

Lifetime Health and Economic Benefits of Weight Loss Among Obese Persons

ABSTRACT

Gerry Oster, PhD, David Thompson, PhD, John Edelsberg, MD, MPH, Amy P. Bird, BS, and Graham A. Colditz, MD, DrPH

Objectives. This study estimated the lifetime health and economic benefits of sustained modest weight loss among obese persons.

Methods. We developed a dynamic model of the relationship between body mass index (BMI) and the risks and costs of 5 obesity-related diseases: hypertension, hypercholesterolemia, type 2 diabetes, coronary heart disease (CHD), and stroke. We then calculated the lifetime health and economic benefits of a sustained 10% reduction in body weight for men and women aged 35 to 64 years with mild, moderate, and severe obesity.

Results. Depending on age, gender, and initial BMI, a sustained 10% weight loss would (1) reduce the expected number of years of life with hypertension, hypercholesterolemia, and type 2 diabetes by 1.2 to 2.9, 0.3 to 0.8, and 0.5 to 1.7, respectively; (2) reduce the expected lifetime incidence of CHD and stroke by 12 to 38 cases per 1000 and 1 to 13 cases per 1000, respectively; (3) increase life expectancy by 2 to 7 months; and (4) reduce expected lifetime medical care costs of these 5 diseases by \$2200 to \$5300.

Conclusions. Sustained modest weight loss among obese persons would yield substantial health and economic benefits. (*Am J Public Health.* 1999; 89:1536-1542)

The prevalence of obesity in the United States has increased significantly in recent years, from 25.4% for the period 1976 to 1980 to 35.2% for the period 1988 to 1994.¹ This trend may have far-reaching consequences, as obesity plays an important etiologic role in a variety of diseases, including hypertension, type 2 diabetes, coronary heart disease (CHD), and stroke.² In the Nurses' Health Study,³ for example, relative risks of type 2 diabetes were reported to be 40.3, 54.0, and 93.2 among women with body mass indexes (BMIs; weight in kilograms divided by the square of the height in meters) of 31 to 32.9 kg/m², 33 to 34.9 kg/m², and 35 kg/m² and more, respectively, relative to those with BMIs of less than 22 kg/m². In the Health Professionals Follow-Up Study, mildly obese men (BMI = 25-28.9 kg/m²) were reported to have a 50% higher risk of CHD than men with BMIs of less than 23 kg/m²; moderate obesity (BMI = 29-32.9 kg/m²) and severe obesity (BMI ≥ 33 kg/m²) were associated with a nearly 2-fold and more than 3-fold increase, respectively, in risk of CHD.⁴

Fortunately, there is mounting evidence that these risks can be reversed. Weight loss has been reported to improve blood pressure, lipid levels, and glucose tolerance among overweight persons with hypertension, dyslipidemia, and diabetes, respectively.⁵⁻¹¹ It also has been found to reduce medication requirements among both hypertensive and diabetic patients.^{12,13} While weight loss yields important benefits, recidivism is inordinately high. On average, two thirds of the weight that is lost by patients who complete weight-loss programs is regained within 1 year, and almost all of it is regained within 5 years.¹⁴

The high rate of recidivism following weight loss may be due in part to recently discovered genetic factors that determine obesity status and to complex biochemical systems that tend to maintain body weight.¹⁵⁻¹⁷

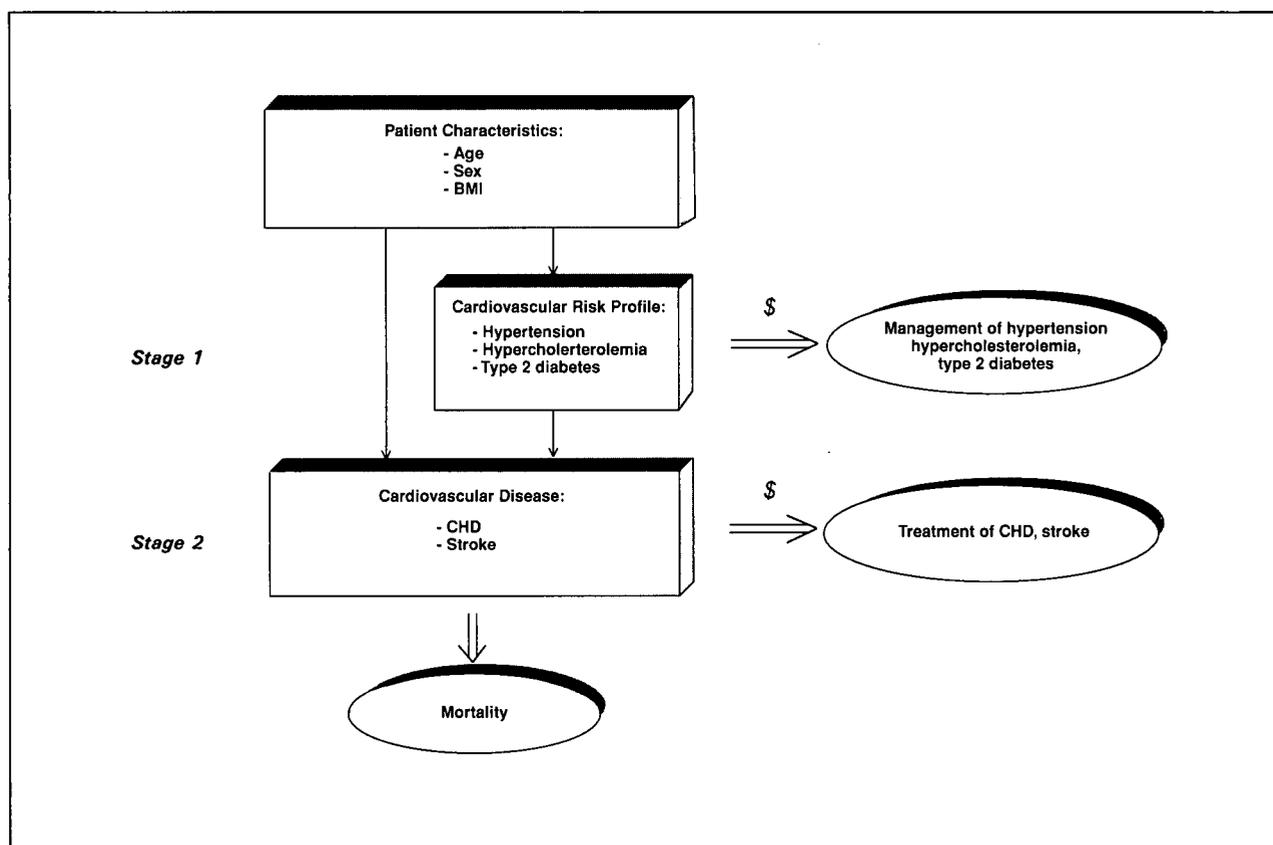
These findings have led to the conceptualization of obesity as a chronic disease condition rather than a problem caused by overeating, inadequate physical activity, and lack of willpower. As such, obesity may require lifelong treatment, as with other chronic diseases. While the etiologic role of obesity in various disease conditions might lead one to suspect that the lifetime health and economic benefits of such therapy would be substantial, data are in fact limited on the long-term consequences of intentional weight loss among obese persons. Although consideration has been given to the lifetime health and economic benefits of modifying other risk factors, such as cigarette smoking and elevated cholesterol levels,¹⁸⁻²⁰ no such analysis has been performed for weight loss.

In this study, we consider the expected lifetime health and economic benefits of weight loss among persons who are obese, using a dynamic model of the relationship between BMI and the risks and costs of hypertension, hypercholesterolemia, type 2 diabetes, CHD, and stroke. These 5 diseases (and their sequelae, such as end-stage renal disease resulting from type 2 diabetes) have been reported to account for approximately 85% of total obesity-attributable medical care costs, which totaled \$45.8 billion in 1990.²¹ We focus attention on the benefits of sustained modest (i.e., 10%) weight loss among men and women aged 35 to 64 years with initial BMIs of

Gerry Oster, David Thompson, John Edelsberg, and Amy P. Bird are with Policy Analysis, Inc (PAI), Brookline, Mass. Graham A. Colditz is with the Department of Medicine, Channing Laboratory, Brigham and Women's Hospital and Harvard Medical School, Boston, Mass.

Requests for reprints should be sent to Gerry Oster, PhD, Policy Analysis, Inc (PAI), 4 Davis Ct, Brookline, MA 02445 (e-mail: goster@pai2.com).

This article was accepted February 19, 1999.



Note. BMI = body mass index; CHD = coronary heart disease.

FIGURE 1—Illustration of cost-of-obesity model.

153711

27.5 kg/m², 32.5 kg/m², and 37.5 kg/m² (representing mild, moderate, and severe obesity, respectively).

Methods

Description of Model

Our analysis is based on a dynamic model of the relationship between BMI and 5 diseases for which obesity is an established risk factor: hypertension, hypercholesterolemia, type 2 diabetes, CHD, and stroke. The model is composed of a set of interrelated equations that estimate the lifetime risks and costs of these diseases for men and women in relation to current age and BMI; it is similar to models previously employed by 2 of the authors in analyses of the benefits of smoking cessation^{18,19} and by 1 of the authors in an analysis of the consequences of cholesterol reduction.²⁰

Because some of the diseases for which obesity is a risk factor—namely, hypertension, hypercholesterolemia, and type 2 diabetes—are themselves risk factors for CHD and stroke, our model is structured in 2 stages to embody the complex nature of these relationships (Figure 1). In stage 1, we generate predicted risks of hypertension, hypercholesterolemia, and type 2 diabetes for men and women, respectively, in each future year of life in relation to BMI. In stage 2, we estimate the risks of CHD and stroke during each future year of life on the basis of BMI and the predicted risks of hypertension, hypercholesterolemia, and type 2 diabetes from the first stage of the model. Our analysis therefore reflects obesity's direct effects on future cardiovascular disease risks as well as its indirect effects on these risks as a result of changes in blood pressure, serum cholesterol, and glucose metabolism.

Expected medical care costs are generated and tallied in both stages of the model.

In the first stage, the expected costs of hypertension, hypercholesterolemia, and type 2 diabetes in each future year of life are calculated on the basis of the estimated risks of these diseases and corresponding estimates of medical care costs; to avoid double-counting, costs of cardiovascular disease (e.g., heart attack, stroke) attributable to these 3 diseases are not included in this stage. In the second stage, the expected costs of CHD and stroke in each future year of life are calculated in similar fashion on the basis of future risks of these diseases and their associated medical care costs. The perspective of our analysis was societal.

In our calculations, we hold BMI constant at its initial value as we "age" persons over time; all other risk factors are adjusted to age-appropriate values. Our model accordingly generates a stream of annual BMI-specific disease risks and economic costs running from current age to 99 years of age. We translate this lifetime stream of costs into a

summary measure of economic burden by discounting and summing these annual estimates, after adjusting for the probability that persons would survive to each future year.

In our model, we conservatively assume that all obesity-attributable mortality arises as a result of elevated risks of CHD and stroke; hence, we assume that persons can die of CHD, stroke, or causes unrelated to these diseases, but not directly of any other complications of hypertension, hypercholesterolemia, or type 2 diabetes (e.g., kidney disease). The probability of survival to any given future age is therefore calculated as the sum of 3 conditional probabilities: (1) survival given prior onset of CHD, (2) survival given prior onset of stroke, and (3) survival free of these diseases. Life expectancies are calculated as customary by summing annual probabilities of survival, from current age through 99 years of age.

To estimate the effects of sustained weight loss, we assume that obese persons of any given age and sex who reduce their BMI as a result of intervention adopt the risk characteristics of those who are at that lower level naturally. Thus, for example, a 45-year-old man with an initial BMI of 30 kg/m² who reduces his body weight by 10% is assumed to adopt the risk profile of another man of the same age whose naturally occurring BMI is 27 kg/m² (i.e., 30 kg/m² minus 10%). For modeling simplicity, we assume that the benefits of weight loss accrue immediately and that postintervention BMI would be maintained throughout all the remaining years of life.

A technical appendix that sets forth all model equations is available from the authors upon request.

Estimation of Model

We estimated our model by using data from a variety of secondary sources, which we describe in detail below. All costs were estimated at 1996 price levels; the Medical Care Component of the Consumer Price Index for All Urban Consumers was used to adjust prices, where necessary. Future costs were discounted on the basis of a real annual rate of 3%, consistent with recommendations of the US Public Health Service Task Force on Cost-Effectiveness in Health and Medicine.²²

Stage 1: Risks of Hypertension, Hypercholesterolemia, and Type 2 Diabetes

We used data from the Third National Health and Nutrition Examination Survey (NHANES III)²³ to estimate risks of hyper-

tension, hypercholesterolemia, and type 2 diabetes (as well as mean levels of diastolic blood pressure and total serum cholesterol) for men and women, respectively, in relation to age and BMI. NHANES III was a large national survey designed to obtain information on the health and nutritional status of the US population through interviews and physical examinations. The variables of interest were expressed as a function of age, age squared, and BMI, and techniques of regression analysis (logistic for dichotomous measures, linear for continuous measures) were employed to estimate these equations separately for men and women with data from NHANES III. We then used the estimated β coefficients of these regressions to predict mean values of the variables of interest in each age-sex-BMI stratum. Because of sample size limitations, we limited our attention to persons aged 35 to 84 years. In the model, we accordingly assumed that the risks of hypertension, hypercholesterolemia, and type 2 diabetes would remain constant at levels prevailing at 84 years of age throughout all remaining years of life (i.e., to 99 years of age).

To estimate the risk of hypertension, we considered NHANES III survey respondents to have hypertension if (1) their mean diastolic blood pressure was more than 90 mm Hg, (2) their mean systolic blood pressure was more than 160 mm Hg, or (3) they responded yes to a question concerning current use of drug therapy to control their blood pressure. To estimate the risk of elevated serum cholesterol, we similarly considered survey respondents to have hypercholesterolemia if (1) their total cholesterol level was higher than 240 mg/dL or (2) they responded yes to a question concerning current use of drug therapy to reduce their cholesterol level. In NHANES III, blood pressure was measured up to 6 times; we used the average of all available readings for both systolic and diastolic blood pressure. Since treatment could mask the true biologic relationships between BMI and both blood pressure and total cholesterol level, we excluded persons who were receiving antihypertensive or cholesterol-lowering medications from these analyses.

In NHANES III, persons with diabetes were not specifically identified as having type 2 or type 1 diabetes. We considered all persons (other than those with gestational diabetes) who reported that they had diabetes to have type 2 diabetes if (1) they were 30 years or older at onset, or (2) they were 19 to 29 years of age at onset and were not taking insulin, or (3) they were 19 to 29 years of age at onset and were currently taking insulin but did not begin taking insulin for at least 12 months following disease onset.²⁴

Stage 1: Costs of Hypertension, Hypercholesterolemia, and Type 2 Diabetes

We estimated the annual cost of treating patients with hypertension (\$670) by averaging the costs of pharmacological therapy and provider services in the first, second, and all subsequent years following diagnosis, as reported by Odell and Gregory.²⁵ Unpublished data from NHANES III indicate that 80.9% of all persons with elevated blood pressure receive antihypertensive therapy.²³ In the model, we accordingly estimated that the expected annual cost of each case of hypertension was \$542 (or 80.9% of \$670).

Our estimate of the annual cost of treating patients with hypercholesterolemia (\$705) was based on a recent study reported by Oster and colleagues.²⁶ Unpublished data from NHANES III indicate that only 22.0% of all persons with hypercholesterolemia receive cholesterol-lowering medications.²³ In the model, we therefore assumed that the expected annual cost of each case of hypercholesterolemia would be \$155 (or 22.0% of \$705).

Age- and sex-specific estimates of the costs of type 2 diabetes were derived from a study reported by Huse and colleagues.²⁷ Costs of hospital care, physician care, nursing home care, drugs, and other professional services associated with type 2 diabetes, as well as those associated with type 2 diabetes-induced atherosclerosis, blindness, glaucoma, cataract, and end-stage renal disease, were included in this estimate; diabetes-attributable costs of CHD and stroke were excluded to avoid double-counting. The annual costs of type 2 diabetes for men aged 35 to 64 years and 65 to 99 years were \$2025 and \$2806, respectively. Corresponding estimates for women were \$2374 and \$4454.

Stage 2: Risks of CHD, Stroke, and Mortality

We used data from the Framingham Heart Study to estimate the risks of CHD and stroke for men and women in relation to age, BMI, and other risk factors. Initiated in 1948, the Framingham Heart Study has followed a cohort of more than 5000 men and women 30 to 62 years of age at study entry (as well as their offspring) to document the risk factors for cardiovascular disease.

Using data from the Framingham Heart Study, we estimated logistic functions to predict the risks of CHD and stroke over 2 years as a function of age, age squared, BMI, diastolic blood pressure, total cholesterol level, presence of type 2 diabetes, and current smoking habit. Separate logistic functions were estimated for men and women aged

35 to 64 years and 65 to 84 years; only primary events were considered. As in prior research,²⁸ CHD was defined to include sudden death, nonsudden death, myocardial infarction, unstable angina pectoris, and stable angina pectoris. Stroke included both hemorrhagic and ischemic stroke. Logistic functions were estimated from data for the original cohort of Framingham Heart Study participants; 2-year risks were halved to approximate annual risks.

Once these functions were estimated, we used them to predict the risks of CHD and stroke among persons 35 to 84 years of age on the basis of age, sex, BMI, and the corresponding predicted values of diastolic blood pressure, serum cholesterol level, and risk of type 2 diabetes (i.e., from stage 1). In the absence of data for persons 85 to 99 years of age, risks of CHD and stroke were assumed to be constant and invariant with respect to BMI; we estimated these risks by using the logistic functions for persons 65 to 84 years of age, setting age at 85 years and all other risk factors at NHANES III-derived mean population values for men and women 85 years and older. Since cigarette smoking could have confounded our estimates of CHD and stroke risk, we estimated risks for nonsmokers only.

For both CHD and stroke, we assumed that onset of disease would have an impact on mortality only in the first 10 years following onset; we assumed that thereafter mortality rates would return to general population levels. Mortality following CHD was estimated from unpublished data from the CHD Policy Research Institute (Karen Kuntz, ScD, written communication, August 1997); mortality following stroke was estimated from the results of a Rochester, Minn, study of survival after first cerebral infarction.²⁹ In the absence of data, mortality rates following CHD or stroke for persons 85 to 99 years of age were assumed to be equal to those of the general population.

General population mortality rates for men and women aged 35 to 84 years were obtained from US Vital Statistics³⁰; rates for persons aged 85 to 99 years were obtained from the Social Security Administration (unpublished data, 1997). For persons younger than 85 years, age- and sex-specific mortality rates from causes other than CHD or stroke were estimated by subtracting CHD and stroke mortality from general population mortality rates for the US population.³⁰

Stage 2: Costs of CHD and Stroke

As with survival following onset of CHD or stroke, we assumed that both of these diseases would give rise to medical care

TABLE 1—Expected Years of Life^a With Hypertension, Hypercholesterolemia, and Type 2 Diabetes Prior to Intervention and Expected Disease-Years Averted Following 10% Weight Loss, by Sex, Current Age, and Initial Body Mass Index (BMI)

Sex and Current Age, y	Expected Years of Life With Disease/Disease-Years Averted, by Initial BMI		
	27.5 kg/m ²	32.5 kg/m ²	37.5 kg/m ²
Hypertension			
Men			
35–44	12.0/2.0	15.9/2.6	19.8/2.9
45–54	10.4/1.7	13.5/2.0	16.3/2.1
55–64	8.2/1.2	10.4/1.5	12.4/1.4
Women			
35–44	15.9/2.1	19.9/2.6	23.7/2.9
45–54	14.7/1.9	18.1/2.2	21.0/2.2
55–64	12.7/1.5	15.2/1.6	17.2/1.4
Hypercholesterolemia			
Men			
35–44	9.7/0.5	10.7/0.6	11.7/0.7
45–54	7.6/0.4	8.3/0.5	9.0/0.5
55–64	5.2/0.3	5.8/0.3	6.2/0.4
Women			
35–44	14.6/0.6	15.7/0.7	16.7/0.8
45–54	12.9/0.5	13.8/0.6	14.6/0.6
55–64	10.0/0.4	10.7/0.4	11.3/0.4
Type 2 Diabetes			
Men			
35–44	3.0/0.7	4.6/1.1	6.7/1.7
45–54	2.8/0.6	4.3/1.0	6.3/1.5
55–64	2.4/0.5	3.6/0.8	5.2/1.2
Women			
35–44	3.7/0.7	5.3/1.1	7.5/1.7
45–54	3.4/0.6	4.9/1.0	6.8/1.5
55–64	3.0/0.6	4.2/0.9	5.8/1.2

^aAmong persons assumed initially free of coronary heart disease and stroke.

costs over a period of 10 years (or up to age 99 years for events occurring after age 90 years). Age-at-onset-specific estimates of the annual cost of CHD for men and women in each of the 10 years following onset were obtained from the CHD Policy Research Institute (Karen Kuntz, ScD, written communication, August 1997). These estimates represent a weighted average of the expected costs of myocardial infarction, cardiac arrest, and angina pectoris, and they include costs for hospital care, prescription drugs, physician services, laboratory tests, and rehabilitation services for the diagnosis and treatment of acute myocardial infarction, coronary revascularization, annual maintenance therapy after acute myocardial infarction, cardiac arrest (fatal or nonfatal), and congestive heart failure. Estimates also include expected costs of secondary events.

Estimates of the annual costs of stroke were obtained from a study of the cost-effectiveness of stroke prevention in high-risk patients.³¹ First-year costs, amounting to \$12 634, included costs for inpatient hospital and professional services as well as inpatient rehabilitation. Age-specific costs of stroke in

years 2 through 10 were obtained by annualizing the monthly costs of the following: nursing home care; all visits to physicians, physical therapists, and speech therapists; home health care; and inpatient acute and rehabilitation care for recurrent strokes. These costs were estimated to be \$3171, \$5356, and \$9421 annually for persons aged 35 to 74 years, 75 to 84 years, and 85 years and older, respectively.

Analyses

We used our model to examine the effects of a sustained 10% reduction in body weight on the following: expected years of life with hypertension, hypercholesterolemia, and type 2 diabetes; lifetime incidence of CHD and stroke; life expectancy; and lifetime medical care costs of these 5 diseases (in 1996 dollars, discounted at 3% annually). We estimated these measures for men and women aged 35 to 44 years, 45 to 54 years, and 55 to 64 years with initial BMIs of 27.5 kg/m², 32.5 kg/m², and 37.5 kg/m², representing persons with mild, moderate, and severe obesity, respectively.

TABLE 2—Expected Lifetime Number of Cases^a of Coronary Heart Disease and Stroke Prior to Intervention and Expected Lifetime Number of Cases Averted Following 10% Weight Loss, by Sex, Current Age, and Initial Body Mass Index (BMI) (Per 1000 Persons)

Sex and Current Age, y	Expected Lifetime Number of Cases/Cases Averted per 1000 Persons, by Initial BMI		
	27.5 kg/m ²	32.5 kg/m ²	37.5 kg/m ²
Coronary Heart Disease			
Men			
35-44	385/19	426/28	476/38
45-54	380/17	418/26	464/36
55-64	354/12	379/17	412/25
Women			
35-44	287/18	325/26	372/36
45-54	286/18	324/26	371/36
55-64	271/16	304/23	345/32
Stroke			
Men			
35-44	143/5	153/7	163/7
45-54	150/6	162/8	174/9
55-64	165/8	181/10	198/13
Women			
35-44	166/1	168/1	169/1
45-54	167/1	169/1	170/1
55-64	172/2	175/2	178/2

^aAmong persons assumed initially free of coronary heart disease and stroke.

TABLE 3—Savings in Expected Lifetime Medical Care Costs^a of Selected Obesity-Related Diseases,^b by Sex, Current Age, and Initial Body Mass Index (BMI)

Sex and Current Age, y	Savings in Lifetime Medical Care Costs, by Initial BMI ^c		
	27.5 kg/m ²	32.5 kg/m ²	37.5 kg/m ²
Men			
35-44	\$2300	\$3500	\$4900
45-54	\$2600	\$3800	\$5300
55-64	\$2300	\$3500	\$4800
Women			
35-44	\$2200	\$3300	\$4600
45-54	\$2500	\$3700	\$5100
55-64	\$2700	\$3800	\$5200

^aAmong persons assumed initially free of coronary heart disease and stroke.

^bCoronary heart disease, stroke, hypertension, hypercholesterolemia, and type 2 diabetes.

^cIn 1996 US dollars.

Results

Years of Life With Hypertension, Hypercholesterolemia, and Type 2 Diabetes

Expected years of life with hypertension, hypercholesterolemia, and type 2 diabetes before intervention and the expected numbers of disease-years averted as a result of a sustained 10% reduction in body weight are presented in Table 1. Depending on current age, sex, and initial BMI, weight loss would reduce the expected number of years of life with hypertension by 1.2 to 2.9 and the expected number of years with type 2 dia-

betes by 0.5 to 1.7; estimated reductions for hypercholesterolemia are more modest, ranging from 0.3 to 0.8 years. Benefits are comparable for men and women.

Lifetime Risks of CHD and Stroke

Lifetime risks of CHD and stroke before intervention and the expected reduction as a result of a sustained 10% weight loss are presented in Table 2. Lifetime risks of CHD decline by 12 to 38 cases per 1000, depending on current age, sex, and initial BMI; reductions are comparable for men and women and are slightly larger (absolutely as

well as relatively) among those who are younger. Risk reductions for stroke are generally more modest, ranging from 1 to 13 cases per 1000, and are greater for men than women.

Life Expectancy

Depending on current age and initial BMI, a sustained 10% weight loss would increase life expectancy by 2 to 7 months among men and by 2 to 5 months among women (data not presented). Estimated gains in life expectancy are greatest for younger persons and persons with the highest initial BMIs.

Lifetime Medical Care Costs

A sustained 10% reduction in body weight would decrease expected lifetime medical care costs of hypertension, hypercholesterolemia, type 2 diabetes, CHD, and stroke by \$2300 to \$5300 for men and \$2200 to \$5200 for women (Table 3). Economic benefits are greatest for men 45 to 54 years of age and women 55 to 64 years of age. Most of the cost savings associated with weight loss derive from expected reductions in the costs of treating type 2 diabetes, CHD, and hypertension (data not presented).

Discussion

Our findings suggest that modest sustained weight loss may yield substantial health and economic benefits. Among persons who are moderately obese, for example, a 10% weight loss would reduce the expected number of years of life with hypertension and diabetes by 1.5 to 2.6 and 0.8 to 1.1, respectively, depending on age and sex. Lifetime risk of CHD would be reduced by 17 to 28 cases per 1000. Expected lifetime medical care costs of the 5 diseases of interest—hypertension, hypercholesterolemia, type 2 diabetes, CHD, and stroke—would decline by approximately \$3300 to \$3800 per person.

While the estimated benefits of weight loss appear substantial, we believe that our findings may be conservative. Modeling complexity prevented us from examining the relationship between changes in BMI and the risks and costs of other diseases that have been linked to obesity, including gallbladder disease,³² osteoarthritis of the knee,³³ and endometrial cancer.³⁴ In addition, although available evidence suggests that average body weight increases with age among US adults,¹ we assumed that BMI would remain constant throughout all remaining years of life in the absence of intervention. This assumption was

necessitated by the lack of data characterizing change in weight over time in relation to initial BMI. Had such an effect been included in our model, it is likely that our estimates of the lifetime health and economic benefits of weight loss would have been larger.

Additional limitations of our analysis should be noted. For one, we assumed that the benefits of weight loss could be expressed in terms of differences in naturally occurring disease risks corresponding to the preintervention and postintervention BMIs that we examined. In fact, it is unknown whether sustained weight loss modifies obesity-related disease risks in this fashion, because no large-scale, long-term intervention studies have been reported to date. We note that our approach has been used in model-based analyses of other interventions, including cholesterol reduction²⁰ and smoking cessation.^{18,19}

Findings reported from epidemiologic studies of the long-term effects of weight loss generally have not lent support to the notion that losing weight improves health and increases longevity.^{35,36} Most such studies have focused on the effects of weight change on all-cause mortality, and they have reported that mortality is in fact *highest* among persons who lose weight or gain excessive weight and *lowest* among those who gain a modest amount. However, these studies are subject to a number of important limitations. For one, they often do not distinguish voluntary from involuntary weight loss; the latter may be indicative of subclinical disease affecting mortality risk. In addition, although these studies typically control for smoking habit, they do not control for recent smoking cessation, which is associated with weight gain as well as improved mortality. They also do not control for weight cycling (i.e., repeated weight loss followed by weight gain), which may increase mortality risk independent of actual weight change.³⁷

Finally, these studies rarely account for relative weight or other important risk characteristics at study entry. Weight loss among persons who are already underweight may increase mortality risk, for example, and survivors of a heart attack or stroke may voluntarily lose weight but nonetheless have higher mortality rates than persons who gain a modest amount but do not have preexisting cardiovascular disease. A recent prospective study that controlled for many of these potential confounders reported substantial reductions in mortality among middle-aged women with obesity-related health conditions after intentional weight loss.³⁸

Intervention studies suggest that patients can achieve a 10% weight loss with reduced-calorie diets, exercise, behavioral modifi-

cation, pharmacological therapy, or programs that combine more than one of these approaches.^{39,40} Relatively few patients, however, seem to be able to maintain their reduced weight over time. It may be argued, therefore, that our analysis is largely irrelevant at this time because lifelong maintenance of a 10% weight loss is unrealistic. More effective therapies for obesity are likely to emerge, however, with growing acceptance in the medical community that obesity is in fact a chronic illness requiring long-term management and ongoing research concerning the genetic and biochemical mechanisms regulating body weight. From this perspective, our findings serve to highlight the potential benefits of new therapies and accordingly may serve as a stimulus for biomedical research.

The health and economic consequences of obesity have drawn considerable interest in recent years. To the best of our knowledge, our study is the first to examine the lifetime health and economic benefits of intentional weight loss among persons who are obese. Our findings suggest that a modest (i.e., 10%) weight loss—maintained for life—would yield important benefits in both human and economic terms. □

Contributors

G. Oster, D. Thompson, and J. Edelsberg wrote the paper with input from G. A. Colditz and A. P. Bird. All authors have approved the final version of the paper and take responsibility for its content.

Acknowledgments

Funding for this research was provided by Knoll Pharmaceutical Company, Mount Olive, NJ.

We thank the following people, without whom this study would not have been possible: Ralph B. D'Agostino, PhD, of the Department of Mathematics, Boston University; George W. Petty, MD, of the Department of Neurology, Mayo Clinic, Rochester, Minn; Paula Goldman, MPH, Karen Kuntz, ScD, and Milton Weinstein, PhD, of the CHD Policy Research Institute Inc, an affiliate of the Harvard School of Public Health and the Brigham and Women's Hospital, Boston, Mass; and Karen Hirsh, MPH, Scott Thompson, and G. Rhys Williams, MS, of Knoll Pharmaceutical Company.

References

1. Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960–1994. *Int J Obes*. 1998;22:39–47.
2. Pi-Sunyer FX. Medical hazards of obesity. *Ann Intern Med*. 1993;119(pt 2):655–660.
3. Colditz GA, Willett WC, Rotnitzky A, Manson JE. Weight gain as a risk factor for clinical diabetes mellitus in women. *Ann Intern Med*. 1995;122:481–486.
4. Rimm EB, Stampfer MJ, Giovannucci E, et al. Body size and fat distribution as predictors of

- coronary heart disease among middle-aged and older US men. *Am J Epidemiol*. 1995;141:1117–1127.
5. MacMahon SW, Macdonald GJ, Bernstein L, Andrews G, Blacket RB. Comparison of weight reduction with metoprolol in treatment of hypertension in young overweight patients. *Lancet*. 1985;1(8440):1233–1236.
6. Wassertheil-Smoller S, Blafloux MD, Oberman AS, Langford HG, Davis BR, Wylie-Rosett J. The Trial of Antihypertensive Interventions and Management (TAIM) study: adequate weight loss, alone and combined with drug therapy in the treatment of mild hypertension. *Arch Intern Med*. 1992;152:131–136.
7. Grimm RH Jr, Flack JM, Grandits GA, et al. Long-term effects on plasma lipids of diet and drugs to treat hypertension. *JAMA*. 1996;275:1549–1556.
8. Doar JWH, Wilde CE, Thompson ME, Sewell PFI. Influence of treatment with diet alone on oral glucose-tolerance test and plasma sugar and insulin levels in patients with maturity-onset diabetes mellitus. *Lancet*. 1975;1(7919):1263–1266.
9. Wing RR, Koeske R, Epstein LH, Nowalk MP, Gooding W, Becker D. Long-term effects of modest weight loss in type II diabetic patients. *Arch Intern Med*. 1987;147:1749–1753.
10. Osterman J, Lin T, Nankin HR, Brown KA, Hornung CA. Serum cholesterol profiles during treatment of obese outpatients with a very low calorie diet. Effect of initial cholesterol levels. *Int J Obes*. 1992;16:49–58.
11. Pories WJ, MacDonald KG Jr, Morgan EJ, et al. Surgical treatment of obesity and its effect on diabetes: 10-y follow-up. *Am J Clin Nutr*. 1992;55(suppl):582S–585S.
12. Schotte DE, Stunkard AJ. The effects of weight reduction on blood pressure in 301 obese patients. *Arch Intern Med*. 1990;150:1701–1704.
13. Collins RW, Anderson JW. Medication cost savings associated with weight loss for obese non-insulin-dependent diabetic men and women. *Prev Med*. 1995;24:369–374.
14. Methods for voluntary weight loss and control: NIH Technology Assessment Conference Panel. Consensus Development Conference, 30 March to 1 April 1992. *Ann Intern Med*. 1993;119(pt 2):764–770.
15. Rosenbaum M, Leibel RL, Hirsch J. Obesity. *N Engl J Med*. 1997;337:396–407.
16. Comuzzie AG, Allison DB. The search for human obesity genes. *Science*. 1998;280:1374–1377.
17. Woods SC, Seeley RJ, Porte D Jr, Schwartz MW. Signals that regulate food intake and energy homeostasis. *Science*. 1998;280:1378–1383.
18. Oster G, Colditz GA, Kelly NL. *The Economic Costs of Smoking and Benefits of Quitting*. Boston, Mass: DC Heath; 1984.
19. Oster G, Colditz GA, Kelly NL. The economic costs of smoking and benefits of quitting for individual smokers. *Prev Med*. 1984;13:377–389.
20. Oster G, Epstein AM. Primary prevention and coronary heart disease: the economic benefits of lowering serum cholesterol. *Am J Public Health*. 1986;76:647–656.
21. Wolf AM, Colditz GA. The cost of obesity: the US perspective. *PharmacoEconomics*. 1994;5(suppl 1):34–37.

22. Weinstein MC, Siegel JE, Gold MR, Kamlet MS, Russell LB, for the Panel on Cost-Effectiveness in Health and Medicine. Recommendations of the Panel on Cost-Effectiveness in Health and Medicine. *JAMA*. 1996;276:1253-1258.
23. National Center for Health Statistics. *Third National Health and Nutrition Examination Survey, 1988-1994, NHANES III Adult, Exam, and Laboratory Data Files* [book on CD-ROM]. Hyattsville, Md: Centers for Disease Control and Prevention; 1996. Public Use Data File Documentation 76200.
24. Rewers M, Hamman RF. Risk factors for non-insulin-dependent diabetes. In: Harris MI, Cowie CC, Stern MP, Boyko EJ, Reiber GE, Bennett PH, eds. *Diabetes in America*. 2nd ed. Bethesda, Md: National Institutes of Health; 1995:179-220.
25. Odell TW, Gregory MC. Cost of hypertension treatment. *J Gen Intern Med*. 1995;10:686-688.
26. Oster G, Borok GM, Menzin J, et al. Cholesterol-Reduction Intervention Study (CRIS): a randomized trial to assess effectiveness and costs in clinical practice. *Arch Intern Med*. 1996;156:731-739.
27. Huse DM, Oster G, Killen AR, Lacey MJ, Colditz GA. The economic costs of non-insulin-dependent diabetes mellitus. *JAMA*. 1989;262:2708-2713.
28. Shurtleff D. Section 30: some characteristics related to the incidence of cardiovascular disease and death: Framingham study, 18-year follow-up. In: Kannel WB, Gordon T, eds. *The Framingham Study: An Epidemiological Investigation of Cardiovascular Disease*. Washington, DC: Dept of Health, Education and Welfare; 1974. DHEW publication (NIH) 74-599.
29. Petty GW, Brown RD, Whisnant JP, Sicks JD, O'Fallon WM, Wiebers DO. Survival and recurrence after first cerebral infarction: a population-based study in Rochester, Minnesota, 1975 through 1989. *Neurology*. 1998;50:208-216.
30. National Center for Health Statistics. *Vital Statistics of the United States, 1992, Vol II: Mortality, Part B*. Washington, DC: Public Health Service; 1996.
31. Oster G, Huse DM, Lacey MJ, Epstein AM. Cost-effectiveness of ticlopidine in preventing stroke in high-risk patients. *Stroke*. 1994;25:1149-1156.
32. Stampfer MJ, Maclure KM, Colditz GA, Manson JE, Willett WC. Risk of symptomatic gallstones in women with severe obesity. *Am J Clin Nutr*. 1992;55:652-658.
33. Felson DT, Anderson JJ, Naimark A, Walker AM, Meenan RF. Obesity and knee osteoarthritis: The Framingham Study. *Ann Intern Med*. 1988;109:18-24.
34. Ballard-Barbash R, Swanson CA. Body weight: estimation of risk for breast and endometrial cancers. *Am J Clin Nutr*. 1996;63(suppl):437S-441S.
35. Andres R, Muller DC, Sorkin JD. Long-term effects of change in body weight on all-cause mortality. *Ann Intern Med*. 1993;119(pt 2):737-743.
36. Pamuk ER, Williamson DF, Serdula MK, Madans J, Byers TE. Weight loss and subsequent death in a cohort of US adults. *Ann Intern Med*. 1993;119(pt 2):744-748.
37. Lissner L, Odell PM, D'Agostino RB, et al. Variability of body weight and health outcomes in the Framingham population. *N Engl J Med*. 1991;324:1839-1844.
38. Williamson DF, Pamuk E, Thun M, Flanders D, Byers T, Heath C. Prospective study of intentional weight loss and mortality in never-smoking overweight US white women aged 40-64 years. *Am J Epidemiol*. 1995;141:1128-1141.
39. Wadden TA. National Institutes of Health Technology Assessment Conference: treatment of obesity by moderate and severe caloric restriction: results of clinical research trials. *Ann Intern Med*. 1993;119(pt 2):688-693.
40. National Task Force on the Prevention and Treatment of Obesity. Long-term pharmacotherapy in the management of obesity. *JAMA*. 1996;276:1907-1915.

Copyright of American Journal of Public Health is the property of American Public Health Association and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.