Genetic influence on left ventricular hypertrophy in endurance athletes

MariaAngela D'Amico[1], Pascal Izzicupo[1], Alessia DiFonso[1], Adriana Bascelli[1], Mariangela Carrideo[2], Sabina Gallina[2], Angela DiBaldassarre[1]

[1] Department of Medicine and Aging Science, Human Morphology Section, University G. D'Annunzio of Chieti-Pescara, Italy
[2] Department of Human Movement, University G. D'Annunzio of Chieti-Pescara, Italy

Exercise activates the renin-angiotensin system (RAS), thus modulating myocardial growth. A long period of training may upregulate of angiotensin-converting enzyme (ACE) so inducing the adaptive left ventricular hypertrophy (LVH), common in endurance athletes. LVH is multifactorial, but genetic factors seems to play the foremost role. Particularly, the presence of double deletion (D) of intron-16 of ACE gene is correlated to highest plasma and tissue enzyme levels, which seems to be associated with more extent of LVH in endurance athletes rather than presence of insertion/deletion (ID) genotype. ACE influences the myocardial cell growth also through the angiotensin type 1 receptors (AGTR1), the major mediator of cardio-circulatory effects of angiotensin II (AngII). It was identified a single nucleotide polymorphism of AGTR1 gene implicated in cardiovascular diseases. A1166C polymorphism is likely related to increased Ang II sensitivity, but its association with LVH remains still undemonstrated. Our aim was to evaluate the role of ACE and AGTR1 polymorphisms in LVH promoting in endurance athletes.

A group of 74 male endurance athletes, aged 25-40 years, were enrolled. All participated primarily in isotonic sports, training for at least >10 h/week, for at least 5 years. The ACE genotypes were: DD 35, ID 36 and II in 3. The group II was excluded from the analysis due to its small size. LVMI was significantly higher in group DD rather than in ID. The group DD showed a slightly higher prevalence of subjects with LVH (LVMI >131g/m2) than ID, (p=0.120). No association was found between ACE-DD and LVH. Concerning the role of AGTR1 polymorphism, the highest LVMI was found in 15 athletes with ACE-DD and AGTR1-AC/CC genotypes; the lowest LVMI value was found in ACE-ID and AGTR1-AA, whereas LVMI in subjects with ACE-DD+AGTR1-AA was similar to ACE-ID+AGTR1-AC/CC group. The presence ACE-DD+AGTR1-AC/CC was strongly associated with LVH(p=0.029). Subjects with LVH showed longer left ventricular isovolumetric relaxation time and higher end-systolic wall stress. LVMI may be greater in the presence of ACE DD and AGTR1-AC/CC polymorphisms.

Key words
Athletes' heart, angiotensin, ACE, polymorphism