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Influence of age and type of aversive stimulus in cardiovascular consequences of chronic emotional stress: Role of autonomic and vascular mechanisms

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Several lines of evidence correlate psychosocial factors with the pathogenesis of cardiovascular diseases. Adolescence has been proposed as an ontogenic period of vulnerability to stress. Nevertheless, the impact of stressful events during adolescence in cardiovascular activity is poorly understood. In the present study we investigated the impact in cardiovascular function of adolescent and adult rats of two chronic stress protocols: the repeated restraint stress (RRS) and chronic variable stress (CVS). Also, the long-lasting effects in adulthood of stressful events during adolescence were investigated. CVS increased blood pressure and heart rate regardless of the age, whereas RRS increased blood pressure selectively in adults. Assessment of the cardiac autonomic tonus realized by intravenously administrating methyl atropine (muscarinic receptor antagonist) and propranolol (β-adrenoceptor antagonist) revealed that CVS-evoked resting tachycardia was associated with increased cardiac sympathetic activity in adults while a decreased cardiac parasympathetic activity was observed in adolescent animals. RRS also decreased cardiac parasympathetic activity in adolescents, but this effect was followed by a reduction in intrinsic heart rate. Analysis of vascular reactivity to vasoactive agents indicated that both stress regimens increased the vasoconstrictor response to the α1-adrenoceptor agonist phenylephrine in adolescent animals. Also, results indicated that RRS, but not CVS, reduced the vasodilator response to acetylcholine and the nitric oxide donor sodium nitroprusside in both adolescent and adult animals. All alterations observed during adolescence were reversed in adulthood. These findings indicate an age- and stress type-specific influence in stress-evoked cardiovascular/autonomic changes. Moreover, data suggest absence of cardiovascular consequences in adulthood of stress during adolescence.

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In vitro cytotoxic and genotoxic evaluation to ascertain toxicological potential of Ketoprofen

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Ketoprofen analagesic and anti-inflammatory properties are well known but little work is done about its cytotoxic activity and the potential to damage the DNA. The present study was designed to evaluate the cytotoxic and genotoxic potential of ketoprofen. MTT dye (3-(4, 5-Dimethylthiazol-2-yl)-2, 5-diphenyltetrazolium bromide) was used to assess cytotoxicity in which confluent monolayer of Vero cells were incubated in the presence of increasing concentrations of ketoprofen. Genotoxicity was evaluated by SCGE (single cell gel electrophoresis) assay or comet assay. Lymphocytes were separated from the mice blood and treated with different concentrations of ketoprofen. Lymphocytes were incorporated in agarose gel on cavity slides and visualized for strand break to asses DNA damage. Ketoprofen concentrations 8 mM, 6 mM, 4.5 mM, 3.3 mM, 2.5 mM, 1.8 mM, 1.4 mM, 1 mM, 0.5 mM were used for both cytotoxic and comet assay. The results of cytotoxic assay showed significant ($p<0.001$) cytotoxicity at 8 mM, and 6 mM concentrations. The cytotoxic concentration for 50% of cells (CC50) value was calculated at 5.2 mM concentration. In case of the comet assay ketoprofen presented DNA damaging potency, creating significant ($P<0.001$) DNA damage at 8 mM concentration, a moderate damage at 6 mM concentration and a mild damage at 4.5mM concentration which was evident from the comet tail lengths and changes in head diameter. DNA damage index was calculated for each concentration of ketoprofen and compared with the control. The data advocates that ketoprofen possesses cytotoxic and genotoxic potential at higher concentrations and its dosage should be carefully monitored to avoid its toxicity.

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