N-METHYL-D, L-ASPARTIC ACID STIMULATING LH SECRETION *IN VIVO* IN THE ADULT MALE GUINEA PIG PARTIALLY VIA NON-GnRH RECEPTOR PATHWAY

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Guinea pig as a research model has some advantages. For example, several aspects of its reproductive function are more close to those of primate, i.e. its reproductive cycle is composed of a true luteal phase and the distribution of the hypothalamic GnRH neurons is more similar to that of the primate. However, guinea pig also has some particularities in its endocrine axes and metabolism pathways, which by comparison with other species may provide new insight. Previous studies in rats and monkeys showed that N-Methyl-D,L-Aspartic Acid (NMA) stimulated luteinizing hormone release via the hypothalamus. In the male guinea pig we observed that NMA could induce strong LH secretion, which could be blocked by the NMDA receptor antagonist, DL-2-amino-5-phosphonovaleric acid (AP5). Administration of Cetrorelix, an GnRH receptor antagonist, before introducing NMA could only partially block the LH release induced by NMA, whereas it could completely block the LH secretion induced by GnRH. It is concluded that NMA induced LH secretion in the male guinea pig was only partially mediated by GnRH receptor.

EFFECT OF TMP ON BURN INJURY PAIN MEDIATED BY $P2X_3$ RECEPTOR

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Peripheral administration of P2X agonists rapidly causes nocifensive behavior in experimental animals and pain sensation in humans. Most of the nociceptive response to peripheral ATP is mediated by $P2X_3$ receptor. The effects of tetramethylpyrazine (TMP) on rat burn injury pain mediated by $P2X_3$ receptor was investigated. First degree and superficial second degree burn injury models were adopted. Mechanical withdrawal threshold and thermal withdrawal latency were measured and the $P2X_3$ receptor expressions of nerve terminal in burn injury skin were detected by immunohistochemistry. After burn, the mechanical withdrawal threshold (MWT) and thermal withdrawal latency (TWL) in group IIIA (first degree foot burn +NS group) and group IIIB (superficial second foot burn +NS group) were lower than that in group I (sham foot burn), lasting for 24 and 96 hours respectively ($p<0.01$). After hour 24, there was no difference in MWT and TWL between group IIA (first degree foot burn +TMP group) and group I ($p>0.05$). However, there was difference between group IIB (superficial second foot burn +TMP group) and group I ($p<0.01$) until hour 72 ($p>0.05$). At day 3 post burn, the $P2X_3$ receptor expressions at the burn injury skin nerve terminal in group VIA (first degree back burn +NS group) and group VIB (superficial second back burn +NS group) were significantly increased compared with other groups ($p<0.05$). Post-treated with TMP, the $P2X_3$ receptor expressions at the nerve terminal in group VA (first degree back burn + TMP group) and VB (superficial second back burn +TMP group) were markedly decreased. These results suggest that TMP may alleviate burn injury pain mediated by $P2X_3$ receptor. This work was supported by the National Natural Science Foundation of China (No. 30260030).

PROTECT ACTION OF PUERARIN TO INTESTINE AND EXTRAINTESTINAL ORGAN IN INTESTINAL ISCHEMIA-REPERFUSION INJURY

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The aims of this study were to study the protect action of Puerarin to the lesion of intestine and extraintestinal organ, and to monitor concentration changes of cellular signal transductive factor-NO and cellular factor receptor-TNF-α in serum by intestinal ischemia-reperfusion injury. 30 rats were divided into three groups, 10 per group. The control group was injected N.S in abdomen. Animal model groups were injected Puerarin in abdomen with the dose of 100 mg/kg, 200mg/kg once daily, for 5 days. On the sixth day, 30 minutes after inject of medicine, rats were anesthetized to build the model of intestinal ischemia-reperfusion injury (IIRI). 45 minutes after superior mesenteric artery was closed, blood flow was recovered for 90 minutes, and then at the end of experiment, blood was obtained to detect the concentration of TNF-α and NO in serum. Part of liver, intestine, lung and kidney were used to observe the pathological diversity. We found Puerarin could relieve the pathological lesion of intestine and extraintestinal organ and increase the concentration of NO ($P<0.05$), and decrease the concentration of TNF-α ($P<0.05$). These results suggest that Puerarin have preserve action to the lesion of intestine and extraintestinal organ - liver, lung and kidney in IIRI in rats, which may related to concentration changes of cellular signal transductive factor NO and TNF-α in a dose-dependent manner.

PROTECTIVE EFFECT OF TELMISARTAN ON PANCREATIC β CELLS OF TYPE 2 DIABETIC RATS

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The aim of this study was to investigate whether Telmisartan had a protective effect on structure and function of pancreatic β cells of type 2 diabetic rats induced by high fat and fructose diet. The normal Sprague-Dawley (SD) rats were randomly divided into three groups: Standard Chow Diet (SCD) group was kept on a standard diet (4 % [w/w] fat, 51 % carbohydrate and 19 % protein); High Fat and Fructose Diet (HFFD) group was kept on a special diet (13 % [w/w] fat, 65 % D-fructose, 10 % protein); and High Fat and Fructose Diet with Telmisartan (HFFD + Tel) group was kept on the same special diet and treated with Telmisartan. Fasting Serum Glucose (FSG) and Fasting Serum Insulin (FSI) were measured every two weeks. Intraperitoneal injection Glucose Tolerance Test (IGTT) was performed to analyze insulin sensitivity. Insulin Release Test (IRT) was performed to evaluate function of pancreatic β cells. Body weight and pancreatic tissue weight of SD rats were measured, and islet morphology was assessed by haematoxylin and structure of pancreas was observed by eosin staining and immunohistochemistry. Reverse Transcription Polymerase Chain Reaction (RT-PCR) was performed to test the level of Glucose Transporter 2 (Glut 2) mRNA in pancreatic tissue. SD rats on high fat and fructose diet developed diabetes (blood glucose>9.0 mmol/l) due to insulin resistance and selective destruction of pancreatic β cells associated with severe loss of immunoreactivity of insulin and decrease of Glut 2 mRNA. In contrast, diabetic SD rats on treatment with Telmisartan remained normoglycaemic, and exhibited normal pancreatic islets and appropriate ability of insulin secretion. Our results indicate that Telmisartan had a protective effect on structure and function of pancreatic β cells of type 2 diabetic rats.

EFFECTS OF SINOMENINE ON CO/NO-cGMP SIGNALING CASCADE IN THE CEREBELLUM AND SPINAL CORD OF MORPHINE-DEPENDENT AND WITHDRAWAL MICE

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The CO/NO-cGMP signaling cascade plays an important role in morphine dependence and withdrawal. To explore the effect of sinomenine on the CO/NO-cGMP signaling cascade in the cerebellum and spinal cord of morphine -dependent and morphine-withdrawal mice, mice were subjected to injection of morphine with an increasing dose for 5 d (d1: 10 mg/kg, d2: 20 mg/kg, d3: 30 mg/kg, d4: 40 mg/kg, d5: 50 mg/kg, s.c.), and then were treated with sinomenine (40 mg/kg, i.p.) for another 5 d. Naloxone (4 mg/kg, i.p.) was used to develop acute withdrawal, and the withdrawal syndromes (including body weight, teeth chattering, twisting, straightening, sneezing, and ptosis) were investigated. The mRNA levels of HO2, nNOS, sGCα1 and sGCα2 in the cerebellum and spinal cord were determined by semi-quantitative RT-PCR, respectively. The results obtained were as follows: (1) Sinomenine restored the decrease of body weight and alleviated the signs of withdrawal in mice. (2) Sinomenine reduced the increase of mRNA level HO2, nNOS, sGCα1, and sGCα2 in the cerebellum and spinal cord resulting from morphine dependence. (3) Administration of sinomenine only did not develop physical