



राष्ट्रीय प्रतिरक्षाविज्ञान संस्थान National Institute of Immunology

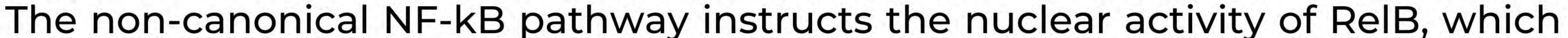
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GRADUATE STUDENT SEMINAR



INVESTIGATING THE DENDRITIC CELL-INTRINSIC ROLE OF THE ReIB-NF-kB PATHWAY IN INSTRUCTING GUT IMMUNITY

NAVEEN KUMAR SYSTEMS IMMUNOLOGY LABORATORY

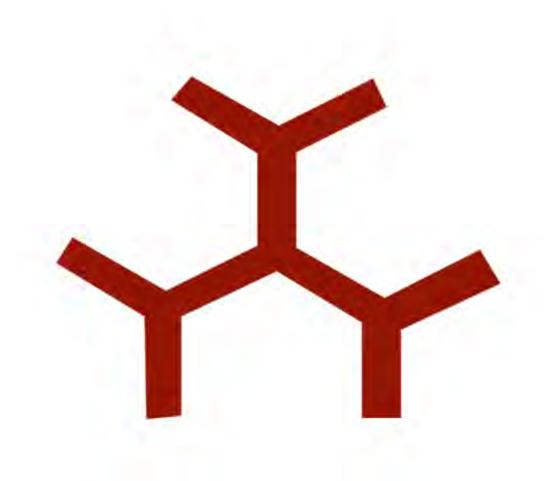


orchestates differentiation and maturation of immune cells, including dendritic cells (DCs). DCs are known to balance intestinal homeostasis and immunity. If DC-intrinsic RelB activity also modulates gut immunity remains unclear. Here, we report that the genetic ablation of the RelB in DCs accumulated regulatory T cells in the gut, augmented gut luminal IgA, and promoted eubiosis, thereby restraining experimental colitis in mice. However, a deficiency of RelB in DCs also compromised immunity against the gut pathogen Citrobacter rodentium. Our ex vivo studies explained that impaired RelB signaling augmented the abundance of β -catenin owing to the reduced synthesis of Axin1, which directs β -catenin for degradation, and that β -catenin transcriptionally upregulated Raldh2, imparting a retinoic acid-dependent tolerogenic functions in these DCs. Taken together, we report a role of noncanonical RelB signaling in DCs in instructing immunity at the gut interface.

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GP TALWAR AUDITORIUM, NII







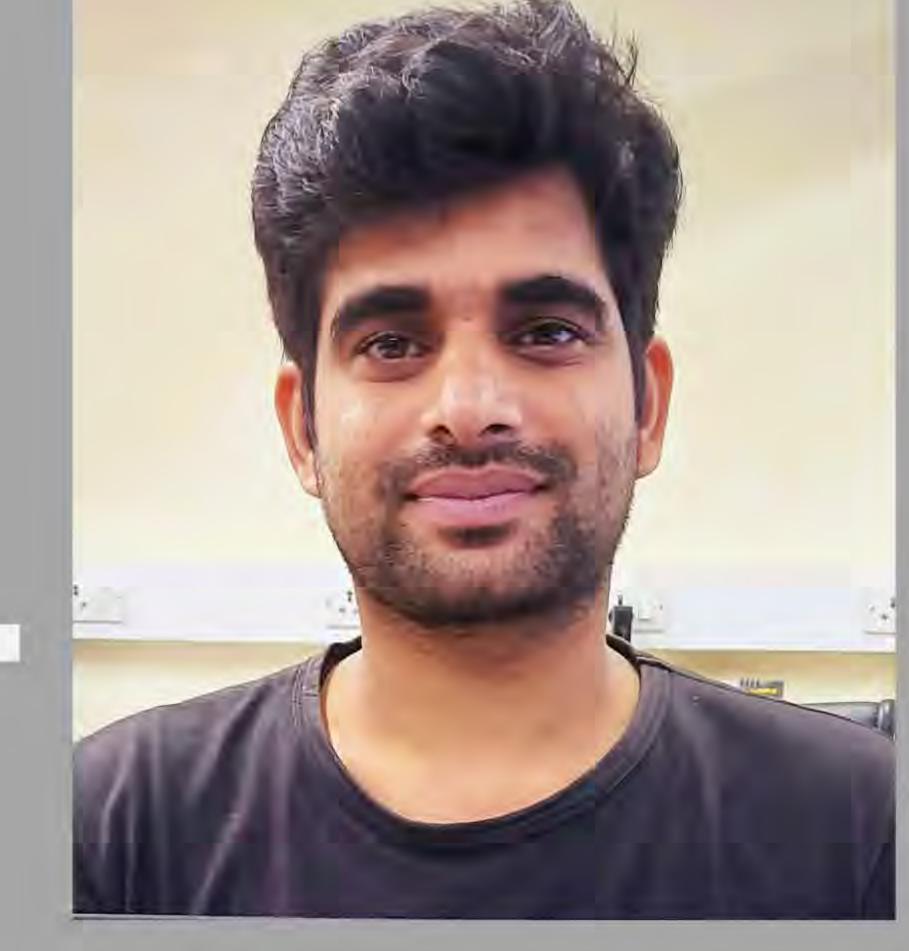
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GRADUATE STUDENT SEMINAR



METABOLIC PROFILE OF FOLLICULAR T HELPER (Tfh) CELLS

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Follicular T Helper (Tfh) cells are a unique subset of CD4+ T cells that are specialized for providing B cell help. Tfh cells are indispensable for devising the Germinal centres (GCs) and GC-derived humoral immunity. The Tfhlineage-specific transcriptomic landscape is well-defined. However, the metabolic pathways underlying the functionally potent Tfh cells are not established. Here, we applied the targeted and non-supervised approaches to identify the metabolic axes of human Tfh differentiation and function. Utilizing the human Tfh-cell differentiation model, we found that the inhibition of productive glycolysis led to the increase in Tfh differentiation mediated by BCL-6 driven program, but reduced B cell help function. We further observed that the compromised Tfh functions were associated with

reduced fatty acid utilization and suppressed fatty acid synthesis. Our study suggests a crucial role of fatty acid metabolism in conferring the potent function to Tfh cells.

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