Biological Psychology 113 (2016) 46–51

Contents lists available at ScienceDirect

Biological Psychology

journal homepage: www.elsevier.com/locate/biopsycho

# A smaller magnitude of exercise-induced hypoalgesia in African Americans compared to non-Hispanic Whites: A potential influence of physical activity

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ARTICLE INFO

*Article history:*
Received 1 July 2015
Received in revised form
22 September 2015
Accepted 15 November 2015
Available online 28 November 2015

*Keywords:*
Race and ethnicity
Central pain modulation
Minority health

ABSTRACT

This study compared exercise-induced hypoalgesia (EIH) between African Americans (AAs, $n = 16$) and non-Hispanic Whites (NHWs, $n = 16$), and examined the potential influence of physical activity (PA) on the racial/ethnic difference in EIH. The PA levels were quantified using a questionnaire, and intensity of electrical stimulus to produce moderate pain was individually determined. Participants squeezed a hand dynamometer at 25% of their maximal strength for three minutes, followed by a three-minute post-exercise rest. Numeric ratings to electrical stimulus at the pre-determined intensity were recorded every one minute during and after exercise. Compared to NHWs, AAs reported less lifestyle PA. Both AAs and NHWs showed EIH, but AAs exhibited a smaller magnitude of EIH than NHWs. However, this difference in EIH disappeared after controlling for the lifestyle PA levels. The results suggest that AAs exhibit less efficient pain modulation than NHWs, and AAs' reduced PA could potentially explain the observed difference in EIH.



## 1. Introduction

Research has been conducted examining racial/ethnic difference in sensitivity to experimental pain stimuli in African Americans (AAs) and non-Hispanic Whites (NHWs), and results from this research generally indicate that AAs are more sensitive to a variety of experimental pain stimuli compared to NHWs (e.g., heat, cold, ischemic stimuli) (Campbell & Edwards, 2012; Edwards, Fillingim, & Keefe, 2001; Rahim-Williams, Riley, Williams, & Fillingim, 2012). Also, physiological thresholds of spinal reflex responses are found to be lower in AAs compared to NHWs (Campbell, France et al., 2008). Together, these observations suggest an increased sensitivity to experimental pain stimuli among AAs compared to NHWs.

Muscular contraction has been found to produce naturally-occurring pain in the exercising muscles (Cook, O'Connor, Eubanks, Smith, & Lee, 1997; Umeda, Newcomb, Ellingson, & Koltyn, 2010). It has been suggested that the localized muscle pain during exercise may occur due to stimulation of Type III and Type IV afferent fibers via increased intramuscular pressure and biochemical products produced during muscular contraction (Ellingson & Cook, 2013; O'Connor & Cook, 1999), and research indicates that even submaximal exercise involving smaller muscles can produce a mild-to-moderate intensity of muscle pain (Umeda et al., 2010). Consistent with the increased sensitivity to experimental pain stimuli among AAs, our recent study then demonstrates that AAs report a greater intensity of muscle pain during submaximal isometric exercise compared to NHWs (Umeda, Williams, Marino, & Hilliard, 2015).

It has been suggested that pain sensitivity is determined by the complex interactions of endogenous pain modulatory mechanisms that can either inhibit or facilitate nociceptive transmission (Millan, 2002); therefore, it is possible that the consistent observations on the increased pain sensitivity among AAs potentially suggest a functional difference in central pain modulatory processing between AAs and NHWs. Interestingly, evidence shows that exercise does not only produce localized muscle pain, but also reduces sensitivity to experimental pain stimuli in healthy adults (Koltyn, 2000; Naugle, Fillingim, & Riley, 2012). This hypoalgesic phenomenon has been termed as exercise-induced hypoalgesia (EIH) in the literature, and previous research shows that hypoalge-

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[1] (http://www.depts.ttu.edu/hess/)

http://dx.doi.org/10.1016/j.biopsycho.2015.11.006
0301-0511/

sia occurs during and immediately after exercise with a variety of experimental pain stimuli (e.g., pressure, electrical, thermal stimuli) in a systemic manner (e.g., contralateral to exercised muscles) (Koltyn, 2000; Naugle et al., 2012). Also, more recent research indicates that exercise reduces the magnitude of temporal summation of pain (Koltyn, Knauf, & Brellenthin, 2013; Vaegter, Handberg, & Graven-Nielsen, 2014) and that endocannabinoid system may be involved in EIH (Koltyn, Brellenthin, Cook, Sehgal, & Hillard, 2014). Therefore, these data collectively demonstrate that pain modulatory processing within the central nervous system is involved in EIH, suggesting that EIH can be used as a laboratory test to examine central pain modulatory processing. However, no study has been conducted to date to compare the EIH responses between AAs and NHWs.

Very little is currently known regarding the factors that influence the function of central pain modulatory processing; however, the increasing body of evidence from recent research appears to suggest that the function of central pain modulatory processing may be influenced by physical activity (PA) levels. For example, there is some evidence that physically active individuals show reduced sensitivity to experimental pain stimuli compared to less physically active individuals (Andrzejewski, Kassolik, Brzozowski, & Cymer, 2010; Ellingson, Colbert, & Cook, 2012; Freund et al., 2013; Johnson, Stewart, Humphries, & Chamove, 2012), and exercise interventions successfully reduce pain sensitivity among healthy adults (Anshel & Russell, 1994; Jones, Booth, Taylor, & Barry, 2014). Furthermore, several studies have been conducted examining the potential influence of regular exercise on central pain modulatory processing using the other laboratory test termed as conditioned pain modulation (CPM), which has been suggested to be mediated by descending pain modulation (Le Bars, 2002; van Wijk & Veldhuijzen, 2010). Results from the studies then show the potential benefits of regular exercise on central pain modulatory processing using this experimental paradigm (Geva & Defrin, 2013; Naugle & Riley, 2014; Umeda, Lee, Marino, & Hilliard, 2015). In contrast, one study indicates that both physically active and less active adults show the comparable EIH responses (Vaegter, Handberg, Jorgensen, Kinly, & Graven-Nielsen, 2014), whereas the other study indicates that endurance athletes show a smaller magnitude of CPM responses compared to healthy adults (Tesarz, Gerhardt, Schommer, Treede, & Eich, 2013). However, the null findings from these studies may be due to several methodological factors, including operational definition of active individuals and conditions of experimental testing. Therefore, although more research is needed in this area, it appears that there is some empirical evidence in the literature suggesting that PA levels may influence the function of central pain modulatory processing. This potential influence of PA on central pain modulatory processing is important to consider the functional difference in central pain modulatory processing in AAs and NHWs because it has been shown that AAs are less physically active compared to NHWs (Hillier, Tappe, Cannuscio, Karpyn, & Glanz, 2014; Trost, Owen, Bauman, Sallis, & Brown, 2002) and spend more time for sedentary behaviors (Cohen et al., 2013). Therefore, the purpose of this study was to compare the functional difference in central pain modulatory processing between AAs and NHWs using the EIH paradigm, and examine the potential influence of PA on the racial/ethnic difference in the EIH responses.

## 2. Materials and methods

### *2.1. Participants*

Healthy adults who identified themselves as AA or NHW were recruited to participate in the study. The inclusion criteria for this study were (1) 18–30 years of age, (2) self-identification as African American or non-Hispanic White, (3) no medical conditions diagnosed by their physician, and (4) no medication use. The participants were excluded from the study if (1) they indicated medical contraindications for exercise, (2) they were currently pregnant or breastfeeding, or (3) they planned to be pregnant or breastfeed in the near future. The two groups were matched based on age (±3 years) and gender. The study protocol was fully approved by an institutional review board, and all participants signed a consent form before participating in the study.

Power analysis was performed to estimate a sample size to accurately detect significant difference in EIH between AAs and NHWs. Effect size was first calculated using our pilot data, and the analysis was performed with a large effect size, an $\alpha = 0.05$, and a power = 0.80. The analysis indicated that approximately 12–17 AAs and 12–17 NHWs would be needed for this study.

### *2.1. Measures*

#### *2.1.1. Physical activity levels*

To quantify the PA levels, the Baecke Physical Activity Questionnaire (BPAQ) was used in this study. The BPAQ consists of three subscales to estimate work, sport, and leisure related PA, and reliability and validity of the BPAQ have been well-investigated in the previous studies (Baecke, Burema, & Frijters, 1982; Jacobs, Ainsworth, Hartman, & Leon, 1993). The work-related PA subscale assesses the type of occupation and the activity levels associated with their occupation. The sport-related PA subscale quantifies the amount of PA that the participants are recreationally engaging in (e.g., playing basketball, swimming), whereas the leisure-related PA subscale quantifies the amount of lifestyle PA associated with routine, daily activities (e.g., walking, riding a bike), except for the recreational PA. Scores from the three subscales are summed to compute the total PA levels, with the higher scores indicative of more PA. Although the BPAQ does not estimate the actual time spent for different type or intensity of PA, previous research has shown that the BPAQ scores correlate to other measures of PA and physical fitness (e.g., PA history, VO2 max, % body fat, etc.) in expected directions (Jacobs et al., 1993).

#### *2.1.2. Pain catastrophizing*

The Pain Catastrophizing Scale (PCS) was used in this study to assess pain catastrophizing, a psychological trait regarding one's negative thoughts and feelings related to pain experience (Sullivan, Bishop, & Pivik, 1995). The PCS consists of the rumination, magnification, and helplessness subscales, and scores from the three subscales were summed to obtain the total score, with the higher scores indicative of greater pain catastrophizing. Previous studies have examined reliability and validity of the PCS (Osman et al., 1997; Sullivan et al., 1995), and it has been shown that pain catastrophizing is a psychological factor that may influence the EIH responses in healthy adults (Naugle, Naugle, Fillingim, & Riley, 2014).

### *2.2. Procedures*

The participants were asked to visit our laboratory for two sessions. Upon arrival at our lab for the first session, the participants were first asked to sign a consent form, and then to complete a questionnaire regarding demographics and general health, the BPAQ, and the PCS.

The participants were then asked to squeeze a hand dynamometer as hard as possible with their dominant hand to measure the maximal voluntary contraction (MVC). The MVC assessment was conducted twice, and the average MVC was used to calculate a target force (25% MVC) for the exercise protocol in the next session. The MVC assessment was followed by a familiar-