Aerobic Exercise and Cold Pressor Test Induce Hypoalgesia in Active and Inactive Men and Women

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Abstract

Background. Physical inactivity is a risk factor for chronic pain. Several mechanisms play a role in pain chronification including impairment of pain inhibition.

Objective. This study compared the efficiency of pain inhibitory systems between physical active and inactive healthy subjects. It was hypothesized that active subjects had more efficient pain inhibition compared with inactive subjects.

Design. A randomized, crossover study with 2 days of data collection.

Methods. Fifty-six (28 females) subjects participated in this study. Subjects were subgrouped into active (n = 30) and inactive (n = 26). Conditioned pain modulation (CPM) was assessed by cold pressor testing. Exercise-induced hypoalgesia (EIH) was assessed after 15 minutes bicycling at a heart rate corresponding to 75% VO2max. A control session of 15 minutes quiet rest was also included. Pressure pain thresholds (PPTs) were recorded at the dominant arm and leg before, immediately after, and 15 minutes after conditioning and exercise as well as before and after rest. PPTs were also recorded during conditioning.

Results. At baseline, PPTs in inactive men were increased compared with inactive women (P < 0.003). During cold pressor test and after exercise, PPTs increased to the same degree in active and inactive subjects, and the CPM and EIH responses were correlated (P < 0.05). The CPM response immediately after cold pressor test was maintained in women but not in men.

Conclusions. Cold pressor stimulation and aerobic exercise caused comparable multisegmental increases in PPT in active and inactive men and women. The CPM and EIH responses were correlated, but they have different temporal manifestation of hypoalgesia.

Key Words. Exercise-Induced Hypoalgesia; Conditioned Pain Modulation; Pressure Pain Thresholds; Physical Activity; Pain Modulation

Introduction

Physical inactivity may be a risk factor for development of chronic pain and has been shown to facilitate neuronal...
responses to minor muscle insults [1]. Regular exercise has been linked to alterations in pain perception and central processing of pain where, e.g., athletes have significantly higher pain tolerance compared with normally active controls [2]. Few studies in healthy subjects have examined the relationship between physical activity and pain modulation efficacy such as conditioned pain modulation (CPM) and exercise-induced hypoalgesia (EIH). Interestingly, impaired CPM is often found in chronic pain patients [3] in whom also physical activity is often reduced [4]. Studies comparing pain modulation in athletes and normally active controls during a cold pressor test have so far been inconclusive, with one study showing increased CPM and one study showing decreased CPM in athletes [5,6]. A recent study found that greater amount of self-reported physical activity as well as greater amount of vigorous physical activity predicted greater pain modulation, assessed as change in thermal pain sensitivity during cold pressor test [7]. However, the CPM protocol used in these previous studies assessed the effect of cold pressor test on heat pain, primarily affecting the skin, which may not be relevant to most musculoskeletal pain conditions, where assessment of the pressure pain sensitivity may be more relevant [8].

Pain modulation is often assessed by recordings of pain thresholds before and after paradigms of CPM [9,10] and EIH [11–13]. CPM is typically triggered by applying a painful conditioning stimulus, e.g., cold pressor test [9], whereas EIH is often assessed by moderate to high intensity aerobic exercise [14]. In healthy subjects, both paradigms are known to cause an acute decrease of the pain sensitivity [15,16]. Both opioidergic and non-opioidergic mechanisms have been proposed for CPM and EIH although studies comparing the two phenomena are sparse. The results of a recent study indicated that CPM may contribute to the EIH response, due to the painful experience during exercise [17]. Although similarities exist [18], differences in both temporal and spatial manifestations of CPM and EIH have been documented [19].

No studies have so far examined a potential difference in pain modulation of the pressure pain sensitivity between normally active and inactive healthy subjects after both cold pressor test and exercise. The primary purpose of this study was to compare the effects of a cold pressor test and an exercise condition on pressure pain thresholds in normally active and inactive healthy subjects. Secondary purpose was to compare temporal and spatial manifestations of CPM and EIH. It was hypothesized that both cold pressor test and exercise would have greater effects on pressure pain thresholds in active subjects compared with inactive subjects and that the temporal and spatial manifestations of CPM and EIH would be different.

Materials and Methods

Subjects

Active and inactive subjects were recruited by advertisement at the local university. An a priori power analysis determined that at least 26 participants per group were required to examine the effect of CPM and EIH on pressure pain, with a power of 0.80, \( \alpha \leq 0.05 \), and a large effect size. None of the participating subjects had any relation to the authors’ research departments. Subjects were subgrouped into active and inactive groups based on their self-reported amount of physical activity in an average week during the last 3 months. Subjects performing less than 30 minutes of aerobic physical activity per week were classified as inactive and subjects performing more than 60 minutes of moderate to high intensity aerobic physical activity were classified as active. Thirty minutes of aerobic physical activity per week was chosen to ensure that inactive subjects were indeed inactive but also to reach 26 participants in each group as required. Walking at normal speed was not included in the total time of aerobic activity per week. Fifty-six healthy subjects (age: median 22 years [range 20–30 years]; body mass index [BMI]: median 22.1 kg/m² [range 16.4–38.7]; 28 women) were included in this study. The active subjects (N = 30) were primarily students at the Institute of Sports Science at the University of Southern Denmark, whereas the inactive subjects (N = 26) were typically students studying law, history, and medicine at the University of Southern Denmark. Three of the active subjects also performed some strength exercises in their regular exercise routine. None of the inactive subjects performed any physical activities of moderate or high intensity.

None of the included subjects suffered from neurological, psychological, and cardiovascular diseases, had any pain, or used any pain medication during the week prior to participating. All subjects were asked to refrain from physical exercises, coffee, and nicotine on the days of assessment. The study was conducted in accordance with the Declaration of Helsinki, approved by the local ethical committee (S-20120014), and all subjects provided written informed consent.

Procedure

The subjects participated in two sessions approximately at the same time of the day and separated by 1–3 weeks. In the first session, subjects were introduced to the procedures. CPM was assessed by cold pressor test on the dominant hand in one of the sessions, and EIH was assessed through an aerobic bicycling exercise in the other session. The order of the conditions was randomized and counterbalanced between the two sessions. Each session lasted approximately 60 minutes. On the same day as performing the cold pressor test, subjects also performed a control condition, which was 15 minutes of quiet rest. The order between the cold pressor test and quiet rest conditions was randomized and counterbalanced. The CPM effect in the current protocol has previously been shown to be short lasting [19], and a 20 minutes recovery interval was maintained between the two conditions.

Pressure pain thresholds (PPTs) at the arm and leg were used to assess the efficacy of the cold pressor stimulation, exercise, and rest. PPTs were recorded before and
immediately after the control condition as well as before, immediately after, and 15 minutes after the cold pressor test and exercise condition. PPTs were also recorded during cold pressor test, when the hand had been immersed in the cold water for 1 minute, as the CPM response has been shown to be significantly larger during cold pressor test compared with after the cold pressor stimulation [19].

**Pressure Algometry**

PPTs were assessed using a handheld pressure algometer (Somedic AB, Hörby, Sweden) with a stimulation area of 1 cm². The rate of pressure increase was kept approximately at 30 kPa/s, and the first time the pressure was perceived as pain, the subject pressed a button and the pressure intensity defined the PPT. Two PPT assessments were completed for each site. Reliable measures of PPT in healthy humans have been demonstrated when PPT is calculated as the mean of trials compared with a single trial [20], and the average of the two measurements was used for statistical analysis. Twenty-second intervals between assessments were kept. PPT measurements were conducted with the subject lying supine on a plinth. The two assessment sites were located and marked. Site one was located in the middle of the dominant quadriceps muscle, 10 cm proximal to the base of patella. Site two was located in the middle of the dominant biceps brachii muscle, 10 cm proximal to the cubital fossa.

**Conditioned Pain Modulation**

The cold pressor test was performed with the subject comfortably lying supine while immersing the dominant hand into a tank containing circulating ice water at 1–2°C. The subject’s hand was immersed to 5 cm above the wrist for 2 minutes. PPT assessments were performed after immersion of the hand for 1 minute, immediately after termination of the cold pressor test, and 15 minutes after. Rating of pain intensity during cold pressor stimulation was obtained on a 0–10 numerical rating scale (NRS), with 0 defined as “no pain” and 10 “as worst imaginable pain” just before the hand was removed from the ice water. After the PPT assessments were performed, the subject was asked to relax quietly on the plinth for a 15-minute recovery period, which was concluded with a final set of PPT assessments.

**Exercise-Induced Hypoalgesia**

All subjects performed a 15-minute bicycling exercise. In the beginning of the first session, the age-related target heart rate (ATHR) corresponding to 50% VO₂ max and 75% VO₂ max were determined. In short, based on a previously described method [21], the relation between heart rate (HR) and VO₂ max was used to determine the HR adequate for obtaining the target intensities. The bicycling intensity was chosen based on previous studies that have shown robust EIH at this intensity [12,19,22,23]. The seat post of the stationary cycle (Ergomadic 928E, Monark Exercise AB, Vansbro, Sweden) was adjusted so that the subject had approximately 5-degree bend at the knee during the bottom phase of the pedal stroke. An HR monitor (Monark Heart Rate Monitor, Monark Exercise AB) was strapped around the subject’s chest. Subjects were instructed to maintain a pedal rate as close to 70 rounds per minute as possible throughout the 15 minutes. The first 2 minutes was used as warm-up. Resistance was then increased over the next 3 minutes until the ATHR was achieved by the beginning of the fifth minute where after the subject continued bicycling for additional 10 minutes. HR was monitored constantly, and resistance was manipulated if necessary to keep the HR at the desired level. Rating of perceived exertion (RPE: 6–20) and HR (beats/min) due to the exercise condition was obtained just before reaching the 15 minutes of bicycling. After 15 minutes of bicycling, PPT assessments were performed as described. After the PPT assessments were performed, the subject was asked to relax quietly on a plinth for a 15-minute recovery period, which concluded with a final set of PPT assessments.

**Statistics**

Results in text and figures are presented as mean and standard deviation, unless otherwise specified. In case the data did not pass the Kolmogorov–Smirnoff test for normality, nonparametric tests were used accordingly. A Mann–Whitney U-test was used to analyze time spent on physical activity, age, BMI, RPE during bicycling, HR during bicycling, and rating of pain intensity (NRS) during cold pressor test and bicycling between the two groups. A three-way repeated-measures analysis of variance (RM-ANOVA) was performed to analyze the effect of gender and physical activity (active vs inactive) on baseline PPTs, with gender and physical activity as group factors and assessment site (arm and leg) as repeated measure. The effect of repeated pain assessments and multiple conditions on PPTs was analyzed with a two-way RM-ANOVA of the PPTs prior to each condition with condition (cold pressor test, exercise and quiet rest) and site (arm and leg) as repeated measures. The effect of sequence between cold pressor test and quiet rest on PPTs prior to rest was analyzed with a two-way RM-ANOVA with factor site (arm and leg) as repeated measures and sequence (order: “cold pressor test—quiet rest” or “quiet rest—cold pressor test”) as group factor.

The effect of quiet rest, cold pressor test, and exercise on PPT was analyzed with a four-way RM-ANOVA with factor time (rest: before and after; cold pressor test: before, during, immediately after, and 15 minutes after; exercise: before, immediately after, and 15 minutes after) and site...
(arm and leg) as repeated measures and gender and physical activity as group factor.

For comparison of the CPM response and the EIH response, a four-way RM-ANOVA was used with the factor conditions (cold pressor test and exercise), site (arm and leg) as repeated measures and gender and physical activity as group factor. In case of significant factors or interactions in the RM-ANOVAs, the Newman–Keuls (NK) test was used for post hoc comparisons incorporating correction for the multiple comparisons. In case of significant factors or interactions, effect sizes were calculated based on Hedges' $g$, due to dissimilar group sizes.

Spearman’s Rank Order correlation was run to determine the relationship between the time spent on physical activity and the CPM response (PPTs before cold pressor test subtracted from PPTs during cold pressor), the relationship between the time spent on physical activity and the EIH response (PPTs before exercise subtracted from PPTs immediately after exercise), the relationship between pain intensity during cold pressor test and the CPM response, as well as the relationship between the CPM response and the EIH response. To correct for multiple correlations, $P$ values were Bonferroni corrected. $P$ values less than 0.05 were considered significant.

**Results**

**Physical Activity Behavior**

The time spent on aerobic physical activity reported by the active subjects (median: 180 minutes; range: 60–420 minutes) was significantly longer compared with inactive subjects (median: 0 minute; range: 0–30 minutes; Mann–Whitney $U: P < 0.001$). There was no significant difference between the two physical activity groups for age (active: median 22 years, range 20–28; inactive: median 22 years, range 21–30; Mann–Whitney $U: P > 0.9$) and BMI (active: median 22.0 kg/m$^2$, range 16.4–38.7; inactive: median 22.3 kg/m$^2$, range 19.5–26.0; Mann–Whitney $U: P > 0.2$).

**Pain Sensitivity at Baseline**

A significant interaction between gender and physical activity was found for baseline PPTs ($F[1,52] = 6.76$, $P < 0.02$), with post hoc test showing significantly higher PPTs in inactive men (485.8 ± 237.0 kPa, mean of both sites) compared with inactive women (316.1 ± 95.9 kPa; NK: $P < 0.003$). A significant main effect for site was also found in the ANOVA ($F[1,52] = 25.02$, $P < 0.001$), with post hoc test showing significantly higher PPTs at the quadriceps site (397.3 ± 153.1 kPa) compared with PPTs at the biceps site (322.0 ± 127.2 kPa; NK: $P < 0.001$).

Overall, there was no significant difference among the baseline PPTs prior to the three conditions ($F[2,110] = 1.67$, $P = 0.19$). There was a significant effect on PPTs prior to the resting condition depending on sequence between cold pressor test and rest ($F[1,54] = 4.56$, $P < 0.04$), with post hoc test showing signifi-

ificantly higher PPTs prior to rest when cold pressor test was performed before rest compared with PPTs when rest was performed before cold pressor test (NK: $P < 0.04$).

**Quiet Rest in Active and Inactive Subjects**

There was no significant change in PPTs after quiet rest compared with before quiet rest ($P > 0.4$; Figure 1).

**Conditioned Pain Modulation in Active and Inactive Subjects**

CPM effects in active and inactive subjects are summarized in Table 1. All 56 subjects completed the cold pressor test. There was no significant difference in the pain intensity rated during cold pressor test between the two physical activity groups (Table 2) and likewise no significant correlation between NRS and change in PPTs during cold pressor test.

As illustrated in Table 1, the effect sizes indicate that inactive subjects had a modest effect on PPTs compared with active subjects during cold pressor test, but no significant main effect or interactions with the factor physical activity were found in the ANOVA. However, a significant two-way interaction between time and gender was found in the ANOVA ($F[3,156] = 3.23$, $P < 0.03$; Figure 2), with post hoc test showing significantly increased PPTs during the cold pressor test compared with baseline, immediately after and 15 minutes after in both men and women and significantly increased PPTs immediately after cold pressor test compared with baseline in women only (NK: $P < 0.002$). There was no significant correlation between time spent on physical activity and the CPM response during cold pressor test (quadriceps: $r[56] = −0.085$, $P = 0.53$; biceps: $r[56] = −0.106$, $P = 0.44$).

**Exercise-Induced Hypoalgesia in Active and Inactive Subjects**

There was no significant difference in RPE, or HR during bicycling between the two physical activity groups (Table 2) and likewise no significant correlation between RPE or HR and change in PPTs immediately after exercise.

EIH responses in active and inactive subjects are summarized in Table 1. As illustrated in Table 1, the effect sizes indicate that active subjects had a modest effect on PPTs compared with inactive subjects after exercise, but no significant main effect or interactions with the factor physical activity were found in the ANOVA. However, a significant main effect of time was found in the ANOVA ($F[2,104] = 20.37$, $P < 0.001$; Figure 3), with post hoc test showing significantly increased PPTs during exercise compared with baseline (NK: $P < 0.001$). There was no significant correlation between time spent on physical activity and the EIH response immediately after exercise (quadriceps: $r[56] = 0.094$, $P = 0.49$; biceps: $r[56] = 0.081$, $P = 0.55$).
Comparing CPM and EIH Responses

A significant interaction between condition and site was found in the ANOVA (F[1,35] = 10.27, P < 0.003), with post hoc test showing significantly increased CPM response at both sites (quadriceps: 30.2 ± 34.2% of baseline; biceps: 41.9 ± 33.3% of baseline) compared with the EIH response (quadriceps: 18.6 ± 20.7% of baseline; biceps: 11.1 ± 15.8% of baseline; NK: P < 0.005). The CPM response was significantly larger at the biceps site compared with the quadriceps site (NK: P < 0.009).

There was a weak but significant correlation between the CPM response and the EIH response (quadriceps: r[56] = 0.347, P = 0.009; biceps: r[56] = 0.330, P = 0.013; Figure 4).

Discussion

This study is the first to compare the manifestations of CPM and EIH in normally active and inactive healthy subjects. Robust EIH and CPM responses compared with a control condition were found in both active and inactive

Table 1  Mean (95% CI, N = 56) percentage change in pressure pain thresholds compared with baseline recorded at the quadriceps and biceps sites during cold pressor test (CPM) and after exercise (EIH)

<table>
<thead>
<tr>
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<th>Active Subjects % Baseline</th>
<th>Inactive Subjects % Baseline</th>
<th>Effect Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPM response quadriceps</td>
<td>37.1 (26.0–48.2)</td>
<td>39.2 (21.1–57.2)</td>
<td>−0.06</td>
</tr>
<tr>
<td>CPM response biceps</td>
<td>43.7 (32.3–55.0)</td>
<td>51.8 (33.5–69.9)</td>
<td>−0.22</td>
</tr>
<tr>
<td>EIH response quadriceps</td>
<td>22.9 (14.6–31.2)</td>
<td>17.9 (6.6–29.2)</td>
<td>0.20</td>
</tr>
<tr>
<td>EIH response biceps</td>
<td>19.9 (7.7–32.2)</td>
<td>16.1 (4.3–27.9)</td>
<td>0.12</td>
</tr>
</tbody>
</table>

Effect size between groups is based on Hedges’ $g$. 

Figure 1 Mean (± SEM, N = 56) pressure pain threshold (PPT) recorded at quadriceps (quad) and biceps (biceps) muscles before and immediately after 15 minutes quiet rest in active and inactive subjects.
men and women. The CPM effect was maintained immediately after the cold pressor test in women but not in men.

Pain Sensitivity at Baseline

The pressure pain sensitivity at baseline was significantly lower in inactive men compared with inactive women but without significant difference between active men and women. The finding in inactive subjects is in agreement with previous studies reporting increased experimental pain sensitivity in women [24,25]. There was no significant difference in PPTs at baseline between active and inactive subjects. Previous comparison of the pressure pain sensitivity in active and inactive subjects is sparse, but the present observation is in agreement with a recent

Table 2 Mean (range) rated pain intensity (NRS: 0–10) during cold pressor test as well as perceived exertion (RPE: 6–20) and heart rate (beats/min) after 15 minutes of bicycling

<table>
<thead>
<tr>
<th></th>
<th>Active Subjects</th>
<th>Inactive Subjects</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cold pressor test</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercise</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity, NRS (0–10)</td>
<td>8 (4–10)</td>
<td>8 (2–10)</td>
<td>0.16</td>
</tr>
<tr>
<td>Rating of perceived exertion, RPE (6–20)</td>
<td>15</td>
<td>14</td>
<td>0.23</td>
</tr>
<tr>
<td>(11–18)</td>
<td>(12–17)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>167</td>
<td>167</td>
<td>0.79</td>
</tr>
<tr>
<td>(148–179)</td>
<td>(155–177)</td>
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</table>

Figure 2 Mean (± SEM, N = 56) pressure pain threshold (PPT) recorded at quadriceps (quad) and biceps (biceps) muscles before, during, immediately after and 15 minutes after cold pressor test in men and women. The cold pressor test was applied to the dominant hand. Significantly different compared with baseline (*, NK: $P < 0.05$).
meta-analysis, which concluded that although significant difference in pain tolerance between athletes and normally active controls, PPTs were comparable [2].

Conditioned Pain Modulation in Active and Inactive Subjects

Cold pressor test decreased the pressure pain sensitivity, which is in agreement with previous studies [15,26]. Although the effect sizes in this present study do indicate that the inactive subjects have a better CPM effect (small effect size) compared with active subjects, there was no significant difference in the CPM response in active subjects compared with inactive subjects. This finding was unexpected. First of all, because regular physical activity has been linked to decreased pain ratings to repeated noxious stimuli [27] and increased CPM effects [28]. However, reduced pain inhibition during cold pressor test have been shown in athletes compared with normally active controls in a recent study [5] so the activity related effects may be highly dependent on the level of activity.

Exercise-Induced Hypoalgesia in Active and Inactive Subjects

EIH occurred with multisegmental manifestations following 15 minutes of bicycling at ATHR corresponding to 75% VO$_2$ max in line with previous studies [12,19,22,23]. Although, the effect sizes indicate that the active subjects had a better EIH (small effect size) compared with inactive subjects, there was no significant difference in the EIH response in active subjects compared with inactive subjects. These findings are supported by findings from two small studies with 20 active subjects and nine inactive
subjects [31] and 10 athletes and 10 nonathletes [32], respectively. Pressure pain ratings during an ischemic tourniquet test was assessed before and after maximal treadmill exercise with no difference in the EIH response between active and inactive subjects [31], and pain ratings during cold pressor test were assessed before and after a submaximal running exercise with no difference in the EIH response between athletes and nonathletes [32]. More EIH in inactive subjects compared with active subjects could have been anticipated due to the unfamiliar physical stress during exercise, which might activate a larger stress-induced analgesic response compared with active subjects. Studies have indicated that there appears to be a dose-related hypoalgesic effect [19,33] and had a more intense cycling exercise (e.g., 85% VO2 max) or a different exercise modality been used, it may have resulted in a different outcome.

EIH was found to have manifestations in local as well as remote body parts, indicating systemic mechanisms responsible for EIH. This finding is in agreement with previous findings of multisegmental EIH effects [34,35].

EIH after bicycling was comparable in men and women. Past research indicated that women produce more hypoalgesia than men in response to exercise [19,36,37]. Possible reasons for the discrepancy are different exercise and pain outcome protocols. Compared with men, Sternberg et al. [37] showed increased EIH in women after running 10 minutes at 85% of maximal HR, and Koltyn et al. [36] found a larger EIH response in women after submaximal isometric exercise when using a thermal pain measure as outcome.

**Similarities in CPM and EIH**

Both the cold pressor stimulation and the exercise paradigm produced multisegmental increases in PPTs. Moreover, a significant correlation was found between the CPM response and the EIH response. These findings indicate
that CPM and EIH could partly be mediated through a common mechanism. Elevated blood pressure has been repeatedly associated with reduced sensitivity to painful stimuli [38,39], and this relationship may be the result of inhibitory baroreceptor effects on the central nervous system [40,41]. Cold pressor test [42] and acute exercise [43] cause systemic increases in blood pressure, which could explain the multisegmental effects. Although similar multisegmental increases in PPTs were found in this study, a difference in temporal manifestations between CPM and EIH was also found. In general, cold pressor test caused an increase in PPTs during cold pressor test, whereas a significant increase in PPTs after exercise was also found immediately after as well as 15 minutes after exercise.

Limitations regarding the interpretation of the results from this study should be taken into consideration. First of all, the nonsignificant difference in CPM and EIH between active and inactive subjects may be due to the small sample size, which could be leading to a type 2 error. Second, subgrouping based on subjects physical activity status was not derived based on an exhaustive physical performance test and determination of VO2 max but on self-reported physical activity behavior during an average week over a 3-month period, which may create some concern in terms of interpretation of the findings. Determination of VO2 max, muscle strength, or the use of accelerometers will improve the methodology of future studies investigating the pain inhibitory systems in active and inactive subjects. Third, the experimental design did not take into consideration potential carry over effects from the cold pressor test to the control condition, which was the case. This could potentially influence the results in the resting condition. However, the duration of the significant CPM response was less than 15 minutes, indicating short lasting effects.

Conclusions

Cold pressor stimulation and aerobic exercise caused multisegmental increases in pressure pain thresholds in active and inactive healthy men and women. No significant differences in the CPM and EIH responses between active and inactive subjects were found. The CPM and EIH responses were positively correlated, but they have different temporal manifestation of hypoalgesia. Aerobic exercise can produce hypoalgesic effects in addition to the general health improvement, regardless of the individual’s physical activity behavior, which may be relevant for rehabilitation purposes.

References


