

Autism Spectrum Disorder (ASD) & Special Needs

Revealing the environmental body burden that may help contextualize neurodevelopmental vulnerability.



ASD is increasingly understood as a complex gene–environment condition in which individual detoxification capacity plays a central role.^[1] Many children with ASD carry variants in UGT glucuronidation enzymes, CYP450 isoforms, and paraoxonase genes that reduce their ability to clear environmental chemicals, leaving them disproportionately vulnerable to body burden accumulation.^[2,4] TOXDetect measures urinary metabolites of pesticides, plasticizers, industrial chemicals, and endocrine disruptors whose mechanistic pathways—dopaminergic disruption, thyroid axis interference, neuroinflammation, and oxidative stress—overlap substantially with recognized features of ASD neurobiology.^[2,3] By identifying elevated chemical exposures, TOXDetect may help clinicians contextualize neurodevelopmental presentations and support individualized, evidence-informed intervention strategies.^[4,15]



When Might Providers Consider the TOXDetect Profile?

Diagnosed or suspected ASD with environmental exposure history: Child or patient with confirmed or evolving ASD diagnosis and known proximity to agricultural areas, industrial sites, or high-use pesticide regions.^[5,6,7]

Unexplained behavioral regression or developmental plateau: Sudden or gradual loss of previously acquired skills, increased repetitive behaviors, or communication regression without clear medical etiology.^[2,4]

Sensory sensitivities, irritability, or sleep disruption co-occurring with neurodevelopmental concerns: Overlapping symptom clusters commonly reported alongside ASD that may reflect neurochemical dysregulation associated with toxicant exposure.^[3,5]

Family or personal history of chemical sensitivity: Maternal chemical intolerance – a marker of heightened physiological reactivity – has been associated with elevated rates of ASD and ADHD in offspring.^[17]

Co-occurring ADHD, anxiety, or mood dysregulation in a neurodevelopmental context: Phthalates and BPA have both been associated with inattentive and hyperactive-impulsive behaviors, particularly in children with co-occurring ASD.^[8,9,14]

Prenatal or early-childhood toxicant exposure suspected: Maternal organophosphate or perchlorate exposure during pregnancy has been epidemiologically linked to increased neurodevelopmental risk in offspring.^[6,7,12,13]

What the TOXDetect Profile Measures

Analyte Class	Everyday Exposure Sources	ASD & Special Needs Relevance
Organo-phosphate Pesticides (DEP)	Fruits and vegetables treated with chlorpyrifos, diazinon, or malathion; proximity to agricultural fields ^[6,7]	Prenatal exposure associated with up to 3.3-fold increased ASD risk; mechanism involves acetylcholinesterase inhibition disrupting cholinergic neurodevelopment ^[6,7]
Bisphenols (BPA, BPS)	Polycarbonate plastics, food can linings, thermal receipt paper, BPS-containing "BPA-free" products ^[8,9]	Children with ASD show ~10% reduced glucuronidation efficiency for BPA; associated with neuroinflammation and disruption of sex steroid signaling ^[8,9]
Atrazine Mercapturate (AM)	Drinking water and groundwater in Midwest agricultural regions; corn and sorghum crops ^[10,11]	Established dopaminergic neurotoxicant in animal models; associated with anxiety, social deficits, and hypothalamic-pituitary-gonadal axis disruption ^[10,11]
Perchlorate (PERC)	Contaminated groundwater, fireworks residue, rocket fuel byproducts, some leafy vegetables ^[12,13]	Competitively inhibits thyroid iodide uptake; maternal hypothyroidism linked to increased ASD/ADHD risk; EPA research confirms structural brain malformations in offspring ^[12,13]
Phthalates (MEHP, MEOHP)	Flexible PVC products, personal care products, food packaging, IV tubing, flooring ^[3,14]	Anti-androgenic endocrine disruption associated with inattentive/hyperactive ADHD behaviors, especially pronounced in children with co-occurring ASD ^[14]
Acrylamide (NAE)	High-temperature cooked starchy foods (french fries, chips, bread crust), cigarette smoke ^[15]	Impairs neuronal migration; causes oxidative stress, mitochondrial dysfunction, and disrupted BDNF/CREB signaling—pathways implicated in ASD neuropathology ^[15]
Parabens (Butylparaben)	Personal care products, cosmetics, pharmaceuticals, food preservatives ^[5]	Endocrine disruptors with estrogenic/androgenic activity; share hormonal disruption pathways relevant to neurodevelopment ^[5]
VOC Panel (Benzene, Styrene, Acrylonitrile)	Vehicle exhaust, industrial emissions, tobacco smoke, solvent-based products, plastics manufacturing ^[5]	Central nervous system (CNS) toxins metabolized via CYP450 and glutathione conjugation—pathways frequently impaired in ASD; elevated oxidative stress and neuroinflammatory burden ^[4,5]
HEMA (Ethylene Oxide / Vinyl Chloride)	Sterilized medical equipment, tobacco smoke, industrial chemical manufacturing ^[4,5]	International Agency for Research on Cancer (IARC)-classified carcinogens; detoxified via epoxide hydrolase and glutathione S-transferase—enzymes functionally impaired in some children with ASD ^[4,5]

Related Conditions

Condition	Clinical Encapsulation
Autism Spectrum Disorder (ASD)	Cumulative toxic body burden interacting with impaired detoxification genetics—particularly UGT, CYP450, and glutathione pathways—represents the prevailing framework for gene-environment interaction in ASD; no single toxicant is established as causal. ^[2,3,4,16]
ADHD & Attention Dysregulation	Early childhood phthalate and BPA exposures have been associated with inattentive and hyperactive-impulsive ADHD behaviors, with effects especially pronounced in children with co-occurring ASD. ^[8,9,14]

Related Conditions (Cont)

Condition	Clinical Encapsulation
Neurodevelopmental Delay	Prenatal organophosphate exposure has been epidemiologically associated with a 60% increased likelihood of ASD diagnosis when mothers reside within one mile of treated agricultural fields during gestation. ^[6,7]
Thyroid-Mediated Developmental Risk	Perchlorate competitively inhibits thyroid iodide uptake, and maternal hypothyroidism during pregnancy has been epidemiologically linked to increased ASD and ADHD risk in offspring. ^[12,13]
Dopaminergic & Social Behavior Dysregulation	Atrazine reduces striatal dopamine via vesicular dopamine uptake interference—a mechanism directly relevant to ASD neurobiology—and has been associated with anxiety and social deficit phenotypes in animal models. ^[10,11]
Mitochondrial & Oxidative Stress Burden	Acrylamide exposure has been associated with mitochondrial dysfunction, oxidative stress, and impaired BDNF/CREB signaling—biological mechanisms that converge with recognized neuropathological features of ASD. ^[15,5]

How the TOXDetect Profile Supports Clinical Reasoning

Supports Targeted Exposure Reduction:

Elevated analyte results can guide clinicians in prioritizing specific exposure source modifications—such as dietary changes, water filtration, or product swaps—tailored to the individual patient's burden profile.^[5,15]

Informs Individualized Detoxification Support:

Results may help support clinical reasoning around detoxification interventions, such as glutathione precursors for VOC/vinyl chloride burden, iodine sufficiency assessment alongside elevated perchlorate, or sauna/perspiration approaches for phthalate reduction.^[5,12]

Adds an Environmental Lens to Neurodevelopmental Workup:

TOXDetect complements genetic, metabolic, and functional assessments by providing actionable data on the environmental contributors that interact with neurodevelopmental genetic susceptibility.^[16,3]

How to Order

To order the TOXDetect 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.

References

1. Main PAE, Angley MT, O'Doherty CE, Thomas P, Fenech M. The potential role of the antioxidant and detoxification properties of glutathione in autism spectrum disorders: a systematic review and meta-analysis. *Nutr Metab (Lond)*. 2012;9(1):35.
2. Hallmayer J et al. Genetic heritability and shared environmental factors among twin pairs with autism. *Arch Gen Psychiatry*. 2011. <https://pmc.ncbi.nlm.nih.gov/articles/PMC3944636/>
3. New Patterns ABA. Environmental causes and risk factors of autism. <https://www.newpatternsaba.com/blog/environmental-causes-risk-factors-of-autism>
4. Rossignol DA, Genuis SJ, Frye RE. Environmental toxicants and autism spectrum disorders: a systematic review. *Transl Psychiatry*. 2014;4(2):e360. Published 2014 Feb 11. doi:10.1038/tp.2014.4
5. MosaicDX. TOXDetect Panel Clinical Report Reference. MDX_Report_TOXDetect_ENG.pdf
6. Duke Superfund. Organophosphate pesticides and autism. <https://sites.nicholas.duke.edu/superfund/organophosphate-pesticides-and-autism/>
7. Shelton JF et al. Neurodevelopmental disorders and prenatal residential proximity to agricultural pesticides. *BMJ*. 2019;364:l962.
8. Autism.org. New research points to alterations in BPA excretion pathway in children with autism/ADHD. <https://autism.org/new-research-points-to-alterations-in-bpa-excretion-pathway-in-children-with-autism-adhd/>
9. Rowan University. Researchers find BPA links to autism, ADHD. <https://today.rowan.edu/news/2023/09/researchers-find-bpa-links-to-autism-adhd.html>
10. Bardullas U et al. Chronic atrazine exposure causes disruption of the dopaminergic system. *Neurotoxicol Teratol*. 2011. <https://pubmed.ncbi.nlm.nih.gov/24561379/>
11. Hossain MM et al. Dopaminergic toxicity of atrazine. *PMC*. <https://pmc.ncbi.nlm.nih.gov/articles/PMC1853311/>
12. The Conversation. From fireworks to iodine and IQ: the perchlorate connection. <https://theconversation.com/from-fireworks-to-iodine-and-iq-the-perchlorate-connection-86595>
13. EPA. EPA research provides important information on health effects of perchlorate exposure. <https://www.epa.gov/sciencematters/epa-research-provides-important-information-health-effects-perchlorate-exposure>
14. ECHO Cohort. Early childhood phthalate exposures associated with ADHD behaviors in childhood and adolescence. <https://pubmed.ncbi.nlm.nih.gov/38692176/>
15. Motttram DS et al. Acrylamide neurotoxicity: mechanisms and developmental implications. *PMC*. <https://pmc.ncbi.nlm.nih.gov/articles/PMC9561965/>
16. Hallmayer J et al. Twin study implicating gene-environment interaction in ASD. *PubMed*. <https://pubmed.ncbi.nlm.nih.gov/28475976/>
17. Heilbrun LP, Palmer RF, Jaen CR, Svoboda MD, Perkins J, Miller CS. Maternal Chemical and Drug Intolerances: Potential Risk Factors for Autism and Attention Deficit Hyperactivity Disorder (ADHD). *J Am Board Fam Med*. 2015;28(4):461-470.

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