

1 RESEARCH ARTICLE

2 Running Head: Reduced fixed perceived effort power output with muscle pain

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4 Elevated muscle pain induced by a hypertonic saline injection reduces
5 power output independent of physiological changes during fixed
6 perceived effort cycling

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27 ABSTRACT

28 Pain is a naturally occurring phenomenon that consistently inhibits exercise
29 performance by imposing unconscious, neurophysiological alterations (e.g., corticospinal
30 changes) as well as conscious, psychophysiological pressures (e.g., shared effort demands).
31 Although, several studies indicate that pain would elicit lower task outputs for a set intensity
32 of perceived effort, no study has tested this. Therefore, this study investigated the impact of
33 elevated muscle pain through a hypertonic saline injection on the power output,
34 psychophysiological, cerebral oxygenation, and perceptual changes during fixed perceived
35 effort exercise. Ten participants completed three visits (one familiarisation + two fixed
36 perceived effort trials). Fixed perceived effort cycling corresponded to 15% above gas
37 exchange threshold (mean RPE = 15; hard). Before the 30-minute fixed perceived effort
38 exercise, participants received a randomised, bilateral hypertonic or isotonic saline injection
39 in the vastus lateralis. Power output, cardiorespiratory, cerebral oxygenation, and perceptual
40 markers (e.g., affective valence) were recorded during exercise. Linear mixed model
41 regression assessed the condition and time effects and condition \times time interactions.
42 Significant condition effects showed that power output was significantly lower during
43 hypertonic conditions
($t_{107} = 2.08, p = .040, \beta = 4.77$ Watts, 95%CI [0.27 to 9.26 Watts]). Meanwhile all
45 physiological variables (e.g., heart rate, oxygen uptake, minute ventilation) demonstrated no
46 significant condition effects. Condition effects were observed for deoxyhaemoglobin changes
47 from baseline ($t_{107} = -3.29, p = .001, \beta = -1.50 \Delta\mu\text{M}$, 95%CI [-2.40 to -0.61 $\Delta\mu\text{M}$])
48 and affective valence ($t_{127} = 6.12, p = .001, \beta = 0.93$, 95%CI [0.63,1.23]). Results infer
49 that pain impacts the self-regulation of fixed perceived effort exercise, as differences in
50 power output mainly occurred when pain ratings were higher after hypertonic versus isotonic
51 saline administration.

52

53 NEW & NOTEWORTHY

54 This study identifies that elevated muscle pain through a hypertonic saline injection caused
55 significantly lower power output when pain is experienced but does not seem to affect
56 exercise behaviour in a residual manner. Results provide some evidence that pain operates on
57 a psychophysiological level to alter the self-regulation of exercise behaviour due to

58 differences between conditions in cerebral deoxyhaemoglobin and other perceptual
59 parameters.

60 **Keywords:** effort; exercise behaviour; muscle pain; psychophysiology; self-regulation.

61

62 INTRODUCTION

63 Effort-based decision-making is central to task performance (1). Ultimately,
64 individuals will enact a behaviour if the subjective evaluation about whether the potential
65 reward meets/exceeds the effort to obtain the outcome (2). Naturally, exercise imposes a
66 catalogue of new sensory and perceptual experiences (3) that impact the perceived value of a
67 task (2,4). Consequently, it becomes important for individuals to self-regulate their behaviour
68 and psychophysiological state to promote a continued investment of effort (5).

69 Muscle pain is a perception arising from the integration of nociceptive stimulations of
70 type III and IV muscle afferents (6). Notably, pain has been observed to consistently inhibit
71 exercise performance (3,7-12). On the one hand, the nociceptive element tends to impose
72 numerous, inhibitive neurophysiological alterations along the corticospinal pathways (13,14).
73 For instance, Martinez-Valdes et al. (15) identified that during conditions with higher
74 nociception, the recruitment threshold of fatigue-prone, fast-twitch fibres was lowered
75 whereas fatigue-resistant, slow-twitch fibres saw reduced firing rates. Concomitantly,
76 numerous studies demonstrate that experimental methods which increase nociception/pain
77 (e.g., hypertonic saline, ischaemia, electrical, and/or thermal stimulation) causes an increase
78 in corticospinal inhibition as well as a decrease in corticospinal excitability (13-17). Thus, the
79 underlying nociceptive aspect to pain elicits a compensatory increase in central drive to
80 maintain an exercise intensity compared to conditions with less/lower nociceptive stimulation
81 (10,11). Thereby increasing perceptions of effort for a set intensity of exercise (12,18).

82 On the other hand, pain also inflicts conscious, psychophysiological changes (19). To
83 illustrate, pain has evidenced a marked impact on the hedonic (e.g., less pleasurable) and
84 motivational (e.g., less willing to apply effort) aspects of the affective experience causing
85 people to feel and perform worse when in pain (20). Subsequent data from
86 neurophysiological studies indicate an increased activation of cortical areas associated with
87 inhibitory control (21), particularly when performing with a negative affective valence due to
88 pain (1,19,20). In turn, continued engagement in inhibitory control is believed to exact a

89 motivationally fatiguing effect (22) as well as being associated with a subjective feeling of
90 effort (1). Therefore, it is unsurprising that during painful tasks which require inhibitory
91 control, a given exercise intensity feels more effortful (1,18).

92 In summary, past studies imply that pain and its underlying nociceptive component
93 tend to have negative psychophysiological effects (19) as well as a net inhibitive effect on
94 corticospinal transmission of central drive (13,14). Therefore, for a fixed task intensity like a
95 time-to-exhaustion trial, a compensatory increase in central drive is required to maintain the
96 intensity causing a higher perception of effort for a given intensity (18). Alternatively, when
97 the task paradigm is flipped to a fixed perceived effort task, pain conditions would be
98 expected to cause a reduced intensity/workload compared to non-painful conditions.
99 However, no study has tested this yet. Moreover, as pain is a compelling sensory and
100 emotional experience that must be endured when undertaking exercise (23) it is important to
101 understand the methods that individuals use to self-regulate and cope with pain without
102 compromising exercise performance (5,23).

103 Therefore, the aims of this study were twofold. Primarily, the present study aimed to
104 investigate the impact of elevated pain perceptions through a hypertonic saline injection on
105 power output and psychophysiological state during a fixed perceived effort task. Second, the
106 present study also aimed to investigate the self-regulatory responses (i.e., changes in power
107 output [behavioural] and cerebral haemodynamics [cognitive] as indicators of the self-
108 regulatory strategies) that were used to maintain a fixed perceived effort during hypertonic
109 (painful) or isotonic (placebo-control) conditions.

110 It was hypothesised that mean power output would be lower in the hypertonic versus
111 isotonic condition (condition effect). Second, it was hypothesised that the decreases over time
112 in power output would be steeper in the hypertonic versus isotonic condition (condition \times
113 time interactions). It was also hypothesised that changes in cerebral oxygenation markers
114 from baseline would be greater in the pain versus isotonic condition indicating more
115 inhibitive control (24,25). Finally, a series of secondary hypotheses were made that markers
116 of physiological strain (e.g., heart rate, ventilatory parameters, blood lactate) would be lower
117 in the hypertonic than the isotonic condition, whilst perceptual markers like affective valence
118 would be lower in the hypertonic versus isotonic condition.

119

120 MATERIALS AND METHODS

121 PARTICIPANTS

122 Ten healthy and recreationally trained cyclists (two female) with a mean \pm SD age:
123 28.9 ± 6.6 years, height 175.8 ± 6.1 cm, mass: 72.1 ± 8.0 kg, physical activity: 6.1 ± 2.9
124 hours.week⁻¹, maximum relative oxygen uptake ($\dot{V}O_{2\cdot kg^{-1}}$): 52.6 ± 7.2 mL.kg⁻¹.min⁻¹
125 volunteered to participate in this study. An α -priori calculation using an effect size ($dz =$
126 1.09) from (11) which used an identical saline injection procedure, $\alpha = .05$, and $\beta = 0.8$,
127 determined a required sample size of 10 to determine a sufficient effect on power output
128 during a fixed perceived effort trial with an actual $\beta = 0.82$. All participants reported at least
129 three years of cycling experience, current engagement in cycling activity, and an ‘excellent’
130 $\dot{V}O_{2\max}$ according to (26) to qualify for this study. All participants were free from any
131 musculoskeletal injuries in the previous six months, with no cardiovascular disease,
132 neurological disorders, or blood-borne viruses, and participants did not use dietary
133 supplements or medication throughout the entire study. Prior to all data collection sessions,
134 participants abstained from food (2 hours), caffeine (4 hours), analgesics (8 hours), alcohol
135 (48 hours), and refrained from vigorous exercise (48 hours). Female participants reported
136 being eumenorrheic and were scheduled so that all visits were conducted within the same
137 stage of menses (luteal phase). All participants provided written informed consent before
138 testing for this School of Sport and Exercise Sciences Research Ethics Advisory Group
139 approved study (Prop #11_20_21) which was conducted according to the scientific principles
140 outlined within the Declaration of Helsinki.

141

142 PROCEDURES

143 The present study implemented a randomised, single-blinded, within-subject design
144 whereby the lead researcher was blinded to which conditions were being completed. Initially,
145 the researchers aimed to complete a double-blinded design however, the infusion of
146 hypertonic saline may naturally be distinguished from the isotonic saline by participants (7,9-
147 12). On three separate occasions (Figure 1) participants were required to visit the same
148 laboratory. Each visit was conducted at the same time of day (± 2 hours) in similar ambient
149 environments (mean \pm SD temperature: 19.6 ± 3.8 °C, humidity: 51.9 ± 8.4 %, barometric
150 pressure: 751.9 ± 7.7 mmHg). Each visit was separated by a minimum of three days and
151 maximum of seven days.

152 At the start of each session, participants' anthropometrics were recorded, and they
153 were provided with a full brief of the procedures, equipment, and perceptual scales.
154 Participants were fitted to the functional near infrared spectroscopy (fNIRS) device (Artinis
155 Medical Systems BV: PortaLite MK II, Arnhem, Netherlands) and asked to sit completely
156 still for five minutes during baseline measures. Participants were also fitted with a heart rate
157 monitor (Cyclus 2: ANT+, Leipzig, Germany) to assess heart rate on a beat-by-beat basis and
158 provided a 20 μ L resting blood lactate sample from the right index finger to be assessed using
159 an automated lactate analyser (Biosen: C-Line, EKF Diagnostics, GmbH, Barleben,
160 Germany). Finally, participants provided baseline values for each perceptual scale (see
161 'perceptual scales').

162 Participants performed identical ten-minute warm-ups at a rating of perceived effort
163 (RPE) of 11; "light", on the cycle ergometer (Cyclus2, Leipzig, Germany). After the warm-
164 up, participants were afforded five minutes of passive recovery before remounting the cycle
165 ergometer to begin the respective exercise tasks for each session. During all exercise tasks,
166 participants were fitted to a calibrated gas analyser system (Cortex Metalyser: Model 3B,
167 Leipzig, Germany) to assess pulmonary ventilation (e.g., $\dot{V}O_2 \cdot \text{kg}^{-1}$, minute ventilation [\dot{V}_E],
168 and breathing frequency) on a breath-by-breath basis. After exercise, participants completed a
169 short questionnaire pack where on completion they were debriefed and exited the laboratory.

170

171 VISIT 1 – RAMPED INCREMENTAL TEST AND FAMILIARISATION

172 The first visit consisted of a ramped incremental test and a familiarisation to fixed
173 perceived effort cycling with bilateral hypertonic saline administration. The ramped
174 incremental test involved an initial three-minute stabilisation period at 80% starting intensity
175 (males = 80 Watts, females = 40 Watts). Participants were asked to cycle at a comfortable
176 cadence $\sim 80 \text{ revolutions} \cdot \text{min}^{-1}$ and were recommended to gradually increase cadence over the
177 course of the test. The incremental ramped test began at 100 Watts (males) or 50 Watts
178 (females) with $25 \text{ Watts} \cdot \text{min}^{-1}$ increments. These intensities were selected according to pilot
179 test data to ensure ramped incremental tests lasted between eight – twelve minutes as
180 previously recommended (27).

181 During the ramped incremental tests, breath-by-breath analysis of oxygen
182 consumption ($\dot{V}O_2$), carbon dioxide expulsion ($\dot{V}CO_2$), \dot{V}_E , and breathing frequency were
183 taken. An RPE response was obtained at each minute (including starting intensity and at the

184 point of exhaustion). Finally, a blood lactate sample was taken at the point of exhaustion.
185 Cerebral oxygenation via fNIRS, affective valence, and pain intensity were not measured
186 during the ramped incremental test. Task cessation demarcated when the participant believed
187 they reached volitional exhaustion or if cadence fell below 60 revolutions.min⁻¹ for more than
188 five seconds despite strong verbal encouragement.

189 After the ramped incremental test, participants received 15 minutes passive recovery
190 and were then prepared for a ten-minute fixed perceived effort cycle at RPE 15; “hard” after
191 receiving a bilateral hypertonic saline intramuscular injection for familiarisation. A full
192 explanation of the fixed perceived effort trials can be seen in ‘Visits 2 & 3 – fixed perceived
193 effort trials’.

194

195 DETERMINATION OF FIXED PERCEIVED EFFORT INTENSITY IN VISITS 2 & 3

196 Using the \dot{V} -slope method (28), gas exchange threshold (GET) was matched to the
197 point at which $\dot{V}O_2$ values above and below the breakpoint of $\dot{V}CO_2$ diverged from the
198 intersection of the two linear regression lines. Secondary criteria including ventilatory
199 equivalents (first divergence of ventilatory equivalent of oxygen and carbon dioxide), end-
200 tidal volumes (first divergence of end-tidal volumes for oxygen and carbon dioxide),
201 respiratory exchange ratio (reaching a value of 1.00), and a secondary researcher confirmed
202 GET identification (26). Once GET was determined, $\dot{V}O_2$ values 15% above GET (GET_{+15%})
203 were calculated. Plotting GET_{+15%} $\dot{V}O_2$ against power output from the ramped incremental
204 test, a regression equation ($y = mx + c$) derived what power output corresponded to the
205 GET_{+15%} $\dot{V}O_2$. Finally, power output data was plotted against ramped incremental RPE
206 responses in which a similar regression equation was used to identify RPE (RPE_{+15%GET}) at
207 the corresponding power output at GET_{+15%}. This RPE was rounded to the nearest whole
208 number and used as the RPE reference for subsequent fixed perceived effort cycling in visits
209 2 and 3 (mean \pm SD RPE_{+15%GET} = 14.7 \pm 0.4, 8n = RPE 15; “hard”, 2n = RPE 14; between
210 “somewhat hard” and “hard”).

211

212 VISIT 2 & 3 – FIXED PERCEIVED EFFORT TRIALS

213 Both experimental sessions were single-blinded and randomised. After the same
214 preparation, baseline, and warm-up protocols as Visit 1, participants were prepared to receive
215 two simultaneous, bilateral saline injections before commencing a 30-minute fixed perceived
216 effort cycle. Injections involved a bolus of 1 mL saline (hypertonic = 5.85% NaCl, isotonic =

217 0.9% NaCl) injected into the middle third of the muscle belly of the vastus lateralis on each
218 leg. Injection sites were measured and marked to ensure consistent locality of injection. Sites
219 were cleaned with an alcoholic swab and saline was manually infused using a 3 mL Luer-Lok
220 syringe (BD, New Jersey, USA) connected to a 3.8 cm 25-gauge hypodermic needle
221 (SurGuard2, Terumo, Japan) over a 20 second window (insertion, five second pause, ten
222 second infusion period, five second pause, withdrawal). A hypertonic saline model was
223 utilised as several studies have validated its ability to mimic exercise-induced pain
224 experiences across different physical task modalities (9-11,16,17,29,30) as well as
225 demonstrating its replicability (29) although it does present some difficulties with blinding as
226 participants can distinguish which condition they are completing which may generate some
227 confounding effects on behavioural, motivation (1,19), and other psychophysiological indices
228 such as hyperventilation (29).

229 Immediately after the injection procedure, participants began cycling and ramped up
230 to the required RPE (mean \pm SD time to begin fixed perceived effort task: hypertonic = $27 \pm$
231 9 s, isotonic = 29 ± 9 s). Following this, the fixed perceived effort trial commenced. During
232 this, power output, heart rate, gas parameters, cerebral oxygenation parameters via fNIRS,
233 and pain measurements were assessed continually whilst affective valence and blood lactate
234 were assessed every five minutes.

235 Crucially, the task was a fixed perceived effort trial (see [31]), therefore, throughout
236 the trial, participants were blinded from all performance-related variables (e.g., power output,
237 time on task) except for cadence. In doing so, participants' sole focus was to maintain a fixed
238 perceived effort. Participants were asked to maintain a cadence between 80 - 90 (± 2)
239 revolutions.min⁻¹ that was replicated across both sessions (mean \pm SD 86 ± 3 revolutions.min⁻¹
240 ¹). However, power output could be changed at any point throughout the exercise to maintain
241 the fixed perceived effort using virtual gears on the Cyclus2 ergometer console which
242 changed the resistance at the set cadence. The researcher provided a reminder of the RPE
243 definition (32) and need for the participant to be at a fixed perceived effort every two
244 minutes.

245 ***Please Insert Figure 1 – Figure of Research Protocols***

246

247 fNIRS MEASUREMENT

248 Cerebral oxygenation was assessed through a portable fNIRS device. The device was
249 placed on the surface of the forehead aligned with the left prefrontal cortex between Fp1 and
250 F3 (international EEG 10-20 system) as this aligns with relevant cerebral centres for
251 executive motor control (33). Prior to application, the skin was wiped with an alcohol swab
252 and a thin transparent film was placed over the site to prevent any sweat interfering with the
253 device. To protect from light interference, a black bandana was placed over the device which
254 held it stationary. Furthermore, the wire leading from the optode to the laptop was taped
255 tightly onto the cycle ergometer and adjoining table to avoid movement artifacts. Pre-
256 calibration adjusted an age-dependent differential path-length factor and data were sampled at
257 10 Hz from six optodes at wavelengths between 760 – 850 nm according to manufacturer’s
258 guidelines. Data were sampled from single, long-separation channels. Moreover, according
259 with the manufacturer’s guidelines and prior studies (34), a low-pass filter of 0.1 Hz was
260 applied to all participant data and a visual inspection of all data was completed to identify and
261 remove any movement artifacts present in the data. A five-minute resting baseline was
262 completed at the beginning of each session, whereby any fNIRS data obtained during
263 subsequent exercise tasks was represented as changes from baseline (Δ) (35). Therefore,
264 fNIRS data during exercise was expressed as change in oxyhaemoglobin ($\Delta\text{O}_2\text{Hb}$),
265 deoxyhaemoglobin (ΔHHb), total haemoglobin (ΔtHb), and tissue saturation index ($[\text{TSI}] =$
266 $\Delta\text{O}_2\text{Hb}/\Delta\text{tHb} \times 100$) compared to resting baseline with an arbitrary average baseline value
267 denoting 0 μM , in accordance with previous research (36,37).

268

269 PERCEPTUAL SCALES

270 RPE SCALE

271 The 15-point Borg RPE scale (38) denoted *how hard, heavy, and strenuous does the*
272 *exercise consciously feel to drive the working muscles and for your breathing* (31).
273 Responses ranged from 6; “no effort”, *like when you were sat during the fNIRS baseline*
274 *doing absolutely nothing* to 20; “maximum effort”, *like giving everything you have got like at*
275 *the end of a $\dot{V}\text{O}_2$ max test*. Appropriate anchors were given before exercising to facilitate the
276 consistency of participant responses (39,40).

277

278 AFFECTIVE VALENCE SCALE

279 The feeling scale (41) denoted *how are you feeling at the present moment of the*
280 *exercise*. Responses ranged on an 11-point Likert scale from +5 “I feel very good” to -5 “I
281 feel very bad” with a middle value of 0 denoting “neutral”.

282

283 PAIN MEASUREMENT

284 During experimental exercise trials, a continual rating of exercise-induced pain
285 intensity was obtained by participants using a moveable cursor on an electronic VAS that
286 sampled a recording every five seconds. Responses ranged from 0 = “no pain” to 100 =
287 “worst imaginable pain” (8). This device was placed on the handlebars of the ergometer for
288 ease. Participants were instructed to anchor the uppermost pain rating to the worst exercise-
289 induced pain they had previously experienced (8,42).

290 Pain quality was assessed using the long form McGill pain questionnaire (43) to
291 assess several pain elements such as sensory, affective, and evaluative qualities. Therefore,
292 the McGill pain questionnaire allows a more multidimensional consideration of pain that goes
293 beyond the simple magnitude of pain. Each category contains adjectives that are ranked in
294 ascending order according to implied pain intensity (e.g., descriptor one assigned a value of
295 1). A subclass rating index denoted a sum for each subclass and a total pain rating index
296 denoted a sum of all subclasses. The McGill pain questionnaire was administered after each
297 fixed perceived effort exercise task where participants were required to select one word from
298 each subcategory if any of the descriptors applied.

299

300 ANALYSIS

301 Power output data was averaged across each minute of the 30-minute fixed perceived
302 effort trials. All other continuous data (e.g., physiological [except blood lactate], cerebral
303 oxygenation markers) and pain intensity ratings were averaged across six, five-minute time
304 zones (e.g., time zone 1 = minute 00:00 – 04:59). Affective valence and blood lactate were
305 analysed according to the minute they were extracted (e.g., minute 0, 5, etc).

306 All data were exported to Jamovi (JAMOVI: v 2.3, Sydney, Australia) and was
307 assessed for normality and symmetry using a Q-Q plots and a Shapiro-Wilk test before any
308 further analysis. Any data that exceeded 2SD from the group mean was excluded from further
309 analysis although subsequent analysis evidenced that no participants data exceeded 2SD from

310 the group mean. A series of paired samples t tests were conducted to assess differences
311 between conditions in resting responses for perceptual markers and blood lactate.

312 A random-intercepts linear mixed-effects models regression was conducted to assess
313 the condition and/or time effects as well as the condition \times time interactions on all dependent
314 variables data. Condition effects observed differences between hypertonic and
315 isotonic(placebo-control) conditions. Time effects observed differences over the course of the
316 30-minute perceived effort task. Condition \times time interactions observed the differences
317 between conditions in changes to a set variable over time. The generalised form for the linear
318 mixed model regression is presented below (a) showing that the grouping/cluster variable was
319 each participant.

320 (a) (Dependent Variable) = Condition + Time Zone + Condition:Time Zone +
321 (1|Participant)

322 The variable of *condition* and *time* were set as fixed effects. Models were fitted
323 according to the group intercept. Results from the linear mixed-model regression were
324 reported as t values as time was entered as a continuous variable. Another benefit to this
325 method is that reporting of estimated marginal means (β -coefficient) denotes the raw mean
326 differences between the two conditions as an effect size with supplementary 95% confidence
327 intervals (95%CI). A normality test was conducted on the residual values and if they violated
328 normality, a Wilcoxon signed ranks test was reported with a rank biserial correlation (r)
329 denoting effect size. All data reported for the mixed models regression is according to
330 isotonic – hypertonic comparisons with positive t and β values showing a higher value in the
331 isotonic versus hypertonic condition.

332 Data from the McGill pain questionnaire underwent a basic frequency analysis
333 whereby each descriptor was assigned a score (1 – 5) according to its severity. Each of the 20
334 categories of descriptors were grouped according to their subclass and a total score for each
335 subclass was calculated for each condition and participant. Next all subclass totals were
336 calculated to also create a total pain rating index across each condition and participant. Mean
337 scores across the cohort for each subclass as well as the total pain rating index underwent a
338 series of t tests to assess the differences between conditions. For clarity, only descriptors
339 which were selected by over one third of the cohort are presented in Table 1. A Wilcoxon
340 signed ranks test was reported if data violated normality and a Cohen's d was reported to
341 denote effect size. The alpha level for all tests was set at $P \leq 0.05$.

342

343 RESULTS

344 STANDARDISATION

345 Prior to beginning the experimental fixed perceived effort cycling trials, all
346 participants rated no pain (0), and blood lactate was not significantly different between
347 conditions (hypertonic = 1.53 m.mol^{-1} versus isotonic = 1.45 m.mol^{-1} , $p = .327$, $d = .18$).
348 In addition, affective valence did not differ between conditions prior to exercise (hypertonic =
349 2.2 versus isotonic 2.6, $p = .111$, $d = .21$).

350

351 POWER OUTPUT AND PHYSIOLOGICAL MARKERS

352 Power output was found to be significantly lower in the hypertonic compared to
353 isotonic condition with significant main effects for condition ($t_{107} = 2.08$, $p = .040$, $\beta =$
354 4.77 Watts [0.27,9.26]) being observed. Power output also decreased over time in both
355 conditions with main effects for time
356 ($t_{107} = -6.11$, $p = .001$, $\beta = -5.80 \text{ Watts}$ [-7.66,3.94]) being observed (Figure 2). The
357 trajectories of power output changes did not significantly differ between conditions as there
358 was no condition \times time interaction ($t_{107} = -1.32$, $p = .189$, $\beta = -1.78$ [-4.41,0.86]).

359

Please Insert Figure 2 – Power Output

360 There were no differences in heart rate between conditions ($t_{107} = 1.69$, $p =$
361 $.094$, $\beta = 1.82 \text{ b.min}^{-1}$ [-0.29,3.92]). However, heart rate did increase across both
362 conditions as a significant main effect for time
363 ($t_{107} = 5.63$, $p = .001$, $\beta = 1.77 \text{ b.min}^{-1}$ [1.15,2.39]) was observed (Figure 3a).
364 Trajectories in heart rate changes did not differ between conditions ($t_{107} = -1.17$, $p =$
365 $.246$, $\beta = -0.73$ [-1.97,0.50]).

366

366 Similarly, $\dot{V}\text{O}_2.\text{kg}^{-1}$
367 ($t_{107} = 1.34$, $p = .182$, $\beta = 0.57 \text{ mL.min}^{-1}.\text{kg}^{-1}$ [-0.26,1.39]) and \dot{V}_E $t_{107} = 1.43$, $p =$
368 $.157$, $\beta = 2.12 \text{ L.min}^{-1}$ [-0.79,5.04]), did not demonstrate a significant condition effect.
369 However, $\dot{V}\text{O}_2.\text{kg}^{-1}$ ($t_{107} = -5.29$, $p = .001$, $\beta = -0.65 \text{ mL.min}^{-1}.\text{kg}^{-1}$ [-0.90, -0.41])
370 and \dot{V}_E ($t_{107} = -4.31$, $p = .001$, $\beta = -1.88 \text{ L.min}^{-1}$ [-2.73, -1.02]) did demonstrate
371 significant changes in values over time (Figure 3b and c). No significant condition \times time

372 interactions were observed for $\dot{V}O_2 \cdot \text{kg}^{-1}$ ($t_{107} = -0.86, p = .394, \beta = -0.21 [-0.70, 0.27]$)
373 or \dot{V}_E ($t_{107} = -1.10, p = .273, \beta = -0.96 [-2.67, 0.75]$).

374 Breathing frequency was not significantly different between conditions ($t_{107} =$
375 $1.72, p = .088, \beta = 1.00 \text{ breaths} \cdot \text{min}^{-1} [-0.14, 2.14]$) and did not differ over time
376 ($t_{107} = 1.82, p = .072, \beta = 0.31 \text{ breaths} \cdot \text{min}^{-1} [-0.02, 0.64]$) (Figure 3d). In addition,
377 breathing frequency did not show a significant condition \times time interaction ($t_{107} =$
378 $-0.32, p = .750, \beta = -0.11 [-0.77, 0.56]$). Finally, no significant main effects for
379 condition ($t_{127} = 1.84, p = .068, \beta = 0.45 \text{ m} \cdot \text{mol}^{-1} [-0.03, 0.92]$), or time ($t_{127} =$
380 $-1.29, p = .200, \beta = -0.02 \text{ m} \cdot \text{mol}^{-1} [-0.04, 0.01]$), were observed for blood lactate. To
381 add, condition \times time interactions for blood lactate ($t_{127} = -0.27, p = .789, \beta =$
382 $-0.01 [-0.05, 0.04]$) were insignificant (Figure 4).

383 ***Please Insert Figure 3, Panels a - d – Figures of Cardiorespiratory Changes***

384 ***Please Insert Figure 4 – Figure of Blood Lactate***

385

386 CEREBRAL OXYGENATION MARKERS

387 A condition effect for $\Delta O_2\text{Hb}$ was not observed ($t_{107} = -1.71, p = .091, \beta =$
388 $-1.48 \Delta\mu\text{M} [-3.17, 0.22]$). However, a significant main effect for time ($t_{107} = 6.81, p =$
389 $.001, \beta = 1.72 \Delta\mu\text{M} [1.22, 2.22]$) was observed for $\Delta O_2\text{Hb}$ as it increased over the course of
390 the exercise in both conditions (Figure 5a). The linear mixed-model regression showed no
391 condition \times time interaction for $\Delta O_2\text{Hb}$ ($t_{107} = -0.70, p = .486, \beta = -0.35 [-1.35, 0.64]$).

392 Alternatively, ΔHHb ($t_{107} = -3.29, p = .001, \beta = -1.50 \Delta\mu\text{M} [-2.40, -0.61]$)
393 and ΔtHb ($t_{107} = -4.15, p = .001, \beta = -5.46 \Delta\mu\text{M} [-8.04, -2.88]$) were observed to be
394 significantly lower in the isotonic compared to hypertonic condition (Figure 5b and c). Both
395 ΔHHb ($t_{107} = 4.04, p = .001, \beta = 0.54 \Delta\mu\text{M} [0.28, 0.80]$) and ΔtHb ($t_{107} = 5.65, p =$
396 $.001, \beta = 2.18 \Delta\mu\text{M} [1.42, 2.94]$) also showed a significant time-based main effect with both
397 increasing over the course of the exercise. However, no significant condition \times time
398 interaction was noted for ΔHHb ($t_{107} = -0.44, p = .659, \beta = -0.12 [-0.64, 0.41]$) or
399 ΔtHb ($t_{107} = -0.83, p = .407, \beta = -0.64 [-2.15, 0.87]$).

400 Lastly, no significant condition ($t_{107} = 1.94, p = .055, \beta = 0.52 \% [-0.01, 1.04]$)
401 or time ($t_{107} = -0.58, p = .566, \beta = -0.04 \% [-0.20, 0.11]$) main effects were found for

402 Δ TSI. Also, there was not a significant condition \times time interaction for Δ TSI ($t_{107} =$
403 1.91, $p = .059$, $\beta = 0.30 [-0.01, 0.60]$).

404 ***Please Insert Figure 5, Panels a – c – Figures of Cerebral Oxygenation Responses***

405

406 PERCEPTUAL MARKERS

407 Affective valence was found to be significantly lower in the hypertonic compared to
408 isotonic condition with a significant condition main effect ($t_{127} = 6.12$, $p = .001$, $\beta =$
409 0.93 [0.63, 1.23]), as well as a significant main effect for time ($t_{127} = -3.96$, $p = .001$, $\beta =$
410 $-0.03 [-0.04, -0.02]$). Notably, time-based changes in affective valence differed between
411 condition as a linear mixed-model regression also observed a significant condition \times time
412 ($t_{127} = -3.16$, $p = .002$, $\beta = -0.05 [-0.08, -0.02]$) interaction. Particularly, affective
413 valence responses were more negative in earlier stages of the exercise in the hypertonic
414 compared to isotonic condition (Figure 6a).

415 Pain ratings were significantly higher in the hypertonic compared to isotonic
416 condition ($t_{127} = -5.90$, $p = .001$, $\beta = -9.97 [-13.28, -6.66]$) (Figure 6b). However,
417 time-based main effects were not significant
418 ($t_{127} = -1.78$, $p = .077$, $\beta = -0.15 [-0.32, 0.01]$). Trajectories in the changes of pain
419 ratings were significantly different between conditions with a significant condition \times time
420 interaction ($t_{127} = 6.00$, $p = .001$, $\beta = 0.95 [0.61, 1.28]$). Particularly, pain decreased then
421 plateaued in the hypertonic condition and pain increased then plateaued in the isotonic
422 condition.

423 ***Please Insert Figure 6, Panel a - b – Perceptual Responses***

424 Table 1 demonstrates the dimensional quality of perceived pain during trials. Total
425 scores for subclasses of sensory and affective domains did not demonstrate significant
426 differences between conditions, however, a moderate effect ($d = .55$) in the sensory and a
427 large effect ($d = .80$) in the affective domain were observed. Total scores for dimensions of
428 evaluative ($Z = 2.392$, $p = .017$, $d = .67$), miscellaneous ($t = 3.139$, $p = .012$, $d =$
429 $.50$), and PRI ($Z = 2.075$, $P = .038$, $d = 0.84$) did demonstrate significant differences
430 between conditions with moderate and large effect sizes.

431 ***Please Insert Table 1 – Table of MPQ Responses***

432

433 DISCUSSION

434 This study aimed to investigate the impact of elevated muscle pain through a
435 hypertonic saline injection on the power output changes, psychophysiological state, and
436 cerebral oxygenation variables during a fixed perceived effort exercise task. Knowledge of
437 the changes in the power output, psychophysiological indices and cerebral haemodynamics
438 also contributed to a secondary question which explored the self-regulatory strategies that
439 were used to maintain a fixed perceived effort during conditions of pain (hypertonic) or a
440 placebo-control (isotonic).

441 The main finding of the present study is that the hypertonic condition elicited a
442 significantly lower power output (by an average of 5 Watts) than the isotonic condition.
443 Alongside which, there were no significant condition effects on any physiological variables
444 like heart rate, $\dot{V}O_2 \cdot kg^{-1}$, \dot{V}_E , breathing frequency, or blood lactate. However, differences in
445 power output between conditions were paired with significant differences in pain intensity
446 and quality responses which were found to be significantly higher in the hypertonic compared
447 to isotonic condition. Likewise, this study demonstrated significantly worse/more negative
448 affective valence responses in the hypertonic compared to isotonic condition. Finally, there
449 was a significantly higher change in deoxyhaemoglobin levels from baseline in the
450 hypertonic versus isotonic condition.

451 Findings pertaining to power output confirmed our initial hypothesis. Numerous
452 studies have demonstrated a reduced task output (e.g., power output, force, duration on task)
453 during painful compared to non-painful conditions (7-12). Notably, muscle pain imposes
454 neurophysiological alterations such as changes in corticomotor conductance of central drive
455 (13,14,16) and muscle fibre recruitment (15,17) as well as heightened psychophysiological
456 demands such as reduced affect (19,20). The psychophysiological consequences of pain are
457 confirmed in this study. Namely, this study observed lower/worse affective valence responses
458 during the hypertonic versus isotonic condition. Inferring that individuals may have
459 experienced a less hedonic experience (20) due to the pain with further implications on their
460 motivation to continue exercising at the same perception of effort (44), thus resulting in a
461 negatively valenced affective response (45). Furthermore, the changes in deoxyhaemoglobin
462 from a resting baseline were significantly higher in the painful hypertonic versus less painful
463 isotonic condition. Specifically, cerebral oxygenation measures were taken from the

464 prefrontal cortex which several recent studies have indicated is linked to executive function
465 (46). Therefore, the results of this study imply that individuals during the painful, hypertonic
466 condition engaged in more inhibitory control (a subset of executive function) to cope with
467 pain (47-49). Notably, continued inhibitory control is closely associated with increases in
468 effort due to enhanced activity of cortical areas (18,20,46,47) associated with effort
469 processing as well as a motivationally fatiguing effect (22). Consequently, it is expected that
470 exercise in the presence of higher pain is more effortful than exercise without pain (1,3,5,18).
471 When the task paradigm is switched to a fixed perceived effort trial, it is expected that the
472 task output such as power output would be lower within conditions of pain versus a control
473 (7,9-12). Yet, some caution is warranted when considering haemodynamic responses as there
474 are potential confounds involving the autonomic nervous regulation of blood flow during an
475 exercise of vigorous intensity that could impact the raw changes in oxygenation markers that
476 were measured (34,50).

477 However, it was interesting to note that there were no differences in any of the
478 physiological/cardiorespiratory markers despite significant differences in power output,
479 leading the authors to reject some aspects of their secondary hypotheses. Certain models of
480 exercise regulation insist that exercise behaviour is governed by afferent feedback loops that
481 relay information through the central nervous system concerning metabolic and
482 proprioceptive changes (51). Yet, the results of this study appear in conflict with this
483 suggestion as physical outputs at a constant perceived intensity were not proportional to the
484 subconscious changes in cardiorespiratory and metabolic parameters that were monitored.
485 Alternatively, it may be worthwhile acknowledging other models (e.g., psychobiological
486 model [1]) which claim that afferent feedback impacts exercise behaviour *via* changes in
487 effort perceptions. Relatedly, a recent study by Mauger et al. (52) discerned that after trained
488 cyclists were administered tramadol (a very potent painkiller), performance in a subsequent
489 time-trial was significantly faster compared to a placebo-controlled condition. In addition,
490 Mauger and colleagues (52) required participants to conduct a fixed intensity cycle prior to
491 their time-trial and found that RPE responses were significantly lower after tramadol
492 ingestion versus control. Therefore, some indications could be made to justify the effect
493 afferent feedback like nociception/pain has on the exercise performance due to its combined
494 neurophysiological *and* psychophysiological influences on effort perceptions (7).

495 Consecutively, this study aimed to explore the self-regulatory strategies that operate
496 during fixed perceived effort cycling in the presence of painful (hypertonic) or less/non-

497 painful (isotonic) conditions. Mainly, condition \times time interactions can illustrate the
498 differences in the changes for power output (behavioural) or cerebral haemodynamics
499 (cognitive) self-regulation over time. Furthermore, researchers of this study were aware that a
500 hypertonic saline procedure typically peaks at \sim 3 minutes and dissipates within \sim 5-6 minutes
501 after administration (9-12,29,30) yet the fixed perceived effort task lasted 30 minutes.
502 However, this generated another question as to whether a pain experience imposes *residual*
503 effects at later stages of an exercise task as previous studies have shown that even after a pain
504 experience, neurophysiological markers do not immediately return to baseline, perhaps due to
505 a retained motor adaptation (15).

506 Results conflicted our prior hypotheses with no significant condition \times time
507 interactions for power output, any markers of physiological strain, or cerebral oxygenation
508 parameters. Figure 2 illustrates that both conditions exhibited an expected decrease in power
509 output (31) but the rate at which power output decreased was unaffected. Meanwhile,
510 markers of physiological strain (Figure 3) indexed a plateau which would be expected for
511 certain markers like breathing frequency during fixed perceived effort exercise (53).
512 Similarly, changes in oxy-, deoxy-, and total haemoglobin over the course of the fixed
513 perceived effort bouts were not significantly different between conditions (Figure 5). Instead,
514 the only significant condition \times time interactions that were observed related to the pain
515 intensity and affective valence responses (Figure 6). Naturally, differences in pain intensity
516 responses were expected as the hypertonic condition evoked higher perceptions of pain
517 compared to the isotonic at the start of the exercise whereas the progressive engagement in
518 exercise caused naturally occurring muscle pain to reach similar levels in the latter stages of
519 the task (8). Second, the affective valence responses exhibited that the painful hypertonic
520 saline conditions caused affect to become more negative/worse much sooner and whereas the
521 isotonic condition caused affect to become negative at a much steadier rate. However, it is
522 interesting that this difference in affective valence did not instigate any differences in self-
523 regulatory behaviour (i.e., changes in power output) as some may expect (54).

524 Consequently, two main conclusions can be drawn about the self-regulation of
525 perceived effort during conditions of pain versus less/non-painful conditions. First, it appears
526 that pain does prompt a difference in task outputs at a set perception of effort as shown by the
527 condition effects for power output and cerebral oxygenation markers. A second conclusion is
528 that the pain ratings and power output data indicate that pain does affect the perception of
529 effort and associated outputs but only when it is *experienced*. Alternatively, pain does not

530 seem to demonstrate any *residual* effects which impact exercise behaviour at a later stage of a
531 task when elevated muscle pain has dissipated. To illustrate, there were no significant
532 condition \times time interactions suggesting that although higher pain ratings at the start of the
533 exercise may be indicative of increased engagement in inhibitory control, this may not be an
534 enduring effect on exercise behaviour as prior resource models of self-regulation would
535 suggest (5).

536

537 LIMITATIONS AND FUTURE RESEARCH

538 Some aspects of this study's methodological approach could be adapted in future
539 studies to understand more about the effect of pain on perceived effort and the subsequent
540 self-regulation of exercise behaviour. One note is that this study did not control for the
541 volume of the saline bolus in accordance with muscle mass. Instead, all participants were
542 administered a bolus of 1 mL of saline. As a result, those with lower vastus lateralis mass
543 may have experienced a higher intensity of pain versus those with greater muscle mass.
544 Observations of the pain data (Figure 6b) does show a varied response to the hypertonic
545 saline when it was most potent (minutes 0 and 5). As a result, this may in part, contribute to
546 the slightly larger variances in power output (95%CI = 0 – 9 Watts lower in the hypertonic
547 versus isotonic condition over 30 minutes).

548 Another aspect of the varied power output response may have been due to the
549 duration of the fixed perceived effort task. As noted previously, whilst the 30-minute task
550 duration afforded researchers to observe any potential residual effects of pain on exercise
551 behaviour, the differences in later stages of the task were negligible (2 – 4 Watts). Thus,
552 skewing the observed effects and increasing the likelihood of a type II error. However, the
553 results did show an average difference of 10 - 25 Watts at minutes 0 – 5 whilst the pain
554 intensity was higher due to the hypertonic saline (Figure 2). A result that is both statistically
555 as well as physiologically meaningful. In context, individuals experiencing high levels of
556 pain are likely to conduct a given task at a much slower rate with potentially inferior
557 performance (7-12,20-22,29). To add, an overall average (i.e., the entire 30-minute group
558 mean) exhibited a ~5 Watts lower power output in the painful versus isotonic condition.
559 Though this result may not be entirely meaningful for everyday situations, it is still
560 statistically significant and could still be considered relevant to elite sporting populations. For
561 instance, RPE responses ~15; "hard" are commonplace at the initial phases of a prolonged

562 time-trial (55). Therefore, if a competitor can gain an initial advantage due to a higher power
563 output at the start of a race-type situation due to being free from any existing pain, this is
564 contextually meaningful (55).

565 Finally, whilst this study aims to incorporate the best practice for fNIRS measurement
566 (34), some aspects of data collection were not viable. For example, Pinti et al. (34) suggests
567 that the additional use of short separation channels to obtain fNIRS data may allow a better
568 interpretation of fNIRS neuroimaging data when analysed with linear mixed model
569 regression like those used in this study. To add, short separation channels can detect
570 additional noise from extracerebral signals (e.g., cardiac cycles) which can subsequently
571 factor into the analysis of data to eradicate confounds such as systemic interference as a
572 consequence of the exercise. However, as this study was concerned with oxy-/deoxy-
573 haemoglobin changes at the prefrontal cortex, long separation, single channels we used due to
574 the need for penetration to deeper tissues (e.g., versus muscle fNIRS). Though filters
575 identical to previous studies in the area were used to eradicate potential noise and confounds
576 (33,35-37), some caution is warranted in the interpretation of fNIRS data.

577 In accordance with these shortcomings, future research may wish to control for the
578 volume of saline that is applied according to muscle mass. Furthermore, the duration of a task
579 could be curtailed to fit the expected time saline procedures remain effective (~5-6 minutes).
580 Beyond, other suggestions for future research could involve other markers of cognitive effort.
581 Whilst several studies have hinted towards cerebral oxygenation markers as being indicative
582 of cognitive effort (47-50), other methods such as pre-ejection period and eye-tracking (e.g.,
583 measurement of pupil diameter and/or variability in fixation locations) are potentially
584 effective at measuring cognitive load/effort through another physiological approach (56,57).
585 Characteristically, exercise tasks impose physical and cognitive demands, but little is known
586 about ways in which individuals choose between applying physical or cognitive effort (2,4).
587 Therefore, future research could explore this area as it could shed light into how
588 psychophysiological constructs like pain and effort are regulated and influence exercise
589 behaviours and performance.

590

591 CONCLUSION

592 The current study aimed to investigate the impact of elevated pain perceptions
593 through a hypertonic saline injection on power output and psychophysiological state during a
594 fixed perceived effort task. It was observed that the painful hypertonic condition caused a
595 significantly lower power output, a greater increase in deoxyhaemoglobin compared to rest,
596 and a lower/worse affective response compared to a placebo-controlled isotonic condition.
597 However, there were no differences in any markers of physiological strain between
598 conditions. Therefore, it may be that the regulation of exercise behaviour like power output is
599 not directly related to physiological parameters but may operate via the perception of effort.

600 In addition, the present study also aimed to investigate the changes in power output
601 [behavioural] and cerebral haemodynamics [cognitive] as indicators of the self-regulatory
602 strategies that were used to maintain a fixed perceived effort during conditions of pain
603 (hypertonic) or a control (isotonic). However, no significant condition \times time interactions
604 were detected for power output, physiological, or cerebral oxygenation markers. Therefore, it
605 was concluded that pain impacts the self-regulation of fixed perceived effort exercise, as
606 differences in power output mainly occurred when pain ratings were higher after hypertonic
607 versus isotonic saline administration.

608 An emphasis in our discussion highlights the potential impacts our approach may
609 have for the conclusions on pain's effect of perceived effort and subsequent exercise
610 behaviour. Furthermore, we pose potential avenues for future research to account for the
611 shortcomings of our approach and other ways that physical and cognitive effort contributions
612 operate during self-regulated exercise tasks.

613

614 DATA AVAILABILITY

615 Data is available upon request from the corresponding author in raw and analysed forms.
616 Data can be seen at: <https://www.doi.org/10.17605/OSF.IO/3JVU2> with some additional
617 materials related to the study provided.

618

619 SUPPLEMENTARY MATERIALS

620 Some supplementary materials can also be found at:
621 <https://www.doi.org/10.17605/OSF.IO/3JVU2>

622

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632

633 DISCLOSURES

634 All authors declare that there are no competing interests with the study and content of this
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638

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640 No disclaimers are required as part of this work.

641

642 AUTHOR CONTRIBUTION

643 Author CAO contributed to the development, data collection, analysis, writing and editing of
644 the study manuscript. Authors RN and SAS contributed to the data collection and editing of
645 the study manuscript. Author CLF contributed to the development of the study. Author ARM
646 contributed to the development of the study, data collection and editing of the study
647 manuscript. All authors have read and approved the submitted manuscript.

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829

830 FIGURE LEGENDS

831 Figure 1. Visual representation of Study 3 protocols. W represents power output. \wedge indicates
 832 affective valence and self-efficacy measurements. \blacklozenge represents blood lactate measurements.
 833 \ast represent rating of perceived effort (RPE) measurements. fNIRS represents functional near
 834 infrared spectroscopy measures.

835 Figure 2. Mean group (thick line) and individual (thin lines) power output data during fixed
 836 perceived effort trials. Significant condition (\ast) and time (§) effects illustrated. Error bars
 837 denote standard deviations from the mean.

838 Figure 3. Mean group (thick line) and individual (thin lines) (a) heart rate, (b) relative oxygen
 839 uptake ($\dot{V}O_2 \cdot \text{kg}^{-1}$), (c) minute ventilation (\dot{V}_E), (d) breathing frequency cardiorespiratory data
 840 during fixed perceived effort trials. Significant condition (\ast), time (§), and condition \times time
 841 (\dagger) effects illustrated. Error bars denote standard deviations from the mean.

842 Figure 4. Mean group (thick line) and individual (thin lines) blood lactate responses during
 843 fixed perceived effort exercise. Error bars denote standard deviations from the mean.

844 Figure 5. Mean group (thick line) and individual (thin lines) (a) oxyhaemoglobin ($\Delta O_2\text{Hb}$),
 845 (b) deoxyhaemoglobin (ΔHHb), (c) total haemoglobin (ΔtHb) changes during fixed perceived

846 effort trials. Significant condition (*) and time (§) effects illustrated. Error bars denote
 847 standard deviations from the mean.

848 Figure 6. Mean group (thick line) and individual (thin lines) (a) affective valence, (b) pain
 849 intensity perceptual responses during fixed perceived effort trials. Significant condition (*),
 850 time (§), and condition × time (‡) effects illustrated. Error bars denote standard deviations
 851 from the mean.

852

853 TABLES

Table 1. Frequency of descriptors selected and mean ± SD subclass scores for pain quality.

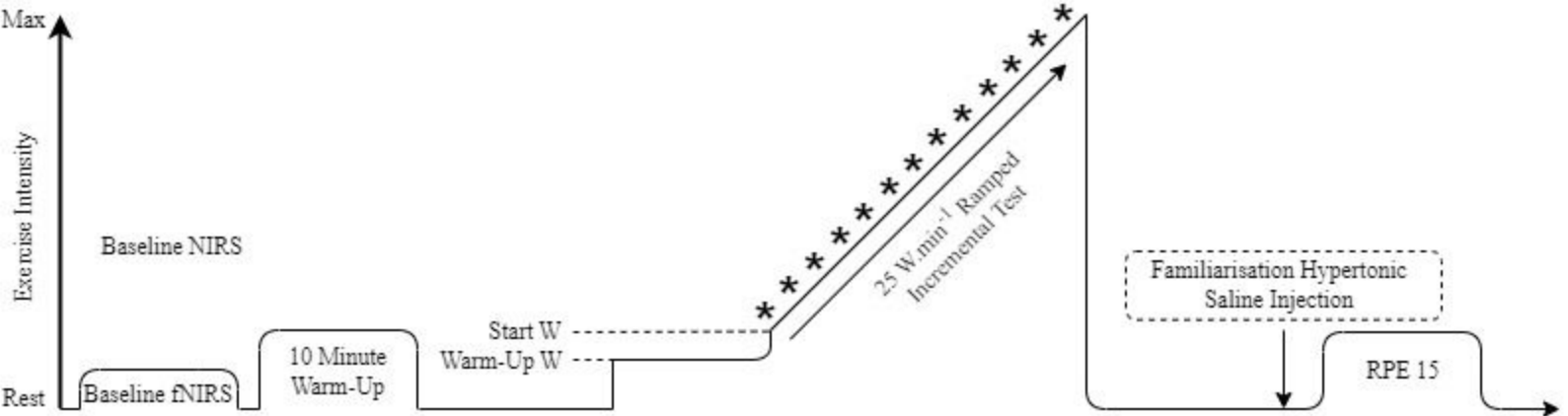
<i>Subclass</i>		Hypertonic	Isotonic	
<i>Sensory</i>		Hot (40%) Sharp (50%) Tender (60%) Burning (40%) Throbbing (50%) Tugging (50%)	Hot (60%) Sharp (50%) Tender (60%) Pricking (40%) Dull (40%) Aching (40%) Pulling (50%) Tingling (50%) Pressing (60%)	
	SRI	17 ± 5	14 ± 6	#
<i>Affective</i>		Gruelling (40%) Tiring (70%) Sickening (40%) Fearful (40%) Wretched (40%)	Gruelling (40%) Tiring (70%)	
	SRI	5 ± 3	3 ± 2	‡
<i>Evaluative</i>	SRI	Intense (60%) 3 ± 1	Annoying (40%) 2 ± 2	*#
<i>Miscellaneous</i>		Tight (40%) Radiating (40%)	Tight (80%) Spreading (40%) Nagging (50%)	
	SRI	5 ± 2	4 ± 2	*#
	<i>PRI (T)</i>	30 ± 8	22 ± 11	*‡

Legend: Subclass Rating Index (SRI); Pain Rating Index Total (PRI) all presented as mean ± SD.
 * denotes significant difference between conditions, # denotes a moderate effect size, ‡ denotes a large effect size.

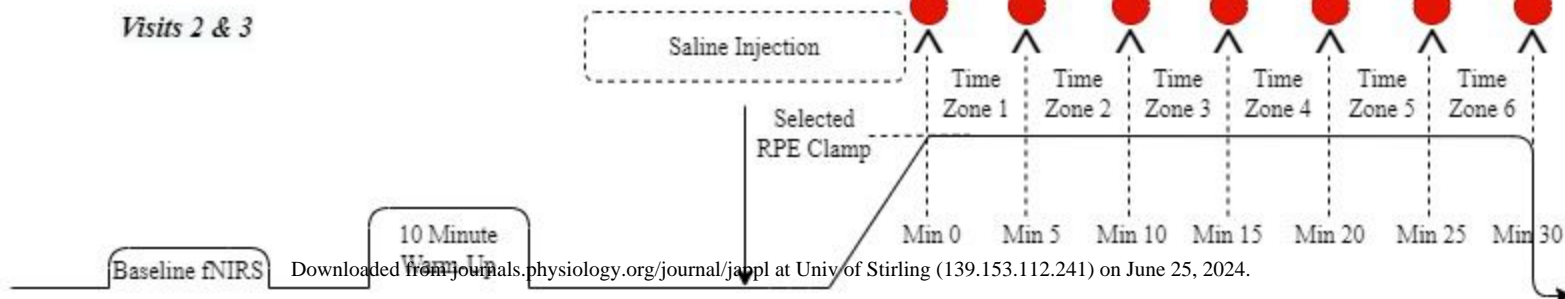
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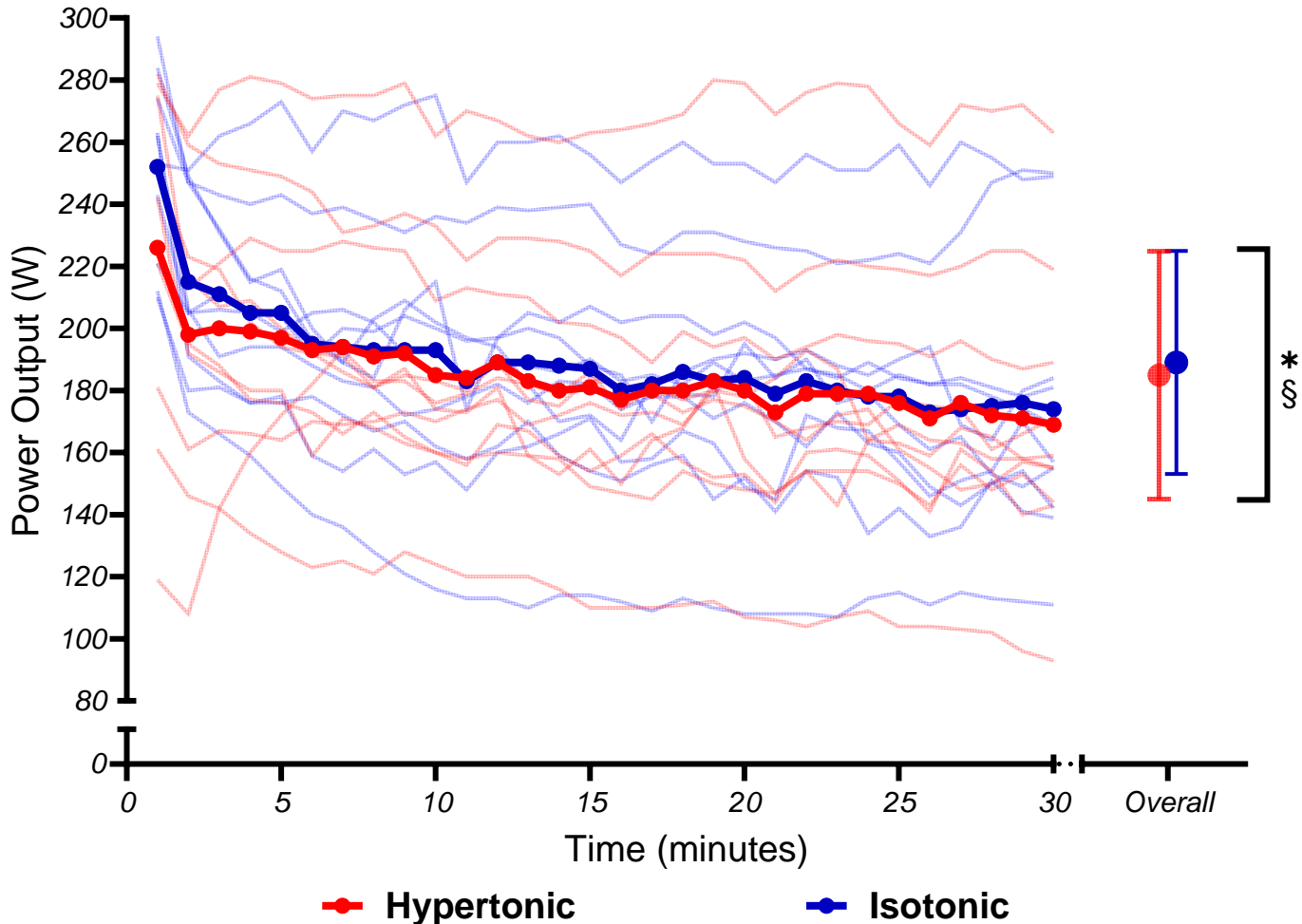
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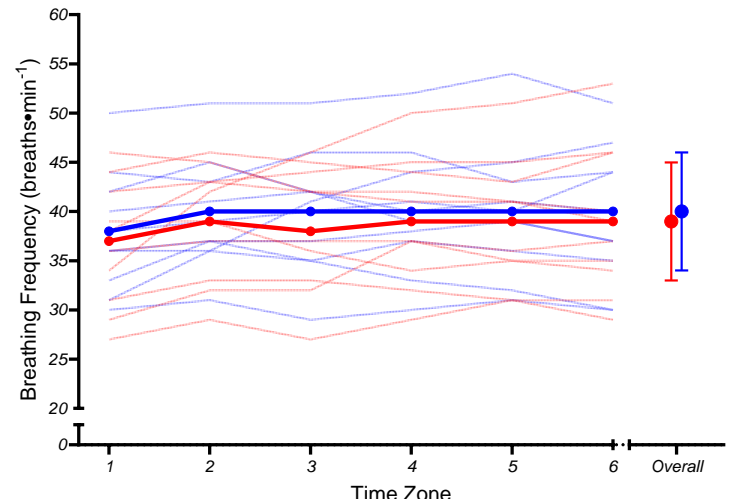
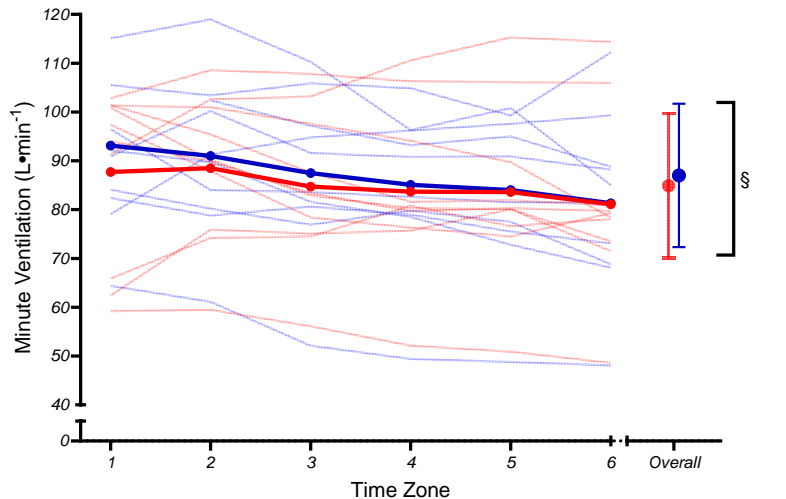
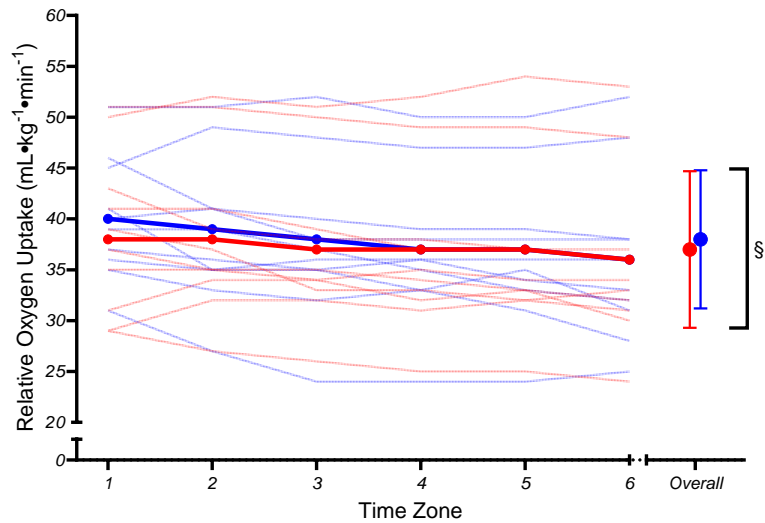
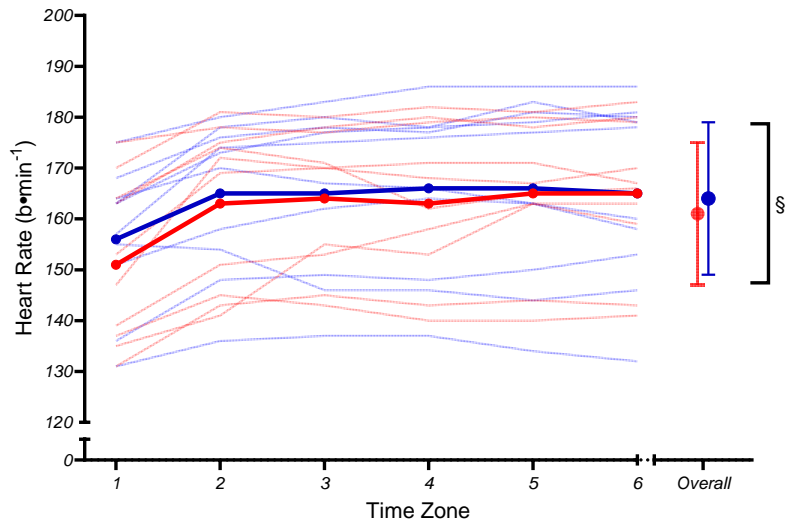
Visit 1

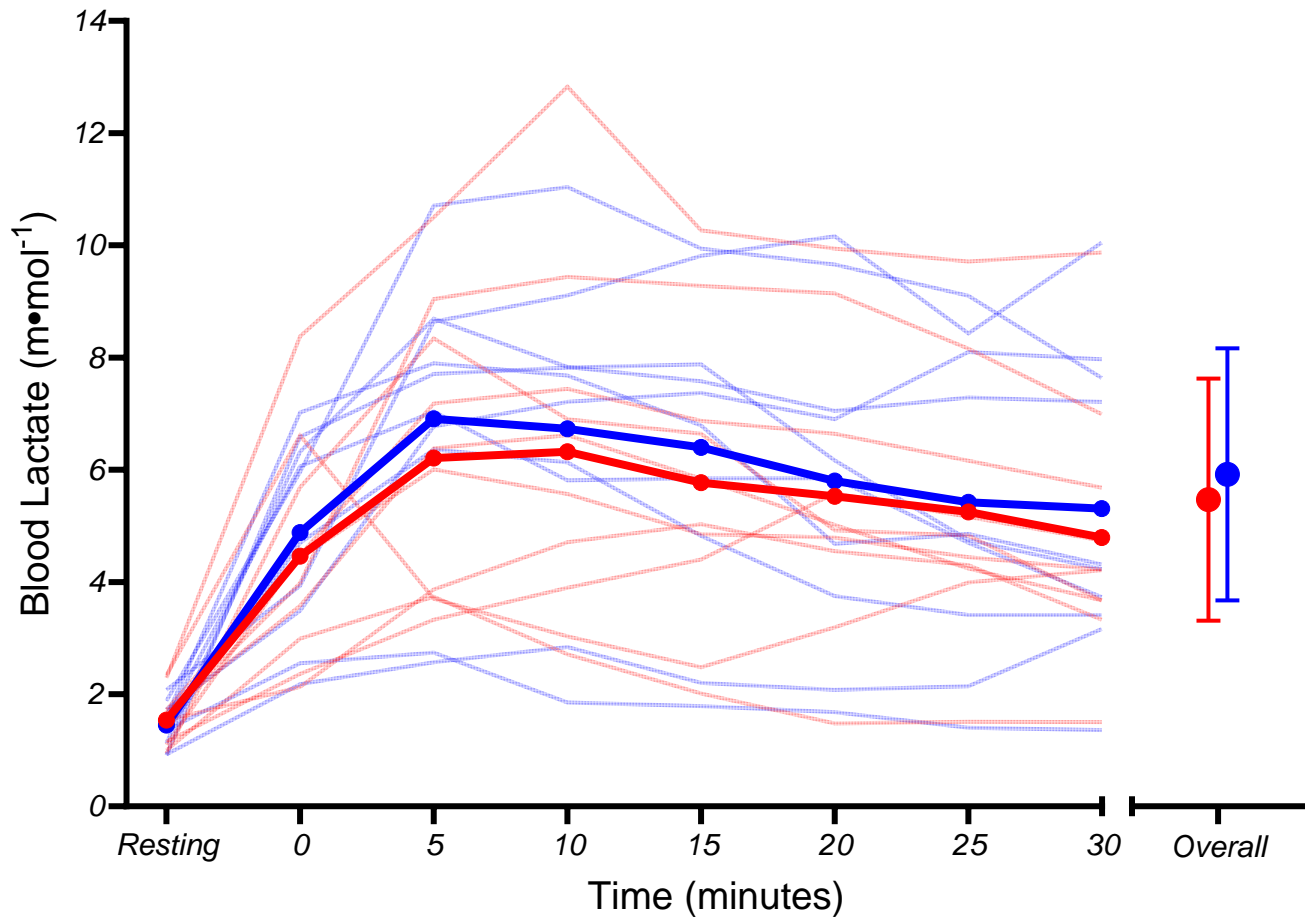


Visits 2 & 3



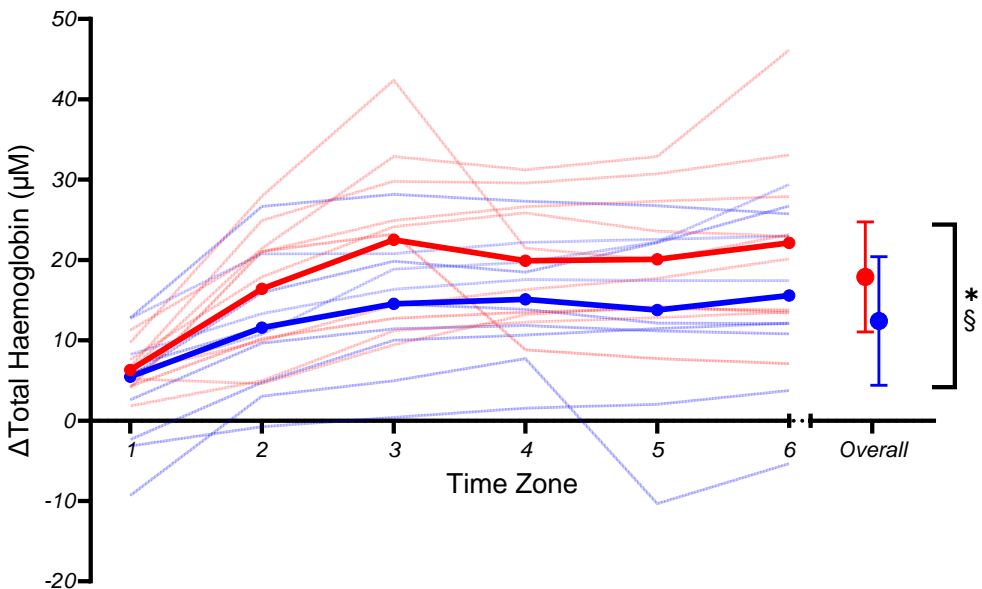
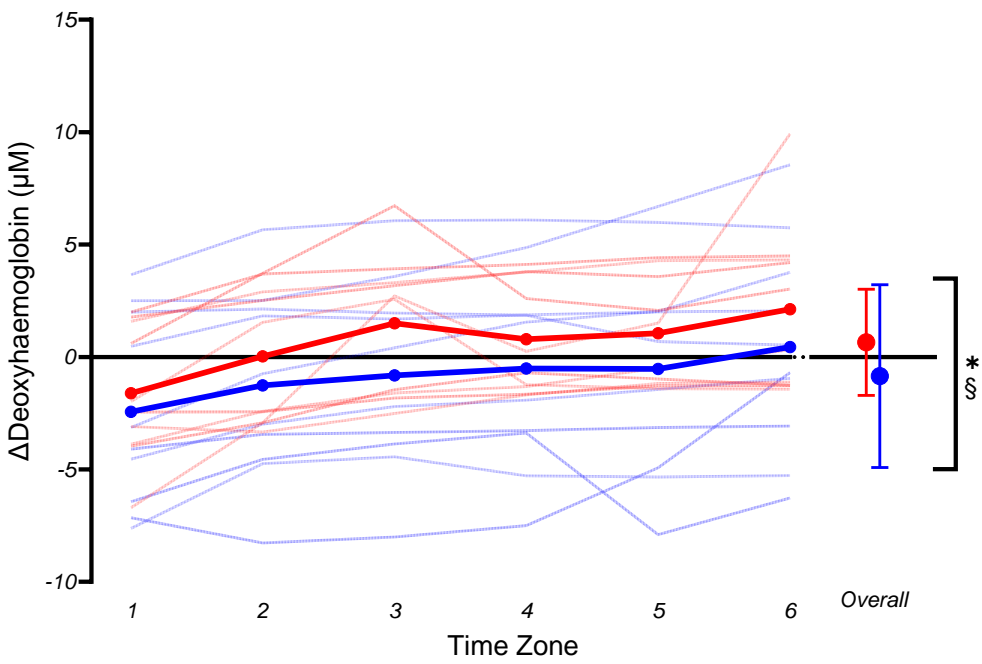
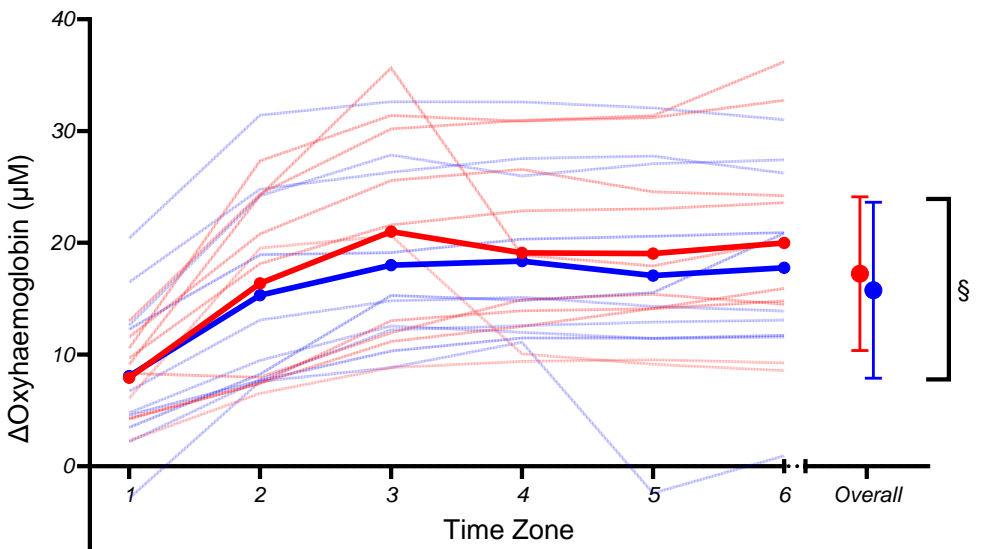




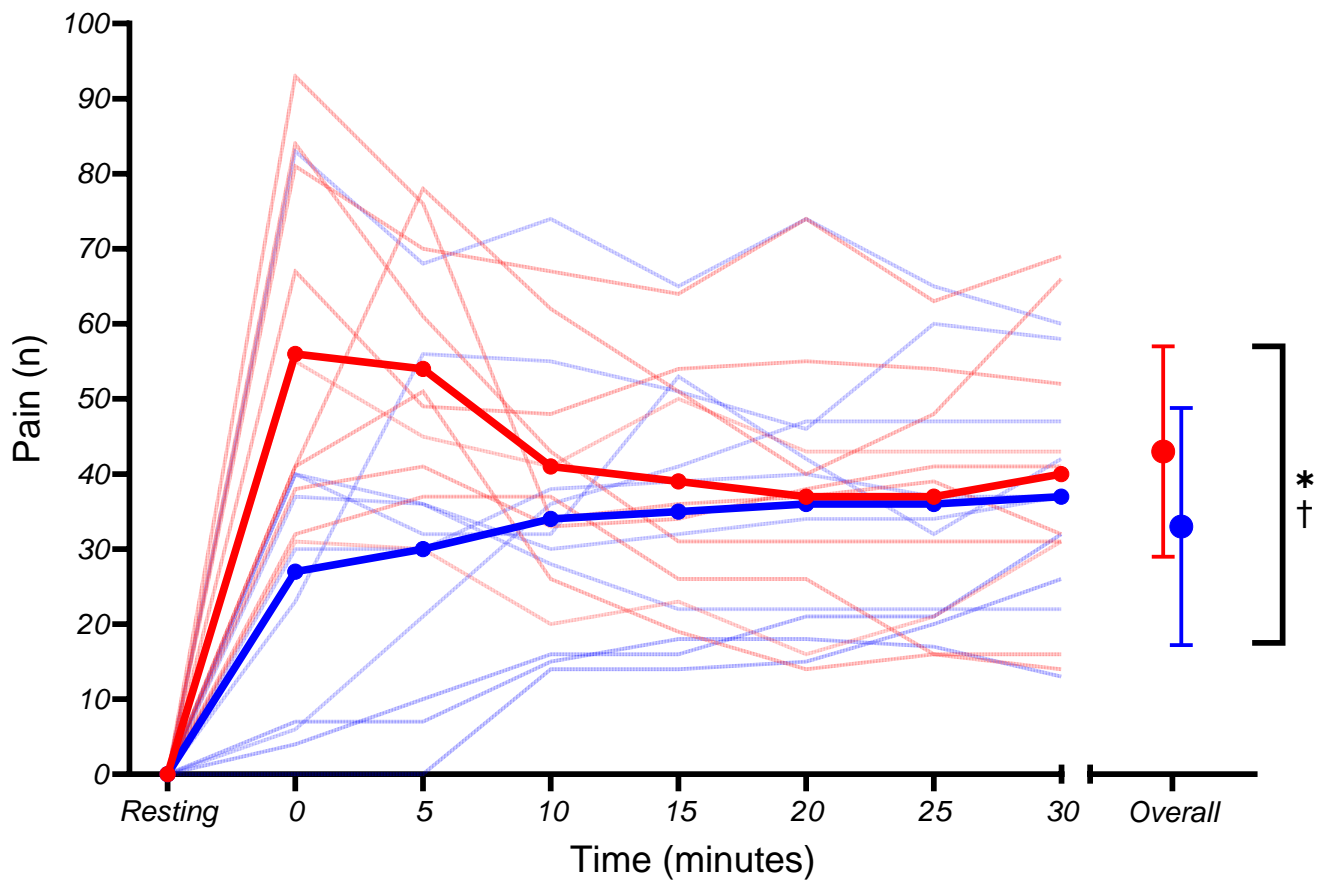
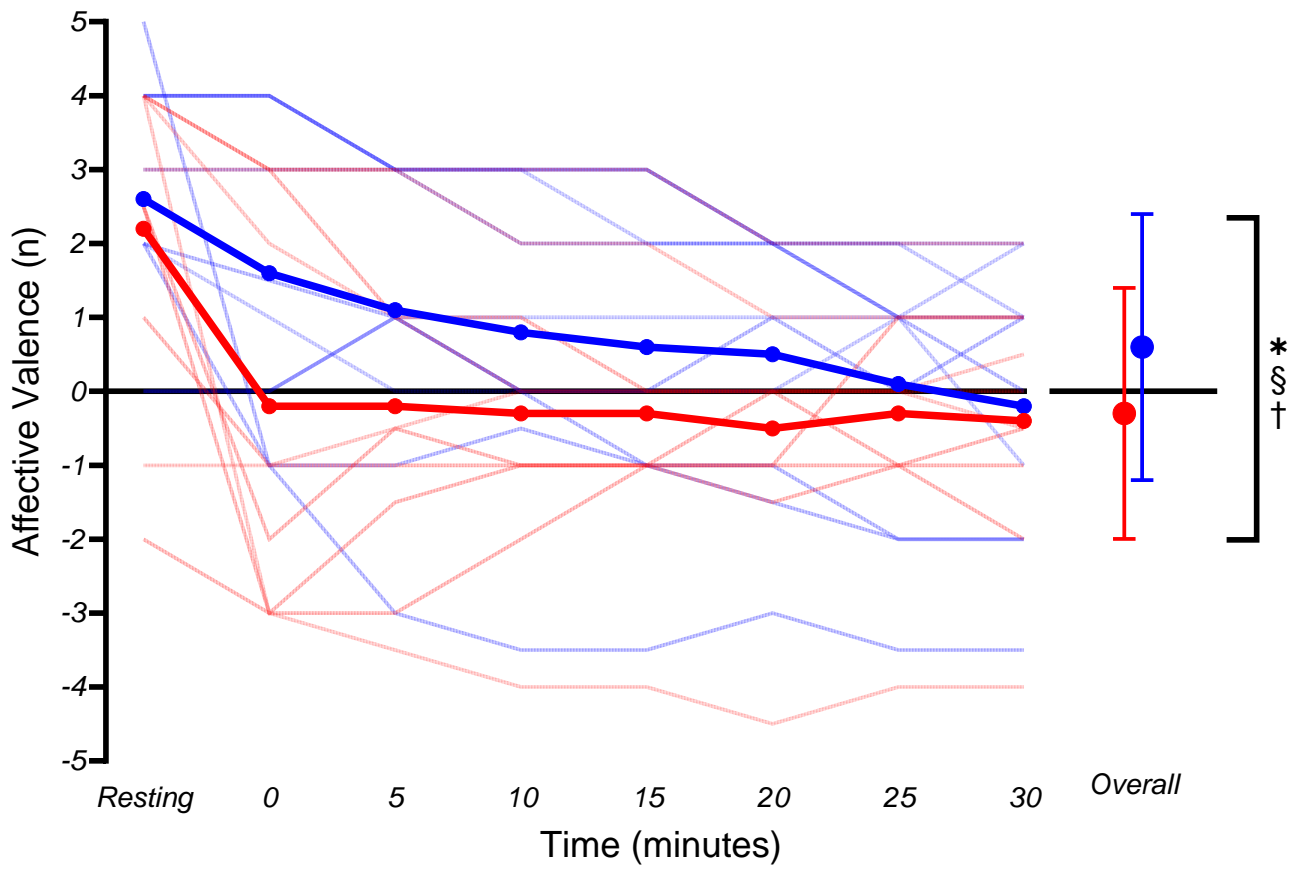


Hypertonic

Isotonic



—●— Hypertonic —●— Isotonic



—●— Hypertonic —●— Isotonic

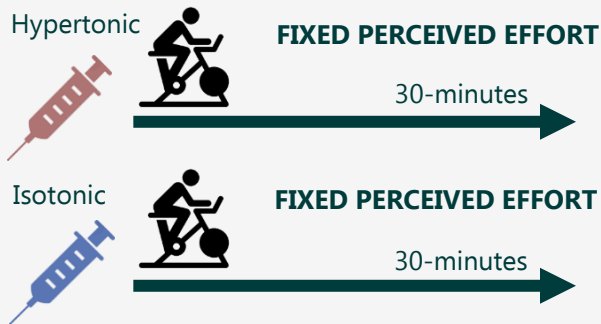
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	<i>PRI (T)</i>	30 \pm 8	22 \pm 11	*‡

Legend: Subclass Rating Index (SRI); Pain Rating Index Total (PRI) all presented as mean \pm SD. * denotes significant difference between conditions, # denotes a moderate effect size, ‡ denotes a large effect size.

Reduced fixed perceived effort power output with muscle pain

METHODS



Measures

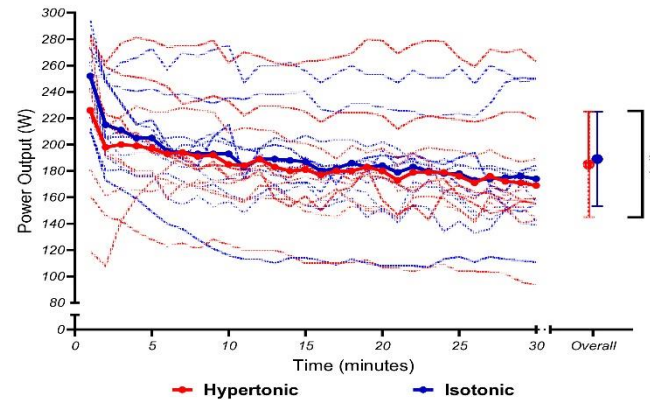
Power Output

Cardiorespiratory Measures

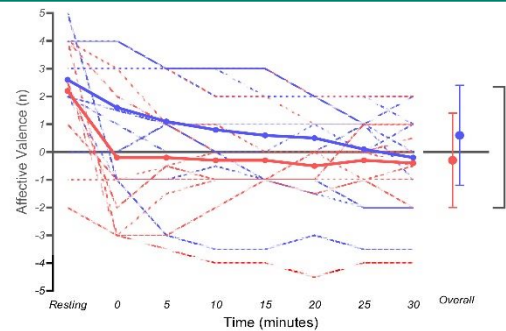
Cerebral Oxygenation

Perceptual Measures

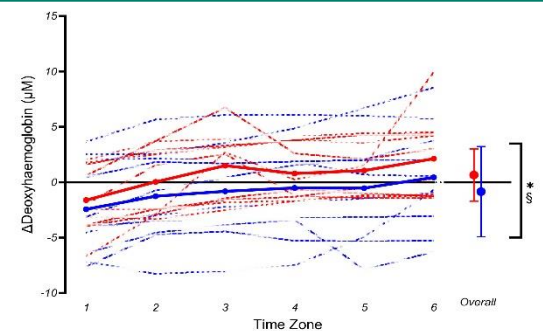
OUTCOME



Lower power output in the hypertonic versus isotonic condition



Lower affective valence and earlier decreases in the hypertonic versus isotonic condition



Higher deoxyhaemoglobin change from baseline in the hypertonic versus isotonic condition

Changes in power output, affect, and deoxyhaemoglobin between hypertonic (red) and isotonic (blue) conditions. Significant condition (*), time (§), and condition x time (†) effects shown.

CONCLUSION

- Increased muscle pain from a hypertonic saline injection causes a lower power output at a fixed perception of effort compared to placebo-controlled isotonic condition.

- Differences in power output coincide with when elevated muscle pain is experienced.