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The effect of ischemic preconditioning on repeated sprint cycling performance.

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1 **Abstract**

2 **Purpose:** Ischemic preconditioning enhances exercise performance. We tested the hypothesis
3 that ischemic preconditioning would improve intermittent exercise in the form of a repeated
4 sprint test during cycling ergometry. **Methods:** In a single-blind, crossover study, fourteen
5 recreationally-active males (mean \pm SD; age 22.9 ± 3.7 years, height 1.80 ± 0.07 m, mass
6 77.3 ± 9.2 kg) performed twelve 6 s sprints following four 5 min periods of bilateral limb
7 occlusion at 220 mmHg (ischemic preconditioning) or 20 mmHg (placebo). **Results:**
8 Ischemic preconditioning resulted in a 2.4 ± 2.2 , 2.6 ± 2.7 and $3.7 \pm 2.4\%$ substantial increase
9 in peak power for sprints 1, 2 and 3 respectively, relative to placebo, with no further changes
10 between trials observed for any other sprint. Similar findings were observed in the first three
11 sprints for mean power output following ischemic preconditioning (2.8 ± 2.5 , 2.6 ± 2.5 and
12 $3.4 \pm 2.1\%$, for sprints 1, 2 and 3 respectively), relative to placebo. Fatigue index was not
13 substantially different between trials. At rest tissue saturation index was not different between
14 trials. During the ischemic preconditioning / placebo stimulus there was a $-19.7 \pm 3.6\%$
15 decrease in tissue saturation index in the ischemic preconditioning trial, relative to placebo.
16 During exercise there was a $5.4 \pm 4.8\%$ greater maintenance of tissue saturation index in the
17 ischemic preconditioning trial, relative to placebo. There were no substantial differences
18 between trials for blood lactate, electromyography (EMG) median frequency, oxygen uptake
19 or rating of perceived exertion (RPE) at any time points. **Conclusion:** Ischemic
20 preconditioning improved peak and mean power output during the early stages of repeated
21 sprint cycling and may be beneficial for sprint sports.

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

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24 **Introduction**

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

35 Research to date has primarily focussed on events of an endurance nature and has identified
36 improvements in peak oxygen uptake ($\dot{V}O_{2max}$; 13), power output at $\dot{V}O_{2max}$ (12), running
37 time trial performance (3), 1000 m rowing performance (25) and time to task failure (7)
38 following ischemic preconditioning. Relatively little research has focused on performance
39 during shorter durations and the findings are conflicting. For example, an improvement in
40 100 m swimming performance was observed in elite national level swimmers (24) but no
41 effect of ischemic preconditioning was demonstrated on single 30 m running sprint
42 performance (19) or cycling exercise at 130% $\dot{V}O_{2max}$ (12).

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

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

53 Ischemic preconditioning may improve aerobic metabolism as evidenced by increased
54 $\dot{V}O_{2\max}$ (13), accelerated $\dot{V}O_2$ kinetics (34) and improved oxygenation of skeletal muscle (38)
55 and it may therefore reduce the performance related decline in power output associated with
56 repeated sprint exercise. Secondly, in ischemic reperfusion injury models, ischemic
57 preconditioning enhances PCr resynthesis following ischemia (1, 29) and thus may enhance
58 the ability to recover between sprints. Therefore, the aim of this study was to investigate the
59 effect of ischemic preconditioning on repeated sprint cycling performance. Given the
60 apparent ability of ischemic preconditioning to improve aerobic metabolism and promote PCr
61 resynthesis it was hypothesized that it would improve repeated sprint cycling performance by
62 reducing the rate of fatigue.

63

64 **METHODS**

65 **Participants**

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

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

76 **Experimental Overview**

77 All participants reported to the laboratory for four exercise trials. In the initial trial,
78 data were obtained on individual anthropometric characteristics such as body mass, height
79 and four skinfolds (subscapular, biceps brachii, triceps brachii, and iliac crest). During this
80 trial participants were familiarised with the repeated sprint cycling protocol, consisting of
81 twelve 6 s cycle sprints with 30 s of passive recovery between each sprint. Trial 2 was a
82 repeat of the first, to further familiarise the participants with the exercise protocol. Trials 3
83 and 4 were the experimental trials which consisted of either ischemic preconditioning or
84 placebo treatment prior to the exercise protocol. The experimental trials were performed in a
85 counterbalanced manner, separated by 5-7 days to ensure no possible carryover of acute
86 ischemic preconditioning (28). During both trials respiratory gas exchange,
87 electromyography (EMG) of the vastus lateralis (VL) and near-infrared spectroscopy (NIRS)
88 of the VL were recorded. Participants indicated their rating of perceived exertion (RPE, 6 –
89 20; Borg's scale) and blood was taken from the earlobe at rest and following sprints 4, 8 and
90 12 before being subsequently analyzed for lactate. Participants performed all of their trials at
91 the same time of day (± 1 h) and laboratory conditions were controlled at approximately 20°C
92 and 38% relative humidity during all trials. Participants were instructed to maintain their
93 normal diet, to refrain from any form of intense physical activity and caffeine for the 24 h
94 period prior to testing, and not to eat for 3 h before each trial.

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95 **Experimental Measures**

96 *Ischemic Preconditioning*

97 In trials 3 and 4 exercise was preceded by ischemic preconditioning or placebo,
98 performed in a supine position using bilateral occlusion (3, 13). In the ischemic
99 preconditioning trial automatic occlusion cuffs (14.5 cm width - Delfi Medical Innovations,
100 Vancouver, Canada) were positioned proximally around the thigh and inflated to 220 mmHg
101 for 5 minutes followed by 5 minutes of reperfusion. This procedure was repeated four times
102 (3). The placebo trial was identical to the ischemic preconditioning trial except that the cuffs
103 were inflated to 20 mmHg. The time delay between the cuff removal and the beginning of the
104 warm up for the exercise test was 30 minutes as ischemic preconditioning has been
105 demonstrated to improve exercise performance within 45 minutes of the final cuff inflation
106 (3).

107 *Repeated-sprint cycling*

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

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

124

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

125 *Cardiorespiratory Measures*

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

136 *Muscle EMG*

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

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

157

158 *NIRS Measurements*

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

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

179 *Blood Lactate Measurement*

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

185 **Statistical Analysis**

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

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189 threshold value for the smallest meaningful change for mean and peak power output was set
190 as 0.8% (2). For all other data, the smallest worthwhile or important effect for each
191 dependent variable was the smallest standardised (Cohen) change in the mean: 0.2 times the
192 between-subject SD for baseline values of all participants (8). Qualitative descriptors were
193 assigned to the quantitative percentile scores as follows: 25–75% *possible*; 75–95% *likely*;
194 95–99% *very likely*; >99% *almost certain* (20). A substantial effect was set at > 75%. Effect
195 size was calculated using threshold values for Cohen's *d* statistics (0.2; small, 0.5; moderate
196 and 0.8; large). Data are presented as mean \pm SD or percent change from placebo ($\% \Delta \pm 90\%$
197 confidence interval ($\pm 90\%$ CI)), percent likelihood that the difference between conditions
198 was larger or smaller (% likelihood) and effect size). An effect was deemed unclear if its
199 confidence limits overlapped the thresholds for both the smallest beneficial and the smallest
200 harmful effect, that is, if the effect could be substantially positive and negative.

201

202 **RESULTS**

203 The maximal peak power (mean \pm SD) obtained during the repeated sprint cycling test was
204 1594 ± 208 and 1630 ± 192 W for placebo and ischemic preconditioning, respectively.
205 Qualitative analysis revealed that performing ischemic preconditioning before sprint activity
206 led to a *likely* increase in maximum peak power output ($2.5 \pm 1.9\%$, 93%, small ($\% \Delta$, %
207 likelihood, effect size)). Raw peak and mean power output data for each sprint are presented
208 in Figures 1 and 2, respectively. Ischemic preconditioning, relative to placebo, resulted in
209 substantial increases in peak power output for sprints 1 ($2.4 \pm 2.2\%$, 89% *likely*, small), 2 (2.6
210 $\pm 2.7\%$, 87% *likely*, small) and 3 ($3.7 \pm 2.4\%$, 97% *very likely*, small) only, with effects
211 unclear for the remaining sprints. Mean power output followed a similar pattern with
212 substantial increases in sprints 1 ($2.8 \pm 2.5\%$, 91% *likely*, small), 2 ($2.6 \pm 2.5\%$, 88% *likely*,

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

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

233 **DISCUSSION**

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

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

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

252 It is recognized that high energy compounds are important for energy production during
253 repeated sprint activity, with total anaerobic contributions of ATP production during a single
254 6 s sprint being 6%, 50% and 44% from ATP, PCr and anaerobic glycolysis, respectively
255 (18). Whilst speculative, it is possible that the increased power production in the first three
256 sprints in the ischemic preconditioning trial may have been a result of increased ATP
257 production from anaerobic sources. Following ischemic reperfusion injury ATP content is
258 maintained in rabbit and mice heart muscle as a result of ischemic preconditioning via
259 increased concentration of PCr and PCr / ATP ratio (29) or increased anaerobic glycolysis
260 (23). To date little evidence is available on concentrations in skeletal muscle, however

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

267 Originally, it was hypothesised that ischemic preconditioning would improve aerobic
268 metabolism and thus improve the ability to recover between sprints. Markers of aerobic
269 fitness such as $\dot{V}\text{O}_{2\text{max}}$ and $\dot{V}\text{O}_2$ kinetic parameters are related to the ability to offset fatigue
270 during a repeated sprint effort (14, 30), whilst an increased aerobic energy production
271 contributes towards power production in the latter stages of repeated intense exercise (6, 18,
272 30). Previous research employing one bout of circulatory occlusion prior to the start of an
273 exercise bout has demonstrated accelerated pulmonary $\dot{V}\text{O}_2$ kinetics (34). Moreover ischemic
274 preconditioning has been shown to increase $\dot{V}\text{O}_{2\text{max}}$ (13), suggesting that the method may be
275 used to help maintain power output during repeated sprint exercise, via improved PCr
276 resynthesis (9, 18, 34). However, data from the present study does not support this theory as
277 evidenced by the similarity between trials for $\dot{V}\text{O}_2$ during the repeated sprint protocol.

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

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285 (11). This may explain the maintenance of power output in the latter sprints in the ischemic
286 preconditioning condition, despite the higher power outputs early in the trial and place the
287 emphasis on greater O₂ delivery. It should be noted, however, that muscle oxygenation is not
288 a limiting factor during repeated sprint activity (39). Instead, it may be that ischemic
289 preconditioning increases blood flow to skeletal muscle (40), thereby improving power
290 maintenance by increasing microvascular pressure, and/or by increasing metabolite washout
291 (27). However, this mechanism is questionable given that blood flow returns to resting levels
292 within 20 minutes of cuff release (7).

293 Previous research investigating ischemic preconditioning in exercise involving sprint activity
294 has provided conflicting evidence. Elite swimming performance (100 m) is enhanced
295 following ischemic preconditioning (24); however, the time taken to complete the event was
296 ~66 s, thus not typical of a sprint experienced in team sports. Moreover, no effect of ischemic
297 preconditioning has been demonstrated during ‘all-out’ sprint exercise at 130% $\dot{V}O_{2max}$ or 30
298 m land based sprint running (12, 19). Whilst these results differ from the ones in the current
299 study they may be explained by the timing of the preconditioning strategy. Previous studies
300 have performed a warm up immediately post the preconditioning stimulus and moved straight
301 into the exercise regime (19). In the current study, the warm up was started 30 minutes post
302 the ischemic preconditioning stimulus to make the research more applicable to an athletic
303 setting. Current research investigating performance immediately after the ischemic
304 preconditioning stimulus may be confined to a laboratory setting due to the impracticality of
305 performing a similar action in an athletic event. It may be that the extra recovery time
306 following the ischemic preconditioning stimulus is more beneficial for sprint related activity
307 as demonstrated by the increased power output in the first three sprints. Due to the evidence
308 in a controlled laboratory environment and protocol in the current study, future research

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

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

319

320 **Acknowledgements**

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

322

323 **Conflict of Interest**

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

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472 **Figure Legends**

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

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

479 **Table Legends**

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

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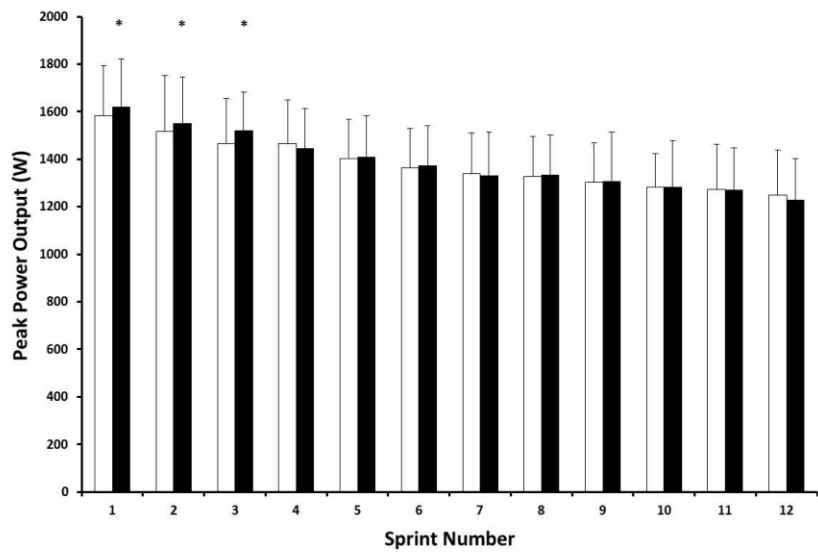


Figure 1.

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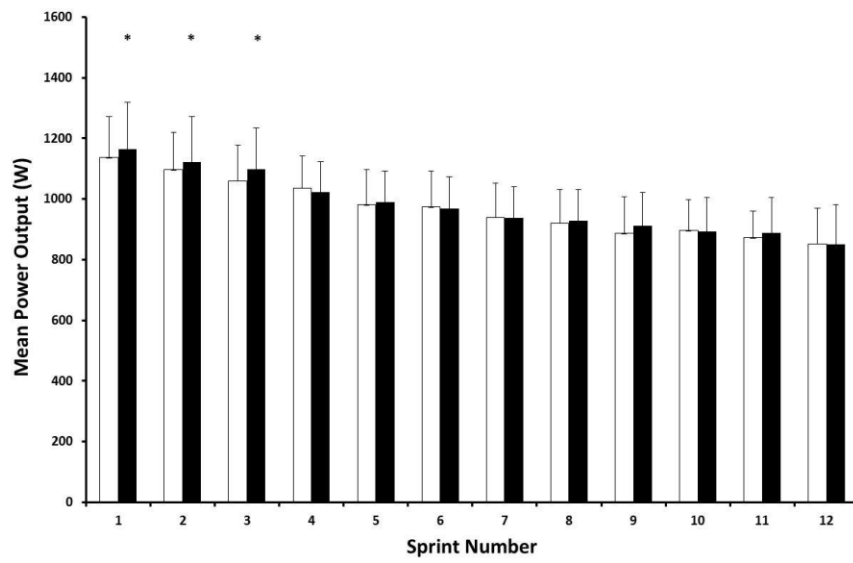


Figure 2.

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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).