

POSTER 105

DON'T BE SUCH A BABY! OR THE EFFECTS OF THE ENVIRONMENT AND T CELLS ON NEONATAL IMMUNITY

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It is known that neonates are highly susceptible to microbial infections and allergic reactions. This susceptibility is due to a lack of Th1 cells and an excess of its Th2 counterparts. However, the mechanism underlying this Th1/Th2 imbalance has not been clearly elucidated. Although both Th1 and Th2 cells are present in the primary response, only Th1 cells up-regulate the IL-13R α 1 chain. Consequently IL-13R α 1 can associate with IL-4R α to form a heteroreceptor through which IL-4 from Th2 cells can signal and cause the apoptosis of Th1 cells upon secondary re-challenge with antigen. Formation of this IL-13R α 1/IL-4R α heteroreceptor is influenced by two factors: the neonatal environment and intrinsic T cell factors. Previous studies demonstrate that the lack of IL-12 in the neonatal environment supports IL-13R α 1 up-regulation and Th1 apoptosis. This lack of IL-12 is due to a low frequency of CD8 α +CD4- DCs, the main producer of IL-12. However, by day 6 postpartum, this DC subset reaches a significant accumulation and produces sufficient IL-12 that down-regulates IL-13R α 1 and restores Th1 responses. Interestingly, T cells also contribute to the Th2 bias of neonatal immunity as evident by the fact that when adult T cells are transferred into a neonatal environment, they do not up-regulate IL-13R α 1. Current studies show that T cells from 8d mice are no longer susceptible to antigen induced IL-13R α 1 up-regulation when transferred into the neonatal environment. Determining the mechanism as to why 8d T cells are resistant to this up-regulation is ongoing, but indicates the involvement of the IL-12R β 2 chain.