OPEN ACCESS Freely available online

ISSN: 2471-2663
Clinical & Medical Biochemistry

Commentry

Composition of Autoimmune Disorders in T cells and B cells

Sander Vernino*

Department of Immunology, New York University School of Medicine, New York, USA

DESCRIPTION

The immune system is a collection of organs and cells that help keep us healthy. It does this by distinguishing between what we call our self (our own normal cells and tissues) and what makes us sick, such as bacteria, viruses and fungi. We call these pathogens. The immune system can also recognize and destroy cancer. B lymphocytes or B cells are effectors of humoral immunity and provide defense against pathogens through a variety of functions, including antibody production. B cells constitute approximately 15% of peripheral blood leukocytes and arise from hematopoietic stem cells in the bone marrow. This is where their antigen receptors (surface immunoglobulins) are assembled.

B cells, along with T cells, form the core of the adaptive arm of the immune system. They are continuously generated throughout the lifecycle of the organism in the primary lymphoid tissue, the bone marrow, from hematopoietic stem cell progenitors that undergo successive developmental stages. Each developing B cell expresses its own B Cell Receptor (BCR), which is composed of two identical heavy chain proteins and two identical light chain proteins. In both humans and rodents, when it is confirmed that developing B cells express the correctly assembled BCR on the cell surface and do not express the BCR, immature IgM B cells leave the bone marrow and enter the blood and move towards the spleen.

Through these mechanisms, B cells are involved in both autoimmune diseases traditionally thought to be antibody-mediated and autoimmune diseases commonly classified as T-cell mediated. This new understanding of the role of B cells has opened up new therapeutic options for the treatment of autoimmune diseases. This mechanism involves in different roles of the B cells in autoimmunity. B-cell involvement in systemic lupus erythematosus, rheumatoid arthritis, and Type 1 diabetes and current B-cell-based therapies. However, an increasing understanding of the complexity of the immune system has had a profound impact on how we view the autoimmune diseases and their pathogenesis. The reciprocal roles of T cell help for B cells during adaptive immune responses and B cell help in $CD4^+$ T cell activation are increasingly recognized. The observation that most autoantibodies in conventional autoantibody-mediated diseases are of her IgG isotype and harbor somatic mutations strongly suggests that T cells serve autoimmune B cell responses. Similarly, B cells serve as important antigen-presenting cells in autoimmune diseases traditionally thought to be T-cell mediated.

A key feature of autoimmune diseases is loss of B cell resistance and inappropriate production of autoantibodies. Over 80 different autoimmune diseases have been described, including Multiple Sclerosis (MS), Rheumatoid Arthritis (RA), and Systemic Lupus Erythematosus (SLE). Clonally silent B cells escape cell death and can be induced to proliferate and secrete autoreactive antibodies in otherwise healthy individuals as part of random events such as viruses that induce strong activation signals. Although B-cell-derived cytokines normally drive autoreactive T-cell responses during autoimmune development, a subpopulation of B-cells, also known as regulatory B-cells, act as anti-inflammatory cytokines. There is a compelling evidence that immune responses are negatively regulated (IL-10, IL-35, etc.) or contact dependent. Although B-cell depletion may eliminate most circulating B cells in the periphery, the clinical outcome of B-cell depletion therapy in autoimmune diseases is limited to B-cell activation or activation induced by the provided tissue microenvironment. It varies from person to person because survival signals are different.

CONCLUSION

Autoimmune diseases can be mediated by autoantibodies and/or autoreactive T cells, and tissue damage can result from direct attack on antigen-bearing cells, immune complex formation, or local inflammation. Autoimmune diseases caused by antibodies that bind to cellular receptors and cause overactivation or inhibition of receptor function fall into a special class. T cells may be directly involved in inflammation and cell destruction, and are also required to maintain autoantibody responses. Similarly, B cells are critical for maintaining autoantigen-specific T cell responses. It can be an antigen-presenting cell.

Correspondence to: Sander Vernino, Department of Immunology, New York University School of Medicine, New York, USA, E-mail: Vernino_S@hotmail.com

Received: 02-Jan-2023, Manuscript No. CMBO-23-19788; **Editor assigned:** 05-Jan-2023, PreQC No. CMBO-23-19788 (PQ); **Reviewed:** 19-Jan-2023, QC No CMBO-23-19788; **Revised:** 26-Jan-2023, Manuscript No. CMBO-23-19788 (R); **Published:** 02-Feb-2023, DOI: 10.35841/2471-2663.23.9.145

Citation: Vernino S (2023) Composition of Autoimmune Disorders in T cells and B cells. Clin Med Bio Chem. 9:145.