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Randomised control study of oxidative stress in whole body vibration exercise

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ABSTRACT

Whole Body Vibration (WBV) causes increased blood flow and oxygen consumption, which contribute to free radical generation, yet levels of oxidative stress and antioxidant activity induced by WBV are unknown. This study compares oxidative stress of WBV with a known muscle-damaging protocol and metabolic equivalent exercise. Twenty-one untrained volunteer females (23.9 ± 1.0yr) were randomly allocated into one of three groups: Vibration training (WBV), Downhill Running (DHR) or Walking (WLK), and completed 8-weeks training of 3x20-min per week. Blood samples for Creatine Kinase, oxidative stress (F2-isoprostane), antioxidant enzyme activity of Glutathione Peroxidase (GPx) and Total Antioxidant Capacity (TAC) were collected at baseline, immediately following, and 24-hours after the first exercise session, and immediately following the final session. There was greater muscle damage in DHR ($p = 0.02$) compared with WBV and WLK at 24h post-exercise, but no difference between WLK and WBV. All groups showed increased F2-isoprostane levels immediately post-exercise ($p < 0.05$), but lower than baseline at 24-hours and 8 weeks. The WBV F2-isoprostane level changed the least and absolute levels were similar to WLK. There was a time main effect ($p < 0.001$) for GPx with activity greatest in DHR for acute and chronic responses. The TAC assay revealed immediate and 24hr post increases for DHR and WLK, but not WBV. An acute bout of WBV does not incur significant muscle damage, or oxidative stress, which could be advantageous to patient/elderly groups, but there is a GPx antioxidant training effect. Oxidative stress was highest immediately post-exercise, and WBV incurred the lowest percentage-change indicating its low damaging impact, and therefore safe use with vulnerable populations.

1. Introduction

Whole-Body Vibration (WBV) or Vibration Training has been shown to be an effective neuromuscular training tool, leading to improvements in leg muscle strength (Verschueren et al., 2004), power (Adams et al., 2009) and vertical jump performance in a wide-variety of subject cohorts under various different training regimes (Abercromby et al., 2007; Broadbent et al., 2010; Verschueren et al., 2004; Sands, McNeal, Stone, Russell, & Jemni, 2006). Moreover, research is now available about the potential for WBV as a therapeutic tool for back pain-relief (Abercromby et al., 2007; Jun Iwamoto et al., 2004), increased Bone Mass Density (Abercromby et al., 2007; Gusi, Raimundo, & Leal, 2006; Jordan, Norris, Smith, & Herzog, 2005; Verschueren et al., 2004), lumbar flexibility (Lee & Chow, 2013), elderly balance (Abercromby et al., 2007), and subsequent improvements in quality of life in the elderly (Bruyere et al., 2005).

Some effects of WBV that are seen as positive may also have an adverse effect, where the elevated blood flow is associated with WBV (Alessio et al., 2000; Broadbent et al., 2010; Rittweger, Beller, Felsenberg, & Rittweger, 2000; Sands et al., 2006), particularly an increased lower extremity blood flow (Games & Sefton, 2013; Lohman III, Petrofsky, Maloney-Hinds, Betts-Schwab, & Thorpe, 2007). Increased vasodilation leads to an increased total surface area of micro-vessels in muscles, theoretically enhancing gas exchange (Games & Sefton, 2013) and waste metabolism between blood and muscle fibres (Mester,

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Kleinöder, & Yue, 2006). However, an increased blood flow also has the potential to increase the concentration of reactive oxygen species (ROS) and therefore cause elevated oxidative stress in participants (Alessio et al., 2000), particularly those who are untrained (Moflehi, Kok, Tengku-Kamalden, & Amri, 2012). Such ROS have the potential to cause oxidative damage to a range of molecular structures including lipids, proteins and DNA, leading to inflammation and/or loss of function via cellular destruction (Urso & Clarkson, 2003).

Physical activity induces production of ROS with the elevated oxygen consumption and metabolism associated with acute aerobic (Sacheck, Milbury, Cannon, Roubenoff, & Blumberg, 2003; Urso & Clarkson, 2003) and anaerobic exercise (Alessio et al., 2000). This response is thought to be intensity dependent, with both high and moderate intensity exercise having been shown to increase lipid peroxidation (Seifi-Skishahr, Siahkohian, & Nakhostin-Roohi, 2008), an indicator of oxidative stress. Concentrations of F2-isoprostane indicate the peroxidation of esterified arachidonic acid which is an omega-6 fatty acid in the phospholipids of cell membranes. F2-isoprostane concentrations have high levels of specificity and sensitivity (Schmitz et al., 2008) as an oxidative stress marker, and it remains stable making it the most reliable and accurate measure in a wide range of fluids (Dalle-Donne, Rossi, Colombo, Giustarini, & Milzani, 2006). It is often measured in conjunction with an indirect marker of membrane damage, such as Creatine Kinase concentration, to assess levels of exercise-induced damage (Seifi-Skishahr et al., 2008). The resultant muscle damage enhances free radical production and activity, which in turn regulates antioxidant activity and synthesis.

The body has a cellular antioxidant 'reserve' through an existing level of endogenous antioxidant enzymes which are active at low levels of oxidative stress. However, antioxidant adaptation requires up-regulation of the synthesis of these enzymes which is stimulated by chronic free radical production (Allen & Tresini, 2000; Ji & Zhang, 2014). There are several measureable markers of antioxidant activity such as Total Antioxidant Capacity (TAC) and Glutathione Peroxidase (GPx) enzyme activity. The TAC is a general marker of oxidative stress by measuring low molecular weight, chain breaking antioxidants (Young, 2001), while GPx is more specific in reducing peroxide particularly that involved in lipid peroxidation of cellular membranes.

Despite evidence of elevated markers of oxidative stress with different forms of exercise and a suggestion of increased risk of oxidative stress with an increased blood flow, there is scant published research into the risk of oxidative stress posed by WBV training/exercise sessions. Theodorou et al. (2015) looked at redox activity by measuring Thiobarbituric acid (TBARS) and TAC in low trained middle-aged women. They found no increases in oxidative stress or antioxidant regulation although TBARS only measures the by-product malondialdehyde and not all lipid peroxidation end products (Dalle-Donne et al., 2006).

Understanding any possible detrimental effect of WBV is important as it is being heralded as both a training tool enhancing the strength and power of athletes (Cormie, Deane, Triplett, & McBride, 2006; Delecluse, Roelants, & Verschueren, 2003; Rønnestad, 2009a, 2009b) and also as a therapeutic exercise to improve balance (Bogaerts et al., 2007; Cheung et al., 2007; Gusi et al., 2006; Kawanabe et al., 2007), physical function (Furness & Maschette, 2009; Iwamoto, et al, 2012; Merriman, Brahler, & Jackson, 2011; Mori et al., 2006), muscular strength (Bogaerts et al., 2009; Roelants, 2004; Verschueren et al., 2004; von Stengel et al, 2012). Therefore, the present study was undertaken to investigate the effects of two different modes of acute and chronic exercise with WBV on markers of oxidative stress and antioxidant activity. It will be compared with downhill running, well-known as a muscle damaging exercise and increasing oxidative stress, and walking at a rate of metabolic equivalence as a control group.

2. Methods

2.1. Participants and Ethical Clearance

Twenty-one untrained females (Table 1) consented to take part in the study after all procedures were explained. Potential participants completed a health screening questionnaire and were excluded if they participated in any regular recreational exercise or sport training, injured, smokers, or currently taking anti-inflammatory/pain-relief medication or dietary antioxidant supplements. Additionally, all participants were instructed to refrain from taking any antioxidant supplements, and not increase their dietary vitamin C or E intake prior to or during the 8 weeks. All participants were informed both verbally and in writing about the potential risks and gave written informed consent for their participation in this study, which was approved by the Institute's Human Ethics Committee; Southern A; 10/04; approved 05.03.2010.

2.2. Experimental Design

Blinded to experimental conditions, participants randomly self-selected one of three training groups; Whole body vibration training (WBV), Downhill Running (DHR), or Walking (WLK) group. Each participant completed an eight-week training program, comprising of 3x20-min supervised sessions of exercise per week, at the same time of day for individuals in order to negate circadian rhythm effects. The DHR and WLK exercises were conducted on a treadmill, with all exercises conducted within a laboratory. Sessions were separated by a minimum of 24-hours. Morning baseline venous blood samples were collected prior to the first exercise session, immediately following and 24-hours after the first exercise session (acute measures), and immediately after the final exercise session (chronic measures). Venous samples were also collected for subsequent analysis of markers of muscle damage and oxidative stress.

The WBV exercise was delivered in 10x1-min bouts in a ratio of 1 min:1 min (WBV:rest) totalling 20 minutes at 26 Hz (maximum frequency of the machine) using a sinusoidal vibration trainer (Galileo® Control 0544; Novotec Medical GmbH, Pforzheim, Germany) with knees set at a 20° angle measured by goniometer (Baseline Instruments, Auckland, New Zealand) to dampen the vibration from reaching the brain level.

Shoeless feet were placed in a standardised position on the machine (middle toe on either foot aligned to 18cm from the central axis) to deliver an amplitude (A) of ~6.8mm and 3.02g peak angular acceleration. The DHR parameters were 20-min continuous treadmill running at a -10.5% (6°) gradient and a self-selected speed of between 9–12 km·h-1, which participants voluntarily increased over the 8 weeks up to the maximum 12 km·h-1. Walking sessions consisted of 20-min continuous treadmill walking at 4.5 km·h-1, which equated to equivalent METs for WBV at 26 Hz; A = 6mm (Rittweger et al., 2000; Rittweger, Schiessl, & Felsenberg, 2001), at neutral angle i.e. with no incline or decline.

2.3. Protocol

On the first visit to the lab, body mass and height was measured using electronic scales (model UC-321; A&D Co. Ltd., Tokyo, Japan) and a standard laboratory stadiometer (model 26SM; Surgical and Medical Products, Seven Hills, NSW, Australia), details of participants are shown Table 1. A cannula (Becton-Dickinson Ltd., Oxford, UK) was inserted into an antecubital vein, and initial blood sample drawn; 6ml was collected in serum separation (SST) tubes for CK analysis and 8-iso-Isoprostane (referred to as F2-isoprostane) analysis (Becton-Dickinson Ltd., Oxford, UK), while 4ml in EDTA tubes were used for, Total Antioxidant Capacity (TAC as μM Trolox equivalents) and Glutathione Peroxidase activity (GPx, U/L). Blood samples in EDTA tubes were spun immediately in a centrifuge (Medifuge; Heraeus Sepatech, Berlin, Germany) for 10 min at 1000 g before blood plasma samples were divided into two 1.5ml aliquots and frozen at -80°C for further analysis. Blood samples in SST tubes were left to stand for 30 min prior to being spun in a centrifuge for 20 min at 3000 g, after which blood serum samples were also divided into two 1.5ml aliquots and frozen at -80°C.

2.4. Blood Analysis

Lipid peroxidation was assessed using a commercially available immunologic assay ELISA kit (8-iso-Prostaglandin F2α ELISA Kit; Cell Biolabs, Inc., San Diego CA, USA) to measure F2-isoprostane levels in plasma. These kits are sensitive to detect between 49 pg/mL and 2000,000 pg/mL of 8-isoprostane, where normal human plasma baseline is 40-100 pg/mL. The absorbance for the standard curve and samples were read at 450 nm (Benchmark Plus Microplate Spectrophotometer, Biorad Laboratories Pty Ltd, Auckland, New Zealand). Creatine Kinase (CK) activity was assayed using standard enzyme colourimetric analysis (Roche/Hitachi cobas c system TCK, Roche, Mannheim, Germany) with absorbance measured at wavelength 546/340 nm. The lower and upper limits for detection were 3 U/L 2000 U/L respectively where U/L x 0.0167 = μkat/L. Both F2-isoprostane and CK levels were presented as units per litre (U/L).

Total Anti-oxidant Activity was measured using a colourmetric ELISA kit (QuantiChromTM Antioxidant Assay kit; BioAssay Systems, California, USA). In a 20 μl sample detection ranges from 1.5-1000 μM Trolox equivalents with absorbance measured at wavelength 570 nm. Glutathione Peroxidase (GPx) enzyme activity was measured by colourimetric assay (EnzyChromTM EGPx-100; BioAssay Systems, CA, USA) with a detection range of 12-300 U/L GPx activity per 10 μL sample. Samples were read at wavelength 340nm.

2.5. Statistical Analysis

Creatine Kinase activity, F2-isoprostane levels, and percentage changes from baseline for F2-isoprostane, TAC and GPx were compared between groups at the different measurement points via a Repeated Measures ANOVA (group x measurement time point). Bonferroni Post-hoc tests were carried out if main effects were identified. Paired analysis between the final and immediately post values of each participant was performed to determine any chronic training effects. Significance was set at 5%, and results are presented as mean ± standard error mean. Treatment effect-sizes were calculated in accordance with Cohen's d (Cohen, 1988), with clinical-importance inferred at Moderate (0.5-0.8) and Large (0.8+) effect-sizes in accordance with Page (Page, 2014).

3. Results

There were no statistical differences between groups for age or anthropometric measures, as shown in Table 1.

Table 1: Anthropometric measures of participants, and group distribution. Group and overall means ± SEM are reported for anthropometric measures.

	n	Age (decimal years)	Height (m)	Weight (kg)	BMI ($kg \cdot m^{-2}$)
Downhill Running	6	26.4 ± 2.5	1.63 ± 0.03	63.7 ± 3.7	24.0 ± 1.2
Vibration Training	8	22.6 ± 1.4	1.63 ± 0.03	67.3 ± 5.4	25.0 ± 1.5
Walking	7	23.2 ± 1.3	1.69 ± 0.01	78.6 ± 6.9	27.7 ± 2.6
Overall	21	23.9 ± 1.0	1.65 ± 0.02	70.1 ± 3.5	25.6 ± 1.2

The Creatine Kinase from DHR was significantly elevated compared with WBV (p = 0.034) and WLK (p = 0.013) at 24h post exercise, but not between WLK and WBV (Figure 1). As expected, a large effect size of 2.02 was observed at 24h post exercise with DHR, while WBV and WLK had small effect sizes (0.13 and 0.04, respectively).

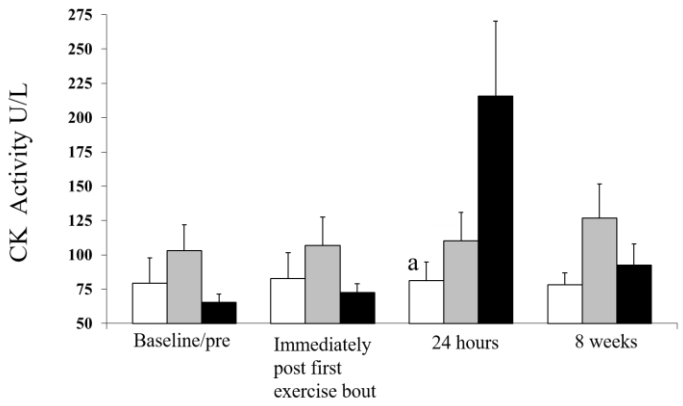

Figure 1: Mean ± SEM for Creatine Kinase activity (U/L). Walking (white), whole body vibration (grey), and downhill running (black) at baseline/pre, immediately post, 24 hours post, and after 8 weeks training.
[a] Significant difference ($p < 0.05$) between running and walking.

There were significant ($p < 0.05$) Measurement Period and Group main effects for F2-isoprostane concentration (Figure 2) and percentage change in F2-isoprostane ($p < 0.05$) (Figure 3), for which WBV always appeared the most stable showing the least change. All groups showed an increase in F2-isoprostane concentration immediately post-exercise (moderate-effect size for DHR of 0.66; trivial-to-small effect sizes for WBV and WLK respectively), but lower than baseline at 24h ($p < 0.05$) and 8 weeks but these trends were not significant (Figure 2). Although there was a significant main effect for the Measurement Period for percentage change of F2-isoprostane (Figure 3), there was no difference between groups. A small non-significant training effect was calculated from the paired F2-isoprostane analysis ($p = 0.081$).

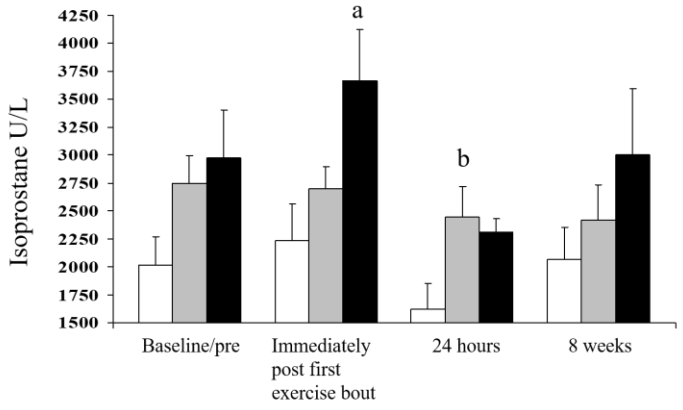

Figure 2: Mean ± SEM for F2-Isoprostane concentration (U/L). Walking (white), whole body vibration (grey), and downhill running (black) at baseline/pre, immediately post, 24 hours post, and after 8 weeks training.
[a] Significant difference ($p < 0.05$) between running and walking.
[b] Significant difference ($p < 0.05$) between running and whole body vibration.

The general TAC assay revealed acute increases immediate post and 24-hr post exercise (Table 2) for DHR (356 µM ± 29, 334 µM ± 22 respectively) and walking (319 µM ± 12, 320 µM ± 14), but not WBV (305 µM ± 14, 302 µM ± 11).

The more specific antioxidant GPx assay revealed a significant time main effect ($p < 0.001$) for all exercise modes. Post-hoc analysis revealed DHR had the highest activity for both acute, with effect sizes compared with baseline 0.72 immediately post; 1.22 at 24-hr post and chronic responses (effect size = 1.55 at 8 weeks), followed by WBV and WLK (Figure 4). However, effect sizes for WLK were 0.26; 0.74; 0.87 respectively, and for WBV were small or moderate at 0.13; 0.18; 0.65 respectively.

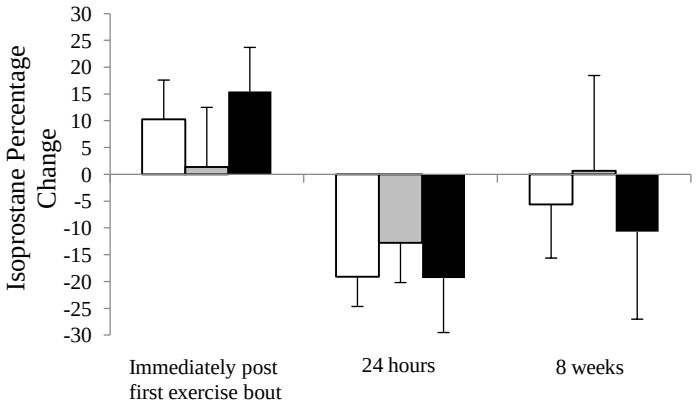

Figure 3: Percentage change from baseline in F2-Isoprostane concentration. Walking (white), whole body vibration (grey), and downhill running (black).

Table 2: Total Antioxidant Capacity response to exercise. Mean ± S.E.M. for Total Antioxidant Capacity (µM).

	Walking	Whole body vibration	Downhill running
	n = 7	n = 8	n = 6
Baseline/pre	305.8 ± 9.4	303.3 ± 13.7	326.9 ± 24.9
Immediately post first exercise bout	319.1 ± 12.2	305.1 ± 14.4	355.9 ± 29.3
24 hours	320.2 ± 14.2	301.8 ± 11.1	333.7 ± 21.5
8 weeks	334.0 ± 7.6	316.5 ± 6.6	320.1 ± 23.5

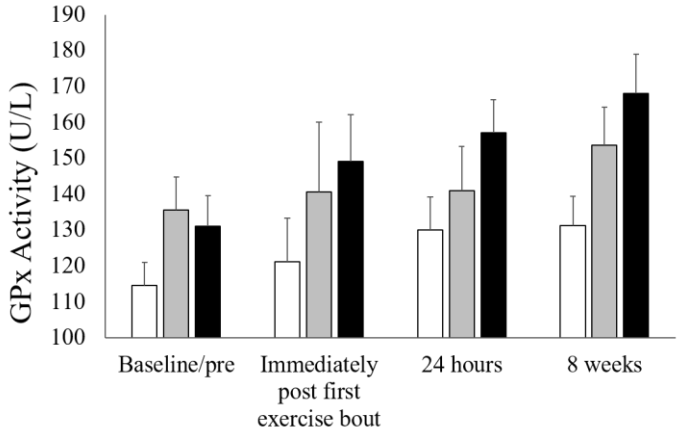

Figure 4: Mean ± SEM for GPx activity (U/L). Walking (white), whole body vibration (grey), and downhill running (black) at baseline/pre, immediately post, 24 hours post, and after 8 weeks training. There was a significant time effect for all modes of exercise.

4. Discussion

The results reveal all three exercise modes increased lipid peroxidation oxidative stress damage immediately post-exercise, but lower than baseline at 24h and 8 weeks. Furthermore, the levels of muscle damage and oxidative stress were intensity and type of exercise dependent, with oxidative stress following the same pattern over 8-weeks for each exercise mode. General TAC antioxidant activity increased after an acute bout of DHR exercise and walking but not WBV, while GPx antioxidant activity appeared to increase over the 8 weeks for all exercise modes indicating a training effect, although this appeared smallest in the WBV suggesting a lower production of ROS.

4.1. Muscle Damage and Oxidative Stress

Clearly muscle damage was incurred with the DHR as shown by the significantly increased CK measured 24 hours post exercise (Figure 1). Although some studies have reported vibration therapy for recovery purposes minimises certain markers of inflammation such as muscle soreness and IL6 (Aminian-Far, Hadian, Olayaei, Talebain, & Bakhtiary, 2011; Broadbent et al., 2010), it does not minimise CK (Broadbent et al., 2010). In comparison to the activity known to cause muscle damage i.e. DHR, the CK level produced during WBV and walking at 4.5 Km.h-1 did not change throughout the study, indicating the corresponding intensities were not sufficient to cause significant amounts of muscle damage.

Although free radical production has been linked with muscle-damaging exercise (Aoi et al., 2004) and the ensuing inflammatory response (MacIntyre, Reid, & McKenzie, 1995), there appears to be a mis-match in time to peak. In this current study F2-isoprostane levels peaked for DHR immediately after an acute bout of exercise and had reduced back to baseline levels by 24 hours (Figure 2), whereas CK appears to peak by 24 hours post exercise (Figure 1). This time to peak mis-match may be due to the rapid formation of reactive oxygen species under favourable conditions and their short life span. An increase in oxidative stress resulting from acute exercise has been previously documented (Kingsley, Wadsworth, Kilduff, McEneny, & Benton, 2005; Thompson et al., 2001), and is predicted to occur when the production of free radicals overwhelms the amount of available antioxidants (Kanter, 1998). Balance of reactive oxygen species formation and the quenching of these free radicals is resumed within 24 hours even after a 50 Km marathon (Mastaloudis, Leonard, & Traber, 2001), whereas CK is a slower cumulative process.

As DHR was the highest intensity exercise mode, it is not surprising that in the current study it also had the most lipid peroxidation oxidative stress damage with the first bout of exercise. When looking at the absolute and percentage change in oxidative stress, WBV appeared to induce the smallest change in F2-isoprostane levels for an acute response. This agrees with Theodorou et al. (2015), although they used Thiobarbituric acid (TBARS) which measures malondialdehyde and is only one of the products of lipid peroxidation and so offers a narrow view of the oxidative stress occurring. This means the oxidative stress damage in WBV was minimal compared to DHR, and even in comparison to the metabolic equivalent exercise of walking, such that any inherent antioxidant levels could cope with the challenge. This is supported by the TAC results (Table 2) which show an increase for DHR and walking after acute exercise but not for WBV. Seifi-Skishahr et al. (2008) reported that oxidative stress was intensity dependent which would indicate that walking was more strenuous that WBV. It was initially thought that the rapid co-contractions of the large muscle groups of the legs associated with WBV would increase oxygen utilisation, and therefore increase the probability of creating ROS. Therefore, the reduced oxidative stress damage demonstrated in this study is particularly important with respect to patient and elderly groups, for whom the negative effects of such damage can be significant (Goodyear-Bruch & Pierce, 2002). Furthermore, it appears the dynamic exercise of walking incurs more oxidative stress damage compared with WBV even though they are equivalent in METs. This may be attributable to the fact walking is an eccentric exercise, like DHR, which causes more muscle damage than WBV. Therefore, it is the degree of muscle damage from the type of exercise that causes increased oxidative stress and not the energy cost per se, at least for lipid peroxidation, indicating the potential of WBV as a safe exercise tool for patient and elderly groups.

4.2. Pattern of Oxidative Stress

The overall pattern of percentage change in lipid peroxidation was the same for all three exercise modes as seen in Figure 3, i.e., it increased with an acute bout of exercise, decreased during recovery 24-hours later, and was slightly less than baseline levels at 8-weeks. The lower than base-line levels at 8 weeks is indicative of a small chronic exercise effect, presumably due to an up-regulation of antioxidant activity (Parker, McGuckin, & Leicht, 2014). The effect is small overall probably because the training regime was not a high intensity and spanned only 8 weeks. Whole body vibration appears to have the smallest training effect, but as it had the least change in oxidative stress

throughout the study there would have been a lower stimulus to enhance any antioxidant enzyme concentrations. Nevertheless, GPx activity increased for all exercise modes over time (Figure 4), and not surprisingly DHR appears to have the greatest chronic increase. Eight weeks of WBV training does appear to enhance GPx with a moderate effect size compared to the large effects of WLK and DHR, which indicates WBV can produce oxidative stress which can stimulate up-regulation of at least one antioxidant. Although Theodorou et al. (2015) reported no antioxidant increase they only used TAC as the biomarker, which is most prominent in urate and excludes the contribution from antioxidant enzymes (Young, 2001). In contrast GPx is more specific to reducing the level of lipid peroxidation by quenching hydrogen peroxide and is prevalent in skeletal muscle. Limitations to this study include the low number of participants, however functional and training effects with WBV have been seen with typically small numbers (<10/group) (Torvinen et al., 2002). The authors concede larger groups may have created more differentiation between the WLK and WBV groups. The oxidative stress peak may have been higher at 1-2 hours post exercise as reported by (Michailidis et al., 2007), however the measurement time points in this study do show distinct patterns for F2-isoprostane across all the exercise modes. Some may see the recruitment of female participants as a limitation since oestrogen has been shown to exhibit antioxidant properties in animal studies (Tsuda et al., 2005), however work by Chung, Goldfarb, Jamurtas, Hegde, and Lee (1999) and Schmitz et al. (2008) reported that in humans the menstrual cycle phase had minimal influence on oxidative stress. Therefore, respective menstrual cycles were not accounted for in this study, but perhaps should be considered in further work.

Although diet was not controlled, all participants were instructed to refrain from taking any anti-oxidant supplements, or increasing their dietary intake of vitamins C or E. A further limitation was the lack of a pre-determined fitness level for participants. Although the participants in this study were a convenient sample set recruited from university students they were specifically recruited because they did not take part in any recreational sport or training activities. Furthermore, in this study the WBV exercise was being compared directly with the metabolic equivalent of walking at 4.5 km.hr-1, while the DHR exercise was included to show the potential level of muscle damage for a relative comparison, and hence this was not a training study per se. Although the self-selected speed of DHR could be viewed as a limitation, it must be remembered that participants were untrained. A minimum of 9 km.hr-1 was set, and all were encouraged to work at their maximum speed in each session, with the majority achieving 12 km.hr-1 within the first few weeks. Moreover, all other parameters of this exercise (length of time, angle of incline, weekly-frequency) were standardised across this group.

There are diurnal variations in the background oxidative stress levels in urine (Dalle-Donne et al., 2006), and albeit this is unknown in plasma, the sampling was always done in the morning as a precaution. However, the authors note that urine analyses for F2-isoprostane may have reinforced the results. The authors further acknowledge that lipid peroxidation is just one form of free radical damage which can occur in many body tissues, but it is more specific to skeletal muscle, and accurate due to its stability. Other factors that influence oxidative stress e.g., acidosis, catecholamine auto-oxidation, or ischemia-reperfusion syndrome were not considered for these participants, but the authors acknowledge they may have played a minor role in the oxidative stress levels measured.

4.3. Conclusion

An acute bout of WBV exercise does not incur significant muscle damage, and although lipid peroxidation increases immediately post-exercise it is small and decreases by 24h indicating adequate antioxidant protection for acute bouts of WBV. There is also an up-regulation of antioxidants due to chronic vibration training. We conclude that WBV exercise presents as a safe training and exercise tool, and suggest that further work is warranted to explore the effectiveness of WBV exercise in both the recovery from injury or as a warm-up/muscle activation activity.

4.4. Practical Applications

This research indicates that whole body vibration is a safe exercise that will not increase oxidative stress above that of moderate walking in those with compromised health. Furthermore, it can give protective effects of antioxidants as in regular exercise. As such, the findings demonstrate the potential of WBV as a safe exercise modality for the untrained, patients, and elderly groups.

Conflict of Interest

The authors declare no conflict of interests.

Author Contributions

Dr Lark conceived the idea, and both authors designed the experiment, participated in data collection, analyses and final editing of the manuscript.

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References

Abercromby, A. F. J., Amonette, W. E., Layne, C. S., McFarlin, B. K., Hinman, M. R., & Paloski, W. H. (2007). Vibration exposure and biodynamic responses during whole-body vibration training. *Medicine & Science in Sports & Exercise, 39*(10), 1794-1800. 1710.1249/mss.1790b1013e3181238a3181230f.

Adams, J. B., Edwards, D., Serviette, D., Bedient, A. M., Huntsman, E., Jacobs, K. A., . . . Signorile, J. F. (2009). Optimal frequency, displacement, duration, and recovery patterns to maximize power output following acute whole-body vibration. *The Journal of Strength & Conditioning Research, 23*(1), 237-245.

Alessio, H. M., Hagerman, A. E., Fulkerson, B. K., Ambrose, J., Rice, R. E., & Wiley, R. L. (2000). Generation of reactive

oxygen species after exhaustive aerobic and isometric exercise. *Medicine & Science in Sports & Exercise, 32*(9), 1576-1581.

Allen, R., & Tresini, M. (2000). Oxidative stress and gene regulation. *Free Radical Biology and Medicine, 28*(3), 463-499.

Aminian-Far, A., Hadian, M., Olayaei, G., Talebain, S., & Bakhtiary, A. (2011). Whole-body vibration and the prevention and treatment of delayed-onset muscle soreness. *Journal of Athletic Training, 46*(1), 43-49.

Aoi, W., Naito, Y., Takanami, Y., Kawai, Y., Sakuma, K., Ichikawa, H., . . . Yoshikawa, T. (2004). Oxidative stress and delayed-onset muscle damage after exercise. *Free Radical Biology and Medicine, 37*(4), 480-487.

Bogaerts, A., Delecluse, C., Claessens, A. L., Coudyzer, W., Boonen, S., & Verschueren, S. M. P. (2007). Impact of whole-body vibration training versus fitness training on muscle strength and muscle mass in older men: A 1-year randomized controlled trial. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences, 62*(6), 630-635.

Bogaerts, A. C. G., Delecluse, C., Claessens, A. L., Troosters, T., Boonen, S., & Verschueren, S. M. P. (2009). Effects of whole body vibration training on cardiorespiratory fitness and muscle strength in older individuals (a 1-year randomised controlled trial). *Age and Ageing, 38*(4), 448-454. doi:10.1093/ageing/afp067

Broadbent, S., Rousseau, J. J., Thorp, R. M., Choate, S. L., Jackson, F. S., & Rowlands, D. S. (2010). Vibration therapy reduces plasma IL6 and muscle soreness after downhill running. *British Journal of Sports Medicine, 44*(12), 888-894.

Bruyere, O., Wuidart, M.-A., Di Palma, E., Gourlay, M., Ethgen, O., Richy, F., & Reginster, J.-Y. (2005). Controlled whole body vibration to decrease fall risk and improve health-related quality of life of nursing home residents. *Archives of Physical Medicine and Rehabilitation, 86*(2), 303-307. doi:10.1016/j.apmr.2004.05.019

Cheung, W.-H., Mok, H.-W., Qin, L., Sze, P.-C., Lee, K.-M., & Leung, K.-S. (2007). High-frequency whole-body vibration improves balancing ability in elderly women. *Archives of Physical Medicine and Rehabilitation, 88*(7), 852-857. doi:10.1016/j.apmr.2007.03.028

Chung, S. C., Goldfarb, A. H., Jamurtas, A. Z., Hegde, S. S., & Lee, J. (1999). Effect of exercise during the follicular and luteal phases on indices of oxidative stress in healthy women. *Medicine & Science in Sports & Exercise, 31*(3), 409-413.

Cohen, J. (1988). Statistical power analysis for the behavioral sciences. 2nd. In: Hillsdale, NJ: Erlbaum.

Cormie, P., Deane, R. S., Triplett, N. T., & McBride, J. M. (2006). Acute effects of whole-body vibration on muscle activity, strength, and power. *Journal of Strength and Conditioning Research, 20*(2), 257.

Dalle-Donne, I., Rossi, R., Colombo, R., Giustarini, D., & Milzani, A. (2006). Biomarkers of oxidative damage in human disease. *Clinical chemistry, 52*(4), 601-623.

Delecluse, C., Roelants, M., & Verschueren, S. (2003). Strength increase after whole-body vibration compared with resistance training. *Medicine and Science in Sports and Exercise, 35*(6), 1033-1041.

Furness, T. P., & Maschette, W. E. (2009). Influence of whole body vibration platform frequency on neuromuscular performance of community-dwelling older adults. *The Journal of Strength & Conditioning Research, 23*(5), 1508-1513.
doi: 1510.1519/JSC.1500b1013e3181a1504e1508f1509.

Games, K., & Sefton, J. (2013). Whole‐body vibration influences lower extremity circulatory and neurological function. Scandinavian *Journal of Medicine & Science in Sports, 23*(4), 516-523.

Goodyear-Bruch, C., & Pierce, J. D. (2002). Oxidative stress in critically ill patients. *American Journal of Critical Care, 11*(6), 543-551.

Gusi, N., Raimundo, A., & Leal, A. (2006). Low-frequency vibratory exercise reduces the risk of bone fracture more than walking: a randomized controlled trial. *BMC Musculoskeletal Disorders, 7*(1), 92.

Iwamoto, J., Otaka, Y., Kudo, K., Takeda, T., Uzawa, M., & Hirabayashi, K. (2004). Efficacy of training program for ambulatory competence in elderly women. *The Keio Journal of Medicine, 53*(2), 85-89.

Iwamoto, J., Sato, Y., Takeda, T., & Matsumoto, H. (2012). Whole body vibration exercise improves body balance and walking velocity in postmenopausal osteoporotic women treated with alendronate: Galileo and Alendronate Intervention Trail (GAIT). *The Journal of Musculoskeletal and Neuronal Interactions*, *12*(3), 136-143.

Ji, L., & Zhang, Y. (2014). Antioxidant and anti-inflammatory effects of exercise: role of redox signaling. *Free Radical Research, 48*(1), 3-11.

Jordan, M. J., Norris, S. R., Smith, D. J., & Herzog, W. (2005). Vibration training: An overview of the area, training consequences, and future considerations. *The Journal of Strength & Conditioning Research, 19*(2), 459-466.

Kanter, M. (1998). Free radicals, exercise and antioxidant supplementation. *Proceedings of the Nutrition Society, 57*(01), 9-13.

Kawanabe, K., Kawashima, A., Sashimoto, I., Takeda, T., Sato, Y., & Iwamoto, J. (2007). Effect of whole-body vibration exercise and muscle strengthening, balance, and walking exercises on walking ability in the elderly. *Keio Journal of Medicine, 56*, 28-33.

Kingsley, M. I., Wadsworth, D., Kilduff, L. P., McEneny, J., & Benton, D. (2005). Effects of phosphatidylserine on oxidative stress following intermittent running. *Medicine & Science in Sports & Exercise, 37*(8), 1300-1306.

Lee, T., & Chow, D. (2013). Effects of whole body vibration on spinal proprioception in normal individuals. Paper presented at the Engineering in Medicine and Biology Society (EMBC), 35th Annual International Conference of the IEEE.

Lohman III, E. B., Petrofsky, J. S., Maloney-Hinds, C., Betts-Schwab, H., & Thorpe, D. (2007). The effect of whole body vibration on lower extremity skin blood flow in normal subjects. *Medical Science Monitor Basic Research, 13*(2), CR71-CR76.

MacIntyre, D. L., Reid, W. D., & McKenzie, D. C. (1995). Delayed muscle soreness. *Sports Medicine, 20*(1), 24-40.

Mastaloudis, A., Leonard, S. W., & Traber, M. G. (2001). Oxidative stress in athletes during extreme endurance

exercise. *Free Radical Biology and Medicine, 31*(7), 911-922. doi:http://dx.doi.org/10.1016/S0891-5849(01)00667-0

Merriman, H. L., Brahler, C. J., & Jackson, K. (2011). Systematically controlling for the influence of age, sex, hertz and time post-whole-body vibration exposure on four measures of physical performance in community-dwelling older adults: A randomized cross-over study. *Current Gerontology and Geriatrics Research*, 2011. doi:10.1155/2011/747094

Mester, J., Kleinöder, H., & Yue, Z. (2006). Vibration training: benefits and risks. *Journal of Biomechanics, 39*(6), 1056-1065.

Michailidis, Y., Jamurtas, A. Z., Nikolaidis, M. G., Fatouros, I. G., Koutedakis, Y., Papassotiriou, I., & Kouretas, D. (2007). Sampling time is crucial for measurement of aerobic exercise-induced oxidative stress. *Medicine and Science in Sports and Exercise, 39*(7), 1107-1113.

Moflehi, D., Kok, L.-Y., Tengku-Kamalden, T.-F., & Amri, S. (2012). Effect of single-session aerobic exercise with varying intensities on lipid peroxidation and muscle-damage markers in sedentary males. *Global Journal of Health Science, 4*(4), 48-54. doi:10.5539/gjhs.v4n4p48

Mori, S., Tuji, S., Kawamoto, M., Yamada, E., Miyamato, K., Tanaka, S., . . . Yamamoto, T. (2006). Six month whole body vibration exercises improves leg muscle strength, balance as well as calcaneal bone mineral density of community dwelled elderly. *Journal of Bone and Mineral Research, 21*(Suppl.), S249.

Page, P. (2014). Beyond statistical significance: Clinical interpretation of rehabilitation research literature. *International Journal of Sports Physical Therapy, 9*(5), 726-736.

Parker, L., McGuckin, T. A., & Leicht, A. S. (2014). Influence of exercise intensity on systemic oxidative stress and antioxidant capacity. *Clinical Physiology and Functional Imaging, 34*(5), 377-383.

Rittweger, Beller, Felsenberg, & Rittweger, J. r. (2000). Acute physiological effects of exhaustive whole-body vibration exercise in man. *Clinical Physiology, 20*(2), 134-142.

Rittweger, J., Schiessl, H., & Felsenberg, D. (2001). Oxygen uptake during whole-body vibration exercise: comparison with squatting as a slow voluntary movement. *European Journal of Applied Physiology, 86*(2), 169-173.

Roelants, M., Delecluse, C., Goris, M., & Verschueren, S. (2004). Effects of 24 weeks of whole body vibration training on body composition and muscle strength in untrained females. *International Journal of Sports Medicine, 25*(01), 1-5. doi:10.1055/s-2003-45238

Roelants, M., Delecluse, C., & Verschueren, S. M. (2004). Whole-body-vibration training increases knee-extension strength and speed of movement in older women. *Journal of the American Geriatrics Society, 52*(6), 901-908. doi:10.1111/j.1532-5415.2004.52256.x

Roelants, M. C. S. M. (2004). Whole-body-vibration training increases knee-extension strength and speed of movement in older women. *Journal of the American Geriatrics Society, 52*(6), 901-908. doi:10.1111/j.1532-5415.2004.52256.x

Rønnestad, B. R. (2009a). Acute effects of various whole-body vibration frequencies on lower-body power in trained and untrained subjects. *The Journal of Strength & Conditioning Research, 23*(4), 1309-1315.

Rønnestad, B. R. (2009b). Acute effects of various whole body vibration frequencies on 1RM in trained and untrained subjects. *The Journal of Strength & Conditioning Research, 23*(7), 2068-2072.

Sacheck, J. M., Milbury, P. E., Cannon, J. G., Roubenoff, R., & Blumberg, J. B. (2003). Effect of vitamin E and eccentric exercise on selected biomarkers of oxidative stress in young and elderly men. *Free Radical Biology and Medicine, 34*(12), 1575-1588.

Sands, W. A., McNeal, J. R., Stone, M. H., Russell, E. M., & Jemni, M. (2006). Flexibility enhancement with vibration: Acute and long-term. *Medicine & Science in Sports & Exercise, 38*(4), 720-725.

Schmitz, K. H., Warren, M., Rundle, A. G., Williams, N. I., Gross, M. D., & Kurzer, M. S. (2008). Exercise effect on oxidative stress is independent of change in estrogen metabolism. *Cancer Epidemiology, Biomarkers and Prevention, 17*(1), 220-223. doi:10.1158/1055-9965.epi-07-0058

Seifi-Skishahr, F., Siahkohian, M., & Nakhostin-Roohi, B. (2008). Influence of aerobic exercise at high and moderate intensities on lipid peroxidation in untrained men. *The Journal of Sports Medicine and Physical Fitness, 48*(4), 515-521.

Theodorou, A. A., Gerodimos, V., Karatrantou, K., Paschalis, V., Chanou, K., Jamurtas, A. Z., & Nikolaidis, M. G. (2015). Acute and chronic whole-body vibration exercise does not induce health-promoting effects on the blood profile. *Journal of Human Kinetics, 46*, 107-118. doi:10.1515/hukin-2015-0039

Thompson, D., Williams, C., Kingsley, M., Nicholas, C. W., Lakomy, H. K. A., McArdle, F., & Jackson, M. J. (2001). Muscle soreness and damage parameters after prolonged intermittent shuttle-running following acute vitamin C supplementation. *International Journal of Sports Medicine, 22*, 68-75.

Torvinen, S., Sievanen, H., Jarvinen, T. A., Pasanen, M., Kontulainen, S., & Kannus, P. (2002). Effect of 4-min vertical whole body vibration on muscle performance and body balance: a randomized cross-over study. *International Journal of Sports Medicine, 23*(5), 374-379. doi:10.1055/s-2002-33148

Tsuda, M., Iwai, M., Li, J.-M., Li, H.-S., Min, L.-J., Ide, A., . . . Suzuki, H. (2005). Inhibitory effects of AT1 receptor blocker, olmesartan, and estrogen on atherosclerosis via anti-oxidative stress. *Hypertension, 45*(4), 545-551.

Urso, M. L., & Clarkson, P. M. (2003). Oxidative stress, exercise, and antioxidant supplementation. *Toxicology, 189*(1-2), 41-54.

Verschueren, S. M., Roelants, M., Delecluse, C., Swinnen, S., Vanderschueren, D., & Boonen, S. (2004). Effect of 6-month whole body vibration training on hip density, muscle strength, and postural control in postmenopausal women: a randomized controlled pilot study. *Journal of Bone and Mineral Research: The Official Journal of the American Society for Bone and Mineral Research, 19*(3), 352-359. doi:10.1359/JBMR.0301245

von Stengel, S., Kemmler, W., Engelke, K., & Kalender, W. A. (2012). Effect of whole-body vibration on neuromuscular performance and body composition for females 65 years and older: a randomized-controlled trial. *Scandinavian Journal of Medicine & Science in Sports, 22*(1), 119-127. doi:10.1111/j.1600-0838.2010.01126.x

Young, I. (2001). Measurement of total antioxidant capacity. *Journal of Clinical Pathology, 54*(5), 339-339.