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The Effects of Anticipatory Stress on Pain Responses in Male and Female Athletes

Sarah Nagle
Haverford College
Caryn Dolich, Emily Hurwitz, Jeff Ratliff
Wendy Sternberg, Thesis Advisor
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Abstract

Athletic competition elicits a stress response that is both physical and psychological in nature. Additionally, following athletic competition, an increase in pain threshold (analgesia) has been observed. However, the mechanisms that produce analgesia due to athletic competition have not been systematically investigated. The present study sought to determine if the psychological stress in anticipation of an athletic competition is alone able to induce an analgesic state by evaluating sympathetic nervous system measures (heart rate, blood pressure), cortisol levels, self-rated anxiety scores, and pain thresholds in male and females soccer players and in female basketball players prior to a game and a practice, and on a non-competitive day. The results suggest that there is an increase in pain threshold (analgesia) in anticipation of both an athletic contest and a team practice session and that this increase may be associated with increases in cortisol levels, sympathetic nervous system measures, and self-rated measures of anxiety. Future research on coaching styles, winning expectancies, and the social context surrounding the competition would help clarify the present findings.
The Effects of Anticipatory Stress on Pain Responses in Male and Female Athletes

Pain signals the body of tissue damage (real or potential). Feelings of pain are generated so that the body does not undergo more harm, attention can be paid to the painful area, and the problem can be fixed. However, during the stress response, mechanisms within the body that are potentially detrimental long-term are activated so that the body can more effectively respond to stress without being hindered by pain, a phenomenon known as stress-induced analgesia. For instance, during competition, athletes do not tend to exhibit a pain reaction to injuries that typically elicit large pain responses in other environmental circumstances. The present study evaluates the stress response in anticipation of athletic competition in male and female athletes. More specifically, this study sought to determine if pain thresholds are increased in anticipation of an athletic competition as compared with a non-competitive or practice session in male and female athletes as a result of stress-induced analgesia.

Pain versus Nociception

Pain signals the presence of potential or actual tissue damage and is therefore essential to the well being of a person. There is, however, a difference between the perceptual experience of pain and the physiological experience of nociception. Pain is a complex perception, the brain’s unique interpretation of sensory input influenced by both the environment and one’s emotional state (Basbaum & Jessell, 2000). Nociception, in contrast, is a physiological experience that refers to activity in the neural circuits.
Interestingly, the brain itself lacks nociceptors, and therefore cannot experience pain (Carlson, 2004).

Beecher (1956, as cited in Gatchel, 1999) first demonstrated that the pain experience could be modified by psychological factors through studies of World War II soldiers. He compared the requests of soldiers returning from battle for pain medication to the requests of civilians with similar wounds and found that only 25% of the soldiers requested pain medication as compared with over 80% of the civilians. Many soldiers expressed that their pain was not sufficient to require medication, suggesting the presence of a psychological component to pain.

Even before Beecher, individuals have always had a desire to understand and to control pain. As far back as 4000 B.C., the ancient Egyptians attempted to treat pain; over 2000 years ago, acupuncture was used to alleviate pain in China. As time progressed, medical practitioners began to understand that pain was both physical and psychological. The Greek physician Hippocrates (400-300 B.C.), for instance, believed that pain and other physical or mental illnesses resulted from an imbalance of the four humors, or bodily fluids. By the 17th century, the study of medicine was more scientific, as Decartes argued that pain was a result of a specific type of activity in the sensory nervous system. More recently, 19th century scientists argued that one must better understand pain in order to treat it more effectively (Gatchel, 1999).

**Primary Afferent Nociceptors**

The understanding of pain has evolved since the 19th century. Scientists now understand that the perceptual experience of pain begins with primary afferent
nociceptors, which are relatively unspecialized nerve cells throughout the skin and in deep tissues that respond exclusively to noxious stimuli. Primary afferent nociceptors project from the site of tissue damage (real or potential) to the dorsal root ganglion, a collection of cells that supply information to the spinal cord.

There are three major types of nociceptors, all of which transmit nociceptive information fairly slowly. However, there are both relatively fast and relatively slow nociceptive pathways which are each important for different reasons. The three types of nociceptors are A\(\delta\) mechanothermal, A\(\delta\) mechanosensitive, and polymodal C fibers; each plays a unique role in the perception of pain. A\(\delta\) mechanothermal and A\(\delta\) mechanosensitive nociceptors both have fairly small diameters, are thinly myelinated, and conduct relatively quickly (as compared with other nociceptors) at speeds of approximately twenty meters per second (Fields, 1987). A\(\delta\) mechanothermal and A\(\delta\) mechanosensitive nociceptors are activated by different stimuli; A\(\delta\) mechanothermal nociceptors are stimulated by extreme temperatures of greater than 45°C or less than 5°C whereas A\(\delta\) mechanosensitive nociceptors are activated by intense pressure. Polymodal C fibers also have small diameters, but are nonmyelinated, and therefore conduct at much slower rates than the other two (about two meters per second). Nociceptive C fibers are classified as polymodal because they are activated by mechanical, chemical, and thermal stimuli (Basbaum & Jessell, 2000).

All nociceptors have two main functions: transduction and transmission. Initially, during transduction, a noxious stimulus (chemical, mechanical, or thermal) depolarizes the nociceptor and creates a nerve impulse that can be recognized by the brain. Subsequently, through the process of transmission, nerve impulses are conducted to the
spinal cord and to other central nervous system structures to create the sensation of pain. Transmission generates several distinct pain responses such as withdrawal reflexes or the subjective reaction to the event (Fields, 1987).

Application of a noxious stimulus generates two separate sensations. An early, sharp, brief prickling described as the first pain, is produced by lower threshold Aδ fibers (mechanothermal or mechanosensitive depending on the nature of the stimulus). Following the first pain is a dull and prolonged second pain, for which the higher threshold polymodal C fibers are responsible (Fields, 1987). Therefore, each type of fiber contributes uniquely to an individual’s perception of pain.

**Nociceptive Pathways in the Central Nervous System**

Primary afferent nociceptors project to the dorsal horn of the spinal cord where they interact with spinal cord neurons. The axons of spinal cord neurons cross to the anterolateral quadrant of the spinal cord on the side opposite the nociceptors and project to higher centers in the brain that contribute to the pain response (Fields, 1987).

The majority of primary nociceptive afferents enter the spinal cord through the dorsal root. Axons in the dorsal root carry sensory information to the spinal cord and the brain. As the dorsal root approaches the spinal cord, at the ventrolateral section of the dorsal root there is a separation between the small diameter myelinated axons and the unmyelinated axons. Consequently, when the primary afferent nociceptors enter the dorsal horn of the spinal cord, large diameter and small diameter nociceptive fibers are completely separated (Fields, 1987).
In the dorsal horn of the spinal cord, there are three types of neurons: projection neurons, excitatory interneurons, and inhibitory interneurons. Each type of neuron contributes uniquely to the experience of nociception. Primarily found in sections of the dorsal horn that receive direct input from primary afferent nociceptors (laminae I, II, and V), projection neurons fire maximally in response to noxious stimuli and send nociceptive information to higher brain centers that play a role in pain perception. Excitatory interneurons, in contrast, send nociceptive information to projection cells, to other interneurons, or to motor neurons that control spinal reflexes. Inhibitory interneurons too send nociceptive information to other neurons and play a role in the control of nociceptive transmission (Fields, 1987).

The dorsal horn is divided into ten distinct sections, or laminae. The neurons in the superficial layers of the dorsal horn (primarily laminae I, II, and V) play an important role in nociception. Lamina I neurons are predominately projection cells that receive input directly from myelinated and unmyelinated nociceptors and that respond maximally to noxious stimulation. Most importantly, lamina I projection neurons extend to higher brain centers, such as the thalamus, that have an important role in pain processing that will be discussed later (Fields, 1987).

The majority of the neurons in lamina II are interneurons (excitatory and inhibitory) that respond to both nociceptive and nonnociceptive information. Lamina II interneurons make connections with other lamina II interneurons or projection neurons in laminae I or V. Stalk cells of lamina II are excitatory interneurons that send axons to neurons in lamina I projection cells (Fields, 1987).
Like lamina I neurons, cells in lamina V as well as in deeper layers of the dorsal horn respond maximally to noxious stimuli. However, fewer primary afferent neurons terminate in lamina V than in laminae I or II. Lamina V consists of both projection neurons and interneurons; lamina V interneurons make connections with cells in laminae I and II and lamina V projection neurons carry nociceptive information to higher brain centers such as the brainstem and the thalamus. Deeper layers of the dorsal horn also contain nociceptive neurons that project to higher brain regions such as the thalamus or the reticular formation, but their functions are more complex (Fields 1987).

Nociceptive information from neurons that make up the laminae of the dorsal horn is carried to the brain through five major ascending pathways; each pathway carries nociceptive information to different brain regions, contributing differentially to the pain response. The pain response is comprised of both an affective and a sensory component; the aspect of the pain response to which each ascending pathway contributes depends on the brain region to which it projects. The sensory-discriminative facet of pain signals the location, intensity, and quality of the noxious stimulus (Purves et al., 2004). The sensory-discriminative characteristic of pain is generally consistent within an individual and even among individuals. It includes information such as identification of the stimulus and pain threshold. Conversely, the affective-motivational component (pain tolerance), the unpleasant feeling that accompanies the perception of pain, is not consistent across individuals as the level of anxiety in response to a noxious stimulus varies from person to person. The affective-motivational component of the pain response signals autonomic activation, the fight-or-flight response to a noxious stimulus (Fields,
Each nociceptive pathway in the spinal cord projects to a brain region that is involved in either the sensory-discriminative or affective-motivational aspect of pain.

The major ascending pathway in the spinal cord that transports nociceptive information is the spinothalamic tract, which consists of neurons from lamina I as well as deeper layers of the dorsal horn. Axons of the spinothalamic tract terminate in the thalamus, which sends the nociceptive information to the somatosensory cortex. Axons originating in the lateral cervical nucleus (which receives input from neurons in deeper lamina layers) form the cervicothalamic tract, which also terminates in the thalamus (Basbaum & Jessell, 2000).

The thalamus is the relay center of the brain. It receives auditory, somatosensory, and visual signals and sends them to the cerebral cortex. Therefore, it takes incoming nociceptive information and sends it to areas of the cerebral cortex that aid in both the sensory-discriminative and the affective-motivational perception of pain (Basbaum & Jessell, 2000).

The thalamus is comprised of the medial nuclear group and the lateral nuclear group; each plays a different role in pain processing. The medial nuclear group of the thalamus receives input from neurons in deeper layers of the dorsal horn and from the reticular formation of the brainstem. Neurons in the medial nuclear group are important in the affective-motivational processing of nociceptive information and in the activation of a nonspecific arousal system. Conversely, the lateral nuclear group of the thalamus gets input from neurons of laminae I and V of the dorsal horn. This section of the thalamus is concerned with the sensory-discriminative processing of information about the location of an injury (Basbaum & Jessell, 2000).
The spinoreticular tract also carries nociceptive information to the thalamus. However, this pathway consists of neurons from deeper layers of the dorsal horn and transports nociceptive information to the reticular formation. The reticular formation is located in the brainstem; its structures that aid in the perception of pain are the periaqueductal gray matter and the medulla, which primarily contribute to pain modulation. The reticular formation is the arousal center for the brain and is therefore involved in the affective-motivational aspect of pain perception (Basbaum & Jessell, 2000).

Neurons from laminae I and V comprise the spinomesencephalic tract, which also projects to the reticular formation and the periaqueductal gray matter. The periaqueductal gray matter is particularly important in pain suppression. Opioid receptors in the periaqueductal gray matter respond to opiate analgesics, agents of pain suppression that will be discussed in more detail later. Within the brain, neurons of the spinomesencephalic tract (via the spinobrachial tract) also project to the amygdala, part of the limbic system that regulates the brain’s response to emotion. Therefore, the spinomesencephalic tract carries information important for the affective-motivational component of pain (Basbaum & Jessell, 2000).

Axons from neurons in laminae I and V as well as deeper layers comprise the spinohypothalamic tract which projects to brain regions important for the regulation of the endocrine system and autonomic control such as the hypothalamus. The hypothalamus connects the nervous system to the endocrine system. The spinohypothalamic tract is therefore essential for the autonomic responses that
accompany the perception of pain such as increases in heart rate and blood pressure (Basbaum & Jessell, 2000).

Other regions in the brain, primarily in the cerebral cortex, are necessary for the perception of pain. Regions in the cerebral cortex generally receive projections from other brain regions (such as the thalamus) that indicate the presence of noxious stimuli. The somatosensory cortex, the cingulate cortex, the insular cortex, and the frontal lobe all respond to nociceptive input. Each of these areas except for the frontal lobe is activated in response to thermal stimuli. The cingulate cortex, mainly the cingulate gyrus and the anterior cingulate cortex, are involved in pain processing. The cingulate cortex is a part of the limbic system and is therefore essential for processing the emotional component of pain. The insular cortex receives projections from medial nuclei of the thalamus and therefore contributes to the autonomic component of the pain response. The insular cortex is also important for the integration of the sensory, affective, and cognitive components of pain perception (Basbaum & Jessell, 2000). Clinical observations of patients with frontal lobe lesions in addition to the presence of a pathway that carries nociceptive information from the thalamus to the frontal lobe indicate that the frontal lobe may be important in processing the affective component of pain (Fields, 1987).

Pain Modulation

In addition to the multiple ascending pain pathways in the spinal cord and in the brain that contribute to the perception of pain, there are circuits that are integral for modulating nociceptive information and altering the pain experience. At the spinal cord, the initial site of pain modulation, nociceptive and nonnociceptive fibers interact to
determine what nociceptive information is projected to higher brain centers (Basbaum & Jessell, 2000).

The gate control theory of pain, introduced by Melzack and Wall, first established the idea that modulatory events within the spinal cord in addition to nociceptive peripheral activity contributes to the experience of pain. Observations of neurons in the spinal cord led Melzack and Wall to question existing theories of pain modulation. They observed a number of phenomena: (1) Neurons in laminae I and V obtain excitatory input from both nonnociceptive Aβ fibers (fibers of larger diameter that are not involved pain processing) as well as nociceptive Aδ and C fibers; (2) Aβ fibers inhibit neuronal firing of cells in lamina V through activation of interneurons in lamina II; and (3) Aδ and C fibers can excite neurons in lamina V and simultaneously inhibit the firing of lamina II interneurons activated by nonnociceptive Aβ fibers (as cited in Basbaum & Jessell, 2000).

From these observations, Melzack and Wall refuted the standing theory, which argued that there was a direct relationship between stimulus and sensation, pain was the result of straight-through transmission, and instead hypothesized that the pain experience was a complex phenomenon with multiple opportunities for modification. Furthermore, the balance of activity in nociceptive and nonnociceptive fibers within the spinal cord appeared to alter pain perception, with nociceptive fibers opening and the nonnociceptive fibers closing a gate to the control of noxious input (Melzack & Wall, 1965).

The idea that pain perception could be modulated within the spinal cord and ascending pathways to the brain led to the idea of descending inhibition – influence from higher order brain centers could also close the gate. Evidence for descending inhibition
of pain has come from brain stimulation studies. Stimulation of certain brain regions, such as the periaqueductal gray matter of the midbrain, produces analgesia but not insensitivity to sensory stimuli; this indicates the presence of unique descending modulatory pathways for pain. When the periaqueductal gray matter was stimulated in rats, they could still respond to touch, pressure and temperature, but surprisingly, they did not respond to pain induced by hemostat-applied pressure or typically painful surgical procedures, lacking characteristic withdrawal reflexes (Reynolds, 1969).

Stimulation of other brain regions such as the parabrachial nucleus, the reticular formation, and the locus coeruleus, a region responsible for the physiological reactions to stress, produces analgesia as well. Brain stimulation produces analgesia because neurons project from the brain to the dorsal horn of the spinal cord, make inhibitory connections with neurons in laminae I, II, and V, and prevent them from firing (Basbaum, Clanton, & Fields, 1976; Purves et al., 2004).

Additionally, endogenous opiate-containing circuits in the dorsal horn inhibit the firing of nociceptive neurons to enable brain stimulation to produce analgesia (Basbaum & Jessell, 2000). Scientists discovered that naloxone, an opiate antagonist that blocks the action of opiates, reversed analgesia produced by stimulation of the periaqueductal gray matter in rats (Akil, Mayer, & Liebeskind, 1976). Naloxone blocks the action of opiates by binding to a receptor (the µ receptor), displacing the opiate peptide, but not activating the receptor, and therefore enabling pain to return. Reversal of analgesia by naloxone indicates that stimulation-produced analgesia is opioid-mediated (Basbaum & Jessell, 2000).
Opiates (endogenous and exogenous) themselves can effectively produce analgesia along the same descending pathways active during stimulation-produced analgesia. Three major classes of opiate receptors have been identified in the brain: µ, δ, and κ. Appropriately, there are also three opioid peptides – enkephalins, β-endorphin, and dynorphins. Each type of peptide is present at sites associated with the processing or modulation of nociception in the body. Enkephalins, active at both µ and δ receptors, are primarily in the periaqueductal gray matter, the rostral ventral medulla, and laminae I and II of the dorsal horn of the spinal cord. Beta-endorphin (along with ACTH) is released into the bloodstream in response to stress and is therefore primarily found in the region of the brain related to the stress response, the hypothalamus. Dynorphins typically bind to κ receptors in the periaqueductal gray matter, the rostral ventral medulla, and laminae I and II of the dorsal horn of the spinal cord (Basbaum & Jessell, 2000).

Stimulation-produced analgesia in humans, as in animals, works through endogenous opioid pathways. Electrical stimulation of the central gray matter in humans has been used to relieve chronic pain that was not alleviated by narcotic analgesics (suggesting that stimulation-produced analgesia can be a more effective analgesic than non-endogenous opioids); naloxone, however, blocks the analgesic effects of brain stimulation (Hosobuchi, Adams, & Linchitz, 1977). Stimulation of the periaqueductal gray matter in humans results in an increase of β-endorphin, the opiate peptide involved in the stress response. This suggests a connection between stimulation-produced analgesia, endogenous opiate pathways, and the stress response, indicating that stress can act as a natural trigger for stimulation-produced analgesia (Hosobuchi, Rossier, Bloom, & Guillemin, 1979).
Pain Measurement

A range of methods in both animal and human models have been used to measure pain responses to different types of stimuli. Analgesia in animal models is generally measured by reflex tests (tail flick, flinch jump) or thermal stimuli (hot plate test). Reflex tests evaluate analgesia at the level of the spinal cord. During the tail flick test, heat is applied to the tail of a rodent and latency to flick the tail is measured to determine analgesia. However, during the flinch jump test, a series of electrical shocks of increasing intensity are given to the animal and behavioral responses such as flinch threshold, jump threshold, and escape threshold measure analgesia. Thermal stimuli such as the hot plate test evaluate analgesia at levels higher than the spinal cord in the central nervous system. During the hot plate test, the rodent is placed on a hot plate, and their latency to lick their paw or jump is recorded (Amit & Galina, 1986).

Multiple types of stimuli are used to assess analgesia in human models as well. As in animal models, analgesia in humans can be measured by reflex, ischemic, and thermal tests. Lower limb reflex tests attempt to measure pain objectively by stimulating the sural nerve by electrodes on the surface of the skin and recording flexion reflexes from a lower limb through surface electrodes (Willer, Dehen, & Cambier, 1981). Thermal and ischemic pain tests, however, record a more perceptual measure of pain, requiring subjects to indicate when a stimulus first becomes painful. Ischemic pain tests, for example, replicate the aching pain due to illness by restricting blood flow in the arm with a tourniquet, having the subject contract his or her arm muscle and subsequently indicate the intensity of the pain (Johnson & Tabasam, 2003). During temperature tests,
subjects are asked to indicate when a cold or hot stimulus first becomes painful. The cold pressor test requires subjects to place an arm in a plastic container filled with ice water; analgesia measurements are made based on changes in pain intensity and unpleasantness ratings. Similarly, latency of a withdrawal response from a radiant heat source also measures analgesia. During this test, subjects place their forearms and fingertips over the radiant heat source and remove them when the source becomes too painful; the temperature at which subjects withdraw from the heat source indicates their pain threshold (Sternberg, Bokat, Kass, Alboyadjian, & Gracely, 2001).

*The Stress Response*

In humans, two endocrine systems dominate the stress response. In reaction to a stressor, the medulla of the adrenal gland secretes epinephrine while the cortex of the adrenal gland secretes glucocorticoids. Both psychological (e.g. financial problems) and physical (e.g. starvation) stressors activate epinephrine and glucocorticoid secretion, disrupting a person’s physiological balance, and activating a “fight-or-flight” response. An individual can either face the threat directly or flee (Sapolsky, 1992). Although a person’s behavioral response to a threat depends on the nature of the stressor, multiple types of stressors trigger a coordinated set of physiological changes (epinephrine and glucocorticoid secretion) in all individuals. This way, the body can mount a successful response to the perceived threat and most effectively reestablish its physiological balance (Taylor et al., 2000).

A cascade of events prefaces epinephrine and glucocorticoid secretion. When a stressor is perceived, the hypothalamic-pituitary-adrenal (HPA) axis is activated, initially
releasing corticotrophin-releasing hormone (CRH) from the hypothalamus. CRH causes the pituitary gland to release adrenocorticotropic hormone (ACTH), which is carried to the adrenal cortex and results in the release of cortisol, a glucocorticoid. Additionally, the sympathetic nervous system is activated and epinephrine is released (Sapolsky, 1992).

Several studies have demonstrated the rise in stress hormones, such as cortisol, prior to athletic competition, suggesting that games and matches are sufficient stressors to elicit a stress response (Bateup, Booth, Shirtcliff, & Granger, 2002; Salvador, Suay, Gonzalez-Bono, & Serrano, 2003).

In a preliminary study, we tested cortisol levels of four female varsity college soccer players prior to and following both a practice and a game. Saliva samples were taken six times over three sessions: baseline (baseline one and baseline two were separated by two hours), practice (directly before and directly afterwards), and competition (directly before and directly afterwards). Each sample was taken at a similar time during the day so not to be confounded with the natural daily pattern of cortisol secretion. The results of the study suggest a trend towards an increase in cortisol levels prior to a game as compared with a practice or a baseline session, an increase in cortisol following both a practice and a game (as compared with samples taken before the game or practice), and an overall heightened cortisol response during the competition condition (Figure 1). This trend in cortisol secretion suggests that athletes perceive meaningful athletic competition as a more stressful event than a practice or a non-competitive session.

Stressors (such as competition) differentially affect cortisol responses in males and females. Males display a greater increase in cortisol concentrations than females do
in response to a noxious stress, perhaps indicating that the male HPA axis is more susceptible to stress (Zimmer, Basler, Vedder, & Lautenbacher, 2002). Separate research has suggested that sex differences in the stress response can be explained by different behavioral coping strategies in males and females. While males typically exhibit a “fight-or-flight” response to stressors, females, in response to their social role, are more inclined to respond in an affiliative “tend-and-befriend” manner. Females respond to a perceived threat by protecting their young and reducing neuroendocrine responses that could jeopardize the health of their offspring (tending) and affiliating with social groups to reduce their vulnerability to potentially harmful situations (befriending) (Taylor et al., 2000).

**Stress-Induced Analgesia**

Research of both animal and human models indicates that there are two distinct systems by which stress can induce analgesia; stress-induced analgesia can be both opioid mediated and non-opioid mediated. Opioid and non-opioid mechanisms are differentially activated in both animal and human models, and the majority of research supports the presence of both mechanisms in the analgesic stress response.

Opioid mediated stress-induced analgesia requires the release of endogenous opiates in the central nervous system. Opiate peptides such as β-endorphin are secreted by the pituitary gland during the stress response to induce analgesia through an opioid mechanism just as in stimulation-produced analgesia (Amir, Brown, & Amit, 1980).

Multiple experiments in rodents indicate that stress-induced analgesia is primarily opioid mediated. A stressor commonly used in animal models is the swim stress, which
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consists of both a physical stress (exercise) and a psychological stress (uncertainty, a need for survival). Analgesia (measured via a hind limb flick in response to a hot plate) elicited by the swim stress in mice is reversed by naloxone under most circumstances (Willow, Carmody, & Carroll, 1980). In particular, opioid mediated stress in animals appears to predominate during swims of longer duration (greater than 3 minutes) and in warm (greater than 32°C) water (Tierney, Carmody, & Jamieson, 1991; O’Connor & Chipkin, 1984).

With changes in water temperature and swim duration, however, animal studies have suggested the presence of a strong non-opioid component to swim stress-induced analgesia; the analgesic effects present both at the beginning of a swim and following a cold water swim are not reversed by naloxone, suggesting that they are non-opioid mediated (Tierney, Carmody, & Jamieson, 1991; O’Connor & Chipkin, 1984). For example, naloxone does not affect analgesia in mice induced by a 15°C swim. However, dizocilpine, a NMDA antagonist completely blocks analgesia in mice following that same swim, indicating that cold-water swims are primarily non-opioid mediated (Marek, Mogil, Sternberg, & Liebeskind, 1992).

The non-opioid mechanism of stress-induced analgesia is not completely known, but studies similar to the one mentioned above have suggested that the NMDA receptor is an important component of non-opioid stress-induced analgesia (Marek, Mogil, Sternberg, & Liebeskind, 1992). The neuropeptide vasopressin has also been suggested to mediate non-opioid stress-induced analgesia because vasopressin antagonists lead to a reduction in pain threshold. As with all non-opioid analgesics, vasopressin does not bind to the opioid receptor, μ as opioid analgesics do. Although multiple systems appear to
play a role in non-opioid mediated analgesia, more research regarding its mechanism is undoubtedly necessary (Amit & Galina, 1986).

Stress-induced analgesia studies using human models also suggest that opioid and non-opioid mediated reactions to stress arise under different circumstances. In humans, both intensity and temporal pattern of the stressor regulate which system is activated. Opioid mediated stress-induced analgesia appears to result when a stressor is brief or weak, whereas non-opioid mediated stress induced analgesia occurs when a stressor is more intense or prolonged (Willer et al., 1981; Janal, Colt, Clark, & Glusman, 1984).

Illustrating that weak and brief stressors are opioid mediated is research demonstrating that early in the pain experience, naloxone can partially reverse the analgesic effects (measured via nociceptive flexion reflex) of a repetitive stress (anticipation of inescapable and noxious foot shock) in humans. However, pain thresholds increase again after approximately twenty minutes, suggesting the presence of a non-opioid system as well (Willer et al., 1981). Stress-induced analgesia due to other stressors, such as novice parachute jumping, is also mediated by an endogenous opioid system. While $\beta$-endorphin levels increased in every subject, pain sensitivity to electrical stimulation and pressure pain decreased significantly when naloxone was administered to subjects as compared with a saline placebo (Janssen & Arntz, 2001).

Multiple types of stressors can activate both opioid and non-opioid systems in stress-induced analgesia. Exercise is one stressor that has been studied in depth, particularly in humans. Thermal pain thresholds of pilots, for instance, increased significantly following cycle ergometry exercise (Kemppainen, Hamalainen, & Kononen, 1998). Exercise appears to be a stressor that is mediated by both opioid and non-opioid
mechanisms. Pain thresholds to thermal, cold pressor, and ischemic stimuli increased in runners in response to a 10km run at about 85% of maximum aerobic capacity; naloxone, however, was only able to reverse analgesia in ischemic stimulation (Janal, et al., 1984).

Exercise is a stressor primarily because it is physically taxing. Studies have consistently shown that physical activity generates a hormonal stress response such that cortisol, plasma β-endorphin, and catecholamines all increase significantly during exercise (Droste, Greenlee, Schreck, & Roskamm, 1991). However, the degree of the increase depends on the intensity, duration, and type of exercise in addition to the physical fitness level of the subject (Moya-Albiol, Salvador, Gonzalez-Bono, Martinez-Sanchis, & Costa, 2001).

Athletic competition is a personally meaningful form of exercise that is both psychologically and physically stressful and has been shown to induce analgesia in humans. Past research comparing changes in pain thresholds (analgesia) of male and female athletes immediately following athletic competition to a baseline session has suggested that athletic competition alters an athlete’s response to noxious stimuli. Pain sensitivity was evaluated in male and females athletes (track runners, basketball players, and fencers) via the cold pressor test and arm and finger withdrawal latencies to noxious heat two days before, directly following, and two days after a competition. Immediately following competition, athletes were less sensitive to both the cold pressor test and noxious heat on the arm. Additionally, most athletes showed increased pain sensitivity to noxious heat on the fingertips directly after competition as compared with baseline days. The authors explain the difference in changes in pain sensitivity by indicating that fingertips have more sensory innervations than do forearms and therefore a heightened
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awareness to peripheral stimulation during an athletic competition is expected (Sternberg, Bailin, Grant, & Gracely, 1998).

The primary limitation of the aforementioned study was its failure to separate the physical and psychological stresses associated with athletic competition; it is unknown which type of stressor generated an increase in pain threshold (analgesia) that was observed in athletes’ arms following competition. This distinction is important because of the considerable difference in physical exertion between the conditions coupled with the ability of exercise to increase stress levels and induce an analgesic state in individuals. During the competition session, athletes’ pain responses were evaluated directly following competition, and so the athletes had just exercised vigorously. However, during the other sessions (baseline and control), both athletes and non-athlete controls had not exercised vigorously within 12-24 hours. Although the authors did not attempt to separate the physical and psychological stresses associated with athletic competition they created an opportunity for future studies to do so (Sternberg, Bailin, Grant, & Gracely, 1998).

Attempts to separate the psychological and physical stressors associated with competition have generated mixed results. One study attempted to distinguish between the physical and cognitive facets of athletic competition by requiring subjects to perform both the physical aspect of athletic competition alone (treadmill running) and the cognitive aspect of athletic competition alone (a sedentary videogame competition). The cold pressor test was administered to athletes and non-athletes on a baseline day and immediately following a track meet, treadmill exercise, or videogame playing. The results of this study indicate the presence of sex differences; that is, stress responses to
athletic competition in male and female athletes appear to result from different factors. In male athletes, athletic competition is stressful because of the psychological aspects associated with it, whereas athletic competition is stressful for females because of its physical aspects (Sternberg et al., 2001).

Although it is quite possible that different aspects of competition trigger an analgesic response in male and female athletes, it is also likely that the sedentary competition of videogame playing was not sufficiently meaningful to induce analgesia in females. Competition most likely has to be meaningful to induce a physiological stress response; competing against another person in videogames is less likely to be meaningful for most females compared to males due to lower frequency of the activity and lower competence at the task. At the same time, videogame competition may not induce the same psychological state as athletic competition. As a result, questions still remain as to whether or not the psychological stress associated with athletic competition alone is enough to induce analgesia.

Each of the above studies evaluated analgesia following an athletic competition. It is equally as important to measure analgesia in anticipation of an athletic competition because pain threshold induced by athletic competition may begin to diminish directly following a game or race. It is also of interest to evaluate anticipatory stress because, as previously mentioned, research has suggested a rise in stress hormones (cortisol) prior to competition (Bateup et al., 2002; Salvador et al., 2003), but does not indicate if there is a corresponding increase in sympathetic nervous system measures (heart rate and blood pressure) and pain threshold (analgesia), both of which are typically associated with the stress reaction.
The present study attempts to address the limitations of the previous studies. Its purpose is three-fold; the study aims to (1) Assess anticipatory stress in male and female athletes through cortisol levels, sympathetic nervous system measures (heart rate and blood pressure), and pain threshold (analgesia measured by thermal stimuli) due to athletic competition as compared with a practice and a baseline condition; (2) Determine if the observed increases in stress (measured by cortisol levels) in anticipation of athletic competition will also be detected in sympathetic nervous system measures (heart rate and blood pressure) and pain threshold (analgesia); and (3) Determine if the psychological stress due to competition is alone able to induce an analgesic state.

In order to dissociate the analgesic effects of competition from the analgesic effects of exercise, subjects in the present study participated in an equivalent amount of exercise during each testing session. During practice and game sessions, subjects completed a team warm-up and during the baseline session, each participant rode an exercise bike for five minutes. Exercise-induced analgesia is only produced in humans following a high level of exercise with a workload of at least 74% of aerobic capacity (Pertocaara, Huopaniemi, Virtanen, & Johansson, 1984). Each testing session (baseline, practice, and game) was not aerobically challenging for the participants, and therefore was unlikely to lead to exercise-induced analgesia.

We anticipate that meaningful competition creates a more psychologically stressful environment for an athlete than does a practice or a baseline session. We therefore expect that sympathetic measures (heart rate and blood pressure), cortisol levels, and pain threshold will all increase in male and female soccer players and in
female basketball players in anticipation of a game relative to a practice and a baseline session.

**Method**

**Experiment 1**

*Subjects*

Subjects included 9 male and 11 female Haverford College varsity soccer players between 18 and 21 years of age. They were recruited depending on their playing time. Athletes who started or played at least 25% of each game were asked to participate in the study. In addition to an orientation, all subjects participated in three sessions over a one week period – baseline, practice, and game and were reimbursed $30.00 for their time. The sessions were counterbalanced to rule out repeated measures effects.

*Exercise*

Since the experimental testing sessions involved warm-up exercise, we strove to make the exercise portion of each session similar so as to rule out potential differences caused by exercise. Baseline sessions took place in the lab and subjects were first asked to ride a stationary exercise bike for five minutes at 100 watts and 70-80 rpm. Because cardiovascular fitness was different for each subject (especially between men and women), subjects were asked to use the above as a guideline but to focus on increasing their heart rate and “working up a sweat.” Practice sessions were conducted after the team had warmed up – about 30 minutes into practice. Team warm-ups included jogging and stretching. Similarly, game sessions were conducted about 30 minutes into the warm-up and 15 minutes before the game.
Tests of Pain Threshold

Subjects were tested for pain threshold on both the forearm and fingertips during each session. Tests for pain threshold were conducted by application of a radiant heat source (Medoc Neurosensory Analyzer) to the subject’s forearms and fingertips. The probe was removed from the subject’s skin and the temperature at which the subject first reported pain was recorded.

The subject was oriented to the machine and the process of sensory decision making during his or her orientation session by a series of warmth demo sessions. Each subject was then briefly put through the actual test so that they could determine when the probe became painful. During each session, every subject was asked to verbally indicate when the probe became painful.

The heat probe started at each subject’s arm temperature which was initially measured via a noncontact infrared thermometer (Kent Scientific Model C-1600MP). It increased at a rate of 1 °C per second until the subject indicated it was painful or the temperature hit the maximum of 50 °C. Heat was applied to three places on each forearm and the three middle fingertips on each hand for a total of twelve measurements. The temperatures at which the subject indicated that the heat had turned into pain were averaged separately for forearm and fingertips. The pain threshold was determined as the difference between the subject’s initial arm temperature and the average temperature at which he or she said the stimulus was painful for the forearm and the fingertips.

Body Awareness Questionnaires
BAQs were paper and pencil questionnaires given to each subject during each session (Appendix A). They consisted of twenty-two statements that addressed the subjective reports of anxiety and alertness in each individual at that particular time. The statements were accompanied by a Likert Scale from 1 (not at all) to 4 (very much so), and subjects were asked to circle a number one to four in response to each question (O’Connor, Raglin, & Morgan, 1996).

**Practice/Game Intensity and Importance Ratings**

During the game and practice sessions, subjects were asked how much effort the upcoming game or practice would cause them to exert (Appendix B). Each subject was asked to circle a number between 0 (nothing at all) and 13 (maximal). Additionally, during the game session, subjects were asked to rate the importance of the game on a scale from one to ten, with ten being extremely important. These measurements were not used during the baseline session.

**Blood Pressure/Heart Rate**

Blood pressure and heart rate were measured during each session with a portable device (HEM-609 Wrist Blood Pressure Monitor). The measurements were taken approximately five to seven minutes after the subject had completed exercising so that exercise would not interfere with the measurements.

**Cortisol Assay**
During every session, each subject was asked to chew on a piece of gauze for 30 seconds or until it was saturated. The gauze was then squeezed into a mini Eppendorf tube and refrigerated. All samples were assessed using the Active Cortisol EIA Kit, obtained from DSLabs in Arlington, Texas, with a standard assay procedure. Absorbance was read on a plate reader with a 450 nm absorbance filter. Each sample and standard was tested in duplicate and results were averaged and assessed. A standard curve was then constructed and the average absorbance value for each unknown sample was determined.

**Procedure**

In an orientation session, subjects were explained the experiment, asked to sign a consent form (Appendix C), and familiarized with the heat probe. Following the orientation, each session was set up in the same way. Subjects first participated in a brief amount of exercise, were then tested for pain threshold, they filled out body awareness questionnaires (and in the game and practice sessions they provided importance and intensity ratings), they were tested for blood pressure and heart rate, and finally were asked to chew on a piece of gauze so that we could obtain a saliva sample for cortisol assays.

**Results**

**Experiment 1**

*Physiological Measures*

A mixed factorial ANOVA was calculated to study the effects of day (baseline, practice, game) on the systolic blood pressure of male and female soccer players. A
significant main effect of day was found such that systolic blood pressure increased from baseline to practice to game (F(2,34)=4.55, p=.018). Post hoc analysis using Fisher’s LSD showed that systolic blood pressure on game day was significantly higher than on practice day (p=.02) and baseline day (p=.008), which were not significantly different from one another (Table 1, Figure 2). However, there was no main effect of sex on systolic blood pressure and there was no significant day by sex interaction.

A mixed factorial ANOVA was calculated to measure the effects of day (baseline, practice, game) on the diastolic blood pressure of male and female soccer players. A significant main effect of day was found such that diastolic blood pressure increased from baseline to practice to game day (F(2,34)=33.43, p<.001). Post hoc analysis using Fisher’s LSD showed that diastolic blood pressure on game day was significantly higher than on practice day (p<.001) and baseline day (p<.001), and diastolic blood pressure on practice day was significantly higher than on baseline day (p=.04). Diastolic blood pressure was highest on game day and lowest on baseline day (Table 1, Figure 2). There was no main effect of sex on diastolic blood pressure. However, there was a significant day by sex interaction (F(2,34)=3.58, p=.039) such that diastolic blood pressure in males on practice day was significantly greater than on baseline day (p=.002) whereas diastolic blood pressure was not significantly different for females between practice and baseline days.

A mixed factorial ANOVA was calculated to measure the effects of day (baseline, practice, game) on the heart rate of male and female soccer players. A significant main effect of day was found such that heart rate increased from baseline to practice to game day, (F(2,34)=17.65, p<.001). Post hoc analysis using Fisher’s LSD showed that heart
rate on game day was significantly higher than on practice day (p=.001) and baseline day (p<.001) and that heart rate on practice day was significantly higher than on baseline day (p=.03). Heart rate was highest during game day and lowest during baseline day (Table 1, Figure 2). However, there was no main effect of sex on heart rate and there was no significant day by sex interaction.

**Skin Temperature**

A mixed factorial ANOVA was calculated to measure the effect of day (baseline, practice, game) on skin temperature in male and female soccer players. A significant main effect of day on skin temperature was found (F(2,38)=27.65, p<.001). Post hoc analysis using Fisher’s LSD suggest that skin temperature on baseline day was significantly higher than skin temperature was on practice day (p<.001) and game (p<.001) day (Table 1, Figure 4).

**Body Awareness Questionnaires**

A mixed factorial ANOVA was calculated to measure the effect of day (baseline, practice, game) on BAQ scores for male and female soccer players. A significant main effect for day was found, such that BAQ scores increased from baseline to practice to game sessions (F(2,36)=9.91, p<.001). Post hoc tests using Fisher’s LSD suggests that BAQ scores on game and practice days were significantly higher than on baseline day (p<.001, p=.02 respectively). The difference in BAQ scores between practice day and game day approached significance (p=.058). Self-rated measures of anxiety were highest
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on game day and lowest on baseline day (Table 1, Figure 5). However, no main effect of sex was found and there was no significant day by sex interaction.

**Perceived Intensity**

A mixed factorial ANOVA was calculated to measure the effect of day (practice, game) on perceived intensity in male and female soccer players. A significant main effect of day on perceived intensity was found such that perceived intensity ratings increased from practice to game sessions (F(1,14)=59.66, p<.001). Male and female soccer players rated games as more intense than practices (Table 1, Figure 6). Post hoc analysis using Fisher’s LSD showed that perceived intensity of games was higher than that of practices (p<.001).

**Cortisol**

A mixed factorial ANOVA was calculated to measure the effect of day (baseline, practice, game) on cortisol levels in both male and female soccer players. A significant main effect for day was found, such that cortisol levels increased from baseline to practice to game sessions (F(2,20)=3.94, p=.003). Post hoc tests using Fisher’s LSD suggest that cortisol levels were significantly higher on game day compared to baseline day (p=.01) and practice day (p=.01) in both male and female soccer players (Table 1, Figure 7).

**Pain Threshold**
Pain threshold was measured by taking the difference between initial skin temperature and the temperature at which the subject reported that the stimulus was painful. A mixed factorial ANOVA was calculated to measure the effect of day (baseline, practice, game) on pain threshold in both arms and fingertips in male and female soccer players. A significant effect of day was found such that pain threshold measurements in both the arm and the fingertips increased from baseline to practice to game day \((F(2,36)=20.61, p<.001; F(2,36)=21.89, p<.001\) respectively). Pain thresholds in both the fingertips and on the arm are lowest on baseline day and highest on game day (Table 1, Figures 8 & 9). Post hoc tests using Fisher’s LSD showed that arm pain threshold is significantly greater on both game day and practice day than on baseline day \((p<.001, p<.001\) respectively). Post hoc analysis on fingertip pain threshold suggests that fingertip pain threshold is significantly greater on both game day and practice day than on baseline day \((p<.001, p<.001\) respectively).

**Game Importance Rating and Pain Threshold**

A Pearson correlation coefficient was calculated to investigate the relationship between how important the subject rated the game and the pain threshold (in terms of difference between arm temperature and the average temperature at which the subject reported pain) for the subject’s arm and finger. A significant positive relationship was found between the game importance rating and the subject’s arm pain threshold \((r(12)=.593, p=.025\). The more important a subject rated the game, the greater arm pain threshold (or lower the pain sensitivity) he or she displayed (Figure 10). A positive relationship approaching significance was found between the game importance rating and
the subject’s finger pain threshold ($r(12)=.593$, $p=.077$). The more important a subject rated the game, the greater finger pain threshold he or she showed (Figure 11). The two correlations found were independent of starting arm temperature.

Discussion

Experiment 1

The results of experiment 1 indicate that multiple stress measures increase in anticipation of meaningful competition as compared with a baseline condition in male and female soccer players. Consistent with prior research, the results of the present study suggest that stressors can increase sympathetic nervous system measures (heart rate, blood pressure), cortisol levels, and pain threshold (Sternberg et al., 1998; Sternberg et al., 2001; Salvador et al., 2003). Both cognitive and physical stressors involved in competition can trigger this rise in objective stress measurements and analgesia (Faulstich et al., 1986).

In experiment 1, anticipatory stress measured via cortisol levels increased from baseline to practice to game days, with game day cortisol levels being significantly greater than cortisol concentrations on both practice and baseline days (Figure 7). This is expected, as a rise in cortisol in both males and females has been observed in response to a stressor as well as in anticipation of a stressful event (Bateup et al., 2002; Salvador et al., 2003; Kivlighan et al., 2005). Cortisol rises in anticipation of competition are typically observed up to 24 hours in advance of the contest and are due to the mental and physical demands associated with the competition. Higher pre-competition cortisol levels are associated with more mental preparation for the event and increased competitiveness (Edwards, Wetzel, & Wyner, 2006).
In the present experiment, sympathetic nervous system measures (heart rate, blood pressure) were consistent with the expected rise in stress, increasing from baseline to practice to game sessions (Figure 2). Heart rate and both systolic and diastolic blood pressures in male and female soccer players were significantly greater before a game than before a practice or during a baseline session. Additionally, heart rates of male and female soccer players and diastolic blood pressure of male soccer players were significantly greater during practice sessions than during a baseline condition. In combination, elevations in cortisol concentrations and sympathetic nervous system measures on game days suggest that, in both male and female athletes, all objective measures of stress, and therefore anxiety levels, are increased in anticipation of a meaningful athletic competition as compared with a non-competitive session.

Parallel to the rise in objective stress measures and in support of prior studies, the results of the present study suggest that athletes display a rise in pain threshold (analgesia) in response to athletic competition as compared with a non-competitive testing session (Sternberg et al., 1998; Sternberg et al., 2001). Additionally, the present study suggests that pain threshold increases in anticipation of a meaningful athletic competition (Figures 8 & 9). In male and female soccer players, pain threshold on both the arm and fingertips is significantly greater during game and practice sessions than during a baseline session.

Both sympathetic nervous system measures and pain thresholds are elevated prior to both games and practices relative to a baseline session, suggesting that both games and practices represent a meaningful competition for male and female soccer players. During games athletes are competing against another team whereas during practices athletes are
competing against their teammates for playing time. Furthermore, subjective stress measurements, evaluated by scores on self-rated body awareness questionnaires support the argument that practices are indeed personally meaningful competitions that can induce a heightened stress response. In male and female soccer players, scores on body awareness questionnaires are significantly higher on practice and game days than on baseline day (Figure 5). Therefore, both games and practices should be considered forms of meaningful competition. This was an unexpected finding that will be further investigated in experiment 2.

Together, both sympathetic nervous system measures (heart rate and blood pressure) and self-rated anxiety scores indicate that, for athletes, competitive environments (games and practices) as compared with non-competitive situations lead to an increase in anxiety levels and a subsequent rise in pain threshold (analgesia). Athletes associated a high degree of intensity with practice sessions, but they perceived the intensity of a game to be significantly greater than that of a practice session (Figure 6). This suggests that analgesia in response to meaningful competition is partially mediated by the psychological stress associated with the contest.

Additional support for this hypothesis was found in a correlation between game importance rating and arm pain threshold. Before the game, soccer players were asked to self-rate the importance of the game on a 10-point scale. One would expect that a game of greater importance is perceived by an athlete as a more stressful event than a game of less importance. A positive relationship was found between the game importance rating and arm pain threshold such that the more important an athlete rated the game, the greater
pain threshold in the arm he or she exhibited, further supporting the psychological stress due to competition was alone able to induce an analgesic state (Figure 10).

While the results of experiment 1 support our main hypotheses, there is one major issue – the confound of ambient temperature. Pain threshold increased prior to games and practices as compared with a baseline condition, but ambient temperature followed an identical pattern (Figure 4). Air temperature was cooler outside than it was in the lab, and it was therefore colder during practice and game sessions than it was during the baseline condition. This is important because the ambient temperature was directly correlated with skin temperature such that skin temperature was significantly higher during baseline sessions than during practice or game sessions. The cooler ambient temperature could have altered pain threshold measurements. To control for this confound, a second experiment was designed that would take place entirely indoors, with the expectation that skin temperature would be more consistent across testing sessions and the results would not be confounded by air temperature.

Additionally, the second experiment would only include female subjects. Prior studies have found a difference in pain threshold in males and females, but there are no consistent findings for sex differences in pain threshold following stress-induced analgesia. Because no sex differences were observed in experiment 1 and circumstances allowed easier access to female basketball players, experiment 2 uses only female athletes as subjects.

Method

Experiment 2
The purpose of experiment 2 was to control for the confound of ambient temperature that appeared in experiment 1 and to further investigate the interaction between the stress response to competition and analgesia from the perspective of a second sport. Subjects included nine female Haverford College varsity basketball players between the ages of 18 and 22. Only females participated in experiment 2 because no sex differences were found in experiment 1.

Subjects were recruited based on playing time; athletes who started in games or usually played at least 25% of the game were asked to participate in the study. The conditions and procedures were the same as those used in experiment 1 with one exception. The practice sessions took place approximately 30 minutes before the beginning of practice and the game sessions were conducted approximately 50 minutes before the start of the game. As in experiment 1, the sessions were counterbalanced to control for practice effects and the participants were reimbursed $30.00 for their time.

Results

Experiment 2

Physiological Measures

A repeated measures ANOVA was calculated to study the effect of day (baseline, practice, game) on the systolic blood pressure of female basketball players. No significant differences were found ($F(2,14)=2.61$, $p>.05$). However, there were trends indicating that systolic blood pressure on baseline day was lower than on practice ($p=.07$) day or game ($p=.06$) day (Table 2, Figure 3).

A repeated measures ANOVA was calculated to evaluate the effect of day (baseline, practice, game) on the diastolic blood pressure of female basketball players.
No significant differences were found ($F(2,14)=1.28, p>.05$). Day did not influence diastolic blood pressure in female basketball players (Table 2, Figure 3).

A repeated measures ANOVA was calculated to test the effect of day (baseline, practice, game) on the heart rate of female basketball players. A significant effect of day on heart rate was found ($F(2,14)=12.30, p=.001$). Post hoc analysis using Fisher’s LSD indicated, in female basketball players, heart rate on baseline day was significantly lower than on practice ($p=.003$) or game ($p<.001$) days. Heart rate did not differ between practice and game days (Table 2, Figure 3).

**Skin Temperature**

A repeated measures ANOVA was calculated to test the effect of day (baseline, practice, game) on the skin temperature of female basketball players. A significant effect of day on skin temperature was found ($F(2,16)=26.86, p<.001$). Post hoc analysis using Fisher’s LSD revealed that skin temperature on baseline day was significantly greater than on practice ($p<.001$) day or game ($p<.001$) day (Table 2, Figure 4).

**Body Awareness Questionnaire**

A repeated measures ANOVA was calculated to study the effect of day (baseline, practice, game) on BAQ scores in female basketball players. No significant difference was found ($F(2,16)=.98, p>.05$). The condition did not influence BAQ scores in female basketball players (Table 2, Figure 5).

**Perceived Intensity**
A repeated measures ANOVA was calculated to measure the effect of day (practice, game) on perceived intensity. A significant main effect of day on perceived intensity was found such that perceived intensity ratings increased from practice to game sessions (F(1,8)=16.67, p=.004). Female basketball players rated games as more intense than practices (Table 2, Figure 6). Post hoc analysis using Fisher’s LSD showed that perceived intensity of games was much higher than that of practices (p=.003).

Cortisol

A repeated measures ANOVA was calculated to test the effect of day (baseline, practice, game) on cortisol levels in female basketball players. No significant effect of day on cortisol levels was found (F(2,8)=2.09, p>.05). The condition did not influence cortisol levels in female basketball players (Table 2, Figure 7).

Pain Threshold

Pain threshold was evaluated by taking the difference between initial body temperature and the temperature at which the subject first reported that the stimulus was painful. A repeated measures ANOVA was calculated to measure the effect of day (baseline, practice, game) on pain threshold in both the arms and fingertips of female basketball players. A significant effect of day was found such that pain threshold measurements in both the arms and in the fingertips increased from baseline to practice to game day (F(2,16)=12.17, p<.001 and F(2,16)=5.44, p=.016 respectively). Pain thresholds in both the arm and the fingertips are lowest on baseline day and highest on game day (Table 2, Figures 8 & 9). Post hoc tests using Fisher’s LSD showed that arm
pain threshold is significantly lower in baseline day than on both practice day (p=.001) and game day (p<.001). Post hoc analysis on fingertip pain threshold suggests that fingertip pain threshold is significantly lower on baseline day than on both practice day (p=.012) and game day (p=.011).

Discussion

Experiment 2

Experiment 2 was designed to control for the confound of ambient temperature present in experiment 1 and to investigate the relationship between stress and pain threshold from the perspective of a second sport. Experiment 1 revealed no sex differences with respect to pain threshold and levels of anxiety. Therefore, only female basketball players were tested in experiment 2 because we had greater access to them than to the men’s basketball team.

The results from experiment 2 indicate that both objective and subjective levels of anxiety are not consistently elevated prior to a meaningful competition in female basketball players. Although the heart rates of female basketball players were higher before games and practices than they were during a non-competitive session, blood pressure was not (Figure 3). Both systolic and diastolic blood pressures showed no significant differences between each of the conditions. Systolic blood pressure, however, did show a trend towards an increase from baseline to practice to game day. Additionally, self-rated levels of anxiety were also inconsistent. Scores on body awareness questionnaires did not differ between the conditions; however, as in experiment 1, basketball players in experiment 2 perceived the intensity of a game to be greater than that of a practice (Figure 5, Figure 6 respectively). Furthermore, salivary
cortisol levels failed to show differences between the conditions (Figure 7). The inability to detect significant differences in cortisol levels between the conditions was most likely due to technical problems encountered while running the cortisol assay on the basketball samples.

Despite the differences between the first and second experiments in levels of anxiety, changes in pain threshold (analgesia) in response to each condition were consistent between the two experiments. As in athletes in experiment 1, pain thresholds of both the arms and fingertips in athletes in experiment 2 were significantly lower during the baseline session than during the practice and game sessions. Unfortunately, subjects in experiment 2 did not provide consistent ambient temperatures during each condition. Similar to experiment 1, the skin temperatures of subjects in experiment 2 were significantly higher in the laboratory during the baseline session than they were in the basketball arena during the practice and game sessions (Figure 4). Here exists a potential confound because the rise in skin temperature parallels the increase in pain threshold (analgesia) observed during the game and practice sessions as compared with the baseline condition.

It was difficult to draw conclusions from the experiment 2 data for a number of reasons. The cortisol concentrations were inaccurate because of problems encountered while running the assay. Additionally, limited access to the basketball players resulted in a low subject number and a testing time far in advance of the actual practice or game. Finally, as in experiment 1, the skin temperatures of athletes in experiment 2 were lower in the practice and game conditions than during the baseline session. This could serve as
a potential confound because the depression in skin temperatures during the practice and
game sessions is accompanied by a rise in pain threshold.

General

The purpose of this experiment was three-fold: (1) To assess anticipatory stress in
male and female athletes through cortisol levels, sympathetic nervous system measures
(heart rate and blood pressure), and pain threshold due to athletic competition as
compared with a practice and a baseline session; (2) To determine if the observed
increases in stress (measured by cortisol levels) in anticipation of athletic competition
will also be detected in sympathetic nervous system measures and pain threshold; and (3)
To determine if the psychological stress due to competition is alone able to induce an
analgesic state. We hypothesized that meaningful competition would create a more
psychologically stressful environment for an athlete than would a practice or a non-
competitive session. We therefore expected that sympathetic nervous system measures
(heart rate, blood pressure), cortisol levels, and pain threshold would all increase in male
and female soccer players and female basketball players in anticipation of a game relative
to a practice or a baseline session.

The present research supports previous findings that indicate that pain threshold
(analgesia) is increased in response to athletic competition (Sternberg et al., 1998;
Sternberg et al., 2001). Additionally, the present study had novel findings; in particular,
pain threshold (analgesia) increased in anticipation of a meaningful athletic competition
in both male and female soccer players and female basketball players (Figures 8 & 9). The present study also expanded upon past findings because it eliminated exercise as a
potential confound. In every condition, each subject participated in an equivalent and brief amount of physical activity so that the analgesic effects of exercise for each participant were consistent throughout the study. This allowed us to directly investigate the effect of the psychological stress due to competition on pain threshold in athletes. Furthermore, the severity of exercise during each session (team warm-up and stationary biking) was minimal and is not likely to have altered the pain thresholds of athletes. Exercise-induced analgesia is only produced in humans following a high level of exercise with a workload of at least 74% aerobic capacity (Partocaara et al., 1984). Each testing session was not aerobically challenging for the participants, and therefore was unlikely to lead to significant changes in pain threshold due to exercise.

Although the results of experiments 1 and 2 display similarities in pain threshold changes, objective measures of anticipatory stress in the experiments generated inconsistent results. Male and female soccer players displayed a constant rise in stress measures from baseline to practice to game sessions in both sympathetic nervous system measures (heart rate, blood pressure) and salivary cortisol levels. Women basketball players, however, did not demonstrate the same pattern in anticipatory objective stress measures. Neither sympathetic nervous system measures (with the exception of baseline heart rates being significantly lower than game and practice heart rates) nor cortisol levels in female basketball players were significantly different between the testing conditions.

Subjective levels of anxiety also differed between soccer and basketball players. While male and female soccer players displayed an increase in self-rated anxiety during practice and game sessions as compared with a baseline session, female basketball
players did not exhibit a pattern (Figure 5). Perhaps the best explanation for this is through the physiological nature of subjective arousal: increases in heart rate and blood pressure are typically accompanied by increases in subjective arousal (Rowland, Kaariainen, & Houtsmuller, 2000; Coventry & Hudson, 2001). Without the change in heart rate and blood pressure, there may be no basis for a change in subjective arousal for female basketball players. Therefore, changes in self-rated anxiety during meaningful competition may not exist in female basketball players because of the lack of sympathetic nervous system differences across conditions. Similar differences were observed between soccer and basketball players in a correlation between game importance and pain threshold. In male and female soccer players, but not basketball players, the more important a game was rated, the greater pain threshold (analgesia) that person exhibited (Figure 10). Although differences were present between the groups in self-rated anxiety levels and the correlation between game importance and pain threshold, both soccer and basketball players behaved in a similar fashion with respect to perceived intensity ratings. Each group of athletes perceived games to be significantly more intense than practices (Figure 6).

Despite differences in multiple measures of anxiety, in both groups of athletes, “meaningful competition” appeared to signify not only a contest against another team but also a practice session during which teammates were competing against each other for playing time. In both soccer and basketball players, pain threshold increases were observed in anticipation of both games and practices as compared with a baseline session, suggesting that each type of competition was meaningful and sufficient to induce an analgesic response. The results of experiment 2 further support this claim; in male and
female soccer players, both self-rated anxiety scores and sympathetic nervous system measures were elevated during practice and game sessions as compared with a non-competitive session.

However, it is unclear why cortisol levels of male and female soccer players did not exhibit the same elevation pattern (concentrations during both practice and game sessions being significantly greater than a baseline session) as the other sympathetic nervous system measures of anxiety. Increases in cortisol are associated with greater mental preparation (Kivlighan et al., 2005). Mental preparation for a game begins at least a day in advance when athletes begin to think about the competition and what they need to do to win. However, practices are more routine than games, occurring daily rather than once or twice a week. Practices therefore require much less mental preparation than games, and most athletes do not begin to think about the practice session until it has begun. Differences in mental preparation between practice and game sessions could also contribute to the differences in perceived intensity between game and baseline sessions observed in both groups of athletes. Both male and female soccer players and female basketball players perceived the intensity of a game to be greater than the intensity of a practice session (Figure 6). It is therefore likely that the difference in cortisol concentrations between practice and game sessions could be due to the different mental preparation associated with games and practices.

The inability for basketball players to display the same pattern of sympathetic nervous system activation, cortisol secretion, and subjective stress measures could be due to methodological issues. There are a number of possible explanations that could account for the differences in results between soccer and basketball players. Significantly fewer
basketball players (9) participated in the study than did soccer players (20); the low subject number could contribute to the inability to find significant differences in subjective and objective stress measures between each experimental condition. This was unavoidable because fewer athletes play in a basketball game than in a soccer game; only five players are on the court at a time in a basketball game whereas eleven players are on the field at a time during a soccer game. This makes it difficult to have a large group of subjects who are basketball players.

The disparity in the timing of testing between experiments 1 and 2 also may have contributed to a differential stress response between soccer and basketball players. Due to subject access, soccer players were tested 10-15 minutes before the game and directly following a practice warm up. Basketball players, however, were tested almost one hour before the game and nearly thirty minutes before the practice. Testing, conducted too far in advance of the event, may not yield stress reactions as prominently as testing performed directly before the contest.

Non-methodological issues could also account for the differences in stress response between soccer and basketball players. Soccer is a “player’s game.” During a game, athletes are expected to make their own decisions concerning what to do in every situation. The coach has limited control over the outcome of the game – he or she cannot call a timeout or a play. Even substitutions are limited so that a coach cannot take a player out of the game to explain something and immediately send him or her back into the match. In basketball, however, coaches are part of the team. They control what plays are run, call timeouts to change the game plan or to stop the opposition’s momentum, and are constantly instructing players on what to do (Hargreaves, 1990). Perhaps the nature
of soccer creates a situation in which players feel more pressure than basketball players – soccer players are expected to make decisions on their own throughout a game; they cannot rely on coaches’ decisions as much as basketball players can. This theory is supported by past research that has suggested that performance pressure leads to an increase in both subjective arousal and cortisol levels (Scholtz, Schulz, Hellhammer, Stone, & Hellhammer, 2006).

Other differences in stress reactions may arise as a result of winning expectancies and social norms. Sympathetic nervous system responses (blood pressure, heart rate) of both male and female soccer players demonstrate an increase from baseline to practice to game; those of female basketball players, however, do not exhibit the same significant differences (Figure 2, Figure 3). Research has suggested that individuals who expect to win have a greater increase in heart rate and blood pressure in anticipation of the event than those who do not expect to win. More specifically, the cognitive factor of winning expectancy rather than the actual playing of the game elicits a stress response to competition (Smith, Allred, Morrison, & Carlson, 1989; Ladouceur, Sevigny, Blaszczynski, O’Connor, & Lavoie, 2003; Wulfert, Roland, Hartley, Wang, & Franco, 2005).

Furthermore, social context can also influence sympathetic nervous system activation. In particular, losing is especially stressful for men because contemporary society expects men to succeed. Winning, however, is particularly stressful for females because they often fear success (Holt-Lunstad, Clayton, & Uchino, 2001). The record of the men’s soccer team that participated in the present study was five wins, eleven losses, and one tie. The record for the women’s soccer team, on the other hand, was fourteen
wins, five losses, and one tie. Undoubtedly, there was a success difference between each program that conflicted with social norms. Therefore, it is possible that the stress reactions of the members of each soccer team were heightened due to social factors concerning winning or losing. The women’s basketball team record was six wins and nineteen losses. Lacking the expectation to win and the social stress associated with winning, it is not surprising that female basketball players exhibited less of a sympathetic nervous system response to competition.

The role of the stress response to competition and the reason for the anticipatory rise in stress measures prior to competition is largely debated. This rise has been suggested to be beneficial for an athlete for three reasons: (1) To organize resources needed for physical activity; (2) To aid memory, learning, and emotions important for performing well; and (3) To regulate other stress-sensitive systems in the body (Kivlghan, Granger, & Booth, 2005). Researchers generally agree that the activation of the stress response is advantageous to an athlete because it mobilizes the physiological resources necessary for the contest (Salvador et al., 2003). Although the role of the stress response in anticipation of competition is similar for all athletes, the present study indicates that activation of the stress response is more complex. Meaningful competition does not simply elicit the same stress response for each athlete. Research has suggested that hormones and behavior do not necessarily display a cause and effect relationship, but rather show a bidirectional association (Kivlghan et al., 2005). Meaningful competition may enhance the likelihood for increases in cortisol concentrations, sympathetic nervous system responses, and self-rated levels of anxiety, but not necessarily cause them. Under the appropriate conditions, the stress response is triggered. Winning expectancies, social
context, coaching styles, and the nature of the sport all play a role in an athlete’s stress response to meaningful competition and should therefore be considered in athletes who claim to feel no pain following an injury.

Presently, we do not know the exact set of circumstances that will trigger an anticipatory stress and analgesic response to competition. However, the present study suggests that winning expectancies, social norms and the nature of the sport may play an important role. Perhaps the environmental context of the present study was not suitable to maximally elicit a stress and analgesic response. More studies, therefore, must be conducted that evaluate situations that maximally trigger an athlete’s stress response to competition. For instance, studies could evaluate variations in the stress response due to competition between winning and losing teams perhaps by asking whether or not the individual expects to beat the opponent. It would also be valuable to take into account the performance level of an athlete (do novices or experts exhibit a greater stress and analgesic response?) and the nature of the sport (individual versus team). Further exploration of practice as a form of meaningful competition is essential so that a more complete understanding of the psychological factors that induce analgesia during competition is reached. The complexity of this study’s results opens many doors for future research in this field. More research is necessary to completely understand the mechanisms that trigger analgesia due to athletic competition.

The present study had multiple shortcomings, most notable were the differences in skin temperature in subjects between the baseline session and the game and practice sessions, the irregularities in the cortisol assay from experiment 2, and the difference in testing times between both groups of athletes. Both the irregularities in the cortisol assay
for experiment 2 and the difference in testing times between both experiments have previously been discussed. In both experiment 1 and experiment 2, skin temperature was significantly lower during the practice and game sessions than it was during the baseline session. This could contribute to the pain threshold differences noted between the sessions. Perhaps this is the result of ambient temperature: the temperature on the soccer field and in the gym was cooler than in the lab. However, this could also be in response to the stress associated with competition. During the stress response, in addition to sympathetic nervous system activation, blood is sent to organs that need more oxygen. This diverts blood flow away from the skin, reducing its temperature. The reduction in skin temperature that often accompanies the stress response is typically associated with subjective feelings of anxiety (Wofford, 2001). If reductions in skin temperature are a component of the stress response to meaningful competition, it may be very difficult to measure thermal pain thresholds in response to stress. Perhaps a different type of pain measurement should be devised to further investigate the effects of anticipatory stress in pain threshold measurements in male and female athletes. Despite these flaws, the results suggest that meaningful competition (game or practice) does induce an analgesic response in male and female athletes.

The aforementioned success of the women’s soccer program may have influenced the correlation between game importance rating and pain threshold that was observed in soccer but not basketball players (Figure 10). When a team is consistently winning and has a chance to make conference and NCAA playoffs, every game is important. However, games may not be perceived of as important if a team is frequently losing and does not have a chance to make playoffs. The game importance rating is also a
subjective measure; subjects are rating the importance of a game on a relative rather than an absolute basis. Therefore, it is possible that a ceiling effect occurred in soccer players’ game importance ratings, as each player rated the importance of a game between a 7 and a 10 on a 10-point scale, and over half of the subjects rated the game importance as a 10.

Despite limitations, the results of the present study have important implications for the self-report of pain for injuries that occur not only during a competition, but also during a team practice. When faced with an injury during a game or a practice, athletes may not accurately assess its severity. Therefore, athletic trainers and coaches must objectively evaluate the situation and its environmental context so that the athlete does not further injure himself.
References


Figure 1: Cortisol levels before and after competition, a practice and a baseline session in four female soccer players. Cortisol concentrations were the same before and after a practice and during two sessions on a baseline day. Cortisol concentrations, however, were elevated prior to a competition as compared with a practice or baseline session and were greatly increased following a competition.
**Table 1**: Physiological and Pain Threshold Measurements Separated by Day (Experiment 1).

<table>
<thead>
<tr>
<th></th>
<th>Baseline Mean ± SEM</th>
<th>Practice Mean ± SEM</th>
<th>Game Mean ± SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Systolic Blood Pressure (mmHg)</strong></td>
<td>135.9 ± 5.20</td>
<td>139.7 ± 5.6</td>
<td>156.3 ± 3.98</td>
</tr>
<tr>
<td><strong>Diastolic Blood Pressure (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>74.6 ± 3.86</td>
<td>89.25 ± 4.2</td>
<td>102.5 ± 3.30</td>
</tr>
<tr>
<td>Female</td>
<td>83.0 ± 3.29</td>
<td>82.5 ± 3.58</td>
<td>100.9 ± 2.85</td>
</tr>
<tr>
<td><strong>Heart Rate (bpm)</strong></td>
<td>68.3 ± 2.46</td>
<td>76.4 ± 2.9</td>
<td>87.4 ± 3.45</td>
</tr>
<tr>
<td><strong>Body Awareness Questionnaires</strong></td>
<td>33.3 ± .988</td>
<td>36.3 ± 1.6</td>
<td>38.45 ± 1.13</td>
</tr>
<tr>
<td><strong>Skin Temperature (°C)</strong></td>
<td>31.4 ± .221</td>
<td>26.3 ± .898</td>
<td>26.0 ± .831</td>
</tr>
<tr>
<td><strong>Pain Threshold - Arm (°C)</strong></td>
<td>11.4 ± .764</td>
<td>15.9 ± .990</td>
<td>16.2 ± 1.20</td>
</tr>
<tr>
<td><strong>Pain Threshold – Fingertips (°C)</strong></td>
<td>14.4 ± .911</td>
<td>18.85 ± 1.12</td>
<td>19.6 ± 1.28</td>
</tr>
<tr>
<td><strong>Cortisol (µg/dL)</strong></td>
<td>.269 ± .076</td>
<td>.270 ± .065</td>
<td>.467 ± .097</td>
</tr>
</tbody>
</table>
Figure 2: Changes in heart rate, diastolic blood pressure, and systolic blood pressure from baseline to practice to game days in male and female soccer players. *Heart rate and blood pressure (systolic and diastolic) was significantly greater for male and female soccer players during game sessions than during practice or baseline sessions. Additionally, heart rates of male and female soccer players and the diastolic blood pressure of male soccer players were significantly greater during practice sessions than during baseline sessions.
**Figure 3:** Changes in heart rate, diastolic blood pressure, and systolic blood pressure from baseline to practice to game days in female basketball players. * Heart rates of female basketball players were significantly lower during baseline sessions than during practice and game sessions.
**Figure 4:** Differences in skin temperature during each condition in male and female soccer players and female basketball players. * The skin temperature of male and female soccer players and female basketball players was significantly greater during a baseline session than during a practice or game session.
Figure 5: Self-rated anxiety scores in each condition in male and female soccer players and female basketball players. * BAQ scores were significantly greater in male and female soccer players during a game session than during a practice or baseline session. Scores in male and female soccer players were also significantly higher during a practice session than during a baseline session.
Figure 6: Perceived game and practice intensity in male and female soccer players and female basketball players. * Male and female soccer players and female basketball players perceived the intensity of a game to be greater than that of a practice.
Figure 7: Cortisol concentrations over three conditions in male and female soccer players and female basketball players. * Cortisol concentrations in male and female soccer players were significantly greater during a game session than a practice or baseline session.
Figure 8: Pain threshold in the fingertips in male and females soccer players and female basketball players across three conditions. * Finger pain thresholds were significantly lower during a baseline session than a practice or a game session in both male and female soccer players and female basketball players.
Figure 9: Pain threshold in the arm in male and female soccer players and female basketball players across three conditions. * Arm pain thresholds were significantly lower during a baseline session than a practice or a game session in both male and female soccer players and female basketball players.
Figure 10: Correlation between game importance rating and pain threshold (arm) in male and female soccer players. A significant correlation was found such that the more important a soccer player rated the game, the greater his or her arm pain threshold.
Figure 11: Correlation between game importance rating and pain threshold (fingertips) in male and female soccer players. No significant correlation was found.
Table 2: Physiological and Pain Threshold Measurements Separated by Day (Experiment 2).

<table>
<thead>
<tr>
<th></th>
<th>Baseline Mean ± SEM</th>
<th>Practice Mean ± SEM</th>
<th>Game Mean ± SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic Blood Pressure (mmHg)</td>
<td>128.25 ± 7.65</td>
<td>143.5 ± 8.30</td>
<td>144.25 ± 6.40</td>
</tr>
<tr>
<td>Diastolic Blood Pressure (mmHg)</td>
<td>87.4 ± 5.08</td>
<td>95.0 ± 3.06</td>
<td>92.9 ± 3.50</td>
</tr>
<tr>
<td>Heart Rate (bpm)</td>
<td>63.1 ± 3.4</td>
<td>74.1 ± 2.1</td>
<td>77.5 ± 2.9</td>
</tr>
<tr>
<td>Body Awareness Questionnaires</td>
<td>31.7 ± 1.1</td>
<td>31.8 ± 1.3</td>
<td>33.1 ± 1.2</td>
</tr>
<tr>
<td>Skin Temperature (°C)</td>
<td>31.7 ± .42</td>
<td>28.8 ± .20</td>
<td>28.9 ± .20</td>
</tr>
<tr>
<td>Pain Threshold - Arm (°C)</td>
<td>13.0 ± .85</td>
<td>16.1 ± .73</td>
<td>16.6 ± .45</td>
</tr>
<tr>
<td>Pain Threshold – Fingertips (°C)</td>
<td>16.2 ± .67</td>
<td>18.4 ± .70</td>
<td>18.5 ± .70</td>
</tr>
<tr>
<td>Cortisol (µg/dL)</td>
<td>.493 ± .163</td>
<td>1.105 ± .251</td>
<td>.852 ± .156</td>
</tr>
</tbody>
</table>
Appendix A

Body Awareness Questionnaire

Subject #: ____________________  Time: ____________________
Date: ____________________  Day/Condition: ______________

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.

<table>
<thead>
<tr>
<th>Statement</th>
<th>1= Not at all</th>
<th>2= Sometimes</th>
<th>3= Moderately so</th>
<th>4= Very much so</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I feel tense</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2. I am aware of my breathing</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3. My fingertips feel numb or tingle</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4. I feel lightheaded and dizzy</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5. I feel calm</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6. My heart is pounding</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7. My mouth is dry</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8. I feel nervous</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9. I have a lump in my throat</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10. I feel confident</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11. My hands are shaking</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>12. I am having difficulty breathing</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>13. My head is throbbing</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>14. I am afraid</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>15. I feel weak and fatigued</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>16. I feel mentally relaxed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>17. I feel shaky inside (butterflies)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>18. My vision is blurred</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>19. I have chest discomfort or pain</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>20. I feel cold</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>21. I feel like yawning</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>22. I feel steady</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
Appendix B

RATINGS OF PERCEIVED INTENSITY SCALE

For the game or practice session you are about to participate in, indicate the degree of effort or intensity you expect to exert:

<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>

(Game Day Only):
On a scale of 1-10, (with 10 being extremely important), how important is the game you are about to participate in?
Appendix C

PARTICIPANT’S INFORMED CONSENT

Subject name: ______________________

Thank you for participating in our study. We are interested in how human pain responses are altered by participating in athletic competition. We will be testing your pain sensitivity on three separate occasions as a part of the experimental procedures. Today, we will familiarize you with the procedures and allow you to get comfortable with the testing apparatus. Then, we will test your pain sensitivity three times, one of which must be just prior to an athletic competition or practice session.

Your pain threshold to a heat stimulus will be assessed during the course of the study. We will place a square probe on your arm, which will heat from a neutral temperature until it gets hot. You will indicate when the stimulus becomes painful, then it will shut off immediately. This will be repeated for 6 trials, on your fingertips and forearm. The temperature range used in this study 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. It is important to note that exposure to these stimuli can in no way affect your performance in your event.

We will also ask you to rate your subjective perceptions of nervousness and anxiety and will have you provide us with saliva samples on each occasion for analysis of hormones. We will attach a blood pressure cuff to your wrist, which will record your heart rate and blood pressure.

You will not be identified by name in the data presentation. You will be informed of the study hypotheses 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, 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 neurological condition that might impair your ability to respond to the stimulus, and you are not currently under the care of a physician for treatment of a pain condition.

Signature: ______________________
Date: ______