assessed while the plasma level of VPO1 in patients and the expression of VPO1 in arterial tissues was measured. Cultured human aorta vascular smooth muscle cells were treated with ANGII, and the proliferation activity, VPO1 expression, H2O2 and HOCl level were examined. The effect of VPO1 RNA interference, apocynin, catalase and PD98059 on VPO1 expression and the proliferation activity of cells were observed.

**Results** The VPO1 level/expression was significantly increased in patients with essential hypertension and in spontaneously hypertensive rats concomitant with definite vascular remodeling by evaluating the intima-media thickness, pressure-strain elastic modulus and stiffness index of carotid artery in patients, as well as the media thickness, lumen diameter, media thickness/lumen diameter ratio and mean nuclear area in artery media in spontaneously hypertensive rats. The angiotensin II-stimulated cell proliferation of human aorta smooth muscle cells was inhibited by knockdown of VPO1 using small hairpin RNA. Moreover, the NADPH oxidase inhibitor, apocynin, the hydrogen peroxide scavenger, catase, but not the ERK1/2 inhibitor, PD98059 attenuated Ang II-mediated upregulation of VPO1 and generation of hypochlorous acid.

**Conclusions** VPO1 is a novel regulator of vascular smooth muscle cell proliferation via NADPH oxidase/H2O2/VPO1/ERK1/2 pathway and plays an important role in vascular remodeling during hypertension.

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**ANGIOTENSINII MODULATES ION PUMPS OF SMOOTH MUSCLE CELLS DERIVED FROM UMBILICAL ARTERY OF HUMAN NEONATES WITH HYPERTENSIVE FAMILY HISTORY**

**Methods** Ion pump activities in cultured HUASMCs were detected by spectrophotography. The mRNA expression of sodium pump \( \alpha_{1} \)-subunit and plasma membrane Ca\(^{2+}\)-ATPase isoform 1 (PMCA1) in FH\(^{+} \) and FH HUASMCs was measured by RT-PCR.

**Results** Sodium pump, calcium pump activities in FH\(^{+} \) HUASMCs were higher than those in FH\(^{+} \) group (p<0.05), but the mRNA expression of sodium pump \( \alpha_{1} \)-subunit and PMCA1 showed no difference between two groups. In FH\(^{+} \) group, after 24-h treatment, AngII (1x10\(^{-7} \) mol/L) elevated the activities of sodium pump (4.62±0.26 vs 3.52±0.33) and calcium pump (4.00±0.31 vs 3.01±0.32), and up-regulated sodium pump \( \alpha_{1} \)-subunit mRNA expression (0.94±0.09 vs 0.69±0.05, n=5, p<0.01), however higher concentration AngII (1x10\(^{-6} \) mol/L) suppressed the activities of sodium (2.47±0.27) and calcium pump (1.79±0.27), and down-regulated sodium pump mRNA expression (0.44±0.05). Whereas, in FH\(^{-} \) groups, both concentration (1x10\(^{-6} \) and 1x10\(^{-7} \) mol/L) of AngII suppressed the activities of sodium pump (3.49±0.34, 2.21±0.35 vs 4.70±0.44) and calcium pump (2.83±0.31, 1.87±0.16 vs 4.27±0.48), but only AngII (10\(^{-6} \) mol/L) down-regulated their mRNA expression (\( \alpha_{1} \)-subunit: 0.51±0.13 vs 0.35±0.09, PMCA1: 0.165±0.049 vs 0.397±0.046, n=5, p<0.01).

**Conclusions** The activity of sodium pump and calcium pump is increased in FH\(^{+} \) HUASMCs. AngII inhibits both Na\(^{+} \) and Ca\(^{2+} \) ion pumps activities and mRNA expression in FH\(^{+} \) HUASMCs, and may have biphasic effects on ion pump activities and mRNA expression in FH\(^{+} \) HUASMCs.

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**ROLE OF MONOCYTE CHEMOTACTRUCT PROTEIN-1 ON LARGE ARTERIAL STRUCTURAL AND FUNCTIONAL CHANGE IN PREHYPERTENSIVE SUBJECTS**

**Background and Objects** Elevated blood pressure causes a change in vascular remodelling and arterial stiffness. Dynamic development of the inflammatory reaction may play a role in the early increase of blood pressure. Monocyte chemoattractant protein-1 (MCP-1) which has a chemotactic effect on monocytes/macrophages, is an initial factor of inflammation. However, whether monocyte chemoattractant protein-1 (MCP-1) is altered in the change of large arterial structure and function in prehypertensive subjects has been incompletely investigated.

**Method** According to the criteria of JNC7, 160 subjects were divided into three groups: (1) normotensive group (n=57), (2) prehypertensive group (n=50) and (3) hypertensive group (n=53). Brachium-ankle pulse wave velocity (BaPWV) was measured by an automatic wave-form analyser (Form FVV/ABI) and carotid artery intima-media thickness (IMT) was determined ultrasonographically. MCP-1 mRNA level were obtained by real time RT-PCR.

**Result** In prehypertensive subjects, MCP-1, baPWV and IMT levels are higher than that in normotensives (p<0.01) and lower than that in hypertensives (p<0.01). MCP-1 mRNA level correlated linearly and significantly with baPWV and IMT (p<0.01), even after adjustments for confounding variables.

**Conclusions** Large artery remodelling has been found in prehypertensive subjects. PWV and IMT were closely related to the level of blood pressure. MCP-1 may play a role structural and functional vascular changes in prehypertensive subjects.