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Neonatal Mast Cells and Transplacental Ige Transfer

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Abstract

Atopic diseases, including food allergy, allergic rhinitis, atopic dermatitis, and asthma, exhibit some degree of heritability. Although large cohort studies suggest that this heritability emerges at least in part from the interaction of genetic, epigenetic, and environmental conditions

Keywords: Immuno; Infection; Vaccine; Body

Introduction

Studies in humans and mice have also suggested a more direct role for maternal immune status in the development of atopic disease [1]. Murine studies have shown that sensitizing pregnant females to ovalbumin (OVA) increases the susceptibility of pups to OVA-induced bronchoconstriction; however, OVA-sensitized pups were found to exhibit increased susceptibility to the unrelated protein casein, suggesting an acquired, antigen-independent transfer of atopic susceptibility. In humans, maternal (but not paternal) circulating total IgE concentration is correlated with infant IgE levels and the development of eczema [2]. Although IgE plays a major role in human atopic disease, it is not thought to cross the maternal-fetal interface and is rarely detected in the circulation of either neonatal mice or humans. Thus, the mechanism(s) underlying atopic heritability are not yet fully understood. Mast cells (MCs) are long-lived tissue-resident granulocytes that are widely recognized as central effector cells in human atopic diseases [3]. They emerge from circulating committed progenitors that populate the tissues during embryonic life. In tissues such as the skin, neonatally acquired MCs (at least in mice) persist into adulthood. MCs express the high-affinity IgE receptor FcεR1α on their surface, through which they are able to efficiently capture circulating IgE [4]. Following IgE cross-linking, MCs release a broad range of pro inflammatory mediators into the extracellular environment, including preformed histamine and proteases, rapidly synthesized lipid pro inflammatory mediators, as well as transcriptionally regulated chemokines and cytokines. MC capture of IgE also stabilizes the molecule. Although circulating IgE has a half-life of only days, IgE can be maintained on the surface of MCs for months. Thus, MCs have the potential to serve as a tissue-resident "reservoir" of IgE even under conditions in which no circulating IgE can be detected [5]. In a recent issue of Science, Used a series of murine model systems and ex vivo studies of human MCs to establish that fetal MCs can bind maternally derived IgE and can be subsequently activated in an antigen-specific manner both in vitro and in vivo [6]. Pups that are born to ragweed-sensitized mothers and challenged intranasally with ragweed at 4 weeks of age developed pulmonary eosinophilia and airway hyper reactivity. The authors observed that transfer of maternal IgE is dependent on the neonatal Fc receptor (FcRN) and that FcRN knockout mice were protected from both ragweed-induced airway hyper reactivity and pulmonary eosinophilia. Further supporting a role for FcRN in the transfer of IgE across the maternal-fetal interface, the authors observed that IgE and IgG colocalized in placental endothelial cells of wild-type mice, whereas neither was observed on endothelial cells in FcRN knockout mice [7]. Finally, the authors found that MCs in 14- to 20-week-old human fetal skin and lung were histologically mature, expressed FcεR1, and were able to bind IgE. These findings suggest that maternal transfer of IgE can sensitize fetal MCs for antigen-specific activation responses in the postnatal period.

Although these results are fascinating and potentially important, additional studies will be required to determine the degree to which maternal IgE transfer influences the development of allergic disease in human children. It is possible that maternal IgE may contribute to infant sensitization to allergens via the skin, especially because maternal IgE was previously found to be correlated with the development of infant eczema [8].

This process could also potentially be exacerbated by genetic polymorphisms and epigenetic modifications associated with barrier integrity such as the loss-of-function allele of filaggrin that has previously been linked to both atopic dermatitis the development of peanut allergy. Maternal IgE transfer could also help to explain why only a subset of patients with filaggrin loss-of-function polymorphisms go on to develop atopic dermatitis and why the risk of dermatitis in children of mothers with filaggrin mutations depends partly on the presence of maternal IgE sensitization. Although the convincingly demonstrated that fetal skin MCs bind maternal IgE in mice, they did not evaluate IgE binding by MCs in mouse mucosal tissues such as the lung (in which MCs exhibited greatly decreased FcεR1 expression) or intestinal tissue (in which fetal MCs undergo far more rapid turnover than in the skin) [9]. Experimental sensitization in mice results in high levels of IgE specific to a single antigen that may not replicate the function of endogenously generated IgE in humans. Whereas mouse pups in experimental settings are typically weaned by 3 weeks of age, human development is a much slower process. Studies of MC IgE retention in patients with hypogamma globulinemia have established that IgE on adult skin MCs has a half-life of approximately 16 to 20 days. Thus, by the time a human infant is introduced to solid foods at 4 to 6 months of age, little maternal-derived IgE is likely to be retained on tissue MCs. Finally, although parental atopy clearly increases the risk of atopic disease development in infants, there is little evidence in the literature to support antigen-specific transmission of risk. Given that IgE and MCs evolved to serve roles in host defense, it is possible that transfer of maternal IgE to long-lived fetal MCs, especially those in the skin, may reflect evolutionary selection at some point in mammalian

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development (Fig 1). Both MCs and IgE play a protective role in skin defense against parasitic insects such as ticks, suggesting that maternal IgE transfer may provide protection against ectoparasites. The same mechanism may confer passive immunity to skin-invasive helminths in areas with high endemic infection rates. Additionally, a series of studies by the Galli laboratory have identified a role for MC proteases in degrading xenobiotic reptile and insect venoms, and a specific role for IgE was determined for honey bee venom and Russel viper venom. Thus, maternal transmission of IgE may also provide a degree of protection to infants against stings and bites that are potentially otherwise lethal, particularly in populations with high levels of exposure and sensitization.

This mechanism could provide passive neonatal barrier protection against helminths, ticks, or venoms while also potentially transmitting some risk for antigen-specific hypersensitivity (Figure 1).

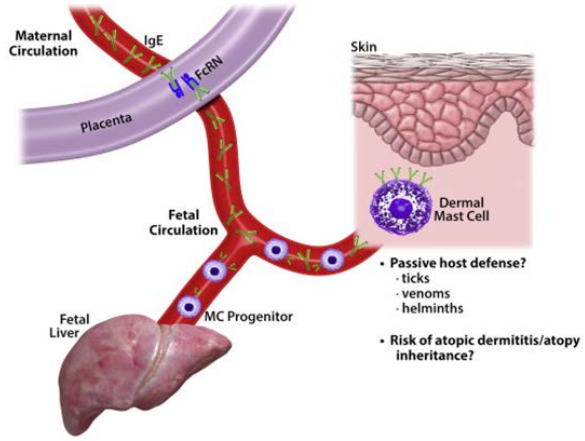

Figure 1: Maternal IgE can access the fetal circulation via placental FcRN. The prenatally acquired IgE may be taken up by fetal MCs (seeded in the tissues by committed progenitors from fetal liver, hematopoietic endothelium, and yolk sac).

Conclusion

In conclusion, definitively established that both IgG and IgE are capable of crossing the maternal-fetal interface via FcRN and that the IgE that passes to the fetus in this manner can be bound by MCs in both mice and humans. From an evolutionary perspective, maternal transmission of IgE may have evolved to help protect newborn mammals from xenobiotic venoms and ectoparasites. Although this study illustrates the potential for antigen-specific transfer of IgE sensitization, further focused studies will be required to determine whether such transfer contributes to atopic disease susceptibility and development in humans.

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