Dynamics of the power-duration relationship during prolonged endurance exercise and influence of carbohydrate ingestion

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Abstract

We tested the hypotheses that the parameters of the power-duration relationship, estimated as the end-test power (EP) and work done above EP (WEP) during a 3-min all out exercise test (3MT), would be reduced progressively following 40 min, 80 min and 2 h of heavy-intensity cycling, and that carbohydrate (CHO) ingestion would attenuate the reduction in EP and WEP. Sixteen participants completed a 3MT without prior exercise (control), immediately after 40 min, 80 min and 2-h of heavy-intensity exercise while consuming a placebo beverage, and also after 2-h of heavy-intensity exercise while consuming a CHO supplement (60 g/h CHO). There was no difference in EP measured without prior exercise (260 ± 37 W) compared to EP following 40 min (268 ± 39 W) or 80 min (260 ± 40 W) of heavy-intensity exercise; however, after 2-h, EP was 9% lower compared to control (236 ± 47 W; $P<0.05$). There was no difference in WEP measured without prior exercise (17.9 ± 3.3 kJ) compared to after 40 min of heavy-intensity exercise (16.1 ± 3.3 kJ), but WEP was lower ($P<0.05$) than control after 80 min (14.7 ± 2.9 kJ) and 2-h (13.8 ± 2.7 kJ). Compared to placebo, CHO ingestion negated the reduction of EP following 2-h of heavy-intensity exercise (254 ± 49 W) but had no effect on WEP (13.5 ± 3.4 kJ). These results reveal a different time course for the deterioration of EP and WEP during prolonged endurance exercise and indicate that EP is sensitive to CHO availability.

New and Noteworthy

The parameters of the power-duration relationship (critical power, CP, and the curvature constant, W') have typically been considered to be static. Herein, we report the time course for reductions in CP and W', as estimated using the 3-min all-out cycle test, during 2 h of heavy-intensity exercise. We also show that carbohydrate ingestion during exercise preserves CP, but not W', without altering muscle glycogen depletion. These results provide new mechanistic and practical insight into the power-duration curve and its relationship to exercise-related fatigue development.

Introduction

The parameters of the hyperbolic power-duration relationship, the critical power (CP) and the curvature constant (W′, which represents a fixed work capacity above CP), are important determinants of endurance exercise performance (25, 32, 50). CP is considered to be a metabolic or fatigue threshold which separates the 'heavy' from the 'severe' exercise intensity domains (8, 40, 41). In the heavy-intensity domain (<CP), intramuscular metabolic homeostasis is maintained and pulmonary $\dot{V}O_2$ attains a delayed steady-state (4, 26, 41). In contrast, in the severe-intensity domain (>CP), intramuscular metabolic homeostasis is not achieved, a $\dot{V}O_2$ 'slow component' develops that drives $\dot{V}O_2$ inexorably to its maximum, and exercise tolerance is predictably limited as a function of the power output above CP and the size of the W′ (4, 26, 40, 41).

The CP and W′ are conventionally estimated using 3-5 severe-intensity prediction trials in which constant power outputs are maintained until the limit of tolerance, with the asymptote (representing CP) and the curvature constant (representing W′) of the power-time relationship subsequently being determined mathematically (30, 41, 48). More recently, a 3-min all-out cycle test against fixed resistance (3MT) has been developed, during which external power output declines hyperbolically with time, which permits a more expeditious assessment of CP and W′ (7, 48). The 3MT has been shown to provide valid and reliable estimates of CP and W′, where the mean power output during the last 30 s of the test (end-test power, EP) represents CP, and the work completed above EP (WEP) represents W′ (7, 38, 48). We have recently reported that the 3MT continues to provide valid (13) and reasonably reliable (12) estimates of CP and W′ following 2 h of heavy-intensity cycle exercise. In these studies we found that prolonged endurance exercise consistently and profoundly altered the power-time relationship, with CP and EP falling by ~10% and W′ and WEP falling by ~20% (12, 13). These results have important implications for our understanding of fatigue development, and

for performance prognosis, during endurance exercise. At present, however, the time course over which EP and WEP deteriorate during prolonged endurance exercise is not known. Elucidating the dynamic changes in EP and WEP *during* prolonged exercise may provide insight into the determinants of fatigue and underpin the development of interventions to attenuate the decline in performance during such exercise.

The mechanistic bases for the reductions in EP and WEP after 2 h of heavy-intensity exercise are likely multifactorial, and may include muscle glycogen depletion (12, 13, 30). It is well known that glycogen depletion increases with the duration of heavy-intensity exercise (17, 18). If glycogen depletion impacts the power-duration relationship, then reductions in EP and WEP would be expected to become much more substantial following longer, compared to shorter, bouts of heavy-intensity exercise. Carbohydrate (CHO) ingestion is known to benefit prolonged endurance exercise performance by sparing muscle [glycogen], better maintaining blood [glucose] and/or providing stimulation to the central nervous system via the 'pleasure and reward' centers of the brain (21, 23). During long-duration events, which deplete muscle glycogen stores, exogenous CHO intake is particularly important to maintain high rates of CHO oxidation (20), with the greatest performance enhancement observed at ingestion rates of 60-80 g/h (44). While the reductions in EP and WEP were not significantly correlated with changes in muscle [glycogen] following 2 h of heavy-exercise in our previous study (13), the relationship between muscle CHO availability (in the muscle and circulation) and the power-time relationship is likely to be complex, and it is possible that CHO ingestion may offset the reductions in EP and WEP reported following 2 h of heavy-intensity cycling (12, 13)

The purpose of this study was to: 1) investigate the dynamic changes in EP and WEP during and following 2 h of heavy-intensity exercise; and 2) determine the effect of 60 g/h of CHO ingestion, compared to a placebo, on EP and WEP during and following 2 h of heavy-intensity exercise. We hypothesized that EP and WEP would be reduced progressively

following 40 min, 80 min and 2 h of heavy-intensity exercise, and that CHO ingestion would attenuate the declines in EP and WEP after 2 h of heavy-intensity exercise.

Methods

This paper reports the results of two experiments. The first experiment was conducted to investigate possible changes in EP and WEP after 40 min, 80 min and 2 h of heavy-intensity exercise and the second experiment was conducted to investigate the effect of CHO ingestion on changes in EP and WEP after 2 h of heavy-intensity exercise.

Participants

Experiments I and II were conducted on the same group of participants. Sixteen males (mean ± SD: age = 34 ± 6 years, height = 1.78 ± 0.07 m, body mass = 79.1 ± 7.6 kg, peak O_2 uptake ($\dot{V}O_{2peak}$) = 52.5 ± 7.3 $mL \cdot kg^{-1} \cdot min^{-1}$) took part in the experiments. They were all competitive athletes (comprising two runners, six cyclists, four triathletes, three Crossfit athletes, and one squash player) but were not professional/elite. The participants were instructed to arrive at the laboratory in a rested and hydrated state, to avoid alcoholic drinks and strenuous exercise for 24 h prior to testing, and to maintain their habitual diet throughout the study. The participants recorded their diet for 24 h prior to the first experimental session and replicated this prior to each subsequent visit. The experimental procedures were approved by the Institutional Research Ethics Committee at the University of Exeter and informed consent was obtained from each participant prior to testing. One participant did not consent to having muscle biopsies taken in Experiment II. All exercise tests were separated by a minimum of 24 h but the tests in which the 3MT was completed following a bout of heavy-intensity exercise (see below for more details) were separated by at least 72 h (i.e., 7 ± 4 days).

Experimental design

Participants reported to the laboratory on 7 occasions over a 7-week period (± 2 weeks). The tests included a ramp incremental exercise test for the determination of $\dot{V}O_{2peak}$ and gas exchange threshold (GET), a 3MT familiarisation trial, a 3MT performed in a rested state which served as a control (C-3MT), and on subsequent visits: a 3MT preceded by 40 min (40-3MT), 80 min (80-3MT) or, on two occasions, 2 h of heavy-intensity exercise. A placebo drink was consumed during the 40-3MT, 80-3MT and during one of the 2 h visits (120-$3MT_{PLA}$), while CHO was consumed during one 2 h visit (120-$3MT_{CHO}$). The 120-$3MT_{PLA}$ visit was used for both experiments. The other trials were administered in a randomized and counterbalanced order.

All exercise tests were conducted using the same electrically-braked cycle ergometer (Lode Excalibur Sport, Groningen, Netherlands). During all tests, except the 3MTs, the participants cycled at a self-selected pedal rate (70-90 rpm). For each participant, the self-selected pedal rate used in the ramp incremental test was recorded and replicated in each subsequent visit. The ergometer seat and handlebar configuration were adjusted for comfort during the first visit and were recorded and then replicated in subsequent visits. Exercise tests took place in an air-conditioned laboratory with ambient temperature of 20°C and relative humidity of 60%.

Determination of $\dot{V}O_{2peak}$ and gas exchange threshold

During visit 1 the participants completed a ramp incremental exercise test. The ramp protocol consisted of a 3-min baseline of pedalling at 20 W after which the power output was increased by 30 W/min until the participant was unable to continue. The limit of tolerance was determined when cadence fell >10 rpm below the target cadence for more than 5 s despite strong verbal encouragement. $\dot{V}O_{2peak}$ was determined as the highest 30-s mean value recorded during the test. The GET was defined according to the procedures of Beaver et al. (2). The GET and $\dot{V}O_{2peak}$ were used to normalize the fixed resistance for the 3MTs.

148 *Three-min all-out tests (3MTs)*

149 The resistance for the 3MT was applied using the linear factor function of the ergometer and
150 was calculated as: linear factor = power output/preferred cadence^2 where the power was
151 50%Δ (i.e., GET plus 50% of the interval between the GET and ramp test peak power output).
152 Visit 2 (familiarization test) and visit 3 consisted of a C-3MT performed with no prior
153 exercise. The 3MT protocol began with a 3-min baseline of pedalling at 20 W. During visits
154 4-7, a 60 s pause was administered after the constant power output bout, during which the
155 participants had 30 s of passive recovery and then were instructed to cycle for the last 30 s
156 against a resistance of 20 W. This was done to replicate the 1-min pause that was required to
157 obtain a muscle biopsy in Experiment II. A 5 s countdown was given prior to the 3MT and the
158 participant was instructed to increase cadence to ~110-120 rpm. Strong verbal encouragement
159 was then given for an all-out effort from the onset of the 3MT. Participants were not informed
160 of the elapsed time. Instructions were given to reach peak power output as quickly as possible
161 and to maintain the all-out effort throughout the test. The EP was subsequently calculated as
162 the mean power output over the last 30 s of the test, and the WEP was calculated as the work
163 done above EP during the 3MT, i.e. the mean power output above EP for each second of the
164 test was multipled by the number of seconds of exercise performed above EP to compute the
165 total work done above EP (48). The results of the 3MT were deemed valid only when the
166 $\dot{V}O_{2peak}$ attained exceeded 95% of the $\dot{V}O_{2peak}$ determined in the initial ramp incremental test.
167 Results determined from the C-3MT, 40-3MT, 80-3MT, 120-3MT$_{PLA}$ and 120-3MT$_{CHO}$ were
168 consequently termed C-EP and C-WEP, 40-EP and 40-WEP, 80-EP and 80-WEP, 120$_{PLA}$-EP
169 and 120$_{PLA}$-WEP, 120$_{CHO}$-EP and 120$_{CHO}$-WEP, respectively.

170 *Experiment I: Effect of 40 min, 80 min and 2 h of heavy-intensity exercise on the parameters*
171 *of the power-duration relationship*

The constant power output applied during the 40 min, 80 min and 2 h heavy-intensity exercise bouts was calculated as the power output at the GET plus 25% of the difference between the GET and C-EP (25%Δ1; 12). Experiment I consisted of three heavy-intensity exercise bouts of 40 min, 80 min and 2 h followed by a 3MT. The visits began with a 3-min baseline of pedaling at 20 W for attainment of baseline measurements, after which the power output abruptly increased to the target power output for 40 min, 80 min or 2 h. Participants were instructed to hold their desired cadence throughout the constant power output bout. Participants were provided with a 100-ml opaque plastic bottle containing 1 mL of apple-flavored sweetener (Myprotein.Co, UK), with no caloric value, added to 94 mL of water to ensure that supplements were indistinguishable from, and of equal volume to, the CHO solution used in Experiment II (see below). The first bottle was given at -3 min, as participants wore a mask between -3 – 15 min for measurements of pulmonary gas exchange, and thereafter a bottle was given every 15 min. During the 40-3MT the last beverage was consumed at 30 min; during the 80-3MT visit the last beverage was consumed at 75 min; and during the 120-3MT$_{PLA}$ the last beverage was consumed at 105 min. A clock with time remaining was visible during the constant power output bout and participants were allowed to listen to music; however, both were withdrawn 2 min prior to the 3MT. Participants were instructed to stop pedalling for 30 s after the constant power output bout, and at 30 s, they were instructed to start cycling again at 20 W. This was administered to ensure the same rest period was provided between the four constant power output tests (both experiments) and the 3MTs and so that muscle biopsies could be taken in the 30 s window during the 2 h visits for experiment II. Pulmonary gas exchange data were attained at the following time points during the 40-3MT visit: -3-15, 25-30 and 35-40 min; the 80-3MT visit: -3-15, 25-30, 55-60 and 75-80 min; and the 120-3MT$_{PLA}$ visit: -3-15, 25-30, 55-60, 85-90 and 115-120 min; and continuously throughout the 3MT. A blood sample from a fingertip was collected every 20

min during the constant power output bouts for the analysis of [lactate] and [glucose]. Heart rate (HR) was recorded (Garmin FR70, Garmin Ltd, Schaffhausen, Switzerland) every 5 s during all visits. Before and after the test, participants were weighed in minimal clothing to assess changes in body mass.

Experiment II: Effect of CHO ingestion on the parameters of the power-duration relationship

The 120-3MT$_{PLA}$ and 120-3MT$_{CHO}$ visits included 2 h of heavy-intensity exercise immediately followed by a 3MT. The 120-3MT$_{PLA}$ exercise trial was the same as that in Experiment I. The procedures for the 120-3MT$_{CHO}$ visit were identical to the 2 h visit described in Experiment I, except for the ingestion of CHO. During the 120-3MT$_{CHO}$ visit participants consumed 60g/h of CHO (Maurten drink mix 320, Biotech center, Gothenburg, Sweden). Participants were given the same bottle as described in Experiment I, containing 94 ml of Maurten drink mix (15g of CHO), every 15 min during the constant power output bout. A muscle biopsy was taken prior to exercise and again at 120 min during the 30 s rest period between the constant-power-output bout and the 3MT.

Measurements

Pulmonary gas exchange. Pulmonary gas exchange was measured breath-by-breath and averaged over 10 s periods during all visits. Participants wore a face mask (Hans Rudolf 7450 Series V2^{TM} Mask, CareFusion, Germany) and inspired and expired gas volume and gas concentration signals were sampled continuously at 100 Hz (Vyntus, CareFusion, Germany) via a capillary line connected to the mask. These analysers were calibrated before each test with gases of known concentration and the turbine volume transducer was calibrated using a 3-L syringe (Hans Rudolph, MO). The volume and concentration signals were time-aligned by accounting for the delay in capillary gas transit and analyzer rise time relative to the volume signal. The baseline $\dot{V}O_2$ period during all visits were defined as the mean value

recorded over the final minute during the 3-min warm up period at 20 W. Fat and CHO oxidation rates were calculated from $\dot{V}O_2$ and $\dot{V}CO_2$ using the following stoichiometric equations with the assumption that protein oxidation during exercise did not change (24):

CHO oxidation ($g \cdot min^{-1}$) = [4.21 ($\dot{V}CO_2$) – 2.692 ($\dot{V}O_2$)]

Fat oxidation ($g \cdot min^{-1}$) = [1.695 ($\dot{V}O_2$) – 1.701 ($\dot{V}CO_2$)]

Muscle biopsies. Muscle samples were obtained from one incision from the medial region of the *m. vastus lateralis* under local anesthesia (1% lidocaine) using the percutaneous Bergström needle biopsy technique under suction (3). Muscle samples were taken at rest and immediately post 2 h of heavy-intensity exercise during the 120-3MT_{CHO} and 120-3MT_{PLA} visits. The post-exercise biopsies were taken while participants remained on the cycle ergometer and were typically collected within 10 s of the completion of the exercise bout. Biopsy samples were immediately frozen in liquid nitrogen and stored at −80°C for subsequent analysis.

Muscle glycogen concentration. Muscle samples were freeze-dried prior to dissection from connective tissue, fat and blood. Muscle glycogen was extracted from ~1 mg d.w. muscle and hydrolysed to glucose units in 1M HCl at 95°C for 3 h. The addition of hexokinase catalyzed the reaction of glucose with adenosine triphosphate to glucose-6-phosphate, and then to 6-P-gluconolactone with NADH+ in the presence of G-6-PDH enzyme, producing the fluorescent detectable NADPH (28). Reactions were measured on a Fluoroskan (Fluoroskan™ Microplate Fluorometer, ThermoFisher Scientific, Mass. USA), with Excitation 355 nm and Emission 460 nm filters. Glycogen was reported in units of mmol of glucose per kg dry muscle.

Blood analyses. All fingertip blood samples (~25 µl) (visit 4-7) were collected into capillary tubes and analysed within 60 s for blood [lactate] and [glucose] using an automated lactate analyser (Stat2300, Yellow Spring Instrument, Yellow Springs, OH).

Statistical analysis

For experiment I, one-way ANOVAs with repeated measures were used to assess differences over time during the 40-3MT, 80-3MT and 120-3MT$_{PLA}$ tests in respiratory gas exchange variables, HR, blood [lactate] and blood [glucose]. To assess the difference in these physiological variables at common time points within the 40-3MT, 80-3MT and 120-3MT$_{PLA}$ a repeated measures ANOVA (condition x time) was used. One-way ANOVA with repeated measures was used to assess differences in EP, WEP, total work done (TWD), peak power output, $\dot{V}O_{2peak}$, and body mass between C-3MT, 40-3MT, 80-3MT and 120-3MT$_{PLA}$.

For experiment II, one-way ANOVAs with repeated measures were used to assess differences in the EP,WEP, TWD, peak power output, muscle glycogen concentration and $\dot{V}O_{2peak}$, as well as differences in respiratory gas exchange variables, blood [glucose] and blood [lactate] between C-3MT, 120-3MT$_{PLA}$ and 120-3MT$_{CHO}$. Differences in the change in muscle [glycogen] (from rest to post-exercise) between the 120-3MT$_{PLA}$ and 120-3MT$_{CHO}$ visits were analysed using a paired sample t-test. The relationships between the change in muscle [glycogen] and the changes in in EP and WEP were determined using Pearson product-moment correlation coefficients.

Statistical significance was accepted at $P<0.05$. Significant interactions and main effects were followed up with Bonferroni post hoc tests. Data are reported as mean ± SD.

Results

The $\dot{V}O_{2peak}$ in the ramp incremental test was 4.12 ± 0.45 L·min^{-1}, the peak power output was 360 ± 41 W and the GET was 132 ± 7 W. The 25%Δ1 for the 2 h constant power output bouts was 164 ± 28 W.

Experiment I: Dynamic changes in the parameters of the power-duration relationship following 40 min, 80 min and 2 h of heavy-intensity exercise

During the 40-3MT visit, the relative intensity did not change between 10-15 min (63% ± 7% $\dot{V}O_{2peak}$) and 35-40 min (63% ± 5% $\dot{V}O_{2peak}$; $P>0.05$). The relative intensity increased during both the 80-3MT visit (from 10-15 min: 63% ± 7% $\dot{V}O_{2peak}$ to 75-80 min: 65% ± 5 % $\dot{V}O_{2peak}$; $P<0.05$) and the 120-3MT$_{PLA}$ visit (from 10-15 min: 64% ± 8% $\dot{V}O_{2peak}$ to 115-120 min: 68% ± 7% $\dot{V}O_{2peak}$; $P<0.001$). HR increased over time during all the constant power output bouts (Table 1). During the 40-3MT visit, RER did not change significantly from 10-15 min to 35-40 min; however, RER decreased during both the 80-3MT and the 120-3MT$_{PLA}$ visits (Table 1).

Body mass decreased during the 40-3MT, 80-3MT and 120-3MT$_{PLA}$ trials (Table 1). Body mass was lower post-exercise in the 120-3MT$_{PLA}$ visit compared to the 80-3MT and 40-3MT visits with no differences between the 80-3MT and the 40-3MT visit (Table 1).

The power output profiles during the C-3MT, 40-3MT, 80-3MT and 120-3MT$_{PLA}$ are shown in Fig. 1. There were no differences in $\dot{V}O_{2peak}$ between C-3MT (4.03 ± 0.40 L·min^{-1}), 40-3MT (4.19 ± 0.40 L·min^{-1}), 80-3MT (4.15 ± 0.41 L·min^{-1}), 120-3MT$_{PLA}$ (4.08 ± 0.51 L·min^{-1}) and the ramp incremental test ($P>0.05$). There was no differences in EP between C-EP (260 ± 37 W), 40-EP (268 ± 39 W) and 80-EP (260 ± 40 W; $P>0.05$). However, 120$_{PLA}$-EP was lower than EP in all the other conditions (236 ± 47 W; $P<0.05$; Fig. 2A). There was no difference in WEP between C-WEP (17.9 ± 3.3 kJ) and 40-WEP (16.1 ± 3.3 kJ), but both 80-WEP (14.7 ± 2.9 kJ; $P<0.05$) and 120$_{PLA}$-WEP (13.8 ± 2.7 kJ; $P<0.05$) were lower than C-WEP; 80-WEP was also lower than 40-WEP ($P<0.05$; Fig. 2B). Results for peak power output and TWD during the 3MTs are shown in Fig. 2C and Fig. 2D, respectively. Blood

293 [lactate] immediately after the 3MT was higher in the 40-3MT (8.8 ± 2.1 mM) compared to
294 the 120-3MT$_{PLA}$ visit (7.0 ± 2.1 mM; P<0.05) with no difference after any other 3MTs.

295 *Experiment II: Influence of CHO ingestion on changes in the power-duration relationship*
296 *following 2 h of heavy-intensity exercise*

297 In both the 120-3MT$_{PLA}$ and 120-3MT$_{CHO}$ tests, the relative intensity increased from ~64%
298 $\dot{V}O_{2peak}$ at 10-15 min to ~68% $\dot{V}O_{2peak}$ at 115-120 min (Fig. 3A). HR increased during both
299 120-3MT visits (Table 1). There was no difference in relative intensity or HR between the
300 120-3MT$_{CHO}$ and 120-3MT$_{PLA}$ visits at any time point (Table 1 and Fig. 3A). RER decreased
301 during both the 120-3MT$_{CHO}$ and 120-3MT$_{PLA}$ visits, but was higher at 115-120 min in the
302 120-3MT$_{CHO}$ trial compared to the 120-3MT$_{PLA}$ (Fig. 3B). CHO oxidation was higher in the
303 120-3MT$_{CHO}$ bout compared to the 120-3MT$_{PLA}$ bout at 85-90 and 115-120 min (Fig. 3C).
304 Blood [glucose] was higher during the 120-3MT$_{CHO}$ bout compared to 120-3MT$_{PLA}$ at all time
305 points at and beyond 40 min (Fig. 3D). There was no difference in blood [lactate] measured
306 during the 120-3MT$_{CHO}$ and 120-3MT$_{PLA}$ trials. However, blood [lactate] was higher post
307 3MT during the 120-3MT$_{CHO}$ (8.4 ± 0.5 mM) compared to the 120-3MT$_{PLA}$ visit (7.2 ± 0.5
308 mM; P<0.05).

309 The power output profiles during the C-3MT, 120-3MT$_{CHO}$ and 120-3MT$_{PLA}$ are shown in
310 Fig. 4. There were no differences in $\dot{V}O_{2peak}$ between C-3MT, 120-3MT$_{CHO}$, 120-3MT$_{PLA}$ and
311 the ramp incremental test (P>0.05). 120$_{PLA}$-EP was 9% lower (236 ± 47 W) than C-EP (260 ±
312 37 W; P<0.05) and 7% lower than 120$_{CHO}$-EP (254 ± 49 W; P<0.05; Fig. 5A). 120$_{CHO}$-EP
313 was not different from C-EP (Fig. 5A). C-WEP was 22% higher (17.9 ± 3.3 kJ) than 120$_{PLA}$-
314 WEP (13.8 ± 2.7 kJ; P<0.001) and 24% higher than 120$_{CHO}$-WEP (13.5 ± 3.4 kJ; P<0.001;
315 Fig. 5B). 120$_{CHO}$-WEP was not different from 120$_{PLA}$-WEP (Fig. 5B). Results for peak power
316 output and TWD are shown in Fig. 5C and Fig. 5D, respectively.

There was insufficient muscle tissue in one biopsy sample for completion of muscle glycogen analyses, and therefore the muscle glycogen data are for n=14. Muscle [glycogen] decreased over the 2 h heavy-intensity exercise bouts in both the 120-3MT$_{CHO}$ and 120-3MT$_{PLA}$ trial (Fig. 6). Muscle [glycogen] was lower following the 2 h bout in the 120-3MT$_{PLA}$ trial compared to 120-3MT$_{CHO}$ trial; however, the change in muscle [glycogen] was not different between trials (Fig. 6). There was no correlation between the change in muscle [glycogen] over the 2 h exercise bout and the changes in EP or WEP in either the 120-3MT$_{PLA}$ or the 120-3MT$_{CHO}$ bout.

To increase sample size and therefore increase confidence in our analysis, we combined the data from the present study with those from Clark et al. (13) to create a data set containing 28 participants who underwent muscle biopsies and completed a 3MT both at rest and following 2 h of heavy-intensity exercise. The pooled data show that muscle [glycogen] at rest was correlated with control EP (Fig. 7A) and that muscle [glycogen] following 2 h of heavy-intensity exercise was correlated with both fatigued EP (Fig. 7B) and fatigued WEP (r = 0.43; P<0.05). The change in EP following 2 h of heavy-intensity exercise was not correlated with the change in muscle [glycogen] (Fig. 8C, 8D). However, the percentage change in muscle [glycogen] following 2 h of heavy-intensity exercise was correlated with both the absolute change in WEP (Fig. 7E) and the percentage change in WEP (Fig. 7F).

Discussion

This is the first study to investigate the influence of different durations of prolonged endurance exercise, and CHO ingestion during exercise, on the parameters of the power-duration relationship. We used the 3MT as a practical and expeditious method to appraise changes in the power-duration relationship because it has been shown that the EP and WEP

provide valid estimates of CP and W′, respectively, both in the absence of prior exercise (7, 48) and following 2 h of heavy-intensity cycle exercise (13). The principal novel findings were that: (1) EP was not altered by 40 min or 80 min but was significantly reduced after 2 h of heavy-intensity exercise; (2) WEP was not altered by 40 min but was significantly reduced after 80 min and 2 h of heavy-intensity exercise; (3) the reduction in EP following 2 h of heavy-intensity exercise was negated when CHO was consumed; and (4) the reduction in WEP following 2 h of heavy-intensity exercise was not rescued by CHO ingestion. These results reveal disparate time courses for the changes in EP and WEP during heavy-intensity exercise: WEP appears to fall in an approximately linear fashion and becomes significantly reduced when exercise duration ≥80 min; while EP is preserved for at least 80 min and is only significantly reduced when exercise duration approaches 2 h. Importantly, our results show that EP (but not WEP) is preserved following 2 h of heavy-intensity exercise when 60 g/h CHO is consumed, suggesting that CP (but not W′) is influenced, at least in part, by CHO feeding. These findings may have significant implications for performance prediction and the formulation of optimal race (pacing and nutritional) strategies.

I: Dynamic changes in the parameters of the power-duration relationship following 40 min, 80 min and 2 h of heavy-intensity exercise

Consistent with our previous research, we found that 2 h of heavy-intensity exercise led to a decline of both EP and WEP (12, 13). Specifically, there was a ~9% fall in EP and a ~22% fall in WEP compared to C-3MT, results which are in close agreement with our previous findings of an 8-11% reduction in EP and a 20-22% reduction in WEP following 2 h of heavy-intensity exercise (12, 13). The key novel findings of the present study were that there were no significant changes in either EP or WEP compared to the control condition following 40 min of heavy-intensity exercise whereas, following 80 min of exercise, EP was unchanged but WEP was significantly reduced (by 17%). Our study therefore reveals differences in the

time course of changes in EP and WEP during endurance exercise, with WEP deteriorating more rapidly than EP. These results may provide insight into the physiological mechanisms underpinning the power-duration relationship and their relationship with the fatigue process during endurance exercise.

Several factors likely contribute to the changes observed in the power-duration parameters following 80 min and/or 2 h of heavy-intensity exercise. An increase in the O_2 cost of sustaining the constant power output was observed during the 2 h (from ~64% to ~68% of initial $\dot{V}O_{2peak}$) and 80 min (from ~63% to ~65% of initial $\dot{V}O_{2peak}$) but not the 40 min exercise bout (stable at ~63% of initial $\dot{V}O_{2peak}$) and occurred concomitantly with a decrease in RER in the 2 h (from ~0.91 to ~0.84) and 80-min (from ~0.93 to ~0.91) exercise bouts but not in the 40 min bout (from ~0.93 to ~0.92). The progressive loss of efficiency is therefore related in part to a shift in substrate utilisation from CHO towards fat oxidation during the 80 min and 2 h exercise bouts. Given that EP reflects a critical oxidative metabolic rate (1), a loss of efficiency (i.e., a higher $\dot{V}O_2$ per watt of power output) would necessarily result in a reduced EP.

A substantially increased core temperature and consequent redistribution of blood flow away from skeletal muscle to facilitate heat exchange might also increase the overall O_2 cost of exercise (36). Moreover, significant dehydration, due to the sweat rate exceeding the rate of fluid replacement, could compromise cardiac output and muscle O_2 delivery (42). Given that EP is an index of oxidative metabolic function (29, 47), these changes could reduce efficiency and/or exacerbate muscle metabolic perturbation and fatigue development. In our study, however, participants exercised in an air-conditioned lab (20^{o} C) and consumed 380 ml of fluid per hour, *pro rata*, such that body mass losses were minimal (≤1.0 kg; ≤1.2%). Such small changes in body mass suggest that issues related to thermoregulation probably did not

391 contribute appreciably to the decline in 3MT performance we observed and certainly indicate
392 that, if hyperthermia did occur, it likely affected subjects similarly.

393 It appears that CP represents an important boundary for neuromuscular fatigue development
394 (9, 40) such that central fatigue, which may be determined by a reduction in voluntary
395 activation measured by motor nerve stimulation, makes a greater contribution to fatigue
396 development in the heavy-intensity domain compared to the severe-intensity domain (4, 9,
397 46). It is possible, therefore, that the development of central fatigue during the 80 min and 2 h
398 heavy-intensity exercise bouts in the present study impaired performance in the subsequent
399 3MT, reducing EP and/or WEP. Consistent with this interpretation, it has been reported that
400 acetaminophen ingestion, which would be expected to blunt central fatigue development by
401 attenuating the sensation of pain and preventing any reduction in central motor drive, led to
402 greater muscle activation over the last 30 s of a 3MT and a higher EP compared to the placebo
403 condition (31).

404 The reduction in WEP after just 80 min as well as after 2 h of heavy-intensity exercise is
405 intriguing. Significant changes in muscle [ATP] and/or muscle metabolic status (i.e.
406 substantial depletion of phosphocreatine or accumulation of hydrogen ions) would not be
407 anticipated during heavy-intensity exercise (4) such that the development of peripheral fatigue
408 through these mechanisms is unlikely to explain the reduced WEP. The lack of effect on peak
409 power output during the 3MT following 80 min and 2 h of heavy-intensity exercise might
410 suggest that the reduced WEP at these time points is related not to high-energy phosphate
411 depletion but rather to impaired ATP production via anaerobic glycolysis which may, in turn,
412 be related to muscle glycogen depletion. Consistent with this, a reduction in WEP has been
413 reported when glycogen depletion has been invoked by dietary restriction (30) or the
414 performance of 2 h of heavy-intensity exercise (13). We note here that the evaluation of WEP
415 is less reliable than EP (12) such that possible error in the estimates of WEP is a possible

limitation of the present study; however, the reduction in WEP at 80 min and 2 h of exercise is substantial relative to the likely error.

When we combined the data from the present study with those from Clark et al. (13), we found a significant correlation between the change in WEP, but not EP, and the change in muscle [glycogen] following 2 h of heavy-intensity exercise. The progressive decline in muscle [glycogen] that would be expected during prolonged heavy-intensity exercise (18, 19) could therefore contribute to the reduction in WEP during the 80 min and 2 h exercise bouts. It is interesting to note, however, that WEP was significantly reduced despite muscle glycogen depletion being far from complete (~36% of the resting value was remaining following 2 h of exercise) at the start of the 3MT. An important consideration in this regard is that muscle glycogen depletion can be localised to subcellular compartments such that muscle performance might become impaired even when 'global' muscle [glycogen] appears sufficient (34). The present data suggest that absolute muscle [glycogen] may influence the rate of energy generation through anaerobic glycolysis, and/or influence contractile function via effects on sarcoplasmic reticulum calcium release (37), thereby reducing WEP.

II: Influence of CHO ingestion on changes in the power-duration relationship following 2 h of heavy-intensity exercise

We tested the hypothesis that changes in the power-duration relationship following 2 h of heavy-intensity exercise would be mitigated when CHO, compared to placebo, was consumed during exercise. A striking observation was that the reduction in EP following 2 h of heavy-intensity exercise was eliminated by 60 g/h of CHO ingestion. Specifically, in the placebo condition, EP was reduced by 9% compared to C-EP, consistent with previous findings (12, 13); however, when CHO was ingested during exercise, there was no significant reduction in EP compared to C-EP. In contrast, WEP was significantly reduced in both the placebo and

CHO conditions (22% and 24% reductions, respectively) compared to C-WEP, with there being no difference between the two conditions. An important novel finding of the present study is therefore that EP, but not WEP, may be preserved by CHO ingestion during endurance exercise of up to 2 h duration.

Significant muscle glycogen depletion was evident following 2 h of heavy-intensity exercise both when subjects ingested CHO (53% reduction in [glycogen]) and when they ingested a placebo beverage (64% reduction in [glycogen]). Muscle [glycogen] was higher following 2 h of heavy-intensity exercise with CHO compared to placebo ingestion but this may be explained by the tendency for muscle [glycogen] to be higher at baseline in the CHO condition. The reduction in muscle [glycogen] over the 2 h exercise bout was not different between conditions despite there being greater CHO availability and utilization (evidenced by higher RER, a greater rate of CHO oxidation and higher blood [glucose]) when CHO was ingested. Similarly, Smith et al. (45) reported that ingestion of 60 g/h of CHO during 2 h of exercise at 77% of $\dot{V}O_{2peak}$ did not spare muscle glycogen, compared to placebo, but did enhance subsequent 20-km time trial performance, an effect that the authors suggested was due to the increased rate of CHO oxidation. In light of the results of the present study, it is possible that the participants in Smith et al. (45) had a higher CP and could therefore sustain a higher power output during the 20-km time trial following CHO compared to placebo ingestion.

The increased rate of CHO oxidation observed in the present study, consequent to CHO ingestion, likely contributed to the preserved EP we observed. However, given the greater proportional development of central fatigue during heavy-intensity endurance exercise (4, 9, 46), it is possible that other factors also contributed. In the placebo condition, blood [glucose] was significantly lower than the baseline value at 100 and 120 min of exercise (~3.8 mM) whereas it remained stable throughout exercise in the CHO condition (~4.8 mM). It is

possible that this difference in blood [glucose] attenuated central fatigue development (14, 35) and enabled a higher muscle activation during the subsequent 3MT. Similarly, the detection of CHO by sensors in the mouth, even when the CHO is not swallowed, may attenuate decrements in exercise performance associated with fatigue (10, 16, 22). Specifically, it appears that CHO receptors in the oral cavity may signal an impending increase in CHO availability to higher brain regions (11, 16, 23). It is possible therefore that, in addition to direct effects on skeletal muscle metabolism, the ingestion of CHO during exercise reduced central fatigue and enabled enhanced motor output and contractile function (35) during the subsequent 3MT.

In the present study, no significant correlations were found between the changes in muscle [glycogen] and EP or WEP in either condition, possibly due to the relatively small sample size. However, when the data from the present study were pooled with those of Clark et al. (13), we found a positive relationship between EP and muscle [glycogen] both at rest and following 2 h of heavy-intensity exercise. These results suggest a link between aerobic fitness and muscle glycogen storage, which might be mediated by training status (15). Moreover, when the data from the two studies were combined, we found that, following 2 h of heavy-intensity exercise, muscle [glycogen] was correlated with WEP and also that the change in muscle [glycogen] over the 2 h exercise bout was correlated with the change in WEP.

The physiological mechanisms underpinning W′ have not been entirely resolved (5, 33, 43). However, because CP is closely related to the proportion of type I muscle fibers (29, 47), it has been suggested that W′ may reflect the metabolic, contractile and/or fatigue-related characteristics of type II muscle fibers (47, 51). The fall in WEP we observed might therefore reflect the specific effects of 2 h of heavy-intensity exercise on the type II fiber population, including glycogen depletion (52). There is evidence to suggest that W′ is related to the capacity for substrate-level phosphorylation, with PCr availability being important for the

achievement of peak power output in the 3MT (39, 47, 49). It has been reported that W′ is reduced following glycogen depletion induced by dietary CHO restriction, with there being no effect on CP (30). The results of the current investigation are consistent with these findings in showing that WEP is reduced when glycogen depletion is evident following 2 h of heavy-intensity cycling, irrespective of effects on EP.

The different effect of CHO feeding during endurance exercise on EP and WEP provide novel insight into the mechanisms underpinning these parameters (and, by extension, CP and W′). While muscle glycogen depletion was not altered compared to the placebo condition, the maintenance of euglycaemia by CHO ingestion during prolonged exercise resulted in a preservation of EP in the face of a decline in WEP. This suggests that, following 2 h of heavy-intensity exercise, EP may be modulated by central fatigue whereas WEP may be more directly related to muscle glycogen availability. Further studies are required, with more direct measurements of central and peripheral fatigue, to explore this possibility.

Implications for Performance

The results of the present study have several potentially important implications for athlete performance diagnostics and race practice. The disparate time courses for the degradation of EP and WEP we observed during prolonged endurance exercise is of particular interest. The maintenance of EP for ~80 min of such exercise indicates that performance for events which are sustained below EP can be faithfully predicted from measurements made in the rested state whereas, for longer events, the fall in EP render such predictions more complicated. Indeed, inter-individual variation in the reductions in EP and WEP *during* endurance exercise, reflecting fatigue resistance, is likely an important but previously overlooked component of success in endurance sports (12). The fall in WEP during heavy-intensity exercise occurred much earlier than the fall in EP, with the group mean reduction being 10% after 40 min (not

514 significant), 18% after 80 min and 23% after 2 h. This relatively early reduction in WEP may
515 impact on an athlete's ability to draw upon W′, limiting their ability to respond to, or initiate,
516 surges in pace above CP, a factor that should be carefully considered when developing race
517 tactics.

518 Our findings also emphasise the importance of CHO consumption during prolonged
519 endurance exercise by showing that ingestion of 60 g/h CHO enabled EP to be maintained
520 compared to a 9% reduction in EP when placebo was consumed. The performance
521 implications of this difference, whether measured in terms of the mean speed that may be
522 sustained throughout a race or in the ability to finish strongly, are likely to be profound.
523 Depending on the power being sustained and the extent of the fall in CP during prolonged
524 endurance exercise, it is possible that an athlete may eventually transition from the heavy- to
525 the severe-intensity exercise domain, with the inevitable outcome that power must fall and/or
526 exercise will become intolerable. This highlights the important interaction between
527 physiology and nutrition during endurance exercise. Maintaining a high rate of CHO ingestion
528 while exercising at a high intensity is an important consideration, and a potential limiting
529 factor, for extreme human endurance challenges such as the attempt to run a <2 h marathon
530 (6, 27).

531 It is important to acknowledge that our results are specific to the conditions of our study.
532 Naturally, dynamic changes in EP and WEP would likely be different if other combinations of
533 exercise intensity and duration were assessed. Moreover, the effect of CHO ingestion on the
534 change in EP is also likely influenced by exercise intensity, exercise duration and the rate
535 (and type) of CHO consumption (22). Finally, the extent to which our results in cycling may
536 be extrapolated to other exercise modalities is presently unclear. These questions and the
537 nature of the interaction between these key variables might be the subject of future
538 investigations.

539 *Conclusion*

540 In conclusion, the EP and WEP, surrogates of the parameters of the power-duration
541 relationship, CP and W′, respectively, were reduced following 2 h of heavy-intensity cycling.
542 WEP, but not EP, was also reduced following 80 min of heavy-intensity cycling. CHO
543 ingestion during 2 h of heavy-intensity cycling abolished the reduction in EP, but not WEP.
544 These results suggest that, following prolonged endurance exercise, W′ may be sensitive to
545 local muscle glycogen availability, presumably via a limitation to energy production by
546 anaerobic glycolysis, whereas CP may be sensitive to global CHO availability, perhaps via its
547 relationship with central fatigue development. Practitioners should be aware that dynamic
548 changes in the parameters of the power-duration relationship *during* heavy-intensity exercise
549 present a challenge to the use of these parameters to predict performance during endurance
550 sports events. It is clear, however, that CHO supplementation represents a practical and
551 effective intervention to constrain the deterioration of CP during endurance exercise.

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739

740 **Figure Legends**

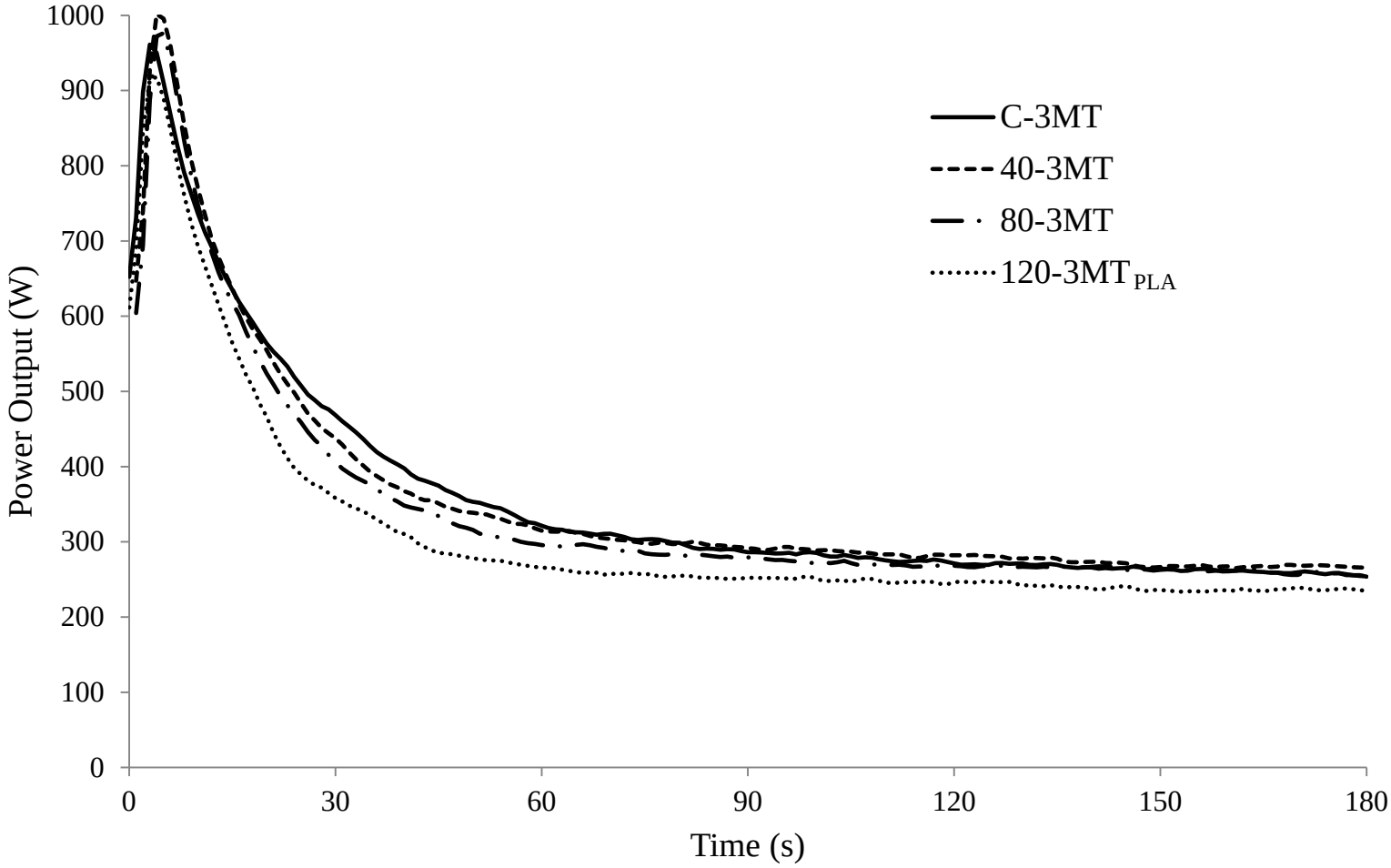
741 **Figure 1.** The group mean power profiles during the 3-min all-out test (3MT) measured with
742 no prior exercise (C-3MT), and when preceded by 40 min (40-3MT), 80 min (80-3MT) and 2
743 h (120-$3MT_{PLA}$) of heavy-intensity exercise. Error bars are not shown for clarity. The end test
744 power was significantly reduced at 2 h compared to control and the work done above the end
745 test power was significantly reduced at 80 min and at 2 h. See text for further details.
746
747 **Figure 2.** Group mean end test power (A), work done above end test power (B), peak power
748 outputs (C) and total work done (D) during the 3-min all-out test (3MT) measured with no
749 prior exercise (C-3MT), and following 40 min (40-3MT), 80 min (80-3MT) and 2 h (120-
750 $3MT_{PLA}$) of heavy-intensity exercise. a = different from C-3MT (P<0.05), b = different from
751 40-3MT (P<0.005), c = different from 80-3MT (P<0.05).
752
753 **Figure 3.** Group mean relative intensity (%$\dot{V}O_{2peak}$) (panel A), respiratory exchange ratio
754 (panel B), carbohydrate oxidation (panel C) and blood [glucose] and [lactate] (panel D)
755 during 2 h of heavy-intensity cycling while ingesting carbohydrate (white symbols) or
756 placebo (black symbols). * = different from placebo (P<0.05), a = different from 10-15 min
757 (P<0.05), b = different from 25-30 min (P<0.05), c = different from 55-60 min (P<0.05), d =
758 different from 85-90 min (P<0.05), $= different from baseline (P<0.05), £ = different from 20
759 min (P<0.05).
760
761 **Figure 4.** The group mean power profiles during the 3-min all-out test (3MT) measured with
762 no prior exercise (C-3MT) and when preceded by a 2 h-heavy intensity exercise bout with
763 ingestion of carbohydrate (120-$3MT_{CHO}$) or placebo (120-$3MT_{PLA}$). Error bars are not shown
764 for clarity. The end test power was significantly reduced at 2 h compared to control when

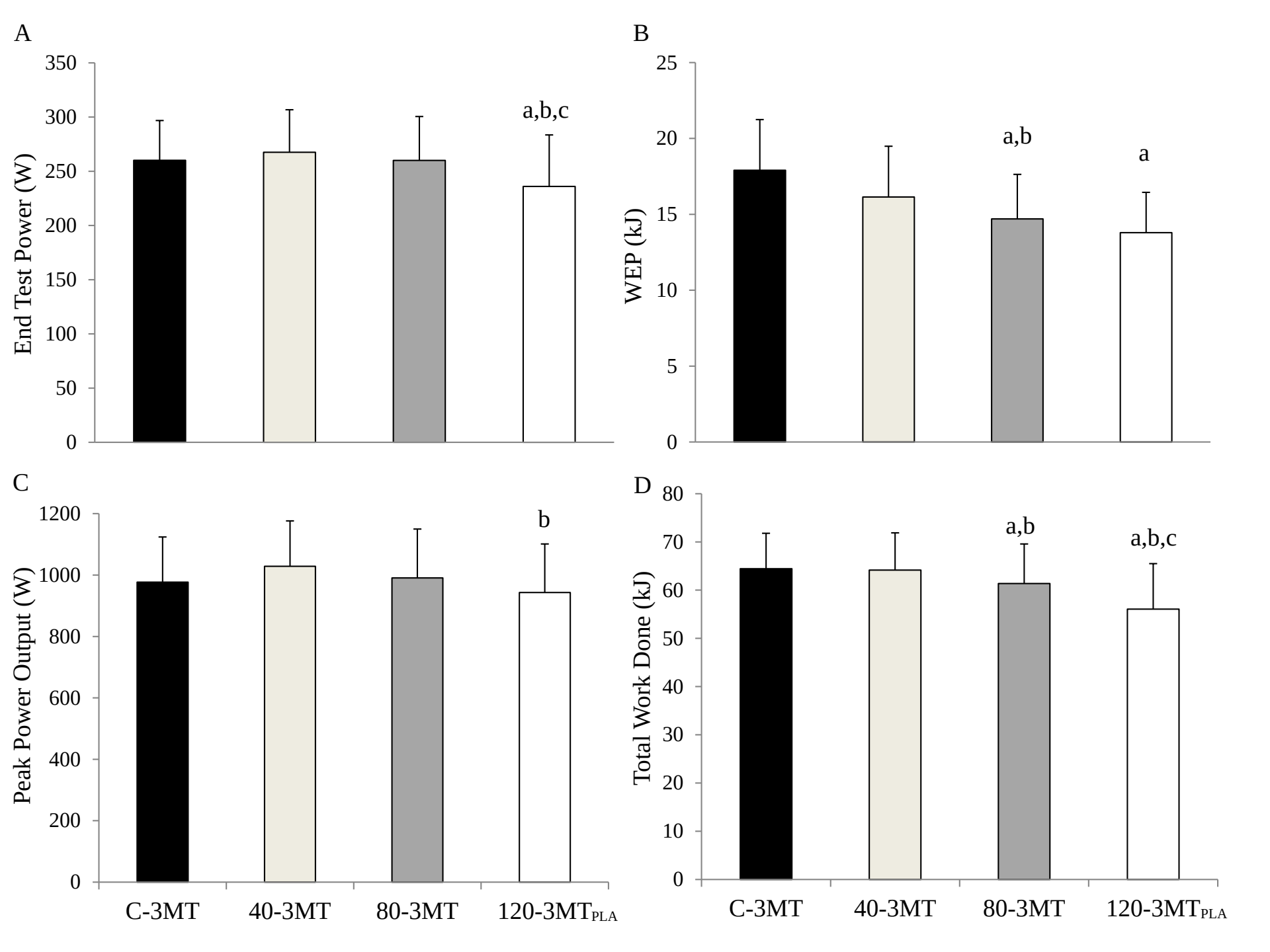
765 placebo was ingested but was not different to control when carbohydrate was ingested. The
766 work done above the end test power was significantly reduced compared to control
767 irrespective of placebo or carbohydrate ingestion. See text for further details.
768
769 **Figure 5.** Group mean ± SD end test power (A), work done above end test power (B), peak
770 power outputs (C), and total work done (D) during the 3-min all-out test measured with no
771 prior exercise (C-3MT) and after 2 h of exercise while ingesting carbohydrates (120-$3MT_{CHO}$)
772 or placebo (120-$3MT_{PLA}$). * = significant difference (P<0.05).
773
774 **Figure 6.** Group mean ± SD muscle [glycogen] before and after 2 h of heavy-intensity
775 exercise while consuming carbohydrate (CHO) or placebo (PLA). a = different from pre-
776 exercise values (P<0.001), b = different from CHO condition (P<0.05).
777
778 **Figure 7.** Combined results from present study (black circles) and Clark et al. (13) (white
779 circles) (n=28). Correlation between muscle [glycogen] at rest and end test power (EP)
780 without preceding exercise (panel A) and correlation between muscle [glycogen] and EP after
781 2 h of heavy-intensity exercise (B). Correlations between the percentage change in muscle
782 [glycogen] and the absolute (panel C) and percentage (panel D) change in EP over 2 h of
783 heavy-intensity exercise. Correlations between the percentage change in muscle [glycogen]
784 and the absolute (panel E) and percentage (panel F) change in WEP over 2 h of heavy-
785 intensity exercise. ** = P<0.005; * = P<0.05.

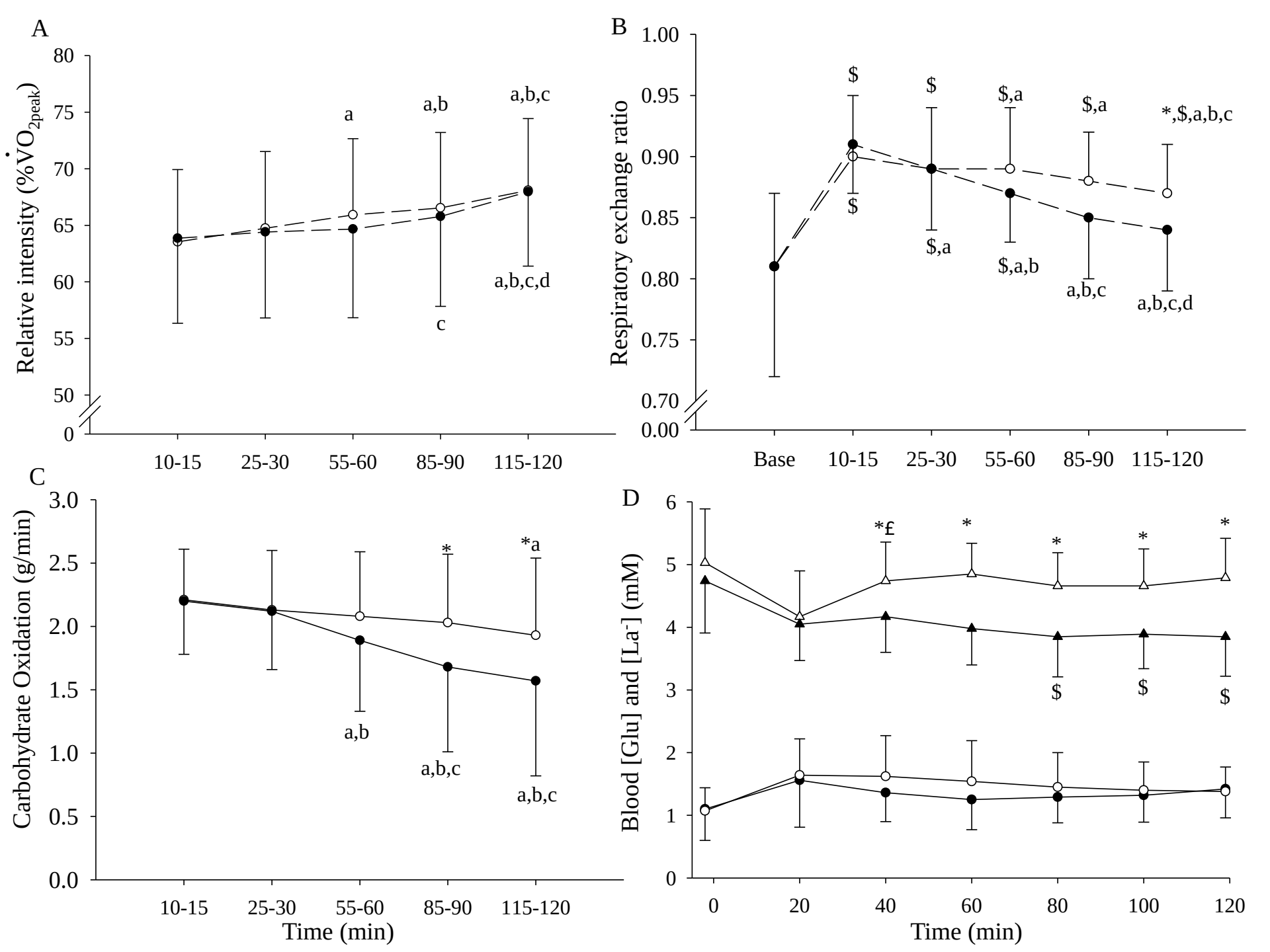
Table 1. Mean ± S.D. body mass, heart rate and RER during heavy-intensity exercise performed for various durations of heavy-intensity exercise

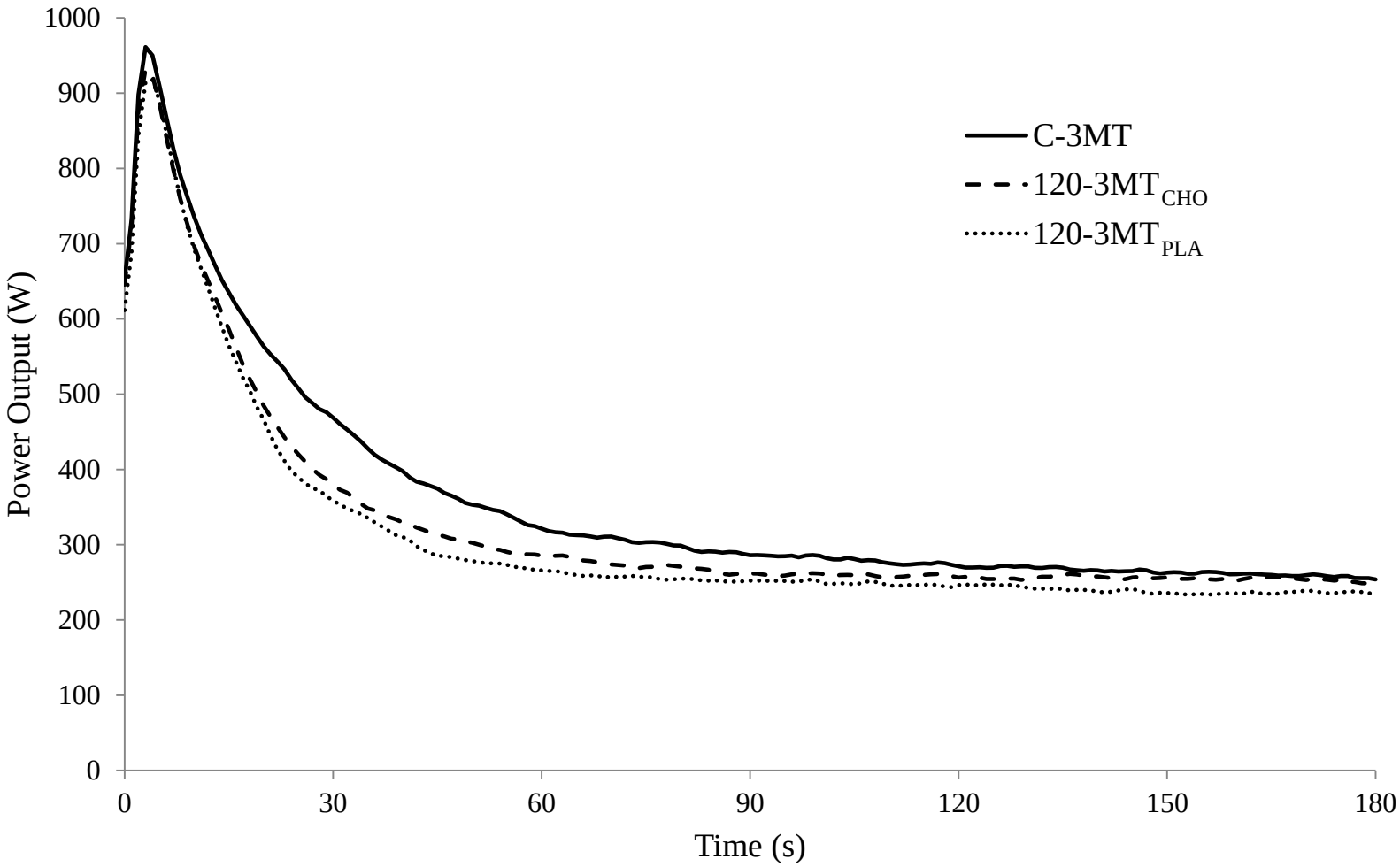
	Body mass (kg)		Heart Rate ($b \cdot min^{-1}$)		RER	
	Pre exercise	Post exercise	10 - 15 min	Last 5 min of exercise	10 - 15min	Last 5 min of exercise
40-3MT	79.2 ± 7.6	78.9 ± 7.6[ab]	134 ± 16	138 ± 16[ab]	0.93 ± 0.03	0.92 ± 0.04[b]
80-3MT	79.3 ± 7.6	78.7 ± 7.5[ab]	133 ± 15	141 ± 17[ab]	0.93 ± 0.04	0.91 ± 0.05[ab]
120-3MT$_{PLA}$	79.2 ± 7.5	78.2 ± 7.3[a]	134 ± 18	150 ± 16[a]	0.91 ± 0.04	0.84 ± 0.05[a]
120-3MT$_{CHO}$	79.4 ± 7.6	78.5 ± 7.4[a]	130 ± 16	149 ± 17[a]	0.90 ± 0.05	0.87 ± 0.04[ab]

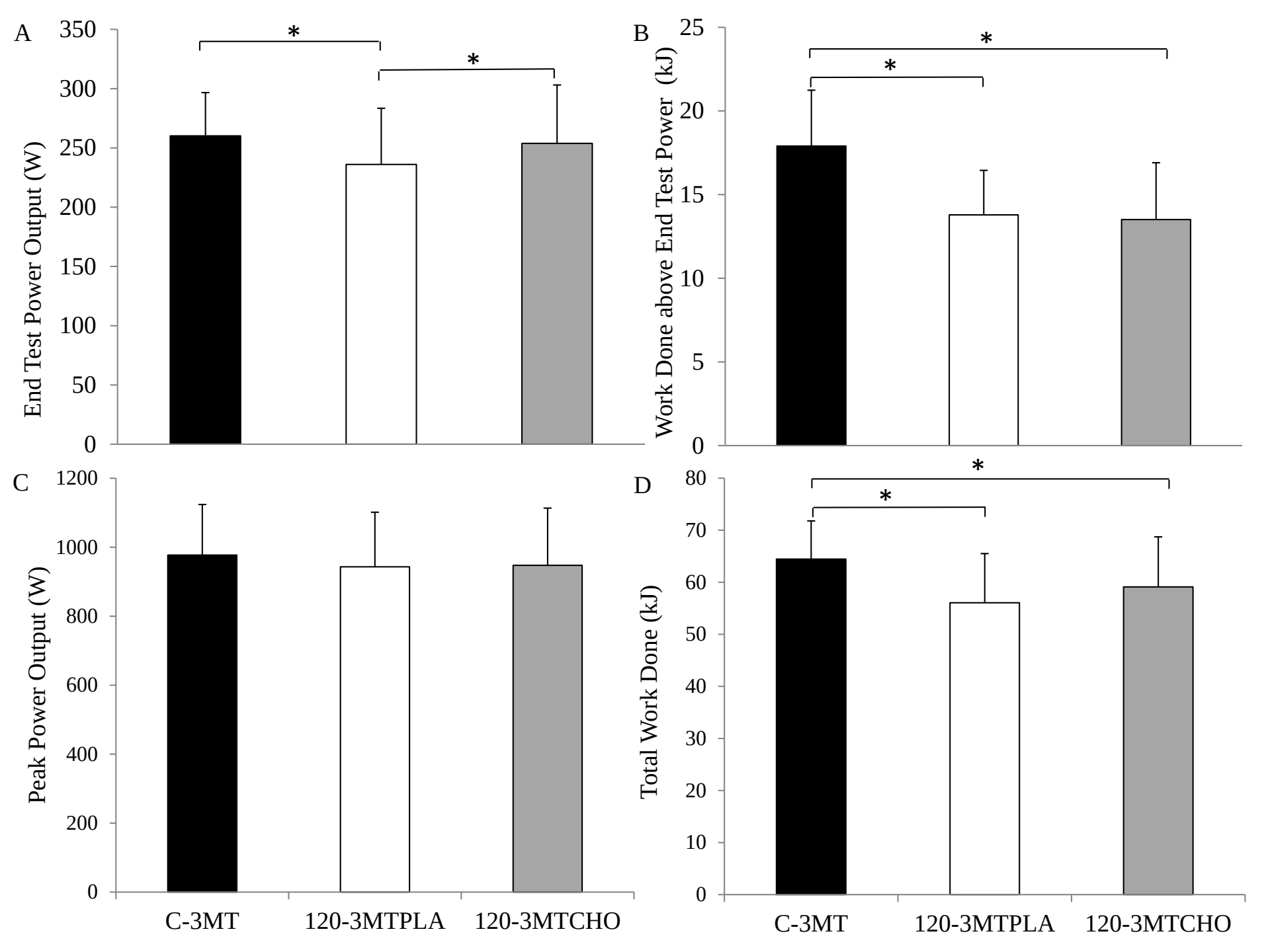
40-3MT, 40 min of heavy-intensity exercise; 80-3MT, 80 min of heavy-intensity exercise; 120-3MT$_{PLA}$, 120 min of heavy-intensity exercise consuming water; 120-3MT$_{CHO}$, 120 min of heavy-intensity exercise consuming carbohydrates. [a] = different from start of exercise measurements, $P<0.05$; [b] = different from 120-3MT$_{PLA.}$, $P<0.05$.

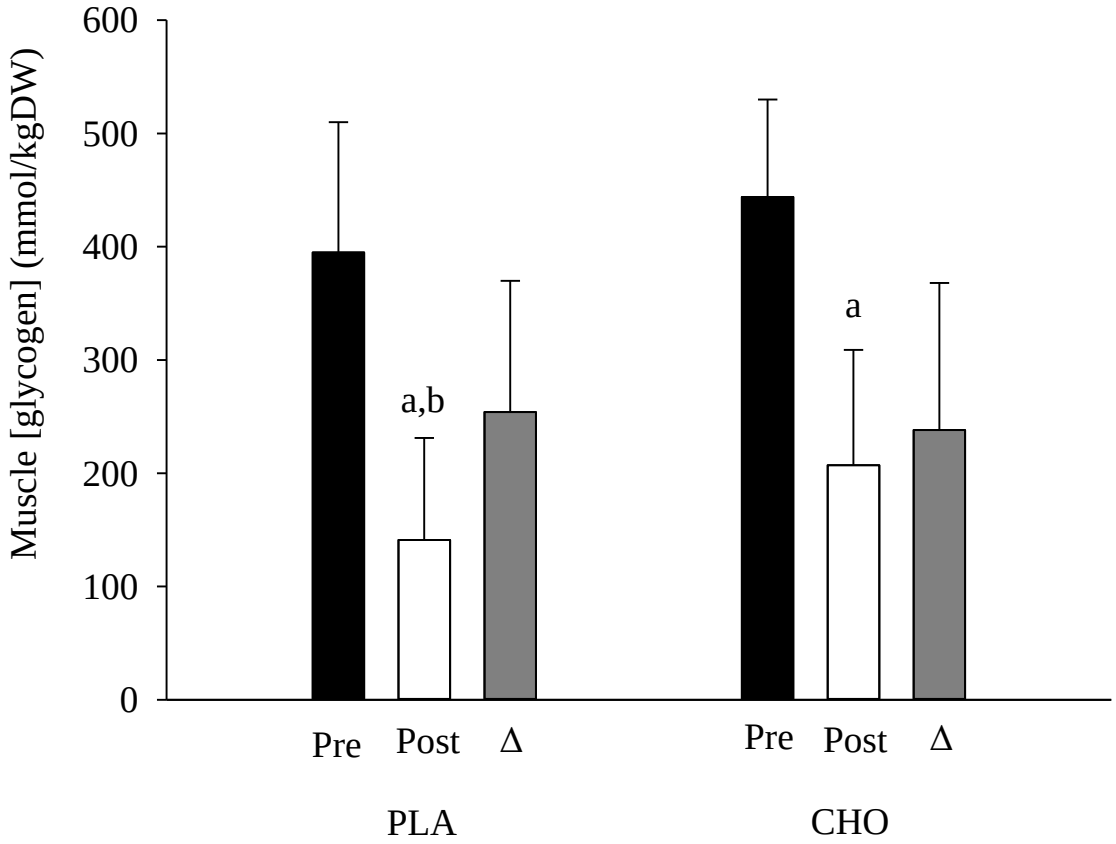

1000
900
800
700
600
500
400
300
200
100
0
Power Output (W)
0
30
60
90
120
150
180
Time (s)
C-3MT
40-3MT
80-3MT
120-3MT PLA

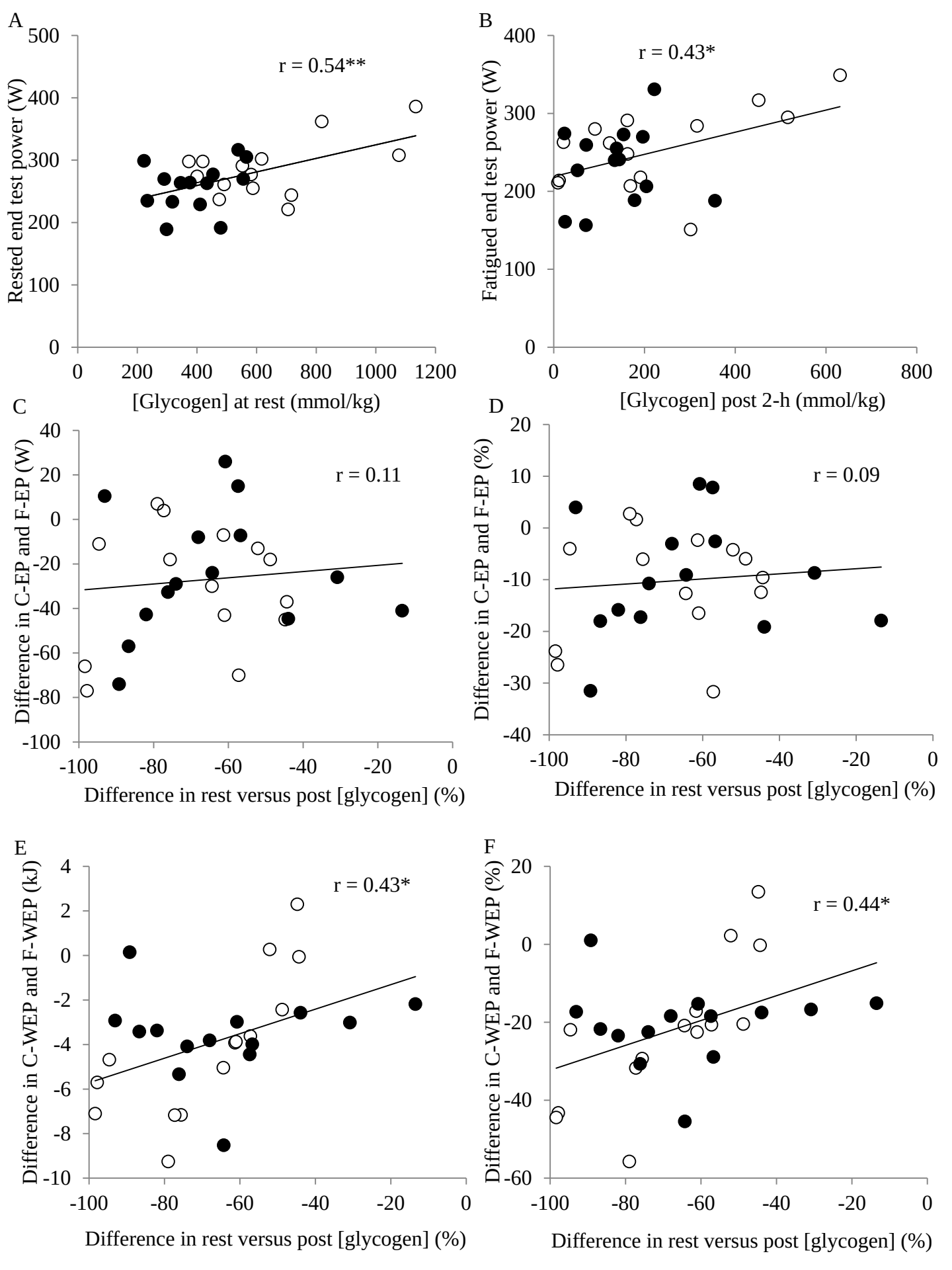

A
End Test Power (W)
350
300
250
200
150
100
50
0
a,b,c
B
WEP (kJ)
25
20
15
10
5
0
a,b
a
C
Peak Power Output (W)
1200
1000
800
600
400
200
0
b
D
Total Work Done (kJ)
80
70
60
50
40
30
20
10
0
a,b
a,b,c
C-3MT
40-3MT
80-3MT
120-3MTPLA
C-3MT
40-3MT
80-3MT
120-3MTPLA


A
Relative intensity (%$\dot{V}O_{2peak}$)
80
75
70
65
60
55
50
0
a
a,b
a,b,c
c
a,b,c,d
10-15
25-30
55-60
85-90
115-120
B
Respiratory exchange ratio
1.00
0.95
0.90
0.85
0.80
0.75
0.70
0.00
$
$
$
$,a
$,a
$,a
$,a,b
a,b,c
*,$,a,b,c
a,b,c,d
Base
10-15
25-30
55-60
85-90
115-120
C
Carbohydrate Oxidation (g/min)
3.0
2.5
2.0
1.5
1.0
0.5
0.0
*
*a
a,b
a,b,c
a,b,c
10-15
25-30
55-60
85-90
115-120
Time (min)
D
Blood [Glu] and [La-] (mM)
6
5
4
3
2
1
0
*£
*
*
*
*
$
$
$
0
20
40
60
80
100
120
Time (min)


1000
900
800
700
600
500
400
300
200
100
0
Power Output (W)
0
30
60
90
120
150
180
Time (s)
C-3MT
120-3MT CHO
120-3MT PLA


A
End Test Power Output (W)
350
300
250
200
150
100
50
0
*
*
B
Work Done above End Test Power (kJ)
25
20
15
10
5
0
*
*
C
Peak Power Output (W)
1200
1000
800
600
400
200
0
D
Total Work Done (kJ)
80
70
60
50
40
30
20
10
0
*
*
C-3MT
120-3MTPLA
120-3MTCHO
C-3MT
120-3MTPLA
120-3MTCHO


Muscle [glycogen] (mmol/kgDW)
600
500
400
300
200
100
0
a,b
a
Pre
Post
Δ
Pre
Post
Δ
PLA
CHO


A
r = 0.54**
Rested end test power (W)
[Glycogen] at rest (mmol/kg)
B
r = 0.43*
Fatigued end test power (W)
[Glycogen] post 2-h (mmol/kg)
C
r = 0.11
Difference in C-EP and F-EP (W)
Difference in rest versus post [glycogen] (%)
D
r = 0.09
Difference in C-EP and F-EP (%)
Difference in rest versus post [glycogen] (%)
E
r = 0.43*
Difference in C-WEP and F-WEP (kJ)
Difference in rest versus post [glycogen] (%)
F
r = 0.44*
Difference in C-WEP and F-WEP (%)
Difference in rest versus post [glycogen] (%)