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Mucosal Immune System in Health and Disease

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Editorial

The system could also be viewed as an organ that's distributed throughout the body to supply host defense against pathogens wherever these may enter or spread. Within the system, a series of anatomically distinct compartments are often distinguished, each of which is specially adapted to get a response to pathogens present during a particular set of body tissues. The previous part of the chapter illustrated the general principles underlying the initiation of an adaptive immune response in the compartment comprising the peripheral lymph nodes and spleen [1]. This is the compartment that responds to antigens that have entered the tissues or spread into the blood. A second compartment of the adaptive immune system of equal size to this, and located near the surfaces where most pathogens invade, is the mucosal immune system (commonly described by the acronym MALT). Two further distinct compartments are those of the body cavities (peritoneum and pleura) and the skin. Two key features define these compartments. The first is that immune responses induced within one compartment are largely confined in expression thereto particular compartment. The second is that lymphocytes are restricted to particular compartments by expression of homing receptors that are bound by ligands, referred to as addressing that are specifically expressed within the tissues of the compartment [2-4]. We will illustrate the concept of compartmentalization of the system by considering the mucosal system. The mucosal surfaces of the body are particularly susceptible to infection. They are thin and permeable barriers to the inside of the body due to their physiological activities in gas exchange (the lungs), food absorption (the gut), sensory activities (eyes, nose, mouth, and throat), and reproduction (uterus and vagina). The necessity for permeability of the surface lining these sites creates obvious vulnerability to infection and it's not surprising that the overwhelming majority of infectious agents invade the human body through these routes.

A second important point to bear in mind when considering the immunobiology of mucosal surfaces is that the gut acts as a portal of entry to a vast array of foreign antigens in the form of food. The system has evolved mechanisms to avoid an active immune reaction to food antigens on the one hand and, on the opposite, to detect and kill pathogenic organisms gaining entry through the gut. To complicate matters further, most of the gut is heavily colonized by approximately 1014 commensal microorganisms, which sleep in symbiosis with their host [5]. These bacteria are beneficial to their host in some ways. They provide protection against pathogenic bacteria by occupying the ecological niches for bacteria within the gut. They also serve a nutritional role in their host by synthesizing vitamin K and some of the components of the vitamin B complex. However, in certain circumstances they can also cause disease, as we will see later [6, 7].

In order to know the phenomenon of oral tolerance, it's essential to know how orally delivered antigens are presented to T cells. Two routes of antigen presentation of soluble food antigens are characterized which will induce T-cell responses favoring tolerance instead of immune activation. The first is presentation of soluble food antigens by the antigen-presenting cells of the gut and other peripheral lymphoid organs. In the absence of inflammatory stimuli, antigen presentation by dendritic cells favors the induction of tolerance instead of T-cell activation. Dendritic cells in Peyer's patches have been shown to express IL-10 and IL-4, in contrast to similar cells in peripheral lymph nodes which express IFN-γ and IL-12. However, this heterogeneity of cytokine responses doesn't fully explain tolerance to food antigens [8]. These could also be detected within the bloodstream after feeding and there's evidence that the induction of tolerance to food antigens takes place in lymph nodes and spleen as well as in the mucosal lymphoid system. The second possible route of presentation of food antigens is by the enterocytes of the gut, which express MHC class I and MHC class II molecules within the absence of co-stimulatory molecules and thus may induce energy on presenting antigens to intraepithelial lymphocytes.

We will discuss each of these mechanisms of tolerance further, where we consider how the loss of tolerance to self-tissues may contribute to the development of autoimmune disease. As we will see, one of the strategies for treating allergy and autoimmune disease is to attempt to manipulate the nature of the antigen-specific response to stimulate T cells with the properties of such regulatory T cells [9, 10].

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Conflict of Interest

None

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