The Immune System, Inflammation, and Parasitic Worms

Intestinal helminth, or worm, parasites infect millions of people and animals worldwide and cause significant morbidity. The immune system reacts to the parasites with type 2 inflammation, characterized by activating certain immune cells and intestinal epithelial cell responses that lead to worm expulsion. Recent studies have revealed that basophils, a rare type of white blood cell, are key players in type 2 inflammation, but exactly how basophils function in this context remains unknown. The Notch signaling pathway, a molecular lock-and-key that can rapidly communicate messages of inflammation to a variety of cell types, is a potential candidate for controlling basophil responses following parasite infection.

In preliminary studies, the lab of Elia Tait Wojno, Microbiology and Immunology, showed for the first time that the Notch signaling pathway is active in basophils in a mouse model, following infection with Trichuris muris (T. muris), an intestinal parasite. They then demonstrated that Notch signaling contributes to optimal basophil function and helminth clearance following infection.

Tait Wojno’s group now hypothesizes that during intestinal helminth infection, basophils upregulate Notch and respond to Notch ligands, creating the conditions for optimal basophil function and efficient helminth expulsion. To test this, they are studying how the Notch signaling pathway regulates basophil population expansion and differentiation during type 2 inflammation caused by T. muris infection. They are also testing how Notch signaling promotes basophil function and contributes to type 2 inflammation in vivo and the role of Notch ligands or helminth proteases in this process. The results of these studies will expand understanding of intestinal type 2 inflammation and the role of basophils and will inform the development and use of therapies that target Notch to treat type 2 inflammatory diseases.

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