Attenuated gastric distress but no benefit to performance with adaptation to octanoate-rich esterified oils in well-trained male cyclists

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Thorburn, Megan S., Bodil Vistisen, Rhys M. Thorp, Mike J. Rockell, Asker E. Jeukendrup, Xuebing Xu, and David S. Rowlands. Attenuated gastric distress but no benefit to performance with adaptation to octanoate-rich esterified oils in well-trained male cyclists. J Appl Physiol 101: 1733–1743, 2006.—We investigated the effects of modifying a normal dietary fatty acid composition and ingestion of high-fat exercise supplements on gastrointestinal distress, substrate oxidation, and endurance cycling performance. Nine well-trained male cyclists completed a randomized triple-crossover comprising a 2-wk diet high in octanoate-rich esterified oil (MCFA) or twice long-chain fatty acids (LCFA). Following the diets, participants performed 3 h of cycling at 50% of peak power followed by 10 maximal sprints; while ingesting either 1) a carbohydrate (CHO)+MCFA-rich oil emulsion after the 2-wk MCFA-rich dietary condition (MC-MC, Intervention) and 2) one of the LCFA-rich dietary conditions (LC-MC, Placebo) or 3) CHO only following a LCFA-rich diet (LC-CHO, Control). During the 3-h ride MCFA-adaptation decreased octanoic-acid oxidation by 24% (90% confidence interval: 14–34%). The CHO+MCFA-rich oil emulsion reduced endogenous fat oxidation by 61% (33–89%) and 110% (89–131%) in the MC-MC and LC-MC conditions, respectively, and MCFA-adaptation reduced endogenous-carbohydrate oxidation by 10% (~3–23%). MCFA-adaptation attenuated gastrointestinal distress and nausea during the sprints, but the effect of the oil emulsion was to lower sprint power by 10.9% (7.7–14.1%) in the LC-MC condition and by 7.1% (5.7–8.5%) in the MC-MC condition, relative to the LC-CHO control; every one unit increase in nausea decreased mean power by 6.0 W (3.2–8.8 W). We conclude that despite some attenuation of endogenous-carbohydrate oxidation and gastric distress following adaptation to a MCFA-rich diet, repeat sprint performance was substantially impaired in response to the ingestion of a CHO+MCFA-rich oil emulsion.

medium-chain fatty acids; structured triacylglycerols; dietary adaptation; supplementation

THE FINITE AVAILABILITY of endogenous glycogen as an energy substrate contributes to the development of fatigue and is therefore a limiting factor in athletic performance during prolonged strenuous exercise (23). An increase in plasma free fatty acid (FFA) and fat oxidation has been shown to spare endogenous glycogen, most reliably via fat infusion studies (38). However, fat infusion during racing conditions is not feasible, and long-chain triglyceride ingestion before or during exercise has not been shown to enhance performance (14, 32, 40). More recently, interest has focused on the ingestion of medium-chain triglycerides (MCTs). Unlike long-chain fatty acids, medium-chain fatty acids (MCFA) can be absorbed more rapidly and directly from the intestinal lumen (5, 13), and their entry into mitochondria does not appear to be rate limited by the acyl-carnitine transfer system to the extent of long chain fatty acids during high-intensity exercise (33). Therefore, ingestion of MCFAs during exercise has the potential to enhance performance by elevating plasma FFA concentrations and sparring muscle glycogen (17, 35, 36).

In trials feeding 25–30 g MCT suspensions, the oxidation of MCFA reached 30% compared with 70% oxidation when coingested with carbohydrate (CHO); plasma FFA and ketone concentrations were elevated, but no overall change in total lipid or carbohydrate oxidation was observed (8, 19, 20). In contrast, 86 g MCT coingested with CHO has resulted in elevation of serum FFA concentration, reduced reliance on endogenous carbohydrate, and performance enhancement (2.5% decrease in 40-km time-trial time) relative to CHO-only ingestion with no reports of gastrointestinal distress (35). In contrast, Jeukendrup et al. (22) and Goedecke et al. (11, 12) did not observe performance benefits when feeding up to 85 g of MCT coingested with CHO during endurance and ultra-endurance exercise, which was related to the development of moderate-to-severe gastrointestinal distress. Additionally, no clear differences between fat and CHO oxidation with or without MCT ingestion were observed in these later studies.

Chronic dietary MCT adaptation may be one way to attenuate gastric distress when ingesting MCTs during exercise. Both Misell et al. (26) and Öörik et al. (29) reported decreased gastrointestinal distress symptoms at rest with 1–2 wk of dietary MCT adaptation. Unfortunately, participants did not ingest MCT-rich supplements during exercise in either of these studies, giving no indication whether the dietary adaptation period decreased gastrointestinal distress during exercise (26, 29).

Furthermore, the ingestion of structured triacylglycerols has also been investigated as a method to alleviate the gastrointestinal distress associated with MCT intake. Specifically, structured triacylglycerols containing MCFAs in the sn-1 and sn-3

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MCFA ADAPTATION AND EXERCISE PERFORMANCE

Standardized training in lab
Light training
No training

Positions and a long chain fatty acid in the sn-2 position are well tolerated and rapidly metabolized when ingested at rest (41). Vistisen et al. (36) investigated exercise supplements with structured triacylglycerol-CHO emulsions during endurance exercise. Despite similar gastrointestinal distress ratings between the structured triacylglycerol-CHO and CHO-only supplement conditions during exercise, structured triacylglycerol ingestion had no effect on performance.

Therefore, the aim of this study was to see if MCFA-rich dietary adaptation would alleviate gastrointestinal distress and increase the rate of MCFA oxidation while ingesting a CHO + MCFA-rich oil emulsion during endurance cycling. To do this we altered the fatty-acid composition of a normal mixed diet for 2 wk by supplementation with foods containing randomly esterified MCFA-rich structured oils. A decrease in gastrointestinal distress or increase in MCFA oxidation was anticipated to lead to performance benefits.

MATERIALS AND METHODS

Participants
Nine well-trained male cyclists aged 37 yr (SD 7) and weighing 81 kg (SD 8) completed the study. All cyclists had been training for at least 8–10 h/wk and riding competitively for at least 6 mo. Mean VO2 max and peak power output were 4.8 l/min (SD 0.5) and 357 W (SD 21), respectively. Before beginning experimentation, all participants read the study information sheet, were informed of their rights, screened for precluding health conditions, and signed a consent form. The experimental protocol of this study was approved by the Massey University Ethics Committee (Protocol 03/143).

General Design
Cyclists participated in a randomized double-blind triple-crossover protocol in which three dietary conditions modifying the normal fatty acid composition were completed over three consecutive 2-wk experimental blocks (Fig. 1). The conditions were 1) placebo, 2-wk diet containing long-chain fatty-acid-rich oil with the intervention CHO + MCFA-rich esterified oil emulsion ingested during exercise (LC-MC); 2) Intervention, 2-wk diet containing MCFA-rich esterified oil with the CHO + MCFA-rich oil emulsion ingested during exercise as above (MC-MC); and 3) Control, 2-wk diet containing long-chain fatty-acid rich oil with a CHO-only exercise supplement (LC-CHO). All participants began an individualized and standardized training regimen 2-wk before the first baseline 3-h ride and performance test (Fig. 1). The training was repeated for each subsequent 2-wk dietary condition. The training protocol included three-to-five supervised and workload-standardized lab sessions where they ingested 500 ml of the dietary supplement during exercise. On the morning of each test, participants reported to the lab between 6 and 8 AM in a fasted state, and the parameters of the cycle ergometers were adjusted according to the subject’s own racing cycle angles. Two or three cyclists completed the testing procedure at once and this same cohort completed all subsequent experimental blocks and cycling tests to repeat any effects of personality-induced psycho-stimuli.

Protocols
Maximal oxygen uptake (VO2 max) and peak power output (PPO) were measured using a progressive exercise protocol on an electronically braked cycle ergometer (VeloTron Racer Mate, Seattle, WA). After a warm-up period, the test started at a workload of 3.33 W/kg body mass. The first stage duration was 150 s, after which the load was increased by 50 W, and then by 25 W for every subsequent 150-s stage. Exhaustion was defined as the time at which the participant could no longer maintain a pedal cadence of 50 RPM after three warnings. VO2 max was measured online with a calibrated SensorMedics Vmax Spectra Series gas analyzer (SensorMedics, Yorba Linda, CA) and taken as the highest attained 20-s average oxygen uptake. PPO was defined as the last completed work rate plus the fraction of time spent in the final noncompleted work rate multiplied by the 25 W work rate increase.

Familiarization. Approximately 1-wk following the VO2 max test, a shortened version of the performance test (see below) was performed as a procedural familiarization trial. Participants cycled for 2-h at 50% PPO and performed six maximal sprints interspersed with recovery periods at 40% PPO. An 8% CHO-based solution was ingested every 20-min, and participants practiced recording gastrointestinal distress and ratings of relative perceived exertion.

Baseline test. This test provided information on gastric responses to naive exposure of the CHO + MCFA-rich esterified-oil emulsion supplement (see below), doubling as a full practice trial of the testing...
procedure and a ride to deplete endogenous glycogen stores to lower the background $^{13}$C enrichment. Participants rode for 3 h at 50% PPO, primarily for assessment of physiological responses but also as a long preload/depletion ride prior to the repeated-sprint performance test, which consisted of 10 maximal-effort sprints interspersed with recovery periods as described above.

Immediately before the start of exercise, participants ingested a double quantity of the CHO+MCFA-rich esterified oil supplement. Every 20-min throughout the 3-h steady-state ride, participant gastrointestinal distress, exertion ratings, and heart rate were recorded before ingesting the next supplement, the quantity of which was based on individual PPO calculated from a base reference of 220 ml/20 min for a PPO of 400 W. Participants ingested between 180 and 213 ml of supplement/20 min (360–426 ml double bolus). During the sprints, participants ingested each drink supplement throughout the 20 min as they liked, and reported gastrointestinal distress and exertion immediately after sprints 1, 4, 7, and 10.

$^{13}$C background trial. A 3-h ride at 50% PPO was performed on day 11 of each 2-wk supplementation block (Fig. 1) to determine background breath $^{13}$C-enrichment required for later calculation of octanoic-acid oxidation. During the ride, participants ingested the experimental drink supplement, containing all components except the esterified oil. Every 20 min during the 3-h ride, external respiratory gas was collected for indirect calorimetry (Sensormedics) and $^{13}$C breath enrichment, the latter into 10-ml evacuated rubber-capped test tubes (Labco, High Wycombe, UK) from a 5-liter mixing chamber. For all experiments, the Sensormedics mass flow sensor and gas analyzers were calibrated before testing and every hour during the 3-h exercise test. Following testing, the raw minute volume and gas fractions were adjusted if drift was greater than 3% following each hour of sampling. Drift between the initial and verification calibrations was assumed to be linear and the raw data were adjusted accordingly.

3-h Ride. On day 14 of each 2-wk block, participants repeated the protocol undertaken during the baseline exercise test (Fig. 1). Upon reporting to the lab, participants had a 20-gauge cannula inserted into the antecubital vein of their right forearm (Becton Dickinson Medical). A two-way stopcock valve (Becton Dickinson Medical) was connected to the cannula and a 5-ml blood sample was taken immediately and transferred into vaccutainers containing lithium heparin (Becton Dickinson, Franklin Lakes). Triplicate breath samples were collected from the mixing chamber for resting breath $^{13}$C enrichment. Immediately before exercise began, participants consumed a double bolus of drink supplement. Every 20 min during the 3-h ride data variables were collected in the order of exertion and gastrointestinal distress scales, indirect calorimetry, $^{13}$C breath enrichment, and a blood sample. Immediately following data collection, another bolus of supplement was ingested.

Performance test. After completion of the 3-h ride, participants dismounted their cycles and were allowed to use the toilet and stretch. Upon remounting their ergometers, participants completed 10 maximal sprints interspersed with recovery periods as described above. No verbal encouragement was provided to the participants, the only information provided during the sprints was elapsed work completed (kilocalories) shown on the computer screen. Participants were given a verbal countdown in preparation for the start of each sprint and at 20, 10, 5, and 2 kilocalories to go in preparation for the end of each sprint. Supplement ingestion continued during the sprint procedure with the allocated quantity ingested ad libitum every 20 min. Immediately following sprints 1, 4, 7, and 10, exertion and gastrointestinal distress data were collected. Blood samples were also taken from six of the nine participants after sprints 1, 4, 7, and 10.

The power output of the VeloTron ergometer during both constant power and self-selected resistance modes was verified against 12-gauge SRM cranks (Rudolph Schulten, Jülich, Germany). Power was between 0 and 1.5% of the SRM over the range of workloads tested (50–700 W). The calibration of the three ergometers used was tested twice during the experimental period. The wattage deviation was consistently 0.5–1.5% from factory calibration.

Diet and Supplements

Diet and dietary supplementation. Immediately following the baseline test, participants started their first 2-wk diet condition. The diets consisted of food and drink supplements containing either the MCFA-rich oil (see Randomized-Esterified Oil) or LCFA oil in prescribed meal and snack replacements. The LCFA oil was a blend of one-third canola and two-thirds palm oil. Palm oil has a high content of long-chain saturated fatty acids, which provided control for saturation characteristics relative to trioctanoin. Both MCFA- and LCFA-rich diets were administered ad libitum in identical fashion. Foods and supplements were prepared on site by food technologists and catering services and included muffins, chocolate-flavored sports fudge bars, curries, bolognese sauce, and milk-like drinks. Each item excluded any ingredient naturally enriched with $^{13}$C (maize, sugarcane) and was produced to provide 15 g of experimental oil (either LCFA or MCFA rich) per serving and a fixed macronutrient composition: 15, 55, and 30% energy from protein, carbohydrate, and oil, respectively. Participants were instructed to eat a set number of study food portions per day based on a quantity determined by body mass calculated as proportional to a 75-kg cyclist ingesting 90 g of the experimental oils per day. In practice, the quantity of randomized esterified oil ingested during the 2-wk diet (intervention condition) was 100 (SD 11) g/day or 67 (SD 7) g MCFA/day. Participants recorded all food ingested over the first 2-wk experimental block and replicated that daily pattern over the proceeding two blocks. In addition to eating the foods and supplements during the day, participants were instructed to ingest the sports bars and milk-like drinks during training sessions to facilitate exercise gastrointestinal adaptation. Participants were also provided with extensive lists and instructed not to eat any foods naturally enriched in $^{13}$C beginning immediately after the baseline test (cane sugar and maize products) but to otherwise maintain their normal diet while incorporating the study foods. Instructing participants in this way has been shown to be effective in reducing background $^{13}$C enrichment (18, 19). Participants verbally reported adhering to the dietary interventions. Three participants reported experiencing slight cases of diarrhea and belching while on the intervention (MC-MC) diet, although none claimed to be aware of which condition they were on at the time. Both the LCFA- and MCFA-rich diets were otherwise well tolerated.

Exercise supplements. Four different milk-like drinks and emulsions were formulated for use during exercise. The drinks were flavored identically for placebo purposes. The drink containing the CHO+MCFA-rich esterified oil was made in two forms: the supplement ingested during the initial baseline and LC-MC and MC-MC performance (sprints) tests contained 5.7% esterified oil, 2.6% wheat-derived dextrose and 5.6% maltodextrin, 1.5% sodium caseinate emulsifier, 0.08% salt, colors, chocolate and vanilla flavoring; and an identical supplement ingested during the 3-h ride in the MC-MC and LC-MC dietary conditions but also containing esterified oil with 1-$^{13}$C octanoic-acid tracer incorporated into the esterified oil. The third, placebo-control exercise drink used in the LC-CHO condition contained the same ingredients as above minus the esterified oil and with addition of 4.0% milk powder to emulate color and taste. The
fourth drink used during the three $^{13}$C background trials consisted of identical ingredients to the drink containing the MCFAs-enriched esterified oil minus the oil and $^{13}$C tracer. The mean quantities of MCFAs-enriched esterified oil emulsion ingested were 112 g (SD 6.5) (75 g MCFAs, SD 4.3) and 44 g (SD 5.2) (29 g MCFAs, SD 3.5) during the 3-h ride and performance test, respectively.

In producing the exercise supplements, the oil was first heated to 50°C. Dry ingredients were mixed with water, colors, and flavors, and the two mixtures were then combined and homogenized to disperse the fat droplets (giving the drinks a milky appearance) and refrigerated. The drinks were made 1–5 days before ingestion and had a minimum refrigerated shelf life of 10 days.

Randomized-Esterified Oil

A blended oil mixture consisting of one-third canola (LCT) and two-thirds trioctanoin oil was mixed in a batch reactor by an impeller at 230 rpm. After the vacuum reached 100 mbar, the oil was dried for 30 min at 90°C. Temperature was then decreased to 60°C and 0.1 wt% sodium methoxide was added while stirring. Any air in the reactor was removed using vacuum and N$_2$ systems. After 30 min, the reaction was stopped by addition of a 5 wt% citric acid-water solution. The oil was washed three to four times until pH was below 7 and dried again before deodorization in a conventional batch deodorizer. Vacuum was adjusted to less than 5 mbar, stripping steam consumption was adjusted to 4 wt%, and temperature was raised to 160°C for 2 h. After deodorization, the oil was cooled by tap water circulation with N$_2$ protection and stored in a −25°C freezer.

$^{13}$C$_1$-Octanoic Acid-Enriched Esterified Oil

The 1-$^{13}$C$_1$-octanoic acid-enriched esterified oil was produced by esterification of 1-$^{13}$C-labeled octanoic acid (C8:0; Cambridge Isotope Laboratories, Andover, MA), oleic acid (18:1n-9) and glycerol in the proportion of 3:1:1:6. Components were mixed 10:1 with lipase (Novozym, Novozymes, Bagsvaerd, Denmark) at 60°C with constant stirring. Vacuum removed excess water from the process by passing the air through anhydrous Na$_2$SO$_4$ (JT Baker, Deventer, Holland). After a 24-h incubation period, the reaction was stopped by separating the oil and enzymes with filtering. The oil was then kept at −20°C until use. The $^{13}$C$_1$-enriched oil was mixed into the randomized esterified oil in six batches in an appropriate dilution that yielded a mean oil $^{13}$C$_1$-enrichment of 84 8‰ vs. PDB (SD 30) (0.006854 $^{13}$C/$^{12}$C ratio), which was confirmed to yield a detectable breath signal in pilot trials.

Psychometric Scales

Perceived exertion (soreness of legs and effort of cycling) and gastrointestinal distress (nausea, fullness/bloating, stomach cramp, reflux/burping) markers were measured using scales modeled from Borg’s CR10 scale (2). The verbal descriptors were associated with the scale: nothing 0, very weak 1, weak or mild 2, moderate 3, strong 5, very strong 7, extremely strong 10, and absolute maximum 13.5. Participants were instructed to make a pen mark on a continuous scale, rating the strength of their exertion or distress. For example, a question on their rating of perceived exertion was “how sore did your legs feel during the last sprint?” and a question on their rating of gastrointestinal distress was “how much discomfort do you feel in your stomach with respect to fullness/bloating?” The numerical value for each verbal anchor was not displayed on the scale charts, so as not to distract the participant from their rating, as the numerical value increased factorially in accordance with the CR10 scale by $\times$1.6. The CR10 scale used to construct the psychometric scales for this study was previously shown to be reliable and valid (3) and the coefficients of variation (CVs) for the present scales ranged from 21 to 79% of a scale unit (D. S. Rowlands, unpublished observations).

Analyses

$^{13}$C Enrichment. Breath samples were analyzed for $^{13}$C/$^{12}$C by gas chromatography continuous flow isotope ratio mass spectrometry (Finnigan Delta XP, Bremen, Germany). The enrichment of the esterified oil was measured using Elemental Analyzer isotope ratio mass spectrometry.

Calculations. Oxidation rates (g/min) of exogenous octanoic acid and total fat and CHO were calculated from $^{13}$C-enrichment and indirect calorimetry measurements. Isotopic enrichment of expired air was expressed as the delta per million difference ($\delta^{13}$C) between $^{13}$C/$^{12}$C ratio of the sample and a known laboratory reference standard (Pee Dee Belemnite; PDB) according to the formula: 31°C = ($^{13}$C/$^{12}$C ratio sample/$^{13}$C/$^{12}$C ratio standard) − 1)/100‰, where $^{13}$C/$^{12}$C standard = 0.0112372 (6). The amount of octanoic-acid oxidized is then calculated according to the formula: exogenous octanoic-acid oxidation (g/min) = $\dot{V}$CO$_2$ × ($\delta^{13}$CO$_2$ − $\delta_{bkg}$)/($\delta_{bkg}$ − $\delta_{ing}$), in which $\delta_{bkg}$ is the $^{13}$C enrichment of expired air in the 3-h background trial, $\delta_{exp}$ is the $^{13}$C enrichment of expired air during the 3-h ride with $^{13}$C-enriched esterified oil ingestion, and $\delta_{ing}$ is the $^{13}$C enrichment of the oil in the ingested exercise supplement, and k is the amount of CO$_2$ (liters) produced via oxidation of 1 g octanoic acid on a glycerol backbone (k = 1.2369 1/CO$_2$/g trioctanoin) (18). A conversion factor of 34.19 kJ/g was used to estimate MCFAs contribution to energy expenditure (25). Total carbohydrate and fat-oxidation rates were calculated using the non-protein respiratory quotient (16): carbohydrate oxidation (g/min) = 4.210 × $\dot{V}$CO$_2$ − 2.962 × $\dot{V}$O$_2$; fat oxidation (g/min) = 1.695 × $\dot{V}$O$_2$ − 1.701 × $\dot{V}$CO$_2$.

Conversion factors of 15.64 kJ/g (9) for CHO and 40.81 kJ/g (30) for fat oxidation were used to estimate contribution to energy expenditure. The oxidation rate of other fats was the total fat oxidation rate minus the octanoic acid oxidation rate.

Calculation of exogenous substrate oxidation is affected by the delayed equilibration of $^{13}$CO$_2$ originating from the tissues with the large endogenous HCO$_3$ pool. However, a physiological steady-state condition occurs relatively rapidly during exercise, and $^{13}$CO$_2$ in the expired air will be equilibrated with the $^{13}$CO$_2$/H$^{13}$CO$_2$ pool from around 60 min of exercise (31). As a consequence, calculations on substrate oxidation were only reported from 60 to 180 min of exercise.

Plasma. Blood was collected into lithium heparin-containing tubes, immediately centrifuged at 2,000 g for 12 min, aspirated into Eppendorf tubes, and snap-frozen in liquid nitrogen before being stored in a −80°C freezer until analysis. Lactate, glucose, potassium, and acid-base variables were analyzed using an automated blood gas analyzer (Bayer Rapidlab 800 system, Bayer HealthCare, Tarrytown, NY). Before analysis, a quality control test was run to evaluate the system for imprecision and inaccuracy. Two-point calibrations for all parameters were performed every 45–60 samples. One-point calibrations for all parameters were performed every 15–20 samples. One-point calibrations for PCO$_2$, glucose, and lactate were performed every three samples.

Sprint data. Mean power (J/s) for each interval was calculated from the inbuilt efficiency factor and kJ conversion factors in the Velotron software code: kcal=4.186×1.000×0.25/t, where kcal is the energy (kilocalories) expended by the participant during the sprint, 4.186 is the conversion factor from kilocalories to kilojoules, 0.25 is the efficiency factor of an exercising person, and t is the time it took the participant to complete the sprint in seconds.

Statistics

General method. The effect of diet condition on the outcome variables was estimated with mixed modeling (Proc Mixed) in the Statistical Analysis System (SAS9.1, SAS Institute, Cary, NC). Most dependent variables were analyzed after log transformation to reduce or eliminate effects of nonuniformity of error; the exceptions were variables where the unit or expression was as a percent (error non-
Facilitized), data sets containing numerals $<$0or $<$0<, and the psychometric data, where data heteroscedasticity is somewhat accounted for by the scale power conversion. For measures of performance, metabolism, and other dependent physiological variables, the effects of the diets were compared in a three-way model, whereas the analysis of psychometric variables also included the baseline exercise test responses. Qualitative and quantitative mixed linear models were applied to the time series data sets for the 3-h exercise and during the sprint procedure. In the former, sample time or sprint number were the qualitative repeated measures; whereas in the latter, sample time or sprint number were numeric x-axis variables. In the sprint performance data set in the quantitative model, sprint 10 was omitted from the model because it did not conform to the observed general linear decline in mean power evaluated between sprints 1 and 9. Analysis is based on the reduction in mean power per sprint. More negative means greater relative fatigue emulsion ingested during exercise; LC-MC, long-chain fatty-acid rich diet with the CHO + MCFA-rich esterified oil emulsion ingested during exercise; LC-CHO, long-chain fatty-acid rich diet with a CHO-only during exercise.

### Covariance parameters
In addition to the normal between-cyclist variance, cyclist$\times$varLC-MC and cyclist$\times$varMC-MC were specified random effects that assigns extra (fixed) variance associated with moving from the control to the intervention oil conditions. In the quantitative models, the covariances associated with moving between time points or sprints were included, again with extra variance assigned to the intervention conditions. The within-cyclist standard deviation was estimated from the residual variance for all data sets.

### Mechanisms analysis of the relationship between power output and nausea
The trends in mean power output and nausea ratings during the sprint procedure were compared using within-subject modeling. For each cyclist and experimental condition, a polynomial with linear and quadratic components was fitted to the data. The positional and linear slope effects derived from the polynomial analysis for nausea (there was no evidence for a quadratic effect) were included as covariates in separate repeated-measures analyses of the effects of treatment on the positional and linear slope effects for mean power. Reduction of the treatment effect by the covariate indicates the extent to which changes in sprint mean power were attributable to change in the covariate.

### Presentation of data
Measures of centrality and spread for participant descriptive, raw stable isotope, and dietary variables are raw means and standard deviations. Means derived from the analysis of log-transformed variables are back-log-transformed least-squares, or adjusted, means. The associated spread around these least-squares means is represented by percentage (geometric) standard deviations or factor standard deviations (SDf), implying $\times$/?. For example, for a plasma-glucose concentration of 5 mmol/l with a between-subject standard deviation of 20% (SDf 20), the typical variation is $5 \times 1.20$ to $5 \div 1.20$, or 6 to 4.16 mmol/l. Data in graphs and text are shown as least-squares means. Data are rounded to two significant digits or in some cases three, where the first digit is “1”.

In keeping with recent trends in inferential statistics, and the guidelines for American Physiological Society journals (7), we made magnitude-based inferences about population (true) values of the effect of the dietary intervention on outcomes by expressing the uncertainty in the effect as 90% confidence limits (CL) or interval (CI) and as probabilities that the true value represents a practically important substantive change (beneficial or detrimental; positive or negative; 1). The smallest substantive or worthwhile change in sprint performance variables was assumed to be a reduction or increase in sprint time of >1.1%. This value was derived from 0.5 times (15) the average of estimates of CV for road time trial and repeated sprint mean power (15, 39). The smallest standardized (Cohen) change in the mean for biochemical and psychometric variables was assumed to be 0.20 times the between-subject standard deviation for the value in the control group (LC-CHO condition; 4). The statistical outcomes were qualified according to the rule that if both the chance of a substantial benefit and detriment are >5%, then the true effect was assessed as unclear (could be beneficial or detrimental, increase or decrease). Otherwise, chances of substantial benefit or detriment were inferred as follows: <1%, almost certainly not; 1–5%, very unlikely; 5–25%, unlikely; 25–75%, possible; 75–95%, likely; 95–99%, very likely; >99%, almost certain (1). In the case where the chance that the benefit or detriment is <5% and the chance of the effect being trivial is greater than detriment or benefit, the likelihood of the effect being trivial is qualified. The statistical approach emphasizes precision of estimation rather than null hypothesis testing ($P$ values and statistical significance) and provides a qualitative interpretation of the practical importance defined by the confidence limits and the smallest important or non-trivial effect (7, 34).

### RESULTS

#### Performance

Mean power for sprints 1–9 was reduced by 7.1% and 10.9% in the MC-MC and LC-MC conditions relative to the LC-CHO condition, but the difference between MC-MC and LC-MC was unclear (Table 1, Fig. 2). There was no clear difference in the decline in mean power, (slope or fatigue effect) between conditions.

<table>
<thead>
<tr>
<th>Measure</th>
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<td>Sprints 1–9 Mean power</td>
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<td>Almost certainly detrimental</td>
<td>$-10.9 \pm 3.2$</td>
</tr>
<tr>
<td>Fatigue</td>
<td>$-3.4 \pm 5.9$</td>
<td>Unclear</td>
<td>$2.6 \pm 4.2$</td>
</tr>
<tr>
<td>Sprint 10 Mean power</td>
<td>$-3.4 \pm 3.9$</td>
<td>Unclear</td>
<td>$-5.4 \pm 6.5$</td>
</tr>
</tbody>
</table>

Values are means ± SD. *±90%CL: add and subtract this number to the mean effect to obtain the 90% confidence limits for the true difference. *Mean power is the overall mean power effect. *Fatigue is the percentage difference between the conditions in the linear decline in power evaluated between sprints 1 and 9. Analysis is based on the reduction in mean power per sprint. More negative means greater relative fatigue emulsion ingested during exercise; LC-MC, long-chain fatty-acid rich diet with the CHO + MCFA-rich esterified oil emulsion ingested during exercise; LC-CHO, long-chain fatty-acid rich diet with a CHO-only during exercise.

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Table 1. Comparison of the three dietary conditions on performance

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Values are means ± SD. *±90%CL: add and subtract this number to the mean effect to obtain the 90% confidence limits for the true difference. *Mean power is the overall mean power effect. *Fatigue is the percentage difference between the conditions in the linear decline in power evaluated between sprints 1 and 9. Analysis is based on the reduction in mean power per sprint. More negative means greater relative fatigue emulsion ingested during exercise; LC-MC, long-chain fatty-acid rich diet with the CHO + MCFA-rich esterified oil emulsion ingested during exercise; LC-CHO, long-chain fatty-acid rich diet with a CHO-only during exercise.
Mechanisms Analysis

For every one-unit increase in the position of the nausea curve, the position of the mean power curve decreased by 6.0 W (90% CI: 3.2– 8.8 W; likelihood of substantial decrease 93%). When the influence of nausea was added as a covariate in the polynomial analysis, the reduction in mean power in the LC-MC and MC-MC conditions, relative to the LC-CHO condition, was made unclear (Fig. 3).

Substrate Metabolism

Breath 13C-enrichments during the 3-h background trials are shown in Fig. 4. Accordingly, mean peak octanoic-acid oxidation rates at the 180-min sample during the LC-MC and MC-MC conditions, respectively, were 0.43 (SD 42%) and 0.38 (23%) g/min.

A statistical summary of substrate oxidation over the 60- to 180-min period of the 3-h ride is presented in Table 2. Overall relative energy contributions are shown in Fig. 5. In summary, octanoic acid oxidation was lower but endogenous fat oxidation higher in the MC-MC condition relative to the LC-MC condition (Table 2). Energy derived from the oxidation of endogenous fat was substantially reduced by the oil emulsion (MC-MC and LC-MC conditions), relative to the LC-CHO condition. There were statistically small reductions [Cohen effect-size statistic, ES (4)] in energy derived from endogenous carbohydrate oxidation of ES 0.22 and 0.32 in the MC-MC minus LC-CHO and the MC-MC minus LC-CHO comparisons.

The pattern of substrate oxidation over the 3-h ride is illustrated in Fig. 6. The increase in the octanoic acid oxidation rate from the 60th to the 180th min sample points was 0.14 g/min in the LC-MC condition and 0.16 g/min in the MC-MC condition. The difference in the increase in LC-MC relative to MC-MC (modeled slope effect) from 60 to 180 min was by factor of 0.85 (90% CI: 0.75– 0.97). The observed total carbohydrate oxidation rates changed little (<10%) from 60 to 180 min. In contrast with octanoic acid oxidation, endogenous fat oxidation rates declined from 60 to 180 min. In the LC-MC, MC-MC, and LC-CHO conditions, the respective declines were 0.08, 0.27, and 0.06 g/min. The difference in the decline in MC-MC relative to LC-CHO was 1.5-fold (1.0–2.3). The remaining observed between-treatment slope differences for endogenous fat oxidation were unclear.

Psychometric Scales

A statistical summary of gastrointestinal distress (nausea, fullness/bloating, stomach cramp, reflux) and exertional parameters (leg soreness, perceived effort) is shown in Tables 3 and 4. The responses for nausea, fullness/bloatingedness, and perceived effort were selected to illustrate the magnitude and temporal effects of treatment and exercise duration (Fig. 7). 3-h ride. Substantial increases in gastrointestinal distress were recorded in the MC-MC and LC-MC conditions, relative to LC-CHO; exertion, however, was likely to be greater in the LC-MC condition only, relative to the LC-CHO condition (Table 3). The increase (slope) in scale unit rating from the sample at 20 min to the 180th min was likely to be greater in the LC-MC condition relative to the LC-CHO condition by 1.6 scale units (90% CI: 0.4–2.8 scale units) for nausea, 0.8 scale units (0.4–1.2) for cramp, 1.2 scale units (0.8–1.6) for reflux, and 1.7 scale units (0.5–2.9) for fullness/bloating; fullness/bloating also increased 1.5 scale units (0.2–2.8) more in the LC-MC condition relative to the MC-MC condition. In the MC-MC condition the difference in the increase in LC-MC relative to MC-MC (modeled slope effect) from 60 to 180 min was by factor of 0.85 (90% CI: 0.75–0.97). The observed total carbohydrate oxidation rates changed little (<10%) from 60 to 180 min. In contrast with octanoic acid oxidation, endogenous fat oxidation rates declined from 60 to 180 min. In the LC-MC, MC-MC, and LC-CHO conditions, the respective declines were 0.08, 0.27, and 0.06 g/min. The difference in the decline in MC-MC relative to LC-CHO was 1.5-fold (1.0–2.3). The remaining observed between-treatment slope differences for endogenous fat oxidation were unclear.

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condition, the increase in scale unit rating was greater relative to the LC-CHO condition by 1.9 (0.7–3.1) for nausea and 1.6 (0.3–2.9) for reflux. The remaining comparisons were unclear.

**Performance test.** The performance test accentuated the effect of the LC-MC treatment on all gastrointestinal distress and exertional parameters, relative to the LC-CHO condition. Likely substantial increases in nausea and fullness/bloating were recorded in the MC-MC condition, but the effects of this condition on stomach cramp, reflux, and leg soreness were less clear, and there was a decrease in perceived effort, relative to the LC-CHO condition. Relative to the LC-MC control, the MC-MC treatment substantially attenuated most markers of gastrointestinal distress and perceived effort.

The incline (slope) in scale unit rating from sprints 1 to 10 was likely to be greater in the LC-MC condition relative to the MC-MC condition by 2.2 scale units (90% CI: 0.3–4.0 scale units) for nausea, 1.8 scale units (0.5–3.0) for reflux, and 1.3 scale units (0.3–2.3) for fullness/bloating. In the LC-MC condition relative to the LC-CHO condition, the increase was greater by 2.3 scale units (0.6–4.0) for nausea and 1.13 scale units (0.1–1.1) for fullness/bloating. The remaining comparisons were unclear.

**Plasma, Electrolytes, Acid-Base Status, Glucose, and Lactate**

The effects of treatment condition on plasma bicarbonate, potassium, glucose, and lactate concentrations during the exercise are shown in Fig. 8. No clear differences were observed between conditions for hydrogen ions (not shown) or standard bicarbonate concentrations during the 3-h ride or performance test.

**Glucose.** During the 3-h ride, glucose was 6.9% (2.3–11.5%) and 12.6% (9.3–15.9%) lower in the LC-MC and MC-MC conditions, respectively, relative to the LC-CHO condition, and the difference between LC-MC and MC-MC was 5.4% (−0.2 to 11.0%). During the performance test, glucose was overall 5.3% (2.4 to 8.2%) and 9.9% (7.3 to 12.5%) lower in the LC-MC and MC-MC conditions, relative to the LC-CHO condition.

Table 2. Summary of the effect of the three dietary conditions on substrate metabolism from the 60th to 180th min of the 3-h ride

<table>
<thead>
<tr>
<th>Substrate</th>
<th>Substrate Oxidation* g/min</th>
<th>Substrate Oxidation* kJ/min</th>
<th>Effect Comparisons (% ±90% CLb and Qualitative Inference)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate</td>
<td>1.9 (24)</td>
<td>2.0 (22)</td>
<td>2.0 (16)</td>
</tr>
<tr>
<td>Octanoic acid</td>
<td>0.32 (19)</td>
<td>0.37 (40)</td>
<td>—</td>
</tr>
<tr>
<td>Endogenous fats</td>
<td>0.31 (71)</td>
<td>0.28 (46)</td>
<td>0.61 (37)</td>
</tr>
</tbody>
</table>

* The effects of dietary condition are the back log-transformed least-squares mean value for 60 to 180 min sampling points inclusive with corresponding percentage between-subject standard deviations (SDf) in parentheses. To determine the range of the percentage standard deviation, convert the SDf to a factor then multiply and divide by the mean.  ±90%CL: add and subtract this number to the mean effect to obtain the 90% confidence limits for the true difference.

Fig. 5. Mean substrate oxidation as percentage contribution to total energy expenditure during the 3-h rides.
Table 3. Summary of the overall effect of the three dietary conditions on gastrointestinal distress and exertional parameters during the 3-h ride

<table>
<thead>
<tr>
<th>Parameter</th>
<th>MC-MC minus LC-CHO</th>
<th>LC-MC minus LC-CHO</th>
<th>MC-MC minus LC-MC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea</td>
<td>1.0±0.4</td>
<td>0.9±0.7</td>
<td>0.1±1.1</td>
</tr>
<tr>
<td>Fullness/bloating</td>
<td>1.1±0.6</td>
<td>0.8±1.0</td>
<td>0.3±1.2</td>
</tr>
<tr>
<td>Stomach cramp</td>
<td>0.2±0.2</td>
<td>0.4±0.3</td>
<td>−0.2±0.3</td>
</tr>
<tr>
<td>Reflux</td>
<td>0.9±0.7</td>
<td>0.7±0.9</td>
<td>0.2±1.3</td>
</tr>
<tr>
<td>Leg soreness</td>
<td>0.3±0.5</td>
<td>0.5±0.5</td>
<td>−0.2±0.6</td>
</tr>
<tr>
<td>Perceived Effort</td>
<td>0.4±0.5</td>
<td>0.7±0.6</td>
<td>−0.3±0.8</td>
</tr>
</tbody>
</table>

a 90% CL: add and subtract this number to the mean effect to obtain the 90% confidence limits for the true difference.

**DISCUSSION**

The purpose of this study was to determine if a 2-wk high-MCFA dietary adaptation period would attenuate gastrointestinal distress, increase octanoic-acid oxidation rate, and enhance performance while ingesting a CHO+MCFA-rich oil emulsion during endurance cycling in a sample of well-trained male cyclists. The CHO+MCFA-rich oil emulsion resulted in mild to moderate gastrointestinal distress ratings during the repeated-sprint performance test with naive (baseline) exposure and in the control dietary condition (LC-MC), but dietary-MCFA adaptation attenuated these effects. During the 3-h preloading exercise high amounts of ingested MCFA were oxidized. However, MCFA-adaptation reduced octanoic-acid oxidation, increased endogenous-fat oxidation, and lead to a likely small reduction in endogenous-carbohydrate oxidation. Despite these gastrointestinal and metabolic effects, almost certain substantial impairments of sprint performance were observed in the LC-MC and MC-MC conditions relative to the LC-CHO control.

The MCFA-dietary adaptation intervention was intended to alleviate gastrointestinal distress associated with MCFA ingestion during exercise and to therefore permit the potential benefits to fuel-substrate supply and performance to be provoked. Indeed, there was a mild reduction of gastrointestinal distress, but this was insufficient to improve sprint performance relative to the two dietary control conditions. Nausea was found to have a moderately-strong negative effect on sprint mean power and to our knowledge this is the first time that the association between nausea and sprint performance has been quantified in this line of research. Such an effect suggests that performance may have been, at least partly, limited by a central mechanism. Noakes et al. (28) propose that fatigue is regulated by a central governor, resulting from the integration of physiological, biochemical and sensory feedback from the

Table 4. Summary of the overall effect of the 3 dietary conditions on gastrointestinal distress and exertional parameters during the performance test

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<th>MC-MC minus LC-MC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea</td>
<td>1.2±0.6</td>
<td>3.1±0.6</td>
<td>−1.9±0.5</td>
</tr>
<tr>
<td>Fullness/Bloating</td>
<td>1.1±1.0</td>
<td>2.5±0.5</td>
<td>−1.4±1.2</td>
</tr>
<tr>
<td>Stomach cramp</td>
<td>0.1±0.6</td>
<td>0.5±0.3</td>
<td>−0.4±0.5</td>
</tr>
<tr>
<td>Reflux</td>
<td>0.6±1.2</td>
<td>2.3±0.6</td>
<td>−1.7±1.2</td>
</tr>
<tr>
<td>Leg soreness</td>
<td>0.6±0.6</td>
<td>1.4±1.6</td>
<td>−0.8±1.7</td>
</tr>
<tr>
<td>Perceived effort</td>
<td>−1.0±0.3</td>
<td>0.9±0.5</td>
<td>1.9±0.5</td>
</tr>
</tbody>
</table>

a 90% CL: add and subtract this number to the mean effect to obtain the 90% CLs for the true difference.
periphery to prevent complete energy depletion. According to this model, nausea may have induced peripheral fatigue before rate limitation by energy supply, such as muscle-glycogen depletion.

In response to naïve exposure, previously only Goedecke et al. (11) have also found CHO+MCT ingestion to clearly impair performance relative to CHO-only during ~5-h of exercise. In their study, the performance of intermediary sprints became increasingly worse, and time to complete the final 200-kJ time trial was longer by 13% (90%CI: 8 to 18%) with CHO+MCT ingestion. In an earlier study, Goedecke et al. (12) also observed mean impairments of 3% and 2.5% in 40-km time-trial performance following a 2-h ride reported as statistically insignificant (P-values not provided) when participants ingested high MCT+CHO and low MCT+CHO supplements, respectively, compared with CHO-only. On the other hand, other authors have reported non-statistically clear (P-value >0.05) trends toward performance improvements with CHO+MCT ingestion. Jeukendrup et al. (22) and Vistisen et al. (36) observed on average 1% and 1.5% improvements in constant work or constant time tests, respectively, when participants ingested a CHO+MCT supplement compared with CHO-only. The only group reporting a clear performance benefit as a 2.5% (90%CI: 0.5 to 4.5) enhancement in 40-km time following a 2-h preload was also the only group reporting no gastrointestinal distress (35). This finding is in contrast to all of the other studies feeding >30 g of MCT, including the present study. Van Zyl et al. (35) suggested that the increase in performance in their study was due to the observed increased availability of plasma fatty acids and the subsequent sparing of endogenous carbohydrate (implying muscle glycogen). This result contrasts the present findings, as well as that of others.
(12, 20–22) which show that MCT ingestion during exercise spares only endogenous fat rather than endogenous carbohydrate utilization.

The impaired performance in this and several of the previous studies might be largely attributed to the substantial gastrointestinal distress (11, 12, 22); indeed the present study provides quantitative evidence through the relationship with nausea. Additionally, it has been suggested that MCFAs may not even reach the systemic circulation as MCFAs (36), which would exclude utilization of their potentially beneficial properties in the muscle cells. There are several possibilities for the fate of MCFAs other than oxidation in muscle mitochondria, including: 1) elongation in the liver before release into the systemic circulation, limiting their ability to rapidly enter the muscle mitochondria (36); 2) conversion into ketone bodies because of the rapid build-up of acetyl-CoA in the mitochondria, which are then either used as energy substrate by the body, or excreted through the urine and breath (10, 24); or 3) not absorbed by the gut and excreted in the feces.

Despite the unknown fate of ingested MCFAs, it has been demonstrated that some are oxidized, either in the form of MCFAs, elongated fats, or ketone bodies. Using the $^{13}$C stable-isotope method, it has previously been shown that MCFAs can be oxidized up to $\sim 0.15$ g/min when co-ingested with CHO (19). In the present study, however, the maximum mean octanoic-acid oxidation rate was almost 3-fold greater at 0.43 g/min. These oxidation rates were reached in the 180th min of the 3-h ride, and approximated MCFA ingestion rates. The elevated rates of octanoic-acid metabolism observed in this study suggest a unique quality of our supplements that promoted high absorption and oxidation rates. Fat digestion is normally slow because triacylglycerols aggregate into large droplets in the small intestine, slowing the action of pancreatic lipase. However, our supplements were suspended in an emulsion, which distributes lipids into smaller droplets, thereby increasing their accessibility to pancreatic lipase. Also, cyclists were fed MCFAs and long chain fatty acids in the form of structured triacylglycerols, which may have enhanced MCFA absorption due to the increase in pancreatic secretion which is promoted by long-chain triglyceride, but not MCT, ingestion (27). Therefore, more extensive hydrolysis of the structured triacylglycerols may have occurred compared with MCT solutions, leading to greater absorption, blood concentrations, and mitochondrial oxidation.

Interestingly, mean octanoic-acid oxidation rates in the intervention MC-MC condition were almost certainly 24% lower than that observed in the dietary-adaptation control LC-MC condition (Table 2). Data were checked for errors, so we have no clear explanation for this finding that was opposite to that anticipated. It may be possible that dietary MCFA adaptation increased nonoxidative metabolism of the ingested octanoic acid. Sprint power output and plasma-lactate concentrations were higher in the LC-CHO condition, relative to both MCFA-rich supplement conditions (Fig. 8). Higher exercise intensity would also explain the elevated plasma $K^+$ concentrations observed during the performance test in the LC-CHO condition, as $K^+$ release from the intracellular space is proportional to exercise intensity (37).

In summary, the ingestion of a CHO+MCFA-rich esterified oil emulsion resulted in high exogenous MCFA-oxidation rates, but induced mild-moderate gastric distress symptoms that was quantitatively associated with a substantial impairment in sprint performance. These effects were attenuated by 2-wk’ adaptation to a diet rich in octanoic acid but insufficiently so to result in a benefit to performance from the MCFA-rich exercise supplement. We conclude that the majority of recent research into the possible ergogenic effects on male endurance performance of MCFA ingestion, in the form of native or structured oils, does not merit the need for further investigation. Even with the relatively tolerable gastrointestinal distress and the high MCFA-oxidation rates in the present study, predominantly only negative performance outcomes were observed. However, the results of the present study do warrant further investigation into the effects of nausea on performance, and the mechanisms behind this detriment.

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