Stress Induced Analgesia in Competitive Athletes

By: Emily Hurwitz

Based on an experiment by: Caryn Dolich, Emily Hurwitz, Sarah Nagle and Jeff Ratliff
Introduction

In humans the brain regulates all sensory input. The sense of pain, which involves central nervous system mechanisms that inhibit nociceptive afferent fibers, can in some cases activate analgesic states when perception of pain would be detrimental to survival. Such reduced sensitivity to noxious stimuli can be elicited by environmental events. This idea that natural or experimentally induced stress can lead to reduced pain sensitivity is termed stress induced analgesia (SIA).

According to anecdotal evidence, athletes can continue competing even when faced with severe injuries. Such evidence supports the idea that SIA can occur in humans during naturally occurring events. A limited number of studies have documented competition-induced analgesia in a controlled setting. However, existing research findings on stress induced analgesia and its physiological effects in humans are inconclusive and inconsistent. The current study was designed to measure the effects of the anticipatory stress associated with athletic competition (measured by cortisol levels) on pain thresholds.

Pain Modulation

The sensation of pain, along with touch, temperature, pressure and proprioception is considered a somatic sensation. Furthermore, the protective and subjective nature of pain distinguishes it from other sub modalities because only pain warns of potential injury. Unlike the other senses, the conditions under which a person experiences pain affects the intensity of the pain. Additionally, the subjective level of intensity varies in individuals under similar conditions (Basbaum, 2000).
The unpleasant sensation of pain is a unique perceptual process. Pain, alone among the sensory systems (vision, hearing, and smell), is both a sensory and emotional experience that is associated with actual or potential damage to tissues and other body parts. To study pain effectively a distinction must be made between the neural mechanisms of nociception and the emotional sensation of pain. Nociceptors, specialized sensory receptors located in peripheral tissues, are activated by noxious stimuli (Basbaum, 2000). Therefore, nociception is the process of sensing physiological pain, whereas pain is a mental perception.

The individualized and subjective nature of pain makes it difficult to define and treat clinically. There is no one stimulus that will prompt the same pain response in a large cross section of individuals. More so than other sensory modalities, pain is influenced by a subject's emotional state as well as environmental factors. For example, athletes often do not recognize their injuries until after they stop competing, and soldiers wounded in battle report that they do not feel pain (Basbaum, 2000). In addition, Clark and Mehl (1971) found that sex and age may contribute to attitudes associated with pain (Clark & Mehl, 1971).

Pain Measurement

Investigators have used several different stimuli to measure the perception of pain. Pain studies in humans typically employ electrical, temperature, or pressure measurements. Subjects who participated in a study by Janssen and Arntz (2001) were asked to rate subjective pain on a scale from “not painful” to “extremely painful” after electrical pain stimulation on the subject’s ankle (Janssen & Arntz, 2001). Temperature, either hot or cold, is also a useful measurement of pain. Sternberg et al. (1998) employed
the cold-pressor test in order to assess both sensory discriminative and unpleasantness features of the pain experience. The cold-pressor test assesses such measures by obtaining subjective ratings of intensity and discomfort while the subject's arm is submerged in a bucket of ice-water. The same researchers used withdrawal latency from a radiant heat source as a measure of pain (Sternberg et al., 1998). Koltyn et al. (2001) assessed pain perception while 3000g of pressure was applied to the right forefinger for two minutes. While pressure was applied with a pain stimulator ( Forgione-Barber), subjects were asked to note when the stimulus became painful (Koltyn et al., 2001).

Additional methods of measuring pain have been employed in studies with rodents. One common method involves the tail-flick test (D’Amour and Smith) that tests the latency of the spinally-mediated withdrawal reflex of the tail in response to the application of radiant heat (Akil et al., 1976). An alternative method involves applying shocks to the tail through disk electrodes. The rodent’s response to pain was measured by quantifying squeaking, lurching, and escape attempts (Mayer et al., 1971). Such methods are limited to animal studies since tail shocks cause tissue damage.

History

While scientists have studied and attempted to understand pain since the earliest recorded history, the systematic study of pain is fairly new. The treatment of pain was mentioned on a piece of Egyptian papyrus dating from 4000 BC. Acupuncture therapy, a traditional Chinese treatment that is still employed for pain reduction, originated in ancient China almost 2,000 years ago.

One of the earliest theories of pain was conceptualized by René Descartes, a French philosopher, using a medical mechanistic approach. He theorized that pain is an
activity in the sensory nervous system that travels from the skin directly to the brain by means of a “straight-through” channel. Descartes proposed that, “a flame applied to the foot sets particles in the foot into activity, and that motion is transmitted up the leg and back into the head” (Gatchel & Turk, 1999). Afterwards the person feels pain and behaves accordingly.

In 1894, Von Frey proposed a more formal model of pain called the *specificity theory of pain*. This theory hypothesized that specific sensory receptors were responsible for the conduction of sensations including pain, warmth, touch, and pressure. Differences in the structure of the receptors accounted for the presence or lack of sensitivity to specific stimulation. At that time scientists believed that pain had central as well as peripheral mechanisms (Gatchel & Turk, 1999).

Goldschneider proposed the *pattern theory of pain*, an alternative explanation to Von Frey’s specificity theory. Goldschneider explained that nerve impulse patterns are first produced and coded at the peripheral site of stimulation and then transmitted to the central nervous system, eventually resulting in sensations of pain. The quality and type of sensation depended on the patterning and quantity of the discharge from the peripheral nerve fiber. Goldschneider explained that a minimal tactile stimulus to an area could create the sensation of touch, whereas a stronger pattern of tactile stimulation could prompt pain (Gatchel & Turk, 2000).

While support exists for both of these theories, there are actions and behaviors that cannot be explained by Goldschneider or Von Frey. Both theories fail to mention the significance of psychological factors in the process of pain perception. As researchers adopted biopsychosocial approaches in medicine, pain researchers used a similar
comprehensive methodology in their studies. Preliminary work by Beecher (1956) demonstrated that the psychological status of a person can affect his or her response to pain. Beecher studied wounded soldiers returning from battle during WWII. Beecher compared the soldiers’ requests for medicine and their perceived pain to civilians who had similar wounds. The contrasts are striking; only 25% of the soldiers actively requested medicine compared to an overwhelming 80% of the civilians. Beecher also noted that the soldiers reported having little to no pain or denied having pain from their wounds. Beecher suggests that the differences in the psychological and emotional states of the subjects accounts for the dissimilar pain reports (Beecher, 1956).

Since Beecher’s ground-breaking study, other research has been conducted that demonstrates the importance of the individual's psychological state. The gate control theory of pain, by Melzack and Wall (1965), was the first to successfully account for the psychological and physiological interaction in pain perception. The gate control theory explains that stimulation of the skin evokes nerve impulses that are then transmitted to three spinal cord systems. Inputs from ascending nonnociceptive afferents and descending nociceptive afferents to the spinal cord can alter the upward flow of pain information to the brain (Melzack & Wall, 1965). In other words, nonnociceptive afferents “close” and nociceptive afferents “open” a gate to the central transmission of noxious input. This theory introduced the psychological nature of downward central nervous system modulation and the existence of different types of pain (Fordyce & Steger, 1979). While the original gate control theory of pain signaled a major advance in pain research, the theory has since forth been reformulated to integrate neurophysiology, neurotransmission, and endogenous opioids.
Nociception

Von Frey’s theory still does not account for the psychological nature of pain, but its main finding concerning modulation at the spinal cord and the presence and process of nociception is currently believed to be correct. This complex process is initiated by primary afferent nociceptors in the peripheral nerve that respond specifically to stimuli that produce pain in humans. There are three known types of nociceptors, thermal, mechanical, and polymodal; each one contributes in a distinct way to the quality of the intensity of pain: (Fields, 1987).

Since the skin is one of the most densely innervated surfaces in the body researchers can easily apply noxious stimuli to the skin to effectively study the three classes of nociceptors. Cutaneous nerves, located in the skin, are easily identified and can be isolated for electrophysiological study. By studying cutaneous nerves researchers have determined that afferent nociceptors exist (Fields, 1987).

Thermal nociceptors respond to extreme temperatures (> 45°C or < 5°C). They are thinly myelinated Aδ fibers with small-diameters that conduct signals at about 5-30 m/s. Mechanical nociceptors are activated by strong pressure applied to the skin. They are also thinly myelinated Aδ fibers that conduct at the same rate as thermal nociceptors (5-30 m/s). Polymodal nociceptors are activated by high-intensity mechanical, chemical, or thermal (extreme hot or cold) stimuli. Polymodal nociceptors also have small diameter, nonmyelinated C fibers that conduct slowly (less than 1.0 m/s) (Basbaum, 2000).

The three classes of nociceptors are well distributed in the skin and deep tissues and frequently work together. For example, when a person stubs his toe, a sharp pain is felt right away, followed by a feeling of aching and perhaps burning. The immediate
pain, the “first pain”, is transmitted by Aδ fibers that transmit information from thermal and mechanical nociceptors. The slow dull pain, the “second pain”, activated by the polymodal nociceptors, is transmitted by C fibers (Basbaum, 2000).

These observations on pain transmission are supported by psychophysical studies in which researchers, selectively block the myelinated fibers while leaving the C fibers intact. In these studies the first pain disappears and the second pain is more intense and prolonged. The most logical way to explain this effect is that the myelinated nociceptor afferents both excite and inhibit nociceptive cells (projection cells) located in the spinal cord. Input from the myelinated nociceptors arrives at the spinal cord first and either excites the projection cell causing first pain or activates an inhibitory interneuron. Inhibition will suppress the pain response until the projection cell receives input from the slower, unmyelinated nociceptors (Fields, 1987).

Pathways from the Dorsal Horn to the Brain

Nociceptive afferent fibers usually terminate in the dorsal horn of the spinal cord, with each distinct class ending on one of the six distinct layers, or laminae, of the dorsal horn. Each class of primary afferent neurons can only be activated by specific stimuli. The anatomical organization of neurons in the dorsal horn corresponds with the neurons’ function (Basbaum, 2000).

Nociceptive neurons are located in lamina 1, as well as in the substantia gelatinosa (lamina II). Most of these neurons receive input from Aδ and C fibers. Neurons in lamina I only respond to noxious stimulation and are therefore labeled nociceptive-specific neurons and project to higher brain centers. Lamina II, however, is
made up of interneurons that respond to both noxious and nonnoxious stimuli (Basbaum, 2000).

Laminae III and IV contain monosynaptic neurons that have small, topographically organized receptive fields and respond mostly to nonnoxious stimuli. Lamina V contains neurons that connect to the brain stem and to parts of the thalamus. The neurons receive input from Aβ, Aδ, and C fibers in addition to nociceptive input from visceral structures. Lamina VI neurons receive input from afferent fibers, from muscles, and joints but do not contribute to the communication of nociceptive messages (Basbaum, 2000).

After the nociceptive information is sent from the periphery to the dorsal horn, it is then transmitted to the thalamus and the cerebral cortex along five ascending pathways: the spinothalamic, spinoreticular, spinomesencephalic, cervicothalamic, and spinothalamic tracts.

The spinothalamic tract, the most prominent of all the ascending nociceptive pathways, is comprised of axons that project from laminae I and V-VII to the contralateral side of the spinal cord and terminate in the thalamus. While there are several nuclei in the thalamus that process nociceptive information, two are especially important: the lateral and medial nuclear groups. The lateral nuclear group is made up of the ventroposterior medial nucleus, the ventroposterior medial and lateral nuclei, and the posterior nucleus. Neurons in the nuclei, as well as the spinal neurons that project to them, have small receptive fields. A small receptive field corresponds with how accurately the brain can localize an injury. The lateral thalamus is therefore thought to be involved with mediating information about the location of an injury (Basbaum, 2000).
The medial nuclear group in the thalamus includes the central lateral nucleus and the intralaminar complex. Neurons in the medial thalamus typically respond to noxious stimuli but also project to the basal ganglia and various cortical regions. In addition to processing nociceptive information, the nuclei respond to stimuli that activate an arousal system (Basbaum, 2000).

The spinoreticular tract, unlike the spinothalamic tract, does not cross the midline and terminates in the reticular formation and the thalamus. The spinomesencephalic tract’s major destination is the amygdala, a major structure in the limbic system involved with emotion. Therefore, researchers believe that the spinomesencephalic tract adds to the emotional component of pain. The cervicothalamic tract and the spinohypothalamic tract receive input from laminae III and IV and I, V, and VIII respectively. The cervicothalamic tract ends in the medulla while the spinohypothalamic tract projects to the supraspinal autonomic control centers and activates neuroendocrine and cardiovascular responses to noxious stimuli (Basbaum, 2000).

The ascending pathways reach the brain, but questions remain as to how these specific structures contribute to the perception of pain. Findings from positron emission tomography (PET) studies suggest that pain is generated through the interplay and communication between cortical and subcortical structures; there is not just one central mechanism that acts as a master switch for the production of pain. Areas including but not limited to the anterior and posterior cingulate cortex, the periventricular and paeriqueductal gray areas in the midbrain, the thalamus and the cerebral cortex uniquely contribute to the perception, and modulation of pain (Tolleet al., 1999).
Melzack and Wall, the creators of the gate control theory, were the first to suggest that the activation of descending pain pathways results in pain inhibition. Strong evidence supports the idea that electrical stimulation of the brain produces analgesia that creates these inhibitory neurons. A study by Reynolds (1969) indicated that specific (focal) application of electrical currents in and around the midbrain central grey area in rats produced analgesia. Analgesia was measured by the lack of a response to aversive stimuli while general motor functions remained intact (Reynolds, 1969).

Two years later Mayer et al. (1971) showed that stimulation in mesencephalic and diencephalic sites eliminated responsiveness to intense pain in rats. The lack of response was specific to pain stimuli; the majority of animals responded normally to other sensory stimuli including visual, auditory, and tactile. Another relationship -- the one between reward and analgesic areas-- was also studied since several analgesic areas are components of the reward system. The results suggest that while the strength and existence of the relationship between analgesia and reward varies from structure to structure, analgesia cannot be explained due to pleasant sensations that distract from unpleasant stimuli (Mayer, 1971).

In several additional experiments, stimulation of the periaqueductal gray area produces a specific analgesic (pain reduction) effect that doesn’t just numb overall sensation in the stimulated area; the region simply feels less pain (Hosobuchi et al., 1977; Hosobuchi et al., 1979).

**Pharmacological Analgesia**

Stimulation-produced analgesia shares several common features with analgesia produced by opiates such as morphine and codeine. Both mechanisms of analgesia
involve the same pathway and are effective in areas around the third ventricle, the cerebral aqueduct, and rostral portions of the fourth ventricle. In addition, a narcotic antagonist, naloxone, can partially block both stimulation and opiate-produced analgesia suggesting that a neural system exists that uses an endogenous substance similar to morphine to produce analgesia (Akil, 1976).

Evidence for the existence of endogenous opiate mechanisms of pain modulation led to the discovery of opiate receptors in the brain. Since the brain could not have evolved receptors for such a plant compound as opiates, it follows that naturally occurring opiates that bind to the receptors exist in the human body (Sapolsky, 1992).

The discovery of opiate receptors (Pert & Snyder, 1973) triggered a search for opioids. The endorphins, enkaphalins, and dynorphins were discovered (Hughes et al., 1975) and located soon after in the pituitary, brain, and various peripheral organs. Opioids caused analgesia when injected into the receptor sites and opiate receptor antagonists blocked analgesia in regions that had been previously mapped as parts of the pain pathways. Such findings provide additional support for the existence of stimulation produced analgesia. Such regions, including the relay sites in the dorsal horn, the periaqueductal gray area and the raphe complex, were shown to contain opiate receptors (Sapolsky, 1992). The discovery of the location of these compounds enabled scientists to map descending analgesia pathways.

*Stress induced analgesia (SIA)*

Descending analgesia pathways can also be activated during extreme stress and emotional arousal in a phenomenon known as stress induced analgesia (SIA). Originally, such pain reduction, or SIA, was thought to be solely psychological.
Numerous studies in the 1970s, however, led to the discovery of the neurochemical nature of SIA. Researchers focused their studies on morphine, heroin, and opium, opiates that share similar chemical structures and are analgesic.

Additional research on the endogenous nature of pain focused on the two endocrine pathways that both involve the adrenal gland and govern the stress response and perhaps subsequent analgesia. Walter Cannon and Hans Selye, two researchers who are credited with the emergence of the study of stress physiology, emphasized the nonspecificity of the stress-response; the idea that the appraisal of a stimulus as painful is dependent on an individual’s perception (Sapolsky, 1992). Cannon termed this reaction the fight or flight response because vertebrates either face a threat (‘fight’) or attempt to escape (‘flight’). Similar situations can trigger different responses depending on the individual.

The hypothalamic pituitary adrenal axis (HPA) becomes activated in response to a stressful environmental challenge. The HPA axis contains parts of the hypothalamus, pituitary gland, and adrenal cortices. The hypothalamus releases corticotrophin-releasing hormone (CRH) in response to a threat. CRH then stimulates the pituitary gland where adrenocorticotropic hormone (ACTH) is released. ACTH stimulates the adrenal that releases glucocorticoids. In humans and primates the dominant form of glucocorticoids is cortisol. Cortisol, the “stress hormone” helps restore homeostasis after stress. To summarize, during periods of stress, a chain reaction occurs in the release of hormones (first CRH then ACTH and finally glucocorticoids) (Sapolsky, 1992).

The relationship between the physiological response to stress and pain reduction is further evidenced by the co-release of ACTH and β-Endorphin. As ACTH is released,
β-Endorphin is also secreted. β-endorphin, an endogenous opiate compound, helps regulate pain perception during stress by binding to opioid receptors and producing analgesia (Sapolsky, 1992).

SIA in Animals

Typical means of inducing stress in animals that facilitate systematic study of SIA include immobilization, forced swimming, restriction, and footshock. Amir & Amit (1979), for example, demonstrated that footshock produces an analgesic response that can be antagonized by naloxone, an opioid receptor antagonist (Amir & Amit, 1979). The analgesic response to such stressors is equivalent to those caused by morphine at a dose of 5-10mg/kg however, SIA lasts for less than 30 minutes (Bodnar et al., 1978). It is important to note, though, that the type, intensity, and duration of the stressor used impacts not only the potency of its effect but also determines the neuronal mechanism used to mediate such an effect (Yamada & Nabeshima, 1995).

Evidence suggests that there are at least two types of SIA: opioid-mediated and non-opioid mediated analgesia. In a study by Lewis and colleagues (1980) it was shown that long-term (20 minute) electric footshock produces naloxone sensitive analgesia while a short-term (3 min) footshock produces analgesia that is insensitive to naloxone (Lewis et al., 1980). It was therefore concluded that the number or duration of shocks, not the stressor type, determines whether the stressor produces opioid or non-opioid analgesia. A follow up study by Grau et al. (1981) examined what happens when rats are exposed to inescapable, continuous shocks, as opposed to the intermittent shocks employed in the Lewis study. The continuous shocks, similar to the intermittent shocks, cause naloxone insensitive analgesia at first followed by a naloxone reversible analgesic response. This
suggests that the form of SIA is determined by the number of shocks and duration of exposure, not the type of stressor used (Grau et al., 1981). With all of the available evidence Terman et al. proposed a general rule that opioid-mediated pain-inhibitory systems are triggered when a stressor is short or weak, while non-opioid analgesia systems are recruited when the stressor is longer or more intense (Terman et al., 1984).

**SIA in humans**

Compelling results from animal studies have led to the controlled study of stress induced analgesia in humans. In humans, anecdotal evidence suggests that stress results from natural occurring events. Such evidence has led to the study of exercise as a stressor. Dancers and athletes have reported instances where they have continued a strenuous activity while enduring a serious injury, and afterwards report feeling no pain. Such evidence points towards the notion that strenuous physical activity can alter an individual's perception of pain (Koltyn, 2000).

Inescapable stress has been shown to induce analgesic effects. A study by Janssen and Arntz (2001) investigated whether SIA could be demonstrated in humans performing a parachute jump for the first time. The results are consistent with animal studies. After a parachute jump, subjects who received the opioid antagonist naloxone reported higher pain sensitivity than subjects who received a placebo. Results therefore provided evidence that stress alters pain perception, and that the stress response is reversible by naloxone. This result provides further evidence for the existence of opioid-mediated analgesia in humans (Janssen & Arntz, 2001).

The most common hypothesis in the study of exercise induced analgesia suggests that exercise activates the endogenous opioid system that causes the observed analgesic
response as supported by the Janssen study previously mentioned (Koltyn, 2000).

Running and cycling, have been used as models of exercise in humans. The effect of exercise on the pain response is tested with noxious a stimulus (electrical, temperature, or pressure) that is applied before and after the exercise activity.

Several studies of pain perception following exercise have used noxious dental pulp stimulation techniques. Pertovaara et al. studied changes in dental pain threshold following cycling exercise of varying intensities. Cycling levels were increased in a stepwise fashion from 50-200W. Dental pain thresholds tended to increase with harder workloads (Pertovaara as cited in Koltyn, 2000). In a study by Droste et al. (1991), dental pulp and finger pain thresholds were measured in addition to plasma hormone levels (β-endorphin, cortisol, and catecholamines) before, during and following cycle ergometer exercise to exhaustion. The hormones were found to be significantly elevated during exercise in both conditions; participants were either given naloxone or a placebo prior to the activity. In studies conducted by Droste et al. (1991) and Pertovaara (1984) pain threshold measures were significantly elevated at maximum exercise (exercise until exhaustion); exercise produced analgesic effects. The results also indicate that naloxone had no effect on the analgesic response (Droste, 1991). This finding is counter to the hypothesis that exercise induced analgesia occurs due to the activation of the endogenous opioid response.

A number of researchers have observed analgesic responses to a variety of noxious stimuli following exercise (Kemppainen et al., 1986; Kemppainen et al.1985; Koltyn et al., 1996, as cited by Koltyn, 2000). The results for exercise-induced analgesia appear to be more consistent for studies that used electrical or pressure stimuli to produce
pain compared to temperature stimulation. Additionally, analgesia following exercise seems to be the most reliable when exercise is performed at intensities greater than 70% of maximal aerobic capacity (Perovaara & Kemppainen, 1992 as cited by Koltyn, 2000). Human exercise intensity results are consistent with the dichotomy observed in the animal literature between opioid and non-opioid mediated analgesia. Non-opioid–mediated pain inhibitory systems are triggered when the stressor is longer or more intense (Terman et al., 1984).

Although there appears to be plenty of evidence in support of exercise induced analgesia, Padawar and Levine (1992) argue that the observation of analgesia after exercise is confounded by repeated testing effects. Studies of exercise induced analgesia usually start with the administration of a measurement of pain prior to exercise, and end with a measurement of pain after exercise. Padawar and Levine believe that the analgesia is observed because of the pre and post test paradigm; previous exposure to a painful stimulus results in reduced sensitivity and leads to higher pain thresholds following exercise (Padawar and Levine, 1992).

Padawar and Levine controlled for the reduced sensitivity confound by using pre and post tests for some participants, but only using the post-test for the other participants. Experimenters used the cold pressor stimulus to measure pain threshold and whether it varies in response to exercise. The only significant analgesic results were found for the pre-exposure group, suggesting that the exercise induced analgesia effect found in previous studies is an artifact of design (Padawar and Levine, 1992).

Koltyn et al. (2001) studied the effect of isometric exercise (resistance exercise) on pain perception and blood pressure in men and women. Participants rated their pain
perception after being exposed to 3000g of pressure on their right forefinger before and after an isometric hand-grip exercise. Researchers found interesting gender differences; women had lower pain thresholds, systolic, and diastolic blood pressure before exercise while analgesia after exercise was observed more consistently in women than in men. Women had increased pain thresholds and lower pain ratings after maximal and submaximal isometric exercise while men only experienced lower pain ratings after maximal isometric exercise. The cause for gender differences in pain perception may be due to differences in blood pressure, an interaction may exist between pain regulatory mechanisms and control for blood pressure (Koltyn et al., 2001).

Varied experimental conditions could contribute to the inconsistent findings on the effect of exercise on pain perception. Experimental paradigms have typically used cycling, weight lifting, and running as the exercise measure. Pertovaara et al. (1984) displayed only a weak response of the hypothalamic-pituitary adrenocortical (HPA) axis after exercise in an experimental setting (Pertovaara et al., 1984).

These experimental activities are equivalent to daily practice sessions and fail to serve as ‘stressors’ (Janal et al., 1984). Researchers attempted to investigate whether competing in an athletic event, as opposed to participating in noncompetitive exercise, results in a lessened sensitivity to noxious stimuli. Sternberg et al. (1998) suggest that there must be a cognitive evaluation of the situation as stressful in order to stimulate the SIA mechanism. Sternberg and colleagues chose to systematically test pain threshold in fencers, track runners, and basketball players. They employed two different measures of pain threshold: the cold-pressor test, in addition to a heat probe test in which withdrawal latencies from a radiant heat source were obtained bilaterally on fingertips and forearms.
Pain threshold and ratings of noxious cold were obtained two days before, during and two days after competition. Athletes’ pain measurements were compared to twenty non-athlete controls.

The results suggest that competition can modulate behavioral responses to noxious stimuli depending on the pain test used, the body region tested, and the sport in question. All athletes reported pain from the cold-pressor test to be less intense and less unpleasant following competition as compared to baseline measurements. Pain perception for the heat source varied depending on the body region tested: withdrawal latencies increased when tested on the forearm of basketball players and track athletes whereas the same athletes exhibited pain enhancement when latencies were measured on fingertips. This difference between body parts was hypothesized to result from the presence of hypervigilance located where dense sympathetic innervation takes place—i.e. the fingertips (Sternberg et al., 1998).

Overall, the results show that athletic competition can produce SIA. While such findings provide a better understanding of exercise and competition induced analgesia, the researchers were unable to tease apart the relationship of stress appraisal during competition and the analgesic effects of exercise. The lack of agreement about the existence of exercise-induced analgesia supports the hypothesis that the stress component of competition contributes to the pain inhibition associated with physical exertion. It is unclear, however, whether the stressful psychological state that is produced during competition is sufficient to produce analgesia in the absence of exercise (Sternberg et al., 1998).
A subsequent study in the same laboratory sought to dissociate competition-specific effects from exercise-only effects. Sternberg et al. (2001) examined the analgesic effect of exercise-related stress and the stressful cognitive components of competing, independent of exercise. They did so by comparing subjects’ pain ratings of a cold pressor test after playing a competitive video game (competition condition), after a track meet (exercise + competition), or after running on the treadmill for ten minutes (exercise condition) (Sternberg et al., 2001).

The findings supported previous results of athletic competition-induced analgesia; cold pressor pain ratings were significantly lower following running in a competitive track meet compared to subjects’ own baseline values. Gender differences were also observed in the pain inhibition response to the laboratory manipulations. Treadmill running reduced cold pressor pain ratings in women, but not men, whereas the sedentary video game task induced an analgesic state in men only. The lack of analgesia in women following the video game competition, does not necessarily suggest that women are noncompetitive. Researchers hypothesize that females and males must appraise an activity as being stressful in order to elicit analgesia. Perhaps the video game competition did not adequately activate analgesia pathways in women, as it did for men, because the experience of video game competition was possibly not rewarding or engaging and therefore may have failed to evoke a personal investment into the game. Such sex differences could be due to a variation in the reason why men and women believe athletic competition is stressful (and therefore produces analgesia) (Sternberg et al., 2001).

Several studies indicate that hormone levels fluctuate during competitive athletic encounters (Suay et al., 1999). Studies indicate that cortisol, the stress hormone and the
dominant form of glucocorticoids in humans, may have an impact on behaviors such as aggression, arousal, and mobilization of physiological resources, typical of athletes in competition (Booth et al., 1989). A study of a collegiate women’s rugby team revealed an increase of cortisol levels 20 minutes prior to the matches compared to measurements obtained 24 hours before and immediately after league matches. Post game hormone levels were even higher than pre game measurements (Bateup et al., 2002). Elevated levels of cortisol suggest the athlete has both an “enhanced energy availability…and a psychological state characterized by high motivation to win and self-confidence” (Salvador et al., 2003).

Do cortisol levels increase before athletic competition because of anticipation of competition or from anticipation of physical activity? If cortisol levels rise with aggression and other behaviors typical of athletes in competition, one would expect to see elevated pre-activity cortisol levels before periods of intense competition but perhaps not prior to non-competitive, physically strenuous bouts of activity (i.e. practice sessions). A pilot study was conducted that compared cortisol levels in four female varsity intercollegiate soccer players prior to a practice, a game, and on a baseline day. While the magnitude of the changes of cortisol levels was not significant, the data does indicate a slight trend; levels were higher on competition day than on a practice day and the baseline measure (Figure 1). If athletes do experience a rise in cortisol levels before a competition, the physiological change could be beneficial since it facilitates the availability of energy before the event (Arthur, 1987 as cited by Salvador, 2003).

The previous research on the topic of stress induced analgesia and its hormonal effects in humans is inconclusive and inconsistent. It is unclear whether males and
females respond to competition and exercise-related stress in the same way and what mediates that response. The current study was designed to assess the effects of competition on anticipatory stress measured by cortisol levels and pain thresholds. Therefore, the analgesic effects in male and female soccer players and female basketball players were compared after a slight warm up before a practice, before a game, and on a baseline day (after cycling for a fixed amount of time) using heat threshold temperatures. Sympathetic measures (HR\BP), cortisol saliva samples, and a body awareness questionnaire were taken at the above-stated testing days. Subjective intensity measures, how much effort the athlete expected to exert in the upcoming game, were collected at practice and game days and the importance scale was assessed on game days.

It is hypothesized that pain thresholds will increase before meaningful competition (game) relative to a practice and baseline measure in both males and females. Cortisol and sympathetic measures will follow the same trend. Since both sexes will participate in competitive situations with similar levels of arousal and physical rigor, no sex differences are expected.

*General Methods*

*Subjects*

Two experiments were conducted. Twenty members (11 females and 9 males) of the Haverford College varsity women’s and men’s soccer team and nine female members of the Haverford College varsity basketball team (NCAA division III) were recruited from their respective teams. Subjects were given $30 for their participation, $10 for each testing day. Procedures were explained to all subjects and informed consent was obtained.
prior to experimental testing. Procedures were approved by the Haverford College Human Subjects Committee before the experiment.

**Apparatus**

Pain measures were obtained using a TSAII Medoc Thermal Sensory Analyzer. Pain thresholds in response to the heat stimulus were obtained bilaterally on fingertips (excluding the thumb and fourth finger) and the volar surface of the forearm. Experimenters placed the heat probe on six surfaces on the subjects' arms and six on their fingertips. Subjects were instructed to say “pain” when the sensation they experienced changed from hot to painful. The experimenter would immediately remove the heat probe from the subject’s body and terminate the stimulus. The probe reached its maximum temperature if/when it reached 50°C (a temperature greater than 50°C would cause tissue damage).

**Skin Temperature**

Since surface skin temperature fluctuations might influence pain threshold (Wu et al., 2001), Skin temperature was measured during all testing sessions (practice, baseline, game). Skin temperature measurements were taken before application of the heat probe along the inner surface of the arm using a noncontact infrared thermometer (Kent Scientific Model C-1600MP). Start temperature of the probe was entered prior to each testing trial.

Since skin temperature significantly differed between testing sites in experiment 1, experiment 2 was designed to help control for such differences by completing all testing indoors.
Cortisol

Saliva samples were obtained at all testing sessions. Subjects chewed on a piece of gauze until fully saturated. Experimenter extracted the saliva by squeezing the gauze into an eddendorf tube. Saliva was stored at 4°C until the assay was conducted. Enzyme immunoassay salivary cortisol kit (DSLabs, Arlington, TX) was used to determine the levels of cortisol in the participants’ saliva in response to anticipatory stress.

Body Awareness

Subjective measurements of levels of alertness and anxiety were assessed using the body awareness questionnaire. The questionnaire consists of 22 questions that are paired with forced choice responses using a 4-point version of the Likert scale, from 1 (not at all) to 4 (extremely). Sample questions include, “I feel butterflies”, “I feel calm”, “My palms are sweaty”, etc. (Appendix A).

Perceived Intensity/Importance measures

Subjective measurements of levels of intensity (from 1 being “not at all” to 13 being extremely intense) were taken before a practice and a game. On game day, subjects were also asked to, “rate on a scale of 1-10, how important is the game you are about to compete in?” (1 being not important, 10 being extremely important) (Appendix B).

Procedure

All subjects were tested on three occasions, a baseline session, a practice session, and a game session. The order of testing days was counterbalanced between subjects meaning that some subjects participated in the baseline session first, some in the practice session first and others in the game session first. Subjects were randomly assigned to one of six different possible orders of sessions. During the game and practice sessions, pain
responses and associated measures were obtained after the soccer teams’ routine warm ups but prior to competition, approximately 45 minutes prior to the start of the game. During the baseline session, we collected data after a five minute cycling session in which we instructed subjects to simulate the rigor of a typical pre-game and pre-practice warm-up. At all three sessions physiological values, skin temperature, pain threshold, and survey answers were assessed.

Data analysis: experiment 1

Final pain threshold temperatures from the subject's arms or fingers were averaged separately. Pain threshold was defined as the difference between the average pain threshold temperature and the starting skin temperature of the volar surface of the arm. This method was used since skin temperature varied significantly depending on where the session took place (outside or inside).

Day and sex effects on the various dependent variables were determined by 3 x 2 repeated measures ANOVA with day as the within-subjects factor and sex as the between-subjects factor. A fisher LSD test was run to determine which means were significantly different from one another.

All saliva samples were assayed for salivary cortisol (ug/dl) using an enzyme immunoassay kit (DS labs, Arlington, Texas). All samples were assayed in duplicate, and all samples from the same subjects were tested in the same assay run. After the assay a plate reader was used to read well absorbances at a wavelength of 450nm using a 450nm absorbance filter. A standard curve was constructed to determine the absorbance levels for each unknown. Day and sex effects on cortisol levels were determined by a 3 x 2 repeated measures ANOVA with day as the within-subjects factor and sex as the
between-subjects factor. A fisher LSD test was run to determine which means significantly differed from one another.

Bivariate Pearson correlations were calculated to investigate the relationship between various dependent variables.

Results: Experiment 1

Physiological measures

Systolic Blood Pressure

A main effect of day (baseline, game, or practice) was observed on systolic blood pressure (F[2, 34]=4.63; P=.017). Participating in a soccer game significantly raised systolic blood pressure compared to a practice (p=.008) or baseline session (p=.02) which were not different from one another (Figure 2 and Table 1). There was no sex by day interaction, thus this pattern was apparent in both males and females.

Diastolic Blood Pressure

A main effect of day was observed on diastolic blood pressure (F[2, 34]=32.837; P<0 .0001). There also existed a significant day x sex interaction (F[2,34]=3.487; P=.042) although there were no overall sex differences. In males and females, diastolic blood pressure was significantly elevated at a game as compared to practice (p<.001) and baseline sessions (p<.001). Males' diastolic blood pressure, however, was also significantly higher at practice than at baseline sessions (p=.002) (Figure 2 and Table 1).

Heart Rate

A main effect of day was observed for heart rate (F[2, 34]=18.968; P<.001). Heart rate was significantly different on all three days; it was higher at the game session than at
the practice session (p=.001) and baseline session (p<.001) and HR was significantly higher at the practice session compared to baseline (p=.03) (Figure 2 and Table 1).

**Skin Temperature**

A main effect of day was observed for initial skin temperature (F[2, 16]=26.86; P<.001). Skin temperature measured just prior to pain testing was significantly higher at baseline than at practice and game sessions (p<.001). There was no significant difference, however, between practice and game days (Figure 3).

**Body Awareness**

A main effect of day was observed for subjective stress ratings as measured on the body awareness questionnaire (F[2, 36]=9.992; P<0.0001). Subjects reported being slightly but not significantly more aroused on game day compared to the practice (p=.058) and significantly more aroused on game day compared to baseline sessions (p<.001). A similar significant difference was reported when comparing the practice and baseline days (p=.02) (Figure 4 and Table 1).

**Perceived intensity of effort**

A main effect of day was observed for the perceived intensity scale (F[1,14]= 59.67; P<.001). Subjects’ subjective level of intensity was higher at game compared to practice (p<.001) (Figure 5). Intensity measures were not obtained at baseline sessions.

**Cortisol**

Significant effects of day were observed for levels of salivary cortisol (F=[2,20]=3.94; P=.003). Anticipatory cortisol on game day was significantly elevated compared to baseline (p=.011) and practice days (p=.01) (Figure 6). Cortisol levels were
not significantly different on a practice day compared to a baseline session. No sex differences existed nor were there any interaction effects.

*Pain measures*

**Arm**

Significant effects of day were observed for pain threshold differences in the arm (F=[2,36]=20.608; P<0.0001). Pain thresholds on game (p<.001) and practice days (p<.001) were significantly higher than baseline days. Thresholds were elevated (subjects were less sensitive to pain) before a game and a practice compared to a baseline day. There was no significant difference, however, between practice and game days (Figure 7). No sex differences existed nor were there any interaction effects.

**Fingertips**

Significant effects of day were observed for pain threshold differences in fingertips (F=[2,36]=21.887; P<0.0001). Pain thresholds on game (p<.001) and practice days (p<.001) differed significantly from baseline days (Figure 8). Once again, game and practice thresholds were not significantly different from one another and no sex differences or interaction effects existed.

*Correlation: Game importance and pain thresholds*

A Pearson correlation coefficient was calculated to investigate the relationship between game importance and pain thresholds (significance level of p < .05). A significant positive relationship was found between game importance and arm thresholds (r(12)=.593, p=.025). This suggests that the more important the game was, the higher the arm threshold difference and therefore, the greater the reduction in pain sensitivity.
(Figure 9). A positive correlation for game importance and finger thresholds was also observed that approached but did not reach significance.

Discussion: experiment 1

The results of this study suggest that athletic competition and routine practice modulate behavioral responses to noxious stimuli. Athletes exhibited higher pain thresholds prior to a practice and game in both arm and fingertip loci compared to baseline, however, the pain thresholds for both experimental days were not significantly different from one another. The data do, however, suggest a trend in the predicted direction; baseline pain thresholds are lower than practice thresholds, which are lower than pain thresholds on the game day.

Other observations support this trend. Correlational results show that pain thresholds were significantly higher when athletes anticipated an important game. There was a positive correlation between the importance of the game and the athlete's tolerance to pain. The more important the athlete believed the game to be, the less sensitive the subject was to pain. Perhaps we can conclude that anticipatory stress is therefore modulated by meaningfulness and expected effort or intensity. Athletes who expect to play in a meaningful, significant competition get more aroused to play from a behavioral, as well as physiological perspective, than before a less important, less meaningful game.

The body awareness questionnaire provided measured subjective levels of alertness and anxiety. Subjects reported being slightly but not significantly more anxious at a game than at practice and more anxious at practice than at baseline. Elevated scores prior to practice and game sessions suggest that subjects did feel more anxious prior to competition (practice and game) than at baseline. Pain threshold data in both loci (arms
and fingers) followed the same trend although the trend was not strong enough to elicit a positive correlation; in sessions when players reported being more anxious, subjects had higher pain thresholds.

Our interpretation is further supported by the hormonal analysis. Anticipatory cortisol levels were significantly elevated prior to a competition (game session) in both males and females compared to levels prior to a practice and baseline session. The high levels of this hormone suggest that cortisol facilitates an appropriate state in which to compete by enhancing energy availability. The lack of hormonal sex differences in the present study is consistent with the findings of a study by Bateup et al. (2002) in which men and women shared a similar rise in pre-competition cortisol. However, Bateup et al. found that men’s pre-game cortisol, unlike the women’s, was affected by skill and experience- more talented male competitors have lower cortisol than less-skilled men (Bateup, 2002). Since the current study did not evaluate skill-level, such a sex difference could not be replicated.

Levels of subjective intensity were also measured at practice and game sessions. Subjects reported being significantly more intense before a game than before a practice. Such a significant difference is consistent with the observed elevated cortisol levels in anticipation of game-related competition as compared to practice levels.

Measurements across the three testing sessions were taken after routine warm-ups (practice and game), or after a short session on a recumbent bike (baseline). The exercise at baseline was designed to mimic the exercise prior to the other two conditions such that exercise intensity at all three sessions was indeed equal. Previous studies have been unable to separate the cognitive mindset associated with competition from the physical
component of exercise. Research using an equivalent rigor of exercise across testing days would help determine whether athletes exhibit pre-competition stress in addition to whether such a stressful psychological state is sufficient to alone produce analgesia.

Discrepancies exist between physiological measures, cortisol levels, pain threshold, and self-report results in the present study. Systolic blood pressure was significantly raised before a game compared to prior to a practice or baseline day. Females’ diastolic blood pressure followed the same trend while males’ diastolic blood pressure was also significantly higher at practice than at baseline sessions. If physical exertion or elevated blood pressure alone leads to analgesia we would expect to observe higher pain thresholds when blood pressure was elevated.

Likewise, if pain threshold was a direct product of heart rate we would expect that the pain results would follow the same pattern as heart rate. Heart rate was significantly elevated before a game compared to before a practice although such a significant difference was not observed between pain thresholds at practice and game sessions. Subjects’ thresholds on game day did not significantly differ from practice levels. It is therefore more likely that observed analgesia was a result of anticipatory stress, not physical exertion.

Researchers believe that the stress component of competition contributes to the pain inhibition associated with physical exertion (Sternberg, 1998). In the context of competitive sport, cognitive appraisal relates to the evaluation of the significance of a particular stressful encounter. A physical or mental error, for example, and its relevance for the athlete’s psychological (e.g. confidence, satisfaction) and physical well-being (e.g. pain, effort) impacts performance quality (Anshel et al., 2001). The amount of cognitive
involvement affects mental stressors depending on such variables as degree of exerted effort, volitional task involvement, and concerns regarding an individual's performance. Therefore, unlike physical stressors, mental stressors are more vulnerable to variability across sessions (Faulstitch, 1986).

Pain data support our hypothesis that anticipation of physical exertion is sufficient to activate pain inhibition. The analgesic effects of anticipatory competition, however, are not significantly different at practice compared to a game. This suggests that athletes appraise competition within their squad (practice) to be almost as competitive as a game against intercollegiate opponents. While subjective intensity reports suggest otherwise, body awareness measurements are consistent.

An additional explanation for our inability to find significant differences in the pain threshold results in the game setting and practice session could be due to the confound of outside temperature. Our testing during game and practice sessions were conducted outside while the baseline sessions were completed indoors. Skin temperature was significantly higher at baseline than at practice and game sessions. It is therefore possible that increased time to reach threshold solely reflects a lower starting temperature while pain threshold remains unchanged. A follow-up experiment was conducted using similar methods to experiment 1, but controlled for temperature. We assessed anticipatory stress and analgesia and measure cortisol in female basketball players before a game, practice and on a baseline day. All sessions were completed indoors to control for temperature in order to determine whether subjects’ pain thresholds are a product of analgesia or outside temperature. Since no gender differences were found in experiment
1, we only included females in our second study due to their willingness to participate and availability.

Methods: Experiment 2

Procedure

Experimenters followed the same procedure in experiment 2 as was employed in experiment 1. All subjects (female basketball players) in experiment 2 were tested on 3 occasions, a baseline session, a practice session, and a game session. Testing occurred after a pre-game and pre-practice warm ups but 50 minutes before the competition. All testing sessions will be completed indoors to control for temperature; practice and game sessions in the gymnasium and baseline sessions in the psychology laboratory.

Data Analysis

Total pain threshold ratings were obtained by taking the difference between the average pain threshold temperature on the subject's arms or fingers and the starting skin temperature of the volar surface of the arm. This method of analysis was used for two main reasons; individual differences in initial body temperature could influence final pain thresholds and to remain consistent with methods employed in experiment 1.

Day effects on the various dependent variables were determined by various factorial ANOVAs with day as the within-subjects factor. Analyses for each sport were kept separate to control for unavoidable differences due to the nature of the sport. A fisher LSD test was run to determine which means were significantly different from one another. Missing data were excluded.

All saliva samples were assayed for salivary cortisol (µg/dl) using an enzyme immunoassay kit (DS labs, Arlington, Texas). All samples were assayed in duplicate and
all samples from the same players were tested in the same assay run. After the assay a plate reader was used to read well absorbances at a wavelength of 450nm using a 450nm absorbance filter. A standard curve was constructed to determine the absorbance levels for each unknown.

Analyses of bivariate Pearson correlations were run to investigate the relationship between various dependent variables.

*Physiological measures*

*Systolic blood pressure*

There was no significant day effect on systolic blood pressure ($F[2,14]=2.61; P=.109$). Trends approached significance in the predicted direction; baseline systolic blood pressure was less than practice and game. Systolic blood pressure on the practice day was also less than on game day (Figure 9, Table 2).

*Diastolic blood pressure*

Unlike experiment 1, there were no significant effects in diastolic blood pressure across the three testing days (Figure 10, Table 2).

*Heart Rate*

A main effect of day was observed for heart rate ($F[2, 14]=12.30, P=.001$). Heart rate on game ($p<.001$) and practice days ($p=.003$) differed significantly from baseline days (Figure 10). Game and practice heart rate were not significantly different from one another.

*Skin Temperature*

A main effect of day was observed for initial skin temperature ($F[2, 16]=26.86; P<.001$). Skin temperature measured just prior to pain testing was significantly higher at
baseline (p<.001) than at practice and game sessions. There was no significant difference, however, between practice and game days (Figure 3).

**Body Awareness**

Unlike experiment 1, there was no significant day effect on body awareness responses across the three testing days (Figure 4).

**Perceived intensity**

A main effect of day was observed for the perceived intensity scale (F[1,8]=16.67; P=.003). Subjects’ subjective level of intensity was higher at game compared to practice (p=.003). Intensity measures were not obtained at baseline sessions (Figure 5).

**Cortisol**

Due to irregularities in the cortisol assay, readings on salivary cortisol levels were inconclusive; readings were much higher than anticipated across testing days. Cortisol levels did not significantly differ across testing days (Figure 6).

**Pain Measures**

**Arm**

Significant effects of day were observed for pain threshold differences in the arm (F=[2,16]=12.71; P<.001). Pain thresholds on game (p<.001) and practice days (p=.001) were significantly higher than baseline days. Subjects were less sensitive to pain before a game and a practice than on a baseline day. There was no significant difference, however, between practice and game days (Figure 7).

**Fingertips**

Significant effects of day were observed for pain threshold differences in fingertips (F=[2,16]=5.44; P<.05). Pain thresholds on game (p=.011) and practice days
Correlations

Pearson correlation coefficients were calculated to investigate the possible relationships between game importance, game intensity measures, and body awareness responses and pain thresholds (significance level of p < .05). No significant correlations were found.

Discussion: experiment 2

Basketball players showed analgesia on both game and practice days compared to baseline. A trend exists in the predicted direction such that practice thresholds were less than game day thresholds; game and practice thresholds were, however, not significantly different from one another.

Although experiment 2 was performed entirely indoors, the results point to a significant difference in the starting temperature as a function of testing site. Practice and game testing were performed in the gymnasium which was on average about 2°C cooler than where baseline testing occurred. The confound of ambient temperature may still impact pain results taken at both game and practice sessions. It is unclear whether a subject’s pain thresholds are a product of the temperature or a product of analgesia.

The difference in skin temperature at soccer games and practices compared to baseline was substantially larger than the observed temperature difference between basketball testing sessions. The weather during soccer season reached cooler temperatures than temperatures during basketball testing sessions in the gymnasium. The magnitude of pain sensitivity, however, was about equal between both sports. Skin
temperature may therefore have a minimal impact on perception of pain when exposed to a heat stimulus. Systolic blood pressure values suggest that the rigor of exercise at all three testing sessions were not significantly different. It is accepted that blood pressure and heart rate vary in the short term in response to physical activity. Heart rate, which is more immediately affected by physical activity than blood pressure (Portier et al., 2001), did vary according to testing day. Practice and game situations induced a higher heart rate than the baseline exercise session. Such increased heart rate levels at practice and game days could be due to a combination of anxiety levels and physical activity.

One main factor that may moderate basketball players’ experience of anticipatory stress involves the possibility of winning. Gamblers, for example, become aroused by the expectancy and prospects of winning money (Wulfert et al., 2005). The participating basketball team in the present study had an overall losing record and rarely expected to beat their opponents. Since anxiety in anticipation of competition is related to the prospect of winning, the teams’ losing outlook may have lowered levels of anticipatory stress. Game and practice days elicited similar levels of anxiety and therefore pain perception did not differ; analgesia was observed before both practices and games.

Irregularities in the cortisol assay prevent interpretation of the results. Hormonal levels did not significantly change as a function of day and absorbance levels are inconclusive. Due to our within subject experimental design, several subjects had to be excluded if one sample across the three testing days was atypical or missing, leading to a small sample size.
General Discussion

As stated earlier, analyses for each sport were kept separate due to an unbalanced sex ratio and inherent differences in each sport. Research involving athletic competition has been criticized due to factors involving heterogeneous samples. In studying sport, researchers must obtain large enough sample sizes, control for age, differentiate between types of sports, and distinguish between outstanding and average performance levels (Egloff and Gruhn, 1995).

Basketball is by nature a high scoring game whereby good scoring opportunities are frequently presented. This is in contrast to the small number of truly good scoring opportunities which occur in sports such as soccer where scoring chances are rare. A defensive mistake in soccer, for example, may carry more importance and cause a higher degree of anxiety than a defensive mistake in a basketball game or practice. If a defensive mistake in a basketball game results in a basket for the opposition, many opportunities to make up the score deficit are likely to occur (Dunn & Nielson, 1996).

Athletes who participated in soccer or basketball, however, experienced similar levels of analgesia prior to practice and game situations. Originally, the practice session was intended to test whether athletes (both males and females) experienced stress and subsequent analgesia in anticipation of routine exercise. The present study provides evidence that the competitive aspects of inter-squad competition and its physical stressors aroused the SIA mechanism.

Elevated levels of subjective intensity on game day suggest that players consider games to be more intense than practice. Since intensity ratings are completely self-reported it is unclear whether subjects simply reported higher intensity because of the
game-driven environment or because they actually felt more intense and more focused on
competition. Much of the existing research on anxiety in sport is too focused on the
characteristics of the athlete, while ignoring the characteristics of the environment in
which the behavior took place. Situational variables have been shown to affect emotions
elicited by various competitive environments (Dunn and Nielson, 1996). Situational
variables involved in an actual athletic competition include but are not limited to the
members of the audience (friends and family), officiating, teammates, and opponents.
Perhaps the athletes are describing the situation and environment as being intense, as
opposed to their individual intensity levels.

Additional stressful situational components of a team's practice session impact
levels of nervousness. The behaviors of the coach play an important role in producing
athlete anxiety (Dunn and Nielson, 1996). During practices players are being evaluated
by coaches and compete against each other to determine who is faster, stronger or simply
better. Coaching decisions during a practice influence an athlete’s playing time, in
addition these decisions often affect an athlete’s confidence in his or her playing ability.
Dunn and Nielson (1996) noted that performance feedback in general was in itself a
contributing factor in creating anxiety for an athlete.

Hormonal levels in soccer players, however, were significantly elevated before a
game compared to practice. Cortisol has been identified as a reliable marker of stress.
Both analgesia and elevated cortisol levels measured before games suggest that game
situations can be considered as reliable stressors. Practice session cortisol levels did not
differ from hormonal levels at baseline. Many outside factors also contribute to the
overall physiological status of a player including conditioning activities, practice
schedules, academic demands, psychological stressors, in addition to competition (Kraemer et al., 2004). Such factors consistently affect a student-athlete’s overall levels of stress and may confound possible cortisol elevations in anticipation of practice.

Research on the role of cortisol in competition is guided by a theoretical model that upholds that in order to gain and maintain status, the competitor must out-stress the opponent through dominant behavior, physical aggression, or performing better. Slight increases in cortisol prepare individuals for action (Kivlighan et al., 2005). Researchers choose sporting events to study hormones and competition since the setting is highly organized; teams have consistent pre and post-game routines and individuals can be tested in natural game and practice settings.

Previous research suggests that environmental stress is a natural trigger for the inhibition of pain sensation (Sapolsky, 1992). It follows that athletes experienced both analgesia prior to a game and elevated cortisol levels due to the stress of athletic competition. Stress is believed to activate the endogenous opioid system which will cause the observed analgesic response (Koltyn, 2000). The HPA axis, which contains parts of the hypothalamus, pituitary gland, and adrenal cortices, also controls the reaction to stress. During stress, a chain reaction in the release of hormones occurs in the HPA axis which leads to the release of glucocorticoids. Cortisol, the “stress hormone”, is the dominant form of glucocorticoids in humans (Sapolsky, 1992). One would therefore expect to observe both analgesia and elevated cortisol levels in a stressed individual.

It remains difficult to reconcile the contrasting stress data in the present study. The pain data suggests that athletes consider both practice and game situations stressful, while the hormonal analysis suggests that soccer players’ salivary cortisol levels are
significantly elevated at a game when compared to practice. Two main explanations could shed light on such unexpected differences; either cortisol secretion is more susceptible to minor differences in stress levels than inhibitory pain pathways or practice was not a stressor strong enough to activate the HPA axis.

Perhaps hormonal changes are more susceptible to stress than inhibitory pain pathways; players may have been slightly more stressed prior to a game compared to practice. Such a slight change may have only significantly affected cortisol levels while pain data gathered at game and practice days are not significantly different from one another. Changes in pain perception could only be observed in situations that cause significantly elevated levels of stress such as a soccer game while cortisol elevations may occur due to slightly higher levels of stress.

Alternatively, the lack of elevated cortisol levels at practice compared to baseline could be due to the natural amount of time it takes for activation of the HPA axis to take place. It is possible that when stressed, the body’s endogenous opiate-system responds faster or is more sensitive than the HPA axis’s release of glucocorticoids. Cortisol and additional glucocorticoids are released after a series of reactions that begin in the HPA axis in response to stress. ACTH and β-endorphin are co-released after the hypothalamus releases CRH in response to a stressful stimulus. Both ACTH and β-endorphin have roles in the regulation of stress; ACTH stimulates the adrenal which releases cortisol and β-endorphin binds to opioid receptors which produces analgesia (Sapolsky, 1992). The pain data suggest that anticipation of practice was a strong enough stressor to activate the release of β-endorphin and produce analgesia while the cortisol measurements suggest that players were no more stressed than they were at baseline. Subjects reported feeling
somewhat anxious at practice but perhaps not anxious enough to elicit a strong HPA axis response.

There are several limitations of the present study that one must consider when interpreting the results. The lack of consistent findings could be a result of our limited sample size. Subjects were only included if they expected to play regularly in games, in other words, those who anticipated having a direct impact on the outcome of the game. Due to availability and additional selection criteria we only tested 9-11 players per team. Since significant differences were found between both sports, it was necessary to run separate analyses. Perhaps more conclusions could have been drawn if results from both sports could have been analyzed together.

Outside temperature is an additional variable that is difficult to control. Baseline skin temperature influences the magnitude of pain caused by a heat stimulus (Wu et al., 2001). In order to fully understand the biophysical basis of heat transduction in nociceptors, it is important to first grasp how skin temperature specifically relates to heat pain. Previous studies have found that the time required for pain to be noticed varies inversely with the base temperature (Pertovaara et al., 1988). More recently Wu and colleagues (2001) found that a 4°C increase in baseline skin temperature (from 34°C to 38°C) leads to a small but statistically significant increase in pain ratings to a suprathreshold temperature-controlled heat stimulus. This finding suggests that pain sensitivity related to a temperature-controlled heat stimulus can be influenced by a change in skin temperature (Wu et al., 2001). Thus, the analgesic findings from the present study could be due to the combination of two factors: 1) base temperature, which influenced the sensitivity to the pain stimulus or 2) stress elicited from competitive
situations, which in turn caused analgesia and subsequently higher pain thresholds. Perhaps future studies of heat-related pain threshold should perform all tests in the same exact location.

The current study supports the hypothesis that females experience pre-competition stress in the same way as males. Results show no significant sex differences in all major variables tested. Male and female soccer players exhibited the same analgesic response in anticipation of competition in both games and practices compared to baseline. The cortisol levels in the men and women did not differ across testing days; cortisol was elevated prior to games compared to practice and baseline days.

A debate exists as to whether males and females have similar reactions in anticipation of competition. Taylor et al. (2000) outlined a theoretical rationale that challenges the assumption of gender similarity in gaining status by out-stressing the opponent. The theory states that females, unlike males, respond to challenges by building on social networks (befriending) as a means to reduce stress (Taylor et al., 2000 as cited by Kivlighan, 2005). Other researchers argue that women do compete for status and have observed pre-competition rises in testosterone (a hormone linked to the pre-event phase) and cortisol (Cashdan, 1998; Bateup et al., 2002).

To test the assumptions of gender similarity, further study of biobehavioral responses need to be examined in a social context in which both men and women compete in similar settings (Kivlighan et al., 2005). To further our knowledge of the potential sex differences in coping with pre-event stress, perhaps the present study could be repeated with more male subjects and include a questionnaire to evaluate levels of dominance, competitiveness, and team bonding. An assay to test levels of testosterone
would also help clarify whether genders respond differently to anticipation of competition.

Researchers often choose sporting events to study hormones and competition due to the highly organized setting. There are several advantages to investigating a phenomenon through a naturally-occurring event. Researchers also must avoid interfering with the performance of their subjects who are participating in the event. Therefore, certain variables were out of our control. Pre-game warm ups, for example, were more strenuous than pre-practice warm-ups. Also, due to the nature of the pre-game routine, we had to test our subjects 45 minutes before the actual competition whereas pre-practice measures were taken immediately before practice.

Since all subjects were by nature competitive athletes, subjects reported a desire to be able to “withstand the most pain”- a measure of toughness, pain tolerance, and inter-squad competitiveness. It would have been beneficial to include non-athlete controls to compare overall cortisol and pain threshold levels between athletes and non-athletes. Strategies for coping with pain such as diverting attention and ignoring pain are associated with the ability to function physically and psychologically while competing (Meyers et al., 2001). Such a finding suggests that athletes would have higher pain thresholds and cortisol levels compared to non-athletes. Inclusion of a control group would help clarify whether a difference exists between athletes’ and non-athletes’ pain thresholds and their respective ability to deal with stress.

Competition can be considered a stressor significant enough to evoke analgesia in athletes. The present study found that male and female soccer players and female basketball players experienced analgesia in anticipation of both inter-squad (practice) and
inter-collegiate (game) competition compared to baseline. Soccer players also had elevated cortisol levels prior to games (inter-collegiate competition) compared to practice and baseline measures.

Future research should focus on the impact of variables including but not limited to level of success and skill-level, sex, and sport-type on pre-competition levels of stress. Previous research has suggested that such variables may impact pain perception and how athletes respond to stressful situations. Additional research is needed to clarify whether biobehavioral sex differences in stress-coping mechanisms exist before, during, and after competition. Research should also examine the accuracy of the available mechanisms for pain measurement and whether and how base temperatures affect such measurements. Further study of such variables is necessary to clarify what situational variables contribute to the appraisal of an environment as stressful and what specifically governs the endogenous response to stress.
Works Cited


