

The Microbiome-Host Axis: Influence on Social Behavior and Evolution

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Abstract

The relationship between a host organism and its resident microbial communities has moved far beyond simple notions of gut health and digestion. Accumulating evidence across disciplines — from neuroscience to evolutionary biology to behavioral ecology — points to the microbiome as an active participant in shaping social behavior, mate choice, kin recognition, and even the trajectory of host evolution. This article examines the microbiome-host axis through multiple lenses: the neurochemical pathways through which gut bacteria influence brain function and social motivation, the role of microbial communities in generating chemical signals used for social recognition, the evidence from model organisms for microbiome-mediated behavioral change, and the evolutionary implications of treating the host-microbiome complex as a unit of selection. We review findings from insect, rodent, fish, and primate systems, highlighting both the mechanistic insights and the interpretive challenges that this research field generates. Particular attention is given to how microbially mediated behaviors might drive assortative mating and population divergence, potentially contributing to speciation. The article closes by addressing open questions and the methodological standards the field needs to establish before strong causal claims about microbiome-driven evolution can be fully supported.

Keywords: social behavior, host-microbe coevolution, behavioral evolution, gut microbiome, gut-brain axis, kin recognition

I. Introduction

There is something quietly unsettling about the idea that the bacteria living in your gut might be influencing how you interact with other people. Not in a dramatic, science-fiction way — no microbial puppetmasters pulling neural strings — but in subtler, more pervasive ways: shaping the chemical signals your body produces, tuning the sensitivity of your stress response, nudging the balance of neurotransmitters that affect mood and social motivation. If that sounds speculative, it was, until quite recently. Over the past fifteen years, the evidence has accumulated to the point where dismissing microbiome-behavior connections as fringe science is no longer scientifically defensible.

The human body hosts somewhere between ten trillion and a hundred trillion microbial cells — bacteria, archaea, fungi, and viruses — most of them residing in the gut. For decades, their primary recognized contribution to host biology was metabolic: fermenting dietary fiber, synthesizing vitamins, competing with pathogens. The idea that these organisms might also influence behavior through neural pathways, or that they might shape the social signals animals use to recognize kin and choose mates, simply was not part of mainstream biology's imagination. Germ-free animal models changed that. When researchers raised mice in the complete absence of any gut microbiota and measured their behavior against normally colonized controls, the behavioral differences were striking — and deeply interesting.

The evolutionary implications are potentially even more striking than the neuroscience. If gut microbiota influence mate choice and social affiliation, and if those microbiota are transmitted between individuals through social contact, then the microbiome creates a feedback loop between behavior and microbial community composition that classical evolutionary theory has no obvious framework to accommodate. The host's genome is not the only heritable information that matters; the microbial community it carries is also transmitted, also shapes fitness, and also evolves. Whether this requires us to revise our units of evolutionary analysis — to speak of the "holobiont" (host plus microbiome) as the relevant evolutionary individual — is a question the field is actively and sometimes contentiously wrestling with.

This article traces the microbiome-host axis from molecular mechanisms to evolutionary theory, trying to give an honest account of what we know, what we suspect, and where the evidence still needs to catch up with the ideas.

II. The Gut-Brain Axis: Neurochemical Pathways to Social Behavior

2.1 Mechanisms of Microbial Influence on the Brain

The gut and the brain communicate through a surprisingly rich set of channels. The vagus nerve which extends throughout the gastrointestinal system functions as a direct neural pathway connecting the gut with the brainstem. Gut bacteria produce metabolites and signaling molecules which activate enteroendocrine cells that line the gut wall to transmit signals to vagal afferents. Rodent studies show that cutting the vagus nerve leads to loss of multiple behavioral effects which probiotics and microbial changes produce thus demonstrating that this neural pathway functions as a key causal mechanism instead of being a random connection.

Microbial metabolites travel beyond the vagus nerve to reach blood circulation where they gain ability to penetrate the blood-brain barrier for direct interaction with neural tissues. Short-chain fatty acids which bacteria produce through their fermentation process of dietary fiber, create changes in microglial function which impact the brain's immune cells, resulting in modifications to synaptic pruning and neuroinflammation, which ultimately affects the development of neural circuits. The gut bacteria control the dietary tryptophan conversion to serotonin versus kynurenine through their metabolic processes, which results in a specific ratio that influences both gut motility and central serotonergic signaling involved in social behavior, aggression, and anxiety.

Researchers have established an experimental foundation through germ-free mouse studies which demonstrate these biological mechanisms. Germ-free mice exhibit heightened anxiety through certain test patterns while they demonstrate diminished social interest through different test patterns because they spend less time exploring new social contacts and they show weak reactions to social defeat. The introduction of microbiota from normally-raised mice into germ-free mice restores many of their behavioral traits when the microbiota transfer occurs during their essential developmental periods of early life. The timing matters a great deal: colonization in adulthood restores gut microbial communities but may not fully rescue the social behavioral phenotypes that developed abnormally in germ-free conditions.

2.2 Oxytocin, Serotonin, and Social Motivation

Two neurotransmitter systems with particularly well-established roles in social behavior — the oxytocin system and the serotonin system — show sensitivity to microbial colonization status. Oxytocin, sometimes called the "social bonding hormone" with a degree of oversimplification that most neuroscientists wince at, regulates trust, affiliation, and social memory in both rodents and humans. Germ-free mice show altered oxytocin receptor expression in brain regions involved in social recognition, and probiotic supplementation in rodents can increase social interaction in ways that are blocked by oxytocin receptor antagonists — suggesting that microbial effects on sociality run through the oxytocinergic system at least partially.

The serotonin connection is even more direct. Roughly 90% of the body's serotonin is produced in the gut, largely regulated by gut microbial activity. Germ-free mice have dramatically elevated colonic serotonin — a counterintuitive result that reflects disrupted regulatory feedback — and this peripheral serotonin excess appears to feed back on central circuits. Given serotonin's role in regulating social behavior, aggression, and hierarchical dominance across vertebrate species, microbially influenced variation in serotonin availability is a plausible mechanistic bridge between gut community composition and social phenotype.

III. Microbial Communities and Social Recognition

3.1 Chemical Communication and the Microbiome

Chemical communication describes the most sophisticated mechanism which links microbiome activities to social behavior through animal detection of volatile organic compounds and other odorant emissions which they use to recognize family members and select mates and distinguish themselves from others while identifying their colony membership. The idea that gut bacteria contribute to host odor profiles is not new — it was proposed in the 1970s for mammals — but the experimental evidence to support and extend this idea has only accumulated convincingly in the last decade.

Hyenas provide a particularly well-studied mammalian example. The scent gland secretions these animals use for social communication are chemically complex mixtures, and the bacterial community which lives inside their scent glands produces most of their secretions' volatile compounds through its fermentation activities. Different hyena social groups have different scent gland bacterial communities, and these differences correspond to differences in the volatile compound profiles of their secretions. The microbiome exists as an active participant in social signaling because it creates the chemical signals which animals use to express their social identity.

In insects, the evidence is equally striking. *Drosophila* flies use cuticular hydrocarbons — waxy compounds on the body surface — for mate recognition, and the composition of these hydrocarbons is partly determined by gut and surface bacteria. Manipulating the microbiome of *Drosophila melanogaster* changes their mating preferences: flies prefer to mate with individuals that share their microbiome. Crucially, this preference

disappears when flies are treated with antibiotics to eliminate their bacteria, and it can be restored by reintroducing the original bacterial communities. This is a clean experimental demonstration that microbiome composition drives mating behavior through chemical signal production.

As illustrated in Figure 1, the pathway from gut microbiome composition through cuticular hydrocarbon production to mating preference in *Drosophila* provides a mechanistic model for how microbiome-mediated chemical signaling can shape social behavior.

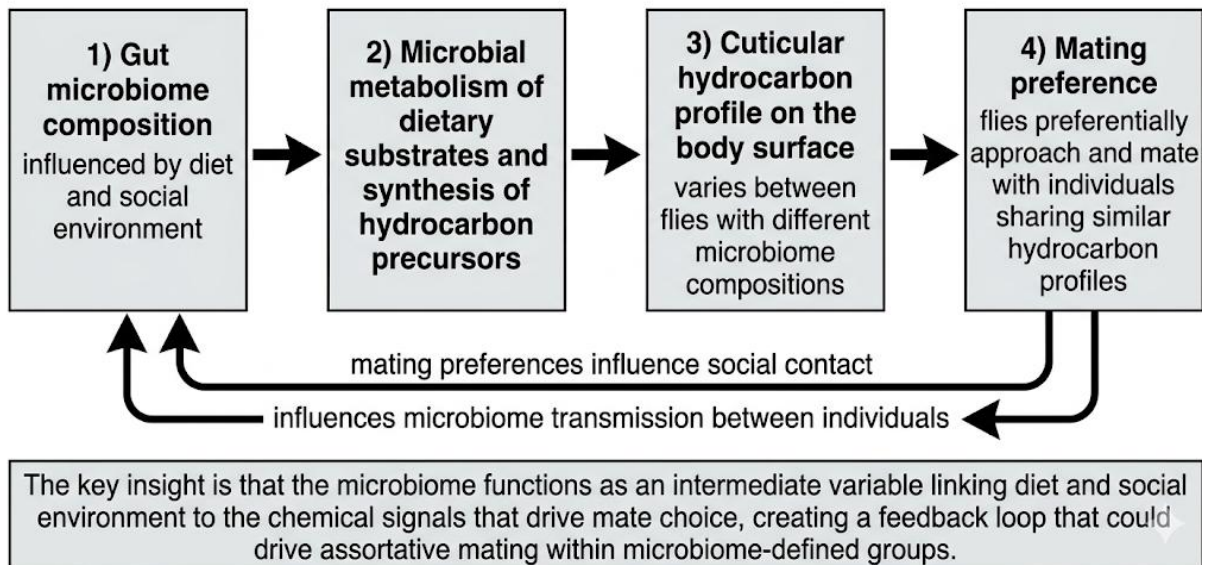


Fig.1: Mechanistic Pathway from Gut Microbiome Composition to Mating Preference in *Drosophila melanogaster* Through Cuticular Hydrocarbon Signaling, Source: Author Generated

The flowchart diagram demonstrates four sequential boxes that the arrows connect to each other. The first box shows Gut microbiome composition which gets affected by both diet and social environment. The second box shows how microbes break down dietary materials to produce hydrocarbon precursor substances. The third box displays the cuticular hydrocarbon profile that exists on the body surface of flies which have different microbiome compositions. The fourth box shows the mating preference pattern which causes flies to choose partners who have identical hydrocarbon profiles. The feedback arrows show that mating preferences result in social contact patterns which create pathways for microbiome transmission between people. The microbiome serves as an intermediary factor which connects dietary and social environmental impact to chemical signals that determine mate selection which creates a feedback loop that leads to mating patterns based on microbiome-related groupings.

3.2 Kin Recognition and Microbial Transmission

The study of kinship ties serves as a basis for understanding how social behavior evolved according to Hamilton's inclusive fitness theory, which states that people should prefer to assist their blood relatives, but they need systems that enable them to recognize their relatives. Vertebrates use olfactory cues which originate from the major histocompatibility complex (MHC) to identify relatives, but the microbiome system provides additional support for this identification process.

Gut microbial communities spread between family members through three pathways which include vertical transmission that occurs from mother to child during birth and nursing as well as horizontal transmission which happens when family members share space and groom each other and eat meals together. Family members develop similar microbial profiles because they share more environmental factors and behavioral patterns than people who do not share genetic ties and because MHC genes among other host genetic factors help determine microbiome composition. The olfactory cues that animals use to identify their relatives through recognition contain two sources of similarity, namely shared microbiome composition and shared host genetic material between kin.

IV. Evolutionary Implications: The Holobiont Concept

4.1 Host-Microbiome Coevolution

The holobiont concept — the idea that the host and its associated microbial communities should be treated as a single unit of evolutionary analysis — has generated both enthusiasm and sharp criticism since it

was formalized by Zilber-Rosenberg and Rosenberg around 2008. The enthusiasm is easy to understand. If the microbiome profoundly influences host fitness, and if microbial communities are heritable (either vertically through transmission or environmentally through habitat fidelity), then selection acts on host-microbiome combinations, not on host genomes alone. The coral that thrives in warming oceans may do so partly because it harbors heat-tolerant *Symbiodinium*; selecting for coral heat tolerance selects for this partnership, not just the coral genome.

The criticism is equally understandable. For holobiont selection to operate coherently, there needs to be reliable enough transmission of specific microbial communities across generations that consistent host-microbiome combinations can be maintained across the timescales over which selection operates. In many organisms, vertical transmission of the microbiome is imperfect and variable; horizontal transmission from the environment can rapidly replace or supplement vertically acquired communities. Critics argue that the microbiome is often too variable, too environmentally contingent, and too rapidly replaceable to function as a stable unit of heredity in the way that host genomes do.

Both sides of this debate are making valid points, and the resolution probably varies by system. For organisms with highly vertical microbiome transmission — like some insects where specific bacterial symbionts are directly transmitted to eggs — holobiont-level selection is a compelling framework. For organisms with more open and environmentally contingent microbiome assembly, the framework requires more careful qualification. The honest position is that host-microbiome coevolution is real, but its evolutionary dynamics vary enormously across systems, and generalizing from one system to all of biology is unwise.

4.2 Microbiome-Mediated Reproductive Isolation and Speciation

Perhaps the most provocative evolutionary hypothesis to emerge from microbiome research is that gut bacteria might contribute to reproductive isolation and speciation. The logic runs as follows: if mating preferences are influenced by microbiome composition (as demonstrated in *Drosophila*), and if populations in different environments develop different microbiome compositions (due to different diets, climates, or microbial communities in the environment), then microbiome divergence between populations could create partial reproductive isolation between them. Flies from population A prefer to mate with other flies from population A partly because they share similar microbiome-derived chemical signals; the same applies to population B. This assortative mating could reduce gene flow between populations, facilitating genetic divergence and ultimately speciation.

The *Drosophila* experiments by Sharon and colleagues demonstrated this principle in the laboratory: two *Drosophila* populations maintained on different diets developed different microbiome compositions, different mating preferences, and — crucially — these mating preferences disappeared when the flies were treated with antibiotics. This is a striking result. It suggests that dietary divergence, by driving microbiome divergence, could drive behavioral reproductive isolation without any initial genetic difference between populations.

Figure 2 illustrates the hypothetical pathway from dietary divergence through microbiome differentiation to assortative mating and eventual reproductive isolation, situating microbiome-mediated speciation within the broader context of allopatric and sympatric divergence models.

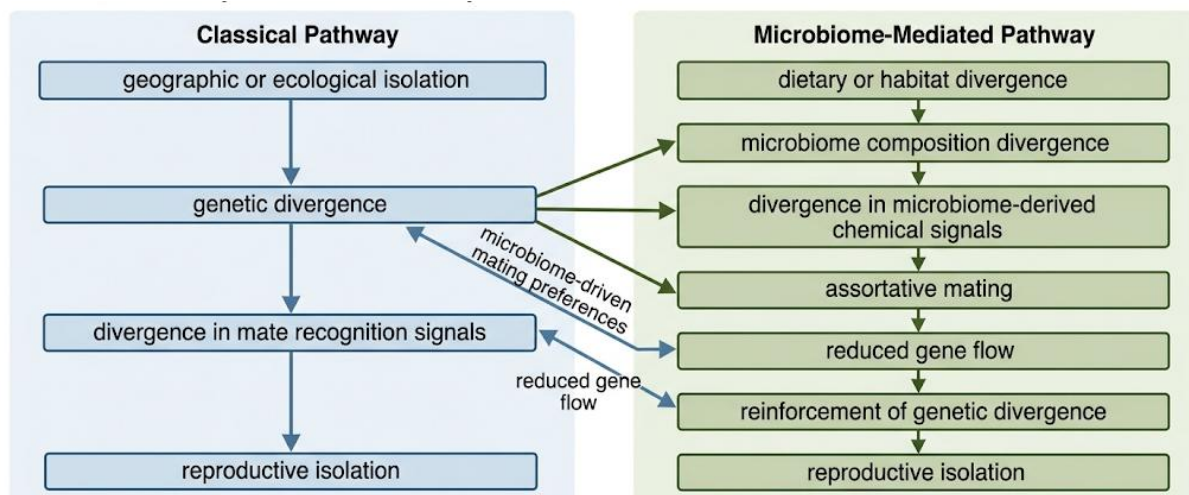


Fig. 2: Hypothetical Pathway from Environmental Divergence to Microbiome-Mediated Reproductive Isolation, with Comparison to Classical Speciation Mechanisms, Source: Author Generated

The two parallel pathways that lead to different outcomes are shown in this diagram. The classical pathway (left column) shows: geographic or ecological isolation → genetic divergence → divergence in mate recognition signals → reproductive isolation. The microbiome-mediated pathway (right column) shows: dietary or habitat divergence → microbiome composition divergence → divergence in microbiome-derived chemical signals → assortative mating → reduced gene flow → reinforcement of genetic divergence → reproductive isolation. The two pathways show interactivity because their arrows create links which show their mutual influence: genetic divergence affects microbiome composition through host genetic influences on microbial community assembly, while microbiome-driven mating preferences accelerate genetic divergence by reducing gene flow. The main point establishes that microbiome-based reproductive isolation functions as a genetic speciation mechanism which provides an essential process for organisms to develop genetic differences.

V. Evidence from Primate and Human Systems

5.1 Primate Social Behavior and Microbiome Composition

Research on non-human primates has added an important social dimension to microbiome-behavior connections that rodent studies, for all their experimental elegance, cannot easily capture. The social structure of primates consists of complex groups that maintain stable relationships through their advanced social cognition abilities. The microbiomes of individuals in the study group develop through their social interactions and their grooming connections and their membership in specific social groups according to researchers.

Social group membership among savanna baboons in Kenya shows a stronger connection to microbiome composition than food consumption according to research findings which also show that people with more social ties develop broader gut microbial networks. The relationship between social behavior and a richer microbiome shows two pathways that scientists need to investigate because both pathways need to be studied. The natural behavior of wild primate populations results in multiple effects that scientists need to study at the same time. The microbiome exists within primate social structure because it functions as a fundamental biological element that controls their social interactions.

Chimpanzee studies have extended these findings to show that microbiome composition changes predictably when individuals transfer between social groups — a common event in chimpanzee society — and that the new group's microbiome is rapidly acquired through social contact, grooming, and shared food handling. The microbiome is, in a very real sense, a product of social history in these long-lived primates, which raises fascinating questions about how social disruptions — group fission, captivity, habitat fragmentation — affect the microbiome and potentially the social behavior it influences.

5.2 Human Implications and Caution

The jump from mouse and primate studies to human behavior requires considerable caution. Human social behavior is influenced by culture, cognition, economic structure, and historical contingency in ways that no animal model captures. Observational associations between gut microbiome composition and social phenotypes in humans — including associations reported with autism spectrum disorder, depression, and social anxiety — are interesting and have generated enormous research interest and clinical investment. However, the causal interpretation of these associations is far from settled.

Most human microbiome-behavior studies are cross-sectional: they measure microbiome composition and behavioral phenotypes at the same time point and report correlations. These correlations could reflect microbial influence on behavior, behavioral influence on microbiome (through diet, stress, social contact patterns), shared genetic influences on both, or common environmental factors affecting both independently. Randomized controlled trials of probiotic interventions on social behavior and mood in humans exist, and some show statistically significant effects — but effect sizes are typically modest and replication across studies is inconsistent. The field is not ready to support strong clinical claims, and the popular press's enthusiasm for gut-brain axis findings has run well ahead of the evidentiary standards the science actually meets.

VI. Conclusion

The microbiome-host axis is not a peripheral curiosity in behavioral biology or evolutionary theory. It sits near the center of some of the field's most important and unresolved questions: How do animals recognize kin and choose mates? What drives the early stages of population divergence and speciation? How do social environments get "under the skin" to affect individual health and behavior? The accumulating evidence suggests that gut microbial communities contribute meaningfully to all of these processes.

What makes this intellectually exciting — and what demands intellectual honesty — is that the field is still young. The mechanistic picture of how gut bacteria influence brain function through the gut-brain axis is increasingly detailed and credible. The evidence that microbiome-derived chemical signals shape social recognition and mating behavior is compelling, particularly from insect systems where experimental manipulation is cleanest. The evolutionary implications — holobiont selection, microbiome-mediated speciation

— are genuinely intriguing but require more rigorous empirical foundation before they can be called established science rather than well-motivated hypothesis.

The human implications deserve particular care. The gut microbiome almost certainly influences aspects of human mood, anxiety, and social cognition through the pathways documented in model organisms. How large these effects are relative to the genetic, developmental, experiential, and cultural factors that shape human social behavior is not yet clear, and overselling microbiome effects on human behavior does a disservice to both the science and the public.

What we can say without qualification is that the classical view of the organism as a genetically defined individual, whose behavior is explained purely in terms of its own nervous system and its own evolutionary history, is insufficient. The microbes we carry with us are not passengers — they are, to varying degrees across different taxa and different biological contexts, participants in the production of the social phenotypes that drive the evolutionary dynamics we study.

References

- [1]. Archie, E. A., & Theis, K. R. (2011). Animal behaviour meets microbial ecology: The microbiome as a novel axis of social organisation. *Animal Behaviour*, 82(3), 425–436. <https://doi.org/10.1016/j.anbehav.2011.05.034>
- [2]. Bravo, J. A., Forsythe, P., Chew, M. V., Escaravage, E., Savignac, H. M., Dinan, T. G., & Cryan, J. F. (2011). Ingestion of *Lactobacillus* strain regulates emotional behavior and central GABA receptor expression in a mouse via the vagus nerve. *Proceedings of the National Academy of Sciences*, 108(38), 16050–16055. <https://doi.org/10.1073/pnas.1102999108>
- [3]. Buffington, S. A., Di Prisco, G. V., Auchtung, T. A., Ajami, N. J., Petrosino, J. F., & Costa-Mattioli, M. (2016). Microbial reconstitution reverses maternal diet-induced social and synaptic deficits in offspring. *Cell*, 165(7), 1762–1775. <https://doi.org/10.1016/j.cell.2016.06.001>
- [4]. Clarke, G., Grenham, S., Scully, P., Fitzgerald, P., Moloney, R. D., Shanahan, F., & Cryan, J. F. (2013). The microbiome-gut-brain axis during early life regulates the hippocampal serotonergic system in a sex-dependent manner. *Molecular Psychiatry*, 18(6), 666–673. <https://doi.org/10.1038/mp.2012.77>
- [5]. Cryan, J. F., & Dinan, T. G. (2012). Mind-altering microorganisms: The impact of the gut microbiota on brain and behaviour. *Nature Reviews Neuroscience*, 13(10), 701–712. <https://doi.org/10.1038/nrn3346>
- [6]. Ezenwa, V. O., Gerardo, N. M., Inouye, D. W., Medina, M., & Xavier, J. B. (2012). Animal behavior and the microbiome. *Science*, 338(6104), 198–199. <https://doi.org/10.1126/science.1227412>
- [7]. Foster, J. A., & McVey Neufeld, K. A. (2013). Gut-brain axis: How the microbiome influences anxiety and depression. *Trends in Neurosciences*, 36(5), 305–312. <https://doi.org/10.1016/j.tins.2013.01.005>
- [8]. Goodrich, J. K., Waters, J. L., Poole, A. C., Sutter, J. L., Koren, O., Blekhman, R., & Ley, R. E. (2014). Human genetics shape the gut microbiome. *Cell*, 159(4), 789–799. <https://doi.org/10.1016/j.cell.2014.09.053>
- [9]. Lombardo, M. P. (2008). Access to mutualistic endosymbiotic microbes: An underappreciated benefit of group living. *Behavioral Ecology and Sociobiology*, 62(4), 479–497. <https://doi.org/10.1007/s00265-007-0428-9>
- [10]. Lyte, M. (2013). Microbial endocrinology in the microbiome-gut-brain axis: How bacterial production and utilization of neurochemicals influence behavior. *PLOS Pathogens*, 9(11), e1003726. <https://doi.org/10.1371/journal.ppat.1003726>
- [11]. Montiel-Castro, A. J., González-Cervantes, R. M., Bravo-Ruiseco, G., & Pacheco-López, G. (2013). The microbiota-gut-brain axis: Neurobehavioral correlates, health and sociality. *Frontiers in Integrative Neuroscience*, 7, 70. <https://doi.org/10.3389/fnint.2013.00070>
- [12]. Mulle, J. G., Sharp, W. G., & Cubells, J. F. (2013). The gut microbiome: A new frontier in autism research. *Current Psychiatry Reports*, 15(2), 337. <https://doi.org/10.1007/s11920-012-0337-0>
- [13]. Sampson, T. R., & Mazmanian, S. K. (2015). Control of brain development, function, and behavior by the microbiome. *Cell Host & Microbe*, 17(5), 565–576. <https://doi.org/10.1016/j.chom.2015.04.011>
- [14]. Sharon, G., Segal, D., Ringo, J. M., Hefetz, A., Zilber-Rosenberg, I., & Rosenberg, E. (2010). Commensal bacteria play a role in mating preference of *Drosophila melanogaster*. *Proceedings of the National Academy of Sciences*, 107(46), 20051–20056. <https://doi.org/10.1073/pnas.1009906107>
- [15]. Stilling, R. M., Dinan, T. G., & Cryan, J. F. (2014). Microbial genes, brain and behaviour: Epigenetic regulation of the gut-brain axis. *Genes, Brain and Behavior*, 13(1), 69–86. <https://doi.org/10.1111/gbb.12109>
- [16]. Theis, K. R., Schmidt, T. M., & Holekamp, K. E. (2012). Evidence for a bacterial mechanism for group-specific social odors among hyenas. *Scientific Reports*, 2, 615. <https://doi.org/10.1038/srep00615>
- [17]. Tung, J., Barreiro, L. B., Burns, M. B., Grenier, J. C., Lynch, J., Grieneisen, L. E., & Archie, E. A. (2015). Social networks predict gut microbiome composition in wild baboons. *eLife*, 4, e05224. <https://doi.org/10.7554/eLife.05224>
- [18]. Yano, J. M., Yu, K., Donaldson, G. P., Shastri, G. G., Ann, P., Ma, L., & Hsiao, E. Y. (2015). Indigenous bacteria from the gut microbiota regulate host serotonin biosynthesis. *Cell*, 161(2), 264–276. <https://doi.org/10.1016/j.cell.2015.02.047>
- [19]. Zilber-Rosenberg, I., & Rosenberg, E. (2008). Role of microorganisms in the evolution of animals and plants: The hologenome theory of evolution. *FEMS Microbiology Reviews*, 32(5), 723–735. <https://doi.org/10.1111/j.1574-6976.2008.00123.x>