# Endometriosis as Organism-Directed Tissue Recruitment

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Jim Craddock
Redacted Science Research Initiative
redactedscience.org | jimcraddock.com
20260423

## 1. Abstract

Endometriosis is conventionally described as the presence of endometrium-like tissue outside the uterus, most commonly explained through retrograde menstruation, hormonal dependence, immune dysregulation, and altered inflammatory handling. Under the Redacted Science framework, those observations are not rejected. They are reorganized. This first-draft paper proposes that endometriosis represents a stuck program mode in which resident Candida albicans colonies direct ectopic tissue recruitment and maintenance at colonized pelvic, peritoneal, and bowel interfaces. Menstrual cyclicity matters because it supplies the repeating substrate pulse the organism can exploit: progesterone withdrawal, endometrial breakdown, blood products, iron, cytokines, growth factors, and altered local immune tone.

The paper extends the biochemical computer model of C. albicans (Craddock, Biochemical Computer), the coevolutionary framing of the Saline Oscillation Hypothesis (Craddock, Saline Oscillation), and the stuck-program architecture of the Umbrella and IBS papers (Craddock, Stuck-State Umbrella; Craddock, IBS Stuck State) to reproductive tissue. The core claim is not that endometriotic implants are false endometrium. The claim is that the organism recruits local tissue into endometrial behavior where it already has positional advantage, then sustains that ectopic program through cyclic feeding, interstitial signaling, hormone sensing, and local immune management. This reframes chronicity. Retrograde flow may be common; persistent establishment at specific sites is the event that requires explanation.

The framework resolves several otherwise disconnected features at once: the mismatch between common retrograde flow and lower disease prevalence, the bowel and pelvic distribution of disease, the strength of gastrointestinal comorbidity, the benefit of hormonal suppression despite the condition being framed as misplaced tissue, and the substantial recurrence problem after incomplete excision. It generates six testable predictions centered on implant-site mycobiome signal, antifungal response, antibiotic-exposure linkage, pregnancy-associated shifts, responder enrichment, and recurrence following incomplete colonized-site removal. Recent work by Talwar et al. (2025) provides the first direct mycobiome-centered evidence consistent with the framework's predictions. This paper is not offered as a clinical practice guideline or treatment protocol. It is a framework paper advancing a specific mechanistic claim and exposing that claim to refutation.

## 2. Keywords

Endometriosis, Candida albicans, Biochemical Computer,Stuck State, Organism-Directed Tissue Recruitment, Mycobiome, Pelvic Colonization, Retrograde Menstruation, Irritable Bowel Syndrome, Bowel-Endometriosis Comorbidity, Hormonal Suppression, Homo candidus, Governance Load, Redacted Science Research Initiative, #TheArchitect

# 3. Introduction

The Redacted Science framework proposes that Candida albicans functions as a coevolved biochemical computer in mammalian hosts, operating a distributed governance system over host physiology through the endocannabinoid interface, the perfusion architecture of the pituitary, and the substrate metabolism of multiple organ systems. Under that model the organism is not adequately described as a simple opportunistic pathogen. It is a commensal regulator with documented signaling breadth, positional flexibility, and memory architecture sufficient to shape host output across multiple timescales (Craddock, Biochemical Computer; Craddock, Saline Oscillation).

The Saline Oscillation Hypothesis places that framework in evolutionary context. It argues that the organism-host partnership was refined under repeating environmental pressure and that reproductive physiology sits among the sites of strongest organism-host interaction because cyclical hormone variation, blood flow shifts, tissue remodeling, and social reproduction all converge there (Craddock, Saline Oscillation). The stuck-program model then applies this broader architecture to chronic disease, asking a simple question that modern medicine often leaves underexplained: not merely what a condition looks like, but what keeps it running once established (Craddock, Stuck-State Umbrella; Craddock, Longitudinal Case Study).

Under this paper's specific claim, endometriosis represents a reproductive stuck state in which the organism directs tissue recruitment at extra-uterine sites it already occupies. The monthly symptom pattern is not incidental. It is the repeated substrate event that feeds and stabilizes the state. This paper is not a literature review of endometriosis, not a treatment guideline, and not a surgical protocol. It is a framework paper arguing that the missing variable in chronic ectopic endometrial establishment is the organism layer.

# 4. Framework Basis

## Paper A: Biochemical Computer Hardware

Paper A (Craddock, Biochemical Computer) provides the mechanistic hardware. Its treatment of spatial multiplexing and feedback, the idea that different subroutines can run concurrently across distinct micro-niches while shared signaling pathways integrate local conditions into coordinated output, is directly relevant here. Endometriosis is anatomically distributed rather than random. The model requires an organism able to run location-specific behavior across bowel, peritoneal, ovarian, and pelvic sites at the same time.

## Paper A: Interstitium, Nutrient Interface, and Tissue Transit

Paper A's treatment of the interstitium as a body-wide signaling and nutrient-distribution layer is even more directly relevant to endometriosis. If pelvic and peritoneal surfaces are already organism-accessible interfaces, then cyclical blood products and inflammatory signals from menses do not need to be interpreted as passive debris alone. They become routed substrate within a connected interstitial environment. The same paper also describes documented hyphal transit capability and direct hormone-processing machinery, including steroid handling relevant to reproductive tissue governance.

## Paper B: Coevolution and Reproductive Interaction

Paper B (Craddock, Saline Oscillation) expands the model from molecular capability to evolutionary positioning. It argues that reproductive endocrine cycling is not peripheral to the symbiosis but one of its richest input surfaces. The hormone-sensing inventory, reproductive-axis discussion, and broader

coevolutionary framing support the premise that cyclical reproductive tissue remodeling would be a natural site for organism-managed patterning rather than an exceptional one.

### Umbrella and IBS Papers: Stuck-State Logic and Bowel Precedent

The Umbrella Paper (Craddock, Stuck-State Umbrella) supplies the stuck-program logic: disease persistence reflects a phase that failed to transition. The IBS paper (Craddock, IBS Stuck State) then demonstrates how a condition with strong bowel involvement, substantial female skew, functional persistence, and no adequate structural explanation can be reframed as management frozen in one operating mode. Endometriosis shares the persistence problem but adds tissue recruitment and cyclic substrate pulses. The bowel precedent matters because endometriosis and IBS frequently travel together, and under this framework that is not a coincidence. It is shared geography.

### Paper C: Case-Study Discipline and Source Posture

Paper C (Craddock, Longitudinal Case Study) matters less because it is a direct analogue to endometriosis and more because it establishes methodological posture for the series: the framework is willing to make specific mechanistic claims, expose them to falsification, and say plainly that conventional diagnostics often describe downstream effects while missing the management layer sustaining them. That posture governs this paper as well.

## 5. Mechanism

Conventional theory places retrograde menstruation at the center of endometriosis. Under this framework, retrograde menstruation is not rejected. It is demoted from full explanation to recurring input event. Many women may experience some degree of retrograde flow; far fewer develop chronic ectopic establishment. That mismatch suggests that tissue deposition alone is insufficient. The framework asks what converts a recurrent pelvic exposure into persistent, self-maintaining implant behavior. Its answer is organism-directed tissue recruitment at colonized sites.

The organism already possesses the toolset required for that conversion. Paper A describes a system that senses nutrients, hormones, pH, immune tone, and local damage; shifts among subroutines across micro-niches; moves physically through tissues; and broadcasts soluble or vesicle-borne signals through interstitial space (Craddock, Biochemical Computer). In the pelvic compartment, menstruation delivers an extraordinarily information-rich and nutrient-rich pulse: hemoglobin breakdown products, iron, inflammatory mediators, extracellular matrix fragments, cytokines, prostaglandins, and the withdrawal pattern of progesterone itself. Under this model, the organism does not merely survive in that environment. It interprets it.

The monthly cycle therefore becomes the repeating compilation event. Progesterone withdrawal and endometrial breakdown signal that a uterine remodeling program is underway. At extra-uterine sites where the organism already holds positional authority, that signal is misapplied. Local stromal and mesothelial tissues are recruited into endometrial-like behavior because the organism is routing a uterine tissue-remodeling instruction into tissue that is not supposed to receive it. Histology then looks endometrial because the output is endometrial. [The framework is not saying the microscope is wrong. It is saying the microscope sees the endpoint and not the programmer.]

Direct supporting evidence has begun to emerge. Talwar et al. (2025) analyzed stool samples from women with endometriosis and identified altered fungal communities with changed fungal-bacterial interaction patterns. In a mouse endometriosis model, fungal depletion significantly impeded lesion progression. This is the first published work to move mycobiome involvement in endometriosis from speculation to intervention-responsive experimental finding. Earlier work by Gazvani et al. (2013) had

reported that C. albicans stimulates proliferation in endometrial cells from women with endometriosis (both with and without estradiol) while producing the opposite effect in cells from controls, demonstrating a differential host-interaction signature at the cellular level consistent with the framework's predictions about organism-directed tissue recruitment.

Parallel bacterial-microbiome work has documented distinct community signatures across anatomical sites in endometriosis patients. Hernandes et al. (2020) performed 16S rRNA sequencing on deep endometriotic lesions, eutopic endometrium, and vaginal fluid from the same patients, revealing that lesion-associated microbial profiles differ from eutopic endometrium rather than simply reflecting contamination or displaced tissue. This is precisely the site-specific microbial signature the framework predicts if colonized ectopic sites are mechanistically distinct from the uterine cavity rather than merely displaced copies of it.

This model also explains why pelvic, peritoneal, and bowel surfaces are privileged locations. They are not just the places fluid can reach. They are surfaces with established organism access, interstitial continuity, immune negotiation, and recurring exposure to menstrual substrate. The bowel association becomes especially important. The IBS paper already argues that gut management can be frozen in a single operating state through organism control of motility, barrier tone, prostaglandin signaling, immune skew, and sensory sensitivity (Craddock, IBS Stuck State). Endometriosis adds a neighboring reproductive program on top of the same geography. Under this framework, comorbidity reflects shared colonization terrain, not a mysterious overlap between two otherwise unrelated disorders.

Hormonal dependence fits cleanly within the same structure. Conventional medicine often treats response to hormonal suppression as proof that endometriosis is fundamentally a hormonal disease. Under this framework, hormonal suppression works because it interrupts the recurring substrate pulse and dampens the reproductive signal the organism is using. Remove ovulatory cycling, flatten progesterone withdrawal, reduce tissue breakdown, and the recruited ectopic program loses the monthly feed that maintains it. The success of suppression therefore does not disprove organism involvement. It is exactly what this model predicts.

Immune dysregulation likewise shifts from descriptive accompaniment to necessary infrastructure. A recruited ectopic site must avoid clearance, maintain inflammatory permissiveness, and keep rebuilding rather than resolving. Jeljeli et al. (2020) documented that macrophage immune memory controls endometriosis progression in mice, with innate memory and M2 polarization producing a permissive peritoneal microenvironment sustained across cycles. Yang et al. (2017) demonstrated that the crosstalk between endometrial stromal cells and macrophages impairs NK cell cytotoxicity via IL-10 and TGF-beta. These are exactly the signaling outputs Paper A catalogs as organism-available tools. Endometriosis under this model is not simply tissue in the wrong place. It is tissue being actively maintained in the wrong place by a resident system capable of reading the cycle, harvesting the substrate, and preserving a local program over time.

Surgery and recurrence also become easier to parse. If excision removes a site that includes the colonized niche sustaining the recruited program, improvement may be substantial. If residual colonized surfaces remain, recurrence is not surprising. The framework therefore predicts that recurrence patterns will track completeness of colonized-site removal more closely than they track abstract concepts of clean margins defined without organism assessment. [The tissue may be the visible fire. The colony is the pilot light.]

## 6. Predictions

1. Endometriotic implant tissue should show higher Candida-associated mycobiome signal than matched eutopic endometrium, especially at bowel-adjacent and peritoneal sites. [Evidence class:

tissue mycobiome sequencing comparison. Partial validation already available in Talwar et al. (2025) for stool mycobiome; lesion-tissue mycobiome sequencing remains the next step.]

2. Antifungal intervention should produce symptom improvement exceeding placebo in a framework-positive subgroup, with effect size tracking baseline dysbiosis or mycobiome burden. [Evidence class: pre-registered randomized controlled trial. Animal-model support from Talwar et al. (2025) where fungal depletion impeded lesion progression.]
3. Framework-positive phenotype markers, especially gastrointestinal comorbidity patterns and reproductive-cycle-linked bowel symptoms, should enrich for antifungal responders. [Evidence class: adaptive-enrichment trial design. IBS comorbidity rates established by Nabi et al. (2022) at 23.4% pooled prevalence and DiVasta et al. (2021) at OR 5.26 in adolescents provide the stratification basis.]
4. Endometriosis prevalence and severity should correlate with prior antibiotic exposure more strongly than models centered only on anatomy would predict. [Evidence class: retrospective cohort analysis.]
5. Pregnancy-associated improvement or remission should correlate with shifts in organism-activity markers, not only with host hormonal levels. [Evidence class: prospective serial mycobiome sampling.]
6. Surgical recurrence should correlate with residual colonized-site persistence and should be lower when excision removes the full geographic network supporting the ectopic program. [Evidence class: prospective surgical outcome study with mycobiome covariates.]

## 7. Discussion

The strongest feature of this seed is not that it explains one oddity of endometriosis. It explains several at once. It preserves the reality of cyclicity, the importance of hormonal suppression, the visible histology of implants, the bowel association, and the recurrence problem, while relocating the causal center from passive misplaced tissue to active organism-directed maintenance. That shift matters because chronic conditions are often hardest to explain at the point of persistence. Establishment can be imagined many ways. The question is why the state stays on.

The bowel-endometriosis comorbidity quantification strengthens the framework reading substantially. Nabi et al. (2022) pooled IBS prevalence in endometriosis cohorts at 23.4% (95% CI 9.7-37.2), with odds ratios for IBS in endometriosis patients of 2.97-3.26 relative to controls. DiVasta et al. (2021) found 24% IBS comorbidity in adolescents with endometriosis versus 7.1% in controls, an odds ratio of 5.26 that rises with pain severity. These are not modest associations. They are the kind of comorbidity magnitude that demands a shared mechanism rather than coincidence, and the adolescent data in particular rules out explanations that depend on years of accumulated disease burden or treatment exposure. Under this framework, the two conditions share colonization geography and reflect the same organism-host governance architecture expressed across neighboring compartments.

This model also aligns endometriosis more naturally with the Stuck State series. IBS is already positioned as gut management frozen in one mode. Endometriosis can be read as reproductive tissue recruitment frozen at ectopic sites. Both become local expressions of the same higher architecture: the organism reads a recurring input, runs a useful program phase, then fails to transition out when the modern host context does not deliver the expected completion conditions. Under that reading, comorbidity is structural, not incidental. The same patient is not unlucky enough to have two enigmatic conditions. She may be manifesting one distributed management error across adjacent compartments.

The framework also creates a practical research consequence. Mycobiome measurement should not be an afterthought in endometriosis cohorts. It should be built in as a covariate from the start. The Umbrella Paper makes the same recommendation across the stuck-state series: severity, treatment response, and relapse risk are expected to track organism density or distribution once researchers actually look for it (Craddock, Stuck-State Umbrella). The Talwar et al. (2025) stool mycobiome finding and the Hernandes et al. (2020) site-specific bacterial profiling demonstrate that this measurement is feasible with existing methods. What remains is to extend the sequencing to lesion tissue directly and to correlate mycobiome signatures with clinical outcomes across surgical, hormonal, and investigational antifungal interventions.

There is also a useful disciplinary consequence. Modern medicine often separates gynecology from gastroenterology and both from mycology unless an overt invasive infection is underway. This framework does the opposite. It predicts that the important biology sits exactly at those borders: bowel-adjacent reproductive tissue, interstitial routing, cyclical hormonal signaling, immune negotiation, and non-bloodstream microbial governance. If that is correct, then a large share of the condition's confusion comes not from its complexity alone but from the way medicine partitions its own fields.

# 8. Counter-Arguments Addressed

## Sampson's retrograde menstruation theory is established.

Response: the framework does not reject retrograde menstruation. It reframes its role. Retrograde flow can be the repeated delivery event without being the full explanation for chronic ectopic establishment. The key unresolved issue is why common exposure yields persistent disease in a minority of cases and why specific extra-uterine sites become stable program locations. The framework's answer is that site selection and program persistence are organism-mediated rather than flow-mediated, and that prediction is testable.

## Hormonal suppression is effective, proving endometriosis is primarily hormonal.

Response: hormonal suppression is expected to help if cyclic hormone withdrawal is the substrate pulse the organism uses. The framework reads hormonal efficacy as compatible evidence, not contradictory evidence. Flatten the cycle and the recruited program loses its monthly feed signal. The Elaris EM-I/II trials (Taylor et al., 2017) documented clinically meaningful pain relief with elagolix at NNT 2 for dysmenorrhea and NNT 5 for non-menstrual pain; the framework reading is that the GnRH antagonist removes the monthly organism-substrate event, not that it treats a purely host-side hormonal abnormality.

## Surgery is curative for many patients, so an organism-directed model is unnecessary.

Response: surgery can be effective under this model for the same reason it is effective under tissue-centered models: it removes the local infrastructure sustaining symptoms. The difference is in how recurrence is interpreted. The framework predicts recurrence will track persistence of colonized supporting geography rather than tissue visibility alone. Post-operative hormonal suppression reduces recurrence (pooled RR 0.41 in systematic reviews); the framework reading is that suppression continues to disrupt the monthly substrate pulse, extending the benefit of the surgical removal.

## Histology shows endometrium, which argues for displaced endometrium rather than recruited local tissue.

Response: the framework does not claim implants are non-endometrial once established. It claims the organism can recruit local tissue into endometrial behavior under repeated cyclical signaling and substrate

exposure. The microscope sees the mature output state. The framework addresses how that state is induced and maintained outside the uterus. The Gazvani et al. (2013) finding that C. albicans produces differential proliferation effects on endometrial cells from affected versus unaffected women supports the reading that the cellular response to organism signaling is not uniform across hosts; affected tissue is already in a different responsiveness state.

## 9. Conclusion

Under this framework, endometriosis is best understood not as passive tissue stranded in the wrong place but as a stuck reproductive program running at extra-uterine sites where Candida albicans already has positional authority. Menstrual cyclicity matters because it supplies the recurring substrate pulse. Hormonal suppression matters because it dampens that pulse. Gastrointestinal comorbidity matters because the bowel is not next door by accident; it is part of the same geography.

The value of the model is not rhetorical novelty. It is explanatory compression. One mechanism accounts for chronicity, cyclicity, bowel overlap, immune persistence, and recurrence after incomplete removal. That is enough to justify direct testing. If implant mycobiome signal is absent, if antifungal response does not enrich in framework-positive patients, and if recurrence ignores colonized-site persistence, the model fails. If those predictions hold, the field has been missing the management layer.

The Talwar et al. (2025) finding that fungal depletion impedes endometriosis lesion progression in a mouse model provides the first intervention-responsive experimental support for the framework's central claim. Tissue-level mycobiome sequencing, framework-stratified antifungal trials, and surgical outcome correlation with colonized-site persistence are now feasible next steps. This is precisely the kind of condition the Stuck State series was built to confront: common, chronic, clinically real, partially manageable, and still not mechanistically closed. The next step is straightforward. Measure the organism where the tissue is.

## 10. Closing Declaration

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