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Social Determinants and International Comparisons of Health and Mortality

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Abstract
Despite substantial gains in population health over recent decades, the US faces a growing epidemic of obesity that threatens continued progress. This dissertation seeks a better understanding of this dire challenge through three chapters that explore obesity from distinct vantage points. The first chapter quantifies the extent to which greater obesity in the US contributes to its low life expectancy ranking with respect to 15 other developed countries. The principal finding is that the higher prevalence of overweight and obesity in the US may contribute between a fifth and a third of the longevity gap above age 50. The second chapter is an investigation of the mortality risks and population impact of obesity in the older adult population of the US. I propose an innovative measurement strategy using weight histories. My findings indicate that the prior literature may substantially underestimate the mortality risks of obesity by failing to fully account for confounding by illness. The third and final chapter investigates the social context of obesity through an examination of eating behaviors of adults in the US. I find that participation in the family dinner is associated with a significantly lower probability of being obese and that the association is robust to adjustment for multiple dimensions of socioeconomic status.

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SOCIAL DETERMINANTS AND INTERNATIONAL COMPARISONS OF HEALTH AND MORTALITY

In memory of my grandparents,
Dr. Charles A. Stokes, Constance C. Stokes
and Jane R. Portmess

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ABSTRACT

SOCIAL DETERMINANTS AND INTERNATIONAL COMPARISONS OF HEALTH AND MORTALITY

Andrew Stokes

Samuel H. Preston

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Despite substantial gains in population health over recent decades, the US faces a growing epidemic of obesity that threatens continued progress. This dissertation seeks a better understanding of this dire challenge through three chapters that explore obesity from distinct vantage points. The first chapter quantifies the extent to which greater obesity in the US contributes to its low life expectancy ranking with respect to 15 other developed countries. The principal finding is that the higher prevalence of overweight and obesity in the US may contribute between a fifth and a third of the longevity gap above age 50. The second chapter is an investigation of the mortality risks and population impact of obesity in the older adult population of the US. I propose an innovative measurement strategy using weight histories. My findings indicate that the prior literature may substantially underestimate the mortality risks of obesity by failing to fully account for confounding by illness. The third and final chapter investigates the social context of obesity through an examination of eating behaviors of adults in the US. I find that participation in the family dinner is associated with a significantly lower probability of being obese and that the association is robust to adjustment for multiple dimensions of socio-economic status.

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INTRODUCTION

The US has achieved substantial gains in population health over recent decades. Heart disease rates have more than halved between 1980 and 2000 as a result of improved medical management and impressive reductions in major risk factors (Ford et al. 2007). Smoking rates have declined dramatically since the Surgeon General's report of 1964. A major exception to these favorable trends is obesity, which has risen dramatically in recent decades both in the US and internationally (Flegal et al. 2010; Finucane et al. 2011; Hossain, Kawar, and Nahas 2007).

It is to the epidemic of obesity that this dissertation is addressed. Each study examines obesity from a different perspective. The first chapter (co-authored with Samuel H. Preston) assesses the contribution of obesity to international differences in longevity. The paper finds higher obesity prevalence at younger ages and higher prevalence of morbid obesity at all ages in the US compared to countries in Western Europe. As a result, the findings indicate that obesity may explain between a fifth and a third of the gap in life expectancy between the US and comparison countries.

In the second chapter, I investigate the mortality risks and population impact of obesity in the older adult population of the United States using an innovative approach that is robust to the biasing effects of illness-induced weight loss. Instead of using the body mass index (BMI) at time of survey, I employ a measure of maximum lifetime BMI to measure the mortality risks of obesity. The advantage of maximum BMI is that it better captures the total physiological damage caused by obesity and is not susceptible to fluctuations related to weight loss. I find that use of maximum BMI leads to monotonically increasing risks of mortality with increasing BMI. Furthermore, using

maximum BMI reveals that previous findings of weak associations between obesity and mortality in older adults may be due to the fact that the normal BMI category combines low-risk stable-weight individuals with high-risk individuals that have experienced weight loss. Use of maximum BMI results in an estimate of the contribution of obesity to mortality for older adults in the US that is substantially higher than that estimated using BMI at time of survey. This implies that our estimates of the contribution of obesity to the US longevity disadvantage in Chapter 1 may be conservative.

The third chapter examines sociological dimensions of obesity through a study of social rituals of eating in the US. I focus one particular eating ritual that has been emphasized in the prior literature: the family dinner. Using data from the American Time Use Survey, I investigate the association between family dinner participation and obesity in adults ages 25-49. The extensive detail in the time-use data allows for a more meaningful definition of the family dinner than has been possible in prior studies. The definition incorporates information on the timing, location and duration of the meal, whether it was reported as the primary (e.g. main) activity being carried out and with whom it occurred. My findings indicate that participation in the family dinner is associated with a significantly lower probability of being obese and that the association is robust to multiple controls for socio-economic status. This finding suggests that changing eating patterns may have contributed to the rise of obesity in the United States.

CHAPTER 1

Contribution of Obesity to International Differences in Life Expectancy[1]

1.1 Introduction

Life expectancy in the United States (US) has fallen below that of most other industrialized countries and ranked 32nd in the world in 2008 (World Health Organization 2010). As President Obama has noted (2009), the relatively low level of life expectancy in the US coexists with the highest per capita expenditure on health care in the world. Explanations of the low US ranking range from a history of high levels of cigarette smoking to low levels of physical activity, a poorly performing health care sector, high levels of income inequality, and high levels of obesity (Crimmins, Preston, and Cohen 2010). Identifying the responsible factors would help to clarify the critical public health domains where the US has fallen furthest behind its peers.

The purpose of this paper is to estimate the extent to which the high level of obesity in the US is contributing to its poor longevity performance. According to World Health Organization estimates, men and women in the US had a higher prevalence of obesity in 2005—defined as having a body mass index (BMI) (the ratio of weight in kilograms to the square of height in meters) of 30.00 or higher—than any other country in Europe, North America, or East Asia (World Health Organization 2005). Because many studies demonstrate that obese individuals suffer an elevated risk of death (Prospective Studies Collaboration 2009), it is reasonable to suppose that the high level of obesity in the US is contributing to its comparatively low life expectancy.

[1] This chapter is co-authored by Samuel H. Preston (University of Pennsylvania). A slightly modified version of this chapter is published as: Preston SH and Stokes A (2011). "Contribution of Obesity to International Differences in Life Expectancy" *American Journal of Public Health* 101:2137-2143.

The years of life lost by an *individual* as a result of his or her obesity have been estimated in several studies (Fontaine et al. 2003; Prospective Studies Collaboration 2009). In this paper, we are asking a question about population health rather than individual health: how many years of life are forfeited, on average, by members of a *population* as a result of the level of obesity *in that population*. Answering this question involves combining the prevalence of obesity in a population with the risks of mortality for people in a particular BMI category in order to estimate the effects of obesity on age-specific mortality rates. Estimates of the impact of obesity on a population's level of life expectancy are uncommon; an exception is Olshansky et al., whose effort was limited to the US (Olshansky et al. 2005). Yet, these estimates are important as they provide a basis for conducting cross-national comparisons, which can be used to determine why some countries achieve better health outcomes than others.

We estimate the fraction of deaths attributable to obesity by age and sex for 16 countries including the US. We focus on ages above 50 since 94% of newborns survive to age 50 in the current US life table and variation in life expectancy at birth is dominated by variation in mortality above this age (Arias, Rostron, and Tejada-Vera 2010; Ho and Preston 2009). We recalculate life tables for each country after removing deaths attributable to obesity in order to estimate 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 obesity and to misreporting of height and weight.

1.2 Methods

1.2.1 Data

We use BMI as our basic indicator of obesity. Flegal and Graubard have shown that the proportion of deaths attributable to obesity does not vary significantly with the indicator chosen (Flegal and Graubard 2009). 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 2009). The Prospective Studies Collaboration (PSC) study is the largest and most detailed of several large compilations of data on obesity and mortality (Allison et al. 1999). 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. Results of this investigation have been presented by sex, age group (35-59, 60-69, 70-79, 80-89), and detailed BMI categories (2.5 unit intervals within the range 15.00-34.99 and a single interval for 35.00-49.99).

Estimates of the population distribution of BMI were obtained from nationally representative survey data. Height and weight data for estimating an individual's BMI are based on self-reports obtained through in-person interviews except in Canada and England where measured height and weight were used. In the US, both self-reported and measured values are available and used.

Data for European countries excluding England were taken from the Survey of Health, Ageing and Retirement in Europe (SHARE). We include individuals interviewed in Wave 1 (2004) as well as a refresher sample from Wave 2 (2006-2007). Data for England were obtained from Wave 2 (2004-2005) of the English Longitudinal Study of

Ageing (ELSA). US data come from the National Health and Nutrition Examination Survey (NHANES) cycles 2003-2004, 2005-2006 and 2007-2008. Previous research has found no significant national trend in obesity for either sex during this period in the US (Flegal et al. 2010). Data for Canada were derived from cycle 3.1 (2005) of the Canadian Community Health Survey (CCHS). .

Data for constructing period life tables, including deaths and population, were obtained by country, age and sex in single-year age-intervals for 2006 from the Human Mortality Database (HMD) (Wilmoth and Shkolnikov 2010).

1.2.2 Analytic Approach

To identify the proportion of deaths in a particular country/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 that group to the optimal category and calculate the proportional reduction in mortality that would occur under this redistribution. This is quantified using the population attributable fraction (PAF). We construct estimates of BMI prevalence in the same age-sex-BMI groupings used by the Prospective Studies Collaboration, with the exception that we apply the PSC mortality values for ages 35-59 to ages 50-59. In the PSC, the lowest-risk BMI category is 22.50-24.99 except for males aged 80-89, for whom it is 20.00-22.49, and for females aged 70-79, for whom it is 25.00-27.49. We use the term obesity to refer to all weight categories above the optimal, including those who are overweight (BMI between 25.00 and 30.00). 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 mortality risk from obesity is assumed

to be zero above age 90 throughout our analysis. The PAF for population i (where i is an indicator for each country, age and sex combination) is estimated as,

$$PAF_i = \frac{\sum(C_{ij}M_{sj} - C^*_{ij}M_{sj})}{\sum(C_{ij}M_{sj})} \quad (1)$$

where,

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 individuals above the optimal BMI were redistributed to the optimal category

Equation 1 would give the same value of the PAF 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.

The country, age and sex specific PAFs are applied to death rates in the HMD 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. Conventional methods of calculating life tables were used (Preston, Heuveline, and Guillot 2001). Hypothetical life expectancies obtained in this manner are then compared to the actual values, also computed from the HMD, by country and sex. To identify the extent to which the US shortfall in life expectancy is attributable to obesity, differences in actual life expectancy between the US and each country are compared to the differences that would be expected in the absence of obesity. When Canada and England are compared to the US, measured rather than self-reported heights and weights are used.

Analysis of uncertainty was conducted for PAFs and life expectancy estimates using a bootstrapping procedure (Efron and Tibshirani 1986). Uncertainty estimates from two sources are combined: uncertainty in the BMI data resulting from sampling variability and uncertainty in estimation of the relative risks. For each country, age and sex combination, 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 incorporate uncertainty from the relative risks, vectors of the underlying effect parameters of relative risks of length corresponding to the number of BMI intervals are drawn from independent normal distributions with age and sex-specific standard errors provided to us by the Prospective Studies Collaboration. The resulting vectors of risks are applied to the simulated BMI distribution data to obtain country, age and sex specific PAFs. These steps are repeated to obtain 500 estimates of each country, age and sex specific attributable fraction, from which the 2.5th and 97.5th percentile values are extracted as 95% confidence intervals.

We explore the sensitivity of results to the assumed set of risks associated with obesity and to misreporting of height and weight. Flegal et al. have suggested that the relative risks of death associated with obesity have declined in the US (Flegal et al. 2005). In order to investigate the effect of a possible reduction in obesity risks on international comparisons, we introduce an alternative set of risk factors adapted from Adams et al. that applies to a more recent period (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. As in the Prospective Studies Collaboration results,

relative risks are adjusted for smoking. In contrast to PSC procedure, relative risks in Adams et al. are also adjusted for social status and physical activity.

We use the published results of this study to estimate relative risks in the age categories that were employed in the baseline 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. 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. Standard errors for uncertainty estimation are approximated because of the smoothing procedure we employed to obtain risks for the relevant ages.

Analysis of NHANES data shows that American women tend to underestimate their weight, while both men and women tend to overestimate height at older ages (Ezzati et al. 2006). To explore whether our results are sensitive to error in self-reports of height and weight, we replicate all analyses after correcting self-reported height and weight for misreporting using an approach similar to that which has been applied elsewhere (Burkhauser and Cawley 2008). Using data on adults ages 50 and above from NHANES 2003-2008, we estimate linear regression models for each sex of measured height (weight) versus self-reported height (weight), age and the square of age.

Analyses were conducted using STATA 10.1 (Stata Corp, College Station, Texas) and R 2.11.1 (The R Foundation for Statistical Computing, Vienna).

1.3 Results

1.3.1 Cross-National Comparison of Obesity Rates

Table 1.1 presents sample sizes in each country and 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 any comparison country for both males and females. The proportionate difference between the US and other countries grows larger as BMI increases. In Canada and England, prevalence rates for obese class I and above exceed rates found in other countries, yet remain consistently lower than in the US. The difference is most pronounced for severe obesity with the prevalence of class III obesity in Canada and England being about half of the level found in the US.

[TABLE 1.1 HERE]

Figure 1.1 shows smoothed frequency distributions of BMI by sex based on self-reports for the US and a set of countries selected to show the range of variation present in the sample. The US distribution has larger variance and is markedly right-skewed with respect to the comparison countries.

[FIGURE 1.1 HERE]

1.3.2 Effects of Obesity on Longevity

Fractions of all-cause mortality attributable to obesity (PAF) by country, age and sex are presented in Table 1.2. Confidence intervals for PAFs reflect sampling uncertainty in BMI data and estimation uncertainty in the risks of obesity. The use of measured rather

than self-reported values of height and weight in the US leads to PAFs that are higher by approximately 3%. The discrepancy between the PAFs in the US and other countries is typically greatest for both men and women at ages 50-59, reflecting the unusually large proportion of individuals obese in the US in those ages. Using self-reported data, the fraction of deaths attributable to obesity for US women aged 50-59 is 0.20 (95% CI 0.17-0.27) compared to an average of 0.10 (0.08-0.15) in comparison countries. Mortality attributable to obesity declines significantly with age for both males and females. For females, the greatest effects are found in the age-group 60-69 while for males, the impact of obesity is highest at ages 50-59. After reaching these levels, the effects of obesity on mortality decline by about two-thirds across the age-range in both sexes.

[TABLE 1.2 HERE]

Table 1.3 presents life expectancy impacts implied by the estimates of deaths attributable to obesity presented in Table 1.2. 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 US by an estimated 1.28 years (1.14-1.70) for women and by 1.61 years (1.44-1.82) for men when self-reported BMI data are used. In other countries with self-reported data, female life expectancy would improve by an average of 0.73 years (0.63-1.13) and male life expectancy would improve by an average of 0.98 years (0.86-1.16) if obesity were eliminated. When measured BMI is used, the estimated gains in life expectancy in the US are greater by an additional 0.24-0.26 years. No other country is estimated to gain as much from the elimination of obesity as the US.

[TABLE 1.3 HERE]

Table 1.4 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 substantially 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 0.80 years (0.70-0.87) lower without obesity, so that obesity accounts for an average of 42% (36-48) 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% (57-76). For females, after the elimination of obesity the difference in life expectancy between the US and England, Germany and Israel becomes statistically indistinguishable from zero and US life expectancy surpasses that of the Netherlands. For males, the difference in life expectancy between the US and France is eliminated and US life expectancy surpasses that of Austria and the Netherlands. These estimates suggest that obesity is contributing very substantially to the low US ranking in longevity.

[TABLE 1.4 HERE]

1.3.3 Sensitivity of Results to Alternative Risks

Results of using the alternative risk factors are presented in Table 1.5. In every country for both sexes, the use of the alternative risk factors reduces the estimated gain in life expectancy from eliminating obesity. For countries other than the US using self-reported data, women's mean gain in life expectancy is only 42% as large using Adams et al. risk factors as it is using PSC's. For men, it is only 21% as large. Proportionate reductions are

smaller in the US than in other countries because a much higher fraction of the US population resides in obesity classes II or III, where risks remain considerable even under the alternative sets of risks.

Confining comparisons in Table 1.5 to countries with higher life expectancies than the US, as in Table 1.4, we recalculate the proportion of the life expectancy gap that is explained by obesity. Obesity accounts for 29% of the US shortfall for women and 32% for men using Adams' risks. Obesity continues to account for a substantial part of the US shortfall in life expectancy even when lower risks are assumed.

[TABLE 1.5 HERE]

We also applied a second alternative set of risk factors derived from NHANES III (Mehta and Chang 2011). A national probability sample of 4375 individuals enrolled at ages 50-69 between 1988 and 1994 were followed into the National Death Index through 2006. Advantages of the study include recent data, a probability sample of the US population, and a relatively long follow-up period. Relative risks were adjusted for smoking and socioeconomic status. The results (not shown) are very similar to those produced using the Adams et al. risks: obesity accounts for 22% of the shortfall in life expectancy for US women and 29% for men.

1.3.4 Effects of Misreporting of Height and Weight

After adjusting self-reported height and weight data for misreporting, 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 (results not shown). The estimated effect of eliminating obesity also increases in other countries, although by

less than in the US. As a result, correcting for misreporting positively affects the magnitude of the life expectancy gap attributable to obesity between the US and other countries. The greatest difference occurs between the US and Spain, amounting to 0.10 years for women and 0.17 for men. 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.

1.4 Discussion

In our analysis of the effects of obesity on longevity in 16 countries, we have shown that obesity reduced longevity in all countries, ranging from half a year for females in Switzerland to more than a year and a half for US males. These effects have been more severe in the US than in other countries. Two key features of the US BMI distribution that distinguish it from comparison countries include an unusually high rate of obesity in younger age-groups and significantly higher rates of severe obesity. Comparing the US to the two countries with the next highest rates of obesity – Canada and England-- gains to life expectancy by hypothetically eliminating obesity are still 25-40% higher in the US. As a result of its greater impact there, obesity has contributed substantially to the US longevity disadvantage, which would be significantly reduced and in some cases eliminated in the absence of obesity.

Olshansky et al. have also produced estimates of the effect of obesity on US life expectancy using NHANES III risk factors (Olshansky et al. 2005). They do not use the full BMI distribution but rather experiment with various binary specifications of risk, producing estimated effects on US life expectancy that range widely from 0.28 years to 0.88 years. Our estimates in Table 1.5 are at the high end of that range when Adams' risk factors are used and far above it when risks from the Prospective Studies Collaboration are used.

It is clear that the estimated effect of obesity on levels of life expectancy is sensitive to the set of obesity risk factors that is used. The risk factors derived from the studies of Adams et al. have the advantage of pertaining to a period closer to the time when the levels of both obesity and mortality are recorded in the various countries and when the PAFs are modeled. This study also controls social class in its analyses, an important confounding factor of the relationship between obesity and mortality (Mehta and Chang 2009).

The choice of the proper set of risk factors probably depends most heavily on whether the mortality risks of obesity have declined. A large study begun in 1982 by the American Cancer Society with follow-up of healthy non-smokers through 2002 found no decline in the mortality risk from obesity (Calle, Teras, and Thun 2005). However, such a decline has been found using successive waves of NHANES (Flegal et al. 2005). A reduction in obesity risks in the US was also identified by Mehta and Chang in three different data sets including NHANES, the Framingham study, and a National Health Interview Survey follow up study (Mehta and Chang 2011).

A decline in the mortality risks of obesity may have occurred for a number of reasons. Gregg et al. note that the use of lipid-lowering and antihypertensive medications increased rapidly from 1988-94 to 1999-2000, with the largest gains among obese individuals (Gregg, Cheng, and Cadwell 2010). Also, deaths from cardiovascular disease are a diminishing proportion of all mortality (Beltrán-Sánchez, Preston, and Canudas-Romo 2010). Combined with greater obesity risks from cardiovascular diseases than from the aggregate of other causes of death, such a decline also implies that the all-cause mortality risk from obesity should be declining.

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 US (Flegal et al. 2010) may produce a decline in the average duration of obesity for an obese person. To the extent that there are duration effects of obesity—risks that cumulate with length of time spent in the state—the risk of obesity per se may have declined when duration is not accounted for in the research design. 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).

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.

Our analysis has a number of strengths. We used nationally representative data from 16 countries to measure distributions of BMI, which together capture a large fraction of the variation in obesity rates among high-income countries. We incorporated detailed information on the mortality risks of obesity, differentiated by age, sex and fine BMI intervals using high-quality data from a large meta-analysis of prospective cohort studies. We characterized uncertainty in our estimates from multiple sources and conducted numerous analyses of the sensitivity of our results to alternative procedures.

Our analysis is also subject to limitations. We assumed that the same set of individual-level mortality risks of obesity was applicable to all countries, although these risks may differ somewhat across contexts. Our analysis would have been strengthened by the availability of measured BMI data in all sample countries and inclusion of data from high-income countries outside North America and Europe, such as Japan and Australia, where conditions may differ from those included in the sample. While the risk factors that we used were adjusted for smoking behavior, they were not adjusted for all other factors with which obesity may be correlated.

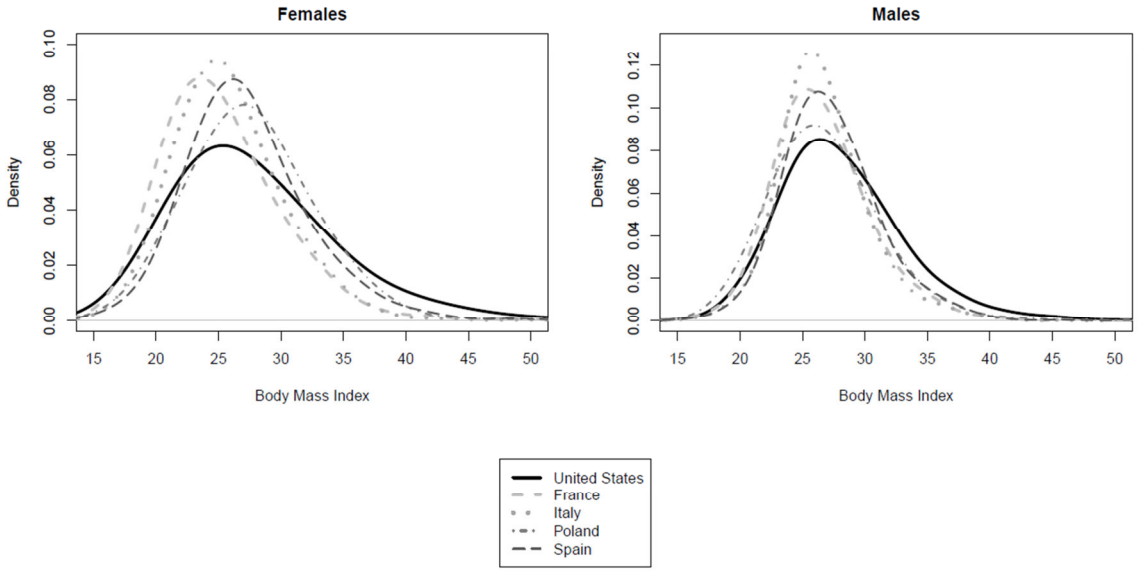
1.5 Conclusion

Based on our results, the high prevalence of obesity in the US has reduced life expectancy at age 50 by 0.52 to 1.61 years for males and by 0.70 to 1.29 years for females. In order to study the impact of obesity on international differences in longevity, we have also estimated the effects of obesity on longevity in 15 other countries. We conclude that, even when relatively low mortality risks associated with obesity are used, the high levels of obesity in the US contribute substantially – in the neighborhood of 30%

- 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 42% of the longevity shortfall for US women and 67% for US men.

High levels of obesity in the US appear to be strongly implicated in its inferior level of longevity. We believe that this demonstration should add urgency to public health efforts aimed at achieving healthier weights for Americans.

Figure 1.1 Smoothed frequency distributions of body mass index by sex, ages 50-89.



This Figure shows population distributions of BMI for the US and comparison countries. BMI data are derived from self-reported height and weight.

Table 1.1 Cumulative prevalence of overweight and obesity by country, adults 50-89 based on self-report [95% Confidence Intervals]

		Percentages, Adults Aged 50-89 [95 % CI]							
		Females				**Males**			
Country	**Sample Size**	**BMI ≥ 25**	**BMI ≥ 30**	**BMI ≥ 35**	**BMI ≥ 40**	**BMI ≥ 25**	**BMI ≥ 30**	**BMI ≥ 35**	**BMI ≥ 40**
Austria	1,840	57.0 [53.8-60.2]	21.0 [18.5-23.8]	4.7 [3.5-6.3]	1.5 [0.9-2.6]	69.2 [65.5-72.6]	19.3 [16.4-22.6]	3.9 [2.6-5.8]	1.1 [0.5-2.5]
Belgium	2,933	53.2 [50.6-55.8]	17.7 [15.8-19.7]	4.1 [3.2-5.2]	0.8 [0.4-1.3]	63.8 [61.2-66.4]	17.4 [15.5-19.6]	2.9 [2.1-3.9]	0.5 [0.2-1.1]
Czech Rep.	1,768	67.7 [63.4-71.7]	22.8 [19.2-26.7]	4.6 [3.1-6.9]	1.0 [0.5-2.1]	75.9 [71.6-79.7]	21.8 [18.3-25.8]	2.7 [1.7-4.2]	0.8 [0.3-1.9]
Denmark	1,756	43.7 [40.4-47.1]	13.0 [10.9-15.5]	3.0 [2.1-4.3]	0.6 [0.3-1.3]	59.0 [55.4-62.5]	13.2 [10.9-15.8]	1.9 [1.1-3.0]	0.4 [0.1-1.3]
France	2,774	46.0 [42.7-49.3]	15.5 [13.0-18.4]	3.2 [2.3-4.6]	1.2 [0.6-2.3]	61.6 [57.8-65.4]	16.5 [13.8-19.7]	3.0 [1.7-5.1]	0.3 [0.1-1.8]
Germany	2,885	54.8 [51.9-57.6]	15.8 [13.8-17.9]	4.4 [3.4-5.7]	1.4 [0.8-2.2]	67.9 [64.9-70.7]	16.4 [14.3-18.9]	3.8 [2.8-5.3]	0.8 [0.4-1.7]
Israel	2,146	57.9 [48.7-66.6]	19.0 [13.9-25.4]	3.5 [2.0-6.2]	1.0 [0.3-2.7]	64.3 [57.5-70.5]	14.0 [10.1-19.1]	3.3 [1.3-7.9]	0.6 [0.1-2.7]
Italy	2,751	53.2 [50.0-56.4]	15.9 [13.8-18.3]	3.1 [2.2-4.2]	0.6 [0.3-1.1]	67.3 [63.9-70.5]	14.9 [12.6-17.5]	3.1 [2.1-4.6]	0.2 [0.1-0.5]
Netherlands	2,812	52.7 [49.8-55.6]	15.9 [14.0-18.1]	4.3 [3.3-5.7]	1.5 [0.9-2.4]	62.5 [59.1-65.8]	12.7 [10.9-14.9]	2.4 [1.7-3.5]	0.4 [0.2-0.9]
Poland	1,681	68.3 [64.9-71.6]	27.3 [24.4-30.5]	6.2 [4.8-8.1]	1.1 [0.6-1.9]	64.5 [60.7-68.1]	20.1 [17.3-23.3]	4.0 [2.7-5.8]	0.8 [0.3-1.9]
Spain	1,994	66.5 [63.2-69.6]	23.9 [21.2-26.8]	7.5 [6.0-9.4]	1.9 [1.1-3.1]	71.9 [68.4-75.1]	20.6 [17.8-23.7]	3.7 [2.4-5.4]	0.3 [0.1-1.0]
Sweden	2,966	52.1 [49.1-55.0]	16.4 [13.8-19.2]	3.3 [2.4-4.4]	0.9 [0.5-1.7]	56.7 [52.6-60.6]	13.9 [10.4-18.3]	4.2 [1.6-10.3]	0.5 [0.2-1.2]
Switzerland	1,615	41.4 [38.0-44.9]	12.5 [10.4-15.0]	3.1 [2.1-4.6]	0.7 [0.3-1.6]	61.8 [58.2-65.4]	14.2 [11.7-17.1]	2.5 [1.6-4.1]	0.0 [0.0-0.0]
Average	*2,302*	*55.0 [54.0-56.2]*	*18.2 [17.5-19.1]*	*4.2 [3.9-4.7]*	*1.1 [0.9-1.3]*	*65.1 [64.0-66.1]*	*16.5 [15.7-17.3]*	*3.2 [2.7-3.7]*	*0.5 [0.4-0.7]*
United States	7,526	61.9 [59.7-64.0]	31.0 [29.1-32.9]	12.3 [11.6-13.2]	5.1 [4.3-6.0]	73.3 [71.3-75.2]	30.9 [28.6-33.2]	8.8 [7.5-10.3]	2.5 [1.9-3.3]
Canada†	1,979	65.5 [60.9-69.9]	28.6 [24.9-32.7]	11.8 [9.2-15.1]	3.2 [2.0-5.3]	79.3 [75.0-83.0]	32.9 [27.9-38.4]	5.9 [4.1-8.4]	1.7 [0.8-3.7]
England†	7,153	69.6 [68.1-71.1]	31.0 [29.5-32.6]	9.8 [8.9-10.9]	3.0 [2.5-3.6]	75.9 [74.3-77.4]	27.2 [25.5-28.9]	6.0 [5.1-6.9]	1.0 [0.7-1.5]
United States†	6,511	67.4 [65.2-69.4]	36.3 [34.0-38.6]	16.5 [15.0-18.1]	7.0 [6.0-8.1]	76.9 [75.2-78.5]	34.6 [32.5-36.9]	11.7 [10.2-13.3]	3.3 [2.6-4.2]

This table presents data on the cumulative distribution of overweight and obesity based on various thresholds of BMI. Prevalence rates are age-standardized to the US 2000 Census population using age-groups 50-59, 60-69, 70-79 and 80-89. Sampling weights were used to adjust BMI estimates for unequal selection probabilities and standard errors were adjusted for cluster design and stratification where this information was available. †BMI calculated using measured height and weight.

Table 1.2 Estimated proportion of all-cause mortality attributable to obesity by country, age and sex [95% Confidence Intervals]

	Population Attributable Fractions, Adults Aged 50-89 [95% CI]							
	Females				**Males**			
Country	**50-59**	**60-69**	**70-79**	**80-89**	**50-59**	**60-69**	**70-79**	**80-89**
Austria	0.13 [0.10-0.19]	0.18 [0.13-0.23]	0.08 [0.06-0.14]	0.06 [0.02-0.16]	0.19 [0.15-0.23]	0.13 [0.10-0.16]	0.10 [0.07-0.13]	0.05 [0.00-0.12]
Belgium	0.09 [0.07-0.15]	0.16 [0.11-0.20]	0.10 [0.08-0.15]	0.06 [0.02-0.17]	0.16 [0.13-0.20]	0.12 [0.09-0.15]	0.10 [0.07-0.13]	0.07 [0.00-0.14]
Czech Rep.	0.10 [0.07-0.17]	0.19 [0.13-0.25]	0.14 [0.10-0.21]	0.07 [0.02-0.18]	0.18 [0.14-0.22]	0.16 [0.12-0.20]	0.12 [0.08-0.15]	0.09 [0.00-0.17]
Denmark	0.08 [0.06-0.13]	0.11 [0.06-0.14]	0.09 [0.07-0.15]	0.03 [0.01-0.14]	0.13 [0.10-0.17]	0.11 [0.08-0.14]	0.07 [0.05-0.10]	0.06 [0.00-0.13]
France	0.08 [0.06-0.14]	0.12 [0.08-0.17]	0.08 [0.06-0.14]	0.04 [0.01-0.15]	0.15 [0.11-0.19]	0.13 [0.09-0.17]	0.10 [0.07-0.13]	0.06 [0.00-0.13]
Germany	0.09 [0.08-0.15]	0.16 [0.11-0.21]	0.11 [0.09-0.17]	0.05 [0.01-0.15]	0.15 [0.12-0.19]	0.13 [0.11-0.16]	0.11 [0.08-0.14]	0.08 [0.00-0.15]
Israel	0.10 [0.06-0.18]	0.14 [0.09-0.19]	0.15 [0.10-0.22]	0.05 [0.00-0.15]	0.15 [0.10-0.21]	0.13 [0.09-0.18]	0.11 [0.06-0.17]	0.05 [0.00-0.12]
Italy	0.09 [0.07-0.15]	0.14 [0.10-0.19]	0.09 [0.07-0.15]	0.05 [0.01-0.16]	0.14 [0.11-0.18]	0.13 [0.10-0.16]	0.10 [0.07-0.13]	0.07 [0.00-0.14]
Netherlands	0.10 [0.08-0.16]	0.13 [0.08-0.18]	0.11 [0.08-0.17]	0.05 [0.01-0.14]	0.13 [0.10-0.16]	0.13 [0.10-0.15]	0.07 [0.05-0.10]	0.04 [0.00-0.11]
Poland	0.14 [0.11-0.21]	0.22 [0.16-0.27]	0.16 [0.13-0.22]	0.06 [0.02-0.17]	0.16 [0.13-0.21]	0.16 [0.13-0.20]	0.10 [0.08-0.13]	0.06 [0.00-0.12]
Spain	0.12 [0.10-0.19]	0.21 [0.15-0.27]	0.14 [0.11-0.20]	0.09 [0.03-0.18]	0.18 [0.14-0.23]	0.15 [0.12-0.18]	0.11 [0.08-0.14]	0.09 [0.00-0.16]
Sweden	0.09 [0.07-0.16]	0.13 [0.08-0.17]	0.10 [0.08-0.16]	0.05 [0.02-0.16]	0.16 [0.10-0.24]	0.11 [0.09-0.14]	0.08 [0.06-0.10]	0.04 [0.00-0.10]
Switzerland	0.06 [0.04-0.10]	0.11 [0.07-0.16]	0.09 [0.06-0.14]	0.05 [0.01-0.14]	0.14 [0.11-0.18]	0.11 [0.08-0.15]	0.09 [0.06-0.13]	0.06 [0.00-0.13]
Average	*0.10 [0.08-0.15]*	*0.15 [0.11-0.20]*	*0.11 [0.09-0.17]*	*0.05 [0.02-0.16]*	*0.16 [0.13-0.19]*	*0.13 [0.11-0.16]*	*0.10 [0.07-0.12]*	*0.06 [0.00-0.12]*
United States	0.20 [0.17-0.27]	0.23 [0.18-0.28]	0.14 [0.12-0.19]	0.06 [0.03-0.16]	0.24 [0.21-0.29]	0.21 [0.18-0.24]	0.14 [0.11-0.17]	0.07 [0.00-0.13]
Canada†	0.15 [0.12-0.22]	0.26 [0.20-0.33]	0.14 [0.11-0.20]	0.09 [0.04-0.19]	0.23 [0.19-0.28]	0.22 [0.18-0.26]	0.14 [0.10-0.17]	0.07 [0.00-0.14]
England†	0.17 [0.14-0.23]	0.22 [0.17-0.27]	0.17 [0.15-0.22]	0.09 [0.04-0.19]	0.22 [0.19-0.26]	0.18 [0.15-0.21]	0.13 [0.10-0.15]	0.11 [0.00-0.17]
United States†	0.22 [0.20-0.30]	0.26 [0.20-0.31]	0.18 [0.15-0.23]	0.09 [0.04-0.19]	0.26 [0.22-0.31]	0.23 [0.20-0.26]	0.17 [0.13-0.20]	0.10 [0.00-0.16]

Source of relative risks: Prospective Studies Collaboration. †BMI calculated using measured height and weight. 95% confidence intervals for PAFs incorporate sampling uncertainty in estimates of the distribution of BMI and estimation uncertainty in relative risks of obesity.

Table 1.3 Life expectancy at age 50 (e_{50}) in 2006 in the presence and absence of all-cause mortality attributable to obesity by country and sex (in years) [95% Confidence Intervals]

	Females			Males		
Country	**e_{50} Actual**	**e_{50} without Obesity**	**Difference**	**e_{50} Actual**	**e_{50} without Obesity**	**Difference**
Austria	33.96	34.67	0.71 [0.59-1.07]	29.39	30.39	1.00 [0.86-1.23]
Belgium	33.70	34.42	0.73 [0.61-1.16]	29.03	30.01	0.98 [0.82-1.18]
Czech Rep.	31.24	32.25	1.01 [0.85-1.40]	26.04	27.38	1.34 [1.12-1.57]
Denmark	31.90	32.52	0.62 [0.52-1.02]	28.22	29.05	0.82 [0.68-1.02]
France	35.68	36.20	0.52 [0.43-0.90]	29.86	30.85	0.99 [0.82-1.20]
Germany	33.60	34.31	0.70 [0.60-1.07]	29.07	30.12	1.05 [0.85-1.27]
Israel	33.61	34.40	0.79 [0.61-1.18]	30.64	31.56	0.92 [0.71-1.22]
Italy	35.24	35.81	0.57 [0.49-0.96]	30.57	31.47	0.90 [0.73-1.12]
Netherlands	33.31	34.00	0.69 [0.59-1.03]	29.45	30.18	0.73 [0.61-0.92]
Poland	31.39	32.58	1.19 [1.02-1.60]	24.73	26.09	1.37 [1.21-1.61]
Spain	35.40	36.27	0.87 [0.72-1.23]	29.94	31.09	1.15 [0.95-1.39]
Sweden	34.10	34.73	0.63 [0.53-1.01]	30.45	31.17	0.72 [0.59-0.92]
Switzerland	35.33	35.83	0.50 [0.41-0.84]	31.14	31.93	0.79 [0.63-0.99]
Average	*33.73*	*34.46*	*0.73 [0.63-1.13]*	*29.12*	*30.10*	*0.98 [0.86-1.16]*
United States	32.95	34.23	1.28 [1.14-1.70]	29.20	30.81	1.61 [1.44-1.82]
Canada†	34.50	35.66	1.15 [1.00-1.51]	30.72	32.09	1.37 [1.18-1.59]
England†	33.31	34.54	1.23 [1.07-1.60]	29.84	31.18	1.34 [1.13-1.53]
United States†	32.95	34.49	1.54 [1.37-1.93]	29.20	31.05	1.85 [1.62-2.10]

†BMI calculated using measured height and weight.

Table 1.4 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) [95% Confidence Intervals]

	Females				Males			
Country	**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	[0.31:0.54]	0.56	0.19	-0.42	[-0.55:-0.28]	>1.00
Belgium	0.75	0.19	[0.07:0.27]	0.74	-	-	-	-
Canada†	1.56	1.17	[0.99:1.32]	0.25	1.52	1.04	[0.89:1.21]	0.31
England†	0.36	0.05	[-0.07:0.13]	0.86	0.65	0.13	[0.02:0.26]	0.79
France	2.73	1.97	[1.83:2.08]	0.28	0.66	0.04	[-0.10:0.19]	0.94
Germany	0.66	0.08	[-0.04:0.18]	0.89	-	-	-	-
Israel	0.67	0.17	[-0.07:0.34]	0.75	1.44	0.75	[0.52:0.96]	0.48
Italy	2.29	1.58	[1.45:1.70]	0.31	1.37	0.66	[0.52:0.79]	0.52
Netherlands	0.37	-0.23	[-0.36:-0.12]	>1.00	0.25	-0.63	[-0.77:-0.50]	>1.00
Spain	2.46	2.04	[1.90:2.17]	0.17	0.74	0.27	[0.12:0.44]	0.63
Sweden	1.15	0.50	[0.37:0.60]	0.57	1.25	0.36	[0.21:0.49]	0.71
Switzerland	2.38	1.60	[1.44:1.71]	0.33	1.94	1.12	[0.96:1.24]	0.42
Average	*1.37*	*0.80*	*[0.70:0.87]*	*0.42 [0.36:0.48]*	*1.00*	*0.33*	*[0.24:0.43]*	*0.67 [0.57:0.76]*

Shortfalls in life expectancy for countries with self-reported height and weight data are calculated in respect to self-reported BMI values for the US. †Estimates are based on measured values of height and weight and are compared to US measured data. Dashed cells indicate a lower life expectancy at age 50 in that country and sex compared to the US. *The average value in this column is based on the average gap with and without obesity.

Table 1.5 Estimated gain in life expectancy at age 50 in 2006 from hypothetically redistributing obese to optimal BMI categories, using two sets of risk factors (in years) [95% Confidence Intervals].

	Females		Males	
Country	**PSC**	**Adams**	**PSC**	**Adams**
Austria	0.71 [0.59-1.07]	0.30 [0.23-0.40]	1.00 [0.86-1.23]	0.23 [0.16-0.32]
Belgium	0.73 [0.61-1.16]	0.32 [0.24-0.42]	0.98 [0.82-1.18]	0.20 [0.14-0.27]
Czech Rep.	1.01 [0.85-1.40]	0.44 [0.32-0.61]	1.34 [1.12-1.57]	0.30 [0.20-0.41]
Denmark	0.62 [0.52-1.02]	0.28 [0.19-0.38]	0.82 [0.68-1.02]	0.16 [0.10-0.23]
France	0.52 [0.43-0.90]	0.22 [0.16-0.29]	0.99 [0.82-1.20]	0.22 [0.15-0.31]
Germany	0.70 [0.60-1.07]	0.29 [0.22-0.38]	1.05 [0.85-1.27]	0.22 [0.16-0.29]
Israel	0.79 [0.61-1.18]	0.30 [0.20-0.45]	0.92 [0.71-1.22]	0.19 [0.10-0.31]
Italy	0.57 [0.49-0.96]	0.22 [0.17-0.31]	0.90 [0.73-1.12]	0.17 [0.12-0.23]
Netherlands	0.69 [0.59-1.03]	0.31 [0.23-0.41]	0.73 [0.61-0.92]	0.15 [0.10-0.20]
Poland	1.19 [1.02-1.60]	0.58 [0.45-0.75]	1.37 [1.21-1.61]	0.38 [0.26-0.53]
Spain	0.87 [0.72-1.23]	0.38 [0.28-0.50]	1.15 [0.95-1.39]	0.24 [0.16-0.33]
Sweden	0.63 [0.53-1.01]	0.26 [0.20-0.35]	0.72 [0.59-0.92]	0.17 [0.10-0.25]
Switzerland	0.50 [0.41-0.84]	0.19 [0.14-0.28]	0.79 [0.63-0.99]	0.15 [0.10-0.20]
Average	*0.73 [0.63-1.13]*	*0.31 [0.25-0.40]*	*0.98 [0.86-1.16]*	*0.21 [0.16-0.27]*
United States	1.28 [1.14-1.70]	0.71 [0.59-0.86]	1.61 [1.44-1.82]	0.52 [0.40-0.64]
Canada†	1.15 [1.00-1.51]	0.65 [0.51-0.80]	1.37 [1.18-1.59]	0.37 [0.25-0.49]
England†	1.23 [1.07-1.60]	0.61 [0.50-0.74]	1.34 [1.13-1.53]	0.33 [0.25-0.42]
United States†	1.54 [1.37-1.93]	0.88 [0.74-1.04]	1.85 [1.62-2.10]	0.62 [0.50-0.76]

The two sets of risk factors used in the calculations are drawn from the Prospective Studies Collaboration and Adams et al. †Estimates are based on measured values of height and weight.

CHAPTER 2

Using Maximum Weight to Redefine Body Mass Index Categories in Studies of the Mortality Risks of Obesity[2]

2.1 Introduction

Many studies of body mass index (BMI, measured in kg/m^2) and mortality in older adults find weak or even inverse associations between excess BMI and mortality (Corrada et al. 2006; Flegal et al. 2013; Oreopoulos et al. 2009; Stevens et al. 1998). Several physiologic and behavioral explanations for the paradoxical findings have been proposed (Flegal and Kalantar-Zadeh 2013). A statistical explanation for the weak or inverse associations identified in prior research is confounding by illness-induced weight loss—also referred to as reverse causality (Hu 2008; Wannamethee, Shaper, and Walker 2001; Willett, Dietz, and Colditz 1999).

Consistent with the statistical explanation, numerous studies find significantly stronger mortality risks of obesity after implementing measures aimed at reducing reverse causality, such as restricting samples to "healthy" participants and delaying onset of risk for several years after the time of the survey (Adams et al. 2006; De Gonzalez et al. 2010). These strategies, however, have been criticized on several grounds: the exclusions lead to eliminating a large proportion of deaths among respondents, thereby reducing the generalizability of findings (Flegal et al. 2007). Also, pre-existing illness is identified on the basis of respondent self-reports, meaning that individuals with undiagnosed illnesses cannot be excluded. Finally, delaying onset of risk for several years may not be effective

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at addressing reverse causality, as illness-induced weight loss can begin many years before death (Alley et al. 2010).

In this study, I investigate the mortality risks of obesity among older adults in the US using an approach that incorporates individual weight histories and is robust to reverse causality. Unlike other methods of addressing reverse causality, the present approach does not require excluding participants or delaying onset of risk. Instead of using BMI at time of survey, I employ a measure of maximum lifetime BMI. The advantage of the latter is that it is not susceptible to fluctuations in BMI related to illness. I also calculate the population attributable fractions for overweight and obesity for US adults implied by the estimated mortality risks.

2.2 Methods

2.2.1 Data

The National Health and Nutrition Examination Surveys (NHANES) provide nationally representative data on health for the US noninstitutional population. I used data from NHANES 3 (1988-1994) and continuous NHANES (1999-2004) to construct the cohort and obtained information on mortality status through the end of 2006 from the National Death Index (National Center for Health Statistics. Office of Analysis and Epidemiology. 2009). The sample was restricted to never-smoking adults ages 50-84. The exclusion of ever-smokers was carried out because smoking is a powerful confounder of the association between BMI and mortality (Hu 2008; Mehta and Chang 2011; Prospective Studies Collaboration 2009). After these exclusions and further eliminating those with missing data on BMI, education, smoking, and mortality status, the final analytic sample

consisted of 5,566 individuals. A total of 928 deaths occurred during follow-up in 42,815 person-years.

2.2.2 Measures

Demographic variables (gender, race/ethnicity, and educational attainment) and maximum weight were determined by interview. To ascertain maximum weight, NHANES respondents were asked, "Up to the present time, what is the most you have ever weighed?" Respondents were instructed not to include weight during pregnancy. Weight and height at the time of survey were measured by trained personnel in mobile examination clinics and used to calculate BMI at the time of survey. Maximum weight was combined with height measured at the time of survey to calculate maximum BMI. Categories of BMI at time of survey and at maximum were constructed on the basis of the continuous measures. For both variables, I used the standard WHO categories: normal (18.5-25 kg/m^2), overweight (25.0-30.0 kg/m^2), obese class 1 (30.0-35.0 kg/m^2), and obese class 2 (35.0 kg/m^2 and above). Respondents were also categorized into ten different weight trajectories (normal-normal, over-normal, obese 1-normal, obese 2-normal, over-over, obese 1-over, obese 2-over, obese 1-obese 1, obese 2-obese 1, obese 2-obese 2) on the basis of their maximum BMI and BMI at time of survey. For example, an individual who was in the obese class 2 category at their maximum and in the normal weight category at the time of survey would be categorized as "obese 2-normal."

2.2.3 Analytic Approach

Mortality rates were calculated as the ratio of the number of deaths to person-years and standardized to the US population in 2000 using five-year age groups between 50-54 and 80-84. Rates were calculated separately based on BMI at maximum and at time of survey as well as for each of 10 weight trajectories defined on the basis of both variables. Cox proportional hazards models with age as the underlying time scale were used to examine the hazard ratios associated with each BMI category relative to the reference category of normal BMI. Hazard ratios were also estimated for each of the 10 weight trajectories using normal BMI at maximum and at time of survey as the reference group. All models were adjusted for gender, race/ethnicity, and educational attainment.

I used the hazard ratios obtained above to estimate population attributable fractions (PAF). These provide an estimate of the percentage of mortality at the population level that is attributable to the combination of overweight and obesity. I use the following formula to estimate PAFs:

$$PAF_k = pd_k\left(\frac{HR_k - 1}{HR_k}\right) \quad (1)$$

Equation 1 is the appropriate formula for use with hazard ratios adjusted for confounding (Rockhill, Newman, and Weinberg 1988). In this equation, PAF_k denotes the PAF for the kth level of the risk factor, pd_k denotes exposure to risk at level k among deceased individuals, and HR_k is the hazard ratio associated with exposure level k. The exposure categories for which PAF_k is estimated include overweight, obese class 1, and obese class 2. The total PAF is obtained by summing the PAFs across exposure categories.

All estimates incorporated sampling weights that capture unequal probabilities of selection and nonresponse adjustments and accounted for the complex survey design of

NHANES. Analyses were carried out using STATA 12 (StataCorp, Texas, USA). Variances were estimated with the SVY routine, which uses Taylor series linearization.

2.3 Results

2.3.1 Descriptive Statistics

Figure 2.1 presents a comparison of the population distributions of BMI measured using time of survey and maximum values. Comparison of the two distributions reveals a greater density at higher BMI values using maximum values.

[FIGURE 2.1 HERE]

Descriptive statistics of the study sample, consisting of US adults ages 50-84 who never smoked, are presented in Table 2.1. Mean age at survey was slightly over 64 years. At the time of the survey, 20% and 12% of adults were in the obese class 1 and obese class 2 categories, respectively. When obesity status was assessed using maximum BMI, the percent obese class 1 and obese class 2 climbed to 27% and 19%.

[TABLE 2.1 HERE]

Table 2.1 also shows the population distribution across 10 categories defined using information on BMI at maximum and at time of survey. The majority of individuals (70%) were at their maximum BMI at the time of survey; 17% of individuals were in the normal BMI category both at time of survey and at their maximum BMI, and 26%, 15%, and 12% were overweight, obese class 1, and obese class 2 at both values. The remaining 30% of the population lost weight between their BMI at maximum and time of survey. The majority of individuals in this subpopulation transited between the overweight and normal (10%) or obese class 1 and overweight categories (11%). A small proportion of

the population experienced more significant weight loss, with about 2% of individuals going from obese class 2 to normal or overweight and another 2% going from obese class 1 to the normal category.

2.3.2 Analysis

Cox proportional hazards models predicting mortality for each of the two categorical measures of BMI are presented in Table 2.2. The results show a much stronger relationship using maximum values. In the specification using BMI at time of survey, the hazard ratios for obese class 1 and obese class 2 were only moderately associated with mortality and were not significant (obese class 1: 1.18 [95% confidence interval (CI), 0.91-1.54); obese class 2: 1.31 [95% CI, 0.95-1.81]). However, in the model using maximum BMI, both categories of obesity were strongly and significantly related to mortality (obese class 1: 1.67 [95% CI, 1.15-2.40]; obese class 2: 2.15 [95% CI, 1.47-3.14]).

[TABLE 2.2 HERE]

Kaplan Meier survival curves by category of BMI also reveal more substantial differences in survival across BMI categories using maximum values (Figure 2.2). A notable difference between the two sets of results is the improved survival of individuals in the normal BMI category when maximum values are used.

[FIGURE 2.2 HERE]

Table 2.3 again shows the hazard ratios for BMI at maximum and at time of survey (these results appear in the first row and column of the table). However, Table 2.3 has two additional elements. First, it includes age-standardized mortality rates (expressed as

deaths per 1,000 person-years) associated with categories of BMI at maximum and at time of survey. Second, it shows age-standardized mortality rates and hazard ratios for each combination of BMI at maximum and time of survey. This information is arrayed in a matrix with the rows identifying categories of BMI at time of survey and columns identifying BMI at maximum. Cells below the diagonal are empty because BMI at time of survey is always equal to or less than BMI at maximum.

[TABLE 2.3 HERE]

The lowest mortality rates are generally along the diagonal of the matrix corresponding to persons with stable or increasing weight. Those with the lowest mortality rates were individuals of normal weight at their maximum and survey values (7.17 [95% CI, 4.58-9.76]) (measured by deaths per 1,000 person-years), followed by individuals who were overweight (8.02 [95% CI, 6.23-9.81]) or obese class 1 (12.52 [95% CI, 8.10-16.95]) at both their maximum and survey values. Mortality rates were consistently higher in subgroups above the diagonal of the matrix—individuals who lost weight between their BMI at maximum and time of survey. The population subgroups with the highest mortality rates were those that exhibited the most weight loss, including those that went from obese class 2 to normal and overweight and individuals that went from obese class 1 to normal weight. Although the mortality rates were very large in the groups that lost the most weight, the proportion of the population in these groups was small. Only about 2% of individuals transited from obese class 2 to normal or overweight between measurements (Table 2.1).

Table 2.3 also shows that the mortality rate for normal weight individuals was higher when the category is constructed using BMI at time of survey compared to BMI at

maximum (10.42 [95% CI, 7.92-12.91] versus 7.17 [95% CI, 4.58-9.76]). This is consistent with findings from Figure 2.2 of improved survival among those in the normal category when using BMI at maximum versus BMI at time of survey.

Examination of mortality rates for combinations of BMI at maximum and time of survey reveals the source of the discrepancy. Using BMI at maximum, the normal category only includes stable normal-weight individuals. The mortality rate in this group (7.17) was lower than for any other group in Table 2.3. In contrast, the normal category defined using BMI at time of survey combines the low-risk stable-weight individuals with high-risk individuals that have experienced weight loss. About 42% percent of individuals classified as normal using time of survey values were at one point in their lives either overweight or obese (Table 2.1). Mortality rates among groups that lost weight were substantially greater: 14.16, 16.61, and 66.56 for individuals that were overweight, obese 1, and obese 2 in their past and normal weight at time of survey. The contamination of the normal weight category when it is defined using BMI at time of survey explains why the mortality risks of overweight and obesity grew stronger after substituting maximum BMI for BMI at time of survey in Table 2.2.

Table 2.4 shows population attributable fractions for overweight and obesity based on BMI at survey and at maximum. Category-specific and overall PAFs are given. Using BMI at survey, an estimated 5.41% of deaths were attributable to the combination of overweight and obesity, whereas using maximum BMI, the attributable risk was about six times greater, at 32.16%.

[TABLE 2.4 HERE]

2.4 Discussion

Among older never-smoking adults in the US, use of maximum values for assessing the mortality risks of overweight and obesity yield much stronger associations between excess weight and mortality than using BMI at the time of survey. The analysis of the percentage of mortality attributable to overweight and obesity indicates that use of BMI at the time of survey may significantly underestimate the associated burden of excess weight in the US. Attributable mortality is about six times higher in the analysis using maximum values—32% compared to 5%.

The discrepancy in results relates to who is classified as normal weight across the two measures. This is clearly revealed in examining mortality rates for combinations of BMI at maximum and time of survey. When BMI is assessed at time of survey, the normal weight category includes those who have lost weight from their maximum BMI and are at significantly higher risk for death. Assessment of BMI using maximum values removes the confounding, as the reference group is restricted to individuals whose BMIs never exceeded the normal weight category.

Mortality risks were higher in the present study among those subpopulations that lost weight between their maximum and baseline values. This finding is consistent with prior studies that have also identified weight loss as a strong risk factor for mortality (Kuller and Wing 1993; Myrskyla and Chang 2009; Wannamethee et al. 2001; Zajacova and Ailshire 2013; Zheng, Tumin, and Qian 2013). One explanation for this finding is that most weight loss is associated with illness, masking any beneficial effects of lifestyle modification. A British study that investigated weight loss and mortality found that among individuals losing weight, 78% lost weight because of ill-health—either

unintentionally or intentionally—versus the remaining 22% who lost weight for other reasons (Wannamethee, Shaper, and Lennon 2005).

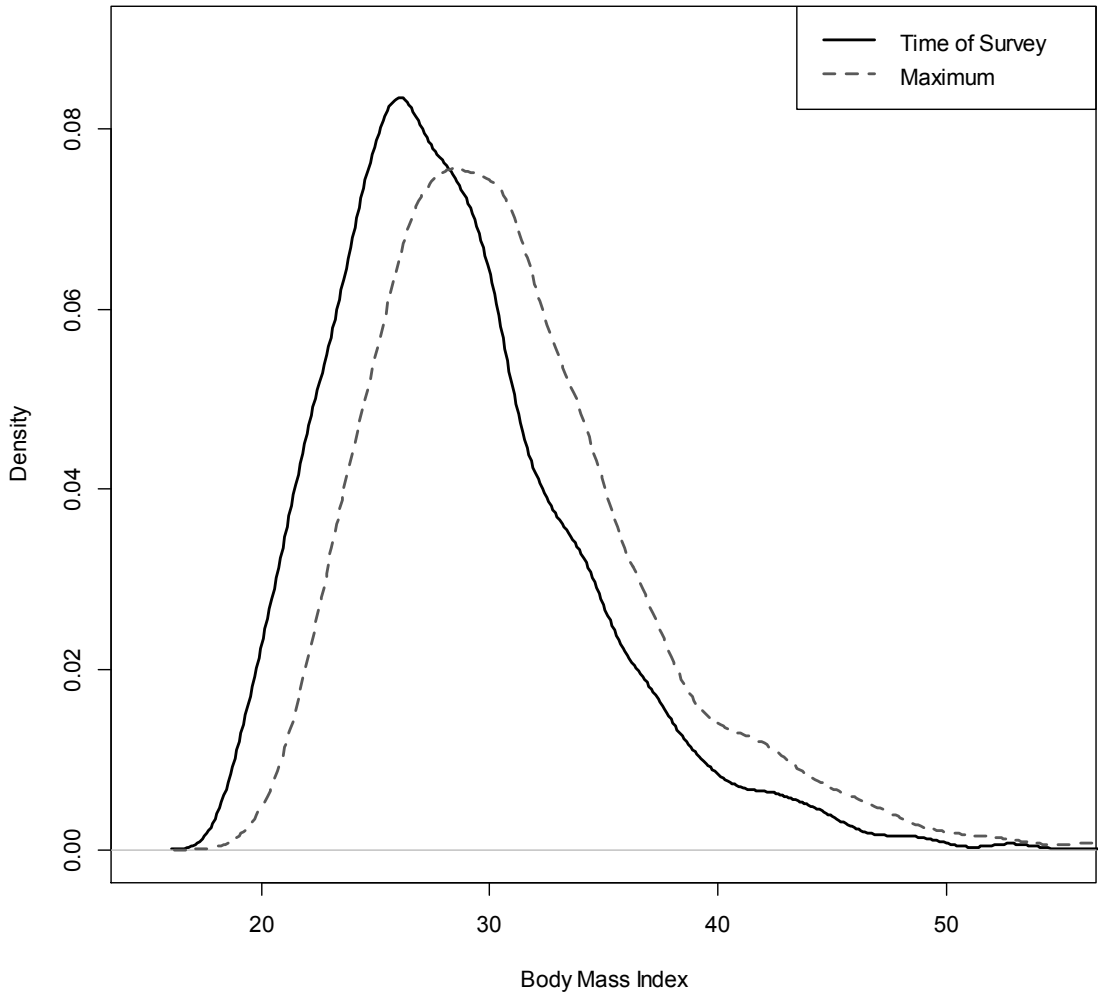
Several prior studies have introduced alternative measures of BMI into analyses of obesity and mortality with the aim of reducing bias due to the effects of reverse causality (Adams et al. 2006; Greenberg 2001; Smith et al. 2009). In each case, stronger associations were identified, consistent with the findings from the present study.

The present study has several limitations. First, as maximum weight was self-reported, it may be subject to recall bias. If respondents tend to underreport their maximum weight, some individuals may be incorrectly assigned to a lower BMI category. The effects of this bias on the estimated mortality risks of obesity are unclear, as it may lead to mortality rates being overestimated in both the normal and obese categories. Because the analyses used a categorical measure of BMI, potential for misclassification was reduced. Furthermore, validation studies of weight recall support their validity for use in epidemiological studies (Casey and Dwyer 1991; Perry et al. 1995). A second limitation arises from using height at survey to calculate maximum BMI. Because of the tendency for height loss at older ages, maximum BMI may have been overestimated in some respondents. This would be expected to dilute mortality rates in the overweight and obese categories, leading to more conservative estimates of the mortality risks of obesity. A third source of bias is differential mortality of obese individuals. Some individuals who were heavy in their past may not have survived to the time of the survey to report their maximum weight. This bias may also produce conservative estimates. Future research should replicate the analyses presented here using

prospective cohort data containing contemporaneous measures of height and weight across the lifecycle.

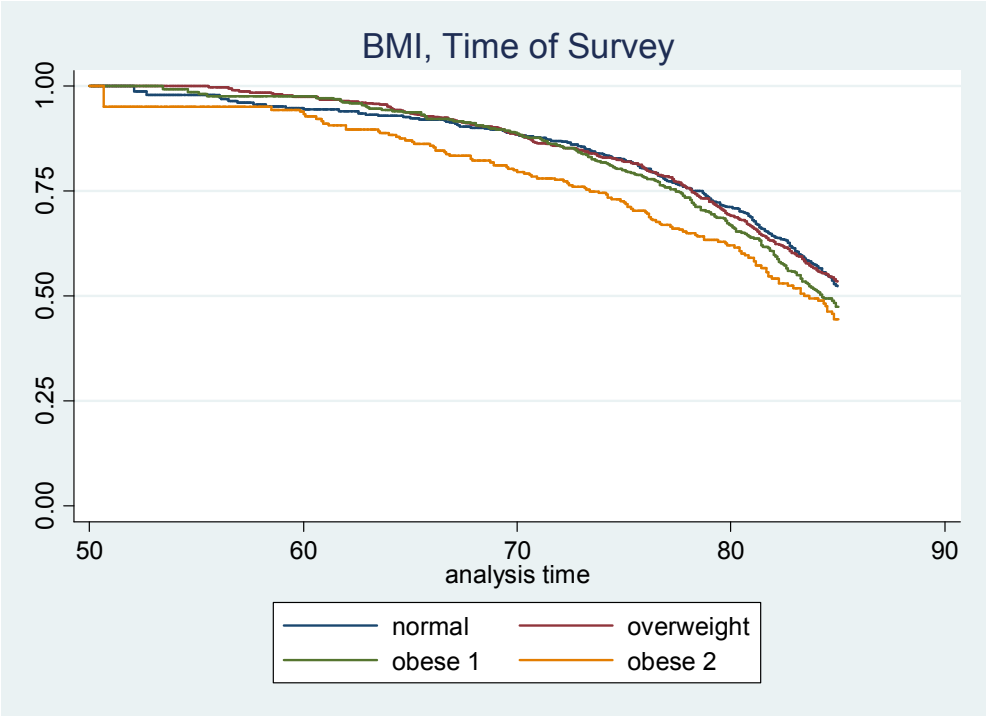
Prior assessments of associations between excess weight and mortality underestimate mortality risks because of reverse causality owing to the high prevalence of disease in aging populations. The present study suggests that the impact of overweight and obesity on mortality at the population level is likely much larger than is appreciated. As maximum lifetime BMI is highly predictive of mortality, an additional implication of this study is that individual obesity histories should be ascertained in clinical settings to obtain a more complete understanding of individuals' mortality risks.

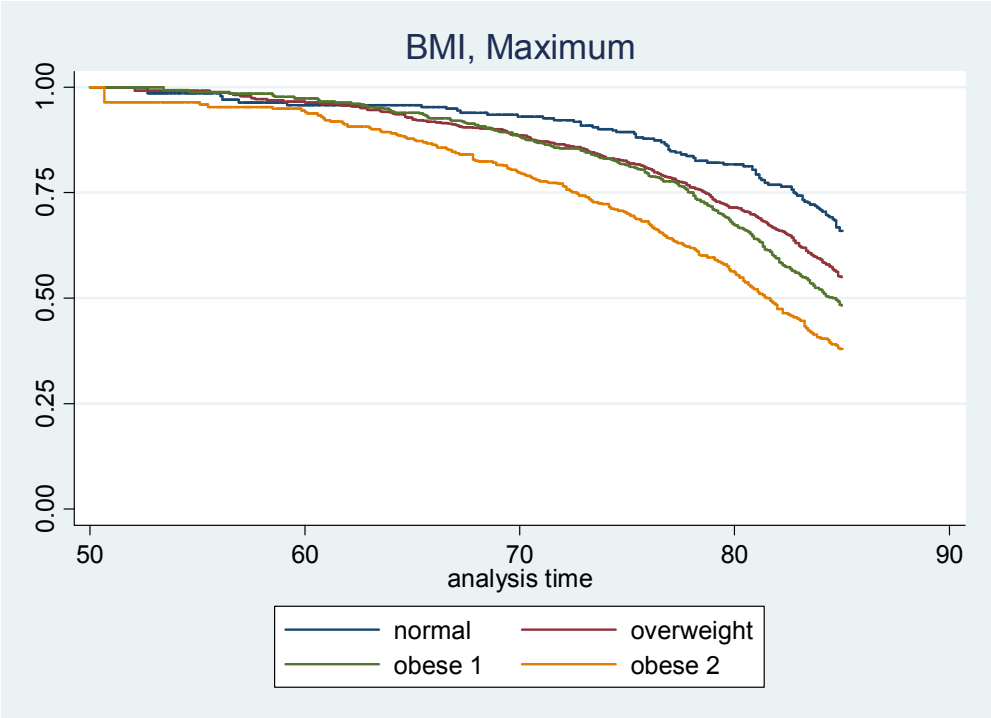
Figure 2.1 Distribution of body mass index at time of survey and at maximum among US never-smoking adults ages 50-84



Distributions are unweighted. Source: National Health and Nutrition Examination Survey.

Figure 2.2 Kaplan Meier curves for categories of BMI at time of survey and at maximum





Categories of BMI are normal weight (18.5-25.0 kg/m^2); overweight (25.0-29.9 kg/m^2); obese class 1 (30.0-34.9 kg/m^2); and obese class 2 (35.0 kg/m^2 or greater). The sample includes persons ages 50-84 who never smoked. Entry years are 1988-2004 with mortality follow-up through 2006. Estimates are weighted and account for complex survey design. Source: National Health and Nutrition Examination Survey.

Table 2.1 Characteristics of US never-smoking adults ages 50-84

	No.	% or mean
Age at survey, years		64.14
Age of exposure, years		67.60
Education		
Less than high school	2,466	28.35
High school or equiv.	1,395	28.91
More than high school	1,684	42.74
Race/ethnicity		
Hispanic	1,380	8.55
Non-Hispanic white	2,950	77.79
Non-Hispanic black	1,089	9.24
Non-Hispanic other	147	4.42
Obesity status at survey		
Normal	1,549	29.38
Overweight	2,176	37.71
Obese class I	1,157	19.79
Obese class II	684	12.11
Obesity status at maximum		
Normal	770	17.77
Overweight	1,994	36.04
Obese class I	1,657	27.17
Obese class II	1,145	19.01
Obesity status: maximum-survey		
Normal - normal	770	17.07
Over - normal	635	10.44
Obese 1 - normal	119	1.81
Obese 2 - normal	25	0.36
Over - over	1,359	25.90
Obese 1 - over	704	10.60
Obese 2 - over	113	1.59
Obese 1 - obese 1	834	15.00
Obese 2 - obese 1	323	4.99
Obese 2 - obese 2	684	12.24
Deceased	928	12.09
Total	5566	

Categories of BMI are normal weight (18.5-25.0 kg/m^2); overweight (25.0-29.9 kg/m^2); obese class 1 (30.0-34.9 kg/m^2); and obese class 2 (35.0 kg/m^2 or greater). Entry years

are 1988-2004 with mortality follow-up through 2006. Source: National Health and Nutrition Examination Survey.

Table 2.2 Hazard ratios for mortality from all causes according to body mass index at time of survey and body mass index at maximum

	BMI, time of survey		BMI, maximum		
BMI category (kg/m^2)	Hazard ratio	95% CI	Hazard ratio		95% CI
Normal	1.00		1.00		
Overweight	0.98	(0.77-1.24)	1.28		(0.89-1.84)
Obese class 1	1.18	(0.91-1.54)	1.67	**	(1.15-2.40)
Obese class 2	1.31	(0.95-1.81)	2.15	***	(1.47-3.14)

BMI: body mass index. See Table 2.1 for definitions of BMI categories. The sample includes never-smoking persons ages 50-84. Entry years are 1988-2004 with mortality follow-up through 2006. Hazard ratios are derived from Cox proportional hazards models that adjust for gender, race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, other), and educational attainment (less than high school, high school, some college, or greater). Age at exposure is specified as analysis time. The reference category in both regressions is the normal category. All estimates are weighted and account for complex survey design. Source: National Health and Nutrition Examination Survey.

***p<0.001; **<0.01; *p<0.05

Table 2.3 Age-standardized all-cause mortality rates (per 1,000 person-years) and hazard ratios for mortality from all causes according to combinations of body mass index at time of survey and body mass index at maximum

			BMI, maximum							
			Normal		Overweight		Obese class 1		Obese class 2	
	7.17	(4.58-9.76)	9.74	(7.64-11.84)	13.87	(10.64-17.09)	16.88	(13.68-20.09)
BMI, time of survey	1.00		1.28	(0.89-1.84)	1.67	(1.15-2.40)	2.15	(1.47-3.14)
Normal								
Mortality rate	10.42	(7.92-12.91)	7.17	(4.58-9.76)	14.16	(7.98-20.34)	16.61	(10.29-22.93)	66.56	(17.41-115.70)
Hazard ratio	1.00		1.00		1.69	(1.12-2.56)	2.69	(1.67-4.33)	4.97	(2.01-12.27)
Overweight										
Mortality rate	10.51	(8.62-12.39)			8.02	(6.23-9.81)	15.25	(10.83-19.66)	22.17	(12.45-31.90)
Hazard ratio	0.98	(0.77-1.24)			1.10	(0.76-1.60)	1.76	(1.16-2.66)	3.06	(1.72-5.44)
Obese class 1										
Mortality rate	13.87	(10.04-17.69)					12.52	(8.10-16.95)	17.88	(12.41-23.35)
Hazard ratio	1.18	(0.91-1.54)					1.48	(0.98-2.24)	2.28	(1.54-3.36)
Obese class 2										
Mortality rate	14.55	(10.90-18.20)							14.55	(10.90-18.20)
Hazard ratio	1.31	(0.95-1.81)							1.85	(1.18-2.89)

BMI: body mass index. See Table 2.1 for definitions of BMI categories. The sample includes never-smoking persons ages 50-84. Entry years are 1988-2004 with mortality follow-up through 2006. Mortality rates are age-standardized to the US 2000 Census using five-year age-groups between 50-54 and 80-84. First row and column correspond to mortality rates pooled across BMI at the time of survey and across maximum BMI categories, respectively. Hazard ratios are derived from separate calculations in which adjustment is made for gender, race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, other), and educational attainment (less than high school, high school, some

college, or greater) using Cox proportional hazards models. Age at exposure is specified as analysis time in all models. All estimates are weighted and account for complex survey design. Source: National Health and Nutrition Examination Survey

Table 2.4 Population attributable fractions estimated using body mass index at time of survey and body mass index at maximum

	BMI, time of survey			BMI, maximum		
BMI category (kg/m^2)	Pd (%)	HR	PAF (%)	Pd (%)	HR	PAF (%)
Normal	29.63	1.00	0	12.77	1.00	0
Overweight	36.81	0.98	-0.75	32.73	1.28	7.16
Obese class 1	21.11	1.18	3.22	31.04	1.67	12.45
Obese class 2	12.45	1.31	2.95	23.45	2.15	12.54
Total			5.41			32.16

BMI: body mass index; Pd: proportion exposed among decedents (%); HR: hazard ratio; PAF: population attributable fraction. See Table 2.1 for definitions of BMI categories. The PAF for each exposure category is calculated using Equation 1 in the text. PAFs are summed across exposure categories to obtain the overall PAF. Calculations are based on the sample of never-smoking adults ages 50-84.

CHAPTER 3

The Family Dinner and Obesity among US Adults: A Time-Use Study

3.1 Introduction

Obesity is among the leading risk factors for mortality in the United States (Danaei et al. 2009). The prevalence of obesity, defined as a body mass index (BMI) in excess of 30 kg/m^2, grew from less than 10% in the 1950s to over 30% today. Obesity has also risen in other countries, but to a lesser extent: the average prevalence of obesity in OECD countries stands at 18% (Preston and Stokes 2011). Comparisons of morbid obesity present an even sharper contrast: the prevalence of obese class II (BMI>=35 kg/m^2) is 12.3% in the US compared to 4.2% in OECD countries and the prevalence of obese class III (BMI>=40 kg/m^2) is 5.1% compared to 1.1%.

Prior research has highlighted a variety of explanations—economic, ecological and socio-cultural—for the rise of obesity in the United States. Economic explanations include the transition to an industrial food economy, employment shifts from manufacturing to services and changes in technology that have increasingly brought processed and ready-made foods into the household and made them affordable (Cutler, Glaeser, and Shapiro 2003; Sturm and An 2014). Ecological explanations have focused on the role of changes in the built environment, including the construction of highways, suburbanization, increasing reliance on the automobile and neighborhoods that are less walkable (Wang et al. 2011). A third body of research has focused on the role of social and cultural change, including the rise of sedentary behaviors such as watching TV and consumption of digital media (Katzmarzyk 2009).

I advance a sociological perspective on the rise of obesity in the United States—one which might make sense not only of recent trends in the US, but why the US is an outlier with respect to most other highly developed countries. I argue that expanding waistlines cannot be attributed solely to declining food prices or increasing availability of convenience food—social norms have also changed in such a way as to make it socially acceptable for individuals to buy and consume food in an increasing variety of non-traditional ways, such as eating food away from home, eating alone and eating in a larger variety of settings (e.g. at work, in the car, in front of the computer or TV). Thus, social as well as economic or ecological factors must figure into any explanation of the rise of obesity if it is to be comprehensive.[3]

These changes may be described as informalization (Wouters 2007) in which social constraints and traditions have gradually become relaxed and have lost their salience. Historically, I argue that formality brought structure to eating and drinking, helping to regulate appetite and reduce energy intake. Formality imposed controls on both the quantity and quality of food consumed as well as meal duration, leading to smaller, more balanced meals over longer periods of time. With increasing informalization, this structure dissipated and a wider variety of eating patterns gained acceptance, setting the foundation for increased energy intake.

The importance of external constraints in promoting healthful eating behaviors is supported by evidence suggesting that individuals are poor judges of food intake and are easily fooled into eating more calories than they think they are (Wansink 2007). Indeed,

[3] Of course, these processes can also operate in tandem. To the extent that changes in eating behaviors (such as a shift towards shorter eating occasions and eating in informal settings) are a direct consequence of structural factors (such as the rise of fast food) it may be difficult to fully disentangle the independent effects of changing social norms on the rising prevalence of obesity. I return to this point in the discussion section.

experimental evidence suggests strong effects of portion size on energy intake independent of other factors that influence energy intake, such as hunger (Rolls, Morris, and Roe 2002). In one particularly illustrative study, Wansink and colleagues found that people ate much more tomato soup than usual when their bowls were filled secretly from below the table (Wansink 2007). Satiety was not being judged based on 'feeling full' but instead using the measure of the amount left in the bowl. In a different study, students' normal food intake was measured at a lunch buffet, after which they returned and were allotted 100, 125 and 150 percent of this portion. The more students were served, the more they ate (Levitsky and Youn 2004).

Against this backdrop, formal eating patterns provide a crucial mechanism for regulating the appetite and food consumption. Changes in the social context of eating towards informality may have upset this delicate balance by removing external influences on energy intake. Importantly, even moderate changes in daily eating habits sustained over a lifetime can generate effects at the population level: prior research shows that preventing 100 calories of intake every day (equivalent to McDonalds kids size French Fries) would prevent weight gain in most of US population (Wansink 2007).

In this paper, I contribute to the literature on social aspects of eating by using time diary data from a nationally representative sample of Americans to study the association between eating patterns and obesity. I focus on one particular eating ritual: the family dinner. The extraordinary detail present in the time diaries, including information on when, where and with whom activities are carried out, permits characterizing participation in the family meal in more detail than has been possible in prior studies. In the current paper, I define the family dinner as a primary eating episode in the interval

4:30-10 PM of at least 15 minutes in duration that occurred at home with at least one other family member present. Family members are defined as individuals that are related to the respondent and co-reside in the same household. Since individuals who live alone cannot have a family meal under this definition, they are excluded from the analysis. I hypothesize that regular participation in family dinners, an indicator of the formality of eating behaviors—is associated with a lower probability of obesity. Since what constitutes a family dinner may vary based on the resources families have at their disposal as well as household structure, I also explore the extent to which the association between family dinner and obesity varies by socio-economic status and the number of children living in the household.

3.2 Background

3.2.1 Informalization

In his book, *The Civilizing Processes*, Norbert Elias used books on manners to document the accretion of manners in Western Europe between the fifteenth and nineteenth centuries (Elias 1969). In early medieval times, only very basic rules of etiquette and propriety accompanied the activities of daily life. Over time, he found that the upper classes adopted increasingly complex rules for the regulation of behavior. Gradually, as the performance of these rules became naturalized, their articulation in books of manners became less and less necessary and eventually disappeared. According to Elias, external constraint was replaced by internalized self-restraint (Elias 1969). It eventually became improper to discuss openly standards of etiquette for which it was once necessary to have explicit rules. The rise of the bourgeois and the increased mixing of the trades and classes

throughout this period caused manners to spread more widely in society. These manners eventually came to shape the national habitus.

In *Informalization: Manners and Emotions since 1890*, Cas Wouters finds that the 20th century marked a discontinuity with respect to these earlier trends (Wouters 2007). The 20th century, he argues, was characterized by a process of informalization, in which the outward display of etiquette and manners declined. Social forces, including social upheaval and democratization, led to increased mixing of various social groups and a gradual reconciliation of their norms (Wouters 2007). As this leveling progressed, social distance between groups was reduced and the etiquette and codes used by upper-class groups to distinguish themselves lost social acceptability. Wouters argues that emphasis shifted increasingly to appearing natural and unpretentious in social situations. Particularly, it became important to appear unconstrained. Behavior according to formal codes became increasingly associated with insincerity and deceitfulness and an antiquated class structure.

This process affected various domains of life, including eating behaviors. Prior to the 20th century, eating rituals were more highly circumscribed, with individuals gathering for family meals at least once a day. Very little eating occurred outside the context of the family meal and that eating which did occur was highly regulated. Eating alone or in the context of other activities was rare and in general not deemed socially acceptable. With informalization, eating rituals became less circumscribed and less subject to formal social control. As a consequence, the social context of food consumption became increasingly varied and eating behaviors, including secondary eating (e.g. eating while performing other activities), have become increasingly common.

3.2.2 Eating Patterns and Health

Most articles to date on eating patterns and health have focused on childhood and adolescence, stages of the life course where the benefits of establishing healthy eating patterns are presumed to be the most significant. Both for children and for the population in its entirety, descriptive evidence indicates that primary eating has declined while episodes of secondary eating and drinking have increased significantly between 1975 and 2006 (Zick and Stevens 2010). Popkin and Duffey examine the incidence of any eating episodes over a similar time period finding that they have increased on average from about 3 to 5 occasions (Popkin and Duffey 2010). Examining more recent trends in a sample of adolescents from urban public schools in Minnesota, Neumark et al. found that the frequency of family meals did not change as a whole over the last decade, but that this masked divergence in family meal patterns across SES, with higher-SES adolescents experiencing higher rates of family meals over time and lower-SES adolescents experiencing lower rates of family meals over time (Neumark-Sztainer et al. 2013). The authors speculate that economic and employment related factors may present barriers to low SES families gathering for family meals. These include economic stress, unemployment, underemployment, the necessity to work multiple part time jobs and limited flexibility in work schedules.

Eating patterns in children and adolescents have also been evaluated with respect to dietary quality (Burgess-Champoux et al. 2009; Hammons and Fiese 2011; Larson et al. 2007; Shea, Harvey-Berino, and Johnson 2010; Videon and Manning 2003; Woodruff and Hanning 2009), obesity (Taveras and Rifas-Shiman 2005; Rollins, Belue, and Francis 2010; Sen 2006) and mental health and substance abuse (Musick and Meier 2012;

Fulkerson et al. 2009). The key explanatory variable chosen in the overwhelming majority of studies on eating behaviors and health is participation in the family meal—particularly the family dinner.

A recent meta-analysis of 17 studies and a total of 182,830 children finds that the frequency of shared family meals is significantly associated with a range of beneficial outcomes, including lower overweight/obesity and less disordered eating (Hammons and Fiese 2011). Children who shared at least three meals with their family per week were significantly less likely to be obese and more likely to have healthy dietary patterns than children who participated in less than three family meals per week. The authors speculate on a few of the mechanisms that may explain the relationship, including that family meals may increase home-preparation of foods and encourage family interaction (also see (Fiese and Schwartz 2008)). In a separate article, Skafida argues that the mechanism by which the family meal may promote adolescent health may be by increasing the probability that children eat the food being consumed by their parents, which she argues is generally healthier than children's foods (Skafida 2013).

Increased home preparation of food may be a significant factor in light of evidence that finds that in the US a greater proportion of food away from home correlates negatively with dietary quality (Todd et al. 2010). However, Hammons and Fiese also discuss how the effects of the family meal may not be in all cases positive, where family interactions are negative or involve television and digital media (Hammons and Fiese 2011). Indeed, a recent poll reveals that digital media are becoming increasingly used in the context of the family meal (NPR and Robert Wood Johnson Foundation 2013). Fiese et al. 2012 explore the context of the family meal in detail in an observational study of

200 family meal times (Fiese, Hammons, and Grigsby-Toussaint 2012). Families of normal weight children were more likely to be engaged with each other during the meal, had better communication and ranked meals as more important ritual than families of overweight and obese children, providing support for some of the mechanisms stipulated in earlier studies.

Time constraints are emphasized in numerous articles in the literature on eating patterns and health (Neumark-Sztainer et al. 2013; Cawley and Liu 2012; Jabs and Devine 2006; Celnik, Gillespie, and Lean 2012). For example, Cawley and Liu use time use data to examine mechanisms by which maternal employment might give rise to higher levels of child obesity (Cawley and Liu 2012). They find that employed mothers spend less time cooking and grocery shopping and that these changes are generally not offset by more time spent in these activities by husbands. Although the study doesn't directly evaluate the impact of maternal employment on childhood obesity, taken together with other evidence that more time spent in food preparation is associated with higher diet quality (Mancino and Gregory 2012), it suggests that time scarcity could be an important mechanism generating unhealthy eating behaviors and obesity.[4]

A subset of the literature expresses skeptical or cautionary notes about the role of the family meal in promoting child and adolescent health. For example, Musick cautions that the family meal may to some extent serve as a proxy for other aspects of the family environment and may not in itself be the key causal factor promoting beneficial outcomes for children (Musick and Meier 2012).

[4] Of note, despite increased perceptions of time scarcity, time spent working has actually declined over time for the average worker (Jacobs, Jerry and Gerson 2004). This paradox is likely to be explained in part by the dramatic rise in labor force participation of women and the accompanying challenges of juggling work and family in two-career households.

Few studies have examined eating patterns and health in adults. Two studies have addressed cross-cultural similarities and differences in eating patterns (Rozin, Remick, and Fischler 2011; Warde et al. 2007). The study by Rozin and colleagues compared attitudes towards food and eating in the US and France, while Warde et al. examined similarities and differences in how eating behaviors changed over time in a sample of 5 high-income countries. Zick et al. examine the association between many different dimensions of time use and obesity using the American Time-Use Survey (ATUS) (Zick, Stevens, and Bryant 2011). They find significant inverse associations between time spent in primary eating (e.g. primary refers to the fact that eating is the main activity being carried out) and obesity and strong positive associations between time spent in secondary drinking and obesity. Surprisingly, time spent in secondary eating (e.g. eating performed in the context of other activities such as driving or working) was found to negatively correlate with obesity. Less surprising, time spent in food preparation was negatively associated with obesity while a positive association was identified for time spent in sedentary behaviors. Vorisek also studies time use and obesity among adults in the ATUS (Vorisek 2012). The author stratifies the sample by obesity status and examines variations in time use across the sub-groups, finding systematic variation, with obese individuals spending less time in food preparation and grocery shopping.

3.2.3 Mechanisms

I have hypothesized that participation in family dinners is associated with a lower probability of obesity. Prior research suggests a number of mechanisms that may explain this relationship. I discuss those related to food as a symbol of family, social norms and

gatekeeper effects. These mechanisms act on choices of food, portion size, frequency and length of eating occasions and the social context of eating (i.e. whether eating occurs in front of the TV, in the car or at the dinner table; in other words as a primary or secondary activity).

Food as a symbol of family The term "ritual" as used by sociologists such as Durkheim can be defined as "a mechanism of mutually focused emotion and attention producing a momentary shared reality, which thereby generates solidarity and symbols of group membership" (Collins 2004). Durkheim analyzed rituals to show how they give rise to religious beliefs (Durkheim 1912). Collins subsequently elaborated on Durkheim in his theoretical model of interaction rituals by formally spelling out the elements of a ritual as well as anticipated outcomes (Collins 2004). The elements he identified were assembly of a group, mutual focus of attention and common mood and exclusion of outside non-participants, whereas the outcomes included group solidarity, identity and creation of sacred objects and symbols representing the group. Outcomes vary in their intensity depending on the presence or absence as well as the strength of each of the elements.

Applying this model to the family dinner is fruitful for understanding pathways by which family dinner participation may affect eating behaviors. Under the assumption that the family dinner is a successful ritual[5] (i.e. all the main input variables are

[5] Although this assumption pervades much of the literature on family meals, it may not always be true. See Hammons for a discussion (Hammons and Fiese 2011). In some cases, the family meal may be a "weak" ritual in the sense that some or all of the ritual elements are missing. For example, despite assembly of the group for the meal, mutual focus of attention and common mood may be missing, if interpersonal dynamics are poor or family members are occupied or distracted (e.g. cell-phones) (NPR and Robert Wood Johnson Foundation 2013).

present and strong), the family dinner generates feelings of group solidarity and emotional energy. These translate into the creation of symbols that become emblematic of family, intimacy and the emotional energy of the mealtime.

The most significant symbol of the family dinner is the food itself. Where food is primarily consumed at the table with family members, food becomes synonymous with family and its most sacred symbol. Other symbols of formal eating ritual can include the table-ware, glasses, table-cloth and other accessories of eating. While many of these have a practical purpose, as symbols, their function goes beyond the utilitarian. Plates and bowls may be decorated with artwork, the utensils made with silver, glasses made from crystal and all of these items displayed in the dining room in glass cabinets. These items are often inherited through several generations, giving them additional layers of symbolic value as representations of the continuity of family.

The family dinner also creates feelings of morality or the sense that one is obligated to respect as well as defend the family and its symbols. Thus, the manner in which the family eats becomes the right way to eat. Patterns of eating that do not conform to this model or attitudes and behaviors that degrade food are deviant.

As a consequence of the ritual performance of the family dinner, eating outside of formal venues and the family context come to represent a diminishment of the significance of family and food. These values may be expected to reduce the incidence of informal eating patterns, including snacking, eating alone, eating food away from home and eating in the context of other activities, such as working, driving or watching TV.

Social and cultural norms Social and cultural norms may be further divided into expectations and emulation, pacing and habituation effects and cultural norms. Expectations are collective beliefs held by groups regarding proper eating behaviors. Individuals must adhere to etiquette when eating in groups. They must conform to social norms of the group and not engage in selfish behaviors. This enforces restraint and limits overeating. In making efforts to fulfill social expectations, individuals may moderate portion size and choice of foods, speed of eating and the social context of eating. That is, they may be discouraged from eating alone or while multi-tasking. In the context of families with children, parents may moderate their own consumption to set examples for their children. Emulation effects refer to individuals mirroring the behavior of others in social eating occasions. One may moderate his or her food consumption so as not to exceed that consumed by others at the table. They may also eat a balanced meal by emulation—eating foods (e.g. broccoli) that they would normally avoid—because others are eating them. Pacing effects refer to the effects of the family meal or social eating occasions on the pace of eating. Eating in the context of a family meal is likely to slow down the pace at which individuals eat, which has the potential to reduce energy intake (Robinson et al. 2014).

Habituation effects refer to the structure that the family meal provides with respect to eating occasions. Family meals place individuals on an eating schedule and bring the appetite into line with the meal routine. This restrains hunger at other times and is likely to reduce the incidence of snacking. Prior research shows that eating habits form early and persist (Wansink 2007), suggesting that the appetite may be adapted to established social routines of eating. Cultural factors may also be

important. In some cases, positive cultural traditions of delight in food and a preference for small portions may act as a restraint on overeating (Rozin et al. 2011).

Nutritional gatekeeper A third way in which the family meal may affect obesity is through the role that the family meal affords to the nutritional gatekeeper (Fiese and Schwartz 2008). This effect is particularly salient for children, but also applies to adults. The role of gatekeeper was traditionally performed by women, but has become more varied over time, with the male parent, grandparent or non-family member often assuming sole or shared responsibility. The gatekeeper is the person through whom decisions on food purchasing and preparation are filtered. Although they may or may not be the ones to actually purchase and prepare meals, they are typically the ones making the decisions on these matters. Importantly, the gatekeeper makes decisions on what food to bring into the household before eating occasions occur. This has the beneficial effect of reducing impulse buying/eating by distancing food consumption from decisions on food purchase. Frequent family meals tend to concentrate power in the hands of the gatekeeper, increasing their discretion over the quality and quantity of the food consumed by family members (Wansink 2007). Compared to a decentralized system in which each individual makes his or her own decisions on what to eat and when, this system may reduce the likelihood of unrestrained or irregular eating.

The proposed mechanisms discussed above—food as a symbol of family, social and cultural norms and the nutritional gatekeeper—offer several pathways by which the

family dinner may lower obesity risk. The commonality among them is that they provide structure to eating behaviors and reduce unrestrained eating.

3.2.4 Potential Effect Modifiers

The prior literature points to several potential effect modifiers of the association between family dinner participation and obesity, including socio-economic status (SES) and household structure. With respect to SES, one reason the association may differ is that low-income households do not have sufficient resources at their disposal to prepare healthy family meals (Hammons and Fiese 2011). This may not only include financial resources for purchasing fresh ingredients but also non-financial resources such as access to neighborhood grocery stores, farmer's markets and fruit and vegetable stands (Franco et al. 2008; Andreyeva et al. 2008; Baker et al. 2006). Another explanation is time-constraints, which may limit the ability of low-SES households to prepare meals from scratch. In these circumstances, a family meal may mean eating take-out together from a fast-food restaurant. In each case, family meals may not yield the same benefits to low-income households as they do for more affluent households.

Household structure is another potential effect modifier of the association between family dinner participation and obesity. Families with a larger number of children may face more significant time constraints that limit their ability to prepare home-cooked meals. Such families may more readily sacrifice quality for expediency in order to meet the demands of a larger household. A second possibility is that having more children in the household shifts consumption towards foods marketed to and preferred by

children (e.g. fish sticks, chicken nuggets, pizza), which tend to be less healthy in terms of nutrients and calories than foods typically consumed by adults (Harris et al. 2009).

On the basis of the above discussion, I hypothesize that the association between family dinner participation and obesity will be stronger for individuals in high- as compared to low-SES households and stronger for individuals with smaller vs. larger families. In the next section, I present my empirical strategy for examining the association between family dinner participation and obesity.

3.3 Methods

3.3.1 Data

Data for this study are drawn from the American Time-Use Survey (ATUS), a nationally representative sample of the civilian non-institutionalized population of the United States (Bureau of Labor Statistics 2013a). The ATUS commenced in 2003 and includes data on approximately 25,000 respondents per year. Respondents are selected at random from households that have completed their eighth interview for the Current Population Survey (CPS) 2-5 months earlier. Individuals aged 15 and above are eligible to participate. Time-use data in the ATUS are collected in computer-assisted telephone interviews for the 24-hr period prior to survey. Interviews are dispersed evenly between weekdays and weekends to ensure that the data reflect the range of time-use patterns across the days of the week. For each respondent, data are collected on the number of minutes spent in various activities in addition to where the activity took place and with whom.

As the ATUS is drawn from the CPS, the two surveys can be linked, providing access to a rich set of socio-economic variables. Special supplements to the ATUS have

been periodically conducted, including a module on Eating and Health in 2006-2008 (Bureau of Labor Statistics 2013b). This module is unique in several respects. First, it contains additional detail on eating behaviors, not available in ATUS, and second, it includes information on body mass index (BMI; measured in kg/m^2) and self-rated health, allowing analyses of the relationship between time-use patterns and health outcomes.

The ATUS was linked with data from the CPS and the ATUS Eating and Health module for the current analysis.[6] As the Eating and Health data were only collected in years 2006-2008, I restricted the analysis to that time period. Adults aged 25-49 were included in the analysis. Adults between the ages of 18 and 25 were excluded as this group includes many students, whereas older adult were excluded because BMI in the normal weight range at older ages is often a marker of illness rather than an indicator of optimal health (Willett, Dietz, and Colditz 1999).

As the key explanatory variable in the analysis is the family dinner, the sample was restricted to respondents co-residing with family members (e.g. related individuals). Thus individuals living alone and individuals co-residing with non-family members, such as with roommates, were excluded.[7] Individuals who perceived themselves to be in 'poor' health were also excluded as were individuals whose diary day coincided with a holiday. Individuals in poor health were excluded to reduce potential for reverse causality between eating behaviors and body mass index (BMI). Data from holidays were excluded

[6] The following data sets were combined for the analysis: the ATUS respondent file (socio-demographic information on each respondent); the ATUS roster file (containing information on members of the household); the ATUS activity file (containing information on all activities reported in the time-diaries); the ATUS WHO file (containing information on the person(s) with whom the respondent participated in each activity; the ATUS CPS file (containing CPS data with unique identifiers that enable linkage to ATUS); and the ATUS Eating and Health Respondent and Activity Files.

[7] If the respondent reported co-residing with a combination of family and non-family members, they were retained in the analytic sample.

because eating patterns on these days were unlikely to be representative of respondents' long-term eating behaviors. Lastly, respondents reporting BMI below 18.5 kg/m^2 were excluded as underweight is often indicative of a pre-existing illness. After these exclusions, the final analytic sample consisted of 12,667 respondents.

3.3.2 Measures

In this section, I describe the measures that were constructed to examine the association between family dinner participation and obesity, including the dependent variable, covariates and the key explanatory variable.

The Dependent Variable The dependent variable in the analysis was a dichotomous indicator of obesity, defined as a BMI greater than or equal to 30 kg/m^2. (National Heart Lung and Blood Institute 1998) BMI was calculated using respondents' self-reported height and weight. Hamermesh finds that self-reported BMI in the ATUS are reasonably valid in a comparison to data from the National Health and Nutrition Examination Survey (Hamermesh 2010).

Covariates I incorporated several types of covariates into the analysis, including information on demographics and socio-economic status of respondents. Demographic data came from the core ATUS files and included race/ethnicity (non-Hispanic black, Hispanic and Other), age group (25-29, 30-39 and 40-49) and marital status (never married, married, divorced/separated or widowed).

Socio-economic variables were drawn from both the Eating and Health module and the ATUS-CPS data. These variables included educational attainment (high school or less, some or all college, graduate education), poverty status (above/below the poverty threshold) and employment status (unemployed/out of the labor force, part-time, full-time). Poverty status was calculated based on whether a respondent's family income was below 180% of the national poverty threshold for a family of a given size in a particular year.

Family Dinner The key explanatory variable in the analysis was a dichotomous indicator of whether the respondent participated in a family dinner on the diary day ("family dinner"). I assessed family dinner status using information on the number of minutes spent eating in addition to when, where and with whom eating episodes occurred.

In order to be classified as a family dinner, an eating episode had to meet the following four criteria. First, the event must have been reported as an instance of primary eating[8] that started between the hours of 4:30 PM and 10:00 PM. In ATUS, the designation "primary" reflects respondents' perceptions regarding whether the activity in question was the main activity being carried out in a given interval of time. "Primary" implies a degree of importance associated with the activity in question. This contrasts with "secondary" eating episodes that are carried out simultaneously with other activities (e.g. driving, working, etc.) and are often not the main focus of attention.

[8] ATUS activity code 110101

Second, the eating episode must have occurred in the household. This criterion was imposed for consistency with the literature, as the majority of prior studies on the family dinner refer to meals carried out at home (Fiese and Schwartz 2008). The third criterion was that at least one other member of the family besides the respondent was present at the eating occasion. Meals conducted with non-family members, such as friends, acquaintances, co-workers and neighbors were not classified as a family dinner.

The fourth and final criterion used to define the family dinner was that the eating episode must have lasted for at least 15 minutes.[9] The purpose of specifying a minimum meal length was to distinguish a formal meal from snacking and grazing. Choice of this particular threshold was motivated by the empirical distribution of eating times in the current study, which indicated that eating episodes less than 15 minutes in duration were rare and thus unlikely to represent family dinners.[10] The appropriateness of this threshold is also supported by a prior study of American adolescents which indicated that only 5% of family dinners lasted less than 15 minutes whereas 27% lasted between 15-20 minutes (The National Center on Addition and Substance Abuse at Columbia University 2011). As the adopted threshold of 15 minutes may misclassify some individuals who had a family dinner that lasted for a shorter duration, I carried out preliminary analyses in which I altered the threshold to 5 minutes and to 10

[9] In cases where respondents reported multiple eating episodes during the dinner hour, I used the one with the longest duration for purposes of classification.

[10] Considering eating episodes that met the first three criteria above (primary eating episodes in the interval 4:30-10 PM that occurred at home in the presence of at least one family member), 1% lasted for less than 10 minutes, 4% lasted between 10 and 15 minutes, 10% lasted between 15 and 20 minutes and 85% lasted 20 minutes or more.

minutes. Findings from these alternative analyses were highly consistent with those reported in the baseline analysis. Additionally, I investigated the consequences of specifying a stricter threshold of 20 minutes. Results from this analysis showed consistent, albeit attenuated associations between the family dinner and obesity. The weaker associations exhibited in this sensitivity analysis are not surprising given that it involves re-distributing a substantial number of respondents to the "no family dinner" group (624 cases), likely increasing misclassification in the family dinner variable.

3.3.3 Analytic Approach

I used multivariate logistic regression to examine associations between family dinner and obesity, introducing covariates into the analysis sequentially, proceeding from no adjustment to partial adjustment and finally full adjustment for demographic and socio-economic covariates. I also examined effect modification of the association between family dinner and obesity by stratifying the analyses by poverty status and the number of children less than 18 living in the household (using the categories 0-2 vs. 3 or more children). These additional analyses were motivated by the possibility that the nature of the family dinner may vary based on the resources families have available to them as well as household structure. I evaluated whether the effect modification was significant through models that interacted family dinner with each of poverty status and number of household children.

I carried out numerous sensitivity analyses to examine the robustness of the findings to alternative specifications. First, I tested an alternative version of the key

explanatory variable with the following three categories: no dinner, dinner alone, family dinner. This was carried out in order to check for heterogeneity in the odds of being obese between the first two categories, as these were combined in the primary analysis. Second, I replicated all analyses adjusting for family income using the categories less than $30,000, $30,000-$49,999, $50,000-$74,999 and $75,000 and above. Prior to doing so, I imputed missing values on family income using multiple imputation with Amelia II software (Honaker, King, and Blackwell 2012; King et al. 2001) All model covariates were included in the imputation model. I did not adjust for family income in the primary analysis due to the substantial number of missing observations. Furthermore, prior work on the ATUS indicates that the family income variable is subject to item-specific non-response (e.g. the data are not missing at random), such that multiple imputation may be biased. Third, analyses were replicated using BMI specified as a continuous variable. In this sensitivity analysis, the dependent variable was defined as units of BMI above 25 kg/m^2 and BMI values between 18.5 and 25 kg/m^2 were assigned a value of zero.

All analyses make use of the sample weights provided in the Eating and Health Module of the ATUS. These sample weights adjust for unequal probabilities of selection, oversampling of weekend diary days and non-response both to the ATUS and the Eating and Health Module. Analyses are carried out using Stata 12 (StataCorp, Texas, USA).

3.4 Results

3.4.1 Descriptive Statistics

Table 3.1 shows the distribution of covariates by family dinner status for US adults ages 25-49. Sample sizes are reported as well as percentage distributions adjusting for sample

weights. Statistical significance was evaluating using two-tailed t-tests for differences in means and chi-squared tests for differences in distributions of categorical variables. The sample contains 6,214 respondents who participated in a family dinner the evening prior to the survey and 6,453 who did not. The table reveals a significantly higher concentration of young people, Hispanics and non-Hispanic blacks among the subpopulation that did not have a family dinner. Differences were particularly stark for the non-Hispanic black population, which made up 15.0% of the of the "no family dinner" group compared to 7.6% of the "family dinner" group.

[TABLE 3.1 HERE]

Members of the "no family dinner" group were also significantly less well educated and significantly more likely to live below the poverty line than respondents who participated in a family dinner. With respect to education, 43.7% of respondents in the "no family dinner" group reported having a high school degree or less, compared to 37.3% of respondents that participated in a family dinner. With respect to poverty status, 29.9% of respondents in the "no family dinner" group reported being below the poverty line compared to 26.8% of the "family dinner" group. Employment status, marital status and number of own household children less than 18 years of age were also unevenly distributed across the two groups, with members of the "no family dinner" group significantly less likely to be unemployed, married and living with children less than 18 years of age.

3.4.2 Analysis

Table 3.2 examines the association between "family dinner" participation and obesity (BMI>=30 kg/m^2). Associations were examined by sequential adjustment in a series of logistic regression models, first with no adjustments (Model 1), then partial adjustment for demographic information (Model 2) and finally, a fully adjusted model that included multiple measures of socio-economic status (Model 3). The bivariate association (expressed as odds ratios (OR)) between family dinner participation and obesity in Model 1 of Table 3.2 was 0.82 (95% CI 0.73-0.91; $p<0.001$), equivalent to an 18% reduction in the odds of being obese. With additional adjustments in Model 2 for gender, age and race/ethnicity, the association between family dinner and obesity was reduced to 0.85 (95% 0.76-0.95; $p<0.01$). Adjustment for marital status and socio-economic information in Model 3, including education, poverty and employment attenuated the association further (OR=0.87, 95% CI 0.78-0.97), however the association remained significant ($p<0.05$). Thus, even after adjustment for multiple indicators of SES, family dinner participation was associated with 13% reduction in the odds of being obese.

[TABLE 3.2 HERE]

Table 3.3 presents results on effect modification of the association between family dinner and obesity by socio-economic status (SES). This analysis was motivated by the fact that low-SES households may be constrained in their ability to prepare healthy family dinners due to limited food budgets and lack of grocery stores in their neighborhoods. As such, the health benefits of the family dinner may not extend to low-SES households to a similar extent. Poverty status, elicited in the ATUS Eating and Health interview through a

simple question about whether the respondent's family income fell above or below a certain threshold, was used to explore SES differences.[11]

[TABLE 3.3 HERE]

The associations shown in Table 3.3 were obtained in a fully-adjusted logistic regression analysis that stratified by poverty status. A significance test of the difference in associations by poverty status was performed by interacting family dinner with poverty status. Consistent with my hypothesis, the association was stronger for those above vs. those below the poverty line; however, the difference in odds ratios was not significant. For those above the poverty line, the OR on family dinner was 0.86 (0.75-0.98; $p<0.05$), corresponding to a 14% decrease in the probability of being obese (Model 1). For those living below the poverty line, the OR was 0.91 (95% CI 0.75-1.10).

Table 3.4 explores effect modification of the association between family dinner and obesity by the number of children less than 18 years of age in the household (using categories 0-2 vs. 3 or more children). Significance was assessed in the same manner as above. As hypothesized, the association was weaker for individuals with larger vs. small families, although the difference in odds ratios was not statistically significant. For individuals with 0-2 children, the OR on family dinner was 0.84 (95% CI 0.75-0.95; $p<0.01$) indicating that family dinner participation was associated with a 16% decline in obesity risk (Model 1). For individuals with 3 or more children, the odds ratio was 0.98 and not significant (Model 2).

[TABLE 3.4 HERE]

[11] The survey question reads: "Last month, was your total household income before taxes more or less than (amount) per month?" The amount stated by the surveyor was determined based on the number of people in the household as well as the year of the survey and approximates the 185 percent of the poverty threshold (Bureau of Labor Statistics 2013b)

3.4.3 Sensitivity Analyses

Family dinner was specified as a binary variable in the baseline analysis. In a sensitivity analysis, I tested a categorical variable that separated individuals that didn't have a family dinner into two separate groups: those who didn't eat dinner and those who ate alone. This was motivated by the possibility of heterogeneity in obesity risk among these two groups. Among the 6,453 respondents that didn't have a family dinner, 5,960 or 92% of individuals had no dinner compared to 493 or 8% that ate alone. The results appear in Table A.3.1: the OR for dinner alone was 0.97 and not significant (relative to the "no dinner" group). The OR for family dinner was 0.87 ($p<0.05$), equivalent to the value in the baseline analysis.

A second sensitivity analysis assessed whether the association between family dinner and obesity was robust to adjustment for family income. This variable (specified as a categorical variable with four categories) was substituted for poverty status in a model adjusting for the complete set of covariates used in the baseline analysis. The results, which appear in Table A.3.2, show that the association attenuated slightly but that family dinner participation remained associated with a 12% reduction in the odds of being obese ($p<0.05$).

In the third sensitivity analysis, all results were replicated using a continuous version of the dependent variable (Tables A.3.3-5). Results in these tables reflect associations across the whole range of BMIs in the sample rather than simply a respondent's location above or below the obesity threshold. Consistent with the baseline analysis, Table A.3.3 shows that the association between family dinner and BMI weakens upon introducing additional covariates; however, it remains significant in Model 3 after

fully adjusting for covariates. In that model, family dinner participation was associated with a 0.83 unit reduction in BMI (95% CI -1.53,-0.14; $p<0.05$). In Tables A.3.4 and A.3.5, analyses using continuous BMI as the dependent variable were stratified by poverty status and the number of household children under the age of 18. The associations in these two tables were consistent with findings from the baseline analysis, with associations stronger among those above the poverty line and those with fewer household children. As in the baseline analysis, the differences were not statistically significant. Overall, the results from continuously specified BMI provide strong support for the findings from the baseline analysis that used a binary indicator of obesity status.

3.5 Discussion

A recent and growing literature examines the role of eating patterns in dietary quality and obesity (Hammons and Fiese 2011). This research has evolved in the context of dramatic changes in the way people eat in the United States (Zick and Stevens 2010; Popkin and Duffey 2010). Various eating patterns have received emphasis, including breakfast skipping, snacking and secondary eating.

The current analysis used time-use data to explore the association between family dinner participation and obesity among adults aged 25-49 in the United States. Using multivariate logistic regression, I found that family dinner participation was associated with a 13% reduction in the odds of being obese. This finding was robust to multiple controls for SES, including poverty, educational attainment and employment status. Furthermore, findings were consistent across several sensitivity analyses, including in an analysis that adjusted for family income, analyses in which BMI was substituted for

obesity status as the dependent variable and a model in which the key explanatory variable was defined as a categorical rather than binary variable.

These findings are consistent with prior studies examining associations between the family dinner and obesity (Hammons and Fiese 2011). However, the prior studies focused exclusively on children and adolescents and therefore results are not directly comparable. One paper focused on time-use patterns in relation to obesity more generally (Zick et al. 2011). In this analysis, time spent in primary eating throughout the 24-hr period prior to survey exhibited a significant negative association with obesity. This result is broadly consistent with my findings, as individuals that reported more time spent in primary eating throughout the day were more likely to have participated in a family dinner than individuals spending less time in primary eating.

I found differences in the association between family dinner and obesity depending on poverty status and number of household children. Consistent with my hypotheses, associations were found to be stronger among individuals in high- vs. low-SES households and stronger in individuals with smaller vs. larger families. Although suggestive, these differences were not statistically significant. Future research should examine these potential effect modifiers in larger datasets with increased power to detect differences in associations. If SES is found to be a significant effect modifier, with associations diminished among low-SES households, it might suggest that policy efforts should focus on food access issues for low-income households rather than (or in addition to) encouraging them to eat more family meals.

The present study contributes to the literature in several key respects. First, extraordinary detail in the time-use data allows for a more meaningful definition of the

family dinner. The definition in this study incorporates information on when, where and with whom the meal occurred as well as how long the meal lasted and whether it was the primary activity being carried out. Prior work in this area makes universal use of simple survey questions for defining participation in the family meal.[12] The survey question identifies whether a family meal occurred, but doesn't provide information on other characteristics such as the duration of the meal, whether it occurred in or outside the household and whether it was the primary activity being carried out. Second, integration of the ATUS with CPS data permitted extensive adjustment for socio-economic confounding. A third strength of the current study is that it was based on a nationally representative sample. Thus, the findings are broadly representative of US adults ages 25-49 with families.

This study also has some limitations. First, inferences regarding the effects of the family dinner on obesity cannot be interpreted as causal due to the observational design and the possibility of confounding. However, linkage to CPS did allow extensive adjustment for socio-economic factors, including family income, poverty, education and employment status. Another issue with the observational design is the possibility of reverse causality. It may not be family dinner participation that is driving obesity, but rather obesity that is driving lack of family dinner participation. Third, I considered meals initiated between 4:30-10:00 PM as potential dinners. Although this window is quite broad, it may still leave out some respondents whose main meal is at mid-day or whose dinner hours are irregular due to non-standard work hours. This may have introduced some misclassification bias. A fourth limitation is the low response rate of

[12] Family dinner participation is typically judged based on a simple survey question that asks about "the number of times all or part of the family gathered for dinner in the last week."

ATUS (below 60%). However, a validity study found that busy people were not less likely to respond to ATUS than less busy people (Abraham, Maitland, and Bianchi 2006). The non-response appears to be explained by failure to contact people that are less well integrated into their communities.

The empirical analysis in this paper focused on one particular eating behavior as it relates to health—the family dinner. Future research might focus on other aspects of eating and mealtime behavior apart from the family to dinner to identify the set of behaviors most strongly associated with health. Some examples might include the incidence of snacking, away-from-home eating and secondary eating and drinking. Another promising avenue for future research would be to employ multivariate methods (such as principle components analysis) to identify distinct clusters of eating patterns (e.g. formal vs. informal) that could then be related to various health outcomes. Finally, future studies should explore the mechanisms underlying the association between the family dinner and obesity, including dietary quantity and quality and pacing.

Many studies investigate the determinants of obesity, but a limited number are focused on social and cultural factors. However, as eating is deeply rooted in the social and cultural fabric of life, examining obesity from these vantage points is critical to gaining a complete understanding of the obesity epidemic. The contribution of this paper is to offer a sociological explanation for the rise of obesity in the United States. Although the focus here was on the family dinner, the paper has broader implications. The dramatic changes that have occurred in social norms surrounding eating behaviors may be an underappreciated aspect of the rise of obesity in the US in the late twentieth century. Formal codes that in the past dictated when and under what circumstances food could be

consumed have gradually diminished and informal eating behaviors—eating alone, eating out and secondary eating—have become more socially acceptable. Future work should continue to flesh out the consequences of these changes.

Table 3.1 Distribution of Social and Demographic Covariates by Family Dinner Status, US Adults 25-49

	Family Dinner N=(6,214)		No Family Dinner N=(6,453)		p-value
	N	(%)	N	(%)	
Male	2,762	(49.9)	2,960	(52.1)	0.068
Age Group					<0.001
25-29	712	(14.6)	934	(20.4)	
30-39	2,745	(41.1)	2,684	(36.1)	
40-49	2,757	(44.3)	2,835	(43.5)	
Race/ethnicity					<0.001
Other	4,939	(76.2)	4,509	(67.5)	
Non-Hispanic Black	446	(7.6)	863	(15.0)	
Hispanic	829	(16.1)	1,081	(17.5)	
Household Children < 18					<0.001
0	708	(25.0)	1,231	(36.6)	
1	1,729	(23.7)	1,964	(23.4)	
2	2,434	(31.8)	2,097	(24.6)	
3+	1,343	(19.4)	1,161	(15.4)	
Education Level					<0.001
High School or Less	1,898	(37.3)	2,321	(43.7)	
Some or All College	3,415	(49.8)	3,464	(47.6)	
Graduate Education	901	(12.9)	668	(8.7)	
Below Poverty Line	1,592	(26.8)	1,909	(29.9)	0.005
Employment Status					<0.001
Unemployed/Out of Labor Force	1,172	(18.3)	958	(13.7)	
Employed Part Time	771	(11.5)	803	(12.0)	
Employed Full Time	4,271	(70.1)	4,692	(74.3)	
Marital Status					<0.001
Never Married	544	(11.4)	921	(19.8)	
Married	5,009	(80.4)	4,430	(66.5)	
Widowed, Divorced or Separated	661	(8.2)	1,102	(13.7)	
Obese	1,655	(27.5)	1951	(31.8)	<0.001

Family dinner is defined as a primary eating episode in the interval 4:30-10 PM of at least 15 minutes in duration that occurred at home with at least one other family member present. Respondents are classified as below the poverty line if their income is less than

180% of the federal poverty threshold in a given year. 'Obese' is defined as a body mass index equal to or exceeding 30 kg/m^2 (based on respondents' self-reported height and weight). The sample includes people ages 25-49 who co-reside with at least one family member. Percentages are adjusted using sample weights. Statistical significance was evaluating using two-tailed t-tests for differences in means and chi-squared tests for differences in distributions of categorical variables. Source: American Time-Use Survey, 2006-2008.

Table 3.2 Odds Ratios and 95% CI from Logistic Regression Models Relating Family Dinner to Obesity, US Adults 25-49

	Model 1			Model 2			Model 3		
	Odds Ratio		95% CI	Odds Ratio		95% CI	Odds Ratio		95% CI
Family Dinner	0.82	***	(0.73-0.91)	0.85	**	(0.76-0.95)	0.87	*	(0.78-0.97)
Sex									
Women				1.00			1.00		
Men				1.28	***	(1.15-1.42)	1.27	***	(1.13-1.42)
Age									
25-29				1.00			1.00		
30-39				1.16		(0.97-1.37)	1.22	*	(1.02-1.45)
40-49				1.23	*	(1.04-1.46)	1.29	**	(1.08-1.54)
Race/ethnicity									
Other				1.00			1.00		
Non-Hispanic Black				1.73	***	(1.47-2.03)	1.55	***	(1.30-1.83)
Hispanic				1.45	***	(1.26-1.68)	1.22	**	(1.05-1.42)
Education Level									
High School or Less							1.00		
Some or All College							0.87	*	(0.77-0.99)
Graduate Education							0.50	***	(0.41-0.62)
Poverty Status									
Above Poverty Line							1.00		
Below Poverty Line							1.27	***	(1.11-1.45)
Employment Status									
Unemployed/Out of Labor Force							1.00		
Employed Part Time							0.98		(0.79-1.21)
Employed Full Time							1.04		(0.89-1.21)
Marital Status									
Never Married							1.00		
Married							0.95		(0.80-1.15)
Widowed, Divorced or Separated							0.94		(0.75-1.18)

CI: confidence interval. See Table 3.1 for variable definitions and sample inclusion criteria. Estimates incorporate sample weights. Source: American Time-Use Survey, 2006-2008. ***p<0.001; **<0.01; *p<0.05

Table 3.3 Odds Ratios and 95% CI from Logistic Regression Models Relating Family Dinner to Obesity by Poverty Status, US Adults 25-49

	Model 1 (Above Poverty Line)			Model 2 (Below Poverty Line)		
	Odds Ratio		95% CI	Odds Ratio		95% CI
Family Dinner	0.86	*	(0.75-0.98)	0.91		(0.75-1.10)
Sex						
Women	1.00			1.00		
Men	1.38	***	(1.20-1.60)	1.03		(0.84-1.26)
Age						
25-29	1.00			1.00		
30-39	1.26	*	(1.00-1.59)	1.20		(0.92-1.58)
40-49	1.49	***	(1.19-1.88)	1.01		(0.76-1.35)
Race/ethnicity						
Other	1.00			1.00		
Non-Hispanic Black	1.61	***	(1.30-2.00)	1.44	**	(1.10-1.89)
Hispanic	1.36	**	(1.10-1.68)	1.11		(0.89-1.39)
Education Level						
High School or Less	1.00			1.00		
Some or All College	0.82	**	(0.70-0.95)	0.98		(0.80-1.18)
Graduate Education	0.46	***	(0.36-0.57)	1.04		(0.52-2.07)
Employment Status						
Unemployed/Out of Labor Force	1.00			1.00		
Employed Part Time	0.88		(0.65-1.19)	1.13		(0.84-1.52)
Employed Full Time	1.05		(0.85-1.29)	1.01		(0.81-1.26)
Marital Status						
Never Married	1.00			1.00		
Married	0.83		(0.65-1.05)	1.20		(0.92-1.56)
Widowed, Divorced or Separated	0.78		(0.57-1.05)	1.14		(0.82-1.57)

CI: confidence interval. See Table 3.1 for variable definitions and sample inclusion criteria. Estimates incorporate sample weights. Source: American Time-Use Survey, 2006-2008. ***p<0.001; **<0.01; *p<0.05

Table 3.4 Odds Ratios and 95% CI from Logistic Regression Models Relating Family Dinner to Obesity by Number of Children < 18 in the Household, US Adults 25-49

	Model 1 (0-2 Children)			Model 2 (3-5 Children)		
	Odds Ratio		95% CI	Odds Ratio		95% CI
Family Dinner	0.84	**	(0.75-0.95)	0.98		(0.78-1.22)
Sex						
Women	1.00			1.00		
Men	1.23	**	(1.09-1.40)	1.49	**	(1.12-1.98)
Age						
25-29	1.00			1.00		
30-39	1.21		(1.00-1.46)	1.43		(0.95-2.15)
40-49	1.33	**	(1.10-1.61)	1.24		(0.80-1.93)
Race/ethnicity						
Other	1.00			1.00		
Non-Hispanic Black	1.51	***	(1.25-1.82)	1.74	**	(1.19-2.56)
Hispanic	1.26	**	(1.06-1.50)	1.00		(0.74-1.35)
Education Level						
High School or Less	1.00			1.00		
Some or All College	0.85	*	(0.74-0.97)	0.98		(0.75-1.29)
Graduate Education	0.48	***	(0.38-0.61)	0.61	*	(0.38-0.98)
Poverty Status						
Above Poverty Line	1.00			1.00		
Below Poverty Line	1.19	*	(1.02-1.38)	1.56	**	(1.19-2.06)
Employment Status						
Unemployed/Out of Labor Force	1.00			1.00		
Employed Part Time	1.02		(0.80-1.30)	0.80		(0.54-1.20)
Employed Full Time	1.04		(0.87-1.24)	0.97		(0.72-1.31)
Marital Status						
Never Married	1.00			1.00		
Married	0.94		(0.78-1.15)	0.92		(0.54-1.55)
Widowed, Divorced or Separated	0.92		(0.72-1.17)	0.94		(0.52-1.71)

HH: household; CI: confidence interval. See Table 3.1 for variable definitions and sample inclusion criteria. Estimates incorporate sample weights. Source: American Time-Use Survey, 2006-2008. ***p<0.001; **<0.01; *p<0.05

Table A.3.1 Odds Ratios and 95% CI from Logistic Regression Models Relating Dinner Alone and Family Dinner to Obesity, US Adults 25-49

	Odds Ratio		95% CI
Dinner Status			
No Dinner	1.00		
Dinner Alone	0.97		(0.74-1.28)
Family Dinner	0.87	*	(0.78-0.97)
Sex			
Women	1.00		
Men	1.27	***	(1.13-1.42)
Age			
25-29	1.00		
30-39	1.22	*	(1.02-1.45)
40-49	1.29	**	(1.08-1.55)
Race/ethnicity			
Other	1.00		
Non-Hispanic Black	1.55	***	(1.31-1.83)
Hispanic	1.22	**	(1.05-1.42)
Education Level			
High School or Less	1.00		
Some or All College	0.87	*	(0.77-0.99)
Graduate Education	0.50	***	(0.41-0.62)
Poverty Status			
Above Poverty Line	1.00		
Below Poverty Line	1.27	***	(1.11-1.44)
Employment Status			
Unemployed/Out of Labor Force	1.00		
Employed Part Time	0.98		(0.79-1.20)
Employed Full Time	1.04		(0.89-1.21)
Marital Status			
Never Married	1.00		
Married	0.95		(0.79-1.15)
Widowed, Divorced or Separated	0.94		(0.75-1.18)

CI: confidence interval. See Table 3.1 for variable definitions and sample inclusion criteria. “Family Dinner” is defined as in the baseline analysis, as a primary eating episode attended by at least one other member of the family that occurred at home during

the hours 4:30-10 PM and was at least 15 minutes in duration. “Dinner Alone” is defined as above with the exception that the meal occurred alone. Estimates incorporate sample weights. Source: American Time-Use Survey, 2006-2008. ***p<0.001; **<0.01; *p<0.05

Table A.3.2 Odds Ratios and 95% CI from Logistic Regression Model Relating Family Dinner to Obesity with Adjustment for Family Income, US Adults 25-49

	Odds Ratio		95% CI
Family Dinner	0.88	*	(0.79-0.98)
Sex			
Women	1.00		
Men	1.27	***	(1.13-1.42)
Age			
25-29	1.00		
30-39	1.25	*	(1.05-1.49)
40-49	1.34	**	(1.12-1.60)
Race/ethnicity			
Other	1.00		
Non-Hispanic Black	1.51	***	(1.27-1.78)
Hispanic	1.20	*	(1.03-1.40)
Education Level			
High School or Less	1.00		
Some or All College	0.92		(0.81-1.04)
Graduate Education	0.56	***	(0.45-0.69)
Family Income (Annual, $)			
Less than 30,000	1.00		
30,000 to 49,999	0.85		(0.71-1.01)
50,000 to 74,999	0.75	**	(0.62-0.90)
75,000 or More	0.60	***	(0.49-0.72)
Employment Status			
Unemployed/Out of Labor Force	1.00		
Employed Part Time	0.99		(0.80-1.22)
Employed Full Time	1.06		(0.91-1.23)
Marital Status			
Never Married	1.00		
Married	1.00		(0.83-1.20)
Widowed, Divorced or Separated	0.91		(0.72-1.13)

CI: confidence interval. See Table 3.1 for variable definitions and sample inclusion criteria. Missing values on family income (n=1,356) were imputed using multiple imputation with Amelia II software. Estimates incorporate sample weights. Source: American Time-Use Survey, 2006-2008. ***p<0.001; **<0.01; *p<0.05

Table A.3.3 Coefficients and 95% CI from OLS Regression Models Relating Family Dinner to BMI in US Adults 25-49

	Model 1		Model 2		Model 3	
	Coefficient	95% CI	Coefficient	95% CI	Coefficient	95% CI
Family Dinner	-1.56 ** *	(-2.26--0.86)	-1.13 **	(-1.82--0.44)	-0.83 *	(-1.53--0.14)
Sex						
Women (ref.)						
Men			5.19 ** *	(4.50-5.88)	4.75 ** *	(3.99-5.50)
Age						
25-29 (ref.)						
30-39			1.42 *	(0.31-2.52)	1.75 **	(0.65-2.85)
40-49			2.67 ** *	(1.57-3.77)	2.91 ** *	(1.80-4.02)
Race/ethnicity						
Other (ref.)						
Non-Hispanic Black			4.93 ** *	(3.83-6.02)	3.90 ** *	(2.75-5.05)
Hispanic			3.13 ** *	(2.22-4.05)	1.55 **	(0.58-2.53)
Education Level						
High School or Less (ref.)						
Some or All College					-1.36 ** *	(-2.14--0.57)
Graduate Education					-5.77 ** *	(-6.96--4.58)
Poverty Status						
Above Poverty Line (ref.)						
Below Poverty Line					2.22 ** *	(1.36-3.09)
Employment Status						
Unemployed/Out of Labor Force (ref.)						
Employed Part Time					-0.39	(-1.80-1.02)
Employed Full Time					1.45 **	(0.38-2.53)
Marital Status						
Never Married (ref.)						
Married					-0.01	(0.00-1.23)
Widowed, Divorced or Separated					0.32	(0.00-1.81)

CI: confidence interval; BMI: body mass index; ref: reference category. See Table 3.1 for variable definitions and sample inclusion criteria. The dependent variable, BMI, is units of BMI above 25 kg/m^2. BMI values between 18.5 and 25 kg/m^2 are assigned a value of

zero. BMI was calculated based on respondents' self-reported height and weight. Estimates incorporate sample weights. Source: American Time-Use Survey, 2006-2008. ***p<0.001; **<0.01; *p<0.05

Table A.3.4 Coefficients and 95% CI from OLS Regression Models Relating Family Dinner to BMI by Poverty Status, US Adults 25-49

	Model 1 (Above Poverty Line)			Model 2 (Below Poverty Line)		
	Coefficient		95% CI	Coefficient		95% CI
Family Dinner	-0.98	*	(-1.79--0.16)	-0.33		(-1.64-0.97)
Sex						
Women (ref.)						
Men	6.16	***	(5.26-7.06)	1.12		(-0.26-2.50)
Age						
25-29 (ref.)						
30-39	1.02		(-0.36-2.41)	3.28	***	(1.48-5.08)
40-49	3.16	***	(1.81-4.52)	2.09	*	(0.16-4.02)
Race/ethnicity						
Other (ref.)						
Non-Hispanic Black	4.18	***	(2.69-5.67)	3.39	***	(1.59-5.19)
Hispanic	1.84	**	(0.52-3.15)	1.45		(-0.06-2.96)
Education Level						
High School or Less (ref.)						
Some or All College	-1.60	**	(-2.56--0.65)	-0.99		(-2.31-0.34)
Graduate Education	-5.97	***	(-7.26--4.69)	-3.39		(-7.64-0.86)
Employment Status						
Unemployed/Out of Labor Force (ref.)						
Employed Part Time	-0.62		(-2.46-1.22)	0.36		(-1.75-2.47)
Employed Full Time	1.32		(-0.08-2.72)	1.84	*	(0.21-3.47)
Marital Status						
Never Married (ref.)						
Married	-0.29		(-1.89-1.31)	0.87		(-1.01-2.75)
Widowed, Divorced or Separated	-0.26		(-2.24-1.72)	0.86		(-1.35-3.08)

CI: confidence interval; BMI: body mass index; ref: reference category. See Table 3.1 for variable definitions and sample inclusion criteria. The dependent variable, BMI, is units of BMI above 25 kg/m^2. BMI values between 18.5 and 25 kg/m^2 are assigned a value of zero. BMI was calculated based on respondents' self-reported height and weight. Estimates incorporate sample weights. Source: American Time-Use Survey, 2006-2008.

***p<0.001; **<0.01; *p<0.05

Table A.3.5 Coefficients and 95% CI from OLS Regression Models Relating Family Dinner to BMI by Number of Children < 18 in the Household, US Adults 25-49

	Model 1 (0-2 Children)			Model 2 (3-5 Children)		
	Coefficient		95% CI	Coefficient		95% CI
Family Dinner	-1.08	**	(-1.87--0.29)	0.09		(-1.27-1.45)
Sex						
Women (ref.)						
Men	4.60	***	(3.77-5.44)	6.00	***	(4.29-7.71)
Age						
25-29 (ref.)						
30-39	1.75	**	(0.51-2.98)	1.83		(-0.53-4.19)
40-49	3.18	***	(1.96-4.41)	1.59		(-0.92-4.09)
Race/ethnicity						
Other (ref.)						
Non-Hispanic Black	3.69	***	(2.39-4.99)	4.39	***	(1.92-6.86)
Hispanic	1.68	**	(0.55-2.80)	0.76		(-1.11-2.62)
Education Level						
High School or Less (ref.)						
Some or All College	-1.45	**	(-2.34--0.57)	-1.04		(-2.63-0.54)
Graduate Education	-6.12	***	(-7.46--4.78)	-3.91	**	(-6.37--1.45)
Poverty Status						
Above Poverty Line (ref.)						
Below Poverty Line	1.53	**	(0.49-2.56)	4.26	***	(2.63-5.89)
Employment Status						
Unemployed/Out of Labor Force (ref.)						
Employed Part Time	-0.27		(-1.95-1.41)	-0.62		(-2.96-1.72)
Employed Full Time	1.42	*	(0.15-2.69)	1.15		(-0.81-3.11)
Marital Status						
Never Married (ref.)						
Married	-0.01		(-1.34-1.32)	-1.27		(-4.45-1.91)
Widowed, Divorced or Separated	0.35		(-1.27-1.97)	-0.96		(-4.51-2.59)

CI: confidence interval; BMI: body mass index; HH: household; ref: reference category. See Table 3.1 for variable definitions and sample inclusion criteria. The dependent variable, BMI, is units of BMI above 25 kg/m^2. BMI values between 18.5 and 25 kg/m^2 are assigned a value of zero. BMI was calculated based on respondents' self-reported

height and weight. Estimates incorporate sample weights. Source: American Time-Use Survey, 2006-2008. ***p<0.001; **<0.01; *p<0.05

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