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The study of neonatal immunity: Creation of an IL13Rα1 deficient mouse
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Each year approximately 1.44 million newborns worldwide die from infections during the first four weeks of life. This is due in part to the inability of the developing neonatal immune systems to mount a Th1 immune response, which is responsible for clearing bacterial and viral infections. Previous studies in our laboratory have shown that expression of the IL-13 receptor α1 subunit (IL13Rα1) is upregulated in neonatal Th1 cells, and this overproduction is hypothesized to signal for their death. Currently, a knockout mouse deficient in expression of IL-13Rα1 is not available and the development of an IL-13Rα1 knockout mouse will allow studies to substantiate this hypothesis and possibly provide new insights into the relationship between IL-13Rα1 signaling and neonatal immunity. If we can better understand the mechanisms that involve IL13Rα1 and the development of Th1 immune responses, then this receptor can be targeted in novel vaccine strategies to protect newborns from infection.