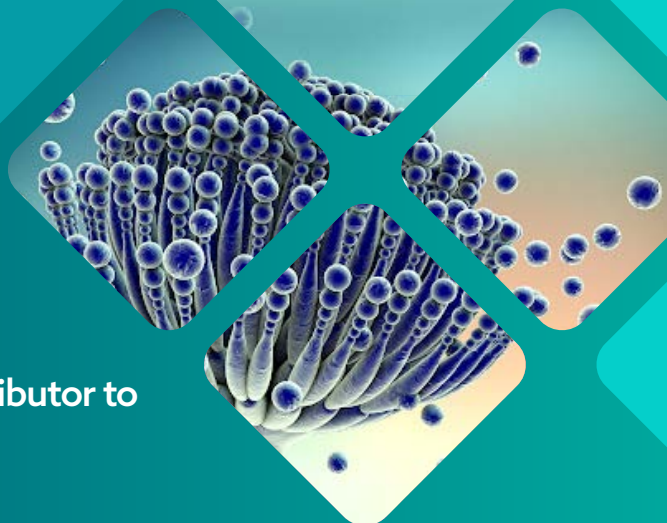


Autism Spectrum Disorder (ASD) & Special Needs

Assessing Mycotoxin Burden as a Modifiable Contributor to Symptom Complexity in ASD



ASD rarely comes alone. Many individuals with ASD also experience gastrointestinal problems, attention-deficit/hyperactivity disorder (ADHD), or other psychiatric conditions—and these are the same areas that mycotoxin exposure can affect. [1] A measurable mycotoxin burden may worsen symptoms in people who already have biological vulnerabilities like poor detoxification, leaky gut, or inflammation.^[5,7,12] The MycoTOX Profile can help identify these environmental contributors, giving practitioners a more complete picture of what's driving the clinical presentation.^[16]



When Might Providers Consider the MycoTOX Profile?

GI Distress with Unclear Etiology: Chronic constipation, diarrhea, bloating, or dysbiosis unresponsive to standard interventions may reflect mycotoxin-driven disruption of intestinal tight junction proteins and gut microbiome composition.^[1,2]

Behavioral Regression or Symptom Plateau: Sudden worsening of core ASD symptoms, increased irritability, or loss of previously acquired skills without a clear clinical explanation may warrant assessment of environmental mycotoxin burden as a contributing factor.^[5,12]

Attention, Mood & Dopamine-Related Symptoms: Co-occurring ADHD-like patterns, anxiety, depression, or reward-processing deficits may have mechanistic overlap with ochratoxin A (OTA)–associated dopamine depletion documented in animal models.^[3,4,11]

Sleep Disruption Unresponsive to Intervention: Persistent difficulties with sleep onset or maintenance—common in 50–80% of ASD children—may carry a neuroinflammatory component that mycotoxin burden assessment can help contextualize.^[1,5]

Known or Suspected Water-Damaged Building Exposure: Residence, school, or childcare settings with visible mold or prior water damage represent a primary exposure pathway for clinically relevant mycotoxin classes including OTA and gliotoxin.^[6,12]

Restricted or Grain-Heavy Dietary Patterns: Sensory-driven ASD feeding patterns that favor grains, breads, pastas, and crackers^[14] may significantly increase dietary mycotoxin load, as these foods carry disproportionately higher contamination risk.^[15]

What the MycoTOX Profile Measures

Analyte Class	Everyday Exposure Sources	ASD & Special Needs Relevance
Ochratoxin A (OTA)	Grains, coffee, dried fruits, wine, beer, processed cereals ^[2,6]	Most studied mycotoxin in ASD context; associated with gut permeability disruption, dopamine depletion, hippocampal injury, and dysregulation of microRNA-132—a pathway implicated in ASD-related gene regulation (MeCP2, PTEN). ^[3,6,10]

What the MycoTOX Profile Measures (Cont)

Analyte Class	Everyday Exposure Sources	ASD & Special Needs Relevance
Aflatoxins (AFM1)	Corn, peanuts, tree nuts, dried figs, spieces, grain-based foods ^[4]	Can cross the blood-brain barrier; associated with disruption of serotonergic and dopaminergic systems, oxidative stress, and pro-inflammatory cytokine activation in preclinical models. ^[4,5]
Trichothecenes	Wheat, barley, oats, rye—staples in many restrictive ASD diets ^[14]	Inhibit ribosomal protein synthesis; promote intestinal epithelial injury, immune dysregulation, and neuroinflammatory cytokine cascades (TNF- α , IL-1 α). ^[5,7]
Zearalenone (ZEA)	Corn, wheat, soy, fermented grain products ^[14]	Estrogenic mycotoxin with endocrine-disrupting properties; associated with immune modulation and oxidative stress in neural tissue in animal models. ^[5]
Gliotoxin (and other important mycotoxins)	Indor air from water-damaged buildings; Aspergillus and Candida species ^[12]	Potent immunosuppressant associated with mitochondrial dysfunction and oxidative stress; clinically relevant when indoor environmental mold exposure is suspected. ^[5,8]

Clinical Applications & Evidence Context

Condition	Clinical Encapsulation
GI Disorders (Dysbiosis, Constipation, Diarrhea)	OTA disrupts intestinal tight junction proteins and promotes dysbiosis; given that up to 85% of ASD individuals have GI comorbidities, mycotoxin-driven gut permeability may be a clinically relevant and potentially modifiable contributing mechanism. ^[1,2,6]
ADHD / Attention Deficits	OTA has been shown to deplete dopamine and impair hippocampal function in animal models, offering a plausible mechanistic basis for the attention deficits that co-occur in ~50–70% of ASD individuals. ^[3,11]
Anxiety & Mood Dysregulation	Aflatoxin B1 disrupts serotonergic and dopaminergic signaling in preclinical models; OTA-associated dopamine depletion has been proposed as a potentiating factor for anxiety and mood symptoms common in ASD. ^[4,11]
Sleep Disorders	Mycotoxin-associated neuroinflammation and mitochondrial dysfunction may disrupt CNS signaling pathways underlying sleep architecture, relevant to the 50–80% of ASD children with clinically significant sleep difficulties. ^[1,5]
Epilepsy / Seizure Activity	Mycotoxin-driven disruption of the blood-brain barrier and glial activation are documented in preclinical studies ^[5] ; since epilepsy affects an estimated 25–40% of individuals with ASD ^[1] , environmental burden assessment may be a component of the broader clinical picture in patients with treatment-resistant or atypical seizure presentations — though a direct mechanistic link between mycotoxin exposure and seizure threshold has not been established in the literature.
Neuroinflammation & Immune Dysregulation	Mycotoxins generate reactive oxygen species, deplete glutathione, and activate microglia to secrete TNF- α and interleukins—pathways that overlap substantially with the near-universal neuroinflammatory underpinning described in ASD research. ^[5,7]

How the MycoTOX Profile Supports Clinical Reasoning

Assesses Modifiable Environmental Burden:

Quantifies specific urinary mycotoxin classes, providing actionable data to support discussions around environmental remediation, dietary modification, and targeted supportive interventions.^[6,17]

Maps to ASD-Relevant Molecular Pathways:

Ongoing research notes that OTA has been associated with dysregulation of microRNA-132—a pathway directly implicated in regulation of ASD-associated genes MeCP2 and PTEN—representing one of the more precise molecular threads connecting mycotoxin burden to ASD biology.^[10]

Supports Integrative Test Pairing:

MycoTOX results can contextualize findings from complementary testing such as the Organic Acids Test (OAT) for mitochondrial dysfunction markers, gut dysbiosis signatures, and detox pathway capacity—building a more complete biological burden picture.^[5,8]

How to Order

To order the MycoTOX Profile test, contact your Mosaic Diagnostics representative or visit [www.mosaicdx.com]. Specimen collection kits and requisition forms are available through your account portal. For clinical support or interpretation questions, contact our clinical education team.

This document is intended for licensed healthcare professionals and is for informational and educational purposes only. The test results are not intended to diagnose, treat, cure, or prevent disease. All clinical interpretation should be done by a qualified healthcare professional in the context of the individual patient. Statements regarding associations between health symptoms and conditions do not constitute diagnostic claims.

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