Resistance Training Associated with Nandrolone Decanoate Alters Myostatin and Atrogin-1 RNAm, but not MyoD and Murf

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Androgenic-anabolic steroids (AAS) are spread among athletes and no athletes in order to improve performance or physical appearance. AAS targets the satellite cells in skeletal muscles, which are essential for muscle growth and repair. The understanding of AAS mechanism of action in skeletal muscle is critical for a better comprehension of muscular physiology under AAS use. The aim of this study was to investigate the effects of overload aquatic plyometric training associated to AAS supraphysiological dose on the expression of modulators of skeletal muscle pathways of atrophy and hypertrophy. Wistar rats were grouped into: sedentary (S); trained (T); S with AAS (SA); and T with AAS (TA). Exercised groups performed jumps in water: 4 sets of 10 jumps each and 30-second of rest interval between series, for 7 weeks with a progressive overload of 50 to 80% of body weight. AAS (5 mg/kg – supraphysiological dose) was injected sc twice a week. After last exercise session animals were killed.. Myostatin, MyoD, Atrogin-1 and Murf mRNA expression was determined in the gastrocnemius muscle extracts by real-time RT-PCR. The exercise did not change RNAm expression of any studied genes while AAS or its association with training increased atrogina-1 and reduced myostatin RNAm. The expression of MyoD and MURF was not altered by AAS. These results suggest that the positive effects of AAS on skeletal muscle hypertrophy may also involve downregulation of myostatin. However, atrophy pathways may be also activated.

Key words: aquatic plyometric training; nandrolone decanoate; overload.

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