Environmental Inflammation and Tic Disorders:

A Review of Neuroimmune Pathways in Tourette Syndrome

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Author's Note

This educational publication explores emerging scientific literature regarding neuroinflammation, immune dysfunction, and neurological symptom presentation in Tourette syndrome and related tic disorders. The purpose of this paper is to encourage awareness, discussion, and continued scientific exploration regarding neuroimmune mechanisms and potential environmental contributors to neurological symptoms. Current research remains evolving, and this publication is not intended to diagnose, treat, cure, or prevent any medical condition. Individuals experiencing medical concerns should consult qualified healthcare professionals.

ABSTRACT

While Tourette syndrome has historically been associated with dopamine dysregulation, emerging evidence involving neuroinflammation, immune activation, and environmental inflammatory exposures suggests that tic disorders may involve broader neuroimmune mechanisms than previously recognized.

Recent studies have demonstrated associations between inflammatory cytokines, microglial activation, blood-brain barrier dysfunction, and altered dopaminergic signaling in individuals with TS. Parallel findings have also been observed in environmental exposure research involving water-damaged buildings, mold exposure, and mycotoxin-related neuroinflammation. This paper reviews current literature regarding neuroimmune pathways involved in TS and explores whether environmental inflammatory exposures may represent overlooked contributing or exacerbating factors in susceptible individuals. While direct causal evidence remains limited, the overlap between inflammatory mechanisms observed in TS and environmentally induced neuroimmune dysfunction warrants further scientific investigation.

Tourette syndrome (TS) is a neurodevelopmental disorder characterized by persistent motor and vocal tics. Historically, research has focused heavily on abnormalities within dopaminergic signaling pathways as the primary mechanism underlying tic disorders. However, growing evidence suggests that immune dysregulation, neuroinflammation, and environmental factors may also contribute to the development or exacerbation of symptoms.

Recent studies have identified elevated inflammatory cytokines, microglial activation, autoimmune responses, and blood-brain barrier disruption in some individuals with TS (Wu et

al., 2025). Additionally, infection-triggered syndromes such as Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections (PANDAS) further support the possibility that immune-mediated inflammation may influence tic expression.

At the same time, literature involving exposure to water-damaged buildings and mold-associated illness has identified similar neuroinflammatory pathways, including cytokine activation, oxidative stress, neuroimmune dysfunction, and neurological symptom presentation. Although current evidence does not establish a direct causal relationship between environmental mold exposure and Tourette syndrome, the overlap between these inflammatory mechanisms raises important questions regarding environmental contributors to tic disorders in susceptible individuals.

Neuroinflammation in Tourette Syndrome

Recent literature increasingly supports the role of neuroinflammation in TS pathogenesis. Wu et al. (2025) describe how infections, allergens, and inflammatory processes may activate peripheral immune responses, resulting in elevated inflammatory cytokines capable of crossing the blood-brain barrier. Once within the central nervous system, these cytokines may contribute to neuroinflammation and abnormal activation of microglial cells.

Microglia serve as the immune defense cells of the brain. Under chronic inflammatory conditions, however, excessive microglial activation may disrupt neuronal signaling and contribute to injury within dopaminergic pathways associated with motor regulation. Wu et al. (2025) further note that inflammatory signaling pathways, including JAK2/STAT3 and NF-κB

activation, may impair dopaminergic neurons within the striatum, potentially contributing to excessive dopamine release and tic manifestation.

The relationship between inflammation and dopamine dysregulation is significant because the basal ganglia and striatal circuits involved in movement control have long been implicated in TS. This evolving neuroimmune model expands traditional understandings of tic disorders beyond purely neurotransmitter-based explanations.

You can even visually reference the dopamine pathway with this central formula relationship:

Neuroinflammation → Microglial Activation → Dopamine Dysregulation → Tic Expression

Infection-Triggered Tic Disorders and Autoimmunity

Research involving infection-triggered neuropsychiatric syndromes further strengthens the argument for immune involvement in tic disorders. PANDAS describes a subgroup of children who experience sudden-onset obsessive-compulsive symptoms and tics following streptococcal infection. Proposed mechanisms include the production of anti-neuronal antibodies that mistakenly target brain tissue involved in movement regulation.

Wu et al. (2025) explain that anti-neuronal antibodies may interact with neuronal surface antigens and activate microglia through calcium/calmodulin-dependent protein kinase II (CAMK II) pathways, resulting in altered dopaminergic function. These findings suggest that immune activation alone may significantly influence neurological symptom presentation in vulnerable individuals.

Environmental Inflammatory Exposures and Neuroimmune Dysfunction

Environmental exposure research has identified multiple biological mechanisms capable of producing neuroinflammatory responses similar to those discussed in TS literature. Occupants of water-damaged buildings have reported neurological and neuropsychiatric symptoms associated with chronic exposure to microbial contaminants, mycotoxins, endotoxins, and inflammatory volatile organic compounds.

Studies involving mold and mycotoxin exposure have demonstrated elevated inflammatory cytokines, oxidative stress, immune dysregulation, and neurological dysfunction. Experimental animal models have additionally shown that mold inhalation may activate innate immune responses within the brain, impair neurogenesis, and alter behavioral functioning.

Although direct evidence linking mold exposure specifically to Tourette syndrome remains limited, the mechanistic overlap is notable. Both TS neuroinflammation models and environmental exposure research involve:

- cytokine elevation
- microglial activation
- blood-brain barrier disruption
- oxidative stress
- dopaminergic pathway impairment

These shared mechanisms suggest that environmental inflammatory exposures may represent potential contributing or exacerbating factors in tic disorders among susceptible individuals.

Limitations of Current Research

Despite growing interest in neuroimmune models of TS, several limitations remain. Current research has not established definitive causation between environmental mold exposure and Tourette syndrome. Many studies remain observational, mechanistic, or animal-based rather than large-scale human clinical trials.

Additionally, TS is likely multifactorial, involving genetic, neurological, immunological, and environmental components that interact differently across individuals. Environmental inflammatory exposures should therefore be considered possible contributing factors rather than singular causes.

Future research investigating environmental exposure histories, inflammatory biomarkers, and neuroimmune dysfunction in tic disorder populations may help clarify these relationships.

Conclusion

Emerging evidence suggests that neuroinflammation and immune dysregulation may play important roles in the development and expression of Tourette syndrome and tic disorders. Research involving inflammatory cytokines, microglial activation, autoimmune responses, and dopaminergic disruption continues to expand understanding of TS beyond traditional neurotransmitter models.

Simultaneously, environmental exposure literature involving water-damaged buildings and mold-associated illness has identified overlapping neuroimmune mechanisms capable of affecting neurological function. While current evidence does not prove that environmental mold

exposure causes Tourette syndrome, the biological similarities between these inflammatory pathways warrant further investigation.

A broader neuroimmune framework may ultimately improve understanding of tic disorders and encourage more comprehensive approaches to neurological and environmental health assessment.

Reference

Wu, X., Hao, J., Jiang, K., Wu, M., Zhao, X., & Zhang, X. (2025). Neuroinflammation and pathways that contribute to Tourette syndrome. Italian Journal of Pediatrics, 51(1), 63. https://doi.org/10.1186/s13052-025-01874-3