Center for Demography and Ecology
University of Wisconsin-Madison

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Yan Yu

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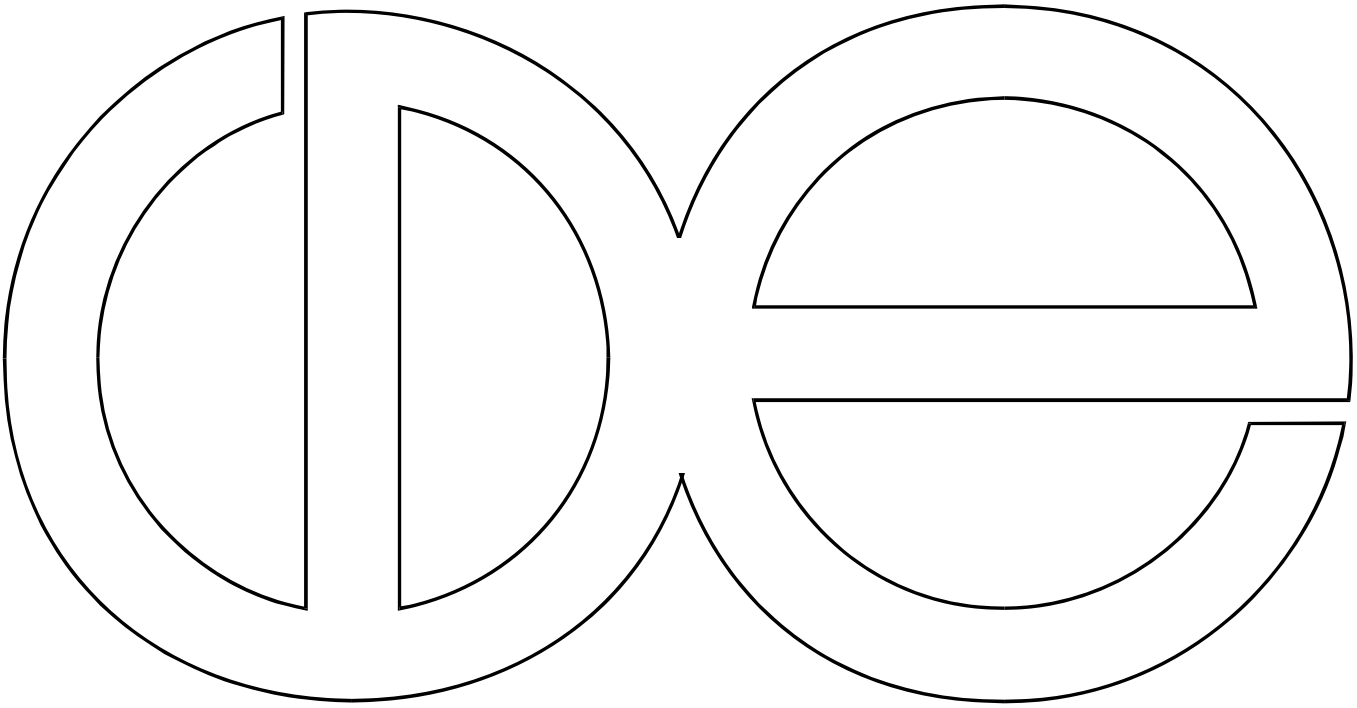

Re-Examining the Age Effect on the Weight-Mortality Relationshhip: Age of Death, Age of Weight and Cohort Effect*

Yan Yu

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

Abstract

The majority of US adults are overweight or obese. Contemporary cohorts have been gaining weight continuously well up to the oldest ages. The negative health consequences of excess body weight have been well-documented. An intriguing question is whether these consequences decline over the life course, and excess body fat is no longer a significant mortality risk factor in old age. Previous studies concluded with a decline over age in the excess mortality of the overweight or obese, and call for weight guidelines that adjust for age. However, most prior research was based on comparing cross-sectional age groups that belong to a multitude of birth cohorts. In addition to the pitfall of cohort distortions, all previous studies used time since baseline as analysis time, leading to an age overlap in the comparison groups that makes it impossible to determine the age to which the weight disparities in mortality pertain. This study adopts an age- and cohort-specific framework to examine the age effect on the weight-mortality relationship for five cohorts born in the first half of the 20th century. To make weight disparities age-specific, age rather than time since baseline is used as analysis time. The analysis finds no decline in the long-term mortality consequences of baseline weight over the age of mortality followup. Mortality differentials tend to be stronger among later cohorts. When cohort differences are ignored, weight disparities in mortality may assume a rising or declining age pattern, and even if the age pattern is not distorted, estimates of mortality differentials may be biased towards nil.

1 Background

Do the negative health consequences of excess body fat vary over age? This is an important question. Age variations in mortality differentials throw light on the process of aging, and point to the potential for improvement of health at increasingly older ages. Empirical research has examined the age effect for a variety of characteristics such as education (Lauderdale 2001), race (Corti et al. 1999) or gender (Vallin 1993). Unlike social demographic characteristics that are relatively fixed in adulthood, body weight changes over the life course. Therefore, the age-effect on the weight-mortality relationship can be conceptualized in two related but different ways. First, age refers to the age of death, and weight at a fixed age may have long-term mortality consequences that are variable over the subsequent ages. Second, age refers to the age of weight measurement. As weight and health are an inter-related dynamic process that unfolds over an individual's life course, the heterogeneities of weight groups with respect to health and their mortality implications may vary depending on the age when weight is classified.

Figure 1 graphs the two aspects for one single cohort born in 1941. Suppose cohort members entered the study and had their weight measured in 1971 when they were aged 30 (Point A in the graph). One interest is how the mortality consequences of weight A vary subsequently from B at age 40 to C at age 50. We refer to this as the long-term age effect, and it focuses on the age of death. Further suppose that cohort survivors were measured again for their weight at B. Another interest is whehter the mortality implications of weight at age A differ from those of weight at age B.

These two age effects share common sources of variations. The social, biological and population process underlying the weight-mortality relationship may affect both the long-term age patterns and the patterns over the age of weight in a similar fashion. Life conditions associated with excess body fat, such as lower socio-economic status, physical inactivity and unhealthy diet, tend to be stable or accumulate over the life course. On the other hand, aging could be accompanied by changes in the biological mechanisms through which body fat has been understood to affect the physique. With declining rates of lipolysis

at older ages, for example, excess fat could turn less harmful (Ostman et al. 1969). Survivors to old age may be selected for good health, perhaps more so in the heavier groups due to their higher early mortality. The selection mechanism, however, should be weak because of low overall mortality before old age in most low-mortality countries. As age unfolds over historical time, progress in medical and health care could reduce or expand the weight disparities, depending for example on differential access to care. These underlying forces may conteract against each other but make the the weight-mortality relationship variable over the long run and over the age of weight measurement in a similar way.

The two age effects are distinguished in how weight change is handled. The long-term age effect ignores the subsequent change of weight, whereas a focus on the age of weight measurement explicitly updates weight classification. As shown in Figure 1, the first perspective uses weight measure at age A only, and the second perspective updates weight A by weight B. This updating is useful in practical settings because it allows for the evaluation of a patient's relative chances of survival based on a more recent weight or a future target weight. Implicitly, the updating should change the heterogeneities of weight groups, which could make the weight-mortality relationship more or less prone to confounding by pre-existing illnesses, leading to an artificial age effect. This is because weight change, weight loss in particular, is more common among adults who are older, in excess weight, in poor health (Losonczy et al. 1995) or likely to die early (Barone et al. 2006). Body weight in old age has been found to predict mortality less well than weight in young or middle ages (Peeters et al. 2003).

An artificial age effect may also arise from measurement error. For reasons not really understood, body composition changes over age, even when weight does not change. As lean mass turns into fat mass, and more fat mass is redistributed to abdominal cavity, Body Mass Index (BMI), the most commonly used measure of body fat, may become less valid. Calculated as weight in kilograms divided by squared height in meters, BMI does not distinguish between lean and fat mass, and assumes that at the same level of height, most of the variability is due to body fat. Although BMI is strongly correlated with fat mass among middle-aged adults, this assumption may be questionable among old adults (Seidell and

Visscher 2000; Willett et al. 1999), making weight less predictive of mortality at old ages.

To sum up, common social and biomedical processes may underlie the variations of weight disparities over the age of mortality followup and over the age of weight. On the other hand, age of weight implies the extent of heterogeneities with respect to health, a source of variation from which the long-term weight diparities are free.

Prior weight research has failed to distinguish the two age effects, and addressed neither issue adequately. To illustrate the research design in previous studies, suppose an analysis uses data from the 1971-75 National Health and Nutrition Examination Survey with mortality followed through 2000, as shown in Figure 2. The typical approach is to divide up the sample into groups based on age at study entry (young, middle-aged and old), and examine how the groups differ in the relatiosnhip between baseline weight and mortality averaged between the baseline and the end of follow-up in 2000. A consistent finding is that the excess mortality of the overweight or obese is higher among the younger than older groups (Bender et al. 1999; Calle et al. 1999; Stevens et al. 1998).

Should this finding be interpreted as a declining age effect? To answer this question, it is necessary to acknowledge that baseline age groups belong to different birth cohorts (born in 1901-50 in the above illustration). Rapid socio-economic and technological improvements of the last century have left among surviving cohorts a large amount of variation in life history. For example, earlier cohorts had more exposure to infections and malnutrition, which could impact the chemical composition and functioning of the vital system; and the physiological adaptation to malnutrition and trauma in early life may change the body weight-survival relationship. As suggested in Fogel (2004), in an infectious environment with a shortage of food supplies, a small body may be optimal for short-run survival but deteriorate at a faster rate in later life and entail a health cost over the long run. Later cohorts, on the other hand, bore the brunt of the obesity epidemic in the late 1980s and the 1990s. They experienced a high prevalence of the metablic syndrome (Ford et al. 2004), an obesity-related constellation of metablic factors that often put individuals at an increased risk of cardiovascular diseases and diabetes, and on multiple-drug treament that are not only costly but also have side effects and require lifelong use (Hu 2005). In a cross-sectional

sample, these cohort differences would show up as differences among age groups.

Acknowledging cohort effect, a few studies followed the same cohort and analyzed how mortality differentials by weight at study baseline vary in the follow-up time, that is, between the entry and end of the study in the NHANES example. The findings are mixed. In the Seven-Country Study for European men aged 40-59 in 1958 and followed through 1985, no temporal variation is found among non-smokers in the mortality ratios of having a BMI of 30 and greater, relative to having a BMI between 18.5 and 25, at 15, 20 and 25 years from the baseline (Visscher et al. 2000). In a sample of California Seventh-Day Adventist white women aged 55-74 in 1960 and followed up for 26 years, the temporal pattern for the mortality of the highest BMI quintile is increasing, when compared with the first BMI quintile, and decreasing when compared with the second BMI quintile (Lindsted and Singh 1997).

Neither type of variations (across cross-sectional age groups or over follow-up time) could be appropriately interpreted as age effect, however. This is because all previous studies used time since baseline as analysis time (i.e., the difference between event calendar year and baseline year in Figure 2), and the age to which the mortality differentials pertain is not clear. In a time-since-baseline framework, when age or cohort groupings are broad and mortality follow-up is long (e.g. over 10 years), age overlap occurs both within and across categories. A late cohort or young age group at the baseline may attain an age in follow-up time that is older than that of an early cohort or old group at the baseline. The same overlap would occur within a cohort or age group. The issue is negligible when the follow-up time is short, for example, five years, but in weight research, it is typical to follow mortality for well over 10 years. Although time since baseline is the standard approach in medical research and appropriate for analyzing treatment effect in clinical trials, it is confusing and ineffective to study age effect in samples with a multitude of birth cohorts.

Despite the conceptual ambiguities and methodological limitations, previous findings have been interpreted to suggest that the negative health consequences of excess weight decline over age, and led to some wide-spread myths about weight management strategies over the life course. It has been suggested that some additional fat may provide a cushion

for old age survival, and weight guidelines should be age-specific, rather than applying to all age groups (Andres et al. 1985; Heiat et al. 2001; Heiat 2003). As representative of cohorts spending the prime of their life in the 1980s and 1990s, American women born in 1931-1940, for example, had their mean BMI increase from 23.5 in their 20s to 29.6 in their 50s, an increase of nearly six BMI units in forty years. This gain, however, is smaller than the 2.25 BMI units per decade which, according to one estimate based on comparing cross-sectional age groups, would incur no additional mortality risk as one grows older (Bender et al. 1999).

The implication that optimum survival can only be achieved through continuous weight gain is not uncontroversial and actually disturbing. As mortality differentials are commonly measured by relative indicators, some researchers pointed out that absolute mortality differences among weight groups actually increase over age, leading to a higher number of deaths due to excess weight at older ages (Stevens 1998). It has also been emphasized that both the quantity and quality of life matter, and well-documented evidence exists for the association between weight gain and the incidence of coronary heart disease, and its risk factors such as Type II diabetes and hypertension (Willett et al. 1999). The combination of evidence would thus imply that one should put on weight and get sicker to live a longer life. Is the road to longevity paved by "a pandemic of mental disorders, chronic diseases and disabilities" (Gruenberg 1977)? Such puzzles deserve further investigation.

The goal of the current study is to distinguish and analyze meaningful age patterns of weight disparities in mortality that are not contaminated by cohort effect. For this purpose, I study five ten-year birth cohorts born in the first half of the 20th century that are all surveyed in the first three cross-sectional samples of the National Health and Nutrition Examination Surveys (NHANES) (respective baselines in 1971-75, 1976-80 and 1988-94), and analyze how weight at the survey baseline affects age-specific mortality through the year 2000 for each birth cohort.

Specifically, I first ask if mortality differentials by baseline weight vary as people pass through subsequent ages up to the year 2000. A grasp of the long-term mortality implications of weight sets the stage for addressing the second question: Do these differentials

vary depending on the age to which body weight pertains? The cross-sectional NHANES series allow the *same* birth cohort to be observed at *different* ages for *different* individuals. For each cohort, respondents in the Third NHANES, for example, are on average older than those in the first two. Taking advantage of this feature, I use survey timing to proxy the age of weight measurement. The interest simplifies to whether weight disparities differ across surveys. Lastly, I explore whether survey/age differences can be explained by weight change history, should that be warranted.

This study improves on prior research in two important ways. First, it adopts an age- and cohort-specific framework that would allow us not only to analyze meaningful age patterns, but also to compare age patterns with and without the influence of cohort differences, and formally examine the role of cohort distortions. Second, it interprets age in two distinct ways, and uses age of weight measurement to explicitly accommodate the feature of weight change, albeit at the population level. Weight group heterogeneity and confounding by pre-exisiting conditions is a perennial problem in weight research. As pre-existing conditions tend to vary over age and cohort, this analytic framework is useful to understand the weight-mortality relationship in general.

It should be pointed out that the age effect studied here or previously (Bender et al. 1999; Calle et al. 1999; Stevens et al. 1998) does not correspond to the age effect in an age-period-cohort analysis. As two of the three quantities (age, period and cohort) determine the third one, a large literature exists to solve the identification problem and analyze the three effects separately (Hobcraft et al. 1982). This paper makes no such attempt but analyzes cohort trajectories as they age over time. Thus, the age effect captures the actual life course experience of individuals in one historical period, rather than being the age effect per se.

2 Data and Methods

Data are drawn from the first three National Health and Nutrition Examination Surveys (NHANES) with respective baselines in 1971-75 (N1), 1976-80 (N2) and 1988-94 (N3) (National Center for Health Statistics 1973, 1978, 1981, 1994), and mortality followed

through December 2000 via a linkage with the National Death Index. Each survey baseline interviewed and examined a probability sample of the US non-institutionalized population. The analysis sample includes five evenly spaced 10-year birth cohorts born between 1901 and 1950: 1901-10 (C1), 1911-20 (C2), 1921-30 (C3), 1931-40 (C4) and 1941-50 (C5). All five cohorts are surveyed in all three surveys.

Figure 3 describes the cohort life-lines in the NHANES, and the cohort classification system used in this paper. The shaded areas refer to the years and ages spanned by survey baseline.[1] The data structure for the three samples appears similar to the one in Figure 2. What differs is the analysis scheme. Under the new design in Figure 3, age (the vertical axis) is the analysis time. This makes it possible to evaluate mortalities and mortality differentials by weight groups that are age-specific. In the old scheme as discussed Figure 2, time since baseline (the horizontal axis) is analysis time, which blurs the age classification system and makes it impossible to determine the ages to which mortality or mortality differentials pertain.

The age range for each cohort can be found in Figure 3, and is also summarized in Table 1 for the N2 and N3 sample. Despite some overlap, each cohort entered the surveys at different ages, and on average, cohort members are older in N3 than N2 (or N1). Note that the three NHANES are cross-sectional, and each survey samples different individuals, even though they may belong to the same birth cohort.

Person-month data are used in the mortality analysis. The statistical analysis uses N2 and N3 confidential mortality data accessed remotely through the Research Data Center (RDC) at the National Center for Health Statistics. Out of confidentiality conerns, the remote access disallows calculating age-specific mortality rates without using a statistical model. To get a sense of the age, cohort and survey patterns of mortality differentials before fitting models, I use two publicly available NHANES mortality files: the complete N3 mortality data, and the N1 mortality data through 1992. The N1 and N2 mortality data

[1]The baseline quadrangles are chipped off at the corners as a result of the NHANES sampling restriction by age: 24-74 in N1 and 30-74 in N2. N3 has no age restriction. However, its publicly available file topcodes age at 90. Although the statistical analysis uses the N3 confidential file, which has accurate age information for all cases, those aged 90 and above are deleted in the analysis sample for comparability with the public file.

through 1992, both released in the public domain, are based on an earlier mortality linkage. As compared with the most recent 2000 linkage, the earlier linkage understates 65 deaths (out of 4669) in N1, and 239 deaths (out of 2384) in N2. The N2 public file, therefore, is not used. The N1 sample is not used in the statistical analysis, due to its lack of baseline information on smoking and weight history.

Body Mass Index (BMI) is calculated as weight in kilograms divided by height in squared meters. Body weight and height are measured through a standard protocol at the baseline. The World Health Organization Guidelines (WHO 2000) are used to define four BMI (weight) groups: Underweight (BMI $<$ 18.5), Normal weight (18.5 $\leq$ BMI $<$ 25), Overweight (25 $\leq$ BMI$<$ 30) and Obese (BMI $\geq$ 30).

Table 2 (males) and Table 3 (females) present summary information for the analysis samples, after deleting 23 cases in N2 and 44 cases in N3 that are missing for BMI measurement. One can note the small number of deaths for the two most recent birth cohorts, in particular in the underweight category with single-digit or zero deaths. As the number of underweight cases is too small to allow for a full-blown analysis of age and cohort patterns of mortality, the analysis focuses on the two excess weight groups. Underweight excess mortality is largely due to manifest or occult diseases that lead to weight loss. A causes-of-death analysis traced the underweight mortality elevation primarily to chronic respiratory conditions (Flegal et al. 2007). In the analysis about how mortality differs by weight at age 25, an age when illness-related weight loss should be minimal, no mortality excess is found for underweight women (Yu 2008).

The person-month data are first used to directly calculate and graph age-specific mortality rates and mortality ratios by weight groups for each cohort and all cohorts combined. Then the statistical analysis uses a discrete version of the parametric Gompertz model, characterized by an exponential increase of mortality over age a:

$$h(a) = exp(\beta + \gamma \cdot a) \tag{1}$$

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

scale and shape of the mortality curve.

Interest centers on how each of the two parameters (β and γ) varies by weight group W, and whether this variation differs across cohort C. In the simple case where both β and γ depend on W, and this dependence does not differ by C, the model becomes:

$$\begin{aligned} \beta &= b_0 + b_1 \cdot W \\ \gamma &= b_2 + b_3 \cdot W. \end{aligned}$$

By way of substitution, the corresponding equation for age-specific mortality $h(a)$ can be expressed as:

$$h(a) = exp(b_0 + b_1 \cdot W + b_2 \cdot a + b_3 \cdot W \cdot a)$$

Thus a weight term in the γ equation is equivalent to an interaction between weight and age ($W \cdot a$) on the mortality outcome $h(a)$, with b_3 capturing age variations in how mortality differs by weight. Following the hierarchical modeling principle, any term in the γ equation (interaction effect) is therefore predicated on its presence in the β equation (main effect). In the exposition, however, the word interaction refers only to terms that interact between cohort and weight, or between cohort, survey and weight, because the actual computing would be modeling β and γ, rather than $h(a)$.

Let W_W denote the β and γ equation in the above example. It follows that interactions between cohort and weight on both β and γ can be denoted as C×W_C×W. Using this notation, six candidate models of interest can be written as:

$M1$: C_C

$M2$: CW_C

$M3$: CW_CW

$M4$: C×W_C×W

$M5$: C×W_CW

$M6$: C×W_C

$M1$ is the baseline model, under which mortality shape (γ) and scale (β) vary by cohort, but

neither by weight status. All other five models include terms for weight. Under the next two models, mortality differs by weight, and weight differentials are age-constant under $M2$ and age-varying under $M3$. The two models share an absence of cohort-weight interaction, thus assuming no cohort differences in how weight affects mortality.

Interaction terms C$\times$W are included in three subsequent models $M4$, $M5$ and $M6$. Of the three, $M6$ is the simplest because C$\times$W is on the β equation only and no W term appears on the γ equation, specifying that the weight effect on mortality is constant over age, but differs across cohorts. Under $M4$ and $M5$, weight effect varies by both age and cohort. Age variations differ across cohort under $M4$, but not under $M5$. So $M4$ is the most complete model.

Each of the six models is referred to as a set, rather than one specific model, because under each specification except $M1$ and $M2$, model variants can be entertained by collapsing cohorts or weight groups in the interaction terms, while maintaining the five-cohort groupings in the main effects. The collapsing is determined empirically by examining estimates in the full model, while giving due consideration to logical ordering.

The analytical strategy is to fit, for each survey sample, all six sets to the data, and select the best variant of each set to be compared across the six to choose the best of all. A comparison of $M4$, $M5$ and $M6$ leads to the best model when cohort effect is accounted for; a comparison of $M2$ and $M3$ chooses a model that ignores cohort effect. Comparing the two chosen models formally tests whether cohorts differ in the weight-mortality association. Model selection is based on the AIC statistic. The one with the smallest AIC is the best model, and those within two units of each other are tied (Burnham and Anderson 2002). When two models are tied, priority is given to no age variation (with fewer or no terms on the γ equation) in order to be conservative about the age patterns of mortality differentials. This decision is made in consideration of the small sample size, and the consequent large fluctuations in age-specific mortality rates, to be shown shortly in the results section.

After modeling the age and cohort patterns of weight disparities separately for each survey sample, I combine the N2 and N3 data to examine survey differences in the weight-mortality relationship, as a proxy for differences arising from age of weight measurement.

Four models are of interest in the combined analysis:

$M7$: Having no survey differences in weight disparities (baseline model);

$M8$: Having survey differences in weight disparities;

$M9$: Having survey differences and accounting for weight change;

$M10$: Accounting for weight change only (deleting survey differences).

Comparing $M7$ and $M8$ addresses the question whether weight disparities differ between N2 and N3. If $M8$, the model incorporating survey differences, is preferred over $M7$, the answer is positive. Given survey differences, comparing $M9$ and $M10$ explores whether survey differences could be explained by weight change history. If $M10$, the more parsimonious model that deletes survey differences, is preferred, then the data would indicate that weight change explains away the variations in mortality differentials over the age of weight measurement. Again, model comparison is based on the *AIC*.

The baseline model in the combined analysis is chosen on the basis of the preceding separate analysis. Given the small number of deaths observed earlier, this exploratory study assumes the same long-term age patterns of weight disparities for N2 and N3. That is, there is no survey variable on the γ equation. As it turns out that the constant age pattern fits the N2 and N3 data reasonably well (that is, $M6$ with no weight terms on γ), I use $M6$ as the baseline and relabel it as $M7$. Interest simplifies to whether survey, weight and cohort interact on the β equation only, and if so, whether this interaction vanishes after accounting for weight change.

Survey timing is a simple and crude approximation to age of weight measurement. Due to long survey durations (five years in N1 and N2 and seven years in N3), for each birth cohort, age of weight measurement varies within a survey, and overlaps across surveys, as shown in Figure 3 and Table 1. Instead of pooling the N2 and N3 sample and dividing up the whole age range in some arbitrary manner, however, this approximation scheme keeps the integrity of each survey and allows for easy interpretation.

For completeness, I replicate previous studies by using the Cox model with time since baseline as analysis time, and analyzing how weight disparities differ among baseline age groups. The replication exercise uses the separate and combined N2 and N3 sample. Three

baseline age strata are classified: under 55, between 55 and 70, and 70 and above. Differences in the weight-mortality relationship across the cross-sectional age groups are captured by interactions between weight groups and age strata in a stratified Cox model.

Throughout this paper, weight groups are defined using weight and height measured at the survey baseline. Weight change history is based on baseline measured weight and weight at age 25 retrospectively self-reported. Using 5% as a cut-off point, weight change is coded into four categories: stable weight, weight loss, weight gain and missing weight change. Since adult weight is correlated with life conditions that affect mortality, all statistical analysis takes into account compositional differences in educational achievement (high school vs. no high school), race (whites vs. non-whites) and baseline smoking status (never, former and current smokers). All covariates appear on both the β and γ equation. Thus, each covariate's association with mortality is allowed to vary over age. All analysis uses sample weights, and is done separately for men and women.

3 Results

As point of departure, Table 4 presents replication results for the male and female samples. As compared with the normal-weight, the relative mortality of obese men, and obese or overweight women (shaded in the table) have a consistent pattern of decline from young to old groups. For example, in the N2 and N3 combined sample, mortality ratio for obese women is 1.77 (95% C.I. 1.43-2.11) in the under-55 age group, 1.23 (1.08-1.38) in the 55-70 age group, and 0.85 (0.71-0.99) for those aged 70 above. In other words, compared to the normal-weight, the obese suffer an 80% mortality elevation among young and middle-aged adults, but enjoy a 15% mortality advantage among the old.

Mortality protection is also notable among overweight men, and overweight women in the older groups, whereas the underweight men or women are subject to elevated mortality at all ages. These results about the weight-mortality relationship in general and its age group differences largely agree with the existent literature (Bender et al. 1999; Calle et al. 1999; Heiat 2003; Stevens et al. 1998). As discussed earlier, since the age groups belong to

various birth cohorts, and overlap in age during the mortality follow-up, these differences in mortality ratios could be distorted by cohort differences, and have no bearing on whether excess fat is more or less harmful at older ages.

The age- and cohort-specific analysis starts with calculating age-specific mortality rates averaged over a three-year interval for each birth cohort, and further within each weight group, using the N1 and N3 sample. These rates are graphed on a logarithmic scale in Figure 4 (males) and Figure 5 (females), and in Figure 6 and Figure 7 by weight group. An overall linear pattern can be observed, and more clearly so in the first two figures, indicating that the exponential assumption of the Gompertz model is reasonable.

These mortality rates produce age-specific mortality ratios for each birth cohort, shown in Figure 8 (males) and Figure 9 (females). The fluctuations of the ratio curves mirror those of the mortality curves: The ratio is zero if no death in the numerator weight group, and goes to infinity if no death in the reference group, which shows up as a gap in the curve. Despite the large fluctuations, the curves appear to be flat, suggesting a constant age pattern and no change in the long-term mortality consequences of body weight.

These age patterns are cohort-specific and free of cohort influences. Excess mortality for the overweight or obese appears to differ across cohorts: stronger in the more recent cohorts, and diminished or reversed in the early cohorts. These cohort differences are particularly strong for females, both in N1 and N3. Ignoring cohort differences leads to a remarkable decline over age in the mortality ratios, as shown in Figure 10. For males, there is little excess mortality for the two heavy groups except for the N1 obese. This could be due to the high prevalence and intensity of smoking across all five male cohorts, suppressing the mortality hazard of excess weight and obliterating cohort differences. Male age patterns when ignoring cohort differences tend to be flat, and clustered around the reference line, as shown in Figure 11.

Note no substantive interpretation is attached to these cohort differences. Instead they are treated as nuisance parameters. Notable cohort differences in weight and health history that could impact the weight-mortality relationship include exposure to infections and mal-nutrition in early life (among early cohorts), and to excess weight gain in mid-life (among

late cohorts). The differences observed here, however, could have been influenced by age effect, since earlier cohorts tend to be older at study entry and weight measurement. An interest in cohort patterns would require data for weight measurement at comparable ages for different cohorts, for example weight at age 25 retrospectively reported in the NHANES. Measured adult weight at the same age for all cohorts would be ideal but have more limited availability. For the purpose of this study, it suffices to analyze age differences within cohort.

Mortality ratios differ between N1 and N3, and the direction of differences varies across cohort or age at study entry. The passage of time/age wipes out or reverses mortality differentials among the three early cohorts, but accentuates overweight or obese excess mortality among the two late cohorts. The decrease among the old adults is consistent with previous findings showing that among those aged 70+, recalled age-50 weight predicts mortality better than current weight at age 70+ (Losonczy et al. 1995). The rise of excess mortality among recent cohorts/young or middle-aged adults is a new phenomenon, but agrees with the analysis about age-25 weight that finds an increase in the late 1990s in overweight/obese excess mortality (Yu 2008).

What could explain these differences between the surveys and the divergence among the cohorts? The last part of the study explores the role of weight change. In progressing from N1 (or N2) to N3, all cohorts age but at different stages of the life course, which may impact the extent and nature of weight change, and its mortality implications. Percentages having lost or gained 5% of weight at age 25 are shown in Table 5 (males) and Table 6 (females), separately for the N2 and N3 sample.[2] Missing for age-25 weight is coded as a separate category, rather than being deleted. After the underweight, the normal-weight is the most likely to have experienced weight loss. Of interest is how this differential among the baseline weight groups changes between N2 and N3. Across the table columns, the absolute differences between the normal-weight and obese in the percentage having lost weight generally expand in N3 for the early three or four cohorts, but change little or narrow for the late cohorts. Note that in the two recent female cohorts, the percentage of

[2]The N1 baseline sample collects no information about weight at age 25, and is not used here.

weight loss among the normal-weight actually decreases from N2 to N3. This is due to the crudeness of the weight change measure. Based on two data points, the measure does not capture the complexity of weight change (e.g., multiple gains and losses, or losses and gains).

Weight gain also varies across the surveys, but in a different way. The N3-N2 differences in 5% gain are small for all weight groups in the three early cohorts, but dramatic in the two late cohorts. For those born in 1941-50, percentage weight gain increases from 28 in N2 to 44 in N3 in the normal-weight men, and from 46 to 75 in the normal-weight women. The corresponding figures for the obese show a much smaller increase, but the relative measure may mask the full extent of weight change, as the obese most likely have a large denominator value.

Table 7 shows the weight composition (percentage of severe obesity) among the obese in N2 and N3. Unlike the earlier cohorts, the latest cohort experienced a large increase in severe obesity (BMI 40 and greater) among those having a BMI 30 and greater: from 3% to 10% for males, and from 12% to 19% for females. This reflects the general weight trend, with the population BMI distribution shifting to the right and a big increase in the part of the population in the extremely heavy categories (Flegal et al. 2002; Sturm 2003). The brunt of the obesity epidemic has been borne by recent cohorts spending the prime of their life in the late 1980s or the 1990s. In the NHANES data as shown in Table 8, percentage obese (or severly obese) is stable across the NHANES for earlier cohorts, but more than doubles from N1 (or N2) to N3 for later cohorts.

Unfortunately, the analysis samples are small. With a single-digit number of deaths, a finer distinction of obesity is not realistic for a mortality analysis. However, this analytical limitation does not mitigate the reality that over age/time, a greater obesity prevalence and a higher excess mortality combine to exacerbate the negative health consequences of obesity at the population level.

Next, I turn to results from the Gompertz model that take into account education, smoking and race in the N2 and N3 sample.

Modeling Age Patterns: Age of Death and Cohort Effect

Goodness-of-fit statistics for selected models is presented in Table 9, where the first column describes the model, the second and third columns give the negative double of the log-likelihood ($-2LogL$) and df, and the last column, the AIC ($= -2LogL + 2df$). In the male N2 and female N3 sample, $M6$ has an AIC value at least two units lower than all other five values, and clearly fits the data better. Thus $M6$ is chosen as the preferred model. Under $M6$, mortality ratios for N2 men and N3 women are age-constant, but vary by cohorts.

For N3 men, the three cohort models are tied with each other based on the AIC: $AIC(M4)$=11176.296; $AIC(M5)$=11176.902; $AIC(M6)$=11176.857. Obese relative mortalities increase over age for the earliest three cohorts under $M4$, and for all five cohorts under $M5$, and are age-constant under $M6$. However, none of the age variations are statistically significant at $\alpha = .05$. The observed ratios in Figure 8 show no obvious age patterns either. Although the variations of the curves are detected by the model as age pattern, they could also be purely sampling fluctuations. Thus, to be conservative, we choose $M6$ as the preferred model for N3 men. For N2 women, the preferred model according to the AIC is $M4$, under which relative mortality increases over age for overweight women in the earliest three cohorts but stays constant for all other cohort-weight combinations.

Figure 12 (males) and Figure 13 (females) present the relative mortalities and 95% confidence intervals for the overweight and obese, as compared with the normal-weight. These mortality ratios and uncertainty intervals are also shown in Table A-1 and Table A-2, calculated from the parameter estimates and standard errors under the preferred models shown in Table A-3, and in Table A-4 in the Appendices. Under the preferred models, a constant age pattern fits all cohort-weight groups except C1-C3 for N2 females. In the three exceptions, overweight relative mortalty exhibits a rising age pattern.[3] These model estimates are consistent with the earlier observations of the N1 and N3 data that do not account for education, race or smoking status. Thus even considering uncertainty in model selection, there is no deline over age in weight disparities in mortality.

[3]For these groups, only the initial age has a ratio estimate that is statistically significant at $\alpha = .05$ (lower than 1), which is almost certainly due to extrapolation rather than being real.

Weight disparities stay constant over age, but differ across cohort. Early cohorts generally have little or no mortality differentials, whereas late cohorts experience excess mortality related to being obese or overweight. The cohort differences are stronger in N3 than N2, and for females than males.

Do cohort differences matter? What age patterns would emerge if cohort differences are ignored? According to the AIC values in Table 9, the preferred model when ignoring cohort distinctions for N2 males and N3 females is $M3$, under which relative mortalities vary over age for at least one weight group, and for N3 males and N2 females, $M2$, under which relative mortalities are age-constant.[4] None of these chosen models are better than the preferred cohort models, according to the *AIC*. This suggests that cohort differences should not be ignored when analyzing the age effect.

Ratio estimates under $M3$ or $M2$ are shown in Figure 14 (males) and Figure 15 (females), contrasted with estimates under the preferred cohort model. Depending on the magnitude and direction of cohort differences, a rising or declining age pattern would appear when cohort differences are ignored. In the N3 female sample, as a result of a distinct and strong pattern of increase from early to late cohorts, the constant age pattern of overweight or obese excess mortality appears to decline over age when cohorts are not differentialed. When age patterns are not distorted (for N2 females and N3 males), the ratio estimates are biased towards no excess mortality.

Noteworthy is that mortality ratio estimates change when cohorts progress from N2 to N3. Take for instance female obese relative mortality shown in Figure 13 and in Table A-1 in the Appendices. The ratio estimate is 0.92 (95% C.I. 0.68-1.16) for C1, and 1.42 (95% C.I. 1.22-1.61) for C2-C5 in N2, and in N3, 0.98 (95% C.I. 0.77-1.18) for C1-C3, 2.2 (95% C.I. 1.55-2.85) for C4, and 3.46 (95% C.I. 2.20-4.72) for C5. Thus, from N2 to N3, mortality differentials stay unchanged or decrease among the three early cohorts, and increase among the two late cohorts. This within-cohort difference between N2 and N3 is also observed for the overweight group and in the male data, albeit to a lesser extent.

[4] In the N3 male and N2 female sample, $M2$ is chosen instead of $M3$, which has an *AIC* value smaller than that of $M2$, but the difference is less than two units.

Modeling Age Patterns: Survey Timing/Age of Weight Measurement

To formally examine survey differences, I pool the N2 and N3 sample, and compare models with and without survey differences in the weight-mortality association. The results from the separate samples indicate that a reasonable baseline model is $M6$, under which mortality ratios are age-constant, but differ among cohorts. I re-label the baseline as $M7$ for the pooled sample, and add survey differences to the baseline to obtain $M8$. According to the AIC values shown in Table 10, $M8$ provides a better fit (Males: $AIC(M8) = 31467.008$, $AIC(M7) = 31483.010$; females: $AIC(M8) = 29439.790$, $AIC(M7) = 29442.947$), indicating that weight disparities in mortality differ between N2 and N3.

Ratio estimates under $M8$ are presented in Table A-5 in the Appendices. Results of the combined and separate analysis are consistent. From N2 to N3, mortality ratios for the obese or overweight decrease among the earliest three cohorts but increase among the latest two cohorts. Note that the 95% confidence intervals of the ratio estimates between the two surveys mostly overlap. These comparisons, however, suffer from the flaws of multiple testing. The global test using the AIC is free of this problem, and suggests a better fit of $M8$ when survey differences are taken into account.

What possible health processes could explain these differences? The last analysis focuses on weight change over the life course. Compared with later cohorts, earlier cohorts entered the N3 sample probably above a critically old age. As shown in Table 1, age of study entry averaged 65 and above for C1-C3, and below 60 for C4-C5. At older ages, people are more likely to experience illness-related weight change, weight loss in particular (Losonczy et al. 1995), possibly increasing the heterogeneities of weight groups and confounding the weight-mortality relationship. Among younger adults, in contrast, the increase over age is stronger for weight gain.

The comparison is over two models with ($M9$) and without ($M10$) survey differences, but both including the four-category weight change measure (stable, loss, gain or missing), evaluated using weight at age 25 and the survey baseline, and a 5% cut-off. The idea is to dertermine whether survey differences in weight disparities persist after taking into account weight change. Based on the AIC, the model with survey differences $M9$ is substan-

tially better than the one deleting survey differences $M10$ (Males: $AIC(M9)$ = 31440.765, $AIC(M10)$ = 31457.803; Females: $AIC(M9)$ = 29418.971, $AIC(M10)$ = 29421.437). Results are similar when we use 10% as the cut-off point, or an absolute measure of weight change. Thus, mortality differentials vary depending on the age of weight measurement, and this age-dependence is not explained away by weight change between age 25 and survey baseline.

4 Summary and Discussion

The majority of US adults are overweight or obese. Contemporary cohorts have been gaining weight continuously well up to the oldest ages (Wang et al. 2007). The negative health consequences of excess body weight have been well-documented (WHO 2000). An intriguing question is whether these consequences decline over the life course, and excess body fat is no longer a significant mortality risk factor in old age. Previous studies concluded with a decline over age in the excess mortality of the overweight or obese (Bender et al. 1999; Calle et al. 1999; Stevens et al. 1998), and call for weight guidelines that adjust for age (Heiat 2003). However, most prior research was based on comparing cross-sectional age groups that belong to a multitude of birth cohorts. In addition to the pitfall of cohort distortions, all previous studies used time since baseline as analysis time, leading to an age overlap in the comparison groups that makes it impossible to determine the age to which the weight disparities in mortality pertain. This study adopts an age- and cohort-specific framework to examine the age effect on the weight-mortality relationship for five cohorts born in the first half of the 20th century (from 1901 to 1950). To make weight disparities age-specific, age rather than time since baseline is used as analysis time.

A further distinction is made between age of death and age of weight measurement to analyze two distinct aspects of age variations: the change of weight disparities over the long-term period of mortality followup subsequent to age at study entry, and the change of disparities over the age of weight measurement. For social-demographic characteristics that are fixed relatively early in life, this distinction is unnecessary, and the two aspects

are one and the same. For body weight, this distinction is crucial to understanding the weight-mortality relationship, because health- or aging-related weight change may affect the heterogeneities and mortality consequences of weight groups.

Analyzing data from the first three National Health and Nutrition Examination Surveys (NHANES) with respective baselines in 1971-75 (N1), 1976-80 (N2) and 1988-94 (N3) and mortality through 2000, I find no decline in the long-term mortality consequences of baseline weight over the age of mortality followup. Except for a few cohort-weight groups, the age pattern is constant. In these few exceptions, the relative mortalities of the overweight or obese appear to rise moderately over age of death. As compared with the normal-weight, substantial age-constant excess mortality (mortality ratio and 95% C.I.) is identified for a series of cohort-weight groups: a) obese men among the four early cohorts in N2 (1.28, 1.11-1.46) and among the two late cohorts in N3 (1.47, 1.08-1.86); b) overweight women among the two late cohorts in N2 (1.65, 1.20-2.10), and in N3, among those born in 1921-40 (1.29, 0.98-1.59) and among the last cohort (3.19, 1.92-4.46); and c) obese women among the four late cohorts in N2 (1.42, 1.22-1.61), and in N3, among the fourth cohort (2.22, 1.55-2.85) and the fifth cohort (3.46, 2.20-4.72).

Cohorts differ in how mortality varies by body weight. Mortality differentials tend to be stronger among later cohorts. Since cohorts enter the surveys at different ages, and cohort differences could be distorted by age effects, no substantive interpretation is attached to these patterns. Instead, they are treated as nuisance parameters, with a focus on how they distort age patterns. When cohort differences are ignored, weight disparities in mortality may assume a rising or declining age pattern, and even if the age pattern is not distorted, estimates of mortality differentials may be biased towards nil.

Despite the lack of change over age of death, weight disparities vary over the age of weight. With survey timing (N1 or N2 vs. N3) as a proxy for the age of weight measurement, across surveys, overweight or obese excess mortality tends to diminish or reverse direction among the three early cohorts, who on average enter the N3 at age 60 or above, but increase among the two late cohorts, who enter the N3 before age 60.

To explore whether these variations could be explained by weight history, the last part

of the analysis accounts for weight change between age 25 and the survey baseline. Unfortunately, the crude measure based on two weight data points fails to provide an adequate explanation for the variations over the age of weight measurement. As birth cohorts also enter the NHANES at different time periods, one may wonder whether these results could be explained by advances in medical technology and health care that could have reduced or eliminated weight-related health disparities (Flegal et al. 2005). The support for this convergence hypothesis is not strong. If it was the case, we would be left with the question of why the overweight/obese young or middle-aged adults have been left out.

Younger adults' increase in excess mortality from N1 (or N2) to N3 are consistent with the results in the analysis on age-25 weight that finds a significant increase in the late 1990s in the excess mortality of the overweight or obese based on age-25 weight (Yu 2008). In connection with the well-documented weight gain among contemporary cohorts (e.g., 6 BMI units from between ages in the 20s to 50s for American women born in 1931-40), it appears that technological progress has failed to counterbalance the extended and intensified exposure to excess fat such that an increasingly large percentage of recent cohorts may have fared relatively worse as they pass the prime of their life.

The decline of weight disparities among old adults are consistent with previous findings based on comparing the mortality implications of concurrent and retrospective weight for the elderly (Losonczy et al. 1995). A likely explanation is the increasing heterogeneities of weight groups and measurement error that make BMI a less valid measure of body fat and less predictive of mortality. Although body fat could be less harmful at old age (Ostman et al. 1969), theoretically, this mechanism also affects the long-term mortality effect of excess fat. Given the finding of no decline over the age of death, biological mechanisms appear to at most balance out other social or population processes such as the accumulation of disadvantages, mortality selection and overall historical health improvement. Current evidence, therefore, is not sufficient to support old age as a license for not managing weight or weight gain. Measurement issues would continue to be important in future research on the elderly.

The findings are not directly comparable to prior research. Previous studies found that

the relative mortalities of the overweight or obese decline over cross-sectional age groups that belong to a multitude of birth cohorts (Bender et al. 1999; Calle et al. 1999; Stevens et al. 1998), a result that is replicated with the NHANES data here. Under this typical approach of comparing different ages and different cohorts, age effect could be confounded by cohort differences in the weight-mortality relationship. Additionally, the prior approach used time since baseline as analysis time, rather than using age as done here to make age-specific comparisons. Under the time-since-baseline framework, it is impossible to determine the age to which mortality differentials pertain. In spite of the lack of comparability, the new findings suggest that the declining pattern that has been referred to as age effect in the literature largely results from cohort distortions and the greater heterogeneities of weight groups at older ages.

That weight differentials in mortality are constant over age of mortality followup, and that among young or middle-aged adults, excess overweight or obese mortality increases over age of weight measurement are important findings to understand contemporary mortality patterns. Recent studies found that in the 1990s, county-level life expectancy at birth declined for US women in 180 counties (Ezzati et al. 2008), and as compared with Japan and France, the rise in female life expectancy at age 65 slowed down (Meslé and Vallin 2006). The study adds to this growing body of evidence and points to excess weight as the risk factor underlying the population trends. At the same time, it sheds light on the potential for further mortality improvement. If weight control can be implemented effectively at a sufficiently early age, lasting mortality reduction could be achieved up to the oldest ages. As suggested by Hill et al. (2003), small changes in behavior or in the environment that facilitates such behavioral changes could have a large impact. This may take the form of walking 15 to 20 minutes for a mile, or having three bites fewer of a typical premium fast-food restaurant burger, and doing it everyday.

The study has limitations. The age- and cohort-specific analysis requires a large sample with sufficient death events. The small number of deaths in recent cohorts for some weight groups, especially in the 1988-94 NHANES data, creates large uncertainty in the estimates, and in some cases, in model selection. Although consistent age, cohort and survey patterns

are observed both in the raw data and statistical analysis, the estimation and selection of models would benefit from larger samples. When the NCHS implements a new round of mortality linkage with the National Death Index, we would have the chance to observe and analyze mortality for a longer period of time. If sample size allows, a more refined classification of obesity would be informative.

The cross-sectional NHANES series makes it possible to track the same cohort at different time periods and different ages for different individuals. This approach avoids the issue of loss to follow-up, proxy reporting, or change of study mode (e.g. from measuring to respondent reporting weight) that tend to plague longitudinal data. However, longitudinal data follow the same individuals, and track changes in individuals' weight or health conditions prospectively. This information would be useful to understand the health processes underlying the changes in mortality differentials at different ages. Alternative measures of body fat would also be worthwhile. Measurement issues would continue to be important topics in the research for the elderly.

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Table 1: Age at Study Entry and Exit, 1976-80 and 1988-94 NHANES, Mortality through 2000

	1976-80 NHANES (N2)				1988-94 NHANES (N3)			
	Entry			Exit	Entry			Exit
Birth Cohort	Min	Max	Mean	Max	Min	Max	Mean	Max
C1: 1901-10	65	75	70	99	78	89	83	99
C2: 1911-20	55	69	62	89	68	83	74	89
C3: 1921-30	45	58	51	79	57	73	65	79
C4: 1931-40	35	49	41	69	48	63	55	69
C5: 1941-50	30	38	32	59	38	53	44	59

Table 2: Male Sample Size, 1976-80 and 1988-94 NHANES, Mortality through 2000

Birth Cohort and Baseline Weight	1976-80 NHANES (N2) N	Deaths	Person-months	1988-94 NHANES (N3) N	Deaths	Person-months
C1: 1901-10						
Underweight	35	35	3312	30	28	1114
Normal-weight	394	353	53160	242	202	15188
Overweight	366	327	55350	232	176	15511
Obese	121	112	15866	44	38	2984
C2: 1911-20						
Underweight	23	18	3711	17	13	1164
Normal-weight	543	369	99156	331	175	26627
Overweight	615	400	121162	369	188	31471
Obese	196	135	36134	152	70	13459
C3: 1921-30						
Underweight	8	6	1473	19	16	1023
Normal-weight	277	108	64294	329	107	30575
Overweight	302	113	72692	510	121	49664
Obese	106	49	24040	294	67	29282
C4: 1931-40						
Underweight	7	2	1835	8	1	861
Normal-weight	278	57	70933	269	41	27902
Overweight	325	46	84939	395	44	41784
Obese	107	26	27356	249	29	25839
C5: 1941-50						
Underweight	12	1	3088	5	1	519
Normal-weight	314	38	81549	358	31	38706
Overweight	244	20	64854	477	20	52409
Obese	75	6	19774	301	20	32983
Total	4348	2220	904677	4631	1388	439065

Table 3: Female Sample Size, 1976-80 and 1988-94 NHANES, Mortality through 2000

Birth Cohort and Baseline Weight	1976-80 NHANES (N2) N	Deaths	Person-months	1988-94 NHANES (N3) N	Deaths	Person-months
C1: 1901-10						
Underweight	28	25	3685	32	29	2104
Normal-weight	427	339	75387	246	172	18114
Overweight	391	292	74168	216	143	17266
Obese	229	168	41534	85	56	6558
C2: 1911-20						
Underweight	43	32	8005	26	19	1587
Normal-weight	605	293	136183	325	128	29125
Overweight	504	253	111148	339	114	31918
Obese	347	201	73557	266	101	23228
C3: 1921-30						
Underweight	23	8	5145	24	11	2115
Normal-weight	348	83	86889	341	60	35321
Overweight	240	45	62165	428	89	42040
Obese	174	58	41816	409	67	41306
C4: 1931-40						
Underweight	31	5	7780	22	5	2184
Normal-weight	412	39	108890	279	22	29128
Overweight	193	26	50489	322	27	33656
Obese	154	21	40508	386	43	38777
C5: 1941-50						
Underweight	32	2	8500	19	0	2086
Normal-weight	426	12	114384	380	10	41914
Overweight	146	11	38729	381	18	41046
Obese	125	8	33123	505	25	53544
Total	4878	1921	1122085	5031	1139	493017

Table 4: Replicating the Comparison of Baseline Age Groups, 1976-80 and 1988-94 NHANES (N2 and N3), Mortality through 2000*

Baseline Weight and Age Group	Males		Females	
	Mortality Ratio	95% C.I.	Mortality Ratio	95% C.I.
Normal-weight	1	–	1	–
Underweight				
N2 + N3				
Age $\leq$ 55	1.5	0.68-2.31	1.72	1.05-2.38
Age [55, 70)	1.55	1.19-1.91	1.77	1.4-2.13
Age 70+	1.87	1.51-2.24	2.06	1.68-2.44
N2 Sample				
Age $\leq$ 55	1.41	0.51-2.31	1.62	0.91-2.32
Age [55, 70)	1.25	0.86-1.63	1.57	1.18-1.97
Age 70+	1.62	1.02-2.23	1.66	0.98-2.33
N3 Sample				
Age $\leq$ 55	2.51	0.26-4.75	2.52	0.58-4.45
Age [55, 70)	2.92	1.99-3.85	2.76	1.79-3.73
Age 70+	2.1	1.63-2.56	2.18	1.72-2.64
Overweight				
N2 + N3				
Age $\leq$ 55	0.8	0.63-0.98	1.42	1.11-1.73
Age [55, 70)	0.89	0.79-1	1.01	0.88-1.14
Age 70+	0.89	0.78-1	0.89	0.77-1.01
N2 Sample				
Age $\leq$ 55	0.86	0.65-1.06	1.28	0.94-1.62
Age [55, 70)	0.96	0.84-1.07	0.99	0.85-1.13
Age 70+	0.91	0.72-1.1	0.97	0.78-1.17
N3 Sample				
Age $\leq$ 55	0.56	0.23-0.89	1.7	0.96-2.45
Age [55, 70)	0.72	0.52-0.92	1.09	0.76-1.42
Age 70+	0.86	0.72-0.99	0.83	0.68-0.98
Obese				
N2 + N3				
Age $\leq$ 55	1.24	0.97-1.5	1.77	1.43-2.11
Age [55, 70)	0.95	0.82-1.09	1.23	1.08-1.38
Age 70+	0.87	0.71-1.02	0.85	0.71-0.99
N2 Sample				
Age $\leq$ 55	1.36	1.04-1.69	1.72	1.33-2.11
Age [55, 70)	1.11	0.94-1.29	1.27	1.1-1.44
Age 70+	1.14	0.84-1.45	0.89	0.65-1.12
N3 Sample				
Age $\leq$ 55	0.76	0.36-1.17	1.63	0.93-2.33
Age [55, 70)	0.69	0.45-0.92	1.06	0.73-1.39
Age 70+	0.73	0.55-0.9	0.8	0.62-0.97

* Sample weights are used; all models include covariates for education, race and smoking status.

Table 5: Percentage Having Changed 5% of Age-25 Weight at the Survey Baseline, 1976-80 and 1988-94 NHANES Males, Weighted

Birth Cohort and	1976-80 NHANES (N2)				1988-94 NHANES (N3)			
Baseline Weight	Missing	Stable	Loss	Gain	Missing	Stable	Loss	Gain
C1: 1901-10								
Underweight	6.9	19.1	72.1	1.9	22.5	0	77.5	0
Normal-weight	8.4	29.2	33.6	28.8	13.7	23.1	41.4	21.8
Overweight	7.4	16.1	5.2	71.3	11.3	18.6	11.5	58.6
Obese	5.5	3.8	2.6	88.1	3.2	3.3	11.5	81.9
C2: 1911-20								
Underweight	4.5	0	95.5	0	1.5	19	79.5	0
Normal-weight	5.3	28	28.8	37.9	10.1	29.2	28.9	31.8
Overweight	5	13	4.7	77.3	5.1	15.6	4.9	74.4
Obese	6.7	5.7	1.7	85.9	3.9	8.4	1.2	86.5
C3: 1921-30								
Underweight	9.3	22.5	58.2	10	4.7	4.7	90.5	0
Normal-weight	3.8	33.3	25.7	37.1	5.8	30.6	31.2	32.4
Overweight	4.8	21.5	5.1	68.6	3.2	12.8	7.1	76.9
Obese	2.4	4	1.2	92.4	2.7	6.1	0.5	90.7
C4: 1931-40								
Underweight	0	17.6	82.4	0	7.3	25.1	67.6	0
Normal-weight	2.5	41.9	18.7	36.9	1.6	31.4	25.5	41.5
Overweight	3.5	24.5	6.5	65.6	3.1	17.3	7.7	71.8
Obese	0.8	15.3	4	79.9	0.7	4.6	1.7	93
C5: 1941-50								
Underweight	0	29.2	65.9	4.8	20.6	27.5	29.6	22.4
Normal-weight	1.8	54.4	15.9	27.9	3.3	36	16.4	44.3
Overweight	1.9	30.1	11.5	56.4	2.3	13.4	6.3	78
Obese	0.8	18.7	5	75.5	4.7	10.1	3.8	81.4

Table 6: Percentage Having Changed 5% of Age-25 Weight at the Survey Baseline, 1976-80 and 1988-94 NHANES Females, Weighted

Birth Cohort and Baseline Weight	1976-80 NHANES (N2) Missing	Stable	Loss	Gain	1988-94 NHANES (N3) Missing	Stable	Loss	Gain
C1: 1901-10								
Underweight	3.8	5.6	71.8	18.8	11.8	9.2	79	0
Normal-weight	6.3	25.8	18.4	49.6	14.1	24.7	29.4	31.8
Overweight	8.1	7.7	2.8	81.4	13.7	7.9	4.1	74.2
Obese	10.4	3.5	1.5	84.6	14	2.3	3.8	79.9
C2: 1911-20								
Underweight	13.7	27	57.9	1.3	8.1	10.1	81.7	0
Normal-weight	4.3	27.2	10.9	57.5	7.1	24.2	19.3	49.4
Overweight	6.1	6.1	3.5	84.3	8.1	6.6	2.4	82.9
Obese	9.2	3.2	1.5	86	10.3	1.5	3	85.3
C3: 1921-30								
Underweight	0	12.4	71.9	15.7	1.5	11.9	86.7	0
Normal-weight	3.4	23.8	11.1	61.7	4.9	24	12.9	58.2
Overweight	2.2	5.2	3.3	89.3	6.8	3.2	3.1	87
Obese	7	2.5	2	88.5	8.5	1.3	1.2	89
C4: 1931-40								
Underweight	5.1	48.4	27.8	18.7	7.9	17.4	72.7	2
Normal-weight	3.3	24.5	9.7	62.5	3.7	19.2	4.7	72.4
Overweight	3.2	9.1	1.5	86.2	3.4	4.2	2.2	90.1
Obese	4.7	2.1	0.5	92.6	4.7	1.7	0.6	93
C5: 1941-50								
Underweight	0	49.5	36	14.5	2	54.5	22.8	20.7
Normal-weight	1.2	40.6	12.2	46	1.2	17.9	6.4	74.5
Overweight	2.7	10	6.4	80.9	3.9	1	0.5	94.6
Obese	5.7	9	2.2	83.2	5.9	3.9	4	86.1

Table 7: Percentage of Severe Obesity (BMI 40 and Greater) among the Obese (BMI 30 and Greater) at the Baseline, 1976-80 and 1988-94 NHANES (N2 and N3), Weighted

	Males		Females	
Birth Cohort	N2	N3	N2	N3
C1: 1901-10	0.8	3.4	10.5	4.3
C2: 1911-20	3.7	1.3	10.9	13.2
C3: 1921-30	2.8	3.4	14.3	13.2
C4: 1931-40	4.2	7.2	15.3	16.2
C5: 1941-50	2.8	10.1	11.5	18.8

Table 8: Percentage of Obesity (BMI 30 and greater) or Severe Obesity (BMI 40 and Greater), 1971-75, 1976-80 and 1988-94 NHANES (N1, N2 and N3), Weighted

	Males			Females		
Birth Cohort	N1	N2	N3	N1	N2	N3
Obesity						
C1: 1901-10	10.8	12.8	9.8	24.8	19.6	15.6
C2: 1911-20	15.5	14.1	18.4	24.7	23.0	25.6
C3: 1921-30	15.7	15.0	27.7	17.7	21.0	31.0
C4: 1931-40	13.2	14.5	27.3	15.2	17.9	33.8
C5: 1941-50	10.6	11.0	22.0	11.7	16.0	28.6
Severe Obesity						
C1: 1901-10	0.1	0.1	0.3	1.4	2.1	0.7
C2: 1911-20	0.9	0.5	0.2	2.4	2.5	3.4
C3: 1921-30	0.7	0.4	1.0	2.5	3.0	4.1
C4: 1931-40	0.6	0.6	2.0	2.5	2.7	5.5
C5: 1941-50	1.1	0.3	2.2	1.6	1.8	5.4

Table 9: Separate Analysis of 1976-80 and 1988-94 NHANES (N2 and N3), Goodness of Fit Statistics for Selected Gompertz Models*

Model Description	$-2LogL$	df	AIC
Male N2 Sample			
M1: C_C	20438.332	18	20474.332
M2: CW_C	20420.714	21	20462.714
M3: CW_CW	20414.548	22	20458.548
M4: CxW_CxW	20400.557	28	20456.557
M5: CxW_CW	20401.689	26	20453.689
M6: CxW_C	20403.503	24	20451.503
Male N3 Sample			
M1: C_C	11174.443	18	11210.443
M2: CW_C	11145.741	21	11187.741
M3: CW_CW	11142.056	22	11186.056
M4: CxW_CxW	11128.296	24	11176.296
M5: CxW_CW	11128.902	24	11176.902
M6: CxW_C	11130.857	23	11176.857
Female N2 Sample			
M1: C_C	18650.267	18	18686.267
M2: CW_C	18632.317	21	18674.317
M3: CW_CW	18628.986	22	18672.986
M4: CxW_CxW	18605.815	25	18655.815
M5: CxW_CW	18611.595	24	18659.595
M6: CxW_C	18616.070	23	18662.070
Female N3 sample			
M1: C_C	10930.719	18	10966.719
M2: CW_C	10909.853	21	10951.853
M3: CW_CW	10892.705	23	10938.705
M4: CxW_CxW	10879.374	28	10935.374
M5: CxW_CW	10881.201	27	10935.201
M6: CxW_C	10881.250	25	10931.250

* Sample weights are used; models include covariates for education, race and smoking; preferred models are in **bold**.

Table 10: Combined Analysis of 1976-80 and 1988-94 NHANES, Goodness of Fit Statistics for Selected Gompertz Models*

Model Description	$-2LogL$	df	AIC
Males			
M7: M6 (No Survey Difference)	31419.010	32	31483.010
M8: M6, Survey Difference	31397.008	35	31467.008
M9: M6, Survey Difference, Weight Change	31358.765	41	31440.765
M10: M6, Weight Change (No Survey Difference)	31387.803	35	31457.803
Females			
M7: M6 (No Survey Difference)	29380.947	31	29442.947
M8: M6, Survey Difference	29373.790	33	29439.790
M9: M6, Survey Difference, Weight Change	29340.971	39	29418.971
M10: M6, Weight Change (No Survey Difference)	29347.437	37	29421.437

* Sample weights are used; models include covariates for education, race and smoking; two model comparisons are made: $M7$ vs. $M8$ and $M9$ vs. $M10$; preferred models are in **bold**.

Figure 1: Two Age Effects

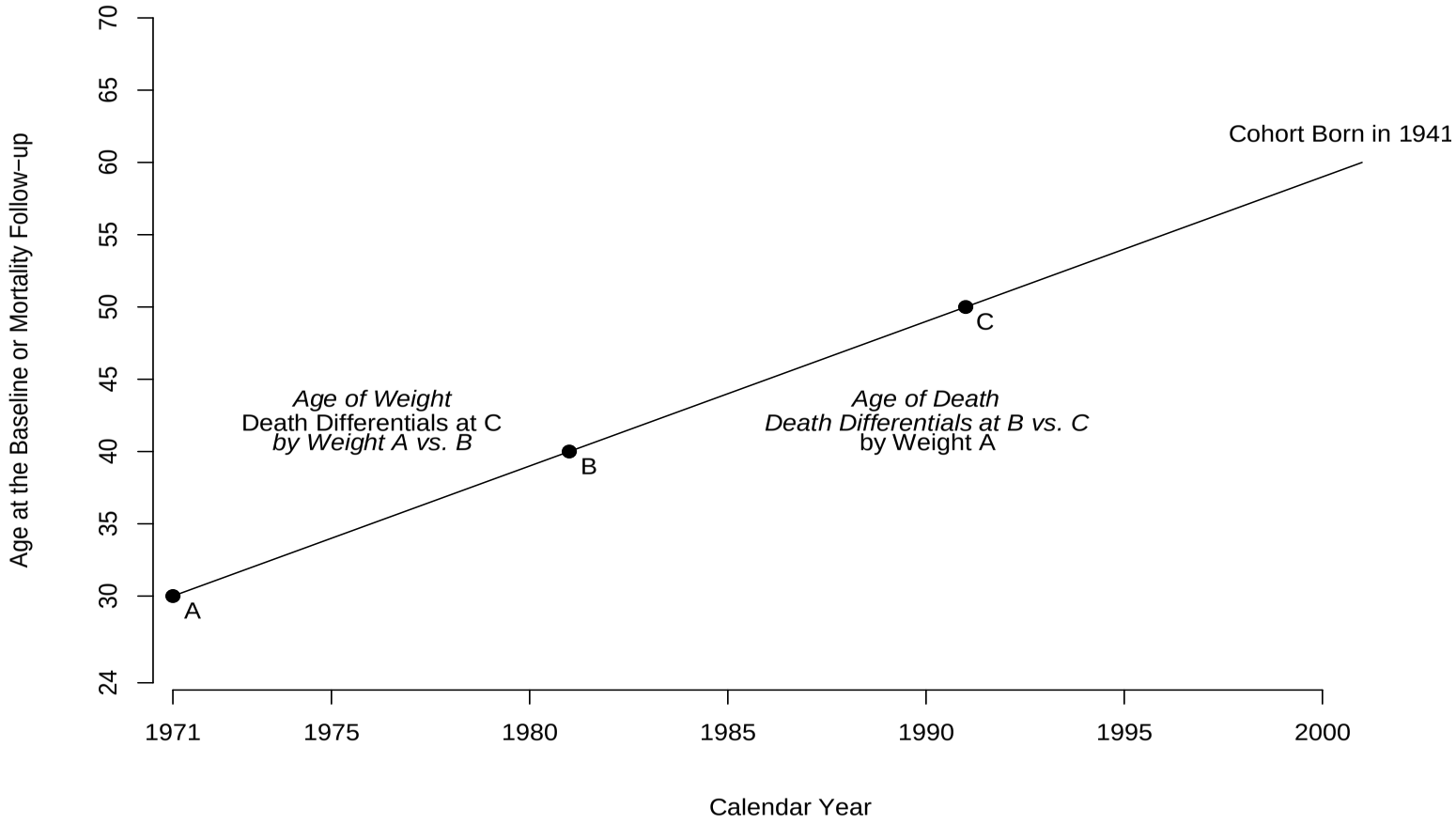


Figure 2: Comparing Cross-Sectional Age Groups

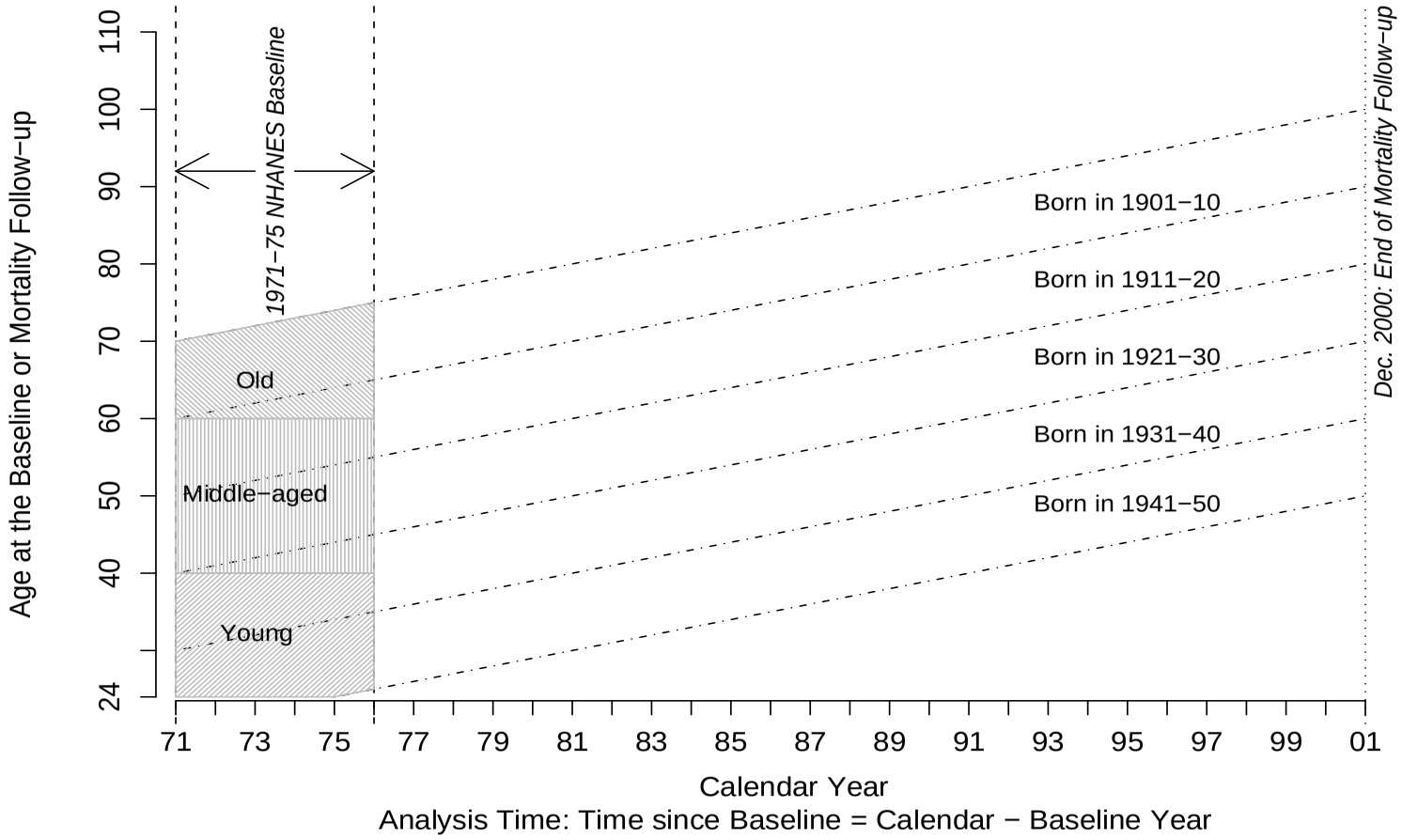


Figure 3: Age and Cohort Classifications of Analysis Samples, NHANES

Figure 4: Observed Age-specific Mortality Rates on the Logarithmic Scale, By Cohort, 1971-75 NHANES through 1992 (N1) and 1988-94 NHANES through 2000 (N3), Males, Weighted

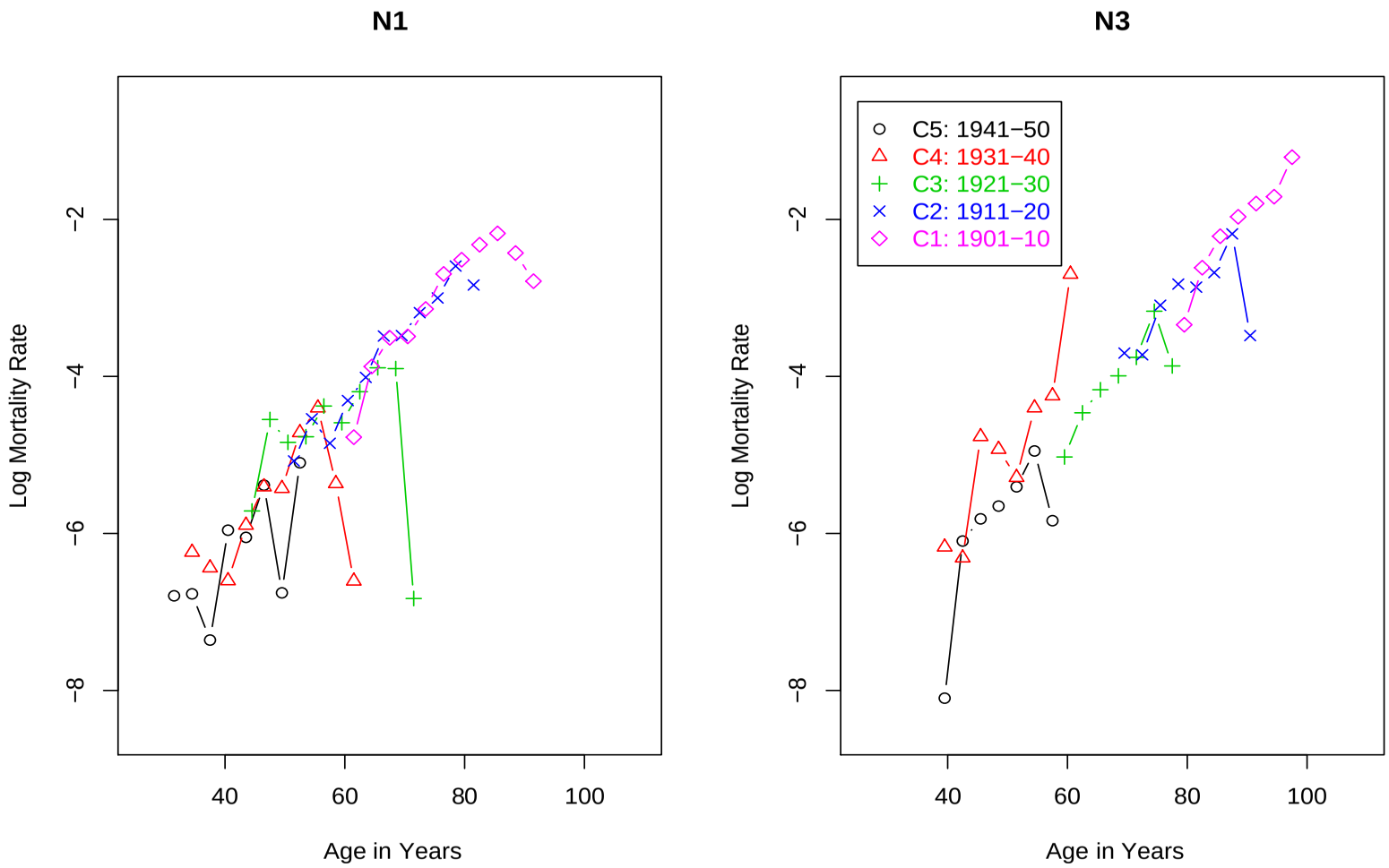


Figure 5: Observed Age-specific Mortality Rates on the Logarithmic Scale, By Cohort, 1971-75 NHANES through 1992 (N1) and 1988-94 NHANES through 2000 (N3), Females, Weighted

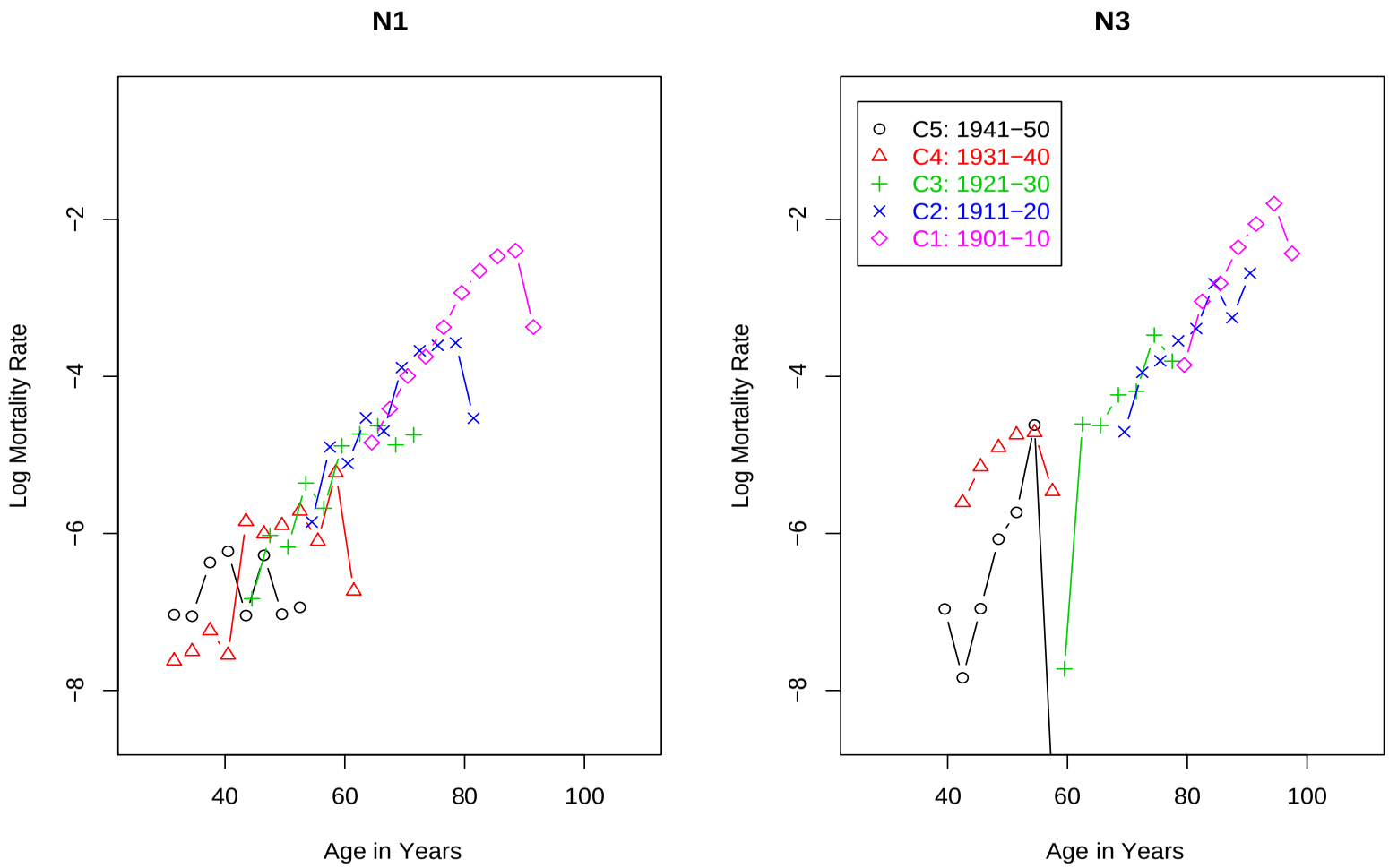


Figure 6: Observed Age-specific Mortality Rates on the Logarithmic Scale, By Cohort and Baseline Weight, 1971-75 NHANES through 1992 (N1) and 1988-94 NHANES through 2000 (N3), Males, Weighted

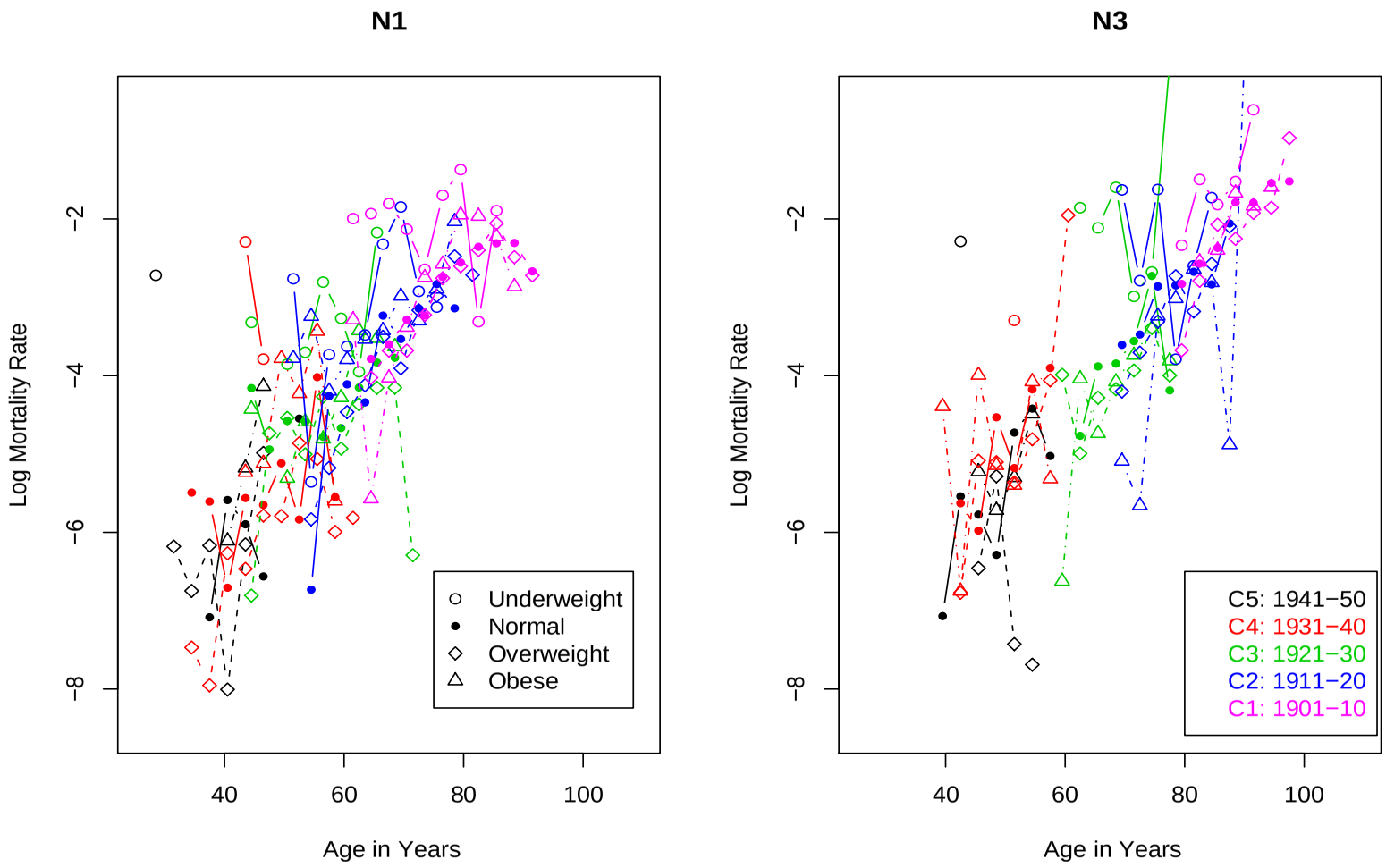


Figure 7: Observed Age-specific Mortality Rates on the Logarithmic Scale, By Cohort and Weight Group, 1971-75 NHANES through 1992 (N1) and 1988-94 NHANES through 2000 (N3), Females, Weighted

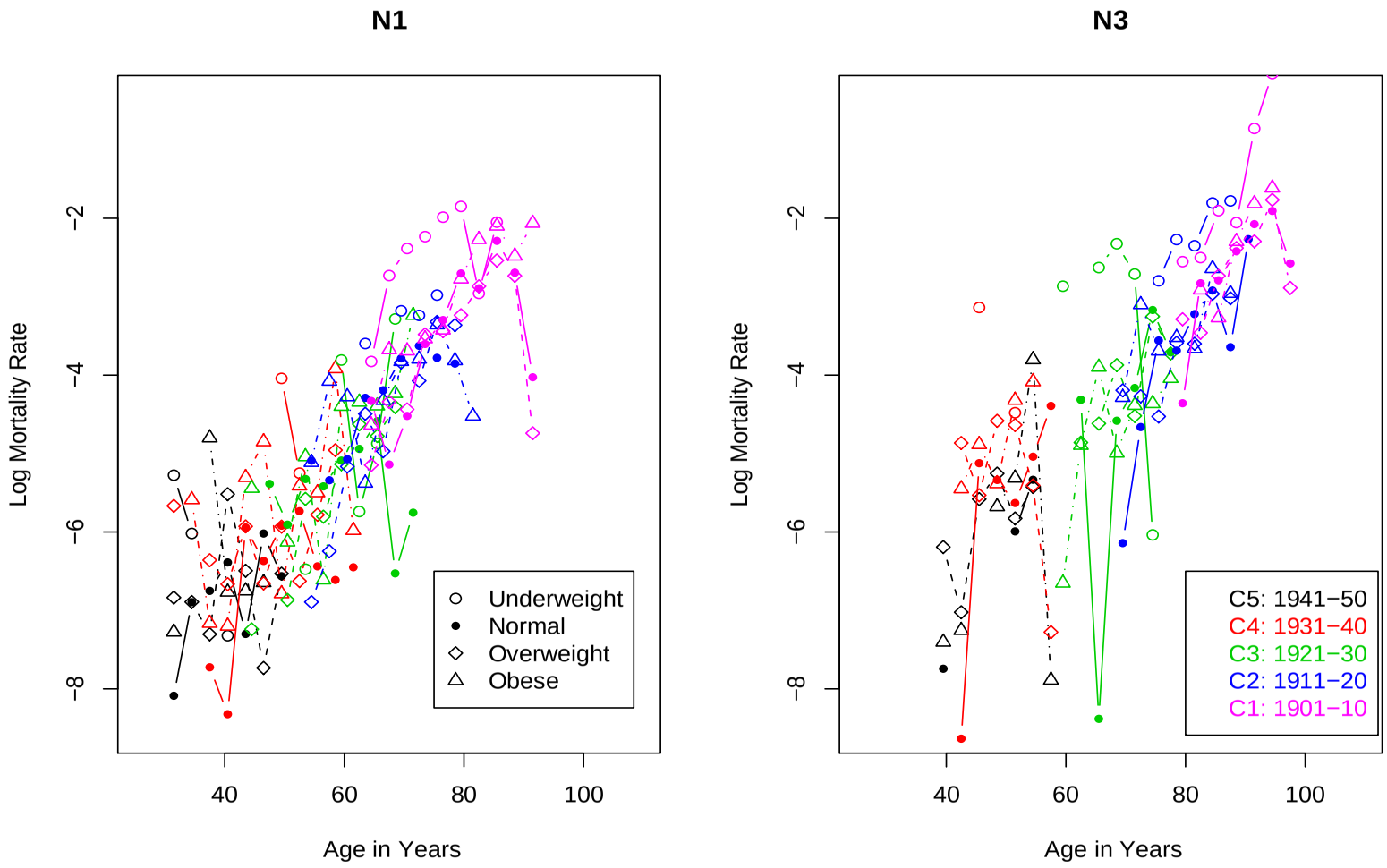


Figure 8: Observed Age-specific Mortality Ratios, By Cohort, 1971-75 NHANES through 1992 (N1) and 1988-94 NHANES through 2000 (N3), Males, Weighted

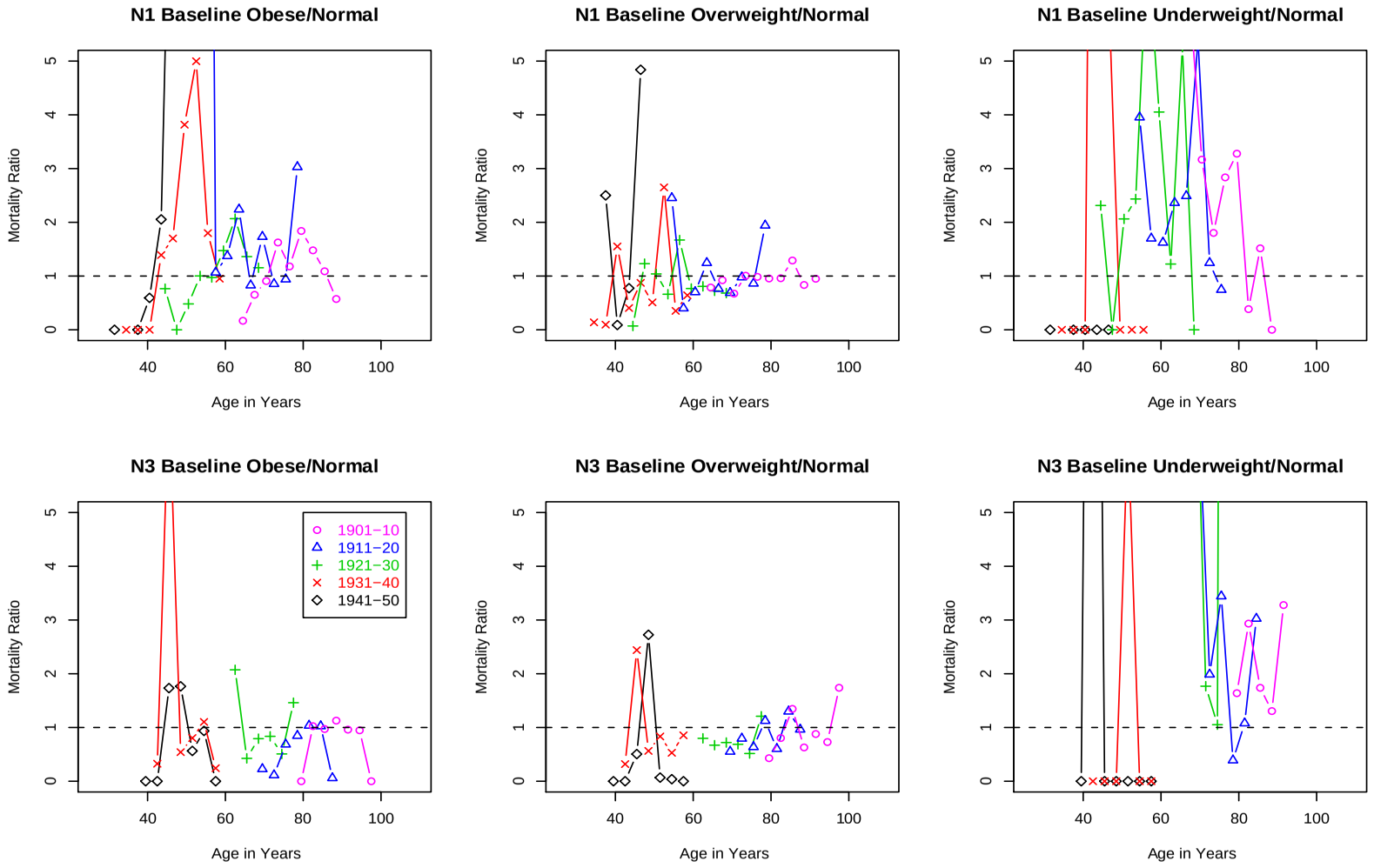


Figure 9: Observed Age-specific Mortality Ratios, By Cohort, 1971-75 NHANES through 1992 (N1) and 1988-94 NHANES through 2000 (N3), Females, Weighted

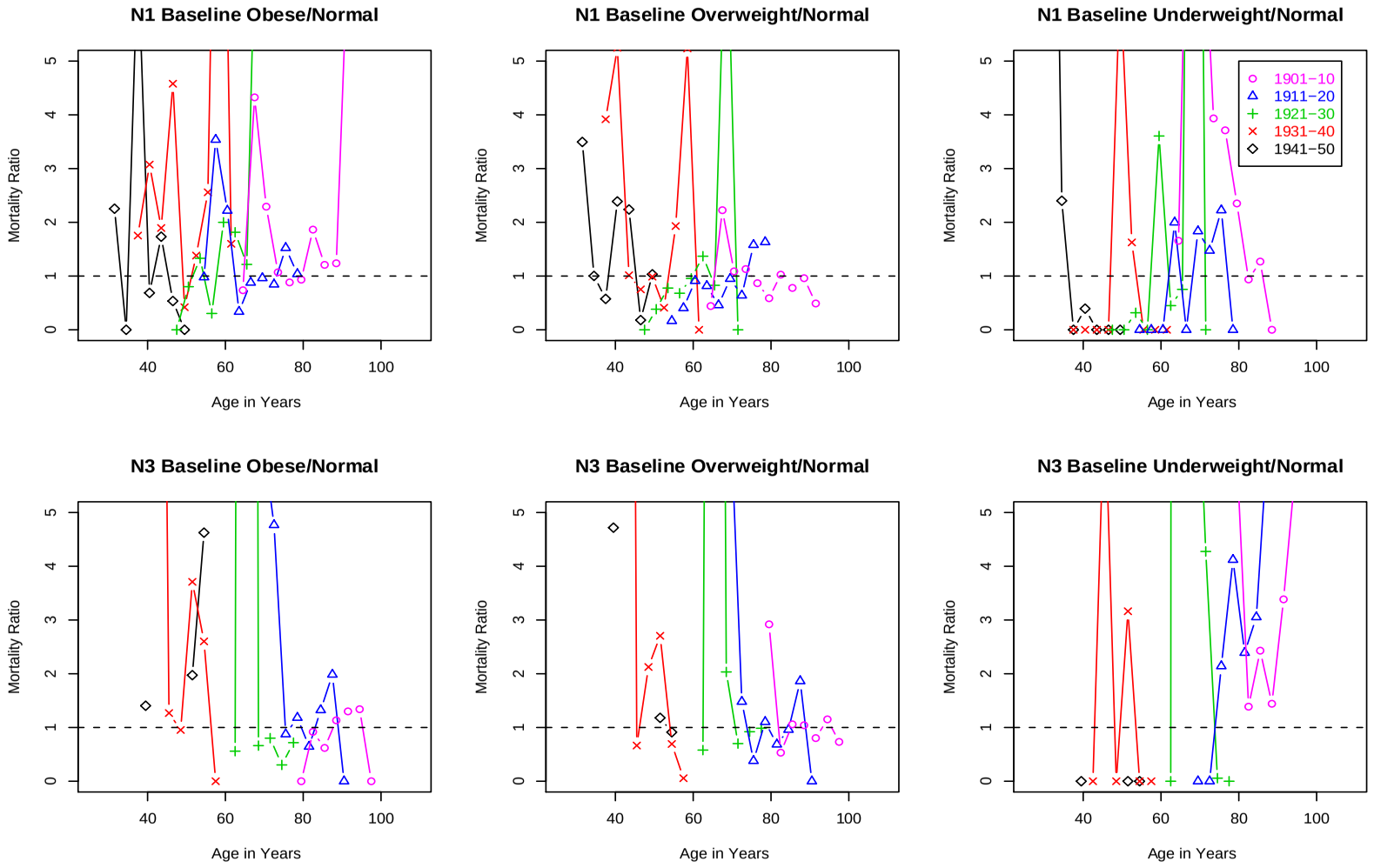


Figure 10: Observed Age-specific Mortality Ratios, Combining Cohorts, 1971-75 NHANES through 1992 (N1) and 1988-94 NHANES through 2000 (N3), Females, Weighted

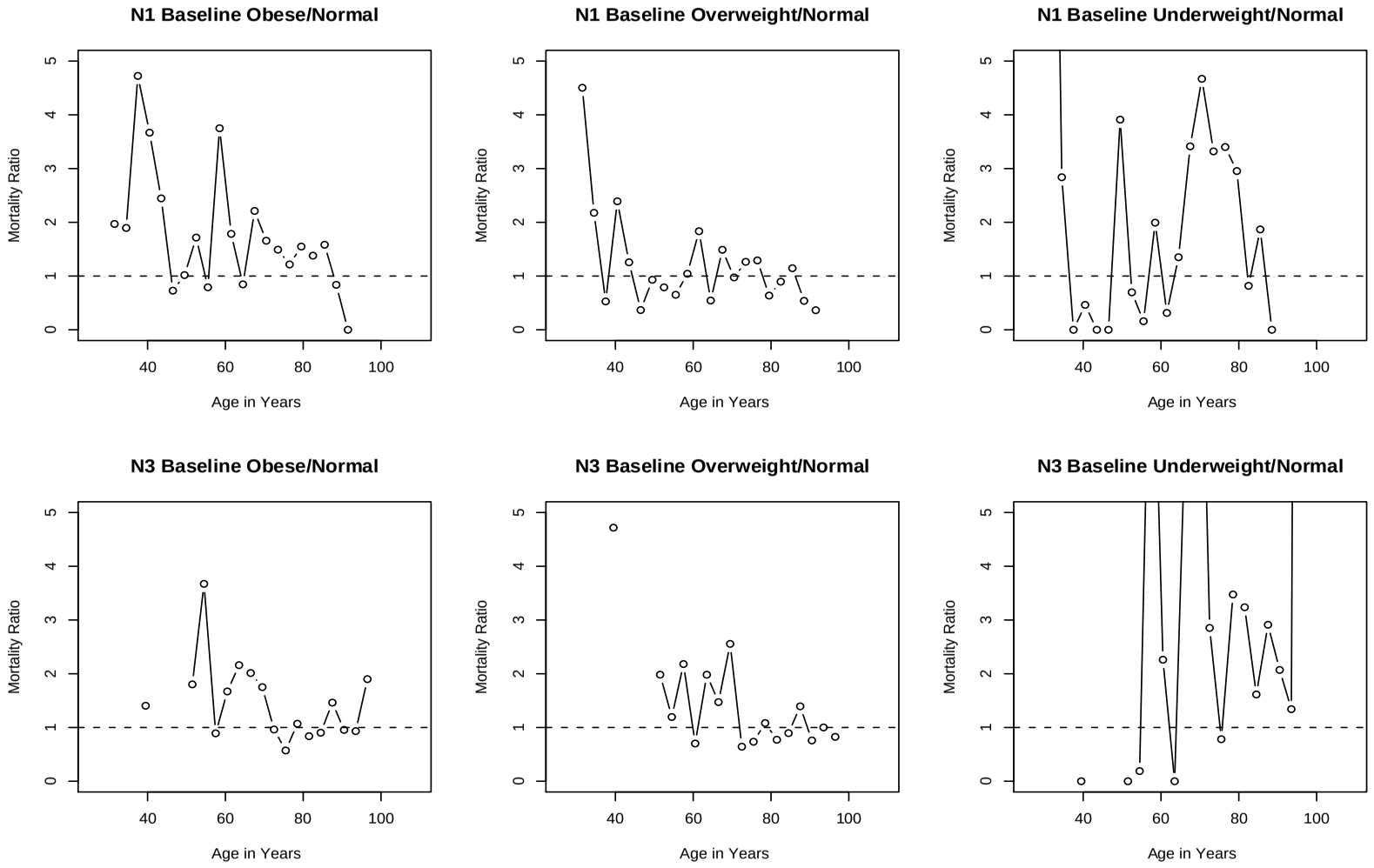


Figure 11: Observed Age-specific Mortality Ratios, Combining Cohorts, 1971-75 NHANES through 1992 (N1) and 1988-94 NHANES through 2000 (N3), Males, Weighted

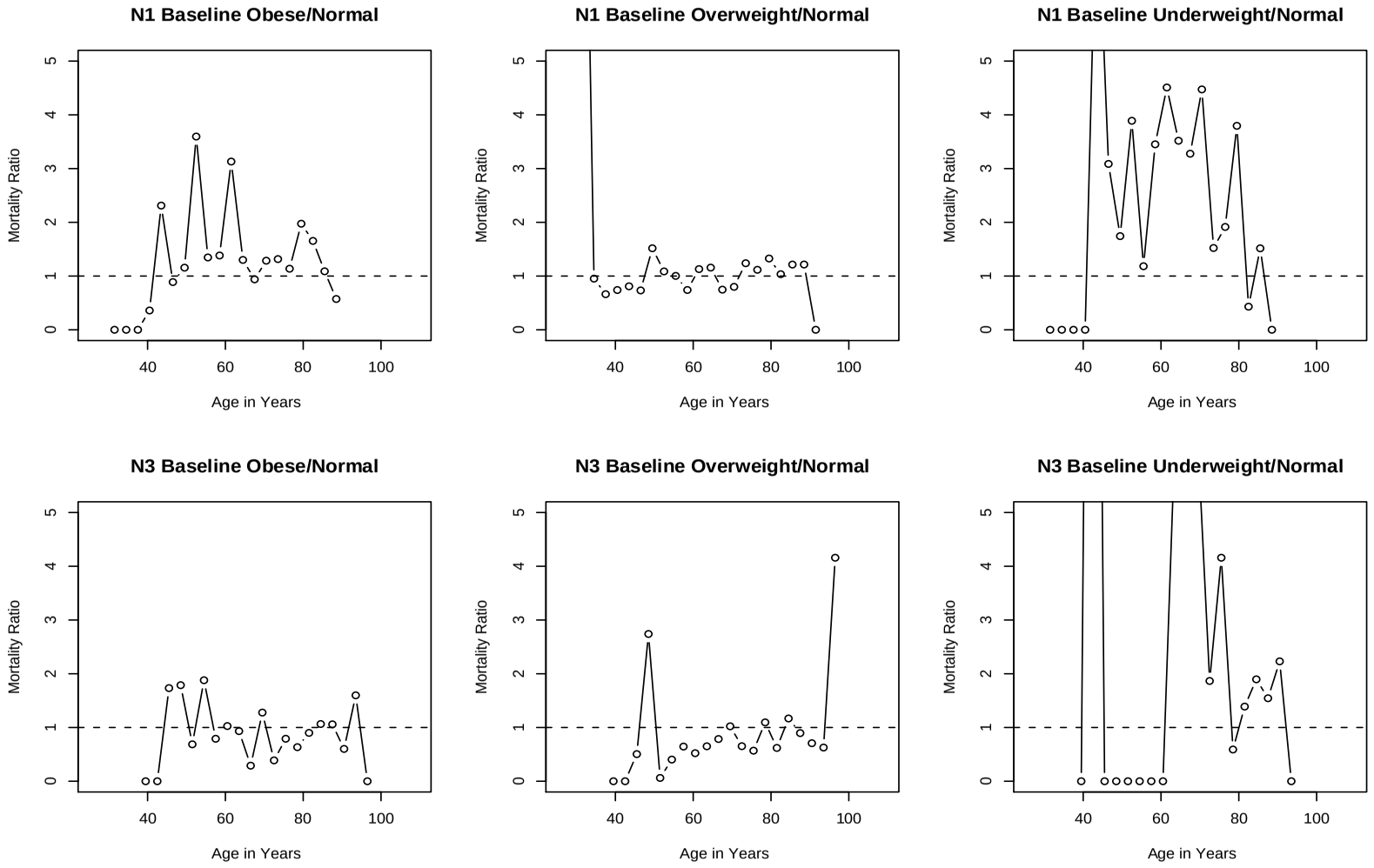


Figure 12: Estimated Mortality Ratios and 95% C.I., Separate Analysis of 1976-80 and 1988-94 NHANES (N2 and N3), Males, Weighted

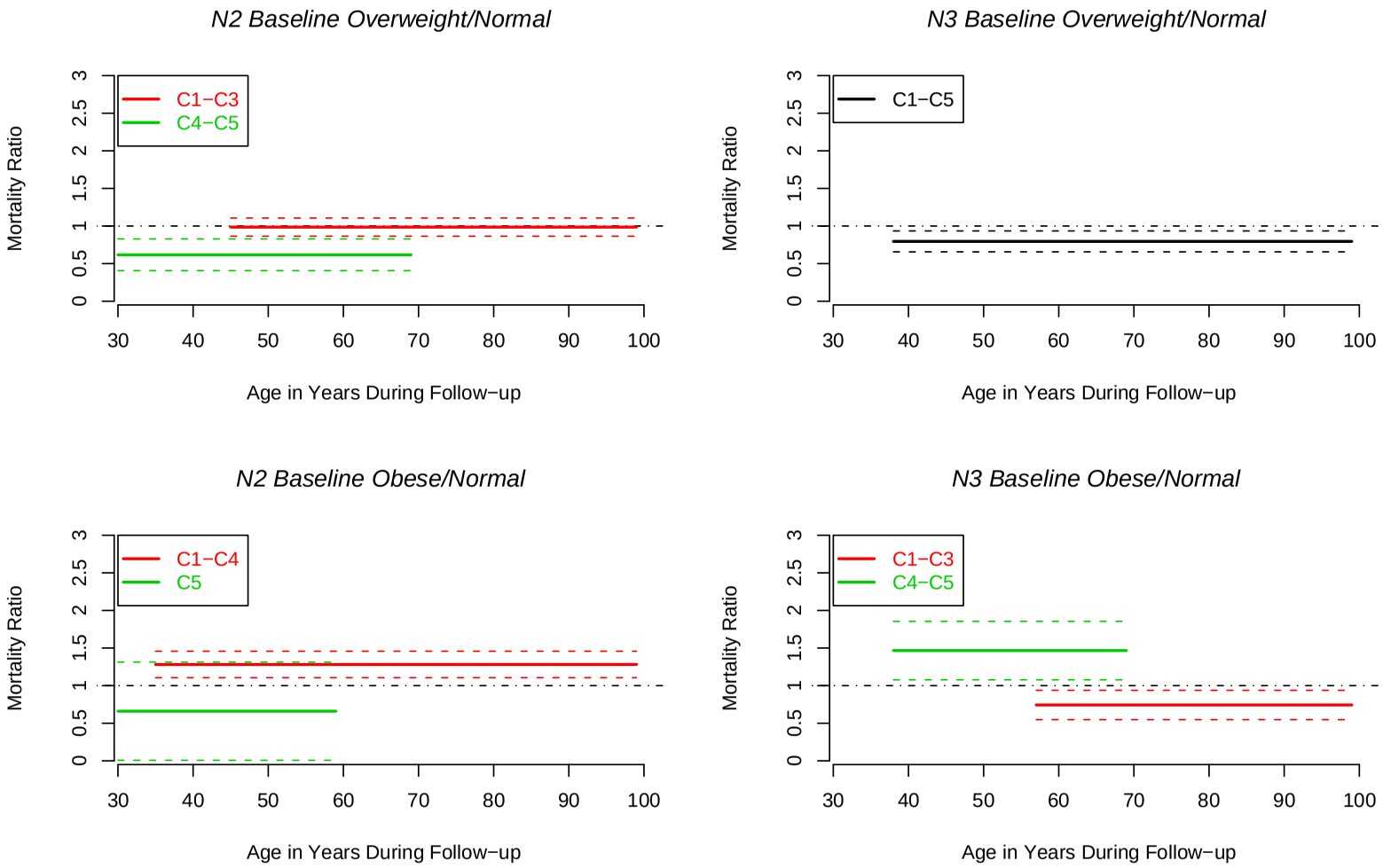


Figure 13: Estimated Mortality Ratios and 95% C.I., Separate Analysis of 1976-80 and 1988-94 NHANES (N2 and N3), Females, Weighted

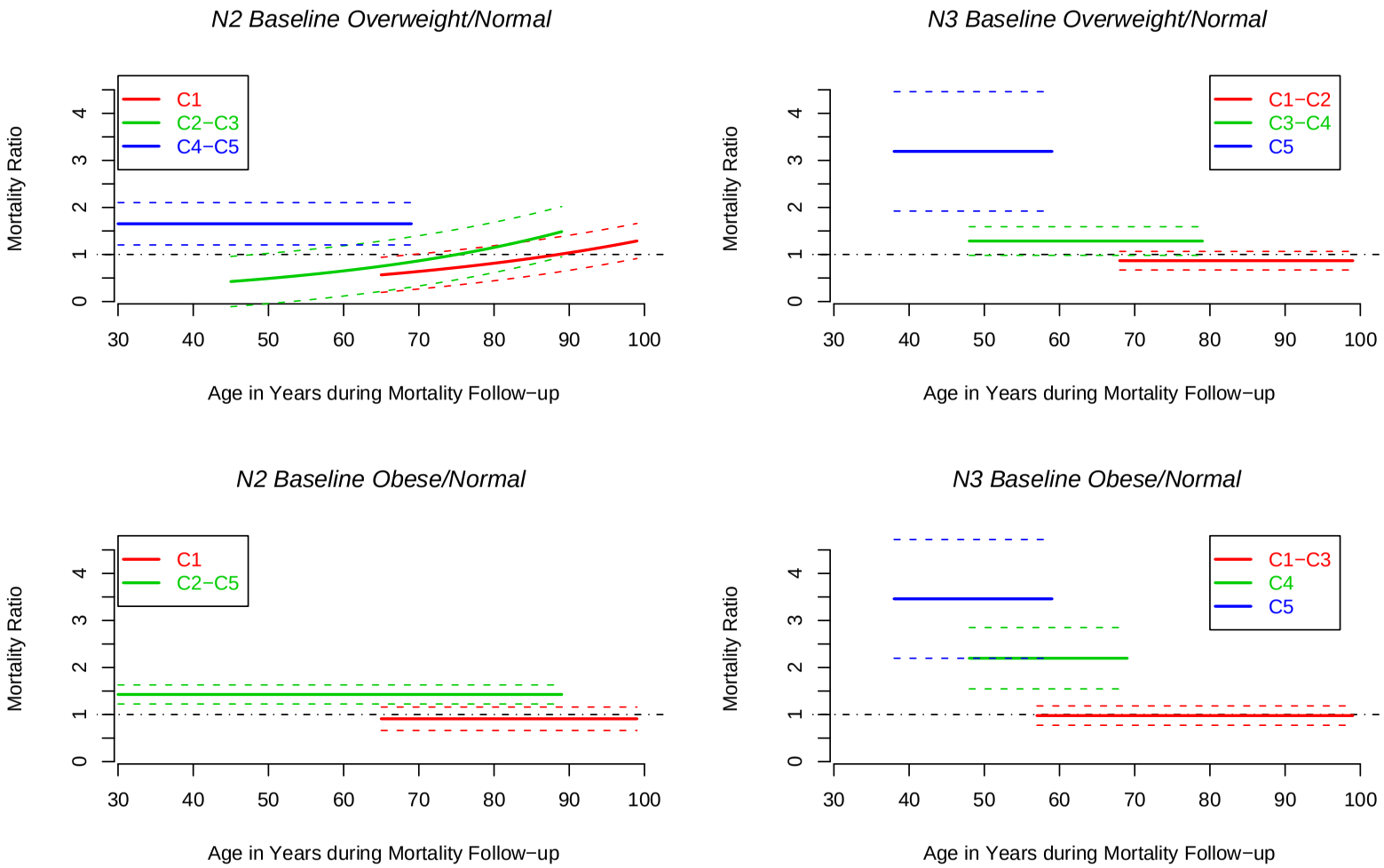


Figure 14: Age Patterns and Cohort Distortions, Mortality Ratios Under Models with and without Cohort Distinctions, 1976-80 and 1988-94 NHANES (N2 and N3), Males, Weighted

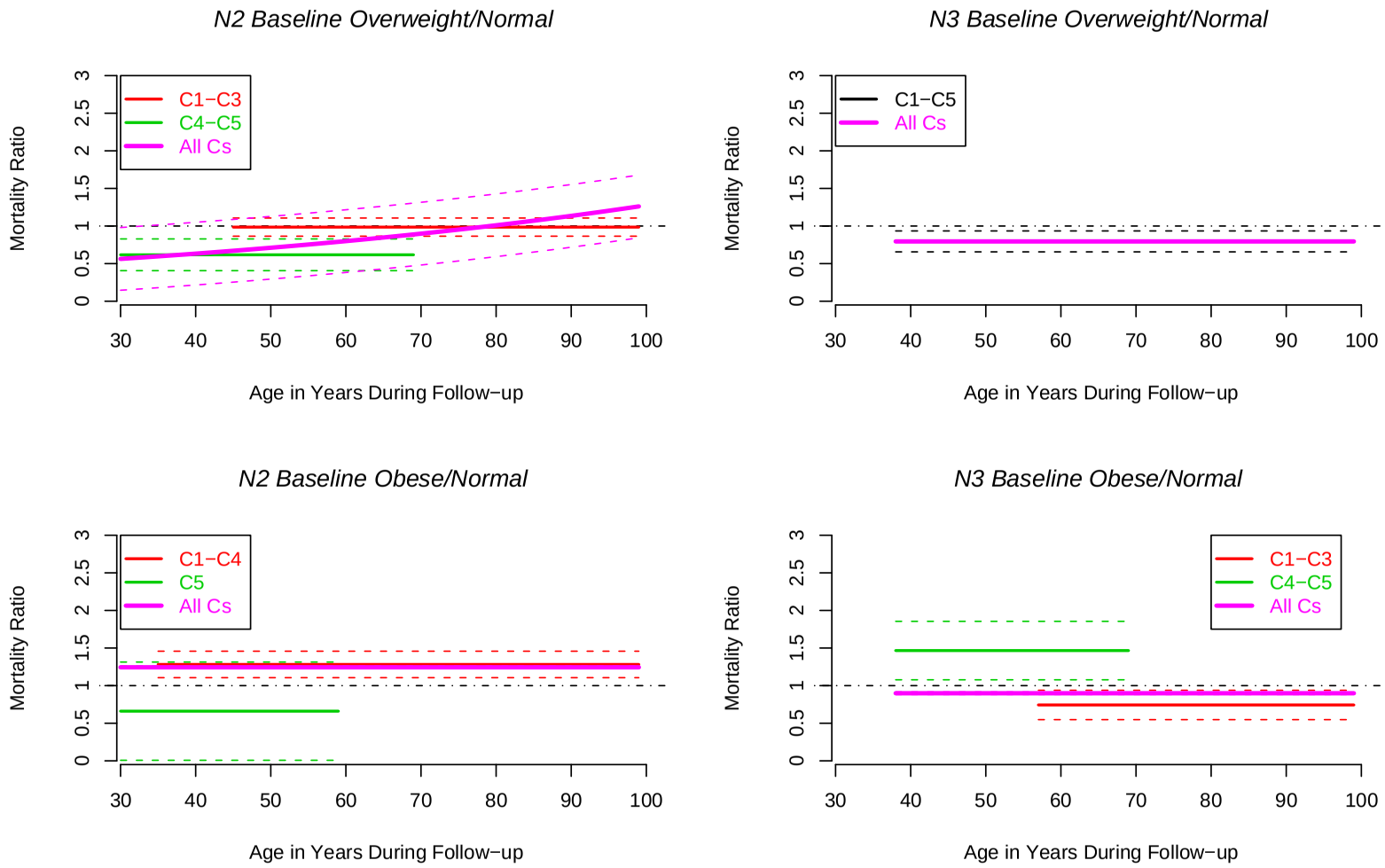
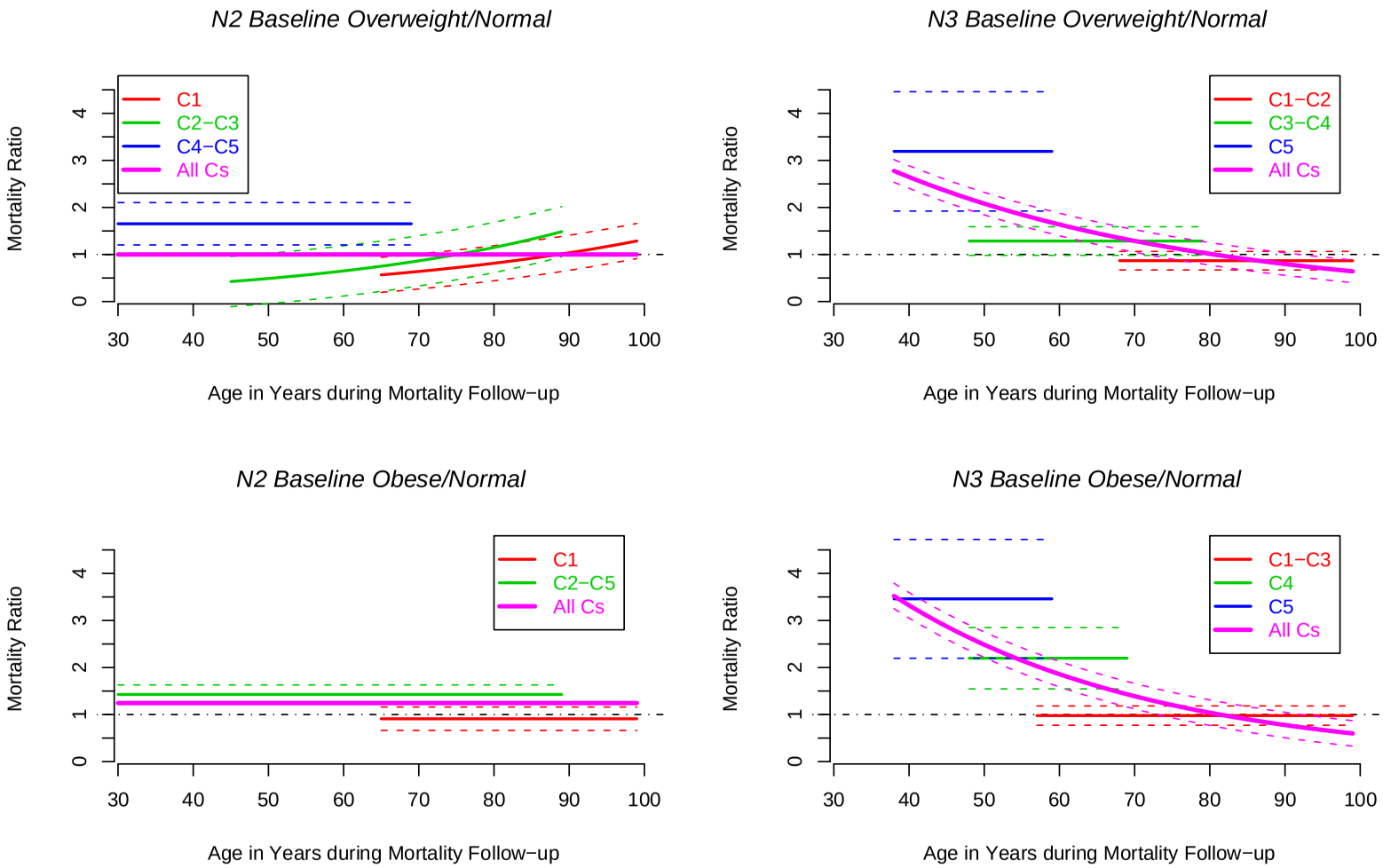


Figure 15: Age Patterns and Cohort Distortions, Mortality Ratios Under Models with and without Cohort Distinctions, 1976-80 and 1988-94 NHANES (N2 and N3), Females, Weighted



Appendices

Table A-1: Separate Analysis of 1976-80 and 1988-94 NHANES (N2 and N3), Estimated Mortality Ratios and 95% Confidence Intervals, Under Preferred Gompertz Models in Table 9*

Baseline Weight and Birth Cohort	1976-80 NHANES (N2) Estimate	1976-80 NHANES (N2) 95% C.I.	1988-94 NHANES (N3) Estimate	1988-94 NHANES (N3) 95% C.I.
Males				
Normal-weight	1	–	1	–
Underweight				
C1: 1901-10	1.54	1.1-1.98	2.36	1.72-2.99
C2: 1911-20	1.54	1.1-1.98	2.36	1.72-2.99
C3: 1921-30	1.54	1.1-1.98	2.36	1.72-2.99
C4: 1931-40	0.3	0-1.13	2.36	1.72-2.99
C5: 1941-50	0.3	0-1.13	10.18	5.99-14.37
Overweight				
C1: 1901-10	0.99	0.86-1.11	0.8	0.66-0.93
C2: 1911-20	0.99	0.86-1.11	0.8	0.66-0.93
C3: 1921-30	0.99	0.86-1.11	0.8	0.66-0.93
C4: 1931-40	0.62	0.41-0.83	0.8	0.66-0.93
C5: 1941-50	0.62	0.41-0.83	0.8	0.66-0.93
Obese				
C1: 1901-10	1.28	1.11-1.46	0.74	0.55-0.94
C2: 1911-20	1.28	1.11-1.46	0.74	0.55-0.94
C3: 1921-30	1.28	1.11-1.46	0.74	0.55-0.94
C4: 1931-40	1.28	1.11-1.46	1.47	1.08-1.86
C5: 1941-50	0.66	0.01-1.31	1.47	1.08-1.86
Females				
Normal-weight	1.00	–	1.00	–
Underweight				
C1-C5	1.59	1.23-1.95	2.18	1.72-2.64
Overweight				
C1: 1901-10	See Table A-2	See Table A-2	0.87	0.67-1.06
C2: 1911-20	See Table A-2	See Table A-2	0.87	0.67-1.06
C3: 1921-30	See Table A-2	See Table A-2	1.29	0.98-1.59
C4: 1931-40	1.65	1.2-2.1	1.29	0.98-1.59
C5: 1941-50	1.65	1.2-2.1	3.19	1.92-4.46
Obese				
C1: 1901-10	0.92	0.68-1.16	0.98	0.77-1.18
C2: 1911-20	1.42	1.22-1.61	0.98	0.77-1.18
C3: 1921-30	1.42	1.22-1.61	0.98	0.77-1.18
C4: 1931-40	1.42	1.22-1.61	2.2	1.55-2.85
C5: 1941-50	1.42	1.22-1.61	3.46	2.2-4.72

* Sample weights are used; all models include covariates for education, race and smoking status; negative lower bounds are changed to zero.

Table A-2: Mortality Ratios and 95% Confidence Intervals, Overweight/Normal-weight, 1976-80 NHANES Females, Under the Preferred Gompertz Model in Table 9*

Birth Cohort and Selected Ages	Estimate	95% C.I.
C1: 1901-10		
Age 65	0.57	0.19-0.94
Age 75	0.72	0.35-1.09
Age 85	0.92	0.55-1.29
Age 95	1.17	0.80-1.54
Age 99	1.29	0.91-1.66
C2-C3: 1911-30		
Age 45	0.42	0-0.96
Age 55	0.56	0.03-1.10
Age 65	0.75	0.22-1.28
Age 75	1.00	0.46-1.53
Age 85	1.33	0.79-1.86
Age 89	1.49	0.95-2.02

* Sample weights are used; all models include covariates for education, race and smoking status; negative lower bounds are changed to zero.

Table A-3: Separate Analysis of 1976-80 and 1988-94 NHANES (N2 and N3), Parameter Estimates and Standard Errors, Under Preferred Gompertz Models in Table 9*

Baseline Weight and Birth Cohort	1976-80 NHANES (N2) Estimate	S.E.	1988-94 NHANES (N3) Estimate	S.E.
Males				
Normal-weight	0	–	0	–
Underweight				
C1: 1901-10	0.4322	0.1818	0.8575	0.2109
C2: 1911-20	0.4322	0.1818	0.8575	0.2109
C3: 1921-30	0.4322	0.1818	0.8575	0.2109
C4: 1931-40	-1.196	0.7659	0.8575	0.2109
C5: 1941-50	-1.196	0.7659	2.3203	0.6706
Overweight				
C1: 1901-10	-0.0146	0.062	-0.2287	0.0796
C2: 1911-20	-0.0146	0.062	-0.2287	0.0796
C3: 1921-30	-0.0146	0.062	-0.2287	0.0796
C4: 1931-40	-0.4825	0.137	-0.2287	0.0796
C5: 1941-50	-0.4825	0.137	-0.2287	0.0796
Obese				
C1: 1901-10	0.2483	0.0794	-0.2969	0.1155
C2: 1911-20	0.2483	0.0794	-0.2969	0.1155
C3: 1921-30	0.2483	0.0794	-0.2969	0.1155
C4: 1931-40	0.2483	0.0794	0.3829	0.1638
C5: 1941-50	-0.4147	0.4099	0.3829	0.1638
Females				
Normal-weight	0	–	0	–
Underweight				
C1-C5	0.4631	0.1443	0.7801	0.1589
Overweight				
C1: 1901-10	See Table A-4	See Table A-4	-0.1421	0.1081
C2: 1911-20	See Table A-4	See Table A-4	-0.1421	0.1081
C3: 1921-30	See Table A-4	See Table A-4	0.2511	0.1369
C4: 1931-40	0.5022	0.1783	0.2511	0.1369
C5: 1941-50	0.5022	0.1783	1.1604	0.3624
Obese				
C1: 1901-10	-0.0844	0.1282	-0.022	0.106
C2: 1911-20	0.3475	0.0842	-0.022	0.106
C3: 1921-30	0.3475	0.0842	-0.022	0.106
C4: 1931-40	0.3475	0.0842	0.7874	0.2242
C5: 1941-50	0.3475	0.0842	1.2412	0.3463

* Sample weights are used; all models include covariates for education, race and smoking status.

Table A-4: Estimates for Selected Parameters and Variance-Covariance Matrix of Preferred Gompertz Model (*M*4 in Table 9), 1976-80 NHANES Females (N2)*

Parameter	Estimate	Variance-Covariance Estimate (1)	(2)	(3)
Overweight				
β Equation				
(1): C1-C3	-2.1356	0.453238	–	–
γ Equation				
(2): C1	0.00201	-0.00045	4.519E-7	–
(3): C2-C3	0.00237	-0.00050	4.961E-7	5.685E-7

* Sample weights are used; all models include covariates for education, race and smoking status.

Table A-5: Combined Analysis of 1976-80 and 1988-94 NHANES, Estimated Mortality Ratios and 95% Confidence Intervals*

Baseline Weight and Birth Cohort	1976-80 NHANES (N2) Estimate	95% C.I.	1988-94 NHANES (N3) Estimate	95% C.I.
Males				
Normal-weight	1	–	1	–
Underweight				
C1: 1901-10	1.45	1.05-1.86	2.68	1.55-3.82
C2: 1911-20	1.45	1.05-1.86	2.68	1.55-3.82
C3: 1921-30	1.45	1.05-1.86	2.68	1.55-3.82
C4: 1931-40	0.26	0-1.1	0.48	0-1.29
C5: 1941-50	1.45	1.05-1.86	2.68	1.55-3.82
Overweight				
C1: 1901-10	0.98	0.87-1.1	0.82	0.67-0.96
C2: 1911-20	0.98	0.87-1.1	0.82	0.67-0.96
C3: 1921-30	0.98	0.87-1.1	0.82	0.67-0.96
C4: 1931-40	0.61	0.42-0.8	1.05	0.51-1.59
C5: 1941-50	0.68	0.04-1.32	0.51	0.36-0.66
Obese				
C1: 1901-10	1.27	1.1-1.44	0.76	0.58-0.95
C2: 1911-20	1.27	1.1-1.44	0.76	0.58-0.95
C3: 1921-30	1.27	1.1-1.44	0.76	0.58-0.95
C4: 1931-40	1.27	1.1-1.44	0.92	0.44-1.4
C5: 1941-50	0.68	0.04-1.32	1.13	0.53-1.73
Females				
Normal-weight	1	–	1	–
Underweight				
C1-C5	1.77	1.49-2.04	1.77	1.49-2.04
Overweight				
C1: 1901-10	0.94	0.84-1.05	0.94	0.84-1.05
C2: 1911-20	0.94	0.84-1.05	0.94	0.84-1.05
C3: 1921-30	0.94	0.84-1.05	0.94	0.84-1.05
C4: 1931-40	1.48	1.06-1.91	1.48	1.06-1.91
C5: 1941-50	2.74	1.9-3.58	2.74	1.9-3.58
Obese				
C1: 1901-10	1	0.78-1.22	0.77	0.52-1.02
C2: 1911-20	1.32	1.13-1.52	1.02	0.81-1.23
C3: 1921-30	1.32	1.13-1.52	1.02	0.81-1.23
C4: 1931-40	1.5	1-2.01	2.26	1.61-2.9
C5: 1941-50	2.11	1.21-3.01	3.16	2.19-4.13

* Under $M8$ in Table 10; sample weights are used; all models include covariates for education, race and smoking statu; negative lower bounds are changed to zero.

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