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PS1-19

MECHANISM OF MASTOPARAN-INDUCED CATECHOLAMINE RELEASE FROM PORCINE ADRENAL MEDULLARY CELLS

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Release of catecholamine from porcine adrenal chromaffin cells exposed to mastoparan, a wasp venom peptide which activates GTP-binding proteins and phospholipase A_2, was evaluated. Release of catecholamine was dependent on mastoparan concentration. This release was, however, independent of extracellular and intracellular calcium concentration. Ca^{2+} channel blockers did not abolished their responsiveness to mastoparan. Pretreatment of the cells with bromophenacyl bromide, a phospholipase A_2 inhibitor, was without effect. [^{3}H]Inositol phosphates formation was not altered by mastoparan. Pretreatment of the cultures with pertusis toxin was without effect. Moreover mastoparan induced catecholamine release from permeabilized cells was not inhibited after the treatment with GDPßS. These results indicate that catecholamine release induced by mastoparan is not mediated by activating of GTP-binding proteins which regulate phospholipase C or phospholipase A_2.

ⓒ PS1-20

URINARY NEOPTERIN AND BIOPTERIN LEVELS IN PATIENTS WITH DEPRESSION

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Determination of urinary neopterin and biopterin levels is the method of choice to investigate the metabolism of tetrahydrobiopterin (BH_4) which is the cofactor of aromatic amino acid hydroxylases, the enzymes catalyzing the biosynthesis of norepinephrine, serotonin and dopamine. Levels of urinary neopterin and biopterin in 26 patients with depression and 45 control subjects were determined by high performance liquid chromatography with fluorescence detection. The present results did not show a significant difference of biopterin levels (mean $\pm$ SD nmol/mmol creatinine) between the normal (614 $\pm$ 267) and depressed subjects (635 $\pm$ 281), while an obvious reduction of neopterin level in patients with active depression (441 $\pm$ 261) compared with the control subjects (604 $\pm$ 318) ($P < 0.03$)was noted, and the neopterin level tended to return to "normal" (567 $\pm$ 181) as the active depressive symptoms had remitted. These results failed to support the presumption that BH_4 deficiency might play a role in the pathophysiology of depression, but on the other hand, the changes of neopterin level, which was also an indicator of the state of immune response in mammals, indicated a correlation between depression and neopterin. The relationship between depression, urinary neopterin level, and immune response deserves further exploration.

122