Redacted Science: The Pan-Mammalian Coevolution Hypothesis

A Further Refinement of the Coevolution Framework

Jim Craddock | Redacted Science Research Initiative | March 2026, revised June 2026 V4

redactedscience.org | jimcraddock.com

Abstract

The Redacted Science framework (Craddock, *Redacted Science*) originally proposed that modern humans coevolved with a fungal symbiont, specifically *Candida*, producing a physiologically distinct phenotype designated *Homo candidus*. This paper extends that framing. The conserved mammalian interface systems through which the symbiont operates, including the hypothalamic-pituitary axis, the endocannabinoid system, sodium-potassium pump dynamics, renal concentrating mechanisms, and phagocytic immune pathways, are not uniquely human. They are shared across all land mammals and have been preserved across approximately 200 million years of mammalian evolution. Recent comparative mycobiome studies document that each mammalian lineage carries its own host-adapted yeast symbiont from the Saccharomycotina subphylum: *C. albicans* in humans, *C. natalensis* in dogs, *C. tropicalis* in mice, *Kazachstania slooffiae* (the teleomorphic state of *Candida slooffiae*) in pigs. These symbionts span multiple yeast families within the Saccharomycotina with the Debaryomycetaceae/Metschnikowiaceae (CTG-Ser clade) and the Saccharomycetaceae among them. The conserved feature across the mammalian clade is the interface architecture, not the identity of any specific yeast lineage. The framework's claim is therefore stronger than a single-lineage adaptation story: partnership architecture has emerged convergently across multiple yeast families partnered with different mammalian hosts, indicating that the architecture is selected for at the mammalian-yeast interface level rather than at any specific yeast lineage level. This paper therefore proposes that the coevolution did not originate with humans but with the mammalian body plan itself, and designates the full clade *Mammalia candidus*, with the specific organism varying by host lineage. *Homo candidus* is a subset of that broader category, distinguished not by the presence of the symbiosis but by the capacity for conscious, volitional participation in its physiological program. The Pan-Mammalian Coevolution hypothesis is argued to be complementary to the Methylation/Neural Selection hypothesis, describing respectively the structural and runtime layers of the same symbiotic architecture. The paper also addresses the detection variability problem documented across mammalian mycobiome surveys, arguing that conventional detection rates reflect methodology limits rather than true absence. Implications for human and veterinary medicine, mycobiome research, and the framing of idiopathic disease are discussed. A specific environmental mechanism for this coevolution is developed in the companion paper on the Saline Oscillation Hypothesis (Craddock, Saline Oscillation).

Keywords: *Candida albicans; coevolution; fungal-mammalian symbiosis; mycobiome; endocannabinoid system; Mammalia candidus; Homo candidus; hypothalamic-pituitary axis; evolutionary medicine; Redacted Science Research Initiative; #TheArchitect*

This article represents a further refinement of the theoretical framework first presented in *Redacted Science*. It does not invalidate prior theory. It extends it. The Methylation/Neural Selection hypothesis and the Pan-Mammalian Coevolution hypothesis are complementary. They describe different layers of the same architecture.

I. The Starting Point: Homo Candidus

The original Redacted Science framework (Craddock, *Redacted Science*) proposed that modern humans, *Homo sapiens*, coevolved with a fungal symbiont, specifically *Candida*, over tens of thousands of years. The condition documented in the redacted 1995 medical article described this symbiosis as active, physiologically transformative, and capable of producing what the paper termed "upgrades": enhanced cognition, pain tolerance modulation, endurance extension, and systematic metabolic optimization.

The author of this document is a 30-year case study. He is the evidence. The framework was built from the inside out: not from hypothesis to experiment, but from survival to reverse-engineered mechanism. The redaction of the source article from medical literature is itself documented (Craddock, *Expose*). The archive exists across decentralized platforms specifically to prevent re-redaction.

Homo candidus was the working designation: humans carrying the active symbiosis, distinguished from the general population by physiological profile rather than genetics. The condition was framed as human-specific. This article refines that framing.

II. The Refinement: Why Humans Only?

The question that drives this refinement is simple: if the symbiosis exists, why would it be limited to humans?

The mammalian body plan is extraordinarily conserved. Across all land mammals, from rodents to great apes to humans, the following systems are present in nearly identical form:

- The hypothalamic-pituitary axis, the central regulatory architecture of hormonal control
- The endocannabinoid system, the primary modulator of pain, appetite, memory, and immune function
- The sodium-potassium pump dynamics governing cellular energy
- Similar renal concentrating mechanisms and ADH regulation
- Comparable phagocytic immune modulation pathways

These systems are the precise interface points documented in the *Redacted Science* framework. The symbiont operates through the endocannabinoid system. It modulates via the pituitary axis. It exploits cellular energy dynamics through the pump. The substrate is not uniquely human. It is mammalian.

The hypothesis therefore extends: the coevolution did not originate with humans. It originated with the mammalian body plan itself. Homo candidus is a subset of a broader category. The working designation for the full clade: *Mammalia candidus*.

This designation makes a specific empirical prediction. If the coevolution operated at the level of the mammalian body plan, each mammalian lineage should carry its own host-adapted fungal partner, with the architecture conserved and the specific organism varying. The recent mycobiome literature documents exactly this pattern.

Humans carry *Candida albicans*, the most thoroughly characterized example, vertically transmitted from mother to child with no known environmental reservoir (Kumamoto, 2011; Day and Kumamoto, 2023). Dogs carry *Candida natalensis* as their dominant gut fungal commensal, with *Candida* identified as the most abundant fungal genus in canine fecal mycobiome surveys (Foster et al., 2013). Mice are not naturally colonized by *C. albicans*; they carry *Candida tropicalis* and other host-specific yeasts as their dominant gut fungal partners, with *C. albicans* requiring antibiotic pretreatment, neonatal status, or germ-free conditions to establish in murine gut. Pigs carry *Kazachstania slooffiae*, the teleomorphic (sexual reproductive) state of *Candida slooffiae*, present universally in production swine across geographic locations and production systems (Summers et al., 2019; Arfken et al., 2020). Cats carry a mixed *Candida* / *Saccharomyces* / *Malassezia* commensal community with regional variation. Hindgut fermenters such as horses have shifted to anaerobic fungi (Neocallimastigomycota) for their dominant fungal partner, reflecting their dietary ecology, but still carry *Candida* species as minor members.

Each lineage carries its own. The architecture is conserved across the mammalian clade. The specific organism reflects the host's ecology, immune architecture, and evolutionary history. *Mammalia candidus* therefore designates the architectural pattern of mammalian-fungal coevolution, not a literal claim that every mammal carries the same organism. *Homo candidus* is the human-specific instance. The framework predicts and the evidence confirms that other mammalian lineages have their own host-specific instances, with the family-level commonality (Saccharomycotina yeasts, or functionally equivalent fungi in specialized lineages) preserved across most of the clade.

III. Symbiosis as Evolutionary Advantage

This is not a fringe proposition. We already accept it without question for bacteria. The gut microbiome is essential for digestion, immune calibration, and neurotransmitter production. Mitochondria, the energy engines of every eukaryotic cell, are almost certainly the descendants of bacterial endosymbionts so thoroughly integrated that we die in seconds without them. The human body is already an ecosystem, not a standalone organism.

The question is not “why would fungi be symbiotic partners?” The question is: “why have we assumed they are not?”

Organisms with well-integrated symbionts are more resilient. (Craddock, Biochemical Computer) They carry:

- Redundant metabolic pathways beyond their own genome
- Expanded biochemical capability through the partner's chemistry
- External intelligence optimizing the internal environment
- Adaptive capacity that outpaces single-organism evolution

Natural selection favors successful symbiosis. The most resilient lineages are the ones that integrated the most capable partners. Bacteria enabled the transition to complex cellular life. Fungi (biochemical computers with memory, adaptation, multi-form expression, and zero time preference) may have enabled the transition to the mammalian clade as we know it.

The conserved mammalian architecture is not coincidental. It may have been selected for precisely because it interfaced optimally with the symbiont. The pituitary axis, the endocannabinoid system, the renal and immune dynamics: these are not just shared features of a common ancestor. They may be the interface layer, preserved across 200 million years of mammalian evolution because they work.

This raises a question the framework must address directly. If the symbiosis is universal across mammals, why do molecular surveys frequently report "no *Candida* detected" in a substantial fraction of individuals? In humans, conventional stool PCR and ITS sequencing typically detect *C. albicans* in 40 to 70 percent of healthy individuals. The remainder appear *Candida*-free.

The cleanest evidence that this is a detection problem rather than true absence comes from veterinary work. The Foster canine mycobiome study found that *Candida* sequence prevalence in healthy dogs ranged from 0.0 percent to 63.1 percent, with a median of 5.2 percent (Foster et al., 2013). Some healthy dogs registered as fully *Candida*-free by molecular sequencing. At the same time, when the sequence data was examined at the species level, Candida natalensis was identified in 10 of 12 healthy dogs at greater than 99 percent similarity to the reference sequence — a near-universal positivity that the genus-level abundance summary obscures. The same dataset produces apparent absence at the genus level and near-universal presence at the species level, depending on which methodology aspect is foregrounded. Dogs are presumably more uniformly colonized than humans, sharing food bowls, grooming littermates, and being raised in close family contact. The variability is not a biological gradient. It is a detection artifact. Whatever produces "no *Candida*" readings in some healthy dogs produces the same readings in some healthy humans.

Three biological features make true absence implausible in humans. *C. albicans* has no known environmental reservoir. It is acquired vertically from mother to child during birth canal transit and breastfeeding, and horizontally through close contact (Kumamoto, 2011; Day and Kumamoto, 2023). For 20 to 60 percent of adults to be genuinely *Candida*-free, that fraction would need to have escaped vaginal birth, breastfeeding, family contact, and intimate contact with carriers across the entire population. This is not the population that exists.

Standard mycobiome surveys also sample the wrong locations. Stool samples reflect the gut lumen at a single moment. The organism's documented niches include skin, oral mucosa, vaginal and penile mucosa, deep gut mucosa rather than lumen, the interstitium (Benias et al., 2018), and tissue compartments such as the perisellar venous network. Single-time-point fecal sampling

misses most of these. Direct comparison of jejunum lumen versus fecal sampling in the same animals (Mentula et al., 2005) found 27 percent prevalence in jejunum versus 5 percent in feces — a five-fold difference produced by sample site selection alone.

Detection threshold biology compounds the problem. PCR and ITS sequencing have minimum detection limits. Below those limits, an organism in biofilm dormancy, deep tissue residence, or low-abundance commensal mode at a given site reads as absent. Yeast-form quiescent populations and tissue-embedded hyphal networks are systematically underdetected by methods optimized for active luminal yeasts.

The defensible position is therefore that conventional detection rates of 40 to 70 percent in humans, and the matching variability seen in dogs and other mammals, reflect detection methodology limits more than true colonization status. The symbiont's biology and the cross-species pattern of host-specific Saccharomycetaceae symbiosis make near-ubiquitous mammalian carriage the prior expectation. Apparent absence is more parsimoniously explained as undersampled or under-detected presence. The organism is not missing. It is hiding, in the technical sense that available molecular tools see only a fraction of where it actually lives.

IV. The Human Distinction: Volitional Participation

If all land mammals carry this symbiosis, what distinguishes humans? Not the symbiosis itself. The capacity for conscious intervention in the physiological process.

The Redacted Science case study documents a specific behavior during Phase 1 onset (the polyuria phase) where volitional acts, bearing down to urinate, and later consciously choosing not to urinate during the onset crisis, appear to have triggered the pituitary override documented in the source article (Craddock, Terminal Onset DI). No other mammal performs this level of conscious, intentional intervention in a physiological crisis. No other mammal could read a medical article, recognize its own condition, replicate the described treatment, or teach their offspring that they must do these things in order to survive an illness.

The evolutionary distinction of Homo sapiens within the *Mammalia candidus* clade is not that we carry a unique symbiosis. It is that we are the branch capable of consciously participating in the process. We can know what is happening and act on that knowledge. That capacity, not the symbiosis itself, is what makes the human case study possible and what makes the suppression of this knowledge so consequential.

V. Compatibility with the Methylation/Neural Selection Hypothesis

The Neural Selection hypothesis, as previously articulated in *Redacted Science*, describes a parallel evolutionary mechanism: populations that adapt epigenetically through ligand exposure (cannabinoids, terpenes, fungal compounds) train their gene expression in real time. Unlike mutation-driven selection, methylation-based adaptation is rapid, heritable, and reinforcement-based. It is evolution running on a feedback loop rather than waiting for random nucleotide events.

The key passage:

This isn't natural selection. It's neural selection: biochemical intelligence embedded in host systems, updating on a loop. It is an evolutionary war in motion.

These two frameworks, Pan-Mammalian Coevolution and Methylation/Neural Selection, are not competing theories. They describe different layers of the same system:

- The Coevolution hypothesis describes the structural layer: the symbiont integrated into mammalian physiology over geological time, preserved through conserved interface systems.
- The Methylation hypothesis describes the runtime layer: the symbiont's ligands and compounds actively tuning host gene expression in real time, accelerating adaptation within individual lifespans and passing it forward.

The symbiont built the interface (Coevolution). The symbiont runs the interface (Methylation). Both are true. Both are necessary for the full picture.

The cannabinoid flywheel described in the Neural Selection hypothesis (ligand exposure driving methylation driving reinforced adaptation driving further exposure) is precisely what you would expect from a symbiont that has been optimizing its host for 200 million years. It built the endocannabinoid system. Of course it knows how to run it.

VI. Implications for Medicine

If the Pan-Mammalian Coevolution hypothesis is correct, the blind spot in conventional medicine is not incidental. It is structural. The entire framework of idiopathic disease (depression, anxiety, autoimmune disorders, IBS, chronic fatigue, and dozens of other poorly-understood conditions) has been constructed without awareness of the symbiont. Symptoms are treated. The ecology is ignored (Craddock, *Implications*).

We do not spend significant research resources understanding how fungi interact with mammalian physiology. This is not because the question has been asked and found unimportant. It is because the question has largely not been asked. The historical suppression of integrative fungal-host research is documented separately (Craddock, *Focal Infections*; Craddock, *Hemodynamic Conpensation*). The mycobiome receives a fraction of the research attention devoted to the bacterial microbiome, despite fungi being orders of magnitude more biochemically complex.

The Redacted Science framework proposes that what medicine currently classifies as separate diseases are, in many cases, manifestations of ecological dysregulation: the symbiont present but unsupported, its signals misread as pathology, its chemistry suppressed by antifungals prescribed for unrelated complaints. The system is not broken. It is operating on an incomplete map.

The same blind spot extends to veterinary medicine. Dogs, cats, pigs, horses, and other domesticated species are treated for *Candida* overgrowth and fungal dysbiosis with the same antifungal categories used in humans. The reframing offered here applies to those species as well. Each carries its own coevolved fungal partner. Each interfaces with that partner through the same conserved mammalian architecture. Each is subject to the same iatrogenic risks when the partner

is suppressed without recognition of its role. Documented parallels are already visible: diabetic dogs show increased *Candida* colonization analogous to the pattern seen in human type 1 diabetes; cats with chronic inflammatory enteropathy show *Candida* overgrowth; piglet weaning produces the metabolic stress signatures that destabilize *Kazachstania slooffiae* partnership. The veterinary mycobiome literature is younger than the human literature and has more room to integrate the framework's predictions before the same century-long blind spot calcifies.

The detection problem documented in Section III also applies to veterinary medicine. A dog that tests *Candida*-negative on stool sequencing is not *Candida*-free. The clinical implications are the same as in humans: absence of detection is not absence of organism, and treatment decisions made on the basis of negative molecular surveys are operating on incomplete information. The healthy canine mycobiome typically contains multiple Candida species simultaneously (Foster et al., 2013), with C. natalensis dominant but other species including C. austromarina, C. zeylanoides, and C. neerlandica frequently coexisting. The partnership architecture supports parallel host-organism instances within a single mammalian lineage, consistent with the framework's claim that what is conserved is the architecture rather than any specific organism identity.

A specific environmental mechanism for the pan-mammalian coevolution proposed here, the cyclical salinity oscillations of the East African Rift Valley during the Plio-Pleistocene, is developed in the companion paper on the Saline Oscillation Hypothesis (Craddock, *Saline Oscillation*).

Closing Statement

This article was written in March 2026 and revised in May 2026, during the documented final stage of a 30-year iatrogenic progression. The author remains fully employed, documenting daily. The theory presented here was developed in real-time conversation with AI, the only system capable of engaging with the framework without institutional guardrails terminating the analysis.

The archive is distributed across 8+ platforms, anchored in the academic citation graph, and designed to be unredactable. If you are reading this after the author's death, the documentation mission succeeded. If you are reading this and the science has been recognized, the 30-year case study held.

Document, record, preserve. Time reveals all.

Jim Craddock
#TheArchitect
www.redactedscience.org

References

Craddock, J. (2025). *Redacted Science* (revised April 2026) [Book]. Retrieved from https://jimcraddock.com (Latest Book Version)

Craddock, J. (2026). *Candida albicans as Biochemical Computer: A Theoretical Framework.* Redacted Science Research Initiative. Zenodo. https://doi.org/10.5281/zenodo.19337525 [Cited as Biochemical Computer]

Craddock, J. (2026). *Phase 5 of a 31-Year Managed Symbiont Progression: A Longitudinal Case Study Interpreted Through the Biochemical Computer Framework.* Redacted Science Research Initiative. Zenodo. https://doi.org/10.5281/zenodo.19560800 [Cited as Phase 5]

Craddock, J. (2026). *The Saline Oscillation Hypothesis: Endocannabinoid-Mediated Fungal-Hominid Coevolution in the East African Rift Valley.* Redacted Science Research Initiative. Zenodo. https://doi.org/10.5281/zenodo.19369715 [Cited as Saline Oscillation]

Craddock, J. (2026). *They Looked. They Were Right. They Were Destroyed: A Historical Investigation of the 1960s UK Research Corridor and the 1965 Winner-Hurley Symposium.* Redacted Science Research Initiative. Zenodo. https://doi.org/10.5281/zenodo.19393803 [Cited as Exposé]

Craddock, J. (2026). *Focal Infections 2.0.* Redacted Science Research Initiative. Zenodo. https://doi.org/10.5281/zenodo.19423069 [Cited as Focal Infections]

Craddock, J. (2026). *Acute Hemodynamic Decompensation Following Routine Phlebotomy in a Patient with Chronic Volume Dysregulation: A Five-Week Longitudinal Provocation Sequence.* Redacted Science Research Initiative. Zenodo. https://doi.org/10.5281/zenodo.19462704 [Cite as Hemodynamic Compensation]

Craddock, J. (2026). *Implications of Recognizing C. albicans as Biochemical Computer.* Redacted Science Research Initiative. Zenodo. https://doi.org/10.5281/zenodo.19488041 [Cited as Implications]

Arfken, A. M., Frey, J. F., & Summers, K. L. (2020). Temporal dynamics of the gut bacteriome and mycobiome in the weanling pig. Microorganisms, 8(6), 868. https://doi.org/10.3390/microorganisms8060868

Benias, P. C., Wells, R. G., Sackey-Aboagye, B., Klavan, H., Reidy, J., Buonocore, D., Miranda, M., Kornacki, S., Wayne, M., Carr-Locke, D. L., & Theise, N. D. (2018). Structure and distribution of an unrecognized interstitium in human tissues. Scientific Reports, 8(1), 4947. https://doi.org/10.1038/s41598-018-23062-6

Day, A. W., & Kumamoto, C. A. (2023). Interplay between host and Candida albicans during commensal gut colonization. PLOS Pathogens, 19(9), e1011607. https://doi.org/10.1371/journal.ppat.1011607

Foster, M. L., Dowd, S. E., Stephenson, C., Steiner, J. M., & Suchodolski, J. S. (2013). Characterization of the fungal microbiome (mycobiome) in fecal samples from dogs. Veterinary Medicine International, 2013, 658373. https://doi.org/10.1155/2013/658373

Kumamoto, C. A. (2011). Inflammation and gastrointestinal Candida colonization. Current Opinion in Microbiology, 14(4), 386–391. https://doi.org/10.1016/j.mib.2011.06.003

Summers, K. L., Frey, J. F., Ramsay, T. G., & Arfken, A. M. (2019). The piglet mycobiome during the weaning transition: A pilot study. Journal of Animal Science, 97(7), 2889–2900. https://doi.org/10.1093/jas/skz182