During volitional eccentric muscle contraction, as in all other types of voluntary muscle contraction, the EMG interference signal recorded by use of surface electrodes during eccentric muscle contraction constitutes a complex outcome of motor unit recruitment and motoneuron firing frequency (rate coding). In addition, the net EMG signal amplitude relies on the specific summation pattern of the individual motor unit action potentials (MUAPs), which among other things is affected by the degree of motor unit synchronization.

A neural regulatory mechanism that limits the recruitment and/or discharge rate of motor units has been suggested to exist during maximal voluntary eccentric muscle contraction, since in untrained subjects the EMG recorded during eccentric muscle contraction of maximal volitional effort appear to be markedly reduced compared to that of maximal concentric contraction (Westing et al. 1991, Seger & Thorstensson 1994, Aagaard et al. 2000, Komi et al. 2000).

Notably, this apparent inhibition in motoneuron activation during maximal eccentric contraction appears to be down-regulated with certain types of strength training. Thus, the observed suppression in eccentric EMG signal amplitude was partially abolished in parallel with a gain in maximal eccentric muscle strength following intense heavy-resistance strength training (Aagaard et al. 2000, Andersen et al. 2005).

The specific neural pathways responsible for the suppression in muscle activation during maximal eccentric contraction remain unidentified. During maximal voluntary muscle contraction, efferent motoneuronal output is influenced by central descending pathways, afferent inflow from group Ib Golgi organ afferents, group Ia and II muscle spindle afferents, group III muscle afferents and by recurrent inhibition. All of these pathways may exhibit adaptive plasticity with training.

A possible mechanism responsible for the selective increase in eccentric muscle strength with strength training could be down-regulation in spinal inhibitory interneuron activity mediated via Golgi organ Ib afferents. Furthermore, the H-reflex appears to be markedly suppressed during maximal eccentric muscle contraction in vivo, suggesting the presence of presynaptic Ia afferent inhibition (Duclay & Martin 2005). It is possible, therefore, that strength training leads to reduced presynaptic inhibition of muscle spindle Ia afferents during maximal eccentric muscle contraction, which will increase the excitatory inflow to the spinal motoneuron pool and thereby increase EMG and maximal eccentric muscle strength.

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