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Impact of an acute bout of vibration on muscle contractile properties, creatine kinase and lactate dehydrogenase response

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Abstract

The aim of this study was to assess the effects of a bout of whole body vibration (WBV) on muscle response and to determine whether this stimulus leads to muscle damage. Thirty healthy and physically active participants (mean $\pm$ SD; age: 21.8 ± 2.0 years; height: 176.7 ± 5.8 cm; body mass: 76 ± 6.8 kg and BMI: 23.1 ± 3.7 kg·m⁻²) participated in this study. Participants were randomly allocated in one of two groups, one of them performed a bout of 360 s WBV (frequency: 30 Hz; peak-to-peak displacement: 4 mm) (VIB) and the other one adopted a sham position (CON). Muscle contractile properties were analysed in the rectus femoris (RF) by using tensiomyography (TMG) 2 min before the warm-up and 2 min after intervention. Muscle damage was assessed by determining plasma creatine kinase (CK) and lactate dehydrogenase (LDH) levels at three time points; 5 min before warm-up and 1 h and 48 h after the intervention. TMG results showed a significant decrease in maximal displacement ($p < 0.05$) and delay time ($p < 0.05$) in VIB and in delay time ($p < 0.05$) and relaxation time ($p < 0.05$) in CON. Muscle damage markers showed significant group differences ($p < 0.05$) for CK 1 h after the intervention. In addition, differences for CK 1 h after the intervention from baseline ($p < 0.05$) were also observed in VIB. In conclusion, a 6-min bout of WBV results in an increase of muscle stiffness in RF and increased CK levels 1 h after intervention (returning to baseline within 48 h).

Keywords: muscle damage, vibration training, tensiomyography

Introduction

Whole body vibration (WBV) training is a relatively new neuromuscular training method that is gaining popularity. Studies using vibration training have shown different effects on muscle performance, such as strength (McBride et al., 2010), jump ability (Da Silva-Grigoletto, De Hoyo, Sañudo, Carrasco, & García-Manso, 2011) and power output (Da Silva-Grigoletto et al., 2009, 2011).

Improvements in muscle function with WBV are thought to be associated with enhanced neural excitation (Cardinale & Bosco, 2003; Rittweger, Mutschelknauss, & Felsenberg, 2003) and possibly by increased reflex activation (Cardinale & Bosco, 2003). Sensory receptors that modulate muscle stiffness detect this through reflex muscular activity

and attempt to dampen the vibratory waves (Cardinale & Bosco, 2003). Every cycle of WBV induces a successive and frequency-dependent activation of the muscle spindles, which leads to muscle contraction (Ritzmann, Kramer, Gruber, Gollhofer, & Taube, 2010). During the WBV training, muscle excitation is transmitted via Ia afferents and generates numerous discharges at the α -motoneuron pool, which lead to muscle stiffness to mitigate the vibration transmission (Ritzmann et al., 2010). However, the adaptations following WBV are inconsistently reported in the literature (Rittweger, 2009). Recently, Ritzmann, Kramer, Gollhofer, and Taube (2011) showed that after WBV the reflex response was depressed returning to baseline values after just a few minutes. Thus, according to these authors, performance

improvements after WBV are not likely to be caused by spinal facilitation, therefore an alternative mechanism must be responsible for the performance improvements reported in some studies.

Possible explanations for the reported improvements in muscle function with WBV include the effects of WBV on muscle temperature (Cochrane, Stannard, Firth, & Rittweger, 2010) and some authors attribute improvements to what they describe as post-activation potentiation (PAP; Cardinale & Bosco, 2003; McBride et al., 2010). The PAP is commonly associated with a maximum or submaximal muscle contraction, which results in improved muscle response (Tillin & Bishop, 2009). The mechanisms, which facilitate PAP, are related to the phosphorylation of light chain regulatory myosin (RCL), increased recruitment of motor neurons and a possible change in pennation angle. However, a previous stimulation could also lead to fatigue and therefore, a subsequent response may be determined by the PAP-fatigue balance (Sale, 2002). Consequently, this phenomenon is similar to a bell-shape where muscle stiffness contributes to the potentiation effects but muscle fatigue or even muscle damage may decrease it (Hunter et al., 2012). However, the links between muscle damage and muscle stiffness are not well defined, several studies seem to indicate a clinically significant relationship between muscle stiffness and muscle damage (Brockett, Morgan, & Proske, 2004; Hody, Rogister, Leprince, Wang, & Croisier, 2011; McHugh et al., 1999). In this sense, McHugh et al. (1999) demonstrated greater strength loss, pain, muscle tenderness, and muscle damage in those participants with stiff muscles compared to those with compliant muscles. These authors found that participants with stiffer hamstring muscles experienced greater muscle damage, despite exercising at the same relative intensity.

One possible explanation to this interaction was attribute to the presence of eccentric muscle contractions in WBV training (Rittweger et al., 2003), which are known to induce, in some situations, muscle fibre damage (Zhou, Li, & Wang, 2011). In this sense a significant correlation between muscle damage (plasma creatine kinase – CK – concentrations) and muscle stiffness was found after intense eccentric exercise (Hody et al., 2011).

On the other hand, in order to better understand this relation, several studies have analysed the muscle damage associated with different exercise interventions using biological markers together with strength, stiffness, pain and electromyographic response (Chapman, Newton, McGuigan, & Nosaka, 2008; Miyama & Nosaka, 2007). However, while markers such as CK and LDH are commonly used for diagnosis of muscle damage, even after WBV training (Gojanovica, Feihlb, Liaudetb, Gremiona, &

Waeberb, 2011), techniques to assess complementary muscle contractile properties test are infrequently used.

Recently, a new technique known as tensiomyography (TMG) is being used in studies focused on muscle contractile properties and function in response to a low-level electrical stimulus (Tous-Fajardo et al., 2010). TMG uses a high precision ($4\ \mu\text{m}$) digital pre-tension displacement transducer sensor placed perpendicular to the muscle belly to record radial muscle belly displacement (Hunter et al., 2012). TMG has also been used to assess the level of muscle activation and even muscle stiffness in different sporting activities (García-Manso et al., 2011; Križaj, Simunic, & Zagar, 2008; Rey, Lago-Peñas, Lago-Ballesteros, & Casáis, 2012). Moreover, this technique has been used to effectively detect fatigue after cycling at maximal oxygen uptake power output (Carrasco, Sañudo, de Hoyo, Pradas, & da Silva, 2011) and following an ultra-endurance triathlon (García-Manso et al., 2011). To our knowledge, only Hunter et al. (2012) correlated different TMG parameters with muscle damage measured with CK levels after an eccentric elbow flexion.

Therefore, and considering that none studies have been conducted after vibration and the causes of the adaptations to WBV observed in training studies have not been pinpointed yet, the aim of the current study was to assess the effects of one bout of WBV on muscle response and to determine whether this stimulus leads to muscle damage. Consequently, we hypothesised that an acute bout of WBV would induce muscle damage (assessed by CK and LDH levels), accompanied by elevated muscle stiffness and fatigue (assessed by TMG).

Methods

Participants

Thirty healthy male volunteers were recruited for this study (mean $\pm$ SD; age: 21.82 ± 2.01 years; height: 176.67 ± 5.79 cm; body mass: 76 ± 6.81 kg and BMI: 23.14 ± 3.66 kg·m⁻²). Medical histories were reviewed by a physician to assess suitability for the study. Each subject completed 'The International Physical Activity Questionnaire' (IPAQ; Craig et al., 2003) in order to determine the level of physical fitness prior to the study, this allowed recruitment of recreationally active subjects, according to the criteria established by Varo et al. (2003). Additionally, subjects with osteoarticular conditions (including fracture or injury) were excluded. The study was conducted according to the Declaration of Helsinki, and the protocol was fully approved by the local research ethics committee before recruitment. After a detailed explanation about the aims, benefits and risks

involved in this investigation, all participants gave written informed consent. Table I shows the descriptive data of the selected participants.

Study design and procedure

Subjects were randomly allocated to one of two different protocols: continuous WBV protocol (VIB; $n=15$) or a sham position (CON; $n=15$). All participants were familiarised with the vertical vibrating platform (Pro5 Airdaptive, Power Plate North America, Inc., Northbrook, IL) and the proper positioning. In the VIB group the WBV stimulus was induced during 360 s to the plantar surfaces of the feet at a frequency of 30 Hz with a peak-to-peak displacement of 4 mm according to that indicated by the manufacturer, considered the optimal combination to get the greater muscle performance (Da Silva-Grigoletto et al., 2009, 2011). CON participants received no vibration (360 s). All participants adopted a squat position, knees and ankles flexed at 60° (knee extension = 0°) and 90° , respectively, as measured by an electronic goniometer. This position was reported to be the one where the electromyographic activity was highest in the RF according to Ritzmann, Golhofer, and Kramer (2012). To avoid bruising, all participants wore no sport shoes. All tests were preceded by a 5-min warm-up consisting of cycling on a cycloergometer (Ergoline 900[®], Ergometrics, Bitz, Germany) at 60 W and 60 rpm. The blood samples were obtained 5 min before warm-up and 1 h and 48 h after intervention and TMG tests were performed 2 min before the warm-up and 2 min after the bout of WBV. All tests and interventions were performed at the same time of day. Participants were not allowed to consume water and food during and until 1 h after the intervention.

TMG measurements

Displacement-time curve recordings allow for muscle contractile properties to be assessed (Valenčič & Knez, 1997). The validity of the TMG parameters has been reported in previous studies (Križaj et al., 2008; Pišot et al., 2008). Despite the aforementioned validity, some limitations have been reported with this technique. TMG-derived contractile parameters

were very sensitive to alterations in regard to inter-electrodes position and also variations in muscle response. The difficulties in repositioning both the sensor and the electrodes in the same area may also affect TMG measurements and may affect its reliability. Finally, some other intrinsic factors such as skin conductivity, subcutaneous fat thickness or motor nerve branching may also affect inter-individual variability (Tous-Fajardo et al., 2010). Therefore, to ensure the reliability of the measurement in the current study we proceeded according to indications proposed by the authors (Tous-Fajardo et al., 2010).

The main outcome variables determined in the current study were: maximal radial displacement (Dm), time from the onset of electrical stimulus to 10% of Dm (Td), time from 10 to 90% of Dm in the ascending curve (Tc), time between 50% of Dm on both sides of the curve (Ts), and time from 90 to 50% of Dm on the descending curve (Tr). These outcomes were previously assessed in the literature with low Dm values indicating greater muscle stiffness (Dahmane, Valencic, Knez, & Erzen, 2001; Križaj et al., 2008; Valencic, Knez, & Simunic, 2001; Rey et al., 2012; Hunter et al., 2012), meanwhile with AQ5 these changes are associated with increments in Td and Tr seems to indicate muscle fatigue (Dahmane, Djordjevic, Simunic, & Valencic, 2005; Garcia-Manso et al., 2011).

Radial displacements were measured under static and relaxed conditions, with the participant in the supine position and the knee joint fixed at an angle of 60° (0° corresponding to full extension of the knee). The measured limb was positioned on a wedge foam cushion to keep a fixed knee angle. A digital displacement transducer (GK 40[®], Panoptik d.o.o., Ljubljana, Slovenia), which incorporates a spring of $0.17 \text{ N}\cdot\text{mm}^{-1}$, was set perpendicular to the muscle belly to acquire radial displacement. Sensor location was determined anatomically according to Delagi, Perotto, Iazzetti, and Morrison (1975) and marked with a dermatological pen. Two square ($5 \times 5 \text{ cm}$) 2 mm thick self-adhesive electrodes (Compex Medical SA, Ecublens, Switzerland) were placed symmetrically 5 cm ($\pm 3 \text{ cm}$) to the sensor tip.

A TMG-S1 stimulator (EMF-Furlan and Co. d.o.o., Ljubljana, Slovenia) was used to induce electrical pulses (duration: 1 ms; initial intensity: 30 mA). For each TMG assessment intensity was progressively increased (10 mA intervals) until there was no further increase in Dm or the maximal device output (110 mA) was reached. Between consecutive measurements rest periods of 15 s were allowed to minimise the effects of fatigue and potentiation. In any case, none of the participants reported discomfort during electrical stimulation.

Table I. Characteristics of the participants (Mean $\pm$ SD).

	Age (years)	Height (cm)	Body mass (kg)	BMI ($\text{kg}\cdot\text{m}^{-2}$)
VIB	22.12 ± 3.14	176.21 ± 4.90	77 ± 5.46	23.65 ± 3.48
CON	20.87 ± 1.69	176.98 ± 6.07	75 ± 3.21	23.24 ± 1.88

VIB = vibration group; CON = control group.

CK and LDH measurements

For the assessment of both enzymes, blood samples (10 ml) were obtained from the antecubital vein before and 1 h and 48 h after a bout of WBV. Blood samples were dispensed immediately into a tube (5 ml) containing potassium EDTA 3K⁺ or an eppendorf tube (5 ml) for haemoglobin concentration and cell volume determination and stored on ice (−4°C). After 20 min of mixing (Spiramix-10, Denley Instruments, Sussex, UK), haemoglobin concentration (in duplicate by the cyanmethaemoglobin method) and packed cell volume (in triplicate by spun haematocrit) were measured to allow calculation of changes in plasma volume relative to the volume in different moment. Then blood samples (5 ml) were centrifuged at 3000 rpm for 10 min at 20°C in a centrifuge AllegraTM X-12R (Beckman Coulter, Inc., Fullerton, CA, USA). After removing the plasma, the aliquots were stored at a temperature of −80°C in eppendorf tubes labelled for later analysis. Measurements were carried out by two chains Accellerator Core-Lab (Abbott, Chicago, IL, USA), each composed of six analyzers: three teams Architect i2000 immunoassay and three Architect c16,000 spectrophotometry (used in the CK and LDH analysis). In each biochemical quantity gauges were used and reagents specific to the commercial house. All parameters have been subjected to daily internal quality control of Inter-QC (VITROS®) and monthly external quality control of the Spanish Society of Clinical Biochemistry and Molecular Pathology (SEQC).

Statistical analyses

Means and standard deviations of the mean (SD) were calculated for each variable. Normality was checked using the Kolmogorov-Smirnov test and all variables were normally distributed. A 2 × 2 analysis of variance (ANOVA) was performed to compare TMG measurements between protocol (VIB and CON) and moment (before and after intervention). A 2 × 3 repeated measures ANOVA was performed to compare changes in the muscle damage enzymes

and haemoglobin concentrations and haematocrit levels between protocols (VIB and CON) and moment (before, 1 h and 48 h after intervention). When a significant *F*-value interaction was achieved, a Tukey post hoc test was used to examine where significant differences occurred. The level of significance was set at *p* < 0.05. All analyses were performed using SPSS v.18 (SPSS Inc., Chicago, IL).

Results

Table II represents the TMG response on RF before and after the interventions. ANOVA test showed no significant inter-group differences before and after the intervention. Intra-group analysis revealed statistically significant changes for Dm (−22.78%; *F*(1,29) = 6.718; *p* < 0.05) and Td (−7.45%; *F*(1,29) = 5.289; *p* < 0.05) in the VIB group, while significant differences in Td (−8.96%; *F*(1,29) = 6.070; *p* < 0.05) and Tr (−41.54%; *F*(1,29) = 4.349; *p* < 0.05) were observed for the CON group.

No significant differences in percentage changes in haemoglobin and haematocrit levels between interventions and time points considered were found. Figure 1 shows the time course of CK and LDH in both groups. ANOVA test showed no significant group interaction on both CK and LDH levels before the intervention. However, significant differences were observed for CK 1 h after intervention (*p* < 0.05), although these differences were not found at 48 h. The group by moment analysis showed significant differences for CK 1 h after intervention in the VIB group (+11.05 ± 2.03%; *F*(1,29) = 4.289; *p* < 0.05), but not for the CON group. There were no significant differences in the LDH levels at any of the measurement time points.

Discussion

This study aimed to assess the acute effect of a bout of WBV on muscle stiffness or local muscle fatigue assessed by TMG. In addition, we wanted to determine whether continuous exposure to WBV might cause a change in muscle damage markers (CK and LDH). Results showed a significant decrease in Td in

Table II. Tensiomyographic parameters from rectus femoris in VIB and CON groups (Mean ± SD).

	Dm (mm)			Td (ms)			Tc (ms)			Ts (ms)			Tr (ms)		
	PRE	POST	<i>p</i>	PRE	POST	<i>p</i>	PRE	POST	<i>p</i>	PRE	POST	<i>p</i>	PRE	POST	<i>p</i>
VIB	9.13 ± 2.12	7.05 ± 2.26	0.015	23.48 ± 1.98	21.73 ± 2.17	0.029	30.82 ± 6.71	28.87 ± 5.94	0.408	120.03 ± 61.63	89.62 ± 47.60	0.142	73.42 ± 53.91	43.96 ± 34.62	0.086
CON	9.51 ± 2.77	7.75 ± 3.06	0.112	23.65 ± 1.97	21.53 ± 2.17	0.020	31.97 ± 4.14	30.11 ± 6.94	0.382	117.93 ± 52.03	96.30 ± 53.60	0.272	75.09 ± 48.04	43.90 ± 32.34	0.046

VIB = vibration group; CON = control group; Tc = contraction time; Ts = sustain time; Tr = relaxation time; Dm = maximal displacement; Td = delay time.

Figure 1. Changes respect pre-test on creatine kinase (A) and lactate dehydrogenase (B) levels 1 h and 48 h after intervention (mean $\pm$ SD).

*Significant inter-group differences ($p < 0.05$) measured by ANOVA test; #Significant intra-group differences ($p < 0.05$) measured by ANOVA test.

both groups but also in Dm for the VIB group and Tr in the CON group. Results also showed a significant increase in CK levels 1 h after intervention for the VIB group. In addition, analysis 48 h after exposure showed that CK returned to baseline levels in both groups. A significant group by moment interaction in CK was found 1 h after the intervention, while non-significant effects for LDH were reported.

Authors such as Križaj et al. (2008) indicated that TMG parameters are very sensitive to muscle fatigue and potentiation. Consistent with this statement several authors reported that Dm, Ts and Tr are the most accurate indicators (Dahmane et al., 2001, 2005; García-Manso et al., 2011; Valencic et al., 2001). Thus Dm, which could be equated to electrically evoked peak twitch torque, is determined by both the number and the type of muscle fibres recruited by the electrical stimulus, and could be considered as a measure of muscle belly stiffness (Pišot et al., 2008). In this sense, a decrease in Dm after an exercise may indicate an increase in muscle tone and greater muscle stiffness (Hunter et al., 2012). Another possible explanation to this response is the exercise-induced muscle damage, as some of the myofilaments became acutely stretched during the eccentric contractions and can no longer overlap due to inhomogeneities within the sarcomere (Proske & Allen, 2005). This process causes an

increased tension in the passive cellular structures (Morgan & Proske, 2004) and consequently could lead to a Dm decrease (Hunter et al., 2012). Moreover, some authors have found an association between muscle fatigue and a decrease in Dm (Carrasco et al., 2011; García-Manso et al., 2011). However, this response is not consistent with our results due to other contraction time parameters such as Ts and Tr are needed to complement this response (Križaj et al., 2008). Thus, when the muscle fatigue appears, a decreased in Dm together with an increased in Tr and Ts can be observed (Križaj et al., 2008). In this sense, Simunic, Rozman, and Pisot (2005) Leer fonticamfound a decrease in all temporal outcomes after a non-fatiguing exercise; by contrast, when the exercise resulted in muscle fatigue these time parameters increased. In the current study, one may consider that the decrease in Dm, but also in some temporal parameters, could indicate greater muscle stiffness in VIB for RA but not a sign of local muscle fatigue.

Taken into account the above-mentioned responses, it would be interesting to hypothesise the possible link between TMG parameters and muscle damage biological markers. Indeed, in a recent study, Hunter et al. (2012) related a decrease in Dm with muscle damage measured with CK levels after an elbow flexor eccentric exercise. A similar response was observed in our study where the decreases in Dm were accompanied with significant increases in CK levels 1 h after the intervention in the VIB group. This is in agreement with Spitzenpfeil, Schwarzer, Fiala, and Mester (1999), who analysed the effect of a 21 days strength-training programme (36 sessions) on muscle response. When the strength training was combined with vibration (24 Hz and 2.5 mm) a greater increase in the CK levels was observed when comparing with the strength training alone. However, the total load used by Spitzenpfeil et al. (1999) was greater to the one applied in our current study and considering that these authors reported a short-term impairment in muscle function, although an adaptive long-term response, it may be considered that a greater WBV stimulus could lead to an increase in this muscle damage response. In this line, Gojanovica et al. (2011) showed an increase in CK levels after a WBV bout (26 Hz, 15 mm) measured between 24 and 72 h post-exercise. However, this response only was only observed in 25% of participants. Differences in WBV protocols and other methodological issues can explain these inconsistencies.

A similar response to our study has been reported after different strength training protocols. Hurley et al. (2007) reported peak CK levels a few hours after performance of strengthening exercises, and it was also reported that after a passive recovery these

levels might be elevated up to 24 h (Brancaccio, Maffulli, Buonauro, & Limongelli, 2008). Resistance exercise protocols that involve exclusively eccentric muscle actions (Paschalis et al., 2007) cause more sustained muscle damage vs. traditional resistance exercise protocols that involve both concentric and eccentric muscle actions (Rodrigues et al., 2010). After eccentric exercises the enzymatic levels can be elevated even 72–96 h after the intervention (Zhou et al., 2011). For this reason, and taking into account the eccentric component of the WBV (Rittweger, Beller, & Felsenberg, 2000; Rittweger et al., 2003), we can speculate that our load (intensity or duration) was not enough to induce significant muscle damage 48-h post-exercise. As participants in this current study were physically active, this may have provided a protective effect or may result in muscle damage markers being permanently elevated (Kratz et al., 2002). It may be the case, therefore that the changes identified after exercise may be lower on trained subjects than on untrained subjects (Garry & McShane, 2000).

As a novelty in the current study we measured muscle damage by means of plasma LDH levels. To our knowledge this is the first study conducted to assess this response after a long bout of WBV. Only Gojanovica et al. (2011) evaluated the response of LDH after intermittent WBV protocol and neither observed changes in this biomarker. While this response is not apparent after WBV, different studies have analysed the relationship between LDH and strength training reporting statistically significant increases 24–72 h after intervention (Rodrigues et al., 2010). Normally, serum LDH activity has been shown to be elevated 24 h after bouts of exercise and is maintained for 48–72 hours (Bessa et al., 2008). In addition, serum CK activity increases to a greater extent vs. the serum activity of other muscle proteins such as LDH (Bessa et al., 2008). In fact, after a traditional strength training, CK levels tend to increase (Rodrigues et al., 2010), whereas LDH levels exhibit lower fluctuation (Rodrigues et al., 2010). This response is similar to that observed in the present study, where statistically significant changes were only observed for CK and not for LDH.

At this point one may wonder why previous studies recommended WBV for muscle damage prevention (Aminian-Far, Hadian, Olyaei, Talebian, & Bakhtiary, 2011) while our results indicate possible hazards effects. It must be noted that WBV increases muscle spindle activity and muscle pre-activation which results in greater background tension and less disruption to excitation–contraction coupling (Aminian-Far et al., 2011; Bakhtiary, Safavi-Farokhi & Aminian-Far, 2007). Theoretically, with an increase in muscle pre-activation, a greater number of motor units and muscle fibres would be recruited, which

may reduce myofibrillar stress during repeated muscle contractions, leading to accelerate recovery (Kosar, Putland, & Candow, 2011). Thus, untrained adults who maintained a static half-squat position for 60 s on a WBV platform (35Hz, 5 mm) prior to performing 6 sets of 10 maximal voluntary isokinetic eccentric knee extensors contractions experienced a decrease in CK levels and soreness compared to participants who did not perform WBV before exercise (Aminian-Far et al., 2011). Furthermore, when the effects of vibration therapy (50 Hz for 60 s) are assessed prior to walking downhill in young adults, Bakhtiary et al. (2007) discovered that WBV resulted in a significant reduction in muscle soreness and subsequent decrease in plasma CK levels post-exercise compared to subjects who did not perform WBV. These results contrast with the finding reported in our study and suggest that WBV is an effective intervention to attenuate muscle damage following intense exercise training. However, it must be taken into account that in these studies the vibration stimuli was lower (60 s), which may explain the differences shown with our study (360 s); it appears that when the duration of the stimulus is higher the effects are totally opposite.

In conclusion, our results clearly show an elevation of CK activity 1 h after WBV in the VIB group and an immediate increase in muscle stiffness. However, attending to TMG analysis, we cannot confirm the presence of local muscle fatigue. Also, our intervention induced a transient effect on CK response so plasma levels of this enzyme returned to basal values 48 h after WBV exposure. Probably, higher intensity bouts of WBV (higher frequency and greater peak-to-peak displacement than those used in our study) could provoke remarkable muscle fatigue and muscle damage. In any case, our results can help professionals in the prescription of safe WBV protocols for a wide spectrum of population.

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