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Effects of Dietary Acid Load on Exercise Metabolism and Anaerobic Exercise Performance

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Abstract

Dietary acid load, quantified as the potential renal acid load (PRAL) of the diet, affects systemic pH and acid-base regulation. In a previous cross-sectional study, we reported that a low dietary PRAL (i.e. alkaline promoting diet) is associated with higher respiratory exchange ratio (RER) values during maximal exercise. The purpose of the present study was to confirm the previous findings with a short-term dietary intervention study. Additionally, we sought to determine if changes in PRAL affects submaximal exercise RER (as a reflection of substrate utilization) and anaerobic exercise performance. Subjects underwent a graded treadmill exercise test (GXT) to exhaustion and an anaerobic exercise performance test on two occasions, once after following a low-PRAL diet and on a separate occasion, after a high-PRAL diet. The diets were continued as long as needed to achieve an alkaline or acid fasted morning urine pH, respectively, with all being 4-9 days in duration. RER was measured during the GXT with indirect calorimetry. The anaerobic performance test was a running time-to-exhaustion test lasting 1-4 min. Maximal exercise RER was lower in the low-PRAL trial compared to the high-PRAL trial (1.10 ± 0.02 vs. 1.20 ± 0.05, p = 0.037). The low-PRAL diet also resulted in a 21% greater time to exhaustion during anaerobic exercise (2.56 ± 0.36 vs. 2.11 ± 0.31 sec, p = 0.044) and a strong tendency for lower RER values during submaximal exercise at 70% VO_2max (0.88 ± 0.02 vs. 0.96 ± 0.04, p = 0.060). Contrary to our expectations, a short-term low-PRAL (alkaline promoting) diet resulted in lower RER values during maximal-intensity exercise. However, the low-PRAL diet also increased anaerobic exercise time to exhaustion and appears to have shifted submaximal exercise substrate utilization to favor lipid oxidation and spare carbohydrate, both of which would be considered favorable effects in the context of exercise performance.

Key words: Renal acid load, alkaline diet, substrate oxidation, respiratory exchange ratio.

Introduction

Common variations in the composition of Western diets can alter systemic acid/base balance, as reflected by changes in blood pH of ~0.03 pH units, (Giannini et al., 1999; Yancy et al., 2007) and changes in urine pH of ~1.0 pH unit (Buclin et al., 2001). The effect of diet on systemic acid load can be quantified by calculating the potential renal acid load (PRAL) of the diet, which is based on dietary intakes of protein and mineral ions (Remer and Manz, 1995). In general, fruits and vegetables promote systemic alkalinity, while meat, grains, and cheese promote systemic acidity. We previously proposed (Niekamp et al., 2012) that a low-PRAL (alkaline promoting) diet increases circulating bicarbonate availability and as a result, increases the capacity to produce "non-metabolic" CO2 from the bicarbonate buffering system during maximal exercise. Because non-metabolic CO2 production is responsible for increasing the respiratory exchange ratio (RER) above the 1.00 during maximal intensity exercise (Robergs et al., 2004), this would be expected to increase maximal exercise RER. Indeed, in a cross-sectional study (Niekamp et al., 2012), we found that men and women who habitually consumed a low-PRAL diet had higher RER values at the end of a progressive incremental treadmill exercise test to exhaustion than those who were consuming a high-PRAL (acid promoting) diet (1.21 ± 0.01 vs. 1.15 ± 0.01 RER units, p < 0.05). Additionally, dietary PRAL and maximal exercise RER were inversely correlated (r = -0.43, p = 0.001), such that lower PRAL (more alkaline) diets were associated with higher maximal RER values (Niekamp et al., 2012). However, this earlier study was observational and the results need to be substantiated with intervention studies.

If dietary acid load does affect acid-base regulation and serum bicarbonate levels during exercise, it is also possible that dietary PRAL could affect the development of acidosis during high-intensity exercise and consequently alter high-intensity exercise performance (for comprehensive review on the effects of acidosis on muscle fatigue, see reference (Cairns, 2006)). More specifically, a low-PRAL diet would be expected to improve anaerobic exercise performance by inducing alkalosis (higher blood pH) and increasing bicarbonate availability. Such effects have been well documented in response to sodium bicarbonate loading (Carr et al., 2011a; McNaughton et al., 2008; Peart et al., 2012). However, to our knowledge, no studies have evaluated the effect of broad dietary patterns, such as high- and low-PRAL diets, for ergogenic effects during high-intensity anaerobic exercise.

The purpose of the present study was to perform an intervention study to confirm and expand on our previous cross-sectional findings (Niekamp et al., 2012). The primary aim was to evaluate the hypothesis that a short term (4-9 days) low-PRAL diet results in a higher respiratory exchange ratio during maximal exercise as compared

to that after a high-PRAL diet. A second aim of the present study was to determine if a low-PRAL diet improves anaerobic exercise performance. Finally, as an exploratory aim, we sought to determine if dietary PRAL affects respiratory exchange ratio during submaximal exercise.

Methods

Participants and screening

Men and women (n = 10), aged 18-60 years were recruited from the Saint Louis metropolitan area. Volunteers completed a medical history and medications questionnaire, which was used along with criteria from the American College of Sports Medicine, to classify each individual as low, moderate, or high risk for medical complications during exercise (American College of Sports Medicine, 2014). Volunteers were excluded if they were at moderate or high risk for medical complications. Both trained and untrained individuals were eligible to participate, provided that they were willing to undergo maximal exercise testing and undergo substantial dietary alterations for several days with the goal of changing dietary PRAL. The study was reviewed and approved by Saint Louis University Institutional Review Board and all participants provided informed written consent to participate in the study.

Study design

The study was a cross-over trial in which participants underwent exercise testing on two occasions, once after following a low-PRAL diet and once after following a high-PRAL diet. The intervention sequence was randomized and counterbalanced such that half of the participants underwent the low-PRAL intervention first and the others underwent the high-PRAL intervention first.

Dietary interventions

For each of the dietary interventions, the study dietitian provided the subjects with specific instructions on how to modify their habitual diets to achieve a low- or high-PRAL diet. The study dietitian was in contact with the participants (via telephone or email) every day during the dietary interventions to encourage compliance and to provide specific food suggestions as needed. The general strategy used for the low-PRAL diet was to increase the consumption of alkaline-promoting foods such as fruits and vegetables and to reduce the consumption of acid-promoting foods such as meats, cheeses, and grains (Welch et al., 2008). More specifically, participants were instructed to consume 6-8 cups of vegetables and >4 servings of fruit each day. Because there is a tendency for lower energy intake with diets that are rich in fruits and vegetables, such as the low-PRAL diet, participants were instructed to eat frequently and consume energy dense foods during the low-PRAL trial, such as starchy vegetables (e.g. sweet potatoes), dried fruits (e.g. dates and raisins), and plant sources of fat (e.g. avocado, coconut, nuts, seeds). Foods with moderate PRAL values (e.g. legumes, yogurt, egg whites, quinoa) were allowed and were used to ensure that energy and macronutrient intakes were adequate. The participants were also advised to minimize the consumption of all meats, cheeses and common grains (most of which are high-PRAL) during the low-PRAL diet. During the high-PRAL diet, participants were instructed to consume at least 3-4 servings of common grains (e.g. wheat, corn, and oats), 3 servings of meat, and 3 servings of cheese (especially hard cheeses such as parmesan) each day while minimizing the intakes of fruits and vegetables. Moderate PRAL foods were allowed as desired as long as it did not displace high-PRAL foods from the diet. In general, the high-PRAL diet required less intensive counseling from the dietitian because it closely resembled the baseline diet of the participants.

The goal during the low PRAL diet was to achieve a dietary PRAL of ≤-1 mEq/d and during the high-PRAL diet the goal was PRAL ≥15 mEq/d; these cut points were based on PRAL values of the high and low PRAL tertiles that were observed in our previous cross-sectional study on 57 middle-aged men and women (Niekamp et al., 2012). Food portions were adjusted as needed during both dietary interventions to maintain energy balance as evidenced by body weight changes. The diets were a minimum of 4 days in duration but were maintained as long as needed to achieve a fasted morning urine pH of ≥7.0 in the low-PRAL trial and ≤6.0 in the high-PRAL trial, as described below. Participants were instructed to avoid antacid supplements and medications, which have their own effects on systemic acid/base status.

Dietary assessment and calculation of PRAL

Participants recorded 3-day food diaries at baseline and during the dietary interventions. Nutrient analysis of the diaries was performed with Food Processor SQL software (version 10.6.0 ESHA Research, Salem, OR). Dietary PRAL was calculated based on the following equation:

$$PRAL = P \times 0.0366 + Pro \times 0.4888 - [K \times 0.0205 + Ca \times 0.0125 + Mg \times 0.0263]$$

Where the units for PRAL are mEq/d and P is phosphorous in mg/d, Pro is protein in g/d, K is potassium in mg/d, Ca is calcium in mg/d, and Mg is magnesium in mg/d (Remer & Manz, 1995).

Urinary assessment

Urine pH was measured to the nearest 0.5 pH unit with pH strips (pH 5.0-10.0, catalog #9588, ColorpHast, EMD Chemicals, Inc., Gibbstown, NJ) and used to inform decisions about the duration of the diets and to confirm that the diets adequately altered dietary acid load. The goal during the low-PRAL intervention was to achieve a urine pH of ≥7.0 and during the high-PRAL intervention the goal was ≤6.0 (Welch et al., 2008). Fasted morning urine pH was self-monitored by the participants every morning during the intervention; if urine pH was not within the desired range, subjects were asked to follow the dietary intervention for additional days as needed and to achieve the urine pH goals. On the morning of exercise testing, urine pH was measured by the investigators to confirm that the pH goal had been attained.

Graded exercise testing

Indirect calorimetry (MedGraphics CardioO$_2$; Medical

Graphics Corporation, St. Paul, MN) was performed during a graded treadmill exercise test (GXT) to exhaustion. Prior to each test, a 3-liter air syringe (Hans Rudolph, Inc., Shawnee, KS) was used to generate a series of flow profiles for calibrating the pneumotach flow meter on the calorimeter. The carbon dioxide and oxygen analyzers were calibrated prior to each test with medical grade gases of known carbon dioxide and oxygen concentrations. The GXT was initiated at a speed determined during warm-up to increase HR to ~70% of age-predicted maximal heart rate (HRmax) and a grade of 0%. Thereafter, the grade was increased by 2 percentage points every 2 minutes until the subject could no longer continue due to fatigue. For each participant's second test, the treadmill test protocol was replicated and if needed, additional stages were added to the end of the test until the subject could no longer continue due to fatigue. The protocol did not include a familiarization test. Peak VO_2 was considered a "true VO2max" if the following criteria were met: (1) measured HRmax $\geq$ age predicted HRmax minus 10 beats/min, and (2) rating of perceived exertion $\geq$19 on the 6-20 point Borg scale. Age predicted HRmax was calculated as 208-0.7 x age (yr) (Tanaka et al., 2001). All subjects met these criteria during both tests. A maximal RER criterion was not included in this assessment because maximal exercise RER was a study outcome.

Submaximal RER values were determined by establishing a linear regression equation for each test that described the relationship between oxygen uptake and RER. Metabolic data from early in the test, when RER values temporarily decline, and during a VO_2 plateau, if present, were not used in the development of the regression equations. The subject-specific equations were then used to determine RER values at oxygen uptakes equivalent to 70%, 80%, and 90% of VO2max.

Oxygen pulse was calculated as absolute oxygen uptake divided by HR and was used as an index of left ventricular stroke volume (Whipp et al., 1996). As a crude retrospective estimate of work efficiency, oxygen consumption was evaluated for each subject at a stage during the graded exercise test that coincided with ~85% of VO_2max. For each subject, the same stage was used for the low- and high-PRAL trials. In accordance with published equations (American College of Sports Medicine, 2014), the VO_2 data were normalized to percentages of predicted VO_2 based on the treadmill speed and grade.

Anaerobic performance testing

After completing the graded exercise test, the participants underwent 10 min of seated rest and then performed the anaerobic exercise performance test. Performance was measured as time-to-exhaustion while running on a treadmill with the speed set at the same speed used during the graded exercise test and a treadmill grade that was 2 percentage points steeper than that achieved during the last full stage of the graded exercise test.

Statistical analyses

Paired t-tests were used for means comparisons. Pearson correlation analyses were used to evaluate bivariate associations. Statistical analyses were performed with SPSS Statistics software (version 20, IBM Corporation, Armonk, New York). $P \leq 0.05$ was considered significant. Data are presented as means $\pm$ standard errors. An a priori power analysis was used to determine the smallest detectible effect size of high and low PRAL diets on RERmax. Based on an alpha of 0.05, a desired statistical power of 0.80, n=10 in a repeated measures design, and a standard deviation of repeated RERmax measures of 0.04 (based on our previous research (Niekamp et al., 2012)), it was expected that a treatment effect of $\geq$0.04 RERmax units would be detectable.

Results

Subjects

All 10 of the participants who were enrolled in the study complete all aspects of the study and provided evaluable data. Six (60%) of the participants were women and 3 (30%) were exercise trained (exercising $\geq$ 5 hours per week). All subjects were in the normal range for BMI (22.0 $\pm$ 0.86). Average age was 42 $\pm$ 5 yr (range: 19-60 yr). Baseline PRAL was 8.6 $\pm$ 5.7 mEq/day, which is greater than those reported for European adults (-4 to -7 mEq/d) (Welch et al., 2008) and similar to, or slightly greater than those reported for individuals in the United States (2-8 mEq/d) (Remer et al.,, 2003; Thorpe et al., 2008).

Adherence to dietary interventions: PRAL, nutrient intakes, and urine pH

During the dietary interventions, PRAL was 33 $\pm$ 8 mEq/day in the high PRAL intervention and -21 $\pm$ 4 mEq/day in the low PRAL intervention (Table 1; p < 0.001 between trials). Dietary intakes of protein, phosphorous, potassium, and calcium (all of which are used in the calculation of PRAL) differed between treatment conditions (Table 1); however, magnesium intake (also used in PRAL calculations) did not differ between trials. Total energy intake, carbohydrate intake, and fat intake did not differ between trials (Table 1).Total energy intake was not different from the estimated energy requirement (EER) for weight maintenance in physically active individuals (Institute of Medicine, 2002) (low-PRAL: 97 $\pm$ 7% of EER, p = 0.441; high-PRAL: 113 $\pm$ 11% of EER, p = 0.194). Carbohydrate intake exceeded the recommended dietary allowance (RDA) of 130 g/day (Institute of Medicine, 2002) in the low-PRAL (245 $\pm$ 20% of RDA, p > 0.0001) and high-PRAL trials (272 $\pm$ 49% of RDA, p = 0.004). Furthermore, carbohydrate intake did not differ significantly from the 5 g/kg/day minimum recommended carbohydrate intake to prevent glycogen depletion in athletes (American Dietetic Association et al., 2009) (low-PRAL: 101 $\pm$ 12% of recommendation, p = 0.467; high-PRAL: 105 $\pm$ 14% of recommendation, p=0.266) All participants achieved the urine pH goals for both interventions; accordingly, urine pH on the testing day (i.e. last day of dietary intervention) for the two diets differed significantly (Figure 1; 7.1 $\pm$ 0.1vs. 5.8 $\pm$ 0.1; p $\leq$ 0.0001). Participants followed the high PRAL diet for 4.3 $\pm$ 0.1 days before achieving the urine pH goal of $\leq$6.0, whereas 6.8 $\pm$ 0.4 days were required to achieve the urine pH goal of $\geq$7.0 in the low PRAL intervention.

Table 1. Body weight, PRAL and nutrient intakes during the high and low PRAL interventions. Values are means (± SE).

	Low PRAL Trial	High PRAL Trial
Body Weight, kg	66.5 (4.8)	66.2 (4.7)
PRAL, mEq/d	-21 (4)	33 (8) ***
Protein, g/d	60 (4)	110 (17) *
Phosphorous, mg/d	608 (97)	1062 (128) **
Potassium, mg/d	2829 (316)	1904 (201) *
Calcium, mg/d	556 (40)	945 (145) *
Magnesium, mg/d	235 (38)	232 (27)
Total energy, kcal/d	2208 (138)	2664 (337)
Carbohydrate, g/d	319 (25)	353 (63)
Carbohydrate, % of energy	58 (3)	52 (3)
Carbohydrate, g/kg body weight	5.3 (.7)	5.1 (.6)
Total fat, g/d	71 (9)	93 (12)
Total fat, % of energy	29 (3)	32 (2)
Saturated fat, g/d	19 (7)	30 (4)
Fiber, g/d	35 (3)	26 (2) *
Sodium, mg/d	3090 (440)	3842 (560)

*, ** and *** denote $p < 0.05$, 0.001 and 0.001 respectively from paired t-tests. PRAL, potential renal acid load. Nutrient intakes are based on nutrient analysis from 4-day food diaries.
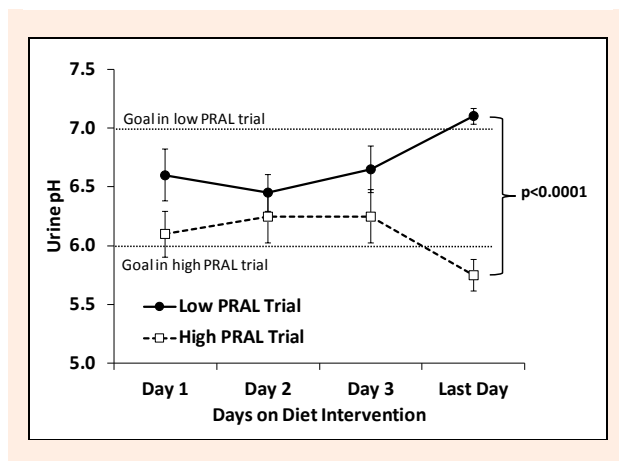

Figure 1. Fasted morning urine pH during the dietary intervention for the low- and high-PRAL interventions. The objective was to attain the pH goal in 4 days; however, up to 9 days were required for some participants. "Last day" indicates urine pH on the last day of the dietary intervention (i.e. 4 to 9 days), which was also the morning during which outcomes assessments were performed. Values are means±SE. Paired t-tests were used for means comparisons. PRAL, potential renal acid load.

Respiratory exchange ratio

Maximal exercise respiratory exchange ratio was greater during the high PRAL treatment as compared to the low PRAL treatment (Figure 2). Furthermore, during all submaximal intensities, there were tendencies (p-values of 0.059 to 0.077) for higher RER values in the high PRAL condition (Figure 2). The differences in RER between trials were not correlated with age for any of the exercise intensities (all $p \geq 0.317$).

Maximal oxygen uptake, heart rate, and work efficiency

Both tests for all subjects met the criteria for "true" VO_2max. There were no differences between trials for VO_2max or maximal exercise VCO_2 (Figure 3). HRmax was 97 ± 2% of age-predicted maximum in the high PRAL trial and 98 ± 1% in the low PRAL trial, with no differences between trials (Figure 3). Mean oxygen pulse

was 14.9 ± 1.2 mL/beat in the low-PRAL trial and 14.2 ± 1.1 mL/beat in the high-PRAL trial; these means were not statistically different (p = 0.153). Submaximal exercise gross work efficiency did not differ (p = 0.481) between the low-PRAL trial (97 ± 5% of predicted VO_2) and the high-PRAL trial (95 ± 7% of predicted VO_2).
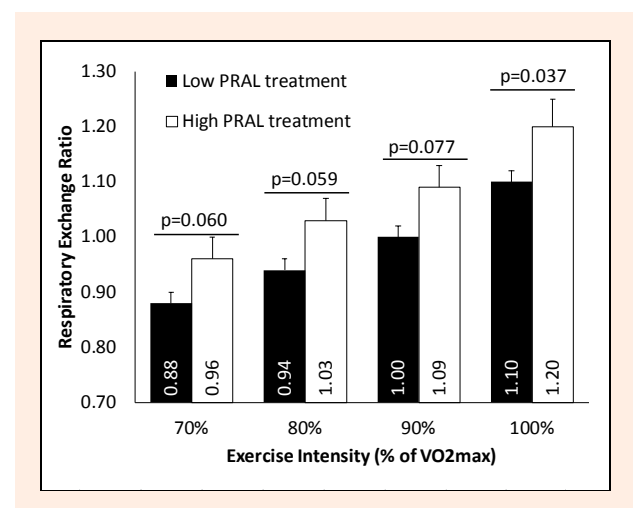

Figure 2. Respiratory gas exchange ratio (VCO_2 production/VO_2 consumption) during submaximal and maximal-intensity exercise during the graded exercise test. Values are means±SE. Paired t-tests were used for means comparisons.

Exercise performance

Time to exhaustion during the anaerobic exercise performance test was 21% greater (p = 0.044) in the low PRAL trial as compared to the high PRAL trial (Figure 3). There was a non-significant tendency for association between the magnitude of difference in PRAL between trials and the magnitude of difference in anaerobic exercise time to exhaustion between trials (r = −0.58, p = 0.082), such that greater reductions in PRAL between the high and low PRAL trials were associated with greater increases in anaerobic exercise performance. Based on linear regression, this translates to a 10 unit greater reduction in dietary PRAL being associated with a 6 sec (~5%) greater increase in time to exhaustion. Time-to-exhaustion during

the graded exercise test did not differ between trials (Figure 3; p = 0.120). The difference in exercise performance time between trials was not correlated with age for either of the time-to-exhaustion tests (all p ≥ 0.512).
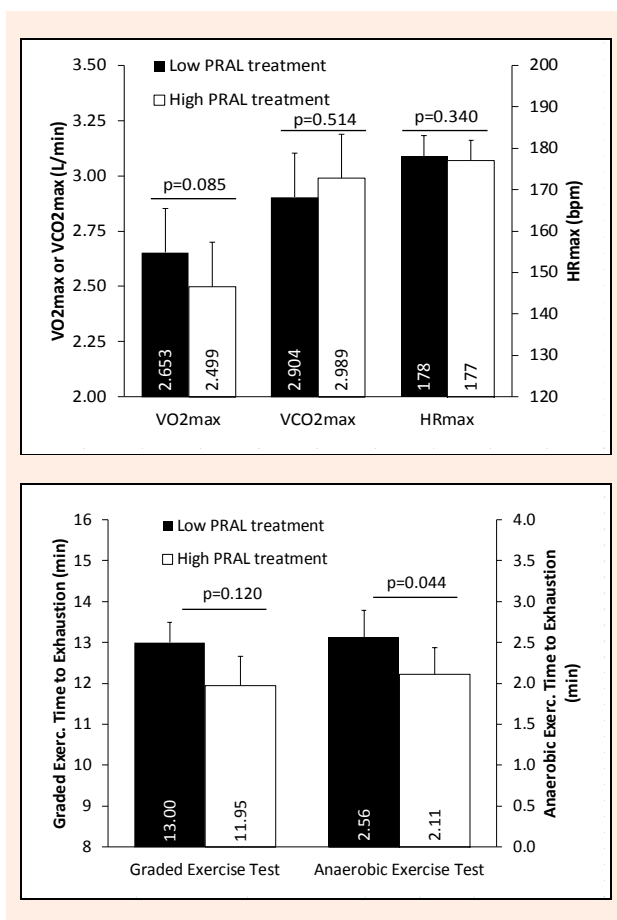

Figure 3. Oxygen consumption, carbon dioxide production, and heart rate during maximal exertion at the end of the graded treadmill exercise test to exhaustion (top panel) and exercise performance time-to-exhaustion during the graded exercise test and anaerobic exercise test (bottom panel). Values are means±SE. Paired t-tests were used for means comparisons.

Discussion

Based on our previous study (Niekamp et al., 2012), we proposed that, when compared to an acid promoting (high-PRAL) diet, a short-term alkaline promoting (low-PRAL) diet increases systemic pH and increases maximal exercise respiratory exchange ratio, which is heavily dependent on bicarbonate buffering of exercise-induced acid production. As expected, the low-PRAL diet increased fasted morning urine pH, suggesting systemic alkalinity. However, contrary to our hypothesis, the low-PRAL diet resulted in a lower RER_{max}, not higher. Based on the aforementioned premise that a low-PRAL diet increases systemic alkalinity, and based on evidence that alkalizing agents such as sodium bicarbonate increase anaerobic exercise performance (reviewed in (Cairns, 2006)), we also hypothesized that a low-PRAL diet increases anaerobic exercise performance. Indeed, time-to-exhaustion during high-intensity treadmill running (lasting ~1-4 min) was 21% greater after consuming a low-

PRAL diet as compared to a high-PRAL diet, indicating that anaerobic exercise performance was augmented.

It is not clear why the short-term, low-PRAL dietary intervention in the present study resulted in a lower RER_{max}, while in our previous cross-sectional study (Niekamp et al., 2012), a habitual low-PRAL diet was associated with higher RER_{max} values; however, some issues warrant consideration. First, it is possible that the short-term dietary intervention was not sufficient to alter serum bicarbonate levels and thus wouldn't be expected to affect non-metabolic CO_2 production and RER_{max}. Although we did not measure serum bicarbonate or related measures such as pH or CO_2 (and future studies should include such measures), bicarbonate levels are closely and inversely associated with urine pH (Unwin and Capasso, 2001). Based on this relationship and the urine pH data from the present study, serum bicarbonate would be predicted to be ~12% lower in the high-PRAL condition than in the low PRAL trial (Unwin and Capasso, 2001). Furthermore, while a lack of change in serum bicarbonate levels might explain a lack of effect on RER_{max}, it does not explain why short-term changes in dietary PRAL yielded opposite effects to those from our previous cross-sectional study. A more likely explanation relates to the effects of chronic versus acute alterations in dietary acid load. In the previous cross-sectional study, participants were categorized into tertiles of dietary PRAL based on their habitual dietary intakes, which had presumably been consumed for months or even years. In contrast, the present study involved a short-term intervention, which based on urine pH, did not alter systemic pH until the day of testing. Clearly, future studies are needed to evaluate the time-course of responses to changes in dietary PRAL.

A major and novel finding form the present study is that anaerobic exercise performance was enhanced by the low-PRAL diet. Furthermore, despite lower maximal RER values after the low-PRAL diet, there were tendencies for a longer time-to-exhaustion during the GXT (p = 0.12) and greater VO_2max (p = 0.08). It seems likely that the augmented anaerobic exercise performance could have resulted from an alkaline environment created by the consumption of low PRAL foods, and possibly by an increase in bicarbonate availability. Indeed, it generally accepted that bicarbonate loading improves anaerobic exercise performance by enhancing acid buffering capacity (Carr et al., et al., 2011b; McNaughton et al., 2008; Peart et al., 2012). However, as we and others have demonstrated, a major limitation to bicarbonate loading is that it frequently causes severe gastrointestinal distress (Cameron et al., 2010; Carr et al., 2011a; Kahle et al., 2013; Siegler et al., 2012); furthermore, standard ergogenic doses (300 mg/kg) result in sodium doses of 4-9 grams, which is well above the recommended upper limit of 2.3 g/d (Institute of Medicine, 2005). Therefore, a low-PRAL diet, which focuses on high intakes of fruits and vegetables and has the added benefits of being rich in phytochemicals, fiber, antioxidants, and other nutrients, may be an attractive alternative to bicarbonate loading for improving anaerobic exercise performance.

Another novel finding was the strong tendency for lower submaximal exercise RER values during the low-

PRAL diet, suggesting that the low-PRAL diet altered substrate utilization to favor greater lipid oxidation and lower carbohydrate oxidation. Based on published equations (Ferrannini, 1988) and RER data from the 70% VO$_2$max submaximal intensity, lipid oxidation was higher in the low-PRAL trial than in the high-PRAL trial (37% vs. 12% of total energy expenditure) while carbohydrate oxidation was lower in the low-PRAL trial (66 vs. 96% of total energy expenditure). While this apparent carbohydrate-sparing effect of a low-PRAL diet might have implications for delaying glycogen depletion during long-duration endurance exercise, the results should be considered preliminary, as the exercise and indirect calorimetry protocols were not optimized for the evaluation of substrate utilization during steady-state, submaximal exercise. We are currently conducting a follow-up study to confirm these findings. One possible explanation for the shift in substrate utilization might be that the low-PRAL diet, because it was lacking in grains (i.e. a rich source of carbohydrate), was carbohydrate deplete and as a consequence, low glycogen levels caused the low carbohydrate oxidation rates (Starling et al., 1997). However, subjects were encouraged to eat substantial quantities of carbohydrate-rich fruits during the low-PRAL diet and thus there was no difference in carbohydrate intake between trials. Furthermore, total energy intake and carbohydrate intake met or exceeded recommended intakes in both trials, making it unlikely that energy or carbohydrate deficiency altered substrate utilization rates. Another possibility is that the shift in systemic pH altered the activity of enzymes that regulate lipid and carbohydrate oxidation. In support of this possibility, the activity of carnitine acyl transferase-I, one of the rate limiting enzymes in lipid oxidation, is highly pH-sensitive within the physiologic range (pH 6.8-7.0) (Bezaire, Heigenhauser, & Spriet, 2004) in a manner that would favor lipid oxidation during a more alkaline state.

A study limitation is that the broad dietary changes that were needed to alter PRAL also altered other dietary factors that might have had their own effects on the study outcomes. One such factor is dietary nitrates, which acutely improve exercise performance (Lansley et al., 2011; Larsen et al., 2007; 2010; Murphy et al., 2012). Because vegetables are nitrate-rich (Hord et al., 2009), and because large quantities of vegetables were consumed during the low-PRAL diet, it is possible that nitrates might have enhanced exercise performance during the low-PRAL trial. However, this scenario does not seem likely because nitrates improve exercise performance by increasing work efficiency (i.e. less oxygen cost to perform exercise) (Vanhatalo et al., 2010) and we did not observe differences in efficiency between trials. It is also possible that the greater protein intake in the high-PRAL diet may have contributed to the lower fat oxidation rates observed in the low-PRAL trial; however, while studies have demonstrated that acute ingestion of dietary protein suppresses postprandial fat oxidation (Benton & Swan, 2007), fasting lipid oxidation is not altered by a long-term high-protein diet and actually reduces carbohydrate oxidation (Linn et al., 2000), which is opposite to that observed in the present study. The large age range included

in the present study (19-60 yr) might also be viewed as a limitation; however, this also makes the results generalizable to a larger population. Furthermore, based on the lack of correlation between age and the main study outcomes, the responses to low and high-PRAL diets appears to be relatively uniform across the wide age range, making the results applicable to young and older men and women.

Practical implications of the present study are that the dietary manipulation of PRAL might benefit athletes. Events heavily dependent on anaerobic metabolism, where performance is often limited due to acidosis, would be expected to improve. These would be events lasting 1-5 minutes such as 100-200 meter swimming or 400 – 800 meter running events. Additionally, based on the preliminary evidence of lower RER values during submaximal exercise, a low-PRAL diet may have a glycogen sparing effect by decreasing carbohydrate oxidation and increasing lipid oxidation, and thus might be beneficial for long-duration exercise performance, in which glycogen depletion is of concern (e.g. marathon running, long-distance bicycle races, etc.). In order to achieve PRAL levels that are similar to those used in this study, high consumption of fruits and vegetables should be encouraged while minimizing consumption of meats and grains. For competitive athletes, a high-carbohydrate diet can be maintained without a grain-rich diet by consuming carbohydrate-rich fruits and vegetables such as fresh and dried fruits, fruit juices, and potatoes for at least 4-7 days before competition. Anecdotally, and based on the ~50% longer time required to achieve the urine pH goal, it was more difficult for participants to adhere to the low-PRAL diet. This is not surprising in light of the acidic nature of the subjects' diet at baseline (i.e. less dietary change was required). However, because the low-PRAL diet is rich in fruits and vegetables, and deplete of many unhealthy foods such as cheese, fatty meats, and refined grains, a low-PRAL diet not only provides exercise performance benefits, but might also reduce chronic disease risk.

Conclusion

In conclusion, short-term changes in diet to decrease systemic acid load does not increase maximal exercise respiratory exchange ratio and may even reduce it. This is in contrast to our previous study, which showed that long-term consumption an alkaline diet was associated with higher maximal RER values. The present study also showed that an alkaline-promoting diet (i.e. rich in fruits and vegetables and low in meat and grains) improves anaerobic exercise performance by 21%. This finding has obvious implication for athletes. However, it might also be relevant for individuals who have limited ability to perform physical activities of daily living, such as elderly frail individuals and patients with heart failure. Future studies, ideally with larger sample sizes, are needed to evaluate the effects of low- and high-PRAL diets on other populations. Furthermore, additional studies are needed to compare the metabolic effects of long- and short-term changes in PRAL and to elucidate the mechanisms by which such diets alter substrate utilization and exercise

performance.

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Key points

- Short-term (4-9 days) changes in the acid or alkaline promoting qualities of the diet, quantified as potential renal acid load (PRAL), alter systemic pH, as evidenced in the present study by changes in fasted morning urine pH. Low-PRAL (alkaline promoting) diets are characterized by high intakes of vegetables and fruits with limited consumption of meats, cheeses, and grains while high-PRAL diets are characterized by the opposite dietary pattern.
- An alkaline promoting (low-PRAL) diet increases anaerobic exercise performance, as evidenced by greater time-to-exhaustion during high-intensity treadmill running.
- Preliminary evidence suggests that an alkaline promoting (low-PRAL) diet increases lipid oxidation and may have a carbohydrate-sparing effect during submaximal endurance exercise, although further studies are needed.
- In contrast to what has been observed in response to habitual/long-term dietary patterns, a short-term low-PRAL diet does not increase maximal exercise respiratory exchange ratio and even appears to lower it. This suggests that short-term and long-term alterations in PRAL have different physiologic effects on this parameter.

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