

论文

醛固酮合成酶基因及环境因素对高血压交互作用

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摘要:

目的 探讨汉族人群醛固酮合成酶基因(CYP11B2)启动子区-344C/T和第三外显子K173R多态性位点与原发性高血压(EHT)关系。**方法** 按病例对照研究方法选择来自黑龙江省哈尔滨市的2组人群(210例EHT患者和391名对照及补充的182例EHT患者和189名对照),利用聚合酶链反应-限制性片段长度多态性及直接测序技术分别对其进行-344C/T与K173R基因型检测,问卷调查和实验室检查获取传统危险因素。**结果** 总人群和男性中,-344C/T基因型分布在高血压组和对照组间差异均有统计学意义($P<0.05$),而K173R基因型分布仅在女性高血压组和对照组间差异有统计学意义($\chi^2=11.16, P=0.0038$);总人群中,-344C/T CC+TC、K173R KK分布与体质指数、腰围、血脂异常、高血糖对EHT均存在正交互作用,饮酒、吸烟与-344C/T CC+TC分布同时出现对EHT发生有促进作用;按性别分组的亚群中,除男、女血脂异常、女性血糖升高与-344C/T CC+TC分布同时存在对EHT存在负交互作用外,其余均为正交互作用。**结论** -344C/T、K173R基因多态性分布在汉族EHT与正常人群中存在差异;-344 C/T CC+TC、K173R KK与传统危险因素联合作用可促进EHT发生。

关键词: 原发性高血压(EHT) 基因多态性 危险因素 交互作用

Effects genetic polymorphisms of CYP11B2 and enviromental factors on development of essential hypertension

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Abstract:

Objective To explore the relationship between genetic polymorphisms of -344C/T in the promoter region, K173R in the exon-3 of aldosterone synthase gene (CYP11B2) and essential hypertension (EHT), and to identify whether classical risk factors interact with genetic polymorphisms in the development of the disease. **Methods** We used a nested case-control design to investigate the relationship between genetic polymorphisms of -344C/T, K173R and the risk of EHT. The genetic polymorphisms were detected with polymerase chain reaction-restriction fragment length polymorphism (PCR-RELP) in 210 EHT patients and 391 normal controls (182 complementary cases and 189 matched controls) from Harbin city. Information on several classical risk factors were collected. **Results** The differences in 344C/T genotype frequency distribution between EHT patients and normal controls were statistically significant in men and all subjects ($P<0.05$). The difference in K173R genotype frequency distribution between EHT patients and normal controls was statistically significant in women ($P<0.01$). Overall, there were several synergistic effects between the studied polymorphisms and classical risk factors, including overweight, abdomen obesity, hyperglycemia and dyslipidemia. The presence of -344C/T CC+TC (and also K173R KK) increased the odds of developing EHT when combined with the classical risk factors (synergism index >1). Alcohol drinking and smoking also increased the risk of EHT in the presence of -344C/T CC+TC. In the subgroup analyses, dyslipidemia interacted negatively with -344C/T CC+TC for both men and women, and hyperglycemia interacted negatively with -344C/T CC+TC in women. In addition, the risk factors interacted positively with -344C/T CC+TC as well as K173R KK. **Conclusion** The differences in -344C/T and K173R genotype frequency distributions between EHT patients and normal controls are statistically significant in Han people. Moreover, the combined effects of -344C/T CC+TC, K173R KK, and some classical risk factors significantly contribute to the development of the EHT.

Keywords: essential hypertension gene polymorphism risk factor interaction

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