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Fundamental Aspects of Immunology in Microbiology

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Description

Immunity boosters protect by inducing effector mechanisms that can rapidly control replicating pathogens and inactivate their toxic components. The main role of B cells in the efficacy of current vaccines should not undermine the importance of T cell responses. T cells are essential for the induction of high affinity antibodies and immunological memory and directly help protect current vaccines such as Bacille Calmette Guérin (BCG). It may play a more important role than previously thought in certain diseases such as whoping cough and will be a major effector for new targets with major intracellular localization such as tuberculosis.

New methods have been developed that allow the assessment of more and more microbial-related immune parameters, including humans. This development raises new questions about the correlation between the optimal markers to evaluate and provide protection. Identifying the immune correlation of the mechanism, or at least the surrogate for microbial efficacy, is an essential advantage for the development of new microbes or the optimization of immune strategies with available microbes. Therefore, their destinations generate considerable interest.

Over the last decade, there has been increasing awareness of the complexity of the immune system and its determinants, including at the genetic level of the host, using systems biology approaches to various processes and networks in response to immunization. Evaluating the interaction of is an attempt to isolate and characterize several components of the vaccination response. Induction of antigen-specific immune effectors (and/or immunological memory cells) by the immunization process does not imply that these antibodies, cells, or cytokines represent or correlate vaccine efficacy. This requires formal evidence that vaccine-mediated protection of vaccinated individuals depends on the presence of specific markers, such an antibody titers and the number of antigen-specific cells above a specific threshold. B cells are essentially activated in the lymph nodes that drain the injection site.

The microbial antigens that reach the subcapsular sinus by diffusion of free fluid are taken up by specific subcapsular sinus macrophages and migrate to the B cell zone. B cells with surface B cell receptors 49 capable of binding vaccine antigens are activated and migrate to the interface between B cells (follicles) and the T cell zone. There, B cells attack T cells and begin their proliferation. The cumulative amount of co-stimulation signals received by B cells determines the fate of B cells. Fifty protein antigens (incorporated and presented as small peptides on the surface of APC) activate TFH cells. It induces a highly efficient B cell differentiation pathway through a specific structure Germinal Center (GC) in which antigen-specific B cells proliferates and differentiates into antibody-secreting plasma cells or memory B cells.

Polysaccharide antigens that do not mobilize TFH cells for the reaction do not trigger GC, so they only trigger short-lived plasma cells, resulting in a weaker, less persistent antibody response with no immunological memory. Proof that B memory cells persist long after the vaccine antibody disappears and their rapid reactivation upon exposure to the antigen have a direct impact on the immunization program. First, it means that the immunization schedule should not be restarted from the beginning, but should be resumed from where it was interrupted, regardless of the length of the interruption. Second, it means that if a person is regularly exposed to natural booster immunity, it may not be necessary to include booster doses in a particular immunization schedule. Without additional childhood immunization, up to 50% of adolescents or young adults were immunized against tetanus or hepatitis B in infancy may have no history of response and infant memory-induced immune forever. Please note that it may not continue.

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