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MORTALITY

The burden of mortality of obesity at middle and old age is small. A life table analysis of the US Health and Retirement Survey

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Abstract The evidence of effect of overweight and obesity on mortality at middle and old age is conflicting. The increased relative risk of cardiovascular disease and diabetes for overweight and obese individuals compared to normal weight is well documented, but the absolute risk of cardiovascular death has decreased spectacularly since the 1980s. We estimate the burden of mortality of obesity among middle and old aged adults in the Health and Retirement Survey (HRS), a US prospective longitudinal study. We calculate univariate and multivariate age-specific probabilities and proportional hazard ratios of death in relation to self-reported body mass index (BMI), smoking and education. The life table translates age specific adjusted event rates in survival times, dependent on risk factor distributions (smoking, levels of education and self reported BMI). 95% confidence intervals are calculated by bootstrapping. The highest life expectancy at age 55 was found in overweight (BMI 25–29.9), highly educated non smokers: 30.7 (29.5–31.9) years (men) and 33.2 (32.1–34.3) (women), slightly higher than a BMI 23–24.9 in both sexes. Smoking decreased the population life expectancy with 3.5 (2.7–4.4) years (men) and 1.8 (1.0–2.5) years (women). Less than optimal education cost men and women respectively 2.8 (2.1–3.6) and 2.6 (1.6–3.6) years. Obesity and low normal weight decreased population life expectancy respectively by 0.8 (0.2–1.3) and 0.8 (0.0–1.5) years for men and women in a contemporary, US population. The burden of mortality of obesity is limited, compared to smoking and low education.

Keywords Obesity · Burden of mortality · Body-Mass Index · Life expectancy · Behavioral risk factors

Abbreviations

BMI	Body mass index
HRS	Health and Retirement Survey
CI	Confidence interval
AHEAD	Asset and Health Dynamics Among the Oldest Old

Introduction

In 2004, Mokdad et al. [1] calculated that obesity was overtaking smoking as actual cause of death. Equating the health risks of smoking with those of obesity struck the imagination [2, 3]. Subsequent corrections were major: the original 365,000 attributable deaths came down to 112,000, the ranking of obesity as actual cause of death dropped from the 2nd to 7th place [1, 4]. What happened to obesity?

We documented high mortality among obese and overweight persons in the Framingham Heart Study (FHS) before [5]. However, the FHS describes the life history of a cohort born between 1900 and 1920, alive at 1948–50 and followed up throughout the second half of the twentieth century. The cohort lived throughout the coronary heart disease epidemic, reaching its peak at the end of the 1960s [6, 7]. Since then, cardiovascular disease death rates have been declining considerably [8]. Cardiovascular disease with coronary heart disease is by far the most important cause of excess death in obesity [9]. These secular changes in cardiovascular mortality might explain the bewildering

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range of results linking BMI to mortality, even in the same age groups [9].

We studied the mortality experience of a recent cohort, the combined HRS and AHEAD studies which began in 1992 and 1993, respectively, and were merged in 1998. The design is comparable to the more recent Survey of Health, Ageing and Retirement in Europe (SHARE), allowing for later international comparisons [10]. We described the burden of mortality of overweight, obesity, smoking and education with life tables. Life tables translate age specific death rates in life expectancies, taking into account competing mortality risks.

Data and methods

Data

We used the RAND Health and Retirement Survey (HRS) data file containing the HRS and the Asset and Health Dynamics Among the Oldest Old (AHEAD) [11]. More information is available elsewhere (http://hrsonline.isr.umich.edu/). The HRS and AHEAD surveys include a nationally representative sample of initially non-institutionalized persons born in 1931–1941 (HRS, aged 51–61 in 1992) and in 1923 or earlier (AHEAD, aged 70 and older in 1993). Sampled persons were re-interviewed over two years. Response was on average 86% (HRS) and 90% (AHEAD). We selected white non-Hispanic men and women of whom date of birth, gender, level of education, Body Mass Index (BMI), and smoking status was available. Data on vital status and month and year of death are obtained through the mortality register (the National Death Index) and through HRS answers. The sample includes surveyrounds from 1992 to 2002, covering 6 waves.

BMI, weight loss and self-report of health

Body Mass Index (BMI in kg/m^2) is determined by self-reports of height and weight at the first contact. We define the BMI groups according to the classification of the World Health Organization (www.who.int/bmi): underweight, BMI smaller than 18.5, normal weight, BMI of 18.5–24.9, overweight, BMI of 25–29.9, obese, BMI of 30–34.9 and severely obese, BMI greater than or equal to 35. Because our analyses showed heterogeneity among the normal weight individuals, we split this group into low normal weight with BMI of 18.5–22.9 and normal weight with BMI of 23–24.9. Because weight loss at older ages can be an indication of deteriorating health and increased risk of dying we exclude persons with BMI less than 18.5 and use for every individual the BMI value that was reported earliest in the survey period. We start counting exposures and events after three years follow up. Respondent's self-reported general health status is used to control for morbidity at baseline, ranging from 1 for excellent to 5 for poor.

Smoking status and educational attainment

Smoking status is divided into three groups: never smokers, past smokers and current smokers. We use the oldest available information on smoking status. We distinguish three groups of educational attainment: Less than high-school or General Educational Development (GED), High-School graduate and College graduate and above.

Methods

We estimated the hazard of mortality by age for males and females and for each determinant of interest. Since individuals may enter and leave observation anytime during the survey, left-truncation is taken into account and we used the Nelson–Aalen estimator to determine the mortality hazard rather than the more common Kaplan–Meier estimator. The Nelson–Aalen estimator determines the cumulative hazard whenever an event (death) occurs [12]. Age, education and smoking adjusted mortality rates by BMI were estimated using Poisson (loglinear regression).

Next we estimated Cox proportional hazard ratios, comparable to relative risks of death by BMI, smoking and education. Single year age is used as the timescale for the baseline hazard instead of time, accounting for left truncation and right censoring. Schoenfeld residuals with significance level set at 5% tested the proportionality assumption [13]. Because the mortality hazard for women was not proportional over age, we divided the data in groups of under age 80 and 80 and over.

Mortality rates are translated in annual probabilities using the Poisson assumption which define stratified life tables calculating life expectancy (e_x) at age $x \geq 55$. Confidence intervals for the life expectancies and differences in life expectancies were calculated using bootstrapping with 1000 replicates. To check the confidence intervals we also applied Chiang's analytical method using the rule of variance of a linear function [14], which gave practically the same results.

To assess the effect of a determinant as a cause of loss of life in a population we multiply the age-specific baseline hazard by the relative risk estimated by the Cox model, weighted by the actual or hypothetical proportions of the population [15]. This is a counterfactual thought experiment comparable to the calculation of population attributable risk. However, the life table takes into account age at death and

recalculates changes in mortality as changes in life expectancy. Interactions between BMI and smoking are only significant for males aged 80 and over. Since this group contains rather few individuals and interactions have no effect on other variables and did not improve the model, we did not include interactions in the model.

Results

The final sample describes 16192 non-Hispanic white persons aged 55 and over. Table 1 gives the characteristics of the sample population. Persons were observed between May 1995 and December 2002. About 2,354 individuals died during the period under survey. The mean follow-up was 7.8 years (range 0–10.8 years), including the three first years to avoid weight loss caused by fatal disease. Figure 1 shows the age, smoking and education adjusted mortality by BMI. Variation in all cause mortality was small between BMI 23 and 36 among men and between BMI 20 and 33 among women.

The life expectancy at age 55 based on observed age specific death rates for males and females is respectively 23.7 (95% confidence interval 23.2–24.1) and 28.1 (27.6–28.6) (see Table 2, univariate analysis). The actual life expectancy of the white American population in 1997 was 23.6 for men and 27.7 for women. After adjusting for smoking and education (see Table 2, multivariate analysis), overweight men lived 0.6 years longer (0.0–1.2) than normal weight men (BMI 23–24.9). There were no statistically or clinically significant differences in survival of

Table 1 Sample population by sex, BMI, smoking status, education and age at baseline

	Males		Females		Total	
Total sample	7281	100.0%	8911	100.0%	16192	100.0%
Low normal weight	912	12.5%	2515	28.2%	3427	21.2%
Normal weight	1361	18.7%	1684	18.9%	3045	18.8%
Overweight	3621	49.7%	2997	33.6%	6618	40.9%
Obese	1095	15.0%	1179	13.2%	2274	14.0%
Severely obese	292	4.0%	536	6.0%	828	5.1%
Never smoked	1881	25.8%	4608	51.7%	6489	40.1%
Stopped smoking	3893	53.5%	2686	30.1%	6579	40.6%
Currently smoking	1507	20.7%	1617	18.1%	3124	19.3%
Low education	2016	27.7%	2348	26.3%	4364	27.0%
Medium education	3536	48.6%	5288	59.3%	8824	54.5%
High education	1729	23.7%	1275	14.3%	3004	18.6%
<65	3931	54.0%	4559	51.2%	8490	52.4%
65–74	1375	18.9%	1514	17.0%	2889	17.8%
75–84	1617	22.2%	2146	24.1%	3763	23.2%
85+	358	4.9%	692	7.8%	1050	6.5%

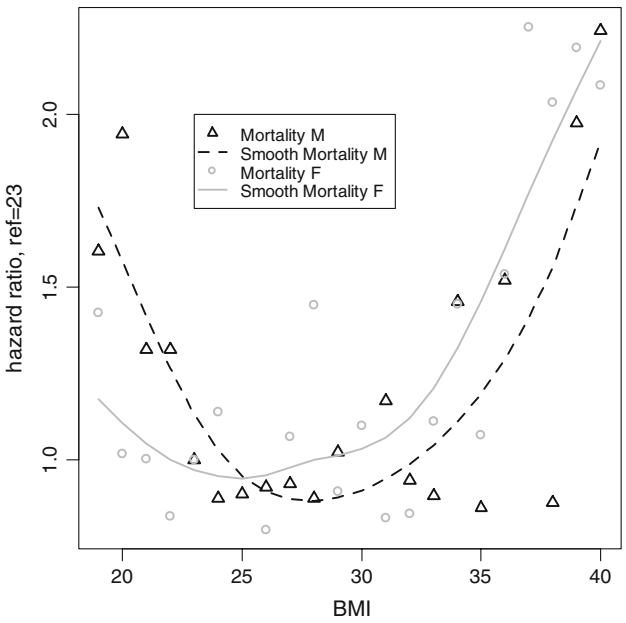

Fig. 1 Age, smoking and education standardized proportional mortality hazard ratios by rounded BMI for ages 55–80 (lines are discrete splines weighted by standard errors)

overweight women or obese men and women, relative to a high normal weight (BMI 23–24.9). However, severe obesity did shorten life of men with 3.0 years (2.2–3.8) and of women with 5.2 years (4.4–6.1) compared to normal weight. Low normal weight (BMI 18.5–22.9) cost 2.4 years for males compared to normal weight (1.8–3.0). Stratifying for smoking status shows that the increased hazard of death of low normal weight is related to current or former smoking, but not non-smoking.

After adjusting for BMI and education, male and female smokers lived on average 8.2 years (7.3–9.0) and 7.5 years (6.8–8.2) shorter, while quitters lived respectively 3.8 years (2.9–4.6) and 2.3 years (1.6–3.1) years shorter. Highly educated men lived 3.3 years (2.5–4.0) (moderate education) and 4.5 years (3.8–5.3) (low education) longer, highly educated women lived respectively 2.4 (1.4–3.5) and 4.6 (3.6–5.6) years longer. The optimal lifestyle in terms of life expectancy is lived by overweight, highly educated persons who never smoked. Figure 2 compares the observed life table and the multivariate model of this optimal lifestyle, showing a median survival at age 55 of 32.1 and 34.3 years, which is a total life expectancy of 87.1 and 89.3 for men and women. The multivariate Cox proportional hazards model (Table 3), adjusting for smoking and levels of education confirms the univariate life table findings.

To limit the possibility of reverse causation (people with low weight because of existing disease), we studied weight loss. 1284 people lost 10% or more weight in less than three waves (or on average in less than 6 years), which conferred a hazard ratio of death equal for both sexes, 2.6

Table 2 Life expectancy at age 55 by life style determinants using stratified univariate analysis and multivariate model (95% confidence limits between parentheses)

	Univariate stratified		Multivariate modeled	
	Males	Females	Males	Females
Total population	23.7 (23.2–24.1)	28.1 (27.6–28.6)	24.2 (23.9–24.6)	28.6 (28.1–29.1)
Low normal weight	20.9 (19.5–22.4)*	28.2 (27.1–29.2)	21.9 (21.5–22.4)*	28.6 (28.1–29.2)
Normal BMI[a]	23.7 (22.5–24.9)	28.5 (27.2–29.8)	24.4 (24.0–24.8)	28.7 (28.1–29.4)
Overweight	24.6 (23.8–25.4)	28.8 (27.7–30.0)	25.0 (24.5–25.4)	29.4 (28.7–30.0)
Obese	24.0 (22.2–25.8)	28.5 (26.9–30.1)	24.3 (23.7–24.9)	28.6 (27.8–29.4)
Severely obese	21.0 (18.9–23.1)*	23.1 (21.1–25.0)*	21.4 (20.7–22.0)*	23.5 (23.0–24.1)*
Never smoking[a]	28.2 (27.1–29.2)	30.5 (29.8–31.3)	27.8 (27.0–28.5)	30.4 (29.8–30.9)
Stopped smoking	24.2 (23.4–25.0)*	27.8 (26.7–28.9)*	24.0 (23.6–24.3)*	28.0 (27.5–28.6)*
Current smoker	18.7 (17.6–19.8)*	22.4 (21.2–23.8)*	19.6 (19.3–19.9)*	22.9 (22.5–23.3)*
Low education	21.2 (20.2–22.2)*	25.6 (24.3–26.9)*	22.5 (22.2–22.9)*	26.6 (26.2–27.1)*
Medium education	23.5 (22.7–24.3)*	28.7 (27.7–29.6)*	23.8 (23.4–24.2)*	28.7 (28.2–29.3)*
High education[a]	27.5 (26.2–28.8)	31.0 (29.4–32.6)	27.0 (26.4–27.7)	31.2 (30.3–32.1)
Overweight, highly educated non-smoker	29.5 (26.4–32.7)	33.5 (30.3–36.8)	30.7 (29.5–31.9)	33.2 (32.1–34.3)

Differences are caused by confounding and interaction

[a] Reference category

* $P < 0.05$ compared to reference category

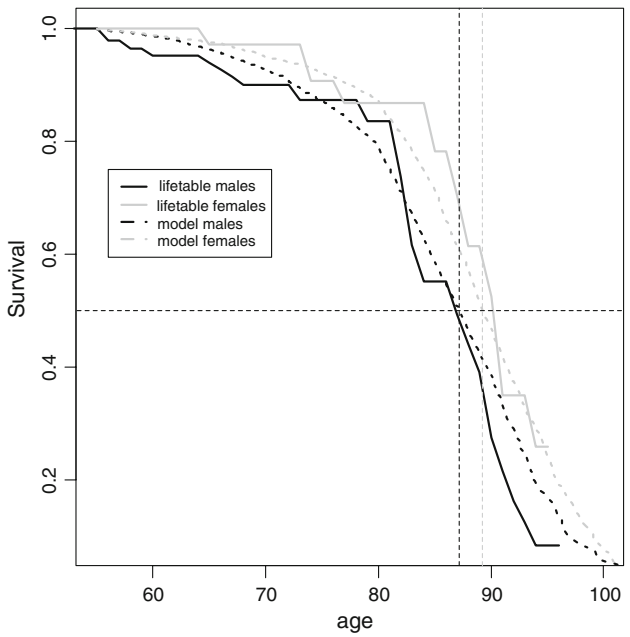

Fig. 2 Survival function and median age at death (for 55+) of the multivariate model and the actual lifetable for the overweight, non-smoking, highly educated population

(2.1–3.2) (between 2 waves or 4 years) and 1.9 (1.6–2.2) (between 3 waves or 6 years). However 2554 people, men and women losing weight over 3 waves or more (>6 years) saw their hazard of death lowered: a PHR of 0.6 (0.5–0.7). We modeled the proportional hazards after excluding those who lost weight, but the changes in hazard ratios were minor. Waiting five years instead of three before counting events did not change the results, except for lowering the statistical power. Waiting three years does not exclude reverse causation entirely but limits it sufficiently.

Loss or gain in life expectancy

The multivariate model of Table 2 can also be interpreted as a counterfactual experiment measuring the life expectancy if the whole imaginary cohort was obese, highly educated, etc. This can be compared to the modeled average population life expectancy. If everybody would be overweight, non smoker and highly educated, the population life expectancy would be 6.5 (5.2–7.7) years (men) and 4.6 (3.4–5.8 years) (women) higher. If nobody would have ever smoked, population life expectancy would be 3.5 (2.7–4.4) years (men) and 1.8 (1.0–2.5) years (women) higher. Less than optimal education cost men and women respectively 2.8 (2.1–3.6) and 2.6 (1.6–3.6) years (considering low education causal and ignoring selection that might lead to lower levels of education). If everybody would have been overweight instead of (morbid) obese or (low) normal weight, population life expectancy would increase with 0.8 (0.2–1.3) for males and 0.8 (0.0–1.5) year for females. Figure 3 is comparable to calculations of attributable risks but shows losses or gains in life expectancy, attributable to the selected risk factors. Losses or gains in life expectancy by each risk factor are weighted by the prevalence of that risk factor in the population illustrating the importance of the contributions of risk factors to

Table 3 Cox proportional hazard ratios (age under and over 80) with 95% confidence intervals

	Males						Females					
	55–80			80+			55–80			80+		
	CI											
Model 1												
Low normal weight	1.59	1.25	2.02	1.14	0.90	1.44	1.05	0.80	1.39	0.99	0.82	1.20
Normal weight	1.00			1.00			1.00			1.00		
Overweight	0.95	0.78	1.16	0.88	0.71	1.08	1.05	0.81	1.37	0.79	0.65	0.96
Obese	1.05	0.82	1.36	0.88	0.62	1.25	1.02	0.74	1.42	0.94	0.71	1.24
Severely obese	1.42	0.98	2.04	1.37	0.70	2.69	1.94	1.36	2.76	1.84	1.26	2.68
Model 2												
Low normal weight	1.42	1.11	1.81	1.1	0.87	1.39	1.01	0.77	1.34	1.01	0.84	1.34
Normal weight	1.00			1.00			1.00			1.00		
Overweight	0.96	0.79	1.18	0.86	0.70	1.06	1.03	0.79	1.34	0.80	0.66	0.98
Obese	1.08	0.83	1.39	0.85	0.60	1.21	1.07	0.77	1.49	0.95	0.72	1.26
Severely obese	1.47	1.02	2.12	1.31	0.67	2.58	1.95	1.36	2.79	1.80	1.23	2.64
Never smoked	1.00			1.00			1.00			1.00		
Stopped smoking	1.77	1.41	2.22	1.28	1.05	1.56	1.47	1.18	1.84	1.20	1.02	1.41
Currently smoking	3.23	2.53	4.12	1.45	1.07	1.96	2.86	2.28	3.58	1.98	1.52	2.58
High education	1.00			1.00			1.00			1.00		
Low education	1.89	1.49	2.39	1.37	1.07	1.75	2.53	1.71	3.76	1.18	0.92	1.52
Medium education	1.69	1.35	2.11	1.07	0.83	1.38	1.99	1.36	2.91	0.91	0.71	1.18

Model 1 describes the association of BMI with mortality

Model 2 adjusts for smoking and education

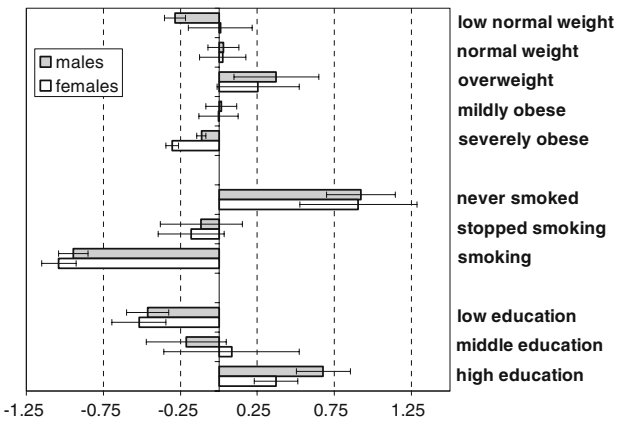

Fig. 3 Losses or gains in average population life expectancy attributed to risk factors, adjusted for each other (95% confidence limits)

the total population life expectancy. Obesity costs severely obese men 2.8 years of life, but because of low prevalence (4%), severe obesity contributes only −0.1 years to life expectancy on population level.

Discussion

The HRS confirms findings from other studies: being overweight at middle and older ages does not increase or even decreases the risk to die [4, 9, 16]. Only higher levels of BMI increase mortality, which implies that the burden of mortality of obesity is limited even in present day US. In the HRS study, the burden of mortality of overweight was not higher than of a BMI between 23 and 24.9 and even mild obesity did not show excess mortality. Low normal weight on the other hand did increase the hazard rate significantly for men. The burden of past and current smoking is far higher, decreasing total life expectancy with 3.5 (men) and 1.7 years (women) in the total population. Poor education also has a larger impact on mortality than body weight. Only severe obesity knows a high mortality. These results, indicating lower mortality at high normal BMI and overweight and higher mortality at low normal weights are in line with many other studies [17].

Limitations

The HRS is a general purpose study of health and retirement. We have no data on the effect of increased BMI's at younger ages, but people die rarely at younger ages. On the other hand, increasing prevalence of obesity in the future might be successfully tackled by innovative technology. The strength and weakness or our study is the recent but short period of time, namely 10 years in the 1990s. The short period makes it difficult to make statements about the

future life course. On the other hand, the recent observation window gives a reliable picture of recent mortality, not of mortality in the past fifty years.

The HRS contains self-reported BMI. Measured BMI tends to be underreported by self report, especially for women with a higher body weight [18]. For overweight and obese women the mean difference was −1.14 BMI [19]. This would only strengthen our findings as the threshold of BMI where mortality hazards start to increase (see Fig. 1) would move up even higher. However, underreporting of BMI's does lead to underestimation of prevalence of obesity and hence to slightly underestimated loss of life expectancy on population level. Metha and Chang [20] show that the prevalence underestimation is small. BMI, while being used in health promotion, is but a fair measure of adiposity [18, 21–23]. Weight is the sum of fat-free mass and body fat, with opposite effects on health [23]. At a BMI of 26–27, the mean percentage of body fat is 26 but may vary between 16% and 40% [22]. Wasting associated with loss of muscle mass sharply increases the risk of death, while high levels of body fat maintain BMI at normal levels. Waist circumference (as proxy of adiposity) and midarm muscle circumference (as proxy of muscle mass) are far better predictors of mortality risks than BMI [21, 23].

We limited the analysis to all cause mortality, lacking cause specific data. Both overweight and obesity are associated with higher rates of diabetes and cardiovascular mortality [9]. Older studies like the Framingham Heart Study and to a lesser extent the Cancer Prevention Study II did detect a high excess risk of death in obesity [5, 24]. However, cardiovascular mortality rates have halved since the 1970s, particularly after 1980. Smoking, high blood pressure and increased cholesterol levels came down sharply, partly as a consequence of successful cardiovascular risk management [8]. This may well explain the disappearing excess mortality in more recent studies. Our analysis is comparable to the NHANES data, a recent meta-analysis of 26 prospective studies and many more [4, 9, 17, 25]. Increased morbidity and mortality from cardiovascular diseases and diabetes is a common observation, but mortality of all causes is lower at high normal weight and overweight. Even the Framingham Heart Study shows conflicting results, likely depending on choices made in age bands and calendar periods used [26].

Several studies show that healthier people at baseline show lowest mortality risks at lower BMI than less healthy people: among women, non-smokers, or those free from disease, BMI related to lowest mortality are lower [24, 25, 27, 28]. Among men, former and current smokers or those with disease, BMI related to lowest mortality are higher. In current populations of middle aged and elderly persons, the former categories are far smaller. The striking differences between the large populations of former smokers and never smokers might suggest other than causal explanations for lowered mortality at lower BMI in these groups. Risk avoidance of people living a prudent life can cause both lowered mortality and lower weight.

Fatal disease causing weight loss can never be fully excluded in observational studies. We assessed potential reverse causation by various sensitive analyses. We waited five years instead of three, excluded persons who lost weight or excluded individuals who reported their health status as poor at baseline. None of these altered the results materially. Longer term weight loss, was associated with an increased, not a decreased life expectancy.

As senescence and degenerative diseases cause loss of weight, a higher weight may extend life by offering increased reserves.

Lowered mortality of obesity should not lead to complacency [29, 30]. Firstly, while mortality is increased at very high levels of BMI, wasting together with adiposity may increase the mortality risks of "normal" BMI at middle and old age [21, 23]. Waist circumference as a marker of adiposity and midarm muscle circumference are not so difficult to obtain and explain better mortality risks at increasing ages than does BMI. Insertion tapes seem to give more valuable information than balances. Secondly, obesity may be the exact opposite of smoking: while smoking is still fairly fatal, obesity became largely non-fatal [4, 8]. Smoking prevents morbidity by premature death [31, 32]. Older HRS results on obesity showed little change in total life expectancy but shortened active life expectancy (active life expectancy is the ability to perform the activities of daily life) [33, 34]. The price to pay for the lowered mortality of obesity may well be increased morbidity and health care costs in the extended life.

While some researchers predict that obesity may cause life expectancy to decline [35], we conclude that the burden of mortality of obesity among white Americans aged 55 and over is limited to a still small fraction of severely obese people. Highly educated overweight never smokers may anticipate at age 55 a total life expectancy of 85.7 years and 88.2 years.

Further studies should identify the causes why overweight protects against all cause mortality, while cardiovascular mortality is still increased [9]. The interaction between overweight, increased cardiovascular risk and successful cardiovascular risk management may cause interesting dilemmas for future health policy. While the burden of mortality of obesity is fairly small, the burden of morbidity and obesity associated health care costs may increase, partly as a consequence of successful health care interventions promoting survival of the obese. Future research should focus more on morbidity and disability than on the rather limited excess mortality.

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