Commentary

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The Role of Gut Microbiota in Stroke Prevention and Systemic Inflammation

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Description

The gut microbiota, the diverse population of microorganisms residing in the human gastrointestinal tract, has emerged as a critical regulator of overall health and disease. Recent scientific investigations have illuminated the profound influence of gut microbiota on the Central Nervous System (CNS) and cardiovascular health, underscoring its pivotal role in stroke prevention. Stroke, a leading cause of disability and death worldwide, is predominantly caused by either ischemia or hemorrhage in the brain. Emerging evidence suggests that the gut microbiota not only influences systemic inflammation and metabolic pathways but also directly impacts cerebrovascular function and stroke risk. Understanding the dynamic interplay between the gut and the brain through the gut-brain axis is essential for developing innovative therapeutic strategies aimed at reducing stroke incidence. Systemic inflammation is a well-established risk factor for stroke, and the gut microbiota plays a crucial role in modulating inflammatory responses. The gut is home to trillions of microbes that produce metabolites such as Short-Chain Fatty Acids (SCFAs), which are derived from dietary fiber fermentation. SCFAs, including butyrate, propionate, and acetate, have anti-inflammatory properties and help maintain the integrity of the intestinal barrier. A robust gut barrier prevents the translocation of pathogenic bacteria and endotoxins into the bloodstream, thereby reducing systemic inflammation. Conversely, dysbiosis, an imbalance in the gut microbiota, can lead to the release of pro-inflammatory cytokines and an increase in circulating Lipopolysaccharides (LPS), which are associated with endothelial dysfunction and heightened stroke risk. Hypertension is the most significant modifiable risk factor for stroke, and the gut microbiota has been implicated in blood pressure regulation. Certain gut bacteria produce metabolites that interact with the Renin-Angiotensin System (RAS), a critical regulator of blood pressure. For example, SCFAs have been shown to activate G-protein-coupled receptors on vascular endothelial cells, leading to vasodilation and reduced blood pressure.

Lipid metabolism and atherosclerosis

Atherosclerosis, the buildup of fatty deposits in arterial walls, is a significant contributor to ischemic stroke. The gut microbiota influences lipid metabolism by modulating bile acid synthesis and enterohepatic circulation. Specific bacterial species convert primary bile acids into secondary bile acids, which regulate cholesterol levels and prevent the formation of atherosclerotic plaques. Dysbiosis, however, can disrupt this balance, leading to hyperlipidemia and increased plaque formation. Moreover, certain gut bacteria produce Trimethylamine (TMA) from dietary choline and carnitine, found in red meat and eggs. TMA is subsequently converted into Trimethylamine N-Oxide (TMAO) in the liver, a compound linked to enhanced atherosclerosis and cardiovascular events, including stroke. Strategies to modulate TMAO levels through dietary changes or microbiota-targeted therapies could, therefore, play a role in stroke prevention. The gut-brain axis, a bidirectional communication network between the gut microbiota and the CNS, is instrumental in neuroprotection and stroke prevention. The vagus nerve, immune system, and microbial metabolites serve as conduits for this interaction. SCFAs, for instance, influence microglial activity, the brain's resident immune cells, and modulate neuroinflammation, a key factor in stroke pathogenesis. Additionally, gut microbes produce neurotransmitters such as Gamma-Aminobutyric Acid (GABA), serotonin, and dopamine, which regulate neuronal excitability and stress responses. Dysbiosis has been associated with increased permeability of the Blood-Brain Barrier (BBB), rendering the brain more vulnerable to ischemic injury. Interventions aimed at restoring gut microbiota balance could enhance BBB integrity and confer neuroprotection against stroke.

Dietary interventions

Diet plays a crucial role in shaping gut microbiota composition and functionality, offering a promising avenue for stroke prevention. A fiber-rich diet promotes the growth of beneficial microbes that produce SCFAs, which, as mentioned, have anti-inflammatory and vasodilatory effects. The Mediterranean diet, characterized by high consumption of fruits, vegetables, whole grains, and olive oil, has been associated with a reduced risk of stroke. This dietary pattern enhances gut microbiota diversity and reduces the abundance of pro-inflammatory species. Conversely, a Western diet, high in saturated fats, sugars, and processed foods, fosters dysbiosis and exacerbates stroke risk factors. Personalized nutrition, guided by gut microbiota profiling, could optimize dietary interventions for stroke prevention. Probiotics and prebiotics have garnered attention as therapeutic agents for modulating gut microbiota and reducing stroke risk. Probiotics, live beneficial bacteria, can enhance gut

barrier function, reduce inflammation, and lower blood pressure. For example, strains of *Lactobacillus* and *Bifidobacterium* have demonstrated potential in mitigating hypertension and improving lipid profiles. Prebiotics, on the other hand, are non-digestible fibers that selectively promote the growth of beneficial microbes. In combination, probiotics and prebiotics can synergistically enhance gut health and mitigate risk factors associated with stroke. Clinical trials are underway to evaluate the efficacy of synbiotics (a combination of probiotics and prebiotics) in preventing cardiovascular and cerebrovascular diseases. Recent studies have introduced the concept of the microbiota-gut-vascular axis, highlighting the role of gut microbiota in vascular health. The interaction between gut microbes and endothelial cells influences vascular tone, permeability, and inflammation. For instance, SCFAs regulate endothelial nitric oxide production, a key factor in maintaining vascular homeostasis. Dysbiosis-induced endothelial dysfunction can precipitate thrombosis, a major cause of ischemic stroke. Understanding the mechanisms underlying the microbiota-gut-vascular axis could unveil novel therapeutic targets for stroke prevention.