

NCCS

GUEST SEMINAR



**Environmental factors are
mechanisms of hematopoietic
dysregulation in lupus pathogenesis**

Dr. Neelakshi Jog

Principal Scientist

Translational Immunology

Bristol Myers Squibb, USA

18 April 2023 (Tuesday)

10 AM

Venue: Charaka

Dear all,

A guest seminar has been scheduled as given below -

Title of the talk: 'Environmental factors are mechanisms of hematopoietic dysregulation in lupus pathogenesis'

Date: **18 April 2023** (Tuesday) Time: **10 AM** Venue: **Charaka**

Speaker: **Dr. Neelakshi Jog** (Principal Scientist -Translational Immunology, Bristol Myers Squibb, USA)

Speaker's LinkedIn profile: <https://www.linkedin.com/in/neelakshi-jog-ph-d-9bbaa14>

Abstract:

Systemic lupus erythematosus (SLE, lupus) is a prototypical systemic autoimmune disease, which remains a major cause of early mortality in young women of childbearing age. SLE is characterized by autoantibody production, multi-organ involvement and periods when disease symptoms worsen (flares). The underlying cause of exacerbation of autoimmune inflammatory response in SLE remains unclear and hinders development of effective treatment options. Although environmental factors, such as Epstein-Barr virus (EBV) infections along with genetic factors are implicated in the disease onset, how viral infections contribute to cellular dysfunction required to generate the autoimmune response in SLE is unknown. A possible contribution of neutrophils along with dysregulated adaptive immune responses to lupus flares has been suggested although the precise mechanisms are unclear. A granulopoiesis gene signature has been reported in SLE patients, however, whether SLE patients have dysregulated granulopoiesis and whether this is a result of the inflammatory burden is not known. Using data derived from lupus patients and unaffected donors, we will discuss the role of EBV, as a model of viral infection, in initiation of autoimmune response. We will also discuss how the initial autoimmune response can influence the generation and dysfunction of neutrophils (granulopoiesis); which can cascade into an uncontrolled inflammatory response, resulting in SLE disease flares and damage. These studies will lead to a better understanding of the underlying mechanisms in lupus flares and identify new therapeutic targets and biomarkers.