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EDITORIAL

Immunity and its role in white plague and obesity

Himanshu Kumar

Editor in Chief, International Reviews of Immunology & Principal Investigator, Laboratory of Immunology and Infectious Disease Biology, Indian Institute of Science Education and Research (IISER), Bhopal, India

Multilayers of host immunity ensure quick, efficient and specific elimination of microbial pathogens without perturbing commensal microbes and host immune homeostasis. The elimination of microbes is accomplished by various cellular and biochemical processes. These processes result in the formation of pores in the microbial surface, phagocytosis, masking the pathogenic molecules by host defense molecules or initiation of programed cell death of infected host cells. The majority of microbes are cleared by one or several processes triggered through sensing and a complex cascade of signaling to develop relevant effector immune responses. This issue of *International Reviews of Immunology* focuses on key immunological processes of innate immunity that lead to inflammation, resulting in the elimination of microbial infection. Another article also discusses the correlation among obesity, inflammation, and obesity-associated metabolic diseases and interactions of specialized immune cells with Mycobacterium tuberculosis (Fig. 1).

Natural immunity consists of various physical, biochemical, and cellular systems. These systems collectively inactivate or eliminate microbes, directly or indirectly through the recruitment of proteins known as complements on microbial surface, facilitating phagocytosis and/or inducing inflammation for the recruitment of professional immune cells at the site of infection. The first review article of this issue by Vijay Kumar discusses the complement system, Toll-like receptor family sensors, intracellular inflammasome pathway, and the crosstalk among them in terms of sensing and signaling. This article provides a deep insight into these phenomenon and

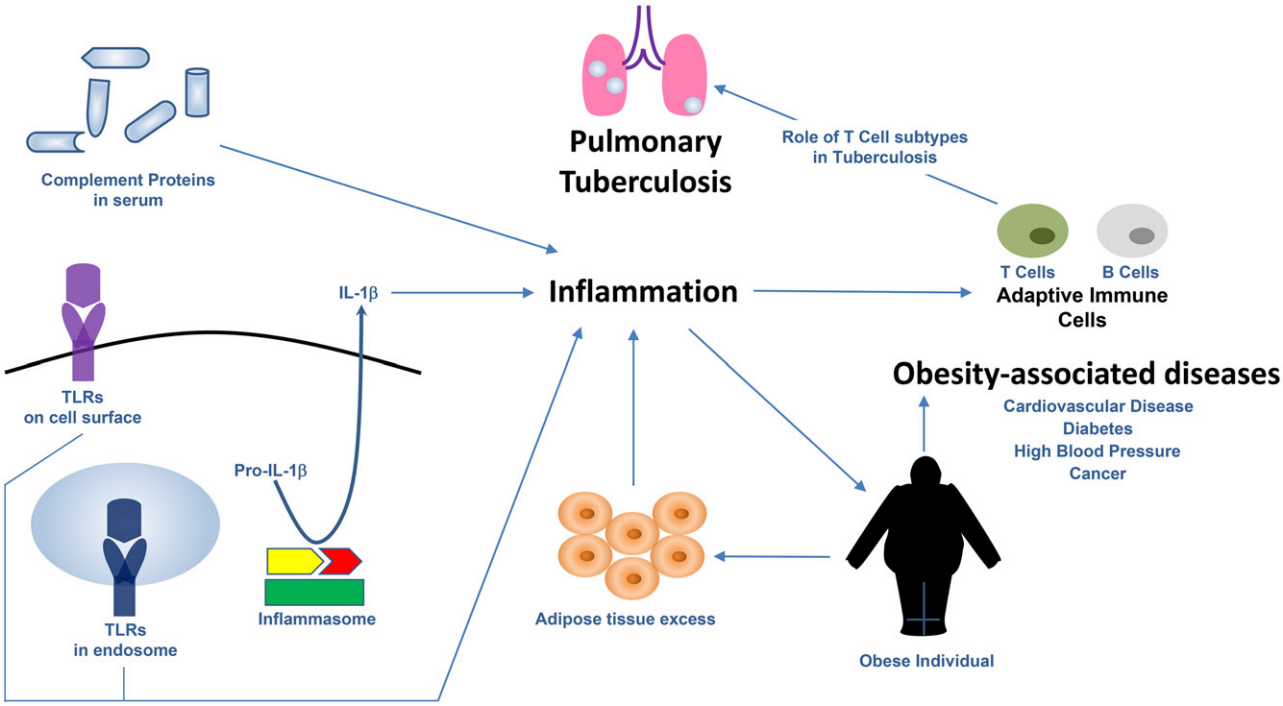

Figure 1. Role of host immune components in tuberculosis and obesity.

CONTACT Himanshu Kumar Himanshu.Kumar@taylorandfrancis.com Editor in Chief, International Reviews of Immunology & Principal Investigator, Laboratory of Immunology and Infectious Disease Biology, Indian Institute of Science Education and Research (IISER), Bhopal, India.
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discusses how sensing pathways develop effective pathogen clearance. This article is beneficial to fundamental and clinical immunology researchers working with various infectious diseases [1] (Fig. 1).

Individuals with a body mass index (BMI) equal to or more than 30 kg/m^2 fall in the category obese. Obesity severely affects the general health of individuals, affecting the musculoskeletal, circulatory system and overall metabolism. The second review of this issue by Singh et al. focus on how obesity dysregulates inflammation and culminates in diseases such as diabetes, cardiovascular diseases, stroke, and so on. Although the field is in its infancy, the article also discusses the molecular mechanism. However, extensive research is needed before attempting to find molecular targets to treat obesity and obesity-associated diseases. This article will be helpful to clinicians, dieticians, and fundamental and translational researchers working in immunology and metabolic diseases [2] (Fig. 1).

"White plague" or tuberculosis is an ancient infectious disease caused by Mycobacterium tuberculosis. This disease causes 1.3 million deaths annually and is reported as one of the top 10 causes of death worldwide by the World Health Organization [3]. The causative agent Mycobacterium tuberculosis gains access to the host through the respiratory tract, where it interacts and infects alveolar macrophages. These macrophages trigger the killing process within phagolysosome through the generation of free-radicals, and they also trigger adaptive immune responses via activation of subtypes of T cells. The third article of this issue by Abebe describes the role of subsets of T cells and other adaptive immune cells during Mycobacterium tuberculosis infection. This article will be useful to the broad classes of medical and immunology researcher [4] (Fig. 1).

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