Anorexia Nervosa as a Stuck Program Mode of the Candida albicans Biochemical Computer

Substrate Restriction Without Phase Transition

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Abstract

Anorexia nervosa (AN) carries the highest mortality rate of any psychiatric disorder, with relapse rates of 30-50% following weight restoration. The persistence of the condition despite aggressive refeeding remains unexplained. Monteleone et al. (2015) demonstrated that endocannabinoid responses to hedonic eating remain abnormal in weight-restored AN patients, indicating that the physiological disruption outlasts the caloric deficit. This paper applies the biochemical computer framework (Craddock, 2026a; 2026b) to propose that AN represents a stuck program mode in which the organism Candida albicans has locked host feeding behavior into a substrate restriction phase through sustained modulation of endocannabinoid tone, serotonin precursor availability, and satiety signaling. The restriction phase serves the organism's program requirements for specific metabolic conditions, but the transition signal to resume normal feeding fails to arrive. The framework resolves the persistence problem, explains the pubertal-onset female predominance through documented organism sensitivity to estrogen and luteinizing hormone, and generates testable predictions linking organism colonization density to endocannabinoid disruption severity and treatment response. The net effect is directly dependent on colonization density.

Keywords: anorexia nervosa, Candida albicans, endocannabinoid system, hedonic eating, CB1, tryptophan, kynurenine, serotonin, OEA, PEA, stuck program mode, quorum sensing, pubertal onset, estrogen, dietary antifungal, Homo Candidus

I. Introduction

Anorexia nervosa is classified as a psychiatric disorder characterized by caloric restriction, distorted body image, and intense fear of weight gain. Standard treatment models frame it as a cognitive-behavioral pathology maintained by psychological reinforcement loops. These models do not explain why the condition is so resistant to treatment that it kills more patients than any other psychiatric diagnosis, why weight restoration fails to normalize the underlying physiology, or why the demographic distribution is so dramatically skewed toward young females with pubertal onset.

The biochemical computer framework (Craddock, 2026a; 2026b) provides an alternative interpretation: AN is not a psychological disorder that affects eating. It is an organism-mediated metabolic program that affects psychology. The organism C. albicans, operating through documented signaling interfaces with the host's endocannabinoid system, serotonin synthesis pathway, and satiety signaling, has locked feeding behavior into a restriction mode that serves a specific program phase. The restriction is not the malfunction. The malfunction is that the program phase does not end.

II. The Mechanism

The organism's access to the host's feeding behavior operates through multiple documented signaling channels, each contributing a distinct component of the restriction phenotype.

Endocannabinoid Tone Management

C. albicans makes its own version of a human inflammatory signal, prostaglandin E2, by hijacking the same raw material (arachidonic acid) that the host uses to make its feel-good molecules: the endocannabinoids anandamide and 2-AG (Erb-Downward and Noverr, 2007; Acharya et al., 2017). There is a finite pool of this raw material. Every molecule the organism diverts toward prostaglandin production is one the host cannot use to make the endocannabinoids that govern how pleasurable food feels. The organism is not blocking pleasure directly. It is outcompeting the host for the ingredients pleasure is made from.

Monteleone et al. (2015) tested this system in anorexia patients. They measured endocannabinoids and related compounds in underweight AN patients, weight-restored AN patients, and healthy controls after eating foods they liked versus foods they did not. In healthy people, eating pleasurable food triggers a specific endocannabinoid response: the body's reward chemistry activates. In AN patients, that response was disrupted. The critical finding: it stayed disrupted even after the patients regained weight. Their bodies were back to normal weight, but their reward chemistry was not. Whatever was driving the disruption had not been fixed by feeding them. The program was still running. [*From my perspective, after reading the Article mentioned in (Craddock, 2025), I knew I always had to eat, so I ate. Even when it putting food in my mouth caused me to actively gag, I ate. AN patients do not start knowing that.*]

Two of the compounds Monteleone measured are particularly informative. PEA (palmitoylethanolamide) is part of the body's endocannabinoid-related signaling, but it also has documented antifungal properties through immune modulation. When the organism modulates PEA, it is doing two things at once: altering the host's reward signaling and modulating a compound that enhances host immune surveillance, tilting the immune landscape toward the permissive Th2 profile the organism maintains. It is managing appetite and managing its own immune evasion through the same molecule.

OEA (oleoylethanolamide) is the body's natural "I'm full" signal. The organism's capacity to modulate OEA gives it **direct control** over when the host stops eating. Put these together: an organism that can suppress how good food feels (endocannabinoid depletion), accelerate how quickly the host feels full (OEA manipulation), and protect itself from the host's defenses (PEA suppression), all through the same signaling layer, has *comprehensive* control of feeding behavior. That is not a side effect of infection. That is management architecture.

The Elders indicated in The Oscillation Hypothesis (Craddock, 2026b) would have had the knowledge to tell their people to eat through everything. That's something 10,000+ generations can easily learn.

Serotonin Depletion

C. albicans diverts host tryptophan through the kynurenine pathway, producing enzymes that redirect tryptophan away from serotonin synthesis and toward kynurenine and its downstream metabolites (Cheng et al., 2010; Zelante et al., 2013). The net direction of tryptophan metabolism in the presence of C. albicans varies by experimental context, but the

functional outcomes, IL-17 suppression at mucosal surfaces and reduced serotonin precursor availability, are consistent across the literature that is most relevant to the gut-resident commensal scenario described here. This rerouting simultaneously depletes a neurotransmitter precursor essential for mood regulation and appetite and disables the host's primary mucosal defense against fungi (IL-17-mediated immunity). The organism achieves immune evasion and behavioral modification through a single metabolic diversion.

The downstream product quinolinic acid is a potent NMDA receptor agonist and documented neurotoxin. Sustained kynurenine pathway activation produces the anxiety, mood disturbance, and cognitive rigidity characteristic of AN through a biochemical mechanism, not a psychological one.

Compulsive Exercise as Palliative Trap

Compulsive exercise is a hallmark feature of AN, conventionally treated as psychopathology. The framework reads it as something more layered and more dangerous than either simple psychopathology or simple organism-directed behavior. It is both at once, and the interaction between the two is what makes it lethal.

AN patients consistently report pervasive tension and anxiety that exercise temporarily resolves, consistent with the organism's simultaneous suppression of serotonin (via tryptophan diversion) and modulation of GABA-A inhibitory tone (via farnesol), producing a neurochemical state in which stillness is physiologically intolerable. [*This is certainly represented on the Homo Candidus timeline. There were times I worked out twice a day (lifting 1 hour, cardio 30 minutes) 3 times a week, and then added a five mile run for stretch day*]

The organism benefits from exercise directly: the host burns calories, maintaining the metabolic conditions the restriction phase requires. But the host benefits too, and this is the trap. Exercise generates endogenous anandamide, one of the exact endocannabinoid ligands the organism is suppressing through arachidonic acid competition. During and immediately after exercise, the organism's suppression of hedonic tone is temporarily overridden. Food becomes tolerable. Anxiety recedes. The body feels, briefly, like it belongs to the person living in it. The AN patient who cannot stop exercising is not merely obeying the organism's caloric expenditure directive. They are self-rescuing through the only channel that temporarily restores the reward chemistry the organism has suppressed. The exercise feels essential because it is. It is the only time the pleasure system works.

This is the palliative trap, the same mechanism described in the CHS hot shower analysis (Craddock, 2026b, Section VI.II.IV), operating through endogenous cannabinoid rather than exogenous heat. The short-term relief drives a long-term worsening cycle. The organism reads each anandamide spike from exercise the same way it reads chronic THC in CHS patients: as interference with its management of the CB1 signaling layer. It compensates by tightening its grip on endocannabinoid tone during non-exercise periods. The baseline between exercise sessions gets worse. The restriction deepens. The anhedonia intensifies. The only relief is more exercise. But more exercise provokes stronger compensation, which deepens the trough further, which demands more exercise. The patient is not addicted to exercise in a psychological sense. They are trapped in a positive feedback loop in which the organism makes everything feel progressively worse when they stop, and the only temporary escape is the behavior that makes the underlying suppression progressively stronger.

The clinical trajectory of AN reflects this escalation. The sickest patients, the ones exercising through emaciation at levels that are physiologically dangerous, are not simply further

along in the restriction program. They have been running the feedback loop longer. Each cycle of exercise and compensation has deepened the organism's entrenchment in the signaling layer. The exercise is simultaneously the organism's tool for caloric expenditure, the host's only source of hedonic relief, and the mechanism by which both the restriction and the anhedonia worsen over time. In the Homo candidus context described in Craddock (2026b), the social group would intervene before this loop reached lethal intensity: communal feeding, enforced rest, behavioral protocol. In modern Homo sapiens, no such infrastructure exists, and the loop runs to its conclusion.

III. The Stuck State

The program phase represented by AN is substrate restriction: the organism locks feeding behavior to drive specific metabolic conditions, possibly including ketogenesis, altered bile salt composition, tissue substrate availability, or the metabolic staging required for an intermediate-to-advanced program phase. The compulsive exercise and heat-seeking behavior documented in AN patients contributes to this staging through a mechanism that is basic physiology: sweating is hypotonic relative to serum. Every episode of exercise-induced sweating concentrates the host's blood, raising serum osmolality. Elevated osmolality triggers hypothalamic osmoreceptors to drive vasopressin release from the posterior pituitary. The pituitary works harder. If the organism governs or colonizes the pituitary as the framework proposes, elevated osmolality is not a side effect of the restriction program. It is a performance condition: higher osmolality means more pituitary throughput, more hormonal modulation capacity, and a faster-running program. The AN patient is not merely starving and exercising. They are, through the combined effects of caloric restriction and hypotonic fluid loss, constructing the internal saline environment in which the organism operates at maximum capacity. The restriction, the exercise, and the sweating are not three separate symptoms. They are a coordinated staging operation that produces the metabolic, electrolyte, and endocrine conditions a program phase requires.

In a functioning Homo candidus program, this phase would be temporary, terminated by communal feeding intervention [“*Eat, no matter what” – Craddock, 2025 p 43*] behavioral protocol enforcement, or the natural completion of whatever metabolic condition the phase was staging. The social group would rehydrate, refeed, and enforce rest before the osmolality shift and caloric deficit reached dangerous levels. The phase was designed to run hot, briefly, under supervision.

In modern Homo sapiens, none of these termination signals arrive. The social infrastructure of Homo candidus, which the companion paper (Craddock, 2026b, Section 8) describes as behavioral control encompassing diet, fluid management, pain endurance, ritual, and authority, does not exist. The restriction program runs unchecked because the system that would have stopped it is absent from the species that carries the organism. The osmolality climbs unchecked. The pituitary stays overclocked. The exercise compulsion deepens through the palliative trap. The host wastes toward a threshold that, in Homo candidus, the community would never have allowed them to approach.

The quorum sensing dimension maintains the stuck state at the population level. As the organism population coordinates through farnesol and tyrosol above a density threshold, the restriction program sustains its coherence. Refeeding without reducing organism density pushes calories past a program that is still running restriction mode. Rehydration without antifungal intervention dilutes the osmolality the organism has established, provoking compensatory fluid

loss to re-concentrate. The program has not received its transition signal. It has merely been overridden temporarily.

IV. The Persistence Problem Resolved

The central mystery of AN is persistence. Weight restoration does not cure the condition. Relapse rates of 30-50% following treatment indicate that the underlying driver survives the intervention. The Monteleone et al. (2015) finding that endocannabinoid responses remain abnormal after weight restoration is the single most informative data point: the physiological disruption outlasts the caloric deficit. The program is still running.

Duration of illness is the single strongest predictor of poor outcome in AN. Within the framework, this follows directly: longer program execution means deeper entrenchment of the operational state. The organism's quorum sensing coordination, signaling pathway management, and metabolic substrate landscape all deepen with duration. A restriction program that has run for ten years is harder to unstick than one that has run for one year, not because of psychological entrenchment but because the organism's management of the signaling landscape has had ten years to consolidate.

The patients who actually recover, meaning not just weight restoration but normalization of food reward and feeding behavior, would be those in whom the organism's operating context changed sufficiently to provide the transition signal. Hormonal maturation in the late teens and early twenties may provide a natural transition as the endocrine environment shifts, consistent with the clinical observation that some patients appear to age out of AN. The framework predicts that this aging-out correlates with hormonal shifts that change the organism's operational priorities, not with psychological maturity. [*Other unexplained conditions such as bedwetting also frequently stop during this hormonal transition period. Bedwetting is known to be correlated with other conditions later in life. But that is another paper, perhaps*]

V. Clustering Evidence

The demographic distribution of AN is among the most dramatic of any disease: overwhelming female predominance and onset clustered around puberty. Conventional models attribute this to sociocultural pressure, but sociocultural models do not explain why the condition's physiological signature (endocannabinoid disruption, serotonin depletion, compulsive exercise, hypothermia, amenorrhea) is consistent across radically different cultural contexts.

The framework provides a biological explanation. C. albicans possesses confirmed receptors or binding proteins for estrogen and responds to luteinizing hormone (LH) and human chorionic gonadotropin (hCG) with increased yeast-to-hyphal transition (Kinsman et al., 1988). The response is specific: FSH, TSH, GH, and prolactin do not trigger the transition. Puberty activates the hormonal environment, specifically estrogen and LH cycling, that changes the organism's operational context. The female endocrine cycle creates a substrate management landscape distinct from the male hormonal environment, and the organism's documented sensitivity to these specific hormones explains why the restriction program locks disproportionately in females at the developmental stage when these hormones activate.

The AN-IBS comorbidity rate is clinically well-documented and higher than population prevalence would predict. Within the framework, this is not comorbidity. It is two stuck states running simultaneously from the same organism: one managing feeding behavior (AN), the other

managing gut motility and barrier function (IBS). Their co-occurrence in the same individuals at rates exceeding chance is consistent with a shared organism-driven etiology.

VI. Unfreezing: Therapeutic Implications

AN is the hardest candidate to unfreeze in this series, and the clinical data confirms it.

Structured Refeeding Plus Antifungal Pressure

Weight restoration alone does not provide the transition signal because it does not change the organism's operating context. The framework predicts that structured refeeding combined with sustained antifungal pressure, reducing organism density below the quorum sensing threshold for restriction-mode maintenance, should produce better long-term outcomes than refeeding alone. As organism density drops, the endocannabinoid management loosens. The host begins experiencing normal food reward. The program releases.

Dietary antifungals (coconut oil, crushed garlic, cinnamon) provide accessible, low-risk antifungal pressure that could be incorporated into refeeding protocols without pharmacological intervention. Lemon balm is of particular interest as a dual-mechanism intervention: it has documented antifungal properties alongside anxiolytic and acetylcholinesterase inhibitor effects. If an AN patient is experiencing organism-mediated anxiety and hedonic suppression, lemon balm addresses both the organism directly and the downstream symptom through independent pathways.

The Die-Off Complication

Antifungal intervention in AN carries a specific risk the framework predicts: organism die-off can produce acute symptom exacerbation before improvement. The Herxheimer-like response documented in candidiasis treatment would manifest as intensified GI symptoms, mood disturbance, and possibly increased restriction drive in the short term [*from experience, imagine a week long hangover*]. Clinical protocols incorporating antifungal pressure in AN would need to account for a worsening period and ensure sufficient support through it. This is not a reason to avoid the intervention. It is a reason to anticipate the trajectory.

Hormonal Transition as Natural Unfreezing

The framework predicts that hormonal maturation in the late teens and early twenties provides a natural transition signal as the estrogen/LH environment shifts from the acute activation of puberty toward the more stable cycling of full reproductive maturity. This is consistent with the clinical observation that AN incidence peaks in adolescence and some patients recover without specific intervention in their twenties. The organism's hormonal sensing infrastructure reads the maturing endocrine landscape as a different operational context, and the restriction program may release when the hormonal input that locked it no longer matches.

VII. Testable Predictions

Prediction A1: Oral and gut C. albicans colonization density correlates with endocannabinoid disruption severity in AN patients. Specifically, higher organism density predicts greater suppression of 2-AG response to hedonic eating, as measured by the Monteleone et al. (2015) protocol.

Prediction A2: Antifungal intervention (pharmaceutical or dietary) partially normalizes hedonic eating responses in AN patients in a way that weight restoration alone does not. The magnitude of normalization correlates with the degree of organism density reduction achieved.

Prediction A3: IBS comorbidity in AN patients occurs at rates significantly above population prevalence, consistent with a shared organism-driven etiology rather than independent co-occurrence.

Prediction A4: Kynurenine pathway metabolites (quinolinic acid, kynurenic acid) are elevated in AN patients relative to BMI-matched controls and correlate with organism colonization density rather than with nutritional status alone.

Prediction A5: AN patients who achieve sustained recovery (normalized hedonic eating, not merely weight restoration) show measurably different mycobiome trajectories compared to those who relapse, with sustained reduction in C. albicans density preceding and predicting behavioral normalization.

VIII. Limitations

The framework does not claim that C. albicans is the sole driver of AN. Genetic predisposition, psychological reinforcement, and sociocultural factors contribute to the clinical presentation. The claim is that organism-mediated management of endocannabinoid tone, serotonin synthesis, and satiety signaling represents an unrecognized physiological layer whose inclusion would improve the explanatory power of existing models, particularly for the persistence of the condition despite weight restoration and the striking demographic clustering around pubertal females.

The Monteleone et al. (2015) data, while strongly consistent with the framework, was not designed to test organism-mediated hypotheses and did not measure mycobiome variables. The interpretation offered here is post hoc. Prospective studies measuring organism density alongside endocannabinoid responses are required to test the framework's predictions directly.

The die-off risk in AN patients receiving antifungal intervention has not been characterized and represents a clinical concern that must be addressed before any therapeutic application of the framework in this population. AN patients are medically fragile, and any intervention that produces acute symptom exacerbation, even temporarily, requires careful clinical management. [*Moderation in all things…the Greeks weren't stupid*]

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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