THE SIGNIFICANCE OF ENDOGENOUS TESTOSTERONE ON MUSCULAR RATE OF FORCE DEVELOPMENT IN RESPONSE TO STRENGTH TRAINING

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It is known that testosterone interact with skeletal muscles and is a component of an endocrine system (e.g. growth hormone, IGF-I, insulin, cortisol) which is reported to mediate adaptive changes in skeletal muscles in response to strength training (ST) (Kraemer et al. 1990, Kvorning et al. 2006, 2007). However, it is not known if the level of endogenous testosterone influences the quality of muscle in terms of changes in contractile Rate of Force Development (RFD).

Endogenous secretion of testosterone can be suppressed by the use of GnRH analogues (Cockshott 2000). Thus, we hypothesized that suppression of endogenous testosterone will inhibit the adaptation to ST leading to a less pronounced increase in RFD. Twenty-two young men participated in this randomized, placebo-controlled, and double-blinded intervention study. The participants were randomized for treatment with a GnRH-analogue (goserelin, 3.6 mg every 4 weeks) or placebo for a period of 12 weeks (goserelin group (G) n=12, placebo group (P) n=10). The ST period of 8 weeks started after 4 weeks of treatment and included exercises for all major muscles (3 – 4 sets per exercise x 6 – 10 repetitions with corresponding 6 – 10 repetition maximum (RM) loads, 3/week). Isometric dynamometry (KinCom) and blood sampling were performed at weeks 4 and 12. Resting serum testosterone decreased significantly (p<0.01) in G from 22.6 ± 1.6 (mean ± SE) nmol/l to 2.0 ± 0.1 (week 4), whereas it remained unchanged in P. In G isometric quadriceps RFD decreased 8 % (p<0.05) in the very initial contraction phase (0 – 50 ms) following 8 weeks of ST, whereas P showed no changes. Isometric quadriceps strength increased in P (p<0.05), while unchanged in G. It could be an indication that overtraining was present after ST since RFD remained unchanged in P and decreased in G. However, it seems that suppression of testosterone to some extent leads to impaired training adaptability in terms of contractile RFD. Suppressed endogenous testosterone levels have been shown to affect adaptation to ST in terms of decreased changes in isometric strength and muscle mass (Kvorning et al. 2006). In addition, testosterone acts on neural tissue through different mechanisms, ranging from neurotransmitter synthesis and release to development and remodelling of synaptic circuitry (Alonso-Solis et al. 1996). Moreover, changes in circulating steroid hormone levels may impair the sensitivity of the neuroendocrine system (Alonso-Solis et al. 1996). Together this may influence the ability to exert large contractile RFD in G. In conclusion, 10 – 20 fold lower resting testosterone levels in G resulted in significant decreases in RFD after ST.

Alonso-Solis et al., Cell Mol Neurobiol 16: C357-C382, 1996.  

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