Oven Access



Commentary

Vaccines and Mucosal Immunity

Christopher Hull MD*

Department of Dermatology, University of Utah, Salt Lake City, Utah, 30 North 1900 East, Salt Lake City, UT 84132

There is currently great interest in developing mucosal vaccines against a spread of microbial pathogens. Mucosal induced tolerance also seems to be a promising form of immunomodulation for treating certain autoimmune diseases and allergies. Here we review the properties of the mucosal system and discuss advances within the development of mucosal vaccines for cover against infections and for treatment of varied inflammatory disorders.

The mucous membranes covering the aero digestive and the urogenital tracts as well as the eye conjunctiva, the inner ear and the ducts of all exocrine glands are endowed with powerful mechanical and chemical cleansing mechanisms that degrade and repel most foreign matter. In addition, a large and highly specialized innate and adaptive mucosal immune system protects these surfaces, and thereby also the body interior, against potential insults from the environment. In a healthy human adult, this local immune system contributes almost 80 % of all immunocytes. These cells are accumulated in, or in transit between, various mucosa-associated lymphoid tissues (MALT), which together form the most important mammalian lymphoid organ system [1].

The mucosal system has three main functions: (i) to guard the mucous membranes against colonization and invasion by potentially dangerous microbes which will be encountered, (ii) to stop uptake of undegraded antigens including foreign proteins derived from ingested food, airborne matter and commensal microorganisms, and (iii) to stop the event of probably harmful immune responses to those antigens if they do reach the body interior. At variance with the systemic immune apparatus, which functions during a normally sterile milieu and sometimes responds vigorously to invaders, the MALT guards' organs that are replete with foreign matter? It follows that upon encountering this plethora of antigenic stimuli; the MALT must economically select appropriate effector mechanisms and regulate their intensity to avoid bystander tissue damage and immunological exhaustion [2].

The MALT represents a highly compartmentalized immunological system and functions essentially independent from the systemic immune apparatus. It is comprised of anatomically defined lymphoid micro compartments like the Peyer patches, the mesenteric lymph nodes, the appendix and solitary follicles within the intestine, and therefore the tonsils and adenoids at the entrance of the aero digestive tract, which serve as the principal mucosal inductive sites where immune responses are initiated. Small but numerous clusters of immature lymphocytes and dendritic cells have also been described in the sub epithelial compartment of the mouse intestine and may represent sites of extrathymic lymphopoiesis; such crypto patches have not been found in humans, however. The MALT also contains diffuse accumulations of large numbers of lymphoid cells in the parenchyma of mucosal organs and exocrine glands, which form the mucosal effector sites where immune responses are manifested. Consistent with a high degree of compartmentalization, the MALT is populated by phenotypically and functionally distinct B cell, T cell and accessory cell subpopulations as compared with systemic lymphoid tissues, and has also developed strong restrictions upon lymphoid cell recirculation between mucosal sites [3].

Effector mechanisms

In addition to the barrier function, mechanical cleansing mechanisms and different chemical antimicrobial factors or defensins provided by the liner epithelium of various mucosal tissues, the mucosa contains variety of other cells of the innate immune system, including phagocytic neutrophils and macrophages, DCs, NK cells and mast cells. Through a variety of mechanisms these cells contribute significantly to host defense against pathogens and for initiating adaptive mucosal immune responses.

The adaptive humoral immune defense at mucosal surfaces is to a large extent mediated by secretory IgA (SIgA) antibodies, the predominant immunoglobulin class in human external secretions. The resistance of SIgA to proteases makes these antibodies uniquely suited for functioning in mucosal secretions (Box 1). The induction of IgA against mucosal pathogens and soluble protein antigens depends on T helper cells, although IgA immunity to commensal flora could also be thymus independent and of low affinity. In humans, transforming growth factor (TGF)- β and interleukin (IL)-10 in concert with IL-4 have been shown to promote B-cell switch to IgA and differentiation into IgA-producing cells [4].

Mucosal cytotoxic T lymphocyte (CTL) responses have been described after oral, nasal, rectal or vaginal immunization, and recently also after transcutaneous immunization31. Mucosal CTLs have been shown to be crucial for the immune clearance of pathogens in several animal models of infection with enteric or respiratory viruses and intracellular parasites. In most studies, wild-type or attenuated viruses and bacteria are required to induce CTLs in mucosal tissues. There are, however, exceptions to the present rule, inasmuch as use of certain adjuvants like cholera toxin and related enterotoxins can promote mucosal CTL development when administered orally or nasally with soluble proteins and peptides. Besides CTLs, interferon (IFN)-yproducing CD4+ T cells, induced either by the live pathogens or by mucosal immunization with inactivated vaccines together with cholera toxin or other mucosal adjuvants, have been found to be important for mucosal immune defense to both viral and bacterial infections; their protective mechanism (s), however, remain to be defined. Thus, appropriate adjuvants or delivery systems, or both, may critically favor the induction of protective mucosal cellular responses, and this notion is of importance for developing mucosal vaccines against intracellular pathogens.

*Corresponding author: Christopher Hull MD, Department of Dermatology, University of Utah, and 30 North 1900 East, Salt Lake City, UT 84132, E-mail: Christopher.hull@hsc.utah.edu

Received: 03-May-2022, Manuscript No. jmir-22-63173; Editor assigned: 05-May-2021, PreQC No. jmir-22-63173 (PQ); Reviewed: 21-May-2022, QC No. jmir-22-63173; Revised: 27-May-2022, Manuscript No. jmir-22-63173 (R); Published: 31-May-2022, DOI: 10.4172/jmir.1000147

Citation: Hull C (2022) Vaccines and Mucosal Immunity. J Mucosal Immunol Res 6: 147.

Copyright: © 2022 Hull C. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

For many years, mucosal immunity and mucosal vaccines have attracted but their due share of research and development, considering that the majority infections and environmental allergies have a mucosal portal of entry [2, 5].

But in recent years, methodological advances allowing more intense study of mucosal immune responses have led to growing interest in both trying to better understand the specific features of mucosal as compared with systemic immunity, and to develop mucosal vaccines for preventing mucosal infections and for treating allergic or autoimmune diseases. Methods that facilitate the monitoring of mucosal immune responses in humans including infants and young children-the major target groups for vaccination against infectious diseases-have been developed, primarily for measuring secretory antibody responses. But practical assays for assessing mucosal T cell reactivity in clinical and in field settings are still scarce and methods for predicting efficacy of candidate mucosal immunotherapeutics in humans are lacking [5].

Acknowledgement

None

Page 2 of 2

Conflict of interest

None

References

- Macpherson AJ, Macpherson AJ, Gatto D, Sainsbury E, Harriman GR, et al. (2000) A primitive T cell-independent mechanism of intestinal mucosal IgA responses to commensal bacteria. Sci 288: 2222-2226.
- Stoel M (2005) Restricted IgA repertoire in both B-1 and B-2 cell-derived gut plasma blasts. J Immunol 174: 1046-1054.
- Goodrich ME, McGee DW (1998) Regulation of mucosal B cell immunoglobulin secretion by intestinal epithelial cell-derived cytokines. Cytokine 10: 948-955.
- Asano T, Kaneko H, Terada T, Kasahara Y, N Kondo (2004) Molecular analysis of B cell differentiation in selective or partial IgA deficiency. Clin Exp Immunol 136: 284-290.
- Klavinskis LS, Bergmeier LA, Gao L, Mitchell E, Ward RG, et al. (1996) Mucosal or targeted lymph node immunization of macaques with a particulate SIVp27 protein elicits virus-specific CTL in the genito-rectal mucosa and draining lymph nodes. J Immunol 157: 2521-2527.