Competition vs. Exercise-Induced Analgesia in Male and Female Athletes and Non-Athletes

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Abstract

Pain sensitivity in 52 college male and female athletes and non-athletes was assessed at baseline and then after exercise and competition while workload was held constant. Subjects pedaled on a stationary bike for 20 minutes at 60% maximum capacity in both exercise and competition conditions. Non-exercising, repeated-pain testing controls were tested at the same time intervals. Pain sensitivity was measured by heat pain threshold, thermal scaling and the cold pressor test. Subjects showed a significant decrease in pain sensitivity between baseline and exercise conditions (on thermal scaling) and again between exercise and competition conditions (on thermal scaling and heat pain threshold). Repeated pain testing in non-exercising subjects revealed a significant increase in heat pain threshold of athletes between their first and third testing sessions as well as significantly greater pain ratings of female non-athletes than female athletes on the cold pressor. Possible conclusions are that exercise and competition produce like-strength analgesia regardless of sex and athletic status, and that athletes show a lessened response to pain over time.
Pain has been the focus of investigation for as long as humans have been examining ways to control it. From carefully calculated torture of highly sensitive body parts to careful application of a precisely appropriate anesthetic, the study of pain is one that persists due to its “natural” importance. Central to perceptual experience as the salient warning of noxious stimuli, pain is a strong motivator of behavior and hence an important subject for psychological research.

To approach pain as a topic for study, the adaptive function of pain must be considered. The presence of pain-sensing circuitry in the human nervous system serves most obviously to alert the sensing being of danger so that the danger may be recognized as such and avoided. Thus an organism who can feel pain is afforded protection by this ability. However, pain can also be debilitating and construed as disadvantageous in certain situations such as when a wounded animal needs to fight or escape but might be hampered from doing so by intense pain. This construal of pain as potentially “disadvantageous” might seem to conflict with the idea of pain as “protection”, yet evidence shows that the body’s perceptual system may account for this variance of the adaptive function of pain by inhibiting pain in response to certain forms of stress. The phenomenon of analgesia, a body state in which pain threshold is heightened, supports this view. Certain forms of stress trigger adaptive, physiological mechanisms within an organism’s brain which in turn, through descending (from brain down) nerve pathways, causes an analgesic state known as “stress-induced analgesia.” The environmental stressors that trigger stress-induced analgesia within an organism are varied. However, the type of stressor researchers generally think of when considering stress induced analgesia is aptly defined as “an event, internal or external to an animal, that poses a real
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or perceived threat to the maintenance of the animal’s homeostasis” (Yamada & Nabeshima, 1994). Endocrine responses to these types of stressors, such as the release of adrenocorticotropic hormone into the bloodstream, occur reliably enough so that researchers are capable of unifying diverse forms of environmental stimuli into a single type under the common label “stressors.” Forced cold water swim, electric shock and food deprivation are examples of stressors which have been used in a laboratory to induce analgesia (Amit & Galina, 1986). Of interest to the current study, however is the finding that exercise and competition have been identified as stressors capable of inducing analgesia in humans.

Having emerged only recently as a scientifically documented phenomenon, endogenous analgesia highlights the adaptive capability of the body to modulate pain perception. However, the early models generated by researchers to explain how the nervous system perceived pain did not account for such adaptive modulation of pain and instead concentrated on how a painful stimulus was directly encoded by the peripheral nervous system and communicated to the brain. Researchers at the time argued over whether or not there were specific receptors for pain.

The specificity theory of pain, proposed by Von Frey in 1894, hypothesized that there were indeed specific sensory receptors for painful stimuli just as there were specific receptors for other sensations (like warmth or pressure). Yet almost contemporaneously, an alternative theory called the pattern theory of pain proposed by Goldschneider asserted that there were no specific receptors for pain, but rather specific patterns of nerve fiber discharges that were responsible for the discrimination between different sensations (Gatchel, 1999). However, much evidence then amassed which suggested that
the central nervous system exerted a top-down control over pain perception and so both theories became inadequate. Neither theory could account for the finding that the brain could perceive the same painful stimulus differentially or that psychological factors (such as anxiety) could affect pain perception.

An empirical demonstration of analgesia by Beecher (1956), however, presented evidence that discounted both theories. Beecher systematically noted the differences in perceived pain between wounded WWII soldiers and wounded civilians through their subjective reports and pain killer requests for similar procedures (Gatchel, 1999). His striking results that soldiers recently returned from battle felt significantly less pain contributed to the factors which made a revision of pain theory necessary. Thus a new theory replaced the old with the advent of the **gate control theory** (1965).

Proposed by Melzack and Wall, the gate control theory proposed that the perception of pain was modulated by the central nervous system and hence accounted for the impact of psychological factors on pain perception. It accounted for the finding that the firing of nonnociceptive afferents (peripheral nerves which communicate to the central nervous system information other than the pain message) inhibits the firing of nociceptive afferents (peripheral nerves which do communicate a pain message to the central nervous system) in the spinal cord. The discovery of this “gate” for the pain message which could be “opened” by nociceptive stimulation and “closed” by nonnociceptive stimulation popularized a pain reduction therapy tactic in which nonnociceptive afferents in the spinal cord were stimulated by electrodes on the skin (Basbaum & Jessel, 2000). The theory also viewed pain perception as a complex system which operated not only with different brain structures (all of which could influence the
pain message passed to the brain from the peripheral nervous system), but with different types of sensory pain receptors for different types of pain. However, the gate control theory is no longer a complete explanation of pain perception since it does not account for new chemical understandings of the pain message.

The evidence amassed in the last fifty years has extensively expanded our knowledge of pain. Nervous system anatomy has become much more classifiable as techniques have improved and allowed researchers to probe even microscopic levels of activity. Due to such advancement, it has been possible to identify specific sensory receptors for pain. Von Frey’s 1894 specificity theory of pain was therefore confirmed half a century after it had been so hotly debated.

These specific pain receptors are called “nociceptors” and must be present in a tissue for the encoding of the pain message. Sensitive to mechanical, thermal and chemical noxious stimuli, nociceptors encode the pain message and transmit it to the central nervous system. It is in the central nervous system, in the brain more specifically, where the subjective perceptual event of pain occurs.

Examination of the nerves which start the pain message and perform this transduction process has revealed three basic types of nociceptors: Aδ mechanoreceptors, Aδ mechanothermal receptors and C-polymodal receptors. Type C polymodal nociceptors are the slowest conducting of the three types since speed of transmission is positively correlated with wide axonal diameter and the presence of a myelin sheath and C nociceptors are small and unmyelinated. Conversely, type Aδ nociceptors are faster conducting by merit of their possessing a larger diameter as well as a myelin sheath. Type C fiber afferents are the most common element in the peripheral
nerve (3/4 of all primary afferents in primates) and are overwhelmingly (completely in
humans) nociceptive (Fields, 1987). The C fiber nerves in humans are all “polymodal” in
that they all respond to stimulus from several modalities; they are responsive to noxious
thermal, mechanical and chemical stimuli applied to the skin.

Type A\(\delta\) nociceptors (mechanothermal receptors and mechanoreceptors) respond
differentially to noxious stimuli. The type A\(\delta\) nociceptors responsive to noxious
mechanical as well as noxious thermal stimuli are A\(\delta\) mechanothermal receptors. This
type of nociceptor constitutes about 20-50% of A\(\delta\) nociceptors. The remainder of A\(\delta\)
ociceptors are referred to as high-threshold mechanoreceptors.

A\(\delta\) nociceptors are referred to as high-threshold mechanoreceptors because
although they do respond to noxious mechanical stimuli just as mechanothermal A\(\delta\) do,
they do not discharge in response to noxious thermal stimuli unless it is of high
intensity—for these neurons do not respond to the first application of noxious thermal
stimulus by definition. Instead A\(\delta\) mechanoreceptors respond and give progressively
larger responses to \textit{repeated} noxious thermal stimuli and their discharge does increase as
the stimulus intensity increases into the range that produces tissue damage. Therefore
when a A\(\delta\) mechanoreceptor is sensitized to thermal stimulus, it behaves much like a A\(\delta\)
mechanothermal receptor. Sensitization, or hyperalgesia, occurs as a result of repeated
stimulus and as a neuronal reaction to substances in the extracellular space (such as
what’s been released from damaged cells).

The unique contributions of these different types of nociceptors to the perceptual
experience of pain is most clearly evidenced by an experimental manipulation which
selectively blocks either A\(\delta\) or C nociceptors. Brief intense stimuli delivered to the distal
limb has been observed to give rise to two distinct sensations: an initial sharp and brief, pricking sensation (first pain) and then a prolonged, dull sensation (second pain).

However, when researchers selectively block A\(\delta\) nociceptors, the first pain sensation vanishes. Conversely, when C nociceptors are selectively blocked, second pain vanishes (Fields, 1987). Thus it appears that A\(\delta\) nociceptors are the peripheral nerve responsible for fast communication of the first, pricking pain and that C nociceptors are the peripheral nerve responsible for the communication of the second, duller pain. The faster conduction velocity of A\(\delta\) nociceptors relative to C nociceptors (from their greater diameter and myelin sheath) logically compliments this observation since the A\(\delta\) nociceptor pain is perceived first. Also, it stands to reason that the first pain communicated by the A\(\delta\) nociceptors, if it is thermal stimulus, is communicated by the A\(\delta\) mechanothermal receptors rather than the high-threshold A\(\delta\) mechanoreceptors since the latter does not respond to thermal stimulus unless sensitized. Despite the attraction to assign these distinct qualities of the pain experience to the activity of the distinct types of nociceptors, doing so would be an oversimplification of the process of transduction of painful stimuli since any naturally occurring stimulus will simultaneously activate a broad range of receptors and nociceptors.

The communication of the pain message within the central nervous system begins with the ascension of the message up the spinal cord and then from the spinal cord to the brain along five ascending pathways. The five pathways which carry the pain message from the spinal cord to the brain terminate in either the thalamus or the cortex. These pathways include the spinothalamic tract (the most prominent of the ascending nociceptive pathways of the spinal cord which transmits the information from the spinal
cord to the thalamus), the spinoreticular tract (which communicates the message to both the thalamus and the reticular formation of the cortex), the cervicothalamic tract (which terminates in the thalamus and the midbrain), the spinothalamic tract (brings the message to brain’s autonomic control centers such as the hypothalamus), and the spinomesencephalic tract (which carries the message to the mesencephalic reticular formation and the periaqueductal gray matter of the cortex and, through a link with the parabrachial nuclei, sends the pain information on to the amygdala, a sub-cortical structure of the neural system involved in emotion (limbic system). The spinomesencephalic tract is thought to contribute the affective component of pain due to it’s connection to this emotional system (Basbaum & Jessel, 2000).

The dissection of the pain experience as having an affective-motivational component (“unpleasantness”) and a sensory-discriminative component (“intensity”) is a commonly accepted division due to evidence which includes the identification of distinct brain regions related to the distinct affective and sensory experiences of pain. Tolle, Kaufmann & Siessmeier et al. (1999), utilizing the brain imaging technique of positron emission topography, found that the degree of activation of certain brain regions significantly corresponded to independent subjective ratings of unpleasantness and intensity. The coding of pain intensity appeared to be related to activity in the periventricular gray matter as well as to the posterior cingulate cortex, whereas the encoding of pain unpleasantness appeared to be related to the activity in the posterior sector of the anterior cingulate cortex. The results of this data therefore support the notion that spatially distinct regions within the brain specifically process the sensory and affective components of pain.
The specific anatomy of the nervous system which corresponds to the activation of analgesic mechanisms (the previously discussed modulatory circuitry) has also begun to be identified. The periaqueductual gray region of the cortex is one brain structure which, upon stimulation, produces analgesia (Basbaum & Jessel, 2000). Rats with stimulating electrodes implanted in this region underwent abdominal surgery and did not have aversive reactions to any of the surgical procedures (Reynolds, 1969). The analgesia produced, however, is not a heightening of general sensory thresholds since only pain perception, and not other sensory perception, is affected. For example, the rats who were operated upon, although showing no aversive response to the surgical procedure, did show startle and struggle when there were quick movements in their visual field (Reynolds, 1969). Research has shown that the animal experiencing analgesia from stimulation in this region maintains responsiveness to other sensory stimuli such as touch, pressure and temperature; it just feels less pain (Basbaum & Jessel, 2000). Therefore the periaqueductal gray matter is a brain region specific to the modulation of nociceptive information in particular through a descending (from brain to periphery) pathway.

The heightening of the pain threshold achieved by analgesia by descending inhibition occurs at the level of the spinal cord. The firing of peripheral nociceptive neurons is not affected by descending pathways. However, the greater electrical charge (greater ion influx) required to cause a firing of a spinal cord nociceptive neuron as a result of brain stimulation is evidence of the brain's ability to use a descending pathway to raise the pain threshold (Basbaum & Jessel, 2000). Inhibitory connections from the periaqueductal gray region of the cortex to the nociceptive neurons in the dorsal horn of the spinal cord (accomplished mostly via excitatory connections with the serotone
neurons of the nucleus raphe magnus of the medulla) enable that brain region to inhibit the communication of the pain message within the spinal cord (Basbaum & Jessel, 2000). The pain message in the spinal cord is also inhibited by virtue of other descending pathways, such as those that originate in the brain regions of the medulla and pons. These pathways inhibit spinal cord neurons by both direct and indirect inhibitory actions.

The chemical identity of the inhibitory messages has also been analyzed. So far, the chemicals associated with communication of the analgesic message have been identified as opioid. Opiates are a type of chemical most certainly known to cause analgesia since experimental evidence implicating opiates as the identity of the neurotransmitters involved in communication of the analgesic message is extensive. Morphine and codeine, for example, are opiates which have long been used by doctors to lessen the pain of their patients because the human body responds to opiate administration by decreasing pain sensitivity.

The notion that our bodies have endogenous opioids, and therefore possess a “natural narcotic”, was confirmed further by the discovery of opioid receptors in the nervous system (Basbaum & Jessel, 2000). This discovery revealed specifically that the body is prepared to receive opiates as neurotransmitters and recognizes opiates as a valid form of inter-neuronal communication. High concentrations of opioid receptors in central nervous system structures specifically identified as important to the modulation of pain (ie: periaqueductal gray matter, ventral medulla, dorsal horn of the spinal cord) further implicate opiates as carriers of the analgesic message. Opiate-induced analgesia has also been shown to utilize the same pathways as the stimulation-produced analgesia (such as that produced by stimulation of periaqueductal gray matter (Reynolds, 1969)).
because microinjections of the opiate morphine into those specific, pain-mediating brain 
regions produces a powerful analgesia by inhibition of the nociceptive neurons in the 
dorsal horn of the spinal cord (Basbaum & Jessel, 2000).

However, when neurotransmitters are discussed in terms of their role in 
communication of the analgesic message, they are referred to as “opioid” or “nonopioid” 
although those terms specifically mean “µ-opioid” and “non-µ opioid” respectively. 
Because a high correlation exists between the strength of an analgesic and its affinity for 
binding to the opioid receptor type µ, affinity for receptor µ serves as a fundamental 
distinction between types of analgesics (Basbaum & Jessel, 2000). Although other 
classes of endogenous opiates (such as opiates κ and δ) exist, experimental manipulation 
which blocks µ opiate receptors through the administration of the drug naloxone has 
demonstrated the strength of µ opiates as communicators as the analgesic message. 
Naloxone acts as an opiate antagonist by specifically binding to opioid receptors (with 
the highest affinity for type µ) without itself activating the receptor. Therefore after 
administration of naloxone, the µ opioid receptor is blocked and any analgesia caused by 
µ opioid receptor communication vanishes. The analgesic effect of the opiate morphine, 
for example, vanishes when an injection of naloxone is made into pain modulating 
regions such as the periaqueductal gray region or the serotenergic nucleus of the medulla 
(Basbaum & Jessel, 2000). Also, the pain relief afforded to human patients with 
intractable pain problems through electrical stimulation of the periaqueductal and 
periventricular gray matter was reversed with administration of naloxone (Hosobuchi, 
Adams & Linchitz, 1976). Hence, analgesic effects were eradicated when µ opiate 
receptor communication in the nervous system was disabled.
However, research has demonstrated that analgesia does not always completely vanish with the administration of naloxone and the disablement of µ opiate receptors. Thus a non-µ opioid, or “naloxone-insensitive”, category of analgesics is revealed. Although this category is one which could contain opioids which are just not type µ, it is nonetheless commonly referred to in the literature as “nonopioid”. Conversely, the µ opioid category is simply referred to as “opioid” despite the fact that it only really encompasses one type (µ) of several endogenous opioids (such as κ and δ).

A model which attempts to explain the existence of both opioid and nonopioid systems is the collateral inhibition model. The collateral inhibition model proposes that one analgesic system, which is initially activated by the specific intensity and temporal pattern of the stressor, inhibits the other system. Some researchers have reasoned that this would be adaptive for an organism since one pain-inhibition system would always remain functional (Kirchgessner, Nodnar, & Pasternak, 1982 as cited by Amit & Galina, 1986). Evidence for the existence of collateral inhibitory mechanisms between opioid and nonopioid mediated analgesia has been provided by Steinman, Farris, Mann, Olney, Komisaruk, Willis and Bodnar (1990). They showed that the analgesia produced by the opiate morphine is significantly reduced when rats are exposed to a nonopioid, analgesia-inducing stressor (like continuous cold water swim) prior to morphine administration (Yamada & Nabeshima, 1995). This result indicates that the ability of opiates to produce analgesia is somehow inhibited if the nonopioid form of analgesia is also induced. Therefore, just as the collateral inhibition model would have stipulated, the activation of one of the analgesic mechanisms inhibited the other.
Investigation into the question of when nonopioid vs. opioid analgesic systems are activated has only definitively illustrated that the type of system activated depends on the nature of the stress. Research that sought to define the discrimination between the opioid and nonopioid pain modulation systems pointed to the trend that naloxone was less likely to eradicate analgesic effects when the stressor which induced the analgesia was one of higher intensity and longer duration. Thus a shift from the opioid system to the nonopioid system was triggered by an increase in severity of stressful stimuli, such as colder water and continuous instead of intermittent swimming in a forced swim (Mogil, Sternberg, Bailian, Liebeskind, & Sadowski, 1995). However, for exceptionally severe stress, a shift from nonopioid to opioid systems occurs. It has been suggested that for such severe stress (20-30 minutes of intermittent foot-shock or 60-80 tailshock delivered to rats), the organism has learned the stress is inescapable and this perception of “learned helplessness” results in the shift to opioid analgesia (Maier, Sherman, Lewis, Terman & Liebeskind, 1982).

However, the shift from opioid to nonopioid systems that accompanies increased severity of stress may not be uniform cross species since the female Quackenbush mouse exhibits the opposite: a shift from nonopioid to opioid systems with increasing severity of the stress (Tierney, Carmody, & Jamieson, 1991 as cited by Mogil et al., 1995). This opposite finding contrasts to the trend found in rats as well as in other mice (Mogil et al., 1995). Possible reasons for this apparent divergence in findings are the researchers’ utilization of different rodent species and different stress stimuli. Consequently, the exact conditions which would reliably produce either the opioid or nonopioid analgesia cross
species are not clear. What is clear is that increasing severity of stressful stimuli results in a shift of one neurochemical analgesic system to another.

The nature of a stressor is also an important determinant of whether the stressor can or cannot induce analgesia. The stressors of exercise and competition are two stressors that can, depending on their nature (exercise parameters), induce analgesia. Research (discussed below) suggests that the analgesia produced by exercise is not uniform in that different analgesic mechanisms are triggered (opioid or non-opioid) by different exercise (aerobic vs. anaerobic, for example), only some forms of stimulus pain used to measure pain sensitivity (such as cold pressor vs. thermal pain)\(^1\) elicit analgesic response, and women may be more stressed by exercise than men. The stressor of competition may also not be uniformly impactful and analgesia-inducing cross sex and different individuals.

Research examining the analgesia induced by exercise has yielded highly non-unified results and indicates a need to standardize experimental procedures along several parameters. One study by Olaussson, Eriksson, Ellmarker et al. (1986) demonstrated the diverse analgesic systems (opioid and nonopioid) involved in exercise-induced analgesia by pain testing subjects before and after 20 minutes of leg and arm exercises (as cited in Koltyn, 2000). Subjects experienced significantly elevated dental pain thresholds after exercise, yet naloxone only attenuated analgesia in subjects following arm exercise. Thus exercise can trigger both naloxone-sensitive (µ opioid) and naloxone-insensitive (non-µ opioid) analgesia mechanisms and the type of exercise performed (here, arm or leg)

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\(^1\) The cold pressor measure of pain sensitivity is done by submerging a subject’s arm in ice water for a period of time while they continuously rate the pain they feel. The thermal pain measure of pain sensitivity can be done by applying noxious, hot stimulus to the skin and measuring at what temperature a subject feels pain (heat pain threshold) or by exposing the subject to different hot temperatures and recording their pain ratings (thermal scaling).
contributes to the determination of which one of the systems will be activated. This finding is further complicated by the results of a study by Janal, Colt, Clark et al. (1984) which found that post-run analgesia was reversed by naloxone only in response to an ischaemic pain measure stimulus (pressure) but not for the thermal pain measure stimulus (as cited in Koltyn, 2000). Thus the detection of exercise-induced analgesia not only depends on the condition of whether naloxone has been administered to the subject, but also on the particular stimulus used in the pain measure. In sum, in order to detect whether opioid or nonopioid systems have been activated, experimenters must use an array of pain stimuli for the pain measure (in combination with opioid receptor blockers).

Use of more than one painful stimulus to measure analgesia in subjects is also now validated as a standard method in analgesia research due to many study results such as the above which indicate that analgesia detected by using one pain stimulus may not be detected by using another. Because pain threshold after exercise is altered differentially for different types of painful stimuli, current research must follow this multi-stimulus pain methodology. Which stimulus allows detection of which analgesia after which exercise is still unclear. This is so because trends of which stimulus allows detection of which analgesia after which exercise are not clear (or at least in the mind of this researcher).

Possibly due to the great lack of uniformity in methodology in measurement of pain sensitivity, different exercise stressors—aerobic, resistance and isometric—do not produce uniform analgesia. Aerobic exercise has led to analgesia which in one study (one mile run with ischemic pain measure, Haier, Quaid, and Mills, 1991) was sensitive to naloxone administration and which in another study (cycling on an ergometer with a
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dental pain measure, Droste, Greenlee, Schreck, et al., 1991) was not (as cited by Koltyn, 2000). A third study (6.3 mile run at 85% aerobic capacity) detected naloxone-sensitive analgesia in response to aerobic exercise only with ischemic pain measure and not thermal pain measure, therefore supporting the possible deduction that aerobic-exercise-induced analgesia uses µ-receptor opiates that can counteract ischemic, but not thermal or dental pain (Janal et al., 1984). Resistance exercise (75% maximum resistance capacity and ischemic pain measure (Kotlyn & Arbogast, 1998)) and isometric exercise (isometric contraction to exhaustion and ischemic pain measure (Koltyn & Ekholm, 1995)) have also resulted in analgesia (as cited by Kotlyn, 2000). However, the lack of a naloxone condition in these resistance and isometric exercise studies prevents drawing conclusions about whether resistance and isometric-exercise-induced analgesia is mediated by µ-receptor opiates or not. Thus although it is clear that exercise can lead to analgesic state, the way in which the analgesic mechanisms work remains nebulous. The physiological mechanisms which dictate analgesic response to distinct stimulus ought to become more clear as all researchers adopt the multi-pain stimulus methodology, manipulate exercise parameters by incrementally increasing intensity of different forms of exercise and selectively block suspected analgesic communicators.

Competition has also been documented as a cause of analgesia. Sternberg, Bailin, Grant, and Gracely (1998) found that both male and female athletes (basketball player, fencers, and track athletes) experienced analgesia immediately after athletic competition in response to cold pressor and thermal pain stimulus. The analgesia experienced by these athletes could have been due to the stress of competition as well as exercise because competition independent of exercise has been illustrated to cause analgesia. Such a
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competition, specifically a sedentary videogame competition, was found by Sternberg, Bakat, Kass, Alboyadjian, and Gracely (2001) to produce an analgesic response to cold pressor pain in males (though not females). This result supports the idea that competition-induced analgesia operates by a separate mechanism than exercise-induced analgesia since subjects in a study by Janal et al. (1984) did not show analgesic response to cold pressor pain after exercise (Koltyn, 2000).

The reason that the sedentary videogame competition did not induce a like analgesia in females could be due to competition’s exclusivity as a trigger for analgesic mechanism in males, competition’s triggering of a unique female analgesic mechanism that produces analgesic response to stimulus pains other than cold pressor, or videogame competition’s inability to be a meaningful competition for females. The greater female apathy towards the videogame competition detected by researchers throughout the course of the experiment supports the latter conclusion, as does research which suggests videogames are more popular with males (Brown, Holzer, Brown, & Brown, 1997 as cited by Sternberg et al., 2001). The ability of competition as a stressor to produce analgesia therefore might be sex-dependent or dependent upon whether the competition is personally meaningful.

In order to compare male and female pain sensitivity in response to competition, one must recognize the significant baseline sex difference in pain sensitivity; the reliable sex-difference in the literature is that females are in general more sensitive to painful stimuli than males. Early studies that relied on subject reports of pain were criticized since it was hypothesized that males would report less pain than females despite similar sensation. This hypothesis was born out of research which illustrated that male children
are socialized to repress emotional display and that men feel obligated to display stoicism in response to pain (Miaskowki, 1999). However, even studies which utilized methods of monitoring subject pain perception without subject report echoed the previous trend. Ellermeier & Westphal (1995) found that the greater pain reported by females than males in response to pressure pain stimulus was significantly correlated with pupil dilations, a pain indicator beyond voluntary control. Studies done on rats also showed greater female response to pain (thermal and electric shock) (Beatty & Beatty, 1970 and Marks & Hobbs, 1972 as cited by Miaskowski, 1999). Additional evidence establishes the general fact that a sex-difference in pain perception does exist because studies superseding a possibly biased subjective report have also found sex difference in pain response. Paulson, Minoshima, Morrow, and Casey (1998) used brain imaging techniques and discovered a greater activation in the prefrontal cortex of females in response to thermal pain stimuli. Greater female response to κ-opiates, such as a significantly stronger and prolonged analgesia, has also been observed (Gear, Miaskowski, et al., 1996 as cited by Miaskowski, 1999). Thus reliable and consistent sex-differences in basal pain sensitivity exist and suggest different levels of perceived stress between males and females. Consequently, such sex difference in pain sensitivity at baseline may be responsible for the presence of relative sex difference in pain sensitivity observed after competition.

Testosterone, a predominantly male hormone, may also play a role in the observed sex-differences towards competition because not only is it much more prevalent in males and responsible for biological sex-differences, but also because testosterone has been shown to influence competitive drive in particular. Rising testosterone levels cause an individual to become more assertive, seek competition, and act dominantly (Mazur,
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Booth, & Dabbs, 1992). The finding of Singh (1990) that top level female athletes report more competition anxiety than top level male athletes may serve as evidence of a distinct, positive orientation of males towards competition which is rooted in or mediated by males’ greater levels of testosterone. Increased testosterone levels in competitors pre-competition (of both athletic and non-athletic type) is also a consistent finding which supports a positive relationship between testosterone level and competitive drive. Animal studies also indicate a relationship between testosterone and competition since a significant positive correlation has been observed between testosterone level and competition-associated constructs such as dominance (Sielinski & Vnadernbergh, 1992 with male house mice and Setchell & Dixson, 2000 with male mandrills) and aggression (Alber, Petrovic, & Walsh, 1988 with rats and Delville, Mansour, & Ferris, 1994 with hamsters). Zielinski & Vandenbergh even manipulated testosterone level as an independent variable in their study and found that high testosterone mice achieved higher dominance as a result of the manipulation. Therefore it is possible that higher level of testosterone in men may therefore lead them to perceive more stress than women in response to competition.

Research examining the fluctuation of testosterone levels in response to competition in males has shown that testosterone levels after a successful competition are only significantly related to how much the individual perceives themselves as responsible for the success. Gonzalez-Bono, Salvador, Serrano, and Ricarte (1998) monitored testosterone levels in basketball players and found that the more winning-team members attributed the successful outcome to external factors, the lower their testosterone levels were post-game. They also found that the more losing-team members attributed the
unsuccessful outcome to external factors, the higher their testosterone levels were post-game. A subsequent study by Gonzalez-Bono, Salvador, Ricarte, Serrano, and Arnedo (2000) reasserted the results of the first study by finding that the testosterone levels of basketball team members after successful competition were significantly correlated with external attribution of success. Thus it appears that the more an individual perceives himself as contributing to the success, the higher his testosterone levels will be. The fluctuation of testosterone in individual competition, such as a Judo match, also mirrors this positive relationship between testosterone level and perceived personal competitive excellence; Serrano, Salvador, Gonzalez-Bono, Sanchis, and Suay (2000) found Judoists’ testosterone levels were positively associated with self-appraisal of performance and attribution of outcome to personal effort. The fluctuation of testosterone levels in females, however, has not been examined in relationship to personal effort in competition.

Different testosterone patterns in males and females have been documented though, and Mazur, Sussman, and Edelbrock (1997) found that females do not show the pre-contest rise in testosterone that males show and that female testosterone level decreases throughout the competition. However, this study examined female testosterone level in response to a videogame competition, which, for reasons discussed earlier, may not be a meaningful competition for females and hence not one which can elicit the biological responses usually induced by competition. Thus the differences between the sexes in analgesic response to competition may be due to the degree of perceived effort which itself may be modulated by testosterone. A stronger analgesia may derive from a
greater perceived effort in response to competition and that competitive effort in turn may be made greater by men’s greater amount of testosterone.

Another possible analgesic difference between males and females is one which discriminates between exercise and competition. It has been suggested previously in this paper that men might have an analgesic mechanism triggered by competition alone which women do not have. However, evidence suggests that women may have an analgesic mechanism which men do not have; one for which exercise is a more capable trigger. The finding by Sternberg et al. (2001) that women experience analgesia (cold pressor measure) more readily in response to exercise (10 min. run at 85% max. heart rate capacity), as well as the like finding by Koltyn, Malani, Stegner, and Tobar (2001) (but with pressure pain measure after isometric exercise), suggest that exercise may induce greater analgesic response in women.

Athletic status has also been shown to affect perceived pain in that athletes seem to feel less pain in response to a cold pressor stimulus than non-athletes. Researchers have found that athletes report less pain than non-athletes in response to cold pressor pain (Hall & Davies (1991); Sullivan, Tripp, Rodgers, and Stanish (2000); Sternberg et al. (2001)). Research comparing athlete vs. non-athlete response to pain other than cold pressor pain, such as thermal pain, does not indicate a significant difference (Sternberg, 2001). Possible reasons athletes give lower pain ratings are that individuals with higher pain thresholds choose become athletes and that athletic training includes a frequent confrontation with, as well as demand for forbearance of, pain. Because studies have illustrated that classical conditioning can produce an analgesic response, it stands to reason that pain-confronting athletic training may cause athletes to experience a rise in
basal pain sensitivity. Thus cues of exercise and competition may produce analgesia more readily in athletes because athletic training frequently pairs those cues with pain. If this possible difference in basal pain sensitivity is due to the ascending component, then athletes may have a lower density of nociceptors or have a higher threshold for transduction. If due to the descending component, then athletes may more readily inhibit the pain message because of athletic training. Because of this possible difference in pain sensitivity, studies examining analgesia, especially after stressors of exercise and competition, must control for athletic status.

The present study seeks to check for differences in analgesia across sex and athletic status in response to competition. To evaluate whether females indeed have an analgesic mechanism for which competition is the trigger, the present study attempts to measure analgesia in males and females after a cycling competition. Since past research detected such a competition-triggered, analgesic mechanism only in males possibly because videogame competition is a meaningful competition for males only, the competition used in this study may be able to induce analgesia in females. Since the researchers know of no research which would designate a cycling competition as one significantly more meaningful to males than females, it is assumed that any perceived sex differences will be due to a difference in the effect that a general cognitive mindset of competition has on males and females (especially since an exercise condition will control for the exercise-induced analgesic effects that a cycling competition could elicit).

Athletes and non-athletes will serve as subjects so that analgesia produced in response to exercise and competition can be compared cross populations for whom those stressors may operate differently. Subjects will report to the lab on three successive days for pain
testing. The first day all subjects will be tested for a baseline pain sensitivity and have their maximum exercise capacity determined. The second day, subjects will exercise on a stationary exercise bike for 20 minutes at 60% maximal exercise capacity and then have their pain sensitivity assessed. The third day, the subjects will be told that they are in competition with another subject to travel the “furthest” on the bike in the given 20 minute period. However the workload between the exercise and competition conditions will be kept constant by the computer software that controls the bike’s pedal resistance, therefore isolating the competitive mindset as the only difference between exercise and competition conditions. Subjects will include both male and female athletes and non-athletes as well as repeated pain-testing controls. The athlete subject population will consist of track athletes since a previous study by Sternberg et al. (1998) showed that track athletes had the greatest analgesic response after athletic competition than other collegiate athletes of other various sports (such as fencing and basketball). Pain measures used will be the cold pressor test, suprathreshold thermal scaling, and thermal heat threshold. Stress levels will be measured each session by taking blood pressure and heart rate, salivary samples for analysis of the hormone cortisol, and subjective arousal ratings. These measures, along with subjective ratings of effort that will be given following both the exercise and competition bouts, will serve to check whether the manipulation of the competition was successful.

I hypothesize that, consistent with past literature, males will show less pain sensitivity than females, athletes will show less pain sensitivity than non-athletes, and all experimental subjects will show less pain sensitivity after exercise (including exercise involved in competition) than at baseline. I also predict that only males will show
significantly more analgesia in the competition condition whereas for females it will be relatively the same as the exercise condition.

Method

Subjects and Design:
The design was a 2x2x2x3 mixed factorial in which subjects were assigned to either the experimental (exercise) or repeated-pain-testing control condition and tested on three different days: Baseline day, exercise day and competition day (though controls had only baseline day 3 times). Pain sensitivity and stress level were assessed each time. Subject gender and athletic status (athlete or non-athlete) were also independent variables across which data were analyzed.

A total of 52 student subjects were recruited through signs posted around Haverford and campus-wide e-mails. Subjects included equal numbers of male and female track athletes and non-athletes. Experimental subjects (N=32) were compared with control subjects of the same sex and athletic status who served as repeated-pain-testing controls (N=20). Experimental and repeated-testing control subjects were paid $50 and $25 respectively for their participation.

Pain Measures
The pain measures obtained were cold pressor ratings, thermal heat threshold and suprathreshold thermal ratings.

A Medoc thermal stimulator delivered thermal stimulus through a 30x30mm Peltier thermode. In the thermal heat threshold procedure, the thermode was placed on the volar surface of the subject’s forearm and rose from the neutral temperature of 32°C
(at a rate of 1°C/sec) until the subject reported that pain threshold has been reached. The device was then reset to 32°C and the procedure repeated 4 times so that a stable pain threshold was attained. The average of those 4 values was used in analysis as the subject’s heat pain threshold for a given day.

In the thermal direct scaling procedure, suprathreshold pain ratings were determined by presenting four stimulus temperatures (42°C, 44°C, 46°C, 48°C) for a duration of 5 seconds to the volar surface of the forearm. Stimulus sequence was completely randomized within the block with the possibility of successive stimuli of the same intensity. Subjects were administered 2 blocks of stimuli at each scaling session. The 8 total stimulus presentations within each performance of the thermal direct scaling procedure were delivered to 8 different parts of the volar forearm surface (in order to avoid the effect of hypersensitivity in one previously stimulated spot). Stimuli were delivered at about 10 second intervals between the 8 different forearm sites. The thermode had a rise and fall time of 4°C/second and had a duration of 5 seconds at each stimulus temperature. Subjects gave pain ratings on the Gracely box scales (See Appendix A) in response to each stimulus intensity. The Gracely box scales allow subjects to rate pain intensity and unpleasantness on separate scales that consist of 0-20 numerical ratings anchored in descriptor terms. The pain ratings used in analysis were those the subject gave in response to the initial presentation of a given temperature.

The cold pressor test consisted of subjects placing their arm (up to the elbow) in the center of a mesh screen submerged in a bucket of ice water (ice is not in contact with a subject’s arm) for a maximum of 90 seconds or until the subject removes his/her arm in which case a maximum score of 20 was assumed for the remainder of the 90 seconds).
Pain ratings on the Gracely box scales were obtained every 15 seconds, and in the case that a subject removed his/her arm before the end of 90 seconds, a maximum rating of 20 was assumed for whatever time remained. The sum of pain ratings for the 90 second period on each scale was the value used in analysis.

Stress Measures

Measures of subjects’ stress levels were attained through several different avenues: hormone analysis, measure of cardiovascular activity, and subjective arousal. Salivary samples were taken to determine the levels of the stress hormone cortisol, blood pressure and heart rate were recorded, and subjects rated their subjective state of arousal with the Body Awareness Questionnaire (see Appendix B). All measures were obtained just prior to pain testing.

Procedure

Day 1

Subjects reported to the lab on “Day 1” of the 3 day sequence and, after giving informed consent (see Appendix C), were presented with a medical history questionnaire (see Appendix D). Athletes reported to the lab at a time of day before their scheduled practice so analgesic effects from their practice exercise were avoided. Before being exposed to any painful stimulus, subjects were first acquainted with the thermal stimulator device in a “warm threshold” procedure so that they could practice making perceptual judgments about thermal stimuli. This initial practice consisted of exposing subjects’ volar forearm
surface to the thermode which started at 20°C and rose consistently until they detected (and reported) warmth.

Baseline thermal pain threshold was then determined for all subjects with the same device through the thermal threshold test. Suprathreshold pain ratings were then obtained from all subjects with the same device through the direct thermal scaling test. Finally, subjective measures of pain were recorded for the cold pressor test.

Subjects’ baseline stress levels were also attained immediately prior to pain testing so that the recorded stress response was not elevated by the pain testing.

On all 3 days, repeated-pain-testing control subjects reported to the lab and had their pain sensitivity tested with the first thermal pain threshold test, then the thermal scaling test and then the cold pressor test. Stress level was also assessed.

The maximal exercise capacity of each subject in the exercise group was determined on an exercise bike (Lode Corival cycle ergometer) using the standard Maximal Exercise Capacity Test, wherein subjects pedal at increasing resistance until they are unable to maintain a fixed pace (50 rpm).

**DAY 2**

For experimental subjects, the second day was exercise day. They exercised non-competitively for 20 minutes on the ergometer at 60% maximal capacity. Blood pressure and heart rate were assessed immediately after exercising. Measures of stress level were also obtained. Pain sensitivity was assessed first with the thermal threshold test, then the thermal scaling test and finally the cold pressor test. Borg’s Ratings of Perceived Effort scale (RPE; see Appendix E) was filled out after both the non-competitive (Day 2) and
competitive (Day 3) exercise bouts to serve as check that the contrived competition was meaningful.

**DAY 3**

For exercising subjects, the third day was competition day. Subjects were told that they were competing against another subject (sex and athletic status matched) with respect to “distance” traveled on the ergometer. Each subject waited in the room while the other subject had their turn (20 minutes) cycling. They were informed that the subject who traveled the farthest would receive $5 at the end of the session and the subject who traveled the farthest out of all of the subjects (same sex) would receive an additional $25. However, this was a deception and no one could have won since distance traveled was not even recorded. Also, the workload of the subject was fixed by the computer software (Turbofit) controlling the cycle resistance. Workload in the competition condition was therefore matched with workload in the exercise condition without the subjects’ knowledge. After cycling each subject immediately had heart rate and blood pressure measured and pain sensitivity assessed. Salivary samples were obtained and the RPE and BAQ filled out. After the second cyclist finished pain testing both subjects were informed of the deception, debriefed and paid for their participation. A lottery for a $30 prize was held for all subjects who underwent the deception in the experimental condition.

_Data Analysis_
A 2x2x2x3 factorial ANOVA was performed to test for significant main effects and interactions. Fisher’s LSD post-hoc was performed for any significant effects indicated by the ANOVA.

A main effect of day in the experimental group and not the pain-testing control group would confirm hypotheses if pain ratings were lower (and stress levels higher) after exercise and competition than they were at baseline. An interaction between day and gender would confirm a central hypothesis if males gave lower pain ratings than females on competition day. A main effect for gender and athletic status across trials would follow expectations if males and athletes generally gave lower pain ratings than women and non-athletes respectively.

Results

Stress Measures

Heart Rate

For all experimental subjects, there was a main effect of day ($F_{2,56} = 92.09, P = .00$). Heart rate significantly increased from baseline day to exercise day ($P = .00$) and from exercise day to competition day ($P = .00$) (See Table 1). There were no significant differences between the heart rate of control subjects cross day. (Fig. 1)

Table 1

<table>
<thead>
<tr>
<th>HR in beats/minute</th>
<th>Baseline</th>
<th>Exercise</th>
<th>Competition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>76.19</td>
<td>111.53</td>
<td>126.25</td>
</tr>
<tr>
<td>Standard Error</td>
<td>1.93</td>
<td>3.59</td>
<td>4.10</td>
</tr>
</tbody>
</table>
In both experimental and control groups, there was a main effect of athletic status ($F_{1,16}=5.52, P=.03$ in controls, $F_{1,28}=5.98, P=.02$ in experimental subjects) in that athletes had significantly lower heart rate than non-athletes.

In the experimental group, there was a significant sex x athletic status interaction ($F_{1,28}=7.94, P=.01$) in that male non-athletes had significantly higher heart rate than all other experimental subjects.

**Blood Pressure**

Neither systolic nor diastolic blood pressure of control subjects differed significantly cross day, and no significant differences in diastolic blood pressure were found cross day in either experimental or control groups.

A main effect of sex ($F_{1,16}=8.38, P=.01$) as well as sex x athletic status interaction ($F_{1,16}=10.17, P=.01$) was found in systolic blood pressure of controls. Post hoc analysis revealed that female non-athlete controls had significantly lower systolic blood pressure ($M=115.33\pm3.64, P < .05$) than all other subject groups (female athletes $M=128.80\pm3.64$, male athletes $M=127.73\pm3.64$, male non-athletes $M=137.47\pm3.64$).

A significant sex x athletic status ($F_{1,16}=14.23, P=.00$) was also found in diastolic blood pressure of controls in that female non-athletes had significantly lower diastolic blood pressure than female athletes ($P=.03$), whereas male non-athletes had significantly greater diastolic blood pressure than both female non-athletes ($P=.00$) and male athletes ($P=.01$) (See Table 3).

**Table 3**

<table>
<thead>
<tr>
<th>Controls</th>
<th>Female Non-Athletes</th>
<th>Female Athletes</th>
<th>Male Non-Athletes</th>
<th>Male Athletes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diastolic Blood Pressure</td>
<td>89.73\pm2.89</td>
<td>77.60\pm2.89</td>
<td>74.07\pm2.89</td>
<td>83.73\pm2.89</td>
</tr>
</tbody>
</table>
Competition vs. Exercise-Induced Analgesia

Cortisol

There were no significant differences found in cortisol levels across day for either the experimental or control group.

A sex x athletic status interaction was present in the experimental group ($F_{1,28}=5.00, P=.03$) in that male athletes had significantly higher cortisol levels ($M=.52\pm.08$) than female athletes ($M=.21\pm.08, P=.01$).

Subjective Arousal (Body Awareness Questionnaire)

In the experimental group, there was a main effect of day ($F_{2,56}=12.22, P=.00$) in that subjects were significantly less aroused (had lower scores on the BAQ) on baseline day ($M=27.18\pm.72$) than they were on exercise day ($M=30.56\pm.93, P=.00$) or competition day ($M=31.94\pm.99, P=.00$).

A main effect of day was found in control subjects ($F_{2,32}=3.96, P=.03$) in that their subjective arousal was significantly higher on Day 1 ($M=26.95\pm.75$) than it was on Day 3 ($M=24.60\pm.65, P=.01$).

There was also a day x sex x athletic status interaction ($F_{2,56}=5.13, P=.01$) in the experimental group in that male athletes were significantly more aroused on competition day ($M=33.25\pm1.98$) than they were on baseline day ($M=25.38\pm1.43, P=.00$) or exercise day ($M=28.63\pm1.86, P=.02$), whereas male non-athletes were similarly aroused on all days. Female athletes were also similarly aroused on all days, whereas female non-athletes were significantly more aroused on exercise day ($M=33.38\pm1.86, P=.00$) and competition day ($M=32.75\pm1.98, P=.00$) than they were on baseline day ($M=25.13\pm1.43$).

(Fig. 2)
Ratings of Perceived Effort

A main effect of day ($F_{1,28}=28.65$, $P=.00$) and day x athlete interaction ($F_{1,28}=15.26$, $P=.00$) was found in experimental subjects’ ratings of perceived effort after exercise and competition. Athletes perceived themselves as putting in significantly more effort on competition day ($M=5.44 \pm .52$) than exercise day ($M=9.44 \pm .69$, $P=.00$) whereas non-athletes did not significantly differ in their RPE score cross day. (Fig. 3)

Skin Temperature

A main effect of day in experimental subjects was found ($F_{2,54}=4.37$, $P=.02$) in that skin temperature was significantly lower on competition day ($M=31.07 \pm .20$) than it was on baseline day ($M=31.71 \pm .14$, $P=.01$).

There was a significant main effect of athletic status in control subjects ($F_{1,16}=6.02$, $P=.03$) in that control athletes had a significantly lower skin temperature ($M=31.28 \pm .21$) than control non-athletes ($M=32.02 \pm .21$). This effect may be due to a more efficient cardiovascular system of athletes.

Pain Sensitivity Measures

Heat Pain Threshold

There was a main effect of day in the experimental group ($F_{2,56}=5.16$, $P=.01$) in that subjects had a significantly higher threshold on competition day ($M=45.72 \pm .43$) than they did on either exercise day ($M=45.14 \pm .40$, $P=.03$) or baseline day ($M=44.93 \pm .48$, $P=.00$). Pain threshold of control subjects did not significantly differ cross day. (Fig. 4)

A significant interaction between day and athletic status was found in repeated-testing controls ($F_{2,32}=4.34$, $P=.02$) in that athletes had a significantly higher pain threshold on Day 3 ($M=46.53 \pm .65$) than they did on Day 1 ($M=45.35 \pm .60$, $P=.00$).
Thermal Scaling

In the experimental group, a significant day x temperature interaction was found \((F_{6,168}=2.70, \ P=.02)\) in that subjects gave significantly lower pain ratings at 42°C on competition day \((M=4.81 \pm .67)\) than they did on exercise day \((M=6.34 \pm .61, \ P=.04)\) or baseline day \((M=7.50 \pm .69, \ P=.00)\). (Fig. 5 and 6) No significant difference was found in pain ratings across day at 44°C, 46°C or 48°C.

No significant difference in thermal pain ratings across day, or day x temperature interaction, was found in repeated-testing controls. (Fig. 5 and 6)

A significant main effect of temperature was present in both experimental and control groups \((F_{3,84}=148.05, \ P=.00\) for experimental, \(F_{3,48}=107.59, \ P=.00\) for control). Although pain ratings for different temperatures were not always significantly different from one another, pain ratings did increase significantly with increase in temperature for both experimental and control groups. (Fig. 6)

Additionally, experimental group pain ratings revealed a temperature x scale x day interaction \((F_{6,168}=2.41, \ P=.03)\). Post-hoc analysis revealed that on the intensity scale, experimental subjects gave significantly higher pain ratings at 42°C \((M=8.72\pm.81)\) on Day 1 than on Day 2 \((M=7.34\pm.63, \ P=.02)\), as well as on Day 2 than on Day 3 \((M=5.28\pm.70, \ P=.00)\). Although a day x temperature interaction in unpleasantness did not reach significance in experimental subjects \((F_{6,168}= 1.98, \ P=.07)\), mean unpleasantness ratings decreased across day with the most magnitude at 42°C as well. At 48°C, decrease in ratings of intensity was almost significant \((P=.06)\) from Day 1 \((M=11.49\pm.60)\) to Day 3 \((M=10.08\pm.54)\).
A main effect of scale was also found in controls ($F_{1,16}=36.53, P=.00$) in that their intensity ratings ($M=12.24+.60$) were significantly higher than unpleasantness ratings ($M=9.53+.73$). A significant scale x day x sex x athlete interaction ($F_{2,32}=4.43, P=.02$) revealed that all control subjects except male non-athletes gave significantly higher intensity ratings than unpleasantness ratings each day ($P < .05$ for difference between intensity and unpleasantness ratings each day for male athletes, female athletes and female non-athletes) (see Table 2). Male non-athletes only showed intensity ratings that were significantly higher than unpleasantness ratings on Day 2 ($P=.01$).

**Table 2**

<table>
<thead>
<tr>
<th></th>
<th>Female Athletes</th>
<th>Female Non-Athletes</th>
<th>Male Athletes</th>
<th>Male Non-Athletes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Day 1</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>I:</strong></td>
<td>12.95±2.44</td>
<td>14.95±2.44</td>
<td>12.65±2.44</td>
<td>10.35±2.44</td>
</tr>
<tr>
<td><strong>U:</strong></td>
<td>9.30±3.10</td>
<td>11.45±3.10</td>
<td>9.45±3.10</td>
<td>9.00±3.10</td>
</tr>
<tr>
<td><strong>Day 2</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>I:</strong></td>
<td>12.40±2.80</td>
<td>13.95±2.80</td>
<td>10.90±2.80</td>
<td>12.05±2.80</td>
</tr>
<tr>
<td><strong>U:</strong></td>
<td>8.65±3.51</td>
<td>11.95±3.51</td>
<td>8.40±3.51</td>
<td>9.75±3.51</td>
</tr>
<tr>
<td><strong>Day 3</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>I:</strong></td>
<td>11.45±2.40</td>
<td>13.95±2.40</td>
<td>10.85±2.40</td>
<td>10.45±2.40</td>
</tr>
<tr>
<td><strong>U:</strong></td>
<td>8.30±2.62</td>
<td>11.60±2.62</td>
<td>7.45±2.62</td>
<td>9.05±2.62</td>
</tr>
</tbody>
</table>

Furthermore, the Day 1 intensity ratings of male athlete controls were significantly higher than their Day 2 and Day 3 intensity ratings ($P=.04$ and $P=.00$, respectively) and their unpleasantness ratings on Day 1 also were significantly greater than on Day 3 ($P=.02$). Female athlete controls showed a significant decrease of intensity ratings from Day 1 to Day 3 as well ($P=.01$). Conversely, male non-athlete controls showed a significant increase in intensity ratings from Day 1 to Day 2 ($P=.05$).

*Cold Pressor*
Pain ratings were not significantly different across day for experimental or control subjects.

A significant sex x athlete interaction was found in controls ($F_{1,16}=8.05, P=.01$) in that female non-athletes gave significantly higher pain ratings ($M=103.17 \pm 7.30$) than female athletes ($M=71.7 \pm 7.30, P=.00$). (Fig.7) Female non-athletes in the experimental group also gave the highest pain ratings relative to other experimental subjects (on their baseline day), although their ratings were not significantly higher than any other group (See Fig. 8).

Both experimental and control groups showed a main effect of scale ($F_{1,28}=17.53, P=.00$ experimental, $F_{1,16}=9.73, P=.01$ control). The main effect of scale in both experimental and control groups was that intensity ratings ($M=84.34 \pm 4.23$ for experimental, $M=90.15 \pm 3.48$ for control) were significantly higher than unpleasantness ratings ($M=79.77 \pm 4.46, P=.00$ for experimental, $M=85.75 \pm 3.94, P=.01$ for control).

Discussion

The results of this study indicate that males and females do experience analgesia as a result of a competitive mindset. Both male and female experimental subjects had significantly lower pain sensitivity after competition than they had after exercise or at baseline on the heat pain threshold and thermal scaling tests. Sex did not affect basal pain sensitivity in either experimental or control conditions and athletic status decreased it in female controls on the cold pressor test only.

The success of the competition manipulation in eliciting a state of competition in the experimental group is evidenced against significant increases in heart rate, subjective arousal and perceived effort expended on competition day compared to exercise day.
Although subjective arousal as measured by the Body Awareness Questionnaire (BAQ) was not significantly higher on competition day than exercise day for all experimental subjects, male athletes showed significantly higher subjective arousal scores after competition than they did on exercise (whereas repeated testing controls’ BAQ scores actually decreased significantly from Day 1 to Day 3) (Fig.2). Similarly, athletes perceived themselves as expending significantly more effort on the competition day than they did on exercise day (Fig.3). No subjects indicated that they had known of the deception when asked after debriefing.

In checking for internal validity, one notes that skin of experimental subjects was significantly colder on competition day than it was on baseline day. This result alone implies that the lower pain ratings given by experimental subjects in response to noxious thermal stimuli on competition day could have been due to their colder skin. However, because skin temperature was only significantly colder between competition and baseline day, whereas differences in pain sensitivity were significant between competition and exercise day, the variance of skin temperature does not correspond to the variance of pain sensitivity. This temporal mismatch of magnitude of variance between skin temperature and thermal pain ratings indicate that cooler skin is not the reason subjects gave lower pain ratings in response to thermal pain on competition day.

The finding of significantly decreased pain sensitivity in repeated-pain-testing controls appears to threaten the study’s validity as well since it suggests that the decrease in pain sensitivity in the experimental group may just be due to repeated pain testing and not to the experimental manipulation. However, due to where these significant
differences in controls fell temporally, on both thermal scaling and heat pain threshold
tests, the validity of the manipulation is not compromised.

On the heat pain threshold test, the significant effect in controls was a higher heat pain threshold on Day 1 than on Day 3 in athletes. However, the pain sensitivity change in the experimental condition does not correspond to the change in control condition because whereas heat pain threshold in repeated-pain-testing athlete controls only elevated significantly in between Days 1 and 3, the entire experimental group experienced elevated heat pain threshold between Days 2 and 3. Therefore the rise in heat pain threshold of experimental athletes, because it occurred between a smaller time interval, does not temporally map onto the rise in control athletes. Also, the significantly higher heat pain threshold of controls on Day 3 is an effect isolated to control athletes, which indicates that the heat pain threshold change observed in experimental non-athlete subjects is not due to repeated pain testing. Furthermore, because all subjects in the experimental condition and not just athletes did experience a significant rise in heat pain threshold between exercise and competition, evidence of analgesia after competition remains.

On the thermal scaling test, the significant effect in the control condition was that male and female athlete controls gave significantly lower pain ratings across day. Male athletes’ intensity ratings were greater on Day 1 than on Days 2 and 3 and their unpleasantness ratings decreased between Days 1 and 3. Also, the intensity ratings of female athlete controls also decreased significantly between Days 1 and 3.

Firstly, the decrease in unpleasantness ratings across day given by control male athletes can be disregarded as a factor responsible for parallel decreased pain sensitivity
in experimental male athletes since only ratings of intensity significantly decreased across day in experimental subjects; decrease in unpleasantness cross day did not even reach significance in experimental subjects. Secondly, experimental subjects gave significantly lower intensity ratings on Day 3 than on Day 2 whereas there was never a difference between Days 2 and 3 observed in controls. Thus difference in pain sensitivity between exercise and competition is not due to a repeated pain testing effect which decreases sensitivity between 2nd and 3rd time pain tested. In sum, the validity of the manipulation appears uncompromised.

A main finding of the current study is that both men and women experienced similar magnitude of analgesic response after exercise and after competition. Thus the analgesia that women experience after athletic competition is due to the competitive component as much as men’s. This finding that men and women produce similar analgesia in response to exercise was not expected because past research indicated that females produce more analgesia than males in response to exercise (Sternberg et al. 2001, Koltyn et al., 2000) and because in past research, only men showed analgesia after a competition without exercise (a videogame competition, Sternberg et al. 2001). So although females and males had in the past shown equal strength analgesia after athletic competition (Sternberg et al, 1998), it was reasoned that women’s greater analgesia in response to exercise could have accounted for the similar analgesia between the sexes after physical competition (assuming that men’s analgesia was due more to the competitive component whereas female analgesia was more due to the exercise component). To reconcile past research to the fact in this study women experienced the same amount of analgesia as men in response to both the competitive and exercise
component of athletic competition, one may consider the different type of competition and parameters of exercise which researchers employed in the past.

The type of competition used in past research was likely ineffective at inducing analgesia in females because it was a videogame competition, which is a type specifically excitatory to males. Since videogame competition is not generally a personally meaningful competition to females, it did not produce a competitive mindset of which females are capable—one which can elicit analgesia. The competition used in this study, in contrast, was one which is not specifically more excitatory to males by merit of its type. Thus the type of competition in this study, one personally meaningful to females, effected a competitive mindset in females which allowed an analgesia induced by that mindset to emerge.

The ability of competition as a stressor to induce analgesia is supported strongly by results. Experimental subjects experienced significantly greater analgesia after competition than they did after exercise on thermal scaling and heat pain threshold tests. Furthermore, because exercise alone was not enough to elevate heat pain threshold yet competition was, the potency of competition as an analgesia-inducing stressor is emphasized.

Although heat pain threshold and thermal scaling tests showed analgesia, the cold pressor test in this study did not show the expected effect of decreased pain sensitivity in response to exercise and competition. In fact, it did not register any significant effects in the experimental condition. This finding contrasts with the finding of Sternberg et al. (1998) of a highly significant effect of athletic competition on the cold pressor test. The reason that cold pressor test did not find significant analgesia in response to athletic
competition in the current study is most likely due to experimental procedure which made
the cold pressor test the last test administered. Analgesic effects had probably diminished
in the circa. 10 minute period which passed between exercise and administration of the
cold pressor test. This amount of time between the end of the analgesia-inducing stressor
and the cold pressor test might have compromised the amount of analgesia still
detectable. To discover if this is the case, a replication of this study’s manipulations with
cold pressor test administered immediately after exercise would suffice.

The finding that subjective measures of stress were sex and athletic status
dependent (Fig. 2), whereas pain alterations were not, indicate that either the subjective
measure of stress used was not a highly accurate indicator of stress level, or analgesia is
related only to a specific dimension of stress which was not separated out in the
subjective measure. Specific dimensions of stress to which analgesia might be more
specifically related (and along which sex and athletic status differences are not manifest)
are competitive and exercise stress. The subjective response, however may have
measured the aforementioned stresses along with other stresses, like social anxiety stress
and academic stress, that physically manifest themselves enough to be measured by the
Body Awareness Questionnaire as well. Subjects’ response to the subjective measure
also may have been tailored to standards of social desirability.

Similarly, subjective ratings of perceived effort differed significantly between
athletes and non-athletes, yet pain alterations did not (Fig. 3). Although athletes
perceived themselves as putting in significantly more effort on competition day than
exercise day whereas non-athletes perceived themselves as putting in the same effort,
both athletes and non-athletes experienced similar strength of analgesia in response to
exercise and competition. Between-subject variations of perceived stress of competition do not manifest as variations of analgesia. This indicates that an analgesic mechanism triggered by competition is triggered equally for all subjects regardless of athletic status.

The unexpected finding that men and women showed equal strengths of analgesia after exercise in this study is in contrast to past research which has indicated that women show greater analgesia in response to exercise than men. One possible reason for this discrepancy is that experimenters in the past used different exercise parameters. Females have shown a greater analgesic response to exercise in past research after running for 10 minutes at 85% max heart rate (Sternberg et al., 2001) and after isometric exercise (Koltyn et al., 2001), yet they did not show greater analgesia than males after this study’s exercise of pedaling on a bike for 20 minutes at 60% maximum capacity (as determined by the max. capacity test). Because differing intensity of exercise employs different analgesic systems to become activated (opioid or nonopioid), it is possible that the different parameters of exercise in other studies caused activation of a different analgesic system in females which resulted in their stronger analgesic response. Also, because females have shown significantly stronger and prolonged analgesia to κ opiates, females in other studies may have experienced activation of the same analgesic system as males (an analgesic system different than the one activated in this study) more intensely (Gear, Miaskowski, et al, 1996 as cited by Miaskowski, 1999). More research into what analgesic systems (opioid or nonopioid, for example) are activated to what extent by given exercise parameters would benefit understanding this issue. Using opiate receptor blockers such as naloxone would help determine whether observed sex difference in response to exercise is due to activation of neurochemically disparate analgesic systems.
Additionally, sex differences in response to exercise are not always observed (Kanarek, Gerstein, Wildman, Mathes, & D’Anci, 1998, Koltyn, Trine, Malani, Stegner, & Tobar, 2001, Poudevigne, O’Connor, & Pasley, 2002). Koltyn et al. (2001), for example, found that women experienced greater analgesia (on a thermal pain measure) after “submax” isometric exercise whereas there were no sex differences after “maximum” isometric exercise.

Also averse to expectations, a sex difference in basal pain sensitivity was not found in this study. The expected lower basal pain sensitivity in males was only closest to being evident in repeated testing controls on one type of pain test (cold pressor) when male and female non-athletes were compared (p = .07). Because sex difference in pain sensitivity only came close to emerging at repeated baseline measurements (Fig. 7) and not in the experimental condition (Fig. 8), it suggests that a stressor like exercise or competition may bring men and women to a common level of decreased pain sensitivity and that a sex difference in basal pain sensitivity may be more evident in response to cold pressor pain (than to thermal pain). However, since past research shows no such trend of more significant sex difference on the cold pressor test in particular, and most findings support a decreased basal pain sensitivity in males, I can only fathom that this study did not produce significant results because the sex difference is a subtle effect and to elicit it requires a greater sample size.

However, an athletic-status-dependent sex difference in basal pain sensitivity was elicited in controls in that female non-athletes gave significantly greater pain ratings than female athletes in response to cold pressor and males did not exhibit such a difference (Fig. 7). This finding, that athletic status only decreased basal pain sensitivity in females,
contrasts with past research’s findings of significant effect of athletic status on basal pain sensitivity regardless of sex (Hall & Davies, 1991, Sullivan et al., 2000, and Sternberg et al., 2001). However, Hall & Davies (1991) produced a finding similar to this study’s in that their female non-athlete subjects reported significantly higher pain ratings than female athletes as well. However, their female non-athlete group also gave significantly higher pain ratings than male athletes and non-athletes, an effect not present in our study. Because our results in the experimental condition on baseline day show female non-athletes once again to have the highest pain ratings (though non-significant), our study helps confirm that athletic status is at least a significant variable with regard to female basal pain sensitivity in response to cold pressor. Why our study did not find a parallel effect of athletic status on male basal pain sensitivity in response to cold pressor could be because although athletic status can have an effect on male basal pain sensitivity, it is a weaker effect in males. A possible reason for a stronger effect of athletic status in females could be the fact that males in our culture are socialized to be athletic more than females, thus making even “non-athlete” males more athlete than non-athlete females (Koivula, 1995).

The lack of effect of athletic status on pain sensitivity in the experimental group was an unexpected finding. Differences in basal pain sensitivity, at least in regards to athletic status, did not persist when subjects were stressed by either exercise or competition. The effect of athletic status in this study can therefore be seen as not strong enough to affect those stressed by exercise. Such a result suggests that when the body confronts an analgesia-inducing stressor, it adjusts pain sensitivity to a similar level across subject populations with different basal pain sensitivities.
The effect of athletic status was also confined to one stimulus pain measure: cold pressor. This finding that the effect of athletic status is primarily evident in response to cold pressor pain more than other types of stimulus pains used to measure pain sensitivity (such as thermal pain measures like heat pain threshold and thermal scaling) is consistent with past research (Hall & Davies, 1991, Sternberg et al., 2001, Sullivan et al, 2000). Failure to find difference in basal pain sensitivity between athletes and non-athletes on tests other than the cold pressor test is also consistent with past research which failed to find an effect of athletic status on thermal and ischemic pain measures (Janal, Glusman, Kuhl, & Clark, 1993). Since athletes are possibly exposed to cold pain more due to the routine icing of injuries, this effect of athletic status on cold pressor may be the result of habituation to a commonly encountered stimulus. Future research which takes into account athletes’ past exposure to cold pain could elucidate whether decreased pain sensitivity to cold pain is due to past cold pain confrontation. A control group for such a future study should include non-athletes who have had to repeatedly ice injuries.

Another interesting effect that athletic status had on pain sensitivity indicates that athletes more quickly adapt to pain in general (and are adapted to cold pain). This effect emerged in repeated-pain-testing controls on both thermal scaling and heat pain threshold tests. In heat pain threshold, athlete heat pain threshold on Day 3 was significantly lower than it was on Day 1, whereas non-athletes did not have significantly lower heat pain threshold on Day 3. Similarly on thermal scaling, female athletes gave significantly lower intensity ratings on Day 3 than Day 1, whereas female non-athletes did not. Male athletes’ intensity and unpleasantness ratings on thermal scaling also significantly decreased across day, whereas male non-athletes’ did not.
Together these findings suggest that athletes are better able to cope with repeated pain exposure. They also support the notion that whatever lower pain sensitivity athletes may have is due to their frequent confrontation with pain as in general (and hence not just adaptation to the cold type of pain) in athletic training. Therefore athlete confrontation with pains commonly encountered in the course of athletic training, such as muscle pain or cold pain, may enable athletes to more easily adapt to other types of pain (such as the thermal pain presented by our procedure). Because it is not known how adaptation to specific pain (cold pain, muscle pain) enables a quicker adaptation in response to another type in the same category (thermal pain), future research could address this question.

Another question for future research to potentially investigate is whether athletes’ faster rate of adapting to pain developed with exposure to pain, or has just always existed as a “natural talent” of the nervous system of people who choose to be athletes. Research attempting to answer this question should address what factor is responsible for athletes’ reduced response to repeated presentation of painful stimuli. Whether ability to reinterpret painful stimuli or something else altogether is the main factor responsible, researchers should proceed to see if potential factors (such as reinterpretation ability) are variables which significantly alter in response to frequency of exposure to pain. If they do, and they do so in a magnitude that statistically corresponds to observed individual differences in pain adaptation rate, then the dominant reason athletes better cope with pain is not that such ability is their “natural talent”.

Although it is almost impossible to empirically dismiss “natural talent”/present-at-birth-and-stable-thereafter ability as responsible for differences in pain perception and coping, data imply that frequency of past exposure to pain is the reason. Since a strong
trend of decrease in pain sensitivity to repeated presentation of stimulus pains existed for
all subjects in the study, all subjects adapted to pain the more frequently they were
exposed to it. Thus previous exposure to pain is a variable which has shown itself to be
significant in decreasing pain sensitivity between trials and possibly significant in
decreasing pain sensitivity between non-athlete and athlete subject populations. Whereas
it makes sense that a nervous system which more frequently encounters pain would have
a more developed response to it, it makes little sense that—and is not immediately
apparent how-- ability to reinterpret painful stimuli as less severe (perhaps a higher order
cognitive task) would be present at birth rather than learned with experience.

Contrary to expectations, greater male testosterone level did not cause males to
exhibit greater analgesia in response competition. Although males have a much higher
testosterone level than females, the magnitude of the analgesia induced by competition in
our study was the same or both sexes. This finding that testosterone does not make
competition a more able inducer of analgesia is puzzling when one considers past
research which suggests testosterone is proportional to competitive drive (Mazur, Booth,
& Dabbs, 1992). Although competitive drive does increase analgesia (as evidenced by
our result of greater analgesia after competition than just exercise as well as by the
analgesia induced by a sedentary videogame competition (Sternberg et al, 2001)), it was
expected that males would possess more of a competitive mindset and consequently
stronger analgesia. However, our results suggest that the “amount” of competitive drive
which is contributed by testosterone does not cause more of a competitive mindset or
stronger analgesia in males. However, the role of testosterone in the current study may be
significant to results, but not in the way that was initially reasoned. Unlike previously
thought, testosterone is not a variable that allows males to experience a greater magnitude of analgesia after competition. However, it may be strong enough a variable to change pain sensitivity by a less direct route which is more clearly evidenced through our understanding of how testosterone affects athletic status, and how athletic status affects pain sensitivity in turn. Therefore although testosterone does not manifest itself directly as a force immediately proportional to analgesia after competition, it is possible that it acts significantly as a force by encouraging athletic behavior. Due to increased male engagement in such behavior, it is possible that male non-athletes are more “athlete” than female non-athletes. Hence male non-athletes might have confronted the pain associated with athletic training more than female non-athletes, and consequently all difference in pain sensitivity cross athletes and genders may be simply due to the single variable of past exposure to pain.

Attribution of greater athletic status to males derives from male socialization as well as greater male testosterone level. Because males in our culture are socialized to be athletic more than females (Koivula, 1995), they are more likely to engage in athletic activity as they conform and meet cultural expectations. This effect of socialization upon athletic behavior, however, is compounded with the effect greater male testosterone level has upon athletic behavior. Empirical demonstration that testosterone increases athletic behavior can be found in studies of girls with Congenital Adrenal Hyperplasia (CAH). These girls, who are exposed to more testosterone in the womb yet socialized as girls, show significantly more rough and tumble play as children (Hines & Kaufman, 1994). On both child and parent report, CAH girls scored higher than control girls on athletic play. Therefore testosterone and socialization encourage athletic behavior in males. So
although males may not officially be athletes, the amount of athletic activity they engage/have engaged in is probably significantly greater than that of females.

Furthermore, data from the current study possibly support testosterone’s contribution to athletic status. On the cold pressor test, a pain measure consistently shown to be particularly susceptible to differences in athletic status, female athletes of our study gave significantly lower pain ratings than female non-athletes, whereas the difference between male athletes and male non-athletes was not significant. Thus the speculated, unofficial athletic status of male non-athletes may have been the reason that difference of official athletic status did not affect a difference in pain ratings between males as much as it did in females.

Longitudinal studies which correlate frequency of exposure to pain and pain sensitivity might further determine the strength of pain sensitivity’s dependence on that single variable. Performing such studies have the potential to empirically demonstrate that sex and athletic difference in pain sensitivity is due to the single variable of past exposure to pain.
Figure 1

Heart rate increases after exercise and more so after competition. For all experimental subjects, heart rate increased significantly between Day 1 to Day 2, and again between Day 2 to Day 3. Heart rate of control subjects did not differ across day.
Figure 2

Subjective Arousal as measured by BAQ changes across day. Experimental subjects were significantly less aroused at baseline than they were on Day 2 or Day 3. Conversely, controls were significantly more aroused on Day 1 than they were on Day 2 or Day 3. Sex x athletic status interaction in the experimental group: Male athletes were significantly more aroused on competition day than at baseline, and female non-athletes were significantly more aroused on both exercise and competition days than they were at baseline.
Figure 3

Ratings of perceived effort across day. RPE significantly increased between exercise and competition for athletes, but not non-athletes.
Figure 4
Heat pain threshold across day. Heat pain threshold on Day 3 significantly heightened from Day 2 and baseline levels for experimental subjects, whereas controls did not differ significantly across day.
Figure 5

Pain ratings at 42°C across day. Pain ratings at 42°C were significantly lower for experimental subjects on Day 3 than they were on Day 2 or Day 1, whereas controls did not give significantly different pain ratings cross day.
Figure 6

Pain ratings in response to different temperatures across day. All subjects gave significantly higher pain ratings with increase in temperature. Experimental subjects gave, at 42°C, significantly lower pain ratings on Day 3 than they did on Day 1 and Day 2, whereas controls did not give significantly different pain ratings cross day at that temperature.
Figure 7
Control cold pressor ratings as a function of sex and athletic status. Control female non-athletes gave significantly higher CP pain ratings than female athletes.
Table: Cold pressor ratings of baseline day experimental male and female athletes. Experimental female non-athletes gave non-significantly higher CP pain ratings than female athletes or male athletes and non-athletes.
References


Competition vs. Exercise-Induced Analgesia


Appendix A  Body Awareness Questionnaire

Subject #: __________________________  Time: __________________________
Date: __________________________  Day/Condition: __________________________

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

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

CONSENT FORM

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

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

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

Pain testing involves the following:

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

We will ask you questions about your health and diet, and will periodically also ask you to rate your subjective perceptions of nervousness and anxiety. We will have you provide us with saliva samples on each occasion for analysis of hormones, and we will measure your heart rate and blood pressure during each session.
You will not be identified by name in the data presentation. You will be informed of the study hypotheses (and results, if interested) after participation in the experiment. Please do not discuss the study procedures with anyone until after the study is concluded.

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

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

Signature: ________________________
Date: ______
CONSENT FORM

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

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

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

Pain testing involves the following:

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

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

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

You will be asked to perform the following exercise tasks:

- Today your fitness level will be tested by performing a ~10-minute exhausting bout of cycling exercise. During the two remaining sessions, your exercise task will consist of a 20 min cycling bout, while trying to maintain a particular cycling intensity.
We will ask you questions about your health and diet, and will periodically also ask you to rate your subjective perceptions of nervousness and anxiety. We will have you provide us with saliva samples on each occasion for analysis of hormones, and we will measure your heart rate and blood pressure during each session.

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

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

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

Signature: ________________________  
Date: ______
Appendix C

Medical History Questionnaire

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

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

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

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

Gracely Box Scales: Intensity

0
1 2 3
FAINT

4
5 6
WEAK

7
8 9 10
MILD

11
12 13 14 15 16 17 18 19 20
EXTREMELY INTENSE

VERYSLIGHTLY INTENSE

BARELY STRONG

MODERATE

VERY WEAK

VERY MILD

MILD

SLIGHTLY INTENSE

STRONG

INTENSE

VERY INTENSE

EXTREMELY INTENSE
Appendix E: Gracely Box Scales: Unpleasantness

20
19
18
17
16
15
14
13
12
11
10
9
8
7
6
5
4
3
2
1
0

VERY INTOLERABLE
INTOLERABLE
VERY DISTRESSING
SLIGHTLY INTOLERABLE
VERY ANNOYING
DISTRESSING
VERY UNPLEASANT
SLIGHTLY DISTRESSING
ANNOYING
UNPLEASANT
SLIGHTLY ANNOYING
SLIGHTLY UNPLEASANT

NEUTRAL