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Dentistry

Perspective

Multifaceted Role of IL-33/ST2 from Autoimmune Diseases to Periodontal Health

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DESCRIPTION

The Interleukin-33/Suppression of Tumorigenicity2 (IL-33/ST2) axis is an important component of the immune system that influences various biological processes, including those related to oral health. This pathway involves the cytokine IL-33 and its receptor ST2, which together modulate immune responses and inflammation. Recent research has highlighted the significant role of this axis in oral diseases, particularly autoimmune disorders and periodontal conditions.

IL-33/ST2 axis overview

IL-33, a member of the interleukin-1 family, is produced by a variety of cells, including epithelial cells, endothelial cells and fibroblasts. It is released in response to cellular stress or injury and acts through its receptor, ST2, which is found on several immune cell types. The interaction between IL-33 and ST2 initiates a signaling cascade that influences immune cell activation and inflammatory responses.

In the context of autoimmune diseases and periodontal diseases, the IL-33/ST2 axis can significantly impact disease progression and severity. Understanding its role in these conditions can offer insights into potential therapeutic approaches.

IL-33/ST2 axis in autoimmune diseases

Autoimmune diseases occur when the immune system erroneously targets and attacks the body's own tissues. The IL-33/ST2 axis has been implicated in several autoimmune disorders, including Systemic Lupus Erythematosus (SLE), Rheumatoid Arthritis (RA) and Sjogren's syndrome.

Systemic Lupus Erythematosus (SLE): SLE is characterized by widespread inflammation and tissue damage. Elevated levels of IL-33 have been observed in the serum of SLE patients. This increase in IL-33 correlates with disease activity and severity. IL-33 can activate ST2-positive immune cells, leading to the production of pro-inflammatory cytokines that contribute to the systemic inflammation seen in SLE. Furthermore, IL-33 promotes the survival of autoreactive T cells, which exacerbates the autoimmune response.

Rheumatoid Arthritis (RA): RA is a chronic inflammatory condition affecting the joints. Research indicates that IL-33 levels are elevated in the synovial fluid of RA patients. The interaction of IL-33 with ST2 on immune cells in the joint environment enhances the production of inflammatory mediators, such as Tumour Necrosis Factor alpha (TNF-alpha) and IL-6, which drive joint inflammation and damage. Inhibition of the IL-33/ST2 axis in experimental models of RA has shown a reduction in disease severity, suggesting a potential therapeutic target.

Sjogren's syndrome: Sjogren's syndrome is an autoimmune disorder that primarily affects the salivary and lacrimal glands, leading to dryness of the mouth and eyes. Elevated IL-33 levels have been detected in the saliva and serum of patients with Sjogren's syndrome. The IL-33/ST2 interaction contributes to the inflammatory process in these glands, promoting the infiltration of immune cells and the subsequent glandular damage. Targeting IL-33 or ST2 could potentially mitigate the inflammatory response and alleviate symptoms in affected individuals.

IL-33/ST2 axis in periodontal diseases

Periodontal diseases, including gingivitis and periodontitis, are characterized by inflammation and tissue destruction in the oral cavity. The IL-33/ST2 axis plays a significant role in the pathogenesis of these diseases by influencing the immune response and tissue remodeling.

Gingivitis: Gingivitis is the earliest stage of periodontal disease and is marked by inflammation of the gingiva. IL-33 is upregulated in gingival tissues during gingivitis and its expression correlates with disease severity. The IL-33/ST2 pathway enhances the recruitment and activation of immune cells, such as T Helper 2 (T^H2) cells, which release cytokines that exacerbate the inflammatory response. This chronic inflammation can lead

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to the progression from gingivitis to more severe periodontal conditions.

Periodontitis: Periodontitis represents a more advanced stage of periodontal disease, involving deeper periodontal tissue destruction and bone loss. IL-33 levels are elevated in the periodontal tissues of patients with periodontitis. The interaction of IL-33 with ST2 on immune cells in the periodontal environment contributes to the chronic inflammation and tissue degradation observed in this condition. IL-33 promotes the production of Matrix Metalloproteinases (MMPs), enzymes that degrade extracellular matrix components and lead to tissue destruction. Additionally, IL-33 enhances the proliferation of osteoclasts, cells responsible for bone resorption, further contributing to alveolar bone loss.

Therapeutic implications

Given the role of the IL-33/ST2 axis in autoimmune and periodontal diseases, targeting this pathway presents a potential strategy for therapeutic intervention. Research into inhibitors of IL-33 or ST2 is ongoing, with the goal of mitigating the inflammatory responses associated with these diseases.

Autoimmune diseases: For autoimmune conditions like SLE, RA and Sjogren's syndrome, modulating the IL-33/ST2 axis could reduce systemic inflammation and tissue damage. Potential therapies could include monoclonal antibodies targeting IL-33 or ST2, or small molecules that inhibit their interaction. Clinical trials are necessary to evaluate the efficacy and safety of these approaches.

Periodontal diseases: In periodontal diseases, targeting the IL-33/ST2 axis could help control inflammation and prevent tissue destruction. Possible treatments might involve local delivery of IL-33 inhibitors to the periodontal tissues or systemic administration of drugs that block ST2 signaling. Such therapies could potentially improve outcomes for patients with chronic periodontal conditions and reduce the need for invasive procedures.