INTRODUCTION

Anabolic steroids in sports is also known as "badboys" drug, because of their use as doping for performance enhancing and winning at any cost purposes. Some athletes take anabolic steroids to increase muscle mass and strength, others to train harder and recover faster. This is not all the truth about anabolic steroids. Endogenous anabolic steroids have also important role in fair play or clean competition, as anabolic and antieatabolic effects are useful in increase of muscle mass, strength. Train harder and recover faster. Intensive and high volume exercise training accompanies with increase of catabolic process. Anabolic steroids may block catabolic effect of cortisol, so prevent muscle protein breakdown and speed recovery after exercise training. Use of changes in endogenous hormone ensemble is part of modern athletes training program which helps the min performance enhancement and is a sign of a wise training management.

Catabolic Action of Corticosteroids

Glucocorticoids, steroid hormones that affect the carbohydrates metabolism, have anti inflammatory effect and many side effects of catabolic action. The catabolic effect of glucocorticoids depends on individual tissues. In contrast to the loss of protein from skeletal muscle, bone, lymphoid tissue, and smooth muscle, there is an increase in the rate of cardiac, hepatic, and urogenital activities [1]. The catabolic action of glucocorticoids in skeletal muscle depends on the type of muscle fibers [2]. Muscle fibers, which have the lowest oxidative capacity (IIB / X) among the striated muscle fibers, are most sensitive to the catabolic action of glucocorticoids. In these fibers (IIB / X) the activity of non-lysosomal proteases increases significantly after glucocorticoid excess [2]. Excess of glucocorticoids depress testosterone and insulin levels [3,4], although the inhibition of protein synthesis after glucocorticoid administration plays a lesser role in the atrophy of muscle fibers than accelerated protein catabolism [1]. It is well known that the content of lysosomes in type IIB/X muscle fibers is low; the role of lysosomal proteases in the development of corticosteroid myopathy is not significant. The process of atrophy starts from peripheral myosin filaments. Actin filaments are more resistant to the catabolic action of glucocorticoids. Muscle weakness in corticosteroid myopathy is the result of destruction and atrophy of the myofilbrils [5).

The higher the degree of atrophy, the lower the muscle elasticity and the higher the tone. Muscle tone is dependent on changes in innervations. It has been shown that glucocorticoid myopathic fast-twiftch (FT) muscles' neuromuscular synapses are destroyed [6]. A decrease of titin and myosin [7] and of the ratio of nebulin and myosin heavy chain (MyHC) in myopathic muscle [8] shows that these changes in contractile and elastic proteins are the result of elevated catabolism of the above mentioned proteins in skeletal muscle. This is the reason for reduced elasticity and generation of tension in glucocorticoid-caused myopathic muscle.


catabolic Action and Muscle Function

Moderate endurance type of exercise is effective in retarding muscle atrophy [9] and protecting against wasting [10]. The effect of moderate exercise in inducing a less pronounced catabolic effect of glucocorticoid is caused by the elevation of antieatabolic activity of this type of exercise and related with the endogenous action of androgens in the stimulation of antieatabolic activity in type IIB/X muscle fibers [2]. Skeletal muscles with higher oxidative capacity, particularly slow twitch (ST) fibers, are less sensitive to the catabolic action of glucocorticoids. This phenomena was explained by the lesser elevation of proteolytic activity in ST Oxidative muscle fibers, but in muscle fibers where oxidative capacity is low (IIB/X), the catabolic activity was more pronounced [11]. As ST oxidative muscle fibers are involved in the maintenance of static body posture, in slow repetitive
movements, and being functionally active even when FT fibers are passive, it may be also explain why there is no significant catabolic action of glucocorticoids and aatrophy of ST muscle fibers [11]. Atrophy of muscle fibers with low oxidative capacity is the result of inhibition of insulin-like growth factor-1 (IGF-1) [12] and upregulation of two genes, myostatin and glutamatesynthase [13]. Muscle activity increases the synthesis rate of myofibrillar proteins [14] via a mammalian target of rapamycin-activating proteins within the nitrogen-activated protein kinase signalling [15]. The recovery from the last exercise session, particularly from intensive exercise, is faster in the young than in the elderly [16].

Catabolic Action and Contractile Apparatus

Muscle fibers and myofibrils of glucocorticoid-caused myopathic FT glycolytic muscle are thinner. Myosin filaments disappeared completely from one fifth of the area of myofibrils of myopathic muscle [5]. The intensive destruction of myofibrils and degradation of contractile proteins, including MyHC IIb isoform [17], are the main reasons for reduced muscle strength, motor activity, and weakness in glucocorticoid-caused myopathic rats [18,19]. Destruction of myofibrils starts from the periphery of glycolytic muscle fibers. Myosin filaments disappeared from some sarcomeres [11]. The second reason why myofibrils of myopathic muscle fibers are thinner is the slower myofibrillar protein synthesis rate and assembly of filaments. The decrease of the MyHC IIb iso form relative content and the increase of the MyHCId isoform show that the quantitative changes in myofibrils are significantly related to the qualitative remodelling of thick myofilaments in myopathic glycolytic muscle fibers [18]. Changes in the myofibril ultra structure of myopathic muscle fibers are also related to the functional modification of glycolytic muscle fibers. These modifications were not observed in muscle fibers with higher oxidative capacity in myopathic rodents [18]. Glucocorticoids caused wasting in senescent and young rodents as a result of the loss of FT fibers, their myofibrils, contractile proteins, and conversion of muscle fibers with low oxidative capacity into higher oxidative capacity. The MyHC and actin synthesis rate decreased by about 20–30% and contractile proteins turned over very slowly after glucocorticoid treatment.

REFERENCES


