View metadata, citation and similar papers at core.ac.uk

brought to you by CORE

provided by St Mary's University Open Research Archive

- The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

The effect of ischemic preconditioning on repeated sprint cycling performance.

STEPHEN D. PATTERSON, NEIL E. BEZODIS, MARK GLAISTER AND JOHN R. PATTISON

School of Sport, Health and Applied Science, St Mary's University, Strawberry Hill, Twickenham, TW1 4SX, UK.

Corresponding Author:

Dr Stephen D. Patterson

School of Sport, Health and Applied Sciences

St Mary's University, Twickenham

Waldegrave Road

Strawberry Hill

Twickenham, UK

Phone: +44 2082402357

Fax: +44 2082404212

E-mail: Stephen.Patterson@smuc.ac.uk

Running Title: Ischemic preconditioning and sprint performance

Disclosure of Funding: None

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

Abstract

Purpose: Ischemic preconditioning enhances exercise performance. We tested the hypothesis that ischemic preconditioning would improve intermittent exercise in the form of a repeated sprint test during cycling ergometry. **Methods:** In a single-blind, crossover study, fourteen recreationally-active males (mean ± SD; age 22.9 ± 3.7 years, height 1.80 ± 0.07 m, mass 77.3 ± 9.2 kg) performed twelve 6 s sprints following four 5 min periods of bilateral limb occlusion at 220 mmHg (ischemic preconditioning) or 20 mmHg (placebo). **Results:** Ischemic preconditioning resulted in a 2.4 ± 2.2, 2.6 ± 2.7 and 3.7 ± 2.4% substantial increase in peak power for sprints 1, 2 and 3 respectively, relative to placebo, with no further changes between trials observed for any other sprint. Similar findings were observed in the first three sprints for mean power output following ischemic preconditioning (2.8 ± 2.5, 2.6 ± 2.5 and 3.4 ± 2.1%, for sprints 1, 2 and 3 respectively), relative to placebo. Fatigue index was not substantially different between trials. At rest tissue saturation index was not different between trials. During the ischemic preconditioning / placebo stimulus there was a -19.7 ± 3.6% decrease in tissue saturation index in the ischemic preconditioning trial, relative to placebo. During exercise there was a 5.4 ± 4.8% greater maintenance of tissue saturation index in the ischemic preconditioning trial, relative to placebo. There were no substantial differences between trials for blood lactate, electromyography (EMG) median frequency, oxygen uptake or rating of perceived exertion (RPE) at any time points. **Conclusion:** Ischemic preconditioning improved peak and mean power output during the early stages of repeated sprint cycling and may be beneficial for sprint sports.

Key Words: Ischemia, occlusion, power output, multiple sprint, fatigue

- The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

Introduction

Ischemia-reperfusion injury underpins the damage caused by either disease and /or deliberately imposed interruption of blood supply to tissues. However, since 1986, brief and repeated bouts of ischemia / reperfusion, known as ischemic preconditioning, have been demonstrated to protect many organs, including the myocardium (32), liver (35) and skeletal muscle (21), from the damage caused by a subsequent prolonged ischemic event. In addition to the clinical use of ischemic preconditioning, this technique has also been applied immediately before exercise to improve performance. Across a range of various exercise modes, performance has been enhanced by 1-8% (3, 12, 13, 24, 25) which makes it potentially beneficial for athletic events where such small margins are the difference between winning or losing.

Research to date has primarily focussed on events of an endurance nature and has identified improvements in peak oxygen uptake ($\dot{V}O_{2max}$; 13), power output at $\dot{V}O_{2max}$ (12), running time trial performance (3), 1000 m rowing performance (25) and time to task failure (7) following ischemic preconditioning. Relatively little research has focused on performance during shorter durations and the findings are conflicting. For example, an improvement in 100 m swimming performance was observed in elite national level swimmers (24) but no effect of ischemic preconditioning was demonstrated on single 30 m running sprint performance (19) or cycling exercise at 130% $\dot{V}O_{2max}$ (12).

Repeated sprint exercise provides a model to investigate transitions from high to low metabolic work, a common feature of many team sports. The major energy demands of repeated sprint exercise are derived from phosphocreatine (PCr) and anaerobic glycolysis (18), and recent work suggests a strong relationship between PCr resynthesis and recovery of repeated sprint performance (31). Alternatively, there is an increased reliance on aerobic

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

energy production during the latter stages of repeated intense exercise as evidenced by a larger reduction in anaerobic energy production than performance (18, 30) and increased muscle oxygen uptake (6). Furthermore, reducing (5) or enhancing (4) oxygen availability during repeated exercise impaired or enhanced performance, respectively, which suggests that the aerobic system plays an important role, possibly through faster PCr resynthesis.

Ischemic preconditioning may improve aerobic metabolism as evidenced by increased $\dot{V}O_{2max}$ (13), accelerated $\dot{V}O_2$ kinetics (34) and improved oxygenation of skeletal muscle (38) and it may therefore reduce the performance related decline in power output associated with repeated sprint exercise. Secondly, in ischemic reperfusion injury models, ischemic preconditioning enhances PCr resynthesis following ischemia (1, 29) and thus may enhance the ability to recover between sprints. Therefore, the aim of this study was to investigate the effect of ischemic preconditioning on repeated sprint cycling performance. Given the apparent ability of ischemic preconditioning to improve aerobic metabolism and promote PCr resynthesis it was hypothesized that it would improve repeated sprint cycling performance by reducing the rate of fatigue.

METHODS

Participants

In a randomized, single blind, crossover study, fourteen healthy males (mean ± standard deviation (SD); age 22.9 ± 3.7 years, height 1.80 ± 0.07 m, mass 77.3 ± 9.2 kg) recreationally active in repeated sprint sports such as field hockey, soccer and rugby, volunteered to participate. Participants were naïve to the effect of ischemic preconditioning on exercise performance and were not informed about the rationale of the study. They were

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

fully informed of all procedures and associated risks before completing a training history questionnaire and providing written, informed consent. Participants reported they had actively been involved in sport for an average of 12 years, with time spent training each week reported as 6.7 ± 2.3 hours. Approval for the study's procedures was granted by St Mary's University Ethics Committee which conformed to the Declaration of Helsinki.

Experimental Overview

All participants reported to the laboratory for four exercise trials. In the initial trial, data were obtained on individual anthropometric characteristics such as body mass, height and four skinfolds (subscapular, biceps brachii, triceps brachii, and iliac crest). During this trial participants were familiarised with the repeated sprint cycling protocol, consisting of twelve 6 s cycle sprints with 30 s of passive recovery between each sprint. Trial 2 was a repeat of the first, to further familiarise the participants with the exercise protocol. Trials 3 and 4 were the experimental trials which consisted of either ischemic preconditioning or placebo treatment prior to the exercise protocol. The experimental trials were performed in a counterbalanced manner, separated by 5-7 days to ensure no possible carryover of acute ischemic preconditioning (28). During both trials respiratory gas exchange, electromyography (EMG) of the vastus lateralis (VL) and near-infrared spectroscopy (NIRS) of the VL were recorded. Participants indicated their rating of perceived exertion (RPE, 6 – 20; Borg's scale) and blood was taken from the earlobe at rest and following sprints 4, 8 and 12 before being subsequently analyzed for lactate. Participants performed all of their trials at the same time of day (±1 h) and laboratory conditions were controlled at approximately 20°C and 38% relative humidity during all trials. Participants were instructed to maintain their normal diet, to refrain from any form of intense physical activity and caffeine for the 24 h period prior to testing, and not to eat for 3 h before each trial.

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

Experimental Measures

Ischemic Preconditioning

In trials 3 and 4 exercise was preceded by ischemic preconditioning or placebo, performed in a supine position using bilateral occlusion (3, 13). In the ischemic preconditioning trial automatic occlusion cuffs (14.5 cm width - Delfi Medical Innovations, Vancouver, Canada) were positioned proximally around the thigh and inflated to 220 mmHg for 5 minutes followed by 5 minutes of reperfusion. This procedure was repeated four times (3). The placebo trial was identical to the ischemic preconditioning trial except that the cuffs were inflated to 20 mmHg. The time delay between the cuff removal and the beginning of the warm up for the exercise test was 30 minutes as ischemic preconditioning has been demonstrated to improve exercise performance within 45 minutes of the final cuff inflation (3).

Repeated-sprint cycling

The exercise protocol consisted of twelve 6 s sprints with resistance set at a torque factor of 1.0 $N \cdot m \cdot kg^{-1}$ on a cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) with individual participant cycling position being established during visit 1 and then replicated on each subsequent visit. Participants performed a standardized warm-up, consisting of 3 minutes of cycling at 120 W, followed by two maximal 6 s sprints, with 1 minute between efforts followed by 5 minutes of passive rest. Toe clips were used to secure the feet to the pedals and strong verbal encouragement was provided throughout each trial. Participants performed each sprint with the pedals in the same starting position and were instructed to sprint as fast as possible maintaining maximal effort until asked to stop. Each sprint was initiated by illuminating a series of 20 light emitting diodes (LEDs) which were synchronized with the EMG recording. During the 30 s rest period after each sprint

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

participants remained seated on the ergometer. Mean and peak power output were calculated for each condition. The percentage decrement score (S_{dec}) for all 12 sprints was calculated as the percent difference between total and ideal peak power output, where total power represents the sum of peak power values from all sprints (S_n where n = 1:12) and ideal power represents the number of sprints multiplied by the highest peak power (S_{best}) achieved (20).

$$S_{dec}(\%) = \left[1 - \frac{(S_1 + S_2 + \ldots S_{12})}{S_{best} \times 12}\right] \times 100$$

Cardiorespiratory Measures

Respiratory gas exchange was measured during the entire exercise protocol through breath-by-breath analysis using an open spirometric system (Oxycon Pro, Jaeger, Hoechburg, Germany). The gas analyser was calibrated prior to each trial using oxygen and carbon dioxide gases of known concentrations (Cryoservice, Worcester, UK), and the turbine volume transducer was calibrated using a 3 L precision syringe (Hans Rudolph Inc, Shawnee, USA). During the trials participants breathed room air through a facemask (Hans Rudolph, Kansas City, MO, USA) that was secured in place by a head-cap assembly (Hans Rudolph, Kansas City, MO, USA). Respiratory gas exchange data were subsequently averaged on a 1 s basis and then averaged for the overall exercise protocol, so that the total time of analysis was 432 s ((12 × (6 s sprint + the following 30 s recovery periods)).

Muscle EMG

The EMG activity of the VL muscle of the right leg was recorded at 1000 Hz using a data acquisition system (Biopac MP150, Biopac Systems Inc. CA, USA). Before placement of the electrodes, the overlying skin was prepared. The hair was shaved and the skin thoroughly cleaned with alcohol to reduce skin electrode interference. Pre-gelled disposable

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

hypoallergenic 1 cm snap-electrodes (Performance Plus, Vermed, VT, USA) were fixed two-thirds of the distance along a line from the anterior spina illaca superior to the lateral side of the patella (17). Electrode centres were placed 2.0 cm apart, parallel to the direction of muscle fibres, with a reference electrode located above a prepared site on the shaft of the tibia. The EMG electrode placement was marked on the skin by indelible pen to ensure similar placement of electrodes between experimental trials. EMG recording was initiated by a digital trigger coincident with the start of each 6 s sprint. The start of each sprint was identified from the square wave pulse provided by the synchronization trigger and the subsequent 6 s of data were used for the analysis of each individual sprint. The raw EMG data were band pass filtered to remove the signal outside of the 20 – 500 Hz range. To investigate the difference in VL EMG frequency between the two conditions, the filtered EMG data from each sprint were transformed in to the frequency domain using a fast Fourier transformation and the median frequency (MDF) of the resulting power spectrum density was calculated. The MDF values from each of the 12 sprints were then analysed using linear regression, and the gradient of this line was extracted as a representation of the change in frequency (fatigue) across the 12 sprints (33).

NIRS Measurements

During experimental trials, muscle oxygenation of the left VL was continuously monitored using portable NIRS apparatus which is a wireless spatially resolved dual-wavelength spectrometer (Portamon, Artinis Medical Systems, BV, The Netherlands). Changes in tissue saturation index (TSI, expressed as a %) were measured using two wavelengths (750 and 850 nm), using an arbitrary value for the differential pathlength of 3.83 (10). During rest and prior to the preconditioning procedure a measure of TSI was taken.

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

During the preconditioning and placebo procedures, TSI was averaged over the duration of each 5 minute period of ischemic preconditioning and the value used was for the portion of time the cuff was inflated only (4 × 5 minutes of pressure). During the repeated sprint cycling protocol, TSI was calculated as an average across all the sprints and recovery time, in a similar manner to oxygen uptake data described above. The NIRS device was positioned on the left VL using the same procedures described above for the EMG placement (for the opposite leg). As with EMG placement an indelible pen was used to mark the placement of the device and to ensure similar placement between trials. The NIRS device was covered with a black light-absorbing cloth to prevent contamination from ambient light. During all tests the NIRS device was connected to a personal computer by Bluetooth for data acquisition (10 Hz). Skinfold thickness was measured at the site where the NIRS probe was attached before each trial using Harpenden skinfold calipers (British Indicators Ltd, UK). For all participants, the calculated value of skin and subcutaneous tissue thickness was less than half of the distance between the source and the detector.

Blood Lactate Measurement

The right ear lobe was cleaned using an alcohol swab and punctured using an automated lancet. At rest and immediately following sprints 4, 8 and 12, a blood sample was drawn using a 20 µl capillary tube (EKF Diagnostics, Barleben, Germany). The whole blood sample was hemolysed in a pre-filled micro test tube and analysed using a blood lactate/glucose analyser (Biosen C_Line, EKF Diagnostics, Barleben, Germany).

Statistical Analysis

Data were analysed using a contemporary magnitude-based inferences approach (22) because small changes in performance can be meaningful in athletes. Data were log transformed to reduce non-uniformity of error except for RPE due to its interval nature. The

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

threshold value for the smallest meaningful change for mean and peak power output was set as 0.8% (2). For all other data, the smallest worthwhile or important effect for each dependent variable was the smallest standardised (Cohen) change in the mean: 0.2 times the between-subject SD for baseline values of all participants (8). Qualitative descriptors were assigned to the quantitative percentile scores as follows: 25–75% *possible*; 75–95% *likely*; 95–99% *very likely*; >99% *almost certain* (20). A substantial effect was set at > 75%. Effect size was calculated using threshold values for Cohen's *d* statistics (0.2; small, 0.5; moderate and 0.8; large). Data are presented as mean ± SD or percent change from placebo (%Δ ± 90% confidence interval (± 90% CI)), percent likelihood that the difference between conditions was larger or smaller (% likelihood) and effect size). An effect was deemed unclear if its confidence limits overlapped the thresholds for both the smallest beneficial and the smallest harmful effect, that is, if the effect could be substantially positive and negative.

RESULTS

The maximal peak power (mean ± SD) obtained during the repeated sprint cycling test was 1594 ± 208 and 1630 ± 192 W for placebo and ischemic preconditioning, respectively. Qualitative analysis revealed that performing ischemic preconditioning before sprint activity led to a *likely* increase in maximum peak power output (2.5 ± 1.9%, 93%, small (%Δ, % likelihood, effect size)). Raw peak and mean power output data for each sprint are presented in Figures 1 and 2, respectively. Ischemic preconditioning, relative to placebo, resulted in substantial increases in peak power output for sprints 1 (2.4 ± 2.2%, 89% *likely*, small), 2 (2.6 ± 2.7%, 87% *likely*, small) and 3 (3.7 ± 2.4%, 97% *very likely*, small) only, with effects unclear for the remaining sprints. Mean power output followed a similar pattern with substantial increases in sprints 1 (2.8 ± 2.5%, 91% *likely*, small), 2 (2.6 ± 2.5%, 88% *likely*,

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

small) and 3 (3.4 ± 2.1%, 98% *very likely*, small) for the ischemic preconditioning trial, relative to placebo, and the effects on the remaining sprints were deemed unclear. During the repeated sprint cycling protocol, fatigue was evident in both trials as represented by S_{dec} values of 13.2 ± 5.6% and 14.7 ± 5.9% for placebo and ischemic preconditioning, respectively. Qualitative analysis revealed a *possibly* greater fatigue rate when repeated sprint cycling was performed following ischemic preconditioning (13.5 ± 16%, 64% *possible*, small).

Blood lactate was not different at rest prior to the placebo and ischemic preconditioning trials (mean ± SD; 1.1 ± 0.2 and 1.0 ± 0.3 $mmol.L^{-1}$, respectively; *unclear*, trivial). Blood lactate was *possibly* higher when measured at sprints, 4, 8 and 12 in the ischemic preconditioning (Table 1). Relative to placebo, the effects of ischemic preconditioning on perceived exertion at sprints 4, 8 and 12 were -0.1 ± 0.6, 0.2 ± 0.7 and 0.1 ± 0.8 (arbitrary units), respectively, with qualitative analysis interpretation deeming differences and effect sizes as unclear or trivial. Data for TSI are presented in Table 1. Briefly, effects for TSI at rest, between trials were unclear. During the occlusion / preconditioning stimulus there was an *almost certain* decrease in TSI during the ischemic preconditioning trial, relative to placebo. During exercise there was a *likely* higher increase in TSI in the ischemic preconditioning trial when compared with placebo (Table 1). At rest and during exercise, differences in oxygen uptake between trials were unclear (Table 1). The rate of change in MDF of EMG was *possibly* higher in the ischemic preconditioning trial, relative to the placebo trial (Table 1).

DISCUSSION

The main aim of this study was to investigate the effect of ischemic preconditioning on repeated sprint cycling performance. Relative to placebo, the results showed that ischemic preconditioning was associated with a 2 – 4% increase in both mean and peak power output

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

in the early phase of the protocol. The improvement in power output is similar to other ergogenic aids used during this type of exercise (15, 16) and to performance improvements observed following ischemic preconditioning using different exercise modes (12, 24).

The present investigation is the first to demonstrate an improved power output following ischemic preconditioning during a repeated sprint protocol. Despite rejecting our hypothesis, we did observe substantial increases in both peak and mean power output for the first three sprints. Previous research has demonstrated an improved muscle force production following ischemia and reperfusion in animal (21, 26) and human models (27). Due to the original aim and thus design of the study it was not possible to determine the contribution of increased motor unit recruitment to improved performance, although it does remain a possibility. EMG amplitude has previously been demonstrated to increase in skeletal muscle of animals following ischemic preconditioning (36), suggesting increased motor unit recruitment. In the only relevant human study, muscle fibre conduction velocity, which measures the speed of action potential or excitatory impulse, is increased during isometric exercise; yet ischemic preconditioning did not play a role (37).

It is recognized that high energy compounds are important for energy production during repeated sprint activity, with total anaerobic contributions of ATP production during a single 6 s sprint being 6%, 50% and 44% from ATP, PCr and anaerobic glycolysis, respectively (18). Whilst speculative, it is possible that the increased power production in the first three sprints in the ischemic preconditioning trial may have been a result of increased ATP production from anaerobic sources. Following ischemic reperfusion injury ATP content is maintained in rabbit and mice heart muscle as a result of ischemic preconditioning via increased concentration of PCr and PCr / ATP ratio (29) or increased anaerobic glycolysis (23). To date little evidence is available on concentrations in skeletal muscle, however

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

increased PCr production has been observed using ^{31}P MRS in recovery from an ischemic event (1). Therefore it is possible that improved power output may be a result of increased anaerobic energy contribution early in the sprint protocol. Within the current study a *possible* increased blood lactate concentration was observed following the fourth sprint in the ischemic preconditioning trial, giving further weight to this suggestion although it was not a substantial effect.

Originally, it was hypothesised that ischemic preconditioning would improve aerobic metabolism and thus improve the ability to recover between sprints. Markers of aerobic fitness such as $\dot{V}O_{2max}$ and $\dot{V}O_2$ kinetic parameters are related to the ability to offset fatigue during a repeated sprint effort (14, 30), whilst an increased aerobic energy production contributes towards power production in the latter stages of repeated intense exercise (6, 18, 30). Previous research employing one bout of circulatory occlusion prior to the start of an exercise bout has demonstrated accelerated pulmonary $\dot{V}O_2$ kinetics (34). Moreover ischemic preconditioning has been shown to increase $\dot{V}O_{2max}$ (13), suggesting that the method may be used to help maintain power output during repeated sprint exercise, via improved PCr resynthesis (9, 18, 34). However, data from the present study does not support this theory as evidenced by the similarity between trials for $\dot{V}O_2$ during the repeated sprint protocol.

Alongside an increased aerobic metabolism, ischemic preconditioning has been associated with improved muscle oxygenation during and in recovery from exercise (38). As expected, TSI was *almost certainly* decreased by 20% during the preconditioning stimulus, relative to placebo, which is similar to a previous investigation (25). However, during exercise, TSI was *likely* maintained at a higher level in the ischemic preconditioning trial. Since TSI reflects the dynamic balance between O_2 supply and O_2 consumption, the greater TSI observed during the ischemic preconditioning trial is indicative of an improved O_2 delivery at the muscle level

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

(11). This may explain the maintenance of power output in the latter sprints in the ischemic preconditioning condition, despite the higher power outputs early in the trial and place the emphasis on greater O_2 delivery. It should be noted, however, that muscle oxygenation is not a limiting factor during repeated sprint activity (39). Instead, it may be that ischemic preconditioning increases blood flow to skeletal muscle (40), thereby improving power maintenance by increasing microvascular pressure, and/or by increasing metabolite washout (27). However, this mechanism is questionable given that blood flow returns to resting levels within 20 minutes of cuff release (7).

Previous research investigating ischemic preconditioning in exercise involving sprint activity has provided conflicting evidence. Elite swimming performance (100 m) is enhanced following ischemic preconditioning (24); however, the time taken to complete the event was ~66 s, thus not typical of a sprint experienced in team sports. Moreover, no effect of ischemic preconditioning has been demonstrated during 'all-out' sprint exercise at 130% $\dot{V}O_{2max}$ or 30 m land based sprint running (12, 19). Whilst these results differ from the ones in the current study they may be explained by the timing of the preconditioning strategy. Previous studies have performed a warm up immediately post the preconditioning stimulus and moved straight into the exercise regime (19). In the current study, the warm up was started 30 minutes post the ischemic preconditioning stimulus to make the research more applicable to an athletic setting. Current research investigating performance immediately after the ischemic preconditioning stimulus may be confined to a laboratory setting due to the impracticality of performing a similar action in an athletic event. It may be that the extra recovery time following the ischemic preconditioning stimulus is more beneficial for sprint related activity as demonstrated by the increased power output in the first three sprints. Due to the evidence in a controlled laboratory environment and protocol in the current study, future research

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

should focus on the mechanisms for improved performance and application of ischemic preconditioning in events which mimic actual performance events.

In conclusion, ischemic preconditioning of skeletal muscle *likely* increases both mean and peak power output in the first three sprints by 2-4% during the early stages of repeated sprint cycling. This was in contrast to our hypothesis that ischemic preconditioning would improve S_{dec} through aerobic metabolism. Moreover S_{dec} was not substantially different between trials, possibly due to maintenance of TSI in the ischemic preconditioning condition. Further research is required to establish the mechanisms for increased power output during repeated sprint cycling following ischemic preconditioning. Overall the results of this study suggest that ischemic preconditioning is a potential aid for improving sprint based performance.

Acknowledgements

The authors thank all the participants who volunteered for this study.

Conflict of Interest

The authors have no conflicts of interest that are relevant to the content of this article. The results of the present study do not constitute endorsement by ACSM

- The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

330

331

332 **REFERENCES**

333 1. Andreas M, Schmid AI, Keilani M, Doberer D, Bartko J, Crevenna R, Moser E, Wolzt
334 M. (2011). Effect of ischemic preconditioning in skeletal muscle measured by
335 functional magnetic resonance imaging and spectroscopy: a randomized crossover
336 trial. J Cardiovasc Magn Reson. 2011; 13(1): 32
337 2. Argus CK, Driller MW, Ebert TR, Martin DT, Halson SL. The effects of 4 different
338 recovery strategies on repeat sprint-cycling performance. *Int J Sports Physiol*
339 *Perform*. 2013; 8(5): 542-548.
340 3. Bailey TG, Jones H, Gregson W, Atkinson G, Cable NT, Thijssen DH. Effect of
341 ischemic preconditioning on lactate accumulation and running performance. *Med Sci*
342 *Sports Exerc*. 2012; 44(11): 2084-2089.
343 4. Balsom PD, Ekblom B, Sjodin B. Enhanced oxygen availability during high intensity
344 intermittent exercise decreases anaerobic metabolite concentrations in blood. *Acta*
345 *Physiol Scand*. 1994; 150: 455-456.
346 5. Balsom PD, Gaitanos GC, Ekblom B, Sjodin B. Reduced oxygen availability during
347 high intensity intermittent exercise impairs performance. *Acta Physiol Scand.* 1994;
348 152: 279-285.
349 6. Bangsbo J, Krustrup P, González-Alonso J, Saltin B. ATP production and efficiency
350 of human skeletal muscle during intense exercise: effect of previous exercise. *Am J*
351 *Physiol Endocrinol Metab*, 2001; 280: 956-964.
352 7. Barbosa TC, Machado AC, Braz ID, Fernandes IA, Vianna LC, Nobrega AC, Silva
353 BM. Remote ischemic preconditioning delays fatigue development during handgrip

354 exercise. *Scand J Med Sci Sports*. 2014; 15. doi: 10.1111/sms.12229. [Epub ahead of
355 print].
356 8. Batterham AM, Hopkins W G. Making meaningful inferences about magnitudes. *Int
357 J Sports Physiol Performance* 2006; 3(4): 547-557
358 9. Bogdanis GC, Nevill ME, Boobis LH, Lakomy HK. Contribution of phosphocreatine
359 and aerobic metabolism to energy supply during repeated sprint exercise. *J Appl
360 Physiol*. 1996; 80(3): 876- 884.
361 10. Buchheit M , Bishop D , Haydar B, Nakamura FY, Ahmaidi S. Physiological
362 Responses to Shuttle Repeated-Sprint Running. *Int J Sports Med.* 2010; 31(6): 402-
363 409.
364 11. Buchheit M, Ufland P. Effect of endurance training on performance and muscle
365 reoxygenation rate during repeated-sprint running. *Eur J Appl Physiol.* 2011; 111(2):
366 293-301.
367 12. Crisafulli A, Tangianu F, Tocco F, Concu A, Mameli O, Mulliri G, Caria MA.
368 Ischemic preconditioning of the muscle improves maximal exercise performance but
369 not maximal oxygen uptake in humans. *J Appl Physiol*. 2011; 111(2): 530-6.
370 13. de Groot PC, Thijssen DH, Sanchez M, Ellenkamp R, Hopman MT. Ischemic
371 preconditioning improves maximal performance in humans. *Eur J Appl Physiol*. 2010;
372 108(1): 141-6.
373 14. Dupont G, Millet GP, Guinhouya C, Berthoin S. Relationship between oxygen uptake
374 kinetics and performance in repeated running sprints. *Eur J Appl Physiol* 2005; 95(1):
375 27-34.
376 15. Faulkner SH, Ferguson RA, Gerrett N, Hupperets M, Hodder SG, Havenith G
377 Reducing muscle temperature drop after warm-up improves sprint cycling
378 performance. *Med Sci Sports Exerc*. 2013; 45(2): 359-365.

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

16. Foster J, Taylor L, Chrismas BC, Watkins SL, Mauger AR . The influence of acetaminophen on repeated sprint cycling performance. *Eur J Appl Physiol*. 2014; 114(1): 41-8.
17. Freriks B, Hermens H, Disselhorst-Klug C, Rau G. The recommendations for sensors and sensor placement procedures for surface electromyography. In: Hermens H, Freriks B, Merletti R, Stegeman D, Blok J, Rau G, et al., editors. European recommendations for surface electromyography. Enschede (The Netherlands): Roessingh Research and Development BV; 1999. p. 15–54.
18. Gaitanos GC, Williams C, Boobis LH, Brooks S. Human muscle metabolism during intermittent maximal exercise. *J Appl Physiol*.1993; 75: 712–719.
19. Gibson N, White J, Neish M, Murray A. Effect of ischemic preconditioning on land-based sprinting in team-sport athletes. *Int J Sports Physiol Perform*. 2013; 8(6): 671-676.
20. Glaister M, Stone MH, Stewart AM, Hughes M, Moir GL. The reliability and validity of fatigue measures during short-duration maximal-intensity intermittent cycling. *J Strength Cond Res*. 2004; 18(3): 459–62.
21. Gürke L, Marx A, Sutter PM, Frentzel A, Salm T, Harder F, Seelig J, Heberer M. Ischemic preconditioning improves postischemic skeletal muscle function. *Am. Surg.* 1996; 62: 391– 394.
22. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc*. 2009; 41(1): 3–13.
23. Janier MF,Vanoverschelde JL, Bergmann SR. Ischemic preconditioning stimulates anaerobic glycolysis in the isolated rabbit heart. *Am J Physiol*. 1994; 267(4):H1353-60.

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

403 24. Jean-St-Michel E, Manlhiot C, Li J, Tropak M, Michelsen MM, Schmidt MR,
404 McCrindle BW, Wells GD, Redington AN. Remote preconditioning improves
405 maximal performance in highly trained athletes. *Med Sci Sports Exerc*. 2011; 43(7):
406 1280-1286.
407 25. Kjeld T, Rasmussen MR, Jattu T, Nielsen HB, Secher NH (2014). Ischemic
408 preconditioning of one forearm enhances static and dynamic apnea. *Med Sci Sports*
409 *Exerc*. 2014; 46(1):151-155.
410 26. Lawson CS, Downey JM. Preconditioning: state of the art myocardial protection.
411 *Cardiovasc Res*. 1993; 27(4): 542-50.
412 27. Libonati JR, Howell AK, Incanno NM, Pettee KK, Glassberg HL. Brief muscle
413 hypoperfusion/hyperemia: an ergogenic aid? *J Strength Cond Res*. 2001; 15(3): 362-6.
414 28. Loukogeorgakis SP, Panagiotidou AT, Broadhead MW, Donald A, Deanfield JE,
415 MacAllister RJ. Remote ischemic preconditioning provides early and late protection
416 against endothelial ischemia-reperfusion injury in humans: role of the autonomic
417 nervous system. *J Am Coll Cardiol*. 2005; 46(3): 450–456.
418 29. Lukes DJ, Lundgren A, Skogsberg U, Karlsson-Parra A, Soussi B, Olausson M.
419 Ischemic Preconditioning Can Overcome the Effect of Moderate to Severe Cold
420 Ischemia on Concordant Mouse Xeno–Heart Transplants. *Transplant Proc*. 2005;
421 37(8):3332-4.
422 30. McGawley K, Bishop DJ. Oxygen uptake during repeated-sprint exercise. *J Sci Med*
423 *Sport*. 2014 Feb 10. pii: S1440-2440(14)00030-9. doi: 0.1016/j.jsams.2014.02.002.
424 [Epub ahead of print.
425 31. Mendez-Villanueva A, Edge J,Suriano R, Hamer P, Bishop D. The Recovery of
426 Repeated-Sprint Exercise Is Associated with PCr Resynthesis, while Muscle pH and
427 EMG Amplitude Remain Depressed. *PLOS One*. 2012; 7(12): e51977

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

428 32. Murry, CE, Jennings RB, Reimer KA. A delay in lethal cell injury in ischemic
429 myocardium. *Circulation*. 1986; 74(5): 1124-1136.
430 33. Ng JKF, Richardson CA, Kippers V, Parnianpour M, Bui BH. Clinical applications of
431 power spectral analysis of electromyographic investigations in muscle tension.
432 *Manual Therapy*. 1996; 2: 99-103.

433 34. Paganelli W, Pendergast DR, Koness J, Cerretelli P. The effect of decreased muscle
434 energy stores on the VO2 kinetics at the onset of exercise. *Eur J Appl Physiol Occup*
435 *Physiol. 1989;* 59**:** 321–326

436 35. Pang CY, Yang RZ, Zhong A, Xu N, Boyd B, Forrest CR. Acute ischaemic
437 preconditioning protects against skeletal muscle infarction in the pig. *Cardiovasc Res.*
438 1995; 29(6): 782–788.
439 36. Phillips, D.J., Petrie, S.G., Zhou, B.-H., Guanche, C.A., and Baratta, R.V. Myoelectric
440 and mechanical changes elicited by ischemic preconditioning in the feline hindlimb.
441 *J. Electromyogr. Kinesiol*. 1997; 7(3): 187–192.
442 37. Rongen GA, van Dijk JP, van Ginneken EE, Stegeman DF, Smits P, Zwarts MJ
443 Repeated ischaemic isometric exercise increases muscle fibre conduction velocity in
444 humans: involvement of Na(+)-K(+)-ATPase, *J Physiol*. 2002; 540(3): 1071-8.
445 38. Saito T, Komiyama T, Aramoto H, Miyata T, Shigematsu H. Ischemic
446 Preconditioning Improves Oxygenation of Exercising Muscle in Vivo. *J Surg Res*.
447 2004; 120(1): 111-8.
448 *39.* Smith KJ, Billaut F. Influence of cerebral and muscle oxygenation on repeated-sprint
449 ability. *Eur J Appl Physiol*. 2010; 109(5): 989-99.

• The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

450 *40.* Wang WZ, Stepheson LL, Fang XH, Khiabani KT, Zamboni WA. Ischemic
451 preconditioning-induced microvascular protection at a distance. *J Reconstr Microsurg*
452 2004; 20: 175–181.

453

454

455

456

457

458

459

460

461

462

463

464

465

466

467

468

469

- The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

Figure Legends

Figure 1. Peak power output data during twelve maximal 6 s sprints following ischemic preconditioning (solid bars) or placebo (open bars). Data are mean ± SD. * indicates substantially different from placebo (> 75% likelihood).

Figure 2. Mean power output data during twelve maximal 6 s sprints following ischemic preconditioning (sold bars) or placebo (open bars). Data are mean ± SD. * indicates substantially different from placebo (> 75% likelihood).

Table Legends

Table 1. Statistical summary of the differences between ischemic preconditioning and placebo for oxygen uptake, tissue saturation index, EMG, and blood lactate.

- The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

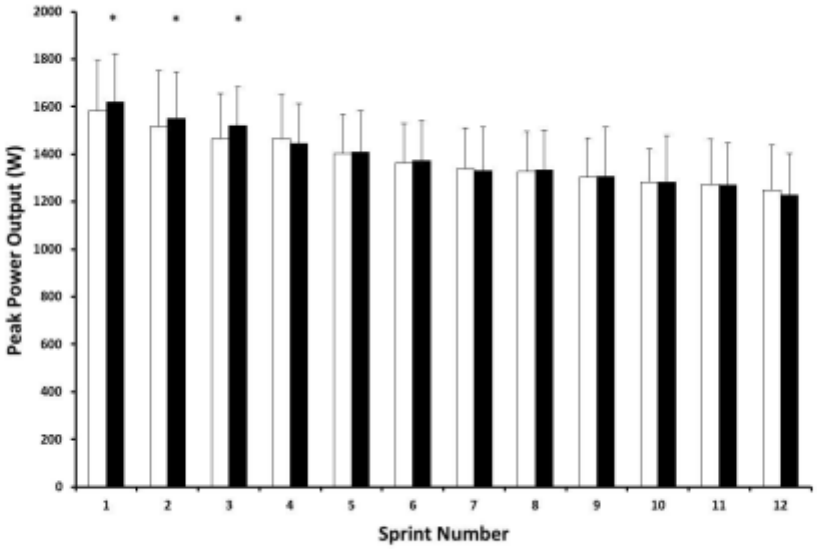

Figure 1.

- The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

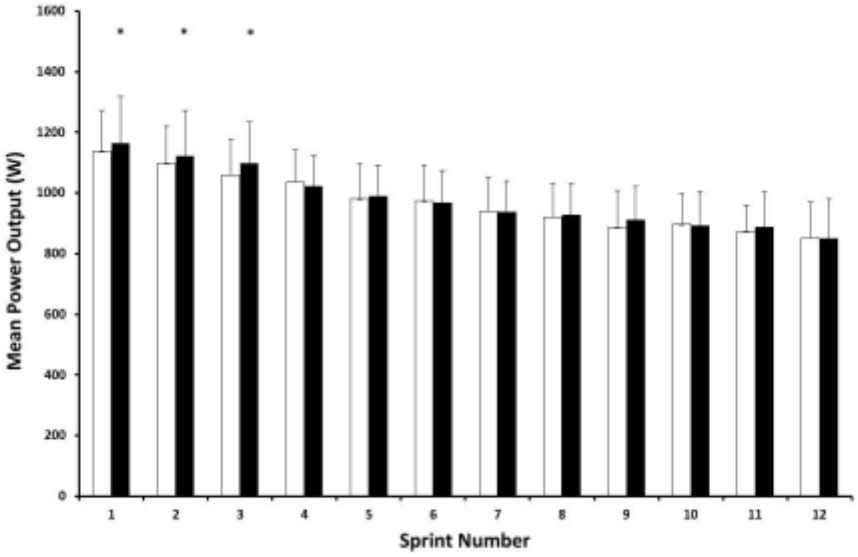

Figure 2.

- The is a non-final version of an article published in final form in Medicine and Science in Sport and Exercise (2015) Aug;47(8):1652-8. doi: 10.1249/MSS.0000000000000576

Table 1.

	Placebo	**Ischemic preconditioning**	**Mean Change[a]; ± 90% CI (%)**	**Qualitative Inference[b] (% Likelihood)**	**Effect Size (Qualitative Descriptor)**
$\dot{V}O_2$ Rest ($L.min^{-1}$)	0.4 ± 0.1	0.4 ± 0.1	1.9 ± 15.6	Unclear	0.08 (trivial)
$\dot{V}O_2$ Exercise ($L.min^{-1}$)	2.6 ± 0.3	2.7 ± 0.4	4.3 ± 7.3	Unclear	0.29 (small)
TSI Rest (%)	71.8 ± 5.1	73.0 ± 4.0	1.7 ± 3.6	Unclear	0.24 (small)
TSI Occlusion (%)	72.3 ± 5.5	58.0 ± 4.2	-19.7 ± 3.6	**Almost Certainly decreased (100%)**	2.77 (Large)
TSI Exercise (%)	57.7 ± 5.0	60.9 ± 6.0	5.4 ± 4.8	**Likely Increased (93%)**	0.56 (Moderate)
Rate of change in EMG MDF (Hz/sprint)	-0.04± 0.43	-0.28 ± 0.42	48.9 ± 69.7	Possibly Higher (73%)	0.37 (small)
Sprint 4 Blood Lactate ($mmol.L^{-1}$)	6.9 ± 2.1	7.5 ± 2.3	7.1 ± 11.4	Possibly Higher (50%)	0.19 (trivial)
Sprint 8 Blood Lactate ($mmol.L^{-1}$)	9.6 ± 2.8	10.2 ± 2.3	4.3 ± 6.4	Possibly Higher (25%)	0.12 (trivial)
Sprint 12 Blood Lactate ($mmol.L^{-1}$)	11.1 ± 3.5	11.8 ± 2.7	5.5 ± 6.4	Possibly Higher (26%)	0.13 (trivial)

90% CI = 90% confidence interval

a Mean change refers to ischemic preconditioning minus placebo trial.

b Inference about the magnitude of the effect

Bold inferences (% likelihood) indicate conditions with substantial change (> 75% likelihood).