Committee on Oversight and Reform
Subcommittee on the Environment

Hearing on
"The Devil They Knew: PFAS Contamination and the Need for Corporate Accountability, Part II"

September 10, 2019

Dr. Denise Rutherford, Senior Vice President of Corporate Affairs, The 3M Company

The Honorable Harley Rouda (D-CA)

1. **In your written testimony submitted to the Subcommittee, you stated: "Importantly, the weight of scientific evidence has not established that PFOS, PFOA, or other PFAS cause adverse human health effects." In addition, you testified at the hearing that "there's no cause and effect for adverse human health effects."**

   a. **Can you explain what type of scientific study would be necessary to establish causality in humans?**

At the hearing on September 10, 2019, I testified that a review of the scientific evidence shows no cause-and-effect relationships between adverse human health effects and exposure to per- and polyfluoroalkyl substances, or PFAS, including PFOS (or perfluorooctane sulfonate) and PFOA (or perfluorooctanoic acid), at the levels the general population has been exposed to.

Causal inference rarely can be made based on the results of a single study. A broad range of methodologic perspectives and tools (such as study design, statistical modeling, and biological reasoning) are needed to make such a determination. The process of making a causal determination generally involves assessing the quality of individual studies (of different designs and methodologies) and the consideration of a range of plausible explanations for observed associations including the role that various possible biases, confounding, and other factors may have played in the observed results. Ultimately, causality can only be inferred after a synthesis of the evidence under a set of accepted guidelines—such as those outlined by the English epidemiologist Sir Austin Bradford Hill. Other than temporality, there is no single criterion that is either necessary or sufficient to establish whether an observed association is causal. Kenneth Rothman et al., *Modern Epidemiology* ch. 2 (2008). Rather, each of the guidelines must be assessed, and the body of evidence must be evaluated as a whole.

   b. **What level of statistical significance, if any, in an associative relationship between PFAS chemicals and health effects would you believe is suitable for inferring causality?**

A "p-value," which measures the "level of statistical significance," can be a useful statistical measure, but it can also be misused and misinterpreted. Ronald Wasserstein & Nicole Lazar, *The ASA Statement on p-Values: Context, Process, and Purpose,* 70:2 Am. Statistician, 129-133 (2016). The p-value is a statement about data in relation to a specified hypothetical

explanation, and is not a statement about the explanation itself. A level of statistical significance does not measure the size of the effect, or the importance of the result. The American Statistical Association explains, "Statistical significance is not equivalent to scientific, human, or economic significance. . . . Any effect, no matter how tiny, can produce a small p-value if the sample size or measurement precision is high enough, and large effects may produce unimpressive p-values if the sample size is small or measurements are imprecise. Similarly, identical estimated effects will have different p-values if the precision of the estimates differs." *Id.* at 132. Accordingly, there is not a single level of statistical significance that is sufficient by itself to infer causality. For a discussion of the appropriate approach for assessing causality, see the response to Rouda 1.a.

c. Since establishing true causality in humans would require experimental conditions that are highly unethical and unfeasible with human subjects, why does 3M believe that causality is the standard that must be met before conceding the harmful health effects of PFAS?

3M does not agree that experimental conditions are required to evaluate or establish causality in humans. For a discussion of the appropriate approach to assessing causality, see the response to Rouda 1.a. Moreover, from a scientific perspective, one cannot say that a substance has harmful health "effects" without first establishing a "cause-and-effect" relationship between the substance and the observed phenomenon in humans.

d. Are you aware of studies that establish "cause-and-effect" for adverse health effects in animal subjects? If the answer is yes, please cite those studies and explain why the evidence of adverse health effects in animals is insufficient for making inferences about human health effects.

Toxicology studies are intended to produce a biological response by administering increasing amounts of the test substance to laboratory animals. A wide variety of substances have been shown to cause an adverse response if administered at a sufficiently high dose, and PFOA and PFOS are no different. The levels of exposure that laboratory animals receive in toxicology studies are often much greater than levels typically found in the environment or in humans, and this has generally been the case for laboratory tests on animals exposed to PFOA and PFOS.

Because of the nature of toxicology studies, some of these studies have shown "cause-and-effect" relationships in laboratory animals between PFOA and PFOS exposure at the high levels administered in those studies and certain health outcomes. For example, 3M has conducted or sponsored more than 100 toxicology studies that have resulted in the observation of varying biological responses in the animal subjects. PFOA and PFOS studies involving laboratory rodents, for example, have shown biological responses including decreased body weight, increased liver weight, lowering of blood lipid, and occasionally, when animal subjects are given very high doses well beyond levels typically found in the environment, mortality.

The evidence of adverse health effects observed in some animal studies is insufficient by itself to make inferences about human health for a number of reasons. First and foremost, the doses in those toxicology studies greatly exceed the exposure levels that humans encounter in the

environment, both historically and today. (For further discussion of the relative levels of exposure between animal subjects in toxicology studies and the general human population, see the response to Comer 5.) Second, for a given study, other factors may also limit its relevance to human health. As noted below with specific reference to the Sprague Dawley rat bioassays, the mode or modes of action that give rise to the health effect in animals may not be present in humans. Lastly, in many cases, separate human health studies have looked at the same endpoints reported in the animal studies and failed to show any association between exposure to PFOA or PFOS and that health outcome in humans.

Beyond 3M's own studies, there are other studies that have similarly involved the administration of high (sometimes progressively higher) doses of PFOA or PFOS in order to elicit a response in the studies' animal subjects. Given the number of potentially relevant studies, I have not attempted to summarize the extent to which each of those studies establishes a cause-and-effect relationship between exposure to PFOA or PFOS and an adverse health outcome in laboratory animals. Some such relationships, however, are well known. Examples of studies that have shown causal links involve the development of liver, pancreatic acinar cell, and testicular Leydig cell tumors in laboratory rats exposed to PFOA through their diet for their entire two-year lifetime. Specifically:

- *Liver tumors in rats*: There have been three lifetime bioassays conducted in Sprague Dawley rats with dietary administration of PFOA. Lisa Biegel et al., *Mechanisms of Extrahepatic Tumor Induction by Peroxisome Proliferators in Male CD Rats*, 60 Toxicological Sci., 44-55 (2001); John Butenhoff et al., *Chronic Dietary Toxicity and Carcinogenicity Study With Ammonium Perfluorooctanoate in Sprague-Dawley Rats*, 298 Toxicology, 1-13 (2012); National Toxicology Program (NTP)*, Draft Report: NTP Technical Report on the Toxicology and Carcinogenesis Studies of Perfluorooctanoic Acid Administered in Feed to Sprague Dawley Rats* (2019). The highest dietary PFOA concentration in two of the studies (Biegel et al. (2001); Butenhoff et al. (2012)) was 300 parts per million (ppm) while the dietary concentration in the most recent study (NTP (2019)) included both perinatal exposures up to 300 ppm in the mothers and post-weaning exposures that ranged up to 80 ppm PFOA in the male offspring. Increased incidence of liver tumors was observed in the Biegel et al. (2001) and NTP (2019) studies. The mode of action for the development of liver tumors in rats likely involves the activation of liver nuclear receptors such as PPARα. It is generally recognized that this mode of action in producing liver tumors in rats is not relevant to humans. Agency for Toxic Substances and Disease Registry (ATSDR), *Draft Toxicological Profile for Perfluoroalkyls* (Draft for Public Comment June 2018); U.S. Environmental Protection Agency (EPA), *Health Effects Support Document for Perfluorooctanoic Acid* (2016); J.C. Corton et al., *The PPARα-Dependent Rodent Liver Tumor Response is Not Relevant to Humans: Addressing Misconceptions*, 92 Archives of Toxicology, 83-119 (2018); Frank J. Gonzales & Yatrik M. Shah, *PPARα: Mechanism of Species Differences and Hepatocacinogenesis of Peroxisome Proliferators*, 246 Toxicology, 2-8 (2008); J. Christopher Corton, *Cancer Risk Assessment* ch. 17, 439-481 (2010); J. Christopher Corton et al., *Mode of Action Framework Analysis for Receptor-Mediated Toxicity: The Peroxisome Proliferator-Activated Receptor Alpha (PPARα) as a Case Study*, 44 Critical Revs. Toxicology, 1-49 (2014); James Klaunig et al., *Mode of*

Action Analysis of Perfluorooctanoic Acid (PFOA) Tumorigenicity and Human Relevance, 33 Reproductive Toxicology, 410-418 (2012).

- *Pancreatic acinar cell tumors in rats*: Among those same three bioassays in Sprague Dawley rats with dietary administration of PFOA, there was an increased incidence of pancreatic cell tumors reported by Biegel et al. (2001) and by NTP (2019). The proposed mode of action for the development of pancreatic acinar cell tumors in rats involves stimulation by the cholecystokinin (CCK) enzyme; this mechanism is likely not applicable in humans. James Myer et al., *Species- and Dose-Specific Pancreatic Responses and Progression in Single- and Repeat-Dose Studies with GI181771X: A Novel Cholecystokinin 1 Receptor Agonist in Mice, Rats, and Monkeys*, 42 Toxicologic Pathology, 260-274 (2014); Klaunig et al. (2012).

- *Testicular Leydig cell tumors in rats*: Among the same three bioassays, the two studies in which the rats received a 300 ppm PFOA dose (Biegel et al. (2001); Butenhoff et al. (2012)) showed an increased incidence of testicular Leydig cell adenomas. The third study (NTP (2019)) did not, and the study acknowledged that the absence of testicular Leydig cell adenoma was likely due to the lower maximum dietary PFOA dose used post weaning (80 ppm) or differences among rat stocks between the studies. Proposed modes of action for the development of testicular Leydig cell tumors in rats include increased interstitial estradiol stimulated by aromatase induction and activation of PPARα in the liver. Lisa Biegel et al., *Effects of Ammonium Perfluorooctanoate on Leydig Cell Function: In Vitro, In Vivo, and ex Vivo Studies*, 134 Toxicology and Applied Pharmacology, 18-25 (1995); Klaunig et al. (2012). The relevance of these modes of action to humans has not been established. Klaunig et al. (2012).

Many other studies are collected and summarized in a number of reports by government agencies, including the ATSDR *Draft Toxicological Profile for Perfluoroalkyls* (2018) and the EPA's Drinking Water Health Advisories for PFOA and for PFOS (2016).

2. You also testified the following before the Subcommittee when asked about the link between adverse health effects and PFAS chemicals: "I do appreciate that links and associations are indicated in those scientific studies."

a. Is it the 3M Company's position that the benefits of no regulations on PFAS chemicals outweigh the potential consequences for human health indicated by the "links and associations" you mentioned? If so, why?

No, that is not 3M's position.

I testified at the hearing on September 10 that there are associations between PFAS exposure and human health effects that are indicated in scientific studies, but that there are also inconsistencies in the data and that establishing human health impacts is complex.

3M supports science- and risk-based regulation of PFAS compounds at the federal level and has been actively urging the adoption of such regulations and supporting related legislation. At the same time, regulations not based on science or risk can impose substantial costs on society

with no demonstrable benefit, making it impracticable to produce useful and life-saving products and unnecessarily draining resources that could be better spent on other health or environmental concerns.

3. You also testified before the Subcommittee that 3M is "continuing our studies, and we will work proactively with scientific bodies .... We do agree additional study is required."

a. Please provide a complete list to the Subcommittee of the current studies 3M is conducting or funding (either wholly or partially) regarding PFAS, including the researchers conducting each study and a description of each study.

I testified at the hearing on September 10 that 3M is continuing its human health studies, and I agreed with the government agencies and other bodies that have called for additional study.

3M is currently conducting the following on-going environmental science and treatability studies related to legacy PFAS compounds (including PFOA and PFOS):

- Degradation of Poly- and Perfluoroalkyl Substances (PFASs) in Water via High Power, Energy-Efficient Electron Beam Accelerator (Dr. Cleston Lange, principal investigator; in collaboration with Fermi National Accelerator Laboratory)
- Analytical Method Development and Chemical Characterization of 3M Legacy AFFF (Dr. Cleston Lange, principal investigator)
- Synthesis and Characterization of Analytical Standards for the Quantification of Legacy 3M PFAS (Dr. Cleston Lange, principal investigator)
- Method Validation of a New Low-Level Total Organic Fluoride Analytical Method (Dr. Sudha Marimanikkuppam, principal investigator)
- Expansion of a Direct Injection Isotope Dilution LC/MS/MS Method for the analysis of PFAS in Aqueous Environmental Matrices (Susan Wolf, principal investigator)
- Development of Stack Testing Methods for Measurements of PFAS in Air (Dr. Brian Mader, principal investigator)
- Biodegradation of 3M Legacy AFFF (Dr. Cleston Lange, principal investigator)
- Evaluation of Granular Activated Carbon to Treat Water Containing 3M Legacy AFFF (Dr. Brian Mader, principal investigator)
- Soil Adsorption/Desorption of 3M Legacy AFFF (Chelsie Grochow, principal investigator)
- Hydrolysis and Photolysis of 3M Legacy AFFF (Susan Wolf, principal investigator)
- Evaluation of Ion Exchange Resins to Treat Water Containing PFAS (Jim Kotsmith, principal investigator)
- Field Evaluation of the Destruction Efficiency of AFFF in an Industrial Waste Incinerator (Dr. Brian Mader, principal investigator)
- NIST AFFF SRM Inter-Comparison. (Dr. Cleston Lange, principal investigator)

3M is funding the following on-going treatability study related to legacy PFAS compounds (including PFOA and PFOS), conducted by researchers at Brown & Caldwell:

- Evaluation of Ultrafiltration, Nanofiltration, Reverse Osmosis and Granular Activated Carbon to Treat Landfill Leachate

3M is currently conducting the following on-going health studies related to legacy PFAS compounds (including PFOA and PFOS):

- Biomonitoring of Serum PFAS Levels (Geary Olsen, principal investigator)
- 90-Day Dietary Study in Mice with PFHxS (Sue Chang, principal investigator)

3M is funding the following on-going health studies related to legacy PFAS compounds (including PFOA and PFOS), conducted by researchers at Ramboll:

- PBPK Model Simulation of Potential Chemical Interactions between PFAS and Lipoproteins
- Investigation of the Association of Clinical Thyroid Disease with PFOA and PFOS Concentrations Using Quantitative Bias Analysis with Pharmacokinetic Models
- Quantitative Bias Analysis of the Association Between Subclinical Thyroid Disease and Two Perfluoroalkyl Substances
- Updated Meta-Analysis of the Association of Birthweight with PFOS
- Calibration of a Human Perfluorooctanoic Acid PBPK Model with Plasma and Urine Data from a Controlled Human Exposure Study

3M is funding the following on-going health study related to legacy PFAS compounds (including PFOA and PFOS), conducted by researchers at the University of Minnesota:

- Mortality and Cancer Incidence in Perfluorooctanesulfonyl Fluoride Production Workers at the 3M Decatur Workforce

3M also provides unrestricted research grant funding to researchers at Cornell University, Pennsylvania State University, the University of Kansas Medical Center, and the University of Minnesota. That funding may be used to support the study of PFAS, including legacy PFAS compounds such as PFOA and PFOS.

4. You also testified before the Subcommittee that, "as we moved forward, we saw that [PFOA and PFOS] did bioaccumulate, meaning that they would build up over time with continued exposure, for these two particular materials."

a. Please explain why and how 3M decided that bioaccumulation was a health risk sufficient to justify voluntary phase-out of these chemicals?

I testified at the hearing on September 10 that, in addition to our knowledge about bioaccumulation, our testing capabilities improved over time, allowing us to detect PFAS compounds at lower and lower concentrations.

Bioaccumulation refers to the tendency of PFOA and PFOS to persist in the environment and in the human body, with the potential to build up over time if exposure continues. As technology improved in the 1990s, 3M learned that PFOA and PFOS, in addition to being

persistent, were also widely prevalent. With that knowledge of persistence and prevalence, 3M moved voluntarily to phase out the use and manufacture of perfluorooctanyl chemistries, including PFOA and PFOS. Importantly, even with evidence of both prevalence and persistence, 3M believed the science had not at that time—and still has not—shown that exposure to PFOA and PFOS at the levels present in the environment and in living organisms at the time of the phase out, let alone today, causes adverse health effects in humans. 3M explained at the time of the phase out, "While this chemistry has been used effectively for more than 40 years and our products are safe, our decision to phase out production is based on our principles of responsible environmental management. . . . All existing scientific knowledge indicates that the presence of these materials at these very low levels does not pose a human health or environmental risk."

b. Would 3M agree that the health risks associated with bioaccumulation justifies regulation of PFOA and PFOS by the federal government? If no, why are the dangers of bioaccumulation sufficient to justify a voluntary phase-out but insufficient to justify federal regulation?

3M supports science- and risk-based regulation at the federal level. The fact that PFOA and PFOS bioaccumulate is one important consideration that should be weighed in any regulatory assessment, but bioaccumulation alone does not indicate that health effects will occur.

The Honorable James Comer (R-KY)

1. In your testimony, you stated that the weight of scientific evidence today doesn't support a finding that PFAS cause harmful human health effects. Some of my colleagues suggested that this statement was inconsistent with testimony and documents in the public record. Can you clarify 3M's position on the state of scientific knowledge about the impacts of PFAS, and explain the support for that position?

There has been significant study of PFAS and human health outcomes. Although a subset of human health studies has identified associations between PFAS exposure and a given health outcome or biomarker, reporting associations from individual studies is not the same as establishing causation. In fact, in many cases, the observed association could not be replicated by subsequent studies. In other cases, the association has been attributed to reverse causation. Overall, the relevant evidence has not established a cause-and-effect relationship between exposure to PFAS and adverse human health outcomes at the levels encountered in the environment, historically or today.

This is not just 3M's position. Several health agencies and expert panels have offered their view that the present scientific evidence does not establish that PFOA and PFOS cause human health effects; for example:

- The Michigan Science Advisory Panel opined, "[C]ausality between a PFAS chemical and a specific health outcome in humans has not been established in the current scientific literature." Michigan PFAS Science Advisory Panel, *Scientific Evidence and Recommendations for Managing PFAS Contamination in Michigan*, at 10 (Dec. 7, 2018).

- ATSDR states in its draft toxicology profile that "cause and effect relationships have not been established for any of the [health] effects, and the effects have not been consistently found in all studies." Agency for Toxic Substances and Disease Registry (ATSDR), *Toxicological Profile for Perfluoroalkyls*, at 635-36 (Draft for Public Comment June 2018).

- The Australian Expert Health Panel on PFAS found that "[t]here is no current evidence that supports a large impact on an individual's health" as a result of high levels of PFAS exposure. Expert Health Panel for Per- and Poly-Fluoroalkyl Substances, *Report to the Minister*, at 3 (March 2018).

- The International Agency on Cancer (IARC) evaluated PFOA and characterized it as only "possibly carcinogenic to humans" while finding that there was "limited evidence" in both humans and experimental animals "for the carcinogenicity of [PFOA]." International Agency on Cancer (IARC) Monographs on the Evaluation of Carcinogenic Risks to Humans, *Some Chemicals Used as Solvents and in Polymer Manufacture: Perfluorooctanoic Acid* Vol. 110, 97-98 (updated Dec. 22, 2016). This is the same category IARC assigns to exposure to radio frequency electromagnetic fields, such as those produced by wireless devices, including cell phones.

- The National Toxicology Program (NTP) evaluated PFOA and PFOS and concluded they are "presumed to be an immune hazard to humans," but not "known" to be such (which is NTP's highest hazard identification conclusion). National Toxicity Program Monograph, *Immunotoxicity Associated with Exposure to Perfluorooctanoic Acid or Perfluorooctane Sulfonate* (Sept. 2016). That conclusion was based, in part, on "moderate confidence in the body of evidence for an *association* between exposure to PFOA or PFOS and" suppressed antibody response from human studies. *Id.* at 13 fig. 3 (emphasis added). And notwithstanding its conclusion, NTP finds that "the body of evidence for human studies provides low confidence for PFOA or PFOS associations with infectious disease." *Id.* at 31-32.

2. In your testimony, you referenced studies of 3M workers who had occupational exposure to PFAS. How should the results of those studies inform policymaking concerning PFAS?

Policymaking should be informed by and based on an evaluation of the full breadth and depth of scientific evidence on the potential health effects of PFAS exposure. Some of the most important scientific studies in this regard come from the many years of occupational health research of 3M and DuPont workers exposed to PFOA or PFOS in the course of their job duties, including exposure at levels substantially higher than would be encountered in the environment by the general population, either today or historically. While certain isolated studies have suggested an association between the higher exposure to PFAS in that population and certain health outcomes, those associations have not tended to be replicated in subsequent study and with more robust analysis. And taken together, the worker health studies do not indicate adverse health effects caused by the elevated exposures to PFAS.

3. In your testimony, you stated that 3M announced its phase out of PFOS and PFOA in 2000. Could you provide us with more information about how that decision was implemented?

3M is committed to proactive stewardship of its products and acted in that spirit with the benefit of advances in sampling and detection technology and additional information regarding the prevalence of PFOA and PFOS in the environment and in humans. 3M also understood that those compounds were persistent and could bioaccumulate in the environment and in the human body over time as exposure continued. Although the science had not—and still has not—shown that exposure to PFOA and PFOS at the levels 3M was finding causes adverse health effects in humans, 3M made the decision voluntarily to stop using and manufacturing PFOA and PFOS. Once 3M made that decision, it worked with the Clinton Administration EPA to announce the voluntary phase-out of those compounds in 2000. 3M phased PFOA and PFOS out of most of its commercial products by 2002 and, with the introduction of ADONA in 2008, stopped all use of PFOA. EPA applauded those measures, saying, "3M deserves great credit for identifying this problem and coming forward voluntarily." Environmental Protection Agency, Press Release, "EPA and 3M Announce Phase Out of PFOS" (May 16, 2000).

4. The regulatory requirements around the production and use of chemicals in commerce have evolved substantially over time. How does that relate to 3M's production and use of PFAS chemicals?

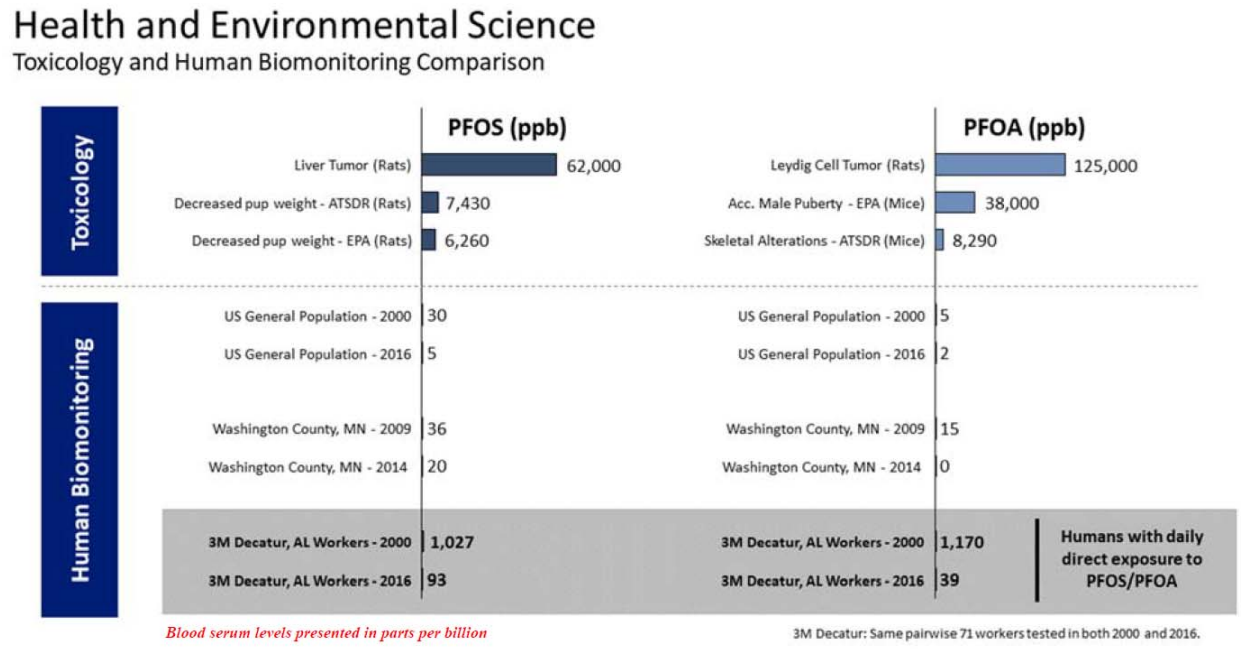
Requirements for the manufacture, testing, marketing, use, and disposal of chemicals have developed over time as our collective knowledge of the nature and potential effects associated with given chemical compounds evolves. Congress has played a key role in that development through enactment of environmental statutes like the Toxic Substances Control Act (TSCA), Emergency Planning and Community Right to Know Act, and Resource Conservation and Recovery Act, among others. Federal agencies, including in particular the EPA, use those and other statutory authorities, coupled with sound science, in the deliberative rulemaking process. 3M works with regulators to contribute to the continuing evolution of scientific understanding to inform the regulatory process. When it comes to PFAS, this is most evident in the decision to phase out the use of PFOA and PFOS. (*See* Response to Rouda 4.a.; Response to Comer 3.) 3M also provides EPA with its studies of PFAS under the TSCA Section 8(e) reporting program. And since the phase out of PFOA and PFOS, newly designed alternative PFAS compounds have gone through agency review and testing, as EPA requires.

5. Your testimony suggested that there are significant differences in exposure levels between animal toxicology studies and the level of PFAS generally found in the environment. Could you provide us with additional information on that?

Toxicology studies in laboratory animals are generally designed to administer increasing doses of a given substance until a response is observed. The result of this is that the doses—including those that may elicit a response—in a toxicology study are often higher than any dose that a human or other organism might expect to receive as a result of environmental exposure. In the case of PFOA or PFOS, the doses that elicit certain responses in laboratory animals are typically higher than what a human or other organism would face based on historic and current levels in the environment. As a result, those laboratory animal subjects have concentrations of

the particular PFAS up to tens of thousands of times higher than what has been found in the general human population.

The chart included in my testimony provided some examples of the concentrations of PFOA and PFOS found in laboratory animal subjects and the concentration in the general population. The rats in the studies that identified increased incidence of Leydig cell tumors, for example, had PFOA serum levels of 125,000 parts per billion. PFOA levels in the general population, by comparison, are approximately 2 parts per billion, as of 2016, and were approximately 5 parts per billion in 2000, at the time the phase out began. The chart from my testimony, which includes other examples for both PFOA and PFOS is reproduced below:



6. In your testimony, you discussed five commitments that 3M was making to address PFAS. Please tell us more about them.

We are continuing to make progress on the five commitments I made in my testimony:

1. *Continue ongoing remediation at 3M manufacturing sites*. 3M is working with federal, state, and local authorities at sites where we produced or disposed of PFAS as part of our manufacturing processes. This is an important responsibility we feel we have to the communities where we live and operate.

2. *Ensure appropriate disposal of Aqueous Film Forming Foam (AFFF) containing PFAS.* Some of 3M's former customers may still have supply of 3M-manufactured, PFAS-containing AFFF. We are working with those customers to help ensure that unused AFFF is properly handled. In some cases, this means 3M will work with customers to take back the unused product for disposal.

3. *Support nationwide science-based regulation of PFAS*. 3M remains committed to working with regulators to advance science-based regulation at the national level, including in particular the priorities identified in the EPA's PFAS Action Plan, such as evaluation of whether to set a Maximum Contaminant Level for PFOA, PFOS, and other PFAS under the Safe Drinking Water Act. 3M continues to conduct, fund, and support studies involving PFOA and PFOS to increase our scientific knowledge of these compounds, as it has for many years.

4. *Establish a clearinghouse for best practices on detection, measurement, and remediation*. 3M is moving forward with the plans announced in my testimony in September to establish a clearinghouse to share best practices and technology for detecting, measuring, and, where appropriate, remediating PFAS. We are in the process of collecting past studies and best practices for that purpose.

5. *Support coordinated research on PFAS*. 3M is working to advance efforts to enlist a respected, established, and independent scientific body to conduct a comprehensive review of the existing science on PFAS, inform the public of its findings, and set an agenda for further research to address further questions. In addition, 3M continues to conduct and support various ongoing research related to PFAS.