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Effect of whole body vibration frequency on neuromuscular activity in ACL-deficient and healthy males

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ABSTRACT: Whole-body vibration (WBV) has been shown to enhance muscle activity via reflex pathways, thus having the potential to contrast muscle weakness in individuals with rupture of the anterior cruciate ligament (ACL). The present study aimed to compare the magnitude of neuromuscular activation during WBV over a frequency spectrum from 20 to 45 Hz between ACL-deficient and healthy individuals. Fifteen males aged 28±4 with ACL rupture and 15 age-matched healthy males were recruited. Root mean square (RMS) of the surface electromyogram from the vastus lateralis in both limbs was computed during WBV in a static half-squat position at 20, 25, 30, 35, 40 and 45 Hz, and normalized to the RMS while maintaining the half-squat position without vibration. The RMS of the vastus lateralis in the ACL-deficient limb was significantly greater than in the contralateral limb at 25, 30, 35 and 40 Hz ($P<0.05$) and in both limbs of the healthy participants (dominant limb at 25, 30, 35, 40 and 45 Hz, $P<0.05$; non dominant limb at 20, 25, 30, 35, 40 and 45 Hz, $P<0.05$). The greater neuromuscular activity in the injured limb compared to the uninjured limb of the ACL-deficient patients and to both limbs of the healthy participants during WBV might be due to either augmented excitatory or reduced inhibitory neural inflow to motoneurons of the vastus lateralis through the reflex pathways activated by vibratory stimuli. The study provides optimal WBV frequencies which might be used as reference values for ACL-deficient patients.

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INTRODUCTION

Rupture of the anterior cruciate ligament (ACL) is a common sports-related injury, which requires appropriate rehabilitation protocols for the athlete's complete recovery [1]. Persistent weakness of the knee extensor muscles has been reported as one of the major issues in rehabilitation following ACL injury [2]. The exact mechanisms underlying the loss of muscle strength due to ACL rupture, however, are unclear [3]. An important factor contributing to weakness is a failure in voluntary activation of the knee extensors despite no structural damage to the muscle or innervating motoneurons [4]. It is thought that abnormal afferent discharge from the knee may alter the excitability of reflex pathways within the spinal cord, which in turn would reduce the excitability of the knee extensors' α-motoneuron pool by preventing supraspinal centres from fully activating the muscle [4]. A number of interventions based on traditional overload techniques involving active either open or closed kinetic chain exercises have been carried out to contrast muscle weakness in ACL-deficient patients [5,6]. However, it has been argued that active exercises may not be effective to target neuromuscular mechanisms underlying muscle weakness and, hence, to achieve the patient's complete recovery of strength [5,7].

Whole-body vibration (WBV) has slowly emerged as an alternative method of neuromuscular overload to enhance physical training, due to previous reports of improved strength in the lower limb muscles after vibratory exercise [8,9,10,11]. In three studies from different authors [12,13,14], WBV exercises after ACL reconstruction were proven to be effective for achieving complete recovery of neuromuscular control, although the neural mechanisms underlying such improvement remain elusive [15]. Enhanced muscle contraction during WBV has been demonstrated to be evoked via the stretch reflex pathway [16,17]. Acute changes in motor output, in fact, have been associated with increased sensitivity of muscle spindles, which would lead to facilitation in homonymous α motoneurons [8]. The neuromuscular response to WBV has been shown to depend on the type, frequency, amplitude and duration of the oscillatory stimulus as well as on the body position on the vibration platform [18,19,20,21]. Among all of these factors, vibration frequency has received increased attention as it appears to have an important effect on the magnitude of the neuromuscular response [13,18,21]. Cardinale and Lim [18], for instance, observed a gradual rise in neuromuscular activation of the vastus lateralis muscle up to 30 Hz, which was followed by a gradual decrease in activation as WBV frequency increased. Based on these findings, it has been suggested that WBV frequency should be individualized to the optimal value corresponding to the maximal amplitude of muscle activity, in order to fully maximize the excitatory inflow to motoneurons and, hence, optimize the training stimulus to the neuromuscular system [9,22]. WBV at optimal frequencies, therefore, may have the potential to specifically target neuromuscular mechanisms underlying the injury-related muscle weakness in ACL-deficient patients by maximizing the stretch reflex contribution to the overall motor output. To the best of the authors' knowledge, however, there are no studies investigating the lower limb muscle activity in ACL-deficient patients during exposure to WBV at different frequencies.

Therefore, the purpose of the present study was to compare the magnitude of neuromuscular activation between ACL deficient and healthy individuals during WBV over a frequency spectrum from 20 to 45 Hz. As it has been suggested that exposure to WBV would increase muscle activity via reflex pathways, it was hypothesized that the WBV stimulus would enhance neuromuscular activity in both ACL-deficient and healthy participants.

MATERIALS AND METHODS

Participants. Fifteen male patients (age: 28.1 ± 3.8 years; height: 1.74 ± 0.1 m; body mass: 68.3 ± 10.3 kg) with unilateral isolated ACL rupture were recruited to participate in the study. Inclusion criteria were occurrence of ligament rupture in the dominant limb from 30 to 60 days before testing and full range of motion of the knee joint. Exclusion criteria were concomitant injury to any other knee ligament or lower limb muscle, associated meniscus tear, and previous surgery on either knee. Fifteen healthy and physically active male volunteers (age: 28.8 ± 3.5 years; height: 1.78 ± 0.1 m; body mass: 73.7 ± 12.2 kg), with no disorder or history of knee injury, served as the control group. None of the subjects were experienced with WBV training. With approval of the local Ethics Committee, the study was carried out in accordance with the Declaration of Helsinki; informed consent was obtained from all participants.

Experimental procedures

The participants were exposed to synchronous vertical oscillations at 2-mm peak-to-peak amplitude using a WBV platform (NEMES Double-Vibe; BoscoSystem Technologies, Rieti, Italy). Each subject stood barefoot on the platform to eliminate any damping of mechanical oscillations that could be due to footwear. During the exposure to WBV, participants were asked to maintain a static half squat position with an angle of 60° at the knee joint (full extension: 0°) and to distribute their weight evenly over the forefoot and hindfoot bilaterally. The knee joint angle was checked with a goniometer prior to administration of WBV. The following frequencies were administered to participants in a continuous incremental order: 0 (no vibration), 20, 25, 30, 35, 40, 45 Hz. The increase in frequency occurred in steps of 5 seconds with a total duration of 30 seconds of WBV preceded by 5 seconds in the static position without vibration. Throughout the exposure to vibration, the investigators made sure that the participants' trunk did not lean laterally, the knee angle was held constant and the heels were not raised from the platform.

Two self-adhesive silver/silver chloride electrodes, with a diameter of 4 mm (Blue Sensor Ag/AgCl type NF-00-S/12, Ambu A/S, Ballerup, Denmark), were placed over the vastus lateralis muscle of both limbs with a 20 mm inter-electrode distance according to current recommendations [23]. This muscle was considered to be representative of the knee extensors muscle group, as in previous studies [24,25,26]. Before applying the electrodes, the skin was shaved and gently abraded with fine sandpaper. Medical adhesive tape and an elastic band were used to fix the sEMG cables to the skin in order to minimize any motion artefacts that could be encountered during the vibration. The sEMG cables included a pre-amplifier (gain: 1k) and a Butterworth band-pass filter (cut-off frequencies: 8-600 Hz). Signals were then full-wave root mean square (RMS) converted with an averaging time constant of 100 ms and then sampled at 100 Hz using a portable EMG system (MuscleLab 4020e, Ergotest Technology AS, Langesund, Norway), as previously described [9,18]. Test-retest reliability of the sEMG measurements during the continuous incremental WBV protocol has been previously shown to be 0.90 [27].

Data analysis and statistics

Average RMS was computed off-line for each frequency condition (0, 20, 25, 30, 35, 40, and 45 Hz) as the mean value of a central 4-second window within the available 5 seconds. The RMS values of each WBV frequency from 20 to 45 Hz were then normalised for the RMS value obtained at 0 Hz and expressed as percentages [20]. In all participants, the optimal vibration frequency (OVF) was identified as the WBV frequency with the highest normalized RMS value for each limb.

All data were normally distributed in terms of skewness and kurtosis (all values $<|2|$). Statistical comparisons of the data (normalized RMS) between frequencies (20, 25, 30, 35, 40, and 45 Hz), groups (control and ACL group) and limbs (dominant and non dominant in healthy participants; injured and uninjured in the ACL-deficient patients) were carried out by two-way ANOVA for repeated measures followed by post-hoc Student's t-test with Bonferroni correction. Statistical significance levels were set at $P < 0.05$. Unless otherwise specified, data were presented as mean ± standard error of the mean.

RESULTS

The ANOVA showed a significant main effect of group ($F = 32.1$, $P < 0.01$), limb ($F = 13.9$, $P < 0.01$) and frequency ($F = 54.2$, $P < 0.01$), as well as significant group × limb ($F = 5.8$, $P < 0.05$),

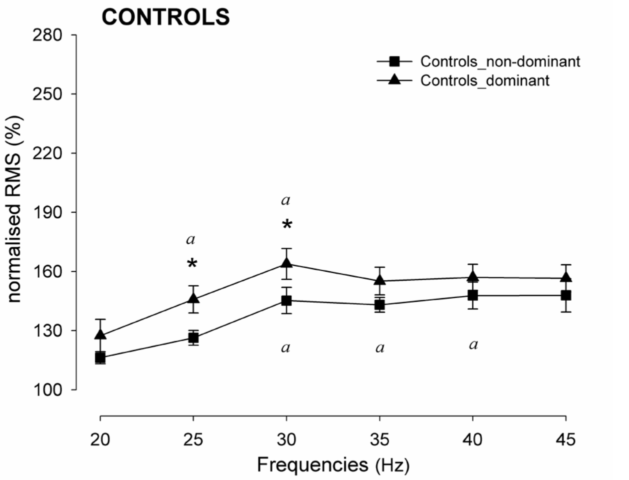

FIG. 1. Normalized RMS of the vastus lateralis muscle at 20, 25, 30, 35, 40 and 45 Hz in both the dominant (triangles) and non dominant (circles) lower limb of the healthy participants. Data (mean±SE) are reported as percentage of RMS at 0 Hz. * = Significantly different from non dominant limb ($P < 0.05$); a = Significantly different from 20 Hz ($P < 0.05$).

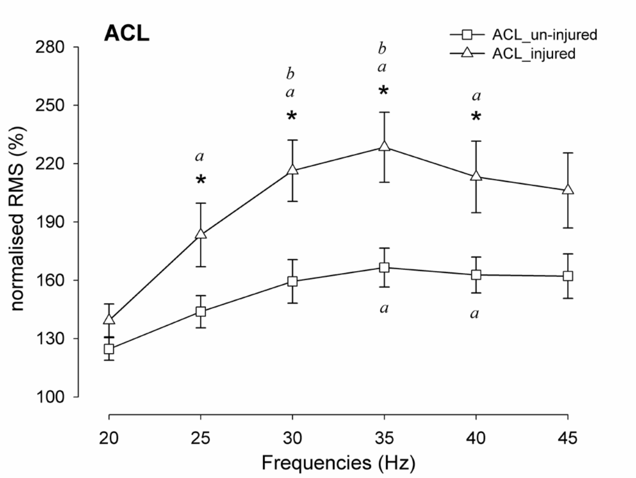

FIG. 2. Normalized RMS of the vastus lateralis at 20, 25, 30, 35, 40 and 45 Hz in both the injured (circles) and the uninjured (triangles) lower limb of the ACL-deficient patients. Data (mean±SE) are reported as percentage of RMS at 0 Hz. * = Significantly different from uninjured limb ($P < 0.05$); a = Significantly different from 20 Hz ($P < 0.05$); b = Significantly different from 25 Hz ($P < 0.05$).

TABLE 1. Optimal vibration frequency in the injured and uninjured limb of the ACL-deficient patients and in the dominant and non dominant limb of the healthy participants

		Optimal vibration frequency	
		Median (Hz)	Interquartile range (Hz)
ACL	Injured	35	35-45
	Uninjured	35	30-45
CONTROL	Dominant	30	30-40
	Non-dominant	30	30-40

Note: Data are reported as median and interquartile range. Each participant maintained a static half squat position on a WBV platform and was exposed to synchronous vertical oscillations at 2-mm peak-to-peak amplitude.

group × frequency ($F = 12.7$, $P < 0.01$) and limb × frequency ($F = 3.8$, $P < 0.01$) interactions for the normalized RMS of the vastus lateralis muscle.

Figure 1 shows the normalized RMS of the vastus lateralis muscle in both limbs of the control group for each WBV frequency. The post-hoc analysis revealed that, in healthy participants, RMS of the dominant limb was higher at 25 and 30 Hz compared to 20 Hz, while RMS of the non dominant limb was higher at 30, 35 and 40 Hz compared to 20 Hz. Moreover, the RMS was significantly higher in the dominant limb with respect to the non dominant limb at 25 and 30 Hz.

Figure 2 shows the normalized RMS of the vastus lateralis muscle in both limbs of the ACL group for each vibration frequency. The post-hoc analysis revealed that, in ACL-deficient patients, the RMS of the injured limb was higher at 25, 30, 35, and 40 Hz compared to 20 Hz, and at 30 and 35 Hz compared to 25 Hz, while RMS of the uninjured limb was higher at 35 and 40 Hz compared to 20 Hz. Moreover, the RMS was significantly higher in the injured limb with respect to the uninjured limb at 25, 30, 35 and 40 Hz.

The post-hoc analysis also revealed that the RMS of the injured limb in ACL-deficient patients was significantly higher with respect to the dominant limb of healthy participants at 25 Hz (183.32 ± 63.39% vs 145.88 ± 26.77%), 30 Hz (216.37 ± 61.05% vs 163.87 ± 30.30%), 35 Hz (228.44 ± 69.80% vs 155.15 ± 27.12%), 40 Hz (213.32 ± 74.03% vs 157.03 ± 25.58%) and 45 Hz (206.97 ± 64.69% vs 156.55 ± 26.48%), and with respect to the non dominant limb of healthy participants at 20 Hz (139.34 ± 32.76% vs 116.31 ± 11.98%), 25 Hz (183.32±63.39% vs 126.41 ± 14.46%), 30 Hz (216.37 ± 61.05% vs 145.28 ± 25.79%), 35 Hz (228.44 ± 69.80% vs 143.10 ± 14.49%), 40 Hz (213.32 ± 74.03% vs 147.81 ± 26.38%) and 45 Hz (206.97 ± 64.69% vs 147.91 ± 32.64%).

Visual inspection of Table 1 shows that median values of either limb's OVF differed between groups, being 30 Hz in healthy participants and 35 in ACL-deficient patients. The ANOVA, however, did not show any significant effect of either group or limb on OVF.

DISCUSSION

The main finding of the present study was that the magnitude of activation in the vastus lateralis muscle during WBV was greater in the injured limb of ACL-deficient patients than in the uninjured limb and in both limbs of healthy participants.

In the healthy participants and in the uninjured limb of ACL-deficient patients, the enhanced neuromuscular activation of the vastus lateralis muscle during WBV with respect to 0 Hz (i.e. no vibration) is in agreement with the results of previous studies on both locally applied [28] and whole-body vibratory stimuli [18,21]. Local vibration of 10 to 200 Hz on a muscle belly or a tendon has been shown to elicit the so-called tonic vibration reflex, wherein motoneuron excitation may be mainly attributed to activation of the primary endings of muscle spindles [16]. Similarly, exposure to WBV during squatting is believed to evoke muscle contraction mainly via the stretch reflex pathway [8,17]. The parabolic relationship between WBV frequency and vastus lateralis muscle activity, with the latter peaking on average at 30-35 Hz in healthy participants and in the uninjured limb of ACL-deficient patients, is in agreement with previous reports on healthy individuals [9,18,27]. As previously argued by others [9,18,27], this might be due to the initial enhancement of the excitatory inflow up to 30-35 Hz followed by a gradual predominance of the inhibitory inflow to the motor output at higher frequencies.

In the injured limb of ACL-deficient patients, neuromuscular activation of the vastus lateralis muscle was remarkably greater than in the contralateral uninjured limb and in both limbs of healthy participants throughout the WBV frequency spectrum from 20 to 45 Hz. Based on previous findings suggesting that WBV would increase muscle activity via the stretch reflex pathway [16,17], it is reasonable to argue that the enhanced muscle activity in the ACL-deficient limb may reflect either an increased excitatory inflow or a decreased inhibitory inflow to the net sum of neural influences acting on the stretch reflex pathway of the vastus lateralis muscle. During WBV, the mechanical oscillatory stimuli would elicit simultaneous activation of neuromuscular spindles as well as other joint, skin and vestibular receptors [8]. A number of excitatory and inhibitory influences of both central and peripheral origin, hence, may impact on the stretch reflex pathway to modulate the magnitude of neuromuscular activation recorded in the vastus lateralis muscle during WBV. In healthy limbs, appropriate levels of muscle stiffness are preserved in an attempt to dampen the vibratory waves by regulating such reflex muscle activity, mainly through the γ-muscle spindle system [8]. However, this may not be the case in ACL-deficient limbs, as abnormal efferent activity of the γ motoneurons serving the knee extensors has been reported following ACL injury [4,29]. It is thought that the injury-related lack of afferent information from mechanoreceptors in the ACL may disrupt γ efferent activity and, in turn, lead to abnormal γ-loop sensitivity [4]. Alternatively, or perhaps concurrently, an increase in the discharge of group II and III knee afferents, which is due to the greater translation of joint surfaces in the ACL-deficient knee with respect to the healthy one during WBV, may contribute to the net excitatory effect on both γ and α motoneurons [30]. Yet, the possibility of central modulation of stretch reflex excitability cannot be ruled out, as previous studies have provided direct evidence for the contribution of supraspinal centres in the spinal modulation of the stretch reflex gain in the knee extensor muscles [31,32]. Irrespective of whether such modulation of stretch reflex excitability is due to either central or peripheral mechanisms, any increase in reflex-induced muscle activity during WBV may represent an effective way to enhance the motoneuron output despite the diminished rate of voluntary activation of the knee extensors in the ACL-deficient limb.

The WBV frequency corresponding to the maximal EMG amplitude of the vastus lateralis muscle, referred to as OVF, did not differ between the ACL-deficient group and healthy participants and occurred on average at 30-35 Hz. In healthy individuals, it has been suggested that providing a vibratory stimulus at the optimal frequency is essential in order to fully maximize the excitatory inflow to motoneurons and, hence, to have a positive effect on the neuromuscular system [8,18,33]. Nevertheless, in the ACL-deficient individuals it is uncertain whether vibrating at optimal frequency has positive or negative effects on the neuromuscular system due to the lack of studies aimed at evaluating muscle performance in ACL-deficient patients following WBV at different frequencies. Therefore, future studies should address both acute and chronic effects in ACL-deficient patients exposed to different WBV frequencies.

CONCLUSIONS

In conclusion, the present study showed that WBV stimuli led to greater neuromuscular activation of the vastus lateralis muscle in the injured limb of ACL-deficient patients compared to the contralateral uninjured limb over a frequency spectrum from 25 to 40 Hz and to both limbs of healthy participants (dominant limb over the spectrum from 25 to 45 Hz and non dominant limb over the spectrum from 20 to 45 Hz). This might be attributed to a reflex-mediated increase in excitatory influences on the motoneurons of the vastus lateralis muscle during WBV, which may represent a compensatory mechanism aimed at overcoming the diminished rate of voluntary activation of the knee extensors in the ACL-deficient limb. Further studies are warranted to highlight the neural mechanisms underlying the differences in the magnitude of neuromuscular activation in the knee extensors between ACL-deficient and healthy participants during WBV.

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