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Mediate Protection and Age-Associated Changes in Vaccine Responses

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Editorial

Vaccines protect by inducing effector mechanisms capable of rapidly controlling replicating pathogens or inactivating their toxic components. Vaccine-induced immune effectors are essentially antibodies—produced by B lymphocytes—capable of binding specifically to a toxin or a pathogen. Other potential effectors are cytotoxic CD8+T lymphocytes that may limit the spread of infectious agents by recognizing and killing infected cells or secreting specific antiviral cytokines and CD4+ T-helper (Th) lymphocytes [1]. These Th cells may contribute to protection through cytokine production and provide support to the generation and maintenance of B and CD8+ T-cell responses. Effector CD4+Th cells were initially subdivided into T-helper 1 (Th1) or T-helper 2 (Th2) subsets depending on their main cytokine production, respectively. This dichotomy became outdated as Th cells were increasingly shown to include a large number of subsets with distinct cytokine-producing and homing capacities [2]. A recently identified critical subset of vaccine-induced CD4+Th cells are follicular T-helper (Tfh) cells: they are specially equipped and positioned in the lymph nodes to support potent B-cell activation and differentiation into antibody secreting-cells and were identified as directly controlling antibody responses and mediating adjuvant city. Another important subset are T-helper 17 (Th17) cells which essentially defend against extracellular bacteria that colonize the skin and mucosa, recruiting neutrophils and promoting local inflammation. These effectors are controlled by regulatory T cells (Tregs) involved in maintaining immune tolerance. Most antigens and vaccines trigger B- and T-cell responses, such that there is no rationale in opposing vaccines favouring antibody production and T-cell responses. In addition, CD4+T cells are required for most antibody responses, whereas antibodies exert significant influences on T-cell responses to intra-cellular pathogens. Age-associated changes in T-cell responses are reflected by a progressive decline in naïve T cells, reflecting declining thymic output. This is associated with a marked accumulation of large CD8+clones presumably resulting from prior infections [3]. These large T-cell clones have reached a state of replicative senescence, and homeostatic mechanisms negatively influence the size of the naïve and effector memory T-cell subsets.289 In response to influenza immunization, healthy elderly people mount CD4+responses initially similar to those of young adults but that fail to maintain or expand.306 This does not reflect a functional impairment of CD4+T memory cells, but a shift of the T-cell pool from naïve to memory effector CD4+ T cells. The failure to maintain CD4+responses reflects a lower induction of new Tem cells in relation to lower IL-7 levels. Other studies indicated that frail elderly subjects mount blunted and delayed Th1 responses to influenza vaccination, which correlated positively with their reduced total and IgG1 antibody response.308 Limitations also affect the expansion of infection-driven influenza-specifc CD8+T cells.308 Strategies to enhance vaccine-induced protection in aging people include the use of higher vaccine doses 309 and/or specific adjuvants [4]. This was recently demonstrated by formulating the IgE glycoprotein of varicella-zoster in the novel AS01E adjuvant. Nevertheless, limitations of effector memory and of GC responses may continue to require the more frequent administration of certain vaccine boosters to compensate for the brevity of B- and T-cell vaccine-induced responses in elderly people. Innate and adaptive antibody and T-cell–mediated cellular immune responses decline with age, which increases the frequency and severity of infections and reduces the protective effects of vaccinations [5].

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Conflict of interest

None

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