protein oxidation, protein degradation, DNA fragmentation and DNA mutation in skeletal muscle. Performance is mainly dependent on the intensity of these changes.

PURPOSE: In the present study, the effect of an aerobic exercise training on expression of antioxidant enzymes (Cu,Zn-superoxide dismutase-SOD, Mn-SOD, catalase and gluthatione peroxidase) and heat shock proteins (HSP 60 and 70) in rat soleus muscle was investigated. **METHODS**: Adult male rats were trained at 65% of VO_{2max} for 5 weeks, 1 hour per day, 7 days per week. After the last session of exercise, the soleus muscle was removed and frozen in liquid nitrogen for RNA extraction and RT-PCR analysis. **RESULTS**: The exercise program imposed increased the expression of catalase (33%), Cu,Zn-SOD (83%), Mn-SOD (127%), HSP 60 (149%) and HSP 70 (200%) as shown in Table 1.

Table 1: Expression of antioxidant enzymes and heat shock proteins (HSP). The values are expressed as arbitrary units in relation to G3PDH mRNA.

Genes	Arbitrary units (mean ± SEM) of 6 determinations	
	Control group	Trained group
catalase	0.70 ± 0.07	0.93 ± 0.04*
Cu,Zn-SOD	0.59 ± 0.06	1.08 ± 0.04*
Mn-SOD	0.41 ± 0.16	0.93 ± 0.10*
gluthatione peroxidase	0.91 ± 0.09	1.01 ± 0.07
HSP 60	0.35 ± 0.10	0.87 ± 0.09*
HSP 70	0.29 ± 0.07	0.87 ± 0.10*

* $p \leq 0.05$ as compared with control rats.

CONCLUSION: The increase in expression of antioxidant enzymes and HSP may play an important role to protect the skeletal muscle from oxidative stress induced by exercise.

Supported by CAPES, CNPq and FAPESP.

1648 *Board #103* **9:30 AM - 11:00 AM**

Ultrasound And Cryotherapy Combined Therapy To Control Delayed Onset Muscle Soreness

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Muscle injuries are, after the ligament sprains, the most frequent injury of soft tissues in athletes. The study of the Delayed Onset Muscle Soreness (DOMS) from an Eccentric Exercise Protocol (EEP) has been proved as a valuable experimental model, where the muscular damage is comparable to a clinical muscular injury. The main characteristics of DOMS are soreness, muscle injury and functional impairment, where the effects of therapeutic Ultrasound (US) and cryotherapy (CRY) are normally oriented.

PURPOSE: To determine if the combined therapy of US and CRY, after an EEP, is able to control the DOMS.

METHODS: Thirty seven healthy men and women were recruited for a simple-blind, placebo-controlled trial. Parameters as difference with the baseline Pain (dVAS) with a 20 grs/mm compression, mid arm relaxed girth (dMAG), maximal isometric voluntary contraction (dMIVC), and rest elbow angle (dREA) and Total Plasma Creatine Kinase activity (CK) were measured 72 hours before, immediately after, and 24, 48, 72, 96 and 168 hours after a 80% MIVC nondominant elbow flexors eccentric-exercise protocol. Subjects were randomly assigned in one of the follow groups: Control-Control (CON-CON) without EEP and any treatment (n=8), Control-Exercise (CON-EXC) with EEP and no kind of treatment (n=10), US sham+CRY (USC-S) with EEP and placebo US treatment plus CRY (n=10) and US+CRY (USC) with EEP and US treatment plus CRY. The CRY was a 20 minutes cold pack protocol applied immediately after, 12, 24, 36, 60, 84 and 108 hours after the EEP; while the US was applied by 10 minutes, 36, 60, 84 and 108 hours after the EEP, with a 0,04-0.16 W/cm² variable SATA.

RESULTS: No statistical differences ($p>0.05$) were observed for any studied parameter at the within group CON-CON analysis, confirming the statistical baseline. CK levels were lower in USC group compared to CON-EXC group at 48 hours after EEP (356.73 ± 436.93 v/s 1402.80 ± 1491.81 U/L, $p<0.05$) and higher in USC-S group compared to CON-CON group at 96 and 168 hours after EEP (1801.88 ± 1316.70 v/s 185.71 ± 132.78 U/L, and 1449.88 ± 1038.73 v/s 131.63 ± 57.22 U/L, $p<0.05$, respectively), without statistical significances between USC and CON-CON groups any time. At the dMAG, USC-S group were lower than USC group at 24 and 48 hours after EEP (-0.08 ± 0.39 v/s 0.54 ± 0.76 cms, and 0.00 ± 0.44 v/s 0.62 ± 0.72 cms, $p<0.05$, respectively). At the dREA, USC group were lower than USC-S group at 48 hours after EEP (-25.00 ± 9.79 v/s -43.38 ± 10.65, $p<0.05$).

CONCLUSIONS: These results showed that the therapeutic US itself and combined with CRY may be considered as one answer for the treatment for acute muscle injuries, more studies must be done to determine the exact US and CRY doses.

1649 *Board #104* **9:30 AM - 11:00 AM**

Effects Of Skeletal Muscle Specific Fkbp12 Deficiency On Eccentric Contraction-induced Injury In Mouse Muscle

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Central Core Disease (CCD) is an inheritable skeletal muscle disease that results in weakness and motor development delays. Although CCD is known to stem from mutations in the ryanodine receptor (RYR1), the exact etiology of CCD is unknown. **PURPOSE**: To test the hypothesis that removal of FKBP12, a 12-kD binding protein known to bind to RYR1 sites near the locus of some CCD mutations, would induce central core lesions and exacerbate strength deficits in mouse anterior crural muscles after eccentrically biased exercise. **METHODS**: Skeletal muscle specific FKBP12 deficient mice were created using the CreLoxP gene recombination technique with Cre transgene expression under the regulation of the muscle creatine kinase promoter. Anterior crural muscle strength was measured in FKBP12 deficient (n=11) and control (n=11) mice before, immediately after, and at 3, and 14 d after performance of 150 eccentric contractions in vivo. Isometric force and caffeine sensitivity (1-50 mM) were measured in EDL muscles in vitro. Histological analyses were used to characterize muscle damage and central core lesions in TA muscles. **RESULTS**: Body weight (BW) was 8% lower in FKBP12 deficient mice compared with controls (i.e., Cre, wild-type). Before injury there were no differences between groups in isometric torque as a function of stimulation frequency (20-400 Hz) except at 20 Hz, with peak isometric torque normalized to BW being 15% lower in FKBP12 deficient mice. There was no difference in peak torque normalized to BW on the first eccentric contraction between FKBP12 deficient (204.1±3.5 Nmm/kg) and control (202.0±3.3 Nmm/kg) mice. Relative decreases (43.0±0.9%) in peak eccentric torque over the 150 contractions were similar for both groups. There were no differences in isometric torque deficits between groups immediately after, or at 3 and 14 d after injury. Although peak isometric specific force in the EDL muscle was not different between groups, FKBP12 deficient mice exhibited a greater (20%) response to 50 mM caffeine at 14 d. TA muscles from both groups exhibited cellular damage, however, no evidence of central core lesions was found. **CONCLUSIONS**: Skeletal muscle FKBP12 deficiency does not exacerbate strength deficits or induce central core lesions after eccentric contraction-induced injury in mouse anterior crural muscle.

Supported by NIH grant 2 R01 AR41802-11.

1650 *Board #105* **11:00 AM - 12:30 PM**

Effect Of Chronic Exposure To Stretch-shortening Cycles On Apoptotic Markers In Skeletal Muscle Of Aged Rats

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We have observed that chronic exposure to stretch shortening cycles (SSC) exhibits hypertrophy and increased muscular strength in young adult rats but decreased strength and maladaptation in aged rats. This indicates that recovery from SSC is impaired with aging. **PURPOSE:** The objective of this pilot study was to examine if apoptosis is involved in this impaired muscle adaptation during chronic exposure to SSC in aged rats. **METHODS:** Left dorsiflexor muscles of 30 mo old Fisher Brown Norway rats (bodyweight 338 ± 32 g, *N*=5) were exposed *in vivo* to SSC under anesthetization 3 days per week over a 4.5 week period. The right dorsiflexor muscle served as an intra-animal control. Each exposure consisted of 8 sets of 10 SSC with two minutes rest between sets. Tibialis anterior muscles of both limbs were then dissected. Protein content of heat shock protein (HSP) 72, apoptosis inducing factor (AIF), and cytoplasmic superoxide dismutase (CuZnSOD) was determined in the extracted cytosolic protein fraction by Western immunoblot. Apoptotic DNA fragmentation and total cytosolic cytochrome c level was assessed by ELISA. Moreover, a spectrofluorometric enzyme assay was used to measure the protease activity of caspase-3 and -9. **RESULTS:** We did not find difference in the protein content of HSP72, AIF, and CuZnSOD between the SSC and control muscles ($P>0.05$). Caspase-9 enzyme activity was similar in the SSC and control samples ($P>0.05$). However, the level of apoptotic DNA fragmentation, cytochrome c, and caspases-3 protease activity tended to increase by 11% ($P=0.056$), 30% ($P=0.061$), and 39% ($P=0.088$) in the SSC muscle when compared to contralateral control muscle, respectively. **CONCLUSION:** These pilot data are consistent with the hypothesis that apoptosis may be involved in the maladaptation of aged skeletal muscle during chronic SCC stimulation. Nevertheless, our results are limited by the sample size and further investigation is warranted to confirm our findings with inclusion of a larger number of animals.

Supported by grants from NIH R01AG021530 and NIOSH.

1651 *Board #106* **9:30 AM - 11:00 AM**

Effects Of Isometric And Eccentric Contractions On Apoptosis Of Skeletal Muscle In Male And Female Rats

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PURPOSE: It is well known that the eccentric muscle contraction (ECC) lead to more severe histological muscle damage than isometric muscle contraction (ISO). Recent studies indicated that exercise stimulates the expression of proteins related in apoptosis and increase apoptosis myonuclei. The purpose of this study was to examine the effect

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