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Cord blood hemopoietic progenitor cell toll like receptor expression and function: a mechanism underlying allergic inflammation in early life?

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Objective/purpose

Neonatal immune responses to environmental stimuli, mediated *via* TLR, may determine the development of atopy in childhood. Since hemopoietic mechanisms are involved in development and maintenance of allergic inflammation, we investigated alterations in progenitor expression and differentiation profiles after stimulation with TLR agonists.

Methods

Freshly isolated, CD34-enriched human CB cells were stimulated with 10 μg/mL lipopolysaccharide (LPS) or 5 μM CpG ODN overnight. Flow cytometric analyses were used to evaluate surface and intracellular expression of TLR-2, TLR-4, TLR-9, as well as the hemopoietic cytokine receptors (HCR) IL-5R, IL-3R and GM-CSFR; methylcellulose cultures were performed to assess CD34+ cell differentiation capacity into Eo/B CFU.

Findings

After TLR agonist stimulation, CD34+ cell TLR-2, -4 and TLR-9 percentage expression increased significantly ($p = 0.005$), whereas HCR expression decreased ($p = 0.01$); however, mean fluorescence intensity of all receptors was found to be increased. Stimulation with a combination of TLR agonists and hemopoietic cytokines induced increased IL-5- and IL-3-responsive Eo/B CFU ($p = 0.02$), when compared to hemopoietic cytokine stimulation alone.

Deliverables

CB CD34+ progenitor cells significantly express TLR, and TLR ligation directly affects both TLR and HCR expression. These receptor alterations allow modulation of progenitor cell differentiation capacity into eosinophils and basophils, key cells involved in allergic inflammation. These findings may highlight an alternate innate immune pathway of microbial influence on the development of allergic inflammation in early life.

Relevance

These findings may suggest that activation of TLR-mediated hemopoietic mechanisms during the neonatal period could be a forerunner for the development of infant atopy and allergic inflammation, thereby providing a novel therapeutic target for preventative measures against infant allergy.

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