Mediators of Inflammation

Hindawi

Special Issue on

IL-17 and Related Cytokines Involved in Autoimmune Diseases: Present and Future Perspectives

CALL FOR PAPERS

Autoimmune disorders encompass a wide range of diseases, including rheumatic, dermatologic, and gastroenteric. With over 80 autoimmune diseases currently identified, the key feature amongst the majority of them is the inability of the immune system to turn off processes targeted against healthy tissue following inflammatory responses.

Regardless of the type of disease in question, there are two common elements in all these conditions: firstly, the attack of T cells against self-antigens and the activation of B cells to produce autoantibodies against host tissue; secondly, the production and systemic release of proinflammatory cytokines (e.g., IL-17, IL-22, IL-23, IL-35, and INF-α) and chemokines that amplify the inflammatory milieu at the site of injury.

Interleukin-17 (IL-17), a hallmark cytokine of T-helper 17 (Th17) cells, plays critical roles in host defense against bacterial and fungal infections, as well as in the induction of inflammatory response aimed at protecting our body against invading pathogens. However, increasing preclinical and clinical evidences have demonstrated that IL-17 (also known as IL-17A) possesses a dark side contributing to the tissue destruction that occurs in chronic inflammatory and autoimmune diseases such as psoriasis, rheumatoid arthritis, and multiple sclerosis. These phenomena seem to be correlated with the capability of IL-17 to amplify the inflammatory scenario through synergy with other cytokines and chemokines. The revolutionary discovery that IL-17-producing T cells were central drivers in autoimmune diseases has positioned this cytokine as promising therapeutic target.

Another T cell subset with indispensable role in immune responses is the T regulatory cell (Treg). These cells possess extremely powerful and pleiotropic means to control immune responses and we know that, at least in some cases, their absence or inability to work fuels the autoimmune mayhem. Interestingly enough, we now know that Tregs and Th17 are cross-regulated mainly by the relative contributions of cytokines IL-6 and TGF-β. While cellular immunotherapy for autoimmune diseases still represents a promising way forward (e.g., using T regulatory cells), in order to develop efficient long-lasting treatments, we still have to conquer fundamental knowledge on the mechanisms of how these two subsets affect each other.

It is at this crossroad that we seek to invite investigators to submit high quality, original research articles, centered on preclinical research regarding the role of IL-17 or Tregs and related cytokines in autoimmune diseases. Review articles that discuss recent discoveries in basic research are also encouraged.

Potential topics include but are not limited to the following:

- IL-17 family members and related cytokines/chemokines as targets for therapy of autoimmune diseases
- Th17/Treg cells involved in disease pathogenesis
- Biomarkers which can predict Th17/Treg dysfunction in autoimmune disease
- Involvement of innate cells (e.g., neutrophils) in the production and/or regulation of Th17/Treg cytokines

Authors can submit their manuscripts through the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/mi/miad/.

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