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The decorin and myostatin response to acute whole body vibration: impact of adiposity, sex, and race

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BACKGROUND: Traditional forms of exercise affect immune, metabolic, and myokine responses and contribute to a multitude of health benefits. Whole body vibration (WBV) has recently emerged as an exercise mimetic that may be more tolerable for those individuals that cannot perform traditional exercise. However, the myokines response to acute WBV in humans has yet to be fully elucidated.

OBJECTIVE: To characterize the decorin and myostatin response to acute whole body vibration (WBV) and determine the impact of adiposity, sex, and race.

SUBJECTS: One hundred twenty-nine adults (32.8 ± 0.4 years, 66.7% female, 53.5% non-Hispanic Black) were recruited as part of an ongoing, longitudinal twin cohort parent study. Participants were classified into three groups: those with obesity (OB: $\geq 30 \text{ kg/m}^2$), those who are overweight (OW: ≥ 25 and $< 30 \text{ kg/m}^2$), or those with normal weight (NW: $< 25 \text{ kg/m}^2$) based on BMI.

METHODS: Blood was collected at baseline (PRE), immediately post (POST), and 1 h (1H), 3 h (3H), and 24 h (24H) post WBV. The acute WBV protocol consisted of 10 cycles of 1 min of vibration exercise followed by 30 s of standing rest.

RESULTS: The response was similar between NW and OW, so these groups were combined for analysis (NW/OW: BMI $< 30 \text{ kg/m}^2$). Overall, circulating concentrations of decorin were higher ($p < 0.001$) POST ($8.80 \pm 0.19 \text{ pg/mL}$) and significantly lower ($p < 0.005$) at 1H ($8.66 \pm 0.19 \text{ pg/mL}$) and 3H ($8.68 \pm 0.19 \text{ pg/mL}$), compared to PRE ($8.71 \pm 0.19 \text{ pg/mL}$). Decorin POST was greater ($p = 0.016$) in the OB group ($8.82 \pm 0.18 \text{ pg/mL}$) compared to the NW/OW group ($8.77 \pm 0.20 \text{ pg/mL}$). Overall, myostatin was higher ($p = 0.002$) POST ($54.93 \pm 1.04 \text{ pg/mL}$) and lower ($p < 0.001$) at 24H ($49.13 \pm 1.04 \text{ pg/mL}$) compared to PRE ($53.49 \pm 1.04 \text{ pg/mL}$). The myostatin response was lower ($p < 0.001$) in female and non-Hispanic White individuals compared to male and non-Hispanic Black individuals, respectively.

CONCLUSIONS: A single bout of WBV can facilitate the release of decorin and myostatin into circulation, a similar response to traditional exercise. Additionally, adiposity, sex and race should be considered when evaluating the myokines response to WBV.

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INTRODUCTION

Obesity continues to be a public health crisis with over a third of US adults having a body mass index (BMI) of $\geq 30 \text{ kg/m}^2$ [1, 2]. The total health and financial burden of obesity cannot be overstated, and individuals with obesity are at an increased risk for cardiovascular disease, metabolic disorders, and several cancers [3, 4]. While there may be a hereditary component to obesity [5], the increased adiposity is likely due to a combination of high caloric diets, excessive calorie consumption, and reduced physical activity [3].

Physical activity and/or exercise is an effective method that not only aids in the treatment and prevention of obesity, it is protective against obesity-related cardiac and metabolic risk factors [6]. Individuals with obesity, however, are less likely to start or maintain regular exercise due to the physical and mental limitations that are associated with increased adiposity [7–9].

Accordingly, this vicious cycle of physical inactivity leading to weight gain is difficult to break and further exacerbates the obesity-related impairments in overall health and wellbeing.

Whole body vibration (WBV) has emerged as an exercise mimetic that represents an alternative to conventional exercise modalities such as walking, running or biking [10, 11]. WBV requires little movement, making this low-impact exercise alternative more tolerable than traditional exercise, and may be more appealing to individuals with greater adiposity who may have joint pain or limited range of motion [12]. While the mechanisms that mediate the beneficial effects of traditional exercise have yet to be completely elucidated, myokines may certainly play an important role [13]. Myokines are signaling molecules released by skeletal muscle in response to muscle contractions. Myokines facilitate many key roles throughout the body such as assisting with increases in glucose metabolism and

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fat oxidation, contributing to post-exercise reductions in systemic inflammation, and inducing muscle hypertrophy [14–16].

Three myokines of particular interest are IL-6, myostatin, and decorin. Circulating IL-6 increases with traditional exercise in a dose response manner and has roles in muscle building and insulin sensitivity [17–19]. Myostatin is secreted by skeletal muscle and is a negative regulator of skeletal muscle growth [20]. Myostatin decreases following acute bouts of both resistance and aerobic exercise and contributes to decreased proliferation and differentiation of myoblasts [21–24]. Accordingly, loss of function of myostatin leads to muscular hypertrophy [25]. In contrast, decorin is released by skeletal muscle contraction and acts as an antagonist to myostatin. Concentrations of decorin are increased following acute bouts of resistance exercise, and this has been shown to upregulate pro-myogenic factors such as Mighty, Myod1, and follistatin. Multiple bouts of acute exercise lead to a chronic increase in decorin expression, which likely contributes to increased muscle mass over time [26].

Studies on the myokine response to WBV have been limited. In humans, a single bout of WBV has been shown to increase circulating concentrations of IL-6 and irisin [27, 28]. In addition, an increase in myostatin following WBV was observed in mice [29]. Whether or not WBV could facilitate changes in circulating decorin and myostatin in humans has yet to be investigated. Indeed, similar to traditional modalities of exercise, the effects on immune and metabolic systems following a single bout of WBV have been shown to be dependent on adiposity [11, 28]. In fact, the percent change in IL-6 following WBV was greater in normal weight compared to individuals with obesity [28]. Whether adiposity plays a role in the decorin and myostatin response to WBV has not been explored. Thus, the purpose of this study is to characterize the myokine response, specifically decorin and myostatin, to acute WBV in humans, and determine the impact that adiposity plays in this process.

METHODS

Experimental design

All participants reported to the Laboratory of Integrated Vascular and Exercise Physiology (LIVEP) at the Georgia Prevention Institute for two visits, a preliminary visit and an experimental visit. The preliminary visit consisted of the informed consent process, anthropometric measures, and body composition assessment. Height and weight, determined using a stadiometer and standard platform scale (CN20, DETECTO®, Webb City, MO), were used for calculation of body mass index (BMI). Total body fat, fat-mass, and fat-free mass were determined using dual-energy X-ray absorptiometry (QDR-4500W; Hologic, Waltham, MA). Resting systolic and diastolic blood pressures were evaluated using established protocols [30]. For the experimental visit, participants reported to the LIVEP in the morning following an overnight fast and having abstained from moderate-to-vigorous physical activity for 24 h prior to performing the WBV protocol. An IV was placed in the antecubital fossa, and blood samples were collected at baseline (PRE), immediately post WBV (POST), and 1 h (1H), 3 h (3H), and 24 h (24H) post WBV.

Participants

One hundred twenty-nine apparently healthy men and women (55 twin pairs and 19 singletons) ages 18–45 years were recruited as part of an ongoing, longitudinal twin cohort parent study. Body mass index (BMI) was used to assign participants into three groups: those with obesity (OB: $\geq 30 \text{ kg/m}^2$), those who are overweight (OW: ≥ 25 and $< 30 \text{ kg/m}^2$), or those with normal weight (NW: $< 25 \text{ kg/m}^2$).

Body composition and skeletal muscle index

Fat-free soft tissue mass and fat mass were assessed by dual-energy X-ray absorptiometry (QDR-4500W; Hologic Inc., Waltham, MA). The residual derived from the regression of fat-free soft tissue mass on height and fat mass was used as an index of skeletal muscle mass (SMI). This index is a proxy for skeletal muscle health, independent of the influence from both height and fat mass, and has been widely used when assessing skeletal

muscle depletion in general population studies [31, 32]. A positive residual indicates a relatively muscular individual. Participants were further stratified into low, intermediate, and high muscle mass groups according to even distribution of SMI tertiles.

Acute whole body vibration protocol

A synchronous WBV platform (Power Plate Pro 5, Performance Health Systems) was used for this investigation. Participants were instructed to remove any footwear and stand mid-center on the platform with a loose grip on the front rails. Vibration frequency was set to 30 Hz as this frequency has been demonstrated to elicit muscle activation, yet is well below the frequency in which harmful side effects may occur [12]. Vibration amplitude was set to 2.5 mm. These settings yielded a calculated peak acceleration of 4.5 g (44.4 m/s). The acute WBV protocol consisted of 10 cycles of 1 min of vibration exercise followed by 30 s of standing rest, which has been shown to induce changes in the metabolic and myokines profile [28]. During the vibration portion of the protocol, participants were instructed to stand in a static squat position, consisting of knee flexion ($\sim 60^\circ$) with a stable non-flexed trunk.

Clinical laboratory and myokine analysis

An intravenous catheter was inserted into an antecubital vein and a 10 mL blood sample at baseline (PRE) was obtained. Fasting concentrations of total cholesterol (TC), high-density lipoproteins (HDL), low-density lipoproteins (LDL), triglycerides (TG), and glucose were assessed using standard core laboratory techniques (Laboratory Corporation of America Holdings, Burlington, NC). Hemoglobin A_{1c} (HbA_{1c}) was determined using standard core laboratory techniques (Laboratory Corporation of American Holdings, Birmingham, AL). Additional blood samples were collected immediately following (POST), 1 h (1H), and 3 h (3H) post-WBV. Participants returned to the LIVEP in a fasted state 24 h (24H) following WBV and a final venous blood sample was obtained. Blood was separated via centrifugation and plasma samples were aliquoted, flash frozen in liquid nitrogen, and stored at -80°C until further analysis. High sensitivity ELISA kits were used to determine the plasma concentrations of myostatin (R&D Systems, Minneapolis, MN) and decorin (Abcam, Cambridge, United Kingdom) according to manufacturer's instructions. The lower limit of detection for the myostatin and decorin assay kits are 31.3 pg/mL kit and 0.76 pg/mL, respectively.

Statistical analyses

All statistical analyses were performed using Stata 17.0 (StataCorp). The major goals of the analyses were to characterize the pattern of the myokine response to an acute bout of WBV (i.e., time effect), as well as test whether the response was dependent on sex, race, adiposity status and skeletal muscle mass (i.e., interactions between time and these parameters). Since the myokine response was similar between normal weight and overweight groups, these two groups were combined as a normal weight/overweight group for analysis and presentation of the findings. Due to the complex design (i.e., multiple time points within one individual and potential twin pairs within a family), multilevel linear mixed models were used, as previously described [32], with myokine concentrations as the dependent variables, and age, sex, race, adiposity status and skeletal muscle mass as independent variables. Families and individuals were modeled as random effects in each analysis. The tests of interest included the main effect of time, the main effect of sex, race, adiposity status and skeletal muscle mass. These variables were also modeled with their individual interactions with time. Overall data were evaluated without the interaction of time. Values are presented as mean \pm SEM unless otherwise noted. An alpha < 0.05 was considered statistically significant for all analyses.

RESULTS

Participant characteristics and clinical laboratory values

Participant demographics and clinical laboratory values are presented in Table 1. Roughly equal numbers of non-Hispanic Black (NHB) and non-Hispanic White (NHW) were included in each group. The group with obesity was significantly older ($p < 0.001$) and as expected had a greater BMI ($p < 0.001$), body weight ($p < 0.001$), and body fat percentage ($p < 0.001$). In addition, triglycerides ($p = 0.009$), fasting glucose ($p < 0.001$), and HbA_{1c}

Table 1. Participant characteristics by adiposity group.

	All participants (N = 129)	NW/OW (N = 65)	OB (N = 64)	p value
Age (years)	32.8 ± 0.4	30.7 ± 0.6	34.9 ± 0.6	<0.001
Percent of females (%)	66.7	50	50	
Percent of NHB (%)	53.5	43.5	56.5	
Height (cm)	168.7 ± 0.8	168.0 ± 1.2	169.3 ± 1.1	0.433
Weight (kg)	90.2 ± 2.6	68.7 ± 1.4	112.2 ± 3.1	<0.001
BMI (kg/m ²)	31.6 ± 0.8	24.3 ± 0.4	38.9 ± 0.9	<0.001
Body fat (%)	37.8 ± 0.9	32.4 ± 1.1	43.6 ± 0.9	<0.001
Handgrip (kg)	34.0 ± 1.1	33.9 ± 1.7	34.1 ± 1.4	0.956
Total cholesterol (mg/dL)	172.4 ± 3.2	170.5 ± 4.2	174.7 ± 4.8	0.513
HDL (mg/dL)	52.4 ± 1.2	56.4 ± 1.6	48.4 ± 1.7	<0.001
LDL (mg/dL)	100.6 ± 2.8	97.5 ± 3.8	104.1 ± 4.2	0.252
Triglycerides (mg/dL)	99.4 ± 6.1	83.1 ± 6.1	115.3 ± 10.3	0.009
Fasting glucose (mg/dL)	86.5 ± 1.0	82.1 ± 0.8	86.2 ± 1.7	<0.001
HbA _{1c} (%)	5.4 ± 0.05	5.2 ± 0.04	5.7 ± 0.07	<0.001

Data are presented as mean ± SEM. Bold values indicate statistically significant differences between NW/OW (BMI < 30) and OB (BMI ≥ 30) groups. BMI body mass index, HDL high-density lipoprotein, LDL low-density lipoprotein, HbA_{1c} hemoglobin A1c, NHB non-Hispanic Black.

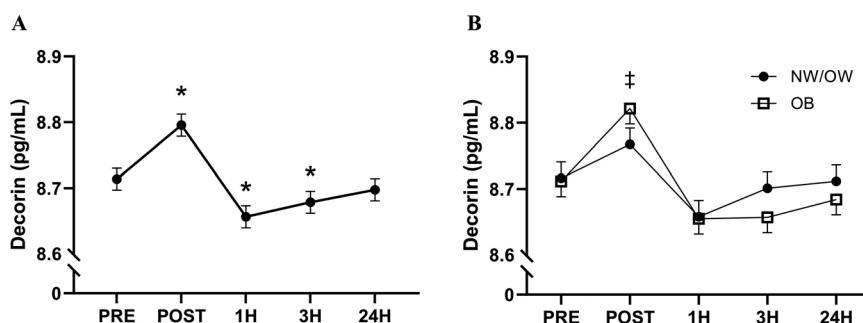


Fig. 1 Decerin Response to Acute Whole Body Vibration. Decerin response at PRE, POST, 1H, 3H, and 24H after WBV across **A** the entire cohort and **B** stratified into NW/OW (BMI < 30) and OB (BMI ≥ 30) groups; $n = 129$ (NW/OW = 65, OB = 64). Multilevel linear mixed models. Data presented as mean ± SEM. *indicates a significant ($p < 0.05$) difference from PRE circulating decerin concentrations. ‡indicates a significant ($p < 0.05$) difference between NW/OW and OB groups.

($p < 0.001$) were all higher in the group with obesity vs. the group who is normal weight/overweight, and HDL ($p < 0.001$) was lower in the group with obesity compared to the group who is normal weight/overweight. All other characteristics and clinical laboratory values were similar between groups. Given the effect of myokines on glucose, similar findings were observed even when controlling for baseline differences in circulating blood glucose.

Myokine response to WBV

Decorin and WBV. The overall decerin response to WBV over time is presented in Fig. 1A. Circulating concentrations of decerin increased significantly ($p < 0.001$) immediately POST (8.80 ± 0.19 pg/mL) WBV compared to PRE (8.71 ± 0.19 pg/mL). In contrast, decerin was significantly lower at 1H (8.66 ± 0.19 pg/mL; $p < 0.001$) and 3H (8.68 ± 0.19 pg/mL; $p = 0.005$) compared to PRE. Concentrations of decerin returned to PRE values by 24H (8.70 ± 0.19 pg/mL; $p = 0.194$). Figure 1B illustrates the significant adiposity x time interaction for the decerin response. The response to WBV was not significantly different between the groups with normal weight vs. overweight, so these groups were combined for analysis (NW/OW: BMI < 30 kg/m²). While PRE concentrations of decerin were similar between NW/OW (8.72 ± 0.20 pg/mL) and OB (8.71 ± 0.19 pg/mL) groups, there was a significantly ($p = 0.016$) greater increase in decerin immediately POST WBV in the group with obesity (8.82 ± 0.19 pg/mL) as compared to the group who is normal weight/overweight.

(8.77 ± 0.20 pg/mL). Decerin was similar between groups at 1H (OB: 8.65 ± 0.19 pg/mL vs. NW/OW: 8.66 ± 0.20 pg/mL; $p = 0.939$), 3H (OB: 8.66 ± 0.19 pg/mL vs. NW/OW: 8.70 ± 0.20 pg/mL; $p = 0.111$), and 24H (OB: 8.68 ± 0.19 pg/mL vs. NW/OW: 8.71 ± 0.20 pg/mL; $p = 0.359$) time points, reflecting that differences in adiposity only affected the decerin response immediately post WBV. Neither race ($p = 0.736$), sex ($p = 0.395$), nor SMI ($p = 0.476$) had a significant effect on the decerin response to WBV.

Myostatin and WBV. The overall myostatin response to WBV over time is presented in Fig. 2A. There was a significant ($p = 0.002$) increase in circulating concentrations of myostatin immediate POST (54.93 ± 1.04 pg/mL) WBV compared to PRE (53.45 ± 1.04 pg/mL). In addition, myostatin at 1H (53.68 ± 1.04 pg/mL; $p = 0.678$) and 3H (54.01 ± 1.04 pg/mL; $p = 0.257$) was similar to PRE; however, myostatin decreased significantly ($p < 0.001$) from PRE at 24H (49.13 ± 1.04 pg/mL) following WBV. Concentrations of myostatin were similar between the group with obesity, the group who is overweight, and the group with normal weight following WBV and no differences in the myostatin response over time was observed between the group with obesity, group who is overweight, and group with normal weight. Looking at the main effect of sex, race, and SMI without the interaction of time, overall, myostatin was significantly greater in men (58.01 ± 1.04 pg/mL; $p < 0.001$) and NHB individuals (56.54 ± 1.02 pg/mL; $p = 0.001$) as

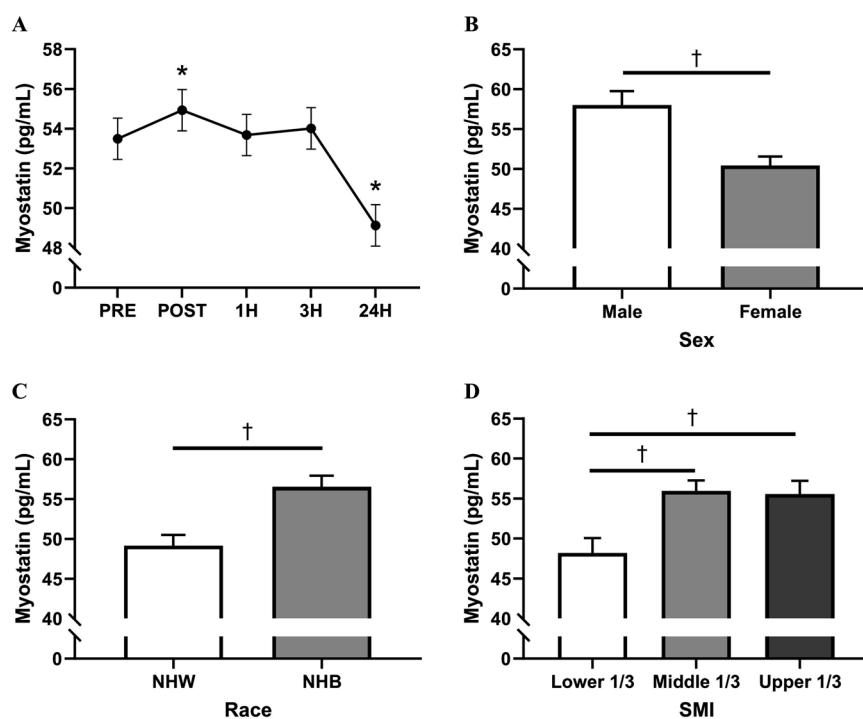


Fig. 2 Myostatin Response to Acute Whole Body Vibration. Myostatin response at PRE, POST, 1H, 3H, and 24H after WBV across **A** the entire cohort; $n = 129$. Without the interaction of time, overall concentrations of myostatin comparing differences in **B** sex, **C** race, and **D** tertiles of skeletal muscle index (SMI); $n = 129$ (male = 43, female = 86, NHW = 60, NHB = 69, lower 1/3 = 43, middle 1/3 = 43, upper 1/3 = 43). Multilevel linear mixed models. Data presented as mean \pm SEM. *indicates a significant ($p < 0.05$) difference from PRE circulating myostatin concentrations. †indicates a significant ($p < 0.05$) difference between demographic groups.

compared to women (50.43 ± 1.01 pg/mL) and NHW individuals (49.15 ± 0.93 pg/mL), respectively (Fig. 2B, C). While there were differences in the overall concentrations of myostatin following WBV across sex and race, the interactions between sex \times time ($p = 0.253$) or race \times time ($p = 0.090$) of the myostatin response to WBV were not statistically significant. Moreover, without the interaction of time, overall circulating concentrations of myostatin were significantly lower in individuals in the lowest tertile of skeletal muscle index (SMI) (48.18 ± 1.08 pg/mL) as compared to those in the intermediate (55.94 ± 0.76 pg/mL; $p = 0.004$) or high (55.58 ± 0.94 pg/mL; $p = 0.041$) tertile of SMI (Fig. 2D).

DISCUSSION

For the first time, the present investigation has documented the myokine response to WBV and determined the impact of adiposity on the response. Findings demonstrate that decorin increases immediately POST and is reduced at 1H and 3H following WBV. In addition, Myostatin increases immediately POST and decreases 24H following WBV. Further, the decorin response to WBV varies between the group who is normal weight/overweight and the group with obesity, with circulating decorin concentrations increasing more in individuals with obesity immediately post WBV. Additionally, myostatin concentrations following WBV were lower in women, NHW, and those in the lower tertile of SMI. These data support the observation of a meaningful physiological response following a single bout of WBV.

Decorin response to WBV

Decorin is released by contracting muscles and increased decorin concentrations following traditional resistance exercise has been shown to contribute to muscle hypertrophy [26]. This muscle growth is likely due to decorin's antagonizing effects on myostatin, an inhibitor of muscle growth [20, 33]. In the present investigation, circulating concentrations of decorin were significantly increased

immediately POST WBV. This higher POST WBV response is consistent with previous studies of traditional exercise that demonstrate an increase in decorin concentrations immediately following acute resistance training [26]. Collectively, these data provide evidence that WBV can mimic a similar physiological response that traditional exercise induces, and thus has the potential to provide similar health benefits.

In addition, the immune, metabolic, and IL-6 response to WBV does vary between individuals with obesity and those who are of normal weight [28]. In the present study, individuals with obesity exhibited a significantly greater increase in circulating decorin immediately POST WBV compared to individuals who are normal weight/overweight. Consequently, it is conceivable that the increase in decorin can promote skeletal muscle growth [26] following WBV by stimulating muscle spindles and alpha-motoneurons [34]. Muscle growth has significant impacts on overall health such as increasing metabolic rate and ultimately lowering risk for several chronic diseases, including cardiovascular disease [35]. In fact, increases in decorin may have therapeutic potential as it has recently been used in pre-clinical studies to decrease muscle loss in murine models of muscular dystrophies [36].

It is noteworthy that decorin is also expressed in adipose tissue, and its expression is increased in individuals with obesity [37]. Therefore, whether this differential response in plasma decorin between the group who is normal weight/overweight and the group with obesity immediately POST WBV is due to increased release of decorin from muscles or adipose tissue is unclear. Regardless of the source, increased decorin has a positive effect on glucose metabolism, muscle building, and fat loss [26, 38, 39]. Accordingly, the increase in plasma decorin following acute WBV could offer metabolic benefits that may be specific to individuals with obesity. Given the ease and convenience, WBV could even be used as a more favorable form of exercise to reduce risks of chronic diseases in populations with limited mobility or low motivation to engage in time-consuming traditional exercise.

It is important to note that the acute upregulation of decorin following WBV identified in the present study might have other effects on the body aside from its potential role in skeletal muscle health. For example, decorin can downregulate receptor tyrosine kinases [40], which are often expressed in many cancer cells, and downregulation of tyrosine kinases have been shown to combat tumorigenic signaling pathways [41]. Whether WBV can induce these decorin related benefits in cancer is unclear, however further exploration of how WBV effects signaling pathways would be useful to increase our understanding and fully define the health impacts of WBV.

Myostatin response to WBV

Myostatin is a negative regulator of muscle growth [20] and traditional exercise has been shown to decrease myostatin, providing a mechanism for exercise to promote skeletal muscle growth [21]. In the present investigation, circulating concentrations of myostatin were significantly increased immediately POST WBV in both groups. In addition, Myostatin was significantly lower at 24H following WBV compared to PRE. This response is consistent with previous studies that have documented a decrease in plasma myostatin 24 h following acute bouts of traditional exercise as well as an increase immediately after [42–44]. Taken together, these data provide evidence to indicate that acute bouts of WBV can facilitate a positive physiologic myostatin response that is similar to traditional modalities of acute exercise.

While there are health benefits to acute bouts of exercise, it is routine, persistent, chronic exercise that ultimately reduces the risk for chronic diseases [45]. In a meta-analysis that investigated the myostatin response to traditional resistance exercise training, chronic traditional exercise spanning from 5 weeks to 6 months in length, was found to have the most profound decrease in myostatin concentrations [46]. These longer-term decreases in myostatin are likely more favorable for continuous and substantial muscle gain. It is important to emphasize, however, chronic training results from repeated bouts of acute training. Therefore, if the acute physiological response to WBV observed in the present study has a compounding effect, it would identify WBV as a veritable exercise replacement with the ability to promote a similar long-term health benefits as traditional chronic exercise. Accordingly, further studies that investigate the myokine response following chronic WBV are certainly warranted.

On average, men and NHB individuals have increased muscle mass as compared to women and NHW individuals, respectively [47, 48]. Findings from the present study also document that the overall myostatin response to WBV was higher in men compared to women, and in NHB individuals compared to NHW individuals. Similarly, individuals with the lowest skeletal muscle index exhibited the least amount of overall circulating concentrations of myostatin following WBV. It is conceivable that individuals with increased muscle mass would exhibit lower concentrations of myostatin, which would allow for more muscle growth.

However, a positive correlation between myostatin concentrations and muscle mass has previously been documented [49] and aligns with the findings of the present investigation. This “myostatin paradox” may be explained by the fact that myostatin is released by skeletal muscle contraction, consistent with the findings of the present investigation that documented a significant increase in myostatin immediately post WBV [20, 49]. This myostatin paradox also demonstrates that muscle regulation and growth is extremely complex and is likely controlled by a multitude of factors, with circulating myostatin only being one piece of the puzzle. Consequently, it is plausible that individuals with an increased muscle mass have increased concentrations of pro-myogenic factors to balance this increase in circulating myostatin under basal conditions; however, future investigations are needed to test this hypothesis.

CONCLUSION

Data from the present investigation provides evidence that a single bout of WBV can facilitate muscle activation and promote the release of myokines in the general population. Specifically, acute bouts of WBV led to an initial increase in both decorin and myostatin, followed by a decrease at 24H, a physiological response that favors muscle growth and is consistent with the myokine response to traditional exercise. Additionally, circulating concentrations of decorin immediately following WBV were greater in individuals with obesity compared to individuals who are normal weight/overweight and myostatin concentrations in response to WBV were lower in women, NHW, and those in the lower tertile of SMI. While future studies would need to be done to fully assess the chronic effects of WBV on skeletal muscle health, the present findings provide the foundation to support the use of WBV as an exercise replacement with the capability of reducing the risk of chronic disease, particularly in populations that cannot or do not engage in traditional exercise.

DATA AVAILABILITY

The dataset used and analyzed during the current study is available from the corresponding author on reasonable request.

REFERENCES

1. Agha M, Agha R. The rising prevalence of obesity: part A: impact on public health. *Int J Surg Oncol*. 2017;2:e17.
2. Ogden CL, Fakhouri TH, Carroll MD, Hales CM, Fryar CD, Li XF, et al. Prevalence of obesity among adults, by household income and education—United States, 2011–2014. *Morb Mortal Wkly Rep*. 2017;66:1369–73.
3. Eckel RH. Obesity and heart disease: a statement for healthcare professionals from the Nutrition Committee, American Heart Association. *Circulation*. 1997;96:3248–50.
4. Hotamisligil GS. Inflammation, metaflammation and immunometabolic disorders. *Nature*. 2017;542:177–85.
5. McPherson R. Genetic contributors to obesity. *Can J Cardiol*. 2007;23:23A–7A.
6. Schuler G, Adams V, Goto Y. Role of exercise in the prevention of cardiovascular disease: results, mechanisms, and new perspectives. *Eur Heart J*. 2013;34:1790–9.
7. Pietilainen KH, Kaprio J, Borg P, Plasqui G, Yki-Jarvinen H, Kujala UM, et al. Physical inactivity and obesity: a vicious circle. *Obesity*. 2008;16:409–14.
8. Okifuji A, Hare BD. The association between chronic pain and obesity. *J Pain Res*. 2015;8:399–408.
9. Sarwer DB, Polonsky HM. The psychosocial burden of obesity. *Endocrinol Metab Clin North Am*. 2016;45:677–88.
10. Totosy de Zepetnek JO, Miyatani M, Szeto M, Giangregorio LM, Craven BC. The effects of whole body vibration on pulse wave velocity in men with chronic spinal cord injury. *J Spinal Cord Med*. 2017;40:795–802.
11. Zago M, Capodaglio P, Ferrario C, Tarabini M, Galli M. Whole-body vibration training in obese subjects: a systematic review. *PLoS ONE*. 2018;13:e0202866.
12. Cardinale M, Wakeling J. Whole body vibration exercise: are vibrations good for you? *Br J Sports Med*. 2005;39:585–9.
13. Leal LG, Lopes MA, Batista ML Jr. Physical exercise-induced myokines and muscle-adipose tissue crosstalk: a review of current knowledge and the implications for health and metabolic diseases. *Front Physiol*. 2018;9:1307.
14. Glund S, Deshmukh A, Long YC, Moller T, Koistinen HA, Caidahl K, et al. Interleukin-6 directly increases glucose metabolism in resting human skeletal muscle. *Diabetes*. 2007;56:1630–7.
15. Mizuhara H, O'Neill E, Seki N, Ogawa T, Kusunoki C, Otsuka K, et al. T cell activation-associated hepatic injury: mediation by tumor necrosis factors and protection by interleukin 6. *J Exp Med*. 1994;179:1529–37.
16. Zunner BEM, Wachsmuth NB, Eckstein ML, Scherl L, Schierbauer JR, Haupt S, et al. Myokines and resistance training: a narrative review. *Int J Mol Sci*. 2022;23:23.
17. Christiansen T, Bruun JM, Paulsen SK, Olholm J, Overgaard K, Pedersen SB, et al. Acute exercise increases circulating inflammatory markers in overweight and obese compared with lean subjects. *Eur J Appl Physiol*. 2013;113:1635–42.
18. Pedersen BK, Hoffman-Goetz L. Exercise and the immune system: regulation, integration, and adaptation. *Physiol Rev*. 2000;80:1055–81.
19. Munoz-Canovas P, Scheele C, Pedersen BK, Serrano AL. Interleukin-6 myokine signaling in skeletal muscle: a double-edged sword? *FEBS J*. 2013;280:4131–48.
20. McPherron AC, Lawler AM, Lee SJ. Regulation of skeletal muscle mass in mice by a new TGF-beta superfamily member. *Nature*. 1997;387:83–90.
21. Hittel DS, Axelson M, Sarna N, Shearer J, Huffman KM, Kraus WE. Myostatin decreases with aerobic exercise and associates with insulin resistance. *Med Sci Sports Exerc*. 2010;42:2023–9.

22. Hulmi JJ, Kovanen V, Selanne H, Kraemer WJ, Hakkinen K, Mero AA. Acute and long-term effects of resistance exercise with or without protein ingestion on muscle hypertrophy and gene expression. *Amino Acids*. 2009;37:297–308.

23. Louis E, Raué U, Yang Y, Jemioł B, Trappe S. Time course of proteolytic, cytokine, and myostatin gene expression after acute exercise in human skeletal muscle. *J Appl Physiol*. 2007;103:1744–51.

24. Joulia D, Bernardi H, Garandel V, Rabenoelina F, Vernus B, Cabello G. Mechanisms involved in the inhibition of myoblast proliferation and differentiation by myostatin. *Exp Cell Res*. 2003;286:263–75.

25. Kambadur R, Sharma M, Smith TP, Bass JJ. Mutations in myostatin (GDF8) in double-muscled Belgian Blue and Piedmontese cattle. *Genome Res*. 1997;7:910–6.

26. Kanzleiter T, Rath M, Gorgens SW, Jensen J, Tangen DS, Kolnes AJ, et al. The myokine decorin is regulated by contraction and involved in muscle hypertrophy. *Biochem Biophys Res Commun*. 2014;450:1089–94.

27. Huh JY, Mougijs V, Skraparlis A, Kabasakalis A, Mantzoros CS. Irisin in response to acute and chronic whole-body vibration exercise in humans. *Metabolism*. 2014;63:918–21.

28. Blanks AM, Rodriguez-Miguel P, Looney J, Tucker MA, Jeong J, Thomas J, et al. Whole body vibration elicits differential immune and metabolic responses in obese and normal weight individuals. *Brain Behav Immun Health*. 2020;1:100011.

29. Cariati I, Bonanni R, Pallone G, Romagnoli C, Rinaldi AM, Annino G, et al. Whole body vibration improves brain and musculoskeletal health by modulating the expression of tissue-specific markers: FNDC5 as a key regulator of vibration adaptations. *Int J Mol Sci*. 2022;23:10388.

30. Kapuku GK, Treiber FA, Davis HC, Harshfield GA, Cook BB, Mensah GA. Hemodynamic function at rest, during acute stress, and in the field: predictors of cardiac structure and function 2 years later in youth. *Hypertension*. 1999;34:1026–31.

31. Bosy-Westphal A, Muller MJ. Identification of skeletal muscle mass depletion across age and BMI groups in health and disease—there is need for a unified definition. *Int J Obes*. 2015;39:379–86.

32. Hao G, Pollock NK, Harris RA, Gutin B, Su S, Wang X. Associations between muscle mass, physical activity and dietary behaviour in adolescents. *Pediatr Obes*. 2019;14:e12471.

33. Kishioka Y, Thomas M, Wakamatsu J, Hattori A, Sharma M, Kambadur R, et al. Decorin enhances the proliferation and differentiation of myogenic cells through suppressing myostatin activity. *J Cell Physiol*. 2008;215:856–67.

34. Cardinale M, Bosco C. The use of vibration as an exercise intervention. *Exerc Sport Sci Rev*. 2003;31:3–7.

35. McLeod JC, Stokes T, Phillips SM. Resistance exercise training as a primary countermeasure to age-related chronic disease. *Front Physiol*. 2019;10:645.

36. Iqbal A, May U, Prince SN, Jarvinen TAH, Heydemann A. Systemically administered homing peptide targets dystrophic lesions and delivers transforming growth factor-beta (TGFbeta) inhibitor to attenuate murine muscular dystrophy pathology. *Pharmaceutics*. 2021;13:1506.

37. Bolton K, Segal D, McMillan J, Jowett J, Heilbronn L, Abberton K, et al. Decorin is a secreted protein associated with obesity and type 2 diabetes. *Int J Obes*. 2008;32:1113–21.

38. Svard J, Rost TH, Sommervoll CEN, Haugen C, Gudbrandsen OA, Mellgren AE, et al. Absence of the proteoglycan decorin reduces glucose tolerance in overfed male mice. *Sci Rep*. 2019;9:4614.

39. Ward M, Ajuwon KM. Regulation of pre-adipocyte proliferation and apoptosis by the small leucine-rich proteoglycans, biglycan and decorin. *Cell Prolif*. 2011;44:343–51.

40. Goldoni S, Iozzo RV. Tumor microenvironment: modulation by decorin and related molecules harboring leucine-rich tandem motifs. *Int J Cancer*. 2008;123:2473–9.

41. Neill T, Schaefer L, Iozzo RV. Decorin: a guardian from the matrix. *Am J Pathol*. 2012;181:380–7.

42. Hulmi JJ, Tannerstedt J, Selanne H, Kainulainen H, Kovanen V, Mero AA. Resistance exercise with whey protein ingestion affects mTOR signaling pathway and myostatin in men. *J Appl Physiol*. 2009;106:1720–9.

43. Kazemi F. The correlation of resistance exercise-induced myostatin with insulin resistance and plasma cytokines in healthy young men. *J Endocrinol Invest*. 2016;39:383–8.

44. Domin R, Dadej D, Pytka M, Zybek-Kocik A, Ruchala M, Guzik P. Effect of various exercise regimens on selected exercise-induced cytokines in healthy people. *Int J Environ Res Public Health*. 2021;18:1261.

45. Pedersen BK, Saltin B. Exercise as medicine—evidence for prescribing exercise as therapy in 26 different chronic diseases. *Scand J Med Sci Sports*. 2015;25:1–72.

46. Khalafi M, Aria B, Symonds ME, Rosenkranz SK. The effects of resistance training on myostatin and follistatin in adults: a systematic review and meta-analysis. *Physiol Behav*. 2023;269:114272.

47. Abe T, Kearns CF, Fukunaga T. Sex differences in whole body skeletal muscle mass measured by magnetic resonance imaging and its distribution in young Japanese adults. *Br J Sports Med*. 2003;37:436–40.

48. Araujo AB, Chiu GR, Kupelian V, Hall SA, Williams RE, Clark RV, et al. Lean mass, muscle strength, and physical function in a diverse population of men: a population-based cross-sectional study. *BMC Public Health*. 2010;10:508.

49. Kim JS, Cross JM, Bamman MM. Impact of resistance loading on myostatin expression and cell cycle regulation in young and older men and women. *Am J Physiol Endocrinol Metab*. 2005;288:E1110–9.

AUTHOR CONTRIBUTIONS

MNB was responsible for data analysis, data interpretation, drafting and finalizing the manuscript. KN was responsible for recruiting participants, data collection, and manuscript review. JT was responsible for data collection, biological sample processing and manuscript review. RAH and XW conceived the project, secured funding, contributed to data collection/analysis, data interpretation, drafting the manuscript and finalizing the manuscript.

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COMPETING INTERESTS

The authors declare no competing interests.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

All study protocols were performed in accordance with the human subjects regulations and approved by the Institutional Review Board at Augusta University (IRB#1323570). Informed consent was obtained from all participants.

ADDITIONAL INFORMATION

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