PP.26.15 ACUTE EFFECTS OF VENOM ACANTHOSCURRIA JURUENICOLA IN BLOOD PRESSURE AND BLOOD GLUCOSE IN RATS

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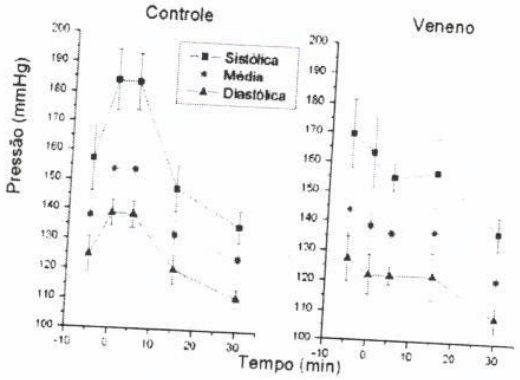
Objective: The study of the chemical composition of animal toxins aims to understand the action of the effects of venoms on humans. During such research, it was discovered active compounds that could exert therapeutic functions, leading to the development of various drugs currently used. In Brazil, 43 milion inhabitants are diagnosed with hypertension. Complications arising from these disease generates huge spending for the public health. The goal of this project is to study the acute effects of the components from Acanthoscurria juruenicola on blood glucose levels and blood pressure.



Design and method: Winstar rats, adult, male, 350g to 400g. In the control group is administered saline. In the experimental group, the venom is administered at a dose of 300 micrograms per kilogram of body mass diluted in saline. 12 hours before the start of the procedure, the rat is placed in fasting food restriction, only water ad libitum. Then, the animal is sedated and anesthetized. The blood pressure was measured by a carotid access coupled to a pressure transducer, and blood glucose was assessed by strips filled with tail blood. The computer registers the blood pressure. 1 minute before the injection of the solution, the glucose is measured. In the next 1, 5, 10, 20 and 30 minutes following the administration of the solution, glucose is continously measured. The amounts recorded allowed the construction of graphs.

Results: The study of the results shows a large increase in blood pressure seconds before the application of saline in the control group. This does not happen to the experimental group, very likely because of the action of the venom. About the glucose levels, the control group showed an increase. This is not what happens to the experimental group, most likely due to the action of the venom.

Conclusions: The venom shows an antihiperglycemic and antihypertensive effect. It has already being identified the influence of Acanthoscurria juruenicola on blood pressure and glucose. At the end of this phase, will be tested molecules already purified.

PP.26.16 PARENTERAL ADMINISTRATION OF COENZYME Q10 AFTER EXPERIMENTAL INFARCT MYOCARDIUM DECREASES THE MORTALITY AND LIMITS MYOCARDIAL HYPERTROPHY

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Objective: It was shown in our previous study that CoQ10 injected intravenously (i.v.) after the onset of acute myocardial ischemia limited myocardial remodeling and improved the cardiac function on the 21st post-operative day. The aim of the present study was to estimate whether CoQ10 injected intramuscular (i.m.) after acute myocardial infarction has cardioprotective efficacy.

Design and method: The experiments were carried out on the rat model of acute MI induced by coronary artery ligation. Solubilized CoQ10 (10 mg/kg) was administered i.v. (n=6) or i.m. (n=7) in 10 min after occlusion. Control infarct rats (n=7) and sham operated rats (n=6) received saline. Mortality, the degree of myocardial hypertrophy, CoQ10 tissue levels were evaluated on the 21st day after coronary occlusion. The CoQ10 content was measured by HPLC with electrochemical detection. Statistical analysis was performed with two-tailed t-test.

Results: After coronary occlusion 42% untreated infarct rats were died. No rat was died in the groups after i.v. or i.m. CoQ10 injection. Rats with MI had large aneurisms replacing muscle wall in the area supplied by the occluded coronary artery. So, thickness of interventricular septum was used for estimate of the degree of myocardial hypertrophy. Thickness of interventricular septum in control infarct rats was larger ($p<0.05$) than that in sham operated rats. Administration of CoQ10 considerably decreased the development of myocardial hypertrophy: thickness of interventricular septum in infarct rats received i.v. or i.m. CoQ10 injection was less ($p<0.05$) than that in control infarct rats and not differed from sham operated rats. Plasma and hepatic CoQ10 levels in rats after CoQ10 i.v. or i.m. administration were higher about by 66% ($p<0,05$) and 100% ($p<0,01$) respectively, than those in the control rats.

Conclusions: Single i.v. or i.m. administration of CoQ10 in 10 min after acute MI decreases the mortality and reduces the development of myocardial hypertrophy. The parenteral forms of CoQ10 for urgent therapy of cardiac ischemic events could be beneficial.

PP.26.17 FUNCTIONAL MICROVASCULAR RAREFACTION IS REVERSED BY SIMVASTATIN AND LOVASTATIN IN THE BRAIN AND SKELETAL MUSCLE OF SPONTANEOUSLY HYPERTENSIVE RATS

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Objective: Microvascular rarefaction is an aggravating factor of hypertensive end-organ damage. However, the microcirculatory effects of statins in hypertension remain unknown. Thus, this study was designed to investigate the acute effects of simvastatin and lovastatin on cerebral and muscular microcirculation in Spontaneously Hypertensive Rats (SHR).

Design and method: Male normotensive Wistar rats (WKY) and SHR were divided into 4 groups of 6 animals each: WKY-CTL and SHR-CTL treated with 0.9% saline solution, and SHR+SIM and SHR+LOVA treated with simvastatin (SIM) and lovastatin (LOVA) 30 mg/kg/day during 3 days orally by gavage. We investigated brain and skeletal muscle (SM; gracilis muscle) functional capillary density (FCD) using intravital fluorescence videomicroscopy after IV injection of fluorescein-isothiocyanate (FITC)-labeled dextran. All surgical procedures and protocols were approved in accordance with the internationally accepted principles for the Care and Use of Laboratory Animals (CEUA license # L-48/12).

Results: SIM administration reduced SBP in SHR (SHR-CTL 203±3 vs. SHR+SIM 172±6 mmHg; $p< 0.001$; in contrast LOVA treatment was not able to reduce SBP (SHR+LOVA 192±3 mmHg). SHR showed a significantly lower FCD in the gracilis muscle compared to WKY (SHR-CTL 210±17 vs. WKY-CTL 338±16 capillaries/mm²; $p<0.01$). SIM (SHR+SIM 447±20 capillaries/mm²) and LOVA (SHR+LOVA 418±22 capillaries/mm²) treatment reverted functional capillary rarefaction in the SM of SHR (SHR-CTL 210±17 capillaries/mm²; $p<0.001$). Cerebral FCD was reduced in SHR compared with WKY (SHR-CTL 337±61 vs. WKY-CTL 421±35 capillaries/mm², $p<0.05$). The administration of SIM (SHR+SIM 530±31 capillaries/ mm²) and LOVA (SHR+LOVA 471±37 capillaries/mm²) during 3 days was capable to increase cerebral FCD in SHR (SHR-CTL 337±61 capillaries/mm²; $p< 0.05$).

Conclusions: Acute treatment with simvastatin and lovastatin significantly reversed microvascular rarefaction in hypertensive rats. In addition to cholesterol-lowering effects, statins could turn out to be a new therapeutic approach for improving microcirculatory function in hypertensive patients. However, it is necessary to further investigate the mechanisms and pathways which may additionally play important roles in statin-mediated cardiovascular protection in hypertension.

PP.26.18 CHRYSIN AND LUTEOLIN ALLEVIATE EXAGGERATED VASOCONSTRICTION ASSOCIATED WITH INSULIN RESISTANCE

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Objective: Chrysin and luteolin are two plant flavonoids with PPAR-γ stimulating activity. Here, we investigated the protective effect of chrysin and luteolin from exaggerated vasoconstriction associated with insulin resistance.

Design and method: Insulin resistance was induced in rats by fructose drink-