The Effects of Competition and Exercise on Pain Perception

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Abstract

One of the most ubiquitous examples of stress-induced analgesia that is easily observed is the pain reduction athletes experience during competition. There is anecdotal evidence for this phenomenon but there is an omission in the literature of conclusive systematic investigations of athletes' responses to noxious stimuli under competitive stress. The purpose of the present study was to examine the nature of stress involved in interpersonal competition, and to determine which component of athletic competition, psychological stress or physical exertion, is a trigger for endogenous pain inhibitory systems. The results demonstrate stress-induced analgesia as a result of strenuous exercise in athletes and non-athletes—an increase in pain threshold and a reduction in pain ratings that increases under competitive stress. The present study will add to the research on the contribution of stress to athletic competition induced analgesia and identifies how the circumstances necessary to elicit a stress-induced analgesic response during competition interact with gender and athletic status.
The Effects of Competition and Exercise on Pain Perception

In competition athletes will often continue to compete even after sustaining painful injuries. The ability of athletes to perform when injured is anecdotal evidence of pain modulation systems in humans responsible for a reduction in sensitivity to noxious stimuli. The effective inhibition of nociceptive afferent fibers is a form of endogenous analgesia. Environmental stressors such as threatening or aversive events can activate the mechanisms of this system. The existence of “stress-induced analgesia” has been well established in laboratory animals but few studies have reliable evidence of analgesic states in human subjects confronted with stressors in experimental or naturalistic situations. The objective of the present experiment is to systematically study the effects of competitive exercise on the perception of noxious stimuli.

Previous studies have shown that competing in athletic events can produce analgesic states in college athletes (Sternberg, Bailin, Grant, & Gracely, 1998; Sternberg, Bokat, Kass, Alboyadjian, & Gracely, 2001). Efforts were made to discriminate between the different components of competition, the physical exertion involved in competition and the cognitively stressful components of competition that are independent of exercise. Researchers demonstrated that there are differences in the analgesic effects produced by the different components of the experience of competition, and that these differences may be sex dependent. In females, a low intensity treadmill run produced analgesia, whereas in males only, sedentary video game competition had this effect (Sternberg et al., 2001). However, the exercise component of that previous study may not have been significantly stressful enough to elicit a stress-induced analgesic response in males. In response to these findings researchers suggest that exercise produces analgesia if, perhaps only if, it is construed as stressful (Sternberg et al., 2001).
Thus, based on previous research the hypothesis of the present study is that physical exertion must include some element of “stress” in order to activate pain suppressing mechanisms. The goal of the present study is to examine this phenomenon by manipulating the psychological state associated with interpersonal competition while holding physical exertion constant. In the current experiment, subjects will exercise and also participate in contrived competition while physical exertion remains the same under both conditions. This design will allow the experimenters to address the question of whether or not the exercise component of competition is solely responsible for the observed analgesic effects, or if there is something unique about the notion of interpersonal competition—if competition is an environmental stressor capable of activating the pain suppressing mechanism of stress-induced analgesia.

*Nociception, Pain and Analgesia*

The physiological system being studied in the current experiment is the neural mechanism of pain perception. Noxious stimuli applied to the skin activate specialized sensory receptors called nociceptors (Basbaum & Jessell, 2000). There are different classes of nociceptors that differ in mode of activation, speed of transmission and size. For example, some Aδ fibers are thermal nociceptors, myelinated, small in diameter (2-5 \(\mu M\)), and are activated by extreme temperatures (greater than 45°C and less than 5°C). Polymodal nociceptors, C fibers are unmyelinated and have the smallest diameter (0.3-3.0 \(\mu M\)). Polymodal axons like C fibers are activated by chemical, mechanical, and thermal stimuli. These primary afferent nociceptors are characterized by different rates of conduction; C axons conduct signals at velocities less than 1-2 m/s whereas A\(\delta\) fibers conduct signals at 5-30 m/s (Basbaum & Jessell, 2000; Fields, 1987). Not only do nociceptive C primary afferents constitute the majority of axons in peripheral nerves, but experimental evidence has also demonstrated that C and A\(\delta\) nociceptors are the primary...
contributors to pain sensation. They are also responsible for the quality and intensity of the sensation (Fields, 1987).

Nociceptors have peripheral terminals innervated throughout the skin. Transduction begins in the primary afferent with the activation of receptors in peripheral tissues in the form of chemical, mechanical, or thermal energy, which is then converted into electrochemical nerve impulses. The coded information is then transmitted to structures in the central nervous system. Specifically, primary afferent nociceptors terminate in the dorsal horn of the spinal cord and activate ascending tracts that relay the pain message to the brain. There are nociceptive-specific neurons that respond exclusively to noxious stimulation, and these signals ascend to the brainstem, through the thalamus, and finally to the cortex to be processed (Fields, 1987; Basbaum & Jessell, 2000).

It is important to recognize the distinction between nociception and pain perception. As Basbaum and Jessell (2000) explain, while nociceptors are activated by noxious stimuli, pain is a mental perception, “An unpleasant sensory and emotional experience…[is] a product of the brain’s elaboration of sensory input,” which is separate from the actual neural mechanics of the pathway (pp.473-474). This is an important distinction to make because pain is the basis of the dependent variable measured in the present study, which is different than nociception. Pain is an individual and subjective experience; different painful stimuli will be experienced differently between individuals. “There are no ‘painful stimuli’—stimuli that invariably elicit the perception of pain in all individuals…[For example] athletes often do not detect their injuries until their game is over,” (Basbaum & Jessell, 2000, pp.474). This issue and the distinction between pain and nociception are important for experimenters to be cognizant of when studying the experience of pain.
In order to study pain it is also crucial to understand that there are two components of the sensation of pain. First, pain intensity is considered the sensory aspect of painful sensations (Gracely, McGrath, Dubner, 1978). Secondly, there is also evidence for an affective dimension of pain which may be defined as the cognitive component or the emotional arousal of a painful experience, referred to in this study as "unpleasantness" (Gracely et al., 1978; Jensen & Karoly, 1992). Positron emission tomography studies have confirmed that there are different brain areas responsible for coding different components of pain: intensity and unpleasantness (Tolle et al., 1999).

Pain responses in the current study will be quantified using ratio scales that separately assess the sensory intensity and affective dimensions of pain. The Gracely Box Scales will be used for measures of intensity and unpleasantness (Gracely et al., 1978). This measure is a 20-point scale composed of a combination of numerical rating scales and verbal analogue scales, in other words, each numerical value is associated with a verbal descriptor (see Appendix A) (Jensen & Karoly, 1992; Gracely et al., 1978).

The perception of noxious stimuli before and after the experimental manipulation is essentially what is measured in the current study—how competition induces changes in pain sensitivity. The present study involves pain testing on three different measures. Pain responses will be obtained for the following measures: cold pressor test (ischemic pain), thermal heat threshold test, and suprathreshold thermal scaling test (pain ratings obtained for different temperatures). These are standard pain tests widely used in the field of pain research and in clinical settings (Nielsen, 1997). Some pain researchers believe that stimulus response tasks, such as the thermal scaling procedure, are better and more informative than threshold
determination because not only is the data from suprathreshold procedures more descriptive in terms of response characteristics (intensity and unpleasantness) than pain thresholds, but also thresholds are relatively consistent and thus not a good measure of changes in pain perception (Nielsen, 1997; Price & Harkins, 1992). Multiple pain measures have been included in the present study in order to measure pain and assess analgesia as thoroughly, fully, and accurately as possible.

Experimenters in the field of pain research have observed that the sensation of pain does not always coincide with nociception. This occurs because pain is not a direct result of nociception alone, but can be regulated by non-nociceptive activity. Discrepancies arise as a result of endogenous pain modulation systems that affect pain perception. Melzack and Wall proposed the Gate Control Theory of Pain in 1965 to address why the experience of pain and nociception do not always correspond. Their primary theory involves a “gate” at the level of the spinal cord that determines the upward flow of information based on communication between afferents. Excitatory and inhibitory afferents incoming from the periphery interact in the spinal cord. This local circuitry in the spinal cord is the gate-keeper that influences and modulates the transmission of nociceptive input from peripheral fibers to higher order processing structures in the brain. Melzack and Wall's theory also included the possibility that “downward” modulation could play a role in the experience of pain and introduced the idea that exogenous factors are important to the process of pain perception (Melzack & Wall, 1965; Gatchel, 1999). Support for the existence of descending internal pain modulation systems were based on evidence that stimulation of the brain, in the periaqueductal gray region (PAG), induces selective analgesia in animals and humans, a phenomenon referred to as stimulation produced analgesia. There is converging evidence that these effects are reversed by naloxone and are mediated by changes in
the descending pathways. (Reynolds, 1969; Hosobuchi, Adams, & Linchitz, 1977; Akil, Mayer & Liebeskind, 1976). The evidence shows that stimulation of the PAG blocks spinally mediated responses to noxious stimuli because the blockade involves the descending pathways that inhibit nociceptive neurons. The descending pathway is composed of neurons in the PAG, which make excitatory connections with neurons of the raphe nucleus that in turn have inhibitory connections in the dorsal horn of the spinal cord (Basbaum & Jessell, 2000).

As Melzack and Wall suspected, there are descending analgesic pathways involved in pain modulation. This system relies on endogenous opioids, which are largely responsible for and involved in the body’s intrinsic analgesia systems. Opioid peptides are analgesic agents that lead to a reduction in pain sensitivity. Opiate receptors are located at key points in the pain pathway including the central terminals of the primary afferent fibers, the spinal cord, areas of the brain, and in peripheral areas in skin, joints, and muscles (Basbaum & Jessell, 2000). Since opiate receptors are located throughout the body, the release of endogenous opioids can produce analgesia in response to many different factors. In fact, opioid mediation was found to play a crucial role in stimulation produced analgesia; the release of opioids occurs in response to electrical stimulation and results in analgesia (Hosobuchi, Adams, & Linchitz, 1977; Akil, Mayer & Liebeskind, 1976).

**Stress Induced Analgesia**

Since endogenous opioids are capable of reducing the sensation of pain, it is logical to conclude that stimuli that activate the release of these analgesic agents will lead to decreases in pain perception. Stress is an example of a naturalistic psychological factor, the experience of which is correlated with endorphin release that induces analgesia. Experiments have shown that in response to stressful stimuli, opioid peptides (such as β-endorphin) are released (Yamada &
However, the release of β-endorphin is not conclusive evidence of opioid mediated stress-induced analgesia; experimental proof of naloxone antagonism is more conclusive evidence of opioid mediation. Opiate antagonists, like naloxone, effectively block opioid activity. Subsequently, this mechanism of action is used as a helpful diagnostic tool to investigate the occurrence of opioid mediation.

Stress-induced analgesia is difficult to study in humans. In order to investigate these effects, stress must be induced in the lab in an ethical way. Some investigators rely on observations of real-life psychological stress. For example, researchers have investigated opiate mediation systems and stress-induced analgesia in novice parachute jumpers (Janssen & Arntz, 2001). Subjects were given naloxone or a placebo immediately after a jump and the results showed a decrease in pain sensitivity in the placebo condition only, accompanied by an increase in plasma β-endorphin levels in both groups (Janssen & Arntz, 2001). This evidence demonstrates that stressors can result in elevated levels of endogenous opioids and analgesia but these effects are inhibited by naloxone. The experimenters concluded that their data represent opioid mediated stress-induced analgesia in humans, and they suggest that real life stress triggers an endogenous opioid system to attenuate the potential pain of actual or imminent physical harm (Janssen & Arntz, 2001).

In addition to opioid mediated stress-induced analgesia, there is also non-opioid mediated analgesia (Yamada & Nabeshima, 1995). The stress-induced analgesia response, in some cases, can be sensitive to naloxone or not (Lewis, Cannon, & Liebeskind, 1980). Evidence indicates that the nature of the stress-induced analgesia may be dependent on the intensity, duration, and temporal pattern of the stressor (Yamada & Nabeshima, 1995). One such theory suggests that the decision to recruit the activation of opioid or non-opioid analgesia systems is determined by the
intensity and duration of the stimulus. Opioid mediated pain inhibition occurs when a stressor is short or weak, and when stressors are more intense and prolonged non-opioid mediation occurs (Terman, Shavit, Lewis, Cannon, & Liebeskind, 1984). The relationship between opioid and non-opioid systems of mediation has been classified as a mutually inhibitory interaction, in what is referred to as the collateral inhibition model (Kirshgessner, Bodnar, & Pasternak, 1982).

Stress-induced analgesia is a phenomenon that has been well documented, at least in animal studies, using a number of different stimuli such as inescapable shock, cold water swim, restraint, and centrufrugal rotation (e.g., Drugan, Ader, & Maier, 1984; LaBuda, Sora, Uhl, & Fuchs, 2000; Rizzi et al., 2001). Stress stimulates an endocrine response involving stimulation of the hypothalamic pituitary adrenocortical axis and results in the secretion of various hormones and activation of sympathetic nervous system activity (Yamada & Nabeshima, 1995; Terman et al., 1984). Cortisol is a physiological marker of stress-induced adrenal cortical activity (Janssen & Arntz, 2001). Psychological stimuli have particularly potent effects on the pituitary adrenal system, therefore, cortisol levels should reflect the amount of stress the subject is experiencing (Mason, 1971). In an experimental situation hormone assays can quantify the level of cortisol present in the sample and can be used in research as a measure to observe stress.

Previous research has demonstrated that competitive situations result in such hormonal changes. A large portion of these studies show an increase in cortisol after contests, however, in addition to competition, these situations also usually involve physical effort which may be sufficient to produce hormonal changes (Serrano, Salvador, González-Bono, Sanchis, & Suay, 2000). In the present study, hormonal responses will be assessed through the analysis of cortisol levels, which will permit us to measure the levels of stress experienced and hopefully allow
insight into differences in the physical and psychological stress of exercise with and without the impression of competition.

*Exercise Induced Analgesia*

As described above, stress is one example of a mechanism that activates endogenous pain modulatory systems. In addition, the experience of exercise is shown to result in analgesia. Research has demonstrated that exercise stimulates endogenous opioid activity in human subjects (Thorén, Floras, Hoffman, & Seals, 1990; Goldfarb & Jamurtas, 1997). Thus, it seems logical to conclude that exercise may serve as a trigger for endogenous analgesia pathways (Sternberg et al., 1998; Fuller & Robinson, 1993). This phenomenon is referred to as “Exercise-induced analgesia.” Researchers have reported evidence to support the existence of exercise-induced analgesia. For example, one study demonstrated that a 6.3-mile run resulted in analgesia in runners (Janal, Colt, Clark, & Glusman, 1984). Experimenters also determined that β-endorphin levels increased and that the exercise-induced analgesic effects could be blocked by naloxone, which indicates the involvement of opioid mediation (Janal et al., 1984).

However, research results on the effects of exercise-induced analgesia have been unpredictable. One study found hypoalgesia, decreased pain sensitivity, after intense exercise and increased β-endorphin levels, but failed to find a correlation between increased β-endorphin levels and decreases in pain perception (Oktedalen, Solberg, Haugen, & Opstad, 2001). Also in a review of the literature, Kotlyn (2000) found that results of studies vary in consistency depending on the mode of pain assessment. Studies that use painful electrical and pressure stimuli show more consistent results than studies that rely on temperature stimuli to produce pain (Koltyn, 2000). This summary of human studies also revealed that naloxone administration does
not always influence the exercise-induced analgesic response, and this finding indicates that there is a parallel non-opiate mediation system at work (Kotlyn, 2000).

Padawer and Levine (1992) proposed that the experimental evidence of the relationship between exercise-induced opioid activity and exercise-induced analgesia is an artifact of testing procedure. These researchers showed no analgesic effects for exercise and instead identified pain testing as a confounding variable. The inclusion of a control group that did not exercise eliminated differences in pain perception after exercise and showed that reduced pain sensitivity may occur on the second pain assessment of most studies regardless of exercise due to the effects of repeated testing (Padawer & Levine, 1992). However, there is debate in the literature regarding the criticisms presented by Padawer and Levine. Researchers claim that their results are in fact evidence of an exercise-induced analgesia system but it is unclear whether exercise produces analgesia in all testing situations and on all pain tests. (Fuller & Robinson, 1993; Pertovaara & Kemppainen, 1992; Droste & Greenlee, 1992). The confounds of analgesia associated with pain assessment represent an inadequacy present in most previous studies that had reported evidence of exercise-induced analgesia (Sternberg et al., 1998; Padawer & Levine, 1992).

Perhaps the evidence of exercise-induced analgesia, if it is not an artifact of repeated testing, has been misinterpreted. The reduction in pain perception after exercise observed in the literature, may actually be due to acute stress as a result of the pain testing procedure. As mentioned earlier, stress-induced analgesia is produced in the presence of an environmental stressor that is significant enough to warrant pain suppression. The pain testing may be a confounding variable in previous studies due to the stressful nature of the testing itself. Pain testing certainly qualifies as a stressor according to Yamada and Nabeshima (1995), who define
a stressor as, “An event, internal or external to an animal, that poses a real or perceived threat to the maintenance of the animal’s homeostasis,” (pp.133). Padawer and Levine (1992) indicate that the pain testing that occurs before exercise may be the source of analgesia as opposed to the act of exercising.

“The measurement of pain is not a passive procedure, however; it can be highly reactive. Given the range of environmental stimuli known to produce stress-induced analgesia (SIA) via stress mechanisms…one would expect that pain, as a stressor, would of itself produce SIA, and results cited as evidence for an EIA [exercise-induced analgesia] effect may just as well be due to the analgesic effect of the pain pretest (or repeated testing) as to the exercise per se.” (Padawer & Levine, 1992, pp.132)

However, there is an alternative explanation for the lack of conclusive evidence of exercise-induced analgesia. Inconsistent findings may be a result of inconsistent appraisal of particular exercise paradigms as stressful; exercise manipulations used in previous contrived experimental circumstances may not be uniformly stressful to all subjects. Perhaps, activation of the stress-induced analgesia pathways requires cognitive appraisal of a situation as stressful, which is a primary unanswered question that led to a need for the present study (Sternberg et al., 1998).

**Stress Induced Analgesia in Athletic Competition**

One of the most ubiquitous examples of stress-induced analgesia observed in everyday life is the pain reduction athletes experience during competition. Sternberg et al. (1998) interpreted this observation as evidence for the activation of endogenous analgesia mechanisms and began systematically studying the influence of athletic competition on analgesia in an experiment that tested athletes on various pain measures before and immediately following a competition. The results demonstrated that participating in athletic competition significantly reduced pain sensitivity to noxious stimuli (Sternberg et al., 1998). However, there were several
methodological concerns that need to be addressed in future studies. For example, there was a need to use improved testing equipment and to address previous difficulties testing athletes immediately after competition. The duration of pain modulation induced by competition is not known and earlier studies varied in the duration between the conclusion of the competition and when the pain testing was conducted. Also, the athletic competition involved exercise, and the design of previous experiments did not allow experimenters to discern between competition specific effects and exercise only effects.

Thus, in order to separately assess the pain inhibitory effects of the stressful components of athletic competition, (stress of interpersonal competition vs. physical activity) a later study also included the study of sedentary competition and non-competitive exercise. Experimenters tested male and female athletes after a track meet, but also included lab sessions where subjects were asked to compete head-to-head against another subject in a video game competition or to engage in exercise on a treadmill for a fixed period of time. Sternberg et al. (2001) reported analgesia in male and female athletes after the track meet, but the results of the lab sessions were sex-specific. Male subjects (but not females) exhibited analgesia after the video game competition, where as females (but not males) showed an analgesic response to the treadmill exercise (Sternberg et al., 2001). In response to these findings, researchers suggest that exercise produces analgesia only if it is construed as stressful (Sternberg et al., 2001). Also, it is possible that men and women may experience competition differently in that men find competition stressful and women find the exercise involved in competition stressful. The Sternberg et al. (2001) study was possibly limited by the intensity of the exercise and sex biases in the video game task, which may have been arousing or salient only to males. The researchers suggested that the threshold for exercise-induced activation of endogenous opioid systems in humans may
differ between the sexes, and if the treadmill intensity is too low, then their ability to test men's response to exercise is compromised (Sternberg et al., 2001). Regardless, the results of this study demonstrate a need for further investigation of this phenomenon and lead directly to the questions addressed by the current study. Is it the case that competition needs to be accompanied by physical exertion to trigger analgesic responses in women? Could exercise alone trigger stress-induced analgesia in men if it is strenuous enough?

The purpose of the present study is to investigate the role of competitive stress in activating endogenous pain suppressing mechanisms in response to exercise, in order to determine if the psychological state of competition or strenuous physical exertion are necessary to trigger a stress induced analgesic response. To accomplish our goal of separating stress from the exercise involved in competition, “competition” will be manipulated (absent or present) while exercise intensity is held constant. Subjects will first exercise in the lab and later will exercise in a contrived competition followed on each occasion by assessment of pain perception. Subjects will be under the impression that they are competing, however, the actual power that they exert will be controlled by the experimenter; exercise intensity will be the same when they completed the non-competitive exercise task. This manipulation will allow us to observe individual differences in pain sensitivity resulting from exercise versus competitive exercise. Thus, the effect of the competitive stress on pain sensitivity will be identified.

*Stress-Induced Analgesia: Influence of Sex and Athletic Status*

There are additional questions the present experiment intends to investigate in regards to stress-induced analgesia in competition. Through a review of the literature it is clear that sex and athletic status may influence this process.
This study will explore sex differences in the ability of competition to produce stress-induced analgesia. The evidence from previous studies in this area suggest that physical exertion must include an element of “stress” to activate pain suppressing mechanisms in males, whereas exercise alone may be considered a stressor for women capable of eliciting stress-induced analgesia (Sternberg et al., 2001). Female and male track athletes have exhibited analgesia after competition, which certainly involves both physical exertion and stress (Sternberg et al., 1998). Taken in consort, these findings suggest that the contributions of stress to athletic competition induced analgesia are different in men and women. For example, in males the stress of competition induces analgesia, but for females the exercise component of competition is stressful and is responsible for inducing analgesia, not necessarily the psychological state of competition.

There is also evidence to suggest that males and females perceive pain differently overall. Often, females demonstrate decreased thresholds, greater ability to discriminate between stimuli, higher pain ratings, and less tolerance for noxious stimuli compare to males (Berkley, 1997). The data on sex differences is considered by some to be inconclusive because they are inconsistently observed and small, however when they do appear, the differences are usually in the same direction and replicable in animals (Miaskowski, 1999; Berkley, 1997).

Regardless, some studies do report that male and female subjects show differential responses to painful stimuli (Ellermeier & Westphal, 1995; Paulson, Minoshima, Morrow, & Casey, 1998; Sheffield, Biles, Orom, Maxiner, & Sheps, 2000). Women rate thermal stimuli more unpleasant and intense than men (Sheffiled et al., 2000; Sternberg et al, 2001). One study confirms that there are sex differences in the brain areas involved in pain perception via positron emission tomography (Paulson et al., 1998). This study demonstrated that there may be a unique pattern of cerebral and cerebellar activity in response to pain, and that in males and females,
patterns of activity overlap in general (Paulson et al., 1998). However, females do rate perceived intensity of noxious heat stimuli higher and this difference is correlated with greater activation in certain structures of the forebrain, the contralateral thalamus and the anterior insula (Paulson et al., 1998).

There is also evidence that experimenter gender is a psychosocial factor that influences pain responses and is dependent on gender in that men report less pain to female experimenters (Ellermeier & Westphal, 1995; Miaskowski, 1999). All of the experimenters in the present study are female. Therefore, if female experimenters have an effect on only male subjects, then sex differences found in the current study may be exaggerated by or an artifact of experimenter gender. However, experimenter gender will not be a confound in assessing the analgesic manipulations because subjects will be tested by a female experimenter on all testing days.

An additional variable the present study will investigate is how athletic status (athlete vs. non-athlete) influences individual differences in pain and analgesic responses. Previous research has demonstrated that there are differences in pain sensitivity between athletes and non-athletes. Athletes consistently report significantly lower pain ratings than non-athletes on experimentally delivered somatic noxious stimuli (Sternberg et al., 2001; Hall & Davies, 1991; Janal, Glusman, Kuhl, & Clark, 1994). For example, the noxious cold threshold for male runners is significantly higher than control males who do not perform regular exercise (Janal et al., 1994). In this study male runners also discriminated among noxious thermal stimuli better than controls (Janal et al., 1994). Researchers concluded that athletes' capacity to tolerate pain better, at least decreased sensitivity to noxious cold, is attributed to adaptation to athletic training (Janal et al., 1994; Freischlag, 1981). However, the generalizability of the Janal et al. (1994) study across sex is limited because only male athletes were studied.
Hall and Davies (1991) observed differences in pain perception related to athletic status and found interactions between athletic status and gender on responses for different pain dimensions. The results showed that non-athletes reported higher affective pain ratings than athletes (Hall & Davies, 1991). Also, female non-athletes rated pain intensity higher than female athletes, male athletes, and male non-athletes (Hall & Davies, 1991). Athletes in general reported higher pain intensity than affect, but the reverse was true for non-athletes whose affective reports were significantly higher than intensity reports (Hall & Davies, 1991).

Therefore, pain tolerance may in part depend upon the type and the frequency of painful experiences the individual is exposed to. Athletes may experience more pain than non-athletic individuals on a regular basis, due to strenuous training and painful injuries sustained in competition, subsequently influencing their pain sensitivity (Hall & Davies, 1991; Janal et al., 1994). An alternative explanation is that an altered sensitivity to pain is required to become an athlete or that such individuals are drawn to sports.

These ideas are particularly relevant to the current study because athletic status may mediate differences in pain sensitivity, but also exercise may be more stressful for non-athlete since athletes participate in strenuous exercise during training.

Hypotheses

The major theoretical hypothesis of the present study is that exercise must be stressful in order to activate analgesic mechanisms. Therefore, if competitive exercise is considered stressful it should produce a greater analgesic response than exercise alone. If there are sex differences in this process, females may find the non-competitive exercise task stressful and demonstrate decreased pain sensitivity and no increase in analgesia in response to competition. Males may find the competition stressful showing more analgesia after competitive exercise than just
exercise. Alternatively, the exercise task may be sufficiently stressful to produce analgesia in both sexes.

Stress in the current study is observed and measured as individual differences in perception in pain, effort exerted, and sympathetic arousal. As evidence of the effectiveness of the experimental manipulation an increase in cortisol should be observed under stressful conditions, primarily during competitive exercise and to a lesser extent during exercise only.

Overall, it is expected that pain ratings reported by females will be higher than those given by males. Also in accord with previous research, athlete’s pain ratings should be lower than ratings reported by non-athletes (e.g. Hall & Davies, 1991; Janal et al., 1994).

The purpose of the control group is to determine the effects of repeated testing on pain ratings. There is no change expected in the non-exercising control group across testing sessions. Experimental and control groups should not significantly differ in pain sensitivity during the initial session.

Method

Participants

A total of fifty-two subjects from Haverford College participated in the study. Forty subjects participated in the exercise manipulation and twenty participants, who did not engage in exercise, serve as repeated testing control subjects. The subject population was composed of both athletes and non-athletes, including an equal number of male and females in each category. Athletes were recruited from the Haverford track team and non-athletes were recruited from the general campus population via signs posted around campus and in athletic buildings. There were exclusion criteria for participating in the study because of the high intensity exercise involved; subjects for whom exercise is contraindicated were not able to participate. Eligibility was
determined by a series of questions given to potential subjects during initial contact with the experimenter and repeated again on the first day subjects report to the lab. Eligible subjects were monetarily compensated at the conclusion of their participation. Exercising subjects received fifty dollars and repeated testing control subjects were given twenty-five dollars.

**Forms and Questionnaires**

In order to verify eligibility for participation, subjects completed the Medical History Questionnaire (see Appendix B). Subjective ratings of physiological arousal were assessed by the Body Awareness Questionnaire that asks subjects to rate, on a 4 point scale, the degree to which they were currently experiencing symptoms of sympathetic arousal (see Appendix C). Also, subjects were asked to rate the perceived effort (RPE) exerted during the exercise tasks on the Borg's RPE scale (see Appendix D), which measures the amount of personal investment in the task.

**Physiological Measures**

Subjects had their blood pressure and heart rate measured using the Omron Blood Pressure and Heart Rate Meter, an inflatable pressure cuff that is placed on the wrist. Experimenters also obtained the subject's skin temperature.

**Hormone Assay**

Salivary samples were collected from each subject for analysis of the levels of the stress hormone cortisol.

**Pain Testing Equipment**

A Medoc thermal stimulator was used to obtain thermal heat threshold and suprathreshold thermal ratings. This device is widely used in research and clinical settings and is approved by the FDA. The device can produce temperatures from 0°C to 50°C, which are
delivered by a 30 x 30 mm Peltier thermode (thermal probe) that is placed on the volar surface of
the forearm. Temperatures within this range do not cause tissue damage.

Pain Threshold

During the thermal heat threshold test, the thermal probe were placed on the dominant arm and began at a neutral temperature (32°C). The temperature of the probe rose 1°C every second until the subject reported that the stimulus was painful. This indicates that the pain threshold has been reached and the device returns to neutral. This procedure was then repeated three times and the average temperature of the four trials was recorded as the pain threshold.

Thermal Scaling Test

During the thermal scaling procedure, the subject was presented four stimulus temperatures (42°C; 44°C; 46°C; 48°C) twice, for a total of 8 stimuli at 8 different sites of the volar forearm. The order of the stimulus sequence was randomized. The stimuli were delivered in 10-second intervals. The thermal probe will began at neutral and rose (4°C/sec), and peaked at the target temperature and remained at that temperature for a duration of 5 seconds. At the end of the five seconds the subject was asked to make pain ratings on the Gracely box scales of intensity and unpleasantness. These scales consist of numerical ratings (0-20) accompanied by descriptor terms (see Appendix A). The first response given on these scales was used in the analysis as the dependent measure.

Cold Pressor Test

The cold pressor test required subjects to submerge their non-dominant arm (up to the elbow) in the center of a bucket of ice-cold water for a maximum of ninety seconds. The ice was not in contact with the skin; it was separated from the center by a mesh screen. The subjects are asked for numerical ratings (on the Gracely box scales) of pain intensity and unpleasantness
every fifteen seconds for ninety seconds or until the subject removed their arm (after which the maximum rating was given for the remaining time).

**Pain testing Demonstration**

Subjects were shown the highest temperature produced by the thermal probe that was used in the study, and received a demonstration of the cold pressor test and then given an opportunity to withdraw from the study. Subjects practiced making perceptual judgments about thermal stimuli using a “warm threshold” activity, during which subjects make a decision about when the thermal probe first feels warm. The probe started at 32°C and increased in temperature until the subject reported a warm sensation.

**Exercise Equipment**

The exercise component of the study was conducted on a cycle ergometer (Lode Corival) controlled by Turbofit software. This program allows the experimenters to control the workload (in Watts); the pedal resistance varies inversely to the pace at which the subject pedals (rpm or revolutions/minute). The device in this study allowed power output to be fixed without the direct knowledge of the participant.

If at any time during an exercise session a subject felt faint, dizzy, experienced chest pain, or had trouble breathing, they were instructed to discontinue exercise. Water and clean towels were available to the subjects.

**Maximal Capacity Test**

Exercising subjects completed a maximal exercise capacity test. In this case, subjects began with a two minute warm up period where they cycled at their own pace at 10 W. Subjects were then asked to pedal at a fixed rate (50 rpm) while the power increased (2W/sec) until they
were unable to maintain the pace of 50 rpm. The power at which the test ends is considered the maximal capacity.

Noncompetitive Exercise Task

During the exercise session, subjects cycled at a fixed workload that is 60% of the subject's maximal capacity.

Competitive Exercise Task

Subjects again cycle at a fixed workload that is 60% of the subject's maximal capacity. However, the subjects were told that they are competing against another subject with regards to distance traveled. During the competitive task, when a subject cycled another subject was present, who is waiting for their turn to cycle. Subjects were told that the cyclist who traveled the farthest on the ergometer between the two subjects present or out of all the subjects would receive five dollars and twenty-five dollars respectively. Competing subjects were matched for sex and athletic status. Thus, whether or not the subject believed that they are in a competition served as the experimental manipulation. All subjects were entered in a lottery for an opportunity to win a thirty-dollar prize, the same amount as the incentive previously offered in the competition condition. The only deception then was the impression of competition since each subject had an equal chance to win the prize.

Design

The present study looked at the change in pain ratings on standard pain measurement stimuli. The current study is 2 x 2 x 2 x 3 factorial design. Sex (male or female) and athletic status (athlete or non-athlete) are between-subject variables that are investigated. There is also a between-subject “Group” variable, comparing the experimental groups, who receive the exercise manipulation, to a control group that repeats the pain testing three times, which will allow
experimenters to determine if there are any repeated testing effects. The within subject "Day" variable refers to the competition, exercise only and baseline sessions. The experimental subject’s baseline ratings are compared to ratings after the two exercise sessions that either do or do not involve competition.

Procedure

Subjects reported to the lab for three separate sessions. Subjects were briefed about the procedures involved in participating and signed a consent form (see Appendix E). All subjects received a demonstration of what is involved in pain testing during the first visit to the lab and was be given an opportunity to withdraw from the study before further testing.

Subjects who were not exercising had their pain tested on three separate occasions. Exercising subjects were scheduled to participate at a time of day before any regularly scheduled exercise or athletic practice. On these occasions the following occurred:

Day 1: Obtained Medical and Fitness History; Subjective and physiological measures of arousal; Pain testing demonstration; Baseline pain testing followed by the maximal exercise test.

Day 2: Cycling task for 20 minutes (non-competitive), workload was set to 60% of the subject's maximal capacity. Exercise is followed immediately by subjective and physiological measures of arousal, rating of perceived effort, and pain testing.

Day 3: Competitive cycling task for 20 minutes. The workload was the same as the previous day, but the subject that the cyclist was told they were competing against was be present, waiting for their turn to cycle. Subjects were tested on the subjective and physiological measures of arousal, rating of perceived effort, and on the pain tasks immediately following their cycling task.

Cold pressor ratings, thermal heat threshold, and suprathreshold thermal ratings served as the pain measures in this study. Subjects were tested on the pain measures immediately
following both the non-competitive and competitive exercises tasks. After both subjects had
completed the competitive task and pain testing, they were informed that the workload was fixed
and they were not actually in competition. The nature of the deception required by the
experimental manipulation did not allow the order of the exercise sessions to be counterbalanced
between competitive and non-competitive tasks. Hence, there was a separate group of control
subjects who share the same characteristics (sex and athletic status) as experimental subjects that
were tested on the pain measures on three separate occasions.

Data Analysis

To assess the effects of participating in strenuous exercise and interpersonal competition
a series of 2 x 2 x 3 mixed-factorial analyses of variance (ANOVA) will be conducted on pain
response data for experimental and control subjects separately. Further analyses consist of Fisher
LSD post-hoc tests. The threshold analysis will use an average threshold calculated from the four
thresholds taken on each testing day. Analysis of the first presentation of the stimulus is used in
the analysis of thermal scaling data. Cold pressor analyses use the sum of the intensity and
unpleasantness ratings. A main effect for the Day variable is expected where the experimental
manipulation should show changes across testing day (in pain testing and stress measures) but
there should be no changes in the control group. The changes predicted in the experimental
group may be mediated by interactions between gender and athletic status.

Results

In order to study the effects of the experimental manipulation on analgesia, analysis of
the control subjects was first conducted to identify any effects of repeated testing as an
alternative explanation for reduction in pain sensitivity in the experimental group. Control
subjects were recruited separately, compensated differently, and were considered participants in
a separate study conducted in parallel with the exercise study, and therefore data from the control subjects were analyzed separately.

**Analysis of Pain Data for Control Subjects**

Repeated measure multivariable Analysis of Variance (MANOVA) were calculated first using only control subjects, to test the effect of repeated testing on pain sensitivity. No significant effect of testing day on heat pain thresholds was observed \((F(2, 32)=1.57, p>.05)\). There was also no effect of testing day on thermal scaling pain ratings \((F(2, 32)=2.61, p>.05)\) or cold pressor ratings \((F(2, 32)=0.21, p>.05)\). Therefore, repeated testing does not appear to alter pain sensitivity.

The only repeated testing effect observed demonstrated an interaction between day and athletic status for pain threshold \((F(2, 32)=4.34, p<.05)\). Pain thresholds were elevated on Day 3 \((M=46.53, SD=.65)\) compared to Day 1 \((M=45.35, SD=.60)\) in control athletes. However, Day 2 \((M=45.99, SD=.77)\) thresholds were not significantly different from Day 1 or Day 3. This result indicates that although athlete's sensitivity to painful stimuli decreases with the frequency with which they are exposed to it, this effect does not account for differences in analgesia between noncompetitive and competitive exercise.

Significant main effects of temperature \((F(3, 48)=107.59, p=.00)\) and scale \((F(1, 16)=36.53, p=.00)\) on thermal scaling analyses were found. Pain ratings increased as temperature increased from \(42^\circ C \ (M=6.67, SD=0.72)\), to \(44^\circ C \ (M=9.67, SD=0.69)\), to \(46^\circ C \ (M=12.46, SD=0.71)\), and to \(48^\circ C \ (M=14.75, SD=0.65)\). Pain intensity ratings \((M=12.2, SD=0.60)\) were higher than unpleasantness \((M=9.53, SD=0.73)\) ratings in controls. These same effects and patterns are observed in the experimental subjects.

**The Effects of Sex and Athletic Status in Control Subjects**
Analysis of the pain ratings, physiological, and subjective arousal data of control subjects did demonstrate some interesting effects related to sex and athletic status. Repeated measure multivariable Analysis of Variance (MANOVA) were conducted for each measure: blood pressure, pulse, cortisol levels, skin temperature, body awareness and the three pain measures.

Control Pain Measurements

Although there were no differences between the sexes or athletes and non-athletes in heat pain thresholds or thermal scaling, an interaction between sex and athletic status on cold pressor ratings was observed \( (F(1, 16)=8.06, p<.05) \). Female athletes pain ratings \( (M=71.7, SD=7.30) \) were significantly lower than female non-athletes \( (M=103.17, SD=7.30) \) but male athletes \( (M=93.46, SD=7.30) \) did not differ from male non-athletes \( (M=83.46, SD=7.30) \) (see Figure 1).

Control Physiological and Subjective Data

Blood Pressure

The physiological data demonstrated significant effects for sex and athletic status in systolic and diastolic blood pressure (see Figure 2). No significant effect of testing day was observed in either systolic or diastolic blood pressure. However, there was a main effect of sex in systolic blood pressure \( (F(1, 16)=8.38, p<.05) \) and an interaction between sex and athletic status \( (F(1, 16)=10.17, p<.05) \). Systolic blood pressured did not differ between male athletes \( (M=127.73, SD=3.64) \) and male non-athletes \( (M=137.47, SD=3.64) \) but a difference did exist between female athletes \( (M=128.80, SD=3.64) \) and non-athletes \( (M=115.33, SD=3.64) \). Female non-athletes had significantly lower systolic blood pressure than all other groups. An interaction for sex and athletic status was also observed in diastolic blood pressure \( (F(1, 16)=14.23, p<.05) \). Male athletes \( (M=77.60, SD=2.89) \) were lower than male non-athletes \( (M=89.73, SD=2.89) \).
Female non-athletes ($M=74.07, SD=2.89$) diastolic blood pressure was significantly lower than female athletes ($M=83.73, SD=2.89$) and male non-athletes.

**Heart Rate**

A significant main effect of athletic status on heart rate ($F(1, 16)=14.23, p<.05$) indicated that non-athletes have a higher resting heart rate ($M=78.43$) than athletes ($M=66.57$) (see Figure 3). There was no significant effect of day on pulse ($F(2, 32)=0.40, p>.05$) (see Figure 4).

**Skin Temperature**

There was no effect of day on skin temperature ($F(2, 32)=3.24, p>.05$). However, there was a significant main effect for athletic status ($F(1, 16)=6.02, p<.05$), athlete's skin temperature was significantly lower than non-athletes, ($M=31.28, SD=0.21$) and ($M=32.02, SD=0.21$) respectively.

**Cortisol Data**

There were no significant effects of testing day on cortisol levels ($F(2, 32)=0.44, p>.05$) nor were any main effects or interactions noted for the sex and athletic status variables.

**Body Awareness**

A significant main effect of testing day was observed on the body awareness questionnaire (a measure of subjective arousal) ($F(2, 32)=3.96, p<.05$), which decreased across day. Self-reported ratings of body awareness were significantly lower on Day 3 ($M=24.60, SD=0.65$) compared to Day 1 ($M=26.95, SD=0.75$), however ratings on Day 2 ($M=25.80, SD=0.57$) were intermediate and did not significantly differ from the other days (see Figure 5).

**Analysis of Pain Data for Experimental Subjects**
In order to study the effects of strenuous exercise and competitive stress on pain, repeated
measure mixed factorial Analysis of Variances (MANOVA) were conducted on the three pain
measurements, heat pain threshold, thermal scaling task, and cold pressor.

Pain Threshold
A significant main effect of testing day on average heat pain threshold was observed
\( F(2, 56)=5.16, p<.05 \). Thresholds decreased significantly across day, baseline \( (M=44.92,\ SD=0.48) \), exercise \( (M=45.14, SD=0.40) \), and competition \( (M=45.72, SD=0.42) \). Pain thresholds after competition were significantly higher than those displayed at the baseline session and following an equally intense bout of exercise (see Figure 6). Correlation analysis between skin
temperature and threshold was calculated to determine whether skin temperature and thresholds
are correlated overall. On Days 1 and 2 skin temperature is not correlated with threshold,
however, Day 3 skin temperature was negatively correlated to Day 3 threshold \( r = -.37, p<.05 \).

Thermal Scaling Task
The main effect for testing day on pain ratings averaged across stimulus temperatures
was marginally significant \( F(2, 56)=2.87, p=.06 \), the trend is in the expected direction
decreasing from baseline, exercise, to competition, \( (M=10.34, SD=0.58) \), \( (M=9.88, SD=0.62) \),
and \( (M=9.16, SD=0.57) \) respectively. Significant main effects for temperature \( F(3, 84)=148.05,\ p=.00 \)
and scale \( F(1, 28)=64.87, p=.00 \) were observed. These effects were similar to those
observed in control subjects. Pain ratings increased as temperature increased from 42º C
\( (M=6.22, SD=0.52) \), to 44º C \( (M=8.47, SD=0.50) \), to 46º C \( (M=10.68, SD=0.55) \), and to 48º C
\( (M=14.13, SD=0.56) \). Pain intensity ratings \( (M=10.82, SD=0.51) \) were higher than
unpleasantness \( (M=8.93, SD=0.53) \).
The effect of testing day was dependent on temperature, evidenced by a significant interaction between day and temperature ($F(6, 168)=2.70, p<.05$). At 42º C the differences in pain ratings significantly decreased across baseline, exercise, and competition, ($M=7.50, SD=0.69$), ($M=6.34, SD=0.61$), and ($M=4.81, SD=0.67$) respectively (see Figure 7). At intermediate and higher temperatures, the trend is in the expected direction but the changes are not significant (see Table 1 and Figure 8). These results indicate a decrease in pain sensitivity following exercise and even larger decrease in pain sensitivity after a competition.

There was an effect of athletic status on thermal scaling pain ratings that approached significance ($F(1,28)=3.38, p=.07$). Pain ratings of athletes ($M=8.94, SD=0.71$) were lower compared than non-athletes ($M=10.80, SD=0.71$).

A significant interaction of testing day, temperature, and scale was found ($F(6,168)=2.41, p<.05$). The components of the thermal pain ratings, intensity and unpleasantness, were analyzed separately. At 42°C and 48°C unpleasantness ratings were significantly higher on baseline day compared to exercise day and competition day, and competition ratings were lower than exercise ratings. At 44°C and 46°C pain ratings did decrease across day, baseline ratings were significantly higher than competition but exercises ratings are intermediate and do not significantly differ from competition. Similar patterns were also found in the intensity ratings. At 42°C baseline intensity ratings are significantly higher than ratings following exercise and competition, and the increase in analgesic response between exercise and competition days is significant. (see Table 2 and Figure 11).

The same main effect of temperature was found for intensity ($F(3,81)=137.01, p=.00$) and unpleasantness ratings ($F(3,84)=133.70, p=.00$) where as temperature increases pain ratings increase.
Competition/Exercise and Pain 31

Cold pressor

Contrary to expectations, there was no significant effect of testing day on cold pressor ratings ($F(2,56)=1.63, p>.05, \text{n.s.}$). This finding does not support the hypotheses of the present study. However, the ratings did decrease across day in the expected direction baseline ($M=84.44, SD=4.07$) > exercise ($M=82.00, SD=4.83$) > competition ($M=79.73, SD=4.85$) but the differences did not reach statistical significance (see Figure 9). There was a significant effect of scale as seen in the other pain measurements; the intensity ($M=84.34, SD=4.30$) ratings were higher than unpleasantness ratings ($M=79.77, SD=4.46$).

Physiological and Subjective Data for Experimental Subjects

Blood Pressure

There was no significant main effect of day on systolic blood pressure across day ($F(2,56)=2.67, p=.07$). A main effect for sex approached significance ($F(1,28)=3.67, p=.06$), this sex difference mirrored the effect observed in controls on systolic blood pressure, males ($M=135.91, SD=3.63$) were higher than females ($M=126.08, SD=3.63$). There were no significant effects of testing day, sex, or athletic status on diastolic blood pressure.

Heart Rate

As expected, there was a significant main effect of testing day observed on heart rate ($F(2,56)=3.56, p<.001$). Heart rate increased significantly across day after exercise ($M=111.53, SD=3.59$) and competition ($M=126.25, SD=4.10$) compared to baseline ($M=76.19, SD=1.92$) (see Figure 4).

There was a significant main effect for athletic status ($F(1,28)=5.98, p<.05$) and a significant interaction between sex and athletic status ($F(1,28)=7.94, p<.05$). Like in control subjects, heart rates in athletes ($M=98.50, SD=3.55$) were significantly lower than in non-athletes.
(M=110.81, SD=3.55). Male non-athletes (M=122.91, SD=5.03) displayed significantly higher heart rates than male athletes (M=96.41, SD=5.03) and female athletes (M=100.5, SD=5.03) or female non-athletes (M=98.70, SD=5.03) (see Figure 3).

Skin Temperature

A significant effect of testing day on skin temperature was found (F(2,54)=4.37, p<.05) Skin temperature on Day 3 (M=31.07, SD=0.20) was significantly lower than skin temperature on Day 1 (M=31.71, SD=0.14). However, there is no significant change in skin temperature between Day 2 and Day 3.

Body Awareness

There was a significant main effect for testing day (F(2,56)=12.23, p<.05) on self-reported sympathetic arousal, which increased across day (M=27.19, SD=0.71), (M=30.56, SD=0.93), (M=31.94, SD=0.99). There was a significant interaction between day, sex, and athletic status (F(2,56)=5.13, p<.05). Subjective arousal does not significantly differ between baseline, exercise and competition (M=29.38, SD=1.43), (M=30.75, SD=1.86), (M=30.62 SD=1.97) in male non-athletes, but male athletes report elevated sympathetic arousal after competition (M=33.25, SD=1.98) as compared to baseline (M=25.38, SD=1.43) and exercise (M=28.62, SD=1.86). Female non-athletes are aroused equally on exercise (M=33.8, SD=1.86) and competition day (M=32.75, SD=1.97), which differ significantly from baseline (M=25.12, SD=1.43). Female athletes however do not significantly differ in their self-reported arousal on exercise (M=29.50, SD=1.86) and competition (M=31.12, SD=1.98) compared to baseline (M=28.88, SD=1.43) (see Figure 5).

Ratings of Perceived Effort
There was a significant main effect for testing day ($F(1,28)=28.65, p<.05$) and a significant day by athletic status interaction ($F(1,28)=15.26, p<.05$) on ratings of perceived effort (RPE) expended on the cycling task. As expected RPE significantly increased on the competition day ($M=9.28, SD=0.48$) compared to the exercise day ($M=6.97, SD=0.36$). A significant interaction between testing day and athletic status was found ($F(1,28)=15.26, p<.05$).

After intense exercise, athletes reported exerting significantly less effort ($M=5.44, SD=0.52$) than non-athletes ($M=8.50, SD=0.52$). However after equally intense exercise involving competition, athletes reported significantly higher effort ($M=9.44, SD=0.69$) exerted than they had after the same task on the previous day ($M=5.44, SD=0.52$), but non-athletes RPE rating did not change significantly between Day 2 ($M=8.50, SD=0.52$) and Day 3 ($M=9.12, SD=0.69$) (see Figure10).

Cortisol

There were no significant effects of testing day on cortisol levels ($F(2,56)=0.65, p>.05$)

Discussion

The primary interest of this study was to examine interpersonal competition as a stressful stimulus, and to determine which component of athletic competition, psychological stress or physical exertion, is a trigger for endogenous pain inhibitory systems and to identify differences in this phenomenon based on sex and athletic status. As expected, competitive exercise is a stressor capable of activating stress-induced analgesia, and appears to be considered more stressful than strenuous exercise alone, at least in some subjects. Experimental subjects show a larger reduction in pain sensitivity on competition day than when completing the non-competitive exercise, but not all subjects rated this day as "more stressful". Differences in the experience of strenuous physical exertion and competitive stress are related to sex and athletic status.
The thermal scaling data are consistent with the hypothesis that competition and intense exercise are stressors that activate endogenous pain inhibitory mechanisms. Pain ratings decrease from baseline in response to exercise and competition. Experimental subjects rated thermal noxious stimuli at 42°C less intense and less unpleasant after competition and exercise as compared to baseline (see Figure 7 and Figure 8). The increase in analgesia between exercise day and competition day indicates that although intense exercise is a stressor capable of initiating a stress-induced analgesia response, in athletic competition the added element of competitive stress enhances the effects of stress-induced analgesia.

The existence of competitive stress-induced analgesia is further confirmed by the pain threshold data. Heat pain thresholds were significantly higher, suggesting less pain sensitivity, following competition as compared to baseline and equally intense exercise (see Figure 6). This result indicates that exercise is not sufficient to raise thresholds but that a competitive element is necessary to induce analgesia, which supports the hypotheses of the present study. Sternberg et al. (1998) reported a similar finding, track athletes and basketball players exhibited forearm analgesia due to athletic competition. These findings confirm that competition is uniquely capable, compared to exercise, of activating stress-induced analgesia in some subjects.

The hypotheses of the current study were based on previous findings in the literature that exercise must be evaluated as stressful in order to activate analgesic mechanisms. There is physiological evidence that all experimental subjects were aroused after exercise and competition tasks, such as the significant increase in heart rate observed. Self-reports of sympathetic arousal, assessed by the Body Awareness Questionnaire, reveal the differences in the experience of exercise and competition by different groups. Female non-athletes exhibited increased arousal that did not differ between exercise and competition days. Male athletes do not
report elevated arousal after exercise compared to baseline even though the physiological data show that pulse increases, however, after competition they do show elevated arousal compared to baseline and the same power exercise. Male non-athletes and female athletes show no difference in arousal by testing day, even though again the physiological data show significant increases in pulse across day (see Figure 5). These results partially support the hypothesis based on previous findings that men, particularly athletes, may find competition stressful and women, especially non-athletes, may find the exercise involved in competition stressful.

In this case differences in arousal between female non-athletes and female athletes may be due to the physical exertion required by these tasks which could be particularly strenuous in female non-athletes as compared to female track athletes who regularly participate in intense exercise. Male athletes may show increased arousal in response to competition as compared to male non-athletes because competitive stress is particularly salient in male track athletes who participate in competition regularly and are also used to competing against each other as they did in the present study.

Furthermore, the results of the body awareness questionnaire are complemented by the athletic status differences observed in ratings of perceived effort. Self-reported ratings of the intensity and effort required of each task increased between exercise and competition day in athletes only. Athlete's ratings of perceived effort were lower than non-athletes after exercise but then made a dramatic increase following competition, which did not differ from non-athletes (see Figure 10). Thus, according to the ratings perceived effort data, non-athletes found competition stressful but no more than exercise and athletes found competition profoundly more stressful than exercise.
If athletes find the same power exercise more intense, effortful, and stressful when they are under the impression that they are competing, then the hypothesis that competitive stress has a greater psychological effect on athletes than non-athletes, in comparison to strenuous exercise in the absence of competition is confirmed.

In summary, the results of the current study suggest that strenuous exercise is capable of producing stress-induced analgesia in males, females, athletes and non-athletes, and this reduction in pain ratings increases under competitive stress. The present study also confirms that sex and athletic status mediate the subjective evaluation of exercise and competitive exercise as stressful.

*Pain Measurement Analyses and Limitations of the Current Study*

While the results of the current study support the hypotheses presented, there are elements of the study that conflict with the original hypotheses. For example, the thermal scaling data failed to find significant decreases in pain ratings from baseline in response to exercise and competition at 44°C, 46°C, and 48°C. However, a significant reduction in pain sensitivity was found at the lowest temperature (42°C). One possible explanation is that higher temperature noxious stimuli were too painful to observe sizeable analgesic effects. These temperatures are all near or above the baseline threshold average, which is plausibly why endogenous pain inhibition is not significant enough to overpower the intensity and unpleasantness of these sensations.

The analysis of the day, temperature, and scale interaction on thermal scaling ratings reveal that when intensity and unpleasantness are separated, there are significant effects of testing day on both components. Following exercise and competition, experimental subjects rated the thermal noxious stimuli as significantly less unpleasant at most of the stimulus temperatures. The testing day and temperature effect of intensity ratings showed a similar pattern
as the unpleasantness ratings but only at the lowest temperature. This result further supports the hypothesis that competitive stress in combination with physical exertion has a larger stress-induced analgesia effect compared to strenuous exercise in the absence of competition. Again, failure to reach significance at all temperatures may be related to pain thresholds where intermediate and high temperatures are above the average pain threshold and consequently are too painful to demonstrate efficacious stress-induced analgesia. However, this argument is more difficult to make when looking at the components of thermal scaling ratings separately because the unpleasantness ratings significantly decrease across baseline, exercise, and competition days at the highest and lowest temperatures but do not significant decline at intermediate temperatures.

The cold-pressor data are to some extent consistent with the hypothesis that competitive stress can activate endogenous pain suppressing mechanisms. The expected trend was observed, cold pressor pain ratings decreased following exercise and decreased further following competition. Unfortunately, these differences in pain ratings across testing day did not reach statistical significance. Some studies have shown that thermal and ischemic pain were reduced following exercise in all subjects on various measures, but there was no analgesic response observed on the cold pressor test (Janal et al., 1984). However, there are studies that have reported significant reductions in cold pressor pain in athletes after athletic competition (Sternberg et al., 1998). The replicability of analgesia with repeated cold pressor tests has been criticized because the results are mixed (Kotlyn et al., 2000). In an effort to address this discrepancy, the present study involved multiple pain measures. The threshold test and thermal scaling task were successful in identifying stress-induced analgesia as a result of competition;
however, if the power of the study were increased the analgesic effects found on the other measures may have been significantly detected by the cold pressor test.

*The Effects of Sex and Athletic Status*

A secondary interest of the present study was to examine how pain sensitivity and physiological and self-report stress responses vary with differences in sex and athletic status. Contrary to the hypotheses presented, there were no significant differences in pain thresholds or thermal scaling pain ratings mediated by sex and athletic status. However, there was a marginally significant effect of athletic status on thermal scaling pain ratings. Athletes' pain ratings were lower than non-athletes. This finding indicates that athletes may have reduced pain sensitivity. However, this effect did not reach statistical significance and the present study fails to identify the robust athletic differences in pain sensitivity that have been reported in previous research (Sternberg et al., 2001; Hall & Davies, 1991; Janal, Glusman, Kuhl, & Clark, 1994).

One interesting difference between athletes and non-athletes found in control subjects is related to pain threshold. Athletes' pain thresholds increased between Day 1 and Day 3 by approximately one second. Apparently, athletes seem to think that painful stimuli are less painful when they are frequently exposed to it. The implications of this result in the outcome of the current study need to be addressed. Experimental subjects did exhibit elevated thresholds on Day 3, however, there was no interaction of athletic status observed. The repeated testing effect in control athletes observed on Days 1 and 3 may partially contribute to the analgesic effect in athletes found in the experimental group. However, repeated testing does not appear to influence changes between Days 2 and 3 in the control group, therefore are not considered responsible for significant differences in pain thresholds between Day 2 and 3 in the experimental group.

There were no significant differences between the sexes or athletes and non-athletes in
heat pain thresholds or thermal scaling ratings, but a significant interaction between sex and athletic status on cold pressor ratings was observed (see Figure 1). Female non-athletes in the control condition rated the cold pressor pain as more intense and unpleasant than female athletes, who did not differ significantly from male athletes or non-athletes. Therefore, differences in pain ratings in females arise from differences in athletic status. These findings, as well as the athletic differences in thresholds after repeated testing, are consistent with the expected hypothesis that athletes may experience more pain than non-athletic individuals on a regular basis, due to strenuous training, consequently influencing their pain sensitivity (i.e. Hall & Davies, 1991). In a study by Hall and Davies (1991) a similar result with the cold pressor test showed that female athletes displayed lower perceived intensity and unpleasantness than female non-athletes, but female athletes did not differ from male athletes or non-athletes. Therefore, the present study confirms previously established athletic differences, which influence the experience of cold pressor pain, at least in females.

The present study replicates a sex difference in systolic blood pressure that has been established in previous studies (Kotlyn et al., 2001). In control and experimental subject, males had higher systolic blood pressures than females, and an interaction with athletic status showed female non-athletes had the lowest systolic blood pressure (see Figure 2). Blood pressure in the experimental group was not influenced by testing day, which is expected because blood pressure is typically invariant and independent from the cardiovascular effects of exercise, like an increase in pulse. Additionally, athletes in the control group had a lower resting heart rate than non-athletes (see Figure 3). This result could have been anticipated given that athletes may be expected to be in better cardiovascular condition than non-athletes.

An additional difference between athletes and non-athletes was observed in skin
temperature. Athletes in the control group were on average one degree cooler than non-athletes, which is important because skin temperature may influence the perception of thermal noxious stimuli (discussed below). Therefore, it is important to consider the implication of athletic differences in skin temperature in the current study. Further analysis established that athletic differences in skin temperature were not found in the experimental condition and thus would not influence pain threshold assessment.

The body awareness questionnaires were intended to assess sympathetic arousal. A significant effect of testing day was found in both conditions. Control subjects body awareness levels were lower on the third day of testing compared to the first day (see Figure 5). This result indicates that perhaps subjects were more comfortable on the third day because they were familiar with the procedures. Repeated testing seems to decrease body awareness, which is important because the opposite effect is expected in the experimental group.

Indeed, in experimental subjects body awareness increased in male athletes in response to competition, and in female non-athletes body awareness increased and remained elevated during exercise and competition (see Figure 5). These results support the hypotheses presented in the current study that sex differences in stress-induced analgesia may arise from what the sexes evaluate as stressful and subsequently self-report more arousal, in particular, competition in men more so than women.

Given that men and women experience differences in the psychological state associated with athletic competition we would anticipate that these body awareness and rating of perceived effort effects contribute to sex differences in analgesia. The original hypothesis would predict that women would exhibit analgesia following strenuous exercise and no increase in analgesia following competition, where as men were expected to show greater analgesia or only analgesia
during athletic competition as opposed to demonstrating an analgesic response during strenuous exercise in the absence of competition. Contrary to this expectation there were no differences in analgesia between the sexes—men and women both exhibited analgesia following exercise and increased analgesia after competition. Therefore, perhaps the differences observed in psychological state were not profound enough to translate into sex differences in analgesia during athletic competition.

Alternative Explanations and Limitations of the Current Study

As mentioned earlier, the heat pain threshold data analysis demonstrates that exercise is not sufficient to raise threshold but that a competitive element is necessary to induce analgesia. This evidence is critical in substantiating the theories and hypotheses of the present study. However, it could be argued that increases in pain thresholds are not a product of stress-induced analgesia but a function of skin temperature. There was a negative correlation found between skin temperature and threshold on Day 3. The present study does not disprove that the increase in threshold on Day 3 is an artifact of skin temperature. However there is no difference in skin temperature between Day 2 and 3, whereas an increase in pain threshold was found on Day 3 compared to Day 2. Consequently, differences in skin temperature cannot be responsible for the increase in analgesia observed in thresholds after exercise versus competition (see Figure 6). It is possible that there is no direct relationship between skin temperature and threshold, but instead changes in these items are caused by a common underlying mechanism of stress-induced analgesia following competition. Previous studies have shown that heat pain thresholds were only minimally influenced by changes in skin and body temperature (Kotlyn, 2000). Regardless, future research is necessary in order to clarify how changes in skin temperature that accompany exercise interact with pain threshold.
Additionally, future research should aim to identify the most informative time and way to obtain cortisol information. Cortisol assays were included in the present study based on the expectation that cortisol levels would effectively reflect the levels of stress experienced by the subjects. However, there was no significant effect of testing day on cortisol levels. This result does not mean that subjects did not experience stress because the Body Awareness Questionnaire and the Ratings of Perceived Effort documented the occurrence of stress in subjects. Perhaps saliva samples were not taken at the appropriate time, stress levels may peak in the beginning of exercise or competition and the duration of changes in hormone levels was not considered. Future research would benefit from information on or the investigation of the optimal time to obtain hormone samples under the conditions of athletic competition. There are studies that show cortisol levels demonstrate anticipatory peaks prior to competition (Filaire, Maso, Sagnol, Ferrand & Lac, 2001). Therefore, it may be more informative to assess cortisol levels just before competition in the future.

The present study included a group of subjects that had their pain tested on three occasions in order to control for the effects of repeated testing. Overall, there were no effects of testing day in the controls, which means simply undergoing pain testing three times does not result in changes in pain sensitivity.

However, there are at least two concerns raised by the control data. Athletes did demonstrate a decrease in pain ratings from Day 1 to Day 3. Consequently, we are unable to determine whether or not the decrease in pain ratings between baseline and competition in athletes is partially due to the effects of repeated testing. Also, at 42°C there is a decreasing trend in pain ratings in the control group (see Figure 8). Although the differences across Days 1, 2, & 3 are not significant in controls, the trend is concerning because it mirrors the analgesia observed
in experimental subjects and could suggest that these effects are due to repeated testing and not the experimental manipulation.

Finally, the present study does not control for the effects of repeated cycling. Due to the nature of the deception involved in the design of the study, the exercise session and the competition session could not be counterbalanced. However, the effects of repeated cycling if there were effects would be in the opposite direction of the expected stress-induced analgesia effects of competition. We could anticipate that participating in a second cycling session would be less stressful because the subjects would be familiar with the protocol. Therefore, repeated cycling is less likely to be a confounding variable of the observed stress-induced analgesia effects.

In conclusion, the present study provides evidence that the analgesic effects of the activation of the stress-induced analgesia pathways as a result of exercise increase in response to the competitive stress involved in athletic competition. The results also indicate that sex and athletic status influence the subjective experience of competitive stress and stress induced analgesia.
References


receptor knockout mice reveals normal function of the delta opioid receptor system.

*Brain Research, 869,* 1-5.


Author Note

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Miriam Meister and Jillian Scavone, Psychology Students, Department of Psychology, Haverford College.

I would like to acknowledge my collaborators on this project for their assistance in designing the experiment and special thanks to Professor Sternberg, our thesis advisor, for overseeing the fabrication of this experiment.
Table 1

Pain Ratings on Thermal Scaling Task

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Baseline</th>
<th>Exercise</th>
<th>Competition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
</tr>
<tr>
<td>42°C</td>
<td>7.50*</td>
<td>0.69</td>
<td>6.34 *</td>
</tr>
<tr>
<td>44°C</td>
<td>8.20</td>
<td>0.72</td>
<td>8.78</td>
</tr>
<tr>
<td>46°C</td>
<td>11.13</td>
<td>0.71</td>
<td>10.67</td>
</tr>
<tr>
<td>48°C</td>
<td>14.79</td>
<td>0.53</td>
<td>14.04</td>
</tr>
</tbody>
</table>

* = Statistically significant changes in pain ratings from baseline, $p < .05$.
† = Statistically significant change in pain ratings from exercise, $p < .05$. 
Table 2

Thermal Scaling Ratings of Intensity and Unpleasantness across testing day.

Unpleasantness

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Baseline</th>
<th></th>
<th>Exercise</th>
<th></th>
<th>Competition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>42°C</td>
<td>6.28</td>
<td>0.66</td>
<td>5.34*</td>
<td>0.63</td>
<td>4.34* †</td>
</tr>
<tr>
<td>44°C</td>
<td>7.06</td>
<td>0.67</td>
<td>7.75*</td>
<td>0.82</td>
<td>7.63*</td>
</tr>
<tr>
<td>46°C</td>
<td>10.06</td>
<td>0.76</td>
<td>9.59</td>
<td>0.67</td>
<td>9.25*</td>
</tr>
<tr>
<td>48°C</td>
<td>13.91</td>
<td>0.62</td>
<td>13.38*</td>
<td>0.80</td>
<td>12.53* †</td>
</tr>
</tbody>
</table>

* = Statistically significant changes in pain ratings from baseline, \( p < .05 \).
† = Statistically significant change in pain ratings from exercise, \( p < .05 \).

Intensity

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Baseline</th>
<th></th>
<th>Exercise</th>
<th></th>
<th>Competition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>42°C</td>
<td>8.72</td>
<td>0.80</td>
<td>7.34*</td>
<td>0.63</td>
<td>5.28* †</td>
</tr>
<tr>
<td>44°C</td>
<td>9.34</td>
<td>0.81</td>
<td>9.81</td>
<td>0.84</td>
<td>9.25 †</td>
</tr>
<tr>
<td>46°C</td>
<td>12.20</td>
<td>0.72</td>
<td>11.75</td>
<td>0.70</td>
<td>11.22* †</td>
</tr>
<tr>
<td>48°C</td>
<td>15.69</td>
<td>0.62</td>
<td>14.72*</td>
<td>0.77</td>
<td>14.56 *</td>
</tr>
</tbody>
</table>

* = Statistically significant changes in pain ratings from baseline, \( p < .05 \).
† = Statistically significant change in pain ratings from exercise, \( p < .05 \).
Figure Captions

Figure 1. Athletic differences in Sum of Cold Pressor Ratings for control subjects.

Figure 2. Sex and athletic status differences in systolic and diastolic blood pressures.

Figure 3. Difference in heart rates between athletes and non-athletes.

Figure 4. Heart rate influenced by testing day.

Figure 5. Differences in body awareness across testing day between the sexes and based on athletic status.

Figure 6. Pain thresholds in experimental and control subjects.

Figure 7. Evidence of stress-induced analgesia on thermal scaling ratings at 42°C in experimental subjects.

Figure 8. Thermal Scaling Pain Ratings.

Figure 9. Experimental and Control Cold Pressor Sums.

Figure 10. Difference between athletes and non-athletes on ratings of perceived effort.

Figure 11. Evidence of stress-induced analgesia on thermal scaling ratings in experimental subjects. An interaction between testing day, temperature, and scale.
Figure 1

Cold Pressor ratings of control subjects. * Significant difference in female non-athletes compared to female athletes, $p<.05$. 

[Graph showing the comparison of Cold Pressor ratings between male athletes, male non-athletes, female athletes, and female non-athletes]
Systolic and diastolic blood pressure in control subjects. *Significant difference: female non-athletes lower than female athletes, male athletes, and male non-athletes in systolic and diastolic blood pressure. Male non-athletes had significantly higher diastolic blood pressure compared to male athletes, $p<.05$. 
Figure 3

Heart rate means separated by condition and athletic status. *Significant difference in heart rate, non-athletes' pulse is higher than athletes in both groups, p < .05.
Mean heart rates on three testing days. * Significant increase in pulse on competition day compared to baseline and exercise in experimental subjects (pulse on exercise day also significantly higher than baseline), $p<.05$. No significant change across testing day in control subjects.
Body awareness rating of male and female subjects across all three testing days. Male athletes body awareness is higher during competition than any other session. Female non-athletes body awareness is elevated on exercise and competition day. Male non-athletes and female athletes show no significant changes in body awareness across testing day. Body awareness in control subjects significantly decreases between Day 1 and Day 3, \( p < .05 \).
Figure 6

Heat Pain Threshold

Pain thresholds of subjects on three testing days. * Significant increase in threshold in experimental subjects on competition day compare to exercise and baseline thresholds. No significant change in control subject thresholds across testing day, $p<.05$. 
Thermal scaling pain ratings decrease across day. There is a significant decrease in pain ratings from baseline following exercise that decrease further following competition, $p<.05$. There are no significant changes in pain ratings of control subjects.
Thermal scaling pain ratings separated by stimulus temperature. Pain ratings tend to decrease from baseline to exercise to competition in experimental subjects, however, these changes are significant at 42°C and only marginally significant at 44°C, 46°C, and 48°C. There are no significant changes in the control group.
Figure 9

Cold Pressor ratings across testing day in experimental and control subjects. There is a decrease in pain ratings in the experimental group that approaches significance but there is no significant change in control subjects.
Ratings of perceived effort for experimental subjects. Significant increase on competition day compared to exercise day in athletes, \( p < .05 \).
Unpleasantness ratings on the thermal scaling task for experimental subjects. The pain ratings on competition day and exercise ratings are lower than baseline ratings at all temperatures except 44°C. There is also a significant difference between exercise and competition ratings at the lowest and highest temperatures, $p<.05$.

Intensity ratings on the thermal scaling task for experimental subjects. The pain ratings on competition day and exercise ratings are lower than baseline ratings at 42°C and 48°C, $p<.05$. There is also a significant difference between exercise and competition ratings at all the temperatures except the highest.
Appendix A

Gracely Box Scales of Intensity and Unpleasantness.

<table>
<thead>
<tr>
<th>INTENSITY</th>
<th>UNPLEASANTNESS</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>18</td>
<td>18</td>
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<td>8</td>
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<td>6</td>
<td>6</td>
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<tr>
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<tr>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

EXTREMELY INTENSE

VERY INTENSE

INTENSE

STRONG

SLIGHTLY INTENSE

BARELY STRONG

MODERATE

MILD

VERY MILD

WEAK

VERY WEAK

FAINT

NO PAIN SENSATION

VERY INTOLERABLE

INTOLERABLE

VERY DISTRESSING

SLIGHTLY INTOLERABLE

VERY ANNOYING

DISTRESSING

VERY UNPLEASANT

SLIGHTLY DISTRESSING

ANNOYING

UNPLEASANT

SLIGHTLY ANNOYING

SLIGHTLY UNPLEASANT

NEUTRAL
Appendix B

Medical History Questionnaire, subjects who answer yes to any questions between 10 and 21 are excluded from participating in the study because exercise would not be advised.

**Medical History Questionnaire**

The purpose of this questionnaire is to obtain information about your medical history. It is important that you answer each question honestly and completely in order to minimize the risks associated with your participation in this research. Please ask us if you need clarification about any of the questions. Put a question mark (?) next to any questions that you are not certain about.

1. __________ 4-digit ID (select a number that you can remember)
2. _________ Gender
3. __________ If a female, number of days since the start of your last menstrual period
4. __________ If a female, are you taking any contraceptive medication?
5. __________ If a female, are you pregnant?
6. _________ Age  7.__________ Height  8.__________ Weight
9. _________ Does your mother or father have high blood pressure (i.e., hypertension)?
10. __________ Do you now have, or have you ever had, any heart trouble?
11. __________ Do you frequently suffer from pains in your chest?
12. __________ Do you often feel faint or have spells of severe dizziness?
13. __________ Do you now have, or have you ever had, high blood pressure?
14. _________ Do you have a bone or joint problem, such as arthritis, that has been aggravated by exercise, or might be made worse with exercise?
15. _________ Have you ever fainted during exercise?
16. _________ Has any member of your family died of a heart attack prior to the age of 50?
17. _________ Have you ever had a seizure?
18. _________ Do you regularly smoke cigarettes?
19. _________ Have you ever had an unexplained episode of irregular heart beats, trembling, sweating, difficulty breathing or intense anxiety.
20. _________ Do you have any pain that you have been experiencing for more than a month?
21. _________ Is there a good physical reason not mentioned above why you should not engage in vigorous physical activity? If so, describe it:
Appendix C

The Body Awareness Scale is a subjective measure of a subject’s state of arousal.

**Body Awareness Questionnaire**

<table>
<thead>
<tr>
<th>Subject #: ____________________</th>
<th>Time: ____________________</th>
</tr>
</thead>
<tbody>
<tr>
<td>Date: ________________________</td>
<td>Day/Condition: ____________</td>
</tr>
</tbody>
</table>

**Directions**: A number of statements appear below which people have used to describe their body awareness at different points in time. Read each statement and then circle the appropriate number to the right of the statement to indicate how you FEEL RIGHT NOW AT THIS MOMENT. There are not right or wrong answers. Do not spend too much time on any one statement, and try to give the answer that seems to best describe your feelings right now.

1= Not at all 2= Sometimes 3= Moderately so 4= Very much so

1. I feel tense 1 2 3 4
2. I am aware of my breathing 1 2 3 4
3. My fingertips feel numb or tingle 1 2 3 4
4. I feel lightheaded and dizzy 1 2 3 4
5. I feel calm 1 2 3 4
6. My heart is pounding 1 2 3 4
7. My mouth is dry 1 2 3 4
8. I feel nervous 1 2 3 4
9. I have a lump in my throat 1 2 3 4
10. I feel confident 1 2 3 4
11. My hands are shaking 1 2 3 4
12. I am having difficulty breathing 1 2 3 4
13. My head is throbbing 1 2 3 4
14. I am afraid 1 2 3 4
15. I feel weak and fatigued 1 2 3 4
16. I feel mentally relaxed 1 2 3 4
17. I feel shaky inside (butterflies) 1 2 3 4
18. My vision is blurred 1 2 3 4
19. I have chest discomfort or pain 1 2 3 4
20. I feel cold 1 2 3 4
21. I feel like yawning 1 2 3 4
22. I feel steady 1 2 3 4
Appendix D

Borg's ratings of perceived effort (RPE) scale.

**RATINGS OF PERCEIVED INTENSITY SCALE**

For the amount of exercise you just completed, indicate the degree of effort or intensity you exerted:

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Nothing at all</td>
</tr>
<tr>
<td>1</td>
<td>Very, very weak</td>
</tr>
<tr>
<td>2</td>
<td>Very weak</td>
</tr>
<tr>
<td>3</td>
<td>Weak</td>
</tr>
<tr>
<td>4</td>
<td>Moderate</td>
</tr>
<tr>
<td>5</td>
<td>Somewhat strong</td>
</tr>
<tr>
<td>6</td>
<td>Strong</td>
</tr>
<tr>
<td>7</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Very Strong</td>
</tr>
<tr>
<td>9</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Very, very strong</td>
</tr>
<tr>
<td>12</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Maximal</td>
</tr>
</tbody>
</table>
Appendix E

Consent forms for control and experimental subjects, respectively.

CONSENT FORM

I, _________________________________, agree to participate in a research study titled "EFFECT OF EXERCISE ON PAIN RESPONSES" conducted in the laboratory of Dr. Wendy Sternberg from the Department of Psychology at Haverford College. I understand that my participation is voluntary. I can stop taking part without giving any reason, and without penalty, and I will be paid for my time. I can ask to have all of the information about me returned to me, removed from the research records, or destroyed.

If you volunteer to take part in this study, the following will occur:

Today, we will familiarize you with the procedures involved in the study. You will then return on two separate occasions. During all three sessions (including today), you will have your pain sensitivity tested on three different measures (described below). For participating in all three sessions, we will pay you a total of $25. You will receive partial payment if you discontinue participation. You will be paid at your final session (if you do not return for the 3rd session, contact us so we can send you a check).

Pain testing involves the following:

• Cold Pain Sensitivity: Placing one arm (up to the elbow) in a bucket of ice water for a maximum of 90 seconds. You will rate the pain sensation periodically during the immersion on a 0-20 scale, although you may take your arm out at any time if the pain becomes unbearable.

• Heat Pain Test: You rate the pain sensation (on the same 0-20 scale) that results from a heat probe placed on your forearm for a duration of 5 seconds at a time. The probe will take on 4 different temperatures, some warm, some hot. There will be 8 different placements, each lasting 5 seconds, and the temperature will vary randomly. The temperature range used in this study (and that can be produced by the heat generating apparatus) cannot cause tissue damage. Thus, the heat stimuli cannot burn you. You may have a slight red mark on your skin where the probe comes in contact with your skin, but you will not have any residual pain or any injury as a result of this study.

• Heat Pain Threshold: We will use the same apparatus as in the heat pain test, and the temperature will slowly rise until you say it is painful, then it will cease. Again, no tissue damage can result from exposure to the stimulus.

We will ask you questions about your health and diet, and will periodically also ask you to rate your subjective perceptions of nervousness and anxiety. We will have you provide us with saliva samples on each occasion for analysis of hormones, and we will measure your heart rate and blood pressure during each session.

You will not be identified by name in the data presentation. You will be informed of the study hypotheses (and results, if interested) after participation in the experiment. Please do not discuss the study procedures with anyone until after the study is concluded.

If you have any questions during the study or do not understand any of the information presented on this consent form, please ask.

Your signature below indicates that you have read the description of the experiment and give consent to participate. Your signature also indicates that you have had a physical examination by a physician within the last 5 years and you have not been diagnosed with any of the following conditions: Raynaud’s disease, high blood pressure, chronic pain condition, heart disease, chest pain, frequent dizziness, fainting spells, high blood pressure, arthritis, seizures, smoking, family history of early death due to heart attack, or unexplained heart irregularity or intense anxiety.

Signature: ________________________________
Date: ______
CONSENT FORM

I, _________________________________, agree to participate in a research study titled "EFFECT OF EXERCISE ON PAIN RESPONSES" conducted in the laboratory of Dr. Wendy Sternberg from the Department of Psychology at Haverford College. I understand that my participation is voluntary. I can stop taking part without giving any reason, and without penalty, and I will be paid for my time. I can ask to have all of the information about me returned to me, removed from the research records, or destroyed.

If you volunteer to take part in this study, the following will occur:

Today, we will familiarize you with the procedures and have you perform on the exercise bicycle. You will then return on two separate occasions. During both of these sessions, you will engage in exercise. During all three sessions (including today), you will have your pain sensitivity tested on three different measures (described below). For participating in all three sessions, we will pay you a total of $50. You will receive partial payment if you discontinue participation ($10 for first session; $20 for the second session). You will be paid at your final session (if you do not return for the 3rd session, contact us so we can send you a check).

Pain testing involves the following:

- **Cold Pain Sensitivity:** Placing one arm (up to the elbow) in a bucket of ice water for a maximum of 90 seconds. You will rate the pain sensation periodically during the immersion on a 0-20 scale, although you may take your arm out at any time if the pain becomes unbearable.
- **Heat Pain Test:** You rate the pain sensation (on the same 0-20 scale) that results from a heat probe placed on your forearm for a duration of 5 seconds at a time. The probe will take on 4 different temperatures, some warm, some hot. There will be 8 different placements, each lasting 5 seconds, and the temperature will vary randomly. The temperature range used in this study (and that can be produced by the heat generating apparatus) cannot cause tissue damage. Thus, the heat stimuli cannot burn you. You may have a slight red mark on your skin where the probe comes in contact with your skin, but you will not have any residual pain or any injury as a result of this study.
- **Heat Pain Threshold:** We will use the same apparatus as in the heat pain test, and the temperature will slowly rise until you say it is painful, then it will cease. Again, no tissue damage can result from exposure to the stimulus.

You will be asked to perform the following exercise tasks:

- **Today** your fitness level will be tested by performing a ~10-minute exhausting bout of cycling exercise. During the two remaining sessions, your exercise task will consist of a 20 min cycling bout, while trying to maintain a particular cycling intensity.

We will ask you questions about your health and diet, and will periodically also ask you to rate your subjective perceptions of nervousness and anxiety. We will have you provide us with saliva samples on each occasion for analysis of hormones, and we will measure your heart rate and blood pressure during each session.

You will not be identified by name in the data presentation. You will be informed of the study hypotheses (and results, if interested) after participation in the experiment. Please do not discuss the study procedures with anyone until after the study is concluded.

If you have any questions during the study or do not understand any of the information presented on this consent form, please ask.

Your signature below indicates that you have read the description of the experiment and give consent to participate. Your signature also indicates that you have had a physical examination by a physician within the last 5 years and you have not been diagnosed with any of the following conditions for which strenuous exercise (or exposure to the painful stimuli used in this experiment) would be contraindicated: Raynaud’s disease, high blood pressure, chronic pain condition, heart disease, chest pain, frequent dizziness, fainting spells, high blood pressure, arthritis, seizures, smoking, family history of early death due to heart attack, or unexplained heart irregularity or intense anxiety.

Signature: ________________________ Date: ______