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Short Communication

Short Chain Fatty Acids Role in Gut Morphology

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ABSTRACT

A substantial body of evidence supports that the gut microbiota plays a pivotal role in the regulation of metabolic, endocrine and immune functions. In recent years, there has been rising recognition of the participation of the gut microbiota in the modulation of multiple neurochemical pathways through the highly interconnected gut-brain axis. Though amazing scientific breakthroughs over the last few years have prolonged our knowledge on the communication between microbes and their hosts, the underpinnings of microbiota-gut-brain crosstalk remain to be determined. Short-chain fatty acids (SCFAs), the main metabolites produced in the colon by bacterial fermentation of dietary fibers and resistant starch, are speculated to play a key role in neuro-immunoendocrine regulation. However, the fundamental mechanisms through which SCFAs might influence brain physiology and behavior.

Keywords: Gut microbiota; Short chain fatty acids; Brain physiology

DESCRIPTION

The modulation of gut physiology by the CNS and its effects on gut function such as motility, secretion, blood flow, nociception, and immune function during neurological stressors are well-documented. Further, brain to gut signaling can directly affect the microbiota, either *via* immune system or gut functions such as motility, release of neurotransmitters and intestinal immune tone [1]. Comparatively, gut to CNS signaling has been studied for a short period, and the mechanisms underlying this crosstalk are starting to be understood. It is noteworthy that several brain disorders have been linked to imbalances in the microbial composition of the gut. However, whether these alterations in the microbiota are induced by brain signaling or whether brain dysfunction is driven by changes in the gut microbiota remains to be fully determined. Although a more compelling causal relationship between altered gut microbial composition and brain dysfunction is still needed, it has been shown that disruption in the neuronal and microbial organization in prenatal and postnatal periods of mammalian development may lead to the onset of neurodevelopmental and other brain disorders later in life. In a similar way, growing evidence has shown that alterations in maternal microbiome during pregnancy, such as use of antibiotics or probiotics, variations in diet, immune activation, and exposure to stress can modulate the microbiome, neurodevelopment, and behavior of offspring in both rodents and humans [2]. Furthermore, distribution mode and early-life incidences such as nourishing changes, infection, and antibiotics treatment have a huge effect on the gut microbiota composition with a long-term impact on brain and also behaviour. Under physiological circumstances, activation of immune cells and production of cytokines can have a minor impact in the CNS [3]. However, chronic systemic inflammation, mostly in the cases of infections, has long been related with behavioral alterations and cognitive dysfunction. It is now widely known that peripheral insults that cause a systemic inflammatory response might affect ongoing inflammation in the CNS mostly by microglial activation, production of inflammatory molecules, as well as recruitment of peripheral immune cells into the brain, thus determining a cerebral inflammatory milieu that may seriously affect neuronal function [4]. Notable, during gut pathologies with increased penetrability of the intestinal barrier, the translocation of bacterial products can increase the production of cytokines and impact the Blood-Brain Barrier (BBB), leading to more intense harmful effects. Additional, it has already been shown that several bacterial strains can modify levels of neurotransmitter precursors in the gut lumen and even self-sufficiently synthesize (or modulate the synthesis of) a number of Neurotransmitters, serotonin (5-HT), including γ-Amino Butyric Acid (GABA), Dopamine (DA), and noradrenaline (NA). These neurotransmitters can possiblyinfluence microglial activation and several cerebral functions. Moreover, the

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sympathetic branch of the autonomic nervous system is also involved in intestinal homeostasis and immune regulation [5]. Conversely, the gut microbiota can interrelate with the CNS *via* gut modulation or directly *via* metabolites and endotoxin translocation from the lumen to the circulation. Likely signal transducers involved in the communication of the microbiota with the CNS include enterochromaffin cells, which can quandary numerous microbial products and secrete serotonin into the lamina propria, increasing colonic and blood concentrations of 5-HT.

CONCLUSION

Gut-brain message can also be attained through vagus nerve signaling. Vicissitudes in enteric neuron activity apparent by the vagus nerve are essential for mediating satiety, stress, and mood. Given the close physical nearness, gut bacteria can interact with and trigger the vagus nerve, thus exerting effects upstream to the CNS. This notion is in full contract with early educations performance that oral inoculation with pathogens or probiotics persuades activation of the vagal sensory neurons that innervate the GI moving the regulation of CNS functions. However, whether the vagus nerve is triggered by physical interaction with bacteria or finished soluble microbial mechanisms remain to be determined. Finally, bacterial metabolic byproducts including SCFAs are often considered key candidate mediators of gut-brain communication, and altered SCFA production has been established in a variety of neuropathologies.

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