Years of Life Lost Due to Obesity

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M ANY PUBLIC HEALTH OFFI-cials and organizations have tried to warn the public about the dangers of obesity.[1,2] One of the strongest warnings came from the US surgeon General who stated that a failure to address overweight and obesity "could wipe out some of the gains we've made in areas such as heart disease, several forms of cancer, and other chronic health problems."[3]

For a health message to be effective, it should be both understood by the recipient and the information should be perceived as relevant.[4] In trying to convey the magnitude of the deleterious effects of obesity, investigators have presented information on the health care costs due to obesity,[5] the relative mortality rates among obese vs nonobese persons,[6] and the number of annual deaths that might be attributable to obesity.[7] These messages may not be effective for individuals in the United States, who may be less interested in population-level effects. One way to quantify the individual effect is in terms of the expected number of years of life lost (YLL). The *YLL* is defined as the difference between the number of years a person would be expected to live if he/she was not obese and the number of years expected to live if the person were obese.

For editorial comment see p 229.

Context Public health officials and organizations have disseminated health messages regarding the dangers of obesity, but these have not produced the desired effect.

Objective To estimate the expected number of years of life lost (YLL) due to overweight and obesity across the life span of an adult.

Design, Setting, and Subjects Data from the (1) US Life Tables (1999); (2) Third National Health and Nutrition Examination Survey (NHANES III; 1988-1994); and (3) First National Health and Nutrition Epidemiologic Follow-up Study (NHANES I and II; 1971-1992) and NHANES II Mortality Study (1976-1992) were used to derive YLL estimates for adults aged 18 to 85 years. Body mass index (BMI) integer-defined categories were used (ie, <17; 17 to <18; 18 to <19; 20 to <21; 21 to 45; or ≥45). A BMI of 24 was used as the reference category.

Main Outcome Measure The difference between the number of years of life expected if an individual were obese vs not obese, which was designated YLL.

Results Marked race and sex differences were observed in estimated YLL. Among whites, a J- or U-shaped association was found between overweight or obesity and YLL. The optimal BMI (associated with the least YLL or greatest longevity) is approximately 23 to 25 for whites and 23 to 30 for blacks. For any given degree of overweight, younger adults generally had greater YLL than did older adults. The maximum YLL for white men aged 20 to 30 years with a severe level of obesity (BMI >45) is 13 and is 8 for white women. For men, this could represent a 22% reduction in expected remaining life span. Among black men and black women older than 60 years, overweight and moderate obesity were generally not associated with an increased YLL and only severe obesity resulted in YLL. However, blacks at younger ages with severe levels of obesity had a maximum YLL of 20 for men and 5 for women.

Conclusion Obesity appears to lessen life expectancy markedly, especially among younger adults.

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To our knowledge, only 1 study has attempted to calculate YLL estimates due to obesity. Stevens et al[8] found that YLL estimates were greater for older than younger obese individuals. This seems counterintuitive and may be the result of the investigators restricting their analysis to a 12-year period rather than estimating YLL across the remaining life span. This may have led to marked underestimation of YLL and may have created a spurious positive correlation between baseline age and YLL. Therefore, we conducted a study estimating YLL across the entire life span to provide meaningful values.

METHODS
Method of Calculation

The YLL due to obesity can be estimated by combining 3 types of information[1]: an estimate of the distribution of body mass index (BMI) for each year

of adult life[2]; an estimate of the hazard ratio (HR) for death given various BMI levels in each year of adult life (ages 18-85 years); and the probability of death during each year of adult life.[3] Each of these types of information was obtained from existing public use data sources. The steps used to calculate YLL due to obesity are available at http://www.soph.uab.edu/statgenetics/Research/Tables/YLL-Calculation-Steps.pdf.

BMI Distribution

We derived population-based data on BMI distribution from the Third National Health and Nutrition Examination Survey (NHANES III). NHANES III collects information about health and diet in a representative sample of the civilian, noninstitutionalized US population. This survey also estimates the prevalence of a variety of health conditions and risk factors, such as obesity. NHANES combines a home interview with health tests, which are performed in a mobile examination center. NHANES III has been described in detail elsewhere.[9,10] In brief, NHANES III represents a 6-year study, involving two 3-year phases, conducted from 1988 to 1994. The data used to derive estimates of the population-based BMI distribution are from both phases of the NHANES III data collection. Mexican Americans, non-Hispanic blacks, and older adults were oversampled in NHANES III to ensure weighted, unbiased estimates from these particular groups. Herein, only black and white subjects were used because sample sizes of other groups were generally insufficient to allow reasonably precise estimates.

Body mass index was divided into ordered, nonoverlapping categories starting with less than 17 (ie, 17 to <18; 18 to <19; 20 to <21; 21 to 45; or ≥45). Second, for each race and sex group, the proportion of individuals within each BMI category was estimated. The proportions were calculated using a smoothing procedure based on moving averages. This procedure was necessary because continuous scales made categorical with fine gradations may re-

sult in insufficient data to produce precise estimates. Smoothing is a nonparametric way to minimize this problem. Specifically, we first estimated the probability of being in each of a series of 34 overlapping BMI intervals starting with the interval BMI of less than 17, then progressing through BMI intervals with widths of 5 (≤13 to <18; ≤14 to <19; ≤44 to <49; and the open interval of ≥45). For each interval, we assigned subjects a score of 1 if their observed BMI was within the interval and zero if it was not. This resulted in 34 binary variables. Each of these binary variables was then regressed on age to a third-degree polynomial via logistic regression.

We chose a model with a cubic polynomial because past research, which included the NHANES I data, showed that (over the adult life span) the change in BMI with age is convex and can be well characterized by a third-degree polynomial or less.[11] Using the resulting logistic regression equations, the probability of being in each of these broad intervals was calculated for each age from 18 to 85 years. Then, within each age, the probability of being in each integer-defined BMI category was calculated as the moving average of all the broader BMI bins containing the integer-defined BMI category of interest. These smoothed proportions were used to represent the probability of being in a given BMI category at each age.

Third, also using NHANES III data, we estimated the mean BMI within each BMI category for each race and sex group. This was simply estimated as the observed arithmetic mean within each sex by race by BMI category grouping.

The HR for BMI Levels in Each Year of Adult Life

To estimate the HR for death at a given BMI level in each year of adult life, we combined data from the First National Health and Nutrition Epidemiologic Follow-up Study (NHEFS) and the NHANES II Mortality Study (NH2MS).[12]

The NHEFS is a national longitudinal study that was designed to investigate the

relationships between clinical, nutritional, and behavioral factors assessed in NHANES I and II and subsequent morbidity, mortality, and hospital use, as well as changes in risk factors, functional limitation, and institutionalization.[12] The NHEFS includes all persons aged 25 to 74 years who completed a medical examination during NHANES I from 1971 to 1975 (n=14 407). Follow-up vital statistics were collected through 1992. Of all the participants, 96% have been successfully traced at some point through follow-up in 1992.[12]

The NH2MS cohort is composed of adults who were aged 30 to 75 years at the time of their NHANES II examination between 1976 and 1980 (n=9252). Two participants were unable to be followed-up because of incomplete personal identifying data and are considered lost to follow-up. The NH2MS cohort members were tracked through 1992 by searching national databases containing mortality and cause-of-death information. The 2 datasets were combined to increase sample size and precision of estimates.

Using these data, Cox regression models were used with mortality as the outcome and BMI, BMI[2], age, age[2], and their interaction terms as covariates. Potential age, race, and sex interactions were accounted for by fitting separate models to each race and sex group. Interactions of BMI and BMI[2] with age[2] were not used because prior research indicated that change in the BMI parameters appears to be roughly linear in age.[13] In addition, removal of such terms greatly reduced collinearity. Despite this, there may be some collinearity, particularly among variables such as age and age[2]. However, this is not a major concern in this situation because collinearity will increase the variance of the coefficients, but will not bias the parameter estimates or the overall predictions. Prior to estimating the Cox regression model, BMI was transformed to BMI inverse (1/BMI). We chose inverted BMI as a predictor instead of BMI because prior research has shown that inverted BMI is more suitable for modeling the convex but asymmetrical relationship be-

tween BMI and mortality.[14,15] Within every sex and age group examined in NHANES III, inverted BMI was more highly correlated with body fat than was BMI.[16] Smoking status (defined as current, former, never, or unknown) was controlled for in all analyses. Using the resulting equation, we calculated the estimated HR for each BMI category at each age interval by substituting the mean BMI in each BMI category for the BMI in the equation and the midpoint of the age interval (eg, 18.5 for 18- to 19-year-olds) for age in the equation. As a sensitivity analysis, we also calculated the estimated HRs across the categories of BMI using the sample weights that account for the complex sampling design of NHEFS and NH2MS. These weighted estimates were extremely similar to the unweighted estimates and when they differed, the HRs for obesity were higher with than without weighting, suggesting that our results may be conservative.

To test the proportionality assumption, Schoenfeld residuals were computed for each variable in the model. When the proportionality assumption is met, Schoenfeld residuals should be independent of survival time. Therefore, we plotted these residuals against survival time for each independent variable involving BMI (ie, BMI; BMI^2; $BMI \times age$; $BMI^2 \times age$) and also computed that Spearman nonparametric correlation between these residuals and survival time. This was performed separately for each of the 4 sex and race groups. In each group, all 4 plots gave no indication of a nonrandom pattern of association. Moreover, across all 4 variables in all 4 groups, the largest Spearman r^2 for the correlation with survival time was less than 0.5% of the variance. Thus, there is substantial evidence for the validity of the proportionality assumption. All logistic regression models were fit using SPSS statistical software (Version 10.0.7; SPSS Inc, Chicago, Ill).

Life Tables

The life table for the total population of the United States in 1999 was used to derive information on the probability of death during each year of life (http://www.cdc.gov/nchs/data/nvsr/nvsr50/nvsr50_06.pdf). For each race- and sex-specific age interval, we obtained an estimate of the total probability of death within the interval, which was conditional on having lived to the start of that interval.

Estimate of YLL

Using data from the (1) US Life Tables (1999); (2) Third National Health and Nutrition Examination Survey (NHANES III; 1988-1994); and (3) First National Health and Nutrition Epidemiologic Follow-up Study (NHANES I and II; 1971-1992) and NHANES II Mortality Study (1976-1992), we were able, for each age interval, in each of 4 race and sex groups, to obtain an estimate of the probability of death within the interval, which was conditional on having lived to the start of that interval and being in the first BMI category. The probability of death within the interval conditional on having lived to the start of that interval and being in a given BMI category was also estimated. For a person of a given age in a given BMI category, we estimated their expected age of death (operationally defined as the median age of death for a person with their starting age and BMI). Finally, the YLL for a person of a given age in a given BMI category is the difference relative to being in the reference category (ie, BMI=24). A BMI of 24 was chosen as the reference category because it represents the upper bound of desirable weight.

RESULTS

The YLL due to obesity estimates for each sex and race group (compared with a BMI of 24) for ages 20, 30, 40, 50, 60, and 70 years are displayed in **FIGURE 1** and **FIGURE 2**. A complete tabulation of the YLL estimates across the entire range of BMI is posted on our Web site (http://www.soph.uab.edu/statgenetics/Research/Tables/YLL.htm). Among whites, the overall pattern suggests a J- or U-shaped association. For example, YLL would be 1 to 9 in individuals with low BMIs (<17 to 19) and 9 to 13 in individuals with high BMIs (≥35). White men aged 20 years with BMIs greater than 45 are estimated to have 13 YLL relative to white men aged 20 years with BMIs of 24. A similar, although less pronounced pattern occurs throughout the upper range of BMI (Figure 1). Among white women, the pattern shows less variability across the range of BMI distribution as a function of age. White women aged 20 to 30 years with BMIs greater than 45 are estimated to have 8 YLL due to obesity (Figure 1).

The overall pattern of findings among blacks was somewhat different. Among black men, estimated YLL due to obesity did not begin for any age group until a BMI of 32 was reached. Obesity appeared to be associated with a slightly increased life expectancy across much of the moderate obesity range for the older age groups (60-70 years). Twenty was the maximum YLL found among black men aged 20 years with BMIs greater than 45 (Figure 2). The maximum YLL for black women aged 20 years with BMIs greater than 45 was 5 (Figure 2).

COMMENT

Using 1999 US Life Tables and population-based data to derive estimates of the BMI distribution for each year of adult life, as well as the HR for death for each BMI category in each year of adult life, we found that obesity has a profound effect on life span. That is, mirroring the association between BMI and mortality, a J- or U-shaped association between BMI and YLL at all ages was observed. Moreover, the optimal BMI (ie, the BMI associated with the greatest longevity) for adults aged 18 to 85 years is approximately 23 to 25 for whites and approximately 23 to 30 for blacks.

A 20-year-old white male with a BMI greater than 45 is estimated to have 13 YLL due to obesity. Assuming a life expectancy of 78 years, this translates into a 17% reduction in total life expectancy. When one considers that a 20-year-old white male is expected to live another 58 years, a 13-year reduction

due to obesity actually represents a 22% reduction in remaining years of life. White women aged 20 years with BMIs greater than 45 are estimated to have 8 YLL due to obesity, which is a 10% reduction in total life expectancy. Assuming 61 years of life remaining for a 20-year-old woman, an 8-year reduction due to obesity actually represents a 13% reduction in remaining years of life. Thus, our findings suggest that obesity has a marked effect on the life span of whites, and that the risk of increased YLL was greatest at younger ages.

The pattern of findings among blacks was somewhat different. Obesity was associated with only slightly increased life expectancy across much of the overweight and moderate obesity range for most age groups. A consistent reduction in expected life expectancy was not observed until BMIs of 37 to 38 for women and 32 to 33 for men were reached. The maximum YLL was 20 among black men aged 20 years with BMIs greater than 45. This is a 29% re-

duction based on a life expectancy of 70 years and a 40% reduction in remaining years of life. Given the small sample size of black men with extremely high BMIs, it is worth considering the more conservative estimate obtained among black men with BMIs of 44 to 45. For these 20-year-olds, the YLL was 11, which is a 16% reduction in total life expectancy and a 22% reduction in remaining years of life. Among black women aged 20 years with BMIs greater than 45, the YLL was 5, which is a 6.6% reduction in total life expectancy of 76 years and an 8.9% reduction in remaining years of life. Despite the different pattern of findings from whites, we also observed that YLL were greatest at younger ages.

In one respect, our findings run counter to those reported by Stevens et al[8] in that the YLL attributable to obesity increased with age, but declined for those in the oldest decade (70-79 years). A possible explanation for the divergent findings is that we estimated YLL

across the remaining years of life while Stevens et al restricted the analyses to a 12-year period using data from the Cancer Prevention Study I. This approach may have led to both an underestimation of YLL and a spurious positive association between baseline age and YLL. This is because the method of estimating YLL used by Stevens et al defined the observed YLL to be zero for all subjects who did not die within the duration of the study. This results in an overall underestimation of YLL because those individuals who will die before the median life span for their demographic group (ie, would have an YLL >0), but did not die during the study, are coded as YLL=0. Moreover, younger individuals (independent of BMI) are less likely to die than older people within any defined period. This presumption results in a greater underestimation for younger rather than older individuals and potentially leads to a spurious positive correlation between YLL and age. Our YLL esti-

Figure 1. Years of Life Lost Among White Men and Women
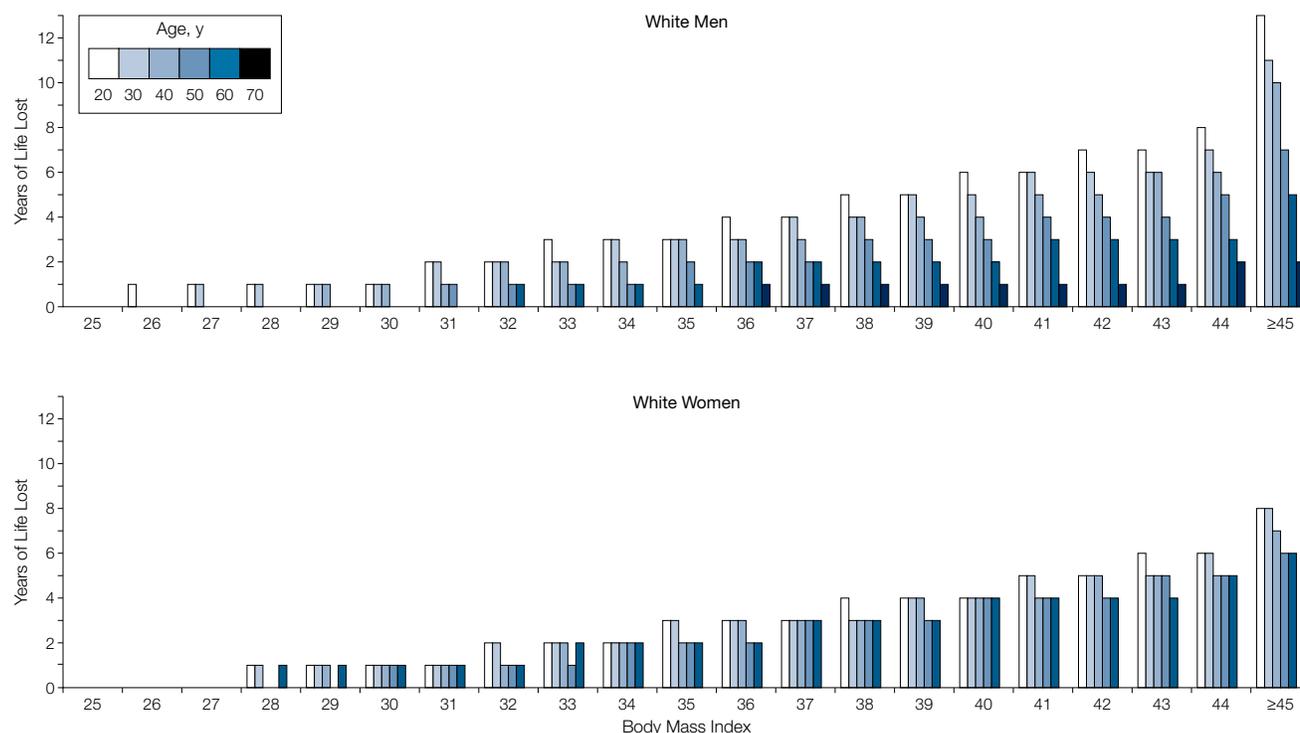
mates also seem markedly higher than those obtained by Stevens et al who considered all levels of obesity as a single class (ie, BMI ≥30) so the results are not easily compared directly with ours. Nevertheless, the YLL associated with obesity did not exceed 1 for any age or sex group considered.

There was some difference in the pattern of findings in YLL due to obesity between whites and blacks. This is actually consistent with what has been observed regarding the association between BMI and mortality between these races.[17] For example, in a cohort of 2731 black women who were members of the Kaiser Foundation Health Plan and

were followed-up for 15 years, Wienpahl et al[18] found an essentially flat association between BMI and mortality across the entire range of BMI. Other investigators[19-22] have obtained similar results, which suggests that the effect of a given BMI increase on mortality rate may be different and sometimes less deleterious among blacks relative to whites.

A recent review[17] of the literature on the association between BMI and mortality in blacks indicates that obesity resulted in a smaller increase in mortality among blacks compared with whites. Using data from the Cancer Prevention Study II, a J- or U-shaped pattern

between BMI and relative rates of mortality among whites was obtained. In contrast, the pattern among black men and women suggests that the only category in which the relative mortality rate is consistently and substantially elevated is among black women with BMIs of less than 18.5.

At least 4 possible explanations may account for the differential relationship between moderately high levels of BMI, mortality, and YLL across races. First, the true causal effect of BMI on mortality rate may differ between blacks and whites. Second, blacks and whites may be exposed to different competing risks for mortality and this may

Figure 2. Years of Life Lost Among Black Men and Women
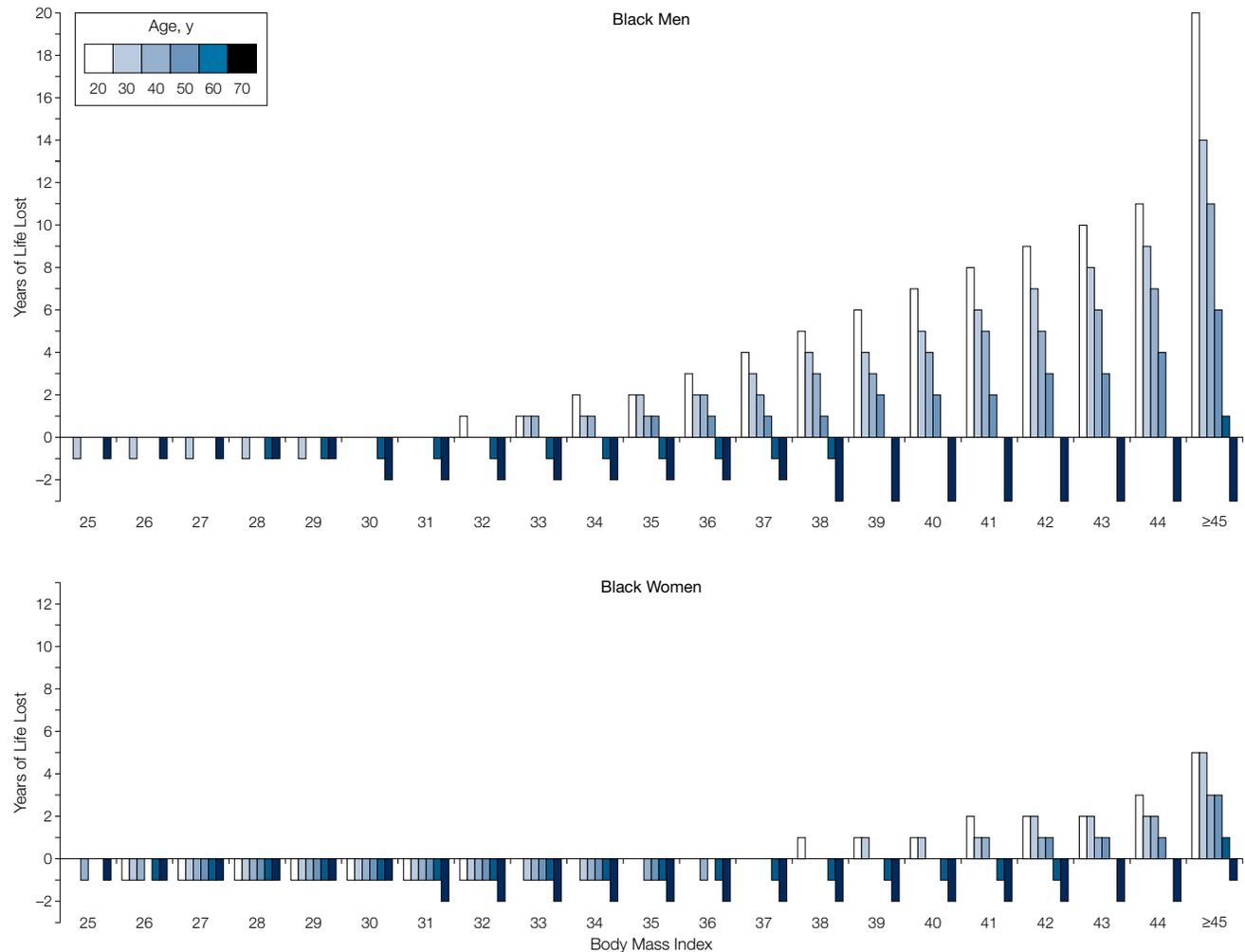
affect the outcome.[23] That is, distributions of age, health status, socioeconomic status, and other variables that can affect mortality rate may differ by race and this can affect the estimated HR which, in turn, influences the YLL estimates. For example, homicide is the leading cause of death among blacks aged 15 to 34 years, and the third leading cause of potential YLL.[24] Thus, it is possible that homicides, a cause of death presumably independent of BMI, may disproportionately account for excess deaths among blacks, particularly males, thereby altering the influence of obesity on YLL estimates. Third, there may be different distributions of confounding variables among blacks compared with whites. Fourth, the critical variable may be body composition, not BMI, and the relationship between metabolism, BMI, and body composition (central adiposity) may differ by race, especially among women.[25,26] These 4 potential explanations are neither mutually exclusive nor exhaustive.

We also observed some sex differences in the association between obesity and YLL. On the whole, independent of race, men were at increased YLL risk. Among white men, the increased YLL risk was generally more uniform across age than it was for white women. This is consistent with Stevens et al,[8] who also found that YLL was higher among men than women with comparable BMIs.

The results of this study should be interpreted in light of several limitations. Although elevated BMI is the primary index of obesity used in most studies, it is a proxy of adiposity and may not provide the best estimate of the association between obesity and outcomes such as mortality or YLL. Body mass index involves fat mass and fat-free mass, so its use may mask any differential health consequences associated with fat mass and fat-free mass,[27-30] which suggests that future studies should obtain not only BMI, but direct measures of body composition as well.

Due to insufficient sample size, the association between obesity and YLL could not be reliably estimated for other racial/ethnic groups, including Mexican-Americans and Pacific-Islander Americans, groups that are known to have high prevalences of overweight and obesity. Until databases of sufficient size are available for these racial groups, the association between BMI and YLL will remain unknown.

A related issue concerns the relatively small sample size of individuals in the upper BMI categories. We chose to collapse the upper BMI categories at 45 or greater, as opposed to some other cut point because of the increasing proportion of individuals with BMIs above 40 and the markedly accelerated increase in mortality rate for individuals with high BMI levels.[31,32] To address whether the relative sparseness of data in the upper BMI categories may have rendered YLL estimates imprecise, we reversed the order of the pi's for the upper BMI categories for white men and black women to evaluate the extent to which this influenced YLL estimates. Less than 1% of the YLL estimates for white men in the upper BMI categories were altered, and in all of these cases, the changes were minimal (ie, ±1 year). For black women, there were more changes (approximately 8%), but the magnitude of these changes on YLL estimates were small (±1 year). Thus, although sample sizes in the upper BMI categories were modest, our results appear to be quite robust to any misestimation of the frequency distribution of BMI at the upper end of the BMI continuum.

Although an estimate of YLL due to obesity is interesting and important, it is only 1 consequence of obesity. A recent review[33] indicates that obesity significantly impairs quality of life, arguably a more potent marker of the effect of obesity because quality-of-life deficits are experienced in the moment rather than anticipated sometime in the future.

We controlled for smoking status as a potential confounder of the association between BMI and YLL. Specifically, we categorized individuals as current, former, or never smokers, and unknown. The number of cigarettes smoked was not taken into account because of the limitation of the data. We also did not consider that changes in smoking prevalence are occurring over time. Given that smoking affects both body weight and mortality rate, this may affect the YLL associated with obesity at future time points. Thus, future researchers may wish to reestimate YLL values at periodic intervals to see if changes occur.

We also did not control for the possible confounding effects of subclinical or occult disease. This is because it is unclear exactly how to control for something that is, by definition, unobservable. Excluding individuals who died early in follow-up (eg, ≤5 years) to reduce confounding due to preexisting disease has been shown to be ineffective and possibly even exacerbating.[28,29,34] Nonetheless, within the context of BMI and YLL studies, it is possible that preexisting occult disease could confound the association between BMI and YLL and lead spuriously to a diminution in the observed increase in YLL across the upper range of BMI. To the extent that confounding due to occult disease exists, our YLL estimates are likely to be underestimates.

Our YLL calculations assume that an individual's BMI remains constant across age. We sought to address the question: Given that an individual has a BMI of 37, for example, how much longer would he/she be expected to live if the BMI was 24? This is different than addressing the question: Given that an individual has a BMI of 37, for example, and gains or loses weight at the same rate as others with BMIs of 37, for example, how much longer would he/she be expected to live if the BMI was 24 and gained or lost weight at the same rate as others with BMIs of 24? The former question is more germane to public health and clinical communication.

Given that we used information from 3 separate and different data sets in our calculation procedure, we were unable to provide confidence intervals for our YLL estimates. We are unaware of any developed analytic formula that would allow easy calculation of SEs and confidence intervals. Computer-

intensive sampling procedures could be used, but that is a topic for future research. It is likely that our YLL estimates will be least precise when there is less data such as among blacks and among extremely obese individuals.

Finally, we did not determine statistical power a priori. Although, given our sample size, this is not a limitation per se for most of our analyses, it should be noted that as one moves to the extremes of the distribution of predictor variables under study, fewer data are available and estimates become less stable, and the HRs can substantially influence estimates of YLL. Therefore, it will be useful for future research to replicate and refine the results herein using larger data sets, particularly among nonwhites.

There are also several strengths of the study. We used large nationally representative samples to derive our estimates of BMI distribution and HR of death across the life span. We also used an analytic approach that generated YLL estimates for each remaining year of life along the range of BMI. Moreover, our YLL estimates are likely conservative because we did not exclude cohort members (eg, ever smokers, weight fluctuators).

Our results confirm that obesity is a major public health problem that appears to lessen life expectancy markedly, especially among individuals in younger age groups. Among blacks, the pattern of findings suggests that overweight and obesity may not decrease life expectancy until a BMI of approximately 32 to 33 for men and 37 to 38 for women is reached. At these BMI levels, longevity begins to decrease, especially among individuals in the younger age categories. Our estimates of YLL due to obesity strongly support the public health recommendation for adults to avoid obesity.

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