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Highlights in Mucosal Immunology

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"The Journal of Mucosal Immunology Research" covers broad scope on the frontiers of mucosal immunology.

The immune systems have elaborative mechanism to protect the body from life-threatening pathogens. The mucosal surfaces of upper respiratory and gastrointestinal tracts, reproductive organs and secretory glandular tissues, which can exceed 400 m^2 in humans, are particularly sensitive to infections. These organs regulate the functions of memory B cell, T cells and IgA producing plasma cells. An epithelium cell layer and intestinal microbial circumstances function as innate immunity and a physical barrier in defense against pathogens.

A mucosal immune system (MIS) has been provoked to maintain homeostasis against stressful elements in higher mammals. It might be independent from systemic immunity and the largest immune organ in human body. The mucosal membranes have the potential to secrete antibodies (IgA) and to inactivate pathogens. For mucosal immune system, Ag specific mucosal effector cells such as IgA producing plasma cells and memory B and T cells are essential. Specific immunocompetent cells such as B1-B cell, γδ T cell and NK22 cell support MIS. The amount of IgA produced in mucosal cells is more than 75% of the total amount of antibodies produced in the entire body. The mucosal inductive sites consist of mucosa associated lymphoid tissue (MALT), nasopharyngeal associated lymphoid tissue (NALT); gut associated lymphoid tissues (GALT), and the others.

Epithelial-derived transforming growth factor (TGF)-β1 has a pivotal effect during influenza A infection [1]. TGF-β1 regulates immune responses to suppress inflammatory reaction. Their study elucidated the relationship between local epithelial microenvironment and initial immune responses.

On the other hands, ulcerative colitis (UC) and Crohn's disease (CD) are main inflammatory bowel diseases (IBD) induced by disturbance of the MIS. Genetic factors, environmental microbial flora and the immune response are supposed to lead to IBD. NOX1 missense mutation associated with UC [2]. A rare NOX1 mutation is related with early stage of intestinal inflammation. In IBD, there are functional defects of the immune barriers. Structural changes are induced by inflammatory cytokines such as tumor necrosis factor (TNF), IL-13 and IFN-γ. Hence, anti-TNF antibody has been applied to the therapy of IBD and has been shown to improve IEC-mediated barrier function in IBD. Plasma cells of patients with IBD express Granzyme B (GrB) and show cytotoxicity [3]. GrB expressing CD19+ and IgA+ cells were significantly more frequent in IBD than in normal intestinal mucosa. CD19+ cells promote intestinal epithelial cell (IEC) apoptosis and IL-21 increase the ability of GrB expressing cells to induce IEC apoptosis.

In conclusion, the MIS is a key part of the overall immune system and plays a crucial role in higher mammals. It might be very effective to control mucosal barrier for preventing infectious and inflammatory diseases. Through mucosal immunology, we could advance the appropriate treatment of various diseases.

References

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