Obesity in Adulthood and Its Consequences for Life Expectancy: A Life-Table Analysis

Anna Peeters, PhD; Jan J. Barendregt, PhD; Frans Willekens, PhD; Johan P. Mackenbach, MD, PhD; Abdullah Al Mamun, BSc(Hons), MSc; and Luc Bonneux, MD, PhD, for NEDCOM, the Netherlands Epidemiology and Demography Compression of Morbidity Research Group*

Background: Overweight and obesity in adulthood are linked to an increased risk for death and disease. Their potential effect on life expectancy and premature death has not yet been described.

Objective: To analyze reductions in life expectancy and increases in premature death associated with overweight and obesity at 40 years of age.

Design: Prospective cohort study.

Setting: The Framingham Heart Study with follow-up from 1948 to 1990.

Participants: 3457 Framingham Heart Study participants who were 30 to 49 years of age at baseline.

Measurements: Mortality rates specific for age and body mass index group (normal weight, overweight, or obese at baseline) were derived within sex and smoking status strata. Life expectancy and the probability of death before 70 years of age were analyzed by using life tables.

Results: Large decreases in life expectancy were associated with overweight and obesity. Forty-year-old female nonsmokers lost 3.3 years and 40-year-old male nonsmokers lost 3.1 years of life expectancy because of overweight. Forty-year-old female non-smokers lost 7.1 years and 40-year-old male nonsmokers lost 5.8 years because of obesity. Obese female smokers lost 7.2 years and obese male smokers lost 6.7 years of life expectancy compared with normal-weight smokers. Obese female smokers lost 13.3 years and obese male smokers lost 13.7 years compared with normal-weight nonsmokers. Body mass index at ages 30 to 49 years predicted mortality after ages 50 to 69 years, even after adjustment for body mass index at age 50 to 69 years.

Conclusions: Obesity and overweight in adulthood are associated with large decreases in life expectancy and increases in early mortality. These decreases are similar to those seen with smoking. Obesity in adulthood is a powerful predictor of death at older ages. Because of the increasing prevalence of obesity, more efficient prevention and treatment should become high priorities in public health.

The increasing prevalence of overweight and obesity, coupled with their associations with death, disability, and disease, has led to their identification as a major, potentially preventable cause of premature morbidity and death (1–9). However, it is difficult to estimate the public health impact of overweight and obesity because of complex interactions with age; smoking; and obesity-related risk factors, such as diabetes, hypertension, and lipid disorders (8, 10–12). The observed relationship between body mass index (BMI) and mortality has been described as J-shaped; mortality increases as a result of underweight, overweight, and obesity. However, preexisting illness and inadequate control of smoking may cause at least part of the increased mortality at very low weight (8).

Consequently, there have been no robust estimates of life expectancy lost as a result of obesity. A primary reason is the lack of understanding of probable, healthy, or unhealthy weight trajectories over the life course. Conclusions regarding appropriate weight trajectories between adulthood and older age are complicated by uncertainties about age-appropriate measurements of obesity and the effects of smoking, obesity-associated risk factors for cardiovascular disease, and unintended weight loss (13, 14).

We provide an estimate of the effect of obesity and overweight in adulthood on life expectancy, implicitly taking into account the various possible weight trajectories throughout the life course. We take advantage of the cohort follow-up made available by the Framingham Heart Study to analyze the differences in life course for various BMI groups. We make no assumptions about the relationship between BMI and mortality at older ages. Our primary objective was to analyze the reductions in life expectancy associated with overweight and obesity at 40 years of age.

METHODS

Data Source

The Framingham Heart Study is a longitudinal study with excellent follow-up on mortality. The original study cohort involved 5209 adults, age 28 through 62 years, residing in Framingham, Massachusetts, between 1948 and 1951 (15). To examine the effect of overweight and obesity in adulthood, we used the data from more than 40 years of follow-up (examinations 1 through 21) on age at death for persons 30 through 49 years of age at baseline (*n* = 3607). Height and weight were measured at baseline (7, 15). Smoking status at baseline was defined categorically as self-reported current smoker or nonsmoker. No information was available on smoking status before study entry. Information on all three variables was available for 3582 participants (99%). Because the relationship between weight and mortality is affected by underlying disease (8, 14, 16), we excluded participants who had cardiovascular disease (17)

at baseline or died within 4 years of follow-up (63 participants, including 50 deaths). Because our analysis focused on the risk for death associated with overweight and obesity, we also excluded 62 underweight persons (BMI < 18.5 kg/m^2). The final analyses involved 3457 participants (1550 men and 1907 women).

We analyzed the effect of potential confounders on the relationship between obesity and mortality (6, 8, 18). Of the 3457 participants examined, hypertension and diabetes status was available for all participants, and physical activity level was available for 2893 (84%) participants. Total serum cholesterol level was available for 2127 (62%) participants and was therefore not taken into account. We defined hypertension at baseline as either systolic blood pressure of 160 mm Hg or greater or diastolic blood pressure of 95 mm Hg or greater in two repeated measurements. Physical activity (a continuous index derived from hours of activity and rest) was not available until examination 4 (approximately 8 years after baseline). Level of education at baseline was available for 3350 (97%) participants. Potential confounders were analyzed by using only complete cases.

BMI Group Classification

Body mass index at baseline was calculated as weight in kg/height in m^2. We defined three BMI categories based on World Health Organization guidelines (2): group I (normal weight), BMI of 18.5 to 24.9 kg/m^2; group II (overweight), BMI of 25 to 29.9 kg/m^2; and group III (obese), BMI greater than or equal to 30 kg/m^2 (including 19 people with BMI > 40 kg/m^2).

Survival Analysis

We used S-Plus 2000 (MathSoft, Inc., Seattle, Washington) for all statistical analyses. Survival curves for each BMI group were compared by using Kaplan–Meier plots. We assessed the association between BMI group at baseline and mortality over the 40 years of follow-up by using Cox proportional hazards analysis, with age as the time scale. The effect of BMI was analyzed separately within strata defined by sex and smoking status at baseline. We tested the proportionality of hazards assumption by analysis of the Schoenfeld residuals (19, 20). Statistical significance was set at the 5% level.

Life Course Analysis

Within each stratum, we estimated age-specific mortality rates for each BMI group by using Poisson regression analysis; age at follow-up and BMI group at baseline were categorical variables. Although the hazard ratios estimated for BMI group from this analysis are equivalent to those estimated from the Cox analyses, Poisson regression also optimizes the hazard associated with each age at follow-up. Life tables were derived for each BMI group, representing populations that were 40 years of age and free of cardiovascular disease at study entry. Conversions between mortality rates and probabilities assumed that within each single age interval, the hazard is constant. The life expectancy

Context

Middle-aged adults who are overweight or obese may have shorter life expectancies than normal-weight adults, but how much shorter?

Contribution

This analysis of data from the Framingham Heart Study from 1948 to 1990 showed that, on average, adults who were obese (body mass index [BMI] ≥ 30 kg/m^2) at age 40 years lived 6 to 7 years less than their normal-weight counterparts. Adults who were overweight (BMI, 25 to 29.9 kg/m^2) and did not smoke lived about 3 years less than normal-weight nonsmokers. Adults who were obese and smoked lived 13 to 14 years less than normal-weight nonsmokers.

Cautions

Descriptions of lost life expectancy do not necessarily predict length of life that could be gained from obesity prevention or treatment programs.

–The Editors

at 90 years of age was assumed to be a constant 4.53 for men and 5.05 for women for each BMI group (based on life expectancies of the total Framingham Study sample [21]). The main outcome measure, life expectancy at 40 years of age, was calculated as the mean age at death within a life-table population. Confidence intervals for the life table measures were calculated by using a bootstrap procedure, based on 10 000 replicates. We report the bootstrap bias–correct, adjusted 95% CIs (using the bias-corrected accelerated percentile interval algorithm) (22). Although computationally demanding, the bootstrap procedure is easier than an analytical alternative that includes both the variance of the Poisson model and the variance of the life table.

Role of the Funding Source

The Framingham Heart Study was conducted and supported by the National Heart, Lung, and Blood Institute (NHLBI) in collaboration with the Framingham Heart Study Investigators. The NHLBI reviewed this article for scientific content and consistency of data interpretation with previous Framingham Heart Study publica-

Table 1. Characteristics of Original Framingham Heart Study Participants, Age 30 to 49 Years at Baseline (1948–1951)

BMI Group*	Female Nonsmoker	Female Smoker	Male Nonsmoker	Male Smoker
		n (%)		
Group I	522 (52)	624 (70)	115 (38)	577 (46)
Group II	353 (35)	206 (23)	143 (47)	533 (43)
Group III	136 (13)	66 (7)	44 (15)	138 (11)

* Group I is normal weight, group II is overweight, and group III is obese. BMI = body mass index.

Figure. Kaplan–Meier survival estimates for body mass index (*BMI*) groups I, II, and III within sex and smoking strata.
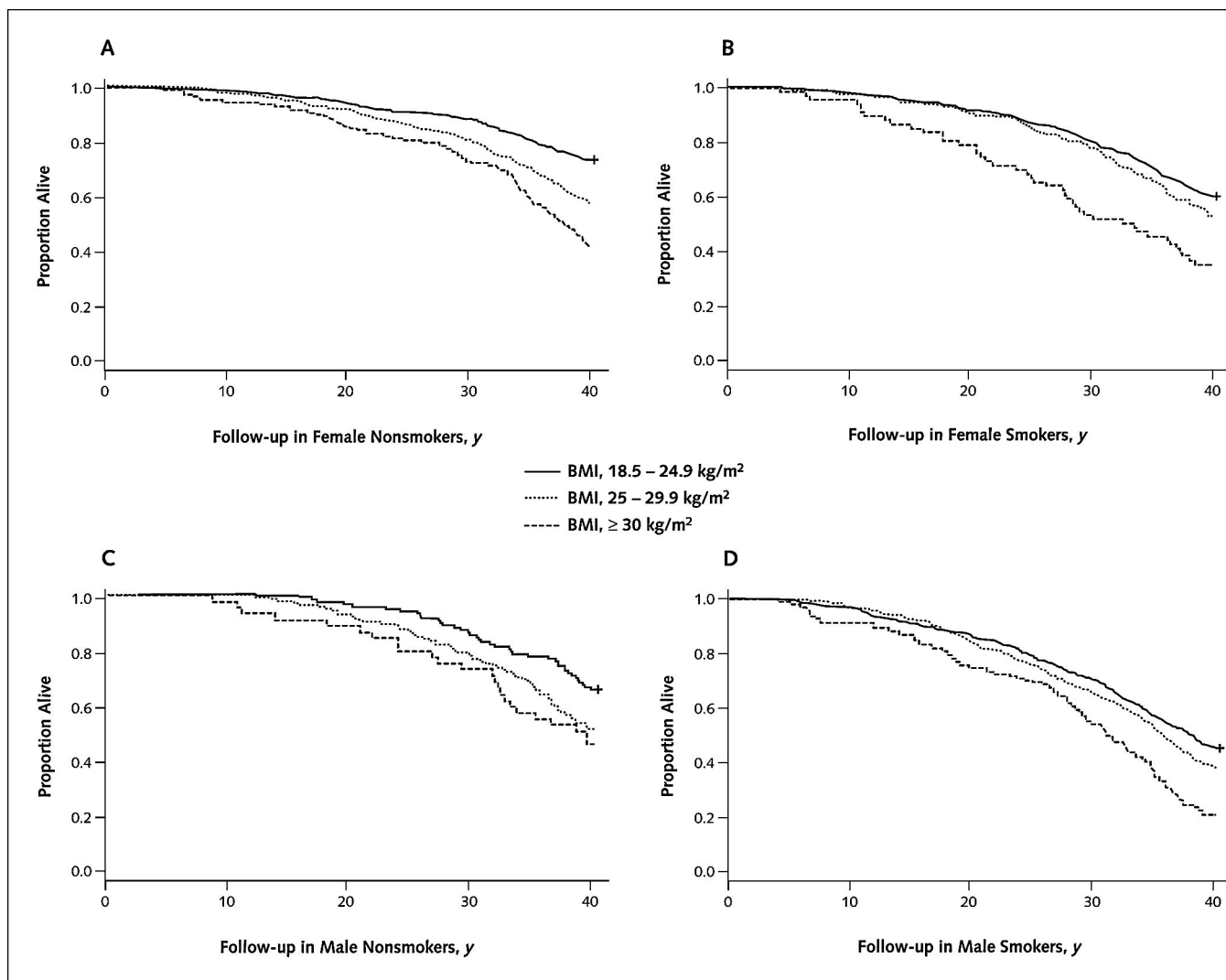
tions; significant comments were incorporated into the text before submission for publication. The NHLBI had no role in the design, conduct, analyses, and reporting of the study or in the decision to submit the manuscript for publication. The Netherlands Heart Foundation and the Netherlands Organization for Scientific Research funded our study. Neither had any role in the design, conduct, analyses and reporting of the study or in the decision to submit the manuscript for publication.

RESULTS

The characteristics at baseline within the Framingham Study cohort were generally as expected: The probability of death increased with each higher category of BMI group, the relationship between the prevalence of smoking and BMI group was inverse (**Table 1**), and age generally increased with each higher category of BMI group (7, 8, 23). Although male nonsmokers were a small group and may represent an unusual cohort for that time, they were analyzed in the same way as the other groups. We did this because male nonsmokers had BMI-related risks similar to those of female nonsmokers and to findings in previous studies examining the relationship between BMI and mortality.

BMI and Survival

With participants categorized by BMI at baseline, we used Cox proportional hazards analysis to determine the relative rate of death over the 40 years of follow-up. We found that sex did not significantly modify the effect of BMI but that smoking status at baseline did, as has been previously described (10, 12, 24, 25). Additional analyses were performed separately for strata defined by sex and smoking status at baseline. The **Figure** illustrates the empirical survival curves for each BMI group within each of the four strata: female nonsmokers, female smokers, male nonsmokers, and male smokers. The survival disadvantage

associated with BMI group II compared with BMI group I is apparently smaller in smokers than in nonsmokers. The hazard ratios for mortality associated with BMI group were generally consistent between strata, although neither male nor female smokers in BMI group II showed an increased mortality risk (**Table 2**). The proportional hazards assumption seemed appropriate for BMI, both by analysis of the Schoenfeld residuals and by comparison of the Cox-derived hazard ratios for two distinct follow-up periods with approximately equal numbers of events (follow-up years 4 to 28 [725 deaths]; and follow-up years 29 to 40 [919 deaths]). For all nonsmokers, sex-adjusted hazard ratios for BMI group II were 1.47 in period 1 (95% CI, 1.06 to 2.04) versus 1.53 in period 2 (CI, 1.19 to 1.97), and sex-adjusted hazard ratios for BMI group III were 2.05 (CI, 1.38 to 3.05) versus 2.27 (CI, 1.67 to 3.1). Hazard ratios in the two periods were similar for each stratum.

Although earlier studies report a lack of risk associated with overweight in smokers (8, 12, 23), this relationship is complicated by the fact that lower weight may reflect longer duration or stronger intensity of smoking (8), which

are themselves risk factors for death. We analyzed the effect of various measures of smoking intensity (including future smoking habits and number of cigarettes smoked per day) and of excluding deaths within the first 20 years of follow-up to account for smokers who have lower weight because of underlying disease. In all analyses, the hazard ratios for BMI group II remained 1. Therefore, why overweight confers an excess risk among nonsmokers but not smokers remains unexplained.

Adjustment for physical activity and education had no effect on the risks associated with BMI in any of the four strata (**Table 2**) (6, 8). As expected (6, 8), adjustment for hypertension and diabetes led to reductions in the mortality hazard ratios for obesity (**Table 2**). In participants without diagnosed hypertension or diabetes at baseline, the mortality hazard ratios for body weight were similar to those of the total study sample (**Table 2**). We performed our primary analyses on the total sample (n = 3457) without further adjustment. Secondary analyses were performed on the 3025 participants without hypertension or diabetes at baseline.

Table 2. **Mortality Hazard Ratios for Body Mass Index Groups II and III Relative to Body Mass Index Group I, Derived from 40 Years of Follow-up and Adjusted for Age***

BMI Group†	Mortality Hazard Ratio (95% CI)			
	Female Nonsmoker	Female Smoker	Male Nonsmoker	Male Smoker
Primary analysis with all participants (n = 3457)				
Group I‡	1.00	1.00	1.00	1.00
Group II	1.51 (1.20–1.91)	1.01 (0.80–1.28)	1.42 (0.96–2.09)	1.14 (0.98–1.33)
Group III	2.27 (1.72–2.99)	2.01 (1.45–2.78)	1.92 (1.16–3.19)	1.92 (1.55–2.39)
Participants without hypertension or diabetes at baseline (n = 3025)				
Group I‡	1.00	1.00	1.00	1.00
Group II	1.38 (0.88–2.14)	1.06 (0.89–1.25)	1.51 (1.17–1.96)	1.02 (0.78–1.32)
Group III	2.20 (1.17–4.14)	1.95 (1.50–2.55)	2.35 (1.69–3.27)	1.84 (1.25–2.69)
All participants after adjustment for hypertension and diabetes at baseline (n = 3457)				
Group I‡	1.00	1.00	1.00	1.00
Group II	1.41 (0.94–2.10)	1.09 (0.93–1.27)	1.48 (1.17–1.86)	0.89 (0.69–1.13)
Group III	1.58 (0.91–2.75)	1.62 (1.28–2.03)	1.95 (1.46–2.61)	1.75 (1.25–2.45)
Participants with physical activity information from examination 4 and education information from baseline examination (n = 2819)				
Group I‡	1.00	1.00	1.00	1.00
Group II	1.56 (1.02–2.39)	1.16 (0.98–1.38)	1.57 (1.21–2.04)	0.99 (0.76–1.31)
Group III	1.86 (1.06–3.29)	1.82 (1.41–2.33)	2.19 (1.60–3.00)	1.83 (1.24–2.71)
Participants with physical activity information from examination 4 and education information from baseline examination after adjustment for physical activity and education (n = 2819)				
Group I‡	1.00	1.00	1.00	1.00
Group II	1.56 (1.02–2.39)	1.15 (0.97–1.37)	1.50 (1.15–1.95)	0.99 (0.76–1.30)
Group III	1.86 (1.06–3.29)	1.79 (1.39–2.30)	2.03 (1.47–2.79)	1.83 (1.23–2.72)

* BMI = body mass index.
† Group I is normal weight, group II is overweight, and group III is obese.
‡ Group I is the reference group.

Table 3. **Effect of Overweight and Obesity in Adulthood on Life Expectancy at 40 Years of Age***

BMI Group†	Life Expectancy or Years Lost (95% CI), y			
	Female Nonsmoker	Female Smoker	Male Nonsmoker	Male Smoker
Primary analysis with all participants (*n* = 3574)				
Life expectancy at 40 years of age				
Group I	46.28 (45.00 to 47.46)	40.21 (38.90 to 41.50)	43.35 (40.82 to 45.96)	36.31 (35.14 to 37.48)
Group II	42.99 (41.50 to 44.39)	40.05 (38.15 to 42.03)	40.30 (38.00 to 42.49)	35.00 (33.85 to 36.15)
Group III	39.19 (36.96 to 41.45)	33.00 (29.75 to 36.85)	37.54 (33.84 to 41.70)	29.65 (27.78 to 31.42)
Years lost from the age of 40 years relative to BMI group I				
Group II	3.29 (1.42 to 5.07)	0.16 (−2.12 to 2.36)	3.05 (−0.36 to 6.39)	1.30 (−0.34 to 2.79)
Group III	7.08 (4.58 to 9.57)	7.21 (3.18 to 10.62)	5.82 (1.36 to 10.53)	6.66 (4.55 to 8.82)
Participants without hypertension or diabetes at baseline (*n* = 3025)				
Life expectancy at 40 years of age				
Group I	46.91 (45.64 to 48.21)	40.85 (39.41 to 42.12)	44.35 (41.64 to 47.08)	36.84 (35.70 to 38.24)
Group II	43.83 (42.12 to 45.22)	40.66 (38.62 to 43.15)	41.72 (39.45 to 44.28)	36.31 (35.09 to 37.61)
Group III	39.90 (37.25 to 42.44)	34.67 (30.98 to 38.43)	37.50 (32.94 to 43.30)	30.21 (27.92 to 32.66)
Years lost from the age of 40 years relative to BMI group I				
Group II	3.08 (1.30 to 5.11)	0.20 (−2.82 to 2.39)	2.63 (−0.92 to 6.06)	1.19 (−1.10 to 2.26)
Group III	7.01 (4.29 to 9.98)	6.18 (2.34 to 10.20)	6.85 (0.36 to 9.98)	6.63 (4.01 to 9.37)

* All results are from life tables representing cohorts initially 40 years of age and free of cardiovascular disease. BMI = body mass index.
† Group I is normal weight, group II is overweight, and group III is obese.

BMI, Life Expectancy, and Premature Mortality

We calculated the implications of the increases in risk for death on the life course of 40-year-old participants, classified as normal weight, overweight, or obese, and determined their differences in life expectancy and premature mortality. We focus on nonsmokers first. Forty-year-old women and men in BMI group III lost 7.1 and 5.8 years of life, respectively, compared with 40-year-old nonsmoking women and men in BMI group I (**Table 3**). In addition, 40-year-old nonsmoking women and men in BMI group II lost 3.3 and 3.1 years of life, respectively, compared with nonsmoking women and men in BMI group I, although the latter result was not statistically significant (**Table 3**). We observed similar differences in life expectancy in the participants who did not have hypertension or diabetes at baseline (**Table 3**).

The decreases in life expectancy were reflected in increased probabilities of premature death (defined as death before the age of 70 years) (**Table 4**). Compared with BMI group I, BMI group II had 4 to 5 more deaths/ 100 persons and BMI group III had 10 to 11 more deaths/ 100 persons between 40 and 70 years of age. This represents a 115% (women) and 81% (men) increased risk for premature death in BMI group III (**Table 4**). We observed increases of a similar magnitude in the participants without hypertension or diabetes at baseline (**Table 4**).

Compared with smokers in BMI group I, smokers in BMI group II had no significant loss of life expectancy (**Table 3**). However, female and male smokers in BMI group III had a large decrease in life expectancy compared with normal-weight smokers: 7.2 and 6.7 years, respectively (**Table 3**).

Although smoking status at baseline was not our pri-mary analysis, the difference in life expectancy between smokers and nonsmokers at 40 years of age ranged from 5 to 7 years; our findings are similar to those previously reported (26) (**Table 3**).

Effect of BMI in Adulthood Independent of Future BMI

Overweight and obesity identified in adulthood predict substantial decreases in life expectancy; however, these decreases may be unrelated to the effect of BMI in adulthood. Rather, they may be the result of the independent effects of overweight and obesity later in life. We analyzed whether BMI in adulthood has an effect on mortality independent of BMI later in life. The survival patterns presented in the **Figure** and the analysis of the mortality risks within two distinct follow-up periods suggest that obesity in adulthood exerts an immediate effect on mortality.

We also analyzed the patterns of change in BMI and BMI group over 20 years of follow-up. Of the participants who had BMI and smoking status measured at examination 10 (20 years after entry to the study, representing 64% of those still alive), 1977 survived for at least 4 years after this examination. Although most persons in each BMI group were in the same BMI group 20 years earlier, a substantial proportion of persons in BMI groups II and III were in a lower BMI group 20 years earlier (**Table 5**). The BMI at age 30 to 49 years was a significant predictor of death from age 50 to 69 years, independent of BMI group at 50 to 69 years of age. For persons in BMI group II or III at age 50 to 69 years, the risk for death was significantly higher among those who had been in BMI group III at 30 to 49 years of age (**Table 5**). Significant weight gain or loss may be worse for health than is a consistent weight

throughout adulthood, but the numbers in our study are too small to allow valid interpretation.

With BMI as a continuous variable, BMI at age 30 to 49 years was associated with a hazard ratio of 1.05 (CI, 1.02 to 1.09) for mortality from age 50 to 69 years, after adjustment for BMI at age 50 to 69 years, sex, age, and smoking status. In this analysis, the mortality hazard ratio associated with BMI at age 50 to 69 years was 1.00 (CI, 0.97 to 1.03). This finding is in contrast to the mortality hazard ratio of 1.04 (CI, 1.02 to 1.06) associated with BMI at age 50 to 69 years in an analysis adjusting only for sex, age, and smoking status.

DISCUSSION

The effect of overweight and obesity in adulthood on life expectancy and premature death is striking. Among 40-year-old nonsmokers without previously diagnosed cardiovascular disease, overweight was associated with a 3-year decrease in life expectancy; obesity was associated with a 7-year decrease for women and 6-year decrease for men. These decreases were based on comparisons with persons of normal weight at age 40 years. Because the increased mortality risk is spread across all ages, there are also large increases in premature mortality; compared with persons in the normal-weight range, obese women were 115% more likely to die before age 70 years and obese men were 81%

more likely to die before age 70 years. Our study did not analyze underweight persons (BMI < 18.5 kg/m^2).

Obese smokers had a similar decrease in life expectancy: They lost 7 more years of life than smokers of normal weight. The double burden of obesity and smoking resulted in obese female smokers' losing 13 years and obese male smokers' losing 14 years compared with nonsmokers of normal weight. Although overweight (BMI, 25 to 29.9 kg/m^2) male smokers lost approximately 1 year of life expectancy, overweight female smokers showed no survival disadvantage compared with smokers of normal weight. Studies differ on the effects of smoking on the relationship between body weight and mortality (12, 27, 28). In our sample, the interaction between smoking status and the effect of overweight on mortality was significant. We could not explain this finding, even after accounting for future smoking habits, numbers of cigarettes smoked, and medium-term mortality.

Our primary analyses stratified by sex and smoking status and adjusted only for age. Adjustment for education and physical activity did not change the relative risks associated with overweight and obesity. The results of our primary analyses do not differ greatly from those obtained after analysis of only participants without hypertension or diabetes at baseline. We argue against adjusting for such factors as hypertension and diabetes because they are

Table 4. Effect of Overweight and Obesity in Adulthood on Premature Death between 40 and 70 Years of Age*

BMI Group†	Female Nonsmoker	Female Smoker	Male Nonsmoker	Male Smoker
Primary analysis with all participants (*n* = 3574)				
Percentage mortality between 40 and 70 years of age (95% CI), %				
Group I	9.36 (7.56–11.59)	18.72 (16.10–21.74)	12.62 (8.73–17.75)	26.72 (23.58–29.73)
Group II	13.85 (11.18–16.73)	19.02 (15.50–23.28)	17.43 (12.74–23.07)	29.74 (26.53–33.03)
Group III	20.09 (15.34–24.85)	34.35 (24.90–43.84)	22.86 (14.31–32.91)	45.03 (39.11–51.57)
Number of excess deaths/100 population relative to BMI group I, *n*				
Group II	4.49	0.30	4.81	3.02
Group III	10.73	15.63	10.24	18.31
Percentage increased risk for premature death relative to BMI group I, %				
Group II	48.04	1.62	38.15	11.32
Group III	114.74	83.51	81.17	68.54
Participants without hypertension or diabetes at baseline (*n* = 3025)				
Percentage mortality between 40 and 70 years of age (95% CI), %				
Group I	7.99 (6.17–10.09)	18.02 (15.35–20.98)	11.42 (7.33–16.85)	24.68 (21.37–27.71)
Group II	11.86 (9.45–15.15)	18.36 (14.19–22.8)	15.42 (10.91–21.47)	25.85 (22.49–28.98)
Group III	17.82 (13.31–23.64)	30.61 (21.94–40.96)	23.37 (12.37–37.12)	42.51 (34.69–50.79)
Number of excess deaths/100 population relative to BMI group I, *n*				
Group II	3.86	0.34	4.00	1.17
Group III	9.83	12.59	11.95	17.83
Percentage increased risk for premature death relative to BMI group I, %				
Group II	48.31	1.89	35.01	4.74
Group III	122.94	69.88	104.60	72.22

* All results are from life tables representing cohorts initially aged 40 years and free of cardiovascular disease. BMI = body mass index.
† Group I is normal weight, group II is overweight, and group III is obese.

Table 5. **Independent Effects of Body Mass Index in Adulthood and in Older Age on Mortality***

BMI Group at Baseline†	BMI Group at 20 Years after Baseline		
	Group I	Group II	Group III
Proportional distribution (number) of survivors at 20 years after baseline by BMI group at baseline, % (n)			
Group I	89 (695)	41 (352)	5 (16)
Group II	11 (84)	54 (467)	47 (160)
Group III	0.4 (3)	4 (38)	48 (162)
Hazard ratios (95% CIs) from regression on 20-year mortality of BMI group trajectory (the combination of BMI group at baseline and 20 years later)‡			
Group I	1	1.08 (0.86–1.37)	1.36 (0.51–3.68)
Group II	1.40 (1.00–1.97)	1.16 (0.94–1.42)	1.46 (1.10–1.94)
Group III	NA	2.10 (1.39–3.20)	2.00 (1.54–2.58)

* BMI = body mass index; NA = not applicable.
† Group I is normal weight, group II is overweight, and group III is obese.
‡ Adjusted for sex, age, and smoking status at 20 years after baseline. The reference category is those participants who are in BMI group I at baseline and 20 years after baseline. Participants who died within the first 4 years of survival time were excluded.

downstream physiologic effects of obesity (8, 29). The decrease in mortality risk associated with obesity after adjustment for hypertension and diabetes may reflect true confounding, the increased mortality risk of those with more severe obesity (associated with early development of hypertension or diabetes), or a combination of both. Furthermore, if future risk profiles are related to the association between obesity and mortality, adjustment for any of these during follow-up would bias assessment of the true mortality risk associated with obesity.

A high degree of correlation of BMI measured throughout adult life (30) and the uncertain long-term effects of weight loss interventions (2) support the rationale for identifying BMI in adulthood as a target for prevention. Our study confirms that the relative risk for death associated with increased BMI in adulthood remains relatively constant throughout life (12, 31). We also showed that overweight and obesity in adulthood are strong predictors of death after taking into account future BMI (6). As a result, overweight and obesity are even more important targets. Our results strongly support prevention as a first line of public health action (14, 32, 33).

Our study has several advantages when compared with other studies. In contrast to a previous study (34), we used observed mortality follow-up of all causes for people with BMI classified in adulthood. Differences in life expectancy were not as great among BMI groups in this earlier theoretical study, probably because the researchers limited the consequences of obesity to only cardiovascular disease and diabetes and kept BMI constant during the life course (34). Our study also benefits from use of the Framingham Study data set. The Framingham Study provides excellent long-term follow-up on mortality and provides independently determined data on height and weight. Without such an extended follow-up period, it is not possible to empirically analyze the long-term effects of overweight and obesity in adulthood.

The most important outstanding question is to what extent the observed association between obesity and decreased life expectancy is causal and applicable to today's populations. The risks associated with obesity that were defined 50 years ago may not be the same as those defined today because of differences in the obese population and treatment. However, the hazard ratios reported here are consistent with those derived from more recent studies (6, 8, 10, 12). For example, the first 24 years of follow-up in the Framingham Study were associated with hazard ratios of 1.4 for overweight in female nonsmokers and 1.8 for obesity in female nonsmokers. The Nurses' Health Study, which also analyzes younger women (age 30 to 55 years), has followed participants since 1976 and reported hazard ratios of 1.3 to 1.6 for overweight in healthy female nonsmokers and 2.1 to 2.2 for obesity in healthy female nonsmokers (8).

Mortality rates have improved greatly over the last 50 years. However, the Framingham Heart Study cohort is relatively healthy, and its life expectancy is similar to that of the 1990 Massachusetts population (21, 35). After each age-specific mortality rate is reduced by 30% (approximating the reductions in mortality between 1970 and 2000), the loss of life expectancy associated with obesity decreased only very slightly (data not shown). Although these results will not represent the absolute life expectancy of today's populations, they are a robust estimation of the relative magnitude of life expectancy lost due to increasing body weight.

The major limitation of our study is that we could not identify what proportion of the described loss of life expectancy is a direct consequence of obesity and would therefore be prevented through obesity prevention. Obesity clusters with other mortality risk factors, such as low physical activity levels, diabetes, hypertension, and high lipid levels. Although we accounted for some of these factors at baseline, physical activity level was not available until 8

years after the baseline examination and lipid levels were not available in a sufficient proportion of the population. It would be of interest to analyze the effects of obesity in young adults still free of other related risk factors. In addition, although many of these variables were available during follow-up, our study did not have enough power to analyze mortality differences associated with different trajectories of BMI and related risk factors. Future studies should determine whether these large survival deficits are associated with specific, preventable risk factor profiles or can be avoided if an "optimal" weight trajectory is maintained throughout life.

We conclude that obesity in adulthood is associated with a decrease in life expectancy of about 7 years, both in men and women, smokers and nonsmokers. The magnitude of this loss is similar to that associated with smoking. As with smoking, obesity caused increased mortality decades after onset. In the Framingham Study cohort, overweight and obesity in middle age decrease the life expectancy of nonsmokers by approximately 2 years. We expect the potential impact on today's populations to be much greater because obesity is twice as prevalent in U.S. adults now as it was in the Framingham Study (1, 2).

The smoking epidemic in the western world is waning; however, a new fear should be the increasing prevalence of overweight and obesity in young adults, which heralds another potentially preventable public health disaster. This time, we must pay attention earlier and firmly establish research for more effective prevention and treatment as top priorities in public health.

APPENDIX: MEMBERS OF THE NETHERLANDS EPIDEMIOLOGY AND DEMOGRAPHY COMPRESSION OF MORBIDITY RESEARCH GROUP

Anna Peeters, PhD, Erasmus MC (Rotterdam, the Netherlands); Jan J. Barendregt, PhD, Erasmus MC (Rotterdam, the Netherlands); Frans Willekens, PhD, University of Groningen (Groningen, the Netherlands); Johan P. Mackenbach, MD, PhD, Erasmus MC (Rotterdam, the Netherlands); Abdullah Al Mamun, BSc(Hons), MSc, University of Groningen (Groningen, the Netherlands); Luc Bonneux, MD, PhD, Erasmus MC (Rotterdam, the Netherlands); Fanny Janssen, MSc, Erasmus MC (Rotterdam, the Netherlands); Anton Kunst, PhD, Erasmus MC (Rotterdam, the Netherlands); and Wilma Nusselder, PhD, Erasmus MC (Rotterdam, the Netherlands).

From Erasmus MC, Rotterdam, and University of Groningen, Groningen, the Netherlands.

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Current Author Addresses: Drs. Peeters, Barendregt, Mackenbach, and Bonneux: Department of Public Health, Erasmus MC, PO Box 1738, 3000 DR Rotterdam, the Netherlands.
Drs. Willekens and Al Mamun: Population Research Center, University of Groningen, PO Box 800, 9700 AV Groningen, the Netherlands.

Author Contributions: Conception and design: A. Peeters, J.P. Mackenbach, L. Bonneux.
Analysis and interpretation of the data: A. Peeters, J.J. Barendregt, J.P. Mackenbach, A. Al Mamun, L. Bonneux.

Drafting of the article: A. Peeters, L. Bonneux.
Critical revision of the article for important intellectual content: A. Peeters, J.J. Barendregt, J.P. Mackenbach, L. Bonneux.
Final approval of the article: A. Peeters, J.J. Barendregt, J.P. Mackenbach, L. Bonneux.
Provision of study materials or patients: A. Peeters.
Statistical expertise: J.J. Barendregt, F. Willekens, A. Al Mamun.
Obtaining of funding: J.J. Barendregt, J.P. Mackenbach, L. Bonneux.