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Apoptotic Changes in Aging Testis

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Cell apoptosis is an active process which occurs in the course of aging. The direct result of the aging of the testis is reduction in levels of testosterone (T) and results of this are disrupting of physiological processes and dramatically worsened the quality of life for millions of older men. According to the theory of oxidative stress, with the leading factors free radicals (ROS- Reactive oxygen species), mitochondria are the biological clock of aging and the main place of oxidative injury in the course of this process. ROS attack primarily mitochondria and their DNA (mt DNA), increased permeability of the mitochondrial membrane, reduced membrane potential of mitochondria and these are the early signs of beginning apoptotic changes in the cells. Mitochondrial damage caused by ROS disrupts steroidogenesis in the testis and at the same time the process of steroidogenesis generates sub-products, which could also be responsible for functional insufficiency and apoptotic changes in aging LC. Apoptosis is a complex process which is regulated from multiple levels pro-and anti-apoptotic proteins and long-term damage to the balance between the two groups causes apoptotic changes in cells. One of this proteins is tumor suppressive protein p53 – promotes the expression of a number apoptotic genes. At this stage, the role of p53 in the control of apoptosis in human steroidogenic LC in the aging testis is not well investigated which determines the aim of the present study. We used material from human testis of patients between 56, 68 and 80 years old, embedded in paraffin and prepared for immunohistochemistry. Our results demonstrated the increased intensity of the immunoexpression of p53 in the course of the aging and these findings categorically point out the role of this protein in the regulation of testis apoptosis.

Key words: apoptosis, Leydig cells, aging, immunoexpression, p53.