

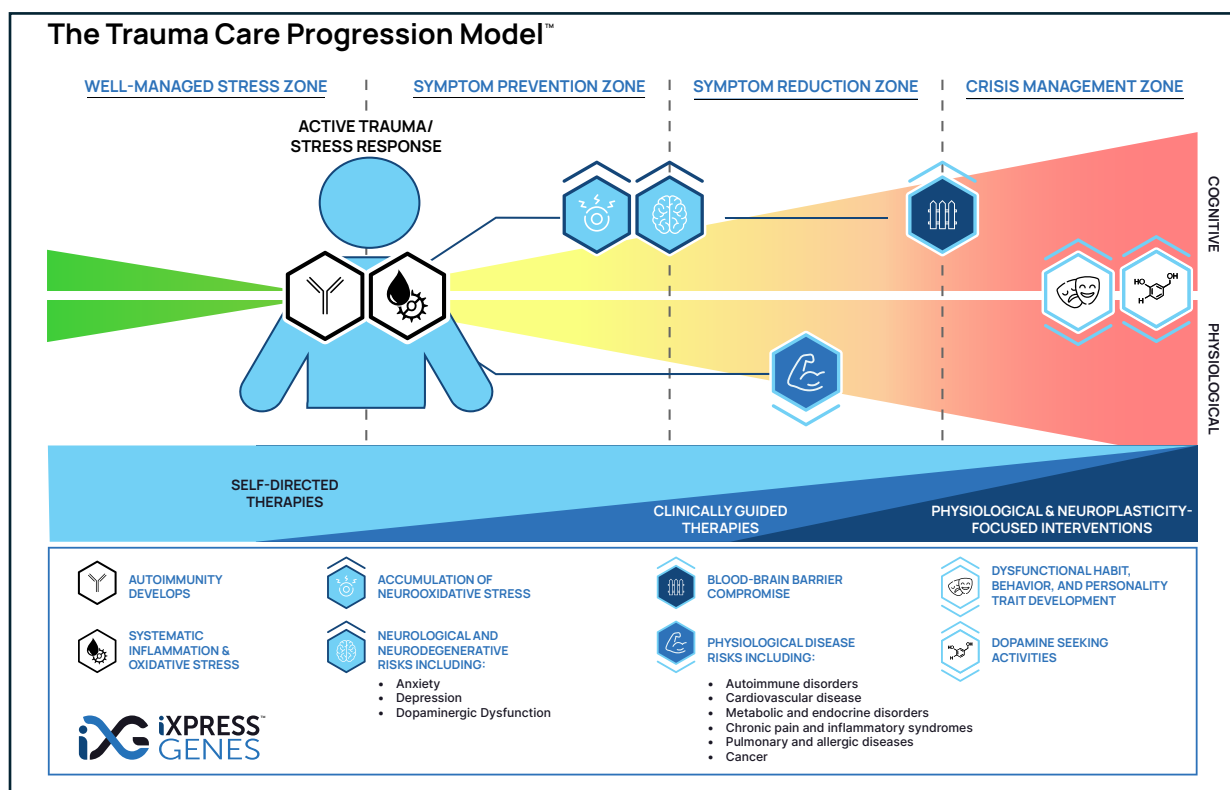
Trauma Autoimmune Indicator™ Test: A Peripheral Biomarker-Based Assessment of Chronic Stress and Neurocognitive Risk

Introduction

Chronic psychological trauma and unresolved stress are increasingly recognized as key contributors to systemic inflammation, oxidative stress, and long-term neurological dysfunction. At \$225 retail, the Trauma Autoimmune Indicator™ (TAI) test offers a novel, low-burden, precise, scalable, and low-cost method for assessing an individual's biological response to chronic stress using a panel of validated blood-based expressed gene biomarkers. This approach identifies early indicators of unresolved systemic inflammation and blood-brain barrier compromise— a state in which the brain's protective barrier becomes abnormally permeable, allowing potentially harmful substances to enter neural tissue. By identifying these biological changes before the onset of overt disease, the TAI™ test supports proactive health interventions aimed at preserving cognitive function and overall well-being. Integrating advances from immunology, neurobiology, and psycho-neuroendocrinology, the TAI™ test translates decades of peer-reviewed research into a practical tool for preventive medicine and brain health optimization.

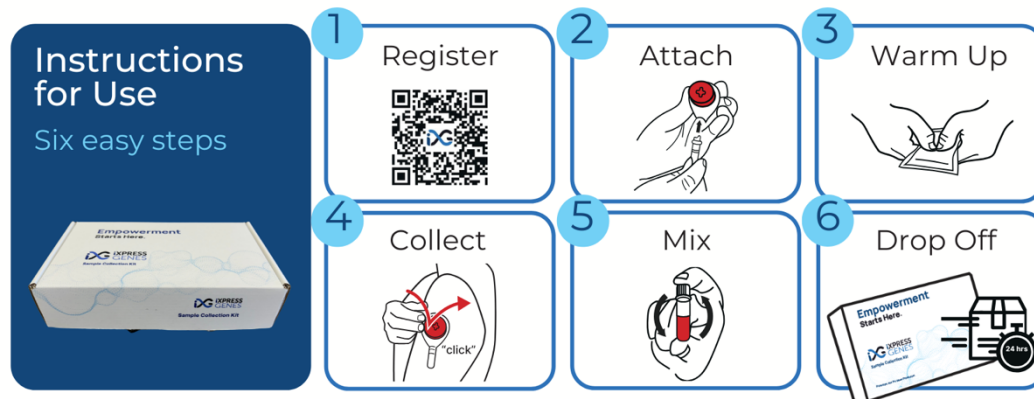
TAI™ Testing: The Trauma Care Progression Model™

The TAI™ test supports a wholistic model along a trauma continuum that is being developed by iXpressGenes to help provide global clinical context of trauma-induced disease and the underlying physiology it detects.



Blood Collection

The TAI™ test requires a standard peripheral blood draw, which can be performed in-clinic or at home using a validated self-collection kit. For clinical settings with on-site phlebotomy, venipuncture samples are collected in EDTA tubes. Alternatively, for simplicity, remote testing, or self-directed collection, the Tasso+ Kit or Red Drop device we employ enables patients to collect a capillary blood sample from the upper arm in a user-friendly, sterile device. This method supports decentralized testing models for greater accessibility. All collected samples are shipped or collected by courier and processed in the iXG CLIA-approved laboratory located in Huntsville, Alabama.



Explanation of Results

Analytes Evaluated: IL6, TNF- α , IL-1 β , IL-8, MCP-1, CRP, IL-10, IFN- γ , IL-17, CD163, NOS2, TGF- β , COL1A1, HMOX1, COX-2, NLRP3, Caspase-1, Cytochrome C, and LDH

In addition to detailed test results that can be used for clinicians to conduct individualized research for their patients, the TAI™ test delivers a simplified discussion falling in one of four categories which compliment the Trauma Care Progression Model™ to aid in interpretation and clinical action:

- **Non-inflammatory** Neither systemic nor neuroinflammatory markers are elevated. Immune and barrier functions appear well-regulated. This seems to represent a balanced physiological state. There is no evidence of chronic stress physiology or neurovascular compromise. Preventive strategies are recommended to maintain this resilience.
- **Chronic Stress and Inflammation Present** Systemic inflammation and oxidative stress present, but no compromise blood–brain barrier (BBB) disruption appear to be present. Literature reports have associated similar profiles with unresolved psychological or physiological stress manifesting through elevated pro-inflammatory and oxidative markers. The immune system is persistently activated, which, if unaddressed, may predispose to future neuroinflammatory risk and lead to cognitive dysfunction such as anxiety and depression.
- **Blood–Brain Barrier (BBB) Compromise** Biomarkers are present consistent with systemic inflammatory load and potential blood–brain barrier disruption. Literature reports have associated similar profiles with historical trauma, infection, or injury. Although the immune system may not currently be in a complete pro-inflammatory state, the barrier between brain and blood is impaired, potentially allowing immune or toxic insults to enter the central nervous system.

- **Chronic Stress, Inflammation, and BBB Compromise** Biomarkers consistent with both chronic systemic inflammation and potential blood–brain barrier (BBB) permeability.

Trends over time can inform trajectory and treatment response. A shift towards a non-inflammatory state supports therapeutic success; conversely while rising profiles may flag imminent relapse or unresolved biological trauma, even in the absence of self-reported symptoms.

Potential Use Cases

The TAI™ test is designed to be applied across a range of clinical environments, enabling early detection, tailored treatment, and preventive interventions:

- **High Stress Occupations:** Occupational health providers and clinicians working with veterans, active-duty military, or public safety professionals can utilize the TAI™ test for proactive identification of unresolved physiological stress before the onset of functional decline or psychiatric decompensation. Elevated inflammatory markers can guide early interventions such as structured rest, behavioral health referral, or anti-inflammatory treatments to preserve performance and long-term cognitive resilience.
- **Family Medicine and Primary Care:** In routine annual checkups, TAI™ serves as a screening tool for identifying patients with subclinical inflammation who may be at elevated risk for stress-related comorbidities such as cardiovascular disease, autoimmune disorders, or neurodegenerative conditions. Its use in asymptomatic individuals offers a proactive approach to lifestyle or pharmacological interventions before disease manifests.
- **Psychiatrists and Trauma Therapists:** In behavioral health settings, the TAI™ test provides an objective physiological measure to complement subjective symptom reporting. Elevated biomarkers can validate trauma exposure in patients with PTSD, generalized anxiety, or major depression. This data can help guide pharmacotherapy choices, monitor inflammation-related treatment resistance, and provide reassurance to patients that their symptoms have a measurable biological basis.
- **Self-Insured Employers and Occupational Health Clinics:** TAI™ can be deployed in workplace wellness programs to detect chronic stress in high-risk roles or demanding environments. Early identification of biologically stressed employees enables targeted wellness interventions that improve productivity, reduce burnout, and lower downstream healthcare costs by mitigating inflammation-driven disease before it develops.
- **Monitoring Treatment Efficacy:** For mental health providers and integrative care teams, serial use of TAI™ can measure the biological impact of therapy. Effective interventions may reduce cytokine and oxidative stress markers even before subjective improvement is evident. This is particularly important in conditions like PTSD or depression where patients often experience a lag between biological recovery and symptom relief. TAI™ can inform adjustments to treatment plans based on objective data.
- **Relapse Prevention and Early Intervention:** For patients with a prior history of trauma or chronic stress, the TAI™ test offers a sensitive tool for monitoring recurrence. As these individuals are primed for exaggerated immune responses to renewed stress, TAI™ can detect inflammatory reactivation before cognitive or emotional deterioration is clinically apparent. This enables preemptive interventions—such as therapy intensification or pharmacologic support—to halt the trajectory toward relapse.

Supporting the Evolving Modalities of Trauma Care

In addition to evidence based nutritional supplementation to support recovery, there is a rapidly expanding field of trauma recovery modalities—from traditional cognitive therapies to emerging

neurophysiological interventions such as MDMA-assisted psychotherapy, ketamine infusions, hyperbaric oxygen therapy, and psychedelic-based treatments like psilocybin and ibogaine. While many of these approaches show promise, they often lack broad clinical acceptance due to limited objective validation outside of controlled trials. The TAI™ test offers a powerful tool to help bring these therapies into the mainstream by providing measurable and objective, biologically based markers of immune activation, oxidative stress, and blood–brain barrier integrity. By demonstrating physiological change in response to treatment—regardless of the modality—TAI can support evidence-based adoption, guide individualized care, and help clinicians, researchers, and innovators validate the effectiveness of new and integrative approaches in a way that resonates across disciplines.

Conclusion

As the clinical landscape of trauma recovery continues to evolve, the need for objective, biologically grounded data has never been greater. The TAI™ test offers healthcare professionals a practical, science-based tool to assess the physiological impact of unresolved stress—providing a clearer picture of the body’s inflammatory and neuroimmune status when patient symptoms may be vague, delayed, or absent altogether. By translating complex immune and oxidative markers into a clear interpretive framework, TAI™ supports personalized treatment decisions grounded in measurable biology rather than subjective reporting.

Notably, the TAI™ test does not prescribe any specific therapeutic path, but rather serves as a diagnostic compass—helping clinicians identify when more support may be needed, when treatment is taking effect, or when early intervention could prevent relapse. Whether you practice mindfulness-based interventions, trauma-informed cognitive therapies, or are exploring the role of physiologically focused and neuroplasticity-oriented modalities such as MDMA-, psilocybin-, ketamine-, or ibogaine-assisted psychotherapy, the TAI™ test offers a unifying biomarker-based foundation to inform care across approaches. This includes integrative strategies such as targeted nutritional interventions, which modulate inflammation and oxidative stress and support brain health through diet.

This test complements—not replaces—clinical judgment, therapeutic alliance, and patient-reported outcomes. As the field of trauma recovery moves toward a more integrated biopsychosocial model, tools like TAI help bridge disciplines by contributing objective, actionable insights into an otherwise invisible biological burden.

At its core, the TAI test is a clinical tool to support evidence-based, individualized care—and a step toward transforming trauma care forever.

References and Recommended Reading

Bonney, S., Dennison, U., Fuller, A., et al. (2019). IFN- γ drives cerebral endothelial activation and breakdown of BBB tight junctions in inflammation. *Journal of Neuroimmunology*, 328, 35–42.

<https://www.sciencedirect.com/science/article/pii/S0165572819300114>

Chen, Y., Li, G., & Huang, Y. (2019). Tumor necrosis factor- α mediates blood–brain barrier dysfunction induced by microglial activation. *Journal of Pharmacological Sciences*, 140(3), 249–257.

<https://www.sciencedirect.com/science/article/pii/S1347861319300896>

Choi, A. M. K., & Alam, J. (1996). Heme oxygenase-1: Function, regulation, and implication in response to oxidative stress. *American Journal of Physiology-Lung Cellular and Molecular Physiology*, 270(6), L800–L821.

<https://journals.physiology.org/doi/full/10.1152/ajplung.1996.270.6.L800>

- Danese, A., Pariante, C. M., Caspi, A., Taylor, A., & Poulton, R. (2007). Childhood maltreatment predicts adult inflammation in a life-course study. *Proceedings of the National Academy of Sciences*, 104(4), 1319–1324. <https://www.pnas.org/content/104/4/1319>
- Dantzer, R., Cohen, S., Russo, S. J., & Dinan, T. G. (2018). Resilience and immunity. *Brain, Behavior, and Immunity*, 74, 28–42. <https://www.sciencedirect.com/science/article/pii/S0889159117304397>
- Hauptmann, J., Johann, S., Marini, F., Kitic, M., Nieke, J., & K ry, P. (2020). Interleukin-1 promotes autoimmune neuroinflammation by suppressing endothelial HO-1 at the blood–brain barrier. *Acta Neuropathologica*, 140(4), 549–567. <https://link.springer.com/article/10.1007/s00401-020-02193-3>
- Huo, K., Sun, Y., Liu, D., & Liu, Y. (2020). Stress-induced nitric oxide synthase and its effect on blood-brain barrier permeability. *Cell Stress and Chaperones*, 25(5), 815–823. <https://link.springer.com/article/10.1007/s12192-020-01100-z>
- Kebir, H., Kreamborg, K., Ifergan, I., Dodelet-Devillers, A., Cayrol, R., Bernard, M., ... & Prat, A. (2007). Human TH17 lymphocytes promote blood–brain barrier disruption and central nervous system inflammation. *Nature Medicine*, 13(10), 1173–1175. <https://www.nature.com/articles/nm1651>
- Liu, Y., Ho, R. C., & Mak, A. (2017). Interleukin (IL)-6, tumour necrosis factor alpha (TNF- ) and soluble interleukin-2 receptors (sIL-2R) are elevated in patients with major depressive disorder: A meta-analysis and meta-regression. *Journal of Affective Disorders*, 190, 264–274. <https://www.sciencedirect.com/science/article/abs/pii/S0165032715003399>
- Madrigal, J. L. M., Hurtado, O., Moro, M. A., Lizasoain, I., Lorenzo, P., Castrillo, A., & Leza, J. C. (2003). Induction of cyclooxygenase-2 accounts for restraint stress-induced oxidative status in rat brain. *Neuropsychopharmacology*, 28(9), 1579–1588. <https://www.nature.com/articles/1300206>
- Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin*, 137(6), 959–997. <https://psycnet.apa.org/fulltext/2011-17585-001.html>
- Montalvo-Ortiz, J. L., Wang, F., Holley, C. L., et al. (2022). Central and peripheral immune dysregulation in posttraumatic stress disorder: Convergent multi-omics evidence. *Journal of Clinical Medicine*, 11(10), 2747. <https://www.mdpi.com/2077-0383/11/10/2747>
- Munjiza Jovanović, A., Ivković, M., Brkić, M., et al. (2021). Childhood maltreatment correlates with higher concentration of TGF-  in adults with major depression. *Psychiatry Research*, 301, 113987. <https://www.sciencedirect.com/science/article/abs/pii/S0165178121001184>
- Passos, I. C., Vasconcelos-Moreno, M. P., & Costa, L. G. (2015). Inflammatory markers in post-traumatic stress disorder: A systematic review, meta-analysis, and meta-regression. *Molecular Psychiatry*, 20(12), 1480–1488. <https://www.nature.com/articles/mp2015133>
- Pieper, C., Pieloch, P., & Galla, H. J. (2013). Pericytes support neutrophil transmigration across the blood–brain barrier via IL-8. *Brain Research*, 1524, 1–11. <https://www.sciencedirect.com/science/article/abs/pii/S0006899313012286>
- Walsh, J. G., Muruve, D. A., & Power, C. (2014). Inflammasomes in the CNS. *Nature Reviews Neuroscience*, 15(2), 84–97. <https://www.nature.com/articles/nrn3638>
- Yang, J., Chu, Y., Yang, X., Gao, Y., Li, X. M., & Chen, H. (2020). Peripheral injury causes blood–brain barrier disruption and cognitive impairment via IL-6 and IL-1 . *Journal of Neuroinflammation*, 17(1), 154. <https://jneuroinflammation.biomedcentral.com/articles/10.1186/s12974-020-01811-y>
- Yao, Y., & Tsirka, S. E. (2014). The roles of microglia and monocyte chemoattractant protein-1 in neuroinflammation. *Cellular and Molecular Life Sciences*, 71(4), 683–697. <https://link.springer.com/article/10.1007/s00018-013-1388-3>
- Yin, D., Wang, X., & Gao, Q. (2013). Essential role of IL-10/STAT3 in chronic stress-induced immune suppression. *Brain, Behavior, and Immunity*, 32, 86–95. <https://www.sciencedirect.com/science/article/pii/S0889159113000107>