

The effects of magnesium supplementation on exercise performance

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ABSTRACT

FINSTAD, E. W., I. J. NEWHOUSE, H. C. LUKASKI, J. E. MCAULIFFE, and C. R. STEWART. The effects of magnesium supplementation on exercise performance. *Med. Sci. Sports Exerc.*, Vol. 33, No. 3, 2001, pp. 493–498. **Purpose:** To determine the effects of magnesium (Mg^{2+}) supplementation on performance and recovery in physically active women using the sensitive and recently advanced measure of ionic Mg^{2+} (iMg). **Methods:** Participants ($N = 121$) were screened for [iMg] in plasma, with 44 (36.4%) exhibiting [iMg] below the normal range of 0.53–0.67 $mmol \cdot L^{-1}$ (4). Thirty-two subjects (21 ± 3 yr) representing a broad range of [iMg] (0.54 ± 0.04 $mmol \cdot L^{-1}$) completed the main 14-wk study. At baseline, participants submitted to a resting blood pressure measurement, and they completed both an anaerobic treadmill test and an incremental (aerobic) treadmill test. For the latter, values for workload, oxygen uptake, and heart rate were obtained at both anaerobic threshold and maximal effort. Blood samples for iMg, total serum Mg^{2+} (TMg), erythrocyte Mg^{2+} (EMg), Ca^{2+} , K^+ , Na^+ , hemoglobin, hematocrit, lactate, and glucose were also collected pretest, and 4, 10, 30 min, and 24 h posttest. Subjects received 212 $mg \cdot d^{-1}$ Mg oxide or placebo in a double-blind fashion and were retested after 4 wk. After a 6-wk washout period, the testing was repeated with a treatment crossover. **Results:** Ionic Mg^{2+} increased with Mg^{2+} treatment versus placebo ($P < 0.05$); however, performance and recovery indices were not significantly affected. **Conclusion:** Four weeks of 212 $mg \cdot d^{-1}$ Mg oxide supplementation improves resting [iMg] levels but not performance or recovery in physically active women. **Key Words:** MAGNESIUM DEFICIENCY, IONIC MAGNESIUM, PHYSICALLY ACTIVE, WOMEN, $\dot{V}O_{2max}$, ANAEROBIC THRESHOLD, ANAEROBIC TREADMILL TEST, RECOVERY

Magnesium (Mg^{2+}) is an essential mineral and co-factor for over 400 enzymatic reactions with central roles in the control of neuronal activity, cardiac excitability, neuromuscular transmission, muscular contraction, vasomotor tone, and blood pressure. The intracellular functions of Mg^{2+} are achieved through the formation of magnesium adenosine triphosphate (Mg^{2+} -ATP)—a substrate for a wide variety of enzymes that act to breakdown fatty acids, amino acids, and glucose during energy metabolism (1). Magnesium plays a vital role in regulating cell growth, reproduction, and membrane structure by regulating deoxyribonucleic acid and ribonucleic acid synthesis and structure. Magnesium also serves as a Ca^{2+} channel blocker so that an inadequate amount of the mineral would lead to hypertension and arrhythmias (1). The maintenance of an adequate Mg^{2+} status is therefore of utmost importance for optimal exercise performance and recovery.

Recommended intakes are 200 and 250 $mg \cdot d^{-1}$ for women and men, respectively, in Canada (16), and 280 and 350 $mg \cdot d^{-1}$, respectively, in the United States (14). As values from 70 to 100% of the Recommended Dietary Allowance (RDA) are considered adequate for an individual, cause for concern results when intakes are less than 70% of the RDA

(17). Well over 50% of the normal population may have marginal Mg^{2+} deficiencies as they have Mg^{2+} intakes below that of the RDA (19). Lukaski (18) reports that typical Western diets, which are high in protein and/or fat, may not contain sufficient amounts of Mg^{2+} . Also, due to the increasing use of fertilizers (lacking Mg^{2+}) and food processing (removing Mg^{2+}) in practice today, intakes have declined from 500 $mg \cdot d^{-1}$ to about 175–225 $mg \cdot d^{-1}$ (2). In addition, lower Mg^{2+} intakes and, thus, marginal Mg^{2+} deficiencies are more likely to be found in women rather than men (7,17).

Concerning athletic populations, Seelig (24) notes that up to half consume diets containing less than the RDA. Athletes in particular tend to have increased needs for Mg^{2+} most likely due to greater urinary and surface losses during periods of exercise training (7,12,20,22). Because recommended intakes of the mineral are based on nonathletic populations, and because the literature regarding the actual Mg^{2+} status of athletes is limited, one can still question whether the observed dietary intakes are sufficient for the amount of energy that the athletes are expending and the potential increased Mg^{2+} losses.

Research has been equivocal in regard to Mg^{2+} supplementation's effect on exercise performance. Some studies have been supportive (9,10,13,15,21), whereas others (23,25,27–29) found no benefit to exercise performance. Although it may be assumed that a suboptimal intake of Mg^{2+} could result in physiological impairments, research

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has relied on insensitive indicators of Mg^{2+} status if Mg^{2+} status was measured at all.

It is therefore not clear whether Mg^{2+} supplementation, beyond the maintenance of an adequate dietary intake of the mineral, is effective in enhancing performance and recovery from exercise. In addition, little research has concentrated on physically active women who may be at the highest risk for Mg^{2+} deficiency. Thus, the purpose of this investigation was to examine the effects of Mg^{2+} supplementation on Mg^{2+} levels as well as the effects of Mg^{2+} supplementation on performance and recovery in physically active women. This would be achieved by utilizing the highly specific, sensitive, and recently advanced measure of ionic Mg^{2+} (iMg) assay, and by employing a randomized, double-blind, placebo-controlled crossover experimental design. It was hypothesized that Mg^{2+} supplementation would increase Mg^{2+} levels as well as performance and recovery from exercise.

METHODS

Participants. Once written informed consent was obtained (Ethics Advisory Committee, Lakehead University), 121 apparently healthy, physically active women between the ages of 17 and 43 residing in the Thunder Bay, Ontario, Canada, region were screened for participation based on iMg status. Those who qualified to continue the study were 20 marginally Mg^{2+} -deficient subjects and 20 who possessed [iMg] levels in the upper range of normal. A power test utilizing previous test data on $\dot{V}O_2$ max values (9) indicated the sample size of 40 should be sufficient to reach a power of 0.80 in detecting an effect size of $3 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$. The normal range for [iMg] is $0.53\text{--}0.67 \text{ mmol}\cdot\text{L}^{-1}$ (4). Although the intent of screening based on iMg status was to permit later statistical analysis with initial [iMg] values being an independent variable, the lability of [iMg] made this proposition problematic. Over the course of 3 wk from the time of screening to the date of the first testing period, many subjects changed their iMg classification from low to normal. As well, the correlation of iMg values between the two time periods when no treatment had been initiated was low (see Results). Selection criteria for subjects included: A. no clinical history of proven or suspected hypersensitivity to Mg^{2+} supplements; B. the expectation and willingness to maintain regular physical activity (i.e., performing at least three workouts at or above 75% maximum intensity, for at least 2 h total, per week) throughout the study; and C. the expectation and willingness to maintain regular dietary intakes throughout the study. Exclusion criteria for subjects included: A. not performing the incremental treadmill test at all four test sessions, B. not ingesting at least 75% of pills given per treatment period, C. being ill or injured (enough to prevent exercising) for more than 1 wk during the treatment periods, and D. not maintaining a training load equal to $\pm 25\%$ of their regular load. Enrolment of participants continued until the requisite number of subjects had been identified.

Measurements. Selected subjects underwent three day dietary analysis and anthropometric measurements (height, weight, and sum of five skinfolds). Physiological testing included: A. resting blood pressure; B. incremental treadmill test with measurement of workload, heart rate, and expired gases—for assessment of $\dot{V}O_2$ max, anaerobic threshold, maximal workload, and submaximal running efficiency; C. blood sampling pretest, and 4, 10, 30 min, and 24 h posttest, for assessment of [iMg], [TMg], [iCa], [plasma lactate], [plasma glucose], [plasma K^+], [plasma Na^+], [hemoglobin], hematocrit; D. anaerobic treadmill test; and E. subjective and objective measures of overtraining recorded daily in a training diary (for control purposes).

Four testing periods (T1, T2, T3, and T4) were scheduled. During the weeks of T1 and T3, subjects filled out three consecutive days (including one weekend day) of self-report dietary records. Dietary intakes were assessed using computerized diet analysis software (Diet Analysis Plus™, West Publishing Co., St. Paul, MN). Subjects were asked to refrain from unaccustomed strenuous exercise for 48 h (and any strenuous exercise 24 h) before exercise testing. They were also asked to refrain from alcohol consumption 24 h (and caffeine consumption 6 h) before testing. They were advised to consume a light carbohydrate meal 2–3 h before reporting to the exercise tests which took place between 1:00 and 9:00 p.m. Anthropometric measurements were performed by a Certified Fitness Appraiser according to national standards (11). Harpenden calipers were used to measure skinfolds that were taken from the triceps, biceps, subscapular, iliac crest, and medial calf regions. Percent body fat was predicted using the Durnin-Womersley method (11).

Resting blood pressure (performed by a Certified Fitness Appraiser using an AMG Med. Professional Series sphygmomanometer) and resting heart rate (using a Polar Vantage XL® heart rate monitor from Polar CIC Inc., Kempele, Finland) were obtained after the subject rested in the sitting position for 10 min. Blood was collected in 7-mL green-topped Vacutainer® tubes (lithium-heparin added) by antecubital venipuncture. The tourniquet was applied gently and released before the actual blood draw.

The multi-test Stat Profile™ Ultra Analyzer® model 11–3C (Nova Biomedical Canada Ltd., Mississauga, Ontario) was utilized for the immediate analyses of [iMg], [iCa], [Na^{2+}], [K^+], [hemoglobin], hematocrit, [glucose], and [lactate] from 25 μg of whole blood. The instrument was housed in the same laboratory as the exercise testing, and the analyses were performed by the same technician throughout the study. The precision of the instrument for [iMg] was estimated at $\pm 0.03 \text{ mmol}\cdot\text{L}^{-1}$, as determined by repeat testing on the same sample of blood, both within and between days, on an individual not involved in the study. All analyses were within their respective reference ranges as determined by NOVA quality control substances.

Before the incremental treadmill test, the subjects warmed up on the treadmill (Quinton Instruments, Seattle, WA) for 5 min. The initial treadmill speed was $2.22 \text{ m}\cdot\text{s}^{-1}$ and during the test was increased by $0.22 \text{ m}\cdot\text{s}^{-1}$ every

minute. The treadmill grade remained horizontal until the subject had completed two workloads past the workload at which the subject's respiratory exchange ratio (expired $\text{CO}_2/\text{inspired O}_2$) passed a value of 1.0. At that time, the speed no longer increased but the grade increased by 2% each min. Subjects continued until exhaustion. Expired gases were sampled and analyzed by a SensorMedics \dot{V}_{max} System® metabolic (Yorba Linda, CA) with $\dot{V}\text{O}_2\text{max}$ and \dot{V}_{Epeak} determined as the highest 30-s mean. Heart rates were monitored by a telemetric heart rate monitor and recorded 30 s into each workload. Anaerobic thresholds were assessed via examination of expired gas values according to the method described by Beaver et al. (6). Two investigators blinded to the study, and familiar with anaerobic threshold estimations, independently analyzed the $\dot{V}_{\text{E}}/\dot{V}\text{CO}_2$ curves to pick out the lowest point. These researchers then met to compare estimates. Discrepancies (more than one min differences) were resolved by examining other expired gas indices of anaerobic threshold. Efficiency of running at a submaximal workload was assessed by noting the workload during the T1 test that corresponds to 65% of $\dot{V}\text{O}_2\text{max}$. At T2, T3, and T4, the oxygen consumption was noted at this same workload.

The anaerobic treadmill test was performed 48 h after the incremental treadmill test, and according to the protocol described by Bouchard et al. (8). In brief, the test required the subjects to exert a maximal effort on the treadmill with the speed set at $3.31 \text{ m}\cdot\text{s}^{-1}$ and the grade set at 20%. Time to exhaustion (approximately 30–90 s) was the only variable measured.

Encouragement was given equally to all subjects for all treadmill tests. The protocols described above remained identical for each of the four testing periods. The timing in terms of day of week and time of day were consistent for each subject with a few exceptions to accommodate subjects' schedules.

The screening of the 121 subjects for [iMg] was conducted over 3 d. Three weeks later, the first testing session (T1) began. Each of the four testing sessions (T1, T2, T3, and T4) involved the following: Day 1 consisted of the distribution of dietary records (T1 and T3 only), and submission to anthropometric tests, resting heart rate and blood pressure measurement, and an incremental treadmill test with pretest, and 4, 10, and 30 min posttest blood withdrawal. Day 2 involved only the 24-h posttest blood withdrawal. Day 3 consisted of the anaerobic treadmill test and the dispensing of pills (Mg^{2+} supplement or placebo) (T1 and T3 only).

In a double-blind manner, subjects were randomly assigned to begin treatment with either $212 \text{ mg}\cdot\text{d}^{-1}$ (two pills of 106 mg elemental Mg^{2+}) Mg oxide (C. E. Jamieson and Company Ltd., Windsor, Ontario) or matching placebo. The placebo contained 222 mg dicalcium phosphate, 12 mg purified stearic acid, 6 mg coscarmellose sodium, 4 mg silicon dioxide, and 276 mg microcrystalline cellulose. Both the Mg^{2+} supplement and the placebo were in tablet form, and were identical in appearance, consistency, and taste. Subjects were instructed to consume the contents of one

TABLE 1. Characteristics of screened subjects ($N = 121$ subjects).

Parameter	Means \pm SD	Range
Age (yr)	21.5 ± 4.2	17–43
Height (m)	165.8 ± 6.2	151.5–178.0
Weight (kg)	63.2 ± 8.2	45.0–87.0
Resting [iMg] level ($\text{mmol}\cdot\text{L}^{-1}$)	0.54 ± 0.04	0.46–0.69
Systolic blood pressure (mm Hg)	109.9 ± 8.8	94–130
Diastolic blood pressure (mm Hg)	66.8 ± 7.9	50–90

tablet twice per day (one just before breakfast and one just before dinner). The treatment lasted 4 wk (28 ± 3 d), which was followed by a 6-wk (42 ± 3 d) washout period. Treatments were then reversed for the final 4 wk.

A randomized, double-blind, crossover (Mg oxide vs. placebo) design was employed. Testing (identical to that noted above) occurred every 4 wk (at T1, T2, T3, and T4). Subjects were divided into two treatment groups, one for which the order of pill administration was Mg^{2+} supplement first and placebo second (group M/P), and the other, *vice versa* (group P/M).

Statistical analysis. Correlational coefficients were used to establish relationships between the baseline measures of dietary intakes, hematological assays, and performance variables. The dietary intakes and training diaries were examined so that any discrepancies in routine would be taken into account. To test whether a carryover effect was present (i.e., if the washout period was ineffective) an independent *t*-test was performed on the [iMg] change scores from pre to posttreatment (5). Once the carry-over effect was disclaimed (see Results), the data were pooled to test for differences between treatment groups. The differences between treatments were measured using Student's *t*-test for paired samples on change scores from baseline values to treated values. To investigate whether training state affects the efficacy of Mg^{2+} supplementation, ANOVAs were run. Training state was classified as above or below the T1 mean using $\dot{V}\text{O}_2\text{max}$ values and again using time to exhaustion on the anaerobic treadmill test. The accepted level of significance was $P < 0.05$ for all statistical tests.

RESULTS

The characteristics of the 121 subjects screened for the study are listed in Table 1. Of the 121 originally screened for this study, 44 (or 36.4%) were marginally Mg^{2+} -deficient according to the criteria set by Altura et al. (4), i.e., they exhibited an [iMg] of less than $0.53 \text{ mmol}\cdot\text{L}^{-1}$. Most subjects maintained their training regimens throughout the study, although there was some seasonal fluctuation, especially as several did not exercise consistently during the washout period, which took place over the Christmas season. However, no differences in training, lifestyle, or dietary intake were apparent between treatment groups. Subjects participated in sporting activities, which were aerobic (most often running, cross-country skiing, and swimming) and/or anaerobic (most often weight training, soccer, basketball, and volleyball). All participants possessed regular menstrual patterns and none reported gastrointestinal distress throughout the study.

TABLE 2. Baseline characteristic of subjects completing entire study.

Parameter	All Subjects	Group M/P	Group P/M	t-Test Result
Age (yr)	21.2 ± 3.1	20.8 ± 1.8	21.4 ± 3.8	<i>t</i> (30) = -0.46
Height (cm)	165.5 ± 5.6	166.3 ± 7.2	165.0 ± 4.2	<i>t</i> (30) = 0.63
Weight (kg)	61.5 ± 5.3	61.9 ± 7.1	61.2 ± 3.8	<i>t</i> (30) = 0.34
Predicted percent body fat (%)	22.5 ± 4.0	23.1 ± 4.4	22.1 ± 3.8	<i>t</i> (30) = 0.70
Resting [iMg] level (mmol·L ⁻¹)	0.57 ± 0.03	0.56 ± 0.04	0.57 ± 0.03	<i>t</i> (30) = -1.04
Mg ²⁺ intake (mg·d ⁻¹)	320.4 ± 123.2	305.8 ± 133.5	325.6 ± 115.0	<i>t</i> (30) = -0.45
VO ₂ max (mL·min ⁻¹ ·kg ⁻¹)	49.8 ± 6.3	49.4 ± 6.8	50.2 ± 6.1	<i>t</i> (30) = -0.34
V _E peak (L·min)	109.2 ± 12.3	109.3 ± 11.1	109.2 ± 13.3	<i>t</i> (30) = 0.04
Maximum heart rate (beats per min)	195.1 ± 9.5	200.0 ± 7.2	193.9 ± 9.5	<i>t</i> (30) = 1.96
VO ₂ at an aerobic threshold (mL·min ⁻¹ ·kg ⁻¹)	37.4 ± 4.6	38.3 ± 4.9	36.7 ± 4.4	<i>t</i> (30) = 0.94
Anaerobic treadmill test result(s)	41.7 ± 14.3	41.0 ± 16.8	42.2 ± 12.8	<i>t</i> (26) = 0.24
Systolic blood pressure (mm Hg)	114.3 ± 8.9	116.3 ± 9.9	113.0 ± 8.1	<i>t</i> (30) = 1.04
Diastolic blood pressure (mm Hg)	69.4 ± 9.5	70.2 ± 12.4	69.0 ± 7.3	<i>t</i> (30) = 0.35

Group M/P, magnesium—1st, placebo—2nd (*N* = 13); Group P/M, placebo—1st, magnesium—2nd (*N* = 19). Total of 32 subjects or 30 for Mg²⁺ intake; means ± SD. Independent *t*-tests performed on treatment groups; * *P* < 0.05.

Before completing the study, seven subjects dropped out from group M/P (*N* = 13 as a result), and one dropped out from group P/M (*N* = 19 as a result). Of these eight individuals, two became injured, three became ill, and three experienced scheduling difficulties; none were included in the analysis.

Table 2 lists the characteristics of the 32 subjects completing the entire study as well as the characteristics of both treatment groups, M/P and P/M, with independent *t*-tests performed between groups. Because there were no significant differences between treatment groups on baseline measures, further analyses were justified. The mean dietary Mg²⁺ intakes were 320 ± 123 mg·d⁻¹ at T1 and 333 ± 127 mg·d⁻¹ at T2. Each of the two sets of dietary analyses revealed that six (or 20%) had intakes less than the Canadian Recommended nutrient intake (RNI) amount of 200 mg of Mg²⁺, and the same number had less than the 70% RDA amount. There was no correlation between Mg²⁺ intake and [iMg] (*r*₍₃₂₎ = -0.27) at T1.

In studies with crossover designs such as in this study, it is necessary to test for a carry-over effect, i.e., to see if the supplemental Mg²⁺ (or the placebo) still carried any effect from the first treatment period to the second treatment period. Armitage and Hills (5) also refer to this effect as the “treatment by period” interaction, where “treatment” is the factor representing the Mg²⁺ supplementation or placebo, and “period” is the factor representing the two periods of treatment. An independent *t*-test was performed between treatment groups on [iMg] change scores from pre to post-treatment (*t*₍₃₀₎ = -0.05, *P* > 0.05). Because the test did not reveal a treatment by period interaction, the carry-over effect was dismissed; thus, data from the two treatment periods were pooled (or combined) to form two categories—Mg²⁺-treated (M), and placebo-treated (P).

The lability of [iMg], as was noted in Methods, confounded its use as an independent variable. To highlight the lability of [iMg], in the 3-wk interval between the initial screening of potential subjects and T1, resting [iMg] significantly increased from 0.532 ± 0.046 mmol·L⁻¹ to 0.567 ± 0.032 mmol·L⁻¹, *t*₍₃₁₎ = 4.70; *P* < 0.05 (*N* = 32). Whereas 50% would have been classified as marginally deficient using the screening results, only 15.6% were marginally deficient at T1. The correlation for resting [iMg] levels

between screening and T1 was *r* = 0.45. Lability can also be highlighted by noting correlations between resting [iMg] levels from pretreadmill test to 24 h posttest and these were *r* = 0.55 (T1), *r* = 0.35 (T2), *r* = 0.80 (T3), and *r* = 0.59 (T4).

Despite lability concerns, pooled data revealed that the increase in resting [iMg] was significantly more for M (+0.044 mmol·L⁻¹) than P (+0.028 mmol·L⁻¹), *t*₍₃₁₎ = 2.28; *P* < 0.05 (see also Table 3). Performance and recovery index values did not differ significantly between treatments. However, a trend was indicated for V_Epeak values as they increased slightly more for M (+2.25 L·min⁻¹) vs P (-2.01 L·min⁻¹) (*t*₍₂₅₎ = 1.75; *P* = 0.09).

Pooled data revealed no differences in Mg²⁺ supplementation's affect between those with the higher and lower VO₂max values (*P* = 0.50) nor when using higher vs lower time to exhaustion scores on the anaerobic treadmill test (*P* = 0.60).

DISCUSSION

Results indicated that 4 wk of treatment with 212 mg·d⁻¹ Mg oxide was successful in raising resting [iMg] levels. Previous research has discovered slight but insignificant increases in [TMg] and [EMg] (after three weeks of supplementation with 387 mg·d⁻¹ Mg pidolate) (26). It may be that the [iMg] measure is more sensitive than [TMg] or [EMg] as previously claimed (2,3). Therefore, the use of this assay to assess the effects of Mg²⁺ treatment on performance and recovery may be advantageous over other hematological measures of Mg²⁺.

The significant increase in [iMg] levels from screening to T1 may be due to the subjects' increased awareness and interest about their nutritional habits from their involvement in such a study. Natural regression toward the mean may have also led to this increase. Because there were mostly weak correlations between screening and T1 [iMg] values and between pretest and 24 h posttest resting [iMg] values, it seems that these levels were fairly labile within individuals. The instrument itself does not appear to account for this as the precision (coefficient of variation) of the ion-selective electrode for Mg²⁺ has been reported to be excellent, i.e., less than 6% for control samples (4), and indeed quality control samples

TABLE 3. Physiological testing parameters by treatment.

Physiological Variable	Treatment	Raw Scores		Change		t-Test Result
		Baseline	Treated	Actual	%	
Resting [iMg] (mmol·L ⁻¹)	M	0.563 ± 0.045	0.607 ± 0.037	+0.044	+7.82	t(31) = 2.28*
	P	0.563 ± 0.038	0.591 ± 0.043	+0.028	+4.97	
V̇O _{2max} (mL·min ⁻¹ ·kg ⁻¹)	M	49.66 ± 6.54	50.70 ± 6.65	+1.04	+2.09	t(26) = 0.95
	P	49.77 ± 5.96	50.20 ± 6.69	+0.43	+0.86	
V _{Epeak} (L·min ⁻¹)	M	107.21 ± 13.59	109.46 ± 14.47	+2.25	+2.10	t(25) = 1.75
	P	107.54 ± 12.64	105.53 ± 13.95	-2.01	-1.87	
Maximal workload (% grade)	M	3.25 ± 2.63	3.13 ± 2.43	-0.12	-3.69	t(28) = -0.14
	P	3.66 ± 1.86	3.59 ± 2.35	-0.07	-1.91	
Maximal heart rate (beats per min)	M	197.00 ± 8.35	195.81 ± 7.84	-1.19	-0.60	t(28) = -0.66
	P	195.34 ± 9.54	195.52 ± 6.99	+0.18	+0.09	
V̇O ₂ at AT (mL·min ⁻¹ ·kg ⁻¹)	M	38.31 ± 3.95	39.12 ± 4.30	+0.81	+2.11	t(30) = 0.22
	P	38.16 ± 5.40	38.75 ± 4.20	+0.59	+1.55	
Anaerobic test (s)	M	36.54 ± 14.74	37.18 ± 13.50	+0.64	+1.75	t(27) = 1.06
	P	38.47 ± 11.87	37.80 ± 13.72	-0.67	-1.74	
Systolic BP (mm Hg)	M	115.09 ± 9.45	110.94 ± 7.72	-4.15	-3.61	t(31) = -1.26
	P	114.31 ± 8.53	112.69 ± 8.91	-1.62	-1.42	
Diastolic BP (mm Hg)	M	68.94 ± 9.03	68.00 ± 7.73	-0.94	-1.36	t(31) = -0.36
	P	68.84 ± 6.91	68.59 ± 7.80	-0.25	-0.36	

Pooled data based on treatment: M, magnesium; P, placebo. Baseline and Treated, tests performed before and after treatment, respectively (means ± SD). Student's *t*-tests performed on change scores; * *P* < 0.05.

and repeated sampling on the same blood yielded consistent values in the present study. Physiological effects could thus be deduced to be the cause of the variability, although at this point the source of the physiological perturbation is unknown. Some control was in place regarding previous exercise and diet including alcohol and caffeine consumption. The variability of [iMg] confounded clarification of the role Mg²⁺ status plays in the efficacy of Mg supplementation. The variability of [iMg] within individuals over time must be researched in more detail as the assessment, classification and diagnosis of Mg²⁺ status based on these values are put in jeopardy.

The mean dietary Mg²⁺ intakes of 320 ± 123 mg·d⁻¹ (for T1) and 333 ± 127 mg·d⁻¹ (for T2) are well above both the RNI of 200 mg·d⁻¹ and the RDA of 280 mg·d⁻¹. Altura (2) claimed that female subjects (without regard to activity level) ingest approximately 175–225 mg·d⁻¹ Mg²⁺ (60–80% of the RDA), whereas Lukaski (18) noted that female athletes take in about 168–182 mg·d⁻¹ of the mineral (60–65% of the RDA). It seems that participants in this study possess Mg²⁺ intakes that are higher than normal. Apart from being regular exercisers between the ages of 17 and 29, the subjects appeared to be quite diverse in regard to dietary habits. Some subjects gave conscientious effort to obtaining a healthy diet, whereas others tended toward highly refined convenience foods that often lack Mg²⁺ in adequate amounts. In addition, because there was no correlation between Mg²⁺ intakes and [iMg] levels ($r_{(32)} = -0.27$), it may be that there is a wide variability in the way in which Mg²⁺ is metabolized. This may have meant that the supplementation would have had less effect on some participants.

There were no significant effects of Mg²⁺ supplementation on performance during (or recovery from) aerobic or anaerobic exercise. Previous studies have discovered benefits of Mg²⁺ treatment on O₂ (10, 22) consumption at submaximal workloads, total workload (26), and time to exhaustion (9). Magnesium treatment has also been shown to improve submaximal performance by decreasing sub-

maximal V̇O₂, V_E, and HR (9, 21). Yet all of these studies involved higher dosages of Mg²⁺ supplementation and either used subjects with lower mean Mg²⁺ intakes or did not report intakes at all. Moreover, only Vecchiet and colleagues (26) incorporated the highly controlled crossover experimental design as utilized in this study. These choices may have contributed extensively to reporting significant effects. In addition, it is difficult to speculate on the possibility of Mg²⁺ deficiencies in the research of Brilla and Gunter (9) as well as that of Ripari and coworkers (21) because Mg²⁺ status was not assessed. The results of the present study are more in line with those of Ruddel et al. (23), Weight et al. (28), and Weller et al. (29). Weight and coworkers utilized a crossover design and a lower dose of Mg²⁺ (116 mg·d⁻¹) and found no significant effects on performance in subjects with high Mg²⁺ intakes (372 ± 122 mg·d⁻¹). The only study to specifically screen for a low [TMg] was that of Weller et al., and they did not find a benefit of Mg supplementation on exercise performance in subjects whose initial [TMg] values were in the low-normal range.

On the other hand, it is possible that the present study is lacking in certain areas that may have been critical for obtaining positive effects of Mg²⁺ supplementation. The relatively high mean Mg²⁺ intake of the participants and the relatively low dosage of Mg²⁺ supplementation may have prevented the opportunity for significant increases in absorption of the mineral in the body. Although this study built on previous research in the area by utilizing the sensitive [iMg] measure to assess Mg²⁺ status, the discovery that [iMg] appears to be rather labile compromises its usefulness (at least until further research can elucidate all of the physiological factors contributing to the variability). As well, because reference ranges for [iMg] levels have not yet been well established, it is difficult to confirm whether some subjects were initially Mg²⁺-deficient or not.

There appears to be nothing to suggest that initial training state (specifically V̇O_{2max} and anaerobic treadmill test

performance) affects the response to Mg^{2+} supplementation in this group of subjects. A review of the literature may have suggested otherwise because those studies revealing no effects with Mg^{2+} supplementation (23,25,27,29) all incorporated trained subjects, whereas those finding a positive effect (9,10,13,15,21,26) predominantly used untrained subjects. The homogeneity of this study's subject pool may not have permitted a clear distinction between the higher and lower training status groupings.

This study concluded that 4 wk of $212\text{ mg}\cdot\text{d}^{-1}$ Mg oxide supplementation significantly improved resting [iMg] levels

but not performance or recovery in a group of physically active females.

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REFERENCES

1. ALTURA, B. M. Basic biochemistry and physiology of magnesium: A brief review. *Magnesium and Trace Elements* 10:167–171, 1991–92.
2. ALTURA, B. M. Importance of magnesium in physiology and medicine and the need for ion sensitive electrodes. *Scand. J. Clin. Lab. Invest.* 54(Suppl. 217):83–89, 1994.
3. ALTURA, B. T., J. L. BURACK, R. Q. CRACCO, et al. Clinical studies with the NOVA ISE for iMg^{2+} . *Scand. J. Clin. Lab. Invest.* 54(Suppl. 217):53–68, 1994.
4. ALTURA, B. T., T. L. SHIREY, C. C. YOUNG, et al. Characterization of a new ion selective electrode for ionized magnesium in whole blood, plasma, serum, and aqueous samples. *Scand. J. Clin. Lab. Invest.* 54(Suppl. 217):21–36, 1994.
5. ARMITAGE, P., and M. HILLS. The two-period crossover trial. *The Statistician* 31:119–131, 1982.
6. BEAVER, W. L., K. WASSERMAN, and B. J. WHIPP. A new method for detecting anaerobic threshold by gas exchange. *J. Appl. Physiol.* 60:2020–2027, 1986.
7. BOHMER, D. Magnesium deficit: Relation to physical performance. In: *Magnesium and Physical Activity*, L. Vecchiet (Ed.). New York: Parthenon, 1995, pp. 149–156.
8. BOUCHARD, C., A. W. TAYLOR, and S. DULAC. Testing maximal anaerobic power and capacity. In: *Physiological Testing of the Elite Athlete*, J. D. MacDougall, H. A. Wenger, and H. J. Green (Eds.). Canada: Canadian Association of Sport Sciences, Mutual Press, 1982, pp. 61–74.
9. BRILLA, L. R., and K. B. GUNTER. Effect of magnesium supplementation on exercise time to exhaustion. *Med. Exerc. Nutr. Health* 4:230–233, 1995.
10. BRILLA, L. R., and T. F. HALEY. Effect of magnesium supplementation on strength training in humans. *J. Am. Coll. Nutr.* 11:326–329, 1992.
11. CANADIAN SOCIETY FOR EXERCISE PHYSIOLOGY. *Certified Fitness Appraisal Resource Manual*. Gloucester, ON: Canadian Society for Exercise Physiology, 1995, pp. 111–113.
12. DEUSTER, P., A., DOLEV, S. B. KYLE, R. A. ANDERSON, and E. B. SCHOOMAKER. Magnesium homeostasis during high-intensity anaerobic exercise in men. *J. Appl. Physiol.* 62:545–550, 1987.
13. DRAGANI, L., M. A. GIAMBERARDINO, and L. VECCHIET. Effects of magnesium administration on muscle damage from physical exercise. In: *Magnesium and Physical Activity*, L. Vecchiet (Ed.). New York: Parthenon, 1995, pp. 253–260.
14. NATIONAL RESEARCH COUNCIL (U.S.)/SUBCOMMITTEE ON THE TENTH EDITION OF THE RDAs. *Recommended Dietary Allowances*, 10th Ed. Washington, DC: National Academy Press, 1989.
15. GOLF, S. W., S. BENDER, and J. GRUTTNER. On the significance of magnesium in extreme physical stress. *Cardiovasc. Drugs Ther.* 12(Suppl. 2):197–202, 1998.
16. HEALTH AND WELFARE CANADA. *Nutrition recommendations: Report of the scientific review committee*. Ottawa, ON: Minister of Supply and Service Canada, 1990, pp. 136–140.
17. LUKASKI, H. C. Micronutrients (magnesium, zinc, and copper): are mineral supplements needed for athletes? *Int. J. Sport Nutr.* 5:S74–83, 1995.
18. LUKASKI, H. C. Prevention and treatment of magnesium deficiency in athletes. In: *Magnesium and Physical Activity*, L. Vecchiet (Ed.). New York: Parthenon, 1995, pp. 211–226.
19. MCDONALD, R., and C. L. KEEN. Iron, zinc and magnesium nutrition and athletic performance. *Sports Med.* 5:171–184, 1988.
20. RESINA, A., L. GATTESCHI, M. G. RUBENNI, et al. Changes in serum and erythrocyte magnesium after training and physical exercise. In: *Magnesium and Physical Activity*, L. Vecchiet (Ed.). New York: Parthenon, 1995, pp. 199–209.
21. RIPARI, P., G. PIERALISI, M. A. GIAMBERARDINO, A. RESINA, and L. VECCHIET. Effects of magnesium pidolate on some cardiorespiratory submaximal effort parameters. *Magnesium Res.* 2:70, 1989.
22. ROSE, L. I., D. R. CARROLL, S. L. LOWE, E. W. PETERSON, and K. H. COOPER. Serum electrolyte changes after marathon running. *J. Appl. Physiol.* 29:449–451, 1970.
23. RUDDER, H., C. WERNER, and H. ISING. Impact of magnesium supplementation on performance data in young swimmers. *Magnesium Res.* 3:103–107, 1990.
24. SEELIG, M. S. Consequences of magnesium deficiency on the enhancement of stress reactions: preventive and therapeutic implications (a review). *J. Am. Coll. Nutr.* 13:429–446, 1994.
25. TERBLANCHE, S., NOAKES, T. D., DENNIS, S. C., D. W. MARAIS, and M. ECKERT. Failure of magnesium supplementation to influence marathon running performance or recovery in magnesium replete subjects. *J. Sports Nutr.* 2:154–164, 1992.
26. VECCHIET, L., G. PIERALISI, M. D'OVIDIO, et al. Effects of magnesium supplementation on maximal and submaximal effort. In: *Magnesium and Physical Activity*, L. Vecchiet (Ed.). New York: Parthenon, 1995, pp. 227–237.
27. WEIGHT, L. M., K. H. MYBURGH, and T. D. NOAKES. Vitamin and mineral supplementation: effect on the running performance of trained athletes. *Am. J. Clin. Nutr.* 47:192–195, 1988.
28. WEIGHT, L. M., T. D. NOAKES, D. LABADARIOS, J. GRAVES, P. JACOBS, and P. A. BERMAN. Vitamin and mineral status of trained athletes including the effects of supplementation. *Am. J. Clin. Nutr.* 47:186–191, 1988.
29. WELLER, E., P. BACHERT, H. MEINCK, B. FRIEDMANN, P. BARTSCH, and H. MAIRBAURL. Lack of effect of oral Mg-supplementation on Mg in serum, blood cells, and calf muscle. *Med. Sci. Sports Exerc.* 30:1584–1591, 1998.