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ORIGINAL ARTICLE

Acute whole-body vibration elicits post-activation potentiation

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Abstract Whole-body vibration (WBV) leads to a rapid increase in intra-muscular temperature and enhances muscle power. The power-enhancing effects by WBV can, at least in part, be explained by intra-muscular temperature. However, this does not exclude possible neural effects of WBV occurring at the spinal level. The aim of this study was to examine if muscle twitch and patellar reflex properties were simultaneously potentiated from an acute bout of WBV in a static squat position. Six male and six female athletes performed three interventions for 5 min, static squat with WBV (WBV+, 26 Hz), static squat without WBV (WBV−) and stationary cycling (CYCL, 70 W). Transcutaneous muscle stimulation consisting of a single 200 μs pulse and three patellar tendon taps were administered prior to and then 90 s, 5, 10 min post-intervention. Ninety-seconds after WBV+ muscle twitch peak force (PF) and rate of force development (RFD) were significantly higher ($P < 0.01$) compared to WBV− and CYCL. However the patellar tendon reflex was not potentiated. An acute continuous bout of WBV caused a post-activation potentiation (PAP) of muscle twitch potentiation (TP) compared to WBV− and CYCL indicating that a greater myogenic response was evident compared to a neural-mediated effect of a reflex potentiation (RP).

Keywords Twitch · Muscle contractile properties · Stretch reflex · Electrical stimulation · Rate of force development

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Introduction

Acute vertical sinusoidal whole-body vibration (WBV), eliciting rapid eccentric/concentric of the leg extensors (Cardinale and Bosco 2003; Rittweger et al. 2003; Rittweger et al. 2001), improves performance of these muscles in the short-term (Cochrane and Stannard 2005; Cochrane et al. 2008b; Cochrane et al. 2008c; Torvinen et al. 2002). This transient effect is thought to be mediated by a rapid reflex-mediated stretch-shortening (Cardinale and Bosco 2003; Rittweger et al. 2003; Rittweger et al. 2001) likely to involve the tonic vibration reflex (TVR), which stimulates the muscle spindles (Cardinale and Bosco 2003; Rittweger et al. 2001). Practically, WBV application leads to enhanced anaerobic power (Cochrane and Stannard 2005; Cochrane et al. 2008c). Previous work from this laboratory demonstrates that WBV leads also to a rapid increase in intra-muscular temperature (Cochrane et al. 2008b). Intra-muscular temperature in itself enhances muscle power (de Ruiter and de Haan 2000). Therefore, it seems that the power-enhancing effects by WBV can, at least in part, be

explained by intra-muscular temperature. However, this does not exclude a possible neurogenic potentiation from WBV, but limited research has been performed on assessing such spinal effects, with varying results and a lack of comparison to other modalities (Hopkins et al. 2008; Melnyk et al. 2008; Rittweger et al. 2003). For example, Rittweger et al. (2003) reported an enhancement of the patellar tendon stretch reflex following WBV in combination with squatting exercise to exhaustion, whereas Hopkins et al. (2008) found no such effect after 5 × 1 min intermittent vibration exposure of WBV (26 Hz). The results that WBV does not enhance spinal reflex excitability are supported by the finding that superimposing the Jendrassik manoeuvre upon WBV failed to enhance metabolic rate (Cochrane et al. 2008a). Nevertheless, stretch reflexes seem to be active during WBV, as we have reported a temporal association between EMG activity and muscle contractility; however, WBV was only performed at a very low frequency (6 Hz) (Cochrane et al. 2009). It is possible that the acute effects of WBV are related to its influence on non-neurally mediated events during the contractile process, such as those which occur distal to the neuromuscular junction.

Depending on the extent of prior work done, the muscles' performance can either be impaired through fatigue or enhanced, most likely through a phenomenon known as post-activation potentiation (PAP) (Sale 2002). PAP has been measured by either muscle twitch response or twitch potentiation (TP) and H-reflex or reflex potentiation (RP) (Grange et al. 1995; Guillich and Schmidtbleicher 1996; Sweeney et al. 1993; Trimble and Harp 1998). Enhanced TP has been reported following contractile activity such as a series of evoked twitches or sustained maximal voluntary contraction (MVC) (Sale 2002), or heavy resistance exercise prior to completing an explosive movement (Hodgson et al. 2005). The proposed mechanism to account for TP is believed to involve phosphorylation of myosin regulatory light chains making actin and myosin more sensitive to the intracellular Ca^{2+} signal (Moore and Stull 1984; Sweeney et al. 1993; Zhi et al. 2005). This results in a greater rate of cross bridge attachment for the same Ca^{2+} concentration, which in turn increases twitch tension (Metzger et al. 1989). Conversely, RP is thought to elicit reflex activity in the spinal cord, by increasing the activation of the α motoneurones (Hodgson et al. 2005; Trimble and Harp 1998).

It is therefore possible that acute WBV enhances muscular performance, in part, through PAP, by TP and/or RP; however, this theory remains untested. None of the aforementioned studies (Cochrane and Stannard 2005; Cochrane et al. 2008b; Cochrane et al. 2008c) assessed the presence of PAP by recording muscle twitch or reflex activity. Therefore it is unclear whether PAP exists and if muscle twitch and patellar reflex properties are simultaneously potentiated by an acute bout of WBV.

Therefore, the aim of this study was to compare the acute effect of WBV with another modality such as stationary cycling and control (no WBV) on PAP by assessing TP and patellar RP. Given that WBV seems to induce a high rate of alpha-motoneurone discharge, we postulate that 5 min continuous acute WBV (26 Hz) will result in a greater TP compared to centrally mediated effect of a RP from WBV-induced stretch reflex.

Methods

Participants

Six males (mean age (±SE) 22.8 ± 0.7 years; body mass 83.4 ± 1.7 kg, height 1.78 ± 0.2 m), and six females 22.8 ± 1.5 years; body mass 64.4 ± 1.4 kg, height 1.71 ± 0.2 m) competing in national level sport (field hockey $n = 9$; cycling $n = 1$; soccer $n = 2$) volunteered to participate in the study. Inclusion criteria required no history of neurological or circulatory disorders, no lower-limb or back injury in the previous 6 months and no lower-limb or back surgery in the previous 2 years. Participants were tested to determine if they had a measurable patellar tendon reflex response, and those with a measurable reflex were then asked to take part in the study. Written informed consent was obtained from the participants and ethical approval was granted by the University Human Ethics Committee.

Study design

Every participant performed three interventions, namely static squat with WBV (WBV+), the same static squat regimen without WBV (WBV−) and stationary cycling (CYCL), in a randomised balanced order with 24 h separating each intervention.

The WBV+ and CYCL were assumed to be matched for heat production. From previous research we have reported that 70 W of stationary cycling for 5 min elicits a change of 0.85°C in muscle temperature (T_m)(Cochrane et al. 2008b), which is comparable to research conducted in our laboratory that T_m increases 0.90°C from 5 min of WBV (26 Hz) with static squat (SS) (40° knee flexion). Therefore we chose to match the interventions based on a continuous 5 min protocol of WBV + SS (26 Hz) and 70 W CYCL.

Participants completed the performance tests at the same time of day and were instructed to strictly refrain from undertaking any vigorous activity 24 h prior to the interventions. All participants were familiarised with equipment and protocols the day before undertaking their first session. All interventions were performed at a constant

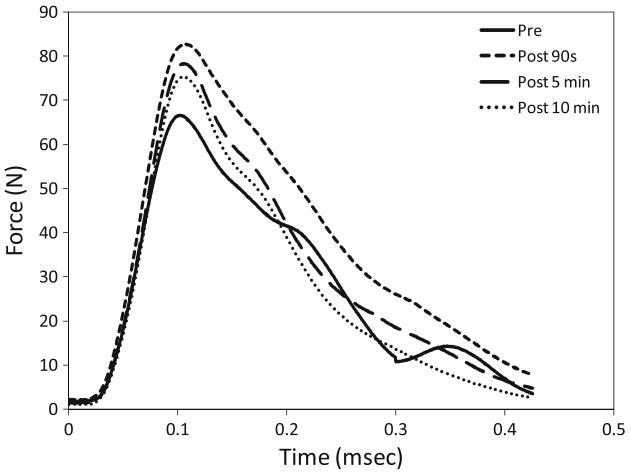

Fig. 1 A typical WBV+ response of transcutaneous muscle twitch for pre-, post-90 s, 5 and 10 min

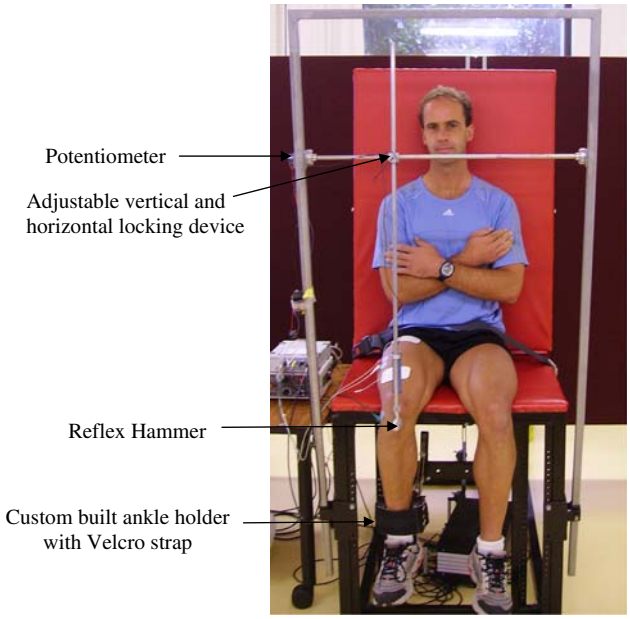

Fig. 2 Participant setup for evoked muscle twitch and patellar tendon reflex

ambient temperature (20.7 ± 0.8°C) and barometric pressure (755 ± 3.5 mm Hg).

Prior to each intervention, ~90 s, 5 and 10 min post-intervention (Fig. 1), each participant underwent transcutaneous muscle stimulation consisting of a single twitch 200 μs pulse and three patellar tendon taps.

Muscle and patellar tendon stimulation

Muscle twitch

Participants were seated in a straight-backed chair apparatus with hips and knees flexed at 90°. To prevent unwanted movement the participant's pelvic region was secured to the chair by an adjustable belt, and arms were crossed with hands placed on the opposite shoulder. The right ankle was secured to a custom-built ankle holder by a Velcro® strap. The ankle holder was connected to a load cell (6000 ICI, Sensortronics, Covina, CA, USA) and DC amplifier (Fig. 2). The load cell was fixed firmly to the undercarriage frame work of the chair apparatus. The evoked force was detected by the load cell, amplified and recorded to an acquisition system (Powerlab, 8/30, and Chart v6.1.1, ADInstruments, Australia) and desktop computer. Prior to the participant's arrival the load cell was zeroed and calibrated.

Two 5 × 9 cm self-adhesive surface electrodes were transversely placed on the distal (8 cm from the inguinal crease) and proximal areas (5 cm from the proximal border of patella) of the quadriceps muscle group. Skin preparation for electrode placement included shaving, gentle abrasion using a gel-based product (Nuprep, D.O. Weaver, USA) and cleansing with an alcohol tissue pad. To ensure that the electrode placement was the same between the interventions a permanent marker pen was used to trace the position of the electrodes. During the familiarisation the maximum twitch response of each participant was determined by a single 200 μs pulse generated by a high-voltage stimulator (model DS 7A, Digitimer Ltd, Welwyn, Garden City, England). This involved increasing the twitch voltage in 50–100 mA steps until a plateau in twitch force was reached. This value was noted as the maximal twitch response and used in the measurements at the time of the three subsequent interventions. A typical twitch stimulus for WBV+ before, ~90 s, 5 and 10 min after the intervention is shown in Fig. 1.

Patellar tendon reflex

A reflex hammer (MLA93 ADInstruments, USA) was extended onto an alloy rod that rotated about a horizontal axis mounted to a steel frame and bolted to the chair apparatus. A 10-bit potentiometer (Model 533, Vishay, Malvern, PA) was fixed to the bearing of the rotating axis which measured the angle of release recorded by a data acquisition system (Powerlab, 8/30, and Chart v6.1.1, ADInstruments, Australia) (Fig. 2). A piezo-electric sensor was located in the head of the hammer that gave an output signal which was indicative of the hammer strike force. The reflex hammer had an adjustable vertical and horizontal locking device that positioned the head of the hammer to strike the centre third of the patellar tendon. The exact contact point of the tendon strike on the skin was then marked with a permanent marker which was used for subsequent trials. For every strike the reflex hammer was raised to 60° from the vertical before release to give consistent and

repeatable tendon reflex response. The angle of the reflex hammer was calibrated prior to every testing session. Three patellar tendon strikes were administered with 15 s rest separating each strike.

Electromyography (EMG)

EMG activity was recorded from the right leg of the vastus lateralis (VL) muscle. Surface pre-gelled Ag–AgCl electrodes, 10 mm diameter (Medicostest, Rugmarken, Denmark) were placed over the muscle belly at an inter-electrode distance of 20 mm with a reference electrode placed over the head of the fibula. The EMG signal was amplified at a predetermined bandwidth filter of 10–500 Hz with a gain of 1,000. The skin of the electrode site was prepared by shaving, gentle abrasion using a gel-based product (Nuprep, D.O. Weaver, USA), and cleansed with an alcohol swab.

Interventions

WBV+

Acute WBV was performed on a commercial machine (Galileo Sport, Novotec, Pforzheim, Germany), which has a motorised teeterboard that produces side-to-side alternating vertical sinusoidal vibrations to the body. To negate the possibility of discomfort to the sole of the foot, participants wore sport shoes and placed their feet on either side of the central axis which corresponded to vibration amplitude (peak-to-peak) of 6 mm, magnitude of 9 g and the vibration frequency was set to 26 Hz for 5 min. This concurred with previous reports that 26 Hz augments jump height from a possible warm-up effect (Bosco et al. 1998; Cochrane and Stannard 2005; Cochrane et al. 2008b). During exposure to WBV the participants were in a static squat position of 40° knee flexion. A manual goniometer was used to set the knee angle, and to ensure that the position was maintained through the intervention, an adjustable hurdle was placed under the participant's gluteal fold.

WBV−

The vibration machine was set to 0 Hz (amplitude 0 mm, magnitude 1 g), with the same static squat position and time parameters used for WBV+.

CYCL

This involved pedalling at a cadence of 50 revolutions per minute on a stationary friction-braked cycle ergometer (Monark 874 E, Varberg, Sweden) for 5 min at 70 W. As mentioned previously, the workload of 70 W was selected from a previous study that showed at 5 min T_m increases to 0.85°C (Cochrane et al. 2008c), which is comparable to work conducted in our laboratory of 0.9°C increase in T_m from WBV+ with static squat performed at 40° knee flexion.

Data analysis

All data were recorded and sampled at 2,000 Hz using a data acquisition system (PowerLab 8/30 ADInstruments, Australia) and was stored off-line on a desktop computer and analysed using chart software (version 6.1.1, ADInstruments, Australia). Prior to analysis all recorded signals were visually checked for recording-related artefacts. The onset of muscle activation (EMG) and force was defined as the change in three standard deviations from resting values of muscle EMG and force. Each muscle twitch at pre-, post-90 s, 5 and 10 min was analysed for the following characteristics (Fig. 3); (1) muscle twitch peak force (PF) was determined at the highest amplitude; (2) time to peak force (TPF) was defined as the time from force onset to PF; (3) rate of force development (RFD) was defined as the average slope of the twitch force–time curve (ΔPF/TPF). For reflex data, the onset of EMG and onset of force was defined as the change in three standard deviations during baseline values conducted over 0.03 s and analysed by the chart software. Individual markers were then placed on the appropriate time interval of EMG, force onset and hammer contact to calculate reflex latency and electromechanical delay. The mean of three hammer strikes at pre-, post-90 s, 5 and 10 min were analysed for (1) electromechanical delay (EMD), defined as the time from the onset of muscle activation to force onset (Fig. 4); (2) patellar reflex latency (RL), defined as the time interval from the reflex hammer impact to onset of muscle activation (Fig. 4); (3) patellar reflex peak force (RPF), calculated as the maximum amplitude and normalised to the amplitude of the reflex hammer impact (Fig. 4). RL and RPF were chosen because they are able to assess changes in muscle spindle sensitivity or motoneurone excitability (Hopkins et al. 2008; Rossi-Durand 2002). Likewise EMD was selected because it is an indirect measure of musculo-tendinous stiffness (Isabelle et al. 2003) and provides information of spindle sensitivity via alpha–gamma co-activation (Hopkins et al. 2008).

Statistical analyses

For all the interventions the mean relative changes were calculated as the difference between post- (90 s, 5 and 10 min) and pre-values. For EMD and RL absolute changes were calculated between post- and pre-values. A 2 factor [time (pre-, post-90 s, 5, 10 min) × intervention (WBV+,

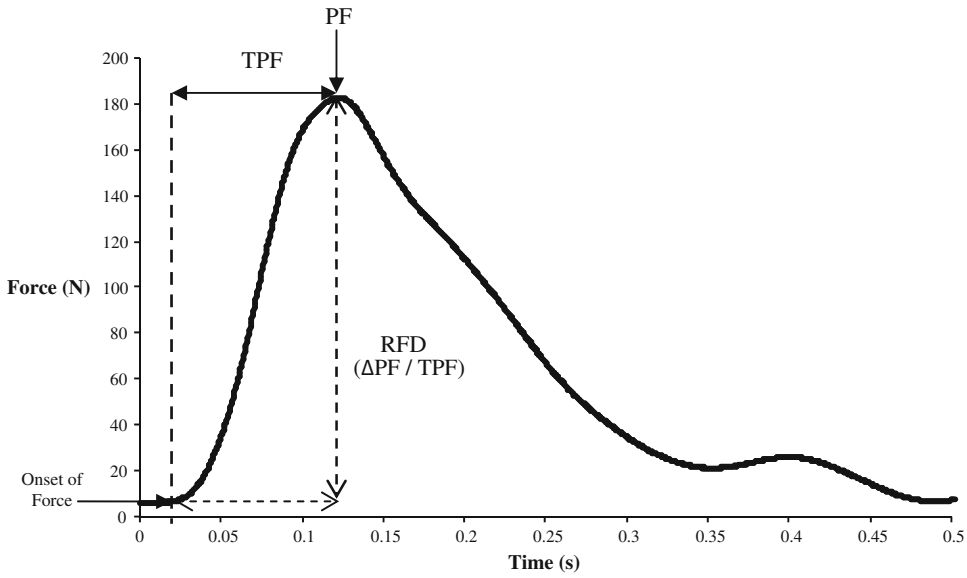

Fig. 3 A typical trace of an evoked muscle twitch response pre-intervention illustrating muscle twitch peak force (PF), time to peak force (TPF) and rate of force development (RFD)

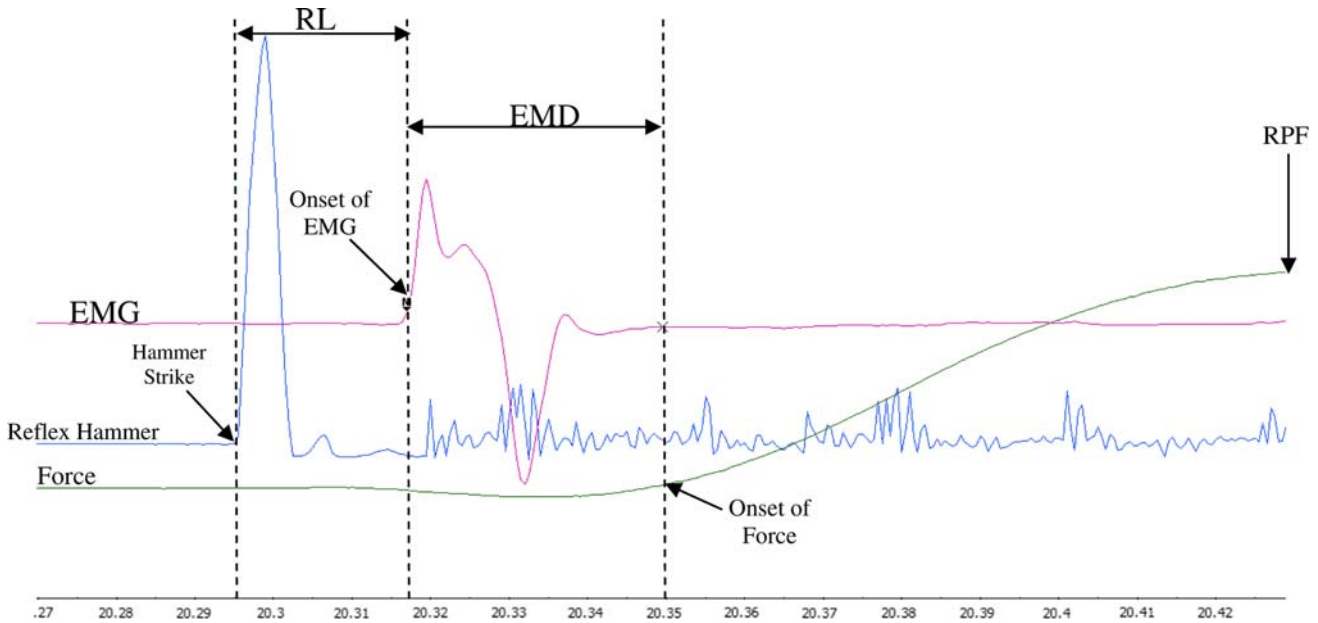

Fig. 4 A typical trace of a patellar tendon reflex pre-intervention showing electromechanical delay (EMD), patellar reflex latency (RL) and patellar reflex peak force (RPF)

WBV−, CYCL)] repeated measures ANOVA was performed to examine the magnitude and significance of these on PF, TPF, RFD, EMD, RL, RPF. For multiple comparisons significance, post hoc pairwise comparisons were performed and adjusted to Bonferroni's rule. Using pre-values intraclass correlation coefficient (ICC) assessed the day-to-day reproducibility of PF muscle twitch and patellar tendon impact strikes. All statistical analyses were performed using statistical software SPSS for Windows Version 16 (Chicago, IL, USA) and level of significance was set at $P < 0.05$.

Results

Muscle twitch peak force (PF)

There was a significant treatment, time and treatment × time interaction effect for PF. Post hoc analyses showed that 90 s after WBV + PF was significantly higher ($P < 0.01$) compared to WBV− and CYCL (Fig. 5), which equated to a 12.4% increase for WBV+, compared to 0.5% WBV−, and −1.7% CYCL. However the potentiation was not evident at post-5 and 10 min WBV+, indicating the

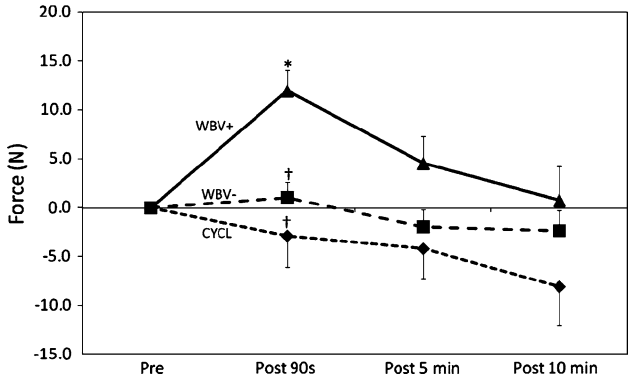

Fig. 5 Mean (±SE) change in muscle twitch peak force for WBV+, WBV− and CYCL. *$P < 0.01$ compared to pre-value, †$P < 0.01$ compared to WBV+

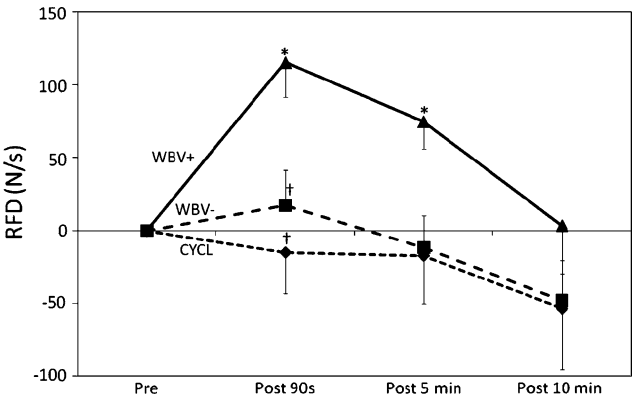

Fig. 6 Mean (±SE) change in rate of force development of muscle twitch for WBV+, WBV− and CYCL. *$P < 0.01$ compared to pre-value, †$P < 0.01$ compared to WBV+

transient nature of WBV on muscle twitch potentiation. No other significant differences over time (post-5 and 10 min) were found between the interventions (Fig. 6).

Time to peak force (TPF)

There was no treatment, time or time × treatment interaction effect for TPF (Table 1).

Rate of force development (RFD)

There was a significant treatment, time and treatment × time interaction effect for RFD (Fig. 6). Post hoc analyses revealed RFD being significantly ($P < 0.01$) higher post-90 s for WBV+ compared to WBV− and CYCL (Fig. 6), which equated to a 11.2% increase for WBV+, compared to 1.2% WBV− and −0.7% CYCL. RFD was significantly higher ($P < 0.01$) at post-90 s and post-5 min compared to pre but returned to baseline levels at post-10 min (Fig. 6). There were no significant increases in RFD at any time point for either WBV− or CYCL (Fig. 6).

Patellar tendon reflex

There was no treatment, time or time × treatment interaction effect for electromechanical delay (EMD), patellar reflex latency (RL) (Table 2) and patellar reflex peak force (RPF) (Table 1). There were no significant differences between gender for any muscle twitch or reflex activity parameters.

Reproducibility

The ICC day-to-day reliability of PF muscle twitch ($r = 0.986$) and patellar tendon impact strike ($r = 0.931$) were highly significant ($P < 0.001$), indicating that a high degree of repeatability and consistency were attained between interventions.

Discussion

The aim of this study was to compare the effect of acute WBV with static squat to that of stationary cycling and control (no WBV) on PAP from muscle twitch and patellar reflex activity. We hypothesised that 5 min of continuous acute WBV would potentiate the muscle twitch response compared to a centrally mediated effect that would potentiate WBV-induced patellar tendon reflex. Our results support our hypothesis that an acute continuous bout of

Table 1 Mean (±SE) changes in TPT and RPF for WBV+, WBV− and CYCL

	TPF (ms)			RPF		
	WBV+	WBV−	CYCL	WBV+	WBV−	CYCL
Pre	0.0	0.0	0.0	0.0	0.0	0.0
Post-90 s	−0.8 (2.0)	1.5 (2.3)	1.5 (2.3)	0.1 (0.9)	0.4 (1.3)	−1.3 (0.7)
Post-5 min	2.1 (1.9)	−5.8 (2.7)	−5.8 (2.7)	−2.8 (0.9)	−1.0 (1.2)	−2.8 (0.3)
Post-10 min	0.0 (1.9)	−7.4 (2.0)	−7.4 (2.0)	1.2 (2.6)	1.2 (1.7)	−2.8 (2.2)

TPT Time to peak muscle force, *RPF* patellar reflex peak force

Table 2 Mean (±SE) absolute EMD and RL, for WBV+, WBV− and CYCL

	EMD (ms)			RL (ms)		
	WBV+	WBV−	CYCL	WBV+	WBV−	CYCL
Pre	26.4 (3.4)	25.9 (2.9)	24.0 (1.6)	32.2 (1.8)	32.8 (1.8)	33.6 (2.0)
Post-90 s	27.0 (2.0)	25.9 (2.9)	23.4 (2.6)	30.0 (2.2)	33.2 (2.0)	33.3 (2.2)
Post-5 min	26.8 (2.8)	24.7 (2.9)	23.3 (2.5)	29.6 (2.2)	32.1 (2.0)	33.3 (2.2)
Post-10 min	26.5 (2.1)	26.1 (3.3)	27.2 (2.5)	30.3 (2.2)	32.6 (2.1)	33.7 (2.3)

EMD Electromechanical delay, *RL* patellar reflex latency

WBV at 26 Hz induces a PAP of twitch potentiation (TP) compared to WBV− and CYCL interventions.

It is reported that acute WBV improves short-term muscular performance above cycling-only exercise (Cochrane and Stannard 2005) which may indicate that WBV has the potential to be considered as a warm-up modality prior to explosive activities such as strength, speed and power (Bazett-Jones et al. 2008; Cochrane and Stannard 2005; Cormie et al. 2006). We have shown that WBV rapidly increases intramuscular temperature, and this contributes to an augmented effect on subsequent muscular activity (Cochrane et al. 2008b). However, it must be assumed that intra-muscular temperature was similar among the different conditions in this study (Cochrane et al. 2008b), and therefore seems likely that mechanisms other than temperature caused the observed effects. One such additional phenomenon is PAP, which is referred to as an increase in muscle performance preceded by a muscle contractile activity (Sale 2002) and is assessed by twitch potentiation (TP), or reflex potentiation (RP) (Hodgson et al. 2005). To our knowledge, this is the first time following WBV that TP and RP were simultaneously measured. The results showed that WBV+ increased PF, RFD with no change in TPF compared to WBV− and CYCL. This concurs with previous research showing that short duration (5–10 s) isometric maximum voluntary contractions enhance PF and RFD (Sale 2002; Vandenboom et al. 1993) whereas TPF remains unchanged (Petrella et al. 1989). The time course of TP reached a maximal level 90 s after WBV+ and returned to baseline levels after 10 min, which is in agreement with other studies (Baudry and Duchateau 2004; O'Leary et al. 1997). Likewise the increase in RFD from acute WBV is supported by other studies that have reported increases in RFD following high stimulation frequencies (>100 Hz) (Abbate et al. 2000; O'Leary et al. 1997; Vandenboom et al. 1993). Hence, as a result of enhancing RFD, explosive activities such as jumping, kicking and throwing may be improved if TP is heightened (Hodgson et al. 2005; Sale 2002). By contrast, one study investigated the effect of acute WBV on muscle activation properties and found no differences in voluntary or simulated maximal rates of force rise between baseline and post-measures of the leg extensor muscle group (de Ruiter et al. 2003). However this study differs from the present one in several ways, as the authors did not include a control group and used an intermittent protocol of 5 × 1 min WBV exposures with 2 min rest between treatments. Moreover, electrical muscle stimulation in that study was in combination with voluntary contractions, whilst the twitch characteristics have been investigated without such interference in the present study.

The explanation for PAP has centred around twitch potentiation (TP) and reflex potentiation (RP) (Folland et al. 2008; Grange et al. 1995; Hodgson et al. 2005; Sweeney et al. 1993; Trimble and Harp 1998). TP is considered to involve the phosphorylation of myosin regulatory light chains making actin and myosin more sensitive to the intracellular Ca^{2+} signal (Moore and Stull 1984; Sweeney et al. 1993; Zhi et al. 2005). This would result in greater cross-bridge interaction for the same intracellular Ca^{2+} concentration, which in turn increases the muscle tension for the same absolute level of neural stimulus (Metzger et al. 1989). However if calcium homeostasis was involved, then one would expect a disproportionate change in RFD and PF, yet the increase in RDF and PF 90 s post-WBV+ were similar (11.2% RFD, 12.4% PF) suggesting that PAP may be prevalent according to the cross-bridge cycle model (Brenner and Eisenberg 1986).

Conversely, RP is thought to elicit reflex activity in the spinal cord, by increasing synaptic efficacy between Ia afferent terminals and α motoneurones of the muscle (Hodgson et al. 2005; Trimble and Harp 1998). WBV is thought to elicit muscle contractions through spinal reflexes (Cardinale and Bosco 2003; Cochrane et al. 2009; Rittweger et al. 2003). However, and in contrast to other exercise modalities, stretch and H-reflexes are typically suppressed during isolated muscle vibration (Arcangel et al. 1971; de Gail et al. 1966). It should be considered that WBV is a combination of muscle vibration and voluntary exercise contractions, and the ambiguous findings of either increased (Melnyk et al. 2008; Rittweger et al. 2003), or unchanged (Hopkins et al. 2008) reflex activity in response to WBV may be due to the complexity of this intervention. Importantly, and in agreement with the findings of Hopkins et al. (2008), we found no changes in reflex magnitude or

latency. Therefore, our findings suggest that TP from WBV may be more important than RP. Further, it should be noted that using a tendon tap to elicit a stretch reflex response does not necessarily provide a comprehensive measure of spindle sensitivity or its contribution, as there are several factors from which the stretch reflex can be modified either by the excitatory or inhibitory elements of the motoneurone pool and/or the mechanical sensitivity of the muscle spindle itself (Hopkins et al. 2008). Given these reservations, we have reported that an acute bout of continuous WBV does not elicit a RP, which is in disagreement with the speculated proposal that changes in muscle performance from WBV are in part caused by neurogenic factors from possible changes in muscle spindle sensitivity (Cardinale and Bosco 2003; Cardinale and Rittweger 2006).

There were no TP or RP effects observed in WBV− and CYCL even though muscle temperature was indirectly matched between the interventions. An explanation for this finding may be due to the force levels being too small to elicit increases in TFP and RFD. It is well documented that heavy pre-loading exercise regimes such as 3–5 repetition maximum (RM) or MVCs are often implemented prior to short-term activities to induce PAP (Clark et al. 2006; Gourgoulis et al. 2003; Young et al. 1998). Therefore it is plausible that the pre-loading activity of WBV− and CYCL are needed to be of higher magnitude to elicit TP and RP, compared to WBV+.

This study has two limitations. Firstly, there is a possibility that the series- or parallel-elastic elements were affected in different ways by the three interventions. However it is unlikely, given that the forces required to reduce musculo-tendinous compliance, such as in stretching, are normally much greater than those used in this study (Morse et al. 2008). Moreover, in the hypothetical case of increased musculo-tendinous compliance one would have expected an increase in EMD, which was not observed in this study (see Table 1). Secondly, the time frame to detect post-reflex responses also needs to be considered (Hopkins et al. 2008). In the current study it took approximately 90 s to initiate the first patellar reflex measurement, due to the time it took to re-connect leads and re-position the participant in the chair apparatus. But this time remains in agreement with other studies that have shown increases in muscular performance (Bosco et al. 1999; Cochrane and Stannard 2005; Cochrane et al. 2008b; Torvinen et al. 2002). It has been shown that a potentiated patellar reflex dissipates after 10–20 s following exhaustive WBV (Rittweger et al. 2003), so it is possible that any RP may have been undetected.

In conclusion, we measured the neuromuscular response of muscle twitch and patellar reflex characteristics, and our results suggest that acute WBV causes PAP mainly downstream from the neuromuscular junction. Muscular performance studies have shown that PAP is induced by heavy pre-load activity which enhances muscular activity (Gourgoulis et al. 2003; Young et al. 1998). Likewise, WBV is also capable of inducing high gravitational loads through vibration frequency and amplitude that act as a pre-loading movement to activate PAP. The augmented TP of the current study may have resulted from an increase in muscle activity due to the vibrations being dampened by the muscles (Ettema and Huijing 1994; Wakeling et al. 2002), which may have caused cross-bridge cycling to increase (Cardinale and Wakeling 2005). However it is untested whether phosphorylation of myosin regulatory light chains and cross-bridge attachment are mediated by WBV and requires confirmation before any conclusion can be drawn on whether WBV elicits TP via this mechanism.

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