THE EVALUATION OF THE AEROBIC EXERCISE EFFECTS ON PAIN TOLERANCE

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Abstract

The exercise hypoaolesic effects are reported. But, the effect of chronic exercise training on pain sensitivity is unclear. The study purpose is evaluation the effect of aerobic exercise training on pain sensitivity in healthy persons. Pressure pain threshold ischemic pain tolerance and pain ratings during ischemic were distinguished in 24 participants before and after 6 weeks of structured aerobic exercise training (n = 12) or after 6 weeks of normal physical activity\textsuperscript{(n = 12)}. The exercise training diet included of cycling three times per week for 30 min under 75% of maximal oxygen consumption reserve condition. There were significant increasing for aerobic fitness (P = 0.004) and ischemic pain tolerance (P = 0.036) in the exercise group after training and there was no significant difference for aerobic fitness (P > 0.1) or pain sensitivity (P > 0.1) in the control group. Moderate to strong intensity of aerobic exercise training increases ischemic pain tolerance in healthy persons.

Key Words: hypoaolesia, ischemic pain, exercise, aerobic, pain rating

Introduction

In healthy persons, there are a lot of studies about hypoaolesic effect of acute exercise (Naugle et al., 2012). Pain sensitivity reduction can be as a result of chronic exercise training in patients with continuous pain (Hoffman et al., 2004; Zwierska et al., 2005) and as an important part of treatment in these patients, exercise training can have important role (Busch et al., 2007; Hayden et al., 2005). Anshel and Russell (1994) examined the effect of 12 weeks of aerobic exercise and resistance exercise on pressure pain tolerance on 48 unfit males (Anshel and Russell, 1994). The studies have done 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 unclear 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 (Sternberg et al., 1998), as well as with the coping strategies used by different individuals (Manning and Fillingim, 2002; Ord and Gijsbers, 2003). Also, the volume, the intensity, the duration and the type of exercise training performed by the athletes have been poorly controlled or quantified. In addition to this, results have depended on the modality and protocol for evoking pain, in particular whether pain thresholds or pain tolerance have been measured. Chronic exercise causes to increase pain tolerance that reported by these cross-sectional investigations, but not pain threshold (Ord and Gijsbers, 2003; Scott and Gijsbers, 1981; Tesarz et al., 2012). This concept is supported by Anshel and Russell (1994), who indicated pain tolerance increase after exercise training. To our knowledge, this is the only study examining the effect of exercise training on pain sensitivity in healthy individuals. So, it is still unclear whether chronic exercise can influence on pain sensitivity independently of athletic status. This research was evaluated the effect of moderate to vigorous intensity of chronic aerobic exercise on pain sensitivity in healthy adults. Pressure and ischemic noxious stimuli were selected as they are arguably the most similar to the pain experienced during physical activity and chronic disease (Scott and Gijsbers, 1981). It was hypothesized that on the base of previous studies, chronic aerobic exercise would increase pain tolerance, but not affect pain threshold.

Materials and methods

Eligibility criteria of the persons included 1) healthy with no history of chronic pain or chronic disease 2) between the ages of 18 and 50 years old and 3) absence of a current diagnosis of depression. Twenty-seven participants that included 5 numbers males and 22 numbers females were selected for this study. All over the 6 weeks, 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 years; control: 2 males and 10 females, 21.8 ± 1.6 years; P = 0.013). The experiment for the exercise group consisted of 20 sessions that 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 weeks period. Before the initial and final assessments, participants were asked to abstain from vigorous exercise for 24 hours and from caffeine for 4 hours. 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 (Main et al., 1992). The Profile of Mood States was used to assess six subscales of mood (tension, depression, confusion, anger, vigor and fatigue) (Pollock et al., 1979). The long form of the International Physical Activity Questionnaire was used to evaluate physical activity levels (Craig et al., 2003). Pressure pain threshold was assessed for four muscular sites (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·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 intra-rater 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 in LabVIEW version 8 (National Instruments) to provide visual feedback of the grip force and auditory tones prompted the start and end of each contraction. During testing, subjective ratings of pain were recorded using a 0–10 numeric pain rating scale every 30 s (15). 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. A VO₂peak 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 pedalling 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 VO₂peak was obtained. Criteria used for the determination of VO₂peak 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. 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 (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 r² for each group, exercise: r² = 0.89, SD = 0.13; control: r² = 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 a = 0.05 level.

Results

The VO₂peak of participants is shown in Table 1. A significant group time interaction was indicated for VO₂peak with (F₁,₂₂ = 24.00, P = 0.001). A significant difference in VO₂peak between groups was observed at baseline (t₁₂ = 3.11, P = 0.02), but this difference disappeared after the intervention (t₂₂ = 0.24, P = 0.81). Exercise training caused a significant increase in VO₂peak (t₁₁ = 5.39, P = 0.004, +14.6%), whereas VO₂peak in the control group (t₁₁ = 1.45, P = 0.72, -2.8%). A significant grouptime interaction was observed for workload (F₁,₂₂ = 11.5, P = 0.003) but not peak HR (F₁,₂₂ = 3.63, P = 0.07) or RPE (F₁,₂₂ = 0.77, P = 0.39) during VO₂peak assessment. For participants in the exercise group, an increase in peak workload was observed in the final compared with the initial VO₂peak assessment (t₁₁ = -2.66, P = 0.08, +8.6%). Conversely, a decrease in peak workload was observed for participants in the control group in the final VO₂peak assessment compared with the initial V′O₂peak assessment (t₁₁ = 2.16, P = 0.2, -6.7%). Peak HR and RPE were unchanged in both groups between each VO₂peak assessment (P > 0.5; Table 1). 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 (t₁₁ = -2.67, P = 0.02, +9.4%), whereas the average RPE during these sessions remained unchanged (t₁₁ = 1.46, P = 0.17) (Table 2). The average RPE across all exercise sessions was 15, which equates to a subjective rating of “hard.”
Table 1. Duration and peak workload, HRmax, RPE, and RER during the maximal aerobic test before and after the intervention.

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>After</th>
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<tbody>
<tr>
<td><strong>Exercise</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VO(_{2})peak (mL·min(^{-1})·kg(^{-1}))</td>
<td>35.9 ± 5.6</td>
<td>41.7 ± 6.6</td>
</tr>
<tr>
<td>Duration (min)</td>
<td>6.5 ± 1.5</td>
<td>7.2 ± 1.6</td>
</tr>
<tr>
<td>Workload (W)</td>
<td>201.5 ± 42.7</td>
<td>218.3 ± 42.2</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>173.8 ± 11.5</td>
<td>176.2 ± 6.8</td>
</tr>
<tr>
<td>RPE</td>
<td>18.7 ± 1.3</td>
<td>18.9 ± 1.5</td>
</tr>
<tr>
<td><strong>Control</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VO(_{2})peak (mL·min(^{-1})·kg(^{-1}))</td>
<td>43.1 ± 4.8</td>
<td>42.7 ± 6.2</td>
</tr>
<tr>
<td>Duration (min)</td>
<td>7.5 ± 1.2</td>
<td>7.2 ± 1.5</td>
</tr>
<tr>
<td>Workload (W)</td>
<td>217.6 ± 31.9</td>
<td>203.2 ± 37.9</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>101.2 ± 12.5</td>
<td>178.3 ± 12.1</td>
</tr>
<tr>
<td>RPE</td>
<td>18.7 ± 1.5</td>
<td>18.6 ± 1.1</td>
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<tr>
<td>RER</td>
<td>1.4 ± 0.8</td>
<td>1.2 ± 0.8</td>
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</tbody>
</table>

Data are presented as mean ± SD. The duration of the maximal aerobic test does not include the warm-up or cool down.*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.

<table>
<thead>
<tr>
<th></th>
<th>First Session</th>
<th>Final Session</th>
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<tbody>
<tr>
<td>Workload (W)</td>
<td>97.6±21.2*</td>
<td>106.3±24.2*</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>161.1±46.5</td>
<td>159.5±47.8</td>
</tr>
<tr>
<td>RPE</td>
<td>15.5±1.4</td>
<td>14.2±1.4</td>
</tr>
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</table>

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

The 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\(_{1,22} = 8.4, P = .008\). 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. 1).

Ischemic pain tolerance was not significantly different between groups at baseline (t\(_{2} = -0.92, P = 1\)). No relationship was observed between the change in duration of ischemic contractions and the change in VO\(_{2}\)peak for either group.

However, when both groups were combined, there was a significant positive relationship between the change in VO\(_{2}\)peak and the change in ischemic pain tolerance.

Pressure and ischemic pain are conveyed by different afferents (mechanosensitive and chemosensitive afferents, respectively) (Giordano, 2005). The afferents activated during the ischemic task are similar to those activated during exercise (Light et al., 2008). 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 (Koltyn, 2002). 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 (Ellingson and cook, 2011; Ruble et al., 2005;
Yarnitsky et al., 2010). 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 (Scholz and Woolf, 2002), whereas pain tolerance additionally involves a strong psychosocial and behavioural component (Baker and Kirsch, 1991). Cross-sectional comparisons of pain sensitivity between athletes and non-athletes indicate that athletes tolerate more pain when exposed to a range of noxious stimuli, whereas pain threshold usually does not differ between the groups (Teszar et al., 2012). This effect is mediated by personality traits, coping strategies and a higher level of pain self-efficacy (Johnson et al., 2012; Ord and Gijsbers, 2003). 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 (Bhalang et al., 2005; Hastie et al., 2005). Despite the conjecture that surrounds the influence of signals from muscle afferents on endurance performance (Amann and Secher, 2010; Marcora, 2010), it is generally accepted that they are important (Amann, 2012). Our results also provide evidence of a systemic hypoalgesia after exercise training, whereby pain tolerance increased in the arm after 6 weeks of training with the legs. This is consistent with reductions in pain sensitivity in non-exercising limbs in healthy adults and patients with peripheral arterial disease (Anshel and Russell, 1994; Zwierska et al., 2005). 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 VO2peak and other cardiovascular parameters when subsequent exercise is performed solely with the upper body (Tordi et al., 2001).

Conclusion

To conclude, the results from this study demonstrated that 6 weeks 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.

References


**PROCJENA EFETATA AEROBNE VJEŽBE NA TOLERANCIJU BOLI**

**Sažetak**

Prijavljeni su efekti hipoalgezijske vježbe. Ali, efekt kroničnog treninga na osjetljivost na bol je nejasan. Svrha istraživanja je procjena efekta aerobnog treninga na osjetljivost na bol u zdravih osoba. Prag pritiska boli, ishemijska tolerancija boli i ocjene boli za vrijeme ishamije su izdvojeni kod 24 sudionika prije i poslije 6 tjedana strukturiranog aerobnog treninga (n=12) ili nakon 6 tjedana uobičajene fizičke aktivnosti (n=12). Prehrana treninga je uključivala vožnju bicikla tri puta tjedno po 30 minuta ispod 75% od stanja rezerve maksimalne potrošnje kisika. Postojalo je značajno povećanje u aerobnom fitnesu (P=0,004) i ishemijskoj toleranciji boli (P=0,036) u grupi treninga nakon treninga i prag pritiska boli i ocjene boli nisu pokazali značajna razlike u ishamiji (P>0,2). Nije postojala značajna razlika za aerobni fitnes (P>0,1) ili osjetljivost na bol (P>0,1) u kontrolnoj grupi. Umjeran do jak intenzitet aerobnog treninga uvećava ishemijsku toleranciju boli u zdravih osoba.

**Ključne riječi:** hipoalgezija, ishemijska bol, vježba, aerobik, ocjena boli

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