Methods: Complete Freud's adjuvant-induced arthritis (CFA) was used as a mouse model of arthritis. CFA was injected in the ankle joints of C57BL/6JRj and MCK-PGC-1α1 transgenic mice. After 2 weeks of inoculation, mitochondrial respiration and Ca^{2+} handling was evaluated by high-resolution respirometry and live cell imaging, respectively. Muscle mitochondrial ultrastructure was analyzed by electron microscopy, while mitochondrial-related protein expression was accessed by immunoblotting.

Results: The CFA treatment significantly reduces mitochondria oxidative capacity (22%, $p < 0.01$). This reduction was accompanied by a decrease in mitochondria number and size (≈40%, $p < 0.01$), and a pronounced decrease in the expression of subunits of mitochondrial complexes (63% for the CI and 38% for complex II and III, $p < 0.01$). A decreased mitochondrial Ca^{2+} uptake (60%, $p < 0.001$) following a fatigue protocol and increased mitochondria-sarcoplasmic reticulum distance (~29%, $p < 0.01$) were also observed in WT-CFA mice. The mitochondrial impairment induced by CFA is reverted in the MCK-PGC-1α1 transgenic mice.

Conclusion: Our data show that arthritis induced impaired mitochondria function in the muscle. Interestingly, the overexpression of PGC-1α1 in transgenic mice was able to mitigate these effects.

The effects of melatonin on exercise performance and cognitive functions in rats with overtraining syndrome

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Abstract

Background: Overtraining syndrome (OTS) occurs with inadequate recovery periods and leads to many symptoms, including poor performance. Melatonin is an anti-oxidant hormone. We aimed to evaluate possible protective effects of melatonin on OTS.

Methods: Male Sprague Dawley rats ($n = 26$) were divided into sedentary, moderate intensity exercise (MIE), overtraining syndrome (OTS), and those with melatonin added to exercise (MEL;15 mg/kg). On the treadmill, MIE were subjected to 7, OTS 9 weeks training protocols. Then, the exercise performance, cognitive functions, depression like behavior and anxiety levels were evaluated. Levels of malondialdehyde, glutathione and myeloperoxidase, catalase, superoxide dismutase activity and histological analysis were done in brain and gastrocnemius samples. Statistical analyses were done by one-way-ANOVA.

Results: Exercise performance was reduced with OTS while MIE or MEL increased performance ($p < 0.05$). Spatial memory was increased in MIE + MEL group, and working memory increased in OTS + MEL group ($p < 0.05$–0.01). Freezing time was increased with OTS and decreased with MEL ($p < 0.05$). With both exercises, the immobilization time was increased and decreased back with MEL ($p < 0.05$–0.001). Brain malondialdehyde was increased in OTS, and decreased with MEL ($p < 0.05$–0.01). Muscle malondialdehyde levels decreased with OTS and were declined more with MEL ($p < 0.05$). OTS in muscle increased glutathione and superoxide dismutase activity, MIE increased catalase levels ($p < 0.05$–0.001). While brain myeloperoxidase levels increased with OTS ($p < 0.05$), decreased with both exercises and MEL ($p < 0.01$–0.001). Increased neuron damage in OTS was improved with MEL.

Conclusion: Our findings suggest that melatonin may improve cognitive functions and exercise performance by limiting oxidant damage and inflammation.

Effect of moderate physical exercise on body composition, physical fitness and serum cytokines levels

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Abstract

Introduction: As more people lead sedentary lifestyle the question is rising – if starting moderate physical activity would ease the problems associated with decreased physical activity.

Aim: To assess moderate physical activity effect on overall muscle strength and wellbeing.

Objectives: (1) Select physically healthy women in their 20–30 who report themselves as lacking physical activity. (2) Evaluate participant physical fitness entering the research. (3) Instruct them to do mild – moderate exercise for 2 months. (4) Evaluate results upon repeated measurements.

Methods: Physical fitness was tested by palm grip test and spirometry and body composition was assessed using bioimpedance method. Serum levels of interleukin 6 (Il-6), fibroblast growth factor 23 (FGF-23) and kidney injury molecule 1 (KIM1) were measured. Participants received instructions for physical activities that they had to do for

2 months until the next meeting. Activities were chosen as mild to moderate and included collecting 10 000 steps daily and an exercise for upper body with added 0.5 kg weight. After 2 months of exercising all measurements were repeated.

Results: 22 participants were selected and divided in 3 groups according to their body mass index (BMI). After 2 months of exercise there was no significant change in spirography data and muscle mass in any of the groups, but slight changes could be observed in muscle strength and cytokines.

Conclusions: Mild to moderate physical exercise may not be enough to increase muscle mass but may play positive impact on overall wellbeing.

Creatine deficiency causes muscle-specific shift in the myosin heavy chain composition

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Abstract

The creatine kinase (CK) system is one of the most important energy transport systems in skeletal muscle, whose function relies on the maintenance of an adequate phosphorylation potential near ATPases. CK allows the fast transfer of a phosphate group from phosphocreatine (PCr) to ADP to resynthesize ATP, and the opposite reaction by transferring a phosphate from ATP to creatine. During contraction, muscles undergo important changes in energy availability, and the CK system is the main rebalancing system. Studies of the consequences of compromised CK function in skeletal muscle have been mainly carried out in $CK^{-/-}$ models exhibiting moderate alterations. However, creatine-deficient mice, especially mice deficient in L-arginine: glycine amidinotransferase ($AGAT^{-/-}$), show greater muscle atrophy than their $CK^{-/-}$ counterparts. Our laboratory previously highlighted a massive metabolic shift in a muscle dependent-manner in $AGAT^{-/-}$ mice. Here, we show that the total lack of creatine also leads to a dramatic shift in the myosin heavy chain (MHC) composition in a muscle dependent-manner. This remodeling of the muscle phenotype involves a chronic activation of the energy sensor AMP-activated kinase (AMPK), which has been suggested as an important player in the muscle phenotypic shift. Additionally, we investigated a potential signaling pathway that could explain this chronic activation of AMPK. Overall, the lack of creatine seems to have severe consequences in a muscle dependent-manner, affecting mainly glycolytic-based muscles, and causing remodeling of their metabolism and their MHC composition towards an oxidative profile.

Exploring structural and functional signatures in respiratory muscles induced by chronic exercise training: Insights for promoting respiratory health

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Abstract

While the role of chronic exercise in musculoskeletal remodeling is well-established, the extent to which it affects the respiratory system is not well-understood. Thus, this study aimed to investigate the effects of different long-term exercise exposures on respiratory functions and diaphragm morphology. To achieve this goal, we conducted a cross-sectional comparison involving sixteen high-level cyclists (CYC), sixteen professional basketball-players (BPL), and fifteen untrained subjects (CON). The testing involved assessing lung function, strength of inspiratory and expiratory muscles, and B-mode ultrasonography of diaphragm muscle. CYC demonstrated the highest values for lungs capacity, followed by BPL, with both athlete groups displaying significantly higher values compared to CON ($p < 0.05$). BPL exhibited the strongest respiratory muscles, while CYC showed the second-highest values, both significantly higher than CON ($p < 0.05$). Additionally, BPL had greater diaphragm thickness at rest and at the end of inspiration compared to CON and CYC ($p < 0.05$). Consequently, both BPL and CYC demonstrated greater diaphragm thickening fractions during tidal and maximal inspiratory breaths ($p < 0.05$). Chronic exercise training induces favorable adaptations in respiratory muscles, as evidenced by improved respiratory function and diaphragm morphology in CYC and BPL compared to CON. Moreover, the specificities of each exercise training, appear to influence the specific adaptations observed in both, structural and functional characteristics of the respiratory muscles. These findings underscore the importance of regular exercise, alongside targeted respiratory muscle training, in promoting respiratory health and optimizing performance.