Raumenų-fascijų stiprinimas

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Skeletal muscle homeostasis and plasticity in youth and ageing: impact of nutrition and exercise

Mechanisms Modulating Skeletal Muscle Phenotype

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Figure 5  Summary of some conditions and factors able to induce skeletal muscle hypertrophy and atrophy. Overload: functional overload imposed by elimination of synergistic muscles; unloading: muscle unloading induced by hindlimb suspension or in conditions of microgravity.
Diagram of muscle anatomy:
- Bone
- Perimysium
- Blood vessel
- Endomysium
- Muscle fiber (cell)
- Epimysium
- Fascicle (wrapped by perimysium)
- Tendon
- Endomysium (between fibers)
Figure 3  Illustration of the changes in biceps brachii and brachialis moment arm lengths with increases in anatomical cross-sectional area. By doubling the anatomical cross-sectional area of the biceps brachii and brachialis, the moment arms of each increase by 27.2% and 37.3%, respectively.
FIGURE 2.16 Dissection of the gluteal region. Transverse adhesions between the superficial and deep fascia at the gluteal fold. At this level, the skin adheres to the deeper planes. As the skin is stretched, the tension is transmitted to the underlying muscles, thanks to the adhesion of the superficial fascia with the underlying planes.
Figure 5

(a) Passive stretch of muscle sarcomeres. As a sarcomere is stretched beyond its slack length, the proximal tandem Ig segments unfold approximately to their contour length (above). After the proximal tandem Ig segments have reached their contour length, further stretching extends the PEVK segment (below). Adapted from Granier & Labett (58). (b) Active stretch of muscle sarcomeres. Upon activation, N2A titin binds to actin (above). Only the PEVK segment (green) extends when active muscle is stretched (below), due to binding of N2A to thin filaments. Reproduced with permission from Nishikawa et al. (99). Copyright 2012, The Company of Biologists Ltd. Abbreviations: Ig, immunoglobulin; PEVK, proline-glutamate-valine-lysine.
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Nordic Hamstring
Flywheel

- Max force (decreasing with fatigue)
- Load
  - Conventional weights
  - Flywheel devices
- Force
  - Max force (decreasing with fatigue)
  - Reps
Naujausios Meta-Analizės
Jėgos pratimai stimuliuoja kepenyse riebalų deginimą – todėl tokie pratimai yra labai gera priemonė, išvalanti riebalus iš kepenų. Tris kartus per savaitę; 40-45 min per dieną; 12 savaičių. (Hashida et al. 2017).
Fizinio aktyvumo Medicina

FA (ištvermės ir jėgos) senjorams pagerina ne tik fizinę, bet ir psichinę sveikatą. Rezultatai reikšmingai matosi net po 12 savaičių. Tai priklauso nuo BDNF padidėjimo po FA (Byun and Kang, 2016).
Ištyrus 35754 žmones, nustatyta, kad jėgos fiziniai pratimai, nepriklausomai nuo aerobinės ištvermės krūvių, yra efektvi prevencinė priemonė prieš antrojo tipo diabetą ir daugybę ŠKS ligų (Shiroma et al. 2017).
Figure 5.
Muscle as an endocrine organ. Exercise triggers the secretion from muscle of numerous factors that impinge on systemic function. See text for details.
Myostatin

Myostatin (MSTN) is a member of the transforming growth factor β (TGF-β) superfamily that is expressed in the developing and adult skeletal muscle. The main function of myostatin is to negatively regulate muscle mass [58]. Evolutionarily, actively limiting muscle growth might have helped to prevent the build-up of energy-consuming muscle mass beyond the needs of the current situation. Accordingly, myostatin null mice exhibit a massive muscle hypertrophy that is characterized by an increased fiber cross-sectional area as well as an elevated number of fibers. The hyperplasia in this animal model most likely originates from accelerated primary and secondary myogenensis. Importantly, the myostatin gene is highly conserved among different vertebrate species. For example, myostatin mutations in some domestic breeds of cattle including the Piedmontese, Belgian Blue and Marchigiana result in a so called double-muscling phenotype and hence pronounced muscle hypertrophy [59-61]. Similar double muscling phenotypes have been observed in sheep and the whipped dog breed that leads to increased muscle mass and racing performance in the latter [62]. Finally, a mutation in the myostatin gene has also been associated with muscle hypertrophy in a male child with extraordinary muscularity and several relatives with self-reported unusual strength [63].
Interleukin-6 (IL-6) has originally been classified as a prototypical pro-inflammatory cytokine while later, anti-inflammatory properties have also been described [82]. Besides the production of IL-6 in activated immune cells, the systemic elevation of IL-6 in patients with metabolic diseases has contributed to the link of IL-6 and inflammation. Moreover, overexpression of IL-6 in transgenic mice results in reduced body mass and impaired insulin-stimulated glucose uptake by skeletal muscle [83]. Thus, IL-6 has been proposed as one of the pro-inflammatory factors that promote the development of peripheral insulin resistance.

Thus, IL-6 release in response to exercise seems to have pleiotropic effects by increasing glucose uptake and fatty acid oxidation locally in skeletal muscle and enhancing insulin secretion, which further increases glucose uptake into muscle cells. At the same time hepatic glucose output [99] and fatty acid release from adipose tissue [101] are stimulated thereby providing energy substrates for the exercising muscle.
Brain-derived neurotrophic factor (BDNF) is a member of the neurotrophin family, is strongly expressed in the brain [132] and to a lesser extent in skeletal muscle [133]. In the CNS, BDNF regulates neuronal development and modulates synaptic plasticity, playing a role in the regulation of survival, growth and maintenance of neurons [134, 135]. Furthermore, hypothalamic BDNF has been identified as a key factor in the control of body mass and energy homeostasis [136]. BDNF also influences learning and memory [137] and brain samples of patients with Alzheimer’s disease exhibit reduced expression of BDNF [138]. Similarly, BDNF serum levels of patients with depression, obesity and type 2 diabetes are decreased [139, 140]. Inversely, exercise increases circulating BDNF levels in humans [141] and recent studies suggest that the brain contributes 70-80% of the circulating BDNF in this context [142]. BDNF mRNA and protein levels are also increased in skeletal muscle in response to exercise and contribute to enhanced fat oxidation by activating AMPK [133]. However, muscle-derived BDNF seems not to be released into the circulation in significant amounts indicating that BDNF primarily acts in an auto- and/or paracrine manner.
Iirisin is cleaved off from FNDC5, a membrane-bound protein in skeletal muscle that is induced by exercise and muscle shivering [167]. Irisin exerts its action on white adipose tissue cells to stimulate UCP-1 expression and other brown fat-like genes thereby inducing browning and thermogenesis of white adipose tissue. Collectively, these effects lead to an increase in energy expenditure and result in an improvement of adiposity and glucose homeostasis [170]. Besides skeletal muscle, FNDC5 is also expressed in the brain [173, 174]. By elevating systemic irisin levels, endurance exercise induces FNDC5 expression in the hippocampus in a PGC-1α-dependent manner, which then leads to increased hippocampal BDNF expression and ultimately neurogenesis in this brain region. Accordingly, peripheral delivery of FNDC5 via adenoviral vectors is sufficient to induce BDNF expression in the brain [175]. Therefore, FNDC5/irisin might be the molecular mediator of exercise-induced neurogenesis in a direct skeletal muscle-brain cross-talk.
Stabilizatoriai ir šešios fascijų grandys
>30 procentų jėgos ir galingumo judesio metu generuoja fascijos