Combined effects of whole-body vibration, resistance exercise, and vascular occlusion on skeletal muscle and performance

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Abstract

The purpose of this study was to evaluate the effects of a new high-intensity training modality comprised of vibration exercise with superimposed resistance exercise and vascular occlusion (vibroX) on skeletal muscle and performance. Young untrained women were randomized to either train in a progressive mode on 3 days per week for 5 weeks ($n = 12$) or to maintain a sedentary lifestyle ($n = 9$). VibroX increased peak cycling power ($+9\%, P = 0.001$), endurance capacity ($+57\%, P = 0.002$), ventilatory threshold ($+12\%, P < 0.001$), and end-test torque ($+15\%, P = 0.002$) relative to the sedentary group. Training load increased by 84.5\% ($P < 0.001$) after vibroX. The increases were paralleled by increases in myosin heavy
chain type 1 vastus lateralis muscle fiber cross-sectional area (+14%, \( P = 0.031 \)) and proportion (+17%, \( P = 0.015 \)), thigh lean mass (+4%, \( P = 0.001 \)), capillary-to-fiber ratio (+14%, \( P = 0.003 \)), and cytochrome c oxidase activity. Conversely, maximal values for oxygen consumption, cardiac output, isokinetic leg extension power and jumping power remained unaffected. Notably, vastus lateralis muscle adaptations were achieved with a very low weekly training volume. We conclude that vibroX quickly increases muscle (fiber) size, capillarization, and oxidative potential, and markedly augments endurance capacity in young women.

**Key words:**
Ischemia, endurance exercise, whole-body vibration, capillarization, vascular occlusion, resistance exercise

**Introduction**
Resistance and endurance exercise produce widely diversified adaptations, with little or no overlap between them. Resistance exercise typically results in increases in maximal muscle power and skeletal muscle mass [26], with little or no increases in maximal oxygen consumption [7] and overall capillary-to-fiber ratio [3, 26]. In contrast, endurance exercise induces increases in maximal oxygen consumption, muscle mitochondrial biogenesis, oxidative enzymes, and capillarization, leading to an increased submaximal exercise capacity [13], with little or no hypertrophy or strength gains.

In sports, rehabilitation, and medicine, a combination of strength and endurance exercise is required to improve performance, restore function, and preserve or restore metabolic health, respectively. However, when resistance and endurance training are performed
simultaneously, *i.e.* when endurance exercise is performed immediately (*i.e.* minutes or a few hours) after resistance exercise, a potential interference in strength development takes place, making such a combination less effective relative to the isolated training form [12, 19]. Thus, there exists an obvious need for new or modified training modalities that can at the same time mediate adaptations both in the strength and endurance domains in a time efficient way.

Therefore, we aimed at engineering an efficient training method capable of concurrently promoting strength and endurance adaptations. To this end, we synthesized a stimulus that was comprised of whole-body vibration with superimposed loaded squats (or heel rises) and sustained vascular occlusion, subsequently termed vibroX (vibration + resistance + occlusion + exercise). These components were chosen because a) vibration has been shown to induce angiogenic factors [24], b) resistive exercise usually mediates an increase in neuromuscular strength and hypertrophy, and c) exercising under ischemic conditions can amplify capillarization, oxidative enzyme activity and endurance capacity [25]. We then determined how a 5-week training regime influenced the mean response in structural and functional measures of neuromuscular strength and endurance in young women. A group of young women without exercise were measured over the same period to account for any systematic biases in measurements that might occur during the acquisition of muscle tissues, tissue processing, body composition assessment, and functional testing. Once the proof of concept that strength and endurance can simultaneously be increased with vibroX is established, we can identify the critical steps mediating this response.

**Methods**

**Participants**
We recruited 21 sedentary young women by placard and randomly assigned them to 2 groups (vibroX: 12 women; no training [CON]: 9 women). Participants in the vibroX group were 23.5 ± 3.0 years old and weighed 60.9 ± 8.1 kg. Their body mass index was 21.6 ± 2.6 kg·m$^{-2}$, and maximal oxygen consumption was 2.21 ± 0.25 l·min$^{-1}$. Participants in the CON group were 24.5 ± 3.8 years old, weighed 60.7 ± 5.4 kg, had a body mass index of 21.8 ±1.7 kg·m$^{-2}$, and their maximal oxygen consumption was 2.21 ± 0.48 l·min$^{-1}$. These characteristics were not different between groups. All study participants were healthy. Nine (vibroX) and 7 (CON) women were taking oral contraceptives. After completing a routine health questionnaire, the participants were informed about the procedures applied and about the associated risks. The participants then signed an informed consent. The experimental protocol was approved by the local ethics committee, and the study was performed in accordance with the Ethical Standards in Sport and Exercise Science Research involving human participants [10].

**Study design and exercise protocol**

The study consisted of 1) pretests; 2) 5 weeks of vibroX with progressively increasing training load or a similar period without exercise for CON; and 3) posttests with concurrent training at unchanged loads to retain possible training adaptations for vibroX (Fig. 1). During the study, macronutrient intake was not monitored, and participants were not given any food or dietary supplements.

*Pre- and posttests*

Before and after vibroX and CON we performed baseline measurements to assess maximal oxygen consumption, maximal cardiac output, cycling endurance capacity, isokinetic leg
extension fatigue resistance, training load, thigh lean mass, as well as vastus lateralis muscle fiber size, fiber phenotype and capillarization. Before the pretests, participants could familiarize themselves with the training and testing procedures on 2 separate occasions.

Exercise regimen

Participants of the vibroX group performed 3 supervised progressive training sessions per week on alternate days for 5 weeks (16 training sessions), followed by 3 training sessions with the same load and time under tension (TUT) as during the last progressive training session (Fig. 1). At the beginning of each exercise session, participants performed a warm-up (3 min), which involved squat and upright heel rise movements on a Galileo® side-alternating vibration plate (Novotec, Pforzheim, Germany).

The warm-up was followed by 6 sets of vibroX: loaded (Multipower®, Technogym, Gambettola, Italy) squats (3 sets) and heel rises (alternating between 2 sets of upright heel rises and 1 set of seated heel rises or vice versa) were performed while standing without footwear on the Galileo® vibration plate oscillating at 30 Hz. A frequency of 30 Hz has been shown to be the optimal frequency for producing the greatest magnitude of response in EMG activity of vastus lateralis muscle in an isometric half-squat position [2].

For both, squat and heel rise exercises (3 sets each), a duty cycle of 4 min on and 1 min off was employed. Participants rested for 5 min between squats and heel rises. Three sets per muscle group were chosen in order to attain occlusion times (i.e. 12 min per session) that were in-between of those previously published [20, 21]. Vascular occlusion during the on phase of the duty cycles was induced by inflating tourniquet cuffs (VBM, Sulz a.N., Germany) affixed to the inguinal fold region of the thigh, or to the proximal portion of the
lower leg, centered in the space between the superior aspect of the gastrocnemius muscle and the inferior edge of the patella to suprasystolic pressure, i.e. 197 ± 3 mmHg (26.26 ± 0.40 kPa). The suprasystolic pressure employed here was the highest pressure that was tolerated by the participants for the duration of the on phase of the duty cycles (4 min each). For both, squat and heel rise exercises, tourniquet cuffs were inflated right before the first set and remained inflated until the 4 min of vascular occlusion were complete. The cuffs were then deflated to a pressure of 100 mmHg (13.33 kPa), and the participants rested for 1 min before the cuffs were quickly reinflated for the subsequent set. Right upon cuff inflation, Galileo® vibration was initiated in parallel with squat or heel rise exercise.

Load magnitude was adjusted from prior knowledge (familiarization sessions) in order to induce volitional failure within 40–60 s of exercise (i.e. 1 set was comprised of 4–6 repetitions with 4 s concentric, 2 s isometric and 4 s eccentric action per repetition). Loads were adjusted progressively during the training period, i.e. in every training session either TUT or load magnitude were increased. Load magnitudes during the first training session corresponded to 57.5 ± 16.0 % and 61.7 ± 21.1 % body mass for the first set of squat and upright heel rise exercise, respectively. For squat sets 2 and 3 the loads were reduced by 10.0 ± 4.0 % and 15.9 ± 6.5 % body mass. Load reduction was 6.8 ± 4.1 % body mass for heel rise sets 2 and 3.

**Experimental procedures**

*Muscle biopsy analyses*

During the pre- and posttesting period of time, we obtained a percutaneous biopsy after local anesthesia with 1% lidocaine from the middle portion of the nondominant vastus lateralis muscle, using a ProMag Ultra device and 14 gauge needles (Angiotech Pharmaceuticals,
Gainesville, FL, USA), as previously described [28]. After removal, the muscle tissue was immediately mounted in an embedding medium (Tissue-Tek®, Sakura, Zoeterwoude, The Netherlands), snap frozen in isopentane cooled to −160 °C with liquid nitrogen, and subsequently stored at −80 °C until use. Consecutive 12 μm sections were cut on a microtome at −25 °C and mounted on glass cover slides for further histochemical and immunohistochemical analyses. For all fiber analyses, only fibers fully encircled by adjacent fibers were evaluated. Measurements were made for at least 50 of each of the main fiber types [i.e. myosin heavy chain (MYH) isoform type 1 and 2]. We stained the serial cryocut cross-sections using the myofibrillar adenosinetriphosphatase (mATPase) method after acid (pH 4.3 and 4.6) and alkali (pH 10.5) preincubation according to Guth and Samaha [9], with minor modifications [18]. Subsequently, we classified the muscle fibers according to their MYH isoform into MYH-1 and MYH-2.

For the analysis of oxidative enzyme activity we incubated consecutive sections in media containing cytochrome c oxidase. The monoclonal mouse anti-human CD31 endothelial cell antibody (DAKO, Carpinteria, Canada, 1:600 dilution) was used as a marker for muscle capillaries, and capillary-to-fiber ratio was calculated by dividing the number of CD31-positive cells by the number of muscle fibers. For all histochemical and immunohistochemical analyses, we used the NIH Image J software (version 1.41o, National Institutes of Health, Bethesda, MD, USA). Arbitrary cytochrome c enzyme activity levels were derived from the measured mean optical density pixel values of the muscle fibers normalized to the background pixel values on the same section.

**Dual-energy X-ray absorptiometry (DXA)**

We used DXA (Lunar iDXA™, GE Healthcare, Madison, WI, USA) to determine the lean
mass of both thighs according to the manufacturer’s specifications.

**Training**

Training loads and number of completed repetitions were continuously recorded. Training performance was retrospectively assessed by comparing loads and repetitions for the first set of vibroX squat exercise between the first and last progressive training session. These measures were chosen because 1-repetition maximum testing is not applicable to vibroX. During the third and last training session, we continuously recorded heart rate (S610i, Polar Electro, Kempele, Finland), and measured blood lactate concentration by drawing 20 µl of arterialized venous blood from an earlobe at rest, after the squat routine, after the heel rise routine, and 10–15 min after exercise. Blood samples were analyzed enzymatically amperometrically with a BIOSEN C_line Sport® (EKF-diagnostic, Barleben, Germany). In addition, we assessed perceived exertion and perceived pain after the third squat and heel rise duty cycle using a visual analogue scale (VAS). The VAS scale consisted of a horizontal line. The word “none” was placed at the left end of the scale, and “very severe” was placed at the right end of the scale. The VAS was scored from 0 to 10, but the participants were unaware of the numbers.

**Isokinetic dynamometry**

We tested maximal knee extension power and fatigue resistance of the nondominant leg using a commercially available dynamometer (Con-Trex MJ, CMV, Dubendorf, Switzerland). Participants performed 2 maximal knee extensions (\(\omega = 3.14 \text{ rad} \cdot \text{s}^{-1}\)), separated by 1 min, to assess maximal power. Subsequently, a fatigue test consisting of 50 alternate maximal voluntary knee extensions and knee flexions (\(\omega = 3.14 \text{ rad} \cdot \text{s}^{-1}\)) was performed to assess end-test leg extension torque (mean value over the last 5 repetitions) and to calculate fatigue
percentage \([(\text{mean torque value of the first 5 maximal repetitions} - \text{end-test torque})/(\text{mean torque value of the first 5 maximal repetitions}) \cdot 100]\).

**Jumping mechanography**

Three vertical countermovement jumps with freely moving arms (separated by 30 s of rest) were performed on a strain gauge ground reaction force platform (Leonardo Mechanograph\textsuperscript{®}, Novotec, Pforzheim, Germany) linked to a desktop computer using an integrated analog digital board and software system (Leonardo Mechanography GRFP version 4.2, Novotec, Pforzheim, Germany) to assess maximal jumping power.

**Graded cycling exercise tests**

We used Innocor\textsuperscript{TM} (Innovision, Odense, Denmark) to determine maximal cardiac output by inert gas rebreathing, oxygen consumption and ventilation by breath-by-breath ergospirometry, as well as arterial oxygen saturation and heart rate during a graded cycling exercise test as previously described [6]. Ventilatory threshold was determined as the power corresponding with a disproportionate increase in minute ventilation.

**Constant-load cycling exercise tests**

We used a constant-load cycling exercise test to assess time to exhaustion at submaximal power as an indicator of endurance capacity. Power at 85\% maximal power of the first cycling exercise test was sustained until volitional exhaustion, *i.e.* the point in time at which the participants stopped pedaling or were no longer able to maintain pedal rate within the required limits.

**Statistics**
We report all group data as mean ± SD. Structural and functional data (except for training load) were analyzed by using a two-way repeated-measures analysis of variance (ANOVA). The factors were trial (pretraining, posttraining) and condition (vibroX, CON). Significant differences were subsequently analyzed by using Tukey’s honestly significant difference post hoc test. Training load data were analyzed by using a paired sample Student’s t-test. We used STATISTICA Version 7.0 (Statsoft, Tulsa, OK, USA) for all statistical analyses, and statistical significance was set at $P < 0.05$.

**Results**

**Training**

Maximal heart rate achieved during vibroX was 94.1% and 91.4% of maximal heart rate during a graded cycling exercise test for pre- and posttraining measurements, respectively (Fig. 2A). Maximal blood lactate concentration during vibroX was 77.3% and 75.4% of maximal blood lactate concentration during a graded cycling exercise test for pre- and posttraining measurements, respectively (Fig. 2B). Perceived exertion and pain were high and did not change over time, indicating that participants exercised with maximum voluntary effort (Fig. 2C). Perceived exertion during vibroX was similar to maximal exercise during a graded cycling exercise test to exhaustion, in which perceived exertion was rated 9.7 ± 0.9 and 9.5 ± 1.2 on a modified Borg scale for pre- and postmeasurements, respectively. During the period of time between the first and last progressive training session, vibroX participants increased the training load by 84.5%, while the number of repetitions (and as such TUT) until volitional failure was the same at both points in time (Fig. 2D).

**Muscle (fiber) size, metabolism, and capillarization**

VibroX resulted in a 16.7% and 13.8% increase in vastus lateralis muscle fiber CSA for
MYH type 1 and 2 fibers, respectively (Fig. 3A,B). However, a testing time by group interaction was observed only for MYH-1 fiber hypertrophy ($P = 0.010$), demonstrating that vibroX increased MYH-1 fiber CSA relative to CON. Concordantly, thigh lean mass increased by 4% in the vibroX group [vibroX: 10.6 vs. 11.0 kg ($P = 0.001$); CON: 10.7 vs. 10.7 kg ($P = 1.000$); testing time by group interaction: $P = 0.008$] (Fig. 3C). Notably, MYH-1 fiber proportion and overall capillary-to-fiber ratio were increased by 14.0% each (Fig. 3D). Furthermore, cytochrome c oxidase activity increased in vibroX, reflecting the shift in the MYH-1 and MYH-2 fibers' metabolic phenotype towards an oxidative phenotype (Fig. 3E,F).

*Submaximal and maximal power, time to exhaustion, and end-test torque*

Maximal power during a graded cycling exercise test was increased by 8.7% in vibroX relative to CON (Fig. 4A). VibroX also increased time to exhaustion during a constant-load cycling test and ventilatory threshold (graded cycling exercise test) by 57.0% and 11.7%, respectively (Fig. 4B,C), relative to CON. Additionally, vibroX improved concentric isokinetic leg extension fatigue resistance by 15% (Fig. 4D), while neither maximal concentric isokinetic knee extension power nor maximal jumping power per kg body mass was increased after vibroX (Fig. 4E,F). Body mass remained unchanged for both groups [vibroX: 60.9 ± 8.1 vs. 61.1 ± 8.0 kg, $P = 0.680$; CON: 60.7 ± 5.4 vs. 60.7 ± 5.6 kg, $P = 1.000$].

*Cardiac output, oxygen consumption, and ventilation*

VibroX increased maximal cardiac output (albeit without significant testing time by group interaction) (Table 1), while no change in maximal oxygen consumption, calculated arteriovenous oxygen difference, or arterial oxygen saturation was observed for either group.
Higher maximal power in vibroX had no effect on ventilation, while a lower maximal power correlated with a reduction in ventilation in CON (Table 1).

**Discussion**

This study showed that 5 weeks of progressive vibroX significantly increased maximal cycling power, ventilatory threshold, time to exhaustion at submaximal cycling power, end-test torque after 50 maximal concentric isokinetic leg extensions, and training load in young women. However, vibroX had no effect on their maximal jumping and concentric isokinetic leg extension power. Increased endurance capacity and strength were commensurate with increased vastus lateralis muscle MYH-1 fiber proportion and hypertrophy, oxidative enzymes, and overall capillarization, while maximal oxygen consumption remained unchanged. Our results demonstrate that vibroX is an effective training modality to rapidly increase submaximal power capacity and neuromuscular strength in young women at the same time. Furthermore, given that performance improvements were achieved with a minimal weekly training volume, time efficiency of vibroX was high.

VibroX is a training modality which required maximum effort from participants. This was evident from the high ratings of perceived exertion and pain, the high peak blood lactate concentration, and the high peak heart rate. In return, time commitment for vibroX was very low, corresponding to approximately 15 min per training session. The increase in neuromuscular strength after vibroX was substantial, as indicated by the +85% increase in squat training load. This increase in training load correlated with increases in muscle fiber CSA and thigh lean mass. VibroX increased CSA of type 1 and 2 fibers to a similar extent (17% and 14% for type 1 and 2 fibers, respectively). However, only MYH-1 fiber hypertrophy was significant between groups over time (Fig. 3A,B), indicating that 5 weeks of
vibroX preferentially promoted type 1 fiber hypertrophy. Notably, vibroX mediated a fast adaptation in muscle fiber CSA, considering that even in the case of high-resistance exercise with different exercise modes (open- and closed-chain) and larger training volume, increases in CSA of vastus lateralis muscle fibers in young women usually require at least 6–8 weeks of training [22, 23].

DXA thigh lean mass increased by approximately 4% in 5 weeks, lending further credence to our finding that vibroX induced muscle fiber hypertrophy. Assuming that segmental training-induced increases in DXA muscle mass are proportional to total lean mass increases after whole-body exercise, an increase of 2.5–3.0% in DXA thigh lean mass can be expected after 10–12 weeks of high-resistance exercise when the participants' diets are not supplemented with protein in the postexercise period [5, 15]. Our finding that the participants undergoing vibroX increased thigh lean mass by 4% in 5 weeks without supplementing protein and furthermore using a very small training volume indicated that vibroX stimulated muscle protein synthesis, either in the immediate post-exercise period and/or in the days after exercise (with or without satellite cell recruitment), leading to a net muscle protein balance.

In addition to the increases in muscle hypertrophy and training load, vibroX concurrently increased MYH-1 fiber proportion, overall capillary-to-fiber ratio, and oxidative enzyme activity. One mechanism possibly orchestrating these muscular adaptations is the activation of peroxisome proliferator-activated receptor γ coactivator 1α (PGC-1α) through multiple isoforms. PGC-1α is a transcriptional coactivator that powerfully regulates many aspects of skeletal muscle oxidative metabolism, including fiber-type switching, mitochondrial biogenesis, and angiogenesis [16, 29]. It has recently been demonstrated in mice that exercise-induced activation of β-adrenergic signaling leads in skeletal muscle to robust induction of PGC-1α from an alternative promoter, which then acts through the orphan
nuclear receptor ERRα (estrogen receptor α) to activate a broad program of angiogenesis, including the induction of VEGF [4]. On the basis of these observations and the data presented here, we speculate that vibroX caused marked activation of the sympathetic nervous system, which activated PGC-1α, which in turn mediated the ‘pleiotropic’ effects of vibroX.

The cellular/organic changes were paralleled by an increase in end-test torque following 50 consecutive maximal isokinetic knee extensions. End-test torque has been shown to correlate with the estimated critical torque [1], which in turn has been suggested to be analogous to the critical power for cycling exercise [1, 14]. Consistent with the notion that dynamometry-derived critical torque is analogous to critical power for cycling exercise, both the increase in ventilatory threshold and the markedly (+57%) increased time to exhaustion during a constant-load cycling test could be explained by an increase in critical power in this study. Notably, we found that endurance capacity could be increased without concurrently increasing maximal oxygen consumption, supporting the conclusion of other authors that the percentage change in submaximal performance is not associated with the percentage change in maximal oxygen consumption [27]. However, we observed that maximal cardiac output increased in the group performing vibroX, albeit without inter-group difference. Probably, this increase was too small for both, a significant inter-group difference and an increase in maximal oxygen consumption. Maybe, a longer training period (8–10 weeks) would have been necessary to observe significant inter-group effects at the systemic cardio-circulatory level.

In contrast to the marked increases in the capability of participant to generate and sustain submaximal power, maximum voluntary isokinetic leg extension power and jumping power
remained unchanged. The fact that maximal concentric isokinetic leg extension power remained unchanged after vibroX indicates that muscle force at this angular velocity ($\omega = 3.14 \text{ rad}\cdot\text{s}^{-1}$) also remained constant (power = force \cdot velocity). Unchanged muscle force can be explained by a concurrent increase in MYH-1 fiber CSA and proportion after vibroX. In fact, the increase in maximal concentric isokinetic force due to an increase in the number of parallel sarcomeres within MYH-1 fibers might have been blunted by the concurrent increase in the share of the muscle CSA consisting of MYH-1 fibers, because MYH-1 fibers have reduced isometric maximal tension [17] relative to MYH-2 fibers. Moreover, MYH-1 fibers display lower maximal shortening velocity and power than MYH-2 fibers [17]. Thus, despite the higher MYH-1 fiber power due to hypertrophy, mixed muscle maximal shortening velocity might be reduced, possibly explaining the unchanged maximal jumping power after vibroX.

We acknowledge that the level of involvement of those women who participated in pre- and post-testing might have been less than that of the exercise group. Thus, our experimental design did not permit adequate assessment of possible ‘reactivity’/placebo effects on performance tests. However, we reported significant cellular and organic changes that clearly cannot be explained by any ‘reactivity’/placebo effect, and which coincided with the observed performance improvements. We interpreted this to mean that performance improvements were a consequence of the cellular and organic adaptations.

In summary, 5 weeks of vibroX concurrently increased power and ventilatory threshold during a graded cycling exercise test, time to exhaustion during a constant-load cycling test, end-test torque after repeated maximal leg extensions, and squat training load. These increases were related to increases in muscle hypertrophy, capillarization, and oxidative enzymes, but not maximal oxygen consumption. We conclude that vibroX is an effective and
time efficient training modality to quickly increase muscle (fiber) size, capillarization, and oxidative potential, and to markedly augment endurance capacity, local fatigue resistance and neuromuscular strength in young women. Thus, we provided for the first time a proof of concept that strength and endurance can be effectively and efficiently increased at the same time. VibroX or derivatives thereof may not only be of interest in clinical settings (deconditioned individuals), but also for highly trained endurance athletes for whom traditional HIT [8] has lost most of its potency. In view of the variously described notion, that adaptations to exercise training and resultant performance improvements are highly specific to the mode of activity [11], it is now worthwhile investigating the molecular bases underlying vibroX adaptations.
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**Figure legends**

*Fig. 1.* Overview of experimental protocol on a weekly scale (−2 to 7). B, biopsy; F, familiarization session with respect to training and testing; Q, graded cycling exercise test; E, constant-load cycling exercise test; C, dual-energy X-ray absorptiometry; M, muscle testing (mechanography and isokinetic dynamometry); T 1–16, training sessions with progressive training load; t 1–3, training sessions with constant (same as during T 16) training intensity. vibroX, Galileo® vibration + resistance exercise + vascular occlusion.

*Fig. 2.* Peak heart rate (A) and blood lactate concentration (B) during vibroX compared to the maximal values obtained during the graded cycling exercise test (GXT) at the beginning and end of the study. Perceived exertion and pain during the 3rd vibroX squat set at the beginning and end of the study (C). Training load and number of repetitions (9–10 s per repetition) during the first squat set of the first and last progressive training session (D). Bars and error bars represent mean values and standard deviations, respectively, for the 12 women undergoing vibroX. vibroX, Galileo® vibration + resistance exercise + vascular occlusion. ***, significantly different first vs. last training session at *P* < 0.001.

*Fig. 3.* Muscle fiber cross-sectional area (CSA) for myosin heavy chain type 1 (A) and 2 (B) fibers, overall capillary-to-fiber ratio (C), myosin heavy chain type 1 fiber percentage (D), and cytochrome c (cyt c) oxidase activity in optical arbitrary units (AU) for type 1 (E) and 2 (F) fibers pre- and posttraining. Bars and error bars represent mean values and standard deviations, respectively, for: (A) and (B), 8 women per group; (C), 7 women per group; (D), 6 and 5 women in vibroX and CON, respectively; (E) and (F), 8 and 7 women in vibroX and CON, respectively. vibroX, Galileo® vibration + resistance exercise + vascular occlusion;
CON, no exercise. *, **, significantly different within group pre vs. post, \( *P < 0.05, **P < 0.01; \uparrow \), pre vs. post differences significantly different between groups at \( P < 0.05 \).

*Fig. 4.* Maximal power during the graded cycling exercise test (GXT, A), time to exhaustion during a constant-load cycling exercise test (CLT, B), ventilatory threshold calculated based on gas exchange during GXT (C), knee extension (KE) fatigue following 50 repeated maximal concentric isokinetic (\( \omega = 3.14 \text{ rad} \cdot \text{s}^{-1} \)) knee extensions (D), maximal concentric isokinetic knee extension power (E), and maximal countermovement jump (CMJ) power per kg body mass (F) pre- and posttraining. Bars and error bars represent mean values and standard deviations, respectively, for: (A), (B), (C), and (F), 12 and 9 women in vibroX and CON, respectively; (D) and (E), 10 and 7 women in vibroX and CON, respectively. vibroX, Galileo© vibration + resistance exercise + vascular occlusion; CON, no exercise. *, **, ***, significantly different pre vs. post within group, \( *P < 0.05, **P < 0.01, ***P < 0.001; \uparrow \uparrow \), \uparrow\uparrow\uparrow, pre vs. post differences significantly different between groups, \( \uparrow\uparrow P < 0.01, \uparrow\uparrow\uparrow P < 0.001 \).
Table 1. Peak cardiac output, oxygen consumption, and ventilation during GXT

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<th>vibroX</th>
<th>CON</th>
<th>vibroX vs. CON</th>
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<td></td>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
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<tr>
<td>Cardiac output (l/min⁻¹)</td>
<td>12.3 ± 2.2</td>
<td>13.4 ± 1.7*</td>
<td>11.9 ± 1.7</td>
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<tr>
<td>Arterio-venous oxygen difference (ml O₂/100 ml⁻¹)</td>
<td>18.2 ± 2.4</td>
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<td>Arterial oxygen saturation (%)</td>
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<td>Oxygen consumption (l/min⁻¹)</td>
<td>2.21 ± 0.25</td>
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<td>Ventilation (l/min⁻¹)</td>
<td>79 ± 15</td>
<td>83 ± 13</td>
<td>78 ± 16</td>
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Values are means ± SD for 12 women (vibroX) and 9 women (CON). GXT, graded cycling exercise test; vibroX, Galileo® vibration + resistance exercise + vascular occlusion; CON, control group. *, **, significantly different pre vs. post within group, *P < 0.05, **P < 0.01; †, pre vs. post differences significantly different between groups at P < 0.05.