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The effect of 10 days of intermittent fasting on Wingate anaerobic power and prolonged high-intensity time-to-exhaustion cycling performance

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Abstract
Many physically active individuals have undertaken intermittent fasting to reduce their daily caloric intake. However, abstaining from meals for a specific length of time may lead to the acute disturbance of highly carbohydrate-dependent exercise performance. The purpose of this study was to observe the effect of 10 days of intermittent fasting on high-intensity type exercises, Wingate anaerobic (WT) and prolonged high-intensity time-to-exhaustion (HIT) cycling test. Twenty participants were randomised into an intermittent fasting (FAS) and a control group (CON). One day after baseline data collection on Day-0 where participants consumed their recommended daily caloric intake (FAS = 2500 ± 143 kcal day\(^{-1}\); CON = 2492 ± 20 kcal day\(^{-1}\)) served over a course of five meals, the FAS group consumed only four meals where 40% was restricted by the omission of lunch (FAS = 1500 ± 55 kcal day\(^{-1}\)). This diet was then continued for 10 days. Data on exercise performance and other dependent variables were collected on Day-2, -4, -6, -8 and -10. A reduction in WT power in the FAS group was observed on Day-2 (821.74 ± 66.07 W) compared to Day-0 (847.63 ± 95.94 W) with a moderate effect size (\(p < .05, ES = 0.4\)), while HIT time-to-exhaustion performance declined over the 10 days with a trend of recovery from a large to a minimum effect size (\(p < .05, ES = 0.8-0.3\)). Body weight and triglyceride were consistently reduced in the FAS group (\(p < .01\)). The present study suggests that intermittent fasting must exceed 10 days to ensure that high-intensity performance does not deteriorate because this length of time seems to be required for effective adaptation to the new dietary regimen.

Keywords: Exercise, performance, nutrition

Highlights
• This study highlights that athletes should be wary when practicing fasting with low caloric intake for Wingate anaerobic test and high-intensity cycling time-to-exhaustion which are highly carbohydrate-dependent exercise performance, since:
  • both performances are shown to be attenuated at the beginning of the practice.
  • Wingate anaerobic performance requires at least 4 days for adaptation.
  • Cycling time to exhaustion requires more than 10 days to adapt.

Introduction
Fasting can be defined as the absence of caloric and fluid ingestion in a specific window of time in a day where the post-consumption period usually lasts for several hours after the first meal (Maughan, Fallah, & Coyle, 2010). The practice of fasting in relation to exercise has long been studied; hence its effect on metabolic regulation is now better understood. In general, it is clear that the acute effect of caloric deprivation is detrimental to exercise performance. However, it is less clear whether the prolonged practice of this diet affects exercise performance in the same way.

Bangsbo, Graham, Kiens, and Saltin (1992) reported that the time-to-exhaustion (TTE) during a repeated bout of 15-s high-intensity exercise was prolonged when carbohydrate was given sufficiently prior to exercise. While, glycogen concentration in the vastus lateralis muscle showed a 14% reduction even after performing only a 6-s bout of exhaustive exercise on a cycle ergometer (Gaitanos, Williams, Boobis, & Brooks, 1993). These studies show that high-intensity
exercise is associated with a rapid breakdown of muscle glycogen, which implies that this type of exercise is dependent on carbohydrate availability. It is therefore plausible that reducing the caloric intake reduces carbohydrate availability and is thus a limiting factor in sustaining a high-power output.

However, it has been suggested that the metabolic rate is not substantially affected by intermittent fasting and caloric deprivation, due to continued oxidative metabolic mechanisms that meet energy need (Maughan et al., 2010). In one study, the rate of carbohydrate utilisation during resting decreases under the fasted state while the energy demand is met by an increased rate of fat oxidation (Cahill et al., 1966). This is a physiological response to spare the limited carbohydrate reserve from energy deprivation to maintain tissue’s basal function. One of the primary responses of fasting is the mobilisation of triglyceride, which leads to an increase in plasma free fatty acids and therefore increases the availability of this energy source to working muscles (Cahill et al., 1966). The intracellular mechanism by which free fatty acid availability is increased is therefore believed to be responsible for suppressing carbohydrate oxidation, which in turn affects the performance of exercises that are highly carbohydrate-dependent. Apart from the reduction in carbohydrate availability during fasting having an effect on exercise performance, where carbohydrates are predominantly utilised as fuel (Artioli et al., 2010; Rankin, Ocel, & Craft, 1996), dehydration could also be a contributing factor, where cardiovascular strain and an increase in lactate concentration could negatively impact exercise performance (Webster, Rutt, & Weltman, 1990).

Studies have consistently shown that acute fasting (1–4 days) is detrimental to high-intensity exercises that require near to maximal oxygen uptake (Gleeson, Greenhaff, & Maughan, 1988; Horswill, Hickner, Scott, Costill, & Gould, 1990; James, Mears, & Shirreffs, 2015; Maughan & Gleeson, 1988; Nindl et al., 2002). In contrast, studies that have examined prolonged intermittent fasting (10–56 days) reported no change in exercise performance (Garthe, Raastad, Reifsnes, Koivisto, & Sundgot-Borgen, 2011; Marttinen, Judelson, Wiersma, & Coburn, 2011; Mourier et al., 1997). Nevertheless, discrepancies exist in studies involving 7 days of fasting, where performance is either attenuated (Jarvis, McNaughton, Seddon, & Thompson, 2002) or unchanged (Filaire, Maso, Degoutte, Jouanel, & Lac, 2001; McMurray, Proctor, & Wilson, 1991; Umeda et al., 2004). Several factors may have contributed to these inconsistencies, such as the consumption of pre-testing meals, re-feeding prior to testing (Artioli et al., 2010; Mendes et al., 2013), the carbohydrate content of the diet (Filaire et al., 2001; McMurray et al., 1991), the severity of the energy deficit during intermittent fasting (Horswill et al., 1990), or the intensity of the exercise tested (Horswill et al., 1990; Jarvis et al., 2002). However, regardless of these factors, recovery in high-intensity exercise performance involving fasting of more than 7 days could be due to adaptations in fuel utilisation.

Since the practice of intermittent fasting is commonly analysed using data from just two-time points (pre and post) in most of the abovementioned studies, it is reasonable to assume that a better understanding can be gained from repeated data collection at recurrent time points. Hence, the main purpose of this study was to observe the chronological changes throughout 10 days of intermittent fasting to shed some light on the relationship between intermittent fasting, high-intensity exercise performance and fuel adaptation. It was hypothesised that there would be a drop in supramaximal anaerobic power and high-intensity cycling TTE during the beginning stage of intermittent fasting and that this adverse effect would become negligible by Day-10 due to adaptation in fuel utilisation.

**Methods**

**Study design**

This study employed a quantitative experimental approach to observe the consecutive effect of intermittent fasting on 30-s of Wingate anaerobic power and on prolonged high-intensity cycling TTE capability. Participants were randomly allocated to either the intermittent fasting (FAS) or control (CON) group and completed two sets of sessions. Each session consisted of 7 days of dietary standardisation and familiarisation. Collection of baseline data was conducted on Day-0 (the last day of familiarisation). Participants then followed their allocated group treatment plan for the next 10 days. During each session, participants performed an exercise trial every 2 days (i.e. Day-2, -4, -6, -8 and -10). The Wingate test (WT) was performed in one session and the prolonged high-intensity cycling test (HTT) was conducted in the other, with the order allocated in a randomised counterbalanced manner. Each session was separated by a 4-week washout period where participants continued their habitual daily activity and diet. A simplified schematic timeline of the study is shown in Figure 1.

**Participants**

Twenty healthy, non-smoking, active male college students with peak oxygen consumption above
2.8 L min\(^{-1}\) consented to participate in this study. Participants were randomly and evenly divided into two treatment groups: FAS and CON. Those in the FAS group were aged 21 ± 1 years CON group 20 ± 1 years. While height in the FAS group was 173 ± 5 cm, weight 67.26 ± 4.94 kg and \(\dot{V}O_2\)peak was 2.91 ± 0.12 L min\(^{-1}\), in the CON group height was 174 ± 6 cm, weight was 66.71 ± 4.08 kg and \(\dot{V}O_2\)peak was 2.98 ± 0.15 L min\(^{-1}\).

During both sessions, participants were obliged to stay in a residential college to facilitate supervision of their prescribed meals. Medical ethics approval was provided by the University of Malaya Medical Centre ethics committee and participants’ written consent was acquired before the experiment was conducted.

Pre-trial standardisation and familiarisation

On the first visit, the participants’ \(\dot{V}O_2\)peak was determined using an Astrand cycle ergometer test protocol until volitional fatigue (Monark 928E Pro \(\dot{V}O_2\) Astrand Testing Bike, Sweden). This graded exercise test started with participants performing a 5-min warm-up at 50 W and a cadence of 60 r min\(^{-1}\). A workload of 100 W was then applied, which was increased by 50 W every 2 min until each participant reached volitional exhaustion. Expired breath was measured continuously using a metabolic chart (COSMED, Quark CPET, Italy). The participants’ \(\dot{V}O_2\)peak was defined as the highest oxygen uptake obtained during the last 30-s interval during the test.

At least 1 week before each experimental trial, participants began a period of dietary standardisation and exercise familiarisation. During this standardisation period, daily dietary intake was computed using the Harris-Benedict equation approximated to participants’ estimated daily requirements (FAS = 2500 ± 143 kcal day\(^{-1}\); CON = 2492 ± 120 kcal day\(^{-1}\)). The prescribed diet with a macronutrient ratio of 53% carbohydrate:19% protein:28% fat in every daily meal was calculated using specialist software (Nutritionist Pro, Diet Analyses Software, USA) and prepared by a researcher (MNN). To maintain the daily hydration status of the participants, water intake was provided at 35 mL kg\(^{-1}\) body mass (2.31 ± 0.25 L) during meals and throughout the day. On the second day of the standardised diet and every 2 days thereafter, participants were familiarised with the two exercise test protocols. The familiarisation was conducted at least three times during the dietary standardisation period until peak anaerobic power and high-intensity TTE reached stability when analysed using the mean plots of each exercise variable (Bland & Altman, 1986).

Experimental trials

The first time point for the collection of data on exercise performance and all the physiological variables was the last day of familiarisation (Day-0). On this day, each group continued their diet as prescribed for pre-trial standardisation and familiarisation. On the following day, they commenced the 10-day experimental trial during which they consumed the diet for their allocated group. Food and water were provided in four (FAS) or five (CON) meals over the day depending on the trial: breakfast (0600), lunch (1200), snack (1700), dinner (2000) and supper (2130) (Table I). In the CON group, the diet was a continuation of their pre-trial standardisation diet. In contrast, in the FAS group, the participants’ daily energy intake was only 60% (1500 ± 55 kcal day\(^{-1}\)) of their estimated energy requirements, with about 40% energy restriction achieved via the omission of lunch. Participants consumed all meals at the allocated times and were prohibited from consuming any food and drink other than that provided. Plain water was provided as a
drink, with the consumption of cocoa, tea, coffee or any caffeine-containing products prohibited during both trials.

The participants arrived at the laboratory at 1500 for all exercise testing sessions, which were undertaken under standard environmental conditions (26 ± 2°C and 75 ± 4% relative humidity). Upon arrival, the participants’ body mass in underwear was measured to the nearest 0.01 kg using an electric weighing scale (InBody370, Body Composition Analyser, Korea). They then provided a urine sample as well as a 3-mL venous blood sample taken from the median cubital vein. Participants then completed the allocated exercise tests, after which a second blood sample was taken.

Wingate test (WT). The anaerobic WT was conducted using a 30-s supramaximal single bout (Monark 894E Ergomedic, Sweden) with participants paddled as many revolutions under load resistance of 0.075 kg from their body weight. Participants completed the test after they had followed a standard stretching and warming-up process of three to four sprint cycles without any load. Verbal encouragement was given to every participant to maintain their pedalling rate throughout the test. Data on absolute peak power (APP) output were collected at the end of the exercise test.

High-intensity cycling test (HIT). Before commencing the prolonged high-intensity test, participants performed a 2-min warm-up at 30% of $\dot{V}O_{2\text{peak}}$ followed by 1 min at 60% of $\dot{V}O_{2\text{peak}}$ on the cycle ergometer. This was immediately followed by the HIT where the timer was started when the participant’s $\dot{V}O_{2\text{peak}}$ reached 90%. Participants were encouraged to cycle at 60 r min$^{-1}$ and the test was terminated if this dropped below 55 r min$^{-1}$. Exhaustion was defined as occurring when the participants could not maintain the workload applied.

Analyses of blood and urine samples

Blood samples were collected in an EDTA vacutainer (BD Vacutainer SST-II Advance, USA) and plasma was separated by centrifugation. The plasma glucose and triglyceride concentrations were determined using an enzymatic colorimetric method (Colorimetric/Fluorometric Assay Kit, Biovision Inc, CA, USA), while the plasma lactate concentration determined using an enzymatic membrane system (YSI 150 Sport, Yellow Spring Instrument, Yellow Spring, OH, USA). The blood samples were analysed in triplicate and the mean value was used for statistical analysis. As for the urine samples, the urine specific gravity (USG) was determined using a handheld refractometer (Atago PAL 10-S, Tokyo, Japan).

Statistical analyses

Data were analysed using SPSS software (version 20; IBM Corp., Armonk, NY, USA). However, first, the Shapiro–Wilk test was carried out to determine the
normality of the sample, the result of which showed that all the data were normally distributed. A t-test was used to compare each group’s body mass and USG at baseline (Day-0). For the analysis of the interaction for each group’s body mass, USG, WT and HIT performances as well metabolite variables in each experimental session, a mixed factor repeated measures a $3 \times 6$ (group × time) ANOVA was used to identify the significant changes of each day compared to the baseline (Day-0). The Bonferroni posthoc test was used to identify the pairwise difference. All statistical significances were set at $p < .05$ and an absolute standardised effect size (ES) was included to support important findings. An ES of 0.2 was considered to denote a minimum difference in all outcome measures with 0.5 being moderate and 0.8, large (Cohen, 1988).

Results

Baseline measurement

The baseline (Day-0) measurement of body mass for the Wingate session [FAS: $67.46 \pm 4.51$ kg; CON: $66.84 \pm 5.15$ kg; $p = .812$] and high-intensity TTE cycling session [FAS: $67.51 \pm 3.47$ kg; CON: $66.64 \pm 6.43$ kg; $p = .792$] showed no difference between groups. There was no difference between groups in the USG in the Wingate session ($p = .103$) and high-intensity TTE cycling session ($p = .253$), which indicates that participants were in a well-hydrated state during the sessions. Exercise performance for both WT ($p = .721$) and HIT ($p = .251$) also showed no significant difference between groups.

Wingate session

There were no time, group or interaction effects ($p > .05$) for body mass. In general, body mass gradually reduced in the FAS group on Day-6, -8 and -10, compared to the baseline ($p < .001$) with an overall percentage reduction of $-1.71\%$ (Day-0: $67.46 \pm 4.51$ kg; Day-10: $66.31 \pm 4.43$ kg). However, the CON group showed no difference in body mass throughout the session ($p > .05$) (Figure 2). There was a time effect in the FAS group’s USG where significant increases were observed on Day-2, -4, -6, -8 and -10 compared to the baseline ($p < .001$).

![Figure 2](image-url)

Figure 2. The top two (A and B) indicate changes in body weight during Wingate anaerobic and prolonged high-intensity tests. The bottom two (C and D) indicate the urine specific gravity during the Wingate session and prolonged high-intensity session. The values shown are $M \pm SD$. (*) denotes significant different from Day-0 ($p < .05$). The circle (○) represents CON and the square (□) represents FAS.
As shown in Table II, for WT performance no interaction effect \( (p = .270) \) was observed, but a time effect \( (p = .003) \) as well as a tendency for a group effect \( (p = .057) \) were noted. Compared to the baseline, performance in the FAS group was significantly reduced on Day-2 by 3.05\% with a minimum ES \( (p = .05, ES = 0.4) \). However, performance recovered from Day-4 onwards, with no differences observed compared to the baseline, while the ES became smaller from that time point onwards \( (p > .05, ES = 0.1) \). No difference in WT performance was observed in the CON group \( (p > .05) \).

There were time \( (p = .001) \) and group effects \( (p = .033) \) but no interaction effect \( (p = .996) \) in glucose concentration pre WT (Figure 3). However, post-WT, the glucose concentration showed no time, group or interaction effects \( (p > .05) \). Post-WT, the glucose concentration in the FAS group increased on Day-4 to Day-10 compared to the baseline \( (p < .05) \). There were no time, group or interaction effects \( (p > .05) \) in the pre- and post-WT lactate concentrations for both groups. On the other hand, there were significant time \( (p = .023) \), group \( (p = .022) \) and interaction \( (p < .0001) \) effects for post-WT triglyceride concentrations in the FAS group. Plasma triglycerides showed a gradual reduction from Day-4 onwards compared to Day-0 \( (p < .01) \).

**High-intensity cycling time-to-exhaustion session**

For body mass over the 10 days of the HIT session, interaction and time effects \( (p < .001) \) were observed, but there was no group effect \( (p = .987) \). Generally,
body mass reduced in the FAS group on Day-4, -6, -8 and -10, compared to the baseline \( (p < .001) \) with an overall percentage reduction of \(-1.55\%\) (Day-0: 67.51 ± 3.47 kg; Day-10: 66.46 ± 4.31 kg). Body mass in the CON group was unchanged throughout the session \( (p > .05) \).

There were time \( (p < .0001) \), group \( (p = .004) \) and main interaction \( (p < .0001) \) effects for TTE, as shown in Table II. Cycling TTE in the FAS group was noted to reduce on Day-2 and throughout the experimental session compared to the baseline. However, there was a trend of recovery during the later phase of this session. Compared to Day-0, TTE in the FAS group reduced on Day-2 \( (p < .0001, ES = 0.8) \), Day-4 \( (p < .001, ES = 0.7) \), Day-6 \( (p < .001, ES = 0.7) \), Day-8 \( (p < .05, ES = 0.5) \) and Day-10 \( (p < .05, ES = 0.5) \).

There were time \( (p = .19) \) and group \( (p = .005) \) but no interaction \( (p = .866) \) effects in glucose concentration during pre-HIT. However, post-HIT, the glucose concentration showed a time effect \( (p < .001) \) with no group \( (p = .651) \) or interaction effects \( (p = .800) \). The post-HIT glucose concentration was higher in the FAS group on Day-4, -6 and -8 compared to the baseline \( (p < .05) \). There were no time, group or interaction effects \( (p > .05) \) for pre- and post-HIT lactate concentrations. As for the post-HIT triglyceride concentration, there were time \( (p < .0001) \), group \( (p = .004) \) and main interaction \( (p < .0001) \) effects where the FAS group showed a gradual reduction from Day-4 onwards \( (p < .001) \) compared to the baseline.

**Discussion**

To the best of our knowledge, this study is the first to report changes in Wingate anaerobic power and high-intensity cycling TTE during 10 days of intermittent fasting. This study made two major discoveries. First, intermittent fasting reduces Wingate performance during the early phase of restriction (Day-2) but performance returns to that seen at baseline from Day-4 onwards. Second, in prolonged high-intensity cycling, TTE is reduced throughout the 10-day period, but a trend of recovery is observed towards the end of the experiment. Based on these findings, it seems that performance recuperation starts to take place on the fourth day, which also suggests that there is a transition between the acute and prolonged practice of intermittent fasting.

The detrimental effect of intermittent fasting on anaerobic performance in the first 2 days is consistent with the study by Rankin et al. (1996), which reported a reduction in anaerobic arm ergometer cycling performance, following acute practice (3 days) of moderate caloric deprivation (≈30%). This attenuation in anaerobic performance could be linked to a reduction in carbohydrate intake (Fogelholm, Koskinen, Laakso, Rankinen, & Ruokonen, 1993; McMurray et al., 1991; Rankin et al., 1996). Fasting and a reduction in caloric consumption prior to the first Wingate exercise test in present study certainly led to a depletion in hepatic glycogen, and to a lesser extent a reduced muscle glycogen level (Dohm, Beeker, Israel, & Tapscott, 1986). These changes in glycogen could also be artefacts of the reduction in total energy intake. However, glycogen utilisation cannot be solely attributed since the nature of the exercise itself is rather brief to trigger changes in performance. Another mechanism that may plausibly be involved in reduced performance is dehydration. Our earlier study found that dehydration at 3% can induce fatigue in a 30-s Wingate cycling test (Naharudin & Yusof, 2013). In this study, the USG level was higher before the exercise in the FAS group compared to the baseline, indicating dehydration. Our findings support those of an earlier study by Webster et al. (1990), which found that dehydration caused a drop in anaerobic power due to cardiovascular strain and an increase in lactate concentration.

However, the reason for performance recuperation from Day-4 onwards is as yet unclear. Although our suggestion is somewhat speculative, it is possible that recuperation could be due to participants’ expectation of what it feels like performing exercise under conditions that are sub-caloric and dehydration after the first test on Day-2. The rate of gluconeogenesis via fat utilisation will eventually increase after few days of caloric deprivation and therefore lower the rate of muscle glycogen depletion (Dohm et al., 1986). This reasoning is supported by our data which revealed that there was a significantly higher plasma glucose level after the WT on Day-4–Day-10, but not on Day-2. This finding is in line with previous studies that found no change in anaerobic performance under moderate caloric deprivation (Artioli et al., 2010; Ferguson et al., 2009; Garthe et al., 2011; Marttinen et al., 2011; Mourier et al., 1997) for more than 7 days.

As evidenced by this study, post-exercise plasma glucose was elevated in both groups to replenish the amount utilised during the high-intensity exercise. It is a well-known fact that a brief high-intensity anaerobic exercise that relies mainly on endogenous carbohydrate stores as a fuel source can produce an increase in plasma glucose within a few minutes. This mechanism was reviewed by Adams (2013), who suggested that, during high-intensity exercise (> 80% of VO\(_{2\text{max}}\)), hormones such as catecholamine
noticeably increase, causing the production of glucose to rise seven- to eightfold while its utilisation is only increased three- to fourfold. Moreover, the rise in plasma glucose following such exercise is not influenced by moderate negative energy balance and hypohydration (Ferguson et al., 2009). Thus, it appears that brief, high-intensity exercise (<30-s) increases the plasma glucose level regardless of the participant’s nutritional intake. Interestingly, a rise in plasma glucose was observed from Day-4 onwards compared to baseline in the FAS group. An adaptation in fuel mobilisation as a consequence of the effects of intermittent fasting in compensating for the exercise demand by way of the additional pathway of gluconeogenesis occurring after at least 4 days of intermittent fasting is probably the reason for this increase in post-anaerobic exercise plasma glucose (Field, 1989).

In the prolonged high-intensity exercise, participants’ cycling TTE dropped throughout the 10 days of intermittent fasting. This suggests that depriving energy intake by up to 40% during intermittent fasting influences exercise performance. Since this mode of exercise is mainly dependent on anaerobic glycolysis and glycogenolysis, the reduction in TTE in the FAS group is likely due to a drop in glycogen availability. This will slow down the muscle’s energy filling rate during highly glycogen-demanding exercises causing a failure in maintaining the cycle cadence. Another factor associated with premature muscular fatigue is the higher lactate accumulation after performing moderate-intensity exercise following 23 h of fasting (Dohm et al., 1986). As cycling was continuously performed until exhaustion, the accumulation of H+ and lactate as by-products of anaerobic glycolysis result in a drop in blood pH (Medbo, 1993). The increased acidity impairs the enzyme activity involved in energy metabolism and reduces maximal muscle fibre recruitment (Allen, Lamb, & Westerblad, 2008). Under similar caloric conditions, TTE has also been reported to be negatively affected by prolonged high-intensity exercise in both acute (1–3 days) (Dolan, Cullen, McGoldrick, & Warrington, 2013; Oliver, Laing, Wilson, Bilzon, & Walsh, 2007) and longer (7 days) periods of caloric deprivation (Jarvis et al., 2002; Symons & Jacobs, 1989). On the other hand, according to McMurray et al. (1991), TTE may decrease during intensive exercise of more than 70% of VO2max, unless adequate carbohydrate.

However, a progressive trend of improvement in TTE throughout the experiment was noted, although it remained lower than at baseline. This, again, could be due to fuel adaptation under intermittent fasting where gluconeogenesis prevents a drop in muscle and hepatic glycogen. Therefore, if a longer period of intermittent fasting were to be carried out, it is likely that the high-intensity exercise TTE would recover. This notion is in line with the study by Ferguson et al. (2009), which showed that, after 3 weeks of fasting, aerobic performance was not affected due to an improvement in the power to weight ratio at 90–100% of VO2max.

In this study, plasma glucose increased from Day-4 onwards in the FAS group, which might be related to induce hypohydration. According to Judelson et al. (2008), hypohydration of 5% is shown to increase stress hormone response (cortisol, epinephrine and norepinephrine). Although the authors implemented active dehydration by exposing participants to a 36–37°C environment, resting in room temperature for more than 10 h is believed to have eliminated the confounding factors associated with heat. Thus, metabolic and hormonal responses are believed to be solely dependent on hypohydration status and this reasoning can be extended to this study. In brief, in the long-term practice of intermittent fasting, while fluid is adequately consumed, the metabolic adjustment will take place on Day-2. However, it will return to the baseline after 10 days.

The progressive reduction in body mass observed in both experimental groups was as predicted and in line with published data (Degoutte et al., 2006; McMurray et al., 1991; Mourier et al., 1997). Negative energy balance and exercise demand are the main reasons implicated in the reduction in body mass. As the energy intake is reduced, the utilisation of stored energy in the form of body fats becomes fundamental in sustaining basal metabolic rate (Hansen, Dendale, Berger, van Loon, & Meeusen, 2007). This is seen through the daily energy expenditure and meeting of the energy requirement for the exercise test (Hansen et al., 2007). In the present study, relatively greater changes in body mass were observed during the high-intensity session compared to the Wingate session, which could be attributed to a larger energy requirement for high-intensity exercise. During the period of food deprivation, stored triglyceride in adipose tissue is catabolised by lipolysis to fuel exercise activity (Mendes et al., 2013). Especially under prolonged high-intensity exercise, food deprivation triggers greater mobilisation of liver glycogen, increases in gluconeogenesis and the utilisation of free fatty acids, which may explain the gradual reduction in triglyceride as intermittent fasting progresses (Gleeson et al., 1988).

The findings of this study imply that 10 days of caloric intake deprivation of around 40% during intermittent fasting has variable effects on two modes of high-intensity cycling performance, namely Wingate anaerobic power and prolonged
high-intensity TTE. Anaerobic power was attenuated at the beginning of intermittent fasting and then immediately recovered thereafter. As for prolonged high-intensity cycling, TTE reduced throughout the study period, yet a trend of recovery was noted towards the final day. Under these conditions, in the case of anaerobic performance, participants should be cautious when performing at their maximal capacity and allow at least 4 days for their body to make adjustments. Notably, for prolonged high-intensity performance, a longer period (in excess of 10 days) might be required to eliminate the effect of fasting. The recuperation in performance, evidenced for both modes of exercise, is suggestive of fuel adaptation to meet the exercise demand when participants are under sub-caloric conditions.

As a final note, it should be borne in mind that the participants in this study were active male college students, therefore, caution is advised in not generalising these findings to elite athletes who may have become accustomed to repeated bouts of caloric deprivation and for whom changes in exercise performance may therefore be negligible.

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Disclosure statement
No potential conflict of interest was reported by the authors.

References


