

Pain tolerance as a mediator of aggressive behavior

By

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Research has shown that the experiences of pain and aggression are linked. Past research supports the notion that individuals with an aggressive history tend to have higher pain thresholds than their less aggressive counterparts. The aim of this study was to test the notion that past aggressive behavior is positively associated with higher pain tolerances, and that higher pain tolerance would be associated with the use of a clearly aggressive response on a laboratory task. Using data from a larger study on the neuroscience of human aggression ($N = 80$), a serial mediation model was tested using both objective and subjective indexes of pain tolerance as mediators. Results indicated that historic aggression was positively associated with both objective and subjective pain tolerance, and objective pain tolerance mediated the relationship between historic aggression and current aggression, whereas subjective pain tolerance did not.

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CHAPTER I
INTRODUCTION

General Introduction

Pain is a ubiquitous human experience. In 2011, at least 100 million American adults reported suffering from chronic pain conditions, with direct medical treatment and lost productivity costing approximately \$560-635 billion annually (Gaskin & Richard, 2012). The authors further suggest that these figures underestimate the full impact of pain as there are likely many acute experiences that go unreported and untreated. Pain is characterized by unpleasant physical and emotional experiences associated with tissue damage, either immediate or anticipated (Merskey & Bogduk, 1994). Pain can be chronic or acute, and it can coincide with other conditions, both physiological and psychological (Loeser & Melzack, 1999). Additionally, due to the subjective nature of pain, it is approached as a construct with great inter-individual variability (Fillingim, 2005) and can be affected by both situational and dispositional factors. For example, pain tolerance, which is thought to be a relatively stable dispositional or trait-like characteristic, is the maximum level of pain that a subject is willing to endure (Merskey & Bogduk, 1994). However, pain tolerance can be affected by environmental factors (Ewart, Elder, Laird, Shelby, & Walker, 2014), demographic variables (Woodrow, Friedman, Siegelau, & Collen, 1972), the experience of chronic pain (Kato et al., 2017), sleep disturbances (Rosseland, Pallesen, Nordhus, Matre, & Blågestad, 2018), and even cognitive processes (Litt, 1988; Wiech, Ploner, & Tracey, 2008).

Variations in the experience of pain can inform a number of social behaviors, such as aggression (Berkowitz, 1983, 1993).

For the purposes of this study, pain tolerance (PT) is defined as the maximum intensity of a painful stimulus that a person is willing to endure (Merskey & Bogduk, 1994). This is a separate, but related, construct to pain threshold, which is the minimum intensity of a stimulus that a participant would describe as painful. Furthermore, sensitization is defined as an increased responsiveness to pain-producing stimuli, and analgesia is the absence of a response in a situation that would normally be painful (Merskey & Bogduk, 1994).

Aggression refers to any behavior that is motivated by an intent to harm or injure another living being who would be motivated to avoid this behavior (Baron & Richardson, 1994). It is important to note that aggression is a behavior and not an emotion often associated with aggression (i.e., anger) or cognitions associated with aggression (i.e., hostility). For a discussion of the differences between aggression, anger, and hostility see (Baron & Richardson, 1994).

Measures of Pain

A wide variety of approaches to measure pain exist, including self-report scales, behavioral observation scales, and physiological responses. Numerous scales have been developed to assess pain, mainly assessing the intensity of pain on a single dimension (Breivik et al., 2008). Melzack and Torgerson (1971) proposed a series of pain descriptors and subtypes of pain. Their classification system includes three classes of pain (i.e., sensory, affective, and evaluative) as well as several subclasses (e.g., splitting, stinging, sharp) of pain. For the purposes of this study, a sensory (behavioral) and evaluative (self-rating) approach to pain associated with electric shock is used.

Experience of Pain

The experience of pain varies across individuals, and the subjective qualities of this experience remain largely unknown. In a study by Coghill, McHaffie, and Yen (2003), the authors attempted to identify objective neural correlates that corresponded with the differences in individuals' subjective reporting of pain. The authors administered thermal stimuli of varying temperatures to a group of seventeen adults. Following this, each adult provided subjective ratings of the perceived pain intensity using mechanical visual analogue scales while engaged in a functional imaging session. Using this information, the authors grouped individuals into high-, moderate-, and low-sensitivity groups. Results indicated that individuals who reported higher sensitivity to pain exhibited greater activation of the primary somatosensory cortex, anterior cingulate cortex, and prefrontal cortex than the individuals who reported lower sensitivity. These findings provide support for the idea that central nervous system correlates for the subjective pain experience exist. Other studies with similar procedures have explored the neural correlates of expectations of pain (Koyama, McHaffie, Laurienti, & Coghill, 2005) and placebo-induced changes in pain perception (Wager et al., 2004). For an extended discussion of this literature, see Coghill (2010).

Less research exists looking at the subjective evaluation of pain tolerance specifically. In a study by Hirsch and Liebert (1998), these researchers examined how expectations and labels may affect performance and modulate PT on a cold pressor task. Using a sample of college women, the authors varied the way in which they presented the temperature of the water to participants, with the three labels for the water being discomfort, pain, and vasoconstriction pain. PT was defined as the amount of time that the participant could keep their hand submerged. Results indicated that PT increased as the label of the water became more benign. Specifically,

participants were able to keep their hand submerged in the cold water for longer when it was associated with “pain” rather than “vasoconstriction pain,” and even longer when it was associated with “discomfort.”

Another study by Masedo and Rosa Esteve (2007) examined the role of coping strategies in the modulation of PT. Using a sample of 219 college-age volunteers and a cold pressor task, the authors assigned participants to one of three conditions to engage in a 20-minute intervention about coping skills. One group was an acceptance-based condition in which the participant was taught to “accept” any thoughts and sensations related to pain. A second group was a “spontaneous coping” condition in which the participant was educated on psychological factors (e.g., negative thoughts) that could be related to pain. Finally, a third group was a “suppression” condition in which the participant was instructed to suppress any thoughts and sensations related to pain. Results indicated that the group that had received the acceptance intervention had a significantly higher PT than the other groups, and the group that had received the suppression intervention had a significantly lower PT than the other groups. These findings suggest that PT can be altered by a cognitive intervention.

Acute Pain and Aggression

A rich literature exists on the effects of pain stimuli on aggression in lower animal species. One of the first demonstrations of the relation between pain and aggression was by O’Kelly and Steckle (1939), who tested several pairs of mice confined to a small cage. Electric shocks were administered through the cage floor via a wire grid. Following the administration of a shock, the mice engaged in nonspecific behaviors thought to reflect aggressive responding (e.g., biting, scratching), often continuing well after the shock terminated. Azrin, Hutchinson, and Hake (1963) replicated this finding with a higher mammalian species, specifically squirrel

monkeys. A shock-aggression effect was observed, but each pair of monkeys required different shock intensities and patterns to elicit aggression, implying that this relationship is much more complex than it appears to be in lower mammalian species. For an extended review of pain and aggression in lower animal species, see Berkowitz (1983).

The role of acute pain in human aggression has also been examined, but the literature is limited. An early study by Berkowitz, Cochran, and Embree (1981) investigated this relationship using a variation of a classic laboratory aggression task, the Buss Teacher-Learner paradigm, to examine how physical discomfort affects workplace performance observed in the laboratory. In this experiment, college-aged women were assigned to engage in a cold pressor task with water that was either moderately cold (18°C) or painfully cold (6°C). While the participant had her hands submerged, she was asked to assess another participant (a confederate) on her ability to answer business-related questions. The participant was allowed to award up to five punishments or five rewards to the confederate based on her performance. Half of the participants were told that the punishments would motivate the worker to perform better, and the other half were told that the punishments would be detrimental and potentially discouraging for the worker. Results showed that the participants in the painfully cold condition issued more punishments than participants in the moderately cold condition; within the painfully cold condition, participants who were told that the punishments would cause harm issued the most punishments of all.

This study included only women participants, but a follow up study by Berkowitz and Embree (1987) generally replicated these results in men. This latter study had a similar design as the previous one, but participants were also assigned to either a condition where they were explicitly permitted to end the cold pressor task at will or a condition where they were encouraged to persist. Participants who had greater control over the termination of the task

reported experiencing less feelings of anger and found the cold water less aversive than participants who were encouraged to keep engaging. However, participants in this former group were more aggressive to their partners than participants in the latter group. These results appear inconsistent with previous reports that higher aversiveness should lead to higher aggression. The authors explain these findings by suggesting that increased aversiveness may not be the sole determinant of increased aggression, suggesting that a reflexive response to pain cannot completely explain aggressive responding in humans.

Other processes, including cognitive appraisals, may modulate aggressive responding. Anderson, Anderson, Dill, and Deuser (1998) explored this idea in a study by measuring participants on trait hostility and then assigning them to one of two conditions: A “pain” condition in which they held an arm at an uncomfortable angle for a prolonged period of time or a no-pain condition in which they supported their arm on a desk. Participants then rated word pairs on their similarity, with some words that were aggression-related and others that were aggression-unrelated. To assess hostile cognitions, participants completed the State Hostility Scale post task (Anderson, Deuser, & DeNeve, 1995).

Results indicated that participants who reported higher trait hostility found more aggressive meaning in both aggressive and ambiguous word pairs. Furthermore, participants in the pain condition reported greater state hostility than participants in the no-pain condition. Although measures of hostile cognition are not directly indicative of aggressive behavior, these results lend support for the idea that the pathways between pain and aggression are complex, and that the relationship with aggressive behavior could be modulated by the individual differences in perception of environmental stimuli. The literature on the acute pain-aggression relationship generally suggests that more intense immediate pain predicts an aggressive response, but these

studies do not account for individual differences in pain tolerance. Indeed, it is possible that individuals with a higher pain tolerance will behave more aggressively when attacked or provoked compared to individuals with a low pain tolerance. Specifically, individuals with a high pain tolerance might experience less pain when during an aggressive interaction, both while administering and receiving punishment (thus failing to inhibit aggression).

Individual Differences in Aggression and Pain Tolerance

Indirect evidence for the relation between pain experiences and aggression comes from pharmacological studies of aggression using substances with antinociceptive properties. For example, alcohol has been shown to have analgesic effects (Thompson, Oram, Correll, Tsermentseli, & Stubbs, 2017), and its effect on aggression has been examined extensively both in field and laboratory studies. Bushman and Cooper (1990) conducted a meta-analysis of 30 experimental studies looking at the relationship between alcohol and aggression accounting for various differences in study design (e.g., placebo versus veridical control drink conditions). Overall, a small effect size ($d = .25$) emerged for alcohol versus control and a medium effect ($d = .61$) for alcohol versus placebo.

Analgesic drugs used in medical practice have also been examined in human studies of aggression. For example, Berman, Taylor, and Marged (1993) administered either 45 mg of morphine or a placebo to twenty-eight male college-aged adults. Participants then engaged in a series of reaction time trials with a fictitious opponent, with the participant being told that the winner of each trial would have an electric shock chosen before each trial delivered to his opponent with intensity of his choosing (on a scale of 1 to 10). Participants completed a total of 21 trials, and mean shock level across trials was calculated. Results indicated that participants in the morphine condition were more aggressive in their initial responses compared to participants

in the placebo group and responded more aggressively throughout the experiment.

Benzodiazepines, a class of tranquilizer with mild analgesic effects, demonstrated a similar role in a meta-analysis by Albrecht et al. (2014).

Pharmacologic studies of drugs with analgesic effects on aggression suggest that experimentally manipulated changes in PT facilitates aggression. Similarly, individual differences in PT could be expected to be related to aggression differentially. Few studies on PT set point and aggression have been conducted. In one study, PT was operationalized in men by administering shocks of gradually increasing intensity until the participant deemed the shock too painful to continue (Niel, Hunnicutt-Ferguson, Reidy, Martinez, & Zeichner, 2007). Once this procedure was complete, the participant competed against a fictitious opponent on a series of competitive reaction time trials and could administer an electric shock as “punishment” to their opponent, regardless of whether they won or lost the reaction time trial. The chosen shock levels were determined by the participant by selecting one of ten buttons, with the buttons labeled from 1 through 10 (ranging from 55 to 100 percent of the ostensible opponent’s shock tolerance). Results revealed a modest positive relationship between PT and aggression ($r = .23$). This study was partially replicated with a more gender-balanced sample of 195 undergraduate students (67% women) and a self-rating (not behavioral) measure of trait aggression (Reidy, Dimmick, MacDonald, & Zeichner, 2009). The authors measured PT using a modified method of limits task similar to the one used in the study by Niel et al. (2007). Results indicated that trait aggression was positively associated with PT in men but not women. Note that Niel et al. (2007) measured current aggressive behavior with a laboratory task whereas Reidy et al. (2009) used a measure of trait aggression. In addition, the highest shock available in Niel et al. to deliver to the opponent was equivalent to the pain threshold, and mean shock selections were used as an index

of aggression. Given that it difficult to infer aggressive intent (i.e., the delivery of a shock intended to harm) from this index, other researchers have employed a “severe” or “extreme” shock option that is ostensibly twice the opponent’s pain threshold (but in actuality is never delivered, see Berman et al., 2009 for an example).

Another line of evidence for heightened pain tolerance and aggression comes from studies of individuals diagnosed with Intermittent Explosive Disorder (IED). IED is the one diagnostic category in the DSM-V (American Psychiatric Association, 2013) for which aggression is a cardinal feature and is characterized by persistent patterns of aggressive behavior that represent a heightened sensitivity to actual or perceived provocation. Previous research has validated IED as a distinct category (Ahmed, Green, McCloskey, & Berman, 2010) with a lifetime prevalence rate of 7.3% (Kessler et al., 2006). For reviews of IED and its associated criteria, see Coccaro (2011, 2012).

Few studies have examined the relationship between IED and pain. One study investigated the prevalence of IED diagnoses in patients reporting “...mixed chronic pain...” (p. 183) and found that almost 17 percent of men and two percent of women met DSM-III criteria for IED (Fishbain, Goldberg, Meagher, Steele, & Rosomoff, 1986). It should be noted that this sample reported other psychiatric diagnoses at elevated rates which may represent higher levels of psychopathology overall. For a review of psychiatric comorbidities in chronic pain patients, see Fishbain, Cutler, Rosomoff, and Rosomoff (1998). Another study compared individuals with and without IED on several different health outcomes (McCloskey, Kleabir, Berman, Chen, & Coccaro, 2010). Results indicated that IED was associated with higher rates of neck/back pain, headaches, and other chronic pain.

Life History of Aggression and Laboratory Measures of Aggression

Aggressive tendencies are thought to be relatively stable across the lifespan (Huesmann & Eron, 1989). For example, Olweus (1979) conducted a review of 16 longitudinal studies examining aggression in men of varying ages. The author's findings indicate that aggression appears to be a stable construct across time, with similar results across different methods of measurement. A more recent study (Kokko, Pulkkinen, Huesmann, Dubow, & Boxer, 2009) examined different forms of adult aggression across time in both men and women ($N = 856$; 436 men and 420 women) and found that a history of aggression predicted physical aggression in adulthood.

Given that aggressive behaviors appear somewhat stable across the lifespan, it is reasonable to assume that individuals high in trait aggression or who have a documented history of aggression would exhibit high levels of aggression on behavioral tasks designed to assess aggression under controlled laboratory conditions. Several studies have been conducted examining the validity of laboratory measures using samples drawn from populations associated with aggressive traits and histories. For example, Giancola and Parrott (2008) evaluated the validity of the inferences that can be drawn from the Taylor Aggression Paradigm (TAP). Participants completed several self-report personality questionnaires that assessed physical aggression, verbal aggression, anger, and hostility. Aggressive performance on the TAP was indexed in several ways, and all indices were associated with all four forms of aggression, most strongly with physical aggression.

A meta-analysis by Bettencourt, Talley, Benjamin, and Valentine (2006) found associations between personality variables derived from the five-factor model of personality (Costa & McCrae, 1992) and aggression, and compared these associated variables (i.e.,

dissipation-rumination, emotional susceptibility, impulsivity, irritability, narcissism, trait aggressiveness, trait anger, and Type A personality) to laboratory measures of aggression. Specifically, the authors examined the aggressive behavior of participants who scored low on these personality variables compared to participants who scored high across a total of 63 experimental studies. These studies involved the use of different types of overt aggression (i.e., physical, verbal, and monetary penalty), targets of aggression, options to aggress, age, and gender. Results indicated that, in general, individuals who scored high on the previously mentioned personality variables exhibited more aggressive behavior in the laboratory than those who scored low. Specifically, fixed-effects analyses indicated that trait aggressiveness and trait irritability predicted aggressive behavior in both neutral and provoking conditions, whereas trait anger, type A personality, dissipation-rumination, emotional susceptibility, narcissism, and impulsivity predicted aggressive behavior in provoking conditions alone.

Current Study

Aims of Study

Based on the literature reviewed, it appears that individual differences in pain tolerance as a function of past aggression might serve as a mechanistic explanation for aggressive behavior observed prospectively. If the relationship holds, this would provide evidence for the role of aggression-associated pain tolerance in future aggressive acts. To date, it appears this possibility has not yet been tested, particularly under controlled laboratory conditions. Thus, the first aim of the study was to examine whether a life history of aggression is positively associated with objective and subjective appraisal of pain tolerance. The second aim of this study was to examine whether the two pain tolerance indexes mediate the relationship between past aggressive acts and aggression observed prospectively in the laboratory using a clear index of aggressive intent.

Finally, as an exploratory analysis, the serial mediational model shown in Figure A1 will provide information about whether the mediating effects of pain tolerance emerge through the subjective evaluation of pain tolerance, an objective index of pain tolerance, or both.

It was predicted that life history of aggression would be positively associated with both objective and subjective pain tolerance. Second, it was predicted that both pain tolerance indices would mediate the relation between past and current aggression observed in the laboratory. Finally, the mediational path from pre-task objective pain tolerance to post-task self-evaluation of pain was explored, and no a priori predictions were made.

CHAPTER II

METHOD

Participants and Data

For the current study, archival data were retrieved from a dataset involved in a larger study on the neuroscience of aggression (see Berman, McCloskey, Fanning, Schumacher, & Coccaro, 2009). The sample consisted of 38 women and 42 men ($N = 80$), ages 18 through 48 years ($M = 24.33$, $SD = 7.27$). “Healthy volunteers” were recruited from the community and were compensated for their time for a study on “personality and psychomotor skills and reaction time.” The recruitment strategy oversampled individuals with a notable history of aggressive behavior by recruiting people “with a short fuse” in order to provide a range of responses with respect to past aggressive acts (see Berman et al., 2009, for details on the recruitment process and detailed characteristics of the sample). The current study employed de-identified data and was therefore exempt from Institutional Review Board review by Mississippi State University (IRB-19-321).

Materials and Procedure

The Life History of Aggression Scale

Participants completed a battery of assessments, one of which was the Life History of Aggression Scale (LHA; Coccaro et al., 1997). The LHA is a semistructured interview consisting of three different subscales (Aggression, Social Consequences and Antisocial Behavior, and Self-directed Aggression). The Aggression Sub-scale (AG) of the LHA was used in the current

study to assess aggressive history and consists of five items: Temper outbursts, physical fighting, verbal aggression, assaults, and aggression towards objects. Items were rated for frequency using scores of 0 (*no events*), 1 (*one event*), 2 (*“a couple” or “a few,” i.e. 2-3 events*), 3 (*“several” or “some,” i.e. 4-9 events*), 4 (*“many” or “numerous,” i.e. 10+ events*), and 5 (*“so many events that they can’t be counted”*). Cronbach’s alpha for the AG in this study was .87.

Objective Pain Tolerance (OPT)

To measure OPT, fingertip electrodes were attached to the middle and index fingers of the participant’s non-dominant hand and one second shocks of increasing intensity were administered in intervals of 100- μ A. The participant noted when they first felt the shock and then again when the shock was “definitely painful,” described as being so intense that they asked that the threshold procedure be terminated. This marked the end of the OPT procedure. OPT was operationalized as the level of current associated with this maximum value measured in μ A. A ceiling of 2.5 mA was used to avoid any potential injury. This procedure was repeated for a fictitious “opponent” ostensibly in an adjoining room, using audio-recorded gender matched voices to simulate the threshold procedure, and overheard by the participant through an intercom system. No feedback about the opponent’s OPT was given to the participant beyond hearing the opponent asking to terminate the procedure when the shock became too painful.

Subjective Pain Tolerance

Perception of the pain threshold was rated post-task (“How uncomfortable was the highest shock you received?”) from 1 (*Not at All*) through 8 (*Very Much*).

The Taylor Aggression Paradigm

The Taylor Aggression Paradigm (TAP; Taylor, 1967) is a classic laboratory task designed to simulate a human aggressive interaction. In the TAP, a participant competes against a fictitious opponent on a simple reaction time test during which electrical shock is both given and received. The participant is informed that the person who “loses” the reaction time test will be administered a shock with intensity as determined by the “winner.” At the beginning of each trial, the participant chooses a shock level from 0-10 or 20. The shock intensity is set according to the individual’s OPT: a level 10 shock is 100 percent of the OPT, a level 9 shock is 95 percent of the OPT, a level 8 shock is 90 percent of the OPT, and so forth. The level 0 option administers no shock and is included to increase ecological validity. The participant is informed that the level 20 shock is twice the intensity of the level 10 shock rated as painful by both the participant and fictitious opponent. After the participant selects the shock level, the shock selected by the “opponent” is shown on a computer monitor. The order of wins and losses, as well as the shocks “selected” by the opponent, are predetermined and computer controlled. Aggressive behavior is operationalized as the number of 20 shocks that the participant tries to administer over the course of 28 reaction-time trials. Evidence for the validity of the inferences that can be drawn from the TAP comes from a meta-analysis by Anderson and Bushman (1997) and a study by Giancola and Parrott (2008).

Procedure

On an initial visit, the participant completed a battery of assessments, one being the LHA. On their next visit (1-4 weeks later), the participant’s (and “opponent’s”) OPT was determined, followed by the TAP. After receiving tasks instructions, the participant completed 28 total reaction time trials divided into four blocks of 6 trials with a transition trial before each block. To

simulate provocation, the “opponent” was programmed to deliver shocks with averages of 2.5, 5.5, 8.5, and 8.5 across the four blocks respectively. The trials before each block are used to smooth the transition between blocks. For the transition trial before block 4, the opponent sets a shock of level 20 and loses, ensuring that the participant never receives a shock greater than the OPT. The task is programmed so that the participant “wins” 50% of the trials. After the TAP, the participant rated his or her perception of the pain associated with the pain threshold determined before the TAP.

CHAPTER III

RESULTS

Descriptive Statistics

Participants' scores on the LHA Aggression subscale ranged from 0 to 22 ($M = 9.73$, $SD = 6.53$). Their objective pain tolerance ranged from 0.23 to 2.47 milliamperes ($M = 1.21$, $SD = 0.75$), and their self-evaluation of pain tolerance ranged from 1 to 8 ($M = 4.46$, $SD = 2.08$). The extreme shock variable was logarithmically transformed to account for skew. Twenty-four participants selected the extreme shock at least once during the TAP (30% of sample), with the total number of extreme shocks ranging from 1 to 14.

Bivariate Correlations

LHA scores were positively correlated with objective pain tolerance ($r = .40$, $p < .001$) and total extreme shocks ($r = .23$, $p = .042$) and were negatively correlated with self-evaluation of pain tolerance ($r = -.32$, $p = .004$). Objective pain tolerance was positively correlated with total extreme shocks ($r = .39$, $p < .001$) and negatively correlated with self-evaluation of pain tolerance ($r = -.29$, $p = .008$). Total extreme shocks were not significantly correlated with self-evaluation of pain tolerance ($p = .069$). See Table B1 for these correlations.

Mediation Analyses

IBM SPSS PROCESS macro, Version 3.3, Model 6 (Hayes, 2017) was used to conduct a serial mediation analysis with objective pain tolerance and self-evaluation of pain tolerance as

possible mediators of the relationship between LHA scores and total number of extreme shocks selected during the TAP. The model (see Figure A1) was also used to explore whether self-evaluation of pain tolerance intervenes in the relationship between objective pain tolerance and extreme shocks (objective pain tolerance → self-evaluation of pain tolerance → extreme shock use). Therefore, objective pain tolerance was included as the first mediator, and self-evaluation of pain tolerance was included as the second.

The overall regression model with total extreme shocks regressed on LHA scores, objective pain tolerance, and self-evaluation of pain tolerance was significant, $R = .41$, $F(3, 76) = 5.05$, $p = .003$. Objective pain tolerance ($b = .0002$, 95% CI [.0001, .0004]) significantly predicted extreme shocks, but LHA scores ($b = .005$, 95% CI [-.013, .024]) and self-evaluation of pain tolerance ($b = -.022$, 95% CI [-.082, .037]) did not. See Table B2 for model results.

The total effect of LHA on total extreme shocks was significant ($b = .018$, 95% CI [.001, .035]), indicating that LHA predicted the total number of extreme shocks when the mediators were not considered in the model. However, as noted above, the direct effect of LHA on total extreme shocks was not significant when the mediators were included in the analysis.

The model provided three different indirect effects calculated using 5000 bootstrap samples. These were estimated as the product of the regression coefficients along the three different pathways between the independent (X) and dependent (Y) variables, through each mediator individually as well as combined. The first indirect effect was estimated from a_1b_1 , indicating that the standardized indirect effect of objective pain tolerance on the relationship between LHA and total extreme shocks (LHA → objective pain tolerance → total extreme shocks) was significant ($\beta = .14$, 95% CI [.04, .25]). That is, participants' objective pain

tolerance increased as a function of their LHA scores, and this higher pain tolerance was associated with more extreme shock selections.

The next indirect effect was estimated from a_2b_2 , indicating that the standardized indirect effect of self-evaluation of pain tolerance on the relationship between LHA and total extreme shocks (LHA → self-evaluation of pain → total extreme shocks) was not significant ($\beta = .02$, 95% CI [-.04, .09]). Therefore, LHA did not predict the participants' self-evaluation of their pain tolerance, which in turn did not predict the number of extreme shocks selected.

The serial indirect effect was estimated from $a_1d_1b_2$, indicating that the standardized indirect effect of both objective pain tolerance and self-evaluation of pain tolerance on the relationship between LHA and total extreme shocks (LHA → objective pain tolerance → self-evaluation of pain → total extreme shocks) was also not significant ($\beta = .007$, 95% CI [-.01, .04]). Thus, objective pain tolerance did not influence how participants evaluated their own rating of pain tolerance when the relationship between LHA and total extreme shocks is considered.

Post-hoc Power Analysis

A post-hoc power analysis was conducted in order to better understand if the regression model was appropriate for detecting the predicted effects. A Monte Carlo power analysis was conducted within RStudio Version 1.1.414 using an app developed by Schoemann, Boulton, and Short (2017). The sample size of 80 was used with a confidence level of 95%. The standardized regression coefficients for each pathway reported above were used to conduct the analysis. Results indicated that the analysis of the first indirect effect (a_1b_1) was powered to a value of .96. The second indirect effect (a_2b_2) was powered to a value of .04. Finally, the third indirect effect

$(a_1d_1b_2)$ was powered to a value of .02. These results indicate that the analysis of the indirect effect of objective pain tolerance on the relationship between LHA and total extreme shocks was appropriately powered for this study, but the other indirect effects may not have been.

CHAPTER IV

DISCUSSION

General Discussion

These results provide support for the idea that a positive relationship exists between a life history of aggression and current aggression demonstrated in a controlled laboratory setting, which is consistent with previous research (Coccaro, Berman, & Kavoussi, 1997). Higher LHA scores significantly predicted more aggressive TAP behavior. Furthermore, pain tolerance appeared to mediate this relationship. Although self-evaluation of pain tolerance was significantly correlated with both LHA scores and objective pain tolerance, it did not significantly mediate the relationship between LHA and TAP performance. Furthermore, the serial mediation model through both objective pain and self-evaluation of pain was not significant.

It was predicted that life history of aggression would be positively associated with both objective and subjective pain tolerance. The results supported this prediction—LHA scores were correlated with both objective pain tolerance and subjective evaluation of pain tolerance. The more aggressive an individual reported their history to be, the higher their objective pain tolerance was, and the less discomfort they would report from the worst shock that they experienced. This interpretation follows the literature; aggression is positively associated with pain, both situationally and dispositionally. However, the literature is limited in how a history of

actual aggressive acts are associated with self-reported pain tolerance, which appears to be a novel finding.

Second, it was predicted that both pain tolerance indices would mediate the relation between past and current aggression observed in the laboratory. This prediction was partially supported. Objective pain tolerance mediated the relationship between LHA scores and TAP performance. Specifically, it is possible that a history of aggressive behavior leads to higher levels of objective pain tolerance, and this increased pain tolerance leads to more aggressive performance on the TAP. However, this effect was not significant for self-evaluation of pain. Higher LHA scores predicted lower ratings of discomfort from extreme shock, but these ratings did not predict TAP performance. This finding contradicts the prediction, possibly because the mechanism through which one evaluates their own pain may not be the same as the mechanism through which they experience objective pain. Other explanations could involve social factors such as masculinity/femininity, where one could be motivated to describe their pain tolerance in ways that are traditionally more socially desirable (Reidy et al., 2009). Additionally, there could have been both sensitization and habituation effects throughout the course of TAP that would change one's response to how they evaluate their pain tolerance after the task.

Finally, the mediational path from pre-task objective pain tolerance to post-task self-evaluation of pain was explored, for which a priori predictions were not made. Results indicated that this path was not significant. That is, higher LHA scores predicted higher objective pain tolerance, but this pain tolerance did not affect how participants evaluated their pain tolerance, which in turn did not affect aggressive TAP performance. This could be a result of these pain tolerance indices operating through different mechanisms, as noted above. However, this analysis also accounts for how objective pain tolerance may affect self-evaluation of pain

tolerance, and although these measures are correlated, it does not appear that objective pain tolerance predicts self-evaluation when entered into this model. This could be due to LHA scores predicting both objective pain tolerance and self-evaluation of pain, accounting for shared variance between these two pain indices. However, given that this analysis could benefit from increased power, this lack of an effect could also be an artifact of a relatively low sample size.

Strengths and Limitations

Though these findings present several interesting relationships to explore in future studies, the current study design is limited by several factors. First, the analysis was not powered appropriately enough to find effects beyond the simple mediation model seen for objective pain tolerance. Future studies, however, can use the effect sizes reported for a priori power calculations to determine required sample sizes.

Next, the cross-sectional nature of these data should be noted. Specifically, the developmental trajectory and relationship of aggression and pain cannot be addressed. Thus, it could be that an increased pain tolerance that may have been present from an early age allows an individual to enter into more aggressive situations without as great a fear of harm (e.g., “taking a punch”). It could also be that heightened pain tolerance leads individuals to behave in ways that are not considered aggressive by themselves but may be perceived by others as aggressive, eliciting an aggressive interaction. In sum, the current study does not aim to determine directionality, as aggression could be a precursor to pain tolerance, but a biological predisposition to higher pain tolerance could also lead to aggression.

Objective pain tolerance as a possible predictor of self-evaluation was most appropriate for this analysis, as the pain tolerance procedure took place pre-task, and the self-evaluation questionnaire was post-task. However, it is just as feasible that one’s appraisal of their own pain

tolerance affects actual pain tolerance. It would therefore be valuable in future studies to include multiple measures of pain tolerance both pre- and post-task. Finally, it should be noted that participants evaluated their pain tolerance using a single-item measure. Future studies would benefit from a more robust measure of self-reported pain—for example, the Pain Sensitivity Questionnaire (Ruscheweyh, Marziniak, Stumpfenhorst, Reinholz, & Knecht, 2009).

Future Directions

Despite these limitations, several tentative clinical applications are reasonable to discuss from the current findings. Assuming pain tolerance is partially determined early in life, developing pain self-awareness interventions that could be administered in a classroom setting could reduce future aggressive acts as well as unintentional injuries. Assuming pain tolerance develops later as a function of aggression, including pain awareness mindfulness techniques in aggressive adults might be useful in anger management packages. Additionally, it may be worthwhile to assist these individuals in becoming aware of pain experienced by others and fostering an understanding that not all individuals experience pain similarly.

Overall, this study provides preliminary evidence for the notion that dispositional pain plays a role in the inception and maintenance of aggression. Given the wealth of literature demonstrating the powerful association between pain and aggression, it stands to reason that the nature of this relationship should be expounded further. Future studies can expand on the present findings with laboratory studies on clinical or community samples or with observational studies in the field.

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APPENDIX A
MODEL OF SERIAL MEDIATIONAL ANALYSIS

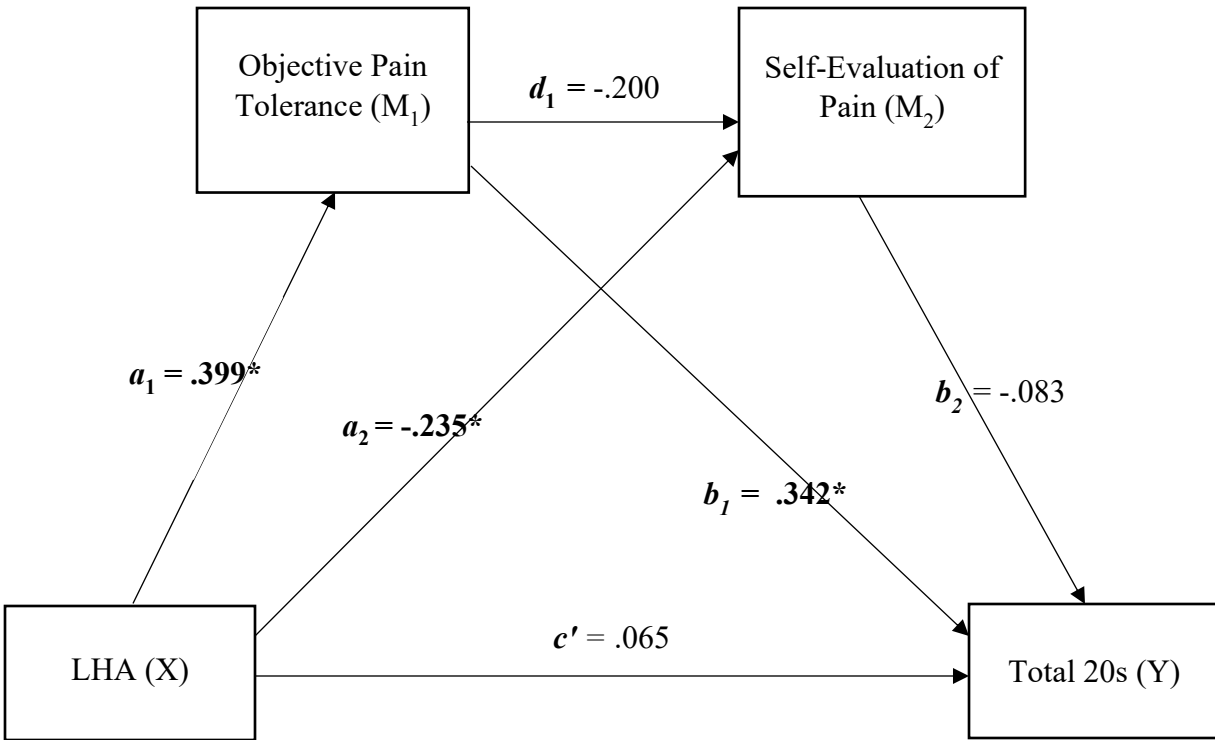


Figure A1. Model of serial mediational analysis of objective pain tolerance and self-evaluation of pain as mediators of the relationship between LHA and Total 20s with standardized coefficients included.

APPENDIX B
CORRELATIONS AND REGRESSION TABLES

Table B1

Bivariate Correlations between LHA, Objective Pain Tolerance, Self-Evaluation of Pain, and Total 20s during the TAP

Variables	1	2	3	4
LHA				
Objective Pain Tolerance	.399*			
Self-Evaluation of Pain	-.315*	-.294*		
Total 20s	.227*	.393*	-.204	

*Note: Correlation is significant at the .05 level (2-tailed)

Table B2

Regression Coefficients, Standard Errors, P-Values, and Model Summary Information for the Serial Mediation Model

	M ₁ (Objective Pain Tolerance)				M ₂ (Self-Evaluation of Pain)				Y ("Extreme" Shocks)						
	<i>b_i</i>	<i>SE</i>	<i>p</i>	95% CI	<i>b_i</i>	<i>SE</i>	<i>p</i>	95% CI	<i>b_i</i>	<i>SE</i>	<i>p</i>	95% CI			
	X (LHA)	<i>a</i> ₁	46.03	11.99	< .001	[22.16, 69.89]	<i>a</i> ₂	-.07	.03	.045	[-.138, -.002]	<i>c</i> '	.0051	.0092	.583
M ₁	-	-	-	-	<i>d</i> ₁	-.0005	.0003	.087	[-.0011, .0001]	<i>b</i> ₁	.0002	.0001	.004	[.0001, .0004]	
M ₂	-	-	-	-	-	-	-	-	-	<i>b</i> ₂	-.0221	.0298	.461	[-.082, .037]	
Constant	<i>i</i> _{M1}	764.38	140.17	< .001	[485.33, 1043.43]	<i>i</i> _{M2}	6.13	.43	< .001	[5.27, 6.99]	<i>i</i> _y	.7345	.2150	.001	[.306, 1.163]
				<i>R</i> ² = .16, <i>MSE</i> = 484535.33, <i>F</i> (1, 78) = 14.74, <i>p</i> < .001				<i>R</i> ² = .13, <i>MSE</i> = 3.35, <i>F</i> (2, 77) = 5.92, <i>p</i> = .004				<i>R</i> ² = .17, <i>MSE</i> = .23 <i>F</i> (3, 76) = 5.05, <i>p</i> = .003			

APPENDIX C

HUMAN RESEARCH PROTECTION PROGRAM APPROVAL LETTER



NOTICE OF DETERMINATION FROM THE HUMAN RESEARCH PROTECTION PROGRAM

DATE: September 03, 2019
TO: Mitchell Berman, Ph.D., Psychology, Andreana Durham;Emily Stafford;Matthew Timmins;Michael Nadorff;Michaela Patoilo;Richard Nelson;Suzanne Amadi
Andreana Durham, Current Honors Student, Psychology, Emily Stafford, Psychology, Matthew Timmins, MS, Psychology, Michael Nadorff, PhD, Psychology, Michaela Patoilo, BS, Psychology, Nathan Barclay, BS, Psychology, Richard Nelson, MS, Psychology, Suzanne Amadi, MS, Student Counseling Services
PROTOCOL TITLE: Pain perception and aggression
PROTOCOL NUMBER: IRB-19-321
Approval Date: September 03, 2019 Expiration Date: September 02, 2024

EXEMPTION DETERMINATION

The review of your research study referenced above has been completed. The HRPP had made an Exemption Determination as defined by 45 CFR 46.101(b)4. Based on this determination, and in accordance with Federal Regulations, your research does not require further oversight by the HRPP.

Employing best practices for Exempt studies is strongly encouraged such as adherence to the ethical principles articulated in the Belmont Report, found at www.hhs.gov/ohrp/regulations-and-policy/belmont-report/# as well as the MSU HRPP Operations Manual, found at www.orc.msstate.edu/humansubjects. As part of best practices in research, it is the responsibility of the Principal Investigator to ensure that personnel added after this Exemption Determination notice have completed IRB training prior to their involvement in the research study. Additionally, to protect the confidentiality of research participants, we encourage you to destroy private information which can be linked to the identities of individuals as soon as it is reasonable to do so.

Based on this determination, this study has been inactivated in our system. This means that recruitment, enrollment, data collection, and/or data analysis CAN continue, yet personnel and procedural amendments to this study are no longer required. If at any point, however, the risk to participants increases, you must contact the HRPP immediately. If you are unsure if your proposed change would increase the risk, please call the HRPP office and they can guide you.

If this research is for a thesis or dissertation, this notification is your official documentation that the HRPP has made this determination.

If you have any questions relating to the protection of human research participants, please contact the HRPP Office at irb@research.msstate.edu. We wish you success in carrying out your research project.

Review Type: EXEMPT
IRB Number: IORG0000467