Carbohydrate supplementation affects blood granulocyte and monocyte trafficking but not function after 2.5 h of running

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ABSTRACT This randomized, double-blind, placebo-controlled study was designed to determine the influence of carbohydrate supplementation on the granulocyte and monocyte response to 2.5 h of high-intensity running [76.7 ± 0.4% of maximal oxygen consumption (VO2max)]. Thirty experienced marathon runners (VO2max 53.4 ± 1.0 mL·kg⁻¹·min⁻¹, age 41.5 ± 1.4 y) were randomly assigned to carbohydrate-supplement (n = 17) and placebo (n = 13) groups. Subjects rested for 10–15 min before a blood sample was taken at 0715, and then ingested 0.75 L carbohydrate beverage or placebo. At 0730 subjects began running at 75–80% of VO2max for 2.5 h, and drank 0.25 L carbohydrate or placebo fluid every 15 min. Immediately after the 2.5-h run (1000), another blood sample was taken, followed by 1.5-h, 3-h, and 6-h recovery samples. Carbohydrate supplementation had a significant effect compared with placebo on the pattern of change in plasma glucose and cortisol, and the blood concentration of neutrophils (F[4, 112] = 5.13, P = 0.001) and monocytes (F[4, 112] = 4.78, P = 0.001), but not on blood granulocyte and monocyte phagocytosis or oxidative burst activity after 2.5 h of intensive running. Am J Clin Nutr 1997;66:153–9.

KEY WORDS Immune system, lymphocytes, glucose, cortisol, phagocytosis, oxidative burst, granulocytes, monocytes, carbohydrate beverage supplementation, marathon running, runners

INTRODUCTION

In previous research conducted on marathon runners, we documented that various measures of immune function, including natural killer cell cytotoxic activity and mitogen-induced lymphocyte proliferation, are depressed for ≥ 3–6 h after prolonged, intensive running (1–6). Endurance running is also associated with sustained neutrophilia, mononcytosis, and lymphopenia, which have been related to elevations in epinephrine and cortisol (4, 7).

Shephard and Shek (8) proposed that nutritional status may modulate the interaction between exercise and immune function in several ways. Theories include 1) a more direct competition between the metabolic needs of the immune cells and the demands of the exercising muscles (eg, when muscle glycogen reserves are depleted and a competition develops for key amino acids), and 2) an alleviation of the potential adverse effects on the immune system of reactive species generated by metabolism or tissue injury (eg, antioxidant supplementation).

Only a few studies have been conducted on the role of nutritional supplementation in the immune response to intense and prolonged exercise, but the results thus far indicate that zinc (9) and glutamine (10) may attenuate some of the negative immune changes that take place during recovery. Vitamin C supplementation, however, has been shown to have no effect on the acute immune response to prolonged running (5).

Carbohydrate compared with water or placebo ingestion during prolonged endurance exercise has been associated with higher blood glucose and lower cortisol and epinephrine responses (11–14). Because of the important role of these stress hormones in regulating the trafficking of immune cells, it is possible that carbohydrate ingestion during prolonged and intensive running may alter the immune response through its influence on blood glucose, cortisol, and epinephrine. This may have important implications for endurance athletes who suffer an increased risk of upper respiratory tract infections after heavy training or marathon-type competitive events (15, 16).

Neutrophils (55–65% of blood leukocytes) and monocytes (3–9% of blood leukocyte) play an important role in nonspecific or innate immunity. These phagocytes act as first-line-of-defense cells to eliminate infectious agents, and are involved in the muscle tissue inflammatory response to exercise-induced injury (17–19). Relatively few studies have been conducted to define the response of these phagocytic cells, especially monocytes, to prolonged intensive exercise. In general, studies indicate that heavy exertion increases the phagocytic activity of neutrophils, but decreases their oxidative burst activity, one of the final steps in the killing process of foreign pathogens (18,

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19). Although epinephrine and cortisol have important effects on the trafficking of neutrophils and monocytes during and after exercise, there is uncertainty regarding the direct or indirect role these stress hormones have on their phagocytic and oxidative burst activity (18-22).

Given the potential role of carbohydrate supplementation in affecting the stress hormone, and therefore the neutrophil and monocyte response to exercise, we designed a randomized, double-blind, placebo-controlled study to investigate the influence of supplemental carbohydrate on the immune response to 2.5 h of intensive running.

SUBJECTS AND METHODS

Subjects

Thirty experienced marathon runners (24 males and 6 females) were recruited who met the following selection criteria: 25–59 y of age, marathon race time of < 4 h within the previous year, average training distance of ≥ 30 km/wk during the previous year, completion of at least two marathon race events, and ≥ 4 y of running experience. Males and females did not differ significantly in maximal aerobic power (54.0 ± 1.0 and 50.9 ± 2.5 mL·kg⁻¹·min⁻¹, respectively), training distance (63.9 ± 3.7 and 66.3 ± 10.3 km/wk, respectively), or ability to perform during the 2.5-h treadmill run [76.7 ± 0.5% and 76.7 ± 0.6% of maximal oxygen uptake (VO₂ max), respectively]. Males and females also did not differ significantly in any of the immune variables measured at baseline or in the pattern of response after the 2.5-h run. Data for the carbohydrate and placebo groups are thus presented for males and females combined. Subjects voluntarily signed an informed consent statement approved by the University Institutional Review Board for Human Studies.

Experimental design

Runners were unevenly randomly assigned into carbohydrate-supplement (n = 17) or placebo (n = 13) groups to account for a greater potential variability in immune responses among runners in the carbohydrate group. Runners from each group recorded food intake for 3 d before the simulated marathon run, choosing foods from a list that ensured a carbohydrate intake of ~60% of total energy. Energy intake was not controlled and runners were instructed to eat the amount of food they typically ingest before marathon race events. Nutrient intake was assessed by using the computerized dietary analysis system FOOD PROCESSOR PLUS (version 6.0; ESHA Research, Salem, OR).

The marathon runners were tested during the months of May and June to avoid confounding due to circannual effects on the immune system. During their first appointment height, weight, body composition, and maximal cardiopulmonary fitness were measured. Body composition was assessed by hydrostatic weighing and VO₂ max was determined by using a graded maximal treadmill protocol (23, 24). Oxygen uptake and ventilation were measured by using the MedGraphics CPX metabolic system (MedGraphics Corporation, St Paul). Maximal heart rate was measured by using the Quinton Q4000 Stress Test System (Quinton Instrument Co, Seattle). Training history and demographic factors were assessed through a questionnaire.

During their second appointment, subjects reported to the Human Performance Laboratory in a 12-h fasted and rested condition at 0700. All subjects indicated that they had avoided intensive exercise for ≥ 12–15 h and that they were healthy and free of symptoms associated with respiratory infections. After they rested quietly for 10–15 min, a blood sample was taken from each subject. Runners consumed 0.75 L of a 6% carbohydrate (Gatorade; Quaker Oats Company, Barrington, IL) or placebo beverage before the run. The beverages were prepared by the Gatorade Sports Science Institute. Treatments were double blinded, and the carbohydrate and placebo beverages were identical in appearance and taste. Except for carbohydrate concentration, the two fluids were identical in sodium (~19.0 mmol/L) and potassium (~3.0 mol/L) concentration, and pH (~3.0).

The marathoners ran on treadmills from 0730 to 1000 (24-h system) at a pace adjusted to elicit a workload approximating 75–80% of VO₂ max. Metabolic and heart rate measurements were made every 20 min during the run to ensure that subjects were maintaining the appropriate workload. Runners ingested 0.250 L carbohydrate or placebo fluid every 15 min during the run. Immediately after the 2.5-h run at 1000, blood samples were obtained from the runners, followed by samples taken at 1130, 1300, and 1600. Subjects drank 500 mL/h carbohydrate or placebo fluid during the first 1.5 h of recovery and then 250 mL/h during the last 4.5 h of recovery. After the blood sampling at 1130, subjects ate a meal ad libitum, choosing foods from the same food list they had adhered to during the 3 d before the study.

Immune measurements

Five blood samples were drawn per subject from an antecubital vein with subjects in the seated position (after 10–15 min of rest except for the immediate postrun sample). Routine complete blood counts were made by our clinical hematology laboratory staff with a Coulter STKS instrument (Coulter Electronics, Inc, Hialeah, FL).

The leukocyte phagocytosis assay used fluorescein isothiocyanate (FITC)-labeled bacteria (Staphylococcus aureus; Molecular Probes, Eugene, OR) to quantify the degree of phagocytosis by granulocytes and monocytes, as we described previously (25). To determine the extent of oxidative burst activity exhibited by granulocytes and monocytes, we used 2',7'-dichlorodihydrofluorescein diacetate (DCF-DA; Molecular Probes), a nonfluorescent molecule that is oxidized to green fluorescent dichlorofluorescein as oxygen radicals are generated in the oxidative burst to kill unlabeled S. aureus. Two-color flow cytometric immunophenotyping (CD45-FITC/CD14-PE) was used to determine percentages of monocytes and granulocytes. Bioparticle reagents of unlabeled and labeled S. aureus were suspended in phosphate-buffered saline at a working concentration of 3 × 10¹¹ bioparticles/L. After determining the number of phagocytic cells in 100 μL whole blood and adding 15 FITC-labeled bacteria per cell, the mean channel fluorescence (FITC) was analyzed to determine the degree of engulfed bacteria (nonphagocytized bacteria were quenched with ethidium bromide; final concentration of 200 μmol/L). To determine the oxidative burst activities, either DCF-DA (final concentration of 100 μmol/L) (basal activity level) or DCF-DA and unlabeled bacteria (stimulated activity level) were added to 100 μL whole blood. After incubating the samples for 60 min
(37 °C) in the dark, lysing the red blood cells, centrifuging for 20 min at 3000 × g and 5°C, and resuspending the pellets, the samples were acquired on the flow cytometer. For each sample, 10,000 phagocytes (monocytes and granulocytes) were acquired. Monocyte and granulocyte populations were analyzed individually for the extent of their phagocytosis and oxidative burst.

**Hormones, glucose, and plasma volume**

Plasma cortisol was assayed by using a competitive solid-phase 125I radioimmunoassay technique (Diagnostic Products Corporation, Los Angeles). For plasma epinephrine, blood samples were drawn into chilled evacuated tubes containing EGTA and glutathione (#RPN532 evacuated tubes; Amersham, Arlington Heights, IL), centrifuged, and the plasma stored at −80°C until analyzed. Plasma concentrations of epinephrine (samples before the run, immediately after the run, and 1.5 h after the run) were determined by HPLC with electrochemical detection (1, 26). Plasma was analyzed spectrophotometrically for glucose (samples before the run, immediately after the run, and 1.5 h after the run) (27). Plasma volume changes were determined by using the method of Dill and Costill (28).

**Statistical analysis**

Data are expressed as means ± SEs and were analyzed by using SPSS/PC+ (SPSS Inc, Chicago). Leukocyte subsets, hormone values, and all immune function measures were analyzed by using 2 (carbohydrate and placebo groups) × 3 or 5 (times of measurement) repeated-measures analysis of variance with interaction statistics reported. The change from baseline for the immediate, postexercise, 1.5-h, 3-h, and 6-h recovery values was compared between groups by using Student’s *t* tests. For some variables, all 30 subjects were combined, and within-group changes were tested by using paired *t* tests. For these four multiple comparisons a Bonferroni adjustment was made with statistical significance set at *P* ≤ 0.0125 and values between this and 0.05 treated as trends. Pearson product-moment correlations for glucose, cortisol, epinephrine, and various immune measures were calculated within the group of marathon runners to test the strength of these associations.

**RESULTS**

Subject characteristics for the runners in the carbohydrate and placebo groups are summarized in Table 1. Groups did not differ significantly in any of the training and fitness variables measured. The 30 marathon runners together were characterized as nonelite but highly experienced and committed to marathon running.

Nutrient analysis of the 3-d food records revealed no significant differences between groups. Energy intake for all subjects combined was 9908 ± 477 kcal/d (2368 ± 114 kcal/d); the proportion provided by carbohydrate was 62.6 ± 1.5%, by fat was 22.3 ± 1.2%, and by protein was 15.7 ± 0.4%.

The carbohydrate and placebo groups did not differ significantly in any of the performance measures taken during the 2.5-h run except for the average respiratory exchange ratio (0.93 ± 0.01 and 0.89 ± 0.01, respectively, *P* = 0.009) and the ending rating of perceived exertion made using the 6–20 scale (14.8 ± 0.3 and 16.3 ± 0.4, respectively, *P* = 0.004). As a group, the 30 marathon runners averaged 11.9 ± 0.2 km/h during the 2.5-h run at a heart rate of 151 ± 2 beats/min or 85.5 ± 0.5% of the maximum heart rate, an oxygen uptake of 40.9 ± 0.8 mL·kg⁻¹·min⁻¹ or 76.7 ± 0.4% of VO₂max, a ventilation of 89.3 ± 3.2 L/min or 63.2 ± 1.4% of maximal ventilation, and a breath rate of 43.4 ± 1.6 breaths/min. The laboratory temperature averaged 23.8 ± 0.2°C, with a relative humidity of 51.9 ± 0.6%. All runners consumed fluids according to the research design, including 2.5 L during the 2.5-h run. The average runner lost 0.35 ± 0.14 kg body weight (0.4 ± 0.2%). Plasma volume changes were minimal and the pattern of change over all time points did not differ significantly between the two groups (*F*₁, ₂₆ = 0.90, *P* = 0.453); for the pre- to immediate postexercise period, plasma volume changes for the carbohydrate and placebo groups were only −1.5 ± 0.4% and −1.0 ± 0.4%, respectively.

Significant group × time interaction statistics were found for neutrophils, lymphocytes, and monocytes (Table 2). For the carbohydrate group, postrun neutrophilia, immediate postrun lymphocytosis, 3-h postrun lymphopenia, and immediate postrun monocytosis were less pronounced than for the placebo group.

The patterns of change over time between carbohydrate and placebo groups for blood monocyte (*F*<sub>4, 112</sub> = 1.09, *P* = 0.367) and granulocyte (*F*<sub>4, 112</sub> = 0.27, *P* = 0.896) phagocytosis, and monocyte (*F*<sub>4, 112</sub> = 0.46, *P* = 0.763) and granulocyte (*F*<sub>4, 112</sub> = 0.49, *P* = 0.746) oxidative burst activity were not significantly different (Table 3 and Figure 1). For both groups combined, a 6-h sustained 50–74% postrun increase in monocyte phagocytosis, 35–44% increase in granulocyte phagocytosis, and a 6-h postrun 13.6% decrease in granulocyte oxidative burst activity were measured (all within-group changes, *P* < 0.01).

The patterns of change over time between groups for serum cortisol (*F*<sub>4, 25</sub> = 3.46, *P* = 0.022; Figure 2) and plasma glucose (*F*<sub>2, 27</sub> = 10.0, *P* = 0.001, but not plasma epinephrine (*F*<sub>2, 26</sub> = 1.94, *P* = 0.164) were significantly different (Table 4), with cortisol higher and glucose concentrations lower in the placebo group after the 2.5-h run.
TABLE 2
Response of blood leukocyte subsets in carbohydrate and placebo groups before and after 2.5-h treadmill running at 76.7 ± 0.4% of maximal oxygen uptake

<table>
<thead>
<tr>
<th>Leukocyte subset</th>
<th>Prerun (0715)</th>
<th>Postrun (1000)</th>
<th>1.5-h postrun (1130)</th>
<th>3-h postrun (1300)</th>
<th>6-h postrun (1600)</th>
<th>Group × time P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neutrophils</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbohydrate (n = 17)</td>
<td>2.99 ± 0.23</td>
<td>5.98 ± 0.43(^1)</td>
<td>7.78 ± 0.23(^1)</td>
<td>8.44 ± 0.49</td>
<td>6.54 ± 0.38</td>
<td>0.001</td>
</tr>
<tr>
<td>Placebo (n = 13)</td>
<td>2.56 ± 0.27</td>
<td>8.03 ± 0.83</td>
<td>8.95 ± 0.76</td>
<td>9.15 ± 0.75</td>
<td>6.63 ± 0.45</td>
<td></td>
</tr>
<tr>
<td>Lymphocytes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbohydrate (n = 17)</td>
<td>1.69 ± 0.13</td>
<td>1.78 ± 0.14(^2)</td>
<td>1.28 ± 0.09</td>
<td>1.62 ± 0.10(^2)</td>
<td>1.89 ± 0.11</td>
<td>0.004</td>
</tr>
<tr>
<td>Placebo (n = 13)</td>
<td>1.85 ± 0.14</td>
<td>2.46 ± 0.17</td>
<td>1.39 ± 0.20</td>
<td>1.36 ± 0.20</td>
<td>1.89 ± 0.16</td>
<td></td>
</tr>
<tr>
<td>Monocytes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbohydrate (n = 17)</td>
<td>0.38 ± 0.03</td>
<td>0.46 ± 0.05(^3)</td>
<td>0.59 ± 0.06</td>
<td>0.63 ± 0.06</td>
<td>0.59 ± 0.04(^4)</td>
<td>0.001</td>
</tr>
<tr>
<td>Placebo (n = 13)</td>
<td>0.39 ± 0.03</td>
<td>0.63 ± 0.07</td>
<td>0.63 ± 0.07</td>
<td>0.54 ± 0.07</td>
<td>0.51 ± 0.05</td>
<td></td>
</tr>
</tbody>
</table>

\(^1\)SE.
\(^2\)Significantly change from prerun between groups: \(^\star\) P < 0.0125, \(^\star\) P < 0.05.

The immediate postrun plasma glucose concentration correlated negatively with plasma cortisol \((r = -0.67, P < 0.001)\) and epinephrine \((r = -0.54, P = 0.002)\). The immediate postrun plasma cortisol concentration correlated positively with the neutrophil cell count \((r = 0.38, P = 0.04)\), but not with the monocyte cell count. The postrun plasma cortisol concentration (average of immediate and 1.5-h postrun concentrations) correlated negatively with the 1.5-h postrun \((r = -0.43, P = 0.018)\) and 3-h postrun \((r = -0.57, P = 0.001)\) blood lymphocyte concentrations. No significant correlations between postrun cortisol and monocyte and granulocyte phagocytosis and oxidative burst activity were found.

DISCUSSION

In this randomized, double-blind, placebo-controlled study, carbohydrate supplementation significantly altered the pattern of change in plasma glucose and cortisol, and blood neutrophil and monocyte cell concentrations, but not in phagocytosis or oxidative burst activity after 2.5 h of intensive running.

Prolonged, intensive exercise is associated with a sustained elevation in blood neutrophils and monocytes and a drop in lymphocytes, changes that have been reported since early in this century \((1-7)\). Granulocytes and monocytes play a central role in host defense, and these phagocytes are the first cells to reach and congregate at the site of infection \((17-19)\). Despite their importance, relatively few investigations have been conducted on the effect of intensive endurance exercise on granulocyte and monocyte function, a curious deficiency given the frequently reported link between heavy exertion and upper respiratory tract infection \((7, 15, 16)\). We found that 2.5 h of high-intensity running strongly increased granulocyte and monocyte phagocytosis of \(S. aureus\) bacteria throughout 6 h of recovery. Oxidative burst activity (stimulated by the bacteria), however, fell 13.6% by 6 h of recovery for granulocytes, while remaining unchanged for monocytes. This finding of granulocyte and monocyte activation but decreased oxidative burst after heavy exertion is consistent with the literature in general \((17, 18)\). Of interest is the growing consensus among investigators that although both moderate- and high-intensity exercise are associated with a sustained increase in blood granulocyte and monocyte phagocytosis and degranulation, only moderate exercise (typically ≤ 60% of \(V_{\text{O}_2}\text{max}\)) tends to enhance oxidative burst activity whereas high-intensity exercise often has the opposite effect \((29-39)\).

However, this may be explained by the fact that these measurements have been made in peripheral blood rather than in specimens taken from the respiratory tract where they might accumulate and function quite differently. Other confounding factors are time and technique. Varying amounts of time that span from when cells are called into action to the time they are studied, and the various methods of collecting cells and the differing techniques used to assess their function all affect the data outcome. It is important to remember that cells of the innate immune system, unlike T

TABLE 3
Blood granulocyte and monocyte oxidative burst activity in carbohydrate and placebo groups after 2.5 h of treadmill running

<table>
<thead>
<tr>
<th>Monocytes</th>
<th>Prerun (0715)</th>
<th>Postrun (1000)</th>
<th>1.5-h postrun (1130)</th>
<th>3-h postrun (1300)</th>
<th>6-h postrun (1600)</th>
<th>Mean fluorescence channel, DCF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate (n = 17)</td>
<td>31.7 ± 2.1</td>
<td>27.9 ± 1.9</td>
<td>36.8 ± 3.1</td>
<td>34.0 ± 2.2</td>
<td>30.0 ± 1.8</td>
<td>445 ± 27</td>
</tr>
<tr>
<td>Placebo (n = 13)</td>
<td>32.9 ± 3.7</td>
<td>30.5 ± 3.1</td>
<td>40.2 ± 4.3</td>
<td>37.3 ± 2.7</td>
<td>28.3 ± 1.8</td>
<td>476 ± 18</td>
</tr>
<tr>
<td>Granulocytes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbohydrate (n = 17)</td>
<td>445 ± 27</td>
<td>476 ± 18</td>
<td>414 ± 24</td>
<td>427 ± 30</td>
<td>376 ± 20</td>
<td></td>
</tr>
<tr>
<td>Placebo (n = 13)</td>
<td>422 ± 24</td>
<td>440 ± 23</td>
<td>417 ± 30</td>
<td>421 ± 33</td>
<td>377 ± 32</td>
<td></td>
</tr>
</tbody>
</table>

\(^1\)SE, DCF, dichlorofluorescin.
FIGURE 1. Pattern of change in blood monocyte or granulocyte phagocytosis for the carbohydrate and placebo groups over time. No significant differences were found. FITC, fluorescein isothiocyanate.

and B lymphocytes, respond rapidly to their immediate local environment. The increased number of these cells in circulation in the early postexercise response (<6 h) of our subjects may be preceding a migration to the respiratory tract tissues, a migration that is responsible for delivering large numbers of cells to that compartment where they can then respond to local instructions delivered by tissue cytokines. Of interest is the report by Müns (36) that phagocytes from nasal lavage samples in runners after heavy exertion have a reduced phagocytic capacity for ≥1 d. Together the
data indicate that after heavy exertion blood phagocytes may have an increased phagocytic capacity (perhaps an initial inflammatory response), but after migrating to the upper respiratory tract, receive local signals that suppress their activity.

The factors that regulate these disparate responses have still not been defined clearly. After prolonged, intensive exercise muscle cells are injured, prompting an inflammatory acute phase response with involvement by neutrophils, monocytes, macrophages, and cytokines (18, 29, 31, 37). Neutrophils appear to infiltrate all metabolically active tissues after exercise, including the heart, liver, and skeletal muscle (29). Although cytokines are thought to mediate the immune response, various hormones, including cortisol, growth hormone, prolactin, and thyroxe have been shown to have some regulatory effect on the function of phagocytes (32, 33, 37, 38).

We sought to alter the hormonal response to heavy exertion through carbohydrate ingestion and then study whether the immune response of phagocytes was affected. Release of corticotropin and cortisol during exercise has been linked in part to decreases in blood glucose concentrations (11, 13, 14). That work showed that during and after prolonged cycling, blood glucose concentrations are higher and cortisol lower in carbohy- drate- compared with placebo-supplemented subjects. Carbohydrate ingestion appears to have a variable effect on the epinephrine response to exercise (13, 14). Although drops in blood glucose are not typical during and after long-distance running, the data from the present study suggest that carbohydrate ingestion (at a rate of 60 g/h during exercise) is associated with higher plasma glucose and lower plasma cortisol concentrations after 2.5 h of intensive running. For all subjects, there was a strong negative correlation between postrun plasma glucose and cortisol concentrations, with a moderately negative effect on epinephrine. The difference in postrun cortisol concentrations in the carbohydrate and placebo groups was enough to significantly alter the pattern of change in circulating leu- kocyte subsets but not blood granulocyte and monocyte function. It has not yet been determined whether functional changes occur in phagocytic function within the respiratory tract.

Smith et al. (33) were also able to alter the hormonal response to exercise through carbohydrate supplementation, but then failed to show that the pattern of change in blood neutrophil oxidative burst activity was affected after 1 h of cycle ergometer exercise. Thus, it would appear that factors other than hormones play a more important role in the regulation of granulocyte and monocyte phagocytosis and oxidative burst activity after heavy exertion.

In summary, carbohydrate supplementation before, during, and after 2.5 h of intensive running had a significant effect in raising plasma glucose concentrations, decreasing plasma cortisol, and attenuating changes in circulating leukocyte subsets. For 6 h after the running bout a strong increase in blood granulocyte and monocyte phagocytosis was measured,
whereas granulocyte oxidative burst activity fell slightly, changes that were unaffected by carbohydrate supplementation. Clearly, further studies are required to resolve the issues raised by our findings, particularly as they pertain to long-term consequences in health and disease.

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