SPINAL REFLEX EXCITABILITY AND HOMOSYNAPTIC DEPRESSION AFTER A BOUT OF WHOLE-BODY VIBRATION

KRISTOF KIPP, PhD,[1] SAMUEL T. JOHNSON, PhD,[2] JEFFREY R. DOERINGER, MS,[2] and MARK A. HOFFMAN, PhD[2]

[1] Department of Physical Medicine and Rehabilitation, University of Michigan, 325 East Eisenhower, Ann Arbor, Michigan 48109, USA
[2] Department of Nutrition and Exercise Sciences, Oregon State University, Corvallis, Oregon, USA

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ABSTRACT: Although whole-body vibration (WBV) affects neuromuscular performance, it remains unclear whether the effects are due to spinal reflex potentiation or inhibition, or differ between muscle groups. This study aimed to identify the effect of WBV on measures of spinal reflex excitability (H-reflex) and homosynaptic depression (HD) in the soleus (SOL) and medial gastrocnemius (MG) muscles. H-reflex and HD measurements were made in the SOL and MG muscle of 20 participants before and after a bout of WBV. H-reflex and HD were measured every 15 seconds for 10 minutes post-WBV and averaged at 1-minute increments. H-reflex amplitude was depressed for the first minute post-vibration, whereas the effect of HD was reduced for the first 2 minutes post-vibration. WBV significantly decreases spinal reflex excitability and HD, but it does so transiently and independent of muscle group.

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Whole-body vibration (WBV) training is a popular technique used by individuals in an attempt to enhance neuromuscular performance.[1–3] WBV has been shown to affect strength, balance, and power after both short-term[4–8] and repeated exposure.[9–12] In addition to use in the sports environment,[13] WBV is also being used in rehabilitation settings.[14–16] Despite its rise in popularity, the mechanisms responsible for the changes seen with WBV exposure remain unidentified.[1]

The primary mechanism that has been proposed to account for the observed changes in motor output after WBV is potentiated reflex activity of the Ia afferent–motoneuron synapse.[5,6] However, recent work has demonstrated that reflex activity either remains unchanged or decreases after a bout of WBV.[4,17,18] Although these findings contradict the assertion that improved functional outcomes are due to potentiation of reflex activity, it often remains overlooked that the net changes in reflex activity may be driven by a variety of mechanisms that serve to integrate sensorimotor information at the spinal level.[19] One such mechanism is rate-dependent modulation of the Ia afferent–motoneuron synapse arising from prior activation of homonymous muscle afferents. Referred to as homosynaptic depression (HD), this modulation acts as a presynaptic mechanism within the Ia afferent–motoneuron synapse that regulates the effectiveness of afferent feedback.[20,21] Although it has been proposed that altered muscle activation after WBV could be partially modulated by HD,[2] this hypothesis has not been previously tested. A study of the changes in spinal reflex excitability and HD after WBV would provide mechanistic insight into sensorimotor integration at the spinal level after short-term WBV exposure.

Another question related to neural effects of WBV is whether muscles of different fiber types are affected to a similar extent. Armstrong and colleagues[17] observed distinct recovery patterns of spinal reflex excitability of the soleus muscle in individuals after WBV and hypothesized that individual differences in muscle fiber composition may have accounted for the observed patterns. However, muscles that consist of different fiber types also vary in the number of muscle spindles.[22] Considering that the number of muscle spindles may affect the function of the Ia afferent–motoneuron synapse and affect either spinal reflex excitability and/or HD, the response to WBV may not be homogeneous across muscles. Measuring spinal reflex excitability and HD in muscles with known differences in fiber type composition and numbers of muscle spindles, such as the soleus and medial gastrocnemius,[22,23] would help test this hypothesis.

The purpose of this study was therefore twofold: (1) to identify the effects of WBV on spinal reflex excitability and HD; and (2) to examine how different muscles respond to WBV with regard to the aforementioned measures over time.

METHODS

In order to identify the effects of WBV on spinal reflex excitability and HD we measured the amplitude of H-reflexes and calculated the ratio between two successive stimuli in the soleus (SOL) and medial gastrocnemius (MG) of participants during quiet standing before and after a brief bout of WBV. Twenty (11 men, 9 women) recreationally active, healthy adults (27.4 ± 4.4 years), with no known neurological deficits, were recruited for this study. All participants provided written informed consent approved by the university institutional review board before participating in the study.

Abbreviations: ANOVA, analysis of variance; HD, homosynaptic depression; H-reflex, Hoffmann reflex; MG, medial gastrocnemius; SOL, soleus; WBV, whole-body vibration

Key words: H-reflex, pre-synaptic inhibition, neuromuscular excitability, muscle spindle, post-activation depression

Correspondence to: K. Kipp; e-mail: kristof@med.umich.edu

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Experimental Protocol. The experimental protocol consisted of the following steps: (1) elicitation of maximum M-wave and Hoffmann reflex (H-reflex); (2) determination of test H-reflex; (3) collection of pre-vibration baseline data; (4) administration of WBV intervention; and (5) collection of post-vibration reflex data.

Data Collection. Soleus and medial gastrocnemius electromyography (EMG) was recorded with surface electrodes (BIOPAC Systems, Inc., Goleta, California) at a frequency of 2000 Hz. Areas of the skin where recording electrodes were placed were shaved and cleaned with alcohol. Lubricated surface EMG recording electrodes (Ag/AgCl) were placed longitudinally along the midline of the muscle belly of the soleus and medial gastrocnemius with an interelectrode distance of 2 cm. Soleus and medial gastrocnemius reflex responses were elicited with a stimulating electrode (2 cm^2) placed over the tibial nerve in the popliteal fossa and a carbon–rubber dispersal electrode (3 cm^2) just superior to the patella on the distal thigh. The stimulating electrode was connected to a stimulator (Model S88; Grass Technologies, West Warwick, Rhode Island). Reflexes were elicited with a 1-ms square-wave pulse delivered to the tibial nerve while participants were standing quietly with normal posture (i.e., hands hanging at the side, knees slightly flexed). A partial recruitment curve was established to identify the maximum H-reflex and maximum M-wave. The test reflex intensity was set such that an H-reflex equal to 10% of maximum M-wave was evoked. This intensity remained constant for the duration of the study.

The paired pulse technique to assess HD consisted of two stimulations delivered with a 100-ms interstimulus interval. The first stimulation produced an unconditioned reflex response that was used as a measure of spinal reflex excitability (H-reflex), whereas the second stimulation resulted in a conditioned response reflective of the rate-dependent inhibition due to the reflex activation history induced by the first stimulation (Fig. 1). A 15-second interval was used between trials. To determine the amount of HD, the ratio of the conditioned to the unconditioned reflex response was subtracted from 1 and multiplied by 100% as follows:

$$\%\mathrm{HD} = [1 - (\text{Conditioned reflex response} / \text{Unconditioned reflex response})] \times 100\% \quad (1)$$

For all 10 paired stimulations, the amplitudes of the M-wave, unconditioned response (H-reflex), and conditioned response were measured in the SOL and MG. The average of the 10 trials was used as the baseline.

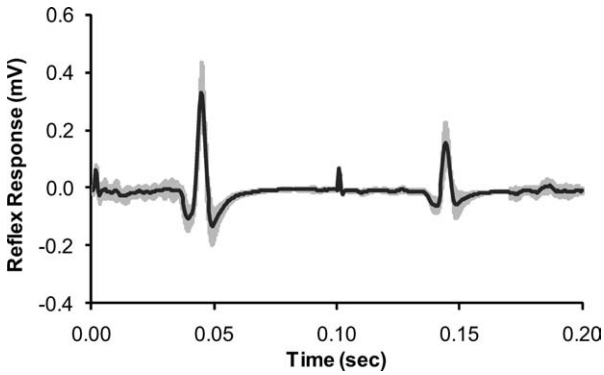

FIGURE 1. Ensemble average of 10 paired reflex recordings (thick dark line = mean, gray area = ±1 SD) for a single participant during pre-vibration data collection. The first response that occurs between 0.04 and 0.05 second is the unconditioned response (i.e., the H-reflex) and the second response that occurs 0.1 second later is the conditioned response. The percentage of homosynaptic depression is the determined from (eq. 1).

For the WBV intervention participants stood quietly, with the same posture as during the reflex recordings, on a vibration plate for 5 minutes (TurboSonic, Hood River, Oregon). The WBV plate was set to vibrate at a frequency of 25 Hz at 2–4-mm amplitude. Post-vibration data collection started immediately after the intervention and continued with reflex measurement every 15 seconds for the next 10 minutes. Post-vibration data were averaged every minute of the 10-minute monitoring period.

Statistical Analysis. Two separate 2 (muscle) × 11 (time) analyses of variance (ANOVAs) with repeated measures on the second factor were used to analyze the data. The dependent variables were the peak-to-peak amplitude of the unconditioned reflex and percent HD. The independent variables were muscle and time. The initial alpha level for significance testing was set at 0.05. Assumptions of the test statistics were verified with the Mauchly test of sphericity. Greenhouse–Geisser adjustments were made if the assumptions of sphericity were not met. Post hoc testing used paired and unpaired *t*-tests for time and muscle comparisons, respectively. Bonferroni adjustment was made to the alpha level ($\alpha_{\mathrm{Bonferroni}} = 0.005$) to account for the multiple comparisons ($n = 10$) between baseline and all respective post-WBV time intervals (e.g., Pre-Min1, Pre-Min2…Pre-Min10).

RESULTS

Spinal reflex excitability was influenced by WBV (Table 1). Although the interaction between time and muscle was not significant, significant main effects for time and muscle existed (main effect; $P < 0.05$). Specifically, H-reflex amplitude of the SOL and MG was significantly depressed (post

Table 1. Peak-to-peak H-reflex amplitude (mean ± SD) of soleus (SOL) and medial gastrocnemius (MG) before and after whole-body vibration.

	H-reflex amplitude	
Time	SOL*	MG
Pre	0.66 ± 0.30	0.23 ± 0.21
1 min	0.43 ± 0.26†	0.17 ± 0.17†
2 min	0.55 ± 0.37	0.20 ± 0.20
3 min	0.56 ± 0.31	0.20 ± 0.20
4 min	0.62 ± 0.39	0.21 ± 0.19
5 min	0.61 ± 0.39	0.18 ± 0.13
6 min	0.70 ± 0.50	0.22 ± 0.23
7 min	0.63 ± 0.36	0.19 ± 0.14
8 min	0.71 ± 0.44	0.20 ± 0.16
9 min	0.68 ± 0.40	0.20 ± 0.15
10 min	0.66 ± 0.41	0.20 ± 0.17

Pre = before whole-body vibration; 1 10 min = respective minutes after whole-body vibration.
**Significant difference vs. MG (main effect; $P < 0.05$).*
†Significant difference vs. Pre (post hoc; $P < 0.005$).

hoc; $P < 0.005$) during the first minute post-WBV. Further, H-reflex amplitude was consistently greater in the SOL than in the MG (main effect; $P < 0.05$).

The percent homosynaptic depression was also influenced by WBV (Table 2). Once again, the interaction between time and muscle was not significant; however, the analysis indicated separate significant main effects for time and muscle (main effect; $P < 0.05$). Specifically, the percentage of HD was significantly depressed in the SOL and MG (post hoc; $P < 0.005$) during the first 2 minutes post-WBV. Further, the amount of HD was consistently greater in the SOL than the MG (main effect; $P < 0.05$).

DISCUSSION

The purpose of this study was to determine whether WBV affects spinal reflex excitability and homosynaptic depression in the SOL and MG. Our results indicate that an acute bout of WBV significantly affected spinal reflex excitability and homosynaptic depression. More specifically, we observed a decrease in the peak-to-peak amplitude of the H-reflex and a decrease in the percentage of HD after WBV exposure. The depression in each of these measures was short-lasting and returned to pre-vibration baseline levels after 1 and 2 minutes, respectively. Although the finding of acute depression of H-reflex amplitude after WBV coincides with that of other investigators,[17] no previous study has examined the effects of WBV with respect to any spinal modulatory mechanism. As HD represents a pre-synaptic mechanism that regulates the function of the Ia afferent–motoneuron synapse,[20,21] a change in HD would indicate altered pre-synaptic control. A decrease in HD would thus indicate less pre-synaptic inhibition and, consequently, improve the transmission efficiency between the Ia muscle spindle afferents and the α-motoneurons.

The decreased HD may help explain the previously observed positive effects of WBV despite the decreased or unchanged levels in spinal or stretch-reflex excitability.[4,18] Several ideas lead to this conclusion. Pre-synaptic inhibition of the Ia afferent–motoneuron synapse is decreased at the onset of contraction.[24] Functionally, reduced pre-synaptic inhibition would facilitate a high reflex gain and enhance motor output during the execution of high-power ballistic movements.[24] Indeed, a recent cross-sectional study found that power-trained athletes displayed less pre-synaptic inhibition than endurance-trained athletes.[25] With that in mind, perhaps the observed improvements in motor performance following WBV are not from increased spinal reflex excitability, but from the change in HD. Although this hypothesis is speculative and remains to be tested more rigorously, it presents a novel perspective in explaining the mechanisms underlying WBV-induced performance increases.

In addition, we examined how WBV influences spinal reflex excitability and homosynaptic depression in different muscles. Our results indicate that WBV-induced changes in H-reflex amplitude and HD did not differ between the SOL and MG muscles and follow a similar time-course after WBV. Although contrary to our hypothesis, the lack of differences between muscles is an interesting result, because it is well established that these muscles differ in a number of physiological aspects. Most notably, the SOL consists of a higher percentage of slow-twitch muscle fibers and

Table 2. Percent HD (mean ± SD) of soleus (SOL) and medial gastrocnemius (MG) before and after whole-body vibration.

	Percent HD	
Time	SOL*	MG
Pre	85 ± 84	74 ± 84
1 min	77 ± 76†	59 ± 72†
2 min	78 ± 72†	55 ± 68†
3 min	83 ± 79	66 ± 76
4 min	82 ± 75	68 ± 75
5 min	88 ± 92	69 ± 77
6 min	84 ± 84	67 ± 80
7 min	86 ± 86	61 ± 71
8 min	89 ± 89	69 ± 79
9 min	89 ± 90	73 ± 82
10 min	84 ± 80	64 ± 68

Pre = before whole-body vibration; 1–10 min = respective minutes after whole-body vibration.
**Significant difference vs. MG (main effect; $P < 0.05$).*
†Significant difference vs. Pre (post hoc; $P < 0.005$)

contains more muscle spindles than the MG.[22,23] The results therefore indicate that neural responses are similar between muscles despite different fiber types and numbers of muscle spindles.

A limitation related to the conclusions in this study is that we only measured one inhibitory mechanism: homosynaptic depression. Other pre- or post-synaptic mechanisms cannot be excluded in their role in modulating spinal reflex excitability after WBV. Likewise, the potential role of supraspinal centers in modulating WBV-induced changes in neural function cannot be excluded, although cortical contributions to spinal reflex excitability and HD during standing are thought to be minimal.[20] Another limitation applies to the generalizability of our findings, because the effects of the amplitudes and frequencies used in WBV protocols may have differential effects on the neuromuscular response.[26,27] Additional research that examines neuromuscular changes after WBV is thus warranted.

In conclusion, we observed that WBV significantly depressed peak-to-peak H-reflex amplitudes during the first minute post-vibration. Further, HD decreased during the first 2 minutes post-vibration. Neither of these changes differed between muscles, as spinal reflex excitability and HD decreased equally in the SOL and MG. These results illustrate that WBV influences spinal reflex excitability and HD, but does so independent of muscle and does so only briefly. Although the changes in spinal reflex excitability do not support the common notion that WBV-induced changes in motor output occur as the result of reflex potentiation, the observed changes in HD point to a particular mechanism that may play a direct role in improving neuromuscular function after WBV.

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