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Social support, but not neutral non-support, attenuates both stress and pain following a cold pain task:
Evidence for the implementation of social support alongside medical treatment regimens

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Social support, but not neutral non-support, attenuates both stress and pain following a cold pain task:
Evidence for the implementation of social support alongside medical treatment regimens

by

Matthew Heard Roberts

Social support has been demonstrated to attenuate pain and stress independently. The lack of a neutral non-support group to control for the effect of another person’s presence on pain and stress reduction has made understanding the mechanisms of social support’s efficacy challenging. In particular, it is currently unknown whether it is merely the presence of another person in the room or the active social support that is responsible for producing anti-nociceptive effects. The current study examined the role of social support in the modulation of stress and pain following a cold pressor task. Following baseline rest, healthy female undergraduates underwent the pain task alone (n=25), or with a confederate in the room providing either no support (non-support condition; n=25) or verbal emotional support (support condition; n=26). Every 20 seconds during the pain task participants silently rated their pain levels on a visual analog scale (VAS). Immediately following completion of the task, participants separately rated the intensity and the unpleasantness of the pain with a VAS. Cardiovascular measures of heart rate (HR) and blood pressure (BP), as well as subjective ratings of mood and anxiety, were assessed both at baseline and immediately following the pain task. Results indicated that participants in the support condition had attenuated delta (stress minus baseline) systolic BP and delta HR, responses to the pain task, and also reported less overall pain, pain intensity, and pain unpleasantness compared to both control conditions. A post-task assessment revealed that participants in the support condition reported less task difficulty, tension, and effort than both control groups. No group differences in delta subjective ratings of affect or anxiety were found. This study is the first to demonstrate that social support, not simply the presence of another person, attenuates both pain and cardiovascular stress in response to a laboratory pain task. Given the negative effects of stress on health and treatment outcome, these findings bear significant clinical implications for the incorporation of active social support into painful medical procedures and standard pain treatment regimens.
Introduction

Stemming from Hippocratic teachings, the concept of *primum non nocere* (first do no harm) remains a fundamental cornerstone of modern medical ethics, driving the balance between maximizing treatment efficacy and minimizing procedural invasiveness (Smith, 2005). The obligation to mitigate pain and stress during treatment, however, is more than an ethical consideration. Several studies and meta-analyses have demonstrated that increased stress levels during the course of a patient's treatment have widespread, deleterious effects on the both the patient's health and recovery (Detillion, Craft, Glasper, Prendergast, & DeVries, 2003; Schelling et al., 2003; Christian, Graham, Padgett, Glaser, & Kiecolt-Glaser, 2006; Lupien, Maheu, Tu, Fiocco, & Schramek, 2007). Citing the neurological overlap between stress, pain sensation, and general well-being, Riva, Wirth, and Williams (2011) argue that modern healthcare needs to place greater emphasis on the psychosocial health of each patient, and that further research is needed in this area. Given the central role that social relationships play in psychosocial and physiological health (House, Landis, & Umberson, 1988), the major purpose of the current project was to demonstrate that social support has the ability to simultaneously attenuate both pain and stress responses, making it an ideal candidate for implementation into the healthcare setting. In doing so, the present study also attempts to reconcile the methodological inconsistencies and oversights that have made drawing meaningful conclusions about the effects of social support on stress and pain difficult.

Stress & Social Support

In response to external stressors, either real or imagined, the body engages sympathetic stress networks across both the peripheral nervous system and the
hypothalamic-pituitary-adrenal (HPA) axis (Matteri, Carrol, & Dyer, 2000). Sympathetic nervous system activation induces a rapid release of epinephrine from the adrenal medulla, preparing the body for the fight or flight response (Charmandari, Tsigos, & Chrouso, 2005). Concurrently, the paraventricular nucleus of the hypothalamus releases corticotropin-releasing hormone (CRH), which subsequently triggers the release of adrenocorticotropic hormone (ACTH) in the anterior pituitary (Dallman & Hellhammer, 2011). Receptors in the adrenal cortex then bind with ACTH, stimulating the release of cortisol, which triggers a breakdown of glycogen into glucose in the liver and muscle tissue, suppresses immune function, and regulates activation of the sympathetic nervous system (Brown & Fisher 1985). Through these various processes, sympathetic and HPA axis activation leads to increased heart rate, blood pressure, and eventually salivary cortisol levels—all of which are commonly used to gauge physiological stress (Rozanski, Blumenthal, Davdison, Saab, & Kubzansky, 2005).

In evolutionary terms, this physiological stress mechanism is both essential and effective in its ability to prepare the body to respond appropriately to external threats. Even at low levels, however, persistent activation of the stress response has been causally linked to widespread health problems. These issues can include, but are not limited to, higher rates of depression, mental disorders, increased risk of stroke and coronary heart disease, decreased immune function, increased susceptibility to cancer and infectious disease, as well as negative changes in daily behavior and mood (Glaser et al., 1987; Anashensel, 1999; Bao, Meynen, & Swaab, 2007; Jones, O’Connor, Conner, McMillan, & Ferguson, 2007; Dimsdale, 2008). Moreover, these detrimental effects are often exacerbated when coupled with psychophysically stressful, invasive medical
treatments such as surgery, chemotherapy, or pharmaceutical regimens (Christian et al., 2006).

Growing evidence from laboratory-induced stress paradigms suggests that social support may be a cost-effective and non-invasive method to reduce both cardiovascular and adrenocortical indicators of stress during medical treatment. As a multidimensional, dynamic, and social phenomenon, Cobb (1976) operationally defined social support as any number of supportive interactions that leads the subject to “believe he is loved, esteemed, or valued, and that he belongs to a network of…supportive persons to [help] deal with crisis and change” (p. 300). At the theoretical level, it has been hypothesized that social support is capable of buffering stress because it provides someone under duress with an effective psychological coping mechanism, thereby reducing the perceived threat level of the stressor (Cohen & McKay, 1984; Thoits, 1995).

There is also a strong empirical basis substantiating the idea that a psychological variable such as social support may have strong physiological effects. In a study of college women, Christenfeld et al. (1997) demonstrated that verbal social support from a friend or stranger significantly attenuated participants’ cardiovascular response to a speech stressor compared to controls that did not receive social support. Another study by Heinrichs, Baumgartner, Kirschbaum, and Ehlert (2003) demonstrated that verbal social support provided during a standardized stress task led to blunted levels of free salivary cortisol compared to those who underwent the task alone. These findings have been further substantiated by a meta-analytical study on the effects of social support on stress by Thorsteinsson and James (1998), which concluded that, across 22 independent studies, social support had a moderately strong effect size (0.61) in its ability to diminish
cardiovascular stress responses, and an even stronger effect size (0.83) in its ability to attenuate cortisol responses to laboratory stressors. Furthermore, increased daily social support has been linked to decreased morbidity and mortality (Berkman, 1985; Uchino, 2006), facilitated wound healing (Christian et al., 2006), decreased inflammation (Mezuk, Diez-Roux, & Seeman, 2010), in addition to many other positive life-outcomes (as reviewed by Uchino, Cacioppo, & Kiecolt-Glaser, 1996). Although these studies have not yet been conducted in hospital settings or in clinical trials, they provide a strong empirical basis for the implementation of social support as supplementary to current medical treatment regimens.

Though the physiological pathways involved in the human stress response are fairly well understood, there is less clarity regarding the precise mechanisms through which social support influences this extensive chain of events. At a conceptual level, social support has both a main effect and buffering effect through which it bolsters health and mitigates the negative effects of the body’s cardiovascular and adrenocortical stress responses (Cohen, 1988; Uchino, 2006, Ditzen & Heinrichs, 2013). The buffering effect represents the protective influence of social support that reduces an individual’s psychophysiological response during or after a stressful event (Wheaton, 1985). In contrast, the main effect of social support is defined as its beneficial influence on a wide range of health indices, independent of the presence of a stressor (Ditzen & Heinrichs, 2013). The main effect of social support is determined by a person’s perceived access to social support, i.e. the support a person feels he or she receives throughout day-to-day life (Wills & Shinar, 2000).
It is important to note that these two salutogenic effects of social support are not mutually exclusive, but rather work concurrently much of the time and may share similar mechanisms (Ditzen & Heinrichs, 2013). Because perceived access to social support can affect psychological health, anxiety levels, hypertension, immune function, etc., it can mediate the body’s stress response (Kiecolt-Glaser, Gouin, & Hantsoo, 2010; Heard, Whitfield, Edwards, Bruce, & Beech, 2011). It is possible, therefore, that perceived access to social support (i.e. the main effect of social support) may influence the efficacy of the pain- and stress-reducing effects of social support during stress (i.e. the buffering effect). Unfortunately, only one behavioral study to date has examined perceived access to social support when measuring the buffering effect of laboratory-induced social support on stress. The results of this study by Schwerdtfeger and Schlagert (2011) suggest that the buffering effect of social support may only be effective in reducing laboratory-induced cardiovascular stress in individuals with high levels of perceived access to social support. Contrastingly, individuals with low perceived access to social support did not show reductions in cardiovascular stress when given support during a stress task (Schwerdtfeger & Schlagert, 2011). If perceived access to social support can moderate the buffering effect, as these results suggest, then perceived access to social support may have been an unknown third variable in all the previous research on social support and stress in which it was not assessed.

Furthermore, only one neuroimaging study has examined the physiological link between the main and secondary buffering effects of social support. Using functional magnetic resonance imaging (fMRI), Eisenberger, Taylor Gable, Hilmert, and Lieberman (2007) found that social support in day-to-day life is negatively correlated with levels of
neural activity in the dorsal anterior cingulate cortex during a stress task (dACC), a region highly associated with the regulation of autonomic stress responses, as well as Brodmann’s Area 8, a region associated with uncertainty and distress (Volz, Schubotz, & von Cramon, 2005). Based on the lack of experimental evidence in this area, Uchino, Carlisle, Birmingham, and Vaughn (2011) argue that further research is warranted to examine the role that perceived access to social support may play in mediating or modulating the buffering effect of social support on stress.

**Pain & Social Support**

In the human body, the process of nociception begins when free nerve endings located in dermal tissue or other organs become activated by the presence of a noxious stimulus, be it mechanical, thermal, or chemical (Belmonte & Cervero, 1996; Hucho & Levine, 2007). Once transduced, the electrical signal travels up afferent nociceptive fibers, terminating at the contralateral dorsal horn of the spinal cord where the information is then relayed to the hypothalamus and thalamus via the spinothalamic tract (Ruda, Bennet, & Dubner, 1986). The direct connection from the spinal dorsal horn to the supraspinal autonomic control centers of the hypothalamus are subsequently responsible for triggering the autonomic stress response following the sensation of pain (Kandell, Schwartz, & Jessel, 2000). Because human pain sensation has three distinct perceptual components, the pain pathways diverge after passing through the thalamus (Smock, 1999). The first aspect of pain perception, the purely sensory aspect of the intensity of the pain, is relayed from the ventral posterior thalamic nucleus to the primary and secondary somatosensory cortices where the source of the pain is localized (Kandell, Schwartz, & Jessel, 2000). The second aspect of pain perception is the emotional unpleasantness
associated with the physical pain, processed primarily in the anterior cingulate cortex and insular cortex (Treede, Kenshalo, Gracely, & Jones, 1999). The final type of pain perception, the association of pain with its potential emotional long-term implications, such as depression in response to chronic pain, is processed in the prefrontal cortex (Price, 2000). Despite the fact that the separate perceptual components of pain are physiologically distinct, all three dimensions of pain are attenuated by social support (Brown, Shffield, Leary, & Robinson, 2003; Holtzman et al., 2004; Eisenberger et al., 2011).

The link between social support and pain was first proposed by Panskepp, Herman, Conner, Bishop, and Scott in 1978 after realizing that opiates were as effective for the treatment of pain as they were for calming socially isolated animals. It was not until much later, however, that behavioral studies were able to support this theory (Paulson & Altmaier, 1995), and an additional fifteen years until neuroimaging studies were able to confirm the presence of a physiological link between the two (Younger, Aron, Parke, Chatterjee, & Mackey, 2010). In fibromyalgia and cancer patients, social support is negatively associated with the intensity of chronic pain sensation (Zaza & Baine, 2002; Holtzman, Newth, & Delongis, 2004). More recently, social support has also been demonstrated to lessen pain sensation and increase pain thresholds during laboratory-induced thermal and cold pain tasks (Brown et al., 2003; Montoya, Larbig, Braun, Preissl, & Birbaumer, 2005). Furthermore, Master et al. (2009) substituted direct social support (having a person present in the room providing social support) with a photograph of a significant other during both a thermal and cold pressor task (CPT). They found that merely having a photograph of their significant other attenuated pain
perception as much as holding hands with the significant other during each task (Master et al., 2009).

Two recent neuroimaging studies have revealed some of the brain regions associated with social support and its connection to pain processing. Using functional magnetic resonance imaging (fMRI), Younger et al. (2010) found that, compared to control photos of a non-romantic acquaintance, presenting pictures of a person’s significant other during a pain task led to decreased activation of nociceptive relay centers in the thalamus and autonomic control centers in the brainstem. In addition, increased blood-oxygen-level-dependent (BOLD) activation in several dopaminergic reward centers of the brain was only observed in the social support condition, but not the visual distraction condition, leading the authors to conclude that distraction could not have been responsible for the observed analgesic effects of social support (Younger et al., 2010). A similar experiment conducted by Eisenberger et al. (2011) found that social support during a thermal pain task was negatively correlated with BOLD activation in the anterior cingulate cortex (ACC) and anterior insula, regions directly associated with the perceptual unpleasantness of pain. Social support during this task was also associated with increased activity in the ventromedial prefrontal cortex (vmPFC), an area associated with safety signaling and fear extinction. Moreover, there was a significant decrease in pain unpleasantness ratings across all participants in the support condition compared to controls that did not receive support (Eisenberger et al., 2011).

**Methodological Issues in Current Literature**

Although these neuroimaging studies have gradually begun to identify the brain regions associated with the buffering effect of social support, there is still a large gap
between pain and stress research with regard to social support. This lack of understanding concerning social support and its anti-nociceptive and stress-reducing effects can be broken down into operational and conceptual issues.

The first is an operational problem: much of the current literature on the stress- and pain-reducing effects of social support has incongruous and divergent methodologies. The lack of methodological uniformity between pain and stress research with regards to social support has made it unfeasible to conclude that social support could concurrently attenuate both pain and stress. Moreover, a lack of control for potential confounds in previous research has resulted in third variable problems, further blurring the relationship between social support and the reduction of pain and stress.

The first methodological incongruity addressed in the present study pertains to the question of who should provide the support to participants. It has been hypothesized that support from one’s close friends or significant others would be the most genuine (and thereby the most effective) support (Glynn et al., 1999). Yet, this type of study design introduces a wide range of third-variable problems (i.e., how close the participant is to their supporter, the length of their relationship, how supportive the person is, how much the supporter says, etc.). Christenfeld et al., (1997), however, demonstrated that verbal support from either a close friend or confederate equally attenuated cardiovascular stress measures compared to an alone condition. As such, the present study used trained confederates pretending to be participants for the support and non-support conditions. This provided precise control over the timing, quality, and quantity of the support given, while simultaneously eliminating the third-variable problems associated with using friends as supporters. Though confederates have been employed in social support and
stress research (e.g., Glynn, Chirstenfeld, & Gerin, 1999; Hilmert, Kulik, & Christenfeld, 2002), no previous research has used a confederate to examine the effects of social support on pain. Additionally, though one previous study assessing the effects of social support on stress levels has controlled for the presence of another person in the room (Phillips, Gallagher, & Carroll, 2009), this experimental design has not been implemented in a study assessing the effects of social support on pain perception. Without the inclusion of a non-supportive control group alongside the standard alone control, there is no way to determine whether it is the social support, and not merely the comfort of having another individual in the room, that is responsible for attenuating pain and stress. The present study therefore implemented a third, neutral non-support condition, to eliminate the presence of another person in the room as a potential confound. Consequently, the present study is the first to unify the support conditions between stress, pain, and social support research.

More concerning still, of the many stress studies in which a supporter was present during the stressor, less than 10% controlled for evaluation effects (Thorsteinsson & James, 1999), a phenomenon in which the subject feels like he or she is being evaluated by the supporter, often confounding the beneficial buffering effects of social support (Fontana et al., 1998). Due to the evaluative nature of stress tasks implemented in social support research, the present study utilized a pain task as a stressor. Doing so not only allowed us to test the effects of social support on both stress and pain with a single stressor, but also reduced the risk of evaluation effects. This was accomplished by having participants silently rate their pain by pointing to a pain scale that was out of view of the confederate (if present). Lastly, only two studies to date have evaluated the modulatory
role of perceived access to social support on the buffering effect of social support on stress and pain, something the present study addressed.

Due to the extremely complex interplay between human stress responses, nociception, and perceptions of social support, it is very difficult to predict how social support may affect pain and stress at the same time. This methodological issue is central to the second, more conceptual problem: although social support has been shown to independently reduce pain and stress in separate studies, it is not known whether the same buffering can attenuate both variables concurrently. This uncertainty is due to a phenomenon known as stress-induced analgesia, in which heightened stress levels lead to decreased pain sensitivity (Butler & Finn, 2009; Yilmaz et al., 2010). Though the complete mechanisms behind stress-induced analgesia are not fully understood, its anti-nociceptive effects appear to occur as a result of activation of the descending inhibitory pain pathway (Butler & Fin, 2009). This relationship between higher stress levels and lower pain ratings, however, is at odds with the hypothesized blunting of both stress and pain in response to social support. Because participants in control conditions would be expected to have greater stress responses to the pain task (e.g., Gluck et al., 2004) than participants in support conditions, the stress-induced analgesia paradigm would predict the control group to have lower pain ratings. This, however, is at odds with the currently proposed capacity of social support to simultaneously attenuate both stress and pain. Given the high level of individual variability in stress-induced analgesia (Butler & Fin, 2009), as well as evidence from recent literature suggesting that stress-induced analgesia is inconsistently induced in laboratory settings, it is unclear whether or not social support would be able to concurrently buffer both the pain and stress response (Rhinehardt,
Kleindienst, Treede, Bohus, & Schmahl, 2013). The present study is the first to potentially demonstrate that social support can overcome stress-induced analgesia by reducing both stress and pain concurrently.

Our primary research questions were as follows: 1) can social support concurrently attenuate both stress and pain responses following a cold pain task, and 2) how does perceived access to social support influence the relationship between social support, pain, and stress? We hypothesized that participants in the support condition would have significantly attenuated levels of heart rate and blood pressure, as well as decreased pain ratings compared to the alone and non-support groups. We also predicted that, based on the results of Schwertfeger and Schlagert (2011), perceived access to social support would moderate the efficacy of social support on pain and stress levels.

Methods

Participants

Female undergraduates (n = 76) between the ages of 18 and 21 responded to an advertisement for research investigating the influence of the menstrual cycle on pain and stress. Based on self-reported medical history during an online screening process, participants were excluded based on a history of any of the following in the past year: high blood pressure, a chronic pain condition, any cardiovascular, seizure, neuroendocrine, respiratory, or gastrointestinal disorder, hepatic or renal impairment, or Raynaud’s disease. Any participants who reported suicidality, severe depression or anxiety, smoked tobacco, regularly took any neural stimulants (e.g., for ADHD), blood pressure medication, or psychotropic medication for anxiety or depression were also excluded. Oral contraceptive use was assessed as a potential covariate, but was not
exclusionary. The research was approved by the college’s Institutional Review Board, and all participants received partial course credit for their time.

Support Manipulation

Each participant was randomly assigned to one of three support conditions: alone (n = 25), nonsupport (n = 25), or support (n = 26). In the alone condition, the participant completed the testing session with only the experimenter present. Participants in the nonsupport and support conditions completed the testing session alongside a female confederate, who participants believed was also taking part in the study. All research assistants were thoroughly trained for their roles as confederates in the nonsupport and support conditions in order to maintain consistency between trials and between research assistants.

During the pre-task wait period and cold pressor task, confederates in the support conditions periodically provided scripted statements of verbal emotional support, (e.g., “You’re doing great! Remember you’re not alone.”), and congratulated participants upon completion of the task. In the non-support condition, the confederate did not interact with the participant in any way and was instructed to read a magazine while in the same room.

Questionnaires & Surveys

Prescreening questionnaire. All prospective participants completed an online questionnaire containing basic health and demographic information relevant to the exclusionary criteria and research questions. Due to the effects of depression (Bär et al., 2007; Ang et al., 2011), trait anxiety (Schmidt & Cook, 1999), and perceived stress (Crowley et al., 2011) on laboratory pain and stress, each of these psychological variables were assessed in the present study. Severity of depression was quantified using the Beck
Depression Inventory (BDI), a 21-question multiple-choice survey. Trait anxiety levels were measured using the Spielberger Trait Anxiety Inventory (STAI trait), a 20-question Likert-scale questionnaire. Self-perception of stress was quantified using the Perceived Stress Scale (PSS), a 10 question multiple-choice inventory. Finally, perceived global access to social support was quantified using the Interpersonal Support Evaluation List (ISEL), a 40-question inventory that measures access to social support along four distinct subscales. To control for the effects of birth control on salivary cortisol levels, participants identified in the prescreening questionnaire whether or not they used birth control, and if so, what type.

**Baseline questionnaires.** As previous studies have suggested that baseline affect (Papousek et al., 2010), state anxiety (Thompson, Keogh, & French, 2011), and sleep quality (Goodin, Smith, Quinn, King, & McGuire, 2012) have been shown to affect pain and stress, participants completed three questionnaires assessing each of these prior to the baseline rest period. Affect was analyzed with the Positive and Negative Affect Schedule (PANAS), a 20-item multiple-choice survey. State anxiety levels were assessed with the Spielberger State Anxiety Inventory (STAI state), a 20-question Likert-scale questionnaire. Recent sleeping habits and sleep quality were measured by the Pittsburg Sleep Quality Index (PSQI), a 10-question varied-response survey.

**Post-task questionnaires.** Following the CPT, participants completed a cold-pressor task assessment, in which they had to rate the difficulty, tension, effort, and concentration levels felt during the CPT on a VAS scale from 0-10. Participants also completed a second iteration of the PANAS and STAI-state to determine how affect and anxiety levels differentially changed across conditions. Additionally, participants
completed a brief manipulation check of how supported they felt during the CPT on a series of 7-point Likert scales across six different axes: supportive-unsupportive, friendly-unfriendly, accepting-rejecting, close-distant, warm-cold, and helpful-unhelpful.

**Experimental Protocol**

**Testing session & participant arrival.** Eligible participants were scheduled for a 90-minute laboratory session starting between 1:30 and 4:30 pm during the first 14 days (non-luteal phase) of their menstrual cycle. On the day of testing, participants refrained from exercising strenuously, drinking more than a single caffeinated beverage in the morning, eating or drinking (except water) one hour prior to the study, consuming any alcohol 12 hours prior to the study, or taking any antihistamines, pain medication, and neural stimulants. The experimenter obtained verbal confirmation that participants had followed all testing-day requirements during the laboratory visit.

Upon arrival, the experimenter guided participants in the nonsupport and support conditions to a waiting room and informed them that the second participant (confederate) had not yet arrived. Following the arrival of the confederate, both the participant and confederate completed informed consent forms. The experimenter then asked the participant and confederate to draw cards to determine who would complete the task first. The card draw was rigged to ensure that the real participant always went first. In the alone condition, participants signed consent forms immediately upon arrival.

**Baseline rest and pre-task instructions.** Following consent, the experimenter escorted the participants and confederates (if present) to separate rooms in the lab area where they started working on the baseline questionnaires. The participants then began a ten minute baseline rest period, during which an automatic blood pressure cuff collected
cardiovascular measures. At the end of the rest period, participants provided a baseline salivary sample. Following an explanation of the cold pressor task, participants underwent a 5-minute wait period either alone (alone condition) or with the confederate present (nonsupport & support conditions) while the experimenter prepared the cold water bath. In the support and non-support conditions, the confederate read her magazine silently during this time. In the support condition, confederates engaged the participant briefly to wish her good luck.

**Cold pressor pain task.** Participants then underwent a cold pressor task (CPT) by submerging their dominant hand up to the wrist in a circulating tank of water at 4.0 (± 0.1) °C for up to three minutes or until they were no longer willing or able to tolerate the pain (tolerance). During the task, participants nonverbally rated their pain every 20 seconds by pointing to a VAS scale from 0 (no pain) to 100 (most intense pain imaginable) that was out of view of the confederate (if present) to eliminate evaluation effects. At the end of the three minutes or at tolerance, participants rated the intensity and unpleasantness of their pain on a separate VAS scale immediately before removing their hand from the water. After drying their hand, participants began their post-task questionnaires, starting with the task assessment.

**Post-task recovery.** In the support and non-support conditions, fifteen minutes after the start of the CPT, the experimenter escorted the confederate out of the testing area under the pretense that it was her turn to begin her baseline rest period. The participant continued to sit quietly in the testing area until all salivary samples had been obtained (see Cortisol Sampling below). Before debriefing the participants, the researcher asked follow-up questions to determine if the participant knew the true purpose of the
study. For the support and non-support conditions, the experimenter also asked what they thought the other participant’s (i.e., confederate’s) role was in the study. No participants correctly identified the purpose of the study or were aware of the confederate’s deception.

**Cardiovascular Sampling**

Cardiovascular measurements were taken at minutes 0, 5, and 10 during the baseline rest period, minutes 0 and 5 of the wait period, and subsequently every 5 minutes starting immediately after the participant removed her hand from the water until the end of the experiment. All cardiovascular measurements were obtained using a programmable automatic blood pressure cuff (OSCAR 2, Suntech Medical).

**Cortisol Sampling**

Saliva was collected in 1.5 mL Eppendorf tubes at the end of the baseline rest period and minutes 15, 20, 25, and 30 post-CPT. Saliva samples were frozen at -20°C immediately after collection to prevent enzymatic degradation. As this study is part of a larger parent project, the analyses of the sampled salivary cortisol will be presented in a future publication.

**Data Analysis**

Group differences by support condition in demographic factors and subjective measures (perceived stress, depression, sleep quality, perceived access to social support, baseline state anxiety, trait anxiety, and affect) were examined using a multivariate ANOVA for continuous variables and chi square analyses for dichotomous variables as appropriate. Group differences in baseline cardiovascular measures were also
investigated using a multivariate ANOVA. Where significant results emerged, post-hoc analyses with Bonferonni corrections were conducted.

Because significant group differences existed in perceived stress (see Results below, Table 1), all hypothesis testing was conducted using perceived stress as a covariate. Multivariate ANCOVAs were run to analyze group differences in pain intensity, pain unpleasantness, mean CPT pain (mean CPT pain was calculated by averaging each participant’s pain ratings during the CPT), time to tolerance, as well as perceived task difficulty, tension, concentration, and effort. Separate ANCOVAs were also used to analyze delta scores (stress minus baseline) for systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), negative affect, positive affect, and state anxiety. Where group differences emerged, Sidak corrected post-hoc comparisons were performed.

In order to determine if perceived access to social support had a moderating effect on pain ratings or physiological stress measures, a quartile split of perceived access to social support was conducted. A multivariate ANCOVA was then used to determine if the relationship between support condition and pain and physiological stress variables depended on whether participants reported high (4th quartile) or low (1st quartile) perceived access to social support.

**Results**

**Demographics and Subjective Measures**

No significant group differences emerged for oral contraceptive use or racial ethnicity, Table 1. Furthermore, BMI, age, trait anxiety, depression, sleep quality, perceived access to social support, baseline state anxiety, affect, SBP, DBP, and HR did
not differ between groups ($ps > .05$), Table 1. There were, however, significant group differences in perceived stress, $F(2,72) = 3.91, p = .024, \omega^2 = .07$, as the alone group had significantly higher perceived stress than the non-support group, $p = 0.021$, Table 1.

**Cold Pressor Pain Task**

Analyses revealed significant main effects of support condition on pain intensity ($F(2,72) = 17.17, p < .001, \omega^2 = .30$), unpleasantness ($F(2,72) = 15.68, p < 0.001, \omega^2 = .28$), and mean pain during the CPT ($F(2,72) = 37.87, p < .001, \omega^2 = .50$), Figure 1. In each case, the support condition had lower pain ratings than either control condition ($ps < .001$). Analysis of task assessment revealed a main effect of support condition on CPT difficulty ($F(2,72) = 16.873, p < .001, \omega^2 = .30$), tension ($F(2,72) = 7.967, p = .001, \omega^2 = .16$), and effort, ($F(2,72) = 26.961, p < .001, \omega^2 = .41$), Figure 2. Subsequent post-hoc analysis indicated that, in each case, the support conditions had lower task ratings than either control group. Concentration and time to tolerance, however, did not differ between support conditions, ($p > .05$). As expected, a main effect of support condition on subjective feelings of social support during the task was observed ($F(2,71) = 17.48, p < .001, \omega^2 = .30$). Post-hoc analysis revealed that the support group felt significantly more supported than the alone and non-support groups ($p < .001$).

**Cardiovascular Responses to Cold Pressor Pain Task**

Analyses revealed a main effect of delta SBP ($F(2,72) = 9.042, p < 0.001, \omega^2 = .18$) and HR ($F(2,72) = 15.01, p < .001, \omega^2 = .27$), as the support condition showed attenuated increases compared to both control conditions ($ps < .01$), Figure 3. There was also a marginally significant trend for a main effect of delta DBP, $F(2,72) = 3.067, p = .053, \omega^2 = .05$, in which the support condition had marginally lower delta DBP than the
alone condition \((p = .062)\). Furthermore, no main effects of delta state anxiety, positive affect, or negative affect were found \((ps > .05)\).

**Perceived Access to Social Support**

Perceived access to social support did not independently affect any pain and stress variables and did not moderate the relationship between social support condition and pain and stress outcomes \((ps > .05)\).

**Discussion**

Our hypothesis that social support would significantly attenuate cardiovascular responses to cold pressor pain relative to those who did not receive support was substantiated, as participants in the support group had significantly lower delta SBP and delta HR than those in the alone and non-support conditions. Our second hypothesis that social support would reduce pain perception during the CPT compared to those who did not receive support was also supported. Participants in the support condition had significantly lower ratings of pain intensity, unpleasantness, and mean pain during the CPT compared to those in the alone and non-support conditions. Our final hypothesis that the effects of social support on pain and stress would be moderated by perceived access to social support was not substantiated, as no significant interaction was found between support condition and perceived access to social support on pain ratings or cardiovascular stress.

Our finding that social support resulted in attenuated cardiovascular stress levels following a stress-inducing laboratory pain task is consistent with previous research on the buffering effect of social support on physiological stress (Kirschbaum et al., 1995; Fontana et al., 1998; Glynn et al., 1999; Hilmert et al., 2002; Phillips et al., 2009). In
contrast, however, McClelland & McCubbin (2008) also assessed the effects of social support on stress following a pain task, though they found no effect of social support on cardiovascular responses. When interpreting this finding, however, one must take into consideration that supporters and participants were not allowed to make eye contact or interact in any way throughout the entire experiment (McClelland & McCubbin, 2008). In this way, their support condition was functionally no different than the non-support condition in the present study, for which no buffering effect of social support was found. It is therefore not surprising that they did not find a causal link between social support and cardiovascular stress following a pain task (McClelland & McCubbin, 2008). In contrast to the current report, the majority of these previous studies used a speech task to induce acute stress in the laboratory (Thorsteinsson & James, 1999). Thus, the present study is the first to demonstrate that social support has the ability to attenuate cardiovascular stress levels following a laboratory pain task. This finding is clinically relevant in a healthcare setting, where both painful and stressful procedures are commonplace and can lead to negative health outcomes.

While supported participants showed attenuated cardiovascular stress responses to the CPT in the present study, the support manipulation had no effect on self-reported changes in state anxiety or affect. Although Salovey, Rhothman, Detweiler, and Steward (2000) proposed a theoretical basis for the interaction between social support and emotional state, previous studies assessing changes in state anxiety and affect in response to social support have produced inconsistent results. Some have validated the presence of a buffering effect of social support on emotional state (Ditzen et al., 2008; Dizten & Heinrichs, 2013), while others have found no causal link between the two (Kamarck et
al., 1995; Gramer et al., 2010). One potential factor responsible for these inconsistencies in the literature could be differences in the length of the stressor. Standardized speech tasks like the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993) are approximately ten minutes in length, while the CPT lasts, at most, three minutes. Although this length of time is sufficient to induce a physiological stress response, it may not be sufficient to induce changes in psychological state. Furthermore, elapsed time from the end of the stress task to measurement of state anxiety and affect may have contributed to why the present and previous studies failed to find differences based on support manipulation. Because these subjective states are not assessed during the CPT itself, but rather up to 2 minutes following the task, participants' knowledge that the stress task is already behind them may reduce state anxiety and buffer changes in affect (Fontana et al., 1999).

In order to verify that differences in length of pain exposure between conditions did not influence stress, time to tolerance was assessed in the present study. Differences in length of exposure to the cold water during a CPT can influence measures of cardiovascular stress, in that the longer a participant leaves their hand in the water, the larger the cardiovascular stress response (Lowery, Fillingim, & Wright, 2003). No main effect of support condition on time to tolerance, however, was found. Thus, differences in exposure length did not contribute the differences observed in cardiovascular stress response between groups.

Our finding that participants in the support condition experienced significantly less pain than those who did not receive support is consistent with other studies analyzing the effects of social support on pain in a laboratory setting (Brown et al., 2003; Jackson et
al., 2005, Master et al., 2009; Younger et al., 2010). In each of these previous studies, however, support was provided by a close friend or significant other. The present study is therefore the first to demonstrate that social support from a same-sex confederate can significantly attenuate pain levels. Given that support from a confederate may be perceived as less meaningful as support from a significant other or close friend (Glynn et al., 1999), this is an important finding because friends and family may not always be available to provide support in a healthcare setting.

The fact that participants in the support condition exhibited not only reduced cardiovascular stress levels, but also reduced pain ratings compared to both control groups appears to directly oppose stress-induced analgesia (SIA). In SIA, increased acute stress elicits decreased sensitivity to pain (Butler & Finn, 2009). Although the SIA is known to inhibit descending pain pathways, little else is known about its mechanism (Yilmaz et al., 2010). Given that the alone and non-support conditions in the present study exhibited significantly higher levels of cardiovascular stress than those in the support condition following the pain task, SIA would predict that both control groups would have lower pain levels than the support group. Although the novel finding of the present study that social support can simultaneously reduce both pain and stress may appear to be incompatible with what is known about SIA, they are not entirely irreconcilable. For one, possible ceiling effects associated with the SIA paradigm could potentially explain these conflicting accounts. Although the support condition did have attenuated cardiovascular stress relative to controls, all three support conditions had increased cardiovascular stress over baseline. If SIA is activated by a threshold level of psychophysiological stress over baseline, and any additional stress does not increase its
anti-nociceptive effects, then this could explain our current findings, as SIA would have affected all groups equally. Even in such a case, only the active support could account for the lower pain ratings observed in the support group relative to controls. An alternate explanation for the present findings in light of SIA literature could be that the psychological and cardiovascular stress levels caused by the cold pressor task were not sufficient to trigger SIA at all. Still another possibility is that SIA was indeed greater in both control conditions than in the support condition, but the buffering effect of social support was so much stronger than the buffering effect of SIA on pain levels that the effects of SIA were not noticeable in comparison. Because the analgesia appears to involve decreased activity in the anterior cingulate cortex and insula, whether it is induced by SIA (Yilmaz et al., 2010) or social support (Eisenberger et al., 2011), it may not be possible to rule out SIA as a confound in social support and pain literature.

In addition to reduced pain and cardiovascular responses, participants in the support condition had significantly lower ratings of task difficulty, tension, and effort than those in alone and non-support conditions. Given that a person’s subjective experience during a procedure can often play a role in determining the outcome of medical treatment (Di Blasi, Harkness, Erst, Georgiou, & Kleijnen, 2001; Keefe, Rumble, Scipio, Giordano, & Perri, 2004; Sullivan et al., 2009), the fact that support from a stranger can bolster one's positive perception of the task is clinically relevant. Although the effects of social support on difficulty, tension, and effort during a pain task have not been evaluated before the present report, there are studies yielding mixed results that have analyzed task difficulty and effort following stress tasks involving serial addition (Phillips et al., 2009) or speech tasks (Gramer et al., 2010). Following the serial addition
task, participants receiving social support rated the task as both less difficult and requiring less effort than those who completed the task alone (Phillips et al., 2009), a result consistent with our own. In contrast, following a speech task, no differences were found in task difficulty or effort between support and alone conditions (Gramer et al., 2010). The fact that Gramer et al. (2010) did not control for evaluation effects, while Phillips et al. (2009) and the present study did, could potentially explain the lack of consistency between these studies, as participants may have felt evaluated, and therefore found the task as more difficult and requiring more effort.

The present study also found that perceived access to support was not found to moderate social support's ability to attenuate pain or stress. Although we expected to see that perceived access to social support moderated the strength of the buffering effect, the fact that this hypothesis was not supported only makes a stronger case for the inclusion of social support alongside medical treatment. The efficacy of the buffering effect of social support on pain and stress does not appear to be contingent upon high perceived access to social support for college females. Consequently, there may be little cause for concern that the provision of social support during medical treatment would only benefit a portion of this population (those with high perceived access to social support). Therefore, a greater number of patients than previously thought may benefit from social support in a healthcare setting. Due to the limited sample size and sample population of the present study, further research is needed to corroborate this conclusion.

This finding, however, contradicts the results of Schwerdtfeger and Schlagert (2011), showing that the buffering effect of social support on cardiovascular stress was only observed in individuals with high, rather than low, perceived access to social
support. There are, however, several distinct methodological differences between the study by Schwerdtfeger and Schlagert (2011) and the present report that could account for this inconsistency. First, participants in their study were not randomly assigned to conditions, but were rather asked by the experimenters prior to the study to assign themselves to either a social interaction or alone condition. This lack of random assignment could have inherently introduced several third-variable problems to their study, as the participants’ ability to find someone willing to accompany them to the study may have influenced their perception of access to social support. Because the participants knew that they could either participate in the study alone or with a supporter, demand effects could have influenced their pain and stress ratings, whereas participants in the present study were blind to the support condition and true purpose of the experiment. Additionally, the manipulation of support in their study lacked structure and consistency in terms of the participants’ interactions with the supporters (either partners, close friends, or fellow students) during a 10-minute rest period while the experimenter was out of the room (Schwerdtfeger & Schlagert, 2011). Importantly, this interaction cannot be considered to play a role in the buffering effect of social support, as there was no stressor for the social interaction to buffer. Instead, the interaction occurred during a rest period and before participants even knew they would undergo a speech stress task. Thus, the interaction that Schwerdtfeger and Schlagert (2011) actually found does not represent the interaction between perceived access to social support and the buffering effect of social support like the present study, but rather something else altogether.

The strengths of the present study include methodological implementations that allowed us to control for potential third-variables left uncontrolled in previous studies
assessing the effects of social support on both stress and pain. First, the inclusion of a neutral, non-support condition enabled us to verify that it is social support, and not the presence of another person, responsible for attenuating cardiovascular stress and pain ratings. Thus, if the buffering effect of social support occurs by providing the supported individual with an enhanced ability to cope with a stressful situation, we can now rule out the presence of another person as the source of this enhanced coping. In a healthcare setting where non-supportive others, be they medical staff or other patients, are almost always present, this is an extremely important distinction. Additionally, given the importance of having the support interactions seem genuine (Thorsteinsson & James, 1999), all confederates were rigorously trained by the experimenter over a four week period to ensure that their support was as genuine and natural in appearance as possible. This extensive training period also allowed verification that the support was consistent between confederates in terms of support statements, inflection, timing, and body language. Furthermore, in order to eliminate the confounding effects of evaluation on social support, in which a person's stress levels may be inflated if they feel that their performance is being judged during a difficult task (Thorsteinsson & James, 1999; Schwabe, Haddad, & Schachinger, 2008), all pain ratings made by participants were out of sight of the confederates. No participants in the support or non-support condition reported feeling evaluated by the other participant (i.e. confederate) or experimenter.

The effects of distraction on pain and stress could potentially be a serious confound in social support research, as both visual and auditory distraction can lead one's concentration away from the pain or stress of the present situation, thereby influencing buffering measures of pain and stress (Campbell et al., 2010; Wright & Raudenbush,
Given that verbal social support could create auditory distraction, the present study assessed task concentration between each support condition. As no group differences were found in concentration ratings, we can conclude that distraction did not act as a confound in our study.

Although the present study filled many methodological gaps and rectified several procedural inconsistencies between previous studies of social support's effect on stress and pain, there were still some methodological limitations worth mentioning. The biggest limitation of the present study was the inclusion of only female college students. Because we only had female laboratory assistants, were males to be included in the study, they would have been supported by female confederates. Males tend to artificially deflate their ratings of pain when in the presence of female strangers, perhaps in order to appear more macho (Levine & De Simone, 1991; Sheffield & Carroll, 1993; Lowery et al., 2003). As such, we would have been unable to determine the relative influence of social support on pain ratings in males potentially trying to appear more macho in front of a female.

Moreover, previous research has demonstrated that women respond more consistently to laboratory induced pain than men (Bär, Greiner, Letsch, Kobele, & Sauer, 2003), so the inclusion of only women in the present study helped to minimize unnecessary variability.

Another factor that limits the generalizability of the present study was the use of a three minute pain task in a controlled laboratory setting to elicit pain and stress responses. Though there is a strong case to be made that the same effects would exist in a hospital setting, this cannot be assumed as necessarily true until clinical trials assessing the
efficacy of social support in its ability to reduce pain and stress, and to improve treatment outcomes alongside standard medical treatments have been completed. Further research is still needed to gain a more holistic and comprehensive understanding of the effects of social support. A follow-up expansion of the present study would benefit from examining the effects of social support from either a male or female confederate on participants from both genders. As the present study only assessed the effects of social support on women of 18-21 years of age, the present study could also be expanded to assess the effects of social support on stress and pain across a wider age range of both participants and confederates. Furthermore, given the physiological differences between cold, thermal, and ischemic pain in terms of sensory processing mechanisms (Schull, Kaplan, & O'Brien, 1981; Lautenbacher, Rollman, & McCain, 1994; Girdler et al., 2005), additional studies examining other types of pain would be illuminating. In addition, future examination of cortisol stress levels in addition to cardiovascular measures would further strengthen the association between social support and the attenuation of stress. Most importantly, however, the effects of social support on pain and stress need to be examined in a clinical, hospital setting, as this will be the final proving ground before can be implemented alongside standard medical procedures.

One of the central purposes of interventional medicine is to reduce the pain, stress, and subjective experience of patients (Smith, 2005). The process of diagnosing, treating, or preventing most medical conditions, however, often requires some form of invasive medical procedure such as surgery, pharmaceutical drug regimens, physical therapy, or exposing the body to radiation, all of which can either directly or indirectly cause pain, stress, and patient discomfort. Not only are these effects of medical treatment
counterproductive to the central purpose of medicine in the short-term, increased stress also can undermine or even counteract the success of the treatment (Schelling et al., 2003; Christian et al., 2006).

In summary, the present study found that positive verbal emotional social support from a confederate significantly attenuated both cardiovascular stress, pain ratings, and subjective assessments of the pain task's difficulty, tension, and effort compared to controls who did not receive support or were alone during the task. Moreover, these robust findings cannot be explained by differences in length of exposure to the pain task, evaluation effects, demand effects, concentration, the presence of another individual during the CPT, or perceived access to social support. Given these results, there is a strong case to be made for the inclusion of social support alongside current medical treatment regimens. Doing so has the potential to not only decrease patients' pain during the course of treatment, but also to improve the success of procedural outcomes through social support's buffering effect on physiological stress. Whether it is doctors, nurses, newly hired additional hospital staff, or a combination of each that provide the extra social support to patients, any short-term incurred cost or loss in efficiency could be far outweighed by the salutogenic effects of social support.
Table 1. Mean (±SEM) of baseline demographic factors and subjective measures as a function of support condition

<table>
<thead>
<tr>
<th></th>
<th>Alone n=25</th>
<th>Non-Support n=25</th>
<th>Support n=26</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral Contraceptive Use (%)</td>
<td>10 (40%)</td>
<td>12 (48%)</td>
<td>14 (54%)</td>
</tr>
<tr>
<td>Racial Ethnicity (% Non-White)</td>
<td>7 (28%)</td>
<td>8 (32%)</td>
<td>9 (35%)</td>
</tr>
<tr>
<td>Age</td>
<td>18.80 (±0.18)</td>
<td>18.84 (±0.18)</td>
<td>18.92 (±0.17)</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>23.64 (±0.79)</td>
<td>23.48 (±0.63)</td>
<td>23.38 (±0.82)</td>
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<tr>
<td>Trait Anxiety</td>
<td>41.28 (±1.84)</td>
<td>37.24 (±1.67)</td>
<td>36.35 (±1.36)</td>
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<tr>
<td>Depression</td>
<td>10.16 (±1.50)</td>
<td>6.88 (±0.99)</td>
<td>6.92 (±0.81)</td>
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<tr>
<td>Sleep Quality</td>
<td>5.08 (±0.47)</td>
<td>4.84 (±0.44)</td>
<td>4.54 (±0.46)</td>
</tr>
<tr>
<td>Perceived Access to Social Support</td>
<td>23.48 (±0.64)</td>
<td>24.44 (±0.74)</td>
<td>25.26 (±0.58)</td>
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<td>Perceived Stress A</td>
<td>18.32 (±1.51)</td>
<td>13.24 (±1.09)</td>
<td>15.27 (±1.08)</td>
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<tr>
<td>Baseline State Anxiety</td>
<td>34.96 (±1.74)</td>
<td>30.60 (±1.19)</td>
<td>30.58 (±1.46)</td>
</tr>
<tr>
<td>Baseline Positive Affect</td>
<td>26.08 (±1.21)</td>
<td>28.48 (±1.29)</td>
<td>30.23 (±1.23)</td>
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<tr>
<td>Baseline Negative Affect</td>
<td>13.72 (±0.83)</td>
<td>11.68 (±0.39)</td>
<td>12.31 (±0.53)</td>
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<tr>
<td>Baseline SBP</td>
<td>119.72 (±1.45)</td>
<td>119.49 (±1.37)</td>
<td>118.64 (±1.47)</td>
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<td>Baseline DBP</td>
<td>70.93 (±1.42)</td>
<td>71.49 (±1.26)</td>
<td>71.09 (±1.23)</td>
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<td>Baseline HR</td>
<td>66.92 (±1.63)</td>
<td>67.60 (±1.38)</td>
<td>67.21 (±1.51)</td>
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<tr>
<td>Post-CPT State Anxiety</td>
<td>43.12 (±2.46)</td>
<td>38.72 (±2.30)</td>
<td>36.80 (±1.87)</td>
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<td>Post-CPT Positive Affect B</td>
<td>22.64 (±1.41)</td>
<td>27.48 (±1.59)</td>
<td>29.36 (±1.54)</td>
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<tr>
<td>Post-CPT Negative Affect</td>
<td>16.04 (±1.22)</td>
<td>14.44 (±1.24)</td>
<td>13.32 (±0.63)</td>
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<td>Post-CPT SBP B</td>
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<td>136.64 (±1.91)</td>
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<td>Post-CPT DBP</td>
<td>82.84 (±1.63)</td>
<td>82.40 (±1.60)</td>
<td>78.28 (±1.69)</td>
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<td>Post-CPT HR C</td>
<td>77.84 (±1.45)</td>
<td>78.28 (±1.36)</td>
<td>71.40 (±1.77)</td>
</tr>
</tbody>
</table>

A Alone > Non-Support, p<.05
B Support < Alone, p<.05
C Support < Alone and Non-support, p<.05
Figure 1. VAS ratings of mean (± SEM) pain intensity, unpleasantness, and mean pain during the CPT by support condition.

Main effect of group on pain intensity, unpleasantness, and mean CPT pain, $F(2,72) = 15.68 - 37.87$, $ps < .001$.
Support < Alone, $ps < .001$
Support < Non-Support, $ps < .001$
Figure 2. VAS ratings of mean (± SEM) task difficulty, tension, concentration, and effort by support condition.

Main effect of group on difficulty, tension, and effort, $F(2,72) = 7.97 - 26.96, ps < .01$.
Support < Alone, $ps < .01$
Support < Non-Support, $ps < .01$
Figure 3. Mean (+SEM) change (stress-baseline) in cardiovascular measurements by support condition.

Main effect of group on Delta SBP and Delta HR, $F(2,72) = 9.04 - 15.00, ps < .01$.
* Support < Alone, $ps < .01$
* Support < Non-Support, $ps < .01$
A marginally significant trend was found for diastolic blood pressure, $F(2,72) = 3.067, p = .053$
* Support < Alone, $p = .062$
References


