Synopsis of Original Research Paper

Studies on activation of mast cells in contact dermatitis

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Mast cells contribute to allergic inflammation by releasing chemical mediators in response to activation with cytokines and/or IgE/antigen. Here we report that when mouse bone marrow-derived mast cells were activated with IgE/antigen in the presence of interleukin (IL)-10 and IL-1β, but not other cytokine combinations, there were two phase of prostaglandin (PG)D_2 generation, in which the first phase occurred within 1 hr and the second phase from 2 to 10 hr. The delayed phase PGD_2 generation paralleled the *de nova* induction of cyclooxygenase (COX)-2 protein irrespective of the constant expression of COX-1 and was abrogated by COX-2 specific inhibitor. Detailed examination of individual effect of IL-10, IL-1β and IgE/antigen on COX-2 expression revealed that IgE/antigen or IL-10 each initiated and stabilized COX-2 mRNA expression, whereas IL-1β stabilized COX-2 protein without affecting its mRNA level. Whereas expression of cytosolic phospholipase A_2 ($cPLA_2$) was unchanged under any culture condition, expression of type II secretory PLA_2 ($sPLA_2$) transcript was induced by 5 hr in cells treated with IL-10 + IL-1β independent of IgE/antigen, accompanied by increase in $sPLA_2$ activity. Substantial suppression of delayed phase PGD_2 generation by anti-$sPLA_2$ antibody suggests the functional linkage of the two induced prostanoid-biosynthetic enzymes, $sPLA_2$ and COX-2, to provide PGD_2 in the delayed phase.