Effects of 8 wk of 16:8 Time-restricted Eating in Male Middle- and Long-Distance Runners

AIDAN J. BRADY[1], HENRY M. LANGTON[1], MOLLIE MULLIGAN[1,2], and BRENDAN EGAN[1,3,4]

[1]School of Health and Human Performance, Dublin City University, Dublin, IRELAND; [2]School of Agriculture and Food Science, University College Dublin, Dublin, IRELAND; [3]National Institute for Cellular Biotechnology, Dublin City University, Dublin, IRELAND; and [4]Florida Institute for Human and Machine Cognition, Pensacola, FL

ABSTRACT

BRADY, A. J., H. M. LANGTON, M. MULLIGAN, and B. EGAN. Effects of 8 wk of 16:8 Time-restricted Eating in Male Middle- and Long-Distance Runners. *Med. Sci. Sports Exerc.*, Vol. 53, No. 3, pp. 633–642, 2021. **Purpose:** Eight weeks of time-restricted eating (TRE) in concert with habitual exercise training was investigated for effects on body composition, energy and macronutrient intakes, indices of endurance running performance, and markers of metabolic health in endurance athletes. **Methods:** Male middle- and long-distance runners ($n = 23$) were randomly assigned to TRE ($n = 12$) or habitual dietary intake (CON; $n = 11$). TRE required participants to consume all of their dietary intake within an 8-h eating window (so-called 16:8 TRE), but dietary patterns, food choices, and energy intake were *ad libitum* during this window. Participants continued their habitual training during the intervention period. Participants completed an incremental exercise test before (PRE) and after (POST) the 8-wk intervention for the assessment of blood lactate concentrations, running economy, and maximal oxygen uptake. Fasted blood samples were analyzed for glucose, insulin, and triglyceride concentrations. Dietary intake was assessed at PRE, MID (week 4), and POST using a 4-d semiweighed food diary. **Results:** Seventeen participants (TRE, $n = 10$; CON, $n = 7$) completed the intervention. Training load did not differ between groups for the duration of the intervention period. TRE resulted in a reduction in body mass (mean difference of −1.92 kg, 95% confidence interval = −3.52 to −0.32, $P = 0.022$). Self-reported daily energy intake was lower in TRE at MID and POST (group–time interaction, $P = 0.049$). No effect of TRE was observed for oxygen consumption, respiratory exchange ratio, running economy, blood lactate concentrations, or heart rate during exercise, nor were there any effects on glucose, insulin, or triglyceride concentrations observed. **Conclusion:** Eight weeks of 16:8 TRE in middle- and long-distance runners resulted in a decrease in body mass commensurate with a reduction in daily energy intake, but it did not alter indices of endurance running performance or metabolic health.
Key Words: BODY COMPOSITION, INTERMITTENT FASTING, LACTATE THRESHOLD, NUTRITION PERIODIZATION, RUNNING, TRAINING LOAD

Athletes and practitioners often pursue novel strategies regarding exercise training, nutrition, and recovery with the aim of enhancing performance in competition. Nutrition periodization is a strategy that is of heightened interest in the last decade, with the manipulation of energy and macronutrient intakes suggested to modulate the adaptive response to exercise training and consequently improve performance outcomes (1). Intermittent fasting (IF) is one aspect of nutrition periodization that is receiving increased scrutiny given its reported benefits on body composition and markers of cardiometabolic health, independent of intentional caloric restriction (2–4). IF involves abstaining from food for a sustained period, longer than the traditional overnight fast, with *ad libitum* food intake only permitted during a narrow eating window. IF includes a range of approaches such as prolonged or alternate day fasting (ADF) and time-restricted eating (TRE). TRE is a daily routine with a narrow window of eating (e.g., 4 to 8 h), usually with *ad libitum* food and calorie intake in that window, and with fasting being undertaken for the remainder (e.g., 16 to 20 h) of the 24-h cycle. A form of TRE known as "16:8," i.e., 16 h of fasting with an 8-h eating window each day, is an increasingly common approach to IF (5–9).

To date, investigations in active adults undertaking TRE have focused primarily on changes in body composition and muscle strength during resistance exercise training (7–10). In trained males performing three resistance training sessions per week, 8 wk of 16:8 TRE resulted in a decrease in fat mass compared with a control group, with no difference between groups for changes in maximal strength or muscle cross-sectional area of the upper arm and thigh (7). In active females, similar improvements in skeletal muscle hypertrophy and muscular strength occurred in response to 8 wk of supervised resistance training, whether participants consumed a control diet or engaged in 16:8 TRE (8).

Address for correspondence: Brendan Egan, Ph.D., School of Health and Human Performance, Dublin City University Glasnevin, Dublin 9, Ireland; E-mail: brendan.egan@dcu.ie.
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To our knowledge, no randomized controlled trial has investigated the effect of a period of TRE on body composition and/or performance indices in athletic populations undertaking aerobic exercise training. However, IF has been examined in the context of aerobic exercise training in rodents (11,12). In one study of mice subjected to 4 wk of IF in the form of ADF and treadmill training, an increase in endurance capacity, an upregulation of molecular pathways involved in mitochondrial biogenesis, and a shift toward greater contribution of fat to energy expenditure was observed (11). If such effects were reproduced in humans, IF may be a promising nutrition strategy to augment the adaptive response to exercise training (4), as demonstrated by other forms of nutrition periodization such as those that manipulate carbohydrate availability before, during and after training (1). However, differences in the effects of different IF regimens have been noted in preclinical models, as well as differences between rodents and humans in a variety of responses to fasting and IF (3). Therefore, whether effects on exercise metabolism and aerobic fitness outcomes observed in preclinical rodent models translate into humans is presently unknown.

The aim of the present study was to investigate the effect of 8 wk of 16:8 TRE, when combined with habitual exercise training, on body composition, energy and macronutrient intakes, indices of endurance running performance, and markers of metabolic health in a group of trained male middle- and long-distance runners. Stated as a null hypothesis, we hypothesized that 8 wk of 16:8 TRE would have no effect on either the primary outcome measure, that being body mass, or the aforementioned secondary outcome measures.

METHODS

Participants. Twenty-eight trained male middle- and long-distance runners (mean ± SD; age, 36.4 ± 7.4 yr; range, 20–49 yr; height, 1.79 ± 0.06 m; body mass, 75.6 ± 9.3 kg; body fat, 12.4 ± 4.3 kg; $\dot{V}O_{2peak}$, 57.7 ± 8.5 mL·kg^{-1}·min^{-1}) were recruited through local running clubs, e-mail, and word of mouth. Participants were required to be currently competing in middle- and long-distance running, considered in this study as events equal to 1500 m and above, and self-reported to be currently engaging in a minimum of 5 d of training per week and to have done so for at least the previous 24 months. All participants gave written informed consent to participate after written and verbal explanations of the procedures. Ethical approval (permit no. DCUREC/2019/0029) was obtained from the Dublin City University Research Ethics Committee in accordance with the Declaration of Helsinki.

Experimental design. This study used a randomized, parallel group, PRE–POST experimental design to compare the effects of 8 wk of TRE or habitual dietary intake (control group [CON]) on body composition, energy and macronutrient intakes, indices of endurance running performance, and markers of metabolic health. The study was single blind as the investigators involved in the analysis of the respective parameters at POST were unaware of the assigned intervention group. All participants completed a familiarization day with a battery of assessments used and were assessed with the same battery of assessments at baseline (week 0; PRE) and upon completion of the intervention (week 8; POST). During each visit to the laboratory, participants completed an incremental exercise test to volitional fatigue. Fasted blood samples and measures of body composition were taken during the week of each laboratory visit. Of the n = 28 participants who completed the familiarization day, n = 23 participants (Table 1) met the inclusion criteria and were pair-matched (with n = 1 unmatched) based on age, height, body mass, and maximal oxygen uptake ($\dot{V}O_{2peak}$), with each pair then randomly assigned to TRE or CON (Fig. 1). Randomization was performed using sealed envelopes drawn from an opaque container that contained an equal distribution of TRE and CON envelopes. Once an envelope had been drawn, it was not returned before the subsequent randomization of the next pair of participants. Both groups were instructed to continue with their habitual training routine for the duration of the study. The duration of the eating window and training logs were completed daily, and semiweighed food diaries were completed after 0 (PRE), 4 (MID), and 8 (POST) wk of the intervention.

The primary outcome measure was change in body mass from PRE to POST. Based on the data from Moro et al. (7) who reported changes in body mass in response to 16:8 TRE during resistance exercise training, the required sample size to detect a difference at a type I error rate (α) of 0.05 and a power ($1-\beta$) of 0.8 was n = 11 per group. Secondary outcomes included changes in body composition, energy and macronutrient intakes, indices of endurance running performance assessed by blood lactate concentration, running economy (RE), and $\dot{V}O_{2peak}$ in response to incremental exercise and fasting blood glucose, insulin, and triglyceride concentrations.

Pretrial preparation. All exercise testing was completed between April and September. Testing was performed between 1000 and 2000 h, but each participant completed their respective exercise tests at PRE and POST at the same time

TABLE 1. Participant characteristics of CON and TRE at PRE.

	CON (n = 7)	TRE (n = 10)	P
Anthropometry			
Age (yr)	39.9 ± 3.0	35.9 ± 8.6	0.266
Height (m)	1.81 ± 0.06	1.79 ± 0.05	0.542
Body mass (kg)	73.13 ± 6.06	72.17 ± 6.68	0.767
Fat mass (kg)	10.30 ± 2.67	10.90 ± 4.00	0.735
FFM (kg)	62.86 ± 4.92	61.25 ± 3.52	0.442
Indices of endurance running performance			
$\dot{V}O_{2peak}$ (mL·kg^{-1}·min^{-1})	59.8 ± 5.2	60.4 ± 4.9	0.810
FBLC 2 mM (km·h^{-1})	13.8 ± 1.4	14.3 ± 1.2	0.412
HR at FBLC 2 mM (bpm)	158 ± 13	163 ± 11	0.411
%HR_{max} at FBLC 2 mM	89.7 ± 5.1	88.5 ± 4.7	0.622
$\dot{V}O_2$ at FBLC 2 mM (mL·kg^{-1}·min^{-1})	48.5 ± 3.2	48.8 ± 4.5	0.896
%$\dot{V}O_{2peak}$ at FBLC 2 mM	81.5 ± 6.4	80.8 ± 3.2	0.767
RE at FBLC 2 mM (mL·kg^{-1}·km^{-1})	212.5 ± 14.8	204.7 ± 12.6	0.263
Markers of metabolic health			
Glucose (mM)	4.97 ± 0.36	5.22 ± 0.42	0.219
Insulin (mU·L^{-1})	3.44 ± 0.60	3.55 ± 0.73	0.746
HOMA-IR (AU)	0.76 ± 0.15	0.83 ± 0.22	0.503
Triglycerides (mg·dL^{-1})	69.2 ± 13.7	65.5 ± 20.1	0.686

Data are presented as mean ± SD. Resting blood samples were taken after an 8-h fast.
HOMA-IR, homeostatic model assessment-insulin resistance; $\dot{V}O_2$, oxygen consumption; $\dot{V}O_{2peak}$, peak oxygen uptake.

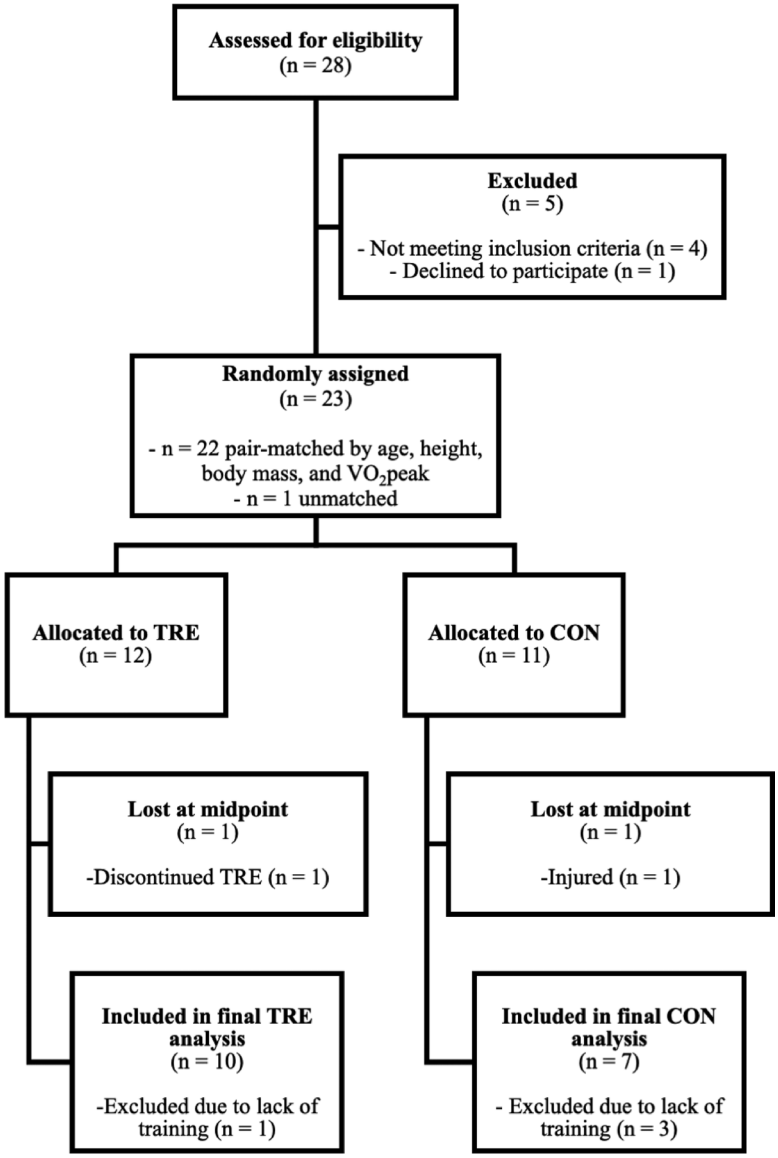

FIGURE 1—CONSORT flow chart. $\dot{V}O_{2peak}$, peak oxygen uptake.

of day ±2 h to minimize the effect of diurnal variations on performance. Participants were asked to abstain from alcohol for 48 h prior and caffeine for 3 h prior, to each test, and to refrain from strenuous exercise training on the day before each test. Participants were prescribed a standardized meal to be consumed ~2 to 3 h before the exercise test, which provided ~510 kcal with a macronutrient composition of 58 g carbohydrate, 47 g protein, and 9 g fat, and was consumed with 500 mL of water. Participants wore the same running shoes for the exercise tests at PRE and POST and were allowed to complete a self-selected stretching routine before being fitted with a heart rate (HR) monitor (H10; Polar, Kempele, Finland) and a face mask (7450 V2 Mask; Hans Rudolph, Inc., KS). The mean ± SD values for ambient temperature, relative humidity, and barometric pressure were 18.6°C ± 0.4°C, 53.6% ± 6.6%, and 763.1 ± 4.3 mm Hg, respectively, at PRE and 18.4°C ± 0.7°C, 54.3% ± 6.3%, and 755.2 ± 3.6 mm Hg, respectively, at POST.

Exercise testing. All exercise tests were conducted on a motorized treadmill (T170; COSMED, Rome, Italy). The exercise test began at a speed of 9 $km \cdot h^{-1}$ and a gradient of 1%, increasing by 1 $km \cdot h^{-1}$ every stage until a participant reached a blood lactate concentration of ≥2 mM, a method used previously in endurance athletes to demarcate lactate threshold and facilitate comparison between athletes (13). Each stage below 2 mM was 6-min in duration to ensure steady-state was achieved (14) and was interspersed with a 30-s rest interval to facilitate the collection of a 15-µL capillary blood sample from the earlobe for determination of blood lactate concentrations (Lactate Pro 2; Arkray, Kyoto, Japan) (15). Rating of perceived exertion (RPE) (Borg 6–20 scale) and HR were recorded during the last 30 s of each stage. Once participants met this criteria of ≥2 mM blood lactate concentration, subsequent stages were reduced to 1 min in duration and increased by 1 $km \cdot h^{-1}$ every minute up to 16 $km \cdot h^{-1}$, after which treadmill gradient was increased by 1% until volitional

fatigue. Expired air was collected and analyzed throughout the test using an automated breath-by-breath (BxB) system (Quark RMR, COSMED), which was calibrated before each test according to manufacturer recommendations. Oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), RER, and RE were calculated from a 60-s average of BxB measurements during the last 90 s of each stage below blood lactate ≥2 mM. RE is expressed as the volume of oxygen required to run 1 km relative to body mass ($mL \cdot kg^{-1} \cdot km^{-1}$). The speed at a blood lactate concentration of 2 mM (fixed blood lactate concentration [FBLC] 2 mM) was calculated using a polynomial equation. $\dot{V}O_2$ at FBLC 2 mM was calculated using a standard linear equation and used to calculate RE at FBLC 2 mM. $\dot{V}O_{2peak}$ was recorded as the highest 20-s average observed during the test.

Body composition. Stature and body composition were assessed on a separate morning during the week of each laboratory testing session, as these measurements were taken between 0630 and 0930 h in an overnight fasted (~10–12 h) state. Participants also refrained from exercise training on the morning of these visits and were advised to consume 500 mL of water 2 h before the assessment. Compliance with these conditions was confirmed verbally by each participant upon arrival. After voiding of the bladder, body mass was measured to the nearest 0.1 kg using a calibrated digital scale (model 703; SECA, Hamburg, Germany), and height was measured to the nearest 0.1 cm using a portable stadiometer (model 231, SECA). Body composition was assessed using bioelectrical impedance analysis (BIA) (DC-430U Dual Frequency Analyzer; Tanita, Arlington Heights, IL) with fat mass and fat-free mass (FFM) generated from the analysis.

Blood analysis. Fasted venous blood samples were taken by a qualified phlebotomist during the same visit as the body composition assessment. Blood was collected in plastic tubes either silicone coated (4 mL; serum) or containing sodium heparin (2 mL; plasma) (Plus Blood Collection Tubes; Becton Dickinson, Franklin Lakes, NJ) for subsequent analysis of glucose, insulin, and triglyceride concentrations. All collection tubes were prechilled, and blood samples were stored on ice before centrifugation at 3000*g* for 15 min at 4°C, after which aliquots of serum and plasma were separated for storage at −80°C until later analysis. Plasma glucose and serum triglycerides were measured using the RX Daytona™ chemical autoanalyzer and appropriate reagents as per the manufacturer's instructions (Randox Laboratories, Crumlin, UK: assay codes GL3815 and TR3823, respectively). Serum insulin was determined by an enzyme-linked immunosorbent assay according to the manufacturer's instructions (10-1113-01; Mercodia, Uppsala, Sweden). Samples for insulin analysis were run in duplicate across two plates with PRE and POST samples from the same participant analyzed on the same plate. Inter- and intraplate variability was calculated using a 4-well pooled sample. Plates 1 and 2 had a coefficient of variation of 5.9% and 3.4%, respectively.

Dietary intervention. Participants in the CON group were instructed to continue with their habitual dietary patterns for the duration of the intervention. Participants in the TRE group were instructed to consume all of their dietary intake within an 8-h window, typically between 1200 and 2000 h. Minor adjustments to exact start or finish of this window were permitted to allow a meal to be consumed after the final training session each evening, or to facilitate a competitive event, but no permissions were given at any point to extend the 8-h eating window on a given day. Only water was permitted to be consumed outside of the 8-h eating window. No restrictions were placed on the frequency of meals or type of food consumed, and no dietary advice was provided other than the instruction about the 8-h eating window. Therefore, the dietary intake within the daily eating window was considered to be *ad libitum* and without caloric restriction. All participants (both CON and TRE) were required to complete an online diary (Google Sheets) on a daily basis of the times at which they started and finished eating each day, which was frequently examined by an investigator (thereby unblinded, and not involved in the final data analyses) to monitor adherence to TRE. A participant's adherence with the eating window in TRE was required to be ≥80% to be included in the final analysis.

Before each laboratory visit and during week 4 (MID) of the intervention, each participant completed a 4-d semiweighed food diary. Detailed instructions on how to measure and record all food and fluid intake during this period were provided. Participants were asked to weigh each food item, or to provide a household measure equivalent, and were instructed to record each food item immediately after consumption. Food diaries were analyzed using an online nutrition analysis software package (Nutritics Dietary Analysis Software; Nutritics, Dublin, Ireland) to calculate energy and macronutrient intakes averaged over 4 d at each time point.

Training log. Participants were instructed to continue with their habitual training routine for the duration of the intervention. None of the participants reported completing any resistance exercise training sessions during the intervention period, and with the exception of cycling training sessions spread randomly throughout both groups during the intervention period that amounted to less than 10 sessions across the entire cohort, all other training sessions recorded were in the form of running sessions. Quantification of training load was performed using the session RPE method (16,17). Briefly, training load was calculated by multiplying session duration (min) by session RPE (Foster 1–10 scale) and is reported in arbitrary units (AU). A custom online training diary was created by the investigators (Google Sheets) and was provided to each participant to record their daily session(s) duration and session(s) RPE. Diaries were monitored daily to ensure data were being entered correctly and to monitor adherence. Participants were also required to record and provide the activity data from their GPS-enabled smart watches (Garmin, Olathe, KS) to the investigators to verify training sessions were being completed. To be included in the final analysis, a participant was required to have completed a minimum of 28 sessions during the intervention period.

Statistical analysis. Graphical representation of data was performed using GraphPad Prism version 8.4 (GraphPad Software

Inc., San Diego, CA). Data are presented as mean ± SD unless otherwise stated. Participant characteristics were compared between groups at PRE using an independent-samples *t*-test. Univariate ANCOVA, with values at PRE for each respective outcome parameter as the covariate, was used to investigate differences between groups at POST for all primary and secondary outcome parameters, except for training parameters and dietary intake. ANCOVA and independent-samples *t*-tests were performed using the Statistical Package for the Social Sciences (version 25; IBM, Chicago, IL). Estimates of effect size from the ANCOVA were determined using partial eta squared (η_p^2) with thresholds of ≥0.0099, ≥0.0588, and ≥0.1379 interpreted as small, moderate, and large effects, respectively, as recommended by Cohen (18) and discussed elsewhere (19). A two-way (group × time) mixed ANOVA was performed in GraphPad Prism and used to determine differences in energy and macronutrient intakes (time levels: PRE, MID, and POST), training load, session duration, session RPE, and session count during the intervention (time levels: weeks 1 through 8). When a main effect of group or a group–time interaction effect was indicated, *post hoc* testing was performed with Tukey's correction, and multiplicity-adjusted *P* values are reported for the comparison of TRE to CON at the respective time points. For null hypothesis statistical testing, the significance level was set at $\alpha \leq 0.05$ for all tests.

RESULTS

Participants. Of the 23 participants who met the inclusion criteria and were randomized at PRE, 17 (TRE, $n = 10$; CON, $n = 7$) were included in the final analysis (Fig. 1). Four participants failed to complete the minimum amount of training sessions, one participant was injured, and one participant did not respond to researchers at MID for food diary analysis. No difference in baseline characteristics were observed between groups (Table 1). Average compliance with the 8-h eating window in TRE was 94.5% ± 5.1%. The average duration of the daily eating window was 7.9 ± 0.2 and 12.9 ± 1.0 h ($P < 0.001$) in TRE and CON, respectively.

Training load. The average weekly training load reported was 1614 ± 410 AU and 1718 ± 476 AU in TRE and CON, respectively. The average number of sessions completed was 42 ± 12 and 46 ± 12 for TRE and CON, respectively. No main effect of group, time, or group–time interaction effects were observed for training load, session count, or session RPE (Table 2). A main effect of time was observed for session duration ($P = 0.017$), with *post hoc* pairwise comparisons revealing that week 7 was higher than weeks 1, 2, 3, and 4 (all $P < 0.05$) in TRE. No main effect of group or group–time interaction effect for session duration was observed (Table 2).

Dietary intake. A group–time interaction effect was observed for energy intake ($P = 0.049$) (Fig. 2A) with no main effects of group or time observed. The average daily energy intake in TRE decreased by 5.2 ± 3.6 kcal·kg^{-1}·d^{-1} between PRE and MID ($P = 0.040$), which was equivalent to 394 ± 228 kcal, but despite being directionally lower by 3.2 ± 8.5 kcal·kg^{-1}·d^{-1} between PRE and POST, which was equivalent to 265 ± 606 kcal, this difference was not statistically significant ($P = 0.413$). A main effect of group was observed for carbohydrate intake ($P = 0.027$) (Fig. 2B). Carbohydrate intake was significantly higher at PRE in TRE (4.4 ± 1.2 g·kg^{-1}·d^{-1}) compared with CON (2.7 ± 0.7 g·kg^{-1}·d^{-1}, $P = 0.004$). A trend in the interaction effect for CHO intake was observed ($P = 0.086$) with carbohydrate intake increasing at MID and POST compared with PRE in CON, but decreasing at MID and POST compared with PRE in TRE. A trend in the interaction effect for fat was also observed ($P = 0.091$), but no main effect of group ($P = 0.673$) or time ($P = 0.608$) (Fig. 2D). No main effect of group or time or group–time interaction was observed for protein intake (Fig. 2C).

Body composition. After adjustment for values in the respective parameters at PRE, body mass in TRE was significantly lower at POST compared with CON (mean difference of −1.92, 95% confidence interval [CI] = −3.52 to −0.32 kg, $P = 0.022$, $\eta_p^2 = 0.321$) (Fig. 3). Fat mass (mean difference of −0.51, 95% CI = −1.21 to 0.19 kg, $P = 0.139$, $\eta_p^2 = 0.150$) and FFM (mean difference of −1.15, 95% CI = −2.46 to 0.16 kg, $P = 0.081$, $\eta_p^2 = 0.202$) were not significantly different between groups.

Indices of endurance running performance. After adjustment for values in the respective parameters at PRE, no difference in $\dot{V}O_{2peak}$ (mean difference of −0.9, 95% CI = −3.8 to 2.0 mL·kg^{-1}·min^{-1}, $P = 0.505$, $\eta_p^2 = 0.032$), FBLC 2 mM (mean difference of 0.4, 95% CI = −0.6 to 1.5 km·h^{-1}, $P = 0.355$, $\eta_p^2 = 0.061$), HR at FBLC 2 mM (mean difference of 4.1, 95% CI = −2.3 to 10.6 bpm, $P = 0.193$, $\eta_p^2 = 0.118$), %HR_{max} at FBLC 2 mM (mean difference of 1.5, 95% CI = −1.9% to 5.0%, $P = 0.355$, $\eta_p^2 = 0.061$), $\dot{V}O_2$

TABLE 2. Overview of training sessions performed during the 8-wk intervention period.

	Group	Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	*P* (Time)	*P* (Group)	*P* (Interaction)
Session count (*n*)	CON	5.9 ± 0.9	6.1 ± 1.3	6.0 ± 1.0	6.7 ± 1.3	5.7 ± 1.0	5.1 ± 2.7	5.6 ± 2.6	6.4 ± 1.9	0.256	0.473	0.463
	TRE	6.0 ± 2.9	5.9 ± 2.3	5.5 ± 1.7	5.3 ± 1.5	5.0 ± 1.9	5.1 ± 1.9	5.0 ± 1.6	4.9 ± 2.0			
Session duration (min)	CON	64 ± 11	69 ± 21	62 ± 13	66 ± 10	68 ± 12	64 ± 20	72 ± 21	71 ± 12	0.017*	0.348	0.334
	TRE	55 ± 17	58 ± 19	51 ± 16	58 ± 22	62 ± 21	62 ± 20	82 ± 21*	63 ± 16			
Session RPE	CON	4.1 ± 1.3	3.7 ± 1.0	4.5 ± 1.8	4.4 ± 1.2	4.7 ± 0.7	4.4 ± 0.8	4.5 ± 1.4	4.6 ± 1.2	0.527	0.077	0.566
	TRE	5.1 ± 1.6	5.4 ± 1.1	5.4 ± 1.4	5.0 ± 1.4	5.6 ± 1.1	5.2 ± 1.4	5.1 ± 1.7	5.2 ± 1.3			
Training Load (AU)	CON	1661 ± 550	1574 ± 571	1687 ± 717	1934 ± 497	1790 ± 405	1536 ± 840	1566 ± 506	2056 ± 650	0.882	0.410	0.358
	TRE	1551 ± 686	1700 ± 498	1442 ± 662	1637 ± 794	1665 ± 1020	1611 ± 744	1852 ± 779	1481 ± 587			

Data are presented as mean ± SD.
* $P < 0.05$ vs weeks 1, 2, 3, and 4.

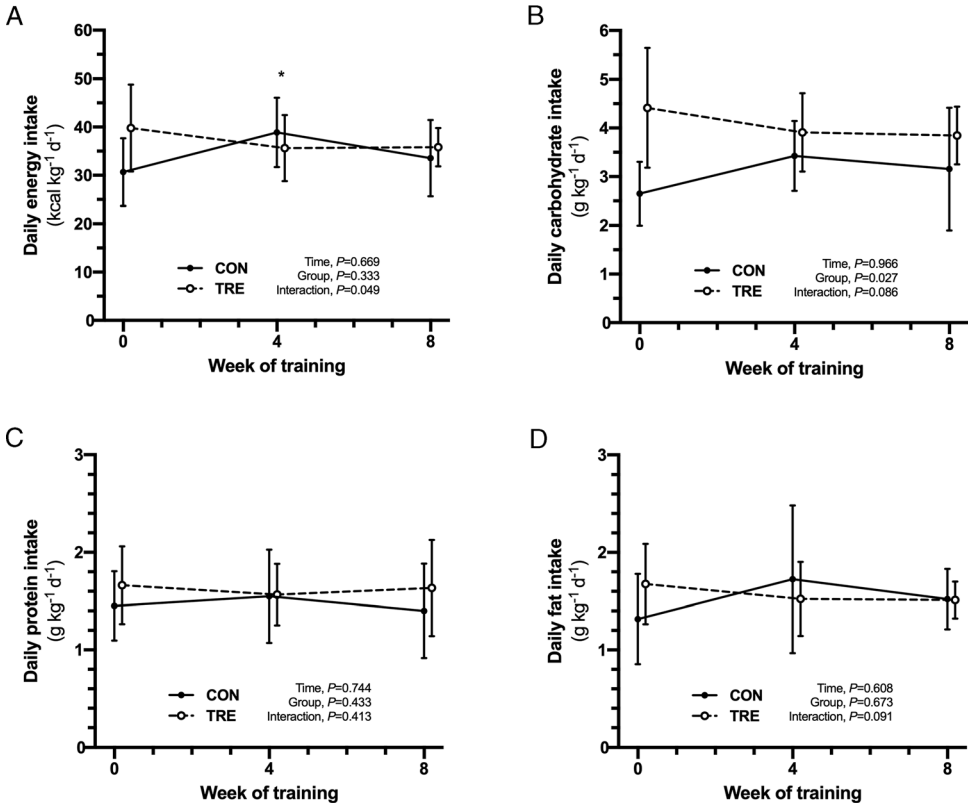

FIGURE 2—Energy (A), carbohydrate (B), protein (C), and fat (D) intake at PRE (week 0), MID (week 4), and POST (week 8) intervention. Data are presented as mean values with error bars representing SD. *$P < 0.05$ compared with PRE within TRE.

at FBLC 2 mM (mean difference of 0.6, 95% CI = −2.7 to 3.9 mL·kg^{-1}·min^{-1}, $P = 0.713$, $\eta_p^2 = 0.010$), %$\dot{V}O_{2peak}$ at FBLC 2 mM (mean difference of 2.1, 95% CI = −2.5% to 6.8%; $P = 0.337$, $\eta_p^2 = 0.066$), and RE at FBLC 2 mM (mean difference of −3.6, 95% CI = −10.1 to 2.8 mL·kg^{-1}·km^{-1}, $P = 0.245$, $\eta_p^2 = 0.095$) were observed between TRE and CON at POST (Table 3). No differences between groups were observed in relative $\dot{V}O_2$, %$\dot{V}O_{2peak}$, blood lactate concentrations, RE, RER, HR, or %HR_{max} at any stage below FBLC 2 mM.

Markers of metabolic health. After adjustment for values in the respective parameters at PRE, blood analysis showed no differences between TRE and CON for fasting glucose (mean difference of 0.22, 95% CI = −0.22 to 0.65 mM, $P = 0.307$, $\eta_p^2 = 0.074$), fasting insulin (mean difference of 0.32, 95% CI = −0.41 to 1.1 mU·L^{-1}, $P = 0.359$, $\eta_p^2 = 0.060$), or fasting

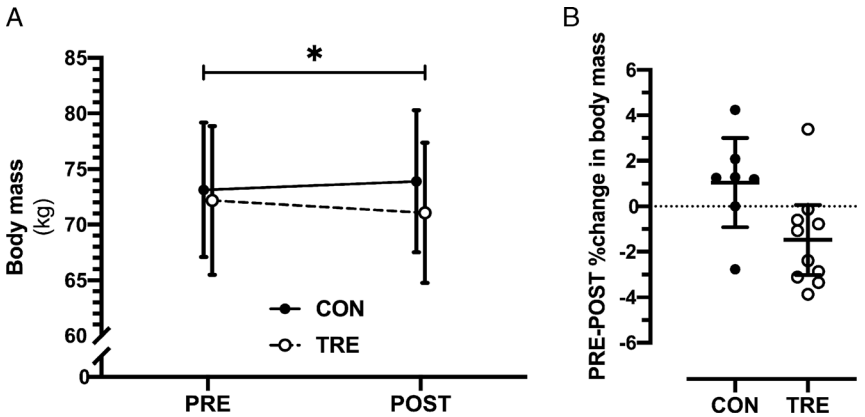

FIGURE 3—Body mass at PRE and POST (A), and percentage change in body mass between PRE and POST (B) for each group. Data are presented as mean values with error bars representing SD (A) or 95% confidence intervals (B). *Circles* represent individual data points from the respective groups. *$P < 0.05$ for TRE vs CON from ANCOVA.

TABLE 3. Change in measures of anthropometry, running performance and metabolic health after an 8-wk TRE intervention, with these POST data presented as unadjusted mean ± SD and ANCOVA-adjusted mean ± SE.

	Unadjusted		Adjusted		
	CON	TRE	CON	TRE	*P*
Anthropometry					
Body mass (kg)	73.90 ± 6.40	71.06 ± 6.29	73.36 ± 0.57	71.44 ± 0.48	0.022*
Fat mass (kg)	10.24 ± 2.52	10.23 ± 3.30	10.54 ± 0.25	10.02 ± 0.21	0.139
FFM (kg)	63.64 ± 5.50	60.81 ± 3.65	62.65 ± 0.47	61.50 ± 0.39	0.081
Indices of endurance running performance					
$\dot{V}O_{2peak}$ (mL·kg^{-1}·min^{-1})	59.6 ± 5.6	59.2 ± 4.6	59.9 ± 1.0	59.0 ± 0.9	0.505
FBLC 2 mM (km·h^{-1})	13.4 ± 1.6	14.2 ± 0.9	13.6 ± 0.4	14.0 ± 0.3	0.355
HR at FBLC 2 mM (bpm)	153 ± 17	162 ± 11	156 ± 2	160 ± 2	0.193
%HR_{max} at FBLC 2 mM	87.2 ± 4.9	88.0 ± 3.7	86.8 ± 1.2	88.3 ± 1.0	0.355
$\dot{V}O_2$ at FBLC 2 mM (mL·kg^{-1}·min^{-1})	47.1 ± 4.9	47.9 ± 3.2	47.2 ± 1.2	47.8 ± 1.0	0.713
%$\dot{V}O_{2peak}$ at FBLC 2 mM	79.3 ± 7.1	81.0 ± 3.3	79.0 ± 1.6	81.1 ± 1.4	0.337
RE at FBLC 2 mM (mL·kg^{-1}·km^{-1})	212.0 ± 12.3	203.0 ± 9.9	208.8 ± 2.3	205.2 ± 1.9	0.245
Markers of metabolic health					
Glucose (mM)	5.00 ± 0.35	5.26 ± 0.41	5.03 ± 0.15	5.24 ± 0.13	0.307
Insulin (mU·L^{-1})	3.10 ± 0.49	3.45 ± 0.78	3.11 ± 0.26	3.44 ± 0.22	0.359
HOMA-IR (AU)	0.69 ± 0.14	0.81 ± 0.23	0.70 ± 0.08	0.81 ± 0.07	0.295
Triglycerides (mg·dL^{-1})	76.1 ± 37.3	79.7 ± 48.1	74.2 ± 16.1	81.0 ± 13.5	0.752

Resting blood samples were taken after an 8-h fast.
*$P < 0.05$ based on outcome of ANCOVA of adjusted means.
HOMA-IR, homeostatic model assessment-insulin resistance; $\dot{V}O_2$, oxygen consumption; $\dot{V}O_{2peak}$, peak oxygen uptake.

triglyceride (mean difference of 6.8, 95% CI = −38.5 to 52.1 mg·dL^{-1}, $P = 0.752$, $\eta_p^2 = 0.007$) concentrations, or for HOMA-IR (mean difference of 0.11, 95% CI = −0.11 to 0.33 AU, $P = 0.295$, $\eta_p^2 = 0.078$) (Table 3).

DISCUSSION

The present study investigated the effects of 8 wk of 16:8 TRE, combined with self-selected habitual training, on body composition, energy and macronutrient intakes, indices of endurance running performance, and markers of metabolic health. Compared with habitual dietary intake and exercise training, TRE resulted in a reduction in body mass commensurate with a reduction in energy intake. TRE did not affect exercise training or indices of endurance running performance, nor were any effects on blood markers of metabolic health observed.

IF is of growing interest in athletic populations (4), a fact that may be attributable to proposed benefits in fuel utilization and body composition observed in rodents undertaking IF combined with exercise training (11,12). Although there is an increasing body of evidence demonstrating beneficial effects of IF and aerobic exercise training on blood lipids and other cardiometabolic health markers in overweight and obese populations (2,20), the present study is the first to investigate the effect of a period of IF in the form of 16:8 TRE in an endurance-trained population. Previous studies of TRE in athletic populations have focused primarily on resistance training with mixed effects reported. Trained females who engaged in 8 wk of 16:8 TRE with resistance training demonstrated minor increases in energy intake but similar gains in FFM and an overall reduction in fat mass compared with a control diet (8). In resistance-trained males, after 8 wk of 16:8 TRE, energy intake was unchanged, but body mass (−0.40 ± 1.76 kg) and fat mass (−0.96 ± 1.72 kg) were reduced (7). By contrast, self-reported energy intake was reduced by ~650 kcal on nontraining days (4 d·wk^{-1}) in young recreationally-trained males who utilized an *ad libitum* 4-h eating window (20:4 TRE) compared with unrestricted eating on training days (10). However, despite this reduction in calories on nontraining days, body composition was unchanged after 8 wk of intervention (10). Athletes with a better understanding of nutrition requirements for their mode of exercise make better nutrition decisions (21). Therefore, counseling participants in such studies may alter eating behavior and be a confounder when aiming to isolate the effects of TRE as an intervention. Moreover, the use of nutrition supplements (8) and standardized diets (7), including intentional calorie restriction (9), makes it difficult to assess the true extent of IF on energy intake and body composition in the exercising adults studied to date.

In the present study, no dietary advice other than explanation of the eating window to the TRE group was provided. No restrictions were made on meal frequency or timings with participants simply directed to eat *ad libitum*, be that in terms of calories or types of food, during their respective eating windows. TRE resulted in a reduction in self-reported energy intake of 394 ± 228 kcal·d^{-1} by MID, but despite being directionally lower at POST by 265 ± 606 kcal·d^{-1}, this difference was not statistically significant because of the large interindividual variation. The pattern of reduced energy intake on TRE days after 4 wk, but returning toward PRE intervention values after 8 wk, was a pattern that was apparent in a previous study of active males engaged in TRE while undertaking resistance training (10). Longer-term studies of TRE will be required to investigate whether changes observed after 4 to 8 wk are sustained during longer duration adherence to TRE, or whether compensatory behaviors consistently emerge that result in daily energy intakes that are similar to habitual, pre-TRE intakes. That said, in the present study, these declines in energy intake were largely explained by downward trends in carbohydrate intake over the 8-wk period and fat intake in the first 4 wk of the intervention. Coincident with the decrease in energy intake in TRE, a reduction in body mass of ~1.5% (or ~1.1 kg) was observed from PRE to POST. This loss of body mass cannot definitively be attributed to the effect of TRE on energy intake, given that energy expenditure was not measured as part of the

energy balance equation. However, exercise training load was unchanged throughout the 8-wk period, and participants were asked to maintain all other daily activity habits as normal. Although overall body mass was reduced in TRE, examination of the individual components of fat mass and FFM showed that both were directionally lower, with FFM trending toward the threshold of 0.05 chosen for statistical significance. The small sample size of participants completing the intervention in the present study cannot be disregarded in this light and likely resulted in this analysis being underpowered for null hypothesis statistical testing. However, effect sizes for decline in body mass (η_p^2 = 0.321), fat mass (η_p^2 = 0.150), and FFM (η_p^2 = 0.202) are all interpreted as large effects.

This downward trend in FFM with TRE was observed despite protein intake ranging from 1.4 to 1.7 $g \cdot kg^{-1} \cdot d^{-1}$ at all time points for both groups, in line with the recommended daily intake for endurance athletes (22). Alternatively, the decline in daily carbohydrate intake in TRE may have negatively effected body glycogen stores, which in turn could have reduced total body water given the association of each gram of glycogen with ~3 g of water (23). This change in total body water would have the effect of both reducing overall body mass but also resulting in a lower quantity of FFM when measured by BIA (24). Of course, the use of self-reported food diaries to monitor dietary intake is not without limitation. Issues of underreporting and altered behavior are common, and thus the results from these records should be interpreted accordingly (25). Moreover, the measurement of body composition by BIA is not without limitation because BIA is known to under- or overestimate fat mass and FFM compared with dual-energy x-ray absorptiometry (26). That said, foot-to-foot bioelectrical impedance analyzers similar to that used in the present study demonstrate good reliability for the assessment of body composition in active males, with between-day coefficients of variation of ~2.0%–2.5% reported (27,28). In the present study, the reduction in fat mass in TRE was ~0.67 kg, equivalent to a relative reduction of ~6.2%. By contrast, the reduction in FFM in TRE was only 0.7%. Therefore, changes in FFM lie within the typical error of measurement for BIA and should be interpreted with caution, but the 6.2% reduction in fat mass is likely to be the major component of the observed reduction in body mass. Therefore, with these data and caveats all considered, we contend that 8 wk of 16:8 TRE resulted in practically meaningful changes in body mass and composition, in addition to energy and macronutrient intakes, which warrant further investigation in future studies.

An important finding of the present study is that this period of TRE did not affect a variety of parameters used to monitor exercise training. No differences were observed between groups for training load, duration, intensity, or frequency throughout the study. Although there were inter- and intraindividual variations in the weekly training load, this is reflective of modern training programs, which alternate between periods of increasing volume and intensity with periods of recovery (17). The participants were also directed to continue with their self-selected habitual training routine for the duration of the intervention period. Given the different types of training periodization used by endurance athletes (1), these variations in training load are not unusual. Although the reduction in energy intake did not result in a decline in training performance, it is important to note that the training loads reported in the present study are considerably lower than those of elite endurance athletes (1614 vs ~3350 AU) (17). Higher training loads require a greater energy intake, with particular reliance on carbohydrate for high-intensity and prolonged aerobic exercise (29). The aforementioned changes in self-reported dietary intake in TRE led to a 14% and 11% reduction in carbohydrate intake at MID and POST, respectively. The manipulation of carbohydrate intake is a common nutritional strategy among endurance athletes with the aim of improving metabolic flexibility and augmenting mitochondrial biogenesis but requires careful planning and consideration of the energy requirements for exercise training (30). The observed decline in energy and carbohydrate intake appears to be spontaneous in nature rather than a conscious or intentional reduction and, therefore, if prolonged may lead to a state of low energy availability in the absence of a decline in training load. Persistent low energy availability can lead to a variety of health and performance decrements, such as impairing muscle protein synthesis and blunting the adaptive response to training and increasing the risk of injury and illness (31). In this regard, future investigations should examine the long-term effects of TRE in more elite endurance athlete populations, especially those with greater training loads.

Blood lactate concentrations, RER, and RE (oxygen cost of running at a given velocity) at incremental running speeds were used in the present study as indicators of improved endurance running performance and substrate utilization during submaximal exercise. Along with $\dot{V}O_{2max}$, lactate concentrations and RE are the best indicators of training adaptation and running performance in endurance athletes (32,33). The lack of change in these metrics may be a consequence of an inadequate training stimulus because of the aforementioned unstructured training because significant improvements in these metrics have been observed in trained participants during short-term training programs (34). However, we deliberately chose not to include a structured exercise training intervention in the present study as our aim, *a priori*, was to examine the effects of TRE itself rather than TRE combined with a progressive overload on aerobic fitness and indices of endurance running performance. By contrast, when IF in the form of ADF was combined with intensive exercise training in mice, endurance running capacity was improved, in addition to a range of metabolic changes (11). For example, trained ADF mice demonstrated reductions in RER with elevated concentrations of ketone bodies, indicative of a shift in the contribution to energy provision from substrate utilization of carbohydrate to fat and an improvement in metabolic flexibility. The expression of peroxisome proliferator-activated receptor alpha, an important regulator of genes involved in fatty acid uptake, was increased along with the increased expression of pyruvate dehydrogenase kinase 4, an inhibitor of the pyruvate dehydrogenase kinase complex, thereby potentially contributing to the

reduction in glycolytic flux (11). Reductions in RER, indicative of a reduction in the reliance on carbohydrate to energy provision, do not always translate into improvements in exercise performance (29,35). The increased availability and oxidation of free fatty acids, which is a consequence associated with fasting and low carbohydrate ketogenic diets, is now recognized as having an inhibitory effect on glycolytic flux rather than a "glycogen sparing" effect as previously hypothesized (29). However, in contrast to the results observed in mice undertaking ADF and exercise training, results of the present study do not support these findings, with no changes in RER or RE at any submaximal intensity and no change in lactate concentrations observed. Lastly, no changes in fasting glucose, insulin, or triglyceride concentrations were observed. These findings are in contrast to a previous report in resistance-trained males undertaking 16:8 TRE for 8 wk that observed reductions in glucose, insulin, and triglyceride concentrations in the TRE group (7). The reason for this discrepancy between the studies warrants further investigation, especially given the often-stated cardiometabolic benefits of IF (2). Likely explanations are the greater level of aerobic fitness at baseline in the present study, and the persistent high level of aerobic exercise training throughout the intervention, meaning that the range for improvements in markers of metabolic health by TRE was relatively narrow.

There are several limitations to be considered in the present study. First, sample sizes of 10 and 7 participants in the TRE and CON groups, respectively, is underpowered relative to the *a priori* sample size (n = 11 per group) required. Several notable trends were observed in relation to change in body composition and dietary intakes, but these will require larger sample sizes in future studies to confirm these findings. The present study was designed to investigate the effect of TRE when undertaken in conjunction with self-selected habitual exercise training, and therefore the results cannot be extrapolated to predict how TRE would interact with the adaptive response to a structured exercise training program designed to improve endurance running performance. Although this experimental approach was used to improve the ecological validity of the study, it resulted in four (n = 1 TRE; n = 3 CON) participants being excluded in the POST analysis for a lack of sufficient training, which also contributed to the small n-sizes per group. In addition to providing a structured exercise training program, it would be worthwhile to investigate whether the time of training, and therefore whether training sessions are performed in the fasted or fed state of TRE, would effect the adaptive response under this approach. Finally, the indices of endurance running performance and substrate utilization may not be sensitive enough to detect changes at a whole body level with just 8 wk of intervention. At a molecular level, changes have been observed in previous work in rodents in shorter time frames (11,12), and given the differences between rodents and humans in the response to IF (3), it may be that a longer duration of intervention would be needed to observe changes at a whole body level in humans. Future studies involving tissue samples would be needed to fully elucidate changes, if any, at a molecular level in human skeletal muscle in response to TRE.

In summary, 8 wk of 16:8 TRE in trained middle- and long-distance runners resulted in a spontaneous reduction in self-reported energy intake and a reduction in body mass, but it did not impair exercise training or alter indices of endurance running performance. The reduction in body mass was composed of directional reductions in both fat mass and FFM being large effect sizes, albeit not reaching the threshold for statistical significance, and in the case of FFM may be confounded by the limitations of the assessment of body composition by BIA. These preliminary observations will therefore require further investigation in future studies. The spontaneous reduction in energy intake represents a potential alternative to alter body composition with *ad libitum* food intake within a narrowed eating window, rather than intentional caloric restriction. However, the reductions in carbohydrate intake and FFM as a consequence of TRE require further investigation to explore whether this dietary intervention produces adverse long-term effects on energy availability or training adaptations. In this regard, future work should investigate the effect of TRE using a structured exercise program over a prolonged period and examine the effect of TRE in an elite population with greater training loads.

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The authors declare the results of this unfunded study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation and do not constitute endorsement by the American College of Sports Medicine.

REFERENCES

1. Mujika I, Halson S, Burke LM, Balagué G, Farrow D. An integrated, multifactorial approach to periodization for optimal performance in individual and team sports. *Int J Sports Physiol Perform*. 2018;13(5):538–61.
2. de Cabo R, Mattson MP. Effects of intermittent fasting on health, aging, and disease. *N Engl J Med*. 2019;381(26):2541–51.
3. Lee JH, Verma N, Thakkar N, Yeung C, Sung H-K. Intermittent fasting: physiological implications on outcomes in mice and men. *Physiology (Bethesda)*. 2020;35(3):185–95.
4. Levy E, Chu T. Intermittent fasting and its effects on athletic performance: a review. *Curr Sports Med Rep*. 2019;18(7):266–9.
5. Chow LS, Manoogian ENC, Alvear A, et al. Time-restricted eating effects on body composition and metabolic measures in humans who are overweight: a feasibility study. *Obesity (Silver Spring)*. 2020;28(5):860–9.
6. Gabel K, Hoddy KK, Haggerty N, et al. Effects of 8-hour time restricted feeding on body weight and metabolic disease risk factors in obese adults: a pilot study. *Nutr Healthy Aging*. 2018;4(4):345–53.
7. Moro T, Tinsley G, Bianco A, et al. Effects of eight weeks of time-restricted feeding (16/8) on basal metabolism, maximal strength, body composition, inflammation, and cardiovascular risk factors in resistance-trained males. *J Transl Med*. 2016;14(1):290.

8. Tinsley GM, Moore ML, Graybeal AJ, et al. Time-restricted feeding plus resistance training in active females: a randomized trial. *Am J Clin Nutr*. 2019;110(3):628–40.
9. Stratton MT, Tinsley GM, Alesi MG, et al. Four weeks of time-restricted feeding combined with resistance training does not differentially influence measures of body composition, muscle performance, resting energy expenditure, and blood biomarkers. *Nutrients*. 2020;12(4):1126.
10. Tinsley GM, Forsse JS, Butler NK, et al. Time-restricted feeding in young men performing resistance training: a randomized controlled trial. *Eur J Sport Sci*. 2017;17(2):200–7.
11. Marosi K, Moehl K, Navas-Enamorado I, et al. Metabolic and molecular framework for the enhancement of endurance by intermittent food deprivation. *FASEB J*. 2018;32(7):3844–58.
12. Real-Hohn A, Navegantes C, Ramos K, et al. The synergism of high-intensity intermittent exercise and every-other-day intermittent fasting regimen on energy metabolism adaptations includes hexokinase activity and mitochondrial efficiency. *PLoS One*. 2018;13(12):e0202784.
13. Seiler KS, Kjerland GØ. Quantifying training intensity distribution in elite endurance athletes: is there evidence for an "optimal" distribution? *Scand J Med Sci Sports*. 2006;16(1):49–56.
14. Kuipers H, Rietjens G, Verstappen F, Schoenmakers H, Hofman G. Effects of stage duration in incremental running tests on physiological variables. *Int J Sports Med*. 2003;24(7):486–91.
15. Gullstrand L, Sjüdin B, Svedenhag J. Blood sampling during continuous running and 30-second intervals on a treadmill: effects on the lactate threshold results? *Scand J Med Sci Sports*. 2007;4(4):239–42.
16. Foster C, Florhaug JA, Franklin J, et al. A New Approach to Monitoring Exercise Training. *J Strength Cond Res*. 2001;15(1):109–15.
17. Stellingwerff T. Case study: nutrition and training periodization in three elite marathon runners. *Int J Sport Nutr Exerc Metab*. 2012; 22(5):392–400.
18. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. New York: Academic Press; 1969.
19. Richardson JTE. Eta squared and partial eta squared as measures of effect size in educational research. *Educ Res Rev*. 2011;6(2):135–47.
20. Bhutani S, Klempel MC, Kroeger CM, Trepanowski JF, Varady KA. Alternate day fasting and endurance exercise combine to reduce body weight and favorably alter plasma lipids in obese humans. *Obesity (Silver Spring)*. 2013;21(7):1370–9.
21. Holtzman B, Ackerman K. Measurement, determinants, and implications of energy intake in athletes. *Nutrients*. 2019;11(3):665.
22. Witard OC, Garthe I, Phillips SM. Dietary protein for training adaptation and body composition manipulation in track and field athletes. *Int J Sport Nutr Exerc Metab*. 2019;29(2):165–74.
23. Bergström J, Hultman E. Nutrition for maximal sports performance. *JAMA*. 1972;221(9):999–1006.
24. Kyle U, Bosaeus I, De Lorenzo A, et al. Bioelectrical impedance analysis. Part I: review of principles and methods. *Clin Nutr*. 2004; 23(5):1226–43.
25. Burke LM, Lundy B, Fahrenholtz IL, Melin AK. Pitfalls of conducting and interpreting estimates of energy availability in free-living athletes. *Int J Sport Nutr Exerc Metab*. 2018;28(4):350–63.
26. Achamrah N, Colange G, Delay J, et al. Comparison of body composition assessment by DXA and BIA according to the body mass index: a retrospective study on 3655 measures. Handelsman DJ, editor. *PLoS ONE*. 2018;13(7):e0200465.
27. Dixon CB, LoVallo SJ, Andreacci JL, Goss FL. The effect of acute fluid consumption on measures of impedance and percent body fat using leg-to-leg bioelectrical impedance analysis. *Eur J Clin Nutr*. 2006;60(1):142–6.
28. Swartz AM, Evans MJ, King GA, Thompson DL. Evaluation of a foot-to-foot bioelectrical impedance analyser in highly active, moderately active and less active young men. *Br J Nutr*. 2002;88(2):205–10.
29. Hawley JA, Leckey JJ. Carbohydrate dependence during prolonged, intense endurance exercise. *Sports Med*. 2015;45(1 Suppl):5–12.
30. Stellingwerff T, Morton JP, Burke LM. A framework for periodized nutrition for athletics. *Int J Sport Nutr Exerc Metab*. 2019;29(2):141–51.
31. Mountjoy M, Sundgot-Borgen J, Burke L, et al. International Olympic Committee (IOC) consensus statement on relative energy deficiency in sport (RED-S): 2018 update. *Int J Sport Nutr Exerc Metab*. 2018;28(4):316–31.
32. Joyner MJ, Coyle EF. Endurance exercise performance: the physiology of champions. *J Physiol*. 2008;586(1):35–44.
33. Midgley AW, McNaughton LR, Jones AM. Training to enhance the physiological determinants of long-distance running performance: can valid recommendations be given to runners and coaches based on current scientific knowledge? *Sports Med*. 2007;37(10):857–80.
34. Jones AM, Carter H. The effect of endurance training on parameters of aerobic fitness. *Sports Med*. 2000;29(6):373–86.
35. Burke LM, Hawley JA. Effects of short-term fat adaptation on metabolism and performance of prolonged exercise. *Med Sci Sports Exerc*. 2002;34(9):1492–8.