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Regulation of Thymic Stromal Lymphopoietin (TSLP) receptor expression

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Objective/purpose

TSLP plays a major role in the induction and effector phases of allergic diseases by acting on dendritic cells, mast cells (MCs), T cells and $CD34^+$ hemopoietic progenitor cells. Whereas the cellular origin and the mechanisms regulating TSLP production are well documented, little is known about the regulation of TSLP receptor expression. We analyzed the regulation of TSLP-R with several cytokines, TLR ligands and drugs commonly used to treat asthma.

Methods

Neonatal $CD34^+$ cells were examined by two-colour analysis for the expression of CD34 and TSLP-R after 48 hours of incubation with or without: 1) cytokines (IL-1, TNF, IL-4, IFN-γ, TGF-β) used alone or in combination; 2)inflammatory mediators (PGE2, LTC4 and PGD2); 3) bacterial products (SAC, LPS and PGN); 4) TLR ligands (TLR3, TLR5, and TLR7); 5) dexamethasone and isoproterenol.

Findings

Expression of TSLP-R on $CD34^+$ cells was markedly enhanced by IL-1/TNF; this effect was suppressed by IL-4, IFN-γ and TGF-β and augmented by PGE2. TSLP-R was induced by SAC and PGN but was not affected by LPS or other TLR ligands. Most interestingly, dexamethasone slightly induced TSLP-R and IL-7Rα expression and markedly increased the effect of IL-1/TNF. The enhancing effect of dexamethasone was also observed on $CD14^+$ and $CD56^+$ neonatal cells. Isoproterenol had no effect on the regulation of TSLP-R.

Deliverables

Taken together, our data may explain the synergistic effect of IL-1/TNF on the response of $CD34^+$ cells to TSLP. They further show that TSLP-R expression is markedly regulated by the inflammatory and cytokine environment. The biological consequences of glucocorticoid-induced upregulation of TSLP-R will be examined.

Relevance

Currently, there is no disease-modifying treatment for allergic diseases. Indeed, steroids, the main therapeutic tool for improving the quality of patients' life, do not prevent irreversible tissue-remodeling and the loss of pulmonary function. Current observations indicate that steroids upregulate the receptor of pro-inflammatory cytokine TSLP on the surface of $CD34^+$ cells. The effect of current anti-allergic treatment on the proinflammatory activity of $CD34^+$ cells should be taken into account and these cells could be considered as target for the development of novel therapeutic approaches for atopic diseases.

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