Primate exposure to anthropogenic pollutants: Interactions with the gut microbiome and neuroendocrine SYSTEM

openaccessgovernment.org

Emily Warrender

5 September 2025

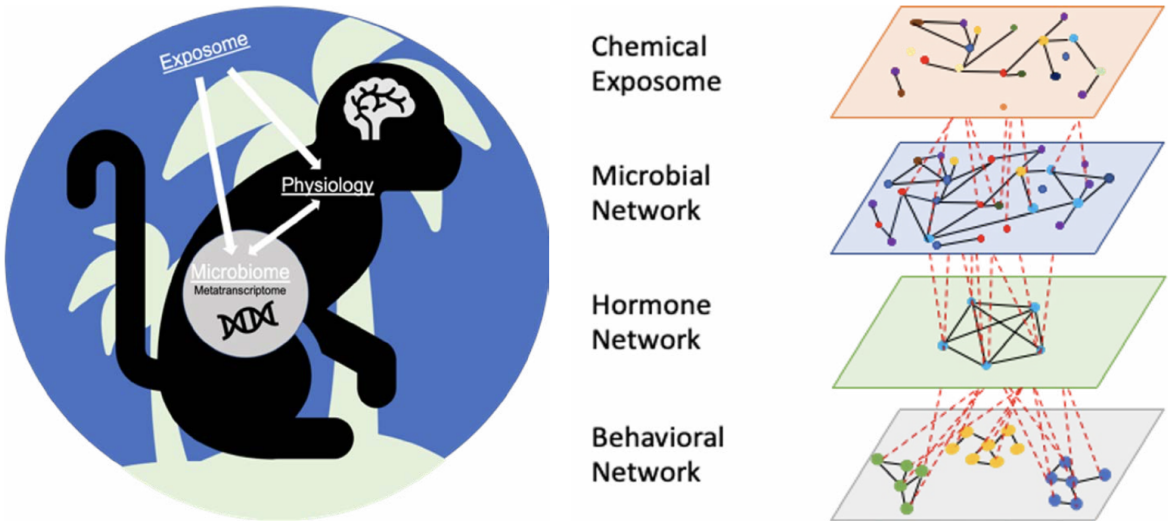

Figure 1. Hypothetical relationships among the chemical exposome, gut microbiome, and neuroendocrine system of a wild primate.

Michael Wasserman of Indiana University discusses interactions among the chemical exposome, microbes, and hormones in wild primates

What is the relevance of the chemical exposome to wild primates?

The use of omics methodology to look at a range of biological molecules from genes to proteins in organisms facing rapidly changing environments globally, especially to explore causal relationships between exposomes and biological outcomes within real-world complex social-ecological systems, offers the potential to address environmental and public health concerns. [1,2]

The exposome is the lifetime sum of environmental exposures, both chemical and non-chemical. [3] The chemical exposome, including pollutants such as pesticides, flame retardants, plastics, and heavy metals, is particularly relevant to the health of primates, as exogenous chemicals can directly affect both the neuroendocrine system through endocrine disruption [4] and the gut microbial community through changes in microbial species composition from species-specific toxicity. [5] It can also indirectly affect primates through their gut microbiome, as microbial species influence host physiology through their own activity, including metabolizing and modulating the effects of exogenous chemicals. [6]

How does the gut microbiome contribute to primate physiology?

Many aspects of primate physiology are affected by microbial symbionts, including metabolism and the immune response. Up to one-third of all molecules circulating in an animal's bloodstream are of microbial origin. [7] One essential role of the microbiome is to facilitate energy availability through fermentation. While primates lack the ability to produce enzymes to break down plant fiber, the microbiome can metabolize it in either the foregut or hindgut and provide short-chain fatty acids (SCFAs) as an energy source to the primate and microbes. SCFAs can also act as signaling molecules to the primate immune system. [8] Butyrate, for example, is a bacterially derived SCFA that modifies the cytokine production profile of T helper cells and promotes intestinal barrier function, thus preventing inflammatory processes. Acetate also helps to maintain intestinal barrier integrity and prevents some Escherichia coli infections. Additionally, other microbial metabolites can regulate the intestinal absorption of iron, [9] which is essential for the innate immune response. [10]

How is the primate neuroendocrine system affected by the gut microbiome?

Gut microbes also alter primate physiology via the neuroendocrine system. [11] For example, microbes can produce neurotransmitters, such as serotonin and dopamine, and influence the functioning of endocrine glands, including the adrenal cortex. [12] The field of microbial endocrinology examines interactions between microbes and their host's neuroendocrine systems. It is built on the premise that microbes have evolved mechanisms to sense host hormones, which they use as environmental cues of habitat suitability. [13]

Some gut microbes can produce neurotransmitters, hormones, and hormone-like metabolites that communicate directly with their host's brain through the afferent neurons of the vagus nerve or indirectly through various endocrine axes. In response, the host sends information back to the gut via efferent neurons from the brain, which elicits physiological responses in the enteric and central nervous system, or changes hormone production along endocrine axes.

Dysbiosis of gut microbes can occur when there is a perturbation in this communication system, which can lead to a myriad of undesirable physiological responses, such as neurological and gastrointestinal disorders. [14, 15] Primate microbial endocrinology utilizes the strengths of studying non-human primates in the wild, including the ability to collect non-invasive samples and data on gut microbial communities, hormones, and behavior across environmental gradients. [16] A recent study using this approach documented significant relationships between gut microbial diversity and fecal cortisol and estradiol in howler monkeys living on Barro Colorado Island in Panama. [17]

Developing a three-factor model: Interactions among the chemical exposome, gut microbiome, and neuroendocrine system in wild primates

The chemical exposome can alter host microbial community composition and diversity through absorption, inhalation, and consumption, while also eliciting specific direct and indirect host physiological responses from the neuroendocrine and immune systems. [5, 18] For example, gut microbial communities of people living in a rural area of Burkina Faso were more diverse than those of people living in an urban area of Italy, with SCFA-producing bacteria more abundant in the rural area, likely due to their low-sugar and high-plant-polysaccharides diet. [19] Non-human primates are subject to similar differences in gut microbial composition and diversity based on diet. A study of five howler monkey groups across habitats of different qualities and one group in captivity found that microbial communities and the quantity of genes in those microbes that control butyrate production and hydrogen metabolism differed, with howlers in 'suboptimal' habitats having reduced microbial diversity and number of genes. [20]

One group of overlooked environmental factors likely altering primate microbiomes is anthropogenic pollutants, increasingly common components of the exposome globally, including in remote, protected tropical forests. [21,22] Pesticide exposure is of particular concern as it has been demonstrated that some pesticides accumulate in the environment after being transported beyond agricultural areas and can negatively affect the gut microbiome and neuroendocrine system. For example, exposure to the pesticide chlorpyrifos in mice resulted in a decrease in beneficial bacteria and an increase in gut permeability and inflammation, [24] as well as inducing obesity and insulin resistance. [25] Recent work has shown that exposure to pesticides and flame retardants likely through air inhalation and consumption of plant foods in Kibale National Park, Uganda, correlates with changes in fecal cortisol levels in juveniles for some primate species. [23] Therefore, clarifying interactions among the chemical exposome, gut microbiome, and neuroendocrine system in wild primates will offer important insight relevant to environmental and public health (Fig. 1).

CLICK HERE for references

Primary Contributor

Michael D Wasserman
Indiana University

Additional Contributor(s)

Kathryn Michelle Benavidez Westrich
Indiana University

Emily Chester
Indiana University