## Abstract

IL-33 is a dual function protein that may function as both a proinflammatory cytokine and an intracellular nuclear factor. In a role of cytokine IL-33 signals via receptor ST2 and induces T helper type 2-associated cytokines in its target cells including mast cells, basophils, eosinophils and natural killer cells. Additionally, it acts as chromatin-associated nuclear factor with transcriptional regulator properties affecting expression of some proinflammatory cytokines. Regulation of this processes is poorly understood, mechanisms underlying synthesis, processing and secretion of IL-33 also remain to be fully explored. The aim of our study was to examine mechanisms probably involved in regulation of IL-33 production and its secretion outside the cell. First, we investigated possibility that IL-33 secretion is affected by stimulation with cytokines TNFα, IFN γ, IL-1β, IL-13, IL-33, TGF-β and IL-10 or stimulation with LPS isolated from *E. coli*. Next we investigated hypothesis that IL-33 is released from cells during cell damage or necrosis and serve as „alarmin". Necrosis was induced in LPS-stimulated cells by freeze-thawing cycles. Besides the presence of IL-33 we tested levels of IL-1α and IL-1β. In our experimental model, we used A549 cell line (alveolar type II-like cells), THP-1 promonocytic cell line and monocytic THP-1dif cells converted from THP-1 cells by incubation with vitaminD3. Cell differentiation was confirmed by upregulated expression of cell surface molecule CD-14, measured with flow cytometer. Cytokine levels were measured by Luminex technology or ELISA. Despite our effort we didn't detect IL-33 in cell supernatants. Our data shows that neither cytokines nor LPS have the ability to induce IL-33 secretion in the cells. Effect of necrosis on IL-33 presence in supernatants was not confirmed. IL-1β was constitutively produced by epithelial A549 cells with no influence by LPS or necrosis. Concentration of IL-1β was affected by stimulation with LPS in both cell lines, THP-1 and THP-1 dif, respectively. Necrosis upregulated levels of IL-1β in supernatants. Dynamics of the IL-1α expression in our experiments confirmed its role as alarmin.