Cephalic phase responses to sweet taste 1–3

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ABSTRACT  The sweet taste of nonnutritive sweeteners has been reported to increase hunger and food intake through the mechanism of cephalic-phase insulin release (CPIR). We investigated the effect of oral sensation of sweetness on CPIR and other indexes associated with glucose metabolism using nutritive and nonnutritive sweetened tablets as stimuli. At lunchtime, 12 normal-weight men sucked for 5 min a sucrose, an aspartame-polydextrose, or an unsweetened polydextrose tablet (3 g) with no added flavor. The three stimuli were administered in a counterbalanced order, each on a separate day at 1-wk intervals. Blood was drawn continuously for 45 min before and 25 min after the beginning of sucking and samples were collected at 1-min intervals. Spontaneous oscillations in glucose, insulin, and glucagon concentrations were assessed as were increments (slopes) of fatty acid concentrations during the baseline period. The nature of the baseline (oscillations: glucose, insulin, and glucagon; and slopes: fatty acids) was taken into account in the analyses of postexposure events. No CPIR and no significant effect on plasma glucagon or fatty acid concentrations were observed after the three stimuli. However, there was a significant decrease in plasma glucose and insulin after all three stimuli. Only the consumption of the sucrose tablet was followed by a postabsorptive increase in plasma glucose and insulin concentrations starting 17 and 19 min, respectively, after the beginning of sucking. In conclusion, this study suggested that oral stimulation provided by sweet nonflavored tablets is not sufficient for inducing CPIR. Am J Clin Nutr 1997;65:737–43.

KEY WORDS  Cephalic-phase insulin response, spontaneous oscillations, plasma glucose, insulin, glucagon, fatty acids, sucrose, aspartame

INTRODUCTION

Preingestive cephalic-phase secretions, triggered by sensory properties of food, shape the organism’s response to the incoming energy load and may enhance nutrient digestion, absorption, and utilization (1). Particular attention has focused on the cephalic-phase insulin release (CPIR) because of its crucial role in limiting postabsorptive hyperglycemia (2, 3). CPIR after exposure to food has been shown in many animal species (2, 4–6) as well as in human subjects (7–9). It is regarded as a conditioned response (10) whose magnitude is determined in part by sensory properties of food (11, 12). Generally, oral exposure to palatable foods, notably sweet ones, is followed by CPIR and by a corresponding drop in plasma glucose concentration (2, 5, 13, 14). A question arises concerning the effect on CPIR after consumption of a sweet but nonenergetic product such as aspartame.

In some studies, the drop in plasma glucose that follows CPIR has been associated with greater hunger sensations and increased short-term food intake (15–20). Some authors have reported higher hunger ratings after subjects consumed aspartame-sweetened solutions than after plain water (18, 19). In another experiment, they reported that the consumption of saccharin-sweetened yogurt, as opposed to plain yogurt, increased food consumption at lunchtime and stimulated further hunger intake after the lunch (20). It has been argued that this appetite enhancement may be the consequence of CPIR occurring after sweet oral stimulation. According to this theory, uncoupling sweet taste and energy would increase hunger sensations and food intake, and may thereby favor obesity. Subsequent research has generally failed to support these findings and the issue remains highly controversial (see review in 21).

Therefore, conclusions about the role of intense sweeteners in the control of energy intake and body weight regulation should consider the effect of sweetness per se on CPIR. Nutritive (2, 22) as well as nonnutritive (23, 24) sweeteners have been shown to elicit the CPIR in rats. However, studies in humans are few because of methodologic difficulties in assessing CPIR. Indeed, the phenomenon is brief, is of small magnitude (14–93 pmol/L), and can occur as early as 2 min after food ingestion. Moreover, the preprandial plasma insulin concentration in humans is ≈50 pmol/L and has been shown to oscillate spontaneously with an amplitude of 7–16 pmol/L and a period of ≈12 min (25, 26). Studies on the effects of food stimuli on plasma insulin must consider these basal oscillations, given that their magnitude can be as large as that reported for CPIR. To separate the CPIR from basal fluctuations, plasma insulin should be assayed over a sufficiently long time before ingestion (≥45 min) and within small intervals of ≤2 min (8). Most studies of CPIR in humans have been conducted with few assays of insulin at arbitrary and variable intervals before, during, and after stimulation. The existence of basal fluctuations suggests that this method might have been a source of artifacts.

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The effect of sweet taste on CPIR in humans has been investigated in several studies. Some authors failed to show insulin secretion after ingestion of nonnutritive sweetened solutions (3, 27). Others found no significant effect on plasma insulin after nutritive and nonnutritive sweetened solutions were tasted and expectedorated (28). One study reported an increase in insulin release after a 3-min lingual application of a glucose solution (29). It is therefore difficult to make a conclusion about the effect of sweet taste on CPIR in humans because the discrepancies in results may be due to different experimental procedures.

Other cephalic responses have been reported in animals and humans. Cephalic-phase glucagon release (CPGR) has been shown in dogs (30) and in rats (13). However, in humans, several authors failed to detect a rise in glucagon during the cephalic-phase period (9, 31, 32), except in one study in which CPGR occurred in five healthy men who consumed a pizza meal (33). Additionally, two early studies in humans (7, 34) suggested anticipatory drops in plasma concentrations of fatty acids just before a meal.

In the present study we tested the hypothesis that sweet taste per se would elicit CPIR in normal-weight men. We also monitored changes in plasma glucose, glucagon, and fatty acids. Because basal insulin, glucagon, and glucose concentrations showed spontaneous ultradian fluctuations (23, 26, 35–37), blood was collected over a prolonged baseline period (45 min) and at 1-min intervals to allow for accurate evaluation of cephalic responses. The stimuli conditions in this study consisted of tablets with no added flavor: a nutritive (sucrose) sweetened tablet, a nonnutritive (aspartame-polydextrose) sweetened tablet, and a nonsweetened (polydextrose) tablet.

SUBJECTS AND METHODS

Subjects

The subjects were 12 normal-weight healthy young men aged 18–27 y. Their body mass indexes (BMIs; in kg/m²) were between 20 and 24. They were nonsmokers, normotensive, and free of metabolic disease. They had stable body weights, were nonrestrained eaters (38), had never followed an energy-restricted diet, and did not habitually use intense sweeteners. The study protocol had been approved by the institutional review board of the University of Paris–Nord. The subjects gave written informed consent before the experiment and were compensated for completing the study protocols.

Sweet stimuli

The stimuli consisted of three carbohydrate-based tablets with no added flavor: the sucrose tablet contained 3 g pure sucrose, the aspartame tablet contained 3 g polydextrose and 18 mg aspartame and had been matched for sweetness with the sucrose tablet in pilot tests, and the placebo tablet contained 3 g polydextrose. Polydextrose is a bland-tasting carbohydrate not absorbed by the small intestine. Perceived sweetness of the stimuli was assessed by using visual analog scales (VASs). After sucking the tablet, subjects were asked to rate perceived sweetness intensity along a 100-mm horizontal line, anchored with the French translation of “not sweet” at the left end and “very sweet” at the right end.

Blood collection

A specially designed double-lumen catheter (Miles 4901; Bayer Diagnostics, Puteaux, France) allowed us to withdraw blood continuously at a constant flow of 2 mL/min and without administration of an anticoagulant (39). Blood was drawn from 45 min before to 25 min after the oral presentation of the stimuli. Seventy blood samples collected at 1-min intervals were obtained with a fraction collector. The blood samples were immediately centrifuged at 1200 × g for 10 min at 5 ± 1 °C and the plasma from each sample was separated into four aliquots and stored at −26 °C for later determination of the various indexes.

Plasma measurements

Plasma assays for glucose, insulin, glucagon, and fatty acids followed standard procedures. Glucose concentrations were measured by an enzymatic assay with a glucose-oxidase technique (glucose analyzer 23A, intraassay CV of 1%; Bioblock, Strasbourg, France), insulin concentrations by a radioimmunoologic method (SB-INSI-5 kit, intraassay CV of 7%; CEA, Git-sur-Yvette, France), glucagon concentrations by a radioimmunologic method (intraassay CV of 9%; Pharmacia-Serono kit; Pharmacia, Saint Quentin en Yvelines, France), and fatty acid concentrations by a colorimetric enzymatic method (intraassay CV of 5%; Nefa C Wako kit; Unipath, Dardilly, France). Analyses of plasma glucose data were based on eight subjects per condition. Analyses of fatty acid concentrations were based on 7 subjects for the placebo, 7 for the aspartame, and 10 for the sucrose condition.

Design and procedures

Subjects arrived at the laboratory on the evening before the test day. They consumed a standard dinner between 2000 and 2030. Dinner was always composed of ham, green beans, hard cheese, stewed apples, and bread. Water was provided ad libitum. On the morning of the test day at 0800, subjects consumed a standard carbohydrate-free breakfast composed of ham, eggs, and coffee. Breakfast composition was intended to stabilize basal concentrations of hormones and metabolites throughout the experimental period. The amount of food provided for dinner and breakfast during the three sessions was determined by the amount consumed on the first occasion.

A catheter was inserted into an antecubital vein of each subject at 1030. Subjects were infused with physiologic saline over 30 min before the placement of the double catheter. Continuous blood drawing began 50 min before the oral presentation of the stimulus. Blood collected during the first 5 min was discarded as waste. Tablets were administered at 1200. Subjects neither saw nor touched the tablets, which were placed in their mouths by the investigator. Subjects were instructed to suck the tablets for exactly 5 min. The investigator verified the compliance of each subject. The order of presentation of the three tablets was counterbalanced across subjects. The study followed a within-subject, double-blind, placebo-controlled design. All subjects participated in three testing sessions, scheduled at exactly 1-wk intervals.

Statistical analyses

The procedure used to separate cephalic-phase secretions from basal fluctuations in plasma glucose, insulin, and gluca-
gon concentrations was based on adjusted plots of sinusoid function (EASYPLOT; Spiral Software, Brookline, MA). Best-fitting sinusoid plots for each individual curve were adjusted on the 45 baseline values obtained before the presentation of the stimulus. Goodness of fit was tested by using correlation coefficients (r) (Figure 1). For cases in which the sinusoid function provided a good fit (P < 0.05), values of the adjusted sinusoid equation were subtracted from observed glucose, insulin, and glucagon values. For cases in which the fit was weak (NS), the arithmetic mean of the 45 baseline data points was subtracted from the observed value. Values obtained from the difference between observed and calculated values are called residual values.

Analyses of plasma glucose, insulin, and glucagon concentrations were based on residual values. Repeated-measures analysis of variance (ANOVA) was used with stimulus type (sucrose, aspartame, or placebo) and time (70 measurements) as the within-subject factors.

To determine within-condition effects (ie, whether significant changes in plasma glucose, insulin, or glucagon occurred compared with baseline), every residual time point in the postexposure period was compared with the baseline mean by using repeated-measures ANOVA. The postexposure period is defined as the time interval between the beginning of sucking the tablet and the end of the blood sampling (45–70 min).

Comparisons of between-condition effects were based on the calculation of areas under the curve (AUCs) of residual values for the 25-min postexposure period. Pilot data from this laboratory as well as examination of individual responses in the sucrose condition suggest that two postexposure phases (pre- and postabsorptive) should be considered. The postexposure preabsorptive phase was defined as the time interval between the beginning of sucking the tablet and the first minutes when rises in plasma glucose and insulin were observed. Therefore, the duration of this phase was set at 0–17 min for glucose and 0–19 min for insulin and glucagon. Consequently, the postabsorptive phase was set at 17–25 min for glucose and 19–25 min for insulin and glucagon. AUCs were then calculated over these two phases and analyzed by using repeated-measures ANOVA with stimulus type and phase (pre- and postabsorptive) as within-subjects factors. When a significant effect of the stimulus type was observed, subsequent comparisons between conditions were tested.

Because plasma concentrations of fatty acids generally showed a steady increase or a steady decline during the pre- and postexposure periods, the dependent measure selected for the analysis was the calculated slope of fatty acid values during the 45-min preexposure and the 25-min postexposure periods. Calculated slopes were then compared by using repeated-mea-

![FIGURE 1](https://example.com/figure1.png)

**FIGURE 1.** Example of a best-fitting sinusoid plot adjusted on the 45 baseline plasma insulin values of an individual curve. Goodness of fit was tested by calculating a correlation coefficient (r). When r is significant (P < 0.05), the calculated equation is prolonged over the 25 min after the beginning of the stimulus. Adjusted equation is \( y = a \times \sin((2\pi/\omega) \times x + \phi) + b \), where \( a \) is amplitude, \( \omega \) is period, \( \phi \) is phase at the origin, and \( b \) is the mean concentration. In this example: \( a = 10.1 \) pmol/L, \( \omega = 12.4 \) min, \( \phi = 0.53 \), \( b = 47.7 \) pmol/L, and \( r = 0.87 \).

![FIGURE 2](https://example.com/figure2.png)

**FIGURE 2.** Mean (± SEM) residual plasma insulin concentrations of 12 normal-weight male subjects before and after 5 min of sucking a nonflavored tablet. Black bars indicate the time of tablet sucking. Significantly different from baseline: *P < 0.05, *P < 0.01.
ures ANOVA with stimulus type and period (pre-compared with postexposure) as the within-subject factors. The critical value for significance was $P < 0.05$. Values are reported as means ± SEMs.

**RESULTS**

**Sweetness ratings**

Sweetness ratings were 74 ± 6.2 mm for sucrose, 57 ± 4.5 mm for aspartame, and 13 ± 3.4 mm for the polydextrose placebo tablet. Repeated-measures ANOVA showed that both sucrose and aspartame tablets were rated as sweeter than the placebo tablet ($P < 0.001$). Moreover, the sucrose tablet was rated as sweeter than the aspartame tablet ($P < 0.05$).

**Plasma insulin**

Individual fluctuations in plasma insulin concentrations were successfully modeled by using a sinusoid function in 34 of 36 cases. (Note that 1 case = 1 subject and 1 stimulus condition, thus, there was a total of 36 cases: 12 subjects × 3 conditions.) The mean amplitude of the fitted oscillation was 5.9 ± 0.5 pmol/L (range: 1.4–14.0 pmol/L) and the mean period was 11.7 ± 0.32 min (range: 7.0–15.8 min).

Mean residual insulin curves in the three experimental conditions are shown in Figure 2. The ANOVA of residual values over the entire 70-min period showed no effect of stimulus type but a significant main effect of time ($P < 0.01$) and a stimulus type–by-time interaction ($P < 0.01$). A separate analysis of the baseline period showed no significant effect of time ($P < 0.05$). In contrast, analysis of the postexposure period showed an effect of time ($P < 0.01$) and a stimulus type–by-time interaction ($P < 0.01$).

Comparisons of single, residual postexposure time points with the baseline mean in each condition are shown in Figure 2. In the sucrose condition, the residual insulin concentration decreased significantly 8–10 min postexposure ($P < 0.01$). Residual insulin increased after 19 min postexposure, though only the value at 22 min was significantly different from baseline ($P < 0.05$). A decrease in plasma insulin was also observed after exposure to the aspartame tablet. This was significant 11–13 min postexposure ($P < 0.01$). No subsequent rise in insulin concentration was observed. In the placebo condition, a significant decrease in insulin concentration was observed 9 and 12 min postexposure ($P < 0.05$) and 23–25 min postexposure ($P < 0.05$).

The ANOVA of insulin AUCs showed a main effect of phase ($P < 0.01$) and a stimulus type–by-phase interaction ($P < 0.05$). A separate analysis of AUCs in the preabsorptive phase showed no main effect of stimulus type. In contrast, analysis in the postabsorptive phase showed a significant main effect of stimulus type ($P < 0.01$). Subsequent comparisons between conditions in the postabsorptive phase showed that the AUC was significantly greater after the sucrose than after the placebo stimulus ($P < 0.01$; Table 1).

**Plasma glucagon**

Sinusoid functions were successfully fitted to the observed plasma glucagon fluctuations in 30 of the 36 cases. The mean amplitude of the oscillations was 19.1 ± 1.7 ng/L (range: 7.2–54.6 ng/L) and their mean period lasted 13.7 ± 0.7 min (range: 6.0–20.6 min).

The ANOVA of residual glucagon values over the entire 70 min did not show any significant effect of stimulus type, time, or stimulus type–by-time interaction. Comparisons of postexposure residual values to the baseline means revealed no significant differences in any of the three conditions (Figure 3). Similarly, no significant effect of stimulus type, phase, or stimulus type–by-phase interaction was observed when AUCs were analyzed (Table 1).

**Plasma glucose**

We failed to detect any cyclic pattern in basal plasma glucose. Therefore, residual values were equal to the difference between the observed values and the arithmetic mean of the 45 basal data points. The ANOVA of residual values over the entire 70 min showed that only time had a significant effect ($P < 0.05$).

Comparisons within each condition showed significant differences of some residual postexposure values and the baseline data (Figure 4). In the sucrose condition, plasma glucose values decreased relative to baseline and 7, 13, and 17 min postexposure ($P < 0.05$). The glucose concentration then rose, reaching a peak at 25 min postexposure but only the value at 23 min was significantly different from baseline ($P < 0.05$). A comparable fall in plasma glucose concentrations was observed in the aspartame condition. However, only values at 13 and 24 min postexposure were significantly different from baseline ($P < 0.05$). No subsequent rise in glucose concentrations was observed in this condition. In the placebo condition, the drop in plasma glucose was significant 1 min ($P < 0.01$) and 2, 8, 20, and 22 min ($P < 0.05$) postexposure. As in the aspartame condition, no subsequent rise in plasma glucose was observed.

The ANOVA of glucose AUCs during the postexposure phase showed a main effect of phase ($P < 0.01$) and a stimulus type–by-time interaction ($P < 0.05$). Separate analyses of postexposure preabsorptive and postabsorptive phases confirmed that there was a significant effect of stimulus type ($P < 0.05$) during the postabsorptive interval only. Comparisons

<p>| TABLE 1 |</p>
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<thead>
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<th>Calculated areas under the curve (AUCs) for plasma glucose, insulin, and glucagon concentrations during pre- and postabsorptive periods (^1)</th>
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<td><strong>Glucose</strong></td>
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<td><strong>Preabsorptive</strong></td>
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<td>Glucose AUC (n = 8) (mmol · min/L)</td>
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<td>Insulin AUC (n = 12) (pmol · min/L)</td>
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<td>Glucagon AUC (n = 12) (ng · min/L)</td>
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\(^1\) $\bar{x} ±$ SEM. Means with different superscript letters are significantly different, $P < 0.05$. 

[Image and Figure references are omitted for brevity, but are assumed to be referenced in the original document.]
between conditions showed that postabsorptive AUCs of plasma glucose were significantly greater after sucrose than after aspartame \((P < 0.01)\) or placebo tablets \((P < 0.05)\) (Table 1).

**Fatty acids**

Plasma fatty acid concentrations rose steadily with time throughout the testing session. As a result, the potential effects of stimulus type on plasma fatty acid were examined by using the slope of the curve as the principal dependent variable. Mean slopes during the baseline and the postexposure periods are shown in Table 2. Repeated-measures ANOVA showed no effect of stimulus type nor of period, and no stimulus type–by-period interaction.

**DISCUSSION**

In the present study, a sinusoid pattern of spontaneous oscillations in the baseline concentrations of plasma insulin and glucagon was observed. This result is consistent with previous reports (25, 26). Plasma glucagon concentrations showed

<table>
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<th>TABLE 2</th>
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<td><strong>Calculated slopes for plasma fatty acid concentrations during the pre- and postexposure periods</strong></td>
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<td>Slopes</td>
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<td>(\mu\text{mol} \cdot \text{L}^{-1} \cdot \text{min}^{-1})</td>
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<td>Preexposure</td>
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\(\bar{x} \pm \text{SEM}\). There were no significant differences between pre- and postexposure periods.
greater interindividual variability and were less amenable to curve fitting. Whereas a sinusoid pattern for insulin pulsatility was successfully established in 34 of 36 cases, only 30 of 36 cases showed a similar pattern with glucagon, and the goodness of fit was less potent. No spontaneous oscillations of plasma glucose were observed. Fluctuations of plasma glucose are known to have low relative amplitude (< 2% of the mean concentration) (26). This makes any oscillatory pattern difficult to discern unless blood glucose concentrations are continuously measured.

The existence of spontaneous baseline oscillations in plasma insulin and glucagon has important consequences for the interpretation of our data. In the present study, analytical procedures were designed to distinguish between potential effects of the experimental conditions and baseline fluctuations in plasma indexes. We believe that this is a state-of-the-art procedures for the study of the cephalic-phase response to foods.

In our experimental conditions, sucrose and aspartame tablets were ineffective in eliciting CPIR. Hitherto, human studies failed to show the occurrence of CPIR in response to simple sweet-tasting stimuli. However, an increase in plasma insulin has been reported after a 3-min lingual application of a glucose solution (29). However, in this study, baseline insulin was measured only once, 1 min before the stimulus.

A review of the published data shows that CPIR has been reported after ingestion of nutritive and nonnutritive sweeteners if they were incorporated into real food or administered with other food related sensory stimuli. Some investigators observed CPIR after the consumption of an aspartame-sweetened strawberry-flavored mousse (9). Others found that neither the simple ingestion of aspartame-sweetened water nor the visual presentation of an appetizing meal was effective in eliciting CPIR (3). However, the combination of these two stimuli associated with the chewing of aspartame-sweetened gum elicited CPIR. In a recent work, tasting and expectorating nutritive and nonnutritive sweetened solutions was not followed by insulin release whereas sham feeding of an apple pie was effective in eliciting CPIR (28). Taken together, these results suggest that CPIR is not triggered by an isolated oral sensation such as sweetness but may need a combination of oral cues associated more closely with an eating context.

No anticipatory drop in plasma fatty acid was found. The increase in fatty acid concentrations throughout the experiment reflects the metabolic state of energy depletion of the subjects. In rats, previous reports showed a steady rise in fatty acid concentrations in the preprandial period (6) and during short phases of fasting (1–6 h) (40). In this study, no effect on the rising slopes of plasma fatty acids was observed. Similarly, no significant change in plasma glucagon was found after ingestion of any of the three tablets.

Although no CPIR was observed after any of the three stimuli, decreases in plasma glucose and insulin concentrations were observed in the postexposure preabsorptive phase after the oral administration of the three tablets. After the sucrose stimuli, these decreases were followed by a mild increase in plasma glucose beginning 17 min postexposure, followed 2 min later by an increase in plasma insulin. This delayed rise in glucose and insulin concentrations reflected postabsorptive modifications after 3 g (50 kJ) sucrose was consumed. Yet, note that the amount of sucrose ingested was very small (3 g) and that the tablet was consumed within the space of 5 min. It is, therefore, probable that glucose entered the hepatic portal flow at a low rate and then rapidly disappeared from venous circulation. At the time of the study (1200), the subjects were in a state of energy depletion, so that circulating glucose was rapidly utilized. Even so, the postabsorptive rise in glucose and insulin concentrations attests to the accuracy and sensitivity of the experimental method.

The examination of individual insulin curves showed that the mere act of placing the tablet in the subject’s mouth had an immediate effect on the regularity of plasma insulin oscillations. In fact, insulin oscillatory cycles tended to flatten out during the 25-min postexposure period regardless of the sweetness or energy content of the tablet. One could argue that the way in which the nonflavored tablets was provided (ie, without being seen or touched) induced stress and led to inhibition of insulin pulsatility as well as CPIR. Indeed, in rats, a nonceptive acute stress applied at the time of ingestion induced an intolerance to glucose, resulting in a marked postabsorptive hyperglycemia. This stress also suppressed the hypoglycemia usually observed at the time of CPIR (41). Moreover, catecholamines have been shown to inhibit the release of insulin in humans (42). Nevertheless, in this study, suppression of insulin pulsatility was not accompanied by concomitant increases in plasma glucose or fatty acid concentrations. This does not support the hypothesis of an eventual inhibition of insulin secretion by an increase in catecholamine concentrations as a result of stress.

In conclusion, the results of this experiment showed that sweet taste provided in the form of sweet tablets did not elicit CPIR. Plasma glucose, insulin, glucagon, and fatty acids were not modified after aspartame or sucrose tablets compared with the nonsweet polydextrose tablet. The present study suggests that sweet taste per se is not a sufficient stimulus for eliciting CPIR. As noted above, such a response may require oral stimulation in the context of normal food consumption.

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REFERENCES

CEPHALIC-PHASE RESPONSES TO SWEET TASTE


