more weight to large versus small samples. Table 2 provides more detailed information (e.g., 95-percent confidence intervals for weight gain and relative risk) regarding each of these investigations.

As indicated in Tables 1 and 2, the average sample size of these investigations was 1,348 (range=28–9,539). The followup period ranged from 1 month to 5 years, with a median followup period of 2 years. Consistent with previous reviews of the smoking and body weight literature (Klesges et al. 1989; US DHHS 1988a), the adjusted average weight gain among smokers who quit was approximately 5 pounds (mean=4.6; range=1.6–11.2 pounds). The weight gain among smokers who quit was considerably greater than the adjusted average gain of 0.8 pounds observed among subjects who continued to smoke (range=0 to +3.5 pounds). Thus, although variability of weight gain is quite marked (Tables 1 and 2), smoking cessation produces approximately a 4-pound greater weight gain than that associated with continued smoking.

A commonly reported, but erroneous, estimate regarding postcessation weight gain is that one-third of smokers gain weight after smoking cessation, one-third maintain body weight, and one-third lose weight after cessation (US DHEW 1977). In the five investigations providing detailed information regarding changes in body weight, the actual percentage of quitters gaining weight appears to be much greater than previously estimated. Considering the results of all five studies and adjusting for sample size, 79 percent of those who quit smoking experienced a weight gain (range=58–87 percent). Over the same followup period, an adjusted average of 56 percent of continuing smokers experienced an increase in body weight (range=33–62 percent) and, as presented above, the average amount of weight gain was less among continuing smokers.

Data allowing computation of a relative risk estimate of weight gain after smoking cessation were available from five investigations. This relative risk estimate compares the likelihood of weight gain in quitters versus continuing smokers. That is, a higher relative risk ratio indicates that the percentage of quitters who gained weight was higher compared with that of corresponding continuing smokers. Overall, the risk of weight gain after cessation was 45 percent greater for quitters (mean=1.45, range=1.31–1.75) than for continuing smokers. This increased risk of weight gain was consistent across differing followup periods, appearing as early as 6 weeks (Rodin 1987; relative risk (RR)=1.75) and lasting up to 6 years after smoking cessation (Noppa and Bengtsson 1980; RR=1.31). Additionally, one investigation found the relative risk of gaining more than 2 pounds after smoking cessation to be 1.38 (Bossé, Garvey, Costa 1980). In another investigation, the risk of gaining more than 10 pounds was 88 percent higher for quitters than for continuing smokers (RR=1.88) (Friedman and Siegelaub 1980).

Although the risk of gaining more than 10 pounds appears to be almost 90 percent greater among quitters than continuing smokers (Friedman and Siegelaub 1980), actual occurrence of 10-pound weight gains was relatively low (20.3 vs. 10.8 percent among quitters and continuing smokers, respectively). Friedman and Siegelaub (1980), with a large sample of quitters (N=2,738) and continuing smokers (N=6,801), presented the percentages of those gaining 20 pounds or more over a median 18-month followup. Among males, 3.7 percent of those who quit smoking gained more than 20 pounds compared with 0.9 percent of those who continued to smoke. Among females, 3.1

TABLE 2.—Details of prospective studies in which change in weight relative to continuing smokers was reported

Reference	Sample	Quit period	Average gain ±SD (lb)	95% CI for average gain (lb)	Results
Bossé, Garvey, Costa (1980)	705 males aged 24–81[a]	≤5 yr	Quitters: 6.35±12.15	(4.80, 7.90)	Gained ≥2 lb: Quit 64.1% (152); Continued 46.4% (217). Gained ≤2 lb or lost: 35.9% (85); 53.6% (251)
			Continuing smokers: 2.01±9.61	(1.14, 2.88)	Relative risk of gaining more than 2 lb=1.38; 95% CI (1.21, 1.58)
Cambien et al. (1981)	475 male Parisians aged 25–35 in control condition of randomized trial	≤2 yr	Quitters: 7.5		
			Continuing smokers and nonsmokers: 2.2		
Coates and Li (1983)	335 asbestos-exposed males, average age 42	1 yr	Quitters: 5.15±7.53	(1.06, 9.24)	Gained: Quit 76.9% (10); Continued 46.3% (149). No change or lost: 23.1% (3); 53.7% (173)
			Continuing smokers: 0.35±7.53	(−0.47, 1.17)	Relative risk of gaining any weight=1.66; 95% CI (1.21, 2.29)
					Quitters: Lost ≥5 lb 15.4% (2); Lost 1–4 lb 7.7% (1); Gained 0–4 lb 30.8% (4); Gained >5 lb 46.1% (6). Continuing smokers: 19.9% (64); 33.8% (109); 23.0% (74); 23.3% (75)

TABLE 2.—Continued

Reference	Sample	Quit period	Average gain ±SD (lb)	95% CI for average gain (lb)	Results
Comstock and Stone (1972)	290 males, aged 40–59	≤5 yr	Quitters: 11.2 Continuing smokers: 2.4		Gained: Quit 87.0% (40); Continued 60.7% (148) No change or lost: 13.0% (6); 39.3% (96) Relative risk of gaining any weight=1.43 95% CI (1.23–1.67)
Friedman and Siegelaub (1980)	9,539 participants aged 20–70 in health screening in California	18 mo (median)	Quitters: 3.18 Males: 4.1 Females: 3.5 Continuing smokers: 0.9 Males: 0.9 Females: 0.9		Gained >10 lb: Quit 20.3% (557); Continued 10.8% (734) Gained <10 lb or lost: 79.7% (2,181); 89.2% (6,067) Relative risk of gaining >10 lb=1.88 95% CI (1.70–2.08)

Males		
Quitters		Continuing smokers
Gained <10 lb	77.6% (930)	88.6% (2550)
Gained >10 lb	18.7% (224)	10.5% (302)
Gained >20 lb	3.7% (44)	0.9% (26)

Females		
Quitters		Continuing smokers
Gained <10 lb	81.2% (1251)	89.6% (3517)
Gained >10 lb	15.7% (242)	8.8% (344)
Gained >20 lb	3.1% (47)	1.6% (62)

TABLE 2.—Continued

Reference	Sample	Quit period	Average gain ±SD (lb)	95% CI for average gain (lb)	Results
Gritz, Carr, Marcus (1988)	554 self-quitters, average age 41.4 yr	1 yr	Quitters: 6.1 Continuing smokers: 0.3		
Hickey and Mulcahy (1973)	88 male smokers surviving first MI, average age 50.2 yr	2 yr	Quitters: 1.6 Continuing smokers: 0.9		
Kramer (1982)	134 participants from a commercial cessation program	≥ 1 yr			Gained / No change or lost Quit 78.0% (46) / 22.0% (13) Continued 56.0% (42) / 44.0% (33) Relative risk of gaining weight=1.39 95% CI (1.09, 1.77)
Lund-Larsen and Tretli (1982)	6,580 Norwegians from CV screening, aged 20–49	≤ 3 yr	Quitters: 6.94 Males: 7.94 Females: 5.95 Continuing smokers: 0.44 Males: 0.88 Females: 0		

TABLE 2.—Continued

Reference	Sample	Quit period	Average gain ±SD (lb)	95% CI for average gain (lb)	Results
Noppa and Bengtsson (1980)	526 Swedish women, aged 38–60[a]	≤ 6 yr	Quitters: 7.7±10.8	(5.2–10.2)	Gained: Quit 80.6% (58); Continued 61.7% (280). No change or lost: 19.4% (14); 38.3% (174)
			Continuing smokers: 2.4±11.5	(1.3–3.5)	Relative risk of gaining any weight=1.31, 95% CI (1.14–1.49)
					Quitters / Continuing smokers: Lost ≥22 lb: 0.0% (0) / 2.4% (11); Lost 11–22 lb: 4.2% (3) / 5.5% (25); Lost 0–11 lb: 15.3% (11) / 30.4% (138); Gained 0–11 lb: 45.8% (33) / 44.5% (202); Gained 11–22 lb: 22.2% (16) / 13.7% (62); Gained ≥ 22 lb: 12.5% (9) / 3.5% (16)
Puddey et al. (1985)	14 quitters and 14 matched smoking controls, aged 24–63	6 wk	Quitters: 3.97		
			Continuing smokers: 0.44		

TABLE 2.—Continued

Reference	Sample	Quit period	Average gain ±SD (lb)	95% CI for average gain (lb)	Results
Rabkin (1984b)	107 participants of cessation program, average age 40	≤3 mo	Quitters: 4.4±3.9 Males: 5.9±4.1 Females: 3.3±3.0 Continuing smokers: 0.7±3.7 Males: 2.2±3.4 Females: –0.4±3.0	(3.1–5.7) (3.8–8.1) (2.0–4.6) (–0.2–1.5) (0.9–3.5) (–1.3–0.4)	
Rodin (1987)	42 participants of smoking cessation program, average age 44	8 wk	Quitters: 3.18 Continuing smokers: 0.30		Gained / No change or lost Quit 58.3% (14) / 41.7% (10) Continued 33.3% (6) / 66.7% (12) Relative risk of gaining any weight=1.75 95% CI (0.84–3.65)
Seltzer (1974)	318 white male veterans from Boston, aged 25–64	≤ 5 yr	Quitters: 7.9 Continuing smokers: 3.5		

TABLE 2.—Continued

Reference	Sample	Quit period	Average gain ±SD (lb)	95% CI for average gain (lb)	Results
Tuomilehto et al. (1986)	496 participants in CV prevention trial in Finland, aged 25–59	≤ 5 yr	Quitters: Males: 8.16 Females: -0.37 Continuing smokers: Males: 2.27 Females: –2.56		

NOTE: SD=standard deviation; CI=confidence interval; MI=myocardial infarction; CV=cardiovascular.

[a]Younger subjects gained more weight.

percent of those who quit smoking gained more than 20 pounds compared with 1.6 percent of those who continued to smoke.

In summary, while approximately four-fifths of smokers who quit will gain weight after cessation, average weight gain is approximately 4 pounds greater than that expected among continuing smokers. The risk of weight gain after cessation is 45 percent greater than the risk associated with continued smoking, although individual weight gains of 20 pounds or more are rare.

Although weight gain is common after cessation, little is known concerning the types of individuals at risk for substantial increases in body weight. Researchers have concluded that women, moderate smokers, and older smokers have the greatest weight control effect from smoking (US DHHS 1988a), although the tremendous variability in body weight changes after cessation has yet to be explained. That is, while the average weight gain after smoking cessation is approximately 5 pounds, individual responses range from weight loss to a weight gain exceeding 20 pounds. Studies are needed that focus carefully on individuals at risk of excessive weight gain after smoking cessation and the differences between these individuals and those who do not gain weight.

Additionally, investigators hypothesize that the relationship between smoking and body weight is attenuated by other health behaviors (Marti et al. 1989). Although the effects of smoking to reduce body weight are acknowledged, individuals who smoke are more likely than nonsmokers to have unhealthy lifestyles associated with increased body weight (e.g., lower levels of physical activity and higher dietary intakes) (Klesges, Eck et al. 1990; Chapter 11).

CAUSES OF POSTCESSATION WEIGHT GAIN

Cross-sectional and longitudinal studies clearly indicate the inverse relationship between smoking and body weight in humans and between nicotine and body weight in animals (Grunberg 1986; Klesges et al. 1989; US DHHS 1988a; Winders and Grunberg 1989). However, no study has included a simultaneous evaluation of the long-term changes in all of the variables that may account for this relationship, including food intake, physical activity, and energy expenditure. Of the currently published investigations, the longest followup period evaluating all three aspects of the energy balance equation has been 8 weeks (Stamford et al. 1986). A recent study evaluated food intake and physical activity changes over a 26-week followup but did not include metabolic measures (Hall et al. 1989). Short-term evaluations do not allow for an adequate determination of predictors of weight gain. This review focuses on those studies that have directly evaluated either food intake, physical activity, and/or metabolic rate as a function of smoking cessation, nicotine administration, or nicotine deprivation. The available data on changes in the energy balance equation that result from smoking cessation are summarized below.

Food Intake

Most short-term evaluations (e.g., 3 days or less) found that food intake, particularly the consumption of sweet foods and simple carbohydrates, increases after smoking cessation. For example in a 1-day experiment, Grunberg (1982a) reported that smokers who were allowed to smoke ate fewer sweet foods, but consumed similar amounts of non-sweet foods, compared with nonsmokers and smokers not allowed to smoke. This between-subjects laboratory study was short term and did not measure body weight changes. In another short-term study, Hatsukami and colleagues (1984) hospitalized 27 smokers for 7 days. 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 abstinence, caloric intake increased significantly in the abstinence group and was accompanied by a 1.76-pound increase in weight compared with baseline. Recently, Duffy and Hall (1988) assessed smokers who differed in degree of eating disinhibition, defined as eating that occurs in situations in which self-control behaviors are disrupted (e.g., binge eating). Smokers who were allowed to smoke before eating ice cream did not show food consumption differences as a function of level of disinhibition. However, results for smokers who had abstained from smoking for 24 hours showed a different pattern. Abstaining smokers who scored high on eating disinhibition ate more than three times (273.6 g) as much ice cream as those who scored low (86.4 g) on eating disinhibition. The results from this investigation indicate that dietary changes following smoking cessation may vary as a function of dieting history, use of cigarettes to curb appetite, and other weight history variables.

Some prospective investigations have qualitatively asked participants who quit smoking if they believed that their dietary intake had changed. These studies also reported that food intake increases after cessation. For example, Manley and Boland (1983) examined the side effects experienced by 94 subjects quitting smoking and whether these side effects varied as a function of relapse. On a withdrawal rating system, those who quit smoking rated themselves as furthest from "optimal" at followup on general appetite and overeating. On a separate rating scale, abstainers also gave higher ratings than relapsers at followup on "eating more." In a study of 53 self-quitters, Black and coworkers (1988) found that of those reporting that they ate more, average weight gain was 6.9 pounds. In contrast, of those reporting that they ate the same or less, average weight gain was 1.4 pounds.

Unfortunately, there are few prospective human investigations that have attempted to quantify carefully food intake changes over time among subjects after quitting smoking. These studies generally indicate that food intake increases after cessation; however, results vary greatly across investigations. Of eight studies to date, two reported clear increases in food consumption after cessation (Leischow and Stitzer 1989; Stamford et al. 1986), four provided qualified support for increased food consumption after cessation (Hall et al. 1989; Klesges et al., in press; Perkins, Epstein, Pastor 1990; Rodin 1987), and two reported no changes in food intake after cessation (Dallosso and James 1984; DiLorenzo et al. 1988).

In what may be the most comprehensive evaluation to date of change in energy balance, Stamford and colleagues (1986) analyzed changes in food intake, physical

activity, and resting metabolic rate in 13 sedentary females who quit smoking for 48 days. Mean daily food intake increased by 227 kcal and explained 69 percent of the variance in changes in weight (4.85 pounds). No changes in physical activity or resting metabolic rate were observed.

To evaluate dietary changes after cessation, Leischow and Stitzer (1989) assigned subjects, in an inpatient setting, to either smoke-ad-libidum (N=6) or quit-smoking (N=9) conditions for at least 14 days after a 4-day baseline period. Results revealed a significant difference in weight gain ($p<0.05$) between smokers and those who quit smoking (2.0 vs. 4.7 pounds, respectively). The weight gain in those who quit smoking was associated with a significant increase in food intake over time compared with continuing smokers.

Four investigations have provided qualified support for dietary changes after cessation. Perkins, Epstein, and Pastor (1990) evaluated caloric intake, resting energy expenditure (REE), and physical activity in seven female smokers for 3 weeks, which included normal smoking (week 1), smoking cessation (week 2), and resumption of smoking (week 3). Total caloric intake did not increase during the week of cessation. However, once smokers resumed smoking during week 3, caloric intake decreased significantly. Caloric intake from alcohol, however, rose from 219 kcal per day in the first week to 432 kcal per day during the week of abstinence. When subjects resumed smoking during the third week, alcohol intake dropped to 129 kcal per day. During the cessation week, REE did not decrease compared with baseline. However, a significant increase in REE was observed when subjects resumed smoking compared with the week of abstinence ($p<0.001$). No changes in physical activity were observed.

Rodin (1987) evaluated changes in food intake and physical activity in 24 subjects who quit smoking and 18 smokers who failed to quit smoking. Subjects who quit smoking gained an average of 3.2 pounds over the 8-week study. Consistent with the literature concerning animals as subjects and some studies using humans (Grunberg 1986; Winders and Grunberg 1989), smokers who gained weight after stopping smoking increased their carbohydrate consumption, particularly sugar. This increase was accompanied by decreased protein consumption. However, these subjects did not increase their total food intake nor did they decrease their levels of physical activity. Levels of physical activity generally increased.

Hall and coworkers (1989) assessed changes in food intake and physical activity among 95 subjects who enrolled in a stop-smoking program. In contrast to all other investigations reviewed in this Section, Hall and coworkers (1989) evaluated long-term changes in food intake and physical activity (for a 6-month followup). Caloric intake increased significantly in one group and marginally in another group during the first 8 weeks of abstinence. Both sugar and total fat increases were noted in the group that significantly increased energy intake. Total dietary intake increased approximately 200 kcal per day over the 8-week period. In assessing 6-month changes, Hall and coworkers (1989) reported a gender difference in caloric intake with time. Among men who quit, mean daily caloric intake decreased by almost 1,000 kcal from a mean of 3,014 kcal during week 1 to 2,035 kcal at week 26. Among women, caloric intake remained stable (mean=1,841 kcal at week 1; mean=1,867 kcal at week 26). However, weight continued to increase for both groups. From the 12-week to the 6-month followup, men

increased their weight 3.56 pounds (8.65 pounds total), and women increased their weight by 4.53 pounds (10.34 pounds total). No changes in physical activity were observed. Weight continued to increase despite no changes from baseline in dietary intake and physical activity in female ex-smokers and despite decreases in dietary intake and no physical activity changes in male ex-smokers.

Klesges and coworkers (in press) reported gender differences in response to smoking cessation. In this study, the food intake and physical activity of 68 smokers and nonsmokers were evaluated during a 2-week period. At the end of the first week, the smokers were paid to quit smoking, and 36 percent were successful at remaining abstinent for the entire week (confirmed by carbon monoxide (CO) readings). Nonsmokers continued to monitor their food intake and physical activity. At the end of the second week, subjects were allowed to return to smoking. In this investigation, female smokers who quit smoking increased their body weight in comparison with nonsmokers. Smokers who quit increased their consumption of mono- and polyunsaturated fats and decreased their intake of fiber. In contrast, males who quit smoking did not change either their weight or dietary intake compared with males in the other groups. No changes in physical activity were detected in any of the groups.

Dallosso and James (1984) reported on 10 subjects who quit smoking and were observed for 6 weeks after they participated in a stop-smoking clinic. Resting metabolic rate dropped by 4 percent in smokers who quit, a drop which was significant only when the data were expressed as per kilogram of body weight. The average food intake increased by 6.5 percent, but this difference was not statistically significant.

DiLorenzo and colleagues (1988) evaluated changes in body weight and caloric consumption in 16 subjects who quit smoking for 5 weeks compared with 11 subjects who continued to smoke and 16 nonsmokers studied over the same time period. Subjects who quit smoking gained an average of 5 pounds over the 5 weeks; the smoking and nonsmoking control groups did not change body weight significantly ($p<0.0001$). This weight gain was not associated with changes in dietary intake.

Physical Activity

In contrast to the findings on dietary intake and smoking cessation, the available data indicate that change in physical activity does not play a role in either differences in body weight between smokers and nonsmokers or the weight gain associated with smoking cessation. The small number of prospective investigations has generally reported unchanged physical activity after smoking cessation (Hall et al. 1989; Hatsukami et al. 1984; Klesges et al., in press; Perkins, Epstein, Pastor 1990; Stamford et al. 1986), and those that found a change in activity reported an increase in physical activity after smoking cessation (Leischow and Stitzer 1989; Rodin 1987). The literature consistently indicates that reduced physical activity after cessation cannot account for postcessation weight gain.

Energy Expenditure

An important and often overlooked variable in energy imbalance leading to weight gain is REE. Approximately 75 percent of total energy expenditure is in the form of metabolism (Ravussin et al. 1982). Ample indirect evidence supports the hypothesis of increased energy expenditure in smokers. That is, given that smokers do not have higher levels of physical activity compared with nonsmokers, the only known mechanism remaining to explain the energy imbalance is some aspect of metabolism (Blair, Jacobs, Powell 1985); smokers' dietary intakes may be the same or higher than those of nonsmokers (Picone et al. 1982; Stamford, Matter, Fell, Sady, Cresanta et al. 1984; Stamford, Matter, Fell, Sady, Papanek et al. 1984); smokers maintain lower body weights than do nonsmokers (Klesges et al. 1989; US DHHS 1988a); and weight gain has been reported in individuals quitting smoking without any dietary and physical activity changes (DiLorenzo et al. 1988; Hall et al. 1989). Additionally, several reports document nicotine-induced reductions in body weight in laboratory animals without a concomitant reduction in food intake (Grunberg, Bowen, Morse 1984; Schechter and Cook 1976; Wellman et al. 1986). However, those few studies that have evaluated metabolic changes in response to smoking cessation among humans have produced inconclusive and equivocal results.

Eight studies have reported either acute changes in REE following smoking or nicotine administration or have reported decreases in REE after smoking cessation. An early study (Glauser et al. 1970) reported decreases in oxygen consumption for seven male subjects who quit smoking for 1 month. Food intake and physical activity were not monitored. Reanalysis of these data (Klesges et al. 1989) revealed that the changes in metabolic rate reported by Glauser and coworkers (1970) were significant only with improper methods of statistical analysis. In the only study that utilized an indirect calorimetry respiration chamber, Hofstetter and coworkers (1986) reported a 10-percent difference in total energy expenditure during a 24-hour period of smoking compared with a 24-hour period of abstinence among eight smokers. However, this difference in energy expenditure disappeared after 24 hours. No changes were observed in mean basal (sleeping) metabolic rate. Diet was held constant.

Perkins and colleagues have conducted a series of studies evaluating the effects of nicotine, in the form of nicotine nasal spray, on changes in REE. In a study of nicotine administration in 18 male smokers, Perkins and colleagues (Perkins, Epstein, Stiller, Marks et al. 1989) reported REE changes that were 6 percent above baseline after nicotine administration, which was significantly greater than the 3-percent increase after placebo administration. Another investigation (Perkins et al. 1989a) sought to determine if nicotine-induced increases in metabolic rate observed at rest were also present during physical activity. Ten male smokers were administered nicotine and were then compared with 10 male smokers who were administered placebo. Metabolic rates increased both at rest and during light exercise. Although the percent change in REE due to nicotine was equivalent both at rest and during activity, the excess energy expenditure (in kilocalories) attributable to nicotine was more than twice as great during exercise. A third study using nicotine nasal spray assessed the combined effects of nicotine and consumption of a meal on REE (Perkins, Epstein, Stiller, Sexton et al.

1989). Eight male smokers were assessed using a repeated measures design. These individuals were given a caloric load (vs. water) and nicotine (vs. placebo). Both the caloric load and nicotine increased REE significantly. However, no interaction between these factors emerged, and the effects were slightly less than additive when combined. Nicotine alone increased REE by 4.95 kcal per hour, food alone increased metabolic rate by 14.30 kcal per hour, but nicotine plus food increased metabolic rate by 17.00 kcal per hour. Finally, in a study of the effects of changes in energy balance as a function of smoking cessation, Perkins, Epstein, and Pastor (1990) evaluated REE in seven female smokers across 3 weeks: normal smoking (week 1), smoking cessation (week 2), and resumption of smoking (week 3). REE did not drop during the week of abstinence compared with baseline. However, a significant increase in REE was observed when subjects resumed smoking compared with the week that they were abstinent.

The effects of smoking and coffee consumption on REE were recently evaluated by Klesges, Brown, and colleagues (1990). Of 45 regular cigarette smokers and coffee drinkers, 15 were randomly assigned to smoke 2 cigarettes, 15 were assigned to drink two standardized cups of coffee, and 15 were assigned to smoke cigarettes and drink coffee. All three groups had acute increases in REE with a similar pattern of response in each group.

In the largest study to date of all-day changes in metabolic rate, Klesges, Coday, and coworkers (1990) evaluated changes in REE among 39 individuals over a 10-hour period using multiple assessments of REE. Of the 30 smokers, 20 were assigned randomly to continuous, regular smoking and 10 were assigned to a no-smoking group. A nonsmoking control group of nine subjects was also evaluated over the same time period. The increase in REE among nonsmokers was not significant. In marked contrast, smokers who did not smoke decreased REE over the course of the day. Additionally, there were two distinct patterns of results among smokers who smoked over time. Of the 20 smokers, 14 (70 percent) markedly increased their REE over time, but 6 smokers (30 percent) decreased REE over time (similar to the pattern of smokers who did not smoke). Closer inspection of the minute-by-minute metabolic changes of those subjects who increased metabolic rate indicated an acute metabolic increase followed by a return to baseline early in the day, or an acute metabolic increase followed by a reduction, but to a level higher than baseline later in the day. In contrast, subjects who had a mean decrease in REE also had an acute metabolic increase followed by a drop below baseline early in the day, or an acute metabolic increase followed by a return to baseline later in the day. Subjects who responded with decreases in REE smoked more (as measured by expired CO) than those who responded with a cumulative increase in energy expenditure. These results are consistent with recent observations of a U-shaped relationship between daily cigarette consumption and body weight, with moderate smokers weighing less than nonsmokers but heavy smokers approximating the body weights of nonsmokers (Albanes et al. 1987).

Four studies found no relationship between smoking and metabolic rate. Burse and coworkers (1982) did not observe chronic changes in resting metabolism in a sample of three smokers who quit for 3 weeks. However, the small sample size in this investigation limits interpretation of the results. Although Robinson and York (1986)

reported an elevated metabolic response to food intake (i.e., thermic effect of food), chronic REE did not change as a function of smoking and total energy expenditure after a meal during the cessation period. Stamford and colleagues (1986) did not find changes in oxygen consumption in 13 subjects who quit smoking for 48 days. These investigators did find marked food intake changes that accounted for 69 percent of the variance of postcessation weight gain. In a study of the chronic effects of smoking status on REE, Perkins and coworkers (1989b) assessed 20 male smokers and 10 male nonsmokers after overnight abstinence from food and caffeine in both groups and after overnight abstinence from smoking in the smoking group. No differences in REE were observed.

Two recent studies evaluating the acute effects of cigarette smoking on REE have provided equivocal findings. In a sample of five occasional and five regular smokers (Warwick, Chapple, Thomson 1987), REE did not increase after smoking, even during the first 15 to 30 minutes after smoking. Additionally, the thermic effect of food was slightly, but not significantly, lower with smoking than without smoking. Dallosso and James (1984) evaluated short- and long-term metabolic changes associated with smoking. The thermogenic (metabolic) response for 1 hour after smoking 1 cigarette was not significant, although an acute increase was observed during the first 30 minutes. However, variability of responses was marked, ranging from a 4.5-percent decrease in metabolic rate to a 9.0-percent increase. No consistent long-term changes in metabolic rate were observed. Rather, the metabolic rate of four smokers clearly decreased after cessation; the rate stayed the same in two smokers and increased in two others.

The literature generally indicates that both dietary and metabolic changes are responsible for weight gain after smoking cessation, but these changes probably occur through complex mechanisms. Physical activity does not appear to be related to postcessation weight gain. Although the pattern generally indicates that both dietary and metabolic factors are involved, there is inconsistency both within and between studies indicating tremendous individual differences in subjects' dietary and metabolic changes after smoking cessation.

Investigators need to try to determine carefully the potential moderator variables of dietary and metabolic changes after smoking cessation. Factors such as gender, age, race, weight history, and concerns about postcessation weight gain may all play a role in predicting dietary changes after cessation. Some individuals, for example, may respond to smoking cessation by dramatically increasing their dietary intake (Duffy and Hall 1988), whereas others may impose dietary restrictions in an attempt to avoid postcessation weight gain (Klesges et al., in press).

There also appears to be tremendous individual variation in the metabolic response to smoking and smoking cessation. Overall, evaluations of short-term, acute responses to smoking generally report increases in metabolic rate as a function of nicotine administration and smoking (Hofstetter et al. 1986; Perkins et al. 1989a; Klesges, Brown et al. 1990), although long-term (overnight or longer) studies generally do not indicate changes in metabolic rate as a function of smoking cessation (Stamford et al. 1986). However, some investigators have reported that the acute effects of smoking have not produced a change in REE (Warwick, Chapple, Thomson 1987).

Research needs to focus on a number of potential moderators of smoking and metabolic rate. Levels of plasma nicotine vary greatly even for the same level of cigarette consumption and for the same nicotine content of cigarettes (US DHHS 1988a). The relationship between nicotine, as well as other constituents of tobacco smoke, and metabolic rate needs to be evaluated carefully. It is also possible that heavier, chronic smokers may habituate to the effects of nicotine over time (US DHHS 1988a) and their metabolic responses may become blunted (Klesges, Coday et al. 1990). Other important moderators, such as years smoked, gender, and relative weight, should also be carefully evaluated in future investigations.

RELATIONSHIP BETWEEN OVERWEIGHT AND ADVERSE MEDICAL AND PSYCHOSOCIAL OUTCOMES

Obesity refers to excess body fat, whereas overweight refers to excess body weight relative to height compared with gender-specific norms (Powers 1980). Obesity and overweight are highly correlated across the population, although some individuals are overweight but not obese (e.g., bodybuilders), and others are obese but not overweight (e.g., a normal weight "couch potato") (Grunberg 1982b). In the context of this Chapter, the relevant data are those that are related to health risks. The most commonly used methods to measure or estimate body fat in studies of health consequences of body size are measures of height and weight in comparison with gender-specific norms (which actually determine overweight) and measurement of subcutaneous fat by skinfold thickness at one or more sites (which determines obesity). Therefore, the data cited in this Chapter are sometimes based on estimates of obesity and sometimes based on estimates of overweight; both terms appear in the text. Normative values for these anthropometric measures have generally been derived in one of two ways: either by averaging the values found in populations of healthy persons or by tabulating values reported to be associated with greatest longevity in population-based studies. Inclusion of data based on these various standard measures provides the most complete information available. Although the volume of research related to obesity and health risk precludes comprehensive review here, a summary of this literature is a useful starting point for examining the health risks of weight gain following smoking cessation.

Large amounts of epidemiologic and clinical data clearly indicate a positive association between excess body weight and medical risk. Cross-sectional, longitudinal, ecologic, and case–control studies indicate that there is a graded relationship between weight and various diseases and disease risk factors. Positive associations have been reported between body weight and glucose intolerance and type II diabetes (Kannell, Gordon, Castelli 1979; Rimm et al. 1972; West and Kalbfleisch 1971; Negri et al. 1988; Hadden and Harris 1987); elevated blood pressure and hypertension (MacMahon et al. 1987; Chiang, Perlman, Epstein 1969; MacMahon et al. 1984; Blackburn and Prineas 1983; Pan et al. 1986); elevated total blood cholesterol and lowered high-density lipoprotein cholesterol (HDL-C) (Jooste et al. 1988; Garrison et al. 1980; Nanas et al. 1987); gout (Larsson, Bjorntorp, Tibblin 1981); kidney stones (Larsson, Bjorntorp, Tibblin 1981); gall bladder disease (Rimm et al. 1972); cardiovascular disease (CVD) (Rabkin, Mathewson, Hsu 1977; Noppa et al. 1980; Garrison and Castelli 1985);

cancers of the endometrium and colon (Garfinkel 1985; Graham et al. 1988; Verreault et al. 1989); arthritis (Anderson and Felson 1988; Felson 1988); and varicose veins. Obese women are more likely than lean women to experience menstrual abnormalities (Hartz et al. 1979) and complications in pregnancy (Abrams and Parker 1988). Obese individuals require more medical care (Tsai, Lucas, Bernacki 1988), experience more complications during and following surgical procedures (Schwartz 1955), and report greater limitations in performing tasks of everyday living (Stewart, Brook, Kane 1980).

The strength and consistency of the data and the understanding of causal mechanisms underlying obesity–disease associations vary from end-point to end-point. Nevertheless, there is little doubt that obesity represents an important health risk that may reduce both the quality and duration of life. The overall evidence linking overweight to disease has led to recommendations from numerous health organizations for individuals in the general population to control their weight as a means of preventing future illness (National Institutes of Health Consensus Development Conference Statement 1985; Subcommittee on Nonpharmacological Therapy of the 1984 Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure 1986; US DHHS 1988b).

Despite convincing data linking obesity to ill health, several issues in the area remain controversial. A key issue that is particularly germane to smoking cessation-induced weight gain is the extent to which modest degrees of overweight represent a health hazard. The most commonly recognized standards for acceptable body weights are those developed by the life insurance industry based on followup studies of policy holders conducted in 1959 and 1979 (Metropolitan Life Insurance Company 1960; Society of Actuaries and Association of Life Insurance Medical Directors of America 1980).

Each of these studies evaluated the mortality of approximately 4,000,000 life insurance policy holders. "Ideal" weight standards that were developed from these studies and widely used in subsequent research represent the gender- and height-specific weights associated with lowest mortality. Overall, a J-shaped relationship is observed between weight and mortality. Lowest premature mortality is associated with body weights that are about 10 percent below the population average. Excess premature mortality is associated with extremely low weights (i.e., body weights more than 10 percent below the standards), and premature mortality increases incrementally for increasing weights above the standard. In the range of weights that encompasses the vast majority of the population (i.e., relative weights of 1.0 to 1.3), the relationship between weight and mortality was approximately linear with each 1-percent increase in weight associated with about a 1-percent increase in premature mortality. Above relative weights of about 1.3, the curve rises even more steeply so that premature mortality may double at relative weights of 1.5 or more (Manson et al. 1987).

The overall relationship between weight and mortality has been confirmed in several other large scale prospective studies. For example, the American Cancer Society followup study of 750,000 men and women from the general U.S. population provides confirmatory data with specific detail on various causes of death (Lew and Garfinkel 1979). Table 3 presents mortality ratios for this study group by weight status for selected causes. Table 4 presents mortality ratios by weight and smoking status. Most

of the deaths associated with leanness occur among smokers, and although the shape of the weight-mortality curves are similar among smokers and never smokers, smokers have nearly twice the mortality rate compared with never smokers over much of the weight distribution. A recent 10-year followup study of 1,700,000 Norwegians confirms these findings in a non-U.S. population with regard to the shape of the weight mortality association and the causes of death at both ends of the distribution (Waaler 1988).

The reported relationship with age further complicates the relationship between body weight and health (Andres et al. 1985). For example, the strongest relationship between body weight and premature mortality holds for younger age groups (i.e., under 40 years of age). In older adults, the relationship between weight and mortality is weak over much of the weight distribution, and in the oldest groups studied (i.e., over 60 years of age), mortality appears inversely related to weight. Indeed, many prospective studies of middle-aged adults have observed little or no prognostic significance of body weight for either total premature mortality or major disease endpoints except at the extremes of the body weight distribution. These findings have led some researchers to argue that concerns about weight and overall health for most individuals have been exaggerated (Keys 1981; Barrett-Connor 1985). In contrast, other investigators have noted that cigarette smoking has not been statistically controlled in many of these analyses, and in addition, pathophysiologic effects of obesity, such as hypertension and hyperglycemia, have been inappropriately adjusted (Manson et al. 1987). Therefore, the health risks of obesity may have been underestimated.

Another issue to consider in the relationship between body weight and health is that all forms of overweight may not pose the same health risks. In particular, health risk may depend on weight status at different times in an individual's life. A study by Abraham, Collins, and Nordsieck (1971), for example, studied 1,087 white males for whom height and weight data were available at ages 9 to 13 and after a period of approximately 40 years. By cross-classifying respondents by childhood and adult weight status, these researchers found that individuals who were at the low end of the weight distribution as children, but who gained weight to reach the high end of the weight distribution as adults, were at significantly higher risk of hypertensive vascular disease and cardiovascular renal disease than were individuals who had high weights both as children and as adults. Similarly, in a report based on the Normative Aging Study, Borkan and colleagues (1986) found age by weight gain interactions, relating weight gain to health risk. Weight gain had a stronger positive association with change in fasting glucose levels for older men compared with younger men; however, weight gain was more strongly related to change in uric acid (positive) and forced vital capacity (negative) in younger men (Borkan et al. 1986).

The importance of timing issues in the relationship between body weight and disease is also apparent in weight cycling. Weight cycling refers to gaining and losing weight repeatedly over time. Such weight fluctuations might occur in individuals who repeatedly diet but are unable to maintain weight losses. Weight cycling might be caused by recurrent illnesses or major fluctuations in lifestyle. Such fluctuations might conceivably also occur among smokers who quit but relapse to smoking on multiple occasions. Several recent reports suggest that weight cycling may be associated with

TABLE 3.—Mortality ratios for all ages combined in relation to the death rate of those 90–109% of average weight

Cause of death	7th rev ICD	Gender	Weight index[a]						
			<80	80–89	90–109	110–119	120–129	130–139	≥140
Total deaths		Male	1.25	1.05	1.00	1.15	1.27	1.46	1.87
		Female	1.19	0.96	1.00	1.17	1.29	1.46	1.89
CHD	420	Male	0.88	0.90	1.00	1.23	1.32	1.55	1.95
		Female	1.01	0.89	1.00	1.23	1.39	1.54	2.07
Cancer, all sites	140–205	Male	1.33	1.13	1.00	1.02	1.09	1.14	1.33
		Female	0.96	0.92	1.00	1.10	1.19	1.23	1.55
Diabetes	260	Male	0.88	0.84	1.00	1.65	2.56	3.51	5.19
		Female	0.65	0.61	1.00	1.92	3.34	3.78	7.90
Digestive diseases	540–542 570–578	Male	1.39	1.28	1.00	1.45	1.88	2.89	3.99
	584–586	Female	1.58	0.92	1.00	1.66	1.61	2.19	2.29
Cerebrovascular diseases	330–334	Male	1.21	1.09	1.00	1.15	1.17	1.54	2.27
		Female	1.33	0.98	1.00	1.09	1.16	1.40	1.52

NOTE: CHD=coronary heart disease.

[a]Calculated by dividing a person's actual weight by the corresponding average weight for the appropriate sex-inch of height-5-yr age group, multiplied by 100.

SOURCE: Lew and Garfinkel (1979).

TABLE 4.—Mortality ratios for all ages combined according to smoking status in relation to those 90–109% of average age

Cause of death	Gender	Smoking status	Weight index[a]						
			<80	80–89	90–109	110–119	120–129	130–139	≥140
All causes of death	Male	Never smoked	0.88	0.75	0.75	0.91	0.98	1.16	1.69
		≥20 cig/day	1.68	1.40	1.34	1.53	1.76	2.00	2.21
		Other	1.22	1.01	0.93	1.04	1.15	1.29	1.66
	Female	Never smoked	1.10	0.88	0.93	1.08	1.20	1.37	1.74
		≥20 cig/day	1.98	1.59	1.64	1.82	2.22	2.30	2.73
		Other	1.53	1.13	1.12	1.40	1.42	1.62	2.04
Coronary artery disease (ICD 420)	Male	Never smoked	0.72	0.66	0.76	0.96	1.04	1.24	1.73
		≥20 cig/day	1.06	1.13	1.33	1.66	1.81	2.11	2.11
		Other	0.91	0.90	0.93	1.12	1.19	1.37	1.84
	Female	Never smoked	0.93	0.82	0.92	1.10	1.29	1.39	1.86
		≥20 cig/day	1.51	1.70	2.12	2.20	3.48	3.79	4.74
		Other	1.54	1.14	1.18	1.88	1.44	2.01	2.33
Cancer, all sites (ICD 140–205)	Male	Never smoked	0.60	0.60	0.66	0.69	0.79	0.90	0.76
		≥20 cig/day	2.07	1.71	1.43	1.46	1.55	1.71	2.00
		Other	1.20	1.03	1.90	0.89	1.05	0.87	1.22
	Female	Never smoked	0.85	0.85	0.96	1.06	1.16	1.19	1.50
		≥20 cig/day	1.49	1.36	1.34	1.50	1.34	1.70	1.49
		Other	1.11	0.98	1.03	1.06	1.16	1.11	1.60

[a]See Table 3 for definition.

SOURCE: Lew and Garfinkel (1979).

elevated premature mortality compared with maintaining a more stable weight over time. In a study by Hamm, Shekelle, and Stamler (1989), for example, CVD and cancer mortality and total mortality were compared among individuals who reported either having gained significant weight (N=133), having remained at the same weight (N=178), or both having gained and lost significant weight (N=98). Both gainers and cyclers had significantly elevated total mortality experience, relative risks of 1.5 and 1.4, respectively, compared with individuals whose weights remained constant. Three recently published abstracts (Lissner et al. 1989; Lissner, Collins et al. 1988; Lissner, Odell et al. 1988) have reported even greater health risks of weight cycling. Using prospective data from the Multiple Risk Factor Intervention Trial (MRFIT) (Lissner, Collins et al. 1988), two prospective studies from Göteborg, Sweden (Lissner et al. 1987), and the Framingham Study (Lissner, Odell et al. 1988), weight cycling was defined as the variability of weights recorded at repeat examinations. Controlling for a variety of possible confounding variables, weight cycling was independently predictive of total premature mortality and CVD mortality. In the analyses based on MRFIT, premature mortality among men with the most variable weights was 36 to 89 percent higher than among men with the most stable weights.

An additional issue to consider in the relationship between body weight and health is the distribution of body fat. Individuals differ in the location of stored adipose tissue. Research data show that individuals who store greater amounts of body fat in the abdominal region rather than in the hips or limbs have elevated cardiovascular risk factors (Gillum 1987; Selby, Friedman, Quesenberry 1989), CVD, and diabetes rates (Freedman and Rimm 1989; Lapidus and Bengtsson 1988) as well as reproductive system cancers among women (Bjorntorp 1988).

Usually measured by the ratio of abdominal circumference to hip circumference or the ratio of trunk versus peripheral skinfolds, a central body fat distribution is positively correlated with absolute body weight. However, in several studies, the centrality of fat distribution has proven to be a much stronger predictor of disease than body weight. A landmark study in this area was conducted by Larsson and colleagues (1984) who reported on 13 years of followup for 792 Swedish men aged 54 years at the time of first observation. Outcome measures were stroke, ischemic heart disease, and all-cause mortality. None of these health outcomes was significantly related to measures of adiposity (body mass index weight/height^2, the sum of several skinfold measurements, and body circumferences). However, the ratio of waist to hip circumference (WHR) was significantly and positively related to all three measures of illness and death. The relevance of this finding for ex-smokers, as discussed below, is that smoking is positively related to WHR and that smoking cessation is associated with a reduced WHR (Shimokata, Muller, Andres 1989).

Compared with pathophysiologic health risks, social and psychological pathologies associated with overweight are not as well established. This situation may reflect the relative absence of research in this area, but it may also indicate the absence of a strong relationship. Obesity is strongly disapproved of and discriminated against in this society (Allon 1973; Grunberg 1982b; Wadden and Stunkard 1985). Overweight individuals are falsely stereotyped as having a variety of undesirable characteristics, including self-indulgence, laziness, lack of self-control, and lack of intelligence.

The perception in this culture of obesity as unattractive has been documented in various populations. For example Richardson (1971), in a study of 10- and 11-year-olds' perception of the likableness of children with a variety of handicaps, found that obese children were judged less attractive than were children with amputations and facial disfigurement or children confined to wheelchairs. Similar biased impressions have been documented among adults and among physicians and medical students (Allon 1973; Maddox and Liederman 1969). Canning and Mayer (1966) found that the prevalence of obese students in college was less than the prevalence of obese students in high school despite no difference in academic performance in high school or in college application rates. A survey of employers indicates that many profess not to hire obese individuals (Roe and Eickwort 1976), and at least one survey of business executives suggests an inverse association between obesity and salary (*Industry Week* 1974). In a survey of college students, Kallen and Doughty (1984) found lower rates of reported dating in overweight subjects, although no less satisfaction with intimate relationships.

Although it is obvious that many overweight individuals are dissatisfied with their personal appearance, desire to lose weight, and frequently make efforts to lose weight (Wadden et al. 1989; Polivy, Garner, Garfinkel 1986; Adams 1980; Guggenheim, Poznanski, Kaufmann 1973; Dwyer, Feldman, Mayer 1975; Dwyer and Mayer 1970; Stewart and Brook 1983; Jeffery et al. 1984), evidence for severe psychological or social impairment in all but the most severe cases of obesity is generally lacking. Moore, Stunkard, and Srole (1962), reporting data from the Midtown Manhattan Study, found higher scores on three measures of psychological disability in the obese compared with the nonobese.

Data from the Rand Health Study and a Dutch population-based study indicated that obese individuals report that their weight imposes some restrictions on their everyday activities and causes them more pain and worry compared with the nonobese (Stewart, Brook, Kane 1980; Stewart and Brook 1983; Seidell et al. 1986). However, Stewart and Brook (1983) also reported that obese persons are less depressed than normal-weight persons, a finding corroborated in a study of British citizens by Crisp and McGuiness (1976). These mixed and inconsistent findings from studies of obese adults also have characterized studies of obese children (Wadden et al. 1989; Wadden et al. 1984). In extremely obese individuals presenting themselves for treatment (i.e., those 75 percent or more overweight), higher levels of psychological disturbance have been reported (Halmi et al. 1980; Atkinson and Ringuette 1967). Even here, it has been questioned whether such pathology is greater than that observed in normal-weight individuals presenting for medical or surgical procedures (Wise and Fernandez 1979; Swenson, Pearson, Osborne 1973). It has been suggested that unwarranted concerns about weight gain may contribute to eating disorders such as anorexia and bulimia (Wooley and Wooley 1984). Data supporting this idea, however, are largely anecdotal (Wadden and Stunkard 1985).

Prospective studies on the effects of weight gain on psychosocial functioning have not yet been reported. Studies of psychological changes accompanying weight loss generally show positive effects, even when weight loss is modest and not well maintained (Wing et al. 1984). Therefore, consistent with intuition, many people feel better

about themselves when they lose weight. However, the extrapolation of these findings to weight gain lacks empirical support.

In summary, although adverse psychological and social consequences of overweight have been much discussed in both lay and professional circles, such effects have not been well documented. Moreover, to the extent that associations have been reported, the direction of causation is unclear. More research in this area is warranted, particularly because the available research is not extensive and much of it is methodologically weak. At this time, data suggest that only the most extreme forms of obesity, the upper 1 or 2 percent of the weight distribution in this domain, pose significant hazards. However, it is important to emphasize that these conclusions reflect the lack of evidence for serious psychosocial problems resulting from modest weight gains. Nevertheless, many persons want to lose weight, many persons seek ways to lose weight, and many persons feel better about themselves when they lose weight.

CHANGE IN WEIGHT-RELATED HEALTH RISKS AFTER SMOKING CESSATION

As documented earlier in this Chapter, smoking cessation is associated with weight gain. An important question is the extent to which this weight gain might lead to elevations in blood pressure, cholesterol, glucose intolerance, or other factors that would offset the benefits of smoking cessation discussed in detail throughout this Report.

Relatively few studies have specifically examined the effect of smoking cessation on weight-related health risks. Seven studies were reviewed for this Report. Gordon and coworkers (1975) reported changes over an 18-year period in weight and related risk characteristics among individuals in the Framingham Study. At entry into the study, 61 percent of men and 40 percent of women smoked cigarettes; at the 18-year followup, 37 percent of men and 31 percent of women continued to smoke. Analyses of changes were restricted to men because of the small numbers of women who quit smoking in this sample. Male quitters were similar to those who continued to smoke in baseline characteristics except that the former group contained more diabetics. The authors interpret this finding as suggesting that ill health is an incentive to stop smoking.

Short-term effects of smoking cessation, defined as the change between the last examination at which smoking was reported and the first examination at which nonsmoking was reported (2-year intervals), included a weight gain of 3.8 pounds, an increase in systolic blood pressure of 1.6 mm Hg, and an increase in serum cholesterol of 0.2 mg/dL. Continuing smokers had an average weight gain of 0.3 pound, increased systolic blood pressure of 0.7 mm Hg, and decreased serum cholesterol of 0.2 mg/dL. For the same time period, nonsmokers had an average weight gain of 0.5 pound, increased systolic blood pressure of 0.7 mm Hg, and increased serum cholesterol of 0.3 mg/dL. Differences among groups in blood pressure and cholesterol changes were not statistically significant. Long-term changes associated with smoking cessation were evaluated by comparing changes between the fourth and the tenth examination, a period of 12 years, among continuing smokers, nonsmokers, and individuals smoking at entry but not smoking from the fourth to the tenth examination. Trends in weight, blood

pressure, serum cholesterol, and blood glucose did not differ significantly among these three groups.

Schoenenberger (1982) reported the relationship between smoking cessation and changes in body weight, blood pressure, and serum cholesterol over 3 years among men in the special intervention group in MRFIT. All men in the study were at high risk for heart disease and were being counseled throughout the study in smoking cessation and dietary changes to effect cholesterol reduction. When necessary, the men were also treated pharmacologically for elevated blood pressure. Results indicated significantly less weight loss in quitters (–0.6 pounds, i.e., a gain of 0.6 pounds) compared with nonsmokers and continuing smokers (5.7 and 3.6 pounds, respectively), no differences in blood pressure change (–9.6, –8.7, and –9.4 mm Hg, respectively, for systolic blood pressure among men not on medication), and greater reductions in serum cholesterol among quitters (–13.4 mg/dL) than in the other two groups (–10.0 and –8.1 mg/dL). The latter effect was interpreted as possibly reflecting a higher level of generalized motivation to reduce risk in the quitting group.

In a 5-year followup study of 2,283 persons with mild hypertension in eastern Finland, Tuomilehto and colleagues (1986) found that 26 percent of men and 35 percent of women who smoked at the time of the initial examination had quit. Among men, smoking cessation was associated with a 7.9-pound weight gain compared with 0.2-pound and 2.2-pound weight gains among nonsmokers and continuing smokers, respectively. Among women, weight loss after smoking cessation averaged 0.7 pound compared with gains of 0.1 pound and 2.2 pounds among nonsmokers and continuing smokers, respectively. Smoking cessation was not associated with a significant increase in blood pressure or serum cholesterol compared with continuing smokers or nonsmokers. Mean arterial pressure fell by 5.0 and 13.1 mm Hg in male and female quitters, respectively, compared with decreases of 6.9 and 8.7 mm Hg among nonsmokers and of 7.0 and 9.6 mm Hg among continuing smokers. Serum cholesterol fell between 0.63 and 0.66 mmol/L across the various subgroups.

Two papers relating smoking cessation to weight-related risks have been published based on data from the Normative Aging Study. The first report examined change over 5 years among 214 continuing smokers and 104 quitters (Garvey, Bossé, Seltzer 1974). An average weight gain of 4.2 pounds, which was accompanied by a 3.6 mm Hg increase in diastolic blood pressure, was observed among quitters compared with continuing smokers. The second report examined the relationship between smoking and body fat distribution, both cross-sectionally and longitudinally between examination visits scheduled 2 years apart (Shimokata, Muller, Andres 1989). Central body fat distribution, which poses increased health risks, as assessed by WHR was positively associated with smoking. Moreover, among smokers, daily cigarette consumption was positively associated with central adiposity. Smoking cessation was associated with increased body weight. However, despite the weight gain, the change in WHR among ex-smokers was small and, in fact, decreased slightly because hip circumference increased. Therefore, based on WHR data only, smoking rather than smoking cessation may pose a weight-related health risk.

Stamford and coworkers (1986) studied the short-term effects of smoking cessation on lipoprotein fractions. Among 13 women who successfully quit smoking for a period

of 48 days, these investigators observed a weight increase of 4.9 pounds. This weight change was accompanied by a nonsignificant increase in total cholesterol of 9 mg/dL and a significant increase in HDL-C of 7 mg/dL. Over the subsequent year, these favorable HDL-C changes were maintained in three individuals continuing to abstain from smoking, but were lost in nine individuals who returned to smoking.

One randomized trial of smoking cessation and weight-related health risks was located for this review. Rabkin (1984a) randomized 107 smokers to smoking cessation and 33 to continued smoking in a comparative study of smoking cessation strategies. A battery of physiologic measures was obtained at baseline and repeated 2 to 3 months following randomization. No differences were found in cessation rates among the different quitting strategies. Physiologic changes observed in the smoking cessation group as a whole (i.e., all those randomized) included a significant increase in weight (1.8 pounds) and skinfold thickness (6.6 mm) compared with the control group (0.4 pound and −7.0 mm), but no significant change in lipid profiles, fasting glucose, or blood pressure. Only 35 subjects in the cessation groups were successful in quitting smoking. Successful quitters gained significant amounts of weight compared with individuals who did not quit (4.4 vs. 0.7 pounds, respectively). Successful quitters also experienced significant increases in HDL-C compared with nonquitters (4.2 vs. 0.1 mg/dL). Changes in other weight-related risk factors did not differ among groups.

The studies reviewed above are consistent in their findings. Individuals who quit smoking and gain weight appear to experience relatively small changes in health-related risk factors such as blood pressure, serum cholesterol, and blood glucose. Moreover, some of the potentially adverse effects of weight gain on health risks are mitigated by changes in lipid profiles and in body fat distribution in a direction predictive of improved health outcomes. It seems likely that only those smokers who have large weight gains after smoking cessation would experience important changes in weight-related risk factors.

The characteristics of individuals most likely to gain harmfully large amounts of weight after smoking cessation merit additional investigation. Bossé, Garvey, and Costa (1980) have reported relevant findings from the Normative Aging Study. Over a 5-year period these investigators found that factors most predictive of weight gain among recent quitters were younger age, leanness of body build, and greater amounts of smoking. The latter finding is confirmed by other studies (Blitzer, Rimm, Giefer 1977; Gordon et al. 1975). There are no data available on specific predictors of excessive weight gain among ex-smokers. Research on predictors of weight gain suggest that those persons most likely to gain weight after smoking cessation may be those who can best afford it because they are relatively lean. They also may be those who need smoking cessation most because they smoke the most.

Quantitatively estimating the extent of health risk associated with weight gain after smoking cessation is a complex process. The health risks of obesity vary with age, the temporal patterning of weight changes, type of obesity, and other risk factors. Moreover, smoking cessation itself appears to have independent effects on some weight-related risk factors that may actually be beneficial.

It has been estimated that the health risks posed by regular smoking double overall mortality rates compared with never smoking (US DHHS 1989). Moreover, as detailed

elsewhere in this Report, there are clear health benefits associated with smoking cessation. The amount of excess body weight that would have to occur to offset the benefits of smoking cessation would have to be considerable. Yet, average weight gains after smoking cessation are only about 5 pounds, bringing most individuals to a weight level similar to that of their nonsmoking peers. As discussed in this Chapter, the proportion of ex-smokers who are likely to gain large amounts of weight (e.g., more than 20 pounds) is small. Therefore, although some individuals may experience these large weight gains, the number of individuals likely to gain enough weight to offset the benefits of smoking cessation is negligible. Also, the likelihood of adverse psychosocial consequences because of small weight gain seems remote for most people. Although further research in this area is warranted, there is little reason to expect weight gain to pose a substantive medical or psychosocial hazard to the vast majority of smokers who are quitting. For those persons who do gain excessive amounts of weight after smoking cessation, the health benefits of cessation still exist, and weight control programs rather than smoking relapse should be implemented. In conclusion, the clear reduction in health risks that results from smoking cessation overshadows any health risks that may result from smoking cessation-induced body weight gain.

STRATEGIES TO CONTROL POSTCESSATION WEIGHT GAIN

Because weight gain after smoking cessation commonly occurs and because many people, particularly young women, report smoking to control weight gain (Klesges and Klesges 1988; US DHHS 1990), strategies that successfully moderate postcessation weight gain may encourage weight-conscious smokers to attempt cessation and may facilitate the efforts of successful quitters to remain abstinent. Only a few controlled investigations have examined interventions for reducing weight gain after smoking cessation. Currently existing behavioral and pharmacologic interventions are summarized below.

Behavioral Methods for Reducing Postcessation Weight Gain

Smoking cessation programs that include a weight control component have not successfully increased smoking cessation. In one study, 79 women were randomly assigned to a 7-week smoking cessation program either with or without weight control information (Mermelstein 1987). At posttreatment and at followup, there were no significant differences in smoking cessation rates between the two groups. Participants in both groups gained weight during treatment; however, the weight increase for the smoking-cessation-plus-weight-control group was significantly less than the increase for the smoking-cessation-only group (1.4 vs. 2.4 pounds).

Several weight control strategies, as adjuncts to smoking cessation, were evaluated by Grinstead (1981). Forty-five subjects were randomly assigned to a 4-week smoking aversion program with one of three weight control interventions. No differences in smoking cessation rates were observed, and there were no weight change differences among the groups. Subjects in all groups gained weight during treatment.