

EXERCISE-INDUCED MECHANICAL HYPOALGESIA IN
MUSCULOTENDINOUS TISSUES OF THE LATERAL ELBOW

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ABSTRACT

Aim: To investigate mechanical sensitivity responses at the lateral elbow to repeated weekly bouts of low load exercise in healthy subjects.

Methods: Thirteen young men (n=6) and women participated in 4 weeks of exercise. Arms were randomly allocated to an eccentric-only exercise protocol (ECC: 5 sets of 20 contractions) or to a concentric-eccentric protocol (CON-ECC: 5 sets of 10 eccentric/10 concentric contractions) performed at 30% maximal wrist extension force. Arms were exercised consecutively within each supervised weekly session. Quantitative measures of pressure pain threshold (PPT) recorded at three sites and maximal force for grip and wrist extension were assessed at baseline, and immediately pre/post exercise at each session. Muscle endurance during 100 maximal grip contractions force was assessed at baseline and one week following the final exercise session.

Results: Regardless of protocol, repeated low load exercise resulted in a time-dependent increase in PPT at all sites post exercise Weeks 3 and 4 and persisting at follow up Week 5 ($P < 0.02$). No significant difference between protocols was evident for any measure. Muscle force and endurance were not significantly augmented compared with baseline.

Conclusion: Mechanical hypoalgesia is induced by repeated low load exercises regardless of exercise mode, and this may prove beneficial if replicated clinically.

INTRODUCTION

Changes in mechanical sensitivity can occur in response to acute resistance exercises in healthy subjects (Koltyn and Arbogast, 1998; Kosek and Lundberg 2003; Marqueste et al. 2004; Slater et al. 2005; Staud et al. 2005). Acute exercise typically results in an immediate exercise-induced hypoalgesia at sites local to the exercised muscles (O'Leary et al. 2007) or at sites both local and remote from the exercised muscles (Staud et al. 2005). Mechanical deep tissue sensitivity changes following acute exercise probably involve multiple interacting peripheral and central pain-modulatory mechanisms (Gibson et al. 2006; Koltyn and Arbogast, 1998; Kosek and Lundberg 2003; Marqueste et al. 2004; Staud et al. 2005). Whether persistent changes in mechanical deep tissue sensitivity occur in response to repeated bouts of exercise loading is unclear, although mechanical tissue adaptations do occur with resultant net collagen synthesis, decreased stress-susceptibility and improved load-resistance of musculotendinous units (Kjaer et al. 2006). Both the degree of load and frequency appear important factors with greater susceptibility to tendinopathies with repeated microtrauma (Wang 2006) or if tendons are stress-shielded against functional cyclical repeat loading (Arnoczky et al. 2007) or subject to repeated high loads (Langberg et al. 1999)

Mechanical loading using exercise is clinically recommended for musculoskeletal health and recovery from tendinopathies. Currently both eccentric and concentric loading is advocated, although systematic review concludes insufficient evidence in favour of eccentric over concentric in lateral elbow rehabilitation (Woodley et al. 2007). Eccentric loading has been recommended specifically in the rehabilitation of

tendinopathies (Alfredson et al. 1998; Ohberg et al. 2004; Knobloch et al. 2007) including lateral elbow tendinopathy (Svernlov and Adolfsson 2001) and combined concentric-eccentric exercise has also been described (Martinez-Silvestrini et al. 2005), both protocols associated with reduced levels of pain (Stasinopoulos and Stasinopoulos 2004). One potentially beneficial effect of performing unaccustomed eccentric exercise compared with concentric or isometric exercises, is attenuation of induced muscle stiffness, soreness and force loss when the same eccentric exercise is repeated (Clarkson et al. 1992). This adaptive or protective effect conferred by a single bout of eccentric exercise is often referred to as the 'repeated bout' effect (Clarkson et al. 1992) and occurs even at low load (Lavender and Nosaka 2006). Whether the attenuated responses described above are also associated with a persistent reduction in mechanical deep tissue sensitivity is unknown, however such a protective effect might be clinically important, as increasing mechanical load too rapidly in musculotendinous conditions such as tendinopathies can result in increased pain and reduced function.

Therefore, the aim of this study using healthy subjects was to compare the effects on deep tissue sensitivity and muscle function in the wrist extensors of two types of repeated low load eccentric exercise. Given that both protocols involved an eccentric exercise component, it was hypothesised that mechanical deep tissue sensitivity and specific muscle function responses to repeated low load exercise would be similar and that both repeated low load exercise protocols would alter deep tissue sensitivity at the lateral elbow but not muscle function.

METHODS

Subjects

A within-group repeated-measures experimental design was used with thirteen healthy subjects (six men, seven women) aged 18-35 (mean \pm SD 27.1 ± 1.4) years. The number of subjects was determined by a difference of 15% for pressure pain thresholds (PPT) and muscle function parameters for interventions with means and standard deviations drawn from a previous study using similar measures following eccentric exercise of wrist extensors (Slater et al. 2005). For this within-group design, a sample size of 12 was required to achieve power of 0.80 with alpha at 0.05. Mean (\pm SD) height and weight were 169.6 (\pm 2.3) cm and 67.7 (\pm 3.3) kg, respectively. Eleven subjects were right hand dominant. Exclusion criteria included a history of upper limb fractures, surgery of the upper limb, musculoskeletal disorders of the shoulder, wrist or hands, any neurological or muscle disorders, use of any form of medication at present or on an ongoing basis and prior training of the forearm muscles. Participants were instructed to continue their daily routine but avoid any form of upper extremity resistance training. This study was approved by Curtin University of Technology Human Research Ethics Committee. Written informed consent was gained from subjects prior to participation in the study.

Study Design

Both arms were exercised, with subject's arms randomly assigned into the eccentric protocol (ECC) or the combined concentric-eccentric (CON-ECC) exercise protocol. Subjects participated in six sessions over six weeks. Reliability of a gripping fatigue test was established at Week 0 and 7 days later at Week 1 prior to commencement of the

exercise bouts. At Week 1, immediately following the fatigue test, subjects commenced the first of four bouts of weekly low load ECC or CON-ECC exercise. At Week 5, repeat testing of all Week 1 pre-fatigue measures was undertaken (no ECC or CON-ECC exercise) (Figure 1). Baseline was defined as Week 1, pre-fatigue testing.

Exercise

Subjects performed two different exercise protocols, one on each arm in a counterbalanced fashion. Subjects were required to perform both ECC and CON-ECC exercise at the same session each week and the parameters for each protocol are fully detailed in Figure 1 (legend). The exercise mode allocated to each arm remained the same throughout testing, while the order of arms exercised was alternated at each session. Based on pilot data, 30% maximal voluntary contraction (MVC) isometric wrist extension, measured at 20° wrist extension was chosen for the physiologic load. Low load was predicted to avoid the tissue sensitizing effects associated with induced muscle soreness following eccentric exercise (Slater et al. 2005). Pilot data was consistent with no induced muscle damage. Exercise was performed using a free weight with the weight kept constant throughout the exercise period. For standardisation purposes, subjects were positioned in sitting with the elbow flexed (30°) and the forearm pronated and stabilised on an adjustable forearm rest.

Deep tissue sensitivity

Pressure pain thresholds (PPT) have been established as a reliable measure of deep tissue sensitivity (Rolke et al. 2005) and have been extensively documented in tennis elbow studies (Vicenzino et al. 2001; Slater et al. 2003; Bisset et al. 2006). PPTs were

recorded using an electronic algometer (Somedic AB, Sweden) with a stimulation area of 1.0cm² and application rate of 50kPa/s until the subject detected the pain threshold. PPT was defined as the point at which the sensation of pressure changed to a sensation of pain. The PPT was measured at three sites: the common extensor origin at the lateral epicondyle (CEO), the musculo-tendinous junction of extensor carpi radialis brevis (ECRB) muscle and the radial head laterally which acted as a control site as previously described (Slater et al. 2005). Three measures were taken at each site, with a thirty seconds interval between measures. The mean of the three trials was recorded as the PPT.

Pain and muscle soreness

Subjects maintained a self-report diary to record any muscle soreness experienced in the exercised arms for 72 hours following each exercise session. Measures of at-rest and movement-related pain were assessed using a visual analogue scale (VAS), consisting of a 10cm line marked at one end as “no pain” and at the other as “worst pain imaginable”. For muscle soreness, subjects completed a modified Likert scale of muscle soreness (Slater et al. 2003) for the upper limb, with 0 defining no soreness and 6 indicating a severe degree of muscle soreness.

Maximal grip force (MGF)

MGF was measured with an electronic digital dynamometer (MIE Medical Research Ltd., Leeds, UK). Subjects were positioned in supine with the arm supported in elbow extension and forearm pronation. Subjects were asked to exert maximal isometric grip

force (kg), which was recorded as the mean of three trials with an interval of 5 seconds between trials (Slater et al. 2005).

Maximal wrist extension force (MWEF)

MWEF was recorded via a force gauge (AFG, range 0-500N, Mecmesin Ltd., England). The force gauge was mounted on a specially designed platform, positioned on a table beside the plinth. The height of the hand attachment and force transducer was kept constant for each subject but could be varied to accommodate various hand sizes. Subjects were positioned in supine with the arm supported in elbow extension, forearm pronation and approximately 20° wrist extension. MWEF (kg) was recorded as the mean of three repeated measures with a 5 seconds interval (Slater et al. 2003).

Muscle endurance

A fatigue test for the wrist extensors was modified from a study in which knee extensor fatigue was investigated (Mouraux et al. 2000). The modified fatigue test comprised 100 maximal isometric gripping actions of 3 seconds duration (10 sets of 10 maximal grips with a 30 seconds rest interval between sets) using the same equipment and testing position as used in the assessment of MGF (kg). A metronome was used to time the procedure. To demonstrate the reliability of the fatigue test, all subjects performed the fatigue test at Week 0 and 7 days later at Week 1 on both arms. Muscle fatigue was calculated using the following fatigue index formula: $100 - ((\text{last 5 repetitions} / \text{first 5 repetitions}) \times 100)$ (Mouraux et al. 2000).

Statistical analysis

Data was analysed using SPSS 17.00 software package. To establish reliability of test measures, subjects undertook repeat measures of all variables for both arms at Week 0 (fatigue test only, no ECC or CON-ECC exercise) and at Week 1 (Figure 1). Measures of PPT, MGF, MWEF and fatigue were found to have high reliability by $ICC_{3,k}$ (Table 1). Baseline (defined as 'pre fatigue' testing at Week 1) values for all quantitative measures were compared between arms by a paired t-test. Mean and standard error (SE) values are given in the text, tables and figures.

A majority of measurements associated with PPT, VAS and motor parameters data met the requirements of a normal distribution as determined by the Shapiro-Wilk normality test. For analysis of chronic effects of repeated ECC and CON-ECC exercise on PPTs, MGF and MWEF a protocol (2 levels: ECC; CON-ECC) by time (Week 1 immediately pre-fatigue test, immediately post exercise at Weeks 1-4 and immediately pre-fatigue at Week 5) repeated measures ANOVA was used, followed by Student Newman-Keuls (SNK) post-hoc tests when significant. To establish the acute effects of low load exercise within each exercise bout at Weeks 2-4, pairwise t-tests were conducted with the significance level adjusted to $P\text{-value} < 0.025$. For fatigue data, protocol (2 levels: ECC; CON-ECC) by time (2 levels: first fatigue test x last fatigue test) repeated measures ANOVA was performed. Significance was accepted at $P < 0.05$.

RESULTS

Baseline comparisons

There were no significant baseline pre-fatigue Week 1 differences between arms in any of the quantitative measures (Table 2).

Deep tissue sensitivity (PPT)

In regard to the chronic temporal effects of exercise on deep tissue sensitivity, no significant difference in PPTs between protocols was demonstrated at CEO ($F_{(5, 60)} = 0.74$; $P = 0.60$), at ECRB ($F_{(5, 60)} = 0.31$; $P = 0.90$) or at RH ($F_{(5, 60)} = 0.94$; $P = 0.46$) from Week 1 pre-fatigue to post exercise Weeks 1-4 and pre-fatigue Week 5 (Figure 2). For both protocols, PPT increased significantly across the four-week exercise period at CEO ($F_{(5, 60)} = 7.30$; $P < 0.001$), at ECRB ($F_{(5, 60)} = 8.75$; $P < 0.001$) and at RH ($F_{(5, 60)} = 3.61$; $P < 0.006$). In comparison with pre-fatigue testing at Week 1, PPTs were significantly higher post exercise at ECRB Weeks 1 (SNK: $P < 0.04$), 3 and 4 and at pre-fatigue testing Week 5 exercise (SNK: $P < 0.001$), at CEO Weeks 3 and 4 and at pre-fatigue testing Week 5 (SNK: $P < 0.02$), at RH Week 2 to Week 4 inclusive and at Week 5 pre-fatigue testing (SNK: $P < 0.02$).

Acute temporal effects of exercise on PPTs were similar regardless of protocol. Compared with pre-exercise, PPTs were significantly higher post exercise for both protocols at ECRB Week 3 ($t_{(25)} = -3.97$, $P < 0.001$) and at Week 4 for both ECRB ($t_{(25)} = -2.85$, $P < 0.008$) and RH ($t_{(25)} = -2.42$, $P < 0.02$) (Figure 3). No significant acute changes in PPT were found at CEO within each exercise bout at Weeks 2, 3 and 4 ($P > 0.05$).

Muscle soreness and pain

No subjects reported muscle soreness, movement-related pain or at-rest pain following any exercise session for either exercise protocol at any time point (data not shown).

Maximal grip force and maximal wrist extension force

In regard to the chronic effects of exercise, an interaction between protocols and time was demonstrated for MGF ($F_{(5, 60)} = 2.65$; $P < 0.03$) (Figure 4), although there were no significant main effects (protocol: $F_{(1, 12)} = 0.03$; $P < 0.87$; time: $F_{(5, 60)} = 1.11$; $P < 0.37$). For MWEF, no significant chronic temporal effects were demonstrated between protocols from pre-fatigue testing at week 1, across all post exercise times Weeks 1-4 to pre-fatigue at week 5 ($F_{(5, 60)} = 0.1184$; $P = 0.328$) and no significant main effects were demonstrated (protocol: $F_{(1, 12)} = 0.12$; $P < 0.73$; time: $F_{(5, 60)} = 1.56$; $P < 0.19$).

Acute temporal effects of exercise on force were similar regardless of protocol: MGF decreased significantly at Week 2 post exercise compared with pre ($t_{(25)} = 2.80$, $P < 0.01$) (Figure 5) but not at Weeks 3 and 4 ($t_{(25)} = 2.80$, $P < 0.34$). Neither protocol demonstrated any significant acute pre-post exercise changes in MWEF at Weeks 2, 3 or 4 ($t_{(25)} = 2.80$, $P < 0.55$).

Muscle endurance

Muscle fatigue was not significantly different between protocols at Week 5 compared with baseline at Week 1 ($F_{(1, 12)} = 3.34$; $P = 0.09$) (Figure 6). Muscle fatigue was not reduced at Week 5 compared with Week 1 for either protocol ($F_{(1, 12)} = 2.331$; $P = 0.15$).

DISCUSSION

This study was novel in investigating the effect of repeated bouts of low load exercise on mechanical sensitivity at the lateral elbow and associated muscle force and fatigue parameters and comparing these responses between two exercise protocols. Following the four weeks of exercise, both repeated low load exercise protocols resulted in localised mechanical hypoalgesia at all three lateral elbow sites and this effect persisted at one week post exercise. Delayed onset muscle soreness was not induced for either protocol and neither protocol augmented any force parameter. No selective benefit for either exercise mode on mechanical sensitivity or muscle function was demonstrated. These results support the study hypothesis.

Deep tissue sensitivity response to repeated bout of low load exercise

Repeated low load ECC and CON-ECC exercise resulted in a time-dependent increase in PPT at all sites post exercise Weeks 3 and 4 and persisting at pre-fatigue testing Week 5. This increase in PPTs is consistent with reduced mechanical sensitivity of deep tissues as a response to repeated low load exercise. In regard to persistent changes in deep tissue sensitivity, the temporal pattern post exercise was different across PPT sites with mechanical hypoalgesia evident at ECRB initially Week 1 post exercise and at RH Week 2 post exercise but not until Week 3 at CEO. These data may reflect chronic mechanical adaptations to low load exercise that are mediated via tissue-specific (osseotendinous and musculotendinous) mechanotransduction processes occurring at differential rates (Kjaer et al. 2006) and differentially mediated at cellular, molecular and tissue levels (Wang 2006). Additionally, as both protocols involved eccentric exercise the chronic decrease in deep tissue sensitivity implies a gradual adaptation of

lateral elbow tissues to repeated loading, consistent with the repeated bout effect. This adaptive response can occur even with low load eccentric exercise (Lavender and Nosaka 2008) as performed in the current study and is characterised by attenuated sensory-motor responses to a subsequent bout of eccentric exercise (Clarkson et al. 1992). The mechanisms underlying this protective effect are thought to involve a number of complex interacting neural mechanisms and mechanical and molecular processes (McHugh 2003). It is unclear however if a persistent decrease in deep tissue mechanical sensitivity parallels this attenuated response as to our knowledge no studies have investigated this specific psychophysical response following repeated sessions of low load exercise. Whether such persistent deep tissue sensitivity changes also occur in response to single exercise modes (ie concentric, isometric) requires further investigation.

As mechanical hypoalgesia did not occur immediately after every single exercise bout, it is unlikely that acute activation of endogenous pain-modulatory systems as described following a single bout of exercise (Koltyn and Umeda 2007) was a dominant mechanism underlying the cumulative exercise-induced mechanical hypoalgesia. In contrast, the further acute decrease in PPT post exercise and post fatigue testing at ECRB (Weeks 3 and 4) and at RH (Weeks 4), does suggest activation of endogenous pain modulatory systems in addition to chronic tissue adaptations outlined above. These PPT data are in accordance with the findings of immediate exercise-induced mechanical hypoalgesia demonstrated in healthy subjects following a single bout of exercise (Koltyn et al. 2001; Staud et al. 2005) with load parameters similar to those described in the current study. The acute post exercise mechanical hypoalgesia elicited at Weeks 3

and 4 is unlikely to be explained by the exercise stimulus, as the protocol parameters did not change across the four weeks. Although speculative, it is plausible that following repeated low load exercise, the potential to more effectively recruit segmental inhibitory or plurisegmental pain modulatory mechanisms is enhanced. In the current study, given the absence of induced tissue damage (i.e., self report indicated no induced pain or muscle soreness following any bout of exercise) the balance between pronociceptive and anti-nociceptive mechanisms may shift in favour of pain inhibition. The acute exercise-induced mechanical hypoalgesia may also reflect the different innervation densities that exist between muscle and tendon (Mense and Simons, 2001). In comparison with tendon (CEO), as exercising muscles including ECRB and the extensor muscles overlying RH activate A β afferents (Mense and Simons, 2001), these muscles may more efficiently recruit endogenous pain modulatory systems. This may explain the more pronounced increase in PPT of ECRB and RH following exercise. It is also possible that in response to ECC and CON-ECC low load exercise muscle, tendon and bone adaptations including deep tissue mechanical sensitivity may not occur at the same rates or to the same degree (Wang 2006). Future studies will incorporate a more distant PPT site as a control to implicate segmental or plurisegmental pain modulatory system activation. Additionally, the possibility that repeated isometric contractions may also generate persistent deep tissue sensitivity changes cannot be excluded. Further investigation is required to establish any additional persistent effect on deep tissue sensitivity of the two pre-exercise fatigue tests, as conducted in the current study.

Muscle strength response to repeated bouts of low load exercise

Within each successive bout no muscle damage appeared to be induced after either protocol, since no delayed onset muscle soreness or significant decreases in force were evident following exercise. Where demonstrated, the modest decrease in force immediately after exercise is most likely due to muscle fatigue rather than damage. As the load was deliberately not increased at each session, no significant increase in grip and wrist extensor force was predicted following the 4 week repeated low load exercise period. Maximal grip force did vary between protocols, however given there were no main effects combined with an inconsistent temporal interaction pattern and the modest force difference (less than 2 kg), these data suggest a clinically insignificant finding. The lack of increase in endurance following the exercise period was most likely due to an insufficient exercise stimulus to induce changes or might indicate resilience to fatigue of the wrist extensors (Finsen et al. 2005).

For the current protocols, 30% maximal isometric wrist extension was chosen for the exercise load as this exceeds many of the demands of the wrist extensor muscle group during activities of daily living (Finsen et al. 2005) and our pilot data indicated this load did not provoke muscle soreness. The frequency of exercise at weekly intervals was also chosen to avoid the potential tissue sensitizing effects and force attenuation commonly associated with provoked muscle damage following unaccustomed eccentric exercise (Cleak and Eston 1992).

CONCLUSION

Repeated low load exercise incorporating an eccentric component is associated with mechanical hypoalgesia of lateral elbow musculotendinous tissues. In healthy subjects,

the current study suggests no significant difference between exercises consisting of eccentric contractions only and of combined concentric and eccentric contractions for exercising the wrist extensors to induce mechanical hypoalgesia. Using low load exercise to generate localised mechanical hypoalgesia may prove beneficial if replicated in patients with lateral elbow tendinopathy.

Figure 1
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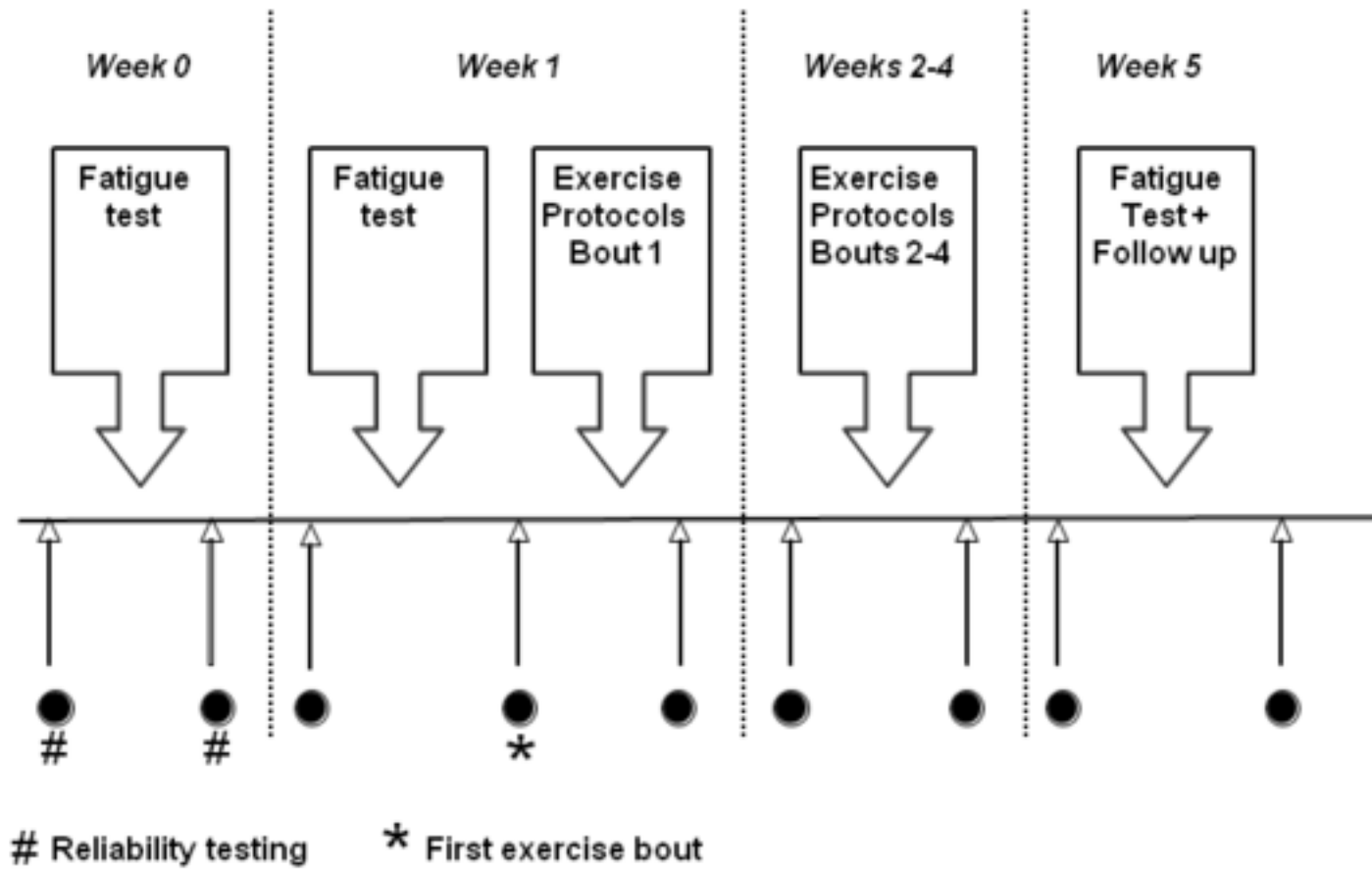


Figure 2
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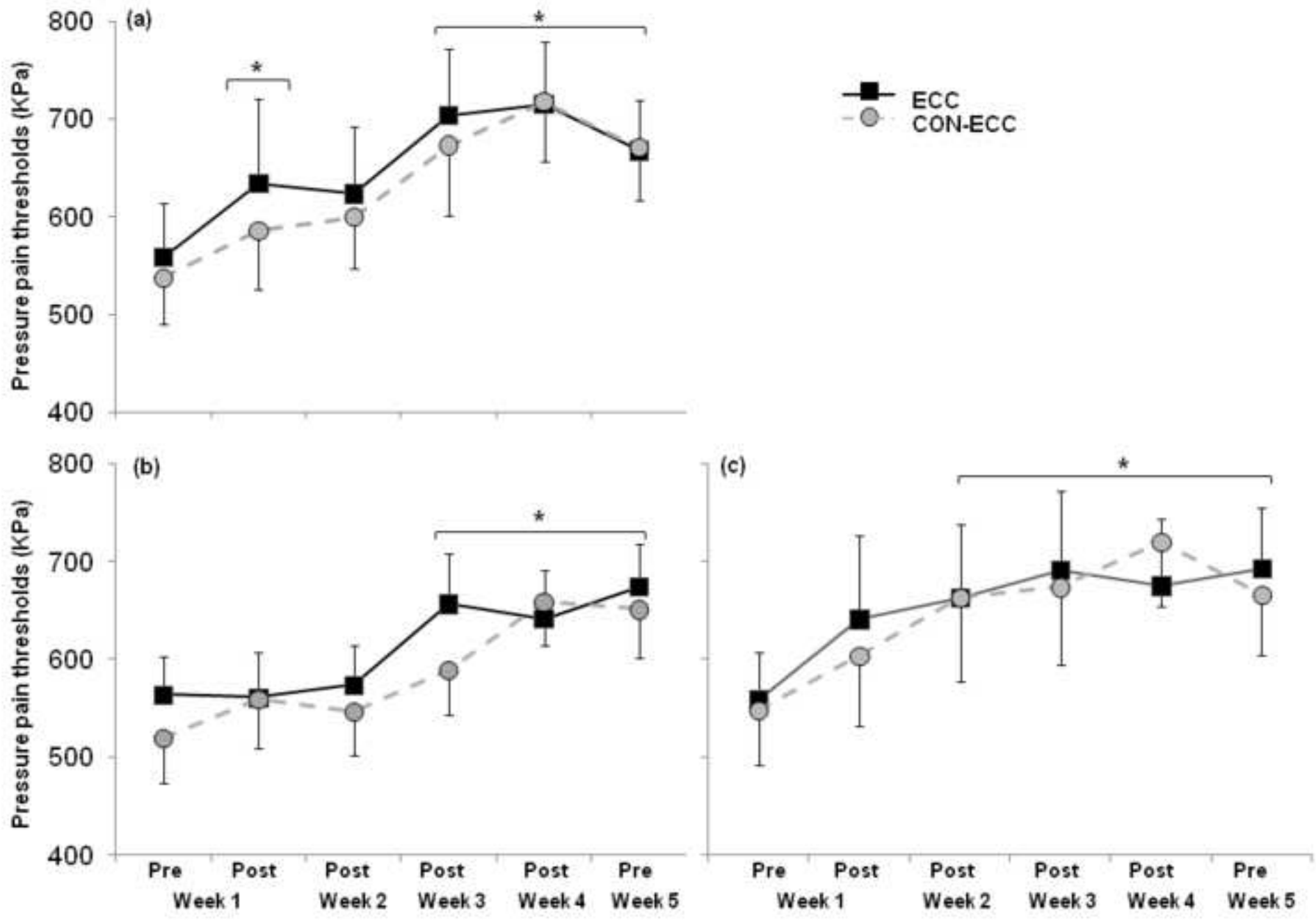


Figure 3
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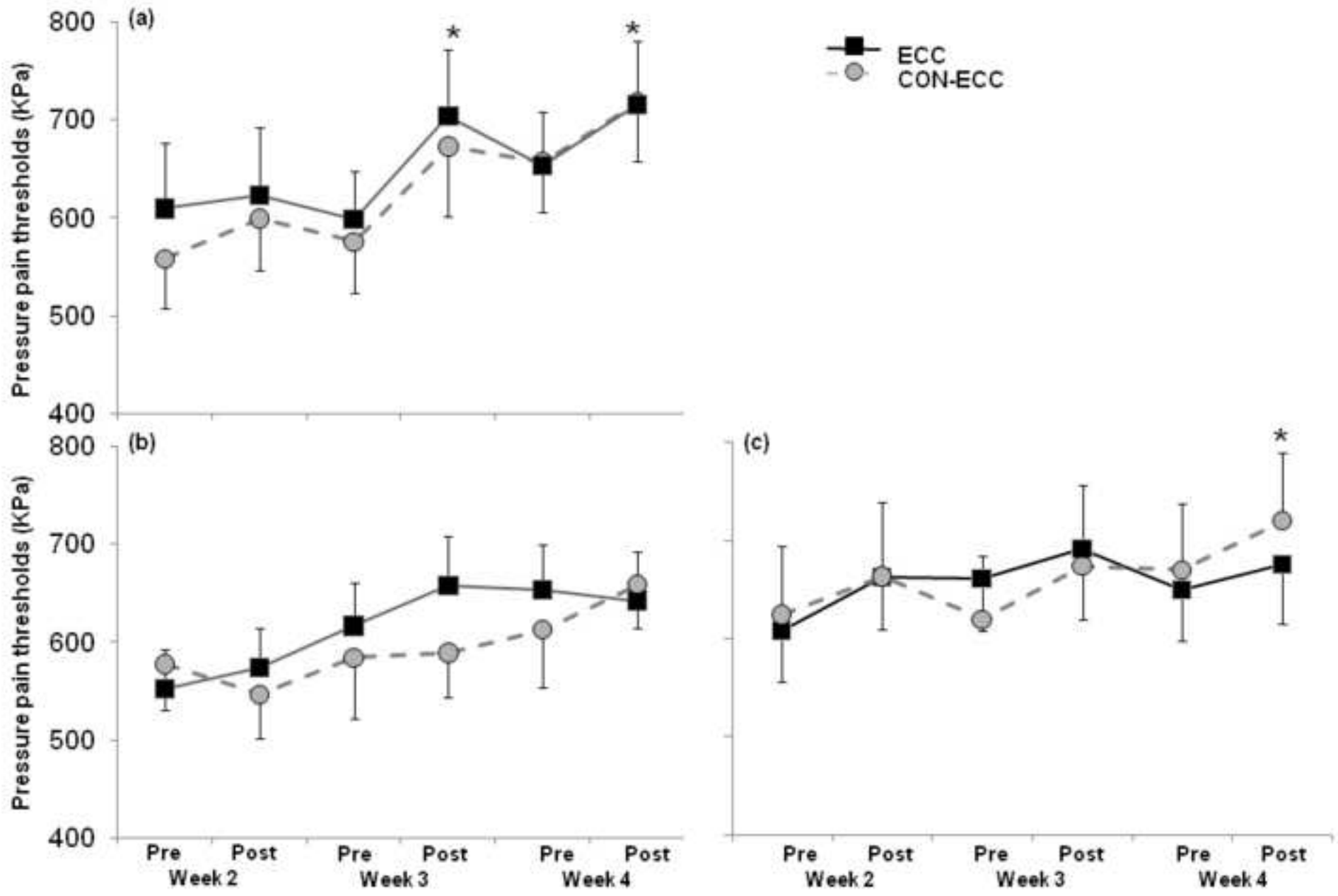


Figure 4
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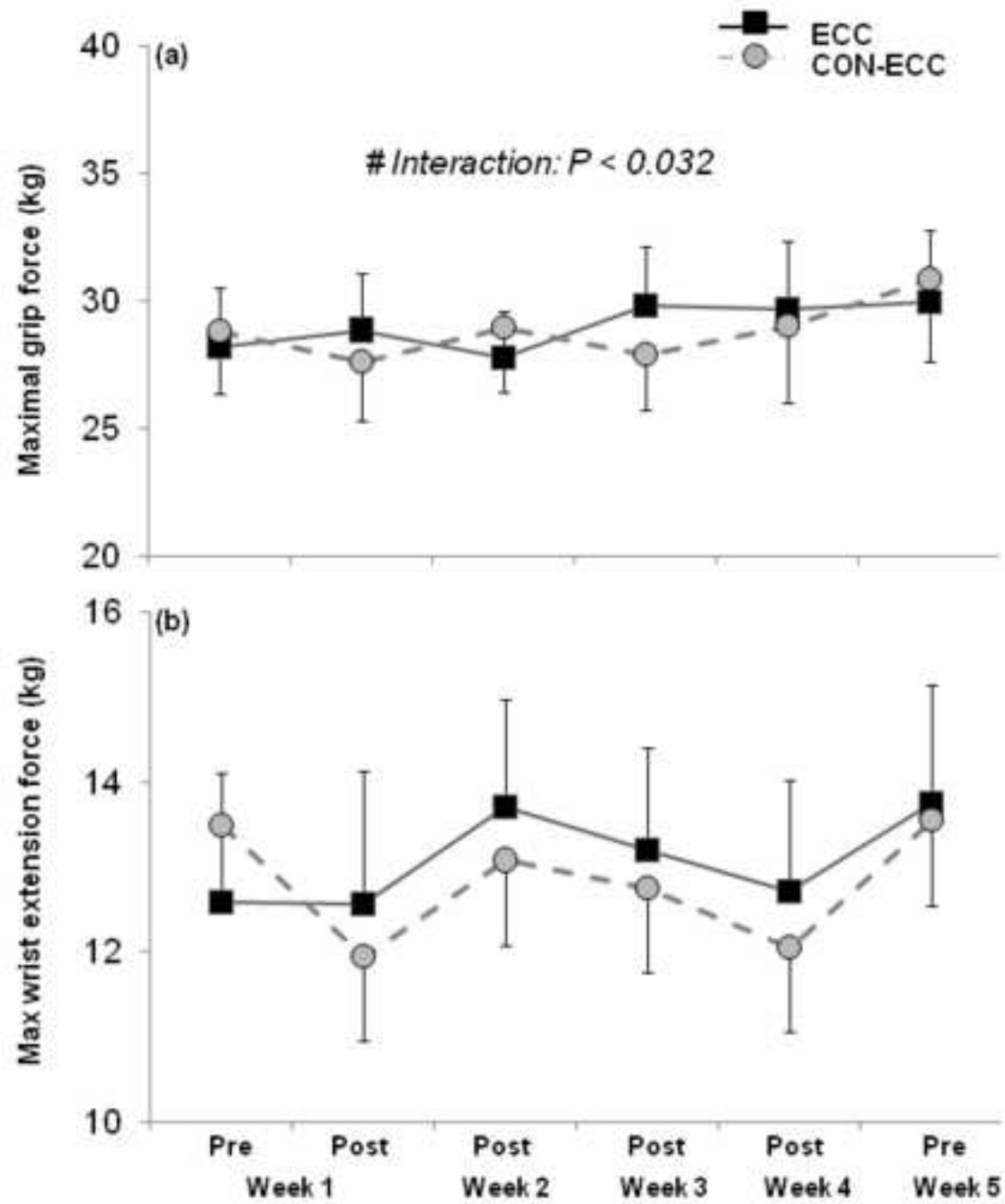


Figure 5
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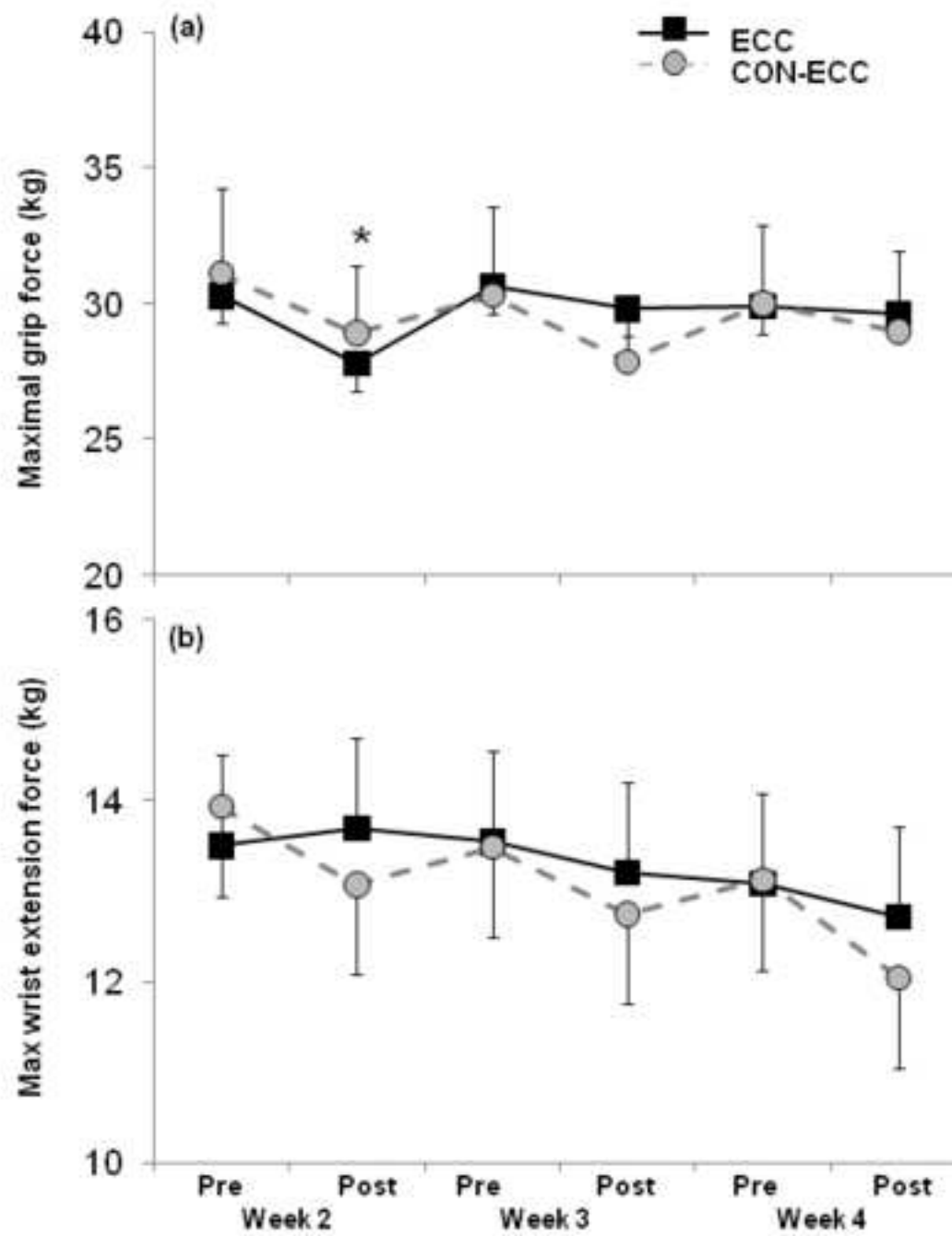
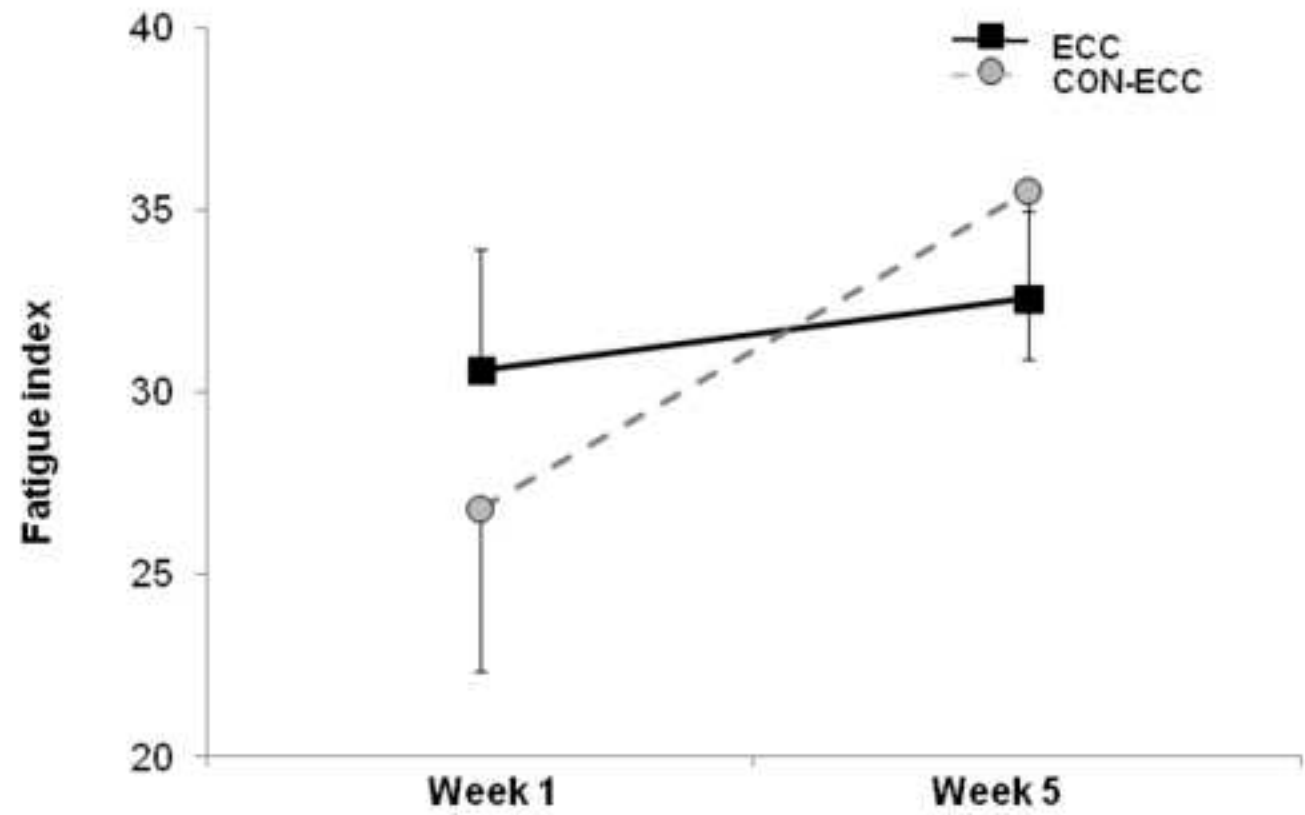


Figure 6
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TABLES

Table 1: Intraclass Correlation Coefficients (ICC) and 95% confidence intervals established by repeating measurements at Week 0 and at Week 1 pre-exercise (baseline) for pressure pain thresholds (PPT) at three sites (CEO = Common extensor origin; ECRB = Extensor carpi radialis brevis; RH= Radial head), maximal grip force (MGF), maximal wrist extensor force (MWEF), and fatigue index. High levels of reliability were demonstrated for all variables as indicated by the ICC values close to 1, variance ratios (expressed by the 'F' statistic) and significant *P*-values.

Variables	ICC (3, k)	95 % Confidence Interval		F	Sig.
		Lower Bound	Upper Bound		
PPT (kPa)					
CEO	0.84	0.63	0.93	6.09	0.001
ECRB	0.73	0.40	0.88	3.72	0.001
RH	0.81	0.58	0.92	5.28	0.001
MGF (kg)	0.93	0.85	0.97	14.94	0.001
MWEF (kg)	0.96	0.92	0.98	27.99	0.001
Fatigue index	0.75	0.35	0.92	3.93	0.002

Table 2: Baseline mean (\pm SE; $n = 13$) values for pressure pain thresholds (PPT) at three sites (CEO = Common extensor origin; ECRB = Extensor carpi radialis brevis; RH= Radial head), maximal grip force (MGF) and maximal wrist extensor force (MWEF) for the eccentrically trained arm (ECC) and concentric-eccentric trained arm (CON-ECC). The results of the comparison between ECC and CON-ECC arms demonstrate no significant baseline differences between arms as indicated by the non-significant *P*-values.

Variables	ECC	CON-ECC	<i>P</i> -value
PPT-CEO (kPa)	563.9 (40.1)	520.0 (46.7)	0.84
PPT-ECRB (kPa)	558.9 (54.5)	537.9 (47.9)	0.17
PPT-RH (kPa)	559.7 (48.0)	548.6 (56.3)	0.95
MGF (kg)	28.2 (2.3)	28.8 (2.5)	0.50
MWEF (kg)	12.6 (1.5)	13.5 (1.9)	0.44
Fatigue index	30.6 (3.3)	26.8 (4.5)	0.32

Table 3

Table 3: Mean (\pm SD; N = 13) values for all variables for the eccentrically trained arm (ECC) and concentric-eccentric trained arm (CON-ECC) Weeks 1-5.

Variables	Week 1		Week 2		Week 3		Week 4		Week 5	
	Pre fatigue	Post-exercise	Pre-exercise	Post-exercise	Pre-exercise	Post-exercise	Pre-exercise	Post-exercise	Pre-fatigue	Post exercise
PPT-CEO (kPa)										
ECC Con	563.9 (144.6)	561.4 (166.1)	552.7 (144.9)	574.8 (147.9)	616.5 (157.5)	657.0 (183.4)	652.6 (170.1)	641.9 (179.1)	674.5 (157.9)	706.8 (180.7)
CON-Ecc	520.0 (168.3)	559.3 (181.9)	577.7 (171.0)	546.6 (161.6)	584.6 (223.1)	589.2 (165.3)	612.7 (214.5)	658.8 (161.0)	651.6 (178.2)	719.1 (213.0)
PPT-ECRB(kPa)										
ECC Con	558.9 (196.4)	634.0 (311.1)	609.6 (242.2)	623.6 (245.7)	598.8 (178.1)	704.4 (243.4)	653.0 (201.0)	715.4 (231.0)	667.9 (185.1)	758.2 (263.1)
CON-Ecc	537.9 (172.9)	586.0 (218.8)	559.1 (184.4)	559.6 (192.0)	576.0 (188.6)	672.8 (259.2)	656.9 (185.6)	717.3 (216.9)	670.6 (193.8)	702.4 (187.4)
PPT-RH (kPa)										
ECC Con	559.7 (173.2)	641.2 (310.1)	608.1 (252.5)	662.8 (275.6)	660.8 (232.1)	691.3 (295.7)	649.5 (245.4)	675.3 (250.1)	692.8 (224.6)	760.7 (286.6)
CON-Ecc	548.6 (202.9)	603.4 (259.4)	624.6 (258.4)	663.3 (307.7)	619.8 (240.0)	674.2 (284.4)	670.4 (230.9)	720.1 (235.6)	666.0 (220.9)	735.4 (251.3)
MGF (kg)										
ECC Con	28.2 (8.4)	28.9 (8.1)	30.3 (8.6)	27.8 (6.5)	30.7 (11.0)	29.8 (8.5)	29.9 (9.2)	29.7 (9.8)	30.0 (10.0)	25.5 (7.3)
CON-Ecc	28.8 (9.0)	27.6 (8.3)	31.1 (11.4)	28.9 (9.0)	30.3 (11.8)	28.1(8.1)	30.0 (10.6)	29.0 (10.8)	30.9 (11.5)	24.2 (7.2)
MWEF (kg)										
ECC Con	12.6 (5.5)	12.6 (5.6)	13.5 (6.1)	13.7 (4.6)	13.6 (5.2)	13.2 (4.3)	13.1 (5.2)	12.7 (4.7)	13.7 (5.0)	13.3 (4.5)
CON-Ecc	13.5 (7.0)	11.9 (5.5)	13.9 (6.2)	13.1 (4.9)	13.5 (5.2)	12.8 (4.5)	13.1 (5.7)	12.1 (5.2)	13.6 (5.5)	13.7 (6.4)

Legend: PPT = pressure pain thresholds; CEO = Common extensor origin; ECRB = Extensor carpi radialis brevis; RH= Radial head; MGF = maximal grip force; MWEF = maximal wrist extensor force