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Acute effects of nicotine on hunger and caloric intake in smokers and nonsmokers

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Abstract. The inverse relationship between smoking and body weight may be due in part to nicotine's effects on reducing hunger and eating. Male smokers and nonsmokers ($n = 10$ each), abstinent overnight from smoking and food, participated in four sessions, involving consumption of a liquid caloric load or water followed by nicotine (15 μg/kg) or placebo via nasal spray every 20 min for 2 h. Hunger and satiety ("fullness") ratings were obtained prior to each dose presentation. At the end of the two sessions involving the caloric load (simulating breakfast), subjects were also presented with typical lunch/snack food items varying in sweet taste and fat content for ad lib consumption. Results indicated that, for both smokers and nonsmokers, the hunger-reducing effects of nicotine occurred only following caloric load consumption, and there was no effect of nicotine on hunger after water consumption. Smokers unexpectedly reported greater satiation than nonsmokers following the caloric load regardless of nicotine or placebo condition. Nicotine also resulted in less caloric intake during the meal, and the decrease was not specific to consumption of sweet, high-fat foods. These results indicate that nicotine reduces appetite, possibly helping to explain the influence of smoking on body weight.

Key words: Nicotine – Hunger – Caloric intake – Smokers – Nonsmokers

Body weight is inversely associated with cigarette smoking, as smokers weigh less than nonsmokers (USDHHS 1988) and gain weight after stopping smoking (Perkins et al. 1987; Hall et al. 1989). Weight gain after cessation may be related to smoking relapse (Klesges and Klesges 1988), although the evidence for this is not entirely consistent (e.g. Hall et al. 1986). Cross-sectional surveys generally do not support the notion that smokers consume fewer calories (Albanes et al. 1987) or are more physically active (Blair et al. 1985; Marks et al. 1990) than nonsmokers. However, prospective research suggests that smoking may acutely decrease caloric intake, compared with periods of smoking cessation (Gilbert and Pope 1982; Hatsukami et al. 1984; Perkins et al. 1990a). In particular, smoking may be related to less preference for sweet taste (Rodin 1987) and less consumption of sweet foods (Grunberg 1982), although these results have not always been found (Bennett et al. 1970; Redington 1984). Animal research indicates that nicotine may be the ingredient of tobacco smoke primarily responsible for the effects of smoking on body weight (Schector and Cook 1976), and results from animal studies of nicotine tend to support findings from human studies of smoking, as nicotine administration reduces body weight, caloric intake, and sweet food consumption in rats (Grunberg 1982; Grunberg et al. 1988b).

Nevertheless, there are many gaps in our knowledge of the effects of smoking on caloric intake. First, there has been almost no research on possible mechanisms to explain reduced caloric intake due to smoking. Smoking may decrease feelings of hunger during fasting, allowing smokers to continue to abstain from eating. On the other hand, given the usual temporal pattern of smoking just *after* a meal (Gilbert and Pope 1982; Hasenfratz et al. 1989; Jarvik et al. 1989), smoking may prolong satiety following a meal and thus inhibit or delay subsequent caloric intake. Results from Gilbert and Pope (1982) suggest the latter may be more likely. In their study, smokers consumed fewer calories during between-meal snacks over a 24-h period of ad lib smoking compared with a 24-h period of abstinence despite consuming as many or more calories during meals.

Second, despite clear evidence of nicotine's effects on caloric intake in animals, little human research has examined effects of nicotine alone, isolated from tobacco smoke, on hunger and caloric intake. A recent study by Lee et al. (1989) suggests that IV nicotine decreases hunger in smokers before a meal but has no effect after a

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meal. Further specification of these nicotine effects in humans may provide directions for research on mechanisms and would help explain how nicotine replacement, such as via nicotine polacrilex, prevents or delays weight gain after smoking cessation (Gross et al. 1989). Alternatively, little or no effect of nicotine alone on hunger or caloric intake would indicate that components of tobacco smoke other than nicotine are responsible for any relationship between smoking and eating.

Third, there has been no research on the anorexic effects of nicotine (or smoking) in nonsmokers. Smaller nicotine effects in smokers versus nonsmokers would indicate chronic tolerance, which has been found with other responses to nicotine in humans (Perkins et al. 1989b). Chronic tolerance would suggest that nicotine exerts greater influence in suppressing hunger and caloric intake when smokers first initiate smoking, perhaps explaining why teenagers who smoke are far more likely than nonsmoking teenagers to believe that smoking helps control weight (Charlton 1984). Lack of research in this area is most likely due to methodological problems in standardizing nicotine dosing between nonsmokers and smokers (Herning et al. 1983).

Finally, none of the human or animal research on the effects of smoking or nicotine on preference for sweet taste has specifically taken into account the mediating effects of fat content of food. Recent research has indicated that, holding sucrose concentration constant, preference for sweet foods increases with increasing levels of fat content (Drewnowski and Greenwood 1983; Perkins et al. 1990b). Thus, the interaction of sucrose and fat content helps determine food preference. Although some animal results have shown that intake of sweet, high-fat foods is suppressed by nicotine (Grunberg et al. 1988b), the specific role of fat content has been ignored, and the smoking/nicotine research has examined food preference as a function of sucrose concentration only. This distinction is important in the present context for several reasons. First, it may help explain the variable results of research examining smoking and nicotine effects on sweet taste preference. In addition, since sweet, high-fat foods are denser in calories than sweet, low-fat foods, reduced preference specifically for sweet, fat taste due to smoking or nicotine may provide important information in understanding smoking's effect on caloric intake and body weight.

The purposes of the present study were 3-fold: 1) to determine the effects of nicotine on hunger and satiety following consumption of a standard caloric load compared with no load (water); 2) examine nicotine's influence on ad lib caloric consumption during a meal (particularly sweet, high-fat foods), and 3) determine possible differences in these nicotine effects between smokers and nonsmokers. In order to isolate effects of nicotine and standardize dosing across smokers and nonsmokers, we employed a nasal spray method of nicotine presentation which has been demonstrated to produce rapid, dose-dependent increases in plasma nicotine similar in magnitude and pattern to, but more reliable than, those of smoking (Perkins et al. 1986, 1989b).

Materials and methods

Subjects. Subjects were ten male smokers and ten male nonsmokers matched on age (mean ± SE of 23.2 ± 1.2 years for smokers versus 21.7 ± 0.8 years for nonsmokers) and body weight (74.8 ± 3.6 kg versus 75.5 ± 2.1 kg, respectively). Smokers smoked a mean of 19.8 ± 0.8 cigarettes per day (range = 15–23) for 5.1 ± 1.1 years (range = 1–10 years), while nonsmokers denied any past regular use of tobacco. All subjects denied current use of other tobacco products, such as chewing tobacco or snuff. Subjects' informed consent was obtained after the nature and consequences of the study were explained.

Design. This study employed a within-subjects design to examine: 1) the effects of nicotine versus placebo on hunger/satiety following caloric versus non-caloric (water) consumption, and 2) the effects of nicotine versus placebo on ad lib caloric consumption. Comparisons between smokers and nonsmokers provided an examination of the effects of smoking status on each. Smokers and nonsmokers each participated in four sessions on four separate mornings, involving consumption of a caloric load (two sessions) or a water load (two sessions), followed by administration of nicotine or placebo (as described below). Ratings of hunger and satiety were obtained throughout each session. Thus, examination of hunger/satiety basically involved one between-subjects factor (smoking status) and two within-subjects factors (caloric/water load, nicotine/placebo). Ad lib caloric consumption occurred only following the two caloric load sessions and thus involved only one within-subjects factor (nicotine/placebo), in addition to the between-subjects factor of smoking status.

Nicotine placebo dosing. Nicotine and placebo were each presented via nasal spray pump. The nicotine dose (15 μg/kg body weight) was similar to the mean nicotine intake of most smokers from a single typical cigarette (Benowitz and Jacob 1983). Each dose presentation consisted of 1.14 ml 0.9% sodium chloride solution together with the designated amount of L-nicotine and peppermint flavoring oil (Lorann Oils, Lansing, MI), which was used to mask the taste and smell of nicotine. The placebo (0 mg) contained only the sodium chloride solution with flavoring oil. Other details of this method have been described previously (Perkins et al. 1986, 1989a, b).

Standardized caloric load. The caloric load consisted of sweetened condensed milk (Borden Inc., Columbus OH), water, and vanilla for flavoring, and contained 67% carbohydrates, 10% protein, and 23% fat. The load amounted to 4.77 kcal/kg body weight and provided 45 kcal per 28.35 g. The water load consisted of an equal volume of water. The caloric load averaged 359 kcal for the subjects in this study and was designed to simulate the amount of caloric consumption during a light breakfast. Subjects found the caloric load palatable, as mean preference for the load was 6.35 ± 0.40, compared with 5.20 ± 0.13 for the water load, on a 1 ("Extremely Dislike") to 9 ("Extremely Like") scale, with 5 as the mid-point ("Neither Like nor Dislike"). There were no differences between smokers and nonsmokers in palatability of either load. Each was served at 5° C and consumed within 2–3 min.

Measures of hunger and satiety. Hunger and satiety ("fullness") were assessed separately, as research indicates the two sensations may not be completely inversely related to each other (Monello and Mayer 1967). Hunger was assessed by having subjects draw a line intersecting a 10-cm visual analog scale (VAS) at the point reflecting how "hungry" they felt at that moment. The VAS was anchored by "Not at all hungry" (0) and "Extremely hungry" (10), with "Somewhat hungry" (5) as the mid-point. Satiety was assessed using a similar 10-cm scale. These scales were adapted from Wardle (1987).

Meal items. Food items presented in the meal were typical lunch or snack items. The 17 items were divided on an a priori basis into

Table 1. Portion sizes and caloric content of each food item presented during ad lib meal

Item	Portion	Macronutrient Composition (% of kcals)			
		kcal	Prot	Carb	Fat
Non-sweet					
Kraft American cheese	1 oz. slice	90	25	9	66
Nabisco Graham cracker, plain	2 (5″ × 2.5″)	110	8	72	20
Pepperidge Farm white bread	2 slices (1.6 oz)	130	11	70	19
Pepperidge Farm wheat bread	2 slices (1.6 oz)	130	14	67	19
Butterball turkey breast, lean	2 slices (2 oz)	70	67	6	27
Hormel ham, lean	3 slices (2.5 oz)	75	71	0	29
Premium saltine crackers	10 (2″ squares)	123	25	66	26
Subtotal		728			
Sweet only					
Food Club seedless raisins	1.5-oz box	94	3	97	0
Mott's sweetened applesauce	4 oz	105	0	100	0
Jello, cherry	1/2 cup	80	10	90	0
"Frozfruit" frozen fruit bar, strawberry	1 (4 oz)	70	6	94	0
Orange	1 (sliced)	64	5	91	4
Subtotal		413			
Sweet fat					
Swiss Miss butterscotch pudding	4.25 oz	160	5	60	35
Reese's peanut butter cup	1 (0.9 oz)	140	10	38	52
Hostess cup cake, cream-filled	1 (1.75 oz)	129	5	63	32
Oreo cookies	3 (1.25 oz)	140	3	58	39
Ice cream, vanilla/chocolate	3 oz	96	9	43	48
Subtotal		665			
Total		1806			

three categories according to taste and fat content: nonsweet, sweet only (nonfat), and sweet/fat (high fat content). The items and their caloric and macronutrient content (USDA 1975) are listed in Table 1. The three categories were employed to determine if nicotine's effects on intake may be specific to sweet, high-fat foods, which are most preferred (Drewnowski and Greenwood 1983). The sweet only foods were low in protein and contained a mean of 1% fat by calories (range = 0–4%), while the sweet/fat foods were also low in protein but contained a mean of 41% fat (range = 32–52%). The nonsweet foods were variable in macronutrient composition and selected based on their lack of sweet taste. The specific items within each category were chosen to provide diversity of selections, and portion sizes were chosen to present items roughly similar in caloric content. Preference for all items was moderately high, as mean ± SE initial preference ratings during subject screening (self-report, without tasting) for each item ranged from 5.65 ± 0.55 (butterscotch pudding) to 8.00 ± 0.25 (ice cream) on a 1 ("Extremely Dislike") to 9 ("Extremely Like") scale, with an overall mean for all 17 items of 6.95 ± 0.15. There were no significant differences between smokers and nonsmokers on these initial ratings for any of the items, indicating equal preference between groups.

Items were arranged on trays in random order. Subjects were allowed to drink water during the meal and were given 20 min to eat as much as desired. Subjects were informed they could ask for additional portions of any item. Using a Sunbeam Precision Electronic Digital Scale (Hanson Scale Co., Shubuta MS), each item was weighed to the nearest gram before and after the meal to determine calories of each consumed. Calorie subtotals for each category of taste (nonsweet, sweet only, sweet/fat) and macronutrient (carbohydrates, fats, and proteins) were also tabulated.

Procedure. Subjects abstained overnight from smoking and food consumption prior to each of the four morning sessions. Overnight smoking abstinence was confirmed by expired air carbon monoxide (CO) reading of ≤13 ppm (Benowitz 1983). Throughout each session, subjects sat in a comfortable armchair in a sound-attenuated experimental chamber. Subjects completed the measures of hunger and satiety every 20 min during a 40-min baseline period. Then, subjects consumed either the caloric or water load, followed 1–2 min later by presentation of either nicotine or placebo. Subjects then remained at rest for 20 min, at which time they completed the hunger and satiety measures just before receiving the second presentation of the same dose previously received. This procedure was designed to assess relatively sustained effects of nicotine on hunger and satiety rather than immediate, and possibly transient, effects. Dose presentation followed by 20 min rest and then completion of measures was repeated four additional times, for a total of six dose presentations over 2 h.

On days in which the water load was consumed, the session ended after completion of hunger and satiety measures at the end of the 20-min rest following the sixth dose presentation. On days in which the caloric load was consumed, subjects were subsequently presented with an additional, seventh dose followed by ad lib consumption of food items during the 20-min meal. The meal was presented on caloric load days to simulate lunch preceded approximately 2 or 2.5 h by a light breakfast (i.e. the caloric load), a more naturalistic pattern of caloric consumption compared with the prolonged fasting induced on water load days (i.e. overnight followed by morning-long fasting). Dose presentations, meal presentation, and calculation of calories consumed were performed by an experimenter blind to the subject's smoking status and to the dose employed in each session.

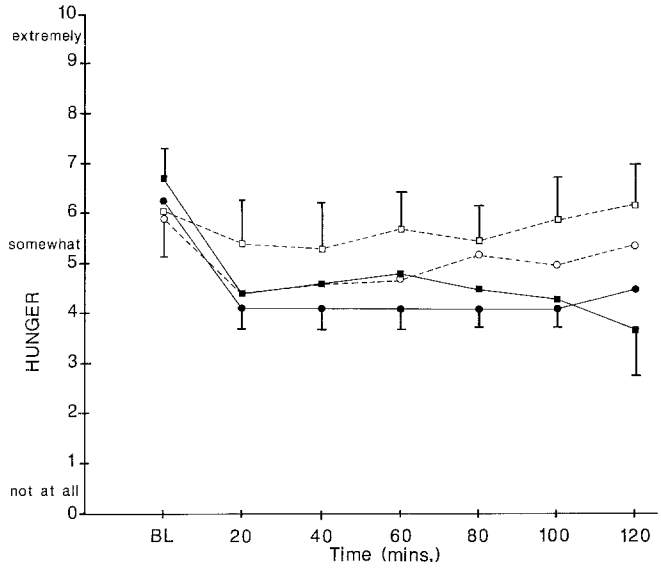
Data analyses. Hunger and satiety were each examined using a 3-factor mixed repeated measures analysis of covariance (ANCOVA), with smoking status as the between-subjects factor, and caloric/water load and nicotine/placebo as the within-subjects factors. The mean of the two baseline (pre-dose) values served as the covariate. Total caloric consumption during the meal was analyzed using a 2-factor analysis of variance (ANOVA), with status and nicotine/placebo as factors. A separate multivariate ANOVA (MANOVA) was performed for the three taste categories (nonsweet, sweet only, and sweet/fat), and for the three macronutrients (carbohydrate, protein, and fat consumption), with each of the MANOVAs employing smoking status and nicotine/placebo as factors, in addition to taste or nutrient category. Follow-up comparisons were performed via Fisher's Least Significant Difference (LSD) *t*-test procedure (Huitema 1981).

Results

Hunger and satiety

There were no significant effects of session or smoking status on baseline (pre-load) ratings of hunger or satiety. As expected, the caloric load significantly decreased hunger [$F(1,17) = 38.12$, $P < 0.001$], as shown in Fig. 1. The main effect of nicotine on reducing hunger was significant [$F(1,17) = 4.28$, $P = 0.05$], as was the interaction of nicotine × caloric load [$F(1,17) = 7.03$, $P < 0.05$]. This interaction was due to nicotine decreasing hunger following consumption of the caloric load [$t(18) = 3.66$, $P < 0.01$] but not following the water load [$t(18) < 1$], also shown in Fig. 1. Follow-up LSD t-tests indicated that the hunger-reducing effect of nicotine following caloric load was significant for smokers [$t(18) = 2.59$, $P < 0.02$] as well as nonsmokers [$t(18) = 4.72$, $P < 0.001$]. A further, exploratory comparison indicated that the magnitude of the effect was greater for nonsmokers compared with smokers [$t(18) = 2.15$, $P < 0.05$].

Caloric load consumption also increased satiety relative to the water load [$F(1,17) = 34.41$, $P < 0.001$], as shown in Fig. 2. In contrast with results for hunger, there was no main effect of nicotine on elevating fullness [$F(1,17) < 1$] and the interaction of nicotine × caloric load



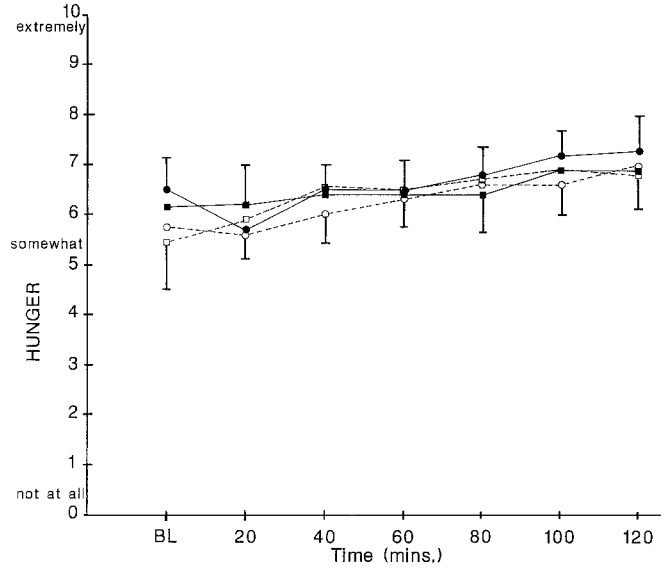
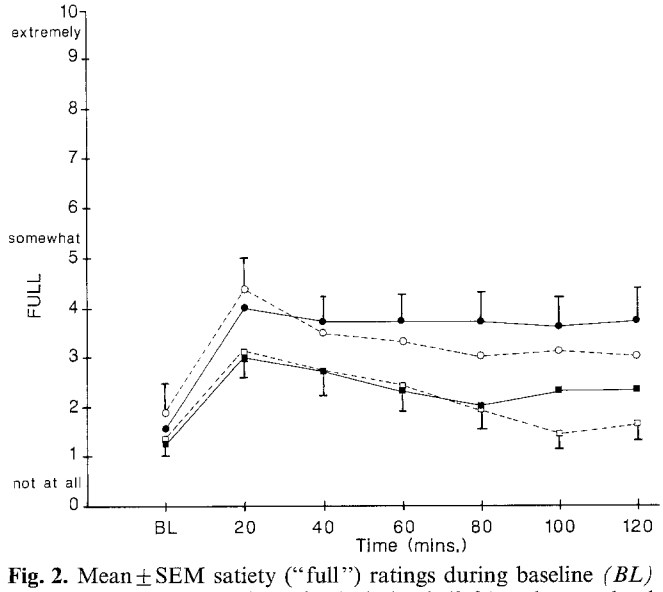


Fig. 1. Mean ± SEM hunger ratings during baseline *(BL)* and following consumption of caloric load *(left)* and water load *(right)* for smokers and nonsmokers after receiving placebo and nicotine every 20 min for 2 h. ○ Smokers/placebo; ● smokers/nicotine; □ nonsmokers/placebo; ■ nonsmokers/nicotine



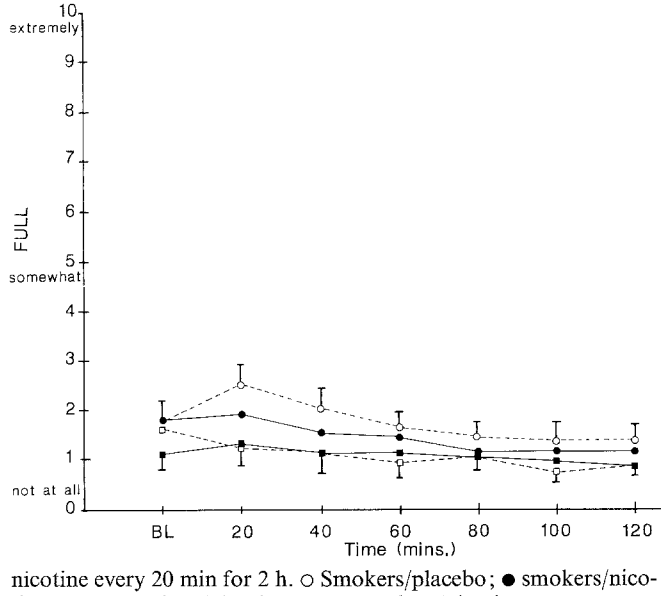


Fig. 2. Mean ± SEM satiety ("full") ratings during baseline *(BL)* and following consumption of caloric load *(left)* and water load *(right)* for smokers and nonsmokers after receiving placebo and nicotine every 20 min for 2 h. ○ Smokers/placebo; ● smokers/nicotine; □ nonsmokers/placebo; ■ nonsmokers/nicotine

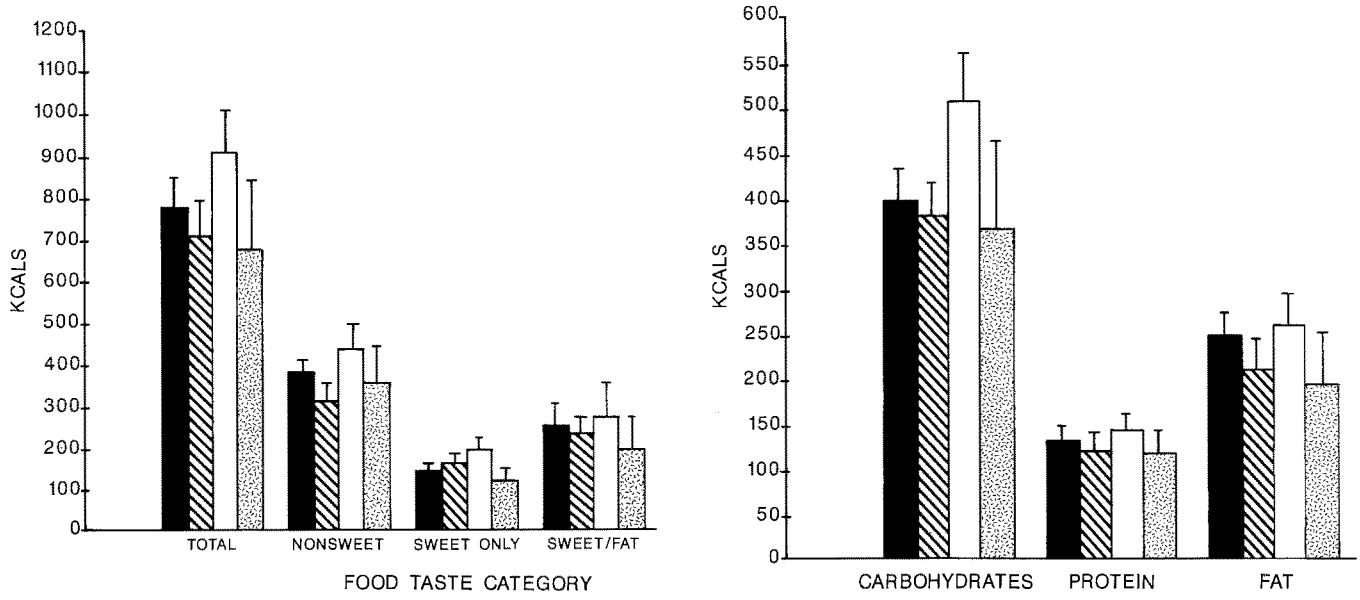


Fig. 3. Mean ± SEM total caloric intake and intake of nonsweet, sweet, and sweet/fat foods *(left)* and of carbohydrates, protein, and fat *(right)* during ad lib meal for smokers and nonsmokers after receiving placebo and nicotine. ■ Smokers/placebo; ▨ smokers/nicotine; □ nonsmokers/placebo; ▨ nonsmokers/nicotine

did not reach significance [$F(1,17) = 2.68$, $P > 0.10$]. However, there was a significant interaction of status × caloric load × time [$F(5,85) = 2.37$, $P < 0.05$]. Follow-up comparisons between groups indicated a more prolonged increase in satiety following the caloric load for smokers versus nonsmokers during *either* the nicotine or placebo condition [$t(18) = 3.10$, $P < 0.01$].

Ad lib caloric consumption

There was no overall difference between smokers and nonsmokers in mean caloric consumption [$F(1,18) < 1$]. However, consumption during the meal was significantly reduced following nicotine compared with placebo [$F(1,18) = 5.27$, $P < 0.05$]. Although the nicotine × status interaction was not significant [$F(1,18) = 1.59$], additional comparisons were performed to determine the consistency of this nicotine effect between smokers and nonsmokers, given that the possible mediating influence of smoking status was a primary focus of the study. These tests revealed that the effect of nicotine on reducing caloric intake was significant for nonsmokers [$t(18) = 2.52$, $P < 0.02$] but not for smokers [$t(18) < 1$], as shown in Fig. 3.

Nonsweet foods were consumed more than sweet only or sweet/fat foods [$F(2,36) = 21.78$, $P < 0.001$], presumably because there were more items from that taste category available to be eaten. More importantly, there was no significant interaction of nicotine × taste category [$F(2,36) < 1$], as nicotine did not differentially affect consumption of nonsweet versus sweet only versus sweet/fat foods, as shown in Fig. 3. There were no other significant effects involving taste category.

There was no overall difference between smokers and nonsmokers in consumption of carbohydrates, protein, and fat [$F(2,36) < 1$ for status × macronutrient interaction], also shown in Fig. 3. However, the interaction of nicotine × macronutrient was marginally significant [$F(2.36) = 3.05$, $P < 0.06$]. Exploratory analyses indicated that nicotine reduced consumption by all subjects of carbohydrates [$t(18) = 3.33$, $P < 0.01$] and fat [$t(18) = 2.12$, $P < 0.05$] but not protein ($t < 1$). The status × nicotine × macronutrient interaction was also nearly significant [$F(2,36) = 2.98$, $P = 0.06$], as the decrease in carbohydrate intake following nicotine was more pronounced in nonsmokers compared with smokers [$t(18) = 5.10$, $P < 0.001$].

Discussion

To the extent that nicotine is the ingredient of tobacco smoke responsible for smoking's effect on body weight, these findings may have implications for explaining this effect. Specifically, nicotine may decrease feelings of hunger in smokers, as well as nonsmokers, following consumption of a caloric load but not in the absence of such caloric consumption. Thus, nicotine may not uniformly suppress hunger under all conditions but may do so only after meal consumption. Nicotine intake via smoking, therefore, may be associated with decreased caloric intake due to nicotine's actions on hunger following a meal, thereby delaying subsequent eating. This notion is supported by the findings of Gilbert and Pope (1982), cited earlier, showing acute effects of smoking on between-meal snacking but not on intake during regular meals. These results contrast with Lee et al. (1989), who found that IV nicotine decreased hunger prior to meal consumption but did not differ from saline after a meal. An explanation for this difference is not immediately apparent, although Lee et al. provided a high-protein

solid meal almost twice the caloric content of ours and maintained steady-state plasma nicotine concentrations, compared with our intermittent presentations of bolus nicotine doses as in smoking (Russell and Feyerabend 1978).

On the other hand, the present study also offered a more direct test of nicotine's acute influence on caloric intake during a meal and revealed that intake was significantly reduced in nonsmokers but not in smokers. This differential effect must be interpreted cautiously, however, since the nicotine × status interaction was not significant. In any event, the possible difference between smokers and nonsmokers in the effects of nicotine on caloric intake is suggestive of chronic tolerance. It is possible, then, that the influence of nicotine on caloric intake (and body weight) may be greater during initial adoption of smoking or during relapse after some period of cessation, perhaps serving as a particularly strong reinforcing effect of smoking. Because plasma levels were not obtained in this study, it is not possible to determine if this tolerance may be dispositional or functional. In contrast, however, the unexpected greater satiety of smokers versus nonsmokers regardless of nicotine or placebo condition suggests that smoking may exert some sort of chronic effect on enhancing processes involved in satiation. Alternatively, unknown differences between groups in factors other than past history of smoking may be responsible for these observations.

There was no difference between smokers and nonsmokers in sweet food intake and no particular effect of nicotine on decreasing consumption of sweet foods, whether low or high in fat. This contrasts with some results from animal research (Grunberg et al. 1985, 1988a) but is consistent with human research on the acute and chronic effects of smoking and nicotine on sweet taste preference (Redington 1984; Perkins et al. 1990a, b). Possible explanations for this disparity include species differences in response to nicotine and the potential that nicotine's anorexic actions may be highly specific to certain sweet foods. Yet, the decline in consumption across taste categories in this study following nicotine would suggest a broader influence of nicotine on food intake, independent of sweet/fat taste.

There is currently little direct evidence of physiological processes by which nicotine acts on central or peripheral sites to decrease hunger and food intake. Nicotine may reduce insulin secretion (Grunberg et al. 1988a), which could directly affect hunger and appetite. Nicotine is also known to have powerful parasympathetic influences which could alter gastro-intestinal tract functioning, such as delaying gastric emptying (Nowak et al. 1987), although some evidence suggests nicotine may accelerate gastric emptying (Grimes and Goddard 1978).

Although the dose employed here was typical of the amount consumed by regular smokers from a single cigarette, these results need to be replicated using a range of doses to determine dose-response relationships between nicotine and hunger or caloric intake. Other doses may reveal greater or smaller differences in responses as a function of prior nicotine exposure (e.g., smokers versus nonsmokers), as has been found with other effects of nicotine in humans (Perkins et al. 1989b). Examination of nicotine's effects on caloric intake in more naturalistic situations may also be useful, since it is possible that the specific pattern of consumption during this ad lib meal setting was not representative of subjects' usual consumption patterns. However, other research supports the validity of such laboratory meal assessments (Obarzanek and Levitsky 1985).

That these effects occurred with presentation of nicotine alone, isolated from tobacco smoke, further supports nicotine as an important component of tobacco smoke which may be responsible for smoking's effect on energy balance and body weight, although other components of smoke may also be actively involved in this relationship. These findings suggest a means by which to explain how nicotine replacement may act to prevent much of the weight gain typically experienced after smoking cessation (Gross et al. 1989). However, given the substantial differences in route and pattern of nicotine administration between our nasal spray method and nicotine gum, further study is needed to specifically determine the effects of nicotine gum on hunger and caloric intake.

Finally, generalization of these results to women is needed, as animal data suggest that nicotine's influence in reducing food intake may be more pronounced in female rats (Grunberg et al. 1987). Women appear to experience greater differences in weight as a function of smoking status (Pagano et al. 1987), and there is emerging evidence that smoking is used as a weight control strategy in women (Klesges and Klesges 1988), perhaps by suppressing hunger or appetite. Since women may be more concerned than men about their weight in general (Feldman et al. 1985) and weight gain following smoking cessation in particular (Sorenson and Pechacek 1987), the effects of nicotine on suppressing hunger and caloric intake may be especially powerful reasons for maintenance of smoking among females.

In conclusion, the results of this study, combined with our previous findings that nicotine produces acute increases in metabolic rate (Perkins et al. 1989a, c), indicate that nicotine may affect both the caloric intake and energy expenditure sides of the energy balance equation and may thus substantially influence body weight of smokers. Better understanding of these effects of nicotine may lead to development of methods to combat increased caloric intake after smoking cessation and thus prevent smoking relapse (Wack and Rodin 1982).

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