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Biopsychosocial Factors That Shape the Acute Physical Pain Experience: The Role of Simpatía

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Biopsychosocial Factors That Shape the Acute Physical Pain Experience: The Role of Simpatía

DISSENYATION

To be submitted in partial satisfaction of the requirements for the degree of

DOCTOR OF PHILOSOPHY

In Psychological Science

by

Amanda M. Acevedo

Dissertation Committee:
Associate Professor Sarah D. Pressman, Co-chair
Associate Professor Belinda Campos, Co-chair
Associate Professor Michelle A. Fortier

2019
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACKNOWLEDGEMENTS</td>
<td>v</td>
</tr>
<tr>
<td>CURRICULUM VITAE</td>
<td>viii</td>
</tr>
<tr>
<td>ABSTRACT OF THE DISSERTATION</td>
<td>xvi</td>
</tr>
<tr>
<td>INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>Background and Theoretical Context</td>
<td>2</td>
</tr>
<tr>
<td>Acute Physical Pain</td>
<td>2</td>
</tr>
<tr>
<td>Ethnic Differences in Pain Sensitivity</td>
<td>7</td>
</tr>
<tr>
<td>Biopsychosocial Factors that Shape the Acute Physical Pain Experience: The Role of Simpatía</td>
<td>9</td>
</tr>
<tr>
<td>The Current Project</td>
<td>18</td>
</tr>
<tr>
<td>AIMS OF THE DISSERTATION</td>
<td>19</td>
</tr>
<tr>
<td>Figure 1: Summary of research aims</td>
<td>19</td>
</tr>
<tr>
<td>Method</td>
<td>20</td>
</tr>
<tr>
<td>Results</td>
<td>34</td>
</tr>
<tr>
<td>Table 1: Sample demographics</td>
<td>35</td>
</tr>
<tr>
<td>Figure 2: Average self-reported pain during cold pressor for Latinos by simpatía value</td>
<td>36</td>
</tr>
<tr>
<td>Figure 3: Conceptual model of the mediation analyses conducted for the current study</td>
<td>37</td>
</tr>
<tr>
<td>Table 2: Results for each pathway of the state emotion mediation models predicting self-reported pain</td>
<td>38</td>
</tr>
<tr>
<td>Table 3: Expressions made during first 30 seconds of pain and correlations with simpatía in Latinos</td>
<td>40</td>
</tr>
<tr>
<td>Table 4: Results for emotion expression mediation models predicting self-reported pain</td>
<td>42</td>
</tr>
<tr>
<td>Table 5: Results for vagal tone mediation models predicting self-reported pain</td>
<td>44</td>
</tr>
<tr>
<td>Figure 4: Participants who remained in cold pressor task, by half-minute</td>
<td>47</td>
</tr>
<tr>
<td>Figure 5: Latino unstandardized coefficients for the association between sex, simpatía, and the propensity to withdraw from pain early</td>
<td>48</td>
</tr>
<tr>
<td>Table 6: Results for state emotion mediation models predicting pain tolerance</td>
<td>49</td>
</tr>
<tr>
<td>Table 7: Results for emotion expression mediation models predicting pain tolerance</td>
<td>52</td>
</tr>
<tr>
<td>Table 8: Results for vagal tone mediation models predicting pain tolerance</td>
<td>54</td>
</tr>
<tr>
<td>Figure 6: Latino and Europeans participants unstandardized coefficients for the association between sex, simpatía, and the propensity to withdraw from pain early</td>
<td>55</td>
</tr>
<tr>
<td>Figure 7: Latino and European participants unstandardized coefficients for the association between simpatía and the propensity to withdraw from pain early</td>
<td>56</td>
</tr>
<tr>
<td>Figure 8: Heart rate response to the cold pressor task (n = 60)</td>
<td>58</td>
</tr>
<tr>
<td>Table 9: Multilevel between person effects of the interaction of simpatía with heart rate recovery</td>
<td>59</td>
</tr>
<tr>
<td>Figure 9: Simpatía by heart rate recovery interaction</td>
<td>60</td>
</tr>
<tr>
<td>Figure 10: RMSSSD response to the cold pressor task (n = 60)</td>
<td>61</td>
</tr>
<tr>
<td>Figure 11: Predicted parasympathetic response to the cold pressor task</td>
<td>63</td>
</tr>
<tr>
<td>Figure 12: PEP response to the cold pressor task</td>
<td>64</td>
</tr>
</tbody>
</table>
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I have had the incredible opportunity to call three smart, powerful, and strong women my academic advisors at UCI: Sarah Pressman, Belinda Campos, and Michelle Fortier. This triad of awesomeness has pushed me to braid my research interests together in ways I originally thought were not possible, so thank you!

Sarah, you were the one who convinced me to come to UCI! After talking with you on the phone after grad recruitment, I knew you were the person I wanted to work with in graduate school. With your help, I mastered my budding knowledge of measuring cardiovascular physiology in the lab and how to clean that data of electrical artifacts. More important though, I learned how essential it is to celebrate even the small accomplishments (like finishing data cleaning, hahaha) when they arrive because academia is filled with too many rejections! I have also learned a lot from you about critiquing research methods and analyses in psychophysiological research, a skill that I think greatly enhanced my success on the post-doc market this year.

Belinda, I am so grateful for the gentle and gradual way we began working together—from simply attending your lab meetings to working on a paper together (that has been accepted as an R&R to CDEMP!). My time in your lab and the work we’ve done together so far was truly formulative in my thinking for my dissertation. I honestly feel so lucky to call you one of my mentors—your eloquent and impactful use of words is something I can only aspire to have one day. With both you and Sarah’s help, I was able to braid my initial research interests together into this dissertation.

Michelle, I am so happy that we had the opportunity to work together on this project! I really appreciate your willingness to continue advising me and working on this collaborative project together after everything that happened this past year. Knowing my dissertation results, it seems we will have several papers that we can write up together soon and I am excited to continue this work with you!

An entire hoard of amazing undergraduate/post-baccalaureate research assistants helped make this dissertation possible. Thank you to Chris Gomez, Robert Twidwell, Elinor Flynn, and Emily Wong for helping out in the development phase of this study. Thank you to Susan Koh, Diana Dominguez-Ortega, Aislinn Beam, Vida Pourmand, and Ashley Griffith for helping me to manage the day-to-day aspects of running this experimental study. Your conscientiousness and organization helped the study to run smoothly. And of course, all of the people mentioned in this paragraph also helped to train the following research assistants in running the study protocol and cleaning the cardiovascular data for this study: Genesis Imani Sanchez, Genesis Ruth Sanchez, Aadil Khan, Alissa Kato, Amber Obenshain, Audrey Lopez-Valdez, Dominique Stevens, Gabby Hernandez, Gina Philbrick, Itzel Garcia, Mary Lamons, Nicolas Nghi Do, Tiffany Szeto, Titus Abraham, Eleanor Meriwether, Sanika Joshi, Marixa Maldonado, and Eduardo Rosales. Thank you all for the countless hours you put in into making this dissertation possible! Also, I need to make a special shout out to one of my first ever study managers, soon-to-be Doctor Lady
Jacquelyn Shader! You helped to solidify a standard for conduct and responsibility in the lab when you helped manage the PA-PAIN study, thank you for being awesome.

“If we cling to belief in God, we cannot likewise have faith, since faith is not clinging but letting go” (Aldous Huxley, The Divine Within, p. 106). My journey so far has definitely tested my faith but I have a number of women to thank for helping me learn how to practice faith in myself and letting go. Carole, I have grown so much from your support and guidance, thank you for pushing me to dig deep during our work together. Jennie, Celina, Nichole, Nicolette, Diane, and Lisbeth, your amazing yogi vibes and teaching skills at YogaWorks helped me establish a yoga practice that I hope to bring with me on the East Coast. During the most difficult times in graduate school, your yoga classes were there to remind me to stay present in the moment and focus on the things I can actually do to reduce my stress, thank you!

Despite moving to California without knowing anyone, I have had the incredible fortune of making some lifelong friends at UCI. To my cohort, I think that our magical rule about not talking about research while hanging out helped to spur the inception of the “pussywillow” along with the special bond we formed with one another our first year. Even as we finished our coursework together, we still made a point to see each other at “nerd prom” and to schedule in cohort happy hours so that we could all stay connected in the sometimes-isolating environment of academia.

Outside my cohort, I have made friends with many intelligent and thoughtful people who I am excited to stay connected with in the future. My labmates- Marie, Johnny, Desi, Brooke, and Emily- grad school truly would not have been the same with you all right in there through it all with me! Johnny, thank you for always being there to remind me about the benefits of going into nature, our camping trip at Joshua Tree will always be a fond California memory. Desi, I admire your strength and drive to pursue science and your commitment to finding fun ways to eat healthy, like having vegetable parties! Brooke, your brain is amazing, I love talking with you about theories and the intricacies of them and am so glad we made friends during your short stint in grad school, haha. Emily, thank you for being so welcoming and engaging from the first time I met you at interview weekend, it really meant a lot to be able to come to you for advice about navigating the first year of grad school and beyond.

Marie, the “Mar” to my “anda”, my undercover friend, my birthday buddy, where do I even begin? It took us a while to solidify our friendship foundation but one fateful Valentine’s Day together set that foundation in stone! You introduced me to all of the great Californian activities - doing yoga and tasting wine (I’m sure there’s more, but let’s focus on the important things). Thank you for being my “no” person, for anticipating when I need support (since I don’t like asking for it), and for understanding my need for “I” time. I cannot wait to take over the East Coast with you and Nicky!

Nicky “booby” Jones, we did it! If only our past selves could see us now! Thank you for always being there when I have a stats question or when I just need to vent. Irvine won’t be the same without us to start shenanigans or spill tea. We’ll have to find a replacement on the East Coast. I’m so excited to see where this next chapter in our lives takes us and I hope that one day an
Acevedo, Jones publication can come into fruition. Also, you introduced me to our Sociology friends! Rodolfo, we don’t need words *hugs*. Sara, my friend! I’m so happy that we were able to become so close recently. I look forward to our future collaboration(s) and grant applications!

I likely would have never even applied to graduate school if it weren’t for my mentors at Alverno College. Kris, it was your research methods class and your subsequent invitation to take the applied research course that made me realize how much I enjoyed psychological science.

Paul, while taking your Physiological Psychology class, I became really interested in the topic and set up a meeting to talk more about career options in psychology. It was at that meeting that I first heard about the field of Health Psychology and now that is what my degree is in! So, thank you, Kris and Paul for illuminating my career path, it was pretty hard to see it at first without the light you ignited in me. Carole, Donna, and Pat, I still remember the multiple courses I took with each of you at Alverno and feel incredibly fortunate to have learned from you all during my time there. Meg, thank you for being such an amazing support system while I was at Alverno.

Finally, thank you to my family—both immediate and extended—for your support and encouragement throughout my lifetime of educating myself. In many ways, you have all taught me early on to put my education first and this has definitely paid off. Mama, I love you so much in the whole wide world! Thank you for always encouraging me and for starting off my process of applying to college. Daddy, I love you! Thank you for being a constant in my life, right down to the voicemail you leave every time I miss your call, “Hey Manda, it’s me, your father, give me a call when you got time, alright? Bye”. I know you wanted to come out to California for my graduation, but I know you’ll be there in spirit! I’ll pour one out in your honor that day too. I’m just glad that we get to celebrate together in Wisconsin. Aunt Mary, thank you for always asking me about and encouraging me to pursue my interests in school. And thank you for always making a point to celebrate my academic accomplishments, it really means a lot. Kayla, thank you for being the best sister ever and for giving me the cutest nephew and niece that an aunt (and Godmother!) could ask for. I love my sister, yes I do! Yaba-daba-doo, thank you for helping organize my graduation party and stuff. Thank you to Angel’s I-III (and Mia, Sophia, and Jenny) for hosting me for Thanksgiving and Christmas in Arizona, that year in particular it was nice to be surrounded by family over the holidays after a couple of years of not being home for the holidays. Herman (and Gale), your favorite niece has graduated with her PhD!! Minnie, thank you for all the work you put into our family, love you. Carl and Linda, Paul and Debbie, thank you always being there, for helping us move when we needed it, and for hosting our holiday family gatherings. To all of my cousins on my mom’s side: Eric, Ryan, Kelly, Adam, Jenna, and Ellie; and to all of my cousins on my dad’s side (and to all of their kids): Angel II, Iris, Adam, Stefanie, Carlitos, and Anyssa; I love you all and wish the best for each of you!

There has been a song that has come up often during key milestones in my graduate career. It came up on Pandora or Spotify while I was analyzing/writing up my second-year project, it came on the morning of the first day of my comprehensive exam and the morning of the last day of the exam, it came on throughout writing my dissertation proposal, and it came on again a lot while writing up this dissertation. This song is called, “End of the Road” by the R&B group, Boyz II
Men, and it is about the end of a relationship. Now that I’ve come to the end of this academic road, I understand how bittersweet this moment also is.

“Although we’ve come/ To the end of the road/ Still I can’t let go/ It’s unnatural/ You belong to me/ I belong to you”.

CURRICULUM VITAE
AMANDA M. ACEVEDO

RESEARCH INTERESTS
Biopsychosocial factors that confer resilience in the experience of stress; how culture influences the experience of emotion; how culture, emotion, and discrimination can impact experience of stress and physical health.

EDUCATION

Doctoral Candidate 2019
University of California, Irvine, Irvine, CA

Dissertation Title: Biopsychosocial factors that shape the acute physical pain experience: The role of simpatía

Honors Bachelor of Arts Degree, Psychology 2012
Alverno College, Milwaukee, WI

Senior Thesis: How behavioral economics can close the gap between cognition and action in health interventions

FELLOWSHIPS

Association of American University Women Dissertation Fellowship, $20,000 2018
Association of American University Women

Graduate Dean’s Dissertation Fellowship, $6,000 2018
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President’s Dissertation Year Fellowship, Honorable Mention, $1,000 2018
University of California, Irvine

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**Graduate Opportunity Fellowship, $17,000**  
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**Post-baccalaureate Fellowship, $27,000**  
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**Graduate Student Mentoring Award, $200**  
University of California, Irvine  
2017, 2016, & 2014

**Citation Poster Award**  
American Psychosomatic Society  
2017

**Minority Travel Grant, $500**  
American Psychosomatic Society  
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**Association of Graduate Students Travel Award, $400**  
University of California, Irvine  
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**Psi Chi Honor Society**  
Alverno College  
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**PUBLICATIONS**


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Wang, D., Mather, M., **Acevedo, A.M.**, & Gruenewald, T.L. (in preparation). Activating the giver within: Does writing about providing support for others attenuate physiological responses to stress?

**PROFESSIONAL PRESENTATIONS** (°Denotes an undergraduate or post-baccalaureate research assistant)

---


RESEARCH EXPERIENCE

Graduate Student Researcher
University of California, Irvine
Supervisors: Sarah Pressman, Ph.D. & Belinda Campos, Ph.D.

Duties:
- Developing study materials and training about 40 Research Assistants for two separate multi-year psychophysiological experiments
- Training Research Assistants to use cardiac physiology and blood pressure equipment; teaching them about cardiac physiology and how to use physiological data cleaning software

Post-baccalaureate Fellow
Post-baccalaureate Research and Education Program
University of New Mexico, Albuquerque
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Duties:
- Reviewed relevant literature regarding diagnosis, treatment, and electroencephalography data for individuals with attention deficit hyperactivity disorder (ADHD)
- Developed protocol and IRB proposal for study on attention and a rhythm and timing therapy in students with ADHD

Research Assistant
Research Center for Women and Girls
Alverno College in Milwaukee, WI
Supervisor: Kris Vasquez, Ph.D.

Duties:
- Reviewed government and industry data to analyze the status of women in Wisconsin in 2012 with regard to health and legislative issues
- Presented strategies for organizing literature to peers

Research Assistant Intern
Stress and Coping Psychophysiology Lab
University of Wisconsin, Milwaukee
Supervisor: Raymond Fleming, Ph.D.
Duties:
- Reviewed relevant literature regarding heart rate variability and relaxation
- Collected data for a relaxation study using electrocardiograph (ECG) equipment; suggested troubleshooting techniques for ECG equipment
- Trained two research assistants to run participants through the study

Research Assistant Intern 2011
Summer Research Opportunity Program (SROP)
University of Wisconsin, Madison
Supervisor: Morton Ann Gernsbacher, Ph.D.
  Duties:
  - Reviewed literature and created materials for a computerized version of vocabulary and grammar tests
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TEACHING EXPERIENCE

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Guest Lectures:

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ADDITIONAL TRAINING
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Mentoring Excellence Program Certificate 2016
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University of California Health Consortium Workshop 2016
  Workshop covered the replication crisis, open science frameworks, grant writing, and applying to faculty positions
University of California Health Consortium Workshop 2015
- Workshop covered missing data, growth curve modeling, and biological assessment
Mindware Technologies Heart Rate Variability and Impedance Cardiography Seminars 2014
- Seminars covered how to collect and analyze cardiovascular physiology data

**PROFESSIONAL SERVICE**

| Association of American University Women’s “Tech Trek” Science Camp Mentor | 2017 & 2016 |
| Summer Research Program Graduate Student Mentor | 2017 & 2016 |
| Psychology Student Association Graduate Student Panel | 2017 |
| Graduate Student Mentor for First-Year Graduate Students | 2016-2017 |
| Developed YouTube Heart Rate Variability Measurement, Stress, and Cardiovascular Data Cleaning Video Tutorial - [https://youtu.be/tFC1KTdLPJ4](https://youtu.be/tFC1KTdLPJ4) | 2016 |
| Graduate Student Panel for Post-Baccalaureate Students | 2016 & 2015 |
| Salivary Bioscience Faculty Search Committee Member | 2016 |
| Hosted Interactive “Psychology Ice Bucket Challenge” Booth at Festival of Discovery | 2015 |
| Graduate Student Panel Member for Latino/a Student Psychological Association | 2015 |
ABSTRACT OF THE DISSERTATION

Biopsychosocial Factors That Shape the Acute Physical Pain Experience: The Role of Simpatía

Amanda M. Acevedo

Doctor of Philosophy in Psychological Science

University of California, Irvine, 2019

Associate Professor Sarah D. Pressman & Associate Professor Belinda Campos, Co-chairs

How do cultural values influence individual responses to stress in the context of experimentally-induced pain? A substantial literature has found ethnic differences in pain whereby ethnic minority groups (e.g., Latinos) report experiencing more pain and less willingness to endure pain during standardized pain tasks in the laboratory setting. However, little is known about the role Latino culture plays in the context of this type of pain. The goals of this dissertation were to examine simpatía, a Latino cultural value emphasizing reduced negative expressions during negative interactions and increased positive expressions during positive interactions, in the context of experimentally-induced pain. Specifically, this dissertation examined 1) whether simpatía was associated with pain (measured via self-report, tolerance, and physiological response) and 2) whether state emotions, emotional expressions, or vagal tone explained the association of simpatía with pain in Latinos. Additionally, this dissertation explored the role ethnic background played in this context as well. To induce pain, a standardized cold pressor task was used with water held at five degrees Celsius. Results indicated that, contrary to hypotheses linking simpatía to improved pain through its association with positive emotion, simpatía was instead associated with worse pain. Specifically, Latinos
high in simpatía self-reported greater pain, were more likely to withdraw early from pain, and exhibited a flatter heart rate recovery slope compared with Latinos low in simpatía. However, no hypothesized mediator (i.e. state emotion, emotion expressions, vagal tone) significantly explained the simpatía-pain association and this association did not hold in European Americans. Taken together, the findings of this dissertation provide evidence for the importance of context in examining the influence of cultural values on the experience of acute physical pain.
Introduction

Millions of Americans suffer from pain each year (Institute of Medicine, 2011) and some groups suffer more than others. Counter to common stereotypes in the medical field and in society more broadly, studies examining the role of ethnicity in pain commonly find that ethnic minorities in the United States (e.g., Latino Americans, African Americans) report experiencing more pain in response to the same pain stimulus compared with European Americans (Rahim-Williams, Riley, Williams, & Fillingim, 2012; Rahim-Williams et al., 2007). Experimental studies examine this phenomenon by inducing pain using the same stimulus across participants and monitoring responses in a controlled environment to understand factors that may explain ethnic differences in pain. A substantial literature focuses on negative factors that exacerbate ethnic differences in pain (e.g., hypervigilance, perceived discrimination), however, there has been a lack of experimental work that examines positive factors that may reduce such differences in pain. As Latinos are one of the fastest growing ethnic minority groups in the United States (Brown, 2014) yet represent an understudied sample within the pain literature (Rahim-Williams et al., 2007), it is important to extend the literature to this group and examine cultural factors that may attenuate pain.

Latino cultural values may be an important factor to consider in the context of pain because when culture is internalized, it is associated with future behaviors such as greater motivation in academics (Fuligni, 2001), fewer externalizing behaviors (Gonzales et al., 2008), and greater positive emotion expressions (i.e. laughing, smiling) during naturalistic social interactions (Holloway, Waldrip, & Ickes, 2009). One cultural factor relevant to Latinos that may influence emotional and physiological pain processes is simpatía. Simpatía is a common cultural script among Latinos and is characterized by an avoidance of interpersonal conflict and the
tendency to strive for interpersonal harmony through an emphasis on positive behaviors that create and sustain positive interactions while at the same time de-emphasizing negative behaviors in social interactions (Triandis, 1989; Triandis, Marin, Hui, Lisansky, & Ottati, 1984). Simpatía has never been studied within the context of acute pain, but it seems plausible that adherence to this cultural script may influence Latinos’ responses to painful stimuli, for better or worse. As this dissertation will review, there are a number of ways in which simpatía might be associated with pain by altering biopsychosocial factors that are relevant to the experience of pain.

This dissertation aims to integrate literatures on biopsychosocial processes known to influence pain with the literature on the cultural construct of simpatía. Pain is a multifaceted experience that evokes multiple responses, and as such, it will be explored accordingly through self-report, behaviors, and cardiovascular responses. The following research questions will be examined: what is the association of simpatía with self-reported pain, pain tolerance (i.e. ability to withstand pain), and cardiovascular responses to pain? What biopsychosocial factors mediate these associations? To explain the rationale for these research questions, the next sections of this dissertation are organized as follows. First, I discuss theories of pain processing and how acute pain is measured (i.e., self-reports, behavior, and physiological response). Second, I highlight the lack of research on Latino samples within the research literature on ethnic differences in pain sensitivity. Third, I discuss simpatía and how it might impact factors associated with pain, namely, state emotion, emotional expressiveness, and vagal tone.

Background and Theoretical Context

Acute Physical Pain
Pain is often categorized as either acute or chronic, with acute pain lasting less than three months and chronic pain lasting more than three months (IASP, 1994). The International Association for the Study of Pain defines physical pain as, “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (1994). There are many external and contextual factors that cause wide variation in naturalistic pain responses that might be better controlled in an experimental environment, including the location of the pain, the context of the pain (e.g., while playing a high-contact sports game, while walking down the street), and other people present when pain occurs. Thus, to better understand the wide individual variation in pain experience in a controlled environment, this dissertation is focused on acute, experimentally-induced pain. Experimental pain is important to assess because of emerging evidence that acute pain is connected to important long-term outcomes. For example, sensitivity to acute pain (operationalized as increased self-reported pain and decreased tolerance for pain) is predictive of greater risk for developing persistent pain after a surgery (Werner, Mjöbo, Nielsen, & Rudin, 2010) and developing future chronic pain conditions (Nielsen, Staud, & Price, 2009).

**Theories of pain processing.** The body does not passively process pain when the sensation occurs because pain does not just have a sensory component; it also has an affective component (e.g., Rainville, 2002). While there are many theories of pain, most of them only have dealt with the sensory component of pain (for a review, see Moayedi & Davis, 2013) and are largely criticized for lacking precision. The gate control theory of pain (Melzack & Wall, 1965) is considered the most widely accepted theory of pain to this day. This theory posits that there is a gate-like mechanism in the spinal cord and, when it is open, pain is perceived. Rubbing the affected area, distraction, and descending information from the brain can inhibit whether or
how much pain is felt by the individual (for a review see Millan, 2002). Importantly, this theory suggests there are a number of psychological factors (e.g., expectations, attention, emotion) that can have an influence on the perception of pain (Melzack, 1999).

Bates (1987) integrated the gate control theory of pain with theories on social learning and social comparison processes to create the biocultural model of pain. This theory posits that cultural factors like attitudes, beliefs, and norms about pain have an influence on emotional expressions (verbal and nonverbal) of pain and on affective processing of pain at the neurological and biological level (Bates, 1987; Bates, 1993; Rahim-Williams et al., 2007). These cultural factors are proposed to contribute to information sent to the pain gate and can inhibit or exacerbate the experience of pain. The biocultural model of pain was proposed in order to explain racial/ethnic differences in pain sensitivity. This model is particularly relevant to the current study because I examined how a cultural construct (simpatía) influences biopsychosocial states that may alter the affective processing of pain and emotional expressions during pain which may initiate descending and ascending inhibitory influences (thus closing the pain gate), respectively.

**How is acute pain measured?** To best understand how individual differences impact responses to pain, experimentally induced pain can be measured in a number of ways, including via self-report, behavior, and physiological responses. Most commonly, participants are asked to self-report how much pain they feel. Although this is a common assessment technique, Mexican Americans high in simpatía tend to exhibit extreme response styles on surveys (Davis, Resnicow, & Couper, 2011). Therefore, in order to fully understand ethnic differences in pain and whether they are due to bias in self-reports, it is necessary to measure pain in other ways besides self-

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1 “Pain response” or “responses to pain” will be used as umbrella terms that capture all of the pain outcomes being assessed.
report, including physiological responses. Specifically, differences in self-reported pain may be due to differences in pain sensation or the psychological response to pain. Alternatively, if findings are consistent across different measures of pain, then this would suggest potential ethnic differences in the socialized response to pain.

Behavioral measures of pain include pain tolerance. Pain tolerance is measured as the amount of time a participant endures pain (Harris & Rollman, 1983). Pain tolerance can be influenced by a number of factors like emotion (e.g., Rhudy & Meagher, 2001) and it represents the one measure of pain that has been predictive of greater risk for developing persistent pain after a surgery (Werner et al., 2010) and developing future chronic pain conditions (Nielsen et al., 2009). Thus, pain tolerance is an important outcome in the study of individual differences, including ethnic differences in pain.

Finally, numerous physiological responses have also been measured during pain including neural (e.g., Salomons, Johnstone, Backonja, & Davidson, 2004), endocrine (e.g., Sharpley, Kauter, & McFarlane, 2009), and cardiovascular responses (e.g., Koenig, Jarczok, Ellis, Hillecke, & Thayer, 2013). Cardiovascular responses may be particularly important to study in the context of pain because exaggerated responses and/or prolonged recovery from acute stressors like pain predict future cardiovascular disease, morbidity, and mortality (e.g., Chida & Steptoe, 2010; Panaite, Salomon, Jin, & Rottenberg, 2015). Additionally, there are numerous interactions between the neural systems that control cardiovascular function and pain perception (Zamir & Maixner, 1986), and the interaction of these systems are especially important in the process of regulating pain (Bruehl & Chung, 2004).

Physiological responses to pain are typically assessed via blood pressure or heart rate response (e.g., Rahim-Williams et al., 2012) with the expectation that blood pressure and heart
rate both increase in response to pain and slowly decrease during recovery from pain. Reactivity to pain is defined as the change in the cardiovascular parameter from baseline to pain, while recovery from pain is defined as the change in the cardiovascular parameter from peak reactivity to a return to baseline or the end of the recovery period that immediately follows pain. One limitation of focusing on blood pressure and heart rate responses in the pain literature is that they reflect the simultaneous influence of both branches of the autonomic nervous system. This means that the autonomic mechanisms through which a certain factor might attenuate heart rate or blood pressure cannot be captured. The autonomic mechanisms also cannot be approximated by heart rate or blood pressure because the autonomic nervous system does not always act reciprocally; the two branches can also co-activate and de-activate together (e.g., Berntson, Norman, Hawkley, & Cacioppo, 2008). Therefore, increases in heart rate are not exactly indicative of an increase in sympathetic nervous system activity and decreases in heart rate are not exactly indicative of an increase in parasympathetic activity. The autonomic mechanisms are important to understand because factors that might alter pain can have different influences on each branch of the autonomic nervous system. The way such factors alter pain can improve our understanding of why there are ethnic differences in pain and how certain factors alter the pain experience, and this knowledge could potentially be applied to interventions meant to reduce experiences of pain.

The typical autonomic reactivity to pain is an increase in sympathetic (i.e. fight or flight) activity and a decrease in parasympathetic (i.e. rest and digest) activity (Koenig et al., 2013). Therefore, both consistent with and extending upon the literature, this dissertation measured heart rate response², parasympathetic response, and sympathetic response to acute physical pain. Overall, the outcomes being assessed represent a robust measure of pain (self-reports, behavior,

² When the terms cardiovascular/ heart rate/ parasympathetic/ sympathetic/ autonomic responses are used, they refer to both reactivity to and recovery from pain.
physiological response) which will help assess when and how individual differences matter during pain. Studies rarely measure all of these outcomes at once. These pain outcomes are often discussed separately in the literature and the association between outcome variables is rarely reported. One exception to this within the pain literature is the association between cardiovascular reactivity and self-reported pain. For example, greater heart rate reactivity is associated with greater self-reported pain (Loggia, Juneau, & Bushnell, 2011). Given this, the association between the outcomes of interest in this study will be investigated within each ethnic group to explore whether the associations are the same across groups.

**Ethnic Differences in Pain Sensitivity**

A meta-analysis examined studies that experimentally induced pain and found medium-to-large effects whereby ethnic minorities tend to be less tolerant of pain and tend to rate their pain as more intense or unpleasant compared with European Americans (Rahim-Williams et al., 2012). This was consistently found across different pain induction techniques including heat, ischemic, pressure, and cold pressor pain. Thermal heat pain occurs when a device is placed on the surface of the skin (usually on the arm or leg) and gradually heats up. Ischemic pain is a process whereby a blood pressure cuff is placed around the participant’s arm and inflated. Cold pressor pain occurs when an individual submerges their hand or foot into a bucket of ice-cold water. The meta-analysis reported on self-reported pain and pain tolerance because there is a surprising lack of research examining the influence of ethnic background on physiological responses to pain. Additionally, the meta-analysis noted that most of the research on this topic has focused on comparing African Americans with European Americans, but fail to address Latino American samples. The lack of research using other samples meant that the meta-analysis
could only calculate average effect sizes for the comparison between African and European Americans.

The few studies that have been done on pain sensitivity in Latino populations have found similar results to the above meta-analysis. For example, one telephone-based cross-sectional survey assessed self-reported pain in a nationally representative probability sample including European, African, and Latino American populations (Portenoy, Ugarte, Fuller, & Haas, 2004). Results suggested that while European American participants reported dealing with pain for a longer duration than the other groups, both the Latino and African American groups self-reported greater pain intensity.

Experimental work is consistent with the above findings as well. Rahim-Williams and colleagues (2007) conducted a study where they experimentally induced pain using three different pain tasks: thermal heat, ischemic pain, and cold pressor pain. This particular study measured both pain threshold (i.e. when someone first reports feeling pain) and tolerance and then calculated a pain range by subtracting each individual’s threshold time from their tolerance time. Across all pain tasks, both the African and Latino American groups had lower pain ranges, suggesting that these ethnic minority groups reached tolerance shortly after they began to feel pain compared to European Americans. This was largely due to differences in pain tolerance and not threshold. In other words, there seemed to be greater variation in how long participants were willing to endure pain (tolerance) and less variation in how long it took participants to indicate that they felt pain (threshold).

My previous work has replicated and extended these findings in a study using the cold pressor task to induce pain (Acevedo & Pressman, in prep). I examined both blood pressure response and self-reported pain as pain outcomes and found that Latinos self-reported greater
pain to the cold pressor compared with European Americans but that there were no differences in blood pressure response. As described above, blood pressure is influenced by both branches of the autonomic nervous system, so while there may not have been differences in the average change in blood pressure, this study could have missed differences in the autonomic pattern of responses in each ethnic group. Overall, the ethnic differences in pain literature has demonstrated consistent differences across ethnic background but has yet to identify factors that consistently attenuate the experience of pain for ethnic minorities.

**Biopsychosocial Factors that Shape the Acute Physical Pain Experience: The Role of Simpatía**

This section will discuss how simpatía might influence three factors that have been shown to alter acute physical pain. Notably, these factors are at the same levels of analysis as the pain outcomes outlined above: self-report, behavior, and physiology. This is important because the factors and outcomes at the same level of analysis (e.g., self-report) may be more strongly associated than factors and outcomes at different levels of analysis. If a factor is associated with outcomes across levels of analysis, this factor would be considered a robust way to alter acute physical pain. However, if a factor is not associated with outcomes at different levels of analysis, this might indicate bias in the factor or a weaker relationship between that factor and all pain outcomes. Further, just as the pain outcomes are interrelated because they occur due to the same event, these factors (self-reported state emotion, emotional expressive behavior, and physiology) are also interrelated.

**Simpatía.** Simpatía is a cultural script common in Latino culture; it is the tendency to strive for interpersonal harmony through the emphasis of positive behaviors in positive interactions and the de-emphasis of negative behaviors in negative interactions (Triandis, 1987).
An individual who is simpatico tends to smooth social interactions and make these interactions more personable by sharing in other’s feelings, being respectful, and striving for harmony by avoiding conflict (Holloway et al., 2009; Sanchez-Burks, Nisbett, & Ybarra, 2000; Triandis, 1989). Simpatía is such a strong cultural script that it influences Latino self-concepts and behavior (Holloway et al., 2009). Specifically, when Latinos answer the question “Who am I?” they respond with a greater proportion of simpatico-related words compared to European Americans. Additionally, in a naturally occurring interaction where two participants were led to believe they were waiting for the experimenter to fix a technical issue, the interaction was covertly recorded and later coded. The authors found that Latinos expressed more positive emotion (i.e. smiling and laughing) during the interaction and gazed directly at their interaction partner more frequently and for longer compared to European and African American participants. This behavior was also reciprocated by the partner of the Latino participant regardless of the partner’s ethnic background (Holloway et al., 2009). This suggests that simpatía has an influence on behavior in the context of social interactions and this is particularly true in Latino contexts. More specifically, individuals high in simpatía demonstrate high state positive emotion, express more positive emotions in positive situations, and express fewer negative emotions in negative situations. While simpatía has never been studied in the context of pain, there is reason to believe that it may influence pain through alteration in state emotion, emotional expressions, and physiology. In the next section, I will outline how simpatía might facilitate each of these factors and, in turn, be associated with pain.

**State emotion.** One pathway through which simpatía might be associated with pain is through state emotion. Specifically, simpatía may facilitate increased positive emotion states and decreased negative emotion states. Positive emotion has been shown to attenuate the negative
effects of the stress response (for a review, see Pressman & Cohen, 2005). For example, induced feelings of calm have been associated with greater parasympathetic nervous system activation and no sympathetic nervous system response to the cold pressor task (Pressman, Acevedo, Leger, & Jenkins, in prep) compared with a neutral condition. Calm may have been the most effective positive emotion induction (happiness and excitement were the other conditions) because it counteracted the known physiological response to acute experimentally-induced pain (an increase in sympathetic activity and a decrease in parasympathetic activity; Koenig et al., 2013). Therefore, one way positive emotion might reduce pain is through attenuating the physiological response to pain.

Positive emotion may also reduce self-reported pain and improve tolerance for pain (e.g., Rhudy & Meagher, 2001). According to the broaden-and-build theory, positive emotions function to broaden awareness, allowing individuals to seek out new opportunities or relationships and build their social resources (Fredrickson, 2004). In the context of pain, positive emotion might broaden the individual’s awareness and attention and/or the individual may more easily call on other social resources while experiencing pain. This, in turn, may help distract the individual from the negative feelings arising from pain and result in reduced self-reports of pain and improved tolerance for pain.

Conversely, positive and negative emotions may bias self-reports (e.g., Cohen, Alper, Doyle, Treanor, & Turner, 2006; Costa & McCrae, 1987; Watson & Pennebaker, 1989). For example, participants receiving experimental exposure to rhinovirus (i.e. common cold) who self-reported high levels of positive emotion reported fewer subjective symptoms then would have been expected given objective markers of symptoms (e.g., mucus weight) in this study (Cohen, Doyle, Turner, Alper, & Skoner, 2003). This finding was replicated in a study that
experimentally exposed individuals to either a rhinovirus or influenza virus (Cohen et al., 2006). Self-reports of high negative emotionality (i.e. neuroticism), for example, have been strongly associated with greater self-reported symptoms and health complaints but is less consistently related to objective markers of illness (Costa & McCrae, 1987; Watson & Pennebaker, 1989).

Measuring both positive and negative emotion addresses an important methodological issue when studying the influence of emotion on pain. Specifically, depending on how negative emotion is measured, it can be strongly correlated with positive emotion (Bagozzi, Wong, & Yi, 1999; Watson, Clark, & Tellegen, 1988). This calls into question whether it is the presence of positive emotion or the absence of negative emotion that drives stress-relevant effects. Thus, given that negative emotionality has been positively associated with greater self-reported physical symptoms (Costa & McCrae, 1987; Watson & Pennebaker, 1989), increased cardiovascular reactivity to stress (e.g., Suls & Wan, 1993) and with narrowing one’s attention (Fredrickson, 2004) to focus on pain, it is critical for researchers to consider both positive and negative emotion. Given that individuals high in simpatía tend to have a higher disposition towards positive emotions (Acevedo, Herrera, Shenhav, Yim, & Campos, under review), they may report feeling more state positive emotions and fewer state negative emotions. Therefore, state emotion will be considered a potential mediator of the association between simpatía and pain responses.

**Emotional expressiveness.** The context of pain can be considered a negative situation where the simpatía cultural script would dictate that negative expressions should be de-emphasized. Therefore, simpatía may be associated with decreased pain by altering the degree to which one verbally and nonverbally expresses their pain as they experience it. Given the lack of research on how simpatía might influence expressions in the context of pain, both positive and
negative behavioral expressions will be examined in this proposal. Which expression is chosen to enact simpatía in the context of pain is important given the literature reviewed below showing differential findings for these various expressions during pain.

Common emotional expressions during pain include the expression of negative emotions. For example, grimacing during pain is so common that it is used as an indicator of pain in nonverbal measurements of pain (e.g., Grunau & Craig, 1987; Sheu, Versloot, Nader, Kerr, & Craig, 2011; Worobey & Lewis, 1989). However, grimacing during pain is rarely considered as a predictor of self-reported pain, pain tolerance, or physiological recovery from pain. One study experimentally manipulated grimacing during acute pain and found reduced self-reported pain compared to a neutral facial expression condition but grimacing was also associated with increased heart rate in response to pain compared to a smiling condition (Pressman, Acevedo, Aucott, & Kraft-Feil, under review). Similarly, swearing during pain has been shown to increase pain tolerance but exacerbates physiological responses to pain compared to not swearing during pain (Stephens & Umland, 2009). This suggests that while negative verbal and nonverbal expressions of pain may lead to improved self-reports and tolerance for pain, it comes with the cost of greater physiological responses to pain. Individuals high in simpatía might enact this cultural script by not grimacing and swearing less to avoid burdening the experimenter with their negative experience. This may be beneficial because, as described above, negative emotion expression does not seem to confer benefits to all outcomes associated with pain.

Another common expression (or lack thereof) during pain is to remain stoic. Individuals high in stoicism may feel as much pain but express the pain less and avoid complaining about it more compared to someone low in stoicism (Janal, 1996). My previous work has supported this as well (Acevedo & Pressman, in prep). This study examined whether ethnic differences
(between Latino and European Americans) in self-reported pain and blood pressure response to pain could be explained by stoic attitudes as measured by the Pain Attitudes Questionnaire (Yong, Bell, Workman, & Gibson, 2003; Yong, Gibson, & Helme, 2001). I found that while stoicism was negatively associated with self-reported pain, it was positively associated with blood pressure reactivity to pain; however, neither stoicism nor blood pressure reactivity explained ethnic differences in pain. Importantly, when I examined whether remaining stoic during pain (i.e. stoic behavior) was associated with these pain outcomes, there was no association. Thus, individuals high in simpatía might enact this cultural script by remaining expressionless, calm, and/or neutral throughout the pain task in order to avoid burdening the experimenter with their negative experience; however, this lack of expression may not alter responses to pain.

Individuals high in simpatía might also enact the cultural script through the use of humor or positive facial expressions during pain. The explicit expression of humor (through jokes, laughter, or smiling) may be beneficial to pain for the same reasons self-reported positive emotion prior to pain may be beneficial. In fact, the way in which humor is induced in experimental work is the same as how positive emotion is sometimes induced in the lab: through funny films/audio. Most of the literature on humor and pain (for reviews see Martin, 2001; Matz & Brown, 1998) has shown that humor improves pain tolerance. For example, after listening to a 20-minute audio tape that either induced laughter, relaxation, boredom, or nothing, participants underwent an ischemic pain task. This study found that individuals who listened to the laughter or the relaxation audio tapes tolerated pain for significantly longer compared to those who listened to the boredom tape or did not listen to anything at all (Cogan, Cogan, Waltz, & McCue, 1987). This suggests that both laughter and relaxation prior to pain may improve pain tolerance.
However, in healthcare research, humor is studied and measured by either recording interactions with patients and coding the interactions for the use of humor or patients are interviewed about the use of humor to cope with their health issues (e.g., Blount, Sturges, & Powers, 1990; Johnson, 2002; Sala, Krupat, & Roter, 2002). For example, a sample of pediatric oncology patients undergoing a painful medical procedure naturalistically used humor and deep breathing to cope throughout the procedure (Blount et al., 1990). This suggests that people use humor as a way to cope with distress and pain in naturalistic, medical settings. Additionally, when primary care visits to a physician were recorded, one study found that high satisfaction with the visit was associated with greater use of humor by both the physician and the patient (Sala et al., 2002). As the experimental and medical research suggests, individuals may use humor as a way to cope and thereby reduce responses to pain. To my knowledge, no one has assessed whether individuals naturally use humor to cope with experimentally-induced pain. Thus, the degree to which humor is naturally used by individuals during pain (especially those high in simpatía) and whether this is associated with improved pain responses across all levels of analysis will be assessed in this dissertation.

One potentially beneficial nonverbal emotional expression is smiling during pain. Individuals high in simpatía might enact this cultural script by smiling more to avoid burdening the experimenter with their negative experience. As mentioned above, one study found that smiling was associated with lower self-reported pain compared to a neutral facial expression condition and with decreased heart rate response to pain compared to a grimacing condition (Pressman, Acevedo, Aucott, & Kraft-Feil, under review). Smiling during pain seems to be a prevalent and natural response to pain for some individuals (e.g., Kunz, Prkachin, & Lautenbacher, 2009) and may communicate that the pain is ‘not that bad’ to others. However, it
is also possible that such smiles expressed naturally during pain are “nonenjoyment” or “miserable” smiles that only involve the lifting of the cheeks and spreading of the lips (i.e., zygomaticus major activation; Ekman & Friesen, 1982). Genuine smiling (i.e. Duchenne smiles) during pain may be adaptive because, as the facial feedback hypothesis posits, the mere act of smiling may alter or induce state positive emotion (Tourangeau & Ellsworth, 1979). Additionally, because Duchenne smiles activate areas in the brain that are associated with positive emotion (Davidson, 1992) and pain has an emotional dimension (Price, 2000), certain types of smiles and positive emotion may work to reduce pain by closing the pain gate through descending inhibition.

Another way in which smiling may work to reduce responses to pain is by directly attenuating physiological arousal due to pain through eye activation (in sincere expressions of positivity) that induces a reflex that lowers heart rate. This reflex is activated when the muscles around the eye (orbicularis oculi) are compressed, which is particularly relevant to a Duchenne smile, as it activates this muscle. This reflex, called the oculocardiac reflex, stimulates the vagus nerve and results in a reduction in heart rate (Lang, Lanigan, & van der Wal, 1991). The vagus nerve is responsible for activation of the parasympathetic nervous system and works through tonic inhibition (i.e., negative feedback) of sympathetic nervous system activity (Thayer & Lane, 2000). In fact, tonic activation of the vagus nerve itself (i.e. vagal tone) may also attenuate responses to pain.

**Vagal tone.** Vagal tone refers to the variation in the time between each heartbeat when an individual is at rest (i.e., at baseline). This variation is specific to vagus nerve activity at the heart. Thayer and Lane (2009) argue that vagal tone can be used as a marker for a number of psychological and physiological dysfunctions because it is linked to emotion regulation
(Appelhans & Luecken, 2006), perceived threat (e.g., Friedman, 2007; Hill et al., 2017), inflammation (e.g., Thayer, 2009), and cardiovascular health outcomes (e.g., Thayer & Lane, 2007; Thayer & Sternberg, 2006). As pain is an emotional and a sensory experience that must be regulated in some way by the individual (with some referring to pain as a “homeostatic emotion”), one study has shown an association between higher vagal tone and lower ratings of pain (Appelhans & Luecken, 2008). Studies that examine this association are lacking (Koenig et al., 2013); therefore, this dissertation will address this gap in the literature.

Simpatía may alter vagal tone which, in turn, might be associated with reduced pain. For example, agreeableness (a construct related to simpatía) is associated with higher vagal tone (Oveis et al., 2009) and, as described above, higher vagal tone is associated with greater emotion regulation skill (Appelhans & Luecken, 2006; Tuck, Grant, Sollers, Booth, & Consedine, 2016) and lower ratings of pain (Appelhans & Luecken, 2008). Beyond agreeableness, simpatía may be associated with higher vagal tone due to the emotion regulation skill it takes to create personable social interactions, share in other’s feelings, and avoid interpersonal conflicts. Thus, vagal tone will be examined as a potential mediator of the association between simpatía and responses to pain.

Of note is the fact that vagal tone and parasympathetic reactivity to and recovery from pain are all measured by the same cardiovascular parameters. This means that this dissertation considers vagal activity to be both a potential predictor and an outcome. This is because the vagus nerve has dynamic feedback and feedforward circuits (Thayer & Lane, 2000) which contributes to both the top-down and bottom-up processing of emotions (like those arising from pain; Park & Thayer, 2014). Additionally, tonic, resting vagal activity has been associated with variability in phasic changes in vagal activity. For example, individuals with lower vagal tone
had reduced phasic vagal activity in response to a fearful face compared to individuals with higher vagal tone (Park, Vasey, Van Bavel, & Thayer, 2014). Thus, it is important to consider both tonic and phasic vagal activity in order to capture the complexity of the physiological pain response.

**The context of experimentally-induced pain.** In thinking through the factors that may link simpatía to pain outcomes in this dissertation, it is important to also consider how the context of experimentally-induced pain might violate expectations and norms relevant to valuing simpatía. Critically, the cultural value of simpatía may not only influence the individual’s own emotions, expressions, or physiology but should also inform expectations during social interactions with others. For example, if you are socialized to expect social interactions characterized by warmth and positivity and find yourself in a context where a neutral, not-warm individual asks you to endure physical pain and offers you no support while you endure it, is this situation likely to trigger the positive behaviors that have been previously linked to simpatía (smiling, laughter)? Or will this context violate the expectations regarding social interactions (as dictated by the cultural value of simpatía) and exacerbate the unpleasantness of the situation? To begin to tease out these complexities, the current study examined the role of simpatía in the potentially norm-violating context of experimentally induced pain.

**The Current Project**

This dissertation will make a number of contributions to the fields of cultural and health psychology. First, it will be the first to study simpatía in the context of pain. The experience of pain is a complex and multidimensional phenomenon and thus requires a complex project that assesses variables at multiple levels of analysis. Thus, this dissertation takes a biopsychosocial perspective that assesses the association of simpatía on biological (vagal tone), psychological...
(state emotion), and social (emotion expressions displayed) factors that are relevant to the acute pain experience. Importantly, simpatía may not have a direct influence on pain, but it might indirectly shape these biopsychosocial factors in ways that alter responses to pain. In order to understand when and how simpatía matters to the acute pain experience, I will measure pain in a number of ways (which is rarely done in the pain literature). This project will create an entirely new literature on the ways in which simpatía might contribute to variation in experiences of acute physical pain. The results of the project could illuminate factors that are associated with acute pain and may point to potential targets of intervention to improve the experience of pain in the future. Figure 1 represents the proposed pathways elaborated below. Each pain outcome is lettered and is associated with Aim #1. Each potential mediator has a letter which corresponds to the letter within Aim #2 discussed below.

**Figure 1. Summary of research aims**

Aim #1: Examine the association between simpatía and pain

A) I hypothesize that simpatía will be associated with lower self-reported pain.

B) I hypothesize that simpatía will be associated with higher behavioral tolerance for pain.
C) I hypothesize that simpatía will attenuate cardiovascular responses to pain (i.e. reduce heart rate and sympathetic response and increase parasympathetic response).

Aim #2: To explore the factors that may mediate the expected associations of simpatía with pain

A) I hypothesize that higher state positive emotion and lower state negative emotion prior to pain will mediate the expected associations of simpatía with pain.

B) I hypothesize that greater stoic expressions or greater positive emotional expressions and fewer negative emotional expressions during pain will mediate the expected associations of simpatía with pain.

C) I hypothesize that vagal tone will mediate the expected associations of simpatía with pain.

Aim #3: To examine whether ethnicity moderates any of the expected associations and found mediation pathways between simpatía and pain

A) I hypothesize that ethnicity will moderate the expected associations between simpatía and pain such that the association between simpatía and pain will be stronger in Latinos than in European Americans.

To test these hypotheses, a laboratory-based study was conducted that used the cold pressor task to induce acute physical pain.

**Method**

**Participants**

A sample of 254 undergraduate students at the University of California, Irvine who self-identified as being of either Latino or European American background, were recruited through either the Department of Psychological Science’s online research subject pool interface (SONA System) for class credit or flyers posted throughout campus for $20. Participants were excluded
from the study if they had ever been diagnosed with or currently had any of the following: a psychological disorder (e.g., depression) for which they were being treated (medication or therapy; \( n = 25 \)), cardiovascular disease (e.g., a heart condition; \( n = 16 \)), chronic pain conditions (e.g., fibromyalgia, arthritis; \( n = 19 \)), a connective tissue disease (e.g., Raynaud’s disease; \( n = 16 \)), or a condition where fainting frequently occurs (e.g., cardiovascular syncope; \( n = 12 \)).

Participants were also excluded if they took pain medication the day of the study (\( n = 11 \)) or were not fluent in English (\( n = 12 \)). In total, 39 potential participants were ineligible for the study. Additionally, out of the 215 participants who were eligible for the study, one participant did not complete the full study. Individuals who checked “White, Caucasian, European” when asked about both their racial/ethnic background and their parents racial/ethnic backgrounds were operationalized as being of European American background\(^3\) (\( n = 63 \)). Individuals who checked either “Mexican, Mexican-American, Chicano”, “Central American (e.g., Guatemala, Costa Rica)”, “South American (e.g., Chile, Columbia)” or who wrote in another description that could be considered Latino (e.g., Puerto Rican, Cuban) for both themselves and their parents were operationalized as being from Latino background\(^4\) (\( n = 123 \)).

Behavior coding was only carried out on videos where participants explicitly gave additional consent for video recording by initialing next to the item, “I consent to allow the researchers to use the video recording from this study for research purposes. Only researchers involved in this project will see these recordings and my identity will not be linked to this recording”. Out of 215 participants, 186 of them consented to video recording and 160 of those participants were Latino (\( n = 105 \)) or European American (\( n = 55 \)).

\(^3\) Hereafter referred to as “European Americans”

\(^4\) Hereafter referred to as “Latinos”
The majority of Latino participants were born in the United States \((n = 100; 82.3\%)\) and identified as female \((n = 101; 82.1\%)\). Of these participants, 71.5\% reported they were from a Mexican background \((n = 88)\), 12.2\% reported they were from a Central American background \((n = 15)\), and the rest of the sample either reported being of South American \((n = 3)\) or mixed Latino background \((n = 17)\). A majority of the participants reported speaking a non-English language at home \((n = 117; 95.1\%)\) and having both parents born outside of the United States \((n = 97; 78.9\%)\). As discussed below, participants came from various educational backgrounds. Additionally, most participants described their families’ socioeconomic status as lower middle class \((41\%; \text{skilled trade, steady employment})\), followed by upper working class \((31.1\%; \text{skilled workers or small farmers, steady employment})\), lower working class \((20.5\%; \text{unskilled workers, employed off-and-on})\), then upper middle class \((7.4\%; \text{professionals, high earned income})\). No participants identified their families as upper upper class (e.g., do not have to work for a living, inherited wealth).

The majority of European American participants were born in the United States \((n = 51; 81\%)\) and identified as female \((n = 43; 69.4\%)\). Of these participants, 60.3\% reported only speaking the English language at home \((n = 38)\) and having both parents born in the United States \((n = 36; 59\%)\). Additionally, most participants described their families’ socioeconomic status as upper middle class \((50\%)\), followed by lower middle class \((38.7\%)\), upper working class \((8.1\%)\), then lower working class \((3.2\%)\). No participants identified their families as upper upper class.

**Procedure**

Participants consented to participate in the study and then separately decided to provide consent to being videotaped throughout the study. Then, they completed a screening
questionnaire. Eligible participants had anthropometric measurements taken (waist and hip circumference) and were then brought into the experiment room where electrodes were placed on participants and both electrocardiograph (Lead II configuration) and impedance cardiography were recorded via BioLab 3.0.13. Female experimenters who either identified as Latina or European American placed electrodes on participants and instructed participants to complete tasks throughout the study. Next, participants completed baseline questionnaires on a tablet assessing characteristics such as simpatía, mood, and stress. Following this, participants completed a five-minute resting baseline period.

Next, participants completed a two-minute room temperature water task in order to get them acclimated to the procedure of the cold pressor task. Participants were asked to self-report their pain every 30 seconds on a tablet while their non-dominant hand was in the water. Following the room temperature task, the participants completed the cold pressor task. During this task, participants put their non-dominant hands in a bucket of ice water (between 4.8°C and 5.2°C) and attempted to keep their hand in the water for as long as they could (with an uninformed ceiling of four minutes). During both the room temperature water and cold pressor tasks, a female experimenter remained in the room with the participant, standing several feet behind the bucket and the participants’ non-dominant hand. After the cold pressor task, participants quietly recovered for five minutes. Upon completing additional questionnaires regarding ethnic background and demographics, participants were asked to guess study hypotheses and then debriefed.

Measures

As done throughout this dissertation, the measures that will be included in this study are organized into self-report, behavior, and physiology measures.
Self-report.

Simpatía. The HCHS/SOL Simpatía scale is a 10-item questionnaire designed to measure how important certain social interactions are (Sotomayor-Peterson, Cabeza De Baca, Figueredo, & Smith-Castro, 2013). Participants were asked, “When interacting with other people, how important is it for you [item]”. Example items include “to avoid conflict at all costs”, “to control your emotions”, and “to make others feel comfortable”. Participants responded on a 0 (not important) to 4 (extremely important) scale. In the present sample, this questionnaire had an internal consistency of $\alpha = .741$ (Latinos: $\alpha = .748$; European Americans: $\alpha = .671$). For this study, the sum of the scale was used with higher scores indicating greater value in simpatía.

Race/ethnicity. Participants were asked to identify their and their parent’s ethnicity.

Self-reported state emotion. A variation of the Profile of Mood States (POMS; McNair et al., 1971; Usala & Hertzog, 1989) was used to measure state emotion (22 items). Positive emotion items were calm, cheerful, energetic, at ease, happy, lively, pleased, full of pep, and relaxed. Negative emotion items were tense, nervous, depressed, angry, hostile, on edge, resentful, sad, tired, sluggish, sleepy, fatigued, and unhappy. Participants were asked, “Consider how you are feeling and evaluate the following items based on how accurately that feeling describes you AT THIS MOMENT.” Participants rated their state emotion on a 1 (not at all accurate) to 5 (extremely accurate) scale. In the present sample, this questionnaire had an overall internal consistency of $\alpha = .705$ (Latinos: $\alpha = .749$; European Americans: $\alpha = .641$); the positive emotion subscale had an internal consistency of $\alpha = .890$ (Latinos: $\alpha = .891$; European Americans: $\alpha = .882$); and the negative emotion subscale had an internal consistency of $\alpha = .860$ (Latinos: $\alpha = .870$; European Americans: $\alpha = .795$). For the current study, all items for the positive subscale were aggregated and all items for the negative subscale were aggregated to
represent average state positive emotion and state negative emotion, respectively. Higher scores for either subscale indicate greater endorsement of that emotion.

_Pain._ Pain experienced during both the room temperature and the cold pressor task was assessed on a numerical rating scale (Glossary, 2000) with the question: “How painful was this task for you on a scale from 1 (not painful at all) to 100 (worst pain possible)?” Including the moment participants placed their hand in the water, participants were asked to report their pain for every 30 seconds they had their hand in the water. Numerical rating scales have been used as this scale has been shown to be associated with other pain assessments (Haefeli & Elfering, 2006; Hjermstad et al., 2011; Kremer, Atkinson, & Igelzli, 1981). These ratings of pain were averaged over time so that all participants, regardless of how long they tolerated pain, had a self-reported pain value in analyses.

_Relevant covariates._ Participants self-reported their sex, age, height/weight (to calculate body mass index), and generation status. Generation status was assessed by asking participants to identify where their parents and both sets of grandparents were born. Additionally, a measure of participants’ waist and hip circumference was taken (to calculate waist to hip ratio). For more information on covariates that are especially relevant to both the independent and dependent variables in this study, see Appendix A.

_Behavior._

_Emotionally expressions._ To examine participant behaviors in response to the cold pressor task, relevant time points for behavioral coding were identified. One experimenter (M.L.) viewed all study videos and, in collaboration with the author, determined that verbal and non-verbal behavior in response to the cold pressor were typically initiated immediately after the beginning of the cold pressor task. This acute pain task could prompt participants who value simpatía to
avoid any negative feelings induced by the pain by either smiling/laughing or remaining stoic during pain. Thus, the 30 seconds after the beginning of the cold pressor task were selected for behavioral coding. Previous research has found that 30 second intervals are appropriate for coding complex social behavior (Campos, Graesch, Repetti, Bradbury, & Ochs, 2009).

A system for coding the video-recorded behavior was then developed by the author and a research assistant (M.L.) which drew from Dr. Campos’s extensive experience coding verbal and non-verbal behavior (Campos et al., 2009; Campos, Keltner, Beck, Gonzaga, & John, 2007; Campos, Schoebi, Gonzaga, & Gable, 2015; Campos et al., 2013; Gonzaga, Turner, Keltner, Campos, & Altemus, 2006). A coding team consisting of five undergraduate research coders (two self-identified Latinas, one self-identified European American, and two from other racial/ethnic backgrounds; three women and two men) who were blind to study hypotheses was formed. All coders coded 39 participants (20.9% of the participants who consented to videotaping) to establish coding reliability. Care was taken to ensure that no coder was ever responsible for coding the video of a participant they personally ran through the study or knew outside of the research setting. To learn the coding system and help the team to reach and maintain reliability, coders met weekly to practice coding and discuss their ratings. While learning the coding system, coders rated video segments that occurred outside of the designated segment time. The weekly learning meetings continued until coders reached appropriate reliability and could confidently apply the coding system and defend their use of the code definitions. At that point, weekly reliability meetings continued as a means to gather coders to share and discuss ratings to ensure that all coders continued to apply codes accurately and consistently. Upon establishing coder reliability, coders individually coded the remaining 79.1% of the participants.
Participant behaviors in the segment were first assigned an overall rating on stoicism (duration and intensity). Specifically, for each 30-second segment, coders rated how long participants remained stoic (i.e., a relaxed, seemingly expressionless face, the face in repose, with eyes open and lips closed; $\alpha = .874$) on a 0 (never) to 5 (~25 secs) Likert scale. Coders also rated how intensely participants exhibited stoicism on a 0 (stoicism absent) to 1 (stoicism present) scale ($\alpha = .714$). How long participants laughed (if at all) was coded on a 0 (never) to 5 (~25 secs) Likert scale, $\alpha = .817$. The duration of non-verbal facial expressions was rated on a 0 (never) to 5 (~25 secs) scale and the intensity of the expression was rated on a 0 (no expression) to 4 (extreme, apparent expression) scale. These nonverbal behaviors included grimaces (i.e., furrowed eyebrows, narrowed eyes, tightened lips, wrinkles around nose, upward mobility of lips, lack of body movement; duration: $\alpha = .751$; intensity: $\alpha = .900$), Duchenne smiles (i.e., wrinkle in the corner of the eyes and lips widening with movement of cheeks; upward curling at corners of mouth and lips; showing of teeth; duration: $\alpha = .767$; intensity: $\alpha = .711$), and social smiles (i.e., lips widening, no wrinkle in corner of eyes, no movement of cheeks; duration: $\alpha = .857$; intensity: $\alpha = .889$).

For this study, frequency/duration and intensity ratings for each expression were combined by taking the product of the expression frequency and expression intensity (as laughter did not have an intensity rating, the raw data was used for this variable). In this way, how often and how much participants made certain expressions were considered at once for the behaviors relevant to this study: stoicism, grimacing, Duchenne smiling, and social smiling.

Pain tolerance. The female experimenter recorded how long participants kept their hand in the cold pressor (in seconds).

Physiology.
Heart rate was recorded as beat-to-beat intervals using BioLab 3.0.13. All raw electrocardiograph (ECG) data were transferred into Mindware HRV 3.0.22 software and edited when artifacts prevented accurate heart rate calculation. R peaks were marked unless artifacts made it impossible to identify them. If this occurred, that part of the segment was removed and research assistants ensured the remaining data in that 60-second segment was at least 30 seconds of continuous data. If 30 seconds of data could not be extracted from a 60 second segment, then it was not included in analyses. Additionally, all raw ECG and impedance cardiography (IMP) data were transferred into Mindware IMP 3.0.22 software and edited in the same way as the HRV program. After this, an averaged ensemble of the ECG and IMP waveforms from each 60 second segment was created. The averaged ensemble was visually inspected for accuracy in labeling event points in the cardiovascular cycle. Once data were cleaned in both programs, several relevant measures were derived from it including heart rate, vagal tone, and parasympathetic and sympathetic reactivity to and recovery from pain.

**Vagal tone.** The root mean-squared successive differences (RMSSD) between adjacent, normal R-R intervals was measured in milliseconds and calculated for each 60-second segment of cleaned data in the HRV program. RMSSD is a valid, stable time-domain measure of the parasympathetic influence on the heart. An average of the five-minute baseline period was used as a measure of vagal tone in this study. Importantly, this variable was considered as a mediator in mediation analyses and was considered a control variable in the analyses where vagal tone was not being considered as a mediator and where parasympathetic response was the outcome variable.

**Heart rate.** Heart rate was extracted from each 60-second segment (in the cleaned data from the HRV program) during baseline, reactivity to and recovery from pain. An average of the
five-minute baseline period was used as a control variable in all analyses where heart rate response was the outcome variable. Heart rate response represented each 60-second segment of heart rate data obtained during the cold pressor task (up to four minutes) and the recovery period that immediately followed (five minutes). For the purpose of mediation analyses, heart rate reactivity slope was calculated as the maximum reactivity point for the sample subtracted from the baseline point and the heart rate recovery slope was calculated as the maximum reactivity point for the sample subtracted from the point at which the sample appeared to return to baseline following stress.

*Parasympathetic response.* Additionally, minute-by-minute measures of RMSSD data (cleaned in the HRV program) obtained during the cold pressor task and the recovery period that immediately followed was used as a measure of parasympathetic response to pain. For the purpose of mediation analyses, RMSSD reactivity slope was calculated as the maximum reactivity point for the sample subtracted from the baseline point and the RMSSD recovery slope was calculated as the maximum reactivity point for the sample subtracted from the point at which the sample appeared to return to baseline following stress.

*Sympathetic response.* Pre-ejection period (PEP) was measured in milliseconds and calculated for each 60-second segment of cleaned data in the IMP program. PEP is a valid, stable time-domain measures of the sympathetic influence on the heart. Minute-by-minute measures of PEP obtained during the cold pressor task and the recovery period was used as a measure of sympathetic cardiovascular response to pain. For the purpose of mediation analyses, PEP reactivity slope was calculated as the maximum reactivity point for the sample subtracted from the baseline point and the PEP recovery slope was calculated as the maximum reactivity point
for the sample subtracted from the point at which the sample appeared to return to baseline following stress.

**Data analysis plan.** All dependent variables were checked for skewness and kurtosis and transformed accordingly. Following appropriate transformations, the dependent variables were checked for outlying values (i.e., three standard deviations above or below the mean). Once these values were identified, a sensitivity analysis was run to determine if these values influenced the results. Additionally, correlations among simpatía, the potential mediator variables, and the outcome variables were assessed. Covariates considered for inclusion in analyses were age, sex, ethnic background, generation status, baseline physiology, socioeconomic status, waist-to-hip ratio, body mass index, and self-reported pain during the room temperature task. If any of these covariates were associated with the dependent variable of interest, they were controlled for within the analysis. Due to the different ways in which each category of dependent variable (self-report, tolerance, physiological responses) in this dissertation are measured, the analysis plan for handling each of these outcomes was different.

**Self-reported pain (Aims 1A-3A).** Specifically, self-reported pain was averaged across the timepoints so that all participants who completed at least part of the cold pressor task were included in analyses. This aggregate was a normally distributed and continuous variable so linear regression was used to examine whether simpatía was associated with pain tolerance (*Aim 1A*) after accounting for covariates. Based on a power analysis with power set at .9 and effect size set at .15 (medium effect size), at least 108 participants were required to achieve statistical power to detect an effect of simpatía on self-reported pain (Faul, Erdfelder, Buchner, & Lang, 2009; Faul, Erdfelder, Lang, & Buchner, 2007).
Next, models testing whether state emotion, emotional expressions, and vagal tone (in separate analyses) mediate the expected associations of simpatía with self-reported pain were conducted (Aim 2A). This was done to investigate the hypotheses that i) state emotion, ii) emotion expressions, or iii) vagal tone mediate the influence of simpatía on self-reported pain. These indirect effects were tested using conditional process analysis using the PROCESS macro Version 3.3 (Hayes, 2017) for SPSS. This regression-based approach to testing for mediation allows for the estimation of both direct and indirect effects in one model. To evaluate the significance of the indirect effect, bias-corrected 95% confidence intervals (CIs) for the estimated effect are provided and the indirect effect is considered statistically significant when the CIs do not include zero. All mediation tests were conducted with 10,000 bootstrapped samples. To achieve the level of power as above in the linear regression analyses (.9) and assuming small-to-medium correlations ($r$'s = .15) between simpatía, self-reported pain, and the mediating variables and standard deviations of 1 for each standardized variable, a Monte Carlo power analysis for indirect effects (Schoemann, Boulton, & Short, 2017) indicated that at least 650 participants would be needed to achieve enough statistical power to detect an effect. Thus, results of these analyses should be treated as exploratory and interpreted with caution.

Finally, the role of ethnicity was considered by adding in the European American sample to examine whether findings remained the same with this sample included and whether ethnic background moderated the influence of simpatía on self-reported pain (Aim 3A). Based on a power analysis with power set at .8 and the effect size set to medium (.25), at least 225 participants were required in these analyses to achieve statistical power to detect an effect (Faul et al., 2009, 2007)
**Pain tolerance (Aims 1B-3B).** Pain tolerance, or the number of seconds the participant endured the cold pressor task (up to 240 seconds), was a non-normally distributed variable that would not be well captured in a linear regression. To investigate the hypothesis that simpatía is associated with pain tolerance (measured as the likelihood of enduring the pain task until the 4-minute uninformed ceiling), proportional hazards discrete time survival analysis was used. This model is based on discrete time-to-event data for withdrawal from pain, with time measured in 30 second units which implies a minimum of 0 (participant withdrew from pain before the first 30 seconds) and a maximum of 8 intervals (participant did not withdraw until after the pain task ended). The predictor variable, simpatía and any covariates were assumed to have a constant effect on pain tolerance across time. In other words, discrete time survival indicators were constrained to load equally onto the latent hazard construct (e.g., Pratschke et al., 2016) and the latent hazard construct was regressed on simpatía and the relevant covariates (Aim 1B). Next, mediation models were tested by adding in paths from simpatía to the potential mediator and from the mediator to the latent hazard construct. This allowed for statistical tests of the direct and indirect paths from simpatía to pain tolerance (Aim 2B). Finally, the role of ethnicity was considered by adding in the European American sample to examine whether findings remained the same with this sample included and whether ethnic background moderated the influence of simpatía on pain tolerance (Aim 3B).

**Cardiovascular responses (Aims 1C-3C).** Cardiovascular responses were measured as minute-by-minute changes in physiology from baseline to room temperature task, cold pressor task, and recovery. To test whether heart rate, RMSSD, or PEP was significantly altered from baseline during the room temperature water task, paired samples t tests were conducted. If the room temperature task significantly altered heart rate, RMSSD, and/or PEP from baseline, then
the last minute of the room temperature task was treated as the “baseline” value because the magnitude of the cardiovascular response depends on the initial level of cardiovascular activity prior to the response (Berntson, Uchino, & Cacioppo, 1994). Following this, a manipulation check was conducted using a one-way repeated measures ANOVA to test whether cardiovascular physiology significantly changed from baseline during stress and contrasts were examined to determine the time of maximal reactivity in order to place the knot for the piecewise regression. Piecewise regression analyses were then conducted to assess the trajectories of heart rate, RMSSD, and PEP over the course of the cold pressor task. This technique allows for examining nonlinear change in time-series data where a known point of change happens (e.g., stress reactivity and stress recovery; Kim, Fay, Feuer, & Midthune, 2000; Rabe-Hesketh & Skrondal, 2012). Further, heart rate, RMSSD, and PEP [Level 1] were clustered within person [Level 2] in all analyses because within person change in cardiovascular responses over time violates the assumption of independence of residuals in ordinary least square regression analysis. Maximum likelihood tests were conducted to examine whether the potential covariates under consideration (see above) improved the model fit. To maintain parsimony, covariates that did not improve model fit were not included. Heart rate, RMSSD, and PEP were dependent variables of interest and were modeled separately. The interaction between simpatía and time was tested in each model after accounting for relevant covariates (Aim 1C). In sum, 3 separate piecewise regression models estimated the effect of simpatía and relevant covariates on: 1) HR, 2) RMSSD, and 3) PEP responses to the cold pressor task. To achieve unbiased estimates in this type of multilevel modeling, the literature recommends at least 50 samples at level two (Maas & Hox, 2004). In this case, this means at least 50 participants are required to achieve unbiased estimates of the participant standard errors.
Next, models testing whether state emotion, emotional expressions, and vagal tone (in separate analyses) mediate the expected associations between simpatía and cardiovascular reactivity and recovery slope were conducted (Aim 2C). This was done to investigate the hypotheses that i) state emotion, ii) emotion expressions, or iii) vagal tone mediate the influence of simpatía on reactivity and recovery slopes. These indirect effects were tested using a percentile bootstrap estimation approach with 10,000 samples implemented with the PROCESS macro Version 3.3 (Hayes, 2017). Again, statistical power is limited in all of the mediation analyses in this dissertation and, therefore, these exploratory results should be interpreted with caution.

Finally, the role of ethnicity was considered by adding in the European American sample to examine whether findings remained the same with this sample included and whether ethnic background moderated the influence of simpatía on cardiovascular responses to pain by re-conducting the piecewise regression models with the inclusion of the European American sample (Aim 3C). For these analyses, I ran sensitivity analyses with and without the individuals who withdrew early from pain (i.e. before the 4-minute uninformed ceiling). To achieve unbiased estimates of the standard errors for both groups of participants (i.e. Latinos and European Americans), then 50 participants in each group is required to achieve such estimates (Maas & Hox, 2004).

**Results**

Sample demographics are reported in Table 1. For clarity, results will be presented aim by aim and one dependent variable at a time.

Table 1

*Sample Demographics*

<table>
<thead>
<tr>
<th>Study Variables</th>
<th>Latino</th>
<th>European</th>
<th>Total</th>
</tr>
</thead>
</table>

34
Americans
(n = 123) | Americans
(n = 63) | Sample
(n = 186)
--- | --- | ---
Females a | 101 | 43 | 144
Born in U.S. a | 100 | 51 | 151
1st Generation a | 24 | 11 | 35
>3rd Generation a | 18 | 44 | 62
Age b | 20.3 (3.3) | 20.5 (3.4) | 20.4 (3.3)
Body Mass Index b* | 24.7 (5.3) | 24.1 (4.6) | 25.3 (5.0)
Waist: Hip b* | 0.83 (0.06) | 0.81 (0.06) | 0.82 (0.06)
Mother’s Education b* | 9.7 (4.6) | 12.2 (5.1) | 10.6 (4.9)
Father’s Education b* | 9.8 (5.0) | 12.9 (5.2) | 10.9 (5.2)

*Significant ethnic differences found; a Numbers in table are the sample size; b Numbers in table are the mean (standard deviation)

**Self-Reported Pain (A)**

On average, Latino participants rated their self-reported pain during the cold pressor as moderately painful (M = 48.22, SD = 24.86) on the 0-100 scale and these ratings did not significantly differ by gender (p = .73).
Figure 2. Average self-reported pain during cold pressor for Latinos by simpatía value.

Aim 1A: Is simpatía associated with self-reported pain? For Aim 1a, only self-reported pain during the room temperature task was positively associated with self-reported pain during the cold pressor task, \( r(122) = .421, p < .001 \). Therefore, average pain during the room temperature task was included as a covariate. A hierarchical linear regression was conducted in order to assess the influence of simpatía on self-reported cold pressor pain after controlling for self-reported pain during the room temperature task. The first model showed that average self-reported room temperature pain was significantly associated with average self-reported cold pressor pain, \( R^2 = .18, R^2_{\text{Adjusted}} = .17, F(1, 120) = 25.89, p < .001, \beta = .42, t(121) = 5.08, p < .001 \). The second model overall was significantly associated with self-reported cold pressor pain, \( R^2 = .21, R^2_{\text{Adjusted}} = .19, F(2, 119) = 15.51, p < .001 \) and this second model was significantly different from the first model, \( R^2_{\text{Change}} = .03, F_{\text{Change}}(1, 119) = 4.40, p = .038 \). This final model accounted for 20.7% of the variance in self-reported pain. Self-reported room temperature pain was again associated with self-reported cold pressor pain such that for every one unit increase in room temperature pain, there was a 1.24-point increase (\( SE = .24 \)) in self-reported cold pressor pain, \( \beta = .43, t(121) = 5.27, p < .0001 \). Contrary to hypotheses, simpatía was associated with greater self-reported cold pressor pain such that for every one unit increase in self-reported simpatía, there was a .79-point increase (\( SE = .38 \)) in self-reported cold pressor pain, \( \beta = .17, t(120) = 2.10, p = .038 \).

Aim 2A: Assessing potential mediators of the association between simpatía and self-reported pain. As in Aim 1A, average room pain was controlled for in analyses. To conceptualize the pathways being estimated in all Aim 2 analyses, see Figure 3.
Do state emotions mediate the association between simpatía and self-reported pain?

To test this, the effects of simpatía on both positive and negative state emotion, controlling for average room temperature pain, were estimated separately. The direct effects model indicated that average room temperature pain, simpatía, and positive emotion explained 22.9% of the variance in self-reported cold pressor pain, $F(3, 111) = 10.96, p < .0001$. Table 2 presents the coefficient estimates for each direct pathway in the mediation model (a: Simpatía $\rightarrow$ State Emotion; b: State Emotion $\rightarrow$ Pain; c: Simpatía $\rightarrow$ Pain) and the indirect pathway from simpatía to pain through state emotion (c'). Simpatía did not directly predict positive emotion, positive emotion did not directly predict self-reported cold pressor pain, and simpatía did not directly predict self-reported cold pressor pain. Additionally, the indirect effect of simpatía on self-reported cold pressor pain through state positive emotion was not statistically significant.

State negative emotion was tested as a mediator of the association between simpatía and self-reported cold pressor pain. The direct effects model indicated that average room temperature pain, simpatía, and negative emotion explained 20.8% of the variance in self-reported cold pressor pain, $F(3, 111) = 9.73, p < .0001$. As Table 2 shows, simpatía directly predicted negative
emotion. Contrary to hypotheses, increases in simpatía were associated with *increased* state negative emotion. State negative emotion, however, did not directly predict self-reported cold pressor pain and simpatía did not directly predict self-reported cold pressor pain. The indirect effect of simpatía on self-reported pain through state negative emotion was not statistically significant. Overall, these results do not support the hypothesis that state emotions explain the association between simpatía and self-reported pain.

Table 2

*Results for Each Pathway of the State Emotion Mediation Models Predicting Self-Reported Pain*

<table>
<thead>
<tr>
<th>Variable</th>
<th>$b$</th>
<th>$SE$</th>
<th>$t$</th>
<th>$p$</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Direct Effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a) Simpatía $\rightarrow$ State Emotion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive Emotion</td>
<td>.001</td>
<td>.013</td>
<td>.037</td>
<td>.970</td>
<td>[-.025, .026]</td>
</tr>
<tr>
<td>Negative Emotion</td>
<td>.023</td>
<td>.011</td>
<td>2.115</td>
<td>.037*</td>
<td>[.001, .044]</td>
</tr>
<tr>
<td>b) State Emotion $\rightarrow$ Pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive Emotion</td>
<td>-5.075</td>
<td>2.817</td>
<td>-1.802</td>
<td>.074*</td>
<td>[-10.657, .507]</td>
</tr>
<tr>
<td>Negative Emotion</td>
<td>1.916</td>
<td>3.444</td>
<td>.556</td>
<td>.579</td>
<td>[-4.908, 8.740]</td>
</tr>
<tr>
<td>c) Simpatía $\rightarrow$ Pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive Emotion</td>
<td>.566</td>
<td>.390</td>
<td>1.451</td>
<td>.150</td>
<td>[-.207, 1.338]</td>
</tr>
<tr>
<td>Negative Emotion</td>
<td>.519</td>
<td>.403</td>
<td>1.289</td>
<td>.200</td>
<td>[-.279, 1.317]</td>
</tr>
<tr>
<td><strong>Boot SE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>95% Boot CI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>c') Indirect Effects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive Emotion</td>
<td>-.002</td>
<td>.072</td>
<td></td>
<td></td>
<td>[-.148, .155]</td>
</tr>
<tr>
<td>Negative Emotion</td>
<td>.044</td>
<td>.092</td>
<td></td>
<td></td>
<td>[-.091, .279]</td>
</tr>
</tbody>
</table>
Note. SE = standard error. CI = confidence interval.

\[ p < .10 \quad \text{and} \quad p \leq .05. \]

**Do emotion expressions mediate the association between simpatía and self-reported pain?** Table 3 presents the means, standard deviations, and association with simpatía for each expression separately by sex. As this table shows, no Latino males in this coding sample grimaced, laughed, or Duchenne smiled during the first 30 seconds of the pain task. Additionally, all Latino males exhibited stoicism at some point during the first 30 seconds of the pain task (i.e., range does not include 0). In females, simpatía was associated with fewer Duchenne smiles, \( r (85) = -.224, p = .039 \). In males, simpatía was only marginally associated with fewer social smiles \( r (20) = -.437, p = .054 \). Due to these sex differences in expressions, sex was also controlled for in mediation analyses. To test the mediation hypothesis, the influence of simpatía on expressions made during pain (i.e., stoicism, grimace, laughter, Duchenne smiling, social smiling) were estimated separately. See Appendix B for means and standard errors of each emotion expression by gender and ethnic background.

Table 3

**Expressions Made during First 30 Seconds of Pain and Correlations with Simpatía in Latinos**

<table>
<thead>
<tr>
<th></th>
<th>Men (n=20)</th>
<th>Women (n=84)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simpatía (^a)</td>
<td>(24.47 (5.6))</td>
<td>(28.37 (5.1))</td>
</tr>
<tr>
<td>Expressions during initial pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stoicism</td>
<td>(4.53 (1.0))</td>
<td>(3.84 (1.8))</td>
</tr>
<tr>
<td>Grimace (^a)</td>
<td>n/a</td>
<td>(-.143)</td>
</tr>
</tbody>
</table>
Laughter 0.00 (0.0) n/a 0.07 (0.3) -.085
Duchenne smile a 0.00 (0.0) n/a 0.54 (1.9) -.224*
Social smile a 0.45 (1.3) -.437+ 1.76 (3.3) -.176

* p ≤ .10  * p < .05  
a Independent samples t-test revealed significant differences between men and women

Note: Fisher z test revealed no significant sex differences between correlation coefficients

Stoicism. The direct effects model indicated that average room temperature pain, simpatía, and stoicism explained 21.2% of the variance in self-reported cold pressor pain, $F (3, 101) = 9.06, p < .0001$. Table 4 presents the coefficient estimates for each direct pathway in the mediation model (a: Simpatía $\rightarrow$ Expressions; b: Expressions $\rightarrow$ Pain; c: Simpatía $\rightarrow$ Pain) and the indirect pathway from simpatía to pain through expressions ($c'$). Simpatía did not directly predict stoicism during pain and stoicism during pain did not directly predict self-reported cold pressor pain. Simpatía, however, directly predicted self-reported cold pressor pain such that greater simpatía was associated with higher self-reported pain. The indirect effect of simpatía on self-reported pain through stoicism displayed during pain, however, was not statistically significant.

Grimacing. The direct effects model indicated that average room temperature pain, sex, simpatía, and grimacing explained 21.1% of the variance in self-reported cold pressor pain, $F (4, 100) = 6.67, p = .0001$. As Table 4 shows, simpatía did not directly predict grimacing during pain and grimacing during pain did not directly predict self-reported cold pressor pain. Simpatía, however, directly predicted self-reported cold pressor pain. The indirect effect of simpatía and self-reported cold pressor pain through grimacing displayed during pain was not statistically significant.
Laughter. The direct effects model indicated that average room temperature pain, simpatía, and laughter explained 20.9% of the variance in self-reported cold pressor pain, $F(3, 101) = 8.94, p < .0001$. As Table 4 shows, simpatía did not directly predict laughter during pain and laughter during pain did not directly predict self-reported cold pressor pain. Simpatía, however, directly predicted self-reported cold pressor pain. The indirect effect of simpatía and self-reported cold pressor pain through laughter during pain was not statistically significant.

Duchenne smiles. The direct effects model indicated that average room temperature pain, sex, simpatía, and Duchenne smiling explained 21.3% of the variance in self-reported cold pressor pain, $F(4, 100) = 6.78, p = .0001$. As Table 4 shows, simpatía significantly predicted Duchenne smiles such that greater value in simpatía was associated with fewer Duchenne smiles during pain. However, Duchenne smiling did not directly predict self-reported cold pressor pain and simpatía marginally predicted self-reported cold pressor pain. The indirect effect of simpatía and self-reported cold pressor pain through Duchenne smiles during pain was not statistically significant.

Social smiles. The direct effects model indicated that average room temperature pain, sex, simpatía, and social smiling explained 22.7% of the variance in self-reported cold pressor pain, $F(4, 99) = 7.28, p < .0001$. As Table 4 shows, greater value in simpatía marginally predicted fewer social smiles during pain. Social smiling did not directly predict self-reported cold pressor pain. However, simpatía significantly predicted self-reported cold pressor pain. The indirect effect of simpatía and self-reported cold pressor pain through social smiles during pain was not statistically significant. Overall, these results do not support the hypothesis that emotions expressed during pain would explain the association between simpatía and self-reported pain.

Table 4
Results for Emotion Expression Mediation Models Predicting Self-Reported Pain

<table>
<thead>
<tr>
<th>Variables</th>
<th>$b$</th>
<th>$SE$</th>
<th>$t$</th>
<th>$p$</th>
<th>95% CI</th>
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<td></td>
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<td><strong>Simpatia $\rightarrow$ Expressions</strong></td>
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</tr>
<tr>
<td>Stoicism</td>
<td>.004</td>
<td>.030</td>
<td>.144</td>
<td>.886</td>
<td>[-.056, .064]</td>
</tr>
<tr>
<td>Grimacing</td>
<td>-.024</td>
<td>.018</td>
<td>-1.336</td>
<td>.185</td>
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<tr>
<td>Laughter</td>
<td>-.001</td>
<td>.004</td>
<td>-.357</td>
<td>.722</td>
<td>[-.009, .007]</td>
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<tr>
<td>Duchenne Smiles</td>
<td>-.062</td>
<td>.031</td>
<td>-2.043</td>
<td>.044*</td>
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<tr>
<td>Social Smiles</td>
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<td>.056</td>
<td>-1.952</td>
<td>.054*</td>
<td>[-.220, .002]</td>
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<tr>
<td>Stoicism</td>
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<td>.542</td>
<td>.589</td>
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<td>Grimacing</td>
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<td>.993</td>
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<tr>
<td>Laughter</td>
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<td>.993</td>
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<td>Duchenne Smiles</td>
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<td>-.578</td>
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<tr>
<td>Social Smiles</td>
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<td>.308</td>
<td>.759</td>
<td>[-1.174, 1.605]</td>
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<td><strong>Simpatia $\rightarrow$ Pain</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stoicism</td>
<td>.843</td>
<td>.377</td>
<td>2.237</td>
<td>.028*</td>
<td>[.096, 1.590]</td>
</tr>
<tr>
<td>Grimacing</td>
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<td>.398</td>
<td>2.028</td>
<td>.045*</td>
<td>[.018, 1.598]</td>
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<td>Laughter</td>
<td>.845</td>
<td>.377</td>
<td>2.240</td>
<td>.027*</td>
<td>[.097, 1.594]</td>
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<tr>
<td>Duchenne Smiles</td>
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<td>.402</td>
<td>1.894</td>
<td>.061*</td>
<td>[-.036, 1.560]</td>
</tr>
<tr>
<td>Social Smiles</td>
<td>.947</td>
<td>.399</td>
<td>2.374</td>
<td>.020*</td>
<td>[.155, 1.738]</td>
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<table>
<thead>
<tr>
<th>$b$</th>
<th>Boot $SE$</th>
<th>95% Boot CI</th>
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</thead>
<tbody>
<tr>
<td><strong>Indirect Effects</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

42
Stoicism  .003  .049  [-.094, .118]
Grimacing  .0004  .069  [-.068, .211]
Laughter  .0001  .021  [-.040, .051]
Duchenne Smiles  .046  .066  [-.055, .205]
Social Smiles  -.024  .084  [-.160, .185]

*Note. SE = standard error. CI = confidence interval. *p < .10  *p < .05.

Does vagal tone mediate the association between simpatía and self-reported pain?

Consistent with the literature (e.g., Thayer & Lane, 2007; Thayer et al., 2009), age was negatively associated with vagal tone (RMSSD: r (119) = -.159, p = .084; RSA: r (119) = -.213, p = .020) so was also controlled for in analyses. No other covariate that was considered was significantly associated with vagal tone. The RMSSD measure of vagal tone was significantly skewed so was natural log transformed for analyses. The effects of simpatía on RMSSD and RSA, controlling for average room temperature pain and age were estimated separately.

RMSSD. The direct effects model indicated that average room temperature pain, age, simpatía, and RMSSD explained 21.4% of the variance in self-reported cold pressor pain, F (4, 113) = 7.71, p < .0001. Table 5 presents the coefficient estimates for each direct pathway in the mediation model (a: Simpatía → Vagal Tone; b: Vagal Tone → Pain; c: Simpatía → Pain) and the indirect pathway from simpatía to pain through vagal tone (c’). Simpatía did not predict RMSSD and RMSSD did not predict self-reported cold pressor pain. However, simpatía significantly predicted self-reported cold pressor pain. The indirect effect of simpatía and self-reported cold pressor pain through RMSSD was not statistically significant.

RSA. The direct effects model indicated that average room temperature pain, age, simpatía, and RSA explained 21.5% of the variance in self-reported cold pressor pain, F (4, 113)
As Table 5 shows, simpatía did not predict RSA and RSA did not predict self-reported cold pressor pain. However, simpatía significantly predicted self-reported cold pressor pain. The indirect effect of simpatía and self-reported cold pressor pain through RSA was not statistically significant. Overall, these results do not support the hypothesis that vagal tone mediates the association between simpatía and self-reported pain.

Table 5

*Results for Vagal Tone Mediation Models Predicting Self-Reported Pain*

<table>
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<tr>
<th>Variables</th>
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<td>Simpatía $\rightarrow$ Vagal Tone</td>
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<tr>
<td>RMSSD</td>
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<td>.012</td>
<td>.991</td>
<td>[-.017, .017]</td>
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<tr>
<td>RSA</td>
<td>.006</td>
<td>.018</td>
<td>.327</td>
<td>.744</td>
<td>[-.030, .041]</td>
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<td>Vagal Tone $\rightarrow$ Pain</td>
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<tr>
<td>RMSSD</td>
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<td>4.299</td>
<td>.446</td>
<td>.657</td>
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<td>RSA</td>
<td>1.112</td>
<td>2.047</td>
<td>.543</td>
<td>.588</td>
<td>[-2.943, 5.168]</td>
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<tr>
<td>RMSSD</td>
<td>.863</td>
<td>.392</td>
<td>2.201</td>
<td>.030*</td>
<td>[.086, 1.639]</td>
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<tr>
<td>RSA</td>
<td>.856</td>
<td>.392</td>
<td>2.184</td>
<td>.031*</td>
<td>[.080, 1.633]</td>
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<table>
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<th>Boot SE</th>
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<td>RMSSD</td>
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<tr>
<td>RSA</td>
<td>-.113, .162</td>
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*Note. SE = standard error. CI = confidence interval. $^*p < .10$ $^*p < .05.$*
Aim 3A: What is the role of ethnicity in the association between simpatía and self-reported pain? In assessing covariates, significant ethnic differences in both waist to hip ratio, $t(184) = -2.17, p = .031; M_{\text{diff}} = -.020, 95\% \text{ CI } [-.037, -.002]$, and body mass index, $t(171.74) = -4.06, p = .00007; M_{\text{diff}} = -2.70, 95\% \text{ CI } [-4.02, -1.39]$ were found. Specifically, Latinos had higher waist to hip ratio ($M = 0.83, SD = .057$) and body mass index ($M = 26.24, SD = 5.44$) compared with European Americans (Waist to hip: $M = 0.81, SD = 0.06$; body mass index: $M = 23.53; SD = 3.50$). Additionally, significant ethnic differences in parental years of education were found (mother: $t(175) = 3.22, p = .002; M_{\text{diff}} = 2.45, 95\% \text{ CI } [.95, 3.95]$; father: $t(168) = 3.74, p = .0003, M_{\text{diff}} = 3.03, 95\% \text{ CI } [1.43, 4.63]$) such that parents of Latino participants had significantly fewer years of education (mother: $M = 9.72, SD = 4.63$; father: $M = 9.82, SD = 4.98$) compared with parents of European American participants (mother: $M = 12.16, SD = 5.15$; father: $M = 12.85, SD = 5.17$). Thus, for all Aim 3 analyses, waist to hip ratio, body mass index, and parental years of education were considered as covariates along with room temperature pain. Importantly, there were no ethnic differences in valuing simpatía such that Latinos ($M = 27.63, SD = 5.38$) and European Americans ($M = 26.44, SD = 4.88$) valued simpatía about the same, $t (183) = -1.46, p = .14$. See Appendix C for intercorrelations between all main study variables for Latinos and European Americans separately.

The final model included ethnicity, simpatía, the interaction between these two variables as predictors, and average parental years of education, waist-to-hip ratio, body mass index, and room temperature pain as covariates, however, the interaction was not significant, $p = .123$. Therefore, the model with all of the variables of interest independently predicting self-reported cold pressor pain was interpreted. This model significantly accounted for 22.4% of the variance in self-reported pain, $R_{\text{adj}}^2 = .196, F (6, 169) = 8.12, p < .001$. Specifically, only average room
temperature pain was significantly associated with cold pressor pain, $B = 1.23$, $SE = .21$, $\beta = .40$, $t (175) = 5.90$, $p < .001$. Ethnic background, however, was not significant ($B = 4.38$, $SE = 3.71$, $\beta = .09$, $t (175) = 1.18$, $p = .24$) and simpatía was marginally significant, $B = .61$, $SE = .32$, $\beta = .13$, $t (175) = 1.92$, $p = .056$. In order to understand whether simpatía was associated with self-reported pain in the same direction as Latinos, the file was split and Aim 1A analyses were repeated with the European American participants. In European Americans, simpatía was not associated with self-reported cold pressor pain controlling for room temperature pain, $B = -.08$, $SE = .52$, $\beta = -.02$, $t (61) = -.16$, $p = .87$.

**Pain Tolerance (B)**

**Aim 1B: Is simpatía associated with pain tolerance?** For Aim 1b, significant sex differences in pain tolerance were found, $t (32.17) = -2.15$, $p = .04$; $M_{\text{diff}} = -44.80$, 95% CI [-87.18, -2.41] such that females tolerated pain for less time ($M = 140.63$, $SD = 97.56$) compared with males ($M = 185.43$, $SD = 84.36$). Therefore, analyses of pain tolerance will include sex as a covariate. The average survival rate for the Latino sample at each time interval is represented in Figure 4 with a maximum time of 240 seconds (i.e., Time = 8).
Figure 4. Participants who remained in cold pressor task, by half-minute.

As can be seen in Figure 4, by the end of the fourth minute of the cold pressor task about half of the Latino participants remained ($n = 60$). Results of the model with simpatía as a predictor of the propensity to withdraw early from pain, controlling for sex are displayed in Figure 5. Contrary to hypotheses, simpatía was significantly associated with an increased propensity to withdraw early from pain ($p = .033$). In other words, for every one-point increase in valuing simpatía, there is a .054 standard deviation increase in the likelihood of withdrawing from pain early.

Figure 5. Latino unstandardized coefficients for the association between sex, simpatía, and the propensity to withdraw from pain early. $s_{\text{total}}$ = simpatía; $w$ = propensity to withdraw from pain. Model fit statistics: LL = -546.69, sample-adjusted BIC = 1,113.09.
Aim 2B: Assessing potential mediators of the association between simpatía and pain tolerance. Regression analyses were used to investigate the hypotheses that i) state emotion, ii) emotion expressions, or iii) vagal tone mediate the influence of simpatía on pain tolerance. These indirect effects were tested using MPlus by adding in the pathways to and from the potential mediator variables (separately) into the model seen above in Figure 5. As in Aim 1B, sex was controlled for in analyses. Results from that aim indicated that simpatía was significantly associated with pain tolerance (i.e., the direct effect is significant).

Do state emotions mediate the association between simpatía and pain tolerance? To test this, the effects of simpatía on both positive and negative state emotion, controlling for sex, were estimated separately. The direct effects model indicated simpatía explained 7.6% of the variance in pain tolerance. Table 6 presents the coefficient estimates for each direct pathway in the mediation model (a: Simpatía → State Emotion; b: State Emotion → Pain; c: Simpatía → Pain) and the indirect pathway from simpatía to pain through state emotion (c’). Simpatía did not directly predict positive emotion. However, positive emotion marginally predicted the propensity to withdraw from pain such that greater positive emotion was associated with a greater likelihood to withdraw from pain early. Simpatía was also a significant and positive predictor of the propensity to withdraw from pain early. The indirect effect of simpatía on the propensity to withdraw early from pain through state positive emotion was not statistically significant.

State negative emotion was tested as a mediator of the association between simpatía and pain tolerance. As Table 6 shows, simpatía directly predicted negative emotion. Contrary to hypotheses, however, increases in simpatía were associated with increased state negative emotion. State negative emotion, however, did not directly predict the propensity to withdraw early from pain. Again, simpatía was associated with a greater propensity to withdraw early from pain.
pain. The indirect effect of simpatía on pain tolerance through state negative emotion was not statistically significant. Overall, these results do not support the hypothesis that state emotions explain the association between simpatía and pain tolerance.

Table 6

_Results for State Emotion Mediation Models Predicting Pain Tolerance_

<table>
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<tr>
<th>Variable</th>
<th>$B$</th>
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<th>$B/SE$</th>
<th>$p$</th>
<th>95% CI</th>
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<td>Simpatía $\rightarrow$ State Emotion</td>
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<tr>
<td>Positive Emotion</td>
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<td>.013</td>
<td>-.061</td>
<td>.951</td>
<td>[-.026, .024]</td>
</tr>
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<td>Negative Emotion</td>
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<td>.010</td>
<td>2.445</td>
<td>.014*</td>
<td>[.005, .044]</td>
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</tr>
<tr>
<td>Positive Emotion</td>
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<td>.196</td>
<td>1.922</td>
<td>.055*</td>
<td>[-.008, .762]</td>
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<td>-.481</td>
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<td></td>
</tr>
<tr>
<td>Positive Emotion</td>
<td>.054</td>
<td>.025</td>
<td>2.149</td>
<td>.032*</td>
<td>[.005, .104]</td>
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<tr>
<td>Negative Emotion</td>
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<td>.026</td>
<td>2.169</td>
<td>.030*</td>
<td>[.005, .044]</td>
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<td><strong>Indirect Effects</strong></td>
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<tr>
<td>Positive Emotion</td>
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<td>.005</td>
<td>-.061</td>
<td>.952</td>
<td>[-.010, .009]</td>
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<tr>
<td>Negative Emotion</td>
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<td>.006</td>
<td>-.486</td>
<td>.627</td>
<td>[-.014, .009]</td>
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</table>

*Note. SE = standard error. CI = confidence interval.

$^+p < .10 \quad ^*p \leq .05.$
Do emotion expressions mediate the association between simpatía and pain tolerance?

As mentioned previously, Table 3 presents the means, standard deviations, and association with simpatía of each expression, separately by sex. Given the sex differences in expressions and in tolerance for pain, sex was controlled for in mediation analyses. To test the mediation hypothesis, the influence of simpatía on expressions made during pain (grimace, stoicism, laughter, Duchenne smiling, social smiling) was estimated separately.

Stoicism. The direct effects model indicated that simpatía explained 7.6% of the variance in pain tolerance. Table 7 presents the coefficient estimates for each direct pathway in the mediation model (a: Simpatía → Expressions; b: Expressions → Pain; c: Simpatía → Pain) and the indirect pathway from simpatía to pain through expressions made during pain (c’). Simpatía did not directly predict stoicism during pain. However, stoicism during pain did directly predict a decreased propensity to withdraw early from pain and simpatía directly predicted an increased propensity to withdraw from pain early. The indirect effect of simpatía on pain tolerance through stoicism displayed during pain was not statistically significant.

Grimacing. As Table 7 shows, simpatía did not directly predict grimacing during pain and grimacing did not directly predict the propensity to withdraw from pain early. Again, simpatía directly predicted an increased propensity to withdraw early from pain. The indirect effect of simpatía on pain tolerance through grimacing during pain was not statistically significant.

Laughter. As Table 7 shows, simpatía did not directly predict laughing during pain and laughing did not directly predict the propensity to withdraw from pain early. Again, simpatía directly predicted an increased propensity to withdraw early from pain. The indirect effect of simpatía on pain tolerance through laughing during pain was not statistically significant.
**Duchenne smiles.** As Table 7 shows, simpatía did not directly predict Duchenne smiling during pain. However, Duchenne smiling during pain was marginally associated with an increased propensity to withdraw from pain early. Again, simpatía also predicted an increased propensity to withdraw early from pain. The indirect effect of simpatía on pain tolerance through Duchenne smiles displayed during pain was not statistically significant.

**Social smiles.** As Table 7 shows, simpatía did not directly predict social smiling during pain and social smiles did not directly predict the propensity to withdraw from pain early. Again, simpatía directly predicted an increased propensity to withdraw early from pain. The indirect effect of simpatía on pain tolerance through social smiling during pain was not statistically significant. Overall, these results do not support the hypothesis that expressions made during pain would mediate the association between simpatía and pain tolerance.

**Table 7**

*Results for Emotion Expression Mediation Models Predicting Pain Tolerance*

<table>
<thead>
<tr>
<th>Variables</th>
<th>B</th>
<th>SE</th>
<th>B/SE</th>
<th>p</th>
<th>95% CI</th>
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<td>Direct Effects</td>
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<tr>
<td><em>Simpatía → Expressions</em></td>
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</tr>
<tr>
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<td>.034</td>
<td>.046</td>
<td>.963</td>
<td>[-.064, .067]</td>
</tr>
<tr>
<td>Grimacing</td>
<td>-.015</td>
<td>.015</td>
<td>-1.017</td>
<td>.309</td>
<td>[-.045, .014]</td>
</tr>
<tr>
<td>Laughter</td>
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<td>.003</td>
<td>-.509</td>
<td>.611</td>
<td>[.007, .004]</td>
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<td>Duchenne Smiles</td>
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<td>.044</td>
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<td>.291</td>
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<td>-1.219</td>
<td>.223</td>
<td>[-.188, .044]</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Stoicisim</td>
<td>-.242</td>
<td>.090</td>
<td>-2.691</td>
<td>.007*</td>
<td>[-.419, -.066]</td>
</tr>
</tbody>
</table>
Grimacing  .203  .139  1.456  .145  [-.070, .476]
Laughter  .433  .720  .601  .548  [-.978, 1.844]
Duchenne Smiles  .117  .069  1.684  .092+  [-.019, .253]
Social Smiles  .071  .049  1.447  .148  [-.025, .166]

Simpatía  →  Pain

Stoicism  .057  .024  2.345  .019*  [.009, .104]
Grimacing  .056  .026  2.201  .029*  [.006, .106]
Laughter  .054  .026  2.111  .035*  [.004, .104]
Duchenne Smiles  .059  .026  2.288  .022"  [.008, .110]
Social Smiles  .060  .025  2.406  .016"  [.011, .110]

Indirect Effects

Stoicism  .000  .008  -.046  .963  [-.016, .016]
Grimacing  -.003  .004  -.698  .485  [-.012, .006]
Laughter  -.001  .002  -.335  .738  [-.004, .003]
Duchenne Smiles  -.005  .007  -.733  .464  [-.020, .009]
Social Smiles  -.005  .005  -1.093  .275  [-.014, .004]

Note. SE = standard error. CI = confidence interval. +p < .10  *p < .05.

Does vagal tone mediate the association between simpatía and pain tolerance? To test this, the effects of simpatía on RMSSD and RSA, controlling for sex and age were estimated separately.

RMSSD. The direct effects model indicated that simpatía explained 7.6% of the variance in pain tolerance. Table 8 presents the coefficient estimates for each direct pathway in the
mediation model (a: Simpatía $\rightarrow$ Vagal Tone; b: Vagal Tone $\rightarrow$ Pain; c: Simpatía $\rightarrow$ Pain) and the indirect pathway from simpatía to pain through vagal tone (c’). Simpatía did not directly predict RMSSD and RMSSD did not predict the propensity to withdraw early from pain. However, simpatía did directly predict an increased propensity to withdraw early from pain. The indirect effect of simpatía on pain tolerance through RMSSD was not statistically significant.

RSA. As Table 8 shows, simpatía did not directly predict RSA and RSA did not predict the propensity to withdraw early from pain. However, simpatía did directly predict an increased propensity to withdraw early from pain. The indirect effect of simpatía on pain tolerance through RSA was not statistically significant.

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Indirect Effects

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<th>.987</th>
<th>[-.001, .001]</th>
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<tbody>
<tr>
<td>RSA</td>
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<td>.001</td>
<td>-.176</td>
<td>.860</td>
<td>[-.003, .002]</td>
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</table>

*Note. SE = standard error. CI = confidence interval. *p < .10 *p < .05.

In sum, no potential mediator (i.e. self-reported state emotion, behaviors expressed during pain, baseline physiology) significantly explained the influence of simpatía on pain tolerance.

**Aim 3B: What is the role of ethnicity in the association between simpatía and pain tolerance?** Given the ethnic differences in body mass index and parental years of education, these variables were considered as covariates along with sex. First, whether the results from Aim 1B replicated after adding in the European American sample was tested. Figure 6 displays the model fit statistics and results of this model. The results among the variables remain the same such that greater value in simpatía was associated with an increased propensity to withdraw from pain early, \( p = .008 \). In other words, for every one-point increase in valuing simpatía, there is a .059 standard deviation increase in the likelihood of withdrawing from pain early.
Figure 6. Latino and Europeans participants unstandardized coefficients for the association between sex, simpatía, and the propensity to withdraw from pain early. \( s_{\text{total}} = \) simpatía; \( w = \) propensity to withdraw from pain. Model fit statistics: \( \text{LL} = -790.08 \), sample-adjusted \( \text{BIC} = 1,604.73 \).

Second, ethnicity, body mass index, and parental years of education were added into the model and the results are displayed in Figure 7. This model indicated that both simpatía \((p = .031)\) and ethnic background \((p = .039)\) were independently and significantly associated with pain tolerance. Specifically, for every one-point increase in valuing simpatía, there is a .060 standard deviation increase in the propensity to withdraw early from pain, and identifying as Latino is associated with a .638 standard deviation increase in the propensity to withdraw early from pain.
Figure 7. Latino and European participants unstandardized coefficients for the association between simpatía and the propensity to withdraw from pain early. Analyses controlled for sex differences in simpatía and ethnic differences in parental education and BMI. Model fit statistics: LL = -804.71, sample-adjusted BIC = 1,644.45. \( s_{\text{total}} = \) simpatía; ethn\( s_r = \) ethnicity; educ\( yrs = \) parental years of education; bmi = body mass index; \( w = \) propensity to withdraw from pain early.

Adding an interaction term between simpatía and ethnicity to the model neither improved model fit nor was statistically significant.

**Cardiovascular Responses (C)**

**Aim 1C: Is simpatía associated with cardiovascular responses to acute pain?** Paired samples \( t \) tests revealed that the room temperature water task significantly altered heart rate and parasympathetic activity from baseline (heart rate: \( t (119) = 6.60, p < .0001 \); RMSSD: \( t (119) = -5.00, p < .0001 \)). The pattern for sympathetic activity changed in a similar direction, however, this change was not significant, \( t (111) = -1.34, p = .18 \). Overall, this suggested that participants became physiologically less aroused during the room temperature task compared with baseline.
(i.e. heart rate decreased, RMSSD increased, and PEP decreased). Therefore, the final minute of the room temperature task was used as the baseline value for analyses.

**Heart Rate.** Following this, manipulation checks (one-way repeated measures ANOVA) revealed a significant effect of time on heart rate with Greenhouse-Geisser correction, $F(2.38, 140.19) = 42.88, p < .0001$, partial $\eta^2 = .421$, and time did not have a linear relationship with heart rate, $F(1, 59) = 1.03, p = .32$, but had a quadratic relationship with heart rate from the room temperature task to the end of the cold pressor task, $F(1, 59) = 62.94, p < .0001$, partial $\eta^2 = .516$. Pairwise comparisons of heart rate at each time point with Bonferroni correction revealed the largest significant mean difference from the room temperature task was the first minute of the cold pressor task, $M_{\text{diff}} = -9.12$, 95% CI [-11.37, -6.86], $p < .0001$. See Figure 8 for the average heart rate response to the cold pressor task for the Latino sample who tolerated pain for the entire 4-minute task (repeated measures ANOVA uses listwise deletion, however, the average response for all Latinos is in Appendix D).

![Heart Rate Response to Cold Pressor for Latinos Who Endured Pain for 4 Minutes](image)

*Figure 8.* Heart rate response to the cold pressor task ($n = 60$).
Over time, heart rate appears to increase (i.e. react) in response to the cold pressor during the first minute of the task. Heart rate decreases significantly from the initial response by the third minute of the cold pressor task, $M_{\text{diff}} = 5.05$, 95% CI [2.55, 7.54], $p < .0001$, and returns to baseline values by the second minute of recovery, $M_{\text{diff}} = -0.22$, 95% CI [-2.62, 2.18], $p > .05$.

Therefore, the knot for the piecewise regression was placed at the first minute of the cold pressor task. In this way, the minute-by-minute heart rate from room temperature task to the first minute of the cold pressor task represented the reactivity slope and minute-by-minute heart rate from the first minute of the cold pressor task to the final minute of the cold pressor task represented the recovery slope. Heart rate was not normally distributed so was log-transformed. Even after this transformation, residual errors of the final model were not normally distributed, therefore, robust standard errors were used to adjust for this assumption violation.

Maximum likelihood tests were conducted to determine whether potential covariates improved model fit prior to testing research questions. For heart rate, maternal years of education significantly improved model fit so was included as a covariate in analyses. Additionally, a model with a random intercept for participants and a random slope for linear time best fit the data. As seen in Table 9, using piecewise regression to test the hypothesis that simpatía would attenuate the heart rate response to pain revealed a significant interaction between simpatía and recovery.

Table 9
Multilevel Between Person Effects of the Interaction of Simpatía with Heart Rate Recovery

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<th>Variables</th>
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<tr>
<td>Intercept</td>
<td>4.222 (0.026) ***</td>
<td>[4.171, 4.274]</td>
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</table>
Reactivity 0.114 (0.009)*** [0.096, 0.133]
Recovery -0.021 (0.002)*** [-0.025, -0.017]
Simpatía 0.002 (0.003) [-0.003, 0.008]
Reactivity X Simpatía -0.001 (0.002) [-0.004, 0.002]
Recovery X Simpatía 0.001 (0.0003)** [0.0003, 0.002]
Maternal Education -0.001 (0.002)** [-0.009, -0.002]

Random intercept ($a_0^2$) 0.011 (0.002) [0.008, 0.015]
Random linear slope ($a_1^2$) 0.00003 (0.00004) [0.000003, 0.00003]
Residual variance 0.003 (0.0004) [0.003, 0.004]

Note. Based on 111 participants with 505 longitudinal records. RSE = robust standard errors; CI = confidence interval. *p < .10 *p ≤ .05. **p < .01. ***p < .001.

Figure 9 visualizes the interaction between simpatía and the recovery period, $z = 2.60$, $p = .009$. Non-transformed values of heart rate are plotted in the figure despite all analyses being conducted using log-transformed values for ease of comparison across other studies. Contrary to hypotheses, greater simpatía was associated with decreased recovery (i.e. a flatter slope) compared with Latinos who valued simpatía less. Running this model only including Latino participants who tolerated pain for 4 minutes (i.e. the maximum time for the cold pressor task in this study; $n = 60$) yielded the same pattern of results.
**Figure 9.** Simpatía by heart rate recovery interaction.

**RMSSD.** Manipulation checks (one-way repeated measures ANOVA) revealed a significant effect of time on RMSSD with Greenhouse-Geisser correction, $F(2.66, 156.99) = 14.72, p < .0001$, partial $\eta^2 = .20$, and time did not have a linear relationship with RMSSD, $F(1, 59) = 2.35, p = .13$, but had a quadratic relationship with RMSSD from the end of the room temperature task to the end of the cold pressor task, $F(1, 59) = 25.76, p < .0001$, partial $\eta^2 = .304$. Pairwise comparisons of RMSSD at each time point with Bonferroni correction revealed the largest significant mean difference from the room temperature task was the first minute of the cold pressor task, $M_{\text{diff}} = 16.09$, 95% CI [6.64, 25.55], $p < .0001$. See Figure 10 for the average RMSSD response to the cold pressor task for the Latino sample who tolerated pain for the entire
4-minute task (repeated measures ANOVA uses listwise deletion, however, the average response for all Latinos is in Appendix E).

Figure 10. RMSSD response to the cold pressor task (n = 60).

Over time, RMSSD appears to decrease (i.e. react) in response to the cold pressor during the first minute of the task. RMSSD increases significantly from the initial response (CP 1) by the final minute of the cold pressor task, $M_{\text{diff}} = -8.94$, 95% CI [-15.91, -1.98], $p = .004$, and returns to baseline values by the final minute of the cold pressor task, $M_{\text{diff}} = 7.15$, 95% CI [-0.37, 14.68], $p = .074$. Therefore, the knot for the piecewise regression was placed at the first minute of the cold pressor task. In this way, the minute-by-minute heart rate from room temperature task to the first minute of the cold pressor task represented the reactivity slope and minute-by-minute heart rate from the first minute of the cold pressor task to the final minute of the cold pressor task represented the recovery slope. RMSSD was not normally distributed so was log-transformed. The assumption that residual errors are normally distributed was violated so robust standard errors were used to adjust for this violation.
Maximum likelihood tests were conducted to determine whether potential covariates improved model fit prior to testing research questions. For RMSSD, no covariate improved model fit. Additionally, a model with a random intercept for participants best fit the data. Using piecewise regression, the hypothesis that simpatía would attenuate the RMSSD response to pain was not supported (see Figure 11). However, the pattern of results appears to be contrary to hypotheses such that greater simpatía was associated with decreased recovery (i.e. a flatter slope) compared with Latinos who valued simpatía less. Running this model only including Latino participants who tolerated pain for 4 minutes (i.e. the maximum time for the cold pressor task in this study) yielded the same pattern of results.

![Predictive Margins with 95% CIs](image)

**Figure 11.** Predicted parasympathetic response to the cold pressor task.
**PEP.** Manipulation checks (one-way repeated measures ANOVA) revealed a marginally significant effect of time on PEP with Greenhouse-Geisser correction, $F(2.95, 156.55) = 1.79, p = .152$, partial $\eta^2 = .03$, and time did not have a linear relationship with PEP, $F(1, 53) = 1.10, p = .30$, but had a marginally quadratic relationship with PEP from the end of the room temperature task to the end of the cold pressor task, $F(1, 53) = 3.37, p = .072$, partial $\eta^2 = .06$. Pairwise comparisons of PEP at each time point with Bonferroni correction revealed the largest mean difference from the room temperature task was the first minute of the cold pressor task ($M_{\text{diff}} = 2.65, 95\% \ CI [-1.30, 6.59], p = .546$), however, this difference was not significant. See Figure 12 for the average PEP response to the cold pressor task for the Latino sample who tolerated pain for the entire 4-minute task (repeated measures ANOVA uses listwise deletion, however, the average response for all Latinos is in Appendix F).

![PEP Response to Cold Pressor Task for Latinos Who Endured Pain for Four Minutes](image)

**Figure 12.** PEP response to the cold pressor task.

Over time, PEP appears to decrease (i.e. react) in response to the cold pressor during the first minute of the task. PEP increases from the initial response (CP 1) by the final minute of the cold pressor task, $M_{\text{diff}} = -1.76, 95\% \ CI [-6.24, 2.73]$, and approaches baseline values by the final
minute of recovery, $M_{\text{diff}} = .283$, 95% CI [-3.39, 3.96]. Therefore, the knot for the piecewise regression was placed at the first minute of the cold pressor task. However, to match the analyses conducted with heart rate and RMSSD and to accommodate for the odd decrease in PEP during the first minute of recovery, the minute-by-minute PEP from room temperature task to the first minute of the cold pressor task represented the reactivity slope and minute-by-minute PEP from the first minute of the cold pressor task to the final minute of the cold pressor task represented the recovery slope\(^5\). PEP was not normally distributed so was cubic-transformed. The assumption that residual errors are normally distributed was violated so robust standard errors were used to adjust for this violation.

Maximum likelihood tests were conducted to determine whether potential covariates improved model fit prior to testing research questions. For PEP, waist-to-hip ratio significantly improved model fit. Additionally, a model with a random intercept for participants and a random slope for linear time best fit the data. Using piecewise regression, the hypothesis that simpatía would attenuate the PEP response to pain was not supported (see Figure 13). However, the pattern of results appears to be contrary to hypotheses such that greater simpatía was associated with greater reactivity (i.e. a steeper slope) compared with Latinos who valued simpatía less. Running this model only including Latino participants who tolerated pain for 4 minutes (i.e. the maximum time for the cold pressor task in this study) yielded the same pattern of results.

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\(^5\) Analyses with the knot placed at the first minute of recovery instead of the first minute of cold pressor yielded a similar pattern of non-significant results for PEP response to the cold pressor task.
Figure 13. PEP response to the cold pressor task.

**Aim 2C: Assessing potential mediators of the association between simpatía and cardiovascular responses to pain.** In Aim 1C, simpatía was only found to be associated with heart rate recovery from pain. Thus, the mediation models will only be tests for the heart rate recovery slope. As with the heart rate response analyses above, years of maternal education was controlled for in analyses.

*Do state emotions mediate the association between simpatía and cardiovascular response to pain?* To test this, the effects of simpatía on both positive and negative state emotion, were estimated separately for heart rate recovery slope.

*Positive emotion.* The direct effects model indicated that maternal years of education, simpatía, and positive emotion explained 6.1% of the variance in heart rate recovery from pain.
but this association was not statistically significant, $F(3, 93) = 2.01, p = .12$. Table 10 presents the coefficient estimates for each direct pathway in the mediation model (a: Simpatía → State Emotion; b: State Emotion → Pain; c: Simpatía → Pain) and the indirect pathway from simpatía to pain through state emotion (c’). Simpatía did not directly predict positive emotion and positive emotion did not directly predict heart rate recovery from pain. Simpatía, however, was marginally associated with a flatter heart rate recovery slope. Additionally, the indirect effect of simpatía on heart rate recovery from pain through state positive emotion was not statistically significant.

**Negative emotion.** The direct effects model indicated that maternal years of education, simpatía, and negative emotion explained 3.7% of the variance in heart rate recovery from pain but this association was not statistically significant, $F(3, 93) = 1.19, p = .32$. As Table 10 shows, simpatía did not directly predict negative emotion and negative emotion did not directly predict heart rate recovery from pain. Simpatía, however, was marginally associated with a flatter heart rate recovery slope. Additionally, the indirect effect of simpatía on heart rate recovery from pain through state negative emotion was not statistically significant. Overall, neither state positive nor state negative emotion explained the found association between simpatía and heart rate recovery.

Table 10

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**Results for State Emotion Mediation Models Predicting Heart Rate Recovery from Pain**
Emotion $\rightarrow$ HR recovery

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Simpatía $\rightarrow$ HR recovery

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<td>.087*</td>
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Indirect Effects

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<td>95% CI</td>
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<td>[-.049, .093]</td>
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Note. SE = standard error. CI = confidence interval.

* $p < .10$  
* $p \leq .05$.

**Do emotion expressions mediate the association between simpatía and cardiovascular responses to pain?** Given the sex differences in expressions, sex was controlled for in mediation analyses. To test the mediation hypothesis that such expressions would mediate the association between simpatía and cardiovascular responses, the influence of simpatía on expressions made during pain (grimace, stoicism, laughter, Duchenne smiling, social smiling) was estimated separately.

**Stoicism.** The direct effects model indicated that maternal years of education, sex, simpatía, and stoicism during pain explained 3.4% of the variance in heart rate recovery from pain but this association was not statistically significant, $F (4, 92) = 0.81, p = .52$. Table 11 presents the coefficient estimates for each direct pathway in the mediation models run for each emotion expression (a: Simpatía $\rightarrow$ Expressions; b: Expressions $\rightarrow$ Pain; c: Simpatía $\rightarrow$ Pain).
and the indirect pathway from simpatía to pain through expressions made during pain (c’).

Simpatía did not directly predict stoicism, stoicism did not directly predict heart rate recovery from pain, and simpatía did not predict heart rate recovery from pain. Additionally, the indirect effect of simpatía on heart rate recovery from pain through stoicism was not statistically significant.

**Grimacing.** The direct effects model indicated that maternal years of education, sex, simpatía, and grimacing during pain explained 3.7% of the variance in heart rate recovery from pain but this association was not statistically significant, $F (4, 92) = 0.88, p = .48$. As Table 11 shows, simpatía did not directly predict grimacing and grimacing did not directly predict heart rate recovery from pain. Simpatía, however, was marginally associated with heart rate recovery from pain. Additionally, the indirect effect of simpatía on heart rate recovery from pain through grimacing during pain was not statistically significant.

**Laughing.** The direct effects model indicated that maternal years of education, sex, simpatía, and laughing during pain explained 3.4% of the variance in heart rate recovery from pain but this association was not statistically significant, $F (4, 92) = 0.81, p = .52$. As Table 11 shows, simpatía did not directly predict laughing during pain, laughing did not directly predict heart rate recovery from pain, and simpatía did not predict heart rate recovery from pain. Additionally, the indirect effect of simpatía on heart rate recovery from pain through laughing was not statistically significant.

**Duchenne smiles.** The direct effects model indicated that maternal years of education, sex, simpatía, and Duchenne smiling during pain explained 3.5% of the variance in heart rate recovery from pain but this association was not statistically significant, $F (4, 92) = 0.83, p = .51$. As Table 11 shows, simpatía did not directly predict Duchenne smiling during pain, Duchenne
smiles did not directly predict heart rate recovery from pain, and simpatía did not predict heart rate recovery from pain. Additionally, the indirect effect of simpatía on heart rate recovery from pain through Duchenne smiles was not statistically significant.

Social smiles. The direct effects model indicated that maternal years of education, sex, simpatía, and social smiling during pain explained 3.3% of the variance in heart rate recovery from pain but this association was not statistically significant, $F (4, 91) = 0.77, p = .55$. As Table 11 shows, simpatía did not directly predict social smiling during pain, social smiles did not directly predict heart rate recovery from pain, and simpatía did not predict heart rate recovery from pain. Additionally, the indirect effect of simpatía on heart rate recovery from pain through social smiles was not statistically significant. Overall, results suggest that no expression made during pain explained the found association between simpatía and heart rate recovery from pain.

Table 11

Results for Emotion Expression Mediation Models Predicting Heart Rate Recovery from Pain

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<tr>
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<tr>
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<td>.060</td>
<td>-1.616</td>
<td>.110</td>
<td>[-.215, .022]</td>
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</table>

Expressions $\rightarrow$ HR Rec
Stoicism        .065  .456  .143  .887  [-.841, .972]
Grimacing      .396  .755  .524  .602  [-1.104, 1.896]
Laughter       -.453  3.322 -.136 .892  [-7.051, 6.145]
Duchenne Smiles .184  .665  .277 .782  [-1.136, 1.504]
Social Smiles  -.030  .255 -.118 .906  [-.536, .476]

**Stimpatia → HR Rec**

<table>
<thead>
<tr>
<th></th>
<th>Boot SE</th>
<th>95% Boot CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stoicism</td>
<td>.235</td>
<td>[.144, 1.636]</td>
</tr>
<tr>
<td></td>
<td>.105</td>
<td>[.050, .521]</td>
</tr>
<tr>
<td>Grimacing</td>
<td>.241</td>
<td>[.144, 1.675]</td>
</tr>
<tr>
<td></td>
<td>.097+</td>
<td>[-.045, .527]</td>
</tr>
<tr>
<td>Laughter</td>
<td>.234</td>
<td>[.144, 1.619]</td>
</tr>
<tr>
<td></td>
<td>.109</td>
<td>[-.053, .520]</td>
</tr>
<tr>
<td>Duchenne Smiles</td>
<td>.239</td>
<td>[.144, 1.656]</td>
</tr>
<tr>
<td></td>
<td>.101</td>
<td>[-.048, .526]</td>
</tr>
<tr>
<td>Social Smiles</td>
<td>.226</td>
<td>[.148, 1.529]</td>
</tr>
<tr>
<td></td>
<td>.130</td>
<td>[-.068, .520]</td>
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</tbody>
</table>

Indirect Effects

<table>
<thead>
<tr>
<th></th>
<th>Boot SE</th>
<th>95% Boot CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stoicism</td>
<td>.0001</td>
<td>[.016, .035]</td>
</tr>
<tr>
<td>Grimacing</td>
<td>-.006</td>
<td>[.024, .037]</td>
</tr>
<tr>
<td>Laughter</td>
<td>.002</td>
<td>[.016, .030]</td>
</tr>
<tr>
<td>Duchenne Smiles</td>
<td>-.004</td>
<td>[.013, .036]</td>
</tr>
<tr>
<td>Social Smiles</td>
<td>.003</td>
<td>[.025, .050]</td>
</tr>
</tbody>
</table>

*Note. HR Rec = Heart rate recovery slope. SE = standard error. CI = confidence interval. *p < .10
**p < .05.

*Does vagal tone mediate the association between simpatía and cardiovascular response to pain?* To test this mediation hypothesis, that vagal tone would mediate the found association between simpatía and heart rate recovery, the influence of simpatía on two different measures of
vagal tone (RMSSD and RSA) were estimated separately controlling for maternal years of education and age.

RMSSD. The direct effects model indicated that maternal years of education, age, simpatía, and log-transformed RMSSD explained 3.6% of the variance in heart rate recovery from pain, $F(4, 96) = 0.89, p = .47$. Table 12 presents the coefficient estimates for each direct pathway in the mediation model (a: Simpatía $\rightarrow$ Vagal Tone; b: Vagal Tone $\rightarrow$ Pain; c: Simpatía $\rightarrow$ Pain) and the indirect pathway from simpatía to pain through vagal tone (c’). Simpatía did not predict RMSSD and RMSSD did not predict heart rate recovery from pain. However, greater value in simpatía marginally predicted a flatter heart rate recovery slope. The indirect effect of simpatía and heart rate recovery from pain through RMSSD was not statistically significant.

RSA. The direct effects model indicated that maternal years of education, age, simpatía, and RSA explained 3.7% of the variance in heart rate recovery from pain, $F(4, 96) = 0.92, p = .45$. As Table 12 shows, simpatía did not predict RSA and RSA did not predict heart rate recovery from pain. However, greater value in simpatía marginally predicted a flatter heart rate recovery slope. The indirect effect of simpatía and heart rate recovery from pain through RSA was not statistically significant.

Table 12

| Results for Vagal Tone Mediation Models Predicting Heart Rate Recovery from Pain |
|---------------------------------|-----|-----|-----|-----|-----|
| Variables | $b$  | $SE$ | $t$  | $p$  | 95% CI  |
| Direct Effects |      |      |      |      |      |
| Simpatía $\rightarrow$ Vagal Tone |      |      |      |      |      |
| RMSSD   | .001 | .009 | .098 | .953 | [-.017, .019] |
| RSA     | .006 | .019 | .339 | .736 | [-.031, .044] |
**Vagal Tone → HR Recovery**

<table>
<thead>
<tr>
<th></th>
<th>Boot SE</th>
<th>95% Boot CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMSSD</td>
<td>.324</td>
<td>[−2.612, 3.260]</td>
</tr>
<tr>
<td>RSA</td>
<td>.308</td>
<td>[−1.125, 1.741]</td>
</tr>
</tbody>
</table>

**Simpatía → HR Recovery**

<table>
<thead>
<tr>
<th></th>
<th>Boot SE</th>
<th>95% Boot CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMSSD</td>
<td>.236</td>
<td>[−.030, .502]</td>
</tr>
<tr>
<td>RSA</td>
<td>.235</td>
<td>[−.031, .501]</td>
</tr>
</tbody>
</table>

**Indirect Effects**

<table>
<thead>
<tr>
<th></th>
<th>Boot SE</th>
<th>95% Boot CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMSSD</td>
<td>.0003</td>
<td>[−.037, .033]</td>
</tr>
<tr>
<td>RSA</td>
<td>.002</td>
<td>[−.047, .045]</td>
</tr>
</tbody>
</table>

*Note.* HR = heart rate. SE = standard error. CI = confidence interval. *p < .10*  *p < .05.*

Overall, state emotions, emotional expressions, and vagal tone considered in this study did not explain the found association between simpatía and heart rate recovery.

**Aim 3C: What is the role of ethnicity in the association between simpatía and cardiovascular responses to pain?** Given the ethnic differences in body mass index, waist-to-hip ratio, and maternal years of education, these variables were considered as covariates. As was done in Aim 1C, the knot for the piecewise regression was placed at the first minute of the cold pressor task, representing when participants, on average, exhibited the greatest physiological response from the room temperature task before beginning to recover. Whether the results from Aim 1C replicated after adding in the European American sample was tested for each cardiovascular outcome variable (heart rate, RMSSD, and PEP).

**Heart rate.** Maximum likelihood tests were conducted to determine whether potential covariates improved model fit prior to testing research questions. For heart rate, no covariate
improved model fit. Additionally, a model with a random intercept for participants and random slope for linear time best fit the data. Upon adding in the European American sample to the piecewise regression, the results indicated that simpatía was still associated with the heart rate recovery slope. When ethnic background was entered into the model independently, it marginally improved model fit so was included. However, when a three-way interaction between simpatía, time, and ethnic background was entered into the model, the model fit did not improve and the interaction was not statistically significant. Thus, the model with ethnic background as an independent predictor and the interaction between simpatía and time was interpreted. As seen in Table 13, there was a significant interaction between simpatía and recovery even after the European American sample was added in.

Table 13

*Multilevel Between Person Effects of the Interaction of Simpatía with Heart Rate Recovery*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Coefficients (RSE)</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed effects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>4.163 (0.019)***</td>
<td>[4.127, 4.200]</td>
</tr>
<tr>
<td>Reactivity</td>
<td>0.128 (0.008)***</td>
<td>[0.114, 0.143]</td>
</tr>
<tr>
<td>Recovery</td>
<td>-0.033 (0.003)***</td>
<td>[-0.039, -0.027]</td>
</tr>
<tr>
<td>Simpatía</td>
<td>0.0001 (0.002)</td>
<td>[-0.004, 0.005]</td>
</tr>
<tr>
<td>Reactivity X Simpatía</td>
<td>-0.001 (0.001)</td>
<td>[-0.003, 0.002]</td>
</tr>
<tr>
<td>Recovery X Simpatía</td>
<td>0.001 (0.0006)*</td>
<td>[0.0003, 0.003]</td>
</tr>
<tr>
<td>Ethnic background *</td>
<td>-0.015 (0.020)</td>
<td>[-0.054, 0.023]</td>
</tr>
<tr>
<td>Random effects</td>
<td></td>
<td>Estimate</td>
</tr>
</tbody>
</table>
Random intercept ($\sigma_0^2$)  & 0.012 (0.002) & [0.010, 0.016]  
Random linear slope ($\sigma_1^2$)  & 0.0002 (0.0001) & [0.0001, 0.0004]  
Residual variance  & 0.003 (0.0004) & [0.003, 0.004]  

*Note.* Based on 179 participants with 663 longitudinal records. RSE = robust standard errors; CI = confidence interval. *a* Latinos coded 1, European Americans coded 0. *p ≤ .05. ***p < .001.

Figure 14 visualizes the interaction between simpatía and the recovery period, $z = 2.48, p = .013$. Non-transformed values of heart rate are plotted in the figure despite all analyses being conducted using log-transformed values for ease of comparison across other studies. Contrary to hypotheses, greater simpatía was associated with decreased recovery (i.e. a flatter slope) compared with individuals who valued simpatía less, regardless of ethnic background. Running this model only including participants who tolerated pain for 4 minutes (i.e. the maximum time for the cold pressor task in this study) yielded the same pattern of results.
Figure 14. Heart rate response to pain is altered by simpatía, regardless of ethnic background.

**RMSSD.** Maximum likelihood tests were conducted to determine whether potential covariates improved model fit prior to testing research questions. For RMSSD, no covariate improved model fit. Additionally, a model with a random intercept for participants best fit the data. Upon adding in the European American sample to the piecewise regression, the results indicated that simpatía was still not associated with the parasympathetic response to pain, despite the interaction between simpatía and time improving model fit. When ethnic background was entered into the model independently, it did not improve model fit so was not included. Additionally, when a three-way interaction between simpatía, time, and ethnic background was
entered into the model, the model fit did not improve and the interaction was not statistically significant. Thus, the model with the interaction between simpatía and time was interpreted.

Figure 15 visualizes the non-significant interaction between simpatía and the parasympathetic response. Non-transformed values of heart rate are plotted in the figure despite all analyses being conducted using log-transformed values for ease of comparison across other studies. Running this model only including participants who tolerated pain for 4 minutes (i.e. the maximum time for the cold pressor task in this study) yielded the same pattern of results.

![Predictive Margins with 95% CIs](image)

**Figure 15.** Parasympathetic response to pain is not significantly altered by simpatía.

**PEP.** Maximum likelihood tests were conducted to determine whether potential covariates improved model fit prior to testing research questions. For PEP, no covariate improved model fit. Additionally, a model with a random intercept for participants best fit the
data. Upon adding in the European American sample to the piecewise regression, the results indicated that simpatía was still not associated with the sympathetic response to pain, adding simpatía to the model independently or as an interaction with time did not improve model fit. When ethnic background was entered into the model independently, it also did not improve model fit. Additionally, when a three-way interaction between simpatía, time, and ethnic background was entered into the model, the model fit did not improve and the interaction was not statistically significant. However, a two-way interaction between ethnic background and time was entered into the model and marginally improved model fit. Thus, the model with the interaction between ethnicity and time was interpreted.

Figure 16 visualizes the non-significant interaction between ethnicity and the sympathetic response to pain, $p$’s $> .15$. Despite the ethnicity by sympathetic response interaction being non-significant, ethnicity independently predicted the sympathetic response trajectory, $z = -2.30$, $p = .021$. Non-transformed values of PEP are plotted in the figure despite all analyses being conducted using cubic transformed values for ease of comparison across other studies. As seen in the figure, European Americans appeared to have a more robust sympathetic response to the cold pressor task and a steeper slope for recovery whereas Latinos appeared to have flatter sympathetic reactivity and recovery slopes. Running this model only including participants who tolerated pain for 4 minutes (i.e. the maximum time for the cold pressor task in this study) resulted in a similar pattern\(^6\) of results.

\(^6\) Considering only participants who endured pain for four minutes, maternal years of education and the interaction between ethnicity and time significantly improved the model fit. Results indicated that ethnicity was no longer a significant independent predictor of the sympathetic response trajectory. However, the ethnicity by reactivity slope interaction was marginally significant, $z = 1.77$, $p = .076$. See Appendix G for a version of Figure 17 that only includes participants who tolerated pain for four minutes.
Figure 16. Ethnic background alters the trajectory of sympathetic nervous system response to pain.

Discussion

This dissertation examined the role that a prevalent Latino cultural value, simpatía, plays in the context of a potentially norm-violating acute stress event: the cold pressor task. Contrary to hypotheses that simpatía would be helpful in the context of pain, the results of this dissertation suggest that simpatía is associated with a worse pain experience in Latinos. Specifically, simpatía was associated with greater self-reported pain, decreased pain tolerance, and a flatter heart rate recovery slope. This association, however, was not explained by any of the hypothesized mediators: state emotions, emotion expressions, or vagal tone. In the context of
experimentally-induced pain, simpatía was associated with fewer expressions in Latinos and did not appear to function similarly in a European American sample.

The findings of this dissertation make key contributions to the literature. First, they suggest that simpatía has links, albeit negative, to the experience of acute, experimentally induced pain but only in Latinos. Second, these findings demonstrate the types of expressions naturally made during pain by an ethnically diverse group and suggests that perhaps nonverbal measures of pain common in medical settings may insufficiently gauge Latino pain (as grimacing during experimental pain did not occur very much). Third, this dissertation replicated results showing ethnic differences in pain such that Latinos reported more pain and were more likely to withdraw from pain early compared with European Americans. Lastly, this dissertation adds to the emotion-pain literature showing that state emotions are weakly associated with self-reported pain and expressions made during pain are associated with pain tolerance. In the following sections, I will discuss the many interesting findings from this dissertation aim by aim and then review the broader implications, limitations, and future directions.

**Aim 1: Is Simpatía Associated with Pain within Latinos?**

The first aim of this dissertation was to determine whether simpatía was associated with experimentally induced pain. Based on prior research (e.g., Acevedo et al., under review; Holloway et al., 2009), it was expected that valuing simpatía would be associated with an increased tolerance for pain and would attenuate both self-reported pain and the cardiovascular response to pain. Both positive emotions and positive facial expressions (Duchenne smiles) have been shown to reduce self-reports of pain and attenuate cardiovascular responses to pain (e.g., Acevedo, Leger, Jenkins, & Pressman, in prep; Kraft & Pressman, 2012; Pressman, Acevedo,
Aucott, Kraft-Feil, under review). However, the results of this study do not support a link between simpatía and positive emotions.

Importantly, the lack of a simpatía-positive emotion association could be due to the context of the experimentally induced pain setting. In the context of the current study, the experimenter was trained to be neutral in affect and un-sympathetic of the participant’s pain. Little is known about what happens when simpatía behaviors are not reciprocated, but at least one study indicates that a lack of behavioral reciprocity during social interactions may exacerbate distress in Latinos (Sanchez-Burks, Blount, & Bartel, 2006). In a field experiment conducted at a large company, managers of European or Latino background took part in a videotaped mock interview in which confederate interviewers either engaged or did not engage in mirroring behaviors (i.e. mimicking nonverbal gestures) throughout the interview (Sanchez-Burks, Blount, & Bartel, 2006). The researchers found that Latino managers self-reported higher anxiety in the non-mirroring condition whereas the anxiety of European Americans did not differ by condition. To the extent that participants sought to engage with the experimenter during pain, the lack of engagement on the part of the experimenter may have triggered greater distress in Latino participants. In remaining neutral throughout the study and especially while the participant experienced pain, the experimenter may have also violated norms and expectations for simpatía during the study. Specifically, if simpatía is associated with greater expectations for positive social interactions, then this experimentally induced pain setting may be a particularly negative experience for such participants.

Diverting attention away from pain through distraction is a well-established method of reducing pain perceptions (e.g., Birnie et al., 2014; Dahlquist et al., 2007; McCaul & Haugtvedt, 1982; Mccaull & Malott, 1984). However, in this expectation-violating context, Latinos high in
simpatía may have been less willing to endure pain compared with those who perceived the pain as something that should be endured. Alternatively, this negative experience may have increased the attention towards pain along with the voluntary nature of the pain. In other words, participants who expected a warm, polite experimenter or a more positive experience in general but instead faced a neutral person during a painful situation may have been more inclined to wonder why they should even try to endure the pain. The voluntary nature of this pain was emphasized extensively prior to the beginning of the cold pressor task for all participants due to concerns about reducing the risk of cardiovascular syncope from occurring during pain. Perhaps these instructions along with the violated expectations for a positive experience increased attention towards the pain, in turn, increasing self-reports of pain in Latinos high in simpatía. Attention towards pain has been shown to increase self-reported pain and decrease pain tolerance in other studies. For example, one study demonstrated that when participants are instructed to look towards the pain, they self-reported more pain (Moseley & Arntz, 2007). Conversely, encouraging participants to continue in the task, to distract themselves, or to reinterpret the meaning of the pain have all been tied to attenuated pain (i.e., decreased self-reported pain, increased pain tolerance) compared with having an empathetic experimenter (Jackson, Iezzi, Chen, Ebnet, & Eglitis, 2005).

Another way this context may have influenced pain in Latinos is the experimenters ethnic background and the degree to which it matched the participants’ ethnic background. Following participant 18, I added in a couple additional questions towards the end of the study (right before being debriefed) including one asking the participant “If you had to guess, what racial ethnic background do you think the experimenter you spoke with today identifies with?” Therefore, we know the self-reported ethnic background of both the participant and the experimenter along
with the participants’ perception of the experimenters’ ethnic background. While outside the scope of this dissertation, there are some indications that the experimenters background may have influenced self-reports of pain in Latinos. Specifically, it appears that Latino participants who had a Latina experimenter self-reported less pain on average \((n = 57, M = 44.69, SD = 24.16)\) compared with Latino participants who had a European American experimenter \((n = 65, M = 51.31, SD = 25.24)\), although this difference was not statistically significant, \(t(120) = 1.48, p = .143\). Therefore, publications coming from this dissertation study will need to consider the ethnic background of the experimenter as it seems to also be relevant to the results of the mediation analyses.

**Aim 2: Do Any Biopsychosocial Factors Explain the Simpatía-Pain Association?**

The second aim of this dissertation was to determine whether the association between simpatía and experimentally induced pain could be explained by certain biopsychosocial factors that have previously been tied to attenuated pain. Based on prior research tying Latinos to a greater likelihood of expressing positive emotions during social interactions (Holloway et al., 2009) and research linking simpatía with dispositional positive emotions and social smiling during stress (Acevedo et al., under review), it was hypothesized that simpatía would be beneficial to pain through a variety of factors that have been tied to pain. Specifically, it was thought that simpatía would be associated with more state positive emotion and less state negative emotion and that these associations would explain the hypothesized negative association between simpatía and pain. However, simpatía did not have strong links to self-reported state emotions prior to pain in the current study. This could have been due to the way emotion was measured in this study because previous work has linked simpatía with a trait-like disposition towards positive emotion rather than high state positive emotion. While this may help
explain the lack of an association between simpatía and positive emotion, the finding that simpatía was actually positively associated with state negative emotion was unexpected.

As mentioned above, however, the ethnic background of the experimenter may help elucidate why simpatía was associated with state negative emotion in this study. I split the data file by the experimenter’s ethnic background and examined whether simpatía was still associated with state negative emotion depending on the experimenter’s ethnicity. I found that the positive association between simpatía and state negative emotion was only significant in Latino participants who had a European American experimenter ($r = .34, p = .007$) but not significant in Latino participants with a Latina experimenter ($r = .03, p = .86$). However, the difference in the strength of these associations did not reach statistical significance (Fisher’s $z = 1.72, p = .085$).

The questionnaire used to assess state emotions in this study was provided after having already interacted with the experimenter. In other words, after providing informed consent, having anthropometric measures taken and cardiovascular monitoring sensors placed on their torso by the experimenter, the participants then completed the state emotion questionnaire. Thus, it is possible that the simpatía violating context of this study had a more negative impact on Latino participants who were high in simpatía and interacted with an out-group, ethnic majority member compared with when such participants interacted with an in-group member.

Additionally, I wanted to pry into the positive association between simpatía and negative emotions further. Within the Latino sample, when examining the association of the items on the simpatía scale with the items on the state negative emotion scale, it appeared that many of the associations between simpatía and the state negative emotion scale were positively and significantly correlated with more low-arousal negative emotion items: tense, sleepy, and unhappy. This suggests that individuals high in simpatía may perhaps find norm-violating
contexts as more tense and less pleasant than individuals low in simpatía. This context, the experimenter’s ethnic background, and the timing of the questionnaire assessing state emotions could have all impacted the (lack of) association between emotions and pain.

The timing of the state emotion assessment may also explain the lack of found associations between positive emotions and reduced pain. This rationale is plausible because the literature linking self-reported positive emotions to reductions in pain has only shown such associations with either an experimentally-induced positive emotion (Acevedo, Leger, Jenkins, & Pressman, in prep) or trait-like positive emotions (Cohen et al., 2006). This suggests that in the current study, what individuals feel in the moment near the beginning of an experiment may not have an impact on their perceptions of pain near the end of the experiment. An alternative explanation for the lack of an association between positive emotions and pain is that this association may be different in Latino samples compared with the more typically studied European American samples. However, this explanation may not be as likely because when examining the raw data, the association between state positive emotion and self-reported pain is negative in both the Latino and European American sample, the correlation coefficients are just too small to reach statistical significance ($r_L = -.159$; $r_{EA} = -.179$).

It was also hypothesized that Latinos high in simpatía might either be more likely to exhibit positive expressions during pain or would be more likely to remain stoic during pain. Results indicated that in Latinos, greater simpatía was associated with fewer smiles made during the first 30 seconds of pain and was not associated with stoicism. These findings are in line with past research by Acevedo and colleagues (under review) who found simpatía was negatively associated with social smiles during a stressor that involved giving a speech and performing an arithmetic task in front of two neutral observers but that this was only true in males. The current
study had only 21 Latino males so cannot strongly interpret gender differences in expressions made during pain for Latinos. However, the data suggest similar patterns to previous research as simpatía was associated with fewer social smiles particularly in Latino males \( r (20) = -.437, p = .054 \) compared with Latinas \( r (84) = -.176, p = .11 \) although the strength of these associations were not statistically different from each other, Fisher’s \( z = -1.09, p = .28 \). Clearly, however, gender on its own had a strong influence on the expressions made during pain because while simpatía was not associated with remaining stoic during pain. As seen in Appendix B, Latino males remained stoic during the initial response to pain more often than Latinas and European Americans of either sex. Additionally, no Latino male expressed a grimace, a social smile, or a Duchenne smile during the initial response to pain. Together with previous work, these findings suggest the importance of examining sex differences within Latinos to better understand the interaction between Latino cultural values that dictate norms for social interactions (i.e., simpatía) and Latino cultural values that dictate norms for gender (i.e., machismo, marianismo).

To my knowledge, no study has examined both machismo/marianismo and simpatía in the context of pain, highlighting a gap in the literature.

Interestingly among Latinos, stoicism was associated with increased pain tolerance while results suggested a marginal negative association between Duchenne smiling and pain tolerance. This appears to contrast with some work finding that displaying a neutral expression is associated with greater self-reported pain during a needle injection compared with displaying a Duchenne smile, social smile, or even a grimace (Pressman, Acevedo, Aucott, & Kraft-Feil, under review). An important distinction between many of the positive facial expression and reduced pain findings (e.g., Kraft & Pressman, 2012; Pressman, Acevedo, Aucott, & Kraft-Feil, under review), is that these previous studies manipulated facial expressions while the current
study measured facial expressions that were naturally made during pain in the laboratory. Across the Latino sample, stoicism was the most common behavior expressed during pain which is consistent with a literature review showing that stoicism is also common among Latinos in chronic pain contexts as well (Hollingshead, Ashburn-Nardo, Stewart, & Hirsh, 2016). The positive association between stoicism and pain tolerance suggests that remaining expressionless during pain for Latinos signals to others that “they are okay” and “can handle the pain”. However, the lack of grimaces among the Latinos in this sample also suggests that measuring pain nonverbally (as is commonly done young or nonverbal individuals in medical settings; e.g., Herr et al., 2006; Sheu et al., 2011) may not adequately capture Latino pain either.

Additionally, the display of smiles naturally during pain and the negative association between smiling and pain tolerance in the current study provides support for the smile of pain (Kunz et al., 2009; Kunz, Prkachin, & Lautenbacher, 2013) that has been described to naturally occur in experimentally-induced pain settings in both pain-free participants and clinical pain patients. This type of smile may serve to signal to others that, “I’m not okay but I can handle this”. In the facial expression literature, this kind of smile is called a ‘miserable smile’ (Ekman & Friesen, 1982) because they are not meant to convey the experience of positive emotion to another person. However, it is also possible that the smiles expressed in this study were ‘false smiles’ which, according to Ekman and Friesen, represent an attempt to cover or mask negative emotions that are felt. The authors hypothesize that an important difference between miserable and false smiles are that miserable smiles would be more likely to be displayed immediately following a negative expression as the combination of those expressions would signal to others first, “I’m not okay” then “but I can handle it”. In comparison, a false smile would exhibit activation of facial muscle movements associated with both a smile and negative emotion at the
same time. Thus, an important future direction for understanding natural facial expressions made during pain is to identify the specific type of smile individuals choose to display.

The biological factor that I hypothesized might explain the association between simpatía and pain was vagal tone, however, this hypothesis was not supported. No work has ever linked simpatía specifically with vagal tone but agreeableness (a construct related to simpatía) has been shown to relate to vagal tone as measured by RSA (Oveis et al., 2009); however, this study was done in a sample comprised of mostly individuals from an Asian background. The results of this study demonstrate that for young adult Latinos, there is no association between simpatía and vagal tone. Additionally, results of this study do not support previous work showing vagal tone is associated with lower pain unpleasantness ratings during the cold pressor task (Appelhans & Luecken, 2008). The evidence for this potential mediator was weak when one considers that only one study linking each pathway from simpatía (really, agreeableness) to vagal tone and from vagal tone to self-reported pain was used to make this prediction. There is a dearth of literature on vagal tone more broadly among Latino samples. As low vagal tone has been associated with a plethora of negative physical outcomes including worse self-rated health (e.g., Jarczok et al., 2015), increased risk for cardiovascular disease and mortality (e.g., Thayer & Lane, 2007; Thayer, Yamamoto, & Brosschot, 2010) and lower vagal tone has been linked to increased self-reported discrimination in African American samples (Hill et al., 2017), it will be important for my future work to examine whether this extends to Latino samples.

**Aim 3: What is the Role of Ethnic Background in the Simpatía-Pain Association?**

Interestingly, ethnicity did not significantly moderate any of the simpatía-pain associations and the findings here were complex. This cannot be explained through ethnic differences in simpatía, however, because both ethnic groups reported valuing simpatía similarly.
Instead, results suggest that either construct of simpatía functions differently in European American culture or that this construct carries a different meaning. Therefore, it is important for future work with larger European American samples to examine whether measures of simpatía are equivalent across cultures. Thus, the simpatía findings (or lack thereof) for European Americans must be considered in light of this important methodological issue in the current literature.

Consistent with the literature (Rahim-Williams et al., 2012), ethnic background was an independent predictor of pain tolerance, however, simpatía did not appear to alter or explain this difference. For European Americans, in order to understand whether simpatía was associated with pain tolerance in the same direction as Latinos, the file was split by ethnic background. In European Americans, simpatía was not associated with propensity to withdraw early from pain, controlling for sex, $B = .046, SE = .06, B/SE = .85, p = .40$. However, it should be noted that the pattern of withdrawing from pain seemed to be different in European Americans compared with Latinos. At least one Latino participant withdrew from pain during every 30 second segment of the 4-minute maximum cold pressor task, however, European American participants either withdrew from pain in the first 90 seconds of the cold pressor or near the end of the 4-minute task (i.e. no European participant withdrew from pain in the middle of the cold pressor task).

This pattern of withdrawal from pain suggests potential differences in attitudes regarding how much experimentally-induced pain should be avoided versus endured. In fact, while the experimental setting can be helpful in isolating certain factors or contexts that matter to our understanding of ethnic differences in pain, it can also exacerbate ethnic differences by creating a different meaning for the pain experienced (Zatzick & Dimsdale, 1990). Perhaps ethnic minority groups in experimentally-induced pain settings are less willing to endure pain for the
sake of science, especially considering the history of unethical research practices targeting people of color (e.g., Tuskegee Syphilis Study). However, while these attitudes may partially explain ethnic differences in pain, the type of pain induced can also exacerbate or attenuate ethnic differences in pain. For example, in a study examining ethnic differences in experimentally induced pain versus acute clinical pain (i.e., standardized oral surgery where a molar was removed for medical reasons), researchers found that ethnic differences in self-reported pain and pain tolerance during the cold pressor task were significant but there were no ethnic differences in the level of self-reported clinical pain (Hyungsuk Kim et al., 2004). As the factors measured in the current study could not elucidate why we found ethnic differences in pain, it would be interesting to replicate this finding in future studies using different pain induction techniques (both in the lab and in naturalistic pain settings).

Limitations and Future Directions

There were many interesting findings in this dissertation but the study is not without limitations. Importantly, the experimental context could have had an impact on the study findings and without having another pain induction task that is more naturalistic to clinical pain, it is difficult to understand the generalizability of these findings outside of the experimental pain context. Thus, extending research on Latinos in pain and on ethnic differences in pain to more pain contexts is an important future direction.

Empirically examining how Latinos and Latino culture impacts responses to pain where and when it naturally occurs is critical, given how understudied Latinos are in the pain literature relative to other racial/ethnic groups. It would be particularly interesting to also examine the relative frequency of bringing family to medical settings where pain must be endured in Latinos compared with other groups. Specifically, future work should examine the degree to which the
patient and their family touch while the patient experiences pain because holding hands with one’s intimate partner has been shown to reduce pain compared with holding a stranger’s hand or not holding a hand (Master et al., 2009).

Another limitation is the lack of sympathetic activation in response to the cold pressor task. This is contrary to previous work by myself and Sarah Pressman (Acevedo, Leger, Jenkins, & Pressman, in prep) and contrary to a literature review demonstrating that experimentally induced pain increases sympathetic activation and parasympathetic withdrawal (Koenig et al., 2013). It is possible that the warmer temperature of the water in this study (5°C) was not stressful enough for the sympathetic nervous system to also be recruited in the physiological response to pain compared with previous studies that found this association. These mixed findings highlight the importance of future work to measure physiological changes in response to experimentally induced pain as a function of water temperature.

There was a lack of variation in sex in the current study that prevented strong interpretations of sex differences among Latinos. This is an important limitation as past research has shown that simpatía is particularly beneficial to physiological responses to stress (Acevedo et al., under review) and cardiovascular health (Merz et al., 2016) in males only. In contrast, benefits of simpatía that are specific to Latinas have yet to be found. Future research should strive to examine the intersection of Latino cultural values and Latino gender identity as it relates to the context of pain and stress more broadly. As the sex of the experimenter has been shown to influence pain ratings such that participants tend to report lower pain ratings when the experimenter was female compared with when the experimenter was male (Levine & Simone, 1991), the use of only female experimenters in the current study could have influenced our
findings. Overall, this suggests the critical importance of examining the influence of both participant and experimenter sex in the study of Latino pain.

**Conclusion**

In sum, the results of this dissertation seem to imply that valuing simpatía could be seen as more of a risk factor for experimentally-induced pain rather than a protective factor as initially hypothesized. In particular, the context of the pain induction for this study seemed to induce a state of negative emotionality in participants high in simpatía, perhaps because they came in expecting a pleasant interaction from the outset of the study. However, this finding does not seem to extend to European American samples, either because measures of simpatía have never been validated in these samples or because simpatía functions differently in this sample compared with Latinos. These findings have important implications for managing Latino pain, suggesting that the experimental pain context may be particularly jarring for Latinos who value simpatía due to violations in expectations for the experience. This hints at the potential for a culturally-tailored intervention that trains health care providers to display simpatía-related behaviors for Latinos undergoing pain to improve pain outcomes. Overall, this dissertation adds to the growing literature on the function of cultural values during experimentally induced pain and brings attention to the need to develop measures of simpatía that are validated in Latino as well as European American samples.
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Appendix A

Relevant Covariates

Although investigating sex and acculturation differences is beyond the scope of this dissertation, it is important to highlight that these differences exist within the context of both simpatía and experimental pain literatures. These factors might alter how much simpatía is enacted as a cultural value and how pain is reported. Thus, these factors will be measured in the proposed study to be controlled for in analyses.

Sex. While research on sex differences in simpatía values is lacking, some work suggests females may value and enact simpatía more often than males (e.g., Durik et al., 2006; Holloway et al., 2009). For example, one would expect males to enact less simpatía-related emotions compared to females because the machismo value (a masculine ideal where men are expected to be dominant, authoritative, and aggressive) is seemingly at odds with being simpatico (Durik et al., 2006). One study found that female-female dyadic interactions exhibited greater duration of talking, partner-directed gazing, and positive affect displays compared to male-male dyadic interactions (Holloway et al., 2009). However, this was not moderated by ethnic background suggesting that simpatía-related behaviors in social interactions may be more gendered than related to ethnic background. There is also research that suggests sex influences experimental pain sensitivity. A meta-analysis found females were less tolerant of pain compared to males, and this effect size was moderate to large (Riley III, Robinson, Wise, Myers, & Fillingim, 1998). Therefore, it is important to enter sex as a covariate in analyses to control for these differences.

Acculturation. There is mixed evidence for whether simpatía values are influenced by acculturation with some work finding no acculturation differences in simpatía (Griffith, Joe, Chatham, & Simpson, 1998) or slight acculturation differences in simpatía (Triandis, Kashima,
Shimada, & Villareal, 1986). There is also mixed evidence for whether pain is influenced by acculturation with some work finding acculturations effects become nonsignificant after accounting for other sociodemographic factors in chronic pain patient populations (Jimenez, Dansie, Buchwald, & Goldberg, 2013). Relatedly, one experimental pain study comparing African and Latino Americans with European Americans found that ethnic identity (the degree to which one affirms and identifies with their ethnic identity) explained ethnic differences in ischemic and heat pain however, did not explain ethnic differences in cold pain (Rahim-Williams et al., 2007). Therefore, it is important to enter acculturation as a covariate in analyses to control for these differences.
Appendix B. Expressions made during the initial 30 seconds of the cold pressor task by sex and ethnic background. Error bars represent standard error of the mean.
Appendix C

Means, standard deviations, and intercorrelation matrices. Latinos (n = 123) below the diagonal, European American (n = 63) intercorrelations above the diagonal

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
</tr>
</thead>
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<tr>
<td>1. Simpatía</td>
<td>27.63</td>
<td>5.38</td>
<td>.24+</td>
<td>.19</td>
<td>.16</td>
<td>-1.7</td>
<td>.24+</td>
<td>.07</td>
<td>.08</td>
<td>.14</td>
<td>.02</td>
<td>-1.4</td>
<td></td>
</tr>
<tr>
<td>2. State PA</td>
<td>2.91</td>
<td>0.74</td>
<td>-0.1</td>
<td>-0.39*</td>
<td>.19</td>
<td>-0.08</td>
<td>.03</td>
<td>.11</td>
<td>-1.2</td>
<td>.14</td>
<td>-1.18</td>
<td>.20</td>
<td></td>
</tr>
<tr>
<td>3. State NA</td>
<td>2.06</td>
<td>0.62</td>
<td>.20+</td>
<td>-0.31**</td>
<td>-0.03</td>
<td>.05</td>
<td>.10</td>
<td>-0.25+</td>
<td>.14</td>
<td>-0.08</td>
<td>.17</td>
<td>-2.0</td>
<td></td>
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<tr>
<td>4. Grimacing</td>
<td>0.26</td>
<td>1.01</td>
<td>-0.09</td>
<td>.11</td>
<td>-0.04</td>
<td>-0.53**</td>
<td>-0.03</td>
<td>.02</td>
<td>-0.02</td>
<td>-0.03</td>
<td>-0.05</td>
<td>.23</td>
<td></td>
</tr>
<tr>
<td>5. Stoicism</td>
<td>3.97</td>
<td>1.70</td>
<td>.01</td>
<td>-0.08</td>
<td>.10</td>
<td>-0.18+</td>
<td>-0.44**</td>
<td>-0.54**</td>
<td>-0.55**</td>
<td>.08</td>
<td>-0.14</td>
<td>.52</td>
<td></td>
</tr>
<tr>
<td>6. Laughter</td>
<td>0.06</td>
<td>0.23</td>
<td>-0.04</td>
<td>.14</td>
<td>-0.10</td>
<td>.33**</td>
<td>-0.29*</td>
<td>.48**</td>
<td>.69**</td>
<td>-0.05</td>
<td>.06</td>
<td>-0.23*</td>
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<tr>
<td>7. Duchenne</td>
<td>0.44</td>
<td>1.69</td>
<td>-0.15</td>
<td>.21+</td>
<td>-0.01</td>
<td>.07</td>
<td>-0.40**</td>
<td>.52**</td>
<td>.22</td>
<td>.01</td>
<td>.20</td>
<td>-0.01</td>
<td></td>
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<tr>
<td>8. Social</td>
<td>1.51</td>
<td>3.07</td>
<td>-0.13</td>
<td>.16</td>
<td>-0.07</td>
<td>.09</td>
<td>-0.73**</td>
<td>.50**</td>
<td>.53**</td>
<td>-0.20</td>
<td>.02</td>
<td>-0.41*</td>
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<td>9. Vagal tone a</td>
<td>3.76</td>
<td>0.04</td>
<td>.02</td>
<td>.16+</td>
<td>-0.14</td>
<td>.06</td>
<td>.05</td>
<td>-0.03</td>
<td>-0.02</td>
<td>-0.13</td>
<td>.04</td>
<td>-0.01</td>
<td></td>
</tr>
<tr>
<td>10. Average pain</td>
<td>48.22</td>
<td>24.86</td>
<td>.15</td>
<td>-0.16+</td>
<td>.07</td>
<td>.09</td>
<td>.04</td>
<td>.05</td>
<td>-0.11</td>
<td>-0.01</td>
<td>.10</td>
<td>-0.19</td>
<td></td>
</tr>
<tr>
<td>11. Pain tolerance</td>
<td>147.28</td>
<td>96.91</td>
<td>-0.17+</td>
<td>-0.16+</td>
<td>-0.03</td>
<td>-0.12</td>
<td>.27**</td>
<td>-0.08</td>
<td>-0.11</td>
<td>-0.14</td>
<td>.03</td>
<td>-0.02</td>
<td></td>
</tr>
</tbody>
</table>

*p < .1  *p < .05  **p ≤ .001  a As measured by natural log-transformed RMSSD (ms)
Appendix D

Heart Rate Response to Cold Pressor for Whole Latino Sample

Time

Heart Rate (bpm)
Appendix E

RMSSD Response to Cold Pressor Task for Whole Latino Sample

[Graph showing RMSSD response over time with error bars for each condition.]
Appendix F

PEP Response to Cold Pressor Task for Latinos

Time

98 100 102 104 106 108 110

RT 2 CP 1 CP 2 CP 3 CP 4 Rec 1 Rec 2 Rec 3 Rec 4 Rec 5
Appendix G

Predictive Margins with 95% CIs

Predicted PEP response for participants who tolerated pain for 4 minutes.