29.17

A SINGLE BOUT OF CONDITIONING PROTECTS MOUSE SKELETAL MUSCLE FROM CONTRACTION-INDUCED INJURY

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Prior injury via pliometric contractions (those during which muscle is stretched) protects muscle from injury during subsequent pliometric contractions. The purposes of this study were to determine whether, in our in situ mouse extensor digitorum longus model, (1) a single bout of pliometric contractions protects muscle from injury during a subsequent bout and (2) passive stretch or isometric contractions also induce protection. An initial bout of 75 pliometric contractions, of 20% strain relative to optimal fiber length, induced a 55% force deficit three days after the bout. Two weeks after the initial bout, a second identical bout of pliometric contractions produced a significantly smaller ($p<0.05$) 19% force deficit at three days. Thus, the initial bout protected muscle from injury during the second bout. Six weeks after the initial bout, a second bout produced a force deficit not significantly different from that for the initial bout, suggesting that protection had been lost. A bout of 75 passive stretches (20% strain) did not result in a force deficit at three days, but did provide some protection from injury following 75 pliometric contractions performed two weeks after the passive stretches (36% force deficit, $p<0.05$). A bout of 75 isometric contractions also did not result in a force deficit at three days, but showed a trend of protection from injury following pliometric contractions performed after two weeks (40% force deficit, $p = 0.09$). The data suggest that neither pliometric contractions nor degeneration/regeneration are required for exercise-induced protection from pliometric contraction-induced injury.

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29.18

FORCE DEFICITS AFTER ACTIVE STRETCHES OF RAT SKELETAL MUSCLES WITH REDUCED COLLAGEN CROSS-LINKS

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Sprague Dawley rats (♀, $n = 6$, age 87 days) were injected 2x daily for 43 days with β-aminopropionitrile (BAPN, 333 $mg \cdot kg^{-1} \cdot day^{-1}$ i.p.) which inhibits the enzyme, lysyl oxidase, responsible for collagen cross-links. Age-matched saline-injected rats served as controls (C, $n = 6$). Isometric forces before dorsiflexion ($a = 3000° \cdot s^{-2}$, ankle position 90° to 40°) with repeated stretches (20) of active plantar flexor muscles at 80 Hz (contraction time 1.1 s, rest periods 3 min) were measured. Isometric forces were measured at 90°, before and 1 hr after the stretches at 5, 10, 20, 40, 60 and 80 Hz. Relative weights of soleus, plantaris and gastrocnemius muscles were not different between groups. Significant reductions in collagen cross-links, pyridinoline, (mol/mol collagen) were found in tendon (22.9%), plantaris (17.1%), and soleus (7.4%) with no changes in collagen content (hydroxyproline) as determined by HPLC. In gastrocnemius medialis, pyridinoline cross-links were 4.4% lower but this did not pass significance ($P = 0.2$). Groups had similar isometric forces at 5, 10, 20, 40, 60 and 80 Hz before the stretches. Isometric force deficits that developed followed similar courses for BAPN- and C-rats. Before the last stretch, deficits were 51.1 ± 2.4% (C) and 54.7 ± 4.6% (BAPN). After 1 hr of rest following the stretches, deficits were similar at 80, 60, 40 and 20 Hz but were 26% and 29% larger at 10 and 5 Hz in BAPN-rats ($P < 0.05$). Isometric force deficits after active stretches of rat plantar flexor muscles with reduced collagen cross-links, pyridinoline, were larger only at low stimulation frequencies. Supported by NIOSH R01-OHAR-02918.

29.19

MUSCLE DYSFUNCTION OCCURS RAPIDLY AFTER RAT HINDLIMB SUSPENSION.

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The purpose of this study was to determine the time course of muscle dysfunction and recovery that occurs in muscles experiencing modified mechanical loading. If a loss of muscle function appears before the well-characterized invasion of inflammatory cells, it would presumably indicate that mechanical loading promotes muscle injury during the early period of increased muscle use. To induce muscle injury and inflammation, rats were suspended for 10d followed by periods of reloading of 0h, 2h, 1d, 3d, 7d or 28d under normal cage activity. Contractile and immunohistochemical measurements were performed on soleus muscles. Muscle function was assessed by measuring maximal isometric specific force (force/cross-sectional area; Po). A significant 35% decrease in Po relative to ambulatory animals was observed following the suspension period. A second drop in Po was recorded after only 2h of reloading when muscles produced only 29% of the Po for ambulatory rats. The deficit in force production was still 38% after 7d of reloading and disappeared by 28d. The contribution of E-C coupling failure to the decrement in Po were also evaluated by incubating soleus muscle in 50 mM caffeine. Results showed that muscle dysfunction and E-C coupling failure are occurring earlier than the well documented time course of inflammatory cell invasion which supports the hypothesis that mechanical stress plays a more important role than inflammation in muscle force decrement following hindlimb suspension Work supported by Fonds de la Recherche en Santé du Québec (FRSQ).

29.20

RADIAL AND LONGITUDINAL STIFFNESS OF SKELETAL MUSCLE FIBERS FOLLOWING UNLOADING.

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Radial and longitudinal stiffness of slow-twitch soleus muscle (SOL) fibers of rats following unloading by hindlimb suspension (HS) were investigated. Rats were divided into 2 groups: the control group and the HS group subjected to 14 days of HS. After HS, SOL was immediately removed from rats. Chemically skinned single muscle fibers were prepared from each muscle tissue. The radial stiffness was evaluated by the relationship between the osmotic pressure and the lattice spacing using an electronmicroscopy in relaxed and rigor states. The osmotic pressure was controlled by the changes in the concentration of Dextran (M.W. 200,000-400,000; 0-25 %). The longitudinal stiffness was evaluated by the relationship between the sarcomere length and resting tension in relaxed state. In relaxed state, the effects of unloading on the radial stiffness were not observed. In rigor state, however, the radial stiffness significantly depressed following HS ($p<0.05$). The longitudinal stiffness significantly decreased following HS ($p<0.01$). Therefore, there may be any changes in the formation of cross-bridges and connectin (titin) filaments following unloading.

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29.21

INTERLEUKIN-6 EXPRESSION IN RESPONSE TO TWO MUSCLE INJURY-INDUCING EXERCISE BOUTS

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The severity of exercise-induced muscle injury and decreased muscle strength is normally lessened with repeated exercise, suggesting an adaptive response that may be mediated by the pro-inflammatory cytokine interleukin-6 (IL-6). The purpose of this study was to: 1) determine the expression of IL-6 mRNA, IL-6, and creatine kinase (CK) in response to eccentric exercise that resulted in injury and decreased strength to the knee extensor muscles, and 2) compare the possible adaptive response in muscle function to IL-6 expression occuring in response to two exercise bouts. Seven untrained males (20.54 ±2.37 years of age) were subjected to two exercise bouts (separated by three weeks) consisting of 7 sets of 10 repetitions at 150% of the one-repetition maximum (1-RM) employing forced-lengthening contractions of the knee extensors of the dominant leg. Blood samples were taken pre- and post-exercise (15 min and 2, 4, 6, 24, 48, and 96 hr after). In addition, 1-RM strength, maximum isometric force (MIF), and muscle soreness ratings were assessed 24, 48, and 96 hr post-exercise. Percent changes from pre-exercise for each post-exercise time point were analyzed with a 2x8 ANOVA ($p < 0.05$). Results revealed that peak IL-6 mRNA expression occurred at 2 hr post-exercise ($p < 0.05$); although, the differences between bouts (bout 1=186%, bout 2=190%) were not significant. However, the peaks for both IL-6 (bout 1=273%, bout 2=259%) and CK (bout 1=244%, bout 2=196%) occurred at 6 hr post-exercise with the differences between bouts only being significant for CK ($p < 0.05$). Peak changes in muscle soreness and decrements in 1-RM strength (bout 1=-29.85, bout 2=-7.91) and MIF (bout 1=-25.99, bout 2=-5.79) occurred at 24 hr post-exercise with the differences between bouts being significantly different. Results of this study indicate that repeated bouts of exercise which induce muscle injury: 1) increase the expression of both IL-6 mRNA and IL-6 upon repeated exercise bouts, 2) have an adaptive response on muscle function compard to the initial exercise bout, and 3) suggest that the adaptive response in muscle function may be a result of the increased expression of IL-6.

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29.22

ADAPTATION TO ECCENTRIC EXERCISE DOES NOT INVOLVE A NEURAL COMPONENT BUT IS INFLAMMATION-DEPENDENT.

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Skeletal muscle can be damaged by eccentric contractions. Interestingly, a single bout of eccentric exercise can provide protection against subsequent damaging contractions. The basis of this adaptation known as the repeated bout effect (RBE) is unclear but hypotheses based on cellular, neuronal and connective tissue response have been proposed. We tested the hypothesis that the RBE does not rely on neural adaptation but rather requires an inflammatory process. Rat extensor digitorum longus muscles were submitted to two bouts of in situ eccentric contractions performed 14d apart. Maximum tetanic tension (Po) was recorded in vitro 2d after each bout. A non-steroidal antiinflammatory drug, diclofenac or placebo was given for 2 or 7d after the first bout (NSAID and ECC groups). Compared to its sham, a 35% decrement in Po was seen 2d after the first bout for the ECC group. The second bout did not induce any damage as absolute and normalized Po (N/cm^2) 2d afterward were not significantly different from the pre-second bout values. When treated with NSAID for 2d, no deficit was seen after the second bout for absolute Po but normalized Po decreased significantly by 11%. When treated for 7d the second bout produced highly significant deficits reaching ~50% of the one for bout 1 suggesting that the adaptive response was strongly impaired. We conclude that the RBE can be seen in a model where neural recruitment strategies are bypassed and that processes associated with the inflammatory response are necessary for this adaptation.

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