HHS Public Access

Author manuscript

J Toxicol Environ Health A. Author manuscript; available in PMC 2016 January 05.

Published in final edited form as:

J Toxicol Environ Health A. 2015 ; 78(9): 571–582. doi:10.1080/15287394.2015.1014079.

ANTIVIBRATION GLOVES: EFFECTS ON VASCULAR AND SENSORINEURAL FUNCTION, AN ANIMAL MODEL

K. Krajnak, **S. Waugh**, **C. Johnson**, **R. G. Miller**, **D. Welcome**, **X. Xu**, **C. Warren**, **S. Sarkisian**, **M. Andrew**, and **R. G. Dong**

Engineering and Controls Technology Branch, National Institute for Occupational Safety and Health, Morgantown, West Virginia, USA

Abstract

Anti-vibration gloves have been used to block the transmission of vibration from powered hand tools to the user, and to protect users from the negative health consequences associated with exposure to vibration. However, there are conflicting reports as to the efficacy of gloves in protecting workers. The goal of this study was to use a characterized animal model of vibration-induced peripheral vascular and nerve injury to determine whether antivibration materials reduced or inhibited the effects of vibration on these physiological symptoms. Rats were exposed to 4 h of tail vibration at 125 Hz with an acceleration 49 m/s^2. The platform was either bare or covered with antivibrating glove material. Rats were tested for tactile sensitivity to applied pressure before and after vibration exposure. One day following the exposure, ventral tail arteries were assessed for sensitivity to vasodilating and vasoconstricting factors and nerves were examined histologically for early indicators of edema and inflammation. Ventral tail artery responses to an α2C-adrenoreceptor agonist were enhanced in arteries from vibration-exposed rats compared to controls, regardless of whether antivibration materials were used or not. Rats exposed to vibration were also less sensitive to pressure after exposure. These findings are consistent with experimental findings in humans suggesting that antivibration gloves may not provide protection against the adverse health consequences of vibration exposure in all conditions. Additional studies need to be done examining newer antivibration materials.

Workers who regularly use vibrating powered and pneumatic hand tools may develop a set of symptoms that have been called hand–arm vibration syndrome (HAVS). The primary symptoms of HAVS include cold-induced vasospasms that result in blanching of the fingers and hands, alterations in tactile sensitivity, pain, and reductions in grip strength and manual dexterity (Griffin, 1990). In some cases, antivibration (AV) gloves have been used in an attempt to reduce the injurious effects of vibration. These gloves may diminish the risk of injury by lowering or preventing transmission of vibration energy from the tool to the user (Milosevic and McConville, 2012). However, studies examining the effectiveness of these gloves produced mixed results; in some cases the use of AV gloves appears to have mitigated the effects of vibration exposure (Jetzer et al., 2003; Brown, 1990). Other studies

Address correspondence to K. Krajnak, PhD, Engineering and Controls Technology Branch, National Institute for Occupational Safety and Health, Morgantown, WV 26505, USA. ksk1@cdc.gov.

Color versions of one or more of the figures in the article can be found online at www.tandfonline.com/uteh

suggest that gloves provide little or no reduction in the vibration transmitted to the fingers (Laszlo and Griffin, 2011; Welcome et al., 2014). A number of factors might be responsible for varying results. These factors include (1) vibration frequency at which the gloves were tested, (2) grip and push force used when handling the tool, (3) fit of the glove, and the (4) type of AV material used within the gloves (Hewitt et al., 2015; Dale et al., 2011; Laszlo and Griffin, 2011; Milosevic and McConville, 2012). Even though there is some question regarding the usefulness of AV gloves, the International Standards Organization (ISO) has developed a standard (ISO-10819) that provides recommendations regarding AV glove testing and criteria that may help employers and workers choose appropriate gloves (Hewitt et al., 2015; McDowell et al., 2013; Welcome et al., 2014).

To determine when gloves are appropriate for use, it is important to understand whether they alter the physical response of the glove and hand to vibration and whether these altered responses of the glove and hand affect pathophysiological responses that lead to HAVS. Biodynamic studies performed in humans and estimations of vibration transmissibility made using computational models of the human finger demonstrated that tissue stresses and strains induced by vibration are greatest at or near the resonant frequency (i.e., the frequency at which the amplitude of the tissue displacement is greater than the amplitude of the vibration-transmitting source; Dong et al., 2004; Wu et al., 2006). The resonant frequency of the human finger is in the 100–300 Hz range, depending on a number of factors including the push- or pull-force used when the measurements were made, and where on the fingers the measurements were taken. It was postulated that increased tissue stress and strain that occur within the resonant frequency range enhance the risk of developing HAVS (Dong et al., 2004; Wu et al., 2007). This hypothesis was been supported by human epidemiological data demonstrating acute changes in vascular function (Bovenzi et al., 2004) and that the risk of developing HAVS, is greater when workers are exposed to vibration from tools with a primary frequency in this range (Bovenzi et al. 2011).

In addition, studies using a rat-tail model of vibration-induced dysfunction also demonstrated that vibration-induced changes in sensorineural function are greatest at or near the resonant frequency (Krajnak et al., 2010, 2012). The resonant frequency of the rat tail is in the same range as that of the human finger (i.e., 100–300 Hz; Welcome et al., 2008; Dong et al., 2004). This is also the resonant frequency range of commonly used AV glove materials (Welcome et al., 2014; Xu et al., 2011). Another study using the rat tail model to assess vibration transmissibility through AV-glove material demonstrated that gel and air-bladder AV materials exerted little effect on the transmission of low vibration frequencies (i.e., less than 60 Hz), but AV material did prevent transmission of high-frequency vibration (i.e., greater than 500 Hz) to the tissue.

Based on these data, the rat-tail model was used to test the hypothesis that vibration-induced changes in peripheral vascular and sensorineural function are reduced with the use of AV glove materials, vascular and sensorineural function was examined in the ventral tail arteries by assessing dose-dependent changes in vascular responsiveness to vasocontricting and vasodilating factors and nerves of rats after a single exposure to vibration. Krajnak et al. (2007) also demonstrated that a single exposure to vibration (4 h at 125 Hz, acceleration of 49 m/s^2) resulted in increased sensitivity of ventral tail arteries to α2c-adrenoreceptor-

mediated vasoconstriction, and that this elevation in sensitivity may be due to a vibration-induced rise in oxidative activity in the arteries (Hughes et al., 2009; Krajnak et al., 2010). Krajnak et al. (2007) also demonstrated that a single exposure to vibration results in a transient reduction in the sensitivity of Aβ nerve fibers to transcutaneous electrical stimulation. Aβ fibers carry information regarding mechanical stimulation from the periphery to the central nervous system. These sensory changes in rats are consistent with responses of humans, where a single exposure to vibration results in a transient vasoconstriction in the blood vessels of the fingers and a shift in sensitivity to a vibratory stimulus (Maeda and Griffin, 1997; Maeda et al., 1995; Maeda, 1994). Therefore, in addition to assessing vascular responsiveness, the hypothesis was also tested that because AV glove materials may exacerbate exposure to vibration at the resonant frequency, physiological and biological correlates of injury to mechanoreceptors may still occur, or may be exacerbated by the use of AV glove material.

METHODS

Animals

Male Sprague-Dawley rats [Hla:(SD) CVF rats; 6 wk of age at arrival; Hilltop Lab Animals, Inc., Scottdale, PA) were used in these studies. Rats were maintained in a colony room with a 12:12-h light:dark cycle (lights on 0700 h) with Teklad 2918 rodent diet and tap water available ad libitum, at the National Institute for Occupational Safety and Health (NIOSH) facility, which is accredited by the Association for Assessment and Accreditation of Laboratory Animal Care International (AAALAC). Rats were allowed to acclimate to the lab for 1 wk prior to beginning the study. All procedures were approved by the NIOSH Animal Care and Use Committee and were in compliance with the Public Health Service Policy on Humane Care and Use of Laboratory Animals and the National Institutes of Health (NIH) Guide for the Care and Use of Laboratory Animals.

Vibration Exposures

The equipment and protocol were similar to those described earlier (Welcome et al., 2008; Krajnak et al., 2006). Rats (n = 8/group) were restrained in Broome-style restrainers and placed on a nonvibrating platform housed within a sound-attenuating chamber. The sound-attenuating chamber kept noise levels to approximately 60 dB during the exposure. A metal platform was attached to a shaker, and the tail was secured to the platform using Soft-tape (Neurotron, Inc., Baltimore, MD), which does not stick to the skin. Care was taken to ensure that the tape was not generating too much pressure and restricting blood flow or compressing the tail (Figure 1). Control rats were exposed to identical conditions except that their tails were secured to platforms mounted on isolation blocks. Half of the exposed and control rats had their tails secured to bare platforms and the other half of the rats had their tails restrained to platforms covered with air-bladder material that was obtained from AV air-bladder gloves (Xu et al. 2011). Strips were cut from the middle finger of the glove and secured to the platform using a double-stick adhesive tape (Impacto, Belleville, Canada, product code BF473). The glove material was secured with the leather side down, in contact with the platform. All exposures were performed at 125 Hz and a continuous acceleration of 49 m/s^2 between 0900 and 1300 h. Rats were euthanized 24 h following the exposure by ip

injection of pentobarbital (100 mg/kg body weight; animal weights at the end of the experiment were (mean ± SEM): controls, 263.4 ± 0.34; glove controls, 276.2 ± 0.84; vibrated, 285.3 ± 1.62; glove vibrated, 267.2 ± 0.88) and exsanguination.

Mechanosensitivity

A Randall–Selitto pressure meter was used to test sensitivity to mechanical stimuli. This pressure meter uses forceps with a force transducer. Pressure can be gradually applied and the force is continually recorded. When the rat flicks its tail, the forceps are released and the applied pressure is automatically registered on the meter. To avoid injury, care was taken to not apply more than 200 g of force. To perform the test, rats were placed into restrainers in a quiet area. The tail was centered on the forceps and pressure was gradually applied until the rat flicked its tail. The test was performed three times, with 1 min of rest between repetitions. Rats were then exposed to either vibration or control conditions. Immediately following the exposure, rats went through an additional set of tests. Average scores were calculated from the three measures taken prior to and after the exposure and used for analyses. Rats were returned to their home cages in the colony room immediately following the sensory test.

Tissue Collection

Rats were anesthetized using pentobarbital (100 mg/mg, ip) and euthanized by exsanguination 24 h after vibration or restraint exposure. Skin samples and ventral tail nerves from the C13–15 region were dissected, placed in molds containing Tissue-Tec OTC compound (Waltham, MA), frozen on dry ice, and stored at −80°C until they were prepared for immunohistochemical (IHC) analysis. The ventral tail artery from this region was removed from the tail, cut into two even segments, and each segment was frozen in liquid nitrogen until enzyme-linked immunosorbent assay (ELISA) was performed from hydrogen peroxide and nitrite/nitrate concentrations. The C16–18 region of the tail was placed into cold Dulbecco's modified Eagle's medium (DMEM) until the ventral tail was dissected and was used to assess dose-dependent changes in responsiveness to vasoconstricting and vasodilating factors.

Ventral Tail Nerve Tissue Preparation

Immunohistochemistry (IHC) for albumin, CD68, cyclic-nucleotide phosphatase (CNPase), tumor necrosis factor (TNF)-α, and interleukin (IL)-1β was performed on sections obtained the ventral tail nerve and the skin.

Primary antibodies were purchased from the following vendors and used at the cited concentrations: mouse anti-rat CD68 (AbD Serotec, Raleigh, NC); rabbit anti-rat albumin (Santa Cruz Biotechnology, Santa Cruz, CA, 1:250); mouse anti-CNPase (Sigma-Aldrich, Indianapolis, IN; final dilution, 1:400 dilution); rabbit anti-IL1β (Abcam, Cambridge, MA; 1:500 dilution), rabbit anti-TNFα (Abcam, Cambridge MA 1:600 dilution). The secondary antibody was Cy3-labeled immunoglobulin (Ig) G (Jackson Immuno Research Labs, West Grove, PA), directed against the correct serotype. These antibodies were used at a final dilution of 1:500. All antibodies were diluted in PBS containing 0.4% Triton X-100. Nerve sections (3–4 section/animal, 100 μm between consecutive sections) were centered under the

objective, and images of each nerve section were captured using a Zeiss LSM510 confocal microscope at a final magnification of 45× and ZEN software (Zeiss International, Inc.; Thornwood, NY). ImageJ software was used to measure the density of albumin staining in each image. A threshold was set and the area of each image that was above threshold was measured. The percent of area stained was used for analyses because it incorporates the density of the staining and the area of the region that was stained.

Cross sections (40 μm thick) from frozen skin samples were cut on a cryostat, placed in phosphate-buffered saline (PBS), and stored at −4°C until processed for IHC. Six separate sets of sections were made for each animal. Each set contained 5 sections, and each section in a set was separated by 200 μm from the next. IHC was performed on a single set of free-floating sections taken from each animal using a slightly modified version of a published protocol (Krajnak et al. 2001). Briefly, sections were fixed in 4% paraformaldehyde diluted in 0.01 *M* PBS for 5 min and then rinsed in PBS. Sections were then incubated in 0.3% hydrogen peroxide in methanol for 30 min, rinsed in PBS, and then incubated in primary antibody diluted in PBS plus 0.3% Triton X-100 and 10% normal serum (PBS-Tx) at 4°C with mild agitation. Sections were rinsed in PBS and incubated in Cy3-labeled donkey IgG (Jackson Immunolabs; West Grove, PA) used at a final dilution of 1:500 in PBS plus Triton X-100, and mounted onto slides and air-dried. Cover slips were applied and secured with Prolong Gold (Life Technologies Corporation; Carlsbad, CA). Images were collected on a Zeiss LSM510 confocal microscope. All images were collected at the same depth from each sample using a 10× air objective. Four images were collected at the same depth to ensure that the hair follicles from each sample had similar properties. Photos were imported into ImageJ and defined boxes were made to identify regions around hair follicles and just below the dermis. Threshold levels were set and the density and percent of area stained were quantified using densitometry as described earlier, and the percent of area stained was used for analyses. IHC for albumin and CD68 in the ventral tail nerve was performed and dual IHC for in the tail skin to determine how AV glove materials affected indicators of acute edema and inflammation.

Microvessel Physiology

Tails were dissected from rats after exsanguination and placed in cold Dulbecco's modified Eagle's medium with glucose (Invitrogen/Gibco; Carlsbad, CA). Ventral tail arteries from the C14–15 region of the tail were dissected shortly after euthanasia, mounted on glass pipettes in a microvessel chamber (Living Systems; Burlington, VT), and perfused with bicarbonated HEPES buffer warmed to 37°C. Arteries were pressurized to 60 mm Hg and allowed to equilibrate for approximately 1 h.

Sensitivity to α2C-adrenorecptor-mediated vasoconstriction and acetylcholine (ACh)-induced redilation using the same artery segments—After 1 h in the chamber, the buffer was changed and the pressure was lowered to 10 mm Hg. Pressure was gradually increased (5 mm Hg every 10 s until reaching a final pressure of 110 mm Hg), and changes in the internal diameter of the artery were recorded. Arteries were then assessed for responsiveness to vasoconstricting and vasodilating agents. All chemicals were purchased from Sigma-Aldrich (Indianapolis, IN) unless otherwise noted. For these

studies, arteries were repressurized to 60 mmHg and allowed to stabilize. UK14304-mediated vasoconstriction was measured in the C13–15 region of the artery. UK14304 was applied to the chamber in half-log increments (−8.5 to −5 *M*), and the internal diameter was recorded after vessels stabilized (approximately 5 min between application of doses). The C16–18 region was dissected and used to examine endothelial-mediated vasodilation. Because ventral tail arteries usually display little basal tone, endothelial-mediated redilation was assessed after arteries were constricted to approximately 50% of their baseline diameters with phenylephrine (PE). Krajnak et al. (2006) previously demonstrated that dose-dependent constriction of ventral tail arteries by PE is not altered after a single exposure to vibration. To assess redilation, acetylcholine (ACh) was added in half-log increments (−9.5 to −5 M) using the same procedures that were used to apply UK14304.

Sensitivity to α2C-adrenorecptor-mediated vasoconstriction and acetylcholine (ACh)-induced redilation using different artery segments— Assessing the effects of pressure in Experiment 1 made arteries more sensitive to UK14304-mediated vasodilation. Therefore, another experiment was performed to determine how arteries responded to UK14304 and ACh without assessing the effects of pressure. The procedures used to measure vascular responsiveness were the same as those described above except lower concentrations of UK14304 (−10 to −5 *M*) were used so that one might determine if there were effects of vibration and the use of AV glove materials on sensitivity to α2C-mediated vasoconstriction.

Nitrate/Nitrite (NO_x) and Hydrogen Peroxide (H_2O_2) Assays

Ventral-tail artery samples from the C12–14 region were dissected and immediately frozen. Tissue samples were homogenized in 200 μl lysis buffer (10 m*M* Tris base, 1 m*M* ethylenediamine tetraacetic acid [EDTA], 1 m*M* EGTA, 150 m*M* sodium chloride, 1% Triton X-100) and NO_x and H_2O_2 concentrations were measured using the NO_x colormetric assay (Caymen Chemical Company; Ann Arbor, MI) and the Fluoro H_2O_2 assay (Cell Technology Inc., Mountain View, CA) following the manufacturers' protocols. Protein concentrations were analyzed using the BCA assay (Pierce, Rockford, IL).

Statistical Analyses

Changes in the internal diameter due to the application of UK14304 were calculated using running means. To calculate the running mean, the 100 measurements of the internal diameter acquired immediately prior to application of the next dose of the drug were used. Running means for the UK14304 data were used because the vessels pulse during application of this drug, and the use of these means provides a more accurate measure of the actual diameter of the artery. Arterial responses to ACh-mediated redilation are more stable, and therefore running means did not need to be calculated. Instead, measures of the internal diameter of the artery were recorded just prior to the application of the next dose of the drug. Dose-dependent changes in internal diameter induced either by UK14304 or ACh were analyzed using a 2 (vibrated vs. control) × 2 (glove vs. bare platform) repeated-measures analysis of variance (MANOVA).

The effective dose (ED) 50 for the vasoconstriction induced by UK14304 and vasodilation induced by ACh also was calculated for each animal. A dose response curve and ED50 and ED50 was calculated for each animal and each agent using Prism GraphPad (version 5.0, San Diego, CA). To determine if there was a treatment-related shift in the ED50, a 2 (vibrated vs. control) × 2 (glove vs. bare platform) analysis of variance (ANOVA) was used. Densitometry levels for immunohistochemical labeling, NO_x concentrations, and H_2O_2 concentrations also were analyzed using two-way ANOVAs as described earlier.

For the Randall–Selitto pressure test, pre and post measures of sensitivity were analyzed using paired *t*-tests. Differences with $p < .05$ were considered significant. All analyses, unless otherwise noted, were performed using JMP 10.0.2 (SAS Institute, Atlanta, GA).

RESULTS

Microvessel Responsiveness and Vascular NO_x and H_2O_2 Concentrations

Sensitivity to α2C-adrenorecptor-mediated vasoconstriction and acetylcholine (ACh)-induced redilation using the same artery segments (Experiment 1)—Curves displaying changes in vascular diameter in response to increasing pressure are presented in Figure 2. The pattern of redilation was not markedly different between any of the groups. Analyses of the vasoconstriction induced by the application of UK14304 demonstrated that there was a significant interaction between the condition and the dose. Arteries from rats exposed to vibration on the bare platform displayed a greater constriction to the lowest dose of UK14304 ($10^{-8.5}$ *M*) than rats from the other three groups (Figures 2B and 2C). At higher doses, arteries from the glove control rats constricted less than arteries from the other 3 groups of rats (Figures 2A and 2D). Although arteries from rats exposed to vibration on the bare platform appeared to be less responsive to ACh-induced redilation, there were no significant differences in redilation among the four conditions (Figures 3A–3D). NO_x concentrations were not significantly different between any of the groups, and H_2O_2 levels were not measureable (data not shown).

Sensitivity to α2C-adrenorecptor-mediated vasoconstriction and acetylcholine (ACh)-induced redilation using different artery segments (Experiment 2)—In this experiment, the dose-dependent constriction in response to UK14304 (using 10^{-10}–10^{-5} *M*; Figure 4A) was not markedly different in the different groups of rats. There also were no differences in the dose-dependent redilation in response to ACh between the groups in this study (Figure 4B).

Randall–Selitto Test—Sensitivity to pressure was tested in a subset of the animals (*n* = 3–4/group). Analyses of the average pressure needed to induce a tail response were similar in all groups prior to the exposure. However, after the exposure, rats in both vibration conditions displayed a reduced sensitivity to pressure (i.e., the force needed to induce tail withdrawal was greater; Figure 5).

Immunohistochemistry (IHC)

In Experiment 1, there were not any marked differences in immunostaining for CD68 and albumin in the skin and the nerves was similar in all groups of rats. In Experiment 2 there were no significant differences in the areas stained with tyrosine hydroxylase, caveolin-1, IL-1β staining, or TNFα.

DISCUSSION

The goal of this study was to use a model of acute vibration exposure to determine whether AV materials might reduce the effects of vibration on peripheral vascular and sensorineural function. Rats were exposed to vibration at 125 Hz, which is within the resonant frequency range of both the rat tail and the air-bubble AV material used in this study. Because exposure was in the resonant frequency range of both the glove material and tail (Xu et al., 2011), it was predicted that the AV material would not protect against vibration-induced changes, or might actually enhance the adverse effects of vibration on peripheral blood vessels and nerves.

In the first group of exposed rats, neither vibration nor restraint affected vascular responses to increasing pressure within the arteries. All arteries displayed a similar pressure-dependent rise in internal diameter. Although increasing pressure did not affect changes in vascular function, the application of the α2C-adrenoreceptor agonist, UK14304, resulted in a dose-dependent constriction at the lowest dose ($10^{-8.5}$ *M*) in all groups. However, arteries from rats vibrated on the bare platform displayed the greatest constriction in response to the application of UK14304 at this dose. These results were different from those of previous studies where acute vibration did not induce significant reductions in vascular diameter until a concentration of approximately $10^{-7.5}$ to 10^{7} *M* (Krajnak et al., 2006, 2009, 2014; Xu et al., 2010). In the second group of animals, vasoconstriction was examined at a broader dose range (10^{-10}–10^{-5} *M*). In this second experiment, there were no significant differences between the groups in UK14304-induced vasoconstriction, and a significant reduction in internal diameter was not seen until a dose of $10^{-7.0}$ *M* was applied. Data suggest that the increased sensitivity of arteries to UK14304-induced vasoconstriction may have been the result of testing the effects of increasing pressure on arteries prior to the application of UK14304. Data also indicate that arteries collected from rats exposed to vibration on the bare platform were more responsive to the application of pressure than arteries from rats in the other three groups. Previous studies demonstrated that an acute exposure to vibration results in pinched endothelial cells and increased oxidative activity in arteries (Krajnak et al., 2006; Curry et al., 2005). Vascular stress (e.g., cold, pressure, chemical insult) also may result in a translocation of α2C-adrenoreceptors from the cytoplasm cell membrane to the surface of the vascular smooth cell (Jeyaraj et al., 2012), thus making it more readily available to be acted upon by adrenoreceptor agonists.

The constriction induced by UK14304 at the higher concentrations was greater in arteries from rats exposed to vibration, or in arteries obtained from rats in the bare platform control group, than in glove controls in the first group of rats. Data suggest that gloves affected vascular responsiveness to UK14304 after vibration exposure. However, arteries from rats exposed to the bare platform were also more sensitive to UK14304. Thus, at these higher

concentrations, it seems likely that AV glove material may also have prevented heat transfer from the tail to the platform, thereby reducing sensitivity of the glove control arteries to UK14304-mediated vasoconstriction at higher doses. Similar changes in UK14304-mediated vasoconstriction at the higher doses were not seen in the second group of rats. As mentioned earlier, this was most likely due to the fact that these arteries were not exposed to increasing pressure prior to the application of the agonist.

In arteries from the first group of animals, rats exposed to vibration on the bare platform tended to be less sensitive to ACh-induced redilation, but there were no significant differences in the dose-dependent responses between groups. In Experiment 2, none of the groups displayed significant changes in the dose-dependent responses to ACh. These results are different from those of previous studies, where ventral tail arteries from rats exposed to vibration on a bare platform were less sensitive to ACh-induced redilation 24 h after exposure to a single bout of vibration (Krajnak et al., 2009, 2014). In these previous studies, changes in vascular responsiveness to ACh were accompanied by increases in oxidative activity and reductions in NO_x concentrations. These changes in the generation of reactive oxygen species (ROS) are in part responsible for the reduction in sensitivity observed in response to ACh (Hughes et al., 2009). In the current study, there were no marked changes in vascular NO_x concentrations, and H_2O_2 was not detectable in the arteries. Thus, although it is unclear why alterations were not observed in vibration-induced changes in vascular measures of oxidative stress, the fact that changes were not seen in oxidative stress is consistent with the finding that changes did not occur in sensitivity to ACh-mediated redilation.

Although the AV glove material did prevent some of the vascular changes in arteries, this material did not attenuate the effects of vibration on sensorineural responses. After exposure to a single bout of vibration, there was a decreased sensitivity to pressure in rats whose tails were vibrated on either the bare or the AV material-covered platform (i.e., the pressure needed to induce a withdrawal response was increased). Sensory information about pressure, like vibration, is detected by Pacinian corpuscles, Merkel disks, and Meissner corpuscles in the skin, and this information is then transmitted to the central nervous system through Aβ and Aδ fibers (Brammer et al., 1987; Bensmaia et al., 2005; Whitehouse and Griffin, 2002). The reduced sensitivity to pressure seen in this study is consistent with previous data showing that there are transient reductions in Aβ-fiber sensitivity after a single exposure to vibration at 125 Hz (Krajnak et al., 2007). These data are also consistent with human studies in which an acute exposure to vibration resulted in a temporary shift in the vibrotactile threshold at 125 Hz (Maeda, 1994).

In humans, an acute exposure to vibration was shown to result in a decrease in blood flow to the fingers and reduced sensitivity of Aβ-nerve fibers (large myelinated fibers carrying pressure and vibration information from the periphery to the central nervous system [CNS]) (Bovenzi et al., 1997, 2004; Giannini and Rossi, 2000). Anatomical and physiological changes in the ventral tail artery and nerve and in skin that are consistent with the physiological changes reported in humans have been reported (Krajnak et al., 2007, 2010, 2012). The acute effects of vibration on vascular and sensorineural function are exaggerated when the exposure occurs at or near the resonant frequency of the appendage being exposed

to vibration (Dong et al., 2012; Welcome et al., 2008; Krajnak et al., 2010, 2012). Based on the findings of this study, gloves may not prevent the adverse effects of resonant-frequency vibration on peripheral sensorineural function.

These findings are consistent with other studies demonstrating that antivibration materials may not protect against the adverse health effects of vibration on peripheral vascular and nervous system in workers using hand tools whose primary frequency is in the resonant frequency range of the finger–hand system (McDowell et al., 2013; Welcome et al., 2014). Workers using antivibration gloves will have reduced finger dexterity and also will tend to grip the tool more tightly. Thus, these workers may experience fatigue of the hand–arm system (Xu et al., 2011). However, gloves may be beneficial by providing protection against the cold, cuts, and abrasions. In addition, other new antivibration glove materials that are more comfortable and allow workers to have increased tactile feedback may protect workers from the negative effects of vibration but allow them to have a better feel for the tool. In addition, other interventions, such as using alternative production techniques, low-vibration machinery, and routine preventative maintenance regimes and limiting the amount of time a worker is exposed to vibration and reducing the vibration transmitted by the tool also may provide protection.

REFERENCES

Bensmaia SJ, Leung YY, Hsiao SS, Johnson KO. Vibratory adaptation of cutaneous mechanoreceptive afferents. J. Neurophysiol. 2005; 94:3023–3036. [PubMed: 16014802]

Bovenzi M, Apostoli P, Alessandro G, Vanoni O. Changes over a work-shift in aesthesiometric and vibrotactile perception thresholds of workers exposed to intermittent hand transmitted vibration from impact wrenches. Occup. Environ. Med. 1997; 54:577–587. [PubMed: 9326161]

Bovenzi M, Giannini F, Rossi S. Vibration-induced multifocal neuropathy in forestry workers: Electrophysiological findings in relation to vibration exposure and finger circulation. Int. Arch. Occup. Environ. Health. 2000; 73:519–527. [PubMed: 11100946]

Bovenzi M, Pinto I, Picciolo F, Mauro M, Ronchese F. Frequency weightings of hand-transmitted vibration for predicting vibration-induced white finger. Scand. J. Work Environ. Health. 2011; 37:244–252. [PubMed: 21046061]

Bovenzi M, Welsh AJ, Griffin MJ. Acute effects of continuous and intermittent vibration on finger circulation. Int. Arch. Occup. Environ. Health. 2004; 77:255–263. [PubMed: 15034718]

Brammer AJ, Piercy JE, Auger PL, Nohara S. Tactile perception in hands occupationally exposed to vibration. J. Hand Surg. Am. 1987; 12:870–875. [PubMed: 2821102]

Brown AP. The effects of anti-vibration gloves on vibration-induced disorders: A case study. J. Hand Ther. 1990; 3:94–100.

Curry BD, Govindaraju SR, Bain JL, Zhang LL, Yan JG, Matloub HS, Riley DA. Evidence for frequency-dependent arterial damage in vibrated rat tails. Anat. Rec. A Discov. Mol. Cell. Evol. Biol. 2005; 284:511–521. [PubMed: 15791580]

Dale AM, Rohn AE, Burwell A, Shannon W, Standeven J, Patton A, Evanoff B. Evaluation of anti-vibration interventions for the hand during sheet metal assembly work. Work. 2011; 39:169–176. [PubMed: 21673444]

Dong RG, Schopper AW, McDowell TW, Welcome DE, Wu JZ, Smutz WP, Warren C, Rakhej S. Vibration energy absorption (VEA) in human fingers–hand–arm system. Med. Eng. Phys. 2004; 26:483–492. [PubMed: 15234684]

Dong RG, Welcome DE, McDowell TW, Xu XS, Krajnak K, Wu JZ. A proposed theory on biodynamic frequency weighting for hand-transmitted vibration exposure. Ind. Health. 2012; 50:412–424. [PubMed: 23060254]

Griffin, MJ. Handbook of human vibration. Academic Press; San Diego, CA: 1990.

Hewitt S, Dong RG, Welcome DE, McDowell TW. Anti-vibration gloves? Ann Occup. Hyg. 2015; 59:127–141. [PubMed: 25381184]

Hughes JM, Wirth O, Krajnak K, Miller R, Flavahan S, Berkowitz DE, Welcome D, Flavahan NA. Increased oxidant activity mediates vascular dysfunction in vibration injury. J. Pharmacol. Exp. Ther. 2009; 328:223–230. [PubMed: 18955588]

Jetzer T, Haydon P, Reynolds D. Effective intervention with ergonomics, antivibration gloves, and medical surveillance to minimize hand-arm vibration hazards in the workplace. J. Occup. Environ. Med. 2003; 45:1312–1317. [PubMed: 14665818]

Jeyaraj SC, Unger NT, Eid AH, Mitra SP, El-Dahdah N, Quilliam LA, Flavahan NA, Chotani MA. Cyclic AMP-Rap1A signaling activates RhoA to induce a2C adrenoreceptor translocation to the cell surface of microvascular smooth muscles. Am. J. Physiol. Cell Physiol. 2012; 303:C499–C511. [PubMed: 22621783]

Krajnak K, Dong RG, Flavahan S, Welcome DE, Flavahan NA. Acute vibration increases α_{2c}-adrenergic smooth muscle constriction and alters thermosensitivity of cutaneous arteries. J. Appl. Physiol. 2006; 100:1230–1237. [PubMed: 16339346]

Krajnak K, Miller GR, Waugh S, Johnson C, Li S, Kashon ML. Characterization of frequency-dependent response of the vascular system to reptitive vibration. J. Occup. Environ. Med. 2010; 52:584–594. [PubMed: 20523237]

Krajnak K, Waugh S, Wirth O, Kashon L. Acute vibration reduces Aβ nerve fiber sensitivity and alters gene expression in the ventral tail nerves of rats. Muscle Nerve. 2007; 36:197–205. [PubMed: 17541999]

Krajnak K, Dong RG, Flavahan S, Welcome DE, Flavahan NA. Acute vibration increases alpha2C-adrenergic smooth muscle constriction and alters thermosensitivity of cutaneous arteries. J. Appl. Physiol. 2006; 100:1230–1237. [PubMed: 16339346]

Krajnak K, Miller GR, Johnson C, Waugh S, Kashon ML. Frequency-dependent effects of vibration on peripheral nerves and sensory nerve function in a rat model of hand–arm vibration syndrome. J. Occup. Environ. Med. 2012; 54:1010–1016. [PubMed: 22785326]

Krajnak K, Rosewell KL, Wise PM. Fos-induction in gonadotropin-releasing hormone neurons receiving vasoactive intestinal polypeptide innervation is reduced in middle-aged female rats. Biol. Reprod. 2001; 64:1160–1164. [PubMed: 11259263]

Krajnak K, Waugh S, Johnson C, Miller R, Kiedrowski M. Vibration disrupts vascular function in a model of metabolic syndrome. Ind. Health. 2009; 47:533–542. [PubMed: 19834263]

Krajnak K, Waugh S, Miller GR, Johnson C. Recovery of vascular function after exposure to a single bout of segmental vibration. J. Toxicol. Environ. Health A. 2014; 77:1061–1069. [PubMed: 25072825]

Krajnak K, Waugh S, Wirth O, Kashon ML. Acute vibration reduced Aβ nerve fiber sensitivity and alters gene expression in the ventral tail nerves of rats. Muscle Nerve. 2007; 36:197–205. [PubMed: 17541999]

Laszlo HE, Griffin MJ. The transmission of vibration through gloves: Effects of push force, vibration magnitude and intersubject variability. Ergonomics. 2011; 54:488–496. [PubMed: 21547793]

Maeda S. Temporary threshold shifts in fingertip vibratory sensation from hand-transmitted vibration and repetitive shock. Nagoya J. Med. Sci. 1994; 57(suppl.):185–193. [PubMed: 7708100]

Maeda, S.; Griffin, M. Consideration of the frequency weighting in ISO 5349 and BS 6842 with respect to temporary changes in thermotactile thresholds, vibrotactile thresholds and circulatory function after acute exposure to hand-transmitted vibration. Finnish Institute of Occupational Health; Helsinki: 1997.

Maeda S, Yonekawa Y, Kanada K, Takahashi Y, Griffin MJ. Vibrotactile temporary threshold shifts induced by hand-transmitted vibration during underwater work. Ind. Health. 1995; 33:89–99. [PubMed: 8543478]

McDowell TW, Dong RG, Welcome DE, Xu XS, Warren C. Vibration-reducing gloves: Transmissibility at the palm of the hand in three orthogonal directions. Ergonomics. 2013; 56:1823–1840. [PubMed: 24160755]

Milosevic M, McConville KM. Evaluation of protective gloves and working techniques for reducing hand-arm vibration exposure in the workplace. J. Occup. Health. 2012; 54:250–253. [PubMed: 22790528]

Welcome DE, Krajnak K, Kashon ML, Dong RG. An investigation on the biodyamic foundation of a rat tail model. J. Eng. Med. (Proc. Inst. Mech. Eng. Part H). 2008; 222:1127–1141.

Welcome DE, Dong RG, Xu XS, Warren C, McDowell TW. The effects of vibration-reducing gloves on finger vibration. Ind. Ergonom. 2014; 44:45–59.

Whitehouse DJ, Griffin MJ. A comparison of vibrotactile thresholds obtained using different diagnostic equipment: the effect of contact conditions. Int. Arch. Occup. Environ. Health. 2002; 75:85–89. [PubMed: 11898881]

Wu JZ, Krajnak K, Welcome DE, Dong RG. Analysis of the dynamic strains in a fingertip exposed to vibrations: Correlation to the mechanical stimuli on mechanoreceptors. J. Biomech. 2006; 39:2445–2456. [PubMed: 16168999]

Wu JZ, Welcome DE, Krajnak K, Dong RG. Finite element analysis of the penetrations of shear and normal vibrations into the soft tissues in a fingertip. Med. Eng. Phys. 2007; 29:718–727. [PubMed: 16962362]

Xu XS, Riley DA, Persson M, Welcome DE, Krajnak K, Wu JZ, Raju RJ, Dong RG. Evaluation of anti-vibration effectiveness of glove materials using an animal model. Biomed. Mater. Eng. 2011; 21:193–211. [PubMed: 22182788]

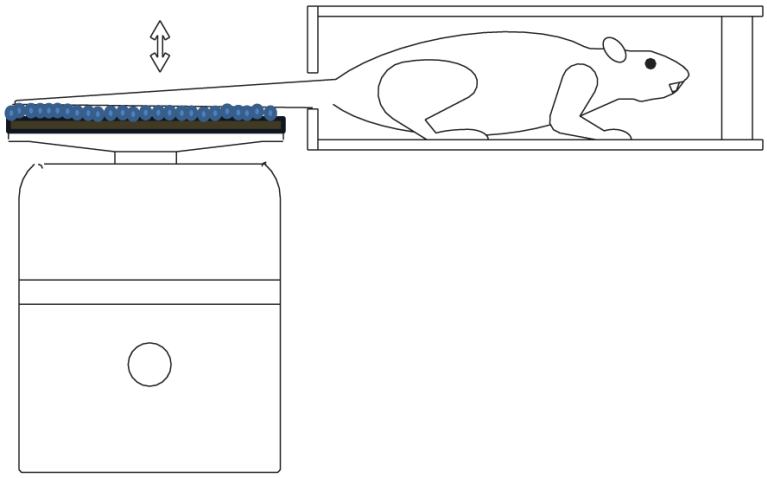

FIGURE 1.

This diagram illustrates how animals were restrained and their tails positioned on the platform. The AV-glove material, including the leather and air pocket antivibration material was secured to the platform using double stick adhesive. The tail was secured either to the bare or covered platform using 4 strips of Soft tape as described in the methods section.

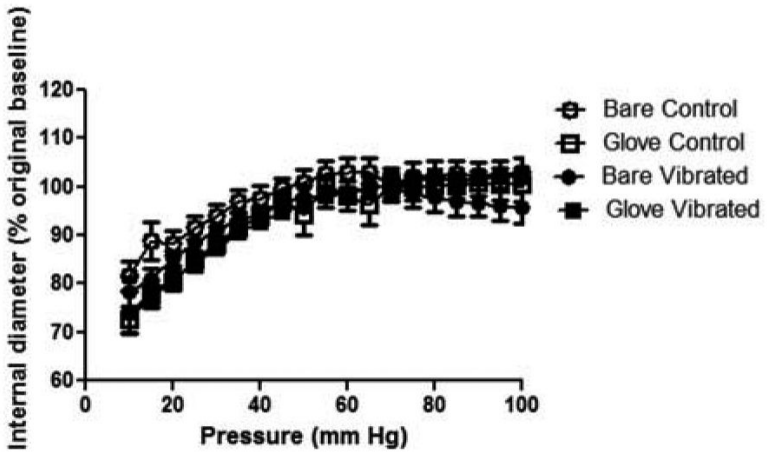

FIGURE 2.

The effects of increasing pressure on the internal vascular diameter. Increasing pressure from 10 to 110 mm Hg resulted in an increase in the internal diameter of all arteries. However, there were no significant pressure-dependent changes between the groups.

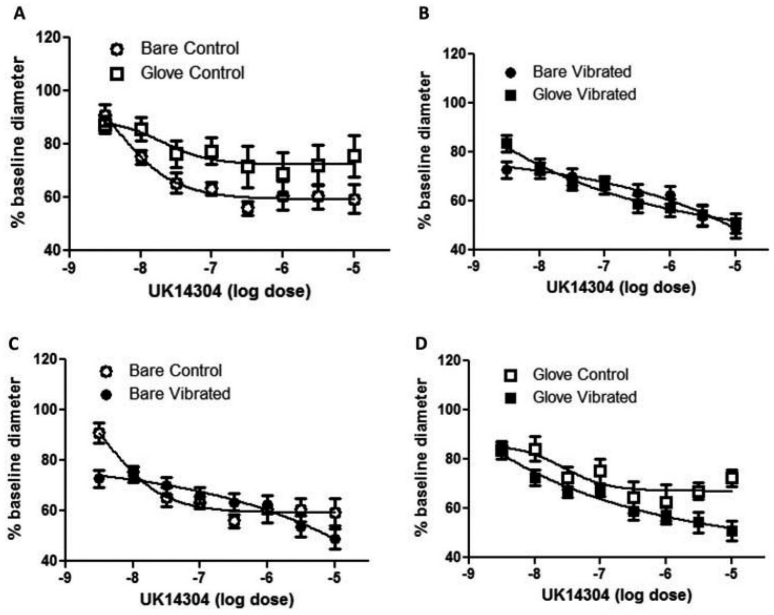

FIGURE 3.

Dose-dependent α2C-mediated vasoconstriction of the ventral tail artery. The dose-dependent response to UK14304 was assessed in arteries after assessing the effects of pressure. The data from the four groups are displayed in four different graphs so that relevant comparisons can be more easily seen: (A) bare control vs, glove control, (B) bare vibrated vs. glove vibrated; (C) bare control vs. bare vibrated; and (D) glove control vs. glove vibrated. All arteries appeared to be more sensitive to the constricting effects of UK14304. Arteries from rats exposed on the bare platform were more responsive at the lowest doses than rats from the other conditions (B and C). At the higher doses, rats from the glove control condition were less responsive to the effects of UK14304 than rats in the other three conditions (A and D). Asterisk indicates significant at $p < .05$ (*n*/group are the same as described in Figure 2).

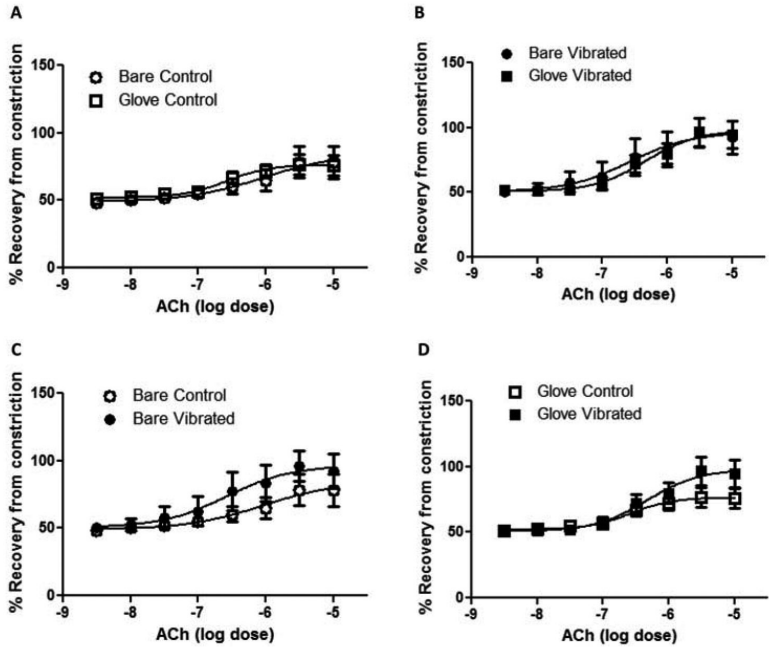

FIGURE 4.

Dose-dependent ACh-mediated vasodilation in the ventral tail artery after preconstruction with phenylephrine. The data from the four groups are displayed in four different graphs so that relevant comparisons can be more easily seen: (A) bare control vs. glove control; (B) bare vibrated vs. glove vibrated; (C) bare control vs. bare vibrated; and (D) glove control vs. glove vibrated. The dose-dependent redilation of arteries to ACh was not affected by treatment (*n*/group are the same as described in Figure 2).

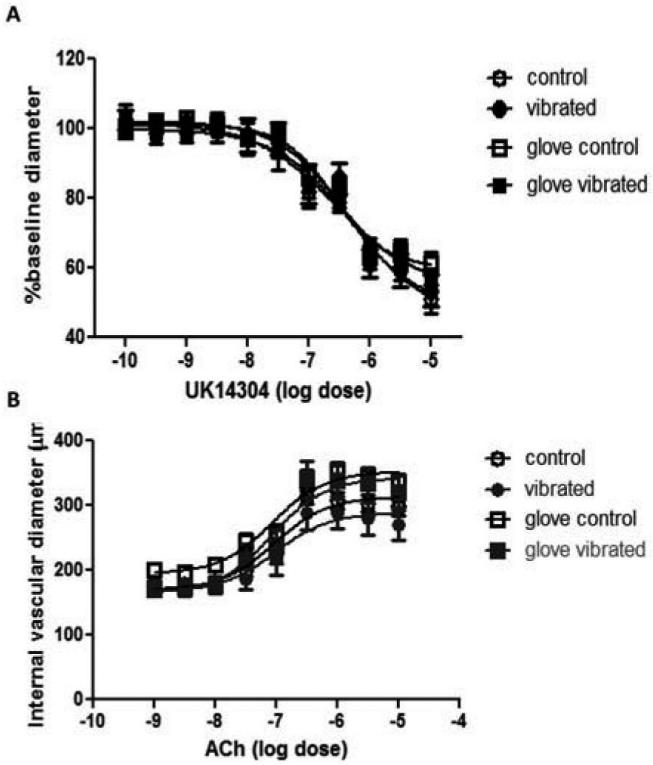

FIGURE 5.

Dose-dependent UK14304-induced vasoconstriction (A) and ACh-mediated redilation (B) of arteries were similar under all conditions.

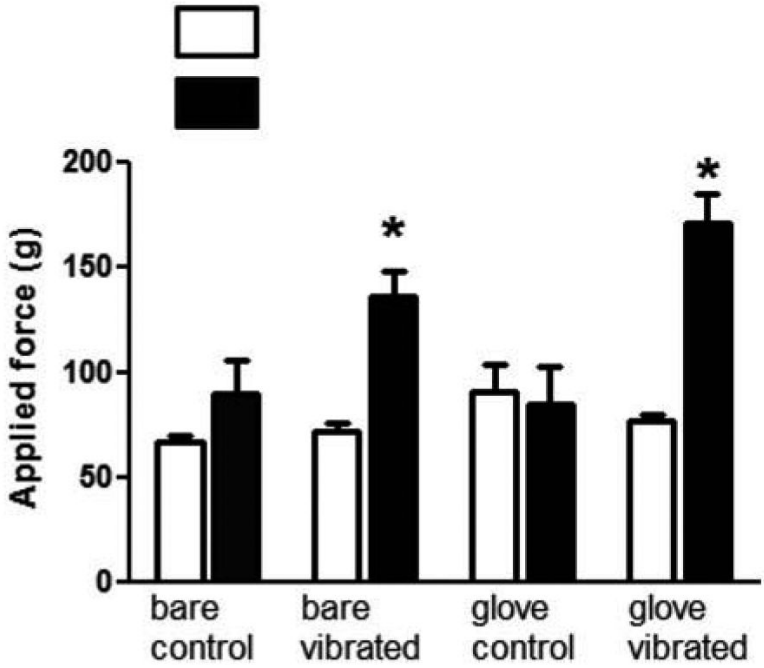

FIGURE 6.

The pressure inducing a withdrawal of the tail was greater postexposure than preexposure in both vibration groups. Asterisk indicates significant at $p < .05$ ($n = 4$/group).