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论著

血管加压素1a与血管加压素2受体在精氨加压素调节缺氧血管平滑肌细胞蛋白激酶C表达中的作用

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摘要 目的 观察血管加压素1a（V_{1a}）受体与V_2受体拮抗剂对精氨加压素（AVP）调节缺氧血管平滑肌细胞(VSMC) 蛋白激酶C（PKC）α，δ和ε亚型表达的影响，以及磷脂酶C（PLC）、磷脂酶D（PLD）和磷脂酶A_2（PLA_2）活性的变化。方法 缺氧培养大鼠肠系膜上动脉VSMC，采用Western蛋白印迹法检测PKC α，δ和ε亚型蛋白表达；采用酶偶联荧光分析法测定PLC和PLD的活性，酸碱滴定法检测PLA_2的活性。结果 缺氧处理1.5 h，VSMC胞膜PKC-α和ε亚型蛋白表达量明显升高，AVP进一步升高胞膜PKC-α和ε的表达。V_{1a}受体拮抗剂$d(CH_2)_5[Tyr^2(Me)]$AVP预处理可明显拮抗AVP诱导的胞膜PKC α和ε亚型蛋白表达升高，同时也明显拮抗AVP诱导的缺氧VSMC中PLC和PLD活性升高。而V_2受体拮抗剂$d(CH_2)[d\text{-}Ile^2Abu^4]$AVP对缺氧诱导的胞膜PKC-α和ε表达增加和VSMC中PLC和PLD活性升高无明显作用。结论 AVP诱导PKC激活的机制可能与V_{1a}受体介导的PLC/PLD途径有关，而V_2受体在这一信号传导途径中可能并不起主要作用。

关键词 受体,血管加压素；血管平滑肌细胞；缺氧；精氨加压素；蛋白激酶 C 磷脂酶C 磷脂酶D；磷脂酶A2
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Vasopressin-1a and vasopressin-2 receptors in argipressin regulating expression of protein kinase C of vascular smooth muscle cell after hypoxia

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Abstract

AIM To investigate the effects of vasopressin-1a(V_{1a}) receptor and V_2 receptor antagonists on argipressin (AVP) regulating the expression of protein kinase C (PKC)-α, δ and ε isoforms of vascular smooth muscle cell (VSMC) and the changes in phospholipases C (PLC), D (PLD) and A_2 (PLA_2) activity. **METHODS** VSMCs from superior mesenteric artery of rats were hypoxia-treated for 1.5 h. The expression of PKC-α, δ and ε isoforms was detected with Western blot. The PLC and PLD activities were assayed by enzyme-coupled fluorimetric analysis, and PLA_2 activity was assayed by acid-base titration. **RESULTS** After hypoxia, the expression of VSMC particulate PKC-α and ε increased, and AVP treatment further increased expression of PKC-α and ε in the particulate fractions. V_{1a}Receptor inhibitor $d(CH_2)_5[Tyr^2(Me)]$ AVP significantly antagonized this effect of AVP, simultaneously, also antagonized AVP-induced increase in PLC and PLD activities of VSMC after hypoxia. But V_2 receptor antagonist $d(CH_2)[d\text{-}Ile^2Abu^4]$ AVP had no significant influence on AVP-induced increase in expression of PKC-α and ε isoforms and the activities of PLC and PLD. **CONCLUSION** AVP induces translocation/activation of PKC isoforms in VSMC mainly through a V_{1a} receptor-dependent PLC/PLD mechanism, while V_2 receptor plays a lesser role in the signal transduction pathway.

Key words receptors vasopressin vascular smooth muscle cell hypoxia argipressin protein kinase C phospholipase C phospholipase D phospholipase A2

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