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Short Communication

Supplementation of Vitamin K2 involved in Cognitive Functions and Diet Composition

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DESCRIPTION

Vitamin K2, also known as Menaquinone, is a fat-soluble vitamin that plays an important role in blood coagulation, bone metabolism, and vascular health. Vitamin K2 has been shown to have various beneficial effects on the nervous system, such as preventing neuronal apoptosis, oxidative stress, and inflammation. However, the gut microbiota, which is the main source of vitamin K2, can be altered by various factors such as antibiotics, diet, stress, and aging. This can lead to gut dysbiosis, which is an imbalance of the microbial composition and function in the intestine [1,2].

Gut dysbiosis can affect the production and absorption of vitamin K2, as well as cause intestinal inflammation and permeability. These changes can have negative consequences for the brain, as the gut-brain axis is a bidirectional communication system that links the intestinal microbiota with the central nervous system. One of the possible mechanisms by which gut dysbiosis can affect the brain is through neuroinflammation, which is a chronic activation of the immune system in brain [3-5].

Neuroinflammation can be triggered by various factors such as infections, injuries, toxins, and stress. It can also be induced by gut-derived pro-inflammatory molecules such as Lipopolysaccharide (LPS), which is a component of the outer membrane of gram-negative bacteria. LPS can cross the intestinal barrier and enter the bloodstream, where it can reach the brain and activate microglia, which are the resident immune cells of the brain. Microglia can produce inflammatory cytokines and Reactive Oxygen Species (ROS), which can damage neurons and impair cognitive functions [6,7].

Therefore, it is possible that vitamin K2 deficiency caused by gut dysbiosis can contribute to neuroinflammation and neurodegeneration [8]. To test this hypothesis, a recent study has investigated the effect of vitamin K2 supplementation on cognitive impairment induced by antibiotics in rats. The rats were divided into four groups: control group (received normal saline), antibiotic group (received ceftriaxone for 10 days), vitamin K2 group (received vitamin K2 for 10 days), and antibiotic+vitamin K2 group (received both ceftriaxone and vitamin K2 for 10 days).

The cognitive functions of the rats were assessed by Morris water maze test, which measures spatial learning and memory. The results showed that antibiotic treatment caused gut dysbiosis, as evidenced by a decrease in the diversity and abundance of beneficial bacteria such as Bifidobacterium and Lactobacillus, and an increase in pathogenic bacteria such as Escherichia coli and Enterococcus faecalis [9]. Antibiotic treatment also reduced the serum level of vitamin K2 by 50%, indicating impaired production and absorption of vitamin K2.

Moreover, antibiotic treatment impaired cognitive functions, as shown by increased escape latency and decreased time spent in the target quadrant in the Morris water maze test. Antibiotic treatment also induced hippocampal damage, oxidative stress, inflammation, and apoptosis, as indicated by increased neuronal degeneration, lipid peroxidation, nitric oxide, TNF-α, IL-6, IL-1β, caspase-3, Bax/Bcl-2 ratio, and TUNEL-positive cells. On the other hand, vitamin K2 supplementation prevented these adverse effects of antibiotic treatment [10]. Vitamin K2 supplementation restored the gut microbiota composition and diversity to normal levels. Vitamin K2 supplementation also increased the serum level of vitamin K2 by 100%, indicating enhanced production and absorption of vitamin K2.

CONCLUSION

Vitamin K2 supplementation also protected the hippocampus from damage, oxidative stress, inflammation, and apoptosis, as indicated by decreased neuronal degeneration, lipid peroxidation, nitric oxide, TNF-α, IL-6, IL-1β, caspase-3, Bax/Bcl-2 ratio, and TUNEL-positive cells. So, it can be concluded that vitamin K2 improved cognitive skills, avoided hippocampus neuronal damage from antibiotics, and lowered intestine and brain inflammation and oxidative stress. It provides evidence for

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the neuro-protective effect of vitamin K2 against gut dysbiosis and neuroinflammation. It also suggests that vitamin K2 supplementation may be a potential therapeutic strategy for preventing or treating cognitive impairment caused by gut dysbiosis. However, further studies are needed to confirm the findings and to elucidate the underlying mechanisms of action of vitamin K2 on the gut-brain axis.

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