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ORIGINAL ARTICLE

Performance and recovery: effects of caffeine on a 2000-m rowing ergometer

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Abstract

Introduction This study investigated the effects of caffeine on subsequent performance and recovery from a 2000-m rowing ergometer.

Materials and methods Nine trained male rowers (18.3 ± 1.1 years) consumed 3 or 6 mg kg^{-1} of caffeine or placebo. In three sequential time trials (TT), 30-min after (Trial1) and 6.5 h after (Trial2) caffeine intervention, participants performed a 2000-m ergometer effort. Power output (PO), stroke rate (SR), and time were monitored during the TTs. Ventilatory variables, serum creatine kinase (CK), blood lactate (BLa), and heart rate (HR) were recorded in Trial1 and Trial2, 30 min after Trial1 (1st recovery) and 30-min before Trial2 (2nd recovery).

Results After ingesting 6 mg kg^{-1} of caffeine, PO was significantly greater in Trial1 (8.2%) compared to the placebo. Also significant increases in $\dot{V}O_{2peak}$ during Trial1 (10.1%) and 2 (13.9%), HR at $\dot{V}O_{2peak}$ (5.5%), $HR_{VO_{2peak}}$ during Trial1 (10.0%) and 2 (11.3%), $\dot{V}O_2/HR_{peak}$ in Trial1 (19.6%) and 2 (17.3%), $\dot{V}O_2$ at lactate threshold (LT) in Trial1 (24.6%), and significant decrease in ventilatory evaluation ($\dot{V}_E$) (9.0%) were shown in 6 mg kg^{-1} compared to placebo. In recovery phase, minute ventilation (MV) was significantly higher in 1st (17.1%) and 2nd recovery (28.7%) and RER increased in 1st recovery (9.9%, $P = 0.04$) in 6 mg kg^{-1} compared to placebo. CK increased significantly in 2nd recovery (14.1%). BLa in 6 mg kg^{-1} was higher than placebo in Trial1 (18.6%) and Trial2 (24.4%).

Conclusion Based on improvements in ventilatory variables, and decreased CK and BLa levels after 6 mg kg^{-1} caffeine ingestion in performance and recovery, this study provides novel data by demonstrating that pre-exercise caffeine ingestion improves performance and subsequent recovery from rowing effort.

Keywords Ergogenic effects · Post-exercise recovery · Ventilatory evaluation · Performance · Anaerobic threshold · Trained rowers

Abbreviations

TT	Time trials
PO	Power output
SR	Stroke rate
CK	Creatine kinase
BLa	Blood lactate
HR	Heart rate
$\dot{V}O_{2peak}$	Peak oxygen uptake
$HR_{VO_{2peak}}$	Heart rate at VO_{2peak}
$HR@\dot{V}O_{2peak}$	Heart rate at $\dot{V}O_{2peak}$
$\dot{V}_E$	Ventilatory evaluation
MV	Minute ventilation
RER	Respiratory exchange ratio
LT	Lactate threshold
$\dot{V}O_2/HR$	Oxygen pulse (O_2 pulse)

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Introduction

Evidences about caffeine ingestion widely declare significant improvement in endurance performances [1–3]. Also there are some, but not too many reports revealing that power-endurance high-intensity performances such as 2000-m rowing time-trial (TT) have been improved after caffeine ingestion [4–7]. On

the other hand, recovery is one of the important aspects of athletic training which is considered for defining the exercise-adaptation cycle in different activities for trained athletes. It is clear that even top-level athletes spend much more time in recovery, rest, and sleep than that of physical training [8]. Recovery process is biphasic, with an initial rapid phase lasting 10 s to a few minutes followed by a slower second recovery phase lasting anywhere from a few minutes to a number of hours or days [9]. Optimal recovery period between training sessions can result in more intensive training at the next session than the latter while reducing the likelihood of overtraining syndrome [10, 11]. In endurance sports, coaches attempt to use a series of high-volume training sessions thereafter by necessary load progression to optimize athletic performance. In some cases, with insufficient recovery periods, excessive overloads in long-term endurance sports cause chronic injuries or overtraining [8]. Rowing is a strength-endurance type sport requiring high-volume training to get higher performance. A 2000-m rowing race including ~ 220 to 260 strokes, equivalent to ~ 5.5 to 7.5 min depending on boat type and weather condition, requires maximal capacity of active muscles and can be classified as a short-term high-intensity endurance performance. The average power of a rowing race in elite rowers is 450–550 W [12]. For a 2000-m race, both aerobic and anaerobic capacities are needed, while power endurance is predominant which presumes to deplete the energy sources of the active muscles [12, 13]. Generally, the more that exercise disrupts homeostasis, the greater the effect on recovery metabolism. The more complete these restorative processes, the greater the ability to generate force or maintain power on subsequent efforts [9]. Hence, it is of practical interest for coaches to hasten recovery of their athletes without increasing the risk of injury. In this basis, several studies have examined the effects of different Trials before, simultaneously or after endurance performances [14] and recovery from that performance [15]. Among Trials, caffeine is one of the mostly used supplements in athletes from wide range of sports [3]. Caffeine has a half-life of 4–6 h which is generally recognized as safe in doses lower than 10 mg kg^{-1} body weight [16]. Also, typical caffeine doses of 3–6 mg kg^{-1} bodyweight ingested prior to exercise reported to make positive effects such as increased work output and time to exhaustion in rowing and running TT lasting 5–60 min [2, 4, 17]. Other studies demonstrated that caffeine improves performance through decreased perceived effort during exercise, positive effects on cognitive parameters such as reaction time and attention, and an increase in Ca^{2+} permeability, cellular potassium uptake, and maintenance of the resting membrane potential of muscle cells [18]. Also caffeine elevated peak (PPO) and mean power output (MPO) in glycogen-depleted cyclists [19]. A study stated that elevation in catecholamine during intensive performance enhances muscle glycogenolysis leading to higher lactate production and power output [20].

In this line, most studies have attempted to explain the effects of caffeine ingestion before sustained high-intensity endurance exercise [4–6]. Although early studies supported a glycogen-sparing mechanism of action [2], the absence of a corroborative change in respiratory exchange ratio (RER), combined with evidence of significant effects in relatively short events (< 60 min) where glycogen availability would not be a limiting factor [1], has led researchers to consider alternative explanations for that.

However, less is known concerning the effects of caffeine ingestion after this type of performance. Several studies have shown positive effects of adding different doses of caffeine to post-exercise carbohydrate feeding on recovery-related variables after strenuous resistance training [21], high-intensity interval-running [22], and cycling time-trial [23]. Moreover, the positive benefits of caffeine on muscle soreness decline after a strength exercise session has been reported [24, 25]. But in reviewing the literature, no previous study has examined the effects of caffeine supplementation on recovery after high-intensity endurance performances. Indeed, the reports of Glaister et al. which found no change in Blood Lactate while RER, V_E, VCO_2, and Heart Rate increased in 30 min recovery after ingesting 5 mg kg^{-1} bodyweight caffeine in incremental strength-endurance cycle ergometer performance is the only source which we found in this regard [15]. Although some findings stated the benefits of caffeine on performance and recovery of aerobic-muscle endurance sports (Running, Cycling, Kayaking, etc.), or the benefits of caffeine on performance after sleep deprivation which destroys recovery process [26], due to the characteristics of rowing performance which highly depends on aerobic and high-intensity strength endurance, especially in leg muscles, it seems that the physiological mechanisms are different from those sports. Also, the different tempo of strokes (~ 34 strokes per min) in elite rowers make it different from higher strokes in kayaking (~ 100 rate per min), cycling (~ 100 rate per min) and running (~ 180 steps per min) which seem more dependent on aerobic capacity than Rowing. Given the importance of better performance and efficient recovery into account, the aim of the present study was to test the hypothesis that ingestion of caffeine in different doses promotes 2000-m ergo rowing TT performance and the next TT performed 6-h post-Trial following better recovery compared with placebo (PL).

Materials and methods

Experimental protocol and procedures

A double-blind, placebo-controlled, cross-over design was utilized in this investigation. Participants were asked to attend seven laboratory sessions. The first session was for

preliminary testing followed by three sequential time trials (TT) in a pre to post-Trial manner (any of the sequences included two times of a 2000-meter ergo rowing with 6-h interval). All results were blinded to the rowers. Each sequence was separated by 6 days to ensure washing out the caffeine ingested (Fig. 1) [6].

Participants

Nine junior elite male rowers who were selected to participate in World Junior Rowing Championships aged (± SD) (18.1 ± 1.1 years), body mass (77.9 ± 8.6 kg), stature (185.7 ± 7.1 cm), body fat (8.4 ± 1.5%), and $\dot{V}O_{2peak}$ (51.38 ± 3.93 ml kg^{-1} min^{-1}) volunteered to participate in the present study, whereas randomly assigned to different groups in a cross-over design. Subjects were non-habitual consumers of caffeine and following routine medical screening, they were questioned for possible reactions to caffeine before being included in the study. Participants were also informed of the purpose of this study and the possible risks involved, and all provided their written informed consent to participate. The study was approved by the ethical committee of Iran Canoe, Rowing and Sailing National Academy and conformed to the Declaration of Helsinki.

Procedures

Diet and exercise control

Participants were directed to continue the same general lifestyle patterns of exercise and nutrition intake during the experiment. They followed the same rowing training sessions including 12 km of sculling with moderate intensity (70–75% HR_{max}) four times a week and weight lifting training 2 session per week in 3 set/8 repetitions/75% × 1 repetition maximum (movements including bench press, military press, bench pull, seated row, biceps curl, and pulley push-downs). Also, nutritional intake was controlled before and 6 h after each experimental trial by providing subjects with a standard diet of 50 kcal kg^{-1} of body mass, composed of 63% carbohydrate (8 g kg^{-1}), 20% fat, and 17% protein. In addition, subjects were asked to refrain from participating in vigorous activity and to avoid the consumption of caffeinated food and beverages in the 6-h period prior to testing. All preliminary testing and experimental trials were performed under standard laboratory environmental conditions before the beginning of the general preparation phase of the athletes' yearly training program.

Supplementation protocol

The study supplementation consisted of 3 or 6 mg kg^{-1} of caffeine (Caffeine Anhydrous, USP; Meridian, Decatur, AL) or placebo (Cellulose). Considerably, caffeine has been included in the 2018 and 2019 WADA Monitoring Program. Therefore, although nowadays it is not considered as Prohibited Substance, it can be included in further editions.

The supplement assignments were blinded to both the participants and the study investigators. The order in which caffeine or placebo was received was counterbalanced and randomly assigned. Thirty minutes before 2000-m TT test, three of the participants ingested 3 mg kg^{-1} of caffeine, three of whom ingested 6 mg kg^{-1} of caffeine, and the other three participants received placebo. Caffeine and placebo were ingested in gelatin capsules with approximately 500 mL of water. In the next sequence, those who previously consumed 3 mg kg^{-1} caffeine received 6 mg kg^{-1} caffeine or placebo and those who had consumed placebo received 3 or 6 mg kg^{-1} caffeine.

Testing protocols

On the preliminary testing session, a progressive incremental exercise test was performed on a Concept IIc rowing ergometer (Concept2, USA) to determine peak oxygen uptake ($\dot{V}O_{2peak}$), HR peak, V_E, blood lactate, and RER. Utilizing Concept IIc rowing ergometer is believed to simulate the metabolic and biochemical demands of on-water rowing and can be used to assess rowing performance. Moreover, the seven-step incremental exercise test was

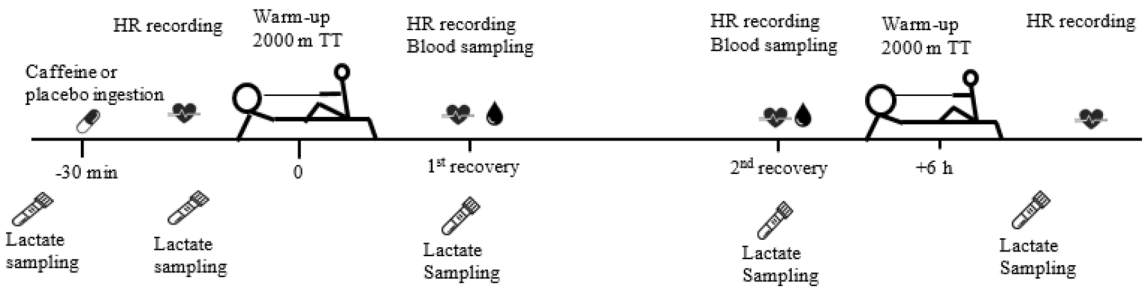

Fig. 1 Schematic of a performance testing sequence illustrating warm-up, 2000-m TT (Trial1 and Trial2), recovery periods (1st and 2nd recovery period), HR (heart rate) monitoring, blood sampling, capillary lactate sampling (immediately before and after), and capsule ingestion

performed as described before which the subjects were accustomed to [27]. Cardiorespiratory-metabolic variables were measured throughout the progressive exercise test, 1st recovery time (30 min after Trial1 for 1 min), and 2nd recovery time (30 min before Trial2) using a gas analyzer (Cosmed K4B2, Italy). Before each test, the gas analyzer was calibrated according to the manufacturer's instructions. Ventilatory threshold (VT), minute ventilation at rest (MV), VO_2 maximal ventilation (V_E), breathing frequency (BF) at rest, maximal HR, and resting RER were recorded. We calculated oxygen pulse (VO_2/HR), VO_{2peak}/HR and VO_{2peak}/LT from the data. In addition, the Lactate threshold (LT) was estimated using the modified V-Slope method as previously was addressed by our laboratory team [28].

For the 2000-m TTs, all subjects performed a 10-min warm-up including 5-min ergo rowing (2 min at 100 W, 2 min at 150 W and 1 min at 175 W) and 5-min stretching before the test, which was replicated before each TT. Power output and stroke rate were updated continuously on the computer display of the rowing ergometer during the TT, and average values were presented for each measure at the completion of the TT. Time to complete the TT and mean power output (MPO) were recorded as the criterion-dependent variable. Heart rate (HR) was recorded using a heart rate monitor (Polar s610i; Polar Electro Oy, Kempele, Finland) during rest, Trial1 (first performance) and Trial2 (second performance). Also it was counted at 1st recovery time (30 min after Trial1) and 2nd recovery time (30 min before Trial2) using Polar H7 heart rate sensor.

Blood collection and analysis

Venous blood samples were collected from the forearm with subjects in a seated position. The first sample was collected at rest 30 min before Trial1 and the second sample was collected 5 min after Trial1. The other two blood samples were donated at 1st and 2nd recovery times. The last blood sampling was done 5 min after Trial 2. Blood was centrifuged at 1500*g* for 15 min at 4 °C. Resulting serum was aliquoted and stored at − 80 °C for analysis. Serum creatine kinase (CK) was measured using colorimetric procedures at 340 nm. Serum CK at 1st and 2nd recovery time was assessed with ELISA assay (Tecan, Infinite F50 microplate reader) and used for the next analyses.

Also, capillary blood samples were obtained via finger prick from the forefinger for blood lactate (BLa) concentration analysis immediately before and immediately after each test. Blood lactate concentrations were analyzed on-site using a lactate analyzer (Lactate Scout, Senslab GmbH Leipzing, Germany).

Statistical analysis

All results are reported as mean ± SD. A paired (Trial1–Trial2) Student's *t* test was run separately to determine whether ergo tests had any difference. A 2 (group and time) × 3 (two caffeine doses and placebo) design repeated-measures ANOVA (with Tukey post hoc) was conducted to compare differences for all variables. The Kolmogorov–Smirnov test was used to test the normality of the distribution. Statistical power was at the 0.88 level where significance was set at $p \leq 0.05$ for all analyses. Effect size was calculated using Cohen's *d*. Statistical analyses were performed using the software program SPSS, version 21.0 (Statistical Package for Social Science, Chicago, IL, USA).

Results

Ventilatory and cardiac characteristics at performance

$\dot{V}O_{2peak}$

As shown in Table 1, mean value of $\dot{V}O_{2peak}$ increased significantly during Trial1 by ingesting 6 mg kg^{-1} caffeine compared to preliminary values (10.1%, $P = 0.03$). Also 6 mg kg^{-1} caffeine ingestion during Trial2 caused to significant enhance in mentioned value when compared to placebo-ingested Trial2 group (13.9%, $P = 0.01$). $\dot{V}O_{2peak}$ in placebo group decreased significantly when compared to preliminary and Trial1 ($P = 0.04$ and 0.01, respectively). 3 mg kg^{-1} caffeine ingestion did not cause significant difference in Trial1 and 2 compared to preliminary values. Also, this variable did not significantly change after ingestion of 6 mg kg^{-1} caffeine or placebo ($P = 0.31$ and 0.56, respectively).

$HR_{\dot{V}O_{2peak}}$

Mean value of HR at $\dot{V}O_{2peak}$ increased significantly during Trial1 and 2 by ingesting of 6 mg kg^{-1} caffeine compared to preliminary values (10.0%, $P = 0.02$ and 11.3%, $P = 0.00$ respectively). Also 6 mg kg^{-1} caffeine ingestion during Trial1 caused significant enhance in this variable than placebo (5.5%, $P = 0.04$). In comparison to preliminary values, there were significant increases in Trial1 and 2 after ingestion of 3 mg kg^{-1} caffeine (7.1%, $P = 0.04$ and 7.6%, $P = 0.03$, respectively).

$\dot{V}O_2/HR_{peak}$

The findings demonstrate that after 6 mg kg^{-1} supplementation, $\dot{V}O_2/HR_{peak}$ significantly increases in Trial1 and 2 (19.6%, $P = 0.00$ and 17.3%, $P = 0.00$), respectively.

Table 1 Preliminary, Trial1, and Trial2 performance values for peak oxygen uptake ($\dot{V}O_{2peak}$), heart rate at $\dot{V}O_{2peak}$ ($HR_{\dot{V}O_{2peak}}$), O_2puls ($\dot{V}O_2$ /HR_{peak}), oxygen uptake in lactate threshold ($\dot{V}O_2$ at LT), and ventilatory evaluation ($\dot{V}_E$) in different ingesting regimens

Group	3 mg kg^{-1}	6 mg kg^{-1}	Placebo
$\dot{V}O_{2peak}$ (ml kg^{-1} min^{-1})			
Preliminary	50.81 ± 4.12	52.21 ± 3.18	51.12 ± 4.49
Trial1	52.72 ± 4.18	57.65 ± 2.57*	52.31 ± 5.37
Trial2	49.76 ± 4.29	53.15 ± 5.31†	46.66 ± 5.03
$HR_{VO_{2peak}}$(b min^{-1})			
Preliminary	170.2 ± 7.1	169.4 ± 6.3	171.4 ± 8.1
Trial1	182.4 ± 6.4*	188.4 ± 5.7*,†	178.6 ± 6.8
Trial2	183.3 ± 7.5*	188.6 ± 5.1*	180 ± 4.2
$\dot{V}O_2$ /HR_{peak} (ml b min^{-1})			
Preliminary	19.74 ± 2.9	19.41 ± 1.9	20.08 ± 3.1
Trial1	20.31 ± 2.5	23.23 ± 2.3*	20.52 ± 2.3
Trial2	18.87 ± 2.2$^{\#}$	22.77 ± 3.8*,†	17.53 ± 2.2*
$\dot{V}O_2$ at LT (l min^{-1})			
Preliminary	2.07 ± 0.37	2.13 ± 0.54	2.04 ± 0.42
Trial1	2.23 ± 0.44$^{\#}$	2.78 ± 0.39*	2.12 ± 0.42
Trial2	1.88 ± 0.36$^{\#}$	2.22 ± 0.34*	1.89 ± 0.39
$\dot{V}_E$ (ml kg^{-1} min^{-1})			
Preliminary	50.81 ± 4.12	52.21 ± 3.74	51.12 ± 4.49
Trial1	52.72 ± 4.18	56.65 ± 4.57*	52.31 ± 5.37
Trial2	48.76 ± 4.29$^{\#}$	53.15 ± 5.31†	46.66 ± 5.03*

Data are means (± SD)

*Significantly greater than preliminary value ($p < 0.05$)

†Significantly different change with Placebo group ($p < 0.05$)

$^{\#}$Significantly different change with 6 mg kg^{-1} ingestion group ($p < 0.05$)

In Trial2, this ratio vas significantly higher in 6 mg kg^{-1} ingestion than 3 mg kg^{-1} (20.6%, $P = 0.00$) and Placebo (29.8%, $P = 0.00$). Also the difference between 3 mg kg^{-1} caffeine and placebo was significant (7.6%, $P = 0.03$).

$\dot{V}O_2$ at LT

Mean value of $\dot{V}O_2$ at LT increased significantly during Trial1 by ingesting 6 mg kg^{-1} caffeine compared to pre-test (30.1%, $P = 0.00$). Also 6 mg kg^{-1} caffeine ingestion during Trial1 caused significant enhance in this variable than 3 mg and placebo ingestion (21.1%, $P = 0.00$ and 24.6%, $P = 0.00$ respectively). In Trial2, there were significant increases in 6 mg kg^{-1} ingestion compared to 3 mg kg^{-1} and placebo ingestion (18.0%, $P = 0.00$ and 17.4%, $P = 0.01$, respectively).

Table 2 Recovery values for minute ventilation (MV), respiratory exchange ratio (RER), and breathing frequency at baseline (rest), 30 min after Trial1 (1st recovery), and 30 min before Trial2 (2nd recovery)

Group	3 kg^{-1}	6 mg kg^{-1}	Placebo
MV (l min^{-1})			
Baseline	12.24 ± 3.08	11.57 ± 2.23	11.93 ± 2.49
1st	13.38 ± 2.56	14.89 ± 2.72*,†	12.71 ± 2.62
2nd	12.37 ± 2.41	14.32 ± 2.59*,†	11.12 ± 2.23
RER			
Baseline	0.87 ± 0.10	0.86 ± 0.11	0.87 ± 0.11
1st	1.02 ± 0.11*$^{\#}$	1.12 ± 0.10*,†	1.03 ± 0.13*
2nd	0.89 ± 0.10	0.91 ± 0.09	0.88 ± 0.11
BF (Breaths min^{-1})			
Baseline	14.6 ± 4.3	14.4 ± 2.1	14.6 ± 3.5
1st	14.9 ± 3.7	14.6 ± 2.8	14.7 ± 2.9
2nd	14.5 ± 2.8	14.8 ± 3.2	14.4 ± 3.1

Data are means (± SD)

*Significantly greater than preliminary value ($p < 0.05$)

†Significantly different change with placebo group ($p < 0.05$)

$^{\#}$Significantly different change with 6 mg kg^{-1} ingestion group ($p < 0.05$)

$\dot{V}_E$

In comparison between preliminary and Trials, there was 8.5% increase for 6 mg kg^{-1} caffeine ingestion in Trial1 ($P = 0.04$) and 9.5% decrease in placebo ingestion in Trial2 ($P = 0.03$). Also, the data revealed that there was significant decrease between 6 mg kg^{-1} ingestion with 3 mg kg^{-1} (9.0%, $P = 0.03$) and placebo ingestion (13.9%, $P = 0.01$).

Recovery and resting baseline values

MV at rest

In minute ventilation, there was 28.2% increase for 6 mg kg^{-1} caffeine ingestion 30 min after Trial1 ($P = 0.00$) and 23.7% increase 30 min before Trial2 ($P = 0.00$). Also, this variable was significantly higher in 6 mg kg^{-1} ingestion from placebo after Trial1 (17.1%, $P = 0.01$) and before Trial2 (28.7%, $P = 0.00$) (Table 2).

RER at rest

Mean value of RER increased significantly 30 min after Trial1 by ingesting 6 mg kg^{-1} caffeine (30.2%, $P = 0.00$) and 3 mg kg^{-1} (17.1%, $P = 0.00$) compared to baseline values. Also 6 mg kg^{-1} caffeine ingestion during Trial1 caused significant enhance in this variable than 3 mg kg^{-1} (9.8%,

Table 3 Average heart rate in different periods and Trials (mean ± SD)

Heart rate	3 mg kg^{-1}	6 mg kg^{-1}	Placebo
Pre-Trial1 (before Trial1)	61.8 ± 5.2	63.1 ± 4.7	62.7 ± 4.4
Trial1	172.9 ± 8.4	171.7 ± 9.9	176.7 ± 9.9
Pre-Trial2 (before Trial2)	62.1 ± 4.6	61.3 ± 5.5	61.8 ± 3.9
Trial2	175.8 ± 6.8	176.8 ± 11.8	174.8 ± 11.8

Table 4 Blood lactate concentrations in preliminary test, during Trial1 and 30 min after Trial1 (1st recovery time), 30 min before Trial2 (2nd recovery time), and during Trial2 of three ingestions (mean ± SD)

Lactate concentration (mmol l^{-1})	3 mg kg^{-1}	6 mg kg^{-1}	Placebo
Preliminary	1.95 ± 0.09	1.96 ± 0.06	1.98 ± 0.06
Trial1	9.26 ± 0.58$^{\#}$	10.40 ± 0.57†	8.77 ± 0.69
1st recovery time	2.01 ± 0.1$^{\#}$	2.35 ± 0.05†	2.04 ± 0.05
2nd recovery time	1.90 ± 0.08‡	1.99 ± 0.07,†‡	1.82 ± 0.09‡
Trial2	10.81 ± 0.59$^{\dagger,\#}$	11.69 ± 0.64†	9.39 ± 0.71

Data are means (± SD)

†Significantly greater than Placebo in the same time ($p < 0.05$)

$^{\#}$Significantly different change with 6 mg kg^{-1} in the same ingestion ($p < 0.05$)

‡Significantly different from 1st recovery time values

$P = 0.04$) and placebo ingestion (9.9%, $P = 0.04$). Also, there was no significant change between three groups 30 min before trial2 (Table 2).

BF at rest

Relative to placebo ingestion, there was no significant difference between breathing frequency in 3 mg kg^{-1} or 6 mg kg^{-1} in Trial1 or 2 ($P = 0.42$). Also, the differences of three ingestions were not significant compared to baseline BF ($P = 0.58$).

Serum CK, BLa, and HR in recovery times

Tables 3 and 4 present effects of 3 or 6 mg kg^{-1} caffeine and placebo ingestion on serum CK, HR, and BLa. CK from 1st recovery period to 2nd recovery period significantly increased (6 mg kg^{-1}: 29.2% from 722.6 ± 121.4 U L^{-1} in 1st to 933.6 ± 128.5 U L^{-1} in 2nd; 3 mg kg^{-1}: 21.8% from 743.3 ± 110.2 U L^{-1} in 1st to 905.9 ± 121.2 U L^{-1} in 2nd; Placebo: 25.6% from 695.3 ± 116.3 U L^{-1} in 1st to 873.3 ± 138.7 U L^{-1} in 2nd recovery time). Also the difference between 6 mg kg^{-1} with 3 mg kg^{-1} (33.9%, $P = 0.00$, $d = 1.42$) and placebo (14.1%, $P = 0.01$, $d = 1.71$) were significant. The HR from Trial1 to Trial2 (6 mg kg^{-1}: Pre = 171.7 ± 9.9 b min^{-1} vs. Post = 176.8 ± 11.8 b min^{-1}; 3 mg kg^{-1}: Pre = 172.9 ± 8.4 b min^{-1} vs. Post = 175.8 ± 6.8 b min^{-1}; Placebo: Pre = 176.7 ± 9.9 b min^{-1} vs. Post = 174.8 ± 11.8 b min^{-1}) showed no significant differences among groups ($P = 0.35$, $d = 0.46$; $P = 0.87$, $d = 0.38$; and $P = 0.65$, $d = 0.17$, respectively).

BLa in all groups increased significantly in both performances compared to baseline. BLa in 6 mg kg^{-1} caffeine ingestion was higher than 3 mg kg^{-1} and placebo in Trial1 (12.3%, $P = 0.01$, $d = 0.76$ and 18.6%, $P = 0.00$, $d = 2.58$, respectively) and 2 (8.1%, $P = 0.03$, $d = 2.18$ and 24.4%, $P = 0.00$, $d = 3.43$). Also, it was significantly higher than 3 mg kg^{-1} and placebo ingestion in recovery1 (16.9%, $P = 0.02$, $d = 0.37$ and 15.1%, $P = 0.01$, $d = 0.61$) and higher than placebo in recovery2 (9.3%, $P = 0.03$, $d = 2.12$). In 3 mg kg^{-1} ingestion, BLa was significantly more than placebo in Trial2 (15.1%, $P = 0.00$, $d = 1.73$) (see Fig. 2).

Mean values of performance variables in Trial1 and Trial2 of three different ingestions are presented in Table 5.

In Trial1, 6 mg kg^{-1} caffeine ingestion significantly improved the 2000-m performance compared to placebo ingestion (2.7%, $P = 0.04$), but this improvement was not shown after 3 mg kg^{-1} ingestion (1.7%, $P = 0.53$). Mean power output (MPO) significantly increased in 6 mg kg^{-1} and 3 mg kg^{-1} ingestion compared to placebo (8.2%, $P = 0.02$ and 4.5%, $P = 0.03$, respectively), as well. In spite of difference in 2000-m time ($P = 0.71$) between 6 mg kg^{-1} and 3 mg kg^{-1} caffeine, this difference was not significant, while MPO for 6 mg kg^{-1} showed significant difference with 3 mg kg^{-1}(4.7%, $P = 0.03$).

In Trial2, although 6 mg kg^{-1} and 3 mg kg^{-1} caffeine ingestion did not show significant difference in 2000-m TT performance compared to placebo ingestion (1.1%, $P = 0.73$ and 0.2%, $P = 0.95$), there was ~ 8 s decrease in 2000 m time in 6 mg kg^{-1} compared to placebo. Also compared to Trial1, record of both of caffeine-ingested groups impaired significantly (4.6%, $P = 0.04$ and 3.9%, $P = 0.04$). MPO decreased significantly in 6 mg kg^{-1} (5.6%, $P = 0.03$) and 3 mg kg^{-1} (5.1%, $P = 0.03$) caffeine ingestion, but not in placebo (0.7%, $P = 0.91$).

In reviewing the data recorded from each rower, six out of nine participants in 6 mg kg^{-1} ingestion ($P = 0.04$) and four participants in 3 mg kg^{-1} ingestion ($P = 0.04$) recorded significantly better 2000-m TT compared to Placebo in Trial2. However, time of all participants showed impairment in Trial2 compared to Trial1 ($P = 0.01$). In addition, all participants in 6 mg kg^{-1} ($P = 0.02$) and three participants in 3 mg kg^{-1} caffeine ingestion ($P = 0.04$) showed significantly increased power output compared to placebo in Trial2. However, all rowers showed impaired power output in Trial2 compared to Trial1 ($P = 0.00$).

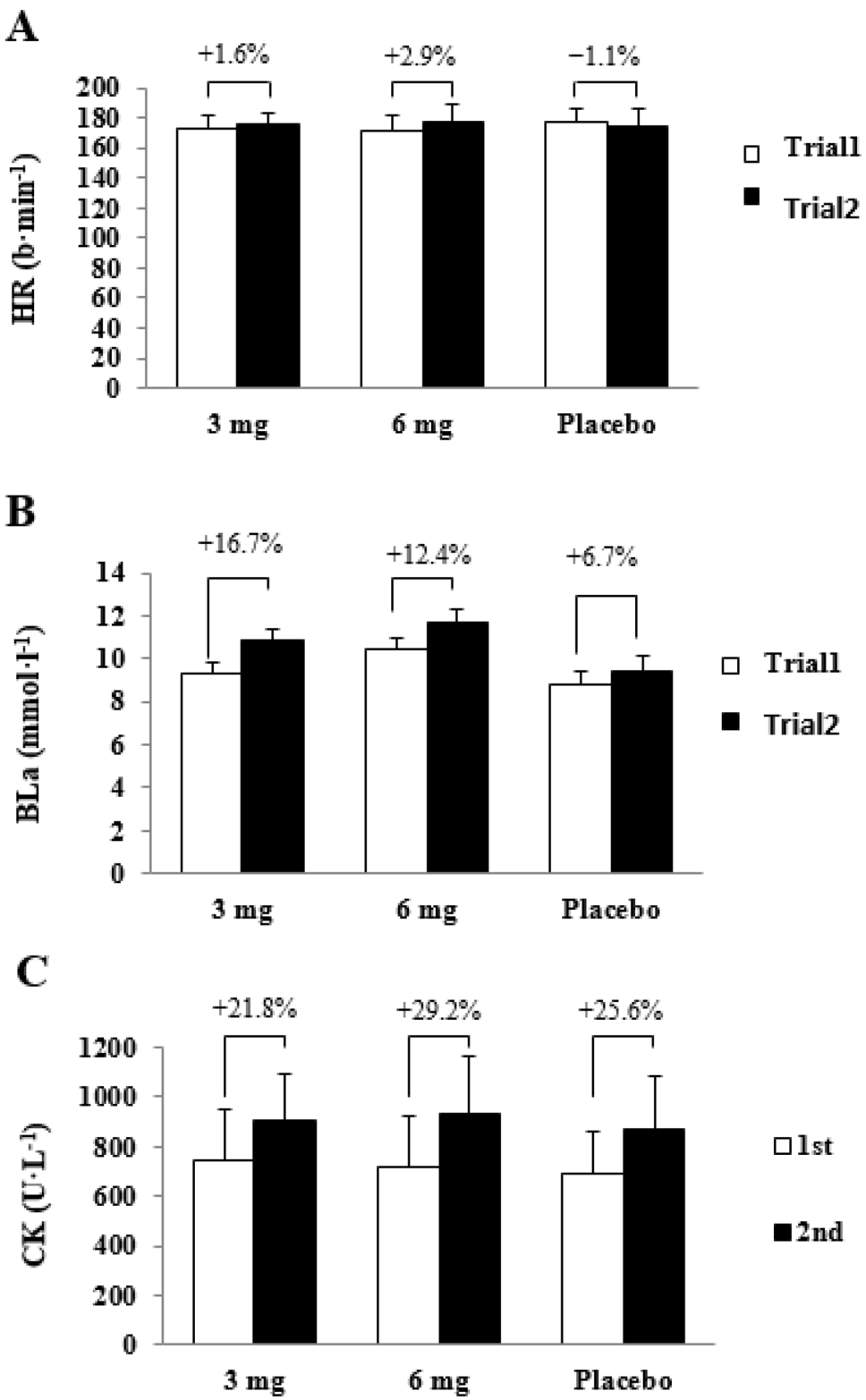

Fig. 2 Effects of 3 or 6 mg kg^{-1} caffeine and placebo ingestion on **a** HR (Heart Rate) from pre-trial (after Trial1) to post-trial (after Trial2), **b** ΔBLa (blood lactate) from pre-trial (after Trial1) to post-trial (after Trial2), and **c** serum ΔCK (Creatine Kinase) from pre-trial (after Trial1) to post-trial (before Trial2). Values above the bars denote percent changes of HR, BLa, and CK (pre- to post-trial)

Discussion

The aim of the present study was to determine the effects of caffeine on ergo rowing performance and recovery. The main findings were that compared to placebo, 6 mg kg^{-1} caffeine ingestion before Trial1 promoted 2000-m record and MPO. However, 3 mg kg^{-1} did not affect any of the variables significantly. Moreover, VO_{2peak}, $HR_{VO_{2peak}}$, $\dot{V}O_2/HR_{peak}$, $\dot{V}O_2$ at

Table 5 Mean performance characteristics (mean ± SD) of three different ingestions in Trial1 and Trial2 ($n = 9$)

Rowing time trial	Stroke rate (rate/min)			Mean power output (W)			2000-m time (s)		
Trials	3 mg kg^{-1}	6 mg kg^{-1}	Placebo	3 mg kg^{-1}	6 mg kg^{-1}	Placebo	3 mg kg^{-1}	6 mg kg^{-1}	Placebo
Trial1	28.50 ± 1.38	28.54 ± 1.69	28.12 ± 1.88	307.2 ± 12.4	321.2 ± 13.2†	296.7 ± 13.1	414.2 ± 12.9	407.1 ± 11.5†	418.4 ± 11.5
Trial2	28.91 ± 2.19	28.72 ± 1.48	28.75 ± 1.58	287.0 ± 12.2*	298.7 ± 11.1*	285.6 ± 14.9	423.3 ± 10.8*	417.8 ± 11.5*	426.3 ± 12.7$^{\#}$

Data are means (±SD)

†Significantly greater than Placebo in the same trial ($p < 0.05$)

$^{\#}$Significantly different changes with trial2 in the same ingestion ($p < 0.05$)

LT, and $\dot{V}_E$ significantly increased in 6 mg kg^{-1} caffeine group, while only $HR_{V\dot{O}_{2peak}}$ increased after ingesting 3 mg kg^{-1} caffeine. Also BLa accumulated more in 6 mg kg^{-1} compared to 3 mg kg^{-1} or placebo. More interestingly, most of these values decreased in Trial2 except BLa and $HR_{V\dot{O}_{2peak}}$.

The other main results demonstrated in recovery phase after Trial1. Our findings revealed that compared to placebo, MV and RER enhanced in 6 mg kg^{-1} caffeine ingestion 30 min after Trial1 and 30 min before Trial2 at rest. CK at 1st recovery time was significantly higher than 2nd recovery in all the groups, but the difference between groups was not significant in 1st and 2nd recovery times.

Performance phase

According to higher $\dot{V}O_{2peak}$, HR at $\dot{V}O_{2peak}$ and $\dot{V}O_2$/HR, and in accordance with improvements in MPO and 2000-m TT, it seems that 6 mg kg^{-1} caffeine ingestion may increase the ability of cardiovascular system to match its oxygen delivery to working muscles with the changes in volume or pressure overload by increasing stroke volume (SV) or a-$\bar{v}O_2$ (arterial-venous O_2 Concentration) difference [28]. Discrepancies in previous studies stating no effect of caffeine on $\dot{V}O_2$ [4, 5, 15] versus a significant increase in this ventilatory variable [30], or no effect of caffeine on V_E in submaximal trials [31, 32] versus a significant increase on V_E [4, 5] make it sense to be discussed.

Previous studies explained the increase in $\dot{V}O_{2peak}$ by (a) increase in oxygen delivery (i.e., increases in SV); and (b) improvement in oxygen utilization by active muscles [33–35]. Given that O_2pulse [the ratio of $\dot{V}O_2$ and HR ($\dot{V}O_2$/HR)] is a good means of appraising SV and a-$\bar{v}O_2$ difference indirectly during exercise [34], an important correlation between the evolution of $\dot{V}O_2$/HR and SV during maximal exercise was reported previously [36, 37]. Hence, increased SV and a-$\bar{v}O_2$ difference seems to corroborate significant increases in V_E and $\dot{V}O_{2peak}$ [29].

Also caffeine may increase Ca^{2+} permeability in the sarcoplasmic reticulum of muscle fibers [20] causing to increase cardiac tissue contractibility which assumes to be a reason for the increase in SV after 6 mg kg^{-1} caffeine ingestion.

The other finding on significant increases in HR_{peak} and BLa is inconsistent with the previous findings [38–40] while it assumes to be correlated to a clear physiological effect of caffeine on 2000-m rowing performance [6]. But in 3 mg kg^{-1} caffeine and placebo ingestion, it seems that increases in HR at $\dot{V}O_{2peak}$ are lonely a response to overload pressure and this change cannot improve performance. In Trial2, both of 2000-m TT and MPO decreased after 3 mg kg^{-1} ingestion. Even though performance

characteristics impaired in this group, 2000-m TT in 6 mg kg^{-1} ingestion was ~ 7 s better than placebo and ~ 9 s better than 3 mg kg^{-1} caffeine ingestion.

In agreement with these findings, the previous studies reported 1% improvement in performance time and 3% improvement in MPO after ingesting 6 and 9 mg kg^{-1} caffeine [5], or 1.4% increase in MPO and a 0.7% reduction in time to complete a 2000-m TT with 6 mg kg^{-1} and 2.7% increase in MPO and 1.3% reduction in 2000-m time with 9 mg kg^{-1} caffeine [4]. By contrast, a study reported no effect of 2, 4, or 6 mg kg^{-1} caffeine on the 2000-m TT rowing performance [6].

Other main finding of the present study was that BLa increased higher in 6 mg kg^{-1} caffeine ingestion than in 3 mg kg^{-1} or placebo immediately after Trial1, while this increase accompanied to higher $\dot{V}O_2$ at LT and $\dot{V}O_{2peak}$. It seems that simultaneous increases in these characters make lactate threshold (LT) possible to approach at higher concentrations [15]. It is a common belief that LT and maximum lactate values are influenced by preceding performance and muscle glycogen stores [41]. Also, it was demonstrated that marked improvements in LT are concomitant with modest changes in the oxidative capacity of trained muscles [42]. However, along with the aforementioned studies, findings in the present study support this notion that improvement in LT is a consequence of the change in $\dot{V}O_{2peak}$ [43].

The research literature often reports an increase in BLa with caffeine following exercise [15, 44] while there are several discrepancies toward these findings [30, 32]. In submaximal performances with high oxygen uptake such as 2000-m rowing TT, the oxidation of pyruvate is limited by inadequate oxygen supply and thus directed toward the formation of lactate in the range of 70–85% of peak power output known as lactate threshold [41].

Recovery phase

Ingesting caffeine pre-exercise to benefit from its ergogenic effect is common. Effects of caffeine persist post-exercise due to its prolonged biological half-life [16] mostly if performance is not too long (≤ 60 min). Present findings demonstrated that MV and RER enhanced in 6 mg kg^{-1} caffeine ingestion in both 1st and 2nd recovery times compared to placebo. Also, CK at 1st recovery time was significantly higher than 2nd in all the groups, but the difference between groups was not significant in 1st and 2nd recovery times.

In this notion, scientists have noted that V_E (MV) is based upon VT and BF which are effective on cardiac output [45, 46]. Sheykhlouvand et al. revealed that improvement of the $\dot{V}$E@VT was mediated by simultaneous increases of both BF@VT and $\dot{V}$T@VT [35]. Present findings for these variables did not show significant changes in BF at 1st and 2nd recovery time. But V_E increased after 6 mg kg^{-1} caffeine ingestion. Previous studies attributed the changes in breathing patterns to respiratory muscle fatigue [47, 48]. Due to impaired recorded time and mean power output (MPO) in Trial2 and elevated BLa and CK after Trial1, respiratory muscle fatigue is presumable. Hence, unchanged BF may accompany deeper ventilation (more tidal volume) in caffeine-ingested group in recovery times. While during Trials, respiratory muscle fatigue leads to rapid shallow breathing through increase in BF and decrease in tidal volume [45].

In reviewing data, BF did not show significant difference between groups and from baseline resting values. But BLa values were higher in 6 mg kg^{-1} caffeine ingestion compared to 3 mg kg^{-1} and placebo in 1st recovery time, and only higher than placebo in 2nd recovery time. Even though performance characteristics impaired in Trial2, 2000-m TT in 6 mg kg^{-1} ingestion was ~ 7 s better than placebo and ~ 9 s better than 3 mg kg^{-1} caffeine ingestion. It seems likely that 6 mg kg^{-1} caffeine ingestion caused better recovery. Then again, this dose-dependent reduction in Trial2 impairment approves the probability of central and peripheral effects of caffeine associated with the antagonism of the various adenosine receptors' subtypes and caffeine-stimulated increase in glycolysis [15]. In 1st recovery time, the significant increase in RER in all the groups compared to baseline revealed that $\dot{V}CO_2$ is more than baseline after Trial1. Moreover, significant increases in RER in 6 mg kg^{-1} and 3 mg kg^{-1} caffeine compared to placebo in 1st recovery time suggest the significant caffeine-induced increase in resting measures of RER. These findings are in accordance with Glaister et al. (2015) who reported RER caffeine-induced increases [15]. Although BLa decreased significantly in all groups in 2nd recovery time compared to 1st recovery time, the likelihood of physiological (ventilatory and cardiovascular) responses to stress on body mechanism, the BLa clearance was more in both of caffeine-digested groups than Placebo. This finding is in contrast with Glaiser et al. who reported no effect of caffeine on BLa clearance in recovery period [15].

CK significantly increased in 2nd recovery time compared to 1st in all groups, whereas increases in 6 mg kg^{-1} caffeine were 14.1% more than Placebo and 33.9% more than 3 mg kg^{-1} caffeine ingestion.

The exercise-induced increase in serum CK concentration is an indirect marker of muscle damage [24]. The efflux of CK from skeletal muscle may occur as a result of increases in the permeability of the muscle cell membrane and/or the temporary reorganization of the intramuscular vasculature [49] resulting in delayed onset of muscle soreness (DOMS) [24]. In our participants, CK concentration at 2nd recovery time was in the range demonstrated by athletes in a previous study [50]. Significant difference following ingestion of 6 mg kg^{-1} caffeine compared to Placebo and 3 mg kg^{-1} ingestion was in contrast with reports of a study about the insignificant difference in CK response with 24- and 48-h

recovery [24]. As an adenosine antagonist, caffeine affects central nervous system activity by blocking adenosine receptors, thus resulting in attenuation of DOMS [21]. On the other hand, caffeine ingestion in post-exercise resting conditions has been shown to result in a reduction in insulin-mediated glucose disposal and increases in epinephrine [25] which might be mediated by both β-adrenergic stimulation and adenosine-receptor antagonism [51] causing to its positive effects on post-exercise muscle glycogen resynthesis [19, 23, 25]. In addition, caffeine resulted in less increase in plasma potassium during prolonged exercise leading to increased removal of potassium from the t tubules and delaying the onset of fatigue processes [25]. Both reduced feelings of pain and soreness and induced glycogen resynthesis may play a role in enhanced performance through increased ability to do work [23, 24]. Albeit Graham et al. (2008) found no difference between caffeine and placebo Trials on post-exercise (70–85% VO_{2max}) muscle glycogen concentrations [39]. This discrepancy can be a result of relatively aerobic nature of those exercises recruited.

As shown in Fig. 3, in Trial2, 6 participants in 6 mg kg^{-1} ingestion and 4 participants in 3 mg kg^{-1} ingestion recorded significantly better 2000-m TT compared to placebo, whereas all participants showed impairment rather than Trial1. Moreover, all participants in 6 mg kg^{-1} and three participants in 3 mg kg^{-1} caffeine group showed significantly increased power output compared to placebo in Trial2, while MPO impaired for all rather than in Trial1. Given that individual changes in MPO and 2000-m TT was variable between participants after 6 mg kg^{-1} caffeine ingestion relative to that of Placebo and 3 mg kg^{-1}, it seems likely that the effects of caffeine on performance and recovery depend on individual metabolic and physiologic status of the rowers. Meanwhile, less relative individual impairments in Trial2 were shown in the rowers whose performance was better in Trial1 (participants coded as 3,4 and 5), and it can be speculated that the more the movement and physiological efficiency is, the less the changes occur by caffeine ingestion. However, this idea needs to be investigated more to establish a stronger basis for the individual effects of caffeine on performance and recovery.

The results stating less impairment after 6 mg kg^{-1} caffeine ingestion support increased capacity of high-intensity interval-running after ingesting caffeine immediately post-exercise and 2-h post-exercise [22]. Although the precise mechanisms underpinning the ergogenic effects of caffeine on exercise performance are not fully understood, based on recent studies, it is reasonable to suggest that the enhanced performance effect in 6 mg kg^{-1} caffeine 6 h after an initial testing session may in part be a result of increased rates of muscle glycogen resynthesis [23], reduced perceptual fatigue and pain, increased muscle ability to recruit motor units [24], and better intracellular Ca^{2+} concentration [7, 20] causing to enforce muscle contraction. The other reason assumes to be related to more BLa reduction at recovery period in 6 mg kg^{-1} caffeine compared to placebo, suggesting that oxidative metabolism assumes to be dominant activity to supply energy during recovery period [15].

However, while there is abundant information on the consequences of caffeine ingestion on performance, information about the effects of pre-exercise caffeine ingestion on subsequent recovery period is scarce. Hence, the aim

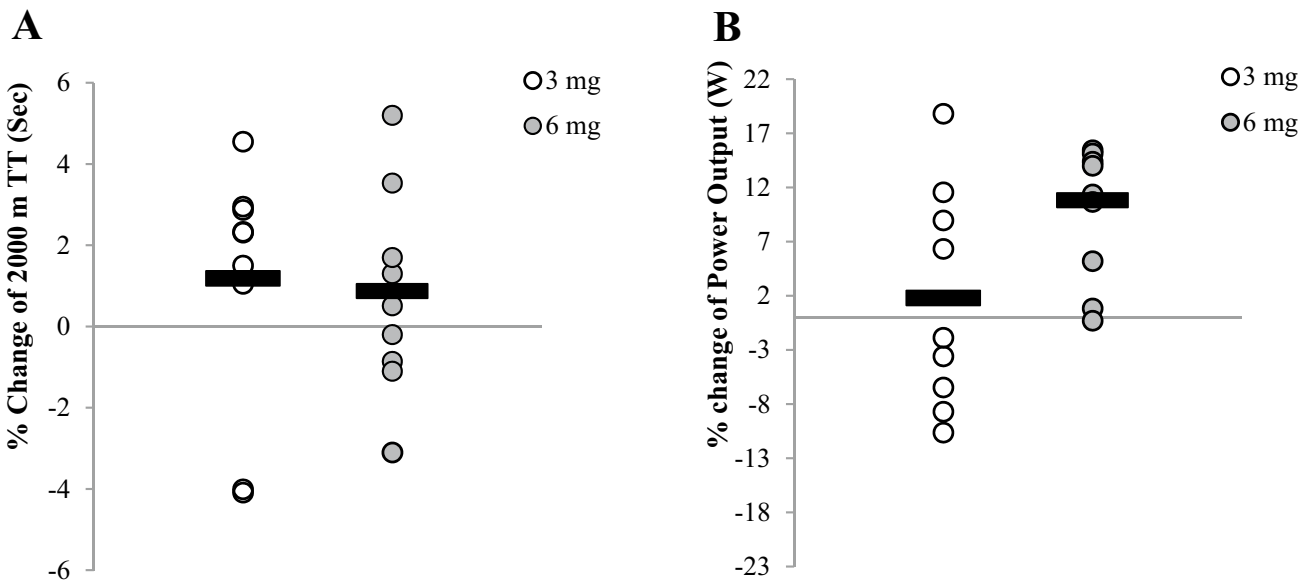

Fig. 3 Individual effects of 3 or 6 mg kg^{-1} of caffeine ingestion on relative percent change in **a** 2000-m rowing TT and **b** power output. Circles show individual percentage change from baseline (placebo) and horizontal bars show mean group percentage change from baseline ($n = 9$)

of the current study was to determine whether ingestion of caffeine improves performance and subsequent recovery period together with any improvement in repeated short-term high-intensity endurance performances compared with the ingestion of PL.

Conclusion

The most striking findings of present study were: (a) the consumption of 6 mg kg^{-1} of caffeine before an intensive rowing TT significantly promoted performance during the next TT performed 6 h later; (b) caffeine affected magnitude of the changes in serum CK, BLa, and ventilatory characteristics.

Although we cannot clearly offer precise mechanisms underpinning this improved performance rather than placebo after recovery period, ability to recruit more motor units and reduce perceptual fatigue together with augmented rates of muscle glycogen resynthesis during recovery after caffeine ingestion might be the reasons. Given into multifactorial role of $\dot{V}O_{2max}$, lactate/ventilatory threshold and oxygen uptake kinetics into account for endurance performance, such improvements in performance could be expected. Based on positive changes in ventilatory variables, and CK and BLa levels with caffeine, this study provides novel data demonstrating that caffeine ingestion improves performance and subsequent recovery from rowing effort. These prescriptions could be applied as a practical method especially for rowers, who need to repeat those relatively intensive competitions to be qualified for upper levels (repechages, semifinals, and finals) in the same day.

Considerably, further studies are needed to investigate the effects of caffeine on other purposed communities than young male rowers and additional metabolites measurements to fully test these hypotheses.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval All methods performed in the study were in accordance with ethical standards of the national research committee and with the 1964 Helsinki Declaration. The study was approved by the ethical committee of Iran Canoe, Rowing and Sailing National Academy.

Informed consent Informed consent was obtained from all individual participants included in the study.

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