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"The Role of CD301b$^+$ Dendritic Cells in Coordinating Innate and Adaptive Immune Responses to Helminth Infection"

By

Alejandro Dávila Pagán
Cell Biology, Neuroscience and Physiology Program
B.S. University of Puerto Rico, Río Piedras Campus, 2016

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Department of Pathology, Immunology and Laboratory Medicine

Friday, March 28th, 2025
Cancer Center, G1196
12:00 P.M.

Join Zoom presentation

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ABSTRACT

Helminth parasites trigger robust type 2 immune responses characterized by eosinophilic inflammation, mucus production, tissue repair mechanisms, and the secretion of type 2 cytokines from both innate and adaptive immune cells. Although dendritic cells (DCs) play a well-established role in bridging innate and adaptive immunity by processing and presenting pathogen-derived antigens in type 1 and type 3 responses, their specific function in helminth infections remains less well understood. Previous studies demonstrated that DCs expressing CD301b (CD301b$^+$ DCs) are required for T helper type 2 (Th2) cell differentiation in skin- and lung-draining lymph nodes following infection with *Nippostrongylus brasiliensis* (*Nb*) in mice. However, their involvement in initiating innate type 2 inflammation and in host protection had not been established. In this study, I show that inducible depletion of CD301b$^+$ DCs during Nb infection results in markedly diminished type 2 immune responses in the lung and impaired worm expulsion. Notably, the reduction in type 2 immune response in the CD301b$^+$ DC-depleted mice extends beyond adaptive immunity, as CD4$^+$ T cell-independent innate type 2 inflammation is also compromised. Moreover, CD301b$^+$ DCs are essential for triggering an early neutrophil- and γδT cells-dependent type 3 inflammation, which in turn promotes the subsequent type 2 inflammation. In Rag1-deficient mice lacking T and B cells, the absence of CD301b$^+$ DCs abolishes *Nb*-induced inflammation altogether, confirming their critical role in regulating innate immunity. Mechanistically, CD301b$^+$ DCs are a critical source of IL-23 and CCL17, which then collectively mobilize γδT cells, neutrophils, and eosinophils. These data indicate that CD301b$^+$ DCs orchestrate both innate and adaptive immune responses against helminth infection, providing new insights into the regulation of immune pathways during such infection.