Reduced postprandial heat production with gavage as compared with meal feeding in human subjects

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LeBlanc, Jacques, Michel Cabanac, and Pierre Samson. *Reduced postprandial heat production with gavage as compared with meal feeding in human subjects.* Am. J. Physiol. 246 (Endocrinol. Metab. 9): E95–E101, 1984.—The postprandial changes in resting metabolic rate (RMR) were measured in eight subjects after the ingestion of 735 kcal taken in the form of a meal or fed by stomach tube. A much larger increase in RMR was found with meal feeding (MF) as compared with tube feeding (TF). Measurements of respiratory quotient (RQ) indicated that the increase in RMR with MF is related to increased glucose oxidation, whereas with TF it is possibly explained primarily by the cost of energy storing. Determination of plasma norepinephrine (NE) indicated that the sympathetic nervous system is activated by MF but not by TF. Similarly, plasma glucose and insulin determinations have shown that the secretion of insulin is increased during food ingestion but only with the MF situation. Thus in the 30 min after the beginning of the meal, an increased glucose oxidation was observed that is possibly related to an enhanced NE and insulin secretion; such effects were nonexistent in the TF situation. These results would not substantiate the notion of the specific dynamic action of food and would indicate that independent of the composition of the ingested nutrients, the postprandial increase in RMR is also influenced by sensory and cognitive stimulations.

feeding; thermogenesis; palatability

IT HAS BEEN REPORTED that feeding rats through a tube placed into the stomach produced a significantly larger increase in body weight gain than allowing normal oral ingestion of food even if the animals received isocaloric diets in both situations (17). It has also been found that increasing caloric intake by as much as 80% by tube feeding did not produce the increased regulatory energy expenditure (diet-induced thermogenesis) (10) that has been described in rats consuming comparable large excess of calories in the form of palatable high-energy diets (16). More recently, LeBlanc et al. (11) have observed a diet-induced thermogenesis with palatable diets of various composition; at the same time it was shown that excess caloric intake was not essential for this action to take place because it was also observed in rats fed palatable diets with low caloric intake. In the present study, it is proposed to test whether the sensory inputs that accompany a meal might play a role in the postprandial increase in metabolic rate of humans. It is already known that insulin is rapidly released after a meal even before the nutrients are absorbed from the intestinal tract (4, 14, 20, 24). It was decided to investigate the insulin and catecholamine secretion and the increase in metabolic rate after the ingestion of a standard meal or after the subjects were given the caloric equivalent by means of a stomach tube, thus bypassing the olfactogustative prandial stimulation.

MATERIALS AND METHODS

Eight healthy men, aged 20–30 yr, and with average weight and height of 65 kg and 1.6 m, gave their written consent to participate in the experiment. Each subject was tested three times at 1-wk intervals and in a randomized order. For each of the tests, the subject came to the laboratory at 0830 h fasting since 2200 h of the preceding day. The subject sat quietly, and a cannula was inserted into the median cephalic vein of the arm for future blood sampling. Two initial measurements of resting metabolic rate (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 while continous O_2 (LB2 analyzer, Beckman) and CO_2 (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 exposed to one of the three following situations. The subjects either *a*) ate a meal, *b*) were fed through a stomach tube, or *c*) were given in control sessions a determined amount of water equivalent to the volume that was tube fed in *b*. All food items were kept at room temperature before being fed to the subjects. Twelve minutes after the beginning of the meal that lasted about 10 min, RMR was measured every 15 min for a total of 90 min. During the same time, blood was collected at 1, 2, 4, 8, 15, 30, 60, and 90 min after the beginning of the meal.

The postprandial heat production of the meal was calculated by substracting from the oxygen consumption measured in the 90-min postprandial period the values obtained when the subjects abstained from eating during the same period of control sessions. The postprandial heat production of tube feeding was calculated in the same manner. The meal contained 3,156 kJ (755 kcal) and was composed of a "submarine" sandwich, a piece of sugar pie, and a soft drink. In the test, during which the subjects were fed by tube (inserted through the nose into the stomach), the same amount of calories was given in the form of a commercial liquid diet prepared from a

mixture of 20% RCF and 80% Ensure (Mead Johnson). Table 1 shows the composition of the two diets. The calculation of caloric content of meals was made from information supplied by manufacturers or from food tables. When the subjects were tube fed, a large syringe was used to push the liquid into the stomach at a rate of 60 ml/min for a total time of 10 min. The same period was allowed when the subjects were eating a meal. Plasma glucose was determined by an enzymatic method (1), insulin by a radioimmunoassay (9) using pork insulin as standard, and catecholamines by a radioenzymatic assay (2).

The results were analyzed by paired t test or by an analysis of variance and the Duncan multiple-range test (3). Area under the curves was calculated according to the trapezoid method. These calculations were made by reference to the pretest levels as a basis for measurement of the effect of either tube or meal feeding.

RESULTS

The average RMR before eating was 3.61 ± 0.42 ml $O_2 \cdot kg^{-1} \cdot min^{-1}$. In the control situation, when the subjects drank only water, in spite of some individual variations, no significant changes in overall RMR were observed during the 90-min period. In the 90 min that followed food ingestion, the integrated elevation in RMR

TABLE 1. *Composition of meal and of food fed by tube*

Type of Feeding	Proteins, g	Lipids, g	Carbohydrates
Meal	26.5	38.1	70.2
Stomach tube	27.4	34.2	76.5

was three times larger when the subjects were meal fed as compared with when they were tube fed (Fig. 1). The average respiratory quotient (RQ) before eating was 0.85. In the 30 min that followed food ingestion, RQ rose significantly but only in the meal-fed situation. Between the 30- and 90-min postprandial period, the increase in RQ was comparable for both the meal- and tube-fed situation. The integrated change in RQ for the whole period was larger with meal than with tube feeding (Fig. 2).

With meal feeding, a significant increase in plasma insulin was observed at *min 2* and *4* from the beginning of the meal and a fall in plasma glucose at *min 1* and *2*. These early changes were not present with tube feeding, but both insulin and glucose increased significantly after 8 min from the beginning of feeding (Fig. 3). This increase is possibly due to the liquid form of the tube-fed diet. During the 15–90 min after both the meal and tube feeding, significant increases in glucose and insulin were observed as indicated on Fig. 4, but no significant differences were found between the two situations.

Large individual variations are observed in plasma NE. For that reason, paired t tests were used to evaluate the significance of the results. The elevation of plasma NE that took place during meal feeding was not observed with tube feeding. When the integrated NE response was calculated, the difference between the two situations was significant only during the feeding period (Fig. 5). A significant fall in plasma E concentration was observed during the feeding period but only in the meal-feeding condition as shown in Fig. 6.

The relative contribution of carbohydrates and lipids to oxygen consumption was estimated at various times
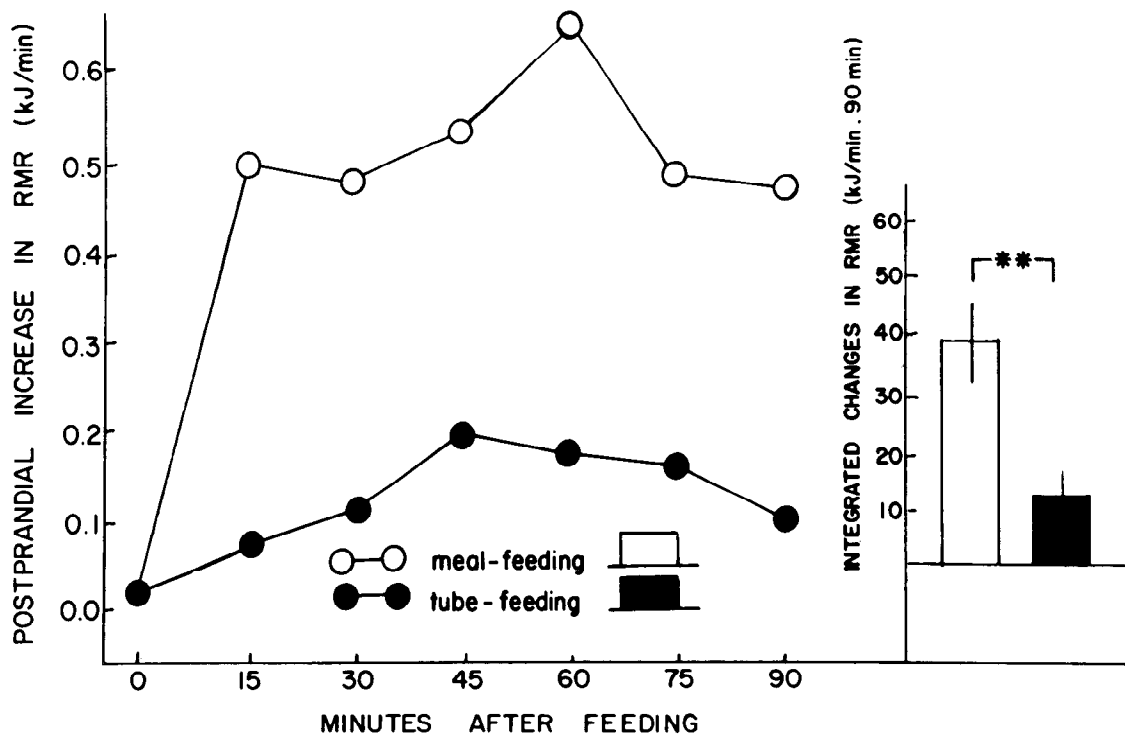

FIG. 1. Increase in resting metabolic rate (RMR) during 90 min after ingestion of 755 kcal fed in form of a meal or by stomach tube. Area under curve is represented as integrated changes in RMR. ** $P < 0.01$.
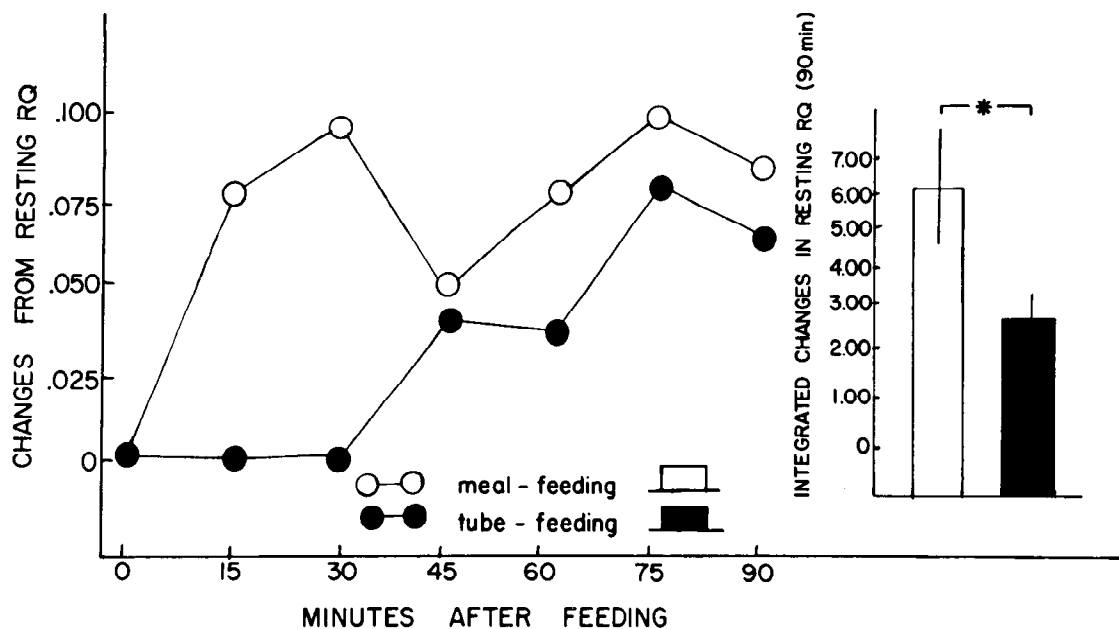
FIG. 2. Increase in respiratory quotient (RQ) during 90 min after ingestion of 755 kcal fed in form of a meal or by stomach tube. Area under curve is represented as integrated changes in RQ. * P < 0.05.
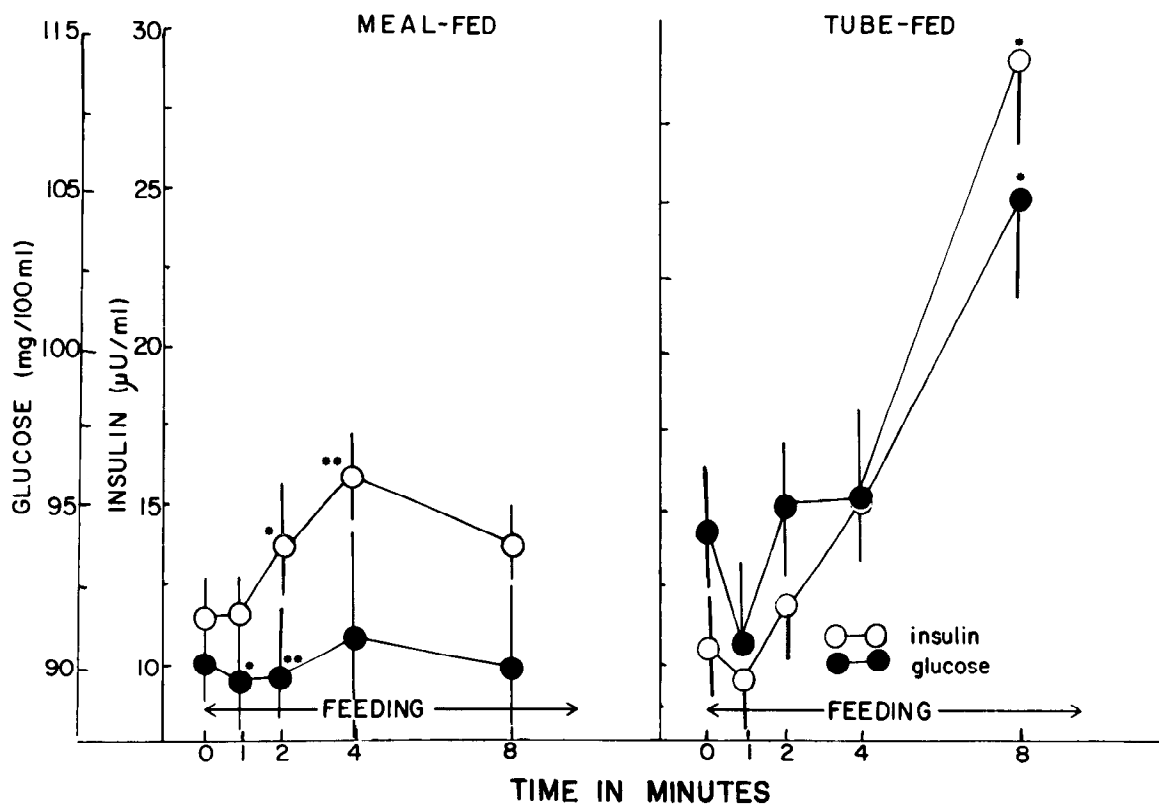
FIG. 3. Plasma glucose and insulin variations during 8 min after ingestion of 755 kcal fed in form of a meal or by stomach tube. * P < 0.05 and ** P < 0.01.

from the calculated RQ. As shown on Fig. 7, the oxidation of glucose was much larger than that of lipids in the postprandial period. Furthermore, although lipid oxidation was not significantly different between the two situations, glucose oxidation was three times larger after meal feeding than after tube feeding.

DISCUSSION

The present experiment shows that meals identical with regard to composition and caloric content produced

four times less heat when the food was placed directly into the stomach by means of a tube as compared with when it was ingested orally. These findings are surprising when reference is made to the classical notion that identifies the postprandial increased energy expenditure with the specific dynamic action (SDA) of food (6, 13, 18).

Obviously, the meal composition and its predicted SDA could not explain the marked differences observed in the above-mentioned situations. Our findings bring
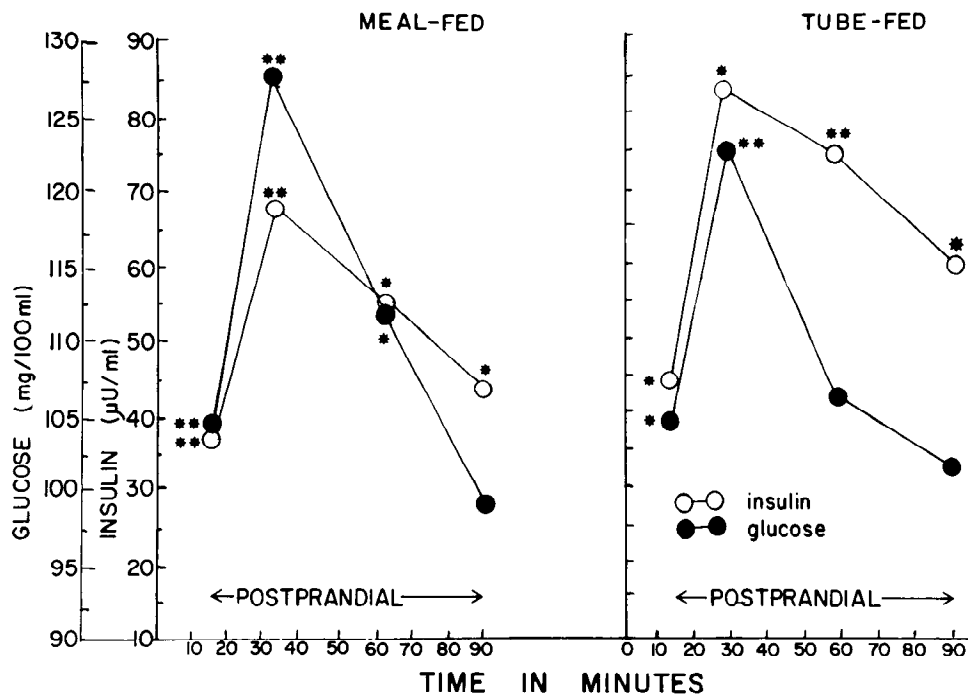
FIG. 4. Plasma glucose and insulin variations between 16 and 90 min after ingestion of 755 kcal fed in form of a meal or by stomach tube. * $P < 0.05$ and ** $P < 0.01$.
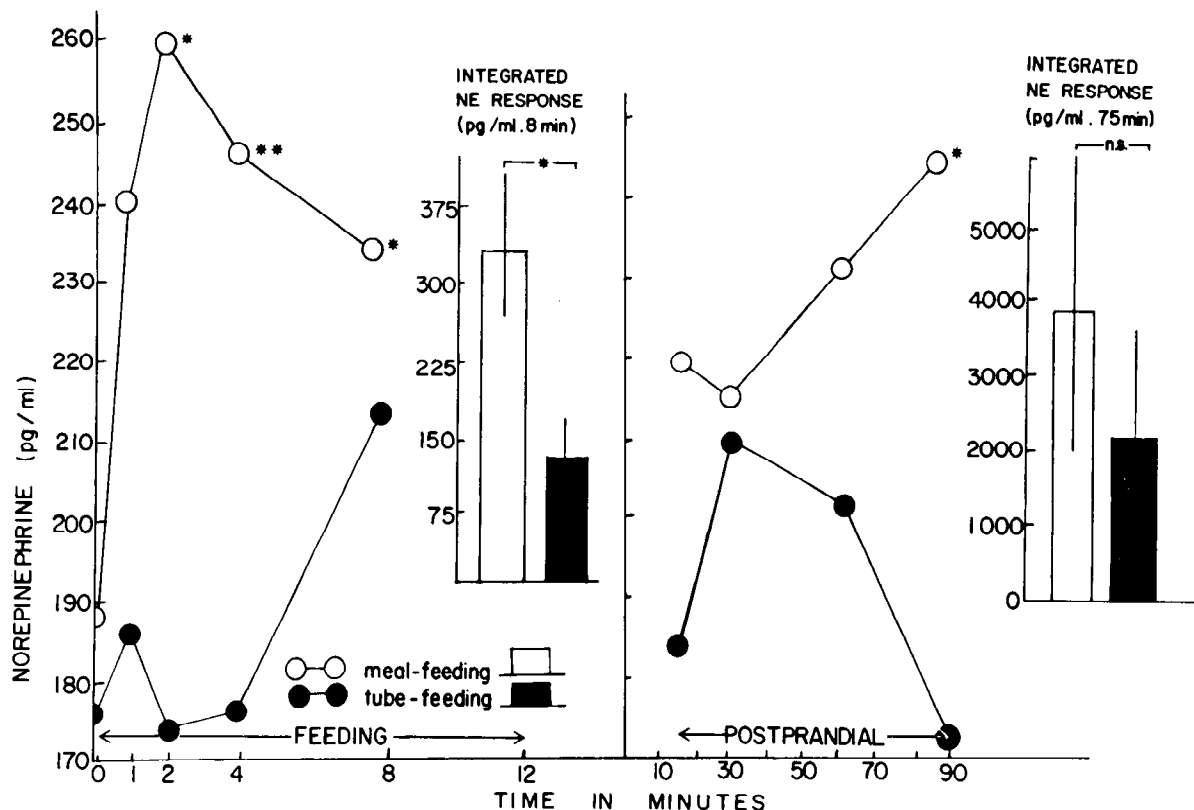

FIG. 5. Plasma norepinephrine (NE) variations in 90 min after ingestion of 755 kcal in form of a meal or by stomach tube. Area under curve is represented as integrated changes in NE. * $P < 0.01$ and NS, $P > 0.05$.

additional new evidence that concurs with the conclusions made in recent years by some investigators regarding the misleading notion of SDA of food (7). The explanation of SDA was based on a predominant contribution of proteins (30% RMR) five to six times larger than that of glucose and fat (6). These data obtained from experiments on animals overestimated the role of protein degradation or synthesis because studies on humans have

shown that a glucose, a gelatin, or a mixed glucose-albumin meal produced within 4 h identical increases of oxygen consumption on the order of 15% (7, 8, 15). Comparable results were found in our study in which postprandial heat production in the meal-fed condition corresponded to 8% of the calories consumed for a period of about 2 h.
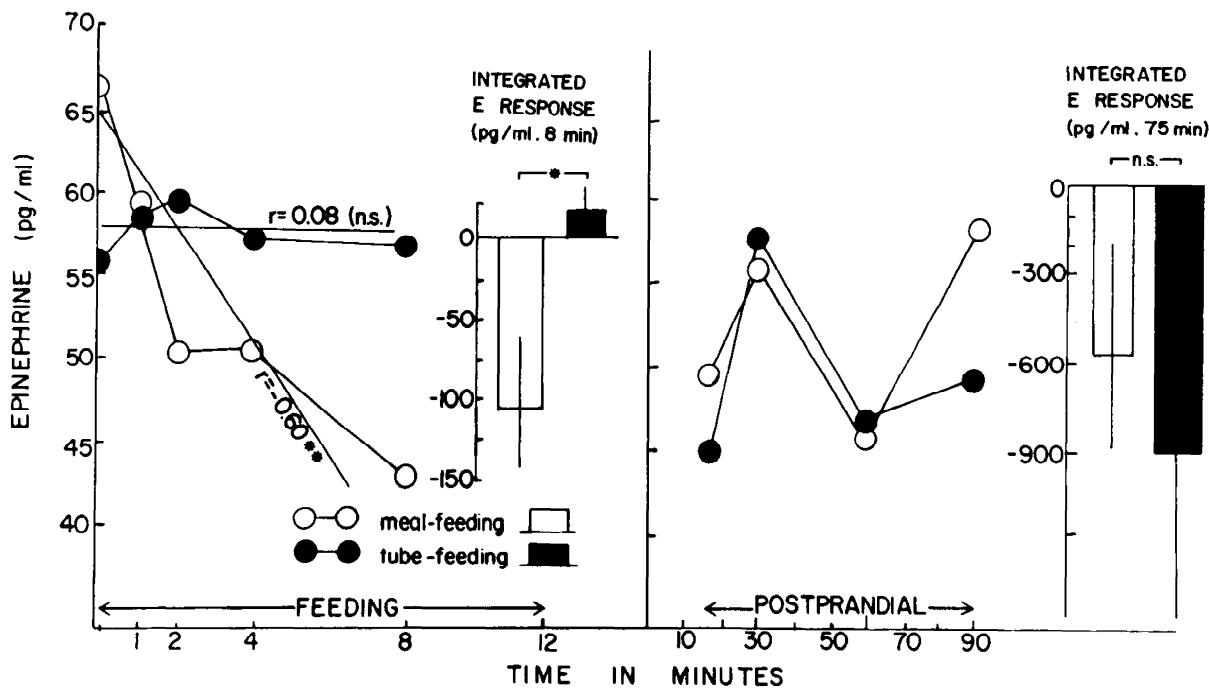
The postprandial heat production in tube-feeding ses-

FIG. 6. Plasma epinephrine (E) variations in 90 min after ingestion of 755 kcal in form of a meal or by stomach tube. Area under or above curve is represented as integrated changes in E. * $P < 0.05$ and ** $P < 0.01$.
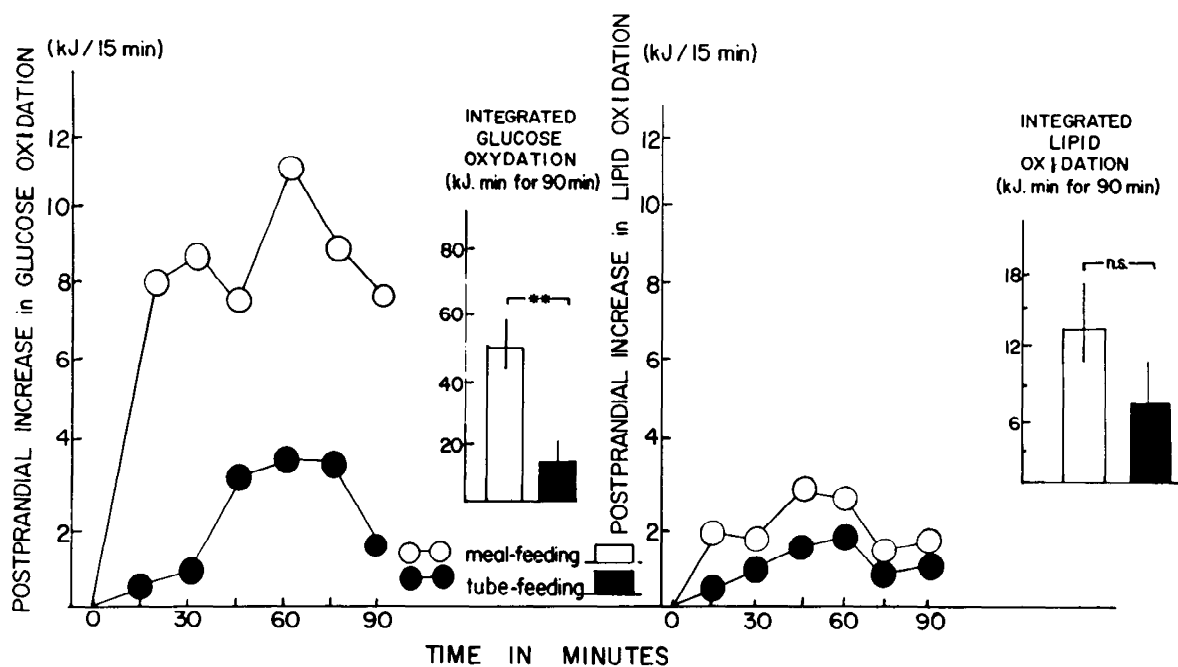

FIG. 7. Increase in glucose and lipid oxydation during 90 min after ingestion of 755 kcal fed in form of a meal or by stomach tube. Area under curve is represented as integrated increase in glucose or lipid oxydation. ** $P < 0.01$ and NS, $P > 0.05$.

sions was four times smaller than that of meal-feeding sessions and amounted to about 2% of RMR. At the same time, the oxidative activity differed greatly between the two situations as depicted by measurements of RQ. In the first 45 min after the beginning of the meal, according to calculations, the proportion of glucose oxidation increased from 50 to almost 80% in the meal-fed situation, whereas no changes were observed when the subjects were tube fed. During the 45- to 90-min postprandial period, although the RQ tended to be identical, the thermic effect of the food remained different in the oral and tube-fed situations. Our results would thus show that factors others than the nutrient composition of the diet must be evoked in order to explain the postprandial increase in oxygen consumption and changes of RQ. We have shown that bypassing the sensory, as well as cognitive input, normally associated with food intake greatly abolished the thermic effect of food. In other words, palatability associated with eating would seem to play a major role in postprandial heat production. Postprandial heat production would thus be an early systemic response to food intake (14) rather than specific dynamic action.

To sort out the relative importance of glucose and fat in the postprandial increase of oxidative metabolism, the following calculations were made. Assuming that comparable proportions of proteins were metabolized in the meal and the tube-fed situation, 15% of the thermic effect of the meal was substracted to take into account the effect of protein metabolism, and the remaining values were used to calculate the amounts of fat and glucose that were oxidized in both the meal- and tube-feeding situations. In terms of absolute values, four times more glucose and two times more fat were oxidized in the 1.5 h that followed the meal than during the same period when food was fed by a tube.

The results obtained with various hormonal determinations will be discussed in the light of these findings. Plasma norepinephrine has been shown to be significantly increased after glucose administration (21, 24), a finding that has been interpreted as evidence of increased sympathetic activity (22). With meal feeding, there was a rapid and sustained increase in NE that did not reach significant levels with tube feeding. Considering the well-known effect of NE on substrate mobilization and utilization, it would not seem impossible that differences in sympathetic activity might explain some of the differences between the thermic effect of a meal as compared with that of food taken by tube. Glucagon was not determined in our study, but considering its prominent role in glucose and fat metabolism and because its secretion is increased following a meal (19), it is possible that the above-mentioned results could also find their explanation in differences in the secretion of this hormone. Differences in insulin secretion were noted between meal and tube feeding. Within the first few minutes after the meal, insulin was found to increase and glucose to decrease. For that reason, an inverse relationship was found between these two variables as shown in Fig. 8. This early insulin release, also termed the cephalic insulin secretion, that accompanies a meal has been explained by a central hypothalamic action mediated by the vagal nerve because it is abolished by atropine (4, 14, 20). When the sensory input that normally accompanied a meal was suppressed as it is by tube feeding, these early effects on glucose and insulin were not found, and a positive relationship was observed between these two variables in the first 8 min after the beginning of tube feeding (Fig. 9). In this case, the elevation of plasma insulin was caused by the increase in plasma glucose, whereas with meal feeding it is the increase in insulin that caused the fall in plasma glucose. Similar findings have been reported recently in rats (12). In the period that followed food ingestion, values for glucose and insulin were not different between meal and tube feeding, a finding that could indicate that the plasma levels of these variables are not directly responsible for the differences in the thermic effect of food between the two situations described.

It is pertinent to ask what proportion of the postprandial increase in oxygen consumption is due to metabolic oxidation as compared with the cost of storing the various nutrients in the form of proteins, lipids, or glycogen reserves. Because the thermic effect of feeding may last 4 h or more (23), it would be difficult to calculate the
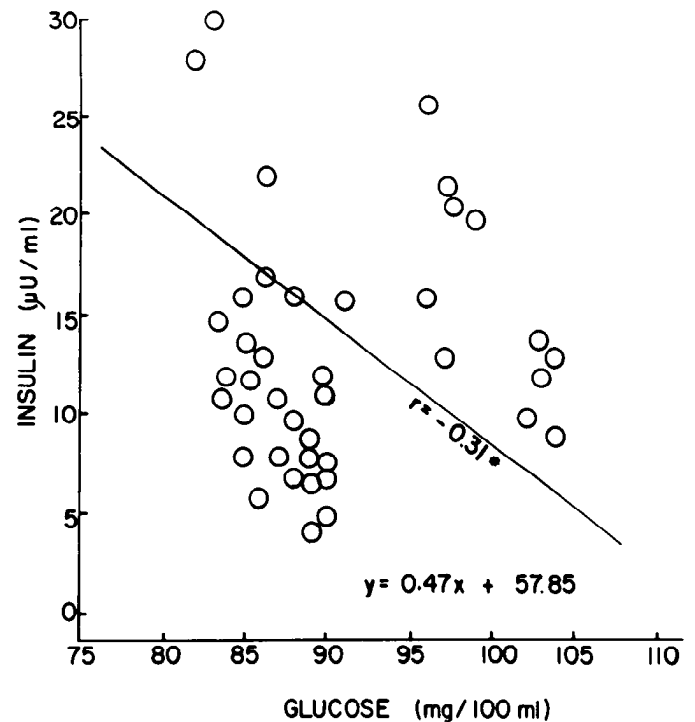

FIG. 8. Inverse relationship between variations of plasma glucose and plasma insulin during 8 min after ingestion of 755 kcal fed in form of a meal. * $P < 0.05$.
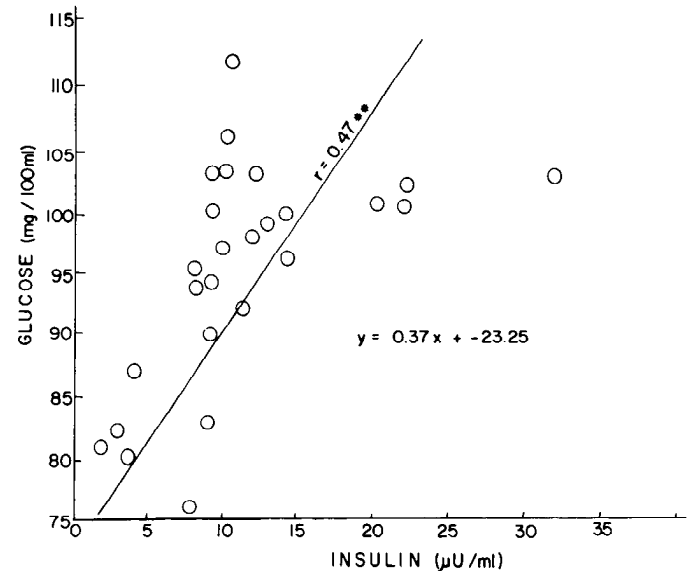

FIG. 9. Relationship between variations of plasma glucose and plasma insulin during 8 min after ingestion of 755 kcal fed by stomach tube. ** $P < 0.01$.

heat increment of feeding (HIF) for our study in which measurements were only made for 90 min. Just the same it would seem possible that the postprandial increase in energy utilization would result primarily from storage of nutrients in the case of tube feeding, whereas, with meal feeding, both storing and oxidation, a more costly process (5), would participate in this action. Our experiment cannot answer whether the substrate for oxidation comes from the body reserves or from the ingested nutrients. However, the fact that the changes in insulin and NE secretion took place very rapidly, even before the in-

gested food could become available in the circulation, could indicate that body reserves were being mobilized and oxidized in the postprandial period. The absence of early insulin and NE response and the smaller effect on oxygen consumption and on RQ observed with tube feeding could indicate that oxidative metabolism was small compared with that of meal feeding.

The present study is limited to the postprandial period extending to 90 min, which is the time when maximum heat production is observed (23) and the time also when glycemia levels have returned to premeal values. However this increased oxygen consumption has been shown to last up to 4 h (23). It is conceivable that, between 90 and 240 min after the meal, factors related to tube feeding, such as gastric emptying time or absorption and assimilation of nutrients, might have been delayed. For these reasons an evaluation of postprandial events for a period longer than 90 min in both tube- and meal-fed subjects would seem to justify further investigation.

For the same reasons, for the 90-min period that follows the meal, the present results suggest that sensory inputs initiated by the ingestion of food activate oxidative processes that would be under endocrine control. If the influence of food palatability is abolished by tube-feeding, the postprandial thermic effect is almost suppressed. It should be interesting to verify whether feeding human subjects highly palatable food at constant energy intake would reduce the efficiency of food and favor an energy imbalance leading to body weight reduction. It has been shown that feeding palatable food to rats in amounts comparable with those consumed by rats on standard laboratory chow gradually produces an enhanced thermogenesis and reduced efficiency of food and body weight gain (11). From the above data we would expect to find an identical response in humans.

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