ACE INSERTION/ DELETION POLYMORPHISM AND LONGEVITY


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Aim: Angiotensin I-converting Enzyme (ACE) exists mainly in the endothelial cells of the whole body and plays an essential role in two physiological systems, one leading to the production of angiotensin II (potent vasoconstrictor) and the other to the degradation of bradykinin. An insertion/deletion (I/D) polymorphism in intron 16 of the human ACE gene has been reported to be related to the levels of the circulating enzyme. A possible relation between ACE polymorphism and longevity has been studied and remains controversial[1-2]. Aim of our study was to compare the frequency of ACE allelic variants between a population of elderly subjects and in a control group. Methods: 117 centenarians subjects recruited from the Sardinia Centenarian AKeA project, and 136 control subjects were recruited and underwent clinical examination and data collection. ACE polymorphisms were determined by the polymerase chain reaction (PCR). Qualitative data were compared between groups by the #2 test. Results: Our results show a difference of statistical significance between centenarians and control group regarding on D/D variant frequency, which is more represented in centenarians. I/D variant frequency is less represented in centenarians. Conclusions: Although ACE gene role in longevity remains controversial and the sample size needs to be enlarged, our study suggests an association between ACE polymorphism and longevity. References: 1 JK Yang, YY Gong, L Xie et al. Lack of genetic association between the angiotensin-converting enzyme gene insertion/deletion polymorphism and longevity in a Han Chinese population. Journal of the Renine Angiotensin Aldosterone System, 2009; 10: 115-8. 2 F Panza, A D’Introno, C Capurso et al. Lipoproteins, vascular-related genetic factors and human longevity. Rejuvenation research, 2007; 10:441-58.

AP0 E ALLELIC VARIANTS IN HEALTHY ELDERLY PEOPLE


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Introduction: In the quest to understand the biological basis of human longevity, it can be supposed that genes and biochemical markers could be implicated in coronary artery disease (CAD), cerebrovascular disease (CVD), and Alzheimer’s disease (AD) and may have a role in human longevity. Among genetic markers, the apolipoprotein E (APOE) gene has been widely examined in different studies because of its well-documented role in AD and vascular diseases. Apolipoprotein E is a polymorphic glycoprotein that plays an essential role related to binding to receptors for the uptake of Chylomicrons and VLDL remnants and of LDL. The three ApoE major isoforms are E3 (Cys112/Arg158), E4 (Arg112/Arg158) and E2 (Cys112/Cys158). In the healthy population, the presence of e4/e4 homozygote genotype is associated with an increased risk of Alzheimer’s disease (AD)[1]. Bennet et coll.[2] conducted a detailed analysis about relationship of apoe status, lipid levels, and coronary outcomes. They showed approximately linear relationships of apoe genotypes (when ordered c2/c2, c2/c3, c2/c4, c3/c3, c3/c4, c4/c4) with LDL-C levels and CAD risk. Aim of the study was to verify the frequencies of APO E allelic variants in a healthy elderly population. Methods: 71 centenarians subjects (range 100-104 years) and 97 control subjects (range 90-99 years) were recruited from the Sardinia Centenarian AKeA project and underwent clinical examination and data collection. ApoE polymorphisms were determined by Real Time-PCR. Qualitative data were compared between groups by the #2 test. Results: Our results reveal that ApoE2/c2 allele frequency shows a marked increase after 100 years old and confirms the hypothesis that this allele is related to longevity. Conclusions: Our results confirm the hypothesis of a probable link between c2 allele and longevity and focus the attention on possible mechanisms underlying this link. References: 1 OY Bang, YT Kwak, IS Joo et al. Important Link Between Dementia Subtype And Apolipoprotein E: A Meta-Analysis. Med J, 2003; 44: 401-413. 2 AM Bennet, E Di Angelantonio, Z Ye et al. Association Of Apolipoprotein E Genotypes With Lipid Levels And Coronary Risk. JAMA, 2007; 298: 1300-1311.