Re-thinking Vaccinology: “ Act Universally, Think NK Cells ” ?

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Received date: August 05, 2017; **Accepted date:** August 23, 2017; **Published date:** September 7, 2017

encoded Bc target epitopes, for example due to spatial rearrangements (i.e., in case of conformational epitopes) or a spontaneous genetic mutation or recombination, may already su f ce for the pathogen to escape a previously naturally induced or vaccine-mediated Ab response. Ab-based vaccines may, therefore, fail to induce protection against distinct, although phylogenetically related, pathogen strains/ serotypes. In addition, immune recognition of cognate T help (T) epitopes may be poor or lacking in a subset of vaccine recipients (so-called 'non-responders') due to MHC polymorphism, thus resulting in the absence of functionally protective Ab responses. Due to the above-mentioned limitations, pathogens may succeed in escaping the host immune response and preserve their f tness. Although immune escape can to some extent be mitigated by enhanced T help through co-

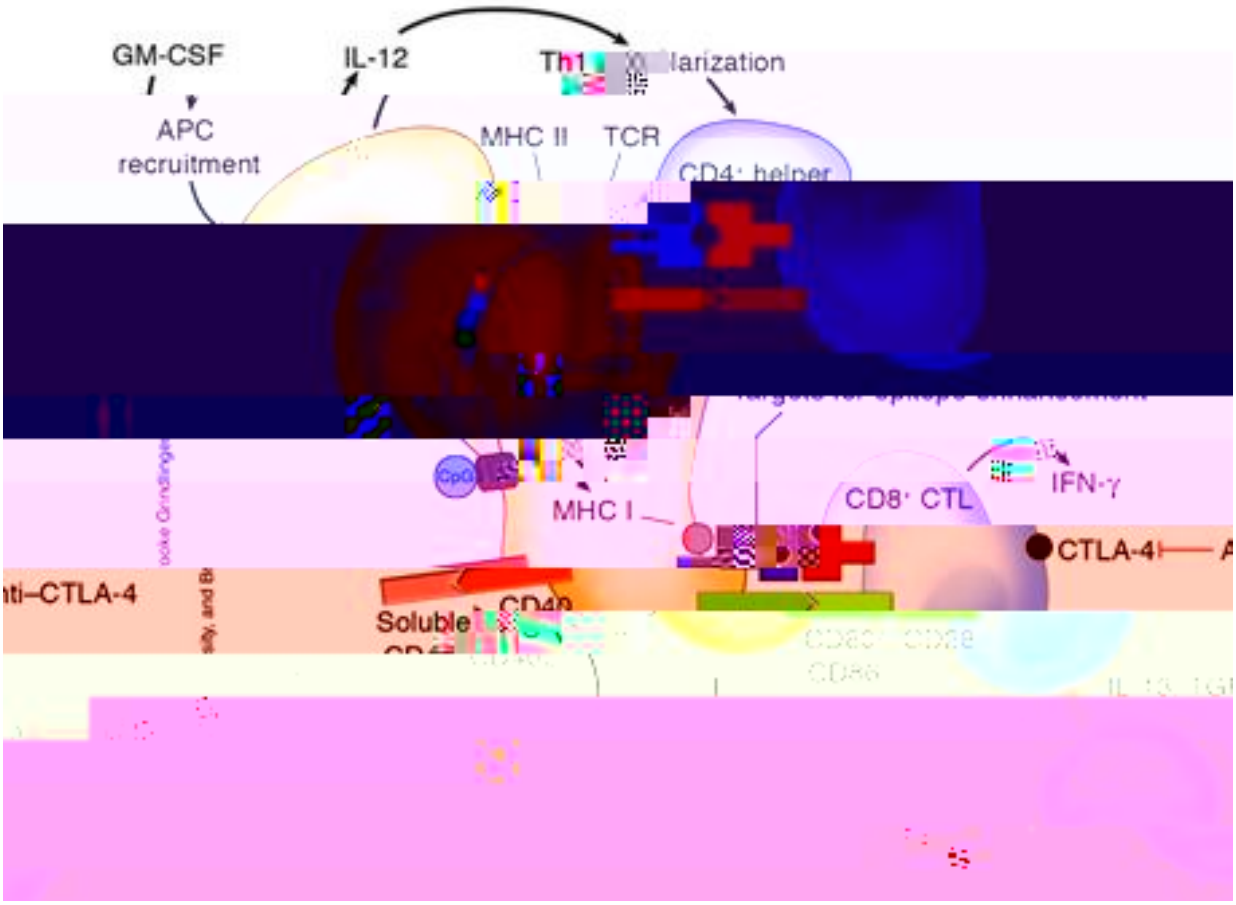

Figure 1: Immunological synapse formation and its role in immune activation of cognate CD4+ T helper cells: CD4+ helper T cells mature and activate APCs through recognition of epitopes presented by class II MHC molecules [MHC II] and interaction of CD40 and CD40 ligand [CD40L]. T e CD40-CD40L interaction causes the APC to upregulate expression of costimulatory molecules such as CD80 and CD86 and to secrete cytokines IL-12 and IL-15. T e costimulatory molecules interact with CD28 on the CD8+ CTL to provide a second CTL activation

therefore, of traditional T 1 adjuvants typically requires sophisticated conjugation technology or formulation with a macromolecular or

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