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1. Introduction

It is well known that blood testosterone level declines during the course of male aging (Feldman et al., 2002; Harman et al., 2001), a phenomena that is associated with the decreases in bone density, muscle mass and strength, sexual function and other physiological parameters (Kaufman & Vermeulen, 2005; Matsumoto, 2002; Vermeulen, 2000). Previous studies reported that serum testosterone concentrations were lower in the male patients with Alzheimer’s disease in comparison to non-demented and age-matched men (Hogervorst et al., 2001; Moffat et al., 2004). Further studies observed that supplementation with testosterone in rats reduced β-amyloid peptide and hyperphosphorylation of τ-protein, two biomarkers of the disease (Gouras et al., 2000; Papasozomenos & Shanavas, 2002; Ramsden et al., 2003). The studies suggest that low blood testosterone is a possible risk factor for the development of Alzheimer’s disease (Rosario & Pike, 2008). The decline in blood testosterone is a progressive process in male aging. Several longitudinal studies on the blood testosterone of aging males indicated that the incidence of hypogonadism increased with age (Feldman et al., 2002; Harman et al., 2001). In addition, many pathological and stress-related factors may accelerate this process. Therefore, delaying the decline in blood testosterone is clinically significant for the health of aging males suffering from hypogonadism.

For delaying the decline in testosterone, understanding the mechanisms responsible for the decline is important. The studies in the last decades reported multiple factors and alterations in aging process that affect the levels of blood testosterone (Wang & Stocco, 2005). The studies further indicated that the primary reason for the decline is the decrease in testosterone biosynthesis during male aging (Chen et al., 1994). Testosterone is principally synthesized in testicular Leydig cells from the substrate cholesterol and released into the blood circulation (Miller, 1988). The rate-limiting step in testosterone biosynthesis is the transfer of cholesterol to the mitochondrial inner membrane to initiate the steroidogenic process in Leydig cells (Stocco & Clark, 1996). This step is regulated by the steroidogenic acute regulatory (StAR) protein, a critical factor in steroid hormone biosynthesis that controls the cholesterol transfer to the mitochondrial inner membrane (Clark et al., 1994; Lin et al., 1995; Wang et al., 1998). However, StAR protein also declines in Leydig cell aging and


This book provides the most up-to-date information on the basic and clinical aspects of endocrinology. It offers both researchers and clinicians experts, gold-standard analysis of endocrine research and translation into the treatment of diseases such as insulinoma, endocrine disease in pregnancy and steroid induced osteoporosis. Investigates both the endocrine functions of the kidneys and how the kidney acts as a target for hormones from other organ systems. Presents a uniquely comprehensive look at all aspects of endocrine changes in pregnancy and cardiovascular effects of androgens.

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