due to the dopachrome tautomerase stimulation.

[PA1-46] [2003-10-10 14:00 - 17:30 / Grand Ballroom Pre-function]

Anti-inflammatory mechanism of melittin, a component of bee venom in Raw 264.7 cells and Synoviocyte

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Bee Venom (BV) has been treated in inflammatory diseases such as rheumatoid arthritis (RA). Bee venom contains several biologically active non-peptide substances as well as two major known peptides; the hemolytic peptide melittin (50%) and the neurotoxic peptide apamin, and a number of minor peptides. Previous our study showed that BV blocked LPS and SNP-induced production of NO and PG through inactivation of NF-κB which regulates expression of COX-2 and iNOS. In this study, we investigated whether melittin, a major componet of BV may play a critical role in the anti-inflammatory effect of BV. We investigated effect of melittin on lipopolysaccharide (LPS) and sodium nitroprusside (SNP)-induced induction of COX-2, cPLA2 and iNOS expression, and production of NO and PGE_2, and activation of NF-κB in a murine marcrophage cell line Raw 264.7 cell and synoviocytes. Similar to the effect of BV, melittin prevented LPS and SNP induced COX-2, cPLA2 and iNOS expression, and the production of NO and PGE_2 through inhibition of transcriptional and DNA binding activation of NF-κB. The inhibitory effect of melittin in some paramters tested was less extent compared to BV, but most of the inhibitory effects of melittin was comparable to the effect of BV, suggesting that melittin may be a causal effective componet of anti-inflammatory effect of BV.

[PA1-47] [2003-10-10 14:00 - 17:30 / Grand Ballroom Pre-function]

Effects of chlorhexidine digluconate on thickness of outer membranes isolated from Cultured Porphyromonas gingivalis.

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To get a better insight into the biophysical mechanism of action of chlorhexidine digluconate, we examined the effect of chlorhexidine digluconate on the thickness of outer membranes isolated from cultured Porphyromonas gingivalis using energy transfer between the membrane surface fluorescent probe (1-anilinonaphthalene-8-sulfonic acid) and the hydrophobic fluorescent probe [1,3-di(1-pyrenyl)propane]. 1-Anilinonaphthalene-8-sulfonic acid quenches the monomer fluorescence of 1,3-di(1-pyrenyl)propane. The significant decreases the thickness of lipid bilayer of the outer membranes by chlorhexidine digluconate were observed even at 0.01, 0.1, 1, 2, 3, 4, and 5 mM. It is presumed that the decrease of membrane thickness was resulted, as discovered in another studies of ours, from the fact that chlorhexidine digluconate caused increase in the rate and range of the lateral and rotational mobility of the membrane lipid bilayer, increased annular lipid fluidity and then resulted in clustering of membrane proteins. Reduction in the membrane thickness causes interdigitation between inner and outer monolayers of the lipid bilayer and membrane expansion and in turn, the membrane expansion accompanies changes of the membrane properties and functions. It is presumed that chlorhexidine digluconate exhibits its specific pharmacological effect by the biophysical mechanism descrived above.

[PA1-48] [2003-10-10 14:00 - 17:30 / Grand Ballroom Pre-function]

Differential regulation of phospholipase Cγ isoforms through FcεRI, high affinity IgE receptor