RECOVERY OF VASCULAR FUNCTION AFTER EXPOSURE TO A SINGLE BOUT OF VIBRATION

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1. INTRODUCTION

Job rotation has been used to reduce worker exposure to hand-transmitted vibration and the risk of developing hand-arm vibration syndrome (HAVS). However, there are no studies indicating what the best rotation schedule is. Immediately following a single exposure to vibration, transient changes in finger blood flow (1) and shifts in vibrotactile sensitivity (2) can be measured in humans. In rats, exposure to a single bout of vibration also results in immediate changes in blood flow, transient shifts in the sensitivity of large myelinated fibers to electrical stimulation (3), and changes in vascular responsiveness to vasoconstriction agents (4). However, it is not known if there are longer-term effects of a single vibration exposure that may affect vascular or sensorineural responsiveness to subsequent exposures. The goal of this study was to use a model of vibration-induced dysfunction to determine if there are residual effects of a single exposure to vibration on peripheral vascular function, and if there are residual effects, the number of days of rest needed for vascular function to recover after a single exposure.

2. METHODS

2.1. Exposure

Male Sprague-Dawley rats [Hla:(SD) CVF rats; 6 weeks of age at arrival; Hilltop Lab Animals, Inc, Scottdale, PA;] were used in this study. Animals were maintained in an AALAC accredited vivarium under a 12:12 LD cycle(lights on 0700 h) with food and water available *ad libitum*. Rats were acclimated to the laboratory for 1 week prior to the beginning of the experiment. On the first day of the experiment, rats were restrained in Broome style restrainers. Each rat had their tail secured to a platform as previously described. Half of the rats were exposed to a single 4h bout of vibration (125 Hz, 49 m/sec^2 rms). The remaining rats served as controls. Control rats had their tails secured to a platform mounted on isolation blocks. Rats were anesthetized using pentobarbital (100 mg/kg, i.p.) and euthanized by exsanguination 1, 2 or 7 days following the exposure. All procedures were approved by the NIOSH Animal Care and Use Committee and were in compliance with CDC and NIH guidelines for the care and use of laboratory animals.

2.2. Vascular Physiology

After dissection, each ventral tail artery was mounted and pressurized (60 mmHg) in a micro-vessel chamber (Living Systems, Burlington VT). Vasoconstriction in response to the α2C-adrenoreceptor agonist UK14304 and the α1-adrenoreceptor agonist phenylephrine (PE) were assessed in separate artery segments. To assess endothelial-mediated vasodilation, phenylephrine, constricted arteries were re-dilated with acetylcholine (ACh). All vaso-modulating factors were added in half-log increments and the internal diameter of the artery was measured after each application of the drug.

2.3. Data Analyses

All data are expressed as a percent change from baseline. Data were analyzed using 2-way repeated measures ANOVAs. Differences were considered significant if $p < 0.05$.

3. RESULTS

One day following exposure to vibration, vasoconstriction in response to UK-14304 was enhanced in vibrated arteries as compared to controls (Figure 1). ACh-mediated vasodilation was not different between the two groups (Figure 2).

Figure 1. One day after exposure, arteries from vibrated rats displayed increased responsiveness to UK14304-mediated constriction (* greater than vibrated arteries, p <0.05).

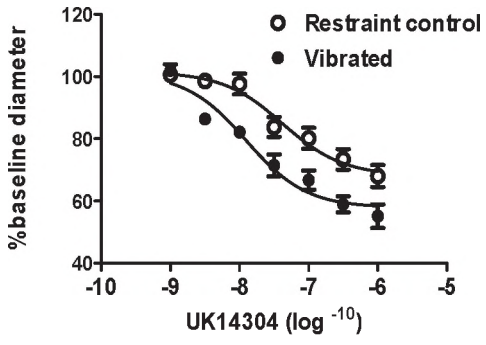
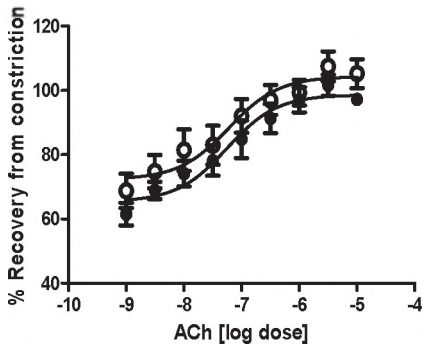
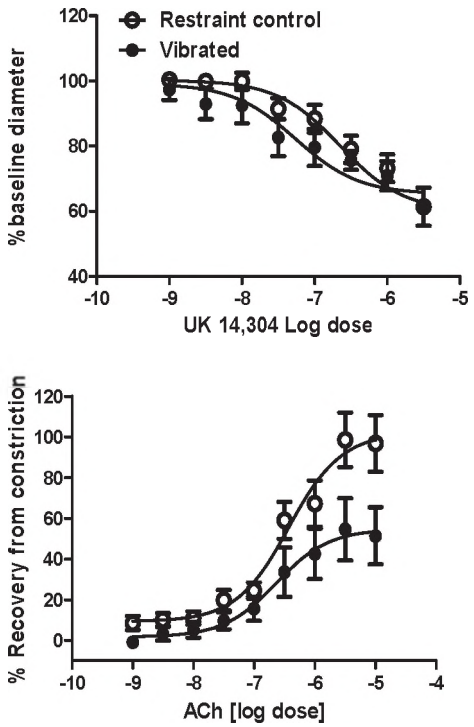


Figure 2. One day after exposure, arteries from vibrated rats displayed unaffected ACh-mediated vasodilation.



After two days of recovery, UK14304-mediated vaso-constriction had returned to control levels (Figure 3, top), but ACh-mediated vasodilation was significantly reduced in arteries collected from vibrated rats (Figure 3, bottom). Responses to vasoconstricting and vasodilating factors were back to control levels after 7 days of recovery (data not shown).

Figure 3. Two days following vibration exposure, vascular responsiveness to UK14304 was similar in control and exposed rats (Top). However, ACh-mediated vasodilation was reduced in vibrated arteries (Bottom;* greater than vibrated arteries, p <0.05).



4. DISCUSSION AND CONCLUSIONS

- Changes in adrenoreceptor-mediated vasoconstriction are apparent for the first 24 h following a single exposure to vibration.
- Endothelial-mediated vasodilation is not affected until 48 h after vibration exposure.
- Changes in responsiveness to vasoconstricting and vasodilating factors may affect vascular responses to subsequent vibration exposures.
- Vascular responsiveness returns to control levels after a 7-day recovery period.
- Additional time-points need to be assessed, but these findings suggest that it may take up to seven days for vascular function to recover after exposure to a single bout of vibration.
- The acute responses of the human peripheral vascular system to vibration are similar to the responses of rats. Thus, it is likely that finger vascular function may also be altered for at least two days following exposure to vibration.
- Work rotation schedules that allow more than two days of recovery between subsequent bouts of vibration exposure may help reduce the risk of developing the vascular dysfunction that is characteristic of HAVS.

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