Awe as a Psychological Analgesic to Acute Physical Pain

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Abstract

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Awe as a Psychological Analgesic to Acute Physical Pain

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Psychological analgesics show great prowess for reducing pain, as supported theoretically with the gate control theory of pain, and as demonstrated in response to laboratory experimental pain paradigms in which psychological components (e.g., positive affect) are manipulated and acute pain responses are attenuated. The current research investigates whether a unique form of positive affect—awe—provides analgesic benefits to participants enduring a laboratory-based, acute pain paradigm (i.e., the cold pressor task; CPT) given previous evidence that positive affect, nature (a frequent elicitor of awe), and other psychological manipulations reduce acute pain. The primary hypotheses were that participants in an awe-manipulation condition (vs. control) would report less pain in response to the CPT, endure the CPT for a longer duration before pain threshold was reached, endure the CPT for a longer duration before pain tolerance was reached, and have lower heart rate, systolic blood pressure, and diastolic blood pressure reactivity to the CPT. I found that participants in the awe (vs. control) condition did not: report less pain, endure the CPT longer before pain threshold or tolerance, or have lower reactivity in heart rate, mean systolic blood pressure, or diastolic blood pressure. Despite no evidence for primary hypotheses, ancillary analyses revealed two notable findings. Specifically, trait awe interacted with experimental condition to predict duration until pain threshold and diastolic blood pressure reactivity during the first minute of the CPT.

Individuals low in trait awe experienced shorter durations until threshold in the awe (vs. control) condition and individuals high in trait awe had greater DBP reactivity during the first minute of the CPT in the awe (vs. control) condition. Together, current study findings suggest that awe may not serve as an analgesic. Accordingly, the findings point toward the need for vigilance in psychological analgesic implementation within multimodal pain treatment.

Table of Contents

Page

List of Tables

Page

List of Figures

Page

Awe as a Psychological Analgesic to Acute Physical Pain

The International Association for the Study of Pain (IASP) defines pain as the unpleasant sensory and emotional experience associated with actual or potential tissue damage (Raja et al., 2020). In addition, pain can be categorized as acute or chronic. Acute pain is pain that serves as an adaptive alarm. Acute, painful stimuli elicit elevation in blood pressure and heart rate, indicative of sympathetic nervous system activation (Millan, 1999). Acute pain also elicits affective and behavioral responses for avoiding the source causing tissue damage or to protect affected tissue from further harm (Auvray et al., 2010; Fernandez & Turk, 1992; Millan, 1999). Further, acute pain is considered to be of recent onset (i.e., defined as present no longer than 3 months) and usually can be attributed to a specific injury or disease (Michaelides & Zis, 2019). Unlike acute pain, chronic pain—pain that persists beyond healing of damaged tissue (Stålnacke, 2011) — serves no adaptive value but is rather maladaptive (Millan, 1999). Individuals with chronic pain often experience other, pain-related negative outcomes, such as disabled physical and social functioning (Zale et al., 2013), increased depression and anxiety (Lerman et al., 2015), and increased risk of suicidality (Ilgen et al., 2008; Von Korff et al., 2005). Without resolution of acute pain through tissue healing, acute pain may become chronic (Loeser & Melzack, 1999; Tompkins et al., 2017). Thus, acute pain is essential for avoiding potentially lethal tissue damage, but it is also important to adequately treat acute pain to prevent the development of chronic pain conditions.

Despite having adaptive value, people try to avoid pain because it is an unpleasant, subjective, physiological experience. Pain is widely accepted as one of the

most common reasons patients seek medical care (Loeser & Melzack, 1999; Radnovich et al., 2014). Pain treatment can reduce the intensity and duration of an injury and shorten the healing process (Loeser & Melzack, 1999). Further, treating acute pain also reduces psychological and physiological after-effects of acute pain, including the risk for development of persistent (i.e., chronic) pain (Daniel, 2008). However, currently available pain treatments are often ineffective at reducing pain (Sinatra, 2010). Thus, pain largely remains inadequately treated, often causing negative health effects, such as reduced quality of life, sleep disturbances, and impaired physical function (Fine, 2001; Majedi et al., 2019; Sinatra, 2010).

Often, the first option chosen to treat pain is pharmacological treatment, with opioids regularly used to treat moderate to severe acute pain (Sinatra, 2010). Though opioids may be effective at treating pain, opioid treatment is quite problematic. Most importantly, treating moderate to severe acute pain with opioids is accompanied by an increased risk of mortality. In 2008, unintentional overdoses of opioids consumed for pain relief were associated with 14,800 deaths in the U.S. (Centers for Disease Control and Prevention, 2011). Further, opioid use for pain relief is accompanied by various other side effects. In a study of 50 post-abdominal-surgery participants (Gan et al., 2004), 96% of respondents indicated they experienced side effects from their opioid pain medications, with 40% of respondents indicating that at least one side effect was severe. Side effects included constipation, mental cloudiness/dizziness, itching, nausea, vomiting, sleep disorders, nightmares/hallucinations, and mood changes/alterations. These potential adverse side effects and the risk of death often motivate patients to discontinue opioid

therapy, interfere with physicians' ability to dose opioids to maximum analgesia, and make physicians reluctant to prescribe opioids in the first place (Sinatra, 2010). As a result, acute pain often ends up being undertreated when opioid therapy is the treatment of choice.

Opioids remain the primary analgesic treatment for acute pain; however, more recently, pharmacological multimodal approaches have gained traction to provide significant benefits to individuals experiencing moderate to severe acute pain (Sinatra, 2010). Nonopioid analgesic drugs also allow for effective pharmacological pain control. These analgesics include nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen (White, 2017). Combining drugs with different mechanisms of action likely provides additive effects for controlling pain (White, 2005). Though combinations of nonopioid analgesics may allow pain control comparable to opioids without the problematic side-effects of opioids, many nonopioid analgesics also have their own risks and limitations. For example, NSAIDs increase the risk of gastrointestinal (GI) bleeding, myocardial infarction (i.e., heart attack), and stroke (Davis & Robson, 2016). Similarly, chronic, heavy use of acetaminophen may result in severe, often fatal, liver damage (Barker et al., 1977). Thus, these drugs come with their own risks, motivating pain researchers to explore non-pharmacological options for pain relief.

Psychological Reduction of Acute Pain

To increase the effectiveness and safety of analgesic treatment, more recent 'multimodal' pain relief techniques have included psychological interventions to reduce pain. Such approaches are often theoretically well-founded. Melzack and Wall's (1965)

gate control theory of pain posits that inputs from the central nervous system reduce or even override painful, nociceptive stimulation in the spine. These central inputs may be psychological in nature and include cognitive, behavioral, and emotional factors. The gate control theory of pain was the first pain theory to propose that higher brain centers and thus, by extension, psychological factors may affect pain signaling (Keefe et al., 1996). Subsequent research has demonstrated that psychological processes play an important role in the subjective experience of pain (e.g., Daniel, 2008; Peck, 1986; VanDalfsen & Syrjala, 1990). Thus, original models focusing mostly on eliminating pain through improved pathology or blocking pain pathways have now evolved into multi-modal theoretical frameworks that rely heavily on psychological factors (Daniel, 2008). Common psychological interventions against acute pain include the following: psychoeducation, relaxation, psychological distancing, distraction, nature exposure, and positive affect inductions.

Psychoeducation

Early research on psychological interventions focused on psychoeducation, often in pre-operative and post-operative settings. Typically, during such a pain intervention, patients receive information on medical procedures (i.e., procedural psychoeducation) and/or physical sensations (i.e., sensory psychoeducation) they may experience during or following a surgery or medical procedure. In a seminal study, Egbert and colleagues (1964) found that providing procedural information to patients resulted in post-surgery shorter inpatient hospital stays, less narcotic use, and lower physician ratings of patient pain. Later research supports the notion that providing procedural and sensory

information results in less pain-related outcomes, such as limited post-operative medication use of hospitalized patients under 50 years of age when provided sensory information (d = .66; Johnson & Leventhal, 1974) and increased patient pain tolerance in response to a laboratory shock pain task when provided both procedural and sensory information (d = .66; Staub & Kellett, 1972) when compared to control conditions.

One suggested mechanism by which psychoeducation reduces pain is through anxiety reduction. Patients commonly experience significant levels of fear and anxiety about surgeries or medical procedures (VanDalfsen & Syrjala, 1990). Anxiety is directly linked to worsened pain outcomes (Peck, 1986). Thus, researchers have explored the effects of psychoeducation on reduced postoperative anxiety. Indeed, providing procedural and sensory information to patients has direct anxiolytic effects: Felton et al. (1976) showed that outlining preoperative, operative, and postoperative procedures/equipment and usual postoperative discomforts to surgical patients decreased both pre- and post-operative anxiety and increased post-operative psychological well-being. Similarly, Garland et al. (2017) demonstrated in hospital patients that psychoeducation decreased both pain intensity and anxiety.

Relaxation

Relaxation is another technique that is effective at reducing acute pain responses. A relaxation response is a state characterized by parasympathetic dominance, reduced blood pressure and heart rate, muscle relaxation, and/or improved mood (Schaffer & Yucha, 2004). Relaxation techniques with documented analgesic effects include hypnosis and guided imagery.

Hypnosis. Hypnosis is a state of consciousness in which a subject maintains focused attention and reduced peripheral awareness, enhancing their capacity to respond to suggestions from another person to change their perceptions, memory, and voluntary actions (Elkins et al., 2015; Garland et al., 2017; Kihlstrom, 1985). Reviews (Kendrick et al., 2016; Montgomery et al., 2000; Patterson & Jensen, 2003) suggest that hypnosis is effective at reducing pain intensity in both laboratory and clinical settings. Further evidence corroborates this conclusion: Hypnosis reduces postoperative anxiety and pain intensity (d = 1.27 and d = .40, respectively, Garland et al., 2017; g = .40 and g = .25, respectively, Kekecs et al., 2014). Hypnosis is also likely effective at reducing pain by decreasing blood pressure and heart rate (Patterson & Jensen, 2003).

Guided Imagery. Guided imagery is a strategy in which an individual uses their imagination to form a mental representation that simulates the sights, sounds, tastes, smells, and touch sensations of a pleasant object, place, event, or situation (Felix et al., 2019). A guided imagery session may involve suggestions for visualizations using live instructions from a clinician or an audio recording. One reason for practicing guided imagery is pain relief (Felix et al., 2019). Researchers evaluated the effects of guided imagery on pain and healing in total joint replacement patients (Lin, 2012) and elderly patients after orthopedic operations (Antall & Kresevic, 2004). Researchers found somewhat encouraging evidence: Individuals that listened to guided imagery audio demonstrated trends towards decreased self-reported pain, anxiety, and length of hospital stay compared to individuals assigned to usual care and bedrest.

Psychological Distancing

Psychological distancing involves cognitively distancing oneself from direct, present experiences (Liberman et al., 2007; Liberman & Trope, 2014). When an individual creates mental distance from their own egocentric perspective, they are engaging in self-distancing (White & Carlson, 2016). Psychological distance from painful stimuli has pain relieving effects (Wang et al., 2019). Specifically, Wang and colleagues (2019) studied the effect of transcending one's self-perspective on pain during a cold pressor task (CPT). The CPT induces pain by having participants submerge their dominant hand into cold water, typically around 4°C. Individuals randomly assigned to a self-distancing group imagined their painful experience as an outside observer, whereas participants randomly assigned to a self-immersed group imagined their painful experience from their own perspective. Participants that assumed a self-distanced perspective experienced a decrease in pain sensation relative to participants who took the self-immersed perspective (d = .41) and control participants who were not specifically instructed to assume either a self-distanced or self-immersed perspective (d = .69).

Another, recent study on pain and psychological distance (Agerström et al., 2019) demonstrates that pain intensity measured in response to a CPT is significantly and negatively associated with different temporal, social, and spatial forms of self-transcendence. Specifically, the researchers found that with more intense pain, participants experienced less imagination of the past (d = .61), perspectives of other people (d = .96), and perspectives of themselves from a spatially distant camera (d = .53). The correlational nature of this data makes it impossible to determine whether pain

reduces self-transcendence, or whether self-transcendence reduces pain. Further empirical support is necessary to determine if greater transcendence reduces pain intensity.

Distraction

Distraction techniques for acute pain involve shifting attention away from painful stimuli toward stimuli that are more enjoyable. Enjoyable stimuli can be internal (e.g., imagery) or external (e.g., visual, auditory) to the individual (Birnie et al., 2017; Bascour-Sandoval et al., 2019). The limited attentional capacity theory and the multiple resource theory both support the idea that multisensory distraction is more effective than distraction on a singular, sensory modality (Birnie et al., 2017). However, singular modality distractors already demonstrate promising effects on acute pain (e.g., Silvestrini et al., 2011; Verhoeven et al., 2011). For example, Silvestrini and colleagues (2011) demonstrated that compared to silence, a pleasant music distractor and an auditory attention task both increased pain tolerance to a cold pressor task, $d = 1.09$ and $d = .81$, respectively. Verhoeven and colleagues (2011) demonstrated that a distraction group experienced CPT pain as less intense compared to a control condition, $d = .46$. An early review of the effect of distraction on pain (McCaul & Malott, 1984) suggested that various distraction techniques increase pain threshold and tolerance and decrease pain ratings compared to no treatment. Similarly, in their review, Bascour-Sandoval and colleagues (2019) suggest that there is sufficient evidence to conclude that auditory distractors, visual distractors, and cognitive distractors are effective at reducing acute pain in adults, but that the analgesic effectiveness of tactile distractors requires further empirical support.

Nature Exposure

Research has used nature views, images of nature, and videos of natural scenery to determine the analgesic effects of nature exposure (e.g., Diette et al., 2003; Miller et al., 1992; Ulrich, 1984). The seminal work of Ulrich (1984) demonstrated that postoperative patients with views of natural landscapes outside their hospital rooms had shorter hospital stays and required fewer analgesics compared to matched patients with views of brick buildings outside their hospital rooms. However, providing views of natural landscapes may not always be practical when treating acute pain. Thus, research has also examined the effects of nature images or murals on pain. Hospitalized patients with exposure to nature images have been shown to use weaker painkillers (Ulrich et al., 1993) and have better statistical odds of pain control (Diette et al., 2003) compared to patients without exposure to nature images. These results replicate in the experimental setting: Vincent and colleagues (2010) showed that without the presence of nature images, participants had significantly higher pain affect ratings. Similarly, videos of nature scenes demonstrate analgesic effects as well. Nature scenery videos accompanied by music significantly reduced pain for patients undergoing dressing change for burn wounds ($d = 4.71$, Miller et al., 1992). Moreover, in the laboratory setting, a soundless nature video increased pain threshold ($d = .80$) and tolerance levels ($d = 1.51$) compared to control in a tourniquet pain paradigm (Tse et al., 2002). Finally, with the advent of virtual reality (VR), nature scenery displayed via VR headset reduced pain experience in response to a CPT and reduced pain as recollected 1 week later (Tanja-Dijkstra et al., 2018).

Positive Affect Inductions

Many studies have experimentally manipulated affect to reduce acute pain. Positive affect is regularly manipulated through the viewing of emotionally evocative images (e.g., from the International Affective Picture System; IAPS) or film clips or through experiencing pleasant odors or music (Finan & Garland, 2015). Research demonstrates that participants viewing pleasant photo inductions report significantly less pain in response to a nociceptive flexion reflex task using electrical stimulation (d = 1.25, Rhudy et al., 2005) and significantly higher pain tolerance in response to a CPT (d = .50, de Wied & Verbaten, 2001) when compared to participants that viewed neutral pictures.

Limitations of Existing Approaches

Though many of the existing psychological approaches to reducing acute pain demonstrate moderate to large effect sizes of pain reduction when compared to conditions in which pain-reduction approaches are absent, existing approaches have many limitations. By nature, psychoeducation may be limited to only operative or procedural acute pain contexts. Physicians may be better able to provide information about painful experiences prior to when they occur or when they are more predictable. However, acute pain does not only occur during operations or procedures but may also take the form of injuries (e.g., broken bones, burns) or pain associated with dying. In these cases, providing procedural or sensory information may not be feasible or effective, such as if acute pain is occurring for unknown reasons. Additionally, there is increasing evidence that other psychological interventions for acute pain outperform psychoeducation. For example, Garland and colleagues (2017) demonstrate in hospitalized patients that mind-

body interventions (e.g., hypnosis) reduce pain severity more than psychoeducation. Thus, maximizing non-pharmacological pain relief requires more than just psychoeducation alone.

Furthermore, researchers warn about the use of relaxation as a main treatment for acute pain (Liossi & Franck, 2008; Seers & Carroll, 1998). With mixed evidence on the effectiveness of relaxation techniques, authors do not recommend relaxation to be used as the sole analgesic technique, but that a multimodal approach including relaxation may be more effective. Moreover, a recent meta-analysis casts some doubt on the effectiveness of some relaxation techniques: Garland and colleagues (2020) found moderate to large effect size improvements in pain outcomes for hypnosis, but not for muscle relaxation or guided imagery. Hypnosis has its own limitations as an approach to acute pain relief: Training hypnosis to patients takes a lot of time (Patterson & Jensen, 2003) and audio recordings of clinicians delivering hypnosis training may not be an effective substitution for live presentation (Kekecs et al., 2014).

Other psychological interventions, such as psychological distancing, distraction, and nature exposure, also have a few limitations when considered for acute pain management. Current empirical evidence is insufficient in demonstrating that psychological distancing reduces pain. Furthermore, psychological distancing requires effort and training both on the part of the acute pain sufferer and clinicians treating such patients, which may not be feasible in all acute pain contexts (e.g., acute injuries). Distraction shows promise as an acute pain-reliever but may be limited to short-term effects. Further, individuals using distraction to alleviate acute pain may effectively

divert attention away from signals alerting to potential tissue damage, thereby exacerbating injuries, and causing sustained pain (Johnson, 2005). Nature exposure also demonstrates strong analgesic benefits, but the physical presence of nature is not always feasible for individuals experiencing acute pain. A lab-based, audiovisual induction of awe may address at least some of these limitations. Thus, it seems to be worthwhile to probe the analgesic benefits of awe.

Awe

Awe is a form of affect in which someone perceives something as vast and transcendent past their current frame of reference (Piff et al., 2015). An individual experiencing awe will need to adjust their current mental structures to accommodate stimuli that transcends their ordinary reference frame (Zhao et al., 2019). Feelings of awe resemble those of wonder, amazement, enlightenment, and admiration (Zhao et al., 2019). However, awe is autonomically and functionally distinct from other forms of affect (Shiota et al., 2011). Awe may be either negatively or positively valanced, though most research observing the health benefits of awe draws upon positive affective experiences. Overall, awe lacks empirical research compared to its more common positive affective counterparts (e.g., contentment, joy). This lack of research may be due to awe's diverse causes and unclear function (Shiota et al., 2007). Perceptually or conceptually complex stimuli, such as panoramic nature views, novel art and music, and remarkable accomplishments of other people (Shiota et al., 2007), tend to elicit awe. Moreover, any experience that challenges one's accustomed frame of reference and causes a need to update or renew schemas to account for deviations from existing

schemas may constitute an awe experience. Awe is frequently manipulated using nature-exposure: Exposure may be either physical, such as sitting in or walking in natural landscapes (Ballew & Omoto, 2018; Lopes et al., 2020) or via media (e.g., videos: McPhetres, 2019; van Elk et al., 2019). The function of awe remains up for debate. Awe may have evolved to induce social submission to powerful others, to seek safe shelters with good vantage points, to respond to natural wonders, or to advance cognition via safe exploration of stimuli eliciting curiosity (Richesin & Baldwin, 2022). Therefore, the function of awe may be to increase survival chances by maintaining social structures, keeping people safe, or increasing curiosity and learning (Richesin & Baldwin, 2022).

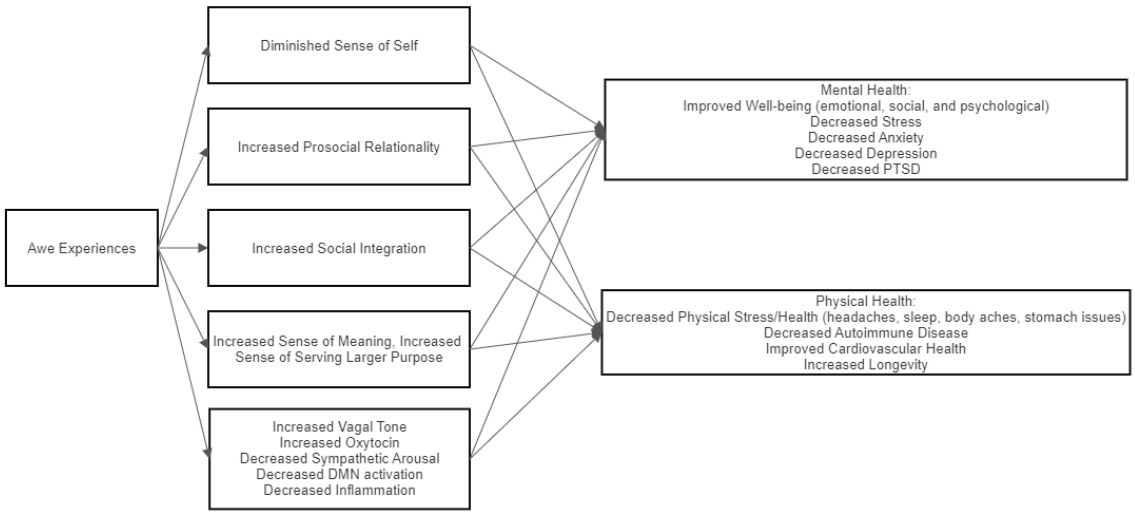
Keltner and Haidt (2003) were the first to first propose two central themes of awe: perceived vastness and a need for accommodation (i.e., the adjusting of mental schemas to integrate a vast novel experience). Awe is associated with a sense of smallness of the self, the presence of something greater than the self, and results in disengagement from self-focused thoughts and emotions (Shiota et al., 2007). Beyond the key features of vastness and need for accommodation, additional factors of awe include altered time perception, connectedness, and physical sensations. Awe elicits a perceptual slowing of time, a sense of connection with the social and non-social environment beyond oneself, and physical sensations of ‘goosebumps’ or chills and facial movements such as widened eyes and a dropped jaw (Yaden et al., 2018). These five factors of an awe experience may be measured using state awe measures. State awe is directly related to an awe-invoking situation (Yaden et al., 2018). Dispositional (i.e., trait) awe is an individual’s characteristic pattern of experiencing awe across different situations (Shiota et al., 2006).

The two, therefore, are distinct constructs. Though both aspects of experiencing awe may be correlated, the two can be measured independently (Yaden et al., 2018).

Monroy and Keltner (2022) recently developed a theoretical model of the mental and physical health outcomes of awe experiences (see Figure 1). Direct outcomes of awe experiences are associated with better mental and physical health outcomes. For example, awe experiences result in various neurophysiological changes associated with improved health: increased vagal tone, decreased sympathetic arousal, increased oxytocin, decreased default mode network (DMN) activity, and decreased inflammation (Monroy & Keltner, 2022). However, this model lacks consideration of an important physical health outcome: pain. It is currently unknown whether awe provides pain-relieving effects. The purpose of the current study is to determine if awe decreases acute pain.

Figure 1

Awe as a pathway to mental and physical health



Note: Monroy and Keltner's (2022) model for awe as a pathway to mental and physical health.

Indirect Evidence for the Analgesic Benefits of Awe

Psychological Evidence. Awe may reduce anxiety and, thereby, reduce pain. The physiological profile of awe is associated with reduced anxiety (John-Henderson et al., 2015 as cited in Monroy & Keltner, 2022). There is preliminary evidence that inducing awe reduces anxiety (Rankin et al., 2020). In a sample of undergraduate students, Rankin and colleagues (2020) induced awe or general positive feelings during a period of waiting for intelligence testing scores and compared outcomes to participants in a neutral control condition that watched a video aimed at gaining participant interest, but not physically or emotionally evoking participants. Participants in the awe condition reported less anxiety relative to participants in the positive affect and neutral conditions. In turn, experiencing anxiety is directly linked to worsened pain (Peck, 1986) and various psychological interventions appear to reduce pain through reducing anxiety (e.g., psychoeducation, guided imagery; Antall & Kresevic, 2004; Garland et al., 2017; Lin, 2012). Thus, because awe seems to reduce anxiety, inducing awe may also reduce pain.

A diminished sense of self may also contribute to decreased pain. The study by Wang and colleagues (2019), in which participants experienced decreased pain when assuming a self-distanced perspective, lends support to the proposed hypothesis that individuals experiencing awe will report less pain compared to individuals not experiencing awe. Awe causes a sense of self-diminishment (Shiota et al., 2007), shifting attention away from a self-focused perspective (Piff et al., 2015). Thus, because self-distancing reduces pain, awe may reduce pain as well.

Physiological Evidence. A key mechanism of relaxation techniques for acute pain relief involves reduced blood pressure and heart rate, which is indicative of sympathetic nervous system withdrawal and parasympathetic dominance. Awe similarly reduces sympathetic nervous system activation (Shiota et al., 2011) and leads to higher parasympathetic activation (Chirico & Gaggioli, 2021). Consequently, these findings also support an analgesic function of awe.

Research on awe and the pro-inflammatory marker interleukin-6 (IL-6) provides further support for the idea that awe may have analgesic properties. Stellar et al. (2015) compared multiple discrete positive emotions and found that dispositional awe was the strongest predictor of reduced IL-6. Inflammatory mediators, such as IL-6, are produced during inflammation and cause pain by direct activation of nociceptors (Matsuda et al., 2019). Though research has not yet established the relationship between *state* awe and pro-inflammatory cytokines, research by Stellar et al. (2015) demonstrates the potential for awe to be associated with lower inflammation and, thereby, reduced pain.

Furthermore, tentative evidence suggests awe is associated with increased oxytocin release (Thomson & Siegel, 2017) and oxytocin has analgesic effects (Bharadwaj et al., 2021). However, the effect of awe on oxytocin production may depend on the type of awe-inducing stimulus. Initial findings on awe and oxytocin demonstrate that awe stemming from observing morally courageous or kind acts increased oxytocin (Thomson & Siegel, 2017). Empirical evidence has not yet demonstrated that awe-induced by experiences other than viewing morally courageous or kind acts affects

oxytocin. Nevertheless, a connection between awe and pain relief is plausible through an effect of awe on oxytocin.

Overall, awe seems to share overlapping mechanisms with other interventions shown to reduce pain (e.g., via anxiety reduction, self-diminishment, sympathetic nervous system withdrawal). Additionally, an awe manipulation may have overlapping components with other interventions shown to reduce pain (e.g., distraction from pain, nature-exposure, positive affect induction). Awe may have differential effects on pain compared to other psychological interventions by combining components of established interventions. Additionally, awe may be feasible to induce in situations where other interventions may not, such as in contexts where training of psychological skills may not be feasible (e.g., emergency medicine) or in contexts where verbal instruction may not be understood (e.g., with children, with people of a different spoken language). Therefore, it seems to be worthwhile to test awe as a new psychological intervention to reduce pain.

The Current Study

The current study relied on a between-subjects design to manipulate the experience of awe and study resulting pain-related outcomes. After completing a short rest period to collect baseline physiological data, participants viewed either an awe-invoking nature video or a nature video without awe-invoking elements. The selected videos had been used before to successfully manipulate the experience of awe (Krenzer, 2018). Components of visual and auditory vastness were present in the awe-invoking nature video, but not in the control nature video. Both videos featured nature elements, however, to control nature video content because nature exposure itself has been shown

to have analgesic effects. While viewing one of these videos, participants engaged in the CPT (von Baeyer et al., 2005). This widely used paradigm involved participants submerging their dominant hand into 4°C (± 1°C) circulating water to induce pain. With their hand in the water, participants indicated when they first experienced a painful sensation (i.e., pain threshold) and when they could no longer tolerate the painful sensation (i.e., pain tolerance). Upon reaching their pain tolerance, participants were allowed to remove their hand from the water. During the CPT, I continuously collected blood pressure (BP) and electrocardiogram (ECG). Next, participants completed a measure of pain intensity experienced during the CPT and rested for 5 minutes as I continued recording BP and ECG. Finally, participants completed a manipulation check measure to determine the effectiveness of the awe manipulation. I hypothesized that individuals experiencing awe relative to individuals in a non-awe control group would report less pain in response to the CPT (H1), endure the CPT for a longer duration before pain threshold was reached (H2a), and endure the CPT for a longer duration before pain tolerance was reached (H2b). I also hypothesized that individuals experiencing awe relative to individuals in a non-awe control group would show a cardiovascular response to painful stimuli that deviates less from baseline (H3). Specifically, I hypothesized that baseline-corrected mean heart rate (H3a), mean systolic blood pressure (H3b), and diastolic blood pressure (H3c) would be significantly lower for individuals experiencing awe relative to individuals in a non-awe control group.

Method

Power

To determine the sample size needed, I initially considered a power analysis based on a published minimum clinically significant difference (MCID) for the primary outcome of interest, i.e., pain reduction on visual analog scale (VAS) as a function of my awe manipulation. A MCID on a VAS indicates the smallest change on the scale that a person would identify as a meaningful change in pain intensity (Gallagher et al., 2001). To calculate an effect size estimate, specifically Cohen's d, using a MCID value, the MCID must be divided by a pooled standard deviation of VAS scores in experimental conditions. Gallagher et al. (2001) and Todd et al. (1996) established a value of 13 mm on a 100 mm scale as the average minimum change in acute pain that is clinically significant. In contrast, an estimation of the standard deviations of VAS scores in my awe and control conditions has not been established in previous studies. To obtain a realistic estimate of these values, given the specifics of sample and methods in my study, I most effectively would need to collect pilot data using my awe manipulation to estimate these standard deviations. Such a pilot study did not seem feasible. Consequently, I chose instead to rely on a general estimate of the effect size to determine the sample size required for my analyses. I completed an a priori power analysis using G*Power 3.1 (Erdfelder et al., 1996). Because of a lack of previous research on the effect of awe on experimentally induced pain, I assumed a medium effect size (d = .50) with a power criterion of 80% and an alpha of .05. The analysis suggested 64 participants per condition.

Participants

I recruited 130 participants (*n*=65 each condition) from the psychology participant pool (SONA system) at Ohio University and from the larger Athens community via email and paper fliers. See Table 1 for demographic information. Following previous studies that have used the CPT to induce pain, I excluded individuals with an open cut or sore on the hand to be immersed, a bone fracture of the limb to be immersed, a history of frostbite, Raynaud's disease, fainting or seizures, or cardiovascular disorder (i.e., high BP, heart disease, arrhythmia/dysrhythmia; Birnie et al., 2012; McIntyre et al., 2020; von Baeyer et al., 2005). These exclusion criteria were intended to avoid adverse events due to increased pain sensitivity associated with existing tissue injury (e.g., cuts, fractures), a history of cold-temperature related tissue injury (e.g., frostbite), or disease-related excessive artery constriction in response to cold (e.g., Raynaud's). Additionally, the exclusion criteria were intended to avoid adverse events due to vasoconstriction and vasodilation associated with submerging and removing the hand from the cold-water stimulus. Consecutive vasoconstriction and vasodilation pose an increased risk of fainting and increased strain on the heart, which may negatively affect persons with a history of fainting, seizures, or cardiovascular disorder. To prevent stress to a fetus or excessive strain on nerve fibers by the CPT, individuals who are currently pregnant or think they may be pregnant or have a neurological disorder did not participate in the study (Birnie et al., 2012; McIntyre et al., 2020; von Baeyer et al., 2005). Individuals with a disorder causing increased pain sensitivity to cold temperatures (e.g., Reflex Sympathetic Dystrophy or Complex Reactive Pain Syndrome; Reimer et al., 2016) were also excluded

from the experiment due to the risk of an intensified pain response to cold-water stimulation. Moreover, due to increased risk of tissue damage from blunted nociceptive response as a consequence of pain medication use, individuals were not eligible to participate in the study if they had taken medications that influence pain (Mischkowski et al., 2021; i.e., ibuprofen within 15 hours, acetaminophen within 15 hours, or aspirin within 24 hours of the study; regular use of pain medicines [e.g., morphine or tramadol] or migraine medications [e.g., sumatriptan or ergotamine]). Participants were also excluded if they indicated regular use of other medications that affect pain. Such medications include some antidepressants (e.g., amitriptyline or duloxetine; Dharmshaktu et al., 2012), some seizure medications (e.g., gabapentin or pregabalin; Eipe et al., 2015; Turan et al., 2004), and some anxiety medications (e.g., clonazepam or lorazepam; Reddy & Patt, 1994).

Table 1

Demographic Information

Variable	*N*	%
Self-reported sex at birth		
Male	43	33.08
Female	87	66.92
Race		
American Indian or Alaska	0	0.00
Asian American or Pacific	6	4.62
Black/African American	7	5.38
Middle Eastern or Arab	2	1.54
White/European American	102	78.46
Other	6	4.62
Mixed	4	3.08
Hispanic origin		
Not Hispanic	123	94.62
Yes, Hispanic	7	5.38

Note. Race information missing for 3 cases

Procedure

All experimental procedures were approved by the Ohio University Biomedical Institutional Review Board and were preregistered (https://osf.io/xf8s7). Individuals interested in participating in the study either directly signed up for a lab session (if they were recruited from the SONA participant pool) or emailed researchers to schedule a lab session (if they were recruited via paper or email flyer). Upon arriving at the lab, participants provided informed consent. Next, participants provided demographic information and completed the Dispositional Positive Emotion Scale – Awe Subscale (DPES-Awe; Shiota et al., 2006) to capture individual differences in the disposition to

experience awe (i.e., trait awe). Afterwards, an experimenter seated participants comfortably for a 5-minute resting baseline data acquisition to obtain baseline BP and ECG. After baseline cardiovascular data was collected, the researcher instructed participants on how to complete subsequent experimental procedures, which involved watching 2 minutes of a video to manipulate awe and then completing the CPT, i.e., continuing to watch the video while submerging their dominant hand up to the wrist in a cold-water reservoir (Model II-BX Chiller, The Wine Well Chiller Company, Milford, Conn.).

Experimental Awe Manipulation

I randomly assigned participants to one of two experimental conditions in which they either watched an awe-inducing or a control nature video. The 4 min 34 s awe-inducing video (view at http://tinyurl.com/9cxf73c3) depicted landscape scenes that shift dynamically in scope (i.e., from close to far away) for portraying vastness. Music accompanying the video also featured shifts (e.g., changes in loudness and intensity, such as through crescendos) to elicit feelings of vastness, which is an important aspect for experiencing awe (Keltner & Haidt, 2003). Similarly, the 4 min 34 s control nature video (view at https://tinyurl.com/3h6nhken) depicted nature scenes and was accompanied by a music soundtrack. However, the control video and music soundtrack lacked the visual shift in scope and auditory shifts in loudness and intensity of the awe-inducing video. Research using these videos has demonstrated that participants in the awe condition experience higher levels of awe than participants in the control condition (Krenzer, 2018). In the current study, participants watched one of these videos on a computer

screen and listened to the accompanying soundtrack through computer speakers to allow for communication between experimenter and participants, which was necessary because participants completed the CPT while viewing the assigned videos. Specifically, the participants watched 2 min of their respective videos before submerging their hand into the cold-water reservoir, following a verbal request from the experimenter. Participants simultaneously watched 2 min of the video and completed the CPT until they could no longer tolerate the painful sensation (i.e., pain tolerance level). Participants were not permitted to maintain their hand in the cold water for any longer than 2 min to prevent cold water immersion injuries consistent with prior work (e.g., Geers et al., 2008). Once pain tolerance or maximum immersion time was achieved, participants removed their hand from the reservoir and watched the remainder of the video.

CPT

During this task, participants submerged their dominant hand into 4°C (± 1°C) circulating water and then verbally indicated as soon as they experienced any painful sensations. Time in seconds passed since task onset served as a measure of pain threshold. Furthermore, participants were allowed to remove their hand from the water when they could no longer tolerate the painful sensation anymore. Specifically, at this point, participants removed their hand from the cold-water reservoir while verbally indicating their intent to do so. Time in seconds since task onset served as a measure of pain tolerance. The participants had short, printed instructions at hand during the CPT.

Following the CPT, participants completed a VAS of pain (Karcioglu et al., 2018). Then, participants completed another 5-minute resting period to obtain measures

of cardiovascular recovery. At this time, BP and ECG were collected. After this period, I stopped cardiovascular data collection, and participants completed the Awe Experience Scale (AWE-S; Yaden et al., 2018). Finally, I debriefed participants and compensated them.

Self-Reported Measures

Trait Awe

I assessed participants' dispositional tendency to experience awe using the awe subscale of the Dispositional Positive Emotion Scale (DPES-Awe; Shiota et al., 2006). The scale consists of 6 items—such as "I have many opportunities to see the beauty of nature"—in which participants indicated their level of agreement on a 7-point scale from 1 (*strongly disagree*) to 7 (*strongly agree*). I averaged responses across items to create a composite measure of trait awe.

State Awe

The Awe Experience Scale (AWE-S) is a 30-item self-report measure of state awe (Yaden et al., 2018). The AWE-S captures different aspects of the awe experience, including alteration of the sense of time (e.g., "I noticed time slowing"), sense of self-diminishment (e.g., "I felt my sense of self shrink"), feelings of connectedness (e.g., "I felt closely connected to humanity"), vastness perception (e.g., "I perceived something that was greater than myself"), physical sensations (e.g., "I had goosebumps"), and a need for accommodation (e.g., "I felt challenged to understand the experience"). Participants indicated agreement with items on 7-point scales from 1 (*strongly disagree*) to 7 (*strongly agree*). The AWE-S, including its subscales, has shown strong internal

consistency (αs≥.80; Yaden et al., 2018). Further, Yaden et al. (2018) demonstrate via confirmatory factor analysis that the scale has adequate construct validity (CFI = .905, RMSEA = .054). I calculated sum scores across items to create a composite measure of awe experienced during the video manipulation.

Pain Intensity

I administered a VAS of pain as an established measure of capturing self-reported pain intensity during the CPT (Karcioglu et al., 2018; von Baeyer et al., 2005). The VAS is administered directly after the painful stimulation (e.g., the CPT) to measure pain intensity, using anchors of 0 (*no pain*) and 100 (*worst imaginable pain*; von Baeyer et al., 2005). The pain VAS has demonstrated high reliability when measuring acute pain (Bijur et al., 2001).

State Anxiety

I assessed participant state anxiety using the state portion of the State-Trait Anxiety inventory (STAI; Spielberger et al., 1983). Participants indicated state anxiety by indicating the extent to which they felt, in the present moment, the given 20 statements on a scale from 1 (*not at all*) to 4 (*very much so*).

Physiological Measures

Blood Pressure

To continuously measure BP throughout the experiment, I used a non-invasive, beat-to-beat system, the CNAP Monitor (CN Systems, Inc., Graz, Austria). To collect a continuous BP wave using the MP160 (Biopac Systems, Inc., Goleta, CA, USA), the participant wore a finger cuff around the index and middle fingers of their nondominant

hand calibrated using an arm cuff around the participant's nondominant upper arm. Additionally, I placed electrodes in a lead II configuration (Constantin et al., 2017) on the participant's torso to measure ECG. ECG was collected using the respective model of the MP160 system (Biopac Systems, Inc., Goleta, CA, USA).

I processed BP data in AcqKnowledge (Biopac Systems, Inc., Goleta, CA, USA) by initially running an arterial blood pressure analysis on the raw BP wave. The arterial blood pressure analysis classified signals, indicating systolic, diastolic, and ECG boundaries. I visually scanned the BP wave with classified signals to ensure systoles and diastoles were appropriately marked for the final minute of the baseline measurement, the first minute and/or the whole length of the CPT, and the entire length of the recovery. I manually fixed the signals for any systoles or diastoles that were inaccurately marked, using R-wave inflections on the ECG wave to help determine placement of missed signals. Following visual scanning, I reran the arterial blood pressure analysis as needed to extract from the properly marked wave systolic blood pressure (SBP) and diastolic blood pressure (DBP) on a cycle-by-cycle basis. For each section of the experimental procedure (e.g., baseline), I highlighted the section of interest and extracted SBP and DBP values. Specifically, I extracted mean SBP and DBP for the final minute of the baseline resting period, the first minute of the CPT task, the length of the individual's CPT task, and the entire 5-minute recovery period. Finally, I determined systolic and diastolic BP reactivity for each participant. I subtracted average systolic BP (in mmHg) from the final minute (most relaxed period) of the resting baseline from average systolic BP in the first minute (most reactive period) of the CPT while the awe manipulation was

taking place (Llabre et al., 1991). Further, because the majority of the participants did not endure the CPT for a minute or longer, I calculated systolic BP reactivity by subtracting mean systolic BP from the final minute of the resting baseline from mean systolic BP during the entire CPT, with the time varying for each participant. I performed the same calculations to determine diastolic BP reactivity as a function of the awe manipulation.

Heart Rate

Heart rate (HR; in beats per minute) was calculated during the arterial blood pressure analysis by extracting the diastolic-to-diastolic time interval for a given cycle (Biopac Systems, Inc., Goleta, CA, USA). Much like BP, for each section of the experimental procedure, I highlighted the section of interest and extracted HR values for the final minute of the baseline resting period, the first minute of the CPT task, the length of the individual's CPT task, and the entire 5-minute recovery period. Finally, I determined HR reactivity by subtracting mean HR from the final minute (most relaxed period) of the resting period from mean HR in the first minute (most reactive period) of the CPT while the awe manipulation was taking place (Llabre et al., 1991). Again, because most of the participants did not endure the CPT for a minute or longer, I calculated HR reactivity by subtracting the average HR from the final minute of the resting baseline from average HR during the entire CPT, with the time varying for each participant.

Deviations from Preregistration

Given participants removed their hand from the cold, circulating water at different times during the CPT, cardiovascular recovery likely started for individuals that removed

their hand from the water soon after the CPT started, and well before the 5-minute recovery period. Similarly, individuals that maintained their hand in the water for the entire duration of the CPT likely started their cardiovascular recovery from the event right at the beginning of the 5-monute recovery period. Therefore, evaluating physiological data over the 5-minute recovery period was abandoned given different start times for cardiovascular recovery during and after the CPT.

Also abandoned was the preregistered technique for manually calculating changes in heart rate: Instead of manually counting R wave inflections of the ECG data and estimating heart rate during 6 second intervals, I used diastole-to-diastole intervals identified electronically by Biopac to observe changes in heart rate. Electronic calculation of changes in heart rate are likely more accurate than human counts and mathematical computation, thereby reducing error. Further, I manually scanned the marked diastoles and the subsequent heart rate waveform for errors, further strengthening the validity of the electronic heart rate calculation. Finally, I opted to evaluate HR, SBP, and DBP reactivity rather than HR, SBP, and DBP amplitude or area-under-the-curve (AUC) given the extensive literature on reactivity values in relation to pain (e.g., Hellström & Lundberg, 2000; Oka et al., 2008). Further, HR, SBP, and DBP reactivity offer a more straightforward, parsimonious analysis of the present data compared to AUC analyses given substantial differences in CPT duration.

Results

Preliminary Analyses

Descriptive statistics and zero-order correlations among study variables are provided in Table 2. Mean pain intensity ratings using the VAS were similar to mean pain intensity ratings in previous literature using a 4°C CPT (e.g., Koenig et al., 2014; Lentini et al., 2020; Hellström & Lundberg, 2000), with particular similarity to mean and standard deviation values observed in Johnson and Petrie's (1997) study evaluating CPT pain intensity ratings under distraction (vs. control) conditions (M =49.33, SD = 21.48 and M =52.77 SD = 18.80 , respectively). Observed mean times to pain threshold in seconds were similar to those observed in previous studies using a 4°C CPT (e.g., Koenig et al., 2014; Streff et al., 2010), though slightly longer on average in the current study when evaluating raw time to thresholds and appearing more similar to mean times reported in previous literature when excluding time to threshold outliers (M = 20.03, SD = 11.60). Finally, mean times to pain tolerance in the current study were similar to those observed in previous research using a 4°C CPT (e.g., Koenig et al., 2014; Brady et al., 2006), with some variability present likely due to a lack of standardization in CPT techniques (e.g., maximum submersion times). However, current study mean times to pain tolerance (and standard deviations) fell between those reported by Koenig and colleagues (2014) and Brady and colleagues (2006).

To ensure proper randomization, I first tested baseline differences in demographics and trait awe between experimentally manipulated awe and control conditions using chi-square and independent samples t-test analyses. Chi-square analyses

revealed that there were no significant differences between awe and control conditions in gender ($\chi^2(2) = 3.071$, $p = .215$), sex ($\chi^2(1) = .035$, $p = .852$), race/ethnicity ($\chi^2(5) = 6.039$, $p = .302$), relationship status ($\chi^2(3) = 2.300$, $p = .513$), or income ($\chi^2(7) = 7.491$, $p = .380$). A chi-square analysis on whether education was significantly different between awe and control conditions approached statistical significance, $\chi^2(4) = 9.302$, $p = .054$. However, because it did not fall below traditional standards ($p < .05$) and because many participants appeared to misinterpret the prompt (i.e., SONA recruited participants frequently indicated their highest level of education was 'high school diploma or equivalent (GED)' instead of 'some college/2-year degree (AA)'), education was not included as a covariate in subsequent analyses. Independent samples t-test analyses revealed that there were no significant differences between awe and control conditions in age ($t(128) = 1.191$, $p = .236$), national-level socioeconomic status ($t(128) = -.395$, $p = .694$), or community-level socioeconomic status ($t(128) = .376$, $p = .707$). Importantly, trait awe did not differ significantly between awe and control conditions, $t(128) = -1.232$, $p = .220$. Finally, I completed an independent samples t-test comparing state awe across conditions to check whether the group that viewed the awe video experienced more awe than the control group and to serve as a manipulation check. The difference between the conditions was not significant, $t(128) = -1.446$, $p = .151$), indicating there was not a statistically significant difference in state awe experienced across conditions. However, mean scores demonstrate that participants in the experimental awe group reported, on average, more state awe than the control group (Table 3).

Table 2

Descriptive Statistics and Zero-order Correlations

Variable	*N*	*M (SD)*	1	2	3	4	5	6	7	8	9	10	11
1. Trait awe	130	5.33 (.83)	-										
2. State awe	130	120.84 (29.43)	.309**	-									
3. Pain intensity rating (VAS)	130	52.04 (20.88)	-.004	.081	-								
4. Time to threshold	130	24.45 (23.23)	.002	-.037	-.240**	-							
5. Time to tolerance	130	59.17 (36.75)	-.026	-.037	-.170	.621**	-						
6. HR reactivity during 1st min of CPT	128	5.23 (8.82)	-.018	-.021	-.060	.236**	.396**	-					
7. HR reactivity for duration of CPT	129	7.30 (8.51)	-.010	.012	.036	.011	.063	.863**	-				
8. SBP reactivity for 1st min of CPT	128	-1.98 (14.71)	-.024	-.097	-.020	.112	.053	-.039	-.072	-			
9. SBP reactivity for duration of CPT	129	-1.29 (15.29)	-.016	-.095	-.096	.223*	.223*	.044	-.063	.972**	-		
10. DBP reactivity for 1st min of CPT	128	3.39 (8.64)	-.028	-.054	-.111	.355**	.283**	.168	.058	.672**	.694**	-	
11. DBP reactivity for duration of CPT	129	4.34 (9.50)	-.011	-.049	-.150	.423**	.431**	.233**	.053	.623**	.702**	.960**	
12. State anxiety	130	1.71 (.46)	-.243**	-.138	-.018	-.051	-.034	-.047	-.026	-.005	.001	-.081	-.063

** = $p < .01$; * = $p < .05$; *Note:* Raw data were used for *M(SD)* for time to threshold and trait awe, whereas log-transformed threshold data and mean-centered trait awe data were used for zero-order correlations.

Table 3

Mean and Standard Deviation Values of Study Variables Across Conditions

Variable	Control Condition M (*SD*)	Awe Condition M (*SD*)
State awe	117.12 (21.76)	124.55 (35.28)
Pain intensity rating (VAS)	52.35 (22.02)	51.72 (19.85)
Time to threshold	20.59 (12.51)	19.43 (10.65)
Time to tolerance	60.58 (37.33)	57.76 (36.39)
HR reactivity during 1st min of CPT	5.63 (9.21)	4.83 (8.45)
HR reactivity for duration of CPT	7.89 (8.88)	6.70 (8.14)
SBP reactivity for 1st min of CPT	-3.54 (15.43)	-0.42 (13.91)
SBP reactivity for duration of CPT	-2.78 (16.50)	0.23 (13.92)
DBP reactivity for 1st min of CPT	2.74 (9.40)	4.04 (7.83)
DBP reactivity for duration of CPT	3.83 (10.59)	4.87 (8.32)
State anxiety	1.83 (0.50)	1.60 (0.40)

Note: Raw data were used for *M (SD)* for time to threshold.

Self-Reported Pain Intensity in Response to CPT (H1)

To test whether the awe (vs. control) group reported less pain in response to the CPT, I first checked the assumptions for performing an independent samples t-test on VAS scores across conditions. I conducted a Shapiro-Wilk test of normality to determine whether VAS scores were normally distributed. The results indicated that VAS scores were not normally distributed ($p = .005$). I analyzed whether outliers were affecting the normality of VAS scores (given $M = 52.04$ and $SD = 20.88$) based on a specific distance from the mean (i.e., 3 *SD*s). Values 3 standard deviations above the mean (114.68) and 3 standard deviations below the mean (-10.60) were not possible given the 0-100 rating scale presented to participants. Therefore, no VAS scores were identified as outliers.

Additionally, as indicated in the preregistration, I attempted to establish normality by performing a log-transformation on VAS scores; however, log-transformed VAS scores remained nonnormally distributed (p .< 001). Therefore, I performed a nonparametric Wilcoxon-Mann-Whitney test on VAS scores across conditions. The results of the Wilcoxon-Mann-Whitney test indicated there was not a statistically significant difference in VAS scores between participants in the awe and control conditions, z = -.266, p = .79, thus not supporting H1.

Duration of CPT Endured Until Pain Threshold (H2a) and Pain Tolerance (H2b)

To test that duration of CPT until pain threshold and duration of CPT until pain tolerance was significantly longer in the awe (vs. control) condition, I first checked the assumptions for performing an independent samples t-test on duration of CPT until pain threshold (hereafter 'threshold') and duration of CPT until pain tolerance (hereafter 'tolerance'). First, I conducted a Shapiro-Wilk test of normality to determine whether thresholds were normally distributed. The results indicated that thresholds were not normally distributed (p < .001). Again, I analyzed whether outliers based on a specific distance from the mean (i.e., 3*SD*s) were affecting the normality of thresholds given M = 24.45 and SD = 23.23. Values 3 standard deviations below the mean (-45.24 s) were not possible; however, values 3 standard deviations above the mean (94.14 s) delineated threshold outliers. Once I excluded outliers (n = 6), I log-transformed thresholds and ran another Shapiro-Wilk test of normality. With outliers excluded and values log-transformed, thresholds were normally distributed (p = .271), and I was able to complete an independent samples t-test. The results of the independent samples t-test indicated

thresholds were not significantly different in the awe (vs. control) condition, $t(122) = .19$, $p = .85$, $d = .03$, thus not supporting H2a. I tested the normality of tolerances using a Shapiro-Wilk test of normality. Tolerance was not normally distributed, $p < .001$. I checked for outliers 3*SD* above the mean and 3*SD* below the mean given $M = 59.17$ and $SD = 36.75$. Values 3 standard deviations below the mean (-51.08 s) and values 3 standard deviations above the mean (169.42 s) were not possible given the CPT cutoff of 120 s. Therefore, there were no outliers to exclude. Again, as indicated in the preregistration, I attempted to establish normality by performing a log-transformation on tolerances; however, log-transformed tolerances remained nonnormally distributed (p .< 001). Accordingly, I performed a nonparametric Wilcoxon-Mann-Whitney test on tolerances across conditions. The results of the Wilcoxon-Mann-Whitney test indicated there was not a statistically significant difference in tolerances between participants in the awe and control conditions, $z = -.258$, $p = .80$, thus not supporting H2b.

Cardiovascular Response to CPT: Heart Rate (H3a), Systolic Blood Pressure (H3b), and Diastolic Blood Pressure (H3c)

To test whether the awe group relative to the control group showed a cardiovascular response (i.e., HR and BP reactivity) to painful stimuli that deviated less from baseline, I evaluated whether independent samples t-tests were appropriate. Shapiro-Wilk tests of normality indicated that all reactivity variables—HR reactivity during the first minute of the CPT and during participants' own CPT duration, SBP reactivity during the first minute of the CPT and during participants' own CPT duration, and DBP during the first minute of the CPT and during participants' own CPT duration—

were not normally distributed, *p*s ≤ .004. An analysis of potential outliers 3 *SD* away from the mean indicated single outliers for mean HR during the final minute of baseline (135.624 bpm), mean SBP during the final minute of baseline (170.416 mmHg), and mean SBP during the first minute of the CPT (172.788 mmHg) and two outliers for mean HR during the first minute of the CPT (128.727 bpm and 136.324 bpm). For cases with identified potential outliers, I visually scanned continuous blood pressure waves again to ensure signals were properly located and values were correctly coded. Given signals were adequately placed, values were coded consistently, and outlier values remained within humanly possible values, no participant data with non-missing values were excluded in HR or BP analyses. I conducted nonparametric Wilcoxon-Mann-Whitney tests on HR, SBP, and DBP reactivity variables given nonnormality. Wilcoxon-Mann-Whitney tests comparing heart rate reactivity during the first minute of the CPT and during the participant's individual CPT duration across conditions indicated no significant reduction in the deviation from baseline for the participants in the awe (vs. control) condition, z = -.858, p = .39 and z = -1.126, p = .26, respectively, thereby indicating H3a was not supported. Similarly, systolic blood pressure reactivity during the first minute of the CPT and during the participant's individual CPT duration across conditions revealed no significant differences in the deviation from baseline for participants in the awe (vs. control) condition, z = -.967, p = .33 and z = -.890, p = .37, respectively, indicating a lack of support for H3b. Finally, diastolic blood pressure reactivity during the first minute of the CPT and during the participant's individual CPT duration revealed no significant reduction in the deviation from baseline for participants in the awe (vs. control)

condition, z = -.596, p = .55 and z = -.108, p = .91, respectively, suggesting a lack of support for H3c.

Ancillary Analyses

Given I found no main effects by video manipulation—and in accordance with the preregistration—I evaluated whether associations between video manipulation and outcomes (i.e., VAS pain intensity, thresholds, tolerances, and HR, SBP, and DBP reactivity) were moderated by trait awe. Trait awe did not moderate the effect of condition on VAS scores (b = -5.03, SE = 4.47, t(126) = -1.12, p = .26), tolerances (b = 3.31, SE = 7.90, t(126) = .42, p = .68), HR reactivity during the first minute of the CPT (b = -1.08, SE = 1.93, t(124) = -.56, p = .57) or the duration of the participants' CPT (b = -1.20, SE = 1.85, t(125) = -.65, p = .52), SBP reactivity for the first minute of the CPT (b = 4.75, SE = 3.17, t(124) = 1.50, p = .14) or the duration of participants' CPT (b = 4.81, SE = 3.30, t(125) = 1.46, p = .15), or DBP reactivity for the duration of participants' CPT (b = 3.16, SE = 2.06, t(125) = 1.53, p = .13). However, trait awe did moderate the effect of condition on log-transformed time to pain threshold, b = .11, SE = .05, t(120) = 2.15, p = .03. Simple slope analyses indicated that condition was marginally associated with log-transformed time to threshold for people low (1 *SD* below the mean) in trait awe, b = -.10, SE = .06, t = -1.64, p = .10, suggesting that duration of CPT until threshold was longer for people with low trait awe in the control (vs. awe) condition (see Figure 2). However, condition was not associated with log-transformed time to threshold for people high (1 *SD* above the mean) in trait awe, b = .08, SE = .06, t = 1.41, p = .16, indicating that there was not a significant difference in the duration of the CPT until threshold for

people with high trait awe in the awe or control conditions. Additionally, trait awe marginally moderated the effect of condition on DBP reactivity during the first minute of the CPT, $b = 3.42$, $SE = 1.86$, $t(124) = 1.84$, $p = .07$. Simple slope analyses indicated that condition was marginally associated with DBP reactivity during the first minute of the CPT only for people high (1 *SD* above the mean) in trait awe, $b = 4.16$, $SE = 2.16$, $t = 1.93$, $p = .06$, suggesting that DBP reactivity during the first minute of the CPT was greater for people with high trait awe when in the awe (vs. control) condition (see Figure 3). However, condition was not associated with DBP reactivity during the first minute of the CPT for people low (1 *SD* below the mean) in trait awe, $b = -1.48$, $SE = 2.17$, $t = -.68$, $p = .50$, indicating that there is not a significant difference in the DBP reactivity during the first minute of the CPT for people with low trait awe in the awe or control conditions.

I also evaluated differences in anxiety across conditions to explore potential explanations for null effects. State anxiety scores followed a nonnormal distribution as shown by a Shapiro-Wilk test of normality ($p < .001$). A nonparametric Wilcoxon-Mann-Whitney test on state anxiety across conditions revealed that anxiety significantly differed across conditions, $z = -2.77$, $p = .006$. On average, participants in the awe condition experienced less self-reported state anxiety during the CPT than participants in the control condition (Table 3).

Finally, I explored whether differences in study variables exist across self-reported biological sex. I used nonparametric Wilcoxon-Mann-Whitney tests on awe, pain tolerance, HR reactivity during the first minute of the CPT or the duration of the

participants' CPT, SBP reactivity during the first minute of the CPT or the duration of the participants' CPT, DBP reactivity during the first minute of the CPT or the duration of the participants' CPT, and anxiety across self-reported biological sex. Generally, no significant differences were observed, all *p*s ≥ 25. However, tolerance differed across sexes, $z = -2.19$, $p = .03$, with males demonstrating longer time until tolerance on average ($M = 64.82$, $SD = 36.95$), compared to females ($M = 52.13$, $SD = 33.53$). Also, DBP reactivity during the first minute of the CPT and for the duration of the participants' CPT differed across sexes, , $z = -2.38$, $p = .02$ and $z = -2.37$, $p = .02$ respectively, with males having higher DBP reactivity than females during the first minute of the CPT ($M = 5.99$, $SD = 8.53$ vs. $M = 2.12$, $SD = 8.46$) and for the duration of the participants' CPT ($M = 7.45$, $SD = 9.73$ vs. $M = 3.01$, $SD = 8.99$). Finally, I explored whether differences in log-transformed thresholds existed across self-reported biological sex using an independent samples t-test. Log-transformed thresholds did significantly differ across biological sex, $t(122) = -1.81$, $p = .07$, with males having greater log-transformed durations until threshold ($M = 1.29$, $SD = .21$) than females ($M = 1.21$, $SD = .24$). Given sex differences on some of the key dependent variables, analyses including these dependent variables were redone, controlling for self-reported biological sex. However, no new significant effects of awe on the dependent variables were observed (*p*s ≥ .60).

Figure 2

Interaction between trait awe and condition with log-transformed time to pain threshold

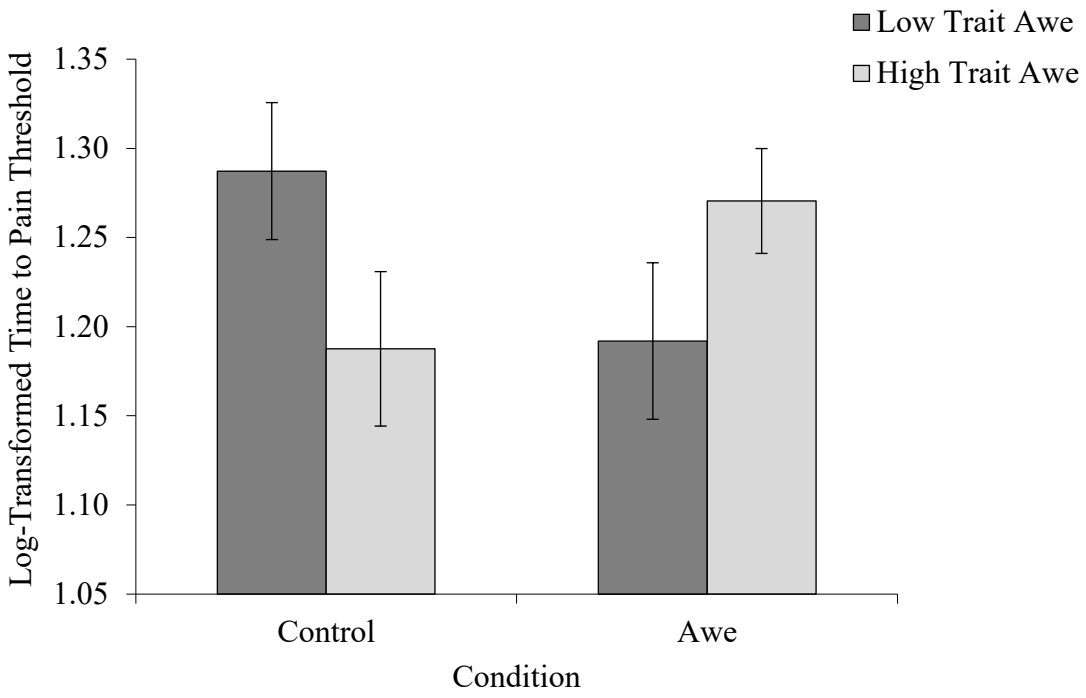
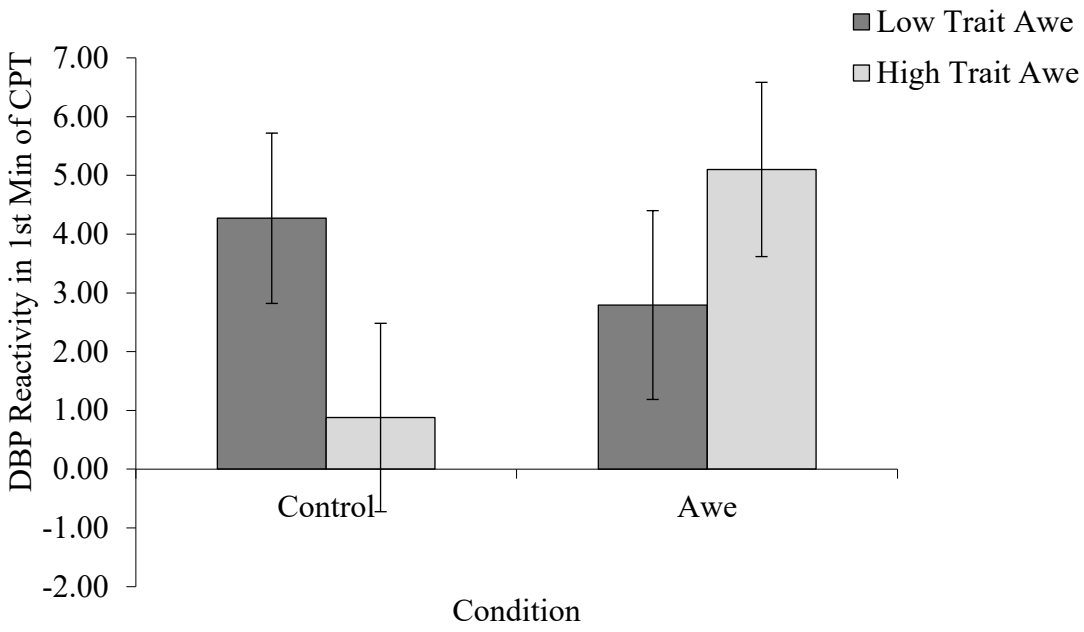


Figure 3

Interaction between trait awe and condition with DBP reactivity during the first minute of the CPT



Discussion

The current study investigated the effect of an awe manipulation (vs. control) on pain-related outcomes (i.e., subjective pain intensity, pain threshold/tolerance, and HR, SBP, and DBP reactivity) in response to a cold-pressor task. While the effects of positive affect (e.g., Finan & Garland, 2015) and other psychological factors (e.g., distraction; Bascour-Sandoval et al., 2019) have been studied in the context of analgesia, the current study is the first to explore the specific role of awe on acute, physical pain. Previous research has shown that positive affect should minimize pain-related outcomes when pain is experimentally manipulated (de Wied & Verbaten, 2001; Finan & Garland, 2015) and awe may be associated with lower inflammation (Stellar et al., 2015), thereby potentially less pain. However, current study findings suggest that awe may not serve as an analgesic. Relative to participants in a control condition, participants exposed to an awe manipulation during the experimental pain task did not have significantly lower pain intensity ratings, longer duration until pain thresholds or tolerances, or smaller deviations from baseline in heart rate, systolic blood pressure, or diastolic blood pressure.

A parsimonious explanation for the null results is that the experimental condition was not strong enough to increase state awe, which then limited the potential to detect differences in key outcomes as a function of the experimental condition. Specifically, the awe manipulation did not sufficiently and reliably induce greater reports of state awe among study participants as anticipated. Though the videos used in the current study appeared to differentially induce self-reported awe in previous research (Krenzer, 2018), a manipulation check demonstrated that state awe scores in the current study only

differed slightly between the awe and the control conditions. Therefore, null effects may largely be explained by a weak experimental manipulation.

Despite no evidence supporting primary study hypotheses, ancillary analyses with trait awe provide additional evidence that awe may not serve as an analgesic. Specifically, trait awe interacted with experimental condition to predict duration until pain threshold. Among people with low inclinations to experience awe, participants experienced shorter durations until threshold when they were in the awe condition than when they were in the control condition. These results indicate pain may be experienced more readily after noxious stimulation when exposed to an awe manipulation.

Additionally, trait awe interacted with experimental condition to predict diastolic blood pressure reactivity during the first minute of the CPT. Among people with high inclinations to experience awe, participants experienced greater DBP reactivity during the first minute of the CPT when they were in the awe (vs. control) condition. This finding indicates the physiological effects of pain may be greater when experiencing awe than when viewing the control nature video. Again, this suggests an awe manipulation may not provide analgesic effects. However, there may be an alternative explanation for why the physiological effects were greater in the awe condition for high-trait awe participants than participants with similarly high-trait awe in the control condition. Awe has frequently been considered a "complex emotion" characterized by both aspects of affective arousal and relaxation (e.g., Chirico et al., 2017; Takano & Nomura, 2023). Emotional arousal increases sympathetic activity (Hoehn-Saric & McLeod, 1988). Therefore, in the current study, participants with a high inclination towards awe that

experienced the awe manipulation may have experienced greater affective arousal—and subsequently demonstrated greater sympathetic activation as evidenced by elevated DBP—because of the manipulation compared to participants with high inclinations towards awe that experienced a neutral control video. However, this interpretation of the results contrasts with research demonstrating that awe produces sympathetic withdrawal and parasympathetic activation (Chirico et al., 2017). Thus, this current research may further contribute to the notion of awe as a "complex emotion" in need of further study and clarification of its physiological effects.

Results of the current study suggest there may be boundary conditions to the types of non-physical inputs that affect pain signaling (e.g., positive affect) and caution must be used when implementing psychological analgesic methods. According to Melzack and Wall's (1965) seminal gate control theory of pain, sensations from noxious stimuli can be blocked by non-noxious stimuli—including non-physical non-noxious stimuli—that override brain signals before painful input reaches the brain or through descending modulation from the brain to the periphery. Considerable research shows positive affect induction results in significantly less pain intensity in response to experimental pain tasks compared to neutral conditions (e.g., Roy et al., 2012; Rhudy et al., 2008; de Wied & Verbaten, 2001), thus supporting the notion that non-physical non-noxious stimuli are analgesic. However, the current study demonstrates that a specific form of positive affect—awe—may not be a sufficient form of non-physical non-noxious stimulation to override pain signaling and reduce pain intensity or other pain-related outcomes in response to a laboratory cold-pressor task. As a result of this study, it remains unclear

whether solely general positive affect or specific forms of positive affect reduce pain signaling and which forms of non-physical non-noxious stimuli serve as analgesics. As a result, it may be necessary to use caution with multimodal analgesics utilizing positive affect to ensure the techniques used effectively reduce pain. This caution is particularly warranted given the pattern of results from the ancillary analyses: The awe induction seemed to contribute to shorter durations until pain threshold for persons low in trait awe and greater DBP reactivity to painful stimuli for persons high in trait awe. The awe induction appeared to exacerbate pain-related outcomes for some people rather than alleviate pain. Further research on dispositional factors and psychological analgesics may be necessary to ensure that the benefits from multimodal analgesic techniques are generalizable.

Strengths, Limitations, & Future Directions

This study had several strengths. First, I utilized a stringent design to test the effects of awe on acute, physical pain. Namely, the stimuli in the experimental groups (i.e., awe and control) featured similar elements, such as nature sights and auditory stimuli, and only differed in the element of awe, providing a stringent test of the effects of awe on pain. Second, additional elements of the study allowed for a high degree of experimental control. Extraneous variables were limited by having a single researcher collect data from all participants using a scripted protocol and having CPT water temperature maintained at 4°C (± 1°C) for each participant with temperature checked before, during, and after each participant's CPT. Moreover, physiological data was collected continuously throughout a baseline period and during the CPT using a

continuous blood pressure monitor rather than taking infrequent, momentary blood pressure assessments, allowing for greater precision in the estimates of physiological reactivity.

Despite the many strengths of the study, there are also limitations to the current study. First, the awe manipulation did not sufficiently and reliably induce high rates of state awe among study participants, limiting the strength in detecting differences in pain-outcomes with the presence or absence of awe as shown by small effects (*d*s = .03) of condition on pain intensity ratings and thresholds. Another limitation of the study includes narrow generalizability of the study findings. This study was conducted in a single laboratory with only one type of experimental pain procedure. Further, I relied on data largely collected from an undergraduate psychology research pool (i.e., SONA; 53.85%) and the sample was largely female (66.92%), white (78.46%), and aged in the late teens to early forties (*M* = 21.67, *SD* = 4.792), limiting the generalizability of findings to a larger population and samples with other demographics.

Future research should consider addressing limitations of the current study. Specifically, future work should be conducted to improve and strengthen the awe manipulation or potentially create a different paradigm. Though the elements of the videos used in the current study were aimed at effectively portraying vastness, such as through dynamic shifts in scope of visual and audio features (Krenzer, 2018), in future research, researchers may manipulate awe to better target the two central themes of awe—perceived vastness and a need for accommodation (Keltner & Haidt, 2003). To better manipulate vastness, researchers may improve participant immersion in videos,

such as by using virtual reality (VR) modalities (e.g., Chirico et al., 2017), projecting video images to larger screens or surfaces, or improving the quality/sharpness of video images. Further, future awe manipulations may attempt to target the need for accommodation by introducing more novel experiences than those portrayed in the current video. One suggested way in which awe may be evoked other than nature is through cognitive elicitation via epiphany—the revelation of profoundness in the ordinary or routine (Keltner & Haidt, 2003). Thus, perhaps researchers may attempt to create epiphany moments for participants by teaching participants something unexpected. To do so, researchers may need to compile various pieces of unexpected information, assess participant knowledge, and tailor the presentations to participants based on participants' previous knowledge to create novel revelations or epiphanies. Another way researchers may attempt to introduce more novel experiences to participants other than nature videos could be to display moments of "moral beauty" or human goodness (Keltner & Haidt, 2003). Perhaps researchers may compile video clips in which participants could witness people helping others (e.g., picking up items that someone else dropped; Piff et al., 2015). Another way researchers may address limitations of the current study in future research includes employing different study designs to reveal more information about the effect of awe on pain-related outcomes. Given the current study was limited in generalizability, researchers may study the relationship between induced awe and pain outcomes with more diverse samples and/or alternative pain induction techniques (e.g., tonic thermal pain, pressure pain, nociception flexion reflex; Finan & Garland, 2015; Reddy et al., 2012). Finally, alternative study designs may further

contribute to scientific knowledge of the impact of awe on pain. Specifically, researchers may use ecological momentary assessment (EMA) to collect self-reported pain ratings immediately after experiencing awe in everyday life. In this type of study design, researchers may elect to recruit clinical samples, such as people who suffer from chronic pain, extending generalizability to samples including chronic pain sufferers.

The findings of the current study point toward the need for vigilance in implementing psychological analgesics within multimodal pain treatment, despite the initial intent to explore awe as a potential effective psychological intervention. This study significantly contributes to psychological pain research by highlighting a case in which a form of positive affect may not affect pain-related outcomes, leaving many unanswered questions and avenues for future research. Although this study did not find evidence for state awe as an analgesic, future work may consider strengthening the experimental manipulation and exploring potential individual differences in the experience of state awe and pain.

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