Irritable Bowel Syndrome as a Stuck Program Mode of the Candida albicans Biochemical Computer

Gut Management Frozen in a Single Operating State

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April 2026

Abstract

Irritable bowel syndrome (IBS) affects 10-15% of the global population and remains a functional diagnosis defined by symptom criteria in the absence of identifiable structural pathology. The Rome criteria classify IBS by what it is not. This paper proposes what it is: a stuck program mode in which the commensal fungal symbiont Candida albicans has locked host gut management into a single operating state. The organism manages gut motility through CB1 receptors, barrier integrity through prostaglandin E2 and immune modulation, visceral pain through TRPV1, microbiome composition through antifungal and antibacterial secretions, and intestinal serotonin through tryptophan diversion. IBS subtypes (IBS-D, IBS-C, IBS-M) represent different frozen modes of this same management system. The framework resolves the persistence of a condition with no structural cause, explains the 2:1 female predominance through documented organism sensitivity to estrogen and luteinizing hormone, accounts for post-infectious IBS as organism reorganization following bacterial disruption, and generates testable predictions linking IBS subtype to organism morphological state and colonization density distribution.

Keywords: irritable bowel syndrome, Candida albicans, gut motility, CB1, TRPV1, prostaglandin E2, visceral hypersensitivity, mast cell activation, tryptophan, kynurenine, serotonin, low-FODMAP, post-infectious IBS, quorum sensing, stuck program mode, Homo candidus, version conflict

I. Introduction

Irritable bowel syndrome is a functional gastrointestinal disorder affecting 10-15% of the global population. It is characterized by chronic abdominal pain, altered bowel habits (diarrhea-predominant, constipation-predominant, or mixed/alternating), bloating, and visceral hypersensitivity. Despite decades of research, no structural cause has been identified. The Rome IV criteria define IBS by its symptom pattern in the explicit absence of demonstrable organic disease. The condition is lifelong, managed but not cured, and represents one of the most common reasons for gastroenterological consultation worldwide.

The absence of structural pathology is the central feature, not a limitation, of IBS. It tells us that the gut hardware is intact. Something is wrong with the management layer. This paper proposes what is managing the gut, why it is stuck, and what produces the specific symptom subtypes that the Rome criteria describe.

The biochemical computer framework (Craddock, 2026a; 2026b) describes C. albicans as a coevolved symbiont operating a phased program within the mammalian host, possessing documented signaling access to the receptor systems governing gut motility, barrier function,

pain perception, immune surveillance, and serotonin metabolism. This program was refined over tens of thousands of generations within the Homo candidus phenotype (Craddock, 2026b), where gut management cycled through coordinated phases under the oversight of social infrastructure that provided dietary protocol, communal feeding, and behavioral intervention when the program required external transition cues. The framework proposes that IBS represents a state in which this management system is frozen in a single operating mode rather than cycling through the coordinated phases a functional Homo candidus program would produce. Modern Homo sapiens carries the organism and its gut management program but lacks the social and physiological architecture that kept the cycling on track. This is a version conflict.

II. The Mechanism

The organism's documented capabilities relevant to gut management span five distinct signaling channels, each of which maps to a recognized IBS symptom domain.

Motility: CB1 Receptor Management

CB1 receptors in the gastrointestinal tract suppress motility when activated. The organism's interaction with the endocannabinoid system, documented extensively in the companion papers (Craddock, 2026a; 2026b), includes modulation of endocannabinoid tone through arachidonic acid substrate competition (Erb-Downward and Noverr, 2007; Acharya et al., 2017). In the CHS analysis presented in Craddock (2026b, Section VI), the framework demonstrates that the organism maintains gut CB1 receptor density at levels that serve its operational requirements, producing the tissue-specific differential between gut and brain CB1 that the standard CHS literature documents but cannot explain (Sharkey and Wiley, 2016). This same CB1 management capacity provides the organism with direct control over gut transit speed. Increased endocannabinoid tone at gut CB1 slows motility. Decreased tone accelerates it. [*The extreme case of 120 hour transit time is noted in (Craddock, 2026c), an overlooked moment in science*]

Barrier Integrity: Prostaglandin and Immune Management

The organism produces authentic prostaglandin E2 from host arachidonic acid (Erb-Downward and Noverr, 2007), modulates dendritic cell function through farnesol (Leonhardt et al., 2015), drives Th2 polarization through PGE2-mediated immune skewing, and manages mucosal immune surveillance through the tryptophan-kynurenine diversion that suppresses IL-17 (Cheng et al., 2010; Zelante et al., 2013). Collectively, these capabilities provide comprehensive management of the gut mucosal barrier: its permeability, its immune tone, and its response to luminal contents. Barrier function in IBS patients oscillates between increased and decreased permeability, a pattern consistent with organism management rather than structural damage. [*Sure seems like the required arsenal*]

Visceral Pain: TRPV1 Management

Visceral hypersensitivity is the hallmark of IBS: patients report pain at stimulus intensities that are subthreshold in healthy controls. TRPV1, a receptor responsive to noxious heat, capsaicin, and endogenous lipid mediators including anandamide, is expressed throughout the gut and is a documented component of the organism's signaling interface (Craddock, 2026b, Section VI). If the organism manages TRPV1 sensitivity as part of its gut management program, a stuck state in which TRPV1 sensitivity is maintained at an elevated level would produce

visceral hypersensitivity as a direct consequence. The organism is not causing pain. It is maintaining a sensory alert state that serves a program phase, and the phase is not ending.

Microbiome Composition

C. albicans exerts direct antimicrobial effects against bacterial populations in the gut through secreted metabolites, biofilm competition, and immune landscape management (reviewed in Craddock, 2026b, Section 5.4). The organism does not exist passively alongside the bacterial microbiome. It manages the bacterial landscape as part of its operational toolkit. The gut microbiome alterations consistently documented in IBS patients, including shifts in Firmicutes/Bacteroidetes ratios, reduced diversity, and altered short-chain fatty acid profiles, may reflect the organism's management of its bacterial environment rather than an independent dysbiosis.

Serotonin: Tryptophan Diversion

Approximately 90% of the body's serotonin is produced in the gut, where it regulates motility, secretion, and visceral sensation. The organism's capacity to divert tryptophan through the kynurenine pathway (Cheng et al., 2010; Zelante et al., 2013) provides direct modulation of gut serotonin availability. Reduced serotonin synthesis in the gut alters motility patterns and visceral sensitivity, two of the three cardinal features of IBS. The tryptophan diversion serves the organism's immune evasion requirements (suppressing IL-17) while simultaneously producing the gut symptoms that define IBS as a clinical entity.

III. The Stuck State: Subtypes as Frozen Modes

In a functional program [*Homo candidus*], the organism cycles its gut management through coordinated phases: adjusting motility, barrier function, and pain sensitivity based on program requirements. These phases serve different purposes, including gut clearance during substrate transitions, extended processing during absorption-intensive phases, and heightened vigilance during immune challenge. The cycling is what produces normal, variable but regulated bowel function.

IBS subtypes represent the loss of cycling: the organism locked into a single mode of gut management without the transition signal to advance.

IBS-D (diarrhea-predominant): motility acceleration stuck in the on position. The organism is driving rapid transit for a phase-specific purpose, such as gut clearance or substrate flushing, without the braking signal that would return motility to baseline. Endocannabinoid tone at gut CB1 is reduced, removing the motility brake. Transit time drops. The host experiences urgency, frequency, and loose stools because the gut is running a clearance program that was designed to be temporary. [*From experience, temporary can mean a long time, but the key is not permanent*]

IBS-C (constipation-predominant): motility suppression stuck in the on position. The organism is holding gut contents for extended processing or absorption, maintaining elevated endocannabinoid tone at gut CB1 to suppress motility. The release signal that would restore normal transit does not arrive. The host experiences infrequent, difficult bowel movements because the gut is running a retention program that does not end.

IBS-M (mixed/alternating): the organism oscillating between modes without achieving the stable cycling a functional program would produce. The transition signals are partially

functional, sufficient to initiate a mode shift but insufficient to stabilize in either mode. The host experiences unpredictable alternation between diarrhea and constipation because the management system is hunting for a stable state it cannot reach.

Visceral hypersensitivity is common across all three subtypes because TRPV1 management is a separate channel from CB1 motility management. The organism can be stuck in any motility mode while simultaneously maintaining TRPV1 at elevated sensitivity. The sensory alert state runs independently of the motility program.

The IBS subtypes illustrate the *version conflict* between the organism's program and its modern host with particular clarity. The gut management system is cycling correctly from the program's perspective. It is executing clearance phases, retention phases, and immune vigilance phases exactly as designed. The problem is that the transition signals it expects, signals that in Homo candidus came from communal dietary protocol, managed feeding schedules, and social behavioral cues, do not exist in modern Homo sapiens. The program initiates a phase and waits for the transition signal. The signal never comes. The phase runs. The patient gets a diagnosis.

IV. The Persistence Problem Resolved

The reason no structural cause has been found for IBS is that the structure is intact. The management layer is stuck. Every endoscopy, colonoscopy, CT scan, and biopsy in IBS comes back normal because there is nothing wrong with the gut hardware. The abnormality is in the software: an organism-mediated management system frozen in a single operating state due to the version conflict noted above.

This is why IBS is lifelong. The organism's management of the gut is continuous. As long as the organism is present and above the quorum sensing threshold required for coordinated management, the stuck state persists. Symptom fluctuation over time reflects variation in organism density, dietary inputs, and hormonal cycling, all of which modulate the organism's operational context without providing the full transition signal that would release the stuck mode.

The Rome criteria, by defining IBS in the absence of organic disease, have inadvertently created a diagnostic category that perfectly describes a management-layer disorder. The framework provides the management layer the criteria were implicitly acknowledging: a biological system that is present, active, and measurable, but has never been measured in this context. The management layer is not the problem. The body is not the problem, it is an interface problem created by a version conflict.

V. Clustering Evidence

IBS shows a 2:1 female predominance globally. The organism's documented sensitivity to estrogen and LH (Kinsman et al., 1988) provides a direct biological mechanism: the female hormonal cycle modulates the organism's operational state with monthly periodicity, and the gut management program responds to these hormonal inputs. The female predominance in IBS, like the female predominance in AN, is not sociocultural. It is a consequence of the organism's hormonal sensing infrastructure responding to the female endocrine environment.

IBS onset typically occurs in young adulthood, the same developmental window as AN, consistent with hormonal activation changing the organism's operational context at puberty and the early reproductive years. [*This is not the only stuck state that can arrive at this time. C. abicans is aware of all system changes. Major changes are more likely to provoke a response*]

Post-infectious IBS, the development of IBS following an acute gastrointestinal infection, is well-documented in the gastroenterological literature. The framework provides a clear mechanism: the acute infection disrupts the bacterial populations the organism manages as part of its gut toolkit. During the reorganization period following the infection, the organism reconfigures its gut management program in the context of an altered bacterial landscape. If the reorganization does not achieve stable cycling, the organism locks into a single management mode, and post-infectious IBS is the result. The trigger was bacterial, but the persistence is fungal.

Mast cell activation in IBS, increasingly recognized as a contributor to symptoms, connects directly to the organism's documented capabilities. C. albicans cell wall components (alpha-mannan, beta-glucan) trigger mast cell degranulation through pattern recognition receptors (Castro et al., 1994; Sakurai et al., 2012). Histamine release from mast cells drives the acute symptom flares, visceral pain, and altered motility that characterize IBS exacerbations. The organism is not incidentally present while mast cells activate. It is the trigger.

VI. Unfreezing: Therapeutic Implications

Low-FODMAP Diet

The low-FODMAP diet is the current gold-standard dietary intervention for IBS, with demonstrated efficacy across multiple randomized controlled trials. FODMAPs are fermentable carbohydrates metabolized by gut organisms. Within the framework, the low-FODMAP diet works because it directly changes the substrate landscape the organism and its managed bacterial populations read. By reducing fermentable substrates, the diet shifts the metabolic environment in which the organism operates, potentially providing the input that breaks the stuck mode.

The framework predicts differential response across subtypes because the same substrate change shifts the organism's context differently depending on which mode it is stuck in. This prediction is testable and would provide mechanistic specificity that current FODMAP research lacks: if organism density and morphological state correlate with FODMAP response, the framework gains evidential support.

Exercise

Physical exercise improves IBS symptoms through mechanisms that remain incompletely characterized in the standard literature. The framework identifies dual pathways: mechanical gut stimulation provides a physical input the organism reads as an environmental change, and the metabolic/hormonal shifts of exercise (cortisol dynamics, post-exercise endocannabinoid tone changes, autonomic rebalancing) supply multiple simultaneous inputs that can break single-mode lock. The combination of mechanical and biochemical signals during exercise provides more diverse transition input than dietary change alone.

Probiotics

Probiotics show modest, inconsistent benefit in IBS across meta-analyses. The framework explains the inconsistency directly: probiotics add bacteria to an environment managed by an organism whose density and operational state are not measured. The same probiotic could help one patient, where it shifts the bacterial landscape enough to change the organism's management context, and do nothing for another, where the organism's density is sufficient to override whatever the probiotic introduced. Probiotic trials that do not control for

mycobiome state are shooting in the dark, and the framework predicts they will continue to produce inconsistent results.

Dietary Antifungals

Coconut oil (lauric acid, caprylic acid), garlic (allicin), and cinnamon (cinnamaldehyde) provide dietary antifungal pressure accessible without prescription. Lemon balm is of particular relevance for IBS: it has documented antifungal properties alongside well-established antispasmodic effects on the gut smooth muscle. For an IBS patient whose visceral pain and motility disruption are organism-mediated, lemon balm addresses the organism directly while simultaneously relieving the downstream gut symptom through an independent pathway. The framework predicts that dietary antifungal intake concurrent with low-FODMAP should outperform either intervention alone. The substrate change provides the transition signal; the antifungal pressure reduces the organism's capacity to resist the transition by lowering density below the quorum sensing threshold maintaining the stuck mode.

VII. Testable Predictions

Prediction I1: IBS subtype (IBS-D, IBS-C, IBS-M) correlates with organism morphological state (yeast-to-hyphal ratio) and/or colonization density distribution pattern within the gut. Specifically, the framework predicts that IBS-D patients show different organism density or morphological profiles at colonic biopsy than IBS-C patients.

Prediction I2: Post-infectious IBS onset correlates with measurable mycobiome reorganization following the triggering infection. Specifically, C. albicans density or strain composition shifts detectably during the post-infection period in patients who develop IBS versus those who do not.

Prediction I3: The AN-IBS comorbidity rate is significantly higher than predicted by independent co-occurrence of two conditions with their respective population prevalences, consistent with a shared organism-driven etiology.

Prediction I4: Low-FODMAP diet response magnitude correlates with baseline C. albicans colonization density. Patients with higher organism density show either greater or lesser FODMAP response depending on subtype, providing mechanistic specificity that current dietary trials lack.

Prediction I5: Mast cell activation severity in IBS patients correlates with fecal C. albicans density, consistent with organism cell wall components (beta-glucan, alpha-mannan) serving as the primary mast cell trigger through pattern recognition receptor engagement.

VIII. Limitations

IBS is a heterogeneous condition with multiple recognized contributors including visceral hypersensitivity, gut-brain axis dysfunction, altered intestinal permeability, bile acid malabsorption, and small intestinal bacterial overgrowth. The framework does not claim that C. albicans is the sole driver. It proposes that organism-mediated gut management represents an unrecognized layer whose inclusion would explain the specific patterns that existing models

describe but do not mechanistically resolve: the absence of structural pathology, the subtype-specific symptom patterns, the female predominance, and the post-infectious onset trigger.

The subtype-as-frozen-mode model is the most novel and least supported component of the paper. While each individual mechanism is documented, the mapping of subtypes to specific organism management states is inferred from the framework rather than demonstrated by existing data. Prospective studies correlating organism state with subtype are required.

This paper is part of a series applying the biochemical computer framework to chronic disease. The companion umbrella paper (Craddock, 2026d) describes the stuck-program model and selection methodology. The foundational framework is described in Craddock (2026a) and Craddock (2026b).

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