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Perception and Hedonics of Sweet and Fat Taste in Smokers and Nonsmokers Following Nicotine Intake

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PERKINS, K. A., L. H. EPSTEIN, R. L. STILLER, M. H. FERNSTROM, J. E. SEXTON AND R. G. JACOB. *Perception and hedonics of sweet and fat taste in smokers and nonsmokers following nicotine intake.* PHARMACOL BIOCHEM BEHAV **35**(3) 671–676, 1990.—Nicotine's effects on reducing perception and/or hedonics of sweet and fat taste may lead to less intake of sweet tasting, high-fat foods by smokers, helping to explain their generally lower body weights. Smokers and nonsmokers (n = 10 males each) rated perception (intensity and sensitivity) and hedonics (liking) of sweet/fat taste in milk samples varying both in sucrose (0, 5, 10, 20% w/w) and fat (0.1, 3.5, 11.7, 37.6% w/w) concentration on two occasions, once following intermittent presentation of nicotine (15 μg/kg) via measured dose nasal spray and the other following placebo. Nicotine significantly reduced perceived intensity of fat but not sweet taste and had no effect on sensitivity to either taste. There was no effect of nicotine on hedonics of sweet/fat taste. On the other hand, although there were no differences between smokers and nonsmokers in perception of sweet or fat, hedonics of sweet/fat taste was reduced in smokers regardless of nicotine or placebo intake. Thus, nicotine may acutely decrease fat taste perception without influencing sweet/fat hedonics, while long-term exposure (i.e., being a smoker) may produce chronically decreased taste hedonics without altering perception.

Nicotine Sweet Fat Intensity Sensitivity Hedonics Smokers Nonsmokers

EPIDEMIOLOGIC research has consistently found that cigarette smoking is associated with lower body weight (1,36), and many studies have shown that smokers gain weight after stopping smoking [e.g., (17, 25, 34)]. Although increased metabolic rate due to nicotine intake may explain some of the difference in energy balance between smokers and nonsmokers (26, 29, 30), this effect does not appear to explain the full extent of the difference and reduced caloric intake by smokers may contribute to their generally lower body weights. Indeed, longitudinal research indicates that smoking may acutely decrease caloric intake, compared with periods of smoking abstinence (10, 18, 27). In particular, smoking may be associated with reduced consumption of sweet foods (12), and increased intake of sweet foods after smoking cessation may be particularly important in determining subsequent weight gain (17,34).

Mechanisms to explain smoking's influence on sweet food intake may involve decreased perception (i.e., intensity and sensitivity) and/or hedonics of sweet taste. With regard to sweet taste perception, findings from studies over the past 30 years have not been very supportive of an effect of smoking. Earlier studies found decreased intensity of bitter, but not sweet, taste in smokers compared with nonsmokers (20,31) and no acute effects of smoking on sweet taste sensitivity (20). Similarly, recent studies of sweet taste sensitivity found no acute effects of smoking and no differences between smokers and nonsmokers (32), as well as no effects of cessation (27). However, a study involving smokeless tobacco did suggest that regular users were less sensitive than nonusers to sweet as well as salty taste (22). Interestingly, in that study, acute exposure to smokeless tobacco had no effect on taste intensity in users but reduced taste intensity (i.e., raised sweet taste threshold) in nonusers, suggesting the possibility that tolerance occurs to this effect.

There is somewhat more support for the notion that smoking is inversely related to sweet taste hedonics, but this effect has not consistently been found. Rodin (34) recently showed that smoking cessation resulted in increased hedonics of sweet taste. Similarly, Grunberg (12) reported reduced intake of sweet foods by smokers compared with nonsmokers. However, other research has found no effects of smoking status (32) or of smoking cessation (27) on sweet taste hedonics. Indeed, some studies have shown greater sugar consumption in smokers compared with non- or ex-smokers (2,33), as well as greater hedonics of sweet taste in smokeless tobacco users relative to nonusers (22). Finally, other studies have found that the acute effects of smoking on sweet food consumption or hedonics are rather small (12) or apparent only following a glucose load (32).

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The results of this research leave many questions unanswered concerning smoking's effects on sweet taste perception and hedonics. First, it is unclear what role nicotine may play in these effects with humans. Animal research indicates that nicotine is the active ingredient of tobacco smoke responsible for the effects of smoking on body weight (14,23). More importantly, results from animal studies tend to support some of the effects of smoking in humans described above by demonstrating reduced sweet food intake following chronic nicotine administration (12, 15, 16). Thus, nicotine may be the specific component of tobacco which influences sweet taste hedonics and, possibly, perception. This would be important to document, as it would suggest that smoking treatment strategies involving nicotine replacement (i.e., nicotine polacrilex) may prolong these influences on taste, which could therefore help explain why nicotine replacement reduces weight gain after cessation (8, 9, 11).

Second, to our knowledge, none of the research on effects of smoking on sweet taste perception or hedonics has manipulated fat content of sweet samples. This is an important omission since most sweet foods are high in fat content as well as sugar, and perception and hedonics of sweet foods increases with increasing levels of fat content (6,7). Some animal research has demonstrated that nicotine reduces consumption of sweet, high-fat foods in rats (15), but the specific role of fat content has been ignored, and the smoking/nicotine research has examined taste perception and hedonics as a function of sucrose concentration only. Exploration of nicotine's effect on perception and hedonics of sweet/fat taste may help explain the variability in results of previous research. In addition, since sweet, high-fat foods are denser in calories than sweet, low-fat foods, reduced hedonics specifically of sweet/fat taste due to smoking or nicotine may provide important information in understanding smoking's effect on caloric intake and body weight.

Finally, there may be differences between smokers and nonsmokers in the acute effects of nicotine on taste perception and hedonics, as suggested by the study of smokeless tobacco users and nonusers by Mela (22), noted above. Such differences could indicate that long-term exposure to tobacco or nicotine produces adaptation to nicotine's effects, suggestive of chronic tolerance. Chronic tolerance would indicate that nicotine's effects on sweet and fat taste perception and/or hedonics are more pronounced when smokers initially adopt the smoking habit, thus perhaps explaining the strong belief in smoking's weight controlling effects among new smokers (5).

The present study examined the effects of nicotine intake on perception and hedonics of milk samples varying in sucrose and fat content and compared responses to nicotine between smokers and nonsmokers. Nicotine was presented via measured dose nasal spray to isolate nicotine's effects from those of other components of tobacco smoke and to equate nicotine exposure across subjects varying in experience inhaling tobacco smoke [i.e., smokers vs. nonsmokers (19)].

METHOD

Subjects

Smokers and nonsmokers ($n = 10$ males each) were similar with respect to age and body weight. Mean (range) age was 23.2 (18–29) years for smokers and 21.7 (19–26) years for nonsmokers, while mean body weight was 74.7 (58.2–94.5) kg for smokers and 75.5 (65.2–83.6) kg for nonsmokers. Smokers smoked a mean of 19.8 (15–23) cigarettes per day for 5.1 (1–10) years and denied current use of other tobacco products, such as chewing tobacco or snuff. Nonsmokers denied any past regular use of tobacco.

Sweet/Fat Taste Samples

There were 16 milk samples varying in sucrose and fat content (4 levels of each) for determination of sensitivity and hedonics of sweet/fat taste. Each sample contained 10 ml of skim milk (0.1% fat w/w), whole milk (3.5%), half and half (11.7%), or heavy cream (37.6%), with 0 (0% w/w), 0.62 (5%), 1.25 (10%), or 2.50 ml (20%) of sucrose dissolved thoroughly in each. This procedure is similar to that of Drewnoski and Greenwood (6). Samples were prepared and then refrigerated (7°C) for approximately 1 hr before testing.

Nicotine and Placebo Presentations

Nicotine and placebo were presented via nasal spray pump, which has been shown to produce reliable linear, dose-dependent increases in plasma nicotine (28,29). The nicotine dose was 15 μg per kg of body weight, or a mean of 1.1 mg, which is similar to the typical nicotine consumption of most smokers from one cigarette (4). The dose consisted of 1.14 ml of 0.9% sodium chloride solution together with L-nicotine and 10 mg of a peppermint flavoring oil (Lorann Oils, Lansing, MI), which was used to mask the smell of nicotine. The placebo (0 mg) contained only the sodium chloride solution with masking agent. This dosing method has previously been described in more detail (26, 28–30).

Procedure

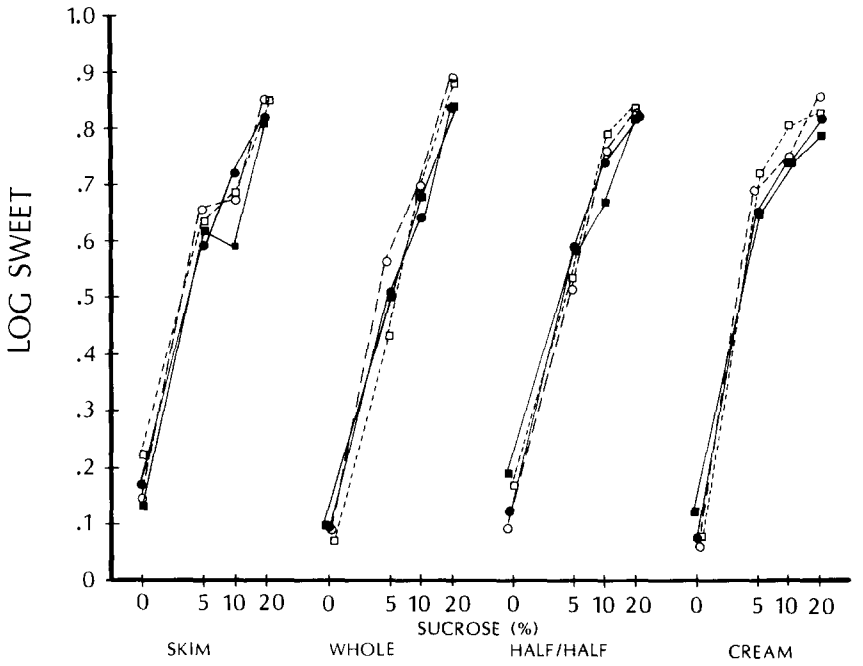
Subjects participated in 2 sessions on 2 separate mornings, each after overnight abstinence from smoking, confirmed by expired air carbon monoxide (CO) reading of ≤ 13 ppm (3). Nonsmokers also provided expired air samples to ensure equal treatment between groups and to maintain experimenter blindness to subject smoking status. Subjects arrived at the lab at 8:00 a.m. Throughout each session, subjects sat in a comfortable armchair in a sound-attenuated experimental chamber and were allowed to consume only water while quietly watching television. Subjects were presented with nicotine during one session and placebo during the other via nasal spray every 20 min for 2 hr prior to taste testing. This procedure was designed to simulate typical morning exposure of regular smokers to nicotine. Order of nicotine vs. placebo was counterbalanced between the two sessions.

After a final, seventh dose presentation, subjects engaged in the test of sweet/fat taste perception and hedonics. The 16 samples were presented individually in random order. Subjects were instructed in the "sip and spit" method of taste testing, in which they were to place the sample in their mouth, move it around for a few seconds without swallowing any, and then spit it out into a cup, at which time they were to provide hedonic and intensity ratings, according to the procedures of Redington (32). Hedonics was determined by rating the sample for "liking" on a 1 ("Extremely dislike") to 9 ("Extremely Like") scale, with 5 ("Neither like nor dislike") as the midpoint. Intensity was determined by rating the sample for sweetness on a 1 ("Not at all sweet") to 9 ("Extremely sweet") scale, with 5 ("Moderately sweet") as the midpoint, and for fattiness on a similar 1 ("Not at all fatty") to 9 ("Extremely fatty") scale. (Sweet and fat taste sensitivity were each derived from the slopes of the intensity ratings across sucrose and fat concentrations, as noted below.) The testing of each sample was separated by 1 min, during which subjects made their ratings, rinsed their mouths with water, and waited for the next sample.

This protocol was approved by the Institutional Review Board for Biomedical Research at the University of Pittsburgh.

Analyses

Each of the ratings for sweet and fat intensity was first converted to log units and the slope of the regression of log



SLOPES OF SWEET TASTE SENSITIVITY:

	SKIM	WHOLE	HALF/HALF	CREAM
○ Smokers/Plac	.295	.325	.318	.350
● Smokers /Nic	.278	.302	.306	.334
□ Nonsmokers/Plac	.258	.323	.293	.329
■ Nonsmokers/Nic	.278	.306	.261	.297
Overall means across groups	.277	.314	.294	.327

FIG. 1. Relation of sucrose concentration with log of sweet intensity rating and slopes of sweet taste sensitivity at each level of fat concentration for smokers and nonsmokers following placebo and nicotine.

concentration on log intensity was calculated to derive measures of sensitivity to each taste (32,35). Larger slopes indicate greater sensitivity to each taste. The logs of sweet and fat taste intensity ratings and the hedonic ratings were each analyzed by a four-factor mixed, repeated-measures ANOVA, involving smoking status as the between-subjects factor, and nicotine/placebo, sucrose concentration (4 levels), and fat concentration (4) as within-subjects factors. The sensitivity slopes for sweet and fat taste were analyzed by a similar three-factor ANOVA, without the sucrose (sweet taste analysis) or fat (fat taste analysis) concentration as factors since the slopes of each represented the relationship of intensity rating with concentration.

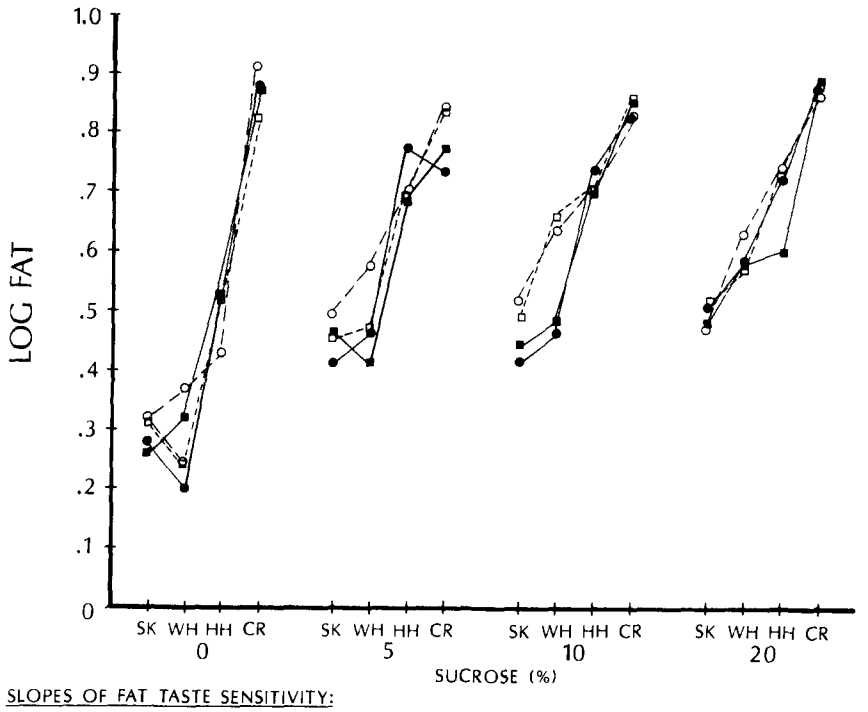
RESULTS

Taste Intensity and Sensitivity

The relationship between sucrose concentration and sweet taste intensity is presented in Fig. 1 at each level of fat concentration for smokers and nonsmokers following placebo and nicotine. As expected, sweet taste intensity was significantly related to sucrose concentration, $F(3,54) = 238.08$, $p<0.001$. Sweet taste intensity was also related to fat concentration, $F(3,54) = 4.26$, $p<0.01$, and to the interaction of sucrose × fat concentration, $F(9,162) = 5.68$, $p<0.001$, as the higher sucrose concentrations tended to be perceived as "sweeter" at the higher fat concentrations while the lowest (0%) sucrose concentration was perceived as less sweet at higher fat concentrations. Nicotine, however, did not affect intensity of sweet taste, $F(1,18) = 1.22$, ns. Furthermore, smoking status was unrelated to sweet taste intensity, as there were no significant main or interaction effects involving smoking status.

The relationship between fat concentration and fat taste intensity is presented in Fig. 2 at each level of sucrose concentration for smokers and nonsmokers following placebo and nicotine. Similar to sweet taste intensity, fat taste intensity was significantly related to fat concentration, $F(3,54) = 113.68$, $p<0.001$, sucrose concentration, $F(3,54) = 16.76$, $p<0.001$, and the sucrose × fat interaction, $F(9,162) = 10.13$, $p<0.001$. The sucrose × fat interaction was due to the lower fat concentrations being perceived as more "fatty" at the higher sucrose concentrations. Nicotine significantly reduced intensity of fat taste, $F(1,18) = 5.23$, $p<0.05$, and this effect appeared to be more prominent at lower fat concentrations, although the interaction of nicotine × fat concentration was not significant, $F(3,54) = 2.00$, $p>0.10$. However, as with sweet taste intensity, there were no main or interaction effects of smoking status on fat taste intensity.

Sensitivity slopes for sweet taste at each level of fat concentration are also presented in Fig. 1, and slopes for fat taste at each level of sucrose concentration are presented in Fig. 2. Similar to intensity, sensitivity to sweet taste was significantly related to fat concentration, $F(3,54) = 3.06$, $p<0.05$, as sweet sensitivity in-



SLOPES OF FAT TASTE SENSITIVITY:

○ Smokers/Placebo	.180	.126	.113	.148
● Smokers/Nicotine	.203	.137	.157	.131
□ Nonsmokers/Placebo	.169	.138	.132	.128
■ Nonsmokers/Nicotine	.206	.118	.146	.131
Overall Means Across Groups	.189	.130	.137	.134

FIG. 2. Relation of fat concentration with log of fat intensity rating and slopes of fat taste sensitivity at each level of sucrose concentration for smokers and nonsmokers following placebo and nicotine (SK, skim milk; WH, whole milk; HH, half/half; CR, heavy cream).

creased at higher levels of fat concentration. Sensitivity to fat taste was also significantly related to sucrose concentration, $F(3,54) = 5.13$, $p<0.01$, but sensitivity *decreased* from 0% to 5% sucrose and remained decreased across sucrose concentrations. However, nicotine had no significant effect on sensitivity to either sweet taste, $F(1,18) = 1.32$, ns, or fat taste, $F(1,18)<1$, and there were no differences between smokers and nonsmokers on sensitivity to either taste (both $Fs<1$). There were also no significant interaction effects involving nicotine or smoking status on sweet or fat taste sensitivity.

Taste Hedonics

The relationship between taste hedonics and sucrose concentration is presented at each level of fat concentration in Fig. 3 for smokers and nonsmokers following placebo and nicotine. Taste hedonics was significantly related to sucrose concentration, $F(3,54) = 57.62$, $p<0.001$, and to sucrose × fat interaction, $F(9,162) = 6.30$, $p<0.001$, but not to fat concentration, $F(3,54) = 1.15$, ns. The sucrose × fat interaction appeared due to decreased hedonics for 0% but increased hedonics for 5% and 10% sucrose at higher levels of fat. There was no effect of nicotine on taste hedonics, $F(1,18)<1$, and no interactions involving nicotine. However, mean hedonic ratings, regardless of whether they followed nicotine or placebo, were lower for smokers compared with nonsmokers, $F(1,18) = 4.21$, $p = 0.055$. This difference was equal across sucrose concentrations, as the interaction of smoking status × sucrose was not significant, $F(3,54) = 1.42$, ns. Although there were no other significant interactions, smokers appeared to show a nonlinear hedonic response to sucrose concentration at the highest fat level (Fig. 3). For smokers, peak hedonic rating ["break point" (7)] was for 5% sucrose following nicotine and 10% sucrose following placebo. For nonsmokers, hedonics peaked at 10% sucrose following nicotine and at 10 and 20% sucrose following placebo. Thus, at the highest fat concentration, hedonic ratings of smokers tended to decline or remain level from 5–20% sucrose, while hedonic ratings for nonsmokers tended to continue to increase.

DISCUSSION

The results of this study indicate that nicotine acutely reduces the intensity of fat taste in both smokers and nonsmokers, but it has no effect on intensity or sensitivity to sweet taste in either group. Furthermore, lack of differences in taste perception between smokers and nonsmokers following placebo indicates no

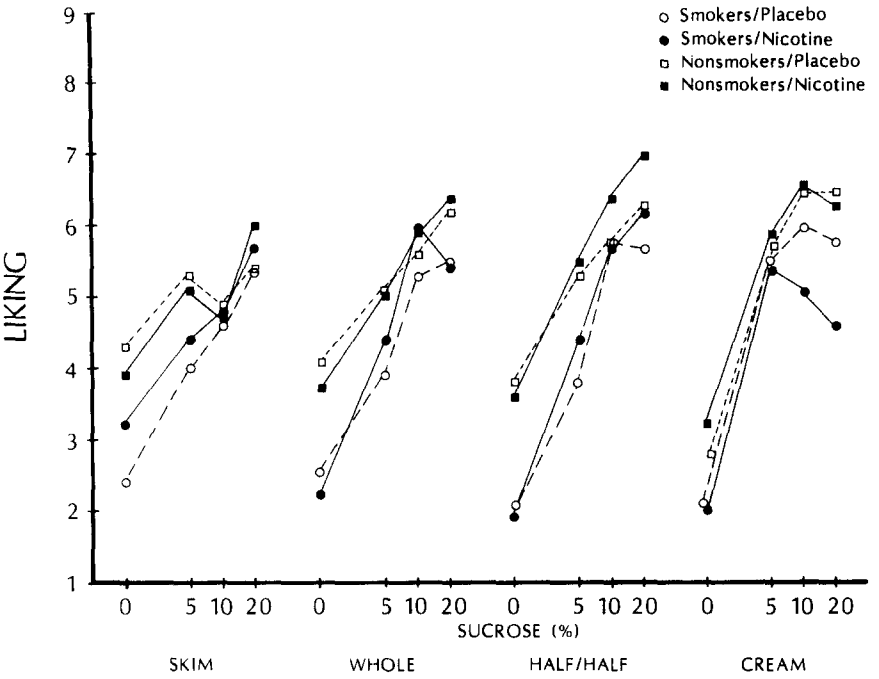


FIG. 3. Relation of sucrose concentration with hedonic rating at each level of fat concentration for smokers and nonsmokers following placebo and nicotine.

chronic effect of smoking on taste perception. These findings are consistent with past research showing no chronic or acute effects of smoking on sweet taste perception [e.g., (20, 31, 32)]. Only research on smokeless tobacco (22), noted earlier, has found a difference in sweet taste perception as a function of any chronic or acute tobacco use. Thus, while the results of this and previous studies indicate that smoking or nicotine intake does not affect sweet taste perception, it is conceivable that specific use of smokeless tobacco may alter taste perception, perhaps because of the oral site of contact.

Similarly, acute nicotine intake did not affect hedonics of sweet/fat taste in smokers or nonsmokers, suggesting that if there is any acute effect of smoking on taste hedonics it must be via nonnicotine constituents of tobacco smoke. However, smokers did show marginally reduced hedonics of sweet/fat taste compared with nonsmokers, regardless of whether they received nicotine or placebo. This finding indicates that regular smoking may gradually induce a chronic alteration in taste hedonics which is unaffected by acute nicotine intake, although smoking apparently has no chronic effect on perception of either sweet or fat taste. In addition, the effect of smoking on hedonics may be quite long term, as we recently found no change in sweet taste hedonics in female smokers after week-long cessation, although only sucrose concentration and not fat concentration was manipulated in that study (27). Nonetheless, Rodin (34) reported an increase in hedonics of sweet taste (also with no manipulation of fat) among ex-smokers 2 weeks after smoking cessation, compared with continuing smokers. This effect was apparent primarily at the highest sucrose concentration (1.0 molar vs. 0.60 molar in the present study) and was due as much to a decline in hedonics across time among continuing smokers as to an increase in hedonics among ex-smokers. On the other hand, another recent study found the opposite effect; greater hedonics of sweet taste among smokeless tobacco users compared with nonusers (22). It may be important to point out that the present study and the study of smokeless tobacco users examined only males, while Rodin's (34) sample consisted primarily of female smokers. Thus, some of the inconsistency across these studies may be related to gender of subject samples, as well, perhaps, as variability in other relevant characteristics such as length of smoking history. Finally, it is possible that our results have little or no bearing on whether regular smoking produces a chronic change in taste hedonics since the smokers and nonsmokers of the present study may have differed in other relevant ways, besides smoking status, which affect sweet taste hedonics. Yet, smokers and nonsmokers were matched on age and body weight and we know of no other possible differences between groups.

Although nicotine's lack of acute effect on sweet taste perception was matched by its lack of effect on taste hedonics, other results may cast some doubt on the relation between taste perception and hedonics. Smokers and nonsmokers did not differ on sweet or fat taste perception, but smokers had reduced hedonics of sweet/fat taste. Furthermore, as noted previously, Mela (22) found decreased sensitivity but increased hedonics of sweet taste in smokeless tobacco users compared with nonusers. Thus, findings on the effects of tobacco use or nicotine on taste perception should be kept distinct from findings on taste hedonics (21).

The lack of effects of acute nicotine intake on taste perception and hedonics in this study suggests that prevention of weight gain after cessation due to nicotine replacement may be via mechanisms other than its effect on sweet taste perception and hedonics. However, as with smokeless tobacco, the oral site of nicotine contact with nicotine polacrilex may lead to effects on taste different from nicotine intake via other routes. Aside from examining route of nicotine intake, further research should examine the separate actions of nicotine and other smoke components since it is still possible that nonnicotine constituents of tobacco smoke may independently affect taste hedonics and, perhaps, perception. It is clear that such research should also take into consideration the fat content of sweet samples, as this study

showed that interaction of sucrose and fat content was significantly related to both sweet taste intensity and hedonics. In addition, possible differences between males and females should be examined, as noted previously. Animal research has found gender differences in nicotine effects on sweet food intake (16), and epidemiological research indicates females may experience greater differences in body weight as a function of smoking (24). Finally, the possibility of a gradual change in taste hedonics following cessation should be explored longitudinally and directly compared with change in caloric intake and body weight in order to more clearly determine the relationships among hedonics, intake, and weight gain.

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