OPEN ACCESS Freely available online

Lupus: Open Access

Perspective

Impact of Interferons in Systemic Lupus Erythematous

Lars Boni*

Department of Medicine and Health Science, University of Gothenburg, Gothenburg, Sweden

DESCRIPTION

Systemic Lupus Erythematosus (SLE) is a complicated autoimmune illness characterized by tissue inflammation, which manifests as rashes, inflammatory arthritis, serositis, glomerulonephritis, central nervous system involvement, and hematological abnormalities. SLE is more common in women of childbearing age, with a 9:1 female to male ratio, and it is more common in black and hispanic women. Genome Wide Association Studies (GWAS) have played an important role in finding SLE susceptibility genes in various ancestral cultures. Additional risk factors for illness development include environmental, hormonal, and infectious variables. Treatment methods for SLE patients vary depending on illness symptoms and activity, but the goal is to achieve remission or low disease activity using a combination of immunomodulatory and immunosuppressive medicines.

Hydroxychloroquine, methotrexate, azathioprine, mycophenolate mofetil, and cyclophosphamide are examples of commonly used treatments. Biologics targeting specific cellular targets and cytokines are currently being used to treat SLE. Belimumab, a monoclonal antibody that suppresses the survival of autoreactive B lymphocytes and targets B Lymphocyte stimulator (BLys), is approved for use in a number of SLE symptoms, including lupus nephritis. Based on considerable preclinical and clinical research, the type I Interferon (IFN) pathway has now emerged as a new therapeutic target. SLE is distinguished immunologically by abnormal autoreactive T and B cell activation, autoantibody synthesis against nuclear antigens, and immune complex formation. Furthermore, innate immunity plays an important role in the immunopathogenesis of SLE. Considerable emphasis has been placed on the generation of type I IFN by plasmacytoid Dendritic Cells (pDCs), which activates various immune cells and is essential for B-cell activation and autoantibody production. In SLE, there is increased interest in therapeutic targeting of type I IFNs. This review will go over the role of type I IFNs in SLE. Immunopathogenesis begins with a basic introduction of type I interferons and lupus murine models. It will also address translational studies in SLE patients that highlight the role of type I interferons in SLE, as well as more recent clinical trials that lead to the use of therapies targeting type I interferons in clinics for the treatment of SLE.

The phosphorylated STAT1-STAT2 heterodimer forms an essential complex in the cytosol with IFN-Regulatory Factor 9 (IRF9) called IFN-Stimulated Gene Factor 3 (ISGF3) in the canonical pathway. The ISGF3 complex enters the nucleus and binds to conserved regions in ISGs known as IFN-Stimulated Response Elements (ISREs) to activate transcription of antiviral and antibacterial genes. IFN can also communicate *via* non-canonical pathways including STAT1 homo dimers that translocate straight to the nucleus and bind to activated sequences in gene promoters. IFN-signalling frequently employs sequences. IFN may also communicate with other STATs, such as STAT3, STAT4, and STAT5, which are involved in cytokine signalling pathways. Several reviews have gone into great length about non-canonical IFN signalling pathways and their effects.

Type I IFN signalling has a wide range of impacts. Hundreds of ISGs activated by IFN have been discovered at the transcriptional level using microarrays and, more recently, RNA sequencing experiments. IFN stimulated the human fibrosarcoma cell line HT1080. There were 40 known ISGs and 82 unique genes among the 122 ISGs that regulated apoptosis, angiogenesis, protein kinase receptors, and synthetases, among other things. Their research uncovered over 300 genes that control biological activities such as adhesion, death, immunological regulation, and cell proliferation.

CONCLUSION

The IFN pathways play an important role in the pathogenesis of SLE and have sparked interest in the diagnosis, prognosis, and therapy of SLE patients. Further research is needed, however, to determine the extent to which IFN gene signatures can be exploited in clinical practice. The discovery of anifrolumab underlines the promise for targeting type I IFN in the treatment of SLE, but more research on anifrolumab and other treatments targeting type I IFN is needed to establish how far targeting type I IFN will be used in the treatment of SLE.

Correspondence to: Lars Boni, Department of Medicine and Health Science, University of Gothenburg, Gothenburg, Sweden, E-mail: lars.boni53@yahoo.com

Received: 13-Feb-2023, Manuscript No. LOA-23-22222; **Editor assigned:** 15-Feb-2023, PreQC No. LOA-23-22222 (PQ); **Reviewed:** 06-Mar-2023, QC No. LOA-23-22222; **Revised:** 14-Mar-2023, Manuscript No. LOA-23-22222 (R); **Published:** 23-Mar-2023, DOI: 10.35248/2684-1630.23.8.221

Citation: Boni L (2023) Impact of Interferons in Systemic Lupus Erythematous. Lupus: Open Access. 8:221.