Ethnic Differences in the Perception of Pain through Vagal-Nociceptive Networks

A Senior Research Thesis

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By

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Abstract

Individual differences in pain perception have been well documented to factors such as age, gender, culture, and ethnicity. As the response to nociceptive stimuli involves activation of the sympathetic nervous system, physiological measures are used to observe changes in neural activity associated with pain perception and the autonomic nervous system. While research on ethnic differences in pain perception has been conducted through various psychophysiological measures, underlying mechanisms through vagally mediated pathways, specifically indexed by vmHRV, have not been thoroughly investigated. The current study aims to investigate the situation in a sample of healthy undergraduate students of various ethnicities. Participants completed a full psychological evaluation and basic health questionnaires prior to starting the experiment. Physiological data was recorded throughout the experiment. A 5-minute baseline period was measured with the participant’s right hand on a thermal plate at 36˚ C. Afterwards, the temperature was raised at a controlled rate up to 52˚ C. Participant’s pain threshold and tolerance was recorded at their respective temperatures and time after onset of increasing temperature. Results indicate European Americans (EAs) and non-European Americans (non-EAs) differ in pain threshold \((p = .041, r = .25)\) and tolerance \((p = .035, r = .26)\) in respect to temperature. EAs and Non EAs also differ in pain threshold \((p = .037, r = .26)\) and marginally for tolerance \((p = .061, r = .23)\), with respect to time. No differences between EAs and Non EAs for vmHRV were observed. Moreover, regardless of ethnicity, changes in vmHRV from baseline to after the painful stressor, were marginally associated with pain tolerance \((r(61) = -.238, p = .060)\). In addition to determine how different ethnicities differ in pain perception – possibly as a result of different ethnicity related stressors - the current study looks at how differences in
autonomic regulatory systems and vagal-nociceptive network is an underlying mechanism for these differences.

Acknowledgments

I would like to thank everyone in the Emotions and Quantitative Psychophysiology Lab for all of the training and support they have provided over the past 4 years. First and foremost I would like to thank my advisors Dr. Julian Koenig and Dr. Julian Thayer for providing mentorship throughout my time in the lab, without which this project would not have been possible. I would also like to thank DeWayne Williams for his continuous support and enthusiasm through the completion of this project, providing a supportive environment to work in and willing to answer any question at a moment’s notice.

Introduction

Pain

Pain is defined as an unpleasant sensory or emotional experience associated with actual or potential tissue damage (International Association for the Study of Pain). Overall, it is a subjective experience with many external factors moderating the perception – such as age, culture, gender, personality, and sympathetic arousal. The sensory–discriminative and affective-cognitive dimensions (Millan, 1999) are two different components that are responsible for our overall experience of pain. The sensory-discriminative component concerns perception and detection of noxious stimuli while the affective-cognitive component concerns the relationship between pain, mood, and the capacity to cope (Millan, 1999).

The type of nociceptors (A-delta or C fibers) activated are responsible for the type of the pain experienced. Pain can be somatic or visceral. Pain is characterized as first pain or second pain, eliciting signals in the fast conducting A-delta fibers or slow conducting C fibers
respectively. The myelination of A-delta fibers is responsible for its role in “fast” pain transmission. Because of this myelination, first pain is characterized as a transitory and sharp response, responsible for sharp, mechanical and thermal pain. It has also been widely known as the sensory – discriminative component of pain. The stimulation of these fibers has been associated with the activation of the primary and secondary somatosensory cortex (Ploner et al., 2002).

C fibers are slower in conduction of pain signals due to the lack of myelin on the fibers. The types of pain felt through these fibers are dull and aching polymodal (mechanical, heat and chemical) pain. This “second pain” is largely responsible for the affective component of pain, with large responses in the anterior cingulate cortex (ACC) and second somatosensory cortex (Ploner et al., 2002).

These fibers are part of the Anterolateral System, conveying pain, temperature, as well as crude touch. The ascending pathways transport sensory information to structures in the brainstem through projection neurons (Willis, 1985). All fibers synapse in the dorsal horn of the spinal cord, where there are three major paths responsible for the perception of pain leading to the brain - the spinothalamic tract, the spinoreticular tract, and the spinomesencephalic tract. The spinothalamic tract is responsible for the location and perception of pain (sensory-discriminative component). The information gets relayed to the sensory cortex, which further relays information to the reticular formation. This is then relayed to areas such as the hypothalamus and thalamus. The relay between the reticular formation and thalamus and hypothalamus provide insight to the autonomic response to pain. The spinoreticular tract is responsible for the emotional aspect of pain, while the spinomesencephalic tract is responsible for the modulation of the pain experience (affective-motivational component). The spinomesencephalic tract projects into the
Ethnic Differences in vmHRV and Pain Perception

Bhatt

periaqueductal grey (PAG), superior colliculus and nucleus cuneiformis in the midbrain. The reciprocal connections between the PAG and limbic system explain the affective-motivational component of pain experience (for review of cortical representation of pain, see Treede et al., 1999). The descending pain analgesia system acts to modify pain perception. These pathways either amplify or suppress nociceptive information via the PAG and the rostral ventromedial medulla (Mayer & Liebeskind, 1974). The spinomesencephalic tract originates in multiple parts of the spinal cord and projects to the PAG and parabrachial nucleus (PBN), most notably terminating in the amygdala, which allows for the influence of psychological processes dealing with emotion (Millan, 2002).

An important component of thermal nociception is the vanilloid receptor type 1 (TRPV1; Caterina et al., 2000). It is a ligand-gated non-selective cation channel expressed in the central nervous system (CNS) and the periphery on dorsal root ganglion (DRG) neurons (Valtschanoff et al., 2001). They can be activated (produce a conformational change in the protein and thus activating the neuron by opening the ion channel) by multiple types of stimuli including capsaicin, low pH, and the modality related to the current study, heat (Szallasi and Blumberg, 1999). Activation of this receptor plays a key role in thermal (heat) nociceptive transmission. The sensation of burning temperatures is dependent on the voltage-dependent activation curve of TRPV1. Specifically, as temperature increases, a leftward shift in the voltage-dependent activation curve of TRPV1 is observed (Voets et al., 2004).

Pain can be considered as allostatic load on the body. Allostasis can be thought of the ability of physiological systems, including the cardiovascular, neuronal, immune, and endocrine systems, to maintain stability when confronted with a stressor (McEwen, 1998). Pain is an adaptive signal that warns an individual of real or potential damage. However, persistent pain
leads to cumulative strain on the brain, allostatic load, and alterations in normative psychological processes such as perception, emotion, cognition, and motivation that are closely intertwined with physiological responses of interoception and autonomic function (Simons et al., 2014).

One of pain’s primary functions is to promote survival. It can broadly be categorized as either adaptive pain or maladaptive pain. Adaptive pain’s role is to contribute to an organism’s survival by protecting it from injury or promoting healing when an injury has occurred (Woolf, 2004). This type of pain triggers our sympathetic nervous system (SNS; fight – or – flight) response to activate (Jänig, 1995). Prolonged exposure to these stimuli can cause many different physical and psychological complications, including hypervigilance, muscular reactivity, escape/avoidance, and guarding behaviors that maintain or exacerbate pain and promote pain-related disability (Verbunt et al., 2003).

*Neurovisceral Integration Model (NIM)*

The autonomic nervous system (ANS) is comprised of the inhibitory parasympathetic nervous system (PNS) and the excitatory SNS. The PNS is alternatively referred to as the “rest and digest” system (e.g. decrease heart rate, respiration rate, pupil size). The SNS is referred to as the “fight-or-flight” system (e.g. increase heart rate, pupil size, induce sweating). The SNS is associated with subcortical structures in the brain (e.g. amygdala), and thus, associated with the fear response and emotions that serve as its precursors. The activity of the SNS and PNS on autonomically-regulated organs (e.g., the heart) works in opposition of each other. In addition, it is responsible for the visceral sensory sensation of pain. Overall, it is posited that activity from sub-cortical brain structures are mechanisms of inhibitory control through descending pathways via the vagus nerve, to the periphery organs (i.e. the heart). This is such that the PNS is in dominance while at rest, keeping the body at an optimal healthy state (i.e. autonomic balance;
Thayer & Lane, 2000). Both the SNS and PNS regulate the heart, creating time intervals between heartbeats called heart rate variability (HRV).

Activity of the vagus nerve (10th cranial nerve) is indicative of many visceral motor functions. Specifically, it is responsible for innervating activity of the heart, lungs, and abdominal organs via the parasympathetic nervous system. The vagus nerve exhibits bidirectional activity due to having both afferent and efferent fibers. In relation to the heart, efferent fibers originate in the brain stem and terminate in the sino-atrial (SA) node. The inhibitory nature of these fibers slows heart rate by decreasing SA firing (Levy & Warner, 1994). Vagal afferent fibers originate in the heart and project to the nucleus tractus solitaries in the medulla, providing continuous feedback to the brain that facilitates regulation (Porges, Dous-sard-Roosevelt, Portales, & Greenspan, 1996).

Vagally mediated HRV (vmHRV) is a reputable physiological measure of individual differences over the past decades, characterized as the variability in milliseconds of subsequent R-R intervals – or the inter-beat-intervals (IBI) on an electrocardiogram (ECG). It is a measure of the dual innervation between sympathetic and parasympathetic influences on heart rate that yields information about autonomic flexibility, thereby representing the capacity for regulated emotional responding (Appelhans & Luecken, 2006). Overall, it is proposed that an individual with high vmHRV is representative of a healthy and adaptive organism. Increased vmHRV is indicative of greater emotion regulation capacity, cognitive flexibility, and overall adaptability to a previous, ensuing, and future stressor (Thayer & Lane, 2000).

**Pain and Vagal Activity**

The vagus nerve is involved in acute nociceptive pathways at many levels. Experimental evidence shows that activation of vagal afferent fibers by electrical stimulation could facilitate or
inhibit responses of dorsal horn neurons in response to applying noxious heat to the skin (Ren, Randich, & Gebhart, 1989.) The role of vagal afferents based on this study in nociception is to “facilitate perception of relevant stimuli” and “inhibition of nociceptive transmission via linkage with known endogenous pain control systems” (Ren, Randich, & Gebhart, 1989). The network responsible for this phenomenon is bidirectional, linking descending inhibitory pathways from cerebral structures to the dorsal horn. These pathways are capable of modulating nociceptive information. Descending inhibition is relayed by the nucleus tractus solitarius (NTS) to the rest of the brain. The NTS receives major input from the vagus nerve and is representative of the first relay station for vagally mediated nociception (Haines, 2004). Decreased vagal activity may be representative of greater somatic and visceral input via the spinothalamic tract, which provides a mechanism for decreased pain threshold and increased pain sensitivity (Ammons et al., 1983).

It has been argued that the relative dominance of the sympathetic nervous system (SNS) over the parasympathetic nervous system (SNS) plays a major role in chronic pain conditions. (Barakat et al., 2012; Martínez- Martínez et al., 2014). One of the most prominent ways to measure ANS activity is vmHRV (Task Force, 1996). Heart rate (HR) is controlled via the PNS and SNS activity in concert and is under tonic inhibitory control via the vagus nerve (PNS dominance; Jose & Collison, 1970). Other measures of autonomic activity include blood pressure (BP), skin conductance response (SCR) – a measure of sympathetic response – and respiration rate.

**Ethnic Differences**

Ethnic differences and ethnicity-related stressors have been studied as a predictor of a well-being of health (Williams, Spencer & Jackson, 1999). Previous research suggests that the subjective experience of racial bias contributes to racial disparities in health (Williams,
Ethnic Differences in vmHRV and Pain Perception

Neighbors, & Jackson, 2003). Ethnic discrimination is a stressor that involves unfair treatment due to one’s ethnicity and is highly prominent in the United States. Contrada et al. (2001) found that ethnicity-related stress may influence physical and psychological well-being including depressive symptoms, life satisfaction and symptoms of physical illness.

Individual differences in ethnicity have been well documented with respect to autonomic activity. Research in ethnic differences of vmHRV has shown inconsistent results regarding to baseline vmHRV (Dorr, Brosschot, Sollers & Thayer, 2007; Liao, Barnes, Chambless, Simpson, Sorlie, Heiss, 1995; Faulkner, Hathaway & Tolley, 2003; Lampert, Ickovics, Horwitz & Lee, 2005). A recent meta-analysis (Hill, Hu, Koenig, Sollers, Kapuku, Wang, Snieder & Thayer, 2014) posits that African Americans (AAs) have higher vmHRV compared to their European American (EA) counterparts. Even with age as in important covariate (Thayer, Yamamoto & Brosschot, 2010; Thayer & Lane, 2007), AAs have higher vmHRV at all age levels compared to EAs (Hill et al., 2014). Moreover, ethnic differences in pain sensitivity have been well documented across various pain modalities. Specifically, Rahim-Williams et al. (2012) found that AAs displayed a lower pain tolerance and threshold compared to EAs. Additionally, it has been observed in children that Asians had higher pain sensitivity than Caucasians, who had higher pain sensitivity compared to AAs and Hispanics (Lu, Zeltzer & Tsao, 2013), even when controlling for gender and age.

Present Study

The present study aims to identify mechanisms that could possibly be underlying ethnic differences in experimental pain response. Nociceptive transmission involves activation of the SNS which is also indicative of autonomic activity. As the vagus nerve is a major inhibitory pathway on pain processing and indicative of autonomic activity, it is hypothesized that changes
and differences in vagal-tone among different ethnicities can provide a physiological pathway and the underlying mechanism underlying ethnic differences in noxious experiences. In line with existing evidence, it is hypothesized that AAs will show greater baseline vmHRV compared to EAs. AAs will show greater pain sensitivity indexed by lower pain threshold and tolerance levels compared to EAs. Adding to the scientific literature, it is hypothesized that greater vmHRV is associated with lower pain sensitivity in EAs, but not AAs. Greater pain sensitivity is expected to be associated with increased cardiovascular reactivity following the painful stressor. Finally, lower baseline vmHRV, is expected to be associated with increased cardiovascular reactivity to the painful stressor in EAs, but not AAs. Overall, this is proposed to provide further evidence for ethnic differences in nociceptive processes and ANS function.

Methods

Participants

All participants (N = 64; mean age = 19.56, SD = 1.09; 28 female) were recruited from the Research Experience Program (REP) at The Ohio State University (OSU). All participants provided informed consent after being given an explanation of the procedure in order to fulfill a partial course requirement. A physical assessment consisting of acquiring the participant’s height (in), weight (lbs) and waist circumference (in) was then conducted. BMI was calculated using height and weight. These measures are important confounders that will later serve as important covariates when analyzing the HRV data.

Questionnaires

All participants were first instructed to complete a general questionnaire indicating their age, date of birth, socioeconomic status and ethnicity. Questionnaires evaluating positive and negative health behaviors will be assessed using the short form of the International Physical
Activity Questionnaire (IPAQ; Booth, Ainsworth & Pratt, 2013), the Alcohol Use Disorders Identification Test (AUDIT; Barbor et al., 2001), a short version of the Smoking and Tobacco Use Questionnaire (STUQ; Pirkle et al., 1996) and Drug Use Questionnaire (DAST-10; Skinner, 1982). General health status will be measured using the Brief Symptom Inventory (BSI-18; Derogatis, 2000) and the Short General Health Questionnaire (GHQ-12; Goldberg, 1992).

**Physiological Data**

All physiological recordings were collected using a 16-channel bioamplifier (NeXus 16; Mind Media B.V.; Roermond-Herten; The Netherlands). Heart rate (HR) was continuously recorded using a three-lead ECG at 1000 Hz. Three Ag-AgCl hypoallergenic surface electrodes (SKINTACT; Leonhard Lang Gmbh, Austria) were connected to the device and placed (1) below the right clavicle, (2) just below the left ribcage and (3) below the right ribcage. Respiration rate was sampled 32 Hz using a respiration belt placed on the chest. All physiological measurements were collected using the software BioTrace+ (Mind Media B.V.; Roermond-Herten; The Netherlands). The baseline and recovery HRV data was exported from BioTrace+ as inter-beat-interval (IBI) lists via text files in milliseconds and further analyzed using Kubios software (Tarvainen et al., 2014).

**Nociceptive Stimulation and Pain Ratings**

All nociceptive stimulation was applied using an AHP-1800CPV Versatile Cold/Hot Plate (TECA Corp., Chicago, IL, USA). Each participant’s pain *threshold*, pain *tolerance* and pain *intensity* was measured (pain intensity data will be used in subsequent analyses). Pain threshold was defined by the moment the body first perceives the stimulus as painful. The participant verbally indicating “pain” determined pain threshold. Pain tolerance was the
maximum amount of pain the person was able to tolerate, and was determined by the participant taking their hand off of the stimulus.

The process of nociceptive stimulation began with the participant placing their right hand on the thermal plate. The plate was previously set to 36º C. Participants placed their hand on the plate for 5 minutes as a baseline measurement. Subsequently, the temperature rose by 1º C every 10 seconds until it reached 52º C. The machine then stopped increasing in temperature to prevent tissue damage. The time point (seconds) and temperature (intensity; ºC) was recorded for pain tolerance and threshold. Additionally, continuous intensity ratings were collected from the moment of pain threshold until the moment of pain tolerance (not used in current analysis).

Procedure

All of the experiments were conducted within soundproof rooms at the Emotions and Quantitative Psychophysiology Lab at The Ohio State University. The experimenter and the participant were seated in different rooms but able to communicate through a dual microphone-speaker system and could be monitored through a live-feed camera. Participants first provided written consent. They first completed the general socio-demographic, health and psychological questionnaires. Afterwards, they were attached to the physiological equipment as vmHRV and respiration rate were recorded continuously throughout the remainder of the experiment. A 5-minute baseline was followed by the pain task. A recovery period was also measured to index preservative physiological activity in reaction to the painful stressor.

Data Analysis

Due to a lack of participants of ethnic minority, participants were categorized as EAs and non-European Americans (non-EAs) for preliminary analyses. The sample consisted of 40 EAs,
11 AAs, 3 Hispanic Americans, 4 South Asian Americans, 5 Mixed Americans, and 1 Middle Eastern American. All ethnicities other than EA (24 participants) were combined into the “non-EA” group. IBI text files from BioTrace+ were first analyzed in Kubios to correct for any artifacts there may have been present. The root mean squared of successive differences (RMSSD) of R-R intervals was used as a time-domain measure reflecting vmHRV (Stein et al., 1994). High frequency (HF) was used as the frequency domain measure, which represents primarily parasympathetic influences of vagal activity (Saul, 1990). To correct for non-normally distributed data, both measures of RMSSD and HF were natural log (ln) transformed to meet the assumptions of linear analysis (lnRMSSD and lnHF respectively), thus allowing the use of parametric tests (Ellis et al., 2008). Independent sample t-tests were run to determine if there were any differences between EAs and non-EAs during baseline and recovery vmHRV (both lnRMSSD and lnHF). Wilcoxon Mann Whitney tests were also run to determine if there were any differences between ethnic groups on pain threshold and tolerance (measures of both temperature and seconds). Zero-order and Partial correlations were run between measures of vmHRV and pain tolerance and threshold while controlling for age, gender, BMI, physical activity (IPAQ) handedness and whether the participant was EA or non-EA. Zero-order and Partial correlations controlling for the same variables were also run between pain measures and ΔlnRMSSD and ΔlnHF (change in cardiovascular activity from baseline to recovery) to determine if changes in cardiovascular activity were associated with pain sensitivity. Finally, independent sample tests were run to determine if there were differences in ΔlnRMSSD and ΔHF between EAs and non-EAs. The level of significance for all tests was set to 0.05. All analyses were carried out using SPSS 22 (IBM Corp. Released 2013. IBM SPSS Statistics for Macintosh, Version 22.0, Armonk, NY: IBM Corp).
Results

The mean and standard deviation of pain and vmHRV measures can be found in Appendix A. Wilcoxon Mann Whitney tests revealed significant differences between EAs (Median (Mdn) = 38.92) and non-EAs (Mdn = 40.07) for pain threshold in respect to temperature in °C (U = 333.0, n₁ = n₂ = 64, Z = -2.039, p = .041, r = .25; see figure 1) and time (Mdns = 49; 71 respectively) in seconds (U = 330.0, n₁ = n₂ = 64, Z = -2.081, p = .037 r = .26; see figure 3). Tests revealed significant differences in EAs (Mdn = 42.48) and Non-EAs (Mdn = 43.34) for pain tolerance with respect to temperature (U = 328.0, n₁ = n₂ = 64, Z = -2.018, p = .035, r = .26; see figure 2) and marginal differences (Mdns = 105; 113 respectively) with respect to time (U = 345.0, n₁ = n₂ = 64, Z = -1.873, p = .061, r = .23; see figure 4). There were no differences between groups in baseline lnRMSSD (t(62) = -0.715, p = .477, Cohen’s d = -0.138) or lnHF (t(62) = -0.202, p = .840, d = .009). There were also no differences in recovery lnRMSSD (t(62) = -0.406, p = .686, d = -0.034) or lnHF (t(62) = .347, p = .730, d = .169). Zero-order and Partial correlations revealed no significant associations between measures of baseline or recovery vmHRV and measures of pain. However, analyses revealed a trending/marginally significant association of ∆lnRMSSD with pain tolerance temperatures using zero-order (r = -0.238, p = .060) and partial correlations (r = -0.227, p = .095; see figure 5). There were no differences between EAs and non-EAs in ∆lnRMSSD (t(62) = 1.038, p = .303, d = .287) or ∆lnHF (t(62) = 1.523, p = .133, d = .433).
Fig. 1: Mann-Whitney test reveal differences between EAs and ethnic minorities with respect to pain sensitivity (temperature) with EAs experiencing lower pain threshold temperatures. The y-axis is units of rank (smallest number of entire sample is assigned rank of 1, and largest number is assigned rank of \(N\) (64)). of temperature in °C. Horizontal lines indicate median values for each group. *\(p < .05\).
Fig. 2: Mann-Whitney test reveal differences between EAs and ethnic minorities with respect to pain sensitivity (temperature) with EAs experiencing lower pain *tolerance* temperatures. The y-axis is units of rank (smallest number of entire sample is assigned rank of 1, and largest number is assigned rank of $N$ (64)) of temperature in °C. Horizontal lines indicate median values for each group. *$p < .05$.*
Fig. 3: Mann-Whitney test reveal differences between EAs and ethnic minorities with respect to pain sensitivity (time) with EAs experiencing lower pain *threshold* temperatures. The y-axis is units of rank (smallest number of entire sample is assigned rank of 1, and largest number is assigned rank of *N* (64)). of time in seconds. Horizontal lines indicate median values for each group. *p < .05.*
Fig. 4: Mann-Whitney test reveal marginal differences between EAs and ethnic minorities with respect to pain sensitivity (time) with EAs experiencing lower pain threshold temperatures. The y-axis is units of rank (smallest number of entire sample is assigned rank of 1, and largest number is assigned rank of N (64)). of time in seconds. Horizontal lines indicate median values for each group. † = .06.
Figure 5: Changes in cardiovascular activity from baseline to recovery are associated with levels of pain tolerance in respect to temperature. Individuals with lower pain tolerance experienced greater cardiovascular reactivity.

**Discussion**

To summarize the results that partially confirm the hypotheses, differences in pain sensitivity were observed among EAs and non-EAs, with EAs experiencing heightened pain sensitivity in terms of pain threshold and tolerance (lower temperatures and time). There were no differences in baseline or recovery vmHRV (lnRMSSD or lnHF) between groups. No associations were observed between measures of baseline and recovery vmHRV (both lnRMSSD and lnHF) and pain measures (both threshold and tolerance). Cardiovascular reactivity was associated with pain tolerance measures in respect to temperature such that higher pain sensitivity was associated with higher reactivity. No differences in cardiovascular reactivity were observed in terms of ethnicity.
Previous research investigating pain sensitivity in minorities has found results contradictory to the current study in terms of pain sensitivity (for a recent review, see Rahim-Williams et al., 2012). Past studies using the same modality of pain induction as the current study (heat pain) have found that AAs exhibit higher pain sensitivity compared to non-Hispanic Whites (NHWs). In relation to the analyses of the current study, aggregate data from past studies reveal very small effect sizes for pain threshold (unweighted, -0.01, weighted, 0.06) and a moderate effect for pain tolerance (NHW compared to Hispanics; -0.59) when subjected to thermal pain (Rahim-Williams et al., 2012). Even though the original hypotheses aimed to explore differences between EAs and AAs, the current preliminary analysis adds to the growing body of research of ethnic differences in thermal pain perception.

The lack of power due to small sample size in the current study can heavily account for the null findings regarding vmHRV. However, autonomic correlates in relation to thermal nociception have been observed, which would support the hypothesis that vmHRV has a role in thermal nociception. Staud et al. (2007) conducted an fMRI study investigating brain activity to thermally induced (specific to C – fibers and TRPV1 receptors) pain. Regions of the brain associated with vmHRV, such as the anterior cingulate cortex (ACC; reflecting affect) and prefrontal cortex (PFC; reflecting higher order executive functioning) were also associated with these nociceptive pathways, supporting that effects on vmHRV may be observed after analysis of ongoing data collection.

In the present study, the painful stimulus can be considered a physical and psychological stressor. HRV following a stressor (recovery vmHRV) is considered a reliable measure of preservative cognition – an excessive state of worry and rumination about the stressor (Brosschot et al., 2008; Brosschot et al., 2010). As our results show that lower pain tolerance is associated
with increased recovery vmHRV, it can be hypothesized that increased pain sensitivity levels are indicative of greater perseverative cognition. One possible route of investigation would be to analyze the vmHRV data during the exposure to the noxious stimulus. Lower vmHRV during exposure may translate to higher stress, which could be a reason for differences in pain tolerance. This hypothesis could be supported by vmHRV’s ability to be indicative of an individual’s emotion regulation capacity, such that higher vmHRV is associated with greater emotion regulation capacity (Thayer et al., 2012). Thus, individuals with higher vmHRV that have any sorts of cognitive-behavior inconsistencies may show greater effortful control - which has been shown to be associated with vmHRV (Tatterstall et al., 1995; Gillie et al., 2012) - to help alleviate these inconsistencies.

Overall, these preliminary results support the notion of ethnic differences in pain perception, but not as a result of vagally mediated nociceptive pathways. Future analyses could investigate the role of various ethnicity related stressors and their effect on vmHRV and pain perception. The current results do not support ethnic differences in vmHRV. Moreover, changes in vmHRV are associated with perceived pain tolerance levels. In addition to ongoing data collection, further research on ANS changes using outcome measures such as SC and respiration rate would serve useful in understanding autonomic mechanisms underlying ethnic differences pain perception.

References


Ethnic Differences in vmHRV and Pain Perception


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Appendix A
<table>
<thead>
<tr>
<th></th>
<th>Pain Threshold (°C)</th>
<th>Pain Tolerance (°C)</th>
<th>Pain Threshold (sec)</th>
<th>Pain Tolerance (sec)</th>
<th>Baseline LnRMSSD</th>
<th>Baseline lnHF</th>
<th>Recovery LnRMSSD</th>
<th>Recovery lnHF</th>
<th>∆lnRMSSD</th>
<th>∆ln HF</th>
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<tr>
<td>EA</td>
<td>39.510 (2.04)</td>
<td>42.588 (1.576)</td>
<td>58.500 (27.633)</td>
<td>102.050 (21.155)</td>
<td>3.993 (.738)</td>
<td>7.207 (1.249)</td>
<td>4.124 (.776)</td>
<td>7.501 (1.290)</td>
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<td>NON-EA</td>
<td>40.904 (2.656)</td>
<td>43.518 (1.871)</td>
<td>77.167 (38.127)</td>
<td>114.542 (25.468)</td>
<td>4.086 (.599)</td>
<td>7.195 (1.323)</td>
<td>4.148 (.595)</td>
<td>7.277 (1.367)</td>
<td>.062 (.164)</td>
<td>.082 (.210)</td>
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<td>TOTAL</td>
<td>40.033 (2.369)</td>
<td>42.937 (1.738)</td>
<td>65.500 (32.960)</td>
<td>106.734 (23.473)</td>
<td>4.028 (.686)</td>
<td>7.202 (1.267)</td>
<td>4.133 (.708)</td>
<td>7.417 (1.313)</td>
<td>.105 (.257)</td>
<td>.215 (.545)</td>
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Appendix A: Means and standard deviations of all pain and vmHRV measures are included in the table above.