# The Effect of Weighted-Vest Sprints on Run Performance 

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#### Abstract

Warm-ups are thought to acutely increase athletic performance by increasing blood flow, motor unit recruitment, and accelerating metabolism. Post activation potentiation (PAP) may be an underlying mechanism lending to an increase in athletic performance following a warm-up. PAP has been defined as the phenomenon by which muscular performance characteristics are enhanced because of muscle's contractile history. An underlying physiological mechanism of PAP may be myosin regulatory light chain phosphorylation. There may be an acute effect of PAP on athletic performance, which is short in duration involving rapid, powerful movements. Although, PAP's contribution to acute performance enhancement remains speculative, as other mechanisms (e.g., higher-order motor unit recruitment, increased leg stiffness, increased $\mathrm{VO}_{2}$ kinetics) may be at play. Thus, 'pre-conditioning' is used to encompass a multitude of physiological mechanisms contributing to improved sports performance. Several studies have provided evidence of pre-conditioning enhancing cycling and rowing performance; however, there is a paucity of data describing preconditioning's efficacy on distance-running performance. Due to the elastic nature of running (i.e., reutilization of absorbed mechanical energy by leg muscles when contacting the ground) and possible acute increase in leg stiffness following pre-conditioning, incorporating resistance exercises into a distance running warm-up may benefit a distance running population. Thus, the primary purpose of the proposed study was to determine the effect of a pre-conditioning resistance exercise (i.e., PAP induction) on running performance in recreationally trained distance runners. Seventeen recreational male distance runners $\left(\mathrm{VO}_{2 \text { peak }} \geq 50 \mathrm{ml} \cdot \mathrm{kg}^{-1} \cdot \mathrm{~min}^{-1}\right)$ were recruited for this study. The study was a counterbalanced crossover design requiring each participant to visit the laboratory on 5 separate occasions: Visit 1 - Familiarization and baseline testing; Visit 2 - Pre-conditioning with weighted vest or pre-conditioning with no additional weight followed by GXT; Visit 3 - Preconditioning with weighted vest or pre-conditioning with no additional weight followed by GXT; Visit 4 - Pre-conditioning weighted vest or pre-conditioning with no additional weight followed by 1600$m$ time trial (TT); Visit 5 - Pre-conditioning weighted vest or pre-conditioning with no additional weight followed by 1600-m TT. Visits measured the effect of a pre-conditioning loaded exercise, weighted-vest sprints ( $20 \%$ body mass), on 2 endurance running assessments (GXT treadmill run and a 1600-m max effort TT). Additionally, a control trial (pre-conditioning with no additional weight) was completed for each running assessment to identify the effect of pre-conditioning weighted-vest sprints. GXTs assessed $\mathrm{VO}_{2}$ kinetics (time constant and amplitude), submaximal $\mathrm{VO}_{2}(3.13,3.58$, 4.02, and $4.47 \mathrm{~m} \cdot \mathrm{~s}^{-1}$ ), ventilatory thresholds $1\left(\mathrm{VT}_{1}\right) \& 2\left(\mathrm{VT}_{2}\right)$, and peak oxygen uptake ( $\mathrm{VO}_{2 \text { peak }}$ ) via indirect calorimetry; resting lactate, lactate threshold (LT), onset of blood lactate accumulation (OBLA), and peak lactate via finger capillary blood samples; and leg stiffness ( $\mathrm{k}_{\mathrm{leg}}$ ) via accelerometry on a motorized treadmill. The 1600-m TT assessed $\mathrm{k}_{\text {leg }}$ via accelerometry and running performance using time-to-completion and 400-m split times. All variables were analyzed with a repeated measures two-way ANOVA. Statistical analyses were performed with SPSS v.28. Data are reported as mean $\pm$ SD. Significance was accepted at $p<0.05$. Submaximal $\mathrm{VO}_{2}$ at $3.13,3.58$, and $4.02 \mathrm{~m} \cdot \mathrm{~s}^{-1}$ during the GXT was significantly decreased ( $p<0.01$ for all) following weighted-vest sprints compared to control. Resting lactate, and $\mathrm{VT}_{1}$ and $\mathrm{VT}_{2}$ were significantly greater following weightedvest sprints compared to control ( $p<0.05$ for all). Time-to-completion during the 1600-m TT did not differ between warm-up conditions ( $p>0.05$ ). Weighted-vest sprints acutely improved submaximal running economy in recreationally trained distance runners but had no effect on middle-distance running performance during a 1600-m TT. Therefore, weighted-vest sprints may not be useful in improving middle-distance running performance, but may be utilized to improve running economy during submaximal run training.


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## CHAPTER 1: INTRODUCTION

Athletes commonly engage in a general warm-up at a low-intensity ( $50-70 \% \mathrm{VO}_{2 \text { max }}$ ) for approximately 5-10 min, to facilitate an increase in body temperature, acceleration of metabolism, and working capacities of heart and lungs (114) (62). In addition, short bouts of high-intensity (>85\% $\mathrm{VO}_{2 \text { max }}$ ) exercise are performed to enhance blood flow, motor unit recruitment, and oxygen uptake among other physiological processes, demonstrating subsequent short-term increases in performance compared to no warm-up or general warm-up exclusively $(11,78)$. Recently, research investigations have explored techniques to acutely improve athletic performance beyond what has been observed following general and high-intensity warm-ups. Although in opposition to traditional athletic dogma, incorporating resistance exercises into a warm-up may provide athletes with a performance advantage compared to athletes who engage in the typical warm-ups previously described (e.g., 10 min of low-intensity continuous running or cycling, $6 \times 10$ s running sprints without an external load). Resistance exercises prior to athletic performance may acutely promote a physiological mechanism known as post-activation potentiation (PAP).

The induction of PAP following a warm-up has been suggested as a mechanism that may improve exercise performance. During muscular contractions in striated muscle, $\mathrm{Ca}^{2+}$ released from the sarcoplasmic reticulum diffuses into the interior of myofibrils and binds with the regulatory protein troponin, causing a conformational change in tropomyosin and exposes the active binding sites on actin allowing myosin to bind and initiate cross-bridge cycling (i.e., cyclical binding of actin and myosin during muscular contractions) (101). PAP is an enhancement in muscle twitch properties due to contractile history and has been theorized as the transient phosphorylation of the regulatory light chain (RLC) of myosin via myosin light chain kinase (MLCK), which increases Ca $^{2+}$ sensitivity in striated muscle (5). In theory, during muscular contraction the actin-myosin interaction is more sensitive to $\mathrm{Ca}^{2+}$ released from the sarcoplasmic reticulum due to a conformational change of the

RLC, exposing the myosin heavy chain and increasing the rate by which myosin cross-bridges move from a non-force producing state to a force producing state (5), thus enhancing force production during subsequent movements. Therefore, PAP may allow athletes to improve sports performance by increasing muscle force production during a given sports-specific movement (e.g., jump higher, sprint faster).

Numerous studies have been conducted investigating PAP's effect on power-based sports $(5,65,83,138,155,164)$. A meta-analysis conducted by Seitz and Haff (137) concluded that PAP is actuated 5-10 min after maximal voluntary contractions (MVC) and could be an effective conditioning stimulus to increase muscle power output during jump, sprint, throw, and upper-body ballistic performances. However, this phenomenon has also been observed in endurance athletes (i.e., Canadian National Triathletes) following MVCs (67). Skof and Strojnik (143) demonstrated that well-trained middle-distance runners enhanced neuromuscular efficiency (electrical stimulation and electromyography) of the vastus lateralis, vastus medialis, and rectus femoris with the incorporation of bouncing and sprinting into a standardized warm-up. Maximal torque and muscle activation were significantly greater during a maximal knee extension assessment following the warm-up with ballistic movements (143); however, this study only evaluated neuromuscular function, and did not include a direct performance measure (e.g., time trial; TT ) or a control condition. Conversely, Feros et al. (47) utilized a performance assessment with elite rowers and demonstrated significant increases in mean power output (+6.6\%) and stroke rate (+5.2\%), as well as decreased time-tocompletion ( $-1.9 \%$ ) during the first half of a 1000-m rowing ergometer TT following a preconditioning resistance exercise. The warm-up included $5 \times 5 \mathrm{~s}$ ( 2 ss at sub-maximal intensity, and 3 ss at maximal intensity) isometric contractions (i.e., pulling an immovable handle on a rowing ergometer) compared to a standardized warm-up without MVCs (47). Feros et al. (47) suggests a benefit of PAP during the first half of the $T T$, as there was no difference in time-to-completion over
the entire TT. Although speculative, elastic energy provision (i.e., mechanical energy stored in musculotendinous units) may not be optimized due to the non-elastic nature of rowing; thus, PAP may have a greater effect on an elastic modality such as running.

The metabolic cost of running (i.e., running economy) is moderated by contractile and structural proteins constituting the musculotendinous units of the legs, with a decrease in metabolic cost related to a greater capacity to store and release potential energy within these proteins (i.e., increased leg stiffness) (49). During running, when the foot makes contact with the ground, the muscle is forcibly stretched and mechanical energy is absorbed by the muscle (i.e., elastic potential energy) (32). Stored mechanical energy can be subsequently re-utilized during active shortening of the muscle (i.e., concentric contraction) (32). The proposed theory of increased cross-bridge cycling following pre-conditioning resistance exercises may acutely enhance an individual's capacity to store elastic potential energy; thus, increasing energy conservation and reducing the metabolic demands of running (134). Furthermore, individuals that present with type II fiber predominance may show enhanced PAP compared to those with more type I fibers (137). Research suggests that decreased cost of sub-maximal running in middle-distance runners is correlated with type IIx and IIa isoforms (r $=-0.69)(117)$. Only one study to date has investigated the pre-conditioning effect of resistance exercises on the metabolic cost of running and leg stiffness (12). With the use of a weighted vest (20\% body mass), Barnes and colleagues (12) determined that $6 \times 10$ s strides improved $(-6.0 \%$ in oxygen uptake) running economy (i.e., reduced oxygen consumption) during 5 min of sub-maximal treadmill running compared to $6 \times 10$ s strides with no weighted vest. Furthermore, in the aforementioned study a moderate improvement in vertical stiffness was observed ( $12.5 \pm 2.7 \mathrm{kN} \cdot \mathrm{m}^{-1}$ vs $10.3 \pm 2.1 \mathrm{kN} \cdot \mathrm{m}^{-1}$ ) during a vertical jump test. However, it is difficult to infer that leg stiffness increased during running since this was not determined during the running trials. Thus, it remains to
be determined whether an increase in running economy following pre-conditioning resistance exercises is associated with a concomitant increase in leg stiffness during running.

Resistance exercises have been used to study PAP effects on squats jumps (88), loaded countermovement jumps (CMJs) (103), and leg press and leg extensions (isometric and isokinetic) (137). Additionally, a combination of exercises has been utilized for pre-conditioning, referred to as a complex training sequence (65). An example of this is 5 RM squats coupled with 5 CMJ . However, the aforementioned exercises may lack specificity to running, whether sprinting or distance running, and may not optimize subsequent run performance. Running involves a coordinated pattern of lower extremity muscle activation that stabilize and propel the body forward. Forces in the horizontal and vertical planes are produced to move the body forward in space. Horizontal or propulsive forces observed during sprinting hold greater importance to maximal acceleration and speed than vertical ground reaction forces (107). Therefore, exercises that initiate horizontal and vertical propulsive forces may be an effective pre-conditioning stimulus prior to running.

When selecting loaded pre-conditioning exercises, it may be prudent to choose exercises that target muscles that are primarily activated and are the greatest contributors to the movement during the performance activity. Plantar flexors (i.e., gastrocnemius and soleus) and quadriceps have been shown to be the greatest contributors to the vertical ground reaction force compared to other activated muscles during distance running (41). These muscles may provide up to $75 \%$ of the total vertical support impulse during the stance phase and this coordination pattern holds true for speeds up to $7.0 \mathrm{~m} \cdot \mathrm{~s}^{-1}(41)$. Thus, potentiating (i.e., pre-conditioning) the plantar flexors and quadriceps through resistance exercises that target these specific muscles may optimize performance during distance running due to an acute increase in force production. An exercise that may provide adequate utility in muscle potentiation during endurance running is weighted-vest sprinting. Weighted-vest sprinting may provide a movement specific to distance running, in conjunction with
providing an increased force demand with the addition of the weighted vest. The imposed load during a given pre-conditioning activity that is attempting to generate the appropriate PAP response during the targeted activity must match the skill force demand (155).

To date, there are two studies investigating the effects of warming up with a weighted vest on distance running. Barnes et al. (12) observed a moderate improvement in vertical stiffness $(20.4 \% \pm 4.2 \%)$ and large improvement in running economy $(6.0 \% \pm 1.6 \%)$ following six $10-$ s strides with a weighted vest ( $20 \%$ body mass). It seems an adequate stimulus was induced to observe improvements during running in this study. In contrast, O-Neal et al. (111) investigated the effects of $80-\mathrm{m}$ loaded strides ( $10.4 \% \pm 1.1 \%$ body mass) on $5-\mathrm{km}$ running performance, and reported no difference in 5-km time-to-completion following loaded strides compared to unloaded strides (control). The limitation in the previous study may have been an inadequate load as well as submaximal sprinting (i.e., stride), which in combination may not have matched the skill force demand of a $5-\mathrm{km}$ time trial. Furthermore, PAP may diminish before completing a $5-\mathrm{km}$ run, given PAP's time course $(104,137)$. In combination, the results of the two aforementioned studies suggest that weighted-vest sprints may acutely improve distance running performance if 1) the pre-conditioning stimulus matches or exceeds the forces produced during the subsequent running performance and 2) the subsequent running performance is initiated and completed within the PAP time frame.

Due to data demonstrating acute improvements in running economy following weightedvest sprints (12), there may likely be an acute increase in physiological parameters (e.g., ventilatory thresholds $1\left(\mathrm{VT}_{1}\right)$ and $2\left(\mathrm{VT}_{2}\right)$ and lactate threshold (LT)) that are highly correlated with distancerunning performance (121). Currently, there is no literature demonstrating the effects of weightedvest sprinting on physiological parameters during a graded exercise test (GXT). Additionally, it remains speculative whether weighted-vest sprinting has an acute effect on distance-running performance (e.g., TT). The majority of the research showing a PAP effect on performance in
endurance athletes is in modalities such as rowing and cycling; however, these studies either have shown a performance improvement during a portion of the trial or did not include a control $(35,47,141)$. Therefore, it is a necessary to assess the effects of loaded pre-conditioning on distancerunning performance.

## CHAPTER 2: REVIEW OF LITERATURE

The benefits of a warm-up (i.e., general and high-intensity warm-ups) can be explained by increased blood flow (86), recruitment of higher-order motor units (26), elevated oxygen uptake $\left(\mathrm{VO}_{2}\right)$ kinetics (63), and increased anaerobic metabolism (62). Additionally, the phosphorylation of myosin regulatory light chain (MRLC) following a warm-up has been suggested as a mechanism that may positively influence exercise performance. MRLC phosphorylation has commonly been referred to as post-activation potentiation (PAP) (5). PAP has been defined as an enhancement in peak twitch force and rate of force development (RFD), and decrease in the time-to-peak force due to contractile history (e.g., evoked tetanic contraction, evoked twitches, and maximal voluntary contraction (MVC)) (75). Extensive evidence demonstrates enhanced muscle performance produced by changes in muscle twitch properties (17,47,138,141,155). However, few studies adequately demonstrate a direct contribution of PAP to subsequent performance enhancement $(50,83,143)$. Recently, the term post-activation performance enhancement (PAPE) (18) has been proposed due to the paucity of research showing a conflicting timeline between PAPE and PAP in observed increases in force production following a pre-conditioning activity.

PAP may have little effect on increases in subsequent performance due to PAPs time course (i.e., peak increase in force production $10-15$ min post pre-conditioning) $(18,104,138)$, thus some research (18) suggests that PAPE be discerned from PAP, as the two may not be interchangeable. The PAP phenomenon has a short half-life ( $\sim 28 \mathrm{ss}$ ), which may be explained by the phosphorylation
of MRLC, quantified by measuring muscle twitch force responses to a bout of muscular activity (18). However, enhancements in force production are observed for several min following MVCs, which has rarely matched the time course of muscle twitch force responses (164). Consequently, researchers question whether PAP contributes to PAPE in vivo in humans. For the purpose of this review, PAP encompasses all the aforementioned physiological mechanisms contributing to an acute increase in exercise performance.

## Post-Activation Potentiation (PAP): Physiological Mechanisms

There are several proposed modulating factors contributing to acute muscle potentiation following pre-conditioning activities $(24,67,83)$. The two primary modulating factors are the phosphorylation of MRLC and the recruitment of higher-order motor units, which may be the greatest adaptations contributing to an acute increase in muscle power output (134). Evidence for MRLC phosphorylation is sparse in humans (primarily observed in vitro and animal models) $(76,156)$; thus, it is suggested that the enhancement of neuromuscular performance may be an equally contributing mechanism to increasing running and jumping performance (97). Additionally, increased blood flow and $\mathrm{VO}_{2}$ kinetics may be pivotal in acute cycling performance enhancement following pre-conditioning $(153,158)$. Lastly, an acute increase in limb stiffness may play a critical role in the PAP response. In combination, these factors may explain the phenomenon of acute increases in athletic performance following pre-conditioning.

## Muscle Fiber Composition, Type, and Speed of Contraction

Mammalian muscles fibers are composed of sarcomeres, which are the functional units that generate movement (101). Myofibrillar proteins, myosin (thick filaments) and actin (thin filaments), within each sarcomere bind to create cross-bridges and produce muscle contractions (101). A spectrum of fiber type classifications has been reported in the literature, and the differentiation of
these fibers is based on different myosin isoforms and other physiological structures (e.g., mitochondria, myoglobin, capillaries, sarcoplasmic reticulum) (146). The myosin molecule consists of six subunits, two heavy chains and four light chains. Light chains can be subdivided into essential and regulatory light chains (RLC). Although uncertain, the essential light chain is suggested to contribute to the structural stability of myosin (5). The RLC is involved in muscle contraction due to its ability to phosphorylate (3). The energy required to facilitate the myosin-actin interaction and muscle contraction is provided by adenosine triphosphate (ATP), which binds to the myosin heavy chain and is hydrolyzed by adenosine triphosphatase (ATPase), yielding adenosine diphosphate (ADP) and inorganic phosphate $\left(\mathrm{P}_{\mathrm{i}}\right)(101)$. The thin filament is composed of actin, troponin, and tropomyosin. Tropomyosin is pulled from an active myosin binding site on actin when calcium ( $\mathrm{Ca}^{2+}$ ) is released from the sarcoplasmic reticulum and binds to troponin, resulting in a conformational change of troponin (21). In the presence of ATP and $\mathrm{Ca}^{2+}$, the myosin-actin interaction will repeat in a cyclical manner (i.e., myosin binds to actin, pulls, releases, and reattaches) allowing the sarcomere to shorten. This process is commonplace in all mammalian muscle; however, the rate of cross-bridge cycling (i.e., shortening velocity) differs based on morphological discrepancies (e.g., MHC isoforms).

The spectrum of fiber type classifications was originally described based on appearance (101). Fast twitch fibers generally appear white, while slow twitch fibers appear red. The red color is due to large concentrations of myoglobin and capillaries, which contributes to a greater oxidative capacity (101). Furthermore, histochemical analysis of myosin ATPase led to the original division of muscle fibers into type I (slow) and type II (fast), and the velocity of muscle shortening was correlated with myosin ATPase activity (101). ATPase hydrolysis rates for fast fibers are 2 to 3 times greater than slow fibers (150). Additionally, myosin heavy chain isoforms ( $\mathrm{MHCl}, \mathrm{MHClla}$, and MHCIIb) can be assessed to identify fiber types, given that the myosin heavy chain contains the ATPase site (150). The evaluation of myosin ATPase and MHC isoforms has led to the identification
of 7 recognized muscle fiber types including types I, IC, IIC, IIAC, IIA, IIAB, and IIB (118). Therefore, proper classification of fiber types requires a comprehensive investigation of a fiber's constituents such as mitochondrial content, myoglobin and capillary content, myosin ATPase activity, and MHC isoforms.

## Phosphorylation of Myosin Regulatory Light Chains

On the muscular level, PAP has been theorized as the transient phosphorylation of the myosin RLC via myosin light chain kinase (MLCK), which increases $\mathrm{Ca}^{2+}$ sensitivity in striated muscle (75). During contractions, MLCK is activated due to an increase in calmodulin (regulatory protein that binds with $\mathrm{Ca}^{2+}$ and is thought to interact with target proteins in skeletal muscle (e.g., phosphorylase kinase, MLCK, protein kinase)), which accompanies an influx in $\mathrm{Ca}^{2+}$ (93). In theory, the actin-myosin interaction is more sensitive to $\mathrm{Ca}^{2+}$ released from the sarcoplasmic reticulum due to a conformational change of the RLC, exposing the myosin heavy chain and increasing the rate by which myosin cross-bridges move from a non-force producing state to a force producing state, thus enhancing force production during subsequent movements.

The phosphorylation of the myosin RLC has been commonly observed via an evoked muscle twitch, which is defined as a brief muscle contraction in response to a single presynaptic action potential or a single, synchronized volley of action potentials (90). An increase in the force of a twitch contraction has been observed following an evoked tetanic contraction (7-s stimulation of the ankle dorsiflexors at 100 Hz resulting in a $45 \%$ increase in twitch peak torque) (110), a sustained MVC (156), and repeated sub-fusion stimuli (96). Furthermore, these contractile conditioning techniques may increase the RFD in a twitch response and decrease its time-to-peak force (134). Twitch potentiation (TP), as this phenomenon is commonly known, has been readily observed and is reproducible $(5,58,110,156)$, but its optimization may be limited given certain components
regarding participants (e.g., sex, age, training status) and the conditioning activity (e.g., intensity, volume, rest periods).

A PAP response has been observed in both type I and type II fibers; although, athletes with a higher proportion of type II fibers generally have a greater level of potentiation due to a greater concentration of MLCK (measured by histochemical analysis of muscle biopsy sample) (149). Houston et al. (76) observed a significant positive correlation ( $R=0.85, p<0.05$ ) between individual increases in the twitch potentiation ratio (twitch tensions following voluntary contractions divided by baseline twitch tension) and increases in phosphate incorporation in the RLC of fast myosin (i.e., type II) 20s after a maximal voluntary contraction. Furthermore, a large effect (ES = 0.53) of loaded pre-conditioning on PAP has been reported among athletes who engage in intermittent highintensity sports or strength training (137), suggesting that athletes with more type I fibers (generally participating in endurance-based sports) may have a reduced PAP response. However, the shortening velocity of type I fibers is increased following endurance training, with a concomitant increase in MLCK concentration (136). This latter adaptation would increase the capacity of RLC phosphorylation, thus enhancing a PAP response.

## Motor Unit Recruitment

The size of cell bodies of recruited motor units corresponds to the intensity of contraction, commonly known as the "size principle" (72). As the intensity of contractions increase, motor unit recruitment increases from small to large. Thus, the recruitment of high-order motor units (i.e., fast motor units) requires MVCs. This mechanism has been observed in vivo through the reflex pathway by the Hoffmann reflex (H-reflex; Figure 1) method. The H-reflex method is an electrically induced reflex analogous to the mechanically induced spinal stretch reflex (83), which is a monosynaptic reflex that initiates muscle contraction following stimulation of local stretch receptors (19). The H-
reflex method provides an estimate of alpha motoneuron excitability by bypassing the muscle spindle. An electrical stimulus is elicited to la afferent fibers resulting in action potentials that travel toward alpha motoneurons $(5,83)$. Action potentials generated by alpha motoneurons travel along efferent fibers to neuromuscular junctions, producing a twitch response. If force enhancement following a pre-conditioning contraction is due to PAP, alpha motoneuron excitability would increase and lead to an increase in the H-reflex amplitude (recorded as an electromyography (EMG) response) (83). This may describe the increase in peak force and RFD as increasing activation of motor units may lead to increased mechanical output (75). For example, Gullich and Schmidtbleicher (65) measured H-reflex amplitude in the lateral gastrocnemius and soleus muscles before and after five repetitions of a 5-s maximal voluntary plantarflexion contractions with 1-min recovery between contractions. H-reflex amplitude was significantly increased in the lateral gastrocnemius $(+19 \%, p<0.05)$ of the trained speed-strength athletes compared to untrained participants. Similarly, Trimble and Harp (154) reported significant increases ( $p<0.01$ ) in H-reflex amplitude of the lateral gastrocnemius following eight sets of ten repetitions of plantarflexion contractions compared to baseline measures. In both of the aforementioned studies, potentiation occurred $10-60 \mathrm{~s}$ following the pre-conditioning activities, suggesting a delay in potentiation.


Figure 1. Hoffman reflex (H-reflex) and muscle response (m-wave) pathways (1).

## $\mathrm{VO}_{2}$ Kinetics

The utilization of oxygen by active tissues due to oxygen availability, enzyme activity, and/or availability of metabolic substrate (80), and the activation of processes associated with oxidative metabolism (108) are theorized mechanisms responsible for enhanced $\mathrm{VO}_{2}$ kinetics and endurance performance following high-intensity pre-conditioning. $\mathrm{VO}_{2}$ kinetics refers to the transitory period from rest to exercise where a coordinated pulmonary, cardiovascular, and muscular system respond to a rapid increase in the flux of oxygen from the atmosphere to muscle mitochondria resulting in aerobic ATP production (120). Faster $\mathrm{VO}_{2}$ kinetics during the transition to moderate-intensity exercise after heavy-intensity exercise has been associated with elevated baseline active form of pyruvate dehydrogenase (PDHa) activity (66). PDHa catalyzes the irreversible decarboxylation of pyruvate to acetyl-CoA and increased concentrations of PDHa increase the production of ATP via the tricarboxylic acid cycle and oxidative phosphorylation (116). Furthermore, a concomitant increase in the recruitment of higher-order motor units and $\mathrm{VO}_{2}$ kinetics has been demonstrated in recreationally active males following high-intensity pre-conditioning bouts of cycling (5,83). As exercise progresses, there may be a reduced need for further motor unit recruitment if higherthreshold motor units are recruited prior to exercise. This may be explained by the number of muscle fibers innervated by larger motor units, which share the imposed exercise load. Increases in integrated electromyography (iEMG) have provided evidence in support of this theory $(24,153)$.
iEMG enhancement of the vastus lateralis, vastus medialis, and gastrocnemius medialis has been observed during the second of two bouts of $30-$ s all-out sprint cycling (Wingate test) at the onset of exercise compared to the first bout ( $p<0.05$ ) (153) and during the second bout of intense exercise (24). Burnley et al. (24) reported a relationship between increased iEMG and enhanced $\mathrm{VO}_{2}$ kinetics during two bouts of heavy cycle exercise (70\% of the difference between the lactate threshold and $\left.\mathrm{VO}_{2 \text { peak }}\right)$. This relationship was demonstrated by no significant difference ( $p=0.45$ )
between the primary $\mathrm{VO}_{2}$ amplitude/normalized iEMG ratio of the first bouts compared to the second, indicating that $\mathrm{VO}_{2}$ increased concurrently with EMG activity. To support the utility of using pre-conditioning to increase $\mathrm{VO}_{2}$ kinetics and improve middle-distance running performance, Ingham et al (78) investigated the effect of warming up with an high-intensity exercise (200-m run at race pace) prior to an $800-\mathrm{m}$ TT. A faster time-to-completion ( $124.5 \pm 8.3 \mathrm{~s}$ vs $125.7 \pm 8.7 \mathrm{~s}$ ) was observed following the 200-m run compared to control condition ( $6 \times 50-\mathrm{m}$ strides). Additionally, faster $\mathrm{VO}_{2}$ kinetics were observed (as measured by the response time for $\mathrm{VO}_{2}$ to reach steady-state) following 200-m runs compared to the control condition ( $27 \pm 6 \mathrm{~s}$ vs $28 \pm 7 \mathrm{~s}$ ). This evidence indicates that a high-intensity component within a warm-up may improve middle-distance running performance.

## Lactate Threshold, the Onset of Blood Lactate Accumulation, and Ventilatory Thresholds

Sports performance, particularly endurance performance, may be predicated on several physiological params, namely lactate threshold (LT), the onset of blood lactate accumulation (OBLA), and ventilatory thresholds $1\left(\mathrm{VT}_{1}\right)$ and $2\left(\mathrm{VT}_{2}\right)$. These physiological parameters are usually evaluated using a graded exercise test (GXT) and are helpful in determining aerobic endurance performance capacity.

Briefly, lactate is derived from the reduction of pyruvate by NADH following glycolysis (i.e., catabolism of glucose) and is catalyzed by the enzyme lactate dehydrogenase (LDH) (124). Lactate is considered one the most abundant circulating carbon carriers in mammals (along with glucose) and can readily be used as a fuel source at rest and during exercise $(124,130)$. During exercise, lactate can be directly transported into skeletal muscle cells via a monocarboxylate transporter (MCT) and converted to pyruvate via PDH and undergo subsequent oxidization by skeletal muscle mitochondria (124). Additionally, lactate can be converted into glucose in the liver via the Cori cycle (124).

Endurance-trained individuals have an increased ability to utilize lactate to generate ATP during exercise due to increased MCT, LDH, PDH, and mitochondrial content (42). Traditionally, the conversion of pyruvate to lactate is thought to release a proton $\left(\mathrm{H}^{+}\right)$and contribute to a decrease in $\mathrm{pH}(29,53)$, which lead researchers to believe that lactate was the cause of metabolic acidosis. However, recent evidence suggests that lactate acts as a proton buffer, and an increase in lactate is in response to increases in $\mathrm{H}^{+}$from glycolysis and ATP hydrolysis (130). Thus, the rise in lactate with increasing exercise intensity provides an indication of an individual's capacity to buffer $\mathrm{H}^{+}$and prolong exercise. To date, two levels of blood lactate concentration (i.e., LT and OBLA) have been utilized in assessing endurance performance.

In regard to LT, many concepts have been developed and there is ongoing debate about a clear threshold in which blood lactate remains stable during prolong exercise (i.e., steady state). The most commonly used definition of LT is a $>1 \mathrm{mmol} \cdot \mathrm{L}^{-1}$ increase in blood lactate concentration from baseline levels with increasing workload (98). Typically, an exponential rise in blood lactate is observed during a GXT, and a rightward shift of this curve is considered greater endurance capacity (generally following a period of endurance training) $(2,162)$. Studies evaluating the LT report a wide range of blood lactate concentrations. For example, during a incremental rowing ergometer test, Stegmann and Kindermann (147) measured blood lactate of rowing athletes, using a portable lactate analyzer, and observed a mean lactate concentration of $2.3 \mathrm{mmol} \cdot \mathrm{L}^{-1}$ at LT. Chwalbinska et al. (36) observed a mean blood lactate concentration of $2.9 \mathrm{mmol} \cdot \mathrm{L}^{-1}$ at LT in endurance-trained male college students during an incremental cycle ergometer exercise test. As exercise workload and duration progress, a s lactate threshold is achieved, namely OBLA. OBLA is defined as a blood lactate concentration of $4 \mathrm{mmol} \cdot \mathrm{L}^{-1}(142)$. A lactate concentration of $4 \mathrm{mmol} \cdot \mathrm{L}^{-1}$ is thought to be the upper boundary of exercise intensities eliciting constant arterial lactate concentration (i.e., maximal lactate steady state) during endurance performance (142). In the aforementioned study, Stegmann and

Kindermann (147) observed a mean lactate concentration of $4.0 \pm 1.6 \mathrm{mmol} \cdot \mathrm{L}^{-1}$ at the end of a prolonged 50 min exercise bout at a predetermined workload corresponding with OBLA (4 mmol $\cdot \mathrm{L}^{-}$ ${ }^{1}$ ). Rowers that performed the prolonged bout at a workload just above OBLA ( $\left.\sim 4.1 \mathrm{mmol} \cdot \mathrm{L}^{-1}\right)$ demonstrated a mean lactate value of $9.6 \pm 1.2 \mathrm{mmol} \cdot \mathrm{L}^{-1}$ at the end of the exercise bout, indicating that the workload at OBLA may be a maximal workload in which lactate remains in a steady-state (147).

An increase in lactate production is accompanied by an increase in carbon dioxide $\left(\mathrm{CO}_{2}\right)$, produced through the buffering of protons $\left(\mathrm{H}^{+}\right)$by bicarbonate (13). This excess $\mathrm{CO}_{2}$ (i.e., nonmetabolic $\mathrm{CO}_{2}$ ) stimulates an increase in ventilation to offset accumulating $\mathrm{CO}_{2}$ and avoid increasing acidity (13), known as a ventilatory threshold. Two ventilatory thresholds have been determined, which coincide with LT and OBLA. $\mathrm{VT}_{1}$ occurs immediately following LT and can be identified as a concomitant increase in ventilatory equivalent of oxygen $\left(\mathrm{V}_{\mathrm{E}} / \mathrm{VO}_{2}\right)$ and end-tidal pressure of oxygen $\left(\mathrm{P}_{\mathrm{ET}} \mathrm{O}_{2}\right)$ with no concomitant increase in ventilatory equivalent of carbon dioxide $\left(\mathrm{V}_{\mathrm{E}} / \mathrm{VCO}_{2}\right)(115) . \mathrm{VT}_{2}$ occurs immediately following OBLA and can be identified as a concomitant increase in $\mathrm{V}_{\mathrm{E}} / \mathrm{VO}_{2}$ and $\mathrm{V}_{\mathrm{E}} / \mathrm{VCO}_{2}$ and a decrease in end-tidal pressure of carbon dioxide $\left(\mathrm{P}_{\mathrm{ET}} \mathrm{CO}_{2}\right)(115)$. A close relationship between lactate and ventilation parameters has been previously reported (20). Davis et al. (40) investigated the validity of LT detection through nonlinear increases in ventilation and $\mathrm{CO}_{2}$ production and abrupt increase in $\mathrm{P}_{\mathrm{ET}} \mathrm{O}_{2}$ and observed no significant difference between the estimation of LT from gas exchange parameters ( $59.7 \pm 7.1 \% \mathrm{VO}_{2 \max }$ ) and blood lactate concentrations ( $59.8 \pm 7.4 \% \mathrm{VO}_{2 \max }$ ). Furthermore, Caiozzo et al. (31) examined the relationship between gas exchange and blood lactate for the determination of OBLA and observed a correlation of 0.95 between the two parameters. Thus, gas exchange may be an effective noninvasive method to determine blood lactate thresholds and metabolic dynamics during exercise tests.

## Stiffness

The ability of a body, limb, or joint to resist applied force during human locomotion is regarded as stiffness (23). Increased musculotendinous stiffness may reduce the energetic cost of movement due to the storage and return of elastic strain energy (i.e., absorbed and reutilized mechanical energy by musculotendinous units) (49). Roberts et al. (131) observed that mechanical work during level running was produced from the stretch and recoil of tendon and muscle springs, measured with implanted sonomicrometer crystals, of the lateral gastrocnemius. Musculotendinous stiffness and the metabolic cost of running have been significantly correlated ( $R=-0.69, p=0.01$ ) during a graded exercise test to volitional exhaustion (43). Increases in plantar flexor tendon stiffness, measured using ultrasonography and dynamometry, have been observed following heavy resistance training, which were also correlated ( $R=-0.723, p<0.005$ ) with improvements in the metabolic cost of running during three submaximal treadmill velocities (75\%, 85\%, and 95\% of lactate threshold) (48). The elastic strain energy provision from the ankle-plantar flexor unit increases with increasing running speeds on a flat surface (89). Thus, runners with stiffer muscletendon units may show enhanced performance by utilizing elastic strain energy and conserving metabolic energy.

Stiffness in the human body varies, thus stiffness (i.e., vertical, leg, and joint stiffness) needs to be operationally defined. Vertical stiffness is a measure of resistance of the body to vertical displacement after application of ground reaction force, and is commonly used as a reference stiffness measure for the development of leg and joint stiffness models (22). This can be represented mathematically as:

$$
k_{\text {vert }}=F_{\max } / \Delta y
$$

where $k_{\text {vert }}$ is vertical stiffness, $F_{\text {max }}$ is maximum ground reaction force, and $\Delta y$ is displacement of center of mass $(6,10)$. Force platforms are commonly used to measure $F_{\max }$, and double integration
of vertical acceleration is used to calculate $\Delta y$ (139). Furthermore, $\mathrm{k}_{\text {vert }}$ can be determined with the use of pressure sensors or accelerometers, while modelling $\Delta y$ with ground contact time and flight time $(55,74)$.

The measurement of leg stiffness seems to be misconstrued, as researchers often conflate leg stiffness and vertical stiffness. Leg stiffness is defined as resistance to change in leg length after application of internal and external forces (22). The most commonly used model to calculate leg stiffness is the quotient of $F_{\text {max }}$ and $\Delta y$, which is a measure of body stiffness as previously mentioned (106). Indeed, leg stiffness is more accurately represented as the quotient of change in leg length and $\mathrm{F}_{\text {max }}$ :

$$
\mathrm{k}_{\text {leg }}=\mathrm{F}_{\max } / \Delta \mathrm{L}
$$

where $\mathrm{k}_{\text {leg }}$ is leg stiffness, and $\Delta L$ is change in leg length (139). Change in leg length can be measured using a three-dimensional motion capture system (64), where the change in leg length is considered the displacement of the hip joint center relative to foot contact. Several variations of measurement are used to assess leg length including hip joint center relative to a marker on the ball of the foot during ground contact (64) and point of force application between foot contact and when ground reaction force reaches maximum (145), and vertical excursion of the hip joint center relative to the ground (127).

The leg is commonly modeled using a spring-mass model (Figure 2), which is ideal for calculating vertical stiffness when the leg is oriented vertically (30). However, the leg contacts the ground at an angle, moving from a non-vertical position to a vertical position during running (30). Thus, a model that considers the runner's horizontal velocity, time of contact, and the resting leg length, as well as peak vertical ground reaction force is used to calculate stiffness during running (30). Latash and Zatsiorsky (91) suggest that an accurate model must account for components such
as tendons, ligaments, muscles, cartilage, and bone, as well as viscosity, muscle reflex time delays, and central nervous system control, among other components. However, a model that considers all the previously stated components is complicated and mathematical expressions have yet to be developed (30). Therefore, the spring-mass model (Figure 3) incorporating a non-vertical contact is frequently used to estimate leg stiffness during running. By accounting for run speed, the model in Figure 3 accounts for attack angle (i.e., leg contacting ground in a non-vertical position) of the leading leg, with a more vertical leg at footstrike requiring greater leg stiffness whereas a more outstretched leg will require lower leg stiffness.


Figure 2. Spring-mass model used for calculating vertical stiffness when the leg is oriented vertically.


Figure 3. Spring-mass model used for calculating stiffness when the leg contacts the ground in a non-vertical position.

Joint stiffness, specifically knee joint stiffness, has been measured as the quotient of peak knee extension moment and change in sagittal knee joint angle (4,7). Knee joint stiffness can be mathematically represented as

$$
\mathrm{K}_{\text {knee }}=\mathrm{M} / \Delta \theta
$$

where $K_{\text {knee }}$ is knee joint stiffness, $M$ is peak knee extension moment, and $\Delta \theta$ is change in knee flexion angle (i.e., knee flexion excursion) (139).

Tendons bear the same tension as muscles when tendons lie in series with muscle fibers, and a stretch is distributed between muscle fibers and tendon when applied to a muscle (122). An initial steep and linear rise in tension has been observed in a muscle fiber in response to stretch, termed short-range stiffness, followed by a gradual increase or even decrease in tension if stretch continues (125). Short-range stiffness may be due to strain of cross-bridge formation between actin and myosin filaments; thus, beyond short-range stiffness, tension change depends on the level of activation of the muscle and on the amount of passive stiffness (81). Therefore, prior to muscle fibers exceeding short-range stiffness, tendons, if attached to muscle fibers, can absorb a large portion of the movement (105). Even at higher forces, when muscle fiber stiffness is elevated, much of the strain is transmitted to the tendon (122). Increased tendon stretch may enhance the capacity for elastic energy storage and provision during running and constrain total energy expenditure.

Conflicting evidence exists demonstrating an acute increase in musculotendinous stiffness following a loaded pre-conditioning activity. Gago et al. (51) observed significant potentiation in the plantar flexors of trained track and field athletes immediately following a 6-s MVC. Peak twitch (60.6 $\pm 19.3 \%, \mathrm{p}<0.05)$ and RFD ( $90.1 \pm 22.5 \%, \mathrm{p}<0.05$ ) were significantly enhanced, while rising time ( $10.1 \pm 7.7 \%, \mathrm{p}<0.05$ ) and half relaxation time ( $18.7 \pm 5.6 \%, \mathrm{p}<0.05$ ) were significantly reduced from supramaximal evoked twitches; however, Achilles tendon stiffness remained unchanged. The
results from this study suggest that contractile properties of the plantar flexors are influenced by loaded pre-conditioning without affecting tendon properties. A systematic review revealed an immediate decrease in Achilles tendon stiffness of the triceps surae complex following non-stretchshortening cycle (SSC) exercise modes (e.g., isometric contractions) (112); however, recreational runners who performed a single 30 -min bout of running on a treadmill at 12 kmph had no change in Achilles tendon stiffness during a subsequent hoping task (two sets of six one-legged hops) at a selfpaced cadence (46). Thus, individuals with more fatigue-resistant Achilles tendon engage in SSC activities compared to individuals participating in non-SSC activities. Middle- and long-distance modalities that rely on musculotendinous stiffness (e.g., running) may benefit from pre-conditioning activities due to its positive influence on muscular properties with unaffected tendon stiffness. It seems that pre-conditioning may acutely affect muscular properties such as motor unit recruitment, actin-myosin interaction, increased blood flow, etc. within the lower extremity, with no effect on tendons as the aforementioned muscular properties are not present in tendinous tissue. Furthermore, leg and vertical stiffness measurements encompass a plethora of anatomical components compared to tendon stiffness.

Morphological changes in other musculotendinous units (e.g., hamstring and quadriceps complexes), rather than those within the triceps surae complex, may alter limb or vertical stiffness. Vertical stiffness was significantly improved ( $+10.9 \%, \mathrm{p}<0.05$ ) in elite rugby athletes during 3 drop jumps following a back squat resistive load of $93 \%$ of a 1 repetition maximum (1RM) compared to a baseline measurement (38). Similarly, Mori et al. (103) observed significant increases vertical stiffness ( $4.93 \pm 1.60 \mathrm{kN} \cdot \mathrm{m}^{-1}$ vs $4.24 \pm 1.07 \mathrm{kN} \cdot \mathrm{m}^{-1}, \mathrm{p}=0.01$ ) in female volleyball players following 3 repetitions of back squats at $90 \% 1 \mathrm{RM}$ compared to baseline measurements. The results from Mori et al. indicate that loaded pre-conditioning acutely enhances vertical stiffness during activities that involve a SSC, which may be particularly beneficial for distance runners given evidence that
demonstrates a decrease in leg stiffness with fatigue $(52,70)$. Acute increases in leg stiffness at the onset of exercise may ameliorate this reduction in leg stiffness toward the latter stages of middleand distance- running performance, thus increasing the capacity for energy conservation by relying more on elastic strain energy.

## Loaded Pre-Conditioning Prior to Power-based Performance

Prior to recent studies exploring the utility of loaded pre-conditioning warm-ups on endurance-based performance $(12,47)$, loaded pre-conditioning warm-ups were first applied to power-based performance exercises and sports (75). Evidence demonstrating the effect of loaded pre-conditioning on ballistic, intermittent athletic performance is broad $(65,137,155)$, and warrants a closer investigation to support the use of loaded pre-conditioning in endurance-based sports. Young et al. (163) using strength-trained athletes, showed a significant increased jump height of $2.8 \%$ in loaded ( 19 kg ) counter movement jumps after performing 1 set of 5 repetitions of 5 RM half squats, using a recovery time of 4 min , compared to a control condition (no half squats). The authors suggested that a set of heavy resistance exercises results in a degree of heightened neural stimulation, measured using EMG, and results in an improved performance on a subsequent plyometric or maximal power movement (163). Robins (129) found that performance on a s set of countermovement jumps preceded by 7 ss of maximal voluntary isometric contraction (MVIC) squats, resulted in a $17.2 \%$ increase in jump height over a first set not preceded by a set of MVIC squats, although no statistical evidence was given to support this data. Similarly, Radcliffe \& Radcliffe (126) found improved countermovement jump power output ( $244 \pm 5.2 \mathrm{~W}$ vs $241 \pm 4.1 \mathrm{~W}$, $p<0.05)$ when preceded by four sets of four power snatch at $75-85 \% 4 R M$ compared to four sets of four loaded jumps with $15-20 \%$ body weight. Chiu et al., (88) in establishing the effects of PAP on jump squats, utilized 5 sets of 1 repetition at $90 \% 1 R M$ back squat to stimulate PAP and
demonstrated significant increases in peak power, but only in strength-trained athletes compared to non-strength-trained athletes $(p=0.006)$.

Gourgallis et al. (59) demonstrated a mean improvement of $2.39 \%$ on vertical jump height following 5 sets of half squats for 2 repetitions at $20 \%, 40 \%, 60 \%, 80 \%$, and $90 \% 1$ RM compared to pre-warm-up jump height. The authors also indicated that subjects with greater maximal strength levels in the squat experienced greater jump height compared to those with lower maximal strength (4.01\% vs $0.42 \%$ ) (60). Rixon et al. (128) found that PAP, measured by countermovement jump performance, could be induced with isometric squats (3 reps of 3 ss of MVICs in a squat against a fixed resistance with two min recovery between repetitions; $p<0.001$ ), but not with dynamic squats (1 set of 3 repetitions at 90\% 1RM on a Cybex Smith machine). Gilbert et al. (54) demonstrated no increase in maximum voluntary contractions but a significant increase in RFD (on an isometric leg press at 90-degree leg angle, with data measured by a strain gauge linked to an analogue to digital converter) ( $p=0.016$ ), after performance of 5 single repetitions of maximal squat exercises of five single repetitions at $100 \% 1$ RM. This increase in RFD peaked at 20 min post squat ( 54 ) and supports the work of Sale (134) who suggests that PAP will have its greatest effects on RFD, with peak force remaining unchanged.

McBride et al. (100) investigating the effect of PAP on sprinting speed, found that athletes ran $0.87 \%$ faster in a 40 -yard dash when preceded by a set of heavy squats (1 set of 3 repetitions at $90 \%$ of 1 RM) compared to a control (no squats prior to sprinting). Interestingly, no statistical difference was seen in the split times at 10 or $30 \mathrm{~m}(100)$, suggesting that PAP has greatest effects at the highest running speeds. Chatzopoulis et al. (34) investigating the effects of PAP on sprinting speed, found no change in performance over 10 m and 30 m of sprinting following 10 sets of single squats at $90 \%$ 1RM, after 3 min recovery, but a significant improvement was observed after 5 min of recovery for both $10 \mathrm{~m}(1.91 \pm 0.03 \mathrm{~s}$ vs $1.84 \pm 0.02 \mathrm{~s}, \mathrm{p}<0.05)$ and $30 \mathrm{~m}(4.54 \pm 0.08 \mathrm{~s}$ vs $4.43 \pm 0.06$
$\mathrm{s}, \mathrm{p}<0.05$ ) of sprinting. The authors suggested a longer recovery period resulted in diminished fatigue and greater potentiation (34). Yetter et al. (161) found significant improvements in the 10-$20-\mathrm{m}$ section of a 40 -yard sprint following $30 \%, 40 \%$, and $50 \%$ of 1 RM back squats (mean difference $=0.12 \mathrm{~m} \cdot \mathrm{~s}^{-1}, \mathrm{p}=0.001$ ), but not $30 \%, 40 \%$, and $50 \%$ of 1 RM front squats compared to a control condition (no squats), suggesting that there may be differences in the potentiation potential of differing exercises, and that potentiation can affect different aspects of speed performance to varying degrees (e.g., different stages of a 40-yard sprint). Smith et al. (144) also postulated that PAP may be able to increase sprinting speed, following their findings of enhanced $10-\mathrm{s}$ sprint cycling performance ( $p=0.006$ ) following $10 \times 1$ repetition at $90 \%$ of $1 R M$ back squats compared to no squats preceding the cycling performance.

## Pre-Conditioning Activity in Endurance Sports

The incorporation of a 3-6-min high intensity exercise bout ( $60-85 \%$ of peak power output) is suggested to sufficiently increase subsequent endurance performance (11). This may be due to an acceleration in the $\mathrm{VO}_{2}$ response at the onset of exercise or during the transition from low-intensity to moderate- or high-intensity exercise $(11,66)$. Near-maximal sprints ( $5 \times 10$ s at $200 \% \mathrm{VO}_{2 \max }$ ) prior to a performance assessment have been shown to significantly increase peak ( $629 \pm 199 \mathrm{~W}$ vs $601 \pm$ $204 \mathrm{~W}, \mathrm{p}<0.05$ ) and average ( $328 \pm 39.0 \mathrm{~W}$ vs $321 \pm 42.4 \mathrm{~W}, \mathrm{p}<0.05$ ) power during a $2-\mathrm{min}$, maximal kayak performance on an ergometer compared to a low-intensity continuous warm-up (15 $\min$ at $65 \% \mathrm{VO}_{2 \max }$ ) (16). Conversely, a similar protocol ( $5 \times 10 \mathrm{~s}$ cycling bouts of $100 \%$ and $150 \%$ of peak aerobic power, and all-out priming) had no significant effect on 3km cycling TT performance (p $>0.05$ ), and actually decreased performance ( $-5.8 \%$ ) if the sprints were performed maximally (102). Although a similar pre-conditioning protocol was used in these two studies, the discrepancy in results may be a consequence of the different performance assessments; thus, this evidence should not be directly compared.

Intermittent high-intensity bouts $(27,78)$ and single-sprint approaches $(25,78)$ have also significantly improved endurance performance. A single 200 m sprint at race pace performed 20 min before an 800 m TT significantly decreased time-to-completion (124.5 $\pm 8.3 \mathrm{~s}$ vs $125.7 \pm 8.7 \mathrm{~s}, \mathrm{p}<$ 0.05) compared to a traditional warm-up ( $6 \times 50 \mathrm{~m}$ and $2 \times 50 \mathrm{~m}$ strides) used by middle-distance runners (78). Furthermore, inter-repetition recovery duration may be implicated in performance decrements. Five min between pre-conditioning repetitions ( $3 \times 30 \mathrm{~s}$ maximal sprints) and a rest period of 15 min prior to the onset of an exhaustive cycling exercise corresponding to $105 \%$ of the predetermined $\mathrm{VO}_{2 \text { peak }}$ were shown to significantly increase the amplitude of the primary $\mathrm{VO}_{2}$ component (i.e., oxygen uptake from the onset of exercise to steady-state) $\left(9.0 \pm 0.7 \mathrm{ml} \cdot \mathrm{kg}^{-1} \cdot \mathrm{~min}^{-1}\right.$ vs $8.1 \pm 0.9 \mathrm{ml} \cdot \mathrm{kg}^{-1} \cdot \mathrm{~min}^{-1}, \mathrm{p}<0.05$ ) compared to a control with no pre-conditioning (158). These findings indicate that there may be an enhanced ability of working muscles to utilize oxygen at the onset of exercise due to an increase in blood flow and oxidative enzymatic activity. This creates potential for attaining a higher peak $\mathrm{VO}_{2}$ as this may be limited by $\mathrm{O}_{2}$ availability among other parameters. It seems apparent from these data that bouts of high-intensity pre-conditioning activities positively influence $\mathrm{VO}_{2}$ kinetics and endurance performance.

## Loaded Pre-Conditioning Prior to Endurance Performance

Evidence demonstrating the effect of loaded pre-conditioning on ballistic, intermittent athletic performance is broad $(65,137,155)$. Given the theory that acute muscle potentiation should result from a loaded pre-conditioning stimulus if it is greater than the stimulus produced during the subsequent performance, a PAP response may have a greater effect on sub-maximal performance (i.e., middle- and long-distance sports) compared to maximal and supra-maximal activities (e.g., sprinting, jumping, throwing).

To date, there have been 5 studies investigating the effects of pre-conditioning resistance exercises on middle- and long-distance athletes, in which 3 studies included a performance measure (e.g., time trial (TT)). Feros et al. (47) showed a significant increase in mean power output (+6.6\%, p $<0.01$ ) and stroke rate ( $+5.2 \%, \mathrm{p}<0.01$ ), and a decrease in time-to-completion ( $-1.9 \%, \mathrm{p}<0.01$ ) over the first 500 m of a 1000-m rowing ergometer TT in national level rowers following an individualized warm-up including $5 \times 5$ isometric contractions (i.e., pulling an immovable handle on a rowing ergometer). Additionally, a non-significant reduction in 1000-m rowing time of 1.4 ss (-0.8\%) following pre-conditioning isometric contractions was reported compared to a rowing warm-up with no loaded activity. This study suggests an effect of pre-conditioning during the first half of the TT; however, this study may be limited by the individualized warm-up. Additionally, a $6 \%$ time reduction has been observed in trained cyclists during a 20 km cycling TT following a 5-min submaximal warmup with 4 sets of 5RM leg press compared to a standard 5-min low-intensity cycling warm-up (141). It was also reported that the loaded pre-conditioning treatment increased mean power output ( $p=$ 0.06 ) during the first $10 \%$ of the $T T(253.1 \pm 69.4 \mathrm{~W}$ vs $239.3 \pm 61.4 \mathrm{~W})$. Although not measured in these studies, RLC phosphorylation may have contributed to the increase in power output at the onset of the TTs, and the overall reduction in time-to-completion influenced by a combination of other pre-conditioning mechanisms (e.g., motor unit recruitment, enhanced $\mathrm{VO}_{2}$ kinetics).

Barnes and colleagues (12) determined that $6 \times 10$ s weighted-vest pre-conditioning strides improved running economy (i.e., reduced oxygen consumption) during 5 min of sub-maximal treadmill running. A large improvement in the metabolic cost of running (-6.0\%) was observed following weighted-vest strides compared to a bodyweight control condition. Furthermore, a moderate improvement in vertical stiffness was observed (post: $12.5 \pm 2.7 \mathrm{kN} \cdot \mathrm{m}^{-1} \mathrm{vs}$ pre: $10.3 \pm 2.1$ $\mathrm{kN} \cdot \mathrm{m}^{-1}$ ) during a vertical jump test. However, it cannot be inferred that leg stiffness increased during running since it was not determined during the running trials, and leg stiffness was conflated with
vertical stiffness given the stiffness assessment was a vertical jump test. O'Neal et al. (111) also investigated weighted-vest strides prior to a 5-k running TT. Participants were asked to perform four $80-\mathrm{m}$ strides with a $6.8-\mathrm{kg}$ load ( $10.4 \pm 1.1 \%$ of body mass) prior to a $5-\mathrm{k}$ TT on a cross-country running course. The researchers observed no difference in time-to-completion between the unloaded and loaded conditions ( $17.59 \pm 0.92 \mathrm{~min}$ vs $17.78 \pm 0.81 \mathrm{~min}$ ). It seems likely that the loaded condition did not provide an adequate stimulus to match or surpass the force demand of the 5 km TT. Additionally, the 10-min rest combined with 5 km time-to-completion may have exceeded optimal potentiation and PAP may have diminished.

Finally, Chorley and Lamb (35) investigated the effects of loaded cycling sprints (70\% peak power output) on 4 km TT cycling performance. Completion times, mean power output, and mean peak force did not significantly improve following the loaded pre-conditioning activity. Although, a significant increase in the rate of adjustment to oxidative metabolism was observed during the beginning stages of the TT, suggesting that there may be a transient potentiating effect of loaded pre-conditioning. An acute improvement in the metabolic cost of running may be greatly influenced by a temporary increase in leg stiffness, which may not have the same effect on cycling efficiency. Kinetic and potential energy are stored transiently as strain energy in the tendons and muscles that are stretched in response to the ground reaction forces during running (140). The stored energy is converted back to kinetic and potential energy as the foot leaves the ground, conserving metabolic energy (140). Strain energy may be minimal if not absent during cycling due to low ground reaction forces and a lack of SSC (8). Thus, the acute increase in leg stiffness following loaded preconditioning may have a greater effect on metabolic energy expenditure during running compared to cycling.

## Pre-Conditioning Modulating Factors

To optimize the PAP response, there are specific factors to consider which include participant characteristics, the pre-conditioning activity task, and recovery following the preconditioning activity. As previously mentioned, the incorporation of high-intensity bouts (approximately 3-6 min at 60-85\% PPO) during a low-intensity warm-up may adequately prime the muscles for subsequent endurance performances (11). At least 5-10 min following this aerobic warm-up component, a loaded pre-conditioning exercise may actuate a PAP response and ameliorate subsequent performance (83); however, this may only occur under certain conditions.

## Variability in Participant Characteristics

Individuals that present with type II fiber predominance may show enhanced PAP compared to those with more type I fibers, as previously discussed (67). Due to the physiological requirements of endurance sports (i.e., large aerobic capacities), individuals with a large proportion of type I fibers commonly participate in these events. This is particularly true in non-elastic recoil modalities such as cycling and rowing; however, athletes participating in sports with an elastic recoil component (e.g., running) may benefit from a warm-up that includes loaded pre-conditioning. Furthermore, research suggests that well-trained middle-distance runners have a higher proportion of type II fibers compared to moderately-trained runners $(77,117)$, and improved running economy (i.e., lower oxygen consumption at a given running velocity) is correlated ( $R=-0.96, p<0.001$ ) with type Ilx and Ila isoforms (117).

Strength-trained individuals have demonstrated significantly enhanced PAP compared to individuals with little or no strength training experience, which may be due physiological adaptations such as an increased proportion of type II fibers and higher threshold motor unit recruitment (137). Strength training has become commonplace in endurance sports over the past
decade. Well-trained swimmers, rowers, and cyclists that have performed strength training regimes have demonstrated enhanced performance $(9,92,132)$. Rowers and cyclists with strength training experience have acutely improved TT performance following a loaded pre-conditioning activity $(47,141)$. Additionally, well-trained distance runners that regularly perform strength training demonstrated significantly improved running economy following a loaded pre-conditioning activity (12). Furthermore, of equal importance to strength training may be overall training status. The training status of the active muscles in a given sport seems to have a better response to a loaded pre-conditioning activity compared to untrained muscles $(67,104)$. A proposed theory is better intermuscular coordination on a task, likely attributed to the activation of higher threshold motor units (14).

## Loaded Pre-conditioning Activity

The intensity of the loaded pre-conditioning activity may have a pronounced effect on a PAP response. It has been reported that high-load resistance exercises and plyometrics have similar effects (ES $=0.47$ and 0.41, respectively) on the PAP response (137). A small effect ( $E S=<0.2$ ) has been observed following moderate-load (65\%-85\% 1RM) resistance exercises and isometric contractions (137). The aforementioned study conducted by Silva and colleagues (141) observed a moderate increase ( $\mathrm{ES}=0.38$ ) in 20 k cycling performance following a 5RM leg press. Barnes et al. (12) observed a large improvement in the running economy $(-6.0 \%, E S=1.4)$ following $6 \times 10$ s strides with a weighted vest (20\% body mass). Additionally, a small and moderate improvement in peak running speed $(2.9 \%, \mathrm{ES}=0.35)$ and $\% \mathrm{VO}_{2 \max }(-7.2 \%, \mathrm{ES}=0.68)$, respectively, was observed. Only one study to date has investigated the acute effects of plyometric exercises on middle-distance runners (17). Blagrove et al. (17) observed moderate improvements in running economy (-3.7\%, ES = 0.67 ) following six depth jumps from a box equivalent to their best CMJ compared to a control
condition involving bodyweight quarter squats. A trivial effect on time-to-exhaustion was observed during a treadmill run at a running speed eliciting $\mathrm{VO}_{2 \text { max }}$.

Furthermore, multiple sets and few repetitions of a loaded pre-conditioning activity may optimize a PAP response $(137,159)$, which may be mediated by the strength level of the athlete, as described previously. Conversely, a meta-analysis conducted by Seitz et al. (137) concluded that plyometric pre-conditioning activities and traditional high-intensity induced moderate PAP effects (ES $=0.47$ and 0.41 , respectively), and moderate-intensity pre-conditioning activities produced a small PAP effect ( $E S=0.19$ ). Maximal isometric stimuli resulted in a negative effect $(E S=-0.09)$. However, this analysis included research studies investigating the effects of pre-conditioning activities on subsequent jump, horizontal jump, sprint, throw, and upper-body ballistic performances rather than endurance performances. The results from the previous meta-analysis seem to indicate that potentiation responses may differ between power and endurance athletes, which may be due to an enhanced fatigue resistance of endurance-trained athletes.

## Recovery Time Between Pre-Conditioning and Subsequent Performance

Optimizing the PAP response requires consideration of the time between the preconditioning activity and subsequent exercise and further consideration should be given to the structure of the recovery period (e.g., passive or active recovery). There must be adequate time for fatigue to dissipate yet capitalize on the relatively brief period of potentiation (i.e., fatigue/potentiation ratio). A high fatigue/potentiation ratio would indicate increased residual fatigue during the subsequent performance activity, negating potentiation, while a low fatigue/potentiation ratio would indicate diminished fatigue during the performance activity and increased potentiation possible lending to improved performance. The period of acute potentiation has been identified as approximately a 30-min window, with peak potentiation occurring at 12 to 15
$\min (83,104,159)$. Short duration tasks seem to require 5 to 12 min of recovery following heavy resistance exercises $(61,137,159)$ and 1 to 6 min following ballistic exercises (97). PAP temporal profile may be altered by individual's training status (e.g., strength-trained vs. non-strength-trained). Stronger individuals may optimize PAP in a shorter recovery period than weaker individuals, and aerobic fitness may enhance recovery from high-intensity pre-conditioning compared to anaerobic fitness (152). A passive recovery of 4 to 5 min following a loaded pre-conditioning activity has been shown to enhance performance at the beginning stages of middle-distance events (47); however, there was no improvement in overall performance, suggesting that residual fatigue may be present and have a negative effect. The aforementioned study conducted by Barnes et al. (12) used a passive recovery period of 10 min following the pre-conditioning activity, which yielded significantly positive results. An adequate recovery period may be needed to allow for phosphocreatine resynthesis and buffer low pH levels in active muscles (157); however, further investigations are needed for confirmation. Therefore, it seems that a passive recovery period between 5 and 10 min may be sufficient to diminish fatigue and maximize the PAP response for middle- and long-distance performance.

## Performance Activity Following Pre-Conditioning

PAP responses persist longer in aerobic-trained individuals compared to anaerobic-trained individuals $(104,113)$. PAP may benefit events lasting $<3 \mathrm{~min}$ compared with longer events ( $>5 \mathrm{~min}$ ). Research has reported significant improvements over the first half of performance during endurance events following pre-conditioning, as well as non-significant overall improvements $(16,69,78)$. However, none of these studies have incorporated an endurance running performance; thus, PAP may persist for the entire duration of distance running performance.

An endurance performance measure that has commonly been utilized to evaluate the effects of PAP is a TT which may provide the most direct form of applicability. Movement economy and efficiency (e.g., running economy and cycling efficiency) have also been used as assessment measurements following pre-conditioning activities. Although, economy and efficiency assessment outcomes have been conflicting in PAP studies $(12,141)$. Barnes et al. (12) reported improved running economy following weighted-vest strides, while Silva et al. (141) did not observe improvements in cycling efficiency (i.e., decreased $\mathrm{VO}_{2}$ during a 20 km TT ) following a 5 RM leg press. This difference may be due to the intensity of the pre-conditioning activity or the intensity of the performance measure. Furthermore, the elastic nature of running may ameliorate the responsiveness to pre-conditioning activities.

## Summary

Warm-ups are commonplace among middle- and long-distance athletes, with the general goal of enhancing oxygen uptake kinetics. Middle- and long-distance events require large aerobic capacities, thus aerobic priming seems to be efficacious in ameliorating subsequent performance. It has been speculated that a PAP response evoked by a loaded or supramaximal pre-conditioning activity may have a positive influence on endurance sports performance. A PAP response was originally thought to have the greatest effect on exercises that elicit lower threshold motor units (67); however, the majority of studies investigating this phenomenon have used ballistic, intermittent exercises. Few studies have investigated the acute effects of loaded pre-conditioning on middle- and long-distance sports performance.

To date, the evidence demonstrates that loaded pre-conditioning in addition to low intensity warm-ups may provide a performance advantage during the first half of an endurance performance. Further research is required to investigate whether this effect persists for the entire duration of an endurance performance. Research is also warranted regarding the effect of loaded
pre-conditioning on middle- and long-distance running performance. The high impact forces that occur during distance running compared to cycling and rowing may increase the effectiveness of loaded pre-conditioning as there may be an added contribution of acute increases in leg stiffness. Finally, it is not yet ascertained whether sports-specific pre-conditioning movements may maximize the PAP response. However, given the current evidence, it seems likely that loaded pre-conditioning may give athletes a slight advantage (e.g., the difference between first and second in a $1600-\mathrm{m}$ running race) and could easily be implemented into a standard warm-up routine.

## CHAPTER 3: METHODS

## Purpose and Specific Aims

The purpose of this study was to determine the pre-conditioning effect of weighted-vest sprints on physiological parameters during a graded exercise test (GXT) and running performance during a 1600-m time trial (TT) in trained male distance runners. It was hypothesized that weightedvest sprints would increase time to the onset of ventilatory threshold $1\left(\mathrm{VT}_{1}\right)$ and $2\left(\mathrm{VT}_{2}\right)$, lactate threshold (LT), and onset of blood lactate accumulation (OBLA); and decrease the $\mathrm{VO}_{2}$ time constant $(\tau)$ at the onset of exercise, and submaximal $\mathrm{VO}_{2}$ compared to control (sprints without weighted vest) during the GXT. Additionally, leg ( $\mathrm{k}_{\text {leg }}$ ) and vertical ( $\mathrm{k}_{\text {vert }}$ ) stiffness would increase during the GXT following weighted-vest sprints compared to control. Finally, time-to-completion during the $1600-\mathrm{m}$ TT would decrease following pre-conditioning weighted-vest sprints compared to control. Preconditioning weighted-vest sprints may be a feasible application in endurance running competitions (e.g., collegiate and high school track and field) and may acutely enhance endurance running performance, providing a competitive edge.

Specific Aim 1. Determine the effect of pre-conditioning weighted-vest sprints on physiological parameters in trained male distance runners.

To address Aim 1, we assessed submaximal $\mathrm{VO}_{2}, \mathrm{\tau}, \mathrm{VT}_{1}$, and $\mathrm{VT}_{2}$ by measuring oxygen consumption during a GXT using indirect calorimetry (TrueOne 2400, Parvo Medics, Salt Lake City, UT, USA) and LT and OBLA were measured via finger capillary blood samples following pre-conditioning weightedvest sprints.

Specific Aim 2. Determine the effect of pre-conditioning weighted-vest sprints on $\mathrm{k}_{\mathrm{leg}}$ and $\mathrm{k}_{\text {vert }}$ in trained male distance runners.

To address Aim 2, we assessed $\mathrm{k}_{\mathrm{leg}}$ and $\mathrm{k}_{\text {vert }}$ during a GXT and 1600-m TT following pre-conditioning weighted-vest sprints.

Specific Aim 3. Determine the effect of pre-conditioning weighted-vest sprints on running performance in trained male distance runners.

To address Aim 3, we assessed time-to-completion and 400-m splits during a $1600-\mathrm{m}$ TT following pre-conditioning weighted-vest sprints.

## Experimental Design and Methodology \& Materials

## Experimental Design \& Methodology

This study was a counterbalanced crossover design. Participants performed all experimental trials in different orders to reduce sequence effect. Different orders occurred for trials 2 and 3, and 4 and 5. Participants reported to the Human Performance Laboratory at the University of Idaho on 5 separate occasions for testing. Visit 1 involved familiarization and baseline testing. Visits 2 and 3 assessed physiological parameters during a GXT following pre-conditioning with a weighted vest or pre-conditioning with no additional weight. Visits 4 and 5 assessed running performance during a 1600-m TT following pre-conditioning with a weighted vest or pre-conditioning with no additional weight.

Table 1. Example order of visits.

| Visit | Pre-Conditioning Task |
| :---: | :--- |
| 1 | Familiarization and Baseline Testing |
| 2 | Weighted-vest sprints (20\% body mass) |
| 3 | Sprints with no additional weight |
| 4 | Weighted-vest sprints (20\% body mass) |
| 5 | Sprints with no additional weight |

Note: Visits 2 \& 3 are GXT experiment trials and visits 4 \& 5 are 1600-m TT experimental trials.

Each laboratory visit was separated by at least 72 h to diminish the potentiating effects of the previous trial and provide adequate time for recovery. Each trial was performed during the same time of day to avoid the potential influence of the circadian cycle. Environmental conditions (i.e., temperature, humidity, barometric pressure, and wind speed) were monitored and recorded prior to each trial. Participants were asked to refrain from physical activity, caffeine, and alcohol for 24 h prior to all visits. For each visit, participants were asked to consume similar meals the night prior to testing. Twenty-four-hour food logs were reported prior to Visit 2 and returned to participants to replicate meals for subsequent trials to control for an influence of nutrition on running performance. Participants were also asked to refrain from eating 3 h prior to each visit.

## Participants

Seventeen healthy, distance-trained male runners ( $\mathrm{VO}_{2 \text { peak }} \geq 50 \mathrm{~mL} \cdot \mathrm{~kg}^{-1} \cdot \mathrm{~min}^{-1}$ ) aged $18-50$ years were recruited via word-of-mouth and flyers posted on the University of Idaho campus, Washington State University campus, sports stores, and running club mailing lists in Moscow, Idaho and Pullman, Washington.

Participants were engaged in run training $\geq 5 \mathrm{~h}$ per week, at least 3 days per week for the previous 3 months. Participants were excluded if they had existing diseases (e.g., cardiovascular disease, metabolic syndrome, etc.), current musculoskeletal injury (injury-free for the previous 6 months), currently smoking, or regularly using ergogenic supplements (World Anti-Doping Agency
banned substances). Females were excluded due to previously reported sex differences in response to PAP protocols (23). All participants were pre-screened during Visit 1 to ensure their eligibility. The University of Idaho Human Subjects Committee approved this study, and all participants completed an informed consent and medical history questionnaire prior to commencement.

## Testing Procedures

## Visit 1: Familiarization \& GXT Baseline Testing

Participants reported to the lab to sign an informed consent, schedule testing dates and times, fill out a 24-h food log, complete a run training questionnaire, and become familiarized with the equipment (don weighted vest, insert mouthpiece used for metabolic cart, and walk/jog on treadmill) and study protocol. Following the collection of demographics and anthropometrics (height, cm; body mass, kg; DETECTO, Apex-SH, Webb City, MO), participants performed a warm-up on a motorized treadmill for 5 min at a self-selected pace. Following the warm-up, participants performed a GXT to volitional fatigue to assess $\mathrm{VO}_{2 \text { peak }}$ and study inclusion ( $\geq 50 \mathrm{~mL} \cdot \mathrm{~kg}^{-1} \cdot \mathrm{~min}^{-1}$ ). Upon GXT completion, participants were given at least 10 min to rest and recover then asked to don the weighted vest and perform 2-3 ten-s runs on the treadmill at various velocities. The weighted-vest runs following the GXT served as familiarization for subsequent weighted-vest sprint trials.

## GXT Details

Participants performed a GXT on a motorized treadmill (4FRONT, Woodway, Waukesha, WI, USA). A metabolic measurement system (TrueOne 2400; Parvo Medics, Salt Lake City, UT, USA) was used to measure oxygen consumption and carbon dioxide production. Following a 5-min selfselected pace warm-up on a motorized treadmill, the participant began the GXT. The testing procedure consisted of 3-min run stages at progressively higher speeds and transitioned to 1-min run bouts at $\mathrm{VT}_{2}$, at which speed remained constant and grade was increased until volitional fatigue. More specifically, the grade was set to $1 \%$ to mimic over-ground running (79) for stages 1-9 and
began at a speed of $3.13 \mathrm{~m}^{*} \mathrm{~s}^{-1}$ and increased by $0.447 \mathrm{~m}^{*} \mathrm{~s}^{-1}$ until a speed of $5.36 \mathrm{~m}^{*} \mathrm{~s}^{-1}$ or until $\mathrm{VT}_{2}$ was observed, at which point grade was increased by $2 \%$ every 1 min until cessation of exercise. $\mathrm{VT}_{2}$ was indicated by a concomitant increase in $\mathrm{V}_{\mathrm{E}} / \mathrm{VO}_{2}$ and $\mathrm{V}_{\mathrm{E}} / \mathrm{VCO}_{2} . \mathrm{VO}_{\text {2peak }}$ was determined by achieving at least 3 of the 5 following criteria: 1) respiratory exchange ratio $\geq 1.15,2$ ) heart rate $\geq$ $85 \%$ of age-predicted maximum $\left(H R_{\max }=208-(0.7 \cdot\right.$ age $\left.\left.)\right), 3\right)$ a plateau in oxygen consumption despite an increase in exercise intensity ( $<2.0 \mathrm{~mL} \cdot \mathrm{~kg}^{-1} \cdot \mathrm{~min}^{-1}$ increase), 4) a rating of perceived exertion $\geq 18$ on the Borg Scale, 5) voluntary cessation of exercise (28).

## Visits 2 and 3: GXT

## Weighted-Vest Sprints prior to GXT

Upon arrival, height ( cm ) and body mass ( kg ) were measured for each participant. They were asked to change into running attire (loose fitting shirt, running shorts and shoes), be fitted with a wristwatch (Fenix 3, Garmin, Kansas City, Kansas) and a chest-mounted accelerometer (HRMRun, Garmin, Kansas City, Kansas) was fastened to their chest to measure HR continuously and estimate $\mathrm{k}_{\text {leg }}$ and $\mathrm{k}_{\text {vert }}$ (described in detail below). Participants performed a warm-up on a motorized treadmill for 5 min at a self-selected pace. Following the warm-up, participants were asked to perform six 10-s weighted-vest (5.11 TacTec Plate Carrier, Rogue Fitness, Columbus, OH) sprints with $20 \%$ body mass (2) on a motorized treadmill. The 5.11 Tactical Weight Vest held medium and large ballistic plates in front and back pockets to evenly distribute weight. The initial three sprints were performed at a progressively increasing velocity to allow participants to warm up. The initial sprint was performed at a velocity that is $0.447 \mathrm{~m} \cdot \mathrm{~s}^{-1}$ lower than the velocity at $\mathrm{VT}_{2}(\mathrm{vVT} 2)$. The velocity was increased by $0.224 \mathrm{~m} \cdot \mathrm{~s}^{-1}$ for each successive sprint until a velocity that is $0.224 \mathrm{~m} \cdot \mathrm{~s}^{-1}$ greater than $\mathrm{vVT}_{2}$. The last three sprints were performed at $0.224 \mathrm{~m} \cdot \mathrm{~s}^{-1}$ greater than $\mathrm{vVT}_{2}$. One min separated each sprint to allow adequate recovery and full ATP-PC restoration. Participants then passively rested for 10 min before beginning a GXT to volitional exhaustion as described above. Visits 2 and 3
included blood lactate sampling during the GXT (described in detail below). The 10-min rest has been observed as an appropriate time for optimizing a PAP response $(3,12,14,24)$.

## Blood Lactate Measurement during GXT

Blood lactate was measured from a finger capillary sample. With 30 s remaining in each stage during the GXT, $20 \mu \mathrm{~L}$ of blood was collected from participants fingertip with a sterilized 28 G single-use lancet (Unistik 3 Comfort, Owen Mumford Inc., Woodstock, Oxfordshire, United Kingdom). Blood lactate concentration was determined immediately using a blood lactate analyzer (Lactate Plus Version 2, Nova Biomedical, Waltham, MA). The analyzer was calibrated according to the manufacturer's instructions before each running trial. Resting blood lactate concentration was measured at 7 min post-pre-conditioning, and peak blood lactate concentration was measured immediately after cessation of exercise.

Lactate Analysis. Resting lactate was defined as baseline lactate concentration during the 10-min recovery between pre-conditioning and GXT. LT was identified as an elevation of blood lactate above resting levels. To validate LT objectively, a two-phase linear regression model was used to estimate the inflection point of the lactate profile curve (94). OBLA was identified as a blood lactate level of $4 \mathrm{mmol} \cdot \mathrm{L}^{-1}$, and time to OBLA was estimated by plotting blood lactate concentration against time and visually connecting the data points (142). Peak lactate was defined as peak lactate concentration following cessation of exercise.

## $\mathrm{VO}_{2}$ Kinetics Measurement during GXT

$\mathrm{VO}_{2}$ response at the onset of exercise was assessed during the GXT. Pulmonary gas exchange was measured breath-by-breath for 3 min prior to exercise to obtain a baseline $\mathrm{VO}_{2}$. Gas exchange was continuously measured for the duration of the GXT. The time for $\mathrm{VO}_{2}$ to reach steady-state at light-intensity exercise (treadmill running at $3.13 \mathrm{~m} \cdot \mathrm{~s}^{-1}$ ) from baseline $\mathrm{VO}_{2}$ (time constant, $\tau$ ) was assessed.
 (78). During the GXTs, breath-by-breath $\mathrm{VO}_{2}$ after the onset of exercise was modeled using iterative nonlinear-regression techniques using Microsoft Excel. The breath-by-breath data was filtered to remove errant breaths (4 SD from preceding 5 breath average) and subsequently modeled using a monoexponential mathematical equation,

$$
\mathrm{VO}_{2}(\mathrm{t})=\mathrm{VO}_{2}(\mathrm{~b})+\mathrm{A}\left(1-\mathrm{e}^{-\mathrm{t} / \mathrm{\tau}}\right)
$$

where $\mathrm{VO}_{2}(\mathrm{~b})$ is the resting baseline value, taken as a mean of the final 2 min before the start of the GXT; A is the asymptotic amplitude for the exponential term; and $\tau$ is the time constant (equivalent to the mean response time).

## Ventilatory Thresholds

Ventilatory thresholds were determined in accordance with Pallares et al. (115). $\mathrm{VT}_{1}$ was identified as a concomitant increase in ventilatory equivalent of oxygen $\left(\mathrm{V}_{\mathrm{E}} / \mathrm{VO}_{2}\right)$ and end-tidal pressure of oxygen $\left(\mathrm{P}_{\mathrm{ET}} \mathrm{O}_{2}\right)$ with no concomitant increase in ventilatory equivalent of carbon dioxide $\left(\mathrm{V}_{\mathrm{E}} / \mathrm{VCO}_{2}\right) . \mathrm{VT}_{2}$ was identified as a concomitant increase in $\mathrm{V}_{\mathrm{E}} / \mathrm{VO}_{2}$ and $\mathrm{V}_{\mathrm{E}} / \mathrm{VCO}_{2}$ and a decrease in end-tidal pressure of carbon dioxide $\left(\mathrm{P}_{\mathrm{ET}} \mathrm{CO}_{2}\right)$.

Leg and Vertical Stiffness Data Collection during GXT

Data were sampled for the last 60 ss of each 3-min stage with a Garmin Fenix 3 watch with a chest-mounted accelerometer. Accelerometer-derived data (cadence, contact time, and flight time) were extracted from the GarminConnect ${ }^{\text {TM }}$ platform for subsequent calculation of $\mathrm{k}_{\mathrm{leg}}$ and $\mathrm{k}_{\mathrm{vert}}$.

Leq and Vertical Stiffness Analysis. The determination of $\mathrm{k}_{\mathrm{leg}}$ and $\mathrm{k}_{\mathrm{vert}}$ was in accordance with Polson et al. (119). $\mathrm{k}_{\text {leg }}$ and $\mathrm{k}_{\text {vert }}$ were calculated using the sine wave method (106) using running speed, body mass, and leg length along with cadence, contact time, and flight time which was extracted from accelerometer-derived data from the Garmin running watch. Figure 4 presents equations used to calculate $\mathrm{k}_{\mathrm{leg}}$ and $\mathrm{k}_{\text {vert. }} \mathrm{k}_{\text {leg }}$ for each step was determined equation 1 , where $\Delta L$ is
change in leg length, and $F_{\text {peak }}$ is the peak ground reaction force. $F_{\text {peak }}$ was determined by equation 2, where $t_{f}$ is flight time, and $t_{c}$ is contact time. $\Delta L$ was determined by equation 3 , where $\Delta y$ is the change in COM, $v$ is running velocity, and $L_{0}$ is the length of the leg $\left(L_{0}=\right.$ height $\left.(m) * 0.53\right)(106) . k_{\text {vert }}$ for each step was determined by equation $4 . \Delta y$ was determined by equation 5 , where $g$ is gravitational force. The Garmin wristwatch and chest-mounted accelerometer have been validated in the determination of $\mathrm{k}_{\text {leg }}$ and $\mathrm{k}_{\text {vert }}$ during treadmill running (119).

$$
\text { Equation 1: } \quad k_{l e g}=\frac{F_{p e a k}}{\Delta L}
$$

Equation 2: $\quad F_{\text {peak }}=\operatorname{body}$ mass $* 9.81 * \frac{p i}{2} *\left(\frac{t_{f}}{t_{c}}+1\right)$

Equation 3: $\quad \Delta L=L_{0}-\sqrt{L^{2}-\left(\frac{v t_{c}}{2}\right)^{2}}+\Delta y$

Equation 4: $\quad k_{v e r t}=\frac{F_{p e a k}}{\Delta y}$

Equation 5: $\quad \Delta y=\frac{F_{p e a k} t_{c}{ }^{2}}{m p i^{2}}+g \frac{t_{c}{ }^{2}}{8}$

Figure 4. Leg and vertical stiffness calculations as reported by Morin et al. (106)

## Sprints with no additional weight prior to GXT

This visit followed an identical format to Visit 2. Following the warm-up, participants performed six 10-s sprints with no additional weight. The sprints replicated the same procedure as described in Visit 2. Participants then rested for 10 min before beginning the GXT to volitional exhaustion as described above.

## Visits 4 and 5: Pre-conditioning Exercise followed by 1600-m TT

## Weighted-vest Sprints prior to 1600-m TT

Upon arrival to the lab, height ( cm ) and body mass $(\mathrm{kg})$ were measured for each participant. Participants were fitted with a chest-mounted accelerometer and wristwatch for the measurement of HR and subsequent estimation of $\mathrm{k}_{\mathrm{leg}}$ and $\mathrm{k}_{\mathrm{verr}}$. Participants performed a 5 -min self-selected submaximal run on the track to warm up. Following the warm-up, participants were asked to perform six $10-s$ weighted-vest sprints with $20 \%$ body mass (2) on the track. The first three sprints were performed at a progressively increasing velocity to allow participants to warm-up. The participants were asked to perform the initial sprint at their perceived 5-k race pace. The second and third sprints were performed at progressively faster velocities than the first sprint. The last three sprints were performed at a near-maximal velocity (faster than perceived $1600-\mathrm{m}$ race pace). One min separated each sprint to allow adequate recovery and full ATP-PC restoration. Participants then rested for 10 min before beginning a 1600-m maximal effort TT. Time-to-completion and 400-m splits were recorded.

## Leg and Vertical Stiffness Data Collection during 1600-m TT

Data were sampled continuously during the 1600-m TT with a Garmin Fenix 3 watch with a chest-mounted accelerometer. An automatic split was programed into the watch to record each 800-m segment. Accelerometer-derived data (cadence, contact time, and flight time) was extracted from the GarminConnect ${ }^{\text {TM }}$ platform for subsequent calculation of $\mathrm{k}_{\mathrm{leg}}$ and $\mathrm{k}_{\text {vert }}$. Sprints with no additional weight prior to 1600-m TT

This visit followed an identical format to Visit 4. Following the warm-up, participants performed six 10-s sprints with no additional weight. The sprints replicated the same procedure as described in the weighted-vest condition.


Figure 5. Example timeline of participant visits.

## Anticipated Risks and Solutions

Participants were not able to participate if they did not meet the inclusion criteria, presented with an injury that impeded their ability to exercise, or voluntarily asked to discontinue the study for any reason. There was minimal risk of injury during treadmill running due to falling or mis-stepping, which may have resulted in acute musculoskeletal injuries (e.g., ankle sprains, abrasions). To reduce this risk, participants were continuously monitored by several members (3 researchers) of the research team during treadmill running and weighted-vest sprints. If participants experienced dizziness and/or musculoskeletal pain while exercising, they were asked to discontinue the trial and undergo evaluation by a member of the research team as to whether they can perform the trial on a later date. During treadmill running, participants were allowed to stop running at any time by grabbing the safety bars. Immediately, a researcher pulled an emergency stop cord and the treadmill belt rapidly stopped moving.

Participants may have experienced delayed onset of muscle soreness (DOMS) the days following weighted-vest sprints and running performance, which is common following exercise testing. A warm-up and cooldown were utilized to prepare the muscles for physical activity and minimize the occurrence of DOMS.

All research personnel were CPR/AED and first aid certified. In the event of an injury that was beyond the scope of basic life support, EMS would have been promptly contacted, and the participant closely monitored until emergency personnel arrived.

## COVID Precautions

Due to the current pandemic, our laboratory developed several precautions. These precautions were to identify at risk participants that may have brought the virus into the laboratory. After the initial screening process to schedule the participants, they were asked a series of questions and had their forehead temperature measured prior to entering the laboratory.

Questions: Have you felt sick or had a high temperature over the past two weeks? Have you traveled outside of the Palouse region over the past two weeks? Have you been in contact with anyone that has tested positive for COVID over the past two weeks? If so, have you yourself been tested or contacted as part of the contract tracing?

If the participant passed all these questions and their body temperature was within the healthy range, they were asked to continue wearing their mask while they were in the building and performing tasks, with the exception of performing the GXT while attached to the metabolic cart.

For laboratory precautions, federal, state, and local COVID recommendations were followed.

## Statistical and Power Analyses

Sample size estimates were calculated using G*Power 3.1.9.7 based on outcome variables reported by Barnes et al. (12) investigating the effect of weighted-vest sprints on running performance during an incremental treadmill run to exhaustion. Estimates were calculated with an alpha of 0.05 and power of 0.80 . The means and SDs reported for oxygen consumption at $14 \mathrm{~km} \cdot \mathrm{~h}^{-1}$ $\left(46.1 \pm 2.0 \mathrm{ml} \cdot \mathrm{kg}^{-1} \cdot \mathrm{~min}^{-1}\right.$ vs $\left.43.3 \pm 2.1 \mathrm{ml} \cdot \mathrm{kg}^{-1} \cdot \mathrm{~min}^{-1}\right)$ and vertical stiffness $\left(10.3 \pm 2.1 \mathrm{kN} \cdot \mathrm{m}^{-1} \mathrm{vs} 12.5 \pm\right.$ $2.7 \mathrm{kN} \cdot \mathrm{m}^{-1}$ ) resulted in samples sizes of 7 and 12 , respectively. Sample size estimates were also calculated (G*Power 3.1.9.7) based on intraclass correlation coefficients (ICC) reported by Cerezuela-

Espejo et al. (33) describing the reliability of ventilation parameters ( $\mathrm{VT}_{1}$ and $\mathrm{VT}_{2}$ ) between two GXT trials. A reported ICC of 0.98 for $\mathrm{VT}_{1}$ between two $\mathrm{GXTs}(11.5 \pm 1.8 \mathrm{~min}$ vs $11.9 \pm 1.8 \mathrm{~min})$ for a sample size of 12 resulted in a power of 0.94 . A reported ICC of 0.95 for $\mathrm{VT}_{2}$ between two GXTs (15.8 \pm 1.4 min vs $16.2 \pm 1.4 \mathrm{~min})$ for a sample size of 12 resulted in a power of 0.81 . Therefore, 15 participants were recruited for statistical purposes with an anticipated dropout rate of $20 \%$.

All variables were assessed for normality. When normality was violated, a transformation was performed to normally distribute the data. A $\log _{10}$ transformation was used initially; however, several dependent variables violated normality following this transformation. Therefore, a two-step approach to normalizing data was used (151). A repeated measures two-way analysis of variance (ANOVA) was used as a statistical analysis with submaximal $\mathrm{VO}_{2}\left(3.13,3.58,4.02\right.$, and $\left.4.47 \mathrm{~m} \cdot \mathrm{~s}^{-1}\right)$, $\mathrm{VT}_{1}, \mathrm{VT}_{2}$, resting lactate, $\mathrm{LT}, \mathrm{OBLA}$, peak lactate, $\mathrm{k}_{\text {leg, }}, \mathrm{k}_{\text {vert }}, \mathrm{VO}_{2 \text { peak }}$, time-to-exhaustion, baseline $\mathrm{VO}_{2}$, $\tau$, amplitude, $H_{\text {peak }}$, 400-m splits (laps 1, 2, 3), and time-to-completion as dependent variables and warm-up condition (weighted vest and no weighted vest; within-subjects factor) and testing sequence (between-subjects factor) as the independent variables. When significant interaction effects were detected, a least significant difference (LSD) post hoc test was used to analyze the pairwise comparisons. Effect sizes (ES) were determined in accordance with Cohen (37). Threshold values for small, moderate, and large ES were $0.2,0.5$, and 0.8 , respectively (37). Statistical significance was accepted at $\mathrm{p} \leq 0.05$. Statistical analyses were performed using IBM SPSS Statistics v.28. Values are reported as mean $\pm$ SD.

## CHAPTER 4: RESULTS

Seventeen participants were recruited for this study. Two participants were excluded due to inadequate peak aerobic fitness $\left(<50 \mathrm{~mL} \cdot \mathrm{~kg}^{-1} \cdot \mathrm{~min}^{-1}\right)$, and two participants dropped out before completing all visits due to injuries unrelated to the experimental trials. The final analyses include 13 participants with no missing data. Participant characteristics are presented in Table 2.

Table 2. Participant characteristics.

|  | Mean $\pm$ SD | Range |
| :--- | :---: | :---: |
| Age (years) | $29.8 \pm 10.6$ | $19-47$ |
| Body Mass (kg) | $74.2 \pm 5.8$ | $67.0-85.5$ |
| Height (cm) | $182.3 \pm 5.9$ | $175.5-196.0$ |
| $\mathrm{VO}_{2 \text { peak }}(\mathrm{mL} \cdot \mathrm{kg}$ |  |  |
| Weekly Kiloms $\left(\mathrm{min}^{-1}\right)$ | $57.0 \pm 4.9$ | $50.2-65.4$ |
| Weekk ${ }^{-1}$ ) | $39.9 \pm 22.6$ | $16.1-96.6$ |
| Years of Training | $4.5 \pm 1.1$ | $3-6$ |

Note: $\mathrm{N}=13 . \mathrm{VO}_{2 \text { peak }}=$ peak oxygen uptake, $\mathrm{kg}=$ kilogram, km=kilometer, $\mathrm{cm}=$ centimeter, $\mathrm{mL}=$ milliliter, $\mathrm{min}=$ minute.

## Maximal, Submaximal, Lactate, and Oxygen Kinetics Measures during Graded Exercise Test

Maximal, submaximal, lactate, and oxygen kinetics data are presented in Table 3.

Submaximal $\mathrm{VO}_{2}$ group data are presented in Figure 6. Individual time-to-exhaustion data are presented in Figure 7. No significant interactions were observed for all GXT measures ( $p>0.05$, for all). Submaximal $\mathrm{VO}_{2}$ values in the weighted-vest condition at $3.13 \mathrm{~m} \cdot \mathrm{~s}^{-1}(\mathrm{p}<0.001, \mathrm{ES}=0.631), 3.58$ $\mathrm{m} \cdot \mathrm{s}^{-1}(\mathrm{p}=0.005, \mathrm{ES}=0.415)$, and $4.02 \mathrm{~m} \cdot \mathrm{~s}^{-1}(\mathrm{p}<0.001, \mathrm{ES}=0.362)$ were significantly lower compared to the control condition. $\mathrm{VT}_{1}(\mathrm{p}=0.005, \mathrm{ES}=0.273), \mathrm{VT}_{2}(\mathrm{p}=0.001, \mathrm{ES}=0.180)$, and resting lactate ( p $=0.01, \mathrm{ES}=0.928$ ) were significantly greater in the weighted-vest condition compared to the control condition. A significant effect of sequence was observed for $\mathrm{VO}_{2 \text { peak }}(p=0.04)$ and submaximal $\mathrm{VO}_{2}$ at $4.47 \mathrm{~m} \cdot \mathrm{~s}^{-1}(\mathrm{p}=0.01)$. No significant effect of vest nor effect of sequence was observed for $H R_{\text {peak }}$, time-to-exhaustion, lactate threshold, OBLA, peak lactate, baseline $\mathrm{VO}_{2}$ time constant, and amplitude ( $p>0.05$, for all).

Table 3. Performance measures during GXT following control and weighted-vest conditions.

|  | Control | Weighted vest | Main Effect of Vest (p) | Effect of Sequence (p) | Interaction <br> (p) | Cohen's d |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Maximal Performance Measures |  |  |  |  |  |  |
| $\mathrm{HR}_{\text {peak }}$ (beats $\cdot \mathrm{min}^{-1}$ ) | $190 \pm 14^{\ddagger}$ | $188 \pm 12$ | 0.122 | 0.346 | 0.907 | 0.153 |
| $\mathrm{VO}_{2 \text { peak }}\left(\mathrm{mL} \cdot \mathrm{kg}^{-1} \cdot \mathrm{~min}^{-1}\right)$ | $56.8 \pm 4.8$ | $56.5 \pm 5.3$ | 0.467 | $0.038^{+}$ | 0.582 | 0.059 |
| Time-to-exhaustion (s) | $866.4 \pm 160.1$ | $862.5 \pm 151.0$ | 0.658 | 0.942 | 0.232 | 0.025 |
| Submaximal Running Measures |  |  |  |  |  |  |
| $\mathrm{VO}_{2} 3.13 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(\mathrm{~mL} \cdot \mathrm{~kg}^{-1} \cdot \mathrm{~min}^{-1}\right)$ | $36.8 \pm 2.0$ | $35.6 \pm 1.8$ | <0.001* | 0.368 | 0.646 | 0.631 |
| $\mathrm{VO}_{2} 3.58 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(\mathrm{~mL} \cdot \mathrm{~kg}^{-1} \cdot \mathrm{~min}^{-1}\right)$ | $42.5 \pm 2.6$ | $41.5 \pm 2.2$ | 0.005* | 0.662 | 0.734 | 0.415 |
| $\mathrm{VO}_{2} 4.02 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(\mathrm{~mL} \cdot \mathrm{~kg}^{-1} \cdot \mathrm{~min}^{-1}\right)$ | $47.9 \pm 3.0$ | $46.9 \pm 2.5$ | $0.001{ }^{*}$ | 0.666 | 0.707 | 0.362 |
| $\mathrm{VO}_{2} 4.47 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(\mathrm{~mL} \cdot \mathrm{~kg}^{-1} \cdot \mathrm{~min}^{-1}\right)$ | $52.5 \pm 3.2$ | $52.3 \pm 3.1$ | 0.496 | $0.014^{+}$ | 0.942 | 0.063 |
| $\mathrm{VT}_{1}(\mathrm{~s})$ | $331.1 \pm 111.7$ | $364.2 \pm 129.8$ | $0.005^{*}$ | 0.818 | 0.055 | 0.273 |
| $\mathrm{VT}_{2}$ (s) | $648.1 \pm 193.5$ | $682.1 \pm 184.1$ | $0.001{ }^{*}$ | 0.502 | 0.161 | 0.180 |
| Lactate Measures |  |  |  |  |  |  |
| Resting Lactate ( $\mathrm{mM} \cdot \mathrm{L}^{-1}$ ) | $1.1 \pm 0.3$ | $1.6 \pm 0.7$ | 0.014* | 0.602 | 0.455 | 0.928 |
| Lactate Threshold (kg.m. $\mathrm{min}^{-1}$ ) | $169.5 \pm 22.5$ | $173.3 \pm 16.0$ | 0.654 | 0.124 | 0.434 | 0.195 |
| OBLA ( $\mathrm{kg} \cdot \mathrm{m} \cdot \mathrm{min}^{-1}$ ) | $201.5 \pm 26.8$ | $202.5 \pm 23.5$ | 0.550 | 0.515 | 0.410 | 0.040 |
| Peak Lactate ( $\mathrm{mM} \cdot \mathrm{L}^{-1}$ ) | $9.3 \pm 2.1$ | $9.3 \pm 2.4$ | 0.954 | 0.460 | 0.244 | 0.000 |
| Oxygen Kinetics Measures |  |  |  |  |  |  |
| Baseline $\mathrm{VO}_{2}\left(\mathrm{~L} \cdot \mathrm{~min}^{-1}\right)$ | $0.4 \pm 0.1$ | $0.4 \pm 0.1$ | 0.740 | 0.058 | 0.139 | 0.000 |
| Time Constant (s) | $19.4 \pm 5.2$ | $18.3 \pm 5.9$ | 0.115 | 0.859 | 0.598 | 0.198 |
| Amplitude (L•min ${ }^{-1}$ ) | $2.3 \pm 0.1$ | $2.3 \pm 0.2$ | 0.120 | 0.334 | 0.733 | 0.000 |

Note: $\mathrm{N}=13$. $\ddagger$ Data are presented as mean $\pm$ SD; *Significant effect of vest, $\mathrm{p}<0.05$. +Significant effect of sequence, $\mathrm{p}<0.05$. Cohen’s $\mathrm{d}=$ effect size, $\mathrm{VO}_{2 \text { peak }}=$ peak oxygen uptake, $\mathrm{HR}_{\text {peak }}=$ peak heart rate, $\mathrm{VO}_{2}=$ oxygen uptake, $\mathrm{VT}_{1}=$ ventilatory threshold $1, \mathrm{VT}_{2}=$ ventilatory threshold 2 , $\mathrm{L}=$ liter, $\mathrm{mL}=$ milliliter, $\mathrm{kg}=$ kilogram, $\mathrm{min}=$ minute, $\mathrm{mM}=$ millimole, $\mathrm{m}=$ meter, $\mathrm{s}=$ second.


Figure 6. Submaximal $\mathrm{VO}_{2}$ values during the GXT in the weighted-vest and control conditions. †Significant main effect of treatment, $\mathrm{p}<0.01$.


Figure 7. Individual values (solid lines) and mean (dotted line) for time-to-exhaustion during the GXT in the weighted-vest and control conditions.

## Stiffness Measures during GXT

Stiffness data are presented in Table 4. No significant interactions were observed for all stiffness measures ( $p>0.05$ ). No significant effect of vest nor effect of sequence was observed for leg stiffness ( $p>0.05$, for all speeds) and vertical stiffness ( $p>0.05$, for all speeds) at all speeds. No significant effect of vest nor effect of sequence was observed for overall leg stiffness ( $p>0.05$ ) and overall vertical stiffness ( $p>0.05$ ).

Table 4. Stiffness measures during GXT following control and weighted-vest conditions.

|  | Control | Weighted vest | Main Effect of Vest (p) | Effect of Sequence (p) | Interaction <br> (p) | Cohen's d |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Leg Stiffness |  |  |  |  |  |  |
| $\mathrm{k}_{\text {leg }} 3.13 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(\mathrm{kN} \cdot \mathrm{m}^{-1}\right)$ | $10.8 \pm 1.6^{\ddagger}$ | $11.0 \pm 1.7$ | 0.263 | 0.334 | 0.380 | 0.121 |
| $\mathrm{k}_{\text {leg }} 3.58 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(\mathrm{kN} \cdot \mathrm{m}^{-1}\right)$ | $10.3 \pm 1.6$ | $10.4 \pm 1.7$ | 0.195 | 0.153 | 0.223 | 0.061 |
| $\mathrm{k}_{\text {leg }} 4.02 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(\mathrm{kN} \cdot \mathrm{m}^{-1}\right)$ | $9.7 \pm 1.7$ | $10.0 \pm 1.8$ | 0.122 | 0.337 | 0.437 | 0.171 |
| $\mathrm{k}_{\text {leg }} 4.47 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(\mathrm{kN} \cdot \mathrm{m}^{-1}\right)$ | $9.5 \pm 1.9$ | $9.8 \pm 1.8$ | 0.190 | 0.628 | 0.749 | 0.162 |
| $\mathrm{k}_{\text {leg }}$ Overall ( $\mathrm{kN} \cdot \mathrm{m}^{-1}$ ) | $10.0 \pm 1.7$ | $10.2 \pm 1.6$ | 0.098 | 0.467 | 0.278 | 0.121 |
| Vertical Stiffness |  |  |  |  |  |  |
| $\mathrm{k}_{\text {vert }} 3.13 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(\mathrm{kN} \cdot \mathrm{m}^{-1}\right)$ | $26.3 \pm 3.3$ | $26.4 \pm 3.6$ | 0.505 | 0.751 | 0.100 | 0.029 |
| $\mathrm{k}_{\text {vert }} 3.58 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(\mathrm{kN} \cdot \mathrm{m}^{-1}\right)$ | $28.2 \pm 4.0$ | $28.0 \pm 4.0$ | 0.394 | 0.365 | 0.140 | 0.050 |
| $\mathrm{k}_{\text {vert }} 4.02 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(\mathrm{kN} \cdot \mathrm{m}^{-1}\right)$ | $29.9 \pm 3.7$ | $30.1 \pm 3.9$ | 0.463 | 0.726 | 0.190 | 0.053 |
| $\mathrm{k}_{\text {vert }} 4.47 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(\mathrm{kN} \cdot \mathrm{m}^{-1}\right)$ | $32.0 \pm 3.7$ | $32.3 \pm 3.9$ | 0.345 | 0.877 | 0.105 | 0.079 |
| $\mathrm{k}_{\text {vert }}$ Overall ( $\mathrm{kN} \cdot \mathrm{m}^{-1}$ ) | $30.2 \pm 3.5$ | $30.3 \pm 3.8$ | 0.632 | 0.869 | 0.150 | 0.027 |

Note: $N=13$. $\ddagger$ Data are presented as mean $\pm$ SD; Cohen's $d=e f f e c t ~ s i z e, ~ k_{l e g}=$ leg stiffness, $k_{v e r t}=$ vertical stiffness, $k N=k i l o n e w t o n, m=m e t e r$, s=second.

## 1600-m Time Trial

TT group data are presented in Table 5. Individual data for time-to-completion are presented in Figure 8. No significant interactions were observed for all measures during 1600-m TT ( $p>0.05$, for all). Individual $k_{\text {leg }}$ data are presented in Figure 9 . No significant effect of vest nor effect of sequence was observed for all measures during 1600-m TT ( $p>0.05$, for all).

Table 5. Performance measures during 1600-m TT following control and weighted-vest conditions.

|  | Control | Weighted vest | Main Effect of Vest (p) | Effect of Sequence <br> (p) | Interaction <br> (p) | Cohen's d |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Performance Measures |  |  |  |  |  |  |
| Time-to-completion (s) | $327.9 \pm 29.3^{\ddagger}$ | $327.8 \pm 29.2$ | 0.982 | 0.641 | 0.188 | 0.000 |
| 400-m Split 1 (s) | $76.7 \pm 7.9$ | $77.6 \pm 6.8$ | 0.369 | 0.361 | 0.397 | 0.122 |
| 400-m Split 2 (s) | $159.9 \pm 15.7$ | $161.1 \pm 14.1$ | 0.373 | 0.899 | 0.269 | 0.080 |
| 400-m Split 3 (s) | $247.2 \pm 23.5$ | $246.2 \pm 22.2$ | 0.652 | 0.679 | 0.186 | 0.044 |
| Running Metrics |  |  |  |  |  |  |
| Cadence (strides $\cdot \mathrm{min}^{-1}$ ) | $188.0 \pm 11.3$ | $188.1 \pm 12.1$ | 0.886 | 0.601 | 0.449 | 0.009 |
| Stride Length (m) | $1.6 \pm 0.2$ | $1.6 \pm 0.2$ | 0.381 | 0.945 | 0.179 | 0.000 |
| Ground Contact (s) | $0.2 \pm 0.0$ | $0.2 \pm 0.0$ | 0.231 | 0.531 | 0.481 | 0.000 |
| Stiffness Measures |  |  |  |  |  |  |
| $\mathrm{k}_{\text {leg }}\left(\mathrm{kN} \cdot \mathrm{m}^{-1}\right)$ | $9.4 \pm 1.8$ | $10.1 \pm 2.4$ | 0.121 | 0.960 | 0.987 | 0.330 |
| $\mathrm{k}_{\text {vert }}\left(\mathrm{kN} \cdot \mathrm{m}^{-1}\right.$ ) | $36.0 \pm 3.9$ | $37.0 \pm 4.9$ | 0.074 | 0.576 | 0.774 | 0.226 |

Note: $N=13$. $\ddagger$ Data are presented as mean $\pm S D$; Cohen's $d=e f f e c t ~ s i z e, ~ k_{l e g}=$ leg stiffness, $k_{v e r t}=$ vertical stiffness, $s=s e c o n d$, min=minute, $\mathrm{m}=$ meter, $\mathrm{kN}=$ kilonewton.


Figure 8. Individual values (solid lines) and mean (dotted line) for time-to-completion during the 1600-m TT in weighted-vest and control conditions.


Figure 9. Individual values (solid lines) and mean (dotted line) for leg stiffness during the $1600-\mathrm{m} \mathrm{TT}$ in weighted-vest and control conditions.

## CHAPTER 5: DISCUSSION

The purpose of the current study was to investigate the acute effect of weighted-vest sprints on indices of distance-running performance during a GXT as well as investigate the acute effect of weighted-vest sprints on middle-distance running performance during a 1600-m maximal effort TT. The main finding was that time to the onset of $\mathrm{VT}_{1}$ and $\mathrm{V} T_{2}$ was significantly greater in the weighted-vest condition compared to control ( $p<0.005$ and $<0.001$, respectively). Also, the energy cost of running at submaximal speeds $\left(3.13,3.58\right.$, and $4.02 \mathrm{~m} \cdot \mathrm{~s}^{-1}$ ) significantly decreased ( $p<0.01$ for all) following weighted-vest sprints compared to control, indicating that warming up with an external load may improve submaximal middle-distance running performance (i.e., running economy), but performance enhancement diminishes during maximal middle-distance running performance. No difference ( $p=0.98$ ) was observed in time-to-completion between the weightedvest and control (sprints without weighted vest) conditions during the TT.

Distance-running performance is predicated on an ability to maintain a given exercise intensity, namely LT and OBLA, throughout the duration of a running event. Lactate parameters may be influenced by an individual's buffering capacity to prevent the accumulation of metabolic byproducts (e.g., $\mathrm{H}^{+}, \mathrm{CO}_{2}$ ) lending to performance decrements and the cessation of exercise (130). An increase in LT and OBLA are correlated with a decrease in time-to-completion during marathon and 1600-m running performance, respectively (82). Given the observation that run performance improves by increasing LT and OBLA through chronic endurance training (82), an acute increase in these parameters may result in improved run performance. Moreover, blood lactate can be indirectly measured through ventilation, given a high correlation between lactate and ventilation parameters (115). In the current study, small and very small effect sizes were observed for time-to$\mathrm{VT}_{1}(\mathrm{ES}=0.273)$ and $-\mathrm{VT}_{2}(\mathrm{ES}=0.180)$ following weighted-vest sprints compared to control. The significant improvements in ventilation thresholds may have been influenced by an acute increase in
$\mathrm{k}_{\text {leg }}$ during the first few stages of the GXT, lending to an increase in elastic energy provision and decrease in metabolic energy utilization. Furthermore, small improvements in $\mathrm{VT}_{1}$ and $\mathrm{VT}_{2}$ may decrease time-to-completion by several seconds during middle-distance run events, which would likely provide an athletic advantage, as finishing times are generally separated by several seconds.
$\mathrm{VO}_{2 \text { peak }}$ results from the current study (Table 3) corroborate previous findings demonstrating no acute effect of weighted-vest sprints on $\mathrm{VO}_{\text {2peak }}$ during a GXT to volitional exhaustion ( $56.8 \pm 4.8$ $\left.\mathrm{mL} \cdot \mathrm{kg}^{-1} \cdot \mathrm{~min}^{-1} \mathrm{vs} 56.5 \pm 5.3 \mathrm{~mL} \cdot \mathrm{~kg}^{-1} \cdot \mathrm{~min}^{-1}, \mathrm{p}=0.47\right)(12) . \mathrm{VO}_{2 \text { peak }}$ is a function of several physiological constituents including mitochondrial density, capillary density, plasma and blood volume, and red blood cell mass (95), all which likely remain acutely unaffected by a resistance pre-conditioning exercise. Furthermore, time-to-exhaustion did not differ between weighted-vest and control conditions ( $862.5 \pm 151.0 \mathrm{~s}$ vs $866.4 \pm 160.1 \mathrm{~s}, \mathrm{p}=0.66$ ). It seems the potentiation effect of weighted-vest sprints diminished as the GXT proceeded. The time course of potentiation following a pre-conditioning activity in human studies takes at least several minutes to appear (6-10 min) and may last for >15 min $(15,159)$. In the current study, time-to-exhaustion in both warm-up conditions was roughly 14 min , and given the $10-\mathrm{min}$ recovery period following the pre-conditioning activity, the potentiation time course would have been exceeded before the completion of the GXT, potentially explaining unchanged maximal measures. Moreover, $\mathrm{VO}_{2}$ response at the onset of exercise (time constant; $\tau$ ) did not differ between weighted-vest and control conditions ( $18.3 \pm 5.9 \mathrm{~s}$ vs $19.4 \pm 5.2 \mathrm{~s}, \mathrm{p}=0.12)$. A greater time constant may spare anaerobic energy stores as $\mathrm{VO}_{2}$ moves toward steady-state, preserving anaerobic energy supply for use during the latter stages of run performance (28). However, weighted-vest sprints did have an acute effect on run performance during the initial stages of the GXT.

Weighted-vest sprints acutely reduced submaximal $\mathrm{VO}_{2}$ during the first 3 stages of the GXT compared to control ( $p<0.001, p=0.005$, and $p=0.001$ for stages 1,2 , and 3 , respectively), but not
during stage $4(p=0.50)$. Similarly, Barnes et al. (12) observed an acute reduction in $\mathrm{VO}_{2}$ following weighted-vest sprints compared to sprints without a weighted-vest during a 5 -min submaximal run on a treadmill. The authors suggested an acute increase in leg stiffness following weighted-vest sprints may have contributed to the improvement in running economy (i.e., decreased $\mathrm{VO}_{2}$ at a given running speed); however, this cannot be inferred as vertical stiffness (rather than leg stiffness) was assessed during a vertical jump test rather than during the submaximal run (12). In the current study, leg stiffness was estimated during the GXT at each submaximal stage. Very small effects (ES = $0.121,0.061,0.171,0.162$, and 0.121 for stage $1,2,3,4$, and overall, respectively) of weighted-vest sprints on $\mathrm{k}_{\text {leg }}$ were observed during the GXT (Table 4), which may indicate increased elastic energy provision and conserved metabolic energy. The conservation of metabolic energy may also be inferred through the increase in $\mathrm{VT}_{1}$ and $\mathrm{VT}_{2}$ following weighted-vest sprints compared to control (Table 3). Indeed, participants were able to run longer before reaching each ventilatory threshold, suggesting an increase in elastic energy and decrease in metabolic energy, during the initial stages of the GXT. However, no difference in $\mathrm{VO}_{2}$ between weighed-vest sprints and control was observed at $4.47 \mathrm{~m} \cdot \mathrm{~s}^{-1}$, indicating diminished potentiation because the time course of potentiation may have been exceeded at this timepoint (stage 4) during the GXT.

A plausible mechanism in reduced potentiation during stage 4 of the GXT is increasing muscle acidity. Changes in muscle pH should be considered in influencing $\mathrm{Ca}^{2+}$-sensitivity (defined as an increase in cross-bridge-generated muscle force for a given level of muscle fiber activation) (18) when a reduction in muscle potentiation may have occurred during the GXT. A small negative effect on $\mathrm{Ca}^{2+}$ - sensitivity has been observed with decreases in muscle pH following intense muscle contractions (99). Decreased $\mathrm{Ca}^{2+}$ sensitivity may affect the $\mathrm{Ca}^{2+}$ binding constant of troponin C (148), actin-myosin kinetics (87), or $\mathrm{Ca}^{2+}$ concentration in the space adjacent to the myofilaments (56). In the current study, resting lactate concentration (measured 7 min following the pre-
conditioning activity) was significantly greater in the weighted-vest compared to control conditions $\left(1.6 \pm 0.7 \mathrm{mM} \cdot \mathrm{L}^{-1}\right.$ vs $\left.1.1 \pm 0.3 \mathrm{mM} \cdot \mathrm{L}^{-1}, \mathrm{p}=0.01\right)$, suggesting decreased muscle pH at the onset of the GXT. Thus, a higher concentration of $\mathrm{H}^{+}$may have reduced $\mathrm{Ca}^{2+}$ sensitivity and likely does not underpin the enhanced submaximal performance observed during the initial stages of the GXT. Furthermore, the accumulation of $\mathrm{H}^{+}$at $4.47 \mathrm{~m} \cdot \mathrm{~s}^{-1}\left(5.95 \pm 0.90 \mathrm{mmol} \cdot \mathrm{L}^{-1}\right)$ during the GXT may have negated muscle potentiation for the remainder of the GXT, which is reflected in similar maximal measures (Table 3) between the two warm-up conditions. Therefore, the weighted-vest load (20\% body mass) may have exceeded the stimulus required to elicit muscle potentiation while inducing a low level of fatigue at the onset of the GXT. Future research should consider utilizing a reduced weighted-vest load ( $\sim 15 \%$ body mass) to improve both submaximal and maximal running performance.

Although submaximal running economy improved during the first 3 stages of the GXT following weighted-vest sprints compared to control, varying degrees of improvement were observed. A reduced effect of the weighted vest on running economy was observed with increasing speeds ( $E S=0.631,0.415$, and 0.362 for stages 1,2 , and 3 , respectively), which may be due to diminishing potentiation with time. It is difficult to ascertain if potentiation was influenced by exercise intensities as the greater running speeds occurred later in the GXT, thus, it is probable that the optimal time frame for a potentiation response was exceeded. However, a reduced potentiation response has been observed during low-intensity performance activities compared to high-intensity performance activities (137), so it seems likely that an increase in running speed (i.e., increased exercise intensity) would result in greater potentiation. Furthermore, the discrepancy between $\mathrm{k}_{\text {leg }}$ results in the current study and Barnes et al. (12) may be due to exceeding the potentiation timeframe as well as the intensity of the performance activity utilized to estimate $\mathrm{k}_{\mathrm{leg}}$. Ground reaction force during a vertical jump (123) is greater compared to submaximal running (71), thus,
greater stiffness ( $\mathrm{k}_{\text {leg }}$ and $\mathrm{k}_{\mathrm{vert}}$ ) is required to resist deformation of the legs and body upon contact with the ground. It is possible that an acute increase in stiffness is enhanced during a performance activity that elicits a greater ground reaction force. An increase in running speed has been correlated with an increase in $\mathrm{k}_{\operatorname{leg}}(44)$, so it seems likely $\mathrm{k}_{\operatorname{leg}}$ would increase to a greater extent following weighted-vest sprints compared to control as speeds increase during the latter stages of the GXT. Future research may consider investigating the effect of weighted-vest sprints on varying running speeds within the optimal potentiation timeframe.

Building on previous research $(12,111)$, this study sought to apply a loaded warm-up (weighted vest with $20 \%$ BM) to a track and field event (1600-m run) commonly performed by middle-distance runners. Performing weighted-vest sprints prior to a submaximal running assessment and GXT to volitional fatigue has been shown to decrease the energy cost of running ($6.0 \%$ ) and increase peak speed ( $+2.9 \%$ ), respectively (12). However, these assessments may lack direct applicability as they were performed on a treadmill. To the authors knowledge only one study has investigated the acute effect of weighted-vest ( 6.8 kg ) sprints on a simulated middle-distance race (5-km TT) (111). No difference in time-to-completion between weighted-vest sprints and control conditions ( $17.78 \pm 0.81 \mathrm{~min}$ vs $17.59 \pm 0.92 \mathrm{~min}, \mathrm{p}>0.05$ ) was observed (111). Similarly, in the present study, 1600-m time-to-completion did not differ between the weighted-vest and control conditions ( $327.8 \pm 29.2 \mathrm{~s}$ vs $327.9 \pm 29.3 \mathrm{~s}, \mathrm{p}=0.98$. Given evidence that high-level middle-distance runners have increased leg stiffness compared to their recreational counterparts (135), it seems likely that an acute increase in leg stiffness may contribute to reduced 1600-m time-to-completion in the current study. Leg stiffness increased following weighted-vest sprints compared to control in 8 of the 13 participants with varying magnitudes of change ( $0.14 \mathrm{kN} \cdot \mathrm{m}^{-1}$ to $2.9 \mathrm{kN} \cdot \mathrm{m}^{-1}$ ); however, it seems an acute increase in leg stiffness did not contribute to improved running performance during the TT. Although speculative, the acute increase in leg stiffness may have been too small ( $\mathrm{ES}=0.33$ )
to contribute to a reduction in time-to-completion. Furthermore, time-to-completion may not have improved due to persistent fatigue following weighted-vest sprints compared to control, at least through the first 800 m of the TT .

It is probable that the slower first half of the TT may be residual fatigue induced by sprinting with an external load, given the participants' lack of experience training with a weighted vest. Although not significantly different, the first ( $77.6 \pm 6.8 \mathrm{~s}$ vs $76.7 \pm 7.9 \mathrm{~s}, \mathrm{p}=0.37$ ) and $\mathrm{s}(161.1 \pm 14.1$ $s$ vs $159.9 \pm 15.7 \mathrm{~s}, \mathrm{p}=0.37$ ) 400-m laps during the $T T$ were slower in weighted-vest compared to control. Moreover, these recreational runners did not frequently engage in resistance training, which may have increased the fatigue/potentiation ratio. Indeed, large inter-individual variability in recovery duration is associated with numerous factors such as strength, training experience, and muscle fiber classification $(57,68,109)$. Another explanation for the lack of potentiation during the TT may be motor pattern interference effect. The motor pattern of one task has been shown to preserve as a subsequent task is commenced $(85,133)$. Moreover, Cronin et al. (39) observed decreased stride length (range: $-5.2 \%$ to $-16.5 \%$ ) and step frequency (range: $-2.7 \%$ to $-6.1 \%$ ), and increased stance phase duration (range: $12.8 \%$ o $24.5 \%$ ) during weighted-vest sprinting compared to un-resisted sprinting, suggesting differences in overload experienced by muscle groups, and possible motor pattern differences. Thus, weighted-vest motor patterns may have been preserved during the subsequent $1600-\mathrm{m} \mathrm{TT}$ and altered running mechanics. However, in the present study, stride length, cadence, and ground contact during the 1600-m TT (Table 5) did not differ ( $p>0.05$ ) between warmup conditions, indicating no overall change in motor patterns during the $T$. Given nearly identical time-to-completion values between the two conditions, neither a positive nor negative effect of warming up with weighted-vest sprints was observed. Due to no observed differences during the 1600-m TT between warm-up conditions, weighted-vest sprints may not be necessary to acutely induce performance improvements. However, middle-distance runners who are regularly
performing strength training may benefit from warming-up with weighted-vest sprints given evidence demonstrating stronger individuals eliciting greater and earlier potentiation (137); thus, fatigue may be adequately diminished before the onset of a 1600-m TT with a 10-min recovery period.

Overall, pre-conditioning weighted-vest sprints improved submaximal running performance, as measured by $\mathrm{VT}_{1}$ and $\mathrm{VT}_{2}$, and submaximal $\mathrm{VO}_{2}$, compared to pre-conditioning without additional weight. However, pre-conditioning weighted-vest sprints did not improve maximal running performance, as measured by time-to-completion during the $1600-\mathrm{m}$ TT as well as time-toexhaustion during the GXT, compared to pre-conditioning sprints without additional weight. Findings from the current study support the use of weighted-vest sprints to acutely improve running economy, which may be useful when performing submaximal run training or long distance runs where the conservation of metabolic energy becomes crucial toward the end of the run; however, further research is required to confirm these ideas.

## Conclusions

Extensive evidence exists demonstrating the efficacy of loaded pre-conditioning exercises acutely enhancing exercise performance during intermittent, ballistic physical activities such as sprinting, weightlifting, and jumping (34,75, $84,113,138,160,164,165$ ). In contrast, literature supporting the use of loaded pre-conditioning prior to endurance sports performance is sparse $(12,47,141)$. Indeed, loaded pre-conditioning may be more effective in individuals that present a greater proportion of type II muscle fibers (5), hence supporting the use of resistance exercises for acute muscle potentiation in sporting events with powerful, short duration movements. Individuals with predominately type I muscle fibers may not show the same level of muscle potentiation compared to their type II muscle fiber counterparts following loaded pre-conditioning; however,
given the mechanism of increased musculotendinous stiffness underpinning acute muscle potentiation, loaded pre-conditioning may be useful in elastic-recoil sports such as distance running. In the current study, a small, non-significant increase in leg stiffness was observed during both the GXT and 1600-m TT. It remains unclear whether the acute increase in leg stiffness contributed to improved running economy during the first half of the GXT, but it seems running performance during the latter half of the GXT and 1600-m TT was not influenced by weighted-vest sprints. Future research is required to elucidate the effect of weighted-vest sprints on middle-distance running performance in athletes of homogenous fitness and training, frequent engagement in middledistance racing or TTs, and regularly perform strength training.

## Limitations

It is important to note several limitations to the current study. First, participants varied in fitness level $\left(\mathrm{VO}_{2 \text { peak }}\right.$ range: $\left.50.2-65.4 \mathrm{~mL} \cdot \mathrm{~kg}^{-1} \cdot \mathrm{~min}^{-1}\right)$ and training status (run training range: 16.1-96.6 $\mathrm{km} \cdot$ week $^{-1}$ ), which may have resulted in varying magnitudes of potentiation following weighted-vest sprints. Greater potentiation has been associated with increased fitness and training experience in distance-runners (67), and strength-trained individuals demonstrate greater potentiation following loaded pre-conditioning compared to non-strength-trained individuals (88); thus, inter-individual responses due to differing self-reported training status and fitness may have skewed performance outcome measures. Furthermore, the current participants may not have previously engaged in strength training, given the omission of strength training as an inclusion criterion, which may have influenced the results. Second, 1600-m TT familiarizations were not included. Greater variability in performance and pacing between TTs has been demonstrated in less trained participants compared to experienced athletes (73). In recreationally active participants with no prior experience of a TT, the effect size for changes in 20-km cycling TT duration was small (<0.49) after three familiarization TTs (73). Participants in the current study reported an average of 4.9 years of run training
experience, all with at least 1 race (high school, college, or recreational competitions) within the previous year; therefore, 1 or 2 familiarization TTs may have improved exercise performance reproducibility. Third, multiple-trial averaging to ascertain oxygen uptake kinetics to steady-state exercise has been used extensively in research $(26,27,153,158)$. In the current study, only 1 trial for each warm-up condition was used to derive oxygen uptake data, which may have resulted in measurement error. Finally, environmental conditions between participants varied, and weather may have an impact on running performance. Marathon performance in male distance-runners for varying populations was significantly reduced ( $p<0.05$ ) in $10-15^{\circ} \mathrm{C}$ compared to $20-25^{\circ} \mathrm{C}(45)$. Data collection for the 1600-m TTs occurred between April and May with ambient temperature and relative humidity ranging from $10-20^{\circ} \mathrm{C}$ and $40-50 \%$, respectively. Although 1600 m is a substantially shorter distance compared to a marathon, the impact of weather on the current study should be considered.

Institutional Review Board 875 Perimeter Drive, MS 3010 Moscow, ID 83844-301C Phone: 208-885-6162 Fax: 208-885-6014 Email: irb@uidaho.edu
December 01, 2020

## To: Ann Brown

## Cc: Christopher Alfiero, M.S.

From: Sharon K. Stoll, Chair
University of Idaho Institutional Review Board
Title: The effect of pre-conditioning weighted-vest sprints on physiological parameters during a graded exercise test and 1-mile running performance.
Protocol: 20-191, Reference: 011338
Review Type: Process Administratively
Approved: 12/01/2020
Expiration Date: 11/29/2021

On behalf of the Institutional Review Board at the University of Idaho, I am pleased to inform you that the protocol for this research project is approved as offering no significant risk to human subjects.

This study may be conducted according to the protocol described in the application. Amendments must be submitted for IRB approval prior to implementing changes. Research that has been approved by the IRB may be subject to further appropriate review and approval or disapproval by officials of the Institution. Every effort should be made to ensure that the project is conducted in a manner consistent with the three fundamental principles identified in the Belmont Report: respect for persons; beneficence; and justice. As Principal Investigator, you are responsible for ensuring compliance with all applicable FERPA regulations, University of Idaho policies, and state and federal regulations. The Principal Investigator is responsible for ensuring that all study personnel have completed the online human subjects training requirement.

Federal regulations require researchers to follow specific procedures in a timely manner. For the protection of all concerned, the IRB calls your attention to the following obligations that you have as Principal Investigator of this study.

1. For any changes to the study, an IRB Protocol Amendment Request

Form must be submitted to the IRB. The amendment request must be reviewed and approved before implementation.
2. Any unanticipated/adverse events or problems occurring as a result of participation in this study must be reported immediately to the IRB.

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