Psychopharmacology (2005) 179: 430–436
DOI 10.1007/s00213-004-2037-x

ORIGINAL INVESTIGATION

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Calorie restriction increases cigarette use in adult smokers

Received: 17 May 2004 / Accepted: 8 September 2004 / Published online: 25 November 2004

Abstract *Rationale:* Cigarette smokers weigh less than nonsmokers, and smokers often gain weight when they quit. This is a major barrier to smoking cessation, especially among women. However, strict dieting is not recommended during smoking cessation out of concern that it might promote relapse. This concern derives, in part, from the observation that calorie restriction increases self-administration of drugs of abuse in animals. This relationship has never been experimentally demonstrated in humans. *Objectives:* To evaluate whether calorie restriction increases cigarette smoking in humans. *Methods:* Seventeen (nine males, eight females) healthy, normal-weight smokers not attempting to quit were cycled in partially counterbalanced order, double-blind, through four diets—normal calorie (2,000–2,800 kcal/day), low calorie (700 kcal/day deficit), low-carbohydrate (CHO)/normal-calorie, and low-CHO/low-calorie—for 6 days per diet in an inpatient research ward. Smoking was assessed by cigarette counts, breath carbon monoxide (CO) levels, and cigarette craving. *Results:* Compared with the normal-calorie diet, while on the low-calorie diet, subjects smoked 8% more cigarettes ($P<0.02$) and had 11% higher breath CO levels ($P<0.01$). The low-CHO/normal-calorie diet showed no significant effect on either variable, but there was a 15% increase in breath CO levels ($P< 0.05$) on the low-CHO/low-calorie diet. There were no changes in self-reported cigarette craving or mood. *Conclusions:* Consistent with animal studies, moderate calorie restriction was associated with a small but statistically significant increase in cigarette smoking, with no independent effect of CHO deprivation. These findings suggest that dieting may increase smoking behavior and could impede smoking-cessation attempts.

Keywords Cigarettes · Calorie restriction · Human

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Introduction

This study examined the intersection of two important public health problems—tobacco dependence and dieting. Tobacco dependence has emerged as a leading preventable cause of morbidity and mortality in the United States (Mokdad et al. 2004). Concern over weight gain is a major barrier to smoking cessation, especially among women, and weight gain may be a factor in relapse (Fiore et al. 2000; Gritz et al. 1996; Perkins et al. 2001; Pomerleau et al. 2001). Smokers weigh less than non-smokers (Froom et al. 1998) and smoking cessation is associated with weight gain (Froom et al. 1998; Jeffery et al. 2000; Ockene et al. 2000; Varner 1999)—an average of 6 kg in continuously abstinent individuals (Klesges et al. 1997), and dieters trying to lose weight are more likely to smoke (Wee et al. 2001). The reasons for cessation weight gain are not entirely clear, although some studies have found changes in energy intake (Perkins 1992) and/or metabolic rate (Klesges et al. 1989;

Perkins 1993). It is also known that nicotine has an anorectic effect in humans (Jo et al. 2002; Li et al. 2003).

Knowledge of this association between smoking and weight gain can lead to dieting while attempting smoking cessation. However, dieting during smoking cessation tends to increase the risk of relapse (Borrelli et al. 2001; Hall et al. 1992; Ockene et al. 2000), and smokers undergoing rapid weight loss tend to smoke more than those who do not (Niaura et al. 1992). Conversely, female smokers who gain weight during smoking cessation are less likely to relapse than those who maintain their pre-cessation body weight (Borrelli et al. 2001; Ockene et al. 2000). For these reasons, practice guidelines for smoking cessation recommend against dieting during cessation attempts (Fiore et al. 2000).

Animal studies support a link between reduced food or calorie intake ("dieting") and increased drug intake. Food restriction in rodents or monkeys significantly increases the self-administration of a variety of abused drugs, including opiates, stimulants, and phencyclidine (PCP) (Carr et al. 2000; Comer et al. 1995; Carroll et al. 2001; Rodefer and Carroll 1996). Parallels between animal and human drug-taking behavior suggested that this phenomenon might occur in humans and that food restriction might contribute to human drug seeking (Griffiths et al. 1980).

A few anecdotal reports are consistent with an inverse relationship between food intake and drug intake in humans. Caffeine and nicotine consumption was increased in World War II conscientious objectors subjected to "experimental semi-starvation" (Franklin et al. 1948). Conversely, coca leaf chewing was decreased when malnourished Peruvian Indians were fed a high-calorie diet (Hanna and Hornick 1977).

Controlled human experimental studies have not replicated this effect of food restriction. Studies of food deprivation for 18, 19, or 24 h found no effect on cigarette smoking (Bulik and Brinded 1994; Zacny and de Wit 1990), alcohol intake (Bulik and Brinded 1993), or marijuana self-administration (Zacny and de Wit 1991). Calorie restriction to 50% or 25% of normal intake for 3 days also had no effect on cigarette smoking (Lawson et al. 1997; Zacny and de Wit 1990).

Given the failure of controlled human experimental studies to replicate the findings of animal studies, we conducted a prospective study with a longer period of calorie restriction (6 days) to assess the influence of caloric restriction on the smoking behavior of nicotine-dependent men and women. Because of the suggestion that carbohydrate (CHO) intake might have a specific influence on drug use (Carroll 1998), we included an independent manipulation of CHO intake.

Materials and methods

Subjects

Subjects were 17 (9 men and 8 women) self-referred, nicotine-dependent (DSM-IIIR criteria), paid research volunteers, recruited primarily by newspaper advertisements. All applicants were pre-screened by telephone, then scheduled for in-person additional screening and evaluation. Inclusion criteria were: cigarette consumption of one to two packs per day, a Fagerstrom tolerance questionnaire (FTQ) (Heatherton et al. 1991) score of 7 or more (indicating severe nicotine dependence) and body mass index of between approximately 19 and 25. Exclusion criteria included illiteracy or inability to comprehend research procedures; any self-reported use of illegal drugs; average consumption of more than one drink of alcohol per day [to avoid having subjects experience alcohol withdrawal while on the research ward, exclude subjects whose usual diet provided a significant proportion of calories from alcohol, and avoid a possible effect of change in alcohol intake (no alcohol was allowed on the research ward) on smoking (Sayette 2002; Romberger and Grant 2004)]; current major medical problems, or illness associated with changes in appetite [based on medical history, physical examination, blood chemistry profile, complete blood cell count (CBC), urinalysis and a 12-lead electrocardiogram (EKG)]; current clinically significant psychological symptoms [based on a symptom checklist 90-R (Derogatis 1983) general severity index (GSI) T-score of 63 or more]; or current weight-loss attempt through dieting.

There were 35 individuals who underwent the in-person screening, 21 of whom met all eligibility criteria and signed a written consent form approved by the Johns Hopkins Bayview Medical Center Institutional Review Board. Of those who consented, 17 completed the study and were included in the data analysis. These 17 subjects included 7 white men, 5 white women, 1 African-American man, 2 African-American women, 1 Native American man, and 1 Hispanic woman, had a mean±SD age of 35.1±8.1 years and an education level of 12.2±2.4 years. Three subjects were currently married, six never married, and eight widowed, separated, or divorced. Subjects had been smoking for 20.0±7.9 years, currently smoked 30.5±7.0 cigarettes per day, and had a FTQ score of 8.7±0.9. There was no significant difference in degree of nicotine dependence based on FTQ scores between completers and non-completers. Of the four non-completers, one did not start, two dropped out for personal reasons, and one was non-compliant with the protocol.

Diet

Prior to admission to the inpatient General Clinical Research Center (GCRC) of the Johns Hopkins Bayview Medical Center, all subjects were interviewed by a registered dietitian to assess current diet and food preferences. This information, along with the Harris-Benedict equation (Harris and Benedict 1919), was used to construct their normal- and low-calorie diets. After admission, all subjects were cycled (in partially counterbalanced order so that no subject received both of the low-calorie diets consecutively) through each of four diets, spending 6 days on each diet: normal calorie (ranging from 2,000 kcal/day to 2,800 kcal/

day), low-calorie/balanced (700 kcal/day balanced deficit), low-CHO/normal calorie, and low-CHO/low-calorie (700 kcal/day deficit). Low-CHO diets contained 20% of total calories or fewer from simple and complex CHOs. The diets were all made as palatable as possible by choosing foods on an individualized basis which met both study guidelines and subject preferences.

A Latin-square, double-blind, random-assignment design was used. Assignment to diet order was stratified by gender to help control for possible gender effects on smoking behavior and metabolic rate. Subjects remained on the GCRC for six consecutive days. They were released on the evening of the sixth day and returned to the ward on the evening of the following day to start another diet the next morning. This provided a 39-h washout period between diets.

Several procedures were employed to control diet compliance. All food was served and consumed at three specified meal times and one evening snack. Only 15 min was allowed for food consumption at each period. No additional food was allowed. Caffeine was limited to one cup of coffee per day. Meals were weighed by staff before and after ingestion, total daily input\output was documented, and calorie counts were calculated. Vital signs and weights were obtained daily.

The influence of the dietary manipulations was assessed by both physiological and psychological measures. Resting metabolic rate was measured by indirect calorimeter (Deltatrac, Sensormedics, Anaheim, CA) on the first and last days of each diet period. Metabolic rate was measured for 45 min after subjects had rested for 30 min without smoking. Testing was done between 0700 hours and 0900 hours, before breakfast. The calorimeter was periodically calibrated with known concentrations of carbon dioxide and oxygen. Respiratory quotient was calculated as the ratio of carbon dioxide minute volume to oxygen minute volume, as measured by the indirect calorimeter. Urine ketone levels (a measure of ketosis) were measured the morning of each inpatient study day by dipstick (Bili-Labstix, Miles Diagnostics, Elkhart, IN; sensitivity=5–10 mg/dl for detection of acetoacetic acid). Current hunger ("How hungry do you feel right now?") was measured utilizing a 100-mm visual analog scale, which was anchored by "not at all" and "extremely" (Silverstone and Fincham 1978). It was self-administered eight times daily—30 min before and 1 h after each meal and the evening snack.

Cigarette smoking

Subjects kept a pack of their preferred brand of cigarettes with them at all times. Smoking was assessed using two commonly used measures: number of cigarettes smoked, a behavioral measure, and expired breath carbon monoxide (CO) level (a biological measure of actual smoke intake that is considered to reflect nicotine intake and is associated with tobacco dependence severity; Hatsukami et al. 2004; Stratton et al. 2001). Subjects kept a half-hourly log of all cigarettes smoked, measured to the nearest eighth of a cigarette with the help of a cigarette diagram. Daytime napping was discouraged. Expired breath CO levels were measured 30 min before and 30 min after each meal and after the evening snack using an Ecolyzer 2000 (National Draeger, Pittsburgh, PA). Current craving for cigarettes ("desire for a cigarette right now") was assessed by the subjects 30 min before and 1 h after each meal utilizing a 100-mm analog scale (Hughes and Hatsukami 1986).

Mood

Each subject's mood was self-assessed daily 30 min before and 30 min after each meal using a 9-item, 7-point Likert scale (Diener and Emmons 1984). Three indicators of present mood (mean score from all sessions of day 5 and day 6) were analyzed to detect changes among diet conditions: positive mood state (sum of responses to "happy" and "joyful"), negative mood state (score of responses to depressed/blue and "unhappy"), and anxiety (response to "worried/anxious").

Statistical analyses

Planned comparisons were made between data collected during the normal diet and the experimental diets using a one-way, repeated-measures analysis of variance (ANOVA). In order to minimize any carry-over effect from the previous week's diet and to allow time for the effect of the dietary manipulations to become manifest, only data collected during day 5 and day 6 were analyzed—a period of 34 h (data were not collected after 1700 hours on the final inpatient day). Assumptions of normality were violated for the variables food craving and breath CO ($W=0.94–0.95$, $P=0.01$). For these measures, ranks were calculated and used in the ANOVAs.

Because meal time can affect smoking behavior, a separate analysis was also carried out for pre-meal and post-meal data for breath CO, as the most reliable measure of smoking behavior. Pre-meal and post-meal data were defined as the breath CO obtained one half hour preceding meals and one half hour following meals, respectively. Planned comparisons were again made between the experimental diets and the normal diet using a one-way, repeated-measures ANOVA. Ranks were used in the ANOVA because this measure was not normally distributed. All tests were considered significant at a two-tailed alpha level of 0.05.

For significant comparisons, a mean of the subjects' percentage changes in response between the normal and each of the experimental diets was calculated.

For comparison purposes, we did a similar analysis for the number of cigarettes smoked and mean breath CO over the first 3 days of each 6-day diet period. This allowed us to

evaluate the effect of a 3-day diet manipulation, the longest duration used in previously published controlled human experimental studies.

Because some studies have shown sex differences in the effects of smoked nicotine and in smoking cessation treatment (Perkins 2001; Perkins et al. 2002; Piper et al. 2001), we also analyzed data on number of cigarettes smoked and breath CO over the last 2 days of each diet period using a two-way ANOVA (diet×sex). This allowed us to evaluate the influence of sex on any diet effect.

Results

Metabolic parameters

There were no significant differences found between the normal diet and the experimental diets in resting metabolic rate (measured in kcal/day; Table 1). As expected, the respiratory quotient was lower on both ketogenic (both low-CHO) diets than on the normal diet (P=0.001 and P=0.002; Table 1). On the low-CHO/normal calorie, and low-CHO/low-calorie diets, 66% and 59% of the subjects, respectively, exhibited ketonuria of "small" or greater magnitude by dipstick, compared with 0% and 6%, respectively, while on the normal calorie and low-calorie/balanced diets (P<0.001, Fisher's Exact Test). Subjects on the low-CHO/normal-calorie diet who had ketonuria had a significantly lower respiratory quotient than those who did not have ketonuria (0.77±0.04 versus 0.86±0.06, t=3.13, P=0.02), consistent with their primary metabolic substrate being fat rather than CHO. Subjects on the low-CHO/low-calorie diet had similarly low respiratory quotients (0.78±0.07 versus 0.79±0.05, t=0.29, P=0.78) whether or not they had ketonuria, probably because the overall calorie restriction forced more metabolism of fat, regardless of the generation of ketonuria by the CHO restriction. Mean weight loss over the 6-day diet period was 0.07±0.42 kg on the normal-calorie diet. This was significantly different (P<0.005) from weight loss on the low-calorie/balanced diet (0.73±0.57 kg), the low-CHO/normal calorie diet (0.57±0.50 kg), and the low-CHO/low-calorie diet (0.79±0.25 kg).

Cigarette use

While on the low-calorie/balanced diet, subjects smoked a mean±SD of 4.0±5.4 or 8% more cigarettes than when on the normal diet (P<0.02; Table 1). On the low-calorie/balanced diet, 13 subjects demonstrated increased smoking, 3 decreased smoking, and 1 showed no change (P=0.02 by sign test). There was no significant difference between the normal diet and any other diet.

Breath CO levels

Over the final 34 h on the low-calorie/balanced diet, subjects had breath CO levels 11% higher than while on the normal-calorie diet (P<0.008; Table 1), consistent with increased cigarette smoking. While on the low-CHO/low-calorie diet, subjects had CO levels 15% higher than on the normal calorie diet (P<0.04). These differences were present during the post-meal period, but not during the pre-meal period (Table 1).

Cigarette craving (VAS)

There were no significant differences between normal and experimental diets in cigarette craving (Table 1).

Mood

There were no differences between the normal and experimental diets on any measure of mood (Table 1).

Table 1 Effects of calorie restriction (700 kcal/day) and carbohydrate (CHO) restriction (≤20% total calories) on metabolic, cigarette-smoking, and mood variables in 17 adult cigarette smokers (mean, SEM)

Measure	Normal diet	Low-calorie/ balanced diet	Low-CHO/ normal-calorie diet	Low-CHO/ low-calorie diet
Metabolic rate: kcal/24 h (resting)	1415.2, 152.6	1365.8, 140.2	1462.6, 226.8	1414.6, 173.2
Respiratory quotient	0.88, 0.08	0.86, 0.08	0.80, 0.04 **	0.78, 0.04 **
Cigarette use: total no. in last 34 h	50.21, 13.32	54.21, 11.05 *	52.01, 13.23	52.38, 13.81
Cigarette craving: total	44.1, 24.3	41.1, 21.9	43.4, 26.0	39.0, 23.9
Cigarette craving: pre-meal	47.4, 23.9	44.5, 21.0	45.0, 23.5	39.4, 26.0
Cigarette craving: post-meal	43.4, 26.4	40.1, 25.2	43.0, 29.77.2	39.1, 23.5
Breath Co, Ppm: last 34 h	34.87, 16.74	38.77, 17.36 **	36.30, 15.05	39.97, 16.78 *
Breath Co: pre-meal	33.28, 16.37	35.22, 15.38	34.25, 13.73	37.19, 15.75
Breath Co: post-meal	35.14, 17.40	39.63, 18.10 **	36.71, 15.54	40.89, 16.95 *
Positive mood	0.60, 0.33	0.68, 0.37	0.65, 0.33	0.57, 0.29
Negative mood	4.24, 1.36	4.25, 1.40	4.25, 1.36	4.00, 1.28
Anxious mood	0.10, 0.12	0.22, 0.25	0.09, 0.08	0.16, 0.16

**P<0.01 *P<0.05

First 3 days of diet period

There was no significant effect of diet on either number of cigarettes smoked ($P>0.6$) or mean breath CO ($P>0.1$) when considering only the first 3 days of each diet period.

Effect of subjects' sex

There was no significant interaction of subjects' sex with the diet effect on either number of cigarettes smoked ($P>0.5$) or breath CO level ($P>0.4$) over the final 2 days of each diet period.

Discussion

We report here the first controlled, prospective study demonstrating that caloric restriction increases drug use in humans, although the statistically significant effect was of small magnitude and occurred in both measures of smoking with only one of the two low-calorie diets. The balanced low-calorie diet resulted in modest but statistically significant increases in both number of cigarettes smoked and expired breath CO. The low-CHO/low-calorie diet (ketogenic) had no significant effect on number of cigarettes smoked, but did produce a significant 15% increase in expired breath CO over the normal calorie diet. The significant effect of both low-calorie diets on expired breath CO supports the validity of these findings. Expired breath CO, as a biomarker of actual smoke intake, is generally considered a more meaningful measure of smoking with respect to nicotine intake and tobacco dependence severity than is the behavioral measure of number of cigarettes smoked (Hatsukami et al. 2004; Stratton et al. 2001).

The low-CHO/normal-calorie diet (also ketogenic) had no effect on either variable. However, the power to detect effects due to ketosis may have been limited by failure to achieve substantial ketosis in many subjects, as suggested by the fact that one-third or more of subjects on the ketogenic diets did not exhibit ketonuria.

Previous controlled human studies have failed to demonstrate the predicted relationship between calorie restriction and substance intake [cigarettes (Bulik and Brinded 1994; Zacny and de Wit 1990; Lawson et al. 1997), alcohol (Bulik and Brinded 1993), or marijuana (Zacny and de Wit 1991)] that is consistently found in animal studies (Carr et al. 2000; Comer et al. 1995; Rodefer and Carroll 1996). This failure may be due to an important design difference between those studies and the present study. Previous studies used periods of food restriction of up to 3 days, while the present study used a period of 6 days. It is possible that longer periods of food restriction are required to produce measurable changes in drug intake in humans. This interpretation is consistent with our finding of no significant effect on number of cigarettes smoked or breath CO level when considering only the first 3 days of diet manipulation. Most animal studies [but not all (Carroll 1985)] maintained food restriction for at least 1 week (Carr 2002; Rodefer and Carroll 1996). The relationship between duration of food restriction and effect on drug intake remains to be studied. The mechanism of the food restriction effect cannot be determined from this study. Analysis of secondary dependent variables suggests that alterations in cigarette craving and mood were not a factor (Table 1), although the ketogenic effect of the low-CHO diets was not consistent in this sample.

Subjects in this study lived in the structured, artificial environment of a research ward, with constraints on the amount and time of their eating that are not present in the non-research setting. These differences may limit the generalizability of our findings to the real world. However, we believe that the magnitude (25% calorie reduction) and duration (6 days) of food restriction in this study realistically reflect the actual experience of many dieting cigarette smokers and other substance users. Despite the small size of the effect (8% more cigarettes smoked), any increase in smoking tendency might impede the difficult process of smoking cessation. Therefore, our findings support the suggestion in clinical guidelines (Fiore et al. 2000) that dieting during a smoking-cessation attempt may be counterproductive. It may be more appropriate for would-be quitters to simply avoid excess eating, especially of calorie-dense foods, and exercise regularly. Once ex-smokers have achieved stable abstinence, judicious use of dieting may be appropriate if weight gain has been excessive. This approach tends to minimize overall health risk, because the medical consequences of modest weight gain are less serious than those associated with continued cigarette smoking (Fiore et al. 2000; McGinnis and Foege 1993). For smokers not attempting to quit, these findings suggest that dieting may modestly increase health risks to the extent that it increases exposure to cigarette smoke relative to the non-dieting condition. Our findings suggest that more research on the relationship between smoking and dieting is likely to yield important benefits for the treatment of two major public health problems: tobacco use and obesity.

The animal data raise the possibility that caloric restriction may increase use of other drugs of abuse in humans. Because drug abuse often alters the diet of the drug abuser (usually in the direction of decreased intake) (Mohs et al. 1990), our findings have other clinical implications. It is possible that drug-related decreases in food intake could stimulate increased drug use, resulting in further decreases in food intake, stimulating even more drug use, etc., generating a positive feedback effect. Further research is needed to confirm that the inverse relationship between food (calorie) intake and substance use observed in this study with cigarettes (nicotine) also occurs with other substances abused by humans.

Acknowledgements Supported by NIDA intramural funds and NIH grant no. 5MO1-RR-02719. Portions of this work were presented at the 55th and 56th Annual Scientific Meetings of the College on Problems of Drug Dependence, June 1993 and June 1994, respectively. We are indebted to Karen Koffler, M.S., and Helene Pokrywka, M.S., for nutritional assessments; Joan Frank, R.N., for subject care; David Goldsborough, B.A., Nancy Kreiter, M.A., and Matthew Tayback, Sc.D., for help with data analysis; and Lynnia Fiorelli for manuscript assistance.

References

Borrelli B, Spring B, Niaura R, Hitsman B, Papandonatos G (2001) Influences of gender and weight gain on short-term relapse to smoking in a cessation trial. J Consult Clin Psychol 69:511–515

Bulik CM, Brinded EC (1993) The effect of food deprivation on alcohol consumption in bulimic and control women. Addiction 88:1545–1551

Bulik CM, Brinded EC (1994) The effect of food deprivation on the reinforcing value of food and smoking in bulimic and control women. Physiol Behav 55:665–672

Carr KD (2002) Augmentation of drug reward by chronic food restriction: behavioral evidence and underlying mechanisms. Physiol Behav 76:353–364

Carr KD, Gye-Young K, Cabeza de Vaca S (2000) Chronic food restriction in rats augments the central rewarding effect of cocaine and the δ_1 opioid agonist, DPDPE, but not the δ_2 agonist, deltorphin-II. Psychopharmacology 152:200–207

Carroll ME (1985) The role of food deprivation in the maintenance and reinstatement of cocaine-seeking behavior in rats. Drug Alcohol Depend 16:95–109

Carroll ME (1998) Interactions between food and addiction. In: Niesink RJM, Jaspers RMA, Kornet LMW, van Ree JM (eds) Drugs of abuse and addiction: neurobehavioral toxicology. CRC, Boca Raton, pp 286–311

Carroll ME, Campbell UC, Heideman P (2001) Ketoconazole suppresses food restriction-induced increases in heroin self-administration in rats: sex differences. Exp Clin Psychopharmacol 9:307–316

Comer SD, Turner DM, Carroll ME (1995) Effects of food deprivation on cocaine base smoking in rhesus monkeys. Psychopharmacology (Berl) 119:127–132

Derogatis LR (1983) SCL-90R administration, scoring and procedures. Manual II. Clinical Psychometric Research, Towson

Diener E, Emmons RA (1984) The independence of positive and negative affect. J Pers Soc Psychol 47:1105–1117

Fiore MC, Bailey WC, Cohen SJ et al (2000) Treating tobacco use and dependence. Clinical Practice Guideline. Department of Health and Human Services. Public Health Service, Rockville

Franklin JC, Sciele BC, Brozek J, Keys A (1948) Observations on human behavior in experimental semi-starvation and rehabilitation. J Clin Psychol 4:28–45

Froom P, Melamed S, Benbassat J (1998) Smoking cessation and weight gain. J Fam Pract 46:460–464

Griffiths RR, Bigelow GE, Henningfield JE (1980) Similarities in animal and human drug-taking behavior. In: Mello NK (ed) Advances in substance abuse: behavioral and biological research, vol 1. JAI, Greenwich, pp 1–90

Gritz ER, Nielsen IR, Brooks LA (1996) Smoking cessation and gender: the influence of physiological, psychological, and behavioral factors. J Am Med Womens Assoc 51:35–42

Hall SM, Tunstall CD, Vila KL, Duffy J (1992) Weight gain prevention and smoking cessation: cautionary findings. Am J Public Health 82(6):799–803

Hanna JM, Hornick CA (1977) Use of coca leaf in Southern Peru: adaptation or addiction? Bull Narc 29:63–74

Harris JA, Benedict FG (1919) A biometric study of basal metabolism. Rep. Publication No. 279. Carnegie Institute, Washington, DC

Hatsukami DK, Henningfield JE, Kotlyar M (2004) Harm reduction approaches to reducing tobacco-related mortality. Annu Rev Public Health 25:377–395

Heatherton TF, Kozlowski LT, Frecker RC, Fagerstrom KO (1991) The Fagerstrom Test for nicotine dependence: a revision of the Fagerstrom Tolerance Questionnaire. Br J Addict 86:1119–1127

Hughes JR, Hatsukami DK (1986) Signs and symptoms of tobacco withdrawal. Arch Gen Psychiatry 43:289–294

Jeffery RW, Hennrikus DJ, Lando HA, Murray DM, Liu JW (2000) Reconciling conflicting findings regarding postcessation weight concerns and success in smoking cessation. Health Psychol 19:242–246

Jo YH, Talmage DA, Role LW (2002) Nicotinic receptor-mediated effects on appetite and food intake. J Neurobiol 53:618–632

Klesges RC, Meyers AW, Klesges LM, LaVasque ME (1989) Smoking, body weight, and their effects on smoking behavior: a comprehensive review of the literature. Psychol Bull 106:204–230

Klesges RC, Winders SE, Meyers AW, Eck LH, Ward KD, Hultquist CM et al (1997) How much weight gain occurs following smoking cessation? A comparison of weight gain using both continuous and point prevalence abstinence. J Consult Clin Psychol 65:286–291

Lawson RH, Bulik CM, Rodefer JS, Scanlon W, Borger MD (1997) The effect of a reduced energy diet and meal patterns on smoking and coffee drinking in women. Int J Eat Disord 21:137–145

Li MD, Kane JK, Konu O (2003) Nicotine, body weight and potential implications in the treatment of obesity. Curr Top Med Chem 3:899–919

McGinnis JM, Foege WH (1993) Actual causes of death in the United States. JAMA 270:2207–2212

Mohs ME, Watson RR, Leonard-Green T (1990) Nutritional effects of marijuana, heroin, cocaine, and nicotine. J Am Diet Assoc 90(9):1261–1267

Mokdad AH, Marks JSSDF, Gerberding JL (2004) Actual causes of death in the United States, 2000. JAMA 291(10):1238–1245

Niaura R, Clark MM, Raciti MA, Pera V, Abrams DB (1992) Increased saliva cotinine concentrations in smokers during rapid weight loss. J Consult Clin Psychol 60:985–987

Ockene JK, Emmons KM, Mermelstein RJ, Perkins KA, Bonollo DS, Voorhees CC et al (2000) Relapse and maintenance issues for smoking cessation. Health Psychol 19:17–31

Perkins KA (1992) Effects of tobacco smoking on caloric intake. Br J Addict 87:193–205

Perkins KA (1993) Weight gain following smoking cessation. J Consult Clin Psychol 61:768–777

Perkins KA (2001) Smoking cessation in women. Special considerations. CNS Drugs 15:391–411

Perkins KA, Marcus MD, Levine MD, D'Amico D, Miller A, Broge M et al (2001) Cognitive-behavioral therapy to reduce weight concerns improves smoking cessation outcome in weight-concerned women. J Consult Clin Psychol 69:604–613

Perkins KA, Jacobs L, Sanders M, Caggiula AR (2002) Sex differences in the subjective and reinforcing effects of cigarette nicotine dose. Psychopharmacology (Berl) 163:194–201

Piper ME, Fox BJ, Welsch SK, Fiore MC, Baker TB (2001) Gender and racial/ethnic differences in tobacco-dependence treatment: a commentary and research recommendations. Nicotine Tob Res 3:291–297

Pomerleau CS, Zucker AN, Stewart AJ (2001) Characterizing concerns about post-cessation weight gain: results from a national survey of women smokers. Nicotine Tob Res 3:51–60

Rodefer JS, Carroll ME (1996) Progressive ratio and behavioral economic evaluation of the reinforcing efficacy of orally delivered phencyclidine and ethanol in monkeys: effects of feeding conditions. Psychopharmacology (Berl) 128:265–273

Romberger DJ, Grant K (2004) Alcohol consumption and smoking status: the role of smoking cessation. Biomed Pharmacother 58:77–83

Sayette M (2002) The effects of alcohol on cigarette craving. Alcohol Clin Exp Res 26:1925–1927

Silverstone T, Fincham J (1978) Experimental techniques for the measurement of hunger and food intake in man for use in the evaluation of anorectic drugs. In: Garattini S, Saminin R (eds) Central mechanisms of anorectic drugs. Raven, New York, pp 375–382

Stratton K, Shetty P, Wallace R, Bondurant S (2001) Clearing the smoke: the science base for tobacco harm reduction—executive summary. Tob Control 10:189–195

Varner LM (1999) Impact of combined weight-control and smoking-cessation interventions on body weight: review of the literature. J Am Diet Assoc 99:1272–1275

Wee CC, Rigotti NA, Davis RB, Phillips RS (2001) Relationship between smoking and weight control efforts among adults in the United States. Arch Intern Med 161:546–550

Zacny JP, de Wit H (1990) Effects of a 24-hour fast on cigarette smoking in humans. Br J Addict 85:555–560

Zacny JP and de Wit H (1991) Effects of food deprivation on subjective effects and self-administration of marijuana in humans. Psychol Rep 68:1263–1274