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Effects of a Mineral Antioxidant Complex on Clinical Safety, Body Water, Lactate Response, and Aerobic Performance in Response to Exhaustive Exercise

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Background: This investigation examined the safety and efficacy of a silica-based mineral antioxidant complex (MAC) that has been suggested to influence body water and buffer lactate. ***Methods:*** In a double-blind, randomized crossover design, male participants completed testing for 3 conditions: water only (baseline), rice flour (placebo), and MAC supplementation. Participants visited the laboratory on 5 occasions: familiarization, baseline, Testing Day 1, washout, and Testing Day 2. Baseline and Testing Days 1 and 2 consisted of fasting blood, pre- to postexercise body-water assessment and determination of VO_{2peak} on a cycle ergometer. The supplementation protocols were separated by 1 wk and balanced to minimize an order effect. ***Results:*** No differences between conditions were found for heart rate, blood pressure, or serum-safety markers ($p > .05$). Before exercise there were no differences between conditions for total body water (TBW), intracellular water (ICW), or extracellular water (ECW). No significant interactive effects for supplementation and exercise were found for TBW, ICW, or ECW ($p > .05$). A time effect for TBW ($p < .01$) and ICW ($p < .001$) was present. Within-group changes in TBW occurred in the MAC condition, and within-group changes for ICW occurred in the MAC and placebo conditions. Ratings of perceived exertion and blood lactate increased ($p < .05$) with exercise. No significant effects were found for performance variables. ***Conclusions:*** MAC supplementation had no impact on aerobic exercise performance and lactate response. Increases in TBW and ICW occurred after MAC consumption, but these changes appeared to have minimal physiological impact.

Keywords: buffer, silica, supplementation

Colloidal silicate gained popularity when it was reported that the Hunzukuts of West Pakistan tended to live healthier and longer lives than world averages (Keller, 1978; Leaf, 1973; Murray & Murray, 1984; Purdy Lloyd, Wasmund, Smith, & Raven, 2001). The enhanced health and longevity of people in this region have been partially attributed to their consumption of glacial milk (Keller, 1978), which has been found to contain colloidal silicate minerals that have potentially health-promoting qualities primarily involving the formation of water around silica molecules, creating a silica–water interface. The silica–water interface forms a hydrated surface that may promote favorable intracellular effects and absorb elements and compounds including iron, potassium, magnesium, lithium, calcium, and hydrogen (Dove & Rimstidt, 1994; Keller, 1958; Purdy Lloyd et al., 2001). Because of the possible health-promoting effects of silica hydride, commercial production has attempted to manufacture a silicate analog in dietary supplements. To this end, commercially produced silicate particles ranging in diameter from 50 to 100 Å in combination with additional metabolic and antioxidant minerals termed mineral antioxidant complexes (MACs) have been manufactured (Purdy Lloyd et al., 2001). The interface formed by the MAC is stabilized by water and can become saturated with reduced hydrogen ions (H^-), which can serve an important antioxidant role (Dove & Rimstidt, 1994), while also operating as a hydrogen-ion (H^+) buffer. The interaction between the MAC and reduced hydrogen forms the theoretical basis for how supplementing with a silica-based MAC can work to reduce blood lactate and improve body-water or hydration status surrounding exercise (Goldfarb, Bloomer, & McKenzie, 2004; Purdy Lloyd et al., 2001).

At the current time, no study has investigated the ability of a silica-based MAC to affect changes in body water. Silica has been shown to form a chemical interface with water, creating a chemically favorable scenario to improve the ability of the cellular milieu to improve hydration and body-water status, but clinical evidence has yet to be reported. Favorable improvements in body-water status as a result of MAC supplementation could

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have important implications not only for recreationally active individuals but also for clinical populations in which cellular hydration is compromised and presents a physiological challenge. As mentioned previously, evidence of the silica–water interface can be seen through an increased ability to buffer changes in hydrogen-ion status and metabolic accumulation of lactate. In this respect, previous investigations have suggested that MAC supplementation may affect changes in lactate accumulation and therefore affect prolonged exercise (Purdy Lloyd et al., 2001).

High-intensity exercise elicits the rapid formation and accumulation of intramuscular hydrogen ions and increased concentration of lactate. This response is thought to disrupt the homeostatic cellular environment by reducing intracellular pH levels, leading to decreased exercise capacity because of disruptions in transport mechanics, substrate availability, or rate-limiting enzymes' kinetics (Baker, Brandes, & Weiner, 1995; Favero, Zable, Bowman, Thompson, & Abramson, 1995; Fitts & Holloszy, 1976; Spriet, Lindinger, McKelvie, Heigenhauser, & Jones, 1989). Although the contribution of hydrogen ions to fatigue is somewhat controversial (Westerblad, Allen, & Lannergren, 2002), the buffering of hydrogen ions by various systems has been thought to improve exercise performance by restoring intracellular pH levels closer to homeostatic values (Nevill, Boobis, Brooks, & Williams, 1989). In support of this effect, previous investigations have explored the effects of MAC consumption on exercise performance (Glazier, Stellingwerff, & Spriet, 2004; Purdy Lloyd et al., 2001) and lactate buffering (Goldfarb et al., 2004; Purdy Lloyd et al., 2001), providing preliminary evidence of a potential reducing effect and intracellular silica–water interface. The first investigation used a double-blind, randomized, placebo-controlled design in which 6 trained cyclists performed a 40-km bicycling time trial. Blood lactate concentrations were assessed 5 min before the start of exercise and 5 min after the time trial. The authors found MAC supplementation to have no effect on time-trial performance, but blood lactate was significantly reduced 5 min postexercise when participants consumed the MAC compared with placebo (Purdy Lloyd et al., 2001). A later investigation with a similar protocol found MAC supplementation to have no effect on time-trial performance, blood lactate, or energy substrate utilization in highly trained male cyclists (65.3 ± 1.2 ml $\cdot$ kg^{-1} $\cdot$ min^{-1}; Glazier et al., 2004). Furthermore, 42 Sprague-Dawley rats were used to examine the effects of a MAC on postexercise blood lactate concentrations. The rats were randomly assigned to one of three groups: MAC I (pH 8.10), MAC II (pH 7.58), or water. The MAC I and MAC II groups received 8.57 mg of the MAC per 35 ml of water twice daily for 5 days. Rats in each condition were allowed to consume the provided fluid ad libitum, so the magnitude of dosing for each rat varied. After 5 days of supplementation a subgroup of rats in each condition ran on a rodent treadmill for 60 min at a pace of 18 m/min at 0 grade. Pre- and postexercise lactate concentrations were obtained, showing that consumption of the MAC II prevented a significant increase in blood lactate after exercise (Goldfarb et al., 2004). Purdy Lloyd et al. and Glazier et al. did not find MAC supplementation to improve time-trial performance, but their findings that postexercise blood lactate concentrations were significantly reduced after submaximal exercise suggest that MAC supplementation may enhance exercise performance that could be influenced by lactate accumulation, by maintaining blood pH, allowing users to exercise at high intensities for longer periods of time.

Given the results of previous investigations, MAC supplementation may be effective at buffering postexercise blood lactate concentrations after prolonged submaximal exercise (Goldfarb et al., 2004; Purdy Lloyd et al., 2001), but no studies to date have used an exercise bout or exercise intensity that sufficiently challenged the lactate-buffering system, resulting in physiological scenarios where hydrogen-ion accumulation may preclude continued exercise activity. Therefore, the purpose of this investigation was threefold: (a) to examine the effects of acute MAC supplementation on total body water (TBW), intracellular water (ICW), and extracellular water (ECW) before and after maximal exertion exercise; (b) to examine the acute effects of MAC supplementation on lactate buffering and aerobic performance parameters during a high-intensity, maximal exercise protocol performed on a cycle ergometer; and (c) to examine the effects of acute MAC supplementation on clinical serum and whole-blood markers.

Methods

Participants

Fifteen recreationally active men (age 23.6 ± 3.7 years, height 180 ± 8 cm, weight 85 ± 11 kg, $16\% \pm 5\%$ fat, 45.6 ± 1.1 ml $\cdot$ kg^{-1} $\cdot$ min^{-1} O_2) volunteered to participate in the investigation. They completed comprehensive medical-history questionnaires and signed an informed-consent form approved by the University of Oklahoma before participation. Participants were excluded if they had a history of hypertension or hepatorenal, metabolic, neurological, autoimmune, or musculoskeletal disease; were currently taking antihyperlipidemic, thyroid, hypoglycemic, antihypertensive, or androgenic medications; or had taken ergogenic levels of nutritional supplements that may affect muscle mass (e.g., creatine, HMB) or anabolic or catabolic hormone levels (e.g., androstenedione, DHEA) within 3 months before the start of the investigation.

Experimental Design

A double-blind, randomized, crossover design was used (See Figure 1). Each participant visited the laboratory on five occasions: familiarization, baseline, Testing Day 1, washout, and Testing Day 2. Before each visit participants were asked to refrain from strenuous activity for 48 hr;

Figure 1 — A schematic depiction of the experimental design. HR = heart rate; BP = blood pressure.

avoid consuming coffee, alcohol, and vitamin and mineral supplements for 24 hr; fast for 10 hr; and not consume any water 60 min before each testing session. During the first visit (familiarization) participants were fitted for the cycle ergometer (Quinton Corival 400, Seattle, WA) and completed a ramped VO_{2peak} test. Seat and handlebar height were recorded and maintained for each participant during each subsequent VO_{2peak} test. Participants then completed a Physical Activity Recall questionnaire to assess their physical activity level during the previous 6 months and a 2-day food diary before baseline testing. Participants were given a copy of this food diary and asked to repeat the same diet before each testing session. Three days after the familiarization session participants returned to the laboratory for baseline testing (water-only condition) that consisted of resting measures of heart rate and blood pressure, height, weight, and percent body fat; a fasting blood draw; and a preexercise measure of body water (TBW, ICW, and ECW). Participants then drank 237 ml of water 30 min before exercise testing and another 237 ml of water on reaching volitional exhaustion. They then rested quietly for 2 hr before the assessment of postexercise body water. Participants were then randomly assigned via coin flip to consume a 7-day supply of either the MAC or a rice-flour placebo.

Seven days after baseline testing participants returned to the laboratory for Testing Day 1. The protocol on Testing Day 1 was identical to baseline testing with the following exceptions: Body composition was not assessed, and participants consumed three capsules of their assigned supplement with 237 ml of water 30 min before exercise testing and one capsule with 237 ml of water on reaching volitional exhaustion. After Testing Day 1 participants began a 7-day washout period in which no supplement was consumed and returned to the laboratory for preexercise body-water assessment. They then crossed over to the other supplement condition and returned to the laboratory after 7 days of supplementation for Testing Day 2, which was identical to Testing Session 1. A schematic of the study design can be seen in Figure 1.

Body-Water Assessment

Bioimpedance spectroscopy was used to estimate TBW, ICW, and ECW following the procedures recommended by the manufacturer (Imp SFB7, ImpediMed Limited, Queensland, Australia) and described in detail by Matthie et al. (1998). In short, a range of frequencies encompassing low and high ranges that allow electrical current to pass around and through each cell was used, which produces valid estimates of TBW, ICW, and ECW compared with a criterion method such as deuterium oxide and sodium bromide (Matthie et al., 1998; Moon et al., 2008; Van Loan, Withers, Matthie, & Mayclin, 1993). Measurements of TBW, ICW, and ECW were obtained by the same individual while the participant lay in a supine position on a table with arms ≥30° away from the torso and legs separated. Electrodes were placed at the distal ends of each participant's right hand and foot following the manufacturer guidelines. Before electrode placement, excess body hair was removed and the skin was cleaned with alcohol at each site. Electrode locations were marked after initial measurements to allow for accurate placement for subsequent measurements within and between testing days. The average of two trials within ±0.05 L was used as the representative TBW, ICW, and ECW. Before analysis, each participant's height, body mass, age, and sex were entered into the BIS device. The BIS used 256 frequencies internal to the device to estimate TBW, ICW, and ECW. Previous test–retest measurements of 11 men and women measured 24–48 hr apart for TBW, ICW, and ECW using the Imp SFB7 BIS produced ICCs greater than .98 and *SEM*s of 0.48, 0.35, and 0.45 L, respectively.

Maximal Exercise Session

A VO_{2peak} protocol on a cycle ergometer interfaced to a metabolic cart (ParvoMedics, Sandy, UT) was used to determine maximal oxygen uptake of all participants. Participants maintained a pedaling cadence of 60–70 rpm. Testing began with a 2-min warm-up in which they cycled at 20 W. As the testing phase began, the workload increased to 50 W and subsequently increased by 1 W every 3 s (20-W increase per minute) until volitional exhaustion. Exhaustion was determined as the time point at which the participant could not maintain a cadence above 50 rpm despite verbal encouragement. On termination, a 10-min active cooldown was initiated in which participants pedaled at a rate of 50 rpm at 20 W. The test was considered maximal if one of the following criteria was met: RER was ≥1.10 or maximal heart rate was within 10 beats of age-predicted maximal heart rate.

Ratings of Perceived Exertion and Capillary Blood Sampling and Analysis

Ratings of perceived exertion (RPE) using a Borg scale and capillary blood, sampled via finger prick, were obtained 5 min before exercise, 5 min after the onset of exercise, every 90 s until exhaustion, and 5 min after exhaustion. Nonheparinized, chilled, sterile glass capillary tubes were used to obtain blood during the testing session (~25 µl). Blood samples were then immediately analyzed for lactate concentrations using a portable lactate analyzer (Accusport, Boeringer Mannheim, Castle Hill, Australia) in duplicate according to the manufacturer's specifications. Reliability measurements of 8 men and women measured 24 hr apart for blood lactate using this device produced an average duplicate coefficient of variation of .09 and a test–retest reliability of .944.

Supplementation Protocol, Dietary Monitoring, and Physical Activity Analysis

In a double-blind fashion participants were assigned to consume 2,400 mg of either the MAC or rice flour (placebo) per day during each supplementation period. They were instructed to consume four 600-mg capsules

per day—one in the morning, two in the afternoon, and one in the evening—during each supplementation period on nontesting days. Each MAC capsule contained 250 mg of a colloidal silicate mineral and 350 mg of a rice bran flour as an excipient ingredient (Royal Body Care Life Sciences, Irving, TX). The silicate supplement consists of a proprietary colloidal mineral containing food-grade silica, potassium carbonate, potassium citrate, and magnesium sulfate formulated into a spherical nanocolloidal silicate particle. Each placebo capsule contained 600 mg of rice flour. On testing days, participants consumed three capsules with 237 ml of water 30 min before exercise and one capsule with 237 ml of water 10 min after maximal-exertion exercise. Supplements were packaged in generic bottles by Royal Body Care Life Sciences (Irving, TX), and adherence was monitored by having participants return the empty supplement bottles at each testing session and complete a daily supplement log. Dietary intake was monitored with a 2-day dietary recall completed before baseline testing. Participants were given a copy of their 2-day dietary recall and asked to repeat the same diet before each testing session to ensure caloric homogeneity between testing conditions. Caloric intake was assessed using Food Processor III Nutrition Software version 6.8 (ESHA Nutrition Research, Salem, OR). The average physical activity patterns of participants for 6 months before the investigation were monitored using a Physical Activity Recall questionnaire (George, Stone, & Burkett, 1997).

Serum and Whole-Blood Analyses

Serum and whole-blood samples were used to evaluate clinical safety during the supplementation protocol. Serum samples were assayed at a commercial diagnostic laboratory using automated clinical chemistry and hematology analyzers for comprehensive metabolic panels including glucose, triglycerides, cholesterol, HDL cholesterol, LDL cholesterol, total protein, blood urea nitrogen (BUN), creatinine, BUN:creatinine ratio, albumin, globulin, sodium, potassium, chloride, calcium, carbon dioxide, total bilirubin, aspartate aminotransferase, alanine aminotransferase, gamma-glutamyl transpeptidase, and alkaline phosphatase. Whole blood was analyzed for red cell counts, hemoglobin, hematocrit, mean cell volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, red cell distribution width, white blood cell counts, neutrophils, lymphocytes, monocytes, eosinophils, and basophils.

Statistical Analysis

Descriptive statistics of means and standard errors were calculated for average physical activity patterns and calorie and macronutrient consumption. To examine the efficacy of the washout period, separate independent *t* tests were used to learn whether there were differences in TBW, ICW, and ECW and selected serum and whole-blood clinical safety markers between supplementation conditions. Supplementation effects were examined using one-way ANOVAs on changes in all clinical-safety and body-water markers. Separate one-way ANOVAs were used to examine baseline differences between conditions for heart rate, blood pressure, serum clinical chemistry, and whole-blood markers. In response to maximal exercise and recovery from exercise, 3×2 (Condition $\times$ Time) repeated-measures ANOVAs were used to examine the main and interactive effects of supplementation on body water.

Separate 3×8 (Condition $\times$ Time) repeated-measures ANOVAs with Tukey's post hoc comparisons were used to examine blood lactate and RPE responses. Separate one-way ANOVAs were used to assess time to exhaustion, time to ventilatory threshold, time to a respiratory quotient of 1.0, VO_{2peak}, peak power, power at ventilatory threshold, heart rate at peak power, time to exhaustion at ventilatory threshold, and time to exhaustion after reaching a respiratory quotient of 1.0. A probability level of $<.05$ was used to determine significance for each variable. All data are presented as $M \pm SE$. All statistical analyses were performed using SPSS (version 15.0, SPSS Inc., Chicago, IL).

Results

Washout Period and Supplement Compliance

No significant differences were noted between conditions for TBW ($p = .97$), ICW ($p = .82$), or ECW ($p = .88$; Figure 2). In addition, no between-conditions differences were noted for any of the assessed serum and whole-blood clinical-safety markers. No participants reported missing any doses throughout the day. On multiple occasions, participants reported breaking from protocol by missing their lunchtime dose, but all participants subsequently made that dose up later in the day, allowing adequate time between their lunch and evening doses.

Physical Activity, Hemodynamic Parameters, and Nutrient Intake

Self-reported physical activity using a Physical Activity Recall questionnaire yielded an average activity rating of 7.5 ± 0.3, which equates to participation in 1–3 hr of vigorous physical activity per week. Each participant consumed approximately 32.5 ± 10.6 kcal $\cdot$ kg^{-1} $\cdot$ day^{-1}—1.5 ± 0.6 g $\cdot$ kg^{-1} $\cdot$ day^{-1} of protein (19% of total calories), 4.2 ± 1.6 g $\cdot$ kg^{-1} $\cdot$ day^{-1} of carbohydrate (51% total calories), and 1.0 ± 0.4 g $\cdot$ kg^{-1} $\cdot$ day^{-1} of fat (30% of total calories)—and these values remained consistent throughout the investigation. No significant differences were noted for any of the resting hemodynamic parameters that were measured, which included heart rate (water 61.7 ± 1.5, placebo 64.1 ± 3.3, and MAC 63.3 ± 2.9 beats/min; $p = .79$), systolic blood pressure (water 122.5 ± 2.6, placebo 118.8 ± 2.3, and MAC 121.2 ± 2.7 mm Hg; $p = .59$), and diastolic blood pressure (water 80.1 ± 1.7, placebo 79.2 ± 1.0, and MAC 80.9 ± 1.8 mm Hg, $p = .74$).

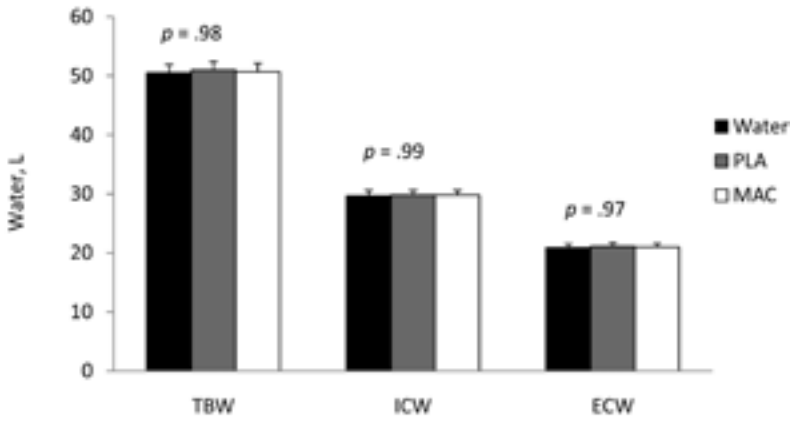

Figure 2 — Supplementation effects on preexercise body water. Data are expressed as $M \pm SE$ ($N = 15$). TBW = total body water; ICW = intracellular water; ECW = extracellular water; PLA = placebo; MAC = mineral antioxidant complex.

Body-Water Assessment

In response to an acute bout of maximal exercise, no condition-by-time interactions were found for TBW ($p = .09$), ICW ($p = .052$), or ECW ($p = .19$), as can be seen in Figures 3, 4, and 5, respectively. Although p values for TBW and ICW approached significance, the changes lack physiological significance (Figures 3[b] and 4[b]). In response to exercise, a significant main effect for time was found for TBW ($p < .01$) and ICW ($p < .001$) but not for ECW ($p = .52$). In this regard, a within-group one-way ANOVA on TBW revealed significant increases after exercise in the MAC ($p < .01$) condition but not the placebo ($p = .16$) and control ($p = .89$) conditions. Similarly, a follow-up one-way ANOVA on ICW revealed significant increases in the MAC ($p < .01$) and placebo ($p < .005$) conditions but not for the control ($p = .13$) condition.

Clinical Safety

There were no significant differences between conditions for heart rate or blood pressure (Table 1). Changes in complete blood cell and clinical serum chemistry markers are presented in Tables 2 and 3. There were no significant differences between conditions for red blood cell count, white blood cell count, or white blood cell differentials over the course of the investigation ($p > .05$). Absolute basophil count did show a tendency to decrease by approximately 25% in both conditions ($p = .06$), but all values remained within normal clinical ranges. Furthermore, there were no significant differences between conditions for serum triglycerides, HDL cholesterol, LDL cholesterol, glucose, hepatic enzymes (i.e., aspartate aminotransferase, alanine aminotransferase), hepatic proteins (i.e., total protein, albumin, and bilirubin), or crude markers of kidney integrity (i.e., alkaline phosphatase, BUN, creatinine; $p > .05$). There were no significant differences between conditions for electrolytes (i.e., sodium, potassium, chloride, calcium) except for carbon dioxide ($p = .03$), but values were within normal ranges for each condition. No participants reported any adverse effects of the supplementation protocol, with the exception of 3 reporting mild stomach cramping and discomfort during the MAC condition.

RPE, Blood Lactate, and Maximal Exercise Performance

No significant condition-by-time interaction was present for RPE, but a significant time effect was present ($p < .001$; Figure 6). No significant condition-by-time interaction was present for blood lactate, but there was a main effect for time ($p < .001$), with blood lactate values in each condition similarly increasing as the exercise test progressed (Figure 7). Finally, supplementation did not affect any performance outcomes between conditions (Table 4).

Discussion

The primary aim of this investigation was to determine the impact of MAC supplementation on changes in body water, blood lactate, and aerobic exercise performance. A secondary aim was to assess the safety and efficacy of 7 days of MAC consumption. Increases in body-water compartments were seen after both forms of supplementation, but these increases lacked physiological significance in our measured outcomes, as exercise performance and the lactate response were unaffected. Supplementation was well tolerated, but the MAC did not appear to affect any measured outcomes.

The hypothesized function of silica supplementation involves the formation of a cellular interface that leads to favorable changes in body-water availability and may serve to buffer lactate and other untoward cellular metabolites (Purdy Lloyd et al., 2001). To investigate the hypothesis that MAC supplementation could influence body-water and cellular-hydration parameters, TBW, ICW, and ECW were measured 30 min before exercise and 2 hr postexercise for each condition. At baseline, no

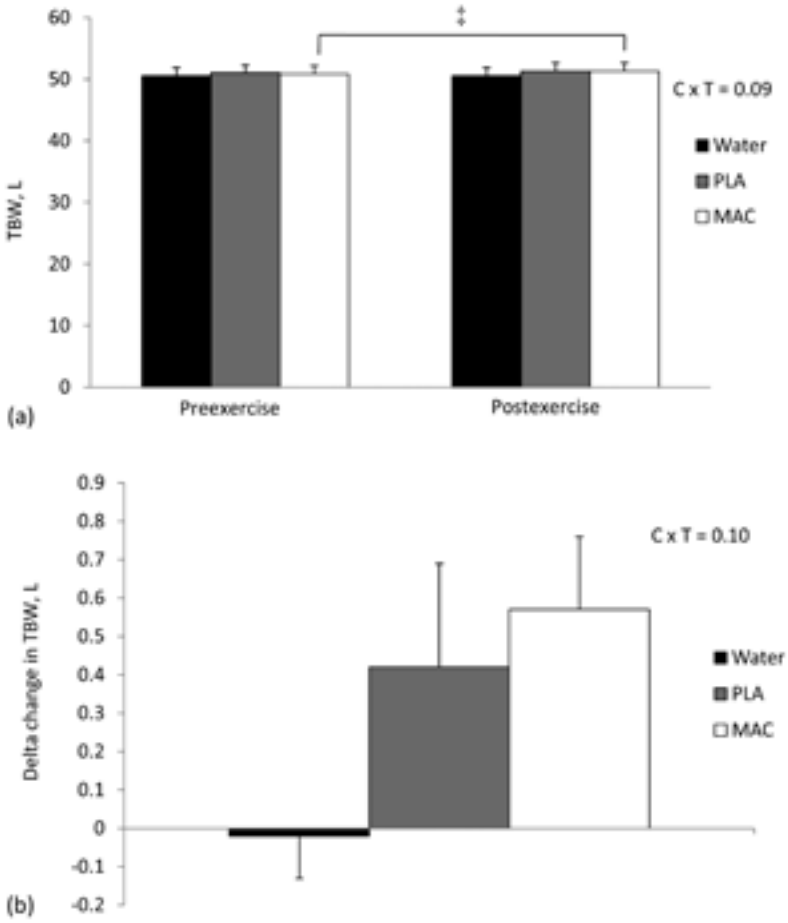

Figure 3 — (a) Effects of supplementation and exercise on total body water (TBW). ‡Significantly different from preexercise value ($p < .05$). (b) Delta change (postexercise – preexercise) in TBW. Data are expressed as $M \pm SE$ ($N = 15$). C × T = Condition × Time interaction as assessed by a two-way ANOVA with repeated measures; PLA = placebo; MAC = mineral antioxidant complex.

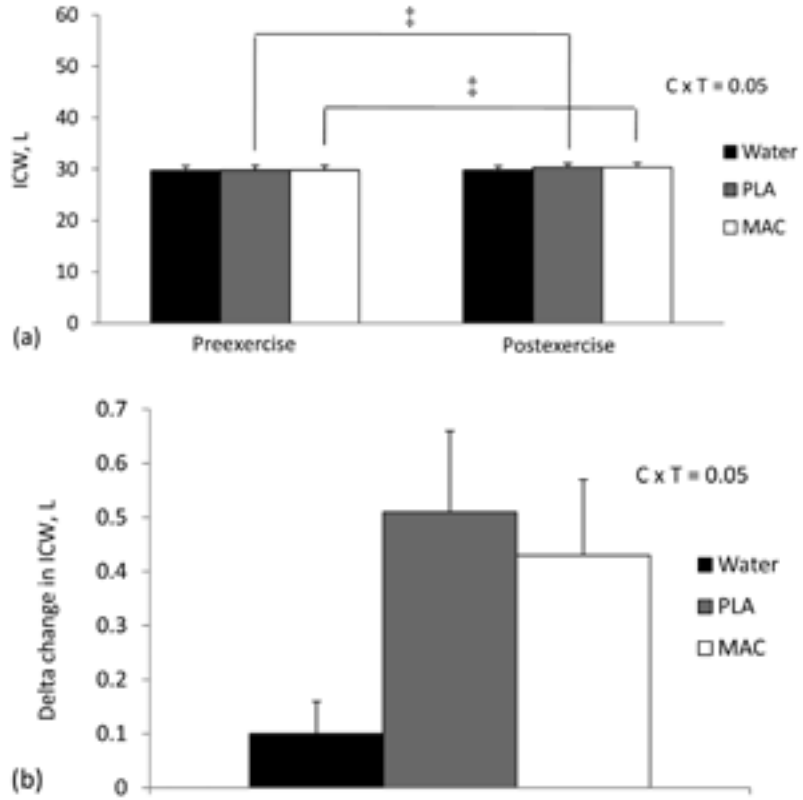

Figure 4 — (a) Effects of supplementation and exercise on intracellular water (ICW). ‡Significantly different from preexercise value ($p < .05$). (b) Delta change (postexercise – preexercise) in ICW. Data are expressed as $M \pm SE$ ($N = 15$). C × T = Condition × Time interaction as assessed by a two-way ANOVA with repeated measures; PLA = placebo; MAC = mineral antioxidant complex.

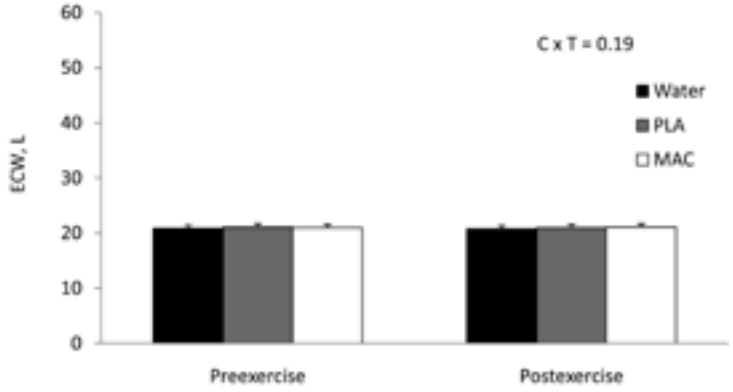

Figure 5 — Effects of supplementation and exercise on extracellular water (ECW). Data are expressed as $M \pm SE$ ($N = 15$). C × T = Condition × Time interaction as assessed by a two-way ANOVA with repeated measures; PLA = placebo; MAC = mineral antioxidant complex.

Table 1 Changes in Serum Lipid and Clinical Chemistry Markers, *M* ± *SE*

Parameter	Baseline	Placebo	Mineral antioxidant complex	*p*
Triglycerides (mg/dl)	103 ± 18	85 ± 7	83 ± 8	.44
Total cholesterol (mg/dl)	157 ± 7	158 ± 8	164 ± 7	.75
HDL cholesterol (mg/dl)	50 ± 3	51 ± 3	52 ± 2	.83
LDL cholesterol (mg/dl)	86 ± 5	90 ± 6	95± 5	.51
Glucose (mg/dl)	88 ± 3	94 ± 2	91 ± 1	.17
Blood urea nitrogen (mg/dl)	16 ± 1	16 ± 1	16 ± 1	.80
Creatinine (mg/dl)	1.0 ± 0.1	1.0 ± 0.1	1.0 ± 0.1	.82
Sodium (mM)	141 ± 1	140 ± 0.3	141 ± 0.4	.72
Potassium (mM)	5.0 ± 0.1	5.0 ± 0.1	5.0 ± 0.1	.59
Chloride (mmol/L)	105 ± 0.6	105 ± 0.5	105 ± 0.4	1.00
CO_2 (mmol/L)	25 ± 0.3	23 ± 0.4	23 ± 0.4	.03*
Calcium (mg/dL)	10 ± 0.1	10 ± 0.1	10 ± 0.1	.98
Alkaline phosphatase (U/L)	61 ± 5	63 ± 5	63 ± 4	.95
Alanine aminotransferase (U/L)	20 ± 2	21 ± 4	20 ± 2	.23
Aspartate aminotransferase (U/L)	23 ± 1	23 ± 2	24 ± 2	.91
Creatine kinase (U/L)	156 ± 24	175 ± 28	205 ± 40	.55

*Baseline > placebo or mineral antioxidant complex.

Table 2 Changes in Whole-Blood Complete Blood Count Markers, *M* ± *SE*

Parameter	Baseline	Placebo	Mineral antioxidant complex	*p*
White blood cell count (10^3/µl)	5.9 ± 1.3	5.3 ± 0.9	5.6 ± 1.3	.42
Red blood cell count (10^6/µl)	5.0 ± 0.1	4.9 ± 0.1	5.1 ± 0.2	.42
Hemoglobin (g/dl)	15 ± 0.1	15 ± 0.2	15 ± 0.2	.44
Hematocrit (%)	45 ± 0.5	44 ± 0.5	45 ± 0.6	.32
Mean cell volume (fl)	91 ± 0.7	90 ± 0.7	91 ± 0.6	.91
Mean cell hemoglobin (pg)	31 ±1.0	30 ±1.1	31 ± 1.0	.87
Mean cell hemoglobin concentration (g/dl)	34 ± 0.1	34 ± 0.2	34 ± 0.2	.38
Red blood cell distribution width (%)	13 ± 0.2	13 ± 0.2	13 ± 0.2	.73
Platelets (10^3/µl)	220 ±11	208 ± 11	215 ± 11	.74
Neutrophils (cells/µl)	3,185 ± 241	2,475 ± 241	2,870 ± 257	.14
Lymphocytes (cells/µl)	2,030 ± 451	1,755 ± 345	1,983 ± 141	.24
Monocytes (cells/µl [1])	460 ± 32	516 ± 50	479 ± 45	.64
Eosinophils (cells/µl)	165 ± 32	214 ± 47	221 ± 42	.56
Basophils (cells/µl)	33 ± 3.1	20 ± 3.7	27 ± 4.5	.05

Table 3 Changes in Heart Rate and Blood Pressure, *M* ± *SE*

Parameter	Group	Measure	*p*
Heart rate			.786
	water	61.7 ± 1.5	
	placebo	64.1 ± 3.3	
	MAC	63.3 ± 2.9	
Systolic blood pressure			.587
	water	122.5 ± 2.6	
	placebo	118.8 ± 2.3	
	MAC	121.2 ± 2.7	
Diastolic blood pressure			.736
	water	80.1 ± 1.7	
	placebo	79.2 ± 1.0	
	MAC	80.9 ± 1.8	

Note. MAC = mineral antioxidant complex.

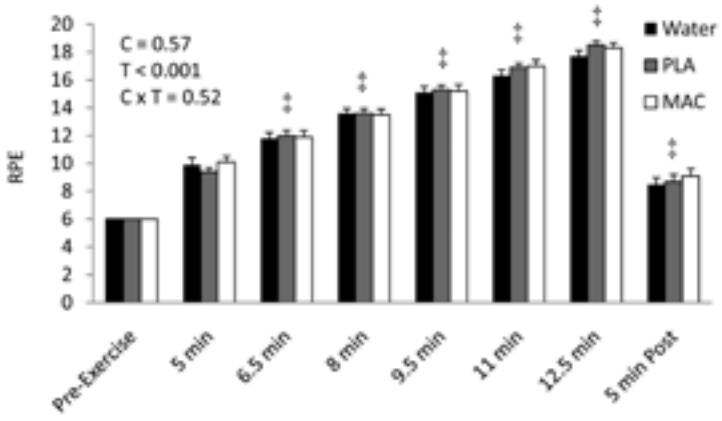

Figure 6 — Effects of supplementation and exercise on ratings of perceived exertion (RPE). Data are expressed as *M* ± *SE* (*N* = 15). C × T = Condition × Time interaction as assessed by a two-way ANOVA with repeated measures; PLA = placebo; MAC = mineral antioxidant complex. ‡Significantly different from preexercise value ($p < .05$).

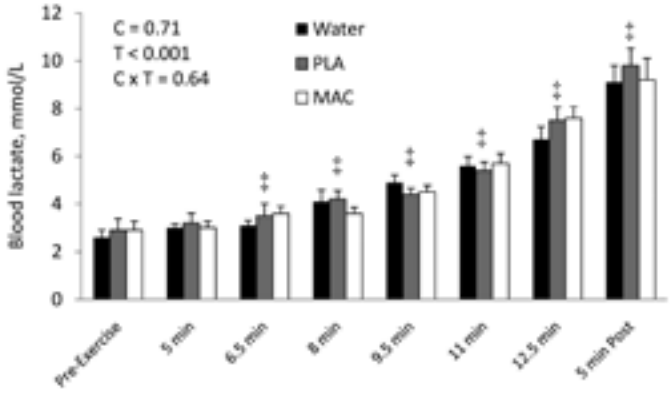

Figure 7 — Effects of supplementation and exercise on blood lactate. Data are expressed as *M* ± *SE* (*N* = 15). C × T = Condition × Time interaction as assessed by a two-way ANOVA with repeated measures; PLA = placebo; MAC = mineral antioxidant complex. ‡Significantly different from preexercise value ($p < .05$).

Table 4 Performance Outcomes, *M* ± *SE*

Parameter	Water	Placebo	Mineral antioxidant complex	*p*
VO_{2peak} (ml · kg^{-1} · min^{-1})	45.6 ± 1.1	44.6 ± 1.5	45.3 ± 1.3	.86
Time to ventilatory threshold (min)	9.2 ± 0.2	8.3 ± 0.2	9.0 ± 0.2	.25
Time to respiratory quotient 1.0 (min)	4.0 ± 0.2	4.5 ± 0.3	4.1 ± 0.2	.58
Time to exhaustion (min)	13.4 ± 0.2	13.3 ± 0.2	13.2 ± 0.2	.90
Time to exhaustion after ventilatory threshold (min)	4.3 ± 0.5	4.9 ± 0.3	4.9 ± 0.4	.41
Time to exhaustion after respiratory quotient of 1.0 (min)	4.0 ± 0.4	4.5 ± 0.3	4.1 ± 0.3	.58
Peak power (W)	285 ± 6	288 ± 6	279 ± 6	.60
Power at ventilatory threshold (W)	204 ± 8	194 ± 8	189 ± 9	.43
Heart rate at peak power (beats/min)	180 ± 2	179 ± 2	177 ± 2	.71

differences were found between conditions for TBW, ICW, or ECW. Moreover, no differences were found between conditions after the 7-day supplementation protocol for each condition. These results suggest that either the available amount of silica hydride molecules when ingested in vivo was not adequate to stimulate changes in the body-water parameters or the MAC did not retain its purported intracellular effect. Body water was also assessed 2 hr after maximal-exertion exercise. TBW was found to be significantly increased during the MAC condition, and ICW was found to be significantly increased during the placebo and MAC conditions. The increase in TBW and ICW was most likely the result of ingested water (474 ml) as part of the study design, but this suggestion is precluded by there being no change in the water-only condition. Although it was unexpected for the placebo (rice flour) to affect hydration, it is possible that this ingredient has osmotic properties that stimulated the changes in body-water compartments. Nonetheless, the measured changes in body water for the placebo and MAC groups did not result in meaningful physiological outcomes; no changes in exercise performance or lactate metabolism were found in the current investigation.

Previous work has reported MAC consumption to have positive effects on lactate buffering (Goldfarb et al., 2004; Purdy Lloyd et al., 2001), which could enhance aerobic performance outcomes; however, these findings have not been consistent. For instance, Purdy Lloyd et al. found 5-min-postexercise blood lactate to be significantly decreased in participants when consuming a MAC compared with a rice flour placebo after a 40-km bicycling time trial. Furthermore, Glazier et al. (2004) used a randomized, double-blind, crossover design and reported that consuming 9.6 g of the MAC (six capsules) over a 48-hr period elicited no effect on lactate and aerobic exercise performance compared with a D-glucose placebo in a performance time trial in well-trained cyclists. However, both investigations (Goldfarb et al., 2004; Purdy Lloyd et al., 2001) used a submaximal exercise protocol and only assessed pre- to postexercise blood lactate concentrations. The maximal-exercise protocol used in the current investigation caused a rapid increase in blood lactate; however, MAC supplementation had no significant effect on RPE, blood lactate concentrations, or any measure of aerobic performance. It is possible that no silica hydride molecules are present in the MAC; previous work using infrared spectrometry analysis has found the MAC to contain no silica hydride molecules (Glazier et al., 2004).

Relative to clinical safety, MAC supplementation had no effect on heart rate, blood pressure, or any of the serum or whole-blood clinical-safety markers that were assessed, except for plasma CO_2 (mmol/L), which was significantly different between conditions. Clinically, plasma CO_2 can be used as a measure of blood pH, which could theoretically be influenced by the active ingredients in the MAC as a result of increased hydrogen-ion buffering (Goldfarb et al., 2004; Purdy Lloyd et al., 2001) or reduced lactate production (Goldfarb, et al., 2004; Purdy Lloyd et al., 2001). Goldfarb et al. postulated that silica hydride supplementation may decrease lactate levels by scavenging hydrogen-ion molecules from NADH to form NAD^+, which would reduce pyruvate conversion to lactate, increasing blood pH. However, plasma CO_2 concentrations for each condition were within the reference range of 21–33 mmol/L and were lowest at baseline, suggesting that any ability of MAC supplementation to alter CO_2 levels by buffering pH was minimal. Nonetheless, our data suggest that short-term (≤7-day) supplementation with a MAC containing silica, potassium, and magnesium as active ingredients is well tolerated for short-duration consumption, although the available research is mixed regarding its efficacy as an ergogenic aid (Glazier, et al., 2004; Purdy Lloyd, et al., 2001), and longer supplementation periods need to be considered relative to clinical safety.

A potential limitation of the current investigation was the effectiveness of the washout period. The selected washout period of 7 days has been employed in previous investigations (Glazier et al., 2004; Purdy Lloyd et al., 2001). The current investigation used preexercise body water as a measure of washout, which has limitations because body water has not been previously found to be influenced by MAC supplementation. However, a prior investigation that used a 7-day washout period found MAC supplementation to significantly reduce postexercise blood lactate concentrations compared with a placebo (Purdy Lloyd et al., 2001). Furthermore, the MAC is composed of commonly ingested minerals that

do not accumulate intramuscularly and thus are not likely to be stored in the body for extended periods of time. In a similar light, changes in serum and whole-blood markers of clinical safety between conditions also suggest that the 7-day washout period was adequate.

Results from the current investigation suggest that acute MAC supplementation is safe, as determined by the effects of supplementation on the clinical chemistry analysis of blood, resting heart rate, and blood pressure. Moreover, the MAC was found to have no meaningful effect on TBW, ICW, ECW, blood lactate, or aerobic performance parameters. Given the safety of acute MAC supplementation and the potential health-promoting benefits of silica hydride (Keller, 1978; Leaf, 1973; Murray & Murray, 1984), future research should examine whether silica hydride bonds can remain intact in the supplement and the human body. Past research has found the MAC to positively influence postexercise blood lactate concentrations (Goldfarb et al., 2004; Purdy Lloyd et al., 2001), but Glazier et al. (2004) found the MAC to contain no silica hydride bonds, which is concerning because silica hydride bonds have been purported to elicit the positive effects of the MAC.

Conclusions

Current results suggest that short-term MAC supplementation is well tolerated as determined by the effects of supplementation on the clinical chemistry analysis of blood, resting heart rate, and blood pressure, but interpretations of safety should be taken with caution because long-term safety has yet to be evaluated. The purported effect of silica supplementation to form a cellular interface of silica-hydride bonds to influence body-water compartments and lactate buffering was equivocal in this investigation. Within-group changes in single conditions after maximal exercise revealed an increase of TBW in the MAC condition and an increase in ICW in the MAC and placebo conditions, and no significant changes occurred in TBW or ICW in the water-only control. However, the increases in TBW and ICW after MAC consumption were small (TBW 1.1% and ICW 1.5% increase) and were not accompanied by measured physiological changes (lactate response or any measured performance variable). Although prior research has found MAC supplementation to affect blood lactate (Goldfarb et al., 2004; Purdy Lloyd et al., 2001), the results were not replicated in the current investigation, and changes in performance were also not found (Glazier et al., 2004; Purdy Lloyd et al., 2001).

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References

Baker, A.J., Brandes, R., & Weiner, M.W. (1995). Effects of intracellular acidosis on Ca2+ activation, contraction, and relaxation of frog skeletal muscle. *The American Journal of Physiology, 268*(1 Pt 1), C55–C63.

Dove, P., & Rimstidt, J. (1994). Silica-water interactions. *Reviews in Mineralogy and Geochemistry, 29*(1), 259–308.

Favero, T.G., Zable, A.C., Bowman, M.B., Thompson, A., & Abramson, J.J. (1995). Metabolic end products inhibit sarcoplasmic reticulum Ca2+ release and [3H]ryanodine binding. *Journal of Applied Physiology, 78*(5), 1665–1672.

Fitts, R.H., & Holloszy, J.O. (1976). Lactate and contractile force in frog muscle during development of fatigue and recovery. *The American Journal of Physiology, 231*(2), 430–433.

George, J.D., Stone, W.J., & Burkett, L.N. (1997). Non-exercise VO2max estimation for physically active college students. *Medicine and Science in Sports and Exercise, 29*(3), 415–423.

Glazier, L.R., Stellingwerff, T., & Spriet, L.L. (2004). Effects of microhydrin supplementation on endurance performance and metabolism in well-trained cyclists. *International Journal of Sport Nutrition and Exercise Metabolism, 14*(5), 560–573.

Goldfarb, A.H., Bloomer, R., & McKenzie, M.J. (2004). Effect of microhydrin on blood lactate, protein carbonyls, and glutathione status in rats before and after aerobic exercise. *International Journal of Sport Nutrition and Exercise Metabolism, 14*(5), 550–559.

Keller, W. (1958). Argillation and direct bauxitization in terms of concentrations of hydrogen and metal cations at surface of hydrolyzing aluminum silicates. *Bulletin of the American Association of Petroleum Geologists, 42,* 233–245.

Keller, W. (1978). Drinking water: A geochemical factor in human health. *Geological Society of America Bulletin, 89,* 334–336.

Leaf, A. (1973). Getting old. *Scientific American, 229*(3), 44–52.

Matthie, J., Zarowitz, B., De Lorenzo, A., Andreoli, A., Katzarski, K., Pan, G., & Withers, P. (1998). Analytic assessment of the various bioimpedance methods used to estimate body water. *Journal of Applied Physiology, 84*(5), 1801–1816.

Moon, J.R., Tobkin, S.E., Roberts, M.D., Dalbo, V.J., Kerksick, C.M., Bemben, M.G., . . . Stout, J.R. (2008). Total body water estimations in healthy men and women using bioimpedance spectroscopy: a deuterium oxide comparison. *Nutrition & Metabolism, 5,* 7.

Murray, M., & Murray, A. (1984). Diet and cardiovascular disease in centenarians of Hunza. *Arteriosclerosis (Dallas, Tex.), 4,* 546A.

Nevill, M.E., Boobis, L.H., Brooks, S., & Williams, C. (1989). Effect of training on muscle metabolism during treadmill sprinting. *Journal of Applied Physiology, 67*(6), 2376–2382.

Purdy Lloyd, K.L., Wasmund, W., Smith, L., & Raven, P.B. (2001). Clinical effects of a dietary antioxidant silicate supplement, microhydrin((R)), on cardiovascular responses to exercise. *Journal of Medicinal Food, 4*(3), 151–159.

Spriet, L.L., Lindinger, M.I., McKelvie, R.S., Heigenhauser, G.J., & Jones, N.L. (1989). Muscle glycogenolysis and H+ concentration during maximal intermittent cycling. *Journal of Applied Physiology, 66*(1), 8–13.

Van Loan, M.D., Withers, P., Matthie, J., & Mayclin, P.L. (1993). Use of bioimpedance spectroscopy to determine extracellular fluid, intracellular fluid, total body water, and fat-free mass. *Basic Life Sciences, 60,* 67–70.

Westerblad, H., Allen, D.G., & Lannergren, J. (2002). Muscle fatigue: Lactic acid or inorganic phosphate the major cause? *News in Physiological Sciences, 17,* 17–21.