E0038  EFFECTS OF ANGIOTENSIN II ON ATPASES IN AORTIC VASCULAR SMOOTH MUSCLE CELLS FROM WISTAR-KYOTO RATS AND SPONTANEOUSLY HYPERTENSIVE RATS

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Aim
To explore the effects of Angiotensin II on the activities of Ca$^{2+}$-ATPase, Na$^{+}$,K$^{+}$-ATPase and mRNA expression levels of the plasma membrane Ca$^{2+}$-ATPase isoform 1 (PMCA1) and Na$^{+}$,K$^{+}$-ATPase a$_1$-subunit in cultured thoracic aortic vascular smooth muscle cells (AMSCs) from Wistar-Kyoto (WKY) rats and spontaneously hypertensive rats (SHR).

Methods
AMSCs isolated from 14-week-old male WKY rats and SHR were cultured and treated with different concentrations ($1\times10^{-9}$, $1\times10^{-8}$, $1\times10^{-7}$ mol/l) of Angiotensin II. The activities of Ca$^{2+}$-ATPase, Na$^{+}$,K$^{+}$-ATPase were measured by biochemistry and enzymology. RT-PCR assay was employed to determine the relative levels of PMCA1 and Na$^{+}$,K$^{+}$-ATPase a$_1$-subunit mRNA in AMSCs.

Results
Low and moderate concentration of Angiotensin II significantly increased the activity of Ca$^{2+}$-ATPase and Na$^{+}$,K$^{+}$-ATPase and up-regulated PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from WKY rats. Low and moderate concentration of Angiotensin II did not affect the activity of Na$^{+}$,K$^{+}$-ATPase and increased its a$_1$-subunit mRNA expression in AMSCs from WKY rats. Low and moderate concentration of Angiotensin II did not affect the activity of Na$^{+}$,K$^{+}$-ATPase and increased its a$_1$-subunit mRNA expression in AMSCs from WKY rats. Low and moderate concentration of Angiotensin II did not affect the activity of Na$^{+}$,K$^{+}$-ATPase and increased its a$_1$-subunit mRNA expression in AMSCs from WKY rats.

Conclusions
In WKY rats, Angiotensin II may have biphasic effects on Ca$^{2+}$-ATPase activity and PMCA1 mRNA expression, and may promote the activity of Na$^{+}$,K$^{+}$-ATPase in a dose-dependent manner in AMSCs. In SHR, Angiotensin II in hyper- and down-regulated PMCA1 mRNA level. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR. Three different concentrations of Angiotensin II significantly decreased the activity of Ca$^{2+}$-ATPase and PMCA1 mRNA level in AMSCs from SHR.

E0040  THE EXPRESSION OF SIGNAL TRANSDUCTION PATHWAY OF TGF1SMAD IN THE RAT CARDIAC TISSUE WITH TYPE 2 DIABETES AND INTERVENTION BY ATORVASTATIN

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Objective
Observation on changes of signal transduction pathway of TGF-β1/Smad in the course of myocardial fibrosis in the rat with type 2 diabetes and preventive effect of atorvastatin.

Methods
The experimental type 2 diabetic rats were yielded by injecting streptozotocin (STZ, 30 mg/kg) and fed with high fat and glucose food, then intervention by atorvastatin (20 mg·kg$^{-1}$·d$^{-1}$) for 12 weeks. Collagen content was observed by Masson staining. RT-PCR was used to observe the gene expression of TGF-β1, Smad2/3, Smad7 and were observed with the immunohistochemistry.

Results
The interstitial collagen accumulation and thickened capillary basement membrane in the atorvastatin (Masson stain: 0.80±0.16) administration group was obviously reduced compared with that of the control group (1.39±0.11, p<0.01). Lisinopril significantly relieved the collagen accumulation in the atorvastatin (Masson stain: 0.80±0.16) administration group and only high dose of Angiotensin II can suppress the activity and α₁ subunit mRNA expression of Na$^{+}$,K$^{+}$-ATPase in AMSCs.

E0039  EFFECTS OF LISINOPRIL ON THE ACTIVITIES AND MRNA EXPRESSION OF ION PUMPS IN AORTIC SMOOTH MUSCLE CELLS FROM SPONTANEOUSLY HYPERTENSIVE RATS

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Objectives
To explore the effects of Lisinopril upon the activities of Na$^{+}$, K$^{+}$-ATPase and Ca$^{2+}$-ATPase and mRNA expression levels of Na$^{+}$, K$^{+}$-ATPase a₁-subunit and plasma membrane Ca$^{2+}$-ATPase isoform 1 (PMCA1) in cultured thoracic aorta vascular smooth muscle cells (ASMCs) isolated from spontaneously hypertensive rats (SHR).

Methods
ASMCs were divided into four groups: Wistar-Kyoto (WKY) control, SHR control, Lisinopril (1×10$^{-7}$) intervened SHR group and Lisinopril (1×10$^{-7}$) intervened SHR group. The activities of ion pumps were detected by spectrophotography and mRNA expressions were measured by real time PCR. The content of Angiotensin II (Ang II) in cells-cultural medium were detected by radioimmunoassay.

Results
The activities of Na$^{+}$, K$^{+}$-ATPase, Ca$^{2+}$-ATPase and the mRNA expression levels of Na$^{+}$, K$^{+}$-ATPase a₁-subunit and PMCA1 in ASMCs from SHR were significantly lower than those from WKY control (p<0.01). Lisinopril significantly increased the activities of Na$^{+}$, K$^{+}$-ATPase and Ca$^{2+}$-ATPase and mRNA expression levels of Na$^{+}$, K$^{+}$-ATPase a₁-subunit and PMCA1 in ASMCs from SHR (p<0.01). Ang II content of culture medium in ASMCs from SHR was significantly more than those from WKY control (p<0.05). Lisinopril attenuated Ang II content of ASMCs culture medium from SHR (p<0.05).

Conclusions
The decreased activities of Na$^{+}$, K$^{+}$-ATPase and Ca$^{2+}$-ATPase may be related to their lower expression of the mRNA in ASMCs from SHR. Lisinopril may increase the activities of two ion pumps and upregulate the mRNA expression of Na$^{+}$, K$^{+}$-ATPase a₁-subunit and PMCA1 in ASMCs from SHR through blocking the generation of Ang II.