Center for Demography and Ecology

University of Wisconsin-Madison

Recent US Trends in Body Weight and Mortality: Using Weight at Age 25

Yan Yu

CDE Working Paper No. 2008-17

Recent US Trends in Body Weight and Mortality: Using Weight at Age 25[*]

Yan Yu

---

[*] Center for Demography and Ecology, University of Wisconsin-Madison. Email: yyu@ssc.wisc.edu

Abstract

The expanding waistlines of the American population have stirred immense research and public interest in how secular changes in body weight affect population health. Big controversies and gaps remain in our understanding about the weight-mortality relationship. This study uses recalled weight at age 25 to classify weight status, and document how mortality differentials changed over time in the US female population. Analyzing data from the National Health and Nutrition Examination Surveys, we find that, in contrast with previous findings based on weight concurrent with the survey baseline, mortality does not differ between the age-25 underweight and normal-weight, and is equally elevated for the age-25 overweight and obese women. Between 1976 and 2004, both *relative* and *absolute* mortality differentials widened. Mortality reversals were observed for the overweight/obese. In 1999-2004, life expectancy at age 25 for the overweight/obese stood at 50.4, as compared with 57.1 for the lean, and 52.6 in 1976-1980.

1 Background

People are living longer. Life expectancy at birth in the US has increased from about 40 years at the turn of the 20th century to 77 years at its end, with about half of the rise achieved during the last three decades of the 20th century. Despite the overall optimism, health experts and the public have been alerted to the looming public health storm of obesity. Excess body mass is associated with a host of fatal diseases such as cardiovascular diseases, hypertension and stroke, cancer, Type II diabetes, gallbladder diseases, and with less life-threatening but debilitating conditions such as osteoarthritis and pulmonary diseases.[1] Since the 1960s, the mean weight of American adults aged 20-74 has increased by 11 kilograms (24 pounds) and the mean Body Mass Index (BMI, calculated as weight in kilograms divided by squared height in meters) has increased by 2.7 and 3.3 units to 27.8 and 28.2 for adult men and women, respectively.[2] Today, about two of every three American adults have a BMI greater than 25 and are considered overweight or obese.[3] This rapid rise in body mass has been observed of all ethnic groups and socio-economic strata. Although the prevalence of overweight and obesity has been higher in more disadvantaged subpopulations, the pace of rise in body weight has been faster among the traditionally less vulnerable groups and the disparities have narrowed.[4]

What are the health implications of these trends in body weight? Olshansky and colleagues argued that obesity is responsible for a reduction of one third to three fourths of a year in life expectancy at birth in the United States in 2000, and that the amount of life shortened would increase in the coming years because “the obese who are now at younger ages carry their elevated risk of death into middle and older ages.”[5] They also suggested that if the current trend in body weight continues, the continuing rise in life expectancy would stop.[5]

These claims, however, are backed up by fragmentary and often conflicting empirical evidence, or even complete blanks in our knowledge. There is little empirical research about whether excess mortality due to obesity has increased over time. Obese mortality relative to the normal-weight declined over the first three National Health and Nutrition

Examination Surveys (NHANES), a finding that has been used to suggest that advances in medical care have reduced the health disparities related to excess weight.[6] However, the surveys overlap in mortality follow-up (through 2000, with respective baselines in 1971-75, 1976-80 and 1988-94). Survey differences are therefore not strictly changes over time.

There has been no research about whether overweight or obese individuals are surviving better than their predecessors. Life expectancy has been rising continuously. It remains an open question if the rising ride has lifted all boats. The prevalence of main cardiovascular disease risk factors such as high cholesterol level, high blood pressure and smoking has decreased since the early 1960s for all weight groups, with the greatest reductions having occurred among the overweight and obese.[7] Ironically, the same analysis found the greatest increase of diabetic prevalence among the heavier groups. Mortality trends within weight subpopulations remain unknown. Nor do we know if at the population level, the reduction of life due to excess weight has been increasing in the past.

In addition to these gaps, big controversies remain in the existent literature about the weight-mortality relationship. Evidence is well-documented for the physiological pathologies of excess fat mass such as hypertension, lipid abnormalities, or insulin resistance that are precursors to cardiovascular heart diseases, and a host of other life-threatening or debilitating chronic diseases.[1] No similar biologic mechanism has been found for lean mass.

The epidemiological studies about body weight and health have less consistent results. In the much-publicized studies using the NHANES data,[6,8] the findings that, compared with normal-weight (BMI between 18.5 and 25), mortality is lower for overweight (BMI between 25 and 30), not higher for moderate obesity (BMI between 30 and 35), but the highest for underweight (BMI less than 18.5) largely agree with the existent literature.[9,10] On the other hand, significant excess mortality for the moderately overweight or obese was detected in studies restricted to healthy non-smokers in the National Institute of Health-AARP sample of those aged 50-71,[11] or younger adults in the Framingham Heart Study for subjects aged 30-49.[12] Since a large majority of the US population is in the middle BMI range between 25 and 35, the conflicting results have led to a great deal of confusion and contention in public health.[13,14] Questions have arisen as to whether we should use current definitions of overweight and obesity to characterize a large segment of the population.[15] The public health concern over obesity has been labeled as alarmist or moral panic.[16]

Underlying these controversies are deep methodological issues.[17] It is an almost universal practice to characterize the population weight distribution using body weight concurrent with the study baseline. Body weight, however, is not a fixed characteristic, but changes over the life-course in connection with health. Weight change, loss in particular, may distort the weight-mortality relationship. In the Established Populations for Epidemiologic Studies of the Elderly with a sample of men and women aged 70+, the heaviest are the least likely to die, and the highest mortality is suffered by the leanest. However, mortality pattern reverses when recalled body weight at age 50 is used. The clue lies in weight loss: The heaviest at age 50 are most likely to have lost 10% of their weight by age 70+, and in their new leaner status, subsequently suffer the highest mortality.[18] Similar pattern reversals were found for cardiovascular diseases and risk factors in the Cardiovascular Health Study.[19] In the NHANES data, excess deaths due to concurrent obesity increase from 112,000 to 165,000 when the reference BMI narrows from 18.5-24.9 to 23-24.9.[6] It is conceivable that the lower end of the reference group (BMI 18.5-24.9) includes a hodgepodge of individuals who have had illness and lost weight, possibly related to earlier heavy weight or smoking, and those who have kept lean weight through a balance of energy intake and consumption.

There is no consensus how to handle illness-related heterogeneities among concurrent weight groups, and for good reasons. Commonly used approaches such as statistical adjustment and sample exclusion remain controversial.[20,21] Take smoking for example. As smokers are more likely to both die early and lose weight as a result of smoking-related manifest or occult diseases, deleting smokers in the sample should make the weight groups less heterogeneous. A simulation study, however, found that the changes in mortality ratios by body weight after excluding smokers are similar to the changes when excluding a numerically identical but randomly picked subset of the sample, and therefore concluded that smokers should not be excluded when analyzing the weight-mortality relationship.[22] Also debatable is whether the physiologic effects of excess weight such as hypertension and diabetes should be adjusted for.[17] Overweight elevates mortality when not controlling for hypertension, and does not when controlling.[23] Results could be different if the diseases are associated with weight loss, which is common among old adults. As body weight and illness are related in a dynamic way, and their inter-relationship varies across populations, how to measure and analyze them remains an analytical challenge.

2 Research Questions

To complement the prior focus on body weight concurrent with the study baseline, and enrich our understanding about mortality differentials and trends, this paper uses body weight at the fixed age of 25 to classify the US female population at three time points roughly spanning the past thirty years, and examine changes over time in mortality differentials by age-25 weight. Specifically, we first use survey data to estimate population distributions for age-25 weight, and mortality ratios by age-25 weight. Second, weight distributions and mortality ratios are combined with national mortality data to calculate three mortality indicators for each age-25 weight group: age-specific mortality rates, life expectancy at age 25 and partial life expectancy between age 25 and 65. Lastly, we calculate years of life shortened due to excess weight at age 25 and the change over time for this index, as done for concurrent weight for the year 2000.[5] The analysis focuses on trends for women, as has been done in other studies on mortality trends in low-mortality countries.[24] Male mortality improvement is yet to catch up, and smoking is still the dominant risk factor.

The strength of using weight status at age 25, as compared with concurrent weight, is that it is less susceptible to health-related weight change. At age 25, illness-related weight loss should be minimal. Using age-25 weight is an attempt to attack the issue of unobserved heterogeneities from an angle different from statistical adjustment or sample deletion that have been commonly used previously. Age-25 weight has additional research merit of its own. Despite the growing body of research on the rapidly expanding waistlines of the nation's children and young adults, little has been done on the long-term mortality effect of childhood obesity, and there has been no study about its time trend. Analyzing data for age-25 weight that have been collected for all adults in existing surveys would provide timely supplementary evidence.

The drawback is that for nationally representative samples, only retrospective self-reported data are available, in contrast to data for concurrent weight that is measured by health professionals, for example in the National Health and Nutrition Examination Surveys. As no previous study has used this measure to classify weight groups in cross-sectional populations, we conduct a series of analyses to check the quality of the retrospectively self-reported age-25 BMI distributions, making use of the well-established historical trend for body weight, and expected mortality selection with respect to age-25 weight. Sensitivity analysis is done to examine if changes over time in mortality differentials by weight are robust to poor data, especially among older age groups. We also compare the results, whenever possible, with findings from the Framingham Heart Study for weight measured at age 30-49,[12] and from the Nurses' Health Study for age-18 weight reported by women aged 24 to 44.[25] It should be noted that there has been a growing literature using retrospective reports of early life characteristics, e.g., overall childhood health status,[26] to examine how early life conditions affect adult health and mortality.

3 Data and Methods

The analysis uses three main data sources: 1) interview and health examination data from three National Health and Nutrition Examination Surveys (NHANES);[27–33] 2) mortality through 2000 for the first two NHANES samples;[34] and 3) US female total mortality data[35] for years that were spanned by the NHANES baselines. The survey data are used to estimate age-25 weight distributions and mortality ratios by age-25 weight. These two sets of quantities are combined with national mortality data to further derive mortality estimates (rates and life expectancies) that are specific for each age-25 weight subpopulation. Life expectancy shortened is calculated in the last step.

Conducted by the National Center for Health Statistics (NCHS), the NHANES consist of a series of cross-sectional samples that represent the non-institutionalized population in the US. Anthropometric and other health conditions data are collected at the baseline health examinations and interviews. This study uses three samples with respective baselines in 1976-80, 1988-94 and 1999-2004, and for convenience, refers to them as N2, N3 and N99. The interval years spanned by each baseline are shown as shaded areas in Figure 1. Baseline retrospective data about age-25 weight are used to estimate the population weight distribution, using the logit model.

The N2 and N3 baseline samples were followed up for mortality through 2000 via a linkage with the National Death Index (NDI). Set up as person-month records and divided into three consecutive time periods (1976-85, 1986-95 and 1996-2000), the mortality data are used to estimate mortality differentials by age-25 weight for each period, using a discrete version of the parametric Gompertz model.

As shown in Figure 1, there is an approximate one-to-one matching of years between the weight distributions and mortality differentials. Take the second time interval for example. The age-25 distribution is estimated from the 1988-94 N3 baseline, whereas mortality ratios, from the person-month records of N2 and N3 subjects surviving in 1986-1995. The analysis takes advantage of this correspondence and combines the two sets of quantities (weight distributions and mortality differentials by weight) to derive, for each of the three time periods, weight-specific mortality from national mortality data, which are obtained from the Human Mortality Database (HMD).

The rationale behind this derivation is that at each time point, population total mortality rate (from HMD) is a weighted average of weight-specific mortality rates, the weights being the population weight distribution (from the NHANES) and the weight-specific mortality rates being summarized by mortality differentials. Sample weights are used when analyzing the NHANES data to represent the US population. It is also assumed that the institutionalized populations, not targeted by the NHANES, have weight distributions and weight disparities in mortality that are similar to the NHANES estimates, as has been implicitly assumed in previous studies.[5,6]

4 Age-25 BMI Distribution

The NHANES measured the weight and height of respondents at the time of survey at a mobile examination center. In addition, the question about usual weight at age 25 was asked of respondents aged 26 and over in N2 and N3, or aged 27 and over in N99. For those aged 25 or 26, we use weight measured at the baseline to impute missing data.

Age-25 Body Mass Index (BMI), calculated as age-25 weight (in kilograms) divided by squared height measured at the baseline (in meters), is used to define two weight groups: lean (BMI < 25), and overweight/obese (BMI ≥ 25). The classification uses the cut-off points specified in the World Health Organization weight guidelines,[1] but collapses the underweight and normal-weight into the lean group because preliminary mortality analysis found no excess underweight mortality relative to the normal-weight. The final results combine the overweight and obese because in both the BMI distribution and mortality analysis, a model that combine the two groups is preferred to one treating the overweight and obese as separate.

The three baseline samples cover a different age range. N2 limits the sample to those aged 74 or younger. All age groups are covered in N3 and N99, but for confidential reasons, age is topcoded at 90 and 85 in the N3 and N99 public files, respectively. Cases aged 85 and above are deleted in these two analytical samples. At older ages, we use extrapolation to estimate population weight distributions, and mortality ratios by weight group (in the next section). After deleting 501 cases in N2, 752 cases in N3, and 319 cases in N99 missing for age-25 BMI, the three sample sizes are: 6865, 7825 and 6590.

Table 1 presents sample sizes (unweighted) and weight distributions (weighted) by five-year age groups for each of the three surveys. The weight distributions are also shown in the left panel of Figure 2. In consistency with the well-documented trend for current weight,[3] proportions of the population who were overweight/obese at age 25 increased across the surveys, notably between the last two surveys and over the younger age groups. About two-fifths of the age-25 population was overweight/obese in N99, nearly doubling that in N2 or N3. The distributional differences across surveys were negligible after age

60. One concern, however, is that at older ages, proportions lean declined. Both historical weight trends and expected mortality differentials by age-25 weight would expect these proportions to increase over age. The retrospective reports of older subjects could be less reliable.

Each survey is analyzed using a binomial logit model, with yearly age and age square as covariates:

$$\log \frac{P(W=2)}{P(W=1)} = \beta_0 + \beta_1 Age + \beta_2 Age^2, \tag{1}$$

where W=1 for the lean, and 2 for the overweight/obese.

The parameter estimates of the three logit models are shown in Table A-1 in the Appendices. As expected, the coefficient estimate is negative for age and positive for age square. At each cross-section, the log odds of overweight/obese decreases by age, but at a slower rate at older ages. Model-based proportions overweight/obese $P(W=2)$ and proportions lean $P(W=1)$ are shown in the right panel of Figure 2. When making estimates for the oldest five-year age groups, instead of completely following the model-based values, I use the first model-based proportion lean that shows a decrement from the preceding age and extrapolate it to succeeding ages. This is to alleviate concerns about the report of older cohorts, typically at older ages at the time of survey, and to preserve sensible historical trends and age patterns.

5 Mortality Ratios by Age-25 Weight

Of interest is how mortality rates differ by age-25 weight, and how these differentials (mortality ratios) change over time and age. The cross-sectional age groups are an important factor to consider as they correspond to birth cohorts that may differ in weight and health history. The quality of retrospective weight report may also differ by age/cohort, as mentioned above.

To estimate mortality ratios related to age-25 weight status, N2 and N3 mortality data through 2000 are remotely accessed through the NCHS Research Data Center.[34] Person-month records in the mortality follow-up are pooled to construct an analytical sample indexed by time period (P1 for 1976-85, P2 for 1986-95 and P3 for 1996-2000), age-25 weight status (lean and overweight/obese), age in months and mortality status. The unweighted analytical sample is summarized in Table 2. The total sample amounts to 1,863,317 person-month records with 2796 deaths. Noteworthy is the small number of deaths for some period-weight categories. The obese in the third period, for example, total 64 deaths (24 from N2 and 40 from N3) for all ages combined. This weakness of the data would constrain the analysis and the interpretation of the results, as we will discuss in geater detail.

One restriction of the remote data access is that it does not permit directly calculating mortality rates without using a statistical model. To explore empirical regularities, the N3 data released in the public domain are utilized. The analysis does not use the publicly available N2 mortality file (through 1992), because it undercounts 239 deaths out of a total of 2384.

For eight 10-year birth cohorts in the N3 sample, the mortality rates of the age-25 overweight or obese relative to the lean (mortality ratios) are calculated and graphed in Figure 3. The large fluctuations in the ratio curves reflect the small number of death events observed above: Mortality ratio is zero if no death in the overweight or obese group, and goes to infinity if no death in the lean group. Despite the large fluctuations, one can tell a mortality excess that is of a similar magnitude for the age-25 overweight and obese. This is contrary to the survival advantage of the overweight reported for weight concurrent with the survey baseline.[6]

Additionally, there is a gradual cohort pattern of mortality ratios increasing from earlier to later cohorts, although the fluctuations are also stronger for later cohorts. Such cohort variations translate into a declining age pattern when no cohort distinction is made, as shown in Figure 4. This cohort/age pattern of mortality differentials could be a consequence of cohort differences in exposure, or an artifact of poor data among old adults, an issue that we will address in a later section on data quality.

The N3 mortality data are further divided into two time periods (1988-95, 1996-00). In Figure 5, mortality ratios for each period are shown in the top graph, and the period ratios (that is, 1996-00 mortality ratios divided by 1988-95 ratios) on the logarithmic scale are shown in the bottom graph. Here the overweight and obese are combined to compare with the lean, since the splitting of the data into two periods have taxed the limited number of deaths. Although the sampling fluctuations prevent a definitive statement, there is some indication of an increase in excess mortality from 1988-95 to 1996-00, especially for the younger age groups. It is important to note the spike of period ratios at about age 40, exceeding 40 on the original scale. That is, the relative mortality in 1996-00 is *impossibly* 40 times that in 1988-95. In a statistical analysis, such observations would have a big influence that should be borne in mind.

The statistical modeling of the data uses the parametric Gompertz function, characterized by an exponential increase of mortality over age a. The model can be written as:

$$h(a) = exp(\beta + \gamma a), \tag{2}$$

where $h(a)$ denotes age-specific mortality rates in the NHANES sample, and β and γ, the scale and shape of the mortality curve.

As SAS, the only statistical software permitted in the NCHS remote access, does not have a procedure for the Gompertz model, we use the complementary log-log function implemented in the SAS logistic procedure to analyze the discrete person-month data constructed above, which would produce comparable results for the underlying continuous-time survival process.[36] (p.216-7) Supplementary analysis using limited publicly available NHANES mortality data confirmed the comparability of model estimates and goodness-of-fit statistics between the Gompertz model in STATA and the discrete analysis in SAS. The limitation with the latter is the inability to examine unobserved heterogeneity, which can be easily implemented in STATA by adding a frailty parameter to the Gompertz model.

The substantive interest in the weight-mortality relationship and its change over time translates into modeling how each of the two parameters (β and γ) vary by two covariates: weight group W (lean, overweight or obese), and time period P (P1, P2 or P3). A weight term in the γ equation is equivalent to an interaction between weight and age on the mortality outcome in a standard regression framework, indicating age variations in how mortality varies by weight. Thus, any term in the γ equation (interaction effect) is predicated on its presence in the β equation (main effect). In the exposition, we use the word interaction only to refer to terms that interact between period and weight, because in computing we would be actually modeling β and γ, rather than $h(a)$.

Using a shorthand to denote the respective dependence of β and γ on covariates, we consider five candidate models:

$M0$: P_P
$M1$: PW_P
$M2$: PW_PW
$M3$: P×W_PW
$M4$: P×W_P

$M0$ is the baseline model, under which mortality shape (γ), and scale (β) vary by period, but neither by weight status. All other models include terms for weight. Under $M1$ and $M2$, mortality differs by weight, but the differentials do not change by time period, as indicated by the absence of an interaction between period and weight. The interaction terms (denoted by P×W) are included in $M3$ and $M4$. $M1$ and $M2$ (or $M3$ and $M4$) differ in whether the weight differences in mortality vary over age, captured by the weight terms in the γ equation. We do not entertain a model with period-weight interactions on γ, thus constraining any age pattern of weight differences in mortality to be period-invariant. This is out of prior considerations over the weakness of the data. Given the small number of deaths, and the tremendous sampling fluctuations observed earlier, this more conservative approach is taken to model time change. Substantively, three-way interactions between weight, age and time are certainly possible when cohort replacement drives the age patterns of mortality differentials to change over time.

Each of the four models can be simplified further by combining periods or weight groups. The three-period classification, however, is maintained in main effects, because we believe that the three periods differ in baseline mortality. The strategy is to select the best out of the four (and the baseline), and then simplify its terms to get a more parsimonious model. Model selections are based on the AIC statistic, with a smaller AIC value indicating a better fit, and models within two AIC units of each other are tied with no clear winner.[37]

Table 3 presents goodness of fit statistics for the fitted models. Based on the AIC, $M3$ emerges as a clear winner out of the five. Under $M3$, mortality differs by weight, and these differences vary by age and time, but the age variations are time-invariant. The superiority of $M3$ to $M4$ (constant age pattern) and to $M2$ (no time change) confirms the earlier observations about the change in mortality ratios over time and cross-sectional age groups.

Re-labeling $M3$ as $M3.1$, three nested models are fit further. $M3.2$ combines the overweight and obese in all weight terms, which is a substantial improvement over the global model $M3.1$ ($AIC(M3.2)$=26382.176; $AIC(M3.1)$=26386.904). However, further simplifications regarding the periodization (time change) result in a poorer fit in $M3.3$ (Mortality differentials are equal between P2 and P3: AIC=26386.613), or no substantial improvement in $M3.4$ (Mortality differentials are equal between P1 and P2: AIC= 26381.029). Therefore, we choose $M3.2$ as the preferred model. Under $M3.2$, mortality rates differ between the lean or not lean, but not between the overweight or obese at age 25. This difference varies over age and among the three time periods.

Figure 6 presents the age- and period-specific mortality ratios and uncertainty intervals estimated under the preferred model $M3.2$. They are calculated from the model parameter and variance-covariance estimates shown in Table A-2 and Table A-3 in the Appendices. In all periods, the overweight/obese women suffered higher mortality than the lean through most ages. In the most recent period (1996-2000), overweight/obese mortality doubled or tripled that of the lean at the youngest ages. This excess declined to about 35% at age 85. All these elevations were statistically significant at $\alpha = .05$, and importantly, they had been increasing steadily over time.

One may ask what if baseline weight is used instead? Results from the same analytical samples but using baseline weight would not be comparable because due to weight change over mortality follow-up, the weight classification of N2 respondents in P3, for example, is different from that in P1, but unknown to the researcher. A cohort-specific analysis of N2 and N3 baseline weight finds a similar expansion of mortality differentials among recent cohorts:[38] Based on weight concurrent with the survey baseline, overweight or obese relative mortalities increase from N2 to N3 among middle-aged adults (born in 1931-50). As N2 and N3 overlap in mortality followup, these increases strictly have no interpretation as change over time.

6 Mortality by Age-25 Weight Status

Age-Specific Rates

Mortality ratios and age-25 BMI distributions, both estimated from the NHANES, are combined with national mortality rates to derive weight-specific mortalities, using the following identities:

$$\begin{aligned} {}_5M_x &= {}_5P_x \cdot {}_5M_x^l + (1 - {}_5P_x) \cdot {}_5M_x^o \\ {}_5M_x^o &= {}_5M_x^l \cdot {}_5R_x \end{aligned} \tag{3}$$

where x denotes age, ${}_5M_x$ total mortality rates, ${}_5M_x^l$ mortality rates for the lean, ${}_5M_x^o$ mortality rates for the overweight/obese, ${}_5P_x$ proportions lean, and ${}_5R_x$ mortality ratios. The two equations are used to solve for the two unknowns (${}_5M_x^l$ and ${}_5M_x^o$).

The various input and output data of Eq. 3 are presented in a tabular format in the Appendices for each time period. Figure 7 graphs the total and weight-specific mortality rates on the logarithmic scale. Over the study period, for most age groups, total mortality and the mortality of those who were lean at age 25 declined continuously, whereas overweight/obese mortality deteriorated, slightly from 1976-80 to 1988-94, but noticeably from 1988-94 to 1999-2004.

To better observe mortality change over time, period mortality ratios (ratios of 1988-94 mortality rates to those in 1976-80, and ratios of 1999-2004 mortality rates to those in

1988-94) are presented in Figure 8, and Table A-7 in the Appendices. Reductions of total mortality diminished in the second half of the period. However, the decline in lean mortality rates showed no sign of abating, averaging about 15% in the first interval and 17% in the second up to age 90. The life chances of the overweight/obese ran a contrary course: Mortality increment averaged about 2% from 1976-80 to 1988-94, and about 13% from 1988-94 to 1999-2004. These trends, combined with a decreasing percentage of the population who were lean at age 25 appear to be responsible for slowed US female mortality reductions at the turn of the 21st century. Between 1988-94 and 1999-2004, the age groups (40-60 and 75+) that experienced the most sluggish reductions in total mortality were also the ones that witnessed the biggest reversals in overweight/obese mortality.

Note that due to age-misreporting, US death rates at age 95+ calculated from vital statistics and the census have been shown to seriously understate the true rates.[39] In Figure 8, the increase over time in the death rates at these ages could be due to improvement in data quality. In this age-specific analysis, total mortality rates at these high ages do not affect the mortality levels and trends of the lower ages. Nor would they have a significant impact on the values of life expectancy at age 25, as analyzed next.

Life Expectancy and Partial Life Expectancy

Age-specific mortality rates are used to calculate life expectancy, assuming that on average, deaths occur half-way through the five-year age interval.[40] Estimates for life expectancy at age 25, and partial life expectancy between age 25 and 65 for each age-25 weight group and the total female population are presented in Figure 9. Total life expectancy at age 25 increased continuously, but the pace of increase slowed down in the second half. The period from 1976-80 to 1988-94 saw an increase of 1.21 y from 53.88 to 55.10, and from 1988-94 to 1999-2004, the increase was almost halved to .66 y. As expected from the trends for age-specific mortality rates, the weight groups diverged in their life chances. The lean did not abate in their march towards longevity, gaining 1.3 y of life in each interval to reach 57 y in 1999-04. The overweight/obese lost .4 y in the first interval, and 1.9 y in the second so that in 1999-2004, their life expectancy at age 25 was 50.4, 2.3 y shorter than 30 years ago, and nearly 7 y shorter than their lean counterparts.

A similar pattern of divergence is observed for partial life expectancy. In each interval, out of the 40 prime years of life, the age-25 lean achieved an increase of about .3 y in each interval to reach 39 y in 1999-04. By contrast, the life chances of the overweight/obese increased by a trivial .06 y in the first interval, and in the second, took a turn for the worse and dropped by .15 y to 37.5 y. Therefore, their expected years of life between 25 and 65 in 1999-04 was .1 y shorter than 30 years earlier, and 1.5 y shorter than the lean.

Years of Life Shortened

Years of life shortened due to overweight/obesity at age 25 is calculated as the difference in life expectancy between the lean weight group and the total population. That is, we ask for each point of time, what US female life expectancy at age 25 (or between age 25 and 65) would have been if all weight groups had had the same mortality rates as those of the lean group.

As shown in Figure 10, years of life lost increased, from one fourth of a year in the 70s to 1.3 y at the dawn of the 21st century. For partial life expectancy, the loss increased from .14 y to .34 y. 1.3 y is much bigger than the estimate based on weight concurrent with study baseline (one third to three fourths of a year) for life expectancy at birth in the year 2000.[5] The health burden of excess fat appears to be understated when using concurrent weight.

7 Data Quality

The innovation to use body weight at the fixed age of 25 to characterize the population weight distribution raises questions about data quality. Age-25 weight is used instead of the commonly used concurrent weight, which is measured by health professionals in the NHANES, but more vulnerable to confounding by health-related weight change for the older section of the population. Age 25 presents the same and shorter exposure to weight change for adults of all ages. However, age-25 weight is not measured but self-reported retrospectively. Before attempting substantive explanations of the findings, it is necessary to examine possible bias due to data quality. For this purpose, we examine: 1) for eight 10-year birth cohorts, the age patterns of the proportion of the population who reported to be lean at age 25 (proportion age-25 lean), 2) for each of the three cross-sectional surveys, proportions missing for age-25 weight, and 3) for the 1945-54 cohort, proportions age-25 lean at specific ages based on retrospective reports and those predicted from weight measured at age 25 and mortality, a contrast to be compared with that between proportions lean based on measured and self-reported current weight. These explorations would provide an idea about the extent and nature of bad data, on the basis of which we further construct two scenarios to gauge the potential bias on mortality trends due to poor retrospective reports, and conduct two sensitivity analyses focusing on the impact of older subjects' retrospective reports on the trends in mortality ratios. Lastly, we compare the results with those from the Nurses' Health Study, where age-18 weight was retrospectively self-reported by women aged 24-44, and from the Framingham Heart Study, where body weight was measured at age 30-49.

Figure 11 presents age patterns of proportion age-25 lean for eight 10-year birth cohorts born between 1895-1974. In this analysis, the same cohort is traced through the cross-sectional NHANES series, making use of the retrospective reports belonging to the same cohort but different individuals at various ages. Age is restricted between 25 and 84, and cases are deleted if foreign-born to avoid migration into a birth cohort. Under the assumption of a consistently higher mortality across adult ages for the age-25 overweight/obese as compared with the age-25 lean, and in consistency with historical weight trend, we expect the proportion age-25 lean to increase by age but decrease across cohort.

Proportions lean appear to have an overall increasing age pattern for the later four female cohorts, but more erratic for earlier cohorts. Later cohorts also have a rather consistent cohort trend so that at each age, later cohorts have a lower proportion than earlier ones. Taking into account sampling variations, most age differences in proportions within cohort are not statistically significant at $\alpha = .05$. While the age patterns do not provide conclusive evidence for the quality of retrospective self-reported age-25 weight, it indicates that data for later cohorts at younger ages are more reliable than earlier cohorts at older ages.

Older people are not only more prone to erratic retrospective reports, but also more likely not to report at all, as shown in Figure 12. Non-response rate increases across cross-sectional age groups from about 2-3% to nearly 10%. On the other hand, data quality appears to have improved over time, as non-response rate is consistently lower in N99 than N2 or N3.

To get a sense of the magnitude of data error in retrospectively reported weight distributions, we next focus on women born in 1945-54 to compare their proportions age-25 lean between age 25 and 60 based on two sources: retrospectively self-reported, and predicted from measured BMI at age 25 and mortality at succeeding ages. This comparison is followed by examining self-reported and measured current weight at these ages for the same cohort.

Measured age-25 BMI is obtained from the measured weight and height of cohort respondents who entered the 1971-75 or 1976-80 NHANES at age 25. Proportions age-25 lean at later ages are predicted recursively from earlier ages, using measured BMI at the exact age of 25, the ratio of overweight/obese mortality to lean mortality, and total age-specific mortality rates, all for the same one cohort. Mortality ratio is estimated from N2 and N3 using cohort data, and total mortality rates are obtained from the Human Mortality Database.

To predict proportions lean at age 30 (${}_5P_{30}$), for example, we use the following equation:

$$ {}_5P_{30} = \frac{{}_5P_{25} \cdot exp(-5 \cdot {}_5M^l_{25})}{{}_5P_{25} \cdot exp(-5 \cdot {}_5M^l_{25}) + (1 - {}_5P_{25}) exp(-5 \cdot R \cdot {}_5M^l_{25})} \quad (4) $$

where ${}_5P_{25}$ is calculated from measured age-25 BMI, mortality ratio R is constant over age at $exp(.8292)$, and age-specific lean mortality rate ${}_5M^l_{25}$ is calculated from cohort total mortality rate using Eq. 3. The proportions at succeeding ages are predicted accordingly. The first two columns of Table 4 compare the proportions lean (BMI 25 or less) based on retrospective self-reports and predictions. The former exceed the latter by about 7-11%, indicating a downward bias in self-reported age-25 weight.

How does this discrepancy compare to the discrepancy between measured and self-reported weight concurrent with the survey baseline? Self-reported concurrent weight has been, and continues to be extensively used in mortality research, although its downward bias has been widely acknowledged.[41] The last two columns of Table 4 shows the two proportions for concurrent BMI. The difference is about 2-3%, much smaller than the discrepancy for age-25 BMI. Thus, retrospective self-reports appear to deviate more from the true than current self-reports, suggesting that in addition to the concern about self-image, recall errors may have also played a big role in retrospective reporting. This is consistent with the results in the two preceding explorations indicating less reliable weight reports among older subjects, to whom the recalled event should be more remote, in the more distant past where body weight was probably less a relevant health issue than in more recent decades.

What implications do the under-reporting of age-25 weight, and its possible change over time have for mortality estimates and their time trend? Under-reporting would bias downward mortality ratios because overweight/obese subjects are misclassified as lean. However, given the way we construct the person-month records from the N2 and N3 data, and divide them into three non-overlapping periods (P1, P2 and P3 as shown in Figure 1), ratio estimates are unlikely to be affected by changes in data quality, since the P3 ratios are estimated from data collected in P1 and P2. Therefore, for simplicity, we first assume no bias in ratio estimates, and examine how biases in the age-25 weight distribution and changes in data quality affect estimates of mortality rates for each weight group and their trends. Then we examine how data quality might affect ratio estimates, and compare our results with those from the Framingham Heart Study and Nurses' Health Study.

Recall that weight distributions (that is, proportions lean) are combined with mortality ratios to derive weight-specific mortality from national total mortality, using Eq. 3. Holding constant mortality ratios and total mortality, under-reporting age-25 weight and inflating the proportion of the population that is healthy (lean) would bias up the mortalities of subpopulations, the lean and overweight/obese alike. We construct two scenarios to examine the impact of weight understatement: reported proportions lean are 10% above the true in all N2, N3 and N9; and 2) reported proportions lean are 5% above the true in N99 for the first four age groups, and 10% above the true in N2 and N3, and for the remaining ages in N99. The quality of retrospective reports improves in N99 because, for example, later cohorts know and recall their age-25 weight better. Under each scenario, we correct the retrospectively reported proportions, and the resulting period ratios of weight-specific mortality rates are shown in Figure 13 (scenario 1) and Figure 14 (scenario 2). Overall, the trend of mortality reductions for the lean, and reversals for the overweight/obese remain unchanged.

Could the time trend in relative mortalities be driven by age/cohort differences in the quality of retrospective reports? That relative mortalities decline over age but increase over time could be due to the less reliable age-25 weight reported by older people in earlier cohorts. To address this concern, two separate mortality analyses are performed: constraining the mortality differentials to be constant over age, and restricting the analytical samples to those whose baseline age is less than 65. The overall findings are similar: Relative mortality rates increase in the most recent period.

We are not aware of prior research using self-reported age-25 weight to classify weight groups and analyze weight trends or mortality differentials at the population level. Based on age-18 weight self-reported by 102,400 women aged 24-44 in the Nurses' Health Study, relative to the mortality of those having a BMI between 18.5 and 21.9, the estimate for obese mortality is 2.79 (95%CI, 2.04-3.81), overweight mortality 1.66 (1.31-2.10), and underweight mortality 0.98 (0.78-1.23).[25] In the Framingham Heart Study for subjects aged 30-49 at the baseline in 1948-51 with measured body weight and mortality through 1990, estimates of obese mortality relative to the normal-weight ranged between 1.5 and 2.3, and differences among weight groups' expected years of life at age 40, between 3 to 7 years.[12] These ratio and expectancy values are comparable to the ones for age-25 weight obtained earlier or in additional calculations for weight-specific life expectancy at age 40 (shown in Table A-8 in the Appendices).

Obviously a bit arbitrary is the extrapolation procedure used to model the age-25 BMI distributions at the older ages. This is done to preserve the well-documented secular trend in body weight, and to be consistent with mortality selection with respect to age-25 weight at younger ages. However, this procedure is unlikely to influence the results for mortality rates at the younger ages, or partial life expectancy. In short, data quality issues are not sufficient to explain away the change over time in mortality differentials by age-25 weight.

8 Discussion

The analysis of population weight distributions based on age-25 weight finds a time trend that is consistent with results based on weight concurrent with the study baseline.[3] Cohort replacement has driven the rising proportion overweight/obese at age 25 to increasingly older ages. In 1999-2004, about two-fifths of Americans at age 25 had a Body Mass Index of 25 and greater. The mortality implications of age-25 weight, however, are strikingly different from current weight. Mortality does not differ between the age-25 underweight and normal-weight, and compared with the combined lean group, the age-25 overweight and obese have equally elevated mortality. At the end of the 20th century, mortality ratio ranged between 3.7 at age 25 and 1 at age 100. Mortality excess was statistically significant up to age 85.

These results contrast starkly with the large body of research based on concurrent weight, which found that being overweight or moderately obese protects or at least does not harm survival, whereas underweight suffers the highest mortality.[9,10] The new findings are consistent with the biomedical mechanisms through which excess fat mass, but not lean mass, has been understood to induce physiologic pathologies and chronic diseases.[1] They are also consistent with results for concurrent weight restricted to healthy non-smokers.[11] Estimates based on concurrent weight tend to be smaller among old adults, but are of a more similar magnitude among young or middle-aged adults.[12,38]

These differences and similarities provide indirect evidence for unobserved heterogeneities that have long been suspected among concurrent weight groups but defied an analytic panacea.[17,21] Among old adults, those with manifest or occult illnesses or those who die early[42] are more likely to experience weight loss. However, weight or disease history is hard to measure, and their dynamic inter-relationship is likely to vary across populations, rendering standard analytical procedures such as regression adjustment or sample exclusion ineffective and controversial.[17] Estimates of excess mortality based on concurrent weight appear to have been distorted or biased downward, especially among old adults.

This study demonstrates that age-25 weight is a useful alternative measure for studying the health burden of excess body fat in cross-sectional populations. At age 25, illness-related weight change should be minimal. In the NHANES series, we suspect that the retrospective self-reports of old subjects are less reliable than younger ones. All evidence, however, suggests an improvement over time in data quality for all ages, probably because body weight has become a more salient feature of health and recall error has diminished. The series of data quality checks and sensitivity analysis indicate robust results for mortality differentials based on age-25 weight.

In addition to providing new and less biased estimates, this is the first study about how the mortality impact of excess weight has changed in the past decades. Age-25 weight disparities in mortality on a variety of metrics widened in the last thirty years of the 20th century. Excess overweight/obese mortality increased. Take the aged 50-54 group for example. Mortality ratio increased from 1.5 in the late 1970s to 2.4 in the late 1990s. Lean mortality rates continued to decline at an unabated pace, whereas mortality reversals were observed for the overweight/obese women since the late 1970s. Their respective life expectancy at age 25 was 57.1 y and 50.4 y (a near 7-y gap) in 1999-2004, as compared with 54.1 y and 52.6 y in 1976-80 (a 1.5-y gap). Partial life expectancy saw a similar widening trend. Weight-related reductions of life increased from .24 y to 1.31 y, and for partial life expectancy, from .14 y to .34 y.

In additional statistical models that include education in three categories (no high school, high school and at least some college), the temporal patterns of relative mortality rates persist. NHANES did not ask about smoking in early adulthood, but current smoking status. Cross-tabulations found no systematic association between age-25 weight and current smoking, which of course is pre-dated by age-25 weight.

It has been suggested that advances in medical technology and health care might have reduced or even eliminated weight-related health disparities.[6] The hypothesis of health improvement/convergence is little supported by this research. Both *relative* and *absolute* mortality of the heavier groups are found to increase over time. The new findings, however, echo observations made by some medical experts, noting the serious side effects of multi-drug treatment for the obesity-related medical complications, and the cost and need for life-long use without a complete cure.[17,43]

What could be responsible for the unfavorable trends then? The clue may lie in the increasing exposure to excess weight. Among children aged 2-19, 5% of them were overweight in 1971-4, and the number increased to 17% in 2003-4.[44] Obese children and adolescents are more likely to develop the metabolic syndrome.[45] Not only did recent birth cohorts start out with more body fat, they also experienced more rapid weight gain over adulthood. Figure 15 shows age-specific proportion overweight or obese (BMI 25 and greater), and proportion severely obese (BMI 40 and greater) for female cohorts born between 1895-1984, using measured concurrent weight and height data in the NHANES series. At each age, the proportions are higher for more recent cohorts. Moreover, the steepness of the curves increases, indicating a greater weight gain over the life course among more recent cohorts.

Under such trends, we can expect that at the time of mortality evaluation, the age-25 overweight or obese are heavier in the most recent period. Note that the role of compositional differences within weight groups with respect to age-25 weight should be small, because as shown in Figure 1, for the period that experienced the significant increase in excess mortality (1996-2000), the age-25 weight classification was based on weight reports that the NHANES collected in earlier periods, where the increase in excess mortality was negligible. What matters more is weight gain over time/age after age 25. Even though based on age-25 weight the weight classification is unchanged, weight concurrent with the most recent period should have increased, probably more so for the age-25 overweight/obese, as suggested by the weight trajectories in Figure 15. While there is no denying the clinical benefits of modern medical treatments and drugs, and all else being equal, they should make excess fat less deadly, these advances appear to have failed to countervail the physiological harms brought by the extended and more intense exposure to excess weight, and for a greater share of the population.

The results provide empirical evidence in support of the claim that the amount of life shortened due to obesity would increase in the future.[5] They also agree with recent studies showing a decline in female life expectancy in 180 US counties,[46] and a slowed improvement in US female life expectancy at age 65, as compared with Japan and France in recent decades.[24] The new results shed light on excess weight as an underlying risk factor for the less favorable trends. Despite an overall improvement in public health, unhealthy life styles and an environment that encourages unhealthy living have led to the deterioration of health for a significant portion of the population and a widening of health disparities. This portends a real storm in public health.

References

1 WHO, *Obesity: preventing and managing the global epidemic, report of a WHO consultation*. Geneva: World Health Organization, 2000.

2 Ogden CL, Fryar CD, Carroll MD, Flegal KM. Mean body weight, height, and body mass index, United States 1960-2002. *Advance Data From Vital and Health Statistics* 2004:347.

3 Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999-2000. *JAMA* 2002:288(14):1723–1727.

4 Zhang Q, Wang Y. Trends in the association between obesity and socioeconomic status in U.S. adults: 1971 to 2000. *Obesity Res* 2004:12(10):1622–1632.

5 Olshansky SJ, Passaro DJ, Hershow RC, et al. A potential decline in life expectancy in the United States in the 21st century. *N Engl J Med* 2005:352(11):1138–1145.

6 Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. *JAMA* 2005:293(15):1861–1867.

7 Gregg EW, Cheng YJ, Cadwell BL, et al. Secular trends in cardiovascular disease risk factors according to body mass index in US adults. *JAMA* 2005:293(15):1868–1874.

8 Flegal KM, Graubard BI, Williamson DF, Gail MH. Cause-specific excess deaths associated with underweight, overweight, and obesity. *JAMA* 2007:298(17):2028–2037.

9 McGee DL. Body mass index and mortality: a meta-analysis based on person-level data from twenty-six observational studies. *Ann Epidemiol* 2005:15(2):87 – 97.

10 Romero-Corral A, Montori VM, Somers VK, Korinek J, Thomas RJ, Allison TG. Association of bodyweight with total mortality and with cardiovascular events in coronary artery disease: a systematic review of cohort studies. *Lancet* 2006:368:666–78.

11 Adams KF, Schatzkin A, Harris TB, et al. Overweight, obesity, and mortality in a large prospective cohort of persons 50 to 71 years old. *N Engl J Med* 2006:355(8):763–778.

12 Peeters A, Barendregt JJ, Willekens F, Mackenbach JP, Mamun AA, Bonneux L. Obesity in adulthood and its consequences for life expectancy: a life-table analysis. *Ann Intern Med* 2003:138(1):24–32.

13 Blair SN, LaMonte MJ. Commentary: current perspectives on obesity and health: black and white, or shades of grey? *Int J Epidemiol* 2006:35(1):69–72.

14 Couzin J. A heavyweight battle over CDC's obesity forecasts. *Science* 2005: 308(5723):770–771.

15 Gronniger JT. A semiparametric analysis of the relationship of body mass index to mortality. *Am J Public Health* 2006:96(1):173–178.

16 Campos P, Saguy A, Ernsberger P, Oliver E, Gaesser G. The epidemiology of overweight and obesity: public health crisis or moral panic? *Int J Epidemiol* 2006:35(1):55–60.

17 Hu FB. Overweight and increased cardiovascular mortality: no French paradox. *Hypertension* 2005:46(4):645–646.

18 Losonczy KG, Harris TB, Cornoni-Huntley J, et al. Does weight loss from middle age to old age explain the inverse weight mortality relation in old age? *Am J Epidemiol* 1995:141(4):312–321.

19 Harris TB, Savage PJ, Tell GS, Haan M, Kumanyika S, Lynch JC. Carrying the burden of cardiovascular risk in old age: associations of weight and weight change with prevalent cardiovascular disease, risk factors, and health status in the Cardiovascular Health Study. *Am J Clin Nutr* 1997:66(4):837–844.

20 Flegal KM, Graubard BI, Williamson DF, Gail MH. Impact of smoking and preexisting illness on estimates of the fractions of deaths associated with underweight, overweight, and obesity in the US population. *Am J Epidemiol* 2007:166(8):975–982.

21 Willett WC, Dietz WH, Colditz GA. Guidelines for healthy weight. *N Engl J Med* 1999: 341(6):427–434.

22 The BMI in Diverse Populations Collaborative Group. Effect of smoking on the body mass index-mortality relation: empirical evidence from 15 studies. *Am J Epidemiol* 1999:150(12):1297–1308.

23 Thomas F, Bean K, Pannier B, Oppert JM, Guize L, Benetos A. Cardiovascular mortality in overweight subjects: the key role of associated risk factors. *Hypertension* 2005: 46(4):654–659.

24 Meslé F, Vallin J. Diverging trends in female old-age mortality: the United States and the Netherlands versus France and Japan. *Popul Dev Rev* 2006:32(1):123–45.

25 van Dam RM, Willett WC, Manson JE, Hu FB. The relationship between overweight in adolescence and premature death in women. *Ann Intern Med* 2006:145(2):91–97.

26 Haas SA. The long-term effects of poor childhood health: an assessment and application of retrospective reports. *Demography* 2007:44(1):113–135.

27 National Center for Health Statistics. Plan and operation of the First National Health and Nutrition Examination Survey: United States, 1971-1973. In: *Vital Health Stat*, 1973, vol. 1 of *1*, 1–46.

28 National Center for Health Statistics. Plan and operation of the HANES I Augmentation Survey of Adults 25-74 Years: United States, 1974-1975. In: *Vital Health Stat*, 1978, vol. 14 of *1*, 1–110.

29 National Center for Health Statistics. Plan and operation of the Second National Health and Nutrition Examination Survey, 1976-1980. In: *Vital Health Stat*, 1981, vol. 15 of *1*, 1–144.

30 National Center for Health Statistics. Plan and operation of the Third National Health and Nutrition Examination Survey, 1988-94. In: *Vital Health Stat*, 1994, vol. 32 of *1*, 1–407.

31 National Center for Health Statistics, NHANES 1999-2000 data files. Available online at http://www.cdc.gov/nchs/about/major/nhanes/NHANES99_00.htm (accessed Oct 16, 2007).

32 National Center for Health Statistics, NHANES 2001-2002 data files. Available online at http://www.cdc.gov/nchs/about/major/nhanes/NHANES01_02.htm (accessed Oct 16, 2007).

33 National Center for Health Statistics, NHANES 2003-2004 data files. Available online at http://www.cdc.gov/nchs/about/major/nhanes/NHANES03_04.htm (accessed Oct 16, 2007).

34 National Center for Health Statistics Research Data Center, NHANES-linked mortality data files. (Remotely accessed January-June, 2008).

35 Human Mortality Database. University of California, Berkeley (USA) and Max Planck Institute for Demographic Research (Germany). Available online at www.mortality.org (accessed Feb 23, 2008).

36 Allison PD, *Survival analysis*. Cary, NC: SAS Institute, 1995, pp. 216-7.

37 Burnham KP, Anderson DR, *Model selection and multimodel inference: a practical information-theoretic approach*. New York: Springer, 2002.

38 Yu Y. Re-examining the age effect on the weight-mortality relationshhip: age of death, age of weight and cohort effect. CDE Working Paper 2008-18, Center for Demography and Ecology, University of Wisconsin-Madison, 2008.

39 Coale AJ, Kisker EE. Defects in data on old-age mortality in the United States: new procedures for calculating mortality schedules and life tables at the highest ages. *Asian Pac Popul Forum* 1990:4(1).

40 Preston SH, Heuveline P, Guillot M, *Demography: measuring and modeling population processes*. Oxford and Massachusetts: Blackwell Publishers, 2001.

41 Visscher TLS, Viet AL, Kroesbergen IHT, Seidell JC. Underreporting of BMI in adults and its effect on obesity prevalence estimations in the period 1998 to 2001. *Obesity* 2006:14(11):2054–63.

42 Barone BB, Clark JM, Wang NY, Meoni LA, Klag MJ, Brancati FL. Lifetime weight patterns in male physicians: the effects of cohort and selective survival. *Obesity* 2006: 14:902–908.

43 Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. *Lancet* 2005: 365(9468):1415 – 1428.

44 Ogden CL, Yanovski SZ, Carroll MD, Flegal KM. The epidemiology of obesity. *Gastroenterology* 2007:132(6):2087 – 2102.

45 Weiss R, Dziura J, Burgert TS, et al. Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med* 2004:350(23):2362–2374.

46 Ezzati M, Friedman AB, Kulkarni SC, Murray CJL. The reversal of fortunes: trends in county mortality and cross-county mortality disparities in the United States. *PLoS Med* 2008:5(4):0557–0568.

Table 1: Age-25 Body Mass Index (BMI) Distribution, Weighted Percentage (Unweighted N), NHANES, Females

Baseline	1976-80 NHANES		1988-94 NHANES		1999-2004 NHANES	
Age Group	BMI $<$ 25	BMI $\geq$ 25	BMI $<$ 25	BMI $\geq$ 25	BMI $<$ 25	BMI $\geq$ 25
25-29	78.1 (598)	21.9 (179)	75 (631)	25 (285)	59.2 (424)	40.8 (335)
30-34	82.2 (526)	17.8 (130)	80.4 (727)	19.6 (245)	69.6 (504)	30.4 (235)
35-39	86.8 (454)	13.2 (70)	82.4 (722)	17.6 (197)	74.6 (469)	25.4 (173)
40-44	85.9 (427)	14.1 (79)	87.1 (679)	12.9 (159)	80.4 (491)	19.6 (145)
45-49	88 (386)	12 (59)	87.6 (456)	12.4 (86)	81.3 (449)	18.7 (124)
50-54	88.3 (414)	11.7 (59)	82.9 (428)	17.1 (99)	81.7 (423)	18.3 (117)
55-59	86.5 (425)	13.5 (78)	84.6 (409)	15.4 (93)	84.9 (308)	15.1 (65)
60-64	87 (1010)	13 (163)	87.4 (495)	12.6 (101)	88.9 (509)	11.1 (103)
65-69	85.7 (852)	14.3 (149)	85.3 (446)	14.7 (102)	84.6 (396)	15.4 (108)
70-74	82.4 (662)	17.6 (145)	80 (455)	20 (125)	85.9 (361)	14.1 (85)
75-79	NA (NA)	NA (NA)	82 (332)	18 (91)	82.2 (272)	17.8 (73)
80-84	NA (NA)	NA (NA)	82.3 (373)	17.7 (89)	84.4 (347)	15.6 (74)
Total N	6865		7825		6590	

Table 2: Person-Months and Deaths in Pooled NHANES II and III Mortality Samples, Females, Unweighted

Time Period and Age-25 Weight	NHANES II (1976-80 to 2000) Total	Deaths	NHANES III (1988-94 to 2000) Total	Deaths
Year 1976-85 (P1)				
Lean	354834	299	0	0
Overweight	50100	50	0	0
Obese	15518	23	0	0
Year 1986-95 (P2)				
Lean	389929	725	292553	288
Overweight	53980	119	51788	69
Obese	16000	39	23605	23
Year 1996-00 (P3)				
Lean	159534	423	338200	482
Overweight	20813	70	61761	122
Obese	6219	24	28335	40

Table 3: Goodness of Fit Statistics for Selected Gompertz Models, NHANES II and III Mortality Samples, Females, Weighted

Model Description	$-2LogL$	df	AIC	AIC - AIC_{min}
M0: P_P, Baseline Model	26414.768	6	26426.768	44.592
No Weight differentials in Mortality Scale or Shape				
M1: PW_P, Weight differentials in Mortality Scale	26378.495	8	26394.495	12.319
M2: PW_PW, Weight differentials in Mortality Scale & Shape	26369.977	10	26389.977	7.801
M3: PxW_PW, Period-Weight Interactions on Mortality Scale				
(1) Full Model	26358.909	14	26386.904	4.728
(2) Overweight + Obese	26362.176	10	26382.176	0
(3) (2), P1 vs. P2 + P3 in Interactions	26368.613	9	26386.613	4.437
(4) (2), P1 + P2 vs. P3 in Interactions	26368.613	9	26381.029	-1.147
M4: PxW_P	26371.502	12	26395.502	23.326

Table 4: Proportion Lean (BMI $\leq$ 25), Women Born in 1945-54, NHANES (1971-75, 1976-80, 1988-94, 1999-05) and Human Mortality Database, Weighted

Age Group (Age)	Age-25 Weight: Retrospectively Self-Reported	Age-25 Weight: Predicted	Current Weight: Self-Reported	Current Weight: Measured
(25)	–	–	0.7326	0.7272
25-34 (30)	0.7925	0.7279	0.7057	0.6759
35-44 (40)	0.8416	0.7297	0.5378	0.5099
45-54 (50)	0.8125	0.7337	0.3587	0.3402
55-60 (60)	0.8329	0.7422	0.3536	0.3139

Figure 1: Survey Baselines and Mortality through 2000, NHANES, 1976-80 (N2), 1988-94 (N3) and 1999-04 (N99)

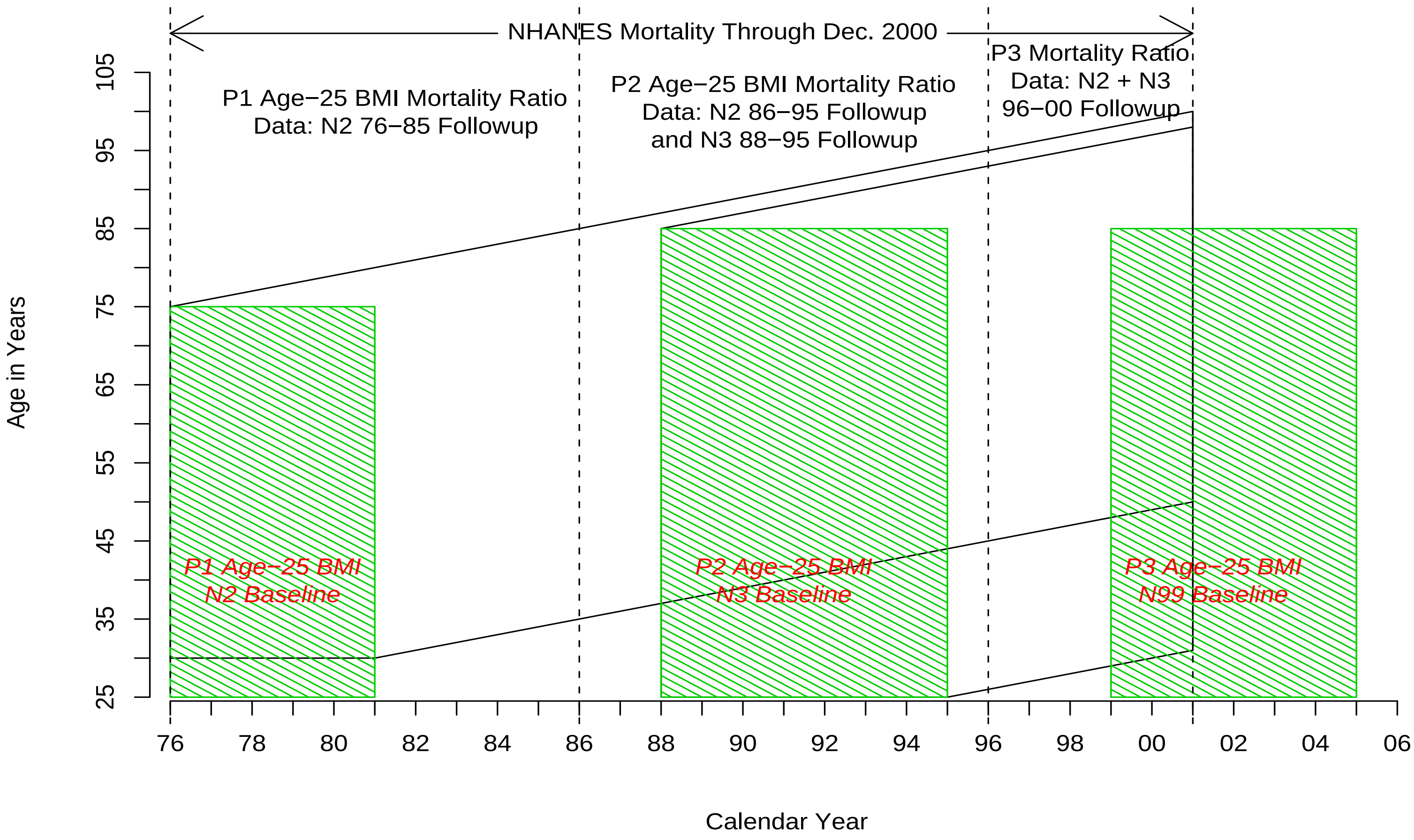


Figure 2: Age-25 Weight Distribution, Retrospectively Reported vs. Modeled, NHANES, Females, Weighted

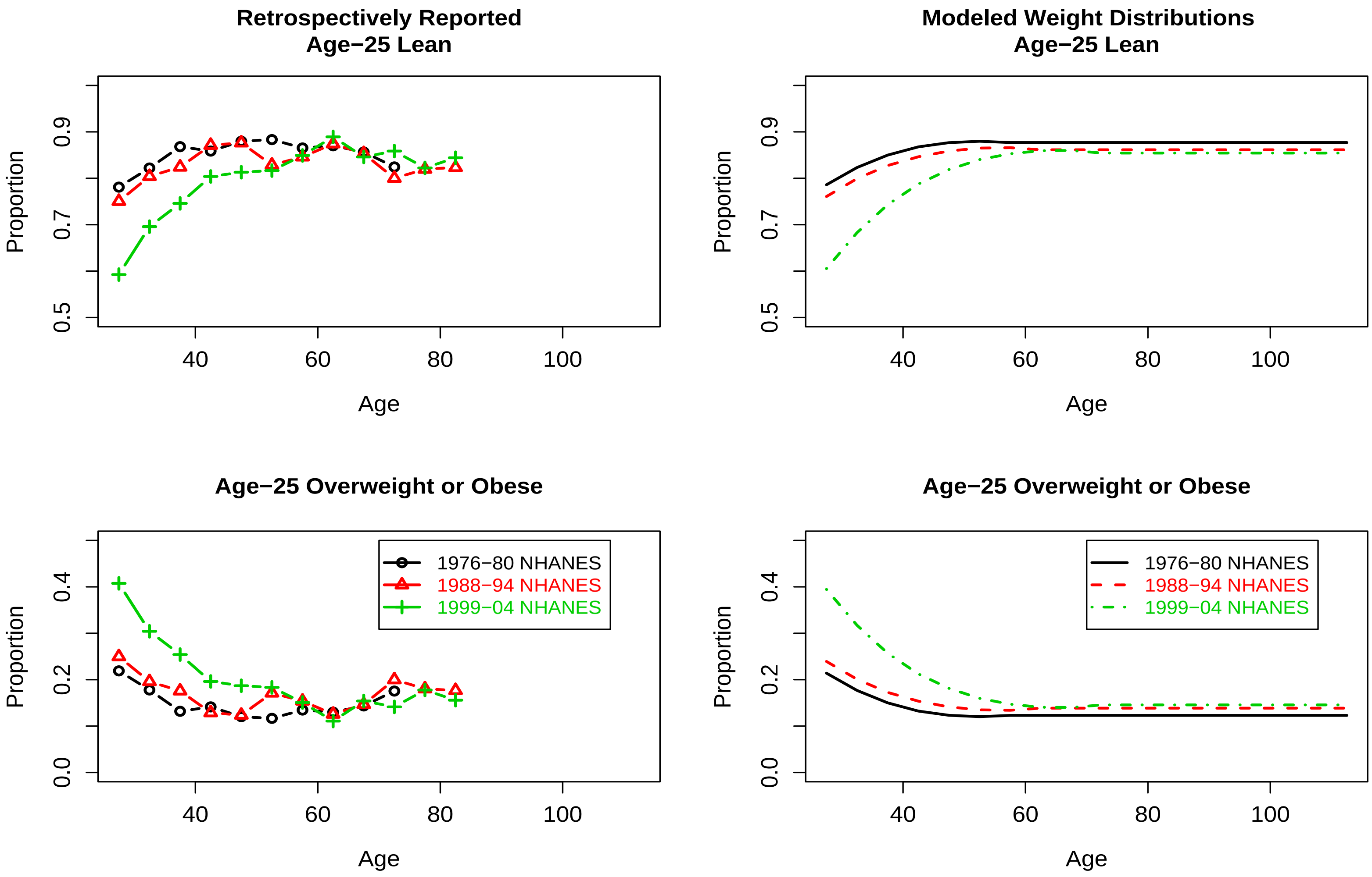


Figure 3: Observed Mortality Ratios by Age-25 Weight, 1988-94 NHANES Mortality Followed through 2000, Females, Weighted, By Birth Cohorts: c.1895-1904 (circle), c.1905-14 (triangle up), c.1915-24 (+), c.1925-34 (×), c.1935-44 (diamond), c.1945-54 (triangle down)

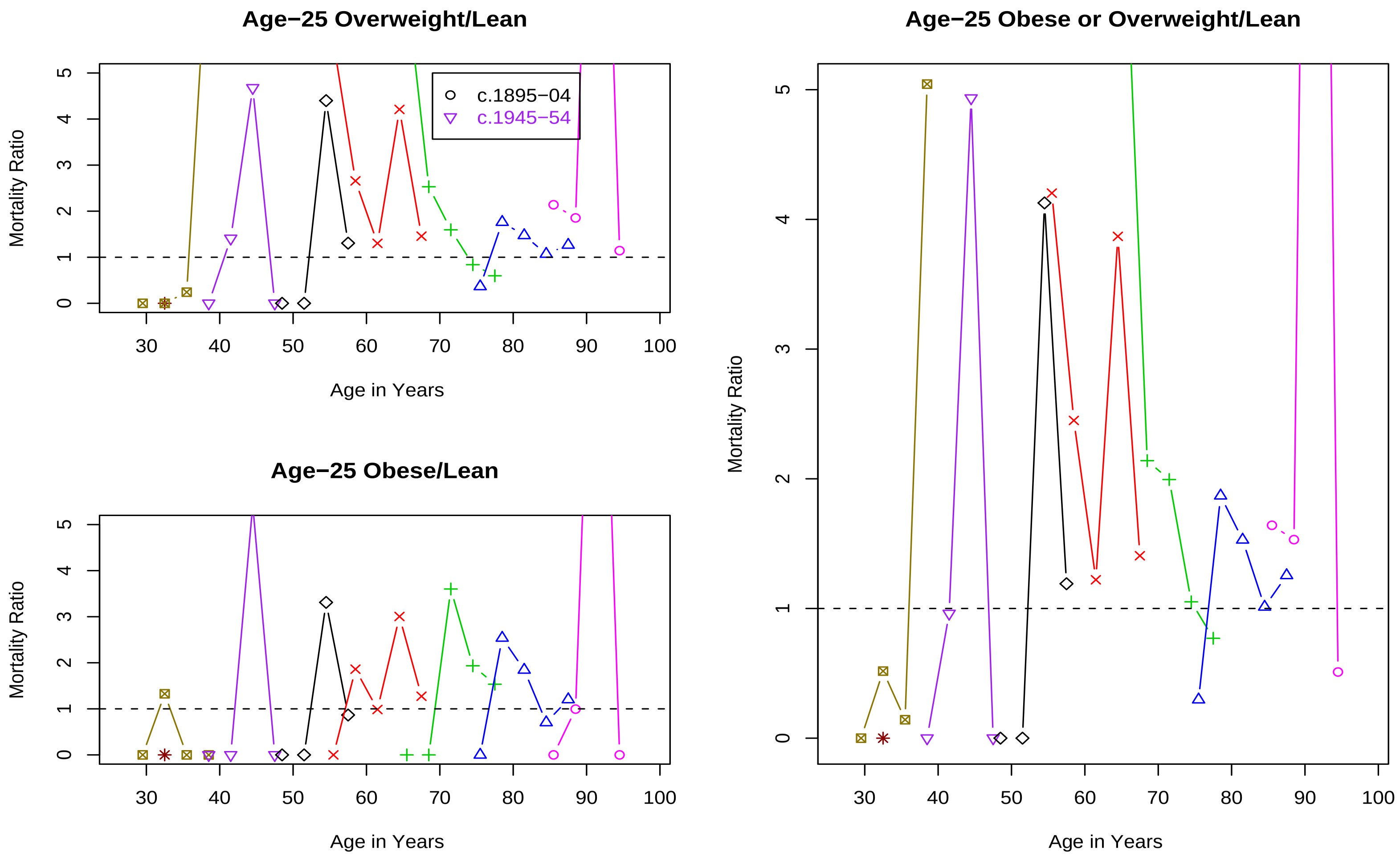


Figure 4: Observed Mortality Ratios by Age-25 Weight, 1988-94 NHANES Mortality Followed through 2000, Females, Weighted, All Birth Cohorts

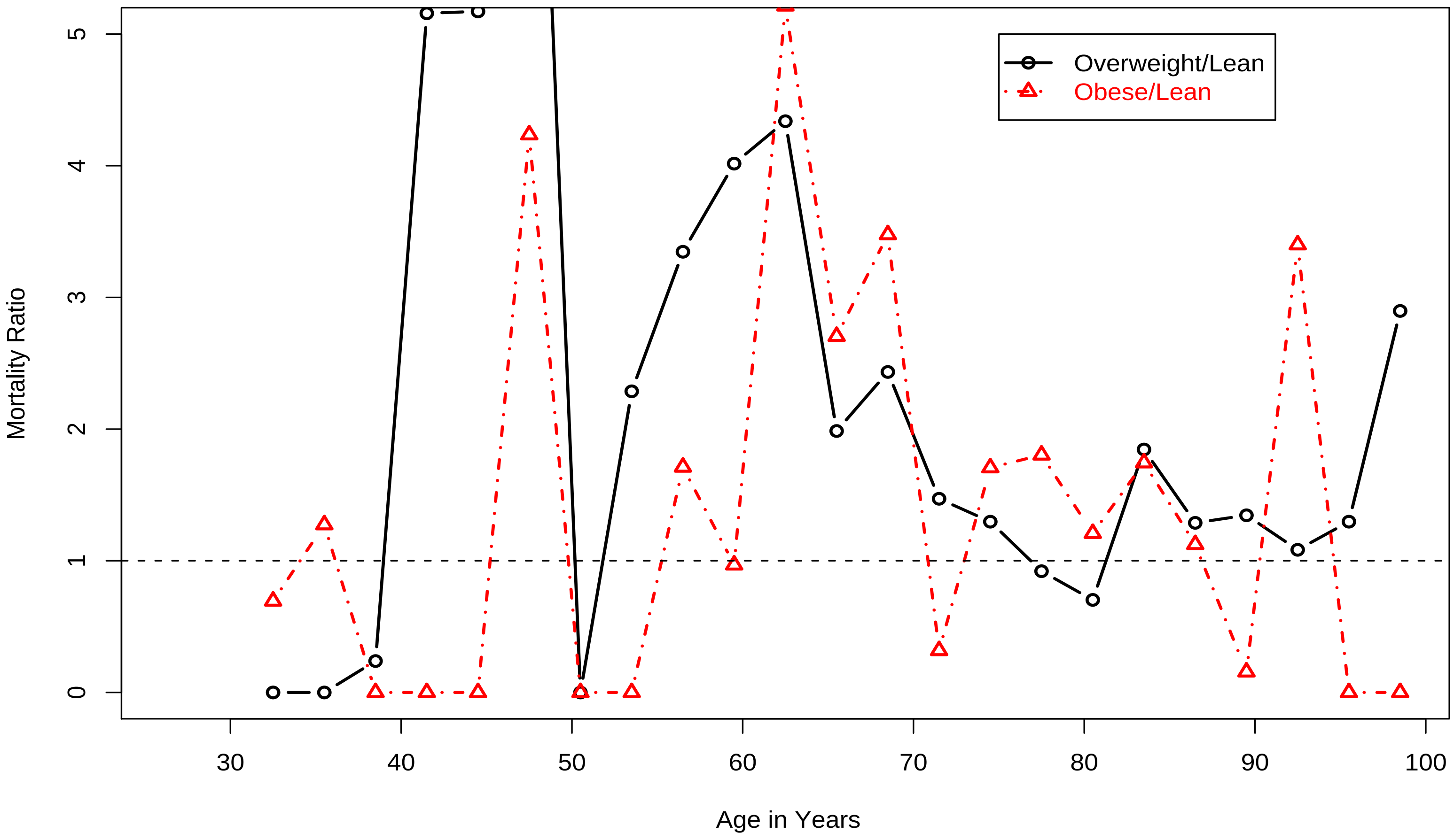
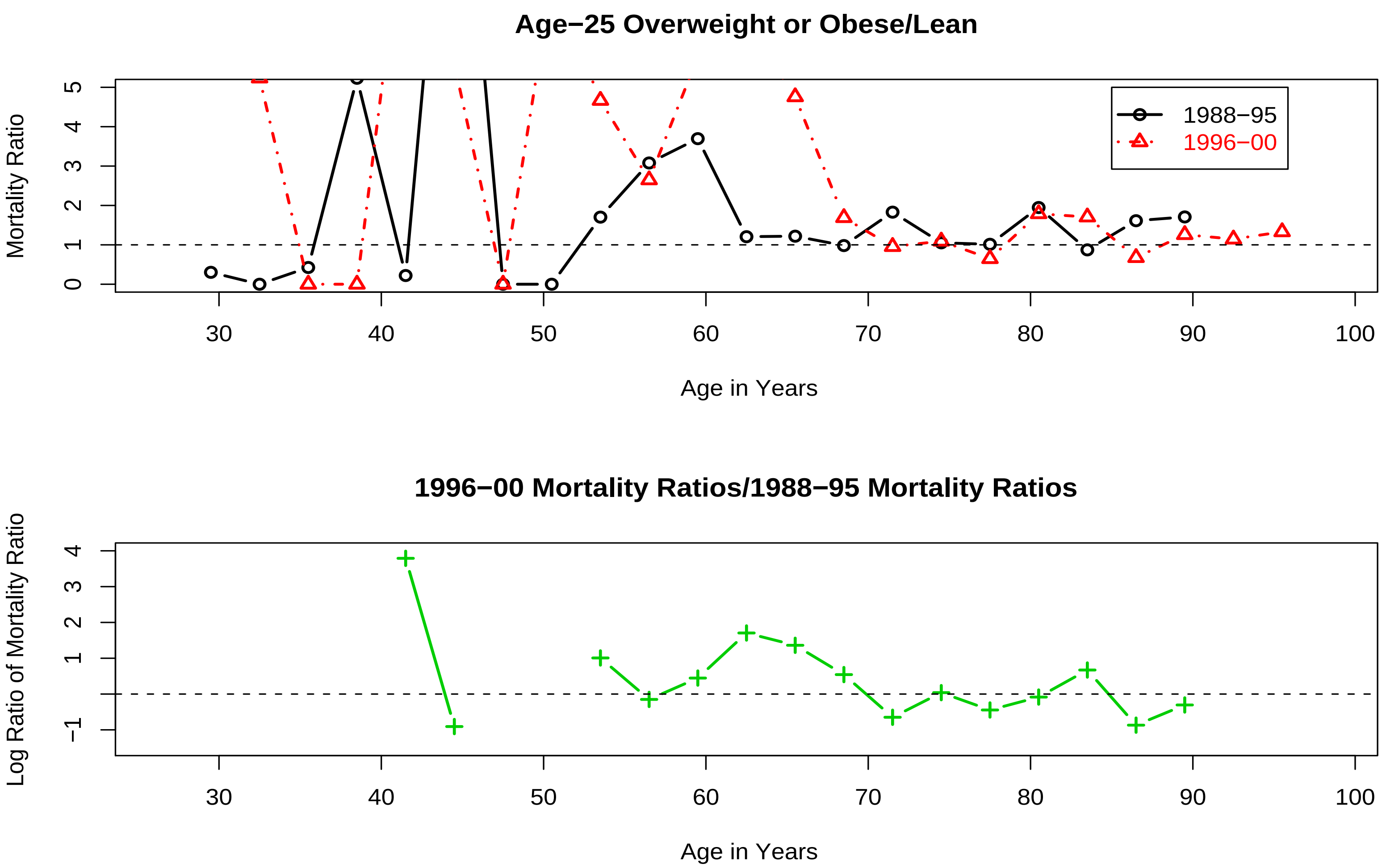


Figure 5: Observed Mortality Ratios by Age-25 Weight, 1988-94 NHANES Mortality Followed through 2000, Females, Weighted, All Birth Cohorts and by Period


Age−25 Overweight or Obese/Lean

1996−00 Mortality Ratios/1988−95 Mortality Ratios

Figure 6: Modeled Mortality Ratios by Age-25 Weight, 1976-80 and 1988-94 NHANES Mortality Followed through 2000, Females, Weighted

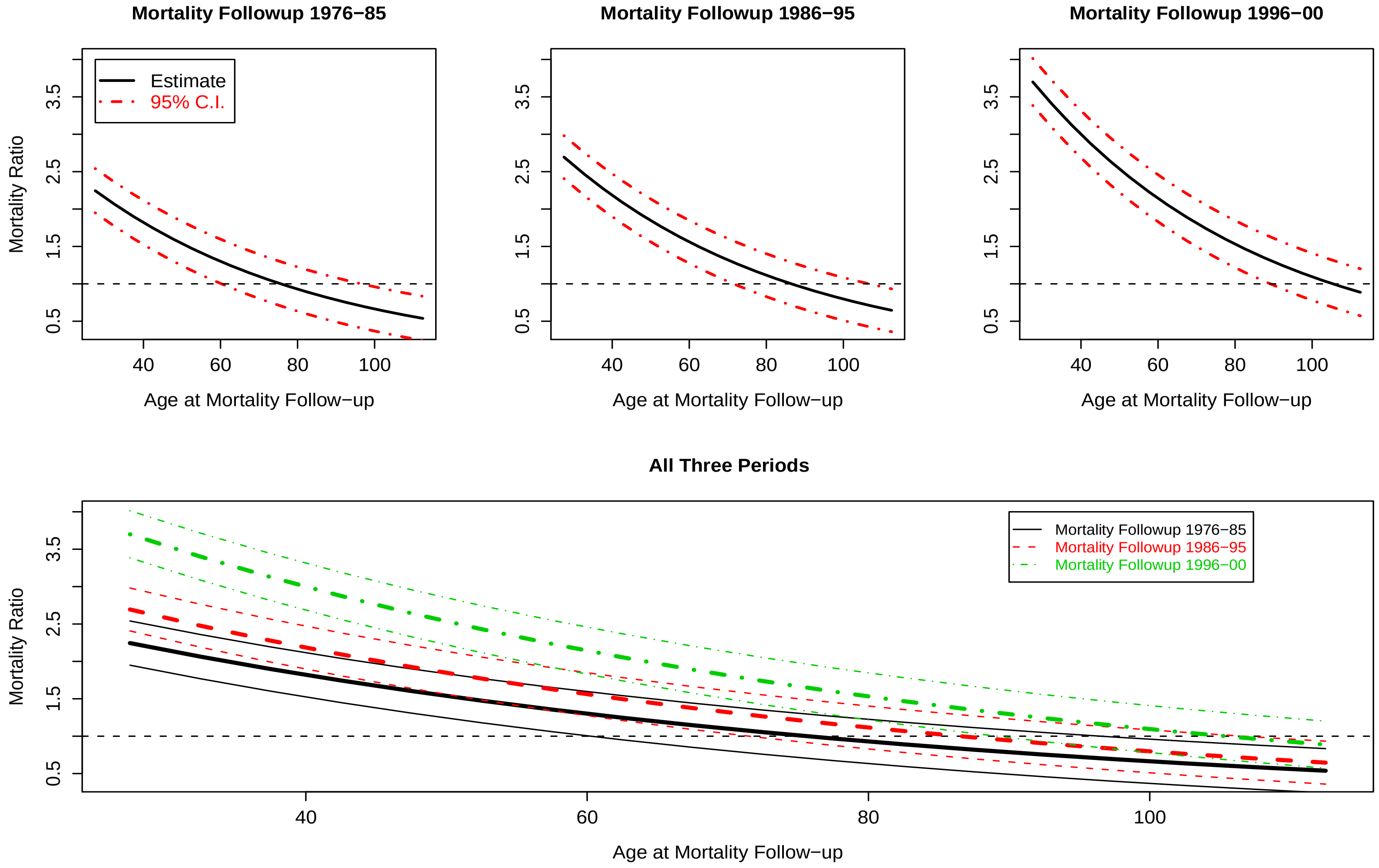


Figure 7: US Female Age-Specific Mortality Rates, NHANES and Human Mortality Database, Total Population and by Age-25 Weight

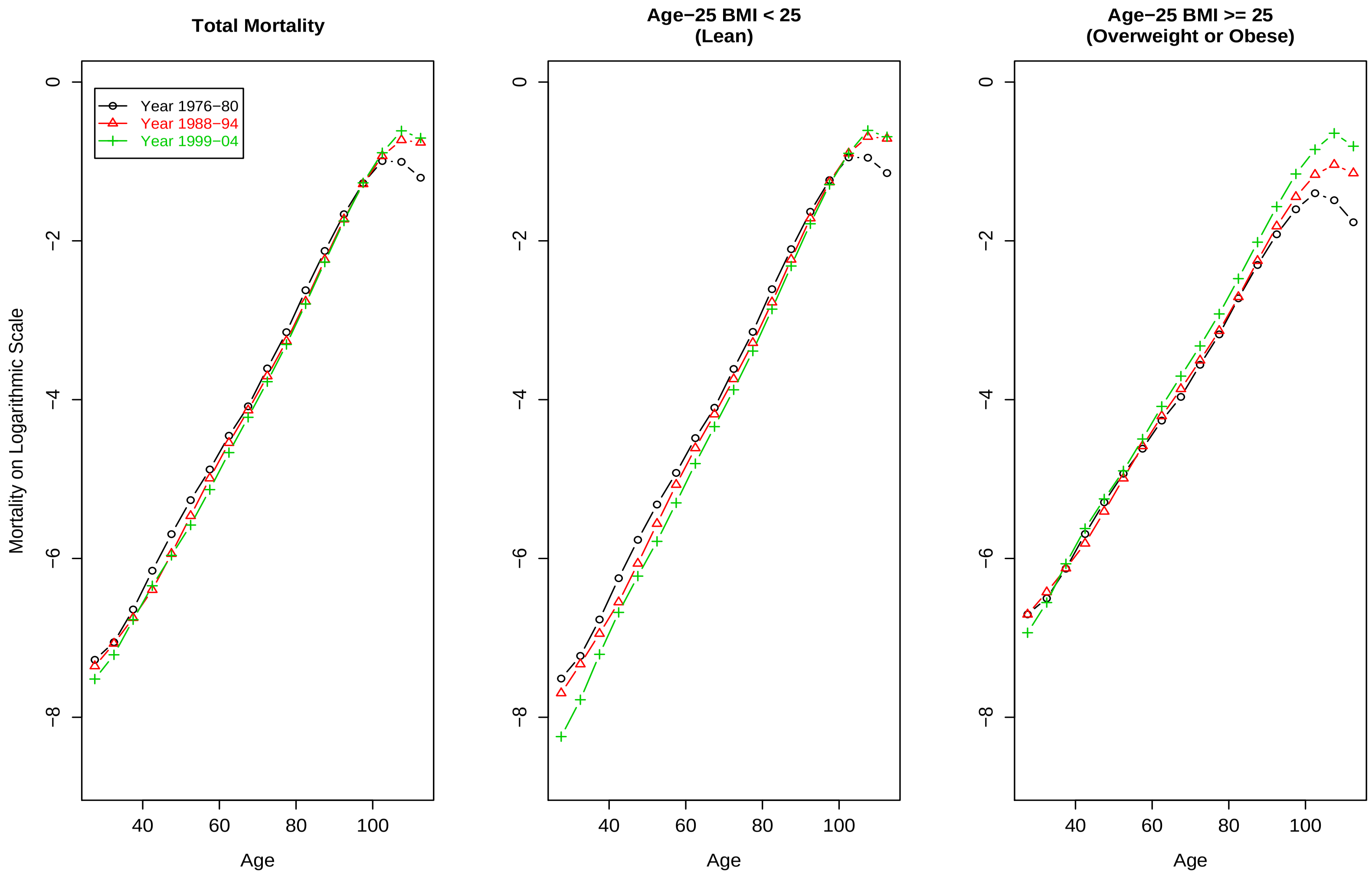


Figure 8: Period Ratios of US Female Age-Specific Mortality Rates, NHANES and Human Mortality Database, Total Population and by Age-25 Weight

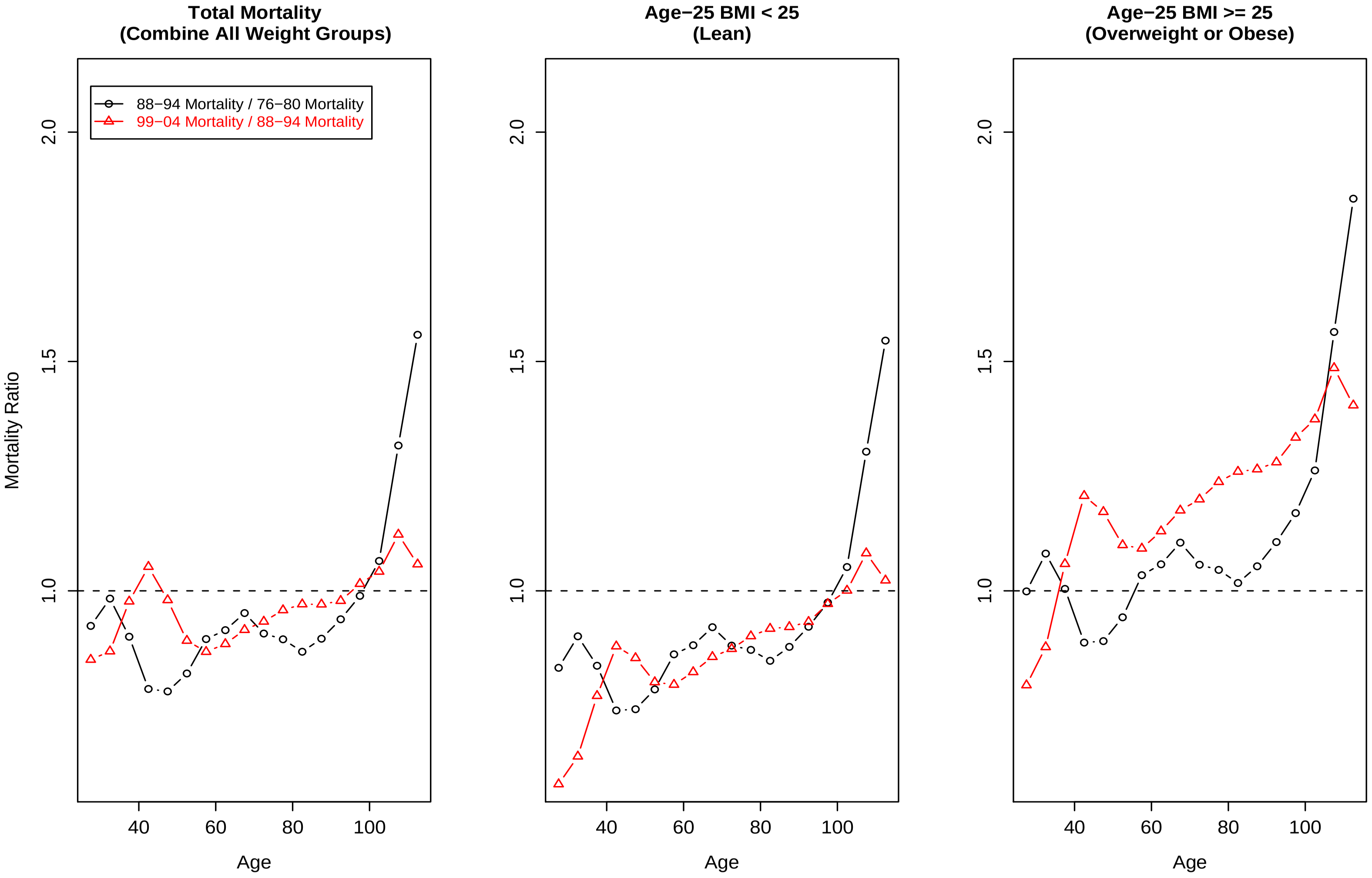


Figure 9: (Partial) Female Life Expectancy, NHANES and Human Mortality Database, Total Population and by Age-25 Weight

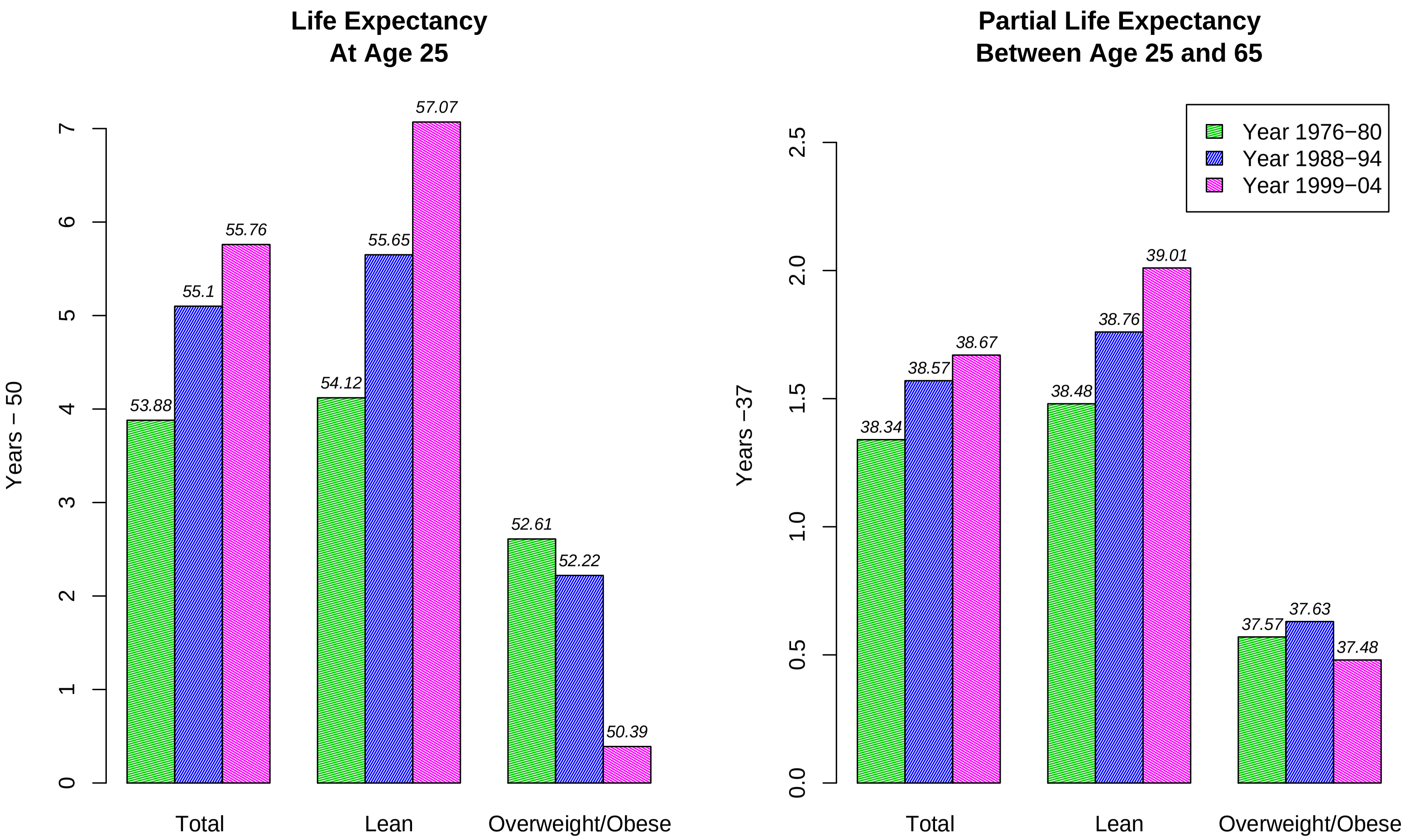


Figure 10: Female Life Expectancy Shortened due to Overweight/Obesity at Age 25, NHANES and Human Mortality Database

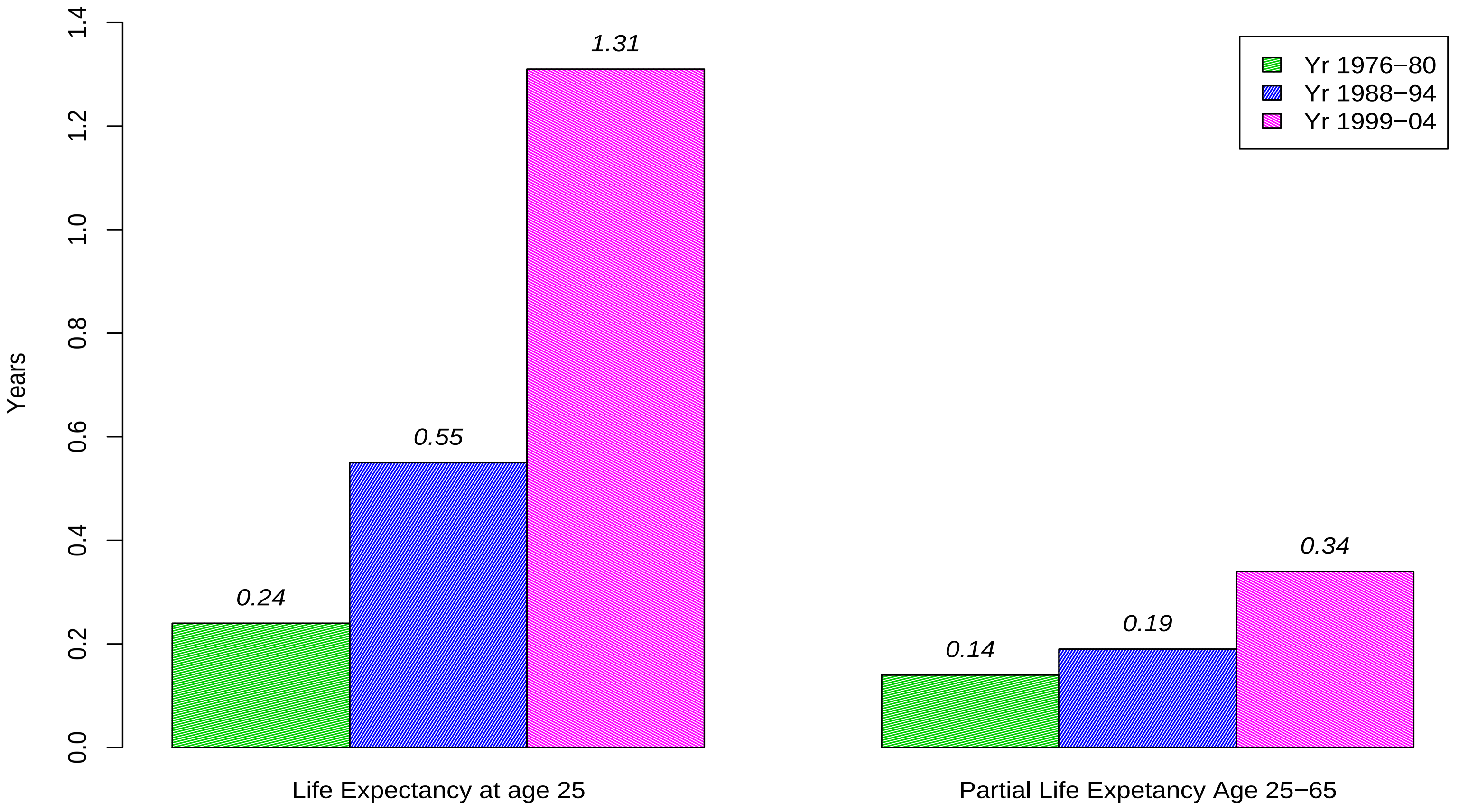


Figure 11: Retrospectively Reported Age-25 Proportion Lean, NHANES (1976-80, 1988-94, 1999-2004), Females, Weighted, By Birth Cohort

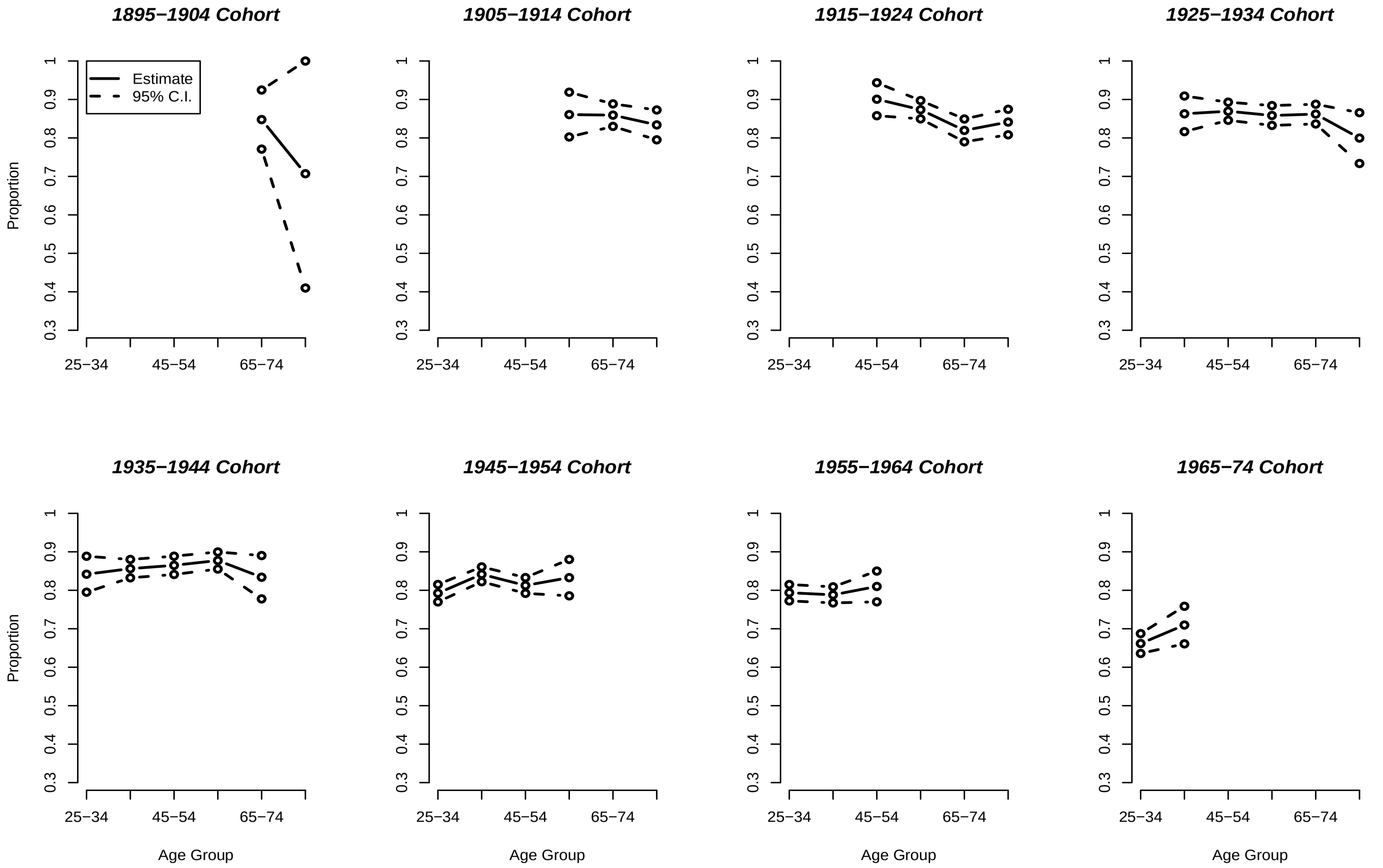


Figure 12: Proportion Missing for Retrospectively Reported Age-25 Weight, NHANES, Females, Weighted

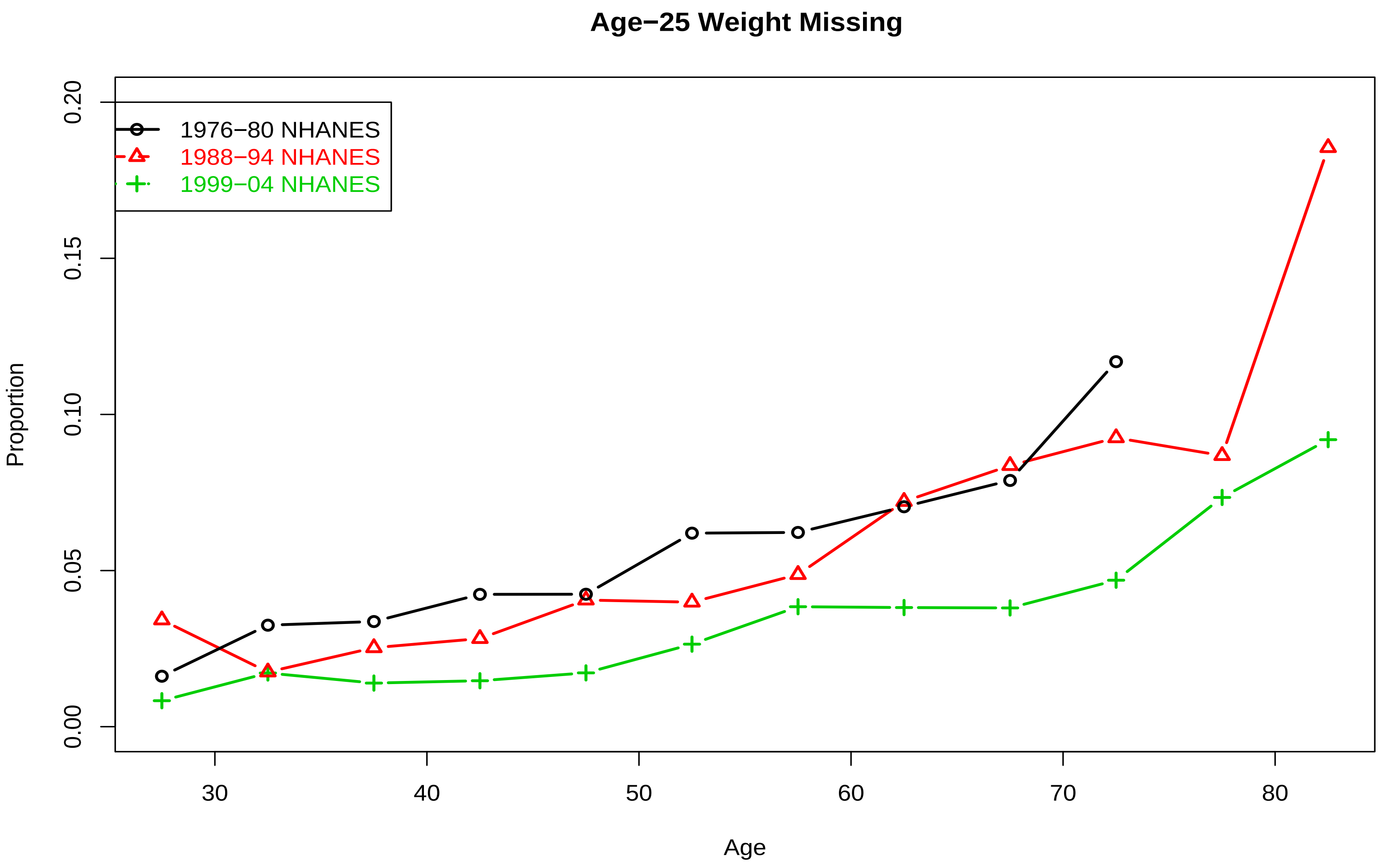


Figure 13: Period Ratios of US Female Age-Specific Mortality Rates, NHANES and Human Mortality Database, Total Population and by Age-25 Weight, Retrospectively Reported Proportion Age-25 Lean Corrected

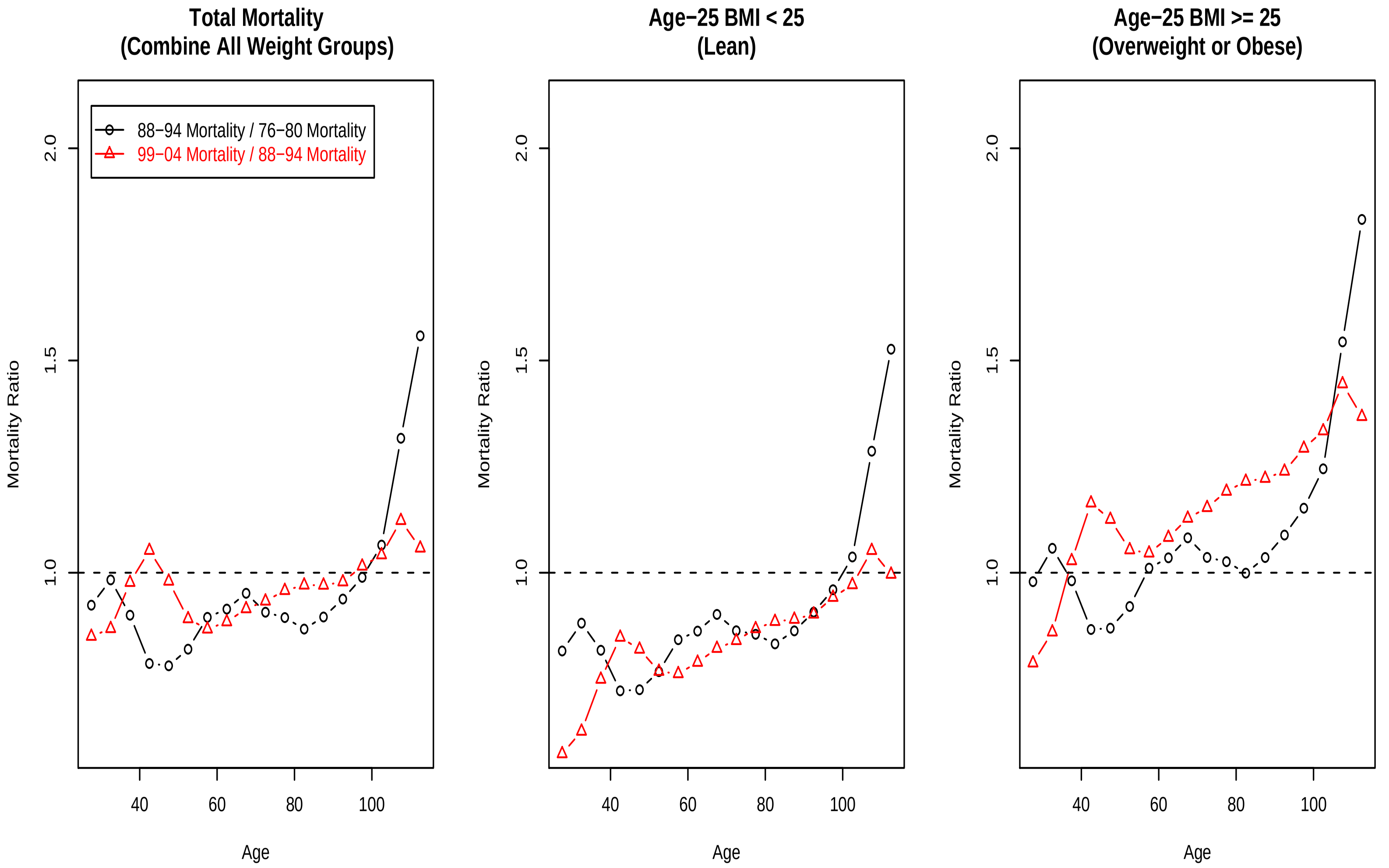


Figure 14: Period Ratios of US Female Age-Specific Mortality Rates, NHANES and Human Mortality Database, Total Population and by Age-25 Weight, Retrospectively Reported Proportion Age-25 Lean Corrected and Allowing for Change in Data Quality across Surveys

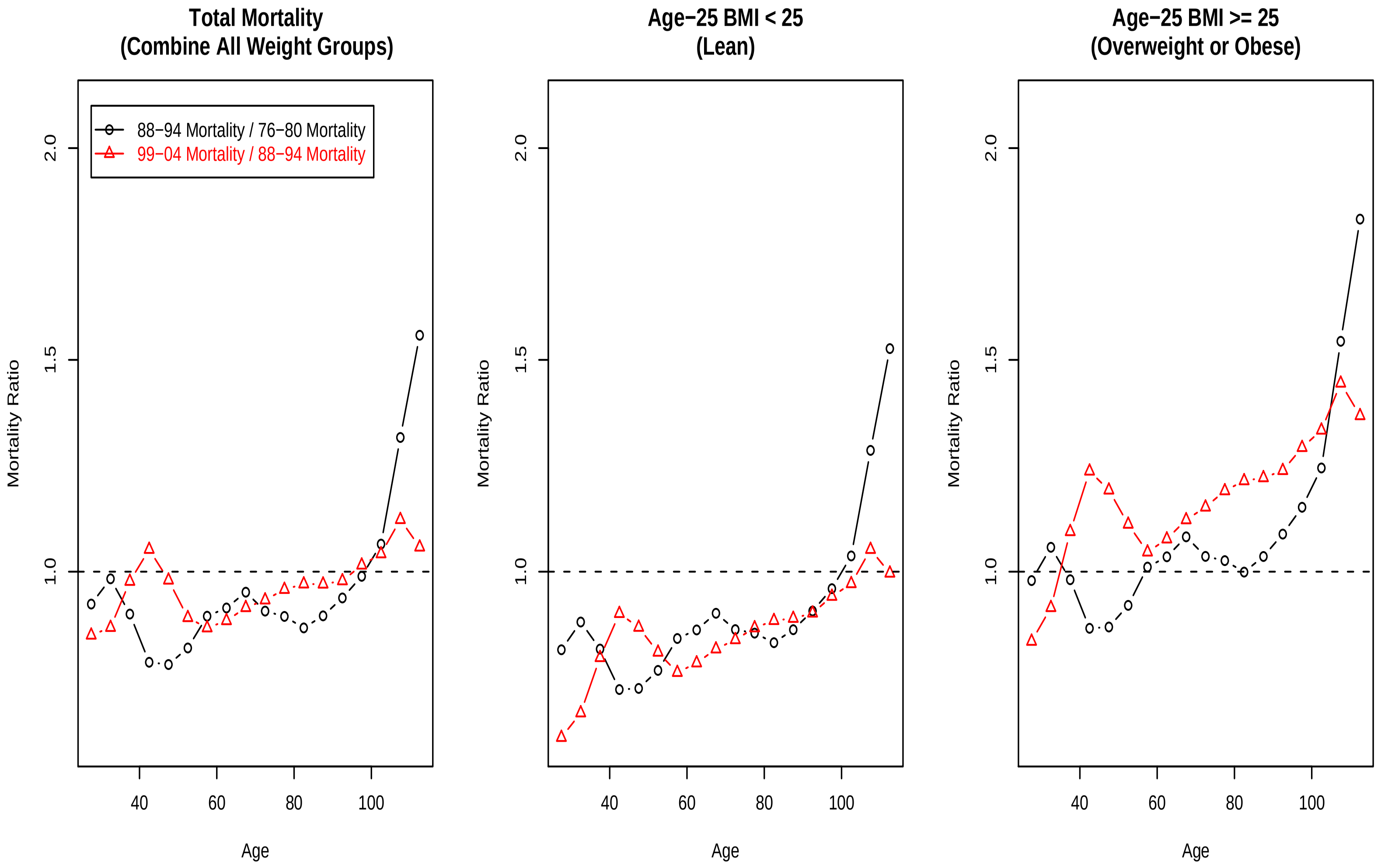
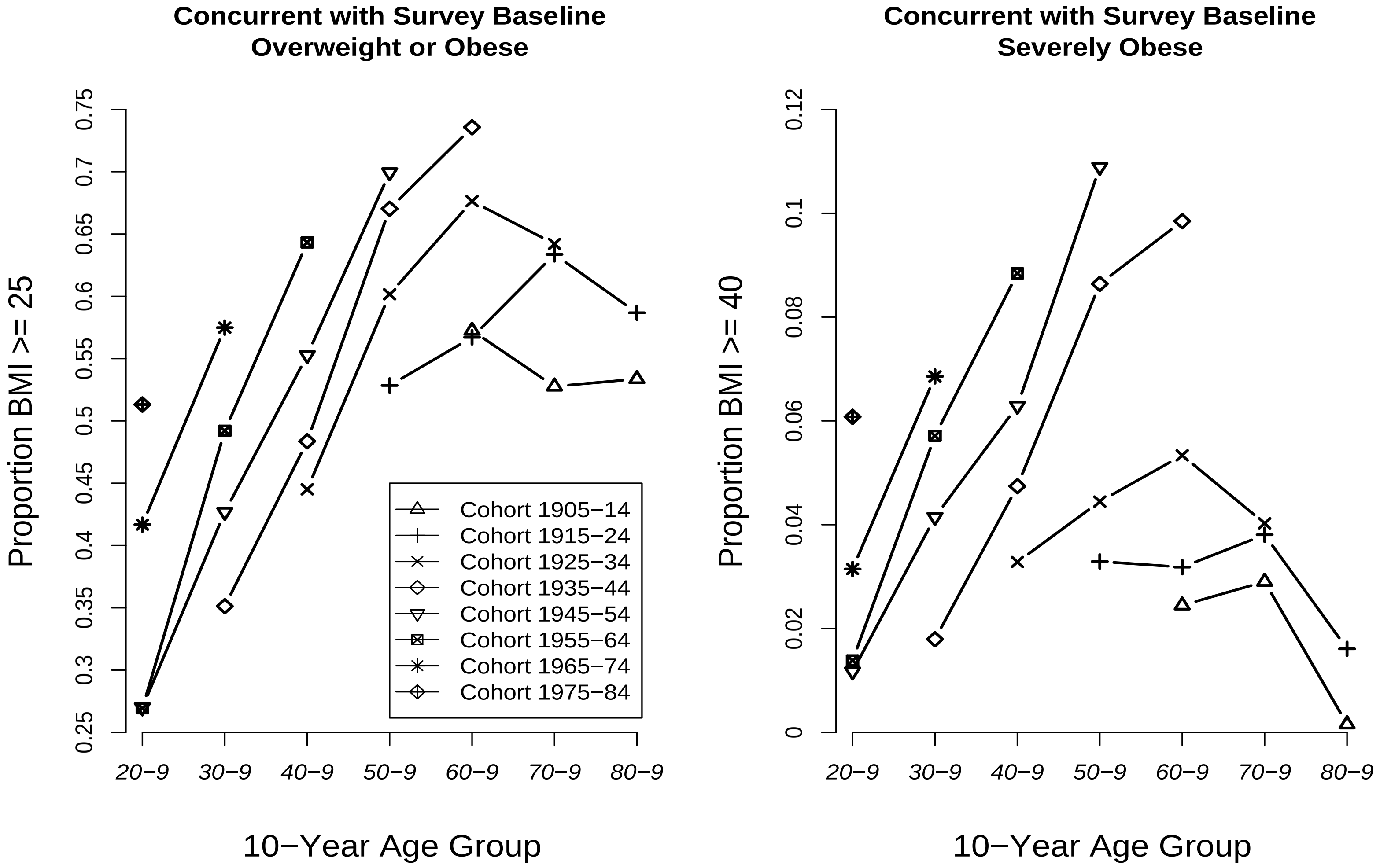


Figure 15: Female Proportion Overweight/Obese, or Severely Obese, NHANES Series (1971-2005), Weighted, by Birth Cohort and Age


Concurrent with Survey Baseline Overweight or Obese

Concurrent with Survey Baseline Severely Obese

Appendices

Table A-1: Parameter Estimates (Standard Errors) for Binomial Logit Models of Age-25 Weight Status, NHANES, Females, Weighted

Parameter	1976-80 NHANES Estimate (S.E.)	1988-94 NHANES Estimate (S.E.)	1999-2004 NHANES Estimate (S.E.)
Intercept	0.9792 (0.3826)	0.8463 (0.3027)	2.2622 (0.3069)
Age	-0.1142 (0.0174)	-0.0983 (0.0129)	-0.1261 (0.0129)
Age Square	0.0011 (0.00018)	0.000889 (0.000124)	0.000973 (0.000126)

Table A-2: Parameter Estimates (Standard Errors), Preferred Gompertz Model $M3.2$ in Table 3, NHANES II and III Mortality Samples, Females, Weighted

Parameter	Estimate (Standard Error)
β Equation (Scale)	
Intercept	-12.1394 (0.3972)
Time Period	
Year 1976-1985 (P1)	ref. (–)
Year 1986-1995 (P2)	-0.8613 (0.4522)
Year 1996-2000 (P3)	-1.0650 (0.4540)
Age-25 Weight Group	
Lean (BMI < 25)	ref. (–)
Overweight or Obese (BMI ≥ 25)	1.2706 (0.3384)
Period × BMI	
BMI $\geq$25 in P2	0.1825 (0.1997)
BMI $\geq$25 in P3	0.4997 (0.2003)
γ Equation (Shape)	
Intercept	0.00662 (0.000502)
Period	
P2	0.00111 (0.000557)
P3	0.00128 (0.000552)
Age-25 Weight	
Overweight or Obese	-0.00140 (0.000378)

Table A-3: Estimated Variance-Covariance Matrix of Selected Parameters, Preferred Gompertz Model $M3.2$ in Table 3, NHANES II and III Mortality Samples, Females, Weighted

Parameter	(1)	(2)	(3)	(4)
β Equation				
(1) Overweight or Obese	0.114547			
(2) BMI $\geq$25 in P2	-0.02004	0.039886		
(3) BMI $\geq$25 in P3	-0.016	0.032161	0.040131	
γ Equation				
(4) Overweight or Obese	-0.00011	-0.00001	-0.00002	1.432E-7

Table A-4: 1976-80 US Female Total Mortality Rates, Age-25 Weight Distribution and Mortality Rates by Age-25 Weight Status, NHANES and Human Mortality Database

Age	Total	Age-25 BMI Distribution		Mortality Ratio	Mortality by BMI	
Group	Mortality	< 25	≥ 25	BMI$\geq$ 25/BMI$<$25	$<$25	$\geq$ 25
25-29	0.00069	0.78605	0.21395	2.24476	0.00055	0.00123
30-34	0.00086	0.82328	0.17672	2.06391	0.00073	0.0015
35-39	0.0013	0.84998	0.15002	1.89762	0.00115	0.00218
40-44	0.00212	0.86768	0.13232	1.74473	0.00193	0.00337
45-49	0.00337	0.87676	0.12324	1.60416	0.00313	0.00503
50-54	0.00517	0.87973	0.12027	1.47491	0.00489	0.00721
55-59	0.0076	0.87699	0.12301	1.35608	0.00728	0.00988
60-64	0.01163	0.87699	0.12301	1.24682	0.01129	0.01407
65-69	0.01684	0.87699	0.12301	1.14637	0.01654	0.01896
70-74	0.02714	0.87699	0.12301	1.05401	0.02696	0.02841
75-79	0.04283	0.87699	0.12301	0.96909	0.043	0.04167
80-84	0.07265	0.87699	0.12301	0.89101	0.07364	0.06561
85-89	0.11918	0.87699	0.12301	0.81922	0.12189	0.09986
90-94	0.18926	0.87699	0.12301	0.75322	0.19519	0.14702
95-99	0.27978	0.87699	0.12301	0.69253	0.29078	0.20137
100-104	0.36987	0.87699	0.12301	0.63674	0.38717	0.24652
105-109	0.36588	0.87699	0.12301	0.58544	0.38554	0.22571
110+	0.29974	0.87699	0.12301	0.53827	0.31779	0.17105

Table A-5: 1988-94 US Female Total Mortality Rates, Age-25 Weight Distribution and Mortality Rates by Age-25 Weight Status, NHANES and Human Mortality Database

Age Group	Total Mortality	Age-25 BMI Distribution		Mortality Ratio	Mortality by BMI	
		< 25	≥ 25	BMI$\geq$ 25/BMI<25	<25	≥ 25
25-29	0.00064	0.76075	0.23925	2.6942	0.00045	0.00122
30-34	0.00085	0.7993	0.2007	2.47713	0.00065	0.00162
35-39	0.00117	0.82745	0.17255	2.27755	0.00096	0.00219
40-44	0.00167	0.8463	0.1537	2.09405	0.00143	0.00299
45-49	0.00263	0.85872	0.14128	1.92534	0.00233	0.00448
50-54	0.00424	0.86505	0.13495	1.77021	0.00384	0.00679
55-59	0.0068	0.86594	0.13406	1.62759	0.00627	0.01021
60-64	0.01063	0.86141	0.13859	1.49646	0.00995	0.01489
65-69	0.01602	0.86141	0.13859	1.37589	0.01523	0.02096
70-74	0.02461	0.86141	0.13859	1.26504	0.02374	0.03003
75-79	0.03831	0.86141	0.13859	1.16311	0.03746	0.04357
80-84	0.063	0.86141	0.13859	1.0694	0.0624	0.06673
85-89	0.10675	0.86141	0.13859	0.98324	0.107	0.1052
90-94	0.17752	0.86141	0.13859	0.90402	0.17991	0.16265
95-99	0.27671	0.86141	0.13859	0.83119	0.28334	0.23551
100-104	0.39394	0.86141	0.13859	0.76422	0.40725	0.31123
105-109	0.48179	0.86141	0.13859	0.70265	0.5025	0.35308
110+	0.46704	0.86141	0.13859	0.64604	0.49113	0.31729

Table A-6: 1999-2004 US Female Total Mortality Rates, Age-25 Weight Distribution and Mortality Rates by Age-25 Weight Status, NHANES and Human Mortality Database

Age Group	Total Mortality	Age-25 BMI Distribution		Mortality Ratio	Mortality by BMI	
		< 25	≥ 25	BMI$\geq$ 25/BMI$<$25	$<$25	$\geq$ 25
25-29	0.00054	0.6055	0.3945	3.69951	0.00026	0.00097
30-34	0.00074	0.68294	0.31706	3.40144	0.00042	0.00142
35-39	0.00115	0.74319	0.25681	3.12739	0.00074	0.00232
40-44	0.00176	0.78754	0.21246	2.87542	0.00126	0.00361
45-49	0.00258	0.81868	0.18132	2.64375	0.00198	0.00525
50-54	0.00378	0.84026	0.15974	2.43075	0.00307	0.00747
55-59	0.0059	0.85282	0.14718	2.23491	0.00499	0.01116
60-64	0.0094	0.85944	0.14056	2.05484	0.00819	0.01682
65-69	0.01466	0.85991	0.14009	1.88929	0.01304	0.02463
70-74	0.02296	0.85435	0.14565	1.73707	0.02074	0.03602
75-79	0.0367	0.85435	0.14565	1.59712	0.03376	0.05392
80-84	0.06116	0.85435	0.14565	1.46844	0.05726	0.08408
85-89	0.1036	0.85435	0.14565	1.35013	0.09857	0.13308
90-94	0.17366	0.85435	0.14565	1.24135	0.16776	0.20825
95-99	0.28096	0.85435	0.14565	1.14134	0.27529	0.3142
100-104	0.41046	0.85435	0.14565	1.04938	0.40753	0.42766
105-109	0.54095	0.85435	0.14565	0.96483	0.54373	0.52461
110+	0.49407	0.85435	0.14565	0.8871	0.50233	0.44562

Table A-7: Mortality Change over Time, Total Population and By Age-25 Weight, US Females, NHANES and Human Mortality Database

Age	1988-1994/1976-1988 Mortality			1999-2004/1988-1994 Mortality		
Group	Total	BMI<25	BMI≥25	Total	BMI<25	BMI≥25
25-29	0.924	0.832	0.999	0.85	0.578	0.794
30-34	0.983	0.901	1.081	0.868	0.639	0.877
35-39	0.9	0.837	1.004	0.977	0.771	1.059
40-44	0.786	0.739	0.887	1.052	0.879	1.207
45-49	0.781	0.742	0.89	0.98	0.854	1.172
50-54	0.82	0.785	0.942	0.891	0.801	1.1
55-59	0.895	0.862	1.034	0.867	0.795	1.092
60-64	0.914	0.881	1.058	0.884	0.823	1.13
65-69	0.952	0.921	1.105	0.915	0.856	1.175
70-74	0.907	0.881	1.057	0.933	0.874	1.199
75-79	0.894	0.871	1.046	0.958	0.901	1.237
80-84	0.867	0.847	1.017	0.971	0.918	1.26
85-89	0.896	0.878	1.054	0.97	0.921	1.265
90-94	0.938	0.922	1.106	0.978	0.932	1.28
95-99	0.989	0.974	1.17	1.015	0.972	1.334
100-104	1.065	1.052	1.262	1.042	1.001	1.374
105-109	1.317	1.303	1.564	1.123	1.082	1.486
110+	1.558	1.545	1.855	1.058	1.023	1.404

Table A-8: Life Expectancy at Age 40, By Age-25 Weight, US Females

Time Period	BMI <25	BMI ≥25
Year 1976-80	39.67	38.71
Year 1988-94	41.13	38.34
Year 1999-04	42.41	36.38

Center for Demography and Ecology
University of Wisconsin
1180 Observatory Drive Rm. 4412
Madison, WI 53706-1393
U.S.A.
608/262-2182
FAX 608/262-8400
comments to:yyu@ssc.wisc.edu
requests to: cdepubs@ssc.wisc.edu