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Population heterogeneity in the impact of body weight on mortality

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\textbf{ABSTRACT}

Existing research provides inconsistent evidence for a relationship between overweight and/or obesity and mortality, and poorly studies the population heterogeneity with respect to the mortality consequence of overweight/obesity. This study investigates how overweight and/or obesity affect mortality and how these effects may vary across sociodemographic groups defined by race, sex, age, education and income by using the U.S. Third National Health and Nutrition Examination Survey (NHANES III) with linked mortality data from 1988 to 2006 (6915 respondents with 2694 deaths). Analysis from Cox proportional hazard model suggests overweight people are at lower risk of death compared to normal weight people, but this protection effect is concentrated in black men, older adults, and people in the lowest income stratum. Class I obesity does not increase mortality risk, but Class II/III obesity does and the harmful effect is concentrated in whites, young and middle adults, and people with higher education and income levels. We discuss these findings in the context of the extant literature and the long-term prospect of life expectancy in the U.S.

Introduction

Obesity has become epidemic in the world, particularly in the United States. Current literature in demography and epidemiology has well documented the prevalence and distribution of obesity across various social demographic characteristics (e.g., Baskin, Ard, Franklin, & Allison, 2005; Centers for Disease Control and Prevention, 2009; Robinson, Gordon-Larsen, Kaufamn, Suchindran, & Stevens, 2009; Wang & Beydoun, 2007), and life course transitions in the obesity status (e.g., Guo & Chumlea, 1999; Harris, Perreira, & Lee, 2009; Scharoun-Lee, Kaufamn, Suchindran, & Stevens, 2009; Wang & Beydoun, 2007), and life course transitions in the obesity status (e.g., Guo & Chumlea, 1999; Harris, Perreira, & Lee, 2009; Scharoun-Lee, Kaufamn, Suchindran, & Stevens, 2009; Wang & Beydoun, 2007). This point is echoed by Stewart, Cutler, and Rosen (2009), which claimed that the gained life expectancy from declining smoking rates will be outweighed by the negative impact of rising obesity.

Less is understood about the impact of obesity and/or overweight on mortality and life expectancy. In fact, extant studies have provided divergent estimates of the percentage of U.S. adult mortality attributable to obesity and overweight. A large number of studies have documented the deleterious effect of obesity (BMI ≥ 30.0 kg/m\textsuperscript{2}) on survival and life expectancy (e.g., Calle, Rodriguez, Walker-Thurmond, & Thun, 2003; Fontaine, Redden, Wang, Westfall, & Allison, 2003; Peeters et al., 2003). It was estimated that 350,000 deaths in the U.S. in 2000 were caused by overweight and obesity, which accounted for about 15.2% of total deaths (Mokdad, Marks, Stroup, & Gerberding, 2004, 2005). These findings lead to a warning that the long-term secular increase in U.S. life expectancy will be threatened by rising obesity (Olshansky et al., 2005). This point is echoed by Stewart, Cutler, and Rosen (2009), which claimed that the gained life expectancy from declining smoking rates will be outweighed by the negative impact of rising obesity.

Some recent studies, however, have found only a moderate impact of obesity on mortality (e.g., Flegal, Graubard, Williamson, & Gail, 2005; Metha & Chang, 2009). For example, Flegal et al. (2005) found obesity was associated with 111,909 excess deaths and overweight (BMI = 25.0–29.9) was associated with 86,094 fewer deaths among U.S. adults in 2000. Therefore, only about 25,815 deaths were attributable to overweight and obesity, which is a much smaller figure than the estimates from Mokdad et al. (2005). Moreover, the deleterious effect of obesity may have been decreasing over time. Metha and Chang (2011) compared the effect of obesity on mortality risks in three datasets (Framingham Heart Study, National Health and Nutrition Examination Survey, and National Health Interview Survey) over the time period 1948–2006 and found the deleterious effect of class I obesity (BMI = 30.0–34.9) significantly declined and even disappeared due to the declining association between obesity and cardiovascular disease mortality, the leading cause of deaths in the U.S. (National Research Council, 2011). The weakening impact of class II/III obesity (BMI ≥ 35), however, is only observed in the NHIS self-reported BMI data.
Recent studies suggest two major reasons for this potential decline, including improvements in medical and surgical treatments (e.g., secondary-prevention medication, treatments for acute coronary syndromes and heart failure) and decreases in heart disease risk factors in the obese such as high cholesterol, high blood pressure and smoking (Ford et al., 2007; Gregg et al., 2005).

Still other studies have not found any significant harmful effect (e.g., Reynolds, Saito, & Crimmins, 2005) and even found a beneficial impact of being obese among older people (Lantz, Golberstein, House, & Morenoff, 2010; Stallard, 2010). These divergent findings lead to uncertainty concerning the impact of obesity on mortality risk.

With reference to the impact of being overweight on mortality risks, some studies found being overweight increases mortality risk (e.g., Calle, Thun, Petrelli, Rodriguez, & Heath, 1999), especially among healthy people (e.g., Adams et al., 2006), while others found overweight adults have similar, or even lower, mortality risks than normal weight adults (e.g., Flegal et al., 2005; Flegal, Graubard, Williamson, & Gail, 2007; Janssen, 2007; Lantz et al., 2010; Metha & Chang, 2009, 2011; Orpana et al., 2009). For example, it was found that after controlling for sociodemographic status and other risk factors, overweight lowers the total mortality rate among adults age 25 and older (Flegal et al., 2005) and decreases the non-cancer and non-cardiovascular disease related mortality (especially chronic respiratory disease, acute respiratory and infectious disease, injuries, and miscellaneous causes) (Flegal et al., 2007). The mechanism for the protective effect of overweight against mortality is not clear, however. It is possible that lean mass and fat mass may reduce mortality risk by affording protection against nutritional and energy deficiencies, the metabolic stresses, the development of wasting and frailty, and the loss of muscle, bone and fat caused by chronic diseases, e.g., heart failure (Janssen, 2007; Oreopoulos et al., 2008). For example, higher cholesterol levels are positively associated with survival in heart failure (Horwich et al., 2001).

Several factors may contribute to the divergent findings in the literature. First, the timing of studies may partially explain the divergent findings on the impact of overweight/obesity on mortality. If the negative impact of overweight/obesity declines over time (Flegal et al., 2005; Metha & Chang, 2011), then studies based on data of earlier periods (e.g., before 1990) are more likely to detect a stronger harmful impact of obesity than later studies. Second, the impact of obesity on mortality and life expectancy may depend on the extent of severity of obesity. A recent Canadian study (Orpana et al., 2009) found that non-morbid obesity (BMI 30–34.9) is not significantly linked with mortality risk, while morbid obesity (BMI > 35) does. This finding has been observed in several other studies as well (e.g., Metha & Chang, 2009). Therefore the effect of obesity in a given study depends on the proportionate shares of class I obesity and class II/III obesity in the sample. Without distinguishing these two levels of obesity, a study may yield misleading and inaccurate estimates of the effects of obesity. Third, the impact of obesity and overweight on mortality and life expectancy may vary by social demographic characteristics that affect sample compositions. For example, while some studies found that obesity did not significantly reduce life expectancy in older adults (Lantz et al., 2010; Reynolds et al., 2005; Stallard, 2010), others found that severe obesity leads to substantial years of life lost among younger adults (e.g., Fontaine et al., 2003). Freedman, Ron, Ballard-Barbash, Doody, and Linet (2006) also reported a more pronounced association between BMI and mortality risks among young and middle-aged adults than among the elderly. Race and gender may also affect the BMI-mortality risk. As reported by Fontaine et al. (2003), optimal BMI (with regard to the greatest longevity) is higher among Blacks than among Whites and the years of life lost due to obesity are more substantial among men than among women. In addition, overweight may provide important nutritional reserves (Janssen, 2007; Oreopoulos et al., 2008) which may be especially important for people with lower socioeconomic status (e.g., income and education) as they have limited access to valuable resources (e.g., food, nutrition, housing, health care). Therefore, there exists population heterogeneity in the BMI-mortality link which may contribute to the divergent findings regarding the links between adiposity and survival but has not been comprehensively assessed.

This study addresses this gap by examining the impact of overweight/obesity on mortality across a wide range of sociodemographic groups defined by age, race, sex, education and income. We test the hypothesis that obesity may be more harmful for younger adults, whites, men, and people with higher socioeconomic status and overweight may protect people with lower socioeconomic status. In addition to explicitly accounting for social heterogeneity of the BMI-mortality link, our approach improves upon previous ones by clearly distinguishing different degrees of obesity and using large nationally-representative samples with a longer time period of mortality data. We use objective physical examination data of weight and height to avoid potential response bias in self-report data that may also have group variations. Findings are adjusted for potential confounders that may bias the estimates of BMI, including sociodemographic characteristics, health insurance, subjective health, 14 chronic conditions, and smoking status. Controlling for chronic conditions and smoking status minimizes the potential bias due to diseases or smoking induced unintentional weight loss and mortality risk. Results indicate that population heterogeneity in the overweight/obese and mortality links is a critical issue that warrants more attention in future studies of this topic.

Methods

Data and sample

We use the data from the Third National Health and Nutrition Examination Survey (NHANES III) with linked mortality data. The NHANES III, conducted between 1988 and 1994 by the National Center for Health Statistics, collected information about health and diet from a nationally-representative sample of civilian, non-institutionalized U.S. population, with an oversample of older adults and racial minorities. We restricted our analysis to non-Hispanic White and non-Hispanic Black because Hispanics are a selective group due to the “healthy migrant” effects (Singh & Siahpush, 2001). Including them would induce sample selection bias in the interpretation of race effects. The follow-up mortality data tracked the mortality status of respondents from the date of survey through December 31, 2006 for periods of up to 18 years. Mortality ascertainment is based on the results from a probabilistic match between NHANES III and the National Death Index (NDI) death certificate records. After deleting all the missing data in the NHANES III with linked mortality data, the final sample includes 6915 respondents. Ethics approval was not required since the study used secondary data without any identifiable personal information.

Variables

The outcome variable is exposure time between the interview and event for the follow-up period of up to 218 months from 1988 to 2006. Respondents who were not identified as deceased by the end of the follow-up period were assumed to be alive. Among the 6915 respondents, 2694 died during this period.

Body mass index (BMI) is the main explanatory variable. We defined four BMI groups based on World Health Organization...
found none of the interactions to be significant. We tested the interaction between each covariate and duration and found none of the interactions to be significant except the one between age and duration, so the proportionality assumption is not violated except for age. In this case, the age effect is a type of average effect over the range of times observed in the data (Allison, 1995). All statistical analyses are adjusted for complex survey designs using sampling weight.

Population-attributable risk fraction

After estimating Cox proportional hazard model, we calculate the population-attributable risk fraction (PAF) to capture the fraction of deaths which can be avoided if the risk factors are removed in the counterfactual situation. We use the same formula as in Metha and Chang (2011) to calculate PAF:

\[ \frac{\sum_j \left( \hat{C}_j \hat{R}_j - \hat{C}_j^{\ast} \hat{R}_j \right)}{\sum_j \left( \hat{C}_j \hat{R}_j \right)} \]

where \( j \) indexes the category of BMI (normal weight, overweight, class I obesity, or class II/III obesity), \( \hat{C}_j \) refers to the proportion of the \( j \)th BMI category in the population, and \( \hat{R}_j \) refers to the relative mortality risk of the \( j \)th BMI category compared to normal weight which can be obtained from hazard ratios (HRs thereafter) in Cox proportional hazard model. \( \hat{C}_j^{\ast} \) is the countefactual proportion of the \( j \)th BMI category in the population when all the respondents in the corresponding \( j \)th category are assigned to normal weight category.

Results

Table 1 describes the baseline sociodemographic characteristics by four BMI groups. Although women were more likely to be in normal weight ranges than men, they were also more likely to have class II/III obesity. Non-Hispanic Blacks had less desirable BMI status and were more likely to be obese (especially class II/III obese) than Non-Hispanic White. People over 70 years old had a smaller proportion of being obese than other age groups which may have resulted from weight loss due to diseases. People with higher socioeconomic status (i.e., education and income) were more likely to be in normal weight ranges than those with lower socioeconomic status although this pattern is not significant for income quartiles. Having insurance did not increase the probability of being in normal weight but did reduce the risk of having class II/III obesity. People reporting good, very good or excellent health were significantly more likely to have normal weight and were less likely to have class II/III obesity. Having no chronic condition was associated with a higher probability of being at a normal weight and a lower probability of being obese. Current smokers were more likely to have normal weight and were less likely to be obese than former and never smokers because smokers tend to be leaner (Metha & Chang, 2009).

Overweight, obesity and mortality risk

Table 2 shows the mortality HRs for overweight and obesity relative to normal weight among the entire sample. In Model 1, overweight was significantly associated with a higher mortality risk compared to the reference group “normal weight” but class I obesity and class II/III obesity were not. After controlling for...
Note: all analyses have controlled age, age squared, education, income, insurance, smoking status, self-rated health and chronic condition.

Mortality hazard ratios for overweight and obesity relative to normal weight from weighted Cox proportional hazard model.

Table 3

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Hispanic Black women</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight</td>
<td>1.00 [Ref.]</td>
<td>1.00 [Ref.]</td>
<td>1.00 [Ref.]</td>
</tr>
<tr>
<td>Class I obese</td>
<td>1.15 (1.03, 1.28)</td>
<td>.99 (0.86, 1.13)</td>
<td>1.00 (0.87, 1.15)</td>
</tr>
<tr>
<td>Class II/III obese</td>
<td>1.15 (0.98, 1.35)</td>
<td>1.40*** (1.19, 1.65)</td>
<td>1.42*** (1.20, 1.68)</td>
</tr>
<tr>
<td>Age</td>
<td>1.08*** (1.05, 1.11)</td>
<td>1.05*** (1.02, 1.08)</td>
<td>1.01 (0.99, 1.03)</td>
</tr>
<tr>
<td>Age squared</td>
<td>1.64*** (1.49, 1.81)</td>
<td>1.55*** (1.40, 1.71)</td>
<td>1.51*** (1.36, 1.67)</td>
</tr>
<tr>
<td>Men</td>
<td>0.80*** (0.69, 0.91)</td>
<td>0.79*** (0.69, 0.91)</td>
<td>0.79*** (0.69, 0.91)</td>
</tr>
<tr>
<td>Education</td>
<td>0.99*** (0.96, 1.00)</td>
<td>0.99*** (0.96, 1.00)</td>
<td>0.99*** (0.96, 1.00)</td>
</tr>
<tr>
<td>Income</td>
<td>0.74* (0.59, 0.92)</td>
<td>0.78* (0.63, 0.98)</td>
<td>0.77* (0.62, 0.94)</td>
</tr>
<tr>
<td>Non-Hispanic White</td>
<td>1.34*** (1.22, 1.51)</td>
<td>1.34*** (1.21, 1.52)</td>
<td>1.34*** (1.21, 1.52)</td>
</tr>
<tr>
<td>Chronic condition</td>
<td>1.17*** (1.14, 1.21)</td>
<td>1.17*** (1.14, 1.21)</td>
<td>1.17*** (1.14, 1.21)</td>
</tr>
<tr>
<td>Former smoker</td>
<td>1.36*** (1.21, 1.52)</td>
<td>1.36*** (1.21, 1.52)</td>
<td>1.36*** (1.21, 1.52)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>2.28*** (2.00, 2.61)</td>
<td>2.28*** (2.00, 2.61)</td>
<td>2.28*** (2.00, 2.61)</td>
</tr>
<tr>
<td>Sample size</td>
<td>6915</td>
<td>6915</td>
<td>6915</td>
</tr>
</tbody>
</table>

*p < .05; **p < .01; ***p < .001.

Attributable mortality for BMI categories

Table 7 lists the estimates of attributable mortality for each higher BMI category for the total sample and by sociodemographic groups. Attributable mortality reflects the percentage of deaths that could be avoided if the corresponding risk factor was eliminated. Values of relative mortality risks are based on the HRs in Tables 2–6 with normal weight as the reference group. Negative attributable mortality as a percentage means the mortality would increase if the corresponding risk factor was eliminated. The PAF analysis based on the entire sample from Model 3 of Table 2 shows that class II/III obesity increased the mortality risk by 42%, which corresponds to 4% (p < .001) of all deaths in this sample between year 1988 and 2006. Additional analysis taking smoking into account suggests that former smoker and current smoker statuses contributed to 7%
among Black men (1.49%; *p < .05) and 25% (*p < .001) of all deaths in this period, respectively. Therefore, smoking-attributable mortality was about 32% (*p < .001), much larger than obesity-attributable mortality.

Overweight protected people who tend to have worse health and significantly contributed to –12.6% (*p < .05), –6.1% (*p < .05), and –7.8% (*p < .05) of deaths for Non-Hispanic Black men, the elderly aged 70 years and over, and people located in the lowest quartile of income distribution, respectively. In other words, if all the overweight people had normal weight, mortality would have been negligible for the elderly aged 70 and over (.21%; *p < .05). The deleterious impact of class II/III obesity was especially harmful for people who tend to have better health than their counterparts, e.g., White women and men vs. Black women and men, young and middle-age adults versus older adults, people with bachelor degree or more versus those without, and people located in higher quartiles of income distribution versus those in lower quartiles. For example, class II/III obesity accounted for 7.5% (*p < .001) and 4.2% (*p < .01) of deaths among White men and women, respectively, but did not significantly contribute to deaths among Black men (1.49%; *p < .05) and women (–2.8%; *p < .05). The attributable mortality percentages were higher among people aged 30–59 (10.2%; *p < .001) and aged 60–69 (4.8%; *p < .05), and were negligible for the elderly aged 70 and over (2.1%; *p < .05).

Class II/III obesity were responsible for a higher percentage of deaths among people with bachelor degree or more (5.3% (*p < .01) than those with less than bachelor degree (3.9% (*p < .001). The class II/III obesity-attributable mortality percentages also increased with income strata, which are 2% (*p < .005), 3.9% (*p < .05), 5.4% (*p < .05), and 8.8% (*p < .001) for 1st, 2nd, 3rd, and 4th quartile, respectively.

Comment

The impact of overweight and obesity on mortality remains a controversial topic despite a decade of research. This study goes beyond the extant research by examining the population heterogeneity of the BMI-mortality link and provides a possible explanation of the divergent findings in the literature. Therefore, the impact of overweight and obesity on mortality risk varies by sociodemographic groups and the population composition of the sample may, then, affect the aggregate BMI-mortality link based on the whole sample. We used the Third National Health and Nutrition Examination Survey (NHANES III) with up to 18 years of mortality follow-up data from 1988 to 2006 to test this hypothesis. Besides the key variables of interest on sociodemographic characteristics including age, sex, race, education and income, we adjusted for health insurance status, self-reported health, preexisting chronic illnesses, and smoking status. To the extent that diseases and smoking may induce weight loss and/or increase mortality risk, controlling for these factors avoids the potential underestimates of the effects of overweight and obesity (e.g., Manson et al., 2007; Willett, Hu, Colditz, & Manson, 2005).

We find that overweight (BMI = 25–29.9) lowers mortality risk in the follow-up period when compared to normal weight even after controlling for chronic conditions. Class I obesity (BMI = 30–34.9) does not significantly raise mortality risk. The positive association between class II/III obesity (BMI ≥ 35) and mortality weakens but remains highly significant even after controlling for comorbidity. After controlling for smoking status, the protective effect of overweight against mortality becomes non-significant whereas the harmful effect of class II/III obesity on survival strengthens. Both results are consistent with those reported in other studies (e.g., Flegal et al., 2005, 2007; Metha & Chang, 2009, 2011; Orpina et al., 2009; Reuser, Bonneux, & Willeksens, 2009). An additional new finding has emerged from our PAF analysis. That is, although class II/III obesity confers a much elevated relative mortality risk compared to normal weight, the percentage of all deaths attributable to this category of obesity is only one eighth of that attributable to smoking during the period of 1988 and 2006.

We further disaggregate the BMI-mortality link by sociodemographic groups defined by sex, race, age, education and income and find that the protective effect of being overweight is concentrated in non-Hispanic Black men, adults aged 70 and over, and people

Table 5

<table>
<thead>
<tr>
<th>Mortality hazard ratios for overweight and obesity relative to normal weight by education levels from weighted Cox proportional hazard model.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than bachelor degree</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>Overweight</td>
</tr>
<tr>
<td>Class I obese</td>
</tr>
<tr>
<td>Class II/III obese</td>
</tr>
</tbody>
</table>

Sample size: 6158
757

Note: all analyses have controlled age, age squared, sex, race, income, insurance, smoking status, self-rated health and chronic condition. *p < .05; **p < .01; ***p < .001.

Table 6

<table>
<thead>
<tr>
<th>Mortality hazard ratios for overweight and obesity relative to normal weight by income levels from weighted Cox proportional hazard model.</th>
</tr>
</thead>
<tbody>
<tr>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>Lower than 25%</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>Overweight</td>
</tr>
<tr>
<td>Class I obese</td>
</tr>
<tr>
<td>Class II/III obese</td>
</tr>
</tbody>
</table>

Sample size: 1731
2226
1499
1459

Note: all analyses have controlled age, age squared, sex, race, education, insurance, smoking status, self-rated health and chronic condition. *p < .05; **p < .01; ***p < .001.
the lowest income quartile. The protective effect of overweight against mortality has been observed in other studies on the elderly (e.g., Lantz et al., 2010).

There may be several explanations for a lower mortality risk associated with overweight. First, survival of the fittest early on eliminates the overweight with severe diseases and leaves those who are more robust to mortality in the surviving sample. This selection process may operate more strongly in the socially disadvantaged group that is subject to premature deaths from additional conditions and causes. The protective effect of overweight can thus become more pronounced for this group. Second, overweight may confer actual benefits for survival as body mass can act as important nutritional reserves in the face of severe physiological and environmental stresses (Janssen, 2007; Oreopoulos et al., 2008).

This protection effect is particularly important in old age as the incidence and prevalence of chronic diseases becomes relatively high, and among lower socioeconomic groups (e.g., Black men, people in the lowest income strata) as their useful resources (e.g., food, nutrition, health care) are very few. The fact that class I obesity but not overweight has a protection effect for Black women may indicate that they have even more limited access to valuable resources than Black men. Third, unobserved diseases and illnesses may contribute to both weight loss and increasing mortality risk. Although we have controlled 14 chronic conditions and smoking status, there could be other unobserved and unmeasured confounding diseases.

Further subgroup analysis also finds that while class II/III obesity does not increase the mortality risk among Black men and women and adults age 70 and over, which has also been observed in some other studies (e.g., Reynolds et al., 2005; Stallard, 2010), it does lead to higher mortality risk among Non-Hispanic White women and men, young and middle-aged adults ages 30–69, people with higher education and more income. The PAF analysis further suggests that morbid obesity (BMI $\geq 35$) does account for a sizable portion of premature deaths in the young and middle aged and that its proportionate contributions to overall deaths increase with levels of education and income.

One possible explanation for these phenomena is that class II/III obesity is especially harmful for healthy people who have fewer competing mortality causes than unhealthy people. For example, obesity was found to increase the mortality risk by at least two to three times among those who are aged 50 and have never smoked (Adams et al., 2006) and to a larger extent among people without preexisting disease (e.g., cancer, heart disease) than those with these diseases (Calle et al., 1999). Appendix I restricts the analysis to people with “good,” “very good,” or “excellent” self-reported health or people without any preexisting chronic condition. Class II/III obesity confers greater HRs of 1.57 ($p < .001$) and 1.75 ($p < .01$) for these healthy subgroups than for the whole sample (1.42; $p < .001$), which supports this explanation that class II/III obesity may be especially harmful for healthy people who have fewer competing mortality causes. Another explanation is that these results are just statistical artifacts. Healthy people have fewer confounding diseases which are linked to both decreased weight and increased mortality risk. Ignorance of these confounding diseases will then lead to an underestimation of the effects of obesity (Manson et al., 2007). Therefore, we tend to obtain larger effects of obesity in healthier samples. This explanation implies that obesity is not particularly more harmful for healthy people, but it is more likely to get an accurate estimate of the obesity impact by focusing on healthy people. Given the wide array of comorbid conditions adjusted in the analyses, this may be a less plausible explanation. But it is still likely that the most disadvantaged group may underreport their comorbid conditions due to the lack of health care access, which may lead to an underestimation of the obesity effects in this group. Future studies are needed to test these possible explanations.

This study uses high quality data to investigate the population heterogeneity with respect to the mortality consequence of excess body mass and overall adiposity. We find that being overweight protects people with fewer resources and at relatively higher mortality risk, e.g., older adults, Non-Hispanic Black men, and lower income groups. Class I obesity is not linked to higher mortality risk except that it reduces mortality risk for Black women. Class II/III obesity does increase mortality risk among Non-Hispanic White women and men, young and middle-aged adults, and people with higher socioeconomic status. Our estimate of Class II/III obesity-attributable mortality in the total sample between year 1988 and 2006 are close to Metha and Chang’s (2009) estimates based on the Health and Retirement Survey 1992–2004 and are much smaller than some previous estimates (e.g., Allison, Fontaine, Manson, Stevens, & VanItallie, 1999; Mokdad et al., 2004, 2005). But that for the young and middle-aged sample does show a sizable percentage of premature deaths in this age group. As the class II/III obesity group continues to increase its proportionate share in the population, its attributable mortality percentage may increase in the future. The degree of this increase will, of course, depend on the size of the mortality risk associated with morbid obesity. If this association weakens over time, its attributable mortality percentage may not increase as much even if the proportion of morbidly obese population increases.

Although this study suggests only modest impact of obesity on mortality and, consequently, life expectancy in this period, the disability-free life expectancy will still be significantly affected by the increasing prevalence of obesity. Alley and Chang (2007) find an increasing functional impairment prevalence among obese people and increasing correlation between obesity and disability from 1988 to 2004, which may be partly induced by prolonged life expectancy among them (Doshi, Polsky, & Chang, 2007). Although overweight protects some from mortality, it may also be linked to higher risks of inflammation, metabolic and cardiovascular disorders as compared to normal weight (Zajacova, Dowd, & Burgard, 2011). Therefore, morbid life expectancy may actually increase in the future among the overweight and obese.

One limitation of this study is the relatively simple measure of obesity. Although BMI is the most commonly measure of adiposity, it has been criticized as not being able to directly measure the body fat and muscle composition and distinguish between central and peripheral adiposity (Oreopoulos et al., 2008). Some research reported inaccuracy of BMI as a measure of obesity in older people (e.g. Heitmann, Erikson, Ellsinger, Mikkelsen, & Larsson, 2000; Visscher et al., 2001) and those with heart failure (Oreopoulos et al., 2011). Future studies can consider more accurate and direct measures of body composition that may be available in other data. For example, the DEXA (Dual Energy X-ray Absorptiometry) in NHANES IV (1999–2004) measures muscle and fat. The challenge is to link such measure with subsequent mortality in a sufficiently long period of follow-up time.

The population heterogeneity in the mortality response to overweight/obesity has not been systematically investigated in the extant literature but can cause serious problems in inference and interpretations of the findings. For example, if some groups (e.g., young adults, White, those with higher education or greater income) are over-represented in the sample, we may not be able to detect the protective effect of overweight but overestimate the deleterious impact of class II/III obesity. And the reverse may occur if they are underrepresented in the sample. Future studies should pay special attention to the sociodemographic diversity of the study population and clearly acknowledge the limitations in the generalizability of results due to the given sample characteristics.
Appendix I. Mortality hazard ratios for overweight and obesity relative to normal weight for people with excellent/very good/good health or without any chronic condition from weighted Cox proportional hazard model

<table>
<thead>
<tr>
<th>Normal weight (18.5–24.9)</th>
<th>People with excellent/very good/good health</th>
<th>People without any chronic condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.00 [Ref.]</td>
<td>1.00 [Ref.]</td>
<td></td>
</tr>
<tr>
<td>Overweight (25.0–29.9)</td>
<td>1.05 (0.92, 1.21)</td>
<td>1.07 (0.94, 1.21)</td>
</tr>
<tr>
<td>Class I obese (30.0–34.9)</td>
<td>1.18 (1.04, 1.34)</td>
<td>1.23 (1.09, 1.39)</td>
</tr>
<tr>
<td>Class II/III obese (35.0+)</td>
<td>1.30 (1.13, 1.50)</td>
<td>1.34 (1.16, 1.56)</td>
</tr>
</tbody>
</table>

Sample size 5068

Note: All analyses have controlled age, age squared, sex, race, education, insurance, smoking status. *p < .05; **p < .01; ***p < .001.

References


