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Mast cells secrete IL-15 by microvesicles shedding upon P2X7 receptor stimulation

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Mast cells are recognized as the key cells of allergic inflammatory reactions. They express and secrete a number of pro-inflamatory cytokines and chemokines. Interleukin-15 (IL-15) is a potent anti-apoptotic cytokine and a regulator of T, B and NK cells differentiation and proliferation. Bone marrow-derived mast cells (BMMCs) constitutively express IL-15 mRNA and this expression is further upregulated by LPS stimulation. However, there is no evidence for IL-15 cytokine secretion from activated mast cells. P2X7 receptor is one of the purinoceptors, which is activated by ATP. Mast cells highly express P2X7 on the cell surface. Recently, we have reported that ATP induces P2X7-mediated apoptosis of BMMCs, as well as triggers pro-inflamatory cytokine secretion, presumably in the time period between commitment to apoptosis and actual cell death. ATP triggers rapid but transient phosphorylation of multiple signaling molecules in BMMCs, including extracellular-signal regulated kinase (ERK), Jak2, and STAT6. Moreover, BMMCs release annexin-V-positive microvesicles upon P2X7 receptor stimulation. These vesicles contain biologically active IL-15, which later appears in the vesicle-free supernatant and stimulates the proliferation of the IL-15-dependent CTLL cell line. The IL-15-containing microvesicles were also found in the supernatants from THP-1 monocytic cell line and bone marrow-derived dendritic cells upon agonistic P2X7 stimulation. Thus, the microvesicle shedding mechanism constitutes one of secretory pathways for a release of IL-15 in mast cells, dendritic cells and monocytes, which might play an important role in regulation by IL-15 of diverse physiological processes or their pathological deviations.