Vitamin and Mineral Supplementation and Neuromuscular Recovery after a Running Race

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1Laboratory of Biomechanics and Physiology, National Institute of the Sport and Physical Education, Paris, FRANCE; 2Faculty of Sport Sciences, University of Burgundy, Dijon, FRANCE; 3Laboratory of University of Toulon-Var, Unit Sporting Ergonomics and Performance, La Garde, FRANCE; and 4School of Human Movement and Exercise Science, University of Western Australia, Crawley, AUSTRALIA

ABSTRACT

GAUCHE, E. J., R. LEPERS, G. RABITA, J.-M. LEVEQUE, D. BISHOP, J. BRISSWALTER, and C. HAUSSWIRTH. Vitamin and Mineral Supplementation and Neuromuscular Recovery after a Running Race. Med. Sci. Sports Exerc., Vol. 38, No. 12, pp. 2110–2117, 2006. Purpose: This double-blind study investigated the effects of vitamin and mineral complex supplementation on the neuromuscular function of the knee-extensor muscles after a prolonged trail running race. Methods: Twenty-two well-trained endurance runners took either placebo (Pl group) or vitamins and minerals (Vm group) for 21 d before the race and for 2 d after the race. Maximal voluntary contractions (MVC) and surface EMG activity of the vastus lateralis (VL) muscle were recorded before (pre) and 1 h (post), 24 h (post 24) and 48 h (post 48) after the race. Central activation ratio (CAR), neural (M-wave), and contractile (muscular twitch) properties of the quadriceps muscles were analyzed using electrical stimulation techniques. Results: The knee-extensor MVC was significantly (P < 0.01) reduced after exercise for both groups (Vm: 36.5 ± 3.0%; Pl: 36.9 ± 2.1%), but MVC recovery was greater for Vm than Pl after 48 h (11%, P < 0.05). The reduced MVC after exercise was associated with a significant reduction in maximal EMG normalized to the M-wave in VL muscle and in CAR for both groups. Characteristics of the muscular twitch were not significantly altered for either groups, whereas M-wave duration increased significantly (P < 0.05) after exercise. Conclusions: The reduction of MVC immediately after the race appeared to result from peripheral mechanisms such as a failure in muscle membrane excitation and, to a lesser extent, from reduced central activation. The cause of the depressed MVC 24 h after the race seemed to be located within the muscle itself. A dietary supplementation of a vitamin and mineral complex does not attenuate the loss of contractile function immediately after the running exercise, and it may accelerate the recovery of maximal force capacity. Key Words: FATIGUE, MAXIMAL VOLUNTARY CONTRACTION, LONG-DURATION EXERCISE, MUSCULAR TWITCH, M-WAVE

Neuromuscular fatigue can be characterized by a transient reduction in maximal voluntary contractions (MVC) (2). Reductions in MVC of the quadriceps muscles have been found after long-duration exercises such as prolonged cycling and running exercises (16,23,27). Eccentric contractions occurring during prolonged running exercises are known to induce damage and to disrupt the contractile properties in human skeletal muscle (8). Indeed, muscle damage results in focal disruption of fiber ultrastructure (8), increased release of intramuscular enzymes, neutrophil and phagocyte infiltration, and loss of contractile performance (2).

Muscle fatigue can be caused by changes in neural and contractile properties of muscle (10). Reduction in quadriceps muscle MVC is frequently associated with a decrease of the integrated EMG (iEMG) in the vastus lateralis and vastus medialis muscles. However, a decrease of iEMG activity during maximal contractions is not always attributable to a decrease of maximal voluntary activation, because the sarcolemma excitability can be modified as well. In some cases, decreases in iEMG can be explained by changes of M-wave amplitude (16,23,27); however, there is evidence that central fatigue occurs after prolonged exercises (23,27). Mechanisms distal to the neuromuscular junction (peripheral mechanisms) can also contribute to muscle fatigue during and after long-duration exercise (27). However, there is no consensus about muscular peak twitch (Pt) changes after long-duration running exercise. Pt has been shown to increase by 18–19% after a 65-km ultramarathon (23) and a 5-h running exercise (27) but was reduced by 8% after a 30-km race (22). During prolonged exercise, metabolic alterations...
in the recruited fibers can also decrease the muscle force-generating capacity by inhibiting the excitation–contraction coupling process.

Nosaka et al. (24) have shown that MVC is negatively impacted by a number of different muscular injuries. Warren et al. (30) observed that free radical damage could be responsible for the muscular tissues damages that can be at the origin of the force loss. In this context, Mastaloudis et al. (18) suggested that endurance exercise causes oxidative stress (28). According to these observations, we can postulate that prolonged running exercise with an important eccentric component will produce free radical and neuromuscular damage, leading to a decrease of maximal strength capacities immediately after and in the days after the race. Antioxidant supplementation might provide beneficial effects against such exercise-induced oxidative tissue damage. Indeed, Maxwell et al. (20) suggested that 400 mg of vitamin C supplementation 3 wk before and 1 wk after exercise may exert a protective effect against eccentric exercise-induced cellular damage. A complex of vitamins and minerals is frequently used by athletes during training and competition periods. These complexes usually contain some substances recognized for their antioxidant effects (e.g., vitamin C, vitamin E, beta-carotene, zinc, selenium, manganese, cooper). Because antioxidants work together to protect organisms from potential side effects of free radicals (29), previous studies have attempted to investigate the efficiency of a combination of several antioxidants as supplements (3,11,19). However, information on the effects of a complex of vitamins and minerals containing antioxidants for preventing exercise-related muscle damage and neuromuscular alterations is limited. In this context, the aim of the present study was to examine the effect of supplementation with a complex of vitamins and minerals on neuromuscular recovery after a prolonged running race. Neural and contractile properties of the knee-extensor (KE) muscles were examined before, 1 h after, and for 2 d following a prolonged trail running race with large gradient changes in two groups of subjects ingesting either a placebo or a complex of vitamins and minerals for 21 d before and 2 d after the race.

METHODS
Approach to the Problem and Experimental Design

This experiment was conducted to examine the effects of supplementation with a complex of vitamins and minerals on neuromuscular function after a prolonged trail running race with significant uphill and downhill portions. Dependent variables in this study included maximal isometric voluntary torque of KE muscles and corresponding EMG activity. To examine the central and peripheral components of fatigue, transcutaneous stimulation of the femoral nerve was performed to analyze central activation ratio (CAR), muscular twitch, and M-waves. Measurements were performed before and 1, 24, and 48 h after the race. Dependent-variables data were analyzed as a function of independent variables (i.e., time and recovery interventions).

Subjects

Twenty-four well-trained endurance runners (age 39.7 ± 1.7 yr (SD); mass 71.2 ± 1.0 kg; height 176.5 ± 1.0 cm) volunteered to take part in this study after they were fully informed of the procedure and the risks involved in this study. Subjects provided a medical history and completed physical activity and diet and supplementation questionnaires to determine eligibility. None had orthopedic or metabolic conditions that could have affected the variables of measurement. No subject had used dietary supplements during at least the last 6 months before the experimental protocol. The subjects had regularly trained in running for 11.9 ± 1.5 yr before the study and had been running 80.8 ± 7.7 km-wk⁻¹ during the last 3 months preceding the experiment. Personal records for the marathon (42.195 km) were 178.7 ± 3.3 min. Written informed consent was obtained from the subjects, and the study was conducted according to the Declaration of Helsinki. This study was approved by the local ethics committee (St Germain en Laye, France) before its initiation.

Experimental Protocol

Preliminary session. During an initial session that took place 1 month before the experiment, each of the 24 subjects performed a continuous, incremental cycling test on an ergocycle (Lode, Excalibur, Groningen, The Netherlands). None of the subjects were regular cyclers. In accordance with the French Huriet’s law (1989), we were constrained to select a cycle ergometer protocol rather than a running protocol. Briefly, the test began with a warm-up at 100 W for 6 min, after which the power output was increased by 30 W every 1 min until volitional exhaustion. During this incremental exercise, oxygen uptake (\( \dot{V}O_2 \)), minute ventilation (\( V_E \)), and respiratory exchange ratio (\( RER \)) were continuously measured every 15 s using a telemetric system (Cosmed K4b², Roma, Italy). The criteria used for the determination of \( \dot{V}O_{2peak} \) were a plateau in \( \dot{V}O_2 \) despite an increase in power output, a RER above 1.1, and a heart rate (HR) above 90% of the predicted maximal HR (13). Maximal oxygen uptake (\( \dot{V}O_{2peak} \)) was the average of the last three highest \( \dot{V}O_2 \) values recorded (54.1 ± 1.5 mL·kg⁻¹·min⁻¹). The maximal aerobic power output (MAP) was the highest power completed for 1 min (342 ± 12 W). Twenty minutes after this test, subjects were familiarized with the isometric measurement apparatus and with the transcutaneous stimulation. This session took place before the supplementation period.

Randomization to treatment group. After the evaluation of \( \dot{V}O_{2peak} \), two groups of 12 subjects were randomly assigned in a double-blind manner to one of two treatment groups: vitamin and mineral supplementation (Vm) (Table 1) (Isoxan Endurance, NHS, Rungis,
TABLE 1. Component of isoxan endurance per day (A) and component of placebo (B) (NHS, Rungis, France).

<table>
<thead>
<tr>
<th></th>
<th>A</th>
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<tr>
<td></td>
<td>Vitamin C</td>
<td>Vitamin B8</td>
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<tr>
<td></td>
<td>200 mg</td>
<td>133 μg</td>
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<tr>
<td></td>
<td>Vitamin E</td>
<td>Vitamin B12</td>
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<td></td>
<td>32 mg</td>
<td>4 μg</td>
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<tr>
<td></td>
<td>Vitamin B5</td>
<td>Magnesium</td>
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<td></td>
<td>20 mg</td>
<td>173 mg</td>
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<td></td>
<td>Vitamin B3</td>
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<td></td>
<td>8 mg</td>
<td>19 mg</td>
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<td></td>
<td>Beta-carotene</td>
<td>Iron</td>
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<td></td>
<td>6 mg</td>
<td>13 mg</td>
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<td></td>
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<td></td>
<td>Vitamin B2</td>
<td>Copper</td>
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<td></td>
<td>5 mg</td>
<td>4 mg</td>
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<td></td>
<td>Vitamin B1</td>
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<td></td>
<td>4 mg</td>
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<td></td>
<td>Vitamin B9</td>
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<td>470 mg</td>
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<td></td>
<td>Lactose</td>
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<td>470 mg</td>
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<td>Reticulene</td>
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<td></td>
<td>Croscarmellose Na</td>
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<td>Magnesium stearate</td>
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<td></td>
<td>Talc</td>
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<td>Sepifilm LP014</td>
<td>33 mg</td>
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<td>3342 Sesisperse Dry</td>
<td>22 mg</td>
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FRANCE) or placebo (Pl) (Table 1). The treatments began 21 d before the race and continued for the 2 d of recovery after the race. The treatments continued during the 2 d after the race to maintain the same experimental condition that had been used during the protocol. All subjects were instructed to maintain their normal diets during the 23 d of supplementation. Capsule consumption, assessed by counting the capsules provided on return of pill bottles, was 99.3% for the Pl group and 99.5% for the Vm group. Isoxan Endurance is a vitamin and mineral supplement that was created by a scientific committee to answer the needs, contributions, and energy cost for athletes who practice long-duration exercise. The daily contribution of Isoxan Endurance was in accordance with the recommended daily allowances for athletes (17). However, the components of Isoxan Endurance per day are 25% greater than the recommended daily allowances because the race involves a significant energy cost for the subjects, defined as the ratio between oxygen uptake during exercise per unit of distance achieved (mL O2·km⁻¹·min⁻¹). The calculation of the energy cost of running (Cr) was done from the relationship between Cr (mL O2·km⁻¹·min⁻¹), speed (V, km·h⁻¹), and VO₂ (mL O2·km⁻¹·min⁻¹) during running (12): Cr = VO₂·V⁻¹. The energy cost of exercise was extrapolated (12), and the component doses of Isoxan Endurance were recomputed (17).

Running exercise. The event used in this study was a fatiguing exercise consisting of a 55-km trail race in the French Alps (6000D, La Plagne, France). The race includes a 6000-m vertical gradient (3000 m up and 3000 m down). The starting point and finishing line were at 694-m altitude, and the highest point of the race was at 3050 m. Because the fatiguing exercise was a race, each subject was well motivated to perform maximally over the distance. Two athletes did not finish the course, and two groups of 11 subjects were constituted for this study. Physical activity after the race was controlled (walking activities were limited, and massages were prohibited).

Neuromuscular performances. The neuromuscular tests, including voluntary and evoked contractions, were performed 2 d before (pre) and 1 h (post), 24 h (post 24), and 48 h (post 48) after the race.

MVC and CAR

Maximal isometric force of the right KE was recorded using an isometric ergometer chair (J. Scnell, Selephon, Germany) connected to a strain gauge (Enertec, Schlumberger, Villacoublay, France). Subjects were comfortably seated, and the strain gauge was securely strapped around the right ankle. The right knee angle was fixed at 100° (0° = knee fully extended). Extraneous movement of the upper body was limited by two harnesses across the chest and the abdomen. For each testing session, the subjects were asked to perform maximal isometric contractions (0 rad·s⁻¹) of 2- to 3-s duration with the KE muscles. The best performance of the three trials was defined as MVC. The subjects were strongly encouraged, and the three trials were executed with a 1-min rest period between each trial. The maximal voluntary activation level was estimated by the CAR method. An electrically evoked twitch was superimposed on the plateau reached during each MVC. The CAR was calculated as:

\[ \text{CAR} = \frac{\text{MVC}}{\text{MVC} + \text{amplitude of superimposed twitch}} \times 100 \]

Electrically Evoked Contractions

All neuromuscular tests were conducted on the right KE. Electrical stimulation was applied to the femoral nerve with a monopolar cathode ball electrode (0.5-cm diameter) pressed into the femoral triangle by the experimenter. A high-voltage stimulator (model DS7, Digitimer, Hertfordshire, United Kingdom) was used to deliver a square-wave pulse of 1-ms duration, 400-V maximal voltage, and intensity ranging from 50 to 110 mA. The optimal intensity of stimulation was set by progressively increasing the stimulus intensity until the maximal isometric twitch torque was achieved. The same intensity was used for the other testing sessions. The anode was a 50-cm² (10 × 5 cm) rectangular electrode (Medicomplex, Ecublens, Switzerland) located in the gluteal fold opposite the cathode. After the optimal intensity of stimulation was found, three single twitches, each separated by 2 s, were applied at rest; these served as control twitches.

The following parameters were obtained from the mechanical response of the evoked twitch: (1) peak twitch (Pt), that is, the highest value of twitch tension production; (2) contraction time (CT), that is, the time from the origin of the mechanical response to Pt; and (3) half relaxation time (HRT), that is, the time to obtain half of the decline in twitch maximal force.

EMG Recording

Electrical activity of the vastus lateralis (VL) and the antagonistic biceps femoris (BF) muscles were recorded by
means of bipolar silver/silver chloride surface electrodes (inter-electrode distance = 20 mm; area of electrode 50 mm²) positioned over the belly of the muscle when it was contracted. The skin was prepared by surface abrasion and cleaned with 33% ether, 33% acetone, and 33% alcohol. Low impedance (≤ 5 kΩ) was obtained by abrading the skin. The impedance was measured with a multimeter (Isotech IDM 93N). The two recording circular electrodes (Blue sensor Q-OO-S, Medicotest S.A.R.L, France) were fixed lengthwise approximately over the motor point. The reference electrode was attached to the kneecap of the right leg. To ensure that the electrodes were precisely at the same place for each testing session, we marked the electrode location on the skin with an indelible marker. Myoelectrical signals were amplified (G = 600) with a bandwidth frequency ranging from 6 Hz to 1.5 kHz. Before online digitization (sampling frequency, 1000 Hz), the signal was filtered with an antialiasing filter (0–500 Hz).

The root mean square (RMS) value was analyzed during the MVC of KE VL muscle for a 0.5-s period after the torque had reached a plateau. Peak-to-peak amplitude (PPA), peak-to-peak duration (PPD), and RMS of the M-wave (RMSM) were determined for the VL muscle during the control twitches performed before the MVC. MVC RMS values were then normalized to the RMS of the M wave for the VL muscle to obtain the RMS/RMSM ratio. This normalization procedure accounted for peripheral influences (neuromuscular propagation failure and/or changes in impedance) from the EMG recordings. A reduction in the MVC RMS without a reduction in RMSM was interpreted as a central activation failure.

Data processing was performed offline with commercialized data analysis and visualization software (Origin 6.1, OriginLab corporation, Northampton, MA; www.originlab.com).

### Statistical Analysis

All data presented are means ± SE (tables and figures). An independent two-way analysis of variance (ANOVA) with repeated measures was used to compare results between treatments and over time. Where significant F ratios were found, a Newman–Keuls significant difference test was used to determine the localization of the variance. For all statistical analyses, a P < 0.05 value was accepted as the level of significance. All the statistical analyses were performed with the Statistica 6.0 software for Windows.

### RESULTS

#### Subjects' performance

The time for the winner of the race was 248 min, and the average time for the subjects participating in the study was 413 ± 54 min, with no difference between the two groups (Pl: 417 ± 55 min, Vm: 454 ± 40 min). After exercise, the body mass loss was 2.9 ± 0.1 kg, which was equivalent to 3.8 ± 2.7% of body mass. There were no statistical differences between groups for body mass loss. During the race, the subjects consumed 4.0 ± 1.3 L of water (Pl: 3.8 ± 1.2 L, Vm: 4.2 ± 1.3 L) and ate 4.1 ± 2.4 PowerGel bars (PowerGel, Inc., Berkeley, CA) (Pl: 4.2 ± 2.6 PowerGel bars, Vm: 4.2 ± 2.4 PowerGel bars). There were no
statistical differences between groups for amount of water consumed or for the number of PowerGel bars eaten.

**Muscular strength**

Figure 1 shows that the KE MVC was significantly reduced after exercise (\(P < 0.01\)) to 63.5 ± 3.0% of the prerace value in the Vm group and to 63.1 ± 2.1% of the prerace value in the Pl group. The return to the preexercise values of the MVC was faster for the Vm group than for the Pl group. Compared with post values, the MVC after 24 h was 25.4 ± 4.4% and 19.8 ± 3.7% higher for Vm and Pl, respectively, with no difference between the two groups. Compared with post values, the MVC after 48 h had improved more for Vm (36.8 ± 4.4%) than for Pl (26.5 ± 3.4%).

**Muscular activity**

Figure 2A shows that compared with pre values, the maximal RMS/RMS\(_M\) of the VL muscle during the MVC decreased significantly (\(P < 0.01\)) from 100 to 52.6 ± 3.9% for Vm group and from 100 to 63.3 ± 2.9% for Pl group after the running exercise, remaining significantly (\(P < 0.01\)) lower during the next 48 h. The CAR was significantly reduced from 95 to 93% (\(P < 0.05\)) after exercise for the two treatment groups (Fig. 2B). Nevertheless, the central activation was not altered after 24 or 48 h for either group.

**Evoked twitch**

Changes in contractile parameters of the muscular twitch are shown in Table 2. Pt and CT were not significantly altered by the race, despite a tendency for Pt to be lower 24 h after the race. HRT remained stable immediately and 24 h after the race for both groups. For the Pl group only, HRT was significantly greater than pre values after 48 h (\(P < 0.05\)).

**M-wave**

PPA of the M-wave was not altered after exercise for either group (Table 3). In contrast, PPD increased significantly (\(P < 0.05\)) for both groups (Vm: 25.2 ± 0.6%, Pl: 7.4 ± 0.5%) after exercise. The changes in PPD after exercise were significantly greater (+17.8%, \(P < 0.05\)) for the Vm group than for the Pl group. PPD values after 24 and 48 h were not different from pre values for either group. RMS\(_M\) for VL muscle was not significantly altered after exercise for either group.

**DISCUSSION**

The main purpose of this research was to examine the effects of supplementation with a vitamin and mineral complex on neuromuscular recovery after a prolonged trail running race with large changes in gradient. The main findings of the present study were i) immediately after the race, a reduction of KE MVC (36%) associated with a reduced maximal RMS/RMS\(_M\) of VL muscle, but a smaller decrease of CAR; ii) no significant change of twitch properties for either treatment group after the race, but an increase of VL muscle M-wave duration that returned to prerace values after 24 h; and iii) a faster recovery of MVC at 48 h after exercise in the Vm group (36.8 ± 4.4% above the postexercise value) compared with the Pl group (only 26.5 ± 3.4% above postexercise).

The present results show that KE MVC decreased by 37% at the end of the race for both treatment groups, which is consistent with previous studies. For example, maximal isometric knee-extension force has been reported to decrease by 30% after a 65-km ultramarathon (23), by 24% after a 30-km running race (22), and by 28% after 5 h of treadmill running (27). The reduction of maximal KE strength in the present study appeared slightly greater than that observed in previous studies and could be explained by the greater changes in gradient (uphill and downhill) in the present race (3000 m up and 3000 m down throughout the 55 km), which might have induced greater muscle damage (1).

The method of superimposed stimulation during MVC used here had been successfully included as part of research investigating the level of voluntary muscle activation of the quadriceps muscle (16,23). The results showed a small, significant reduction of the CAR after exercise, suggesting that a central activation deficit could contribute in part to the decrease of maximal KE force immediately after the race. In addition, the reduction of the VL RMS/RMS\(_M\) observed after exercise suggests that neural drive to this muscle was diminished. The absence of measurements on the vastus

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**TABLE 1.** Values are means ± SE. *\(P < 0.05\), statistically significant compared with preexercise values.

<table>
<thead>
<tr>
<th>PPA (% pre)</th>
<th>Pre</th>
<th>Post</th>
<th>Post 24</th>
<th>Post 48</th>
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<tbody>
<tr>
<td>Pl</td>
<td>100</td>
<td>101.8 ± 7.7</td>
<td>125.8 ± 13.5</td>
<td>95.4 ± 12.8</td>
</tr>
<tr>
<td>Vm</td>
<td>100</td>
<td>103.4 ± 4.2</td>
<td>116.5 ± 18.2</td>
<td>100.4 ± 16.7</td>
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</table>

**TABLE 2.** Values are means ± SE. *\(P < 0.05\), statistically significant compared with preexercise values.

<table>
<thead>
<tr>
<th>PPD (% pre)</th>
<th>Pre</th>
<th>Post</th>
<th>Post 24</th>
<th>Post 48</th>
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<tbody>
<tr>
<td>Pl</td>
<td>100</td>
<td>107.4 ± 0.5</td>
<td>104.7 ± 0.7</td>
<td>97.2 ± 0.5</td>
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<tr>
<td>Vm</td>
<td>100</td>
<td>125.4 ± 0.6</td>
<td>102.5 ± 0.5</td>
<td>94.5 ± 0.6</td>
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**TABLE 3.** Values are means ± SE. *\(P < 0.05\), statistically significant compared with preexercise values.

<table>
<thead>
<tr>
<th>RMS (% pre)</th>
<th>Pre</th>
<th>Post</th>
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<tr>
<td>Pl</td>
<td>100</td>
<td>104.0 ± 1.0</td>
<td>95.9 ± 1.2</td>
<td>117.6 ± 1.7</td>
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<tr>
<td>Vm</td>
<td>100</td>
<td>88.6 ± 0.7</td>
<td>118.2 ± 1.3</td>
<td>119.5 ± 1.7</td>
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</table>
medialis and rectus femoris muscles in the present study does not allow us to speculate on changes in neural drive to these muscles. Central fatigue after long-duration running exercise has previously been shown to occur by several authors (23,27). As recently reviewed by Gandevia (10), central fatigue can originate from a supraspinal site and/or from the spinal level. Supraspinal fatigue after prolonged exercise has been linked to various hormones circulating in the cerebrospinal fluid. In addition, fatigue at the spinal level might result from peripheral reflex inhibition of the α-motoneuron pool and/or disfacilitation of the α-motoneurons by muscle-spindle afferents. It is interesting to note that 24 h after the race, CAR was no longer different from at preexercise, but in contrast, VL RMS/RMSM was still significantly depressed 24 and 48 h after the race for both groups, suggesting that neural drive to VL muscle was still reduced. VL RMS/RMSM corresponds to the activation of only one muscle of KE, whereas CAR represents the global activation of the whole muscle group. The recovery of CAR after 24 h while VL RMS/RMSM is still depressed may be explained by a lesser activation deficit of other KE muscles such as the vastus medialis and rectus femoris. These data suggest that recovery after prolonged exercise could vary among the KE muscles, indicating that a synergist is not representative of the activation pattern of a muscle group.

The unique aspect of the present study was the ability to make measurements on the subjects for 48 h after the race. The results showed that KE MVC values returned progressively to preexercise values for both treatment groups during the 48 h after the race, but MVC recovery was faster for the Vm group than the Pl group after 48 h. Previous experiments using muscle biopsies have shown that myofibrillar damage was more extensive in the days after the eccentric exercise than immediately after the exercise (9). Consequently, force loss after eccentric exercise could last more than 1 wk, especially in untrained subjects (7). However, in the present study, MVC had recovered in part after 24 h for both groups. This finding might be explained by the fact that our population of well-trained endurance runners regularly experienced eccentric contractions during their sessions. Indeed, muscle damage after eccentric exercise is repairable, and adaptation has been demonstrated in muscle that has been subjected to eccentric muscle contractions (25). For example, after repeated bouts of downhill running, muscle damage was reduced when the bout was repeated 3 or 6 wk later (5).

The mechanism to explain this rapid adaptation from one bout of exercise is unclear. Speculation on the mechanisms for such an adaptation have included damage and removal of a pool of susceptible or vulnerable fibers, increased ability to repair initial damage, and increased muscle connective-tissue content (15). Surface EMG recordings during evoked contractions on resting muscle have been used to explore possible changes in neuromuscular propagation. Indeed, M-wave is commonly used in human fatigue experiments as an index of neuromuscular transmission and action potential propagation in muscle fibers. No significant changes in VL M-wave amplitude were found after exercise. In contrast, M-wave duration of the VL muscle significantly increased immediately after the race in both treatment groups, suggesting a reduced conduction velocity of the compound muscle-action potential along the sarcolemma. An increase in VL M-wave duration has already been found in a previous experiment after a 30-km-long trail race (22). The lower muscle-fiber excitability could originate from a reduced chemical gradient for Na\(^+\) and K\(^+\) across the membrane. More specifically, it has been shown that prolonged exercise can lead to loss of K\(^+\) from working muscles, as reflected by the increased concentration of plasma K\(^+\) after marathon running (26), which could lead to possible alterations of sarcolemmal excitability or tubular system excitability (9). It should be noted that for both groups, M-wave duration returned to preexercise values after 24 h, suggesting that recovery of neuromuscular propagation in VL muscle after a long-duration running exercise is completed after 24 h.

In the present study, the twitch contractile properties were not significantly altered by the race, suggesting that the excitation–contraction coupling process was unchanged. Previous studies on long-duration running exercise (23,27) have reported an increase of peak twitch after exercise. The authors attributed the twitch potentiation to a myosin light-chain phosphorylation phenomenon as well as the stretch-shortening contractions of running, because twitch potentiation was not observed after prolonged cycling exercise (16). This latter result was unexpected because it has been shown that glycogen depletion was associated with depressed force, lower Ca\(^2+\) release, and inhibited contractile protein (6), and because of muscle damage. In the present study, the coexistence of potentiation and fatigue could explain the stability of peak twitch observed after exercise. Supplementation with a vitamin and mineral complex did not influence the excitation–contraction coupling process, because no difference was found between the two groups. Previous findings have suggested that contractile performance changes after eccentric exercise-induced muscle damage are marked by a substantial loss of MVC. According to Byrd (4), through the mechanism(s) involved in the disruption of sarcoplasmic reticulum (SR) function after eccentric exercise, oxidative stress and free-radical injury have been implicated in the loss of MVC process. The primary cellular defect seems to be one of excitation–contraction coupling, specifically through failure of transverse tubule–SR communication and/or calcium release by the SR.

In conditions of oxidative stress such as a trail running race, more oxygen radicals are produced, exceeding the cellular antioxidant system and resulting in the peroxidation of polyunsaturated fatty acids in membrane structures (19). Therefore, it has been shown that free-radical injury is an important factor in the origins of exercise-induced muscle damage, and that antioxidant supplementation might attenuate the loss of muscle function. Recently, Mastaloudis et al. (19) investigated the effect of a nutritional dose of 300 mg of vitamin E and 1000 mg of vitamin C during a training period. The antioxidant supplementation failed to attenuate the exercise-induced reduction in.
muscle performance. Nevertheless, they concluded that this form of supplementation reinforces antioxidant status, limits oxidative stress during acute exercise, and prevents lipid peroxidation. Moreover, vitamin E, vitamin C, selenium, and glutathione present in the vitamin and mineral complex used here are very important in controlling the toxic effects of free radicals (28).

By dietary manipulation, several studies have investigated the effects of elevated vitamin E and vitamin C on exercise-induced cellular damage and performance (21). Vitamin E and vitamin C are dietary sources of exogenous antioxidant that have been shown to reduce lipid peroxidation (21). Maxwell et al. (20) suggests that prior vitamin C supplementation may exert a protective effect against eccentric exercise-induced cellular damage. In this context, vitamin C is thought to act in combination with glutathione to protect vital cell structures such as the SR from oxygen radical attack at the surface of the membrane. On the basis of the preceding studies, it should not be surprising that we found a significantly faster recovery of MVC after 48 h of recovery with our vitamin and mineral supplement.

Our results showed that KE MVC values had decreased by 37% at the end of the race for both treatment groups. Jakeman and Maxwell (14) observed similar results after eccentric contractions in the triceps surae of 24 young subjects. These authors showed that MVC decreased to 75% of the initial level in both the placebo and vitamin E–supplemented (400 mg·d<sup>-1</sup> for 21 d) groups. Warren et al. (30) suggested that antioxidant supplementation did not reduce eccentric injury. Indeed, they showed that a dietary supplementation of antioxidants did not attenuate the loss of contractile function immediately after the running exercise. However, antioxidant supplementation could prevent exercise-induced oxidative stress and protect against both oxidative damage and inflammation (28). We can suggest that the protective effect of antioxidants present in the vitamin and mineral complex may have contributed to the faster recovery of MVC and, thus, less damage for Vm than PI after 48 h. Based on our results, more studies are needed to clarify the exact mechanisms responsible for the faster force recovery after a severe running exercise with a vitamin and mineral complex supplementation. If these findings are confirmed, it would be of great interest for athletes to have a vitamin and mineral complex supplement, especially when they experience several races in a short period of time. However we can not exclude that the 2 d of supplementation after exercise might also have contributed to the faster recovery of MVC. Indeed, Bloomer et al. (3) have reported little change in MVC with combined antioxidant treatment, and some studies (3,11) have shown a protection against oxidative stress markers or inflammation markers, but not functional recovery.

**CONCLUSION**

In conclusion, supplementation with a vitamin and mineral complex did not reduce the alterations of the maximal force-generating capacity of the KE muscles that resulted from both peripheral and central mechanisms after a prolonged trail running race. The MVC capacity was still depressed after 24 h, whereas M-wave and central activation returned to pre values, suggesting that the cause of the depressed MVC after 24 h was located within the muscle itself. Interestingly, dietary supplementation of vitamins and minerals did not attenuate the loss of strength immediately after the prolonged running exercise, but it did elicit a modest improvement in MVC recovery at 48 h after the race. Nevertheless, further studies are needed to 1) confirm a faster muscular force recovery after a severe eccentric running exercise during supplementation with vitamins and minerals, and 2) clarify the mechanisms that might underly such a benefit.

This research received financial support from the NHS laboratory located in Rungis (Val-de-Marne, France). We are also grateful to the help of Dr. Le Van P, Dr Vallier J.M, Dr. Joussellin (head of the medical department of INSEP), and Guilleminot CM, for the preparation of the completion of this project.

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