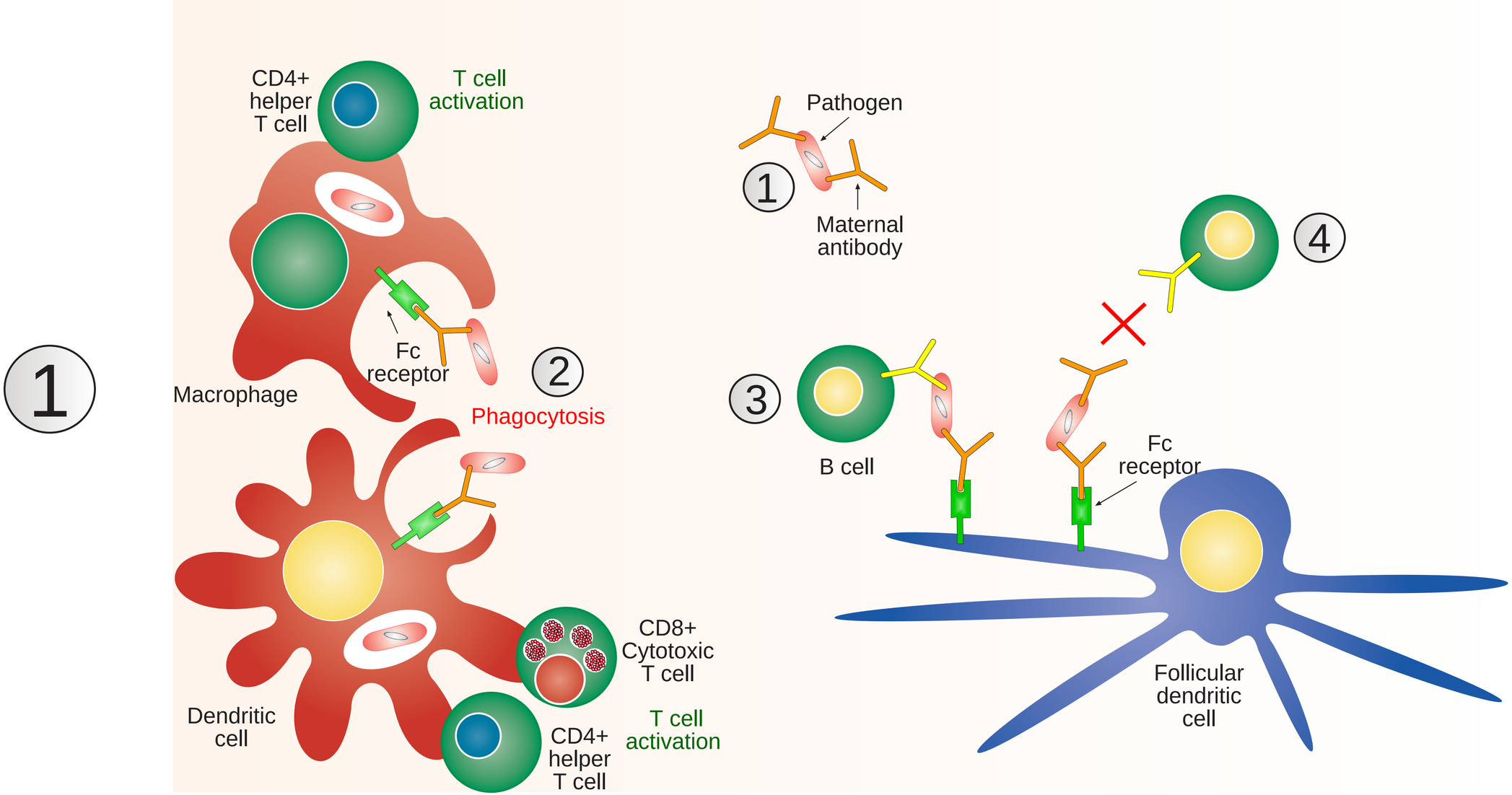
Neonatal immunity

① *Passive immunity*

② *Innate immunity*

③ *Adaptive immunity*

Passive immunity in neonates



Passively aquired maternal antibodies may enhance or inhibit the immune response in neonates as follows: (1) Maternal antibodies coat pathogens and reduce infectivity as well as tag them for destruction by immune cells. However, this can reduce vaccine efficacy when using live replicating vectors. (2) Maternal antibody-coated pathogens are more easily taken up by phagocytes that express Fc receptors. Antigen presentation to T cells is improved in this way. (3) Maternal antibody-coated antigen is trapped by follicular dendritic cells that express Fc receptors and facilitates priming of B cells. (4) B cell epitopes can, however, be masked by maternal antibody and interfere with B cell priming.

Innate immune response in neonates.

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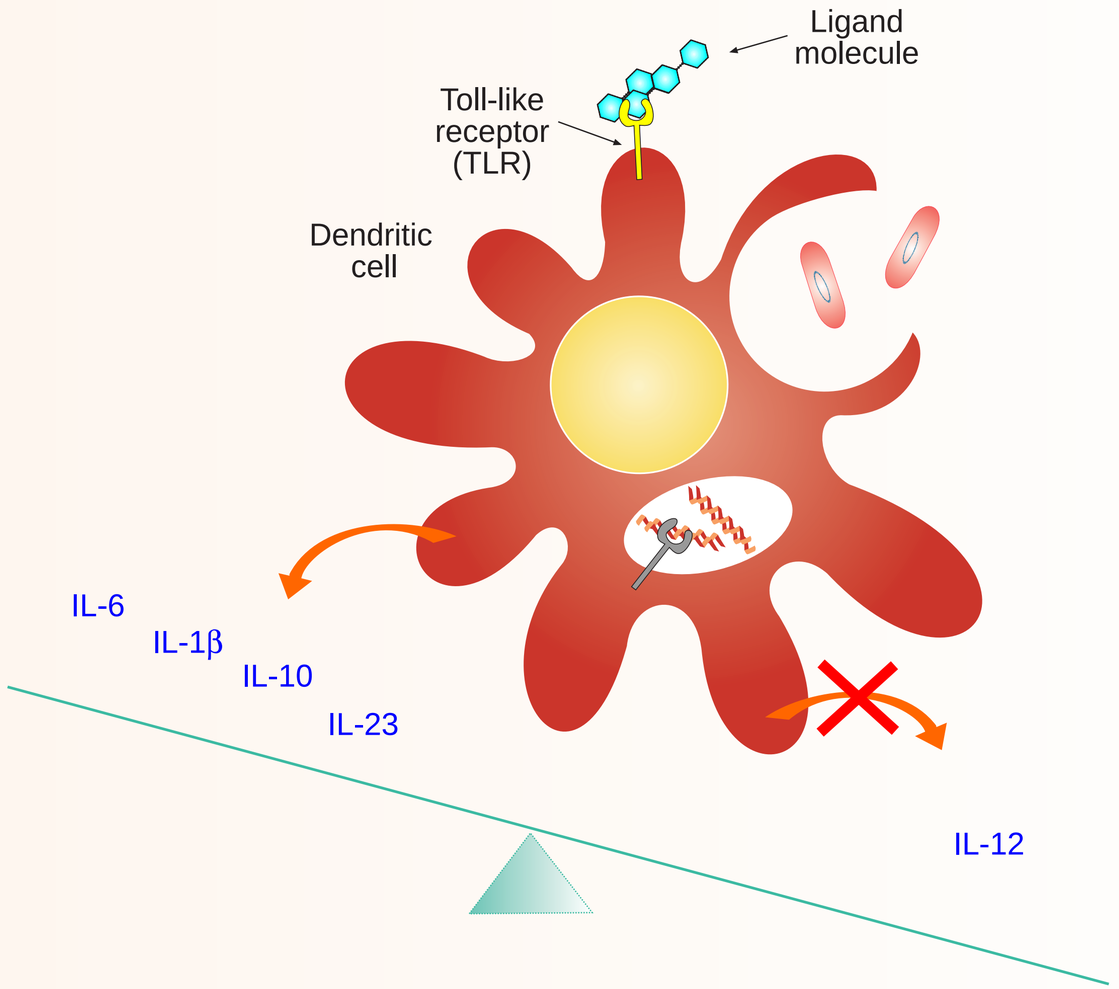
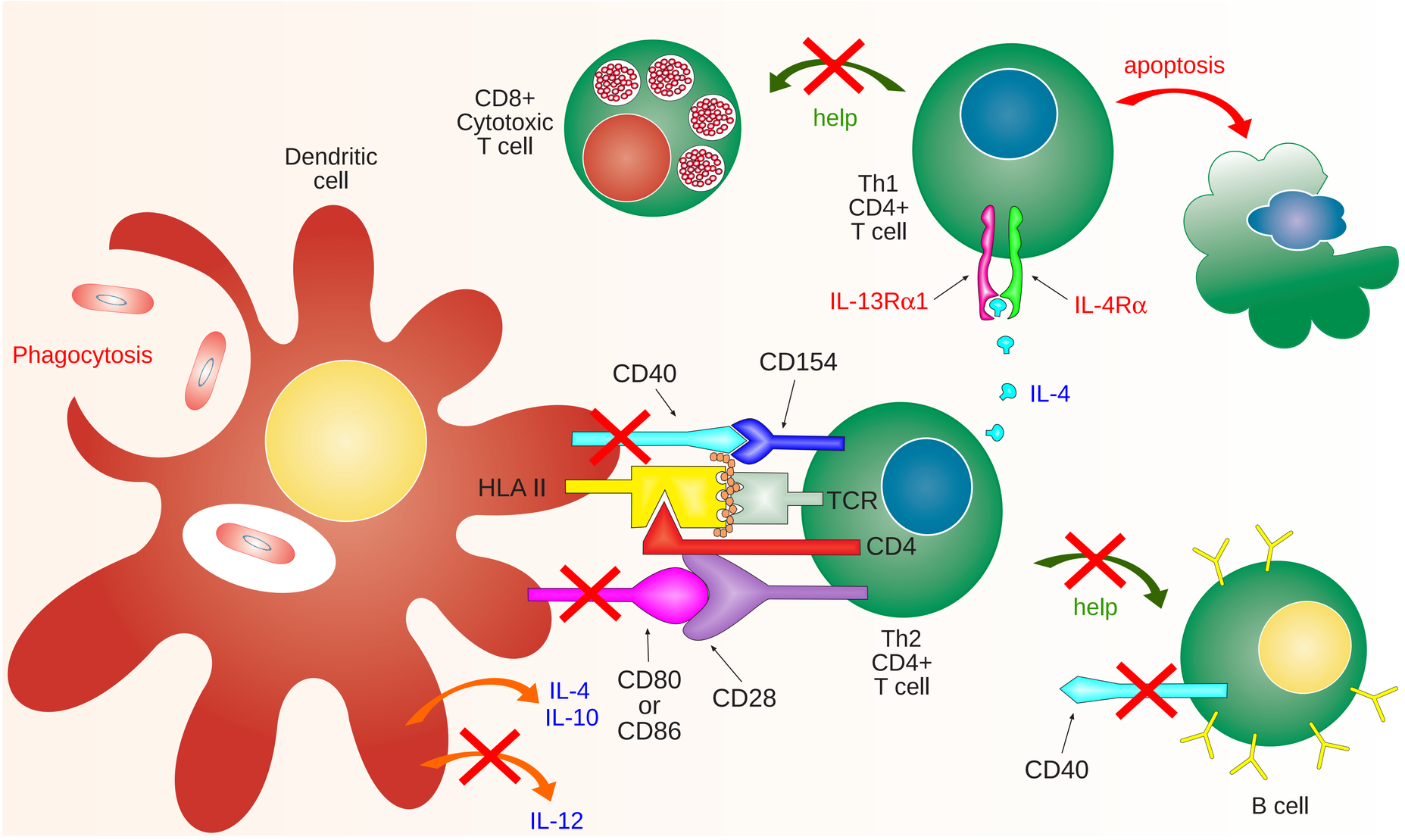
2



Innate immune responses to pathogens that involve the engagement of toll-like receptors in neonatal antigen presenting cells, such as dendritic cells, promotes the secretion of cytokines IL-6, IL-1β, IL-10 and IL-23 and very little IL-12. In turn, IL-10 and IL-23, during T cell activation, promotes the maintence of a Th2 and Th17 T cell differentiation pathway instead of a Th1 response which requires IL-12. Hence the innate immune response via toll-like receptor engagement also plays a part in the polarisation of neonatal cell-mediated T cell responses.

Cell-mediated immunity is impaired in neonates.

3



T cell activation in neonates is impaired and skewed towards a Th2 response. It is thought that low expression of co-stimulatory molecules such as CD80, CD86 and CD40 by activated dendritic cells fail to provide adequate activation signals to naive T cells. Activated dendritic cells preferentially secrete Th2 polarising cytokines IL-4 and IL-10 and low levels of the Th1 polarising cytokine IL-12. Prelimary murine studies have also identified a mechanism contributing to a lack of neonatal Th1 responses where Th1 T cells express a unique receptor for IL-4 that induces apoptosis. IL-4 is abundantly produced by activated neonatal Th2 T cells. T-dependent antibody responses are also impaired in neonates and may be due to a lack of CD40 expression by B cells which is required for antibody synthesis, isotype switching and affinity maturation.