TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Haworth et al. (1980)	536 women (234 nonsmokers, 302 smokers) interviewed last prenatal visit (18%) or within day after delivery (82%)	No smoker/nonsmoker pregnancy weight gain difference		Smoking self-report; pregnancy weight gain data only
Hickey and Mulcahy (1973)	150 men (124 smokers); 6-month, 2-year followups after myocardial infarction	Quitter, reducer, continuing smoker differences not significant		Smoking self-report; postmyocardial infarction may motivate healthy behavior
Holme et al. (1985)	16,202 Oslo men, aged 40–49, screening program; 1,232 (elevated cholesterol or upper quartile coronary risk score) randomly assigned diet/smoking intervention or control; 5-year followup	17% controls, 24% intervention quit; 1- to 2-year-quitter weight increased more than controls, then decreased to below prequit level		Smoking self-report; confounded by high cardiovascular disease risk health intervention; weights not reported
Howell (1971)	Retrospective, 1,121 men, aged 40–54; 15- to 20-year weight gain examinations	Light smokers (<20 cigarettes/day) gained 1.9 lb less than heavy smokers, 3.1 lb less than ex-smokers, 3.6 lb less than never smokers	Smoking rate: lower rate related to less weight gain	Retrospective report
Hughes and Hutchinson (1983)	37 smokers and 19 ex-smokers with pulmonary emphysema followed ≥3 years	Smokers lost 0.32 lb/yr, ex-smokers gained 1.17 lb/yr; significant difference		Smoking self-report; pulmonary emphysema population

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Jenkins et al. (1973)	2,318 men (546 never smokers, 359 previous quitters, 547 light smokers, 866 heavy smokers), aged 39–49, 11 California corporations in Western Collaborative Group Study; changes assessed since age 25; 1960–1969 study	Weight loss more likely for light and heavy smokers than never smokers and quitters		Smoking self-report; weights not presented
Kramer (1982)	175 subjects, commercial cessation program (41 nonparticipants or nonlocated, 59 quitters, 75 continuing smokers) ≥ 1-year followup	76% nonsmokers, 56% smokers gained weight; these smokers mean gain 1.7 lb, these nonsmokers mean gain 3.0 lb		All data self-report; high attrition, data loss; presentation incomplete
Lund-Larsen and Tretli (1982)	12,329 men and women, aged 20–49, cardiovascular disease project; 2 screenings 3 years apart	Smokers mean and relative weight less than nonsmokers; female quitters gained 5.95 lb, male quitters 7.84 lb; smoking-starter men lost 1.98 lb, women 5.5 lb; smokers and nonsmokers little/no change	Sex: men, women weight change/smoking cessation and initiation similar	Self-report
Manley and Boland (1983)	39 male, 55 female smokers, cessation program; randomly assigned, 1 of 3 4-week treatments or attention placebo control; 3-month followup; CO verification	31% abstinent at followup: abstainers averaged 10.93 lb gain, relapsers 6.92 lb		Relapser definition unclear

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Noppa and Bengtsson (1980)	1,302 Swedish women, aged 38–60	Current smokers leaner than nonsmokers; At 6 years, quitters gained 7.72 lb; smoking-starters lost 1.54 lb, nonchangers gained 3.09 lb		Smoking self-report
Pincherle (1971)	222 upper-class male quitters; followup ≥ 1 year after first visit	28% gained weight; 22% lost		Smoking self-report; limited population; incomplete report; no weights presented
Powell and McCann (1981)	29 women, 22 men, 5-day cessation project; 2- and 6-month followup	At 2 months, 54% gained weight, range 3–20 lb, mean 8.96 lb; all subjects mean 4.69 lb		Smoking self-report; no separate abstainer, smoker data; small sample size
Puddey et al. (1985)	66 cessation program volunteers, pair-matched by age, sex, body mass index; randomly assigned experimental, control groups; 2-week baseline, 6-week treatment, 6-week followup; thiocyanate, CO verification	14 quitters gained 3.97 lb; controls 0.44 lb		Small sample size
Rabkin (1984)	40 male, 67 female smokers, assigned to 3 cessation groups; followup 3 weeks post-completion; biochemical verification	67.3% gained weight, average 1.76 lb; skinfold increase 6.6 mm	No age, age at smoking start, rate, relative weight, anxiety correlation to male or female weight change	Small sample size; weight self-report

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Rantakallio and Hartikainen-Sorrin (1981)	12,068 pregnant women, n. Finland, 1966; 15% smokers (smoked after 2 months pregnant); nonsmoking controls matched for age, parity, place of residence, marital status	No smoking/nonsmoking pregnancy weight gain difference		Pregnant women only; smoking self-report; pregnancy weight gain data only
Rush (1974)	162 low-income urban pregnant women, no known medical problems, <140 lb preconception weight; had borne low birthweight infant; randomized controlled nutritional supplementation trial	Mean pregnancy weight gain lower for smokers (0.73 lb/wk) than nonsmokers (0.90 lb/wk)	Smoking rate: higher rate related to lower pregnancy weight gain	Pregnant women only; smoking self-report; pregnancy weight gain data only
Schoenenberger (1982)	4,421 male MRFIT volunteers, aged 35–57, good health but upper 10–15% coronary risk factor score; randomly assigned to intervention or control groups; followup 3 annual visits	With MRFIT intervention, significant body weight decrease in smokers (mean 4.6 lb), nonsmokers (mean 5.8 lb), reducers (mean 3.75 lb); quitters average weight change minimal (mean 0.55 lb)		Smoking self-report; confounded by risk factor reduction program participation; restricted population
Seltzer (1974)	794 adult white male veterans, average age 45; Normative Aging Study; screened for "high" health level, geographic stability; 214 screened at 5 years	At admission, ex-smokers 5.9 lb heavier than nonsmokers, 8.1 lb heavier than current smokers; at 5 years, quitters gained 8.0 lb, continuing smokers 3.5 lb		White veterans; smoking self-report

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Stamford et al. (1986)	13 sedentary women, 48-day successful quitters; 1-year followup	At 48 days, weight increased 4.85 lb; at 1 year, quitters increased 18.07 lb; 3 relapsers reduced weight to baseline levels; per hydrostatic weighing, gain was 96% fat		Small female sample; smoking self-report
Tuomilehto et al. (1985)	10,940 cardiovascular disease prevention program participants, aged 25–59, random sample, e. Finland; selectees with high blood pressure or hypertensive medicine assessed 5 years apart; smoking data from 2,264	Quitters body mass increased 2.31 lb/m^2; starting smokers decreased 1.46 lb/m^2		Smoking self-report; hypertensives
Vandenbroucke et al. (1984)	3,091 Netherlands civil servants, spouses (1,583 men, 1,508 women), aged 40–65, general health exam; 25-year followup	76.6% lean, 65.1% obese men smoked; 22.1% lean, 11.3% obese women smoked		Smoking self-report; restricted population

Gottenborg 1981; Khosla and Lowe 1971) documented increasing weight differences between smokers and nonsmokers with advancing age. Typically, aging smokers failed to gain as much weight as aging nonsmokers.

Three evaluations systematically compared males with females (Bjelke 1971; Kopczynski 1972; Sutherland et al. 1980). Two of the three (Bjelke 1971; Sutherland et al. 1980) reported the differences in body weight between smokers and nonsmokers to be greater in females than in males.

Longitudinal Evaluations of Smoking and Body Weight

Table 3 presents the results of 43 longitudinal evaluations of the effects of smoking on body weight. Consistent with the cross-sectional evaluations, the overwhelming majority (86 percent, 37 of 43) present evidence that smokers who quit smoking gain weight, that people who quit smoking gain more weight than nonsmokers, and that people who initiate smoking lose weight relative to nonsmokers. Of the six studies that did not find these relationships, three limited their examination to smoking and weight changes in pregnant women (Gormican, Valentine, Satter 1980; Haworth et al. 1980; Rantakallio and Hartikainen-Sorri 1981), two relied on participants making broad cardiovascular risk factor reduction efforts in subjects at high risk for cardiovascular disease (Hickey and Mulcahy 1973; Holme et al. 1985), and the remaining study supplied incomplete reports of the data (Kramer 1982). Of those studies on the effects of smoking cessation on weight, the length of followup ranged from 4 days to 7 years. According to these investigations, those who quit smoking gained an average of 6.16 lb (range: 1.76 to 18.07) during the year after cessation.

Daily cigarette consumption was the only moderator variable that received sufficient attention in this group of studies reaching specific conclusions. Seven of nine studies (78 percent) (Blitzer, Rimm, Giefer 1977; Bosse, Garvey, Costa 1980; Comstock and Stone 1972; Friedman and Siegelaub 1980; Hall, Ginsberg, Jones 1986; Howell 1971; Rush 1974) reported a positive relationship between cigarette consumption and weight change; that is, as pretest cigarette consumption increased, postcessation weight gains also increased. Two studies (Carney and Goldberg 1984; Rabkin 1984) did not find a relationship between cigarette consumption and postcessation weight gain.

In summary, there is substantial evidence of an inverse relationship between cigarette smoking and body weight. Of 71 studies reported since 1970, 62 (87 percent) collectively indicate that smokers weigh less than nonsmokers and that people who quit smoking gain weight. Older smokers, females, and those smoking approximately one pack of cigarettes/day may experience the

largest weight control effects of cigarette smoking. Smokers who smoke heavily tend to gain the most weight following smoking cessation. These generalizations are consistent with reviews based on other studies reported since 1880 (Grunberg 1986a). Not all smokers who quit smoking gain weight. Further, for ex-smokers who do gain weight, the amount of weight infrequently poses a serious health risk.

The Role of Nicotine

Animal studies indicate that nicotine administration results in weight loss or decreased weight gains and that cessation of nicotine results in body weight gains greater than those of controls (Bowen, Eury, Grunberg 1986; Grunberg 1982, 1985, 1986b; Grunberg, Bowen, Morse 1984; Grunberg, Bowen, Winders 1986; Grunberg, Winders, Popp 1987; McNair and Bryson 1983; Morgan and Ellison 1987; Schechter and Cook 1976; Wager-Srdar et al. 1984; Wellman et al. 1986). Most of these studies report inverse dose–response relationships between nicotine and body weight.

Recent research on nicotine polacrilex gum with humans corroborates the role of nicotine in body weight effects. Fagerström (1987) reported that subjects who quit smoking were much less likely to gain weight when they consistently used nicotine polacrilex gum. Abstinent subjects who regularly used the gum gained less than 2 lb at a 6-month followup. In contrast, the infrequent gum users gained almost 7 lb ($p<0.05$). Emont and Cummings (1987) reported a significant negative relationship ($r=-0.37$) between the number of pieces of nicotine polacrilex gum chewed per day and weight gain for heavy smokers (>26 cigarettes/day). No such relationship between gum use and weight gain was observed for lighter smokers (<26 cigarettes/day).

Mechanisms Underlying The Relationship Between Smoking and Body Weight

The inverse relationship between smoking and body weight may result from changes in energy intake, changes in energy expenditure, or both. Energy intake involves dietary intake. Energy expenditure is affected by behavioral factors (physical activity) and biological factors (e.g., metabolism). These potential mechanisms are examined below.

Dietary Intake

Several prospective investigations have evaluated dietary intake changes following smoking cessation in humans. Hatsukami and coworkers (1984) hospitalized 27 smokers for a 7-day period. After a 3-day baseline, 20 of the subjects were deprived of smoking for 4 days

while the remaining 7 served as a control group. During this 4-day period of abstinence, caloric intake increased significantly (from 1,397 to 1,651 kcal), which corresponded with a significant 1.76-lb increase in weight. In the most comprehensive study to date, Stamford and coworkers (1986) evaluated changes in dietary intake, physical activity, and resting metabolic rate in 13 sedentary females who quit smoking for a 48-day period. Following smoking cessation, mean daily caloric consumption increased by 227 kcal, which accounted for 69 percent of the variance in postcessation weight gain (4.85 lb). Robinson and York (1986) followed 11 smokers who quit for 7 days. Mean dietary intake significantly increased, but changes in resting metabolic rate were not observed. Dallosso and James (1984) followed 10 subjects for 6 weeks after they participated in a stop-smoking clinic. There was a 4-percent drop in resting metabolic rate in smokers who quit, a drop which was reliable when the data were expressed per kilogram of body weight. The average dietary intake increased by 6.5 percent, but this difference did not reach statistical significance.

Preliminary results of a recent investigation indicate gender differences in the effects of short-term smoking cessation on body weight and food intake (Klesges, Meyers et al. 1987). Female smokers who quit for 1 week increased their body weight and dietary intake significantly more than male smokers who quit. This sex difference is consistent with animal studies (Grunberg, Bowen, Winders 1986; Grunberg, Winders, Popp 1987). Given females' marked concerns regarding postcessation weight gain (Klesges and Klesges, in press), future studies will need to investigate possible gender differences in response to smoking cessation.

Several studies indicate that smokers may differ from nonsmokers in their intake of sweet-tasting simple carbohydrates (sugar) in particular. In a human laboratory study, Grunberg (1982) observed that smokers who were allowed to smoke ate less sweet food than smokers who were not allowed to smoke or nonsmokers. Smokers not allowed to smoke also reported the greatest preference for sweet foods. There were no differences among the three subject groups in consumption of other types of foods. Rodin (1987) conducted a prospective study in which food intake after smoking cessation was carefully evaluated. Smokers who gained weight after quitting smoking increased their sugar consumption in particular. Further, smokers increase consumption of sweet snack foods when they are deprived of cigarette smoking (Duffy and Hall, in press; Perlick 1977). On the other hand, two early investigations (Bennett, Doll, Howell 1970; Richardson 1972) found *higher* sugar consumption in smokers relative to nonsmokers. However, Richardson (1972) found that this difference was because of low-sugar intake in ex-smokers, while Bennett, Doll, and Howell (1970) argued that the differences

were largely due to increased added sugar intake because of hot beverage consumption. These two studies, which are inconsistent with the more recent studies, did not carefully measure all food intake and did not assess intentional changes in food intake to control body weight.

Several animal experiments have documented that food intake decreases during nicotine administration and increases after administration has ceased and that these changes in food intake correspond with changes in body weight (Bowen, Eury, Grunberg 1986; Grunberg 1982; Grunberg, Bowen, Winders 1986; Levin et al. 1987; McNair and Bryson 1983; Wager-Srdar et al. 1984). Consumption of sweet foods by male rats is particulary affected by nicotine (Grunberg 1982; Grunberg et al. 1985). However, nicotine also reduces bland food intake in female rats and has a greater effect on body weight of female rats than of male rats (Grunberg, Winders, Popp 1987; Grunberg, Bowen, Winders 1986; Levin et al. 1987).

Several investigations have reported that changes in body weight in animals also occur without observing decreases in food intake as the result of nicotine administration (Grunberg, Bowen, Morse 1984; Schechter and Cook 1976; Wellman et al. 1986). In one investigation, chronic exposure to cigarette smoke reduced body weight and food intake in rats; however, hamsters exposed to cigarette smoke decreased body weight without reducing food intake (Wager-Srdar et al. 1984). Several methodological factors complicate these results, including the use of different strains of animals, different routes of administration and dosages of nicotine, and whether acute versus chronic effects of nicotine were reported. However, these results indicate that more than the mechanism of food intake was involved in producing nicotine- and smoking-related weight changes.

Data from short-term human studies and several animal experiments indicate that dietary intake is involved with smoking-related energy imbalance. Based on self-reported cross-sectional surveys, it has been reported that smokers' dietary intake is the same as (Albanes et al. 1987; Fehily, Phillips, Yarnell 1984; Fisher and Gordon 1985; Matsuya 1982) or significantly higher than (Picone et al. 1982; Stamford et al. 1984a,b) that of nonsmokers while the smokers simultaneously maintained a lower body weight. Assuming that smokers are not consistently biased in their reports of dietary intake, it appears that either differences in physical activity or metabolic rate are maintaining the body weight differences between smokers and nonsmokers.

Physical Activity

The data available from cross-sectional investigations, short-term prospective studies, and animal investigations seem to indicate that changes in physical activity do not play a role in either differences in

body weight between smokers and nonsmokers or the weight gain associated with smoking cessation. Some cross-sectional investigations have found that smokers have *lower* levels of physical activity compared with nonsmokers (Kannas 1981). Others have not found differences in physical activity and physical fitness between smokers and nonsmokers (Gyntelberg and Meyer 1974; Stamford et al. 1984b; Stephens and Pederson 1983). A recent review (Blair, Jacobs, Powell 1985) that addressed the relationships among exercise, physical activity, and smoking concluded that smoking and physical activity are negatively associated; however, the relationship was extremely weak and variable.

Animal studies on the relationship between nicotine and physical activity have generally found that physical activity plays a small role or fails to correspond to decreases in weight during nicotine administration (Bowen, Eury, Grunberg 1986; Cronan, Conrad, Bryson 1985; Grunberg and Bowen 1985b). One study found that decreases in physical activity after cessation of nicotine appeared to contribute to postdrug body weight increases (Grunberg and Bowen 1985b), but this effect was quite small and occurred only in males.

A few prospective human investigations have evaluated physical activity changes following smoking cessation (Hatsukami et al. 1984; Hofstetter et al. 1986; Klesges, Brown et al. 1987; Rodin 1987; Stamford et al. 1986). These investigations found no changes in physical activity as a result of smoking cessation.

Metabolic Rate

Metabolic rate is an important consideration in energy imbalances associated with smoking cessation because approximately 75 percent of total energy expenditure is in the form of metabolism (Bernstein et al. 1983; Ravussin et al. 1982). Metabolism increases as the result of acute nicotine administration and immediate effects of smoking (Ghanem 1973; Ilebekk, Miller, Mjos 1975; Robinson and York 1986; Schievelbein et al. 1978; Wennmalm 1982). The major question, however, is whether these effects persist long enough to have a direct impact on body weight. Given that (1) smokers do not have higher levels of physical activity compared with nonsmokers (Blair, Jacobs, Powell 1985), (2) some studies report smokers' dietary intakes are the same as or higher than those of nonsmokers (Picone et al. 1982; Stamford et al. 1984a,b), and (3) smokers maintain lower body weights than nonsmokers, it is reasonable to postulate that changes in metabolism contribute to the relationship between smoking and body weight. Additionally, there are several reports in the literature on animals that have documented nicotine-induced reductions in body weight without a concomitant reduction in food intake (Grunberg, Bowen, Morse 1984; Schechter and Cook 1976; Wellman et al. 1986).

Direct evidence supporting a chronic metabolic mechanism that modulates the smoking/body weight relationship is beginning to emerge. Metabolic rate was chronically measured in a study of rat and hamster exposure to cigarette smoke (Wager-Srdar et al. 1984). Higher resting metabolic rates were observed on only one of the test days compared with the pretest in the rat investigation, while no significant differences were observed in the hamster study. Another recent investigation (Wellman et al. 1986) evaluated brown adipose tissue (BAT) thermogenesis at different levels of nicotine and caffeine injections. No differences in BAT thermogenesis were observed in response to either nicotine or caffeine. The group that received a combination of caffeine and nicotine showed a 63 percent increase in BAT thermogenesis.

The few studies that have evaluated metabolic rate changes in response to smoking cessation in humans have produced inconclusive results. Three investigations found metabolic changes after cessation in human smokers. An early report (Glauser et al. 1970) found decreases in oxygen consumption for seven male subjects who quit smoking for 1 month (neither food intake nor physical activity was monitored). A more recent investigation found a 4-percent drop in metabolic rate (reliable when data were expressed per kilogram of body weight) and no significant increase in dietary intake for 10 subjects who quit smoking for 6 weeks (Dallosso and James 1984). In the only study that used a respiration chamber, Hofstetter and others (1986) reported that total energy expenditure was 10 percent higher during a 24-hr period of smoking versus a 24-hr period of abstinence in eight smokers. No changes were observed in physical activity or mean basal (sleeping) metabolic rate (dietary intake was held constant). However, this difference in energy expenditure disappeared after 24 hr.

Three investigations did not find a change in metabolic rate as the result of smoking cessation. Burse and associates (1982, 1975) did not observe changes in resting metabolism in a sample of four smokers who quit for 3 weeks. This investigation did find reliable increases in desire for food, however. In another study, 11 smokers were studied after a 7-day period of smoking abstinence (Robinson and York 1986). Total energy expenditure following a meal did not change during the cessation period. Stamford and colleagues (1986) failed to find changes in oxygen consumption in 13 subjects who quit smoking for 48 days. This investigation did find marked dietary intake changes that accounted for 69 percent of the variance of postcessation weight gain.

There are several possible explanations for the inconsistency observed in the literature on metabolic rate. Different investigators have used different criteria (e.g., resting oxygen consumption, BAT thermogenesis) for operationalizing metabolism. It is possible that

previous dieting history (Brownell et al. 1986) and the use of nicotine polacrilex gum (Fagerström 1987) may directly impact the metabolic response to smoking cessation. It is not clear what the metabolic response to nicotine with added agents is likely to be. For example, one study found that while neither nicotine nor caffeine alone produced a change in BAT thermogenesis, the two combined increased thermogenesis by 63 percent (Wellman et al. 1986). This finding is particularly interesting given that smokers may be more likely to drink caffeinated beverages than nonsmokers (Blair et al. 1980). Finally, the available literature on human studies used very small subject groups, making it impossible to detect subtle but potentially meaningful changes in resting metabolic rate. The small sample sizes do not allow for an evaluation of variables that may potentially moderate the metabolic response to smoking cessation.

Summary of Mechanisms Literature

Changes in dietary intake appear to be involved in weight gains after cessation of smoking or cessation of nicotine administration. Physical activity plays little or no role in the relationship between smoking and body weight. The data on metabolic contributions to postcessation weight gain are suggestive, but further research is needed. Unfortunately, much of the relevant human research literature is characterized by small sample sizes, short followup evaluations, and inadequate evaluations of energy balance following smoking cessation. To date, only one investigation has comprehensively evaluated (i.e., simultaneous assessment of dietary intake, physical activity, and metabolic rate) energy balance changes as the result of smoking cessation. This was a sample of 13 sedentary females followed for 48 days (Stamford et al. 1986). Comprehensive, prospective evaluations of energy balance changes in response to smoking cessation are needed. Additionally, no study has evaluated possible long-term changes in dietary intake, physical activity, and metabolic rate as a result of smoking cessation. The longest followup period reported in the literature to date is 2 months (Dallosso and James 1984). Finally, evaluation of potential moderator variables of dietary intake, physical activity, and metabolic rate as the result of cessation is needed. Gender (Grunberg, Winders, Popp 1987; Klesges, Meyers et al. 1987), previous dieting history (Brownell et al. 1986; Hall, Ginsberg, Jones 1986), pretest levels of lipoprotein lipase (Carney and Goldberg 1984), and the use of nicotine polacrilex gum (Fagerström 1987) appear to be important variables influencing weight gain and need further investigation.

Does the Relationship Between Smoking and Weight Promote Either the Initiation or Maintenance of Smoking Behavior?

Some research attention has been given to body weight as a potential moderator of smoking initiation, maintenance, and cessation. Unfortunately, many investigations do not report weight-related issues (Borkon, Baird, Siff 1983; Eiser et al. 1985; Pederson and Lefcoe 1976; Perri, Richards, Schultheis 1977). The investigations that have evaluated these issues consistently report relationships between body weight and smoking initiation (Charlton 1984a) and maintenance (Klesges and Klesges, in press).

A survey of 16,000 school children (Charlton 1984a) in England found that the heaviest regular smokers were the most likely to agree that smoking controls weight (42.2 percent) compared with those students who never smoked (16.6 percent). Agreement increased with increased levels of smoking. More girls than boys agreed with this statement, and girls were also more likely to be regular smokers. Charlton (1984b) also reported that among the perceived effects of smoking, smokers viewed "calming the nerves" as the most popular reason (72 percent) followed by "smoking keeps your weight down" (39 percent).

Other investigations are consistent with the Charlton (1984a,b) report. In a recent study of 1,000 adolescents in Canada (Feldman, Hodgson, Corber 1985), significantly more girls than boys were concerned about becoming overweight (36 vs. 14 percent, $p<0.001$). In girls 18 years or older, 52.6 percent of smokers reported worrying about their weight, whereas only 31 percent of nonsmokers reported weight-related concerns ($p<0.05$). In a study of smoking intentions among 400 U.S. high school males, Tucker (1983) reported that overweight boys scored much higher on smoking intent than either normal weight or underweight boys ($p<0.005$). Another survey evaluated gender differences in a sample of 221 college cigarette-smoking intenders and nonintenders (Page 1983). Results indicated that females were much more likely to intend to smoke than males. Females were also more likely to believe that smoking maintains body weight, and smoking intenders were also more likely to believe that smoking controls weight. Finally, in a retrospective survey of more than 1,000 young adults (Klesges and Klesges, in press), overweight females reported that they were much more likely (20 percent) to start smoking for weight-related reasons compared with normal-weight females (2 percent). No differences between overweight versus normal-weight males (8 vs. 6 percent) were observed.

Several surveys on smoking maintenance have shown that individuals report that weight control is a powerful motivator to continue to smoke. Physicians who smoked were much more likely than those who had quit (46 vs. 22 percent) to believe that smoking cessation

increases appetite and weight (Fletcher and Doll 1969). Nurses who failed to quit smoking listed (in order) loss of determination, stress, and weight gain as the major reasons for failure (Knobf and Morra 1983). Beliefs regarding the weight-control effects of smoking and quitting differentiate smokers and nonsmokers (Hill and Gray 1984; Loken 1982; Shor et al. 1981). Females are particularly worried about postcessation weight gains (Klesges and Klesges, in press; Sorensen and Pechacek 1987). They are more likely to endorse smoking as an active weight-loss strategy (39 vs. 25 percent) and are more likely to report relapse for weight-related reasons (20 vs. 7 percent) (Klesges and Klesges, in press).

The research cited above is based on self-reports of the weight-control effects of smoking and, as such, could be viewed as an excuse for smoking. Two recent worksite-based investigations evaluated whether pretest concerns regarding smoking and weight-related issues prospectively predicted cessation. Maheu (1985) evaluated 49 subjects who either received a competition-based (n=32) or a no-competition condition (n=17). In the competition-based condition, participants were told that they would be rewarded if those at their worksite lost more weight than those at a neighboring worksite. At a 3-month followup, 78 percent of the subjects in the competition and 76 percent of the subjects in the no-competition condition were reportedly abstinent. Regression analysis at followup indicated that the best pretest predictors of smoking cessation (in order) were negative responses to the questions: (1) "Do you think smoking helps control your weight?"; (2) "Did one of your parents smoke when you were young?"; and (3) "If you have tried to quit before, did you suffer any withdrawal symptoms?" Klesges, Brown, and associates (1987) found that the best predictors of cessation at posttest were pretest cotinine levels and anticipated weight gain as the result of smoking cessation. The best predictors of cessation at followup were the number of coworkers who smoked followed by anticipated cessation-related weight gain.

A recent community survey evaluated predictors of current and former smoking status in a sample of 611 nonsmokers, ex-smokers, smokers who had tried to quit smoking, and smokers who had not attempted cessation (Klesges, Somes et al. 1987). The best predictors of smokers who had never attempted cessation versus those with a history of cessation efforts were a greater concern related to weight control, followed by knowledge of the health consequences of smoking. Smokers who had not attempted cessation were significantly more likely to cite weight-control issues compared with smokers who had made active attempts at smoking cessation. Collectively, these investigations indicate that weight-related concerns may not only predict successful smoking cessation, but also attempted smoking cessation.

Weight gain following smoking cessation as a predictor of smoking relapse has been evaluated in two recent investigations. Hall, Ginsberg, and Jones (1986) found a relationship between smoking status at a 1-year followup and weight gain at 6 months; greater weight gain during the first 6 months predicted continued abstinence. This finding was contrary to expectations. In another investigation, Gritz, Carr, and Marcus (in press) found that continuous abstainers had gained an average of 6.1 lb, relapsers had gained 2.7 lb and subsequently lost half the gain (1.3 lb), and never quitters had gained only 0.3 lb. While it was expected that postcessation weight gain would be predictive of relapse, one would expect that those who have been abstinent from cigarettes would have gained more weight than those whc either failed to quit or those who relapsed, because these latter groups have regained the weight reducing effects of smoking. Additional research will need to evaluate the impact of weight gain on relapsers at the point of relapse compared with the impact on abstainers at a comparable point in time. Further, it is clear that actual weight may have little relationship with subjects' perceptions of their weight status. For example, overweight males consistently view themselves as normal weight, while underweight and normal-weight females consistently view themselves as overweight (Klesges 1983). Very small weight gains in some subjects (e.g., normal-weight females) may be much more predictive of relapse than very large weight fluctuations in others (e.g., overweight males) (Klesges 1983). Future research should evaluate potential variables (e.g., gender, obesity) that may moderate the relationship between weight gain and smoking relapse.

In summary, weight-related issues may be important in the maintenance and cessation of smoking. Weight-reducing effects of smoking may encourage smoking initiation by some people, but the data on this point are currently unconvincing. Future research should focus on who (e.g., males versus females, those with a history of chronic dieting) is most at risk to smoke because of weight-related concerns. In particular, prospective studies on weight-related issues as they predict smoking initiation, cessation, and relapse are needed.

Implications for Tobacco Use

Cigarette smokers weigh less than comparably aged nonsmokers, and many smokers who quit smoking gain weight. This inverse relationship between smoking and body weight is well established, and the role of food intake and energy expenditure as mechanisms for this relationship is currently receiving research attention. The postsmoking weight gains are frequently undesired by the ex-smoker. People are quite aware of the relationship between smoking and body weight, and this relationship may encourage some people to initiate smoking and to keep smoking. However, other people may

modify food intake and avoid weight gains after cessation of smoking.

Summary and Conclusions

1. After smoking cigarettes or receiving nicotine, smokers perform better on some cognitive tasks (including sustained attention and selective attention) than they do when deprived of cigarettes or nicotine. However, smoking and nicotine do not improve general learning.
2. Stress increases cigarette consumption among smokers. Further, stress has been identified as a risk factor for initiation of smoking in adolescence.
3. In general, cigarette smokers weigh less (approximately 7 lb less on average) than nonsmokers. Many smokers who quit smoking gain weight.
4. Food intake and probably metabolic factors are involved in the inverse relationship between smoking and body weight. There is evidence that nicotine plays an important role in the relationship between smoking and body weight.

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