Ulva pertusa modulates DNBS-induced colitis and immune responses in mice

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Outline

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2. *Ulva pertusa*, a Marine Green Alga, Attenuates DNBS-Induced Colitis Damage via NF-κB/Nrf2/SIRT1 Signaling Pathways
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Abstract

Inflammatory bowel disease (IBD) is closely associated with abnormal interactions between intestinal microflora and environmental factors, and its main clinical manifestations include abdominal pain and diarrhea. In recent years, natural products have been recognized as multi-target and safer adjunctive therapeutic strategies, among which *Ulva pertusa* has demonstrated anti-inflammatory and antioxidant potential. Although traditional single-target drugs are effective, their side effects, high costs, and limited ability to address multifactorial pathological mechanisms highlight the need for an intervention capable of simultaneously regulating inflammation, oxidative stress, and immune imbalance. *U. pertusa* may simultaneously influence NF-κB, SIRT1/Nrf2, and innate as well as adaptive immune signaling pathways. Theoretically, *U. pertusa* could relieve inflammation, oxidative stress, and pain concurrently, making it a promising natural candidate worthy of systematic investigation. A mouse colitis model was induced by DNBS (4 mg dissolved in 100 μL of 50% ethanol, administered intrarectally), and *U. pertusa* extract (10, 50, and 100 mg/kg) was administered daily by oral gavage. Clinical disease activity and colonic pathological injury were evaluated, and parameters related to inflammation (such as the NF-κB axis and TLR4/NLRP3), antioxidation (SIRT1/Nrf2), apoptosis (p53, Bax, Bcl-2, Caspase), pain behaviors, and immune cell responses were measured. High doses (50 and 100 mg/kg) of *U. pertusa* significantly reduced the cascade of colonic tissue injury and inflammation induced by DNBS, inhibited NF-κB activation, and concurrently enhanced the antioxidant SIRT1/Nrf2 axis. It also regulated the apoptotic pathway by

downregulating p53, Bax, and Caspase while upregulating Bcl-2, thereby demonstrating anti-inflammatory, antioxidative, and antiapoptotic effect. Under the same model, *U. pertusa* exhibited additional immunomodulatory and analgesic benefits by alleviating abdominal pain behaviors, correcting the overactivation of innate and adaptive immune responses, and modulating TLR4- and NLRP3-related inflammasome signaling, indicating its clinical relevance to immune dysregulation and pain symptoms in IBD. In the DNBS-induced colitis model, *U. pertusa* exerted multiple effects, including anti-inflammatory, antioxidative, immunomodulatory, and analgesic actions, through inhibition of NF-κB, activation of SIRT1/Nrf2, and regulation of TLR4/NLRP3 and apoptotic pathways. This multi-target mechanism provides a promising direction for developing natural adjuvant therapies for IBD, and future studies should focus on component standardization, dose–response relationships, and clinical validation.

Reference

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