Private-Regulation Skepticism: Due Process, Non-Delegation, and Antitrust Challenges*, 37 HARV. J.L. & PUB. POL'Y 931, 955 (2014) ("Nor is there any difference between public and private delegations."); Neil Kinkopf, *Of Devolution, Privatization, and Globalization: Separation of Powers Limits on Congressional Authority to Assign Feder Power to Non-Federal Actors*, 50 RUTGERS L. REV. 331, 358-65 (1998) (describing the nondelegation doctrine as requiring only an intelligible principle, regardless of whether authority is delegated to a private or public non-federal actor). Whether the courts treat public and private delegations differently is not an issue that this memorandum will address.

[27] The Supreme Court has previously found broad delegations to regulate in the "public interest" or in a "fair and equitable" manner to satisfy the intelligible principle test. Nat'l Broad. Co. v. United States, 319 U.S. 190, 216 (1940); Yakus v. United States, 321 U.S. 414, 420 (1944).

[28] *See, e.g.*, Eubank v. City of Richmond, 226 U.S. 137 (1912); Thomas Cusack Co. v. City of Chicago, 242 U.S. 526 (1917); Washington ex rel. Seattle Title & Trust Co. v. Roberge, 278 U.S. 116 (1928); Carter v. Carter Coal Co, 298 U.S. 238 (1936). Sunshine Anthracite Coal Co. v. Adkins, 310 U.S. 381 (1940); Gen Elec. Co. v. N.Y. State Dep't of Labor, 936 F.2d 1448 (2nd Cir. 1991); City of Dallas v. FCC, 165 F.3d 341 (5th Cir. 1999).

[29] Currin v. Wallace, 306 U.S. 1, 15 (1939).

[30] 298 U.S. 238 (1936) [hereinafter *Carter Coal*]. Prior to *Carter Coal*, the Court had upheld relatively broad delegations to private entities. For example, in *St. Louis, I.M. & S. R. Co. v. Taylor*, 210 U.S. 281 (1908), the Supreme Court approved of a law that authorized the American Railway Association to "designate to the Interstate Commerce Commission the standard height of draw bars for freight cars..." *Id.* at 286.

and often are adverse to the interests of others in the same business."[31] Although appearing to characterize the wage and hour provisions as an unlawful "delegation" to a private entity, the Court held that the provision in question was "clearly a denial of rights safeguarded by the due process clause of the Fifth Amendment."[32]

Carter Coal has engendered significant confusion as to whether the Court's holding was based on an extension of the above-outlined nondelegation principles, or was instead grounded in the Fifth Amendment's guarantee of "due process of law."[33] The Due Process Clause, in part, seeks to ensure principles of fundamental fairness, including the notion that decision makers must be disinterested and unbiased.[34] These general principles may be offended when the federal government authorizes a private party to exercise coercive power over another that could be used in a biased or arbitrary manner.[35] As one commentator has summarized: "If a delegation creates the opportunity for private interests to dominate the use of governmental power, then those against whom the power is used may well have suffered deprivations without due process."[36]

In *Carter Coal*, the Court clearly articulated the due process problems involved with providing regulatory authority to private entities:

> The difference between producing coal and regulating its production is, of course, fundamental. The former is a private activity; the latter is necessarily a governmental function, since, in the very nature of things, one person may not be entrusted with the power to regulate the business of another, and especially of a competitor. And a statute which attempts to confer such power

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[31] *Carter Coal*, 298 U.S. at 311.

[32] *Id.* at 311-12.

[33] U.S. CONST. amend. V ("No person shall be...deprived of life, liberty, or property, without due process of law..."). *See, e.g.*, Ass'n of Am. R.R. v. Dep't of Transp., 721 F.3d 666, 671 n.3 (D.C. Cir. 2013), *vac'd*, 135 S. Ct. 1225 (2015) ("At least one commentator has suggested that the 'doctrine forbidding delegation of public power to private groups is, in fact, rooted in a prohibition against self-interested regulation that sounds more in the Due Process Clause than in the separation of powers.' *Carter Coal* offers some textual support for this position, describing the impermissible delegation there as 'clearly a denial of rights safeguarded by the due process clause of the Fifth Amendment.' While the distinction evokes scholarly interest, neither party before us makes this point, and our own precedent describes the problem as one of unconstitutional delegation.") (internal citations omitted); Brief of Professor Alexander Volokh as Amicus Curiae in Support of Petitioners at 2-3, Dep't of Transp. v. Ass'n of Am. R.R., 575 U.S. __, (2015) (arguing that the D.C. Circuit in *Association of American Railroads v. Department of Transportation* was wrong to strike down the statute using a delegation analysis and should have applied a due process analysis instead); A. Michael Froomkin, *Wrong Turn in Cyberspace: Using ICANN To Route Around the APA and the Constitution*, 50 DUKE L.J. 17, 153 (2000) ("The *Carter Coal* doctrine is known as a nondelegation doctrine, but in a way the name is misleading. Unlike the public nondelegation doctrine, which relies on the separation of powers to prevent Congress from making standardless delegations to administrative agencies, the *Carter Coal* doctrine forbidding delegation of public power to private groups is, in fact, rooted in a prohibition against self-interested regulation that sounds more in the Due Process Clause than in the separation of powers.").

[34] *See Carter Coal*, 298 U.S. at 311; *Eubank*, 226 U.S. at 143-44 (invalidating a city ordinance on the grounds that it established "no standard by which the power thus given is to be exercised; in other words, the property holders who desire and have the authority to establish the line may do so solely for their own interest, or even capriciously...."). *See also* Marshall v. Jerrico, Inc., 446 U.S. 238, 242 (1980) ("The Due Process Clause entitles a person to an impartial and disinterested tribunal in both civil and criminal cases."). For a strong defense of the due process approach to private delegations, *see generally* Volokh, *supra* note 26 (identifying additional cases involving city ordinances and state statutes for support of the proposition that the Court has historically used the Due Process Clause to evaluate private delegations).

[35] *See Carter Coal*, 298 U.S. at 311. *See also* David M. Lawrence, *Private Exercise of Governmental Power*, 61 IND. L.J. 647, 659 (1986) ("The concern is that governmental power—power coercive in nature—will be used to further the private interests of the private actor, as to some different public interest. When a public official is permitted to exercise a public power, he is generally expected to do so in a basically disinterested way. The community expects him to act from some conception of what is good for the community or according to standards that seek to further community interests, as opposed to acting to further his narrow private interests.").

[36] Lawrence, *supra* note 34, at 661.

> undertakes an intolerable and unconstitutional interference with personal liberty and private property. The delegation is so clearly arbitrary, and so clearly a denial of rights safeguarded by the due process clause of the Fifth Amendment, that it is unnecessary to do more than refer to decisions of this court which foreclose the question.[37]

It is difficult to predict how, and under what standard, a modern hypothetical reviewing court would evaluate a private delegation. Moreover, it is not entirely clear what effect framing the issue as one of due process, rather than nondelegation, or vice versa, would have on a court's ultimate evaluation of the constitutionality of the delegation.[38] Nevertheless, when considering constitutional limits on private delegations that arise from cases like *Carter Coal*, it would seem that an important consideration is *to whom* power is given, and *over whom* that power may be wielded.

The U.S. Court of Appeals for the District of Columbia Circuit (D.C. Circuit) is the most recent court to offer a thorough explication of *Carter Coal* and the private delegation doctrine. In *Assoc. of American Railroads v. U.S. Department of Transportation*, the circuit court interpreted *Carter Coal* as establishing a strict prohibition on congressional delegations of authority to private entities. This is a position that has not been expressly adopted by the Supreme Court, and has been subject to some criticism.[39] The D.C. Circuit opinion flatly held that "[f]ederal lawmakers cannot delegate regulatory authority to a private entity. To do so would be 'legislative delegation in its most obnoxious form.'"[40] In reaching its holding, the court made a clear distinction between delegations to governmental versus private entities. Whereas Congress need only "prescribe an intelligible principle governing the statute's enforcement," when delegating authority to government agencies, "even an intelligible principle cannot rescue a statute empowering private parties to wield *regulatory authority*."[41] Notably, the D.C. Circuit saw no difference between a due process approach and a nondelegation approach, noting that "in any event, neither court nor scholar has suggested a change in the label would effect a change in the inquiry."[42]

American Railroads involved a challenge to § 207 of the Passenger Rail Investment and Improvement Act of 2008,[43] which delegated authority to Amtrak and the Federal Railroad Administration (FRA) to jointly develop "metrics and standards" to improve enforcement of Amtrak's statutorily established passenger rail service priority.[44] The circuit court struck down the law as an unlawful delegation to a private entity. In determining that Amtrak, which the court found to be a private entity, had been delegated "regulatory authority," the court found it significant that the law placed Amtrak on "equal footing" with the FRA in the development of the performance standards, rather than in the required "advisory or subordinate role."[45] The court did not, however, define what it considered to be the contours of "regulatory authority."

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[37] *Carter Coal*, 298 U.S. at 311-12.

[38] *See Ass'n of Am. R.R.*, 721 F.3d at 671 n.3 ("[I]n any event, neither court nor scholar has suggested a change in the label would effect a change in the inquiry."). Professor Alexander Volokh notes that analyzing delegations to private parties under the Due Process Clause, as opposed to the nondelegation doctrine, is preferable because it "better protects accountability:" Due process "is incorporated against the states through the Fourteenth Amendment;" "preserves the availability of a damages action for injured parties;" and "has consistently been applied to issues of bias and fairness." *See* Brief of Professor Alexander Volokh as Amicus Curiae in Support of Petitioners at 2-3, Dep't of Transp. v. Ass'n of Am. R.R., 575 U.S. __, (2015).

[39] *See generally* Volokh, *supra* note 26.

[40] *Ass'n of Am. R.R.*, 721 F.3d at 670 (quoting *Carter Coal*, 298 U.S. at 311).

[41] *Id.* at 671.

[42] *Id.* at 671 n.3.

[43] P.L. 110-432, Div. B (2008).

[44] *Ass'n of Am. R.R.*, 721 F.3d at 669-70. *See* 49 U.S.C. § 24308(c).

[45] *Ass'n of Am. R.R.*, 721 F.3d at 673.

On appeal, the Supreme Court vacated the D.C. Circuit opinion, holding that Amtrak was in fact a governmental entity.[46] Although disagreeing with the circuit court's characterization of Amtrak as private, the majority opinion did not reflect on the validity of the lower court's prohibition on the delegation of regulatory authority to private entities. Notably, Justices Alito and Thomas appear to have supported the lower court's view in their concurring opinions.[47] Justice Alito also emphasized, as did the D.C. Circuit, that "even the United States accepts that Congress 'cannot delegate regulatory authority to a private entity.'"[48] Nevertheless, while the reasoning in *American Railroads* may be probative of the D.C. Circuit's approach to private delegations, the opinion is not binding precedent within the D.C. Circuit, since it was vacated by the Supreme Court.

Even assuming, *arguendo*, that Congress cannot delegate "regulatory authority" to a private entity, Congress may nonetheless empower a private party to play a more limited role in the regulatory process. The Supreme Court has approved a number of more circumscribed delegations of authority to private entities. *Currin v. Wallace*[49] and *Sunshine Anthracite Coal Co. v. Adkins*[50] provide two such examples, which appear to be unaffected by the Supreme Court's decision in *American Railroads*.

In *Currin*, the Court upheld a law that delegated authority to regulate tobacco markets to the Secretary of Agriculture, but only upon the approval of two-thirds of the growers in the given regional market.[51] The law in question was an example of contingent legislation—or legislation that makes the effectiveness of a delegation contingent upon the occurrence of some future event. Citing to *Carter Coal*, and several other due process cases, the Court stated that "this is not a case where a group of producers may make the law and force it upon a minority."[52] Rather it was Congress, consistent with delegation principles, that had exercised its "legislative authority in making the regulation and in prescribing the conditions of its application."[53] Under *Currin*, it would appear permissible for Congress to delegate to private entities the ability to trigger the exercise of authority in a government official.[54]

The principles established in *Currin* were utilized by the U.S. Court of Appeals for the Sixth Circuit (Sixth Circuit) to uphold an important aspect of the Interstate Horseracing Act (IHA).[55] *Kentucky Division, Horsemen's Benevolent & Protective Association v. Turfway Park Racing Association* involved a challenge to the "horsemen's veto" of the IHA, a provision that prohibits interstate simulcasting of horseraces unless the host track has a written agreement with the necessary "horsemen's group."[56] The

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[46] *American Railroads*, 135 S. Ct. at 1228 ("[T]his Court now holds that, for purposes of determining the validity of the metrics and standards, Amtrak is a governmental entity.").

[47] *See id.* at 1238 (Alito, J., concurring) ("By any measure, handing off regulatory power to a private entity is 'legislative delegation in its most obnoxious form.'"); *id.* at 1254 (Thomas, J. concurring) ("Because a private entity is neither Congress, nor the President or one of his agents, nor the Supreme Court...the Vesting Clauses would categorically preclude it from exercising the legislative, executive, or judicial powers of the Federal Government...For this reason, a conclusion that Amtrak is private—that is, not part of the Government at all—would necessarily mean that it cannot exercise these three categories of governmental power.").

[48] *Id.* at 1237 (Alito, J., concurring).

[49] 306 U.S. 1 (1939).

[50] 310 U.S. 381 (1940).

[51] *Currin*, 306 U.S. at 6.

[52] *Id.* at 15.

[53] *Id.* at 16.

[54] *Currin* also distinguished its facts from those of *Washington ex rel. Seattle Trust Co. v. Roberge*. 278 U.S. 116. In *Roberge*, the Supreme Court struck down a city ordinance that allowed for the issuance of a permit to construct a group home but only if neighboring land owners consented. *Currin* described that case as "a prohibition of an inoffensive and legitimate use of property is imposed not by the legislature but by other property owners." *Currin*, 306 U.S. at 15-16.

[55] 15 U.S.C. §§ 3001-3007.

[56] 20 F.3d 1406 (6th Cir. 1994). The law defines "horsemen's group" as: "with reference to the applicable host racing association, (continued...)

case considered whether the provision constituted an unlawful delegation of authority to a private entity to determine, by either providing or withholding its consent, the permissibility of off-track betting. Relying primarily on *Currin*, the court held that "the horsemen's veto provision does not allow a private party to 'make the law and force it upon a minority'..."[57] Instead, the court viewed the provision as a form of conditional legislation, approved by the Supreme Court in *Currin* and other cases, in which "the Act merely affords the Horsemen a limited power to waive a restriction created by Congress."[58]

In *Adkins*, the Supreme Court upheld a provision of the Bituminous Coal Act of 1937,[59] which authorized private coal producers to propose standards for the regulation coal prices.[60] Those proposals were provided to the National Bituminous Coal Commission (a governmental entity), which was then authorized to approve, disapprove, or modify the proposal.[61] The Court approved of this framework, relying heavily on the fact that the private coal producers played a subordinate role to the Commission, which clearly retained ultimate authority over the regulation of coal prices. Specifically, the Court held:

> Nor has Congress delegated its legislative authority to the industry. The [private coal producers] function subordinately to the Commission. It, not the [private coal producers], determines the prices. And it has authority and surveillance over the activities of these [private parties]. Since law-making is not entrusted to the industry, this statutory scheme is unquestionably valid.[62]

The U.S. Court of Appeals for the Third Circuit (Third Circuit) applied the reasoning in *Adkins* to uphold a private delegation in *United States v. Frame*.[63] In that case, the Beef Promotion and Research Act of 1985[64] created the Cattleman's Beef Promotion and Research Board, a private entity comprised of cattle producers and importers designed to help strengthen the beef industry by coordinating "promotion and research."[65] The Act gives the Board the authority to collect a statutorily established assessment from the beef industry and to "take the initiative in planning how those funds will be spent," but "government oversight" over the Board was "considerable."[66] Relying on *Adkins*, the court held that "no law-making authority" had been entrusted to the Board primarily because the Board was "subject to the Secretary's pervasive surveillance and authority."[67] Board members were appointed, and removable, by the Secretary

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the group which represents the majority of owners and trainers racing there, for the races subject to the interstate off-track wager on any racing day." 15 U.S.C. § 3002(12).

[57] *Turfway Park Racing Assoc.*, 20 F.3d at 1416.

[58] *Id. See* Thomas Cusack Co. v. City of Chicago, 242 U.S. 526 (1917) (upholding a local ordinance that authorized the waiver of a prohibition on billboards if approved by one-half of affected property owners).

[59] 50 Stat. 72 (1937).

[60] *Adkins*, 310 U.S. at 388-89.

[61] *Id.* at 388.

[62] *Id.* at 399.

[63] 885 F.2d 1119 (3d Cir. 1989).

[64] P.L. 99-198, Title XVI, Subtitle A, *codified at* 7 U.S.C. §§ 2901-2911.

[65] *Frame*, 885 F.2d at 1123.

[66] *Id.* at 1128.

[67] *Id.* at 1129. Other lower court opinions suggest that if an agency is overseeing the actions of a private entity, it must do so with diligence. *See, e.g.*, Todd & Co. v. Securities & Exchange Com., 557 F.2d 1008, 1014 (3d Cir. 1977) ("The independent review function entrusted to the SEC is a significant factor in meeting serious constitutional challenges to this self-regulatory mechanism. Since it is a departure from the traditional governmental exercise of enforcement power in the first instance, confidence in the impartiality and fairness of the [private] Association's procedures must be maintained. The SEC, therefore, should not cavalierly dismiss procedural errors affecting the rights of those subjected to sanctions but should insist upon meticulous compliance by the private organization."); First Jersey Secur., Inc. v. Bergen, 605 F.2d 690 (3d Cir. 1979).

of Agriculture and nearly all activities of the Board, including "budgets, plans, or projects," required the Secretary's approval.[68]

Finally, the U.S. Court of Appeals for the Fourth Circuit (Fourth Circuit) has likewise approved of Congress delegating authority to a private entity to play administrative or ministerial roles in the implementation of a governmental program. In *Pittston Co. v. United States*, the court upheld a statutory framework that delegated authority to the Combined Fund, a private entity, to both collect premiums from coal operators and to disperse benefit payments to coal workers.[69] In doing so, the court noted that Congress had "set[] the specific formula for calculating the premiums to be paid" and that the Combined Fund was only "assigned the task of collecting the premiums *designated* by the statute from the persons *specified* by statute."[70] Moreover, the Combined Fund was directed to pay "benefits to the beneficiaries in an amount *specified* by the statute."[71] Because the Fund had no discretion to set the amount of the premium to be collected; the parties from which the premiums were to be collected; or the amount of benefits to be paid, the powers delegated to the private entity were "of an administrative or advisory nature, and delegation of them to the Trustees does not, we conclude, violate the nondelegation doctrine."[72]

Although the scope of Congress's authority to delegate power to private entities appears unsettled, and despite ongoing debates about the proper standards to be applied in such cases, a number of general principles can be gleaned from the above cited precedent. It would appear that broad delegations of regulatory power to private entities are generally disfavored. Furthermore, these delegations may be rejected under the persuasive force of the reasoning used in the vacated decision of *American Railroads* that Congress "cannot delegate regulatory authority to a private entity."[73]

As a result, a law that provides a private entity with ultimate authority to impose regulatory requirements that have a coercive effect on other private parties, or to otherwise exercise broad discretion to formulate policy, would likely raise constitutional concerns. However, some delegations to private entities have withstood constitutional scrutiny. Congress may authorize private entities to engage in more limited regulatory roles. For example, private entities may: trigger authority in a governmental entity; assist or aid a governmental entity in the exercise of its regulatory power; play an advisory or subordinate role to a governmental entity; exercise authority subject to the strict oversight and surveillance of a governmental entity; or administer a regulatory program in a purely ministerial manner.

Application to H.R. 3084

As discussed above, some courts have struck down delegations of regulatory authority to private entities, while other courts have found delegations of administrative or ministerial authority to private entities, like those discussed in *Pittston* and *Frame*, to be permissible.[74] However, the courts have not been particularly clear in defining the differences between an administrative or ministerial authority and one that becomes impermissible because it is regulatory in nature. In *American Railroads*, Justice Thomas described the authority at issue in the case as "the formulation of generally applicable rules of private conduct."[75] In his

[68] *Frame*, 885 F.2d at 1129.

[69] 368 F.3d 385 (4th Cir. 2004).

[70] *Id.* at 395.

[71] *Id.*

[72] *Id.* at 396.

[73] *Ass'n of Am. R.R.*, 721 F.3d at 670.

[74] *See Pittston*, 368 F.3d at 394-96; *Frame*, 885 F.2d at 1128-29.

[75] *American Railroads*, 135 S. Ct. at 1242 (Thomas, J., concurring).

own concurrence in the same case, Justice Alito determined that the power to set metrics and standards is "regulatory power" because private entities may be required to include the metrics and standards in their contracts[76] and "obedience to the metrics and standards materially reduces the risk of liability...."[77]

Taking the case law and these recent statements into account, it appears that a hallmark of regulatory authority is its *coercive* effect on private parties—i.e., whether the authorities delegated to the private entity allow it to impose rules upon other private parties with which those parties are required to comply. Several of the authorities granted to THADA could be viewed as impermissible delegations of authority to a private entity because of their coercive nature.

Authority to Create an Anti-Doping Program, Investigate Violations, and Impose Sanctions upon Violators

Under § 6 of H.R. 3084, THADA is instructed to "develop and administer the Thoroughbred horseracing anti-doping program for covered horses, covered persons, and covered horseraces."[78] In essence, THADA would be tasked with writing rules that define what substances are permitted and prohibited in horseracing. In order for covered persons and covered horses to participate in covered horseraces, they would be required to agree to comply with these rules. H.R. 3084 does provide guidance as to the contents of the anti-doping program. For example, the bill outlines substances that shall be on the initial lists of prohibited and permitted substances.[79] However, THADA would be permitted to amend these initial lists, with full discretion to choose the contents of the final lists. The final lists would not be subject to the approval of a governmental entity and can be changed by THADA at any time, subject to a notice and comment process to be established by THADA.[80]

As part of the anti-doping program mandated by H.R. 3084, THADA would also be responsible for developing procedures to test for the use of prohibited substances and procedures for investigating, charging, and adjudicating program violations.[81] THADA would be granted the same investigatory powers "as the State racing commissions have in their respective states...."[82] These powers could include access to facilities, search and seizure authority, the ability to issue and enforce subpoenas for testimony and documents, and "other investigatory powers."[83] THADA would also be empowered to impose sanctions, in accordance with rules on violations that it establishes.[84] "The rules shall impose up to and including a lifetime ban from horseracing" and shall provide opportunities for violators to reduce the otherwise applicable sanction by satisfying certain conditions.[85]

Authorizing THADA to create these kinds of rules may be regarded as an unlawful delegation of regulatory authority to a private entity. The anti-doping program created solely by THADA, empowered by law with significant discretion in its formulation, is likely to be characterized as imposing coercive requirements that control the conduct of other private entities, namely covered persons. If H.R. 3084 were

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[76] *Id.* at 4 (Alito, J., concurring) ("The fact that private rail carriers sometimes may be required by federal law to include the metrics and standards in their contracts by itself makes this a regulatory scheme.").

[77] *Id.*

[78] H.R. 3084, § 6(a).

[79] *Id.* at § 7(b).

[80] *Id.*

[81] *Id.* at § 6(a)(4)-(5).

[82] *Id.* at § 4(c).

[83] *Id.*

[84] H.R. 3084, § 7(f).

[85] *Id.*

enacted, covered persons and covered horses would be required by federal law to comply with THADA's anti-doping program in order to participate in covered horseraces. If they are suspected of noncompliance, covered persons and covered horses may be subjected to THADA's investigatory powers, which could include the authority to issue subpoenas and search and seize property. Violations of the anti-doping program can lead to sanctions, which are created and imposed by THADA, and can result in a covered person and/or covered horse being banned from thoroughbred racing for life.

The authority granted in H.R. 3084 can be compared to, and contrasted with, the authorities at issue in *Adkins* and the D.C. Circuit's consideration of *American Railroads*. In *Adkins*, a private entity was permitted to propose standards for the regulation of coal prices, which, when implemented, would be coercive requirements placed upon private conduct. That authority was deemed to be lawful because the private entity only *proposed* the rules, and the rules only went into effect if approved by a governmental entity, which could modify the rules as it saw fit. In contrast, under H.R. 3084, THADA, a private entity, would not only propose rules, as the private entity in *Adkins* did, but would also approve and modify those rules with no participation or supervision from a governmental entity. In the absence of government "supervision and surveillance over the activities"[86] of the private entity, the Court's reasoning in upholding the *Adkins* scheme cannot be applied to H.R. 3084.

The D.C. Circuit in *American Railroads* reasoned that the delegation of joint rulemaking authority to a private entity[87] and a governmental entity jointly, where each party had equal authority, constituted an unlawful delegation. If delegating equal authority to one private entity and one governmental entity is unlawful, then delegating authority to a private entity alone, with no government involvement, is also likely unlawful. Therefore, if a reviewing court were to adopt the D.C. Circuit's reasoning, it appears likely that it would consider a grant of rulemaking authority to a private entity like THADA, acting alone, to be unlawful.

Setting the Amount of a Fee

Under H.R. 3084, THADA would be funded through a process by which it establishes a fee "per racing start" adequate to cover implementation of the anti-doping program. State racing commissions would then be required, on a monthly basis, to remit to THADA an amount calculated by multiplying the applicable fee by the number of "racing starts" held in the state over the previous month. Each state racing commission has discretion, subject to applicable state law, to allocate, assess, and collect the amount that is to be remitted to THADA from the thoroughbred horseracing industry (a term that is not expressly defined in the bill.) Thus, while THADA would be the primary actor in establishing the overall cost of its continued operation, it is the state racing commissions that would determine, subject to state law, how to spread those costs amongst the members of the thoroughbred industry. No governmental entity would be involved in either establishing THADA's budget or in determining the amount of the fee.[88]

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[86] *Adkins*, 310 U.S. at 399.

[87] As discussed above, the D.C. Circuit ruled that Amtrak was a private entity. *Ass'n of Am. R.R.*, 721 F.3d at 677. However, this ruling was overturned by the Supreme Court, which determined that Amtrak was a governmental entity for the purposes of this suit. *American Railroads*, 132 S. Ct. at 1233. Therefore, the D.C. Circuit's decision, which was predicated on the fact that Amtrak was a private entity, was vacated. *Id.* at 1233-234.

[88] Under this arrangement, it would appear that the state's may control the sum that must be remitted to THADA by controlling the number of "racing starts" in their state in a given month. It should also be noted that the establishment of THADA as a "nonprofit corporation" may impose implicit restrictions on its finances. When used in law, the term "nonprofit" generally refers to an entity that must be operated on a not-for-profit basis and, as such, is subject to restrictions on its operations and spending that are not applicable to for-profit corporation (e.g., compensation paid by nonprofit entities to officers and others must generally be "reasonable"). *See, e.g.*, D.C. CODE § 29-404.41; NY Not-for-Profit Corp. Law § 202 ; *see also* 26 U.S.C. § 501.

These funding provisions raise several potential delegation concerns. First, may Congress delegate to a private entity like THADA the authority to determine the total cost that must ultimately be paid by a group of private individuals?[89] Several courts have evaluated delegations of authority to private entities to *collect* fees in the past.[90] In determining that the collection of a fee was a ministerial act, those courts focused on the fact that in each instance, the private entity did *not* have the authority to set the amount of the fee. For example, in *Adkins*, the Court upheld a delegation to a group of private coal producers because they acted subordinately to a government entity.[91] This subordination was evidenced, in part, by the fact that the government entity, not the private entity, had the authority to fix reasonable coal prices under the law. In *Frame*, the Third Circuit adopted this reasoning in finding that the collection of assessments across the beef industry by a private entity was not an unlawful delegation.[92] Again, the court focused on the fact that the amount of the assessment was set in statute by Congress and the private entity served a purely ministerial role in collection.[93] Finally, in *Pittston*, the Fourth Circuit upheld the authority of a private entity to collect premiums charged upon members of the coal industry.[94] Here, the court emphasized that the law "set out specific formulas for calculating the premiums to be paid" by each covered operator.[95] The Social Security Commissioner, not the private entity, had complete control over the amount to be paid based on the formula established in statute.[96] In each instance, the courts suggest that allowing the private entity to set the amount of the charge imposed on private parties would transform the delegation from an administrative or ministerial function into a regulatory authority.[97]

Based on this case law, it appears that authorizing THADA to determine the amount of the fee to be remitted by state racing commissions could be found by a reviewing court to constitute an unlawful delegation to a private entity. Under the arrangement that would be established by H.R. 3084, the state racing commissions would essentially act as a middle man: THADA would set the "per racing start" fee; the state racing commission would collect and remit the fee; and the thoroughbred horseracing industry would pay the fee. Therefore, although the state racing commission actually assesses and collects the fee, it could be argued that Congress has delegated authority to THADA to set the total amount that private parties are ultimately required to pay.

It should be noted that nothing in the bill appears to expressly *require* members of the thoroughbred industry to pay the assessed fee. Section 6(b) of the bill provides only that covered persons and their covered horses must, "as a condition of eligibility to participate in covered horseraces," agree to be "bound by...the anti-doping program developed pursuant to subsection (a)."[98] The THADA operation fee

[89] THADA does not directly impose a fee on private entities. Rather, it does so only indirectly, through the state racing commissions.

[90] *See Pittston*, 368 F.3d at 394-96 (evaluating a private entity's authority to collect premiums mandated in law); *Frame*, 885 F.2d at 1128-29 (evaluating a private entity's authority to collect assessments required by law).

[91] *See Adkins*, 310 U.S. at 399 ("Nor has Congress delegated its legislative authority to the industry. The members of the code[, a private entity,] function subordinately to the Commission[, a governmental entity]. It, not the code authorities, determines the prices. And it has authority and surveillance over the activities of these authorities. Since law-making is not entrusted to the industry, this statutory scheme is unquestionably valid.") (internal citations omitted).

[92] *Frame*, 885 F.2d at 1128-29.

[93] *Id.* at 1129 ("Therefore, we hold that the Beef Promotion Act does not constitute an unlawful delegation of legislative authority. In essence, the Cattlemen's Board and the Operating Committee[, private entities,] serve an advisory function, and in the case of collection of assessments, a ministerial one. Congress itself has set the amount of the assessments, while ultimately, it is the Secretary who decides how the funds will be spent.").

[94] *Pittston*, 368 F.3d at 396.

[95] *Id.* at 395.

[96] *Id.*

[97] *See Adkins*, 310 U.S. at 398; *Pittston*, 368 F.3d at 395-96; *Frame*, 885 F.2d at 1128-29.

[98] H.R. 3084 § 6(b).

is not provided for under "subsection (a)." It is only the general statement that "[t]he jurisdiction and authority of [THADA] are hereby imposed... as conditions upon the privilege... to participate in [covered] races," that may compel industry members to pay the THADA operating fee assessed by the state racing commissions. The states, however, could provide the state racing commissions with adequate authority under state law, if that authority does not already exist, to compel payment of the THADA operating fee.

The funding arrangement also raises the question of whether Congress may delegate to a state governmental entity the authority to determine who, and in what proportion, will have to contribute to the state's required remittance to THADA. H.R. 3084 provides the state racing commissions with wide discretion in determining how to collect the THADA operating fee. For example, it appears that the state racing commission could choose to place a larger percentage of the burden on one segment of the industry as opposed to another. This delegation of authority to a state entity is less problematic than a delegation of authority to a private entity. Congress often delegates authority to the states, especially in regard to the enforcement of federal law.[99] Courts have generally not invalidated these arrangements, instead analyzing the delegations under the intelligible principle test of the nondelegation doctrine and noting that such authorizations, when providing the state with the option to exercise federal power,[100] tend to further "another core constitutional value—that of federalism."[101] Moreover, at least one court has previously held that delegations to state governors do not raise the private delegation and due process concerns presented in *Carter Coal,* as a governor, and presumably other state officials, are "motivated to maximize the public good," as opposed to private parties, whose motivations may be "self-serving."[102]

Composition of THADA's Board

Section 5 of H.R. 3084 establishes a board of directors to govern THADA. The board would initially be comprised of the United States Anti-Doping Agency's (USADA) chief executive officer, five USADA board members, and five members "from different constituencies of the Thoroughbred industry who shall be appointed by" USADA.[103] These five additional appointees would be chosen from lists of two candidates submitted by representatives of different groups within the thoroughbred industry, including "owners and breeders, trainers, veterinarians, racing associations, State racing commissions, and jockeys."[104] H.R. 3084 instructs USADA to "provide diversity of industry membership on the board... to the greatest extent practicable," but it may "in its sole discretion," choose more than one person submitted from each thoroughbred constituency's list to serve on the board.[105] Furthermore, if after soliciting two sets of candidate lists from the representative groups, board positions still remain open, USADA "may choose one or more persons at large with substantial experience in the Thoroughbred industry as board

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[99] *See e.g.*, Harold J. Krent, *Fragmenting the Unitary Executive: Congressional Delegations of Administrative Authority Outside the Federal Government*, 85 NW. U.L. REV. 62, 80-82 (1990).

[100] H.R. 3084, rather than providing an option, appears to *require* the state racing commissions to allocate, assess, collect, and remit the THADA operating fee. Although the enforcement mechanism for this requirement is not clear, this memorandum will not address any 10th Amendment concerns that may arise from such an arrangement. Nor does this memorandum address any potential infringement on executive power that may arise from this arrangement. *See* Printz v. United States, 521 U.S. 898, 923 (1997) ("the power of the President would be subject to reduction, if Congress could act as effectively without the President as with him, by simply requiring state officers to execute its laws.").

[101] *Turfway Park Racing Assoc.*, 20 F. 3d at 1417.

[102] Lac Courte Oreilles Band of Lake Superior Chippewa Indians v. United States, 367 F.3d 650, 660 (7th Cir. 2004);

[103] H.R. 3084, § 5(b).

[104] *Id.* The "Thoroughbred industry" is not further defined in the bill.

[105] *Id.* at § 5(b).

members."[106] Board members would serve for terms of three years and "may serve no more than two consecutive full terms."[107]

The composition of the board may raise private delegation or due process concerns if THADA's ability to be a disinterested decision maker is questioned. The ability of the private entity to make fair decisions, free from bias, arbitrariness, and self-interest, is an important consideration with regard to the due process principles discussed above. On the one hand, H.R. 3084 contains a conflict of interest provision that appears to guard against biased decision makers serving on the board. Section 5(c) states that:

> no nominee or board member shall be--
>
> (1) an individual who has a financial interest in or provides goods or services to covered horses;
>
> (2) an official, officer, or serve in any governance or policymaking capacity for any Thoroughbred industry representative; or
>
> (3) an employee or have a business or commercial relationship with any of the individuals or organizations described in paragraphs (1) or (2).[108]

On the other hand, it seems that that USADA, a private entity that may not be presumptively disinterested,[109] is given broad discretion in choosing the five board members that represent the industry.[110] It is possible that one section of the industry, based on USADA's choices, could be over-represented on the board. Arguably more problematic, though, are the provisions regarding filling vacancies on the board once the initial board members reach their term limits. The five members representing the thoroughbred industry will continue to be appointed by USADA following the procedure outlined above.[111] However, vacancies in the six seats initially filled by USADA officials "will be filled pursuant to the provisions of the Authority's bylaws."[112] Since THADA's bylaws are not established by the bill, it is not known how those six board seats will be filled when vacancies arise. Therefore, it is possible that procedures and/or qualifications established in the bylaws may not result in disinterested decision makers and could raise due process concerns.

Distinguishing THADA from the United States Anti-Doping Agency

In support of the bill, it may be argued that THADA's envisioned role under H.R. 3084 is similar to that which is performed by the United States Anti-Doping Agency (USADA) under existing law. A thorough review of these two private entities, however, suggests that they may be distinguished based upon the circumstances of their creation, the primary source of their authority, and the specific powers delegated to each entity by Congress.

USADA serves as the "national independent anti-doping organization for the United States."[113] The agency, which is led by a governing board of "10 independent, experienced, and professional individuals, free from any conflicts of interest," was created by the United States Olympic Committee (USOC) in 2000 in response to a USOC task force recommendation that an independent governing body was

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[106] *Id.* at § 5(b)(5).

[107] *Id.* at § 5(d).

[108] *Id.* at § 5(c).

[109] *See Carter Coal*, 298 U.S. at 311.

[110] H.R. 3084, § 5(b).

[111] *Id.* at § 5(d).

[112] *Id.*

[113] 21 U.S.C. § 2001(b)(1).

necessary to better combat doping in U.S. Olympic sports.[114] USADA's authority primarily flows from its relationship with the USOC and the National Governing Bodies (NGB) for individual Olympic sports.[115] Through a contractual relationship with the USOC, USADA "conduct[s] drug testing, manage[s] test results, investigate[s] potential violations of anti-doping rules, and adjudicate[s] disputes involving anti-doping rule violations for participants in the Olympic and Paralympic movements..."[116] The agency implements this mission through the USADA Protocol for Olympic Movement Testing (Protocol).[117] Under the USOC bylaws, in order for an NGB to remain a member in good standing with the USOC, it must "comply with the... policies and procedures of the independent anti-doping organization designated by the [USOC] to conduct drug testing and adjudicate anti-doping rule violations."[118] More specifically, according to the USOC National Anti-Doping Policy, NGB compliance with the USADA Protocol "shall be a condition" of USOC funding and recognition.[119]

The federal government did not provide funding to USADA until 2002, and it was not until 2006 that Congress designated USADA as the "independent national anti-doping organization for the United States."[120] The entirety of the requirements imposed upon, and powers delegated to, USADA by law are included in 21 U.S.C. § 2001. Pursuant to that statutory provision, USADA shall:

> (1) serve as the independent anti-doping organization for the amateur athletic competitions recognized by the United States Olympic Committee and be recognized worldwide as the independent national anti-doping organization for the United States;
>
> (2) ensure that athletes participating in amateur athletic activities recognized by the United States Olympic Committee are prevented from using performance-enhancing drugs or prohibited performance-enhancing methods adopted by the Agency;
>
> (3) implement anti-doping education, research, testing, and adjudication programs to prevent United States Amateur Athletes participating in any activity recognized by the United States Olympic Committee from using performance-enhancing drugs or prohibited performance-enhancing methods adopted by the Agency;
>
> (4) serve as the United States representative responsible for coordination with other anti-doping organizations coordinating amateur athletic competitions recognized by the United States Olympic Committee to ensure the integrity of athletic competition, the health of the athletes, and the prevention of use by United States amateur athletes of performance-enhancing drugs or prohibited performance-enhancing methods adopted by the Agency.[121]

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[114] *See* S. Rpt. 113-281 at 1 (2014).

[115] U.S. Anti-Doping Agency Protocol for Olympic and Paralympic Movement Testing at 2, *available at* http://www.usada.org/wp-content/uploads/USADA_protocol.pdf ("The USOC has contracted with USADA to conduct drug testing, manage test results, investigate potential violations of anti-doping rules, and adjudicate disputes involving anti-doping rule violations for participants in the Olympic and Paralympic movements... For purposes of transmittal of information by USADA, the USOC is USADA's client.").

[116] *Id.*

[117] *Id.*

[118] Bylaws of the United States Olympic Committee, effective as of September 25, 2015 at 32, *available at* http://www.teamusa.org/~/media/TeamUSA/Documents/Bylaws%20approved%209%2025%2015.pdf.

[119] United States Olympic Committee National Anti-Doping Policy, §§ 4.1-4.2, effective as of January 1, 2015 ("As a condition of membership and recognition by the USOC and in fulfillment of any contractual relationship with the USOC all [NGBs]... shall adhere, in all respects, to the applicable provisions of the... USADA Protocol...") ("NGB compliance... with... the USADA Protocol shall be a condition of USOC funding.").

[120] 21 U.S.C. § 2001(b)(1).

[121] 21 U.S.C. § 2001(b).

In light of this basic description of the history and functioning of USADA, it would appear that THADA, as proposed under H.R. 3084, would differ greatly from USADA. First, the federal government had little involvement in USADA's creation.[122] Indeed, USADA was operating an anti-doping program before it was recognized by Congress as the nation's official anti-doping body or received any federal funding. In contrast, THADA, although private, would be created entirely through an act of Congress.

Moreover, most, if not all, of the coercive power USADA exercises would appear to stem from its relationship with USOC and NGBs, rather than from specific statutory delegations provided to it by Congress. As a result of private arrangements between USOC, USADA, and the NGBs, athletes and NGBs are required to comply with the USADA Protocol as a condition of membership in the USOC and participation in Olympic sports. As characterized by the Office of National Drug Control Policy, "USADA's protocol provides its jurisdiction over organizations and athletes through *contract and agreement*."[123] In contrast, THADA's jurisdiction and authority over covered entitles would arise, not as a result of private contracts and agreements, but as a result of a delegation of coercive authority grounded in federal law. Although compliance with THADA's anti-doping programs would, in fact, be a condition of participation in covered horseraces, it would appear to be a statutorily mandated condition, as opposed to a condition that flows from private contractual arrangements.[124]

Finally, the actual powers delegated to THADA would appear to far exceed those delegated to USADA. As noted above, the federal statutory provision relating to USADA primarily imposes requirements on the agency rather than empowering it.[125] The most significant federal delegation to USADA under federal law is arguably language that authorizes the agency to "implement anti-doping... adjudication programs to prevent" the use of performance enhancing drugs.[126] USADA, however, appears to have had existing adjudicative authority as a result of its arrangement with the USOC.[127] In contrast, H.R. 3084 would directly delegate substantial regulatory, investigative, and enforcement powers to THADA. These powers, which—if H.R. 3084 were enacted—would find their source in federal law, include the authority to issue rules identifying prohibited substances, develop procedures to test for prohibited substances, adjudicate disputes, impose sanctions, and set fees. As a result, it would appear that H.R. 3084 would create an anti-doping authority with jurisdiction over thoroughbred horseracing that differs to a substantial degree from the existing anti-doping framework embodied in USADA.

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[122] USADA was created by the USOC, and although it is federally chartered, receives federal funding, and is governed, in part, by the Ted Stevens Olympic and Amateur Sports Act, 36 U.S.C. §§ 220501 *et seq.*, USOC is a private entity. *See* San Francisco Arts & Ath., Inc. v. U.S. Olympic Comm., 483 U.S. 522 (1987).

[123] *See* Letter from R. Gil Kerlikowske, Director, Office of National Drug Control Policy, to Congressman James Sensenbrenner, Jr., August 7, 2012 (emphasis added).

[124] A hypothetical illustration may be helpful to solidify this distinction as to the primary source of authority for the operation of each of these entities. If the federal authorizations pertaining to USADA were repealed, the agency would likely continue to function without major change. If however, H.R. 3084 were enacted and THADA were established, a subsequent repeal of THADA's statutory authorization would eliminate that entity's authority over thoroughbred horseracing and its activities would be severely impaired.

[125] 21 U.S.C. § 2001(b).

[126] 21 U.S.C. § 2001(b)(3).

[127] See *supra* notes 114-118.

Statement by
Shawn Smeallie
Executive Director, Coalition for Horse Racing Integrity
House Energy & Commerce Subcommittee on Digital Commerce and Consumer Protection
HR 2651 Horseracing Integrity Act
Friday, June 22, 2018

I appreciate this opportunity to voice the strong support of the Coalition for Horse Racing Integrity (CHRI) for H.R. 2651, the Horseracing Integrity Act. CHRI is a broad-based alliance of members of the horse racing industry dedicated to the creation of an independent horse racing anti-doping agency that provides uniform rules and penalties across all 38 racing jurisdictions in the United States.

CHRI's 15 members cover nearly every aspect of racing, including The Jockey Club, the Breeders' Cup, the Water Hay Oats Alliance (WHOA), and the Humane Society of the United States. Our coalition enjoys the support of breeders, such as the Thoroughbred Owners and Breeders Association and the Kentucky Thoroughbred Association/Kentucky Thoroughbred Owners & Breeders; racetracks, including NYRA, which owns Belmont and Saratoga, and Keeneland; and horse owners, such as Frank Stronach, who founded the organization that owns numerous prominent racetracks including Pimlico, Laurel, Santa Anita, and Gulfstream; Jeff Gural, the owner of Meadowlands, Tioga Downs and Vernon Downs; and Centaur Gaming, the owner of Hoosier Park and Indiana Grand racetracks and casinos in Indiana. We also have the strong support from more than 75 trainers, including hall of fame trainers Nick Zito and Jonathan Sheppard.

The coalition, and the legislation it supports, only came into existence because the horse racing industry has been unable to create a binding system of high quality regulations, uniformly applied across all United States racing jurisdictions – a goal the industry has shared since 2001 with the formation of the Racing Medication and Testing Consortium. The current system of 38 separate medication control programs for horses that regularly run across state lines is anachronistic and makes the system vulnerable to cheaters. While many in the horse racing industry have worked to address these challenges on a voluntary basis through the model rules contained in the National Uniform Medication Program (NUMP), adoption of that program has been sporadic and inconsistent: after years of effort, *less than 25% of all racing states have fully implemented the NUMP*.

H.R. 2651 will address this issue by establishing an independent medication control authority that will enforce uniform rules and penalties for horse racing. The new organization, called the Horseracing Anti-Doping and Medication Control Authority, or HADA, will be under the governance control of the non-profit, non-governmental United States Anti-Doping Agency (USADA). USADA has a proven track record of conducting anti-doping and medication control activities for all U.S. Olympic athletes, and its approach can be easily adapted to horse racing via HADA.

HADA's mission will be specifically limited to the very narrow area of medication control – akin to the Financial Industry Regulatory Authority's (FINRA) limited role as an independent, non-governmental agency that regulates the broker/dealer industry. It will not usurp the other facets of horse racing regulation that are conducted by the states.

HADA will be required to create a set of uniform rules, including lists of permitted and prohibited substances and practices. These permitted and prohibited substances and practices will be developed after considering international anti-doping standards and veterinarian ethical standards, and – perhaps most importantly – after consulting racing industry representatives and the public.

HADA will *not be funded by the federal government* – the horse racing industry will pay the funds necessary for the establishment and administration of the authority. The estimated cost of the proposal will be approximately $40-$45 per horse, per start.

The bill will cover all major horse racing breeds, including Standardbreds and Quarter Horses, and places U.S. racing in line with all other international racing jurisdictions by prohibiting the administration of medications on race day.

Opponents of the legislation often say that the current fragmented regulatory system with its myriad racing commissions creating inconsistent rules without centralized investigations, without coordinated penalties, and with woefully underfunded research is perfectly acceptable. They also say the current system is making some progress, and that this bill is a solution in search of a problem. Nothing could be further from the truth. Just because you're not looking, doesn't mean it's not there. From the doping scandals of the Russia Winter Olympics, cycling, baseball, and track & field, it is clear that the use of pharmaceuticals to enable performance enhancement is becoming more and more advanced. Unless you have an effective, comprehensive anti-doping program, the cheaters will ultimately win out.

Few of the key protocols of Olympic testing are common practice for horse racing. For example, out-of-competition (OOC) testing, which is essential for detecting the use of performance-enhancing substances that remain effective beyond their testing detection periods, only accounts for 1% of all testing of Thoroughbreds. OOC testing allows testers to show up unannounced to check for performance-enhancing drugs. In many other horse racing nations, OOC testing is conducted at least 10 times more than is the practice across the United States.

Another effective deterrent is uniform penalties. Under H.R. 2651, if a trainer gets caught cheating, there will be uniform and transparent rules and penalties, and they will be the same from state to state.

H.R. 2651 will ensure that sophisticated laboratory methods will be used to test equine samples. Qualified labs will ensure that cheaters will be caught, and add a layer of protection for trainers by having verifiable split samples to confirm any positive tests. The bill also creates a comprehensive solution for pari-mutuel horse racing by ensuring there are adequate and

competent investigators utilizing modern methodologies to look into suspicious trends and other areas where testing cannot always go.

Another tired argument against the bill is that it adds an expensive new, bureaucratic layer. Actually, we are *replacing* an ineffective, completely voluntary layer with an independent, non-governmental organization that actually knows how to implement and run the best anti-doping program in the world. This bill will require, for the first time, that racing place all jurisdictions under a single rulebook, with USADA's guidance. It will do so by redeploying existing funds supplemented by the stakeholders in the game to create a highly efficient, highly effective and highly coordinated national anti-doping system for pari-mutuel racing that will eclipse the rest of the world.

As we saw with the Olympics, baseball and cycling, once there is a perception that cheating is tolerated, the incentive for all to cheat grows exponentially – if only just to keep up. When there is a strong, independent doping program, athletes worry less that they are competing against a dirty competitor, and are less incentivized to cheat.

Racing needs to go there. The benefits to racing, to the equine athlete, and to the betting public are too great not to make this effort. Polls consistently indicate that the use of performance-enhancing drugs, abuse of therapeutic medications, and inadequate drug testing programs rank as the largest concerns among the sport's advocates and fans. In one recent poll, more than nine in 10 bettors said they want to see uniform medication policies happen faster than they are happening now, and 90% stated that they support having all states work from the same set of rules. It is clear that the fractured nature of medication regulations across all racing jurisdictions has undermined the public's confidence in horse racing, in addition to endangering its human and equine athletes.

Congress, and this Committee, long ago threw a lifeline to the racing industry by passing the Interstate Horseracing Act (IHA). IHA allows wagering across state lines, which now makes up nearly 90% of all wagers. Without it, the industry may not have survived. CHRI now asks the Committee to help us again by ignoring the short-sighted protests of a few and passing the H.R. 2651, the Horseracing Integrity Act. The future of this $40 billion sport and its 400,000 domestic jobs depend on it.

The U.S. is a global leader in production of racehorses: its regulatory system should be a leader as well.

THOROUGHBRED OWNERS AND BREEDERS ASSOCIATION

June 19, 2018

Chairman Bob Latta
Ranking Member Jan Schakowsky
Committee on Energy & Commerce
Unites States House of Representatives
Washington, D.C. 20515

Thank you for allowing me the opportunity to express our support for the Horseracing Integrity Act of 2017 (H.R. 2651).

My name is Dan Metzger, and I am the president of the Thoroughbred Owners and Breeders Association (TOBA). TOBA is a national trade organization whose mission is to improve the economics, integrity and pleasure of the sport on behalf of Thoroughbred owners and breeders.

In 2018, it is common for owners to race their horses in different states. Under the current system, each of an owner's horses may be subject to different medication rules and testing procedures because they compete in different jurisdictions. This system is confusing and nonsensical compared to the uniform regulations in place in other countries.

We believe that the passage of H.R. 2651 is in the best interests of owners and breeders alike. The bill would bring much-needed uniformity to medication standards throughout our country and finally align us with other major international racing jurisdictions.

The Horseracing Integrity Act would also bring independence to drug testing and the adjudication of violations through the creation of the Horseracing Anti-Doping and Medication Control Authority (HADA). This authority would ensure fairness and transparency in how all anti-doping matters and violations are handled. The increased integrity of the sport that would result from HADA's governance would only increase enthusiasm for horse racing and bring new owners and breeders to the game.

TOBA seeks to align itself with causes that benefit our members as well as the racing industry as a whole. We are confident that H.R. 2651 will improve the economics, integrity, and pleasure of horse racing not just for owners and breeders, but for everyone involved in the industry.

For the good of the sport, we ask that the committee chooses to support the Horseracing Integrity Act of 2017.

Dan Metzger
President

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NAARV
NORTH AMERICAN ASSOCIATION OF RACETRACK VETERINARIANS
P.O. Box 55168 | Lexington, KY 40555 | TEL: (859) 429-0652 | Fax: (859) 813-5249

June 19, 2018

The Honorable Bob Latta, Chairman
Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection
United States House of Representatives
2125 Rayburn House Office Building
Washington, D.C. 20515

The Honorable Jan Schakowsky, Ranking Member
Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection
United States House of Representatives
2322A Rayburn House Office Building
Washington, D.C. 20515

Re: North American Association of Racetrack Veterinarians **Opposition** to H.R. 2651

Dear Chairman Latta and Ranking Member Schakowsky:

Esteemed Members of the House Energy and Commerce Committee:

HR 2651 aims to create additional Federal regulation of horse racing specifically by centralizing and standardizing drug testing, listing permitted and prohibited substances (and creating penalty guidelines for violators) and eliminating the use of all race day medications, including the preventive medication Furosemide. At the State level, and more importantly regionally, centralized and standardized post race AND out-of-competition drug testing by accredited labs already exists, as does a detailed and almost exhaustive list of permitted and prohibited substances with appropriate penalty levels for rules infractions. Additionally, all race day medications are already prohibited except for ONE-the highly regulated use of Furosemide (Lasix or Salix).

Furosemide mitigates the severity of EIPH - Exercise Induced Pulmonary Hemorrhage also known as "Bleeding." Its use is restricted to horses that have been placed on the Lasix list by their veterinarians and the dose and time of administration are predetermined and highly regulated. Most in our industry will attest that tremendous strides have been made regionally to ensure a consistent level of regulation of the rules of racing. Critics will cite individual idiosyncrasies within a specific jurisdiction to their point. However, the most unifying concept among horsemen, veterinarians and the betting public alike is the regulated and consistent use of race day Furosemide. As veterinarians, we prioritize the health and wellness of the horse, and to those who work with racehorses, that DEMANDS the use of furosemide during their most stressful endeavor, running a race. Attached please find a bullet point list compiled by Dr. Fenger, Secretary of NAARV, which summarizes, in some detail, the reasons for our collective support for the continued use of Furosemide in racehorses. Thus, as written, we are NOT in support HR 2651.

Professionally submitted,

Nicholas L. Meittinis, DVM

President

NAARV

Furosemide and Horse Racing

Clara K. Fenger, DVM, PhD, DACVIM

- The lungs are specialized organs for the transfer of oxygen and carbon dioxide between the atmosphere around us and the blood.
- This unique purpose is achieved by a microscopically thin membrane lining the tiny air chambers (alveoli) that are the end destination of each breath.
- During exercise of elite athletes, including horses, greyhounds and the most elite of human competitors, the combination of great negative pressures in the chest and the high blood pressure in the pulmonary circulation, culminate in rupture of some of these thin membranes. This rupture causes bleeding into the lungs, a condition called Exercise Induced Pulmonary Hemorrhage, or EIPH.
- Racing horses, as a consequence of their status as elite athletes, experience EIPH almost uniformly. While they may not suffer EIPH in every race, studies have shown that over time all horses will suffer EIPH at some point in their racing careers.
- Furosemide (Lasix) is the only medication clearly demonstrated to ameliorate EIPH, and prevent the severest form, Grade 4/4.
- Criticism about the use of furosemide in horse racing has stemmed from its use in over 90% of the equine racing population; however, like other preventative health measures, such as the use of seatbelts and vaccines, prevention works best when used at all times.
- The use of furosemide as a pre-race treatment, carefully controlled and regulated, is the best example of uniformity within the horse racing industry. All jurisdictions currently require Lasix to be administered 3 to 4 hours before a race, with an "L" listed in the program for complete transparency to the betting public.
- Pre-race furosemide protects the health and welfare of the horse, the rider, and the interests of the betting public.
- The use of furosemide is not a mandatory practice because some individual horses may not tolerate the use of furosemide; therefore, its use is a decision by the trainer after consultation with the horse's owner and veterinarian.
- Critics of furosemide have suggested that the use of diuretics may mask the presence of other illegal substances by making it more difficult to identify drugs in diluted urine. In fact, this is the main reason that furosemide is banned in international human sports. However, research has clearly shown that after 2 ½ hours the diluted urine effect has abated. This is the reason furosemide is administered in horse racing at 3 - 4 hours before the competition.
- It has been pointed out that the World Anti-Doping Association (WADA) considers furosemide a performance enhancing drug. This is based on its use as a diuretic aid to help drop weight in humans who participate in sports with weight classes, such as boxing and wrestling. There is no similar weight class associated with horse racing, making this WADA concern irrelevant in horse racing.
- Both organizations that represent North American racetrack practitioners, the AAEP and NAARV recognize furosemide as a benefit to the health and welfare of the racing horse.

NAARV

HEARING BEFORE THE UNITED STATES HOUSE OF REPRESENTATIVES

COMMITTEE ON ENERGY & COMMERCE

SUBCOMMITTEE ON DIGITAL COMMERCE & CONSUMER PROTECTION

June 22, 2018

Testimony of Chauncey Morris, Executive Director, on behalf of the

Kentucky Thoroughbred Association

I. Introduction

Chairman Latta, Ranking Member Schakowsky, Members of the Committee,

Since 2015, our Association has supported the Horseracing Integrity Act. We have again been asked to endorse the present form of the legislation.

Horses provide direct employment to 56,603 Kentuckians and create a direct economic impact of $3.9 billion. Kentucky is home to the largest foal crop in North America, the biggest and most valuable horse sales in the world, the leading exporter of lives horses in the United States, and counts five Thoroughbred racetracks, which in the past five years, are responsible for generating wagering handle ranking third behind New York and California. Kentucky has achieved such results due to a tradition which emphasizes quality horse racing and a level playing field created by fair state regulation and a persistent drive amongst our regulators to improve the status quo. Our Association represents owners and trainers at Keeneland and Churchill Downs, industry leaders who have been integral to the recent increase in viewership and wagering on Thoroughbred racing.

II. Regulation and Drug Testing

Since 2015, the very existence of the Horse Racing Integrity Act has provided ample opportunity to find common ground, and act as a catalyst to stimulate conversations between important stakeholders to explore ways to improve the integrity of our sport. Concurrently, questions about the ability of our sport to penalize suspected cheaters and protect innocent trainers have occurred, raising important questions on whether the model of State racing commissions possess sufficient authority to investigate and penalize, and more importantly whether laboratories currently used by these same racing commissions are in fact delivering value for our sport and all its participants including: competing owners, trainers whose livelihoods and reputations are on the line if and when a positive is detected, the racetracks paying for current testing, the racing commissions whose reputations as honest adjudicators define the strength or weakness of their industry, and ultimately the horseplayer, i.e. the consumer, who assumes their wager is made in good faith. Indeed, robust and transparent governance and constant qualitative analysis, more than is currently provided, should be placed over each licensed laboratory.

Unfortunately, both the supporters and opponents of the current legislation have been unwilling to compromise. The total amount of money US Thoroughbred racing currently spends to collect samples for drug testing is immaterial if these same samples are subjected to substandard or inconsistent testing, the fallout of which reinforces questions about cheating in popular culture. The Oversight model currently in the legislation grants an impartial third-party total authority over drug testing in the United States, meaning its actions, as an unintended consequence, will have impact on the business plans of racetracks. The elimination of race-day medication in the present form of the legislation is viewed by some desirous of maintaining the status quo as the only goal of its supporters, and the rest of the principles merely serve as a disingenuous "Trojan Horse;" similarly there are some supporters who would be happy to eliminate Lasix and believe those advocating for its use are against progress. While its use places the United States out-of-sync with major racing countries, this issue should be set aside temporarily to seek compromise.

III. Conclusion

It is our belief racing needs to collectively work on a blueprint for ideal governance to tackle these issues, something which legislation is unneeded. Collective efforts through the Racing & Medication Testing Consortium have achieved impressive results since its inception. Our Association remains committed to this process and likewise is committed towards achieving improvement through legislation or alternative means which improves the regulation of medication usage in Thoroughbred racing. The five members of Thoroughbred racing providing oral testimony on June 22nd represent vital constituencies which must come together to best position our sport for the next 100 years.

Statement by
William M. Lear, Jr.
Vice Chairman, The Jockey Club
House Energy & Commerce Subcommittee on Digital Commerce and Consumer Protection
HR 2651 Horseracing Integrity Act
Friday, June 22, 2018

As Vice Chairman of The Jockey Club, it has been my privilege over the past 3+ years to be deeply involved in the development of the regulatory model embodied in HR 2651 and in discussing its features with an array of horse industry constituents. With the evolution of the legislation and the occurrence of events both within and outside of our industry, the focus of certain questions concerning the bill is also evolved. The following are some of the questions most recently posed about the legislation in its current form, together with our responses to those questions. They are presented in an effort to assist the Subcommittee in its consideration of this important initiative.

1. Why have you chosen the Federal Trade Commission?

The FTC was chosen because its jurisdictional mandate includes both the integrity of competition and consumer protection. Its Bureau of Consumer Protection works to prevent fraud, deception and unfair business practices. The use of performance-enhancing drugs and methods in horseracing constitutes a fraud upon not only fellow competitors but also racing fans and those who wager upon horse races through the means of interstate commerce. It also constitutes a threat to the well-being of the horses involved in the races.

2. By withdrawing from the states the authority to regulate medication in horseracing, and mandating their participation in the regulatory system established by this legislation, does this legislation run afoul of the anti-commandeering rules recently invoked by the Supreme Court to invalidate PASPA?

In *Murphy v. National Collegiate Athletic Association*, No. 16-476 (May 14, 2018), the Supreme Court held that the Professional and Amateur Sports Protection Act ("PASPA") was unconstitutional because it "unequivocally dictates what a state legislature may and may not do" in violation of the Tenth Amendment of the federal Constitution and the anti-commandeering doctrine developed thereunder. The anti-commandeering doctrine operates as a limit on the powers of the federal government and prohibits the federal government from "commandeering" the legislative processes of the states by compelling them to enforce federal laws or policies. *New York v. United States*, 505 U.S. 144 (1992). In *Murphy*, the Supreme Court found PASPA to be in violation of the Tenth Amendment because it forced states to pass legislation prohibiting sports betting. Similarly, in *New York*, the Supreme Court found the Low-Level Radioactive Waste Amendments Act violated the Tenth Amendment because the extreme punishment for failure to implement a federal policy ("taking title" to and assuming liability for nuclear waste) was deemed effectively to force states into adopting a federal policy. 505 U.S. 144. In each case, the Supreme Court made a point to highlight that the anti-commandeering principle does not affect the ability of Congress to directly regulate an activity within the scope of its power—

Congress simply cannot commandeer state legislatures and compel them to require or prohibit certain acts.

The Horseracing Integrity Act of 2018 does not run afoul of any Tenth Amendment prohibitions as it does not, in any sense, "dictate what a state legislature may and may not do" or force states into adopting a federal policy or regulatory scheme. For example, although the Horseracing Anti-Doping and Medication Control Authority ("Authority") is authorized to charge fees to fund its operations under the draft legislation, states have the option (a) to collect and remit fees to the Authority or (b) decline to do so, in which case the fees will be collected by the Authority directly from the racetracks and other industry participants as determined by rules promulgated by the Authority. In addition, while the draft legislation permits the Authority to request state and local law enforcement agency assistance in connection with Authority investigations and searches and seizures, the cooperation of such agencies is completely voluntary. In other words, the states retain their sovereign powers to enforce their own laws and *may* choose <u>not</u> to cooperate with the Authority's requests. Consequently, the constitutional limits on commandeering are not violated.

3. Why is this issue important to the public interest rather than simply being a matter of parochial concern within one industry?

The public interest that serves as the foundation of this legislation lies in the protection of horses, the integrity of competition, and in the integrity of wagering. Unlike human competitors, horses involved in competition have no choice regarding the medications put into their systems and the methods used to treat them. This legislation would establish an entity to regulate not only medications which can enhance performance, thereby giving a horse an inappropriate advantage, but also medications which can harm the horse by enabling it to participate in a race when it should not be racing at all. Moreover, unlike other sports, Congress has authorized the use of the instrumentalities of interstate commerce to wager upon horse races. If Congress believes it is in the public interest to allow such wagering, and to establish the conditions under which it may occur, it also follows that it is in the public interest for that wagering to be conducted without corrupt influences. Each year, approximately $11 billion is wagered in the United States on Thoroughbred horse races alone. The results of many of those horse races are impacted by the use of performance-enhancing drugs and methods, but the existence of those in the horse at the time of the race and at the time wagers upon that race are paid is not known until well after the fact when the results of post-race tests are revealed. It is, therefore, critical to the integrity of wagering on horse races that a system be in place which is uniform nationwide and rigorous in order to more effectively prevent the use of performance-enhancing drugs at all, in addition to punishing those who are caught using them.

4. Why will this bill cost more than states are currently paying for medication regulation?

The primary driver of increased costs is the need for more out-of-competition testing. There is no jurisdiction in the United States which today conducts out-of-competition testing at a level commensurate with that practiced by the leading racing jurisdictions around the world. Knowing what we know today about drugs that can be used to boost performance of human and equine athletes, and methods which can be used for those purposes and yet be undetectable in

tests run at the time of or after the competition is concluded, the only way to ensure clean competition is with a robust out-of-competition testing program.

The Association of Racing Commissioners International (which Ed Martin serves as Executive Director) has finally grasped this reality. After 3 years of deliberation, in December 2016, the ARCI approved a model OOCT rule which includes lists of prohibited substances closely modeled after World Anti-Doping Agency (WADA) code. The opposition to our bill has targeted the increased costs to the industry when, in fact, the new ARCI out of competition testing rule will carry with it comparable increased costs.

5. Why can't you address this issue within your industry or by agreement among the states rather than asking Congress to intervene?

The conflict on display today between the two sides represented at the hearing indicates why we have been unable to resolve this within our industry. Unlike most other sports, horseracing has no single governing entity to prescribe rules that all participants must follow. Even within some segments of the industry which are united with respect to this legislation, there is disagreement upon certain important elements of medication regulation. For example, although Mr. Foreman and Mr. Hamelback are united in their opposition to this legislation, they are divided upon one of the most fundamental issues relating to medication regulation, specifically the list of permitted and prohibited medications. Mr. Foreman's group supports the ARCI's so-called NUMP ("National Uniform Medication Program") list of approved medications while the HBPA does not. This is just one example.

The advocates of this legislation only chose to ask Congress to intervene after attempting an industry-based solution and a solution based upon an interstate compact. Many years ago, legislation establishing a state compact to regulate medication was drafted and submitted to several states. Only one state, Kentucky, adopted the legislation. None of the other 30+ jurisdictions that regulate horseracing saw fit to adopt it. An interstate compact binds only the states that adopt it, and then only to the extent that those states are willing to give up their regulatory authority to the compact; and any state is free to withdraw from a compact at any time by action of its legislature.

In reality, there has been much conversation, but only imperfect efforts at achieving national uniformity and appropriate rigor in medication regulation on a state-by-state basis. While many states have, acting individually, adopted all or some portions of the NUMP over a period of several years, at the same time there has been a litany of actions and inactions by various states that demonstrates the ineffectiveness of state-based regulation. For example:

(1) In Pennsylvania, the regulatory authorities resolved a medication violation by declaring two winners in the same horserace and for many years the same regulatory authorities allowed two-year-olds to race while receiving Lasix on race day even though the regulation on their books prohibited the administration of race day Lasix to two-year-olds; that same Pennsylvania regulatory authority became a poster child for grossly ineffective oversight in the criminal trial of a rogue trainer, Murray Rojas.

(2) In the state of Kentucky, which is the birthplace of more than a third of all American thoroughbreds and the home of the Kentucky Derby, a court recently overturned the absolute insurer rule which makes trainers responsible for guaranteeing that the horses within their care run races without having performance-enhancing drugs in their system. This ruling places Kentucky at odds with other major racing jurisdictions, including California, Louisiana, Florida, West Virginia and New Jersey.

(3) In the state of California, which boasts more races and more graded stakes races than any other state and is home to the most respected testing laboratory in America, regulators are encumbered by the most complex and time-consuming rulemaking scheme in the United States (including the Federal government's Administrative Procedures Act). This cumbersome mechanism was on full display in the efforts by California regulators to adopt a new rule regarding the third-party administration of Lasix, one of the key components of the NUMP. Whereas many states adopted that rather straightforward rule change in a matter of months, in California the regulatory process took six years to complete.

(4) In Indiana in 2015, the state Horse Racing Commission terminated the contract with its RMTC-accredited lab after a quality assurance review of the lab found major failures in the lab's testing results: the lab failed to detect seven drug positives, including one Class I substance, in a 26-day period.

(5) In the spring of 2017, the Florida Division of Pari-mutuel Wagering dismissed medication violation charges against more than 50 trainers due to errors in the collection of post-race blood samples.

(6) In May of 2017, the West Virginia Racing Commission rescinded seven earlier rulings of violations for the use of a prohibited drug, naproxen, because the screening levels at various labs were not "in tune with each other."

As noted at the outset of this Statement, the proposed legislation now before the Subcommittee has evolved somewhat from its original form as filed by Representatives Barr and Tonko in July 2015. The changes made to the bill have been in response to suggestions for improvement arising from within our industry and from a variety of sources in Congress. While the proposed legislation has undergone some changes, one thing that has not changed is the critical need for it in America's horseracing industry. Three years ago, working in conjunction with the leaders of the WHOA organization, we posed the following set of questions to those favoring the status quo in the regulation of horseracing medication:

1. Do we have the same medication rules in place in every racing jurisdiction in the United States, or even in all of the major racing jurisdictions?

2. Do we have the same testing rules and procedures in place in every one of those racing jurisdictions, including best practices out-of-competition testing?

3. Do we have the same procedures and standards in place for laboratories, and the same contractual arrangements with laboratories, in every racing jurisdiction? And, are all the labs accredited to international standards?

4. Do we have the same processes for investigation, prosecution and adjudication of rules violations in place in every racing jurisdiction in America?

5. Do we have the same system of penalties in place in every racing jurisdiction, and is there consistency in the application of those penalties across jurisdictions--meaning, does the same offense result in the same penalty in California as it does in New York as it does in Kentucky as it does in Louisiana?

6. Do we have a system in place that can react quickly and uniformly throughout the nation to address the latest new drug that appears on the scene and is being used by those who would cheat to gain an inappropriate advantage?

7. Do we have a medication regulatory authority in place that can speak with one voice to the authorities in other nations when the need to do so arises, such as was the case recently with respect to the British Horseracing Authority's new steroid policy?

We concluded that list of questions with this statement: "We respectfully submit that the answer to each and every one of these questions is 'No.' The big picture, then, is that the status quo features none of the elements that any fair-minded observer would believe to be essential for true uniformity and appropriate rigor in the regulation of medication in Thoroughbred racing. It is time for a change--long past time."

Today, we again respectfully submit that even after the passage of three years, and the expenditure of significant efforts to achieve change by means other than the approach offered by HR2651, the answer to every one of these questions remains an unequivocal "No." The passage of HR 2651 and the implementation of its provisions will at long last allow us to change those answers to "Yes."

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June 20, 2018

The Honorable Bob Latta
Chairman
Committee on Energy & Commerce
United States House of Representatives
Washington, DC 20515

The Honorable Jan Schakowsky
Ranking Member
Committee on Energy & Commerce
United States House of Representatives
Washington, DC 20515

Dear Chairman Latta and Ranking Member Schakowsky:

Thank you for allowing The Stronach Group to share with you why we support the Horseracing Integrity Act of 2017, H.R. 2651.

Please allow me to introduce myself. My name is Belinda Stronach and I am the President and Chairman of The Stronach Group — a company that was founded in 2011 by my father Frank Stronach, who continues to serve as Honorary Chairman.

The Stronach Group is one of the largest owners and operators of Thoroughbred racetracks in The United States. Within our portfolio we hold some of the most recognized brands in the industry, including: Santa Anita Park, “The Great Race Place”; Pimlico Race Course, home of the legendary Preakness Stakes; Gulfstream Park, home of the Pegasus World Cup Invitational — the world’s richest Thoroughbred horse race; Laurel Park; Golden Gate Fields; Portland Meadows; and Rosecroft Raceway.

It is well documented that The Stronach Group is a strong proponent for uniformity in Thoroughbred horse racing and that we support the abolishment of race-day medication.

While our efforts to eliminate the use of race-day medication started years before, in 2014 The Stronach Group issued a letter written by Frank Stronach to our fellow racetrack operators, urging them to come together to discontinue the practice of race-day medication. At that time, we also vowed support for the National Uniform Medication Program (NUMP) hoping that states would adopt and implement the reforms by September 1st, 2014.

STRONACH
GROUP

As this committee is no doubt aware, to date these efforts have not come to fruition. Horses continue to race on medication administered only hours before they race. The United States is the only major racing jurisdiction in the world where this practice remains true.

NUMP still has not been adopted by all jurisdictions. Out of the four states in which we operate, Maryland is the only one to adopt NUMP but has yet to fully implement all of its components.

Our sport will benefit greatly from a level playing field, uniformity and federal oversight. Presently, there are 38 racing jurisdictions with different rules, regulations and penalties. These inefficiencies are both ineffective and costly and compromise the integrity of our sport.

The Stronach Group believes that this much needed reform can be achieved with the introduction of the Horseracing Integrity Act. We need bipartisan legislation that will establish a uniform set of rules, testing procedures, and penalties under the Horseracing Anti-Doping and Medication Control Authority (HADA). Uniformity and regulation exists for many other major sporting platforms, so why not ours?

The Stronach Group believes the sport of horse racing is the last great sporting legacy platform to be modernized. If we expect our sport to grow for future generations, we need to raise our standards. Efforts under HADA to establish oversight and to bring uniformity with the ultimate goal of the elimination of race-day medication will raise the integrity of our sport and, most importantly, will improve the safety of our athletes — both equine and human. It will not only help racing, it will invigorate the entire industry ecosystem from breeding to training and aftercare and will enable our sport to grow and compete on a global scale.

We strongly urge you to vote for the passage of the Horseracing Integrity Act.

Thank you.

Sincerely,

Belinda Stronach
Chairman and President

Ohio State Racing Commission

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John R. Kasich
Governor

Robert K. Schmitz
Chairman

June 19, 2018

Honorable Bob Latta
HOUSE OF REPRESENTATIVES
2448 Rayburn House Office Building
Washington, DC 20515

Dear Congressman Latta:

The Ohio State Racing Commission advocates for the betterment of the horse and horse racing on behalf of those involved in the sport in our great state of Ohio. I am the Chairman of the Ohio State Racing Commission. Gary Koch and Tom Winters are fellow commissioners, both of whom you know from your service in the Ohio General Assembly. The Racing Commission has serious concerns about H.R. 2651, the Horse Racing Integrity Act of 2017.

Ohio has four standardbred tracks and three thoroughbred tracks and sixty-seven county fairs that conduct pari-mutuel horse racing. Ohio hosts the $700,00 Little Brown Jug which is for three-year-old pacers where over 50,000 people descend upon the Delaware County Fair in late September for the Jug. Ohio also has more standardbred horses than any other state in the country. Ohio also is the home of the United States Trotting Association. Ohio also has the $500,00 Ohio Derby for three-year-old thoroughbreds.

If enacted, H.R. 2651 would be significantly burdensome to the horse industry. It would create a new, duplicative regulatory bureaucracy at the federal level, on top of an existing state regulatory structure that is operating in 34 state racing jurisdictions around the country. This system has functioned well for over 100 years. We in Ohio are proud of the job we have done to protect the horse and the integrity of racing. The Ohio State Racing Commission was established in 1933 and has over eighty-five years' experience in protecting the horse and the integrity of racing.

We are concerned that this new proposed bureaucracy would create a new structure of fees and taxes to cover some of the federal costs of the new regulatory structure. Those fees and taxes would come from horse owners and the industry at-large, possibly threatening the economic viability of many in our state. In Ohio, the Ohio State Racing Commission has budgeted over two million dollars for testing of race horses for fiscal year 2019. The Ohio Department of Agriculture analytical toxicology laboratory analyzes test samples for the Ohio State Racing Commission. This laboratory is a premier laboratory and successfully maintained the highest accreditations in North America for equine drug testing. Through the Ohio State University, the Ohio State Racing Commission funds $200,000 for equine research yearly.

In addition to the regulatory burdens noted above which would be created under H.R. 2651, the bill seeks to ban the drug Lasix, or Furosemide. This drug is used in horses to control or prevent

Exercise Induced Pulmonary Hemorrhages (EIPH). Horses often experience EIPHs during intense exercise, such as during races.

Lasix is recognized as an effective, preventative treatment for "bleeding" in the lungs of horses, which is the reason its use is recognized by the North American Association of Racetrack Veterinarians and the American Association of Equine Practitioners as safe and effective. These veterinarians believe that the use of Lasix is in the health and welfare interests of the animals.

According to a recent American Horse Council study, the horse racing industry contributes some $36 billion annually to the national economy and provides about 240,000 direct jobs. Any measure which will add further regulatory and cost burdens will only harm this state and local economies that depend on the industry. The Ohio horse racing industry directly and indirectly generates more than 16,000 jobs.

The Ohio State Racing Commission joins the Ohio Harness Horsemen Association, the United States Trotting Association, the Ohio Horsemen Benevolent & Protective Association (thoroughbred horsemen), and the Racing Commissioners International in urging you to oppose H.R. 2651.

If you or your staff have questions, please contact me.

Sincerely,

Robert Schmitz
Chairman, Ohio State Racing Commission

STONESTREET

June 22, 2018

Chairman Bob Latta
Ranking Member Jan Schakowsky
Committee on Energy & Commerce
Unites States House of Representatives
Washington, D.C. 20515.

Thank you for the opportunity to share my support for HR 2651, the Horseracing Integrity Act of 2017, with the Subcommittee on Digital Commerce and Consumer Protection.

My name is Barbara Banke. I operate a prominent international Thoroughbred racing and breeding operation named Stonestreet. I am also the chairman and proprietor of Jackson Family Wines, the nation's largest seller of premium wine. Thank you for allowing me to share why I believe this legislation is crucial to the future success of horse racing in America.

The federal regulatory framework afforded by this Bill allows the introduction of a single comprehensive scheme that will standardize medication use, testing and penalties across the United States of America. In an industry fueled by dollars from wagers placed across state lines the current variance in state regulations is no longer appropriate for the marketplace. Advocating for federal oversight is not a position I find myself in often, however I have seen it used to great effect in the wine industry in areas affecting consumer trust.

Stonestreet's horses have won graded stakes races, the highest level of competition, in eight different countries. The success of the American Thoroughbred in international competition has raised awareness of our product but the value of our exports will always be capped by our confusing, outdated and conflicting rules. For American horse racing to win in the long term, we must demonstrate that our industry acts with integrity and elevated standards of care to protect the health of our athletes, earn the trust of the public, remain the wagering platform of choice, and promote global bloodstock sales.

Under the Horseracing Integrity Act of 2017, we would achieve meaningful reform. Horse racing would benefit significantly and immediately if we standardize best practices across our industry and every horse race across the United States is run under a single set of rules. I have supported multiple efforts for the horse racing industry to bring uniformity on its own, but we have simply failed. It is time for Congress to act.

If H.R. 2651 were to be passed, we would finally operate under a uniform and effective anti-doping program that would allow our industry to market itself as a sport of authentic competition. In addition to independent application of uniform rules and penalties, HR 2651 requires out-of-competition testing and the use of accredited laboratories, pillars of any worthwhile anti-doping program. These standards should be the bare minimum for any sports industry that relies on wagering for its livelihood.

1000 ALEXANDER MOUNTAIN ROAD — GEYSERVILLE, CALIFORNIA 95441
(707) 431-3200 *phone* (707) 433-9725 *fax*

My family and I are vested, financially and emotionally, in the healthy future of this industry. For those who share our passion for horses and horse racing, H.R. 2651 is in the best interests of every human and equine stakeholder in the industry, and I encourage this committee to support it.

Again, thank you for this opportunity to provide comments.

Barbara R. Banke
Proprietor

Washington, D.C. 20515
Testimony by Matt F. Iuliano
Executive Vice President and Executive Director
The Jockey Club
House Energy & Commerce Subcommittee on Digital Commerce and Consumer Protection
HR 2651 Horseracing Integrity Act
Friday, June 22, 2018

Dear Chairman Latta, and Ranking Member Schakowsky,

This testimony is offered in support of the Horseracing Integrity Act of 2018, H.R. 2651, to more precisely quantify the reported progress of the current system of state-by-state advocacy of the National Uniform Medication Program (NUMP).

The NUMP was intended to unify all jurisdictions authorized to hold pari-mutuel horse racing under a single set of drug-testing rules, enforcement procedures and penalties. By eliminating the variation in rules among states, horses would no longer be subjected to different rules in different states, thus improving the health and safety of the athletes, equine and human, in addition to restoring the confidence of the consumer that the sport is fair and on a level playing field.

The NUMP is a regulatory program that has been advocated through volunteer efforts among the states and is composed of the following four principal components:

- Accreditation of testing laboratories to a code of standards established by the Racing Medication and Testing Consortium (RMTC, a voluntary organization of 23 industry groups designed to develop and promote uniform drug-testing standards)
- Adoption of the Association of Racing Commissioners International (RCI) Controlled Therapeutic Medication Schedule for Horses Version 4.0 (RCI is the umbrella organization of the official governing rule-making bodies for pari-mutuel racing)
- Adoption of the RCI Multiple Medication Violations schedule Version 8.3
- Administration of race-day medications by official veterinarian, the racing veterinarian, or his/her designee only

In December 2016, a fifth element was added when the RCI passed a model rule for out-of-competition testing that included a comprehensive list of substances prohibited from ever being present in samples collected from a horse.

Reports of progress toward adoption of the NUMP have been the subject of disagreement within the industry. The principal source of disagreement can be traced to the various versions of the NUMP that have been passed since it was initially introduced in April 2014. In its original form, the NUMP included regulatory thresholds and administrative guidance for 24 therapeutic medications. Since that time, the document details 23 revisions, including the addition of new therapeutic medications and changes to regulatory thresholds of existing therapeutic medications. The latest version of the NUMP includes 30 therapeutic medications.

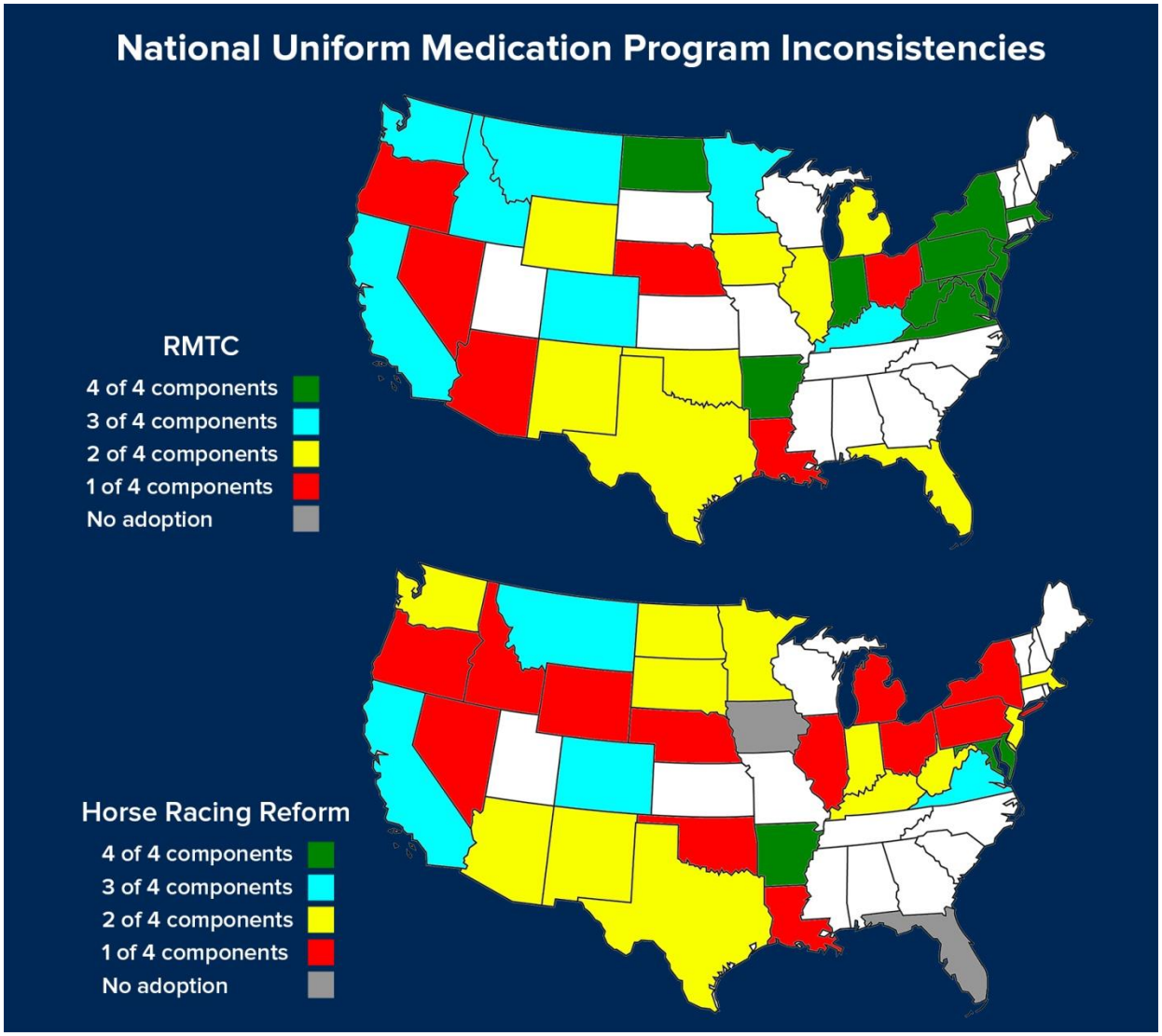
As more medications were added to the list of controlled therapeutic medications, states that had adopted earlier versions of the schedule found themselves out of synch with states adopting later versions. Changes

to regulatory thresholds for the medications further exacerbated the differences among the states, creating a highly fragmented regulatory system.

When the adoption of other components of the NUMP are factored in, such as the Multiple Medication Violation system, administration of race-day medications by official veterinarians or their designee, and the out-of-competition testing protocols, the variance among the states becomes alarming.

Simply put, the various rule-making procedures among the states makes synchronizing all to a single set of drug-testing rules and enforcement procedures an impossible objective.

The graphic below illustrates the stark differences in adoption of the NUMP as reported by the RMTC as compared to when more precise metrics are used.



RMTC reports that 11 of the 38 racing jurisdictions with laws permitting pari-mutuel horse racing have adopted all four elements of the NUMP. A more critical analysis reveals that only two states, Arkansas and Maryland, have adopted all four elements of the RCI NUMP Version 4.0 Controlled Therapeutic Medication Schedule and Version 8.3 of the relevant Model Rules.

Isolating on New York, arguably one of the more prominent racing jurisdictions in the world, material differences exist between drug regulations published by the New York Gaming Commission and those contained in the NUMP, including:

- Cetirizine (antihistamine) is included in the NUMP but is not included in New York
- Withdrawal guidelines differ for 14 of the 30 controlled therapeutic medications
- Regulatory thresholds differ for 8 of the 30 controlled therapeutic medications

When other states are subjected to the same level of scrutiny, similar discrepancies are noted because of either older versions of the NUMP being adopted, outdated regulatory thresholds never being revised, or states just simply failing to keep their rules updated.

The Horseracing Integrity Act, H.R. 2651, will correct these deficiencies in the existing fragmented state-by-state regulatory model by ensuring all racing jurisdictions operate under uniform rules and enforcement procedures.

Please contact me directly with any questions or further information.

ATTACHMENT #1

By training I am a veterinarian, a pharmacologist and a toxicologist and I have been performing, publishing and evaluating research on Lasix in horses since 1975 (Attachment #1 Gabel et al, 1977). I draw attention to this long since published paper because the basic horsemen's opinions and veterinary practitioner evaluations presented in this now long ago 1977 paper have, over the years, been supported by evolving science and published scientific research, as I will be pleased to point out as I present this analysis and opinions.

The story of Lasix and racing horses is a 50 year story of field, i.e., horsemen's and veterinary practitioner evaluations and assessments of the beneficial effects of Lasix in the racing horse. These field evaluations of the beneficial effects of Lasix are set forth in the Gabel 1977 paper and now, forty years later, in 2018, forty years of science has basically confirmed and explained these beneficial effects as our understanding of EIPH, Exercise Induced Pulmonary Hemorrhage has developed. We note that the term Exercise Induced Pulmonary Hemorrhage did not even exist in 1977; as such, the Lasix and EIPH story is a classic story of half a century of scientific research confirming and explaining long standing and well established field observations and clinical experience.

The take home message of this analysis and opinions is that Lasix PROTECTS the lungs of the racing horse, and in so doing PROTECTS the horse and also the jockey. I will begin my presentation with a bullet point summary, I will then present specific sections with literature references supporting each bullet point and then close with a restatement of my bullet points and the take home message that Lasix PROTECTS the lungs of the racing horse and in so doing PROTECTS the horse and also the jockey.

Furosemide in Horses: A Review

Albert A. Gabel, D.V.M., M.S.
Thomas Tobin, D.V.M., Ph.D.
Richard S. Ray, D.V.M., M.S., Ph.D.
George A. Maylin, D.V.M., M.S., Ph.D.

From the Department of Veterinary Clinical Sciences, The Ohio State University, 1935 Coffey Road, Columbus, Ohio 43210 (Gabel, Ray); Department of Veterinary Science, University of Kentucky, Lexington, Kentucky 40508 (Tobin); and Equine Drug Testing and Research, Cornell University, Ithaca, New York 14853 (Maylin).

With additional input by: Gary P. Carlson, University of California; Harold E. Garner, University of Missouri; Jerry R. Gillespie, University of California; Jerry H. Johnson, University of Missouri; Dennis W. Milne, Ohio State University; William W. Muir, Ohio State University, and David Snow, University of Glasgow, and other members of the National Association of State Racing Commissioners Veterinary-Chemist Advisory Committee: Tom Finley, A.V. Villatico, Robert E. Vessiny, Dr. Francis Ozog, Dr. Robert Schoenfeld, Dr. Joe Solomon, Dr. Marvin Beeman, Dr. Joe O'Dea, Dr. Gene Bierhaus, and General Wayne Kester.

1. Injectable furosemide[a] is a potent, effective, safe diuretic which at the usual dose has its peak diuretic effect in about 20 minutes in horses, at which time it increases urine output up to 40-fold for a short time. Most of its diuretic effect is within the first 2 hours after injection.

2. During the first 2 hours, it decreases the pressure in the right side of the heart, decreases left atrial pressure, decreases cardiac output, and concurrently it increases peripheral (systemic) blood vessel resistance and heart rate.

3. It reduces edema in the lungs and airway of horses with certain respiratory diseases by 1 or more mechanisms, perhaps by lowering the pressure of the left atrium and/or increasing capacitance (ability to accept blood) of the vessels of the lungs.

4. The mechanism by which furosemide helps prevent epistaxis may result from the beneficial effect outlined in (3) above since most horses bleed from the lungs. Lasix[a] also appears to affect the platelets of horses during exercise.

5. The blood (plasma) level of furosemide declines rapidly. It can be detected in the urine by screening methods for the first 24 hours and by more sophisticated methods for up to 48 hours after injection.

6. Furosemide does not affect the blood levels of other drugs.

7. Furosemide has little effect on the concentration of certain drugs, such as procaine, in the urine. However, during its peak diuretic effect, within 2 hours after injection, furosemide causes up to 40-fold dilution of certain other drugs such as phenylbutazone. However, furosemide administered more than 4 hours before race time does not significantly reduce the ability to detect those drugs studied to date.

[a] Lasix®, National Laboratories, Inc., Somerville, NJ 08876

8. There is no evidence that furosemide directly or indirectly affects the behavior of the horse.

9. Furosemide cannot make horses race faster or perform better than their innate ability, but in many cases it restores normal performance of horses which bleed.

Introduction

There is no scientific evidence that furosemide will increase the speed or improve performance of a horse beyond its innate capability. In a recently completed double-blind study there was no significant difference in the times of time-trial miles of clinically normal Standardbreds after intravenous furosemide injection compared to saline injection, 2 hours and 10 minutes before the trials.[18] A mechanical prompter was used for uniform psychological stimulation. However, clinical observations indicate that the drug may help a horse to more nearly reach his racing capacity if he is a bleeder, or has certain other conditions, including perhaps early obstructive lung disease "heaves" or diseases in which pulmonary edema occurs, even transiently, during exercise.[12,31]

Mechanism of Action (Diuretic Effect)

The primary effect and use of injectable furosemide has been as a potent diuretic. It has a high degree of efficacy, low-inherent toxicity, and a high therapeutic index in horses.[11] Furosemide produces the increase in urine by inhibiting active electrolyte (inorganic ions such as chloride) transport in that part of the kidney known as the diluting segment of the ascending limb of the loop of Henle.[4,8,21,28] Furosemide produces increased losses of chloride, sodium, other electrolytes, and water.[11] There is little potassium loss in horses.[11] In normal horses, no dehydration or electrolyte imbalance occurs if they have access to water and salt before and after the injection.[22] Its onset of action is rapid.[11,13,20,21,29] The diuretic effect of furosemide peaks within 15 to 30 minutes, when there is up to a 40-fold increase in urine production for a short time.[21,30] Dosed at 1 mg/kg body weight, most of its diuretic effect occurs within 2 hours.[21,30] Doses between 0.01 and 1.0 mg/kg (the dose recommended by the manufacturer) produce a graded increase in urine volume and decrease in its specific gravity.[11,30] The average urine production during a 2-hour period after injection of the usual dose of furosemide is 3 to 12 liters and is dependent on the horse's state of hydration when the drug is given.[20,21,30] Only 1 liter of urine is produced during an average 2-hour control period.[30] Due to the loss of fluid during the period of increased urine production, horses which are dehydrated before the injection make less urine during several hours which follow; therefore, it is sometimes difficult to get a urine sample during this period.[11] Race Commission veterinarians do not find this to be a significant problem.[18]

Furosemide is used by many veterinarians in the treatment of acute tieing-up and azoturia to increase urinary output, since high concentrations of the pigment from damaged muscles can cause kidney shutdown.

Cardiovascular Effects

In resting horses, in addition to increased urine production, the following changes were found to be statistically significant ($P<0.05$)[20] from 15 minutes to 110 minutes after intravenous injection of the usual dose (1 mg/kg) of furosemide:

1. Decreased blood pressure in the right side of the heart, pulmonary artery, and left atrium. This may be due to hypovolemia and/or increasing capacitance (ability to accept blood) of the vessels of the lungs.
2. Decreased venous return and therefore decreased cardiac output.
3. No significant change in systemic arterial blood pressure, although there was a significant increase in peripheral (systemic) vascular resistance and reflex increase in heart rate.
4. Hemoconcentration. Packed cell volume and total protein percentage in the blood increased.

In a recent double-blind study intravenous furosemide injection was compared to saline injection under simulated race conditions using Standardbreds.[18] Hemodynamic and biochemical data were recorded before and during the first "warm-up miles" 15 minutes after injection and before, during, and after the third (time-trial) mile 2 hours and 10 minutes after injection. After furosemide, compared to saline, pulmonary artery pressure was lower during the first warm-up mile, 15 minutes after injection, but not during the time-trial mile 2 hours and 10 minutes after injection. There was no difference in cardiac output immediately after and 10 minutes after each of the miles, nor in the pulse rate during or after each of the miles.

These experiments suggest that, if given during the 2-hour period prior to a race, furosemide might have a negative effect on performance because of a decreased cardiac output.

Respiratory Effects

A clinically important action of furosemide in both man and small domestic animals is its rapid action against pulmonary edema. Since this effect also occurs in patients in which this drug does not produce diuresis, and in patients which are kept hydrated, the volume change is not totally due to diuresis.[7,45]

Recent observations suggest that the pulmonary effects of furosemide may be mediated by prostaglandins released from the kidneys.[7] Another theory is that there may be a tendency for lung edema to develop when mean pulmonary artery pressure exceeds 40 mm Hg by very much.[42,43] Aver-

age mean pulmonary artery pressures of approximately 70 mm Hg have been measured in Standardbreds during time trials.[17] Edema (which may occur transiently) has a negative effect on breathing reflexly through the J-fibers of the vagus nerve.[18] The more severe the exercise, the greater the tendency for pulmonary edema to occur. This may explain why furosemide is used more in Thoroughbreds, which work harder, than in Standardbreds.[21]

The experience of race track veterinarians indicates that the drug appears to have an effect of producing a more nearly optimal performance in Thoroughbreds which have heavy or labored breathing while training or racing, in those which have nasal exudate after racing, and in some which become excited in the paddock.[21]

It is thought that horses with edema and narrowing of the upper airway, especially the pharynx by such conditions as follicular pharyngitis, may race more normally following the use of furosemide, because tissue fluid is removed, enlarging the airway toward normal size.[14,23]

Epistaxis

It is thought that in most cases the blood comes from rupture of small vessels in the lungs, and sometimes the upper airway, when horses have epistaxis following work.[8,14] The experience of many veterinarians shows that furosemide injection approximately 4 hours before exercise is effective in preventing epistaxis in a high percentage of Thoroughbred and Standardbred horses which bleed during or after racing or hard workouts.[23]

In a double-blind study under simulated racing conditions, Standardbred horses were injected with 1 mg/kg of furosemide or saline 15 minutes before the first warm-up mile.[13] Hemostatic function including the blood clotting mechanism and platelets were evaluated prior to injection, immediately following the first warm-up mile, and after the time-trial mile 2 hours and 10 minutes after the injection. There was a slight increase in the retention of platelets in glass bead columns, but this platelet function change could not be substantiated in a follow-up study using furosemide injections in nonexercised horses. No significant changes were found in screening tests of the blood clotting mechanism.

Furosemide may be effective in horses which bleed by reducing the blood pressure of the left atrium and increasing capacitance of the lung vessels.[18]

Blood (Plasma) and Urine Levels—Detection

Furosemide is usually administered to horses by intravenous (IV) or intramuscular (IM) injection at doses of 1 mg/kg or somewhat less. After IV administration of 1 mg/kg, its plasma levels decline rapidly, from about 10 μg/ml (parts per million) initially to 500 ng/ml (parts per billion) at 20 minutes.[20] Thereafter, plasma levels fall more slowly with a half-life of about 30 minutes, to less than 20 ng/ml at 4 hours.[20] Urinary levels of the drug start at about 30 μg/ml and fall rapidly over the first 12 hours to about 500 ng/ml.[20] This is about the detection limit of the thin layer techniques used in routine drug testing screens.[20] However, using gas chromatographic (G.C.) methods, urinary levels of the drug can still be detected up to 48 hours postdosing.[20]

Effects of Furosemide on the Concentration of Other Drugs in Equine Blood and Urine

In no instance is furosemide known to significantly reduce the blood or plasma level of any drug.[24,30]

Treatment with furosemide does not appear to significantly affect the concentration of basic lipid soluble drugs in equine urine.[30] Thus, furosemide has no effect on either plasma or urinary concentrations of procaine in horses treated with 10 mg/kg of procaine HCl intramuscularly.[30] Similar results were observed with methylphenidate[30] In both cases, urinary output of the drug was increased by the greatly increased urine volume, and the concentration did not decrease enough to cause trouble in detection of the drugs.[30] The same effect probably occurs with amphetamine.[30] Glucuronide and sulfate conjugates of narcotics can be diluted up to several fold.[32]

In contrast, however, urinary concentrations of phenylbutazone are reduced up to 40-fold at peak diuresis.[30] This concentration change can greatly reduce the reliability of routine UV screening for phenylbutazone.[25] Concentrations of phenobarbital also are probably affected, though not quite so severely.[9]

In doses usually used, furosemide can greatly reduce the urinary concentrations of some drugs, but only during the first 2 hours and for a much lesser extent up to 4 hours after it is administered; in other drugs it has little effect. In particular, use of furosemide can interfere with routine screening for phenylbutazone during this period of increased urine production (2 to 4 hours). For this reason, blood testing for phenylbutazone should be recommended where medication rules permit the use of furosemide within 4 hours of racing.

Ritalin®, Ciba Pharmaceutical Co., 556 Morris Ave., Summit, NJ 07901.

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ATTACHMENT #2

1/ THE LUNG IS BY NECESSITY A DELICATE TISSUE PRONE TO STRESS FAILURE OF PULMONARY CAPILLARIES:

Lung capillaries must be strong enough to not rupture under the stress of racing, but delicate enough to allow the rapid transfer of oxygen to the red blood cells. Under the stress of racing some incidence of stress failure (rupture) of pulmonary capillaries is inevitable, as described by West el al, 1993, in their paper entitled "***Stress failure of pulmonary capillaries*** *in racehorses with* ***Exercise-Induced Pulmonary Hemorrhage***", Attachment #2 [Our caps, bolding and underlining].

Stress failure of pulmonary capillaries in racehorses with exercise-induced pulmonary hemorrhage

JOHN B. WEST, ODILE MATHIEU-COSTELLO, JAMES H. JONES, ERIC K. BIRKS, RICHARD B. LOGEMANN, JOHN R. PASCOE, AND WALTER S. TYLER
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WEST, JOHN B., ODILE MATHIEU-COSTELLO, JAMES H. JONES, ERIC K. BIRKS, RICHARD B. LOGEMANN, JOHN R. PASCOE, AND WALTER S. TYLER. *Stress failure of pulmonary capillaries in racehorses with exercise-induced pulmonary hemorrhage.* J. Appl. Physiol. 75(3): 1097–1109, 1993.—Bleeding into the lungs in thoroughbreds is extremely common; there is evidence that it occurs in essentially all horses in training. However, the mechanism is unknown. We tested the hypothesis that exercise-induced pulmonary hemorrhage (EIPH) is caused by stress failure of pulmonary capillaries. Three thoroughbreds with known EIPH were galloped on a treadmill, and after the horses were killed with intravenous barbiturate the lungs were removed, inflated, and fixed for electron microscopy. Ultrastructural studies showed evidence of stress failure of pulmonary capillaries, including disruptions of the capillary endothelial and alveolar epithelial layers, extensive collections of red blood cells in the alveolar wall interstitium, proteinaceous fluid and red blood cells in the alveolar spaces, interstitial edema, and fluid-filled protrusions of the endothelium into the capillary lumen. The appearances were consistent with the ultrastructural changes we have previously described in rabbit lungs at high capillary transmural pressures. Actual breaks in the endothelium and epithelium were rather difficult to find, and they were frequently associated with platelets and leukocytes that appeared to be plugging the breaks. The paucity of breaks was ascribed to their reversibility when the pressure was lowered and to the fact that 60–70 min elapsed between the gallop and the beginning of lung fixation. Capillary wall stress was calculated from pulmonary vascular pressures measured in a companion study (Jones et al. *FASEB J.* 6: A2020, 1992) and from measurements of the thickness of the blood-gas barrier and the radius of curvature of the capillaries. The value was as high as 8×10^5 dyn/cm^2 (8×10^4 N/m^2), which exceeds the breaking stress of most soft tissues. We conclude that stress failure of pulmonary capillaries is the mechanism of EIPH.

thoroughbred; lung bleeding; capillary pressure; capillary wall stress; pulmonary vascular pressures; overinflation of lung; furosemide

BLEEDING FROM THE NOSE (epistaxis) in horses has been recognized for many years (23, 35). About 20 yr ago it was shown that in galloping thoroughbreds the bleeding came from the peripheral parts of the lungs (4), and at that time the incidence of bleeding in racehorses was thought to be up to 3%. However, with the introduction of the flexible bronchoscope, Pascoe et al. (31) showed that bleeding from the lung was extremely common, and the term exercise-induced pulmonary hemorrhage (EIPH) was introduced. More recent studies based on the presence of hemosiderin-laden macrophages in tracheal washings indicate that essentially all thoroughbreds in training bleed into their lungs (46). At some racetracks horses that bleed are prohibited from further racing, and EIPH is one of the most serious veterinary problems facing the racing industry today. In addition, the mechanism is of great physiological interest.

The pathogenesis of EIPH is unknown. Possible causes that have been suggested from time to time include chronic lung disease, parasite infestation, bloodborne pathogens, coagulation defects, asphyxia, partial airway obstruction, and uneven mechanical stresses in the lung parenchyma during strenuous exercise (for a review see Ref. 37). In an extensive clinical and autopsy study of the lungs of 26 thoroughbreds with EIPH, O'Callaghan and co-workers (27–29) found multifocal lesions consisting of hemosiderophages, bronchiolitis, and increased connective tissue. They suggested that the etiology was a low-grade bronchiolitis, possibly of viral origin, and postulated that the bleeding came from neovascularization of the bronchial circulation.

Recently two new findings have suggested another basis for EIPH. First, West et al. (45) and Tsukimoto et al. (39) showed that in anesthetized rabbits raising the pulmonary capillary pressure to ≥40 mmHg caused ultrastructural damage to the alveolar wall, including disruption of the capillary endothelium, the alveolar epithelium, or sometimes all the layers. Because the calculated stresses in the capillary walls are extremely high under these conditions, the phenomenon has been called "stress failure." The second finding was extremely high pulmonary vascular pressures measured by catheterization in racehorses galloping on a treadmill. For example, Jones et al. (18) reported mean pulmonary arterial and left atrial pressures of 120 and 70 mmHg, respectively, which indicate that the capillary pressures must have been extremely high. These findings taken together immediately suggest that EIPH could be caused by stress failure of pulmonary capillaries.

In this paper we describe studies in which thoroughbreds with known EIPH were galloped on a treadmill, after which their lungs were prepared for electron microscopy by intravascular fixation with buffered glu-

taraldehyde. We show that the ultrastructural appearances of the lungs are consistent with stress failure of pulmonary capillaries. In addition, we show that the calculated wall stresses of the capillaries are extremely high and conclude that stress failure of pulmonary capillaries is the mechanism of EIPH.

METHODS

Experimental Procedures

Studies were carried out on three thoroughbred horses. First we did a pilot experiment to determine whether electron microscopy of the lung in a horse with known EIPH showed abnormal pulmonary capillaries. Because this was the case, two further horses (experimental *animals 1* and *2*) were specifically studied for this project. The protocols were approved by the Animal Subjects Committee, University of California, Davis, where the horses were run on the treadmill and the lungs were prepared for electron microscopy.

Pilot study. The thoroughbred for the pilot study was a grey gelding aged 6 yr (wt 440 kg). It had been a control animal in a study of the effects of ozone exposure on lung morphology but had not been exposed to ozone. The animal was killed by an overdose of intravenous barbiturate 1 h after galloping on the treadmill at a rate of 14–16 m/s. The lung was then removed, floated on water in a stock tank, and fixed by instilling Karnovsky's solution into the airways at an airway pressure of 30 cmH_2O.

Experiment 1. The thoroughbred for *experiment 1* (*animal 1*) was a chestnut gelding aged 5 yr (wt 430 kg). It had been donated to the University of California, Davis, because it had developed EIPH. On the day of the study, the horse was first walked on the treadmill for 5 min, which was followed by 9 min of trotting, 3 min of cantering, and finally 3 min of galloping at 14 m/s. The heart rate (HR) measured by electocardiogram rose to 218/min. The animal was then removed from the treadmill and walked across to the preparation room ~50 m away. Heparin (20,000 U) was given intravenously, and 10 min later the horse was killed with a large dose of intravenous barbiturate via a jugular vein. The carotid artery was severed, the left forequarter was removed, and the left thoracic cage was cut away. The lungs and heart were then removed, and photographs were taken of the lung to identify the location of the tissue samples that were subsequently taken for electron microscopy. Cannulas were placed in the trachea, pulmonary artery, and left atrium. The lung was then floated on water in a stock tank, and it was inflated three times to a pressure of 25 cmH_2O until all areas were aerated. The lung was then perfused with a saline-dextran mixture (11.06 g NaCl/l, 350 mosM; 3% T-70 dextran) for 10 min to wash out the blood. The alveolar pressure was held at 10 cmH_2O during the washout, which was begun 40 min after the animal was killed. Finally, buffered glutaraldehyde (phosphate-buffered 2.5% glutaraldehyde with 3% T-70 dextran, total osmolarity 500 mosM, pH adjusted to 7.4) was perfused at a pulmonary arterial pressure (Ppa) of 45 cmH_2O, a pulmonary venous pressure of 10 cmH_2O, and an alveolar pressure of 10 cmH_2O. The osmolarity of the fixative was based on the work of Bachofen et al. (1), who showed that this procedure resulted in excellent ultrastructural preservation. The pulmonary venous pressure was then raised to 45 cmH_2O to be the same as the Ppa, and the lung was held at these pressures overnight. Fixation of the lung began 67 min after the end of the gallop and 49 min after death.

Experiment 2. The thoroughbred for *experiment 2* (*animal 2*) was a 3-yr-old filly with known EIPH (wt 545 kg). A catheter was inserted into a jugular vein and was placed in the pulmonary artery by pressure monitoring. Mean Ppa at rest was 39 mmHg referred to the level of the right atrium. After walking on the treadmill for a few minutes, the horse trotted at 4 m/s with the results that the mean Ppa was 60 mmHg and the HR was 102/min. This was followed by 3 min of cantering at 7 m/s, giving a mean Ppa of 90 mmHg and a HR of 171/min. Finally, the horse galloped for 3 min at 13.5 m/s, giving a mean Ppa of 138 mmHg and a HR of 210/min. After the gallop the horse was walked briefly on the treadmill to allow her to cool down, and she was then removed. A twitch was put on the nose, and a bronchoscope was passed through the left nostril. A trickle of fresh blood was seen in the trachea ~100 cm from the nares, that is, ~30 cm above the carina. Heparin (30,000 U) was then given intravenously, and 15 min later the animal was killed with a large dose of intravenous barbiturate. The lungs were removed, cannulated, inflated, and perfused as before except that normal saline rather than a saline-dextran mixture was used to flush out the blood. Perfusion fixation was started using a Ppa of 50 cmH_2O, a pulmonary venous pressure of 40 cmH_2O, and an alveolar pressure of 15 cmH_2O. Fixation of the lung began 50 min after death and 78 min after the end of the gallop. After 15 min of perfusion fixation, the lung was left to fix overnight with pulmonary arterial and venous pressures of 46 cmH_2O and an alveolar pressure of 15 cmH_2O.

Tissue Sampling

The lungs were removed from the stock tank, and the heart, great vessels, and mediastinal tissues were dissected out. Each lung (right and left) was then photographed and cut into 2- to 3-cm transverse slices taken perpendicular to the cephalocaudal axis. Up to 30–35 slices were made from each of the right and left lungs. The slices were inspected for abnormal dark-stained areas and were photographed. Samples were systematically taken from these lesions, from macroscopically clear areas, and from regions intermediate between the two areas. The precise sites of sampling were carefully documented by taking pictures of the slices with the cut samples labeled and left in place. Sixteen to 60 samples (~2 × 0.5 × 0.2 cm) were taken from each horse.

Electron Microscopy

The samples were stored in glutaraldehyde in a refrigerator at 4°C for 10–60 days. They were cut into small blocks, rinsed overnight in 0.1 M phosphate buffer adjusted to 350 mosM with NaCl (pH 7.4), and postfixed for 2 h in a solution of osmium tetroxide in 0.125 M sodium cacodylate buffer adjusted to 350 mosM with NaCl (total osmolarity 400 mosM, pH 7.4). The samples were then dehydrated in increasing concentrations (70–100%) of ethanol, rinsed in propylene oxide, and embedded in Ar-

aldite. Sections (1 μm) were cut with an LKB Ultratome III. They were stained with 0.1% toluidine blue aqueous solution and were examined by light microscopy. Ultrathin sections (50–70 nm) were contrasted with uranyl acetate and bismuth oxinitrate (34) and were examined with a Zeiss 10 electron microscope.

Measurement of Thickness of the Blood-Gas Barrier

Two samples each from *animals 1* and *2* were used. The samples were selected from areas of lung that appeared normal at both the gross and light microscopic levels. From each sample, micrographs were taken by systematic random sampling on five ultrathin sections (1 section from each of 5 blocks). We analyzed 60 micrographs (12 micrographs/block) from each sample. Micrographs of a carbon grating replica (E. F. Fullam, Schenectady, NY), taken with each film, were used for calibration.

A Videometric 150 image analyzer (American Innovision) was used to estimate the thickness of the blood-gas barrier after electronic positive reversal of the 70-mm negative films. Because the resolution of the video images was not always sufficient for the identification of structures, a print of each micrograph was also made and examined during the measurements. The final magnification was ×11,000. Measurements of the blood-gas barrier thickness (profile) were made at right angles to the barrier at systematic random sites relative to the capillaries. These sites were determined by the intersection of the capillary wall with fixed test lines generated by the image analyzer (up to 5 intersections/micrograph). A total of 667 measurements of blood-gas barrier thickness were made.

Measurement of Radius of Pulmonary Capillaries

Capillary radius was measured on selected profiles on micrographs printed at a magnification of ×11,000. The following criteria were used to select capillaries: *1*) the entire capillary was contained within the frame of the micrograph, 2) the endothelial lining was not disrupted, and *3*) the major-to-minor axis ratio was <3. From the total set of 240 micrographs sampled, a total of 47 capillaries met these criteria. Capillary diameter was taken as the average between the major and minor axes.

Calculation of Capillary Wall Stress

The circumferential or hoop stress in the wall of a capillary was calculated using the Laplace relationship

$$S = \frac{Pr}{t}$$

where S is stress (dyn/cm^2), P is pressure (dyn/cm^2), r is radius of the capillary (μm), and t is thickness of the capillary wall (μm) (45). The stress in newtons per meter squared (SI units) is the stress in dynes per centimeter squared divided by 10.

RESULTS

Location of Hemorrhagic Areas and Light Microscopy

At removal, the surface of the lung from *animal 1* showed little macroscopical abnormality, whereas extensive abnormal appearances were seen in the lung from *animal 2*. These regions appeared as light to dark brown stained areas. In both animals the abnormal areas were most prevalent in the dorsocaudal regions of the lungs, as has been described by other investigators (27, 37). In *animal 1* the lesions were mainly confined to this area, but in *animal 2* the damage had progressed to most parts of the lungs. It extended to the anterior portions of the dorsal crest of each lobe, and brown areas were also found in the caudal diaphragmatic surface of the lobes. Although many of those areas may represent earlier lesion sites, solid bright areas of fresh hemorrhage were also seen in the proximity of many of those regions. After slicing, both perfusion-fixed lungs (*animals 1* and *2*) showed dark parenchymal areas of various size (<0.05 to >4 cm^2) in the dorsal region to the midregion along the dorsoventral axis. In *animal 1* these areas were particularly evident in the left lobe where they were found in the most caudal portions of the lung, i.e., in *slices 1* (far caudal) to *6*. They were relatively small, representing less than approximately one-fifth of the total sectional area of each slice. In *animal 2* the distribution of dark areas was considerably more extensive, especially in the most caudal slices (*slices 1–10*) of the left lobe where they represented as much as approximately three-fourths of a slice sectional area. Dispersed smaller dark areas were also seen in the dorsal aspect of more anterior portions of the lung (*slices 28–29*). In other words, macroscopic evidence of lesion was absent only in the few most anterior slices (*slices 4–6*) of the lung in this animal.

A trickle of fresh blood was seen by bronchoscopy ~100 cm from the nares of the same animal after galloping (see METHODS). Macroscopic examination of the sliced lung revealed the presence of streams of blood in the ventral aspect of airways as far caudally as *slices 15* (left lobe) and *20* (right lobe).

Light microscopy examination of 1-μm-thick sections showed extensive accumulation of red blood cells (RBCs) in the interstitium immediately adjacent to the alveoli and in the alveolar spaces at the abnormal sites.

Electron Microscopy

Pilot study. In the pilot study, RBCs were seen in the alveolar wall interstitium (Fig. 1*A*). There was interstitial edema (Fig. 1*B*) and also fluid-filled protrusions into the capillaries (Fig. 1*C*).

Experimental animal 1. In lung tissue from *animal 1*, the peripheral regions of abnormal areas showed RBCs and granular material (edema) in the alveolar spaces as well as in the interstitium. The abnormal areas showed large numbers of RBCs in the alveolar interstitium (Fig. 2*A*) and alveolar spaces (Fig. 2*B*). The epithelial layer lining alveoli with granular material and RBCs was swollen (Fig. 2, *A* and *B*), and interstitial edema was also seen.

Experimental animal 2. In lung tissue from *animal 2* as from *animal 1*, large numbers of RBCs in the alveolar wall interstitium (Fig. 3*A*), interstitial edema, and RBCs and granular edema material in the alveolar spaces were seen. Additional findings were the disruption of the alveolar epithelial layer with intact basement membrane (Fig. 3*B*). Endothelial breaks were also seen, with intact

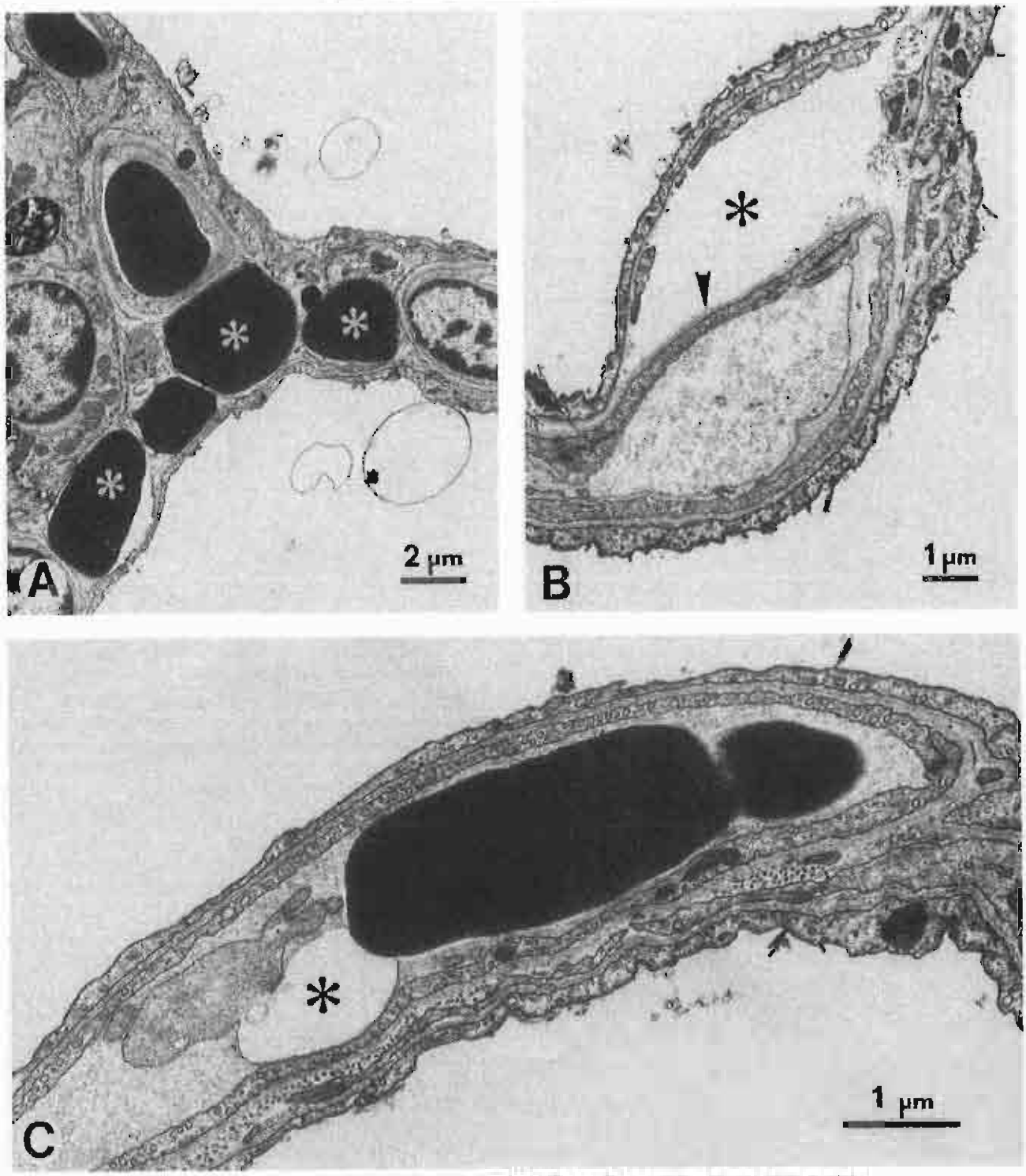

FIG. 1. Electron micrographs showing ultrastructure of alveolar region of lung of thoroughbred horse (pilot study) fixed by airway instillation after maximal exercise. *A*: red blood cells (*) in interstitium. *B*: edema fluid accumulation (*) lifted epithelial layer and interstitial cell cytoplasmic processes from capillary basement membrane (arrowhead). *C*: fluid-filled protrusion (*) into capillary.

basement membrane and several platelets and white blood cell cytoplasmic extensions adjacent to the exposed basement membrane (Figs. 4 and 5). Sections of pseudopods and filipods in close association with the endothelium lining and several cytoplasmic extensions suggested the presence of pulmonary intravascular macrophages (48) in the tissue (Figs. 3–5). Accumulation of electron-dense particles was found in edematous areas (Fig. 6). Measurement of the largest profiles in Fig. 6 suggested a diameter of ≃80 nm for these particles.

Frequency of Abnormal Findings

It was not possible to carry out a rigorous quantitative analysis of the frequency of abnormalities because of the spotty nature of the lesions. However, a systematic ran-

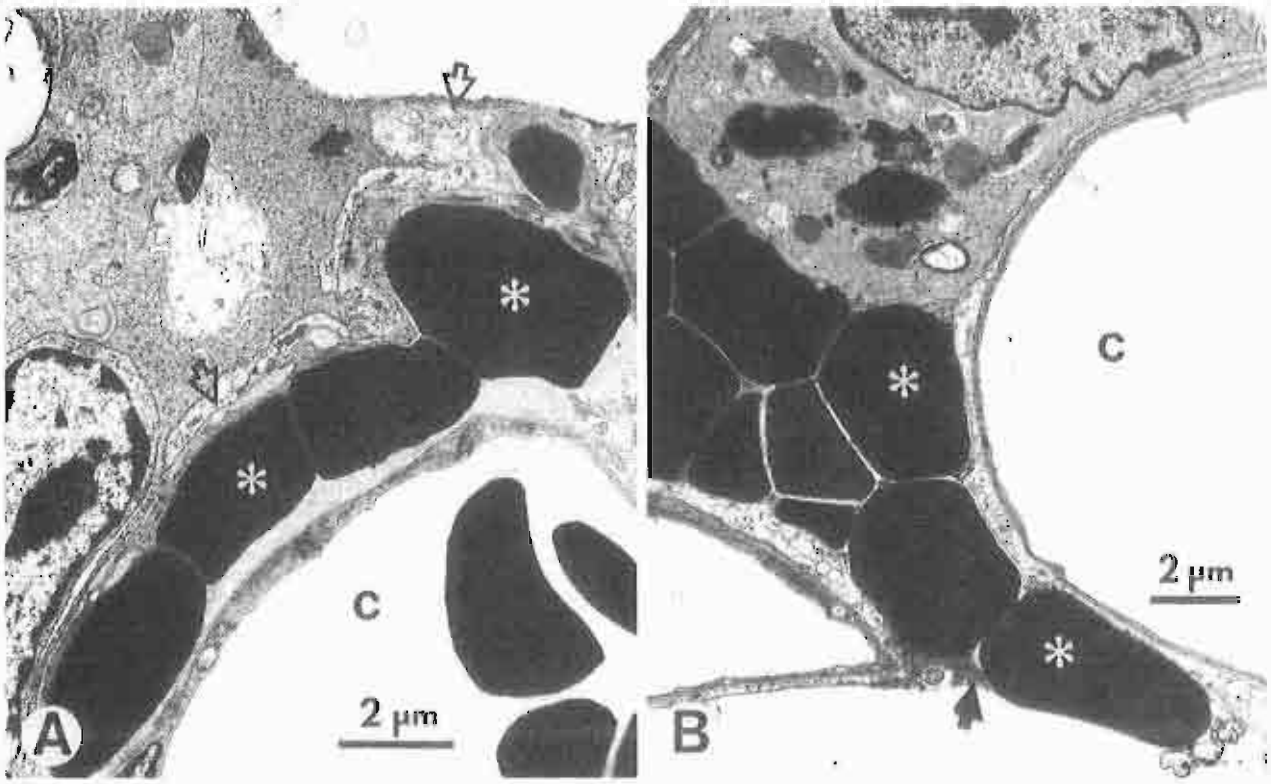

FIG. 2. Electron micrographs showing ultrastructure in abnormal area of lung parenchyma in thoroughbred horse (*animal 1*) perfusion fixed after maximal exercise. *A*: red blood cells (*) in interstitium and accumulation of granular material (solid arrow) in alveolar space. *B*: red blood cells (*) and granular material (solid arrow) in alveolar space. Note swelling of epithelial lining (open arrows) in both micrographs. *c*, capillary.

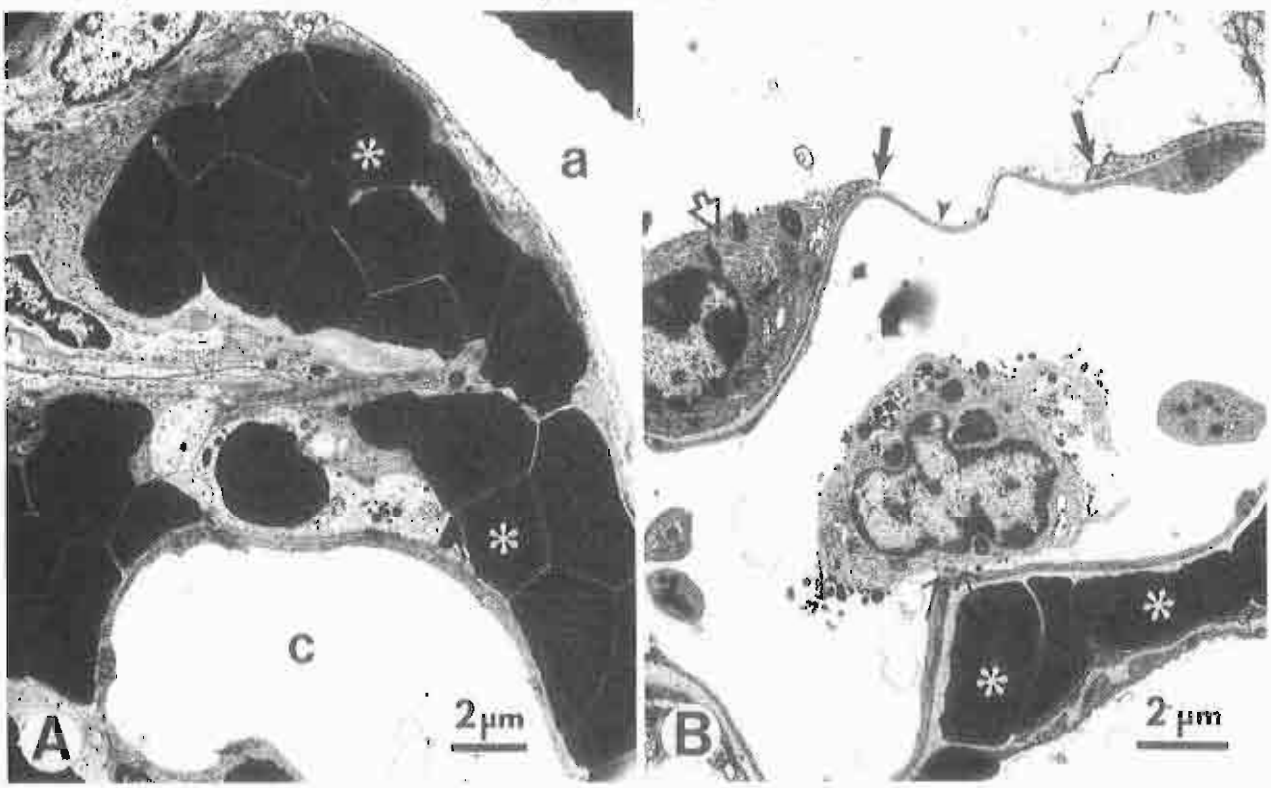

FIG. 3. Electron micrographs showing ultrastructure of lung parenchyma in thoroughbred horse (*animal 2*) perfusion fixed after maximal exercise. *A*: extensive accumulation of red blood cells (*) in interstitium. *B*: discontinuity of epithelium (solid arrows) with intact basement membrane (arrowhead). Note epithelial II cell (open arrow) in alveolar space and red blood cells (*) in interstitium. Large macrophage in capillary lumen shows close apposition of pseudopods to endothelium (thin arrows). *a*, alveolar space.

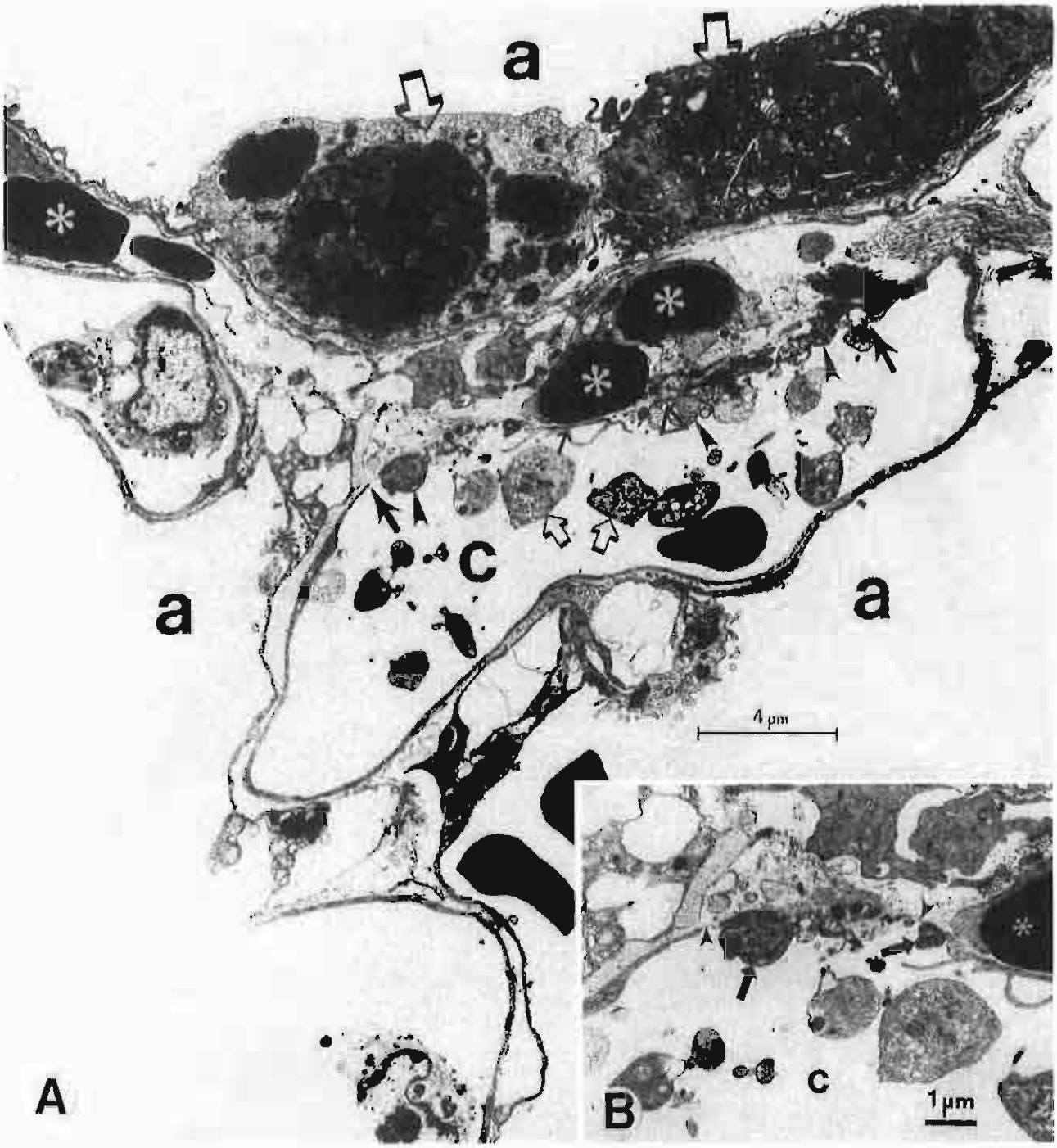

FIG. 4. Electron micrographs showing ultrastructure of lung parenchyma in thoroughbred horse (*animal 2*) perfusion fixed after maximal exercise. *A*: montage showing disruptions of capillary wall (solid arrows) and fragment of endothelium (open arrowhead). Note red blood cells (*) in interstitium, accumulation of platelets (arrowheads) in capillary, and macrophages (large open arrows) on epithelial lining. Filipods in close apposition to endothelium (thin arrow) and some cytoplasmic extensions (small open arrows) suggest presence of pulmonary intravascular macrophages. *B*: enlargement of portion of endothelial disruption from *A* showing platelets (arrows) adhering to exposed basement membrane (arrowheads) and red blood cell (*) in interstitium.

dom examination by light microscopy of 1-μm-thick sections documented the approximate frequency of those abnormalities. Twenty fields were chosen by systematic random sampling in each of two sections of a lesion site and of an intermediate area in *animal 1*. RBCs were found in alveoli (all 20 fields) and in the interstitium (14 of 20 fields) at the lesion site. In the intermediate area where there was an uneven distribution of hemorrhage, RBCs were also found in alveoli (16 of 20 fields) and interstitium (8 of 20 fields). Each field was examined at a magnification of ×400 and included an average of 4.8 ± 0.4 (SE) alveoli and 28 ± 2 capillary sections. A similar sampling in two randomly chosen sections of peripheral areas of lesions in *animal 2* revealed RBCs in alveoli (all 40 fields) and in interstitium in most fields (31 of 40 fields).

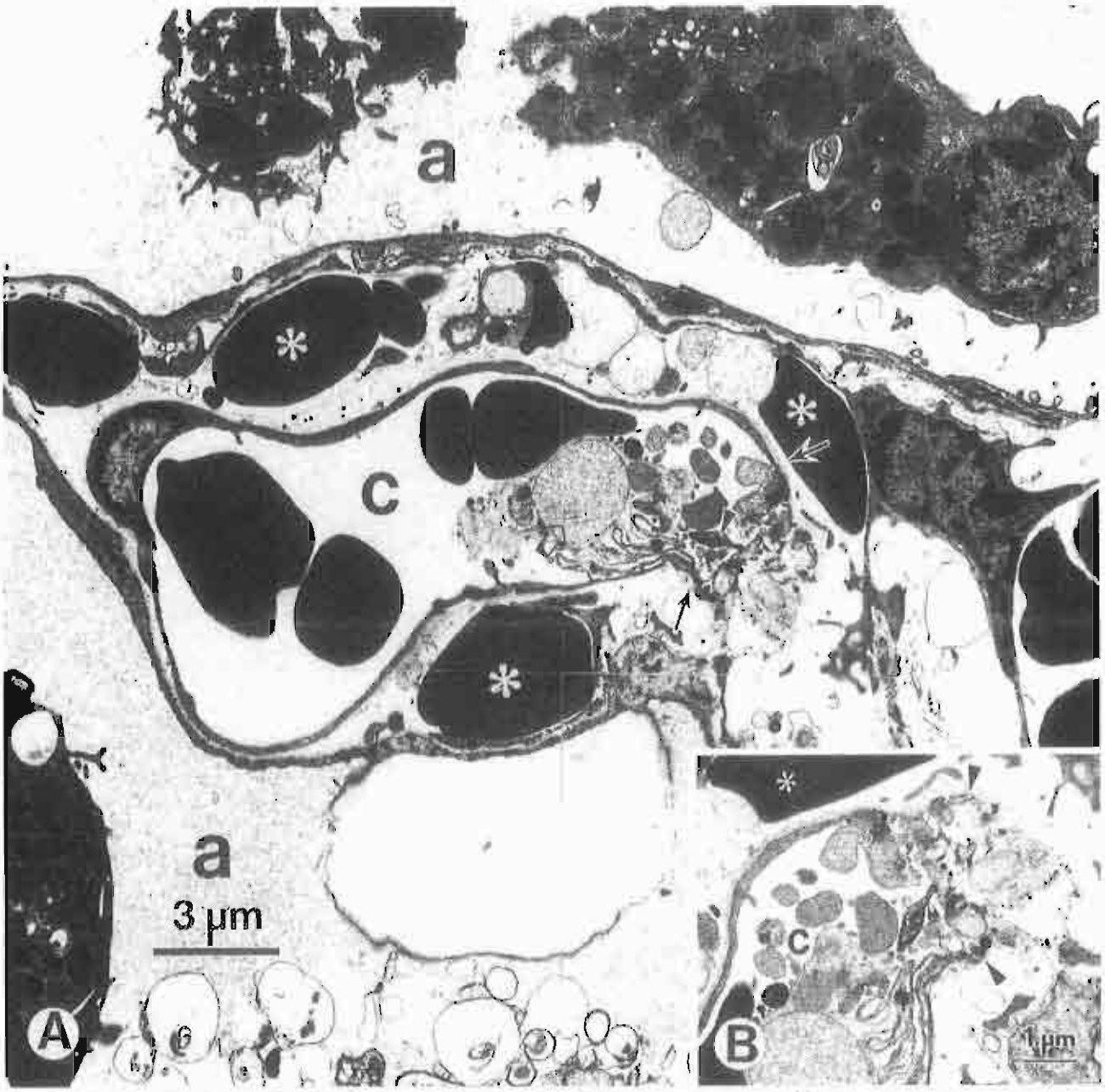

FIG. 5. A: montage showing another site of disruption of capillary wall (thin arrows) with accumulation of platelet (arrowhead) into opening. Note red blood cells (*) in interstitium, pulmonary intravascular macrophage (open arrow) in capillary, and granular and vacuolar material in alveolar space. *B*: enlargement of portion of endothelial disruption from *A* showing platelets passing through and lifting basement membrane (arrowheads). Note red blood cells (*) in interstitium.

Thickness of Blood-Gas Barrier

Figure 7 shows the ultrastructure of pulmonary capillaries, and Fig. 8 is a cumulative plot of total thickness of the blood-gas barrier in normal areas of the lung parenchyma in *animals 1* and *2*. In a previous paper, we demonstrated that the blood-gas barrier in rabbits was thicker when perfused and fixed at a higher transmural pressure (Ptm) (39). Although Ptm differed slightly during preparation of the samples from these two horses, no significant difference was seen between total blood-gas barrier thickness. For both animals 30% of measured blood-gas barrier thickness was <0.51 μm and $>50\%$ was <0.63 μm (Fig. 8).

Radius of Curvature of Capillaries

Using the criteria described under METHODS, we measured pulmonary capillary radii ranging from 2.75 to 4.35 μm [mean 3.35 ± 0.18 (SE) μm]. These criteria possibly underestimated the mean capillary radius by excluding those capillaries too large to fit within the micrograph frame at this magnification. On the other hand, the mean radius of those capillaries that were circular in cross sections but cut obliquely in our preparations was overestimated, because for those capillaries the "true" radius would be represented by the minor axis only. This could yield an overestimation of as much as 100% if all capillaries were circular in cross section. It should be emphasized

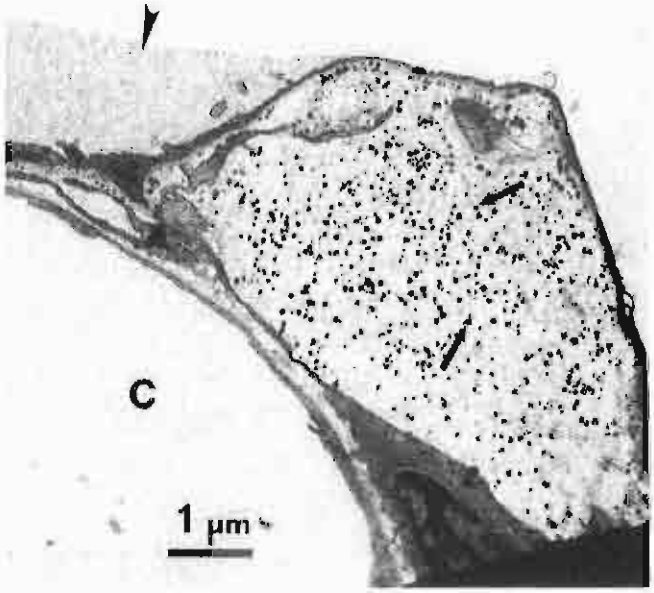

FIG. 6. Electron micrograph showing interstitial edema with accumulation of electron-dense particles (arrows) in lung parenchyma of thoroughbred horse (*animal 2*) perfusion fixed after maximal exercise. Note granular material (arrowhead) in alveolar space.

that the purpose of these measurements was to obtain a representative radius rather than a rigorous mean of all capillary radii. We recognize that some capillaries will have radii of curvature larger or smaller than the value of 3.35 μm obtained here.

Wall Stress of Capillaries During Galloping

As indicated under METHODS, the hoop stress of the capillary wall at any point depends on Ptm, radius of curvature, and wall thickness. Jones et al. (18) reported mean pulmonary arterial and left atrial pressures in horses galloping on a treadmill as 120 and 70 mmHg, respectively. If we assume that capillary pressure is halfway between arterial and venous pressures as reported by Bhattacharya et al. (3), who obtained micropipette measurements in animal lungs, this gives a capillary hydrostatic pressure of 95 mmHg. These investigators also showed that there is a substantial pressure drop along the capillaries and therefore that the pressure at the arterial end of the capillary is higher. In addition, Younes et al. (50) found that with high blood flow pulmonary capillary pressure is closer to arterial pressure than to venous pressure. Therefore, the value of 95 mmHg is a conservative estimate.

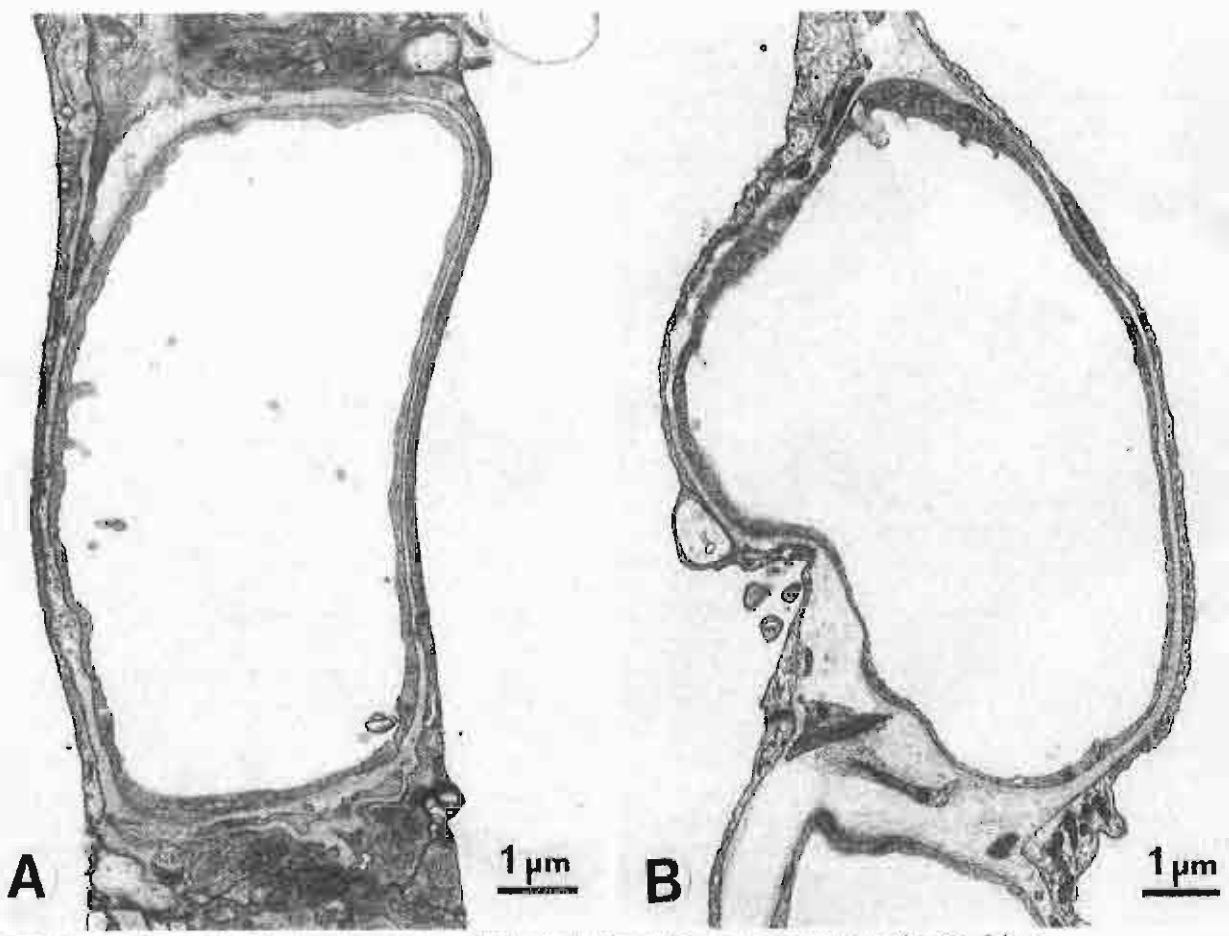

FIG. 7. Electron micrographs showing pulmonary capillaries in alveolar wall in normal parenchymal areas of thoroughbred horses perfusion fixed after maximal exercise. *A*: *animal 1*. *B*: *animal 2*. Total thickness of section of thin side of blood-gas barrier (*right* in both micrographs) is ~0.30 μm.

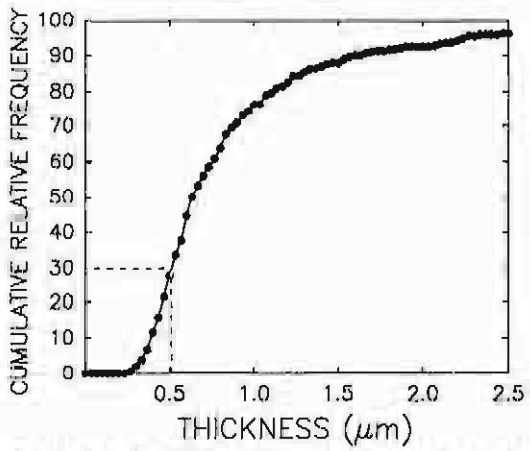

FIG. 8. Cumulative plot of thickness of blood-gas barrier in normal area of lung parenchyma in 2 thoroughbred horses. Note that 30% of thickness measurements are <0.51 μm.

There is considerable variation in both the wall thickness and radius of curvature of the capillaries, and we have not attempted to calculate a mean stress. Instead we have taken reasonable values for both thickness and radius, and we have calculated wall stress based on these values. The actual wall stresses will be both higher and lower than this nominal value.

Figure 8 shows that 30% of the blood-gas barrier thickness is <0.51 μm. As indicated above, the mean value for capillary radius is 3.35 μm. Inserting these values into the Laplace relationship gives the following wall stress

$$(95 \times 1.36 \times 981 \times 3.35)/0.51 = 8.3 \times 10^5 \text{ dyn/cm}^2$$
$$= 8.3 \times 10^4 \text{ N/m}^2$$

This is an extremely high wall stress. It can be compared with wall stress of the normal human aorta of $\sim 9 \times 10^5$ dyn/cm^2 (45), a blood vessel that is strongly armored with large amounts of collagen and elastin. The calculated wall stress of the horse capillaries exceeds the breaking stress of all soft tissues except collagen (49). Little wonder that galloping racehorses bleed into their lungs. Some areas of the blood-gas barrier are clearly thinner than the value of 0.51 μm used above (Fig. 7), so again the wall stress would be higher in these regions. Figure 7 shows the ultrastructural appearances of normal lung in *animals 1* and 2.

DISCUSSION

Ultrastructural Features Consistent With Stress Failure of Pulmonary Capillaries

Many of the ultrastructural findings described here are consistent with stress failure of pulmonary capillaries as the pathogenetic mechanism.

Disruptions of alveolar epithelial and capillary endothelial layers. Epithelial and endothelial disruptions are strong evidence of stress failure in a context in which the calculated stresses of the capillary walls are extremely high. We have seen breaks in the alveolar epithelial and capillary endothelial layers in rabbit lungs when the Ptm of the capillaries was raised to ≥40 mmHg (39). Breaks have also been seen in dog lung capillaries at a Ptm of 70 mmHg (47), and reversible increases in hydraulic conductance have been found in perfused dog lung lobes when the left atrial pressure exceeded ~40 mmHg (33, 38). It is not surprising that similar appearances occur in the horse lung when the capillary pressure approaches 100 mmHg.

An interesting feature of the present study is that the breaks were relatively difficult to find and were frequently associated with cell elements that appeared to be plugging the breaks. For example, Figs. 4 and 5 show endothelial breaks with several platelets and cytoplasmic extensions of leukocytes closely adjacent to the exposed endothelial basement membrane. The basement membrane is highly charged and reactive, and it is not surprising that it causes adherence of platelets and leukocytes. The presence of fibronectin and laminin in the basement membrane may also play a role. Figure 4*A* also shows two alveolar macrophages on the epithelial surface facing the endothelial breaks.

A possible explanation for the presence of the macrophage is that the epithelial layer was also disrupted (but out of the plane of the section and therefore not visible) and the macrophages were attracted to the exposed epithelial basement membrane. Another possibility is that surface receptors on the macrophages were upregulated as a result of substances produced either by the endothelial break itself or by activation of platelets and white blood cells exposed to the highly reactive endothelial membrane. As detailed below, RBCs and granular material accumulations were seen in the alveolar space (Figs. 2, 5*A*, and 6). We have suggested that the inflammatory markers seen in the alveolar fluid of high-altitude pulmonary edema (36) might be explained by activation of platelets and white blood cells by exposed capillary endothelial basement membrane in that condition (43). Lavage studies of rabbit lungs perfused at high capillary Ptm show the presence of the inflammatory marker leukotriene B_4 in the alveolar edema fluid (K. Tsukimoto, N. Yoshimura, M. Ichioka, N. Tojo, I. Miyazato, F. Marumo, O. Mathieu-Costello, and J. B. West, unpublished observations).

As stated above, relatively few sections of open breaks were seen in the alveolar epithelium and capillary endothelium, and we found large numbers of platelets, white blood cells, and macrophages in the immediate vicinity of the breaks. These findings may be related to the relatively long period of time between the occurrence of the very high pressure in the capillaries during galloping and the lung fixation. As pointed out under METHODS, these times were 67 min for *animal 1* and 78 min for *animal 2*. This is a very different time course from our original experiments with anesthetized rabbits and dogs in which the autologous blood perfusion was 1 min, the saline-dextran wash was 3–5 min, and the buffered glutaraldehyde was then infused immediately, all at the same high capillary Ptm (39, 47). Under these conditions large breaks in both the capillary endothelium and alveolar epithelium were easy to find.

We know that some of the breaks are rapidly reversible. In a study of stress failure in rabbit lungs, the autologous blood perfusion was done at a high pressure, but then the pressure was reduced for either 3 or 6 min of saline-dextran perfusion followed by intravascular fixation at the same low pressure (11). It was found that the

number of endothelial breaks decreased from a mean of 7.1 to 2.4/mm and the number of epithelial breaks decreased from a mean of 11.4 to 3.4/mm. Thus a substantial proportion of the breaks closes within 3–6 min of lowering the capillary pressure. It is therefore not surprising that the number of open breaks seen in the horse lung is small, since the pressure was lowered for >1 h before fixation of the lung occurred. The collections of platelets, white blood cells, and alveolar macrophages in the vicinity of the breaks also presumably reflect the amount of time available for these cellular responses to the capillary injury.

It is also possible that some of the abnormal features such as edema and accumulation of RBCs in the interstitium and alveoli represent injury from a previous run. This is particularly true of the accumulation of electron-dense particles (Fig. 6), which suggest the presence of hemosiderin. Electron-dense particles in the lungs of thoroughbreds with a history of EIPH have been previously described (41). Some electron microscopic sections show substantial amounts of collagen in the interstitium of the alveolar wall that might represent a response to capillary injury (Figs. 2*A* and 3). Cords of fibrous tissue were often seen in the immediate vicinity of RBCs in the alveolar interstitium.

RBCs outside capillary lumen in interstitium of alveolar wall. The presence of RBCs is strong evidence of stress failure in this context. The concentration of RBCs in the interstitium is often extremely high, and the cells are tightly packed together. Presumably plasma escaped with the cells but was absorbed from the interstitial space while the RBCs remained. Interestingly, it was seldom possible to see the disruptions in the capillary endothelium through which the RBCs escaped from the capillary. As indicated above, this may be because the disruptions of the endothelial layer are rapidly reversible when the pressure is reduced. Another possibility is that the RBCs track within the interstitium away from the endothelial disruption and the break is then out of the plane of the section.

RBCs and granular material resulting from high-protein edema. RBCs and granular material were frequently seen in the alveolar spaces. This observation is consistent with stress failure and damage to both the capillary endothelial and alveolar epithelial layers. Lavage studies with the rabbit lung preparation show a high concentration of large-molecular-weight proteins together with RBCs in the alveolar edema fluid, indicating a high-permeability type of edema (K. Tsukimoto et al., unpublished observations). This type of edema is also seen in other conditions believed to be caused by stress failure, including neurogenic pulmonary edema and high-altitude pulmonary edema (43). Edema of the alveolar wall interstitium is also consistent with stress failure and is seen in our rabbit preparations (39), although the edema can also be caused by simple hydrostatic edema.

Fluid-filled protrusions into capillary lumen (Fig. 1C). Fluid-filled protrusions into the capillary lumen are also a feature of stress failure in our rabbit preparation (11, 14). They have also been described in other investigators' studies in which stress failure was the apparent mechanism, although it was not recognized as such (16, 26). The protrusions appear to develop as a result of fluid accumulating between the capillary endothelial cell and its basement membrane. Sometimes the electron density of the fluid of the protrusion is much less than that of the plasma, as seen in Fig. 1C. We do not understand the mechanism of these protrusions, but they are a feature of a high capillary pressure and are often associated with other features of stress failure.

Wall Stress of Capillaries During Galloping

As indicated under RESULTS, the calculated wall stress for a capillary of 3.35 μm radius at a Ptm of 95 mmHg is $\sim 8 \times 10^6$ dyn/cm^2. However, it is possible that the capillary Ptm is even greater during parts of the breathing cycle. Although mean alveolar pressure is presumably close to atmospheric pressure, large swings in alveolar pressure probably take place during galloping. For example, it has been shown that esophageal pressure in galloping thoroughbreds can fall transiently to about −20 mmHg with respect to atmospheric pressure (12). Presumably intrapleural pressure changes by approximately the same amount. Much of this fall is transmitted to the alveolar spaces, and although pulmonary vascular pressures may fall along with intrapleural pressure the net result may be an increase in capillary Ptm.

An interesting question is what makes the capillaries of the horse lung so strong. Clearly, if horses developed stress failure at the same capillary Ptm as rabbits do (that is, ~40 mmHg), then they would not be able to withstand the enormous Ptm to which they are subjected during intense exercise. As indicated earlier, we know that there are species differences in the strength of pulmonary capillaries. For example, the capillaries of dog lungs fail at a pressure ~30 mmHg higher than that in the capillaries of rabbit lung (47). There is evidence that the strength of the thin side of the blood-gas barrier comes from the type IV collagen in the extracellular matrix of the capillary wall (44). It may be that the thickness or the composition of the type IV collagen is special in the horse lung and that it confers greater strength as a result.

The reasons for the very high pulmonary vascular pressures in the galloping racehorse are presumably the extremely high aerobic capacities of these animals and the requirement that their cardiac outputs be so high. This in turn means that the filling pressures for the left ventricle are extremely high. These animals have a maximal oxygen consumption of up to 180 ml · min^{-1} · kg^{-1}, which is, for example, more than twice the maximal value of 80 ml · min^{-1} · kg^{-1} in human elite athletes. Specific cardiac outputs of galloping thoroughbreds exceed 750 ml · min^{-1} · kg^{-1}, which is also an extremely high value. Maximum HR values have been measured to be as high as 240/min, and under these conditions the left ventricle apparently needs an enormously high pressure to fill it, which explains the mean left atrial pressures being as high as 70 mmHg. There may also be a substantial pressure drop across the mitral valve because the filling time of the left ventricle is so short and the blood flow rate is correspondingly high.

These animals have developed their extreme athletic ability through >400 yr of selective breeding (6). As an example, >25% of the genetic material in the total gene pool of the modern thoroughbred can be traced back to

four stallions. It is interesting that racing greyhounds also apparently develop EIPH (21). These animals have also been selectively bred for high aerobic capacities.

Possible Objections to Stress Failure as a Mechanism of EIPH

A feature of the pathology of EIPH in the racehorse is that the lesions in the lung are most abundant in the dorsocaudal region (24, 27). At first sight this distribution appears to argue against stress failure as a mechanism because, other things being equal, the capillary pressure would be lower in the dorsal region than the ventral region of the lung because of the hydrostatic pressure gradient. However, there are several reasons why this may be an oversimplification.

First, there is some evidence that pulmonary blood flow is especially high in the dorsocaudal region when the animal is in the normal prone position (9, 17). Presumably this can be explained by a lower intrinsic vascular resistance of the blood vessels supplying this part of the lung. Some measurements in isolated dog lung also suggest that regional blood flow is unexpectedly high in the dorsocaudal part of the lung (2).

Another possibility is that the alveolar pressure falls transiently to low values in the dorsocaudal region during galloping. The diaphragm runs very obliquely in a caudodorsal to cranioventral direction in the horse, with the result that the dorsocaudal lung is bounded by the rib cage above and the diaphragm below. It is possible that displacement of the diaphragm during galloping as a result of movements of the massive abdominal contents could result in large transient falls in alveolar pressure in the dorsocaudal part of the lung. Also, the very long distance from the nares to the dorsocaudal region of the lung could result in unusually low transients of alveolar pressure during inspiration. The horse is an obligate nose breather, and if the airway resistance of this route of airflow were high, then one would expect to see large falls in alveolar pressure during inspiration, which could therefore increase capillary Ptm.

Finally, it is probable that the dorsal alveoli are larger than those elsewhere in the lung because of the distortion that the lung incurs because of its own weight. The most negative pleural pressures in the horse occur in the dorsocaudal region (7). The increased expansion would be expected to contribute to the frequency of stress failure. We have shown that in the rabbit preparation increasing alveolar volume at the same capillary Ptm greatly increases the incidence of stress failure (14). For example, with a constant capillary Ptm of 32.5 cmH_2O, increasing the lung volume from a normal to a high value increased the number of endothelial breaks from a mean of 0.7 to 7.1/mm, whereas the number of epithelial breaks rose from a mean of 0.9 to 8.5/mm. These findings are in line with work by other investigators showing increases in capillary permeability and the development of microvascular injury at high lung volumes (10, 30). It is known that the alveoli at the top of the human lung are relatively large because the lung distorts under its own weight, and the same is presumably true of the horse. Possibly the dorsocaudal region is particularly vulnerable because its situation between the dorsal rib cage and oblique diaphragm referred to above means that downward movements of the abdominal contents during galloping could cause transient increases in alveolar size in that region.

TABLE 1. *Possible ways of reducing stress failure in pulmonary capillaries*

Strengthen capillary wall, possibly by increasing amount or strength of collagen
Reduce capillary wall stress
Reduce capillary hydrostatic pressure, possibly by increasing contractility of left ventricle
Avoid large falls in alveolar pressure, e.g., by relieving any upper airway obstruction
Avoid overinflation of alveoli

Another possible objection to the stress failure explanation for EIPH is the previously referred to pathological findings after a very detailed autopsy study of 26 horses (27–29). As summarized earlier, the principal findings were hemosiderophages, neovascularization of the bronchial circulation, and evidence of bronchiolitis in the affected areas. However, it is possible that the bronchiolitis, including connective tissue reactions, and neovascularization of the bronchial circulation were secondary to the presence of blood in the alveoli and small airways. It should be pointed out that EIPH apparently occurs in essentially all athletically active thoroughbreds from the time that they begin to gallop, and thus there is a long period over which low-grade inflammatory reactions could take place.

It could be argued that, although stress failure of pulmonary capillaries is the mechanism of EIPH, the high pressure within the capillaries comes from the bronchial rather than the pulmonary circulation. It is known that galloping thoroughbreds can have mean systemic arterial pressures >240 mmHg (18). Furthermore, the anatomic study of McLaughlin et al. (25) described anastomoses between the terminal parts of the bronchial arteries and the pulmonary capillaries in horse lung. Thus, a pathway may exist that would allow the pulmonary capillaries to be exposed to the very high pressures in the systemic circulation.

On the other hand, as we have seen, there is strong evidence that the pressure in the pulmonary capillaries can approach or even exceed 100 mmHg during galloping simply as a result of the rise in pressure in the pulmonary circulation itself. Therefore, it is not necessary to invoke any additional factor to account for pressure-induced damage to the pulmonary capillaries. Whether the apparent link between the bronchial and pulmonary circulations plays a critical role is uncertain.

Possible Prevention of EIPH

The main objective of this paper is to provide evidence for a new pathogenetic mechanism for EIPH, namely stress failure of pulmonary capillaries as a result of the extremely high pressures within them during galloping. However, it is natural to speculate on how the condition might be prevented or at least alleviated if this mechanism should be correct.

If EIPH were caused by stress failure of pulmonary capillaries, then the only ways to reduce its severity would be by strengthening the capillary wall or by reducing the capillary wall stress (Table 1). Very little is

known about possible ways of strengthening the capillary wall, although it may be possible to increase the amount or strength of type IV collagen. It is known that patients with chronic increases in pulmonary capillary pressure caused by mitral stenosis or pulmonary venoocclusive disease develop thickened basement membranes in their capillary walls (19, 20, 22). Indeed, it may be that type IV collagen accumulation is continuously regulated by the capillary hydrostatic pressure. However, as pointed out elsewhere (44), the blood-gas barrier has a bioengineering dilemma in that it has to be extremely thin for adequate gas exchange while also being strong enough to avoid stress failure. Thickening the blood-gas barrier is likely to interfere with its diffusion characteristics and therefore oxygen transfer properties, thus reducing aerobic capacity. There is strong evidence that normal thoroughbreds develop arterial hypoxemia when they gallop and that this is caused by diffusion limitation of oxygen transfer in the lung (42). Therefore, additional thickening of the blood-gas barrier is likely to further limit oxygen transfer.

Reducing the Ptm of the capillaries could conceivably be done either by reducing capillary pressure itself or by ensuring that alveolar pressure does not fall transiently to low values. Capillary hydrostatic pressure is high because of the greatly elevated left atrial pressure, which in turn is apparently necessary for adequate filling of the left ventricle. If the contractility of the left ventricle could be increased, then it might be possible to lower left atrial pressure without reducing cardiac output and thus presumably without lowering aerobic capacity.

As regards alveolar pressure, if the large falls during inspiration were caused in part by a high airway resistance resulting, for example, from upper airway obstruction (which is common in thoroughbreds), then relief of the obstruction might be expected to reduce the incidence of EIPH. Indeed, airway obstruction has been suggested as a possible etiological factor (5). However, if the large falls in alveolar pressure are inevitable because of the normal anatomy of the airways and diaphragm, presumably little can be done to alter this factor.

Finally, large alveolar volumes will predispose the subject to stress failure, but if these are caused by distortion of the lung by its weight or by displacements of the diaphragm during galloping, then presumably little can be done to modify these factors.

The drug furosemide is extensively used in states where permitted, but there is little objective evidence that it reduces the incidence of EIPH (32). However, it does reduce pulmonary arterial and right atrial pressure in the exercising horse (13, 15), and so it would be expected to reduce capillary pressure. There is a theoretical basis for its effect on pulmonary vascular pressures, since the drug is a powerful diuretic that reduces circulating blood volume and also an acute venous vasodilator (8). Both of these pharmaceutical actions might be expected to reduce pulmonary vascular pressures. However, the fact that the drug is usually required to be given several hours before a race presumably means that its diuretic action is more important. Whether it is possible to reduce pulmonary vascular pressures with drugs such as furosemide and not interfere with cardiac output and thus aerobic capacity is therefore unclear.

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ATTACHMENT #3

2/ EXERCISE INDUCED PULMONARY HEMORRHAGE, EIPH:

Historically, about 1 % of Horses bleed from their nostrils post-race, called **Epistaxis,** known for at least 300 years. If you endoscope a horse post-race, about 75% of horses show blood in the trachea. These endoscopic findings were first reported by Pascoe et al in 1981, leading to the scientific name, **Exercise-Induced Pulmonary Hemorrhage, EIPH,** Attachment #3.

EIPH is defined as bleeding into the lungs associated with exercise; at some level it occurs in 100% of racing horses. If you do a **Tracheal Wash**, all horses in training show evidence of bleeding into the lungs. The pulmonary damage is CUMULATIVE, and EIPH is equivalent to a production disease in racing horses.

Significant bleeding into the lungs interferes with blood oxygenation (Sanchez et al 2005), slows the horse and is therefore associated with poor racing performance. The bleeding may, at times, be severe enough to cause death, either acutely on the racetrack or soon thereafter, as we will present later.

Gervase Markham's Master-Piece
Curing all DISEASES
in
HORSES
[1766 Edition]
[1568 – February 3, 1637]

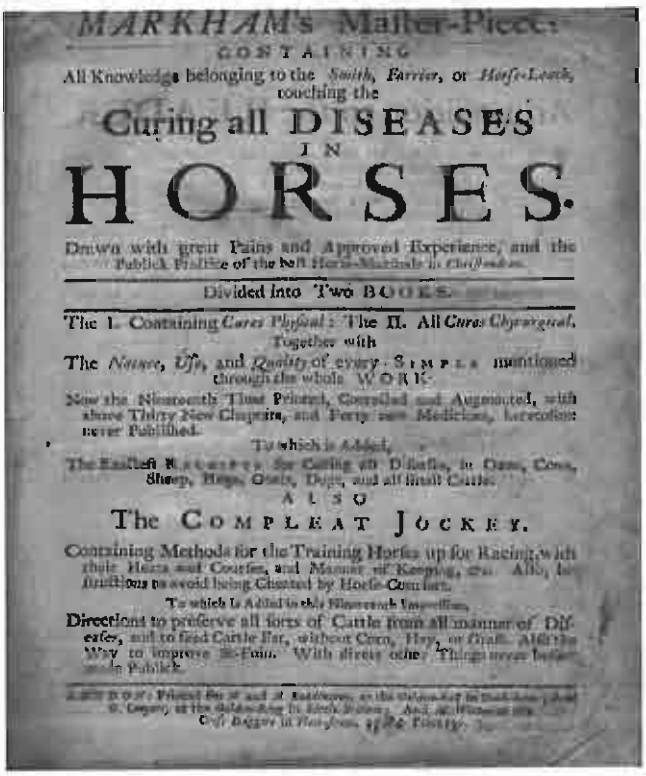
CHAP. 28: ***Of bleeding at the nofe***

Many horfes [efpecially young horfes] are often fubject to this Bleeding at the Nofe, which I imagine proceedeth either from the much abundance of Blood, or that the Vein, which endeth in that Place is either broken, fretted or opened.

[1568 – February 3, 1637]

In somewhat more modern English→

Many horses [especially young horses] are often subject to this Bleeding at the Nose, which I imagine proceedeth either from the much abundance of Blood, or that the Vein, which endeth in that Place is either broken, fretted or opened.



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LONDON: Printed for E. and J. Harrison, at the Golden Key in Pope's-Head-Alley, and G. Conyers, at the Golden Ring in Little Britain. And to be Sold at their Shops. 1683.

CHAP. XXVIII. *Of Bleeding at the Nose.*

MAny Horses (especially young Horses) are often subject to this Bleeding at the Nose, which I imagine proceedeth either from the much abundance of Blood, or that the Vein which endeth in that Place is either broken, fretted, or opened. If it opened many Times by means that Blood aboundeth too much, or that it is too fine or too supple, and so piereeth through the Vein. Again, it may be broken by some violent Strain, Cut, or Blow; and lastly, it may be fretted and gnawn through by the Sharpness of the Blood, or else by some other evil Humour contained therein. The Cure is, according to the ancient Farriers, To take the Juice of the Roots of Nettles, and squirt it up into the Horse's Nostrils, and lay upon the Nape of the Horse's Neck a Wad of Hay dip'd in cold Water, and when it waxeth warm, take it off, and lay on a cold one. Other Farriers use to take a Pint of red Wine, and put therein a Quartern of Bole-Armoniack, beaten into fine Powder, and being made lukewarm, to pour the one half thereof the first Day into the Nostril that bleedeth, causing his Head to be holden up, so as the Wine may not fall out, and the next Day, to give him the other half.

Others use to let the Horse blood on the Breast-Vein, on the same Side that he bleedeth, as several times: Then take, of Frankincense one Ounce, of Aloes half an Ounce, and beat them into fine Powder, and mingle them throughly with the Whites of three Eggs, until it be as thick as Honey, and with soft Hair thrust it up into his Nostrils, filling the Hole full of Ashes, Dung, or Hogs-Dung, or Horses Dung mix'd with Chalk and Vinegar.

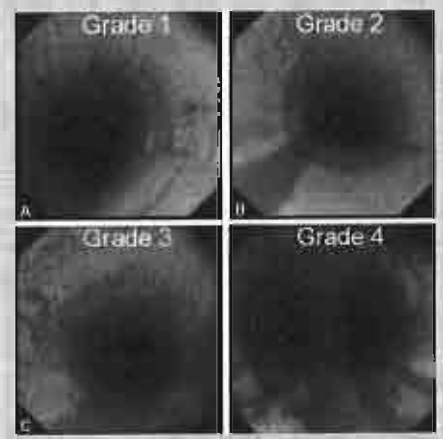
Now for mine own Part, when none of these will remedy and help (as all have failed me at some time) then I have used this: Take two small Whipcords, and with them garter him exceeding hard about some ten or twelve Inches above his Knees of his Fore-legs, and just beneath his Elbows, and then keep the nape of his Neck as cold as may be, with moist Cloaths, or wet Hay, and it will staunch him presently.

CHAP. XXIX. *Of the Bloody Rifts, or Chops in the Palate of the Horse's Mouth.*

THese Chops, Clefts, or Rifts in the Palate of the Horse's Mouth, do proceed (as some Farriers suppose) from the eating of rough Hay, full of Wins, Thistles, or other Prickling-stuff, Provender full of sharp Seed, which by continual pricking and fretting the Furrows of the Mouth, do cause them to rankle, swell, and breed corrupt Blood and stinking Matter, and without speedy Prevention, that Ulcer will turn to the foulest Canker.

Endoscopic grades of EIPH

- Grade 0 = No blood detected
- Grade 1 = One or more flecks of blood or ≤ 2 short, narrow streams (<10% of tracheal surface
- Grade 2 = One long stream or > 2 short streams occupying less than 33% of tracheal circumference
- Grade 3 = Multiple distinct streams of blood covering greater than 33% of tracheal circumference
- Grade 4 = Multiple, coalescing streams of blood, covering more than 90% of the tracheal surface with blood pooling at the thoracic inlet



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Exercise-Induced Pulmonary Hemorrhage in Racing Thoroughbreds: A Preliminary Study

John R. Pascoe, BVSc; G. L. Ferraro, DVM; J. H. Cannon, DVM; R. M. Arthur, DVM; J. D. Wheat, DVM

SUMMARY

Of 235 Thoroughbred racehorses examined with a flexible fiberoptic endoscope within 2 hours of racing to determine the frequency of exercise-induced pulmonary hemorrhage (EIPH), 103 (43.8%) had various degrees of hemorrhage in the tracheal lumen. Two of these horses (0.8%) subsequently had blood flow from the nostrils. Blood seemed to originate from the lung. Statistical analysis of frequency data for 191 horses which finished in 1st, 2nd, and 3rd places did not show any relationship between EIPH and horse's age, sex, or finishing position. However, a trend toward an increased frequency of EIPH with age was shown, by a greater proportion of horses 5 years and older having EIPH. This trend is thought to reflect the chronicity of the pulmonary lesions and an inability of the lung to repair damaged regions while training and racing continued. The efficacy of furosemide for the treatment of EIPH was questioned, since 30 of 56 furosemide-treated horses which were examined had evidence of pulmonary hemorrhage. Nineteen (8%) horses had visible functional abnormalities of the upper respiratory tract.

Frequencies of "bleeding" in racing horses in Australia,[1] England,[2] South Africa,[3,4] and the United States[5] have been reported. These reports were based on observations of hemorrhage at 1 or both nostrils and examination of the upper respiratory tract with a rigid endoscope.[2] The introduction of the flexible fiberoptic endoscope has facilitated the clinical examination of the equine upper respiratory tract. The apparent increase in the number of Thoroughbred horses bleeding (exercise-induced pulmonary hemorrhage; EIPH) during racing or training in southern California was of growing concern to the California Horse Racing Board.* This apparent increase was highlighted by the large number of requests, from horse trainers, for permission to use furosemide as a raceday medication for the control of bleeding.

Accordingly a survey was conducted over 15 consecutive racing days at the Del Mar Thoroughbred Club, southern California, in August and September 1978, to determine the frequency of bleeding in racing Thoroughbreds, the origin of the hemorrhage, and the relationship, if any, between bleeding status and the horse's age, sex, and finishing position in the race. The effect of prerace medication with furosemide on pulmonary hemorrhage was also investigated. Also of interest was the frequency of functional abnormalities of the equine upper respiratory tract in a group of Thoroughbred racing horses.

Materials and Methods

All trainers at the Del Mar Thoroughbred Club race meeting were petitioned for their cooperation in the survey. The reluctance shown by a few trainers to participate placed limitations on the availability of horses for examination. Within these constraints, single endoscopic observations to diagnose EIPH were made on 235 horses. Of these, 191 horses finished in 1st, 2nd, or 3rd place and were examined in the receiving barn stalls immediately after urine test samples were collected, usually within 90 minutes of completion of racing. The remaining 44 horses were voluntarily entered

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* Edmonson AH, California Horse Racing Board Veterinarian: Personal communication, 1978.

in the study by 8 trainers who also trained 54 of the 191 horses which placed. The 44 volunteered horses completed their races, but did not place and were examined at their respective barns after "cooling out" (within 2 hours of completion of racing).

Both nostrils were examined for signs of hemorrhage. Then, with the application of restraint (nose twitch), a 100-cm (length) flexible fiberoptic endoscope[b] was inserted along the ventral meatus for a distance of approximately 25 cm. Visual examination of the caudal part of the nasal cavity was made. The endoscope was advanced slowly forward into the nasopharynx, through the rima glottidis, and down the tracheal lumen for a distance of approximately 60 cm. The following anatomic structures were examined: the caudal nasal cavity, the ethmoid labyrinth, nasopharynx, soft palate, pharyngeal recess, pharyngeal openings of the auditory tubes, epiglottis, arytenoid cartilages and vocal folds, laryngeal floor, and tracheal lumen.

Any unusual changes were recorded and reported to the trainer's veterinarian. Horses with abnormal laryngeal function or other abnormality of the upper airway were reexamined at rest, the following day. Variations in the degree of hemorrhage visible in the trachea were graded numerically: grade I—traces of blood in tracheal mucus (Fig 1), grade II—a streak of blood less than 5 mm wide (Fig 2), grade III—a streak of blood greater than 5 mm wide and less than 15 mm wide (Fig 3), and grade IV—a streak of blood greater than 15 mm wide (Fig 4). Records of horses permitted to use furosemide as a prerace medication were obtained from the California Racing Board veterinarian.

The frequency data obtained from the study were analyzed statistically, using the chi-square test for association. When a significant or marginal chi-square value was obtained, the degrees of freedom on which the overall chi-square was based were partitioned to determine where the significant differences lay.[6]

Results

The ages and sexes of the horses sampled were consistent with these characters of the overall population of horses that were raced during the 15-day period of the survey. All horses categorized as affected with EIPH had evidence of hemorrhage in the tracheal lumen. In no instance did blood originate from the caudal part of the nasal cavity, ethmoid labyrinth, or pharyngeal openings of the auditory tubes. Blood was never present at either nostril at the time of examination. Two horses with EIPH were subsequently reported to have blood flow from the nostrils after they were returned to the stalls.

Of 235 horses examined, 103 (43.8%) had various degrees of blood in the tracheal lumen. Of 103 horses categorized as having EIPH, 73 (70.9%) had not been given furosemide as a prerace medication and before this examination, had not been suspected by their trainers of bleeding. Details of the effect of prerace treatment with furosemide a (Table 1): Of 56 horses treated with furosemide, had blood in the tracheal lumen.

Sixteen horses had blood present in the cau the trachea only, and an additional 20 horses ha blood in the cranial part of the trachea with th increasing caudally. Traces of blood on the dorsal the epiglottis (Fig 5), in addition to blood in th lumen, were noted in 37 horses. Two horses h sprayed circumferentially on the tracheal walls th the length of the trachea. Both horses had coug sively while cooling out.

Although many horses coughed occasionally c cooling-out period, 19 horses coughed almost con Three of these horses had large amounts of soil ac the walls of the nasopharynx, 5 horses had an adn blood and thick white mucopurulent material in th lumen, and another 9 horses had whitish flocculent distributed along the tracheal floor and a hyper inflamed tracheal mucosa. Two 2-year-old colts ha mm clumps of soil lying on the tracheal floor up caudal to the rima glottidis. One of these col moderate amount of blood interspersed with the s cles.

Of 235 horses, 19 (8.1%) had abnormalities of th respiratory tract. Three (1.3%) had left laryngeal h gia, and 9 (3.8%) had various degrees of asymm asynchronous movement of the left arytenoid carti vocal fold. A 3-year-old gelding had complete par the right arytenoid cartilage and vocal fold. Two ho immobile abducted left arytenoid cartilages consist surgical abduction. Two 4-year-old horses had aryt lottic entrapment, and in a 7-year-old cryptorchi was bilateral chondroma of the arytenoid cartilage pharyngeal polyps were seen to protrude from th pharyngeal wall in a 2-year-old colt.

The frequency data obtained for horse's age, s position in race from 191 horses which finished in 1 and 3rd places are shown (Tables 2 through 4). St analysis of the data showed no relationship betwe and the horse's age, sex, and finishing position in th Partitioning of the degrees of freedom (df) on wh overall chi-square value for age was based ($\chi^2 = 5.$ Table 2) indicated that the proportion of horses years and older that had EIPH was larger than the p tion of horses aged 2, 3, and 4 years ($\chi^2 = 4.56$, 1 < 0.05).

Since no relationship between EIPH and finishin tion was found in horses finishing in 1st, 2nd, and 3rd the frequency data of 98 horses of 8 trainers were u

[b] Olympus GIF-D, Olympus Corp of America, New York, NY.

Legends for Illustrations on Facing Page

Fig 1 (Top left)—Grade I EIPH; traces of blood in the tracheal mucus. (The small discrete dots are nonfunctional fiber bundles in the endoscop

Fig 2 (Top right)—Grade II EIPH; a streak of blood less than 5 mm wide.

Fig 3 (Middle left)—Grade III EIPH; a streak of blood greater than 5 mm, but less than 15 mm wide.

Fig 4 (Middle right)—Grade IV EIPH; a streak of blood greater than 15 mm wide.

Fig 5 (Lower left)—Endoscopic view of the epiglottis, arytenoid cartilages, vocal folds, and the aditus laryngis. Note the blood on the dorsal su the epiglottis and the traces on the arytenoid cartilages.

Fig 6 (Lower right)—Endoscopic view of the floor of the larynx showing the pooling of blood with a thinner streak of blood seen in the cranial pa trachea.

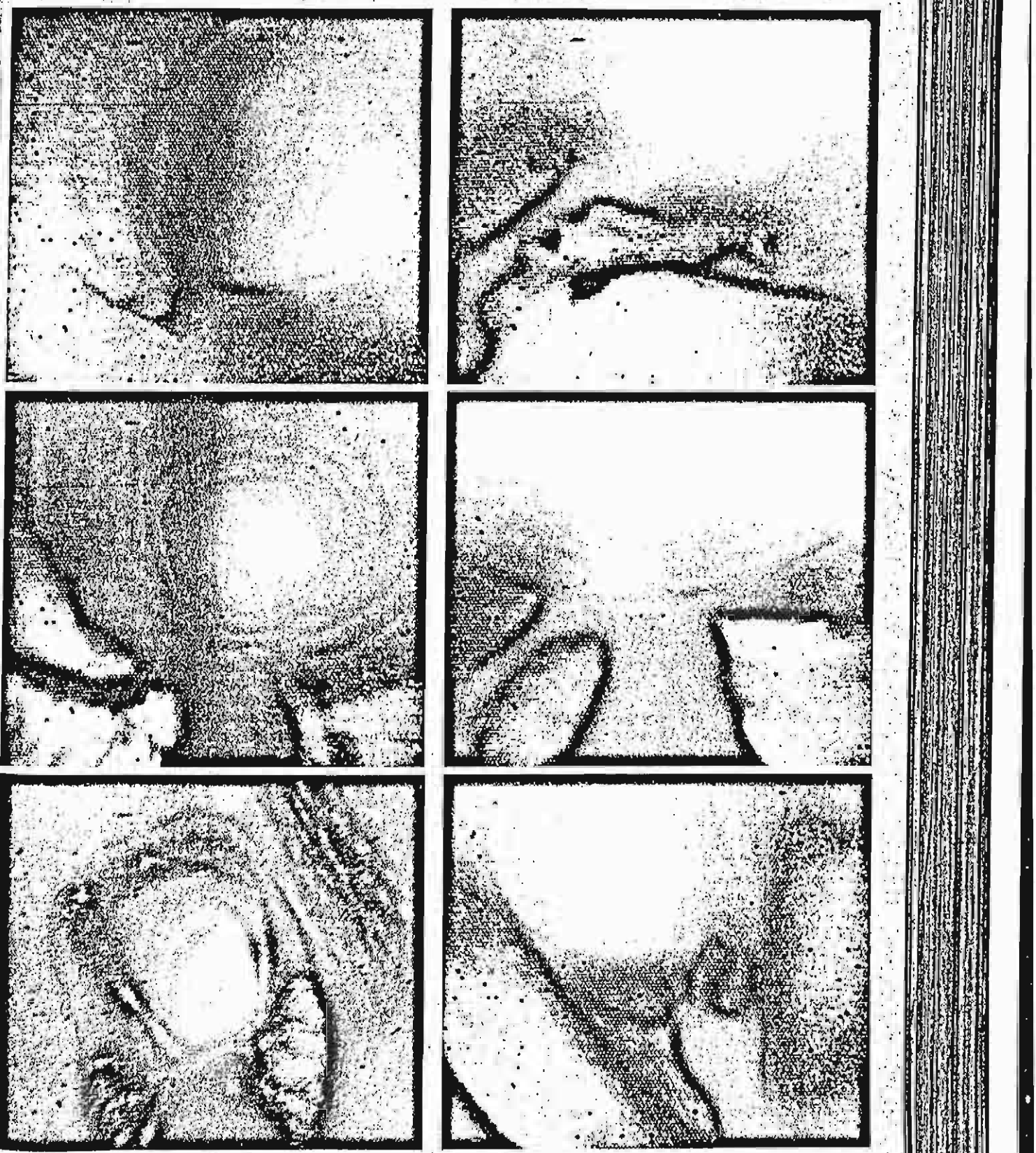

TABLE 1—Relationship Between Prerace Medication with Furosemide and EIPH in 235 Thoroughbred Racehorses

Prerace medication	EIPH		
	Present	Absent	Subtotals
Furosemide	30	26	56 (23.8%)
No furosemide	73	106	179 (76.2%)
Totals	103 (43.8%)	132 (56.2%)	235 (100 %)

TABLE 2—Age Distribution of 191 Thoroughbred Racehorses Finishing in First, Second, or Third Place

EIPH	Age (years)				
	2	3	4	5+	Subtotal
Positive	16	28	17	25	86
Negative	28	36	24	17	105
Total	44	64	41	42	191

Chi-square = 5.13, not statistically significant at $P = 0.05$ level.

TABLE 3—Sex Distribution of 191 Thoroughbred Racehorses Finishing in First, Second, or Third Place

EIPH	Sex			
	Filly	Gelding	Colt	Subtotal
Positive	39	22	25	86
Negative	36	38	31	105
Total	75	60	56	191

Chi-square = 3.7, not statistically significant at the $P = 0.05$ level.

determine whether EIPH was more often diagnosed in horses placing in the first 3 positions. A relationship was not found between EIPH and race-finishing position (Table 5).

Discussion

There has been considerable speculation on the origin of the blood noted in the respiratory tracts of racing horses classed as bleeders. A variety of terms have been applied to this condition—"broken blood vessels,"[7] "bleeding,"[8] "epistaxis,"[2-5] "sporadic idiopathic epistaxis,"[9] and "pulmonary hemorrhage"[2,10]—and some, through common usage, remain entrenched in racing parlance. The acceptance of an association between the presence of blood at 1 or both nostrils of a horse and its origin as being within the nasal cavity or head has persisted in veterinary literature for at least 3 centuries. Even with the advent of endoscopy, little has been achieved to clarify the origin of this hemorrhage. Many of these beliefs were dispelled in a retrospective survey of 174 horses with clinical evidence of hemorrhage at the nostrils.[2] The source of blood was identified in 124 horses by direct examination and the remaining 50 horses were, in the absence of contrary evidence, assumed to be bleeding from the lungs. In addition, in these 50 horses of mixed use, pulmonary hemorrhage was precipitated by competitive exercise only.

Several observations in this survey support the thesis that hemorrhage originates within the caudal part of the lung.[2] A total of 16 horses had a stream of blood in the caudal part of the trachea only. Since the clearing action of the mucociliary blanket in the tracheobronchial tree has a net outward directional flow, that is, away from the lung parenchyma, it seems reasonable to suggest that the blood originated within the lung. Further support is added by the observation that 20 horses had traces of blood in part of the trachea with increasing amounts cau highly unlikely that the blood is inspired into th as suggested by Robertson,[7] since no sites of h external to the trachea were observed. In ad consistently uniform appearance of the blood as located stream within the tracheal lumen with blood deposition on the remainder of the trac would seem to further diminish the likelihood of ring.

On the basis of these findings, the term "e duced pulmonary hemorrhage" is proposed to r previously used terms, as a more accurate desc this clinical entity in the racing horse.

The frequency of bleeding reported in previo varied from 0.25% to 2.5%.[1-4] These data, taken fr records, include "flat racers" and jumpers and w on the observations of hemorrhage at the nostrils. parison, 0.8% (2/235) of the horses examined in th showed blood at both nostrils. The relatively high f (44%) of EIPH reported in this survey reflects th endoscopy for diagnosis. By inference, it raises the that had flexible fiberoptic endoscopy been avail diagnostic technique in the time of the previous would the reported frequencies have been con higher?

Association between the horse's age and EIPH shown in this survey (Table 2). By partitionin associated with the overall chi-square for age, a t wards an increased frequency of EIPH in horses age and older could be shown. Previous studies have i that the frequency of EIPH increased with the horse it being more common in horses aged 4 years and Only a small number of 2- and 3-year-old hors included in previous studies which were not anal statistical methods.[1-3]

This trend toward an increasing occurrence of EI age bears further investigation. It is conceivable th an effect would be a result of the chronicity of the nary lesions and may, in fact, reflect an inability of t to adequately repair the damaged regions in the continued training. It is not known if periods of e rest following episodes of EIPH will diminish the lik of recurrence when training is resumed.

TABLE 4—Distribution of 191 Thoroughbred Racehorses, Fin First, Second, and Third Places

EIPH	Position in race			
	1st	2nd	3rd	Subt
Positive	34	38	14	8
Negative	48	41	16	10
Total	82	79	30	19

Chi-square = 0.75, not statistically significant at the $P = 0.05$ level.

TABLE 5—Distribution of 98 Thoroughbred Racehorses

EIPH	Position in race				
	1st	2nd	3rd	Other*	Sub
Positive	13	8	3	17	
Negative	18	9	3	27	
Total	31	17	6	44	

* Includes horses finishing in 4th or lower positions in their races.
Chi-square = 0.54, not statistically significant at the $P = 0.05$ level.

There was no relationship between sex and EIPH (Table 3), and this is in agreement with an earlier study which did not indicate any sex predisposition.[2] In contrast, a recent South African study suggested that bleeding occurs twice as commonly in geldings as it does in entire males and females.[4] This study based on racing records of observations of hemorrhage at 1 or both nostrils may have yielded different results had endoscopy been used to determine EIPH.

The effect of EIPH on performance is variable. Classically, it was believed that horses that bled during a race showed impaired performance often with a dramatic reduction in speed at some point during the race. Association between finishing position and EIPH was not shown in the present survey (Tables 4 and 5). This finding raises questions about the degree of physiologic impairment to performance caused by EIPH. Certainly horses with EIPH, finishing in the remainder of the field, were more likely to show marked reduction in speed; however, there are numerous other clinical reasons, eg, functional upper airway obstruction and lameness, which can result in alterations in performance.

Whether the performance of horses placing in a race would improve in the absence of EIPH bears further investigation. Although a grading system for EIPH was used, relationship was not seen between the degree of hemorrhage observed and the finishing position. This probably reflects the individual variation in the location of the lesion within the lung parenchyma, the rate of mucociliary clearance, the time of onset of hemorrhage during the race, and the time from completion of racing until examination.

Frequent swallowing after exercise has been reported in 1 bleeding horse and it was suggested that this was an indication that a horse is swallowing blood which is flowing into the pharynx from the trachea.[2] Endoscopic examination permitted observing the movement of blood in a stream along the length of the trachea, pooling in the floor of the larynx (Fig 6), flowing onto the epiglottis (Fig 5), and subsequently being swallowed. Repeated swallowing was a consistent clinical sign of EIPH in many of the horses examined. Often this will be the first indication of EIPH noted by astute horsemen.

The efficacy of furosemide in the management of EIPH remains a contentious issue. Under California Horse Racing Board regulations, the administration of furosemide to authorized horses is not permitted within 3 hours of racing. Of the 56 furosemide-treated horses examined in the present survey, 30 (53.6%) showed evidence of EIPH after racing. Assuming that these horses were legitimately treated because of prior episodes of EIPH, the results create doubts about the efficacy of furosemide treatment for the prevention of EIPH. It is not known, however, if furosemide reduced the amount of hemorrhage these horses may have otherwise experienced.

Clinical time trials with Standardbred horses have not shown any statistically significant differences between 1-mile pace times of furosemide-treated horses and control horses.[11, 12] Comparative data on the performance-related effects of furosemide on horses with EIPH vs untreated horses with EIPH and normal horses are not available. Retrospective analysis of actual racing times in Standardbreds before and after treatment with furosemide has also been reported, with no significant difference in performance being noted.[13] Since previous episodes of EIPH were not a condition for using furosemide under racing conditions in this group of horses, this analysis still does not provide information on the performance-related effects of furosemide in horses with a history of EIPH.

Time trials for examining the effects of furosemide on performance have not been conducted on Thoroughbred horses. A similar retrospective analysis of Thoroughbred performance times has not been carried out. Such an analysis would be further complicated in Thoroughbreds by additional variables including various racing distances, racing handicap weights, and changing track surface conditions.

Several hypotheses relating to the pathogenesis of EIPH have been advanced and were recently reviewed.[14] Substantive data are not available to support these hypotheses, nor are the exact location and nature of the lesion(s) within the lung known. It seems clear that these basic steps in elucidating the pathogenesis of EIPH must be undertaken if a rational clinical approach to the management of EIPH is to be proposed with any certainty.

This preliminary survey of EIPH in racing Thoroughbreds has served to highlight not only the extent of this clinical entity, but to focus attention more directly on the lung as the source of the hemorrhage.

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Effect of Airway Disease on Blood Gas Exchange in Racehorses

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Inflammatory Airway Disease (IAD), exercise-induced pulmonary hemorrhage (EIPH), and upper airway obstruction (UAO) are common respiratory tract diseases that can decrease performance. The purpose of this retrospective study was to compare bronchoalveolar lavage fluid cytology and arterial blood gas analysis during a treadmill test by poorly performing racehorses presented to Purdue University. One hundred thirty-two horses with a history of poor performance were included in this study. Ten horses with no history or diagnosis of EIPH, IAD, or UAO served as controls. Horses were evaluated by rhinolaryngoscopy for upper airway abnormalities and underwent a standardized treadmill test, and samples were collected for blood gas analysis. Horses with IAD or EIPH had a more severe exercise-induced hypoxemia, (mean ± SD; 84.8 ± 1.5 and 86.0 ± 1.7 mm Hg average Pao_2, respectively), than horses in the control group (92.8 ± 2.1 mm Hg). The average Pao_2 of horses with only UAO (88.3 ± 3.3 mm Hg) was not significantly different from control horses. Gas exchanges were the most severely impaired in horses affected with both EIPH and UAO because they exhibited the lowest Pao_2 and highest $Paco_2$ values (66.5 ± 15.2 and 52.2 ± 6.3 mm Hg, respectively).

Key words: Hemorrhage; Inflammation; Lung; Obstruction; Respiration; Ventilation.

Various diseases involving upper and lower airways have been associated with poor performance by racehorses. Inflammatory airway disease (IAD) is a common respiratory disease in young performance horses and is characterized by exercise intolerance, cough, and evidence of various amounts of mucus in the airways on endoscopic evaluation.[1,2] An initial diagnosis of IAD can be made on the basis of the horse's history, physical examination, and clinical signs. However, the diagnosis of IAD can be missed because clinical parameters of affected horses are usually within normal limits and there is no evidence of increased respiratory effort at rest. To confirm the diagnosis, additional diagnostic tests, including endoscopic evaluation, cytologic evaluation of the tracheal wash, and bronchoalveolar lavage fluid (BALF), and lung function tests may be performed.[1]

Horses affected with exercise-induced pulmonary hemorrhage (EIPH) might have evidence of blood in the trachea when evaluated endoscopically. The presence of blood in the trachea has been implicated in decreased performance[2]; however, studies have not been able to identify a relationship between the amount of blood present in the trachea and a horse's finishing position in a race.[3,4] Bleeding originates from the caudodorsal area of the lung and results from stress failure of pulmonary capillaries.[5] An endoscopically guided BALF aspiration enables collection of a sample from the region of interest. A BALF cytologic evaluation accurately reflects the cell population in the terminal airways and alveolar spaces, which aids in the diagnosis of the disease.[6,7] The presence of hemosiderophages in the BALF and tracheal wash fluid is diagnostic of EIPH if no other cause of pulmonary hemorrhage can be identified. Hemosiderophages will remain present in the BALF and tracheal wash for up to 3 weeks and possibly longer after an episode of EIPH.[8]

Upper respiratory tract obstruction (UAO) in racehorses is another common cause of exercise intolerance or decreased performance. The most common UAO are laryngeal paralysis, dorsal displacement of the soft palate, epiglottic entrapment, and dorsal pharyngeal collapse. Other causes that have been implicated are arytenoid chondritis, vocal fold collapse, granulomas, epiglottic hypoplasia, subepiglottic cysts, and nasopharyngeal masses.[9] Diagnosis of UAO is based on endoscopic examination.

Endoscopic evaluations reveal a high prevalence of diseases such as IAD, EIPH, and UAO in racehorses. The objective is to determine whether the findings are responsible for the abnormal performance reported by an owner or trainer. These respiratory diseases negatively affect performance primarily by obstructing airways and impairing gas exchanges and their effects are maximal during strenuous exercise.[10–12] Our hypothesis was that horses with upper and lower airway diseases would have impaired gas exchanges during high-speed treadmill exercise, which would result in more severe exercise-induced arterial hypoxemia, hypercapnia, or both when compared with a control group of healthy racehorses.

The purpose of this study was to compare BALF cytology and arterial blood gas analysis during a standardized treadmill test (STT) in racehorses diagnosed with IAD, EIPH, and UAO with values obtained in healthy racehorses.

Materials and Methods

One hundred thirty-two horses were examined during treadmill exercise between January 1, 1996, and December 31, 2001, and were included in this study. Of these, 122 horses were diagnosed with respiratory disease. Ten horses with no history or diagnosis of EIPH, IAD, or UAO were used as controls. All horses were evaluated on the basis of CBC, serum biochemistry profile, airway endoscopic evaluation, STT, BALF cytology, measurement of blood lactate, blood gas (BG) analysis (Pao_2, $Paco_2$), and serum creatine phosphokinase (CPK) activity before and 6 hours after exercise.

The testing protocol has been previously described[10] and included blood work, BALF cytologic evaluation, and treadmill training on day 1 and additional training, treadmill test (dynamic upper airway endoscopy, BG, blood lactate), tracheobronchial endoscopy 1 hour after ex-

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ercise, and CPK 6 hours after exercise on day 2. An institutional animal care and use committee approved all procedures. A client consent form was signed by the owner at the time of enrollment in the study.

Control horses were clinically normal on the basis of physical examination; lameness evaluation; respiratory tract endoscopy before, during, and 1 hour after treadmill exercise; CBC; and serum biochemistry profile. According to their trainer, these horses were racing satisfactorily and finishing in the top 4 during their last 4 races.

Horses with EIPH had endoscopic evidence of blood in the trachea 1 hour after treadmill exercise. Horses with IAD had moderate to large amounts of mucopurulent secretions present in the trachea 1 hour after treadmill exercise. Upper airway obstruction was detected by endoscopic examination during exercise tests.

BALF Cytology Procedure

The animals were sedated with xylazine hydrochloride (0.4–0.6 mg/kg of body weight) or detomidine (0.02 mg/kg IV) and butorphanol (0.05 mg/kg IV). Lidocaine 0.2% solution was instilled in the airways as the endoscope was advanced into the caudodorsal lung area. A volume of 250 mL of warm 0.9% sterile saline solution was administered. The BALF recovered by suction was placed in a container on ice. All samples were processed within 20 minutes of collection. Specimens were prepared for cytology by cytocentrifugation and stained with Wright's stain. Cells were counted manually.

Standardized Treadmill Test

The treadmill test consisted of a warm-up period of 4 minutes of walking at 2 m/s and 4 minutes at 4 m/s. The treadmill was then stopped, and a videoendoscope was passed through the right nostril and positioned to visualize the caudal nasopharynx and larynx. The treadmill was started and a preprogrammed test was initiated. This test consisted of 120 seconds at 4 m/s, followed by five 1-minute incremental speed steps at 6, 8, 10, 11, and 12 m/s. The incline was set at 10% for Thoroughbreds and 5% for Standardbreds. The treadmill was stopped when the horse showed signs of fatigue and could not keep up with the treadmill speed.

Blood Gas Analysis

The transverse facial artery was catheterized and an arterial blood sample was collected in a heparinized syringe in the last 15 seconds of each step of the STT. Blood temperature was measured by a thermocouple probe inserted ~5 cm from the catheter hub and used for temperature correction of gas tensions by the analyzer.[Ref] A 2-mL aliquot of blood was placed in a dry tube for lactate measurement by a spectrophotometric method. The syringes were then capped and placed on crushed ice for blood gas analysis within 15 minutes of collection.

Statistical Analysis

Normal distribution of cytology, blood gas, and blood biochemistry variables was assessed by the Wilks-Shapiro test.[b] Most BALF cytology variables were nonnormally distributed, so between-group comparisons were assessed by the Kruskal-Wallis analysis of variance tests. All CBC, blood gas, and biochemistry data were normally distributed. For each horse, the minimum Pao_2, the maximum $Paco_2$, and the average Pao_2, $Paco_2$, and blood lactate concentration during the 1st 4 steps of the STT were compared between groups by analysis of variance.[c] Blood gas values at each step of the STT were compared between groups by repeated-measures analysis of variance. When significant main group effects were detected, least significant difference post hoc tests were performed to determine specific group differences. Results were expressed as median (1st–3rd quartiles) for BALF data and mean ± SD for the other data. The significance level was established at $P \leq .05$.

Results

Eighty-three of the 132 horses (62.9%) were Standardbreds, 42 of 132 (31.8%) were Thoroughbreds, and 7 of 132 (5.3%) were from other breeds. Forty-eight out of 132 horses (36.4%) were <3 years old, 44 of 132 were 3–6 years of age (33.3%), and 40 of 132 were >6 years of age (30.3%). All horses enrolled in this study completed at least the 1st 4 steps of the STT (4, 6, 8, and 10 m/s). Eight groups were identified: 28 of 132 (21.2%) had IAD; 20 of 132 (15.2%) had EIPH; 12 of 132 (9.1%) had IAD and EIPH; 10 of 132 (7.5%) had IAD and some type of UAO; 12 of 132 (9.1%) had EIPH and some type of UAO; 38 of 132 (28.8%) had only a diagnosis of upper respiratory tract obstruction; 2 of 132 (1.5%) were diagnosed with a combination of IAD, EIPH, and UAO; and 10 of 132 (7.5%) horses were healthy (control group). Analyses of BALF and blood gas data revealed no significant differences between horses diagnosed with IAD only and horses with IAD and upper respiratory tract obstruction; therefore, data from these 2 groups were pooled. Data from horses with IAD and EIPH and horses with IAD, EIPH, and UAO were also pooled for the same reason. Pooling of data did not change results of the analyses. Adjusting for age did not change the outcomes of the study.

BALF Cytology

Total nucleated cell counts and percentage of hemosiderophages were not significantly different between groups (Table 1).

Lymphocyte percentage and absolute number were significantly ($P \leq .05$) higher in horses with IAD than in control animals.

Alveolar macrophage percentage was significantly higher in control horses and horses with a combination of EIPH and UAO than in horses with IAD ($P < .03$). Alveolar macrophage cell counts were not significantly different between groups.

Four horses from the IAD group had a high percentage of eosinophils (>1%, n = 2) or mast cells (>2%, n = 2). However, percentage or absolute numbers of eosinophils, mast cells, and epithelial cells were not different between groups.

Blood Gas Tensions

Pao_2 decreased significantly as speed increased ($P < .0001$). There were no difference in Pao_2 at each step of the STT ($P = .062$). The average Pao_2 and minimum Pao_2 during the 1st 4 steps of the STT were significantly lower in horses with IAD (84.8 ± 1.5, 71.7 ± 6.7 mm Hg), EIPH (86.0 ± 1.7, 74.3 ± 5.0 mm Hg), IAD and EIPH (86.6 ± 2.5, 68.9 ± 4.9 mm Hg), and EIPH in combination with UAO (82.4 ± 2.3, 66.5 ± 15.2 mm Hg) when compared with control horses (92.8 ± 2.1, 83.2 ± 8.5 mm Hg; $P = .026$ and $P = .001$, respectively). However, the average Pao_2 of horses with only UAO (88.3 ± 3.3 mm Hg) was not significantly different from control horses. Horses diagnosed with IAD or EIPH had a more severe exercise-induced hypoxemia than other horses (Fig 1).

$Paco_2$ increased significantly ($P < .001$) with each ad-

Table 1. Bronchoalveolar lavage fluid cytology analyses from racehorses diagnosed with various respiratory conditions. Results are expressed as median (1st quartile–3rd quartile).

	Cell Count (cells/μL)					
	Control (n = 10)	IAD (n = 25)	EIPH (n = 14)	IAD + EIPH (n = 10)	EIPH + UAO (n = 9)	UAO (n = 10)
Total nucleated cells	436 (326–536)	491 (394–634)	741 (449–1,002)	584 (269–646)	514 (348–620)	425 (277–519)
Neutrophils	8 (4–18)	34 (8–82)	28 (22–56)	24 (7–45)	13 (9–24)	17 (8–24)
Lymphocytes	97[a] (63–156)	185[b] (125–228)	217 (113–351)	154 (104–221)	132 (87–164)	151 (59–192)
Macrophages	294 (246–349)	279 (192–347)	474 (217–572)	286 (245–425)	350 (279–415)	321 (254–343)
Neutrophil %	2.0 (1.0–4.0)	5.0 (3.0–13.0)	4.5 (3.0–6.0)	4.0 (1.7–8.0)	2.0 (2.0–4.5)	3.5 (2.0–5.2)
Lymphocyte %	25.0[a] (15.0–29.2)	37.0[b] (30.8–48.5)	36.0 (28.0–48.0)	34.5 (21.2–39.0)	26.0 (19.5–30.2)	29.5 (17.7–36.2)
Macrophage %	65.0[a] (63.0–76.7)	54.0[b] (44.0–58.0)	49.0 (49.0–67.0)	60.0 (50.5–68.2)	72.0[a] (63.0–78.0)	66.5 (50.2–75.5)
Hemosiderophage %	13.5 (4.7–17.2)	8.0 (1.0–28.0)	48.0 (27.0–52.0)	28.5 (7.5–50.0)	34.6 (19.0–60.0)	2.0 (1.5–20.5)

IAD, inflammatory airway disease; EIPH, exercise-induced pulmonary hemorrhage; UAO, upper airway obstruction.
[ab] Data on a line with different superscripts are significantly different from each other ($P \leq .05$).

ditional step of the STT, but there were no significant differences between groups at each step of the test (Table 2). However, the maximum $PaCO_2$ reached by horses with a combination of EIPH and UAO (52.2 ± 6.3 mm Hg) was significantly higher than the values reached by other groups (Table 2; $P = .03$)

Blood Lactate

Blood lactate concentration increased significantly during the 1st 4 steps of the STT in all groups (1.3 mmol/L at 4 m/s, 1.7 mmol/L at 6 m/s, 2.9 mmol/L at 8 m/s, 6.3 mmol/L at 10 m/s); however, there was no significant difference in blood lactate concentration between groups. Speed for a lactate concentration of 4 mmol/L and peak lactate achieved during STT were not statistically different between groups.

Discussion

Data from this study suggest that racehorses with IAD and EIPH have impaired lung function, as illustrated by the more pronounced degree of exercise-induced hypoxemia they experience during an STT.

BALF Cytology

Bronchoalveolar lavage was performed before, rather than after, exercise on the basis of previous findings that indicated a better ability to differentiate between control horses and horses with IAD.[10] Bronchoalveolar lavage fluid in horses with an endoscopic diagnosis of IAD was characterized by neutrophilia and lymphocytosis, as previously reported.[10,13] Others have reported that BALF from horses with IAD is characterized by an increase in total nucleated cell counts, with the predominant inflammatory cells being neutrophilic, eosinophilic, or mastocytic.[13,14] In this study, some horses with accumulation of mucus in the trachea had a high percentage of eosinophils (>1%, n = 2) or mast cells (>2%, n = 2). However, some horses with excess mucus visible by endoscopy had normal BALF (n = 8) and others, with no detectable mucus accumulation, had inflammatory BALF (n = 14). These findings suggest that a combination of BALF cytologic evaluation performed before exercise and endoscopy of the airways after exercise are helpful to identify horses with IAD.

Distinct pathophysiologic mechanisms could be responsible for the observed differences in BALF phenotypes. Predominance of mast cells and eosinophils in BALF suggest an allergic mechanism (type I hypersensitivity) as the cause of airway inflammation.[15,16] BALF neutrophilia and lymphocytosis might result from persistent viral respiratory tract infection or chronic inhalation of airborne particles.[13]

Exercise-induced pulmonary hemorrhage can be diagnosed by the presence of variable amounts of blood in the trachea after exercise, but a more sensitive indicator is the presence of hemosiderophages in respiratory secretions because quantification of the amount of blood visible in the trachea is subjective.[17] Quantification of red blood cells in a BALF sample is also helpful when there is no endoscopic evidence of bleeding in the trachea.[18] In this retrospective study, animals were classified into the EIPH group by the presence of blood in the trachea after exercise. It is important to remember that not all horses experiencing pulmonary hemorrhage will have evidence of blood in the trachea after strenuous exercise. Indeed, prior episodes of pulmonary hemorrhage in control and IAD horses were revealed by the presence of hemosiderophages in their BALF (13.5 and 8.0% median values, respectively). Our data suggest that racehorses with blood visible in the trachea within 1 hour of a STT have more severe EIPH than horses that do not have blood detectable by endoscopy. For this reason, the presence of hemosiderophages is probably a more sen-

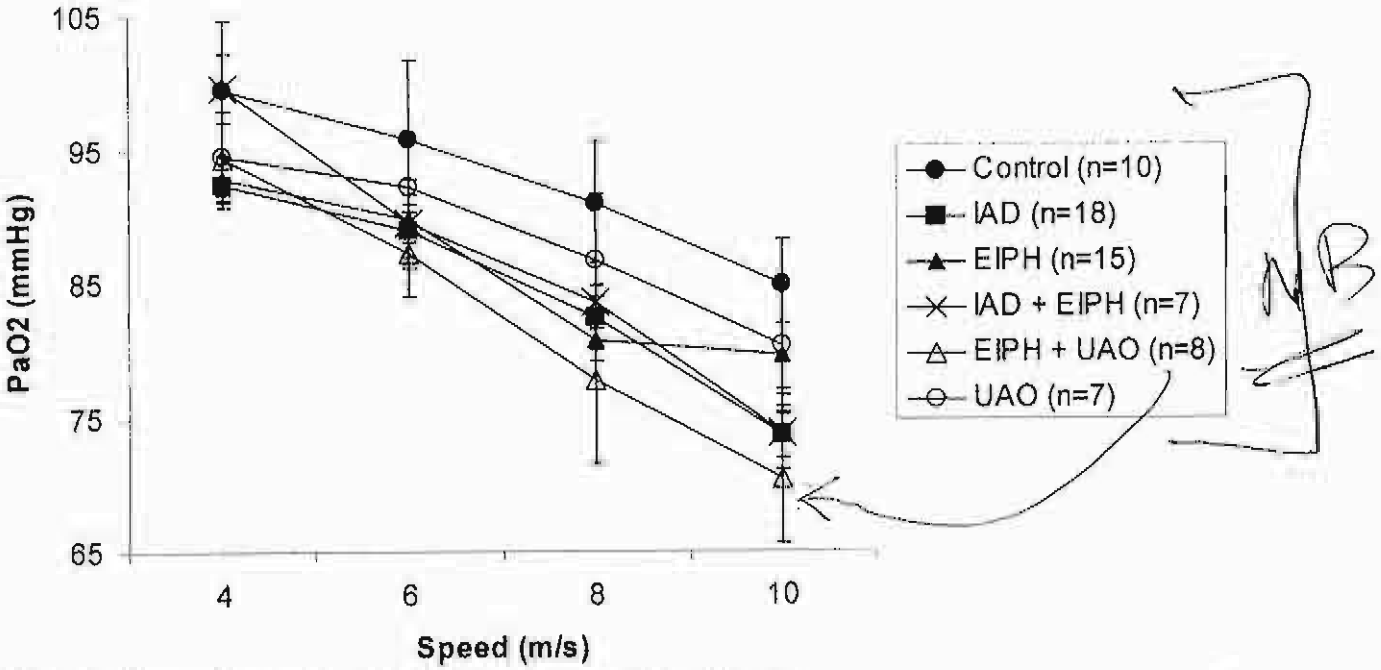

Fig 1. Pao_2 values (mean ± SD) during the 1st 4 steps of the standardized treadmill test. EIPH, exercise-induced pulmonary hemorrhage; IAD, inflammatory airway disease; UAO, upper airway obstruction.

sitive means of determining the severity of EIPH in horses than is endoscopy. Exercise-induced pulmonary hemorrhage is not homogeneous throughout the lung, and with BALF cytology, just a small portion of the lung is being evaluated. For this reason, the endoscope used to perform the BALF cytologic evaluation was wedged in bronchi located in the caudodorsal portion of the lungs, where the bleeding originates. However, regional variation of the percentage of hemodiserophages in BALF is unknown. Such knowledge would improve our ability to diagnose and assess the severity of EIPH.

IAD and EIPH are highly prevalent in racehorses and have been identified as potential causes of impaired performance, especially in younger horses.[2,19] The relationship between airway inflammation and EIPH is a matter of debate. A previous study found an association between IAD and EIPH in young Thoroughbreds in training.[18] It is important to take into consideration that the presence of blood in the airways can in itself cause lung inflammation.[20] Others have proposed that lower airway inflammation plays a role in EIPH pathogenesis.[21] This study found that the majority of horses diagnosed endoscopically with EIPH did not exhibit signs of airway inflammation on the basis of BALF cytology. Further investigation is required to elucidate the relationship between these diseases and their effects on performance.

Blood Gases

In all horses, the degree of exercise-induced arterial hypoxemia worsened as treadmill speed increased throughout the STT, which is consistent with previous reports.[11,12] In addition, we observed that horses with IAD exhibited a significantly lower Pao_2 than control horses during the 1st 4 steps of the STT as previously reported by Couetil et al.[10] Presumably, airway inflammation impaired ventilation-perfusion matching because $Paco_2$ was similar between groups. On the basis of these findings, we recommend treat-

Table 2. $Paco_2$ (mean ± SD) at 4, 6, 8, and 10 m/s during standardized treadmill test (STT).

		$Paco_2$ (mm Hg)				
	n	4	6	8	10	Max[a]
Control	10	40.2 ± 2.66	39.8 ± 5.92	41.2 ± 4.78	44.6 ± 3.37	45.7 ± 2.6
EIPH	17	40.53 ± 2.72	40 ± 4.34	41.38 ± 6.16	43.94 ± 5.45	46.7 ± 4.1
IAD + EIPH	7	38.29 ± 6.23	41.93 ± 3.44	40.89 ± 6.77	43.67 ± 5.53	46.7 ± 2.7
EIPH + UAO	8	37.15 ± 7.44	42.05 ± 3.64	46.41 ± 6.35	48.6 ± 2.49	52.2 ± 6.3[b]
IAD	21	39.84 ± 10.86	40.56 ± 4.61	43.17 ± 3.58	42.61 ± 5.09	47.3 ± 4.4
UAO	6	33.27 ± 8.03	40.35 ± 1.63	37.72 ± 8.57	42.35 ± 5	46.0 ± 3.3

EIPH, exercise-induced pulmonary hemorrhage; IAD, inflammatory airway disease; UAO, upper airway obstruction.

[a] Maximum $PaCO_2$ reached during STT.

[b] Significantly different from other groups ($P = .03$).

ing racehorses with IAD; however, whether current therapies will restore normal gas exchange and performance level has not been demonstrated yet. Horses affected with both EIPH and UAO had a significantly lower Pao_2 level, when compared with horses with EIPH or UAO alone (Fig 1). This lower Pao_2 in horses with both conditions might have been secondary to a relative hypoventilation, as suggested by the significantly higher maximum $Paco_2$ reached by these animals during STT. Also, UAO results in higher pressure swings in alveolar spaces and across capillary beds.[22] Higher pressure swings are more likely to lead to stress failure of pulmonary capillaries and result in EIPH.[23] Therefore, worsening of EIPH in horses that also developed UAO during STT might have impaired gas exchanges further. Successful treatment of UAO is therefore paramount to the management of EIPH when both conditions are detected in a horse. Blood gas data in horses with UAO were not different from controls, and 90% of those horses had <20% of hemosiderophages present in BALF. Therefore, horses with lower airway disease and ≥20% hemosiderophages are likely to have impaired pulmonary gas exchanges, which might adversely affect their performance.

The degree of exercise-induced hypoxemia worsens as a horse's maximum oxygen consumption increases.[11] Other factors such as quality of the horse and level of training are known to affect maximum oxygen consumption and could have confounded our blood gas results.[24,25] However, there was no significant difference in blood lactate concentration and racing times between groups (data not shown), suggesting comparable fitness levels and intrinsic quality of horses between groups. Nevertheless, we cannot exclude the possibility that there could have been large differences in maximal oxygen uptake between the control group and other groups of horses with respiratory disease, which could have resulted in the observed differences in level of exercise-induced hypoxemia.

The finding that horses with UAO had blood gas values similar to control horses was unexpected. Laryngeal hemiplegia, for example, is known to impair ventilation during high-speed exercise and result in a worsening of exercise-induced hypoxemia and hypercapnia.[26] Horses with laryngeal hemiplegia tend to experience progressive worsening of gas exchanges as they exercise. Other types of UAO, such as dorsal displacement of the soft palate, develop more suddenly during high-speed exercise and result in rapid fatigue and stopping of the horse. Because blood samples in this study were collected during the last 15 seconds of each step of the STT, the lack of blood gas abnormalities is likely because horses developing sudden UAO stop quickly and do not complete the step. Therefore, blood is rarely collected while the horse exhibits UAO. This speculation is supported by the finding that, among the 40 horses diagnosed with UAO in this study, 14 exhibited laryngeal hemiplegia and 26 dorsal displacement of the soft palate.

Horses affected with IAD as defined by the presence of mucus in the trachea after exercise are characterized by a neutrophilic and lymphocytic inflammation of the airways, as demonstrated by examination of the BALF. Horses affected by EIPH, as defined by the presence of blood in the trachea after exercise, tend to have a higher percentage of hemosiderophages in the BALF than horses with other respiratory conditions, including UAO. This study also indicates that horses with IAD and EIPH can exhibit some degree of pulmonary gas exchange impairment. Horses affected with UAO and EIPH appear to have the most severe gas exchange impairment. The findings concerning gas exchanges will require further confirmation by studies examining the effect of both aerobic capacity and airway diseases on exercise-induced blood gas changes. Comprehensive evaluation of racehorses by BALF cytology and STT with dynamic airway endoscopy are valuable aids in the diagnosis of causes of decreased performance. Interpretation of arterial blood gas measurement must be used cautiously until future studies validate its use.

Footnotes

[a] Ciba-Corning 238 Blood Gas Analyzer, Norwood, MA
[b] Statistix version 8.0 Analytical Software, Tallahassee, FL
[c] SPSS version 11.5, SPSS Inc, Chicago, IL

Acknowledgments

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ATTACHMENT #4

3/ WATER WITHHOLDING AND EIPH:

American Horsemen long knew that horses run better and bleed less if water was withheld before racing. A four hour water withholding produces a 12 lb or so weight loss, historically a standard pre-race procedure. This long standing field observation of American Horsemen is now scientifically validated, as follows:

4/ OPTIMIZING WATER "WITHHOLDING": LASIX:

When injectable Lasix became available in the late sixties horsemen found that horses bled less and ran better when administered Lasix pre-race. Administration of Lasix can increase the water withholding effect to up to about a 28 pound weight loss, Attachment #4.

THE AMERICAN HORSEMEN'S STORY: BOTTOM LINE

- American horsemen early identified EIPH as a limiting factor in racing performance.
- Correctly identified water withholding as a mechanism of alleviating EIPH.
- Correctly identified facilitation of the water withholding effect with Furosemide.
- 50 or so years later, their Horsemen's perceptions have been scientifically validated.

ATTACHMENT #5

5/ LASIX AND URINE TESTING: THE FOUR HOUR LASIX RULE:

Questions arose concerning the effects of Lasix on urinary detection of other substances. The urinary dilution effect was first shown to be transient, giving rise to the four hour 250 mg IV / rule, which rule led to a need for detention barns, Attachment #5.

J. Equine Vet Sci, 1:203-207, 1981

THE PHARMACOLOGY OF FUROSEMIDE IN THE HORSE
V. The Duration of Reduction of Urinary Concentration of Drugs

Joan Corbie MSc; Thomas Tobin and Thomas Tobin M[illegible] PhD, MRCVS

SUMMARY

The administration of furosemide to horses in IVdoses of 0.5 mg/kg or less reduced drug concentrations in urine for less than 4 hours. The most prolonged reduction observed was that of the glucuronide metabolite of morphine, which required three hours post-dosing to return to control. Urinary concentrations of phenylbutazone were not significantly different from control by two hours post-dosing, while urinary concentrations of fentanyl appeared to return to normal within about two and one-half hours of dosing. Other experiments showed that blood levels of morphine were not significantly reduced by furosemide treatments.

INTRODUCTION

Furosemide (Lasix®)* is a potent, high ceiling diuretic which is the recommended treatment for epistaxis or exercise induced pulmonary hemorrhage (EIPH) in the horse. Since at least 43% of racing horses have some degree of EIPH, a number of racing jurisdictions have allowed the pre-race use of furosemide in horses.[5] However, the principal problem with the approval of furosemide for use in racing horses is that it acts to dilute out certain drugs and drug metabolites in equine urine. This dilution is important because it might reduce the efficacy of testing for some drugs.

Because furosemide is a very short acting drug in the horse, these diluting effects are quite transient. Therefore, one approach to the problem of the diluting effect of furosemide in the horse is to avoid taking urine samples during the period of the diluting effect.

Reviewing the problem in 1977, Drs. Gabel, Tobin, Ray, Maylin and the other members of the Veterinary Chemists Advisory Committee to the National Association of State Racing Commissioners concluded that "furosemide administered more than four hours before race time does not significantly reduce the ability to detect those drugs studied to date."[4] These are the only published recommendations on the duration of diluting effects of furosemide. However, since these workers based this 4-hour estimate on research done using 1.0 mg/kg doses of furosemide, we elected to investigate the duration of the diluting effect of furosemide after administration of smaller (0.5 mg/kg or less) doses of this drug.

It is important that the duration of the diluting effect of prophylactic doses of furosemide be determined. Although the diuretic effects of furosemide are relatively short-lived, the duration of other pharmacological actions of this drug may be more prolonged. Therefore, to enable the use of this drug in horses without undue interference with drug testing, the minimal period for which small, prophylactic doses of furosemide produce their diluting effects should be accurately known. Preliminary reports of this work have been communicated

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MATERIALS and METHODS

Animals: Mature thoroughbred and standardbred horses between 400 and 550 kg were used. The animals were kept at pasture until the day of an experiment when they were housed in box stalls and allowed hay and water *ad libitum*.

All drugs were injected intravenously into the left jugular vein. Blood samples were drawn from the right jugular vein and urine was obtained by bladder catheterization.

Experiment 1:

Phenylbutazone and Furosemide: Three mares were dosed with 4.4 mg phenylbutazone[b] per kg body weight. Urine samples were collected at intervals for 24 hours. The following week, the procedure was repeated on the same animals except that 0.385 mg furosemide per kg body weight was injected intravenously immediately after collection of the 2-hour sample. All samples were stored at 4° C until the analysis was performed.

Two hundred µl of each urine sample was mixed with 2 ml hexane:dichloromethane (2:1) and 2 ml sodium acetate buffer (pH 4.5). The tubes were rotoracked for 5 minutes and centrifuged at 1150 g, 2° C for 15 minutes. A 1 ml aliquot of the solvent phase was evaporated to dryness under nitrogen. The residue was redissolved in 50 µl hexane, 2 µl of which was injected into a Varian 2700 gas chromatograph equipped with a tritium on scandium electron capture detector. Separation was done on a 6-foot glass column packed with 3% OV-101 maintained at 249° C.

Experiment 2:

Fentanyl and Furosemide: Four horses were given 0.001 mg fentanyl[c] per kg body weight and urine samples were collected at intervals (Figure 2) during the next 24 hours. The following week, the same horses were again dosed with fentanyl. Immediately after the 30-minute urine sample was obtained, the animals were given 0.5 mg furosemide per kg body weight. All samples were stored at -20° C for later analysis.

Levels of fentanyl in the urine were determined by a radioimmunoassay method utilizing a commercially available kit.[d] The procedure has been described elsewhere.[1] Briefly, 0.05-0.5 ml urine was incubated with radioactive tracer and antiserum for 2 hours at pH 7.4. Fentanyl in the sample competed with the ^{3}H-fentanyl for binding sites on the fentanyl antibody. After formation of

[b]National Laboratories Corp., Somerville, N.J.
[c]McNeil Laboratories, Fort Washington, PA.
[d]Institut National des Radioelements, Fleurus, Belgium.

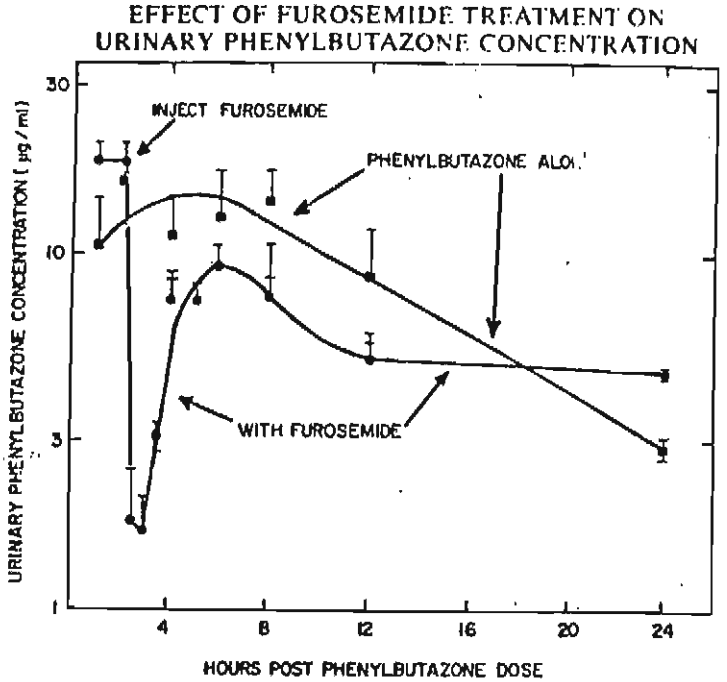


Figure 1. Effect of furosemide treatment on urinary phenylbutazone concentration. Urinary phenylbutazone concentrations following IV dosing with phenylbutazone alone are shown by the solid squares (■-■) and following IV dosing with phenylbutazone and furosemide (0.385 mg/kg) are shown by the solid circles (●-●). Vertical bars represent ± SEM. (Reproduced with permission from "Drugs and the Performance Horse," Charles C. Thomas, Publishers, Springfield, Illinois 62717, 1981).

the immunocomplex was completed, bound and free fentanyl were separated by selective adsorption onto dextran-coated charcoal. The charcoal complex was removed by centrifugation and the radioactivity in the supernatant, due to bound ^{3}H-fentanyl, was measured in a liquid scintillation counter.

Experiment 3:

Morphine and Furosemide: Mares were injected IV with 0.1 mg morphine* per kg body weight. Blood and urine samples were collected (Figure 3-5) during the next 6 hours. Six months later, the same horses were again dosed with the same amount of morphine. Following collection of the 1-hour samples, 0.4 mg furosemide/kg body weight was injected. Blood samples were allowed to clot and serum was removed. Serum and urine samples were stored at -20° C until the analysis was performed.

The methodology for the determination of morphine levels in equine biological samples has been previously described. Urine samples were split, 1 portion analyzed for free morphine, while the other portion was subjected to hydrolysis with β-glucuronidase to allow measurement of total morphine. Samples were cleaned by a combination of liquid-liquid extraction and column chromatography. A strong electron capturing compound was formed by the derivatization of the extracted morphine with pentafluoropropionic anhydride (PFPA). Separation was performed on a SP 2250-DB on 100/120 Supelcoport* column at 235° C in a Varian 3700 equipped with a ^{63}Ni electron capture detector.

RESULTS

Urinary levels of phenylbutazone were reduced by an average of 10-fold during peak diuresis.

Experiment 1 — Urinary concentrations of phenylbutazone quickly recovered, there being no significant difference (paired data t-test, $t = 2.370$, $\alpha = 0.05$) between the treated and untreated horses from 2 hours after the horses had been given the diuretic.

Experiment 2 — Horses were dosed with 0.001 mg/kg of fentanyl and this dose was followed 30 minutes later by either furosemide, 0.5 mg/kg, or normal saline. Urine samples were collected at the indicated periods and analyzed for fentanyl equivalents by RIA. Administration of furosemide caused urinary levels of "fentanyl" to fall about 18-fold within 30 minutes of dosing but urinary "fentanyl" levels had returned to control within about 2½ hours of dosing.

Experiment 3 — Morphine (0.1 mg/kg) was administered to 6 horses and followed 1 hour later with 0.4 mg furosemide/kg body weight. Free morphine plus β-glucuronidase releasable morphine (referred to here as total morphine) fell 13-fold within 15 minutes after the diuretic had been given (Figure 3). Total morphine found in the urine rose rapidly from this low point until the levels were not significantly different (paired data t-test, t

*Eli Lilly and Co., Indianapolis, Ind.

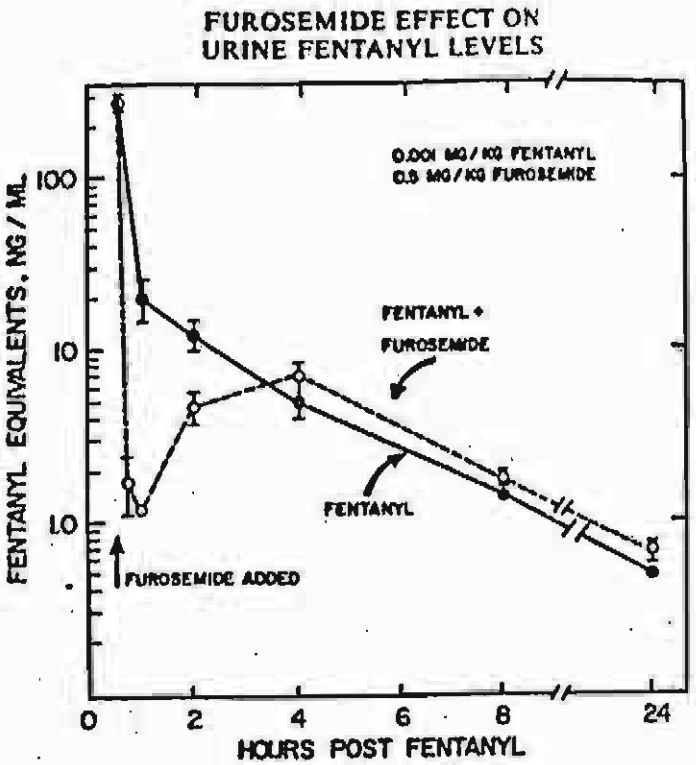


Figure 2. Furosemide effect on urine fentanyl levels. Urinary fentanyl levels were determined by RIA in horses given fentanyl alone (shown by the solid circles, •–•) and given fentanyl and furosemide (shown by the open circles, o–o). Vertical bars represent + SEM. (Reproduced with permission from "Drugs and the Performance Horse," Charles C. Thomas, Publishers, Springfield, Illinois 62717, 1981).

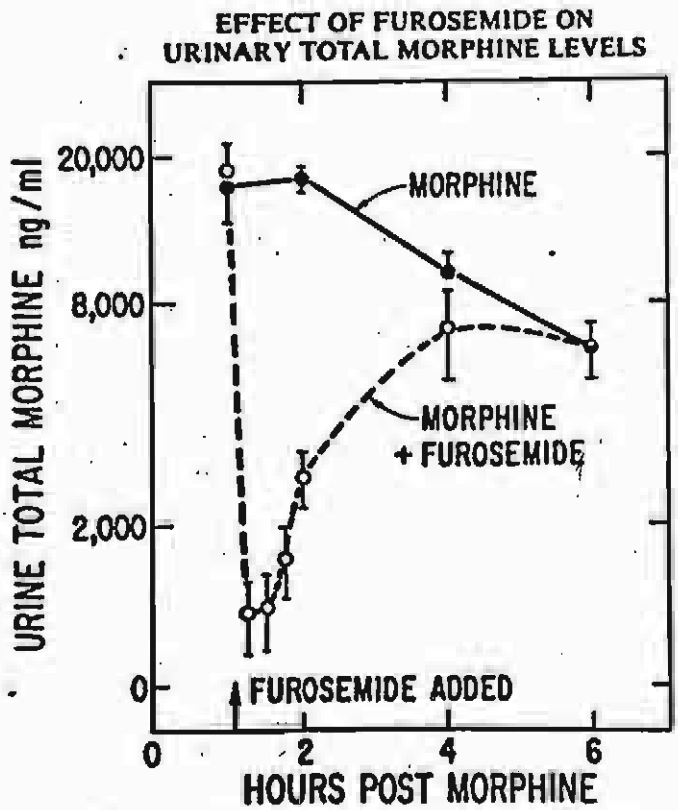
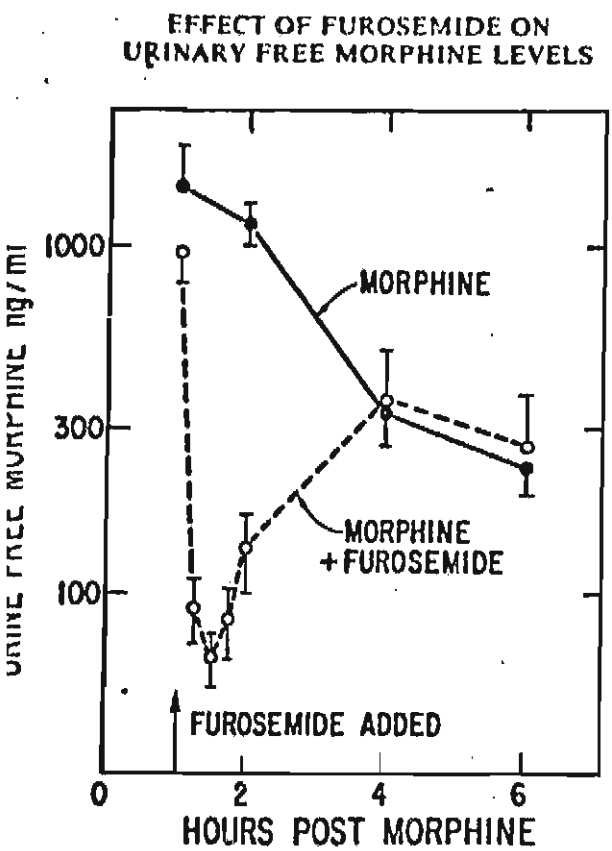


Figure 3. Effect of furosemide on urinary total morphine levels. Horses were dosed with 0.1 mg morphine per kg body weight and urine levels of morphine were analyzed following hydrolysis with β-glucuronidase (shown by the solid circles (•–•). Later, the experiment was repeated but 0.4 mg furosemide/kg body weight was injected 1 hour after the horses



ure 4. Effect of furosemide on urinary free morphine levels. Urine centrations of free morphine in horses dosed with morphine alone shown by the closed circles (•-•) and in the same horses later dosed h morphine and furosemide are shown by the open circles (o-o). tical bars indicate ± SEM.

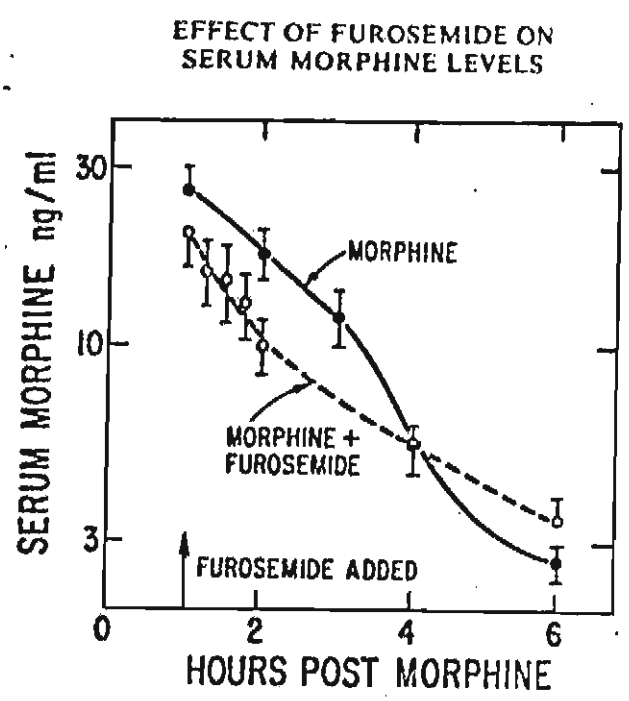


Figure 5. Effect of furosemide on serum morphine levels. Serum morphine levels of 6 horses given 0.1 mg morphine/kg body weight are shown by the closed circles (•-•). Later, the same animals were given the same amount of morphine but were also treated with furosemide (0.4 mg/kg). These serum levels are shown by the open circles (o-o). Vertical bars represent ± SEM.

= 1.026, α=0.05) from control values of furosemide by 3 hours after furosemide administration. Furosemide appeared to have broadly similar effects on total and free morphine levels as seen by comparing Figures 3 and 4.

Serum samples from these same horses were analyzed for morphine and the results are presented in Figure 5. Throughout the entire 5-hour period immediately following furosemide administration, there was no significant difference (2 sample t-test, t = 1.354, α = 0.05) in serum levels of morphine whether or not the horses had received furosemide.

DISCUSSION

Epistaxis, or bleeding from the nose, following exercise has occurred since the earliest days of thoroughbred racing. Historically, the incidence of epistaxis has been low (less than 2%),[3] and a variety of treatments for this condition have been recommended. In the early 1970's, equine practitioners and horsemen began recommending furosemide for the treatment of epistaxis. Since then, it has become the treatment of choice for this condition.

In the late 1970's, work by Pascoe and his associates showed that the classical 2% incidence of bleeding from the nose was a manifestation of a much more extensive problem.[4] Studying horses post-race with a fiberoptic endoscope, Pascoe showed that up to 40% of horses showed evidence of blood in the larynx or trachea post-race. Pascoe therefore renamed the syndrome exercise-induced pulmonary hemorrhage (EIPH)[5] and other work since then has supported Pascoe's observations and conclusions.[6]

The horse appears to be unusually prone to epistaxis, and this predisposition has been attributed to the architecture of the horse's lungs. As a species with poor collateral ventilation at the alveolar level, blockade of the direct entry of air to an alveolus has been theorized to cause blocked alveoli to rupture when the lung expands.[9] Furosemide may act to alleviate EIPH by improving the entry of air into a partially blocked alveolus, either by reducing pulmonary edema or by dilating bronchioles.[9] Alternatively, it may act to reduce epistaxis by reducing congestion.[9] While scientific studies on the efficacy of furosemide for EIPH are not available, there is a clear medical rationale and anecdotal clinical experience to justify its use in EIPH.

The only technical problem with the approval of furosemide for use in racing horses is its ability to dilute out certain drugs in equine urine.[7,8] The maximum duration of this dilution effect must, therefore, be determined to allow evaluation of its potential for use in racing. In this investigation the duration of the diluting effect after small doses of furosemide was determined, and the amount of apparent dilution of post-race urine samples associated with pre-treatment with furosemide was determined.

both the duration and extent of the diluting effects of furosemide would be reduced if the dose of furosemide was reduced. Thus, one might expect that the duration of the dilution effect would be considerably less after a dose of furosemide in the order of 0.4 mg/kg, i.e. the dose commonly used in the treatment of epistaxis, than after 1 mg/kg, the manufacturer's recommended dose for pulmonary edema as was reported earlier.[7,8] In general, this expectation has been borne out by the results obtained.

Both the duration and extent of the dilution after the lower doses of furosemide (less than 0.5 mg/kg) were less than those reported in earlier experiments. Thus, 1 mg/kg of furosemide IV produced about a 40 to 50-fold dilution of the urinary concentrations of both phenylbutazone and the major glucuronide metabolite of pentazocine in a previously reported work.[8] Reducing the dose, however, produced a correspondingly smaller maximal dilution effect, about 10-fold for phenylbutazone, about 18-fold for fentanyl, and about 13-fold for morphine.

Similarly, the duration of the dilution effect was markedly reduced. While the effect lasted for longer than 4 hours when the dose of furosemide was 1.0 mg/kg,[7,8] reducing the dose of furosemide reduced the duration of the effect to about 3 hours or less. For example, the dilution effect when phenylbutazone was used was about two hours, and three hours or less when morphine or fentanyl were the test drugs. Four hours is therefore a conservative time pre-race to administer a prophylactic dose of furosemide. In fact, these data suggest that the drug could be administered in less than 0.5 mg doses as close as three hours prior to sampling with little effect on drug detection in post-race urine samples.

These data further suggest that the diluting effect of furosemide on urinary drug concentrations in actual practice may be relatively small. In other experiments, we measured and compared the concentration of phenylbutazone and its metabolites in the post-race urines of horses with and without furosemide pre-treatment.[9] These data show that the apparent dilution of phenylbutazone and its metabolites in equine urine amounts to less than a 50% reduction in drug concentration in equine urine. Since all practical analytical methods have a margin of safety of much greater than 50%, this reduction in concentration is unlikely to be forensically significant.[9]

It is highly unlikely that less than 0.5 mg furosemide per kg body weight will significantly affect the detection of drugs in blood. This is because small doses of furosemide lead to the elimination of only about 4 liters of fluid from the horse, which is between 1 and 1.5% of the body water of a horse. In general, therefore, furosemide at less than 0.5 mg/kg is unlikely to lead to the elimination of more than 1.5% of the drug in a horse, which is unlikely to be a forensically significant amount.

Further, the fraction of the dose of drug eliminated in the urine in response to furosemide will generally be a lot less than 1.5%. This is because most drugs, either acidic or basic, have an apparent volume of distribution in the horse of much greater than total body water. For example, the tranquilizer acepromazine distributes in the horse in a manner equivalent to its distribution in a volume of over 800,000 liters. The proportion of the total amount of drug eliminated after a dose of furosemide will, therefore, in many cases be only a fraction of 1% of the dose present in the horse at the time of treatment with furosemide.

As a practical matter, therefore, the results reported here show that the diluting effects of doses of furosemide less than 0.5% mg/kg administered IV are essentially over within about three hours of dosing. These data, therefore, support the original conclusions of Gabel, Tobin, Ray and Maylin that dilution is not likely to be significant if the drug is administered four hours before post-time.[4] These results further suggest that four hours prior to post-time may be a relatively conservative time restraint to put on administration of this drug if the dose administered is equal to or less than 0.5 mg/kg.

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ATTACHMENT #6

6/ THE LASIX PLASMA THRESHOLD AND URINARY SPECIFIC GRAVITY TEST:

In the early eighties I was asked by the Kentucky Horsemen's Benevolent and Protective Association to identify a blood level of Lasix that would be equivalent to the four hour rule. We ran a 47 horse study and showed that 1 in 1,000 horses would be expected to exceed 30 ng/ml of Lasix in plasma at 4 hours post 250 mg of Lasix IV (Chay et al 1893, Attachment #6.). This threshold was first introduced in Oklahoma in about 1987, where they set the plasma cut-off at 60 ng/ml. This cut-off was later adjusted upward to 100 ng/ml and, with an added 1.010 urinary specific gravity screen, pioneered by Dr. Richard A. Sams at Ohio State, became the national rule.

Lasix is strictly regulated, now often administered by a third party veterinarian at 4 hours prior to post by rapid IV injection into the jugular vein. Under current rules and modern technologies, including the urinary specific gravity test introduced by Dr. Sams, Lasix does not, to my knowledge, in any way, interfere with drug testing, as I understand was presented by Dr. Richard A. Sams at the June 13-14, 2011, New York Racing Association (NYRA) first International Summit on Race Day Medication at Belmont Park in Elmont, N.Y., Attachment #6.

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THE PHARMACOLOGY OF FUROSEMIDE IN THE HORSE

V. Pharmacokinetics and Blood Levels of Furosemide after Intravenous Administration

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ABSTRACT:

Studies were undertaken to determine blood levels of furosemide in horses after 0.5- and 1.0-mg/kg doses administered iv. Analyses indicated that the pharmacokinetic parameters were dose independent and best described by a three-compartment open model. The α-, β-, and γ-phase half-lives of 5.6, 22.3, and 158.5 min, respectively, were observed after the 0.5-mg/kg dose. Similarly, the respective half-lives after the 1.0-mg/kg dose were 5.8, 24.1, and 177.2 min. After a 0.5-mg/kg dose of furosemide, population frequency distributions were evaluated at 1 hr and 4 hr post-drug administration. At 1 hr after dosing, the blood levels of furosemide in 30 horses were normally distributed. The mean plasma level was 97.9 ng/ml with a range of 41.9 ng/ml to 155.8 ng/ml and a SD of 25.0 ng/ml. Analyses of blood levels of furosemide in 47 horses at 4 hr after drug administration indicated that the population distribution was better fit by a normal curve after log transformation of the values. The mean plasma level at 4 hr post-dosing was 9.6 ng/ml with a range of 4.0 ng/ml to 19.4 ng/ml and a SD of 3.1 ng/ml. Based on this population distribution of the plasma levels, the probability of finding furosemide plasma concentrations above 24.6 ng/ml at 4 hr after anti-epistaxis dose was estimated at less than 1 in 1000.

Furosemide (Lasix), a potent, high ceiling diuretic, is currently the drug of choice for the prophylactic treatment of epistaxis or EIPH[2] in the horse (1). Historically, the incidence of EIPH in horses has been reported as low as 2.5 to 5% (2). However, recent surveys using fiberoptic endoscopy have indicated that at least 43% of racing horses have some degree of this condition (3). Occurrence of EIPH during a race can cause the affected horse to slow or stop abruptly, posing a serious threat to horses and jockeys in a tightly packed field. In an effort to control this condition during races, many racing commissions have permitted the pre-race use of furosemide.

The principal objection to the pre-race use of furosemide is associated with the diuretic response. This diuresis may result in the dilution of illegal drugs and drug metabolites in post-race urine samples, rendering their detection more difficult. Approval of furosemide without controls on its use may therefore make the task of the racing chemist more difficult.

One approach to obviating furosemide's interference with drug screening is to avoid urine collection during the period of diuresis. Studies have shown that furosemide-induced diuresis has a rapid onset and decline (4). After prophylactic doses of furosemide (0.5 mg/kg) administered iv, the diluting effects of furosemide on the levels of drugs and glucuronide metabolites in the urine are essentially complete within 3 hr of dosing (5). Therefore, furosemide administration at prophylactic doses to within 4 hr of post time would result in no effective dilution of post-race urine samples. This procedure has also been suggested by the Veterinary Chemist's Advisory Committee to the National Association of State Racing Commissioners (6).

The currently accepted method of enforcing time rules for furosemide is by means of a detention barn system. In this system, horses to be treated with furosemide are stabled in a secure barn about 5 hr before race time. Furosemide is administered at the approved dose and time pre-race under close and maintained supervision. Although highly visible and effective, such systems are expensive and may not be justified by the magnitude of the drug-diluting effect observed in practice (1). An alternate and less expensive approach to enforcing compliance with a "4-hr" furosemide rule could be to designate a plasma "tolerance level" of furosemide, above which there would be a substantial probability of violation of the time rule. The frequency distribution of furosemide plasma levels at 4 hr after administration of the anti-epistaxis dose can be used as the basis for determining this tolerance level.

Materials and Methods

Horses. Thoroughbred and half-thoroughbred mares, geldings, colts, and fillies were used in these experiments. The mares and geldings were at grass, whereas the colts and fillies were in active training. The horses were brought into individual box stalls before all experimental procedures. Six horses were used for the 1.0-mg/kg furosemide iv kinetic study. Four horses were used to study the kinetics after a 0.5-mg/kg dose. After iv administration of 0.5 mg/kg furosemide, 30 horses were sampled at 1-hr post-dosing and 47 horses were sampled at 4 hr post-dosing.

This work was supported by a grant from the Kentucky Division of the Horsemen's Benevolent and Protective Association and the Kentucky Equine Research Fund.

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[1] Correspondence should be addressed to Dr. Tobin.

[2] Abbreviations used are: EIPH, exercise-induced pulmonary hemorrhage; AIC, Akaike's Information Criterion.

Send reprint requests to: Dr. Thomas Tobin, Kentucky Equine Drug Research Program and Graduate Center for Toxicology, Department of Veterinary Science, University of Kentucky, Lexington, Ky. 40546.

Drug Administration. Pre-drug blood samples were drawn from all horses after which they were dosed with furosemide (Lasix, National Laboratories Corp., subsidiary of American Hoechst Corp., Somerville, N. J.) by rapid iv injection *via* the right jugular vein. Care was taken to insure that the whole dose was injected iv. All subsequent blood samples were drawn from the left jugular vein into 15-ml evacuated heparinized tubes (Becton-Dickinson, Rutherford, N. J.). After centrifugation at 1150 *g*, 5°C, for 15 min, the plasma was removed and stored in 10-ml aliquots at −10°C until assayed. To determine the frequency distributions of plasma levels after 0.5 mg/kg furosemide, blood samples were drawn from the horses at grass at 1 and 4 hr post-dosing. Horses in training were exercised at about 3 hr after dosing and blood samples were drawn at 4 hr after dosing. For the pharmacokinetic studies, doses of 1.0 mg/kg and 0.5 mg/kg furosemide were administered on separate occasions. Blood samples were then collected at 3, 6, 9, and 20 min and at 1, 2, 3, 4, 5, 6, 7, and 8 hr after dosing.

Analytical Method. The analytical method was modified from that of Roberts *et al.* (7) and is based on the method of Lindstrom and Molander (8) with a recovery of approximately 80% when spiked plasma was compared to standards in methanol added directly to the methylating system.

All samples were analyzed in duplicate. Standards were prepared by using dilutions of a 0.1 mg/ml furosemide stock solution dissolved in distilled water with the pH adjusted to 9.5 with concentrated ammonium hydroxide. Appropriate dilutions of furosemide were added to 1-ml aliquots of blank plasma to achieve a concentration range of 2.0 to 50 ng/ml. Standard curves to cover our working range were run with each assay. Early plasma samples were diluted with blank plasma to reduce their concentration to within the linear portion of our working range and to maintain a consistent background. Control plasmas from individual horses, standards, and plasma samples were extracted by using 1 N HCl and 5.0 ml dichloromethane. All samples were mixed at room temperature for 3 min, then centrifuged at 1150*g*, 5°C, for 30 min to achieve phase separation. After centrifugation, the aqueous layer was aspirated and discarded. The proteinaceous plug was dislodged by using a glass pipette and the dichloromethane phase transferred to a clean screw-cap tube. The organic phase was then evaporated to dryness under a stream of prepurified N_2. After evaporation, 2 ml of 0.2 M NaOH, 5 ml of 0.5 M methyl iodide (Fisher Scientific Company, Fair Lawn, N. J.) in dichloromethane, and 50 μl tetrahexylammonium hydrogen sulfate in dichloromethane were added to the residue in each tube. The samples were then incubated by shaking horizontally in a shaker-waterbath at 50°C for 25 min. After removal of the aqueous phase, the dichloromethane layer was transferred to clean screw-cap tubes and evaporated to dryness under a stream of prepurified N_2. Hexane (2.0 ml) and 2.0 ml of 0.5 M NaOH were added to the residue in each tube. The derivatized samples were then mixed at room temperature for 5 min and allowed to stand overnight at 10°C. One milliliter of the hexane layer was transferred to a clean screw-cap tube. Before analysis on the gas chromatograph, each sample was evaporated to dryness under a stream of prepurified N_2 and redissolved in 50 μl of hexane. After vortexing for 15 sec, a 2-μl sample was injected on a Varian 2700 gas chromatograph equipped with a 6-foot column packed with 3% OV-101, and a ^{63}Ni electron capture detector. The operating conditions included: column temperature of 265°C, detector temperature of 310°C, injector temperature 300°C, and a nitrogen flow of 31 ml/min.

Statistical Analysis. The frequency distributions at 1 and 4 hr after a 0.5-mg/kg dose of furosemide were analyzed for normalcy by using the Shapiro-Wilk's statistic. After the best-fit transformation was determined, the curve was estimated by using the methods of moments based on the calculated mean and SD. This distribution curve was then used to determine the plasma levels below which 99.9% of the population was included. This data then served as the basis for determination of plasma tolerance levels for compliance with a "4-hr furosemide rule."

Kinetic Analysis. Individual and mean data were fitted by the NONLIN computer program to a sum of exponentials by using both two and three exponential terms (9). The model which best described the data was chosen based on the application of the AIC (10). The values obtained from the computer analysis were then used to calculate kinetic parameters that were dependent on the number of exponents chosen to best describe the plasma concentration time curve and those independent of such assignment. These parameters were calculated based on both individual and mean values of A, B, C, and α, β, γ (11, 12).

Results

The mass spectrum of plasma sample (3 min after a 1.0-mg/kg dose of furosemide) is shown in fig. 1, confirming the presence of furosemide as the derivative methylated at the carboxylic and sulfonamide moieties. The molecular ion at 372 and the observed fragmentation pattern is consistent with the structural formula for trimethylfurosemide.

Figure 2 shows typical gas chromatograms obtained from methylated furosemide in spiked plasma. The data show a linear increase in detector response with the addition of increasing

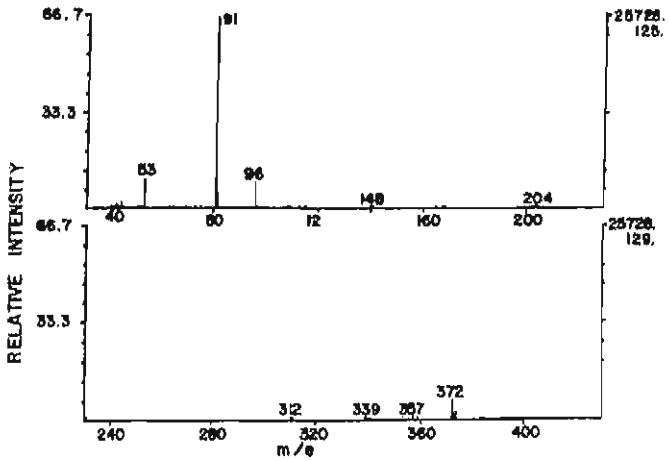

FIG. 1. *The mass spectrum of trimethyl furosemide from an equine plasma (3 min after a 1.0-mg/kg dose of furosemide).*

The molecular ion present at 372 is consistent with the m.w. of trimethyl furosemide.

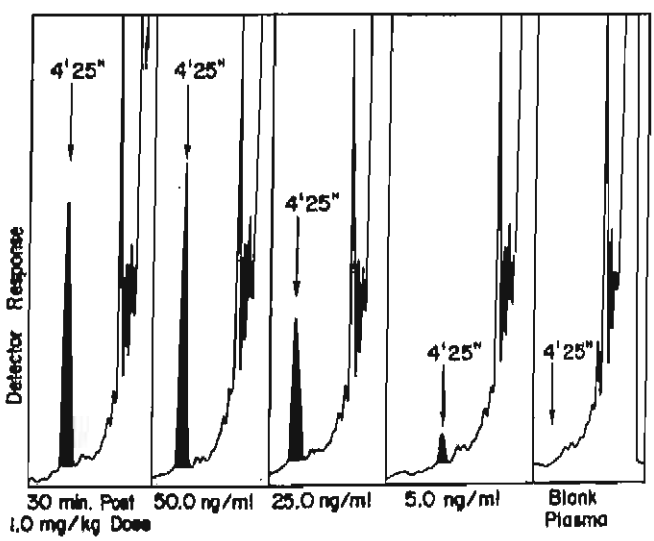

FIG. 2. *Detection of furosemide on a gas chromatograph equipped with an electron capture detector.*

Blank plasma, plasma spiked with furosemide and plasma from a horse dosed with 1.0 mg/kg furosemide were analyzed for furosemide as described in Materials and Methods. The solid peaks represent furosemide which had retention time of 4 min and 25 sec under these conditions. Increasing the concentrations of spiked furosemide yielded increasing detector responses.

amounts of furosemide. A plasma sample (obtained 30 min after a 1.0-mg/kg dose) detected by electron capture detection is shown in the far left panel.

Figure 3 shows a standard curve of furosemide added to 1 ml of horse plasma and analyzed by gas chromatography with electron capture detection. The detector response was quantitated by peak area in mm^2 with a mean range of 10.6 mm^2 to 288.5 mm^2 ($N = 5$). The standard curve is fit by a least squares regression line with a correlation coefficient of 0.990 and a positive intercept on the vertical axis of 4.15. Fitting the curve to the points below 21 ng/ml resulted in a regression line with a correlation coefficient of 0.9980 and a negative intercept on the vertical axis of 1.56. The extrapolated value at the origin was 0.24 ng/ml. To minimize the error when extrapolating the data points, a minimum of four standard points closest in peak area to the unknown were used for determination of concentration. Diluting samples with blank plasma as described in the Materials and Methods section also facilitated control in keeping the concentrations in a linear portion of our curve. There was no significant difference found between the sample duplicates when evaluated by a paired t test ($\alpha = 0.01$, $t = 2.21$). The accuracy of the technique was also reproducible between standard curves. The SE for the standard points indicated in fig. 3 reflects this, representing less than 5% of the plasma concentration. Variability was 8% of the plasma concentration at the lower levels of detection (2 ng/ml).

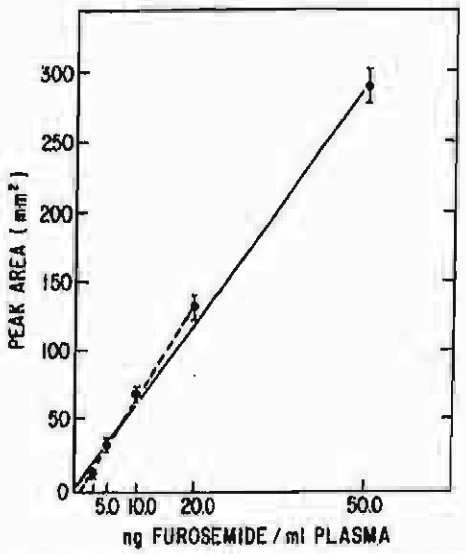

FIG. 3. *Standard curve of furosemide extracted from horse plasma.*

The indicated amounts of furosemide were added to the plasma. The solid circles (●——●) represent the detector response to the added amounts of furosemide as the mean of five experiments ± SE.

Furosemide (1.0 mg/kg) was administered to six horses (415–577 kg) by rapid iv injection. The mean furosemide plasma levels are shown in fig. 4 *A*. The mean plasma level at 3 min after dosing was 7426 ng/ml and fell in a curvilinear fashion to a mean level of 3.0 ng/ml at 8 hr after dosing. At 4 hours, the mean plasma level of furosemide was 11.8 ± 1.4 ng/ml. These results are in good agreement with those previously reported by Roberts and co-workers (7), whose data are represented in fig. 4 *A* by the open circles.

In the prophylaxis of epistaxis in race horses, 0.5 mg/kg iv is the dose most commonly used. We therefore dosed four horses with 0.5 mg/kg furosemide iv and followed plasma levels of the drug for 8 hr. As shown in Fig 4 *B*, plasma levels of furosemide were approximately 5306 ng/ml at 3 min after dosing. Thereafter,

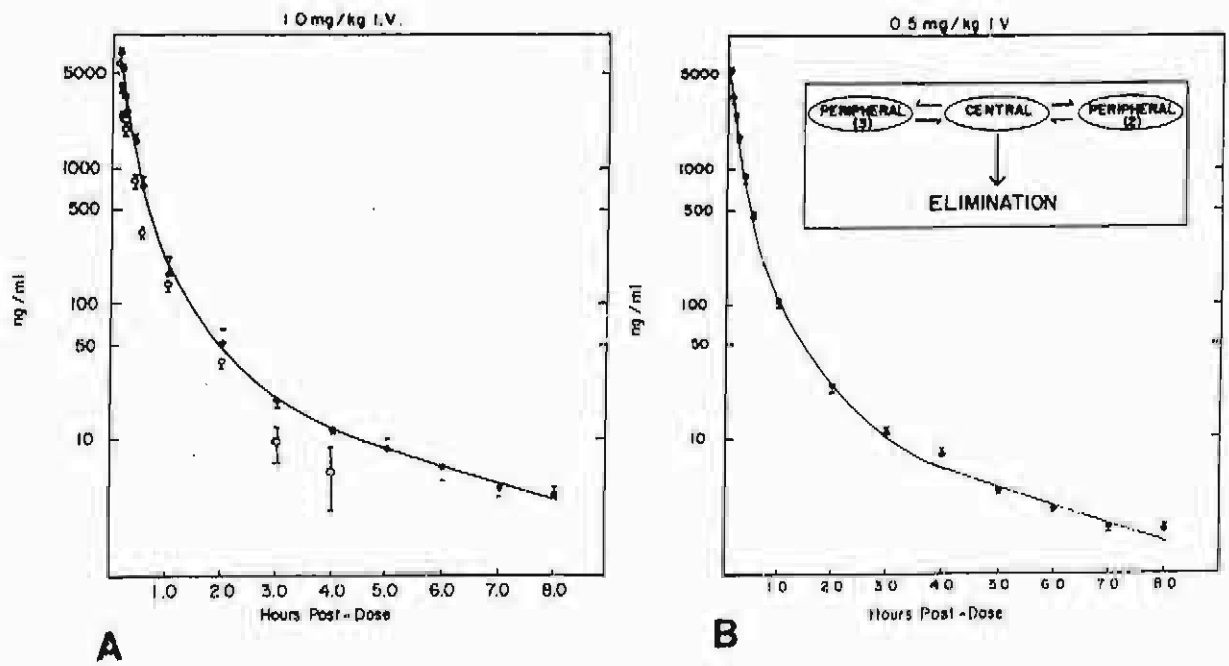

FIG. 4 *A. Plasma levels of furosemide after 1 mg/kg iv.*

The solid circles (●——●) are plasma levels of furosemide in six horses after rapid iv administration of 1 mg/kg furosemide. The open circles (○——○) show data in five similarly treated horses, as reported by Roberts *et al.* (7). The vertical bars represent the SE of the mean. The solid curve is the best fit curve generated by a nonlinear regression analysis.

FIG. 4 *B. Mean plasma levels of furosemide in four horses after a 0.5 mg/kg dose iv.*

The solid circles (●——●) are plasma levels of furosemide after 0.5 mg/kg in four horses.

plasma levels of the drug fell in a curvilinear fashion to about 3 ng/ml at 8 hr after dosing (solid circles, (●——●).

The solid curve in fig. 4 *A* and *B* represents the best fit curve when these data were assessed by nonlinear regression analyses.

Based on the calculated plasma concentrations from the regression analyses, the values for A, B, C, and α, β, and γ (table 1) were derived. Both individual and mean data were evaluated and the mean of the kinetic parameters from individual animals are reported ± SE in table 1. Comparisons for statistical significance between the data (0.5 mg/kg and 1.0 mg/kg) were done with a two-sample *t* test ($\alpha = 0.01$). The *t* values are reported as absolute values in table 1.

The data of fig. 5 show the frequency distribution of furosemide plasma levels ($N = 30$) at 1 hr after a 0.5-mg/kg dose. The range was 41.9 to 155.8 ng/ml, with a mean of 97.9 ng/ml. At 1 hr after dosing, the furosemide plasma levels were normally distributed, with a Shapiro-Wilk's statistic of $p < 0.989$.

The data of fig. 6 *A* show plasma levels of furosemide in 47 horses at 4 hr after a 0.5-mg/kg dose. The mean plasma level was 9.6 ng/ml, with a range of from 4.0 to 19.4 ng/ml. These data poorly fit a normal distribution, with a Shapiro-Wilk's statistic of $p < 0.01$.

When these data were replotted as frequency against log-concentration of drug in plasma (fig. 6 *A*), the data much better fit a normal distribution, with a Shapiro-Wilk's statistic of $p < 0.053$. These data suggest that at 4 hr after drug administration, the distribution of plasma levels obtained in these horses were log-normal.

Based on the assumption that plasma levels of furosemide are log-normally distributed, the probability of an anti-epistaxis dose of furosemide yielding plasma levels above a certain level can be readily calculated. These data are presented in table 2, and show that the probability of an anti-epistaxis dose of furosemide yielding levels of furosemide above 24.6 ng/ml is less than 1 in 1000. Similarly, the probability of plasma levels above a series of different drug concentrations are also presented (table 2).

Discussion

This work confirms and extends the earlier work of Roberts *et al.* (7). Modification of Roberts' method improved the sensitivity of the analytical method about 10-fold and allowed detection of furosemide in equine plasma for up to 8 hr postadministration. The basic characteristics of the detection method, however, are

TABLE 1

Table of kinetic parameters based on the nonlinear regression analyses

	0.5 mg/kg ($N = 4$)	1.0 mg/kg ($N = 6$)	| *t* |
A (ng/ml)	6063.8 ± 145.2	9148.6 ± 666.6	—
B (ng/ml)	675.7 ± 141.2	1016.2 ± 174.0	—
C (ng/ml)	21.6 ± 6.0	32.9 ± 7.2	—
α (min^{-1})	0.1300 ± 0.01	0.1330 ± 0.02	—
β (min^{-1})	0.0330 ± 0.004	0.0320 ± 0.0049	—
γ (min^{-1})	0.0047 ± 0.0007	0.0044 ± 0.0006	—
*t*1/2 α (min)	5.6 ± 0.44	5.8 ± 0.74	0.1341
*t*1/2 β (min)	22.3 ± 3.09	24.1 ± 3.40	0.3674
*t*1/2 γ (min)	158.5 ± 24.69	177.2 ± 28.70	0.4573
K_{12} (min^{-1})	0.0180 ± 0.0049	0.0230 ± 0.0047	0.7319
K_{21} (min^{-1})	0.0420 ± 0.0052	0.0420 ± 0.0070	0.0730
K_{13} (min^{-1})	0.0079 ± 0.0016	0.0064 ± 0.0015	0.6862
K_{31} (min^{-1})	0.0051 ± 0.0008	0.0100 ± 0.0070	0.4094
K_{10} (min^{-1})	0.0907 ± 0.0027	0.0940 ± 0.0130	0.1930
Vc (liters/kg)	0.0740 ± 0.0030	0.2200 ± 0.1100	1.03
Vdss (ml/kg)	164.8 ± 13.82	241.1 ± 19.29	2.88
Cl (ml/kg/hr)	385.5 ± 16.31	503.8 ± 32.88	2.75

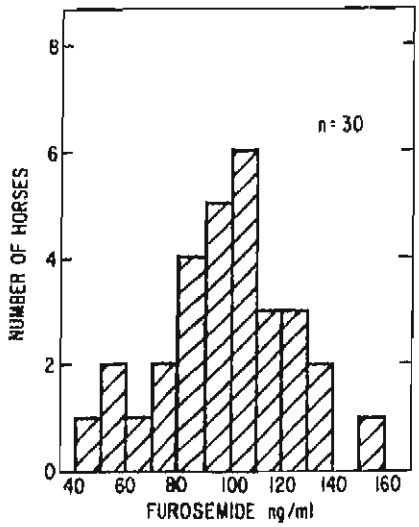

FIG. 5. *Furosemide plasma levels in 30 horses 1 hr after iv administration of 0.5 mg/kg furosemide.*

The vertical bars represent the number of horses found within the indicated ranges of furosemide plasma levels.

those described by Roberts *et al.* (7) and earlier by Lindstrom and Molander (8). The standard curve is linear over small concentration ranges, and the intercept at the foot of the vertical axis is not significantly different from zero.

Analyzing their data, Roberts *et al.* (7) suggested a two-compartment model for furosemide distribution in the horse after its iv injection. Analyzing our data, we elected to use a nonlinear least squares regression analysis with a $1/y^2$ weighting (NONLIN Program) to evaluate the plasma concentration-time curve after both the 1.0-mg/kg and 0.5-mg/kg doses of furosemide. Using the AIC, triexponential rather than biexponential equations were found to better fit the data. Elimination in this model is from the central compartment as shown in the inset to fig. 4 *B*. For the 1.0-mg/kg dose, the following equation best described the data: $C = 9148.6e^{-0.1330t} + 1016.2e^{-0.0320t} + 32.9e^{-0.0044t}$. Similarly after the 0.5-mg/kg dose, the following equation best described the data: $C = 6063.8e^{-0.13t} + 675.7e^{-0.033t} + 21.6e^{-0.0047t}$ where *A*, *B*, and *G* are expressed in nanograms per milliliter and α, β, and γ are expressed in $minutes^{-1}$.

The α- and β-phase half-lives were in good agreement with work previously done in the horse which reported α- and β-phase half-lives after a 1.0-mg/kg dose of furosemide of 5 min and 31.5 to 38.6 min, respectively (7). Similar β-phase half-lives of 30 min and 26 min have also been reported in human volunteers whose furosemide levels were determined fluorometrically (13, 14). Our studies indicated that there were no statistically significant differences (two-sample *t* test $\alpha = 0.01$, see table 1) in any of the kinetic parameters between the two doses administered.

The volume of the central compartment (*Vc*) was 0.074 liter/kg at the 0.5-mg/kg dose and 0.22 liter/kg for the 1.0-mg/kg dose. These findings correlate well with what has previously been reported in the horse (7).

In the triexponential fit, the γ-phase represents less than 6% the total area under the plasma concentration-time curve. For a biexponential fit, the β-phase represents only about 8% the area under the curve. This would indicate that neither the second nor third exponent has much influence on the actual plasma concentration-time curve. The lack of a predominating influence of the terminal phase on the overall curve could be attributed to the

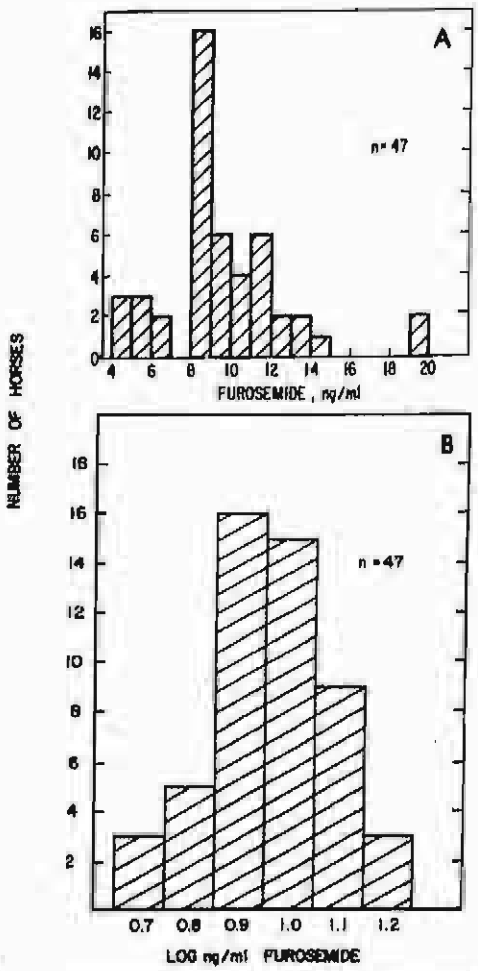

FIG. 6. *Furosemide plasma levels in 47 horses 4 hr after iv administration of 0.5 mg/kg furosemide.*

A, the vertical bars represent the number of horses found within the indicated ranges of furosemide plasma levels; *B*, the vertical bars represent the number of horses found within the indicated ranges of the log of furosemide plasma levels.

TABLE 2

Table of plasma levels of furosemide and average levels based on the population distribution of figure 11

Probability of Overage	Plasma Level of Furosemide
%	*ng/ml*
50	9.10
25	11.30
10	13.74
5	15.44
1	19.24
0.1	24.57
0.05	27.18

rapid decay of drug levels. Based on the AIC the triexponential equation was the simplest description of the data.

Identification of a third pharmacokinetic compartment for furosemide follows a pattern commonly observed when analytical methodology is improved. The ability to analyze for smaller concentrations allows the detection of a slower and slower decay of drug concentration. This pattern suggests that there is no real terminal half-life, but that the apparent terminal slope becomes less and less. Further enhancement of analytical methodology might presumably demonstrate an even slower terminal plasma half-life than that demonstrated here.

We assessed the clearance and volume of distribution, which are parameters that are independent of dose and modeling of the data (11), at steady state. The clearance at the 0.5-mg/kg dose ranged from 341.9 to 420.9 ml/kg/hr ($N = 4$) with a clearance of 385.49 ml/kg/hr, a mean value based on individual data analyses. At the 1.0-mg/kg dose, the clearance ranged from 420.1 ml/kg/hr to 642.3 ml/kg/hr ($N = 6$) with a mean clearance of 503.8 ml/kg/hr. The volume of distribution at steady state (*Vdss*) at the 0.5 mg/kg dose ranged from 126.6 ml/kg to 192.4 ml/kg with a mean *Vdss* of 164.8 ml/kg. At the 1.0-mg/kg dose, the *Vdss* ranged from 184.1 ml/kg to 323.2 ml/kg, with a mean *Vdss* of 241.1 ml/kg. Studying furosemide in human volunteers, similar *Vdss*'s of 110.0 ml/kg, 176 ml/kg, and 210 ml/kg have been reported by other workers (15–17).

The pharmacokinetics of furosemide in racing horses are of considerable forensic interest to racing authorities. Racing authorities may wish to insure that furosemide is administered at a certain period before race time to prevent its diluting effects on drugs and drug metabolites in equine urine. Although a detention barn can be used to enforce such time rules, a more economical method might be a furosemide tolerance level in equine plasma. Based on this consideration, we elected to develop our pharmacokinetic studies in such a way as to determine what plasma levels of furosemide would suggest compliance with a "4-hr" furosemide rule.

At 1 hr after dosing, blood levels ranged from 41.9 ng/ml to 155.8 ng/ml, with a mean of 97.9 ng/ml and a SD of 25.0 ng/ml. By 4 hr after dosing, the blood levels had fallen to a range of 4.0 to 19.4 ng/ml, with a mean of 9.56 ng/ml and a SD of 3.1 ng/ml. The levels found at 1 hr post-dosing were high enough to be clearly distinguished from those found at 4-hr post-dosing.

At 1 hr the furosemide plasma levels were normally distributed, but by 4 hr the distribution appeared skewed. Based on the assumption that the data were fit by a log-normal distribution, a population curve was generated by using the mean plasma level and the SD. This analysis suggested that the probability of a plasma level of 24.6 ng/ml at 4 hr after an anti-epistaxis dose of furosemide was less than 1 in 1000.

As a practical matter, these experiments suggest that a regulatory level of furosemide in equine plasma can be set and used to control the use of furosemide in racing horses. If this level is set at 30 ng/ml in equine plasma, the probability of a random overage would be substantially less than 1 in 1000. On a 10,000-sample per year jurisdiction, with 10% of the horses running on furosemide, the accidental "overage" rate would not be expected to be more than about one or two a year. Because the great bulk of the diuretic and drug-diluting effects of furosemide are over within 1 hr and are essentially complete by 3 hr after iv administration, the probability of a forensically significant drug-diluting effect in horses with less than 30 ng/ml furosemide in their plasma is minimal.

By 4 hr after furosemide administration, the detection of some drugs by thin-layer chromatography is enhanced (18). Therefore, incorporation of a 30 ng/ml furosemide tolerance into medication rules will obviate the possibility of drug dilution effects and may accentuate the drug detection-enhancing properties of this agent.

The data base developed in this investigation can be used to develop a range of furosemide tolerance levels. For example, a tolerance level of 19.2 ng/ml (20 ppb) of furosemide would set the random "overage" rate at about 1% or 1 in 100. Lower tolerance

levels could also be selected, but would be correspondingly stricter, and are not likely to be as useful as higher tolerance levels.

In applying this furosemide tolerance level, it is important that either the analytical method described here be used, or the method used be compared carefully with this method. This is because the only data on furosemide blood levels in the horse published to date are those described in this and previous papers from this laboratory. How closely alternative methodologies would correlate with this data base is not yet known, and should be checked by investigators who elect to use other methodologies.

Acknowledgments. The cooperation and assistance of the J. T. Ward Stables, Inc. is gratefully acknowledged.

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Validation of the AAEP-Industry Position on Furosemide

Dr. Richard Sams, Ohio State University

Summary

During the late 1970's, concern grew among analysts about the effects of furosemide on urinary drug detection. A study involving seven laboratories suggested that the diuresis produced by 1 mg/kg of furosemide IV interfered with urinary drug detection. However, in the early eighties a second inter-laboratory study, using the AAEP recommended dose of furosemide (0.5 mg/kg @ 4 h prior to testing), showed no significant interference with drug detection. This second study was conducted under the auspices of the American Horse Council, and it opened the way for an official industry-wide position on furosemide.

●●●●●

Dr. Sams: Professor Tobin has concisely and accurately described the evolution of the regulatory control of furosemide in horse racing in the United States during the past twenty years. I would like to describe the results of a study that was carried out in the late 1970's that contributed to the concern by racing analysts about the effects of furosemide on drug and metabolite detection in official samples. Then, I will review the evolution of various rules regulating the use of furosemide in racehorses.

The study was designed and undertaken by a group of analysts representing seven laboratories in the United States. Twelve drugs from various chemical and pharmacological classes were administered

to horses by various routes. Furosemide was administered at doses of 0.5 to 1.0 milligrams per kilogram intravenously at various times before and after these drugs were administered. Urine samples were collected from the horses at various times after drug administration, divided, and sent to these seven racing laboratories. The drugs, doses, and routes of administration investigated in this study were:

Drug	Dose	Route
1. apomorphine,	18 mg	IM
2. fentanyl,	0.25 mg	IM
3. pentazocine,	150 mg	IM
4. nalbuphine,	20 mg	IM
5. butorphanol,	15 & 18 mg	IM
6. oxymorphone,	10 mg	IM
7. acepromazine,	10 mg	IV
8. methylphenidate,	150 mg	IV
9. piperacetazine,	12 mg	IV
10. alaphrodine,	30 mg	IV
11. amitriptyline,	100 mg	IV
12. methamphetamine,	150 mg	IV

The seven laboratories tested for all of these drugs by thin layer chromatographic methods, with the exception of fentanyl. Detection of fentanyl administration required the use of a newly-introduced radioimmunoassay procedure, and confirmation required isolation of a hydrolysis product of an oxidative metabolite. The laboratories reported back to Dr. Maylin and myself that the administration of furosemide resulted in some degree of interference in the detection of all these substances except pentazocine and methamphetamine for as long as 6 h after furosemide administration.

The reason for the different doses of furosemide in this study was that there was considerable controversy about the appropriate dose of furosemide to use in the treatment of epistaxis in horses. Only later did

the American Association of Equine Practitioners (AAEP) specify a dose of 250 milligrams administered IV 4 h before post-time as their recommended dose.

Subsequent to the completion of this study, members of the racing community and some outside it became alarmed about the effects of furosemide on drug detection and the effects of uncontrolled use of furosemide on the integrity of racing. This concern prompted the National Association of State Racing Commissioners (NASRC) to prohibit the use of furosemide in, I believe, 1981. As Professor Tobin has pointed out, this action by the NASRC prompted a series of meetings in which the Horseman's Benevolent and Protective Association (HBPA) pressed for some relief from the prohibition of furosemide. The American Horse Council (AHC) assembled a task force on medication and convened a series of meetings to discuss medication issues. They also proposed legislation that would regulate the use of drugs in racehorses. The task force included representatives from various segments of the racing industry including analysts and the HBPA.

A particularly important breakthrough came when the AAEP reported their recommendation with regard to the dose, time, and route of administration of furosemide. This recommendation cleared the way for the committee convened by the AHC to request that the analysts repeat their study of the effects of furosemide on drug detection, but this time with a furosemide dose of 250 milligrams administered intravenously 4 h before collection of the urine samples.

Therefore, a study with numerous representative drugs was conducted in the Cornell University and the Ohio State University Laboratories. Various drugs were administered at these universities. Furosemide (250 mg) or normal saline was administered IV, and urine samples were collected from each of the horses approximately 4 h later. Urine samples were exchanged between the two laboratories by routine testing procedures. Analysts from the laboratories reported that there

was no significant interference between those samples collected after furosemide and normal saline solution. It was concluded that furosemide did not interfere with the detection of any of these drugs if the dose of furosemide was 250 mg and the route of administration was IV.

When these results were reported to the AHC task force on medication, it recommended that the NASRC consider a rule change that would permit furosemide at a dose of 250 mg administered IV route 4 h before race time. That recommendation was considered by the veterinary chemists advisory committee to the NASRC. It's my recollection that the recommendation was accepted by the Veterinary-Chemists Sub-Committee to the NASRC, but as Professor Tobin has just said, I don't believe it was ever formally adopted by NASRC.

Dr. Tobin: This was before the hearings in the Spring of 1983.

Dr. Sams: There was some delay before the recommendation came before the NASRC Veterinary Chemists Sub-Committee.

Dr. Soma: There was a meeting where, I'm assuming, they accepted this because I remember being at that meeting and I don't remember . . . it was about the early 1980's where this whole thing was discussed. I think the meeting at large agreed that 250 milligrams should be administered. Whether it was ever adopted as a specific resolution . . because after that meeting our state went back, Pennsylvania, and based on the vote on the floor made the recommendation that it be used in Pennsylvania.

Dr. Sams: There was indeed considerable discussion on the floor after the veterinary chemists advisory committee had met and discussed this recommendation, and then there was considerable discussion before the full NASRC. I know that a number of racing jurisdictions subsequently adopted rules that allowed furosemide at 250 milligrams IV 4 h before race time; however, my search of the NASRC rules and

other people's searches of those rules has indicated that the recommendation was never adopted by the NASRC.

Dr. Soma: Do you think that was an oversight?

Dr. Sams: I don't know.

Dr. Tobin: My sense was that the NASRC just stood back and allowed the Veterinary-Chemists Advisory report to stand and let the commissions adopt it if they wished.

Dr. Sams: That may very well be. I felt it was important that we review some of the origins of this rule-making process, and I think the take home message that I'd like you to get is that this rule-making process was based upon concerns about the ability of furosemide to interfere with the detection of other substances. This consideration was totally independent of any concerns about effects of furosemide on performance. Thank you.

Dr. Tobin: Thank you, Rick. Having set the stage for the questions about the effects of furosemide on the performance of horses, we now hand over the podium to Dr. Larry Soma. Larry is going to speak on the effects of furosemide on the racing performance of horses.

***Footnote:**

The official history of NASRC-ARCI with regard to furosemide is as follows:

Furosemide was recommended to NASRC by the then Veterinary-Chemists Sub-Committee of NASRC in 1979. In 1981 NASRC voted to ban furosemide; twenty-four jurisdictions voting in support of the

motion, three abstaining, and one "not present". There was no further formal action on furosemide until May of 1996, when the ARCI model rules, which contain specific wording on furosemide regulation were formally adopted by ARCI. These model rules are presented in Appendix I, courtesy of ARCI.

Appendix I

ARCI Model Rule For Furosemide

F. Furosemide (Lasix®)

1. Furosemide (Lasix®) may be administered intravenously to a horse which is entered to compete in a race. Except under the instructions of the official veterinarian or the racing veterinarian for the purpose of removing a horse from the Veterinarian's List or to facilitate the collection of a post-race urine sample, Furosemide (Lasix®) shall be permitted only after the official veterinarian has placed the horse on the Bleeder List.

2. The use of Furosemide (Lasix®) shall be permitted under the following circumstances on association grounds where a detention barn is utilized:

a) Furosemide (Lasix®) shall be administered at the direction of the official veterinarian or his/her designee no less than four hours prior to post time for the race for which each horse is entered.

b) A horse qualified for a Furosemide (Lasix®) administration must be brought to the detention barn within time to comply with the four-hour administration requirement specified above.

c) The dose administered shall not exceed 250 mg nor be less than 150 mg.

d) After treatment, the horse shall be required by the Commission to remain in the detention barn in the care, custody,

and control of its trainer or the owner's designated representative under association and/or Commission security supervision until called to the saddling paddock.

3. The use of Furosemide (Lasix®) shall be permitted under the following circumstances on association grounds where a detention barn is not utilized:

a) Furosemide (Lasix®) shall be administered no less than four hours prior to post-time for the race for which a horse is entered.

b) The Furosemide (Lasix®) dosage administered shall not exceed 250 mg nor be less than 150 mg,

c) The trainer of the treated horse shall cause to be delivered to the official veterinarian or his/her designee no later than one hour prior to post time for the race for which the horse is entered the following information under oath on a foreign provided by the Commission:

(1) the race track name, the date and time the Furosemide (Lasix®) was administered to the entered horse;

(2) the dosage amount of Furosemide (Lasix®) administered to the entered horse;

(3) the printed name and signature of the attending licensed veterinarian who administered the Furosemide (Lasix®).

G. Bleeder List

1. The official veterinarian shall maintain a Bleeder List of all horses which have demonstrated external evidence of exercise induced pulmonary hemorrhage or the existence of hemorrhage in the trachea

post exercise upon endoscopic examination. Such examination must have been performed by or in the presence of the official veterinarian or the racing veterinarian.

2. The confirmation of a bleeder horse must be certified in writing by the official veterinarian or the racing veterinarian and entered on the Bleeder List. Copies of the certification shall be issued to the owner of the horse or the owner's designee upon request. A copy of the bleeder certificate shall be attached to the horse's certificate of registration.

3. Every confirmed bleeder, regardless of age, shall be placed on the Bleeder List.

4. A horse must be removed from the Bleeder List only upon the direction of the official veterinarian, who shall certify in writing to the stewards the recommendation for removal.

5. A horse which has been placed on a Bleeder List in another jurisdiction may be placed on a Bleeder List in this jurisdiction provided that the other jurisdiction's criteria for the identification of bleeders are satisfactory in this jurisdiction.

*This was formally adopted by ARCI on May, 1996

Furosemide in the Horse

Its Actions, Effects, and Regulatory Control

A Testing Integrity Program Seminar
Held at
The New Orleans Hilton Riverside
March 1, 1998

Convened by
Dr. Richard Sams & Dr. Scott Stanley

Editors
J.D. Harkins, Wyndee Carter,
C.G. Hughes & Thomas Tobin

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Presented here is the published version of a workshop seminar entitled "An Overview of Furosemide in the Horse: It's Actions, Effects and Regulatory Control," convened on March 1st, 1998 in New Orleans. This workshop was convened by the Testing Integrity Program (TIP) to bring together the best available scientific data, and the scientific and individual collective memories of TIP members and other invitees. Data, information and opinions presented here are those of individual TIP members and invitees, speaking as individual scientists and not as regulators or on behalf of any regulatory authorities.

Nothing stated herein should be construed as an official TIP position, nor should any of the statements be taken as representing those of any regulatory authority. Readers are also reminded that the scientific process is ongoing, that new facts continually become available, and that previously available data, information, and interpretations may well be re-evaluated and re-interpreted in the light of new facts, concepts, or insights.

Seminar Participants

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Dr. Lawrence Soma
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Dr. Ron Jensen
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Dr. David Tiffany
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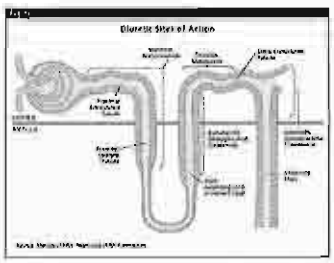
RACE DAY MEDICATION AND DRUG TESTING

Richard Sams, PhD
Director
HFL Sport Science, Inc.
Lexington, Kentucky

Loop Diuretics

- The functional unit of the kidneys is the glomerulus
- Water and electrolytes are normally reabsorbed from the glomerulus
- Waste products are eliminated
- Loop diuretics competitively inhibit Na-K-Cl transporter in the Loop of Henle
- Inhibition of chloride reabsorption decreases driving force for water reabsorption
- More than 98% of the water entering the glomerulus is normally reabsorbed



Race Day Medications

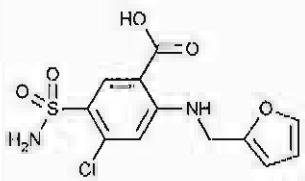
- High Ceiling Loop Diuretics
  - Furosemide
  - Bumetanide
  - Ethacrynic Acid
  - Torsemide
- Fibrinolysis Inhibitors
  - Aminocaproic Acid
  - Tranexamic Acid
- Antihemorrhagic Agents
  - Carbazochrome
  - Etamsylate
- Others

Loop Diuretics

Furosemide

- High ceiling loop diuretic
- Marketed as Lasix™ and Salix™
- Available as oral and parenteral products
- First use in horses reported from late 1960s
- Readily detected in blood and urine by contemporary methods of analysis

Chemical Structure



LOOP DIURETICS

Furosemide and related diuretics

Furosemide

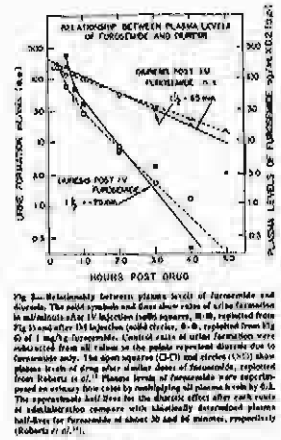
- Synthesized in early 1960s
- Results of clinical trials reported in 1963
- Approved in human medicine for treatment of hypertension from Hoechst (now Sanofi Aventis) in July 1966
- Injectable veterinary product from Hoechst introduced in 1967
- Intervet purchased furosemide from Hoechst and renamed it Salix™

Furosemide

- Lasix™ Injectable available from Hoechst as approved veterinary product in 1967
- First injectable diuretic approved for use in horses
- Indications: For the treatment of edema (pulmonary congestion, ascites) associated with cardiac insufficiency, and acute non-inflammatory tissue edema (US FDA).
- Pioneering work on diuretic efficacy in horses by Dr. Marvin Beeman of Littleton, Colorado

Furosemide

- Pharmacology
  - Dose dependent diuretic effect in horses
  - Decreases reabsorption of electrolytes and water
  - Produces mild metabolic alkalosis
  - Greater diuretic effect after IM administration
- Pharmacokinetics
  - Rapidly cleared by renal mechanisms
  - Extensively protein bound at physiological concentrations
  - Small volume of distribution
  - Not metabolized
  - Excreted rapidly in urine



Furosemide

- Administered to horses to prevent EIPH by late 1960s
- Earliest use of furosemide in bleeders attributed to Dr. Alex Harthill

  Lasix™ use permitted under "permissive medication" programs by mid-1970s
- Use listed in racing programs
- Dose, route, and time of administration were not regulated or standardized
- Urine samples submitted from treated horses were often dilute
- Concerns were raised about effect of furosemide induced diuresis on drug detection
- Veterinary advisory committee to NASRC recommended that NASRC prohibit furosemide in racing
- NASRC voted to prohibit furosemide in racing in 1983
- Several racing commissions followed NASRC recommendation
- Various groups of trainers threatened to boycott racing

Furosemide

- Pharmacology
  - Dose dependent diuretic effect in horses
  - Decreases reabsorption of electrolytes and water
  - Produces mild metabolic alkalosis
  - Greater diuretic effect after IM administration
- Pharmacokinetics
  - Rapidly cleared by renal mechanisms
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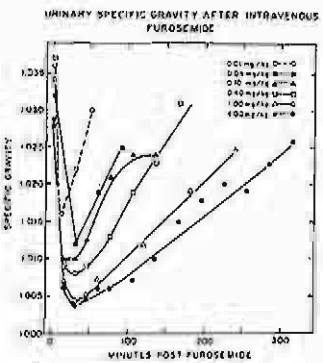
  - Excreted rapidly in urine

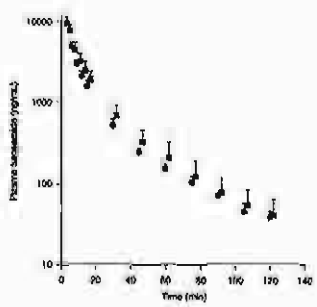


Furosemide

- The AHC (Tom Aronson and Rich Rolapps) took the lead in addressing the furosemide impasse
- They asked the AAEP for a recommended dose, route, and time of administration
- AAEP specified:
  - IV route only
  - 250 mg total dose
  - 4 hours before racing
- Fixing the dose led to studies to determine whether samples collected 5-6 hours after dosing were dilute
- George Maylin and I conducted studies on effects of this dose regimen on detection of drugs and metabolites in urine by TLC methods
- Results indicated no significant effects on detection of ten drugs
- Dose, route, and time were fixed based on these studies
- Various commissions approved furosemide use with dosing restrictions
- All racing commission had approved Lasix use by 1996

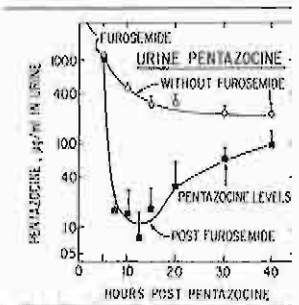
Furosemide

- Pharmacology
  - Dose dependent diuretic effect in horses
  - Decreases reabsorption of electrolytes and water
  - Produces mild metabolic alkalosis
  - Greater diuretic effect after IM administration
- Pharmacokinetics
  - Rapidly cleared by renal mechanisms
  - Extensively protein bound at physiological concentrations
  - Small volume of distribution
  - Not metabolized
  - Excreted rapidly in urine



Furosemide

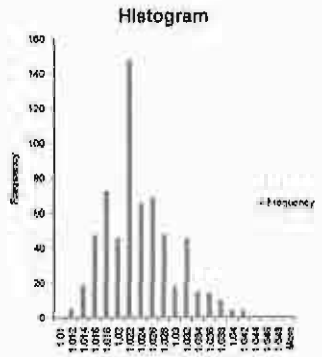
- Effects of diuresis on detection of other drugs
  - Diuresis decreases urine concentration of polar drugs and metabolites up to 50x at peak diuresis – excretion rates are not appreciably affected
    - Pentazocine, morphine, lidocaine metabolites, mepivacaine metabolites, butorphanol, etorphine, nalbuphine, pyrilamine metabolites, glycopyrrolate, tripelennamine metabolites, etc.
  - Diuresis alters the urine concentrations of lipid soluble drugs and metabolites several fold – excretion rates are increased during peak diuresis
    - Caffeine, theophylline, phenylbutazone, flunixin, naproxen, ketoprofen, etc



TOBIN REFERENCE

Furosemide

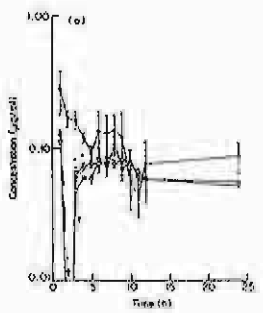
- Dose Limitations
  - IV route only
  - Dose from 100-500 mg
  - Four hours or more before post-time
  - Dose administered by regulatory vet
- Regulatory controls
  - Specific gravity < 1.010 and
  - Plasma or serum concentration over 100 ng/mL
- Evidence for Compliance
  - Urine specific gravity
    - 835 consecutive urine samples from Thoroughbred horses racing on furosemide
    - Furosemide confirmed
    - Mode: specific gravity = 1.002
    - Gives values less than 1.012
    - All values greater than 1.010
  - Serum furosemide concentration



URINE SPECIFIC GRAVITY CUT-OFF

Furosemide

- Effects of diuresis on detection of other drugs
  - Diuresis decreases urine concentration of polar drugs and metabolites up to 50x at peak diuresis – excretion rates are not appreciably affected
    - Pentazocine, acepromazine metabolites, morphine, lidocaine metabolites, mepivacaine metabolites, butorphanol, etorphine, nalbuphine, pyrilamine metabolites glycopyrrolate, tripelennamine metabolites etc
  - Diuresis alters the urine concentrations of lipid soluble drugs and metabolites several fold – excretion rates are increased during peak diuresis
    - Caffeine, theophylline, phenylbutazone, flunixin, naproxen, ketoprofen etc



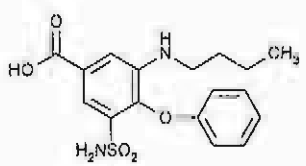
Effect of furosemide on detection Of acepromazine metabolites.

Loop Diuretics

Bumetanide

- High ceiling loop diuretic
- Marketed as Bumex™
- Available as oral and parenteral products
- Detected and reported from horse urine in 1990s where furosemide was not permitted
- Readily detected by contemporary methods of analysis

Chemical Structure



Furosemide

- Effects of diuresis on detection of other drugs
  - Diuresis decreases urine concentration of polar drugs and metabolites up to 50x at peak diuresis – excretion rates are not appreciably affected
    - Pentazocine, morphine, lidocaine metabolites, mepivacaine metabolites, butorphanol, etorphine, nalbuphine, pyrilamine metabolites, glycopyrrolate, tripelennamine metabolites, etc
  - Diuresis may alter the urine concentrations of lipid soluble drugs and metabolites several fold – excretion rates are increased during peak diuresis
    - Procaine, methylphenidate, caffeine, theophylline, phenylbutazone, flunixin, naproxen, ketoprofen, etc

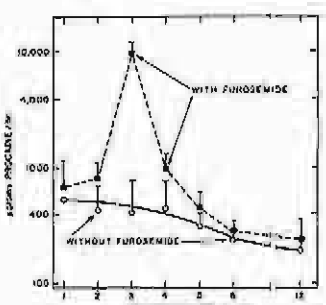


Fig 6--Effect of furosemide on urinary excretion of procaine. The open circles (○ - ○) show urinary elimination of procaine after 10 mg/kg of procaine HCl IM, the solid squares (■ - ■) show elimination of procaine after 1 mg/kg furosemide IV at 2 hours. All experimental points are the means of determinations of 4 different horses ± standard error of the mean.

TOBIN REFERENCE

Loop Diuretics

Bumetanide

- Rapidly cleared by renal excretion
- Half-life shorter than that of furosemide
- More potent than furosemide
- Maximum diuretic effect is equal to that of furosemide

Pharmacokinetics

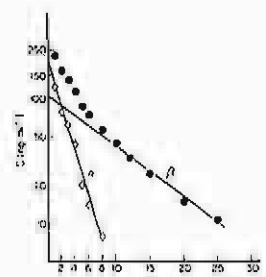


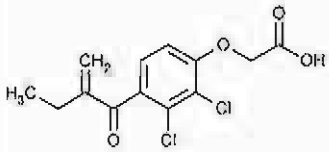
FIG. 1. Mean plasma levels of bumetanide after i.v. injection of 15 μg/kg to five horses.

Loop Diuretics

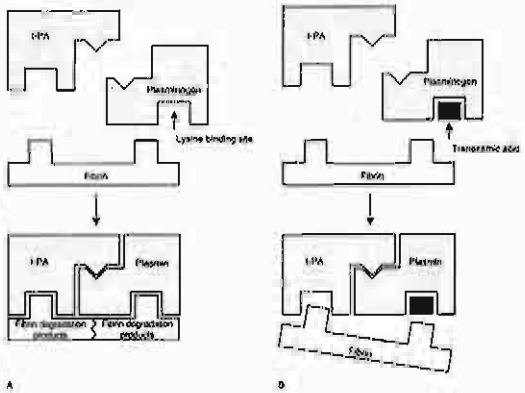
Ethacrynic Acid

- High ceiling loop diuretic
- Marketed as Edecrin™
- Available as oral and parenteral products – generics available
- Detected and reported from horse urine in 1980s where furosemide was not permitted
- Readily detected by contemporary methods of analysis

Chemical Structure



Fibrinolysis Inhibitors

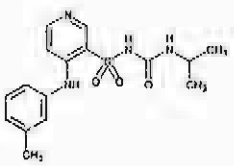


Loop Diuretics

Torsemide

- High ceiling loop diuretic
- Marketed as Demadex™
- Available as oral and parenteral products – generics available
- Detected and reported from horse urine in 2000s
- Readily detected by contemporary methods of analysis

Chemical Structure

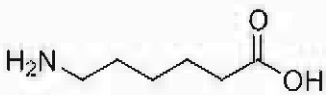


Fibrinolysis Inhibitors

Aminocaproic Acid

- Chemically similar to lysine
- Marketed as Amicar™
- Inhibits fibrinolysis
- Used in human medicine to treat excessive post-operative bleeding (*e.g.*, coronary artery bypass surgery)
- Not approved for use in horses
- Classified as "adjunct bleeder" medication
- Readily detected by contemporary methods of analysis

Chemical Structure



FIBRINOLYSIS INHIBITORS

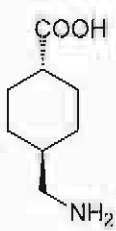
Drugs that inhibit clot dissolution

Fibrinolysis Inhibitors

Tranexamic Acid

- Chemically similar to lysine
- Marketed as Cyklokapron™
- Inhibits fibrinolysis
- Used in human medicine to treat excessive post-operative bleeding (*e.g.*, coronary artery bypass surgery)
- Not approved for use in horses
- Classified as "adjunct bleeder" medication
- Readily detected by contemporary methods of analysis

Chemical Structure



ANTIHEMORRHAGIC AGENTS

OTHER SUBSTANCES

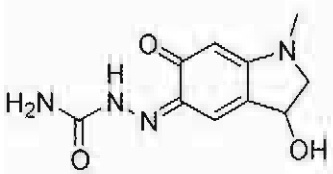
Conjugated estrogens and other substances

Antihemorrhagic Agents

Carbazochrome

- Oxidation product of epinephrine
- Component of Kentucky Red
- Promotes platelet aggregation and adhesion
- Not approved for use in the horse
- Readily detected using contemporary methods of analysis
- Identified as "adjunct bleeder" medication

Chemical Structure



Other Substances

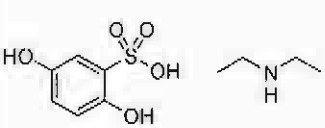
- Conjugated estrogens
  - Endogenous substances without thresholds
- Ergot alkaloids
  - Ergotamine
    - Vasoconstrictor
    - Readily detected

Antihemorrhagic Agents

Etamsylate

- Promotes platelet aggregation and adhesion
- Not approved for use in the horse – not approved for use in US
- Readily detected using contemporary methods of analysis
- Not identified as "adjunct bleeder" medication

Chemical Structure



CONCLUSIONS

ATTACHMENT #7

7/ LASIX PROTECTS AGAINST EPISTAXIS, FRANK BLEEDING FROM THE NOSTRILS:

In 1995 Lasix was approved in New York racing. New York maintained records on the incidence of Epistaxis, frank bleeding from the nostrils post race, and these records show the efficacy of pre-race Lasix in reducing Epistaxis. The New York approval of Lasix reduced the incidence of Epistaxis close to 80%, as shown in Attachment #6. This interpretation was supported by Dr. Anthony Verderosa, the New York Racing Association Chief Examining Veterinarian, who reported a *" >400% decrease"* in the incidence of Epistaxis following the introduction of Lasix. This was the first formal validation of the by then longstanding field observations and experience of American Horsemen with regard to Lasix and Epistaxis, Attachment #7.

THE NEW YORK RACING ASSOCIATION INC.
Anthony R. Verderosa, DVM
Chief Examining Veterinarian
P.O. Box 90, Jamaica, New York 11417-0090
(516) 351-5314

Catastrophic Racing Injuries 1990-1994: 1.3 / 1,000 starts

Post Race Epistaxis ("Bleeders") 1990-1994: 2.6 / 1,000 starts

Catastrophic Racing Injuries 1996-2000: 1.5 / 1,000 starts

Post Race Epistaxis ("Bleeders") 1996-2000: 0.6 / 1,000 starts

5 yr.(1990-1994 vs 1996-2000) statistical comparison:

Catastrophic Racing Injuries: 13% increase

Post Race Epistaxis ("Bleeders"): >400% decrease

NEW YORK THOROUGHBRED HORSEMEN'S ASSOCIATION, INC.

November 10, 2011

Dr. Thomas Tobin
Professor of Veterinary Science
Graduate Center for Toxicology
Room 128C
Maxwell H. Gluck Equine Research Center
University of Kentucky
Lexington, KY 40546-0099

Dear Tom,

Rick wanted me to send you a quick thank you note for providing us with a letter regarding Lasix protocols and the administration of same, which don't comprise the integrity of quality drug detection.

Enclosed is a copy of materials compiled to advance our perspective regarding this important and effective race day medication. Further, also enclosed is a comparative analysis done by Dr. Anthony Verderosa, the New York Racing Association's Examining Veterinarian, on catastrophic racing injuries and post race epistaxis in the years prior and after the permissive use of Lasix in New York in 1995.

Thanks again for your input and support.

Sincerely,

Jim Gallagher
Executive Director

Encs.

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Furosemide, the Prevention of Epistaxis and Related Considerations: A Preliminary Evaluation

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KEY WORDS: Furosemide, Epistaxis, Exercise Induced Pulmonary Hemorrhage [EIPH], Regulatory Approval, Acute/sudden death, Horses, Jockeys, United States.

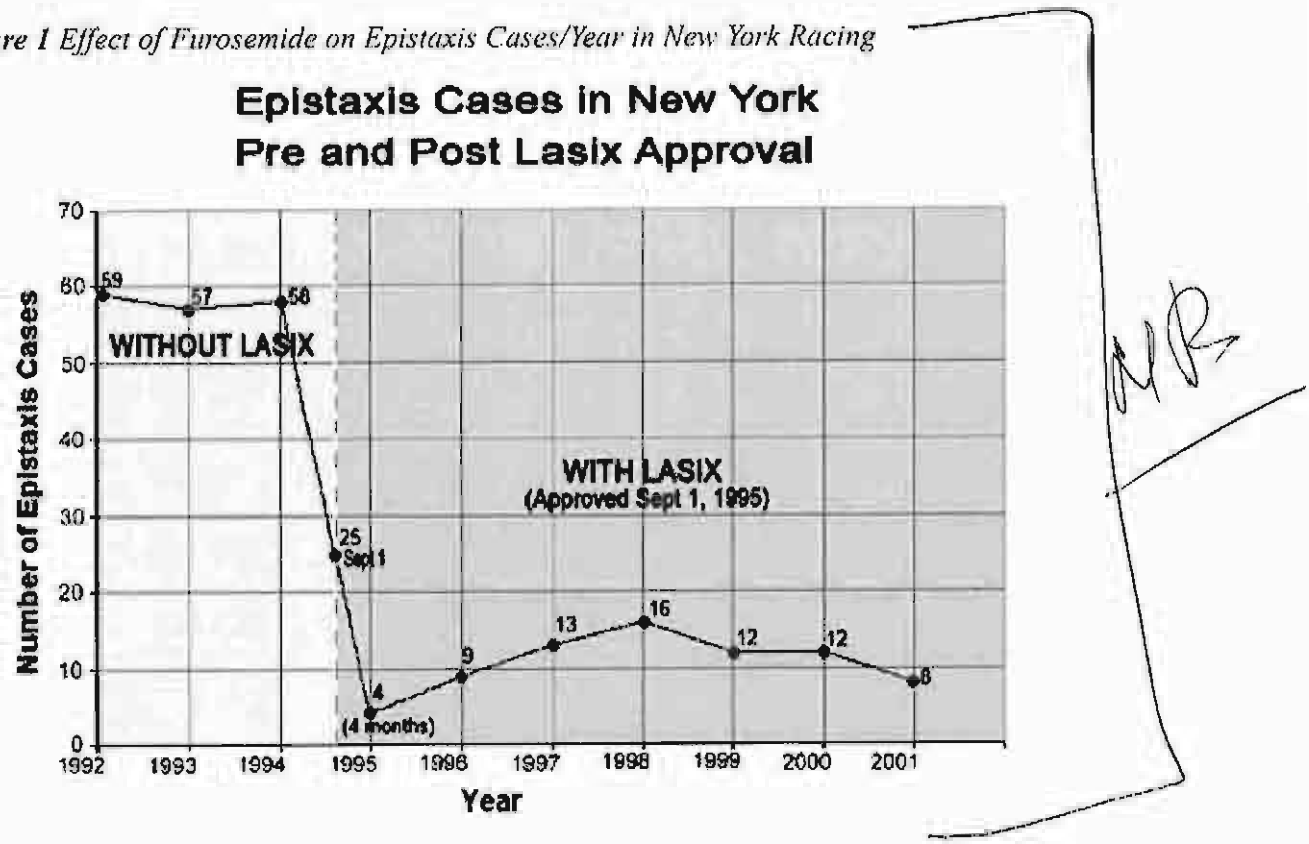
ABSTRACT

Furosemide is widely approved in the United States, Canada and elsewhere in the Americas for the prevention of Exercise Induced Pulmonary Hemorrhage [EIPH]. We review the scientific evidence for the efficacy of furosemide in reduction/ prevention of EIPH, including, presumably, EIPH related acute/sudden death in racing horses. We present evidence from the scientific literature and our own experience, clinical and otherwise [RHG, AMB, DVL, and TT], that EIPH driven acute/sudden death in racing horses has significant adverse health consequences for horses and jockeys. We then outline the adverse effects on equine and human [jockey] health and welfare to be expected when furosemide is not approved for use in racing horses or, where approved, if approval is withdrawn.

FINDINGS

Epistaxis, ie, bleeding from the nose in racing horses, has been observed by horsemen since at least the seventeenth century. [1] In the late 1960s, injectable furosemide (Lasix, Salix) became available in the United States and soon thereafter furosemide was

Figure 1 Effect of Furosemide on Epistaxis Cases/Year in New York Racing



being used in the prevention of epistaxis. This initial use of furosemide was based on clinical experience and, until recently, there was little scientific evidence concerning its efficacy in the prevention of epistaxis. On the other hand, most American racing states have long since approved the use of furosemide on race day for the prevention/alleviation of epistaxis, now known to be a component of Exercise-Induced Pulmonary Hemorrhage [EIPH]. Very recently, however, questions have been raised about whether or not the routine pre-race treatment of racing horses with furosemide for the prevention of epistaxis/EIPH passes "the smell test,"[2] which has led to renewed examination of the scientific basis for the pre-race use of furosemide in North American racing.

We now draw attention to some clinical evidence establishing, in large numbers of racing horses, the efficacy of furosemide in reducing the incidence of epistaxis. These data were first communicated by Mr. Bill Heller in his monograph on Lasix, "Run, Baby, Run,"[3] where these data have remained hidden in plain sight since the 2002 publication of this book. Reviewing this book as part of an overall review of the literature on furosemide in the horse, we noted, on pages 112 and 113, a table entitled "New York By The Numbers, Cases of Epistaxis," dated 1992 to 2001. Inspection of these data immediately clarifies the dramatic reduction in the incidence of EIPH in New York racing following the 1995 approval of furosemide. We now present this data in standard graphical format (Fig 1), which clarifies the remarkable efficacy of furosemide in reducing the incidence of epistaxis in horses racing in New York in the 6 years and 4 months immediately following approval of furosemide in New York racing.

In analyzing these data we note the following facts. The first is that epistaxis is, by definition, clearly observable bleeding ("dripping of blood") from the nose, sometimes defined as from both nostrils. Epistaxis is, however, only one manifestation of what is now known as Exercise-Induced Pulmonary Hemorrhage (EIPH).[4] It is also the only manifestation of EIPH observable without special equipment, and

as such, epistaxis has long been recorded in performance horses.[1-4] Epistaxis is thus the historical "low tech" manifestation of EIPH, with acute sudden death due to EIPH, only recently recognized by science, representing the most severe clinical outcome of this disease.[5,6,7]

All manifestations of EIPH other than epistaxis, including acute/sudden death due to EIPH, require scientific tools and expertise to identify as being EIPH related.[5] As such, the data represented in figure 1 reflect directly on the second most severe manifestation of EIPH, namely epistaxis, and these results speak directly and very compellingly to the efficacy of furosemide in reducing the incidence of both epistaxis and presumably the entire EIPH syndrome in all its various manifestations. The solid circles (O--O) show the total number of epistaxis cases in the previous 12 months of New York racing for 1992 to 2001, inclusive. Furosemide was approved in New York on Sept. 1, 1995, so the 1995 figures are split, with the number 25 representing cases prior to Sept. 1, and the number 4 representing cases from Sept. 1 to Dec. 31, 1995. The annual mean rate of EIPH cases prior to 1994 was 58 per year, and post Sept. 1, 1995, 11.6 per year. These data show that approval of furosemide for use in New York was associated with an immediate and well maintained essentially 80% reduction in the incidence of epistaxis in New York Racing. Data re-plotted from Heller, "Run, Baby, Run," 2002.[3]

With respect to the New York data, review of figure 1 shows that for the years 1992, 1993, and 1994 the numbers of epistaxis cases reported in New York racing were, respectively, 59, 57, and 58, a consistent average of about 58 epistaxis cases per year, which equates to approximately one case per week prior to approval of furosemide. Then, on September 1st, 1995, furosemide was approved for use in New York racing, and the number of epistaxis cases dropped immediately, to a total of 4 cases for the 4 remaining months, September to December, inclusive. In the following year, 1996, the total number of epistaxis cases was 9, followed by 13 in 1997, 16 in 1998, and 12 each in 1999 and 2000, and then a total of 8 cases in 2001, by which time the proportion of horses racing on furosemide in New York had increased to 88.3%. Overall, therefore, in the six calendar years starting on January 1, 1996 and continuing to December 31, 2001, there were a total of 70 epistaxis cases over 6 years, for an average of 11.6 epistaxis cases per year during the first 6 years of furosemide approval in New York.

We also note that this rate of about 11.6 cases of epistaxis per year was remarkably consistent, in that in the last 4 months of 1995, September 1st, 1995 to December 31st, 1995, the first 4 months during which New York raced on Lasix/Salix, there was a total of four cases of epistaxis reported, completely consistent with the subsequent overall average of 11.6 epistaxis cases per year for the following 6 years.

These data are very compelling, and the conclusion to be drawn is that approval of furosemide in racing horses in New York immediately, and we emphasize the word immediately, reduced the incidence of epistaxis by almost 80%. The effect was immediate because it was apparent within the first month of approval of furosemide, and the incidence of epistaxis remained, on average, at essentially the same reduced level in New York racing, 11.6 cases per year, or approximately one case per month, over the following six calendar years. We also note that this reduced rate of epistaxis represents a close to 80% reduction from the baseline 1992-1994 rate of approaching five epistaxis cases per month prior to the regulatory approval of furosemide for use in New York racing.

An unusual aspect of these data is that they have to our knowledge remained unrecognized in the scientific literature. New York was the last major U S racing state to permit use of furosemide, and as such, there was a clearly defined time point after which furosemide was permitted for use in New York. Given the reluctance of the authori-

ties to approve the use of furosemide in New York racing, it may be understandable that these data were not reported earlier. In any event, it is clear from these data that in the actual racing situation, furosemide is remarkably effective in reducing the incidence of epistaxis, and it would have been helpful if these data had been communicated earlier in the scientific literature.

The second point of interest is that these New York data almost certainly underestimate the true efficacy of furosemide in preventing epistaxis. Because New York did not allow the use of furosemide in racing horses, any New York area horses with a propensity to bleed were more likely to race in neighboring jurisdictions that permitted furosemide. Additionally, since EIPH is associated with reduced racing performance, this provided further incentive for horses with any tendency to EIPH to race outside of New York. As such, it is reasonable to assume that the baseline epistaxis rate of 58 per year reported for 1992-1994 actually underestimates the true baseline incidence of epistaxis in North American racing, because horses known to be EIPH prone would tend to have been raced outside of New York. These data, compelling as they are, showing an almost 80% reduction in the incidence of epistaxis after the introduction of furosemide, almost certainly underestimate the true clinical efficacy of furosemide in reducing the incidence of epistaxis.

These data also speak to the lack of effective alternative therapies for epistaxis, non-race day therapies or otherwise.[8-10] Where use of furosemide in the prevention of epistaxis/EIPH is prohibited, the likelihood of use of alternative epistaxis prevention therapies increases. If we make the reasonable assumption that horsemen racing in New York were likely to use any legitimate alternative therapy for epistaxis available to them that did not contravene the rules of racing in New York, we must again assume that the 1992-1994 EIPH incidence figure represent the incidence of epistaxis in the presence of whatever alternative therapies were available to New York horsemen. Again, the demonstrated efficacy of furosemide likely represents the effect of furosemide over and above any possible baseline reducing effect of other available anti-epistaxis therapies.[11]

We must also note that the original clinical observations that furosemide reduced the incidence of epistaxis in racing horses were made in the late nineteen sixties and early nineteen seventies by equine veterinarians and horsemen soon after the introduction of injectable furosemide. These observations were made prior to the introduction of the fiberoptic endoscope and our resulting increased understanding of EIPH and its prevalence in racing horses. The observations reported here fully support these early insightful clinical observations and interpretations and the various decisions since then by equine veterinarians, horsemen and racing authorities to support the prerace use of furosemide in the prevention of epistaxis, as it was then understood, and the entire EIPH syndrome, as it is now understood. As such, these findings leave no reasonable room for doubt that furosemide will also reduce the incidence of acute/sudden deaths during racing due to EIPH.

As well as the data reported here, it is also appropriate for us to acknowledge the recent (2009) highly significant contribution by Hinchcliff, Morley, and Guthrie in this area.[12] These workers performed a classic randomized, blinded, placebo-controlled crossover study on the efficacy of furosemide in preventing EIPH in racing horses. This study established that pretreatment with furosemide reduced the incidence of EIPH in 167 thoroughbred horses under simulated racing conditions at Vaal Racecourse, Johannesburg, South Africa, at, we might note, an altitude of 4,671 ft.

While this study provides strong experimental evidence that pretreatment with furosemide reduces the intensity of EIPH in horses under racing conditions in South Africa, this study did not directly address the efficacy of furosemide in reducing the incidence of epistaxis. We respectfully sug-

Figures 2a and 2b: *Acute/Sudden Death due to EIPH as an Equine and Jockey Safety Hazard*

These photographs record an acute/sudden death EIPH incident in U S Quarter Horse Racing. The horses were moving at approaching 50 mph; the far horse is crashing to the track associated with an acute/sudden EIPH event, and the jockey is being thrown onto the track. The close-up, figure 2b, highlights the blood in the horse's exhalation, consistent with these events being triggered by an acute/sudden EIPH episode. Centennial Racetrack in Littleton, Colorado, altitude 5,389 ft., photographs courtesy of Dr. Richard H. Galley, Willow Park, Texas.

gest that the New York data presented here provides further evidence that is fully supportive of the results obtained by Hinchcliff and his colleagues in their South African study, and extends the clinical efficacy of furosemide to the very effective prevention of epistaxis. Together these studies, as well as approaching 40 years of accumulated clinical experience make an extremely strong scientific case for the use of furosemide in the prevention/alleviation of the EIPH group of syndromes, including the propensity of EIPH to produce acute/sudden death in racing horses.[5,13-17]

Acute/sudden deaths during racing due to EIPH occur when the hemorrhage is sufficiently voluminous to acutely interfere with respiration/blood oxygenation, such that the horse collapses and dies acutely on the racetrack. Although acute/sudden death during racing due to EIPH has long been known to equine practitioners, more recent work has shown that acute/sudden death from EIPH can occur without blood being visible at the nostrils, as shown by the work of Gunson and her colleagues [1988] and others[5], Morales et al [7] and work from our group.[17] Based on this work, it is now very clear that one of the outcomes of EIPH is acute/sudden death of a horse during racing with no obvious external signs of epistaxis. Review of the relevant literature, including the 1988 paper by Gunson and coworkers suggests that in the U S such acute deaths during racing and training occur approximately once per 1,500 Thoroughbred races, and that a substantial proportion, approximately 60% of acute deaths during racing are due to EIPH.[5] As such, the data presented here suggests that pretreatment with furosemide is likely to reduce the instance of such EIPH related sudden deaths during racing by approximately 80%, a very significant contribution to equine and human safety in racing.

Furthermore, we must also note that the Gunson analysis almost certainly underestimates the true incidence of EIPH related acute deaths in racing horses, since this study was carried out after use of furosemide had been approved in Pennsylvania racing (personal communication, Dr. Corinne Sweeney to TT, July 2011). As such, a true estimate of the incidence of EIPH related acute/sudden deaths in racing horses under U S conditions in the absence of furosemide is likely to be substantially greater than the once per 1,500 thoroughbred races reported by Gunson and coworkers, with the expected EIPH related acute sudden deaths figure in the absence of furosemide being between

four and five fold greater, based on the data of figure 1.

With respect to the matter of equine and human safety, the question then becomes what is the incidence of equine and human deaths in racing caused by the EIPH driven acute/sudden death syndrome? At this point, we definitively know from the experience of four of us [RHG, AMB, DVL, and TT] that such EIPH related acute death events occur in racing horses, and that these events also carry clear and highly significant risks for jockeys. The incident represented in figures 2a and 2b occurred at Centennial Racetrack in Littleton, Colorado, in the early to mid 1970's, shortly prior to approval of furosemide for use in Quarter Horse Racing in Colorado. One of us, RHG, was the treating veterinarian. The photograph was taken as the horses reached the finish line and fortunately, as may happen in Quarter Horse racing, there were no horses directly behind the horse that went down. The jockey in question was slightly injured, but the horse did not survive the incident. This photograph therefore records a not an atypical EIPH related acute/sudden death event in Quarter Horse racing, which incident resulted in one equine death and, in this particular case, minor injuries to the jockey. The incident presented in figure 3 represents another EIPH related sudden death incident in racing, this time, in Thoroughbred racing at Ruidoso Downs, New Mexico, altitude 6,720 ft, in the mid 1980s. RHG was again the treating veterinarian and the horse in question was not treated with furosemide. In this particular incident, once the acute/sudden death horse went down, two following horses "went over" the "down' horse/jockey. The horse died on the racetrack, and the jockey sustained career ending injuries [RGH].

These photographic presentations emphasize that EIPH related acute/sudden deaths of horses on the racetrack are not infrequent occurrences, and when they do occur, they have immediate highly significant implications for the health and welfare of the horses and jockeys involved. Additionally, we must also keep in mind that when a horse goes down in a racing situation, there is always a statistical probability of following horses and jockeys becoming involved in the event, and this sequence of secondary events is more likely to occur in Thoroughbred than in Quarter Horse racing.

Figure 3: *EIPH related sudden death. Thoroughbred racehorse, Ruidoso Downs,NM, mid 1980s. Photograph courtesy of Dr. Richard H. Galley, Willow Park, Texas.*

These findings are in good agreement with the clinical experiences of two of us, Dr. A. Morales Briceño and Dr. Diana Villoria Leon, presented in detail elsewhere in their recently published work [2011].[7] Reporting on the incidence of EIPH related acute/sudden death in racing at "Hippodromo La Rinconada," the National Racecourse in Caracas, Venezuela, they recorded 23 cases of acute/sudden death due to/caused by EIPH. These diagnoses were confirmed by full diagnostic necropsies and toxicological examination for medications related to EIPH, which evaluations were performed on each individual animal over the 3- year period from 2008 to 2011, inclusive.

Over these 3 years at "Hippodromo La

Rinconada," there were a total of 44,928 starts, and this population of starters yielded 23 acute /sudden deaths that were on necropsy confirmed as being due to or caused by EIPH. This amounts to one EIPH related acute/sudden death per 1,953 starts. Additionally, we [AMB, DVL] noted the incidence of jockey injuries associated with these EIPH events, which involved 85% of the jockeys riding these horses. Based on this percentage jockey injury rate, there was one jockey injury from EIPH related acute/ sudden death per 2,298 starts over this 3-year period of racing at La Rinconada. At this time, however, we have no data on the nature and severity of the injuries sustained by the jockeys involved in these EIPH related acute/sudden death incidents in Caracas.

This rate of acute/sudden deaths caused by EIPH in racing in Caracas is significantly higher than the rate reported by Gunson and co-workers, who estimated one EIPH related sudden death per 1,500 races, with 9 horse fields. On this basis, the Pennsylvania rate works out at about one EIPH driven acute/ sudden death event per 13,500 starts. This approximately seven fold higher EIPH acute /sudden death rate in Caracas compared with the estimated rate in Pennsylvania is unexpectedly large, and the reasons for this difference are not immediately apparent.

One major difference between the Pennsylvania and Caracas racetracks is the higher altitude of the Caracas racetrack. This racetrack, La Rinconada, at about 2,950 feet above sea level, is elevated compared with Penn National racecourse, at an elevation of 459 feet, and Pennsylvania Park, at an elevation 36 ft. Similarly, Centennial Racetrack, Littleton, Colorado, is at an altitude 5,389 feet and Ruidoso Downs, New Mexico, is at an altitude of 6,720 feet, as noted in figs 2a and 2b and 3.

With respect to the effect of altitude, we note that the principal scientific report to date on the relationship between altitude and EIPH is that of Weideman et al [2003][18] who reported that in South Africa EIPH appeared to be more frequent at sea level than at higher altitudes. If this interpretation is correct it suggests that the altitude of the Vaal Racecourse, Johannesburg, South Africa, at 4,671 ft., made the Hinchcliff et al [12] demonstration of the preventative effect of lasix on EIPH more challenging than such a demonstration would have been at sea level. This interpretation is also consistent with the unusually small number of class 4 EIPH scores in the Hinchcliff data, as pointed out by a colleague in discussions on this matter. Additionally, as a further confounding factor in these EIPH acute/sudden death studies, we note the significant variability in pathological diagnoses on entire equines[16, 17] presumably due at least in part to the extremely large volume of equine tissue to be subjected to histopathological analysis during equine necropsies.

In summary, for reasons that are unclear, and apparently unrelated to altitude, thoroughbred horses racing in Caracas, Venezuela, show an unusually high incidence of EIPH associated acute/sudden deaths, about one EIPH associated acute/sudden deaths per 1,953 starts. This acute/sudden death rate is about six-fold the rate reported in the earlier Pennsylvania study, and the reason or reasons for these differences are not clear. The fact that racing at La Rinconada is at about 2,950 feet above sea level is considered by Weideman et al. to work against the apparent discrepancy, since he and his colleagues consider that altitude above sea level is associated with a reduced incidence of EIPH. We also note, however, that the concept of increased altitude reducing the rate of EIPH is not consistent with the clinical experience of veterinarians, including one of us [RGH], working in the Western United States, where racing takes place at attitudes of up to 6,720 feet at Ruidoso Downs, New Mexico. Unrelated to the role of altitude, however, is the hard reality that 85% of the jockeys involved in EIPH related acute/ sudden death events in horses racing at La Rinconada suffered injury, and that although the extent and severity of these injuries are not available to us, at least one jockey in the

experience of one of us [RHG] suffered a career ending injury associated with acute/sudden death from EIPH [fig 3].

From the data of Gunson and her colleagues we can conservatively estimate the number of EIPH related acute/sudden death related events likely to occur in one year in American Thoroughbred racing. Based on their data, Gunson and their colleagues estimated about one EIPH related acute sudden death per 1,500 starts. Gunson reports nine horse fields, which works out at one EIPH related acute sudden death per 13,500 starts. There are about 417,492 plus thoroughbred racing starts per year in the United States, so this works out at about 31 acute/sudden-death EIPH related events /year in thoroughbred racing in the United States, on the assumption that furosemide is permitted. In the absence of furosemide, however, based on the data in figure 1, we may expect a four to five fold increase in the number of acute sudden-death EIPH related events, to around 155 events/year, or more than three per week.

Gunson also noted that 2 of her 9 reported EIPH cases were acute sudden death in not racing horses, so the final figure is about 120 acute sudden death cases in U S racing per year based on Gunson's data. However, what it is not possible to estimate at this time is the number and intensity of the jockey injuries likely to be associated with these 120 more or less EIPH related acute/sudden-death events in American racing per year in the absence of furosemide, although most of these injuries are likely to be highly significant for the actual individuals involved. [fig 3].

CONCLUSION

These scientific findings, therefore, have implications far beyond equine health and welfare. This is because while pretreatment of racing horses with furosemide serves to reduce the incidence of epistaxis and the various equine pulmonary syndromes associated with intense exercise and EIPH by about 80%, there is every reason to believe that furosemide also serves to reduce, again by about 80%, the incidence of EIPH driven acute/sudden death syndrome in horses in training and racing. By definition, such EIPH related acute/sudden death incidents have the potential to cause severe, including career ending [fig 3] and potentially fatal injuries to jockeys and others riding these horses. As such, the currently in place regulatory approval for use of furosemide in the prevention of EIPH related syndromes in racing horses has a very direct positive and ongoing protective effect on the health and safety of jockeys racing in the United States and elsewhere in the Americas.

Given these scientific realities, we respectfully suggest that it would be unethical and inappropriate, on humane grounds with respect to equine health and welfare, and also on humane and workplace safety grounds with respect to jockeys, for any entity to ban the use of furosemide in racing horses. This is because to do so would be to knowingly significantly increase the risk of serious injury or death for jockeys or others riding racing/performance horses. In sum, any move to disapprove or to withdraw approval for furosemide as an EIPH preventive in racing horses is, from review of the available scientific literature, a move that will immediately and directly increase the risk to life and limb for both the horses and jockeys involved in racing or any other equine event involving exercise sufficiently intense to induce pulmonary hemorrhage.

LIST OF ABBREVIATIONS

[EIPH] Exercise-Induced Pulmonary Hemorrhage

COMPETING INTERESTS

The authors declare no competing interests

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NOTE ADDED IN PROOF

With respect to the data of figure #1 re-plotted from Heller 2002, our attention has recently been drawn to an undated memorandum on the New York Racing Association (NYRA) letterhead of Dr. Anthony Verderosa, DVM, Chief Examining Veterinarian. In this memorandum Dr. Verderosa notes that his analysis of the data on the rates of epistaxis in New York racing for the years 1990 to 2000, that is before and after the introduction of furosemide, showed that the introduction of furosemide in 1995 produced a ">400% decrease" in post race Epistaxis ("Bleeders") EIPH. This is essentially the same conclusion that we drew from what are presumably the same data presented in the Heller book, and appear to fully and independently support our analysis and conclusions presented herein concerning the clinical efficacy of furosemide in preventing epistaxis in New York Racing.

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ATTACHMENT #8

88/ LASIX ALSO PROTECTS AGAINST TRACHEAL EIPH:

A definitive Hinchcliff et al 2009 study performed in South Africa on 167 or so horses showed that pretreatment with Lasix reduced the incidence and severity of tracheal EIPH, again validating the long standing field experience of American Horsemen, Attachment #8 Additionally, a more recent 2015 consensus study authored on Lasix and EIPH authored by Hinchcliff and his colleagues concluded there was *"moderate to high quality evidence that EIPH is progressive . . . ; that it adversely affects racing performance; that severe EIPH is associated with a shorter career duration; [and], that furosemide is efficacious in decreasing the incidence and severity of EIPH,* Attachment #8.

EQUINE

Efficacy of furosemide for prevention of exercise-induced pulmonary hemorrhage in Thoroughbred racehorses

Kenneth W. Hinchcliff, BVSc, PhD, DACVIM; Paul S. Morley, DVM, PhD, DACVIM; Alan J. Guthrie, BVSc, PhD

Objective—To evaluate the efficacy of furosemide for prevention of exercise-induced pulmonary hemorrhage (EIPH) in Thoroughbred racehorses under typical racing conditions.

Design—Randomized, placebo-controlled, blinded, crossover field trial.

Animals—167 Thoroughbred racehorses.

Procedures—Horses were allocated to race fields of 9 to 16 horses each and raced twice, 1 week apart, with each of the 2 races consisting of the same race field and distance. Each horse received furosemide (500 mg, IV) before one race and a placebo (saline solution) before the other, with the order of treatments randomly determined. Severity of EIPH was scored on a scale from 0 to 4 after each race by means of tracheobronchoscopy. Data were analyzed by means of various methods of multivariable logistic regression.

Results—Horses were substantially more likely to develop EIPH (severity score ≥ 1; odds ratio, 3.3 to 4.4) or moderate to severe EIPH (severity score ≥ 2; odds ratio, 6.9 to 11.0) following administration of saline solution than following administration of furosemide. In addition, 81 of the 120 (67.5%) horses that had EIPH after administration of saline solution had a reduction in EIPH severity score of at least 1 when treated with furosemide.

Conclusions and Clinical Relevance—Results indicated that prerace administration of furosemide decreased the incidence and severity of EIPH in Thoroughbreds racing under typical conditions in South Africa. (*J Am Vet Med Assoc* 2009;235:76–82)

Horse racing is a popular, multimillion-dollar industry worldwide, but reports of injuries and other physical disorders in racehorses have harmed public perceptions of the sport and challenged the economic viability of the racing industry. In addition, controversy has been generated by use of medications that are perceived to affect the performance or well-being of racehorses. One of the foremost concerns in this regard is the occurrence of EIPH and the use of medications in an attempt to prevent it. Factors that make this an important issue include the frequency of EIPH, the importance of the disease in terms of the performance and well-being of horses, and the common use of prophylactic treatments. At least 80% of racehorses can be expected to develop the condition at some time during their career,[1,2] approximately 60% of sudden deaths during racing have been attributed to pulmonary hemorrhage,[2] severe EIPH has been shown to adversely affect race performance,[3] and EIPH is believed to adversely affect the overall health of racehorses.[4] Beyond this, management and treatment of EIPH have a substantial economic impact, with the cost of treating EIPH estimated to exceed $100 million annually in the United States alone.[4]

Abbreviations

EIPH	Exercise-induced pulmonary hemorrhage
IQR	Interquartile range
NHRA	National Horse Racing Authority of South Africa
OR	Odds ratio
R	South African Rand

Furosemide is the drug most widely used to prevent EIPH in racehorses and is administered on the day of racing to > 92% of Thoroughbred racehorses in North America (approx 400,000 doses/y).[4,5] However, few studies have examined whether furosemide is effective in preventing the development of EIPH, and the studies that have been performed were not conducted

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under actual racing conditions. Given this lack of evidence and the finding that furosemide can improve the performance of Thoroughbred racehorses,[6] the use of furosemide to prevent EIPH remains controversial. The purpose of the study reported here, therefore, was to evaluate the efficacy of furosemide for the prevention of EIPH in Thoroughbred racehorses racing under typical conditions.

Materials and Methods

Study design—The study was conducted as a randomized, placebo-controlled, crossover field trial. All study participants, including data analysts, were blinded to treatment assignments until statistical analyses related to the primary outcome were completed. The study was conducted at the Vaal Racing and Training facility in Free State Province, Republic of South Africa, between November 20 and 28, 2007, and the study protocol was approved by the institutional animal care and use committees of the University of Pretoria and Colorado State University. For all horses participating in the study, the owner or his or her designee (ie, the trainer) provided informed consent.

Experimental protocol—In an attempt to include horses broadly representative of all horses racing in South Africa, the study was announced at public meetings of trainers, during television programs devoted to horse racing, via racing Web sites, in text messages to trainers, and in advertisements in the local print media inviting owners and trainers to nominate horses for inclusion in the study. Horses considered eligible for participation were Thoroughbred racehorses registered with the NHRA and trained by licensed trainers. Horses were enrolled without knowledge of whether they had previously had EIPH, with the exception that horses with a history of epistaxis during racing or training that had been documented by a veterinarian or steward employed by the NHRA were excluded. At the time horses were nominated for inclusion in the study, the owner or trainer was asked to indicate the specific race or races (eg, 1,300-m race with colts and geldings that had merit ratings ≤ 76) designated for the study during which the horse would be allowed to race.

Horses accepted for inclusion in the study were assigned to race fields on the basis of age, sex, and race record by a professional handicapper who also assigned handicap weights, with each race field consisting of 9 to 16 horses. Enrolled horses raced twice, 7 days apart, with each of the 2 races consisting of the same race field (with the exception of horses withdrawn from the study prior to the second race) and same race distance. Horses carried the same weight, were ridden by the same jockey, started from the same barrier stall, and wore identical tack during the 2 races. Races were run over turf according to the rules of racing of the NHRA, with the exception that administration of furosemide or a placebo prior to each race was permitted for purposes of the present study. In accordance with NHRA rules, blood and urine samples were obtained from selected horses after each race and tested for prohibited medications, including NSAIDs. Owners of horses included in the study were paid a participation fee of R2,000 on completion of the second race. In addition, prize money was paid to the owners of horses that finished first (R28,750), second (R9,200), third (R4,600), fourth (R2,300), or fifth (R1,150) in each race. Prior to each race, trainers were allowed to withdraw (scratch) horses from the race in accordance with the standard rules of racing. Horses that were withdrawn prior to the first race were not allowed to participate in the second race.

Trainers were required to bring participating horses to the racetrack 4.5 hours before the scheduled start time of the race in which they were to compete. As each horse arrived at the track, study personnel confirmed the identity of the horse by checking for a microchip and applied an adhesive tag with a unique identifying number to the mane. Horses were then weighed, placed in stalls, and attended by their grooms. Access to food and water was denied from 4 hours prior to racing until after a tracheobronchoscopic examination was performed following completion of the race. Thirty minutes before the scheduled start of the race, horses were again weighed and moved to the saddling enclosure.

Four hours (± 5 minutes) before the scheduled start of the race, horses were treated with furosemide or a placebo. Each horse received furosemide before one race and a placebo before the other. Treatment order (furosemide prior to the first race and placebo prior to the second race vs placebo prior to the first race and furosemide prior to the second race) was randomly determined by assigning a computer-generated random number to every horse prior to the first race. The first half of each field, as determined by these random numbers, was assigned to receive furosemide prior to the first race and a placebo prior to the second race. The second half of each field was assigned to the opposite treatment order.

Randomization and treatment assignment were performed by an investigator who was not involved in administering any treatments on race days. Individual doses of furosemide[a] (500 mg) and a placebo solution were prepared for all horses prior to the initiation of the study. Each syringe contained 10 mL of solution, and syringes were labeled with horse identification number, race number, and race day. The furosemide solution that was used for the present study had a slight yellow color. Therefore, the placebo solution consisted of saline (0.9% NaCl) solution to which a vitamin B complex solution[b] (0.1 mL/1,000 mL of saline solution) had been added as a coloring agent. Because each 10-mL dose of the placebo solution contained only 0.0001 mL of the vitamin B complex solution, it was considered unlikely to have had any clinically important biological effect, and vitamin B complex solution was not added to the furosemide solution. Furosemide and placebo solutions were administered by IV injection into a jugular vein. Blood samples were collected 15 minutes after treatments were administered and tested for furosemide concentration to verify that the correct treatment had been given.

All races started within 4 minutes of the scheduled start times. At the end of each race, horses were returned to the parade ring, where they were examined by veterinary officials from the NHRA and their tack

was removed. A tracheobronchoscopic examination was then performed. All tracheobronchoscopic examinations were performed by one or the other of 2 teams consisting of 2 veterinarians and 2 lay assistants each. Individuals performing the tracheobronchoscopic examinations were experienced in the procedure, were provided information on the general study protocol, and were specifically asked to thoroughly examine the pharynx, larynx, and trachea to the level of its bifurcation. However, they were blinded to treatment group assignment. All examinations were directly overseen by one of the authors (KWH) and were digitally recorded. After completion of the tracheobronchoscopic examination, horses were released to the care of their trainers.

Maximum environmental temperature on race days ranged from 21.1° to 27.6°C (70.0° to 81.7°F), and minimum environmental temperature ranged from 18.9° to 25.6°C (66.0° to 78.1°F). Maximum humidity ranged from 18% to 73%, and minimum humidity ranged from 14% to 55%. Wind speed during the times that horses raced ranged from 3.4 to 9.2 m/s. A total of 2 mm of rain fell during the time that horses raced on the first race day; 4.2 mm of rain fell on the last of the 4 race days, although this fell after completion of the last race that day.

Assessment of EIPH severity—Digital recordings of each of the tracheobronchoscopic examinations were reviewed by 3 individuals experienced in endoscopic examination of the airway in horses. Individuals scoring the recordings were blinded to identity of the horses and treatment group assignments.

Scoring of EIPH severity was performed by all 3 individuals concurrently, with the digital recording displayed on a large-screen television. Each individual was asked to assign a score from 0 to 4 for severity of EIPH on the basis of a previously reported validated scoring system.[7] Individual scores were then discussed, and if necessary, the examination was reviewed to obtain a consensus score, with consensus scores used in all data analyses.

Data analysis—During design of the study, sample size calculations were performed with standard commercial software.[a] For these calculations, it was assumed that if furosemide were efficacious, the proportion of horses with an EIPH score ≥ 2 would be ≤ 10% following treatment with furosemide, compared with an assumed baseline prevalence of 20% when horses were not treated with furosemide,[3] and that the mean ρ value for repeated observations among subjects would be 0.4. When the α error rate was set at 0.05, sample size calculations indicated that approximately 150 horses would need to complete both arms of the study to achieve a β error rate of 0.2. Assuming that a maximum of 20% of the study subjects would be withdrawn between the first and second arms of the study and that race fields would achieve a minimum of 90% subscription through the use of typical race enrollment methods, we calculated that 12 races with a maximum of 16 horses starting in each race would be required for each arm of the study. No rules for stopping the study or interim analysis of results were put in place.

The primary study outcome was the score for severity of EIPH as determined by means of tracheobronchoscopy. Continuous data were summarized as median and IQR because data were generally not normally distributed, with the exception that differences between pre- and posttreatment body weights of horses were normally distributed and were summarized as mean and SE and elapsed times between the start of racing and tracheobronchoscopy were normally distributed and were summarized as mean and SD. For horses that completed both arms of the study, the EIPH severity score after treatment with furosemide was compared with severity score after treatment with placebo, and the difference between scores was summarized as mean and SD; the Wilcoxon signed rank test was used to determine whether the median difference between scores was significantly different from 0. The Wilcoxon rank sum test was used to compare ordinal and continuous data between groups, and the χ^2 test of homogeneity was used to compare categorical data between groups. The Bowker symmetry test was used to compare paired EIPH severity scores for horses that completed both arms of the study.

Scores for endoscopic severity of EIPH could not be analyzed in their native form (ie, scores of 0 to 4) by means of proportional odds, multinomial logistic regression because assumptions of proportionality were not met. Therefore, scores were dichotomized (0 vs 1 to 4 and 0 or 1 vs 2 to 4) to allow analysis by means of logistic regression. Because various methods have been proposed for analysis of data from crossover studies with binomial outcomes,[8–10] mixed-effects, repeated-measures fixed-effects, and conditional logistic regression models were all used to analyze dichotomized scores. Horse identity was nested within treatment sequence in these analyses to account for random and repeated effects. The primary exposure of interest was treatment (furosemide vs placebo); however, sex, race distance, age, and treatment sequence (furosemide prior to the first race and placebo prior to the second race vs placebo prior to the first race and furosemide prior to the second race) were also evaluated as fixed effects in mixed-effects and repeated-measures modeling. It was not possible to analyze sex, race distance, or age in conditional logistic regression models, as there were no differences in these exposures for paired observations. Age (≤ 3 years old vs ≥ 4 years old) and race distance (1,000, 1,300, or 1,600 m) were analyzed as categorical fixed effects. Exposure variables were analyzed for simple associations with outcome and were included in models with the primary exposure of interest (treatment). Confounding was investigated in multivariable models by evaluating the change in parameter estimates that occurred when variables were included or excluded from the model. Confounding was considered to be present when estimates changed by ≥ 20%. Effect modification was investigated by inclusion of first-order interaction terms. Treatment sequence was included as a random or repeated effect in each model, regardless of whether a significant association could be identified, when treatment sequence was analyzed as a fixed effect. This was considered a conservative method of accounting for incomplete washout,[8–10] even though incomplete washout was not expected.

It was not possible to analyze data on an intent-to-treat basis because tracheobronchoscopy is not routinely

performed after racing and occurrence of EIPH was not known for horses that did not participate. Therefore, data were analyzed on a per-protocol basis. However, use of repeated-measures and mixed-effects logistic regression allowed inclusion of data for horses that only completed the first race (as opposed to requiring that horses complete both arms of the study to be included in analyses), which provided some assurance that missing data for horses that were withdrawn (scratched) did not strongly bias the conclusions of the study.

Analyses were performed with commercial software.[d] A priori, values of $P \leq 0.05$ were determined to be significant.

Results

A total of 328 horses were nominated for inclusion in the study. Of these, 193 (77 females and 116 stallions and geldings) were enrolled in the study by the professional handicapper. Of the 193 horses enrolled in the study, 155 competed in both races, 12 competed only in the first race, and 26 did not compete in either race (Table 1). Horses that participated in the study were from 40 stables (median, 3.5 horses/stable; range, 1 to 14 horses/stable). Twenty-three trainers withdrew at least 1 horse from a study race. Demographic characteristics of horses that did not compete in either race did not differ significantly from characteristics of horses that competed in at least 1 race (Table 2).

Two horses that competed in both races would not allow tracheobronchoscopy to be performed after either race because of their fractious nature, and 1 horse would not allow tracheobronchoscopy to be performed after the second race. Mean ± SD time between the start of racing and tracheobronchoscopy was 41.6 ± 5.9

Table 1—Details of racing conditions for Thoroughbred racehorses enrolled in a study of the efficacy of furosemide for prevention of EIPH.

Race day	Race No.	Distance (m)	Class	Horses nominated	Horses enrolled*	Raced in first race	Raced in second race
A	1	1,300	Maiden fillies	38	18	15	12
A	2	1,300	Maiden colts and geldings	32	17	14	14
A	3	1,300	Maiden colts and geldings	31	18	15	14
A	4	1,600	Maiden colts and geldings	27	14	14	13
A	5	1,600	Maiden colts and geldings	26	14	13	11
A	6	1,600	Maiden fillies	43	18	15	13
B	1	1,000	Fillies and mares (merit ratings ≤ 68)	22	13	9	9
B	2	1,000	Colts and geldings (merit ratings ≤ 72)	37	18	16	16
B	3	1,300	Colts and geldings (merit ratings ≤ 76)	56	18	15	13
B	4	1,300	Fillies and mares (merit ratings ≤ 72)	39	16	15	14
B	5	1,600	Fillies and mares (merit ratings ≤ 68)	35	12	12	12
B	6	1,600	Colts and geldings (merit ratings ≤ 68)	38	17	14	14
			Total	**328**	**193**	**167**	**155**

Of the 328 horses nominated for inclusion in the study, 235 were nominated for 1 race, 90 were nominated for 2 races, and 3 were nominated for 3 races. Horses enrolled in the study raced twice, 7 days apart, with each of the 2 races consisting of the same race field (with the exception of horses withdrawn from the study prior to the second race) and same race distance. Each horse received furosemide (500 mg, IV) before one race and a placebo (saline solution) before the other, and severity of EIPH was scored immediately after the race by means of tracheobronchoscopy.

*Included starters and reserves; the maximum number of horses in each race was 16 starters and 2 reserves.

Table 2—Demographic characteristics of Thoroughbred racehorses enrolled in a study of the efficacy of furosemide for prevention of EIPH.

Variable	Nominated but not enrolled	Raced at least once	Enrolled but did not race	*P* value
No. of horses	135	167	26	NA, NA
Age	4 (3–5)	4 (3–4)	4 (4–5)	0.39, 0.12*
Sex				0.86, 0.34†
Stallion	9	13	0	
Gelding	69	88	15	
Female	57	66	11	
Assigned weight (kg)	NA	57 (56–58)	58 (55–58)	NA, 0.45*
Merit rating‡	65 (55–72)	65 (59–69)	58 (54–65)	0.28, 0.02*
Lifetime No.				
Starts	12 (5–21)	10 (3–22)	12 (8–21)	0.35, 0.19*
First-place finishes	1 (0–2)	0 (0–1)	1 (0–1)	0.06, 0.79*
Second- and third-place finishes	2 (0–5)	2 (0–4)	2 (1–5)	0.50, 0.15*
Finishes earning money	4 (2–9)	4 (0–9)	4.5 (3–8)	0.56, 0.35*
Lifetime earnings ($)	536,250 (220,000–1,018,700)	60,850 (23,000–111,745)	48,750 (23,550–94,490)	0.21, 0.42*

Data are given as median (IQR) or number of horses. *P* values are given as the *P* value for comparisons between horses that were nominated but not enrolled and horses that were enrolled, followed by the *P* value for comparisons between horses that raced at least once and horses that were enrolled but did not race.

**P* value from Wilcoxon rank sum test. †*P* value from χ^2 test of homogeneity. ‡Excludes maidens.

NA = Not applicable.

minutes when horses were treated with furosemide and 42.1 ± 6.0 minutes when horses were treated with saline solution. These values were not significantly (P = 0.63) different.

Scores for endoscopic severity of EIPH ranged from 1 to 4 in 89 of 161 (55.3%) horses after administration of furosemide and in 125 of 156 (80.1%) horses after administration of saline solution (Figure 1); these proportions were significantly (P < 0.001) different. For the 152 horses examined after both races, 87 (57.2%) had EIPH (ie, severity score ≥ 1) after administration of furosemide, whereas 120 (78.9%) had EIPH after administration of saline solution (**Table 3**). None of the horses had severe EIPH (ie, a score of 3 or 4) after administration of furosemide. Overall, 81 of the 120 (67.5%) horses that had EIPH after administration of saline solution had a reduction in EIPH severity score of at least 1 when treated with furosemide. Mean ± SD reduction in EIPH severity score after furosemide administration in the 120 horses that had EIPH after administration of placebo was 0.63 ± 0.08; median reduction in EIPH severity score was significantly (P < 0.001) different from 0.

Results of mixed-effects, repeated-measures fixed-effects, and conditional logistic regression analyses all indicated that horses had significantly lower odds of developing EIPH (ie, severity score ≥ 1) or moderate to severe EIPH (ie, severity score ≥ 2) following administration of furosemide, compared with odds following administration of saline solution (**Table 4**). Horses were 3.3 to 4.4 times as likely to have an EIPH score ≥ 1 following administration of saline solution than they were following administration of furosemide and were 6.9 to 11.0 times as likely to have an EIPH score ≥ 2 following administration of saline solution than they were following administration of furosemide.

Although results of mixed-effects and repeated-measures fixed-effects logistic regression suggested that horses that were ≥ 4 years old were more likely to develop EIPH (ORs, 1.8 and 1.9, respectively; P = 0.04 and 0.07, respectively), no effect modification (ie, an interaction between age and treatment) was detected, and age did not appear to be a confounding variable in these analyses. Development of EIPH was also not associated with sex (P = 0.30 and 0.38, respectively), distance raced (P = 0.38 and 0.99, respectively), or treatment sequence (P = 0.69 and 0.99, respectively) in these analyses.

Mean ± SE weight loss during the 4 hours prior to the start of the race was 12.7 ± 0.33 kg (27.9 ± 0.73 lb) when horses were given furosemide (n = 160) and 5.4 ± 0.28 kg (11.9 ± 0.62 lb) when horses were given saline solution (155). These values were significantly (P < 0.001) different. There was no association between weight loss and development of EIPH, even when controlling for treatment (P ≥ 0.50).

Analysis of blood samples collected 15 minutes after administration of furosemide or placebo confirmed the presence of furosemide in all horses after administration of furosemide and in none of the horses after administration of the placebo.

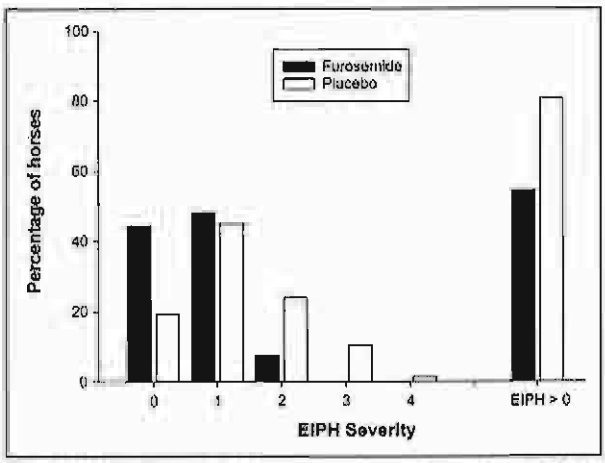

Figure 1—Distribution of scores for endoscopic severity of EIPH in Thoroughbred horses that raced following administration of furosemide (500 mg, IV; n = 161) or a placebo (saline solution; 156).

Table 3—Cross-classification of scores for endoscopic severity of EIPH following racing in 152 Thoroughbred racehorses competing twice under similar conditions each time, except that furosemide (500 mg, IV) was administered prior to one race and a placebo (saline solution) was administered prior to the other.

EIPH score when administered furosemide	EIPH score when administered placebo					
	0	1	2	3	4	Total
0	21	32	10	2	0	65
1	10	32	21	11	1	75
2	1	3	4	3	1	12
3	0	0	0	0	0	0
4	0	0	0	0	0	0
Total	32	67	35	16	2	152

Potential EIPH scores ranged from 0 to 4. Distribution of scores differed significantly (Bowker symmetry test; P < 0.001) between treatments.

Table 4—Results of logistic regression analysis of EIPH severity scores for Thoroughbred racehorses enrolled in a study of the efficacy of furosemide for prevention of EIPH.

Logistic regression analysis method	OR	95% CI	P value
Development of EIPH (ie, severity score ≥ 1)			
Mixed-effects	3.4*	2.0–5.7	< 0.001
Repeated-measures fixed-effects	3.3*	2.1–5.2	< 0.001
Conditional	4.41	2.2–8.8	< 0.001
Development of moderate to severe EIPH (ie, severity score ≥ 2)			
Mixed-effects	7.1*	3.6–14.1	< 0.001
Repeated-measures fixed-effects	6.9*	3.7–13.0	< 0.001
Conditional	11.0†	4.0–30.3	< 0.001

*Odds ratio was adjusted for age. †Odds ratio was not adjusted for age, because this variable did not differ between paired observations.
CI = Confidence interval.
Odds ratios represent the odds that horses would develop EIPH following administration of a placebo (saline solution), compared with the odds that they would develop EIPH following prerace administration of furosemide (500 mg, IV).

Discussion

Results of the present study indicated that prerace administration of furosemide decreased the incidence and severity of EIPH in Thoroughbreds racing under

typical conditions in South Africa. Specifically, horses were substantially more likely to develop EIPH (severity score ≥ 1; OR, 3.3 to 4.4) or moderate to severe EIPH (severity score ≥ 2; OR, 6.9 to 11.0) following administration of saline solution than following administration of furosemide, and the estimated proportion (unadjusted for repeated measures or confounding) of horses that developed EIPH (ie, severity score ≥ 1) following administration of furosemide (89/161 [55.3%]) was significantly lower than the estimated proportion that did following administration of saline solution (125/156 [80.1%]). In addition, 81 of the 120 (67.5%) horses that had EIPH after administration of saline solution had a reduction in EIPH severity score of at least 1 when treated with furosemide.

Important strengths of the present study include the large number of horses examined, the evaluation of horses after standard race conditions, and the use of horses from a population expected to be at risk for developing EIPH (ie, Thoroughbred racehorses in active training and racing). Because various methods have been recommended for analysis of data from crossover studies, we elected to use mixed-effects, repeated-measures fixed-effects, and conditional logistic regression to analyze our data, and results of all 3 analyses were consistent. The strong association between furosemide administration and protection against development of EIPH made it unlikely that unidentified confounding factors or other biases were solely responsible for this effect. The use of a crossover study design enhanced the statistical power of the study over that associated with a parallel-group study design.[11]

Examination of drug effects under actual conditions of use has long been recognized as the best measure of efficacy in human medicine, with randomized, controlled, clinical trials considered to provide the highest degree of evidence for efficacy.[12] However, such trials can be difficult to perform in veterinary medicine, and we are not aware of any previous such studies that have addressed the effects of various preventive measures on the development of EIPH in racehorses.

Results of the present study provide strong evidence that furosemide can help prevent the development of EIPH in Thoroughbred racehorses. As such, its use in racehorses might be justifiable, assuming that other regulatory and policy issues important to the integrity of the sport are adequately addressed.

The mechanism by which furosemide prevents EIPH is unclear, and the present study was not designed to address this issue. It has been speculated that furosemide-induced reductions in body weight are indicative of reductions in body water and intravascular fluid volume and that these reductions in body water and intravascular fluid volume attenuate the exercise-induced increase in pulmonary arterial blood pressure typically associated with exercise, with a consequent reduction in the incidence of alveolar capillary rupture and decreased hemorrhage.[13–15] The amount of weight lost by horses in the present study after furosemide administration was consistent with the amount of weight loss in horses administered furosemide under experimental conditions.[16–19] However, weight loss does not appear to be directly related to the mechanism by which furosemide prevents EIPH, in that we did not identify an association between amount of weight lost and prevention of EIPH in the present study. We have previously shown that EIPH adversely affects the performance of racehorses and that treatment with furosemide improves race performance,[5,6] and results of the present study would seem to suggest that the improved performance associated with furosemide could potentially be attributed to prevention or mitigation of EIPH.

For the present study, we believed that evaluating a large number of horses under actual racing conditions was important because previous studies[13,19] have used experimental models (eg, horses running on a treadmill) that might not reflect racing conditions, had low statistical power because of low numbers of horses, or had limitations in study design or statistical analysis that may have affected their results. Two previous studies[1,20] have examined the effect of furosemide in racehorses under field conditions, although with differing conclusions regarding efficacy. However, neither study was conducted as a randomized, controlled trial, and the data analysis in one of these studies[21] has been criticized.

An important concern with crossover studies is that the time between arms of the study (ie, the washout period) must be sufficiently long to preclude any residual effects associated with the previous treatment. In the present study, we elected to use a washout period of 7 days on the basis of the reported short elimination half-life of furosemide in horses (β half-life, 24 minutes; γ half-life, 177 minutes) and the brief (1-hour) diuretic effect of the drug.[22] The fact that we did not detect furosemide in any of the blood samples collected 15 minutes after administration of saline solution suggested that the washout period was adequate. In addition, there was no evidence that treatment order had an effect on the results of our statistical analyses. Finally, even if there had been a carryover effect in horses that had been treated with furosemide first, this would have acted to make it more difficult to identify a difference between the 2 treatments.

Furosemide reduces mucociliary clearance in humans and causes bronchodilation in ponies with recurrent airway obstruction.[23,24] It is possible, therefore, that furosemide did not actually decrease alveolar bleeding in the present study but simply decreased the rostral progression of blood from the alveoli, diminishing the amount of blood in the trachea at the time of endoscopic examination and resulting in an artifactually low EIPH severity score. Alternatively, bronchodilation secondary to furosemide administration might have favored rostral movement of blood and made the endoscopic score appear worse than it would have been had furosemide not been administered. We believe that the magnitude of either of these potentially conflicting effects is likely to be small in horses without recurrent airway obstruction and bronchoconstriction and would have been unlikely to have materially affected the overall conclusions of the present study.

The present study was performed in South Africa for logistic reasons. However, South Africa has a well-regulated racing industry with horses comparable to those racing in other parts of the world. We believe, therefore, that our results can be generalized to other

racing jurisdictions, particularly given the relative genetic homogeneity of Thoroughbred racehorses,[25] the similarity in training techniques and racing conditions throughout the world,[26] and the characteristics of horses included in our study. Although racing and training conditions in other parts of the world do differ from those in South Africa in minor respects, we do not have any evidence that any of these differences have been demonstrated to have an impact on the frequency or severity of EIPH. Therefore, we believe that results of the present study are relevant to horses racing worldwide.

a. Salix, Intervet SA (Pty) Ltd, Isando, South Africa.
b. Kryovite B Co Super, Kyron Laboratories (Pty) Ltd, Benrose, South Africa.
c. PASS 2007, Number Cruncher Statistical Systems, Kayesville, Utah.
d. SAS, version 9.2, SAS Institute Inc, Cary, NC.

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ACVIM Consensus Statement

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Exercise Induced Pulmonary Hemorrhage in Horses: American College of Veterinary Internal Medicine Consensus Statement

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Background: Published studies of exercise-induced pulmonary hemorrhage (EIPH), when assessed individually, often provide equivocal or conflicting results. Systematic reviews aggregate evidence from individual studies to provide a global assessment of the quality of evidence and to inform recommendations.

Objectives: Evaluate evidence to determine: if EIPH adversely affects the health, welfare or both of horses; if EIPH affects the athletic capacity of horses; the efficacy of prophylactic interventions for EIPH; and if furosemide affects the athletic capacity of horses.

Animals: None.

Materials and Methods: *Systematic review.* A panel of 7 experts was formed to assess evidence in the peer reviewed literature addressing each of the 4 objectives. Methodology followed that of the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE). Publications were assessed for quality of evidence by working groups of the panel, and a summary of findings was presented in tables. Recommendations were based on quality of evidence and were determined by a vote of the panel.

Results: Much of the evidence was of low to very low quality. Experimental studies frequently lacked adequate statistical power. There was moderate to high quality evidence that EIPH is progressive, is associated with lung lesions, that it adversely affects racing performance, that severe EIPH (Grade 4) is associated with a shorter career duration, that furosemide is efficacious in decreasing the incidence and severity of EIPH, and that administration of furosemide is associated with superior race performance.

Conclusions and clinical significance: Strong recommendation that EIPH be considered a disease and a weak recommendation for use of furosemide in management of racehorses with EIPH.

Key words: Bleeding; Lungs; Physiology; Respiratory.

Exercise-induced pulmonary hemorrhage (EIPH) is bleeding that occurs from the lungs of horses during exercise. It occurs in the majority of Thoroughbred and Standardbred racehorses and in many other horses subjected to strenuous exercise.

The perceived importance of EIPH and use of furosemide is illustrated by the results of an internet search[a] (May 26, 2014) that returned over 45,000 results on the term "bleeders in horses", 113,000 on "EIPH", 890,000 using "Lasix and horse", and 905,000 results for "furosemide and horse". Web of Science searches conducted on May 26, 2014 using the terms "exercise-induced pulmonary hemorrhage" or "exercise-induced pulmonary hemorrhage" AND "horse" yielded 368 results, "EIPH" AND "horse" 224 results, and "furosemide or frusemi-

Abbreviations:

ACVIM	American College of Veterinary Internal Medicine
EIPH	exercise induced pulmonary hemorrhage
EP	evidence profile
GRADE	Grading of Recommendations, Assessment, Development, and Evaluation
SoF	summary of findings

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de" AND "horse" 367 results. Further evidence of interest in these topics is the frequency of articles and opinion pieces in the nonscientific veterinary and lay literature.

For the purpose of this consensus statement, EIPH is defined as the presence of blood detected on tracheobronchoscopic examination after exercise, presence of red blood cells in bronchoalveolar lavage fluid, or both. There is no consensus about the concentration of red blood cells in bronchoalveolar lavage fluid that is diagnostic of EIPH, and the definition varies among reports. Interventions to prevent or decrease the severity of EIPH will be referred to as prophylaxis and not as "treatment" given that most interventions are applied before the horse exercises and that some clinicians consider treatment of EIPH to be the management of the consequences of EIPH after exercise.

This consensus statement addresses 4 topics related to EIPH and its prophylaxis in horses. It does not address regulatory issues such as detection of drug administration or the potential masking effect of furosemide on other medications or substances, the pathophysiology of EIPH, risk factors for the condition, or the effect of permitting use of furosemide on the health or racing career of horses.

The objective of this consensus statement is to review the evidence and provide findings and recommendations that address each of the following topics:

1. Does EIPH adversely affect the health, welfare or both of horses? This question is important because evidence of an adverse effect of EIPH on the health or well-being of horses has potential ramifications for use of horses for racing.
2. Does EIPH affect the athletic capacity of horses? An adverse effect of EIPH on athletic capacity might influence decisions on the use of interventions to decrease the severity or incidence of EIPH.
3. Are there effective prophylactic interventions for EIPH? The capacity to manage EIPH is dependent on the existence of medications or interventions that decrease the severity or incidence of EIPH.
4. Does furosemide affect the athletic capacity of horses? An association of furosemide administration with superior performance has been suspected since the drug was first used in race horses and continues to be contentious.[1]

We performed a systematic review providing a series of findings and recommendations rather than a narrative review because of the relatively large number of experimental and observational trials relevant to these topics and the importance of systematically ranking the quality of the evidence. Although both types of review have the capacity to provide a critical evaluation of the literature, only the systematic review is widely recognized as rigorous and, being based on clearly defined methodology, is less likely to be biased.

Increasingly, the shortcomings of providing only assessments of the quality of evidence as the outcome of a systematic review have been recognized, leading to the development of GRADE methodology (Grading of Recommendations Assessment, Development and Evaluation)] which provides a methodology for arriving at findings regarding the body of evidence and making recommendations based on these findings.[2,3] In addition to considering the strength of evidence, the GRADE process considers a number of other factors when making a recommendation (see Supplementary material).[4]

Because randomized controlled trials only rarely have been used in investigations related to EIPH, we expanded our consideration to studies of other designs. We also adopted the GRADE approach to evaluating the quality of evidence of individual studies and then developed a concise statement of our overall confidence in the results of all studies combined. Assessments of evidence included an assessment of the quality of the evidence and the direction of the effect.

Methods

The topic for this consensus statement was developed using policies and procedures of the American College of Veterinary Internal Medicine. The topic was selected after nomination from the ACVIM membership, and confirmed by the ACVIM Board of Regents. Nominations for membership of the consensus panel were solicited from leadership of the ACVIM and ECEIM, and composition and chair of the consensus panel were approved by the Board of Regents of the ACVIM. All members of the panel completed a conflict of interest declaration, which was provided to a representative of the Board of Regents of the ACVIM and the Chair of the panel. Potential conflicts of interest for each panel member are listed separately.

The consensus panel invited input to the process in an email to all members of the Large Animal Specialty and ECEIM on January 7, 2014. Three responses were received.

This consensus statement was developed by a systematic review of the scientific literature related to the 4 topics listed above. Consistent with the GRADE approach,[5] a series of subsidiary outcomes were defined for each of the 4 main topics and were defined as either "critical" or "important".[6] Critical outcomes were those clearly directly related to the topic (e.g. race performance as a critical subsidiary outcome for the topic of "association of EIPH with performance") whereas important outcomes were those related to mechanisms (e.g. blood gas tensions during strenuous exercise as an indirect estimate of the relationship of furosemide with performance, pulmonary fibrosis as an indicator of lung health) or indirect measures of a critical outcome (e.g. run time to fatigue on a treadmill, VO_{2max} as an indicator of athletic capacity). The scientific literature relevant to each of the subsidiary questions was then evaluated for relevance and strength of evidence and each study summarized in an "Evidence Profile" (EP) table.[5] Studies then were aggregated into a "Summary of Findings" (SoF) table that summarized the available literature.[7] Further details are available in the Supplementary material.

Exercise-induced pulmonary hemorrhage was defined as the presence of blood in the airways of horses after exercise. Blood could be detected by tracheobronchoscopic examination, or by enumeration of red blood cells or hemosiderophages in tracheal aspirates or bronchoalveolar lavage fluid. Exercise-induced pulmonary hemorrhage included both occult hemorrhage (evident only on tracheobronchoscopic or cytologic examination of the airways)

and epistaxis. Throughout this document EIPH refers to either outcome (occult EIPH or epistaxis). Epistaxis refers specifically to the presence of blood at the nostrils after racing.

Responsibility for developing the initial search and evaluation of the literature was delegated to a working group for each topic. Each working group then provided Evidence Profile tables, Summary of Findings tables and a written summary for evaluation by the whole panel. Discussion among working group members occurred by email and teleconference. See Supplementary material for details.

Results

Consensus was achieved on all findings by a unanimous vote.

Topic 1. What is the Impact of EIPH on Welfare and Health of Horses?

Exercise-induced pulmonary hemorrhage often is cited as an important factor adversely affecting the health and well-being of athletic horses without provision of evidence supporting the contention. Evidence of systematic examination of affected horses for clinical abnormalities such as fever, cough, or abnormal lung sounds is sparse (Table 1).

Critical Outcome. *Does EIPH produce clinical signs?*: The clinical signs of EIPH often are considered to include: blood in the airways detected by either tracheobronchoscopy or examination of tracheal aspirates or bronchoalveolar lavage fluid, poor performance, epistaxis, abnormalities detected on ultrasonographic or radiographic examination of the thorax, coughing, increased respiratory rate, respiratory distress or changes in behavior. The diagnostic accuracy of these signs varies or has not been well-evaluated. Presence of blood in the airways of a horse after exercise is considered the gold standard for diagnosis of EIPH. Tracheobronchoscopic detection and grading of blood in the trachea or bronchi has been validated as a means of assessing the severity of EIPH (but not the severity of the underlying lesions) and has clinical utility in that it is associated with measures of performance.[8,9] Athletic performance is likely a useful guide to the horse's health.

There is very low quality evidence that EIPH is not associated with coughing and coughing does not appear to be a reliable sign of the presence of EIPH detected by presence of hemosiderophages in tracheal lavage fluid.[10] We located no reports of the frequency of coughing in horses with EIPH diagnosed by tracheobronchoscopy.

Epistaxis after exercise generally is considered an indication of EIPH although epistaxis can result from other causes (e.g. trauma to the head or upper airways, ethmoidal hematoma, guttural pouch mycosis). In the 3 reports of examination of horses with EIPH as evidenced by epistaxis, no evidence of causes other than pulmonary hemorrhage as the source of the blood was identified. There is moderate quality evidence that epistaxis during or soon after exercise is attributable to EIPH.

Radiographic examination of the thorax of horses can demonstrate the presence of densities in the caudodorsal lung fields of some horses with EIPH. Many horses with EIPH have minimal to undetectable radiographic abnormalities and horses without a history of EIPH can have marked abnormalities. There is moderate quality evidence that radiographic examination has low sensitivity in detecting horses with EIPH.[11–14] There is very low quality evidence that ultrasonographic examination has high sensitivity (86%) and low specificity (26%) for detection of EIPH.[15] We identified no evidence regarding increased respiratory rate, respiratory distress, or changes in behavior as clinical signs of EIPH in horses after exercise.

Finding: *There is very low quality evidence of consistent clinical abnormalities in horses with EIPH, with the exception of presence of epistaxis after exercise for which there is moderate quality evidence.*

Important Outcome. *Does EIPH affect blood-gas exchange?*: Arterial blood gas tensions and blood (or plasma) lactate concentrations theoretically could be affected by EIPH. Four observational treadmill studies provided very low quality evidence that EIPH impaired arterial blood gas tensions during intense exercise.[16–19] Studies were marked by inconsistency and imprecision and serious risk of bias.

Three prospective observational studies provide only very low quality evidence that EIPH is associated with higher blood lactate concentrations during exercise.[16,18,19] Studies were marked by low numbers of horses, bias and inconsistency.

Finding: *There is very low quality evidence of an adverse effect of EIPH on arterial oxygen tension during exercise. There is very low quality evidence of an association between higher blood lactate concentrations and EIPH during strenuous exercise.*

Critical Outcome. *Is EIPH a cause of sudden death?*: Quality of evidence regarding the occurrence of sudden death was assessed subjectively because the published data were not appropriate for an EP or SoF. There is low quality evidence of an association between EIPH and sudden death of Thoroughbred horses during racing. Exercise-induced pulmonary hemorrhage occurs in the majority of horses during racing whereas sudden death occurs in 0.08 to 0.29 horses per 1,000 starts.[20] Pulmonary hemorrhage was considered to have contributed to the sudden death during or shortly after racing or training of 50 of 143 horses for which there was confirmation of the cause of death.[21] Other reports of association of pulmonary hemorrhage and death during racing are based on small numbers of cases. Although pulmonary hemorrhage can be present in horses that die suddenly, it is unclear if pulmonary hemorrhage is the primary cause of death or is secondary to another cause of death (e.g. acute heart failure resulting in sudden death and pulmonary hemorrhage). The risk of sudden death in horses with EIPH has not been determined in that an association between EIPH and subsequent sudden death during racing is unclear.

Finding: *There is low quality evidence that EIPH is causally associated with sudden death in race horses and we could locate no evidence of increased risk of sudden death in horses with EIPH.*

Table 1. Summary of Findings table for association of EIPH with health and welfare of horses.

	Quality assessment					Number of horses		Treatment effect			
Outcomes	Study design (n)[a,b]	Bias (n)	Inconsistency	Indirectness	Imprecision	Control	EIPH	Absolute	Relative	Strength of evidence	Comments
Does EIPH cause epistaxis?											
Presence of epistaxis after exercise	III (3)	None	None	Not serious (1)	None	1005 total examinations	726 EIPH cases identified	NA	Incidence of epistaxis in EIPH positive horses 0–6.2%	Moderate	EIPH is associated with epistaxis. Frequency of other causes of epistaxis after exercise is unclear but appears to be low
Does EIPH cause changes that can be detected using imaging techniques?											
Presence of lesions on plain radiographs	III (4)	Serious (3)	Serious (1)	Serious (4)	Serious (1)	30	51	Majority report increased density in caudodorsal lung fields	NA	low	No direct comparison possible among the 4 studies. No demonstration of utility of examination
Presence of changes detectable using ultrasonography	III (1)	Serious	NA	None	None	127	30	Diagnostic sensitivity of ultrasound 55.8% specificity = 25.7%	NA	Low	Single study
Does EIPH cause coughing?											
Evidence of EIPH as a cause of coughing horses	III (1)	None	NA	Serious (indirect assessment of EIPH)	Serious – wide confidence intervals	148	100 coughing horses	OR 0.05–3.5	NA	Very low	No demonstration of coughing in horses with EIPH
Does EIPH affect blood gas exchange and blood lactate concentrations?											
Blood gas tensions during intense exercise	III (3)	Serious – no power estimate or confidence intervals around effect	Serious	None	Serious	22	126	NA	NA	Very low	No pooled estimates of effect available. Inconsistent results. Imprecise results
Blood lactate concentration during intense exercise	III (3)	Serious – no power estimate or confidence intervals around effect	Serious	None	Serious	34	38	NA	NA	Very low	No pooled estimates of effect available. Inconsistent results. Imprecise results
Does EIPH shorten the career of horses?											
Number of lifetime starts	III (1)	No	NA	No	No	744	Grade 4 horses had 15.2 fewer starts than Grade 0 horses	NA	Moderate	$P < .001$. No association between EIPH Grade and duration of career in months.	

Table 1. (Continued)

Outcomes	Quality assessment					Number of horses		Treatment effect		Strength of evidence	Comments
	Study design (n)[*,*]	Bias (n)	Inconsistency	Indirectness	Imprecision	Control	EIPH	Absolute	Relative		
Is EIPH associated with inflammation of the lung?											
Does EIPH cause inflammation in the lungs?	II (4) III (4)	Not serious	None	Serious (4)	Not serious	29	66	Evidence of low-grade inflammation only in horses instilled with autologous blood. No active inflammation in EIPH-affected animals	NA	Moderate	The 4 studies that used inoculation of autologous blood might not reflect processes occurring in EIPH
Does EIPH cause structural changes in the lung?											
Does EIPH cause structural changes in lungs.	III (7) IV (1)	Not serious	None	Not serious	Not serious	10	101	All studies hemosiderin, fibrosis and vascular remodeling in caudodorsal lung fields. 3 showed venous remodeling and 2 changes in bronchioles	NA	High	
Is EIPH a progressive condition?											
Is increasing age associated with greater risk or prevalence of EIPH?	III (4)	Serious – no power estimate or confidence intervals around effect	Serious	Serious -	Serious	569	788	Majority do not detect age effect, including after correction for number of starts.	NA	Very low	No pooled estimates of effect available. Inconsistent results. Imprecise results
Is increasing age associated with greater risk of epistaxis	III (1), IV (2)	Serious – no power estimate or confidence intervals around effect	Serious – explained by confounding of number of starts.	None	Not serious	1,253,159 race starts	NA	Increased risk of epistaxis with increasing time spent racing or age.	NA	Low	No pooled estimates of effect available. Inconsistent results. Imprecise results
Is increasing volume of racing (starts, racing years) associated with increased risk of EIPH or epistaxis	III (2)	Not serious	Not serious	None	Serious	27347	588	OR Epistaxis ~2.8 x for horses 2, 3, 4+ years versus 1 year racing OR for EIPH of 1.8 for ≥ 50 starts versus < 40 starts	1.2–5.3 1.1–2.8	Moderate	Thoroughbreds. Evidence of increase in OR with increasing racing volume
Does EIPH contribute to the pathogenesis of other diseases?											
No relevant publications identified											
Is epistaxis heritable?											
Calculated heritability of epistaxis	IV (2)	Moderate risk (2)	No	No	No	170,234 pedigrees analysed		Lifetime epistaxis risk h^2 = 0.23–0.27	NA	Low	Reporting of results in available studies impairs evaluation of the studies

[*] n = number of studies included.

*Study design (see Supplementary item 5) : Type I - Randomized, placebo controlled, blinded field or clinical trials (high quality RCTs) conducted under conditions of racing or competing. Initial level of evidence - High. Type II - Randomized controlled intervention trials (low quality RCTs) including treadmill studies. Initial level of evidence - Moderate. Type III - Non-randomized controlled trials and prospective observational studies. Initial level of evidence - Low. Type IV - Case series and retrospective observational studies. Initial level of evidence - Very low.

Critical Outcome. *Does EIPH shorten the career of horses?*: The association of EIPH with duration or quality of racing career can be assessed using either EIPH grading or epistaxis as a marker of EIPH severity. The 1 study addressing the association of severity of EIPH and duration of racing career used a single endoscopic examination and provided moderate quality evidence that EIPH Grade 4 is associated with a shortened racing career of Thoroughbred race horses in Australia.[22] Epistaxis is associated with retirement of horses from racing in Australia but whether this is attributable to biologic (i.e., disease) factors or is a consequence of the management of affected horses is unclear.[23] There is moderate quality evidence that EIPH of Grades 1–3 is not associated with a shortened racing career.[22]

Finding: *There is moderate quality evidence that EIPH Grade 1-3 is not associated with a shorter racing career of Thoroughbred horses. There is moderate quality evidence that Thoroughbred horses with epistaxis or Grade 4 EIPH have shorter careers.*

Important Outcome. *Is EIPH associated with inflammation in the lung?*: Early descriptions of airway inflammation (bronchiolitis) in EIPH lungs[24] are not supported by more recent investigations.[35] Experimentally, a single infusion of autologous blood into the airways is followed by increased numbers of alveolar macrophages and hemosiderophages and disappearance of blood with no residual inflammation at 14 days.[25,26] Blood instilled repeatedly also is cleared rapidly and does not result in lesions characteristic of EIPH.[27,28]

The evidence supporting airway inflammation as a cause of EIPH is very weak. During intense exercise, horses are more likely to bleed into regions of lung with local experimentally induced airway inflammation but the role this inflammation plays in the naturally occurring syndrome is unknown.[29] In a large investigation of Thoroughbred racehorses examined monthly, airway inflammation was associated with EIPH as defined both by visible bleeding and hemosiderophages in tracheal wash fluid but the relationship of these observations to recent exercise or racing was not considered.[30] Other large field investigations found no associations between EIPH score and airway inflammation,[31] between cough (a sign of airway inflammation) and number of hemosiderophages,[10] or between tracheal mucus score (a sign of lower airway inflammation) and EIPH score.[31]

Finding: *There is low quality evidence that EIPH leads to inflammation in either the pulmonary parenchyma or airways. There is very low quality evidence that inflammation causes EIPH.*

Critical Outcome. *Does EIPH cause lesions in the lungs?*: Worldwide, lesions are present in the lungs of EIPH-affected horses retired from racing because of repeated exercise-associated epistaxis or EIPH.[33,35,65,97] Similar but less severe lesions described in young horses in training need confirmation.[32] Both gross and microscopic EIPH lesions are bilateral and most prevalent in the caudodorsal region of the lung. Lesions extend to varying degrees along the dorsal border, but never occur in the cranioventral regions. Gross lesions include discoloration of the pleural surface with underlying firm parenchyma that does not fully deflate in excised lungs. Pleural discoloration is a consequence of hemosiderin accumulation that is accompanied by pleural and septal fibrosis and angiogenesis.[24,35] Vascular lesions include extensive remodeling of small pulmonary veins (100–200 μm outer diameter) characterized mainly by accumulation of adventitial collagen and, in some vessels, smooth muscle hyperplasia.[35] In the most severely affected vessels, the vascular lumen is markedly decreased. The distribution of venous remodeling, hemosiderin, and fibrosis is similar to the distribution of pulmonary blood flow in the equine lung.[33] Electron microscopy of lungs from recently exercised horses shows breaks in the capillary endothelium and basement membrane, interstitial and intra-alveolar accumulations of erythrocytes, and interstitial edema that are compatible with capillary stress failure consequent to high intravascular pressure.[34]

Finding: *There is high quality evidence that some horses with EIPH have extensive and characteristic pulmonary lesions.*

Critical Outcome. *Is EIPH a progressive condition?*: There are no studies that report on the incidence of EIPH in a group of horses followed over the course of their career. There is low quality evidence that EIPH detected by endoscopic examination is associated with age when confounding factors, including the number of starts, are not accounted for in the statistical analysis.[9,36,37,46] However, when the number of starts is included, age is not a risk factor for EIPH.[38]

Similarly, there is moderate quality evidence that age is a risk factor for epistaxis when confounding factors are not taken into account.[39–41] When career duration was included in analyses, years spent racing was a significant risk factor (although with considerable imprecision), whereas age was not.

Finding: *There is moderate quality evidence that EIPH is progressive and related to load of racing.*

Critical Outcome. *Does EIPH contribute to the pathogenesis of other diseases?*: We could identify no reports of studies investigating the relationship between EIPH and subsequent infectious or noninfectious lung disease.

Finding: *We did not locate evidence that EIPH is associated with development of other lung diseases.*

Critical Outcome. *Is EIPH heritable?*: Because EIPH of some form occurs in almost all racehorses, there is no phenotypic variance at the level of present/not present, rendering the question of heritability of EIPH likely irrelevant. There is low quality evidence that epistaxis is a heritable trait in racing Thoroughbreds.[42,43] The quality of the evidence is considered to be very low because of difficulties with case identification, inability to exclude non-EIPH related epistaxis, inability to completely characterize pedigrees, and because the heritability measured might be for factors that facilitate the passage of blood from lungs to nostrils rather for those than influence the severity of EIPH.

Finding: *There is no published evidence regarding the heritability of EIPH. There is very low quality evidence of an association of pedigree with occurrence of epistaxis.*

Topic 2. Does EIPH Affect Performance?

The high incidence of EIPH has prompted speculation that EIPH is an important cause of impaired performance in Thoroughbred racehorses. Although this belief is strongly held by many horsemen and veterinarians involved in the care of racehorses, others have suggested that EIPH might be associated with superior performance, being reflective of greater racing effort. Evaluating the association of EIPH with performance requires establishing outcomes or measurements of performance during racing or on the treadmill (Table 2).

Critical Outcome. *Is EIPH associated with the finishing position in a race?*: Seven studies reported on the association of EIPH with finishing position in the race (1 with moderate level of evidence and 6 with low and very low level of evidence). Two studies determined that EIPH detected by tracheobronchoscopic examination was associated with the likelihood of having inferior finishing position races.[44,45] One study examined 744 Thoroughbreds racing in Australia where race-day use of furosemide and nasal strips are prohibited.[45] The other examined 1,003 individual Thoroughbred racehorses (2,118 tracheobronchoscopic examinations) that all received furosemide and had been diagnosed previously with EIPH.[46] The study with the strongest evidence showed that horses that were EIPH negative or had EIPH grade 1 were more likely to win or finish in the first 3 positions.[45] In the 5 studies showing no effect of EIPH on finishing position, pre-race furosemide prophylaxis status for horses was unknown in 4 and unreported in the fifth.[9,37,47–49]

Finding: *There is moderate quality evidence that moderate to severe EIPH in Thoroughbred race horses is associated with increased likelihood of inferior finishing position in a race.*

Critical Outcome. *Is EIPH associated with the finishing time in a race?*: A single study examined 29 EIPH positive Standardbred horses that had at least 1 EIPH negative race.[48] Their average racing times were compared between EIPH positive and EIPH negative horses and no statistical significant difference was detected. The report was of very low quality because of apparent low statistical power, nonrandom selection of horses, and racing time was recorded only in winners.

Finding: *There is very low quality of evidence that EIPH in Standardbred racehorses is not associated with finishing time in a race.*

Critical Outcome. *Is EIPH associated with the distance a horse finishes behind the winning horse in a race?*: A single study evaluated the effects of EIPH on the distance a horse finishes behind the winning horse in a race.[45] Horses with EIPH severity Grade >1 finished significantly further behind the winner than did horses with no evidence of EIPH. For horses with EIPH distance finished behind the winner was associated with grade of EIPH with higher grades finishing further behind the winner. Post hoc testing indicated significant difference in distance finished behind the winner with horses with grade 2 EIPH compared with no evidence of EIPH.

Finding: *There is moderate quality evidence that Thoroughbred racehorses with more severe EIPH finish farther behind the winning horse in a race.*

Critical Outcome. *Is EIPH associated with race earnings?*: A single study evaluated the effects of EIPH on a horse's race earnings. Horses with EIPH severity grade ≤ 1 were about 3 times as likely to be in the highest decile for race earnings when compared to horses with EIPH severity Grade ≥2.[45]

Finding: *There is moderate evidence that severity of EIPH in Thoroughbred racehorses is negatively associated with a horse's race earnings.*

Critical Outcome. *Is there a dose response relationship between the severity of EIPH and performance?*: Three studies of horses racing on a racetrack reported evaluation of the effect of the severity of EIPH on performance.[45–47] The 2 studies providing moderate quality evidence indicated a negative association of the severity of EIPH and performance.[46,47] The strongest study found an apparent dose-response for distance finished behind the winning horse, but not for finishing position as measured categorically (i.e. winning or finishing in the top 3 positions).[45]

Finding: *There is low quality evidence of a dose-response relationship between severity of EIPH in Thoroughbred racehorses and severity of impaired performance.*

Topic 3. Are There Effective Prophylactic Interventions for EIPH?

All investigations of the effect of drugs and nonpharmacological management of EIPH have focused on prevention (i.e. prophylaxis). There are no reports of the efficacy of treatments to decrease severity or progression of lung lesions of EIPH-affected horses nor are there reports of treatment of horses with EIPH (i.e. management of the short term clinical consequences of an episode of EIPH). Likewise, there are no reports of efficacy of interventions applied during training to prevent EIPH during racing.

Critical Outcome. *Is furosemide effective prophylaxis for EIPH?*: A number of low quality investigations conducted both on the treadmill and on the racetrack judged furosemide ineffective as a treatment for EIPH (Table 3).[50–52] These studies simply judged the presence or absence of visible hemorrhage postexercise by endoscopy with no attempt to judge the severity of bleeding. Low quality studies demonstrated a decrease in the number of red blood cells in bronchoalveolar lavage fluid in horses performing standardized exercise tests on a treadmill.[53,54]There was a decrease in severity of EIPH identified in 2 high quality investigations that endoscopically graded bleeding in large numbers of horses running on the racetrack.[55,56]

Finding: *There is high quality evidence that furosemide (0.5–1 mg/kg administered IV 4 hours before strenuous exercise) decreases the severity and incidence of EIPH.*

Important Outcome. *Does furosemide affect pulmonary vascular pressure?*: Pertinent to EIPH, several moderate

Table 2. Summary of findings – Does EIPH affect performance?

	Quality Assessment						Summary of findings				
Outcomes	Study Design	Bias (n)	Limitations	Inconsistency	Indirectness	Imprecision	Summary of results	Results: YES;NO	Dose response (graded EIPH)	Strength of Evidence: High, moderate, low, very low	Comments
Finishing position – Normal racing conditions	III (7)	Low risk (1) Moderate to High risk (6)	Very different approaches to data analysis. Most did not control for potential confounding	Inconsistencies in reported outcomes (performance) and methods of analysis	Nil	Minimal to moderate for pairwise comparisons in the high quality study. Serious for lower quality studies	High quality study for EIPH ≤1: OR for winning = 4.0 (1.5–14.3). OR for finishing in top 3 = 1.8 (1.1–3.1). No difference found in lower quality studies	2 studies found effects (1 high quality, 1 low quality)	Not detected	Mod (n = 1) Very low (n = 6)	Low power and confounding bias could have profound affected low-quality study
Finishing time – Normal racing conditions	III (1)	High risk	Low power, no control of potential confounding	N/A	Nil	Serious	n = 49 horses: EIPH Mean = 2:04.1 (SD = 2.1 second). Non-EIPH Mean = 2:03.3 (SD = 2.1 seconds)	n.s.	Not evaluated	Very low	Very low power
Distance finished behind winning – normal racing conditions	III (1)	Low risk	Low numbers of severely affected horses	N/A	Nil	Moderate for pairwise comparisons related to severe EIPH	n = 744 horses: EIPH ≥ 1 mean = 4.4 m (SE 1.2 m); EIPH Grade 0 mean = 2.6 m (SE 1.1 m)	Significant difference	Yes	Mod	Low numbers of severely affected horses
Race earnings (90th percentile or greater)	III (1)	Low risk	Low numbers of severely affected horses	N/A	Nil	Moderate for pairwise comparisons related to severe EIPH	n = 744 horses: OR for EIPH ≤1 = 3.0 (1.3–8.0) compared to EIPH ≥ 2	Significant difference	Not reported	Mod	Low numbers of severely affected horses
Dose Response Relationship between EIPH and Performance – Normal Racing Conditions	III (3)	Low risk (1) Moderate to High risk (2)	Very different approaches to data analysis. Most did not control for potential confounding. Low numbers of severely affected horses	Inconsistencies in reported outcomes (performance) and methods of analysis	Nil	Minimal to moderate for pairwise comparisons in the high quality study. Serious for lower quality studies.	Dose response relationship was identified in the high quality study for distance finished behind the winner, and was not identified for finishing position. Lower quality studies did not identify an effect.	1 study found an effect in 1 outcome.		Mod (n = 1) Very low (n = 2)	Confounding bias could have profoundly affected low-quality studies

Table 3. Summary of findings of efficacy of furosemide and other interventions for prophylaxis of EIPH.

	Quality Assessment					Number of horses		Treatment effect			
Outcomes	Study design (n)	Bias (n)	Inconsistency	Indirectness	Imprecision	Control	FUR	Absolute	Relative	Strength of evidence	Comments
Is furosemide is an effective prophylactic treatment for EIPH?											
EIPH quantified by scoring tracheal blood postexercise	I (2)	Low	No	No	No	211	211	65% of horses have decreased EIPH score when racing after furosemide	Horses are more likely to bleed after saline than after furosemide treatment	High	"High" evidence rating results from large n, randomized crossover trials, racing conditions, and scoring of tracheal blood
EIPH quantified by BALF RBC count after exercise	Type II (3)	Moderate	No	No	No	23	23	Furosemide reduced BAL RBC count.		Low	The correlation between BALF RBC count and EIPH score is unknown.
EIPH quantified by blood in trachea post exercise (Yes/No)	II (4) IV (1)	High	No	No	No	19 61	19 24	No effect of furosemide	NA	Very Low	None of these studies graded the severity of bleeding
Does furosemide affect pulmonary capillary blood pressure?											
Direct measurement of pulmonary vascular pressures	II (4)	Low	No	No	No	30	30	Furosemide (1 mg/kg, IV) reduced Pcap to 79% of control		Moderate	In treadmill studies, furosemide consistently reduces pulmonary capillary pressure in exercising horses

Table 3. (Continued)

Outcomes	Study design (n)	Quality Assessment				Number of horses		Treatment effect		Strength of evidence	Comments
		Bias (n)	Inconsistency	Indirectness	Imprecision	Control	FUR	Absolute	Relative		
Are nasal strips an effective prophylaxis for EIPH?											
EIPH quantified by blood in trachea post exercise (Yes/No)	II (1)	High	NA	No	Serious – Amount of blood not quantified	7	7	No effect of nasal strips		Low	Single study
EIPH quantified by BALF RBC count after exercise	II (3) III (1)	Low	No	No	No	24	43	Nasal strips reduced BAL RBC count.		Low	Small sample size and low power

quality treadmill investigations have consistently demonstrated that furosemide decreases pulmonary arterial and pulmonary wedge (left atrial) pressures and hence (calculated) pulmonary capillary and transmural pressure during intense exercise.[50,51,57–64] Such decreases in pressure might decrease the likelihood of capillary stress failure.[34]

Finding: *There is moderate quality evidence that furosemide reduces pulmonary vascular pressure during strenuous exercise.*

Critical Outcome. *Is aminocaproic acid an effective prophylaxis for EIPH?*: Two randomized, placebo-controlled treadmill studies found that aminocaproic acid (2–7 g, IV) given 2–4 hours before strenuous exercise test to fatigue did not decrease BALF red blood cells compared to saline placebo.[66,67] However, both studies provided very low quality evidence because of outcome measure imprecision and indirectness (risk of bias), and small sample size (6–8 horses).

Finding: *There is very low quality evidence that aminocaproic acid affects EIPH severity.*

Critical Outcome. *Are bronchodilators effective prophylaxis for EIPH?*: Clenbuterol administered IV alone or in combination with furosemide (10 minutes before exercise) does not affect pulmonary hemodynamics.[50,68] but drug effect on EIPH severity was not assessed. Nine days of clenbuterol treatment in resting horses after intrabronchial instillation of autologous blood did not result in significant change in numbers of red blood cells or hemosiderophages in BALF compared to control.[69] Another study with few horses showed no effect of atropine on EIPH and inconclusive results with ipratropium nebulization.[70] All studies provided very low to low quality evidence because of the low number of horses and lack of blinding.

Finding: *There is very low quality evidence that bronchodilators affect EIPH.*

Critical Outcome. *Are corticosteroids effective prophylaxis for EIPH?*: One treadmill study reported that 3 days of dexamethasone did not prevent EIPH but EIPH severity was not assessed.[71] Neither 9–10 days of inhaled beclomethasone nor oral prednisolone treatment changed either red blood cell number or hemosiderophages in BALF of resting horses after intrabronchial instillation of autologous blood.[69]

Finding: *There is very low quality evidence that corticosteroids affect EIPH severity.*

Critical Outcome. *Are nonsteroidal anti-inflammatory drugs effective prophylaxis for EIPH?*: Very low quality treadmill studies failed to detect an effect of either phenylbutazone (with furosemide) or flunixin meglumine on EIPH (evaluated as presence or absence of blood on endoscopic examination).[61,72]

Finding: *There is very low quality evidence that nonsteroidal anti-inflammatory drug treatment affect EIPH.*

Critical Outcome. *Is pentoxifylline an effective prophylaxis for EIPH?*: Two treadmill studies found that pentoxifylline had no effect on pulmonary hemodynamics when used alone or in combination with furosemide. An effect of pentoxifylline on EIPH (evaluated as pres-

ence or absence of blood on endoscopic examination) was not detected although EIPH severity was not assessed.[58,73]

Finding: *There is very low quality evidence that pentoxifylline affects EIPH.*

Critical Outcome. *Are there other medications that are effective for prophylaxis of EIPH?*: Carbazochrome (with furosemide),[74] equine serum concentrate,[75] conjugated estrogens,[67] endothelin 1-A antagonist[76], nedocromil,[77] nitric oxide,[78] and sildenafil[79] have been investigated as prophylaxis of EIPH in single studies for each drug. The studies are all of very low quality because they were conducted on a treadmill, used low numbers of horses, and the severity of EIPH was not assessed.

Although reportedly used in practice, we could locate no scientific evidence of the efficacy of aspirin or ethamsylate.

Finding: *The studies provided very low quality evidence that these drugs affect EIPH severity.*

Critical Outcome. *Do nasal strips prevent EIPH?*: A low quality treadmill investigation assessing presence or absence of postexercise blood in the airways stated nasal strips were ineffective in preventing EIPH, however, the severity of bleeding was not graded.[80] 4 other studies, undertaken in a limited number of horses, showed that horses had a significant decrease in postexercise BALF RBCs when exercised with nasal strips.[53,54,81,82]

Finding: *There is low quality evidence that nasal strips decrease severity of EIPH.*

Important Outcome. *Are there other miscellaneous nonpharmacological treatments to prevent EIPH?*: Neither herbal formulations[83] nor inhaled water vapor[84] showed evidence of efficacy in preventing EIPH. The studies were of very low quality.

Rest and water restriction before strenuous exercise have been recommended, however there is no scientific evidence that those practices decrease the incidence or severity of EIPH. Nonetheless, several racing jurisdictions have ruled to enforce rest periods ranging from 2 to 3 months for horses with epistaxis.

Finding: *The studies provided very low quality evidence that herbal preparations or inhaled water vapor affect EIPH severity.*

Topic 4. Does Furosemide Affect Performance?

A variety of outcome measures have been used in an attempt to assess performance in horses racing on a track (Table 4). However, standardization of measurements is difficult because numerous intrinsic factors (e.g., sex, age, horse quality, fitness level) and extrinsic factors (e.g., jockey, distance, track conditions, environmental conditions) vary among races and can create confounding bias in results. Control of extrinsic and intrinsic factors is more feasible when horses model race experiences by running on high-speed treadmill, but this model inherently limits the generalizability of results. Additionally, most treadmill studies suffer from small sample size and consequently have low statistical power. The ability to extrapolate performance data obtained during treadmill studies to actual performance during racing has not been established.

Most studies conducted on racetracks have used adjusted race time to cover a standardized distance as an outcome measure of performance[85–90] whereas others have used finish position,[85,89] racing speed or earnings.[85] Treadmill studies have evaluated performance as distance covered or time that horses run until the onset of fatigue.[74,91–93] Alternatively, or in addition, some treadmill studies have reported the effect of furosemide on the energetic cost of locomotion.[91,94,95]

Critical Outcome. *Does furosemide affect performance of horses running on a racetrack?*: Studies in Thoroughbred and Standardbred racehorses have been performed under natural racing conditions[85,88–90] and under simulated racing conditions on a track.[86,87] The study with the highest sample size (n = 22,589) found that mean estimated mile-equivalent race times were 0.56 to 1.09 second faster for horses receiving furosemide prophylaxis compared to horses not receiving furosemide[85] All 4 studies conducted during normal racing conditions were rated as providing moderate quality of evidence because design and analyses helped to minimize risk of bias, used relevant outcome measures (e.g., racing time adjusted for distance), were adequately powered, and showed consistent results. Two studies performed under simulated race conditions did not detect an effect of furosemide on performance as compared to placebo.[86,87] However, both studies provided very low quality evidence because of outcome measure imprecision (risk of bias), small sample size (6–10 horses), and slow running speed achieved during race simulation.

Studies that investigated other performance measures, such as finish position, average racing speed, and race earnings also identifed a consistent benefit for horses receiving furosemide before racing compared to untreated horses.[85,89] The largest study evaluated sex differences, and found that the benefits of furosemide administration on performance were more marked in males and in horses ≤6 years old.[85] These studies were considered to have moderate quality of evidence for these outcomes. No studies investigated the mechanism for superior performance.

Finding: *There is moderate quality evidence that furosemide administered IV 4 hours prior to racing is associated with improved racing outcomes in Thoroughbred and Standardbred racehorses.*

Important Outcome. *Does furosemide affect performance of horses running on a treadmill?*: Five studies examined the effect of furosemide administered to horses performing a standardized test on a high-speed treadmill. Two studies found statistically longer time to fatigue in horses treated with furosemide.[92,93] Furosemide administration before a treadmill test improved the energetic cost of locomotion in 3 studies.[91,94,95]

Quality of evidence for all of these studies was rated down because relevance of performance on a treadmill compared to that on a racetrack is not known, considering that the effect of jockey or sulky and of other horses in the race cannot be replicated in the laboratory (indirectness). Also, performance on treadmill is typically

Table 4. Summary of findings concerning the effect of furosemide on performance.

	Quality Assessment					Number of horses		Treatment effect			
Outcomes	Study design (n)	Bias (n)	Inconsistency	Indirectness	Imprecision	Control	FUR	Absolute	Relative	Strength of evidence	Comments
Performance on the racetrack – Normal racing conditions	III (4)	Low risk (4)	No	No	No	6,001	17,260	−0.12 to −1.09 s*	−0.68 to −0.88%	High	Time to cover a given distance in furosemide treated horses relative to controls.
Performance on the racetrack – Simulated racing conditions	I (1) II (1)	Moderate risk (2)	No	Serious	Very serious	16	16	n.s.		Very low	One study recorded racing time with a stopwatch and in the other, horses raced maximally only during last ¼ mile. Small sample size and very low power
Finish position in race	III (2)	Low risk (1) Moderate risk (1)	No	No	No	5,854	16,804	OR win = 1.4 −1.5	N/A −26%	High	Odds of winning or Improvement in finishing position with furosemide
Treadmill performance	I (5)	Low risk (1) Moderate risk (4)	No	No	No	33	33	13.9 s	N/A	Moderate	Performance measured as extra time run before fatigue with furosemide

n.s., not statistically significant. *$P < 0.05$

judged based on the onset of fatigue which is a subjective assessment that can be influenced by lack of treatment concealment (imprecision).

Finding: *There is low quality evidence that furosemide administered IV 4 hours before treadmill exercise results in delayed onset of fatigue and improved energetic cost of locomotion.*

Discussion and Recommendations

The consensus panel found that there is good quality evidence that the presence of pulmonary lesions in racehorses is associated with epistaxis or repeated diagnosis of EIPH and low quality evidence of no effect of EIPH, excluding epistaxis, on well-being or health of horses. The presence of lesions in lungs of horses with EIPH substantiates our strong recommendation that EIPH be considered a disease and not a variably manifested normal result of strenuous activity in horses. There is only low quality evidence that the disease is progressive but the evidence allows the panel to make a weak recommendation that EIPH be considered a progressive disease, recognizing that further research is needed.

The panel found that there is high quality evidence that furosemide is effective in the prophylaxis of EIPH and makes a weak recommendation for its use in management of racehorses with this disease. The recommendation is weak because the panel recognizes that conditions for use of furosemide in some horses, such as racehorses, is regulated by racing jurisdictions that must consider a broad range of factors (not just efficacy) and that there continues to be extensive discussion among these stakeholders regarding policies and perceived need for furosemide prophylaxis.[96]

The panel makes no recommendation regarding other pharmacological interventions for the prophylaxis of EIPH because of the absence of studies or the very low to low quality of evidence.

The panel notes that many studies intended to test the efficacy of an intervention for prophylaxis of EIPH do not include adequate reporting of the details of the study to permit full evaluation of the quality of evidence, were likely to have a high frequency of Type 2 error rates because of small sample sizes, were conducted on a treadmill (with unknown relevance to actual competition), and did not assess dose–response relationships. Of particular concern to the panel was the large number of reports that had negative results (i.e., the study did not detect an effect of the intervention) but did not make an *a priori* attempt to establish adequate study size or to consider statistical power in interpretation of their results. Failure to detect an effect of the intervention in a study with inadequate statistical power is not the same as demonstration of no effect.

The panel found that there is moderate quality evidence that moderate to severe EIPH is associated with decreased athletic capacity by Thoroughbred racehorses.

The panel found that there is high quality evidence that furosemide administration is associated with improved performance by Thoroughbred and Standardbred racehorses.

Footnote

[b] www.google.com

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Supporting Information

Additional Supporting Information can be found online in Supporting Information:

ATTACHMENT #9

9/ LASIX OPTIMIZES RACING PERFORMANCE:

When a horse bleeds significantly into its lungs it cannot fully oxygenate its blood and its racing performance suffers. Horses showing significant blood in the trachea post-race perform more poorly than horses with minimal or no blood in the trachea. The so called performance "improvement" associated with Lasix is therefore actually more likely a performance PROTECTION against an adverse effect of EIPH, again consistent with the long standing field experience of American Horsemen, Attachment #9.

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Effect of furosemide on performance of Thoroughbreds racing in the United States and Canada

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Objective—To determine the effect of furosemide on performance of Thoroughbreds racing on dirt surfaces at tracks in the United States and Canada.

Design—Cross-sectional study.

Animals—All Thoroughbreds (n = 22,589) that finished a race on dirt surfaces at tracks in the United States and Canada between June 28 and July 13, 1997 in jurisdictions that allowed the use of furosemide.

Procedure—Race records were analyzed by use of multivariable ANOVA procedures and logistic regression analyses to determine the effect of furosemide on estimated 6-furlong race time, estimated racing speed, race earnings, and finish position. Principal component analysis was used to create orthogonal scores from multiple collinear variables for inclusion in the models.

Results—Furosemide was administered to 16,761 (74.2%) horses. Horses that received furosemide raced faster, earned more money, and were more likely to win or finish in the top 3 positions than horses that did not. The magnitude of the effect of furosemide on estimated 6-furlong race time varied with sex, with the greatest effect in males. When comparing horses of the same sex, horses receiving furosemide had an estimated 6-furlong race time that ranged from 0.56 ± 0.04 seconds (least-squares mean ± SE) to 1.09 ± 0.07 seconds less than that for horses not receiving furosemide, a difference equivalent to 3 to 5.5 lengths.

Conclusions and Clinical Relevance—Because of the pervasive use of furosemide and its apparent association with superior performance in Thoroughbred racehorses, further consideration of the use of furosemide and investigation of its effects in horses is warranted. (*J Am Vet Med Assoc* 1999;215:670-675)

Furosemide is a potent diuretic that is ostensibly used in racehorses to prevent exercise-induced pulmonary hemorrhage (EIPH), a condition with a high prevalence in Thoroughbred racehorses.[1-5] Although the effectiveness of furosemide in preventing EIPH has been questioned, furosemide may decrease the severity of bleeding.[7,8,9] Furosemide is commonly administered to racehorses in almost all Thoroughbred racing jurisdictions in the United States and Canada; nonetheless, precise estimates of the frequency of its use are unavailable. To our knowledge, the effect of furosemide on athletic performance of racehorses has not been established, despite the fact that there is experimental evidence that supports a performance-enhancing effect.[10-11,b] Field studies suggest that horses receiving furosemide may race faster than horses that do not.[2,12-14] However, these results have been equivocal, perhaps as a result of small numbers of animals examined and, subsequently, the low statistical power. The purpose of the study reported here was to examine the association between furosemide use and the performance of Thoroughbred horses racing on dirt surfaces at tracks in the United States and Canada.

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Materials and Methods

Database—A commercial database[a] was used to obtain race records for all Thoroughbred horses that raced on dirt surfaces at tracks in the United States and Canada between June 28 and July 13, 1997 in jurisdictions that allowed the use of furosemide. Only the first race for each horse during that time period was included. Horses that did not finish the race were not included in the analyses.

Data obtained included the horse's name, whether the horse received furosemide or phenylbutazone prior to racing (yes vs no), age (y), sex (female, male, or gelding), weight carried (lbs), number of days since last race, race distance (furlongs), racetrack, date the race was held, track variant score, track condition (fast vs not fast), number of horses that started the race, finishing place, lengths finished behind the winner, winner's finishing time (s), purse for the race (dollars), money earned in that race (dollars), lifetime earnings (dollars), number of times the horse had previously received furosemide before a race, number of races in which the horse started during its lifetime, and numbers of lifetime first, second, and third place finishes.

Finishing time was estimated for horses that did not win by use of the following formula: estimated finishing time = winner's time + (0.2 s X lengths finished behind the winner).[3,15,c] Mean racing speed of each horse was estimated by dividing race distance by estimated finishing time. A standardized estimate of the time required to complete a 6 furlong race (1 furlong = 1/8 mile) was then calculated by use of the following equation: estimated 6-furlong race time = 6 furlongs + estimated mean racing speed. Actual distance of races was controlled for in all statistical models.

Track variant score[a] is an indicator of track conditions and quality of competition, with a lower score indicating faster track conditions or better quality of competition on a given day. The track variant score was calculated by use of the following formula: track variant score = 100 − mean speed rating for each type of race on that day. Speed rating[a] is an indicator

of performance for a race and is calculated by use of the following formula: speed rating = 100 points - 1 point for every 0.2 seconds that the winner's finishing time was greater than the track record for that distance during the previous 3 years.

Statistical analyses—Descriptive statistics were calculated, data were summarized, and distributions of continuous variables were evaluated for normality. Furosemide administration (yes vs no) was the primary independent variable of interest and was included in all multivariable analyses. In addition, all available variables thought to have possibly affected or predicted the quality of the horses' performance were controlled in analyses.

Potential associations between furosemide administration and estimated 6-furlong race time, mean racing speed, and race earnings were examined by use of multivariable ANOVA.[8] In addition, the potential interaction between effect of age and effect of furosemide administration was examined in a separate analysis of estimated 6-furlong race time, using age as a categorical variable (2, 3–4, 5–6, 7–8, and 9–14 years). The potential for phenylbutazone administration (yes vs no) to alter the effect of furosemide administration on estimated 6-furlong race time was evaluated by analyzing these main effects and an interaction term for these variables. Only horses that raced in jurisdictions in which use of phenylbutazone was documented were included in this analysis. The potential for the effect of furosemide to vary with race distance was evaluated by use of mean racing speed as the outcome of interest. Only horses that raced at 5, 6, 7, and 8 furlong distances were included in this analysis, as these were the most common race distances.

The distribution of race earnings was highly skewed, and 24.1% of horses did not earn money in these races. Therefore, the natural logarithm of race earnings was used in the analysis. Horses that did not earn money were assigned race earnings of $1 to facilitate this transformation.

Potential associations between furosemide administration and the likelihood of horses winning the race (yes vs no), finishing in the top 3 positions (yes vs no), and earning any money in the race (yes vs no) were evaluated by use of multivariate logistic regression.[9] A logistic-binomial model was used for these analyses.

Available variables that were thought to have possibly affected or predicted the quality of the horses' performance included age, sex, racetrack, race distance, purse, weight carried, number of horses that started the race, number of days since last race, track variant score, track condition, lifetime earnings, number of races in which the horse started during its lifetime, number of lifetime wins, number of lifetime second-place finishes, and number of lifetime third-place finishes. However, as expected, preliminary analyses indicated that there was a great deal of collinearity among these variables. Therefore, principal component analysis[8] was used to create orthogonal (uncorrelated) scores for most of these highly correlated variables (age, race distance, purse, weight carried, number of horses that started the race, number of days since last race, track variant score, track condition, lifetime earnings, number of races in which the horse started during its lifetime, number of lifetime wins, number of lifetime second-place finishes, and number of lifetime third-place finishes).[8,9,11] These uncorrelated scores were then used in the models to account for the variability explained by the original variables. Principle component scores were used in their native form in analyses. All scores were retained in models regardless of loading, as it was considered practically important to control for as much extraneous variation as possible, and this large data set provided sufficient power to allow inclusion of all scores in the analyses.

Four different sets of principal component scores were calculated for use in these analyses. One set of principal component scores was used for analyses of models that used the full data set and was calculated using variables for age, race distance, purse, weight carried, number of horses starting in the race, number of days since last race, track variant score, track condition, lifetime earnings, number of races in which the horse started during its lifetime, number of lifetime wins, number of lifetime second-place finishes, and number of lifetime third-place finishes. A second set of principal component scores was used when investigating the potential interaction between furosemide administration and race distance. Race distance was not used when calculating this second set of principal component scores but instead was analyzed as a fixed effect, using mean racing speed as the outcome of interest. The third set of principal component scores was used when evaluating the potential interaction between furosemide administration and age. All variables previously used to calculate principal component scores were included in this set of scores, except that age category was analyzed as a fixed effect, using estimated 6-furlong race time as the outcome of interest. The fourth set of principal component scores was used when investigating the potential interaction between effect of furosemide administration and effect of phenylbutazone administration. All variables previously used to calculate principal component scores were included in this fourth set of scores, but scores were calculated using only that subset of horses racing in jurisdictions that permitted administration of phenylbutazone.

Furosemide administration, sex, and principal component scores were included in all models as fixed effects. The racetrack at which each horse raced was included in all models as a random effect. Phenylbutazone administration was also analyzed as a fixed effect in 1 model. Interaction terms were estimated using untransformed main effects (not principal component scores). Interaction terms of interest were retained in models with main effects when they were statistically associated with the outcome ($P < 0.05$). The Tukey-Kramer method for multiple comparisons was used to analyze differences in the least-squares means derived from ANOVA models. Odds ratios (OR) and 95% confidence intervals (95% CI) based on likelihood ratio statistics were calculated from logistic regression models.[10]

Results

Race records for 22,589 horses racing in 3,346 races at 49 racetracks were included in this investigation. Of these horses, 41.3% were females, 11.3% were males, and 47.4% were geldings. Horses ranged from 2 to 14 years old; however, most horses were 3 to 4 years old (Fig 1). Race distance ranged from 2 to 14 furlongs; the most common race distance was 6 furlongs, with 35% of the horses racing this distance.

Furosemide was administered to 16,761 (74.2%) horses. Only 1,039 (4.6%) horses received furosemide for the first time during the study period. Records indicated that 19,088 (84.5%) horses had received furosemide prior to racing at least once during their careers. Use of furosemide varied by sex of the horse (71.7% of the females, 66.0% of the males, and 78.3% of the geldings received furosemide), age of the horse, and race distance (Fig 1).

Furosemide administration, sex, principal component scores, and racetrack were significantly ($P < 0.05$) associated with outcome for all statistical models. Estimated 6-furlong race time for horses receiving furosemide was significantly ($P = 0.001$) less than that for horses not receiving furosemide. In addition, a significant ($P = 0.001$) sex-furosemide interaction indicat-

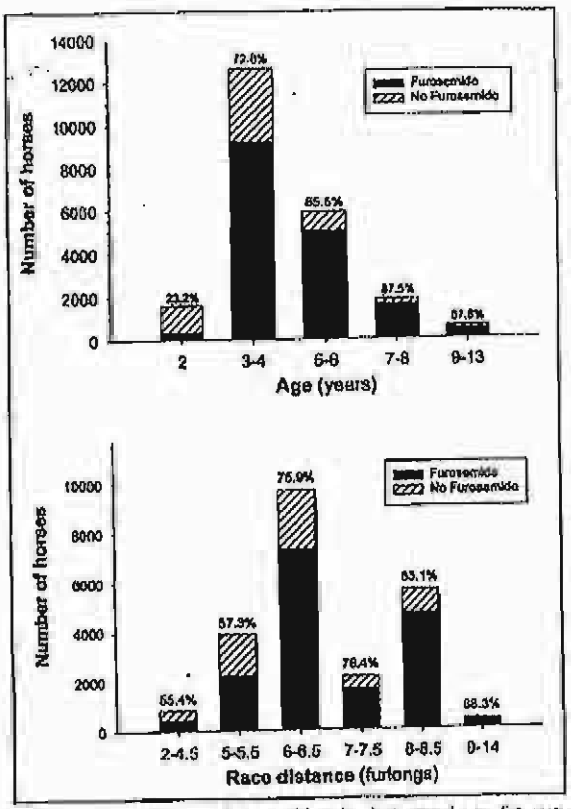

Figure 1—Numbers of Thoroughbreds that raced on dirt surfaces at tracks in the United States and Canada between June 28 and July 13, 1997, grouped according to age (top) and race distance (bottom) and on the basis of whether they did or did not receive furosemide prior to racing. Percentages above each bar represent horses that received furosemide.

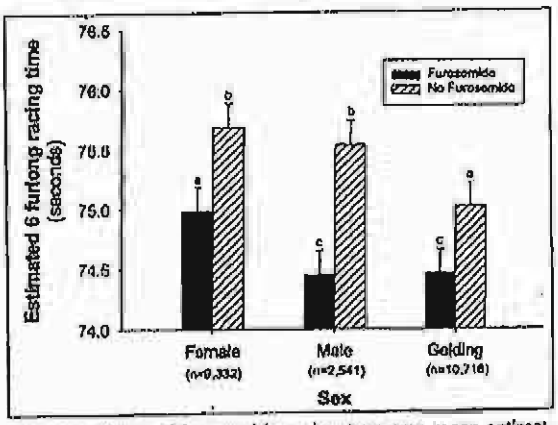

Figure 2—Effect of furosemide on least-squares mean estimated 6-furlong race time (seconds) of Thoroughbreds that raced on dirt surfaces at tracks in the United States and Canada between June 28 and July 13, 1997. Horses were grouped by sex, and groups with different letters were significantly ($P < 0.05$) different. Error bars represent SE.

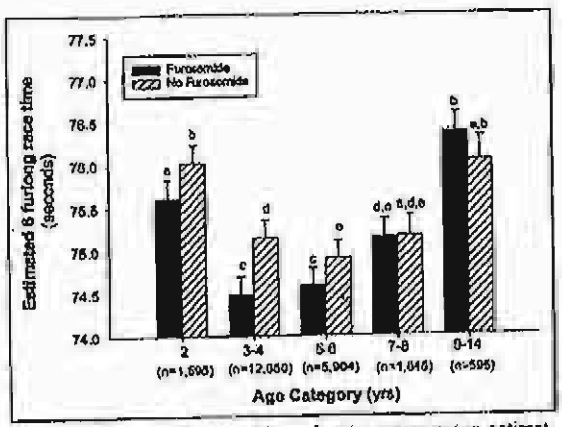

Figure 3—Effect of furosemide on least-squares mean estimated 6-furlong race time (seconds) of Thoroughbreds that raced on dirt surfaces at tracks in the United States and Canada between June 28 and July 13, 1997. Horses were grouped by age, and groups with different letters were significantly ($P < 0.05$) different. Error bars represent SE.

ed that the magnitude of this effect varied with sex of the horse, with the greatest effect in males (Fig 2). When comparing horses of the same sex, horses receiving furosemide had a mean estimated 6-furlong race time that was less than that for horses not receiving furosemide for all sexes ($P < 0.001$). This difference in mean estimated 6-furlong race time was 0.56 ± 0.04 seconds (least-squares mean ± SE) in geldings, 0.70 ± 0.04 seconds in females, and 1.09 ± 0.07 seconds in males.

Analysis of a model that included terms for the interaction of sex and age, the interaction of age and furosemide administration, and the interaction of sex and furosemide administration indicated that the effect of furosemide administration varied significantly ($P < 0.001$) with age category. Among horses < 7 years old, estimated 6-furlong race times for horses that received furosemide were significantly ($P < 0.001$) less than times of horses that did not receive furosemide when comparing horses in the same age category (Fig 3). The greatest difference was observed in horses that were 3 to 4 years old.

Among the 13,519 horses that raced 5, 6, 7, or 8 furlongs, mean racing speed for horses that received furosemide was significantly ($P = 0.03$) faster than speed for horses that did not (Fig 4). Significant ($P = 0.004$) sex-furosemide and distance-furosemide interactions were detected. When comparing horses that raced the same distance, horses receiving furosemide raced faster than did horses that did not. The magnitude of this difference in mean racing speed varied from $2.956 \times 10^{-4} \pm 0.922 \times 10^{-4}$ furlongs/s (least-squares mean ± SE) to $10.322 \times 10^{-4} \pm 0.952 \times 10^{-4}$ furlongs/s (0.06 to 0.21 m/s), depending on the distance of the race. The magnitude of this difference was greatest in horses racing shorter distances.

Horses that received furosemide were 1.4 times as likely to win the race (OR, 1.4; 95% CI, 1.27 to 1.59) and 1.2 times as likely to place in the top 3 positions (OR, 1.2; 95% CI, 1.09 to 1.37) as were horses that did not receive furosemide. Median amount of money earned by horses in the 1 race included in the study was $249 (range, 0 to $600,000), with 75.8% of the horses earning money. Horses that received furosemide were 1.3 times as likely to earn money in the race than were horses that did not receive furosemide (95% CI, 1.18 to

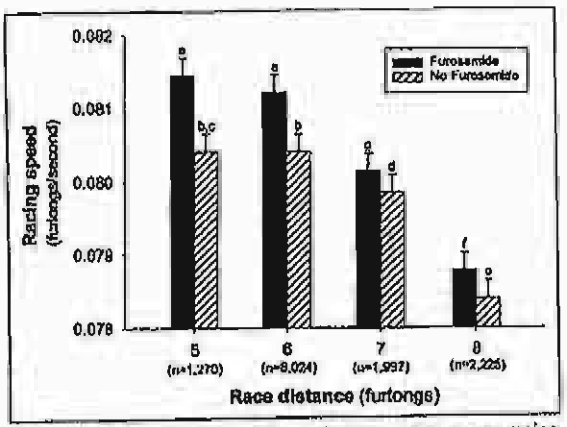

Figure 4—Effect of furosemide on least-squares mean racing speed (furlongs/second) of Thoroughbreds that raced on dirt surfaces at tracks in the United States and Canada between June 28 and July 13, 1997. Horses were grouped on the basis of distance raced, and groups with different letters were significantly ($P < 0.05$) different. Error bars represent SE.

1.41). The difference in geometric mean race earnings between horses that received furosemide and those that did not was $416 (95% CI, $337 to $513; $P < 0.001$).

There were 14,599 horses that raced at tracks at which use of phenylbutazone was permitted. Of these, 4,822 (33.0%) received furosemide alone, 1,787 (12.2%) received phenylbutazone alone, and 6,399 (43.8%) received furosemide and phenylbutazone. After controlling for furosemide administration and a sex-furosemide interaction, there was no significant difference ($P = 0.15$) in estimated 6-furlong race time between horses that did and did not receive phenylbutazone prior to racing. Mean estimated 6-furlong race time of horses that received phenylbutazone was 74.91 ± 0.26 seconds (least-squares mean ± SE); mean estimated 6-furlong race time of horses not receiving phenylbutazone was 75.05 ± 0.26 seconds.

Discussion

For all 6 outcomes assessed in this study (estimated 6-furlong race time, mean racing speed, race earnings, likelihood of winning the race, likelihood of finishing in the top 3 positions, and likelihood of earning money), horses that received furosemide exhibited superior performance, compared with horses that did not receive furosemide. They raced faster, earned more money, and were more likely to win or finish in the top 3 positions than were horses not receiving furosemide. Because of the large study population and resulting statistical power, the magnitude and consistency of the observed effect, and the fact that the study population was likely representative of the population of Thoroughbred horses racing in the United States and Canada, we believe that our results present clear and unequivocal evidence of an association between use of furosemide and superior performance in Thoroughbred racehorses.

Possible explanations for the association between use of furosemide and superior performance include reduction in severity of EIPH,[6] reduction in body weight,[10,11] induction of metabolic alkalosis,[19,20] bronchodilation,[21] and other mechanisms. We consider it unlikely that furosemide would have exerted a performance effect through an effect on EIPH, as there is no evidence that furosemide reduces the prevalence of EIPH in Thoroughbred racehorses.[1] There is also little objective evidence that it reduces the severity of EIPH[6] or that EIPH has a negative effect on the athletic ability of horses, except in the rare case of horses with severe or catastrophic bleeds.[14,22,23] Induction of metabolic alkalosis improves athletic capacity of some human athletes,[24] and furosemide has been shown to induce alkalosis that persists during incremental exercise and during brief, high-speed exercise similar to that performed during a race.[20] However, a performance-enhancing effect from furosemide-induced alkalosis has not been demonstrated in horses.[25-27]

Another explanation for a performance-enhancing effect of furosemide is the acute reduction in body weight that occurs after furosemide administration. Intravenous administration of furosemide has been shown to induce a 2 to 4% reduction in body weight within 4 hours.[10,11,28,b] Because work is a product of mass, velocity, and distance, and given the acknowledged importance of weight carriage when handicapping Thoroughbred racehorses, it would be expected that loss of this amount of weight would have a beneficial effect on athletic ability of furosemide-treated horses. This contention is supported by reports that the furosemide-induced reduction in body weight increases the maximal rate of oxygen consumption, reduces the accumulated oxygen deficit and apparent rate of lactate production, and decreases the rate of carbon dioxide production of horses during intense exertion.[10,11,b] These effects, which are prevented by carriage of weight equal to that lost as a result of furosemide administration, are considered indicative of a performance-enhancing effect of furosemide.[10,11]

The relative importance of furosemide-induced alkalosis and of weight reduction in any performance-enhancing effect of furosemide has not been determined. However, given that the degree of alkalosis induced by furosemide is mild and much less than that induced by sodium bicarbonate administration in studies that did not detect an effect of alkalinization on performance of Thoroughbred horses,[10,24,26,27] we consider it unlikely that furosemide-induced alkalosis was the principle mechanism of performance enhancement. Conversely, the magnitude of the weight loss induced by furosemide is similar to that used to handicap Thoroughbred racehorses and, as such, may represent a physiologically important change. Indeed, as stated previously, furosemide-induced weight loss is associated with a measurable effect on energy metabolism of running horses and this effect is negated by carriage of weight.[10,11,b]

Many extraneous factors may influence the performance of a racehorse, but these factors are often highly correlated. Principal component analysis is a type of multivariate analysis that uses matrix algebra to create orthogonal (uncorrelated) scores from correlated variables. It was useful in the present study, because it allowed all available information from the original variables to be included in the analyses despite the collinearity of these variables. This allowed us to control as many other sources of variation as possible. In

this manner, we could be more confident that observed differences in performance of the horses were associated with administration of furosemide. When the principle component scores were not included in the model, furosemide administration was still associated with superior performance; however, the magnitude of the effect was less. Inclusion of the principle component scores in the model allowed us to develop more refined estimates of the effect of furosemide on the performance of Thoroughbred racehorses.

Estimated race times were used in this study because actual finish times were recorded only for the winning horses. These times were estimated on the basis of a formula widely accepted in the Thoroughbred racing industry for conversion of finish position to estimated race times. The track variant score was used to rate the performance of horses on a particular day and track surface. These calculated variables were used to provide information on the level of performance of the horse and its competition. As these are only estimates, imprecision in the values of these calculated variables may have influenced the precision of estimated differences in horse performance attributable to furosemide administration. However, use of these calculated variables should not have affected the direction of the association or the conclusions reached from these analyses, as estimates would tend to be equally affected for horses that received furosemide and horses that did not.

Several other studies have attempted to determine the effect of furosemide administration on athletic performance of racehorses, but it is difficult to form strong conclusions from these studies, because of the low statistical power. Sweeney et al[2] used a complex prospective crossover study design to evaluate the potential effect that furosemide may have on athletic performance of Thoroughbred racehorses. A total of 131 horses completed the follow-up period and were included in the analysis. Race times were adjusted to 1-mile equivalent race times, using 2 handicapping methods, and ANCOVA was used to adjust race time for the actual race distance. Although the authors found that some horses treated with furosemide raced faster, compared to their initial races when they were not treated with furosemide, this effect was not consistent.

Regardless of the method used by Sweeney et al to adjust race times, mean race times for geldings without EIPH that were treated with furosemide were consistently faster, compared with their initial race times when they were not treated with furosemide.[2] However, race times did not increase when geldings were raced a third time, this time without furosemide treatment, which decreases our ability to draw firm conclusions from this study. Mean race times for females and males without EIPH were also faster when horses were treated with furosemide, but were not significantly different from race times for their initial races. Despite the strengths of this previous study, the relatively small number of horses likely hampered its ability to detect true differences. Only 18 of the horses without EIPH were geldings, the only group that had a consistently significant ($P < 0.05$) decrease in race times.

A study by Soma et al[14] of Thoroughbreds with EIPH compared finish position and race times for 5 races before horses were found to have EIPH (and, therefore, were not yet receiving furosemide) with values for the next 5 races when furosemide was administered. Although there were no significant differences in race times for horses before and after they were treated with furosemide, athletic performance was slightly better after horses received furosemide. However, few horses were enrolled in this study, and effects were inconsistent among the groups of horses examined.

In 2 studies that examined trained Standardbred racehorses, a decrease in the time required for horses to complete 8 furlongs at maximum speed was not detected in association with furosemide administration.[12,13] Results for both studies suggested that race times decreased when furosemide was administered, but differences were not significant. However, only 6 horses were included in each study. A retrospective study[13] of 58 Standardbred horses racing 8 furlongs at Louisville Downs was performed by examining race records of horses for the 1977 season. A comparison was made of race times before and after EIPH was diagnosed, but this study did not attempt to control for extraneous variables. Mean race time for races that horses ran after receiving furosemide was 0.1441 seconds slower than mean time for races that horses ran without first receiving furosemide, but this difference was not significant. Differences between results of that study and results of the present study may have been attributable to differences in breed, racing method, or statistical analyses, or may have been a result of the low numbers of horses included in the previous study.

These previous studies have likely been hampered by low statistical power attributable to small sample sizes. Low statistical power may have prevented conclusive demonstration of small but relevant effects of furosemide on performance in these studies. Even small differences in the performance of racehorses are important, because the margin between winning and losing may be < 0.2 seconds. The study reported here had greater statistical power, which allowed us to identify small effects of furosemide administration. Horses that received furosemide in this study had an estimated 6-furlong race time that was up to 1.09 seconds less than that of horses not receiving furosemide. This is approximately equivalent to a difference of 5.5 lengths (1 length = 0.2 s) at the finish of a 6-furlong race, a difference that could influence the outcome of a race.

The apparent superior performance associated with furosemide administration detected in this study varied, depending on the horse's sex. This interaction in effects is similar to that identified by Sweeney et al[2]; however, Sweeney et al found the largest effect among geldings, whereas in the present study, the largest difference in performance was observed among males. Estimated 6-furlong race times did not differ between males and geldings receiving furosemide in our study, which may reflect a maximal threshold for superior performance of these horses. The apparent effect of furosemide on the speed of racehorses varied in relation to race distance. The greatest effect was observed at shorter distances. The reason for this variation in the effect of furosemide with race distances is unclear.

The effect of furosemide on the estimated 6-fur-

long race time also varied with age of the horse; the greatest differences were observed in younger horses. The reason for this difference is unclear, but it may be that older horses have other factors, such as lameness, that play a more important role in their performance.

All racing jurisdictions require that horses have evidence of EIPH before they are allowed to receive furosemide; however, the rigor of qualification varies considerably between jurisdictions. Therefore, although all horses in this study that received furosemide met the criteria for qualification in their jurisdiction, it was not possible to separate the effect of furosemide from the EIPH status of the horses. This was because it was not known whether horses bled during the race included in this study. Another potential explanation for results of the present study is that EIPH is associated with superior performance in racehorses, and that furosemide administration is a surrogate marker for EIPH.

Experimentally, phenylbutazone has been shown to attenuate the effects of furosemide in resting and exercising horses[19] and to alter cardiovascular function in exercising horses.[30] In the present study, use of phenylbutazone did not alter the effect of furosemide administration on estimated 6-furlong race time.

In the study reported here, it was not possible to determine whether the amount of furosemide administered was associated with superior performance in racehorses. Most racing jurisdictions permitted a range in the dose of furosemide that could be administered, but the actual dose that each horse received was not recorded. Although the study design did not permit us to investigate a dose response, the consistency of the effect and the strength of the association[31] suggest that the observed difference in the performance of horses that received furosemide, compared with those that did not receive furosemide, was a real difference, and that administration of furosemide was associated with superior performance in Thoroughbred racehorses.

[a]Roberts C, Hillidge C, Marlin D. Exercise-induced pulmonary haemorrhage in racing Thoroughbreds in Great Britain (abstr), in *Proceedings*. Intern EIPH Conf 1993;18.

[b]Bayly WM, Slocombe RF, Schott HC, et al. Furosemide administration affects maximal oxygen consumption but not airway mechanics (abstr), in *Proceedings*. Intern EIPH Conf 1994;35–36.

[c]Daily Racing Form, Highs Town, NJ.

[d]MIXED Procedure, SAS statistical software, version 6.12, SAS Institute Inc, Cary, NC.

[e]GENMOD Procedure, SAS statistical software, version 6.12, SAS Institute Inc, Cary, NC.

[f]PRINCOMP Procedure, SAS 6.12, SAS Institute Inc, Cary, NC.

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ATTACHMENT #9

10/ LASIX PROTECTS THE LIVES OF HORSES AND JOCKEYS:

About once in every 1,500 races the bleeding into the lungs is sufficient to asphyxiate the running horse. The horse crashes to the ground, with all the attendant risks for horse and rider. In 1983 Gunson et al reported nine such cases in Pennsylvania racing; Gunson specifically noted that a complete necropsy was required to identify these cases, since death can occur with no overt evidence of pulmonary hemorrhage. We evaluated the first such case reported in Kentucky and wrote the case up specifically including acute sudden death among the clinical presentations of EIPH. Since then sudden death from EIPH has been identified as the principal non-musculoskeletal injury related cause of death in racing horses, Attachment #9

Sudden death attributable to exercise-induced pulmonary hemorrhage in racehorses: Nine cases (1981–1983)

Diane E. Gunson, BVSc; Corinne Raphel Sweeney, DVM; Lawrence R. Soma, VMD

Summary: Pathologic changes are described in 11 horses that died during racing or training; 9 died of acute pulmonary hemorrhage (exercise-induced pulmonary hemorrhage), 1 died of exsanguination, and 1 died of CNS trauma. Cardiac lesions were not found in any horse. Severe engorgement of pulmonary vessels, with hemorrhage into alveoli, airways, interstitium, and subpleural tissues, was observed in all 9 horses that died of exercise-induced pulmonary hemorrhage. Infiltration of eosinophils and/or lymphocytes around vessels and airways was seen in 6 horses. Focally extensive fibrosis was observed in the pleura and interstitium of 6 horses, and collections of siderophages were seen in the fibrous tissue and in the airways. Focal occlusion of bronchioles with inspissated mucus, such as that associated with small airway disease, was found in 4 horses. Underlying respiratory tract lesions, particularly those associated with small airway disease or bronchiolitis, may have a role in fatal pulmonary hemorrhage.

Sudden death in seemingly healthy horses most often is attributed to diseases of the cardiovascular system, such as myocarditis,[1-3] rupture of the heart, aorta, or large arteries,[4,5] ruptured chordae tendineae,[6] and atrial dysrhythmia.[7] Nevertheless, in a recent study of 25 horses, 21 of which died during racing or training, only 7 of the 21 (33%) had lesions sufficient to explain the sudden death.[8] The objective of the study reported here was to determine the incidence and cause of sudden death in exercising horses.

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Criteria for selection of cases

Between January 1981 and July 1983, all horses that died suddenly during racing or training at 2 Pennsylvania racetracks were transported to the necropsy facility of the George D. Widener Hospital at the University of Pennsylvania School of Veterinary Medicine. During the study period, approximately 12,000 races were run, with approximately 9 horses/race. Year-round racing was conducted at both racetracks, and approximately 1,500 Thoroughbreds were housed at each facility. Horses that were euthanatized because of breakdown or other injuries were excluded before referral to the university. Also excluded were horses that died suddenly, but not while racing or training (eg, a horse found dead in its stall one morning was not included). Complete necropsy was performed within 4 to 6 hours of death and included examination of all thoracic and abdominal organs. Tissue specimens were obtained for histologic examination from lungs, liver, kidneys, and organs that appeared abnormal. Multiple lung tissue specimens (>10) were obtained from all horses, including specimens from the cranial, lateral caudal, central dorsal caudal, and hilar regions of both lungs and from the accessory lung lobe. All tissue specimens were fixed in neutral-buffered 10% formalin, and lung specimens were submerged under absorbant towels. After fixation, tissue specimens were embedded in paraffin, and 5-μm sections were cut and were stained with hematoxylin and eosin. Selected sections were treated with Giemsa, toluidine blue, and Masson trichrome stains.

Results

During the period of the study, 11 horses died suddenly without apparent cause of death. Age ranged from 2 to 9 years, and the group included females, geldings, and stallions, representing a reasonable sample of the total population racing at the 2 tracks. Death did not have apparent seasonal incidence.

Two horses died during training; the others died while racing. All the latter horses had completed three fourths of the race. All were in apparent good health immediately before their fatal exercise.

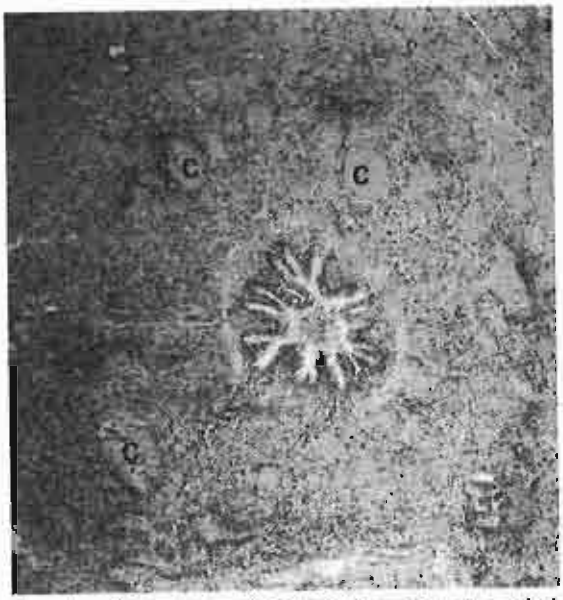

Figure 1—Photomicrograph of a bronchus of horse 3, in which severe hemorrhage separates the various layers—lining epithelium, submucosa, muscularis, and periobronchiolar tissue. Notice bronchial cartilage (c) in outer layers and minimal hemorrhage in lumen. H&E stain; × 26.

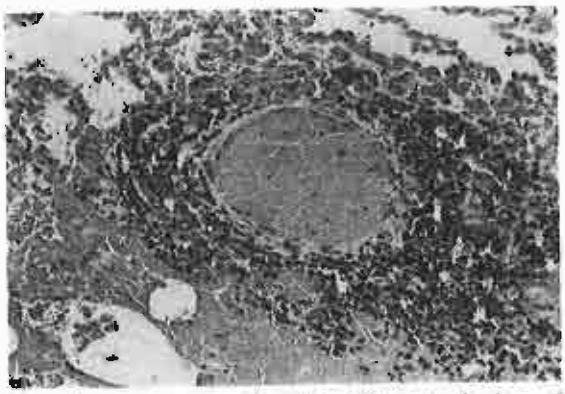

Figure 2—Photomicrograph of a small vein in the lung of horse 5, in which the various layers of the wall contain numerous eosinophils. H&E stain; × 105.

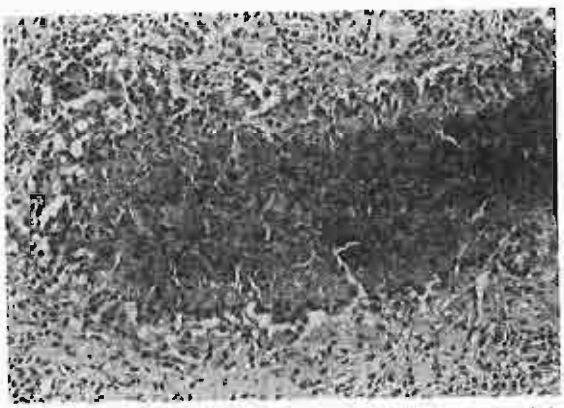

Figure 3—Photomicrograph of a focus of necrotic eosinophils surrounded by fibrous tissue, lymphocytes, macrophages, and giant cells in an area of severely congested and hemorrhagic lung. H&E stain; × 110.

Of the 11 horses, 9 died apparently from exercise-induced pulmonary hemorrhage (EIPH). The nasal cavity, trachea, and bronchi of all 9 horses, except No. 3, were filled with bloodstained froth. The lungs were deep red to black, with a few normal-appearing pink areas in the cranial portion. The lungs were heavy, and felt moderately firm, consistent with being blood-filled. In horses 3 and 5, multiple deep red nodules (1 to 3 cm in diameter) were scattered throughout the lung parenchyma. Focal fibrosis of the pleura, evident as thick white areas over the lung surface, was seen in horses 1 through 5 and horse 9. Some of these areas felt firm and extended into the lung parenchyma. Pathologic changes that could have accounted for the sudden death were not observed in any other system, and macroscopic cardiac lesions were not observed in any horse.

Microscopic examination of the lungs of all 9 horses revealed engorgement of the pulmonary arteries, veins, and capillaries, with hemorrhage into alveoli, bronchioles, bronchi, interstitium, and subpleural tissue. The severity of the engorgement and hemorrhage varied from almost nonexistent to massive in various areas of the lung, but the caudal portion of the lung lobes was the site of the most severe hemorrhage. Pleural and interstitial fibrosis or marked eosinophilic bronchitis and bronchiolitis were accompanied by severe hemorrhage. Some airways with severe eosinophilic and lymphocytic infiltration had extensive hemorrhage, and the lining epithelium, submucosa, muscular layer, cartilage, and peribronchial tissue were separated widely by areas of hemorrhage (Fig 1). Infiltration of eosinophils around and within walls of airways and blood vessels was observed in horses 3, 5, 7, and 9 (Fig 2), and lymphocytes were seen in similar locations in horses 1, 3 through 5, 7, and 9. The severity of the infiltration varied from location to location within the lung—almost nonexistent in some and severe in others. The most severe changes were in the dorsal caudal and hilar areas, but in some horses, multiple, severely affected areas seemed to be randomly distributed. Multiple oblong, oval, or circular foci of degenerating eosinophils resembling parasite migration tracks were observed in the pulmonary parenchyma of horses 3 and 5 (Fig 3). Alternatively, these might have been bronchioles, with attenuated or sloughed bronchial epithelial lining, plugged by bronchial casts of necrotic eosinophils. These eosinophil-containing foci were most numerous within the aforementioned dark red nodules that were visible in the lungs of these 2 horses.

Focally extensive fibrosis of the pleura and interstitium, often including the area around large vessels and airways, were observed in 6 of the horses. Groups of macrophages laden with hemosiderin also were seen within this fibrous tissue, particularly at the junction of the pulmonary parenchyma and the deep layers of the pleura. In horses 1 through 4, 7, and 9, groups of siderophages also were seen in

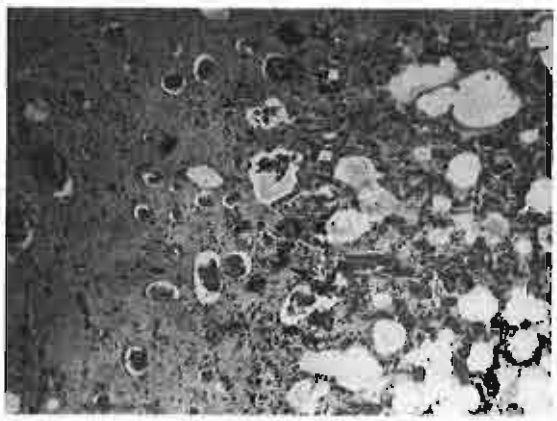

Figure 4—Photomicrograph of a focus of pleural fibrosis, in which are seen congested capillaries and groups of siderophages (arrows). H&E stain; × 40.

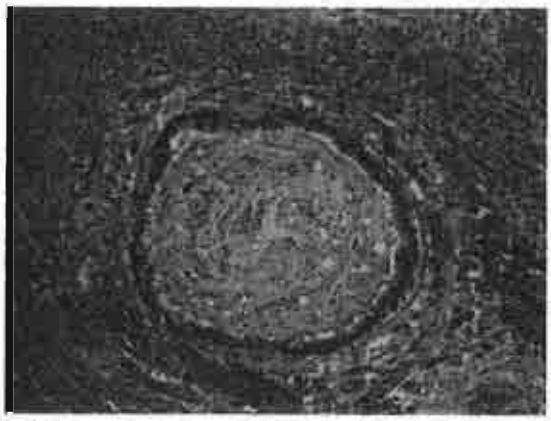

Figure 5—Photomicrograph of mucoid bronchiolitis. The lumen is plugged with mucus and degenerating inflammatory cells. Peribronchiolar connective tissue is infiltrated with lymphocytes and eosinophils, and capillaries are engorged. H&E stain; × 46.

airways (Fig 4), peribronchial connective tissue, alveolar walls, and alveolar lumens. Siderophages infrequently were observed in the alveoli of all horses. Severe focal mucoid bronchiolitis was seen in horses 1 and 3 through 5. In affected areas, the bronchioles and several bronchi were occluded completely with plugs of inspissated mucus and cellular debris (Fig 5). Degenerating inflammatory cells were scattered throughout the lining epithelium, and lymphocytes, eosinophils, and neutrophils infiltrated the bronchiolar walls and peribronchial connective tissue.

In these 9 horses, sudden death was attributed to pulmonary failure secondary to fulminant EIPH. Of the 9, 4 had had epistaxis at the time of death and 5 had previous history of EIPH; the EIPH history of the other 2 horses was unknown.

Horses 10 and 11 were included in the study because they had died during exercise without apparent traumatic cause of death. However, at necropsy, horse 10 had hemoperitoneum attributable to severance of the right internal iliac artery secondary to fracture of the right pubic bone. Other tissue specimens did not have pathologic changes.

Horse 11 had multiple fractures of the left forelimb and third and fourth cervical vertebrae, with macroscopic evidence of spinal cord compression. Other tissues did not have pathologic changes, with the exception of mild pulmonary hemorrhage and eosinophilic peribronchitis in multiple lung specimens.

Sudden death in these 11 exercising horses was attributed to EIPH in 9 horses, exsanguination in 1 horse, and CNS trauma in 1 horse.

Discussion

All horses of this study had lesions sufficient to explain their sudden death during exercise. This differed appreciably from the report of Gelberg et al,[8] who found a 32% probability of such lesions. The majority (82%) of the horses of our study had pulmonary lesions sufficient to explain the sudden death. In the study of Gelberg et al,[8] the authors speculated that sudden death most likely was associated with exercise-induced acute cardiovascular failure; this conclusion was reached despite the fact that only 2 horses had histologic evidence of myocardial disease. Of course, a microscopic lesion causing conduction disturbance, with subsequent heart failure, may have been overlooked.

Epistaxis during or after exercise long has been recognized as a serious problem in racehorses.[9] Its origin in the lungs was suggested,[10] but was not confirmed until fiberoptic endoscopy was used widely.[11,12] Because of its association with exercise, the condition has become known as EIPH. Morbidity of EIPH, its relation to age, and its association with various situations, has been described,[11-13] as has the effect of treatment with various drugs.[12,14-17] Occurrence of EIPH is not random, and it is observed consistently in individual horses.[12,17]

Despite its occurrence in 45 to 86% of racing horses, data are not available on the death rate of horses with EIPH. Our study indicated that EIPH was the cause of death in 82% of the Thoroughbred racehorses that died suddenly (while exercising) of causes unrelated to musculoskeletal trauma. Thus, EIPH should be considered as the most common cause of sudden death in exercising Thoroughbred racehorses.

During the period of the study reported here, approximately 12,000 races were run; therefore, we found the death rate of horses with EIPH to be 1 death/1,500 races.

There is only limited understanding of the pathophysiologic mechanism of EIPH. It is associated with atrial fibrillation,[18] but in human beings, atrial fibrillation is associated with increased left atrial and pulmonary wedge pressures.[19] Paroxysmal atrial fibrillation has been reported in racehorses,[20-23] with most horses reverting to normal sinus rhythm within 24 hours. If transient arrythmia develops in a horse during a race, it might not be detected. Thus, many apparently healthy horses may be exercising with

such arrythmias, and may be at risk for development of EIPH. Exercise-induced pulmonary hemorrhage may be triggered by the asphyxia that develops during breath-holding after horses leave the starting gate.[24] Another hypothesis is that upper airway obstruction, as seen in horses with laryngeal hemiplegia, may initiate pulmonary bleeding during maximal exercise.[25]

Alternative explanations for EIPH have included subclinical respiratory disease resulting in bronchospasm precipitated by exercise.[10,26,27] In our horses, subclinical chronic bronchitis may have been involved, especially in old horses.[26] Bronchiole obstruction and/or scarring might contribute to local increases in perivascular pressure,[26,27] and perhaps might result in pulmonary hemorrhage. Of the horses of this study, 6 had fibrous pulmonary scars, and 4 had eosinophilic bronchiolitis.

In a recent study,[28] extensive small airway disease was observed in close association with the vascular changes in lungs of horses with EIPH. Bronchiolitis was suspected as the early lesion, and increased number and size of bronchial arteries may have resulted.[28] We also suspect that the eosinophils in eosinophilic bronchiolitis associated with allergic lung disease[29] may have a role in alveolar hemorrhage. In the guinea pig, eosinophils contain a metalloprotein that degrades type-I and type-III collagen, which constitute the bulk of lung and blood vessel collagen.[30] Thus, eosinophils could damage the integrity of lung blood vessels and alveolar walls so that increased pulmonary perfusion and intermittent higher airway pressure associated with exercise might precipitate pulmonary hemorrhage. Cytologic findings in tracheobronchial aspirates from many Thoroughbred racehorses were suggestive of small airway disease,[a] and evidence of past pulmonary hemorrhage was detected in many of the horses.

In conclusion, only 4 of the 9 horses that died from pulmonary hemorrhage had epistaxis at the time of death, although all but 1 had blood-stained froth in the airways at necropsy. Thus, pulmonary hemorrhage cannot be ruled out as the cause of death solely because of lack of blood in the nares. Necropsy should include thorough examination of the lungs for the signs of EIPH—deep red to black, heavy, firm lungs, and bloodstained froth in the airways. Detailed examination of the heart and larynx for predisposing lesions also should be performed.

Although the study was designed to include only horses with nontraumatic causes of death, 2 horses that died from trauma were included because of the lack of external signs of such trauma. Because our study indicated that EIPH was the apparent cause of death in the majority (82%) of exercising Thoroughbred racehorses, complete necropsy was necessary to rule out a traumatic cause of death.

[a]Sweeney CR, Roby KA, Soma LR, et al. Cytologic findings from tracheobronchial aspirates of 94 racehorses (abstr). In *Proceedings*. Am Coll Vet Int Med 3rd Annu Sci Prog 1985;125.

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Book Review: Decision Making in Small Animal Soft Tissue Surgery

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As with most textbooks that contain contributions from several authors, the content varies from section to section. The editors are to be complimented for minimizing variations in quality. Of particularly good quality are the discussions of acute and chronic diarrhea and neoplastic disorders. This may be attributable in part to the fact that these topics lend themselves well to the format used in the text. Topics that require judgment on the part of the veterinarian before using the algorithms are not well suited to this. This problem is exemplified by the discussion of management of traumatic wounds. The decision-making process is clearly described, but unless the veterinarian has accurately assessed the wound before following the algorithm, it is difficult to arrive at the intended conclusion. A similar comment could be made regarding the discussion of biliary tract trauma.

Because nearly all topics were handled in the space of 2 pages, including the algorithms, decisions had to be made regarding what material was to be included. In the discussion of megaesophagus, as well as the discussion of vascular ring anomalies, a Heller's myotomy of the lower esophageal sphincter is recommended for treatment of spasm or stricture of the lower esophageal sphincter. The need for this procedure in veterinary medicine is quite limited, and it is used infrequently. The overall poor prognosis associated with surgical treatment of persistent right aortic arch is not emphasized in this section. In the discussion of airway obstruction, it is unclear as to why castellated laryngofissure was the only technique illustrated for treatment for laryngeal paralysis.

Another problem with the algorithm format is illustrated by the section on heartworm disease: In the algorithm accompanying this discussion, the recommendation is made that the presence of liver and kidney disease in conjunction with evidence of right-sided heart disease should be considered an indication that adult worms be removed via pulmonary arteriotomy. It is doubtful that this statement can be universally applied to all animals in this category. The statement also is made that uncomplicated bile peritonitis is aseptic and does not require abdominal drainage. Although it is probably true that uncomplicated bile peritonitis does not require drainage of the abdominal cavity, it is misleading to imply that this is an aseptic condition. In the algorithm accompanying the discussion of linear intestinal foreign bodies, the reader is informed that surgery is invariably required. Recent reports suggest that, in certain instances, these may be managed expectantly.

Potential purchasers of this text should be aware of the intention of the editors and the limitations of the text. Within this context, the text succeeds in its mission and should be a valuable asset to veterinary practitioners and veterinary students seeking to improve their management of small animals requiring surgery.—*[Decision Making in Small Animal Soft Tissue Surgery. By Allen G. Binnington and Joanne R. Cockshutt. 232 pages; illustrated. B. C. Decker Inc, Burlington, Canada. 1988. Available in USA from CV Mosby Co, 11830 Westline Industrial Dr, St Louis, MO 63146. Price $40.00.]*—DALE E. BJORLING

ATTACHMENT #10

10/ LASIX PROTECTS THE LIVES OF HORSES AND JOCKEYS:

About once in every 1,500 races the bleeding into the lungs is sufficient to acutely asphyxiate the running horse. The horse crashes to the ground, with all attendant risks for horse and rider. In 1983 Gunson et al reported nine such cases in Pennsylvania racing; Gunson specifically noted that a complete necropsy was required to identify these cases, since death can occur with no overt evidence of pulmonary hemorrhage. We evaluated the first such case reported in Kentucky and wrote the case up specifically including acute sudden death among the clinical presentations of EIPH. Since then sudden death from EIPH has been identified as the principal non-musculoskeletal injury related cause of death in racing horses, Attachment #10

Sudden death attributable to exercise-induced pulmonary hemorrhage in racehorses: Nine cases (1981–1983)

Diane E. Gunson, BVSc; Corinne Raphel Sweeney, DVM; Lawrence R. Soma, VMD

Summary: Pathologic changes are described in 11 horses that died during racing or training; 9 died of acute pulmonary hemorrhage (exercise-induced pulmonary hemorrhage), 1 died of exsanguination, and 1 died of CNS trauma. Cardiac lesions were not found in any horse. Severe engorgement of pulmonary vessels, with hemorrhage into alveoli, airways, interstitium, and subpleural tissues, was observed in all 9 horses that died of exercise-induced pulmonary hemorrhage. Infiltration of eosinophils and/or lymphocytes around vessels and airways was seen in 6 horses. Focally extensive fibrosis was observed in the pleura and interstitium of 6 horses, and collections of siderophages were seen in the fibrous tissue and in the airways. Focal occlusion of bronchioles with inspissated mucus, such as that associated with small airway disease, was found in 4 horses. Underlying respiratory tract lesions, particularly those associated with small airway disease or bronchiolitis, may have a role in fatal pulmonary hemorrhage.

Sudden death in seemingly healthy horses most often is attributed to diseases of the cardiovascular system, such as myocarditis,[1-3] rupture of the heart, aorta, or large arteries,[4,5] ruptured chordae tendineae,[6] and atrial dysrhythmia.[7] Nevertheless, in a recent study of 25 horses, 21 of which died during racing or training, only 7 of the 21 (33%) had lesions sufficient to explain the sudden death.[8] The objective of the study reported here was to determine the incidence and cause of sudden death in exercising horses.

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The authors acknowledge the cooperation of Drs. F. Oppenlander and R. Solow of Keystone and Penn National Racetracks, respectively, and thank Drs. H. Acland, J. Griffiths, E. Sartin, L. Andersen, D. Shaw, C. Lichtensteiger, and C. Woods for performing some of the necropsies.

This work was sponsored by the Pennsylvania State Horse Racing Commission.

Address reprint requests to Dr. Soma.

Criteria for selection of cases

Between January 1981 and July 1983, all horses that died suddenly during racing or training at 2 Pennsylvania racetracks were transported to the necropsy facility of the George D. Widener Hospital at the University of Pennsylvania School of Veterinary Medicine. During the study period, approximately 12,000 races were run, with approximately 9 horses/race. Year-round racing was conducted at both racetracks, and approximately 1,500 Thoroughbreds were housed at each facility. Horses that were euthanatized because of breakdown or other injuries were excluded before referral to the university. Also excluded were horses that died suddenly, but not while racing or training (eg, a horse found dead in its stall one morning was not included). Complete necropsy was performed within 4 to 6 hours of death and included examination of all thoracic and abdominal organs. Tissue specimens were obtained for histologic examination from lungs, liver, kidneys, and organs that appeared abnormal. Multiple lung tissue specimens (>10) were obtained from all horses, including specimens from the cranial, lateral caudal, central dorsal caudal, and hilar regions of both lungs and from the accessory lung lobe. All tissue specimens were fixed in neutral-buffered 10% formalin, and lung specimens were submerged under absorbant towels. After fixation, tissue specimens were embedded in paraffin, and 5-μm sections were cut and were stained with hematoxylin and eosin. Selected sections were treated with Giemsa, toluidine blue, and Masson trichrome stains.

Results

During the period of the study, 11 horses died suddenly without apparent cause of death. Age ranged from 2 to 9 years, and the group included females, geldings, and stallions, representing a reasonable sample of the total population racing at the 2 tracks. Death did not have apparent seasonal incidence.

Two horses died during training; the others died while racing. All the latter horses had completed three fourths of the race. All were in apparent good health immediately before their fatal exercise.

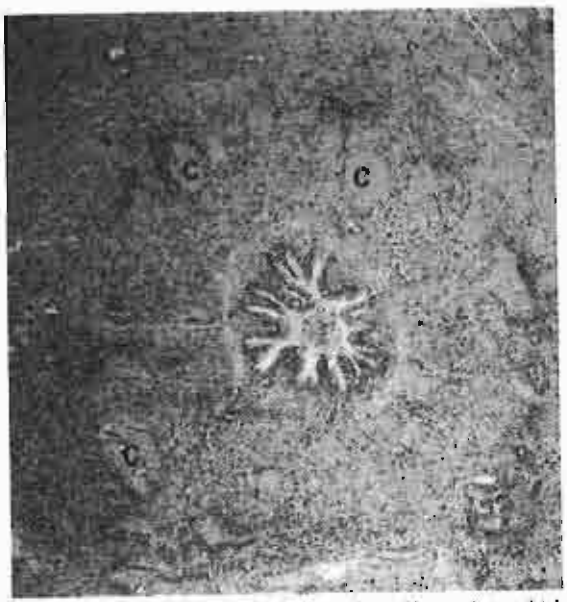

Figure 1—Photomicrograph of a bronchus of horse 3, in which severe hemorrhage separates the various layers—lining epithelium, submucosa, muscularis, and peribronchiolar tissue. Notice bronchial cartilage (c) in outer layers and minimal hemorrhage in lumen. H&E stain; × 26.

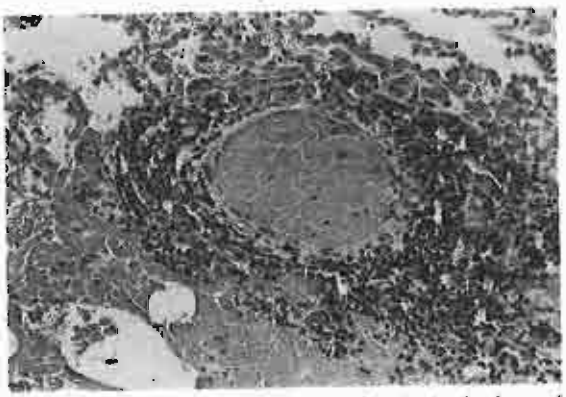

Figure 2—Photomicrograph of a small vein in the lung of horse 5, in which the various layers of the wall contain numerous eosinophils. H&E stain; × 105.

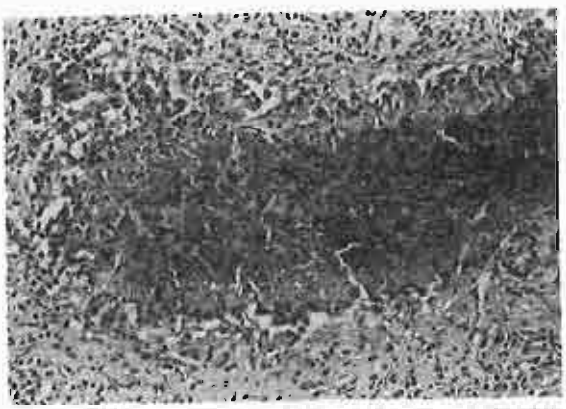

Figure 3—Photomicrograph of a focus of necrotic eosinophils surrounded by fibrous tissue, lymphocytes, macrophages, and giant cells in an area of severely congested and hemorrhagic lung. H&E stain; × 110.

Of the 11 horses, 9 died apparently from exercise-induced pulmonary hemorrhage (EIPH). The nasal cavity, trachea, and bronchi of all 9 horses, except No. 3, were filled with bloodstained froth. The lungs were deep red to black, with a few normal-appearing pink areas in the cranial portion. The lungs were heavy, and felt moderately firm, consistent with being blood-filled. In horses 3 and 5, multiple deep red nodules (1 to 3 cm in diameter) were scattered throughout the lung parenchyma. Focal fibrosis of the pleura, evident as thick white areas over the lung surface, was seen in horses 1 through 5 and horse 9. Some of these areas felt firm and extended into the lung parenchyma. Pathologic changes that could have accounted for the sudden death were not observed in any other system, and macroscopic cardiac lesions were not observed in any horse.

Microscopic examination of the lungs of all 9 horses revealed engorgement of the pulmonary arteries, veins, and capillaries, with hemorrhage into alveoli, bronchioles, bronchi, interstitium, and subpleural tissue. The severity of the engorgement and hemorrhage varied from almost nonexistent to massive in various areas of the lung, but the caudal portion of the lung lobes was the site of the most severe hemorrhage. Pleural and interstitial fibrosis or marked eosinophilic bronchitis and bronchiolitis were accompanied by severe hemorrhage. Some airways with severe eosinophilic and lymphocytic infiltration had extensive hemorrhage, and the lining epithelium, submucosa, muscular layer, cartilage, and peribronchial tissue were separated widely by areas of hemorrhage (Fig 1). Infiltration of eosinophils around and within walls of airways and blood vessels was observed in horses 3, 5, 7, and 9 (Fig 2), and lymphocytes were seen in similar locations in horses 1, 3 through 5, 7, and 9. The severity of the infiltration varied from location to location within the lung—almost nonexistent in some and severe in others. The most severe changes were in the dorsal caudal and hilar areas, but in some horses, multiple, severely affected areas seemed to be randomly distributed. Multiple oblong, oval, or circular foci of degenerating eosinophils resembling parasite migration tracks were observed in the pulmonary parenchyma of horses 3 and 5 (Fig 3). Alternatively, these might have been bronchioles, with attenuated or sloughed bronchial epithelial lining, plugged by bronchial casts of necrotic eosinophils. These eosinophil-containing foci were most numerous within the aforementioned dark red nodules that were visible in the lungs of these 2 horses.

Focally extensive fibrosis of the pleura and interstitium, often including the area around large vessels and airways, were observed in 6 of the horses. Groups of macrophages laden with hemosiderin also were seen within this fibrous tissue, particularly at the junction of the pulmonary parenchyma and the deep layers of the pleura. In horses 1 through 4, 7, and 9, groups of siderophages also were seen in

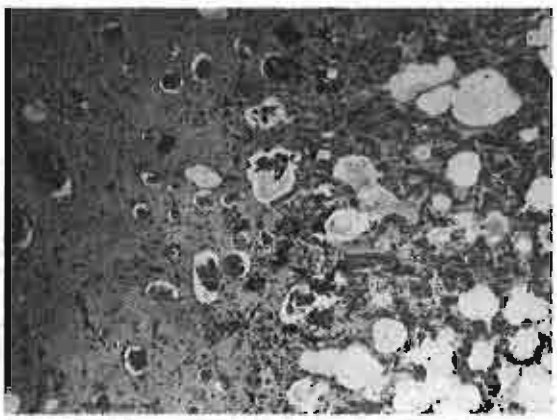

Figure 4—Photomicrograph of a focus of pleural fibrosis, in which are seen congested capillaries and groups of siderophages (arrows). H&E stain; × 40.

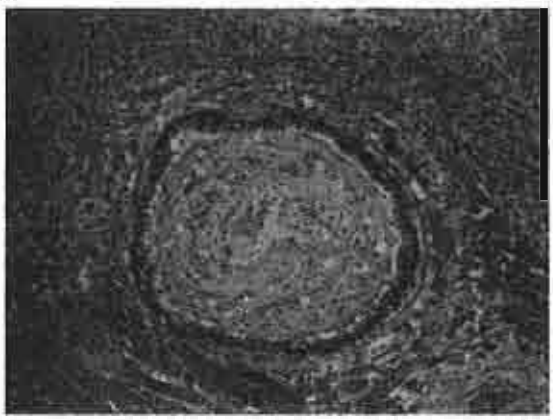

Figure 5—Photomicrograph of mucoid bronchiolitis. The lumen is plugged with mucus and degenerating inflammatory cells. Peribronchiolar connective tissue is infiltrated with lymphocytes and eosinophils, and capillaries are engorged. H&E stain; × 46.

airways (Fig 4), peribronchial connective tissue, alveolar walls, and alveolar lumens. Siderophages infrequently were observed in the alveoli of all horses. Severe focal mucoid bronchiolitis was seen in horses 1 and 3 through 5. In affected areas, the bronchioles and several bronchi were occluded completely with plugs of inspissated mucus and cellular debris (Fig 5). Degenerating inflammatory cells were scattered throughout the lining epithelium, and lymphocytes, eosinophils, and neutrophils infiltrated the bronchiolar walls and peribronchial connective tissue.

In these 9 horses, sudden death was attributed to pulmonary failure secondary to fulminant EIPH. Of the 9, 4 had had epistaxis at the time of death and 5 had previous history of EIPH; the EIPH history of the other 2 horses was unknown.

Horses 10 and 11 were included in the study because they had died during exercise without apparent traumatic cause of death. However, at necropsy, horse 10 had hemoperitoneum attributable to severance of the right internal iliac artery secondary to fracture of the right pubic bone. Other tissue specimens did not have pathologic changes.

Horse 11 had multiple fractures of the left forelimb and third and fourth cervical vertebrae, with macroscopic evidence of spinal cord compression. Other tissues did not have pathologic changes, with the exception of mild pulmonary hemorrhage and eosinophilic peribronchitis in multiple lung specimens.

Sudden death in these 11 exercising horses was attributed to EIPH in 9 horses, exsanguination in 1 horse, and CNS trauma in 1 horse.

Discussion

All horses of this study had lesions sufficient to explain their sudden death during exercise. This differed appreciably from the report of Gelberg et al,[8] who found a 32% probability of such lesions. The majority (82%) of the horses of our study had pulmonary lesions sufficient to explain the sudden death. In the study of Gelberg et al,[8] the authors speculated that sudden death most likely was associated with exercise-induced acute cardiovascular failure; this conclusion was reached despite the fact that only 2 horses had histologic evidence of myocardial disease. Of course, a microscopic lesion causing conduction disturbance, with subsequent heart failure, may have been overlooked.

Epistaxis during or after exercise long has been recognized as a serious problem in racehorses.[9] Its origin in the lungs was suggested,[10] but was not confirmed until fiberoptic endoscopy was used widely.[11,12] Because of its association with exercise, the condition has become known as EIPH. Morbidity of EIPH, its relation to age, and its association with various situations, has been described,[11-13] as has the effect of treatment with various drugs.[12,14-17] Occurrence of EIPH is not random, and it is observed consistently in individual horses.[12,17]

Despite its occurrence in 45 to 86% of racing horses, data are not available on the death rate of horses with EIPH. Our study indicated that EIPH was the cause of death in 82% of the Thoroughbred racehorses that died suddenly (while exercising) of causes unrelated to musculoskeletal trauma. Thus, EIPH should be considered as the most common cause of sudden death in exercising Thoroughbred racehorses.

During the period of the study reported here, approximately 12,000 races were run; therefore, we found the death rate of horses with EIPH to be 1 death/1,500 races.

There is only limited understanding of the pathophysiologic mechanism of EIPH. It is associated with atrial fibrillation,[18] but in human beings, atrial fibrillation is associated with increased left atrial and pulmonary wedge pressures.[19] Paroxysmal atrial fibrillation has been reported in racehorses,[20-23] with most horses reverting to normal sinus rhythm within 24 hours. If transient arrythmia develops in a horse during a race, it might not be detected. Thus, many apparently healthy horses may be exercising with

such arrythmias, and may be at risk for development of EIPH. Exercise-induced pulmonary hemorrhage may be triggered by the asphyxia that develops during breath-holding after horses leave the starting gate.[24] Another hypothesis is that upper airway obstruction, as seen in horses with laryngeal hemiplegia, may initiate pulmonary bleeding during maximal exercise.[25]

Alternative explanations for EIPH have included subclinical respiratory disease resulting in bronchospasm precipitated by exercise.[10,26,27] In our horses, subclinical chronic bronchitis may have been involved, especially in old horses.[26] Bronchiole obstruction and/or scarring might contribute to local increases in perivascular pressure,[26,27] and perhaps might result in pulmonary hemorrhage. Of the horses of this study, 6 had fibrous pulmonary scars, and 4 had eosinophilic bronchiolitis.

In a recent study,[28] extensive small airway disease was observed in close association with the vascular changes in lungs of horses with EIPH. Bronchiolitis was suspected as the early lesion, and increased number and size of bronchial arteries may have resulted.[28] We also suspect that the eosinophils in eosinophilic bronchiolitis associated with allergic lung disease[29] may have a role in alveolar hemorrhage. In the guinea pig, eosinophils contain a metalloprotein that degrades type-I and type-III collagen, which constitute the bulk of lung and blood vessel collagen.[30] Thus, eosinophils could damage the integrity of lung blood vessels and alveolar walls so that increased pulmonary perfusion and intermittent higher airway pressure associated with exercise might precipitate pulmonary hemorrhage. Cytologic findings in tracheobronchial aspirates from many Thoroughbred racehorses were suggestive of small airway disease,[a] and evidence of past pulmonary hemorrhage was detected in many of the horses.

In conclusion, only 4 of the 9 horses that died from pulmonary hemorrhage had epistaxis at the time of death, although all but 1 had blood-stained froth in the airways at necropsy. Thus, pulmonary hemorrhage cannot be ruled out as the cause of death solely because of lack of blood in the nares. Necropsy should include thorough examination of the lungs for the signs of EIPH—deep red to black, heavy, firm lungs, and bloodstained froth in the airways. Detailed examination of the heart and larynx for predisposing lesions also should be performed.

Although the study was designed to include only horses with nontraumatic causes of death, 2 horses that died from trauma were included because of the lack of external signs of such trauma. Because our study indicated that EIPH was the apparent cause of death in the majority (82%) of exercising Thoroughbred racehorses, complete necropsy was necessary to rule out a traumatic cause of death.

[a]Sweeney CR, Roby KA, Soma LR, et al. Cytologic findings from tracheobronchial aspirates of 94 racehorses (abstr), in *Proceedings*. Am Coll Vet Int Med 3rd Annu Sci Prog 1985;125.

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As with most textbooks that contain contributions from several authors, the content varies from section to section. The editors are to be complimented for minimizing variations in quality. Of particularly good quality are the discussions of acute and chronic diarrhea and neoplastic disorders. This may be attributable in part to the fact that these topics lend themselves well to the format used in the text. Topics that require judgment on the part of the veterinarian before using the algorithms are not well suited to this. This problem is exemplified by the discussion of management of traumatic wounds. The decision-making process is clearly described, but unless the veterinarian has accurately assessed the wound before following the algorithm, it is difficult to arrive at the intended conclusion. A similar comment could be made regarding the discussion of biliary tract trauma.

Because nearly all topics were handled in the space of 2 pages, including the algorithms, decisions had to be made regarding what material was to be included. In the discussion of megaesophagus, as well as the discussion of vascular ring anomalies, a Heller's myotomy of the lower esophageal sphincter is recommended for treatment of spasm or stricture of the lower esophageal sphincter. The need for this procedure in veterinary medicine is quite limited, and it is used infrequently. The overall poor prognosis associated with surgical treatment of persistent right aortic arch is not emphasized in this section. In the discussion of airway obstruction, it is unclear as to why castellated laryngofissure was the only technique illustrated for treatment for laryngeal paralysis.

Another problem with the algorithm format is illustrated by the section on heartworm disease: In the algorithm accompanying this discussion, the recommendation is made that the presence of liver and kidney disease in conjunction with evidence of right-sided heart disease should be considered an indication that adult worms be removed via pulmonary arteriotomy. It is doubtful that this statement can be universally applied to all animals in this category. The statement also is made that uncomplicated bile peritonitis is aseptic and does not require abdominal drainage. Although it is probably true that uncomplicated bile peritonitis does not require drainage of the abdominal cavity, it is misleading to imply that this is an aseptic condition. In the algorithm accompanying the discussion of linear intestinal foreign bodies, the reader is informed that surgery is invariably required. Recent reports suggest that, in certain instances, these may be managed expectantly.

Potential purchasers of this text should be aware of the intention of the editors and the limitations of the text. Within this context, the text succeeds in its mission and should be a valuable asset to veterinary practitioners and veterinary students seeking to improve their management of small animals requiring surgery.—*[Decision Making in Small Animal Soft Tissue Surgery. By Allen G. Binnington and Joanne R. Cockshutt. 232 pages; illustrated. B. C. Decker Inc, Burlington, Canada. 1988. Available in USA from CV Mosby Co, 11830 Westline Industrial Dr, St Louis, MO 63146. Price $40.00.]*—DALE E. BJORLING

This paper reviews recent progress in the etiology and pathology of exercise-induced pulmonary hemorrhage (EIPH), a condition which poses an important cause of financial loss to the equine industry. It is hypothesized that all horses experience some grade of EIPH during strenuous exercise. In light of the inflammatory response in lung tissue following infusion of autologous blood and the chronic nature of EIPH in racing horses, every effort should be made to eliminate EIPH during exercise and to ameliorate the inflammatory response once hemorrhage has occurred.

Exercise-Induced Pulmonary Hemorrhage: A Review of the Etiology and Pathogenesis

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Introduction

Exercise-induced pulmonary hemorrhage (EIPH) occurs commonly in performance horses and is an important cause of financial loss to the equine industry. Although the condition has been reported in horses for over 200 years, it has been only within the past 20 years that the source of the bleeding has been documented. This paper reviews recent progress in the etiology and pathology of EIPH. Criteria for classifying the different grades of severity of the syndrome are also offered.

Etiology

During the past 20 years, several theories have been proposed about the etiology of EIPH. In 1974, Cook[1] suggested that hemorrhage from healthy lung was unlikely and that EIPH occurred in horses with chronic lung disease, possibly bronchospasm linked with the early stages of chronic bronchitis and pulmonary emphysema.

In 1980, Robinson and Derksen[2] hypothesized that pulmonary hemorrhage resulted from increased distending forces applied to lung regions not ventilated homogeneously with the rest of the lung. Because lung segments with impeded airways move asynchronously with adjacent lung tissue ventilated normally, this hypothesis was not incompatible with that of Cook.[1] Inelastic scar tissue within the lung parenchyma or pleural adhesions from previous bleeding episodes would be especially susceptible to the accentuated distending forces.

In 1987, O'Callaghan and associates[3] described an increase in bronchial artery circulation in pulmonary areas with evidence of previous EIPH episodes. They proposed that the source of subsequent hemorrhage was the fragile bronchial capillary buds

Continued

in inflamed peribronchial connective tissue and alveolar septa.

Exercise-induced coagulopathies have been proposed to cause EIPH. Reduced platelet adhesiveness and acceleration of thrombin formation with no evidence of fibrinolysis have been reported in exercising horses.[4] One study showed no difference between the clotting profiles of EIPH-positive and normal horses.[5] However, a later study reported that platelet adhesiveness may decrease more in EIPH-positive horses than in non-bleeders following exercise.[6]

A more recent hypothesis proposed by Cook and associates[7] contends that partial respiratory tract obstruction due to recurrent laryngeal neuropathy (RLN) causes EIPH. With upper airway obstruction, a greater negative pressure than normal is created during inspiration. This, along with the presence of pulmonary hypertension during intense exercise, increases the pressure gradient across the alveolar-capillary membrane, resulting in hemorrhage of the alveolar walls.

More recent work by West and associates[8] has persuasively shown that the first step in EIPH is stress failure (microrupture) of the pulmonary capillaries, with RBCs initially entering the interalveolar spaces. The pathological significance of this step is unclear, but evidence suggests that some components in blood are very inflammatory to the alveolar tissue.[9]

The most subtle clinical evidence for EIPH is the presence of hemosiderophages in tracheal washes. Virtually 100% of horses in training show hemosiderophages in their tracheal washes,[10] confirming earlier suspicions that all strenuously exercised horses suffer some degree of lung hemorrhage.

High Blood Pressure and Stress Failure of Pulmonary Capillaries in EIPH

West and associates[8] have recently applied their research on stress failure of pulmonary capillaries (SFPC) in the human lung to the problem of EIPH in horses. The alveolar-capillary membrane is composed of the capillary endothelium, the interstitium, and the alveolar epithelium. The mechanical strength of the alveolar-capillary membrane comes from the Type IV collagen in the interstitium, which is composed of the basement membranes of the capillary endothelial and alveolar epithelial cells.

With electron microscopy, Birks and associates[11] demonstrated an extremely thin (0.535 μm) alveolar-capillary membrane in rabbit lung. At high pulmonary blood pressures, the capillaries bulged into the alveolar spaces. When transmural pressure (capillary pressure minus alveolar pressure) exceeded 40 mmHg, disruption of the pulmonary-capillary wall was observed in rabbits.[12]

In contrast, dogs have a thicker alveolar-capillary membrane (μm 0.759), and transmural pressures in excess of 68 mmHg were required to rupture the membrane.[13] Interestingly, the mean alveolar-capillary membrane thickness of horses is about 0.930 μm. In all species examined, the alveolar-capillary membrane is not uniform in thickness but consists of thin and thick portions. The thinnest walls would have the highest stresses and the greatest probability for failure. The pressure at which SFPC occurs in horses has not been determined. Failure occurs acutely when the pulmonary blood pressure is maximal, typically during intense exercise. Conservative estimates have placed pulmonary capillary pressure in horses during maximal exercise at about 95 mmHg,[14] well above the maximal pressures that can be sustained in rabbit or dog lung without creating stress failure.[12,13] During failure, ruptures appear in the endothelial lining of the pulmonary capillaries, and RBCs escape into the interstitium and alveoli.

However, this theory does not explain the fact that chronic, end-stage EIPH is confined to the dorsocaudal portions of the lung. Because of hydrostatic forces, the greatest intravascular pressures are expected in the ventral portions of the lung. Therefore, EIPH lesions would be expected in the more dependent lung regions. However, one study reported especially high blood flow to the dorsocaudal lung regions.[15] Furthermore, the important pressure measurement is not intracapillary pressure but transmural pressure. Transmural pressure may actually be higher in the dorsocaudal portion of the lung, since alveolar tissue may be squeezed between the diaphragm and lumbar muscle area during strenuous exercise. Furthermore, the dorsal alveoli are probably larger than alveoli in more dependent areas of the lung, and increasing the volume of the alveoli increases the frequency of stress failure in pulmonary capillaries.[16]

It is important to note that the horse is unique among athletic animals in its tendency to become hypoxemic during intense exercise. Even other members of the species (e.g., ponies) maintain normoxia during maximal exercise.[17] The irony concerning the equine alveolar-capillary membrane is that it is too thick to allow adequate gas exchange (physiologically, this limitation is exhibited by arterial hypoxemia during intense exercise) and too thin to protect against the high pul-

Continued

monary capillary pressures generated during intense exercise (thus, the occurrence of EIPH). It appears that the cardiopulmonary vascular system of the racehorse approaches or has reached the physiologic limitations of mammalian systems.

Levels of EIPH

In previous literature about this disease, horses are usually labeled "EIPH-positive" or "EIPH-negative". In light of studies that suggest all racehorses in training have previous evidence of EIPH using BAL[10] and studies that have measured very high pulmonary capillary pressures in exercising horses,[18,19] there may not be any racing horses that are EIPH-negative. If "EIPH-negative" is an inaccurate label for horses, then previous studies that measured the effects of furosemide on EIPH and performance must be evaluated from a different perspective.

We view EIPH as a condition present in all horses subjected to exercise above the level of introductory training. There appear to be at least five separate levels that can be identified based on the location and severity of the pulmonary hemorrhage.

LEVEL 1

Simple rupture of capillary endothelium allows RBCs to escape into the interstitial tissue, but there is no significant loss of cells into the pulmonary alveoli.

LEVEL 2

Rupture of both capillary endothelium and alveolar epithelium allows escape of RBCs into interstitial tissue and alveoli. Following this severity of hemorrhage, hemosiderophages will be present in the fluid of subsequent BALs. Because the amount of blood released into the alveoli may be inadequate to reach the trachea, no endoscopic evidence of EIPH is detectable in Levels 1 and 2 of the disease. Presumably, horses experiencing only Level 1 or 2 of EIPH would be classified EIPH-negative following endoscopic examination.

LEVEL 3

In Level 3 of EIPH, the amount of blood flowing into the alveoli is sufficient to ascend into the trachea and be visualized with endoscopy (Fig. 1).

LEVEL 4 (EPISTAXIS)

Epistaxis occurs in 1 to 2% of racehorses and is evidence of hemorrhage severe enough to be expelled from the nares. Figure 2 is a photograph of epistaxis in a Thoroughbred shortly after finishing a race.

LEVEL 5 (DEATH)

Figure 3 shows the lungs from a horse that experienced Level 5 of EIPH during a race. The horse collapsed 400 meters out of the gate (Fig. 4), fractured C-2 and C-3 of the cervical vertebrae compressing the spinal cord (Fig. 5) as it hit the ground, and died immediately on the track. At necropsy, the entire respiratory passages (nasal cavity, trachea, and bronchi) were filled with bloody froth. The lungs were deep red to black, heavy, and firm, consistent with being blood-filled. Histopathology revealed widespread areas of hemorrhage with bleeding into alveoli, bronchioles, and bronchi. Adjoining alveoli contained erythrocytes and hemosiderin pigment, suggesting previous bleeding episodes. Death was attributed to acute pulmonary hemorrhage.

Although the progression of severity through Level 1 to Level 5 is logical, one study [20] reported that some horses died from EIPH without evidence of epistaxis. At necropsy, those horses exhibited bloody froth in the trachea; however, no blood was evident in the nares.

This five level description of EIPH integrates our knowledge of its epidemiology and pathogenesis and clearly establishes this condition as a continuum. At its least intrusive, this condition occurs in all horses in training and racing; at its most dramatic, it can result in collapse and sudden death of horses early in a race.

While Level 1 is currently not clinically distinguishable from Level 2, the singular event of this level (escape of blood into interstitial tissue) may, ultimately, be the most significant occurrence of EIPH, since it has the greatest potential to produce cumulative pathological changes. It is likely that red blood cells extravasated into the interstitium are cleared via lymphatic drainage, thereby leaving no suggestion (such as hemosiderophages) of past damage. It is also likely that the presence of hemoglobin in the interstitial tissue is more of an irritant than the presence of red cells in the alveolar sinus. Since simple infusion of autologous blood causes definite pathological changes,[21] it seems likely that Level 1 EIPH, possibly associated with minimal clinical signs, could ultimately be a very significant source of long-term pathological changes.

At the other end of the EIPH spectrum, the potential impact of Level 5 events on racing must also

Continued

FIG. 1 — Endoscope view of blood-tinged trachea in a horse following a race. Courtesy of John R. Pascoe, University of California, Davis, California.

FIG. 2 — Epistaxis in a Thoroughbred after finishing a race. Courtesy of Richard H. Galley, Willow Park, Texas.

FIG. 3 — Blood-filled lungs from a horse that experienced Level 5 of EIPH during a race.

FIG. 4 — Longitudinal fracture of C-2 and fracture of cranial portion of C-3 compressing spinal cord.

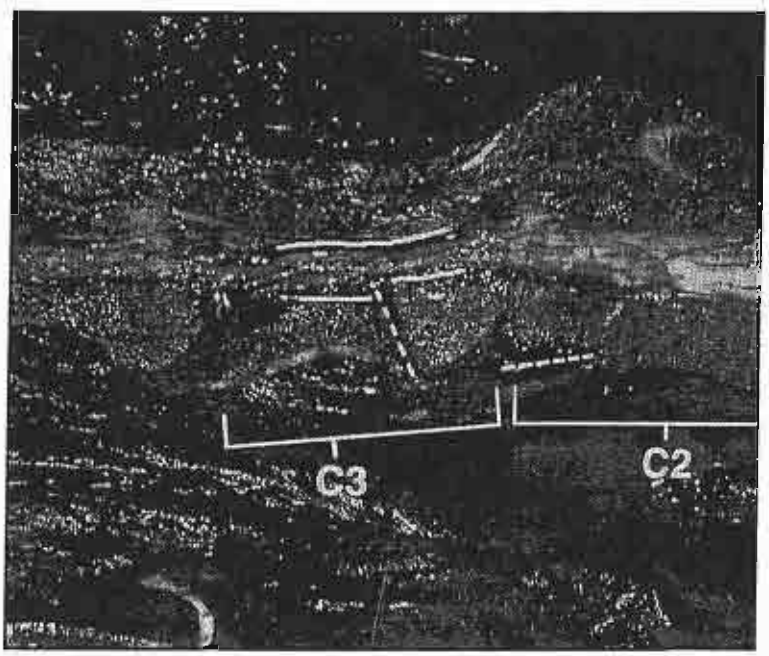


FIG. 5 — Severe discoloration of ~50% of dorsocaudal section of lung. The affected area contains dark blue staining with brown staining and irregular areas of trapped air around the margins. Reproduced with permission from O'Callaghan and associates.[3]

be understood. Massive hemorrhage into the lungs of a racing horse can lead to acute asphyxiation on the track, either during the race or very soon after the completion of the race. The impact of this spectacle is significant on the racing public, and this event needs to be recognized as one sequelae of the EIPH continuum.

Continued

Pathology of EIPH

The first study of EIPH in a large number (813) of Thoroughbreds was from horses racing in Hong Kong and has generated several papers on this topic. Mason and associates[22] performed postmortem examinations on 26 of these horses and reported that 96% of the lungs exhibited bronchiolitis and gross lesions indicative of previous pulmonary hemorrhage.

In another paper from the Hong Kong study, O'Callaghan and associates[9] described the lung pathology in Thoroughbred racehorses of known EIPH status. The most consistent gross finding was a variable amount of dark blue, blue/grey, or blue/brown subpleural staining of lung parenchyma. The lesions were bilaterally symmetrical and typically confined to the dorsocaudal extremities of the lung. In more severely affected animals, the lesions extended cranially along the dorsal surface of the lung. The bilateral symmetry and lesion distribution indicated horizontal spread to other dorsal bronchopulmonary segments rather than proximal extension of a lesion from its distal origin. In contrast, axial spread along an affected bronchus or artery would create lesions on the ventral portion of the lung as well, which was not the case.

In more severely affected lungs, lesions covered 30 to 50% of the lung surface. Stained areas were firmer, with a spleen-like consistency and did not collapse to the same degree as did the normal lung tissue. Similarly, stained areas of the lung did not readily inflate. Another classic finding was the increased bronchial artery circulation in the discolored areas of the lung. In the most severely affected areas, there was extreme vascular hypertrophy of the bronchial arteries.

The effects of furosemide on performance and the aesthetics of horses bleeding during a race have received much attention. However, little concern has been focused on the pathology occurring in lung tissue and the effect of that pathology on future performance. Following disruption of the alveolar-capillary membrane, all of the components of blood enter the pulmonary interstitium and alveoli. Disruption of endothelial cells exposes the endothelial basement membrane, which permits the products of coagulation to accumulate in the injured tissue. This causes microthrombi and impaired microcirculation. As healing occurs, mononuclear phagocytes and mesenchymal cells (immature fibroblasts that can develop into a variety of mature cell types) enter the alveolar tissue. Repair of the interstitium also includes restoration of the extracellular matrix, with the replication of fibroblasts and the deposition of connective tissues in an ordered fashion.[9]

This fibroproliferative response is the same response that prevents surface wounds from becoming life-threatening. A fibroproliferative response to a cut in the skin is appropriate and necessary to restore the barrier between the organism and an inhospitable environment. However, the same response in the lung causes formation of scar tissue that interferes with expansion and contraction of the lungs and impedes gas exchange. In human patients that have experienced maladaptive repair of lung tissue following injury, hypoxemia is a common sequelae.[9] The hypoxemia seen in exercising horses may be initiated or worsened by sequential pulmonary fibrosis from multiple episodes of EIPH.

To better understand the effects of bleeding on lung tissue, Tyler and associates[21] infused single and multiple (5 times at 7-day intervals) doses of autologous blood and saline into specific airways of 11 horses. Reactions to infused blood varied from very mild to severe, with the lesions from multiple infusions being more severe than from single infusions, suggesting EIPH may have a cumulative effect on lung tissue. Although the lesions created in this study were less severe than those seen with naturally occurring EIPH, many similarities were observed including the presence of hemosiderophages in tissues and airspaces, bronchiolitis, and increased connective tissue.

The reduced severity of lesions seen after infusion of blood in comparison with end-stage EIPH is not surprising. If some level of EIPH occurs each time a horse is exercised strenuously, the number of hemorrhagic insults to the lung will be much greater than the number of infusions (maximum = 5 infusions) performed in these experiments. Because naturally occurring EIPH is also accompanied by membrane damage and blood components trapped in interstitial pulmonary tissue, the lesions associated with the naturally occurring disease would be more severe than those created by infusing autologous blood, which was deposited only in the alveoli.

It was concluded that bronchiolitis and increased connective tissue are sequelae rather than antecedents of the pulmonary hemorrhage seen in EIPH. Since strenuous exercise frequently causes bleeding into the lung tissue, pulmonary scarring and loss of function are likely to be common sequelae of strenuous exercise in horses.

Continued

Surprisingly, there was also a marked reaction of lung tissue to multiple saline infusions characterized by bronchiolitis, increased formation of connective tissue, and loss of function. This suggests that even simple bronchiolar lavage carries a significant cost in terms of pulmonary function — a cost which must be weighed against the benefits of lavage as a diagnostic or therapeutic procedure.

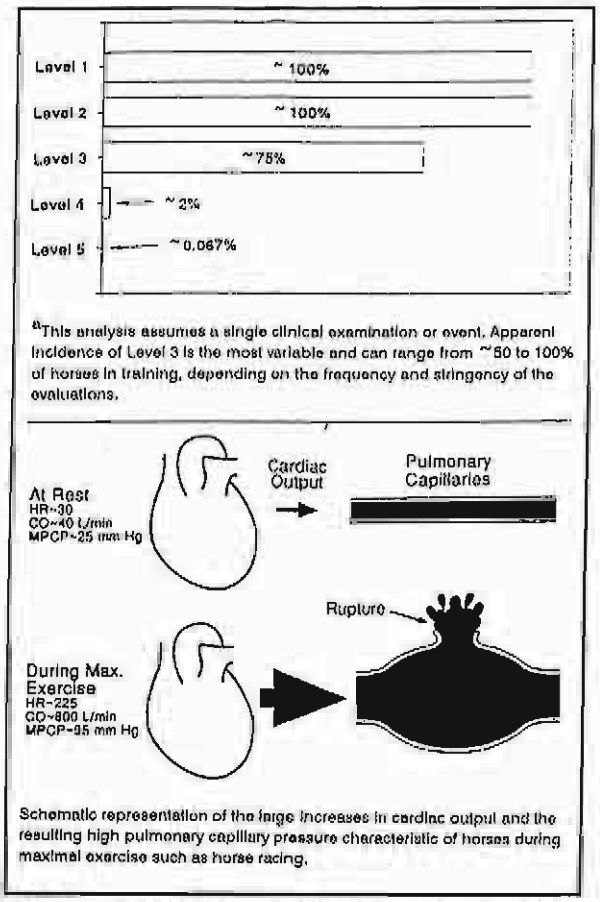
Therefore, the pulmonary healing process appears to be a double-edged sword. Although the fibroproliferative response is required to repair lung injury, the response can also obliterate alveolar air spaces, increase alveolar-capillary membrane thickness, and reduce elasticity of alveolar tissue. All of these sequelae impair gas exchange, a critical and limiting factor in the performance horse.

Summary

Exercise-induced pulmonary hemorrhage (EIPH) was observed in the earliest Thoroughbreds and occurs worldwide in all breeds. During exercise, Thoroughbreds develop exceptionally high pulmonary blood pressures leading to stress failure of pulmonary capillaries. Stress failure of the capillary endothelium allows blood into the pulmonary interstitial tissue, the simplest microscopically identifiable lesion (Level 1) of EIPH.

Failure of the entire capillary-alveolar structure releases blood into the alveolar spaces (Level 2 of EIPH) causing the formation of hemosiderophages, the most subtle clinical sign of EIPH reported to date. All horses in strenuous training show hemosiderophages, suggesting that all race horses experience some grade of EIPH (Fig. 6). More severe hemorrhage results in the appearance of blood in the trachea (Grade 3 EIPH). Most racing horses attain this grade of EIPH if repeatedly exposed to intense exercise. A small proportion (1 to 2%) bleed from the external nares (Grade 4 EIPH), which is the classic "bleeder". Finally, a very small proportion of horses (approximately 0.067%) die acutely during a race or post-race from severe pulmonary failure (Grade 5 EIPH).

EIPH lesions are chronic and cumulative and include hemosiderophages, marked bronchiolitis, vascular hypertrophy, and increased bronchial artery circulation. Multiple infusion of autologous blood into the lungs of horses created lesions similar to but less severe than naturally occurring EIPH lesions. Sequelae of blood in the alveolar tissue includes bronchiolitis, increased connective tissue, loss of function, and neovascularization which may precipitate subsequent episodes of EIPH. It is hypothesized that all horses experience some grade of EIPH during strenuous exercise. In light of the inflammatory response in lung tissue following infusion of autologous blood and the chronic nature of EIPH in racing horses, every effort should be made to eliminate EIPH during exercise and to ameliorate the inflammatory response once hemorrhage has occurred. ■



[a]This analysis assumes a single clinical examination or event. Apparent incidence of Level 3 is the most variable and can range from ~50 to 100% of horses in training, depending on the frequency and stringency of the evaluations.

Schematic representation of the large increases in cardiac output and the resulting high pulmonary capillary pressure characteristic of horses during maximal exercise such as horse racing.

FIG. 6 — Apparent incidence of EIPH in training and racing horses.[a] HR = heart rate, CO = cardiac output, MPCP = mean pulmonary capillary pressure.

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Furosemide, the Prevention of Epistaxis and Related Considerations: A Preliminary Evaluation

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KEY WORDS: Furosemide, Epistaxis, Exercise Induced Pulmonary Hemorrhage [EIPH], Regulatory Approval, Acute/sudden death, Horses, Jockeys, United States.

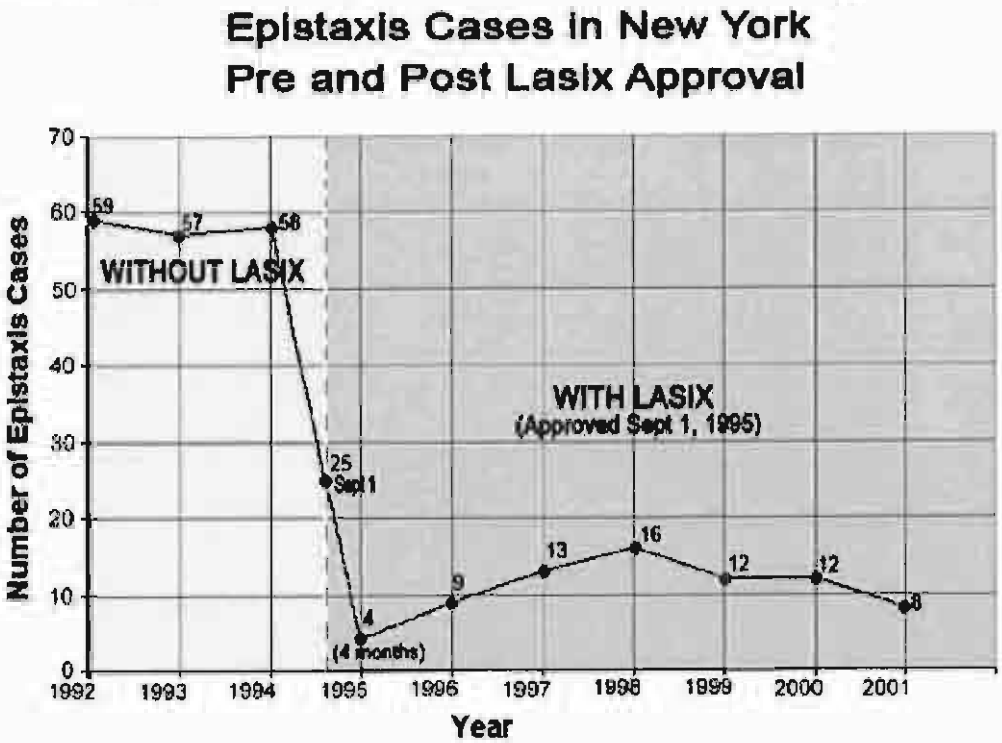
ABSTRACT

Furosemide is widely approved in the United States, Canada and elsewhere in the Americas for the prevention of Exercise Induced Pulmonary Hemorrhage [EIPH]. We review the scientific evidence for the efficacy of furosemide in reduction/ prevention of EIPH, including, presumably, EIPH related acute/sudden death in racing horses. We present evidence from the scientific literature and our own experience, clinical and otherwise [RHG, AMB, DVL, and TT], that EIPH driven acute/sudden death in racing horses has significant adverse health consequences for horses and jockeys. We then outline the adverse effects on equine and human [jockey] health and welfare to be expected when furosemide is not approved for use in racing horses or, where approved, if approval is withdrawn.

FINDINGS

Epistaxis, ie, bleeding from the nose in racing horses, has been observed by horsemen since at least the seventeenth century. [1] In the late 1960s, injectable furosemide (Lasix, Salix) became available in the United States and soon thereafter furosemide was

Figure 1 Effect of Furosemide on Epistaxis Cases/Year in New York Racing



being used in the prevention of epistaxis. This initial use of furosemide was based on clinical experience and, until recently, there was little scientific evidence concerning its efficacy in the prevention of epistaxis. On the other hand, most American racing states have long since approved the use of furosemide on race day for the prevention/alleviation of epistaxis, now known to be a component of Exercise-Induced Pulmonary Hemorrhage [EIPH]. Very recently, however, questions have been raised about whether or not the routine pre-race treatment of racing horses with furosemide for the prevention of epistaxis/EIPH passes "the smell test,"[2] which has led to renewed examination of the scientific basis for the pre-race use of furosemide in North American racing.

We now draw attention to some clinical evidence establishing, in large numbers of racing horses, the efficacy of furosemide in reducing the incidence of epistaxis. These data were first communicated by Mr. Bill Heller in his monograph on Lasix, "Run, Baby, Run,"[3] where these data have remained hidden in plain sight since the 2002 publication of this book. Reviewing this book as part of an overall review of the literature on furosemide in the horse, we noted, on pages 112 and 113, a table entitled "New York By The Numbers, Cases of Epistaxis," dated 1992 to 2001. Inspection of these data immediately clarifies the dramatic reduction in the incidence of EIPH in New York racing following the 1995 approval of furosemide. We now present this data in standard graphical format (Fig 1), which clarifies the remarkable efficacy of furosemide in reducing the incidence of epistaxis in horses racing in New York in the 6 years and 4 months immediately following approval of furosemide in New York racing.

In analyzing these data we note the following facts. The first is that epistaxis is, by definition, clearly observable bleeding ("dripping of blood") from the nose, sometimes defined as from both nostrils. Epistaxis is, however, only one manifestation of what is now known as Exercise-Induced Pulmonary Hemorrhage (EIPH).[4] It is also the only manifestation of EIPH observable without special equipment, and

as such, epistaxis has long been recorded in performance horses.[1,4] Epistaxis is thus the historical "low tech" manifestation of EIPH, with acute sudden death due to EIPH, only recently recognized by science, representing the most severe clinical outcome of this disease.[5,6,7]

All manifestations of EIPH other than epistaxis, including acute/sudden death due to EIPH, require scientific tools and expertise to identify as being EIPH related. [5] As such, the data represented in figure 1 reflect directly on the second most severe manifestation of EIPH, namely epistaxis, and these results speak directly and very compellingly to the efficacy of furosemide in reducing the incidence of both epistaxis and presumably the entire EIPH syndrome in all its various manifestations. The solid circles (O--O) show the total number of epistaxis cases in the previous 12 months of New York racing for 1992 to 2001, inclusive. Furosemide was approved in New York on Sept. 1, 1995, so the 1995 figures are split, with the number 25 representing cases prior to Sept. 1, and the number 4 representing cases from Sept. 1 to Dec. 31, 1995. The annual mean rate of EIPH cases prior to 1994 was 58 per year, and post Sept. 1, 1995, 11.6 per year. These data show that approval of furosemide for use in New York was associated with an immediate and well maintained essentially 80% reduction in the incidence of epistaxis in New York Racing. Data re-plotted from Heller, "Run, Baby, Run," 2002.[3]

With respect to the New York data, review of figure 1 shows that for the years 1992, 1993, and 1994 the numbers of epistaxis cases reported in New York racing were, respectively, 59, 57, and 58, a consistent average of about 58 epistaxis cases per year, which equates to approximately one case per week prior to approval of furosemide. Then, on September 1st, 1995, furosemide was approved for use in New York racing, and the number of epistaxis cases dropped immediately, to a total of 4 cases for the 4 remaining months, September to December, inclusive. In the following year, 1996, the total number of epistaxis cases was 9, followed by 13 in 1997, 16 in 1998, and 12 each in 1999 and 2000, and then a total of 8 cases in 2001, by which time the proportion of horses racing on furosemide in New York had increased to 88.3%. Overall, therefore, in the six calendar years starting on January 1, 1996 and continuing to December 31, 2001, there were a total of 70 epistaxis cases over 6 years, for an average of 11.6 epistaxis cases per year during the first 6 years of furosemide approval in New York.

We also note that this rate of about 11.6 cases of epistaxis per year was remarkably consistent, in that in the last 4 months of 1995, September 1st, 1995 to December 31st, 1995, the first 4 months during which New York raced on Lasix/Salix, there was a total of four cases of epistaxis reported, completely consistent with the subsequent overall average of 11.6 epistaxis cases per year for the following 6 years.

These data are very compelling, and the conclusion to be drawn is that approval of furosemide in racing horses in New York immediately, and we emphasize the word immediately, reduced the incidence of epistaxis by almost 80%. The effect was immediate because it was apparent within the first month of approval of furosemide, and the incidence of epistaxis remained, on average, at essentially the same reduced level in New York racing, 11.6 cases per year, or approximately one case per month, over the following six calendar years. We also note that this reduced rate of epistaxis represents a close to 80% reduction from the baseline 1992-1994 rate of approaching five epistaxis cases per month prior to the regulatory approval of furosemide for use in New York racing.

An unusual aspect of these data is that they have to our knowledge remained unrecognized in the scientific literature. New York was the last major U S racing state to permit use of furosemide, and as such, there was a clearly defined time point after which furosemide was permitted for use in New York. Given the reluctance of the authori-

ties to approve the use of furosemide in New York racing, it may be understandable that these data were not reported earlier. In any event, it is clear from these data that in the actual racing situation, furosemide is remarkably effective in reducing the incidence of epistaxis, and it would have been helpful if these data had been communicated earlier in the scientific literature.

The second point of interest is that these New York data almost certainly underestimate the true efficacy of furosemide in preventing epistaxis. Because New York did not allow the use of furosemide in racing horses, any New York area horses with a propensity to bleed were more likely to race in neighboring jurisdictions that permitted furosemide. Additionally, since EIPH is associated with reduced racing performance, this provided further incentive for horses with any tendency to EIPH to race outside of New York. As such, it is reasonable to assume that the baseline epistaxis rate of 58 per year reported for 1992-1994 actually underestimates the true baseline incidence of epistaxis in North American racing, because horses known to be EIPH prone would tend to have been raced outside of New York. These data, compelling as they are, showing an almost 80% reduction in the incidence of epistaxis after the introduction of furosemide, almost certainly underestimate the true clinical efficacy of furosemide in reducing the incidence of epistaxis.

These data also speak to the lack of effective alternative therapies for epistaxis, non-race day therapies or otherwise.[8-10] Where use of furosemide in the prevention of epistaxis/EIPH is prohibited, the likelihood of use of alternative epistaxis prevention therapies increases. If we make the reasonable assumption that horsemen racing in New York were likely to use any legitimate alternative therapy for epistaxis available to them that did not contravene the rules of racing in New York, we must again assume that the 1992-1994 EIPH incidence figure represent the incidence of epistaxis in the presence of whatever alternative therapies were available to New York horsemen. Again, the demonstrated efficacy of furosemide likely represents the effect of furosemide over and above any possible baseline reducing effect of other available anti-epistaxis therapies.[11]

We must also note that the original clinical observations that furosemide reduced the incidence of epistaxis in racing horses were made in the late nineteen sixties and early nineteen seventies by equine veterinarians and horsemen soon after the introduction of injectable furosemide. These observations were made prior to the introduction of the fiberoptic endoscope and our resulting increased understanding of EIPH and its prevalence in racing horses. The observations reported here fully support these early insightful clinical observations and interpretations and the various decisions since then by equine veterinarians, horsemen and racing authorities to support the prerace use of furosemide in the prevention of epistaxis, as it was then understood, and the entire EIPH syndrome, as it is now understood. As such, these findings leave no reasonable room for doubt that furosemide will also reduce the incidence of acute/sudden deaths during racing due to EIPH.

As well as the data reported here, it is also appropriate for us to acknowledge the recent (2009) highly significant contribution by Hinchcliff, Morley, and Guthrie in this area.[12] These workers performed a classic randomized, blinded, placebo-controlled crossover study on the efficacy of furosemide in preventing EIPH in racing horses. This study established that pretreatment with furosemide reduced the incidence of EIPH in 167 thoroughbred horses under simulated racing conditions at Vaal Racecourse, Johannesburg, South Africa, at, we might note, an altitude of 4,671 ft.

While this study provides strong experimental evidence that pretreatment with furosemide reduces the intensity of EIPH in horses under racing conditions in South Africa, this study did not directly address the efficacy of furosemide in reducing the incidence of epistaxis. We respectfully sug-

Figures 2a and 2b: *Acute/Sudden Death due to EIPH as an Equine and Jockey Safety Hazard*

These photographs record an acute/sudden death EIPH incident in U S Quarter Horse Racing. The horses were moving at approaching 50 mph; the far horse is crashing to the track associated with an acute/sudden EIPH event, and the jockey is being thrown onto the track. The close-up, figure 2b, highlights the blood in the horse's exhalation, consistent with these events being triggered by an acute/sudden EIPH episode. Centennial Racetrack in Littleton, Colorado, altitude 5,389 ft., photographs courtesy of Dr. Richard H. Galley, Willow Park, Texas.

gest that the New York data presented here provides further evidence that is fully supportive of the results obtained by Hinchcliff and his colleagues in their South African study, and extends the clinical efficacy of furosemide to the very effective prevention of epistaxis. Together these studies, as well as approaching 40 years of accumulated clinical experience make an extremely strong scientific case for the use of furosemide in the prevention/alleviation of the EIPH group of syndromes, including the propensity of EIPH to produce acute/sudden death in racing horses.[5,13-17]

Acute/sudden deaths during racing due to EIPH occur when the hemorrhage is sufficiently voluminous to acutely interfere with respiration/blood oxygenation, such that the horse collapses and dies acutely on the racetrack. Although acute/sudden death during racing due to EIPH has long been known to equine practitioners, more recent work has shown that acute/sudden death from EIPH can occur without blood being visible at the nostrils, as shown by the work of Gunson and her colleagues [1988] and others[5], Morales et al [7] and work from our group.[17] Based on this work, it is now very clear that one of the outcomes of EIPH is acute/sudden death of a horse during racing with no obvious external signs of epistaxis. Review of the relevant literature, including the 1988 paper by Gunson and coworkers suggests that in the U S such acute deaths during racing and training occur approximately once per 1,500 Thoroughbred races, and that a substantial proportion, approximately 60% of acute deaths during racing are due to EIPH.[5] As such, the data presented here suggests that pretreatment with furosemide is likely to reduce the instance of such EIPH related sudden deaths during racing by approximately 80%, a very significant contribution to equine and human safety in racing.

Furthermore, we must also note that the Gunson analysis almost certainly underestimates the true incidence of EIPH related acute deaths in racing horses, since this study was carried out after use of furosemide had been approved in Pennsylvania racing (personal communication, Dr. Corinne Sweeney to TT, July 2011). As such, a true estimate of the incidence of EIPH related acute/sudden deaths in racing horses under U S conditions in the absence of furosemide is likely to be substantially greater than the once per 1,500 thoroughbred races reported by Gunson and coworkers, with the expected EIPH related acute sudden deaths figure in the absence of furosemide being between

four and five fold greater, based on the data of figure 1.

With respect to the matter of equine and human safety, the question then becomes what is the incidence of equine and human deaths in racing caused by the EIPH driven acute/sudden death syndrome? At this point, we definitively know from the experience of four of us [RHG, AMB, DVL, and TT] that such EIPH related acute death events occur in racing horses, and that these events also carry clear and highly significant risks for jockeys. The incident represented in figures 2a and 2b occurred at Centennial Racetrack in Littleton, Colorado, in the early to mid 1970's, shortly prior to approval of furosemide for use in Quarter Horse Racing in Colorado. One of us, RHG, was the treating veterinarian. The photograph was taken as the horses reached the finish line and fortunately, as may happen in Quarter Horse racing, there were no horses directly behind the horse that went down. The jockey in question was slightly injured, but the horse did not survive the incident. This photograph therefore records a not an atypical EIPH related acute/sudden death event in Quarter Horse racing, which incident resulted in one equine death and, in this particular case, minor injuries to the jockey. The incident presented in figure 3 represents another EIPH related sudden death incident in racing, this time, in Thoroughbred racing at Ruidoso Downs, New Mexico, altitude 6,720 ft, in the mid 1980s. RHG was again the treating veterinarian and the horse in question was not treated with furosemide. In this particular incident, once the acute/sudden death horse went down, two following horses "went over" the "down' horse/jockey. The horse died on the racetrack, and the jockey sustained career ending injuries [RGH].

These photographic presentations emphasize that EIPH related acute/sudden deaths of horses on the racetrack are not infrequent occurrences, and when they do occur, they have immediate highly significant implications for the health and welfare of the horses and jockeys involved. Additionally, we must also keep in mind that when a horse goes down in a racing situation, there is always a statistical probability of following horses and jockeys becoming involved in the event, and this sequence of secondary events is more likely to occur in Thoroughbred than in Quarter Horse racing.

Figure 3: *EIPH related sudden death, Thoroughbred racehorse, Ruidoso Downs,NM, mid 1980s. Photograph courtesy of Dr. Richard H. Galley, Willow Park, Texas.*

These findings are in good agreement with the clinical experiences of two of us, Dr. A. Morales Briceño and Dr. Diana Villoria Leon, presented in detail elsewhere in their recently published work [2011].[7] Reporting on the incidence of EIPH related acute/sudden death in racing at "Hippodromo La Rinconada," the National Racecourse in Caracas, Venezuela, they recorded 23 cases of acute/sudden death due to/caused by EIPH. These diagnoses were confirmed by full diagnostic necropsies and toxicological examination for medications related to EIPH, which evaluations were performed on each individual animal over the 3- year period from 2008 to 2011, inclusive.

Over these 3 years at "Hippodromo La

Rinconada," there were a total of 44,928 starts, and this population of starters yielded 23 acute /sudden deaths that were on necropsy confirmed as being due to or caused by EIPH. This amounts to one EIPH related acute/sudden death per 1,953 starts. Additionally, we [AMB, DVL] noted the incidence of jockey injuries associated with these EIPH events, which involved 85% of the jockeys riding these horses. Based on this percentage jockey injury rate, there was one jockey injury from EIPH related acute/sudden death per 2,298 starts over this 3-year period of racing at La Rinconada. At this time, however, we have no data on the nature and severity of the injuries sustained by the jockeys involved in these EIPH related acute/sudden death incidents in Caracas.

This rate of acute/sudden deaths caused by EIPH in racing in Caracas is significantly higher than the rate reported by Gunson and co-workers, who estimated one EIPH related sudden death per 1,500 races, with 9 horse fields. On this basis, the Pennsylvania rate works out at about one EIPH driven acute/sudden death event per 13,500 starts. This approximately seven fold higher EIPH acute /sudden death rate in Caracas compared with the estimated rate in Pennsylvania is unexpectedly large, and the reasons for this difference are not immediately apparent.

One major difference between the Pennsylvania and Caracas racetracks is the higher altitude of the Caracas racetrack. This racetrack, La Rinconada, at about 2,950 feet above sea level, is elevated compared with Penn National racecourse, at an elevation of 459 feet, and Pennsylvania Park, at an elevation 36 ft. Similarly, Centennial Racetrack, Littleton, Colorado, is at an altitude 5,389 feet and Ruidoso Downs, New Mexico, is at an altitude of 6,720 feet, as noted in figs 2a and 2b and 3.

With respect to the effect of altitude, we note that the principal scientific report to date on the relationship between altitude and EIPH is that of Weideman et al [2003][18] who reported that in South Africa EIPH appeared to be more frequent at sea level than at higher altitudes. If this interpretation is correct it suggests that the altitude of the Vaal Racecourse, Johannesburg, South Africa, at 4,671 ft., made the Hinchcliff et al [12] demonstration of the preventative effect of lasix on EIPH more challenging than such a demonstration would have been at sea level. This interpretation is also consistent with the unusually small number of class 4 EIPH scores in the Hinchcliff data, as pointed out by a colleague in discussions on this matter. Additionally, as a further confounding factor in these EIPH acute/sudden death studies, we note the significant variability in pathological diagnoses on entire equines[16, 17] presumably due at least in part to the extremely large volume of equine tissue to be subjected to histopathological analysis during equine necropsies.

In summary, for reasons that are unclear, and apparently unrelated to altitude, thoroughbred horses racing in Caracas, Venezuela, show an unusually high incidence of EIPH associated acute/sudden deaths, about one EIPH associated acute/sudden deaths per 1,953 starts. This acute/sudden death rate is about six-fold the rate reported in the earlier Pennsylvania study, and the reason or reasons for these differences are not clear. The fact that racing at La Rinconada is at about 2,950 feet above sea level is considered by Weideman et al. to work against the apparent discrepancy, since he and his colleagues consider that altitude above sea level is associated with a reduced incidence of EIPH. We also note, however, that the concept of increased altitude reducing the rate of EIPH is not consistent with the clinical experience of veterinarians, including one of us [RGH], working in the Western United States, where racing takes place at attitudes of up to 6,720 feet at Ruidoso Downs, New Mexico. Unrelated to the role of altitude, however, is the hard reality that 85% of the jockeys involved in EIPH related acute/sudden death events in horses racing at La Rinconada suffered injury, and that although the extent and severity of these injuries are not available to us, at least one jockey in the

experience of one of us [RHG] suffered a career ending injury associated with acute/sudden death from EIPH [fig 3].

From the data of Gunson and her colleagues we can conservatively estimate the number of EIPH related acute/sudden death related events likely to occur in one year in American Thoroughbred racing. Based on their data, Gunson and their colleagues estimated about one EIPH related acute sudden death per 1,500 starts. Gunson reports nine horse fields, which works out at one EIPH related acute sudden death per 13,500 starts. There are about 417,492 plus thoroughbred racing starts per year in the United States, so this works out at about 31 acute/sudden-death EIPH related events /year in thoroughbred racing in the United States, on the assumption that furosemide is permitted. In the absence of furosemide, however, based on the data in figure 1, we may expect a four to five fold increase in the number of acute sudden-death EIPH related events, to around 155 events/year, or more than three per week.

Gunson also noted that 2 of her 9 reported EIPH cases were acute sudden death in not racing horses, so the final figure is about 120 acute sudden death cases in U S racing per year based on Gunson's data. However, what it is not possible to estimate at this time is the number and intensity of the jockey injuries likely to be associated with these 120 more or less EIPH related acute/sudden-death events in American racing per year in the absence of furosemide, although most of these injuries are likely to be highly significant for the actual individuals involved. [fig 3].

CONCLUSION

These scientific findings, therefore, have implications far beyond equine health and welfare. This is because while pretreatment of racing horses with furosemide serves to reduce the incidence of epistaxis and the various equine pulmonary syndromes associated with intense exercise and EIPH by about 80%, there is every reason to believe that furosemide also serves to reduce, again by about 80%, the incidence of EIPH driven acute/sudden death syndrome in horses in training and racing. By definition, such EIPH related acute/sudden death incidents have the potential to cause severe, including career ending [fig 3] and potentially fatal injuries to jockeys and others riding these horses. As such, the currently in place regulatory approval for use of furosemide in the prevention of EIPH related syndromes in racing horses has a very direct positive and ongoing protective effect on the health and safety of jockeys racing in the United States and elsewhere in the Americas.

Given these scientific realities, we respectfully suggest that it would be unethical and inappropriate, on humane grounds with respect to equine health and welfare, and also on humane and workplace safety grounds with respect to jockeys, for any entity to ban the use of furosemide in racing horses. This is because to do so would be to knowingly significantly increase the risk of serious injury or death for jockeys or others riding racing/performance horses. In sum, any move to disapprove or to withdraw approval for furosemide as an EIPH preventive in racing horses is, from review of the available scientific literature, a move that will immediately and directly increase the risk to life and limb for both the horses and jockeys involved in racing or any other equine event involving exercise sufficiently intense to induce pulmonary hemorrhage.

LIST OF ABBREVIATIONS

[EIPH] Exercise-Induced Pulmonary Hemorrhage

COMPETING INTERESTS

The authors declare no competing interests

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NOTE ADDED IN PROOF

With respect to the data of figure #1 re-plotted from Heller 2002, our attention has recently been drawn to an undated memorandum on the New York Racing Association (NYRA) letterhead of Dr. Anthony Verderosa, DVM, Chief Examining Veterinarian. In this memorandum Dr. Verderosa notes that his analysis of the data on the rates of epistaxis in New York racing for the years 1990 to 2000, that is before and after the introduction of furosemide, showed that the introduction of furosemide in 1995 produced a ">400% decrease" in post race Epistaxis ("Bleeders") EIPH. This is essentially the same conclusion that we drew from what are presumably the same data presented in the Heller book, and appear to fully and independently support our analysis and conclusions presented herein concerning the clinical efficacy of furosemide in preventing epistaxis in New York Racing.

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APPENDIX #1

1/ BASIS FOR MY OPINIONS; MY TRAINING AND EXPERIENCE AS A VETERINARIAN PHARMACOLOGIST AND TOXICOLOGIST:

My opinions are based on my training and experience including my 54 plus years as a veterinarian; I graduated with high standing from the College of Veterinary Medicine, University College Dublin, National University of Ireland, in 1964 and have been an active Member of the Royal College of Veterinary Surgeons [MRCVS] since 1964. I have 48 years of professional experience as a doctoral level pharmacologist [Ph.D. in pharmacology, Medical Center, University of Toronto, 1970], as a faculty member teaching Pharmacology to Medical, Osteopathic and Veterinary students at Michigan State University, 1970 to 1975, and my subsequent experience as a Professor of Veterinary Science and a Professor in The Graduate Center for Toxicology at the University of Kentucky, 1975 to date, of which Center I was Director for a number of years., and I am also a long time Diplomate of the American Board of Toxicology (1980-2015). I am currently licensed to practice veterinary medicine in Kentucky, where my license number is NS 1053, and have in the past been licensed to practice veterinary medicine in Ontario, Canada [1964-1978] and the British Isles, 1964-date.

In the area of equine medications and drug testing and therapeutics, I have written the basic text in this area, "Drugs and the Performance Horse", I have written/published approaching 500 papers and I also hold a number of patents and have created intellectual properties/companies and reference standards related to equine drug detection, drug detection, pharmacokinetics and pharmacodynamics and the therapeutic efficacy of drugs and related therapeutic agents in the horse. With regard to equine medication regulation, in 1982 I performed the research on which the RMTC/ARCI regulatory threshold and withdrawal time guidelines for Furosemide/Lasix are based; among other more recent research contributions and I have also synthesized a significant number of the reference standards and internal standards on which the RMTC regulatory thresholds are based, and a number of these papers and documents are attached to my vitae as [Appendix #1].

Additionally, I have, throughout my career been associated with the creation of a number of highly productive Intellectual Properties at the University of Kentucky, including WTT ELISA tests which company is now part of the Lexington division of Neogen Corp. I have also been awarded the United States Use Patent that underlies the treatment of Equine Protozoal Myelitis [EPM] with Marquis® and related agents [US Patent # 5,883,095 for treatment of EPM, awarded, licensed, Marquis®, August 2001], and more recently, a United States use patent for guanabenz in the horse and other domestic animals [US Patent # 7,074,843, equine tranquilizer awarded, 2006]. Details of my research training and background and publications and intellectual properties are set forth in summary form in the attached curriculum vitae [Appendix #1] or can be found at wwww.ThomasTobin.com

Thomas Tobin *Veterinary Surgeon*

Wednesday, June 20, 2018, Noon

The Honorable Bob Latta, Chairman
Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection
United States House of Representatives
2125 Rayburn House Office Building
Washington, D.C. 20515

The Honorable Jan Schakowsky, Ranking Member
Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection
United States House of Representatives
2322A Rayburn House Office Building
Washington, D.C. 20515

Re: Lasix PROTECTS the Lungs of the Racing Horse and in so doing PROTECTS the Horse and also the Jockey: An Analysis and Opinions.

Dear Chairman Latta and Ranking Member Schakowsky,

Esteemed Members of the House Energy and Commerce Committee:

First, greetings from Kentucky and let me thank you for the opportunity to submit this important information for the record to the Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection for consideration at your June 22, 2018 hearing on H.R.2651, the Horseracing Integrity Act of 2017. This letter and attachments are to make available my current best analysis and opinions concerning the reasons for the approaching 50 years or so approval of Lasix in New World racing. My analysis and opinions in this matter are based on my professional training and experience and my knowledge of the relevant scientific literature, equine pharmacology and the detection, pharmacokinetics, pharmacodynamics and chemistry of the substance in question, Lasix in the horse. The details of my professional training and experience are set forth in Appendix #1, my professional curriculum vitae.

By training I am a veterinarian, a pharmacologist and a toxicologist and I have been performing, publishing and evaluating research on Lasix in horses since 1975 (Attachment #1 Gabel et al, 1977). I draw attention to this long since published paper

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because the basic horsemen's opinions and veterinary practitioner evaluations presented in this now long ago 1977 paper have, over the years, been supported by evolving science and published scientific research, as I will be pleased to point out as I present this analysis and opinions.

The story of Lasix and racing horses is a 50 year story of field, i.e., horsemen's and veterinary practitioner evaluations and assessments of the beneficial effects of Lasix in the racing horse. These field evaluations of the beneficial effects of Lasix are set forth in the Gabel 1977 paper and now, forty years later, in 2018, forty years of science has basically confirmed and explained these beneficial effects as our understanding of EIPH, Exercise Induced Pulmonary Hemorrhage has developed. We note that the term Exercise Induced Pulmonary Hemorrhage did not even exist in 1977; as such, the Lasix and EIPH story is a classic story of half a century of scientific research confirming and explaining long standing and well established field observations and clinical experience.

The take home message of this analysis and opinions is that Lasix PROTECTS the lungs of the racing horse, and in so doing PROTECTS the horse and also the jockey. I will begin my presentation with a bullet point summary, I will then present specific sections with literature references supporting each bullet point and then close with a restatement of my bullet points and the take home message that Lasix PROTECTS the lungs of the racing horse and in so doing PROTECTS the horse and also the jockey.

BULLET POINT SUMMARY:

- The lung is by necessity a delicate structure, because it must facilitate the most rapid possible transfer of inhaled oxygen into the circulating blood.

- During racing ALL horses bleed into their lungs. Significant hemorrhage slows the horse and can cause acute death. This syndrome has been named Exercise Induced Pulmonary Hemorrhage, EIPH.

- Historically, American horsemen were long aware that water withholding pre-race PROTECTED against EIPH and its many adverse effects.

- In the sixties Lasix was found to enhance the water withholding PROTECTIVE effects, and Lasix began to be approved in American racing.

- Concerns about Lasix and drug testing have been fully addressed by the industry.

- Lasix is scientifically proven to PROTECT the lung against EIPH. Horses on Lasix bleed less into their lungs and run closer to their true potential. They are

less likely to suffer sudden death on the racetrack, so Lasix PROTECTS the health, welfare and lives of horses and jockeys.

- Lasix has been approved throughout North America and much of the New World. Use of Lasix in horses is fully equivalent to the Therapeutic Use Exemptions (TUEs) widely used in human athletics.

I will now present the specific scientific data and research publications supporting the above seven bullet points, as follows:

1/ THE LUNG IS BY NECESSITY A DELICATE TISSUE PRONE TO STRESS FAILURE OF PULMONARY CAPILLARIES:

Lung capillaries must be strong enough to not rupture under the stress of racing, but delicate enough to allow the rapid transfer of oxygen to the red blood cells. Under the stress of racing some incidence of stress failure (rupture) of pulmonary capillaries is inevitable, as described by West el al, 1993, in their paper entitled "***Stress failure of pulmonary capillaries*** *in racehorses with* ***Exercise-Induced Pulmonary Hemorrhage***", Attachment #2 [Our caps, bolding and underlining].

2/ EXERCISE INDUCED PULMONARY HEMORRHAGE, EIPH:

Historically, about 1 % of Horses bleed from their nostrils post-race, called **Epistaxis,** known for at least 300 years. If you endoscope a horse post-race, about 75% of horses show blood in the trachea. These endoscopic findings were first reported by Pascoe et al in 1981, leading to the scientific name, **Exercise-Induced Pulmonary Hemorrhage, EIPH,** Attachment #3.

EIPH is defined as bleeding into the lungs associated with exercise; at some level it occurs in 100% of racing horses. If you do a **Tracheal Wash**, all horses in training show evidence of bleeding into the lungs. The pulmonary damage is CUMULATIVE, and EIPH is equivalent to a production disease in racing horses.

Significant bleeding into the lungs interferes with blood oxygenation (Sanchez et al 2005), slows the horse and is therefore associated with poor racing performance. The bleeding may, at times, be severe enough to cause death, either acutely on the racetrack or soon thereafter, as we will present later.

3/ WATER WITHHOLDING AND EIPH:

American Horsemen long knew that horses run better and bleed less if water was withheld before racing. A four hour water withholding produces a 12 lb or so weight loss, historically a standard pre-race procedure. This long standing field observation of American Horsemen is now scientifically validated, as follows:

4/ OPTIMIZING WATER "WITHHOLDING": LASIX:

When injectable Lasix became available in the late sixties horsemen found that horses bled less and ran better when administered Lasix. Administration of Lasix can increase the water withholding effect to up to about a 28 pound weight loss, Attachment #4.

5/ LASIX AND URINE TESTING: THE FOUR HOUR LASIX RULE:

Questions arose concerning the effects of Lasix on urinary detection of other substances. The urinary dilution effect was first shown to be transient, giving rise to the four hour 250 mg IV / rule, which rule led to a need for detention barns, Attachment #5.

6/ THE LASIX PLASMA THRESHOLD AND URINARY SPECIFIC GRAVITY TEST:

In the early eighties I was asked by the Kentucky Horsemen's Benevolent and Protective Association to identify a blood level of Lasix that would be equivalent to the four hour rule. We ran a 47 horse study and showed that 1 in 1,000 horses would be expected to exceed 30 ng/ml of Lasix in plasma at 4 hours post 250 mg of Lasix IV (Chay et al 1983, Attachment #6.). This threshold was first introduced in Oklahoma in about 1987, where they set the plasma cut-off at 60 ng/ml. This cut-off was later adjusted upward to 100 ng/ml and, with an added 1.010 urinary specific gravity screen, pioneered by Dr. Richard A. Sams at Ohio State, became the national rule.

Lasix is strictly regulated, now often administered by a third party veterinarian at 4 hours prior to post by rapid IV injection into the jugular vein. Under current rules and modern technologies, including the urinary specific gravity test introduced by Dr. Sams, Lasix does not, to my knowledge, in any way, interfere with drug testing, as I understand was presented by Dr. Richard A. Sams at the June 13-14, 2011, New York Racing Association (NYRA) first International Summit on Race Day Medication at Belmont Park in Elmont, N.Y., Attachment #6.

7/ LASIX PROTECTS AGAINST EPISTAXIS, FRANK BLEEDING FROM THE NOSTRILS:

In 1995 Lasix was approved in New York racing. New York maintained records on the incidence of Epistaxis, frank bleeding from the nostrils post race, and these records show the efficacy of pre-race Lasix in reducing Epistaxis. The New York approval of Lasix reduced the incidence of Epistaxis close to 80%, as shown in Attachment #6. This interpretation was supported by Dr. Anthony Verderosa, the New York Racing Association Chief Examining Veterinarian, who reported a *" >400% decrease"* in the incidence of Epistaxis following the introduction of Lasix. This was the first formal validation of the by then longstanding field observations and experience of American Horsemen with regard to Lasix and Epistaxis, Attachment #7.

8/ LASIX ALSO PROTECTS AGAINST TRACHEAL EIPH:

A definitive Hinchcliff et al 2009 study performed in South Africa on 167 or so horses showed that pretreatment with Lasix reduced the incidence and severity of tracheal EIPH, again validating the long standing field experience of American Horsemen, Attachment #8 Additionally, a more recent 2015 consensus study authored on Lasix and EIPH authored by Hinchcliff and his colleagues concluded there was *"moderate to high quality evidence that EIPH is progressive . . . ; that it adversely affects racing performance; that severe EIPH is associated with a shorter career duration; [and], that furosemide is efficacious in decreasing the incidence and severity of EIPH.* Attachment #8.

9/ LASIX OPTIMIZES RACING PERFORMANCE:

When a horse bleeds significantly into its lungs it cannot fully oxygenate its blood and its racing performance suffers. Horses showing significant blood in the trachea post-race perform more poorly than horses with minimal or no blood in the trachea. The so called performance "improvement" associated with Lasix is therefore actually more likely a PROTECTION against an adverse effect of EIPH, again consistent with the long standing field experience of American Horsemen, Attachment #9.

10/ LASIX PROTECTS THE LIVES OF HORSES AND JOCKEYS:

About once in every 1,500 races the bleeding into the lungs is sufficient to acutely asphyxiate the running horse. The horse crashes to the ground, with all attendant risks for horse and rider. In 1983 Gunson et al reported nine such cases in Pennsylvania racing; Gunson specifically noted that a complete necropsy was required to identify these cases, since death can occur with no overt evidence of pulmonary hemorrhage at the nostrils. We evaluated the first such case reported in Kentucky and wrote the case up, specifically including acute sudden death among the clinical presentations of EIPH (Harkins et al, 1997). Since then sudden death from EIPH has been identified as the principal non- musculoskeletal injury related cause of death in racing horses, Attachment #10

11/ CLOSING BULLET POINT SUMMARY:

1.1/ All horses RACING bleed into their lungs. Significant bleeding interferes with racing performance and can cause acute death. This condition is now known as Exercise Induced Pulmonary Hemorrhage, EIPH.

1.2/ Historically, American horsemen knew that water withholding before racing PROTECTED against Epistaxis and various adverse effects of EIPH.

1.3/ Starting in about 1969 Lasix was seen to enhance the PROTECTIVE effects of water withholding, and its PROTECTIVE use began to be approved in American racing.

1.4/ Concerns about possible effects on drug testing were addressed by the industry.

5/ Lasix is scientifically proven to PROTECT horses from EIPH. Horses on Lasix bleed less into their lungs and therefore run closer to their true potential. They are less likely to suffer sudden death on the racetrack, Lasix thereby PROTECTS the health, welfare and lives of both horse and rider.

6/ The scientifically regulated use of Lasix has been approved throughout North America and much of the New World. Such approved use of Lasix in racing is entirely equivalent to the Therapeutic Use Exemptions used in human athletics.

I now close this analysis and opinion with a restatement of my basic take home message, namely that administration of Lasix PROTECTS the lungs of the racing horse, and in so doing protects the horse and also the jockey.

This analysis and opinions is based on the information available to me at the time of writing; if there is anything about this analysis and opinions that is unclear or could be better expressed, or if I can be of any further assistance to you, please do not hesitate to contact me; in the meantime, I remain,

Yours sincerely,

Thomas Tobin,
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Diplomate, The American Board of Toxicology 1980-15.
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Statement by
William M. Thomason, Jr.
President and Chief Executive Officer, Keeneland Association, Inc.
House Energy & Commerce Subcommittee on Digital Commerce and Consumer Protection
HR 2651 Horseracing Integrity Act
Friday, June 22, 2018

The Horseracing Integrity Act of 2018, HR 2651, has the full support of Keeneland Association. Keeneland was established in 1935 in the heart of America's most significant Thoroughbred breeding region, and today owns and operates one of the country's iconic racetracks and the world's largest Thoroughbred auction house.

From the beginning, Keeneland's mission was "to create a model race track to perpetuate and improve the sport and to provide a course that is intended to serve as a symbol of the fine traditions in Thoroughbred racing." Inextricably bound to that mission throughout our history have been unwavering commitments to the health and safety of our equine and human athletes and to the protection and enhancement of the American Thoroughbred such that it remains respected and in demand around the world.

True to that mission and those commitments, Keeneland has always presented outstanding Thoroughbred racing. Horses that have run at Keeneland over the years include household names like Bull Lea, Tim Tam, Riva Ridge, John Henry, Holy Bull, Northern Dancer, Forward Pass, Forego, Unbridled, Round Table, Arts and Letters, Honest Pleasure, Alydar, Rachael Alexandra and Spectacular Bid. Twenty-three horses that ran in Keeneland's premier Kentucky Derby prep race, the Bluegrass Stakes, have gone on to win the Kentucky Derby; 13 won the Preakness; 14 won the Belmont; 9 of those horses won two legs of the Triple Crown; and one, Whirlaway, won the Triple Crown. American Pharaoh completed the first ever "Grand Slam" in American Thoroughbred racing by topping off his Triple Crown victories by winning the Breeders' Cup Classic at Keeneland on October 31, 2015.

At the same time, Keeneland has become the world leader in Thoroughbred auction sales. In 2017, Keeneland sold 5,940 Thoroughbreds for a total of approximately $540 million. The buyers came not just from the United States, but also from more than 45 countries from around the world, thus representing an enormous investment of foreign dollars into our American equine products. Horses sold at auction at Keeneland have gone on to achieve enormous success at the racetrack and in the breeding shed. Twenty-two future Kentucky Derby winners, 23 Preakness winners, 20 Belmont Stakes winners and more than 100 Breeders Cup race winners, including 11 winners of the Breeders' Cup Classic, were sold at Keeneland auctions. In 2016, all three winners of the Triple Crown races, Nyquist, Creator, and Exaggerator, had been sold at Keeneland auctions, and later that year at the Keeneland September Yearling Sale, a horse named Justify was sold for $500,000. As we now know, he would go on to become the 13th winner of the Triple Crown, completing that feat earlier this month.

HR 2651 addresses issues critical to the success and growth of racing, breeding, and sales in our great sport. By combining the world's premier medication regulation entity (USADA) with the knowledge and experience of key racing constituencies, the independent authority created by

this legislation will provide both uniformity and appropriate rigor to better ensure clean competition in American racing.

The beneficiaries will include our fans who can enjoy races with confidence they are free from improper influences, participants who can compete knowing they are on an even playing field, the beautiful animals that were "born to run" and deserve to do so without the burden of anything in their systems that could harm them, and the jockeys who can ride the races with confidence their horses are sound and ready to compete.

We especially commend our own Congressman Andy Barr for his leadership in this important initiative and we urge the passage of HR 2651 at the earliest opportunity.

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THE NEW RACING ASSOCIATION, INC.

June 16, 2018

Chairman Bob Latta
Ranking Member Jan Schakowsky
Committee on Energy & Commerce
Unites States House of Representatives
Washington, D.C. 20515.

We would like to thank the committee for allowing us the opportunity to share our support of H.R. 2651, the Horseracing Integrity Act of 2017.

I, Chris Kay, have been the Chief Executive Officer and President of the New York Racing Association (NYRA) since 2013. I, Mike Del Giudice, have led the Board of Directors since June, 2015. NYRA operates three of largest racetracks in the United States by handle, or money wagered on our races: Aqueduct Racetrack, Saratoga Race Course, and Belmont Park, which is home to the third leg of the Triple Crown, the Belmont Stakes.

Since we joined NYRA, we have seen two horses win the Triple Crown: American Pharoah in 2015 and Justify this month. We are very proud that the Belmont Stakes was later named the Sports Event of the Year by Street & Smith's Sports Business Journal.

Within the racing industry, NYRA is known for offering a top racing product to horsemen and our customers. We believe that a significant component of producing the best racing is ensuring the integrity of the sport and protecting the safety of our human and equine athletes. This is not possible without an effective anti-doping system in place. In New York, we have adopted the four components of the National Uniform Medication Program (NUMP) and are known as a leader in safety and welfare initiatives. However, not all racing jurisdictions have adopted every part of the NUMP.

In 2016, NYRA's board voted to support the previous version of H.R. 2651, the Thoroughbred Horse Racing Integrity Act of 2015. Two years ago, we stated our belief that uniform policies nationwide could only help to further the credibility of the sport. Given the insufficient progress made toward uniformity among the 38 racing jurisdictions since NYRA voiced its support for federal legislation. We feel stronger than ever that we need one set of medication rules and enforcement in every jurisdiction in the United States.

With that in mind, we fully support the oversight that would be provided from the passage of the Horseracing Integrity Act of 2017. This bill would enable the Horseracing Anti-Doping and Medication Control Authority (HADA), an independent, private, and non-governmental agency, to ensure that all jurisdictions are on a level playing field and adhere to many of the same regulations already in place at NYRA's tracks.

Nearly two weeks ago, horse racing took center stage of the sports world when Justify won the Belmont Stakes and became the sport's 13th Triple Crown winner. For horse racing to continue to build on the popularity of two Triple Crown winners in four years and gain new lifelong fans, we as an industry must be sure that we are showcasing clean competition backed by a system of strong testing and deterrent measures.

The best way to achieve that goal is by passing the Horseracing Integrity Act of 2017.

Michael J. Del Giudice
Chairman

Christopher K. Kay
Chief Executive Officer and President

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U S T A

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June 19, 2018

The Honorable Bob Latta
Chairman
Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection
United States House of Representatives
2125 Rayburn House Office Building
Washington, D.C. 20515

The Honorable Jan Schakowsky
Ranking Member
Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection
United States House of Representatives
2322A Rayburn House Office Building
Washington, D.C. 20515

Dear Chairman Latta and Ranking Member Schakowsky:

On behalf of the United States Trotting Association (USTA) and our members, I'd like to thank you for the opportunity to submit this important information for the record to the Energy and Commerce Subcommittee on Digital Commerce and Consumer Protection for consideration at your June 22, 2018 hearing on H.R.2651, the Horseracing Integrity Act of 2017.

Since being founded in 1939, the USTA's mission has been to ensure the highest standards of care for Standardbred horses, to support and develop nationwide rules, and to promote the harness racing industry to help ensure economic security. With nearly 16,000 members, the USTA serves a broad constituency including horsemen, racetracks, regulators, media, and racing fans.

The USTA strongly supports breed-specific, consistent medication rules for horse racing but strongly opposes H.R.2651 for these key reasons:

- **A uniform system already exists, and it works well.** The existing system is governed by the Association of Racing Commissioners International (ARCI). If enacted, H.R. 2651 would be significantly burdensome to the horseracing industry because it would create a new, duplicative regulatory bureaucracy at the federal level, on top of an existing state regulatory structure that is operated in 34 state racing jurisdictions around the country. It is a system that has functioned well for over 100 years.

Currently, 99.51% of all post-race tests are clear of any violations. The majority of the scant violations that do occur are for minor therapeutic medication overages. The laboratories are catching violators. A 99.51% clear rate confirms that there is not a "doping" problem in the industry. Hence, the USTA does not believe that this legislation is needed.

H.R. 2651 would create a board whose total makeup has no vested interest in the industry. The bill proposes an independent, self-regulatory, nonprofit corporation to be created, known as the Horseracing Anti-Doping and Medication Control Authority (HAMCA). HAMCA would be charged with developing and implementing a medication control program for Thoroughbreds, Standardbreds, and Quarter Horses. Even though the Department of Agriculture has been charged with protecting animals for over 50 years, HAMCA would instead operate under the ultimate authority of the Federal Trade Commission (FTC), whose mission is to promote competition and protect consumers—not to protect horses. The FTC is the wrong fit for HAMCA.

Furthermore, the HAMCA board would be composed as follows: the chief operating officer of the United States Anti-Doping Agency (USADA), six USADA members, and six members that USADA would appoint. It is possible that the entire harness racing industry may be voiceless on the board. It is imperative that those who know the industry best have a seat at the table.

- **The bill lacks separate regulations governing the use of therapeutic medications for different horse breeds**. H.R. 2651 disregards the differences among racing models that exist for different breeds, particularly the frequency of racing.

  Jockey Club statistics indicate that the average number of races for a thoroughbred in 2017 was 6.21, while USTA statistics show that the average number of starts for all Standardbreds in North America was 18.2 in 2017. For Standardbred pacers, who made up 68.6% of all races last year, the average was 19.4 starts. Clearly, the number of races for a Thoroughbred and a Standardbred can be significantly different; therefore, a one-size-fits-all approach to therapeutic medications is wrong. Experts will tell you that therapeutic medications should instead be tailored to the racing regimen of each breed. That is why state racing commissions and the ARCI recognize the importance of these differences in regulating racing medication.

- **The proposed legislation places an economic burden on small businesses in the industry.** H.R. 2651 threatens the economic well-being of the industry by imposing increased costs for testing that will drive many small business horsemen out of the industry. A cynical view of the legislation would even say that decreased competition is an intended consequence of the bill, which is being pushed largely by those with the resources to thrive within its complexity. Under the legislation, HAMCA will be given unrestricted authority to set its own budget and to impose unlimited taxes and fees on the industry. As with all unrestrained bureaucracies, it will, no doubt, justify its own existence and pay for its unchecked growth by imposing ever-increasing taxes and fees on those it regulates. Historically, Congress has appropriated federal anti-doping funds and granted them to USADA to help cover the cost of drug testing in human sports, which is USADA's sole area of expertise.

H.R.2651 contains no provision for federal funding for HAMCA. Instead, it imposes assessments on state racing commissions as its basic funding mechanism.

HAMCA can also borrow money, and the bill states that assessments on state commissions can be used to liquidate loans. Yet HAMCA gets to determine its own budget, giving it a blank check to impose unlimited new costs on the many small businesses that participate in Standardbred racing.

It is only reasonable to project that the operating costs that HAMCA would impose on racing would be substantial. For example, in calendar year 2016, state racing commission expenditures in Pennsylvania alone exceeded $16 million. If more money is to be spent, instead of supporting a redundant, inexperienced, and uncontrolled new federal bureaucracy, Congress should consider supporting existing medication research as well as other efforts that are being made to improve the sport.

- **The bill's prohibition on the use of the therapeutic medication Lasix has no scientific basis.** H.R. 2651 fails to recognize that the health and well-being of equine athletes is of paramount importance. The use of Lasix (furosemide) has been a 30-year equine welfare policy permitted on race day as a legal, therapeutic medication to mitigate the effects of Exercise Induced Pulmonary Hemorrhage (EIPH) or "bleeding" in the lungs of horses. EIPH occurs in a variety of circumstances and frequently during intense exercise, such as races. All scientific studies and reasoning show that Lasix has no negative impact on the horse. Its use is endorsed by the veterinarians who comprise the industry's gold standard in equine health and welfare, the American Association of Equine Practitioners (AAEP). As the AAEP has determined, Lasix "is in the best interests of the health and welfare of the horse."

  AAEP opposes a Lasix ban, in part, because there is no evidence that Lasix either enhances performance or masks substances that do so. Using the only medication proven to alleviate EIPH is simply the humane way to race horses. Critics of Lasix have suggested that the use of diuretics may mask the presence of other illegal substances by making it more difficult to identify drugs in diluted urine. However, research has clearly shown that the diluted urine effect has abated after 2 ½ hours. For this reason, Lasix is administered in horse racing 3 - 4 hours before competition, thus solving any potential masking problem. In any event, in the decades since the "masking" objection was formulated, scientific testing has become so sensitive that fractions of a picogram (10^{-12} gram) can still be detected.

  Lastly, it is important to note that the use of Lasix is not a mandatory practice; its use is a decision by the trainer after consultation with the horse's owner and veterinarian.

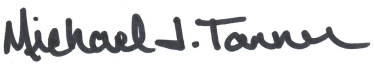
In conclusion, it is of paramount importance to recognize that the racing industry is unified in opposition to H.R. 2651. In addition to the USTA, every organization legally representing horsemen and horsewomen across the country, together with the principal organizations representing the equine veterinary community and the national organization representing independent state racing commissions, all stand in strong and unified opposition. The industry-wide list of opponents includes the following:

- American Quarter Horse Association (AQHA)
- Association of Racing Commissioners International (ARCI)
- California Thoroughbred Trainers Association (CTT)
- Harness Horsemen International and its affiliates
- National Horsemen's Benevolent and Protective Association (NHBPA) and its affiliates
- Thoroughbred Horsemen's Association and its affiliates
- Thoroughbred Owners of California (TOC)
- American Association of Equine Practitioners (AAEP)
- National American Association of Racetrack Veterinarians (NAARV)

According to a recent American Horse Council study, the horse racing industry contributes $36.1 billion annually to the national economy and provides 240,000 direct jobs. Rather than burden this industry with unnecessary costs and regulations, the United States Trotting Association urges Congress to protect us from unneeded and excessive regulation, so we can do our part to grow the state and local economies that make this nation flourish.

Sincerely,

THE UNITED STATES TROTTING ASSOCIATION



Michael J. Tanner
Executive Vice President and CEO

WEST POINT
THOROUGHBREDS

June 21, 2018

Chairman Bob Latta
Ranking Member Jan Schakowsky
Committee on Energy & Commerce
United States House of Representatives
Washington, D.C. 20515

Dear Chairman Latta and Ranking Member Schakowsky:

I would like to thank the committee for allowing me the opportunity to share my support of H.R. 2651, the Horseracing Integrity Act of 2017. My name is Terry Finley, and I'm CEO of West Point Thoroughbreds, the largest Thoroughbred racehorse partnership in the country. Our company owned part of last year's Kentucky Derby winner, Always Dreaming.

I am also a board member of the New York Thoroughbred Horsemen's Association (NYTHA). NYTHA represents the owners and trainers of New York Thoroughbred racing. New York leads the nation in betting handle, purses offered, and graded stakes run. It is truly the epicenter of North American racing. New York also is leading on the issue of doping reform for racing. The New York Racing Association has strongly endorsed the bill, and 26 out of 27 Members of the New York Delegation have co-sponsored HR 2651.

In both capacities as an owner and NYTHA Board Member, I strongly support the Horseracing Integrity Act. I have been a racing fan since childhood, and as it has become my livelihood. I am very concerned about the role of medications in our business. Integrity in any sport is a crucial pillar of success, but in a sport that relies on the wagering dollar, integrity is the very foundation of our existence.

People in the industry have worked hard over the past decades to implement the National Uniform Medication Program (NUMP), but because of our fractured regulatory system, few of the 38 state racing jurisdictions have embraced it. That is why federal legislation is needed. Horse racing doesn't have the luxury of a Commissioner of Racing who can dictate policies that are good for the game. Instead, we have separate fiefdoms that fight like crazy for their piece of the pie, with little regard to the overall health of the industry.

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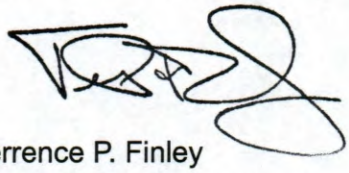
HR 2651 provides a common sense structure to our byzantine anti-doping system. A single, independent, non-governmental, non-conflicted organization will apply uniform rules and penalties. Honest trainers in New York would be able to race in Pennsylvania without hiring a chemist to navigate the different medication rules. Trainers caught cheating in one state would no longer be able to pick up their tack and move their barn to another state. Horse owners and bettors will know they are racing in and wagering on clean races.

HR 2651 will reinvigorate racing.

Racing has many challenges, (all industries do) but integrity shouldn't be one of them. As a member of NYTHA and the proud owner of West Point Thoroughbreds, I strongly urge your Committee to pass HR 2651, the Horseracing Integrity Act.

Respectfully,



Terrence P. Finley

The Water Hay Oats Alliance Membership Roster

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Allen, Donna Versailles KY USA	Allen, Judith Media PA	Allison, A New York NY USA	Allison, Mary Valders WI
Almond, Laura Odenville AL	Altieri, Marion Saratoga Springs NY	Alvarez, Erik Miami FL	Amerman, Jerry Temecula CA USA
Amerman, John Temecula CA USA	Anaya, Joseph Nanuet NY	Ancona, Barbara Colts Neck NJ	Anderson, Sandra Lexington KY
Anderson, Warren Lexington KY	Andreasen, Heather Shakopee MN USA	Andrews, Denny Edmonton Alta Canada	Andros, Kathy Sylvania OH
Ane-Mcgreevy, Cinthia Plantation FL USA	Ann Fogg, Christine Fayetteville GA	Armstrong, Anne Huntersville NC USA	Armstrong, Stewart Kingsville TX USA
Arrington, Susan Springfield VA USA	Arslan, Ridvan loma riac CA	Arthur, Allison Wheeling IL	Assante, Ellen Standish ME
Aten, Joy Hudsonville MI	Athayde Stevens, Angie Sierra Madre CA	Atkins, David Lexington KY	Atkinson, Tommy Scott AZ
Attfield, Roger Nobleton, Ontario Canada	Austin, Penny Nicholasville KY	Ayers, Greg Stewartstown PA	Ayers, Melissa Houston TX
B, Lauren Woodridge IL	Backer, William The Plains VA USA	Bacon, Kathi Corydon IN	Bailis, Carole Gladwyne PA
Baker, Diana Versailles KY	Balding, Ian Kingsclere England	Baldini, Ronald Niantic CT	Ballard, Christine Orangeburg SC
Balzer, John Hampton Bays NY USA	Banahan, Michael Lexington KY USA	Bance, Peter Manakin Sabot VA	Bandler, Barry Boca Raton FL USA
Bandoroff , Holly Lexington KY US	Bandoroff, Conrad Paris KY USA	Bandoroff, Craig Paris KY U.S.	Baptiste , Mo Middleburg VA USA
Barbariantz, Kika Monroe NY USA	Bare, Susan WAshington DC	Bark, Greg New Prague MN USA	Barnes-Euresti, Norma Battle Creek MI
Barnes, John Glen Mills PA	Barry, Rahd Somersby NSW	Barton, Anna Haysi VA	Barton, Frederick Cornelius NC USA
Bartyzel, Chris Amsterdam NY	Bass, Ramona Fort Worth TX	Bass, William Concord NC	Bassett, Taylor Tampa FL USA
Bassett, Ted Midway KY USA	Batsidis, Louis Boca Raton FL US	Baxter, Bethany Frankfort KY	Baygents, Pamela Tucson AZ USA
Bean, Christine Golden Valley AZ	Beavis, Pat Winnipeg Canada	Beck, Antony Lexington KY	Beck, Barb Glendale AZ
Becker, Sandi Glenmoore PA USA	Behrendt, Theresa Saratoga Springs NY USA	Beitz, Gail New York NY	Belden, Jay Bayard NE USA
Bell, David Scottsdale AZ	Bellhouse, Tom Saratoga Springs NY USA	Belling, Mark Milwaukee WI	Belmonte, Kathleen Lake Forest CA
Ben-Meir, Nick Los Angeles CA	Bengston, Joe Sheffield Lake OH USA	Benjamin, Ari New York NY	Benjamin, Chris Phoenix AZ
Benzel, Seth Saratoga Springs NY USA	Berg, Kathleen Stony Plain, Alberta Canada	Bergeron, Nancy Roseburg OR	Berkey, Kathleen Etowah NC USA
Berlin, Celeste Girard PA	Berrios, Manuel Tinton Falls NJ USA	Berry , Jessica Louisville KY USA	Berry, Emma Newmarket, Suffolk UK
Berry, Pamela Odessa FL USA	Berry, Paul Asheville NC	Besaw, Tom Oakdale MN USA	Besenfelder, David Lafayette CA
Beshear, Jane Lexington KY USA	Beshear, Steven Lexington KY USA	Bethel, Bob Gulf Shores AL	Bey, Terri Lexington KY USA

Bialecki, Ed Long Valley NJ USA	Bibb, Renee Burbank CA	Bibby, Candace Indian Wells CA USA	Birsh, Philip New York NY USA
Biszantz, Gary Lexington KY	Bittner, Karl North Syracuse NY USA	Blacker, Dan Monrovia CA USA	Blaho, Karen Williston FL
Blair, Kevin New Braunfels TX	Blair, Proctor Lexington KY USA	Blake, Janice Elmont NY	Blatz-Murff, Pamela Lexington KY USA
Bleakley, James Round Hill VA	Blethen, Valerie Pleasureville KY	Blowen, Michael Georgetown KY	Bloxsom, Marsha Lexington KY
Bobo, Tami Ocala FL	Bobrow, Molly Houston TX	Bodarky, Steven Hazlet NJ USA	Bogdan, Maryanne Sterling Heights MI
Bohrer, Travis Strasburg VA USA	Bole, William Rockford IL	Bolgos, Sally Ann Arbor MI	Bonifaz, Miguel Lima Perú
Bonneau, Sally Indianapolis IN	Bonnie, Cornelia (Nina) Prospect KY	Bonnie, Edward S. Louisville KY	Booker, Hildric Phoenix AZ USA
Booth, Fred and Joan Amity OR	Border, Shanna Humboldt SD	Bouchard, Johanna Waterville ME	Bouchard, Ray Winnipeg Canada
Boultinghouse, Denny Arcadia CA	Bourg, Michelle Lawrenceville GA	Bourne, Bill Dunkirk MD US	Bourque, Rhoda Troy ME
Bowen-Rees, Julian Richmond VA USA	Bowen, Ruthie Versailles KY USA	Bowman, Patricia Lexington KY USA	Boyle, Mike Winchester VA
Bozsum, Mike Surfside Beach SC	Bradley, Charles Delmar NY	Bradley, Paul Lake George NY	Bradley, Ryan San Francisco CA
Bradley, Thomas Woodhaven NY	Brady Cutler, Kim S. Hamilton MA USA	Brady, James Far Hills NJ USA	Brady, Joan Far Hills NJ USA
Brady, Nicholas F. Oldwick NJ USA	Bramlett, Florence Sedro Woollley WA USA	Brass, AJ Houston TX	Braswell, Julie Reddick FL
Bray, Michael Saratoga Springs NY USA	Breathnach, Cormac Lexington KY	Breault, Kathleen Saratoga Springs NY USA	Brendemuehl, Danzel Ocala FL
Breniser, Darryl Denver PA USA	Brewer-Galpin, Jayne Lexington KY	Brewer, Karin San Antonio TX	Brida, Dennis Ballston Spa NY
Brida, Juliane Ballston Spa NY	Briggs, Debbie Rhinebeck NY	Brio, Luis Orange Park FL	Broad, Gary Palm Desert CA USA
Brody, Marlene Ghent NY USA	Brogden, Carrie Lexington KY	Brogden, Craig Lexington KY USA	Broker, John Shakopee MN USA
Bromagen, William Fort Lauderdale FL	Bromley, Jane Naples FL USA	Brooks, Lois Fallston MD	Brothers, Donna Louisville KY USA
Brotherton, Dorothy Richmond VA	Brouillette, Ron Encinitas CA	Broussard, Linda Austin TX	Brown, Alex Lincoln University PA USA
Brown, Cynthia Warner Robins GA USA	Brown, Dennis Hemet CA USA	Brown, Donald Stuart FL USA	Brown, Douglas Keymar MD
Brown, Frank Charleston SC	Brown, J Darrell Lexington KY USA	Brown, James Shakopee MN USA	Brown, John P RR 3 Granton, Ontario Canada
Brown, Lendy Lexington KY USA	Brown, Lynn K Weatherford TX United States	Brown, Michael Lexington KY	Bruels, Mary Gulfport FL USA
Brunet, Norm Nepean Canada	Bryner, Sheryl Monkton MD USA	Buckley, Kathy Charlotte NC	Budge, Christienne Saratoga Springs NY
Buedel, Christina Middle River MD	Bull, Joan Roswell NM	Buller, Lucinda Danville IN	Burke, Julie Lexington KY
Burns, Michael Danville KY	Burton, Darla Nolensville TN USA	Bush, Bing Del Mar CA	Buss, Michael Blackwood NJ
Bussan, Jeff New Canaan CT	Butera, Julie New Orleans LA	Butzow, Barry Eden Prairie MN USA	Buxton, Anne Morriston FL
Buzzell, Gary San Ramon CA USS	Byington, Robert Grand Ledge MI	Byrne, Kathy Chicago IL United States	Byrne, Rob Folsom LA
Byrne, Warren Toronto, Ontario Canada	Caine, Cynthia Munster IN	Calabrese , Toni-lynne Spring Lake NJ USA	Calabrese, Lexi Lexington KY USA
Callahan, Teresa Branchburg NJ	Calmes, Melissa Lexington KY USA	Cambareri, Serafino Mount Kisco NY USA	Campbell, Cot Aiken SC
Campo, Richard Chattanooga TN	Canfield, Bill St. Louis MO	Cantey, Charlsie Okatie SC USA	Canzone, Peter NY
Canzoneri, Dina Childs MD	Capria, Levana Versailles KY USA	Capshaw, Amy Santa Fe NM	Carlisle-Nicholas, John Pleasant Valley NY
Carr-mora, Chellie Fulshear TX United States	Carrano, Lisa Tarpon Springs FL	Carriker, Ashley Tinton Falls NJ USA	Carroll, Christine Gainesville GA

Carroll, Dan Madison CT	Carroll, Janis Madison CT	Carroll, Rick Foxboro MA	Carter D.V.M., Lisa Charlotte MI
Cary, Andrew Lexington KY	Casner, Bill Flower Mound TX	Cassimeris, Lynne Bethlehem PA	Castellano, Mark Far Hills NJ
Caulier- Eimbcke, Beatrice Cynthiana KY USA	Caupp, Denise Greenville OH USA	Cauthen, Doug Lexington KY	Cauthen, Katie Lexington KY USA
Cauthen, Steve Verona KY USA	Cavanagh, Mike Aurora CO	Cecil, Ben Pasadena CA USA	Cerulli, Diane Rhinebeck NY
Chakrabarti, Sumon K Noida, Uttar Pradesh India	Chalfant, Mary North Versailles PA	Chamblin, Debbie Lexington KY USA	Chamblin, Tony Lexington KY
Charest, Roberta The Villages FL	Charles, Betty St. Paul MN USA	Charles, Teresa St. Paul MN USA	Charlston, Mary Ann Hobe Sound FL USA
Chavez, Jose Ocala FL USA	Cheechalk, Colleen Vestal NY	Cheever, Holly Voorheesville NY	Cheng, Paul Plano TX
Childress, Gerardette Park Forest IL	Choi, Irene Denver CO United States	Cholity, Chris Hawthorn Woods IL	Church, Chrissy Crofton MD
Ciampi, Joan Lexington KY	Cimino, Cathy Lynn Saratoga Springs NY USA	Cisna, Craig Lew London WI	Clapton, Alison Lethbridge Canada
Clark, Carrie Lexington KY	Clark, Daryl Plantation FL	Clark, Marsha Mialn IN	Clark, Renee Eolia MO
Clark, Stacie Cedar Valley, Ontario Canada	Clark, Susan Radnor OH	Clarke, James Tyler Lexington KY USA	Clarke, Matthew Chepachet RI United States
Clarke, Steven Harbinger NC USA	Clay, Blythe Versailles KY USA	Clay, Brutus Paris KY USA	Clay, Joseph Paris KY
Clay, Robert Versailles KY USA	Cleary, Leah Moorestown NJ	Clemmens, Nelson Goshen KY USA	Click, Jim Tucson AZ USA
Clott, Sheri Cypress Inn TN USA	Coates, Lou Ocala FL	Coffey, Marialice Macedon NY	Cohen, Darian Oak Park CA
Cohen, Sandra Saratoga Springs NY	Cola, Rosemarie Colts Neck NJ USA	Colebrook, Ben Lexington KY USA	Collazo , Henry Hialeah FL Dade
Colletti, Jerry River Ridge LA	Collier, Kathi Little Torch Key FL	Collingsworth, Amy Lawrenceburg KY USA	Collins, Lincoln Midway KY
Colp, Linda Chesapeake VA	Columbo, Eugenio Boynton Beach FL USA	Combs, Dave Northbrook IL USA	Comfort, Sunny Harrison NY
Cone, Marcia Lexington KY	Conley, Bill Owensboro KY	Contreras, Danielle Anaheim Hills CA	Cook, Lawrence Lanoka Harbor NJ
Cook, Robert Chestertown MD	Cook, Susan Pilot Point TX	Coomber, Vickie Louisville KY USA	Coonan, Brian Ireland
Cooper, Alan London England	Cooper, Patrick Ireland	Copeland, Maryann W. Hollywood CA USA	Copeland, Robert Holton IN
Corbett, Brittany Lexington KY	Corcoran, Jo-Claire Henryville IN	Corcoran, Kathy Cleveland OH USA	Corey, Anthony Boston MA
Corey, Kristen Bethany OK USA	Cornell, Penny Claremore OK USA	Cornett, Kip Lexington KY	Correas IV, Ignacio Lexington KY USA
Couch, Sally Ann Castleblayney, Monaghan Ireland	Coughlin, Joe Grosse Pointe Park MI	Coulter, Karen Barton NY	Counides, Mark Payette ID United States
Courtney, Brenda Lexington KY USA	Courtney, Otis Eden Prairie MN United States	Courtney, Robert Lexington KY USA	Courtney, Stephanie Eden Prairie MN USA
Coutu, Ruth Barre MA	Cowan, Dr. Lyn Eagan MN USA	Cowan, Margaret Galena MD	Cox, Jennie Bedford NY USA
Cox, Virginia Simsbury CT	Cozac, Heller Richmond TX	Creskoff, Bob Ardmore PA	Crismani, Cate Newhall CA USA
Crissey, Alden Dallas TX USA	Cross, Richard Fishtail MT	Crute, Sharon Saratoga Springs NY USA	Cullinan, Barbara North Bergen NJ
Cumming, Alyssa V Versailles KY USA	Cundy, Rod Calgary, Alberta Canada	Cunningham, John Medford MA USA	Cupak, Kathryn Franklin Lakes NJ
Cupingood, Len Norristown PA	Curran, Bob West Islip NY USA	Curran, Pat Valatie NY	Curry, Diane Lexington KY USA
Curtis, Carrie Minneapolis MN	Curtis, Ward St. Petersburg FL	D'Amato, John Boca Raton FL	Dabu, Rick Bloomington MN USA
Dagostino, Nicholas North Las Vegas NV USA	Dailey, Brett Paducah KY USA	Dale, Dennis Grande Prairie, Alberta Canada	Dale, Doug Ridgeway MO
Dallao, Joe Korea	Dalton, Niall Sierra Madre CA USA	Daly, Jacqueline Clearwater FL USA	Daly, Ned Needham MA USA

Damien, Shanna Ocala FL United States	Dane, Keith Gaithersburg MD	Dantico, Ivy Monroe NY	Dartnall, Camilla Marlborough England
Dauterive, Juliette Baton Rouge LA	Davalos, Albert Davis CA	Davenport, Margaret Ballston Spa NY USA	Davenport, Meagan Del Mar CA USA
David, Dr Tom V Metairie LA	Davies, Graham Leesburg VA	Davis, Jody Kansasville WI	Davis, Scott Fort Edward NY USA
Day DVM, William Brenham TX USA	Day, James White Post VA USA	De Francis, Karin Stevenson MD USA	De La Torre III, Jose Hacienda Heights CA USA
De Seroux, Laura Solanbeach CA USA	De Vinney, Erica Landenberg PA	Dean, Marshall Landenberg PA	DeAngelis, Robert Louisville KY
Deeming, Robin Corbett OR USA	Deerinck, Alessandra San Marcos CA USA	Degnan, Sharon Santa Margarita CA USA	Del Gallo, Rinaldo Pittsfield MA USA
Delaforce, Tres Lexington KY	DeLauro, Ginny Amityville NY	Delcastillo, Janet Winter Haven FL	Deline , Jeannine Rochester NY USA
Delozier, Joseph Sparks MD USA	Dempsey, Michael Pensacola FL USA	Dempsey, Susan Belmont MA	DeNardo, Richard Flint MI
Denholtz, Lisa Oceanport NJ USA	Denholtz, Steve Oceanport NJ USA	DeNio, Art El Cerrito CA	Dennis, James Mundelein IL USA
DeRunk, Bill Pittsburgh PA	DeSalvo, Steve Hot Springs AR	DeSmet, Elizabeth Westford MA	DeToro, Alexandra Mequon WI
Devries , Tori Fort Washington MD USA	Dewey, Tamerie Sedro Woolley WA USA	Dickinson, Michael North East MD USA	Dickson, Bill Paris KY
Dickson, Chelsea Lexington KY	Dickson, John Providence RI USA	Diemer, Kathy Nottingham PA	Diers, Patricia Saint Charles IL
Dillon, Dave Hallendale Beach FL	Dillon, Kelsey Minneapolis MN USA	Dilschneider, Adele St Louis MO	DiMaio, Rachel PA
DiMenna, Michael West Chester PA	Disalvo, Maria Whitestone NY	DiVirgilio, Dan Del Mar CA	Dixon, Brittney Lexington KY usa
Dobbs, Janet Voorhees NJ	Doby, Karen Prosper TX	Doneski, Joanne Edgewater MD USA	Donoghue, Daniel Barrington IL
Donovan, Bill Delray Beach FL USA	Donworth, Richie Lexington KY	Dopson, Chris Ontario Canada	Dory, Bill Spruce Grove Canada
Doth, Peter Lexington KY USA	Dougherty, Linda Philadelphia PA	Doyle, Jacqueline Berkshire England	Doyle, Renee Barre MA
Drake, Daniel Media PA USA	Drake, John Pittsburgh PA USA	Drake, Jonathan Sarasota FL	Drew, JoAnn Little Rock AR
Drexinger, Robert Alburtis PA	Drexinger, Sandra Alburtis PA	Driscoll, Noelle Silver Spring MD	Driver, Mitchell Glasgow KY USA
Driver, Monica Flat Rock IN USA	Drysdale, Neil Playa Del Ray CA	Dubuc-Jenkins, Tammy Robertsdale AL USA	Dugan Drysdale, Shawn Playa del Rey CA
Duncan, Greg Charlottesville VA	Dunham, Dr. Christine Crystal MN USA	Dunn, Jane Holly Hill SC	Durand, Emery St Paul MN USA
Durham, Danele Richland Hills TX USA	Dutcher, Michele Boring OR	Dvorkis, Alan Las Vegas NV	Dwyer, Erin Loma Rica CA
Dyer, Karen Nashville TN	Edwards, Dana Manchester KY	Egan, John Richmond VA USA	Egerton, AnnaWebb New York NY
Egerton, Susan Harpers Ferry WV	Elliot, Janet Kirkwood PA USA	Elliott, John Free Union VA USA	Elliott, Sharyn Oshawa, Ontario Canada
Ellis, Angela Ft Worth TX	Ellis, Cassandra Rosemount MN USA	Ellis, Jackie Paris KY USA	Ellis, Tom Rosemount MN USA
Emig, Donna Yardley PA USA	Engel, Melvin Delray Beach FL	Ensom, Jim Orangeville Ontario, Canada	Espinoza, Kristin Sun City AZ USA
Evans, Adiclere Lexington KY USA	Evans, Robert Stamford CT USA	Evans, Sadie Banstead United Kingdom	Ewing, Brad Black Diamond WA
Fagan, Ann Asheville NC	Faine, Lianne Lockeford CA USA	Fantasia, Ava Grafton MA USA	Farish, Bill Versailles KY USA
Farish, Will Versailles KY USA	Farm, Rebah Williston FL USA	Farmer, Abbey Morriston FL	Farmer, Carol Midway KY USA
Farmer, Tracy Midway KY USA	Farrell, Kevin Shakopee MN USA	Farris, Vod Camas WA USA	Farrow, Darlene Rocky Mount NC
Fattorusso, Louis Cedar Knolls NJ USA	Fay, Charles Evanston IL USA	Fay, James Rumford RI	Fay, Stephen Narragansett RI
Feigeles, Scott Long Beach CA	Fenster, Julie M. Manlius NY USA	Ferguson, Sam Ocala FL USA	Fernandez, Rafael Monroe NC
Ferrara, Adele	Ferraro, Greg	Ferraro, Stephen	Figlow, Ralph

Wellington FL	Davis CA	Sierra Madre CA USA	Clarks Summit PA USA
Finley, Debbie Saratoga Springs NY USA	Finley, Terry Saratoga Springs NY USA	Fipke, Chuck Montague, PE Canada	Firestone, Matthew Palm Beach FL USA
Firlotte, Brad Apopka FL USA	Fishbough, Bill Exeter CA USA	Fisher, John R S Coatesville PA	Fitzsimons, Jr., Hugh A Houston TX
Flay, Bobby New York NY	Fleming, Janice Diana TX USA	Fletcher, Sue Paradise Valley AZ	Fluor, Peter Houston TX USA
Fojaco, Victor Wimauma FL	Foley, Tim Chicopee MA USA	Foley, Tom Saint Paul MN USA	Folkerth, Ted Del Mar CA
Ford, Frank Deland FL	Ford, Glenn Deephaven MN USA	Ford, Mark West Midlands United Kingdom	Forrest, Becca Whittier NC USA
Fowler, Louise Minneapolis MN USA	Fox, Jr, Robert La Crescenta CA	Francek, Janet Spokane WA	Frankli N, Dan Prior Lake MN USA
Frassetto, Albert Upper Saddle River NJ	Frassetto, John Upper Saddle River NJ	Freeman Holmes, Katey Loudonville NY USA	Freeman, Bill Tampa FL USA
Freeman, Sharon Vineland NJ	Freeman, Tracey Thousand Oaks CA	French, Catherine Camden SC USA	Freuhling , Rosemary Minnetonka MN USA
Friedman, Lucille Versailles KY	Friel, Ted Ambler PA	Fritz, Jean Lansdale PA	Fritzler, Deb Amherst VA USA
Fujimoto, Kris Wilmington CA	Fulton, John Lake Worth FL	Funke, MIke Shelton CT	Funking, Robert Elmsford NY
Furado, Kathy San Jose CA	Furey, Julie Madison CT	Furey, Margaret Pittsfield MA	Furey, Thomas Marco Island FL
Futch, Haley College Station TX USA	Gagliardo, Nicholas Syosset NY	Gale Mott/Founder, 2nd Chance Equine Paris KY	Galindo , Manuel Anaheim CA USA
Gallagher, Patrick Sierra Madre CA United States	Galsky, Mark Las Vegas NV	Gamboney, Cheryl Arlington VA	Gardner, Frank Midway KY
Gardner, Nina Chatham PA	Garey, Jack Georgetown TX USA	Garofalo, Gregory Saratoga Springs NY	Garratt, David Cape Coral FL
Garten, Annie Middleton WI	Gelbman, Lew Pleasantsville NY	Gelman, Robert Cherry Hill NJ	George, Noelle Grosse Pte Woods MI
Georgiev, Valeri Simi Valley CA	Gerritt, Kevin Jersey City NJ	Gerson, Ken Lexington KY	Ghiorso, Janelle Sonora CA
Gholston, Gary Richmond TX	Gibb, Max Lethbridge Canada	Gill, Amy Lexington KY	Gill, Harriette Lexington KY USA
Gilligan, Patrick Lexington KY USA	Gillispie, Jamie Bristol TN	Ginn, Kerri Easton MD usa	Ginnerty, Kevin McLean VA
Godby, Debbie Lexington KY USA	Godby, Rick Paint Lick KY USA	Goddard, Jim Tacoma WA	Godina, Meg Lakewood CA
Gogue, Karen Long Beach CA	Gold, Rick Las Vegas NV	Goldberg, Jarrod Hankamer TX USA	Goldberg, Stewart Flemington NJ USA
Golden, Richard West Palm Beach FL	Goldstein, David Bala Cynwyd PA	Gonella, Peter Point Pleasant NJ USA	Good, Iris Fayetteville NC
Goodman, Aline Tucson AZ USA	Goodman, H. Greg Lexington KY	Goodman, Hutton Austin TX USA	Goodman, John K. Tucson AZ USA
Goodrich, Alexandra Birmingham AL	Goodrich, Charles Birmingham AL	Goodwillie, Claire Kilkenny Ireland	Gorajec, Joe Indianapolis IN USA
Gosden, John Newmarket UK	Goshen, Martita Sea Cliff NY	Goss, Nancy Hamden CT	Gott, Sheila Lexington KY United States
Gower, Jeff Springfield MO	Graebner, Myles Pendelton KY	Graffard, Lisa-Jane Coye-la-Foret France	Graham, Sheri Versailles KY USA
Grande, Jr, Frank Ocala FL USA	Grant Farrell, Vivian Louisville KY	Gray, Marcia Dumfries VA USA	Gray, William Cottonwood CA USA
Grayeski, Robert Lockport IL	Grdak, Tajana Zagreb Croatia, Europe	Greely, William Lexington KY USA	Green, Finn Frankfort KY
Greenfield, David New York NY	Gregg, Suze Blowing Rock NC	Gribbins, Jim Evansville IN	Grier, Mark Far Hills NJ USA
Griggs, John Lexington KY USA	Griggs, Linda Lexington KY United States	Grimm, Michael Dallas TX USA	Grisolia, Cynthia Versailles KY
Grisolia, Gregory Versailles KY	Gudenrath, Mimi Fayetteville GA	Guidotti, Barbara Pemberton NJ	Gural, Jeffrey R. Standfordville NY
Gurfein, Ron Delray Beach FL USA	Gustafson DVM, Dr Sid Bozeman MT	Guthrie, Margaret Philadelphia PA	Gutterman, Allen Los Angeles CA USA
Guy, Angela Houston TX	Haas, Ginger Tampa FL	Hagale, Jim Strafford MO	Hagan Cosgrove, Joni San Diego CA USA

Haigh, Joanie CA	Hales, Joleen Madill OK	Hall, Jason Eagle ID USA	Hall, Lenny Pahrump NV
Hall, Tom Lexington KY USA	Hamilton, Ken El Dorado AR USA	Hamilton, Lucy Lexington KY	Hancock IV, Arthur Paris KY
Hancock, Alex Lexington KY USA	Hancock, Arthur Paris KY	Hancock, Clay Paris KY	Hancock, Dell Paris KY
Hancock, Hutchi Los Angeles CA	Hancock, Kate Paris KY USA	Hancock, Lynn Lexington KY USA	Hancock, Seth Paris KY USA
Hancock, Staci Paris KY	Hancock, Walker Paris KY	Handel, Hal New Hope PA USA	Hanington, David Bullville NY USA
Hanley, Peter Macomb MI USA	Hannan, Michael Louisville KY	Hannum, Reddy Unionville PA	Harding, Brenda Newburgh NY
Hardy, Hallie Frankfort KY	Harned, Jon Dawson Springs KY	Harper, Kate Encinitas CA	Harrington, Norman Burnsville MN USA
Harris, Kim Hot Springs AR	Harris, Margaret Lexington KY	Harriss, Paul Valders WI	Hartis, Eileen Sealy TX
Hasegawa, Takashi Toyama-ken Japan	Hatton, Vicky Paris KY	Haugh, Mary Marshall Auburn AL	Haughton, Bill Deerfield Beach FL USA
Hawley, Jim Kent WA	Haws, Esq., Nancy Louisville KY USA	Hayes, Jerry Aurora OH	Hayes, Joann Aurora OH USA
Hazard, Holly Falls Church VA	Head, Alec Lexington KY USA	Head, Ghislaine Lexington KY USA	Head-Maarek, Criquette Vauville France
Head, Martine Lexington KY USA	Healy, Tom Rosemount MN	Hebner, Marianne Fallbrook CA	Heller, Robby Easton MD
Henderson, Douglas Reddick FL United States	Hendrickson, John Saratoga Springs NY	Henie, Rob Cardiff CA USA	Herbst, Leanna Ruskin FL
Herrick, Norton Cedar Knolls NJ	Hersey, Rebecca Englewood NJ	Hibberd, Grover Georgetown KY	Hickey, Tom St. Johns Canada
Hicks, Thomas Pompano Beach FL	Higgins, Donna Mount Laurel NJ	Hill, Deborah Winter Springs FL	Hill, Jim Midway KY USA
Hill, Judy Cannon Falls MN	Hilliard, Joanne Lexington KY USA	Hills, George Lexington KY	Hiner, Lezlie Philadelphia PA
Hinkle, Anne Archer Lexington KY USA	Hinkle, Barbara Paris KY	Hinkle, Henry Paris KY	Hinkle, Mary Grace West Hollywood CA
Hinkle, Sam Shelbyville KY	Hinkle, Tom Paris KY USA	Hinson, Dave Cooper City FL	Hirsch, Robert Sunny Isles Beach FL
Hirschle, Robert Wayne PA	Hirsh, Ken New York NY	Hirsh, Lester New York NY	Hirt, Gail Emmett MI
Hnatiuk, Harve Westampton NJ	Hoffman, Geralyn Charlotte NC	Hoffman, Holly Pensacola FL USA	Hogan, Matthew Versailles KY
Holderith, Erika Visalia CA United States	Holderith, Erika Visalia CA USA	Holguin, Diana Bogota Columbia	Hollinger, Joi Lenni PA
Holly, Dale Nicholasville KY USA	Holmes, John Metairie LA	Hood, Rachel Newmarket, Suffolk UK	Hook, Katie Wilmore KY
Hope, Michele Alpharetta GA	Hopkins, Jenni Smithsburg MD	Horton, Linda Peoria IL	Horton, Marian Citrus Heights CA
Host, Jim Lexington KY	Howard, Ginny Lexington KY USA	Howard, Mark Mirror Lake NH	Howard, Neil Lexington KY USA
Howell, Jessica Lexington KY USA	Howell, Jessica Hollywood FL USA	Howes, Lesley Ocala FL USA	Huckabay, Jody Paris KY
Huckabay, Michelle Paris KY	Huddleston, Richard Franklin TN USA	Hudson, Freddie Springfield VA USA	Hudson, Jeff/Virginia Stevensville MT
Huebner, David Kearney NE USA	Hughes, Sydney Lexington KY USA	Hulett, Linda Hondo TX	Hunt, Sue Elmendorf TX
Hurley, Erin Moorestown NJ	Hutchison, Alan Glen Allen VA	Ible, Darrell Jamberoo Australia	Ihle, Kendra Lima OH USA
Ilzhoefer, David Grand Prairie TX USA	Infante, Anthony Tampa FL	Ingram, Lee Ann Franklin TN USA	Ingram, Mike Springfield MO
Ingram, Orrin Nashville TN USA	Inman, Stanley Paris KY	Inselman, Sandra Menasha WI	Irby, Marty Washington, DC USA
Iriarte, Edgardo Buenos Aires Argentina	Irons, Elizabeth Sinks Grove WV USA	Irwin, Barry Versailles KY	Irwin, Gordon Apopka FL USA
Irwin, Kathleen Versailles KY USA	Jackson, Gretchen West Grove PA	Jackson, Michelle Columbus NJ	Jackson, Roy West Grove PA
Jacobs, Kelly	Jaenicke, Kurt	Jaramillo, Jeanclaude	Jarvis, Mike

Floresville TX	Ashland KY USA	Elmont NY USA	Las Vegas NV
Jaworski, Matthew Mississauga, Ontario Canada	Jenkins, Julia West Grove PA	Jenkins, Peter Coeymans Hollow NY	Jennings, Joe Carlisle KY USA
Jensen, Elisabeth Lexington KY	Jessie, John Campbellsville KY USA	Johansson, Nancy Allentown NJ USA	Johnsen, Linda Minneapolis MN
Johnson, Chris Denver CO USA	Johnson, Heather Lexington KY USA	Johnson, Jan Lighthouse Poit. FL USA	Johnson, Joanna Denver CO USA
Johnson, Mary Gahanna OH	Johnson, Peter Montecito CA	Jones, Deborah Huntington Beach CA	Jones, Douglass Lexington KY
Jones, Greg Bristol CT	Jones, John Lexington KY United States	Jones, Libby Lloyd Midway KY USA	Jones, Marie D. Eugene OR USA
Jonsson, Colleen Midway KY USA	Juffet, David East Brunswick NJ	Justice, Bill Lexington KY USA	Justus, Cathy Pagosa Springs CO
Kahn, Matthew Scardsale NY	Kalet, Eric Yardley PA	Kane, John Plymouth MN Usa	Kane, Richard Columbus OH
Kane, Rita Lexington KY	Kanemaru, Ed Arcadia CA	Kanter, Mike Hillsdale NJ USA	Kaplan Chiz, Nancy Shaw MS
Kaplan, Anne Highland Park IL	Karlin, Steve Merrick NY	Kaster, Robert Friday MN USA	Kattner, Emily Minneapolis MN USA
Kaye, Carol Woodbine MD USA	Kayne, Susan Albany NY USA	Keane, Tim Minneapolis MN USA	Keays, Lois Wiarton, Ontario Canada
Kebow, Duke Carlsbad CA	Keeling, Mike Cambridge Canada	Keenan, Chuck Maple Grove MN USA	Keigher, Jason New York NY
Keister, Marie Broadview Heights OH	Keithley, Thomas Landenberg PA	Kelliher, Daniel Kiowa CO USA	Kelly, Jerry Lexington KY USA
Kelly, John Chicago IL	Kelly, Jon Rancho Santa Fe CA	Kelly, Sarah Rancho Santa Fe CA	Kelp, Lars Drammen Norway
Kelzenberg, Anthony Watertown MN USA	Keough, Patty Kent OH	Keppley, Tanya Lancaster PA	Kerr, Sean Brooklyn NY
Kessler, Andrew Annandale VA	Kevorkian, Geirge Bellaire TX	Khalifa, Jayne St. Louis Park MN USA	Khoury, David Las Vegas NV USA
Kieger, Donald Prior Lake MN USA	Killgore , John Juno Beach FL USA	Kinder, Ralph Paris KY	Kinerk, Burt Tucson AZ USA
Kinerk, Nancy Tucson AZ USA	King, Alexa Versailles KY	King, Carolyn Louisville KY USA	King, Karen Sewickley PA
King, Kevin Sewickley PA	Kingsley, Michele Hendersonville NC	Kinisky, Tom Chagrin Falls OH	Kinney, Richard Ballston Lake NY
Kinney, Susan New Tripoli PA	Kinns, Debora Schenectady NY USA	Kirby, John Charles Town WV USA	Kirby, Susan Delhi NY
Kirk, Ronald Lexington KY	Kirk-wagner, Lori Lexington KY	Klein, Jonathan Bethesda MD	Klepaczka, Maria North Tonawanda NY
Klonizos, Kyle Paris KY	Klosek, John Houston TX	Knelman, Jak Lexington KY	Knelman, Suzanne Paris KY
Knowles, Alice Aiken SC	Knudsen, John Chesterfield MO	Koch, Matthew Paris KY USA	Koenig, John Millstone NJ
Kohler, Keith New Bloomfield PA	Kolar, Karen V Lambertville MI	Komorski, Ken Mays Landing NJ	Kouroubacalis, Steve Lynnfield MA
Kowerko, Patricia North Kingstown RI	Krasne, William Washington DC	Kraut, Maureen Perryville KY	Kress, Bernel Jacksonville FL
Kromann, Geraldine Perrineville NJ	Kruger, Ben Los Angeles CA	Kucharski, Natalie Stamford CT	Kuehn, Milo Myakka City FL
Kuhn, Denise Springfield KY USA	Kurgan, John Toronto, Ontario Canada	Kuster, Betsy R Paris KY USA	Kuster, Ted Paris KY
LaBrue, Jimmie Versailles KY	LaDuke, Jason Beverly Hills CA	Lam, Po Liverpool NY	Lamb, Melvin Talbotton GA
Lambert, Christen Waterford VA	Lampman, Jen Liverpool NY	Lange D.O., Lori Lebanon PA	Lange, Kenneth W Nanticoke PA USA
Langert, Shelby Lincoln Park MI	Langford, Elizabeth Bangor ME USA	Langlois, Bryan Lancaster PA USA	Laroche, Lindsay Lafayette CA USA
Lavelle, Patty Middleburgh NY	Lavin, A. Gary Goshen KY USA	Lavin, Elizabeth (Betsy) Goshen KY USA	Lavorato, Tony Darien IL
Lawlor, Elaine Grange Con, Co. Wicklow Ireland	Lawrence, Karen Wellsville OH	Lawrence, Robert Melbourne, Victoria Australia	Lawrence, Sally Lake Charles LA
Lawrence, Stuart	Lawton, Randal	Lay, Jordan	Layton, Margaret

Brooklyn NY USA	Orlando FL USA	Georgetown, Ontario Canada	Paris KY USA
Leak, William Ballston Lake NY USA	Lear, Vange Lexington KY	Lee, Carlene Tyler TX	Lee-Smith, Queen Bloomington MN USA
Lefkow, Len Bathesda MD	Lejzerowicz, Josef Aspen CO USA	Lenert, Dawn Falls Church VA USA	Lennox, Cynthia Pittsburgh PA USA
Lennox, Muriel Toronto, Ontario Canada	Lentini, Lorna New York NY USA	Leonard, James Raleigh NC	Levine, Lara Lexington KY
Levy, Mike Lexington KY	Lillingston, Luke Kilmallock, Co. Limerick Ireland	Lindley, Thomas Maricopa AZ USA	Lindstedt, Berndt Sollentuna Sweden
Lindy, Malcolm Las Vegas NV USA	Lively, Barbara Lewisville TX	Loman, Linda Walnut Hill FL	Long, Paul Warminster PA
Longo, Cynthia Oakton VA USA	Loonin, Larry NYC NY	Lorraine, Jason Kamloops, BC Canada	Loudon, Lucinda Battle Ground WA USA
Louise, Katia Studio City CA	Louw, Robyn Durbanville South Africa	Loveless, Peggi Sidney ME	Lovell, John Brockton MA USA
Lowe, Jeff Versailles KY USA	Luce, Shannon Versailles KY USA	Luce, Shannon Lexington KY USA	Ludlow, Joan Rochester MI
Lunsford, Bruce Louisville KY	Lyman, Marty Cuyahoga Falls OH	Lynch, Judith Boyle Ireland	Lyons, Dr. Sheila MA
M, Kris Corvallis OR	MacArthur, Kim Hemlock MI	Macauley, Frank Madeira Beach FL	MacDonald, Mark Aliso Viejo CA
MacFarlane, Adrienne Ormond Beach FL	Machikas, Brittany Glenmoore PA	Mackay, Elise Locust Valley NY USA	Mackay, Patrick Locust Valley NY USA
Mackintosh, Anne Odessa, Ontario Canada	Magenheim, Neal Manalapan NJ	Maggio, Carol Naples FL	Mahoney, Jean Sicklerville NJ
Makinster, Mike Atlanta GA	Malburg, Barbara Livonia MI	Malkerson, Mary Shakopee MN	Malmstrom, Brent Scottsdale AZ
Mancini, Marietta Boalsburg PA USA	Manganella, Cheryl Brooklyn NY USA	Manner, Karen Arlington Heights IL USA	Manner, Steve Arlington Heights IL USA
Manning, Cheri Alexandria VA USA	Manning, Dr. Monica Saint Paul MN USA	Manning, Kirby Ocala FL	Mansmann, Dick Chapel Hill NC USA
Mardon Stables , Marv Chantler - Loretto. Ontario Canada	Mark, Allen roswell NM	Marks, Phyllis Los Angeles CA	Marsh, Fred Minneapolis MN USA
Marshall, Robin South Island New Zealand	Martin, Ebony Glen Burnie MD USA	Martin, Edward Bourne MA USA	Martin, Kalib Apple Valley MN USA
Massey, Barbara Clearwater FL	Massicotte, Lee Abingdon VA	Massler, Chris Lexington KY USA	Masson, Richard Los Angeles CA
Mast, Henry Caledonia MI	Masters, Cheryl Muncy PA	Masterson, Joan Palm Desert CA	Masterson, Robert Palm Desert CA USA
Mateo, Elizabeth Alpharetta GA USA	Mathayas, Sabeena Minneapolis MN U.S.A	Mathiesen , Hannah Rancho Santa Fe CA USA	Mathis, Michaela Versailles KY
Matthews, Ann Maree Collierville TN USA	Mauro, Joe Clearwter FL	Mayfield, Carroll Aiken SC	McAdam, Christina Fontana CA
McBeath, Gretchen Sunny Isles Beach FL	McCarron, Chris Lexington KY	McCarter, William England	McCarthy, Elisabeth Litchfield CT
McClenathan, Nancy Claremont IL	McCloskey, Sean Chaska MN USA	McCormack, Bernard Janetville, Ontario Canada	McCormack, John Bedford NY
McCrary, Rob Hot Springs AR	McCreery, Kip Palm Beach FL USA	McDonald, James Lexington KY USA	Mcdonald, Katie Seattle WA USA
Mcduffee, David Delray Beach FL USA	Mcfadden, Dale Red Deer Canada	Mcgann, Terry Reno NV USA	McGarity, Leanne Seagoville TX
McGeorge, Wallace Hot Springs AR	McGinnis, James Freehold NJ	McGrath, Jo Ann Charlottesville VA	Mcguffie, Eileen Charleston SC USA
McGuire, William Mora NM	McIngvale, James Houston TX	Mcintosh, Jil Oshawa, Ontario Canada	Mckay, Michael Flushing NY
McKenna, Joe Raleigh NC	McKenzie, Carol Chester SC	McKenzie, Sam Chester SC	Mclean, Nancy Columbia IL USA
McLellan, Roy Scottsdale AZ	McMullen, Andrew Miamisburg OH	McMullen, James Palatine IL	McNally, Thomas Palos Heights IL
McNamara, Chad Mount Upton NY USA	Mcneil, Darren Shakopee MN USA	McNerney, Terry Binghamton NY USA	Mead, Sheila Fisherville KY USA
Mediamolle, Wayne Harahan LA	Meehan, Jan Odessa FL	Melia, Beth Medway MA	Melvin, Sandra Paris KY USA
Mendiola, Patricia	Meng, Ken	Metcalfe, Peter E A	Michela, Dennis

Covina CA	Lexington KY United States	Newmarket, Suffolk England	Phoenicia NY USA
Michelsen, Franklin Cotuit MA	Miller, Bill Weatogue CT	Miller, Connie Danville KY	Miller, Douglas Lake Worth FL USA
Miller, Dr. Alan Vestal NY	Miller, H. Charles Paris KY	Miller, Jonathan Lexington KY USA	Miller, Joseph Midway KY USA
Miller, Kathy Stratford NJ	Miller, Lincoln Palm Beach Gardens FL	Miller, Linda Fort Myers FL USA	Miller, Margie Statesville NC
Miller, Mike Louisburg KS USA	Miller, Thomas Smithville TN USA	Millis, Robert Saratoga Springs NY USA	Mills, Sharon Hendersonville NC
Mineo, Erica Cupertino CA USA	Minott, Geri Cardiff by the Sea CA	Minshall, Barbara Mississauga On Canada	Minshall, Mary North Las Vegas NV USA
Minten, Craig Mulvane KS USA	Mirabito, Jeanne Paris KY USA	Mitchell , Rhonda Center Point TX	Mitchell, Lane B. San Ramon CA USA
Mitchell, Regina Warren OH	Moe, Paul White Bear Lake MN USA	Moe, Roger Erskine MN USA	Moens, Virginia E. Hobe Sound FL
Moes, Dennis No Saint Paul MN USA	Moessner, Warren Ravena NY	Mohammed, Tom Richmond, BC Canada	Montaldo, Rebecca Fayetteville NC USA
Moore, Eleanor Columbia SC	Moore, Jackson Myrtle Beach SC USA	Moore, Michael Lebanon IL	Moore, Michael Montgomery Village MD USA
Moore, Patrick St Louis MO	Morell, Richard Brooklyn NY	Moretti, Marie Reno NV USA	Morrill , Lisa Norfolk VA USA
Morris, Joanie Paris KY USA	Morrison, Vivien Lexington KY	Mortenson, Candace St. Pete Beach FL	Moseley, Trish Hamilton MA
Mosier, Cathy Berea KY USA	Motion, Andrew Upperville VA USA	Motion, Anita Elkton MD USA	Motion, Graham Elkton MD USA
Mott, Briana Versailles KY	Munderloh, Dorothy Jo-Ann Baltimore MD	Munro, Dave Canada	Murga, Jorge Chula Vista CA USA
Murphy, Richard Los Alamitos CA	Murray, Garrett Monkton MD USA	Murray, Marlene Harrisburg PA USA	Murray, Marlene Harrisburg PA USA
Murrietta, Tony Irvine CA USA	Napier, Lisa Cramerton NC United States	Narayanappa, Anand Cave Creek AZ	Nash, Ellen-Cathryn Hoboken NJ USA
Nation, Alicia Eastville VA USA	Naylor, Debora Dover DE	Naylor, Lou Waxahachie TX	Neagle, Jack Lexington KY
Neal, Tamese Corona CA	Neuhart, Fritz Dublin OH	Neuman, Celeste Lexington KY	Neuman, Emler Lexington KY
Nevins, Janet Southbury CT USA	Newman, Liz Givat Brenner England / Israel	Newman, Timothy Wheeling WV USA	Nicholas, Sydney Pleasant Valley NY
Nicholls, Barbara Paris KY	Nichols, William Syosset NY USA	Nicholson, Joe Browne Lexington KY USA	Nielsen, Jason Pittstown NJ USA
Nielsen, Kassandra Woodstock CT	Normile, John Plymouth MI	Norton, Michael Lakewood NJ USA	Norwood, Bernadette Cleveland OH
Novak, Nikki Savage MN USA	O'Brien, Keith Garden City NY USA	O'Brien, Laura Cypress TX USA	O'Brien, Leo West Hempstead NY USA
O'Brien, Robert New York NY USA	O'Connell, Connie Secaucus NJ USA	O'Connell, Ellen Ocala FL	O'Connell, Patrick Lexington KY USA
O'Dwyer, Philip Bothell WA	O'Regan, Kevin Redwood City CA	O'Reilly, Chelsea Gansevoort NY USA	O'Rourke, Dan Denver CO
Ochocki, Robert Riverside CA	Ochs, Judith Chadds Ford PA	Odenkirk, Sharon Vienna VA USA	Okrzesik, Bob Houston TX
Ola, Earl Morriston FL	Olafsen, Matthew Tampa FL	ONeil, Bonnie New York NY USA	Opdycke, Suzette Aztec NM usa
Oppenheim, Bill Scotland, UK	Osborn, Donald Cincinnati OH USA	Osborne, Jennifer Lester PA USA	Osborne, Paul Altadena CA USA
Osenburg, Nicole Baltimore MD USA	Otto, Mark Olivet MI USA	Otway, Teri San Diego CA	Ouellette-Danis, Keely Enfield CT
Overton III, Jesse Bloomington MN USA	Overton, Jesse Bloomington MN	Overton, Lana Bloomington MN United States of America	Overton, Rachel Bloomington MN United States
Owens, Mike Lexington KY	Pachota, Jamie New Hudson MI	Paiement, Luc Mont-Royal, QC Canada	Paige, Jeff Los Angeles CA
Palmer, Ronda Paris KY USA	Panitch, Blaine Willowbrook IL	Papa, Laura New Orleans LA	Papp, Kathryn Cream Ridge NJ
Pappalardo, Phil Edison NJ USA	Parker, Kathleen Bethel PA	Parker, Linda Mount Dora FL USA	Parker, Walter Philadelphia PA USA
Parkinson, Mark	Parks, Christine	Parks, Jerry	Parsons, Michelle

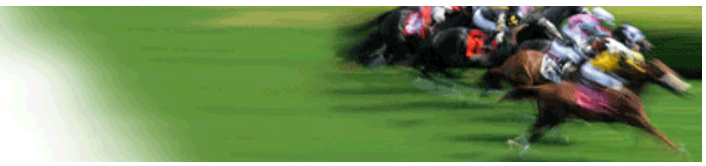
Bethesda MD	Vienna VA	Ocala FL USA	Belle Plaine MN USA
Pascarella, Carl San Francisco CA	Patton, Cory New Orleans LA	Paulhus, Marc Hendersonville NC	Pavey, Linda Cincinnati OH
Pavlichko, Michael Fort Myers FL USA	Payne, KC Arlington TX USA	Peacock, Andrew Austin TX	Pearson, Eryn Princeville IL
Pederson, Wesley Kamloops, BC Canada	Peele, Andrew Allen TX USA	Pegg, Peter Middleburg VA USA	Pelkey, Jill Stokesdale NC
Pendergast, Tom Richmond VA USA	Penn, John Paris KY	Perry, James Dallas TX USA	Pesot, Jeff Basking Ridge NJ
Petter, Jr., Stanley D. Lexington KY	Pflugheber, Jill Hermon NY	Phillips, Jackie Raton NM U.S	Picavet, Christine Fallbrook CA
Picciotti, Marrey Oak Park IL	PIcciotti, William Oak Park IL	PIgg, Rex Friendswood TX	Pilkington, Helen Glen Head NY USA
Plaisance, Douglas Loranger LA	Plant, Emily Missoula MT	Pleshe, Darstan Shakopee MN USA	Plummer, Dick Savage MN USA
Plummer, Jeff Savage MN USA	Plunket, Piers England	Pobjecky, Judith Blacksburg VA	Polivka, Mark Naperville IL
Polk, Hiram Louisville KY USA	Pollard, Catherine New Bern NC USA	Pontrelli, Joanne Huntington NY USA	Pope, David Versailles KY
Popham, Wayne Hamel MN	Potasiewicz, Kendra Gansevoort NY USA	Potter, James Paris KY	Potter, James Afton VA
Pournaras, Ann Flemington NJ	Powell, David Lessard Et Le Chene France	Powers, Walter Harrison OH	Poyah, Anil St Croix Virgin Islands
Pozsar, Randy Raleigh NC	Prado, Edgar Hollywood FL USA	Prather, John Georgetown KY	Prather, Nathan Louisville KY USA
Prather, Steve Hobbs NM USA	Prather, Susannah Georgetown KY	Prekop, Paul Las Vegas NV	Price, Bill & Carrington Mineral Springs NC USA
Priest, DVM, Gary T. Versailles KY USA	Priest, Kathy Versailles KY USA	Primm, Michelle Lexington KY USA	Pringle, Valerie Annapolis MD
Pryor, Sandra St. Paul MN USA	Quickel, Dan Paris KY USA	Quinn, Blake Los Angeles CA	Quinn, Tim NY NY
Racki, Troy Shasta Lake CA	Raglin, Duane Midway KY	Ralph, Donna Viera FL	Ramirez, JeAnna Redwood City CA
Ramirez, Laura Seattle WA	Ramirez, Tommy Lewisville PA	Rampulla, Jimmy Bayside NY	Randolph, Tom White Plains VA
Rankin, Gayla Brock TX USA	Ransom, John Sherman Oaks CA	Ranwick, Sr., Mr. and Mrs. Robert Plymouth MN	Rappa, Antoinette Denver CO
Rarick, Gina Maisons-Laffitte France	Ratza, Maggie Chicago IL USA	Ravenwood, Kira Fredericksburg VA	Ravit, Alexa NY
Rawlins, Nan Hamilton OH	Reagan, Nancy Glen Ellyn IL	Reavis, Dave Louisville KY	Rebhan, Eric Fort Lauderdale FL
Reck, Bill Mechanicsburg PA	Reddoch, Jeff Maurice LA	Redmond, Garrett Paris KY	Redmond, Robin Paris KY usa
Reed B.V.M.S., Dr Tracey Arthur River Australia	Reed, Billy Louisville KY	Reed, Duane Minneapolis MN USA	Reed, Jason Coos Bay OR USA
Reepmeyer, Bill Meredith NH USA	Reeves, Joy Colorado Springs CO	Register, Layton Lexington KY United States	Reid, Carrie Catonsville MD
Reid, Cindy NY	Rekow, Patty River Falls WI	Remondini, Mary Menomonee Falls WI	Rempel, Don Winnipeg, MB Canada
Renfro White, Sandra Lexington KY	Reynolds, Shannon Charlotte NC	Rhodes, Karen Fairfax VA	Richards, Helen Camden SC USA
Richardson, Denise Mount Hope, Ontario Canada	Richmond, Conni Riverside CA	Richter, John & Kris Perkins OK USA	Ricker, Robert Goodlettsville TN
Rickert, Darrel Columbus NE USA	Rickert, Elaina San Dimas CA USA	Rickert, Gene San Dimas CA USA	Rickert, Matthew San Marcos CA USA
Rickert, Tricia Columbus NE USA	Riedel, L.M. Dallastown PA	Riggs, Mary Jane Muncie IN	Ringlee, Elizabeth Delanson NY
Ringler, Maria Harrington DE USA	Rini, Denise Lyndhurst OH USA	Ritter, Carol Tucson AZ	Roach, Bob Moncton Canada
Roach, Robyn Versailles KY	Robbins, Steve Cleveland Heights OH	Roberts, Alicia Parkesburg PA	Roberts, Elizabeth Odessa FL
Roberts, Hailey Stony Point NC	Roberts, Monty Solvang CA USA	Robins, Tylah Swindon England	Robinson, David Cranston RI
Robinson, Diane Lexington KY USA	Robinson, Don Lexington KY	Roche, Cynthia Carpinteria CA USA	Roche, Kathleen Santa Barbara CA USA

Rodriquez, Roberto Temple TX	Roebling, William Princeton NJ	Rogers, Joelle Houston TX USA	Rogers, Kathy Poulsbo WA
Roggenkamp, Edward Versailles KY	Rolfe, Denise Wyncote PA	Rolfe, Randy Wyncote PA	Rollinson, David Osprey FL
Rollinson, David Osprey FL USA	Romanet, Louis Boulogne Billancourt France	Rose, Jim Paris KY USA	Rose, Michael Ft. Lauderdale FL
Rosenberg, Dan Versailles KY	Rosenberg, Neal New York NY	Rosenkranz, Rich Flower Mound TX USA	Ross, Katy Lexington KY USA
Ross, Sharon Urbandale IA	Rossbach, Charles Honesdale PA USA	Roulston, Allison West Palm Beach FL	Rounsefell, Craig Monrovia CA
Roya, Joan Baytown TX	Rozwadowska, Joanna Brzesko Poland	Rubens, Carky Scarsdale NY	Rubin, Mike Beech Island SC
Ruhnke, Chris Cincinnati OH	Ruhnke, Melissa Cincinnati OH	Rullman, Cindy Lexington KY	Rupert, Gaynor South Africa
Russo, Nicholas Lawrenceville VA United States	Ryan, Heather White Hall MD USA	Ryan, Jim Rochester NY United States	Ryan, Marcus Windsor SC USA
Ryan, Mike Kankakee IL USA	Ryder, Bradford Washington DC USA	Saart, Katharine Vista CA	Sahadi, Jenine Del Mar CA USA
Salazar, Michelle Aztec NM USA	Saltzman, Tabitha Indianapolis IN USA	Salvi, Nick Coconut Creek FL USA	Samuels, Rick Lexington KY
Sandahl, Paul New Lennox IL	Sanders, Megan Conyers GA USA	Sanders, Owen St Paul MN USA	Sandhu, Nishaan Lexington KY
Sandmann, Brian Louisville KY	Santangelo, Geogre New York NY	Santulli, Richard Colts Neck NJ	Sanzone, Vincent Commack NY
Saurbier, Marvin Citra FL USA	Sawyers, Diana Stephens AR	Sayre, Lois Laurelville OH USA	Schander, Raoul Stillwater MN USA
Schellenberger, Jessica E Louisville KY	Schettine, Alex Richmond VA USA	Schickli, Dillon Stanwood WA	Schmitz, Steve Pana IL
Schott, Allan Mahwah NJ USA	Schram-Dillon, Cindy Hallandale Beach FL	Schriock, Constance Mcintosh SD USA	Schroer, Debra Fort Pierce FL
Schulz, Stan East Lansing MI USA	Schuster, Louis Minneapolis MN USA	Schuster, Steve Bourne MA	Schweitzer, Rich Cherry Hill NJ
Scotto, Barbara Freeport NY	Seavy, Jo Denver CO	Seidel, Marion Port St Lucie FL	Seitz, II, Fred Versailles KY
Seitz, Frederick J. Versailles KY	Seitz, Joe Versailles KY USA	Sentkowski, Kathleen Gaithersburg MD USA	Seth, Altshuler Matawan NJ
Sexton, Gary Jackson MI	Seyle, Joyce Newington GA	Shaffer, Matthew Highland Park IL	Shaikun, Gerry Sarasota FL
Shanagher, James Loudonville NY USA	Shannon, George Minneapolis MN USA	Sharp, Steve Lexington KY	Shaw, Doug Sarasota FL
Shaw, Simon Bethune SC USA	Shepherd, Patrick Culpeper VA USA	Shepherd, Rebecca Culpeper VA USA	Shepherd, Shannon Highland AR
Sheppard, Jonathan West Grove PA USA	Sherer, Donald Henderson NV USA	Shevick, Mark Saugus CA	Shiflet, Bobby Paris KY USA
Shirley, Patti Tucson AZ USA	Shively, Marilyn Farmington Hills MI	Shoaff, Michelle Oviedo FL USA	Shockey, Patricia Charles Town WV
Shuback, Alan New York NY USA	Shuford, Nancy Hickory NC	Siegel, Bart New York NY	Sigler, Jules Ontario Canada
Silva, Jerry Long Beach NY USA	Simcox, Don Oak Hill VA	Simmer, Kate Philadelphia PA	Simmons, Amanda Orlando FL
Simms, Hunter Lexington KY USA	Simon, Mark Lexington KY USA	Simon, Mary Lexington KY USA	Sinatra, Stephen Manchester CT USA
Sinclair, Carol PA	Singh, Anant Durba SA	Sisung, Elaine Gibraltar MI	Sitongia, Richard Phoenix AZ USA
Skand, David Tampa FL	Skeen, Holly Prescott Valley AZ	Slater, Lysa Lutz FL	Slater, Mary Enfield CT
Slocum, Donna Buckeye AZ	Small, Jane Pittsburgh PA USA	Smeallie, Shawn Alexandria VA USA	Smith, Andrew Eramosa, Ontario Canada
Smith, Connie Prospect KY USA	Smith, Gary Ocala FL	Smith, J David Lexington KY USA	Smith, Jack Prospect KY USA
Smith, Joe Naples FL	Smith, Laura Ormond Beach FL	Smith, Melinda Lexington KY	Smith, Patty Culver City CA
Smith, S. Suzanne Lexington KY US	Smith, Shawn Lansing MI	Smith, Tamme Little Rock AR	Smith, Terry Linden MI
Smoak, Julie	Smoot, James	Snelling, Barry	Snyder, Jennifer

Lexington KY	Charles Town WV	Lexington KY USA	Indialantic FL
Solveyra, Eduardo Alfredo Caba Argentina	Sonder DVM, Claudia Napa CA USA	Sonsteby, Charles Dallas TX USA	Spadafore, Timothy Leesburg VA USA
Spalding, Bobby Paris KY	Sparacello, Paul New Orleans LA	Spear, Amy Jackson WI	Spencer, Sally Pahrump NV
Spicer, Nancy Columbia SC	Spinelli, John DeLand FL	Spiring, Charlie Winnipeg, Manitoba Canada	Spoone, Lisa Bean Station TN
Springer, Bill Warrenton VA	Squires, Jim Versailles KY	Squires, Mary Anne Versailles KY	Srikanth, Sanjay Clifton VA
Stafford, Howard Ocala FL USA	Stalker, Christina Beaverton OR USA	Standish, Jolin Elk Grove CA USA	Steffen, Daniela Gifhorn Germany
Stein, Harvey Gansevoort NY United States	Stepenovitch, Joseph Fort Lauderdale FL USA	Stephens, Art Murrieta CA	Sterck, Greg Covington LA
Sterck, Simone LA	Stern, Samuel Independence MN USA	Stevens, Gary Sierra Madre CA USA	Stevic, Charlene Citra FL USA
Stewart, Cindy Paris KY	Stewart, Shelley Vancouver WA	Stewart, Steve Paris KY USA	Stiff , Lisa Lexington KY USA
Stillman, Kay Berthoud CO USA	Stirrup, Heidi Haymarket VA	Stivers, Sue Nicholasville KY USA	Stockmen, Phil M Edmonton, Alberta Canada
Stokes - Donehower, Pamela Middleburg VA	Storm, Jennifer SeaTac WA	Strawbridge, George Wilmington DE	Strawgate, Pat Coral Gables FL
Stronach, Frank Paris KY	Struder, Ray Farragut TN	Stuart, Ann Weavervillr NC	Stumpf, Tom Saint Paul MN USA
Stuve, Lloyd Savage MN USA	Suchy, Cherie Louisville KY USA	Suffern, Pat Mandeville LA	Sulkes, Marc San Diego CA
Sullivan, David Benbrook TX	Sullivan, Melissa Lexington KY	Sullivan, Michael Sherborn MA	Sullivan, Paul Lexington KY
Sullivan, Thomas Elburn IL USA	Summers-Harmon, Susan Bourbon MO USA	Sunblade, Barbara Parker CO	Super, Denise Pittsburgh PA USA
Susmarski, Ronald Willowbrook IL	Sutton, Dare Eddyville KY USA	Sutton, Joseph Houston TX	Swain III, Jack New York NY USA
Sweezey, Wayne Lexington KY USA	Sweyer, Nancy Peotone IL	Switzer , David Lexington KY USA	Sylvester, Andrew San Francisco CA
Sylvester, Doug Indianapolis IN	Tackett, Kista Louisville MS	Takter, Jimmy East Windsor NJ USA	Talley, Cassandra Houghton MI
Tanz, Meryl Los Olivos CA	Tatham, Deborah Houston TX	Tatham, Tom Houston TX	Taube, Paula Paris KY USA
Taylor, Arlene Hammonton NJ	Taylor, Kitty Lexington KY	Taylor, Lisa Clearwater FL USA	Teague, Larry Hobbs NM USA
Teal, Jimmy Fayetteville NC USA	Theodore, William West Palm Beach FL	Thomas, Alice Edenton NC	Thomas, Susanna Lexington KY USA
Thompson, Alison Paris KY USA	Thompson, Glenn Aiken SC	Thomson, Bob Windham NH	Thren, Barb North Myrtle Beach SC
Tighe, Mike New York NY	Tijou, Dominique Lexington KY	Timmerman, Anna New Orleans LA	Tingberg, Keith Stuart FL
Tirone, Paris Philomath OR	Tirrell, Elyse New York NY USA	Tivnan, Michael Mashpee MA USA	Tobey, Elizabeth Greenbelt MD
Toby, Milt Georgetown KY	Tokarek, Chris Sarver PA	Tokarek, Rich Sarver PA	Tomasello, Nicole Jamesville NY
Tommila, Peter Falmouth ME United States	Tonzi, Eileen Galt CA	Townsend, Darlene Lewes DE	Townsend, Diane Hamilton New Zealand
Townsend, Wallace Georgetown DE	Tracey, Deborah Manukau Heights, Auckland New Zealand	Travis, Joseph Vestal NY USA	Tremper , Amy Helen Boulder CO USA
Troeller, Gordian Upper Bucklebury United Kingdom	Troy, Eric Suffern NY	Trujillo, Aimee Anthony NM	Trujillo, Anthony Anthony NM
Trull, Frankie Middleburg VA	Tucker, Larry Minneapolis MN USA	Turano, Mercy Tampa FL	Turco, Carrie Las Vegas NV
Turcotte, Ron Van Buren ME	Tutcher, Christine Tampa FL	Twigg, Sandy Enterprise OR USA	Ulmer, Janet East Greenville PA
Urbano Grajales , Luis Alberto Lescar France	Van Berg, Jack Hot Springs AR	Van Den Brink, Ben Bennebroek The Netherlands	Van Wieren-Page, Jenny Paris KY USA
Vance, Ethan Hubbardston MI USA	Vandemotter, Heidi Garrettsville OH USA	Vanmeter, William Lexington KY USA	Varone, Robin Fort White FL

Varosky , David Edwardsville PA USA	Vaughan, Cheri Annapolis MD United States	Vaux, Kirk Mercer Island WA	Veneziano, K.R. Monroe NJ USA
Viaene, Jackie Commerce Twp MI	Viarruel, Handel Toronto Canada	Vise, Jr., Allen T. Nicholasville KY USA	Voelker, Marcy Moorestown NJ
Vogt, Debby Klerksdorp South Africa	Vogt, Willy Chicago IL	Voss, Elizabeth Monkton MD USA	Voth, Robert Cleveland OH
Vukovich, Robert Colts Neck NJ	W, S Louisville KY	Wade, James Hartford CT	Wagley, Rachel Lehigh Acres FL USA
Wagner, Chris Triftern Germany	Wagner, Paul Charlottesville VA USA	Wagner, Susan Chatham NY	Waldman, Ric Lexington KY
Walker, Linda Glen Allen VA	Wallace, Kim Langley Canada	Wallace, Madonna Chicago IL	Wallace, Ron Versailles KY
Walpole, Richard Fort Erie Canada	Walter, Christian Rome Italy	Ward, Donna Paris KY USA	Ward, Jo-Anne Brampton Canada
Ward, John Paris KY USA	Warner, Harvey Winnipeg Canada	Washington, Claudie Duluth MN USA	Waterhouse, Gai Kensington, NSW Australia
Watson, George Minneapolis MN USA	Watts, Kelley Latham NY	Waugh, Virginia Anthony FL	Weber, Charlotte Ocala FL
Weber, Melissa Butler PA USA	Webster, Dennis Mineral VA USA	Weddle, Steve Chattanooga TN	Weeks, John South Berwick ME
Weglarz, Paula Frankfort KY	Wegner, Brian Minneapolis MN USA	Weidner, Dianne Lexington KY	Weil, Ed Winnetka IL
Weil, Shannon Cool CA	Weiner, K.C. Houston TX USA	Weinkauf, Keith Levittown NY	Weisbord, Bradley New York New York USA
Weismann, Rodger Watham MA	Wellington, Harold CA	Wells, Kim Woonsocket SD	Wells, Tiana Sonoma CA
Wendlinger, Diana Prescott Valley AZ	Werneth, Coy Ocala FL	West, Murray Paris KY USA	Weston, Allan New York NY United States
Westphal, Patricia Soldiers Grove WI USA	Wheeler, Kaherine Baton Rouge LA	White , Mary Catherine Lexington KY USA	White, Karyn Gillette WY
Whitehouse, Rosemary United Kingdom	Whitfield, Edward Washington DC USA	Whitmire, Gene El Cajon CA	Whitney, Mary Lou Saratoga Springs NY
Wicks, Tom Eden Prairie MN USA	Wiedemer, Carolyn Taylorsville KY	Wienclaw, James Melville NY	Wiesman, Jon York PA USA
Wilhite, Andy Hemet CA	Wilkins, Arlene Mathis TX	Williams, Donna Troy PA	Williams, Rod Hooper UT USA
Willis, Angela Oxford AL USA	Willwerth, Sandra Paris KY	Wilson, Cindy Bokeelia FL	Wilson, Patty Carrollton TX
Wiltz, James St. Paul MN	Wiltz, Jane St. Paul MN	Winterhalter, Janice Aurora CO	Winters, Fred Salvisa KY USA
Wirth, Jennifer Chicago IL USA	Wisniewski, Scott Holland MI	Witkowski, Kath Hilton Head Is. SC	Wohlers, Laura Spring TX
Woitkowski, Matthew Staten Island NY	Wolpert, Elizabeth Albertson NY	Wong, Allen Los Angeles CA	Wong, Daniel Los Angeles CA
Wood, Barbara Woodway TX USA	Wood, Robert Garden City NY	Woolcott, Lauren The Plains VA	Woolcott, Rene Ralph Middleburg VA
Worthington MD, W. Bradley Nashville TN USA	Woskow, Steve Incline Village NV	Wright, Andrew San Diego CA USA	Wright, Jill Thompsons Station TN USA
Wyss, Albert Brooksville FL	You, Yin Los Angeles CA	Young, Leslie Marengo OH	Younker, Bob Cleveland OH USA
Zaborowski, Richard Newark NJ	Zerolo, Michel Miami Beach FL	Zirkle, Cynthia Springfield OH USA	Ziroli, Clem Norton MA
Zoldan, Alan Youngstown OH	Zoldan, Bruce Canfield OH	Zuanetti, Mario Pompano Beach FL USA	

Water Hay Oats Alliance
Stop Drugs in Racing



June 20, 2018

The Honorable Bob Latta
Chairman
Committee on Energy and Commerce
United States House of Representatives
Washington, DC 20515

The Honorable Jan Schakowsky
Ranking Member
Committee on Energy and Commerce
United States House of Representatives
Washington, DC 20515

Dear Chairman Latta and Ranking Member Schakowsky,

Thank you for giving the Water Hay Oats Alliance (WHOA) the opportunity to share our support of The Horseracing Integrity Act of 2017 (HR2651) with you.

The topic of clean racing and anti-doping in horseracing is not a new one. In the 97th Congress, Senator Charles McC Mathias of Maryland introduced *The Corrupt Horseracing Practices* Act and on May 26, 1982 he held a hearing on the bill in his judicial sub-committee. During the proceedings, industry representatives pledged to remedy the doping problems in our sport. Over the last 36 years, Congress has waited, while drug use in racing has continued to get worse. Try as it may, the industry has not been able to solve the problem.

During the 112th Congress, in May of 2011, Congressman Ed Whitfield of Kentucky introduced *The Interstate Horseracing Improvement Act of 2011 (HR1733)* to prohibit the use of performance-enhancing drugs in horseracing. Mrs. Schakowsky was an important co-sponsor to the bill and our efforts.

In the 113th Congress, Congressman Joe Pitts of Pennsylvania introduced *The Horseracing Integrity and Safety Act (HR2012)*, also co-sponsored by Mrs. Schakowsky. But as with Mr. Whitfield's bill in the preceding congress, the legislation had little industry support and failed to move out of committee.

In May of 2012, the Water Hay Oats Alliance (WHOA) was founded as a grassroots movement of like-minded individuals who support federal legislation to prohibit the use of performance-enhancing drugs in the sport of American horseracing. Over the last six years, this alliance has given its members a collective voice as we stand together with one message: drug free racing.

Since WHOA's founding by a handful of thoroughbred owners and breeders, WHOA has grown to over 1,600 members, all with a stake in the game. Today WHOA's membership includes owners and breeders, Hall of Fame trainers and jockeys, equine veterinarians, horse industry professionals, track

owners and race fans from across the United States. Each has pledged to support this legislation so vital to our sport and industry.

In 2015, an important change came about. WHOA along with the Jockey Club, the Breeders' Cup and Humane Society of the United States (HSUS) joined together to found the Coalition for Horseracing Integrity (CHRI). Through the CHRI, fifteen industry groups have been able to come to the table to support Congressman Andy Barr (R-KY), Congressman Paul Tonko (D-NY) and their bi-partisan legislation, *The Horseracing Integrity Act (HR2651)* now before this 115th Congress.

HR2651 grants control over rule-making, testing and enforcement of a uniform anti-doping program to a new independent authority headed by the U.S. Anti-Doping Agency (USADA) - the same entity recognized by Congress as the official anti-doping agency for our Olympic, Pan American and Paralympic sports.

Through HR2651, USADA will create an agency to put the current dysfunctional patchwork of state-to-state rules and penalties under one national uniform anti-doping program, and on a level playing field in step with international standards. WHOA supports the International Federation of Horseracing Authorities (IFHA) rules of racing that ban all performance-enhancing drugs.

It is obvious that after unfulfilled pledges to Congress and years of committee review and discussion, America's racing industry cannot police itself by eliminating the proliferation of performance-enhancing drugs in our sport, nor does it possess the power to adequately punish the purveyors of these drugs. For our sport and industry to survive, we need this important piece of federal legislation.

Doping destroys public confidence in racing, defrauds the betting fan, weakens the genetic pool of our American racehorses and, most importantly, puts the life and limb of our equine athletes and their jockeys at risk.

On behalf of the Water Hay Oats Alliance and its 1,600 members I would ask that you give HR2651 your utmost attention and consideration. Our sport cannot survive much longer without it. American horseracing needs one set of drug and medication rules, one set of testing procedures, one set of penalties. American horseracing needs to join racing jurisdictions from around the world with like rules for drugs and medications. American horseracing needs to reclaim its place among the general public and racing fans alike as a sport with integrity and fair competition. HR2651 is our best hope.

Sincerely,

Staci W. Hancock
WHOA Co-Founder and Managing Member

https://www.nbcwashington.com/investigations/160-Racehorses-Died-From-Injuries-at-Charles-Town-Races-Since-2014-390369632.html

160 Racehorses Died From Injuries Suffered at Charles Town Races Since 2014

By **Scott MacFarlane**, **Rick Yarborough**, **Steve Jones** and **Jeff Piper**

Published at 6:40 PM EDT on Aug 16, 2016 | Updated at 7:54 PM EDT on Aug 17, 2016

A database maintained by the Humane Society of the United States shows the Charles Town race track in Jefferson County, West Virginia stages among the most horse races in the nation. Since 2014, more than 160 thoroughbred racehorses have died from injuries suffered before, during or after race days at Charles Town, the News4 I-Team found. (Published Wednesday, Aug. 17, 2016)

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- *State law requires Charles Town to operate at least 220 racing days each year or to formally request approval for reducing its schedule.*
- *A Humane Society database shows Charles Town stages among the most horse races in the nation, including 13,610 in 2014.*
- *The state racing commission recommended stewards watch races from outside, but at 12 races the I-Team saw, stewards observed from a booth.*

More than 160 thoroughbred racehorses have died from injuries suffered before, during or after race days at the Charles Town race track in Jefferson County, West Virginia, since January 2014, according to a review of state racing records by the News4 I-Team.

Though state records show the track does not suffer from a significantly higher rate of horse mortalities than others nationwide, an I-Team investigation found Charles Town is operating a larger number of races than almost any other track nationwide.

The review also uncovered a series of disagreements between track managers and state racing officials over safety protocols and disciplinary action against horse trainers.

Charles Town Races managers said the state racing agency is failing to follow its own recommended safety protocols and is allowing trainers with a history of horse injuries to continue operating at the track.

The 160 deaths reviewed by the I-Team include a freak and tragic incident in December 2014, in which a horse stumbled, lost its jockey, then broke free in the wrong direction, triggering a head-on collision with other thoroughbreds on the track.

An investigation into the incident ordered by the West Virginia Racing Commission recommended changes in safety protocols to prevent future mishaps, including a recommendation that a state racing steward watch future races with binoculars from an outdoor position along an elevated rail above the grandstand. The investigation found the stewards were instead watching the December 2014 race on a TV monitor from inside a glass-enclosed booth when the incident occurred.

The I-Team review of multiple races at Charles Town in the year since the recommendation was issued found stewards are continuing to watch

races exclusively from inside the glass-enclosed booth, not out on the railing with binoculars. During 12 races viewed by the I-Team, racing stewards watched the race from the recommended position on the railing zero times.

A three-year investigation by the News4 I-Team raises questions about the number of thoroughbreds dying after competing at a West Virginia track. Scott MacFarlane reports.

(Published Tuesday, Jan. 10, 2017)

The stewards would be better positioned to see wrong-way horses and other safety hazards by watching races live along the outdoor rail, Charles Town Races operations manager Erich Zimny said.

“Their employees are not doing what was in their initial recommendations,” Zimny said. “We do feel there is some risk by them not (watching on the rail), but all we can do is bring it to the racing commission's attention.”

When asked why the Racing Commission hasn't ordered the stewards to move outside to a position on the rail, a spokeswoman for the West Virginia Racing Commission said, "Having constant communication with the other two stewards reduces reaction time in the event there is a problem on the track. This is delayed if a steward is outside on the rail viewing the race."

At least two of the other horses euthanized since the beginning of 2014 were overseen by assistant trainer Scott Lane of Pennsylvania. Lane is at the center of a different dispute between the track and the West Virginia Racing Commission.

Lane's horses have suffered at least five break downs at Charles Town. The state racing commission ordered Lane expelled from the track in 2014, after a state investigation concluded Lane misled investigators and showed "a lack of candor" about one of those horses. The horse in question, Diplomatic Gal, died from an injury suffered in a Charles Town race in early 2014, according to state records obtained under the Freedom of Information Act. Footage of the race obtained by the I-Team shows the horse pull up with an apparent injury midrace.

"That's their interpretation," Lane said. "I was here (the day of the workout). There was documentation saying I was here."

Lane said he is dedicated to his horses and often works a 15-hour day to prepare and care for them. "A lot of devotion goes into these horses to make a little bit of money. Half the people who do it love the game." Lane said he was financially impacted by his ejection from the track. "I lost everything," Lane said.

"For someone to come in and accuse me of doing something that I didn't do and take everything from me, that's hard," he said. "But look where I am. I'm right back here."

The state racing commission's investigation said Lane made "false statements" that Diplomatic Gal had undergone a required pre-race workout the day the horse suffered the fatal injury. The investigation also cited a series of other injuries suffered Lane's horses in 2014 and said, "(Lane's track record) is of obvious and serious concern and must be considered in evaluating the ejection period in this case."

Though the West Virginia Racing Commission ejected Lane from Charles Town shortly after, the agency allowed him to return to his operations at the track in June 2015. By August 2015, another of Lane's horses, failed to complete the race and needed a van escort from the track, according to race records.

Charles Town managers said they strongly disagree with the agency's decision to allow Lane to resume his operations at the track. "His ejection from the track was curtailed," Zimny said. "He's back racing horses here. If it was up to us, unilaterally, it would not have happened."

In its statement to News4, the West Virginia Racing Commission said, "The West Virginia Racing Commission utilized the services of an independent party to conduct a full hearing on the matter." The agency confirmed the hearing recommended Lane be allowed to return June 25, 2015, to Charles Town. The agency's statement said, "After reviewing the transcripts of the hearing and recommended decision, the Commission voted to adopt the recommendation of the hearing examiners."

A database maintained by the Humane Society of the United States shows Charles Town stages among the most horse races in the nation. There were 13,610 horse starts at Charles Town in 2014, according to the database. A horse start is the departure of a thoroughbred from the starting gate at the track. Charles Town had almost double the number of starts of some high-profile U.S. race tracks, including Aqueduct and Belmont. Charles Town also had thousands more starts in 2014 than

major Maryland tracks, including Pimlico and Laurel. Only one track nationwide, Gulfstream Park in Florida, had a larger number of horse starts in 2014, according to the database.

State law requires Charles Town to operate at least 220 racing days each year or to formally request approval from the state racing commission to reduce its schedule. The Humane Society of the United States said the length of the race year, and the larger number of horse starts, raises the risk of injuries to the thoroughbreds. “If they don't have enough horses, that's a problem,” Humane Society equine industry expert Keith Dane said. “Horses that would've retired run longer, perhaps with injuries, in order to fill these (races)."

Horse owners and track operators often must reach agreement before state regulators formally grant Charles Town permission to reduce its racing calendar each year.

Joe Funkhouser, head of the Charles Town Horsemen’s Benevolent and Protective Association, said a large majority of horsemen want a “sweet spot,” which he described as a racing calendar of less than 220 days that protects the health of the horses and the health of the local horse industry. He said a smaller minority of horsemen support racing a calendar closer to 220 days each year.

It is unnecessary for legislators to change law to reduce the 220-day race day requirement, Funkhouser said, because racing officials are empowered to make reductions each year based on weather incidents and shortages of horses.

“Safety of our horses is paramount," Funkhouser said. "Everybody’s livelihoods depend on the safety of these horses.”

Leslie Condon, a long horse trainer in Jefferson County, West Virginia, said the length of the race year is not contributing to a higher mortality rate. Condon, whose thoroughbred Lovin' It suffered a mortal injury in March 2015, said the quality of horses racing at Charles Town are no more predisposed to fatal injuries than others.

Condon said race horses are animals for whom special care is provided. "(My horse) lives in a padded room," she said. "He gets a bath every day. He gets groomed every day and a sponge bath every day. He has a vet who visits him on a regular basis. They get more care than if they were just standing in a field."

The West Virginia Racing Commission gave the I-Team this statement:

"We are concerned with the health and well-being of our thoroughbreds in West Virginia and are committed to ensuring that our rules and regulations meet the industry standard at all times for the protection of the animals involved. During the past few years, our rules have gone through an overhaul to include State Veterinarian pre-race exams for all thoroughbreds starting in a race; medication reforms to bring our rules up to industry standards; the collection of injury information by our state veterinarians into the Equine Injury Database; the use of an accredited testing lab; the regulation of the size of toe grabs on horse shoes worn by thoroughbreds; and measures related to rider safety. We have been annually re-evaluating our rules to make sure that we keep up with industry developments."

NOTE: A prior version of this report misidentified the name of a young foal at a trainer's stable in Jefferson County.

Reported by Scott MacFarlane, produced by Rick Yarborough, and shot and edited by Steve Jones and Jeff Piper.

Equine Slaughter
1980-2017 YTD[1]

	Horses Slaughtered in the U.S.				American Horses Exported for Slaughter			
Year	American Horses	Imported from Canada	Imported from Other Countries[2]	Total	To Canada	To Mexico	Total	Total American Horses Slaughtered
	(Numbers of Horses)							
	(4)-(3)-(2) (1)	(2)	(3)	(4)	(5)	(6)	(5) + (6) (7)	(1) + (7) (8)
1980	N/A	N/A	N/A	274, 500	N/A	N/A	N/A	N/A
1985	N/A	N/A	N/A	128,300	N/A	N/A	N/A	N/A
1990	344,385	250	1,065	345,700	68,086	N/A	N/A	N/A
1995	88,142	8,889	12,194	109,225	25,865	N/A	N/A	N/A
2000	40,324	3, 217	3,593	47, 134	24,315	N/A	N/A	**N/A**
2001	51,080	5,167	85	56,332	28,409	245	28,654	**79,734**
2002	40,407	1,843	62	42,312	25,219	774	25,993	**66,400**
2003	48,377	1,684	1	50,062	21,434	818	22,252	**70,629**
2004	60,078	6,105	0	66,183	19,725	4,114	23,839	**83,917**
2005	86,504	7,533	0	94,037	17,324	7,821	25,145	**111,649**
2006	101,123	3,776	0	104,899	21,709	11,080	32,789	**133,912**
2007	28,902	855	4	29,761	32,452	45,609	78,061	**106,963**
2008	0	0	0	0	42,232	56,731	98,963	**98,963**
2009	0	0	0	0	52,405	46,098	98,503	**98,503**
2010	0	0	0	0	53,803	52,862	106,665	**106,665**
2011	0	0	0	0	59,743	67,782	127,525	**127525**
2012					55,781	110,791	166,572	**166572**
2013					42,102	102,554	144,656	**144,656**
2014					40,410	105,375	145,785	**145,785**
2015					40,670	84,938	125,608	**125,608**
2016					29,472	78,724	108,196	108,196
2017					12,273	67,289	78,857	78,857
2018 YTD					3,394	26,576	29,970	29,970

Year to Date = Canada is 2 months behind

Other Countries include Germany, Mexico, Netherlands and the United Kingdom.

All 12,194 horses were imported from Mexico.

*To Canada, late-2009 to present, provided by Statistics Canada source

Sources - Col. (2)**:** Annual Data from USDA, Foreign Agricultural Service (FAS) "FAS Agricultural Import Aggregations and HS-10Digit Import Commities" Commodity Codes 0101901010 & 0101190010 (Live Horses for Immediate Slaughter), Weekly Year to Date data from USDA APHIS, "Canadian Live Animal Imports into the U.S. by Destination," weekly reports listed at http://www.ams.usda.gov/mnreports/WA_LS637.txt; Col. (3) USDA, Foreign Agricultural Service (FAS) "FAS Agricultural Import Aggregations and HS-10Digit Import Commities" Commodity Codes 0101901010 & 0101190010 (Live Horses for Immediate Slaughter); Col. (4) Annual Data from USDA NASS, "Equine Slaughter," query conducted at http://www.nass.usda.gov:8080/QuickStats/index2.jsp; Col. (5): Statistics Canada, Canadian International Merchandise Trade, Commodity Code 0101190010 & 0101900011 (010129 Horses, live, other than pure-bred breeding); Col. (6): USDA Market News Service, "US to Mexico Weekly Livestock Export Summary," weekly reports listed at http://www.ams.usda.gov/mnreports/AL_LS635.txt.

http://thehill.com/blogs/congress-blog/politics/390087-a-bipartisan-approach-to-protecting-racehorses

A bipartisan approach to protecting racehorses

BY MARTY IRBY, OPINION CONTRIBUTOR — 05/31/18 01:40 PM EDT 9
THE VIEWS EXPRESSED BY CONTRIBUTORS ARE THEIR OWN AND NOT THE VIEW OF THE HILL

54

© Getty Images

For centuries, we have respected and admired professional athletes for their agility, speed and endurance. We rightly view athletic competitions as tests of players' natural abilities—talents that have been refined through years of training, experience and intelligence. The same would be true for the animal athletes of horse racing, in a different era. However, according to a 2015 poll conducted by Penn Schoen Berland, 90 percent of bettors and 44 percent of the broader public associate horse racing with performance-enhancing drugs.

This means that the horse racing industry lags behind numerous esteemed professional sports programs that have all taken the steps needed to rid their competitions of illegal drugs and other forms of cheating.

Part of the problem for horse racing, unlike, say, the National Football League, is that no unifying regulatory system exists. Imagine if all 32 professional football teams had different sets of rules in each stadium. That is exactly the situation in each of the 38 state racing jurisdictions throughout the United States. Each one offers a unique set of regulations, allowing corrupt owners and trainers to move racehorses from one jurisdiction to another to avoid penalties or to enjoy more lenient oversight. State racing commissions allow various medications and differing levels of permissible medications. They also impose varying penalties for violations and use numerous, incongruous laboratories to test for the presence of drugs.
Uniform oversight and regulation of the industry are needed to stop unethical trainers and veterinarians from doping horses to improve their chances of winning. The Horseracing Integrity Act, H.R. 2651, can achieve this goal. This bipartisan legislation, introduced in the U.S. House of Representatives by Reps. Andy Barr (R-Ky.), and Paul Tonko (D-N.Y.), will establish a uniform set of rules, testing procedures and penalties.

H.R. 2651 would create a private, independent horse racing anti-doping authority, the Horseracing Anti-Doping and Medication Control Authority (HADA), responsible for developing and administering a nationwide anti-doping program for horse racing.

The authority would be governed by a board composed of the chief executive officer of the United States Anti-Doping Agency (USADA), which is the agency that monitors Olympic sports in the United States. It would also include six individuals from the USADA board, and six individuals selected by USADA who have demonstrated expertise in a variety of horse-racing areas. This new agency would be funded by the industry with no taxpayer funds or taxes on bettors.

According to the 2015 poll, 96 percent of horse racing fans and 83 percent of the public support USADA-led oversight of Thoroughbred racing.

This legislation is crucial to protect the animals and jockeys of an industry that history has shown will not regulate itself.

Overwhelming support for this measure exists among animal welfare groups such as the Humane Society of the United States and the Humane Society Legislative Fund. Backing for the reform is also growing throughout the racing industry where industry insiders are uniting in support of the bill's passage.

Supporters include the New York Racing Association, which operates Belmont Park and Saratoga Racetrack; Frank Stronach, founder of The Stronach Group, which owns several tracks including Pimlico Race Course, where the Preakness Stakes was just won by the Kentucky Derby winner, Justify; The Jockey Club; The Breeders' Cup; Keeneland; and the Water Hay Oats Alliance, which includes 65 racehorse trainers.

As we approach the Belmont Stakes, the last leg of the Triple Crown in Thoroughbred racing, with Justify seeking to become the second horse to win the title in 40 years, let's ask our members of Congress to step up and keep this sport alive by passing the Horseracing Integrity Act. The stakes are highest not for the owners, the trainers, the spectators, or the economy, but for the athletes themselves. We should see that, understand our obligations, and act upon them.

Marty Irby is the Senior Adviser at The Humane Society of the United States and Humane Society Legislative Fund in Washington, D.C.

https://www.horsetalk.co.nz/2018/06/10/american-racing-federal-oversight-hsus-boss/

American racing requires federal oversight, says HSUS boss

June 10, 2018 Horsetalk.co.nz 0 Comments

Spread the word


Kitty Block

The patchwork of horse racing rules across American states has been criticized by the acting head of the Humane Society of the United States, who says federal oversight is necessary.

Kitty Block, the organization's acting president and chief executive, says big events such as today's running of the Belmont Stakes, the third and final race in the Triple Crown series, may draw big crowds.

"But once the race ends and the tracks are empty again, the horse racing industry will find itself in a poor position, lagging behind in the popularity race," she writes in her blog, A Humane Nation.

"The drugging of horses by certain veterinarians and trainers to boost race performance and the continuing scandals surrounding the sale of racehorses to slaughter houses where they are turned into meat, have cast a cloud over the sport.

"Today, at most races other than the Triple Crown, horses run at tracks with increasingly empty bleachers occupied by an aging and shrinking fan base."

Block says the society has been working with stakeholders in the racing industry who want to prioritize animal welfare concerns, like widespread drugging, ending the slaughter of racehorses for human consumption overseas, and expanding second career opportunities.

Current efforts center on the passage of the Horseracing Integrity Act H.R.2651, a federal bill that focuses on medication reform and includes a ban on race-day medication.

“Despite its national and international scope, modern horse racing is still being conducted under outdated state-by-state drug and medication rules and this obsolete model is ripe for change.”

The bill, she says, will make all of the difference.

“There is a strong need for a federal law because unlike other sports, horse racing has no central regulatory body to provide oversight or to sanction those who flout rules.

“Each state’s racing commission determines what drugs are used and what penalties are meted out for violators, and the result is a patchwork of many different sets of rules.

“Many states have extremely permissive medication rules and a lax attitude toward those who break the laws.”

She says the routine drugging of horses to give them a leg up in competition has to end.

“We would not approve of this in any other sport, and we should not turn a blind eye to this practice in the horse racing industry.

“Too many horses have died in recent years as a result of widespread drugging and Congress needs to pass the Horseracing Integrity Act to ensure these abuses are outlawed once and for all.”

Block says that the responsible retiring of racehorses, ensuring them a happy and meaningful life at the end of their track careers, is an industry and owner responsibility.

“While too many horses still lack a sufficient safety net after their racing careers, we are encouraged by some of the industry initiatives for thoroughbred aftercare.

“And while there is still work to do, we are optimistic about the prospects for even better and more innovative programs for aftercare in the thoroughbred racing industry.”

She said the society applauds efforts to date by the thoroughbred racing community in this area. “We would encourage both the standardbred and quarter horse communities to follow the thoroughbred racing industry’s valuable example in this regard.”

She continues: “One principle unites the issues of drugging abuse and aftercare. These incredible equine athletes deserve to participate on a level playing field where their welfare both during and after their racing careers is a priority. And we must work together with every willing partner to ensure this worthy outcome.”

https://www.paulickreport.com/news/the-biz/backers-foes-of-horse-racing-integrity-act-to-testify-before-congress/

Backers, Foes Of Horseracing Integrity Act To Testify Before Congress

by Paulick Report Staff | 06.20.2018 | 9:04am

Five racing executives, two members of the House of Representatives and one animal welfare organization official have agreed to testify before a Congressional subcommittee on Friday to discuss the pros and cons of the Horseracing Integrity Act of 2017.

The House of Representatives' Committee on Energy and Commerce's Subcommittee on Digital Commerce and Consumer Protection will conduct the hearing on Friday, June 22, 2018, at 9:00 a.m. in 2123 Rayburn House Office Building. Invitations were issued by subcommittee chairman Robert Latta (R-OH).

Reps. Andy Barr (R-KY) and Paul Tonko (D-NY), co-chairs of the Congressional Horse Caucus and co-sponsors of the Horseracing Integrity Act, will testify, as will Kitty Block, acting president and CEO of the Humane Society of the United States, an animal welfare group that supports the legislation.

From the racing industry will come five individuals whose organizations have expressed support or opposition to the legislation that would put the regulation of medication policies and enforcement under the umbrella of the United State Anti-Doping Agency – an independent, non-governmental organization that oversees drug testing and enforcement for Olympic athletes and other sports. The legislation, as currently written, would also eliminate the use of all race-day medication.

Providing testimony in support of the bill will be Stuart S. Janney III, chairman of The Jockey Club, and Craig Fravel, CEO of the Breeders' Cup. Both organizations are members of the Coalition for Horse Racing Integrity.

Alan Foreman, chairman and CEO of the Thoroughbred Horsemen's Association, and Eric Hamelback, CEO of the National Horsemen's Benevolent and Protective Association, are expected to speak in opposition to the bill, along with Ed Martin, president of the Association of Racing Commissioners International, a trade association representing state regulators currently responsible for drug policies and enforcement.

New to the Paulick Report? Click here to sign up for our daily email newsletter to keep up on this and other stories happening in the Thoroughbred industry.

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Lane said he is dedicated to his horses and often works a 15-hour day to prepare and care for them. "A lot of devotion goes into these horses to make a little bit of money. Half the people who do it love the game." Lane said he was financially impacted by his ejection from the track. "I lost everything," Lane said.

"For someone to come in and accuse me of doing something that I didn't do and take everything from me, that's hard," he said. "But look where I am. I'm right back here."

The state racing commission's investigation said Lane made "false statements" that Diplomatic Gal had undergone a required pre-race workout the day the horse suffered the fatal injury. The investigation also cited a series of other injuries suffered Lane's horses in 2014 and said, "(Lane's track record) is of obvious and serious concern and must be considered in evaluating the ejection period in this case."

Though the West Virginia Racing Commission ejected Lane from Charles Town shortly after, the agency allowed him to return to his operations at the track in June 2015. By August 2015, another of Lane's horses, failed to complete the race and needed a van escort from the track, according to race records.

Charles Town managers said they strongly disagree with the agency's decision to allow Lane to resume his operations at the track. "His ejection from the track was curtailed," Zimny said. "He's back racing horses here. If it was up to us, unilaterally, it would not have happened."

In its statement to News4, the West Virginia Racing Commission said, "The West Virginia Racing Commission utilized the services of an independent party to conduct a full hearing on the matter." The agency confirmed the hearing recommended Lane be allowed to return June 25, 2015, to Charles Town. The agency's statement said, "After reviewing the transcripts of the hearing and recommended decision, the Commission voted to adopt the recommendation of the hearing examiners."

A database maintained by the Humane Society of the United States shows Charles Town stages among the most horse races in the nation. There were 13,610 horse starts at Charles Town in 2014, according to the database. A horse start is the departure of a thoroughbred from the starting gate at the track. Charles Town had almost double the number of starts of some high-profile U.S. race tracks, including Aqueduct and Belmont. Charles Town also had thousands more starts in 2014 than

major Maryland tracks, including Pimlico and Laurel. Only one track nationwide, Gulfstream Park in Florida, had a larger number of horse starts in 2014, according to the database.

State law requires Charles Town to operate at least 220 racing days each year or to formally request approval from the state racing commission to reduce its schedule. The Humane Society of the United States said the length of the race year, and the larger number of horse starts, raises the risk of injuries to the thoroughbreds. “If they don't have enough horses, that's a problem,” Humane Society equine industry expert Keith Dane said. “Horses that would've retired run longer, perhaps with injuries, in order to fill these (races)."

Horse owners and track operators often must reach agreement before state regulators formally grant Charles Town permission to reduce its racing calendar each year.

Joe Funkhouser, head of the Charles Town Horsemen’s Benevolent and Protective Association, said a large majority of horsemen want a “sweet spot,” which he described as a racing calendar of less than 220 days that protects the health of the horses and the health of the local horse industry. He said a smaller minority of horsemen support racing a calendar closer to 220 days each year.

It is unnecessary for legislators to change law to reduce the 220-day race day requirement, Funkhouser said, because racing officials are empowered to make reductions each year based on weather incidents and shortages of horses.

“Safety of our horses is paramount," Funkhouser said. "Everybody’s livelihoods depend on the safety of these horses.”

Leslie Condon, a long horse trainer in Jefferson County, West Virginia, said the length of the race year is not contributing to a higher mortality rate. Condon, whose thoroughbred Lovin' It suffered a mortal injury in March 2015, said the quality of horses racing at Charles Town are no more predisposed to fatal injuries than others.

Condon said race horses are animals for whom special care is provided. "(My horse) lives in a padded room," she said. "He gets a bath every day. He gets groomed every day and a sponge bath every day. He has a vet who visits him on a regular basis. They get more care than if they were just standing in a field."

The West Virginia Racing Commission gave the I-Team this statement:

"We are concerned with the health and well-being of our thoroughbreds in West Virginia and are committed to ensuring that our rules and regulations meet the industry standard at all times for the protection of the animals involved. During the past few years, our rules have gone through an overhaul to include State Veterinarian pre-race exams for all thoroughbreds starting in a race; medication reforms to bring our rules up to industry standards; the collection of injury information by our state veterinarians into the Equine Injury Database; the use of an accredited testing lab; the regulation of the size of toe grabs on horse shoes worn by thoroughbreds; and measures related to rider safety. We have been annually re-evaluating our rules to make sure that we keep up with industry developments."

NOTE: A prior version of this report misidentified the name of a young foal at a trainer's stable in Jefferson County.

Reported by Scott MacFarlane, produced by Rick Yarborough, and shot and edited by Steve Jones and Jeff Piper.

Equine Slaughter
1980-2017 YTD[1]

	Horses Slaughtered in the U.S.				American Horses Exported for Slaughter			
Year	American Horses	Imported from Canada	Imported from Other Countries[2]	Total	To Canada	To Mexico	Total	Total American Horses Slaughtered
	(Numbers of Horses)							
	(4)-(3)-(2) (1)	(2)	(3)	(4)	(5)	(6)	(5) + (6) (7)	(1) + (7) (8)
1980	N/A	N/A	N/A	274, 500	N/A	N/A	N/A	N/A
1985	N/A	N/A	N/A	128,300	N/A	N/A	N/A	N/A
1990	344,385	250	1,065	345,700	68,086	N/A	N/A	N/A
1995	88,142	8,889	12,194	109,225	25,865	N/A	N/A	N/A
2000	40,324	3, 217	3,593	47, 134	24,315	N/A	N/A	**N/A**
2001	51,080	5,167	85	56,332	28,409	245	28,654	**79,734**
2002	40,407	1,843	62	42,312	25,219	774	25,993	**66,400**
2003	48,377	1,684	1	50,062	21,434	818	22,252	**70,629**
2004	60,078	6,105	0	66,183	19,725	4,114	23,839	**83,917**
2005	86,504	7,533	0	94,037	17,324	7,821	25,145	**111,649**
2006	101,123	3,776	0	104,899	21,709	11,080	32,789	**133,912**
2007	28,902	855	4	29,761	32,452	45,609	78,061	**106,963**
2008	0	0	0	0	42,232	56,731	98,963	**98,963**
2009	0	0	0	0	52,405	46,098	98,503	**98,503**
2010	0	0	0	0	53,803	52,862	106,665	**106,665**
2011	0	0	0	0	59,743	67,782	127,525	**127525**
2012					55,781	110,791	166,572	**166572**
2013					42,102	102,554	144,656	**144,656**
2014					40,410	105,375	145,785	**145,785**
2015					40,670	84,938	125,608	**125,608**
2016					29,472	78,724	108,196	108,196
2017					12,273	67,289	78,857	78,857
2018 YTD					3,394	26,576	29,970	29,970

Year to Date = Canada is 2 months behind

Other Countries include Germany, Mexico, Netherlands and the United Kingdom.

All 12,194 horses were imported from Mexico.

*To Canada, late-2009 to present, provided by Statistics Canada source

Sources - Col. (2)**:** Annual Data from USDA, Foreign Agricultural Service (FAS) "FAS Agricultural Import Aggregations and HS-10Digit Import Commities" Commodity Codes 0101901010 & 0101190010 (Live Horses for Immediate Slaughter), Weekly Year to Date data from USDA APHIS, "Canadian Live Animal Imports into the U.S. by Destination," weekly reports listed at http://www.ams.usda.gov/mnreports/WA_LS637.txt; Col. (3) USDA, Foreign Agricultural Service (FAS) "FAS Agricultural Import Aggregations and HS-10Digit Import Commities" Commodity Codes 0101901010 & 0101190010 (Live Horses for Immediate Slaughter); Col. (4) Annual Data from USDA NASS, "Equine Slaughter," query conducted at http://www.nass.usda.gov:8080/QuickStats/index2.jsp; Col. (5): Statistics Canada, Canadian International Merchandise Trade, Commodity Code 0101190010 & 0101900011 (010129 Horses, live, other than pure-bred breeding); Col. (6): USDA Market News Service, "US to Mexico Weekly Livestock Export Summary," weekly reports listed at http://www.ams.usda.gov/mnreports/AL_LS635.txt.

http://thehill.com/blogs/congress-blog/politics/390087-a-bipartisan-approach-to-protecting-racehorses

A bipartisan approach to protecting racehorses

BY MARTY IRBY, OPINION CONTRIBUTOR — 05/31/18 01:40 PM EDT 9
THE VIEWS EXPRESSED BY CONTRIBUTORS ARE THEIR OWN AND NOT THE VIEW OF THE HILL

54

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For centuries, we have respected and admired professional athletes for their agility, speed and endurance. We rightly view athletic competitions as tests of players' natural abilities—talents that have been refined through years of training, experience and intelligence. The same would be true for the animal athletes of horse racing, in a different era. However, according to a 2015 poll conducted by Penn Schoen Berland, 90 percent of bettors and 44 percent of the broader public associate horse racing with performance-enhancing drugs.

This means that the horse racing industry lags behind numerous esteemed professional sports programs that have all taken the steps needed to rid their competitions of illegal drugs and other forms of cheating.

Part of the problem for horse racing, unlike, say, the National Football League, is that no unifying regulatory system exists. Imagine if all 32 professional football teams had different sets of rules in each stadium. That is exactly the situation in each of the 38 state racing jurisdictions throughout the United States. Each one offers a unique set of regulations, allowing corrupt owners and trainers to move racehorses from one jurisdiction to another to avoid penalties or to enjoy more lenient oversight. State racing commissions allow various medications and differing levels of permissible medications. They also impose varying penalties for violations and use numerous, incongruous laboratories to test for the presence of drugs.
Uniform oversight and regulation of the industry are needed to stop unethical trainers and veterinarians from doping horses to improve their chances of winning. The Horseracing Integrity Act, H.R. 2651, can achieve this goal. This bipartisan legislation, introduced in the U.S. House of Representatives by Reps. Andy Barr (R-Ky.), and Paul Tonko (D-N.Y.), will establish a uniform set of rules, testing procedures and penalties.

H.R. 2651 would create a private, independent horse racing anti-doping authority, the Horseracing Anti-Doping and Medication Control Authority (HADA), responsible for developing and administering a nationwide anti-doping program for horse racing.

The authority would be governed by a board composed of the chief executive officer of the United States Anti-Doping Agency (USADA), which is the agency that monitors Olympic sports in the United States. It would also include six individuals from the USADA board, and six individuals selected by USADA who have demonstrated expertise in a variety of horse-racing areas. This new agency would be funded by the industry with no taxpayer funds or taxes on bettors.

According to the 2015 poll, 96 percent of horse racing fans and 83 percent of the public support USADA-led oversight of Thoroughbred racing.

This legislation is crucial to protect the animals and jockeys of an industry that history has shown will not regulate itself.

Overwhelming support for this measure exists among animal welfare groups such as the Humane Society of the United States and the Humane Society Legislative Fund. Backing for the reform is also growing throughout the racing industry where industry insiders are uniting in support of the bill's passage.

Supporters include the New York Racing Association, which operates Belmont Park and Saratoga Racetrack; Frank Stronach, founder of The Stronach Group, which owns several tracks including Pimlico Race Course, where the Preakness Stakes was just won by the Kentucky Derby winner, Justify; The Jockey Club; The Breeders' Cup; Keeneland; and the Water Hay Oats Alliance, which includes 65 racehorse trainers.

As we approach the Belmont Stakes, the last leg of the Triple Crown in Thoroughbred racing, with Justify seeking to become the second horse to win the title in 40 years, let's ask our members of Congress to step up and keep this sport alive by passing the Horseracing Integrity Act. The stakes are highest not for the owners, the trainers, the spectators, or the economy, but for the athletes themselves. We should see that, understand our obligations, and act upon them.

Marty Irby is the Senior Adviser at The Humane Society of the United States and Humane Society Legislative Fund in Washington, D.C.

https://www.horsetalk.co.nz/2018/06/10/american-racing-federal-oversight-hsus-boss/

American racing requires federal oversight, says HSUS boss

June 10, 2018 Horsetalk.co.nz 0 Comments

Spread the word


Kitty Block

The patchwork of horse racing rules across American states has been criticized by the acting head of the Humane Society of the United States, who says federal oversight is necessary.

Kitty Block, the organization's acting president and chief executive, says big events such as today's running of the Belmont Stakes, the third and final race in the Triple Crown series, may draw big crowds.

"But once the race ends and the tracks are empty again, the horse racing industry will find itself in a poor position, lagging behind in the popularity race," she writes in her blog, A Humane Nation.

"The drugging of horses by certain veterinarians and trainers to boost race performance and the continuing scandals surrounding the sale of racehorses to slaughter houses where they are turned into meat, have cast a cloud over the sport.

"Today, at most races other than the Triple Crown, horses run at tracks with increasingly empty bleachers occupied by an aging and shrinking fan base."

Block says the society has been working with stakeholders in the racing industry who want to prioritize animal welfare concerns, like widespread drugging, ending the slaughter of racehorses for human consumption overseas, and expanding second career opportunities.

Current efforts center on the passage of the Horseracing Integrity Act H.R.2651, a federal bill that focuses on medication reform and includes a ban on race-day medication.

“Despite its national and international scope, modern horse racing is still being conducted under outdated state-by-state drug and medication rules and this obsolete model is ripe for change.”

The bill, she says, will make all of the difference.

“There is a strong need for a federal law because unlike other sports, horse racing has no central regulatory body to provide oversight or to sanction those who flout rules.

“Each state’s racing commission determines what drugs are used and what penalties are meted out for violators, and the result is a patchwork of many different sets of rules.

“Many states have extremely permissive medication rules and a lax attitude toward those who break the laws.”

She says the routine drugging of horses to give them a leg up in competition has to end.

“We would not approve of this in any other sport, and we should not turn a blind eye to this practice in the horse racing industry.

“Too many horses have died in recent years as a result of widespread drugging and Congress needs to pass the Horseracing Integrity Act to ensure these abuses are outlawed once and for all.”

Block says that the responsible retiring of racehorses, ensuring them a happy and meaningful life at the end of their track careers, is an industry and owner responsibility.

“While too many horses still lack a sufficient safety net after their racing careers, we are encouraged by some of the industry initiatives for thoroughbred aftercare.

“And while there is still work to do, we are optimistic about the prospects for even better and more innovative programs for aftercare in the thoroughbred racing industry.”

She said the society applauds efforts to date by the thoroughbred racing community in this area. “We would encourage both the standardbred and quarter horse communities to follow the thoroughbred racing industry’s valuable example in this regard.”

She continues: “One principle unites the issues of drugging abuse and aftercare. These incredible equine athletes deserve to participate on a level playing field where their welfare both during and after their racing careers is a priority. And we must work together with every willing partner to ensure this worthy outcome.”

https://www.paulickreport.com/news/the-biz/backers-foes-of-horse-racing-integrity-act-to-testify-before-congress/

Backers, Foes Of Horseracing Integrity Act To Testify Before Congress

by Paulick Report Staff | 06.20.2018 | 9:04am

Five racing executives, two members of the House of Representatives and one animal welfare organization official have agreed to testify before a Congressional subcommittee on Friday to discuss the pros and cons of the Horseracing Integrity Act of 2017.

The House of Representatives' Committee on Energy and Commerce's Subcommittee on Digital Commerce and Consumer Protection will conduct the hearing on Friday, June 22, 2018, at 9:00 a.m. in 2123 Rayburn House Office Building. Invitations were issued by subcommittee chairman Robert Latta (R-OH).

Reps. Andy Barr (R-KY) and Paul Tonko (D-NY), co-chairs of the Congressional Horse Caucus and co-sponsors of the Horseracing Integrity Act, will testify, as will Kitty Block, acting president and CEO of the Humane Society of the United States, an animal welfare group that supports the legislation.

From the racing industry will come five individuals whose organizations have expressed support or opposition to the legislation that would put the regulation of medication policies and enforcement under the umbrella of the United State Anti-Doping Agency – an independent, non-governmental organization that oversees drug testing and enforcement for Olympic athletes and other sports. The legislation, as currently written, would also eliminate the use of all race-day medication.

Providing testimony in support of the bill will be Stuart S. Janney III, chairman of The Jockey Club, and Craig Fravel, CEO of the Breeders' Cup. Both organizations are members of the Coalition for Horse Racing Integrity.

Alan Foreman, chairman and CEO of the Thoroughbred Horsemen's Association, and Eric Hamelback, CEO of the National Horsemen's Benevolent and Protective Association, are expected to speak in opposition to the bill, along with Ed Martin, president of the Association of Racing Commissioners International, a trade association representing state regulators currently responsible for drug policies and enforcement.

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https://www.thoroughbredracing.com/articles/charles-town-classic-fiasco-response-takes-racing-wrong-direction/

Charles Town Classic fiasco: this response takes racing in the wrong direction

Joe Gorajec | FEBRUARY 25, 2018 |
7 Comments

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Star attraction: Game On Dude winning the $1.2 million Charles Town Classic in 2013. Photo: Coady Photography

This is a case where the cure is worse than the disease. Legislation is currently making its way through the statehouse in West Virginia that would expand its racing commission from three to five members. More importantly, it would mandate that new appointees be "capable and experienced in fields of knowledge relevant to racing". On February 6, 2018, the bill passed in the Senate with a vote of 30-0.

The legislation (aka Senate Bill 393) is likely a well-intentioned response to the recent fiasco of the racing commission's attempt to gut the Charles Town Classic. The state's only Graded race.

On January 23, 2018, in a decision that brought shame and ire on state's regulators, the commission voted to cap the Classic's purse at $300,000.

Its advertised value had been $1.2 million. The immediate backlash from the industry, and, most importantly, West Virginia Governor Jim Justice, who made his displeasure public, virtually assured the issue would be revisited.

Self-regulation

The writing was on the wall two weeks later, when the WV racing commission met to rescind its previous decision.

Evidently, enough damage had been done to prod legislative action.

The increase from three to five members is immaterial. The qualifications mandate, which might look good on paper, is problematic in real life.

The bill, if passed as is, would amount to self-regulation. Which is bad. Very bad. But, first let's look at what the bill says.

Specifically, the bill states, *"Individuals appointed to the commission shall be persons who, by reason of previous training and experience, can be classified as capable and experienced in fields of knowledge relevant to racing."*

One person, qualified as above, would be appointed as a representative for each of the following:

- Tourism
- Thoroughbred horse racing
- Greyhound racing
- Casino (with either race track management or casino experience)
- Veterinary

Two issues are worth raising.

First: potential conflicts of interest. Nothing in the bill prohibits the appointment of a commissioner who has a financial relationship with a person or entity he or she regulates.

For example, can the veterinarian appointed to the commission work on horses racing at the track? Is the casino representative going to be an official of the track or casino?

I am familiar with the Horse Racing Integrity Act of 2017. All parties involved in its drafting of that federal legislation insisted that authority to regulate be entrusted to an independent body. The West Virginia bill takes the industry in the opposite direction.

Each state's pari-mutuel statute across the country varies on whether to allow individuals who are "in the business" to serve as commissioners. Regardless of prohibitions against such situations, the Governor controls this issue by the appointments that are made.

The other issue is having a commission where the majority represents certain segments of the industry.

No commissioner should represent ***any*** segment of the industry. Commissioners should represent the public in the state they serve. They are regulators whose duty requires them to protect the integrity of the sport and the safety of the participants – human and equine. Every commissioner should be mindful to consider the health and prosperity of the industry at-large.

It's only human nature, however, to view the world from the perspective of your experience. When a person is placed on a commission to represent a special interest, that's what he or she will do.

I once had had a commissioner in Indiana, who had ties to the Standardbred industry, declare on a split vote something along the lines of – *"I'm a Standardbred guy and I'm voting for the Standardbreds"*. What an embarrassment. This was supposedly on impartial panel. Even the Standardbred horsemen at that meeting were embarrassed.

It's been my experience as a horse racing regulator in Indiana for 25 years, that, all things being equal, it is best to have commissioners without ties to the industry. I've know a handful of "in-the-business" commissioners who have served their state well – with honor, dignity and objectivity.

But these folks are the exception, not the rule.

It is interesting to note that the commissioner who initiated the purse cut to the Charles Town Classic, Ken Lowe Jr., is certainly qualified under the Senate Bill 393 to retain his seat on the commission. He is a former racehorse owner who served as the HBPA President at Charles Town Race Track.

It's one thing to allow conflicted or potentially biased individuals to serve on a racing commission. It's another to mandate it.

That's what Senate Bill 393 does.

Joe Gorajec has spent his entire adult life in the racing industry and served as the executive director of the Indiana Horse Racing Commission for 25 years (1990-2015). He is also a former chairman of the North American regulators' trade association, the Association of Racing Commissioners International (2008). Now semi-retired, he spends his time consulting, writing and gardening at his central Indiana home.

https://www.paulickreport.com/news/ray-s-paddock/combating-culture-cheating/

Combating A Culture Of Cheating: A Matter Of Trust

by Joe Gorajec | 01.18.2017 | 8:50pm

(First in a two-part series.)

Horse racing has a culture of cheating.

Its problem is drugs. It's what the public calls doping. The methods differ, as do the drugs.

At one end of the spectrum, horsemen and veterinarians inject horses on race day with a wide variety of drugs or other foreign substances.

This practice is one of racing's dirty little secrets, although it's no secret to those who work in the stable area of a racetrack.

There exists a thick, bright line in racing regulation known as the 24-hour rule. This rule prohibits the administration of any drug or foreign substance, other than the anti-bleeder medication Salix (furosemide), within 24 hours of a horse's race. A few states have specific exceptions. This bright line is crossed with such regularity that its practitioners have become blind to its existence.

Horsemen rationalize this cheating by convincing themselves that they are just “helping” the horse. Many of the race day injections are to manage pain, mitigate bleeding, or calm a fractious horse. Several of these drugs are endogenous to the horse and go undetected in post-race testing. The “helping” of the horse is code for “it's not cheating if you don't get caught.”

An example of this culture run amok is the investigation and prosecution of veterinarians and horse trainers at Penn National racetrack by the United States Attorney's Office for the Middle District of Pennsylvania.

On March 27, 2015, The U.S. Attorney's Office issued a press release announcing that four veterinarians had been criminally charged (and had agreed to plead guilty) to conspiracy to unlawfully administer drugs to race horses. The release succinctly describes this activity as follows:

According to the charges, trainers allegedly placed orders for drugs and the defendants after administering the drugs, backdated the billing records to avoid detection. The defendants allegedly submitted false veterinarian treatment reports to the State Horse Racing Commission omitting from those reports any reference to the drugs administered to horses at the track on race day. The filing of these reports and the backdating of billing records were, allegedly, to further the conspiracy by concealing the illegal activity. These acts had the potential to defraud other owners and trainers whose horses were entered in the same race and defrauded the betting public as well.

This type of activity has been common practice on racetracks for decades.

A deterrent to this type of routine race day cheating exists in a national model rule requiring the administration of Salix by a third party. Under the model rule, a veterinarian employed or contacted by the state racing commission or the racetrack administers Salix. The rule is designed for the express purpose of keeping practicing veterinarians (who work for the trainer) out of the horse's stall on race day. Third-party

Salix programs are effective in deterring widespread corrupting influences. It will not, however, stop anyone determined to get an edge on race day.

The status of the third-party Salix model rule is found on the website of the Racing Medication and Testing Consortium (RMTC): 18 states have adopted, 16 states have not.

Far more malevolent and injurious to the sport is what is likely occurring at the other end of the spectrum. That is the use of sophisticated performance enhancing drugs (PEDs) which taint the upper echelon of the sport. The methods for cheating at this level often do not involve simply sneaking into a stall on race day with an illicit drug in a syringe.

Best horses, best races

When it comes to major races, prominent Thoroughbred owner Bill Casner said, “I'll promise you that there will be some horses helped with PEDs (performance enhancing drugs).

Bill and Susan Casner

“It would be incredibly naïve for anyone to think that this (PEDs) does not exist in our game. And especially at the high end because the high end is where all the money is at,” said Casner.

The “high end” to which Casner refers consists of approximately 450 Graded stakes races which are held annually at various tracks. These races make up less than two percent of the approximately 40,000 Thoroughbred races conducted annually. The purses for the Graded stakes, however, account for over $150 million, or 15 percent, of the $1 billion distributed annually.

Casner's path to the pinnacle of the sport is not well traveled. From galloping horses in the early 1960s at Sunland Park in New Mexico, to hoisting the Kentucky Derby trophy in the winner's circle in 2010 as the co-owner of Super Saver, he has witnessed the sport from the inside as few others ever have. His most memorable score, however, did not occur on U.S. soil. In 2009, his WinStar Farm homebred gelding, Well Armed, romped to a 14-length victory in the $6-million Dubai World Cup.

A member of The Jockey Club and the Water Hay Oats Alliance (WHOA), and former chairman of the Thoroughbred Owners and Breeders Association (TOBA), Casner has long been an advocate for clean racing.

Casner claims that a small but significant improvement in performance from illicit drugs is sufficient to drive a trainer to cheat. Especially if they believe their fellow trainers are likely doing the same.

At the elite level of the sport, the incentives to cheat are at their highest, and the financial incentives go well beyond the traditional trainer's purse percentage.

“There is too much difference in the amount of money between a Grade I and a Grade II horse. Grade I horses are stallions. Grade II horses are regional stallions. And Grade III horses stand in [minor state-bred programs],” said Casner.

Nine of the top 10 stallions on *Blood-Horse* magazine's 2016 General Sires List won at least one Grade I stakes during their racing career; 2017 stud fees for these nine horses range from $60,000 to $300,000.

“The share values and the breeding rights that trainers receive become these huge portfolios for them. And this is where they really make their money,” said Casner.

“If EPO can give a horse two or three lengths extra – that is astronomical,” said Casner. “How many races are lost by a nose? How many races are lost by a head? A neck? A length? A length-and-a-half? Two lengths?”

“Performance enhancing drugs work. They make already great athletes, human or animal, even greater,” said Jeff Novitzky.

A matter of trust

Few people in the world have the gravitas and insight to opine on the culture of cheating in sports as does Jeff Novitzky. Once referred to by *TIME* magazine as the Eliot Ness of baseball's “steroid era,” Novitzky has been on the frontline of exposing the cheating of fallen icons such as Barry Bonds, Roger Clemens, Marion Jones, Tim Montgomery, and Lance Armstrong.

Jeff Novitzky

Novitzky served as a federal agent for 15 years with the IRS Criminal Investigations Division, followed by seven years as a special agent for the Food and Drug Administration. He is now the vice president of Athlete Health and Performance with the Ultimate Fighting Championship (UFC), the world's largest mixed-martial arts fight promotion.

Novitzky spoke at the 2016 Jockey Club Round Table Conference in Saratoga Springs, N.Y. One of the most salient takeaways from his presentation involved his personal interaction with users of performance enhancing drugs.

Speaking of the numerous investigations he conducted, Novitzky said, “Throughout those investigations, I got to interview 150 to 200 high profile professional athletes who chose to use performance enhancing drugs. In addition to asking them about where they got the drugs, how they paid for them, and how they were distributed, I always took the opportunity to ask them why they chose to use. It wasn't anything special about me, but I was in a position and they were in a position to be compelled to tell me the truth. In fact, we prosecuted several athletes for not telling the truth.

“So I think in the majority of those 150, 200 conversations I got the truth, and I always took the opportunity to ask, ‘Why did you choose to use performance enhancing drugs? What led you down that path?'

“And the answer I got an overwhelming majority of the time, it came down to one word, and that word was trust.”

Novitzky added: “They said, ‘I didn't trust that my teammates weren't using. I didn't trust that my opponents weren't using, and maybe, most importantly, I didn't trust that my sport's governing bodies cared enough because of the weakness of the program or in some cases total lack thereof.'”

A report titled *Stakeholder Input*, released November 2016 by the Association of Racing Commissioners International, contains a survey in which the issue of horsemen's “trust” is addressed.

In response to the statement *“Doping with designer drugs is rampant,”* 58.1 percent either totally agreed or somewhat agreed. By nearly an identical margin, 57.2 percent of the respondents indicated they either totally or somewhat agreed with the statement *“Most people I know cheat.”*

If horsemen's trust in the effectiveness of their anti-doping program is the determining factor that drives cheating, the racing industry has reason for alarm.

A lack of will

Horse racing in the U.S., to a large degree, is a sport that abides a culture of cheating.

The unwillingness of regulatory bodies to implement common sense deterrents has led to, in many states, risk-free cheating. A clear-cut example is out-of-competition testing.

Out-of-competition testing occurs days, weeks, or months before a horse's race, or between races. Its goal is to determine if horses are training on prohibited drugs that can enhance performance on race day.

Blood doping drugs like Epogen (EPO) cause the body to produce additional blood cells that allow the athlete – horse or human – to increase their oxygen carrying capacity. The drug can only be detected for approximately three days after administration. The performance-enhancing effects will last up to 120 days – which is the life span of a red blood cell.

Many anabolic steroids are like blood doping drugs in that the performance enhancing effects far exceed the short time frame of detection.

States with little or no out-of-competition testing have invited their horsemen to cheat with impunity.

In November 2007, *Blood-Horse* published an op-ed column I wrote on out-of-competition testing. At the time of publication over a year had passed since the development of a method to test for the presence of the blood-doping agent Epogen. Only six states had moved forward to deter and detect this emerging threat by implementing out-of-competition testing.

In the commentary, I hypothesized why the industry had not moved more quickly.

Would some track owners prefer not to endure the inevitable publicity of a successful trainer charged with blood doping? Would some horsemen prefer to not be

inconvenienced by the thought of testing anytime, anywhere, without notice? Are some racing commissions paralyzed by institutional inertia?

Now, 10 years later, we know the answers to all these questions are … yes.

In 2014, of the top 20 states ranked by the number of Thoroughbred races run, 15 conducted little or no out-of-competition testing. These 15 states account for almost two-thirds of all races. Our international counterparts are averaging 10 percent of their testing from out-of-competition sampling, while the U.S. is conducting only 1 percent.

The racing industry's assertion that this failure is due to a lack of funding is disingenuous.

For example, in Indiana in 2015, over 10 percent of the testing for the 120-day Thoroughbred and Quarter horse race meet at Indiana Grand originated from out-of-competition samples. All samples were analyzed for blood-doping agents, a broad spectrum of anabolic steroids, and repartitioning drugs (such as ractopamine and zilpaterol). Samples were taken from horses stabled at the track or at training centers and farms.

The total cost of this program, including sampling and testing, was less than $50,000. To place this in perspective, the cost is less than two purses for maiden special weight races, which at Indiana Grand in 2015 were $32,000 each.

Although Indiana's program is funded by the racing commission, it is also a reasonable expense to be borne by any racetrack or horsemen's association intent on protecting the integrity of its racing program.

The UFC's Novitzky says horse racing's out-of-competition program is a “green light” for cheaters.

When asked what would happen if horse racing's out-of-competition program was applied to human athletics, Novitzky said athletes “would be enhanced to the gills.”

—

Thursday: In the second of this two-part series, Gorajek examines the limitations and challenges of testing laboratories, weaknesses of the current state regulatory system, and a possible solution going forward.

Joe Gorajec served as the executive director of the Indiana Horse Racing Commission for 25 years (1990-2015). He is also a former chairman of the Association of Racing Commissioners International (2008).

This entry was posted in Ray's Paddock and tagged american graded stakes committee, anabolic steroids, association of racing commissioners international, Bill Casner, drug testing, drugs in horse racing, epogen, furosemide, Indiana Grand, Indiana Horse Racing Commission, jeff novitzky, lasix, major league baseball, out of competition drug testing, PEDs, penn national indictments, performance enhancing drugs, race day medications, racing medication and testing consortium, rmtc, salix, steroid era, The Jockey Club, Thoroughbred Owners and Breeders

Association, UFC, Ultimate Fighting Championship, Water Hay Oats Alliance, WHOA by Joe Gorajec. Bookmark the permalink.

https://www.cbsnews.com/news/eight-belles-death-sparks-controversy/

CBS/AP May 5, 2008, 7:26 AM

Eight Belles' Death Sparks Controversy

Kentucky Derby: Is horse racing now facing an image crisis?

With the memory of Barbaro still fresh, Eight Belles' catastrophic breakdown Saturday put increasing focus on a sport already trying to overcome a decline in popularity.

Her death has raised thorny issues about the whole thoroughbred industry, including track safety, whether fillies should be allowed to run against colts, and whether horses are bred too much for speed and not for soundness.

Congressman Ed Whitield of Kentucky, who is trying to toughen regulation of horseracing, told **CBS News correspondent Chip Reid** that breakdowns are far more common than people think, and are on the rise. Whitfield said that one reason for the rise is that the big money is not in racing horses anymore, it's in breeding them.

"These horses really are expendable commodities," Whitfield said. "You want to get the most out of them for a short period of time, and hopefully they are good enough to go into breeding."

A prominent animal rights group got involved Sunday, too, criticizing Eight Belles' jockey for whipping the horse and saying the second-place prize should be revoked.

But to horse people, it wasn't all that simple.

"To make it safer, don't race the horses, don't train them, then they'll live good lives out on the farm," Big Brown trainer Rick Dutrow Jr. said.

"But you have to train them for races, you have to run them and that's where the problems start to set in. They have to be asked to run and sometimes in a particular minute, they're asked to run when they're not ready to give it and then it hurts."

While Big Brown's bid to become the first Triple Crown winner in 30 years will certainly gain momentum in the next couple of weeks, Eight Belles and the sight of fans crying in the stands remained a focal point Sunday.

"Filly's Death Casts Shadow over Kentucky Derby," read The New York Times.

"Tragedy mars Kentucky Derby as the only filly dies after race," the Los Angeles Times' Web site said.

Churchill Downs officials were unsure whether there had been a fatality in the Kentucky Derby. Superintendent Butch Lehr said there hadn't been one in his 41 years at the track.

The death of Eight Belles may have been rare because it occurred well after the finish line, but it's just the latest trauma to happen at a major race on national television.

Two years ago, Derby winner Barbaro shattered his fight rear leg at the start of the Preakness, with more than 100,000 people gasping at the site of the undefeated colt in distress as he was led into an equine ambulance. Barbaro was euthanized eight months later after developing laminitis as a result of the injuries.

Dr. Dean Richardson, the veterinary surgeon who tried valiantly to save Barbaro, told **CBS' *The Early Show*** that Eight Belles' injuries were very different from Barbaro's.

"It is extraordinarily for a racehorse to break down the way Eight Belles did after the race is finished," Richardson said.

Eight Belles suffered fractures of both feet. In the left foot, the fracture was so severe it tore through the skin.

"A horse can get around on three legs temporarily. It's impossible for a horse to get around on just its hind legs," Richardson said.

Now, there are more questions about track safety.

Barbaro's demise helped push forward the installation of synthetic surfaces to replace traditional dirt tracks at several tracks, including Keeneland, Santa Anita, Arlington Park, Hollywood Park, Golden Gate Fields, Del Mar, Turfway and Presque Isle. A new on-track injury reporting program seems to indicate the surface is having the desired effect.

Reports by veterinarians at 34 tracks across the country between June 2007 and early this year showed synthetic tracks averaged 1.47 fatalities per 1,000 starts, compared with 2.03 fatalities per 1,000 starts for horses that ran on dirt.

But not everyone is convinced.

"This is a very big issue and needs to be discussed," two-time Derby winning trainer Nick Zito said. "You're changing the whole game. Big Brown ran on dirt yesterday, he's going for history. You can't tell me the Polytrack is history. It's not yet, there isn't enough data yet."

That's not saying Zito and other horsemen are not interested in making racetracks safer for both horses and jockeys.

"If you told me, `Look, we have a device that these horses can run on pillows and never get hurt the rest of lives,' I'd say, `Where do I sign?'" Zito said. "There's injuries on the Polytrack, too. Now you see why I'm saying it's a big issue."

While breakdowns always have been a part of racing, there has been more of an outcry lately calling for drastic action.

People for the Ethical Treatment of Animals (PETA) issued a statement Sunday calling for the suspension of Eight Belles jockey Gabriel Saez. The group also asked for the "revocation of the second place prize."

Saez was riding in his first Kentucky Derby when Eight Belles broke both front ankles while galloping out a quarter-mile past the finish line.

"What we really want to know, did he feel anything along the way?" PETA spokeswoman Kathy Guillermo said. "If he didn't then we can probably blame the fact that they're allowed to whip the horses mercilessly."

A call to the jockeys' room at Delaware Park, where Saez raced on Sunday, went unanswered.

The Kentucky state racing stewards make decisions on suspensions, but there is no racing at Churchill Downs until Wednesday. At that time, the stewards could review a tape of the race if a formal request is made.

Eight Belles trainer Larry Jones disputed any suggestion that his horse had no business taking on the boys.

"It wasn't that, it wasn't the distance, it wasn't a big bumping match for her, she never got touched," he said. "She passed all those questions ... with flying colors. The race was over, all we had to do was pull up, come back and be happy. It just didn't happen."

On Sunday morning, Jones stood next to his Kentucky Oaks-winning filly, Proud Spell, receiving condolences from friends and fellow trainers.

"Got here at 5 a.m.," Jones said. "Got to go on. It's hard, but it's what we do."

Just then, Barbaro's trainer Michael Matz drove past Jones' barn stopped his car and rolled down the window. On Friday, Matz watched another one of his horses, Chelokee, suffer a life-threatening injury in the Alysheba Stakes. He had just returned from Lexington, where the horse was set for surgery Monday to fuse his injured ankle.

"Sorry, Larry," Matz said.

"I know you know what it's like, thank you," Jones said. "How's yours doin'?"

"Doing good, they're going to operate tomorrow," Matz said.

Dutrow was still basking in Big Brown's victory, well aware that an injury can strike at any time.

"No matter what happens, you're always going to see horses break down on the track," he said. "That is part of this game. It's a very sad part of the game, but you have to go through it.

"For people coming out to the track and seeing that, it's got to make them think, `Man, why would I want to go out there and see that happen to a horse?"' he said. "It's got to be very disappointing to anyone who loves horses."

https://www.paulickreport.com/news/ray-s-paddock/gorajec-barr-tonko-bill-only-path-toward-uniformity/

Gorajec: Barr-Tonko Bill Only Path Toward Uniformity

by Joe Gorajec | 10.15.2015 | 7:57am

The following commentary was written by Joe Gorajec, who on Saturday was relieved of his job as executive director of the Indiana Horse Racing Commission (IHRC), a position he held for nearly 25 years.

The commentary, which Gorajec intended for publication when written, was shared with Indiana commissioners in August with a clearly stated caveat that it represented his personal viewpoint, and not that of the IHRC or its individual members.

Gorajec was advised, in no uncertain terms, that he was not to have the article published.

Having been the executive director of the Indiana Horse Racing Commission since 1990, I have been on the frontline of regulatory policy and enforcement, both in Indiana and nationally, for the past 25 years.

During this time I have seen substantial efforts – with mixed results – to improve uniformity in drug testing and penalties for positive tests. I have also witnessed a largely inadequate, milquetoast response to emerging threats to racing's integrity. Most significant among these threats are blood-doping agents and other drugs that require an extensive out-of-competition program for detection.

I believe the threat to the integrity of our sport is greater now than it was 25 years ago.

We are in this predicament because we lack central governance for drug testing and penalties for violations, which, of course, all other major sports have.

We have been promised and have held out hope that uniformity was achievable and just around the corner. It is not achievable, nor is it around the corner, so you can stop waiting. Uniformity under our current regulatory structure is a mirage.

Lack of uniformity does not equate to lack of effort. In a nutshell, uniformity is *incompatible* with the current structure of individual state prerogatives. Try as we might, we cannot and will not get to our desired level of uniformity with our existing regulatory structure. Once we acknowledge this, it will be much easier to choose a new path leading to true national uniformity.

It should be noted that state regulators (i.e. Commissions) did not create the current model. They inherited it. This occurred decades ago when state legislatures bequeathed to racing commissions the authority and responsibility for equine drug testing.

This lack of uniformity has always been an issue, but emerging threats have made us much more susceptible to the designs of those who cheat. While these threats have increased, racing has become more globalized and the internet has rapidly spread all the shortcomings of our sport into consciousness of our dwindling fan base – and potential new fans.

I was born in Chicopee, Mass., in 1958. In that year, the four major Thoroughbred New England race tracks – Rockingham Park, Suffolk Downs, Narragansett Park and Lincoln Downs – drew 2,680,412 fans to the track. By comparison, that same year, the combined attendance for the Boston Red Sox, Bruins and Celtics was 2,168,412.

All these tracks are nothing but memories, except for Suffolk, which is scheduled to race three days this year. The reasons for the decline of Thoroughbred racing are many. I believe most people will agree that the foundation upon which we must build our sport moving forward is integrity. Our current structure of state prerogatives as it relates to drug testing and penalties has failed to provide this foundation.

For these reasons, I support the Barr-Tonko bill. It places the United States Anti Doping Agency (USADA) in a position to do virtually overnight what the racing industry has been incapable of doing over decades – mandate uniformity in drug testing, procedures and penalties. (Aug. 24, 2015)

Joe Gorajec served as executive director of the Indiana Horse Racing Commission and past chairman of the Association of Racing Commissioners International (RCI). The opinions herein are solely the opinions of the author and do not represent the opinions of the Indiana Horse Racing Commission.

This entry was posted in Ray's Paddock and tagged barr-tonko act, barr/tonko bill, drugs in racing, Horse Racing, horse racing regulations, Indiana Horse Racing Commission, Joe

Gorajec, national uniform medication program, out of competition testing by Joe Gorajec. Bookmark the permalink.

https://thehorse.com/158788/grant-joins-hsus-horse-racing-advisory-council/

Grant Joins HSUS Horse Racing Advisory Council

Barrie Grant, DVM, Dipl. ACVS, has been an equine veterinarian for more than 50 years and has been an official California Horse Racing Board veterinarian since 2009.

By Edited Press Release | Jun 17, 2018 | Welfare and Industry
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The Humane Society of the United States (HSUS) has announced that Barrie Grant, DVM, Dipl. ACVS, has joined its National Horse Racing Advisory Council.

The council is composed of industry professionals and specialists who actively promote higher animal welfare standards within the scope of their involvement in horse racing.

Since its formation in June of 2016, the council has been directing most if its time and resources in advancing federal medication reform legislation that would put an independent, non-financially-conflicted third-party player in charge of setting the rules regarding the medicating of horses. The HSUS said Grant's extensive experience as a veterinarian with racetrack experience will assist the council in making decisions to best protect the welfare of equine athletes.

Grant has been an equine veterinarian for more than 50 years and has been an official California Horse Racing Board veterinarian since 2009. He is board certified in surgery, was a surgeon and partner at San Luis Rey Equine Hospital, and is the author or co-author on more than 100 review papers mainly on surgical procedures and exercise physiology. He was part of the team that developed the surgical technique for treating cervical spinal cord compression. Grant is a lifelong member of both the American Association of Equine Practitioners and the American Veterinary Medical Association.

“I am hopeful that my lifetime of experience with the sport of horseracing and the diagnosis, treatment, management and pathogenesis of the medical conditions of athletic horses will enrich the perspective of the Humane Society of the United States National Horseracing Advisory Council,” said Grant. “It is humbling to be included with the other board members who all bring their special talents, time, and dedication to making racing a sport we all can take pride in.”

Council chairman Joe DeFrancis added, “We are delighted to welcome Dr. Grant to the … National Horseracing Advisory Council. The primary purpose of the council is to facilitate the flow of information and knowledge between the horse racing industry and HSUS, so that the industry and HSUS can work together with maximum effectiveness to solve problems and address important issues concerning the welfare of the equine and human athletes who are the cornerstone of the sport and business of horse racing. Our goal is to have the council comprised of knowledgeable and experienced individuals who represent every aspect and segment of horse racing.

“The knowledge and expertise that a veterinarian of Dr. Grant’s experience and stature brings to the council will be invaluable, and I know I speak for each member of the council when I say how excited we are to have the benefit of his participation.”

https://www.paulickreport.com/news/ray-s-paddock/handel-need-end-slaughter-american-equines/

Handel: We Need To End Slaughter Of American Equines

by Hal Handel | 09.22.2017 | 3:50pm

Throughout history, horses have worked alongside us as companions and partners in work, war and sport; they are living symbols of our nation's spirit.

We have the honor and duty to protect them, which is why I am imploring my colleagues in the horse racing industry to contact their federal representatives in Congress and urge them to cosponsor the Safeguard American Food Exports (SAFE) Act (H.R. 113/S.1706) and maintain the ban on funding horse slaughter in the United States.

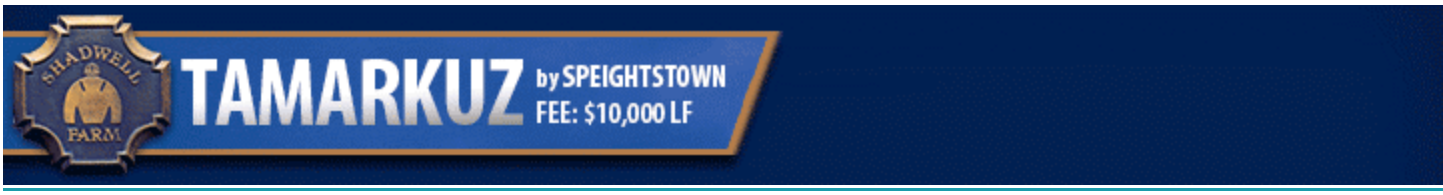
This key federal bill will prevent horse slaughter plants from opening in the U.S. and end the export of horses abroad for slaughter.

Our equine athletes are the lifeblood of our industry, yet too often they are condemned to a horrible death in a slaughter plant. The disreputable, predatory slaughter industry gathers up our loyal and trusting companions only to turn them into meat exports for profit.

Individuals who send horses to slaughter have nothing to do with responsible animal ownership or proper care, nor do they have an ounce of compassion for the graceful and sentient athletes they treat with such brutality. Horses unfortunate enough to end up in the hands of kill buyers suffer terribly at auctions, during transport and during the grisly slaughter process itself.

Horses are no better off being slaughtered in the U.S. than they are abroad. Before the last domestic plant closed in 2007, the United States Department of Agriculture (USDA) documented rampant cruelty at U.S. slaughter plants. There is no reason to believe that bringing slaughter back to the U.S. will make the process humane.

Quite simply, it is a brutal end for animals we have trained to trust us. Nothing about the way that slaughterhouses operate can be made humane for horses. These are lowbrow businesses concerned solely with serving foreign markets and turning a profit, a bloody one at that.



For the last 10 years, Congress has prevented wasteful spending and protected horses by including language in the Agricultural Appropriations Bill prohibiting federal funding for inspection of horse slaughter facilities.

In July, the House Appropriations Committee voted to spend our tax dollars on USDA inspections, which would pave the way for slaughter plants reopening in the U.S., while the Senate Appropriations Committee did not. Efforts to include an amendment in the House Appropriations bill to end this wasteful spending were stymied by pro slaughter politicians who have no interest in horse welfare. The very real possibility that these plants could once again set up shop in the U.S., using our tax dollars to fund USDA inspections, is looming.

Congress will need to reconcile this issue, and as horsemen, we need to take action now to protect our beloved horses by urging our federal representatives to vote to cosponsor the SAFE Act and maintain the current ban on horse slaughter in the U.S.

With the recent announcement by the Trump administration that the 2018 budget will include a 21% cut to the USDA budget, why would Americans want their tax dollars spent on supporting a predatory industry and inspecting meat that ends up on foreign dinner plates?

Horse slaughter also raises serious food safety concerns because drugs administered to horses make their meat unfit for human consumption. Those who work with horses on a daily basis need only glance at the labels on the products in their tack boxes. The overwhelming majority of those products come with a warning: *Not for use in food producing animals.*

There are several other good reasons to reject the return of horse slaughter.

Communities that hosted slaughter plants were stifled economically from the negative stigma of horse slaughter plants and real estate values plummeted.

Additionally, these neighborhoods were burdened with polluted water that often overwhelmed the small towns' septic systems, resulting in constant and horrible stenches.

Furthermore, the transportation of horses to these slaughter facilities was often just as inhumane as the treatment that awaited them. We shouldn't spend millions of American taxpayer dollars just to enable a cruel practice so that a greedy few can peddle tainted horse meat to the public. In the past, this has occurred at the expense of the taxpayer.

The slaughterhouse is not the least expensive way of ending the life of a horse, but it is the greediest and most inhumane way.

It is time for my fellow horse racing enthusiasts to take action and safeguard our horses. I believe that it is our duty to be the voice of our horses and stand up to the abusive horse slaughter industry.

I would strongly encourage you to contact your hometown legislators in both the House of Representatives and the Senate. Let them know that as a member of our country's horse racing industry, you reject horse slaughter. Urge them to take the necessary steps to prevent it from returning to the U.S.

Click here to contact your U.S. Senator and House of Representative member.

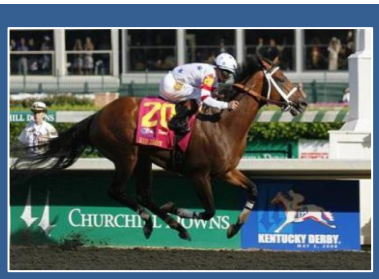
Hal Handel is a former deputy attorney general in New Jersey who supervised the state grand jury investigation into the Tony Ciulla race fixing scandal in the 1970s. He later served as executive director of the New Jersey Racing Commission and served in executive capacities at Monmouth Park, Meadowlands, Philadelphia Park, and the New York Racing Association. He is a past president of the Thoroughbred Racing Associations of North America and a past chairman of the Thoroughbred Racing Protective Bureau.

This entry was posted in NL Article, Ray's Paddock and tagged congress, Hal Handel, Horse Slaughter, house appropriations committee, SAFE Act, Safeguard American Food Exports Act, senate appropriations committee, United States Department of Agriculture, USDA by Hal Handel. Bookmark the permalink.

factsheet

Support the Horseracing Integrity Act of 2017

In the 115th Congress, H.R. 2651 was introduced by Reps. Andy Barr (R-KY) and Paul Tonko (D-NY).



"There has been expressed concern, primarily among examining veterinarians and those who observe the industry, about whether the current medication practices are in the best interest of the horse."

-Dr. Rick Arthur, Equine Medical Director of the California Horse Racing Board

The Problem

Despite its national and international scope, modern horse racing is still being conducted under outdated state-by-state drug and medication rules. This causes risk to the horses running races; confusion for owners and trainers whose horses race across state lines; and inconsistency for bettors who want to be able to fairly evaluate horses. It's clear that when it comes to medication, the horse racing industry can't both promote and police the sport. It needs a national, independent, non-governmental organization like the U.S. Anti-Doping Agency to create and maintain a system that protects horses, horsemen and fans.

The Facts

- Horseracing is a multibillion dollar industry, generating an estimated $40 billion annually and 400,000 jobs.
- The U.S. leads the world in the rate of fatal racing injuries at 1.89 per 1,000 starts (measured over approximately 310,000 starts).
- There is an overuse of therapeutic medication that masks pain and enables an injured horse to race when rest and time off would be more appropriate.
- With a lack of out-of-competition testing, veterinarians and other racing officials have expressed concerns that pre-race exams at racetracks are compromised by the use of drugs that can disguise the unsoundness of a horse.
- On-track betting and interstate, off-track wagering are the financial engines of the horse racing industry.
- Many veterinarians, geneticists, regulatory officials and racing fans believe that America's practice of medicating horses is harmful to the Thoroughbred breed.
- Congress considered banning drugs in horseracing in 1980, but instead allowed each state to make its own decisions on drugs and horseracing. This has resulted in a patchwork of state laws that encourage trainers caught doping their horses to move from state to state and continue doping and racing their horses.

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The Solution

The Horseracing Integrity Act of 2017will ensure equine welfare, protect the integrity of the sport and promote a sustainable and viable horse racing industry in the United States by granting independent control over rule-making, testing and enforcement oversight regarding drugs and medication to a new Authority created by the U.S. Anti-Doping Agency.

USADA – the same agency recognized by Congress as the official anti-doping agency for the Olympic, Pan American and Paralympic sports in the United States – is a national, independent, non-governmental organization with a proven track record of creating uniform standards and science-based oversight to protect the rights of clean competitors and the integrity of competition.

The new Authority, with limited oversight under the Federal Trade Commission (FTC), would be comprised of representatives of USADA and members of the horseracing industry, and would be responsible for:

- developing, publishing, and maintaining rules regarding substances, methods, and treatments that may or may not be administered to Thoroughbred race horses;
- implementing programming related to anti-doping education, research, testing, and adjudication to prevent the racing of horses who have been so affected; and
- establishing uniform rules imposing sanctions, up to and including a lifetime ban from horseracing, for those who violate the rules.

The Act would require that horse racing associations and off-track betting operators recognize the jurisdiction and authority of the independent Authority as a condition of accepting, receiving or transmitting interstate wagers on horse races.

Support for the Horse Racing Integrity Act

This legislation has been endorsed by The Jockey Club, the Breeders' Cup Ltd., the Water, Hay, Oats Alliance (WHOA), The Humane Society of the United States, the Kentucky Thoroughbred Association, Kentucky Thoroughbred Owners & Breeders, the Consignors and Commercial Breeders Association, Meadowlands Racetrack, Tioga Downs, Vernon Downs, Arapahoe Park, The Stronach Group (parent company to the Maryland Jockey Club, The Preakness Stakes, Santa Anita Park, Gulfstream Park, Portland Downs, and Golden Gate Fields) the Humane Society Veterinary Medical Association, and many horse owners, track owners, and trainers.

For more information please contact Marty Irby at mirby@hslf.org

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Myths and Facts Regarding the Horseracing Integrity Act

Myth: Adequate rules and enforcement already exist to prevent doping in horseracing.

Fact: There are no uniform rules to prohibit performance-enhancing drugs and penalize doping violations in horseracing. Almost all American race horses are injected with race day medication, a practice banned by almost all other countries. Trainers can violate medication rules multiple times, seemingly with impunity.

Myth: This bill would create a new federal bureaucracy to regulate horseracing.

Fact: The Act places responsibility for the creation and enforcement of new nationwide rules with an independent, non-governmental oversight authority that may appoint state racing commissions to assist with enforcement, with limited oversight under the FTC. Funding for the anti-doping program mandated by the bill would come from industry, not the taxpayer.

Myth: The federal government has no place in horseracing.

Fact: Federal law already regulates interstate or "simulcast" racing for Thoroughbred, Standardbred (harness), and Quarter horses. This bill would establish a national anti-doping program, managed by an independent, non-governmental authority and would ensure a level playing field wherever interstate wagering on horse races is offered.

Myth: Horseracing groups can solve doping problems without federal legislation.

Fact: Industry groups and state commissions have promised reform for decades. However, since horseracing lacks a national league or commissioner to set and enforce rules, federal legislation that establishes an independent national oversight body charged with developing and enforcing uniform rules is the only viable way to ensure safety and integrity.

Myth: The bill could eliminate the use of beneficial drugs and veterinary care for race horses.

Fact: Nothing in the Horseracing Integrity Act of 2017 prohibits a racehorse from receiving therapeutic care or drugs. Horses should not race when needing such therapy - as doing so can lead to breakdowns, and puts at risk their safety and that of their riders.

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http://www.thoroughbreddailynews.com/julie-krone-joins-humane-societys-horse-racing-council/

Julie Krone Joins Humane Society's Horse Racing Council

Tuesday, April 25, 2017 at 2:18 pm | Back to: Shared News

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Julie Krone | *Adam Coglianese*

Pioneering Hall of Fame jockey Julie Krone has joined the Humane Society of the United States' National Horse Racing Advisory Council. Krone is the first female jockey to win a Triple Crown race and is also the first woman inducted into the National Museum of Racing and Hall of Fame.

Since its formation in June 2016, the Council has been directing most if its time and resources in advancing federal anti-doping legislation that would put an independent, non-financially-conflicted third-party player in charge of setting the rules regarding the same-day medicating of horses.

"I am honored to be selected to participate in The HSUS' Horse Racing Advisory Council and look forward to working with the council members on legislation that will help protect both the equine and human athletes involved in this thrilling sport," said Krone. "As the first woman inducted into the National Museum of Racing and Hall of Fame, I hope my years of experience as a jockey will allow me to help drive changes needed in the industry that will better protect its athletes and allow the industry to prosper."

http://www.drf.com/news/krone-appointed-humane-society-council-racing

Krone appointed to Humane Society council on racing

By Matt Hegarty

Barbara D. Livingston

Julie Krone was inducted to the National Museum of Racing and Hall of Fame in 2000.

Julie Krone, the first woman to be inducted into racing's Hall of Fame, has been appointed to a council at the Humane Society of the United States focusing on racing, the Humane Society announced Tuesday.

The announcement of the appointment came on the same day that Frank Stronach, the owner-breeder who owns the racing company The Stronach Group, announced his support for federal legislation that the Humane Society and several influential racing and breeding organizations have endorsed. The federal legislation would appoint a private, non-profit company, the U.S. Anti-Doping Agency, as the overseer of the sport's medication and drug-enforcement policies.

At the HSUS, Krone will join another Hall of Fame rider, Chris McCarron, along with several racing officials, including Joe De Francis, the former owner of the Maryland Jockey Club; Jim Gagliano, president and chief operating officer of The Jockey Club; Allen Gutterman, a former racing executive; Joe Gorajec, former executive director of the Indiana Horse Racing Commission; and Staci Hancock, a founding member of a group opposed to the use of raceday medication.

The Humane Society racing council was formed last year, in large part to lobby for the passage of the federal legislation. In the release announcing Krone's appointment, the Humane Society said the council "has been directing most of its time and resources in advancing federal anti-doping legislation," in reference to the bill introduced last year.

“I hope my years of experience as a jockey will allow me to help drive changes needed in the industry that will better protect its athletes and allow the industry to prosper,” Krone said in a statement. Krone is married to Daily Racing Form executive columnist Jay Hovdey.

While several racing organizations have endorsed the bill, the legislation is controversial among a number of other racing constituencies. In particular, horsemen have said that the legislation is designed to put in place a ban on the raceday use of the diuretic furosemide, which is legal to administer in the United States and Canada on raceday to mitigate bleeding in the lungs. Horsemen contend the use of the drug is both humane and effective, while opponents maintain that raceday use of any drug places a stain on the sport.

In a statement released Wednesday in support of the legislation, Stronach specifically referenced his opposition to raceday medication (Stronach’s horses in the United States and Canada race on furosemide).

“No raceday medication is a giant step forward,” Stronach said in the statement. “I believe, in the long run, no raceday medication is better for the horses and for the industry.”

The legislation is not expected to gain traction in the next several years, in large part due to the lack of unanimity among racing’s constituencies, racing lobbyists have said.

https://www.nytimes.com/2012/03/25/us/death-and-disarray-at-americas-racetracks.html?_r=1&pagewanted=all

Mangled Horses, Maimed Jockeys

WALT BOGDANICH, JOE DRAPE, DARA L. MILES and GRIFFIN PALMERMARCH 24, 2012

RUIDOSO, N.M. — At 2:11 p.m., as two ambulances waited with motors running, 10 horses burst from the starting gate at Ruidoso Downs Race Track 6,900 feet up in New Mexico's Sacramento Mountains.

Nineteen seconds later, under a brilliant blue sky, a national champion jockey named Jacky Martin lay sprawled in the furrowed dirt just past the finish line, paralyzed, his neck broken in three places. On the ground next to him, his frightened horse, leg broken and chest heaving, was minutes away from being euthanized on the track.

For finishing fourth on this early September day last year, Jacky Martin got about $60 and possibly a lifetime tethered to a respirator.

The next day, it nearly happened again. At virtually the same spot, another horse broke a front leg, pitching his rider headfirst into the ground. The jockey escaped serious injury, but not the 2-year-old horse, Teller All Gone. He was euthanized, and then dumped near an old toilet in a junkyard a short walk from where he had been sold at auction the previous year.

In the next 24 hours, two fearful jockeys refused their assigned mounts. The track honored two other riders who had died racing. As doctors fought to save Mr. Martin's life, a sign went up next to the track tote board: "Hang in there, Jacky. We love you."

On average, 24 horses die each week at racetracks across America. Many are inexpensive horses racing with little regulatory protection in pursuit of bigger and bigger prizes. These deaths often go unexamined, the bodies shipped to rendering plants and landfills rather than to pathologists who might have discovered why the horses broke down.

In 2008, after a Kentucky Derby horse, Eight Belles, broke two ankles on national television and was euthanized, Congress extracted promises from the racing industry to make its sport safer. While safety measures like bans on anabolic steroids have been enacted, assessing their impact has been difficult because many tracks do not keep accurate accident figures or will not release them.

But an investigation by The New York Times has found that industry practices continue to put animal and rider at risk. A computer analysis of data from more than 150,000 races, along with injury reports, drug test results and interviews, shows an industry still mired in a culture of drugs and lax regulation and a fatal breakdown rate that remains far worse than in most of the world.

If anything, the new economics of racing are making an always-dangerous game even more so. Faced with a steep loss of customers, racetracks have increasingly added casino gambling to their operations, resulting in higher purses but also providing an incentive for trainers to race unfit horses. At Aqueduct Racetrack in Queens, the number of dead and injured horses has risen sharply since a casino opened there late last year.

Mr. Martin's injury occurred in a state with the worst safety record for racetracks, a place where most trainers who illegally pump sore horses full of painkillers to mask injury — and then race them — are neither fined nor suspended and owners of those drugged horses usually keep their winnings.

The failure of regulators to stop that cheating is reflected in the numbers. Since 2009, records show, trainers at United States tracks have been caught illegally drugging horses 3,800 times, a figure that vastly understates the problem because only a small percentage of horses are actually tested.

In the same period, according to the Times analysis, 6,600 horses broke down or showed signs of injury. Since 2009, the incident rate has not only failed to go down, it has risen slightly.

The greatest number of incidents on a single day — 23 — occurred last year on the most celebrated day of racing in America, the running of the Kentucky Derby. One Derby horse fractured a leg, as did a horse in the previous race at Churchill Downs. All told, seven jockeys at other tracks were thrown to the ground after their horses broke down.

A state-by-state survey by The Times shows that about 3,600 horses died racing or training at state-regulated tracks over the last three years.

In one 13-day stretch of racing in 2010 at Sunland Park Racetrack and Casino in New Mexico, nine horses died racing, five were hauled away in ambulances and two jockeys were hospitalized, one in critical condition.

"It's hard to justify how many horses we go through," said Dr. Rick Arthur, the equine medical director for the California Racing Board. "In humans you never see someone snap their leg off running in the Olympics. But you see it in horse racing."

Even some of America's most prestigious tracks, including Belmont Park, Santa Anita Park and Saratoga Race Course, had incident rates higher than the national average last year, records show.

Why racehorses break down at such a high rate has been debated for years, but the discussion inevitably comes back to drugs.

Laboratories cannot yet detect the newest performance-enhancing drugs, while trainers experiment with anything that might give them an edge, including chemicals that bulk up pigs and cattle before slaughter, cobra venom, Viagra, blood doping agents, stimulants and cancerdrugs.

Illegal doping, racing officials say, often occurs on private farms before horses are shipped to the track. Few states can legally test horses there.

"They are pharmacist shops," said Dr. George Maylin, the longtime head of New York State's testing laboratory. "Nobody has any control over what they are doing."

Even so, legal therapeutic drugs — pain medicine in particular — pose the greatest risk to horse and rider. In England, where breakdown rates are half of what they are in the United States, horses may not race on any drugs.

At higher levels, pain medicine can mask injury, rendering prerace examinations less effective. If a horse cannot feel an existing injury, it may run harder than it otherwise would, putting extra stress on the injury. As many as 90 percent of horses that break down had pre-existing injuries, California researchers have found.

“This is just a recipe for disaster,” said Dr. Tom David, who until this year was chief veterinarian for the Louisiana Racing Commission. “Inflamed joints, muscles and mild lameness are masked by medication and therefore undetectable to the examining veterinarian.”

While high-profile Triple Crown races get the most attention, the mainstay of racing in America is the lower tier, so-called claiming races. Horses in these races are most vulnerable, in part because regulators often give them less protection from potentially dangerous drugs.

The Times analysis found that horses in claiming races have a 22 percent greater chance of breaking down or showing signs of injury than horses in higher grade races. That lower level of race has been particularly affected by the arrival of casinos.

At Aqueduct, most of the 16 horses that have died so far this year were in the lower ranks, where purses have increased the fastest because of new casino money.

“It’s hard to watch these poor animals running for their lives for people who could really care less if they live,” said Dr. Margaret Ohlinger, a track veterinarian at Finger Lakes Casino and Racetrack in upstate New York. She performs pre-race inspections and treats horses injured in races but is not responsible for their overall care.

Last year at the track, Dr. Ohlinger counted 63 dead horses. That, she said, is more than double the fatalities of five years earlier.

Oversight Undermined

Race officials have always done their best to hide fatal breakdowns, erecting screens around fallen horses and then refusing to disclose the tracks’ accident rates.

But amid criticism that individual state racing commissions lacked the will to make the sport safer, and the threat of federal oversight, the industry promised changes, including new restrictions on the use of drugs, a program to accredit racetracks and drug-testing laboratories and uniform rules for punishing drug violators.

The industry also set up a national database where tracks were asked, but not required, to report injuries with the promise of confidentiality.

So far, the response to these reform measures has fallen short.

Fifty-five tracks pledged that they would seek accreditation, requiring among other things prerace inspections and postmortem examinations, or necropsies. Fewer than half have kept their promise.

“Some tracks do not have the money to spend to meet our standards; others think it’s window dressing and why bother,” said Michael Ziegler, executive director of the National Thoroughbred Racing Association Safety and Integrity Alliance. “Any follow-up with tracks has gone unanswered.”

The laboratory accreditation program, introduced in July 2009, has fared even worse. After calling the program an “unprecedented” step that “ultimately will change the face of drug testing in this country,” a consortium of industry groups that manages it says not a single lab has been accredited.

An association of racing regulators wrote to Congress on May 14, 2010, boasting that with the exception of anti-bleeding medicine, “race day medications are not allowed.” Yet records show that in Florida, a major racing state, trainers continue to use corticosteroids, an anti-inflammatory, on race day.

The national repository for injury reports, maintained by the Jockey Club, the most powerful racing industry group, has been more successful, gathering data from 92 percent of the racing days.

“We put it into a database, and we provide tools back to the racetracks where they can analyze and slice and dice the information themselves,” said James L. Gagliano, president of the Jockey Club, who says the group has encouraged racetracks to make the statistics public. So far, 24 out of 86 tracks have done so.

To assess how often horses get injured, The Times bought data for about 150,000 races from 2009 through 2011, then searched for terms indicating that a horse encountered a physical problem, like “broke down,” “lame” or “vanned off.”

Although the people who chronicle the races, known as chart callers, can be stylistically different, they are taught to use standard industry terms, and their descriptions constitute the official record used by gamblers to evaluate horses.

The analysis showed that during those three years the rate of incidents for horses in the United States was 5.2 per 1,000 starts.

By contrast, Woodbine Racetrack in Toronto, which year after year has one of the lowest breakdown rates in North America, had an incident rate of only 1.4, according to the Times analysis. “One of the differences here is medication is not as permissive as it is in the U.S.,” said Jamie Martin, executive vice president of racing at Woodbine.

According to the analysis, five of the six tracks with the highest incident rates last year were in New Mexico. All are casino tracks, commonly called “racinos.” Ruidoso, where Jacky Martin was injured, topped the list in 2011 with 14.1 incidents per 1,000 starts. Ruidoso attributes its incident rate in part to the failure of horses to acclimate quickly to the track’s elevation. Some horses that appeared to be injured, track officials said, may have simply needed time “to catch their breath.”

Yet no accident over the last three years can match what occurred in a single race on Feb. 29, at Hollywood Casino at Charles Town Races in West Virginia. Eight horses started. Seven fell. One finished. Along the way, seven jockeys were left scattered on the ground.

The next and final race was canceled, not just because it took so long to clear the track, but also because too few jockeys were available or willing to ride.

Drug Violations

It was the day’s first race in Hobbs, N.M. The track was fast and the weather clear. Shortly after noon on Oct. 16, 2010, nine young horses were loaded into the starting gate at Zia Park Casino.

With the finish line a mere 400 yards away, this would be an all-out sprint, horse racing’s equivalent of a drag race. While these races, run by a breed called quarter horses, lack the ebb-and-flow suspense of a longer thoroughbred race, they make up for it in a pure adrenalin rush. The best quarter horses can hit nearly 50 miles an hour.

Three weeks earlier at Zia Park, Mark Anthony Villa was on the back of a quarter horse when it fell just past the finish line, throwing him to the ground. With a herd of thousand-plus-pound animals bearing down on him, Mr. Villa tried to crawl to safety.

He never made it. A horse’s hoof struck him in the head with such force that his helmet shot like a bullet across the track. He died within an instant, leaving a wife and twin children.

For years, track veterans could only speculate as to whether racing quarter horses was more dangerous than racing thoroughbreds. In fact, the Times analysis shows that quarter horses have a nearly 29 percent greater chance of breaking down or showing signs of injury.

With Mr. Villa's death still on the minds of riders and spectators, a gray 2-year-old colt named I Glance at Chicks settled in the 6 hole waiting for the starting bell. For bettors, he was an animal to watch. The horse had won his only race and was trained by Andres Gonzalez, who, according to racing commission records, was not above allowing his horses to race with extra help. Illegal help.

A week earlier, another horse trained by Mr. Gonzalez had raced at Zia Park with 12 times the legal limit of a drug that mimicked steroids. By the end of 2011, Mr. Gonzalez would have amassed a dozen drug violations in just four years. His uncle, Ramon O. Gonzalez Sr., for whom he often worked, had his own lengthy list of violations, including accusations that he drugged 10 horses in just two months.

Whether I Glance at Chicks felt pain as he raced is unknown, but he never challenged for the lead. Shortly after crossing the finish line in fifth place, he broke down. The diagnosis: a bone fracture in his front left leg and ligament damage, injuries from which he could not recover.

A veterinarian, Dr. Clayton McCook, euthanized the colt with an injection of pentobarbital. Afterward, Dr. McCook wrote a note "to whom it may concern," expressing his distress to the authorities over this fatal breakdown and others like it.

"I have had to euthanize several horses due to catastrophic injuries and feel they are occurring in greater numbers than one should expect," Dr. McCook wrote. "I do not pretend to be an expert in racing surfaces, nor in the training of racehorses, but I do know that something appears to be amiss at Zia Park."

According to an analysis of race records, Zia Park in 2010 had the nation's second-highest incident rate, 13.3. Last year, it ranked fourth with a rate of 11.9. After horse owners complained about the track surface, Zia Park officials said they spent $80,000 resurfacing it before the 2011 racing season.

During the three days that a Times reporter visited Zia Park last November, eight horses collapsed, died or were transported off the track. At the time, track officials said it was company policy not to allow a reporter access to the backside where trainers stable their horses.

Christopher McErlean, vice president of racing at Penn National Gaming, which owns Zia Park, said in a statement that the Times analysis used figures "produced by nonmedical professionals for the purpose of handicapping feature races."

Mr. McErlean also said some horses are vanned off as a precaution and may not actually have been injured.

But Zia Park officials said that last year, "a significant number" of horses had to be carried off the track because of exhaustion stemming from the possible abuse of a drug that mimics anabolic steroids as well as "other medication issues."

Mr. McErlean said Penn Gaming endorses tougher penalties for those who violate drug rules.

Without a postmortem exam of I Glance at Chicks, no determination could be made as to whether a pre-existing condition or some other unknown factor might have played a role in his demise. But tests did reveal that the horse had been dosed with a large load of a powerful painkilling medicine called Flunixin.

In at least two states, 2-year-olds may not race with any Flunixin. Not so in New Mexico, where they can run with up to 50 nanograms of the drug, more than double the amount allowed in a higher class of competition called graded stakes races.

But even that higher amount was not enough for Mr. Gonzalez. I Glance at Chicks carried 282 nanograms of Flunixin.

To put that figure in perspective, Dr. Mary Scollay, chief veterinarian for the Kentucky Racing Commission, said she had never seen such high levels in her state.

“When you look at the history of our medication violations — Flunixin — most are under 50 nanograms, 35 nanograms, something like that,” Dr. Scollay said. In fact, she said she had never seen a violation in Kentucky over 104.

In New Mexico, it is common practice.

Tests on horses in New Mexico showed results over 104 nanograms on 68 occasions since 2009, with some registering 1,000 and even 2,400, records show. The levels are so high that regulatory veterinarians in other states say the horses must have been drugged on race day, a practice that is forbidden.

Before the New Mexico Racing Commission could pass judgment on the overdosing of I Glance at Chicks, another horse trained by Mr. Gonzalez tested positive for even higher levels of Flunixin. The extra dosing did not hurt performance. The horse finished first, and its owner, Mr. Gonzalez’s cousin Ramon Gonzalez Jr., got to keep his winnings.

If Andres Gonzalez was worried about how the racing commission viewed his treatment of I Glance at Chicks, he need not have been. Records show he received a warning and nothing more.

Lax Penalties

New Mexico’s racing industry — the tracks and their regulators — has been unusually slow in responding to the safety alarms.

Four of the state’s five racetracks, including Zia Park and Ruidoso, are unaccredited, and the track where Mr. Martin’s injury occurred does not report accidents or positive drug tests to groups that monitor such events.

New Mexico also recorded no positive tests in 2010 and 2011 for the most frequently abused pain medicine in racing, phenylbutazone, a nonsteroidal anti-inflammatory commonly known as “bute.” After The Times asked why none had been found, the new executive director of the state’s racing commission, Vince Mares, said that after researching the question, he discovered that the previous leadership “had cut back on the tests” for financial reasons.

Without a national law regulating drugs in racing, New Mexico regulators can be as lenient as they wish in disciplining drug violators.

Trainers in New Mexico who overmedicate horses with Flunixin get a free pass on their first violation, a $200 fine on the second and a $400 fine on the third, records show.

In Indiana, by contrast, winnings are forfeited after the first drug offense. “If someone who violates the rule thinks the penalties are going to be mild or nonexistent, then breaking the rules is just a cost of doing business,” said Joe Gorajec, the executive director of the Indiana Horse Racing Commission.

New Mexico gives offenders another break: it wipes away Flunixin violations every 12 months, allowing trainers to again overmedicate horses without penalty. Dozens of huge Flunixin overdoses have resulted in warnings only.

Sometimes the same horse is illegally drugged twice. On May 9, 2009, Runawayslew, a horse trained by Andres Gonzalez, raced with two anti-inflammatory drugs. Nineteen days later, under another trainer, Runawayslew raced on cocaine.

To varying degrees, the picture is similar nationwide. Trainers often face little punishment for drug violations, and on the rare occasions when they are suspended, they are allowed to turn their stables over to an assistant. Since January 2005, 116 trainers have had five or more drug violations, and 10 trainers had 10 or more, records show.

In New Mexico, Cody Kelley, an Albuquerque lawyer who represents people accused of violating racing commission rules, including Andres Gonzalez, said punishments were too arbitrary.

“Are there people that cheat at horse racing in New Mexico? Yes, happens everywhere,” Mr. Kelley said. “But I think our commission right now is not equipped to deal with it. What we need are national rules.”

Mr. Mares, the New Mexico racing chief, agrees that his agency needs more uniform penalties to avoid charges of favoritism. “There is an issue of consistency — you can quote me on this,” Mr. Mares said. “It is being addressed.”

New Mexico recently became the first state to temporarily ban all horses from racing on clenbuterol, a drug that aids respiration, but that has been widely abused because it can build muscle.

In recent years, the state commission has had its embarrassments.

One former investigator faces trial on charges of stealing horses while working at the commission. Another trainer’s doping violation was dismissed because the assistant attorney general handling the case neglected to show up in court. And the commission had to drop charges against Ramon O. Gonzalez Sr. for drugging 10 horses because it forgot to file the proper paperwork, according to the state attorney general’s office.

Nonetheless, odds are slim that any of the Gonzalezes — Andres, Ramon Sr. or Ramon Jr. — will show up at a New Mexico racetrack any time soon. In late January, a federal grand jury in Albuquerque indicted them on charges of participating in a drug trafficking scheme tied to one of Mexico’s most notorious drug cartels. All have pleaded not guilty.

Andres Gonzalez was arrested at Sunland Park Racetrack and Casino in New Mexico. His uncle, Ramon Sr., was arrested while pulling a horse trailer that the authorities said was carrying 26 kilograms of cocaine and 500 pounds of marijuana.

Masking Pain, or Healing It

Breakdowns can be caused by a variety of factors, including poor track surface and jockey mistakes. But drugs, often used to mask existing injuries, are the prime suspect.

“It’s not that these medications caused the injuries, but the trainers knew the horses were injured and gave them the meds to get them into the race,” said Dr. Arthur, the veterinarian for the California Horse Racing Board.

Necropsies are considered essential to determining if an existing injury contributed to a fatal breakdown. However, only 11 states require them, a Times survey found.

In California, where necropsies are required, researchers found that a “large majority” of horses had existing problems at the site of their fatal injuries.

“To be fair, some of that is microscopic and may not be readily apparent,” Dr. Arthur said. “We’re trying to figure out why vets and trainers are not identifying injuries prior to catastrophic injuries.”

But many prior ailments are indeed serious. The Times obtained hundreds of necropsy reports on racehorses that died racing in Pennsylvania and found problems that included “severe degenerative joint disease,” “severe chronic osteoarthritis” and pneumonia with “severe, extensive” lung inflammation. One horse had 50 stomach ulcers. Another had just one eye. Pathologists also found metal screws in two horses that had broken bones from previous accidents.

In the United States, horses are usually allowed to run on some dose of pain medication, usually bute. The question, fiercely debated in the racing community, is at what level do therapeutic drugs make racing unsafe?

Virginia’s fatality rate went up after regulators in 2005 raised the allowable level of bute to 5 micrograms from 2 micrograms. “Our catastrophic incidents increased significantly,” said Dr. Richard Harden, equine medical director for the state racing commission.

Virginia returned to the lower level in 2009, though the fatality rate has not come down.

Iowa’s fatality rate rose by more than 50 percent after the state in 2007 allowed a higher level of bute.

Regulatory veterinarians say the higher allowable levels make it difficult for them to spot lameness and injury during prerace examinations. In one study, researchers at Oklahoma State University said they found bute in most of the horses that died racing or training at Oklahomatracks in 2010. Six had both bute and Flunixin, a dangerous practice called “stacking,” the report said.

The researchers also expressed concern that despite fewer races, a record number of horses died, necessitating a “careful re-evaluation of track surfaces, medication/enforcement and prerace examinations.”

But prominent owners and trainers, and even some veterinarians, say evidence linking drugs and breakdowns is unconvincing.

Kent H. Stirling, chairman of the national medication committee for the Horsemen’s Benevolent and Protective Association, said there was “no scientific evidence whatsoever” that 5 micrograms of bute on race day is dangerous.

Mr. Stirling and others say sore horses should not be denied therapeutic medicine when needed. “If you’re a horseman and you’re trying to keep a horse going and keep him happy and healthy as you can, then these therapeutic medications are very helpful,” he said.

Regulators typically view prescription drug violations as more benign than the use of banned substances on horses. And they constitute the bulk of the 3,800 violations that The Times found by surveying racing states.

But others, including racing regulators overseas, say horses should not compete on any drug regardless of type.

“Therapeutic drugs, by definition, are used for healing and curing,” said Arthur B. Hancock III, whose farm produced three Kentucky Derby winners. “Drugs that mask pain and enhance performance are not ‘therapeutic.’ They are what they are: performance-enhancing drugs.”

The industry group that runs graded stakes races had promised to ban all therapeutic drugs for 2-year-olds, but in late February backed off, saying it did not have enough time to bring state regulators on board.

George W. Strawbridge Jr., a prominent breeder and owner, resigned from the group over that decision, calling it “one of the most craven acts” he had seen.

“How on earth did we get to this sorry state?” Mr. Strawbridge said. “The first reason is that in this country there are no significant consequences for doping horses.”

Respecting the Ride

Chris Zamora knows the sensation of riding a sore horse. But one ride in particular stands out.

On Nov. 25, 2008, Mr. Zamora was guiding his horse, Sinful Heart, into the first turn at Zia Park when he sensed something was wrong. “He didn’t want to take the turn,” he said. “He was in pain.”

Sinful Heart drifted out, clipped heels with another horse and fell. A trailing horse tripped over them.

Mr. Zamora, the winner of more than 1,000 races, nearly died in the accident, fracturing his skull, pelvis, ribs and four vertebrae. His lungs collapsed, his liver was lacerated and his heart was compressed. “They had to insert a needle to take the pressure off of my heart,” he said.

Sinful Heart survived to race three more times, in successively cheaper races, never winning before collapsing and dying on the track at Ruidoso.

Four months after his accident, in March 2009, Mr. Zamora returned to the track. But he had changed. No more cheap horses. “I tried to ride quality over quantity,” he said. “I didn’t ride a horse that somebody said was already sore. I scratched more of them at that time than I had in my whole life.”

The best trainers might have been unhappy, he said, but they trusted his judgment and fixed the problem. “They were great horsemen,” Mr. Zamora said, offering the ultimate compliment.

But not all were. Now, he said, some trainers just go to another rider. “These guys will head a horse up until it breaks down completely, and when there’s a man on top of them, it’s bad,” he said.

Other injured jockeys tell similar stories. “I think more should be done for the horse to let him heal naturally than to be getting him to the next race so we can get one more race out of him,” said Randy Meier, a winner of more than 4,000 races, many in the Chicago area.

Along the way, Mr. Meier broke his neck, collarbones, ribs, shoulder, legs, arms, wrist and sternum and developed a brain bleed.

New Mexico jockeys have been hit particularly hard. Not only was Mr. Villa killed and Mr. Zamora and Mr. Martin critically injured, Juan Campos died in an accident in August 2008; Jimmy Ray Coates fell the same year, his heart stopping twice after breaking his femur, shoulder and collarbone; Carlos Rivas had no pulse en route to the hospital after rupturing his aorta in 2010, and the same year Kelsi Purcell fractured multiple vertebrae in a spill.

There were other injuries as well.

“We’ve been through this so many times,” said Terry Meyocks, national manager of the Jockeys’ Guild. More than 50 permanently disabled jockeys receive assistance from the Jockeys’ Guild, he said.

After Mr. Zamora’s accident, Mr. Martin, a friend and hunting partner, had told him not to abandon hope. “You’ll be back,” he said. “You’re in great shape, it won’t be that long. You’re not done. You won’t be in a walker.”

Like all jockeys, Mr. Zamora knew the risks of riding. “Every time you do it, you take a chance one is going to break it off. Even with the soundest horse you take a chance.”

Good jockeys can alter their ride if a horse is sore or about to break down. In some cases, though, there are no hints, no warnings. And that is when jockeys face the greatest danger.

Jacky Martin had no warning.

“I thought he was going to die,” said Adrian A. Ramos, who was riding in the same race. “He hit the ground hard, real hard. I was behind him and I saw everything.”

A Second Chance

The question almost everyone at the track wanted to ask was why. Why did Mr. Martin, at the top of his game, the winner of a record seven All American Futurities, agree to ride a cheap claiming horse with no victories just three days before he was to ride the favorite in the $2.4 million Futurity?

The favorite did eventually win and would have paid him $120,000, the jockey’s standard share. For riding the horse that broke his neck, Mr. Martin took home little more than the cost of a tank of gas.

Until that wrenching moment in the Ruidoso dirt, Mr. Martin at age 56 had been on a redemptive journey to right the wrongs in his life, to help younger jockeys avoid the mistakes he had made and to regain what he had lost: an opportunity to sit atop a racehorse and to coax from it all the power it was willing to give, and nothing more.

For four years, Mr. Martin had been barred from racing after being sentenced to probation in 2006 for poaching deer and possessing less than a gram of methamphetamine. He and his wife, Tracey, also his agent, moved to Louisiana. “I worked horses every day for three and a half years being a gallop boy,” Mr. Martin said. “That’s all I was, a $10 gallop boy.”

In the afternoon, Mr. Martin helped to build fences and even a barn, his wife recounted. “We actually bagged horse manure and sold it and delivered it just to get through,” she said.

It was a steep fall for a man so highly revered in the sport that Mexican businessmen would send armed guards to escort him to high-stakes races south of the border.

“After a time, he took ownership for the wrong things that he did and worked his way through it,” Ms. Martin said. Just as important, friends say, he developed an even deeper appreciation for the role others played in racing, from grooms to horse owners struggling to stay in the game.

In the summer of 2010, Mr. Martin was finally cleared to race, and he returned to Ruidoso unsure of how he would be received. When word spread that “Jacky was back,” owners were eager to extend a helping hand, but most of all, they were eager to win.

And win he did. With the racing season half over, Mr. Martin stormed into the lead to become the top winner and champion jockey for 2010.

“He was so grateful he got a second chance,” Ms. Martin said in December. “He was on the radio saying: ‘People out there need to know that they can be forgiven and succeed. If I can fix my screwed up life, you can too.’ ”

In Mr. Martin’s quest to win an eighth Futurity in 2010, his horse lost by a nose in one of the biggest upsets in the history of that race. But the loss did not diminish the joy he felt competing again.

“It’s just a fairy tale for it to turn out the way it has,” Mr. Martin told a racing publication in 2010.

Mr. Martin fell a year later, on the Friday before Labor Day at the beginning of the final, biggest weekend of racing at Ruidoso. The tens of thousands of spectators, who would later fill the stands and line the distant highway with parked cars, had yet to arrive.

Only a small, quiet crowd, including relatives of riders, trainers and owners, was on hand to watch Mr. Martin go down. One woman screamed because she mistakenly thought her husband had been the one injured.

The authorities did little to determine why Mr. Martin’s horse, Phire Power, broke down. The commission said drug tests found no prohibited substances, but the scope of those tests is unclear, including whether the horse was tested for bute. The state also said the horse’s body did not undergo any postmortem exam before it was destroyed.

Within minutes, Ms. Martin was escorted onto the track to be with her fallen husband. Over the next six months, she would rarely leave his side.

In two days, Mr. Martin had been scheduled to sign autographs at Ruidoso to raise money for injured jockeys. Instead, other jockeys signed autographs to raise money for him.

Since the accident, Mr. Martin has been in and out of hospitals in three cities. He has suffered through infections, pneumonia, nausea, weight loss, bed sores and other problems. He remains paralyzed, unable to move his arms or legs. He breathes with a respirator.

Meanwhile, the racing community has rallied to his side, sending not only words of support but also money to help defray his mounting health care costs. Ruidoso’s owner, R. D. Hubbard, promised $100,000. There have been silent auctions and other fund-raisers. His wife worries that it may not be enough.

Through it all, Mr. Martin refuses to feel sorry for himself.

In December, as he struggled to breathe in a Houston hospital, he told a reporter softly that he had no regrets.

“It’s a bad deal,” he said. “But if I could do it again, I would be right out there doing it. I ride horses. It’s the risk every jockey takes.”

Back home in El Paso, Ms. Martin says her husband derives one of his few pleasures from sitting in his wheelchair next to a window watching horses train silently in the distance.

Mr. Martin’s injury deeply affected Mr. Zamora. He was not only losing a friend from the jock’s room, the sport was losing a rider, a gentleman, who had come to represent the best it had to offer.

“He rode the best horses in the world, but he was worthy of the best horses in the world,” Mr. Zamora said. “He had great hands. He let a runner be a runner instead of going to the whip too early. Them animals loved him, and they ran for him and he understood them. When one didn’t want to run, he let ’em not run. He didn’t take to the whip. You have to understand them — that’s what makes a great horseman. And he was. He was special.”

Last fall, several weeks after Mr. Martin’s spill, Mr. Zamora left the jock’s room for the last time.

“I knew I had come so close, and I couldn’t deal with that.”

Pain, Up Close

It was the third race at Ruidoso on July 11, 2009.

In the stands, Laura and Armando Alvarado sat with their two grandchildren, ages 11 and 14.

The Alvarados were not racing fans, but this was a vacation — they had driven up to the mountain resort from El Paso — and they thought their grandchildren might enjoy watching their first horse race.

Mr. Alvarado took the children down to the rail for a closer look. Ten horses sprinted out of the gate, including a gray Texas-bred quarter horse named Sinful Heart, the same horse that fell several months earlier, nearly killing Chris Zamora.

Just past the finish line, Sinful Heart, with another rider on its back, broke down, collapsing on the track. “The horse is bleeding!” one of the children cried out.

The children were not visibly shaken, but Ms. Alvarado said she was sorry they had to witness death at such a close range. After a few more races, they went shopping.

Five days later, a relative with a passion for racing was visiting the Alvarados, and they all went to the track.

“It was going to be an all-day experience, and I thought how nice to have this man give them all this history and details,” Ms. Alvarado said.

Once again, Mr. Alvarado took the children to the rail to watch the finish of the day’s first race.

This time, a horse broke its leg, pitching its rider — who happened to be Chris Zamora — into the ground, where rider and animal rolled like tumbleweeds across the finish line.

“It was awful,” Mr. Alvarado said. Although Mr. Zamora was not seriously injured, the horse was. “The bone was showing through the skin,” Mr. Alvarado said.

Both children began to cry. “I have never seen anything that horrible close up,” Mr. Alvarado said. “The kids were terrified.”

The horse was euthanized on the track. The family quickly left the premises. Ms. Alvarado said: “I told Armando, just drive. We wanted to get out of there.”

Afterward, her granddaughter said, “I don’t want to go to a racetrack ever again.”

Ms. Alvarado wrote a letter to the editor of the local paper.

“For the sake of the animals and children, we felt compelled to let city officials, agencies and others know of this painful experience and urge you to investigate,” she wrote.

She said she sent copies of the letter to the mayor, the track, its chief veterinarian, the Humane Society and the American Society for the Prevention of Cruelty to Animals.

Ms. Alvarado expected a response.

She never got one, she said.

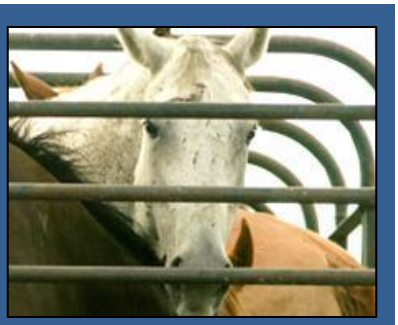
Rebecca R. Ruiz and Matthew Orr contributed reporting from New York.

A version of this article appears in print on March 25, 2012, on Page A1 of the New York edition with the headline: Mangled Horses, Maimed Jockeys. Order Reprints| Today's Paper|Subscribe

factsheet

Support the Safeguard American Food Exports (SAFE) Act (H.R. 113/S.1706)

In the 115th Congress, H.R. 113 was introduced by Rep. Vern Buchanan (R-FL), Jan Schakowsky (D-IL), Ed Royce (R-CA), and Michelle Lujan Grisham (D-NM) and S.1706 was introduced by Sens Robert Menendez (D-NJ), Lindsey Graham (R-SC), Sheldon Whitehouse (D-RI), and Susan Collins (R-ME)



"Here are these exquisite, immensely powerful creatures, who willingly give us their labor in return for our stewardship. They have attended us throughout history, bearing us across frontiers and into battle, pulling our plows, thrilling us in sport, warming us with their beauty… To send these trusting creatures to slaughter is beneath their dignity and ours."

-Laura Hillenbrand, author of "Seabiscuit"

This bill would prevent horse slaughter plants from opening in the U.S. and end the current export of horses abroad for slaughter for human consumption.

Forcing American taxpayers to pay for horse slaughter is fiscally irresponsible. It makes no sense for the federal government to spend millions of taxpayer dollars to oversee new horse slaughter plants. At a time when Congress is so focused on fiscal responsibility and the budget of the USDA's Food Safety Inspection Service is already stretched thin, the USDA should not extend funding for a new program to slaughter horses – a practice that 80% of the American public opposes.

U.S. horsemeat can be dangerous to humans because of the unregulated administration of numerous toxic substances to horses over their lives. American horses are not raised for human consumption, and they are routinely given hundreds of drugs and other substances, both legal and illegal, over their lifetimes that can be toxic to humans if ingested. These substances have not been approved and many have been specifically prohibited by the FDA for use in animals intended for human consumption. For example, a common pain reliever, Phenylbutazone, is known to cause potentially fatal human diseases, and there is no known safe level for residues of this drug in horsemeat. Horses are gathered from random sources, and there is no system in the U.S. to track medications and veterinary treatments given to horses to ensure that their meat is safe for human consumption.

Due to serious food safety concerns, the European Union suspended horsemeat imports from Mexico – where 87% of horses slaughtered for export to the EU are of U.S. origin. EU authorities made the decision after a series of scathing audits that exposed a plethora of problems, including a lack of traceability of American horses and horrific suffering on U.S. soil and in Mexico.

Horse slaughter is inhumane and cannot be made humane. Slaughter is a brutal and terrifying end for horses and is not humane. Horses are shipped for more than 24 hours at a time without food, water, or rest in crowded trucks in which the animals are often seriously injured or killed in transit. Horses are skittish by nature due to their heightened fight or flight response. The methods used to kill horses rarely result in quick, painless deaths; they often endure repeated blows during attempts to render them unconscious and sometimes remain alive and kicking during dismemberment. Before the last domestic plant closed in 2007, the USDA documented rampant cruelty violations and severe injuries to horses, including broken bones protruding from their bodies, eyeballs hanging by a thread of skin, and gaping open wounds.

For more information please contact Holly Gann at hgann@humanesociety.org.

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Supporting Documents for the Horse Racing Integrity Act

1. Articles
    a. 160 Racehorses Died From Injuries Suffered at Charles Town Races Since 2014
    b. A bipartisan approach to protecting racehorses
    c. American racing requires federal oversight, says HSUS boss
    d. Backers, Foes Of Horseracing Integrity Act To Testify Before Congress
    e. Charles Town Classic fiasco this response takes racing in the wrong direction
    f. Combating A Culture Of Cheating A Matter Of Trust
    g. Death of a Derby Winner: Slaughterhouse Likely Fate for Ferdinand
    h. Eight Belles' Death Sparks Controversy
    i. Gorajec Barr-Tonko Bill Only Path Toward Uniformity
    j. Grant Joins HSUS Horse Racing Advisory Council
    k. Handel We Need To End Slaughter Of American Equines
    l. Julie Krone Joins Humane Society's Horse Racing Council
    m. Krone appointed to Humane Society council on racing
    n. Mangled Horses, Maimed Jockeys
    o. The Humane Society of the United States forms national horse racing advisory council
    p. They're embarrassed already but is real humiliation next for these regulators
    q. Trainer Beattie At Rojas Trial: 'Almost Everybody' Illegally Treated Horses On Race Day
    r. What you need to know about the landmark Horse Racing Integrity Act
    s. What's next for Pennsylvania if it doesn't clean up its act?
    t. Why every day is Groundhog Day in U.S. racings rulemaking process

2. Other
    a. Horseracing Integrity Act Fact Sheet
    b. SAFE Act Fact Sheet
    c. The Jockey Club Equine Injury Database

https://www.bloodhorse.com/horse-racing/articles/180859/death-of-a-derby-winner-slaughterhouse-likely-fate-for-ferdinand

Death of a Derby Winner: Slaughterhouse Likely Fate for Ferdinand

Ferdinand, the 1986 Kentucky Derby winner who went on to capture the following year's Horse of the Year title with a dramatic victory over 1987 Derby hero Alysheba in the Breeders' Cup Classic, is dead. *The Blood-Horse* has learned the big chestnut son of Nijinsky II died sometime in 2002, most likely in a slaughterhouse in Japan, where his career at stud was unsuccessful.

Reporter Barbara Bayer, as detailed in an exclusive story in the July 26 issue of *The Blood-Horse*, attempted to learn of Ferdinand's whereabouts after a member of the Howard Keck family that owned and bred the horse inquired about having him returned to the United States, where he began his career at stud. As a racehorse, Ferdinand won eight of 29 starts and earned $3,777,978, retiring as what was then the fifth leading money winner of all time. His victory in the Kentucky Derby gave trainer Charlie Whittingham his first success in that classic, and it was the final career Derby win for jockey Bill Shoemaker.

Ferdinand was retired to stud in 1989 at Claiborne Farm near Paris, Ky., where he was foaled. His initial stud fee was $30,000 live foal, but he achieved little success as a stallion from his first few crops of runners.

Sold to Japan's JS Company in the fall of 1994 at a time when Japanese breeding farms were aggressively pursuing American and European breeding stock, Ferdinand spent six breeding seasons at Arrow Stud on the northern island of Hokkaido, from 1995-2000. Initially popular with local breeders (he was mated to 77 mares his first year), Ferdinand was bred to just 10 mares in his final year at Arrow, and his owners opted to get rid of him.

After efforts by the farm staff to place Ferdinand with a riding club failed, he passed into the hands of a Monbetsu, Japan, horse dealer named Yoshikazu Watanabe and left the farm Feb. 3, 2001. No attempt was made to contact either the Keck family or Claiborne Farm.

Bayer at first was told by Watanabe that Ferdinand had been "given to a friend." When she asked for more information, she was told Ferdinand "was gelded and I think he's at a riding club far away from here." In fact, records showed Ferdinand was bred

to six mares in 2001 and then two in 2002. He spent a period of time at Goshima Farm near Niikappu, where a former handler at Arrow Stud had seen him.

Finally, when Bayer told Watanabe she wanted to see Ferdinand, the story changed yet again. "Actually, he isn't around anymore," she was told. "He was disposed of late last year." Ferdinand's registration in Japan was annulled Sept. 1, 2002, Bayer learned.

"In Japan, the term 'disposed of' is used to mean slaughtered," Bayer wrote in *The Blood-Horse*. "No one can say for sure when and where Ferdinand met his end, but it would seem clear he met it in a slaughterhouse."

"Unfortunately, to those well-versed in the realities beyond the glitter and glory of the racetrack, it comes as no surprise," Bayer wrote. "Ferdinand's story is the story of nearly every imported stallion in Japan at that point in time when the figures no longer weigh in his favor. In a country where racing is kept booming by the world's highest purses and astronomical betting revenues, Ferdinand's fate is not the exception. It is the rule."

"That's just disgusting," said Dell Hancock, whose family operates Claiborne Farm, upon hearing the news of Ferdinand's likely fate. "It's so sad, but there is nothing anyone can do now except support John Hettinger's efforts to stop the slaughter of Thoroughbreds in this country. That wouldn't change anything in Japan...to have this happen to a Derby winner is just terrible."

While the Japanese are among the societies that consume horse meat, it is more likely a slaughtered Thoroughbred would be used for pet food, since the meat consumed by humans is a certain breed of horse raised specifically for that purpose. The slaughter of no longer useful imported breeding stock and many domestic Japanese Thoroughbreds is not uncommon. Shortages of land and the high cost of maintaining a pensioned horse are reasons slaughter is considered an alternate. As in the U.S., where slaughter is also an option available for horse owners, a number of organizations are attempting to provide homes for retired and pensioned racehorses, stallions, and mares. The Japan Racing Association funds one program that currently benefits 90 horses.

Among the people Bayer met and spoke with while trying to learn of Ferdinand's fate was Toshiharu Kaibazawa, who worked as a stallion groom at Arrow Stud during the horse's years there. He called the former champion "the gentlest horse you could imagine. He'd come over when I called to him in the pasture. And anyone could have led him with just a halter on him. ... He'd come over to me and press his head up against me. He was so sweet."

"I want to get angry about what happened to him," Kaibazawa added. "It's just heartless, too heartless."

http://www.humanesociety.org/news/press_releases/2016/06/nat-horse-racing-adv-council-formation-062216.html

The Humane Society of the United States forms national horse racing advisory council

After constructive discussion with other members of the Coalition for Horse Racing Integrity on animal welfare issues and working with thoughtful and progressive leaders committed to elevating the welfare standards in horse racing, The Humane Society of the United States announced the formation of its HSUS National Horse Racing Advisory Council. The council is composed of industry professionals and specialists who continue to promote higher animal welfare standards within the scope of their involvement in horse racing.

"The HSUS is serious about its responsibility to engage with sensible leaders within different industries where there are animal mistreatment issues to find a pathway for reform," said Wayne Pacelle, president and CEO of The HSUS. "Everyone who makes or has made a living from the horse racing industry has a moral obligation to take all reasonable steps necessary to protect and enhance the welfare of the equine athletes who are the heart and soul of the sport and the business of horse racing."

Joe De Francis will chair the council. A long-time animal advocate, he is the former CEO and controlling shareholder of the Maryland Jockey Club, which is the corporate parent of Laurel Park and Pimlico Race Course (home of the Preakness Stakes, the middle jewel of Thoroughbred racing's Triple Crown).

In addition to DeFrancis, council members include a diverse set of stakeholders within the industry, including Jim Gagliano, Stacie Clark-Rogers, Allen Gutterman, Joe Gorajec, Staci Hancock and Chris McCarron.

"I am both honored and excited to be working with The HSUS and with the outstanding and dedicated individuals who will comprise the council," said DeFrancis. "I have every expectation and confidence that the council will be a catalyst for the enactment of federal policies for the betterment of horse racing, to the benefit of all involved: horses, industry participants and fans."

Marty Irby, senior director of rural outreach and equine protection at The HSUS, said: "The establishment of our National Horse Racing Advisory Council is a tremendous step forward for the welfare of equines, the promotion of humane practices and standards both on and off the track, and for the economic vitality and future of the horse racing industry. We are grateful for the opportunity to work with each of these dedicated professionals who recognize the problems that must be solved and want the sport to thrive and flourish, while maintaining the highest standards of animal welfare."

The formation of the council follows the recent release of Pacelle's latest book, The Humane Economy: How Innovators and Consumers are Transforming the Lives of Animals, which delves into the revolution in American business and public policy that is changing how we treat animals and conduct commerce. The book includes an in-depth discussion of how consumer demand for animal welfare improvements is transforming the animal entertainment model.

“The horse racing industry should no longer be an outlier in the humane economy,” added Pacelle. “It’s time for the industry, and the Congress, to adopt a set of independent rules to end doping of horses.”

Media Contact: Stephanie Twining, 301-258-1941, stwining@humanesociety.org

http://www.jockeyclub.com/default.asp?section=Advocacy&area=10

Equine Injury Database

Equine Injury Database

Click here for participating racetracks.

Click here for facts about the EID.

The Equine Injury Database™ is the Thoroughbred industry's first national database of racing injuries. Launched by The Jockey Club in July 2008, the Equine Injury Database seeks to:

- identify the frequency, types and outcome of racing injuries using a standardized format that will generate valid statistics
- identify markers for horses at increased risk of injury
- serve as a data source for research directed at improving safety and preventing injuries

Racetracks, racing organizations and training centers interested in signing up for the Equine Injury Database should contact Kristin Werner Leshney at kleshney@jockeyclub.com or (859) 224-2720.

The following table presents the comparable fatality rates based on the updated analysis of data collected in the Equine Injury Database.

Thoroughbred Only								
Calendar Year	**2009**	**2010**	**2011**	**2012**	**2013**	**2014**	**2015**	**2016**
Rate	2.00	1.88	1.88	1.92	1.90	1.89	1.62	1.54

https://www.thoroughbredracing.com/articles/theyre-embarrassed-already-real-humiliation-next-these-regulators/

They’re embarrassed already, but is real humiliation next for these regulators?

Joe Gorajec | MARCH 14, 2018 |
2 Comments

SHARE

Penn National Race Course in Pennsylvania: the four horsemen in the case had been operating in the state without incident for years

The news of four horsemen suing Tom Chuckas, Thoroughbred Bureau Director of the Pennsylvania Horse Racing Commission, should be of interest to all racing participants across the globe.

This lawsuit involves the authority and tactics of regulators, along with due process protections afforded licensees. These are the type of issues that arise repeatedly everywhere there is a starting gate and a finish line.

This case begins with suspicions of hidden ownership and program trainers (hiding the identity of the actual trainers). Regulators then issued subpoenas and suspensions. Those actions turn into charges of unjustified, unprivileged, and unlawful acts of a Pennsylvania regulator towards its horsemen.

I'll first focus on two critical points: the appropriateness of the subpoenas and the suspensions that followed when four horsemen did not submit the requested information.

The subpoenas

On October 13, 2017, the four horsemen in question - Marcos Zulueta, Juan Carlos Guerrero, Silvio Martin and Sean Mitchell - were served subpoenas by the commission that, according to court filings, were based on anonymous complaints of hidden ownership and program trainers. The requested information included documents covering a three-year period. The specific information included phone records, emails, tax returns and bank statements. Also requested was the location of these trainers' horses at any given time over that three-year period.

The subpoenas allowed 20 days to gather and produce this information and informed the horsemen that failure to comply might result in the suspension of their licenses. The horsemen, believing that the subpoena was unreasonable, overbroad, and oppressive, submitted a motion to quash/amend the subpoena.

In my 25 years as the Director of the Indiana Horse Racing Commission, I have never known of this type of regulatory reaction (or, more precisely, overreaction) to anonymous complaints.

I asked attorney Robin Babbitt to opine. Mr. Babbitt has served as the attorney for Indiana Horse Racing Commission staff since 1994, when pari-mutuel wagering began in the state. Over the course of his years as outside

counsel, Mr. Babbitt never lost a court case representing commission staff while I served as Indiana's Executive Director.

Regarding the issuance of the subpoenas, Mr. Babbitt said, "An administrative subpoena is not something you can take lightly. In regulation you must be careful about laying a foundation for any action you take. This was a situation where there may have been a suspicion of a violation, but that can be a problematic basis upon which to issue a subpoena."

How do you lay that foundation?

Complaints of either hidden ownership or program trainers are not uncommon. In Indiana, we averaged two or three such complaints every year. Our first response would be to collect information to determine if one of our rules had been violated.

Investigators would speak with licensees and others who might possess relevant information. Often, former employees, exercise riders and feed suppliers were just some of the people who provided valuable information.

Stable gate records would be reviewed. These records include the flow of horses in and out of the stable area along with the driver's name and license plate number of the vehicle. Verifying ownership of these vehicles has led to relevant evidence in prosecuting program trainers.

Investigators would order and scrutinize certain winner's circle photographs. We've been surprised at the number of times this has aided our investigation.

These are just a few steps we took. If, after doing our due diligence, we had reason to believe that a violation may have occurred, we would interview the

suspected individuals ‘on the record’ in a tape-recorded interview. We would often follow up the interview with a written request for information.

In Indiana, we would never – ever – issue a subpoena based solely on ‘anonymous’ complaints.

In Pennsylvania, the lack of laying such a foundation was not the subpoena’s only deficiency.

Any information requested in a subpoena must be related to the potential violation. That information must be reasonable, and the person must be given sufficient time to respond. The commission’s subpoena was problematic in all three areas. It would be difficult to get one year of phone records in 20 days, but three years? And how to you track the whereabouts of a stable full of horses retroactively for a three-year period?

The suspensions

Despite their plea to amend the subpoenas, the four horsemen were suspended without a hearing on November 7, 2017.

I believe that this was an extraordinary action. Or, yet again, overreaction.

An immediate suspension without a hearing, also known as a summary suspension, is usually reserved for licensees that pose an immediate threat or danger to the sport or its participants.

Where was the immediate danger? These horsemen had been racing without incident in Pennsylvania for years. None of them had been charged with a rule violation. By simply not complying with a request for information they’re out of business? So much for reasonable enforcement.

Upon an appeal of these suspensions, a hearing was held in front of the full commission on November 29, 2017. The commission subsequently upheld the suspensions and refused to quash or modify the subpoenas.

The Commission gets schooled

The next round in this saga was held on January 11, 2018, in an argument between parties in the Commonwealth Court of Pennsylvania before President Judge Mary Hannah Leavitt.

The venue change from the commission office to the court room was the result of an Application for Stay by the suspended horsemen. This was their opportunity to convince a judge to lift their suspensions temporarily, until a final decision could be made in their case.

My experience in Indiana is that the granting of a stay by a court is an uphill battle for horsemen. Few licensees challenged the Indiana commission in court for a stay. All were denied.

The difficulty in obtaining a stay is meeting the demanding criteria.

Judge Leavitt outlined in her order the criteria the Pennsylvania Supreme Court had established for the granting of a stay:

- The petitioner (horsemen in this instance) must make a strong showing that he is likely to prevail on the merits.
- The petitioner has shown without the requested relief he will suffer irreparable harm.
- The issuance of a stay will not substantially harm other interested parties in the proceedings.
- The issuance of a stay will not adversely affect the public interest.

In her opinion dated January 12, 2018 (the day after the hearing), Judge Leavitt ruled in favor of the horsemen on every criterion.

One finding in Judge Leavitt's decision does not bode well for the Pennsylvania Horse Racing Commission. In a signal of what her final decision may be, she said that the horsemen had made "a substantial case on the merits".

It is an understatement to refer to this particular language as an embarrassment for the Commission.

These horsemen are back racing, but damage, both financial and to their reputation, has already been done. These horsemen had been suspended for over two months, unable to earn a living, based on suspensions that, apparently, should have never been issued.

What happens next?

On February 13, 2018, these same four horsemen filed in United States District Court a complaint against Thomas Chuckas, Thoroughbred Bureau Director of the Pennsylvania Horse Racing Commission.

The complaint alleges, in part:

As a result of Chuckas's unjustified, unprivileged and unlawful acts towards Plaintiffs, they have been deprived and continue to be deprived their constitutionally protected property interest in their trainer's license and their reliance on that license to pursue their chosen profession. These actions also deprive Plaintiff of their liberty right to pursue their chosen profession. Defendant's aforesaid unlawful conduct was knowing and intentional and done with malice.

I have no idea whether this lawsuit has merit.

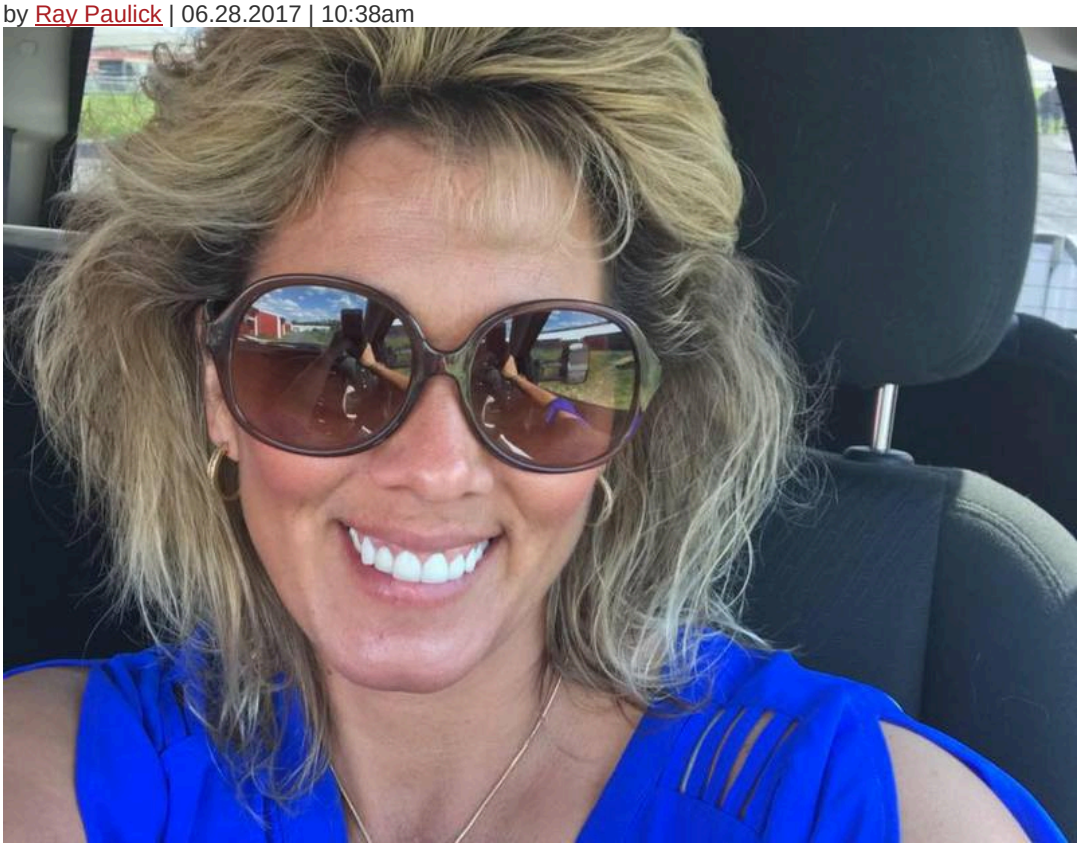
It does appear that its outcome will determine if the commission is merely embarrassed by this whole episode - or humiliated.

Joe Gorajec has spent his entire adult life in the racing industry and served as the executive director of the Indiana Horse Racing Commission for 25 years (1990-2015). He is also a former chairman of the North American regulators' trade association, the Association of Racing Commissioners International (2008). Now semi-retired, he spends his time consulting, writing and gardening at his central Indiana home.

https://www.paulickreport.com/news/ray-s-paddock/trainer-beattie-rojas-trial-almost-everybody-illegally-treated-horses-race-day/

Trainer Beattie At Rojas Trial: 'Almost Everybody' Illegally Treated Horses On Race Day

by Ray Paulick | 06.28.2017 | 10:38am



Stephanie Beattie testified Tuesday at the trial of Murray Rojas in Harrisburg, Pa.

Stephanie Beattie threw fellow horsemen under the bus during her testimony for the prosecution Tuesday afternoon at the federal trial of Murray Rojas, a former rival for leading trainer honors at Penn National in Grantville, Pa.

Beattie admitted she routinely had her horses illegally treated with therapeutic medications on race day by the same veterinarians who counted Rojas as a client.

“Almost everybody did,” Beattie said of the practice. “Ninety-five to 98%. It was a known practice. We wanted to win and they weren't testing for those drugs at that time.”

Beattie, 46, won enough races to be Penn National's leading trainer on three occasions. In 2009, her best year, she won 222 races from 811 starts for earnings of $3.4 million. The previous year, when she won 212 races from 612 starts, she had a win percentage of 35 percent.

But it is two-time Penn National leading trainer Rojas, not Beattie, who is on trial for wire fraud, conspiracy and misbranding of prescription drugs. Assistant U.S. Attorney William Behe has laid out a case with testimony and documents from racing officials, veterinarians and vet assistants alleging Rojas requested and received race-day treatment of horses in order to win purse money, then had billing and treatment records falsified to conceal the cheating.

Beattie is among numerous individuals at Penn National under investigation by the Federal Bureau of Investigation. She resisted cooperating with the FBI at first, Beattie testified, even after Special Agent Bruce Doupe told her, “If you don't want to talk, I'll come to your house at 4:30 in the morning, handcuff you and put you in jail for a very long time.”
Finally, Beattie said, after spending more than $60,000 on legal advice, she decided to cooperate with authorities, submitting to numerous interviews and even wearing a recording device on their behalf.

Despite admitting to years of rule violations in multiple states, Beattie has not been sanctioned by any racing commissions and has faced no criminal charges. It has hurt her business, as shown by a 2016 record of 14 wins from 111 starts and earnings of $217,655.

“This investigation has made things tough for me,” she said.

Beattie also said she has stopped cheating with race-day treatments.

Beattie explained how veterinarian Kevin Brophy established an order form for trainers to fill out their race day medication requests. She said her lists regularly included Kentucky Red, Estrone and Amicar – substances that are not permitted within 24 hours of a race.

Beattie testified that Brophy and other veterinarians informed her of which drugs the state's testing lab was not testing for.

On Monday, Brophy's associate veterinarian, Fernando Motta, testified that Rojas regularly requested and received treatments of Robinul and Estrone on race day for her

horses. Motta beat the test for Robinul, he testified, by administering a lower dose and changing the route of administration to intravenous from intramuscular.

Under cross examination by Robert Goldman, attorney for Rojas, Beattie admitted she never secretly recorded Rojas admitting she had her horses illegally drugged. “There wouldn't be, because we don't talk,” Beattie said.

“You don't like her, do you?” said Goldman, who then revealed that Beattie made fun of Rojas by dressing up like her at a Halloween costume party.

Goldman then recited Beattie's history of medication violations, dating back to her earliest years as a trainer, including a 2005 suspension at Charles Town in West Virginia when officials searched her vehicle and discovered loaded syringes.

Goldman asked: Why did she have injectables?

Beattie responded: “I was giving medication at Lasix time, like everyone else was.”

Beattie denied under oath that she would have shock wave therapy performed on a horse on race day and then have her veterinarian turn in a false name. She said, however, it was “common practice” for horses shipping in to have received shock wave therapy that same day.

She also said she never directed her jockeys or stable employees to use electrical devices, commonly known as buzzers or batteries, to shock horses both during morning workouts and races, as alleged by her former boyfriend and training partner, David Wells. Wells pleaded guilty to charges of rigging a race in a deal with federal prosecutors.

“Did I ever ask them to, no,” Beattie said. “Does it happen at every racetrack, yes. But I never told my jockeys to do it.”

This entry was posted in NL Article, Ray's Paddock and tagged David Wells, drugs in horse racing, Fernando Motta, Kevin Brophy, murray rojas, penn national, Pennsylvania corruption, pennsylvania horse racing, pennsylvania racing, Pennsylvania trial, Ray Paulick, stephanie beattie, William Behe by Ray Paulick. Bookmark the permalink.

https://www.thoroughbredracing.com/articles/what-you-need-know-about-landmark-new-horse-racing-integrity-act/

What you need to know about the landmark Horse Racing Integrity Act

Joe Gorajec | JULY 17, 2017 |
48 Comments

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The new Horse Racing Integrity Act would provide the uniformity in regulation that is the accepted standard in nearly all professional sports.
Photo: NYRA.com

Ask a horse owner or trainer in the United States about racing's anti-doping program and you're likely to receive a quizzical look or a blank stare. That's because 'anti-doping' is not a term most of us associate with horse racing.

If you get any response at all, it's likely to be, "We have a drug-testing program." And indeed, racing does have such a program.

Protecting the integrity of the sport, however, involves much more than just drug testing.

Under a Federal bill that introduced in May (which built upon a similar bill from 2015) Congress would empower the **United States Anti-Doping Agency (USADA)** to take the reins of a new, private, not-for-profit anti-doping authority to create and manage a comprehensive nationwide anti-doping program for horse racing. The proposed legislation, formally known as H.R. 2651, is named the **Horse Racing Integrity Act of 2017 (HRIA)** and it was introduced in the House of Representatives by Rep. Andy Barr of Kentucky and Rep. Paul Tonko of New York.

This bill is materially similar to the version introduced by the same sponsors in 2015, but it has been enhanced in a number of respects. The most significant change is that the scope of the national anti-doping program has been expanded to include Standardbred and Quarter Horse racing. Another important change is a prohibition on administration of any medications on race day, including furosemide (Lasix).

I would strongly recommend that any person with a stake or an interest in the sport of horse racing become familiar with this legislation. The complete 46-page bill can be found here.

I am providing a brief overview of the bill's most important components, along with a description of the status quo. I have included my thoughts on how the new legislation might be applied. In the spirit of full disclosure, I have been, and remain, a staunch advocate for this legislation.

Authority and scope

The legislation would require the creation of an entity named the **Horseracing Anti-Doping and Medication Control Authority** (the 'Authority'). That entity

would be governed by an independent board of directors, a majority of whom come from USADA. It would be responsible for developing and enforcing a national uniform anti-doping and controlled medication program for horse racing.

The program would apply to all Thoroughbred, Standardbred, and Quarter Horse racing. The Authority's jurisdiction would cover all horses and participants involved with racehorses of the three breeds. This would include owners, trainers, veterinarians, grooms, etc.

The Authority's anti-doping and medication control program would supersede individual state racing commission's rules and the Authority would be the sole enforcement authority for all matters covered by the program. This control is limited to the anti-doping and medication control program and does not extend to other matters traditionally governed by state commissions, such as race dates, licensing, and investigating and issuing penalties for all other types of rule violations.

The Authority would be subject to limited oversight by the **Federal Trade Commission (FTC)**. The FTC will be responsible for approving the Authority's rules (after public comment), selecting administrative law judges to hear appeals of the Authority's sanctions and acting as the final appellate body in sanctions matters.

Structure

The Authority will be governed by a 13-member board of directors. The board will consist of the Chief Operating Officer of USADA, six directors from the USADA Board, and six individuals, appointed by USADA from nominees of a variety of equine industry constituencies. Each of the six 'at large' industry board seats is earmarked to people with demonstrated expertise in a variety of areas, such as veterinary treatment of race horses, training race horses, jockeys/drivers, etc.

To ensure independence, all directors will be subject to strict conflict-of-interest standards.

Horse racing expertise

The Horse Racing Integrity Act requires the Authority to establish one or more standing advisory or technical committees in establishing the anti-doping program.

The racing industry would be well served to have the **Racing Medication and Testing Consortium (RMTC)** appointed as the primary standing advisory committee. Established in 2002, the Consortium is an organization that represents 23 racing stakeholder groups. Most of the progress made by the racing industry regarding medication and testing over the past decade has been the result of the efforts of the RMTC.

In order to strengthen the industry's most important advisory committee, the RMTC should expand to include two additional areas of expertise: human anti-doping and international horse racing.

Human anti-doping expertise should be present at all levels of the Authority's structure, including the ground floor of rulemaking and policy development.

The added value of international perspective is consistent with one of the goals of the legislation. The legislation specifically states that "…rules, procedures and enforcement policies should be implemented consistent with internationally accepted best practices ..."

Uniform anti-doping rules

The Horseracing Integrity Act requires the Authority to establish a body of rules for its anti-doping and medication control program, including lists of permitted and prohibited medications, lab accreditation and testing standards, adjudicatory procedures and sanctions.

Currently, most medication and testing rules begin as recommendations from the RMTC. From there, two committees of the **Association of Racing Commissioners International (RCI)** - the Drug Standards and Practices Committee and the Model Rules Committee – consider any recommendations. If approved, the RCI Board of Directors votes to adopt the recommendations as model rules. From that point, to achieve uniformity, each individual state commission (of which there are over 30) must promulgate the model rule.

The state-by-state approval process has frustrated industry stakeholders and fans for decades. Many model rules take years to promulgate – while some states never even try to pass certain rules.

The best way forward under the legislation would be for the Authority to receive recommendations directly from the RMTC and subsequently commence its rulemaking process. This streamlined process would still allow for further opportunity for public comment. Any rule approved would thus become effective simultaneously in all states.

In other words, true uniformity.

List of permitted and prohibited substances

The legislation requires the Authority to develop, maintain, and publish a list of permitted and prohibited substances and methods. The legislation sets the RCI's Uniform Classification Guidelines and Foreign Substances and the Prohibited List of the **World Anti-Doping Agency (WADA)** as a starting point but also makes it clear that the Authority has full latitude to make its own rules (subject to FTC approval).

The Authority will have a head start in providing these required lists. The RCI classification guidelines are current, time-tested, and have served the industry well for over two decades. In December 2016, the RCI adopted a Prohibited Substance List designed after the WADA code.

Under the Authority, the industry will not be left waiting for several years as states attempt local adoption. The Authority would make these lists effective simultaneously in all states.

Laboratory accreditation and testing standards

The legislation requires the Authority to establish procedures, standards and protocols for accredited laboratories. The Authority may extend interim accreditation to those laboratories accredited by the RMTC. Currently, ten of the 14 laboratories conducting testing on race horses in the U.S. are accredited by the RMTC.

The current accreditation process focuses on testing procedures and protocols. In short, laboratories are accredited for what they are capable of doing instead of the work they actually perform for their clients, the commissions.

The deficiency of this accreditation process is one of authority. The RMTC lacks the authority, as does any currently constituted national body, to require laboratories to find certain drugs at a mandated concentration. That authority currently resides with each state racing commission. For that reason, there is a perception of a wide disparity in drug testing between states.

Under the HRIA, the Authority would establish such a national uniform standard. Compliance would be mandatory and uniformity would be achieved.

Testing and sampling

The HRIA permits each state racing commission to choose its own laboratory as long as the laboratory is, and remains, accredited.

In all likelihood, the laboratories currently testing horse racing samples would continue to do so. The primary difference, however, is that these laboratories would be required to perform up to the standards set by the Authority. Failure to meet the Authority’s standards would place a laboratory’s accreditation at risk.

The Authority would also establish standards for **out-of-competition testing (OOCT)** that would be applied uniformly in all states. The OOCT program would require the disclosure of the whereabouts of all horses in training at all times. This would greatly improve the current situation where only a handful of states have an effective deterrent for blood doping.

Investigations

The legislation gives the Authority the responsibility to investigate any violation of its anti-doping rules.

State commissions now have similar authority that is limited to its provincial borders. Most commissions have a Director of Security and/or investigators who conduct investigations into the conduct of licensees and other persons. State investigators have broader responsibility because their investigative authority spans rule violations of any type, whereas the Authority would be limited to its anti-doping rules.

USADA's approach to regulation is highly principled and integrity based. Its independence assures an even-handed, show-no-favorites application of its authority to investigate and sanction. An ideal approach, at least for the most important investigations, would be for the Authority to partner with USADA to utilize its expertise and time-tested strategies and combine them with the specialized knowledge of local commissions' boots-on-the-ground investigators.

Due process

Under the legislation, the Authority has the power to sanction individuals who violate its anti-doping rules. The HRIA requires the Authority to establish a schedule of sanctions for violations and rules for due process that include impartial hearings.

The disciplinary processes that state commissions and USADA currently follow have one procedure in common: the ability to accept an agreed upon sanction. Most trainers accept the RCI Uniform Model Rule for Penalties either as the result of a stewards' hearing or in lieu of a stewards' hearing. Consequently, a very small percentage of alleged violations are appealed.

Presently, the adjudication process for appeals varies from state to state. The most common model allows for state racing commissions to appoint an ALJ (Administrative Law Judge) to conduct an administrative hearing and issue a recommended decision. The commission may adopt, reject, or amend the ALJ's recommended decision. This process is followed in most appeals of an adverse ruling from the Board of Stewards after an initial hearing is provided at the track. In some instances, the initial hearing is by-passed and the first and only administrative hearing is before an ALJ.

A common complaint of the current adjudication process is the perception of a lack of independence. This is the result of ALJs being appointees of the relevant state commission or otherwise affiliated with state government. Under the HIRA, those sanctioned by the Authority will have the right to a speedy appeal covering all the merits of their individual cases, which will be heard by independent administrative law judges not associated with the Authority in any way.

Cost

The cost of the anti-doping program has yet to be determined. Preliminary estimates provided to the **Coalition for Horse Racing Integrity** indicate the additional cost of the new anti-doping program would likely not exceed an average of $60 per start. This estimate assumes that all current funding for anti-doping remains in place. I understand that much of this increased cost comes from the expansion of out-of-competition testing.

Although the cost is expressed in dollars per start, the allocation of cost among stakeholders could vary from state to state. The HRIA provides flexibility in determining the source of funding. The legislation reads as follows:

Each State racing commission shall determine, subject to the applicable laws and regulations of the State, the method by which the requisite amount shall be allocated, assessed, and collected, provided that in no event shall the funds be obtained by means of an increase in the takeout.

Final thoughts

The Horse Racing Integrity Act of 2017 would provide the uniformity in regulation that is the accepted standard in nearly all professional sports. This landmark legislation, however, brings much more than uniform application of standardized rules and protocols. It promotes the principles of integrity, excellence and independence, some of which have been long absent in many racing jurisdictions.

Joe Gorajec has spent his entire adult life in the racing industry and served as the executive director of the Indiana Horse Racing Commission for 25 years (1990-2015). He is also a former chairman of the North American regulators' trade association, the Association of Racing Commissioners International (2008). Now semi-retired, he spends his time consulting, writing and gardening at his central Indiana home.

https://www.thoroughbredracing.com/articles/whats-next-pennsylvania-if-it-doesnt-clean-its-act/

What's next for Pennsylvania if it doesn't clean up its act?

Joe Gorajec | NOVEMBER 07, 2017 |
4 Comments

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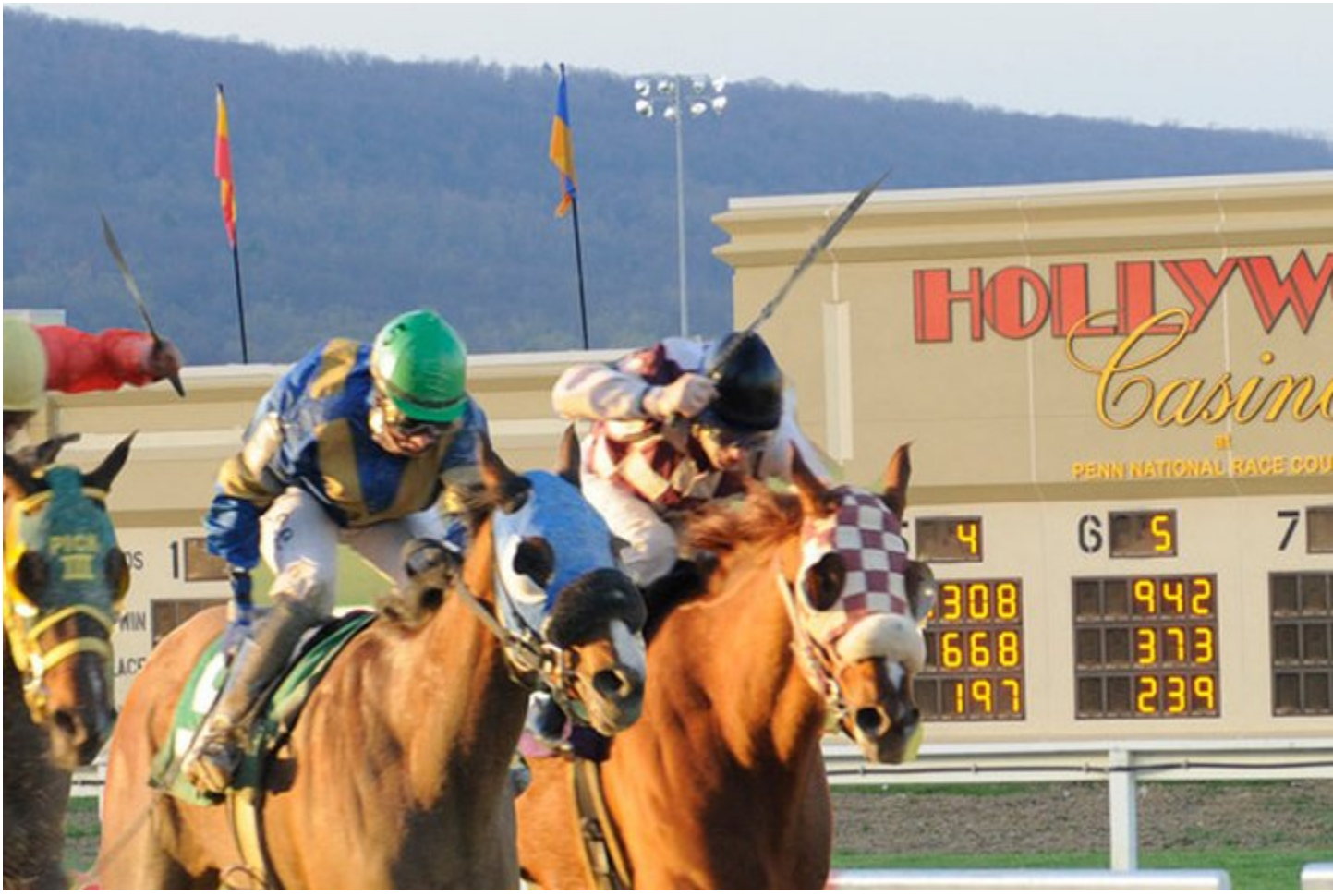

Penn National Race Track: some prosecutions for fraud have involved officials there. Photo: hollywoodpnrc.com

Play the word association game with the term 'Pennsylvania horse racing industry'. Are the most frequent responses 'sleazy' and 'corrupt'?

Unfortunately, Pennsylvania has earned that reputation.

Mr. Stuart S. Janney, Chairman of the Jockey Club, chose to address the troubles in Pennsylvania in his closing remarks at this past summer's Round Table conference in Saratoga Springs, New York.

Mr. Janney said, "What has happened in Pennsylvania recently is disgraceful and sad, especially when you consider that the state is the sixth leading producer of foals and that it hosted approximately 4,000 races and distributed more than $100 million in purses in 2016. Let's start by focusing on the federal trial involving trainer Murray Rojas on charges of fraud, conspiracy, and misbranding of drugs. I think it illustrates what we have to fix and how our problems interconnect.

"Uncontradicted testimony described widespread, in fact, nearly universal, cheating; regulators asleep on the job; a corrupted and ineffectual testing system."

This 'widespread cheating' includes guilty pleas from four veterinarians who treated horses with impermissible medications on race day. This practice, evidently, had been ongoing for over 20 years.

Fraud and conspiracy cases

The corruption in Pennsylvania has not been limited to the case of Ms. Rojas.

In 2014, the clocker at Penn National Race Track pleaded guilty to criminal charges of wire fraud by accepting cash to falsify workouts, thus defrauding the betting public.

Also in 2014, Pennsylvania trainer David Wells was indicted on multiple counts of conspiring to fix races. He was alleged to have administered substances to horses on race day during a four-year period from 2009-2013. Mr. Wells pleaded guilty to one count of "rigging a publicly exhibited contest" as part of a deal with federal prosecutors. He was subsequently ordered to

serve a three-month prison sentence in addition to a mandatory work release program and four-and-a-half years on probation.

In 2015, another Penn National racing official agreed to plead guilty to wire fraud by accepting cash for divulging inside information to trainers involving the entry of horses.

Mr. Janney's Round Table conference remarks did not go unchallenged by the Pennsylvania State Horse Racing Commission. In a letter dated September 26, 2017, to Mr. Janney, Commission Chairman Russell C. Redding characterized Mr. Janney's remarks as "inappropriate and inaccurate", "faulty" and "highly unprofessional".

Mr. Janney's rebuttal can be found here.

Of all Mr. Janney's remarks, the one that most likely hit a nerve was that regulators were asleep on the job.

When prosecutors step in

Mr. Redding counters that, "From day one, this has been a cooperative joint effort between all branches of regulatory and law enforcement … The criminal actions of the veterinarians and horsemen who were successfully prosecuted are reprehensible, but do not confuse that with the actions of the SHRC [the Commission]. There is no evidence of "regulators asleep on the job".

So, were regulators asleep on the job?

Of course, they were.

Mr. Redding just doesn't get it.

Federal prosecutors only involve themselves in these types of rule violations when state regulators are unwilling or unable to effectively carry out their statutory duties to protect the integrity of its racing product.

Simply stated, Pennsylvania regulators wouldn't clean up their own mess, so the federal government stepped in and did it for them.

So, where does Pennsylvania horse racing industry go from here?

It is problematic that most of the people involved in this longstanding and widespread culture of cheating, absent a few individuals, are still involved in racing in Pennsylvania. Once part of this cheating culture, can these people change their ways? Will the newly appointed regulators strive to deter, detect, and forcefully prosecute violations of the rules of racing? Or will they default to the same "hear no evil, see no evil and speak no evil" posture as their predecessors?

What are the remedies if this type of corruption continues?

Slot machine revenue

Is the answer to shut down racing in the state?

If so, this would not be initiated from within Pennsylvania's racing industry. These people are the beneficiaries of the program. Nor would influences outside the state have any bearing on this question. The citizens of Pennsylvania and their elected representative would likely decide. That's

because State lawmakers have the power to direct monies that are derived from slot machines at the track.

The demise of any racing track in the United States would likely be the result of lawmakers discontinuing the flow of slot machine revenue to racing industry. This money, in Pennsylvania and elsewhere, has become the lifeblood of purse funding, and in many cases, is also the revenue that keeps race tracks profitable. Most tracks and horsemen simply cannot rely solely on the revenue derived from pari-mutuel betting.

Should the elected officials in Pennsylvania determine that the racing industry is undeserving of slot machine revenue, its racing program would quickly collapse like a house of cards.

When we play the word association game a decade from now, how will you respond?

I say, “Pennsylvania horse racing industry” and you say, “classy mid-level racing”.

Or “defunct”?

Joe Gorajec has spent his entire adult life in the racing industry and served as the executive director of the Indiana Horse Racing Commission for 25 years (1990-2015). He is also a former chairman of the North American regulators’ trade association, the Association of Racing Commissioners International (2008). Now semi-retired, he spends his time consulting, writing and gardening at his central Indiana home.

https://www.thoroughbredracing.com/articles/why-every-day-groundhog-day-us-racings-rulemaking-process/

Why every day is Groundhog Day in U.S. racing's rulemaking process

Joe Gorajec | APRIL 04, 2018

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In the film Groundhog Day, Bill Murray's character modified his behavior each day in order to pursue his goals, rather than doing the same thing every time

You remember the movie 'Groundhog Day'? Of course, you do! The one where Bill Murray is stuck in Punxsutawney, Pennsylvania. He is living the same day over and over and over again. Every morning he wakes up, desperate to leave Punxsutawney.

For every newly proposed model rule, U.S. racing experiences its own Groundhog Day. We wake up each morning only to confront the same ineffective rulemaking process over and over again, in 30+ different states.

One such example is a set of recommendations made by the Racing Medication and Testing Consortium (RMTC) that, if enacted, would upgrade existing out-of-competition testing rules. The Association of Racing Commissioners International (RCI) voted in December 2016 to approve these recommended rules as a model to be approved separately in each state.

Although I was skeptical, my hope was that all states would rally around this important recommendation and approve it in unison - and within a year.

Lackluster results

I wrote a *TRC* column to that effect in April 2017, titled “Out of Competition testing: after a decade of neglect, what’s next?”

In that piece I opined:

By the way, the new rule is terrific. It is thoughtful, well-crafted, and accompanied by a Prohibited Substance List. This list is modelled after that of the World Anti-Doping Agency (WADA), meaning it is consistent with international anti-doping standards. It greatly expands the number and classes of prohibited drugs. The new list includes, in addition to blood-doping drugs and methods, a broad spectrum of anabolic steroids, peptide hormones, growth factors and related substances. Many of these types of drugs are likely to be detected only in an out-of-competition environment.

Most states can complete the rulemaking process in a year. The start date for this process began when the model rule was adopted by the RCI in December 2016. So, by the end of 2017, we should all know where this is headed.

So where do we stand now – 15 months later.

According to the RMTC’s website, as of March 15, 2018, only seven states have passed this set of rules. These are Arkansas, Delaware, Maine, Maryland, Massachusetts, Pennsylvania and Washington. Four other states are in the process of adoption: California, Colorado, Iowa and New York.

Why such lackluster results?

Because there is no central authority in the U.S. for enforcing national uniformity. The RMTC, which has been the driving force for progress in medication and testing issues for over a decade, does not possess the authority to require compliance. Neither does the RCI, the trade association of racing commissions. It all falls to the individual state racing commissions, which have differing rulemaking processes, personalities and agendas.

There is a perception among racing fans that certain horsemen congregate in racing jurisdictions due to lax regulation regarding drugs and testing. The extent to which this is true is, of course, unknown. I believe that rudimentary logic suggests that this occurs, at least to some degree. Any horsemen seeking an edge with an illegal drug that can only be detected through out-of-competition testing would surely gravitate to states that have little or no such testing.

This situation has been a perennial lament of racing fans.

What saved Bill Murray

The initial out-of-competition rule was drafted in 2006, and some states are just now beginning to implement it. If the system doesn't change, we will be waiting another ten+ years for states to pass the new and improved version.

Is there a way out of this exasperating cycle?

Yes, there is.

Just ask yourself - "What would Bill Murray do?"

In the movie, Murray's character changed his behavior over time. And this is what saved him.

Instead of acting the same way, he modified his behavior each day in order to pursue his goals. He dearly wanted to free himself of the repetitious cycle of Groundhog Day, and, just as important, get his dream girl, played by Andie McDowell.

Can the racing industry change its behavior?

Our goal should be to have a single, national rulemaking process that allows for the expeditious implementation of all medication and drug-testing rules.

We do have a lifeline. It is federal legislation called the *Horse Racing Integrity Act of 2017*.

This legislation, introduced by Congressman Andy Barr (R-KY) and Congressman Paul Tonko (D-NY) calls for a unified rulemaking process the racing industry is lacking. It has the support of over 100 members of the U.S. House of Representatives.

If enacted, this legislation would mandate uniformity in medication and testing rules in all states - just like other professional sports.

The answer to racing's rulemaking dilemma is easy. Just ask yourself, "What would Bill Murray do?"

He sure as hell wouldn't stay in Punxsutawney.

Joe Gorajec has spent his entire adult life in the racing industry and served as the executive director of the Indiana Horse Racing Commission for 25 years (1990-2015). He is also a former chairman of the North American regulators' trade association, the Association of Racing Commissioners International (2008). Now semi-retired, he spends his time consulting, writing and gardening at his central Indiana home.

https://www.thoroughbredracing.com/articles/charles-town-classic-fiasco-response-takes-racing-wrong-direction/

Charles Town Classic fiasco: this response takes racing in the wrong direction

Joe Gorajec | FEBRUARY 25, 2018 |
7 Comments

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Star attraction: Game On Dude winning the $1.2 million Charles Town Classic in 2013. Photo: Coady Photography

This is a case where the cure is worse than the disease. Legislation is currently making its way through the statehouse in West Virginia that would expand its racing commission from three to five members. More importantly, it would mandate that new appointees be "capable and experienced in fields of knowledge relevant to racing". On February 6, 2018, the bill passed in the Senate with a vote of 30-0.

The legislation (aka Senate Bill 393) is likely a well-intentioned response to the recent fiasco of the racing commission's attempt to gut the Charles Town Classic. The state's only Graded race.

On January 23, 2018, in a decision that brought shame and ire on state's regulators, the commission voted to cap the Classic's purse at $300,000.

Its advertised value had been $1.2 million. The immediate backlash from the industry, and, most importantly, West Virginia Governor Jim Justice, who made his displeasure public, virtually assured the issue would be revisited.

Self-regulation

The writing was on the wall two weeks later, when the WV racing commission met to rescind its previous decision.

Evidently, enough damage had been done to prod legislative action.

The increase from three to five members is immaterial. The qualifications mandate, which might look good on paper, is problematic in real life.

The bill, if passed as is, would amount to self-regulation. Which is bad. Very bad. But, first let's look at what the bill says.

Specifically, the bill states, *"Individuals appointed to the commission shall be persons who, by reason of previous training and experience, can be classified as capable and experienced in fields of knowledge relevant to racing."*

One person, qualified as above, would be appointed as a representative for each of the following:

- Tourism
- Thoroughbred horse racing
- Greyhound racing
- Casino (with either race track management or casino experience)
- Veterinary

Two issues are worth raising.

First: potential conflicts of interest. Nothing in the bill prohibits the appointment of a commissioner who has a financial relationship with a person or entity he or she regulates.

For example, can the veterinarian appointed to the commission work on horses racing at the track? Is the casino representative going to be an official of the track or casino?

I am familiar with the Horse Racing Integrity Act of 2017. All parties involved in its drafting of that federal legislation insisted that authority to regulate be entrusted to an independent body. The West Virginia bill takes the industry in the opposite direction.

Each state's pari-mutuel statute across the country varies on whether to allow individuals who are "in the business" to serve as commissioners. Regardless of prohibitions against such situations, the Governor controls this issue by the appointments that are made.

The other issue is having a commission where the majority represents certain segments of the industry.

No commissioner should represent ***any*** segment of the industry. Commissioners should represent the public in the state they serve. They are regulators whose duty requires them to protect the integrity of the sport and the safety of the participants – human and equine. Every commissioner should be mindful to consider the health and prosperity of the industry at-large.

It's only human nature, however, to view the world from the perspective of your experience. When a person is placed on a commission to represent a special interest, that's what he or she will do.

I once had had a commissioner in Indiana, who had ties to the Standardbred industry, declare on a split vote something along the lines of – *"I'm a Standardbred guy and I'm voting for the Standardbreds"*. What an embarrassment. This was supposedly on impartial panel. Even the Standardbred horsemen at that meeting were embarrassed.

It's been my experience as a horse racing regulator in Indiana for 25 years, that, all things being equal, it is best to have commissioners without ties to the industry. I've know a handful of "in-the-business" commissioners who have served their state well – with honor, dignity and objectivity.

But these folks are the exception, not the rule.

It is interesting to note that the commissioner who initiated the purse cut to the Charles Town Classic, Ken Lowe Jr., is certainly qualified under the Senate Bill 393 to retain his seat on the commission. He is a former racehorse owner who served as the HBPA President at Charles Town Race Track.

It's one thing to allow conflicted or potentially biased individuals to serve on a racing commission. It's another to mandate it.

That's what Senate Bill 393 does.

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https://www.paulickreport.com/news/ray-s-paddock/combating-culture-cheating/

Combating A Culture Of Cheating: A Matter Of Trust

by Joe Gorajec | 01.18.2017 | 8:50pm

(First in a two-part series.)

Horse racing has a culture of cheating.

Its problem is drugs. It's what the public calls doping. The methods differ, as do the drugs.

At one end of the spectrum, horsemen and veterinarians inject horses on race day with a wide variety of drugs or other foreign substances.

This practice is one of racing's dirty little secrets, although it's no secret to those who work in the stable area of a racetrack.

There exists a thick, bright line in racing regulation known as the 24-hour rule. This rule prohibits the administration of any drug or foreign substance, other than the anti-bleeder medication Salix (furosemide), within 24 hours of a horse's race. A few states have specific exceptions. This bright line is crossed with such regularity that its practitioners have become blind to its existence.

Horsemen rationalize this cheating by convincing themselves that they are just “helping” the horse. Many of the race day injections are to manage pain, mitigate bleeding, or calm a fractious horse. Several of these drugs are endogenous to the horse and go undetected in post-race testing. The “helping” of the horse is code for “it's not cheating if you don't get caught.”

An example of this culture run amok is the investigation and prosecution of veterinarians and horse trainers at Penn National racetrack by the United States Attorney's Office for the Middle District of Pennsylvania.

On March 27, 2015, The U.S. Attorney's Office issued a press release announcing that four veterinarians had been criminally charged (and had agreed to plead guilty) to conspiracy to unlawfully administer drugs to race horses. The release succinctly describes this activity as follows:

According to the charges, trainers allegedly placed orders for drugs and the defendants after administering the drugs, backdated the billing records to avoid detection. The defendants allegedly submitted false veterinarian treatment reports to the State Horse Racing Commission omitting from those reports any reference to the drugs administered to horses at the track on race day. The filing of these reports and the backdating of billing records were, allegedly, to further the conspiracy by concealing the illegal activity. These acts had the potential to defraud other owners and trainers whose horses were entered in the same race and defrauded the betting public as well.

This type of activity has been common practice on racetracks for decades.

A deterrent to this type of routine race day cheating exists in a national model rule requiring the administration of Salix by a third party. Under the model rule, a veterinarian employed or contacted by the state racing commission or the racetrack administers Salix. The rule is designed for the express purpose of keeping practicing veterinarians (who work for the trainer) out of the horse's stall on race day. Third-party

Salix programs are effective in deterring widespread corrupting influences. It will not, however, stop anyone determined to get an edge on race day.

The status of the third-party Salix model rule is found on the website of the Racing Medication and Testing Consortium (RMTC): 18 states have adopted, 16 states have not.

Far more malevolent and injurious to the sport is what is likely occurring at the other end of the spectrum. That is the use of sophisticated performance enhancing drugs (PEDs) which taint the upper echelon of the sport. The methods for cheating at this level often do not involve simply sneaking into a stall on race day with an illicit drug in a syringe.

Best horses, best races

When it comes to major races, prominent Thoroughbred owner Bill Casner said, “I'll promise you that there will be some horses helped with PEDs (performance enhancing drugs).

Bill and Susan Casner

“It would be incredibly naïve for anyone to think that this (PEDs) does not exist in our game. And especially at the high end because the high end is where all the money is at,” said Casner.

The “high end” to which Casner refers consists of approximately 450 Graded stakes races which are held annually at various tracks. These races make up less than two percent of the approximately 40,000 Thoroughbred races conducted annually. The purses for the Graded stakes, however, account for over $150 million, or 15 percent, of the $1 billion distributed annually.

Casner's path to the pinnacle of the sport is not well traveled. From galloping horses in the early 1960s at Sunland Park in New Mexico, to hoisting the Kentucky Derby trophy in the winner's circle in 2010 as the co-owner of Super Saver, he has witnessed the sport from the inside as few others ever have. His most memorable score, however, did not occur on U.S. soil. In 2009, his WinStar Farm homebred gelding, Well Armed, romped to a 14-length victory in the $6-million Dubai World Cup.

A member of The Jockey Club and the Water Hay Oats Alliance (WHOA), and former chairman of the Thoroughbred Owners and Breeders Association (TOBA), Casner has long been an advocate for clean racing.

Casner claims that a small but significant improvement in performance from illicit drugs is sufficient to drive a trainer to cheat. Especially if they believe their fellow trainers are likely doing the same.

At the elite level of the sport, the incentives to cheat are at their highest, and the financial incentives go well beyond the traditional trainer's purse percentage.

“There is too much difference in the amount of money between a Grade I and a Grade II horse. Grade I horses are stallions. Grade II horses are regional stallions. And Grade III horses stand in [minor state-bred programs],” said Casner.

Nine of the top 10 stallions on *Blood-Horse* magazine's 2016 General Sires List won at least one Grade I stakes during their racing career; 2017 stud fees for these nine horses range from $60,000 to $300,000.

“The share values and the breeding rights that trainers receive become these huge portfolios for them. And this is where they really make their money,” said Casner.

“If EPO can give a horse two or three lengths extra – that is astronomical,” said Casner. “How many races are lost by a nose? How many races are lost by a head? A neck? A length? A length-and-a-half? Two lengths?”

“Performance enhancing drugs work. They make already great athletes, human or animal, even greater,” said Jeff Novitzky.

A matter of trust

Few people in the world have the gravitas and insight to opine on the culture of cheating in sports as does Jeff Novitzky. Once referred to by *TIME* magazine as the Eliot Ness of baseball's “steroid era,” Novitzky has been on the frontline of exposing the cheating of fallen icons such as Barry Bonds, Roger Clemens, Marion Jones, Tim Montgomery, and Lance Armstrong.

Jeff Novitzky

Novitzky served as a federal agent for 15 years with the IRS Criminal Investigations Division, followed by seven years as a special agent for the Food and Drug Administration. He is now the vice president of Athlete Health and Performance with the Ultimate Fighting Championship (UFC), the world's largest mixed-martial arts fight promotion.

Novitzky spoke at the 2016 Jockey Club Round Table Conference in Saratoga Springs, N.Y. One of the most salient takeaways from his presentation involved his personal interaction with users of performance enhancing drugs.

Speaking of the numerous investigations he conducted, Novitzky said, “Throughout those investigations, I got to interview 150 to 200 high profile professional athletes who chose to use performance enhancing drugs. In addition to asking them about where they got the drugs, how they paid for them, and how they were distributed, I always took the opportunity to ask them why they chose to use. It wasn't anything special about me, but I was in a position and they were in a position to be compelled to tell me the truth. In fact, we prosecuted several athletes for not telling the truth.

“So I think in the majority of those 150, 200 conversations I got the truth, and I always took the opportunity to ask, ‘Why did you choose to use performance enhancing drugs? What led you down that path?'

“And the answer I got an overwhelming majority of the time, it came down to one word, and that word was trust.”

Novitzky added: “They said, ‘I didn't trust that my teammates weren't using. I didn't trust that my opponents weren't using, and maybe, most importantly, I didn't trust that my sport's governing bodies cared enough because of the weakness of the program or in some cases total lack thereof.'”

A report titled *Stakeholder Input*, released November 2016 by the Association of Racing Commissioners International, contains a survey in which the issue of horsemen's “trust” is addressed.

In response to the statement *“Doping with designer drugs is rampant,”* 58.1 percent either totally agreed or somewhat agreed. By nearly an identical margin, 57.2 percent of the respondents indicated they either totally or somewhat agreed with the statement *“Most people I know cheat.”*

If horsemen's trust in the effectiveness of their anti-doping program is the determining factor that drives cheating, the racing industry has reason for alarm.

A lack of will

Horse racing in the U.S., to a large degree, is a sport that abides a culture of cheating.

The unwillingness of regulatory bodies to implement common sense deterrents has led to, in many states, risk-free cheating. A clear-cut example is out-of-competition testing.

Out-of-competition testing occurs days, weeks, or months before a horse's race, or between races. Its goal is to determine if horses are training on prohibited drugs that can enhance performance on race day.

Blood doping drugs like Epogen (EPO) cause the body to produce additional blood cells that allow the athlete – horse or human – to increase their oxygen carrying capacity. The drug can only be detected for approximately three days after administration. The performance-enhancing effects will last up to 120 days – which is the life span of a red blood cell.

Many anabolic steroids are like blood doping drugs in that the performance enhancing effects far exceed the short time frame of detection.

States with little or no out-of-competition testing have invited their horsemen to cheat with impunity.

In November 2007, *Blood-Horse* published an op-ed column I wrote on out-of-competition testing. At the time of publication over a year had passed since the development of a method to test for the presence of the blood-doping agent Epogen. Only six states had moved forward to deter and detect this emerging threat by implementing out-of-competition testing.

In the commentary, I hypothesized why the industry had not moved more quickly.

Would some track owners prefer not to endure the inevitable publicity of a successful trainer charged with blood doping? Would some horsemen prefer to not be

inconvenienced by the thought of testing anytime, anywhere, without notice? Are some racing commissions paralyzed by institutional inertia?

Now, 10 years later, we know the answers to all these questions are … yes.

In 2014, of the top 20 states ranked by the number of Thoroughbred races run, 15 conducted little or no out-of-competition testing. These 15 states account for almost two-thirds of all races. Our international counterparts are averaging 10 percent of their testing from out-of-competition sampling, while the U.S. is conducting only 1 percent.

The racing industry's assertion that this failure is due to a lack of funding is disingenuous.

For example, in Indiana in 2015, over 10 percent of the testing for the 120-day Thoroughbred and Quarter horse race meet at Indiana Grand originated from out-of-competition samples. All samples were analyzed for blood-doping agents, a broad spectrum of anabolic steroids, and repartitioning drugs (such as ractopamine and zilpaterol). Samples were taken from horses stabled at the track or at training centers and farms.

The total cost of this program, including sampling and testing, was less than $50,000. To place this in perspective, the cost is less than two purses for maiden special weight races, which at Indiana Grand in 2015 were $32,000 each.

Although Indiana's program is funded by the racing commission, it is also a reasonable expense to be borne by any racetrack or horsemen's association intent on protecting the integrity of its racing program.

The UFC's Novitzky says horse racing's out-of-competition program is a “green light” for cheaters.

When asked what would happen if horse racing's out-of-competition program was applied to human athletics, Novitzky said athletes “would be enhanced to the gills.”

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Thursday: In the second of this two-part series, Gorajek examines the limitations and challenges of testing laboratories, weaknesses of the current state regulatory system, and a possible solution going forward.

Joe Gorajec served as the executive director of the Indiana Horse Racing Commission for 25 years (1990-2015). He is also a former chairman of the Association of Racing Commissioners International (2008).

This entry was posted in Ray's Paddock and tagged american graded stakes committee, anabolic steroids, association of racing commissioners international, Bill Casner, drug testing, drugs in horse racing, epogen, furosemide, Indiana Grand, Indiana Horse Racing Commission, jeff novitzky, lasix, major league baseball, out of competition drug testing, PEDs, penn national indictments, performance enhancing drugs, race day medications, racing medication and testing consortium, rmtc, salix, steroid era, The Jockey Club, Thoroughbred Owners and Breeders

Association, UFC, Ultimate Fighting Championship, Water Hay Oats Alliance, WHOA by Joe Gorajec. Bookmark the permalink.

https://www.cbsnews.com/news/eight-belles-death-sparks-controversy/

CBS/AP May 5, 2008, 7:26 AM

Eight Belles' Death Sparks Controversy

Kentucky Derby: Is horse racing now facing an image crisis?

With the memory of Barbaro still fresh, Eight Belles' catastrophic breakdown Saturday put increasing focus on a sport already trying to overcome a decline in popularity.

Her death has raised thorny issues about the whole thoroughbred industry, including track safety, whether fillies should be allowed to run against colts, and whether horses are bred too much for speed and not for soundness.

Congressman Ed Whitield of Kentucky, who is trying to toughen regulation of horseracing, told **CBS News correspondent Chip Reid** that breakdowns are far more common than people think, and are on the rise. Whitfield said that one reason for the rise is that the big money is not in racing horses anymore, it's in breeding them.

"These horses really are expendable commodities," Whitfield said. "You want to get the most out of them for a short period of time, and hopefully they are good enough to go into breeding."

A prominent animal rights group got involved Sunday, too, criticizing Eight Belles' jockey for whipping the horse and saying the second-place prize should be revoked.

But to horse people, it wasn't all that simple.

"To make it safer, don't race the horses, don't train them, then they'll live good lives out on the farm," Big Brown trainer Rick Dutrow Jr. said.

"But you have to train them for races, you have to run them and that's where the problems start to set in. They have to be asked to run and sometimes in a particular minute, they're asked to run when they're not ready to give it and then it hurts."

While Big Brown's bid to become the first Triple Crown winner in 30 years will certainly gain momentum in the next couple of weeks, Eight Belles and the sight of fans crying in the stands remained a focal point Sunday.

"Filly's Death Casts Shadow over Kentucky Derby," read The New York Times.

"Tragedy mars Kentucky Derby as the only filly dies after race," the Los Angeles Times' Web site said.

Churchill Downs officials were unsure whether there had been a fatality in the Kentucky Derby. Superintendent Butch Lehr said there hadn't been one in his 41 years at the track.

The death of Eight Belles may have been rare because it occurred well after the finish line, but it's just the latest trauma to happen at a major race on national television.

Two years ago, Derby winner Barbaro shattered his fight rear leg at the start of the Preakness, with more than 100,000 people gasping at the site of the undefeated colt in distress as he was led into an equine ambulance. Barbaro was euthanized eight months later after developing laminitis as a result of the injuries.

Dr. Dean Richardson, the veterinary surgeon who tried valiantly to save Barbaro, told **CBS' *The Early Show*** that Eight Belles' injuries were very different from Barbaro's.

"It is extraordinarily for a racehorse to break down the way Eight Belles did after the race is finished," Richardson said.

Eight Belles suffered fractures of both feet. In the left foot, the fracture was so severe it tore through the skin.

"A horse can get around on three legs temporarily. It's impossible for a horse to get around on just its hind legs," Richardson said.

Now, there are more questions about track safety.

Barbaro's demise helped push forward the installation of synthetic surfaces to replace traditional dirt tracks at several tracks, including Keeneland, Santa Anita, Arlington Park, Hollywood Park, Golden Gate Fields, Del Mar, Turfway and Presque Isle. A new on-track injury reporting program seems to indicate the surface is having the desired effect.

Reports by veterinarians at 34 tracks across the country between June 2007 and early this year showed synthetic tracks averaged 1.47 fatalities per 1,000 starts, compared with 2.03 fatalities per 1,000 starts for horses that ran on dirt.

But not everyone is convinced.

"This is a very big issue and needs to be discussed," two-time Derby winning trainer Nick Zito said. "You're changing the whole game. Big Brown ran on dirt yesterday, he's going for history. You can't tell me the Polytrack is history. It's not yet, there isn't enough data yet."

That's not saying Zito and other horsemen are not interested in making racetracks safer for both horses and jockeys.

"If you told me, `Look, we have a device that these horses can run on pillows and never get hurt the rest of lives,' I'd say, `Where do I sign?'" Zito said. "There's injuries on the Polytrack, too. Now you see why I'm saying it's a big issue."

While breakdowns always have been a part of racing, there has been more of an outcry lately calling for drastic action.

People for the Ethical Treatment of Animals (PETA) issued a statement Sunday calling for the suspension of Eight Belles jockey Gabriel Saez. The group also asked for the "revocation of the second place prize."

Saez was riding in his first Kentucky Derby when Eight Belles broke both front ankles while galloping out a quarter-mile past the finish line.

"What we really want to know, did he feel anything along the way?" PETA spokeswoman Kathy Guillermo said. "If he didn't then we can probably blame the fact that they're allowed to whip the horses mercilessly."

A call to the jockeys' room at Delaware Park, where Saez raced on Sunday, went unanswered.

The Kentucky state racing stewards make decisions on suspensions, but there is no racing at Churchill Downs until Wednesday. At that time, the stewards could review a tape of the race if a formal request is made.

Eight Belles trainer Larry Jones disputed any suggestion that his horse had no business taking on the boys.

"It wasn't that, it wasn't the distance, it wasn't a big bumping match for her, she never got touched," he said. "She passed all those questions ... with flying colors. The race was over, all we had to do was pull up, come back and be happy. It just didn't happen."

On Sunday morning, Jones stood next to his Kentucky Oaks-winning filly, Proud Spell, receiving condolences from friends and fellow trainers.

"Got here at 5 a.m.," Jones said. "Got to go on. It's hard, but it's what we do."

Just then, Barbaro's trainer Michael Matz drove past Jones' barn stopped his car and rolled down the window. On Friday, Matz watched another one of his horses, Chelokee, suffer a life-threatening injury in the Alysheba Stakes. He had just returned from Lexington, where the horse was set for surgery Monday to fuse his injured ankle.

"Sorry, Larry," Matz said.

"I know you know what it's like, thank you," Jones said. "How's yours doin'?"

"Doing good, they're going to operate tomorrow," Matz said.

Dutrow was still basking in Big Brown's victory, well aware that an injury can strike at any time.

"No matter what happens, you're always going to see horses break down on the track," he said. "That is part of this game. It's a very sad part of the game, but you have to go through it.

"For people coming out to the track and seeing that, it's got to make them think, `Man, why would I want to go out there and see that happen to a horse?"' he said. "It's got to be very disappointing to anyone who loves horses."

https://www.paulickreport.com/news/ray-s-paddock/gorajec-barr-tonko-bill-only-path-toward-uniformity/

Gorajec: Barr-Tonko Bill Only Path Toward Uniformity

by Joe Gorajec | 10.15.2015 | 7:57am

The following commentary was written by Joe Gorajec, who on Saturday was relieved of his job as executive director of the Indiana Horse Racing Commission (IHRC), a position he held for nearly 25 years.

The commentary, which Gorajec intended for publication when written, was shared with Indiana commissioners in August with a clearly stated caveat that it represented his personal viewpoint, and not that of the IHRC or its individual members.

Gorajec was advised, in no uncertain terms, that he was not to have the article published.

Having been the executive director of the Indiana Horse Racing Commission since 1990, I have been on the frontline of regulatory policy and enforcement, both in Indiana and nationally, for the past 25 years.

During this time I have seen substantial efforts – with mixed results – to improve uniformity in drug testing and penalties for positive tests. I have also witnessed a largely inadequate, milquetoast response to emerging threats to racing's integrity. Most significant among these threats are blood-doping agents and other drugs that require an extensive out-of-competition program for detection.

I believe the threat to the integrity of our sport is greater now than it was 25 years ago.

We are in this predicament because we lack central governance for drug testing and penalties for violations, which, of course, all other major sports have.

We have been promised and have held out hope that uniformity was achievable and just around the corner. It is not achievable, nor is it around the corner, so you can stop waiting. Uniformity under our current regulatory structure is a mirage.

Lack of uniformity does not equate to lack of effort. In a nutshell, uniformity is *incompatible* with the current structure of individual state prerogatives. Try as we might, we cannot and will not get to our desired level of uniformity with our existing regulatory structure. Once we acknowledge this, it will be much easier to choose a new path leading to true national uniformity.

It should be noted that state regulators (i.e. Commissions) did not create the current model. They inherited it. This occurred decades ago when state legislatures bequeathed to racing commissions the authority and responsibility for equine drug testing.

This lack of uniformity has always been an issue, but emerging threats have made us much more susceptible to the designs of those who cheat. While these threats have increased, racing has become more globalized and the internet has rapidly spread all the shortcomings of our sport into consciousness of our dwindling fan base – and potential new fans.

I was born in Chicopee, Mass., in 1958. In that year, the four major Thoroughbred New England race tracks – Rockingham Park, Suffolk Downs, Narragansett Park and Lincoln Downs – drew 2,680,412 fans to the track. By comparison, that same year, the combined attendance for the Boston Red Sox, Bruins and Celtics was 2,168,412.

All these tracks are nothing but memories, except for Suffolk, which is scheduled to race three days this year. The reasons for the decline of Thoroughbred racing are many. I believe most people will agree that the foundation upon which we must build our sport moving forward is integrity. Our current structure of state prerogatives as it relates to drug testing and penalties has failed to provide this foundation.

For these reasons, I support the Barr-Tonko bill. It places the United States Anti Doping Agency (USADA) in a position to do virtually overnight what the racing industry has been incapable of doing over decades – mandate uniformity in drug testing, procedures and penalties. (Aug. 24, 2015)

Joe Gorajec served as executive director of the Indiana Horse Racing Commission and past chairman of the Association of Racing Commissioners International (RCI). The opinions herein are solely the opinions of the author and do not represent the opinions of the Indiana Horse Racing Commission.

This entry was posted in Ray's Paddock and tagged barr-tonko act, barr/tonko bill, drugs in racing, Horse Racing, horse racing regulations, Indiana Horse Racing Commission, Joe

Gorajec, national uniform medication program, out of competition testing by Joe Gorajec. Bookmark the permalink.

https://thehorse.com/158788/grant-joins-hsus-horse-racing-advisory-council/

Grant Joins HSUS Horse Racing Advisory Council

Barrie Grant, DVM, Dipl. ACVS, has been an equine veterinarian for more than 50 years and has been an official California Horse Racing Board veterinarian since 2009.

By Edited Press Release | Jun 17, 2018 | Welfare and Industry
Favorite

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The Humane Society of the United States (HSUS) has announced that Barrie Grant, DVM, Dipl. ACVS, has joined its National Horse Racing Advisory Council.

The council is composed of industry professionals and specialists who actively promote higher animal welfare standards within the scope of their involvement in horse racing.

Since its formation in June of 2016, the council has been directing most if its time and resources in advancing federal medication reform legislation that would put an independent, non-financially-conflicted third-party player in charge of setting the rules regarding the medicating of horses. The HSUS said Grant's extensive experience as a veterinarian with racetrack experience will assist the council in making decisions to best protect the welfare of equine athletes.

Grant has been an equine veterinarian for more than 50 years and has been an official California Horse Racing Board veterinarian since 2009. He is board certified in surgery, was a surgeon and partner at San Luis Rey Equine Hospital, and is the author or co-author on more than 100 review papers mainly on surgical procedures and exercise physiology. He was part of the team that developed the surgical technique for treating cervical spinal cord compression. Grant is a lifelong member of both the American Association of Equine Practitioners and the American Veterinary Medical Association.

“I am hopeful that my lifetime of experience with the sport of horseracing and the diagnosis, treatment, management and pathogenesis of the medical conditions of athletic horses will enrich the perspective of the Humane Society of the United States National Horseracing Advisory Council,” said Grant. “It is humbling to be included with the other board members who all bring their special talents, time, and dedication to making racing a sport we all can take pride in.”

Council chairman Joe DeFrancis added, “We are delighted to welcome Dr. Grant to the … National Horseracing Advisory Council. The primary purpose of the council is to facilitate the flow of information and knowledge between the horse racing industry and HSUS, so that the industry and HSUS can work together with maximum effectiveness to solve problems and address important issues concerning the welfare of the equine and human athletes who are the cornerstone of the sport and business of horse racing. Our goal is to have the council comprised of knowledgeable and experienced individuals who represent every aspect and segment of horse racing.

“The knowledge and expertise that a veterinarian of Dr. Grant’s experience and stature brings to the council will be invaluable, and I know I speak for each member of the council when I say how excited we are to have the benefit of his participation.”

https://www.paulickreport.com/news/ray-s-paddock/handel-need-end-slaughter-american-equines/

Handel: We Need To End Slaughter Of American Equines

by Hal Handel | 09.22.2017 | 3:50pm

Throughout history, horses have worked alongside us as companions and partners in work, war and sport; they are living symbols of our nation's spirit.

We have the honor and duty to protect them, which is why I am imploring my colleagues in the horse racing industry to contact their federal representatives in Congress and urge them to cosponsor the Safeguard American Food Exports (SAFE) Act (H.R. 113/S.1706) and maintain the ban on funding horse slaughter in the United States.

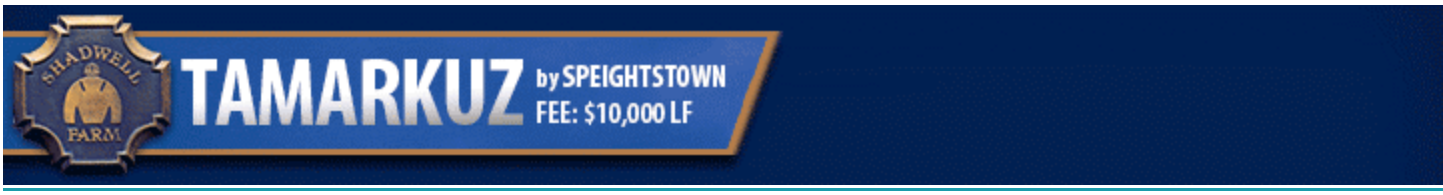
This key federal bill will prevent horse slaughter plants from opening in the U.S. and end the export of horses abroad for slaughter.

Our equine athletes are the lifeblood of our industry, yet too often they are condemned to a horrible death in a slaughter plant. The disreputable, predatory slaughter industry gathers up our loyal and trusting companions only to turn them into meat exports for profit.

Individuals who send horses to slaughter have nothing to do with responsible animal ownership or proper care, nor do they have an ounce of compassion for the graceful and sentient athletes they treat with such brutality. Horses unfortunate enough to end up in the hands of kill buyers suffer terribly at auctions, during transport and during the grisly slaughter process itself.

Horses are no better off being slaughtered in the U.S. than they are abroad. Before the last domestic plant closed in 2007, the United States Department of Agriculture (USDA) documented rampant cruelty at U.S. slaughter plants. There is no reason to believe that bringing slaughter back to the U.S. will make the process humane.

Quite simply, it is a brutal end for animals we have trained to trust us. Nothing about the way that slaughterhouses operate can be made humane for horses. These are lowbrow businesses concerned solely with serving foreign markets and turning a profit, a bloody one at that.



For the last 10 years, Congress has prevented wasteful spending and protected horses by including language in the Agricultural Appropriations Bill prohibiting federal funding for inspection of horse slaughter facilities.

In July, the House Appropriations Committee voted to spend our tax dollars on USDA inspections, which would pave the way for slaughter plants reopening in the U.S., while the Senate Appropriations Committee did not. Efforts to include an amendment in the House Appropriations bill to end this wasteful spending were stymied by pro slaughter politicians who have no interest in horse welfare. The very real possibility that these plants could once again set up shop in the U.S., using our tax dollars to fund USDA inspections, is looming.

Congress will need to reconcile this issue, and as horsemen, we need to take action now to protect our beloved horses by urging our federal representatives to vote to cosponsor the SAFE Act and maintain the current ban on horse slaughter in the U.S.

With the recent announcement by the Trump administration that the 2018 budget will include a 21% cut to the USDA budget, why would Americans want their tax dollars spent on supporting a predatory industry and inspecting meat that ends up on foreign dinner plates?

Horse slaughter also raises serious food safety concerns because drugs administered to horses make their meat unfit for human consumption. Those who work with horses on a daily basis need only glance at the labels on the products in their tack boxes. The overwhelming majority of those products come with a warning: *Not for use in food producing animals.*

There are several other good reasons to reject the return of horse slaughter.

Communities that hosted slaughter plants were stifled economically from the negative stigma of horse slaughter plants and real estate values plummeted.

Additionally, these neighborhoods were burdened with polluted water that often overwhelmed the small towns' septic systems, resulting in constant and horrible stenches.

Furthermore, the transportation of horses to these slaughter facilities was often just as inhumane as the treatment that awaited them. We shouldn't spend millions of American taxpayer dollars just to enable a cruel practice so that a greedy few can peddle tainted horse meat to the public. In the past, this has occurred at the expense of the taxpayer.

The slaughterhouse is not the least expensive way of ending the life of a horse, but it is the greediest and most inhumane way.

It is time for my fellow horse racing enthusiasts to take action and safeguard our horses. I believe that it is our duty to be the voice of our horses and stand up to the abusive horse slaughter industry.

I would strongly encourage you to contact your hometown legislators in both the House of Representatives and the Senate. Let them know that as a member of our country's horse racing industry, you reject horse slaughter. Urge them to take the necessary steps to prevent it from returning to the U.S.

Click here to contact your U.S. Senator and House of Representative member.

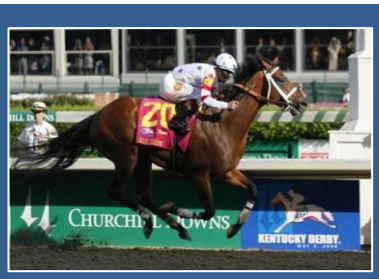
Hal Handel is a former deputy attorney general in New Jersey who supervised the state grand jury investigation into the Tony Ciulla race fixing scandal in the 1970s. He later served as executive director of the New Jersey Racing Commission and served in executive capacities at Monmouth Park, Meadowlands, Philadelphia Park, and the New York Racing Association. He is a past president of the Thoroughbred Racing Associations of North America and a past chairman of the Thoroughbred Racing Protective Bureau.

This entry was posted in NL Article, Ray's Paddock and tagged congress, Hal Handel, Horse Slaughter, house appropriations committee, SAFE Act, Safeguard American Food Exports Act, senate appropriations committee, United States Department of Agriculture, USDA by Hal Handel. Bookmark the permalink.

factsheet

Support the Horseracing Integrity Act of 2017

In the 115th Congress, H.R. 2651 was introduced by Reps. Andy Barr (R-KY) and Paul Tonko (D-NY).



"There has been expressed concern, primarily among examining veterinarians and those who observe the industry, about whether the current medication practices are in the best interest of the horse."

-Dr. Rick Arthur, Equine Medical Director of the California Horse Racing Board

The Problem

Despite its national and international scope, modern horse racing is still being conducted under outdated state-by-state drug and medication rules. This causes risk to the horses running races; confusion for owners and trainers whose horses race across state lines; and inconsistency for bettors who want to be able to fairly evaluate horses. It's clear that when it comes to medication, the horse racing industry can't both promote and police the sport. It needs a national, independent, non-governmental organization like the U.S. Anti-Doping Agency to create and maintain a system that protects horses, horsemen and fans.

The Facts

- Horseracing is a multibillion dollar industry, generating an estimated $40 billion annually and 400,000 jobs.
- The U.S. leads the world in the rate of fatal racing injuries at 1.89 per 1,000 starts (measured over approximately 310,000 starts).
- There is an overuse of therapeutic medication that masks pain and enables an injured horse to race when rest and time off would be more appropriate.
- With a lack of out-of-competition testing, veterinarians and other racing officials have expressed concerns that pre-race exams at racetracks are compromised by the use of drugs that can disguise the unsoundness of a horse.
- On-track betting and interstate, off-track wagering are the financial engines of the horse racing industry.
- Many veterinarians, geneticists, regulatory officials and racing fans believe that America's practice of medicating horses is harmful to the Thoroughbred breed.
- Congress considered banning drugs in horseracing in 1980, but instead allowed each state to make its own decisions on drugs and horseracing. This has resulted in a patchwork of state laws that encourage trainers caught doping their horses to move from state to state and continue doping and racing their horses.

HUMANE SOCIETY
LEGISLATIVE FUND™
1255 23rd Street, NW, Suite 455 Washington, DC 20037
hslf.org

The Solution

The Horseracing Integrity Act of 2017will ensure equine welfare, protect the integrity of the sport and promote a sustainable and viable horse racing industry in the United States by granting independent control over rule-making, testing and enforcement oversight regarding drugs and medication to a new Authority created by the U.S. Anti-Doping Agency.

USADA – the same agency recognized by Congress as the official anti-doping agency for the Olympic, Pan American and Paralympic sports in the United States – is a national, independent, non-governmental organization with a proven track record of creating uniform standards and science-based oversight to protect the rights of clean competitors and the integrity of competition.

The new Authority, with limited oversight under the Federal Trade Commission (FTC), would be comprised of representatives of USADA and members of the horseracing industry, and would be responsible for:

- developing, publishing, and maintaining rules regarding substances, methods, and treatments that may or may not be administered to Thoroughbred race horses;
- implementing programming related to anti-doping education, research, testing, and adjudication to prevent the racing of horses who have been so affected; and
- establishing uniform rules imposing sanctions, up to and including a lifetime ban from horseracing, for those who violate the rules.

The Act would require that horse racing associations and off-track betting operators recognize the jurisdiction and authority of the independent Authority as a condition of accepting, receiving or transmitting interstate wagers on horse races.

Support for the Horse Racing Integrity Act

This legislation has been endorsed by The Jockey Club, the Breeders' Cup Ltd., the Water, Hay, Oats Alliance (WHOA), The Humane Society of the United States, the Kentucky Thoroughbred Association, Kentucky Thoroughbred Owners & Breeders, the Consignors and Commercial Breeders Association, Meadowlands Racetrack, Tioga Downs, Vernon Downs, Arapahoe Park, The Stronach Group (parent company to the Maryland Jockey Club, The Preakness Stakes, Santa Anita Park, Gulfstream Park, Portland Downs, and Golden Gate Fields) the Humane Society Veterinary Medical Association, and many horse owners, track owners, and trainers.

For more information please contact Marty Irby at mirby@hslf.org

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Myths and Facts Regarding the Horseracing Integrity Act

Myth: Adequate rules and enforcement already exist to prevent doping in horseracing.

Fact: There are no uniform rules to prohibit performance-enhancing drugs and penalize doping violations in horseracing. Almost all American race horses are injected with race day medication, a practice banned by almost all other countries. Trainers can violate medication rules multiple times, seemingly with impunity.

Myth: This bill would create a new federal bureaucracy to regulate horseracing.

Fact: The Act places responsibility for the creation and enforcement of new nationwide rules with an independent, non-governmental oversight authority that may appoint state racing commissions to assist with enforcement, with limited oversight under the FTC. Funding for the anti-doping program mandated by the bill would come from industry, not the taxpayer.

Myth: The federal government has no place in horseracing.

Fact: Federal law already regulates interstate or "simulcast" racing for Thoroughbred, Standardbred (harness), and Quarter horses. This bill would establish a national anti-doping program, managed by an independent, non-governmental authority and would ensure a level playing field wherever interstate wagering on horse races is offered.

Myth: Horseracing groups can solve doping problems without federal legislation.

Fact: Industry groups and state commissions have promised reform for decades. However, since horseracing lacks a national league or commissioner to set and enforce rules, federal legislation that establishes an independent national oversight body charged with developing and enforcing uniform rules is the only viable way to ensure safety and integrity.

Myth: The bill could eliminate the use of beneficial drugs and veterinary care for race horses.

Fact: Nothing in the Horseracing Integrity Act of 2017 prohibits a racehorse from receiving therapeutic care or drugs. Horses should not race when needing such therapy - as doing so can lead to breakdowns, and puts at risk their safety and that of their riders.

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http://www.thoroughbreddailynews.com/julie-krone-joins-humane-societys-horse-racing-council/

Julie Krone Joins Humane Society's Horse Racing Council

Tuesday, April 25, 2017 at 2:18 pm | Back to: Shared News

Previous Story | **Next Story**

Julie Krone | *Adam Coglianese*

Pioneering Hall of Fame jockey Julie Krone has joined the Humane Society of the United States' National Horse Racing Advisory Council. Krone is the first female jockey to win a Triple Crown race and is also the first woman inducted into the National Museum of Racing and Hall of Fame.

Since its formation in June 2016, the Council has been directing most if its time and resources in advancing federal anti-doping legislation that would put an independent, non-financially-conflicted third-party player in charge of setting the rules regarding the same-day medicating of horses.

"I am honored to be selected to participate in The HSUS' Horse Racing Advisory Council and look forward to working with the council members on legislation that will help protect both the equine and human athletes involved in this thrilling sport," said Krone. "As the first woman inducted into the National Museum of Racing and Hall of Fame, I hope my years of experience as a jockey will allow me to help drive changes needed in the industry that will better protect its athletes and allow the industry to prosper."

http://www.drf.com/news/krone-appointed-humane-society-council-racing

Krone appointed to Humane Society council on racing

By Matt Hegarty

Barbara D. Livingston

Julie Krone was inducted to the National Museum of Racing and Hall of Fame in 2000.

Julie Krone, the first woman to be inducted into racing's Hall of Fame, has been appointed to a council at the Humane Society of the United States focusing on racing, the Humane Society announced Tuesday.

The announcement of the appointment came on the same day that Frank Stronach, the owner-breeder who owns the racing company The Stronach Group, announced his support for federal legislation that the Humane Society and several influential racing and breeding organizations have endorsed. The federal legislation would appoint a private, non-profit company, the U.S. Anti-Doping Agency, as the overseer of the sport's medication and drug-enforcement policies.

At the HSUS, Krone will join another Hall of Fame rider, Chris McCarron, along with several racing officials, including Joe De Francis, the former owner of the Maryland Jockey Club; Jim Gagliano, president and chief operating officer of The Jockey Club; Allen Gutterman, a former racing executive; Joe Gorajec, former executive director of the Indiana Horse Racing Commission; and Staci Hancock, a founding member of a group opposed to the use of raceday medication.

The Humane Society racing council was formed last year, in large part to lobby for the passage of the federal legislation. In the release announcing Krone's appointment, the Humane Society said the council "has been directing most of its time and resources in advancing federal anti-doping legislation," in reference to the bill introduced last year.

“I hope my years of experience as a jockey will allow me to help drive changes needed in the industry that will better protect its athletes and allow the industry to prosper,” Krone said in a statement. Krone is married to Daily Racing Form executive columnist Jay Hovdey.

While several racing organizations have endorsed the bill, the legislation is controversial among a number of other racing constituencies. In particular, horsemen have said that the legislation is designed to put in place a ban on the raceday use of the diuretic furosemide, which is legal to administer in the United States and Canada on raceday to mitigate bleeding in the lungs. Horsemen contend the use of the drug is both humane and effective, while opponents maintain that raceday use of any drug places a stain on the sport.

In a statement released Wednesday in support of the legislation, Stronach specifically referenced his opposition to raceday medication (Stronach’s horses in the United States and Canada race on furosemide).

“No raceday medication is a giant step forward,” Stronach said in the statement. “I believe, in the long run, no raceday medication is better for the horses and for the industry.”

The legislation is not expected to gain traction in the next several years, in large part due to the lack of unanimity among racing’s constituencies, racing lobbyists have said.

https://www.nytimes.com/2012/03/25/us/death-and-disarray-at-americas-racetracks.html?_r=1&pagewanted=all

Mangled Horses, Maimed Jockeys

WALT BOGDANICH, JOE DRAPE, DARA L. MILES and GRIFFIN PALMERMARCH 24, 2012

RUIDOSO, N.M. — At 2:11 p.m., as two ambulances waited with motors running, 10 horses burst from the starting gate at Ruidoso Downs Race Track 6,900 feet up in New Mexico's Sacramento Mountains.

Nineteen seconds later, under a brilliant blue sky, a national champion jockey named Jacky Martin lay sprawled in the furrowed dirt just past the finish line, paralyzed, his neck broken in three places. On the ground next to him, his frightened horse, leg broken and chest heaving, was minutes away from being euthanized on the track.

For finishing fourth on this early September day last year, Jacky Martin got about $60 and possibly a lifetime tethered to a respirator.

The next day, it nearly happened again. At virtually the same spot, another horse broke a front leg, pitching his rider headfirst into the ground. The jockey escaped serious injury, but not the 2-year-old horse, Teller All Gone. He was euthanized, and then dumped near an old toilet in a junkyard a short walk from where he had been sold at auction the previous year.

In the next 24 hours, two fearful jockeys refused their assigned mounts. The track honored two other riders who had died racing. As doctors fought to save Mr. Martin's life, a sign went up next to the track tote board: "Hang in there, Jacky. We love you."

On average, 24 horses die each week at racetracks across America. Many are inexpensive horses racing with little regulatory protection in pursuit of bigger and bigger prizes. These deaths often go unexamined, the bodies shipped to rendering plants and landfills rather than to pathologists who might have discovered why the horses broke down.

In 2008, after a Kentucky Derby horse, Eight Belles, broke two ankles on national television and was euthanized, Congress extracted promises from the racing industry to make its sport safer. While safety measures like bans on anabolic steroids have been enacted, assessing their impact has been difficult because many tracks do not keep accurate accident figures or will not release them.

But an investigation by The New York Times has found that industry practices continue to put animal and rider at risk. A computer analysis of data from more than 150,000 races, along with injury reports, drug test results and interviews, shows an industry still mired in a culture of drugs and lax regulation and a fatal breakdown rate that remains far worse than in most of the world.

If anything, the new economics of racing are making an always-dangerous game even more so. Faced with a steep loss of customers, racetracks have increasingly added casino gambling to their operations, resulting in higher purses but also providing an incentive for trainers to race unfit horses. At Aqueduct Racetrack in Queens, the number of dead and injured horses has risen sharply since a casino opened there late last year.

Mr. Martin's injury occurred in a state with the worst safety record for racetracks, a place where most trainers who illegally pump sore horses full of painkillers to mask injury — and then race them — are neither fined nor suspended and owners of those drugged horses usually keep their winnings.

The failure of regulators to stop that cheating is reflected in the numbers. Since 2009, records show, trainers at United States tracks have been caught illegally drugging horses 3,800 times, a figure that vastly understates the problem because only a small percentage of horses are actually tested.

In the same period, according to the Times analysis, 6,600 horses broke down or showed signs of injury. Since 2009, the incident rate has not only failed to go down, it has risen slightly.

The greatest number of incidents on a single day — 23 — occurred last year on the most celebrated day of racing in America, the running of the Kentucky Derby. One Derby horse fractured a leg, as did a horse in the previous race at Churchill Downs. All told, seven jockeys at other tracks were thrown to the ground after their horses broke down.

A state-by-state survey by The Times shows that about 3,600 horses died racing or training at state-regulated tracks over the last three years.

In one 13-day stretch of racing in 2010 at Sunland Park Racetrack and Casino in New Mexico, nine horses died racing, five were hauled away in ambulances and two jockeys were hospitalized, one in critical condition.

"It's hard to justify how many horses we go through," said Dr. Rick Arthur, the equine medical director for the California Racing Board. "In humans you never see someone snap their leg off running in the Olympics. But you see it in horse racing."

Even some of America's most prestigious tracks, including Belmont Park, Santa Anita Park and Saratoga Race Course, had incident rates higher than the national average last year, records show.

Why racehorses break down at such a high rate has been debated for years, but the discussion inevitably comes back to drugs.

Laboratories cannot yet detect the newest performance-enhancing drugs, while trainers experiment with anything that might give them an edge, including chemicals that bulk up pigs and cattle before slaughter, cobra venom, Viagra, blood doping agents, stimulants and cancerdrugs.

Illegal doping, racing officials say, often occurs on private farms before horses are shipped to the track. Few states can legally test horses there.

"They are pharmacist shops," said Dr. George Maylin, the longtime head of New York State's testing laboratory. "Nobody has any control over what they are doing."

Even so, legal therapeutic drugs — pain medicine in particular — pose the greatest risk to horse and rider. In England, where breakdown rates are half of what they are in the United States, horses may not race on any drugs.

At higher levels, pain medicine can mask injury, rendering prerace examinations less effective. If a horse cannot feel an existing injury, it may run harder than it otherwise would, putting extra stress on the injury. As many as 90 percent of horses that break down had pre-existing injuries, California researchers have found.

“This is just a recipe for disaster,” said Dr. Tom David, who until this year was chief veterinarian for the Louisiana Racing Commission. “Inflamed joints, muscles and mild lameness are masked by medication and therefore undetectable to the examining veterinarian.”

While high-profile Triple Crown races get the most attention, the mainstay of racing in America is the lower tier, so-called claiming races. Horses in these races are most vulnerable, in part because regulators often give them less protection from potentially dangerous drugs.

The Times analysis found that horses in claiming races have a 22 percent greater chance of breaking down or showing signs of injury than horses in higher grade races. That lower level of race has been particularly affected by the arrival of casinos.

At Aqueduct, most of the 16 horses that have died so far this year were in the lower ranks, where purses have increased the fastest because of new casino money.

“It’s hard to watch these poor animals running for their lives for people who could really care less if they live,” said Dr. Margaret Ohlinger, a track veterinarian at Finger Lakes Casino and Racetrack in upstate New York. She performs pre-race inspections and treats horses injured in races but is not responsible for their overall care.

Last year at the track, Dr. Ohlinger counted 63 dead horses. That, she said, is more than double the fatalities of five years earlier.

Oversight Undermined

Race officials have always done their best to hide fatal breakdowns, erecting screens around fallen horses and then refusing to disclose the tracks’ accident rates.

But amid criticism that individual state racing commissions lacked the will to make the sport safer, and the threat of federal oversight, the industry promised changes, including new restrictions on the use of drugs, a program to accredit racetracks and drug-testing laboratories and uniform rules for punishing drug violators.

The industry also set up a national database where tracks were asked, but not required, to report injuries with the promise of confidentiality.

So far, the response to these reform measures has fallen short.

Fifty-five tracks pledged that they would seek accreditation, requiring among other things prerace inspections and postmortem examinations, or necropsies. Fewer than half have kept their promise.

“Some tracks do not have the money to spend to meet our standards; others think it’s window dressing and why bother,” said Michael Ziegler, executive director of the National Thoroughbred Racing Association Safety and Integrity Alliance. “Any follow-up with tracks has gone unanswered.”

The laboratory accreditation program, introduced in July 2009, has fared even worse. After calling the program an “unprecedented” step that “ultimately will change the face of drug testing in this country,” a consortium of industry groups that manages it says not a single lab has been accredited.

An association of racing regulators wrote to Congress on May 14, 2010, boasting that with the exception of anti-bleeding medicine, “race day medications are not allowed.” Yet records show that in Florida, a major racing state, trainers continue to use corticosteroids, an anti-inflammatory, on race day.

The national repository for injury reports, maintained by the Jockey Club, the most powerful racing industry group, has been more successful, gathering data from 92 percent of the racing days.

“We put it into a database, and we provide tools back to the racetracks where they can analyze and slice and dice the information themselves,” said James L. Gagliano, president of the Jockey Club, who says the group has encouraged racetracks to make the statistics public. So far, 24 out of 86 tracks have done so.

To assess how often horses get injured, The Times bought data for about 150,000 races from 2009 through 2011, then searched for terms indicating that a horse encountered a physical problem, like “broke down,” “lame” or “vanned off.”

Although the people who chronicle the races, known as chart callers, can be stylistically different, they are taught to use standard industry terms, and their descriptions constitute the official record used by gamblers to evaluate horses.

The analysis showed that during those three years the rate of incidents for horses in the United States was 5.2 per 1,000 starts.

By contrast, Woodbine Racetrack in Toronto, which year after year has one of the lowest breakdown rates in North America, had an incident rate of only 1.4, according to the Times analysis. “One of the differences here is medication is not as permissive as it is in the U.S.,” said Jamie Martin, executive vice president of racing at Woodbine.

According to the analysis, five of the six tracks with the highest incident rates last year were in New Mexico. All are casino tracks, commonly called “racinos.” Ruidoso, where Jacky Martin was injured, topped the list in 2011 with 14.1 incidents per 1,000 starts. Ruidoso attributes its incident rate in part to the failure of horses to acclimate quickly to the track’s elevation. Some horses that appeared to be injured, track officials said, may have simply needed time “to catch their breath.”

Yet no accident over the last three years can match what occurred in a single race on Feb. 29, at Hollywood Casino at Charles Town Races in West Virginia. Eight horses started. Seven fell. One finished. Along the way, seven jockeys were left scattered on the ground.

The next and final race was canceled, not just because it took so long to clear the track, but also because too few jockeys were available or willing to ride.

Drug Violations

It was the day’s first race in Hobbs, N.M. The track was fast and the weather clear. Shortly after noon on Oct. 16, 2010, nine young horses were loaded into the starting gate at Zia Park Casino.

With the finish line a mere 400 yards away, this would be an all-out sprint, horse racing’s equivalent of a drag race. While these races, run by a breed called quarter horses, lack the ebb-and-flow suspense of a longer thoroughbred race, they make up for it in a pure adrenalin rush. The best quarter horses can hit nearly 50 miles an hour.

Three weeks earlier at Zia Park, Mark Anthony Villa was on the back of a quarter horse when it fell just past the finish line, throwing him to the ground. With a herd of thousand-plus-pound animals bearing down on him, Mr. Villa tried to crawl to safety.

He never made it. A horse’s hoof struck him in the head with such force that his helmet shot like a bullet across the track. He died within an instant, leaving a wife and twin children.

For years, track veterans could only speculate as to whether racing quarter horses was more dangerous than racing thoroughbreds. In fact, the Times analysis shows that quarter horses have a nearly 29 percent greater chance of breaking down or showing signs of injury.

With Mr. Villa's death still on the minds of riders and spectators, a gray 2-year-old colt named I Glance at Chicks settled in the 6 hole waiting for the starting bell. For bettors, he was an animal to watch. The horse had won his only race and was trained by Andres Gonzalez, who, according to racing commission records, was not above allowing his horses to race with extra help. Illegal help.

A week earlier, another horse trained by Mr. Gonzalez had raced at Zia Park with 12 times the legal limit of a drug that mimicked steroids. By the end of 2011, Mr. Gonzalez would have amassed a dozen drug violations in just four years. His uncle, Ramon O. Gonzalez Sr., for whom he often worked, had his own lengthy list of violations, including accusations that he drugged 10 horses in just two months.

Whether I Glance at Chicks felt pain as he raced is unknown, but he never challenged for the lead. Shortly after crossing the finish line in fifth place, he broke down. The diagnosis: a bone fracture in his front left leg and ligament damage, injuries from which he could not recover.

A veterinarian, Dr. Clayton McCook, euthanized the colt with an injection of pentobarbital. Afterward, Dr. McCook wrote a note "to whom it may concern," expressing his distress to the authorities over this fatal breakdown and others like it.

"I have had to euthanize several horses due to catastrophic injuries and feel they are occurring in greater numbers than one should expect," Dr. McCook wrote. "I do not pretend to be an expert in racing surfaces, nor in the training of racehorses, but I do know that something appears to be amiss at Zia Park."

According to an analysis of race records, Zia Park in 2010 had the nation's second-highest incident rate, 13.3. Last year, it ranked fourth with a rate of 11.9. After horse owners complained about the track surface, Zia Park officials said they spent $80,000 resurfacing it before the 2011 racing season.

During the three days that a Times reporter visited Zia Park last November, eight horses collapsed, died or were transported off the track. At the time, track officials said it was company policy not to allow a reporter access to the backside where trainers stable their horses.

Christopher McErlean, vice president of racing at Penn National Gaming, which owns Zia Park, said in a statement that the Times analysis used figures "produced by nonmedical professionals for the purpose of handicapping feature races."

Mr. McErlean also said some horses are vanned off as a precaution and may not actually have been injured.

But Zia Park officials said that last year, "a significant number" of horses had to be carried off the track because of exhaustion stemming from the possible abuse of a drug that mimics anabolic steroids as well as "other medication issues."

Mr. McErlean said Penn Gaming endorses tougher penalties for those who violate drug rules.

Without a postmortem exam of I Glance at Chicks, no determination could be made as to whether a pre-existing condition or some other unknown factor might have played a role in his demise. But tests did reveal that the horse had been dosed with a large load of a powerful painkilling medicine called Flunixin.

In at least two states, 2-year-olds may not race with any Flunixin. Not so in New Mexico, where they can run with up to 50 nanograms of the drug, more than double the amount allowed in a higher class of competition called graded stakes races.

But even that higher amount was not enough for Mr. Gonzalez. I Glance at Chicks carried 282 nanograms of Flunixin.

To put that figure in perspective, Dr. Mary Scollay, chief veterinarian for the Kentucky Racing Commission, said she had never seen such high levels in her state.

“When you look at the history of our medication violations — Flunixin — most are under 50 nanograms, 35 nanograms, something like that,” Dr. Scollay said. In fact, she said she had never seen a violation in Kentucky over 104.

In New Mexico, it is common practice.

Tests on horses in New Mexico showed results over 104 nanograms on 68 occasions since 2009, with some registering 1,000 and even 2,400, records show. The levels are so high that regulatory veterinarians in other states say the horses must have been drugged on race day, a practice that is forbidden.

Before the New Mexico Racing Commission could pass judgment on the overdosing of I Glance at Chicks, another horse trained by Mr. Gonzalez tested positive for even higher levels of Flunixin. The extra dosing did not hurt performance. The horse finished first, and its owner, Mr. Gonzalez’s cousin Ramon Gonzalez Jr., got to keep his winnings.

If Andres Gonzalez was worried about how the racing commission viewed his treatment of I Glance at Chicks, he need not have been. Records show he received a warning and nothing more.

Lax Penalties

New Mexico’s racing industry — the tracks and their regulators — has been unusually slow in responding to the safety alarms.

Four of the state’s five racetracks, including Zia Park and Ruidoso, are unaccredited, and the track where Mr. Martin’s injury occurred does not report accidents or positive drug tests to groups that monitor such events.

New Mexico also recorded no positive tests in 2010 and 2011 for the most frequently abused pain medicine in racing, phenylbutazone, a nonsteroidal anti-inflammatory commonly known as “bute.” After The Times asked why none had been found, the new executive director of the state’s racing commission, Vince Mares, said that after researching the question, he discovered that the previous leadership “had cut back on the tests” for financial reasons.

Without a national law regulating drugs in racing, New Mexico regulators can be as lenient as they wish in disciplining drug violators.

Trainers in New Mexico who overmedicate horses with Flunixin get a free pass on their first violation, a $200 fine on the second and a $400 fine on the third, records show.

In Indiana, by contrast, winnings are forfeited after the first drug offense. “If someone who violates the rule thinks the penalties are going to be mild or nonexistent, then breaking the rules is just a cost of doing business,” said Joe Gorajec, the executive director of the Indiana Horse Racing Commission.

New Mexico gives offenders another break: it wipes away Flunixin violations every 12 months, allowing trainers to again overmedicate horses without penalty. Dozens of huge Flunixin overdoses have resulted in warnings only.

Sometimes the same horse is illegally drugged twice. On May 9, 2009, Runawayslew, a horse trained by Andres Gonzalez, raced with two anti-inflammatory drugs. Nineteen days later, under another trainer, Runawayslew raced on cocaine.

To varying degrees, the picture is similar nationwide. Trainers often face little punishment for drug violations, and on the rare occasions when they are suspended, they are allowed to turn their stables over to an assistant. Since January 2005, 116 trainers have had five or more drug violations, and 10 trainers had 10 or more, records show.

In New Mexico, Cody Kelley, an Albuquerque lawyer who represents people accused of violating racing commission rules, including Andres Gonzalez, said punishments were too arbitrary.

“Are there people that cheat at horse racing in New Mexico? Yes, happens everywhere,” Mr. Kelley said. “But I think our commission right now is not equipped to deal with it. What we need are national rules.”

Mr. Mares, the New Mexico racing chief, agrees that his agency needs more uniform penalties to avoid charges of favoritism. “There is an issue of consistency — you can quote me on this,” Mr. Mares said. “It is being addressed.”

New Mexico recently became the first state to temporarily ban all horses from racing on clenbuterol, a drug that aids respiration, but that has been widely abused because it can build muscle.

In recent years, the state commission has had its embarrassments.

One former investigator faces trial on charges of stealing horses while working at the commission. Another trainer’s doping violation was dismissed because the assistant attorney general handling the case neglected to show up in court. And the commission had to drop charges against Ramon O. Gonzalez Sr. for drugging 10 horses because it forgot to file the proper paperwork, according to the state attorney general’s office.

Nonetheless, odds are slim that any of the Gonzalezes — Andres, Ramon Sr. or Ramon Jr. — will show up at a New Mexico racetrack any time soon. In late January, a federal grand jury in Albuquerque indicted them on charges of participating in a drug trafficking scheme tied to one of Mexico’s most notorious drug cartels. All have pleaded not guilty.

Andres Gonzalez was arrested at Sunland Park Racetrack and Casino in New Mexico. His uncle, Ramon Sr., was arrested while pulling a horse trailer that the authorities said was carrying 26 kilograms of cocaine and 500 pounds of marijuana.

Masking Pain, or Healing It

Breakdowns can be caused by a variety of factors, including poor track surface and jockey mistakes. But drugs, often used to mask existing injuries, are the prime suspect.

“It’s not that these medications caused the injuries, but the trainers knew the horses were injured and gave them the meds to get them into the race,” said Dr. Arthur, the veterinarian for the California Horse Racing Board.

Necropsies are considered essential to determining if an existing injury contributed to a fatal breakdown. However, only 11 states require them, a Times survey found.

In California, where necropsies are required, researchers found that a “large majority” of horses had existing problems at the site of their fatal injuries.

“To be fair, some of that is microscopic and may not be readily apparent,” Dr. Arthur said. “We’re trying to figure out why vets and trainers are not identifying injuries prior to catastrophic injuries.”

But many prior ailments are indeed serious. The Times obtained hundreds of necropsy reports on racehorses that died racing in Pennsylvania and found problems that included “severe degenerative joint disease,” “severe chronic osteoarthritis” and pneumonia with “severe, extensive” lung inflammation. One horse had 50 stomach ulcers. Another had just one eye. Pathologists also found metal screws in two horses that had broken bones from previous accidents.

In the United States, horses are usually allowed to run on some dose of pain medication, usually bute. The question, fiercely debated in the racing community, is at what level do therapeutic drugs make racing unsafe?

Virginia’s fatality rate went up after regulators in 2005 raised the allowable level of bute to 5 micrograms from 2 micrograms. “Our catastrophic incidents increased significantly,” said Dr. Richard Harden, equine medical director for the state racing commission.

Virginia returned to the lower level in 2009, though the fatality rate has not come down.

Iowa’s fatality rate rose by more than 50 percent after the state in 2007 allowed a higher level of bute.

Regulatory veterinarians say the higher allowable levels make it difficult for them to spot lameness and injury during prerace examinations. In one study, researchers at Oklahoma State University said they found bute in most of the horses that died racing or training at Oklahomatracks in 2010. Six had both bute and Flunixin, a dangerous practice called “stacking,” the report said.

The researchers also expressed concern that despite fewer races, a record number of horses died, necessitating a “careful re-evaluation of track surfaces, medication/enforcement and prerace examinations.”

But prominent owners and trainers, and even some veterinarians, say evidence linking drugs and breakdowns is unconvincing.

Kent H. Stirling, chairman of the national medication committee for the Horsemen’s Benevolent and Protective Association, said there was “no scientific evidence whatsoever” that 5 micrograms of bute on race day is dangerous.

Mr. Stirling and others say sore horses should not be denied therapeutic medicine when needed. “If you’re a horseman and you’re trying to keep a horse going and keep him happy and healthy as you can, then these therapeutic medications are very helpful,” he said.

Regulators typically view prescription drug violations as more benign than the use of banned substances on horses. And they constitute the bulk of the 3,800 violations that The Times found by surveying racing states.

But others, including racing regulators overseas, say horses should not compete on any drug regardless of type.

“Therapeutic drugs, by definition, are used for healing and curing,” said Arthur B. Hancock III, whose farm produced three Kentucky Derby winners. “Drugs that mask pain and enhance performance are not ‘therapeutic.’ They are what they are: performance-enhancing drugs.”

The industry group that runs graded stakes races had promised to ban all therapeutic drugs for 2-year-olds, but in late February backed off, saying it did not have enough time to bring state regulators on board.

George W. Strawbridge Jr., a prominent breeder and owner, resigned from the group over that decision, calling it “one of the most craven acts” he had seen.

“How on earth did we get to this sorry state?” Mr. Strawbridge said. “The first reason is that in this country there are no significant consequences for doping horses.”

Respecting the Ride

Chris Zamora knows the sensation of riding a sore horse. But one ride in particular stands out.

On Nov. 25, 2008, Mr. Zamora was guiding his horse, Sinful Heart, into the first turn at Zia Park when he sensed something was wrong. “He didn’t want to take the turn,” he said. “He was in pain.”

Sinful Heart drifted out, clipped heels with another horse and fell. A trailing horse tripped over them.

Mr. Zamora, the winner of more than 1,000 races, nearly died in the accident, fracturing his skull, pelvis, ribs and four vertebrae. His lungs collapsed, his liver was lacerated and his heart was compressed. “They had to insert a needle to take the pressure off of my heart,” he said.

Sinful Heart survived to race three more times, in successively cheaper races, never winning before collapsing and dying on the track at Ruidoso.

Four months after his accident, in March 2009, Mr. Zamora returned to the track. But he had changed. No more cheap horses. “I tried to ride quality over quantity,” he said. “I didn’t ride a horse that somebody said was already sore. I scratched more of them at that time than I had in my whole life.”

The best trainers might have been unhappy, he said, but they trusted his judgment and fixed the problem. “They were great horsemen,” Mr. Zamora said, offering the ultimate compliment.

But not all were. Now, he said, some trainers just go to another rider. “These guys will head a horse up until it breaks down completely, and when there’s a man on top of them, it’s bad,” he said.

Other injured jockeys tell similar stories. “I think more should be done for the horse to let him heal naturally than to be getting him to the next race so we can get one more race out of him,” said Randy Meier, a winner of more than 4,000 races, many in the Chicago area.

Along the way, Mr. Meier broke his neck, collarbones, ribs, shoulder, legs, arms, wrist and sternum and developed a brain bleed.

New Mexico jockeys have been hit particularly hard. Not only was Mr. Villa killed and Mr. Zamora and Mr. Martin critically injured, Juan Campos died in an accident in August 2008; Jimmy Ray Coates fell the same year, his heart stopping twice after breaking his femur, shoulder and collarbone; Carlos Rivas had no pulse en route to the hospital after rupturing his aorta in 2010, and the same year Kelsi Purcell fractured multiple vertebrae in a spill.

There were other injuries as well.

“We’ve been through this so many times,” said Terry Meyocks, national manager of the Jockeys’ Guild. More than 50 permanently disabled jockeys receive assistance from the Jockeys’ Guild, he said.

After Mr. Zamora’s accident, Mr. Martin, a friend and hunting partner, had told him not to abandon hope. “You’ll be back,” he said. “You’re in great shape, it won’t be that long. You’re not done. You won’t be in a walker.”

Like all jockeys, Mr. Zamora knew the risks of riding. “Every time you do it, you take a chance one is going to break it off. Even with the soundest horse you take a chance.”

Good jockeys can alter their ride if a horse is sore or about to break down. In some cases, though, there are no hints, no warnings. And that is when jockeys face the greatest danger.

Jacky Martin had no warning.

“I thought he was going to die,” said Adrian A. Ramos, who was riding in the same race. “He hit the ground hard, real hard. I was behind him and I saw everything.”

A Second Chance

The question almost everyone at the track wanted to ask was why. Why did Mr. Martin, at the top of his game, the winner of a record seven All American Futurities, agree to ride a cheap claiming horse with no victories just three days before he was to ride the favorite in the $2.4 million Futurity?

The favorite did eventually win and would have paid him $120,000, the jockey’s standard share. For riding the horse that broke his neck, Mr. Martin took home little more than the cost of a tank of gas.

Until that wrenching moment in the Ruidoso dirt, Mr. Martin at age 56 had been on a redemptive journey to right the wrongs in his life, to help younger jockeys avoid the mistakes he had made and to regain what he had lost: an opportunity to sit atop a racehorse and to coax from it all the power it was willing to give, and nothing more.

For four years, Mr. Martin had been barred from racing after being sentenced to probation in 2006 for poaching deer and possessing less than a gram of methamphetamine. He and his wife, Tracey, also his agent, moved to Louisiana. “I worked horses every day for three and a half years being a gallop boy,” Mr. Martin said. “That’s all I was, a $10 gallop boy.”

In the afternoon, Mr. Martin helped to build fences and even a barn, his wife recounted. “We actually bagged horse manure and sold it and delivered it just to get through,” she said.

It was a steep fall for a man so highly revered in the sport that Mexican businessmen would send armed guards to escort him to high-stakes races south of the border.

“After a time, he took ownership for the wrong things that he did and worked his way through it,” Ms. Martin said. Just as important, friends say, he developed an even deeper appreciation for the role others played in racing, from grooms to horse owners struggling to stay in the game.

In the summer of 2010, Mr. Martin was finally cleared to race, and he returned to Ruidoso unsure of how he would be received. When word spread that “Jacky was back,” owners were eager to extend a helping hand, but most of all, they were eager to win.

And win he did. With the racing season half over, Mr. Martin stormed into the lead to become the top winner and champion jockey for 2010.

“He was so grateful he got a second chance,” Ms. Martin said in December. “He was on the radio saying: ‘People out there need to know that they can be forgiven and succeed. If I can fix my screwed up life, you can too.’ ”

In Mr. Martin’s quest to win an eighth Futurity in 2010, his horse lost by a nose in one of the biggest upsets in the history of that race. But the loss did not diminish the joy he felt competing again.

“It’s just a fairy tale for it to turn out the way it has,” Mr. Martin told a racing publication in 2010.

Mr. Martin fell a year later, on the Friday before Labor Day at the beginning of the final, biggest weekend of racing at Ruidoso. The tens of thousands of spectators, who would later fill the stands and line the distant highway with parked cars, had yet to arrive.

Only a small, quiet crowd, including relatives of riders, trainers and owners, was on hand to watch Mr. Martin go down. One woman screamed because she mistakenly thought her husband had been the one injured.

The authorities did little to determine why Mr. Martin’s horse, Phire Power, broke down. The commission said drug tests found no prohibited substances, but the scope of those tests is unclear, including whether the horse was tested for bute. The state also said the horse’s body did not undergo any postmortem exam before it was destroyed.

Within minutes, Ms. Martin was escorted onto the track to be with her fallen husband. Over the next six months, she would rarely leave his side.

In two days, Mr. Martin had been scheduled to sign autographs at Ruidoso to raise money for injured jockeys. Instead, other jockeys signed autographs to raise money for him.

Since the accident, Mr. Martin has been in and out of hospitals in three cities. He has suffered through infections, pneumonia, nausea, weight loss, bed sores and other problems. He remains paralyzed, unable to move his arms or legs. He breathes with a respirator.

Meanwhile, the racing community has rallied to his side, sending not only words of support but also money to help defray his mounting health care costs. Ruidoso’s owner, R. D. Hubbard, promised $100,000. There have been silent auctions and other fund-raisers. His wife worries that it may not be enough.

Through it all, Mr. Martin refuses to feel sorry for himself.

In December, as he struggled to breathe in a Houston hospital, he told a reporter softly that he had no regrets.

“It’s a bad deal,” he said. “But if I could do it again, I would be right out there doing it. I ride horses. It’s the risk every jockey takes.”

Back home in El Paso, Ms. Martin says her husband derives one of his few pleasures from sitting in his wheelchair next to a window watching horses train silently in the distance.

Mr. Martin’s injury deeply affected Mr. Zamora. He was not only losing a friend from the jock’s room, the sport was losing a rider, a gentleman, who had come to represent the best it had to offer.

“He rode the best horses in the world, but he was worthy of the best horses in the world,” Mr. Zamora said. “He had great hands. He let a runner be a runner instead of going to the whip too early. Them animals loved him, and they ran for him and he understood them. When one didn’t want to run, he let ’em not run. He didn’t take to the whip. You have to understand them — that’s what makes a great horseman. And he was. He was special.”

Last fall, several weeks after Mr. Martin’s spill, Mr. Zamora left the jock’s room for the last time.

“I knew I had come so close, and I couldn’t deal with that.”

Pain, Up Close

It was the third race at Ruidoso on July 11, 2009.

In the stands, Laura and Armando Alvarado sat with their two grandchildren, ages 11 and 14.

The Alvarados were not racing fans, but this was a vacation — they had driven up to the mountain resort from El Paso — and they thought their grandchildren might enjoy watching their first horse race.

Mr. Alvarado took the children down to the rail for a closer look. Ten horses sprinted out of the gate, including a gray Texas-bred quarter horse named Sinful Heart, the same horse that fell several months earlier, nearly killing Chris Zamora.

Just past the finish line, Sinful Heart, with another rider on its back, broke down, collapsing on the track. “The horse is bleeding!” one of the children cried out.

The children were not visibly shaken, but Ms. Alvarado said she was sorry they had to witness death at such a close range. After a few more races, they went shopping.

Five days later, a relative with a passion for racing was visiting the Alvarados, and they all went to the track.

“It was going to be an all-day experience, and I thought how nice to have this man give them all this history and details,” Ms. Alvarado said.

Once again, Mr. Alvarado took the children to the rail to watch the finish of the day’s first race.

This time, a horse broke its leg, pitching its rider — who happened to be Chris Zamora — into the ground, where rider and animal rolled like tumbleweeds across the finish line.

“It was awful,” Mr. Alvarado said. Although Mr. Zamora was not seriously injured, the horse was. “The bone was showing through the skin,” Mr. Alvarado said.

Both children began to cry. “I have never seen anything that horrible close up,” Mr. Alvarado said. “The kids were terrified.”

The horse was euthanized on the track. The family quickly left the premises. Ms. Alvarado said: “I told Armando, just drive. We wanted to get out of there.”

Afterward, her granddaughter said, “I don’t want to go to a racetrack ever again.”

Ms. Alvarado wrote a letter to the editor of the local paper.

“For the sake of the animals and children, we felt compelled to let city officials, agencies and others know of this painful experience and urge you to investigate,” she wrote.

She said she sent copies of the letter to the mayor, the track, its chief veterinarian, the Humane Society and the American Society for the Prevention of Cruelty to Animals.

Ms. Alvarado expected a response.

She never got one, she said.

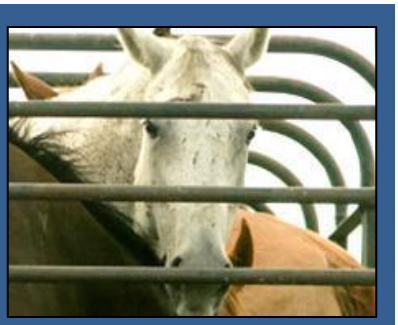
Rebecca R. Ruiz and Matthew Orr contributed reporting from New York.

A version of this article appears in print on March 25, 2012, on Page A1 of the New York edition with the headline: Mangled Horses, Maimed Jockeys. Order Reprints| Today's Paper|Subscribe

factsheet

Support the Safeguard American Food Exports (SAFE) Act (H.R. 113/S.1706)

In the 115th Congress, H.R. 113 was introduced by Rep. Vern Buchanan (R-FL), Jan Schakowsky (D-IL), Ed Royce (R-CA), and Michelle Lujan Grisham (D-NM) and S.1706 was introduced by Sens Robert Menendez (D-NJ), Lindsey Graham (R-SC), Sheldon Whitehouse (D-RI), and Susan Collins (R-ME)



"Here are these exquisite, immensely powerful creatures, who willingly give us their labor in return for our stewardship. They have attended us throughout history, bearing us across frontiers and into battle, pulling our plows, thrilling us in sport, warming us with their beauty… To send these trusting creatures to slaughter is beneath their dignity and ours."

-Laura Hillenbrand, author of "Seabiscuit"

This bill would prevent horse slaughter plants from opening in the U.S. and end the current export of horses abroad for slaughter for human consumption.

Forcing American taxpayers to pay for horse slaughter is fiscally irresponsible. It makes no sense for the federal government to spend millions of taxpayer dollars to oversee new horse slaughter plants. At a time when Congress is so focused on fiscal responsibility and the budget of the USDA's Food Safety Inspection Service is already stretched thin, the USDA should not extend funding for a new program to slaughter horses – a practice that 80% of the American public opposes.

U.S. horsemeat can be dangerous to humans because of the unregulated administration of numerous toxic substances to horses over their lives. American horses are not raised for human consumption, and they are routinely given hundreds of drugs and other substances, both legal and illegal, over their lifetimes that can be toxic to humans if ingested. These substances have not been approved and many have been specifically prohibited by the FDA for use in animals intended for human consumption. For example, a common pain reliever, Phenylbutazone, is known to cause potentially fatal human diseases, and there is no known safe level for residues of this drug in horsemeat. Horses are gathered from random sources, and there is no system in the U.S. to track medications and veterinary treatments given to horses to ensure that their meat is safe for human consumption.

Due to serious food safety concerns, the European Union suspended horsemeat imports from Mexico – where 87% of horses slaughtered for export to the EU are of U.S. origin. EU authorities made the decision after a series of scathing audits that exposed a plethora of problems, including a lack of traceability of American horses and horrific suffering on U.S. soil and in Mexico.

Horse slaughter is inhumane and cannot be made humane. Slaughter is a brutal and terrifying end for horses and is not humane. Horses are shipped for more than 24 hours at a time without food, water, or rest in crowded trucks in which the animals are often seriously injured or killed in transit. Horses are skittish by nature due to their heightened fight or flight response. The methods used to kill horses rarely result in quick, painless deaths; they often endure repeated blows during attempts to render them unconscious and sometimes remain alive and kicking during dismemberment. Before the last domestic plant closed in 2007, the USDA documented rampant cruelty violations and severe injuries to horses, including broken bones protruding from their bodies, eyeballs hanging by a thread of skin, and gaping open wounds.

For more information please contact Holly Gann at hgann@humanesociety.org.

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Supporting Documents for the Horse Racing Integrity Act

1. Articles
    a. 160 Racehorses Died From Injuries Suffered at Charles Town Races Since 2014
    b. A bipartisan approach to protecting racehorses
    c. American racing requires federal oversight, says HSUS boss
    d. Backers, Foes Of Horseracing Integrity Act To Testify Before Congress
    e. Charles Town Classic fiasco this response takes racing in the wrong direction
    f. Combating A Culture Of Cheating A Matter Of Trust
    g. Death of a Derby Winner: Slaughterhouse Likely Fate for Ferdinand
    h. Eight Belles' Death Sparks Controversy
    i. Gorajec Barr-Tonko Bill Only Path Toward Uniformity
    j. Grant Joins HSUS Horse Racing Advisory Council
    k. Handel We Need To End Slaughter Of American Equines
    l. Julie Krone Joins Humane Society's Horse Racing Council
    m. Krone appointed to Humane Society council on racing
    n. Mangled Horses, Maimed Jockeys
    o. The Humane Society of the United States forms national horse racing advisory council
    p. They're embarrassed already but is real humiliation next for these regulators
    q. Trainer Beattie At Rojas Trial: 'Almost Everybody' Illegally Treated Horses On Race Day
    r. What you need to know about the landmark Horse Racing Integrity Act
    s. What's next for Pennsylvania if it doesn't clean up its act?
    t. Why every day is Groundhog Day in U.S. racings rulemaking process

2. Other
    a. Horseracing Integrity Act Fact Sheet
    b. SAFE Act Fact Sheet
    c. The Jockey Club Equine Injury Database

https://www.bloodhorse.com/horse-racing/articles/180859/death-of-a-derby-winner-slaughterhouse-likely-fate-for-ferdinand

Death of a Derby Winner: Slaughterhouse Likely Fate for Ferdinand

Ferdinand, the 1986 Kentucky Derby winner who went on to capture the following year's Horse of the Year title with a dramatic victory over 1987 Derby hero Alysheba in the Breeders' Cup Classic, is dead. *The Blood-Horse* has learned the big chestnut son of Nijinsky II died sometime in 2002, most likely in a slaughterhouse in Japan, where his career at stud was unsuccessful.

Reporter Barbara Bayer, as detailed in an exclusive story in the July 26 issue of *The Blood-Horse*, attempted to learn of Ferdinand's whereabouts after a member of the Howard Keck family that owned and bred the horse inquired about having him returned to the United States, where he began his career at stud. As a racehorse, Ferdinand won eight of 29 starts and earned $3,777,978, retiring as what was then the fifth leading money winner of all time. His victory in the Kentucky Derby gave trainer Charlie Whittingham his first success in that classic, and it was the final career Derby win for jockey Bill Shoemaker.

Ferdinand was retired to stud in 1989 at Claiborne Farm near Paris, Ky., where he was foaled. His initial stud fee was $30,000 live foal, but he achieved little success as a stallion from his first few crops of runners.

Sold to Japan's JS Company in the fall of 1994 at a time when Japanese breeding farms were aggressively pursuing American and European breeding stock, Ferdinand spent six breeding seasons at Arrow Stud on the northern island of Hokkaido, from 1995-2000. Initially popular with local breeders (he was mated to 77 mares his first year), Ferdinand was bred to just 10 mares in his final year at Arrow, and his owners opted to get rid of him.

After efforts by the farm staff to place Ferdinand with a riding club failed, he passed into the hands of a Monbetsu, Japan, horse dealer named Yoshikazu Watanabe and left the farm Feb. 3, 2001. No attempt was made to contact either the Keck family or Claiborne Farm.

Bayer at first was told by Watanabe that Ferdinand had been "given to a friend." When she asked for more information, she was told Ferdinand "was gelded and I think he's at a riding club far away from here." In fact, records showed Ferdinand was bred

to six mares in 2001 and then two in 2002. He spent a period of time at Goshima Farm near Niikappu, where a former handler at Arrow Stud had seen him.

Finally, when Bayer told Watanabe she wanted to see Ferdinand, the story changed yet again. "Actually, he isn't around anymore," she was told. "He was disposed of late last year." Ferdinand's registration in Japan was annulled Sept. 1, 2002, Bayer learned.

"In Japan, the term 'disposed of' is used to mean slaughtered," Bayer wrote in *The Blood-Horse*. "No one can say for sure when and where Ferdinand met his end, but it would seem clear he met it in a slaughterhouse."

"Unfortunately, to those well-versed in the realities beyond the glitter and glory of the racetrack, it comes as no surprise," Bayer wrote. "Ferdinand's story is the story of nearly every imported stallion in Japan at that point in time when the figures no longer weigh in his favor. In a country where racing is kept booming by the world's highest purses and astronomical betting revenues, Ferdinand's fate is not the exception. It is the rule."

"That's just disgusting," said Dell Hancock, whose family operates Claiborne Farm, upon hearing the news of Ferdinand's likely fate. "It's so sad, but there is nothing anyone can do now except support John Hettinger's efforts to stop the slaughter of Thoroughbreds in this country. That wouldn't change anything in Japan...to have this happen to a Derby winner is just terrible."

While the Japanese are among the societies that consume horse meat, it is more likely a slaughtered Thoroughbred would be used for pet food, since the meat consumed by humans is a certain breed of horse raised specifically for that purpose. The slaughter of no longer useful imported breeding stock and many domestic Japanese Thoroughbreds is not uncommon. Shortages of land and the high cost of maintaining a pensioned horse are reasons slaughter is considered an alternate. As in the U.S., where slaughter is also an option available for horse owners, a number of organizations are attempting to provide homes for retired and pensioned racehorses, stallions, and mares. The Japan Racing Association funds one program that currently benefits 90 horses.

Among the people Bayer met and spoke with while trying to learn of Ferdinand's fate was Toshiharu Kaibazawa, who worked as a stallion groom at Arrow Stud during the horse's years there. He called the former champion "the gentlest horse you could imagine. He'd come over when I called to him in the pasture. And anyone could have led him with just a halter on him. ... He'd come over to me and press his head up against me. He was so sweet."

"I want to get angry about what happened to him," Kaibazawa added. "It's just heartless, too heartless."

http://www.humanesociety.org/news/press_releases/2016/06/nat-horse-racing-adv-council-formation-062216.html

The Humane Society of the United States forms national horse racing advisory council

After constructive discussion with other members of the Coalition for Horse Racing Integrity on animal welfare issues and working with thoughtful and progressive leaders committed to elevating the welfare standards in horse racing, The Humane Society of the United States announced the formation of its HSUS National Horse Racing Advisory Council. The council is composed of industry professionals and specialists who continue to promote higher animal welfare standards within the scope of their involvement in horse racing.

"The HSUS is serious about its responsibility to engage with sensible leaders within different industries where there are animal mistreatment issues to find a pathway for reform," said Wayne Pacelle, president and CEO of The HSUS. "Everyone who makes or has made a living from the horse racing industry has a moral obligation to take all reasonable steps necessary to protect and enhance the welfare of the equine athletes who are the heart and soul of the sport and the business of horse racing."

Joe De Francis will chair the council. A long-time animal advocate, he is the former CEO and controlling shareholder of the Maryland Jockey Club, which is the corporate parent of Laurel Park and Pimlico Race Course (home of the Preakness Stakes, the middle jewel of Thoroughbred racing's Triple Crown).

In addition to DeFrancis, council members include a diverse set of stakeholders within the industry, including Jim Gagliano, Stacie Clark-Rogers, Allen Gutterman, Joe Gorajec, Staci Hancock and Chris McCarron.

"I am both honored and excited to be working with The HSUS and with the outstanding and dedicated individuals who will comprise the council," said DeFrancis. "I have every expectation and confidence that the council will be a catalyst for the enactment of federal policies for the betterment of horse racing, to the benefit of all involved: horses, industry participants and fans."

Marty Irby, senior director of rural outreach and equine protection at The HSUS, said: "The establishment of our National Horse Racing Advisory Council is a tremendous step forward for the welfare of equines, the promotion of humane practices and standards both on and off the track, and for the economic vitality and future of the horse racing industry. We are grateful for the opportunity to work with each of these dedicated professionals who recognize the problems that must be solved and want the sport to thrive and flourish, while maintaining the highest standards of animal welfare."

The formation of the council follows the recent release of Pacelle's latest book, The Humane Economy: How Innovators and Consumers are Transforming the Lives of Animals, which delves into the revolution in American business and public policy that is changing how we treat animals and conduct commerce. The book includes an in-depth discussion of how consumer demand for animal welfare improvements is transforming the animal entertainment model.

“The horse racing industry should no longer be an outlier in the humane economy,” added Pacelle. “It’s time for the industry, and the Congress, to adopt a set of independent rules to end doping of horses.”

Media Contact: Stephanie Twining, 301-258-1941, stwining@humanesociety.org

http://www.jockeyclub.com/default.asp?section=Advocacy&area=10

Equine Injury Database

Equine Injury Database

Click here for participating racetracks.

Click here for facts about the EID.

The Equine Injury Database™ is the Thoroughbred industry's first national database of racing injuries. Launched by The Jockey Club in July 2008, the Equine Injury Database seeks to:

- identify the frequency, types and outcome of racing injuries using a standardized format that will generate valid statistics
- identify markers for horses at increased risk of injury
- serve as a data source for research directed at improving safety and preventing injuries

Racetracks, racing organizations and training centers interested in signing up for the Equine Injury Database should contact Kristin Werner Leshney at kleshney@jockeyclub.com or (859) 224-2720.

The following table presents the comparable fatality rates based on the updated analysis of data collected in the Equine Injury Database.

Thoroughbred Only								
Calendar Year	**2009**	**2010**	**2011**	**2012**	**2013**	**2014**	**2015**	**2016**
Rate	2.00	1.88	1.88	1.92	1.90	1.89	1.62	1.54

https://www.thoroughbredracing.com/articles/theyre-embarrassed-already-real-humiliation-next-these-regulators/

They’re embarrassed already, but is real humiliation next for these regulators?

Joe Gorajec | MARCH 14, 2018 |
2 Comments

SHARE

Penn National Race Course in Pennsylvania: the four horsemen in the case had been operating in the state without incident for years

The news of four horsemen suing Tom Chuckas, Thoroughbred Bureau Director of the Pennsylvania Horse Racing Commission, should be of interest to all racing participants across the globe.

This lawsuit involves the authority and tactics of regulators, along with due process protections afforded licensees. These are the type of issues that arise repeatedly everywhere there is a starting gate and a finish line.

This case begins with suspicions of hidden ownership and program trainers (hiding the identity of the actual trainers). Regulators then issued subpoenas and suspensions. Those actions turn into charges of unjustified, unprivileged, and unlawful acts of a Pennsylvania regulator towards its horsemen.

I'll first focus on two critical points: the appropriateness of the subpoenas and the suspensions that followed when four horsemen did not submit the requested information.

The subpoenas

On October 13, 2017, the four horsemen in question - Marcos Zulueta, Juan Carlos Guerrero, Silvio Martin and Sean Mitchell - were served subpoenas by the commission that, according to court filings, were based on anonymous complaints of hidden ownership and program trainers. The requested information included documents covering a three-year period. The specific information included phone records, emails, tax returns and bank statements. Also requested was the location of these trainers' horses at any given time over that three-year period.

The subpoenas allowed 20 days to gather and produce this information and informed the horsemen that failure to comply might result in the suspension of their licenses. The horsemen, believing that the subpoena was unreasonable, overbroad, and oppressive, submitted a motion to quash/amend the subpoena.

In my 25 years as the Director of the Indiana Horse Racing Commission, I have never known of this type of regulatory reaction (or, more precisely, overreaction) to anonymous complaints.

I asked attorney Robin Babbitt to opine. Mr. Babbitt has served as the attorney for Indiana Horse Racing Commission staff since 1994, when pari-mutuel wagering began in the state. Over the course of his years as outside

counsel, Mr. Babbitt never lost a court case representing commission staff while I served as Indiana's Executive Director.

Regarding the issuance of the subpoenas, Mr. Babbitt said, "An administrative subpoena is not something you can take lightly. In regulation you must be careful about laying a foundation for any action you take. This was a situation where there may have been a suspicion of a violation, but that can be a problematic basis upon which to issue a subpoena."

How do you lay that foundation?

Complaints of either hidden ownership or program trainers are not uncommon. In Indiana, we averaged two or three such complaints every year. Our first response would be to collect information to determine if one of our rules had been violated.

Investigators would speak with licensees and others who might possess relevant information. Often, former employees, exercise riders and feed suppliers were just some of the people who provided valuable information.

Stable gate records would be reviewed. These records include the flow of horses in and out of the stable area along with the driver's name and license plate number of the vehicle. Verifying ownership of these vehicles has led to relevant evidence in prosecuting program trainers.

Investigators would order and scrutinize certain winner's circle photographs. We've been surprised at the number of times this has aided our investigation.

These are just a few steps we took. If, after doing our due diligence, we had reason to believe that a violation may have occurred, we would interview the

suspected individuals ‘on the record’ in a tape-recorded interview. We would often follow up the interview with a written request for information.

In Indiana, we would never – ever – issue a subpoena based solely on ‘anonymous’ complaints.

In Pennsylvania, the lack of laying such a foundation was not the subpoena’s only deficiency.

Any information requested in a subpoena must be related to the potential violation. That information must be reasonable, and the person must be given sufficient time to respond. The commission’s subpoena was problematic in all three areas. It would be difficult to get one year of phone records in 20 days, but three years? And how to you track the whereabouts of a stable full of horses retroactively for a three-year period?

The suspensions

Despite their plea to amend the subpoenas, the four horsemen were suspended without a hearing on November 7, 2017.

I believe that this was an extraordinary action. Or, yet again, overreaction.

An immediate suspension without a hearing, also known as a summary suspension, is usually reserved for licensees that pose an immediate threat or danger to the sport or its participants.

Where was the immediate danger? These horsemen had been racing without incident in Pennsylvania for years. None of them had been charged with a rule violation. By simply not complying with a request for information they’re out of business? So much for reasonable enforcement.

Upon an appeal of these suspensions, a hearing was held in front of the full commission on November 29, 2017. The commission subsequently upheld the suspensions and refused to quash or modify the subpoenas.

The Commission gets schooled

The next round in this saga was held on January 11, 2018, in an argument between parties in the Commonwealth Court of Pennsylvania before President Judge Mary Hannah Leavitt.

The venue change from the commission office to the court room was the result of an Application for Stay by the suspended horsemen. This was their opportunity to convince a judge to lift their suspensions temporarily, until a final decision could be made in their case.

My experience in Indiana is that the granting of a stay by a court is an uphill battle for horsemen. Few licensees challenged the Indiana commission in court for a stay. All were denied.

The difficulty in obtaining a stay is meeting the demanding criteria.

Judge Leavitt outlined in her order the criteria the Pennsylvania Supreme Court had established for the granting of a stay:

- The petitioner (horsemen in this instance) must make a strong showing that he is likely to prevail on the merits.
- The petitioner has shown without the requested relief he will suffer irreparable harm.
- The issuance of a stay will not substantially harm other interested parties in the proceedings.
- The issuance of a stay will not adversely affect the public interest.

In her opinion dated January 12, 2018 (the day after the hearing), Judge Leavitt ruled in favor of the horsemen on every criterion.

One finding in Judge Leavitt's decision does not bode well for the Pennsylvania Horse Racing Commission. In a signal of what her final decision may be, she said that the horsemen had made "a substantial case on the merits".

It is an understatement to refer to this particular language as an embarrassment for the Commission.

These horsemen are back racing, but damage, both financial and to their reputation, has already been done. These horsemen had been suspended for over two months, unable to earn a living, based on suspensions that, apparently, should have never been issued.

What happens next?

On February 13, 2018, these same four horsemen filed in United States District Court a complaint against Thomas Chuckas, Thoroughbred Bureau Director of the Pennsylvania Horse Racing Commission.

The complaint alleges, in part:

As a result of Chuckas's unjustified, unprivileged and unlawful acts towards Plaintiffs, they have been deprived and continue to be deprived their constitutionally protected property interest in their trainer's license and their reliance on that license to pursue their chosen profession. These actions also deprive Plaintiff of their liberty right to pursue their chosen profession. Defendant's aforesaid unlawful conduct was knowing and intentional and done with malice.

I have no idea whether this lawsuit has merit.

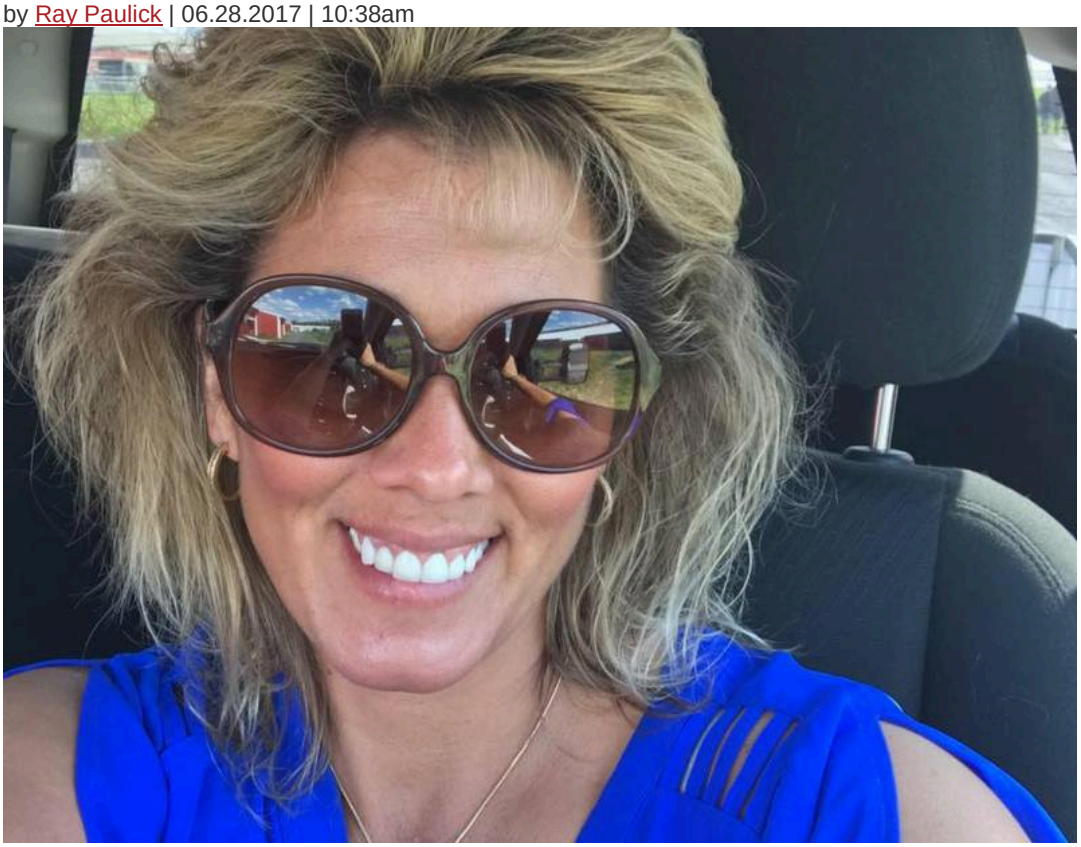
It does appear that its outcome will determine if the commission is merely embarrassed by this whole episode - or humiliated.

Joe Gorajec has spent his entire adult life in the racing industry and served as the executive director of the Indiana Horse Racing Commission for 25 years (1990-2015). He is also a former chairman of the North American regulators' trade association, the Association of Racing Commissioners International (2008). Now semi-retired, he spends his time consulting, writing and gardening at his central Indiana home.

https://www.paulickreport.com/news/ray-s-paddock/trainer-beattie-rojas-trial-almost-everybody-illegally-treated-horses-race-day/

Trainer Beattie At Rojas Trial: 'Almost Everybody' Illegally Treated Horses On Race Day

by Ray Paulick | 06.28.2017 | 10:38am



Stephanie Beattie testified Tuesday at the trial of Murray Rojas in Harrisburg, Pa.

Stephanie Beattie threw fellow horsemen under the bus during her testimony for the prosecution Tuesday afternoon at the federal trial of Murray Rojas, a former rival for leading trainer honors at Penn National in Grantville, Pa.

Beattie admitted she routinely had her horses illegally treated with therapeutic medications on race day by the same veterinarians who counted Rojas as a client.

“Almost everybody did,” Beattie said of the practice. “Ninety-five to 98%. It was a known practice. We wanted to win and they weren't testing for those drugs at that time.”

Beattie, 46, won enough races to be Penn National's leading trainer on three occasions. In 2009, her best year, she won 222 races from 811 starts for earnings of $3.4 million. The previous year, when she won 212 races from 612 starts, she had a win percentage of 35 percent.

But it is two-time Penn National leading trainer Rojas, not Beattie, who is on trial for wire fraud, conspiracy and misbranding of prescription drugs. Assistant U.S. Attorney William Behe has laid out a case with testimony and documents from racing officials, veterinarians and vet assistants alleging Rojas requested and received race-day treatment of horses in order to win purse money, then had billing and treatment records falsified to conceal the cheating.

Beattie is among numerous individuals at Penn National under investigation by the Federal Bureau of Investigation. She resisted cooperating with the FBI at first, Beattie testified, even after Special Agent Bruce Doupe told her, “If you don't want to talk, I'll come to your house at 4:30 in the morning, handcuff you and put you in jail for a very long time.”
Finally, Beattie said, after spending more than $60,000 on legal advice, she decided to cooperate with authorities, submitting to numerous interviews and even wearing a recording device on their behalf.

Despite admitting to years of rule violations in multiple states, Beattie has not been sanctioned by any racing commissions and has faced no criminal charges. It has hurt her business, as shown by a 2016 record of 14 wins from 111 starts and earnings of $217,655.

“This investigation has made things tough for me,” she said.

Beattie also said she has stopped cheating with race-day treatments.

Beattie explained how veterinarian Kevin Brophy established an order form for trainers to fill out their race day medication requests. She said her lists regularly included Kentucky Red, Estrone and Amicar – substances that are not permitted within 24 hours of a race.

Beattie testified that Brophy and other veterinarians informed her of which drugs the state's testing lab was not testing for.

On Monday, Brophy's associate veterinarian, Fernando Motta, testified that Rojas regularly requested and received treatments of Robinul and Estrone on race day for her

horses. Motta beat the test for Robinul, he testified, by administering a lower dose and changing the route of administration to intravenous from intramuscular.

Under cross examination by Robert Goldman, attorney for Rojas, Beattie admitted she never secretly recorded Rojas admitting she had her horses illegally drugged. “There wouldn't be, because we don't talk,” Beattie said.

“You don't like her, do you?” said Goldman, who then revealed that Beattie made fun of Rojas by dressing up like her at a Halloween costume party.

Goldman then recited Beattie's history of medication violations, dating back to her earliest years as a trainer, including a 2005 suspension at Charles Town in West Virginia when officials searched her vehicle and discovered loaded syringes.

Goldman asked: Why did she have injectables?

Beattie responded: “I was giving medication at Lasix time, like everyone else was.”

Beattie denied under oath that she would have shock wave therapy performed on a horse on race day and then have her veterinarian turn in a false name. She said, however, it was “common practice” for horses shipping in to have received shock wave therapy that same day.

She also said she never directed her jockeys or stable employees to use electrical devices, commonly known as buzzers or batteries, to shock horses both during morning workouts and races, as alleged by her former boyfriend and training partner, David Wells. Wells pleaded guilty to charges of rigging a race in a deal with federal prosecutors.

“Did I ever ask them to, no,” Beattie said. “Does it happen at every racetrack, yes. But I never told my jockeys to do it.”

This entry was posted in NL Article, Ray's Paddock and tagged David Wells, drugs in horse racing, Fernando Motta, Kevin Brophy, murray rojas, penn national, Pennsylvania corruption, pennsylvania horse racing, pennsylvania racing, Pennsylvania trial, Ray Paulick, stephanie beattie, William Behe by Ray Paulick. Bookmark the permalink.

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What you need to know about the landmark Horse Racing Integrity Act

Joe Gorajec | JULY 17, 2017 |
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The new Horse Racing Integrity Act would provide the uniformity in regulation that is the accepted standard in nearly all professional sports.
Photo: NYRA.com

Ask a horse owner or trainer in the United States about racing's anti-doping program and you're likely to receive a quizzical look or a blank stare. That's because 'anti-doping' is not a term most of us associate with horse racing.

If you get any response at all, it's likely to be, "We have a drug-testing program." And indeed, racing does have such a program.

Protecting the integrity of the sport, however, involves much more than just drug testing.

Under a Federal bill that introduced in May (which built upon a similar bill from 2015) Congress would empower the **United States Anti-Doping Agency (USADA)** to take the reins of a new, private, not-for-profit anti-doping authority to create and manage a comprehensive nationwide anti-doping program for horse racing. The proposed legislation, formally known as H.R. 2651, is named the **Horse Racing Integrity Act of 2017 (HRIA)** and it was introduced in the House of Representatives by Rep. Andy Barr of Kentucky and Rep. Paul Tonko of New York.

This bill is materially similar to the version introduced by the same sponsors in 2015, but it has been enhanced in a number of respects. The most significant change is that the scope of the national anti-doping program has been expanded to include Standardbred and Quarter Horse racing. Another important change is a prohibition on administration of any medications on race day, including furosemide (Lasix).

I would strongly recommend that any person with a stake or an interest in the sport of horse racing become familiar with this legislation. The complete 46-page bill can be found here.

I am providing a brief overview of the bill's most important components, along with a description of the status quo. I have included my thoughts on how the new legislation might be applied. In the spirit of full disclosure, I have been, and remain, a staunch advocate for this legislation.

Authority and scope

The legislation would require the creation of an entity named the **Horseracing Anti-Doping and Medication Control Authority** (the 'Authority'). That entity

would be governed by an independent board of directors, a majority of whom come from USADA. It would be responsible for developing and enforcing a national uniform anti-doping and controlled medication program for horse racing.

The program would apply to all Thoroughbred, Standardbred, and Quarter Horse racing. The Authority's jurisdiction would cover all horses and participants involved with racehorses of the three breeds. This would include owners, trainers, veterinarians, grooms, etc.

The Authority's anti-doping and medication control program would supersede individual state racing commission's rules and the Authority would be the sole enforcement authority for all matters covered by the program. This control is limited to the anti-doping and medication control program and does not extend to other matters traditionally governed by state commissions, such as race dates, licensing, and investigating and issuing penalties for all other types of rule violations.

The Authority would be subject to limited oversight by the **Federal Trade Commission (FTC)**. The FTC will be responsible for approving the Authority's rules (after public comment), selecting administrative law judges to hear appeals of the Authority's sanctions and acting as the final appellate body in sanctions matters.

Structure

The Authority will be governed by a 13-member board of directors. The board will consist of the Chief Operating Officer of USADA, six directors from the USADA Board, and six individuals, appointed by USADA from nominees of a variety of equine industry constituencies. Each of the six 'at large' industry board seats is earmarked to people with demonstrated expertise in a variety of areas, such as veterinary treatment of race horses, training race horses, jockeys/drivers, etc.

To ensure independence, all directors will be subject to strict conflict-of-interest standards.

Horse racing expertise

The Horse Racing Integrity Act requires the Authority to establish one or more standing advisory or technical committees in establishing the anti-doping program.

The racing industry would be well served to have the **Racing Medication and Testing Consortium (RMTC)** appointed as the primary standing advisory committee. Established in 2002, the Consortium is an organization that represents 23 racing stakeholder groups. Most of the progress made by the racing industry regarding medication and testing over the past decade has been the result of the efforts of the RMTC.

In order to strengthen the industry's most important advisory committee, the RMTC should expand to include two additional areas of expertise: human anti-doping and international horse racing.

Human anti-doping expertise should be present at all levels of the Authority's structure, including the ground floor of rulemaking and policy development.

The added value of international perspective is consistent with one of the goals of the legislation. The legislation specifically states that "…rules, procedures and enforcement policies should be implemented consistent with internationally accepted best practices ..."

Uniform anti-doping rules

The Horseracing Integrity Act requires the Authority to establish a body of rules for its anti-doping and medication control program, including lists of permitted and prohibited medications, lab accreditation and testing standards, adjudicatory procedures and sanctions.

Currently, most medication and testing rules begin as recommendations from the RMTC. From there, two committees of the **Association of Racing Commissioners International (RCI)** - the Drug Standards and Practices Committee and the Model Rules Committee – consider any recommendations. If approved, the RCI Board of Directors votes to adopt the recommendations as model rules. From that point, to achieve uniformity, each individual state commission (of which there are over 30) must promulgate the model rule.

The state-by-state approval process has frustrated industry stakeholders and fans for decades. Many model rules take years to promulgate – while some states never even try to pass certain rules.

The best way forward under the legislation would be for the Authority to receive recommendations directly from the RMTC and subsequently commence its rulemaking process. This streamlined process would still allow for further opportunity for public comment. Any rule approved would thus become effective simultaneously in all states.

In other words, true uniformity.

List of permitted and prohibited substances

The legislation requires the Authority to develop, maintain, and publish a list of permitted and prohibited substances and methods. The legislation sets the RCI's Uniform Classification Guidelines and Foreign Substances and the Prohibited List of the **World Anti-Doping Agency (WADA)** as a starting point but also makes it clear that the Authority has full latitude to make its own rules (subject to FTC approval).

The Authority will have a head start in providing these required lists. The RCI classification guidelines are current, time-tested, and have served the industry well for over two decades. In December 2016, the RCI adopted a Prohibited Substance List designed after the WADA code.

Under the Authority, the industry will not be left waiting for several years as states attempt local adoption. The Authority would make these lists effective simultaneously in all states.

Laboratory accreditation and testing standards

The legislation requires the Authority to establish procedures, standards and protocols for accredited laboratories. The Authority may extend interim accreditation to those laboratories accredited by the RMTC. Currently, ten of the 14 laboratories conducting testing on race horses in the U.S. are accredited by the RMTC.

The current accreditation process focuses on testing procedures and protocols. In short, laboratories are accredited for what they are capable of doing instead of the work they actually perform for their clients, the commissions.

The deficiency of this accreditation process is one of authority. The RMTC lacks the authority, as does any currently constituted national body, to require laboratories to find certain drugs at a mandated concentration. That authority currently resides with each state racing commission. For that reason, there is a perception of a wide disparity in drug testing between states.

Under the HRIA, the Authority would establish such a national uniform standard. Compliance would be mandatory and uniformity would be achieved.

Testing and sampling

The HRIA permits each state racing commission to choose its own laboratory as long as the laboratory is, and remains, accredited.

In all likelihood, the laboratories currently testing horse racing samples would continue to do so. The primary difference, however, is that these laboratories would be required to perform up to the standards set by the Authority. Failure to meet the Authority’s standards would place a laboratory’s accreditation at risk.

The Authority would also establish standards for **out-of-competition testing (OOCT)** that would be applied uniformly in all states. The OOCT program would require the disclosure of the whereabouts of all horses in training at all times. This would greatly improve the current situation where only a handful of states have an effective deterrent for blood doping.

Investigations

The legislation gives the Authority the responsibility to investigate any violation of its anti-doping rules.

State commissions now have similar authority that is limited to its provincial borders. Most commissions have a Director of Security and/or investigators who conduct investigations into the conduct of licensees and other persons. State investigators have broader responsibility because their investigative authority spans rule violations of any type, whereas the Authority would be limited to its anti-doping rules.

USADA's approach to regulation is highly principled and integrity based. Its independence assures an even-handed, show-no-favorites application of its authority to investigate and sanction. An ideal approach, at least for the most important investigations, would be for the Authority to partner with USADA to utilize its expertise and time-tested strategies and combine them with the specialized knowledge of local commissions' boots-on-the-ground investigators.

Due process

Under the legislation, the Authority has the power to sanction individuals who violate its anti-doping rules. The HRIA requires the Authority to establish a schedule of sanctions for violations and rules for due process that include impartial hearings.

The disciplinary processes that state commissions and USADA currently follow have one procedure in common: the ability to accept an agreed upon sanction. Most trainers accept the RCI Uniform Model Rule for Penalties either as the result of a stewards' hearing or in lieu of a stewards' hearing. Consequently, a very small percentage of alleged violations are appealed.

Presently, the adjudication process for appeals varies from state to state. The most common model allows for state racing commissions to appoint an ALJ (Administrative Law Judge) to conduct an administrative hearing and issue a recommended decision. The commission may adopt, reject, or amend the ALJ's recommended decision. This process is followed in most appeals of an adverse ruling from the Board of Stewards after an initial hearing is provided at the track. In some instances, the initial hearing is by-passed and the first and only administrative hearing is before an ALJ.

A common complaint of the current adjudication process is the perception of a lack of independence. This is the result of ALJs being appointees of the relevant state commission or otherwise affiliated with state government. Under the HIRA, those sanctioned by the Authority will have the right to a speedy appeal covering all the merits of their individual cases, which will be heard by independent administrative law judges not associated with the Authority in any way.

Cost

The cost of the anti-doping program has yet to be determined. Preliminary estimates provided to the **Coalition for Horse Racing Integrity** indicate the additional cost of the new anti-doping program would likely not exceed an average of $60 per start. This estimate assumes that all current funding for anti-doping remains in place. I understand that much of this increased cost comes from the expansion of out-of-competition testing.

Although the cost is expressed in dollars per start, the allocation of cost among stakeholders could vary from state to state. The HRIA provides flexibility in determining the source of funding. The legislation reads as follows:

Each State racing commission shall determine, subject to the applicable laws and regulations of the State, the method by which the requisite amount shall be allocated, assessed, and collected, provided that in no event shall the funds be obtained by means of an increase in the takeout.

Final thoughts

The Horse Racing Integrity Act of 2017 would provide the uniformity in regulation that is the accepted standard in nearly all professional sports. This landmark legislation, however, brings much more than uniform application of standardized rules and protocols. It promotes the principles of integrity, excellence and independence, some of which have been long absent in many racing jurisdictions.

Joe Gorajec has spent his entire adult life in the racing industry and served as the executive director of the Indiana Horse Racing Commission for 25 years (1990-2015). He is also a former chairman of the North American regulators' trade association, the Association of Racing Commissioners International (2008). Now semi-retired, he spends his time consulting, writing and gardening at his central Indiana home.

https://www.thoroughbredracing.com/articles/whats-next-pennsylvania-if-it-doesnt-clean-its-act/

What's next for Pennsylvania if it doesn't clean up its act?

Joe Gorajec | NOVEMBER 07, 2017 |
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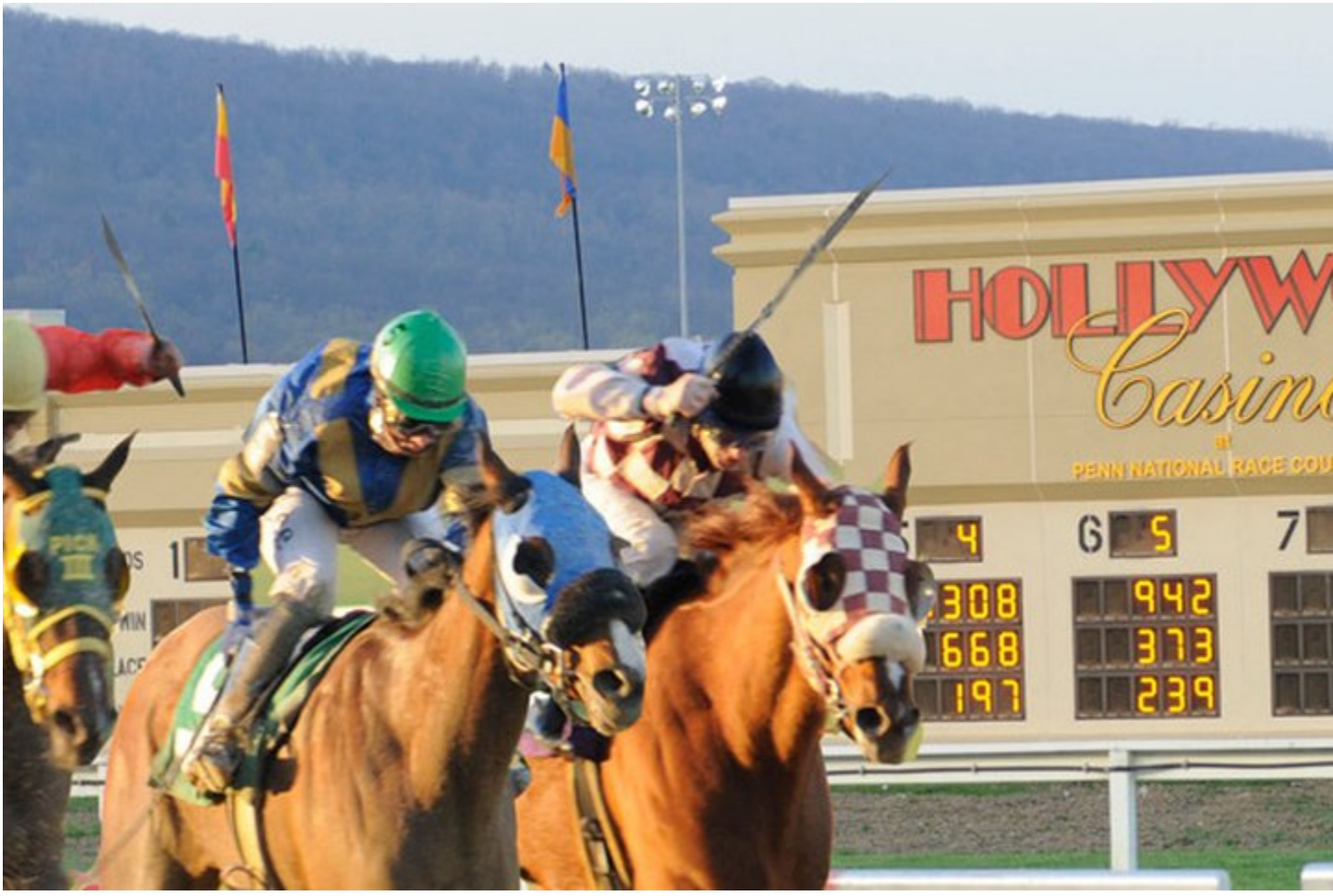

Penn National Race Track: some prosecutions for fraud have involved officials there. Photo: hollywoodpnrc.com

Play the word association game with the term 'Pennsylvania horse racing industry'. Are the most frequent responses 'sleazy' and 'corrupt'?

Unfortunately, Pennsylvania has earned that reputation.

Mr. Stuart S. Janney, Chairman of the Jockey Club, chose to address the troubles in Pennsylvania in his closing remarks at this past summer's Round Table conference in Saratoga Springs, New York.

Mr. Janney said, "What has happened in Pennsylvania recently is disgraceful and sad, especially when you consider that the state is the sixth leading producer of foals and that it hosted approximately 4,000 races and distributed more than $100 million in purses in 2016. Let's start by focusing on the federal trial involving trainer Murray Rojas on charges of fraud, conspiracy, and misbranding of drugs. I think it illustrates what we have to fix and how our problems interconnect.

"Uncontradicted testimony described widespread, in fact, nearly universal, cheating; regulators asleep on the job; a corrupted and ineffectual testing system."

This 'widespread cheating' includes guilty pleas from four veterinarians who treated horses with impermissible medications on race day. This practice, evidently, had been ongoing for over 20 years.

Fraud and conspiracy cases

The corruption in Pennsylvania has not been limited to the case of Ms. Rojas.

In 2014, the clocker at Penn National Race Track pleaded guilty to criminal charges of wire fraud by accepting cash to falsify workouts, thus defrauding the betting public.

Also in 2014, Pennsylvania trainer David Wells was indicted on multiple counts of conspiring to fix races. He was alleged to have administered substances to horses on race day during a four-year period from 2009-2013. Mr. Wells pleaded guilty to one count of "rigging a publicly exhibited contest" as part of a deal with federal prosecutors. He was subsequently ordered to

serve a three-month prison sentence in addition to a mandatory work release program and four-and-a-half years on probation.

In 2015, another Penn National racing official agreed to plead guilty to wire fraud by accepting cash for divulging inside information to trainers involving the entry of horses.

Mr. Janney's Round Table conference remarks did not go unchallenged by the Pennsylvania State Horse Racing Commission. In a letter dated September 26, 2017, to Mr. Janney, Commission Chairman Russell C. Redding characterized Mr. Janney's remarks as "inappropriate and inaccurate", "faulty" and "highly unprofessional".

Mr. Janney's rebuttal can be found here.

Of all Mr. Janney's remarks, the one that most likely hit a nerve was that regulators were asleep on the job.

When prosecutors step in

Mr. Redding counters that, "From day one, this has been a cooperative joint effort between all branches of regulatory and law enforcement … The criminal actions of the veterinarians and horsemen who were successfully prosecuted are reprehensible, but do not confuse that with the actions of the SHRC [the Commission]. There is no evidence of "regulators asleep on the job".

So, were regulators asleep on the job?

Of course, they were.

Mr. Redding just doesn't get it.

Federal prosecutors only involve themselves in these types of rule violations when state regulators are unwilling or unable to effectively carry out their statutory duties to protect the integrity of its racing product.

Simply stated, Pennsylvania regulators wouldn't clean up their own mess, so the federal government stepped in and did it for them.

So, where does Pennsylvania horse racing industry go from here?

It is problematic that most of the people involved in this longstanding and widespread culture of cheating, absent a few individuals, are still involved in racing in Pennsylvania. Once part of this cheating culture, can these people change their ways? Will the newly appointed regulators strive to deter, detect, and forcefully prosecute violations of the rules of racing? Or will they default to the same "hear no evil, see no evil and speak no evil" posture as their predecessors?

What are the remedies if this type of corruption continues?

Slot machine revenue

Is the answer to shut down racing in the state?

If so, this would not be initiated from within Pennsylvania's racing industry. These people are the beneficiaries of the program. Nor would influences outside the state have any bearing on this question. The citizens of Pennsylvania and their elected representative would likely decide. That's

because State lawmakers have the power to direct monies that are derived from slot machines at the track.

The demise of any racing track in the United States would likely be the result of lawmakers discontinuing the flow of slot machine revenue to racing industry. This money, in Pennsylvania and elsewhere, has become the lifeblood of purse funding, and in many cases, is also the revenue that keeps race tracks profitable. Most tracks and horsemen simply cannot rely solely on the revenue derived from pari-mutuel betting.

Should the elected officials in Pennsylvania determine that the racing industry is undeserving of slot machine revenue, its racing program would quickly collapse like a house of cards.

When we play the word association game a decade from now, how will you respond?

I say, “Pennsylvania horse racing industry” and you say, “classy mid-level racing”.

Or “defunct”?

Joe Gorajec has spent his entire adult life in the racing industry and served as the executive director of the Indiana Horse Racing Commission for 25 years (1990-2015). He is also a former chairman of the North American regulators’ trade association, the Association of Racing Commissioners International (2008). Now semi-retired, he spends his time consulting, writing and gardening at his central Indiana home.

https://www.thoroughbredracing.com/articles/why-every-day-groundhog-day-us-racings-rulemaking-process/

Why every day is Groundhog Day in U.S. racing's rulemaking process

Joe Gorajec | APRIL 04, 2018

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In the film Groundhog Day, Bill Murray's character modified his behavior each day in order to pursue his goals, rather than doing the same thing every time

You remember the movie 'Groundhog Day'? Of course, you do! The one where Bill Murray is stuck in Punxsutawney, Pennsylvania. He is living the same day over and over and over again. Every morning he wakes up, desperate to leave Punxsutawney.

For every newly proposed model rule, U.S. racing experiences its own Groundhog Day. We wake up each morning only to confront the same ineffective rulemaking process over and over again, in 30+ different states.

One such example is a set of recommendations made by the Racing Medication and Testing Consortium (RMTC) that, if enacted, would upgrade existing out-of-competition testing rules. The Association of Racing Commissioners International (RCI) voted in December 2016 to approve these recommended rules as a model to be approved separately in each state.

Although I was skeptical, my hope was that all states would rally around this important recommendation and approve it in unison - and within a year.

Lackluster results

I wrote a *TRC* column to that effect in April 2017, titled “Out of Competition testing: after a decade of neglect, what’s next?”

In that piece I opined:

By the way, the new rule is terrific. It is thoughtful, well-crafted, and accompanied by a Prohibited Substance List. This list is modelled after that of the World Anti-Doping Agency (WADA), meaning it is consistent with international anti-doping standards. It greatly expands the number and classes of prohibited drugs. The new list includes, in addition to blood-doping drugs and methods, a broad spectrum of anabolic steroids, peptide hormones, growth factors and related substances. Many of these types of drugs are likely to be detected only in an out-of-competition environment.

Most states can complete the rulemaking process in a year. The start date for this process began when the model rule was adopted by the RCI in December 2016. So, by the end of 2017, we should all know where this is headed.

So where do we stand now – 15 months later.

According to the RMTC’s website, as of March 15, 2018, only seven states have passed this set of rules. These are Arkansas, Delaware, Maine, Maryland, Massachusetts, Pennsylvania and Washington. Four other states are in the process of adoption: California, Colorado, Iowa and New York.

Why such lackluster results?

Because there is no central authority in the U.S. for enforcing national uniformity. The RMTC, which has been the driving force for progress in medication and testing issues for over a decade, does not possess the authority to require compliance. Neither does the RCI, the trade association of racing commissions. It all falls to the individual state racing commissions, which have differing rulemaking processes, personalities and agendas.

There is a perception among racing fans that certain horsemen congregate in racing jurisdictions due to lax regulation regarding drugs and testing. The extent to which this is true is, of course, unknown. I believe that rudimentary logic suggests that this occurs, at least to some degree. Any horsemen seeking an edge with an illegal drug that can only be detected through out-of-competition testing would surely gravitate to states that have little or no such testing.

This situation has been a perennial lament of racing fans.

What saved Bill Murray

The initial out-of-competition rule was drafted in 2006, and some states are just now beginning to implement it. If the system doesn't change, we will be waiting another ten+ years for states to pass the new and improved version.

Is there a way out of this exasperating cycle?

Yes, there is.

Just ask yourself - "What would Bill Murray do?"

In the movie, Murray's character changed his behavior over time. And this is what saved him.

Instead of acting the same way, he modified his behavior each day in order to pursue his goals. He dearly wanted to free himself of the repetitious cycle of Groundhog Day, and, just as important, get his dream girl, played by Andie McDowell.

Can the racing industry change its behavior?

Our goal should be to have a single, national rulemaking process that allows for the expeditious implementation of all medication and drug-testing rules.

We do have a lifeline. It is federal legislation called the *Horse Racing Integrity Act of 2017*.

This legislation, introduced by Congressman Andy Barr (R-KY) and Congressman Paul Tonko (D-NY) calls for a unified rulemaking process the racing industry is lacking. It has the support of over 100 members of the U.S. House of Representatives.

If enacted, this legislation would mandate uniformity in medication and testing rules in all states - just like other professional sports.

The answer to racing's rulemaking dilemma is easy. Just ask yourself, "What would Bill Murray do?"

He sure as hell wouldn't stay in Punxsutawney.

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