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Cardiovascular remodeling in response to physical exercise: …not only training!

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Athletic performance is multifactorial condition which is determined by the complex and poorly understood interactions of both environmental and genetic factors. Although much work has been done to identify the non-genetic components that are associated with performance and susceptibility to the related cardiovascular injuries, there is an ever growing body of research investigating the genetic contribution to these phenotypes. Long-term physical exercise is indeed associated with changes in cardiac morphology and electrocardiographic modifications. Both functional and structural remodelling adaptations together with their magnitude and time-course depend upon training duration, intensity and the vessel beds involved. A well-known adaptive mechanism in trained athletes consists of adaptive left ventricular hypertrophy (LVH), but different extents of LV growth have been reported, independently from the length and the amount of training. It is well known that angiotensin II plays a pivotal role in inducing myocardial hypertrophy. With this in mind, we investigated the influence of polymorphisms of ACE (angiotensin-converting enzyme) and of angiotensin type 1 receptor genes. We also analyzed the effects of genotypic variants of enzymes involved in redox processes. In particular, we focused our attention on the functional role of the C242T variants of NADPH p22phox subunit that, modifying the intensity of the inflammatory and oxidative response to the physical exercise, elicit effects on the cardiovascular district.