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

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Abstract: Participating in a competitive athletic event, for example a routine practice or game, can produce anticipatory elevations in cortisol and pain threshold in male and female college athletes (basketball and soccer players). In an attempt to bridge the theoretical gap between competition-induced analgesia (CIA) and stress-induced analgesia (SIA), the present study investigated the association between subjective and physiological measures of stress to nociceptive response in the context of competitive athletics. Heat withdrawal latencies confirmed expectations that the anticipatory stress of competition can elicit analgesic effects in both basketball and soccer players. Salivary cortisol in soccer players was consistent with this result, showing significant elevations prior to a game compared to practice and baseline sessions, but did not follow this same pattern among basketball players. These results provide evidence for HPA activation of the stress response and its subsequent interaction with the pain pathway in inducing analgesic effects.

Pain sensation is arguably the most distinctive of all the sensory modalities. When we sprain an ankle, overt changes such as swelling and bruising are preceded by sensations of burning, aching, and soreness. Verbalizing exact pain sensation is difficult, but can be universally understood through shared experience of what pain "feels" like. Pain is encompassed in somatic sensation, which includes touch, pressure, and position. Without sensitivity to noxious stimuli, organisms would be unable to avoid potential harm and left unaware of sustained injuries. Like other sensory modalities, pain is a percept—it is a derived perception that the nervous system mediates and brain integrates in response to noxious input. It has been said that pain, "is an unpleasant sensory and emotional experience associated with actual or potential tissue damage" (Basbaum & Jessell, p. 472).

The sensation of pain plays a primitive role to aid organisms in survival and reproduction. If pain is indeed an adaptive response, then why and how, can actual tissue

damage fail to elicit pain in extreme cases? There are circumstances during which pain sensation is not perceived in the presence of a noxious stimulus, suggesting that the inhibition of pain perception is adaptive to an organism's survival. More specifically, these patterns of pain inhibition can be differentially elicited across individuals when exposed under the same conditions. Pain behavior is not just a product of individual differences, however, as external conditions can also influence the intensity of pain sensation.

This specific phenomenon of the brain modulating incoming information, and forming a sensation of pain will be the focus of this paper. Early work on pain behavior concentrated initially on the psychological state of an individual in his or her response to pain. Seminal research by Beecher (1956) compared pain behavior in soldiers and civilians in the classic study of wounded war veterans during World War II. Beecher indirectly assessed pain perception by tracking soldier's requests for pain medication and systematically comparing these to civilian patients with comparable injuries. Dramatic differences were observed between wounded soldiers and civilians; 25% of combat-wounded soldiers requested medication, the remainder of which reported no pain or very little pain. In comparison, civilians with similar wounds reported much more pain, with more than 80% actively requesting pain medication. From these findings, Beecher proposed that the altered psychological states, namely heightened affect and "secondary gain," could considerably alter pain perception. Similarly, objective reports of athletes demonstrate a generalized neglect of injuries sustained during competition, and are detected only after the game is over.

The present study intends to examine athletic competition as a stressor that can activate the HPA-axis stress response and induce pain inhibition (analgesia). Both male and female athletes will be used to reveal potential sex differences in pain responses and to different stressful stimuli, namely to a routine practice situation and a collegiate soccer or basketball game. Stress levels will be assessed using self reports of body awareness and arousal, expected ratings of physical exertion, physiological measures, as well as salivary cortisol levels. Pain behavior will be tested using noxious heat stimulation on the volar surface of the arm and the fingertips to assess pain thresholds. These measurements will be collected on baseline day (after five minutes of cycle ergometry) and prior to our two experimental conditions (game and practice). The specific timing of data collection will explore the phenomenon of anticipatory stress to athletic competition, but also to avoid the potential effects of exercise from confounding the effects of competition.

Anatomy of Pain

~Nociception

The peripheral pain sensory system is composed of primary afferent nerves called nociceptors, which are specialized cells that detect tissue damage. Nociceptors serve two primary roles wherein chemical, mechanical, or thermal energy is transduced into nerve impulses. These signals are transmitted to the spinal cord and brain, where the subjective perception of pain is generated (Fields, 1987). There are three principle classes of nociceptors—thermal, mechanical, and polymodal, all of which work together and are broadly dispersed in the skin and subcutaneous tissues. Thermal nociceptors are sensitive to extreme temperatures which fall under 5° C or above 45° C (Basbaum & Jessell,

2000). Mechanical nociceptors are activated by intense pressure applied cutaneously, whereas polymodal nociceptors are activated after exposure to thermal (hot and cold), chemical, or intense mechanical stimuli (Basbaum & Jessell, 2000). For our purposes, the focus will be on thermal nociceptors.

The primary afferent axons of nociceptors support the diverse functions of the various thermal, chemical, and mechanical nociceptive classes. Three major groups of axons have been classified based on axonal propertie, such as diameter, myelination, or unmyelination—they are C, Aβ, and Aδ. C axons have the smallest diameter and are unmyelinated, thus conducting slowly at velocities ranging from 2m/s to less than 1m/s (Fields, 1987; Basbaum & Jessell, 2000). C fibers transmit characteristic "aching and burning second pain" information from polymodal nociceptors. Myelinated afferents such as Aβ, and Aδ have much faster conduction velocities ranging from 5 to over 100 m/sec (Fields, 1987). Aβ fibers are the thickest and mostly non-nociceptive, and are sensitive to mild mechanical stimuli (Fields, 1987). Aδ fibers are small in diameter and thinly myelinated, transmitting signals at 5-30 m/s from both thermal and mechanical nociceptive classes (Basbaum & Jessell, 2000). Thus, it is logical that Aδ fibers transmit the nociceptive information more quickly than C fibers, generating the initial sharp "first" pain sensation (Basbaum & Jessell, 2000).

The majority of evidence, based on the physical properties of nociceptors and their axons, points to nociceptive primary afferents transmitting at the Aδ or C velocity range. Moreover, Aδ and C primary afferents are most sensitive when noxious stimuli come in contact with their corresponding receptive fields on the surface of the skin.

These fibers are highly accurate in providing information about the location, intensity, and quality of the noxious stimulus.

A more in depth model of Aδ nociceptors reveals that these fibers largely respond to noxious mechanical stimuli, where the remaining 20-50% respond to both heat and mechanical stimuli, specifically to temperatures above or below pain threshold. (Fields, 1987). Aδ nociceptors that are more sensitive to mechanical stimuli are called Aδ mechanoreceptors. Aδ nociceptors that are maximally activated by heat are called mechanothermal nociceptors, and finally those responding to both thermal and mechanical stimuli are labeled high-threshold mechanoreceptors (HTMs) (Fields, 1987). Both mechanothermal and HTMs can become sensitized, whereby repeated noxious stimulation induces progressively larger responses. Behaviorally, this sensitization results in a lowering of threshold temperature for pain perception (Fields, 1987). Sensitization is an important concept that will be taken into consideration in the experimental design of the current study.

Unmyelinated C fibers compose the majority of axons in three-quarters of peripheral nerve primary afferent, of which 90 percent are nociceptive in primate limb nerves (Fields, 1987). In humans, this number increases to 100 percent. C-polymodal nociceptors (C-PMN) respond to noxious chemical, mechanical, and thermal stimuli. Like Aδ myelinated nociceptors, C-PMNs may also undergo sensitization to repetitive bouts with a noxious stimulus. Somewhat paradoxically, C-PMNs have been found to show reduced sensitivity shortly after the application of a noxious stimulus where temperatures above 55° C elicit no activity, suggesting that noxious stimuli may momentarily inhibit pain perception immediately after its withdrawal (Fields, 1987). For

the purpose of the present study, it is important to note that thermal stimuli produce a consistently good correlation between C-PMN activity and subjective ratings of pain intensity (Fields, 1987).

~Dorsal Horn of the Spinal Cord—Laminae I-VII

The three major classes of primary afferents responding to noxious stimuli, Aδ mechanothermal nociceptors, Aδ mechanosensitive nociceptor, and the C-PMN terminate principally in the dorsal horn of the spinal cord. There are six distinct layers in the dorsal horn, in which laminae I-VI are topographically arranged by function (Basbaum & Jessell, 2000). In regards to the nociceptive afferent pathway, only three layers, I, II, and V, are important to discuss. These three superficial layers are the primary sites at which nociceptors synapse. Present evidence designates lamina II (the substantia gelatinosa) as the predominant site for C fiber termination. Aδ fibers terminate in laminae I and V (Fields, 1987). Animal models have refined our knowledge of neural activity at laminae I, II, and V in response to noxious stimulation. The dorsal horn of the spinal cord marks a critical junction from which afferent nociceptive activity is projected to higher order brain regions, where pain becomes a perceptual experience. It is thus important to scrutinize this "integrative component" of the afferent pain pathway.

Most lamina I neurons are specialized to respond to noxious stimulation but also receive input from non-nociceptors; similar to other sensory systems, the convergence of input from primary afferents onto one neuron expands the receptive fields of lamina I neurons (Fields, 1987). A large portion of lamina I neurons are projection cells—many project to the thalamus while others form interconnections among lamina I cells. Lamina

II has been implicated in the somewhat controversial "gate-control theory," which proposes that modulation of afferent inputs can at times shut off activity at the source. Though some of its neurons are activated by noxious input, lamina II consist mostly of interneurons, and projects to the brainstem and thalamus (Fields, 1987). Lamina V cells show great similarity to lamina I cells in that they are maximally sensitive to noxious stimuli, and have a large proportion of cells projecting to the brainstem and thalamus (Fields, 1987). Lamina V cells also show much greater convergence—from both nociceptive and non-nociceptive afferent inputs, and have also been shown to make interconnections among neurons located in laminae I through IV (Fields, 1987).

Substantial evidence indicates that laminae I and V nociceptive neurons are important in transmitting the nociceptive message, which is apparent in their respective projections to the thalamus. Thermal stimulation in the range of 43° C to 50° C elicits activity in both laminae I and V neurons of the spinothalamic tract; as thermal stimuli move into noxious range, human reports of pain increase in conjunction with increased activity in spinothalamic tract cells (Fields, 1987).

The previous concept of sensitization, in which repetitive heat stimuli can lower thresholds of all primary afferent nociceptive classes, manifests behaviorally as greater pain sensitivity (hyperalgesia). Alternatively, rapid noxious stimulation can cause EPSPs in unmyelinated nociceptors to summate in an additive manner, effectively increasing the intensity of pain sensation but not pain threshold (Fields, 1987). Unmyelinated C fibers that demonstrate summation will not sensitize in response to the same stimulus. This suggests that summation occurs within the CNS where the perceptual experience of pain is generated.

~Ascending Pathways

There are five major ascending pathways from the spinal cord to higher order structures in the brain: spinothalamic, spinoreticular, spinomesencephalic, cervicothalamic, and spinohypothalamic. The sheer quantity of ascending nociceptive pathways into the brain is a clear indication that pain is a complex phenomenon. In processing afferent pain sensation to pain perception, three of these pathways in particular, are important. The spinothalamic tract is the largest ascending nociceptive pathway in the spinal cord. Neurons from laminae I and V project to the contralateral side of the spinal cord, ultimately terminating at the thalamus. Clinical observations have demonstrated that spinothalamic tract lesions generate severe deficits in pain and temperature sensation, indicating that normal pain sensation requires this pathway (Basbaum & Jessell, 2000). As its name indicates, the spinoreticular tract terminates in the reticular formation and thalamus. Respectively, these structures are involved in arousal and information processing, which can explain why painful stimuli induce high arousal and escape behaviors. The spinomesencephalic tract consists of axonal bundles from neurons in laminae I and V and projects to both the mesencephalic reticular formation and periaqueductal gray matter (PAG). The PAG contains opiate receptors which opiate drugs bind to, resulting in inhibition of the afferent pain message (analgesic effects). Additionally, some spinomesencephalic axons project to the limbic or affective center, specifically to the amygdala via the parabrachial nuclei. Such projections to neural centers for emotion provide evidence for the role of the spinomesencephalic tract in the affective component of pain.

In particular, the lateral nuclear group of the thalamus receives afferent input from the spinothalamic tract (neurons in laminae I and V) and are thought to be implicated in mediating general information about the location and intensity of a painful sensation (Craig et al., 1996). Recent research has capitalized on the advent of imaging technology to non-invasively study cortical activity in response to noxious stimulation. PET scans have revealed "activity" in cingulate gyrus and the insular cortex during nociception (Craig et al., 1996). One component of the emotional content of pain can be attributed to spinomesencephalic projections to limbic system structures; however PET scans have also implicated other limbic system structures, such as the cingulate gyrus in processing the affective component of pain (Craig et al., 1996). With its projections from various thalamic nuclei, the insular cortex is believed to process information about homeostasis of the body, and thus may be responsible for the autonomic aspects of the pain response. As a result, the insular cortex may be a site for integration of all sensory, emotional, and cognitive components of the pain perception.

Through clinical observation, it has been shown that patients who have experienced damage to any of the aforementioned pathways (from peripheral nerve to cortex), can still experience severe pain. How can this be reconciled with the fact that primary nociceptive afferents project to the brain to form pain perceptions? The simple answer is that both non-nociceptive (Aβ fibers) and nociceptive afferent input (C and Aδ fibers) can play an inhibitory role. When these inhibitory afferents are removed, painful sensation will no longer be inhibited. This notion is supported by pain reports in humans; when myelinated primary non-nociceptive afferents are blocked, leaving only C-fibers remaining intact, sensations from applied noxious skin stimuli are reported as more

painful (Fields, 1987). Additionally, when these same afferent fibers are selectively blocked, summation of second pain is reported to be more intense. From these pain reports, it is apparent that myelinated nociceptors afferents (such as Aδ) have both excitatory and inhibitory connections to projection neurons in the laminae of the spinal cord (Fields, 1987).

Currently it is unclear what the exact function of afferent inhibition is. In studies of primate spinothalamic tract neurons, it has been observed that activation of myelinated Aδ nociceptors more effectively inhibits these neurons in comparison to stimulation of Aβ mechanoreceptors (Fields, 1987). However, the long standing "gate-control" theory posits that pain cannot solely be produced by nociceptive afferent input; rather activity from noxious input is modulated via a balance of nociceptive and non-nociceptive afferent activity (Basbaum & Jessell, 2000). Some theories suggest that these inhibitory inputs serve as a limiting mechanism for reflex responses, or provide more reliable localization of noxious stimuli. This pathway may be implicated in analgesic processes through a potential chemical pathway that amplifies these afferent inhibitory effects, or through descending inhibition.

Descending Pain Inhibition

~Stimulation-Produced Analgesia

The discovery of an endogenous opioid mechanism in the brain and CNS indicates that the CNS not only generates the pain percept, but has the capacity to inhibit the incoming pain message, effectively resulting in analgesia. Yet how is analgesia specifically produced? Reynolds' seminal study in rats (1969) demonstrated pain

inhibition through descending pathways—specifically that of electrical analgesia in rats. Reynolds characterized this pain related phenomenon as the inhibition of pain behavior upon exposure to noxious stimuli while all other motor functions remained intact (Reynolds, 1969). Findings indicated that specific brain stimulation of areas in and around the periaqueductal midbrain structures induced electrical analgesia in the rat. Pain behavior was measured during exploratory laparotomy while the animals underwent continuous brain stimulation. Thus, animals were able to undergo surgery with no additional analgesic or anesthetic. After brain stimulation was terminated, analgesia significantly diminished and responses to noxious stimuli returned fully within five minutes (Reynolds, 1969).

These results were replicated in humans in a clinical study by Hosobuchi and colleagues (1977). After permanent implantation of electrodes in the periventricular and periaqueductal gray matter, human subjects reported reduced pain (Hosobuchi et al, 1977). Patients were able to administer their own stimulation to relieve pain and did so repeatedly and in no particular pattern. Administration of naloxone, a drug that can counteract the effects of analgesia through endogenous opioid mechanisms, reversed pain relief from stimulation in five out of six patients (Hosobuchi et al, 1977). These finding yielded positive clinical applications, and also provide further support for the importance of animals in pain research. Hosobuchi extended these initial findings by measuring the levels of immunoreactive β-endorphin in the ventricular fluid of six patients with intractable pain (Hosobuchi et al, 1979). Stimulation of the periaqueductal gray matter resulted in significant increases in the concentration of β-endorphin in three of the patients with peripheral origin pain. These findings support that relative pain inhibition

achieved via electrical stimulation and endogenous opioid pathways may be modulated by activation of β-endorphin producing areas (Hosobuchi et al., 1977).

As alluded to in the 1977 Hosobuchi study, the periaqueductal gray has a considerable density of opiate receptors and opioid peptides, which is supported by observations that stimulation-produced analgesia shows tolerance and naloxone-reversible pain relief (Terman, 1984; Hosobuchi et al., 1977). Previous research indicates that naloxone is an opioid antagonist, in effect confirming that PAG structures have opioid substrates (Hosobuchi et al., 1977; Hosobuchi et al., 1979). It is also known that non-opioid (naloxone-insensitive) receptors exist in stimulation-produced analgesia, suggesting that there are two analgesic systems—some that are regulated by opioids and others that are not (Terman et al, 1984).

~Descending Pathways—Anatomy of Analgesia

From evidence of inhibitory pain phenomena such as stimulation-produced analgesia (SPA) and stress-induced analgesia (SIA), it is clear that the central nervous system is capable of modulating pain perception and subsequent responses to noxious stimuli through both opioid and non-opioid mediated analgesia. Modulation first occurs at the level of the spinal cord, where afferent nociceptive and non-nociceptive pathways can regulate signals that are sent to the brain, as described previously. SPA functions by way of descending pathways where serotonergic neurons of the nucleus raphe magnus are recruited to inhibit neurons in laminae I, II, and V of the dorsal horn (Basbaum & Jessell, 2000). The discovery of the analgesic properties of opiates like morphine and codeine, not only offers clinical applications, but also allows researchers to study the involvement

of endogenous opiate pathways using opiate antagonists like naloxone. Injection of naloxone into the PAG region or the serotonergic nucleus raphe magnus is found to block opiate receptors, thus reversing stimulation produced pain inhibition (Basbaum & Jessell, 2000).

Findings from endogenous opioid mechanisms of analgesia indicate that the brain has specialized receptors for opiates, which have been identitified as μ, δ and κ (Basbaum & Jessell, 2000). Three endogenous ligands have been found to interact with μ, δ and κ receptors: enkephalins, β-endorphins, and dynorphins (Basbaum & Jessell, 2000). It is noteworthy that β -endorphin is involved in the stress response, which will be further elaborated upon in the context of the stress response. How specifically do these opioid receptors and ligands function in analgesia? At the physiological level of the synapse, all three classes of opioid receptors are found on nociceptive afferent terminals as well as on the dendrites of postsynaptic neurons of the dorsal horn. Although we know a significant amount about the cellular mechanisms of endogenous opioids in producing analgesia, non-opioid mediated analgesia is still not understood, but nonetheless important to acknowledge in both stimulation-produced analgesia (SPA) and stress-induced analgesia (SIA).

~Stress-Induced Analgesia

Unlike stimulation-produced analgesia, an exogenously generated response that bypasses naturally occurring pain inhibition pathways, stress-induced analgesia is a natural primitive response to stressful stimuli. Although these analgesic producing/inducing phenomena operate through the same pathways and result in the

same behavioral output, SPA and SIA are triggered by different stimuli. Stress induced analgesia (SIA) has been observed in rats exposed to stressors like inescapable footshock in a variety of conditions (Terman et al, 1984). Depending on the duration and continuity of the administered shock, analgesia was produced that was either counteracted by naloxone, or insensitive to the opioid antagonist (Terman et al, 1984). Thus, it seems that stress can physiologically trigger both endogenous opioid or non-opioid systems just as SPA can, supporting the contention that SPA and SIA operate through one mechanism. Pain behavior is measured using pain threshold and pain tolerance tests. Using withdrawal latencies from a noxious stimulus, pain threshold indicates the point at which a stimulus becomes "painful." Pain tolerance, in contrast, is the duration of time an individual can withstand painful stimuli. These measures have different applications in the pain behaviors of humans and animals.

In determining which analgesic system is activated, it appears that the properties of the stressful stimuli are important. In humans it has been shown that anticipation of noxious foot shock is stressful, where repetition of the shock elicited analgesic responses manifest in the progressive increase of the pain reflex threshold (Willer et al, 1981). This analgesic response was reversed by naloxone, indicating involvement of an intrinsic opioid system in SIA. It appears that slight nuances in stressful stimuli (i.e. footshock) such as temporal pattern, duration, and intensity, can mediate the activity of either analgesic system (Lewis et al, 1980). Terman and colleagues reported that shorter durations or lower intensities of foot shock administered to rats induced opioid analgesia, as distinguished by naloxone reversal (Terman et al, 1983). In contrast, longer durations or higher intensities of shock elicited non-opioid analgesia (naloxone-insensitive)

(Terman et al, 1983). It has also been noted that depending on the body region shocked, either analgesic system may be activated. For example, front paw shock can produce opioid analgesia while hind paw shock can cause non-opioid analgesia (Watkins et al, 1982). Watkins and colleagues suggest that the nature of the stressor supersedes where the shock is applied; these results demonstrate that either analgesic system can be activated depending on the intensity of the shock, regardless of body region tested (Watkins et al, 1982).

It is important to consider the nature of the stressor when testing in experimental conditions. How natural is a stressful stimulus such as foot shock? Though it may not be encountered in natural environments, it is both useful and effective in demonstrating SIA. Research in SIA responses has demonstrated that natural stressors like fighting, thermal stress, food scarcity, and sexual arousal can also activate analgesia. However, there is still much work to be done in determining what other stressors evoke SIA in humans; one recent line of research has focused on certain types of exercise and athletic competition in inducing SIA (Koltyn et al, 2000; Sternberg et al, 2001).

It has also been demonstrated that analgesia can become a conditioned response, which is adaptive for an organism that is regularly exposed to stress. Through a process of learned association, a previously aversive stimulus (chamber where foot shock occurred) will induce the same analgesic response, even after this environment becomes neutral (chamber without the foot shock). This conditioned response thus prepares the organism for a potentially stressful situation (Terman et al, 1984). Studies have been carefully designed to determine that conditioned analgesia is indeed a "conditioned response to a conditioned mediator" and not to a mediating emotion such as fear (Terman

et al, 1984). The ability for the SIA response to be conditioned has some interesting applications in humans; for example, how might conditioning affect the stress response to a regular athletic activity in relation to one participated in less often?

Like stimulation produced analgesia (SPA), stress-induced analgesia (SIA) can be driven by both opioid and nonopioid mechanisms. Some cases of stress-induced analgesia can be reversed by opiate antagonist naloxone, whereas others cannot. Although future research may elucidate the distinction and purpose of these two mechanisms, for the purpose of this study it is more relevant to understand why and how stress can produce pain inhibition.

Why does Stress Induce Analgesia?

~Fight or Flight

Why have humans or for that matter, living organisms in general, evolved mechanisms for pain inhibition? Without the sensation of pain, organisms would have no means to identify noxious stimuli, or potentially detrimental environments. There would be no system to signal the potential for injury, in turn debilitating our ability to determine appropriate circumstances from which to escape. The long known term, "fight-or flight" refers to the homeostatic change that prepares an organism to face danger or escape from it. Extreme environmental conditions necessitate a different approach from an animals' normal pain response—namely escape, rest, reflex withdrawal, and recovery (Basbaum & Jessell, 2000). A normal pain response would be detrimental to an animal's survival when attempting to escape from a predator or other potentially harmful situation. Under

evolutionary pressures, mechanisms for pain suppression have been selected for as a short term coping strategy to aversive stimuli.

~HPA Axis

The stress-response is mediated by stimulation of the hypothalamic-pituitary-adrenocortical axis (HPA axis), which coordinates two endocrine systems involving the adrenal gland—the medulla and the cortex. When animals are exposed to stressful stimuli, Selye noted glucocorticoids secretion in a nonspecific response called the general adaptation syndrome (Sapolsky, 1992). Upon stimulation of the HPA axis through certain stressors, specifically that of specific psychological stimuli, a carefully timed sequence of endocrine events is initiated; whereby corticotrophin-releasing factor (CRF) is released from the hypothalamus, in turn stimulating the release of adrenocorticotopic hormone (ACTH) from the pituitary gland. The final step in this cascade of events is the release of glucocorticoids from the adrenal gland via stimulation of ACTH. Cortisol is the primary compound released within this class of steroid hormones (glucocorticoids). From stressor to glucocorticoid release, this entire cascade of endocrine events takes only a few minutes.

In addition to these endocrine sequelae, the stress-response also involves the sympathetic nervous system which is divided in two branches—the autonomic (involuntary) and non-autonomic (voluntary). Within the autonomic nervous system, the parasympathetic nervous system mediates "resting and digesting" functions, whereas the sympathetic nervous system activates the "fight-or-flight" response. When an animal is exposed to a stressful stimulus, catecholamines are released. This class of compounds

consists of epinephrine and norepinephrine, which are released from the adrenal medulla and sympathetic nerves, respectively. In combination, both hormones initiate a cascade of homeostatic events in metabolic, immune, and circulatory systems.

Some of these events in particular, include the secretion of β-endorphin by the pituitary gland. β-Endorphin is an opiate ligand involved in inhibitory control of pain perception. During the "fight-or-flight" response, parasympathetic activities are down-regulated to allow the body to mobilize sympathetic functions and conserve energy (Sapolsky, 1992). This suppression in parasympathetic activity is manifest by the release of prolactin, vasopressin, and glucagon (Sapolsky, 1992). Prolactin is involved in reproduction, whereas vasopressin regulates renal function and water retention. Glucagon, a pancreatic hormone, mobilizes the breakdown of energy deposits (fat) in the form of usable energy—glucose (Sapolsky, 1992).

Analgesia is an important component of the stress-response, specifically in preparing the organism for "fight-or-flight." Up until the early 1970s, pain inhibition as a result of SIA, was thought to be a purely psychological phenomenon. However, pain research revealed the biological underpinnings of SIA. Specific relay sites of the pain pathway such as the PAG region and dorsal horn of the spinal cord were found to have three classes of opioid ligands and receptors. Experimental evidence from animals demonstrated that foot-shock stress induces secretion of β-endorphin, though the exact implications of the opiate are unclear in analgesia (Terman et al., 1984). Other stressors were also reported to invoke a supposed "β-endorphin high" in athletes.

β-Endorphin's involvement in opioid mediated SIA can also be demonstrated at the genetic level. The pro-opiomelanocortin (POMC) gene product is the precursor for

ACTH and β-lipotropin (β-LPH) (Yamada & Nabeshima, 1995). β-Endorphin is a derived product of β-LPH (Yamada & Nabeshima, 1995). β-Endorphin has been identified as the only peptide to have affinity for μ-opioid receptors, but also binds to δ-opioid receptors (Yamada & Nabeshima, 1995). It is known that SIA is not entirely dependent on β-endorphin however, as the opiate receptor blocker naloxone, has no effect at times (Yamada & Nabeshima, 1995). This conclusion is consistent with both animal and human models of SIA, in which manipulations of the same stressor can alter which analgesic system (opioid vs. non-opioid) is activated (Terman et al., 1984; Lewis et al., 1980).

As we have seen, SIA has a logical biological foundation involving opioid and non-opioid systems, both of which play a role in pain inhibition; however, psychological factors such as stress can also influence pain perception. For example, one person may consider a particular stressor to be very stressful, whereas another person, exposed to the same stimulus, may perceive it to be minimally stressful. Therefore, differences in stress appraisal lead to subsequent disparities in the stress responses. These are ultimately manifest downstream in the unique analgesic response of an individual (Sapolsky, 1992). Research points to an individual's perception of "lack of control, lack of predictability, and lack of outlets for frustration" as possible factors that can regulate the stress response (Sapolsky, 1992). As a result, psychological factors such as the level of perceived stress of the situation must also be taken into consideration in examining the role of SIA in pain perception. The perceptions of one's environment is essentially a contrived representation generated by the brain, in which subjective experience is constructed from the sensory modalities.

Similar to individual differences apparent in the stress response, there are also distinctive trends across the sexes in glucocorticoid profiles. Females tend to have higher glucocorticoid concentrations in their blood, than do males. This sex difference is not attributed to psychological responses to stress, but results from different hormonal balances—particularly that of estrogen, which alters levels of circulating glucocorticoids in the blood (Sapolsky, 1992). When bound to proteins, specifically corticosteroid-binding globulin, hydrophobic glucocorticoids circulate more easily. Estrogen induces corticosteroid-binding globulin protein synthesis and glucocorticoid circulation is promoted. Ultimately resulting in elevated levels of glucocorticoids in the bloodstream (Sapolsky, 1992).

Although we know both males and females exhibit SIA, Taylor has proposed that females may differ in their biobehavioral response to stress (Taylor et al., 2000). She posits that the phrase "tend-and-befriend" is more suitable description for the female stress response than the long standing notion of "fight-or-flight (Taylor et al., 2000). Despite the interesting implications this theoretical model may have in the overall stress response, it may not directly affect cortisol levels. Instead, the female "tend-and-befriend response may have greater effects on other regulatory hormones, such as oxytocin and other female reproductive hormones (Taylor et al., 2000). Studies of primates suggest that higher levels of endogenous opioids correlate to relative levels of social interaction and maternal behavior (Martel et al, 1993). Upon administration of naloxone, these behaviors were diminished in female monkeys. Jamner and colleagues (1998) found similar results in humans; naltrexone, a long-acting version of naloxone, decreased affiliative behaviors. These behaviors were manifest in the increased time spent in

isolation, reduced time spent with friends, and lower reports of pleasantness of social interactions, compared to men (Jamner et al., 1998). Taylor's research indicates a likely role of this stress response in female affiliative behavior; how this "tend-and befriend" model may affect SIA and pain behavior has yet to be examined.

Experimental Models of SIA

~Animal Models

Animal testing of pain behavior and SIA are critical for understanding the biological mechanisms behind corresponding human behaviors; animal studies can provide insight into the biological, behavioral, and psychological components of pain behavior where human studies cannot. Most of the animal research uses standard stress-inducing paradigms such as swimming and foot-shock to elicit SIA. The rationale for using these artificial "stressors" exists in the inherent novelty and unpredictability of these tasks. For a rat or mouse, contact with a noxious stimulus such as foot-shock, and/or forced participation in swimming are potentially lethal situations, and involve exposure to extreme temperatures and physical exertion, respectively. Based on models of stress and its underlying biological mechanisms, it follows logically that these conditions will demonstrate stress-induced analgesia. Analgesic responses are often measured through observations of pain behaviors like tail-flick or hind-paw shake latencies to a hot plate test.

While foot-shock consistently elicits reproducible pain behaviors in animals, forced swimming provides a larger spectrum of testing conditions to examine analgesia, specifically opioid versus non-opioid mediated responses. Tierney et al.(1991) found that

short bouts of swimming in room temperature water (15 seconds) induced non-opioid mediated analgesia lasting for 10-12 minutes (Tierney et al., 1991). In contrast, longer periods of swimming resulted in opioid analgesia 25-30 minutes after the swimming bout; after three minutes of swimming, endogenous opioid analgesia inhibits the expression of naloxone insensitive analgesia (Tierney et al., 1991). Cooper and Carmody (1982) replicated these findings and extended the research to determine the effects of water temperature on pain behavior. Analgesic responses were apparent after swims as short as 15 seconds, where swims of up to 7.5 minutes showed a progressive increase in the magnitude of analgesia (Cooper and Carmody, 1982). Analgesia was observed in mice that swam in water equivalent to their own body temperature (38°C); when the water temperature was decreased to 21°C, pain threshold increased marginally. Successive drops in water temperature paralleled that of declines in body temperature, both of which were inversely related to the relative pain threshold (Cooper and Carmody, 1982).

Examining the effects of extreme temperature, Giradot and Holloway (1984) swam rats at varying duration rates in 2° C water to induce SIA. Overall results revealed correlations between certain conditions of the cold water stressor and underlying mechanisms of analgesia (Giradot & Holloway, 1984). They measured these analgesic effects through responses to naltrexone, a long-term opiate antagonist. Naltrexone was found to partially block 3.5 minute cold water swim analgesia, significantly block intermittent cold water swim analgesia, and enhance analgesia after 60 separate one second exposures (12/min) (Giradot & Holloway, 1984). Thus, specific parameters of the cold water swim selectively activated either non-opioid or opioid mediated analgesia

Although this is a limited overview of the animal literature, it does illustrate the range of stressors that can induce SIA. It is likely that human and animal stressors share commonalities in characteristics such as novelty, unpredictability, and physical demand. Humans however, can offer insight as to why specific conditions or stimuli may be stressful—a powerful tool for researchers studying SIA.

~Human Stress Models

Research in both animals and humans has demonstrated that the type of stressor, individual differences, and method of pain measurement can have modulatory effects on the analgesic response. Various classes of stressors have been shown to induce analgesia in humans: psychological stress, exercise, painful stimuli, fighting, thermal stress, sexual arousal, and athletic competition.

In studying SIA in the lab, researchers are often confronted with the problem of contrived or unrealistic stressors, particularly in laboratory simulations of fear and anxiety. To avoid this problem Janssen and Arntz (2001), examined real-life stress in novice parachute jumpers. Results revealed both pre-jump and post-jump opioid mediated analgesia, measured by pain behavior responses to electrical stimulation and pressure (Janssen & Arntz, 2001). β-Endorphin was used as a measure of opioid activity, and was elevated only immediately after the jump, which supports the correlation between anxiety reports and increased pain inhibition. This study not only measures a real-life stressful situation, but also raises further questions about the role of pre-anticipatory anxiety/stress in activating endogenous opioid mechanisms.

Psychological stress in the form of a standard mental stress test has been found to induce SIA. Smoking has also been found to induce analgesia through stimulation of the HPA axis, specifically in increased cortisol and β-endorphin output (Girdler et al., 2005). A study investigating sex differences in SIA and "smoking-related analgesia" as a function of pain behavior, revealed that both female and male smokers had higher pain thresholds than did non smokers to ischemic and cold pressor pain, respectively (Girdler et al., 2005). Only females demonstrated SIA in response to the mental stress test (TSST-The Trier social stress test) (Girdler et al., 2005). Paradoxically, male and female smokers showed an inverse relationship between analgesia and HPA-axis function (Girdler et al, 2005). In response to the TSST, smokers demonstrated lower β-endorphin and ACTH concentrations than did non-smokers, which may suggest that chronic activation of the HPA-axis leads to down regulation of the system (Girdler et al., 2005). The study demonstrates that sex and the parameters of the noxious stimulus (pain measures) variably influence pain sensitivity in smokers and non-smokers, as well as SIA (Girdler et al., 2005).

~Exercise-Induced Analgesia in Humans

As demonstrated in animal models, the stressful act of physical exertion in swimming can reduce pain sensitivity. Similarly in humans, personal anecdotes from athletes who suffer injuries during high physical activity report lower pain sensitivity. The human literature on exercise-induced analgesia indicates that both opioid and non-opioid mediated mechanisms can produce analgesia. Which of these systems is activated depends on the type and intensity of the exercise stressor and pain measurement used.

Three classes of physical activity have been extensively studied: aerobic (cycling and running), resistance, and isometric exercise.

A number of issues have been raised concerning exercised-induced analgesia as a potential artifact of the testing procedure, specifically in that stress-induced analgesia may be induced by the pain testing itself. Padawer and Levine (1992) critically examined exercise-induced analgesia, and found significant effects for pain test pre-exposure resulting in analgesia using the cold-pressor test. No significant results were reported however, for analgesia following bicycle ergometry performed at 50 and 70% of maximum heart rate (Padawer and Levine, 1992). A follow-up study by Droste and Greenlee assessed whether exercise intensity played a role in exercise-induced analgesia. Previous research by Kemppainen found that exercise at 74% of maximal aerobic activity produces analgesia, suggesting that perhaps Padawer and Levine's mild exercise condition prematurely discounted exercise-induced analgesia. EIA has also been observed in male endurance athletes where significantly reduced pain sensitivity occurred after running (Fuller & Robinson, 1993). Janal et al. (1984) found EIA in long distance runners after a run of 6.3 miles, working at 85% of maximal aerobic activity. Pain sensitivity was attenuated in thermal and ischaemic tests, but not cold pressor pain (Janal et al., 1984).

Other forms of exercise, such as isometric activity have also provided evidence for exercise-induced analgesia (Koltyn, et al., 2000). After maximal and submaximal bouts of isometric exercise, pain thresholds increased in women (Koltyn et al., 2000). In contrast, pain thresholds increased only after maximal isometric exercise in men (Koltyn et al., 2000). Overall, females demonstrated a more consistent analgesic response to

isometric exercise in comparison to males, who only manifested analgesia in response to specific criterion of the exercise condition (Koltyn et al., 2000). These results suggest the existence of sex differences in pain inhibition; to date this issue has not been studied extensively enough to distinguish what these sex disparities may be. The exercise literature consistently demonstrates that exercise-induced analgesia does exist, but occurs more reliably after strenuous periods of extended exercise. It is probable that the HPA-axis activation occurs only after a prolonged period of physical activity, and as a result, analgesic responses may not be detected under certain testing conditions.

~Athletic Competition-SIA

There is a surprising paucity of research investigating pain behaviors of athletes during competition. Competition involves physical exertion, unpredictability, lack of control, and perhaps most importantly, is a naturally occurring condition. Thus, it is logical that athletic competition should induce SIA. The competition paradigm also has interesting applications in studying stress responses of hierarchically arranged social societies. Mazur proposes in his theoretical model of competition, that gaining, maintaining, and losing status involves both dominance and stress (Mazur, 1985). Under this model, it is clear why natural selection favored those individuals who were more capable of dealing with stress and could use such to their advantage. This model still retains its relevance presently, particularly in contrived competitive situations such as athletics. The same underlying mechanisms for stress exist in athletic competition, making it an ideal topic to study.

Two studies in particular have shed some light on this area, specifically on the effects of competition on pain perception, and sex-dependent aspects of competition-induced analgesia. A study conducted by Sternberg and colleagues (1998) investigated pain perception in athletes (track runners, basketball players, and fencers) and in non-athletes across three testing conditions. Subjects were tested two days prior to, during (immediately after the competitive bout), and two days following participation in an athletic competition. Supra-threshold pain and pain threshold were collected from loci on the forearm and fingertips, using the cold-pressor test and withdrawal latencies from thermal stimuli, respectively. Competition was found to significantly attenuate pain report on the cold-pressor test (Sternberg et al, 1998). Using heat withdrawal latencies, pain threshold increased in the arms, but decreased in the fingers in response to competition (Sternberg et al, 1998). These results indicate that competition can produce analgesic or hyperalgesic states depending on the body region tested and pain measure used. These findings support competition-induced analgesia, but are limited by the fact that analgesia may be a product of exercise rather than competition, since testing followed physical exertion. Despite this limitation, Sternberg and others suggest that results from pain intensity and unpleasantness ratings, as well as self-reported stress and anxiety levels, indicate that the psychological state associated with competition is competition-specific effect.

A follow-up study sought to dissociate competition-specific effects from exercise-only effects, by comparing competition in a non-exercise condition to competition in an exercise condition using both athletes (track runners) and non-athletes (Sternberg et al., 2001). The study defined competition as the cognitive stress associated with competing

against another individual or oneself, in the context of both athletic and sedentary (video-game playing) competition. Results replicated previous findings of competition-induced analgesia, but found notable sex differences in the exercise condition (treadmill running) and sedentary video game competition (Sternberg et al., 2001). After ten minutes of maintaining 85% maximal heart rate, only women demonstrated analgesia, but not men; whereas video game playing induced analgesia in men but not in women (Sternberg et al., 2001). These findings suggest that men and women respond differently to the psychological components of competitive activities, thus invoking varying analgesic effects. The existence of these sex differences can be explained using possible adaptive rationales like Taylor's "tend-and-befriend" model for the female behavioral stress response, and the established concept of "fight-or-flight." Under Taylor's model, a bout of video game playing may not evoke SIA due to the nature of the stressor. Whereas males may find the aggressive content of the auto-racing video game stressful, the absence of any meaningful relationships with the characters in the game may be a mediating factor for females. Perhaps the SIA response would be elicited if females engaged in a sedentary competition condition involving friends and intimate relationships.

The sparse literature on competition indicates that SIA can be produced by the competition stressor, at least in males, and is not simply due to exercise. Sex differences in analgesic responses support the notion that the nature of the stressor can differentially evoke pain inhibition. Specific aspects of the competition stressor may be less salient or personally relevant for women than for men, and vice-versa. Research investigating competition as a stressor indicates that SIA is not mutually exclusive to one gender.

Participation in sports for individuals is voluntary and thus personally relevant for both males and females; whereas contrived laboratory simulations like video-game playing may not be. These studies assume HPA-axis activation from analgesic responses, but do not directly measure these functions. Investigating stress hormones like cortisol is necessary to confirm that competition-induced analgesia is in fact a product of HPA activation.

~Anticipatory Stress in Competition

It has been well documented that cortisol, an essential component of the HPA axis stress response, plays a role in the physiological response to stressful stimuli—specifically in arousal and the mobilization of physiological resources necessary for an anticipated threat or challenge. As one of the primary hormones mediating the stress response, cortisol concentrations can provide biological evidence characterizing the physiological and psychological states of athletes prior to competition. In measuring cortisol levels before competition, increased cortisol and by extension pain inhibition, can be attributed to the competitive mindset but not necessarily to exercise. These elevations may not be initially detectable due to the latency in cortisol response to stress. As a result of the complex cascade of events involved in the stress response, HPA-axis hormones do not act instantaneously. It follows logically that measuring anticipatory cortisol levels before competition will clarify the specific time interval during which athletes begin to mentally and physically prepare for this stressor.

Bateup et al. (2002) investigated cortisol levels one day before, just prior to, and immediately after women's rugby competitions. Prior to this study, the body of

competition research has largely been conducted using male competitors. Findings indicated a pre-game rise in anticipatory cortisol as well as an elevated post-game level, with the post game level being higher than the pre game level (Bateup et al, 2002). Although not entirely relevant to the present study, game fluctuations in cortisol were positively correlated to violations in player expectancies of the opponent's level (Bateup et al, 2002). These findings may have implications in anticipatory cortisol levels, indicating that the importance of a competitive bout or perceived ability of the opponent can influence cortisol levels.

Providing support for lack of sex differences in anticipatory cortisol, Suay and colleague's (1999) study of male judoists demonstrated this adaptive anticipatory rise in cortisol before athletic competition. Cortisol was measured in three sessions: judo competition, ergometry, and a control. Results were characterized by an anticipatory rise in cortisol in response to competition compared to the non-competitive and non-physical effort groups (Suay et al., 1999). The ergometry session did elevate cortisol levels, but not as significantly as the judo fight (Suay et al., 1999).

Applying Taylor's biosocial model of "tend-and-befriend", Kivlighan et al. (2005) recently conducted a study examining potential gender differences in cortisol response to competition. Overall, anticipatory cortisol levels increased in response to competition regardless of gender or athletic experience (Kivlighan et al., 2005). Interestingly, athletic experience had a modulatory effect, wherein athletes with less experience had more elevated anticipatory cortisol levels than did those with more experience. Behavioral and attitudinal measures were administered to assess individual differences in dominance, competitiveness, and team bonding. These measures revealed

that higher anticipatory cortisol levels in females were not associated with competitiveness or pre-competition mental preparation, but instead related to bonding and social affiliation with teammates (Kivlighan et al., 2005). These results parallel other hormonal correlates of competition—namely in reports that pre-competition testosterone in females is significantly correlated to teammate ratings and subjective reports of social connectedness with teammates (Edwards et al., 2006). Males show the opposite effect, in which pre-event elevations of cortisol levels have been correlated to increased competitiveness and pre-race mental preparation (Kivlighan et al., 2005). Additionally, the increase of testosterone during competition, but *not* before, has been associated with teammate ratings and social connectedness with teammates (Edwards et al., 2006). Despite studies indicating analogous physiological patterns of anticipatory cortisol to competition in both males and females, psychological factors associated with gender differences appear to differentially effect the subsequent behavioral responses and motivations activating the HPA axis.

Though plasma cortisol levels are somewhat latent with respect to initial exposure to the stressor, studies have found elevated cortisol prior to competition. Using male judo athletes, Salvador et al. (2003) sought to specify the precise timing of HPA activation, by measuring cortisol at various time increments prior to competition. Cortisol increases have been reported 10-15 prior to the competitive activity and approximately 1 hour before the event, whereas many studies make no mention of this specification at all (Suay et al., 1999; Kivlighan et al., 2005). With the variation in anticipatory cortisol sampling, Salvador et al. took two separate samples prior to the competitive event to clarify the timing in HPA activation. Results demonstrated an overall rise in anticipatory cortisol,

and revealed a progressive decrease as competition approached; samples collected 30-40 minutes before the event were higher in cortisol than those taken immediately prior to the competitive event (Salvador et al., 2003). As a result, it appears that cortisol increases some time before the competitive event, and declines as it approaches. These results are somewhat contradictory in that cortisol plays a role in pre-competition preparation; however, one possible explanation may be a last minute attempt to conserve resources. The observed decline in cortisol immediately before competition may be alternatively explained by the notion of facilitators and debilitators (Eubank et al., 1997). A facilitator is a personality type defined by positive perception of anxiety in relation to performance, whereas debilitators perceive their anxiety as detrimental to performance (Eubank et al., 1997). Findings revealed that debilitators exhibited elevated cortisol levels as the event approached, whereas facilitators had comparatively lower levels (Eubank et al., 1997). Thus, it may be possible that the judo athletes who were tested, collectively resembled the characteristics of debilitators more than facilitators. Individuals who have lower cortisol may also indicate resilience to stressful situations, ultimately resulting in a superior competitor.

Rationale Behind the Current Study

Compared to the depth of pain literature, competition research is relatively understudied. The predominant work in competition has focused on two main areas—analgesia and hormonal responses. The present study intends to merge these two distinct bodies of research, by examining the effects of anticipatory stress on pain perception in male and female athletes. Consistent findings that cortisol levels increase in anticipation

of competition suggest that competition is a valid stressor that can activate HPA function, and induce analgesia. With respect to competition-induced analgesia, we intend to address some of the unanswered questions in the literature concerning exercise-effects and sex differences (Sternberg et al., 1998, 2001). More specifically, by collecting data prior to athletic competition, we can disregard any potential exercise related effects. To further probe potential sex differences in competition-induced analgesia, we will test elite male and female athletes, for whom athletic participation is assumed to be a personally meaningful activity. We intend to replicate earlier findings that competition can modulate the degree and direction of pain threshold depending on the body region tested (Sternberg et al., 1998).

The study will extend earlier research conducted in competition and pain, by investigating the comparative effects of routine athletic practices to game situations. A pilot study suggests the possible relationship between anticipatory cortisol levels and intensity level of the competitive event (practice v. game). Results indicated a number of trends: cortisol increased in anticipation of a game compared to a practice condition and was elevated after both practice and game conditions in relation to "pre" levels (Fig.10). Overall there was a larger cortisol response to game condition in both pre-game and post-game conditions (Fig.10). The relative increase in cortisol post-game compared to pre-game was likely the result of "in-game" latency of cortisol levels.

To demonstrate the relationship between anticipatory stress and pain thresholds in athletic competition, collegiate soccer and basketball players will be tested. The two types of athletes participate in similar competitive activities. The sport of soccer demands mental acuity, team cooperation, physical strength and endurance, and tactile decision-

making; whereas, the sport of basketball involves skill, speed and power, team cooperation, and quick decision making. Both male and female soccer players will be tested to examine potential sex differences. When initial data analyses indicated no significant sex differences, female basketball players were selected to control for the influence of ambient temperature on nociceptive responsiveness.

Data collection will occur just prior to a competitive match and routine practice session and immediately after a short warm-up. These measurements will include pain threshold readings from foci on the forearm and fingertips, salivary cortisol levels, self-reports of body awareness, physiological assessments, ratings of anticipated physical exertion, and game importance scores. In order to control for the minimal effects of exercise during pre-game and practice warm-up, our baseline condition will consist of a five minute bout of cycle ergometry to simulate equivalent physiological conditions before the practice and game conditions.

~Hypotheses

Based on the literature that stress-induced analgesia is exhibited immediately following competition, we expect to find anticipatory analgesia for the same reasons that competition induced analgesia is observed post-competition. If indeed competition-induced analgesia is not simply a result of exercise, a routine practice should induce elevation of anticipatory cortisol and analgesia; generally, practice sessions involve a less intense form of game-day competition, yet still include substantial physical exertion, competition, and an element of the unexpected. As a result, we hypothesize that practice will induce anticipatory analgesia, but to a lesser extent than a competitive game**.** Based

on Sternberg et al.'s study (2001), we expect to see sex differences in practice stress and analgesia. This disparity may result in two possible outcomes: 1) elevated stress and pain inhibition in females but not in males, since females show analgesic responses following mild physical exertion; or 2) vice-versa, since it has been shown that males are more competitive and may thus approach practices more competitively (Koltyn et al., 2000; Kivlighan et al., 2005).

One study investigating pain thresholds in athletic competition using thermal stimuli, obtained results suggested that the degree and direction of analgesia is contingent upon the body region tested (Sternberg et al., 1998). We believe that anticipatory analgesia will exhibit these same effects, in which the pain thresholds will increase on the volar surface of the forearm, but decrease on the fingertips (Sternberg et al., 1998). Due to the inherent disparity between the conditions (practice v. game), it is anticipated that analgesic states will be more pronounced just prior to a game, than before a practice and baseline. Furthermore, pain threshold and cortisol will be greater pre-practice than during baseline testing.

Research conducted on competition and cortisol, support two contrasting bio-behavioral models, namely 'fight-or-flight' and 'tend-and-befriend'. These theoretical behavioral models of the stress response, suggest that gender differences exist in the psychological components of the stressor as well as the resulting behavioral responses to the stressor. In particular, Koltyn (2000) has noted one such sex difference, where females respond more to the exercise component of the competition, and males may respond more to the competition itself. Under these precedents, it is possible that sex differences in anticipatory cortisol and analgesic responses may emerge; however, if the

competitive activity is equally engaging and important for both sexes, these cognitive discrepancies may not be observable in the physiological data. Since our measurements do not include cognitive assessments of pre-game and practice mental states, these contrasting internal states cannot be confirmed.

Methods

~Subjects

Male and female soccer players (*n*=9 and 11, respectively) and female basketball players (*n=9)* were recruited based on his/her "starter status" and playing time for participation in this study with permission from the Haverford College Athletic Department (NCAA Division III).

~Algesiometry

Using the Medoc thermal stimulator, and the "method of limits", the thermal probe began at the subject's skin temperature (obtained at the time of testing) and increased incrementally (at rate of 1°C/sec) until the subject reported that pain threshold had been reached (on average between 41°C and 45°C for healthy subjects). Once pain threshold was reported, the device reset to the neutral temperature. The procedure was repeated 6 times at different loci (3x on forearm, 6x on fingertips) to create a stable baseline pain threshold. Thermal stimuli were delivered by a 30 x 30 mm Peltier thermode (Medoc TSA2001) placed on the volar surface (inner, hairless region) of the forearm or on the fingertips (middle three fingers of each hand) that began at the subject's measured skin temperature. Subjects were instructed to indicate when the stimulus turned from heat to pain, after which the stimulus was terminated. The probe

was shifted to a different location on the forearm or finger, and repeated for a total of 6 trials. In order to practice making perceptual judgments about thermal stimuli, subjects were first exposed to a "warm threshold" procedure, wherein the thermode began at 20°C and increased in temperature until the subject first detected (and reported) warmth, during the initial orientation session (no data collected).

~Saliva Collection and Cortisol Assay

To measure the hormonal changes (cortisol) that may accompany the stress response in anticipation of competitive situations, 500 ml of saliva was obtained from each subject on all testing days by having the players chew on a 2x2 inch square of sterile cotton gauze for approximately 30 seconds. On competition day, samples were collected approximately 5-20 minutes between warm-up and game time for soccer players, and 35-45 minutes between warm-up and game time for basketball players. In the practice condition, samples were collected immediately after a brief warm-up but before practice. Baseline samples were obtained at the same time of day as the collection of pre-game and pre-practice samples after a brief bout of cycle ergometry. Saliva samples were stored at 4°C until assay.

All samples were assayed in the laboratory for salivary cortisol (μg/dl*)* using an EIA kit obtained from DSLabs (Arlington, Texas). The test uses only 25 μl of saliva (for singlet determinations). Absorbance was read from a 96 well plate on a plate reader (450 *nm* absorbance filter). A standard curve was constructed using known cortisol values, to determine absorbance values for each unknown sample.

~Game Importance

We asked subjects to rate the meaningfulness of the game condition in which they were about to participate in on a scale of 1-10, ten being the highest (see Appendix II).

~Body Awareness Questionnaire

Subjects at each of the three experimental sessions provide self-report measures on the Somatic Symptoms Questionnaire (which assesses measures of sympathetic nervous system activation—sweaty palms, butterflies in stomach, heart racing, etc.). See Appendix 1.

~Intensity

We asked subjects to rate the expected intensity level of physical exertion prior to game and practice conditions on a scale of 1-13**,** thirteen being the highest (see Appendix II).

~Procedure

Male and female participants were tested in three different conditions in counterbalanced order, in addition to attending an orientation session prior to the first testing session. The primary goal of the orientation session was to acclimate subjects to the pain testing apparatus and allow them to practice making perceptual decisions about the transition of thermal stimuli. Athletes were tested on three separate occasions: (1) just prior to a NCAA game (immediately after warm-up and 5-15 minutes before game time for soccer players, and 35-45 minutes after warm-up and before game time for basketball players); (2) just prior to a routine practice session (but after pre-practice warm-up); and (3) on a baseline day (immediately after subjects completed five minutes of cycle ergometry to induce equivalent physiological states prior to practice and game conditions). In all three conditions, subjects were first tested for pain withdrawal

latencies at six loci on both the forearm and the fingertips, after which body awareness questionnaires were completed. Heart rate and blood pressure were recorded and salivary cortisol samples collected. In the game condition, subjects were also asked to complete the game importance questionnaire. Subjects were randomly assigned to counterbalanced order of session presentation to control for repeated testing effects. All testing sessions were conducted within one week of the initial orientation session.

Data Analysis

Competition-induced effects of soccer players were determined by mixed design analysis of variance (ANOVA) with sex as a between-subjects factor, and the three testing conditions as the repeated measure (day). Significant F-values were followed by Fisher least significant difference (LSD) post hoc tests where necessary. The level of significance for all tests was established at $P \leq 0.05$. Analysis of competition-induced effects of basketball players was replicated without sex as a between-subjects factor. Results were determined by an ANOVA repeated measure test to determine the effects of testing day on withdrawal latencies, cortisol, body awareness, intensity, physiological measures, and skin temperature. All subjects were counter-balanced for effects of novelty and repeated measure effects. An initial between-group analysis of sport failed to indicate any significant differences, compelling us to analyze each sport separately.

Results-Experiment 1 (Soccer)

~Physiological Measures

A main effect of day was observed on systolic blood pressure ($F[2, 34]= 4.554$, $P=0.018$). Participating in a game significantly increased systolic blood pressure

compared with the practice and baseline conditions (Fig 1). A Fisher LSD post-hoc test revealed that systolic blood pressure on the game day was significantly higher than on the baseline day (P= .008) and practice day (P= .02), which were not significantly different from one another as shown in Table 1. No day x sex interaction or overall main effect for sex reached significance.

A main effect of day was also noted on diastolic blood pressure (F[2, 34]= 33.437, P<.0001). Like SBP, diastolic blood pressure was significantly higher on game days compared to practice (P= .001) and baseline (P<.001). A Fisher LSD post-hoc test also indicated a significant elevation on DBP from baseline to practice (P= .04). A day x sex interaction was observed (F[2, 34]= 3.579, P=.039) wherein DBP was significantly different for all three days (baseline< practice< game). Males had higher diastolic blood pressures in practice compared to baseline (P=.002); whereas female DBP was not significantly elevated from baseline to practice (Fig. 1).

A main effect of day was observed for heart rate (F[2, 34]= 17.647, P<0.0001), which was significantly elevated from baseline to practice (P= .03), baseline to game (P<.001), and from practice to game (P= .001) (Fig. 1). The day x sex interaction and overall main effect for sex were not significant.

~Body Awareness

A main effect of day was noted in body awareness and arousal (F[2, 36]= 9.910, P= 0.0001), wherein game (P< .001) and practice (P= .02) scores were significantly higher than baseline reports (Fig 2). Post-hoc tests also indicate a trend where game arousal was slightly larger than that of practice (P= .058). There was no significant day x sex interaction.

~Intensity

Significant effects of athletic competition were observed on the intensity ($F[1, 14]= 59.67$, $P<.001$) scale with subjects reporting higher expected intensity on game ($P=.004$) day compared to practice ratings (Fig 3).

~Withdrawal Latencies

Withdrawal latency at the forearm loci was calculated by averaging across the different placements of the probe and subtracting the final temperature perceived as painful from the starting skin temperature. The arm difference equals the number of degrees it took to reach pain threshold. A significant main effect of day was apparent for arm difference ($F[2, 36]= 20.608$, $P<0.0001$) as shown in Table 1. Pain threshold in the game ($P< .001$) and practice ($P= .001$) sessions were observed to be significantly higher than that of baseline, but game pain threshold was not significantly different from practice (Fig. 4). Similarly, finger withdrawal latency had a significant main effect of day ($F[2, 36]= 21.887$, $P<.0001$). Practice ($P< .001$) and game ($P< .001$) pain threshold were significantly higher than baseline, but no significant difference was observed between game and practice (Fig. 5). In both arm and finger withdrawal latencies, pain threshold increased from baseline to practice and game, indicating a significant decrease in pain perception before both athletic competition and team practices.

~Skin Temperature

A main effect of day was observed for skin temperature ($F[2, 38]= 27.645$, $P<.001$). Game ($P<.001$) and practice ($P<.001$) skin temperatures were significantly lower than baseline (Fig 6). No significant differences were noted between game and practice.

~Game Importance and Pain Threshold-Arm

A significant correlation between game importance and arm withdrawal latency was found (r[12]= .593, P= .025), indicating that games rated more highly in importance were significantly related to higher arm pain thresholds (Fig 7). The correlation between finger difference and subjective ratings of game importance approaches significance (r[12]= .488, P= 0.077). This finding supports our hypothesis that stress of competition is associated with pain sensitivity with no alternative explanation.

~Cortisol

A significant main effect of day was apparent for cortisol (F[2, 22]= 5.4, P=.012). Salivary cortisol concentration prior to competition was significantly higher than baseline (P<.001) and practice (P<.001) concentrations (Fig. 8).

~Sex Differences

No significant results were observed for gender differences across day, skin and finger differences, body awareness, and physiological measures.

Discussion-Experiment 1

The results of this study support previous findings that athletic competition can induce analgesia and an anticipatory rise in cortisol. Thermal pain threshold significantly increased on both the volar surface of the forearm and fingertips prior to participation in a game and practice compared to baseline difference values. Research on competition induced analgesia has shown evidence that competition can produce analgesic or hyperalgesic states depending on the body region tested and pain measure used. This finding was not replicated in the current study however, which may be a result of a the

anticipatory response to competition and the absence of an EIA confound (Sternberg, 1998). No significant difference in pain threshold (as measured by withdrawal latencies) was found between game and practice, suggesting that cognitively these two situations are equally stressful and physiologically arousing. The significant increase in cortisol just prior to a game but not to practice is consistent with the hypothesis that athletic competition can initiate a SIA response. The absence of a practice elevation in salivary cortisol is not reflected in the pain data, where we did observe a significant elevation in pain threshold from practice to baseline. Though both conditions are capable of inducing analgesia, this deviation from the cortisol data suggest alternative mechanisms by which competition and practice produce analgesia. Alternatively, pre-practice analgesia may be an effect of the testing procedure, as baseline skin temperature can impact heat withdrawal latencies. These effects of baseline skin temperature will later be addressed.

Results from physiological and subjective arousal measures (blood pressure, heart rate, and body awareness ratings) provide further evidence for athletes' pre-competition preparation. Systolic blood pressure was significantly higher on competition and practice day compared to baseline. Diastolic blood pressure showed a similar pattern of game day and practice day levels being significantly higher than baseline. Post-hoc tests of DBP revealed the only sex difference of the study, where males' DBP was significantly higher on practice than baseline day compared to females', who showed no significant variation in DBP between these conditions. Heart rate was significantly higher before competition compared to practice and baseline. Heart rate on practice day was also significantly higher than that of baseline; along with cortisol elevations, these are the only sources of physiological evidence that competition is more arousing than practice. Whether this

result is an artifact of exercise intensity differences (during warm-up) among all three conditions or variability in physiological preparedness cannot be determined from the current study. Results from the Somatic Symptoms Questionnaire, a subjective measure of sympathetic nervous system activation, indicate that reported arousal was significantly higher prior to participating in a game and practice compared to baseline scores. These subjective scores of arousal are consistent with the physiological findings; however the reliability of this inventory is not well documented in the competition and pain literature.

Although our pain data are not entirely consistent with our initial hypothesis that proposed a significant elevation from practice to game day, our cortisol data suggest that practice and game are qualitatively different stressors. It should be noted however, that our testing procedure for pain threshold (heat withdrawal latencies) is susceptible to baseline skin temperature (Wu et al., 2001). If skin temperature was reduced during competition, a false analgesic response could occur as a result of increased withdrawal latency from stimulus onset to conclusion. Even at a lower starting temperature thermal pain threshold will remain unchanged, resulting in a longer heat withdrawal latency and thus a higher pain threshold. Over the course of testing there was precipitous drop in outside temperature, resulting in a significant decrease in skin temperature in both practice and game conditions as compared to baseline sessions (conducted indoors). This parallel decrement creates a potential confound. Wu et al. (2001) found that a decrease of 4°C in the baseline temperature below resting skin temperature resulted in a significant increase in the stimulus needed to reach a subjective pain rating of 5 out of 10. Despite evidence that pain threshold measures (heat withdrawal latencies) are sensitive to baseline skin temperature; it is likely that these effects are minimal in light of the

physiological and cortisol data that support competition as a valid stressor capable of eliciting CIA.

Our interpretation of the data suggests that the effects of baseline skin temperature are indeed "minimal," as game importance scores are significantly correlated to arm withdrawal latencies, where higher subjective importance ratings are associated with greater analgesic effects. Higher game importance ratings suggest greater expected physical exertion and personal investment in the outcome, making the competitive bout more stressful, and subsequently inducing greater analgesic effects. Unlike longer withdrawal latencies (higher pain thresholds), which may simply be a product of lower starting skin temperature rather than stress induced analgesia, the correlation between game importance and arm pain threshold bears no relation to skin temperature. Regardless of starting skin temperature, higher rated game importance is correlated to reduced pain sensitivity.

Results from soccer players were consistent with our hypothesis that anticipation of athletic competition, both in game and practice settings, is a valid stressor that is capable of inducing analgesia. However, without further investigation into the effects of baseline skin temperature on pain threshold, we cannot assume the causative relationship of competition producing analgesic responses. As a result, a follow-up study was conducted where all three conditions took place in an indoor environment to rule out this temperature confound. With no significant fluctuations in ambient temperature across conditions, any significant results we observe will not be attributable to fluctuations in skin temperature. Our next round of subjects were recruited from the Haverford College women's basketball team. Since there were no sex differences in anticipatory cortisol

levels or pain thresholds in experiment one, inclusion of male subjects was considered unnecessary.

Results-Experiment 2 (Basketball)

~Physiological Measures

No significant main effect of day was observed on systolic ($F[2,14]= 2.61$, $P=.11$) or diastolic blood pressure ($F[2,14]= 1.28$, $P=.31$). Though these data not reach statistical significance, calculated means do show a trend where systolic blood pressure on the game day was higher than on the baseline day and practice day (Fig 9).

A main effect of day was observed on heart rate ($F[2,14]= 12.30$, $P=.001$), which was significantly elevated from baseline to practice ($P= .003$) and baseline to game ($P<.001$) as shown in Table 2 (Fig. 9).

~Body Awareness

No significant main effect of day was noted on body awareness and arousal ($F[2,14]= 0.98$, $P=.40$).

~Intensity

Significant effects of athletic competition were observed on the intensity ($F[1, 8]= 16.67$, $P=.004$) scales with subjects reporting higher expected intensity on game ($P=.003$) day compared to practice ratings (Fig 3).

~Pain Latencies

As in Experiment 1, withdrawal latency at the forearm loci was calculated by subtracting the final temperature perceived as painful from the starting skin temperature. The arm difference equals the number of degrees it took to reach pain threshold. A

significant main effect of day was apparent for arm difference ($F[2, 16]= 12.71$, $P<.001$). Pain threshold in the game ($P< .001$) and practice ($P= .001$) sessions were observed to be significantly higher than that of baseline, but game pain threshold was not significantly different from practice (Fig. 4). Similarly, finger withdrawal latency had a significant main effect of day ($F[2, 16]= 5.44$, $P<.05$) as shown in Table 2. Practice ($P=.012$) and game ($P=.011$) pain threshold were significantly higher from baseline, but no significant difference was observed between game and practice (Fig. 5). In both arm and finger withdrawal latencies, pain threshold increased from baseline to practice and game, indicating a significant decrease in pain perception before competition.

~Skin Temperature

An unexpected effect of day was observed for skin temperature ($F[2, 16]= 26.86$, $P<.001$), despite efforts to keep ambient temperature consistent while testing at game and practice conditions. Game ($P<.001$) and practice ($P<.001$) skin temperatures were significantly lower than baseline (Fig 6). No significant differences were noted between game and practice.

~Game Importance and Pain Threshold

No significant correlations were found between game importance ratings and pain thresholds on the arm or finger. Despite the significant relationship between arm difference and game importance in soccer players, the absence of this finding in basketball players suggests an inter-sport difference in game importance ratings.

~Cortisol

No significant main effect of day was noted for cortisol (Fig 8). Three subjects were excluded from the assay due to human error and insufficient salivary volume.

Compared to the robust findings in male and female soccer players, the absence of a significant day effect may be explained by the small sample size (n=5) and/or irregularities that occurred during the cortisol assay.

Discussion-Experiment 2

Findings of competition-induced analgesia in female basketball players are consistent with those from male and female soccer players; both arm and finger heat withdrawal latencies significantly increased prior to participation in a game and practice compared to baseline values. No significant differences were reported between practice and game days. In experiment one, the cortisol and physiological data supported competition-induced analgesia in soccer players. This same data from basketball players makes these analgesic effects difficult to interpret. The only indication of increased physiological arousal prior to competition was observed in heart rate values, where game and practice values were significantly higher than baseline values. None of these same patterns were noted in blood pressure, nor were they apparent in subjective body awareness scores.

It may be possible that the physical activity (short warm-up before practice and game) and a five minute bout of cycle ergometry prior to baseline testing required differential levels of exertion for these two sports. Due to certain testing constraints during basketball warm-up, subjects were asked to warm-up by themselves prior to the "official" team warm-up. In contrast, soccer players were tested immediately after a short bout of team warm-up activity. These disparities may suggest that the warm-up period of basketball players was not physiologically and cognitively sufficient to generate the same

patterns of sympathetic activity as observed in soccer players, namely in elevated blood pressure and subjective body awareness scores. Other mediators of sympathetic activation may be associated with subjective mean scores of game importance and expected physical exertion during competition, which tended to be lower in basketball players than in soccer players.

A striking disparity between experiment one and two was the absence of a significant effect of day on cortisol levels in female basketball players. It should be noted however, that three samples were excluded from our assay (n=5), which in itself had some abnormalities. Nevertheless, these results suggest that pain threshold among female basketball players may not be modulated by the stress of competition. It is possible that the team dynamic had some effect on the perceived stress of competition, specifically if there was a lack of bonding and/or social affiliation among teammates (Kivlighan et al., 2005). Studies have shown that bonding and social affiliation among female teammates is associated with higher levels of anticipatory cortisol, suggesting that a lack thereof among basketball players would effect cortisol output (Kivlighan et al., 2005).

The apparent absence of HPA activation in basketball players may also be explained by disparities in the time interval preceding competition. NCAA warm-up regulations created some limitations for our testing procedures within each sport. Whereas soccer players could be tested 10-20 just prior to practice or game, the earliest testing could occur on basketball players was 35-45 minutes prior to game and practice. Past research on anticipatory cortisol has revealed inconsistencies in the reporting of collection interval, but also in the precise timing of HPA activation. One study noted an

overall rise in anticipatory cortisol, but found a progressive decrease as competition approached; samples collected 30-40 minutes before the event were higher in cortisol than those taken immediately prior to the competitive event (Salvador et al., 2003). Others have been reported an elevation 10-15 minutes prior to the competitive activity and approximately 1 hour before the event (Suay et al., 1999; Kivlighan et al., 2005). These findings are somewhat inconsistent with our results from study one, but also do not explain our null findings in study two.

Our original motivation for conducting experiment two concerned the baseline skin temperature confound we encountered while testing soccer players outside. Yet analysis revealed a significant, albeit lesser, effect of day on skin temperature in basketball players. Skin temperature on practice and game days was significantly lower than baseline values. If baseline skin temperature is the sole factor determining heat withdrawal latencies, it is likely that a significant difference in relative pain thresholds between sports would be revealed. Our data provide evidence against this theory, since no significant difference was found between sport in game pain thresholds despite a considerable difference in temperature from baseline to game between sports (a 5.4°C drop in soccer compared to a 2.8°C decline for basketball).

General Discussion

After data collection was completed from both studies, analysis was conducted with sport as a between-subjects factor and the three testing conditions as the repeated measure (day). Preliminary analysis from cortisol data indicated a significant sport by day interaction, compelling us to examine our data separately within sports. During this

initial data analysis, a significant correlation was revealed between arm withdrawal latency and game importance scores in both soccer and basketball players when the lone outlier score was excluded. This result is consistent with previous findings that competition can induce analgesic effects, and suggests that greater subjective importance attributed to competition can exert stronger analgesic effects (Sternberg, 1998; Sternberg, 2001). Past research of competition-induced analgesia has demonstrated robust sex differences in various conditions of competition both during and post-competition (Sternberg et al., 1998; Sternberg et al., 2001). Our study extends these findings to include the *anticipatory* effects of competition in producing analgesia. With the exception of diastolic blood pressure, no significant sex differences were observed in the current study. The lack of sex differences was somewhat surprising in light of the vast literature regarding sex differences in the stress response and SIA, but may be a product of the competitive stressor under examination.

Subjective assessments like game importance and anticipated physical exertion may offer insight into the cognitive basis for why competition is a stressor. Our data certainly point to these indices as important components of the stress response in modulating pain. Mean scores of expected intensity show game day to be rated as significantly more intense than practice in both basketball and soccer players (Tables 1&2). Notably, basketball players' scores of expected game day intensity were lower relative to soccer game day reports (Tables 1&2). A number of studies have examined the psychological underpinnings of why certain athletes outperform others, and more specifically what distinguishes elite from amateur athletes. Findings from a study of elite vs. non-elite swimmers indicated that elites engaged in more self regulatory thoughts and

behaviors prior to and during competition compared to non-elites (Anshel & Porter, 1996). How these cognitive coping strategies interact with physiological and nociceptive responses has not been extensively explored in the literature. Anshel and Porter's observation that male and female elite athletes have similar psychological characteristics and behavioral tendencies has interesting implications for the present study, as collegiate athletes compete nationally and should therefore be considered under this "elite" qualifier. Thus, the apparent lack of sex differences in our study may be partially attributable to some "elite" quality that male and female soccer players share.

Though skill level has been shown to play a role in the cognitive coping strategies adapted by athletes, how cognitive adaptations are translated into physiological responses is a future challenge for sports psychologists and scientists alike. An alternative route may be game outcome and/or athletic identity. It has been proposed that athletes who assume greater "athletic identity" and are more competitive may show greater pain tolerance, with investment in competitive activity playing a contributing factor (O'Connor, 1999). Indeed, others have shown that athletic identity is associated with win orientation and competitiveness (Tusak, et al., 2005). Those individuals with stronger athletic identity may have greater investment in competition, and therefore perceive these activities as more stressful. Perhaps, our basketball participants were less invested in their season or did not associate to a strong athletic identity compared to soccer players.

In characterizing between sport differences, specifically why cortisol and physiological arousal measures were not consistent across sport, it is likely that minute differences in experimental design played a role. Nonetheless, the absence of statistically significant findings from basketball body awareness indices is consistent with our null

result of the effect of day on blood pressure. It is well documented that sympathetic activity in the periphery requires sufficient time, physical activity, and/or stress to generate significant fluctuations, which is consistent with clinical evidence that chronic stress can result in hypertension (Sapolsky, 1992). Experimental error occurring during our cortisol assay makes it difficult to determine if in fact competition was a legitimate stressor for basketball players. We believe that competition was indeed stressful, but that CIA may not have been produced to the same extent as observed in soccer players. In experiment two, a conceptually inexplicable gap exists between our physiological and subject measures of stress to the observed analgesic response; such a disparity may be explained by the small sample size (n=5 for cortisol; n=8 for all other measures), and greater individual variation in physiological arousal and cortisol levels.

The present findings provide evidence for the theoretical link between stress and pain in athletic competition. The literature on CIA argues that competition is capable of inducing analgesia effects, whereas stress research has consistently demonstrated that athletic competition elicits anticipatory and post-event elevations in cortisol. This study demonstrates the psychological and physiological association between pain and stress interactions in anticipation of competition. While our initial research concerning this relationship looks to be promising, in-game and post-competition patterns must undergo further comparison. Additional research on sex differences in both team oriented and individual sports will advance our understanding about the cognitive basis underlying competitive athletics and its capacity to induce stress and analgesia. Moreover, future research is necessary to support our observation of anticipatory analgesia in response to competition, and the specific time interval during which this system is activated.

Table 1- Soccer (M & F)

Variable	Baseline (N=20) Mean SD	Practice (N=20) Mean SD	Competition (N=20) Mean SD
Systolic BP (mmHG)	135.89±5.21	139.66±5.57	156.27±3.99
Diastolic BP (mmHG)	78.81±2.54	85.90±2.76	101.71±2.20
Males (N=9)	74.62±4.22	89.25±4.27	102.50±3.54
Females (N=11)	81.81±2.43	88.22±2.28	99.00±1.94
Heart Rate (Bpm)	68.33±2.46	76.40±2.90	87.40±3.45
Body Awareness	33.33±0.99	36.31±1.61	38.46±1.13
Intensity (N=17)		6.13±066	11.93±0.37
Skin Temperature (°C)	31.43±0.22	26.29±0.90	26.03±0.83
Arm Difference (°C)	11.39±0.76	15.91±0.99	16.16±1.21
Finger Difference (°C)	14.41±0.91	18.85±1.12	19.64±1.28
Cortisol (µg/dl)	0.269±0.08	0.270±.07	0.0467±0.10

Table 2- Basketball

Variable	Baseline (N=9) Mean SD	Practice (N=9) Mean SD	Competition (N=9) Mean SD
Systolic BP (mmHG)	128.25±7.65	143.50±8.30	144.25±6.40
Diastolic BP (mmHG)	87.4±5.08	95.0±3.06	92.9±3.50
Heart Rate (Bpm)	63.1±3.4	74.1±2.1	77.5±2.9
Body Awareness	31.7±1.1	31.8±1.3	33.1±1.2
Intensity		6.4±0.69	9.8±0.62
Skin Temperature (°C)	31.71±0.42	28.79±0.21	28.92±0.20
Arm Difference (°C)	13.0±0.85	16.1±0.73	16.6±0.45
Finger Difference (°C)	16.2±0.67	18.4±0.70	18.5±0.70
Cortisol (µg/dl) (N=6)	0.493±0.16	1.105±0.25	0.852±0.16

Figure 1.

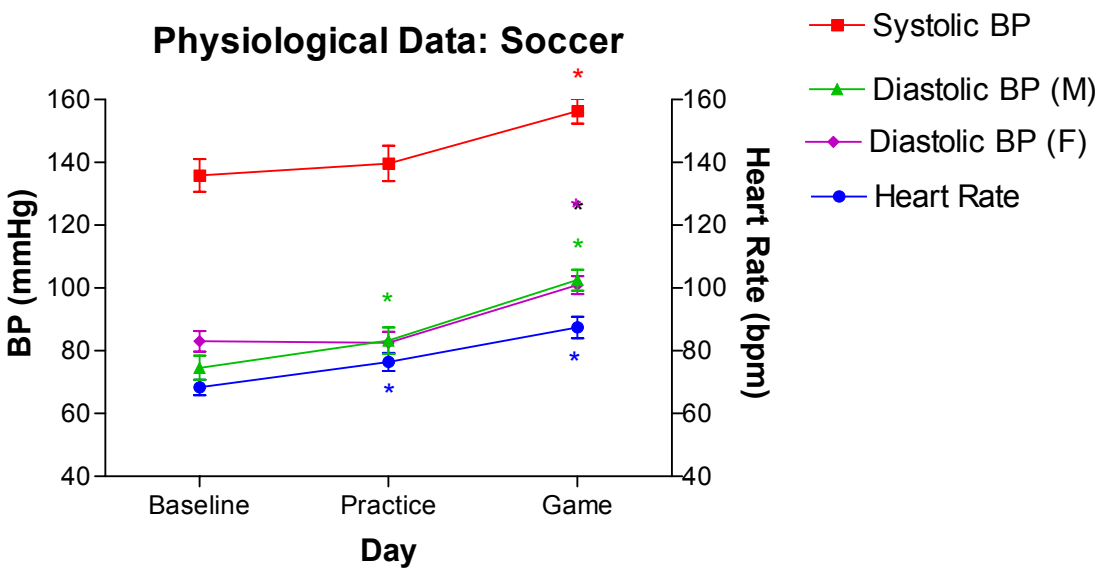

Figure 1. Physiological measures for systolic and diastolic blood pressure, and heart rate on baseline, practice, and game conditions. * Systolic BP was significantly elevated pre-game day compared to pre-practice and baseline values in both sexes. * Diastolic BP in males was significantly elevated from practice to baseline and higher on game day compared to both practice and baseline. * Diastolic BP in females was significantly higher on game day compared to practice and baseline. * Heart rate was significantly elevated from practice to baseline, and higher on game day to practice and baseline days.

Figure 2.

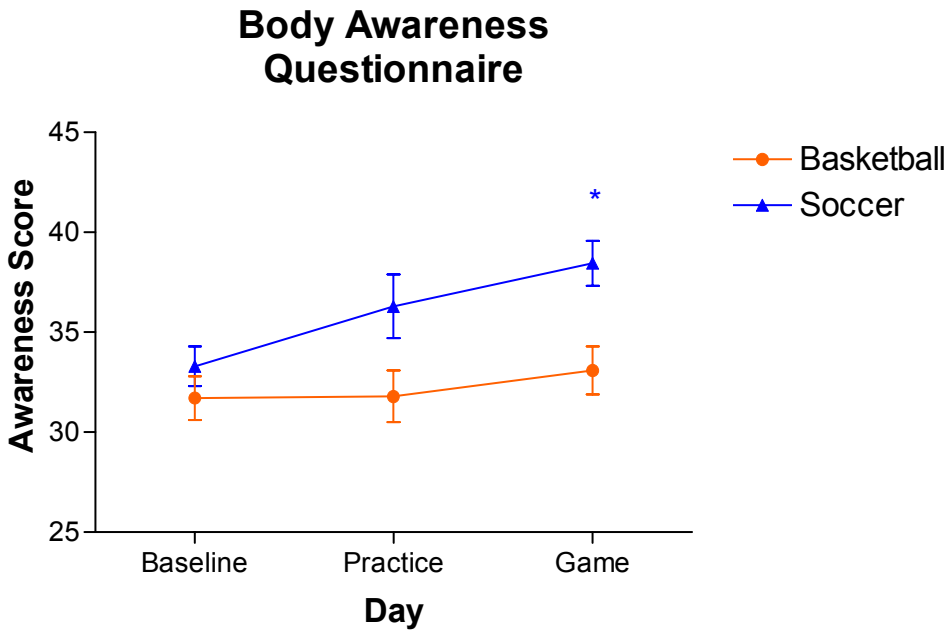

Figure 2. Body awareness scores of athletes across baseline, practice, and game conditions. * For male and female soccer players body awareness scores were significantly higher prior to game day compared to pre-practice and baseline reports.

Figure 3.

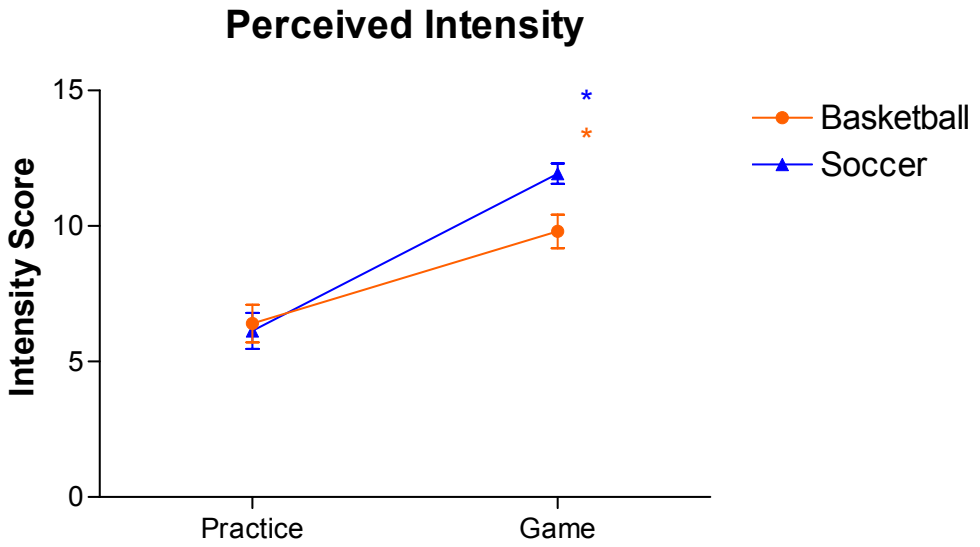

Figure 3. Perceived intensity (of anticipated physical exertion) scores of athletes prior to practice and game conditions. * For male and female soccer players perceived intensity scores were significantly higher prior to game day compared to pre-practice scores. * For basketball players perceived intensity scores were significantly higher prior to a game than before a practice.

Figure 4.

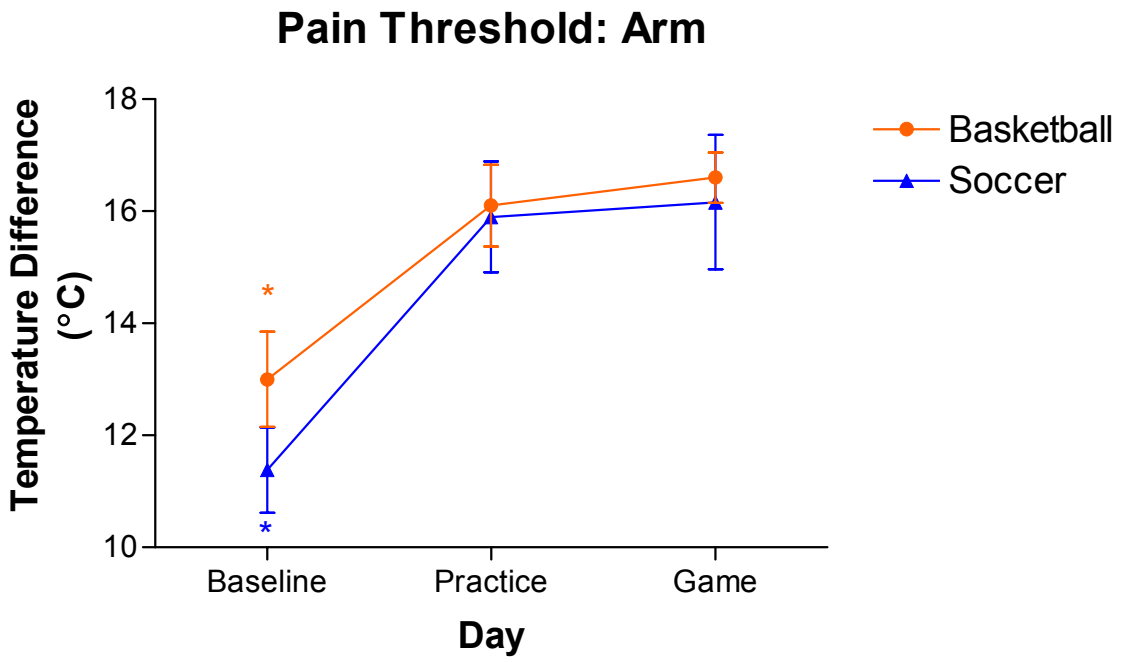

Figure 4. Withdrawal latencies on foci of the forearm of athletes participating in baseline, practice, and game conditions. * Significant elevations in pain threshold prior to practice and game days compared to baseline day in female basketball players. * Significant elevations in arm pain threshold prior to practice and game days compared to baseline day in male and female soccer players.

Figure 5.

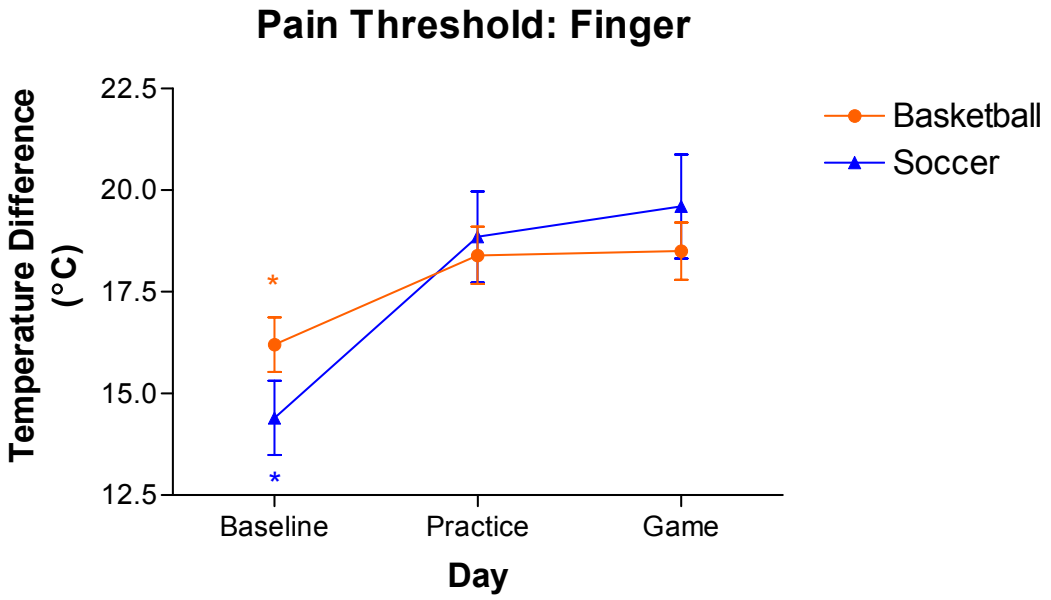

Figure 5. Withdrawal latencies on the fingertips of athletes participating in baseline, practice, and game conditions. * Significant elevations in finger pain threshold prior to practice and game days compared to baseline day in female basketball players. * Significant elevations in finger pain threshold prior to practice and game days compared to baseline day in male and female soccer players.

Figure 6.

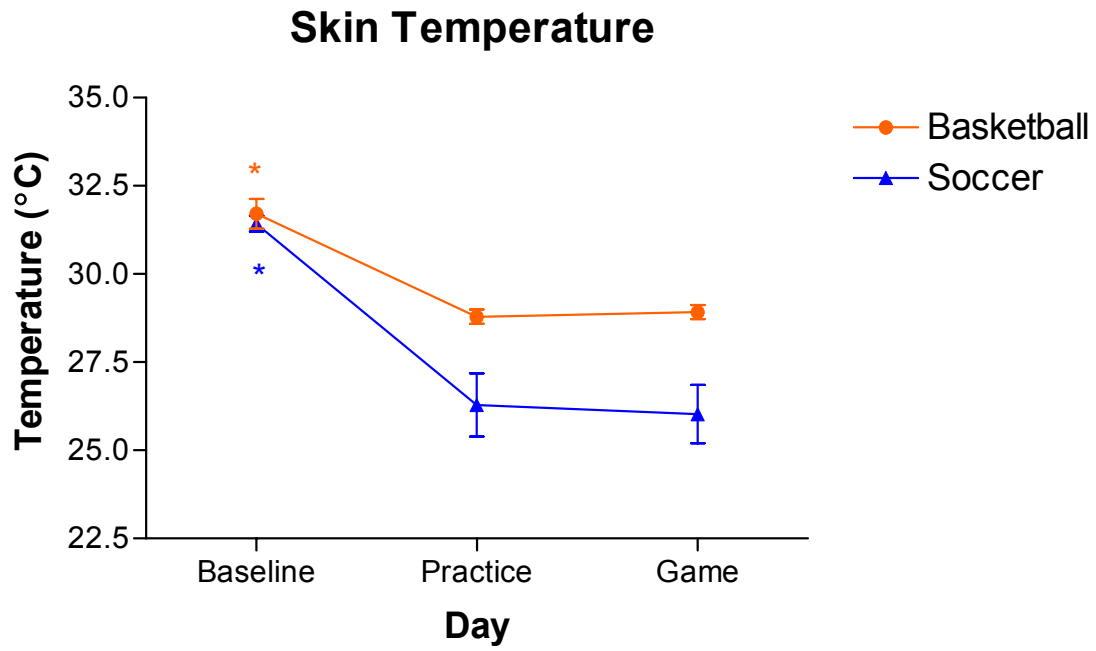

Figure 6. Baseline skin temperature at the start of thermal pain threshold testing of basketball and soccer players participating in baseline, practice, and game conditions. * Significant decreases on practice and game days compared to baseline day in female basketball players. * Significant decreases on practice and game days compared to baseline day in both male and female soccer players.

Figure 7.

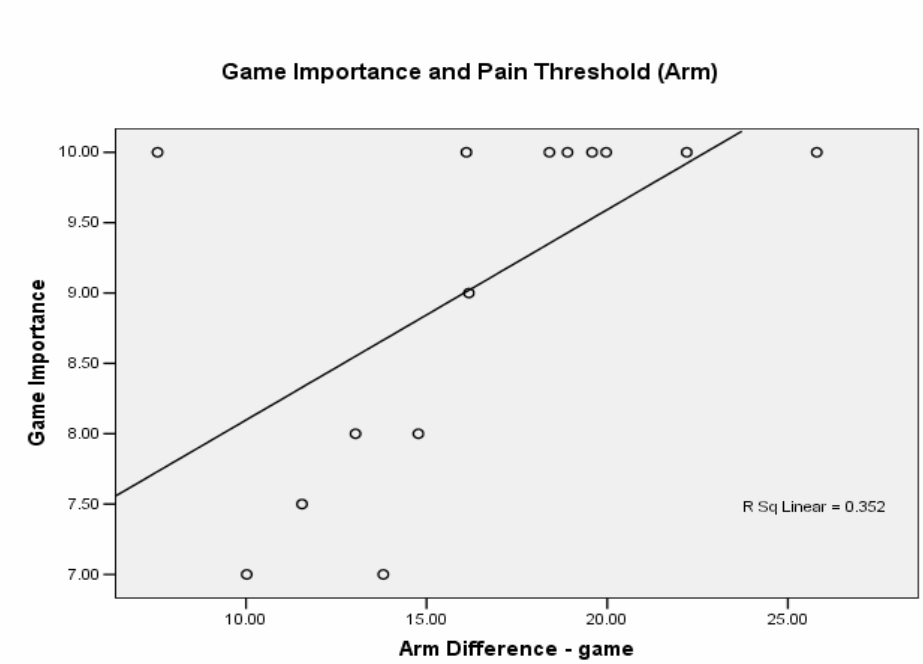

Figure 7. Correlation of game importance ratings (prior to game) of male and female soccer players to arm pain threshold. * Higher reported game importance scores are significantly correlated to higher arm pain thresholds in soccer players.

Figure 8.

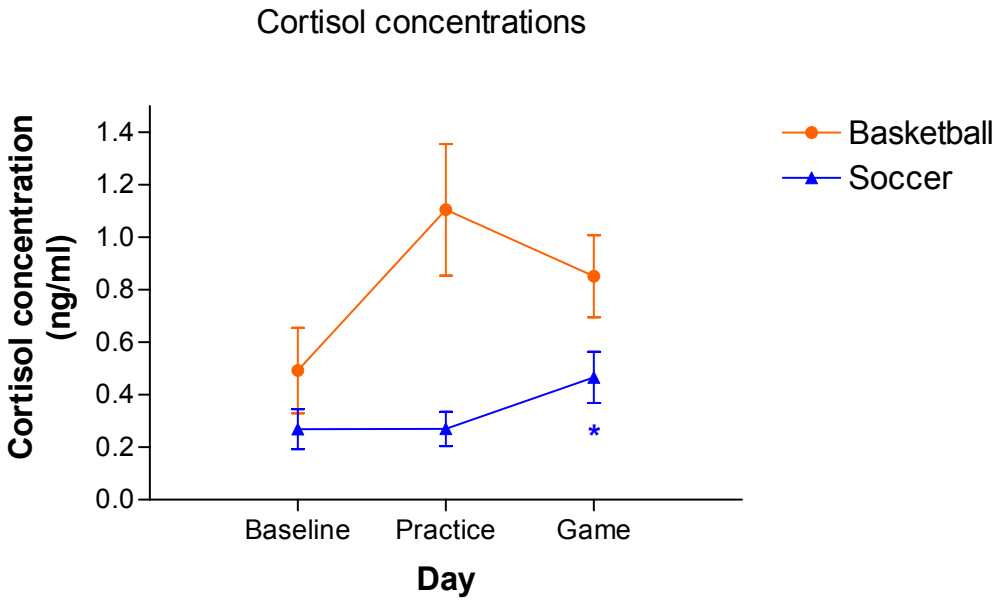

Figure 8. Salivary cortisol concentrations of athletes in baseline, practice, and game testing sessions. * Significantly elevated cortisol concentrations prior to participation in a game compared to practice and baseline days in both male and female soccer players. No significant changes in cortisol were notes for basketball players across testing condition.

Figure 9.

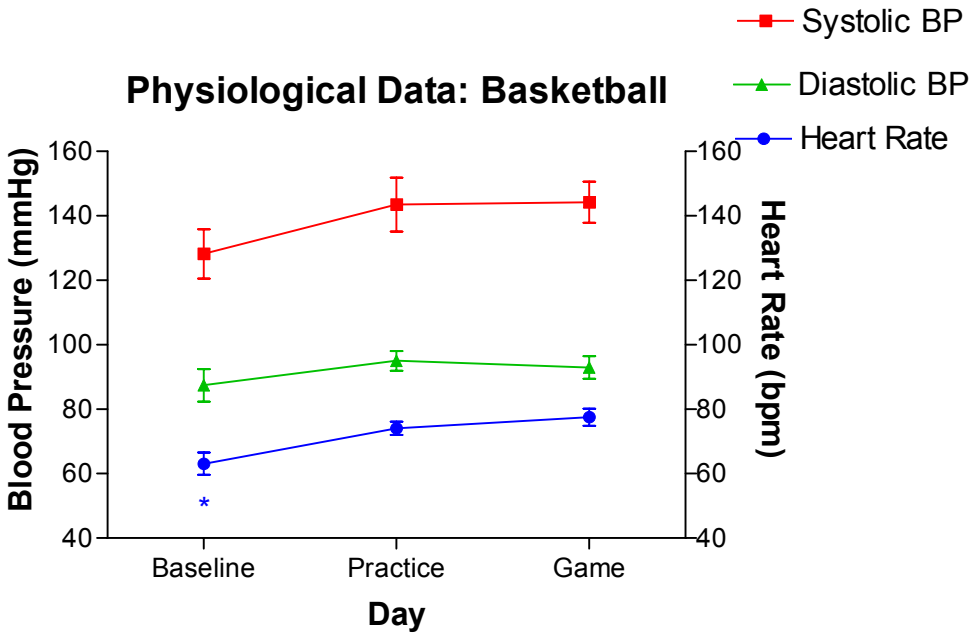

Figure 9. Physiological measures for systolic and diastolic blood pressure, and heart rate on baseline, practice, and game conditions. * Heart rate was significantly elevated on game and practice days compared to baseline day in basketball players. No changes from baseline values were noted in systolic or diastolic BP.

Figure 10.

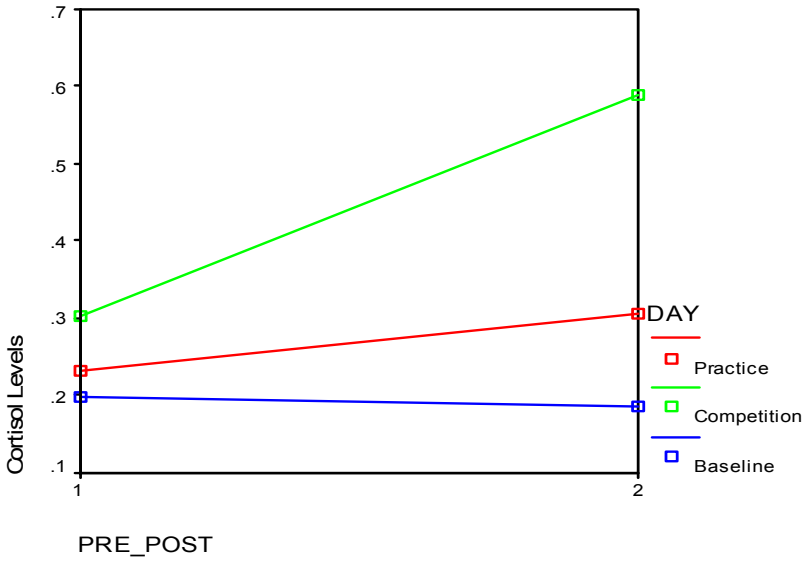

Figure 10. Pre and post salivary cortisol concentrations of female soccer players across baseline, practice, and game conditions. A trend was noted for increased cortisol pre-game compared to pre-practice. Elevations after both practice and game conditions compared to "pre" levels was also noted. An overall elevation of cortisol on game day compared to practice and baseline sessions was observed.

Appendix I.

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.

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

Appendix II.

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:

• 0		Nothing at all
• 1		Very, very weak
• 2		Very weak
• 3		Weak
• 4		Moderate
• 5		Somewhat strong
• 6		Strong
• 7		
• 8		Very Strong
• 9		
• 10		
• 11		Very, very strong
• 12		
• 13		Maximal

GAME IMPORTANCE (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?

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