Redacted Science — Research Roadmap

Jim Craddock | April 8, 2026

This document outlines the remaining research program for the Redacted Science framework. The foundational papers are published and citable:

- **Paper A** — *Candida albicans as a Biochemical Computer*: https://doi.org/10.5281/zenodo.19337525
- **Paper B** — *The Saline Oscillation Hypothesis*: https://doi.org/10.5281/zenodo.19369715
- **Exposé** — *"They Looked. They Were Right. They Were Destroyed."*: https://doi.org/10.5281/zenodo.19393803
- **Focal Infections 2.0**: https://doi.org/10.5281/zenodo.19423069

What follows is the work that remains. Each item builds on the published foundation. Anyone continuing this work should read Paper A first — it establishes the core architecture. Paper B extends that architecture across evolutionary time.

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1. Christoph Thaiss Outreach

Thaiss (Arc Institute / Stanford) published work showing *Parabacteroides goldsteinii* produces medium-chain fatty acids that activate GPR84 on the vagus nerve, causing memory loss. Under the Redacted Science framework, this is not an independent finding — it is a downstream consequence of *C. albicans* managing its gut microbiome toolkit. The organism curates its bacterial environment. Thaiss's data fits directly into the framework's prediction that the symbiont manages host cognition through intermediary microbial populations. An email under 300 words pointing to both Zenodo DOIs is drafted and ready to send.

2. Outreach to Brian Hie (completed)

Hie's Evo 2 model at the Arc Institute is capable of analyzing the approximately 1,300 orphan genes in *C. albicans* — genes with no known homologs. Paper A Prediction 1 states these genes encode the organism's computational architecture. This email has been sent.

3. First and Second Phase Transition Case Studies

The framework describes three distinct transition phases in the organism's long-term host management program. The third transition is partially documented. Case studies for the first and second transitions need to be written, drawing on approximately 30 years of longitudinal personal data. These transitions are observable in retrospect as

coordinated shifts in metabolism, cognition, cravings, and physiological markers — not random symptom clusters.

4. Third Transition Case Study Extension — Urinary Salts

During the third transition, urinary salt composition changes detectably. The extension investigates why routine urinary salt measurement was abandoned in clinical practice. If the organism's metabolic program produces a characteristic salt signature at each phase, dropping that measurement from standard panels eliminated one of the simplest diagnostic windows into colonization status.

5. Tearing Up Without Warning — Mechanism

The framework predicts that the organism's management of the pituitary and downstream hormonal cascades can produce involuntary lacrimation — tearing up without emotional cause. This item investigates the specific mechanism: which hormonal or neurotransmitter pathway, when perturbed by organism activity, lowers the threshold for tear production? This is a clinically observable phenomenon that patients report and physicians currently have no explanation for.

6. Pituitary Cluster — Microadenoma, Breakoff, and Colonization Model

This is the centerpiece. The sella turcica sits outside the blood-brain barrier. The framework proposes that *C. albicans* conducts directed hyphal transit to the pituitary, colonizes it, and builds a microadenoma as its management interface. From this position, it modulates ACTH, cortisol, ADH, and MSH output.

Two modes of pituitary transition are proposed: governance (ongoing managed modulation) and terminal consumption (crisis mobilization — "damn the torpedoes"). The critical clinical question is what happens when a microadenoma breaks off. Under the framework, this is the dividing line between *Homo candidus* (symbiont-managed) and *Homo sapiens* (unmanaged or post-breakoff). The difference should be clinically testable.

Open gaps: the intermediary pathway from systemic electrolyte changes to synaptic Mg^{2+} concentration at NMDA gates; venous anatomy mapping for hyphal transit routes; and *C. albicans* vascular tropism literature.

The silent synapse connection (Harnett lab 2022, cAMP activation route 2024) provides the CNS access pathway — the organism can activate silent synapses via cAMP without requiring direct neural contact.

7. Esophageal Shutdown Mechanism — Case Study

In November 2022, a documented event sequence occurred: localized variceal inflammation (PGE_2 + mast cell degranulation + candidalysin) caused swallowing to stop. The gut emptied. The intestinal wall became the primary substrate. Inflammation resolved. An EGD found nothing. Each step in this sequence uses capabilities documented in Paper A §5. This case study pairs with a substrate progression table mapping the organ-by-organ failure sequence from longitudinal records.

8. Melanin and Pigmentation — Paper C

POMC is cleaved into ACTH and MSH at the pituitary. If the organism drives pituitary overdrive, MSH elevation is a byproduct — producing darker pigmentation independent of UV exposure. The organism also drives melanin locally via PGE_2 and TLR4 melanocyte activation (Tapia 2014, already in Paper A §5.5e). This explains the ER phenomenon of a patient in crisis presenting with "good coloring" — the pigmentation masks the underlying state.

Population-level prediction: colonization density correlates with pigmentation beyond what UV exposure models predict. The saline oscillation hypothesis adds another pigmentation dimension across evolutionary time.

9. Document D — Common Substances and the Biochemical Computer

How common environmental and dietary substances interact with the organism's computational architecture. First substance: glyphosate, which disrupts the shikimate pathway — a pathway *C. albicans* uses for tryptophan and substrate supply. Paired with dicamba, a synthetic auxin mimic that may trigger hyphal transition and quorum sensing at gut concentrations. Additional substances to be identified.

10. Homo Candidus Timeline — "Why This Matters"

The summary document for anyone asking "so what?" Contents: (1) grouped list of top current medical comorbidities that potentially resolve under the framework, organized by category (bacterial infections, autoimmune, metabolic, neurological); (2) estimated current spending per category; (3) mortality data; (4) risks; (5) how medicine would fundamentally change. This is the document that translates the science into policy and clinical implications.

11. Chat with the Compiler — Homo Candidus

A new conversational-format document in the style of the existing "Chat with the Compiler" series. This one focuses specifically on the *Homo candidus* designation — what it means, why it is proposed, and what distinguishes *H. candidus* from *H. sapiens* at the physiological, evolutionary, and clinical level. The conversational format makes the material accessible to non-specialists while preserving technical precision. [Done]

12. Paper A v1.1 — Queued Additions

Four mechanism sections to be added to Paper A: (1) tryptophan/kynurenine rerouting — the organism diverts tryptophan away from serotonin synthesis into the kynurenine pathway; (2) mast cell degranulation via mannan and β-glucan recognition; (3) dopaminergic interface via Gpr1 and clozapine connection; (4) multi-timescale epigenetic architecture — how the organism operates across different temporal scales simultaneously.

13. Paper A v1.2 — Predictions 11 and 12

Two additional testable predictions: (1) VOC (volatile organic compound) output profiling — the organism should produce a characteristic VOC signature detectable in breath or skin emissions; (2) glucose bolus subroutine termination — a controlled glucose load should produce a measurable interruption in the organism's ongoing metabolic subroutine, observable in real-time glucose monitoring data.

14. Paper B — Queued Additions

Six additions to the evolutionary timeline paper: (1) dysentery resistance — managed gut barrier decreases susceptibility, enabling higher population density and social structure; (2) smoking vs. consuming — pulmonary vs. gut delivery changes organism sensing response, ties to fire use timeline; (3) GPR84 MCFA receptor and Thaiss connection; (4) gut microbiome as organism toolkit (B vitamin biosynthesis deficits, SCFA/polyamine reduction, mucus layer degradation, Parkinson's connection); (5) circadian clock loss — *C. albicans* shed autonomous clock genes and reads host rhythms instead; (6) Rogers et al. 2026 two-superarchaic hominin model integration (98% bootstrap support, 19.6% admixture, ~1.3 Ma divergence).

15. What Makes an Ideal Symbiont — Genetic Perspective

[Claude had the wrong idea here. The point is the ideal symbiont is one that kills you when your genetics make you weaker. You both do this. If it gets weaker, you die from Cancer or some other thing. If you get weaker, it kills you. The ideal combination works together

16. DSHEA (1994) — The Regulatory Wall

The Dietary Supplement Health and Education Act of 1994 created a structural barrier between the framework's implications and clinical practice. If the correct intervention is nutritional or metabolic — caloric loading, substrate manipulation, dietary compounds — then physicians literally cannot prescribe it. Doctors prescribe medicines. DSHEA ensures that anything not classified as a medicine stays on the supplement shelf regardless of evidence.

Example: Lemon balm (*Melissa officinalis*). Documented antifungal properties directly relevant to the framework. Also a clinically effective anxiolytic (comparable to benzodiazepines in trials) and an acetylcholinesterase inhibitor (blocks the enzyme that degrades acetylcholine — the same mechanism targeted by Alzheimer's drugs). One plant, three major clinical applications. In France, pharmacies carry it as a treatment. In the United States, it sits in the supplement aisle because of a law passed in 1994. This is not a conspiracy — it is regulatory architecture, and it is a concrete obstacle on the path from framework to clinical application.

Scoring Methodology (retained for future use)

Importance Score (1–10): How much does this advance C. albicans-as-symbiont and H. candidus with scientists?

Criterion	Weight
Testable predictions generated	25%
Mechanistic gap closed	20%
Framework differentiation	20%
Evidence grounding	20%
Cascade potential	15%

Difficulty Score (1–10): How hard to execute given current constraints?

Criterion	Weight
Research depth required	25%
Writing complexity	25%
Estimated sessions	20%
Dependencies	15%
Phone-friendliness	15%

Current Scores

#	Item	Importance	Difficulty
1	Thaiss outreach	7	2
3	First/second transition case studies	8	5
4	Third transition — urinary salts	6	4
5	Tearing up mechanism	5	5
6	Pituitary cluster (microadenoma + colonization)	9	8
7	Esophageal shutdown case study	7	4
8	Melanin/pigmentation (Paper C)	7	6
9	Document D — substances	6	5
10	"Why This Matters" timeline	8	6
11	Chat with Compiler — Homo candidus	7	4
12	Paper A v1.1 additions	8	6
13	Predictions 11 & 12 (v1.2)	6	3
14	Paper B queued additions	8	7
15	Ideal symbiont — genetic perspective	9	7
16	DSHEA (1994) + lemon balm	7	3

This document is a continuity safeguard. The published papers stand on their own. This roadmap is for anyone picking up where I left off.

— Jim Craddock, Tulsa, Oklahoma