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Is the High Level of Obesity in the United States Related to Its Low Life Expectancy?

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Abstract

This paper investigates the effect of obesity on the life expectancy gap between the United States and other developed countries. We perform primary analyses of survey data to identify the distribution of body mass index (BMI) by age and sex in 16 countries. The United States has the highest proportion obese of any population considered. These BMI distributions are combined with three alternative sets of mortality risks by BMI in order to estimate the proportion of deaths attributable to above-optimal weight, by age and sex. These estimates are then converted into their implications for longevity. Our baseline analysis uses the largest, longest, and most internationally-diverse collection of obesity risks, the Prospective Studies Collaboration. Using this set of risks, we estimate that US life expectancy at age 50 in 2006 was reduced by 1.29 years for women and 1.61 years for men as a result of obesity. Using obesity risks that were recorded more recently in the United States, the reduction is in the range of 0.6-0.9 years. Even using these lower risks, we find that differences in obesity account for a fifth to a third of the shortfall of life expectancy in the US relative to longer-lived countries.

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According to World Health Organization estimates, men and women in the United States had a higher prevalence of obesity in 2005 (defined as a Body Mass Index of 30.00 or higher) than any other country in Europe, North America, or East Asia (World Health Organization 2005). At the same time, life expectancy in the United States has fallen below that of most other OECD countries and ranked 32nd in the world in 2008 (World Health Organization 2010).

The purpose of this paper is to identify the extent to which these two phenomena are related to one another. In our baseline analysis, we use the mortality risks associated with various detailed age/BMI categories that were developed in a large international review that synthesized data drawn primarily from North America and Western Europe (Prospective Studies Collaboration 2009a). We apply these risk factors to the distribution of population by BMI in 16 countries during a recent year in order to estimate the fraction of deaths attributable to obesity, by age and sex, beginning at age 50. We recalculate life tables for these countries after removing deaths attributable to obesity to identify the extent of international variation in life expectancy that is attributable to differences in BMI distributions. We explore the sensitivity of results to the assumed set of risks associated with overweight and to misreporting of height and weight.

Methods and Data

In our baseline analysis, we assume that the relative mortality risks in various BMI categories by age and sex that were recorded in a synthesis of 57 prospective studies are applicable to all countries considered (Prospective Studies Collaboration (PSA) 2009a). This is the largest, most recent, and most detailed of several large compilations of data on obesity and mortality (see also Yusuf et al. 2005; McGee & Diverse Populations Collaboration, 2005). The synthesis includes data on 895,000 participants, of whom 63% were from Europe and Israel, 29% were from the US and Australia, and 8% were from Japan. Authors report that the relative risks identified from ischemic heart disease, the principal cause of death through which obesity operates, do not differ significantly between Europe and the US and Australia and that there is no significant heterogeneity across studies in risks of death from stroke or ischemic heart disease. To limit reverse causality, this study excluded the first five years of follow-up, leaving 67,000 deaths of known cause. Associations between baseline BMI and mortality were adjusted for baseline smoking status. BMI values were established through actual measurements except in three studies of US medical personnel, where self-reports were used. Results of this

investigation have been presented by sex, age group, and detailed BMI categories (Prospective Studies Collaboration 2009b).¹

Estimates of the population distribution of BMI were obtained through primary analysis of nationally representative survey data, adjusting for features of survey design. The primary criterion for choice of comparison countries was simply data availability, although countries with higher life expectancies than the US were of particular interest. Estimates were derived, where possible, from the most recently available data. In some countries, survey-years were combined to increase sample size. Height and weight data for estimating an individual's BMI are based on self-report except in England, where only measured values were available, and in the United States and Canada, where both self-reported and measured values are available and used. The majority of data used here were products of the Survey of Health, Ageing and Retirement in Europe (SHARE). This data source is limited to ages above 50, hence the focus of this paper on this age interval. Since 94% of newborns survive to age 50 in the current US life table (Arias et al. 2010), variations in life expectancy at birth are dominated by variations in life expectancy at age 50. Uniform restriction criteria were employed across surveys. The Appendix provides information on sources of data used in this study, including the surveys and years from which data for each country were drawn, sample sizes, features of survey design for which adjustment was made and sample restriction criteria.

Data for constructing period life tables, including deaths and population, were obtained by country, age and sex in single-year age-intervals for 2006, the most recent year for which such data were available for the whole set of countries, from the Human Mortality Database (2010). Conventional methods of calculating life tables were used (Preston, Heuveline, and Guillot 2001).

We exploit the full detail in which risk information is available by constructing estimates of BMI prevalence in the same age-sex-BMI groupings used by the Prospective Studies Collaboration, choosing open-ended categories of BMI<17.5 and BMI≥35.. To identify the proportion of deaths in a particular age/sex category that are attributable to obesity, we hypothetically redistribute the population above the optimal BMI category (i.e., the lowest-mortality category) in a particular age/sex group to the optimal category and calculate the hypothetical death rate after this redistribution. In this thought experiment, we do not change the proportion of persons *below* the optimal BMI category because our interest is in the effect of obesity on mortality.

The proportion of deaths attributable to obesity in population i at a particular age (AF_i) is thus estimated as

$$AF_i = \frac{\sum (C_{ij} M_{sj} - C_{ij}^* M_{sj})}{\sum C_{ij} M_{sj}}, \text{ where} \quad (1)$$

¹ These categories are 2.5 unit intervals between 15-50 kg/m² with the exception of the highest BMI category which spans BMI values between 35-50 kg/m². Estimates are disaggregated by sex for age groups 35-59, 60-69, 70-79 and 80-89.

C_{ij} = proportion of population i in BMI category j

M_{sj} = death rate in BMI category j in the “standard” drawn from PSC data

C_{ij}^* = proportion of population i in BMI category j if all those above the optimal BMI were redistributed to the optimal category²

Equation (1) would give the same value of AF_i if the death rates were in the form of relative risks, e.g., if numerator and denominator were divided by the death rate in the optimal category. By referring to the value produced by equation (1) as the “proportion of deaths attributable to obesity”, it should be clear that we are using the term, obesity, to comprise all weight categories above the optimal.

A key assumption underlying our procedure is that the association between BMI and mortality in the PCS study is causal, reflecting the effect of BMI on mortality. If it is causal, then a redistribution of the population would in fact be expected to change the population’s death rate by the amount indicated in equation 1 (although as Mehta and Chang (2009) point out, it would doubtless not happen instantaneously). A reason to believe that the association may be causal is that BMI is working primarily through cardiovascular diseases, where the physiological connections between BMI and blood pressure, lipoprotein levels, and diabetes would be most likely to manifest themselves (PCS 2009a). In the range of BMI of 25-50 kg/m² each increment of 5 kg/m² was associated with an increase in mortality from heart disease and stroke of 40%, compared to an increase of only 10% in cancers (Ibid.). Relative to other causes of death as an aggregate, a greater responsiveness of cardiovascular mortality to increases in BMI above the optimal category has been demonstrated in many other studies as well (e.g., Calle et al. 1999, Flegal et al. 2007). Below, we consider several possible biases that could affect the results of applying equation (1).

The country- age- and sex- specific attributable fractions are applied to period life table mortality rates in single-year age intervals to estimate what these rates would be if no one were obese. Life expectancy at age 50 is then calculated using the modified death rates. Hypothetical life expectancies obtained in this manner are then compared to the actual values by country and sex. Finally, the effect of eliminating obesity on the life expectancy *gap* between the US and other countries is computed as the change in the life expectancy difference that results from removing obesity-attributable deaths.

Analysis of uncertainty was conducted for attributable fractions and life expectancy estimates. Uncertainty estimates from two sources are combined: uncertainty in the BMI data

² The optimal BMI category for a particular age and sex combination was defined as the category in which the lowest all-cause mortality rate was observed. For males, the lowest risk category was 22.50-24.99 kg/m² for age-groups 50-59, 60-69 and 70-79 and 20.00-22.49 kg/m² for age-group 80-89.. For females, 22.50-24.99 kg/m² was the lowest risk category for age-groups 50-59, 60-69 and 80-89 and 25.00-27.49 kg/m² for the age-group 70-79. We apply the PSC mortality values for ages 35-59 to ages 50-59. The mortality risk from obesity is assumed to be zero above age 90.

resulting from sampling variability and uncertainty in estimation of the relative risks. Bootstrapping is used to address the former uncertainty: for a given country, age and sex, BMI values are sampled randomly with replacement as many times as there are non-missing observations on BMI in that country/age/sex category. To estimate the latter uncertainty, a vector of relative risks of length corresponding to the number of BMI intervals is drawn from independent log-normal distributions with age and sex-specific means and standard deviations as published in Web-Appendix 1 of the PSC study.³ The resulting vector of risks is applied to the simulated BMI distribution data to obtain an age and sex specific estimate of the attributable fraction. These steps are repeated 999 times to obtain 1000 estimates of the age and sex specific attributable fraction, from which the 2.5th and 97.5th percentile values are extracted as 95% confidence intervals. To obtain confidence intervals on the other attributable fractions, the above procedure is repeated for the other country, age and sex combinations. Uncertainty intervals for estimates of life expectancy in the absence of obesity were obtained in a similar fashion.

Analyses were conducted using STATA 10.1 (StataCorp, Texas, USA) and R 2.10.1 (The R Foundation for Statistical Computing).

Results

Table 1 presents the proportion of persons who are in or above the standard BMI categories of overweight (BMI 25-29.99), Obese Class I (BMI 30-34.99), Obese Class II (BMI 35-39.99) and obese class III (BMI 40+). The proportion of individuals exceeding thresholds for class I, II and III obesity is higher in the US than in comparison countries for both males and females. Consistent with other studies (e.g., Ezzati et al. 2006), the proportion of individuals in high BMI categories in the US is greater when measured versus self-reported data on BMI are used. A similar pattern is found in Canada, another country in which both types of data were available.⁴

Countries with the lowest prevalence of obesity include Denmark and Sweden for males and Denmark and Switzerland for women. France has an exceptionally low proportion with BMI above 35 for both men and women; low obesity in France has been attributed to unusually small portion sizes (Rozin et al. 2003). Figure 1 shows smoothed frequency distributions of body mass index by sex based on self-reports for the US and a set of countries selected for

³ Standard errors of relative risks were not directly available and were therefore derived in approximation from reported uncertainty intervals assuming normality of the log-risks.

⁴ Using NHANES and BRFSS data in the US, Ezzati et al. (2006) showed that self-reported BMI elicited through telephone surveys tended towards higher misreporting than when elicited through in-person surveys. Approximately half the Canadian BMI data were obtained through telephone interviews while US NHANES data are from in-person interviews, providing a likely explanation for higher levels of misreporting in Canada relative to the US. See Appendix Table 1 for information on mode of interview by country.

comparison. The US distribution has larger variance and is markedly right-skewed with respect to the comparison countries (standard deviations: 5.95 vs. 4.31; skewness coefficients: 0.99 vs. 0.76 for the US and the average of the other countries).

Fractions of all-cause mortality attributable to obesity (attributable fractions) by country, age and sex are presented in Table 2 including confidence intervals that reflect uncertainty in estimates of the proportions of individuals obese and risks associated with obesity. For the US and Canada, attributable fractions are reported for both measured and self-reported values of height and weight, while in England the fractions are exclusively reported for measured values. For the US and Canada, use of measured versus self-reported values of height and weight leads to attributable fractions that are higher by approximately 3% and 5%, respectively.

Among males, the attributable fractions tend to decline slightly between ages 50-59 and 60-69 and then to decline more rapidly thereafter. Among women, the attributable fractions invariably rise between 50-59 and 60-69 and then declines thereafter. With few exceptions, Americans have the highest attributable fractions at all ages for both sexes. The discrepancy between the attributable fractions in the US and other countries is typically greatest for both men and women at ages 50-59, reflecting the unusually large proportions obese in the US at that age.

These estimated fractions of deaths attributable to obesity have important implications for life expectancy. Table 3 presents life expectancy gains implied by the estimates of deaths attributable to obesity presented in Table 2. Reallocating individuals with higher-than-optimal BMI to the lowest-risk BMI for their age and sex would increase life expectancy at age 50 in the United States by 1.29 years [95% CI] for women and by 1.61 years [95% CI] for men when self-reported BMI data are used. In both cases, hypothetical gains in life expectancy in the US exceed those of any other country. French women and Swedish men have the least to gain for their sex. In every country, men would gain more years of life than women from the elimination of obesity.

Because attributable fractions in the US are larger using measured than self-reported BMI, the estimated gains in life expectancy from eliminating obesity are greater by 0.24-0.27 years when measured BMI is used. An even larger difference in life expectancy gain is observed for Canada, the only other country where the two sets of BMI values can be compared.

Table 4 presents the US shortfall in life expectancy at age 50 and the estimated change in that shortfall if obesity were eliminated. The comparisons are made only to countries with higher life expectancies. Since life expectancy at age 50 in the US would increase significantly more than in other countries through the hypothetical elimination of obesity, the US shortfall would be reduced and in some cases eliminated. US life expectancy for women is 1.37 years lower than the mean of the 12 other countries. It would be an estimated 0.81 years lower without obesity, so that obesity accounts for an average of 41% of the gap. For men, the

equivalent fraction of the difference in life expectancy accounted for by obesity, relative to 10 higher life expectancy countries, is 67%.

On the basis of the PSC risk factors combined with the distribution of BMI in various countries, we would conclude that obesity is a major contributor to the low ranking of US life expectancy.

Potential Sources of Bias in Results

A) Measurement Error

The above calculations assume that BMI is recorded accurately in all countries and that the relative risks of various BMI categories estimated in the Prospective Studies Collaboration are applicable to all countries included in the comparison. The fact that the estimates using BMI are different from those using self-reports in the US and Canada is an indication that self-reports are not free of error. Analysis of NHANES data shows that American women tend to underestimate their weight, particularly at ages 65 and below, while both men and women tend to overestimate height at older ages (Ezzati et al. 2006). The result of these tendencies is greater bias in estimates of body mass index for women than for men and somewhat greater bias at older ages than at younger ages. Larger errors are observed for individuals in the lower and upper extremes of height and weight. A source of systematic error at the population-level is related to the mode of eliciting height and weight data, e.g., in-person versus telephone interviews (Ezzati et al. 2006).

Even if reporting patterns were the same in two countries-- i.e. if people with a particular measured BMI had the same propensity to report themselves in various BMI categories-- there may still be different effects on reported distributions because underlying true distributions were not identical. For example, suppose country A had a higher true proportion in BMI category 25.00-29.99 than country B. Suppose further that the probability of someone in that category reporting that they were 30.00+ were the same in the two countries. Then country A's reported proportion with 30.00+ would be inflated more than country B's by virtue of such misreporting.

We conduct an analysis of the robustness of our findings to errors in self-reported height and weight. Using data from NHANES III on participants with measured and self-reported values of height and weight, Cawley and Burkhauser (2006) model the relationship between measured and self-reported values of height and weight. Models predicting measured values are quadratic in the self-reported values of height and weight as well as in age and are stratified by sex and race/ethnicity (white, black, Hispanic) (see also Michaud et al. 2006).

We assume that the pattern of misreporting of height and weight is systematically related to age, sex and self-reported values of height and weight but not related to geographic context. . Using data from NHANES 2003-2008, we estimate correction equations for individual-

level misreporting of height and weight. We use similar specifications to those of Cawley and Burkhauser (2006); however, regressions are not stratified by race/ethnicity because such stratification is inappropriate in the countries to which the correction equations are to be applied. Mode of interview was similar across data sources, with the exception of Canada, so we do not adjust for it (Appendix Table 1). Coefficients of these equations appear in Appendix Table 2.

Self-reported height and weight in each country in the sample are corrected using the correction equation developed above.⁵ All findings of the paper are reproduced using the adjusted body mass index data. As shown in Table 5, the difference between actual life expectancy at age 50 and life expectancy if obesity were eliminated increases by 0.23 years for US females and by 0.20 for US males when adjustment is made for misreporting. The estimated effect of eliminating obesity also increases in other countries, although by less than in the US. International differences in the effect of this adjustment are not large: the greatest difference occurs between the US and Spain, amounting to 0.10 years for women and 0.17 for men (Table 5). No other differences in the table reach a level of a tenth of a year of life expectancy.

We conclude that errors in self-reported BMI have produced underestimates of the impact of obesity on life expectancy, and that the underestimate is somewhat greater in the US than in most other countries. In this sense, obesity explains more of the gap in life expectancy between the US and other countries than is indicated by self-reports. However, the bias is modest, amounting in only one case to a value larger than 0.10 of the life expectancy gap between US and other countries.⁶

These results pertain to error introduced by bias in self-reports of BMI. Error can also be introduced into our estimates by *random* error in the reporting of BMI. If BMI is measured with error in the studies summarized in the PSC (2009a and b), it is likely to produce “regression dilution bias”, according to which coefficients relating mortality to BMI are biased towards zero (Frost and Thompson 2000). The fact that almost all of the studies reviewed in the PSC used

⁵ The correction equations, when applied to US self-report height and weight data, approximately reproduce the US measured distributions of height and weight. The correction is not exact because the US training data set only including the subset of the US self-report sample for whom clinical measurement of height and weight were also obtained. Before correction for misreporting the percentage of men in the US with BMI 30 kg/m² and above was 32.6 compared to 36.2 using measured values of height and weight (Table 1). After correction, the value increased to 36.1, reducing the discrepancy between self-report and measured values from approximately 3.6 to 0.1% (data not shown). For women the values based on self-report and measured data before correction were 33.5 and 38.6% and the value based on self-report increased to 38.2 after correction, reducing the discrepancy between the estimates from 5.1 to 0.4%. Note that we do not attempt to conduct an out-of-sample validation of the correction equation using Canadian self-reported BMI data against “truth” in that country because that mode of interview differed between Canada and the rest of the sample countries in a manner known to affect patterns of misreporting (see Appendix Table 1) (Ezzati et al. 2006).

⁶ It is possible that we have underestimated the impact of misreporting by using NHANES data to establish correction factors. In NHANES, individuals are aware at the time of the survey that they will also be subjected to clinical measurement of their height and weight, and therefore may be less likely to misreport values relative to individuals in other surveys in which clinical measurement does not follow interview (Ezzati et al. 2006).

actual measures of BMI rather than self-reports reduces the risk of bias from this source. If other measures of adiposity such as waist circumference are more precise predictors of mortality-- a possibility that has frequently been raised (e.g., Alley, Lloyd, and Shardell 2010)-- then using BMI itself produces a form of regression dilution bias with respect to the underlying effects of adiposity.

B) Correlations Among Risk Factors

Apart from error in measuring BMI, there are many forms of potential bias in the set of mortality risks associated with particular BMI levels. A well-recognized form of bias occurs when another variable affects mortality and is correlated with BMI but is not adequately controlled in the research design. One major variable affecting mortality is cigarette smoking. At an individual level, current smokers tend to have lower BMI and higher mortality than non-smokers (Gregg et al. 2005; Adams et al. 2006; Prospective Studies Collaboration 2009a). Like smoking behavior itself, lung cancer mortality tends to be lower among the obese (Flegal et al. 2007). Thus, failure to control smoking behavior can lead to an underestimate of the impact of obesity on mortality.

The risk factors in the Prospective Studies Collaboration are controlled for smoking behavior. The authors report that the risks of obesity and smoking are “roughly additive” (PSC 2009a: 1083). Another meta-analysis of the mortality risks from obesity explicitly tested the significance of the interaction between BMI and smoking in their effects on mortality and found it to be insignificant (McGee 2004: 94). Two large US studies found higher relative risks of obesity among non-smokers than among smokers, but neither explicitly tested the significance of the interaction (Adams et al. 2006; Calle et al. 1999). We have implicitly assumed that the risks of obesity and smoking are additive, which enables us to apply a common set of obesity risk factors to all countries regardless of their smoking prevalence. Because of its history of heavy smoking, mortality in the US shows an unusually large smoking imprint (Preston, Gleib, and Wilmoth 2009). Thus, if obesity risks are actually greater among non-smokers than among smokers, the assumption of additivity may lead to overestimates of the impact of obesity in the US relative to other countries.

Lack of exercise is another variable whose effects can be reflected in obesity risk factors. Obese people maintain, on average, lower levels of exercise (Adams 2006; Koster et al. 2009). A number of studies have demonstrated that obesity and lack of exercise have independent effects on mortality (F. Hu et al. 2004; Koster et al. 2009; G. Hu et al. 2004). The United States not only has the highest level of obesity among OECD countries but the highest proportion of sedentary individuals among countries where exercise levels have been reported (Steptoe and Wickman 2010). But the confounding effect of lack of exercise is not like that of other risk factors because one becomes obese when caloric expenditures fall short of caloric intake. Lack of adequate exercise is intrinsic to becoming obese. The PSC risk factors for obesity are not

controlled for exercise levels. As a result, risks attributable to obesity in our study comprise as well the hazards of inadequate physical activity. But because of the causal relation between obesity and physical activity, this inclusion appears to be less of a bias than an interpretive complexity.

A third variable capable of distorting the measured risks associated with obesity is social class or socioeconomic status. This concept is actually a cluster of conditions and behaviors that are related to health such as access to health care, adequacy of nutrition and shelter, occupational hazards, knowledge and practice of proper health behaviors, skill in dealing with the medical system, compliance with therapeutic regimes, and so on (Marmot, 2004 ; Preston and Taubman 1994). Social class has been shown to be strongly related to mortality in hundreds of studies. Because people in lower social groups are more likely to be obese (Chang and Lauderdale 2005; Sanchez-Vaznaugh et al. 2009), failure to control socioeconomic status can bias the estimated risks from obesity. Discussion of this set of variables is oddly missing from much of the literature on obesity, although certain socioeconomic variables are sometimes controlled. Mehta and Chang (2009) report that controlling for social class variables in the US Health and Retirement Survey reduced the excess risk for BMI levels of 35 and above from 68% to 32% for women and from 45% to 34% among men. Socioeconomic status is not controlled in the PSC study and estimated obesity risks are likely to be biased upwards as a result. The bias may be reduced by the control for smoking, one of the most consequential covariates of social class (Jha et al. 2006).

A prolonged process of weight loss that is often associated with chronic illness can also create a bias in estimated effects of obesity. Since people with a major illness often lose weight because of their sickness, the group of people with lower levels of BMI may contain a disproportionately large number of people who are ill and hence have high mortality risks. Two common ways of dealing with this bias are to exclude people who are reported to be ill at baseline or to delay the counting of deaths and exposure for some number of years beyond the baseline. Adams et al. (2006) demonstrate that large increases in the estimated risk of obesity can occur when either of these strategies is employed. However, no major changes in risk estimates were recorded when these restrictions were imposed on analyses of NHANES or Health and Retirement Survey data (Flegal et al. 2007; Mehta and Chang 2009). In the Prospective Study Collaboration, the second of these strategies was employed: the counting of deaths and exposure was delayed for five years beyond baseline.

C) Possible Trends in the Risk of Obesity

Probably the greatest drawback to using the PSC obesity risk factors is the possibility that the risks from obesity have been declining. Such a decline was reported by Flegal et al. (2007) using successive waves of NHANES. Applied to cardiovascular deaths in the US in 2004, NHANES I risk factors (including exposure from 1971 to 2000) produced 161,290 BMI-

attributable deaths, whereas risks from NHANES III (1988-2000) produced only 46,915. However, the NHANES I risks did not depend on the length of follow-up (15 years vs. 20 vs. 25), an observation that confounds time trends with duration-of-observation effects. In a large study begun in 1982 by the American Cancer Society with follow-up of healthy non-smokers through 2002, no decline was observed in the mortality risk from obesity (Calle, Teras, and Thun 2005). A reduction in obesity risks in the US was identified by Mehta and Chang (2010) in three different data sets including NHANES, the Framingham study, and a National Health Interview Survey follow up study. Three of the six time trends involving two obese categories and three studies were statistically significant. Mehta and Chang (2009) report no significant trend in obesity risks in Health and Retirement Survey participants aged 50-61 who were followed up from 1992 to 2004.

Gregg et al. (2005) provide reasons why the cardiovascular risk associated with obesity may have declined in the US. The prevalence of high cholesterol, high blood pressure, and smoking in successive waves of NHANES declined sharply between 1960-62 and 1999-2000 among obese persons, and the cholesterol decline was significantly faster than among people with lower BMIs. Declines in smoking and high blood pressure were not significantly faster among the obese. The use of lipid-lowering and antihypertensive medications increased rapidly over this period, particularly among the obese, who had a significantly faster increase in antihypertensive drug use than the non-obese. On the other hand, the prevalence of diabetes remained relatively constant over time within BMI groups (*ibid.*) Disability grew more rapidly among the obese than the non-obese between 1988-94 and 1999-2004 (Alley and Chang 2007).

An additional factor that may have reduced relative risks among the obese is the rapid inflow of people into the obese category. A rapid increase such as occurred in the United States (Flegal et al. 2010) implies that the average duration of obesity for an obese person was declining. To the extent that there are duration effects of obesity- risks that cumulate with length of time spent in the stage- the risk of obesity per se should have declined when duration is not accounted for in the research design. Duration effects have been observed in many risk factors (Danaei et al. 2009). The fact that childhood or early adulthood obesity is highly predictive of adult mortality implies that duration effects may be important for obesity (Franks et al. 2010; Gavrilova and Gavrilov 2010).

Finally, that death from cardiovascular disease is a diminishing proportion of all mortality (Beltran-Sanchez et al. 2008), combined with greater obesity risks from cardiovascular diseases than from the aggregate of other causes of death, also implies that the all-cause mortality risk from obesity should be declining.

In order to investigate the effect of a possible reduction in obesity risks on international comparisons, we introduce two alternative sets of risk factors that apply to more recent periods than the PSC results. One alternative set of relative risks is adapted from Adams

et al. (2006). These are derived from a large study of 527,000 enrollees in the NIH-AARP Diet and Health Study that was conducted in six US states and two cities. Enrollees were followed from enrollment in 1995-96 through the end of 2005. The study provides detailed information on mortality risks by age and self-reported BMI. Relative risks are adjusted for smoking, social status and physical activity. Excluding individuals with pre-existing conditions did not affect relative risks already stratified by age. We use the published results of this study to estimate relative risks in the age categories that were employed in the main analysis reported above. To do so, we fit a linear age-trend using weighted least-squares to risks that were originally reported in age intervals 50-65, 56-70, 61-75, and 66-81. We assume again that obesity risks are zero for ages 90 and above. From primary data, we re-calculate the proportions in various BMI intervals in each country to align with the categories used by Adams et al. Again, we use equation (1) to estimate attributable fractions and use the new attributable fractions to modify life expectancy calculations.⁷

The second alternative set of risk factors is derived from NHANES III linked to death certificate data (Mehta and Chang 2010). Initial enrollment at ages 50-69 occurred during 1988 to 1994; individuals were followed into the National Death Index through 2006. The sample size was 4375 individuals and mean follow-up time was 13.3 years. Advantages of the study include recent data, a probability sample of the US population, and a relatively long follow-up period. A disadvantage is the small sample size, which limited the estimation of risk factors to all ages and both sexes combined. Relative risks were adjusted for smoking and socioeconomic status and were not found to be sensitive to the exclusion of individuals with pre-existing conditions (Mehta and Chang 2010). Relative to those of normal weight (BMI 18.5-24.9), overweight people (25.0-29.9) had a 6% added risk, Class I obese people a 1% added risk, and people in combined obese Classes II and III had a 63% added risk. We have applied these relative risks to all ages (50-89) and both sexes.

Results of using these risk factors are presented in Table 6 and in Figures 2a and 2b. In every country for both sexes, the use of the alternative risk factors reduces the estimated gain in life expectancy from eliminating obesity. Using the Adams et al. risk factors, the mean gain is only 44% as large for females and 23% as large for males as the gains from using the PCS risks. Using Mehta/Chang, the gain is 43% as large for women, on average, and 38 % as large for men. Proportionate reductions are not as large in the United States as in other countries because a much higher fraction of the US population resides in obesity Classes II or III, where risks remain considerable under the alternative sets of risks.

Confining comparisons in Table 6 to countries with higher life expectancies than the US, as in Table 4, the mean life expectancy gap for the 12 countries with higher life expectancies is

⁷ The optimal category used in all cases was 23.5-24.9 kg/m².

reduced by 29% for women using Adams' risks to account for the effects of obesity and by 22% using Mehta/Chang's. For men, the equivalent reductions are 33% and 29%.⁸

Thus, differences in the prevalence of obesity continue to explain about 20-35% of the shortfall in US life expectancy relative to countries with superior levels, even when we use much lower sets of obesity risks.⁹ Only if the Adams or Mehta/Chang risk factors were applicable to the US and the PSC risk factors were applicable to other countries would obesity differences play no role in accounting for these mortality disparities. But there is no evidence that such a difference in risk factors exists.

Discussion

Olshansky (2006) has produced estimates of the effect of obesity on US life expectancy using NHANES III risk factors. He does not use the full BMI distribution but rather experiments with various binary specifications of risk, producing estimated effects on US life expectancy that range widely from one-third to three-quarters of a year. Our estimates in Table 6 are at or above the high end of that range for all of the sets of risk factors employed, and are much higher when PSC risks are used. Fontaine et al. (2003) compute the effect of obesity on life expectancy based on NHANES II but their results are not comparable to ours. They compare the life expectancy of an obese person who remains obese to that of an optimal-weight person who remains of optimal weight. Theirs is an individual-level calculation rather than a population-level calculation that recognizes the fraction of individuals in various BMI categories at particular ages.

In order to study the impact of obesity on international differences in longevity, we have estimated the effects of obesity on longevity not only in the US but in 15 other countries as well. We have explored the sensitivity of international comparisons to error in self-reports and to variation in the mortality risk from obesity. We conclude that, even under relatively mild risks, the high levels of obesity in the US contribute substantially- in the neighborhood of 20-35%- to the inferior level of longevity in the US. If the risk factors from the Prospective Studies Collaboration are used, the impact of obesity is substantially larger, accounting for 38% of the longevity shortfall for US women and 69% for US men.

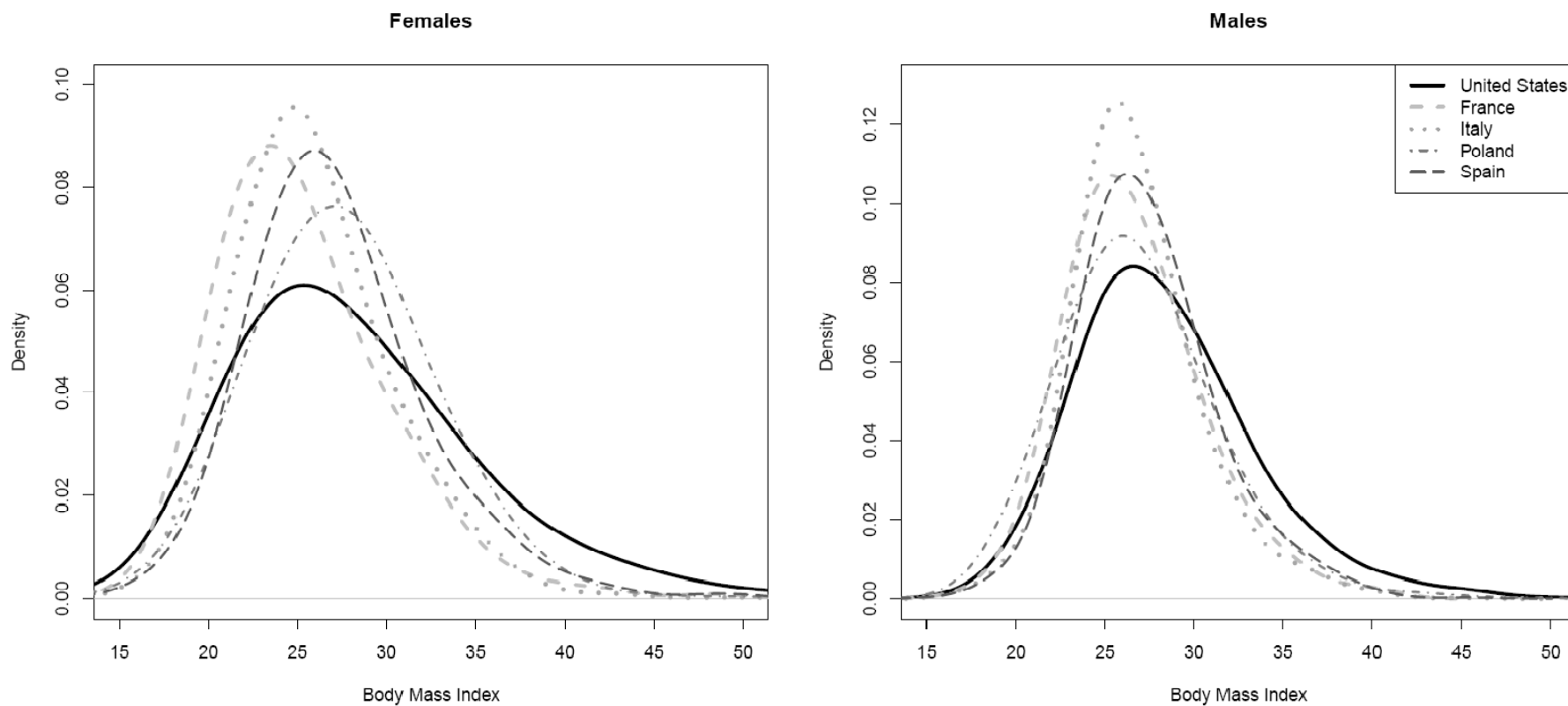
⁸ As in Table 4, this comparison uses self-reported values for Canada and uses the measured US value in the comparison with England.

⁹ Recall that, by construction, Mehta/Chang risks are identical for men and women. Noting that the prevalence of obesity is greater for women than for men in nearly all countries (Table 1), one might assume that using Mehta/Chang risk factors would produce a larger effect on women's than on men's longevity in Table 6. The reason that such an outcome is not observed is that men die at a much faster rate than women at all ages in all countries, so that the same proportionate reduction in death rates for both sexes would result in more life-saving for men than for women.

The risk factors derived from the studies of Adams et al. and Mehta/Chang have the advantage of pertaining to a later period, on average, than those of the PSC. The period is closer to the time when the levels of both obesity and mortality are recorded in the various countries and when attributable risks are modeled. These studies also control social class in their analyses of the impact of obesity on mortality. On the other hand, the Mehta/Chang study is small, and the Adams study is not derived from a probability sample and had a low response rate (18%). Both studies are confined to the US.

If there were a clear-cut trend in the mortality risk of obesity, there would be a strong reason to prefer estimates derived from the two most recent studies. But evidence of a trend is suggestive rather than definitive, since it has not appeared in all analyses where its presence has been investigated and it has not always been statistically significant when it has appeared. As a result, we believe that our results should be interpreted as providing a plausible range of estimates of the impact of obesity on the shortfall in American longevity. And the contribution is substantial regardless of the set of risk factors employed.

Figure 1. Smoothed frequency distributions of body mass index for men and women aged 50-89



Notes. Population distributions of BMI are age-standardized to the US Census population using age-groups 50-59, 60-69, 70-79 and 80-89 and weighted using sample weights. BMI data are derived from self-reported height and weight. Data: United States: 2003-2004, 2005-2006 and 2007-2008 cycles of the National Health and Nutrition Examination Survey (NHANES); France, Italy, Poland, Spain and Sweden: Survey of Health, Ageing and Retirement in Europe (SHARE), Waves I (2004) and II (2006-2007).

Table 1. Prevalence overweight and grades I, II and III obesity and above by country, adults 50-89.

Country	Percentages, Adults Aged 50-89 (95 % CI)							
	Females				Males			
	BMI ≥ 25	BMI ≥ 30	BMI ≥ 35	BMI ≥ 40	BMI ≥ 25	BMI ≥ 30	BMI ≥ 35	BMI ≥ 40
Austria	57.1 [51.8-62.2]	20.9 [16.4-26.2]	4.6 [2.7-7.9]	1.3 [0.6-3.1]	69.3 [65.0-73.3]	17.8 [13.6-22.8]	3.5 [2.3-5.2]	0.9 [0.4-2.1]
Belgium	53.8 [51.2-56.4]	17.9 [16.1-19.9]	4.3 [3.5-5.4]	0.9 [0.5-1.5]	63.5 [60.8-66.1]	17.4 [15.4-19.5]	2.9 [2.1-4.0]	0.4 [0.2-0.9]
Canada	52.9 [51.9-53.9]	18.7 [18.0-19.5]	5.7 [5.3-6.1]	1.9 [1.7-2.2]	65.1 [64.0-66.1]	19.9 [19.0-20.8]	4.1 [3.7-4.5]	0.9 [0.7-1.1]
Czech Republic	69.0 [65.7-72.1]	24.1 [21.5-26.9]	5.4 [4.2-6.9]	1.1 [0.6-2.0]	76.8 [73.4-79.9]	23.7 [20.6-27.1]	3.6 [2.4-5.2]	0.9 [0.4-1.9]
Denmark	43.7 [40.6-46.9]	13.2 [11.1-15.7]	3.4 [2.5-4.6]	0.9 [0.5-1.7]	59.3 [56.0-62.6]	13.0 [10.9-15.4]	2.0 [1.3-3.1]	0.4 [0.1-1.1]
France	46.1 [43.2-49.0]	14.3 [12.3-16.6]	3.4 [2.6-4.3]	1.1 [0.6-1.9]	63.0 [60.8-65.0]	15.6 [13.6-17.7]	2.3 [1.5-3.5]	0.2 [0.0-0.6]
Germany	55.5 [52.8-58.3]	16.2 [14.4-18.2]	4.7 [3.7-6.0]	1.3 [0.8-2.0]	69.8 [67.3-72.2]	16.3 [14.4-18.4]	3.3 [2.5-4.4]	0.7 [0.4-1.4]
Israel	60.5 [57.6-63.4]	19.4 [17.2-21.8]	4.9 [3.7-6.6]	1.2 [0.7-2.1]	67.2 [64.0-70.2]	16.8 [14.6-19.3]	2.7 [1.8-3.9]	0.6 [0.3-1.3]
Italy	56.0 [53.4-58.5]	17.3 [15.4-19.4]	3.7 [2.8-4.9]	0.7 [0.4-1.4]	69.0 [66.3-71.5]	15.5 [13.6-17.7]	2.9 [2.1-3.9]	0.4 [0.2-0.9]
Netherlands	53.3 [50.6-56.0]	16.6 [15.0-18.3]	3.9 [2.9-5.2]	1.3 [0.8-2.4]	61.7 [58.1-65.2]	13.4 [11.3-15.8]	2.5 [1.8-3.5]	0.5 [0.2-1.0]
Poland	69.1 [65.0-73.0]	29.2 [26.3-32.2]	6.6 [5.3-8.2]	1.3 [0.6-2.6]	65.0 [59.9-69.9]	20.9 [17.3-25.1]	3.8 [2.9-5.1]	0.8 [0.3-2.4]
Spain	67.1 [64.2-69.9]	24.9 [22.3-27.6]	7.7 [6.3-9.5]	1.8 [1.1-2.8]	73.4 [70.4-76.2]	20.3 [17.8-23.1]	3.4 [2.4-4.7]	0.4 [0.2-1.2]
Sweden	52.2 [49.4-55.1]	14.5 [12.9-16.2]	3.1 [2.2-4.3]	0.8 [0.4-1.4]	57.5 [54.6-60.4]	12.4 [11.0-13.9]	2.1 [1.5-2.9]	0.4 [0.2-1.0]
Switzerland	41.4 [36.1-46.9]	12.3 [10.4-14.4]	3.0 [2.1-4.2]	0.7 [0.3-1.7]	61.7 [58.8-64.6]	13.7 [11.7-16.1]	2.3 [1.4-3.7]	0.0 [-]
USA	63.1 [60.7-65.5]	33.5 [31.2-35.9]	14.0 [13.0-15.2]	6.1 [5.1-7.4]	74.7 [72.4-76.8]	32.6 [29.9-35.4]	9.5 [8.0-11.3]	2.8 [2.0-3.8]
Canada (M)	65.9 [60.3-71.1]	29.8 [25.4-34.6]	12.5 [9.5-16.3]	3.5 [2.2-5.7]	80.7 [76.2-84.5]	34.4 [28.8-40.5]	6.4 [4.3-9.5]	2.0 [0.8-4.6]
England (M)	70.1 [68.5-71.7]	31.8 [30.2-33.4]	10.3 [9.3-11.5]	3.2 [2.6-3.9]	76.4 [74.8-78.0]	27.7 [26.0-29.5]	6.3 [5.4-7.3]	1.1 [0.8-1.6]
USA (M)	67.7 [65.4-69.9]	38.6 [36.0-41.3]	19.0 [17.2-20.9]	8.5 [7.2-9.9]	78.1 [76.0-80.1]	36.2 [33.6-38.9]	12.7 [10.9-14.7]	3.7 [2.9-4.7]

Notes: Proportions are age-standardized to the US 2000 Census population using age-groups 50-59, 60-69, 70-79 and 80-89. All estimates incorporate survey weights and where applicable are adjusted for features of survey design, including probability sampling, clustering, stratification and non-response (see appendix for more information). 95% confidence intervals for proportions are calculated using logistic transform. Results are based on self-reported height and weight unless indicated as based on measured values (M). For Canada, estimates do not exclude individuals 90 and above because sufficient detail was not available to do so.

Table 2. Estimated proportion of all-cause mortality attributable to obesity by country, age and sex.

Country	Females				Males			
	50-59	60-69	70-79	80-89	50-59	60-69	70-79	80-89
Austria	0.12	0.17	0.09	0.06	0.19	0.13	0.10	0.06
Belgium	0.09	0.16	0.11	0.06	0.16	0.12	0.10	0.07
Canada	0.11	0.15	0.11	0.05	0.17	0.14	0.09	0.06
Czech	0.11	0.21	0.15	0.08	0.18	0.17	0.13	0.07
Denmark	0.08	0.11	0.10	0.03	0.13	0.11	0.08	0.06
France	0.08	0.13	0.09	0.04	0.14	0.12	0.10	0.06
Germany	0.09	0.16	0.12	0.05	0.15	0.13	0.11	0.07
Israel	0.11	0.16	0.14	0.06	0.16	0.14	0.10	0.05
Italy	0.09	0.15	0.11	0.04	0.15	0.13	0.10	0.07
Netherlands	0.09	0.13	0.12	0.05	0.13	0.13	0.07	0.04
Poland	0.14	0.23	0.17	0.06	0.16	0.17	0.10	0.06
Spain	0.12	0.22	0.15	0.08	0.18	0.15	0.11	0.09
Sweden	0.08	0.13	0.10	0.05	0.13	0.11	0.08	0.04
Switzerland	0.06	0.11	0.09	0.05	0.13	0.11	0.09	0.06
United States	0.20	0.23	0.15	0.06	0.24	0.21	0.14	0.07
Canada (M)	0.15	0.26	0.14	0.09	0.23	0.22	0.14	0.07
England (M)	0.17	0.22	0.18	0.09	0.22	0.18	0.13	0.11
United States (M)	0.22	0.26	0.18	0.09	0.26	0.23	0.17	0.10

Notes. Results are based on self-reported height and weight unless indicated as based on measured values (M). Source of relative risks: Prospective Studies Collaboration 2009b. [95% CI to be added.]

Table 3. Life expectancy at age 50 (e_{50}) in 2006 in the presence and absence of all-cause mortality attributed to obesity by country and sex (in years)

Country	Females			Males		
	e_{50} actual	e_{50} without obesity	Difference	e_{50} actual	e_{50} without obesity	Difference
Austria	33.96	34.68	0.72	29.39	30.38	1.00
Belgium	33.70	34.46	0.76	29.03	30.01	0.98
Canada	34.50	35.25	0.75	30.72	31.68	0.96
Czech	31.24	32.30	1.07	26.04	27.43	1.39
Denmark	31.90	32.55	0.65	28.22	29.04	0.82
France	35.68	36.22	0.54	29.86	30.83	0.97
Germany	33.60	34.34	0.73	29.07	30.10	1.03
Israel	33.61	34.44	0.83	30.64	31.55	0.92
Italy	35.24	35.85	0.61	30.57	31.48	0.91
Netherlands	33.31	34.02	0.71	29.45	30.18	0.73
Poland	31.39	32.61	1.22	24.73	26.11	1.38
Spain	35.40	36.28	0.88	29.94	31.10	1.16
Sweden	34.10	34.72	0.62	30.45	31.13	0.68
Switzerland	35.33	35.84	0.51	31.14	31.92	0.78
United States	32.95	34.24	1.29	29.20	30.81	1.61
Canada (M)	34.50	35.67	1.16	30.72	32.09	1.37
England (M)	33.31	34.55	1.24	29.84	31.18	1.34
United States (M)	32.95	34.50	1.56	29.20	31.05	1.85

Notes: Results are based on self-reported height and weight unless indicated as based on measured values (M). [95% CI to be added]

Table 4. US shortfall in life expectancy at age 50 relative to higher life expectancy countries, and change in that shortfall produced by eliminating obesity (in years)

Country	Females			Males		
	Gap in e ₅₀ (Actual)	Gap in e ₅₀ without obesity	Fraction of actual gap attributable to obesity	Gap in e ₅₀ (Actual)	Gap in e ₅₀ without obesity	Fraction of actual gap attributable to obesity
Austria	1.01	0.44	0.56	0.19	-0.43	>1.00
Belgium	0.75	0.22	0.71	-	-	-
Canada (M)	1.56	1.16	0.25	1.52	1.04	0.31
France	2.73	1.98	0.27	0.66	0.02	0.97
Germany	0.66	0.10	0.85	-	-	-
Israel	0.67	0.20	0.70	1.44	0.74	0.48
Italy	2.29	1.61	0.30	1.37	0.67	0.51
Netherlands	0.37	-0.22	>1.00	0.25	-0.63	>1.00
Spain	2.46	2.04	0.17	0.74	0.29	0.61
Sweden	1.15	0.48	0.58	1.25	0.32	0.74
Switzerland	2.38	1.60	0.33	1.94	1.11	0.43
England (M)	0.36	0.05	0.87	0.65	0.13	0.79
<i>Average</i>	<i>1.37</i>	<i>0.81</i>	<i>0.41</i>	<i>1.00</i>	<i>0.33</i>	<i>0.67</i>

Notes: Shortfall is calculated in respect to self-reported or measured BMI values for the US depending on data type of comparison countries (measured height and weight (M) are used for Canada and England and the shortfall attributable to obesity between the US and each of Canada and England is estimated using measured height and weight in the US). Dashed cells indicate a lower life expectancy at age 50 in that country and sex compared to the US.

Table 5. Effect of allowance for BMI misreporting on estimated improvement in life expectancy at age 50 from eliminating obesity (in years).

Country	Females	Males
Austria	0.20	0.12
Belgium	0.19	0.11
Czech	0.29	0.18
Denmark	0.17	0.16
France	0.15	0.12
Germany	0.20	0.17
Israel	0.22	0.08
Italy	0.15	0.11
Netherlands	0.20	0.13
Poland	0.28	0.17
Spain	0.13	0.03
Sweden	0.21	0.14
Switzerland	0.14	0.12
United States	0.23	0.20

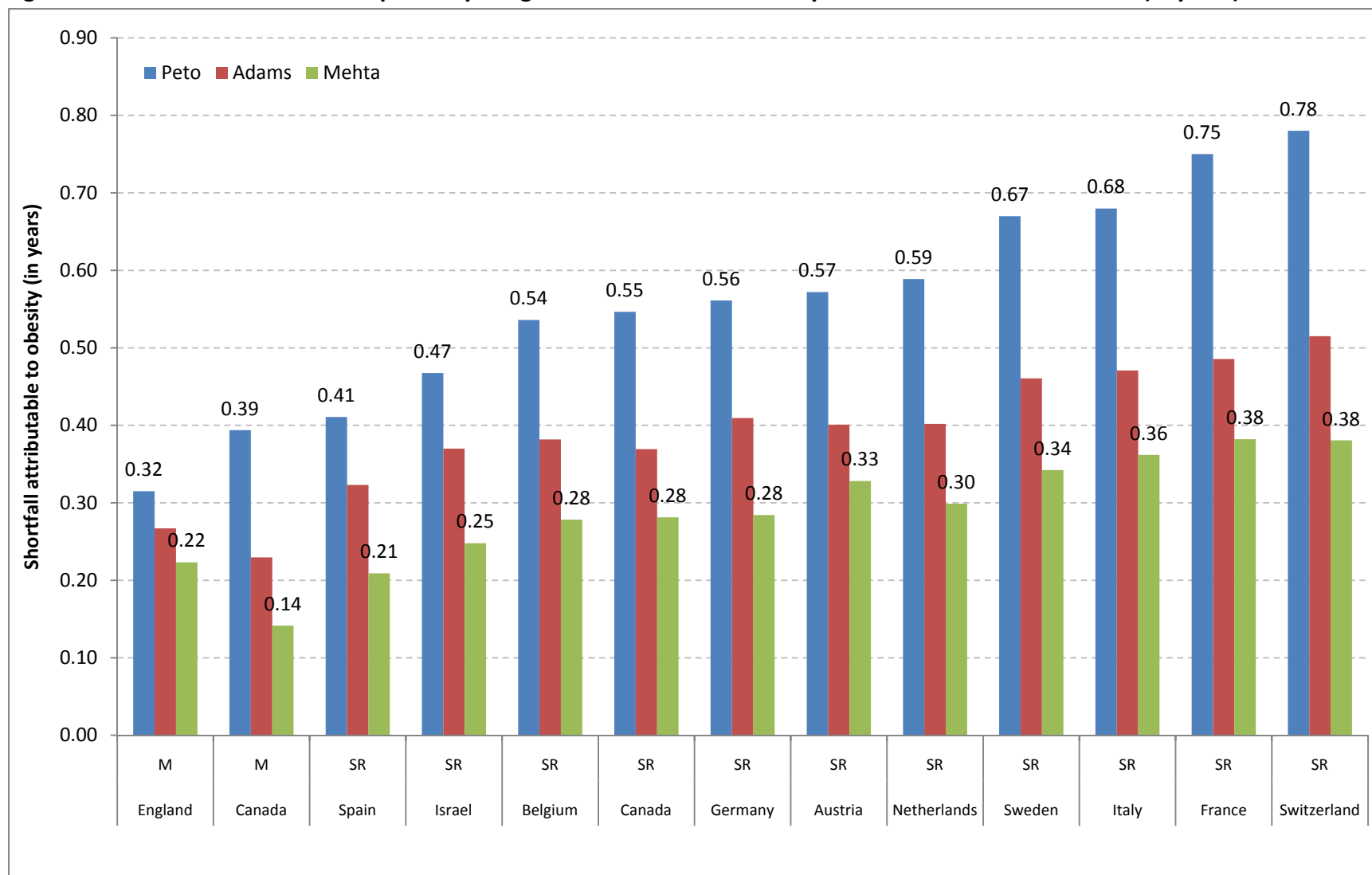
Notes: Corrections applied to countries with self-reported height and weight data. Coefficients of correction equation appear in Appendix Table 2.

Table 6. Estimated gain in life expectancy at age 50 in 2006 from hypothetically redistributing obese to optimal BMI categories, using three sets of risk factors (in years).

Country	Females			Males		
	PSC	Adams	Mehta/Chang	PSC	Adams	Mehta/Chang
Austria	0.72	0.30	0.28	1.00	0.23	0.40
Belgium	0.76	0.32	0.33	0.98	0.20	0.36
Canada	0.75	0.33	0.33	0.96	0.22	0.37
Czech	1.07	0.46	0.41	1.39	0.34	0.46
Denmark	0.65	0.28	0.27	0.82	0.16	0.32
France	0.54	0.22	0.23	0.97	0.19	0.35
Germany	0.73	0.29	0.33	1.03	0.21	0.43
Israel	0.83	0.33	0.36	0.92	0.18	0.34
Italy	0.61	0.23	0.25	0.91	0.17	0.36
Netherlands	0.71	0.30	0.31	0.73	0.15	0.32
Poland	1.22	0.58	0.47	1.38	0.38	0.47
Spain	0.88	0.38	0.40	1.16	0.24	0.42
Sweden	0.62	0.24	0.27	0.68	0.13	0.28
Switzerland	0.51	0.19	0.23	0.78	0.14	0.32
United States	1.29	0.70	0.61	1.61	0.52	0.64
<i>Average</i>	<i>0.79</i>	<i>0.35</i>	<i>0.34</i>	<i>1.02</i>	<i>0.23</i>	<i>0.39</i>
Canada (M)	1.16	0.65	0.64	1.37	0.37	0.46
England (M)	1.24	0.61	0.56	1.34	0.33	0.49
United States (M)	1.56	0.88	0.79	1.85	0.62	0.79

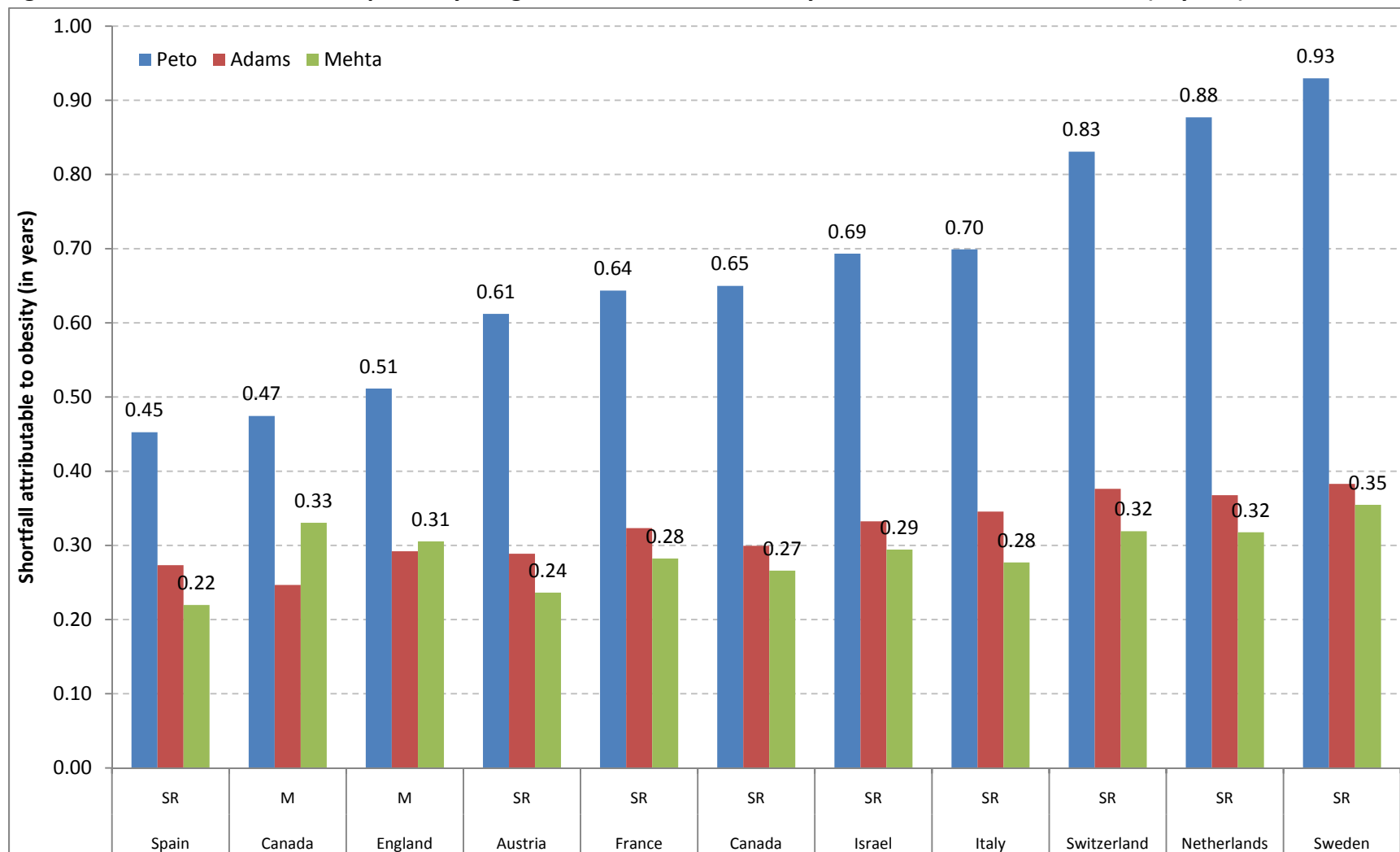
Notes: Results are based on self-reported height and weight unless indicated as based on measured values (M). The three sets of risk factors used in the calculations are drawn from the Prospective Studies Collaboration (PSC) 2009; Adams et al. (2006); and Mehta and Chang (2010).

Figure 2a. Shortfall in female life expectancy at age 50 attributable to obesity for three sets of relative risks (in years)



Notes: SR indicates BMI data based on self-reported height and weight; M indicates BMI data based on measured height and weight. Peto, Adams and Mehta refer to source of relative risks.

Figure 2b. Shortfall in male life expectancy at age 50 attributable to obesity for three sets of relative risks (in years)



Notes: SR indicates BMI data based on self-reported height and weight; M indicates BMI data based on measured height and weight. Peto, Adams and Mehta refer to source of relative risks.

Appendix

Sample Restrictions and Sources of Data

Consistent sample restrictions were applied to each survey to obtain a final analytic sample for estimating the distribution of body mass index. Exclusion of pregnant women was not necessary by virtue of the age-restrictions placed on samples. Body mass index values less than 10 and exceeding 60 were treated as outliers and eliminated from the sample. Individuals for whom self-reported height and weight were elicited through a proxy were dropped from all samples because of concerns with the reliability of these estimates. With respect to measured height and weight, information was available on the reliability of measurement for Canada and England; values deemed unreliable by the attending nurse were dropped. NHANES recorded individuals for whom height and weight were measured wearing clothes or medical devices; such individuals were not dropped from the sample due to inability to implement a similar restriction in other countries in the sample. These individuals, however, constituted a very small fraction of the sample. Specific details on each survey instrument and use of them follow. Appendix Table 1 provides a summary of data sources, years and sample sizes for each country in the sample.

The English Longitudinal Study of Ageing (ELSA) is a sample of the non-institutional population of England aged 50 and over. The sample includes all eligible adults from the 1998, 1999 or 2001 rounds of the Health Survey for England (HSE), which used an equal probability of selection design. ELSA sampling was carried out using clustering and stratification. Body mass index data in ELSA are based on clinical measurement of height and weight in mobile clinics by attending nurses. These data may be subject to measurement variation and examiner effects. Estimates relating to body mass index for England in this paper use Wave 2 nurse data (N=7,666) conducted in 2004-2005 and incorporate weights supplied by ELSA, which correct for systematic differences in response rates across sub-groups. We use the Taylor series linearization to derive estimates of standard errors that reflect the cluster-level PSU design. Information on stratification, however, was not available.

The National Health and Nutrition Examination Survey (NHANES) is a probability sample of the United States population under the auspices of the National Center for Health Statistics (NCHS). The instrument has three main elements: interviews, examination and laboratory testing. Since 1999, NHANES has been conducted as a continuous annual survey; however, data are released in two-year cycles. NHANES includes data on both self-reported and measured height and weight: the former are elicited in interviews while the latter are from clinical examination in mobile clinics by trained nurses in the examination component of the survey. NHANES supply sample weights which capture unequal probabilities of selection, non-response adjustments and adjustments to independent population controls. Weights are provided for separate components of the survey (interview vs. examination). NHANES uses a complex survey design in

which the primary sampling units are counties and the sampling frame was all counties in the US. In some cases, small counties are aggregated to produce larger samples. Prevalence estimates in this paper use NHANES cycles 2003-2004, 2005-2006 and 2007-2008, a period in which there were no significant national trend in obesity in either sex (Flegal 2010). These cycles are combined to obtain a larger sample. Weights are adjusted following procedures recommended in the NHANES documentation (NCHS, 2006). Estimates reflect the sampling weights supplied in NHANES. Standard errors are adjusted for the first stage of the cluster design and stratification using the Taylor series linearization method.

The Survey of Health, Ageing and Retirement in Europe (SHARE) is a cross-national longitudinal probability sample of the non-institutional population of Europe aged 50 and above. Two survey rounds are available: Wave 1 (N=31,115) conducted in 2004 and Wave 2 (N=33821) conducted in 2006-2006. Combined Waves 1 and 2 include Austria, Belgium, Czech Republic, Denmark, France, Germany, Greece, Israel, Italy, Netherlands, Poland, Spain, Sweden and Switzerland. SHARE has a complex survey design with clustering and stratification. However, information on primary sampling units and strata has not been made available for the full set of countries due to restrictions on the data. For this paper, the survey data are treated as a single stage random sample with unequal sampling probabilities as recommended in the SHARE documentation (Mannheim Research Institute for the Economics of Aging, 2009).

Canadian Community Health Survey (CCHS) is a cross-sectional probability survey of the Canadian population, which collected responses from persons aged 12 or older, living in private occupied dwellings in 122 health regions covering all provinces and territories. We use the CCHS cycle 3.1 (2005) public use microdata file (PUMF), which contains self-reported height and weight in the full sample and measured height and weight for a sub-sample. Survey coverage was approximately 98% of the Canadian population aged 12 and over. Three sampling frames were used to select households: an area frame, a list frame of telephone numbers and random digit dialing sampling frame, which accounted for 49%, 50% and 1% of the sample respectively. The CCHS is based upon a complex design, with stratification and multiple stages of selection, and unequal probabilities of selection of respondents. We incorporate survey weights to obtain nationally representative estimates using separate weights for the full and sub-samples provided in the survey. Cluster and stratification information were not available for adjustment of standard errors.

Appendix Table 1: Description of Data Sources

Countries	Survey	Mode of Interview	Type	Year	Sample
Austria	SHARE	In-person	Self-Report	2004; 2006-2007	1840
Belgium	SHARE	In-person	Self-Report	2004; 2006-2007	2933
Canada	CCHS	In-person/Telephone	Self-Report	2005	55703
Canada	CCHS	Examination	Measured	2005	1979
Czech Republic	SHARE	In-person	Self-Report	2004; 2006-2007	1768
Denmark	SHARE	In-person	Self-Report	2004; 2006-2007	1756
England	ELSA	Examination	Measured	2004-2005	7153
France	SHARE	In-person	Self-Report	2004; 2006-2007	2774
Germany	SHARE	In-person	Self-Report	2004; 2006-2007	2885
Israel	SHARE	In-person	Self-Report	2004; 2006-2007	2146
Italy	SHARE	In-person	Self-Report	2004; 2006-2007	2751
Netherlands	SHARE	In-person	Self-Report	2004; 2006-2007	2812
Poland	SHARE	In-person	Self-Report	2004; 2006-2007	1681
Spain	SHARE	In-person	Self-Report	2004; 2006-2007	1994
Sweden	SHARE	In-person	Self-Report	2004; 2006-2007	2966
Switzerland	SHARE	In-person	Self-Report	2004; 2006-2007	1615
USA	NHANES	In-person	Self-Report	2003-2008	7526
USA	NHANES	Examination	Measured	2003-2008	6511

Notes: Surveys: Survey of Health, Ageing and Retirement in Europe (SHARE); Canadian Community Health Survey (CCHS); English Longitudinal Study of Ageing (ELSA); National Health and Nutrition Examination Survey (NHANES). Mode of Interview: refers to how interviews were conducted; Type: refers to whether height and weight data were obtained through self-report or clinical measurement. Sample: refers to final analytic sample after applying sample restriction criteria.

Appendix Table 2. NHANES 2003-2008 Coefficients of Correction Equation

true weight	female (N=3100)	male (N=3195)
weight (kg)	1.11823	0.98502
weight ² (kg)	-0.00063	0.00021
age	-0.15169	-0.08334
age ²	0.00089	0.00047
constant	1.89556	2.76773
true height		
height (m)	-0.42869	0.02846
height ² (m)	0.39685	0.23747
age	0.00104	-0.00043
age ²	-0.00001	0.00000
constant	1.24719	0.99479

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