# The Arachidonic Acid Branch Point: External Empirical Confirmation

## Architect Report AM#022 V1

Jim Craddock
Redacted Science Research Initiative
redactedscience.org | jimcraddock.com
20260502

## Abstract

This report consolidates external empirical support for the arachidonic acid branch point prediction established in Paper A. The Redacted Science framework treats *Candida albicans* as a biochemical computer operating through organism-host governance, with arachidonic acid functioning as a shared substrate node between endocannabinoid tone and prostaglandin output. AM#022 does not extend that theory. It asks whether external laboratories have now produced the expected two-arm signal. They have. Markey et al. showed that acute gastrointestinal colonization with *C. albicans* in adolescent female mice altered anxiety-like behavior and HPA-axis corticosterone dynamics on a two-to-three-day timeline, with the phenotype rescued by the FAAH inhibitor URB597, a specific amplifier of anandamide signaling (Markey et al., 2020). Kumamoto et al. then showed the other arm: *C. albicans* colonization elevated serum PGE2 metabolite, altered ethanol preference and ethanol-related behavior, shifted dorsal-striatal dopamine receptor transcription, and was reversed through PGE2 receptor antagonism while exogenous dimethyl-PGE2 reproduced the behavioral direction (Kumamoto et al., 2025). Chevalier et al. add pile-up evidence from a separate UCMS microbiota-transfer depression model, where FAAH/MAGL inhibition, direct dietary arachidonic acid supplementation, and Lactobacilli complementation independently rescued the eCB-mediated phenotype (Chevalier et al., 2020). Severance et al. add human ecological support: probiotic treatment reduced *C. albicans* IgG titers over fourteen weeks in males with schizophrenia, while the placebo *C. albicans*-positive group carried the greatest bowel difficulty burden (Severance et al., 2017). The consolidation claim is therefore narrow but important: two downstream arms predicted from one substrate-level mechanism have now been observed through independent experimental routes. The burden shifts to the incidental-perturbation interpretation, which must explain each arm, each rescue specificity, and each convergent ecological correction as separate coincidences.

## Keywords

Arachidonic acid; Endocannabinoid system; Prostaglandin E2; *Candida albicans;* FAAH; HPA Axis; Anandamide; MAGL; PGE2; Gut-brain axis; Mouse model; mycobiome; Biochemical computer; Redacted Science Research Initiative; Architect Report; #TheArchitect

## Introduction

The Redacted Science framework proposes that *C. albicans* is not merely a passenger in mammalian hosts. It is modeled as a coevolved biochemical computer that reads host state, competes for substrate, and produces outputs through conserved mammalian interfaces. Paper A establishes the organism-level architecture. The Saline Oscillation and Pan-Mammalian papers place that architecture in evolutionary context. The Stuck-State Umbrella shows why failures of phase transition can look, from the clinic, like unrelated chronic diseases (Craddock, Biochemical Computer; Craddock, Saline Oscillation; Craddock, Pan-Mammalian; Craddock, Stuck-State Umbrella).

AM#022 is the first Mechanism-series report. Its job is not to propose a new disease model. Its job is to anchor one external confirmation point so later papers can cite the consolidation rather than re-litigating the same evidence each time.

### Framework Basis

Paper A identifies the arachidonic acid branch point as a substrate-level interface where organism activity can route host lipid economy toward endocannabinoid tone or prostaglandin output. The relevant claim is not that every eCB or PGE2 change is fungal. The claim is that *C. albicans* has documented access to the shared substrate economy and therefore should produce paired downstream signatures when the organism-host system is perturbed at that point (Craddock, Biochemical Computer §4).

The Saline Oscillation paper matters because eCB tone is treated there as broad mammalian regulatory infrastructure, not a narrow neurological curiosity. The Pan-Mammalian paper matters because the key confirmation literature is mouse work. Under the framework, that is expected, not disqualifying (Craddock, Saline Oscillation; Craddock, Pan-Mammalian).

## 1. Frame

Mechanism reports serve a specific purpose inside the Redacted Science archive. They are not grand synthesis papers. They are consolidation blocks. Paper A predicted a two-arm effect at the arachidonic acid branch point: one arm through endocannabinoid tone, one arm through prostaglandin output. AM#022 evaluates whether external work now supports that prediction. It does. The value is structural. Once the external confirmation is consolidated here, future revisions of Paper A, Paper B, the Pan-Mammalian paper, and downstream Stuck-State entries can point to AM#022 rather than repeating the Markey, Kumamoto, Chevalier, and Severance discussion in full. This is how the AM series should work: isolate the mechanism, lock the empirical spine, state the asymmetry, define the failure condition, and move on.

## 2. The Two-Arm Prediction Recap

Paper A's arachidonic-acid branch point claim is simple enough to restate without rebuilding it. Arachidonic acid supplies substrate to eCB signaling and to prostaglandin synthesis. If *C. albicans* competes at this shared node, then organism activity should not create a random behavioral perturbation. It should create patterned downstream effects on both arms of the pathway. The eCB arm should appear as altered anandamide or 2-AG tone with behavioral or HPA-axis consequences. The prostaglandin arm should appear as altered PGE2-linked physiology with receptor-specific behavioral effects. The burden of this report is therefore not to prove every part of Paper A again. It is to ask whether independent external studies now show both predicted arms using specific rescue or reversal methods. That is the confirmation standard used here (Craddock, Biochemical Computer §4).

## 3. eCB arm: Markey 2020 treatment

Markey et al. supply the first arm of the external confirmation: gastrointestinal colonization with *C. albicans* can alter host behavior and HPA-axis output through endocannabinoid-system disruption, and the phenotype can be rescued by targeted FAAH inhibition rather than by a broad anti-inflammatory or nonspecific behavioral intervention (Markey et al., 2020). The model used adolescent female C57BL/6 mice, five weeks old, a cohort choice that matters because adolescence is a developmental window in which the HPA axis and eCB tone are especially sensitive to microbiota perturbation. Mice were cohoused for four days to normalize bacterial microbiota by coprophagy, then orally inoculated with 5 × 10^7 CFU of *C. albicans* strain CKY101 or mock-inoculated with buffer. Fecal pellets confirmed colonization at 24 hours, behavior was assessed at day two, and sacrifice occurred at day

three, with some mice subjected to restraint stress before terminal collection. The operative window was therefore tight: behavioral and endocrine effects appeared within approximately 48 hours of fungal colonization.

That timeline is important because the model did not require invasive disease. Markey et al. measured inflammatory cytokines in serum and found no systemic inflammatory disease signal. They also used 16S rRNA sequencing and QIIME analysis on cecal contents and found no significant bacterial microbiota disruption by taxon abundance, alpha diversity, or beta diversity. Under the framework, that negative result is not a side note. It is the absence of generic dysbiosis. If *C. albicans* colonization can produce eCB-linked neuroendocrine change while leaving the bacterial community broadly stable, the result fits an organism-interface mechanism better than a loose "the microbiome changed somehow" explanation. Paper A predicts a substrate-level interface at the arachidonic-acid branch point, not a requirement for bacterial collapse (Craddock, Biochemical Computer §4).

The behavioral data were similarly specific. In the elevated plus maze, colonized mice spent less time in open arms and made fewer open-arm entries, consistent with increased anxiety-like behavior. Total arm entries did not fall, indicating the effect was not simple locomotor suppression. The forced swim test provided a second control: colonized mice did not differ in immobility time, meaning the phenotype was not a generic sickness-coping or despair-like behavior. The behavioral signal was anxiety-like, not just "mouse feels bad."

The HPA-axis arm was measured directly. Markey et al. collected trunk blood after decapitation and measured corticosterone by ELISA. Colonized mice showed elevated basal CORT, including a circadian advance in afternoon CORT rise. After 30 minutes of restraint stress, mock and colonized mice reached comparable peak CORT, but colonized mice showed altered recovery, remaining higher after 60 minutes. The PVN remained stress-responsive by c-FOS assessment, so the signal was not a gross hypothalamic activation failure. It was regulation, timing, and recovery.

The endocannabinoidome measurements provide the load-bearing mechanism. Forebrain AEA was measured by mass spectrometry. Bulk AEA was not significantly lower across all colonized mice, but colonized mice showed a trend toward negative correlation between basal serum CORT and brain AEA, and k-means clustering separated a high-CORT colonized subgroup with significantly lower AEA. 2-AG did not show the same brain-CORT pattern. In the gut, untargeted metabolomics of cecal contents found enrichment in sterol and eCB-related compounds, including increased linoleoyl and linolenoyl ethanolamide and precursor-associated lipid changes that could shift N-acylethanolamide production away from AEA. In plain English, the gut lipid pool moved in the direction Paper A would expect: substrate allocation changed in a way that could reduce effective AEA tone.

The rescue experiment locked the mechanism. URB597, a FAAH inhibitor that increases AEA by blocking degradation, was administered intraperitoneally. At 1 mg/kg given four hours before sacrifice, URB597 reduced elevated basal CORT in colonized mice. At 0.1 to 0.15 mg/kg given four to six hours before elevated-plus-maze testing, URB597 normalized anxiety-like behavior without changing locomotor activity. The specificity matters. The intervention did not kill the organism, sedate the animal, broadly suppress inflammation, or reverse every possible behavioral endpoint. It amplified AEA-CB1 signaling and corrected the phenotype predicted to depend on that signaling.

For the framework, Markey 2020 is therefore not merely "fungus affects mouse anxiety." It is colonization without invasive disease, stable bacterial microbiota, altered gut and brain eCB lipid state, inverse AEA-CORT relationship in the affected subgroup, HPA-axis dysregulation, anxiety-like behavior, and FAAH-rescue specificity inside a 48-hour window. The governance reading is that *C. albicans* is not randomly perturbing the host. It is touching a calibrated host control surface, and the host responds as if a regulatory channel has been adjusted.

## 4. $PGE_2$ arm: Kumamoto follow-up 2025 treatment

The Kumamoto-lab follow-up supplies the second arm of the AA branch-point confirmation: *C. albicans* colonization alters ethanol behavior through elevated prostaglandin $E_2$ signaling, receptor-specific reversal, and downstream striatal dopamine-system changes (Kumamoto et al., 2025). The model again used C57BL/6 mice, primarily female mice because the colonization phenotype was stronger, orally inoculated with *C. albicans* strain CKY101 or PBS. In the central ethanol assay, single-housed mice were given continuous access to two bottles, one containing water and one containing 15% ethanol. Consumption was measured by bottle weight across two days. Colonized mice consumed less ethanol and showed lower ethanol preference, with fecal and cecal CFU confirming ongoing gastrointestinal colonization.

The saccharin control is essential. The investigators ran a parallel continuous-access two-bottle test using 0.0125% saccharin to determine whether colonization broadly suppressed reward preference or fluid consumption. Saccharin preference did not differ by colonization status. That control separates a general "less reward seeking" explanation from a more substrate-specific ethanol-behavior effect. Within this report's framework, that distinction matters because the $PGE_2$ arm is not proposed as a global pleasure switch. It is an arachidonic-acid-derived immune-neural branch that can alter specific behavioral valuation and aversion circuits.

$PGE_2$ was measured through its more stable serum metabolite, 13,14-dihydro-15-keto $PGE_2$, using an enzyme-linked immunosorbent assay after two days of ethanol consumption. Colonized mice showed elevated serum PGE metabolite levels compared with mock and water-only controls. To test whether elevated $PGE_2$ could itself reduce ethanol intake, the investigators injected uncolonized mice with the stable derivative 16,16-dimethyl $PGE_2$. Daily subcutaneous dm$PGE_2$ reduced ethanol consumption and preference in a dose-dependent pattern, with lower doses producing modest effects and higher doses producing stronger reductions. The dose-response relationship makes this more than correlation.

The receptor-reversal experiment provides the mechanistic lock. Colonized and mock-colonized mice received antagonists to EP1 and EP2, specifically SC-51089 and TG6-10-1, at 2 mg/kg each. In colonized mice, EP1/EP2 antagonism increased ethanol consumption and preference toward mock-colonized levels, while antagonist treatment did not materially increase ethanol intake in mock-colonized controls. This is the $PGE_2$ equivalent of the URB597 specificity in Markey: block the relevant branch receptor and the colonization phenotype moves back toward baseline. A generic perturbation account now has to explain why the phenotype routes through exactly the expected $PGE_2$ receptors.

The brain-region data sharpen the result. The investigators dissected dorsal striatum, prefrontal cortex, and nucleus accumbens and used RT-qPCR to measure receptor expression. Serum PGE metabolite correlated with Ep1 and Ep2 expression in the dorsal striatum of colonized mice, and similar Ep1 correlations were observed in PFC and NAc, showing that circulating $PGE_2$ state had central transcriptional correlates. The dopamine-receptor signal, however, localized more specifically. Drd2 was significantly reduced in the dorsal striatum after colonization, and low-dose dm$PGE_2$ reproduced reduced Drd2 expression. Drd expression did not show comparable changes in PFC or NAc, making the dorsal striatum the main positive signal rather than one arbitrary brain region among many.

The behavior assays then aligned with that striatal reading. Colonized mice developed ethanol-induced conditioned taste aversion faster than mock mice at the lower 2 g/kg ethanol dose, while high-dose ethanol produced near-complete aversion in both groups. Colonized mice also showed greater susceptibility to ethanol's behavioral effects, including reduced open-field locomotion after ethanol and increased motor coordination impairment on balance-beam testing. EP1/EP2 antagonism reduced the ethanol-induced balance-beam impairment in colonized mice toward mock-colonized performance, although it did not reverse every ethanol phenotype. That partial specificity is not a weakness. It

distinguishes the $PGE_2$-EP1/EP2 contribution to motor-control impairment from other ethanol-sensitive systems.

For the framework, Kumamoto 2025 is the mirror arm of Markey 2020. Markey shows the eCB branch: colonization shifts the host toward reduced AEA-CB1 signaling and FAAH rescue corrects the neuroendocrine phenotype. Kumamoto shows the $PGE_2$ branch: colonization elevates a prostaglandin output, $dmPGE_2$ reproduces the ethanol phenotype, EP1/EP2 antagonism reverses it, and the dorsal striatal dopamine system carries the behavioral valuation signal. Governance reading: one substrate economy, two branch outputs, two receptor-specific rescue patterns. That is not random noise. That is pathway architecture.

## 5. The Asymmetry Argument and Integrated Pile-Up

The asymmetry principle is the central reason AM#022 exists as a mechanism report rather than as a literature summary. A governance interpretation predicts both arms from one mechanism: if *C. albicans* competes for, redirects, or otherwise governs arachidonic-acid substrate allocation, then host outputs should appear on both sides of the AA branch point. One side should implicate endocannabinoid tone, especially AEA or 2-AG depending on tissue and context. The other should implicate prostaglandin signaling, especially $PGE_2$ where immune-neural valuation and inflammatory modulation are engaged. A perturbation interpretation can still explain each paper individually, but it must pay separately each time. It must explain Markey's FAAH-rescue specificity, Markey's AEA-CORT relationship, Markey's stable bacterial microbiota, Kumamoto's elevated serum $PGE_2$, $dmPGE_2$ phenocopy, EP1/EP2 antagonist reversal, dorsal striatal Drd2 signal, Chevalier's AA supplementation rescue, Chevalier's MAGL inhibition rescue, Chevalier's Lactobacilli rescue, and Severance's probiotic-linked reduction of *C. albicans* IgG. The governance model spends one coin. The perturbation model keeps reaching for another.

The Kumamoto cluster is the cleanest demonstration because it is internally paired. Markey et al. show acute gastrointestinal *C. albicans* colonization altering the eCB arm: anxiety-like behavior, basal and recovery-phase CORT dysregulation, cecal endocannabinoidome changes, and rescue with URB597, a FAAH inhibitor chosen because it raises AEA tone (Markey et al., 2020). Kumamoto et al. then show the $PGE_2$ arm in a related lab lineage: reduced ethanol preference, elevated serum $PGE_2$ metabolite, exogenous $dmPGE_2$ reproduction, EP1/EP2 antagonist reversal, and dorsal striatal dopamine-receptor changes tied to ethanol behavior (Kumamoto et al., 2025). The branch-point structure matters more than either phenotype alone. Anxiety and ethanol aversion are not the same clinical domain. CORT regulation and dorsal-striatal valuation are not the same output system. The framework did not need them to be the same. It predicted that one organism-host substrate economy could drive different outputs depending on which arm of the AA branch was being loaded.

Two asymmetry-strengthening arguments follow. First, host-side calibration is a coincidence the perturbation interpretation must explain. In both Markey and Kumamoto, rescue or reversal works through host receptors and host enzymes at the predicted branch: FAAH for AEA tone, EP1/EP2 for $PGE_2$ output. That means the host system is not merely injured. It is responsive at the exact control points the framework identifies. Second, Markey's 48-hour timeline constrains stochastic-perturbation accounts. A random dysbiosis or sickness model has little room to operate when colonization is acute, bacterial community structure remains broadly stable, invasive disease is absent, and receptor-pathway rescue still works. Fast, specific, reversible, and host-calibrated is not the usual fingerprint of generic damage. [*This is why the word "perturbation" starts to look expensive. You can buy one coincidence with it. Maybe two. But once the same branch point keeps showing up in different labs, different behaviors, different assays, and different rescue routes, the invoice gets ugly.*]

Chevalier et al. add the strongest pile-up evidence because they do not begin with *C. albicans*. They use unpredictable chronic mild stress, fecal microbiota transfer, antibiotic-treated or germ-free

recipients, and depressive-like behavior with reduced hippocampal neurogenesis (Chevalier et al., 2020). The transferred UCMS microbiota produced altered fatty-acid metabolism, reduced AA-containing precursors, reduced hippocampal 2-AG, and impaired mTOR signaling. The rescue architecture is the key: JZL184 blocked MAGL and raised 2-AG, dietary arachidonic acid supplied the substrate directly, and *Lactobacillus plantarum* LpWJL complementation restored eCB levels and behavior. The AA supplementation rescue is the decisive point. It shows that the operative substrate is the AA branch point itself, not merely an inference from enzyme blockade. FAAH or MAGL rescue alone could be dismissed as pharmacological forcing. AA rescue says: feed the branch substrate and the phenotype moves.

Severance et al. provide a human clinical pile-up signal at the gut-fungal interface. In a longitudinal, double-blind, placebo-controlled pilot study of outpatients with schizophrenia, participants completed a two-week placebo run-in and then received probiotic or placebo once daily with food for fourteen weeks (Severance et al., 2017). The probiotic contained *Lactobacillus rhamnosus* GG and *Bifidobacterium animalis* Bb12. Blood was collected at study start and study end, and anti-*C. albicans* IgG was measured by ELISA. The analyzable sample included 56 participants, with the male subset carrying the interpretable treatment signal because baseline female antibody levels differed between groups. In males, probiotic treatment significantly reduced *C. albicans* IgG over time compared with placebo. The highest bowel difficulty burden occurred in *C. albicans*-seropositive males receiving placebo. This does not prove the AA branch by itself. It does something narrower but important: ecological intervention at the gut interface moved a fungal exposure marker and the bowel-burden phenotype in the predicted direction.

The pile-up therefore compounds. Markey and Kumamoto establish the two-arm AA structure under direct *C. albicans* colonization. Chevalier shows that independent microbiota-induced behavioral pathology can be rescued by MAGL inhibition, direct AA supplementation, and Lactobacilli complementation, with AA supplementation functioning as the strongest substrate-level confirmation. Severance shows that human probiotic intervention can reduce *C. albicans* antibody burden in a sex-specific clinical subgroup while tracking bowel difficulty. None of these papers were written to validate Redacted Science. That is precisely why they matter.

[*The framework is not asking these papers to salute. It is asking whether they line up when nobody told them where to stand. They do.*]

Asymmetry compounds monotonically as literature accumulates. Each new independent convergence either increases the burden on the perturbation account or strengthens the governance account. A single result can be noise. Two can be coincidence. A two-arm mechanism with receptor-specific reversal, substrate-specific rescue, microbiota complementation, and human fungal-marker movement becomes harder to wave away without multiplying independent explanations. AM#022 does not claim final proof. It establishes the precedent: when external literature keeps rediscovering framework-predicted control surfaces, the correct unit of analysis is no longer the isolated paper. It is the accumulating coincidence count.

## 6. Adjacent Literature Not Built Into the Claim

AM#022 deliberately excludes broader eCB deficiency theories, cannabinoid hyperemesis literature, chronic pain models, and direct clinical intervention proposals. Those literatures may matter later, especially where Russo-style clinical endocannabinoid deficiency intersects with stuck-state disease. They are not needed for this report. The consolidation rests on four cited sources and the preexisting Paper A branch point. That narrowness is a feature, not a defect.

## 7. Scope Limits and Falsification

This report does not establish human causation, does not recommend antifungal treatment, and does not claim every eCB or PGE2 disorder is governed by *C. albicans*. It consolidates mouse-model and

ecological evidence that matches Paper A's two-arm prediction. The falsification path is explicit: if future external work shows the eCB and PGE2 arms diverge from the framework's two-arm prediction, or if independent rescue mechanisms fail to converge on the same arachidonic-acid pathway, this consolidation fails. [*That is the deal. Mechanism papers do not get to hide behind poetry. They either keep absorbing evidence cleanly, or the bridge cracks.*]

## Counter-Arguments Addressed

First, mouse models do not establish human causation. Correct. AM#022 does not claim they do. The framework's Pan-Mammalian extension predicts that organism-host governance architecture is conserved across the mammalian clade, which makes mouse-model evidence framework-consistent at exactly the right level: not as a stand-in for human data, but as direct evidence about the architecture the framework claims is conserved (Craddock, Pan-Mammalian). Severance et al. is included in the citation spine specifically because it supplies a human ecological pile-up signal that mouse work cannot provide on its own. The complete picture is mouse work demonstrating mechanism plus human work demonstrating that the same fungal-ecological intervention moves clinical markers in the predicted direction.

Second, the eCB and $PGE_2$ arms could be explained by separate mechanisms unrelated to organism-host governance. They could, but the architecture is structurally weaker. Governance predicts both arms from one substrate-level claim about the AA branch point. A two-mechanism account requires not just two unrelated mechanisms but also two matching rescue specificities at the predicted branch enzymes and receptors, FAAH for the eCB arm and EP1/EP2 for the $PGE_2$ arm, plus independent convergence of both mechanisms onto host control surfaces the framework named in advance. The rescue specificities are not coincidence-cheap. They are the same control surfaces Paper A predicted before the external work was published.

Third, Chevalier's AA-supplementation rescue could be explained as a generic anti-inflammatory effect of dietary lipid manipulation. The experimental design argues directly against that interpretation. A generic anti-inflammatory account would predict broad rescue patterns without pathway specificity. Chevalier shows three pathway-specific interventions converging on the same eCB-mediated phenotype: MAGL blockade with JZL184 raises 2-AG by enzyme inhibition, direct dietary arachidonic acid supplementation supplies the branch substrate, and *Lactobacillus plantarum* LpWJL complementation restores the eCB lipid signature through ecological intervention. Three different mechanisms, pharmacological, nutritional, and biological, all hitting the same downstream phenotype is the framework's signature pattern, not a generic anti-inflammatory effect.

Fourth, the convergence across studies could reflect confirmation bias by labs predisposed to find this kind of result. Independence across the citation spine is the structural counter. Markey and Kumamoto are Tufts (Boston, USA) on direct *C. albicans* colonization with anxiety and ethanol behavior as primary endpoints. Chevalier is Institut Pasteur (Paris, France) on UCMS depression with fecal microbiota transfer and hippocampal neurogenesis as primary endpoints. Severance is Johns Hopkins (Baltimore, USA) on probiotic intervention in schizophrenia with antibody titers as primary endpoints. Different labs, different countries, different host phenotypes, different organism manipulation routes, different primary endpoints. Confirmation bias in this configuration would require coordinated international misdirection. Independent convergence is the alternative, and it is exactly what the asymmetry argument values.

## Conclusion

AM#022 locks a narrow empirical point: the arachidonic acid branch point predicted in Paper A now has external support on both downstream arms. Markey demonstrates the eCB/HPA arm with FAAH

rescue. Kumamoto demonstrates the PGE2/ethanol/dopamine arm with PGE2 metabolite measurement, receptor reversal, and dimethyl-PGE2 reproduction. Chevalier adds direct AA-substrate rescue, and Severance adds human ecological correction. The framework does not win by volume. It wins, or fails, by integration. Here, the pieces integrate. That is enough for V1: not a victory lap, but a locked mechanism block with a visible failure path.

## Closing Declaration

This Architect Report AM#022 V1 was generated through the Redacted Science Seed Analysis Process, from Seed 022 of the Paper Seed Master, under the Redacted Science Research Initiative. Issued 20260502 by Jim Craddock, #TheArchitect. Authenticity verifiable at jimcraddock.com/architect-reports. Papers claiming any A-series designation not listed in the registry are not authentic Architect Reports.

## References


1. Chevalier, G., Siopi, E., Guenin-Macé, L., Pascal, M., Laval, T., Rifflet, A., Boneca, I. G., Demangel, C., Colsch, B., Pruvost, A., Chu-Van, E., Messager, A., Leulier, F., Lepousez, G., Eberl, G., & Lledo, P.-M. (2020). Effect of gut microbiota on depressive-like behaviors in mice is mediated by the endocannabinoid system. Nature Communications, 11, 6363. https://doi.org/10.1038/s41467-020-19931-2
2. Craddock, J. Biochemical Computer. Redacted Science Research Initiative. https://doi.org/10.5281/zenodo.19337525
3. Craddock, J. Pan-Mammalian Hypothesis. Redacted Science Research Initiative. https://doi.org/10.5281/zenodo.19643601
4. Craddock, J. Saline Oscillation. Redacted Science Research Initiative. https://doi.org/10.5281/zenodo.19369715
5. Craddock, J. Stuck-State Umbrella. Redacted Science Research Initiative. DOI: 10.5281/zenodo.19582484.
6. Kumamoto et al. [Day, A. W., Hayes, E., Perez-Lozada, J., DiLeo, A., Blandino, K., Maguire, J., & Kumamoto, C. A.]. (2025). *Candida albicans* colonization modulates murine ethanol consumption and behavioral responses through elevation of serum prostaglandin E2 and impact on the striatal dopamine system. mBio, 16(11), e02239-25. https://doi.org/10.1128/mbio.02239-25
7. Markey, L., Hooper, A., Melon, L. C., Baglot, S., Hill, M. N., Maguire, J., & Kumamoto, C. A. (2020). Colonization with the commensal fungus *Candida albicans* perturbs the gut-brain axis through dysregulation of endocannabinoid signaling. Psychoneuroendocrinology, 121, 104808. https://doi.org/10.1016/j.psyneuen.2020.104808
8. Severance, E. G., Gressitt, K. L., Stallings, C. R., Katsafanas, E., Schweinfurth, L. A., Savage, C. L. G., Adamos, M. B., Sweeney, K. P., Origoni, A. E., Khushalani, S., Dickerson, F. B., & Yolken, R. H. (2017). Probiotic normalization of *Candida albicans* in schizophrenia: A randomized, placebo-controlled, longitudinal pilot study. Brain, Behavior, and Immunity, 62, 41-45. https://doi.org/10.1016/j.bbi.2016.11.019


## Suggested Additional Citations

None for V1. This draft intentionally uses only the specified citation spine and the Redacted Science corpus.