Two tales of the polarizing immune responses: Th1-mediated host-microbe interaction in tuberculosis vs. Th2-driven childhood asthma

Tanapat Palaga

Immune responses are tailored to deal with invading pathogens while cause minimal damages to the host. In some settings, however, this balance is not maintained and the detrimental outcomes are manifested. Chronic inflammation after bacterial infections or hyper responsiveness to allergens are mediated by two opposing cytokine patterns. *Mycobacterium tuberculosis* (MTB) is an intracellular pathogen which infect mainly macrophages and dendritic cells. The protective immune response is mediated by cell-mediated immune responses mounted mainly by activated macrophages and helper T cells type 1 (Th1).[1] The pathogens can subvert the host immune response and remain persistent in the infected cells for a long time while host immune responses keep them in check. The interaction between host and bacteria at the molecular level and the outcomes of this interaction is thought to shed the light into how MTB survives. In this issue, Faksri et al. studied the MTB Beijing strain, a hypervirulent MTB, and the impact of infection in human pro-monocytic cell line.[2] MTB Beijing strains are phylogenetically divided into ancestral and modern sublineages.[3] The modern sublineage demonstrates higher virulent phenotype and increasing prevalence around the world. Upon infections, macrophages produce cytokines and anti-microbial molecules to control phagocytosed bacteria. Furthermore, cell death of the host is also reported and it is considered to be one mechanism of protective response.[4] The interesting finding reported here is that in contrast to the ancestral sublineage, the modern Beijing sublineage induced higher IL-10 production which is shown to mainly function as a brake for the host immune response. This result fits perfectly with a more virulent phenotype of the modern sublineage shown reported by others. When cell death was investigated, the modern sublineage triggers less necrotic cells death than the ancestral sublineage. Apoptosis of macrophages upon MTB infection helps not only to eliminate the source of bacterial growth but also apoptotic bodies are taken up by neighboring dendritic cells possibly for a cross presentation to CD8+ T cells. In contrast, necrotic cell death triggers inflammatory response and disseminates the bacteria better.[5] The findings by Faksri et al. indicate that the modern lineage of MTB maintains viability of infected cells while suppresses the host immune responses which may result in more persistent infection. Molecular mechanisms leading to this phenotype awaits further investigation.

Asthma is a complex chronic inflammatory and narrowing of the airways. Cytokines produced by Th2 cells and recently identified innate lymphoid cells such as IL-4, IL-5, IL-9 and IL-13, plays pivotal role in asthma.[6] In this issue, Kanchongkittiphon et al. reviewed the relationship between the indoor environment and inner-city childhood asthma. It was speculated that exposure to allergens and/or pollutants may exacerbate asthma condition.[7] The authors reviewed the literature extensively and concluded that inner-city children are exposed to various indoor allergens/pollutants that links to asthma development and exacerbation of existing disease. This conclusion implies that better control of indoor environment may help reducing the incidence or severity of the diseases. Accompanying this review article, two studies on asthma-related topics, the evaluation of the cartoon-aided instruction of intranasal corticosteroid administration technique among Thai children with allergic rhinitis[8] and the prevalence of obstructive sleep apnea in patients with difficult-to-treat asthma[9] are featured.

Also, in this issue, the review article on the pharmacogenetics of drugs-induced hypersensitivity reactions,[10] the research articles on the involvement of p53 and nitric oxide on cytokine-induced

From Department of Microbiology, Faculty of Science, Chulalongkorn University, Bangkok, Thailand 10330
E-mail: tanapat.p@chula.ac.th

apoptosis in human leukemic cell lines[11] and the use of herb to suppress the development of atopic dermatitis[12] are featured.

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