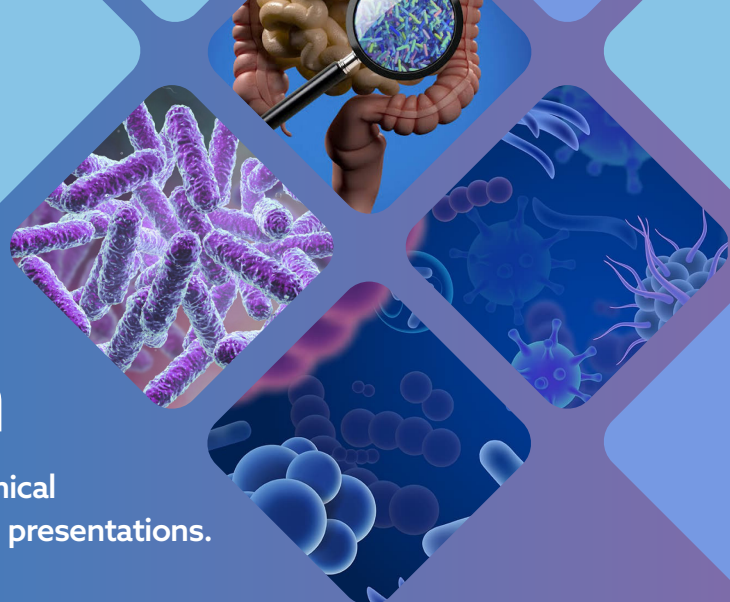


Neurologic & Mental Health

Illuminating the gut-brain connection to support clinical reasoning in cognitive, mood, and behavioral health presentations.



Many patients with brain fog, mood changes, or behavioral concerns also struggle with gut symptoms like bloating, irregular bowel habits, or food sensitivities.^[1,5,6] Research suggests the gut and brain communicate through a shared network involving gut bacteria, immune signals, and digestive byproducts that may influence how these symptoms feel and present.^[1,2] GI360 can help clinicians look at what's happening in the gut as part of a bigger clinical picture to add useful context when gut and brain symptoms overlap.^[1,3,4,6]



When Might Providers Consider the GI360 Profile

Cognitive Complaints + GI Overlap: Patient reports brain fog, memory difficulties, or poor concentration alongside constipation, bloating, or post-meal symptom changes.^[1,2,11]

Mood Dysregulation with Bowel Pattern Changes: Low mood, irritability, or anxiety that fluctuates with GI symptoms, dietary shifts, antibiotic use, or stress.^[1,3,4,6]

Autonomic Dysregulation with GI-related Features: Nausea, early satiety, palpitations, or stress-sensitive bowel changes coexisting with visceral hypersensitivity or anxious arousal.^[1,6,8]

Neurodevelopmental Presentations with GI Comorbidity: Children or adults with ASD, ADHD, or sensory processing differences who also experience GI symptoms, selective eating, or behavioral changes linked to bowel function.^[5,7,8]

Neuroinflammatory or Neuroimmune Concern: Multisystem symptom patterns—fatigue, headache, cognitive slowing, and GI disturbance—that intensify after infections, antibiotics, or dietary disruption.^[1,8,9,12,13]

What the GI360 Profile Measures

Analyte Class	What It Reflects	Neurologic Relevance
Commensal Microbiome (Diversity & Abundance by PCR)	Shaped by diet, antibiotics, early-life exposures, stress, and environmental factors. ^[1,5]	Altered microbial diversity and dysbiosis patterns have been associated with depression, ASD, and neuroinflammatory conditions at the cohort level. ^[3,4,5]
Short-Chain Fatty Acids (SCFAs — Butyrate, Propionate, Acetate)	Produced by fermentation of dietary fiber by gut bacteria; influenced by diet quality and microbiome composition. ^[2,11]	SCFAs may help contextualize neuroinflammatory tone and cognitive resilience; reduced SCFA-producing taxa have been proposed as contributors to Alzheimer's-context cognitive decline. ^[2,11]

What the GI360 Profile Measures (Cont)

Analyte Class	What It Reflects	Neurologic Relevance
Intestinal Inflammatory Markers (Calprotectin, Lactoferrin, Lysozyme)	Reflect mucosal immune activation in response to dysbiosis, pathogens, dietary antigens, or IBD. ^[9,10,13]	Elevated fecal calprotectin has been reported in Parkinson's disease cohorts and studied in ASD-related barrier research; can support clinical reasoning around intestinal inflammatory burden. ^[8,9,10]
Secretory IgA (sIgA)	Produced by gut-associated lymphoid tissue; modulated by stress, diet, and microbiome health. ^[1,6]	Provides context for mucosal immune tone; low or elevated sIgA may help characterize immune dysregulation patterns in neuro-immune-inflammatory presentations. ^[1,6]
Dysbiotic Bacteria & Yeast (Culture-Based)	Overgrowth of pathogenic or opportunistic organisms following antibiotics, diet change, illness, or immune disruption. ^[1,7]	Dysbiotic flora may contribute to barrier disruption and systemic immune signaling; relevant as a contextual finding in multisystem symptom patterns. ^[1,7,8]
Enteric Pathogens & Parasites (PCR + Microscopy)	Acquired via contaminated food/water, travel, animal contact, or environmental exposure. ^[1]	Undetected infectious burden can complicate interpretation of chronic cognitive, mood, or autonomic complaints; identification may support targeted clinical intervention. ^[1]

Clinical Applications & Evidence Context

Condition	Clinical Encapsulation
Major Depressive Disorder / Depressive Syndromes	Associations between depressive disorders and altered gut microbiota composition, supporting mechanistic plausibility for immune, SCFA, and gut-brain signaling pathways as contributing factors to symptom burden. ^[3,4]
Anxiety Disorders / Anxious Arousal	Growing evidence supports mechanistic plausibility for microbiome-mediated signaling in anxiety, with altered bowel motility, visceral hypersensitivity, and autonomic features frequently co-occurring. ^[2,6]
Autism Spectrum Disorder (ASD)	Systematic reviews confirm established cohort-level associations between ASD and altered gut microbiota, GI comorbidity, and intestinal permeability, with mechanistic plausibility for neuroimmune, SCFA, and autonomic pathways. ^[5,7,8]
ADHD / Attentional Dysregulation	Emerging evidence suggests microbiome differences may be present in ADHD cohorts, with mechanistic plausibility for inflammatory and metabolite pathways. ^[7]
Parkinson's Disease	Established association exists for GI involvement in PD, including constipation preceding motor symptoms; fecal calprotectin has been reported elevated in PD cohorts, supporting intestinal inflammation as a contextual marker. ^[9,10]
Alzheimer's Disease / Cognitive Decline	Biological interest in SCFA-mediated modulation of neuroinflammation and cognition is increasing; current evidence remains largely mechanistic and translational. ^[2,11]
Multiple Sclerosis (MS)	Growing observational data support biologic plausibility for gut microbiota, barrier function, and diet-microbiome interactions in MS pathobiology; stool findings are contextual and not disease-specific at present. ^[12,13]

How the GI360 Profile Supports Clinical Reasoning

Characterizes Gut Microbial Ecology

Multiplex PCR-based microbiome profiling provides a structured view of commensal diversity and dysbiosis patterns that may help contextualize neurologic or mental health symptom burden within the broader brain-gut-microbiome axis. ^[1,3,4]

Assesses Intestinal Inflammatory Tone

Fecal calprotectin, lactoferrin, and lysozyme can support clinical reasoning around mucosal inflammatory activity that may amplify systemic immune signaling relevant to neuroimmune-inflammatory presentations. ^[8,9,10]

Identifies Occult Infectious or Dysbiotic Burden

Pathogen, parasite, and culture-based assessment can uncover undetected gut stressors that may complicate interpretation of chronic cognitive, mood, sensory, or autonomic complaints and inform targeted clinical intervention. ^[1,7]

How to Order

To order the GI360 Profile, 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. Martin CR, Osadchiy V, Kalani A, Mayer EA. The Brain-Gut-Microbiome Axis. *Cell Mol Gastroenterol Hepatol*. 2018;6(2):133-148.
2. Qian XH, Xu S, Sun L, et al. Mechanisms of short-chain fatty acids derived from gut microbiota in Alzheimer's disease. *Ageing Res Rev*. 2022;78:101612.
3. Sanada K, Nakajima S, Kurokawa S, et al. Gut microbiota and major depressive disorder: A systematic review and meta-analysis. *J Affect Disord*. 2020;266:1-13.
4. Cheung SG, Goldenthal AR, Uhlemann AC, Mann JJ, Miller JM, Sublette ME. Systematic Review of Gut Microbiota and Major Depression. *Front Psychiatry*. 2019;10:34.
5. Kortenienmi J, Yang B, Eerola E, et al. Systematic review: Autism spectrum disorder and the gut microbiota. *Acta Psychiatr Scand*. 2023;148(3):242-254.
6. Ferrari S, Mulè S, Parini F, et al. The influence of the gut-brain axis on anxiety and depression. *J Tradit Complement Med*. 2024;14(4):237-255.
7. Caputi V, Hill L, Figueiredo M, et al. Functional contribution of the intestinal microbiome in ASD, ADHD, and Rett syndrome. *Front Neurosci*. 2024;18:1341656.
8. de Magistris L, Familiari V, Pascotto A, et al. Alterations of the intestinal barrier in patients with autism spectrum disorders. *J Pediatr Gastroenterol Nutr*. 2010;51(4):418-424.
9. Hor JW, Lim SY, Chong KK, et al. Fecal Calprotectin in Parkinson's Disease and Multiple System Atrophy. *J Mov Disord*. 2022;15(2):106-114.
10. Al-Kuraishy HM, Al-Gareeb AI, Al-Buhadily AK, et al. Calprotectin in Parkinsonian disease: anticipation and patient stratification. *J Neural Transm (Vienna)*. 2024.
11. Chen H, Meng L, Shen L. Multiple roles of short-chain fatty acids in Alzheimer disease. *Nutrition*. 2022;93:111499.
12. Montgomery TL, Peipert D, Kremmentsov DN. Modulation of multiple sclerosis risk and pathogenesis by the gut microbiota. *Immunol Rev*. 2024.
13. Buscarinu MC, Romano S, Mechelli R, et al. Intestinal Permeability in Relapsing-Remitting Multiple Sclerosis. *Neurotherapeutics*. 2018;15(1):68-74.

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