Hormonal factors in reduced postprandial heat production of exercise-trained subjects

JACQUES LeBLANC, PIERRE DIAMOND, JACQUES CÔTÉ, AND ANTOINE LABRIE
Department of Physiology, School of Medicine, Laval University, Québec City, Québec G1K 7P4, Canada

LeBLANC, Jacques, Pierre Diamond, Jacques Côté, and Antoine Labrie. Hormonal factors in reduced postprandial heat production of exercise-trained subjects. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 56(3): 772-776, 1984.—The influence of exercise training on postprandial heat production was investigated in human subjects. Whereas resting metabolic rate was comparable for trained and nontrained subjects, the heat increment of feeding (HIF) after subjects consumed a meal containing 755 kcal was approximately 50% smaller in the trained subjects. Measurements of respiratory quotient also indicated a reduction of about 50% in glucose oxidation associated with exercise training. The levels of plasma norepinephrine increased significantly (P < 0.01) from 200 to 300 pg/ml in the sedentary subjects, but the changes observed in trained subjects were not significant. During the early phase of the meal, plasma levels of insulin were increased, even before nutrients appeared in the blood. Throughout the study the enhanced sensitivity to insulin of the trained subjects was confirmed. The postprandial heat production was diminished in exercise-trained subjects, and it is suggested that this could be related to a reduced activity of the sympathetic nervous system. Another possibility is that this reduction in HIF is related to a facilitation of glucose disposal in the form of glycogen rather than in the form of lipids.

RECENT STUDIES have indicated that the postprandial heat production cannot always be adequately equated with the so-called “specific dynamic action” of the ingested meal (6, 11, 18). For instance, it has been shown that ingesting 755 kcal in the form of a meal produces a much larger increase in the resting metabolic rate (RMR) than if these calories are fed by stomach tube (11). In another study, it has been reported that for a given meal the heat increment of feeding (HIF) is not the same in trained and nontrained subjects (10, 21). Similarly thermogenesis in the rat has been shown to be modified by exercise training (9, 12). Since some of these results were contradictory, the present study was designed to clarify this point. On the other hand, it seemed interesting from a strictly mechanistic or teleological point of view to find out whether exercise training modifies the postprandial increase in energy production. Indeed, it may be argued that if exercise training increases HIF, this action would be contributory to the gradual decrease of body fat associated with the habit of exercise; on the other hand, if training reduces HIF, this could be interpreted as a beneficial sparing effect on the requirement of calories that would be used in priority for the purpose of exercising. To relate our results to hormonal variations, comcomitant plasma catecholamines, glucose, and insulin levels were also determined.

METHODS

Seven trained and seven nontrained men (aged 20-30 yr) participated in this study. The trained subjects had all been long-distance runners for at least 3 yr and were presently running 100-160 km/wk. The subjects came to the laboratory at 8:30 A.M. and had been fasting since 10:00 P.M. of the preceding day. A cannula was then inserted into the median cephalic vein of the arm for future blood sampling. Two initial measurements of RMR were made over a period of 12 min, each separated by a 3-min interval. The volume of expired air was measured by an integrated pneumotacograph, and O2 (Beckman LB-2 analyzer) and CO2 (Allied Scientific) were monitored continuously; the results were averaged over the 12-min period. During this initial resting period, 10-ml blood samples were taken at -30 and -10 min and at time 0. The subjects were then asked to eat a meal containing 755 kcal and composed of a “submarine” sandwich, a piece of sugar pie, and a soft drink. The duration of the meal varied between 8 and 12 min. Twelve minutes after the beginning of the meal, RMR was measured every 15 min for a total of 90 min. Further blood samples were taken at 1, 2, 4, 8, 15, 30, 60, and 90 min after the beginning of the meal. To assess the portion of heat production that was due to the meal, the subjects were asked to come for a second test when all measurements were made except that food was not ingested. This second test from that of the first test, the value of the postprandial increase in heat production was calculated for each subject. These two tests were randomized to eliminate any biased results.

Plasma glucose was determined by enzymatic method (1), insulin by a radioimmunoassay (8) using pork insulin as standard, and catecholamines by a radioenzymatic assay (3). The results were analyzed by paired t test or by an analysis of variance and the
REDUCED POSTPRANDIAL HEAT PRODUCTION

Duncan's multiple-range test (4). Area under the curves was calculated according to the trapezoid method.

RESULTS

The characteristics of the subjects are given in Table 1. No significant differences were found in the premeal RMR between the two groups (3.51 ± 0.41 for trained and 3.68 ± 0.42 ml O₂·kg⁻¹·min⁻¹ for nontrained subjects). In spite of some variations, no changes in overall RMR were observed during the control experiment when food was not eaten. After the ingestion of food, an increase in RMR was observed for all subjects. To better control individual variations, the increments in RMR were determined for each subject by subtracting the results obtained during the control period from the meal period. The final statistical appraisal was made by integrating the increase in RMR over a period of 90 min for both groups of subjects.

After the ingestion of food, the integrated elevation of RMR was significantly greater in the sedentary than in the trained subjects (Fig. 1). A postprandial increase in respiratory quotient was observed, but no significant differences were detected between the two groups of subjects (Fig. 2). Within the first 2 min from the beginning of the meal, a small transient decrease in plasma glucose accompanied by an elevation of insulin was observed in the sedentary subjects. The decrease in plasma glucose was more pronounced in the trained subjects, and it lasted throughout the meal period; however, the increase in plasma insulin was smaller in the latter group (Fig. 3). During the 90 min that followed the meal, peak values for plasma glucose were observed at 30 min, and no differences were noted between the two groups of subjects.

The highest values for plasma insulin were also found at 30 min, and the levels were significantly lower throughout the period in the trained than in the sedentary subjects (Fig. 4). After the meal, plasma norepinephrine increased rapidly in the sedentary subjects as significant differences were observed at 1 (P < 0.05), 2 (P < 0.01), and 4 min (P < 0.05). Integrated plasma NE during the 90-min period was also significantly increased (P < 0.01), as indicated on Fig. 5. In the trained subjects no differences were observed at any time, and through a comparison of the integrated increase in NE for the whole period, a significant difference was observed between the groups of subjects. An overall decrease in epinephrine was observed in both groups of subjects (P < 0.01) as indicated on Fig. 5. The large variations among individuals in plasma catecholamine levels contribute to a widespread SE. To avoid confusion on Fig. 5, these SE are not indicated, and at any event they were not used to evaluate the statistical significance of the results; instead, paired testing was used to that effect. The results obtained for O₂ consumption and respiratory quotient were used to calculate the relative carbohydrate and lipid oxidation. Figure 6 shows that the postprandial increase in glucose oxidation was twice as high in the sedentary as in the exercise-trained subjects. Lipid oxidation also increased significantly after the meal, but in this case, no significant difference was noted between the two groups.

DISCUSSION

The present investigation shows that the increase in heat production that normally follows the ingestion of food is twice as much in nontrained compared with highly trained human subjects. A first conclusion that can be drawn from this study is that factors other than the composition of the diet can influence the calorigenic effect of a meal. It has been suggested that palatability

<table>
<thead>
<tr>
<th>Group</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>Percentage of Fat</th>
<th>V̇O₂max (ml·kg⁻¹·min⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trained</td>
<td>68.1 ± 1.8</td>
<td>176 ± 5</td>
<td>11.1 ± 1.9</td>
<td>62.5 ± 2.4*</td>
</tr>
<tr>
<td>Nontrained</td>
<td>70.4 ± 2.2</td>
<td>175 ± 7</td>
<td>14.6 ± 2.1</td>
<td>44.1 ± 5.5</td>
</tr>
</tbody>
</table>

Values are means ± SE. V̇O₂max, maximal O₂ uptake. * Significant difference between groups (P < 0.01).
could be one of these factors (15), and the results of the present study indicate that the level of training can also play a role in that respect. These results support the findings of some investigators who have concluded that the specific dynamic action of food cannot adequately explain the increased postprandial heat production (6, 11, 17, 18).

A decreased meal thermogenesis associated with exercise training was also found by Tremblay, Côté, and LeBlanc (21), whereas other investigators found opposite results (10). It was argued previously that the level of training of the subjects could explain the opposite results. Indeed, the trained subjects used in the latter study had a maximal \( \text{O}_2 \) uptake of 47 ml \( \text{O}_2 \)·kg\(^{-1}\)·min\(^{-1}\), whereas...
for the trained subjects of our two studies, maximal \(O_2\) uptake was above 60 ml \(O_2\) kg\(^{-1}\) min\(^{-1}\).

The reduction of postprandial heat produced in exercise-trained subjects compared with sedentary subjects is possibly two groups of subjects. Comparable reduction of NE secretion has been reported in trained subjects exercising at various work loads (2, 7). The reason for the reduction of postprandial and postexercise NE secretion in trained subjects is not known. The diminution of sympathetic activity associated with exercise training is, however, compatible with the reduction of postprandial heat production. It also seems possible that this reduction in HIF could be related to a facilitation of glucose transformation into glycogen (6), a less costly caloric process than storing it in the form of lipids (21).

Our results also indicate a significant reduction in the increased postprandial glucose oxidation in the trained subjects, whereas the increased lipid oxidation was comparable for both groups of subjects. Although these findings cannot be explained at this time, they may be important. In terms of energy balance, the diminished postprandial heat production and the reduced glucose oxidation observed in trained subjects indicate a possible increase in food efficiency and a sparing effect on glucose utilization in highly trained subjects.

As reported previously (13, 14), basal plasma insulin levels were found to be lower in trained than in sedentary subjects. Within a few minutes after the beginning of a meal, insulin is shown to increase, and this effect has been explained by a central action mediated by the vagal nerve (11, 16, 20). Although insulin increased in both groups during the meal period, it was larger in the sedentary subjects. At the same time, a decrease in plasma glucose was observed that was more pronounced in the trained subjects. Thus the larger decrease in glucose in the presence of a smaller increase in insulin observed in trained subjects suggests an enhanced insulin sensitivity associated with exercise training. Previous studies have also shown that the insulin requirement during a glucose tolerance test is significantly reduced in exercise-trained subjects (14, 15). During the postprandial period, reduced insulin levels were found in the trained subjects, but the glucose levels were not different between the two groups. Whether these differences in plasma glucose and insulin are directly involved in the differences in the postprandial heat production between trained and sedentary subjects cannot be estimated at this stage.

It is concluded that exercise training reduces the increase of both glucose oxidation and energy expenditure that is normally observed during the period that follows a meal. Results also suggest the direct or permissive effect of norepinephrine and insulin in these actions.

This work was supported by the Canadian Department of National Defence with consignation of the Defence and Civil Institute of Environmental Medicine, Downsview, Ontario, Canada.

J. LeBlanc and J. Côté are members of the Centre de Recherche en Activité Physique, Laval University, Québec City, Québec, Canada.

Received 1 August 1983; accepted in final form 13 October 1983.

For the trained subjects of our two studies, maximal \(O_2\) uptake was above 60 ml \(O_2\) kg\(^{-1}\) min\(^{-1}\).

The reduction of postprandial heat produced in exercise-trained subjects compared with sedentary subjects is possibly two groups of subjects. Comparable reduction of NE secretion has been reported in trained subjects exercising at various work loads (2, 7). The reason for the reduction of postprandial and postexercise NE secretion in trained subjects is not known. The diminution of sympathetic activity associated with exercise training is, however, compatible with the reduction of postprandial heat production. It also seems possible that this reduction in HIF could be related to a facilitation of glucose transformation into glycogen (6), a less costly caloric process than storing it in the form of lipids (21). Our results also indicate a significant reduction in the increased postprandial glucose oxidation in the trained subjects, whereas the increased lipid oxidation was comparable for both groups of subjects. Although these findings cannot be explained at this time, they may be important. In terms of energy balance, the diminished postprandial heat production and the reduced glucose oxidation observed in trained subjects indicate a possible increase in food efficiency and a sparing effect on glucose utilization in highly trained subjects. Related to these findings in humans, it has been found that exercise training in the rat reduces the thermogenesis that is associated with prolonged intake of high-energy diets (12). Admittedly the energy gain by this decrease in HIF observed in trained subjects is small, but it is not negligible even it means a difference of 100–150 kcal/day.

As reported previously (13, 14), basal plasma insulin levels were found to be lower in trained than in sedentary subjects. Within a few minutes after the beginning of a meal, insulin is shown to increase, and this effect has been explained by a central action mediated by the vagal nerve (11, 16, 20). Although insulin increased in both groups during the meal period, it was larger in the sedentary subjects. At the same time, a decrease in plasma glucose was observed that was more pronounced in the trained subjects. Thus the larger decrease in glucose in the presence of a smaller increase in insulin observed in trained subjects suggests an enhanced insulin sensitivity associated with exercise training. Previous studies have also shown that the insulin requirement during a glucose tolerance test is significantly reduced in exercise-trained subjects (14, 15). During the postprandial period, reduced insulin levels were found in the trained subjects, but the glucose levels were not different between the two groups. Whether these differences in plasma glucose and insulin are directly involved in the differences in the postprandial heat production between trained and sedentary subjects cannot be estimated at this stage.

It is concluded that exercise training reduces the increase of both glucose oxidation and energy expenditure that is normally observed during the period that follows a meal. Results also suggest the direct or permissive effect of norepinephrine and insulin in these actions.

This work was supported by the Canadian Department of National Defence with consignation of the Defence and Civil Institute of Environmental Medicine, Downsview, Ontario, Canada.

J. LeBlanc and J. Côté are members of the Centre de Recherche en Activité Physique, Laval University, Québec City, Québec, Canada.

Received 1 August 1983; accepted in final form 13 October 1983.

REFERENCES


7. HAGGENDAL J., L. H. HARTLEY, AND B. SALTIN. Arterial nora-


