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## **Generating a neutralizing antibody against IL13R $\alpha$ 1?**

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Each year approximately 1.44 million deaths result from neonatal infection. Initial insights into the differences between the adult and neonatal immune systems were revealed by Sir Peter Medawar during his studies of organ transplantation. He discovered that skin grafts between different strains of mice, which normally result in a rejection of the graft between adult mice, could be successful if the recipient was initially transferred with splenic cells from the donor mouse during its neonatal stage of development. The phenomenon of neonatal tolerance has gone largely unexplained for nearly 50 years. Recent data from our lab supports a theoretical model for neonatal tolerance in which apoptosis of CD4<sup>+</sup> T helper type I cells precludes rejection of the transplant. More specifically, we have demonstrated the involvement of the type II IL4 cytokine receptor in initiating this apoptotic signal. The IL13R $\alpha$ 1 cytokine receptor subunit is one component of this heterodimeric receptor, creation of an antibody that functionally inactivates this subunit would therefore provide a useful reagent to carry out more articulate studies of the neonatal immune system. Utilizing a baculovirus vector encoding the extracellular domain of IL13R $\alpha$ 1, we have expressed and purified recombinant IL13R $\alpha$ 1 and have further immunized Armenian hamsters to initiate an antibody response. Soon, we will fuse the B cells derived from these animals with immortalized myeloma cells to generate a permanent cell line expressing an antibody specific for IL13R $\alpha$ 1.