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Testosterone hormone and body building

Testosterone is a steroid hormone from the androgen group. In mammals, testosterone is primarily secreted in the testes of males and the ovaries of females, although small amounts are also secreted by the adrenal glands . It is the principal male sex hormone and an anabolic steroid. In both men and women, testosterone plays a key role in health and well-being as well as in sexual functioning. Examples include enhanced libido, increased energy, increased production of red blood cells and protection against osteoporosis. On average, an adult human male body produces about forty to sixty times more testosterone than an adult female body, but females are, from a behavioral perspective (rather than from an anatomical or biological perspective), more sensitive to the hormone However the overall ranges for male and female are very wide, such that the ranges actually overlap at the low end and high end respectively [1] . Androgens promote protein synthesis and growth of those tissues with androgen receptors.

Testosterone effects can be classified as virilizing and anabolic, although the distinction is somewhat artificial, as many of the effects can be considered both .Anabolic effects include growth of muscle mass and strength, increased bone density and strength, and stimulation of linear growth and bone maturation. Virilizing effects include maturation of the sex organs, particularly the penis and the formation of the scrotum in unborn children, and after birth (usually at puberty) a deepening of the voice, growth of the beard and axillary hair. Many of these fall into the category of male secondary sex characteristics [2] . Steroids are dangerous drugs, and when used inappropriately, they can cause a host of severe, long-lasting, and often irreversible negative health consequences. These drugs can inhibit the height of growing adolescents, masculinize women, and alter sex characteristics of men. Anabolic steroids can lead to premature heart attacks, strokes, liver tumors, kidney failure and serious psychiatric problems.

Special points of interest:

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In addition, because steroids are often injected, users risk contracting or transmitting HIV or hepatitis.The anabolic steroids may cause side effects such as: inhibition of natural hormones [3], liver damage [4], increase LDL cholesterol level [5], Gynocomastia (Development of breast tissue in males), development of acne, cardiovascular problems ,Virilization (Development of male characteristics in women), prostate enlargement, high blood pressure, kidney problems, immune system changes and sterility in males and females[6].Body-building supplement fails to strengthen muscle and may harm health. Androstenedione, a popular supplement taken by athletes to enhance sporting performance does not increase muscle development and strength or increase testosterone in the blood but may produce adverse health effects, report researchers from Iowa State University (Ames, IA, USA). Androstenedione is banned by the International Olympic Committee. However, it is allowed by baseball authorities in the USA where, since admission of its use by home-run record holder Mark McGwire, there has been a five-fold increase in androstenedione use by young people. Androstenedione, a testosterone precursor, is normally produced by the adrenal gland and gonads and is converted to testos - terone by 17 b-hydroxysteroid dehydrogenase. It is also produced by some plants e g, the wild yam and is sold as a natural alternative to anabolic steroids The effects of oral androstenedione supplements in men on serum testosterone and on musclefibre size and strength was investigated in a study on 20 men with normal testosterone values, aged 19–29 years. The supplement's effects on blood lipids and liver function were also investigated .The supplement's effects on blood lipids and liver function were also investigated .. All the men did 8 weeks supervised whole-body resistance training. Ten were given 300 mg per day of the supplement during weeks1, 2, 4, 5, 7 and 8; ten were given placebo (JAMA 1999; 281: 2020–28).Muscle strength did not differ between the two groups and testosterone concentrations were unaffected by the supplement. Liver-function enzymes remained normal but there was a reduction in serum HDL cholesterol, and serum oestradiol and oestrone concentra tions were raised in the men taking androstenedione.

Increased oestro - gen values in men are associated with gynaecomastia, and an increased risk of cardiovascular disease and pancreatic cancer. This is the first study to look at androstenedione's effects on men. A previous study, involving two women given a single dose of androstenedione, found that the supplement raised their serum testosterone concentrations [7].

References:

(1)- Bhasin S, Storer TW, Berman N, et al (1996). "The effects of supraphysiologic doses of testosterone on muscle size and strength in normal men". N. Engl. J. Med. 335 (1): 1–7.

(2)- E.R. Freeman, D.A. Bloom, and E.J. McGuire (2001). "A brief history of testosterone". Journal of Urology 165: 371–373 .

(3) Journal of Steroid Biochemistry and Molecular Biology. (2003) 369-375.

(4)- Molano F, Saborido A, Delgado J, Moran M, Megias A. Rat liver lysosomal and mitochondrial activities are modified by anabolic-androgenic steroids. Med Sci Sports Exerc. 1999 Feb; 31(2):243-50.

(5)- Ronald S. Swerdloff, MD, Jason Ng, MD, and Gladys E. Palomeno, MD, GYNECOMASTIA: etiology, diagnosis, and treatment. March 1, 2004.

(6)- . Pol Merkuriusz Lek. Am J Clin Dermatol. 2002; 3(8):571-8. 2. Clin Dermatol. 2004 Sep-Oct; 22(5):419-28. 3. 2004 May; 16(95):490-2.)

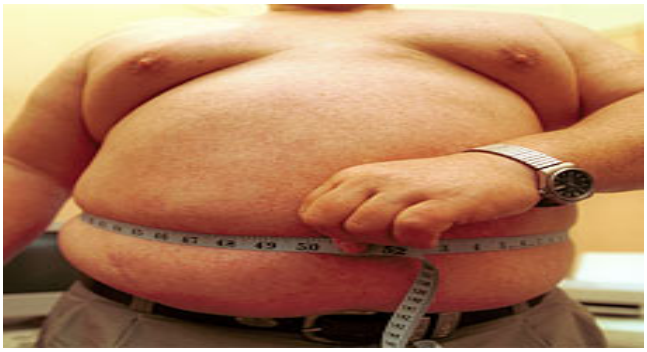
(7)- Karen Birchard. Body-building supplement fails to strengthen muscle and may harm health. THE LANCET Vol 353 June 5, 1999.

Leptin and Obesity

Obesity is a disorder which is due to formation of high amount of body fat that frequently results in a significant impairment of health. Doctors generally agree that men with more than 25% body fat and women with more than 30% are obese. Obesity is a known risk factor for chronic diseases including heart disease, diabetes, high blood pressure, stroke and some forms of cancer. Evidence suggests that obesity has more than one cause: genetic, environmental, psychological and other factors may all play a part [1].40 years ago, the lipostatic theory of energy balance regulation proposed that circulating factors, generated in proportion to body fat stores, acted as signals to the brain, eliciting changes in energy intake and expenditure.The discovery of leptin and its receptors has now provided a molecular basis for this theory [2]. The hormone leptin, produced by adipocytes (fat cells), was discovered about three years ago in mice. Subsequently the human Ob gene was mapped to chromosome 7. Leptin is thought to act as a lipostat: as the amount of fat stored in adipocytes rises, leptin is released into the blood giving signals to the brain by binding to the appetite center, That means'' the body has enough to eat or satiety. ''. A very small group of humans possess homozygous mutations for the leptin gene which leads to a constant desire for food, resulting in severe obesity. This condition can be successfully treated by the administration of recombinant human leptin [3].Thus, circulating leptin levels give the brain input regarding energy storage so it can regulate appetite and metabolism [4]. The relative stability of body weight over the long term and under a variety of environmental conditions that alter short-term energy intake and expenditure provides strong evidence for the regulation of body energy content.Leptin's effects have been shown to cover a broad spectrum of metabolic, neuroendocrine, and behavioral systems, the functions of which are closely tied to nutritional status(5). It has complex effects on the storage and metabolism of fats and carbohydrates. These are mediated both directly, through actions on specific tissues, and indirectly, through CNS endocrine and neural mechanisms. Adipose tissue is both the primary site of leptin production and a major effector organ for many of leptin's actions.

Leptin has been shown to alter glucose and fatty acid uptake and metabolic pathways involved in lipid oxidation and synthesis. Chronically elevated levels of leptin either peripherally or centrally, result in activation of nuclear transduction pathways that trigger a cascade of events leading to reversal of adipocyte maturity and even cell death by apoptosis. However, most overweight people have high levels of leptin in their bloodstream, indicating that other molecules also affect feelings of satiety and contribute to the regulation of body weight. Though, the discovery of leptin and its receptors provided a molecular basis for the lipostatic theory of body energy balance regulation [6], triggering an explosion of research in the field.

Humans and animals with leptin deficiency due to a genetic defect exhibit a similar range of abnormalities, including marked obesity, hyperphagia, and pituitary deficiency, and administration of exogenous leptin in physiological amounts results in reduction of food intake and body weight [7, 8, 9]. However, the role of leptin in obesity of nongenetic origin is unclear. Leptin synthesis and secretion increase as fat stores increase, which suggests that in non–leptin-deficient obese individuals, a state of relative leptin resistance can develop [10].



References:

(1)-Gorden P, Gavrilova O (2003). The clinical uses of leptin. Curr Opin Pharmacol. dec; 3 (6):655-9.

(2)- Kus I, Sarsilmaz M, Colakoglu N, et al (2004). "Pinealectomy increases and exogenous melatonin decreases leptin production in rat anterior pituitary cells: an immunohistochemical study". Physiological Research / Academia Scientiarum Bohemoslovaca 53 (4): 403–8.

(3)- Alonso-Vale, Maria Isabel Cardoso; Sandra Andreotti, Sidney Barnabe Peres, Gabriel Forato Anhe, Cristina das Neves Borges-Silva, Jose Cipolla Neto, Fabio Bessa Lima (November 2004). "Melatonin enhances leptin expression by rat adipocytes in the presence of insulin (abstract)". Am J Physiol Endocrinol Metab. Retrieved on 2007-12-15.

(4)- Erickson JC, Clegg KE (1996). Sensitivity to leptin and susceptibility to seizures of mice lacking neuropeptideY. Nature 381:415–21.

(5)- Kennedy GC (1953). The role of depot fat in the hypothalamic control of food intake in the rat. Proc. R. Soc. 140:578–92.

(6)- Friedman JM, Halaas JL. (1998). Leptin and the regulation of body weight in mammals. Nature 395:763–70.

(7)- Farooqi IS, Jebb SA, Langmack G, et al. (1999). Effects of recombinant leptin therapy in a child with congenital leptin deficiency.

N. Engl. J. Med. 341:879–84.

(8)- Montague CT, Farooqi IS, Whitehead JP, Soos MA, Rau H, et al. (1999). Congenital leptin deficiency is associated with severe early-onset obesity in humans. Nature 387:903–7.

(9)- Pelleymounter MA, Cullen MJ, Baker MB, Hecht R, Winters D, et al. 1995. Effects of the obese gene product on body weight regulation in ob/ob mice. Science 269:540–43.

(10)- Campfield LA, Smith FJ, Guisez Y, Devos R, Burn P. (1995). Recombinant mouse OB protein: evidence for a peripheral signal linking adiposity and central neural networks. Science 269:546–49.

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