The effects of physical activity on muscle atrophy in cancer cachexia

Daniela Krooks och Malin Johansson
Examensarbete för kandidatexamen
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Handledare: Apostolos Theos
Idrottsmedicinska enheten
Institutionen för Samhällsmedicin och Rehabilitering vid Umeå Universitet
Abstract
Physical activity is, among other things, characterized by an increased quality of life and several physiological positive results such as hypertrophy and counteracting atrophy. Cachexia has been proven to degrade muscle proteins and is a contributing factor to death in cancer. Cachectic muscles are resistant to anabolic effects, and this knowledge combined with the many proven positive outcomes on muscle hypertrophy by exercise led this study to investigate the previous studies conducted on this subject further. The design was a descriptive review study and information was collected through 51 articles. The ubiquitin-proteasome system (UPS) plays a significant role in protein degradation, more specifically the E3 ubiquitin ligases MuRF-1 (muscle RING finger protein-1) and MaFbx (muscle atrophy F-box), which are FoxO (forkhead box-O) transcription factors. The UPS can be inhibited by substrates upregulated by physical activity, such as IGF-1 (Insulin-like Growth Factor-1) and PGC-1α (Peroxisome proliferator-activated receptor gamma coactivator 1-alpha). In conclusion, there are a lot of pathways in both cancer cachexia and physical activity that border on each other, but the molecular mechanisms are complex and not always clear.

Key words:
Cachexia, cancer, physical activity, muscle atrophy and protein degradation.