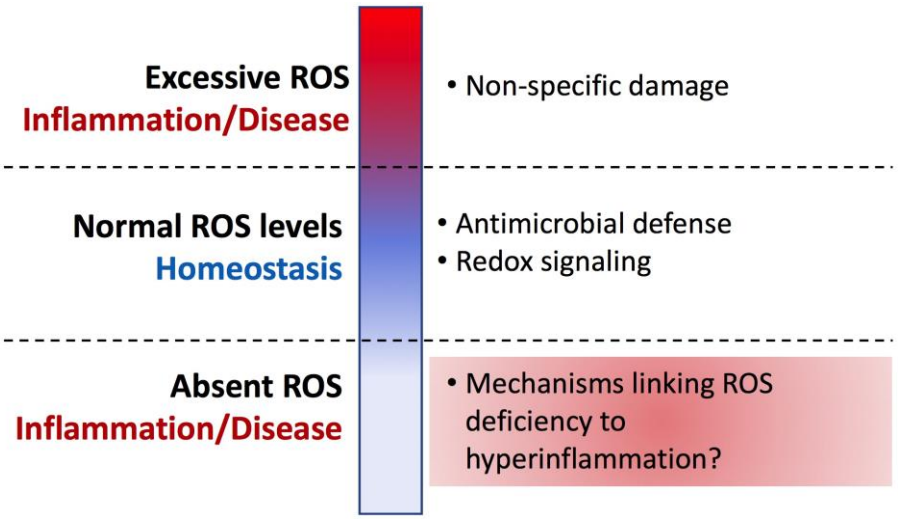
Bagaitkar Lab

Periodontal diseases are highly prevalent, chronic inflammatory disorders caused by dysregulated host responses to microbial colonizers, resulting in the progressive destruction of the tooth-supporting tissues of the periodontium. Persistent or aberrant activation of immune effector pathways by gingival neutrophils (PMN) and macrophages (MΦ) is one of the underlying factors in the immunopathology of periodontitis. However, we understand very little about the cell intrinsic molecular mechanisms that limit or restrain over-activation of these pathways.



The phagocyte NADPH oxidase is a multi-subunit enzyme that generates superoxide, precursor to anti-microbial ROS. Independent of their anti-microbial function NADPH oxidase derived-ROS are increasingly being recognized to play important role in several cellular processes. Our previous work demonstrated that the NADPH oxidase plays a pivotal role in regulating the magnitude, duration and nature of host inflammatory responses to various endogenous and microbial ligands.

However the underlying mechanisms are incompletely understood. Using novel genetic mouse models with targeted deletion of NADPH oxidase functions we are teasing out the relative contributions of neutrophil versus macrophage- derived ROS in regulation of host inflammatory pathways relevant in the oral mucosa and periodontal diseases. Our long-term goals are to understand the role of NADPH oxidase as a key regulator of immune effector responses.