THE PLACEBO EFFECT: INFLUENCE ON RECOVERY DURING REPEATED INTERMITTENT SPRINTS

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ABSTRACT

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Despite the available literature addressing the placebo effect regarding performance, there is a paucity of research addressing the possibility of a placebo effect both within and between bouts of repeated sprint performance. Therefore, the purpose of this study was to determine if the administration of a placebo influences recovery during and between sessions of intermittent sprinting. Ten sprint-trained participants performed a series of repeated sprint tests under two different conditions in a counterbalanced order; one where they were administered a control beverage (i.e., water) and another with an experimental beverage (i.e., placebo). Each testing session consisted of three repeated anaerobic sprint tests (RAST) on a non-motorized treadmill with seven minutes of recovery between sets. Participants were then given 24-hours of recovery whereupon the repeated sprint test protocol was repeated. Ratings of perceived exertion were recorded within five seconds after each sprint. Following each RAST, participants were asked to rate their pain using a visual analog scale which was followed by a blood lactate sample drawn. Participants were then asked to rate their recovery using the perceived recovery status scale 30 seconds before beginning each subsequent RAST. Power was recorded throughout each session from a non-motorized treadmill in order to analyze changes in sprinting performance. Approximately 20 minutes following the final sprint, participants provided a session rating of perceived exertion to rate the global difficulty of the session. A 2x3 repeated measures ANOVA was used to determine significant differences in peak power, mean power, decrement score, recovery score, perceived recovery status, rating of perceived exertion, pain, and blood lactate
between RASTs and conditions. Results from the analyses revealed no significant differences between conditions during the day one testing session. However, results showed the experimental condition produced significantly higher peak power ($p < 0.001$) and mean power ($p = 0.002$) versus the control condition in RAST$_3$ of the day two session. These findings were observed in absence of any other significant difference in metabolic or perceptual strain ($p > 0.05$). In conclusion, it appears the administration of a placebo can attenuate the decline in performance as fatigue increases during repeated sprinting bouts.
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The Placebo Effect in Recovery

Recovery is gaining attention in the literature and represents a growing market for nutritional and supplement companies (Bishop, Jones, & Woods, 2008; Nutrition Business Journal, 2012). The ability to recover within or after a session of repeated, high-intensity exercise is critically important in sport, with studies showing a positive correlation between recovery and subsequent performances (McLester, Bishop, Smith, Wyers, 2003; Glaister, Stone, Stewart, Hughes, Moir, 2005; Jones, Bishop, Richardson, & Smith, 2006; Laurent et al., 2011). Moreover, many sports have intermittent play periods interspersed with recovery periods between bouts of high-intensity efforts where success is dependent on an individual’s ability to recover to maintain optimal level of effort. Additionally, within sports that typically incur events on consecutive days (e.g., tournament play, etc.) it is important for athletes to be recovered and to optimize performance with perhaps only an overnight period of recovery (Montgomery et al., 2008). However, there is a lack of data that notes the impact of a placebo effect on recovery either within a session or on a subsequent day of exercise. This is surprising given the importance and growing interest in recovery in sport and exercise performance with the concomitant interest in supplements intended to augment recovery but is worthy of merit.

Overview of the Placebo Effect

The placebo effect is broadly defined as any treatment or intervention aimed at precipitating positive psychological and/or physiological effects (Shapiro & Morris, 1978; Sher, 1997; Peck & Coleman, 1991). Interestingly, the resultant changes associated with the treatment are most likely a result of the belief and/or expectancy of the intervention to actuate the outcome and not the implicit results of the treatment or intervention (Foad, Beedie, & Coleman, 2008).
That is, the treatment is fundamentally inert but the individual’s belief in the efficacy of the
treatment will most likely elicit the positive changes and manifest the desired physiologic and/or
psychological benefit. Within the literature there have been multiple theories aimed to identify
the factors that may mediate the placebo effect (Pollo, Carlino, & Benedetti, 2011; Peck &
Coleman, 1991; Benedetti, 2006; Benedetti et al., 2003). It seems likely, though, that the placebo
effect may result from interactions among motor, cognitive-verbal, and physiochemical
responses that stem from classical conditioning and/or the expectancy theory and the resultant
activation of endogenous opiates (Benedetti et al., 2003; Benedetti, 2006; Peck & Coleman,
1991; Stewart-Williams & Podd, 2004).

Briefly, classical conditioning is a process in which a neutral stimulus is associated to and
presented with an unconditioned stimulus. A learning effect occurs when a neutral stimulus is
conditioned to cause the same physiological response as an unconditioned stimulus. The
expectancy theory, as it relates to the notion of a placebo effect, states that a resultant change in
performance is largely mediated to the degree that an individual who consumed the treatment
(i.e., placebo) believes it to be beneficial (Beedie, Stuart, Coleman, & Foad, 2006; Benedetti,
2006; McClung and Collins, 2007; Stewart-Williams & Podd, 2004). These improvements in
performance are most commonly associated with the analgesic effect (i.e., pain mediating) that a
placebo is known to induce (Petrovic, Kalso, Petersson, & Ingvar, 2002). While the classical
conditioning and expectancy theories are often viewed as mutually exclusive, it may be that
these theories work synergistically to elicit changes (Stewart-Williams & Podd 2004).

Petrovic, Kalso, Petersson and Ingvar (2002) employed verbal expectancy and opioid
preconditioning to examine the similarity in brain activation during placebo vs. opioid analgesia.
Their findings confirm that the anterior cingulate cortex, an area of the brain with a high
concentration of opioid receptors, is similarly activated during placebo and opioid analgesia trials. This suggests that a placebo may elicit the same neural response associated with decreases in pain sensitivity owing to the binding of opioid receptors. This phenomenon is strikingly similar to that associated with endogenous or exogenous opioid analgesia. Indeed, Benedetti et al. (2003) found that the combination of verbal expectancy and preconditioning with an analgesic injection significantly increases the placebo response when compared to verbal suggestion alone. More recent studies have shown that by using conditioning and/or verbal expectancy, neural changes, similar to those seen after delivery of an analgesic, have been observed (Zubieta, Yau, Scott, & Stohler, 2006; Wager et al., 2004). Moreover, studies have found that pain sensation can negatively impact performance (Farina, Arendt-Nielsen, Merletti, & Graven-Nielsen, 2004; Graven-Nielsen, Svensson, & Arendt-Nielsen, 1997). For example, a negative correlation ($R = -0.45$) between pain sensation and motor unit firing rates has been shown (Farina et al., 2004) while others report a negative effect on time to exhaustion when comparing an experimentally induced pain group to a no pain group (Graven-Nielsen et al., 1997). Collectively, all of this evidence suggests that administration of a placebo may allow an individual to maintain performance by mediating pain and/or fatigue. Theoretically, then, this may allow sustained performance at levels, where under normal conditions, the individual would have exhausted.

The Placebo Effect in Sport and Exercise Performance

Compared to the neurophysiological benefits of the placebo effect identified in mainstream medical and therapeutic fields, there is substantially less known about the placebo effect relative to sport and exercise performance. This is surprising considering the relative interest from researchers and general population. However, there have been a limited number of studies over the past 10 years aimed at quantifying the placebo effect. Beedie, Stuart, Coleman,
and Foad (2006) found that performance during a 10km cycling time trial was positively affected depending on level of expectancy by increasing perceived doses of caffeine. In that study, individuals were deceived to believe that they were administered a caffeine treatment of either 4.5 mg/kg or 9 mg/kg, however all received a placebo. Results revealed an increase in power of 1.3% and 3.1% with increasing dose expectancy (i.e., 4.5 and 9 mg/kg, respectively). Interestingly, there was a reported decrease of 1.4% during the time trial when individuals did not receive the expected dose. In a subsequent study, Beedie, Coleman, and Foad (2007) examined the role of a placebo effect during repeated sprint work. In that study, a placebo group was informed the treatment was ‘proven’ to improve repeated sprint performance whereas another group was informed that the pill would have a detrimental effect on sprint performance, referred to as a “nocebo”. Results show that the nocebo group’s mean sprint performance was negatively impacted while the placebo group’s mean sprint performance improved. Clearly, the available literature seems to indicate that not only the level of expectancy but the direction of expectancy can impact performance. While these studies are novel and reinforce the importance of generating expectancy when using a placebo, there lacks standardized procedures as to how to generate or increase expectancy. For example, Beedie, Stuart, Coleman, and Foad (2007) provided their participants with a pamphlet summarizing findings of published research detailing the positive role that caffeine plays in cycling performance. Additionally, Porcari and Foster (2006) attempted to manipulate participant expectancy by showing participants a brief video that detailed performance benefits that super-oxygenated water may cause. Regardless, it seems that the manner in which an individual is informed of the efficacy of the treatment is not as important as to the degree to which they believe the treatment to work.
The inclusion of ratings of perceived exertion during exercise or sport may provide further support regarding a placebo effect. In essence, RPEs are noninvasive ratings used to assess the psychobiological state of a person at a given moment (Borg, 1982). A primary benefit associated with RPEs is the integrative nature in which they are mediated by both physiological variables and the psychological state of a person to determine the level of exertion (Robertson & Noble, 1997). It has been proposed that RPE is influenced via peripheral afferent information (e.g. blood lactate levels, blood/muscle pH, mechanical strain, muscle damage, pain) which are sent to and interpreted by the brain (Jameson & Ring, 2000; Hampson, St Clair Gibson, Lambert, & Noakes, 2001; Faulkner & Eston, 2007). More recently, the perception of recovery has been studied and has been shown to negatively correlate ($R^2 = .59$) with muscle damage (Sikorski et al., 2013) following high-intensity resistance training, and has also been shown to ‘predict’ performance decrement during repeated sprint work (Laurent et al., 2010). Because interpretations of pain may be reduced if placebo induced analgesia occurs, the measurements of RPE and perception of recovery between a placebo group and control group who complete the same pain inducing exercise seems worthy of merit.

Therefore, the purpose of this study was to determine the effect that the administration of a placebo has on recovery from both a perceptual and performance standpoint during and between sessions of repeated sprint work. It was hypothesized that individuals in the placebo group will demonstrate improved recovery and/or performance at parallel time points when compared to the control group.
Defining the Placebo and Placebo Effect

The placebo is a sensible phenomenon to use as an example of the life history of an artifact (McGuire, 1969). McGuire’s model consists of three progressive stages following the progression of an artifact as it gains merit deeming it worthy of research or scientific inquiry. The first stage is ignorance, and in regards to the placebo effect, ignorance ranged from the early 19th century until the mid-20th century. At its origins, physicians administered placebos stemming from the calming effect that it had (i.e., pain modulation). Interestingly, during this period physicians credited its effect as a result of pure psychological changes in the patient’s perceived pain. Then, the 1950’s and 1960’s ushered the second stage of McGuire’s model, the coping stage. This stage is characterized by the existence of an artifact being recognized by the scientific community and, consequently, ‘controlled for’ in research. The final stage of an artifact is referred to as the exploitation stage where an artifact is studied on its own (Kirsch, 2011). This relatively new interest in the placebo effect has since spurred the proliferation of scientific research aimed to understand the placebo. Chief among these aims are the identification of the mechanisms that drive an inert substance’s ability to cause true physiological and psychological changes.

While not universally accepted, the placebo effect is generally defined as a physiological and/or psychological outcome of a treatment not brought about by the treatment itself but from an expectancy or belief in the treatment to provoke an outcome (Shapiro & Morris, 1978; Sher, 1997; Peck & Coleman, 1991; Stewart-Williams & Podd, 2004). Due to popular culture’s perception of the placebo and how the placebo is used in the medical field, contemporary associations of the word tend to precipitate a negative or, at the very least, inert connotation.
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(Benson & Friedman 1996; Pallanti, 2013). For example, the medical field will often test an active drug against a placebo to determine the effectiveness of the drug as it is compared to an inert modality. However, research has clearly shown genuine physiological effects when an ‘inert’ drug, treatment, or procedure is administered. This, obviously, can be a confounding factor in the assessment of the efficacy of ‘active’ treatments. In turn, this has created a negative stigma and misunderstanding associated with the placebo. Indeed, it has led to some authors proposing that the terms placebo and placebo effect be abandoned altogether in favor of alternative words and definitions that better encapsulate the usage and mechanisms behind the placebo effect (Borkovec, 1985; Kirsch, 1985).

A targeted reason for the misunderstanding of the term ‘placebo effect’ stems from practitioners and researchers often struggling to clearly define it (Peck & Coleman, 1991). Indeed, Price, Finniss, and Benedetti (2008) posit that the difficulty in defining the placebo effect lies in the essential paradox surrounding the concept of a placebo. It is difficult, at best, to understand how an inert substance can produce genuine changes in the individual when the contents or treatment are not known to elicit observed changes that are subsequently observed in the individual. To that end, Stewart-Williams and Podd (2004) have attempted to address this paradox by defining placebo and placebo effect. They define a placebo as “a substance that has no inherent power to produce an effect that is sought or expected” and a placebo effect as “a genuine psychological or physiological effect, in a human or another animal, which is attributable to receiving a substance or undergoing a procedure, but is not due to the inherent powers of that substance or procedure”. Separately defining the two terms allows for a causal relationship to be realized wherein the inert treatment (i.e., placebo) causes genuine changes (i.e., placebo effect) independent of an active ingredient and procedure. That is, administration of a
placebo causes the belief and/or expectancy that, in turn, can influence the neurophysiology and/or biochemistry of the individual; for example pain modulation, decreased cortisol levels, increased growth hormone secretion. The resultant physiological and psychological effects are often times associated with the release of endogenous opioids, classical conditioning and/or expectancy by researchers, and furthermore have been shown to work together to create an analgesic effect (i.e., a diminished sense of pain) (Peck & Coleman, 1991; Amanzio & Benedetti, 1999; Benedetti, Arduino, & Amanzio, 1999).

It is of note that the definition given by Stewart-Williams & Podd (2004) does not state that administration of a placebo yields a directional effect (i.e., positive or negative). Rather, it simply states that a placebo causes an effect; the manifestation of a resultant positive or negative effect is subject to the individual. Consequently, a placebo can genuinely produce the positive effects of a drug as well as the negative side effects pertaining to a drug (Shapiro, Chassan, Morris, & Frick, 1974; Beedie et al., 2006; Duncan, Lyons, & Hankey, 2009). In realizing this phenomenon, the term ‘nocebo’ was coined by Kennedy (1961) to describe the negative effects following the administration of a placebo. Hahn (1997) has since expanded on that definition of nocebo by noting that a true nocebo effect can only be observed when individuals have a conscious expectation for a placebo to elicit a negative effect. This poses another level of complexity to the study of the placebo effect as there are documented cases of placebos yielding both positive and negative changes simultaneously (Shapiro, Chassan, Morris, & Fick, 1974). Indeed, the nocebo and nocebo effect have caused confusion because it stands to reason that an inert treatment administered to an individual may precipitate either a placebo or nocebo effect. Stewart-Williams and Podd (2004) call for a parsimonious naming system where ‘nocebo’ and ‘nocebo effect’ are abolished and ‘placebo effect’ and ‘placebo' remain, regardless of the effect
seen, (i.e., harmful or desirable). Clearly, much more clarity is needed in delineating the verbiage associated with a placebo or nocebo from the research community. While the ambiguity associated with the connotations of the terms of ‘placebo’ and ‘nocebo’ are noted, it is well-supported in the literature that individuals who demonstrate an expectancy of a drug or treatment to elicit change independently manifest psychological and/or physiological changes.

**Psychological Theories in the Placebo Effect: Expectancy versus Classical Conditioning**

Among the first noted placebo-controlled trials was one completed in 1800 by John Haygarth. In that time, a common modality to decrease symptoms of disease was to place metallic rods to the body. It was largely believed metal rods decreased pain via magnetic waves passing through the body. Haygarth, a skeptic of this modality, designed an experiment to determine if the rods decreased pain or if the rods influenced the individuals to perceive less pain. In one trial, Haygarth deceived his patients by treating them with wooden rods fashioned similarly to metal rods (i.e., the placebo). He then treated his patients using the metal rods (i.e., the treatment). Four out of the five patients indicated a feeling of relief in both the wooden and metal rod trials. Haygarth explained the results by stating, “an important lesson in physic is here to be learnt, the wonderful and powerful influence of the passions of the mind upon the state and disorder of the body” (de Craen, Kaptchuk, Tijssen, & Kleijnen, 1999). Haygarth implemented the preconditioning of prior, successful, metal rod modalities, and thus, his patients expected the treatment to work.

What Haygarth’s work spurred has now manifested the two main psychological mechanisms that underlie the placebo effect: the expectancy theory and classical conditioning. There are competing arguments in the research community about which of the two mechanisms exerts greater influence over the placebo. Indeed, literature notes the placebo effect is dependent
on classical conditioning (Ader & Cohen, 1975; Price & Fields, 1997; Voudouris, Peck & Coleman, 1990) while other studies defend that expectancy is the primary mediator to explain a placebo effect (Kirsch, 1991; Montgomery & Kirsch, 1997). While there is literature aimed to advocate both of these theories (i.e., the underlying psychological mechanism(s)), studies show either verbal expectancy or classical conditioning can elicit increased pain tolerance (Amanzio and Benedetti, 1999; Pollo et al., 2001). Indeed, when verbal expectancy and pharmacological pre-conditioning of a placebo are combined, it has been shown that the placebo analgesic (i.e., pain relief) effect is significantly greater than verbal suggestion alone (Benedetti et al., 2003; Voudouris, Peck, & Coleman, 1990). It may also be that the placebo effect a researcher aims to elicit may be dependent on the psychological mechanisms (e.g., classical conditioning and/or expectancy) employed. Benedetti et al. (2003) found that conscious expectancies are more fitting when eliciting a placebo effect on physiological processes such as pain perception and motor performance. In contrast, it seems classical conditioning provides a greater effect when a subconscious physiological effect occurs (e.g., secretion of growth hormone and inhibition of cortisol).

The expectancy theory, as it relates to the notion of a placebo effect, states that a resultant change in performance is largely mediated to the degree that an individual who consumed the treatment (i.e., placebo) believes it to be beneficial (Beedie, Stuart, Coleman, & Foad, 2006; Benedetti, 2006; McClung and Collins, 2007; Stewart-Williams & Podd, 2004). Researchers have employed a variety of techniques to increase the expectancy of a placebo to produce the desired effect, including: informational handouts (McClung and Collins, 2007), videos detailing purported benefits (Foster, Felker, Porcari, Mikat, & Seebach, 2004), and verbal cues from researchers (Kalasountas, Reed, & Fitzpatrick, 2007). Expectancy is most often employed in
placebo studies that involve human subjects because it is assumed that animals lack the cognitive ability to develop expectancies, though Stewart-Williams and Podd (2004) state that this is not necessarily the case.

Classical conditioning, also called Pavlovian conditioning, occurs when an unconditioned stimulus (US) is paired with a neutral stimulus (NS). The unconditioned stimulus causes a natural response, referred to as an unconditioned response (UC). The pairing of stimuli causes a learning effect in which the neutral stimulus becomes a conditioned stimulus (CS). The result is that the newly created conditioned stimulus replaces the unconditioned stimulus to cause a conditioned response (CR). For example, Ader and Cohen (1975) employed classical conditioning in rats to determine if a placebo intervention could impact the immune system. The researchers paired saccharine-flavored (NS) water with an immunosuppressant (US) for three days. The rats were then given saccharine-flavored water (CS) without the immunosuppressant. The rats still exhibited a decreased immune function (CR) without the immunosuppressant being present in the saccharine-flavored water. Ultimately, it is reasonable to conclude that the notion of a placebo effect is well-accepted both within the scientific and lay community. Beyond that, there exist many nebulous factors associated with the placebo effect; from a universal consensus on the definition of a placebo or placebo effect to the causal factor that manifests the effect.

**Neurophysiological Changes Associated with the Placebo Effect**

Though classical conditioning, expectancy and subsequent neurophysiological activation provide a theoretical foundation for *why* the placebo effect works, contemporary research is more concerned with *how* psychological processes facilitate brain structures and brain biochemistry changes to mediate placebo analgesia. Thus, there have been a number of studies conducted aimed at identifying the specific areas of activation and, subsequently, the level of independent
and synergistic effect correlated to the level of analgesia experienced by patients experiencing the placebo effect. While this review is not meant to serve as a thorough discussion of the underlying neurophysiology, it is important to consider the sensory pain pathways relative to placebo analgesia.

Initially, a specialized receptor cell is excited by the paired stimulus. For example, a pain stimulus acts on nociceptor cells that converts the pain signal into electrical neural activity. The afferent electrical activity travels via specialized nociceptive nerve fibers to the dorsal horn of the spinal cord. From the dorsal horn of the spinal cord, the pain sensation travels through the spinothalamic system to the thalamus. The thalamus acts both afferently by sending pain information to the anterior cingulate cortex (ACC), as well as efferently by projecting downwards to the dorsal horn to reduce ascending pain information (Ab Aziz & Ahmad, 2006). The ACC area of the brain has been shown to be an integral part of pain perception showing increased activity following a noxious stimuli (Coghill, McHaffie, & Yen, 2003; Wager et al., 2004) as well as increased activation upon expectation of pain (Rainville, Duncan, Price, Carrier, & Bushnell, 1997). The ACC and thalamus directly communicate with the periaqueductal gray, an area of the brain dense with endogenous opioid receptors which is believed to modulate pain in the spinal cord (Loyd & Murphy, 2009). Binding of opioid receptors allows for the relief of pain and, thus, is thought to largely be the underlying phenomena associated with the placebo effect.

Opponents of the placebo effect argue that analgesia experienced by individuals administered a placebo is due to bias in the methodological approach employed. This bias may cause subjective differences in pain levels without any measurable biochemical or physiological change in the subject (Hróbjartsson & Gøtzsche, 2001). This statement runs contrary to many
studies that found administration of a placebo can manifest changes in brain biochemistry (e.g., release of endogenous opioids) and brain activity (e.g., increased activity in the insula and rostral ACC) both during a pain inducing experience (Levine, Gordon, & Fields, 1978; Coghill et al., 2003) and in anticipation of pain (Rainville et al., 1997; Wager et al., 2011).

Levine, Gordon, and Fields (1978) were the first group of researchers to document release of endogenous opioids, a chemical in the brain that mediates analgesia, and its role in the manifestation of placebo analgesia. In their study, participants were injected with an opioid antagonist (naloxone), which muted the placebo analgesic effect. Naloxone binds to µ-opioid receptors and subsequently blocks endogenous opioids from binding to their target receptors. Thus, this cascade of events would disallow the analgesic effect to occur. Other research has confirmed that brain areas dense with µ-opioid receptors (e.g., rostral anterior cingulate cortex and the periaqueductal gray) demonstrate increased activity coinciding with placebo analgesia. However, this effect is eliminated when naloxone is presented, strengthening the evidence of endogenous opioid release in placebo analgesia (Eippert, Finsterbusch, Bingel, & Büchel, 2009; Scott et al., 2008; Bingel, Lorenz, Schoell, Weiller, & Büchel, 2006). Interestingly, Amanzio and Benedetti (1999) found that naloxone does not negate placebo analgesia when individuals were pre-conditioned with an anti-inflammatory drug. These findings indicate that placebo analgesia may, in fact, be mediated by both opioid and non-opioid systems and may be at least partially dependent on conditioning or expectancy. Benedetti, Amanzio, Rosato, and Blanchard (2011) later found that conditioning-induced placebo analgesia was countered by blocking endogenous cannabinoid receptors revealing that the endo-cannabinoid system may play a role in non-opioid mediated placebo analgesia.
Benedetti et al. (2003) set out to determine how multiple theories thought to be involved in the placebo worked synergistically to produce an analgesic response. The research design implemented a pain-inducing protocol where individuals were asked to estimate pain sensitivity. One experimental group was injected with a saline solution on the second day of testing, which researchers suggested was a powerful pain killer. However, on the first and third days of testing, participants completed the pain inducing protocol with no treatment. Significant increases in pain tolerance ($p < 0.03$) were found on the second day when compared to the first and third day, suggesting that increasing the expectancy of a treatment to temper pain sensitivity could, in fact, result in a decrease in experienced pain sensitivity. Along the same lines, an experimental group was exposed to both a conditional and expectancy component towards a treatment. During the first trial, participants performed a pain inducing protocol with no treatment. Subsequently, the participants performed the pain inducing exercise on days two and three while being treated with an anti-inflammatory steroid, with analgesic properties, in order to create a conditioning stimulus. On the fourth day, participants were administered a saline treatment prior to the exercise. In this instance, researchers manipulated the expectancy effect by informing group four that the injection would decrease their sensitivity to pain. The fifth day followed the same protocol as day one, in that no treatment followed the pain inducing exercise. Ultimately, Benedetti et al. (2003) found that the preconditioning with analgesic ketorolac resulted in significantly greater pain tolerance ($p < 0.05$) than verbal expectations alone. The findings showed that combination of verbal expectancy and preconditioning with an analgesic significantly increases the placebo response compared to verbal suggestion alone. While more studies are needed to further define the exact mechanisms and processes in play, it does seem
clear that the resulting neurophysiological responses to a placebo are influenced by a variety of psychic and physiologic factors that may work independently or synergistically.

The activity of specific brain areas involved in the manifestation of placebo analgesia has been advanced, stemming from novel studies utilizing functional magnetic resonance imaging (fMRI) techniques. For example, Wager et al. (2004) found that individuals in a placebo group exhibit lower activation of the insula, thalamus and anterior cingulate when given painful shocks and heat treatment. The thalamus is known to play an integral role in not only communicating and suppressing noxious signals from the spinal cord to the cerebral cortex at the thalamus (Derbyshire & Jones, 1998), but has also been shown to project down to the spinal cord to inhibit nociceptive signaling before reaching the brain (Millan, 2002). Less activation in the thalamus would modulate the degree of pain felt by an individual. While the analgesic effect of a placebo was originally isolated within the brain, findings by Wager et al. (2004) prompted new research to determine if a placebo administration may elicit changes in the sensation of pain (i.e., nociception) before an afferent signal reached the brain. Eippert et al., (2009) and Goffaux, Redmond, Rainville, and Marchand (2007) determined that administration placebo can, indeed, mediate nociceptive processing in the spinal cord. Although these findings are novel, Atlas and Wager (2012) correctly indicate the paucity of research relative to the placebo effect and nociceptive changes in the spinal cord specifically related to an understanding as to why administration of a placebo does not have a greater effect on more nociceptive regions of the brain (e.g., somatosensory cortices I and II). Future research on this area is certainly warranted and should provide even more insight into the complex regulation of the placebo effect.
The emergence of the placebo effect in sport and exercise

The placebo effect is recognized and controlled for in much of modern medicine research, most popularly by comparing pharmacological effects of a drug to that of a placebo. This is to ensure that any potential physiological or psychological changes observed are a result of the drug rather than effects caused by expectation alone. However, researchers posit that at least some of the noted effects are due to unaccounted differences in the placebo treatment (Kienle and Kiene, 1997). Within the sport and exercise field, Beedie (2007) explains that the disbelief in the placebo effect is increased when one examines the ratio of research validating the placebo relative to the amount of products available that fail to substantiate claims. Via advertising, many of these products attempt to elicit expectation of increased performance upon consumption of the product. That is, rather than the purported ingredients in the product being responsible, it may be that the expectation of ergogeneity is producing a placebo effect that leads to the increase in performance.

Beedie (2007) aimed to quantify the prominence of the placebo effect in sport, as well as personal experiences of change in performance brought about because of false belief in something (e.g., equipment, inert substances, and rituals). In that study, 19 of 30 respondents (97%) said that they believe a placebo could impact performance. Twenty-two of the respondents stated that they recall an occasion where a placebo or false belief positively influenced their performance, and seven of those participants said that expectation in something would likely influence their performance, in the second survey question. It should be noted that the second question was not strictly referring to a placebo, rather a false belief in anything (e.g., equipment, inert substances, and rituals). That notwithstanding, 73% of participants stated that belief alone in some inert treatment or piece of equipment could yield a positive impact on performance. This
qualitative study, along with many studies that have attempted to quantify the placebo effect, help show that the placebo effect has extended beyond traditional, therapeutic medicine and is widely believed to play a prominent role and impact performance in sport and exercise. The following sections of this manuscript are meant to examine and synthesize research that aimed to quantify the impact of the placebo effect in popular modes of exercise and sport: weightlifting, short duration, anaerobic sprint work, and longer duration, aerobic exercise.

Studies Investigating the Placebo Effect in Sport and Exercise Training

**Weightlifting.** Pain is a naturally inhibitory signal. The perception of pain, then, is thought to cause down-regulation in performance in order to preserve an individual from overexertion that may lead to injury. This allows for the preservation an individual’s health status but may also inhibit increases in strength by attenuating the degree of overload achieved during a particular training session. However, through inducing placebo analgesia it may be possible to down-regulate the perception of pain and the resultant inhibitory responses that follow. The chronic down-regulation of inhibition could possibly result in greater muscular and neuromuscular adaptations, thus maximizing training potential. Thus, it is from this platform that the many studies have been directed when studying the placebo effect during weightlifting.

A study published by Ariel and Saville in 1972 is generally regarded as the first study to examine the effect a placebo can have on performance. Fifteen experienced weightlifters were asked to participate in a study to quantify the effects an oral anabolic steroid had on force production. Researchers informed the participants of the benefits of the drug and the positive impact it would have on performance. They were then asked to complete baseline tests, without being administered a placebo, to determine maximal strength for four lifts: bench press, squat, military press, and seated press. All participants were asked to complete a four week program
with no intervention referred to as the “pre-placebo period”. Then, participants completed another maximal strength testing session to re-establish maximal weight lifted following the pre-placebo period. Six subjects were then administered a placebo for the following four week training session with a final maximal testing session to conclude the study.

Results from their study showed strength increases from baseline testing in the pre-placebo trials for bench press, military press, seated press and squat (3.4%, 0.8%, 2.7%, and 2.0%), as well as the placebo intervention (9.6%, 8.5%, 6.2%, and 13.8%, respectively). Significant increases in maximal weight lifted were observed in bench press ($p \leq 0.05$), military press ($p \leq 0.01$), and squat ($p \leq 0.01$) when comparing percent change occurring from baseline to post-testing between the pre-placebo and placebo trials. Participants produced significantly higher percent change in total weight lifted following the placebo intervention compared to the pre-placebo intervention ($p < 0.05$). Researchers did not interview subjects after the study to determine if the subjects experienced a placebo effect of any type (e.g., increased vigor, resistance to fatigue, increased perceived recovery), nor did they attempt to explain the possible mechanism that caused the significant increases in strength following the placebo intervention. Future research would improve on the Ariel and Saville (1972) study, as well as provide possible mechanisms that explain the increase in strength observed in participants who had received a placebo.

Maganaris, Collins, and Sharp (2000) were the first group to conduct research following that of Ariel and Saville (1972) to explore the influence a placebo could have on performance. Similar to Ariel and Saville (1972), researchers chose to deceive participants by informing them they were being administered an anabolic steroid. However, unlike Ariel and Saville (1972), the authors did not inform individuals of possible effects of steroid use, rather, they allowed the
‘street reputation’ of steroids to produce expectancy. Eleven national-level power lifters then completed baseline maximal testing in the squat, bench press, and deadlift. Seven days later they participated in the first experimental trial in which they were administered a placebo pill that they were informed was a fast acting anabolic steroid. The pill was delivered five minutes prior to completing another maximal strength test in the squat, bench press, and deadlift. Before leaving the laboratory individuals were provided two more pills to take during the next week. Seven days after the first session, participants completed an identical testing protocol. Prior to testing, each individual was queried as to how their training changed after being administered the steroid. All individuals reported increased vigor which allowed them to either lift heavier loads or increase repetitions. Six participants were then immediately informed that they had been deceived and were dosing with a placebo. They then completed the second testing trial having been exposed to the deception in the study. The remaining five remaining individuals were allowed to believe they had been taking an anabolic steroid. Results from this study revealed significant improvements in maximal weight lifted in trial one in the bench press, squat, and deadlift (3.5%, 5.2%, and 4.2%, respectively) when compared to baseline \( (p < 0.01) \). During the second testing, the group that believed they were administered an anabolic steroid maintained significant improvements in bench press, squat, and deadlift (3.2%, 4.4%, and 4.0%, respectively; \( p < 0.01 \)) while the group informed of the placebo deception experienced a reduction in maximal weight when compared to baseline (-1.7%, -0.4%, -0.4%, respectively; Beedie & Foad, 2007). The results supported that expectancy of a placebo would produce improvements in performance which could be countered when the expectancy of the placebo was removed.
Kalasountas et al. (2007) implemented a similar testing design to that of Maganaris et al. (2000) to determine if a placebo can positively impact performance versus a control group. An interesting secondary aim was to evaluate the similarity of the placebo group to the control group after being debriefed to the use of a placebo. Researchers assigned 42 participants into three different experimental groups: a control group (con), a group unaware that the ergogenic aid taken was a placebo (PP), and a group who completed the first experimental trial unaware they were administered a placebo but then informed of the inertness of the placebo in the second experimental trial (PN). Before the first trial, participants in the PP and PN groups were given two pills and informed it contained an amino acid that would likely cause an immediate increase in strength. Participants were then asked to perform a maximum bench press and leg press protocol. Forty-eight hours later participants returned to complete the same testing protocol, but prior to testing, the PN group was informed that a prominent coach found that the amino acid pills had no effect on performance and that they would not be given the supplement. The PP group followed the exact same testing procedures for trial two as they did for trial one. Statistical analyses found both the PP and PN groups experienced significant improvements compared to the control group in maximal bench press (13.42 ± 13.71 and 13.58 ± 10.26 vs. 1.86 ± 4.20 pounds, respectively) and leg press (76.76 ± 56.44 and 33.92 ± 33.92 vs. -2.64 ± 16.37 pounds, respectively) in the first trial ($p < 0.01$). They also reported that participants aware of the deception prior to the second trial experienced strength changes in bench press and leg press not significantly different to that of the control group ($p = 0.09$ and $p = 0.12$, respectively). These findings support the second hypothesis that awareness of deception can mediate maximal strength and regress to values similar to a control group.
In another study investigating the effect of placebos on resistance training, Duncan, Lyons, and Hankey (2009) determined if performance would improve when exercise was performed to failure after an oral placebo was administered. Researchers had 12 participants participate in three experimental trials. Within these trials, one of three solutions were delivered: a control trial where they were given water, one trial where they were given a placebo, and lastly a trial where they were given a solution that participants believed contained caffeine, although no caffeine was administered. Participants were provided literature detailing the beneficial results of caffeine supplementation in order to build expectancy for the solution to work. Participants reported to the laboratory one hour before testing to consume a 250 ml solution; however, they were not informed as to what solution they would be drinking. They then completed single-leg extensions at 60% of 1-RM at a cadence of 15 repetitions per minute until failure. Immediately after failure, participants estimated an overall body RPE (RPE-O) and an RPE for the active muscle used during the experiment (RPE-AM). Researchers then queried the participants relative to whether they felt they received the placebo or caffeine solution in order to categorize the trial as perceived placebo or perceived caffeine. The analyses revealed that RPE-O was significantly higher ($p = 0.04$) in the perceived placebo group (14.9 ± 1.6) when compared to both the control (13.8 ± 1.9) and perceived caffeine groups (14.2 ± 1.6). Participants in the perceived caffeine group completed significantly more repetitions (20.3 ± 4.6) than they did in either the control or perceived placebo trial (16.4 ± 4.1 and 18.3 ± 4.1 repetitions, respectively) despite no significant changes in peak heart rate, systolic blood pressure, or diastolic blood pressure. This decrease in performance, resulting from belief in placebo consumption, is also supported in studies using other modes by Beedie et al. (2006) and Clark, Hopkins, Hawley, and Burke (2000) (discussed in a later section).
**Short duration anaerobic running and cycling.** Although weight training and short distance sprinting are similar in nature in that they are characterized by predominant contribution of the anaerobic systems, sprinting requires greater reliance on the cardiorespiratory response to ensure success. Thus, there is an observed increase in afferent signals received by the brain as a result of increased system involvement during short term sprints. The perception of pain in multiple pathways allows for a greater disassociation between perceived and actual stress. This decrease in perceived stress may allow for an increase in performance (Hampson et al., 2001). Plainly stated, it may be that individuals alter their pacing or performance subconsciously relative to the degree of perceived stress interpreted by the brain from the body during this mode of exercise or sport training.

Beedie et al. (2007) designed an experiment to determine the effect that a negative placebo (i.e., the nocebo) may have on performance compared to a positive placebo. Forty-two team-sport athletes participated in the study where each participant ran three, 30 meter sprints separated by two minutes of recovery to establish a baseline for speed and time to complete each sprint (sprints 1-3). They were then given a 20 minute recovery period and within this period received a pill purported to enhance sprint performance (i.e., placebo) or a pill that may decrease sprint performance (i.e., nocebo). Participants then completed the final half of the sprint protocol (i.e., sprints 4-6). Results revealed that the positive belief group (i.e., placebo group) completed the sixth sprint significantly faster (0.133 seconds, \( p < 0.05 \)) than the third sprint. In addition, the nocebo group experienced significantly slower times (i.e., ran slower) after running 1.7% (0.08 seconds) slower in the experimental condition when compared to the control group (\( p = 0.01 \)). No difference in mean speed was observed from baseline to experimental trial (\( p = 0.96 \)) for the positive belief group while the negative belief group ran significantly slower in the experimental
trial than they did in the baseline trial ($p = 0.01$). It seems that the negative belief in a substance will impact performance in the same manner as a positive belief in a substance. The researchers proposed that if negative belief in a substance can have a negative impact on performance, then even a negative belief about an authentic treatment may also negate some positive effects associated with the treatment.

The only other study to have investigated a placebo effect during short duration, anaerobic activity was performed by Wright et al. (2009). Eighteen healthy participants (8 men, 10 women) were asked to participate in a study to validate the benefits of a fast acting creatine monohydrate during a high intensity cycling test. However, the actual purpose was to quantify the effect that administration of a placebo has during a high intensity bout of exercise. The methods employed in this particular study required participants to attend a graduate level academic course that discussed ergogenic aids in order to build expectancy for the placebo to be effective. Participants were asked to report to the laboratory 24 hours prior to testing to prepare and consume a placebo solution which consisted of 300 ml of water and 5 g of maltodextrin or 300 ml of water. This process was repeated 30 minutes before testing. This is the only study that has required participants to prepare the ingredients of the placebo, which was intended to ensure participants would be consuming the ergogenic aid. After completion of a standardized warm-up, participants performed for 30 seconds of exercise using a resistance of 75 g kg$^{-1}$ of body weight on a cycle ergometer. Results for the experimental and control trials were not significantly different in terms of relative peak power (14.44 ± 1.61 vs. 14.35 ± 1.05 W kg$^{-1}$) or relative mean power (7.31 ± 0.94 vs. 7.43 ± 1.02 W kg$^{-1}$). Researchers also found no significant differences in pattern of fatigue between the two trials. Although there were no significant changes in power between the groups, 62% of participants experienced at least a 1.5% increase in mean power.
when administered the placebo while only 36% of participants experienced a power increase of the same magnitude during the control trial.

**Longer duration, aerobic running and cycling.** It has been shown that administration of a placebo has been associated with increases in performance during anaerobic activities (e.g., weightlifting and short distance sprints); however these results may be attributed to the acute nature of the mode of exercise. Thus, these conclusions may not be generalized to all exercise activities, especially those longer in duration. That is, during prolonged, moderate intensity exercise the resultant intensities may not be high enough to elicit the neurophysiological consequence associated with a placebo effect, thus minimizing the potential performance improvements observed in other, more intense modes of exercise or sport training.

A study by Clark et al. (2000) analyzed the effect a placebo and a carbohydrate drink on 40-km cycling time trial performance. The researchers had 54 moderately- to well-trained cyclists complete the study. Participants were informed that they would be placed in one of three test groups: a carbohydrate group, a group that would be given a placebo which contained water with an artificial sweetener added, or a group that had a 50/50 chance of being given either the carbohydrate drink or placebo. Researchers further stratified groups by providing half of the participants in each group the carbohydrate beverage while the other half received a placebo, which gave the researchers six different groups. Researchers informed participants that the carbohydrate should help improve performance when compared to the placebo in order to build expectancy. The participants returned to the laboratory after preliminary testing to complete baseline testing in which all participants were given water. The same liquid consumption protocol was applied to both control and experimental testing sessions. Participants arrived to the lab 30 minutes before testing to consume the designated beverage which equated to eight ml.kg⁻¹
of body mass throughout the trial. They consumed the designated beverage equal to two ml.kg\(^{-1}\) of body mass at two minutes before starting the time trial, as well as 10, 20 and 30-km into the time trial participants. Researchers instructed the participants to complete the 40-km time trial as fast as possible with the only feedback given to them being the percentage of the trial completed. Results from this study showed that the informed carbohydrate produced a 4.3 ± 4.8% improvement in performance; informed placebo, 0.5 ± 8.5% improvement in performance; and 50/50 chance of receiving a placebo or carbohydrate beverage, -1.1 ± 8.5% improvement in performance. The authors went on to quantify the “full placebo effect” by subtracting the average power output of the informed placebo group by the average power of the informed carbohydrate group which yields a value of 3.8\(\%\) \((p = 0.06)\). A 3.8\% increase in power would equate to a 1.6\% decrease in time over a 40-km cycling time trial, which would certainly be practically significant for competitive cyclists, albeit not statistically significant. Analyses also showed substantial variation in power output in the not informed group (~1.6 fold greater than the variation for informed placebo and carbohydrate groups). This variation in power, from baseline to experimental testing, for the not informed group provides indirect evidence demonstrating participant belief in an ergogenic aid may impact performance. The authors also stated that the variation in power could have been due to resignation of effort by the participant when they felt that they hadn’t received an ergogenic aid.

Stemming from findings from Clark et al. (2000), Foster et al. (2004) aimed to determine if a similar effect could be observed on a mode of exercise never examined in placebo research, running. Sixteen well-trained participants were recruited and deceived into believing that the study was attempting to quantify a new ergogenic aid’s influence on a 5-km time trial. Researchers showed the participants a video detailing the purported benefits of super oxygenated
water on endurance performance in order to ensure expectancy. Results from the study revealed no significant differences ($p > 0.05$) between placebo or super oxygenated water conditions in RPE (8.2 ± 1.0 vs. 8.4 ± 1.2), peak heart rate (177 ± 5 vs. 177 ± 6 beats/min), blood lactate (12.2 ± 3.2 vs. 11.4 ± 2.2 mmol), or time to completion (21:54 vs 21:40 min:sec). While time to complete the time trial was not statistically significant ($p = 0.11$), it resulted in a 14-second decrease in time to complete a 5-km run for the placebo group. Similar to results from Clark et al., (2000), findings would be considered a competitively or a practically significant change.

A follow-up study to Foster et al., (2004) was published by Porcari and Foster (2006) to determine the effect of a placebo on 5-km running performance. Researchers deceived 32 experienced runners into believing that super oxygenated water had a beneficial impact on performance. The experimental design employed was a replication of Foster et al. (2004), but with the addition of a habituation 5-km time trial, in addition to the two time trials. Similar to Foster et al. (2004), no significant differences ($p > 0.05$) were observed relative to heart rate, RPE (7.7 ± 1.4 vs. 7.7 ± 1.2) or blood lactate (9.8 ± 3.9 vs. 10.2 ± 3.7 mmol) between control and placebo groups, respectively. That notwithstanding, participants again exhibited significantly faster 5-km run times when they believed they had consumed super oxygenated water than when they consumed control water (19:41 ± 2:32 vs 21:04 ± 3.34 min:sec; $p < 0.05$). Researchers note an interesting relationship between training status and placebo effect as participants who completed the control trial in more than 20 minutes improved their time during the experimental trial by an average of 2 minutes and 22 seconds while runners who completed the control trial in under 20 minutes only improved their experimental trial time by 28 seconds. Whether this difference in improvement is due to the attenuation of a placebo effect in trained (vs. less-
trained) individuals or simply a matter of faster times having less room for improvement is not clear and is worthy of future research.

Beedie et al. (2006) were the first to examine the prospect of a dose-response to a placebo and the potential impact it may have on performance during a 10-km cycling time trial. They conducted a study in which participants were informed that they would take part in baseline testing along with three additional experimental sessions where they would be administered a placebo, 4.5 mg kg\(^{-1}\) of caffeine, and 9.0 mg kg\(^{-1}\) of caffeine. However, a placebo was administered in all testing sessions. Participants were provided research explaining performance benefits associated with consumption of caffeine as well as anecdotal evidence of the benefits of caffeine from elite cyclists in an attempt to build expectancy. While researchers did provide participants with reported benefits of caffeine, they did not inform participants that they were ‘blinded’ to the treatment. Rather, researchers asked participants to identify which experimental trial they completed: placebo, 4.0 mg kg\(^{-1}\) of caffeine, or 9.0 mg kg\(^{-1}\) of caffeine based solely on perceived performance. Findings revealed that participants experienced a 2.2 ± 3.0% increase in power output when they believed they received caffeine, compared to a 1.4 ± 3.1% decrease in power output when they believed they had received a placebo. It appears the perceived caffeine trials reveal a potential dose-response effect when participants believed they received 9.0 mg kg\(^{-1}\) of caffeine. In that trial, participants exhibited an increase in power of 3.1 ± 3.4% and an increase of only 1.3 ± 2.7% when participants believed they were given 4.5 mg kg\(^{-1}\) of caffeine. Moreover, there were no significant changes in heart rate, oxygen uptake, or blood lactate throughout the baseline and experimental changes which may suggest no considerable change in effort throughout the trials. Follow-up interviews showed that five of the seven participants said that they experienced a placebo effect in at least one of the experimental trials; they explained
the placebo effect experienced manifested as pain reduction and fatigue resistance. The authors also state that the two participants that did not experience a placebo effect produced the greatest power outputs while the strongest placebo effect was observed in a participant who had the lowest overall power output, suggesting that training status has a negative relationship with the placebo effect. The possible relationship of training status and placebo response has also been shown by Clark et al. (2000) and Porcari et al. (2006).

McClung and Collins (2007) assessed the effect sodium bicarbonate has on a 1000-meter time trial performance. Sixteen participants (12 men, 4 women) performed five time trials under different informed and actual treatment conditions: a habituation trial, informed drug/ received drug, informed drug/received placebo, informed placebo/received drug, and informed placebo/received placebo. An informational pamphlet was provided to the participants prior to any testing session detailing the benefits of sodium bicarbonate which was provided to “reduce the potential for knowledge, or lack thereof, being an extraneous variable”. Participants were briefed before each trial about what they should experience in terms of pain and discomfort during each trial. They were given 30 minutes to consume 0.3 g kg\(^{-1}\) of the prescribed solution two hours before testing. Time to completion, blood lactate concentration, RPE, and heart rate were measured throughout the testing sessions. Results revealed a significant main effect for belief and time \(p < 0.01\) on performance. This suggests that regardless of the actual treatment condition, times were higher (i.e., diminished performance) when comparing the informed placebo and informed sodium bicarbonate groups. Authors also found that participants who believed that they consumed the sodium bicarbonate reported significantly lower RPE values \(p < 0.05\) than those told that they had not received sodium bicarbonate (5.0 ± 1.8 and 6.1 ± 2.5; respectively). Authors concluded that participant belief of receiving the sodium bicarbonate was
able to have an almost identical impact on performance when compared to receiving sodium bicarbonate.

Placebo research conducted prior to 2009 had employed testing designs involving bouts of short(er) aerobic exercise (e.g., 40-km cycling, 5-km running, and 10-km cycling). To that end, Hulston and Jeukendrup (2009) sought to analyze the placebo effect of a carbohydrate beverage during prolonged aerobic activity. Researchers asked 10 endurance cyclists to take part in a study to determine the effect two carbohydrate beverages had on performance compared to water. Each participant was asked to participate in three experimental trials in which they received water [WAT], placebo [PLA], and a carbohydrate-electrolyte solution [CES] that were separated by at least seven days. Each experimental trial involved 120 minutes of steady-state cycling at 50% of maximal power output. Immediately following the steady-state ride, participants completed a time trial at 70% of maximal power to complete a standardized workload which was accomplished in approximately 60 minutes. Researchers administered 600 ml of the experimental beverage at the onset of steady state exercise followed by an additional 150 ml every 15 minutes. Statistical analyses revealed mean power output during the time trial for the CES (242 ± 27 Watts) to significantly higher ($p < 0.05$) than both PLA and WAT trials (219 ± 17 and 218 ± 22 Watts; respectively). Changes in mean power were significantly different during the final 25% of the time trial for CES and WAT ($p < 0.05$), whereas significant differences in mean power were found during the final 50% and 25% of the time trial when comparing PLA and CES. Mean power output was also maintained throughout the CES trial whereas it decreased in both the WAT and PLA trials. While VO$_2$, volume of oxygen consumption, did not differ significantly between any of the experimental trials, participants in the CES trial were able to oxidize carbohydrates significantly more and fats significantly less
than in the WAT and PLA trials ($p < 0.05$). Results from this finding suggest that a placebo effect over that of an actual ergogenic aid may be both time and intensity specific.

Overall, findings from these studies involving a variety of modes of exercise and sport training suggest that there is most likely a positive change in performance when individuals are administered a placebo. While overall performance generally improved as long as individuals were under the expectancy of improved performance from the placebo, not all studies reveal changes from physiological measures that would reflect significant changes in performance (i.e., blood lactate, heart rate, VO$_2$, etc.). In addition, evidence across all modes of training also seems to suggest that the direction of the expectancy is critical in yielding changes in performance. That is, when an individual is led to believe a positive change in performance should be expected, it tends to manifest, as does a negative change in performance, when that is what an individual is told to expect. Importantly, it does seem to be that the magnitude of the change in performance will be congruent to the degree that the individual is led to believe a change will happen.

Consequently, there may exist a dose-response to the placebo effect much in the same way there is a dose-response to ‘active’ ergogenic aids. Finally, the reviewed studies would also show that there is no universal manner in which individuals develop an expectancy which may range from simple word-of-mouth suggestion, to embellished research findings, to anecdotal evidence from peers.

**Recovery and Susceptibility to Placebo Effect**

Independently, recovery from exercise and sport training along with the placebo effect have gained increased attention in the past 10 years (Bishop et al., 2008; Beedie & Foad, 2009). Interestingly, despite the increase in attention to both fields, these research areas have not converged. This is somewhat surprising given the renewed attention to both fields and the impact
that both have on performance. Indeed, it seems media (i.e., magazines, television commercials, etc.) promoting ergogenic aids are aimed towards the ‘mainstream’ consumer touting improvements in recovery in order to maximize training results. In that sense, it seems there has been a shift of focus, per se, from eliciting improvements during a session to expediting recovery between sessions. This has been mirrored by recent research examining factors associated with time course of recovery (Bishop et al., 2008; McLester et al., 2003; Jones et al., 2006; Laurent et al., 2010), especially during anaerobic work (i.e., resistance training, sprint training). Stemming from these works, recent studies have attempted to develop novel methods that attempt to quantify recovery prior to initiation of subsequent training sessions. Interestingly, these measures strongly rely on measures that are similar to ratings of perceived exertion, in that they attempt to provide a psychophysiological measure of readiness or recovery (Edwards, Bentley, Mann, & Seaholme, 201; Kentta & Hassmen, 1998; Lambert & Borresen, 2006; Laurent et al., 2011).

To that end, Laurent et al. (2011) designed a study to determine if participants are able to correctly estimate sprinting performance by using a perceived recovery status scale (PRS). Researchers found that participants were able to correctly predict their change in performance by using a perceived recovery scale over 80% of the time, both when performance improved and decreased. Sikorski et al. (2013) further validated the use of the PRS as a scale to assess recovery after finding a moderate correlation between the PRS and serum creatine kinase levels which strengthens the view that perceptual measures rely heavily on physiological cues, in this case, muscle damage. Although researchers have used perceived recovery as an explanatory statement relative to an increase in performance, perceived recovery using a validated psychobiological tool has not been measured following administration of a placebo. Beedie and Foad (2009) explain that strength increases seen by Ariel and Saville (1972) when participants were given a
placebo, described to them as an anabolic steroid, could be because of an increased perceived recovery. The authors suggested that when participants felt recovered, they performed at higher intensities in subsequent training sessions despite congruent recovery times between control and experimental conditions. Interestingly, Sikorski et al. (2013) also found a moderate correlation between PRS and leg pain, indicating that nociception may play a role in mediating not only perceived exertion but also perceived recovery. The correlation between pain and PRS, along with the analgesic effect upon administration of a placebo, may warrant research to determine if decreased pain can influence perceived recovery. Further research examining the role of a placebo and placebo effect on recovery, both from a performance standpoint as well as perceived recovery, is worthy of merit and warranted at this time.
Experimental Approach to the Problem

The experimental designs of the available literature regarding the placebo effect, while novel, fail to simulate what typical ergogenic aid consumers would experience in a real world environment. That is, while studies that exist maintain a high-degree of internal validity by implementing a single or double-blind approach, there is perhaps an attenuation of the true expectancy effect that individuals experience in an ecologically valid setting. Popular research designs such as the Latin square design can help explain the factors that mediate the placebo response, but employing this design minimizes individual expectancy, thus reducing the effectiveness of the placebo. When an individual consumes or purchases a supplement, they generally do so expecting it to positively affect performance, thus increasing the expectancy effect. In a no-blind experimental design, participants would expect to see improved performance due to the administration of the ergogenic aid, which would provide researchers an opportunity to gauge a more realistic understanding of the placebo effect in a sport or exercise session. Therefore, in this study, a no-blind approach is taken to improve the expectancy effect as well as maximize ecological validity.

Participants

Ten healthy, asymptomatic men volunteered to participate in the study. An \textit{a priori} power analysis indicated that a minimum of 10 subjects were needed to yield a power of 0.80 for detecting a moderate effect size with significance set at $\alpha = 0.05$. To be included in this study, participants must have reported performing sprint training or competition in an intermittent-type sport (e.g., basketball, football, tennis, soccer) at least two days per week. Criteria for exclusion included any orthopedic or musculoskeletal injury limiting performance or any individual
considered moderate risk or higher according to ACSM guidelines (Thompson, Gordon, & Pescatello, 2010). Prior to testing sessions, participants were instructed to refrain from drinking alcohol 24 hours and caffeine four hours before beginning physical activity. Participants were also instructed to abstain from intense physical activities, other than that asked of them in the study 48 hours before testing. Prior to each testing session, participants were queried regarding adherence to the guidelines set for dietary intake and physical activity. No participants were excluded from testing for the day for having failed to adhere to these guidelines. This study was approved by the local Human Subject Review Board and written, informed consent was obtained prior to testing the participants.

**Maximizing Expectancy**

The participants were provided a brochure after arrival to the lab noting the purpose of the study is to determine the impact an FDA approved substance has on performance during repeated sprint work. The brochure contained a section that provides an overview of previous research regarding the substance that they will be given. However, the results provided to the participants detailed results from studies investigating the placebo effect and exercise performance (Pollo, Carlino, & Benedetti, 2008; Beedie et al., 2007; Wright et al., 2009). The brochure only included an overview of the positive findings from these studies; no author names were included in the brochure to ensure that participants would not be able to search for the studies, relying only on the credibility of the researchers as authority figures, thus ensuring expectancy.

After the participants read the informational brochure, they completed a medical history questionnaire. An inquiry was added to the medical history questionnaire in order to screen for people with a low expectancy of effectiveness. The statement read, “Do you believe nutritional
supplements can have a positive effect on performance?” Participants were asked to rate this on a 1-10 scale, with 1 suggesting they have no belief that ergogenic aids have any positive affect, and 10 confirming the individual fully believes that ergogenic aids can have a positive effect on performance. Results from the expectancy question are outlined in Figure 1. The participants must not have a negative bias as to the effectiveness of ergogenic aids, as this may have decrease the expectancy of the ergogenic aid to work and result in no placebo response. Any participant with a score of five or lower is thought to have a preconceived bias of ergogenic aids and was excluded from the study.

Figure 1. Frequency analysis based upon answers from the expectancy question

Note. A score of six or higher was needed from the participants in order to be considered for the study.

**Measures**

**Rating of perceived exertion (RPE).** Participants reported RPE using the Adult OMNI Scale of Perceived Exertion for running (Utter et al., 2004). The Adult OMNI Scale of Perceived Exertion for running is a 0-10 visually anchored scale with a score of zero signifying extreme ease in completing the activity and a score of 10 signifying that the activity was extremely hard.
Session rating of perceived exertion (S-RPE). Session rating of perceived exertion was reported using the Session RPE (SRPE) scale (Foster et al., 2001). Analogous to the Omni RPE scale, the Session RPE scale is a 0-10 visually anchored scale with zero classified as the exertion felt while at rest and a 10 meaning that the participant was exerting maximal effort. The scale was explained to the participants as the global difficulty felt during the entire session.

Pain. In order to rate pain, participants were presented with a visual analog pain scale (VAS PAIN) (Scott & Huskisson, 1976). The VAS PAIN is a 100 millimeter horizontal line with descriptions of ‘no pain at all’ and ‘almost unbearable pain’ on either end of the scale. Participants were asked to make a horizontal mark on a visual analog scale to assess pain. A ruler was then used to assess where the vertical line was placed on the VAS PAIN scale in order to record the measure of reported pain. For example, a vertical mark placed 78 millimeters away from “no pain at all” would result in a rating of 7.8.

Perceived recovery status (PRS). Perceived recovery status was recorded using a modified Perceived Recovery Status Scale developed by Laurent et al. (2011) (Figure 1). The PRS scale is a 0-10 scale used to determine an individual’s perceived recovery status with a score of zero representing very poor recovery and a score of 10 representing very well recovered.

Decrement score. A decrement score was calculated to analyze the rate of fatigue for each RAST. The decrement score was calculated by dividing the difference of the average power (MPWR) of the cycle and the peak power (PPWR) of the cycle by the PPWR of the cycle and multiplying by 100. Thusly, a higher decrement score means a higher rate of fatigue while a lower decrement score means a lower rate of fatigue (Oliver, 2009).

Recovery score. A recovery score was calculated to determine the amount of power recovered between each RAST. Recovery scores are determined by dividing the difference of
average power of RAST and the subsequent RAST by the average sprint time of the RAST. This value is then subtracted from one and multiplied by 100. Consequently, a higher recovery score means a higher amount of relative power recovered while a lower recover score means that a lower amount of power was recovered (Oliver, 2009)

**Blood lactate concentration.** Blood lactate concentrations were assessed via samples by means of a finger stick and capillary puncture and analyzed by an enzymatic, portable blood lactate analyzer (Lactate Plus, Nova Biomedical Corp., Waltham, Washington USA). The lactate analyzer was calibrated in compliance with the manufacturer’s instructions and has been validated in other studies (Saunders, Feldman, Correia, & Weinstein, 2005).

**Figure 2. The perceived recovery status scale**

<table>
<thead>
<tr>
<th>Perceived Recovery Status Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 Very well recovered / Highly energetic</td>
</tr>
<tr>
<td>9 Well recovered / Somewhat energetic</td>
</tr>
<tr>
<td>8 Adequately recovered</td>
</tr>
<tr>
<td>7 Somewhat recovered</td>
</tr>
<tr>
<td>6 Not well recovered / Somewhat tired</td>
</tr>
<tr>
<td>5 Very poorly recovered / Extremely tired</td>
</tr>
</tbody>
</table>


**Experimental Procedures**

**Familiarization.** Participants reported to the laboratory for a familiarization session. Upon arrival, participants were assessed for height (cm) and body mass (kg), using a stadiometer and beam scale (Detecto Scale Company, Webb City, Missouri, USA). Body fat percentage
estimations were also performed using the 3-site method (men: chest, abdomen, and thigh; women: tricep, iliac, and thigh; Pollock, Schmidt, & Jackson, 1980) by skinfold calipers (Lange, Cambridge, Maryland, USA). The descriptive data for all participants are listed in Table 1.

Participants then performed one running-based anaerobic sprint test (RAST; see Draper & Whyte, 1997), on a Curve non-motorized treadmill (Woodway USA, Inc., Waukesha, Wisconsin, USA). In brief, the RAST consists of six, 35-meter sprints, performed maximally, with 10 seconds of rest between each sprint. The participant were encouraged to ask any questions or express any concerns they may have about the procedures during this session.

Table 1.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>22.2 ± 2.4</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.8 ± .01</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>81.2 ± 7.4</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>8.1 ± 2.4</td>
</tr>
</tbody>
</table>

**Placebo condition.** Following at least 24- hours after familiarization, participants reported to the laboratory 30 minutes prior to testing. Upon arrival, they were given 10 minutes to ingest 600 mL of the placebo (McClung and Collins, 2007). A commercially available, non-caloric, ‘water enhancer’ was used to flavor the water. The two sweeteners used, sucralose and acesulfame potassium, have been shown to be non-nutritive and have no significant effect on blood insulin levels (Mezitis et al., 1996). The first dose of 600 mL was prepared in front of the participants. Researchers extracted 1 mL of the ‘water enhancer’ from a beaker and extracted it...
into an Erlenmeyer flask containing approximately 600 mL of purified water chilled to 10°C. The other dosages of 150 mL were prepared beforehand, with the same concentration as the first dosage.

Immediately after ingestion of the placebo, participants performed a standardized warm-up in agreement with procedures developed by Vetter (2007). This protocol incorporated a four minute walk at 6.04 km/h, a two minute jog at 12.75 km/h on a treadmill, and three rounds of a dynamic warm-up (Table 2). Following the warm-up, each participant performed three RAST protocols (Figure 2). Throughout each RAST, participants were given a 10-second recovery period in which they were asked to straddle the treadmill belt. The investigators gave the participant a five second countdown in which they were prompted to start walking on the belt. At the conclusion of the five second countdown, individuals were given a verbal cue to initiate their sprint. Verbal encouragement was provided to the participants in a similar manner throughout the series of RASTs. Immediately following the completion of the 35 meter sprint participants were given a verbal cue to straddle the treadmill belt again for their 10-second recovery period. Once six sprints had been completed (i.e., one complete RAST) the participants were given a seven minute passive recovery period. The recovery period of seven minutes was chosen to allow optimal phosphocreatine repletion (Harris et al., 1976). During the recovery period, each participant was asked to ingest 150 mL of the placebo beverage, as well as permitted to drink water ad libitum, in accordance with McClung and Collins (2007).

Throughout the entire session of repeated sprint work, heart rate (Polar Inc., Port Washington, New York, USA) was monitored prior to and following each sprint via a telemetered system secured around the participants torso and communicated to a wrist watch. Following each sprint, ratings of perceived exertion (RPE) was provided within a 5-second
period between each sprint. Raw treadmill belt speed data (peak power [watts], mean power [watts], peak speed [km/h], and mean speed [km/h]) from the non-motorized treadmill was recorded via a transducer in the non-motorized treadmill platform and monitored ‘real time’ on a personal computer containing the manufacturer’s computer software (World Wide Software Solutions Firmware version 1.32). Immediately, following (within one minute) each RAST bout, blood lactate concentration was assessed.

Ten seconds into the recovery period between RASTs, participants were presented with a visual analog pain scale (VAS PAIN) and asked to rate their level of pain (Scott & Huskisson, 1976). With 10 seconds remaining in the recovery period, participants were asked to rate their perceived recovery status (PRS). Approximately 15-20 minutes following the total exercise session, participants provided a global rating of perceived effort scale. After the final recovery bout and assessment of SRPE, participants were provided a subsequent 150 mL of the placebo beverage that was promoted to augment overnight recovery. Participants were asked to consume the beverage before leaving the lab to ensure that the beverage is consumed. Finally, participants were reminded to abstain from any physical activity until the second session of the given condition (i.e., day two of the placebo or control condition) was completed the following day. The participants then came in 24-hrs later to complete the same RAST protocol in order to observe the placebo effect in recovery not only within a session, but also between consecutive days of sprint work.

**Control condition.** The control condition followed the same protocol as the placebo condition. However, in this session, individuals in the control condition were not given a placebo, but instead were given 600 mL of water, as well as 150 mL of water during the recovery period between RASTs. Conditions were counterbalanced to ensure that a learning effect would
not impact the measurements that were being recorded. Figure 3 details the entire testing protocol that the participants completed. All testing took place at approximately the same time of the day.

Table 2.

<table>
<thead>
<tr>
<th>Standardized warm-up protocol</th>
<th>Repetitions</th>
<th>Cadence (repetitions per minute)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dynamic warm-up exercise</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toe raises</td>
<td>10</td>
<td>30</td>
</tr>
<tr>
<td>High knee marches</td>
<td>20</td>
<td>30</td>
</tr>
<tr>
<td>Butt kicks</td>
<td>20</td>
<td>30</td>
</tr>
</tbody>
</table>


Figure 3. Schematic illustration of repeated sprint protocol for a single condition.

Note. The illustration includes the sprinting protocol for both day one and day two for a given condition with specific measures detailed at each time point.
Debriefing

After all participants had completed both the control and experimental conditions, a debriefing letter was sent to all via traditional mail. This letter followed the guidelines of the American Psychological Association and institutional review board. A questionnaire was sent to the participants along with the debriefing letter. The questionnaire asked participants if they felt like they were being deceived as to the true nature of the experiment at any time during the experiment. It was revealed that 80% of the respondents did not feel that they were being deceived at any time during the experiment.
Statistical Analysis

A 2 (condition) x 3 (RAST) repeated measures ANOVA was performed to determine main effects for mean power, peak power, decrement score, recovery score, pain, and RPE. Paired t-tests with a Bonferroni correction applied to the alpha level were used to determine any significant differences between condition and/or RAST if a main effect was observed. Additionally, session RPE was analyzed using a dependent t-Test. Effect sizes ($\eta^2$) and statistical power ($N – B$) were also calculated for main effects. Cohen’s $d$ effect sizes for post hoc measures were calculated and classified in accordance with Cohen (1992) (i.e., small effect size $d= 0.20$, medium effect size $d= 0.50$, large effect size $d= 0.80$). All data is presented as a mean ± standard deviation. Statistical significance was determined a priori at $\alpha \leq 0.05$. All data was analyzed using SPSS version 20.0 (SPSS Inc., Chicago, IL, USA).
Peak Power

**Day one.** The peak power achieved in all RASTs for both the control and experimental conditions during day one and day two sessions are modeled in Figure 3. A repeated measures ANOVA revealed no main effect of condition on peak power during day one testing ($F_{1, 9} = 1.80$, $p = 0.21$, $\eta^2_p = 0.17$; N-β = 0.23). There was a significant main effect of RAST on peak power ($F_{2, 18} = 38.80$, $p < 0.01$, $\eta^2_p = 0.81$; N-β = 1.00). Post-hoc measures show peak power significantly decreased from RAST 1 to RAST2 ($p < 0.01$; $d = 0.45$), as well as from RAST2 to RAST3 ($p = 0.001$; $d = 0.50$). Participants also produced a significantly lower peak power in RAST3 than they did in RAST1 ($p < 0.01$; $d = 0.95$). All differences ranged from moderate to large with respect to effect size ($d = 0.45 – 0.95$). There was no significant interaction effect of condition x RAST ($p = 0.55$).

**Day two.** The repeated measures ANOVA revealed a significant main effect of condition on peak power ($F_{1, 9} = 9.50$, $p = 0.01$, $\eta^2_p = 0.51$; N-β = 0.78), as well as RAST on peak power ($F_{2, 18} = 36.80$, $p < 0.01$, $\eta^2_p = 0.80$; N-β = 1.00) on day two. Bonferroni follow-ups revealed peak power was not significantly different in RAST1 ($p = 0.13$; $d = 0.16$) or RAST2 ($p = 0.23$; $d = 0.13$), with congruently small effect sizes. However, participants in the placebo condition produced a significantly higher peak power in RAST3 ($p < 0.01$; $d = 0.41$). Peak power also significantly decreased when comparing each consecutive RAST completed (e.g., RAST1 to RAST2). Peak power for RAST1 was significantly higher when compared to RAST2 ($p = 0.002$; $d = 0.28$), as well as RAST3 ($p < 0.01$; $d = 0.63$). Participants produced significantly higher peak power in RAST3 than they did in RAST2 ($p = 0.003$; $d = 0.50$), as shown in Figure 3. All differences ranged from small to moderate, in terms of effect size, throughout the RAST. There
was also a significant interaction between condition and RAST ($F_{2,18} = 7.00, p = 0.006, \eta_p^2 = 0.44; N-\beta = 0.88$).

**Figure 5.** Peak power achieved during each RAST under control and placebo conditions during day one and day two sessions ($N = 10$).

* $\#$ $\|$ denotes a significant difference in peak power between RASTS during day one ($p < 0.01$)
† $\$, ¥ $\$ denotes a significant difference in peak power between RASTS during day two ($p < 0.01$)
§ denotes a significant difference in peak power between control and experimental conditions ($p < 0.01$)

**Mean Power**

**Day one.** The mean power achieved in all RASTs for both the control and experimental conditions during day one and day two sessions are displayed in Figure 4. A repeated measures ANOVA revealed no main effect of condition on mean power during day one testing ($F_{1,9} = \ldots$
0.47, \( p = 0.51, \eta_p^2 = 0.05; N-\beta = 0.09 \). There was, however, a significant main effect of RAST on mean power (\( F_{2, 18} = 7.35, \ p = 0.005, \eta_p^2 = 0.45; N-\beta = 0.89 \)). Post-hoc measures show mean power was significantly lower when comparing RAST_1 to RAST_2 (\( p = 0.005; d = 0.54 \)), as well as RAST_1 to RAST_3 (\( p = 0.002; d = 0.74 \)). No significant differences in mean power were observed from RAST_2 to RAST_3 (\( p = 0.46; d = 0.14 \)). Significant differences (\( p < 0.05 \)) were found to have correspondingly large effect sizes when compared to the moderate effect sizes calculated for differences that did not reach significance (\( p > 0.05 \)). There was no significant interaction effect of condition x RAST on mean power (\( p = 0.75 \)).

**Day two.** The repeated measures ANOVA revealed a significant main effect of condition on mean power (\( F_{1, 9} = 5.73, \ p = 0.04, \eta_p^2 = 0.39; N-\beta = 0.57 \), as well as RAST and mean power (\( F_{1.3, 11.8} = 4.56, \ p = 0.047, \eta_p^2 = 0.34; N-\beta = 0.56 \)) on day two. Bonferroni follow-ups revealed that mean power between placebo and control were not significantly different in RAST_1 (\( p = 0.22; d = 0.19 \)) or RAST_2 (\( p = 0.26; d = 0.11 \)), with congruently small effect sizes. However participants in the placebo condition produced significantly higher mean power in the third RAST (\( p = 0.002; d = 0.36 \)). Regarding changes in mean power for within a session, RAST_1 was significantly higher when compared to RAST_2 (\( p = 0.011; d = 0.30 \)), as well as RAST_3 (\( p = 0.018; d = 0.47 \)) (see Figure 4). No significant differences in mean power were observed between RAST_2 and RAST_3 (\( p = 0.378; d = 0.32 \)). All differences ranged from small to moderate with respect to effect size. There was also a significant interaction between condition x RAST on mean power (\( F_{2, 18} = 7.00, \ p = 0.006, \eta_p^2 = 0.44; N-\beta =0.88 \)).
Figure 6. Mean power achieved during each RAST under control and placebo conditions during day one and day two sessions (N = 10).

*, # denotes a significant difference in mean power between RASTS during day one (p < 0.05)
†, ‡ denotes a significant difference in mean power between RASTS during day two (p < 0.05)
§ denotes a significant difference in mean power between control and experimental conditions (p < 0.01)

Decrement Score

Day one. Figure 5 shows the decrement of power in all RASTs for both the control and experimental conditions during day one and day two sessions. A repeated measures ANOVA revealed no main effect of condition ($F_{1, 9} = 0.02, p = 0.89, \eta^2 = 0.002; N-\beta = 0.05$), or RAST ($F_{2, 18} = 3.1, p = 0.07, \eta^2 = 0.25; N-\beta = 0.52$) on decrement score during the day one session. Additionally, no significant interaction effect of condition x RAST on decrement score ($p = 0.19$) was found.
**Day two.** A repeated measures ANOVA revealed no main effect of condition ($F_{1,9} = 0.19, p = 0.67, \eta^2_p = 0.02; N-\beta = 0.07$), or RAST ($F_{2,18} = 2.6, p = 0.103, \eta^2_p = 0.22; N-\beta = 0.45$) on decrement score during day two, shown in Figure 5. There was no significant interaction effect of condition x RAST on decrement score ($p = 0.56$).

*Figure 7.* Decrement of power during each RAST under control and placebo conditions during day one and day two sessions ($N = 10$).

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**Recovery Score**

**Day one.** Recovery scores during day one and day two sessions for both control and placebo conditions in respective RASTs are shown in Figure 6. A repeated measures ANOVA revealed no main effect for condition ($F_{1,9} = 0.28, p = 0.61, \eta^2_p = 0.03; N-\beta = 0.08$), or RAST ($F_{1,9} = 0.39, p = 0.55, \eta^2_p = 0.04; N-\beta = 0.09$) on recovery score during day one testing. There
was also no significant interaction effect observed for condition x RAST on recovery score ($p = 0.76$).

**Day two.** The repeated measures ANOVA revealed no significant differences for condition ($F_{1, 9} = 3.31, p = 0.102, \eta^2_p = 0.27; N-\beta = 0.37$), or RAST ($F_{1, 9} = 0.49, p = 0.50, \eta^2_p = 0.05; N-\beta = 0.10$) (Figure 6). However, a significant interaction effect was observed for condition x RAST on recovery score ($F_{1, 9} = 5.72, p = 0.04, \eta^2_p = 0.39; N-\beta = 0.57$).

*Figure 8.* Recovery of maximal power output between each RAST under control and placebo conditions during day one and day two sessions ($N = 10$).
Perceived Recovery Status (PRS)

**Day one.** PRS reported directly before all RASTs for both the control and experimental conditions during day one and day two sessions is displayed in Figure 7. A repeated measures ANOVA revealed no main effect of condition on PRS during day one testing ($F_{1, 9} = 0.41, p = 0.537, \eta_p^2 = 0.04$; $\text{N-}\beta = 0.09$). There was a significant main effect of RAST on PRS ($F_{1.262, 11.357} = 87.12, p < 0.01, \eta_p^2 = 0.91; \text{N-}\beta = 1.00$). Post-hoc measures show significantly higher PRS values, indicating a greater perception of recovery status (i.e., more recovered), before RAST 1 when compared to RAST 2 ($p < 0.01; d = 2.48$), as well as before RAST 2 to RAST 3 ($p < 0.01; d = 0.98$). PRS was also found to be significantly higher before RAST 1 than RAST 3 ($p < 0.01; d = 3.41$). All differences in PRS are considered large with respect to effect size. There was no significant interaction observed for condition x RAST on PRS ($p = 0.50$).

**Day two.** The repeated measures ANOVA revealed no significant main effect for condition on PRS ($F_{1, 9} = 0.11, p = 0.751, \eta_p^2 = 0.01; \text{N-}\beta = 0.06$) on day two. There was a significant main effect of RAST on PRS ($F_{2, 18} = 37.80, p < 0.01, \eta_p^2 = 0.80; \text{N-}\beta = 1.00$). Post-hoc analysis revealed that PRS significantly decreased from RAST 1 to RAST 2 ($p = 0.001; d = 0.99$) and from RAST 2 to RAST 3 ($p < 0.01; d = 1.44$). PRS was also found to be significantly higher before RAST 1 than RAST 3 ($p < 0.01; d = 1.85$) which is displayed in Figure 7. Significant differences ($p < 0.05$) were found to have correspondingly large effect sizes. No significant interaction of condition x RAST on PRS was shown ($p = 0.079$).
Figure 9. Perceived recovery reported directly before the beginning of each RAST under control and placebo conditions during day one and day two sessions (N = 10).

Rating of Perceived Exertion (RPE)

Day one. RPE reported during each RAST for both the placebo and experimental conditions during day one and day two sessions is modeled in Figure 8. A repeated measures ANOVA revealed no significant main effect for condition on RPE ($F_{1, 9} = 0.57, p = 0.47, \eta_p^2 = 0.06; N-\beta = 0.10$) during day one testing. However, analyses reveal a significant main effect for RAST on condition ($F_{1, 289, 11, 599} = 9.68, p = 0.007, \eta_p^2 = 0.52; N-\beta = 0.87$) during day one testing. Post-hoc measures show RPE was significantly lower in RAST 1 when compared to RAST 2 ($p = 0.007; d = 0.80$) but not from RAST 2 to RAST 3 ($p = 0.07; d = 0.94$). Participants also reported
significantly higher ratings of perceived exertion in RAST3 than they did in RAST1 ($p = 0.009; d = 1.39$). All difference were found to be large in regards to effect size. No interaction effect of condition x RAST on RPE was observed ($p = 0.426$).

**Day two.** A repeated measures ANOVA revealed no main effect of condition on perceived exertion during the day two session ($F_{1,9} = 1.55, p = 0.24, \eta^2_p = 0.15; N-\beta = 0.20$). There was a significant main effect of RAST on RPE ($F_{1,1,10.1} = 16.08, p < 0.01, \eta^2_p = 0.64; N-\beta = 0.96$). Post-hoc analyses revealed RPE was significantly lower in RAST1 when compared to RAST2 ($p = 0.001; d = 0.70$) and from RAST2 to RAST3 ($p = 0.015; d = 0.69$). Participants also reported significantly higher ratings of perceived exertion in RAST3 than they did in RAST1 ($p = 0.003; d = 1.33$), as shown in Figure 8. Comparisons of all significant differences ($p < 0.05$) were coupled with correspondingly large effect sizes. No interaction effect of condition x RAST on RPE was seen ($p = 0.069$).

*Figure 10.* Mean rating of perceived exertion reported during each RAST under control and placebo conditions during day one and day two sessions (N=10).
Day one. Figure 9 shows perceived pain reported directly after each respective RAST for both the control and experimental conditions during day one and day two sessions. A repeated measures ANOVA revealed no main effect of condition on perceived pain during day one testing \((F_{1, 9} = 1.67, p = 0.23, \eta_p^2 = 0.16; \text{N-}\beta = 0.21)\). There was a significant main effect of RAST on pain \((F_{2, 18} = 27.03, p < 0.01, \eta_p^2 = 0.75; \text{N-}\beta = 1.00)\). Post-hoc analysis showed perceived pain significantly increased from RAST\(_1\) to RAST\(_2\) \((p < 0.01; d = 0.70)\), as well as from RAST\(_2\) to RAST\(_3\) \((p = 0.004; d = 0.71)\). Participants also experienced significantly more perceived pain in RAST\(_1\) than they did in RAST\(_3\) \((p < 0.01; d = 1.63)\), with congruently large effect sizes. A significant interaction effect of condition \(\times\) RAST on perceived pain was observed \((F_{2, 18} = 6.01, p = 0.01, \eta_p^2 = 0.40; \text{N-}\beta = 0.82)\).

Day two. A repeated measures ANOVA revealed no main effect, but did approach significance, for condition on perceived pain \((F_{1, 9} = 4.17, p = 0.07, \eta_p^2 = 0.32; \text{N-}\beta = 0.45)\) during the day two session. There was a main effect of RAST on perceived pain \((F_{1.1.9.9} = 27.7, p < 0.01, \eta_p^2 = 0.81; \text{N-}\beta = 1.0)\). Post-hoc analysis showed perceived pain significantly increased from RAST\(_1\) to RAST\(_2\) \((p < 0.01; d = 1.03)\), as well as from RAST\(_2\) to RAST\(_3\) \((p = 0.001; d = 0.83)\). Participants also experienced significantly more perceived pain in RAST\(_1\) than they did in RAST\(_3\) \((p < 0.01; d = 1.87)\) as shown in Figure 9. All differences were found to have large effect size. No interaction effect was shown for condition \(\times\) RAST on perceived pain \((p = 0.72)\).
Figure 11. Pain reported directly after completion of each RAST under control and placebo conditions during day one and day two sessions (N = 10).

Blood Lactate

Day one. Blood lactate values observed in their respective RAST for both the control and experimental conditions during day one and day two sessions is modeled in Figure 10. A repeated measures ANOVA revealed no main effect of condition on blood lactate during day one ($F_{1, 9} = 0.16, p = 0.701, \eta^2_p = 0.02; N-\beta = 0.07$), however, there was a significant main effect of RAST on blood lactate ($F_{1.3, 11.3} = 50.14, p < 0.01, \eta^2_p = 0.85; N-\beta = 1.00$). Post-hoc analysis showed blood lactate significantly increased from RAST 1 to RAST 2 ($p < 0.01; d = 1.56$), but not from RAST 2 to RAST 3 ($p = 0.169; d = 0.72$). Blood lactate concentrations were also found to be
significantly higher in RAST3 than they were in RAST1 ($p < 0.01; d = 2.19$). All differences were found to have large effect size. No interaction effect was shown for condition x RAST on blood lactate ($p = 0.25$).

**Day two.** A repeated measures ANOVA revealed no main effect of condition on blood lactate on day two ($F_{1, 9} = 1.60, p = 0.24, \eta_p^2 = 0.15; N-\beta = 0.21$), however, there was a significant main effect of RAST on blood lactate ($F_{2, 18} = 64.0, p < 0.01, \eta_p^2 = 0.88; N-\beta = 1.0$) during the day two session. Post-hoc analyses show blood lactate increased significantly from RAST1 to RAST2 ($p < 0.01; d = 2.09$), and from RAST2 to RAST3 ($p = 0.013; d = 0.49$). Blood lactate concentrations were also found to be significantly higher in RAST3 than they were in RAST1 ($p < 0.01; d = 2.98$) (Figure 10). All differences ranged from moderate to large with respect to effect size. No interaction effect was shown for condition x RAST on blood lactate ($p = 0.69$).
Figure 12. Blood lactate levels immediately following each RAST under control and placebo conditions during day one and day two sessions (N = 10).

![Graph showing blood lactate levels](image)

*, #, denotes a significant difference in blood lactate between RASTS during day one (\( p < 0.01 \))
†, ‡, ¥ denotes a significant difference in blood lactate between RASTS during day two (\( p < 0.05 \))

**Session Rating of Perceived Exertion (SRPE)**

Figure 11 displays the session RPE reported after day one and day two sessions for both placebo and control conditions. A paired t-test revealed that session RPE did not significantly differ between control and placebo conditions during the day one session (\( p = 1.00; d = 0.10 \)) or the day two session (\( p = 0.71; d = 0.68 \)), as shown in Figure 11. While not significantly different (\( p > 0.05 \)) differences in between conditions were found to have moderate effect sizes in the day two session compared to only a small effect size during the day one session.
Figure 13. Session rating of perceived exertion reported after completion of testing for control and placebo conditions during day one and day two sessions (N = 10).
The purpose of this study was to determine the potential effect the administration of a placebo may have on performance and perceived recovery. While some studies have investigated the impact a placebo may have during a single session, this is the first study aimed to examine the influence administration of a placebo may have on recovery both within and between sessions. The most salient finding from this study reveals that administration of a placebo tended to yield improved performance, in terms of power output, following 24-hours of recovery. Indeed, participants in the placebo condition demonstrated significantly higher peak and mean power when compared to the control condition during latter portions of the repeated sprint protocol. Interestingly, changes in power output were observed despite a lack of significant differences in either acute or global perceptual strain (e.g., RPE, SRPE, PRS, and perceived pain), or metabolic strain (e.g., blood lactate).

Statistical analyses revealed the placebo condition produced significantly greater peak and mean power outputs in RAST\textsubscript{3} during the day two session (see Figures 1 and 2). These differences were observed independent of any significant changes in perceptual or metabolic response. This finding confirms previous work noting significantly improved performance in high-intensity work with similar RPE and blood lactate responses upon administration of a placebo (Porcari et al., 2006; Wright et al., 2009). Specific to our methodology, it seems that the administration of a placebo was able to attenuate loss of power output during the later stages of a repeated sprint protocol leading to overall improved performance. There has been previous research noting that most individual accustomed to sprint-type training demonstrate are able to reproduce optimal repeated sprint performance with similar perceptual and metabolic measures following only 24 hours of recovery (Laurent et al., 2010). This notion is confirmed as
participants under all conditions were able to reproduce similar performance during a repeated sprint session. Interestingly, though, administration of a placebo seems to produce attenuation of the decline in mean and peak power output. That is, individual receiving the placebo beverage were able to produce significantly higher power outputs towards the end of the session following 24-hours of recovery. Ultimately, what this may suggest is that the effect of a placebo during this mode of performance may become more pronounced during later stages of the session. During these latter portions is most likely when an individual, under normal circumstances, may experience a loss of optimal performance due to the negative consequence of high-intensity work (i.e., pH disruption, metabolic by-product accumulation, etc.) (Glaister, 2005). It seems, though, that the placebo effect may have mitigated this loss of power output which would have elicited the improved sprint performance during the final RAST of the session.

A unique approach used to identify the placebo effect in the current study was to assess perceived pain during the bout. Surprisingly, this is not a measure that has been investigated in placebo research during repeated sprint training despite indications in the literature of the beneficial role of placebo analgesia. While no significant differences were found with respect to perceived pain, the observed measure of pain in the current study may still lend explanation to the observed differences in mean and peak power during the day two session between conditions. As shown in Figure 7, pain increased significantly similarly throughout each RAST during day one and day two sessions for both the control and placebo conditions. The concomitant increase in pain as work increased was expected and is in agreement with other research investigating the relation of pain to exercise performance (Astorino, Terzi, Roberson, & Burnett, 2011; Cook, O’Connor, Eubanks, Smith, & Lee, 1997). However, no significant difference in perceived pain was observed between the control and placebo conditions at any equivalent time point (e.g.,
RAST1 day one of the placebo condition compared to RAST1 day one of the control condition). Of note is that perceived pain between conditions was lower in the placebo condition, albeit not significantly different, in RAST3 during the day two session despite the placebo condition producing significantly higher peak and mean power outputs. It seems plausible, then, that these findings indicate placebo analgesia may have occurred during RAST3. Research has indicated that perception of pain from active muscles increase as power output increases and it may be that the analgesic effect of the placebo modulated the nociceptive signal (Jameson & Ring, 2000). This, in turn, may have resulted in participants in the placebo condition reporting statistically equivalent levels of perceived pain despite higher peak power outputs. Likewise, increases in mean power output may have been due to a decreased afferent signal to the areas of the brain involved in pain processing (e.g., rACC, thalamus, and PAG) (Loyd & Murphy, 2009).

As seen in Figure 10, participants consistently produced similarly high concentrations of blood lactate at parallel time points between conditions. Given the similarity in blood lactate levels it seems that participants exerted the same amount of effort in both conditions during RAST3 of the day two session. Moreover, it seems plausible that this data may indicate an increased motor unit recruitment strategy that could have facilitated increases in peak and mean power. It may be then, as previous literature suggests, that the significantly lower power produced in the control condition may have been a result of down-regulation of motor unit recruitment rather than a decrease effort (Mendez-Villanueva, Hamer, & Bishop, 2008). Further research is needed to examine if administration of a placebo facilitates motor unit recruitment (e.g., recruitment strategies and/or alterations in muscular recruitment).

It was not surprising and, indeed, expected to see RPE rise throughout the bouts (see Figure 6) as a number of studies have shown that RPE increases as the total amount of work
increases (Pollo et al., 2008; Laurent et al., 2010). Interestingly, during latter stages of sprinting bouts following 24 hours of recovery, RPE during and SRPE following the sprints remained similar between conditions. The levels of perceived exertion reported by the participants were similar despite the placebo condition producing significantly higher mean and peak power. It has been suggested that perceptual strain is a mechanism used subconsciously to regulate performance in an effort to titrate recruitment strategies to both optimize performance and prevent muscle damage (Noakes, 2007). In addition, it has been reasoned that overall muscular strain and pain serve as important afferent sensations mediating RPE (Hampson et al., 2001). More recently, Twist and Eston (2009) found that muscle damage impairs performance, increases RPE, and decreases the amount of work able to be completed. It may be that the administration of a placebo can modulate the sensation of pain due to the inhibition of nociceptive signals at the level of the spinal cord (Millan, 2002) which, in turn, may trigger an up-regulation in performance or, at the very least, the attenuation of a decline in performance as seen in this study. Another novel theory that may help explain the attenuation of the decline in peak and mean power observed in the current study can be explained via the anticipatory perceived exertion performance model (Tucker, 2009). Briefly, this model explains that performance is based upon a pre-exercise template designed to up-regulate or down-regulate performance contingent upon the comparison of a person’s expected RPE to their actual RPE during the activity. It is proposed that the decreased nociceptive signal to the CNS, perhaps due to placebo analgesia, may have allowed for consistent up-regulation of performance via increased motor unit recruitment to muscles that would otherwise have been inhibited due to the noxious environment created by metabolite accumulation (e.g., high blood lactate). It seems that a reduced sensation of pain may have precipitated an up-regulation in performance to allow for
greater maintenance of peak and mean power output in the placebo condition despite similar RPEs reported across both conditions. While there are multiple theories that exist attempting to explain the regulation of perception and performance with respect to fatigue and recovery it is important to note that, in most cases, they are not to be viewed as mutually exclusive, rather, integrative in nature (Laurent & Green, 2009).

In this study, relative measures of fatigue rate and recovery rate were used (i.e., DEC score and REC scores, respectively). Findings from the study show that decrements in performance were not significantly different between RASTs or condition suggesting that the decay in power output within a RAST was unaffected by the administration of a placebo. Consequently, the administration and expectancy of a placebo to improve repeated sprint performance was not effective in terms of mediating fatigue within a set of repeated sprints. Additionally, the ability to recover power was not significantly different between RASTs or condition, similar to what was observed with DEC scores. However, these findings are worthy of closer examination. Although conditions were not significantly different in terms of relative power recovered, the placebo condition did consistently produce higher mean power output reaching significance in RAST3. Thus, the placebo condition did recover absolutely (vs. relatively) a significantly higher amount of raw power when compared to the control. It has been shown that significantly larger recovery of raw power may be due to the modulation of a participant’s exercise template (Tucker, 2009). Simply, this model proposes that work rates are modified to match a pre-exercise template based upon changes in RPE. The expectation of a placebo to improve recovery could have facilitated a modification of a pre-exercise template to allow for an increased power output while the modulation of pain via the analgesic effect of the placebo maintained RPE despite the increased work rate. Therefore, the stability in an
individual’s relative rate of REC (i.e., percent recovered to peak power within a session) despite higher power outputs may be a result of the administration of a placebo and/or placebo analgesia. Further research to investigate this is needed and worthy of merit.

As previously noted, SRPE (i.e., global difficulty) was not significantly different across conditions despite the placebo condition producing significantly higher peak and mean power outputs during the day two session. This would be expected given the lack of overall differences in acute RPEs observed during the session. It appears that an ergogenic effect of placebo administration extends beyond the actual session as overall perceived difficulty of the session was not significantly different between conditions while total work performed was greater. That is, despite significantly higher peak and mean power outputs, participants in the placebo conditions did not feel that the session was any more difficult than when power outputs were reduced.

A unique approach to identify changes in recovery was used in this study as many of the resultant effects of placebo administration may be a consequence of changes in perception. Thus, a novel psychophysiological tool, the Perceived Recovery Status Scale, was used to identify any perceived changes in recovery. The PRS was originally designed to monitor recovery on a day to day basis to ensure that overtraining did not occur or to detect under-recovery (Laurent et al., 2011), however, a modified version of this scale was used to assess changes in perceived recovery status relative to expected performance not only between bouts but within a session as well. Results from the study show that PRS values did not vary between RASTs or condition. This is not surprising when considering that neither REC scores, RPE, nor SRPE were significantly different between conditions. It appears the administration of a placebo does not cause a disassociation between subjective recovery and physiological recovery. Moreover, it
seems beneficial that the administration of a placebo, while ergogenic with respect to power production, will not produce a perceived recovery status to be incongruent with physiological recovery. At least within the context of this investigation, an individual will still be able to determine as to whether they are adequately recovered to perform optimally rather than continue training when insufficiently recovered. This, indeed, seems to be an important finding as under-recovery has been identified as one cause of overtraining which can lead to decreases in performance (Kreher & Schwartz, 2012).

While the findings of the current study are novel, one must also note the limitations of the study before attempting to apply these findings. These findings were observed using only one mode of exercise, repeated intermittent sprinting. While the placebo effect has been shown to influence performance across various modes of exercise (Kalasountas et al., 2007; Beedie et al., 2007; Foster et al., 2004), the findings from this study should only be applied to the placebo effect and recovery from repeated sprint work. Additionally, the sample for the current study was homogenous, as it consisted of only 10 college-aged males with very similar sport and exercise training patterns. Research has both supported (Aslaksen, Bystad, Vambheim, & Flaten, 2011) and opposed (Averbuch & Katzper, 2001) sex differences of expectancy based placebo analgesia, but there is a paucity of research aimed at evaluating sex differences in performance after the administration of a placebo. That notwithstanding, practitioners must be cognizant of the sample tested in the current study when applying findings to a broader population. Similarly, the sample size in the current study was small which may limit the application of findings.

Finally, the greater peak and mean power produced was, in this instance, explained as a decline in peripheral fatigue during the placebo condition in the final RAST of day two when compared to the control condition. However, EMG was not a component of the current study and the
decline in peripheral fatigue via increased neural drive is purely speculative, albeit reasonable. Although EMG data could have provided strong evidence for decreased fatigue during the placebo trial, research has shown that muscular fatigue is partly due to feedback from a central governor. The explanation of placebo effect’s influence on the central governing theory of fatigue in the discussion is thought to have given credibility to the increased peripheral fatigue response devoid of EMG data. Futures studies investigating the placebo effect and its impact on recovery should be aware of those limitations and attempt to rectify them in their methodologies.

In conclusion, findings from the current study support the hypothesis that administration of a placebo can precipitate a positive influence on recovery with respect to repeated sprint performance. Specifically, despite similar perceptual and metabolic response through most of the sessions, there were significant differences found between conditions in the final RAST following 24 hours of recovery. These findings suggest the possible impact of the placebo on performance may be mediated by intensity and exercise induced perturbations of homeostasis. It does seem, at least within this mode of exercise, the manifestation of the placebo effect is seen not initially but as exercise continues. While significant differences between conditions were only identified at a single time point, there exist important practical implications from this study. It seems that during sport or exercise sessions that consist of high(er)-intensities, the placebo effect may provide benefit in sustaining overload of the physiological systems by maintaining power output. This, without any negative consequence to levels of perceived exertion during or after the session which may aid in compliance in subsequent training bouts. Importantly, the ability of a placebo to facilitate a greater recovery of raw power without causing a disassociation between perceived recovery status may have important practical implications. That is, a placebo will facilitate optimal performance to allow for overload via a reduction in the down regulation
of performance while still allowing athletes to detect inadequate recovery. The chronic effect of
increases in daily performance may allow for greater physiological adaptations while
concurrently leaving an athlete’s ability to detect signs of overtraining unaffected. Future work
investigating the effect of a placebo during this type of exercise paradigm over longer periods of
time is certainly worthy of merit and warranted.


Benson, H., & Friedman, R. (1996). Harnessing the power of the placebo effect and renaming it "remembered wellness". *Annual Review of Medicine, 47*(1), 193-199.
doi:10.1146/annurev.med.47.1.193


doi:10.1519/JSC.0b013e31816eb518


doi:10.1073/pnas.1430684100


Draper, N. and Whyte, G (1997). Here's a new running based test of anaerobic performance for which you need only a stopwatch and a calculator. *Peak Performance, 97*, 3-5.


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APPENDIX A: INFORMED CONSENT

Informed Consent

Investigator: Danilo Tolusso
Advisor: Dr. Matt Laurent

Phone: (440) 781-9658
Phone: (419) 372-6904

Project Title: Sport Recovery Beverage’s Effect on Recovery Following Intermittent Sprinting Protocol.

Introduction: You are being asked to participate in a study by Danilo Tolusso, a graduate student in the Kinesiology program, in the School of Human Movement, Sport, and Leisure, at Bowling Green State University. His advisor, Dr. Matt Laurent is an Assistant Professor in the School of Human Movement, Sport, and Leisure Studies at Bowling Green State University. The present study is designed to observe the influence a recovery beverage on recovery during intermittent sprinting.

Purpose: The purpose of this study is to determine the effectiveness of a newly formulated sport recovery beverage on individual’s ability to recover during and following sprint work. If the beverage proves to be effective at improving recovery, then this will move the beverage one step closer to becoming commercially available. Upon completion of the study, you will be given your body fat percentage results that will be recorded at the beginning of the study. Subjects will not receive any monetary reward for taking part in the study.

Procedure: If you are eligible and choose to participate, you will be asked to come in once for a familiarization period where you will fill out and read the informed consent and medical history questionnaire. The information from the medical history questionnaire will be used to place you into a risk category, only people considered low risk will be allowed to participate in the study. Exclusion criteria include obesity, cigarette smoking, high blood pressure, family history of heart disease, and high cholesterol. If you meet any two of these criteria then you will not be allowed to participate. To be included in the study you must be 18 years of age or older, as well as participate in sprint training or a sports involving sprinting at least twice per week. If you are allowed to participate, then you will be asked to fill out a practice session, where you will be able to familiarize yourself with the equipment. Body fat percentage, height, and weight will also be recorded during the familiarization day. You will also be given a daily journal to document caffeine and water consumption. This journal will only need to be filled out the day before you come in for testing.

You will then be asked to complete 4 exercise trials. Each trial will consist of the following:

Warm-up
- You will be asked to consume the 600mL of water or recovery drink 20 minutes prior to exercise
- You will be guided through a warm-up consisting of a 4 minute walk at 3.7 mph, followed by a 2 minute run at 7.5 mph
- After you walk and run, you will be asked to do 3 sets of 10 toe raises, 20 high knee marches, and 20 butt kicks

Intermittent Protocol
- You will be asked to perform 6,35 meter sprints with 10 seconds of recovery between each sprint
- After each sprint is completed, you will be asked to rate how hard you worked on a 0-10 scale
- After you have completed the 6 sprints, you will be allowed to rest for 7 minutes
- You will then be given and asked to drink 150mL of water or recovery substance to drink

- You will be asked to rate your pain using a subjective scale
- Then a minimal amount of blood will be taken via finger prick on your fingertip. The amount of blood drawn out on the finger prick will be very low, about the size of a pen tip
- Lastly you will be asked to rate how recovered you feel using a 0-10 scale
- You will complete the six, 35 meter sprint protocol a total of three times
- After the last set of sprints, you will be seated comfortably for 15-20 minutes and after that time will be asked to rate how hard the entire session was and how effective you feel the recovery beverage was.
- After you have completed the three sets of sprints, you will be given another recovery drink to help speed up overnight recovery

You will come in the next day and complete another three sets of 6x35 meter sprints and under the same procedures.

Between 7-14 days after you have completed the first two rounds of sprint work, either with or without the recovery beverage, you will be asked to complete an identical protocol on back to back days once again. The procedures will be identical except that you will either receive water or the recovery beverage, whichever you did not receive in the first round of testing. The order in which each individual completes the trials is random. However, you will complete the protocol with and without the recovery substance and you will know which trial will be completed prior to your testing day. After the study has been completed, you will receive an e-mail from the primary researcher. This email will consist of a one question questionnaire.

This study will take a total of five days to complete, a familiarization day, two back-to-back testing days with the recovery drink, and two back-to-back testing days without the recovery drink. Each day will take no more than an hour to complete the protocol, for a total of five hours for the entire study. All testing sessions will take place in Eppler South, room 124 or 101.

**Voluntary nature:** Your participation is completely voluntary. You are free to withdraw at any time. You may decide to discontinue participation at any time without penalty. Deciding to participate or not will not affect your grades or your relationship with Bowling Green State University.

**Confidentiality Protection:** All data recorded during the course of the study will be stored on a password protected computer. All documents obtained from the subjects will be stored in a locked filing cabinet in a locked office. Only members of the research team will have access to both recorded data and documents during the study. Subject data will be coded to maintain participant confidentiality and kept for three years upon completion of the study.

**Risks:** The risks of this study are no different than any other high intensity sprinting that you may complete during your training or sporting event. There are potential risks to your health while participating in the study including: 1) cardiovascular injury (heart attack, stroke and death- risk is estimated at <0.01%), 2) Shortness of breath, lightheadedness, dizziness, and nausea, 3) all other possible risks associated with exercise. While there is a risk of a cardiovascular injury, the chance is very low.

The sprinting protocol is very intense; you may become nauseous or lightheaded during or after testing. If you are feeling nauseous or lightheaded, you will be asked to remain in the lab until the symptoms have subsided. If a serious injury does happen, two investigators certified in first aid and CPR will provide immediate care and an ambulance will be called if necessary. You will be required to pay for any medical service that may be needed. In an attempt to avoid any need for medical services, researchers will immediately terminate the testing procedures if you experience chest pain, shortness of breath, wheezing, leg cramps, severe leg pain, light-headedness, confusion, or nausea. If you report or we suspect any of these symptoms during testing, testing will be stopped and you may no longer take part in the study.

**Contact information:** If you have any questions, concerns, or comments, you may contact Danilo Tolusso at (440) 781-9658, danilot@bgsu.edu, or Dr. Matt Laurent at (419) 372-6904, cmlaure@bgsu.edu. If you experience any psychological distress as a result of your participation in this study, then you may contact the Bowling Green State University Counseling Center (419-372-2081). You may also contact the Chair, Human Subjects Review Board at 419-372-7716 or hsrb@bgsu.edu, if you have any questions about your rights as a participant in this research.
The investigators in this study would like to thank you for your time and commitment. Without you, this study would not be possible.

I have been informed of the purposes, procedures, risks and benefits of this study. I have had the opportunity to have all my questions answered and I have been informed that my participation is completely voluntary. I agree to participate in this research.

_____________________________________
Participant Signature
MEDICAL HISTORY QUESTIONNAIRE

All information given is personal and confidential. It will enable us to better understand you and your health and fitness habits. In addition, we will use this information to classify your health status according to the American College of Sport Medicine (ACSM) recommendations for risk stratification (ACSM, 2009). Please let us know if and when you have changed your medication (dose & type), diet, exercise or sleeping habits within the past 24 or 48 hours. It is very important for you to provide us with this information.

NAME______________________________________________ AGE___________________ DATE___________________

OCCUPATION________________________________________________________________________________________

1. **FAMILY HISTORY**

Check each as it applies to a blood relative:

- **Heart Attack**  yes______ no______ unsure______
  - If yes, age at onset ____ yrs; relation to you _____________
- **Sudden Death**  yes______ no______ unsure______
  - If yes, age at onset ____ yrs; relation to you _____________
- **Coronary Revascularization**
  - If yes, age at onset ____ yrs; relation to you _____________

Father’s Age _____ Deceased_____ Age at death_____

(*Before 55 yr. in father or first-degree male relative)

- **Tuberculosis**  yes______ no______ unsure______
- **Stroke**  yes______ no______ unsure______
- **Asthma**  yes______ no______ unsure______
- **High Blood Pressure**  yes______ no______ unsure______
- **Circulatory Disorder**  yes______ no______ unsure______
- **Heart Disease**  yes______ no______ unsure______

Mother’s Age _____ Deceased_____ Age at death_____

(*Before 65 yr. in mother or first-degree female relative)

2. **PERSONAL HISTORY**

Check each as it applies to you:

- **Age** (men ≥ 45 yr; women≥ 55 yr)  yes______ no______
- **Current Cigarette Smoking**  yes______ no______ unsure______
- **Sedentary Lifestyle**  yes______ no______ unsure______
  - Persons not participating in at least 30 min of moderate intensity physical activity on at least 3 days/wk for at least 3 months.
- **Obesity – BMI >30 kg·m⁻²**  yes______ no______ unsure______
  - If yes, give value: __________ kg·m⁻²
- **Waist circum. > 40” men; 35” women**  yes______ no______
- **High Blood Pressure**  yes______ no______ unsure______
  - Systolic Blood Pressure >140mmHg or diastolic >90mmHg
  - If yes, give value: __________ mmHg.
- **Dyslipidemia**  yes______ no______ unsure______
  - Total Serum Cholesterol >200 mg·dl⁻¹; value:______ mg·dl⁻¹
  - LDL-C ≥ 130 mg·dl⁻¹; value:______ mg·dl⁻¹
  - HDL-C ≤ 40 mg·dl⁻¹; value:______ mg·dl⁻¹
  - On lipid lowering medication: yes______ no______ unsure______
- **PreDiabetes**  yes______ no______ unsure______
  - If yes, age of onset:________ years
  - Impaired fasting glucose ≥ 100 mg dl⁻¹; value:______ mg dl⁻¹
  - Impaired glucose tolerance test: yes______ no______
  - (Note: values confirmed by measures on two separate occasions)
- **Negative Risk Factor**: yes______ no______ unsure______
  - HDL ≥ 60 mg dl⁻¹; value:______ mg dl⁻¹

Have you ever had:

- **Diabetes**  yes______ no______ unsure______
- **Tuberculosis**  yes______ no______ unsure______
- **Heart Attack**  yes______ no______ unsure______
- **Angina**  yes______ no______ unsure______
- **EKG Abnormalities**  yes______ no______ unsure______
- **Asthma**  yes______ no______ unsure______
- **Emphysema**  yes______ no______ unsure______
- **Surgery**  yes______ no______ unsure______
- **Stroke**  yes______ no______ unsure______
- **Severe Illness**  yes______ no______ unsure______
- **Hospitalized**  yes______ no______ unsure______
- **Black Outs**  yes______ no______ unsure______
- **Gout**  yes______ no______ unsure______
- **Nervousness**  yes______ no______ unsure______
- **Joint Problems**  yes______ no______ unsure______
- **Allergy**  yes______ no______ unsure______
- **Convulsions**  yes______ no______ unsure______
- **Paralysis**  yes______ no______ unsure______
- **Headaches**  yes______ no______ unsure______
- **Depression**  yes______ no______ unsure______
- **Chest Pain**  yes______ no______ unsure______
- **Arm Pain**  yes______ no______ unsure______
- **Shortness of Breath**  yes______ no______ unsure______
- **Indigestion**  yes______ no______ unsure______
- **Ulcers**  yes______ no______ unsure______
- **Overweight**  yes______ no______ unsure______
- **Hernia**  yes______ no______ unsure______
- **Back Pain**  yes______ no______ unsure______
- **Leg Cramps**  yes______ no______ unsure______
- **Low Blood Pressure**  yes______ no______ unsure______
- **Insomnia**  yes______ no______ unsure______
3. MEDICAL HISTORY

Name of your physician__________________________________________________________________________________

Date of your most recent physical examination________________________________________________________________

What did the physical examination include?_________________________________________________________________

Have you ever had an exercise EKG? Yes_______ No________

Are you presently taking any medications? Yes_______ No_______ _______________________________________________

(Including over-the-counter medications and/or herbs) List name and dosage

Have you ever taken:

Digitalis yes______ no______ unsure______
Nitroglycerin yes______ no______ unsure______
High Blood Pressure yes______ no______ unsure______
Medication
Sedatives yes______ no______ unsure______
Inderal yes______ no______ unsure______
Insulin yes______ no______ unsure______
Pronestyl yes______ no______ unsure______
Vasodilators yes______ no______ unsure______
Other yes______ no______ unsure______
If yes, list medications:

4. EXERCISE HISTORY

Do you exercise? Yes_______ No_______ What activity____________________________________________________________

How long have you been exercising?________________________________________________________

How many days do you exercise? _______________ How many minutes per day? __________________________

What kinds of shoes do you work out in?___________________________________________________________

Where do you usually exercise?______________________________________________________________

Do you monitor your pulse during your workout?_________________________________________________
Do you believe nutritional supplements can have a positive effect on performance?

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5. HEALTH HISTORY

Height______ Weight______

Do you use Health Foods? Yes_____ No_____ List____________________________

Do you take Vitamin pills? Yes_____ No_____ List____________________________

Approximate your daily intake: Coffee_______ tea________ coke______ beer______ wine______ liquor______

Do you smoke or use tobacco products? Yes_____ No_____

If yes, approximate your daily usage: Cigarettes______ Cigars______ Pipes______ Chewing Tobacco______

Did you ever smoke? Yes_____ No_____ How many years?______________ Age when you quit_________

Approximate the number of hours you work per week?______________ Vacations weeks per year___________________

Home Status: Very happy______ Pleasant______ Difficult______ Problem______

Work Status: Very happy______ Pleasant______ Difficult______ Problem______

Do you feel you are stressed? Yes_____ No_____ Unsure_______

Are you worried about your health? Yes_____ No_____ Unsure_______

6. APPROXIMATE A TYPICAL 24 HOUR DAY FOR YOU

Number of hours:

Work
TV
Relaxation/Leisure activities
Driving/Riding
Eating
Exercise
Sleep
TOTAL
Additional information from client interview to further assess health/coronary risk status:

_____________________________________________________________________________________________________
_____________________________________________________________________________________________________
_____________________________________________________________________________________________________
_____________________________________________________________________________________________________

Signature of Tester ___________________________ Date 08/30/09
Findings from lab testing

A supplement company has asked the kinesiology program here at BGSU to validate a new FDA approved recovery supplement that they have designed. The company has performed extensive research on the supplement’s impact on performance and now they are seeking validation of the supplement from an independent agency.

- The supplement company has reported the following findings from their own research:
  - Decreased muscle fatigue
  - Greater increase in sprint speeds achieved
  - A greater recovery rate
  - Faster recovery during workout allowed for greater force production
  - Faster completion times in middle distance aerobic events
  - 75% of subjects ran faster when using the recovery beverage
Hey everyone, my name is Dano Tolusso, and I’m a second year graduate student here at BGSU. I am looking for participants for my thesis. A supplement company asked Dr. Laurent and I to do some testing on a new sports drink they plan on putting out on the market. The company wants us to see if their supplement can improve sprinting performance during repeated sprint bouts. In order to be included in the study you must be classified as low risk, according to American College of Sports Medicine guidelines. You must not have high blood pressure, smoke, or have any type of metabolic disease to be included in this study. In addition, must also already take part in sprint training or a sport that involves brief periods of sprinting at least two days a week. Deciding to either participate or not participate in my study will have no effect on your grade or standing with BGSU. If you have any questions or interest please feel free to contact me. However, you contacting me is only to hear more about the study and what is involved and does not mean you are obligated to actually be in the study. It just means you would like more information.

- I will hand out flyers to all students before speaking.
APPENDIX E: DEBRIEFING LETTER

“Sports Recovery Beverage’s Effect on Recovery Following Intermittent Sprinting Protocol”

Dear Participant;

During this study, you were asked to ingest a sports recovery beverage and perform three maximal sprint bouts on consecutive days. You were told that the purpose of the study was to determine the effectiveness of a newly formulated sports recovery beverage on perceived recovery. The actual purpose of the study was to determine if the placebo effect has an influence on perceptual and physiological recovery. The sports recovery beverage was actually flavored water that we used as the placebo for the study. We wanted to see if you had an improved perceived recovery just because you believed the substance would help you recover. This research may allow for research to get a better understanding about some of the mediating factors behind perceptual recovery, such as belief and expectancy for something to work. In addition, information from this study will help nutrition and supplement companies that are producing recovery beverages know the extent to which their product does or does not actually aid in recovery beyond what is driven by pure belief in a product’s effect.

We did not tell you everything about the purpose of the study because in order to possibly experience a placebo effect, the participants needed to believe that the substance that they were taking would have an effect.

You are reminded that your original consent document included the following information:

Your participation is completely voluntary. You are free to withdraw at any time. You may decide to discontinue participation at any time without penalty. Deciding to participate or not will not affect your grades or your relationship with Bowling Green State University.

If you have any concerns about your participation or the data you provided in light of this disclosure, please discuss this with us. We will be happy to provide any information we can to help answer questions you have about this study.

If your concerns are such that you would now like to have your data withdrawn, and the data is identifiable, we will do so.

If you have questions about your participation in the study, please contact me at 440-781-9658, danilot@bgsu.edu, or my faculty advisor, Dr. Matt Laurent at 419-372-6904 or cmalaure@bgsu.edu.

If you have questions about your rights as a research participant, you may contact the Office of Human Subject Research (419-372-7716, hrsb@bgsu.edu)

If you have experienced any psychological distress as a result of your participation in this study, then you may contact the Bowling Green State University Counseling Center (419-372-2081)

Please again accept our appreciation for your participation in this study.
APPENDIX F: RECRUITMENT FLYER FOR GENERAL POPULATION

ARE YOU INTERESTED IN TESTING OUT A NEW SPORTS RECOVERY DRINK?

A Masters Student is in need of participants to test the effectiveness of a new sports recovery drink. The study will take a total of approximately 5 hours over the course of 5 days.

To qualify for this study:

1. You must already be participating in sprint training or a sport involving bouts of sprinting at least two times a week.
2. Must not have high blood pressure or smoke
3. Must not have any metabolic conditions (e.g. diabetes)

If you are interested or have any questions about the research, please contact:

Dano Tuluoso

Phone: (440) 781-9658

E-mail: danilot@bgsu.edu
APPENDIX G: RECRUITMENT FLYER FOR CLUB ATHLETES

ARE YOU A CLUB ATHLETE INTERESTED IN TESTING OUT A NEW SPORTS RECOVERY DRINK?

A Masters Student is in need of participants to test the effectiveness of a new sports recovery drink. The study will take a total of approximately 5 hours over the course of 5 days.

To qualify for this study:

1. You must already be participating in sprint training or a sport involving bouts of sprinting at least two times a week.
2. Must not have high blood pressure or smoke
3. Must not have any metabolic conditions (e.g. diabetes)

If you are interested or have any questions about the research, please contact:

Dano Toluoso
Phone: (440) 781-9658
E-mail: danilot@bgsu.edu
APPENDIX H: ADULT OMNI SCALE OF PERCEIVED EXERTION FOR RUNNING
(UTTER ET AL., 2004)
APPENDIX I: VAS PAIN SCALE

RAST 1

No pain at all ➔ Almost unbearable pain

RAST 2

No pain at all ➔ Almost unbearable pain

RAST 3

No pain at all ➔ Almost unbearable pain
APPENDIX J: SESSION RPE (FOSTER ET AL., 2001)

<table>
<thead>
<tr>
<th>Rating</th>
<th>Descriptor</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Rest</td>
</tr>
<tr>
<td>2</td>
<td>Very, very easy</td>
</tr>
<tr>
<td>3</td>
<td>Moderate</td>
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<tr>
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<td>Somewhat hard</td>
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<tr>
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<td>-</td>
</tr>
<tr>
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<td>Very Hard</td>
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<td>8</td>
<td>-</td>
</tr>
<tr>
<td>9</td>
<td>-</td>
</tr>
<tr>
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