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Effect of ischemic preconditioning on repeated sprint ability in team sport athletes

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1 **Title:** Effect of Ischemic Preconditioning on repeated sprint ability in team sport athletes.

2

3 **Running Title:** Ischemic preconditioning and repeated sprinting.

4

5 **Keywords:** Preconditioning, Team Sports, Repeated sprint ability, Power

6

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63 Abstract

64 This study investigated whether ischemic preconditioning in a trained population affected
65 repeated sprint performance. A secondary aim was to assess responses according to gender.
66 Sixteen (nine females and seven males) well trained team sport athletes took part in a
67 randomised crossover study design. Participants underwent an ischemic preconditioning and
68 placebo treatment involving three periods of 5 min occlusion applied unilaterally (3 x 5 min
69 occlusion to each leg) at either 220 mmHg or 50 mmHg respectively. Each period of
70 occlusion was followed by 5 min reperfusion. Following treatment 5 x 6 s maximal effort
71 sprints were undertaken on a cycle ergometer against 7.5 % body mass, each interspersed by
72 24 s recovery. Measured parameters included peak power, total power, percentage
73 decrement, post exercise blood lactate and ratings of perceived exertion. No within subject
74 main effect for ischemic preconditioning was observed, neither was there an interaction effect
75 with gender. Effect sizes were trivial ($ES < 0.2$) with the exception of a moderate ($ES < 1.2$)
76 change in post exercise blood lactate in the female cohort ($1.6 \pm 0.4 \text{ mmol}^{-1}$ lower following
77 IPC). Results suggest no benefit to team sport players in utilising ischemic preconditioning as
78 a means of enhancing repeated sprint performance. A lower blood lactate response in female
79 participants following ischemic preconditioning may suggest improved blood flow through
80 vasodilation.

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100 Since early research investigating the effect of ischemic preconditioning on the myocardium
101 the beneficial effect of the treatment has been observed in different tissues within the body,
102 including skeletal muscle (de Groot, Thijssen, Sanchez, Ellenkamp, Hopman, 2010; Crisafulli
103 et al., 2011; Beaven, Cook, Kilduff, Drawer, Gill, 2012; Jean-St-Michael et al., 2011).
104 Research investigating the effect of ischemic preconditioning on exercise has included
105 measures of aerobic capacity (de Groot et al., 2010; Crisafulli et al., 2011), speed (Gibson,
106 White, Neish, Murray, 2013) and recovery following strength and power tasks (Beaven et al.,
107 2012). Data from these studies is equivocal with the suggestion that a pattern of responders
108 and non-responders may exist and that the intervention may be less effective in female
109 populations (Beaven et al., 2012; Gibson et al., 2013).

110 The ability of ischemic preconditioning to exert a beneficial effect on exercise and recovery
111 thereafter appears to be dependent on metabolites, including bradykinin, opioids and
112 adenosine, reaching a critical level (Downey, Davis, Cohen, 2007). Inhibition of one of these
113 metabolites removed the beneficial effect of a single bout of ischemic preconditioning
114 suggesting the existence of a threshold below which occlusion may prove ineffective (Goto et
115 al. 1995). Adopting multiple episodes of ischemic preconditioning has been advocated
116 within the literature to ensure such a threshold is met (de Groot et al., 2010, Crisafulli et al.,
117 2011, Jean-St-Michael et al., 2011).

118 Much of the research to date has focused on the usefulness of ischemic preconditioning on
119 exercise tasks of an endurance nature with positive effects reported for total work, power,
120 exercise time (Crisafulli et al., 2011) and $\dot{V}O_2$ max (de Groot et al., 2010). The effect of
121 ischemic preconditioning on activities of a shorter and more anaerobic nature is less clear: a
122 significant improvement has been reported in elite level swimmers over a distance of 100m
123 (Jean-St-Michael et al., 2011) whilst no improvement was noted when cyclists used the
124 intervention prior to performing supra-maximal efforts (Crisafulli et al., 2011). It should be
125 noted however that in both these studies exercise time was in excess of 60s, considerably
126 longer than typical anaerobic efforts observed in team sport environments (Spender, Bishop,
127 Dawson & Goodman, 2005). When utilised prior to land based sprint activities no effect was
128 reported (Gibson et al., 2013) however the intervention was found to be beneficial on
129 measures of acute and chronic recovery when used between tasks requiring maximal force
130 generation (Beaven et al., 2012). This may in part be due to enhanced muscle recruitment via
131 a desensitizing of the afferent groups III and IV, increased neural drive and force output
132 (Noakes, 2011) Interestingly, it has been suggested that ischemic preconditioning is less
133 beneficial in female populations (Beaven et al., 2012; Gibson et al., 2013)

134 Whilst a number of individual events are characterised by short but intense single efforts,
135 team invasion sports require the performance of multiple bouts of maximal and at times
136 supra-maximal activity (Spencer et al., 2005; Gabbett, 2012; Dwyer & Gabbett, 2012;
137 Impellizeri et al., 2006; Gabbett, 2009). The importance of these repeated sprint efforts to
138 successful performance has been illustrated within rugby league (Gabbett, 2012). Given that
139 ischemic preconditioning has been shown to exert a beneficial effect on performance in high
140 intensity tasks when administered 45 minutes before competition (Jean-St-Michael et al.,
141 2011) its use within the warm up prior to team sports would seem plausible. Ischemic
142 preconditioning has been evidenced to enhance vasodilation, oxygen delivery and ATP
143 sparing (Beaven et al., 2012; Liu et al., 1991; Jennings, Sebbag, Schwartz, Crago & Reimer,
144 2001), adaptations similar to those that could be expected following endurance training.
145 With this in mind a positive effect on repeated sprint activities may be expected given the

146 large aerobic component that exists in exercise of this nature (Dupont, McCall, Prieur, Millet
147 & Berthoin, 2013; Bucheit & Laursen, 2013). The current investigation is designed to assess
148 whether ischemic preconditioning exerts a beneficial effect when used prior to repeated sprint
149 activity performed on a cycle ergometer. Given the large aerobic component that is
150 associated with exercise of this nature it is hypothesised that enhanced oxygen delivery,
151 facilitated through adenosine mediated vasodilation and/or ATP sparing, will provide a
152 beneficial effect on performance. A secondary aim is to compare responses between gender
153 groups to further investigate the assertion that ischemic preconditioning may be less suitable
154 for female populations (Beaven et al., 2012; Gibson et al., 2013)

155 **Methods**

156 **Participants and design**

157 Sixteen participants (7 males and 9 females) volunteered to take part in the study, all with a
158 recognised competition history within the invasion sports of Soccer, Field Hockey and Rugby
159 Union. Mean age, stature and body mass are presented in table I. All participants signed an
160 informed consent document and the study received institutional ethical approval conforming
161 to the code of ethics of the World Medical Association (Declaration of Helsinki).

162 A counterbalanced randomised crossover design was used to assess the impact of a brief
163 period of remote ischemic preconditioning on repeated sprint performance under two separate
164 conditions, experimental and placebo. All participants undertook a prior control with no
165 treatment. In both placebo and ischemic preconditioning trials participants were fitted with a
166 blood pressure cuff positioned around the upper thigh and inflated to 50 mmHg or 220
167 mmHg respectively. Following treatment participants followed a standardised warm up
168 followed by 5 x 6 s sprints against an external load of 7.5 % body mass. Measured variables
169 were peak power, relative peak power (Watts per kilogram body mass), total power,
170 percentage decrement, post assessment blood lactate and ratings of perceived exertion (RPE).

171 ***INSERT TABLE 1 NEAR HERE***

172 **Baseline assessment and control**

173 All participants were required to visit the laboratory on three separate occasions each no more
174 than seven days apart for the collection of control, placebo and experimental data. On their
175 first visit participants' age, stature and body mass were recorded along with a measure of
176 resting blood pressure (Omron RX-3, Kyoto – Japan). Any participants presenting with a
177 blood pressure higher than 140/100 mmHg (systolic/diastolic) were precluded from taking
178 part in the study. These guidelines were in line with ethical approval of the study. Prior to
179 data collection the cycle ergometer (MonarkErgomedic 814c, Stockholm, Sweden) was
180 calibrated according to the manufacturers guidelines and configured to suit the participants
181 preferred cycling position. Participants completed a standardised warm up consisting of five
182 minutes stationary cycling at 60 rpm and against 1kg of external resistance. This was
183 followed by two 3s sprints separated by 60 s to habituate themselves with the requirements of
184 the assessment. The repeated sprint assessment required the performance of 5 x 6 s sprints
185 against 7.5 % of body mass, each separated by a recovery period of 24 s, a protocol used
186 previously in team sport athletes (Blee, Goodman, Dawson & Stapff, 1999; Bishop, Spencer,
187 Duffield & Lawrence, 2001). This protocol was chosen to limit the impact of pacing and
188 provide a sufficient number of sprints for the accurate and reliable assessment of peak power
189 and percentage decrement (Hachana, Attia, Nassib & Shephard, 2012). Blood lactate
190 samples were collected three minutes post the fifth and final sprint using a Lactate Pro

191 analyser (ArkrayInc, Kyoto, Japan. CV 5.66%). RPE data was collected following each of
192 the five sprints. Data from the repeated sprint protocol was collected and analysed using
193 specific software (Cranlea Wingate Software version 3). Following the collection of control
194 data all participants underwent both placebo and experimental conditions in a randomised
195 counterbalanced fashion.

196 Placebo trial

197 On arrival at the laboratory participants had their blood pressure measured as described above
198 to screen for any contraindications to the experimental procedure. Participants were then
199 instructed to adopt a semi-recumbent position on a medical plinth with both legs outstretched.
200 A blood pressure cuff (Boso-roid I aneroid sphygmomanometer, Bosch and Son, Germany)
201 was positioned around the upper thigh, distal to the inguinal fold. For placebo treatment the
202 cuff was inflated by hand to 50mmHg. Each leg was exposed to 5 min of pressure followed
203 by 5min of reperfusion for three consecutive cycles eliciting a total treatment time of 30 min.
204 During reperfusion the contralateral leg was fitted with the cuff and inflated to 50 mmHg in
205 accordance with the protocol for ischemic preconditioning administration described
206 elsewhere (Gibson et al., 2013). During the treatment participants were asked at regular
207 intervals (every minute) to confirm they were able to continue with the protocol. Any
208 participant indicating light headedness, nausea or discomfort had the pressure cuff removed
209 and were omitted from the study (n = 0). Following the final 5 min of reperfusion the
210 participant was supported whilst they stepped down from the plinth and given a moment to
211 ensure they were steady on their feet before commencing the standardised warm up detailed
212 above. The time delay between removing the pressure cuff and commencing the warm up
213 was five minutes.

214 Ischemic preconditioning treatment

215 The ischemic preconditioning treatment followed an identical format to that of the protocol as
216 described above however the blood pressure cuff was inflated to 220 mmHg which has been
217 shown to elicit ischemia by occluding arterial blood flow to the lower legs (Koojiman et al.,
218 2008).

219 Statistical analysis

220 Data was checked for homogeneity of variance using Lavene's test and did not violate the
221 assumption of sphericity using Mauchly's test. All results were non-significant ($P<0.05$) and
222 as such deemed appropriate for parametric analysis. Data were analysed using SPSS for
223 windows (PASW statistics 17.0) and a 3 x 2 mixed factorial ANOVA with significance
224 calculated at $P<0.05$. Due to the practical nature of the investigation effect sizes (Cohen's d)
225 were also used. Effect sizes of <0.2 , <0.6 , <1.2 , <2.0 and >2 were considered trivial, small,
226 moderate, large and very large respectively (Hopkins, Marshall, Batterham & Hanin, 2009).

227 **Results**

228 Table I details the physical characteristics of participants included in the study as a pooled
229 cohort and separated by gender groups. Table II details means \pm SD's for performance
230 variables calculated during the repeated sprint protocol along with corresponding effect sizes
231 comparing control with ischemic preconditioning trials. No significant main effect or
232 interaction with gender was observed for occlusion on peak power or relative peak power
233 ($P>0.05$) as shown in figure 1. Calculated effect sizes were classified as trivial (ES <0.2) for
234 peak power, total power and relative peak power. Within the female cohort a small effect

235 size was detected for percentage decrement whilst for the blood lactate response effect sizes
236 of small and moderate magnitude were calculated for the male and female cohort respectively
237 as shown in figure 2.

238 ***INSERT TABLE II NEAR HERE***

239 ***INSERT FIGURE I NEAR HERE***

240 ***INSERT FIGURE II NEAR HERE***

241 **Discussion**

242 Data collected in the current investigation suggest ischemic preconditioning to exert neither a
243 beneficial nor deleterious effect on absolute and relative peak power, total power or
244 percentage decrement during a repeated sprint protocol. Unlike previously reported data
245 (Beaven et al., 2012; Gibson et al., 2013) there appears to be no difference in response to the
246 intervention when compared by gender group with the exception of post exercise blood
247 lactate. Results would suggest that for events requiring short (<6 s) maximal efforts ischemic
248 preconditioning is not a suitable pre exercise intervention for performance enhancement. The
249 finding that post exercise blood lactate levels may be reduced, especially in a female cohort,
250 combined with non-significant changes in total power and percentage decrement warrants
251 further investigation into the effect of ischemic preconditioning on activities requiring
252 repeated forceful yet sub-maximal efforts, such as those occurring in team sports (Spencer et
253 al., 2005; Gabbett, 2012; Dwyer & Gabbett, 2012; Impellizeri et al., 2006; Gabbett, 2012).
254 The ability to produce similar amounts of work whilst attenuating the production of lactate
255 and/or augmenting its clearance may suggest the intervention to facilitate a greater
256 contribution from aerobic pathways and the sparing of ATP generated via glycolysis (Bailey
257 et al., 2012).

258 Equivocal results are apparent in response to power output following ischemic
259 preconditioning administration. When used as a recovery modality following activities
260 requiring high power output ischemic preconditioning was shown to attenuate reductions in
261 performance, both acutely and chronically (Beaven et al, 2012). In a study conducted with
262 international level swimmers the intervention was shown to improve performance by 0.7 s, a
263 change paralleled with an increased stroke count. This change may be interpreted as being
264 the result of less force exerted per stroke (Jean-St-Michael, 2011). The event duration (~60
265 s) may have provided sufficient time for any decrement in initial peak power following
266 ischemic preconditioning administration to be compensated for by a higher sustained average
267 power in the latter stages of the race. It should be noted that in the present study ischemic
268 preconditioning exerted no significant effect on average, total or peak power. In cyclists
269 exercising supra-maximally for approximately 120 s no beneficial effect of ischemic
270 preconditioning was realised in terms of exercise time or power output (Crisafulli et al.,
271 2011). In studies examining the effect of ischemic preconditioning on swimmers and cyclists
272 however exercise duration was substantially longer than that which has been reported for
273 team sports (Spencer et al., 2005), a sporting population from which the current cohort was
274 drawn. Considering the present study's findings and those reported when ischemic
275 preconditioning was used prior to short land based sprinting (<5 s) (Gibson et al., 2013) there
276 appears to be evidence that would support the existence of a threshold in exercise duration
277 below which the intervention has no effect on performance.

278 A mechanism postulated for the beneficial effect of ischemic preconditioning on exercise is
279 increased blood flow and oxygen delivery to the working musculature via adenosine

280 mediated vasodilation (Beaven et al., 2012; Liu, 1991). There is also evidence of ATP
281 preservation, albeit in canine models (Jennings et al., 2001). In the current investigation no
282 changes in performance following ischemic preconditioning with respect to percentage
283 decrement were reported however moderate effect sizes were measured for post exercise
284 blood lactate in the female cohort with lower values reported following IPC. Lower blood
285 lactate levels following exercise preceded by ischemic preconditioning have been reported
286 elsewhere (Bailey et al., 2012). Following 5 x 3 minute stages of incremental treadmill
287 running ranging from 10 to 14 km.h⁻¹ blood lactate was observed to be 1.07 ± 0.11 mmol⁻¹
288 lower when ischemic preconditioning preceded exercise. In the present study repeated
289 sprints preceded by ischemic preconditioning were shown to illicit a blood lactate response
290 1.6 ± 0.4 mmol⁻¹ lower than control within the female cohort and a corresponding moderate
291 effect size. This reduction was paralleled by non-significant changes in peak power, total
292 power and percentage decrement. It is suggested that future research includes a greater
293 number of sampling points to more fully explain lactate kinetics following exercise preceded
294 by ischemic preconditioning.

295 Whilst improvements in aerobic capacity have been associated with reduced blood lactate
296 following sub-maximal exercise of a given workload (Lorenzo, Minson, Babb & Halliwell,
297 2011) this mechanism is unlikely to explain changes in hematology during the present study.
298 An alternative hypothesis that ischemic preconditioning mimics some of the chronic
299 changes associated with training and its associated improvements in aerobic capacity may be
300 postulated. These include but are possibly not limited to, vasodilation and the associated
301 increases in blood flow that facilitate energy provision via aerobic pathways, sparing of ATP
302 derived from anaerobic glycolytic pathways and potential augmentation of blood lactate
303 clearance. Previous results have shown that the provision of energy via aerobic pathways
304 increases during repeated sprint exercise to compensate for the reduction in glycolysis
305 (Bailey et al., 2012). A strong relationship has also been shown between repeated sprint
306 performance and aerobic capacity (Dupont et al., 2010; Bishop et al., 2004) characterised by
307 an enhanced ability for energy provision via increased capillarisation, blood flow and
308 mitochondrial density. Indeed it was augmentation of blood flow which was suggested as a
309 potential mechanism for ischemic preconditioning providing a beneficial stimulus when used
310 as a recovery modality following tasks requiring maximal force generation (Beaven et al.,
311 2012). Reductions in blood lactate following the use of ischemic preconditioning may be a
312 result of enhanced energy provision via aerobic pathways, ATP sparing and/or enhanced
313 oxidation and clearance rates. Such mechanisms would lend support to the contention that
314 during repeated sprint activities where energy derived from anaerobic processes is
315 compromised as a result of intensity, duration or insufficient between effort recovery,
316 ischemic preconditioning may be beneficial to performance.

317 It was hypothesised that ischemic preconditioning would result in a greater decrement in
318 performance within the female cohort, something that was not evident in the results.
319 Previous studies have shown the intervention to negatively affect performance in female
320 athletes when used prior to land based sprinting activities and as a recovery tool following
321 strength and power activities (Beaven et al., 2012; Gibson et al., 2013). Individual variations
322 in thigh circumference, muscle mass and limb composition and the corresponding level of
323 occlusion caused at an absolute pressure of 220 mmHg (Dempsey & Wagner, 1999) have
324 been cited as potential causes of this discrepancy, along with the perception of discomfort
325 associated with the intervention. RPE data collected in the present study would not support
326 the contention that a greater perception of effort and/or discomfort was associated with
327 ischemic preconditioning in male or female participants. It is acknowledged however that

328 given the relatively low participant numbers in the present study drawing firm conclusions
329 regarding differences that may exist between gender is difficult.

330 Time motion analysis in team sports has suggested mean sprint durations to be between 2-3 s
331 for elite level Soccer, Field Hockey and Australian Rules Football rising to 4.1 ± 1.1 s when
332 mean maximal sprint duration is considered (Spencer et al., 2005). As such the protocol in
333 the present study characterised by 5 x 6 s sprints has been suggested to be representative of
334 field based invasion game activity. For sports that incorporate short (<1 s) accelerative efforts
335 requiring high force output results from the present study would suggest ischemic
336 preconditioning to be an inappropriate pre exercise intervention. If however the sport is more
337 reliant on running sprints (Impellizeri et al., 2006) characterised by high running speeds over
338 longer durations and potentially less forceful accelerations (Gabbett, 2012), the use of
339 ischemic preconditioning may be warranted given non-significant changes in percentage
340 decrement and lower post exercise blood lactate levels in the female cohort. Future research
341 should focus on investigating the effectiveness of IPC as a precursor to land based repeated
342 sprint activities and/or sport specific simulation protocols (Twist & Sykes, 2011)

343 **Conclusion**

344 Ischemic preconditioning exhibited no beneficial effect on markers of performance associated
345 with repeated sprinting characterised by 5 x 6 s efforts, including total power, peak power
346 and relative peak power. Additionally there appears to be no difference in response between
347 gender groups following the intervention as has been reported for single sprint activity and
348 recovery. Interestingly however a moderate reduction in post exercise blood lactate
349 following ischemic preconditioning in the female cohort was observed. This finding may
350 suggest that for repeated sprint protocols of a longer duration, or those involving actions that
351 more closely mimic the demands of team sports, such as collisions or changes of direction
352 ischemic preconditioning may be beneficial to markers of performance.

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497 **Table 1.** Means \pm SD for physical characteristics of participants as a pooled cohort and
498 separated by gender.
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Physical characteristic	Participants (n = 16)	Female (n = 9)	Males (n = 7)
Age (years)	24.1 \pm 2.6	24.0 \pm 3.71	24.2 \pm 1.6
Stature (cm)	174.0 \pm 6.1	171.1 \pm 4.4	177.6 \pm 6.2
Mass (kg)	73.7 \pm 11.8	67.6 \pm 7.1	81.4 \pm 12.5

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541 **Table 2.** Means \pm SD for peak power (PP), peak power adjusted for body mass (RPP), total power (TP), percentage decrement (%Dec) and delta
542 rating of perceived exertion (RPE) along with corresponding effect sizes for control, placebo and ischemic preconditioning repeated sprint trials.
543 Effect sizes correspond to the change between control and ischemic preconditioning trials. Data reported for all participants and by gender
544 group.
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.	Pooled data (n = 16)				Females (n = 9)				Males (n = 7)			
	Control	Placebo	IPC	ES	Control	Placebo	IPC	ES	Control	Placebo	IPC	ES
PP (W)	1583.2 \pm 368.6	1611.7 \pm 461.7	1577.4 \pm 374.1	0.02	1360.4 \pm 247.0	1319.9 \pm 102.3	1353 \pm 184.1	0.03	1869.7 \pm 297.5	1987.0 \pm 476.4	1865.6 \pm 363.9	0.01
RPP (W.kg)	21.4 \pm 3.6	21.7 \pm 4.3	21.3 \pm 2.9	0.04	20.1 \pm 2.7	19.8 \pm 2.7	20.1 \pm 2.0	0.01	23.1 \pm 4.1	24.2 \pm 4.7	22.9 \pm 3.1	0.07
TP (W)	6748.6 \pm 1413.6	6842 \pm 1712.0	6668.6 \pm 1364.0	0.06	5883.5 \pm 895.0	5707.3 \pm 489.2	5788.3 \pm 630.1	0.12	7860.7 \pm 1167.1	8301.0 \pm 1611.5	7800.2 \pm 1211.0	0.05
%Dec	14.1 \pm 5.3	14.4 \pm 5.2	14.8 \pm 4.3	0.15	12.8 \pm 6.2	13.4 \pm 6	14.1 \pm 4.4	0.23	15.8 \pm 3.8	15.7 \pm 3.9	15.8 \pm 4.4	0.01
Bla (mmol⁻¹)	9.3 \pm 2.1	9.0 \pm 2.6	8.2 \pm 2.3	0.51	9.0 \pm 2.4	8.8 \pm 2.4	7.4 \pm 2.0	0.72	9.7 \pm 1.9	9.4 \pm 2.9	9.2 \pm 2.3	0.25
Delta RPE	4.6 \pm 2.3	5.0 \pm 2.9	5.0 \pm 2.3	0.19	3.8 \pm 1.6	4.2 \pm 1.4	4.2 \pm 1.8	0.26	5.6 \pm 2.8	6.0 \pm 2.9	6.0 \pm 3.7	0.18

546 Effect sizes of <0.2, <0.6, <1.2, <2.0 and >2 were considered trivial, small, moderate, large and very large respectively.

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550 **Figure 1.** Mean \pm SD for relative peak power (RPP) and percentage decrement (%Dec)
551 across 5 x 6 s sprints on a cycle ergometer against 7.5% of body mass.

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579 **Figure 2.** Mean \pm SD for the blood lactate (Bla) response following baseline ischemic
580 preconditioning and placebo trials for all participants and separated by gender. * denotes a
581 moderate effect size for differences in Bla response within female participants following
582 ischemic preconditioning compared to baseline trials

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