Aerobic Training Increases Pain Tolerance in Healthy Individuals

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ABSTRACT

JONES, M. D., J. BOOTH, J. L. TAYLOR, and B. K. BARRY. Aerobic Training Increases Pain Tolerance in Healthy Individuals. *Med. Sci. Sports Exerc.*, Vol. 46, No. 8, pp. 1640–1647, 2014. The hypoalgesic effects of acute exercise are well documented. However, the effect of chronic exercise training on pain sensitivity is largely unknown. **Purpose**: To examine the effect of aerobic exercise training on pain sensitivity in healthy individuals. **Methods**: Pressure pain threshold, ischemic pain tolerance and pain ratings during ischemia were assessed in 24 participants before and after 6 wk of structured aerobic exercise training (n = 12) or after 6 wk of usual physical activity (n = 12). The exercise training regimen consisted of cycling three times per week for 30 min at 75% of maximal oxygen consumption reserve. **Results**: Significant increases in aerobic fitness (P = 0.004) and ischemic pain tolerance (P = 0.036) were seen in the exercise group after training, whereas pressure pain threshold and pain ratings during ischemia were unchanged (P > 0.2). No change in aerobic fitness (P > 0.1) or pain sensitivity (P > 0.1) was observed in the control group. **Conclusion**: Moderate- to vigorous-intensity aerobic exercise training increases ischemic pain tolerance in healthy individuals. **Key Words:** PAIN THRESHOLD, PAIN RATING, HYPOALGESIA, ISCHEMIC PAIN, CHRONIC EXERCISE

In healthy individuals, the hypoalgesic effect of acute exercise is well documented (25). Chronic exercise training is also demonstrated to reduce pain sensitivity in patients with persistent pain (16,40), and accordingly, exercise training has become an important part of treatment in these patients (8,15). However, despite the growing evidence for a pain-relieving effect of exercise training in chronic disease populations, the effect of aerobic training on pain sensitivity in healthy individuals is largely unknown. Hence, little is known of how exercise training may modulate pain independently of disease.

To our knowledge, only one study has examined the effect of chronic aerobic exercise on pain sensitivity in healthy participants (5). Anshel and Russell (5) examined the effect of 12 wk of aerobic or resistance or combined aerobic and resistance exercise on pressure pain tolerance in 48 unfit males. The group who completed aerobic exercise training (cycling) tolerated a greater magnitude of mechanical pressure applied to the upper limb. A similar pattern of change was apparent for the lower limb, but the results did not reach significance. Furthermore, the aerobic exercise group, despite tolerating higher intensities of mechanical pressure, reported a more severe subjective appraisal of pain after that training period. Resistance training had no influence on pain tolerance (5). Although the study provides preliminary, but weak, evidence that aerobic exercise training may lead to increased pain tolerance, it has several limitations. The effect of exercise training on pain threshold, and on the duration that the painful stimuli could be tolerated, were not quantified. The volume and intensity of exercise performed by participants was highly varied across the 12-wk period, making it difficult to identify the volume and intensity of exercise needed to elicit the hypoalgesic response. Lastly, maximal aerobic capacity was not measured, so the influence of exercise training on $\dot{V}O_{2max}$, pain sensitivity, and endurance performance cannot be determined.

Numerous other studies have sought to find a relation between fitness or sporting achievement and pain sensitivity based on the anecdotal observation that athletes are more stoical. Findings from these cross-sectional studies are equivocal and have varied depending on the sport and even the phase of the competitive season or the standard of competition from which athletes have been studied (31,33,34), as well as with the coping strategies used by different individuals (23,28). Frequently, the volume, the intensity, the duration, and the type of exercise training performed by the athletes have been poorly controlled or quantified. Furthermore, findings have depended heavily on the modality and protocol for evoking pain, in particular whether pain thresholds or pain tolerance have been measured (31). In general, these cross-sectional investigations provide evidence that chronic exercise

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can increase pain tolerance, but not pain threshold (28,33,36). This notion is supported by Anshel and Russell (5), who demonstrated an increase in pain tolerance after exercise training. To our knowledge, this is the only study examining the effect of exercise training on pain sensitivity in healthy, nonathlete individuals. Therefore, it is still largely unclear whether chronic exercise can influence pain sensitivity independently of athletic status.

The present study was designed to examine the effect of moderate- to vigorous-intensity chronic aerobic exercise on pain sensitivity in healthy adults. Pressure and ischemic noxious stimuli were chosen as they are arguably the most similar to the pain experienced during physical activity and chronic disease (33). It was hypothesized that, based on previous studies, chronic aerobic exercise would increase pain tolerance, but not affect pain threshold.

METHODS

Participants. Participants were recruited using advertisements placed around billboards on campus. Eligibility criteria included 1) apparently healthy with no history of chronic pain or chronic disease, 2) between the ages of 18 and 50 yr, and 3) absence of a current diagnosis of depression. Twenty-seven participants (5 males and 22 females) were recruited for this study. Throughout the 6-wk intervention, three participants withdrew because of injury unrelated to the study, leaving a total of 24 participants who completed the study (exercise: 1 male and 11 females, 24.4 ± 4.3 yr; control: 2 males and 10 females, 21.8 ± 1.6 yr; $P = 0.013$).

Procedures. This study was approved by the University of New South Wales Human Research Ethics Committee. Written informed consent was obtained from each participant before testing. Participants were recruited from the same university staff and student population and on the basis of volunteering for either the exercise or the control group. That is, participants were not randomly assigned but were allocated to either the exercise or control group based on their willingness to participate in either group. For the exercise group, the experiment consisted of 20 sessions, including an initial assessment, 18 exercise sessions, and a final assessment. Control subjects performed only the initial and final assessment and were asked to maintain their regular level of physical activity during the 6-wk period. Before the initial and final assessments, participants were asked to abstain from vigorous exercise for 24 h and from caffeine for 4 h. Compliance to these requests was confirmed verbally at the start of the session.

During their first and last visits and before assessments of pain sensitivity and aerobic capacity, participants completed several questionnaires to assess their psychological status and their physical activity levels. The Distress Risk and Assessment Method questionnaire, which is composed of the Zung Depression Index and the Modified Somatic Perceptions Questionnaire, was used to evaluate distress, depression, and somatization (22). The Profile of Mood States was used to assess six subscales of mood (tension, depression, confusion, anger, vigor, and fatigue) (29). The long form of the International Physical Activity Questionnaire was used to evaluate physical activity levels (9). Height, body mass, and arm and thigh circumferences and skinfolds were recorded. Pressure pain threshold, ischemic pain tolerance, pain ratings during ischemia, and aerobic capacity ($\dot{V}O_{2peak}$) were then assessed as described in the following paragraphs.

Pressure pain threshold was assessed for four muscular sites (trapezius, biceps brachii, rectus femoris, and tibialis anterior). All measurements were made on the right side of the body and in the following rotational order: trapezius, biceps brachii, rectus femoris, and tibialis anterior. Three practice trials were performed on the left trapezius muscle before testing to familiarize the participant with the procedure. The rubber-tipped probe of the handheld algometer (Wagner Force 10 FDX-25; Wagner Instruments, Greenwich, CT) was applied perpendicularly to the participant's skin, and the force was increased gradually at a rate of approximately 1 $kg \cdot s^{-1}$. Participants were instructed to give a verbal command of "stop" when the sensation of pressure turned to pain. This procedure was repeated two more times, for a total of three measurements per site. Pressure pain threshold was recorded as the average of these three measurements. A pilot study examining the reliability of this measure showed high within- and between-session intrarater reliability across all four testing sites (ICC > 0.9).

Ischemic pain tolerance was assessed via a modified submaximal ischemic tourniquet test. Participants grasped, with their dominant hand, a custom-built grip force device that was instrumented with a force transducer (Transducer Techniques MLP-200). Force was sampled at 200 Hz with a 12-bit analog-to-digital device (USB-6008; National Instruments, Austin, TX) and stored in conjunction with the ratings of pain and the target grip force profile. Custom software was written (Labview version 9.0; National Instruments) to provide visual feedback of the grip force and auditory tones prompted the start and end of each contraction. After the determination of the participant's maximal voluntary force, the sleeve of a standard sphygmomanometer was placed around the participant's upper arm, which was then exsanguinated by raising it above the level of the heart for 60 s. The cuff was inflated to 200 mm Hg before the arm was returned to horizontal. Prompted by the auditory tones and monitored by visual feedback, gripping exercise was performed at 30% maximal force for as long as tolerable (4-s contraction and 4-s rest). Pain tolerance was the total time participants were able to sustain the handgrip exercise under ischemic conditions. During testing, subjective ratings of pain were recorded using a 0–10 numeric pain rating scale every 30 s (38). Participants were instructed to choose the number on the scale that corresponded to their level of pain, with 0 = "no pain" and 10 = "worst possible pain." The experimenter was prepared to terminate the procedure if the limit of pain tolerance was not reached by 10 min. This time limit was not made known to participants, who were

instructed only to continue the handgrip exercise for as long as tolerable. The results of a pilot study showed high reliability for the pain tolerance measurement (ICC = 0.94).

A $\dot{V}O_{2peak}$ test was performed on a Monark 828e cycle ergometer (Vansbro, Sweden) with use of an Ultima CPX gas analysis system (Medgraphics, Minnesota USA). Participants were instructed to maintain a pedaling speed of 70 rpm throughout the test. Exercise began with a 5-min warm-up at 35 W, after which the workload increased at a rate of 35 W every 2 min until 105 W was reached. After this, workload increased at a rate of 35 W every 1 min until $\dot{V}O_{2peak}$ was obtained. Criteria used for the determination of $\dot{V}O_{2peak}$ were as follows: no further increase in oxygen consumption despite an increase in workload, HR within ±5 bpm of the participants age-predicted maximum HR, a respiratory exchange ratio > 1.15, and volitional fatigue.

The exercise training consisted of cycle ergometer exercise performed three times per week for 30 min at 75% of HR reserve. The age-predicted maximum HR was determined using the prediction equation $HR_{max} = 207 - 0.7 \times$ age (12). This workload was chosen to correspond to an intensity of 75% $\dot{V}O_{2reserve}$, considered moderate–vigorous intensity (27,35). Each session began with a 5-min warm-up at 35 W and concluded with a 5-min cool down at 35 W. After the warm-up, workload was adjusted to correspond to the intensity that elicited an HR equivalent to 75% HR reserve. Participants were then required to maintain this intensity for 30 min. Measurements of workload, HR, and RPE were recorded every 5 min throughout the exercise sessions. During the exercise intervention, workload was adjusted as necessary to ensure participants maintained their target HR. Participants were required to complete a minimum of 17 exercise sessions to be included in the study. A minimum of 2 d separated the final exercise session and the final assessment.

Data processing and analysis. Pressure pain thresholds for the trapezius and biceps brachii sites and for the rectus femoris and tibialis anterior sites were combined to give an average value for the upper and lower body, respectively. Pain tolerance was the total time that participants were able to sustain the handgrip exercise under ischemic conditions. Pain ratings during ischemia were analyzed in two ways: 1) the slope of the regression line was used to provide the rate of increase in pain rating (i.e., pain ratings per second), and 2) the peak pain rating value was also used (peak pain rating). Linear regression analysis revealed that increases in pain rating were sufficiently linear so that the slope of the regression line was suitable to quantify the increase in pain rating (mean r^2 for each group, exercise: r^2 = 0.89, SD = 0.13; control: r^2 = 0.91, SD = 0.08). Data were analyzed using the Statistical Package for the Social Sciences (version 20; SPSS Inc., Chicago, IL). A repeated-measures ANOVA was used to examine differences between groups and across time. Bonferroni-adjusted paired-sample *t*-tests and independent-sample *t*-tests were also used *post hoc* to examine any within and between group differences, respectively. Significance was set at the $\alpha = 0.05$ level.

RESULTS

Maximal aerobic capacity ($\dot{V}O_{2peak}$). The $\dot{V}O_{2peak}$ of participants is outlined in Table 1. A significant group–time interaction was observed for $\dot{V}O_{2peak}$ ($F_{1,22} = 24.00$, $P < 0.001$). A significant difference in $\dot{V}O_{2peak}$ between groups was observed at baseline ($t_{22} = 3.11$, $P = 0.02$), but this difference disappeared after the intervention ($t_{22} = 0.24$, $P = 0.81$). Exercise training caused a significant increase in $\dot{V}O_{2peak}$ ($t_{11} = -5.39$, $P = 0.004$, + 14.6%), whereas $\dot{V}O_{2peak}$ was not significantly different at follow-up in the control group ($t_{11} = 1.45$, $P = 0.72$, −2.8%).

Workload, HR, and RPE during $\dot{V}O_{2peak}$ assessment. A significant group–time interaction was observed for workload ($F_{1,22} = 11.5$, $P = 0.003$) but not peak HR ($F_{1,22} = 3.63$, $P = 0.07$) or RPE ($F_{1,22} = 0.77$, $P = 0.39$) during $\dot{V}O_{2peak}$ assessment. For participants in the exercise group, an increase in peak workload was observed in the final compared with the initial $\dot{V}O_{2peak}$ assessment ($t_{11} = -2.66$, $P = 0.08$, + 8.6%). Conversely, a decrease in peak workload was observed for participants in the control group in the final $\dot{V}O_{2peak}$ assessment compared with the initial $\dot{V}O_{2peak}$ assessment ($t_{11} = 2.16$, $P = 0.2$, −6.7%). Peak HR and RPE were unchanged in both groups between each $\dot{V}O_{2peak}$ assessment ($P > 0.5$; Table 1).

Exercise training. Six of the exercise participants completed all 18 exercise sessions, whereas the other six completed 17 of the 18 sessions. For the exercise group, there was a significant increase in the average exercise workload between the first and the last exercise session of the intervention ($t_{11} = -2.67$, $P = 0.02$, + 9.4%), whereas the average RPE during these sessions remained unchanged ($t_{11} = 1.46$, $P = 0.17$) (Table 2). The average RPE across all exercise sessions was 15, which equates to a subjective rating of "hard."

Ischemic pain tolerance. The duration of ischemic contractions, and pain ratings during ischemia, for participants in each group are shown in Figure 1. At follow-up, the

TABLE 1. Duration and peak workload, HR_{max}, RPE, and RER during the maximal aerobic test before and after the intervention.

	Before	After
Exercise		
$\dot{V}O_{2peak}$ (mL·min^{-1}·kg^{-1})	36.3 ± 5.5*,**	41.6 ± 6.4*
Duration (min)	6.4 ± 1.4***	7 ± 1.5***
Workload (W)	200.5 ± 41.7	217.7 ± 41.9
HR (bpm)	173.6 ± 11.3	176.1 ± 8.7
RPE	18.8 ± 1.2	18.6 ± 1.4
RER	1.43 ± 0.13	1.33 ± 0.8
Control		
$\dot{V}O_{2peak}$ (mL·min^{-1}·kg^{-1})	42.9 ± 4.9**	41.7 ± 6.1
Duration (min)	7.2 ± 1	7 ± 1.3
Workload (W)	217.9 ± 32.2	203.3 ± 37.3
HR (bpm)	180.9 ± 12.8	178.1 ± 12.2
RPE	18.6 ± 1.4	18.5 ± 1.2
RER	1.37 ± 0.8	1.3 ± 0.7

Data are presented as mean ± SD. The duration of the maximal aerobic test does not include the warm-up or cooldown.
*Significant increase across time within the exercise group, $P = 0.004$.
**Significant difference between groups at baseline, $P = 0.02$.
***Significant increase across time within the exercise group, $P = 0.04$.

TABLE 2. Workload, HR, and RPE during the first and last exercise training session for participants in the exercise group.

	First Session	Final Session
Workload (W)	96.8 ± 20.7*	105.9 ± 23.9*
HR (bpm)	160.7 ± 6.9	158.2 ± 7.8
RPE	15.2 ± 1.8	14.6 ± 1.3

Data are presented as mean ± SD.
*Significant difference between the first and last exercise session, $P = 0.02$.

duration of ischemic contractions was increased in 10 of 12 exercise participants and 5 of 12 control participants. There was a significant group–time effect observed for ischemic pain tolerance ($F_{1,22} = 8.4$, $P = 0.008$). For the exercise group, there was a significant increase in ischemic pain tolerance after training ($t_{11} = -3.15$, $P = 0.036$, +20.3%), whereas ischemic pain tolerance was unchanged in the control group across time ($t_{11} = 1.77$, $P = 0.44$, −3.7%) (Fig. 2A). Ischemic pain tolerance was not significantly different between groups at baseline ($t_{22} = -0.92$, $P = 1$). No relationship was observed between the change in duration of ischemic contractions and the change in $\dot{V}O_{2peak}$ for either group (Fig. 3). However, when both groups were combined, there was a significant positive relationship between the change in $\dot{V}O_{2peak}$ and the change in ischemic pain tolerance ($r^2 = 0.21$, $P = 0.02$; Fig. 3). Presumably, the significant correlation for the combined data occurred mainly because of the way the groups were clustered. That is, an

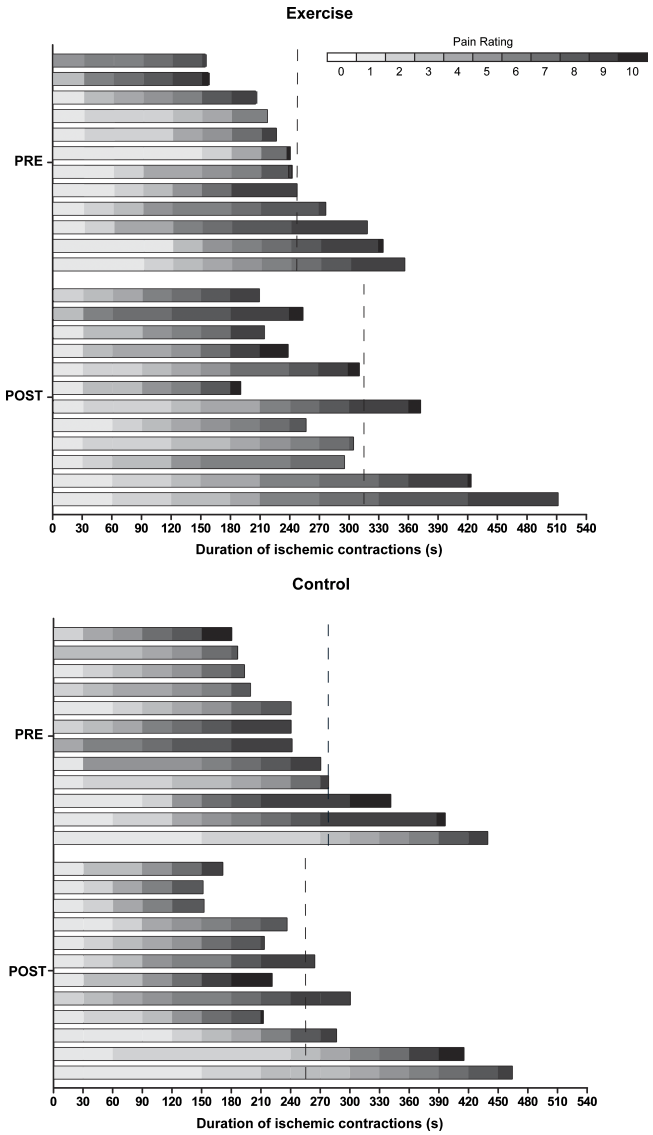

FIGURE 1—Pain tolerance (s) and pain ratings during ischemia for each participant in the exercise group before and after the intervention. The duration of ischemic handgrip exercise performed by each participant is represented by lengths of bars. The dashed lines represent the average duration of ischemic contractions for all participants. Ratings of pain throughout the exercise are represented by shading (scale at top right).

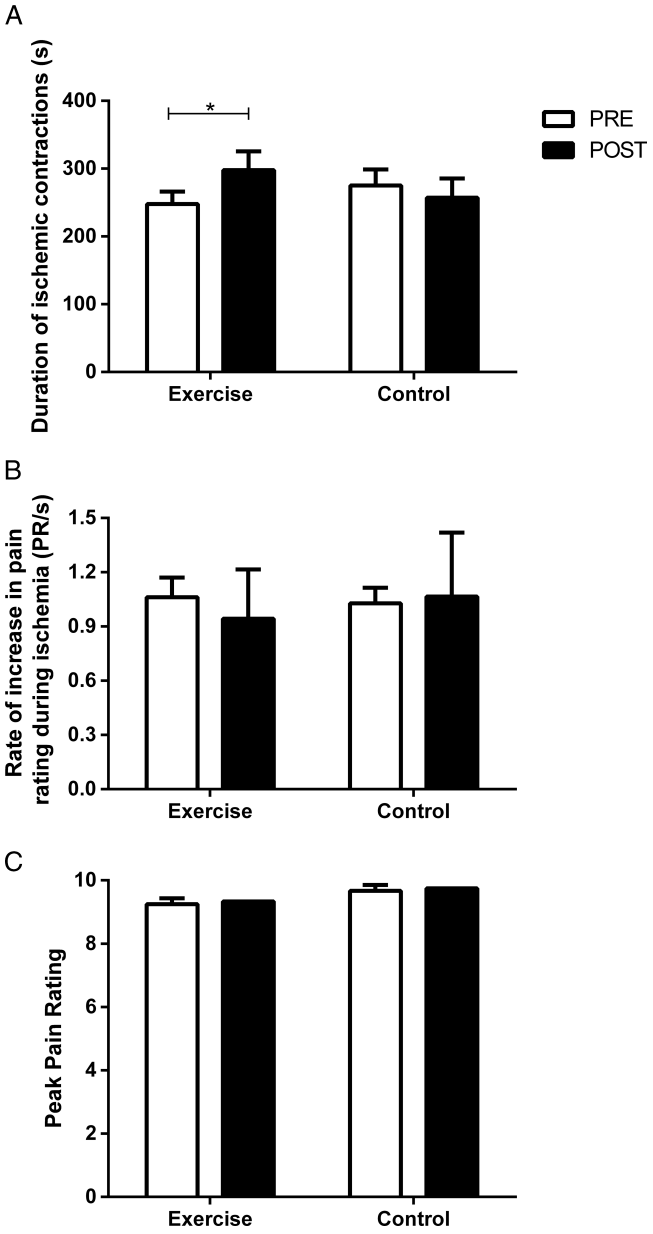

FIGURE 2—Group mean ± SEM data for pain tolerance (s) and pain ratings during ischemia for the exercise and control groups before and after the intervention. A. Duration of ischemic contractions (s). B. The rate of increase in pain rating during ischemia. C. Peak pain rating during ischemia. *Significant difference, $P < 0.05$.

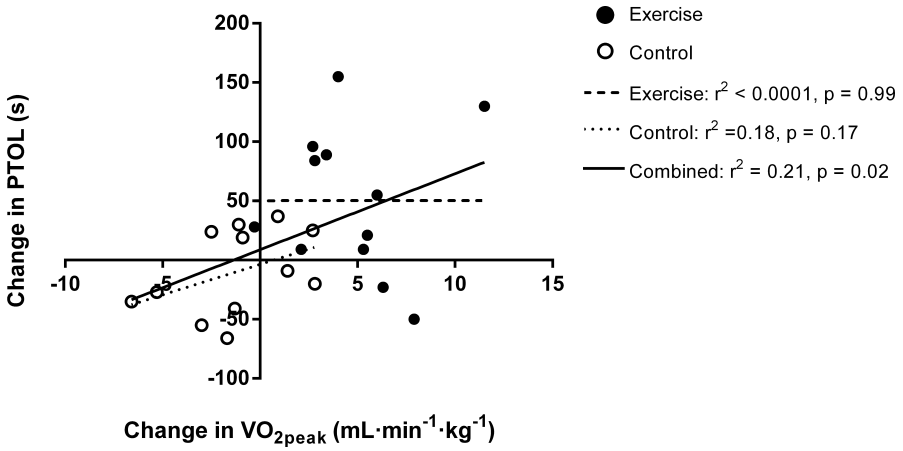

FIGURE 3—The relationship between the change in $\dot{V}O_{2peak}$ (mL·min^{-1}·kg^{-1}) and the change in pain tolerance (s) for the exercise and control groups and both groups combined. Correlation coefficients are reported separately for the exercise and control groups as well as both groups combined.

exercise group with significant changes in $\dot{V}O_{2peak}$ and pain tolerance and a control group with minimal change in these variables.

Pain ratings during ischemia. Pain ratings increased progressively for all participants during the ischemic pain tolerance task (Fig. 1). There was no group effect ($F_{1,22} = 0.14$, $P = 0.7$) or group–time interaction ($F_{1,22} = 1.67$, $P = 0.21$) for the rate of increase in pain during ischemia (Fig. 2B). There was no group effect ($F_{1,22} = 3.3$, $P = 0.08$) or group–time interaction ($F_{1,22} = 0$, $P = 1$) on peak pain rating during ischemia (Fig. 2C).

Pressure pain threshold. There was no significant group effect on pressure pain threshold for either the upper ($F_{1,22} = 0.6$, $P = 0.45$) or lower body ($F_{1,22} = 2.9$, $P = 0.1$). There was no significant group–time effect on pressure pain threshold for either the upper ($F_{1,22} = 1.9$, $P = 0.18$) or lower body ($F_{1,22} = 0.61$, $P = 0.44$; see Fig. 4).

Physical activity. Aside from the exercise group completing the aerobic training intervention, participants were instructed to maintain their usual levels of physical activity. Self-reported physical activity questionnaires identified that all participants adhered to this instruction. There was a significant difference between groups in self-reported walking before the intervention (exercise: 653.8 ± 431 MET·min·wk^{-1}; control: 2477.8 ± 2043.6 MET·min·wk^{-1}; $t_{11.98} = 3.02$; $P = 0.044$). Self-reported moderate physical activity was not different between the groups at baseline (exercise: 986.5 ± 1033 MET·min·wk^{-1}; control: 1718.7 ± 1529 MET·min·wk^{-1}; $t_{22} = 1.37$; $P = 0.73$), or after the intervention (exercise: 527 ± 541 MET·min·wk^{-1}; control: 1792.6 ± 1463 MET·min·wk^{-1}; $t_{13.96} = 2.81$; $P = 0.056$). Similarly, self-reported vigorous physical activity was not different between the groups at baseline (exercise: 939.5 ± 848 MET·min·wk^{-1}; control: 1708.2 ± 1642 MET·min·wk^{-1}; $t_{22} = 1.44$; $P = 0.66$) or after the intervention (exercise: 1398.3 ± 772 MET·min·wk^{-1}; control: 2028.2 ± 1705 MET·min·wk^{-1}; $t_{22} = 1.17$; $P = 1$). Vigorous physical activity level increased, although not significantly, in the final week of the intervention compared with baseline ($F_{1,22} = 6.59$, $P = 0.02$) in the exercise group only ($t_{11} = -2.5$, $P = 0.12$, + 48.5%). No relationship was observed between the change in vigorous physical activity level and the change in ischemic pain tolerance for either the exercise ($r^2 = 0.005$, $P = 0.82$) or control group ($r^2 = 0.19$, $P = 0.15$).

Mood. Regarding the mood of participants on the days of testing before and after the intervention, the Profile of Mood States (POMS) showed no significant difference between the groups for any mood state ($F_{1,22} = 3.4$, $P > 0.08$). A significant effect of time on vigor was observed ($F_{1,22} = 2.96$, $P = 0.04$). Vigor increased significantly in the control group at follow-up compared with baseline ($t_{11} = -3.4$, $P = 0.024$), but all other mood states remained unchanged across time in both

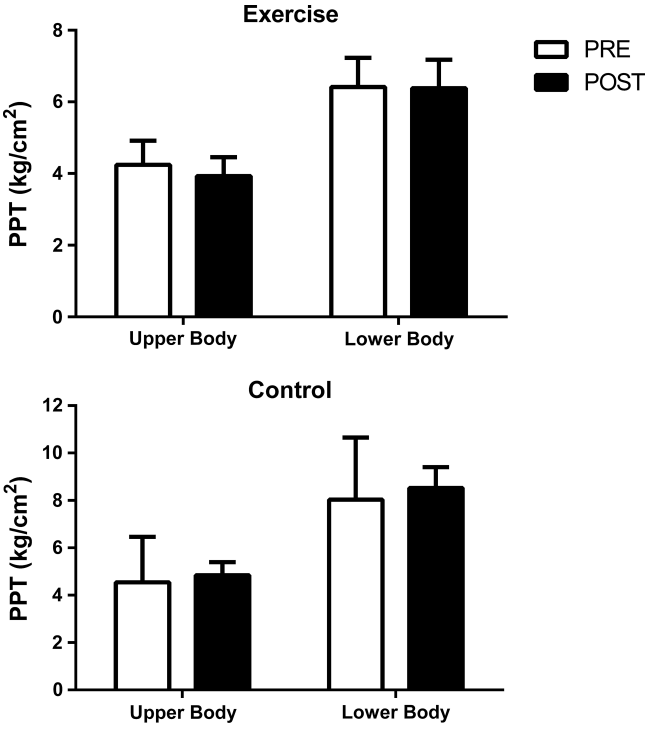

FIGURE 4—Mean ± SEM pressure pain threshold (PPT) for each group before and after the intervention.

groups ($F_{1,22}$ = 3.94, P > 0.24). A significant difference between groups was observed for the Modified Somatic Perceptions Questionnaire at baseline (PRE—exercise: 4.08 ± 2.64; control: 1.17 ± 1.75; $t_{19.08}$ = −3.19; P = 0.02) and after the intervention (POST—exercise: 2.83 ± 2.29; control: 0.83 ± 0.94; $t_{14.59}$ = 2.8; P = 0.04).

DISCUSSION

The results of the present study indicate that in healthy adults, 6 wk of moderate- to vigorous-intensity chronic aerobic exercise increases tolerance to noxious ischemic stimuli. In contrast, pressure pain threshold and pain ratings during ischemia were unchanged after training. The results suggest that increases in pain tolerance may be one psychological aspect of exercise adaptation, which has not been demonstrated previously. This is the first study to clearly demonstrate an effect of chronic exercise on pain sensitivity in healthy, nonathlete adults. Previous cross-sectional studies of athletes have alluded to this adaptation, but findings have been mixed and depended heavily on the context in which pain was assessed.

The specific site and mechanisms of the observed training adaptations are difficult to ascertain and several possibilities exist. In general terms, physiological adaptations may have occurred that resulted in diminished signaling in response to the noxious stimulus. Alternatively, psychological adaptations may have occurred that simply permitted a greater tolerance of a similar level of discomfort, with activity preserved through the pain pathways. Pressure and ischemic pain are conveyed by different afferents (mechanosensitive and chemosensitive afferents, respectively) (13). The current study did not directly test the activity of either the mechanosensitive or chemosensitive nociceptors. Pressure pain thresholds were unchanged in both the upper and the lower body, although these two areas would have experienced different peripheral adaptations to cycle exercise training. Thus, it is unlikely that mechanoreceptor firing was altered.

We also propose that during the tourniquet test, nociceptor activity in the arm was the same pre- and posttraining, as this limb was untrained. Moreover, the arm was occluded from central circulatory influence during testing so that any cardiovascular training adaptations (e.g., increased cardiac output and associated delivery of nutrients to exercising muscle) were unlikely to improve during performance of the task. Therefore, changes in muscle nociceptor afferent signals originating at the periphery are unlikely to account for the increased ischemic pain tolerance or the different response of participants to ischemic but not pressure pain after training. This result provides evidence for a central mechanism as the primary modulator of the increased pain tolerance and suggests a new psychological adaptation to training. The afferents activated during the ischemic task are similar to those activated during exercise (19).Therefore, it is possible that prolonged performance of the ischemic task was facilitated by repeated exposure to the noxious ischemic stimulus during exercise training. Notably, the subjective rating of the ischemic pain was preserved, implying that a similar degree of discomfort was tolerated for a longer duration.

The impact of chronic exercise on ischemic pain tolerance, but not pressure pain thresholds, was perhaps surprising in light of many previous demonstrations that acute bouts of exercise consistently raise pressure pain thresholds (18). The mechanisms underlying hypoalgesia after exercise are unclear. Several theories have been proposed to explain how acute exercise reduces pain sensitivity. These include increases in endogenous opioids, cannabinoids and stress hormones, conditioned pain modulation (a form of endogenous pain inhibition in which pain in one location may inhibit pain in another), and changes in the attentional modulation of pain (i.e., distraction) (10,30,39). However, these remain equivocal for acute exercise and their importance with chronic exercise remains largely unknown. Indeed, the lack of change of pressure pain thresholds in the current study suggests that acute and chronic exercise influence pain sensitivity through different mechanisms.

The aspect, rather than the modality, of pain sensitivity that was assessed may explain the different findings for pressure and ischemic pain in the current study. Pressure pain was measured only as the point at which the mechanical stimulus became painful, whereas the assessment of ischemic pain concerned the capacity of the individual to tolerate a stimulus that was above the threshold for pain. Pain threshold is thought to predominantly reflect muscle nociception (32), whereas pain tolerance additionally involves a strong psychosocial and behavioral component (6). Although the current study reports an increase in tolerance to ischemic pain, Anshel and Russell (5) reported that tolerance to pressure pain increased in healthy adults after aerobic training. However, they assessed pain tolerance as the peak pressure that could be endured rather than the duration for which pain could be tolerated. In addition, cross-sectional comparisons of pain sensitivity between athletes and nonathletes indicate that athletes tolerate more pain when exposed to a range of noxious stimuli, whereas pain threshold usually does not differ between the groups (36). This effect is mediated by personality traits, coping strategies and a higher level of pain self-efficacy (17,28). An early study that compared thresholds and tolerances for different pain modalities in athletes and nonathletes found strong correlations between tolerance across modalities (31). On the other hand, in healthy individuals, when fitness is not taken into account, threshold and tolerance measures within pain modalities are more closely related than threshold measures or tolerance measures across modalities including ischemic, pressure and thermal pain (7,14).

There were several limitations to the present study. Although recruitment of participants was from the same university population of staff and students and conducted concurrently, the allocation of participants to the groups was not randomized. Furthermore, participants in the exercise group received more attention than participants in the control group as they were supervised for all 18 sessions, whereas control participants

only met with the investigator for the initial and final assessment. Therefore, behavioral artifacts cannot be discounted. Second, there was a significant difference in self-reported physical activity and $\dot{V}O_{2peak}$ between the groups at baseline, which may have influenced the results. For example, higher levels of physical activity, particularly vigorous activity, are associated with reduced pain sensitivity (11,26). However, pain sensitivity was not different between the groups at baseline. Moreover, there was no correlation between $\dot{V}O_{2peak}$ and pain sensitivity for either group. Therefore, it is unlikely that the initial difference in aerobic capacity between the groups at baseline influenced the results.

Lastly, based on the Modified Somatic Perceptions Questionnaire, the exercise group reported higher somatization compared with the control group both before and after the intervention. Somatization is the tendency to experience and communicate somatic symptoms in response to psychological stress (e.g., an increased HR and feeling hot all over) (20). This difference could mean that the exercise group had greater attention to the painful stimuli before training and greater scope to change with training. However, the scores for both groups were very low before and after the intervention (exercise group: 4.1 ± 2.6 and 2.8 ± 2.3, respectively; control group: 1.1 ± 0.8 and 0.8 ± 0.9, respectively); out of a possible score of 39 and from a clinical perspective, a score of 12 or more is considered necessary to influence pain sensitivity (22). Moreover, Main (21) found no relation between heightened somatic awareness (i.e., Modified Somatic Perceptions Questionnaire score) and ischemic pain tolerance. Therefore, the between group difference is unlikely to have influenced the results.

One implication of our results is that increasing pain tolerance may contribute to enhanced endurance performance via a greater tolerance of afferent feedback associated with metabolic disturbance in muscles. Despite the conjecture that surrounds the influence of signals from muscle afferents on endurance performance (4,24), it is generally accepted that they are important (1). Discharge of small-diameter muscle afferents, including nociceptors, increases in the presence of metabolites associated with muscle fatigue and this feedback inhibits central neural drive and subsequently performance (3). However, these same muscle afferents can also minimize locomotor muscle fatigue by stimulating ventilator and cardiovascular response to rhythmic exercise (2). Therefore, exercise training may facilitate the development of brain function that increases tolerance of these signals and associated sensations, and this increase in tolerance may contribute to improved endurance performance.

Our results also provide evidence of a systemic hypoalgesia after exercise training, whereby pain tolerance increased in the arm after 6 wk of training with the legs. This is consistent with reductions in pain sensitivity in nonexercising limbs in healthy adults and patients with peripheral arterial disease (5,40). This finding may have important clinical applications for exercise prescription in patients with persistent pain. For instance, patients with persistent pain may gain a pain relieving benefit of exercise by training with unaffected or pain free limbs. This would serve to improve their functional capacity and clinical outcomes, without the risk of exacerbating their symptoms. A transfer of endurance training to untrained limbs has previously been shown after exercise training. That is, exercise training with the lower body can improve $\dot{V}O_{2peak}$ and other cardiovascular parameters when subsequent exercise is performed solely with the upper body (37). This same transfer effect may also apply to pain sensitivity.

To conclude, the results from this study demonstrated that 6 wk of moderate- to vigorous-intensity aerobic exercise training increased pain tolerance in healthy individuals. This demonstration that exercise may influence pain sensitivity independently of disease provides new insight into how some clinical populations with low exercise tolerance and capacity may benefit from aerobic training. That is, increasing pain tolerance in these patients through regular aerobic training may facilitate more exercise as well as exercise at a higher intensity, which may provide greater clinical benefits.

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