



Effects of a 3-week High-Fat-Low-Carbohydrate Diet on Lipid and Glucose Profiles in Experienced, Middle-age Male Runners

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ABSTRACT

International Journal of Exercise Science 12(2): 786-799, 2019. This study examined glucose and lipid marker responses following a 3-week, ad libitum low-carbohydrate-high-fat diet (LCHF; ~70% kcals from fat, <50 g/day of net carbohydrates) versus the habitual mixed macronutrient diet (HMD) of eight middle-age, trained male runners (40 ± 10 years; VO₂peak = 49 ± 4 mL·kg⁻¹·min⁻¹). Blood was drawn at 0600 from an antecubital vein after an overnight fast under conditions of no exercise/heat stress (NEXH; 48 h of restriction from intense exercise) or 24-h after exercise/heat stress (EXH; 60 min run in hot conditions plus 5-km time trial) for both dietary conditions. Glucose improvement during LCHF approached but did not reach statistical significance (p = 0.07). Pre-exercise triacylglycerol did not differ between treatments but decreased > 20 mg/dL (p < 0.05) for both treatments from NEXH to EXH (HMD = 42 ± 16; LCHF = 35 ± 21 mg/dL). Main effects for diet were exhibited for HDL-C during NEXH and EXH (HMD = 48 ± 10 and 50 ± 11; LCHF = 57 ± 13 and 60 ± 13 mg/dL), and LDL-C also increased (p = 0.02) by ~20 mg/dL for LCHF at both collection points resulting in ~30 mg/dL greater total cholesterol for LCHF before and 24-h after exercise (p < 0.05). A 3-week, ad libitum LCHF did not elicit significant negative cardiovascular disease risk in male runners 30-50 years of age with healthy pre-intervention lipid and glucose marker status.

KEY WORDS: Ketogenic diet, HDL-C, Lp(a), vLDL-C

INTRODUCTION

The predominant viewpoints have supported prioritizing carbohydrates as the primary macronutrient for endurance athletes, and current recommendations (22) suggest endurance athletes consume 6-12 grams of carbohydrates per kilogram body mass a day depending on training volume and intensity, but these recommendations have not been without opposition (16,26). Since Phinney and colleagues (16,17) conducted seminal research examining metabolic adaptations to high fat, very low carbohydrate ketogenic diets in well-trained endurance athletes, multiple attempts have been made to explore the potential performance enhancements

for endurance athletes adopting a low-carbohydrate-high-fat and/or ketogenic diets (LCHF) with mixed results (2,3,7,12,13,29).

Considerable efforts have been made to examine the effects of LCHF diets on metabolic syndrome risk factors in both normal weight and overweight men and women (for a review see Volek and Sharman (27)). Although, individual responses display considerable intersubject variability, general trends with LCHF diets in these populations have included: body mass/fat loss, decreased triacylglycerol levels (TG), increased high (HDL-C) and low density lipoprotein cholesterol (LDL-C) with an increase in larger, less dense versus smaller, more dense LDL subfractions. However, less attention has been given to physically active, non-elite populations (12,29).

Low carbohydrate, high fat diets have gained considerable mainstream popularity in the endurance athlete community, but the effects of LCHF on lipid and glucose markers have not been well elucidated in this population. Therefore, the purpose of this study was to determine how lipid and glucose profiles are altered when middle-age male runners accustomed to HMD diets self-selected their food items for 3 weeks with goals of restricting carbohydrate intake to <50 g/day and total calories from fat consumption approaching ~70%. Additionally, the influence of exercise on these markers was examined with data collection taking place 48-h after refraining from exercise and 24-hours after an intense running bout of 75-90 min.

METHODS

Participants

Eight non-elite but well-trained ($\text{VO}_2\text{peak} = 49 \pm 4 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), middle-age, male runners (40 \pm 10 years) were recruited from regional running organizations and completed all study requirements. All runners competed in long-distance road races, and 7 of the 8 participants reported endurance training durations lasting 10-15 h/week. The remaining runner reported training 5-10 h/week. Runners that reported taking any medication related to metabolic syndrome components were excluded from participation. All participants passed two pre-exercise health screening questionnaires and exhibited systolic and diastolic blood pressures of <140 and <90 mm/hg respectively in their initial session. Participants completed written consent prior to any data collection. All procedures were approved by the University of North Alabama's Institutional Review Board.

In the initial session, consumption of a HMD diet was confirmed by completion of a dietary history questionnaire. No runners reported low carbohydrate intake and all runners consumed a wide variety of high carbohydrate foods and beverages. Following completion of training and dietary history questionnaires, nude body mass ($81.7 \pm 1.0 \text{ kg}$) and height ($177.0 \pm 7.8 \text{ cm}$) were assessed on a digital scale (BWB-800, Tanita Co., Alinton Heights, IL) and stadiometer. Body composition ($19.3 \pm 6.0 \text{ BF}\%$) was assessed using bioelectrical impedance analysis (BIA) (seca mBCA 514, seca gmbh & co. Hamburg, Germany). Runners then completed a graded treadmill VO_2peak test (TrueOne2400, Parvo Medics, UT, USA) (10).

Protocol

The data reported in this manuscript was collected as a component of a larger project (10) examining the effects of LCHF on performance, metabolic adaptation, thermoregulatory responses, and change in body composition. Detailed description of the exercise protocols and results are described by Heatherly et al. (10). Following the initial session, participants reported to the laboratory for four additional sessions. Blood was collected following an overnight fast in all four subsequent sessions. In brief, following 48-h of cessation from exercise, participants completed a taxing exercise protocol that included a 50 min run in a hot and humid environment followed by a 5-km time trial (sessions 2 and 4). Markers analyzed before the start of sessions 2 and 4 are operationally defined as non-exercise/heat stress conditions (NEXH). Participants returned 24-h later (sessions 3 and 5) for subsequent blood collections. Markers collected during sessions 3 and 5 are operationally defined as exercise/heat stress conditions (EXH) for clarity. No exercise was allowed between blood collections during sessions 2 and 3 or 4 and 5. The HMD sessions occurred ~ 1 week after the screening session with runners consuming their typical HMD diets. The LCHF phase began immediately after the conclusion of the HMD phase. No dietary interventions were made by the investigative team and all the participants maintained the LCHF intervention for ~21-28 days.

Nutritional Intervention: Significant educational efforts were made during the initial session to prepare runners to transition to the LCHF diet phase. Participants were instructed on how to interpret nutrition labels and assess serving sizes. The intervention diet was based on guidelines from a book written for endurance athletes following a LCHF (25). A copy of the book was given to runners to read prior to the LCHF intervention. Runners were asked to consume ~70% of their daily calories in the form of fat while limiting carbohydrate intake to 50 g or less per day excluding carbohydrates from fiber or sugar alcohols. Runners were provided with an extensive list of LCHF food item choices, recipes, links to websites with recipes for LCHF meals, and examples of daily menus. Additionally, a list of non-LCHF food and beverages were provided to guide runners on what items should not be consumed.

Daily logs were shared with the investigative team for the first 4-7 days of the LCHF phase to confirm that the diet was being followed. Four participants were already using an online nutritional app (MyFitnessPal, Calorie Counter, 2017, Baltimore, MD) daily before the study began. They were instructed to simply continue using the app and provided investigators with printed reports. The remaining 4 participants logged all food daily in a paper journal. At the conclusion of the study, investigators logged their nutrient intake into the same nutrition app. Food and beverage intake were averaged for 3 consecutive days (2 weekdays and 1 weekend day) before the NEXH sessions for both nutritional conditions. Total carbohydrate (minus fiber), protein, and fat equaled 309 ± 155 g/day ($43 \pm 11\%$ kcal/day), 111 ± 50 g/day ($17 \pm 8\%$ kcal/day), 121 ± 47 g/day ($38 \pm 7\%$ of kcal/day) for HMD and 30 ± 13 g/day ($7 \pm 4\%$ kcal/day), 133 ± 43 g/day ($29 \pm 9\%$ of kcal/day), and 137 ± 53 g/day ($64 \pm 9\%$ kcal/day) for LCHF. Total daily caloric intake for HMD (2820 ± 955 kcal/day) exceeded that of LCHF (1886 ± 520 kcal/day) by close to 1000 kcals. Although carbohydrate consumption was approximately 10-fold greater during HMD than LCHF, it should be noted that total daily carbohydrate intake was less than usually promoted for endurance athletes.

Lipid and Glucose Marker Collection and Analysis: Participants were instructed to eat dinner and consume 500 ml of water prior to 1900 and consume an additional 500 ml of water before bed the evening before each experimental session. Participants arrived the following morning between 0500 and 0600 and immediately consumed one more 500 mL bottle of water before being seated in a reclined phlebotomy chair. At minute 10, blood was drawn from an antecubital vein and collected in ethylenediaminetetraacetic acid coated vacutainers for plasma and serum separator vacutainers for serum. Plasma tubes were centrifuged (Centrifuge 83058-42, Cole-Parmer, Vernon Hills, Illinois) for 20 min then the separated plasma was pipetted into 1.5 mL vials that were stored in a -80 °C freezer. Serum vials were allowed to clot for 20 min before being centrifuged and frozen. BIA was then performed to estimate body fat percentage using similar procedures conducted in the screening session and were repeated after blood collection during sessions 2-5. Further, a seven site skinfold test (Lange Calipers, Beta Technology Inc., Cambridge, MD) (1) was completed on both EXH sessions. Skinfold thickness was measured at the chest, triceps, midaxillary, subscapula, abdominal, suprailiac, and mid-thigh sites.

Plasma glucose (Kit#G518-150-Hexokinase, Anaheim, CA) was analyzed in duplicated by an enzymatic colorimetric method. Serum total cholesterol (TC) (Kit# 85430, Cliniaqa, San Marcos, CA) and triacylglycerol (TG) (Kit# 85460, Cliniaqa, San Marcos, CA) were analyzed in duplicated by an enzyme-linked immunosorbent assay. The fractions of serum lipoprotein-cholesterol (VLDL-C, LDL-C, Lp(a), HDL-C) were analyzed by electrophoresis (Cat. # 3438 SPIFE Vis Cholesterol, Helena Laboratory, Beaumont, TX) using the SPIFE 3000 electrophoresis system (Helena Laboratory, Beaumont, TX). In brief, 70 µL of serum samples, in duplicate, were applied to an agarose gel followed by 20 min of electrophoresis at 16 °C with 400 volts. After applying a staining reagent (Cat. # 3438, Helena Laboratory, Beaumont, TX), additional electrophoresis was performed at 30 °C for 15 min. The gel was washed and dried at 70 °C for 20 min, and the density of stained lipoprotein-cholesterol bands were measured in a scanning densitometer (Epson Perfection V 700, Long beach, CA) using Quick Scan 2000 software (Helena Laboratory, Beaumont, TX).

Statistical Analysis

A 2 x 2 (NEXH vs EXH and HMD vs LCHF) repeated measures factorial ANOVA was performed to compare parametric data. If main or interaction effects were found, Bonferroni post hoc adjustment were incorporated when appropriate. Cohen's d effect sizes were calculated for dependent variables at like time points. Individual responses for each variable are presented in Figures 1-7. Participant responses are color coded between figures. Because of the high frequency of no detection of Lp(a) and vLDL-C, data are simply presented by individual responses in Figures 6 and 7 respectively. All data are presented as mean ± SD. An alpha level of 0.05 was determined to represent statistical significance a priori.

RESULTS

A more detailed description and discussion on anthropometric outcomes is available in Heatherly et al. (10). Briefly, body mass and percent fat decreased in LCHF for every comparison excluding BIA during EXH condition (Table 1).

Table 1. Fasting lipid and glucose profiles and anthropometric data (n = 8; mean \pm SD).

| | NEXH | | | EXH | | |
|-----------------------|-----------------|----------------|------|-----------------|----------------|------|
| | HMD | LCHF | ES | HMD | LCHF | ES |
| Glucose (mg/dL) | 96 \pm 9 | 91 \pm 14 | 0.43 | 98 \pm 9 | 88 \pm 5 | 1.43 |
| TG (mg/dL) | 65 \pm 17† | 67 \pm 35‡ | 0.08 | 42 \pm 16 | 35 \pm 21 | 0.38 |
| TC (mg/dL) | 144 \pm 8 | 173 \pm 27† | 1.66 | 141 \pm 12 | 171 \pm 23† | 1.71 |
| HDL-C (mg/dL) | 48 \pm 10 | 57 \pm 13† | 0.78 | 50 \pm 11 | 60 \pm 13* | 0.83 |
| TC/HDL-C ratio | 3.1 \pm 0.6‡ | 3.1 \pm 0.7‡ | 0.03 | 2.9 \pm 0.5 | 3.0 \pm 0.7 | 0.10 |
| LDL-C (mg/dL) | 93 \pm 12 | 114 \pm 26 | 1.11 | 90 \pm 11 | 109 \pm 24* | 1.09 |
| Lp(a) (mg/dL)** | 2.0 \pm 2.4 | 1.9 \pm 3.0 | -- | 1.4 \pm 2.0 | 1.9 \pm 2.9 | -- |
| Body mass (kg) | 81.6 \pm 7.1† | 79.5 \pm 6.4 | 0.31 | 81.1 \pm 7.0† | 78.4 \pm 6.2 | 0.41 |
| BIA body fat (%) | 20.0 \pm 5.3† | 17.9 \pm 4.9 | 0.21 | 19.1 \pm 6.7 | 19.0 \pm 5.5 | 0.02 |
| Skinfold body fat (%) | -- | -- | -- | 18.0 \pm 3.5† | 16.3 \pm 3.5 | 0.49 |

TG = triacylglycerol; TC = total cholesterol; HDL-C = high density lipoprotein cholesterol; LDL-C = low density lipoprotein cholesterol; Lp(a) = lipoprotein A; NEXH = non-exercise/heat stress marker collection; EXH = 24-h post exercise/heat stress marker collection; ES = Cohen's d effect size

† = Significant difference ($p < 0.05$) versus HMD at same collection point.

‡ = Significant difference ($p < 0.05$) versus 24-h post-exercise within treatment.

* = Approached significant difference ($p \leq 0.07$) for main effect of HMD versus LCHF or for LCHF versus HMD at same collection point.

** = not analyzed using parametric statistics due to high number of responses with no detection of Lp(a).

interaction effects were discovered for any lipid or glucose variable. Group results are presented in Table 1 and individual results are presented in Figures 1-7. The main effect for treatment neared but did not reach significance ($p = 0.07$) for glucose. The lack of statistical difference was likely due to two outlier participants that exhibited increases of ~ 30 mg/dL in opposite directions from NEXH condition in HMD to the LCHF condition, resulting in large increases in variance for a small sample size (Figure 1).

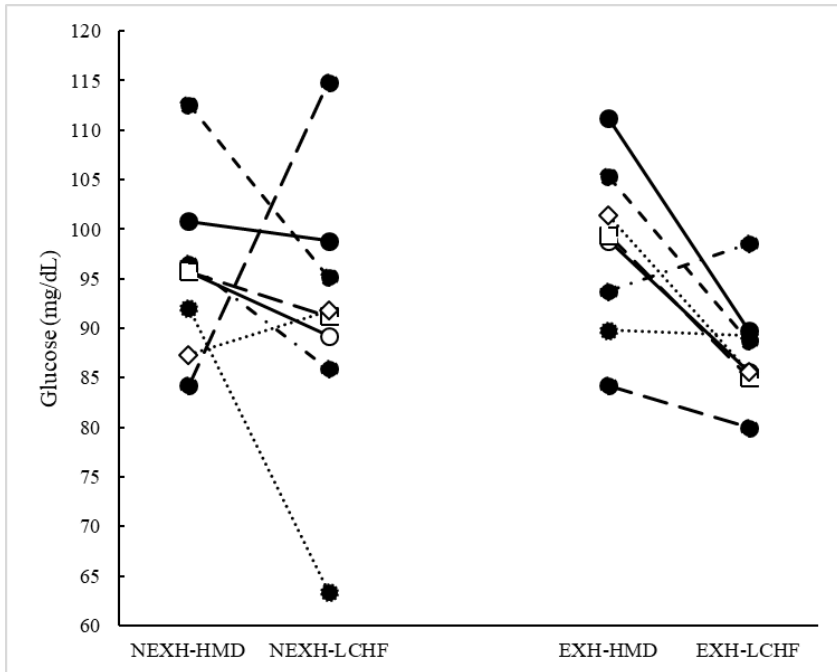


Figure 1. Individual fasted glucose responses after refraining from physical activity (Pre) and ~24 h following experimental exercise procedures (Post).

A significant main effect ($p < 0.001$) was found for TG with HMD and LCHF both decreasing 24-h after EXH, but no effect was found for treatment despite 11 of 16 samples exhibiting an increase of ≥ 9 mg/dL of HDL-C at like collection points between HMD and LCHF and no participants demonstrating a decrease in HDL-C (Figure 2).

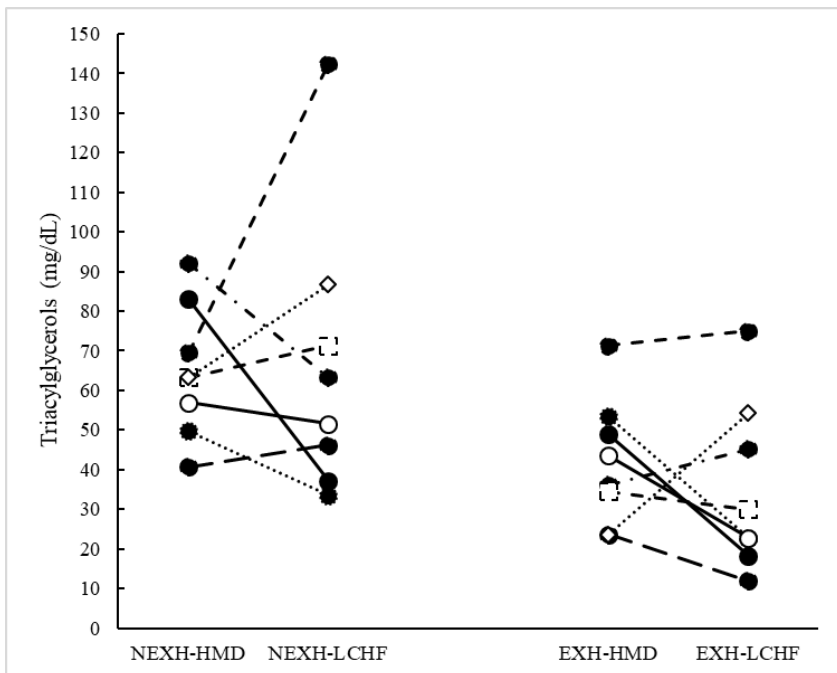


Figure 2. Individual fasted triacylglycerol responses after refraining from physical activity (Pre) and ~24 h following experimental exercise procedures (Post).

A significant main effect was found for TC ($p = 0.004$) with LCHF exceeding HMD before and 24-h after exercise (Table 1). These differences were attributed to approximately 10 and 20 mg/dL increases in HDL-C and LDL-C respectively for both treatments (Table 1; Figures 3-5).

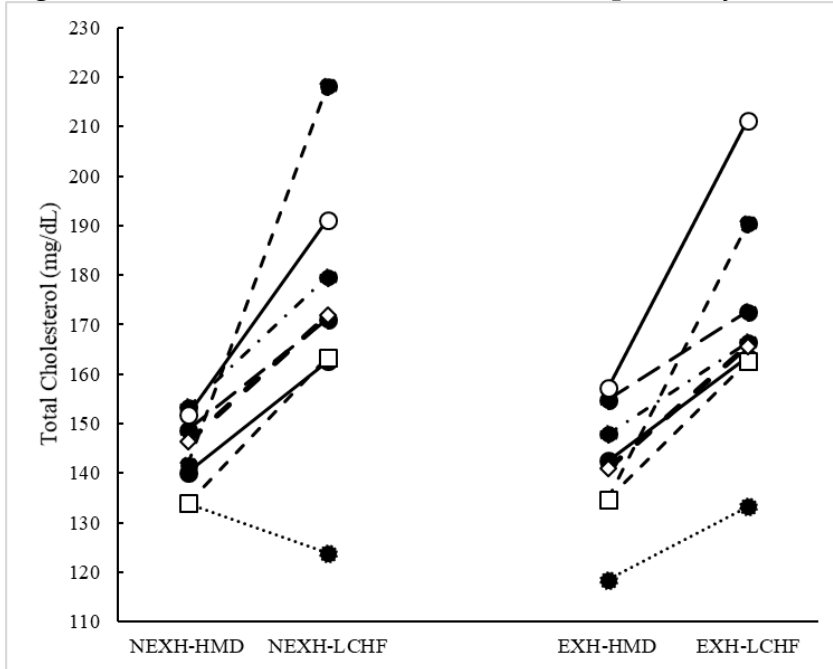


Figure 3. Individual fasted total cholesterol responses after refraining from physical activity (Pre) and ~24 h following experimental exercise procedures (Post).

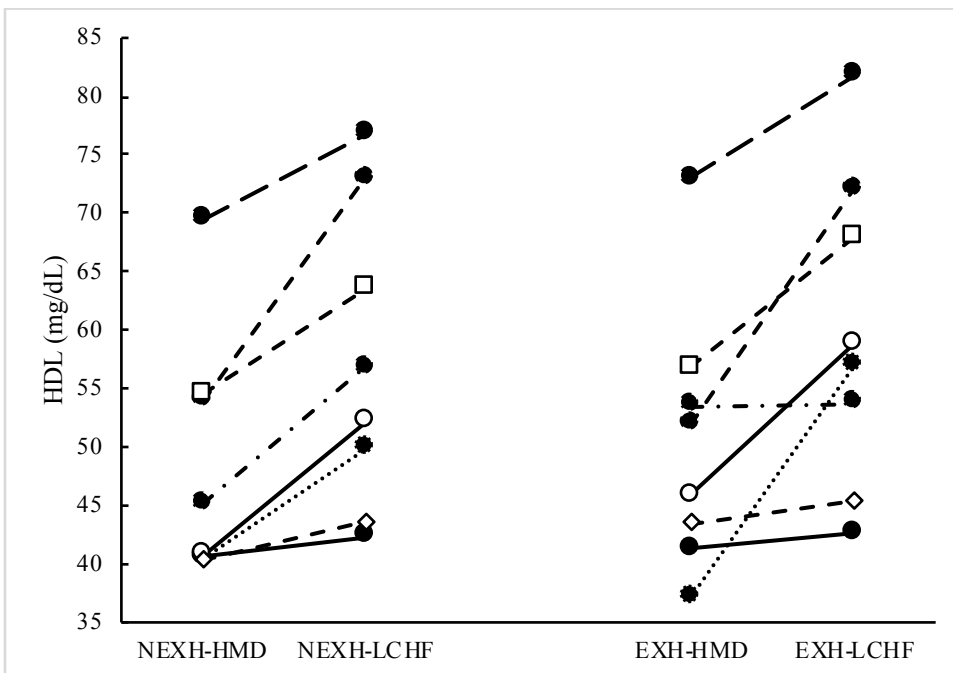


Figure 4. Individual fasted high-density lipoprotein responses after refraining from physical activity (Pre) and ~24 h following experimental exercise procedures (Post).

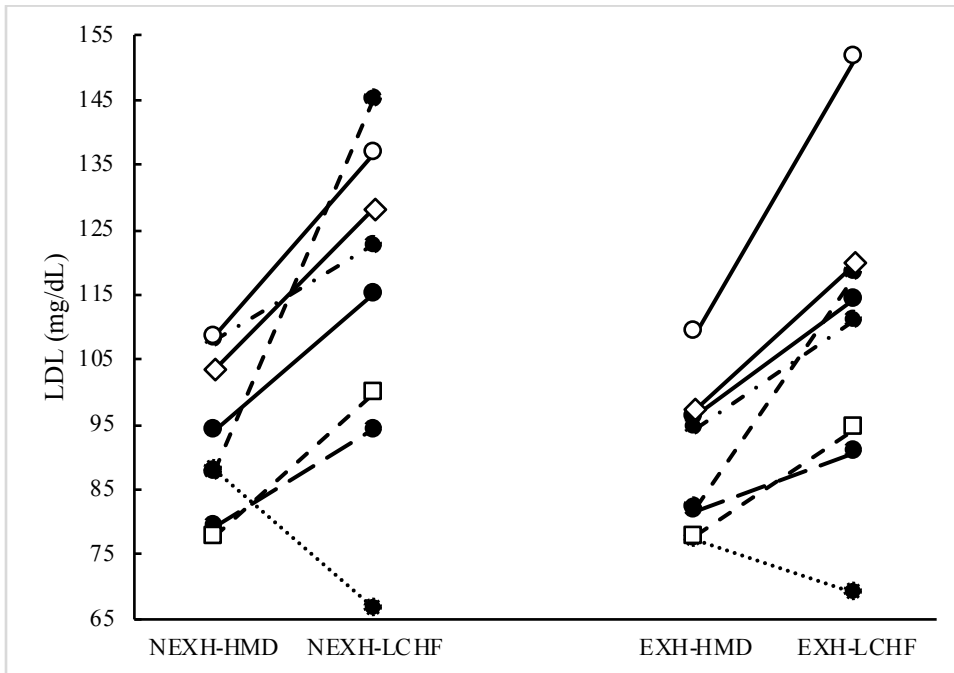


Figure 5. Individual fasted low-density lipoprotein responses after refraining from physical activity (Pre) and ~24 h following experimental exercise procedures (Post).

A main effect for treatment was also found for LDL-C ($p = 0.02$), however after post hoc adjustments only post-exercise LDL-C neared significance ($p = 0.06$). HDL-C exhibited a main effect for time ($p = 0.02$) but with no significance differences for HF or LCHF after post hoc adjustments. A main effect for treatment ($p = 0.004$) was found with higher HDL-C for LCHF versus HMD prior to running ($p = 0.01$) and nearing significance 24-h after exercise ($p = 0.07$) (Table 1). Participants exhibited highly favorable vLDL-C counts with vLDL-C only being detected in 2 of 32 samples (1 during pre-exercise HMD and one during post-exercise LCHF; different participants for each vLDL-C detection; Figure 6). Likewise, Lp(a) was not detected in nearly half of the samples collected, and when Lp(a) was found, a decrease was noted in Lp(a) for LCHF in 6 of the 8 comparisons (Figure 7).

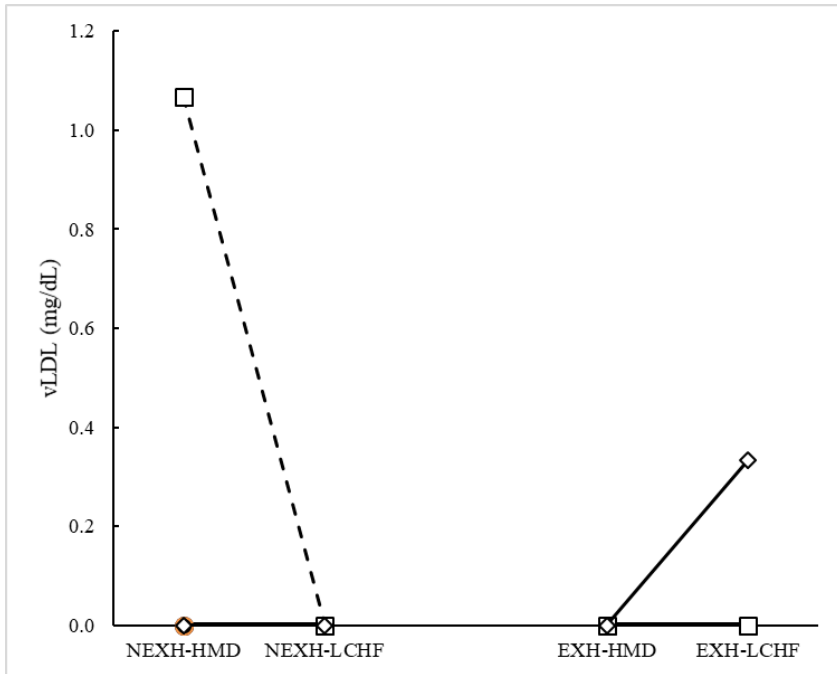


Figure 6. Individual fasted vLDL-C responses after refraining from physical activity (Pre) and ~24 h following experimental exercise procedures (Post).

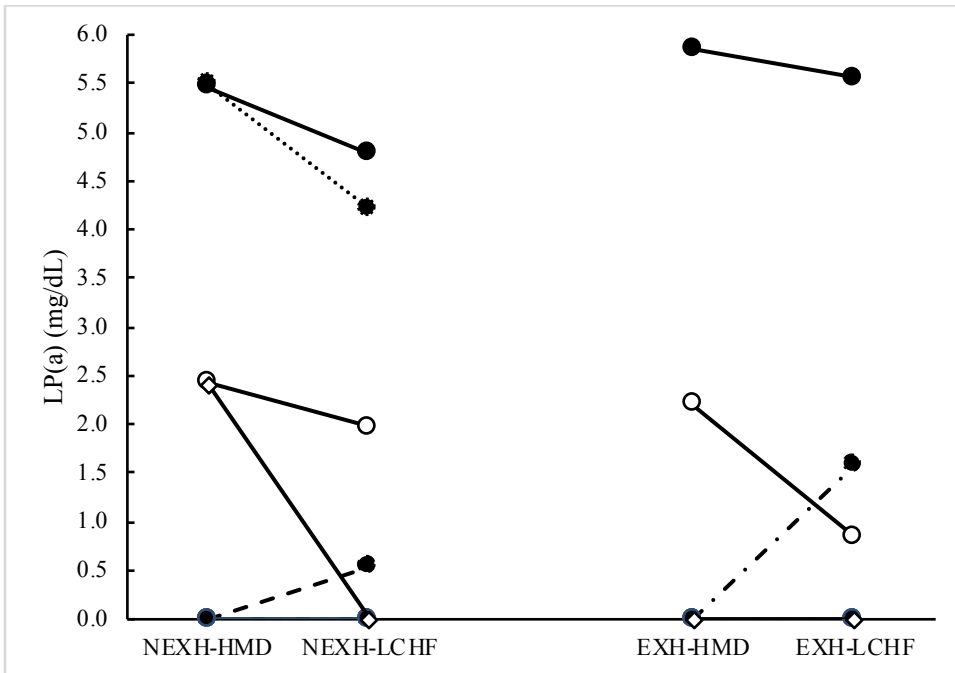


Figure 7. Individual fasted Lp(a) responses after refraining from physical activity (Pre) and ~24 h following experimental exercise procedures (Post).

DISCUSSION

The protection that exercise offers against cardiovascular disease is irrefutable (19), and is further confirmed by the desirable lipid and glucose profiles of the middle-aged runners in the current investigation (Table 1). However, intense marathon training may lead to increased risk of coronary artery calcification in older men versus risk factor matched controls (15). It has also recently been postulated that high carbohydrate diets suggested for endurance athletes could increase risks for multiple health outcomes associated with cardiometabolic diseases (16). Low carbohydrate, high fat diets have become increasingly popular in both the general and running population, but little evidence concerning LCHF on endurance athletes and responses of cardiovascular risk markers is available. The purpose of this study was to determine how switching from a runner's normal carbohydrate rich diet to a LCHF diet would impact lipid and glucose markers. The important findings of this study were (a) when participants were allowed to choose their own food items within the parameters of this study (daily goal of ~70% kcals from fat and <50 g of net carbohydrates/day) and eat to satiety, participants lost weight and improved body composition. This factor must be considered when interpreting the next two findings; (b) LCHF increased total cholesterol possibly due to both elevated LDL-C and HDL-C, but without evidence that vLDL-C or Lp(a) responded in a negative fashion to LCHF; (c) LCHF exhibited trends of no or favorable responses to fasting glucose and TG in nearly all participants; (d) regardless of diet, an acute aerobic EXH significantly and favorably altered the TG and TC/HDL-C profile of participants.

We are aware of two published studies in which non-elite, but regularly physically active adults have undergone 3 or more weeks of LCHF (12,27) and had lipid/glucose status assessed. The first (27) included 8 younger (28 ± 3 years) male mountain bikers with similar body mass, but lower body fat percentage that completed a 4-week isocaloric LCHF intervention. The second study (12) included a mixed gender sample ($n = 13$) with an average age of 31 when excluding one 60 year old participant. Seven participants were endurance athletes. The remaining participants were focused on sport or resistance training. Participants ate ad libitum on a LCHF that limited net carbohydrates to 20 g/day for 35-50 days. The current study supports both previous investigations in that LCHF can be expected to produce ~2.5-3% loss in body mass (Table 1) for the average physically active adult undertaking a LCHF diet (please see (Heatherly et al. 2018) for more detailed discussion concerning body mass/composition shifts). More germane to the current paper, lipid and glucose markers also responded similarly to these past studies supporting the notion that LCHF produce no apparent risk of increasing metabolic syndrome risk factors whether carbohydrate intake is severely restricted (12) or caloric intake is kept constant and only macronutrients are manipulated (29).

A unique aspect of this study compared to the previous investigations is that individual data are reported for each marker. While only approaching a main effect for diet type ($p = 0.07$), when analyzed by participant more striking tendencies are revealed (Figure 1). For example, during NEXH, LCHF seems to have little influence on blood glucose, but following EXH 5 participants had a decrease in fasting glucose of 13-21 mg/dL during LCHF. While only speculation, it is possible that after a rigorous training session, participants selected carbohydrate rich foods

during HMD that were not an option during LCHF or that blood glucose kinetics are altered following an acute longer duration running session. When examined cumulatively with past studies, it appears LCHF has minimal influence on fasting glucose responses in younger, physically active adults with ideal fasting glucose levels. However, older endurance athletes with less favorable glucose responses 24-h after training may potentially benefit from LCHF although further research is needed in this area. Given there was considerable inter-individual variation in carbohydrate intake between participants, it is important to note that our participants fell short (~4 g/kg of body mass) of the 6-12 g of carbohydrates per kg body mass range commonly promoted to endurance athletes by multiple nutrition related organizations (22). Athletes that consume higher relative carbohydrate content may exhibit different responses than the findings reported in this investigation.

With respect to cholesterol, TC and HDL-C were significantly higher following the LCHF intervention during the NEXH sessions and with no significant differences between LDL-C values (Table 1). Our findings agree with others (12,23,29) whom also found increases in TC due to an increase in resting HDL-C values. While only 1 participant (Figure 3) during the LCHF intervention for both NEXH and EXH exceeded the *desirable* category (> 200 mg/dL) (1), this trend of increase in TC alone is likely to be viewed negatively by an endurance athlete's primary care physician. Moreover, it should be noted that an increase in TC is to be expected partly due to increases in both HDL-C (Figure 4) and LDL-C (Figure 5). However, direct implications for the risk of coronary heart disease based on cholesterol count alone is limited if the data fail to differentiate between lipoprotein particle size, density, or count (11,21). Although evidence is limited, one study which did examine particle size during a LCHF intervention showed significant increases to peak LDL diameter and decreases in the number of small and ox-LDL particles (20). This is an important observation given that although the current study showed a non-significant increase in LDL count, potentially due to an increase in LDL particle size, the higher TC observed was not accompanied by raising either vLDL-C (Figure 6) or Lp(a) (Figure 7), both of which are strong predictors for future coronary heart disease risk and atherosclerosis (21). Furthermore, the increase in both TC and HDL-C following the LCHF diet would then explain the lack of differences observed between the TC/HDL-C ratios (3.1 vs. 3.1), which is independently a stronger and more sensitive index of cardiovascular disease risk than mere changes to TC (14). Based on past guidelines supporting HMD and limiting fat intake, it is likely many doctors or dieticians would be hesitant to promote LCHF for middle-age or older endurance athletes. However, the current study adds to the limited data that LCHF are not detrimental to lipid and glucose profiles of metabolically healthy, physically active adult male runners.

Finally, TG and TC/HDL-C decreased for both dietary treatments following EXH, but no effect was exhibited for diet type (Table 1). Similar to glucose responses, the lack of differentiation between dietary interventions may be due to the already favorable TG levels and TC/HDL-C ratios of the current participants and small sample size. Additionally, there is a strong negative correlation between pre-LCHF intervention TG and decreases in TG following LCHF (27). These findings have important clinical implications suggesting that a LCHF diet does not negatively alter TG profiles as often speculated by runners due to its higher fat content and further

strengthens the existing data that exercise provides a protective effect regarding elevated TG levels. To date, there is limited evidence available examining the relationship of lipid and TG changes following an acute aerobic exercise bout in trained male runners. The large body of evidence examining these kinematics have been performed in populations that were untrained and suffering from components of metabolic syndrome (4,5,8,9). However, one study conducted by Yu et al. (1999) did examine lipid and cholesterol changes pre and immediately post exercise in triathletes completing the Hawaii Ironman Triathlon (28). These findings demonstrated that an acute and prolonged bout of exercise significantly increases both HDL-C count and size, while also significantly decreasing TG count following exercise (124 vs. 96 mg/dL). The mechanisms which might explain these changes are detailed in a past study which examined cholesterol and lipid changes in physically active males during a treadmill protocol to exhaustion (6). These findings agree with Yu et al. (1999) which found elevated HDL-C and decreased TG levels 30 min post exercise. The authors postulate that the increase in exercise-induced elevations of HDL-C is due to an increase in the enzyme lecithin:cholesterol acyl transferase which promotes the formation of HDL-C. Additionally, the decrease in TG following exercise is most likely explained by an increase in utilization during and following exercise of free fatty acids and the increase in hormone sensitive lipase. Our study is unique though in that it offers additional insight into lipid and TG changes 24-h following aerobic exercise rather than immediately post as observed in the previous two studies. Therefore, the present study adds to the body of evidence suggesting that exercise, even if performed during a single bout and in trained male runners, can favorably alter lipid and TG responses and thus, offer protection against cardiometabolic diseases regardless of diet intervention.

In conclusion, the current investigation found LCHF results in 1) decreased body mass, 2) increases in TC, HDL-C, and LDL-C, but not TG, TC/HDL-C, vLDL-C, or Lp(a), 3) a trend of improved fasting glucose, particularly the morning after exercise in middle-aged endurance athletes, and 4) favorable improvement in TG and TC/HDL-C 24-h after exercise in trained male runners. The present study demonstrates that if a middle-age male runner undertakes a LCHF diet, overall there are no increased risks for developing cardiovascular disease based on the markers measured and additionally, aerobic exercise may further promote heart health as seen in 24-h reductions to TG and TC/HDL-C levels. The main limitations of the current study are the small sample size, limited observation period of 3 weeks, and that dietary intake was ad libitum and not controlled for caloric content or fat type. With the popularity of LCHF, future studies are warranted to further expand the knowledge concerning these knowledge gaps, including an increased emphasis on the female running population.

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