

राष्ट्रीय प्रतिरक्षाविज्ञान संस्थान

National Institute of Immunology

GRADUATE STUDENT SEMINAR

INVESTIGATING THE DENDRITIC CELL- INTRINSIC ROLE OF THE RelB-NF- κ B PATHWAY IN INSTRUCTING GUT IMMUNITY



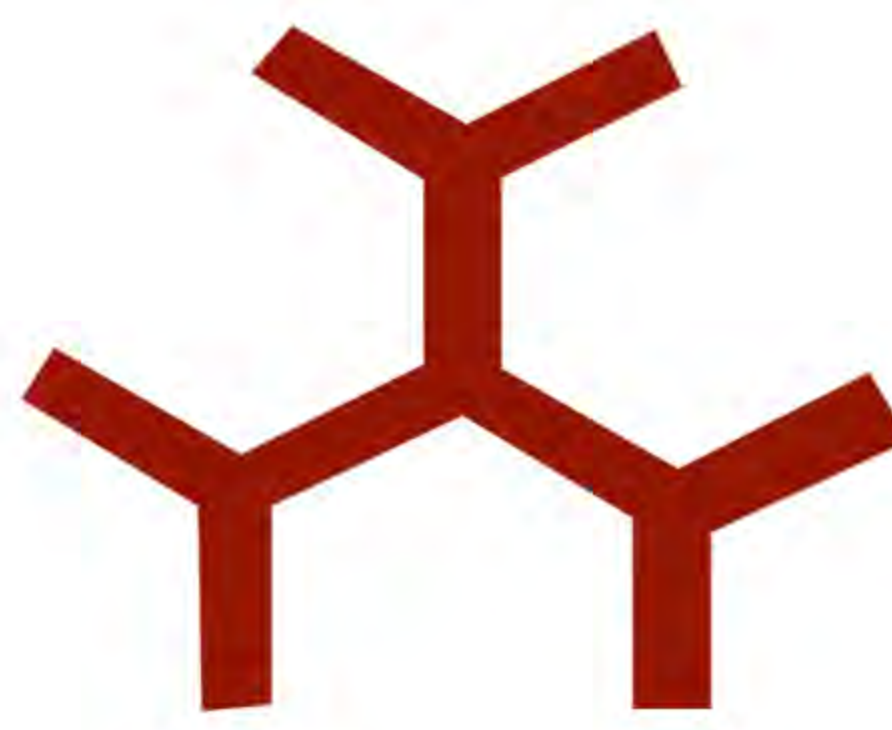
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The non-canonical NF- κ B pathway instructs the nuclear activity of RelB, which orchestrates differentiation and maturation of immune cells, including dendritic cells (DCs). DCs are known to balance intestinal homeostasis and immunity. If DC-intrinsic 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.

20 JULY 2023, 4.00 PM

GP TALWAR AUDITORIUM, NII



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**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 Tfh-lineage-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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