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-13Rα1 chain. Consequently IL-13Rα1 can associate with IL-4Rα 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-13Rα1/IL-4Rα 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-13Rα1 up-regulation and Th1 apoptosis. This lack of IL-12 is due to a low frequency of CD8α+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-13Rα1 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-13Rα1. Current studies show that T cells from 8d mice are no longer susceptible to antigen induced IL-13Rα1 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-12Rβ2 chain.