MODULATED SPINAL EXCITABILITY IN DROP-JUMPS FROM DIFFERENT FALLING HEIGHTS
Leukel Christian, Taube Wolfgang, Gruber Markus, Gollhofer Albert (University of Freiburg, Germany)

Introduction
The stretch-shortening cycle is characterised by stretching of the target muscle (eccentric phase) prior to a subsequent shortening or concentric phase. Stretch reflexes in the eccentric phase were argued to influence the performance of short lasting SSCs in running, hopping and drop-jumps. The short latency component (SLR) of the stretch reflex increased with increased jump height in drop-jumps. However, in jumps from excessive heights, the SLR was diminished (Komi and Gollhofer 1997). The observation that, despite larger impact loads, the neuromuscular activation was decreased was argued to represent a protection inhibition in order to reduce eccentric stress on the tendomuscular system (Komi and Gollhofer 1997). Thereby, changes in the SLR could be induced on the spinal level or could be caused by an altered fusimotor drive. The aim of the present study was to investigate spinal modulations at SLR at different falling heights.

Methods
10 subjects (age 25 ± 4 years) with no history of orthopaedic and neurological disorder participated in this study. Soleus H-Reflex excitability was compared at SLR between jumps from 31 cm (low height, LH) and 76 cm (excessive height, EH). H-Reflex stimulation was used in accordance with the methods described by Simonsen and Dyhre-Poulsen (1999).

Results
H-Reflex amplitudes were significantly smaller at SLR in the EH condition (p = 0.03). Background EMG of soleus and tibialis anterior and M-wave amplitudes did not change between LH and EH.

Discussion
This study showed reduced H-Reflex excitability during the SLR in drop-jumps from EH. Therefore, spinal adaptations seem to be involved in the control of the stretch reflex. However, changes in fusimotor drive cannot be excluded. A decreased excitability of Ia afferents at EH is thought to serve as a protection strategy, functionally to prevent the tendomuscular system from potential injuries due to the high load. There is evidence in the literature showing that the transmission of Ia afferents is modulated as a result of the actual specification of the respective movement (Faist and Pierrot-Deseilligny 1996). Several sources in modulating Ia afferent input are discussed: Thereby, presynaptic inhibition is argued to represent the most likely mechanism.

References


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