Successful Aging and Obesity: Social and Developmental Heterogeneity in Trajectories of Body Weight from Middle to Older Ages

by

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Dedication

To Vlad – only the blessed have a light to guide them through life; he is my love and my guiding light…

To my children, Daniel and Michael – everything I am is for them…

To my Mom, Rebecca – in a perfect world, we would all have her moral clarity and her wisdom…

This is for them.

Acknowledgments

Albert Einstein once observed: "…if we knew what it was we were doing, it would not be called research, would it?" I am not convinced this applies to others, but it deftly applies to me… Embarking on my dissertation work, I thought the what, why, how, and when were all clearly and reasonably planned out. I was so wrong… It is through the support and grace of others that I reached a meaningful destination and it is with immense gratitude that I mention them.

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obstacles. Jersey, Mary, Cathleen and Jane were always there to guide the perplexed in me…

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If “life is the art of drawing sufficient conclusions from insufficient premises” (Samuel Butler), then all that’s valuable in my work is shared with many others. The insufficiencies are solely mine…

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CHAPTER I: INTRODUCTION

OBESITY IN OLDER ADULTS: WHAT WE KNOW, WHAT WE DON'T AND WHY SHOULD WE CARE? FRAMING THE RESEARCH GOALS WITHIN A LIFE COURSE PERSPECTIVE

Overweight and obesity have emerged as a substantial public health concern over the last 20 years, as their prevalence increased rapidly in the US and other developed countries, reaching epidemic proportions (Arterburn, Crane, & Sullivan, 2004; Mokdad et al., 2001; Wang & Beydoun, 2007). Currently, more than 66 percent of US adults age 20 -74 years old are overweight or obese and 33 percent are obese (Flegal, Carroll, Ogden, & Curtin, 2010; Mokdad et al., 2003; Ogden et al., 2006). Data from the successive NHANES study waves show significant increases in the prevalence of overweight and obesity, with most of the increase occurring in the obese category (www.cdc.gov). Further, upward changes in overweight/obesity rates have occurred in all racial/ethnic and age groups (Ogden, Carroll, McDowell, & Flegal, 2007; C. L. Ogden et al., 2006). In older adults, overweight and obesity have been associated with a wide-range of health (Berrington de Gonzalez et al., 2010; Field et al., 2001; Mokdad, Marks, Stroup, & Gerberding, 2004; Must et al., 1999; Olshansky et al., 2005; Profenno, Porsteinsson, & Faraone, 2010), social (Gortmaker, Must, Perrin, Sobol, & Dietz, 1993; Novak, Ahlgren, & Hammarström, 2005) and economic (Finkelstein, Fiebelkorn, & Wang, 2003; Finkelstein, Trogdon, Cohen, & Dietz, 2009; Thorpe, Florence, Howard, & Joski, 2004) adverse consequences.

This work attempts to trace the long-term trajectory of body-weight from middle- to older age and to identify factors (socio-economic and behavioral) that modify its course. Three main considerations provide the motivation for this research. First,

the natural history of weight change over the life course, and especially from middle to older ages, is not well understood because of conflicting evidence from cross sectional and the few existing longitudinal studies (motivation for Essay #1). Second, modification of health behaviors (smoking, alcohol-use, physical activity) has been long advocated as a solution to the obesity epidemic in all age groups, and as a potential path to reducing social health disparities in aging. Yet, the effect of health behaviors, and modifications thereof, on the long term trajectories of body-weight in older adults is still unclear (motivation for Essay #2). Finally, while it is conceivable that individuals vary in their body-weight trajectories over long periods of time, the heterogeneity in body-weight trajectory and its predictors have not been systematically studied (motivation for essay #3).

SPECIFIC AIMS

Essay # 1: Social Stratification of Body-Weight Trajectories in Middle-Age and Older Americans

Existing cross-sectional studies suggest that obesity rates increase robustly with age, from puberty until late middle age, after which they decrease somewhat, such that adults 40–59 years of age are more likely to be overweight or obese compared with younger and older individuals (Hedley et al., 2004; Ogden et al., 2007). Empirical findings from longitudinal studies on body-weight changes over the adult life course are few and mixed. Some studies find that body-weight increases up to middle age (Clarke, O'Malley, Johnston, & Schulenberg, 2009), after which it decreases (Hardy & Kuh, 2006; Kahng, Dunkle, & Jackson, 2004; Woo, Ho, & Sham, 2001), while others find only minor or non-significant reductions in body-weight in people older than 70 (He & Baker, 2004; Villareal, Apovian, Kushner, & Klein, 2005).

Additionally, racial/ethnic and other socio-economic disparities in the prevalence of overweight/obesity have been observed in younger age groups (James, Fowler-Brown, Raghunathan, & Van Hoewyk, 2006; Lewis et al., 2005; Mujahid, Diez Roux, Borrell, & Nieto, 2005; Ogden et al., 2007; Seo & Torabi, 2006). Yet, the

socioeconomic patterning of obesity in older ages has not been systematically studied. In fact, in her opening editorial to a special issue of Research in Aging dedicated to obesity in older ages, Himes (2004) argues that, although racial and ethnic background are often included as control variables in analyses of obesity in older ages, they rarely are the main focus of investigation, and states that "greater understanding is needed given the strong relationship between ethnicity and body size" in old ages (pg. 5). Essay #1 has the following two main aims:

Specific Aim 1: To analyze the patterns of change in body-weight from middle into older age.

Specific Aim 2: To identify and describe racial/ethnic, gender, education, and age differences in body-weight trajectories in older adults.

Essay # 2: The Effect of Stability and Change in Health Behaviors on Trajectories of Body-Mass Index in Middle Aged and Older Americans

Health behaviors, such as smoking (Appel & Aldrich, 2003; Fillenbaum, Burchett, Kuchibhatla, Cohen, & Blazer, 2007; Sulander, Rahkonen, Nissinen, & Uutela, 2007) engagement in physical activity (DiPietro, 2001; Dziura, Mendes de Leon, Kasl, & DiPietro, 2004; Nelson et al., 2007), and alcohol consumption (Arif & Rohrer, 2005), are associated with the risk for overweight/obesity in older adults, but their impact on the trajectory of body-weight is still uncertain. Long-term longitudinal studies of smoking, alcohol use and physical activity status and change-in-status, allowing for multiple variations across time, and concomitantly accounting for other risk behaviors and potential confounders are needed. Essay # 2 has the following aims:

Specific Aim 1: To obtain quantitative estimates of the effects of smoking, physical activity, and alcohol use *status* and *over-time variation* on the long-term trajectory of body-mass index (BMI) starting in middle age.

Specific Aim 2: To examine whether the intra-individual association of health behaviors with the BMI trajectory changed over the period of time under consideration (1992-2006).

Essay # 3: Trajectories of Body-Mass Index from Middle to Older Age and their Socio-Demographic Predictors

Current knowledge on the progression of body-weight starting in middle-age rests on an assumption of population homogeneity; in other words, it assumes that the population follows an "average" pattern of change, and that the observed deviation of individuals from the "average" can be explained in terms of distinctive characteristics, such as gender, race, age, or health status indicators. The underlying heterogeneity in body-weight development in older age has not been studied. This is the primary purpose of Essay #3:

Specific Aim 1: To identify multiple distinct trajectories of body-mass index (BMI) in middle-age and older adults.

Specific Aim 2: To explore socio-demographic differences in the propensity to follow each of the identified trajectories.

OVERARCHING CONCEPTUAL FRAMEWORK

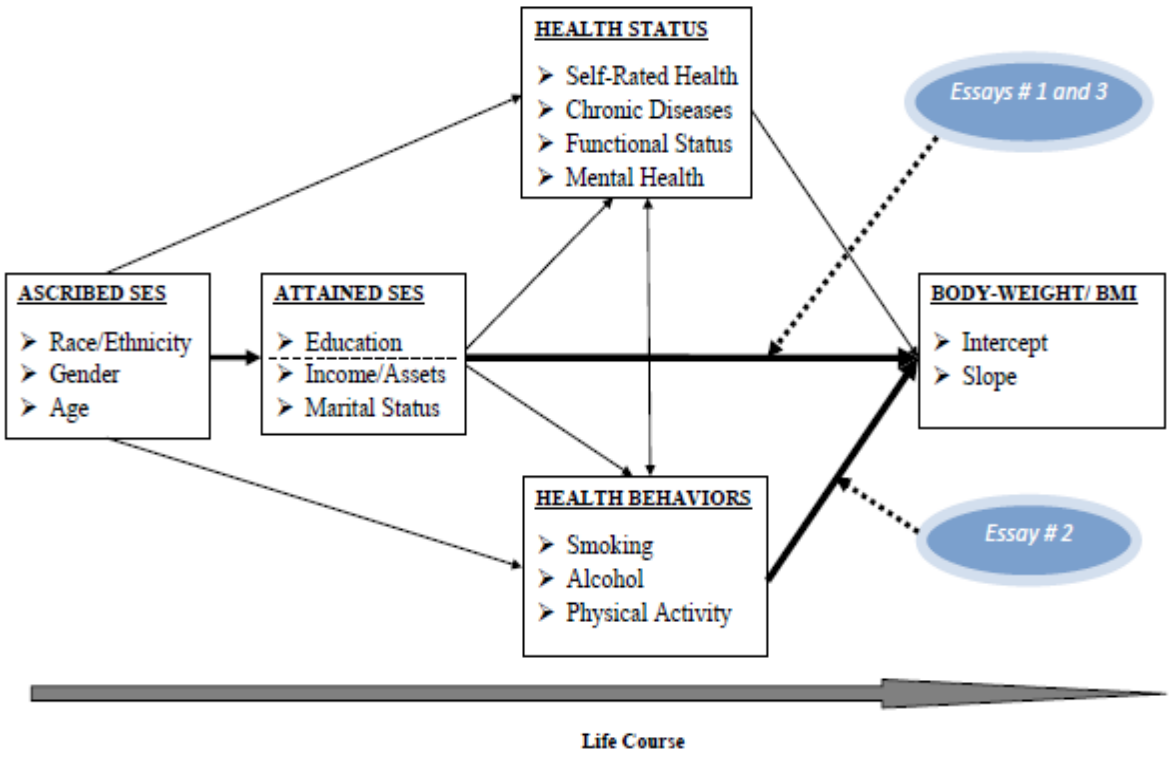
The life course approach to health and health inequalities can inform the study of body-weight changes in older ages. The life course perspective proposes that aging and the health changes associated with it are closely related to early events or to traits displayed earlier in the life course (Elder, 1985; Elder, Johnson, & Crosnoe, 2003; Kuh et al., 2007). This perspective depicts health outcomes as trajectories across time and assumes that the nature, direction, timing and patterns of change are linked to early-life experiences, abilities, and resources (Ben-Shlomo & Kuh, 2002; Elder, 1985). Further, the life course perspective conceptualizes change as a chain of risk, noting that social or biological adversity at any stage in life has the potential to alters the subsequent life course, most often leading to adverse

outcomes (Barker, 2007; Barker, Eriksson, Forsen, & Osmond, 2002), and suggests that the heterogeneity in patterns of aging (Dannefer, 2003; Nelson & Dannefer, 1992) is the result of a differential accumulation of risk factors over successive stages in the life course. Obesity "tracks from cradle to grave" (Eriksson, Forsen, Osmond, & Barker, 2003), and involves biological and social processes that are set in early life and determine the pathways to health in adult life (Wadsworth, 1997). The study of body-weight development is particularly suited to a life course perspective because of its multidimensional - biological, behavioral and social – etiology and its long-term development encompassing both critical periods and accumulating risk effects (Barker, 2007; Gillman, 2004). The life course perspective also creates the conceptual framework for integrating social factors, either ascribed (e.g., race/ethnicity, gender) or established during various life stages (e.g., education, income) as determinants of socially distinct pathways to obesity and related health outcomes.

Recurrent findings documenting racial/ethnic and socio-economic disparities in overweight and obesity, more pronounced and consistent among women (Baltrus, Lynch, Everson-Rose, Raghunathan, & Kaplan, 2005; Baltrus, Everson-Rose, Lynch, Raghunathan, & Kaplan, 2007; Clarke et al., 2009; Seeman et al., 2008; Seo & Torabi, 2006), suggest that social deprivation at various stages in the life course acts as a "fundamental cause" (Link & Phelan, 1995) of later life body-weight trajectory. In fact, the observed stratification of health outcomes along socio-economic and racial/ethnic lines (House, Lantz, & Herd, 2005; Seeman & Crimmins, 2001) persists or even increases into old ages (Alwin & Wray, 2005; Kim & Durden, 2007; O'Rand & Hamil-Luker, 2005). The social stratification of health and aging framework proposes that ascribed characteristics such as age (Riley, 1973; Riley, 1987), race (Williams, 1999; Williams & Jackson, 2005; Williams, 2005) and gender (Bird & Rieker, 1999; Robert & House, 2000), influence socio-economic status attainment (i.e., income, education, occupation) (Adler & Rehkopf, 2008; Robert & House, 2000), which in turn condition exposures to health risks and protective factors (Link & Phelan, 1995) and explain (at least partially) later-life inequalities in health

outcomes (Adler, Marmot, McEwen, & Stewart, 1999; Kaplan, Baltrus, & Raghunathan, 2007; Kawachi & Kennedy, 1999; Lantz et al., 2001). Extrapolating from the social stratification framework, our study attempts to link the *ascribed* social characteristics, mainly race and ethnicity, to the trajectory of body-weight from middle to older ages, both directly and indirectly through *attained* social position (education, income, and assets), marital status, and prior physical and mental health status.

Conceptual Model of Relationships between SES, Health Behaviors, Health Status and Body-Weight over the Life Course



Notes: SES – socio-economic status; BMI – body-mass index. Bold lines represent the main focus of investigation; non-bold lines are treated as moderating/mediating relationships. The model and the theoretical justification for each relationship are further detailed in the essays.

A FEW NOTES ON METHODOLOGY AND STATISTICAL ANALYSIS

Trajectories of health are useful outcome measures, because, as previously discussed, the health status at any point in time can be conceptualized as being the result of multiple transitions, each with its own intercept and pattern of change. Describing the age-norm (i.e., average) body-weight trajectory requires an examination of the intra-personal growth curve or trajectory (i.e., intercept and rate-of-change) over an extended period of time. Further, identifying socio-economic variations in the trajectory of body-weight mandates a separation of intra-personal changes from inter-personal differences. Neither cross-sectional studies, nor studies of changes between two points in time can accomplish this. Cross-sectional studies underestimate the magnitude of age-related weight gain (Nooyens et al., 2008) and cannot discriminate between intra-personal and inter-personal determinants of body-weight. Studies of transitions between two points in time can only estimate the amount and direction of change (i.e., amount of weight gained or lost), but are inadequate in assessing the rate-of-growth, especially if this is not constant, and do not expose the underlying growth curve or trajectory leading to the end-point (Rogosa, 1988).

To enable the estimation of intra- and inter-personal differences in body-weight trajectories, two methodological approaches will be used: (1) hierarchical linear modeling (HLM) (Raudenbush & Bryk, 2002) will estimate an average body-weight trajectory over time and separately quantify the variation from the average trajectory due to inter-personal differences, such as race/ethnicity, gender, education, education, or age-at baseline (Essay #1) or due to differences in patterns of health behaviors (Essay #2); and (2) semiparametric mixture modeling (SPMM) (Jones & Nagin, 2007; Nagin, 2005) will identify distinct group-based trajectories of body-weight and the factors explaining differences in the propensity to follow each of the identified trajectories (Essay #3).

In each study, multiple successively-adjusted models will be tested, in accordance with the classic recommendations for the evaluation of moderation/mediation effects (Baron & Kenny, 1986). The models will be adjusted for:

1. *Non-random missingness* due to mortality, attrition, and proxy status. This is because the apparent decrease in body-weight in older ages may be an artifact of selective survival of leaner healthier individuals, such that the groups surviving into older ages may consist largely of obesity-resistant individuals (Thorpe & Ferraro, 2004). In fact, some studies (Barone et al., 2006) find that controlling for selective mortality explains most of the observed decline in weight after age 65.
2. Multiple *time-varying socioeconomic potential confounders,* such as income, wealth/assets and marital status, to account for within-person changes in socio-economic indicators.
3. *Health status indicators.* Another potential source of bias is the age-related decline in health (Mehta & Chang, 2009; Yang, Bishai, & Harman, 2008). In other words, lower body-weight in older age may be the result of co-existing morbidities and may not represent the "normal" course of body-weight over time (Losonczy et al., 1995).

The conceptual framework and methodological considerations (including advantages, disadvantages, assumptions and limitations) are discussed in detail in the respective section of each essay. Further, concluding thoughts on what inferences can and cannot be drawn from these studies, along with proposals for future research are provided in the Conclusion section.

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CHAPTER II: SOCIAL STRATIFICATION OF BODY WEIGHT TRAJECTORY IN MIDDLE-AGE AND OLDER AMERICANS: RESULTS FROM A 14-YEAR LONGITUDINAL STUDY

II.1. BACKGROUND

Overweight and obesity have emerged as a major public health concern in the US over the past 20 years, as their prevalence increased substantially in all age groups. More than 70 percent of people 70 years and older are overweight with more than 30 percent obese (Ogden et al., 2006). In older ages, obesity has been associated with increased mortality (e.g., Thorpe & Ferraro, 2004), cognitive impairment and dementia (e.g., Profenno, Porsteinsson, & Faraone, 2010), cardiovascular morbidity (e.g., Harris et al., 1997) and functional impairment (e.g., Ferraro, Su, Gretebeck, Black, & Badylak, 2002; Peeters, Bonneux, Nusselder, De Laet, & Barendregt, 2004).

Empirical observations on how body-weight changes over time are mixed (Hedley et al., 2004; Ogden, Carroll, McDowell, & Flegal, 2007). Some cross-sectional studies suggest that body-weight increases up to about age 65 years and declines afterwards (Flegal, Carroll, Kuczmarski, & Johnson, 1998; McDowell, Fryar, Ogden, & Flegal, 2008; Seidell & Visscher, 2000). However, weight loss in old age may be an artifact of selective survival of leaner healthier individuals (Barone et al, 2006; Losonczy et al., 1995; Thorpe & Ferraro, 2004). Longitudinal studies of change in body-weight in older ages are few and limited to relatively short periods of follow-up, ranging from three to six years (Kahng, Dunkle, & Jackson, 2004; Newman et al., 2001; Woo, Ho, & Sham, 2001). The results of such studies are rather inconsistent: while some report that body-weight declines after age 65 (e.g., Kahng, Dunkle, &

Jackson, 2004), others find either no reduction (e.g., Villareal, Apovian, Kushner, & Klein, 2005) or increases in body-weight into older ages (e.g., Barone et al., 2006; He & Baker, 2004).

Charting and describing the trajectory (i.e., estimating the intercept and rate-of-change) of body-weight over time cannot be accomplished through cross-sectional studies or studies of change between two points in time. Cross-sectional studies confound intrapersonal and interpersonal differences, while studies of transitions between two points in time are inadequate in assessing the underlying growth curve or trajectory (Rogosa, 1988). Currently, there is little research on the level and rate-of-change in BMI from middle to older age.

Furthermore, there is limited knowledge concerning how the level and rate-of-change in body-weight in middle and old age vary with key dimensions of social stratification, such as race/ethnicity, gender, and socioeconomic status (SES). Himes (2004), in a special issue of Research in Aging dedicated to obesity in older ages, argues that despite the strong relationship between ethnicity and body-weight, race/ethnicity is seldom the main focus of investigation in obesity studies. Given the association of obesity with poor aging outcomes (e.g., Field et al, 2001) and the well-defined pattern of social inequalities in aging and health (Alwin & Wray, 2005; Kim & Durden, 2007), advancing our understanding of how social stratification dimensions affect the trajectory of body-weight in middle and later life should be viewed as a critical goal.

This study has two specific aims. First, we offer quantitative estimates depicting the trajectory of body-mass index (BMI) by analyzing 14 years (1992 – 2006) of longitudinal data from a national sample of Americans born between 1931 and 1941. Second, we examine how the level and rate-of-change in BMI vary with ascribed (race/ethnicity, gender, age-at-baseline) and achieved (education) social status.

Conceptual Framework

The prevalence of obesity varies significantly with gender, race/ethnicity, and socio-economic status (e.g., Baltrus, Lynch, Everson-Rose, Raghunathan, & Kaplan, 2005; Clarke, O'Malley, Johnston, & Schulenberg, 2009). This is consistent with the perspective of social stratification of aging and health (House, Lantz, & Herd, 2005; Seeman & Crimmins, 2001), which proposes that race (Williams & Jackson, 2005), age (Riley, 1987), and gender (Bird & Rieker, 1999) influence socio-economic status (SES) attainment (i.e., income, education, occupation) (Adler & Ostrove, 1999), marital status (Wyke & Ford, 1992), and exposure to health risks and protective factors (Link & Phelan, 1995), consequently leading to inequalities in health outcomes in later life (e.g., Adler, Marmot, McEwen, & Stewart, 1999; Lantz et al., 2001). This conceptual framework will guide our examination of relationships between social stratification dimensions and trajectories of BMI starting in middle age.

Hypotheses

To the best of our knowledge, there is currently no quantitative depiction of long-term body-weight trajectory after age 50. Existing studies suggest that body-weight increases throughout adult life up to 60-65 years of age, where it levels off or declines (e.g., Ogden et al., 2007; Williamson, 1993). However, most of these results come from cross-sectional or longitudinal studies of short duration. We hypothesize that after age 50, body-mass index (BMI) increases over time following a curvilinear decelerating trajectory *(Hypothesis 1)*.

Racial/ethnic minorities, especially women, are disproportionately represented among the overweight and obese (Center for Disease Control [CDC], 2009; Ogden et al., 2007; Wang & Beydoun, 2007). While some investigators have reported that minority groups also experience higher weight gain over time (Chor, Faerstein, Kaplan, Lynch, & Lopes, 2004; Clarke et al., 2009; Truong & Sturm, 2005), this has not been replicated by others (Lewis et al., 2005). In addition, racial/ethnic

differences in weight gain are confounded with other socio-economic characteristics (Baltrus et al., 2005; Kahn & Williamson, 1991). Because of these inconsistencies and because many studies do not include Hispanics as a separate group in the analysis (e.g., Kahng et al., 2004; Lewis et al., 2005; Mujahid, Diez-Roux, Borrell, & Nieto, 2005), racial/ethnic similarities or differences in body-weight trajectories are not well understood. We hypothesize that, relative to non-Hispanic Whites, Non-Hispanic Blacks and Hispanics exhibit a higher level of BMI and a higher rate of increase over time *(Hypothesis 2)*.

Findings on gender differences in the prevalence of overweight/obesity are mixed. Some studies find no differences (e.g., Ogden et al., 2007; Sobal & Rauschenbach, 2003), while others point to a higher prevalence of overweight in men (Clarke et al., 2009; He & Baker, 2004; Novak, Ahlgren, & Hammarström, 2005; Wang & Beydoun, 2007), and a higher prevalence of obesity in women (e.g., Jenkins, Fultz, Fonda, & Wray, 2003; Williamson, 1993). Over time, women show greater variability in body weight (Williamson, 1993) and tend to gain more weight compared to men (He & Baker, 2004; Truong & Sturm, 2005). Consequently, we hypothesize that, relative to men, women will have a lower BMI level, but a higher rate of increase over time *(Hypothesis3)*.

The educational advantage in overweight/obesity is well documented in younger adults (review in Ball & Crawford, 2005). Both the risk of overweight/obesity (Molarius, Seidell, Sans, Tuomilehto, & Kuulasmaa, 2000; Mujahid et al., 2005; Novak et al., 2005) and the rate of weight gain over time (Baltrus, Everson-Rose, Lynch, Raghunathan, & Kaplan, 2007; Mujahid et al., 2005) are inversely associated with educational achievement. Further research is required to clarify the role of education in shaping the trajectory of body-weight in older adults. We hypothesize that, relative to individuals with less education, those with more education will have both a lower level of BMI and a lower rate of increase over time *(Hypothesis 4)*.

Age differences in the trajectories of health may reflect cohort and aging effects. The prevalence of overweight/obesity has increased in recent cohorts (Reynolds &

Himes, 2007). Further, in younger adults, a more rapid pace of weight gain has been identified (Clarke et al. 2009; Grinker, Tucker, Vokonas, & Rush, 1995; Jacobsen et al., 2001). Extrapolating from these findings, we hypothesize that relative to younger individuals, older individuals will have both a lower level of BMI and lower rates of increase over time *(Hypothesis 5)*.

Consistent with the social stratification of health and aging framework, and because of their previously documented relationship with overweight/obesity, we control in our analyses for other socio-economic characteristics, such as income/assets (Chang & Lauderdale, 2005; Fonda, Fultz, Jenkins, Wheeler, & Wray, 2004) and marital status (Sobal & Rauchenbach, 2003), and for health status (Mehta & Chang, 2009).

II.2. METHODS

Design and Data

Data came from the Health and Retirement Study (HRS). The study started in 1992 by surveying a nationally representative sample of over 12,800 respondents from the 1931-1941 birth cohort, and subsequently added participants from older and younger birth cohorts (HRS documentation at: http://hrsonline.isr.umich.edu/). Because our study's goal is to depict the BMI trajectory starting in middle age, and to minimize the potential for cohort effects (Reynolds & Himes, 2007), we chose to focus on one cohort, namely respondents who were born between 1931 and 1941 (the original HRS birth cohort). A total of 13,565 individuals are in the HRS sample: 12,899 in the original sample (95.1%) and the remaining 666 added as new spouses and partners since the beginning of the study in 1992. Baseline data were obtained in 1992, and follow-up data were gathered every 2 years from 1994 to 2006 (for a total of up to 8 repeated observations over a 14-year follow-up period).

The response rates range from 81.7% (1992) to 89.1% (in 1994). As of 2006, cumulative mortality rate (validated through linkages to the National Death Index) was 19%. When a respondent was unable to be interviewed due to physical or

cognitive limitations, a proxy interview was conducted. The rates of proxy interviews vary by wave and range from 4.8% (1992) to 9.0% (2002).

From the initial sample of 13,565 individuals, 3,116 were excluded for being age-ineligible spouses (i.e., born before 1931 or after 1941). Further, 135 individuals were excluded because they never responded to the survey health sections relevant to our analysis. This resulted in a final analytic sample of 10,314 individuals with a total of 82,512 observations (mean number of interviews completed = 6.4). More than half (55.7%) of the respondents in the final sample completed all 8 interviews.

Measures

The dependent variable Body Mass Index (BMI) was calculated using the following formula:

$$BMI = [\text{Weight (lb)}/\text{Height (inches)}^2] \times 703.$$

Self-reported weight was recorded at each wave; height was recorded for the first interview and verified in the second one. For BMI calculation at each wave we used current weight and initial height.

Several measures of social stratification were included. Indicator variables were created for *gender* (1=Female, 0=Male) and *race/ethnicity* (i.e., non-Hispanic White, non-Hispanic Black, and Hispanic). Race/ethnicity was ascertained through a series of questions designed to ensure mutually-exclusive racial/ethnic categories. The Hispanic sample consisted of Mexican-Americans (60.5%), Cubans (9.8%), Puerto Ricans (8.4%), Dominicans (2.2%) and "other" Hispanic origin (18.9%). Henceforth, non-Hispanic Whites will be referred to as "Whites", non-Hispanic Blacks as "Blacks", while the "Hispanic" category includes only those who at the outset defined themselves as "Hispanics or Latino". *Education* was measured by the number of years of schooling completed (lowest =0, highest =17 years). *Age-at-baseline* was measured as the difference between the baseline (i.e., 1992) and the year of birth (range = 51-61; mean = 55.8, SD=3.17).

Income and *assets* were measured at each wave respectively by total household income (from respondent and spouse only) and total household assets. Because the distributions of both *income* and *assets* were highly skewed, the variables were categorized (quartiles) for all analyses. *Marital status* was measured by binary variable coded as 1= married or living with a partner, 0 = single, divorced, widowed, or separated.

Because health status may operate either as a confounder (Mehta & Chang, 2009) or as a mediator between socio-economic and demographic characteristics and other health outcomes of interest (Seeman et al., 2004), in this case BMI, physical and mental health measures were added as time-varying covariates. *Index of Chronic Diseases* was measured as a count of seven prevalent chronic conditions – heart disease, stroke, high-blood pressure, diabetes, arthritis, chronic lung disease, and cancer. Each disease was coded as 0 (no) and 1 (yes), and a count of 1 (yes) was created (range = 0-7). *Self-rated health* was assessed as a single-item rating of respondent's health (1 = excellent; 2 = very good; 3 = good; 4 = fair; 5 = poor). *Functional status* was represented by the NAGI index (Nagi, 1979), calculated as a count of six items, assessing difficulties with the following activities: (1) stooping, crouching or kneeling; (2) lifting or carrying weights >10 pounds (>4.5 kg); (3) extending the arms above shoulders; (4) standing up after sitting; (5) pulling or pushing a large object; and (6) writing or handling small objects. Each item was scored as 0 (no difficulty reported) or 1 (at least some difficulty reported). NAGI index ranges from 0 (no difficulty) to 6 (difficulties reported in all activities). *Depressive symptoms* were assessed by a count of nine items from the CES-D Scale (Center for Epidemiological Studies Depression Scale) (Radloff, 1977). All nine items were concordant across waves and include: (1) felt depressed, (2) everything was an effort, (3) restless sleep, (4) felt happy, (5) felt lonely, (6) enjoy life, (7) felt sad, (8) couldn't get going, (9) had a lot of energy. For consistency, all items were re-coded to indicate negative feelings, with a value of 0 = no or 1 = yes. CES-D count ranges from 0 (no depressive symptoms) to 9 (maximum depressive symptoms).

Finally, *proxy status* was measured at each wave by a binary variable coded 0 (self-respondent) or 1 (proxy respondent).

Weighted sample descriptive statistics (Gelman, 2007) for all time-varying covariates (Table 1) and time-constant covariates (Table 2) are presented below.

[Tables 1 and 2 about here]

Data Analysis

Hierarchical linear models (Raudenbush & Bryk, 2002) were used to estimate the trajectory of BMI over a period of observation of 14 years (1992-2006).

We modeled the intra-personal changes in BMI with the following equation:

$$Y_{iT} = \pi_{0i} + \pi_{1i} Time + \Sigma\pi_{ki} X_{kiT} + \varepsilon_{iT} , \qquad (1)$$

where Y_{iT} is the BMI of an individual *i* at time *T.* π_{0i} is the intercept of BMI for an individual *I,* and π_{1i} is the slope (intra-personal rate of change) in BMI for individual *i* over time. *Time* is the distance (in years) of assessment from the baseline. X_{kiT} represents the time-varying covariates (e.g., marital status, income, assets) associated with individual *i* at time *T,* and π_{ki} represents the effect of X_k on individual *i's* BMI. ε_{iT} is the random error in BMI for individual *i* at time *T.*

We employed a time-based analytic approach (Alwin, Hofer, & McCammon, 2006) and controlled for inter-personal age-at-baseline differences. We considered both linear and non-linear (quadratic and cubic) patterns of change in BMI. Time was centered at its mean to minimize the possibility of multicollinearity when evaluating quadratic and cubic time (T) functions. Consequently, the intercept for any given model should be interpreted as the BMI at the mean follow-up time (i.e., 7 years into the study). Time-constant predictors, such as race/ethnicity, education, gender, and age-at-baseline, are included in the Level 2 equation (person-level or inter-personal variations model):

$$\pi_{pi} = \beta_{p0} + \Sigma\beta_{pq}X_{qi} + r_{pi} \quad (2)$$

Here, X_{qi} is the qth time constant covariate associated with individual i, and β_{pq} represents the effect of variable X_q on the pth growth parameter (π_p). r_{pi} is a random effect and has a mean of 0.

The HRS involved the over-sampling of Blacks, Hispanics and respondents from Florida. For the multivariate analyses, we decided against the inclusion of sample weights, because many of the attributes on which differential selection weights are calculated (e.g., race/ethnicity, gender, marital status) are explicitly controlled in the models. As such, unweighted ordinary least squares estimates are less biased than and preferable over weighted estimates (Winship & Radbill, 1994). In addition, we ran all models both with and without weights (respondent-level case weights from the 2006 HRS Cross-Tracker file) and obtained very similar results. Finally, we set the statistical significance p-level at less than 0.01, to avoid the over-abundance of significant results due to the very large HRS sample.

Mortality, Attrition and Proxy Status

To minimize the loss of participants due to item missing (Little & Rubin, 2002), multiple imputation was performed (Schafer & Olsen, 1998). Using the NORM software (Schafer, 1997), we imputed 5 data sets and ran HLM analyses using each data set. Parameter estimates and their standard errors were calculated by averaging across the 5 data sets and adjusting for their variance (Raudenbush & Bryk, 2002).

We also addressed the potential for selection bias due to non-ignorable missing data as a result of mortality and attrition. Prior studies document the complicated relation between body-weight and mortality in older age (e.g., Thorpe & Ferraro, 2004). In studies of older populations, the probability of missing data due to mortality and attrition is systematically related to the health outcomes of interest (Harel, Hofer, Hoffman, & Pedersen, 2007) and consequently the critical "missing-at-random" assumption is violated (Little & Rubin, 2002). The potential for

confounding effects of selective mortality and attrition needs to be methodologically addressed (Liang et al., 2010; Mroczek & Spiro, 2005). We included binary variables in the Level 2 equation (Mortality: 0 = alive at the end of the study and 1 = died during study; Attrition: 0 = completed the study period and 1 = dropped out for reasons other than mortality and did not return in subsequent waves) to differentiate respondents who completed the study period from those who died or dropped-out before the study ended. This approach is similar to the pattern-mixture models, in which subjects are classified into different groups, based on their missing data patterns, and estimates are obtained by averaging over the identified patterns (Hedeker & Gibbons, 2006, pp. 302-312). Finally, proxy status was measured as a time-varying covariate (0=self, 1=proxy respondent).

II.3. RESULTS

Trajectory of BMI

Using linear, quadratic, and cubic functions, we mapped the trajectory of BMI between 1992 and 2006. The unconditional model showed that BMI follows a quadratic trajectory, with an intercept of 27.38 ($p<.001$), linear slope of 0.047 ($p<.001$) and a quadratic slope of 0.002 ($p<.01$) (Table 3 - M_0), apparently lending support for Hypothesis 1. However, adjustment for mortality/attrition and additional socio-demographic and health status measures (Table 3 – M_1, M_2, M_3, and M4) rendered the quadratic coefficient non-significant and revealed a largely linear trajectory (intercept b=27.361, $p<.001$; linear slope b=0.067, $p<.001$ in M_4). As the cubic slope coefficient was not significant in the unconditional model (M_0) it was not included in subsequent analyses.

[Table 3 about here]

Racial/Ethnic Differences in BMI Trajectory

The results showed significant racial/ethnic differences in the intercept and linear slope (Hypothesis 2), but not in the quadratic slope (Table 3 - M_2). As Figure 1

illustrates, compared with Whites, Blacks exhibited a higher BMI level (b=1.585, p<.001), but a lower rate-of-increase (b = -0.053, p<.001). Differences between Whites and Blacks remain robust after adjustment for socio-economic and health status factors. However, heterogeneity in SES and health status appears to account for the Hispanics – White differences in the level of BMI, but not in the rate-of-change (M_3 and M_4).

[Figure 1 about here]

Gender Differences in BMI Trajectory

The hypothesized gender differences in BMI trajectory (Hypothesis 3) were not supported (M_2). Men and women had similar BMI levels and linear rates-of-change, except in the model controlling for health status (M_4), in which females showed a lower BMI level (b= -0.259, p<.01).

Educational Differences in BMI Trajectory

Hypothesis 4 was partially supported, as we found significant educational differences in BMI level, but not in the rate-of-change over time. Specifically, higher education was associated with a lower BMI intercept (b = -0.099, p<.001 in M_3). The linear rate-of-change in BMI was similar at different levels of education, except when accounting for baseline BMI (b = -0.002, p<.01 in M_5). These results remained robust when SES (M_3) and health status (M_4) were taken into account (Figure 2).

[Figure 2 about here]

Age Differences in BMI Trajectory

Older age groups had lower levels of BMI (b = -0.063, p<.001 in M_2) and smaller rates-of-change (b = -0.006, p<.001 in M_2). Age differences persisted after adjustment for SES, health status and baseline BMI (M_4 and M_5). As depicted in Figure 3, participants who are older at baseline had a lower intercept and a lower linear rate of weight gain compared with their younger-at-baseline counterparts.

Controlling for all covariates, a one-year increase in age-at-baseline is associated with a decrease of 0.051 in the level of BMI at the mean follow-up time ($p<.001$) and a lower linear rate of increase over time ($b = -0.006$, $p<.001$ in M_5). These findings were consistent with the hypothesized age-at-baseline differences in BMI trajectory (Hypothesis 5).

[Figure 3 about here]

Effects of Other Covariates

Socioeconomic factors, such as income/assets and marital status, as well as health status, may operate as confounding variables (Chang & Lauderdale, 2005; Mehta & Chang, 2009; Sobal & Rauchenbach, 2003) or as mediators (Baltrus et al., 2005; Seeman et al., 2004). In accordance with Baron and Kenny's (1986) recommendation for mediation testing, models 4 and 5 (Table 3) account for these variables. While BMI levels were not significantly associated with assets, higher income predicted a lower BMI in all models ($p<.001$) (M_3). Marital status was positively associated with BMI, such that in every model tested, married people had higher BMI ($p<.001$ for all models) than people who were single, widowed or divorced.

Finally, with the exception of NAGI index, health status measures (chronic diseases, self-rated health and CES-D) showed no association with BMI levels. In particular, those with more functional limitations exhibited higher BMI, even after adjusting for baseline age and BMI.

Mortality, Attrition and Proxy Interview

To control for potential selection bias, mortality, attrition and proxy status were included as confounding variables. Even though the analyses performed with these covariates amount to regression analyses, we recommend against interpreting mortality and attrition as classic "predictors", because both are final events which do not precede any event to be eventually "predicted". As such, they are viewed as

confounding variables, although they may also serve as proxies for poor health (Harel, Hofer, Hoffman, & Pedersen, 2007).

Mortality was negatively associated with the trajectory of BMI in all models; those who died during the follow-up period had a lower BMI (b= -0.436, p<.001) and a lower rate of gain (b = -0.079, p <. 001 in M_1) prior to death. Attrition was negatively associated with the level of BMI, such that people who dropped-out of the study had a lower mean BMI (b = -0.686, p <.001 in M_1). The linear rate-of-change was not associated with attrition in any of the models tested. Proxy status predicted a lower level of BMI (b = -0.606, p<.001 in M_1) in all models. These results suggest that selection bias is a threat in that parameter estimates for the trajectory of BMI would be biased should those measures not be included in the analysis.

II.4. DISCUSSION and CONCLUSIONS

To the best of our knowledge, this is the first study which quantitatively depicts the long-term trajectory of BMI in a national sample of middle aged Americans. Whereas BMI increases in a largely linear fashion over time, this trajectory varies significantly across several dimensions of social stratification, including race/ethnicity, education, and age-at-baseline. However, we observe no gender differences in BMI trajectories.

There are significant racial/ethnic variations in the level and rate-of-change in BMI. Non-Hispanic Blacks enter middle age at a significantly higher BMI level, but they gain less weight over time compared with their White counterparts. After accounting for differences in SES and health status, Hispanics do not differ from Whites in BMI levels, but they tend to gain less weight over time. Our findings differ from those of studies of BMI changes in younger age groups, which show that Hispanics and Blacks exhibit similar trajectories, having higher BMI levels and more accelerated rates of BMI growth compared with Whites (Clarke et al., 2009). Differences in cohort composition (Roshania, Narayan, & Oza-Frank, 2008) and acculturation patterns (Abraido-Lanza, Chao, & Florez, 2005) may account for the

discrepancy in findings. In younger cohorts (Clarke et al., 2009), the risk for overweight/obesity and the rate of change in BMI are similar among minority groups, perhaps due to similar life experiences and exposures (Palmore, 1978). In an older cohort, such as ours, a higher proportion of Hispanics may be foreign-born, first-generation immigrants (Card, 2005), exposed to different early-life environments (nutrition or physical activity patterns) and more likely to preserve cultural norms and values associated with "better" health behaviors (Abraido-Lanza, Chao, & Florez, 2005; Akresh, 2007). Another possible explanation is the underlying heterogeneity within the Hispanic population (Zsembik & Fennell, 2005). A majority (61%) of our sample consisted of Mexican-Americans. Because of the small number of respondents in other Hispanic groups (e.g., Cubans, Puerto Ricans, Dominicans) we were unable to analyze the trajectory of BMI in each of these sub-groups. The approach of combining various Hispanic sub-groups may obscure differences in socio-economic characteristics and in health behavior and further mute underlying differences in BMI trajectories. The same holds true for the underlying heterogeneity within the Non-Hispanic White and Non-Hispanic Black populations. The three commonly-recognized racial groups may fail to capture the considerable biological variability in human populations (Williams, 1999). Additional studies need to further define sub-group differences in BMI trajectories within the White, Black and Hispanic populations.

Contrary to findings from younger groups (e.g., Baltrus et al., 2005), our results show that in middle and old age socio-economic status does not substantially attenuate the relationship between race/ethnicity and BMI trajectory, with the exception of Hispanic/White differences in BMI intercept. This suggests that developmental patterns of body-weight are set much earlier in life (Barker, 2007). Disadvantaged prenatal and childhood environments have been associated with an increased risk of obesity in adulthood (e.g., Ferraro, Thorpe, & Wilkinson, 2003). Racial/ethnic differences in early-life determinants of obesity (Whitaker & Orzol, 2006) and the strong tracking of early-life obesity into adulthood and older ages

(Eriksson, Forsen, Osmond, & Barker, 2003) may explain how childhood racial disparities in body-weight track into adulthood and older ages.

Our study reveals educational differences in the level of body-weight, but not in the rate-of-change. Further, the effect is diminished when baseline BMI is considered, illustrating again the importance of early life factors in setting the course for weight development into older ages (e.g., Baltrus et al., 2007). We also find that the effect of education is not attenuated when considering potentially intervening economic determinants, such as income or assets, or differences in health status. It has been argued that health status and behaviors partially explain socio-economic, including educational, disparities in health trajectories in old age (e.g., Dietz, 2007; Lynch, Kaplan, & Salonen, 1997). According to our findings, the effect of education persists even after adjusting for time-varying measures of health status. Health behaviors partially mediate the association between education (as a measure of SES) and other health outcomes (Lantz et al., 2001) and may mediate the observed educational differences in BMI trajectory. Future examination of health behaviors (smoking, alcohol consumption, physical activity) as potential mediators between education and BMI trajectory is warranted, and may explain the observed decline in the "advantage" of education in younger cohorts (Himes & Reynolds, 2005).

Older age-at-baseline is associated not only with a lower level of BMI, but also with a slower rate of increase over time. Shorter-term longitudinal studies of older respondents (e.g., Dziura, Mendes de Leon, Kasl, & DiPietro, 2004; Kahng et al, 2004) found a decline in body weight after age 65, but our results are consistent with at least one study (Barone et al., 2006), which suggested that cohort and selective survival explain the apparent decline in body weight in older ages. Because of previously documented cohort effects (Reynolds & Himes, 2007) and the potential inadequacy of BMI as a measure of body composition in old ages, we chose to limit our focus to a single cohort of 51 to 61 years old. Our respondents were followed up to age 75 (the age of the oldest respondents at the end of the follow-up period),

revealing that, barring cohort and healthy survivor effects, BMI increases between the ages of 51 and 75.

We should emphasize that in our analysis we used time-based models (i.e., intrapersonal changes over the period of observation), and that interpersonal age differences were controlled in the prediction of both intercept and slope by the introduction of *age-at-baseline* as a time-constant covariate (Alwin, Hofer, & McCammon, 2006). We did not pursue an age-based analysis because HRS data are unsuitable for the correct estimation of the intrapersonal age effects on BMI (discussion in Liang et al, 2008). Briefly, because HRS yields only data collected from respondents from different cohorts at different ages, age and cohort effects are highly confounded, such that the observed age effects represent a combination of cohort- and "true" age effects. Therefore, we cannot infer on the effects of age on intrapersonal BMI development, but only on the effect of time in this specific age group.

Contrary to some prior observations, we find no gender differences in the trajectories of BMI. However, most previous findings come from younger groups (e.g., Baltrus et al., 2005; Clarke et al., 2009). Gender-related socio-economic profiles change across cohorts, and the effects of education (Clarke et al., 2009, Novak et al., 2006), race/ethnicity (Baltrus et al., 2005; Chor et al., 2004) and other measures of SES (Ball & Crawford, 2005; Mujahid et al., 2005) on BMI, vary by gender. In addition, prior studies often do not explicitly account for selective survival (e.g., He & Baker, 2004). Given the significant effect of mortality and attrition observed in our and other (e.g., Barone et al., 2006) studies, and the well-documented gender differences in mortality rates in older ages (with men dying younger), selective survival may explain previously observed gender differences in patterns of change in body-weight.

Several limitations of this research should be noted. Self-reported weight and height were used for BMI calculations across all waves. Individuals tend to over-report their height and under-estimate their weight (Gunnell et al., 2000; Nawaz,

Chan, Abdulrahman, Larson, & Katz, 2001), therefore BMI calculations based on self-reported weight and height are likely to understate the "true" BMI. Three considerations mitigate the potential for bias related to self-reporting. First, if the under-reporting of weight and the over-reporting of height are consistent over time, and there is no indication in the literature to the contrary, the analysis should yield valid estimates of changes in BMI over time. Second, we undertook a comparison of self-reported and interviewer-measured weight and height (available for two distinct random sub-samples of respondents in 2004 and 2006) and found the differences to be rather small in absolute values (not shown; results available upon request). This is also consistent with another analysis of the quality of self-reports of weight and height in HRS (Weir, 2008). Third, within the very limited literature on socio-demographic correlates of bias in self-reporting of weight and height, a review of accuracy of self-reporting in women found no differences among various racial groups (Engstrom, Paterson, Doherty, Trabulsi, & Speer, 2003). Nonetheless, we cannot entirely rule out yet unidentified socio-demographic differences in self-reporting of weight and height; future studies employing measured rather than self-reported weight and height are needed to validate our results.

Conventional anthropometric measures, such as BMI, may be inadequate measures of overall and abdominal fatness in elderly subjects (Prentice & Jebb, 2001; Seidell & Visscher, 2000). Further, interpretations of BMI trajectories in older age should take into account to effect of age-related loss of height. Prior longitudinal studies show that height loss accelerates during the eighth decade of life , peaks after age 80 (Dey, Rothenberg, Sundh, Bosaeus, Steen, 1999; Sorkin, Muller and Andres, 1999) and may account for small artifactual increases in BMI between age 70 and 80 (Sorkin et al., 1999). As some of the respondents in our sample were followed up to age 75, the possibility of upward bias in BMI in later waves in some participants should be considered. We were unable to augment our results with a similar analysis of other body-composition indicators and to assess the potential for artifactual BMI changes due to loss of height because HRS does not collect data on these indicators or on self-reported height in later waves.

Finally, additional studies need to consider a more refined differentiation of racial/ethnic groups, by considering the underlying heterogeneity within the three traditionally studied groups. Race/ethnicity embody life experiences and socio-economic characteristics (Williams, 1999); identifying meaningful ethnic sub-groups representing diverse socio-economic and life course patterns may elucidate some of the complexity in BMI development in adult and older ages. Further, since obesity is linked to an increased risk of early mortality and chronic morbidities, the associations identified here between race/ethnicity, SES and weight trajectories are prone to reinforce and even exacerbate the existing health inequalities in older ages. Our findings highlight the need for long-term longitudinal studies starting in young and middle adulthood, assessing multiple potential mediators and investigating the cumulative effects of racial and socio-economic components (education, income, occupation) on BMI development into old ages.

This research contributes to our understanding of the social stratification of health and aging dynamics by focusing on intra-personal and inter-personal differences in BMI trajectory in a national sample of middle-age Americans over a 14-year period. Our findings complement existing studies of younger and older age groups, to elucidate the dynamics of body-weight development in mid-adult life.

Table 1: Descriptive Statistics for Time-Varying Covariates[a] - weighted[b]

	Baseline (1992)		Wave2 (1994)		Wave3 (1996)		Wave4 (1998)		Wave5 (2000)		Wave6 (2002)		Wave7 (2004)		Wave8 (2006)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Socio-Demographic																
Marital Status	0.77	0.42	0.76	0.43	0.73	0.44	0.71	0.46	0.69	0.46	0.68	0.47	0.67	0.47	0.66	0.47
Proxy Status	0.05	0.21	0.06	0.23	0.06	0.23	0.07	0.26	0.08	0.27	0.08	0.27	0.07	0.25	0.05	0.21
Assets (category)	2.47	1.19	2.51	1.19	2.50	1.20	2.54	1.21	2.57	1.21	2.57	1.21	2.61	1.18	2.64	1.19
Income (category)	2.57	1.10	2.62	1.10	2.59	1.10	2.61	1.11	2.62	1.11	2.63	1.10	2.63	1.11	2.65	1.10
Health																
Self-Rated Health (1-5)	2.55	1.18	2.72	1.20	2.69	1.19	2.83	1.16	2.80	1.16	2.81	1.15	2.86	1.14	2.87	1.13
Index of Chronic Diseases (0-7)	1.16	1.11	2.44	1.47	2.74	1.62	3.02	1.76	3.30	1.88	3.58	1.93	3.80	1.95	4.01	1.94
CES-D count (0-9)	3.77	1.56	3.21	1.61	3.51	1.28	3.64	1.33	3.61	1.35	3.61	1.33	3.54	1.31	3.52	1.34
NAGI count (0-6)	1.44	1.66	1.18	1.56	1.27	1.61	1.32	1.61	1.34	1.61	1.45	1.62	1.51	1.61	1.58	1.64
Body-Mass Index	26.98	4.96	27.10	4.94	27.23	5.05	27.36	5.12	27.53	5.23	27.66	5.30	27.73	5.39	28.07	5.48

N(valid) = 82,512 observations.

[a] Time-varying covariates (Level 1) are those associated with repeated observations within individuals.

[b] Case weights for each wave are respondent-level weights (2006 HRS Cross-Tracker file).

Table 2: Descriptive Statistics for Time-Constant Covariates[a] - weighted[b]

Covariates	Mean/%	SD
Age (1992)	55.83	3.17
Female	52.3%	
Education[c]	12.34	3.05
Non-Hispanic Black	10.3%	
Hispanic	6.5%	
Mortality (between baseline and 2006)	18.7%	
Attrition (between baseline and 2006)	7.1%	

N(valid) = 10,367 respondents
[a]Time-constant covariates (Level 2) are those associated with the individual at baseline (1992).
[b]Case weights are respondent-level 1992 weights (2006 HRS Cross-Tracker File).
[c]Education is measured as "number of school-years completed".

Table 3: Within-persons and Between-persons Differences in Body-Mass Index: Hierarchical Linear Modeling Results

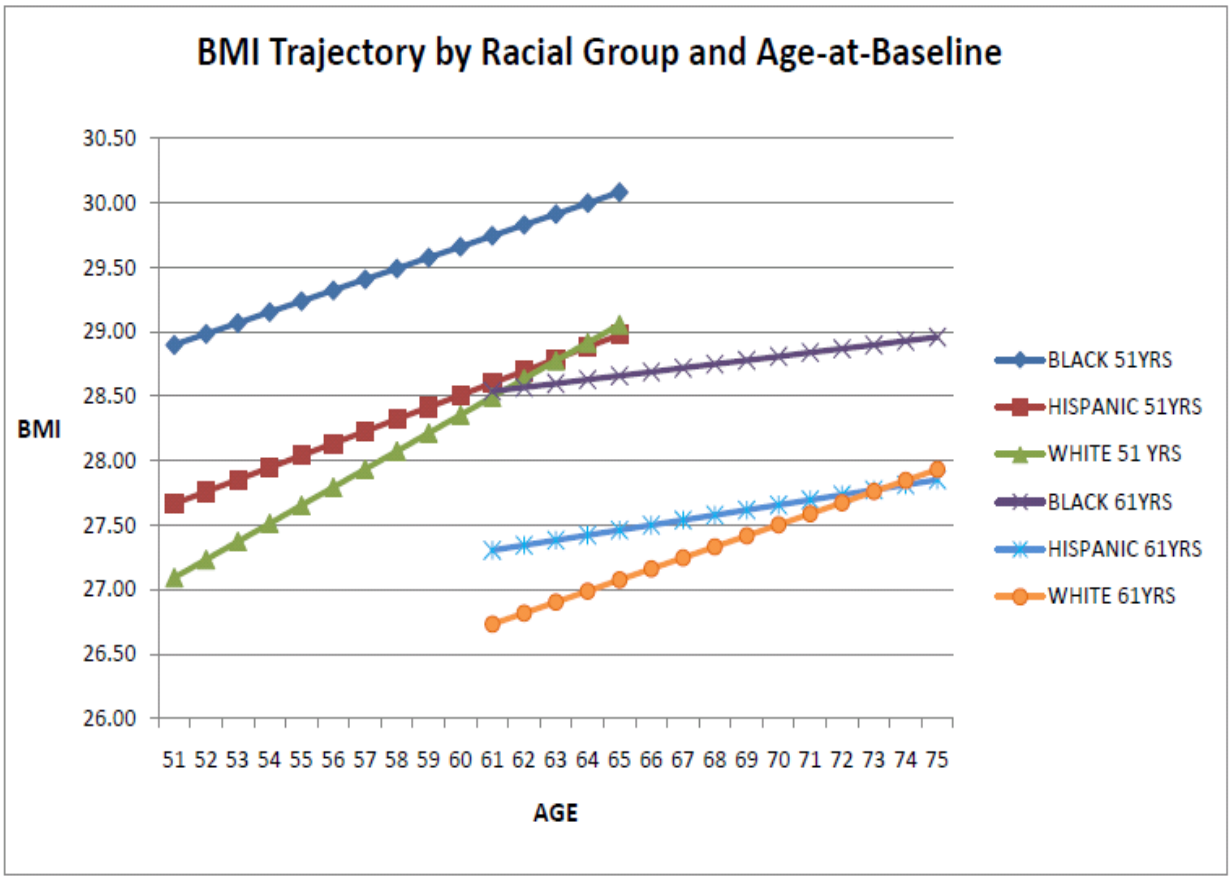
Parameter	Variable	M_0	M_1	M_2	M_3	M_4	M_5
Fixed Effect							
Time-varying variables							
	Proxy		-0.606***	-0.612***	-0.612***	-0.658***	-0.568***
	Marital Status				0.256***	0.285***	0.210***
	Assets				-0.008	0.010	0.013
	Income				-0.095***	-0.078***	-0.059***
	Self-Rated Health					-0.006	-0.003
	Index Disease					0.002	0.001
	NAGI Index					0.241***	0.167***
	CES-D Index					-0.013	-0.019
Intercept	π_0						
	Intercept	27.383***	27.613***	27.370***	27.290***	27.361***	27.517***
	Mortality		-0.436***	-0.531**	-0.610***	-0.713***	-0.690***
	Attrition		-0.686***	-0.790***	-0.754***	-0.708**	-0.197
	Black			1.585***	1.446***	1.417***	0.145
	Hispanic			0.687***	0.221	0.247	-0.144
	Female			-0.146	-0.159	-0.259**	-0.083
	Age (1992)			-0.063***	-0.072***	-0.074***	-0.051***
	Education				-0.099***	-0.085***	-0.009
	BMI (1992)						0.696***
Linear Slope	π_1						

	Intercept	0.047***	0.066***	0.073***	0.075***	0.067***	0.063***
	Mortality		-0.079***	-0.068***	-0.069***	-0.056***	-0.062***
	Attrition		-0.002	0.000	0.001	0.002	-0.018
	Black			-0.053***	-0.054***	-0.056***	-0.008
	Hispanic			-0.047***	-0.046***	-0.047***	-0.032*
	Female			0.010	0.011	0.014	0.007
	Age (1992)			-0.006***	-0.005***	-0.005***	-0.006***
	Education				0.000	-0.000	-0.002**
	BMI (1992)						-0.025***
Quadratic Slope[a]	π_2						
	Intercept	0.002**	0.001	-0.001	-0.001	-0.001	-0.001
Random Effect		VARIANCE	VARIANCE	VARIANCE	VARIANCE	VARIANCE	VARIANCE
Intercept1		20.054***	19.916***	19.510***	19.372***	18.191***	6.835***
Linear		0.074***	0.072***	0.072***	0.071 ***	0.070***	0.060***
Quadratic		0.001***	0.001***	0.001***	0.001***	0.001***	0.002***
Level1 R		7.579	7.558	7.558	7.553	7.525	7.114
AIC		445,993.33	445,607.26	445,411.33	445,326.31	444,765.75	424,447.38

* p< .05, **p< .01, ***p< .001.

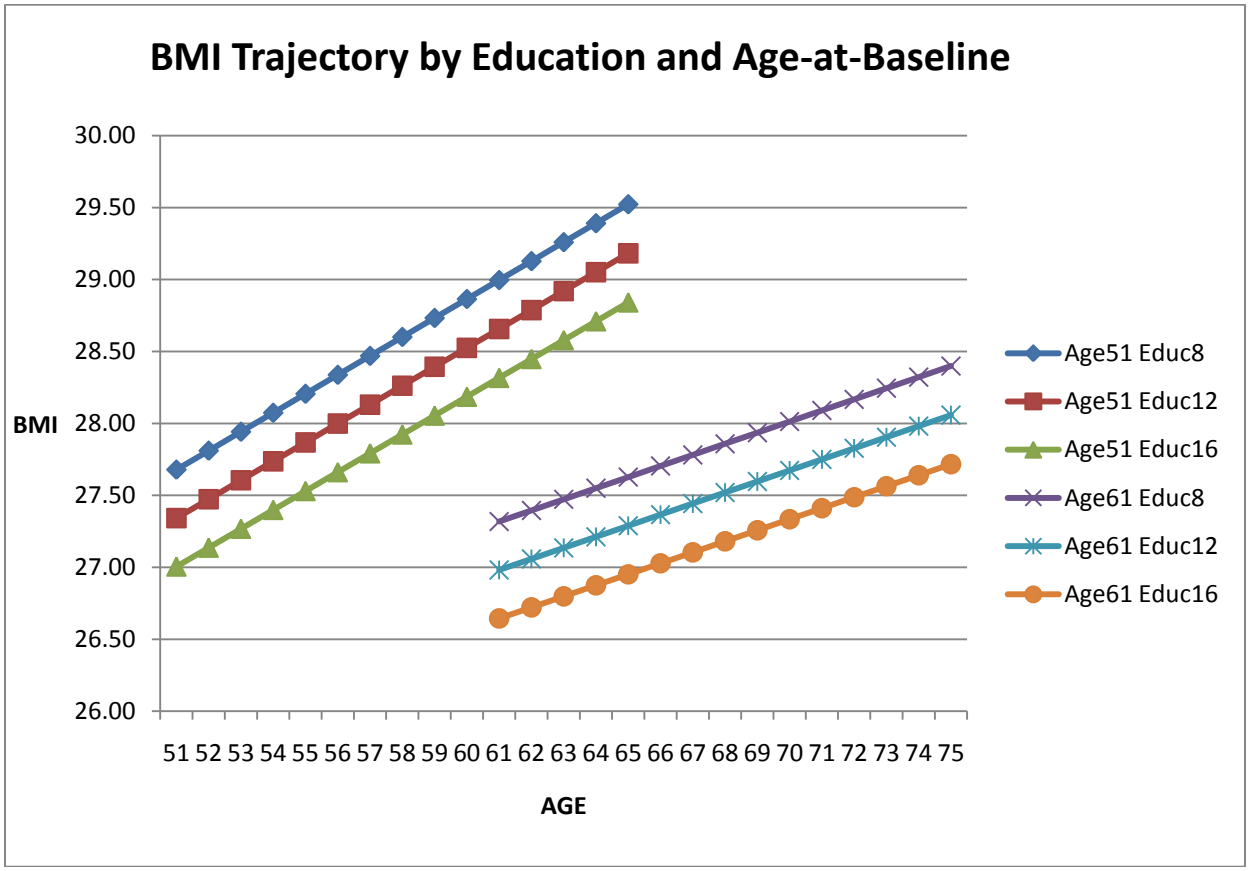
[a] Regression coefficients associated with all covariates on quadratic slope are non-significant in models 1 through 5.

Figure 1: BMI Trajectory by Racial/Ethnic Group and Age-at-Baseline



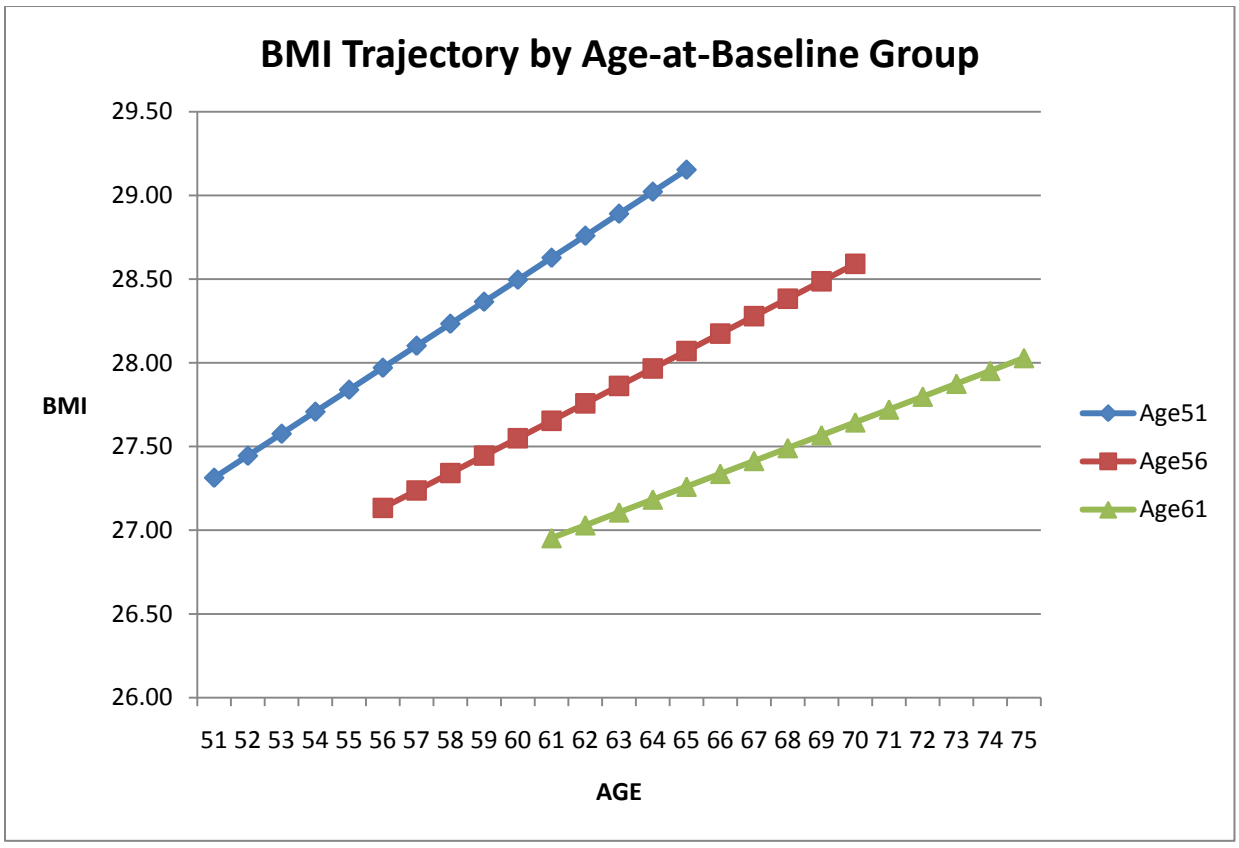
[a] Graph derived based on M_4 (Table 3).

Figure 2: BMI Trajectory by Years-of-Education Completed and Age-at-Baseline



[a] Graph derived based on M_4 (Table 3).

Figure 3: BMI Trajectory by Age-at-Baseline in 1992



[a] Graph derived based on M_4 (Table 3).

Appendix 1: Chapter II

1) Comments on the inclusion of "marital status" as control variable in the models:

The relationship between BMI and marital status is an interesting one and indeed the literature provides evidence of differences in BMI based on marital status, as well as on transitions in marital status (e.g., Sobal, Rauschenbach, & Frongillo, 2003, 1995). We have discussed the possibility of including marital status as a categorical variable (married/separated/divorced/widowed/never married/other), yet we decided against this, based on the following considerations: (1) prior studies of marital status in relation to BMI level or change (e.g., Kahn, Williamson, & Stevens, 1991; Sobal, Rauschenbach, & Frongillo, 2003, 1995) use the two categories "married" and "un-married" (various forms of unmarried grouped under this category), mainly because the theoretical basis for this relationship (e.g., marital role theory, marital selection theory, marital causation theory) provides support for the dichotomous categorization of marital status, (2) marital status was not the main focus of our inquiry, yet it was appropriate to include it as a confounder, due to its links to both BMI and independent variables of interest, and (3) the small number of respondents in categories other than married, divorced and widowed. Further, because we used marital status as a time-varying covariate, the between-wave differences within each sub-category would have been trivial. We felt that in light of existing evidence, the inclusion of a dichotomous marital status variable as a confounder was appropriate, and we did not analyze further the relationship between BMI trajectory and sub-categories of marital status.

2) Potential for expectation bias in interviewer-measured weight and height:

There is a legitimate concern that should the same respondents be measured for weight and height at successive waves, a form of expectation bias in reporting may occur. However, in 2004, the weight and height were measured on a very small random sample of 520 respondents. In 2006, as part of the Biomarkers Data Collection, measured weight and height were collected for a random half of the

households in the HRS sample (7,187 respondents). Only 74 respondents were measured at both waves. In response to the reviewer's concern, we have calculated the differences between self-reported and measured weight and height at both waves (2004 and 2006) for this very small sample (see Table 1A), and found the differences to be *larger* in 2006 compared to 2004. As for the majority of respondents, who were measured at only one wave, the expectation bias should not be an issue - in addressing the controversy about the value of self-reported measures in general, and in the HRS data in particular, David Weir (2008) states that "the self-reports are obtained before respondents are told they will be measured".

Table 1A. Differences between Self-Reported and Measured Weight and Height for the Sub-Sample of Respondents Measured in 2004 and in 2006 (N = 74 respondents).

		2004	2006
Weight (lb.)	Self	183.72	180.24
	Measured	184.82	183.79
	Δ	-1.1	-3.55
Height (inches)	Self	66.78	67.88
	Measured	65.86	65.62
	Δ	0.92	2.26

3) <u>Comments on methodological differences between time-based and age-based analyses and the implications for comparisons of results:</u>

Numerous investigators have undertaken time-based analyses in depicting intrapersonal changes in old age (George & Lynch, 2003; Kelley-Moore & Ferraro, 2004; Taylor & Lynch, 2004). Time-based models specify intra-personal changes as a function of time since a fixed benchmark (usually the beginning of the study)

(Alwin, Hofer, & McCammon, 2006), while in age-based models age, rather than time, is used in estimating the growth parameters. A time-based analysis may yield results different from those of an age-based analysis (see discussion in Liang et al., 2008). To summarize, whereas both approaches correctly identify intrapersonal changes with age (or time in the context of time-based analysis), only age-based analysis correctly identifies cohort effects. Consequently, in a time-based analysis, we can only estimate time or age-related intrapersonal changes while controlling for age differences at the baseline. In this context, age-effects are a combination of cohort- and "true" age-effects. In contrast, given appropriate data (i.e., where members of different cohorts are observed at the same ages over extended periods of time), age-based analyses have the capacity to separate the effects of age and cohort (McArdle & Anderson, 1990). Consequently, a direct comparison of estimates obtained from time-based models (such as in our study) and age-based models (e.g., Grinker et al., 1995) is not warranted.

4) Comments regarding the complicated relation between mortality and BMI development over time. In the following I am describing my line of reasoning regarding the treatment of mortality and attrition and provide evidence to support my decisions.

Treatment of missing data represents a challenge for all surveys, especially longitudinal ones. To draw inferences from the sample under consideration back to the population of interest, assumptions about how the probability of missing data for certain variables is related to baseline covariates and to the outcome of interest have to be made. The fundamental assumption underlying methodological decisions on treatment of missing data is that of "randomness of missing data" (Little & Rubin, 1987, 2002). If the probability of non-response is not related to baseline characteristics or to the outcome of interest, data are "missing completely at random" (MCAR) or "missing at random" (MAR), and listwise deletion and analysis of only those respondents with complete data is appropriate (Little & Rubin, 2002;

Shafer & Graham, 2002; Harel, Hofer, Hoffman, & Pedersen, 2007; Wood, White & Thompson, 2004).

However, this is not the case with mortality in older populations. As the reviewer points out, obesity is associated with higher mortality, those who die tend to lose weight prior to death, and they tend to have worse health status prior to death. Further, mortality in older ages is highly stratified by race/ethnicity and SES (including education) (e.g., Lantz, House, Lepkowski et al., 1998; Smith, Shipley, & Rose, 1990; Sudano & Baker, 2006). Because mortality and selective attrition in older populations are linked to both baseline covariates and to the outcome of interest (in this case BMI), they violate the "missing at random" assumption, and therefore need to be methodologically addressed (Harel, Hofer, Hoffman et al., 2007).

Death is a common and non-random event in gerontological studies. Exclusion of respondents who died results in biased estimates for outcome variables systematically related to mortality (Matthews et al., 2004; Raghunathan, 2004), and undermines inferences to a representative population of aging individuals, because the resulting sample (i.e., the sample excluding those who died) systematically differs from the population from which the original sample was selected (Drivsholm & al, 2006; Harel et al., 2007; Hofer & Hoffman, 2003).

Because mortality and selective attrition violate the "missing at random" assumption, they need to be accounted for in studies of older populations (Hofer & Hoffman, 2007). Two general approaches may be used: pattern mixture models (Hedeker & Gibbons, 1997; Little, 1993, 1995) or selection models (Diggle & Kenward, 1994). In our analysis, we used an approach similar to the pattern mixture models, where sub-groups are identified based on patterns of missing data and membership indicators are included in the models; as such, mortality and attrition were treated as confounding variables, and their inclusion was to adjust for selection bias. This is similar to the approaches used by other investigators in dealing with selection bias (Heckman, 1979; Liang et al., 2010, 2008; Mroczek &

Spiro, 2005). Further, consistent with the recommendation from Harel et al. (2007), we allowed for a distinction between mortality (validated in HRS through the National Death Index) and other-than-mortality attrition (vital status "alive" ascertained in HRS) , by including two distinct indicators. Data limitations precluded consideration of selection modeling techniques, which require that the manner in which the probability of missingness is related to outcomes be specified.

5) Definition of Hispanic population in HRS:

The questions ascertaining "race/ethnicity" in HRS follow the single-race categories approach (Liebler & Halpern-Manners, 2008), such that each individual was uniquely assigned to one of the three mutually exclusive racial groups used in our study. This was done in the following sequence: respondents are asked (1) "do you consider yourself Hispanic or Latino?", (2a) those who answer "yes" were asked follow up questions about the Hispanic group (Mexican American, Puerto Rican, Dominican, Cuban or Other) they consider themselves to belong to (again, one group only allowed), and (3) those who answer "other Hispanic" are asked a follow up question on other Hispanic groups (Spanish, Central America, South America). Those who answered "no" on being asked "do you consider yourself Hispanic or Latino?", were asked follow up questions about defining themselves as "non-Hispanic White" or "non-Hispanic Black". As discussed by Liebler and Halpern (2008), this method uses "self-identification" as the predetermined assignment rule (Parker & Makuc, 2002).

In the HRS sample, 1173 respondents answered "yes" to the question on being Hispanic/Latino. Among the 1173 respondents, more than half (60.5%) or 710 defined themselves as Mexican-Americans, 99 as Puerto Ricans, 116 as Cubans, 26 as Dominicans and the rest as "other" (please see Appendix for details on survey questions pertaining to "race/ethnicity"). In our study, all these respondents were uniquely assigned to the group coded as "Hispanic". This approach is very similar to other studies linking ethnicity and health. For example, Abraido-Larza et al. (2005) studied the effect of acculturation measures on health behaviors among Latinos,

where *Latino ethnicity* was assigned based on self-reported Puerto Rican, Cuban, Mexican, Mexican American, Chicano, other Latin American, or other Spanish national origin or ancestry. This approach is frequently warranted (as is our case) by the very small number of respondents in categories other than Mexican Americans.

In my discussion, I explicitly acknowledge that combining the Latino groups into one over-arching category underestimates cultural and behavioral heterogeneity within this group and potentially reduces or eliminates some of the differences in BMI between Hispanics and Whites/Blacks. Further, in the limitations section, I offer this as a limitation of the study and state that better data are needed for future studies to supplement our findings with findings on specific BMI trajectories within Hispanic sub-groups.

6) Age-related decline in health and the issue of healthy survivor bias:

To address the possibility that unfavorable health status explained at least partially a lower BMi in a subset of sick older adults, various measures of health status were included as time-varying covariates in the analyses. Racial/ethnic and gender differences in age-related health decline (e.g., Liang et al., 2008, 2010) have been reported and major health conditions may result in weight loss (e.g., Mehta & Chang, 2009). As such, similar to mortality, health status is systematically related to both baseline respondent characteristics and to the outcome of interest and needs to be accounted for as a potential confounder. Further, disease presence may act as a biologic mediator between socio-demographic characteristics and the health outcome of interest (Seeman et al., 2004). We include various measures of health status in M_5 (Table 3), and find that only NAGI is significantly associated with trajectory coefficients. Our approach is similar to that frequently employed in studies of mortality attributable to obesity (e.g., Mehta & Chang, 2009; Myrskylä & Chang, 2009; Thorpe & Ferraro, 2004), which include health status measures as confounders or mediators. For example, Thorpe and Ferraro (2004) conclude that the association between obesity and mortality in individuals older than 50 is

entirely mediated by health status; interestingly, this did not hold true for individuals younger than 50, in which the association remains strong even after control for health status.

7) Comments on the exclusion of other (older) cohorts from these studies:

Decision not to include older cohorts was based on the following considerations:

7.1. Strong cohort effects in body-weight have been reported, with younger cohorts being heavier and gaining weight faster than older cohorts (e.g., Clarke et al., 2009; Reynolds & Himes, 2007). As we explain in the Methods section, one of the reasons to include only this cohort, to the exclusion of younger and older cohorts, was an attempt to methodologically rule out the effect of cohort membership on BMI trajectory.

7.2 The explicit goal in this study was to examine the trajectory of BMI starting in middle age. Changes in body-weight in older age are complex; I also acknowledge the possibility that the trajectory of BMI in older cohorts may vary substantially from that in younger cohorts. As such, I believe that describing multiple cohort-based trajectories and identifying the socio-demographic basis for differences among these trajectories cannot be satisfactorily accomplished within the space (word) limitations of a single article. We are currently in the process of analyzing the data for a study aiming to describe differences between younger (WB), middle age (HRS cohort) and older (CODA and AHEAD) cohorts from the Health and Retirement Study and to identify what predicts differences in trajectories.

7.3. Nevertheless, conclusions about the development on BMI during the late seventh and early eight decade of life can be drawn from our results. In our study, a substantial sub-sample is followed past the age previously believed to represent the peak or turning point in body weight (~65 years old) (Hardy & Kuh, 2006). A total of 5,299 respondents (51.4%) were 56 - 61 years old at baseline. They were followed for 14 years, up to age 70 – 75 in 2006 (end of

study). Our results showing that, after control for multiple potential confounders (including mortality and attrition), BMI continues to increase past the point previously thought to represent the peak body-weight and well into the eighth decade of life, are consistent with a number of recent studies. For example, Barone et al. (2006) found that cohort and selective survival account for the peak at age 65, such that the weight gain continues even in older ages. Similar conclusions have been reached by other researchers (Jacobsen et al., 2001; Guo et al., 1999).

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CHAPTER III: THE EFFECT OF STABILITY AND CHANGE IN HEALTH BEHAVIORS ON TRAJECTORIES OF BODY-MASS INDEX IN MIDDLE AGED AND OLDER AMERICANS

III.1. BACKGROUND

Overweight and obesity are increasingly prevalent among older Americans. Over 70% of Americans aged 60 years and older are overweight and one-third are obese (Flegal, Carroll, Ogden, & Curtin, 2010). In older adults, overweight and obesity have been associated with increased mortality (Berrington de Gonzalez et al., 2010; Thorpe & Ferraro, 2004), physical and cognitive morbidity (Field et al., 2001; Peeters, Bonneux, Nusselder, De Laet, & Barendregt, 2004), and reduced active life expectancy (Reynolds, Saito, & Crimmins, 2005); obesity also contributes to higher utilization of healthcare services (Leon-Munoz et al., 2005) and accounts for approximately half of the total $147 billion in obesity-attributable annual US medical expenditures (Finkelstein, Trogdon, Cohen, & Dietz, 2009). Reducing the risk factors for and the consequences of excess weight in older adults should therefore be a critical public health goal.

Smoking (Appel & Aldrich, 2003; Fillenbaum, Burchett, Kuchibhatla, Cohen, & Blazer, 2007), physical inactivity (DiPietro, 2001), and alcohol use (Arif & Rohrer, 2005a) are significant health issues among older adults. Existing studies indicate that smokers are leaner than non-smokers or ex-smokers (John, Hanke, Rumpf, & Thyrian, 2005; Kaplan, Huguet, Newsom, McFarland, & Lindsay, 2003; Sulander, Rahkonen, Nissinen, & Uutela, 2007) and that smoking cessation results in a substantial increase in weight and body fat (Pisinger & Jorgensen, 2007). Older adults' participation in physical activity results in a dose-dependent decrease in body-weight and fat-mass (Irwin et al., 2003; Janiszewski & Ross, 2007), yet the

prevalence of high-intensity physical activities decreases and reported inactivity increases with age, especially among women (DiPietro, 2001). The relationship between alcohol-use and body-weight in this age group has not been systematically studied (Eigenbrodt et al., 2001). In younger groups, a J-shaped relationship has been reported, such that light to moderate drinking is associated with a lower body-weight, whereas heavy or risky drinking has the opposite effect(Arif & Rohrer, 2005b). This inverse relationship is more prominent among women, while the results among men remain inconclusive (Colditz et al., 1991; Liu, Serdula, Williamson, Mokdad, & Byers, 1994).

Our knowledge on health risk behaviors and body-weight in older age can be advanced in at least three respects. First, a recent study has shown that starting in middle age the BMI follows an increasing linear trajectory (Botoseneanu & Liang, 2010), yet to the best of our knowledge, no study to date has examined the impact of health behaviors on the *trajectory* (i.e., intercept and rate-of-change) of body-weight over an extended period of time. The literature on health behaviors and body-weight consists mostly of cross-sectional or two-points transition (weight gain or loss) studies, which provide little understanding of the effect of health behaviors on the *trajectory* of body-weight (Rogosa, 1988).

Second, most epidemiological studies assume a rather stable health lifestyle and do not account for fluctuations in behaviors over time (Mulder, Ranchor, Sanderman, Bouma, & Van Den Heuvel, 1998) or for the observed intra-individual clustering of risky health behaviors (high cigarette consumption, high alcohol intake, and low physical activity) (Chiolero, Wietlisbach, Ruffieux, Paccaud, & Cornuz, 2006; Sneve & Jorde, 2008). Health behaviors are hardly invariable over time (i.e., smokers or drinkers who quit and relapse) (Filozof, Fernandez Pinilla, & Fernandez-Cruz, 2004), especially in old age. Hence, the concomitant incorporation of multiple health behaviors as time-varying covariates is conceptually justified.

Finally, over the last few decades the prevalence of smoking has declined steadily (Appel & Aldrich, 2003; CDC, 2009), as the reported rates of involvement in physical

activities have increased among all age-groups, including older adults (DiPietro, 2001). The strong correlation over time between the rise in obesity and the reduction in smoking in particular has led to suggestions of a causal relationship (Rashad & Grossman, 2004). While population-level studies have examined this link (K. M. Flegal, 2007; Gruber & Frakes, 2006), no study to date has evaluated whether the individual-level association between obesity-related behaviors and body-weight increased or decreased over time.

We have two main aims for this study. First, we offer quantitative estimates of the effects of smoking, physical activity, and alcohol use *status* and *over-time variation* on the long-term trajectory of body-mass index (BMI) starting in middle age. Second, we examine whether the intra-individual association of aforementioned health behaviors with BMI trajectory changed over the period of time under consideration (1992-2006).

III.2. METHODS

Study Population

We analyzed the effect of smoking, physical activity and alcohol-use on BMI trajectory using longitudinal data from a national sample of Americans aged 51 to 61 at baseline, followed for a period of 14 years (1992-2006). Data came from the Health and Retirement Study (HRS documentation at: http://hrsonline.isr.umich.edu/). Consistent with the purpose of this study and to minimize the potential for cohort differences (Reynolds & Himes, 2007), we chose to analyze only the original HRS cohort (birth years 1931-1941). The HRS cohort consists of 13,565 individuals, who were interviewed once every 2 years from 1992 to 2006, for up to 8 repeated observations. The response rates range from 81.7% (1992) to 89.15 (in 1994), with 55.7% of the respondents completing all 8 interviews. When physical or cognitive limitations precluded participation, a proxy interview was conducted. The rates of proxy interviews range from 4.8% (1992) to 9.0% (2002). As of 2006, the cumulative mortality rate, validated through the

National Death Index (NDI), was 19%. We excluded 3,116 (22.9%) cohort-ineligible spouses (born before 1931 or after 1941) and 135 (0.9%) individuals who did not respond to the health assessment survey sections. This resulted in a final analytic sample of 10,314 individuals with an average of 6.4 interviews.

Measures

Body-mass index (BMI). Self-reported weight was recorded at each wave; height was self-reported at baseline (1992) and verified in the second wave (1994). Body-mass index (BMI) was calculated as BMI = [Weight (lb)/Height (inches) ^{2}] x 703, using current weight and initial height.

Health behaviors assessment. Current *smoking* and *alcohol use* status were recorded at each wave. Participants were classified into "non-users" (= 0) or "users" (= 1) at all time points for each of the two behaviors. The frequency of *vigorous physical activities* (i.e., aerobics, running, swimming, or bicycling) was assessed at each wave. Because the coding for physical activities varied slightly across waves, these indicators were re-coded into binary measures, with a score of 0 for "once per week or less" and 1 for "more than once per week" for all time points.

Changes in smoking, alcohol use and physical activity between two adjacent waves were captured by a difference score (i.e., the difference between current (t_i) and previous observation (t_{i-1})) calculated for each wave (i.e., -1 = cessation, 0 = no change, 1 = initiation) (aggregate rates shown in Table 1).

[Table 4 about here]

Other covariates. The social stratification of aging and health framework (House, Lantz, & Herd, 2005) suggests that exposure to health risks and protective factors, including health behaviors, is conditioned by race (Williams & Jackson, 2005), age (Riley, 1987), gender (Bird & Rieker, 1999), socio-economic status (SES) (i.e., income, education, occupation) (Wray, Alwin, & McCammon, 2005), and marital status (Wyke & Ford, 1992). Further, because poor health behavioral risk profiles

are clustered among the low socio-economic status groups (Lynch, Kaplan, & Salonen, 1997; Yang, Lynch, Schulenberg, Roux, & Raghunathan, 2008), we included several socio-demographic and economic covariates in our analyses. Age-at-baseline, gender (1 = female, 0 = male), race/ethnicity (i.e., non-Hispanic White – reference group, non-Hispanic Black, and Hispanic), and education (years of education completed) were measured at baseline (1992) and verified in subsequent waves. Time-varying socio-economic covariates were also included: total household income, total household assets, and marital status (1 = married/living with a partner, 0 = single/divorced/widowed/separated). As the distributions of both *income* and *assets* were highly skewed, the variables were categorized (quartiles) for all analyses (*shown in Supplementary Data Appendix*).

Because health events may trigger changes in health behaviors (Keenan, 2009; Salive et al., 1992) and to account for the potential confounding effects of health status on BMI (i.e., healthy survivor bias) (Mehta & Chang, 2009), measures of physical and mental health were included as time-varying covariates: *index of chronic diseases* (count of seven chronic conditions – heart disease, stroke, high-blood pressure, diabetes, arthritis, chronic lung disease, and cancer; range = 0 - 7), *self-rated health* (single-item rating of respondent's health; range = 1 (excellent) -5 (poor)), *Nagi index of functional limitations* (Nagi, 1979) (count of six items representing reported difficulties with common activities; range = 0 - 6), and *CES-D (depression) score* (Radloff, 1977) (count of nine items from the Center for Epidemiological Studies Depression Scale; range = 0 - 9).

Sample time-varying (Table 5) and time-constant characteristics (Table 6) are presented below.

[Tables 5 and 6 about here]

Data analysis

Hierarchical linear models (HLM) (Raudenbush & Bryk, 2002) were used to estimate the trajectory of BMI from 1992 to 2006. HLMs are well-suited for studies

of individual changes over time, using repeated measures of a construct to estimate a growth trajectory defined by intercept (starting point) and slope (rate-of-change). Intra-individual changes in BMI were specified as follows (Level 1 equation):

$$Y_{iT} = \pi_{0i} + \pi_{1i}Time + \Sigma\pi_{ki}X_{kiT-1} + \varepsilon_{iT} \quad (1)$$

where Y_{iT} is the BMI of individual *i* at time *T*; π_{0i} is the intercept of BMI for individual *i,* and π_{1i} is the slope (intra-personal rate-of-change) in BMI for individual *i* over time; *Time* is the distance (in years) of assessment from baseline; X_{kiT} represents *lagged* time-varying covariates (e.g., smoking, alcohol use, physical activity) associated with individual *i* at time *(T-1)*; and π_{ki} represents the effect of X_k on individual *i*'s *BMI*. ε_{iT} is the random error in BMI for individual *i* at time *T.*

We employed a time-based analysis approach (Alwin, Hofer, & McCammon, 2006) (i.e., intra-personal changes in BMI modeled as a function of time), with control for inter-personal age-at-baseline (Liang et al., 2008) and socio-demographic differences. The analysis evaluated linear, quadratic, and cubic patterns of change in BMI over time. Time was centered at its mean to minimize the possibility of multicollinearity when estimating non-linear time functions; consequently, the intercept for any given model should be interpreted as the BMI at the mean follow-up time. To ensure a clear time-sequencing between dependent and independent variables, each time-varying covariate was represented by two distinct measures: a *lagged measure* (i.e., observation from the previous wave (t-1)) and a *change term* (i.e., difference between current (t) and previous observation (t-1)). To assess whether the effect of each health behavior on BMI trajectory varied over time, we created interaction terms involving the lagged measure and change score with time (e.g., (smoking * time) and (Δ smoking * time)) .

Time-constant covariates, such as race/ethnicity, gender, education, and age-at-baseline are included in the Level 2 equation (inter-individual variations model):

$$\pi_{pi} = \beta_{p0} + \Sigma\beta_{pq}X_{qi} + r_{pi} \quad (2)$$

where X_{qi} is the qth time constant covariate associated with individual i, and β_{pq} represents the effect of variable X_q on pth growth parameter (π_p). r_{pi} is a random effect and has a mean of 0.

In longitudinal studies of health in older populations, mortality and attrition (Mroczek & Spiro, 2005; Murphy et al., 2011) are potential confounders, because they are non-random events correlated with both BMI (Mehta & Chang, 2009) and poor health (Harel, Hofer, Hoffman, & Pedersen, 2007). Similarly to pattern-mixture modeling (Hedeker & Gibbons, 2006), we included indicators for mortality (1 = died during study period, 0 = alive at the end of study) and attrition (1 = attrited for reasons other than mortality and did not return, 0 = completed the study period) in the Level 2 equations. Finally, time-varying proxy status was represented by a lagged measure (i.e., proxy status at previous wave; 1 = proxy respondent and 0 = self) and a change term (i.e., change in proxy status from previous wave).

To minimize the loss of participants due to item missing (Little & Rubin, 2002; Schafer & Graham, 2002), three complete Level 1 and Level 2 data sets were imputed using the NORM software(Schafer & Olsen, 1998). Parameter estimates and their standard errors were calculated by averaging across the three imputed data sets and adjusting for their variance (Raudenbush & Bryk, 2002).

The HRS involved the over-sampling of Blacks, Hispanics and respondents from Florida. We ran all models with and without respondent-level sampling weights and obtained similar results. Consequently, we chose not to weight the data. This approach is also warranted because many of the attributes on which differential selection weights are calculated (e.g., race, gender, marital status) are explicitly controlled for in our multivariate analyses, making unweighted ordinary least squares estimates less biased than, and as such preferable over weighted estimates (Winship & Radbill, 1994).

Finally, because of the large HRS sample, a two-sided $P < 0.01$ was considered to represent statistical significance. All analyses were performed using HLM version 6.0 (Scientific Software International, Lincolnwood, Illinois).

III.3. RESULTS

Changes in BMI over time

Using linear, quadratic and cubic functions, we mapped the trajectory of BMI between 1992 and 2006. BMI increased approximated by a quadratic function, with an intercept of 27.569 ($P < 0.001$), linear slope of 0.048 ($P < 0.001$) and a quadratic slope of 0.002 ($P < 0.01$) (M_1; Table 7). The cubic slope coefficient was not significant and it was not included in subsequent analyses. The quadratic slope became non-significant after adjustment for socio-demographic and health status measures, indicating a linearly increasing BMI trajectory (M_3, M_4; Table 7).

[Table 7 about here]

Health behaviors status and changes

Smoking, alcohol use, and vigorous physical activity, and initiation of each activity, were all significantly associated with lower BMI over time. Smokers had a lower BMI compared with non-smokers (b = -1.263, $P < 0.001$; M_1) and smoking cessation was associated with higher BMI over time (b= -1.048; $P < 0.001$; M_1) (Figure 4). Individuals engaged in vigorous physical activity (b = -0.549, $P < 0.001$; M_1) and those who initiated physical activity (b = -0.381, $P < 0.001$; M_1) had lower BMI over the observation period (Figure 5). Finally, alcohol users (b = -0.329, $P < 0.01$; M_1) and those who initiated drinking (b = - 0.208, $P < 0.001$; M_1) had lower BMI over time (Figure 6) compared with non-users and with quitters.

[Figures 4-6 about here]

With the exception of smoking status, the effects of health behaviors on BMI remained constant over time. The negative effect of smoking on BMI increased with

time (b = -0.047, $P < 0.001$; M_2). The same applies to *change* in smoking status (b = -0.038, $P < 0.001$; M_2). In contrast, the effects of the lagged measures of physical activity and alcohol use and their changes on BMI did not differ significantly over time.

To what extent are the results confounded by socio-economic heterogeneity and health status differences? The effects of alcohol use and its change over time on BMI appear to be explained by heterogeneity in socio-demographic attributes and health status, as their negative effects on BMI were no longer significant when socio-demographic and health status were controlled (M_3). In contrast, the effects of smoking and physical activity remained significant, though attenuated (M_3). Interestingly, the time-interaction effects of smoking and its change on BMI increased when population heterogeneity was taken into account (M_3 compared with M_2; Table 4).

Socio-economic and health status covariates

Individuals who were older at baseline had a lower BMI (b = -0.063, $P < 0.001$; M_4) and a lower linear rate-of-change (b = -0.008, $P < 0.001$; M_4) compared with younger individuals. Women had lower BMI than men (b = -0.163, $P < 0.01$; M_4), with no significant differences in the linear rate-of-change. There were also racial/ethnic and educational differences in BMI over time, although they were no longer significant when health status measures and baseline BMI were included in the model (M_4). Among the health measures considered, only the Nagi Index of functional limitations was associated with a higher BMI level (b = 0.135, $P < 0.001$, respectively b = 0.156, $P < 0.001$; M_4).

Mortality, attrition, and proxy status

The results confirm that BMI trajectory parameter estimates would be biased should mortality, attrition and proxy status measures not be included as potential confounders in the analysis. Those who died during the follow-up period had a lower BMI (b = -0.611, $P < 0.001$; M_4) and a lower rate of weight gain (b = -0.052, $P <$

0.001; M_4) prior to death. Attrition (b = -0.632, $P < 0.001$; M_1), proxy status (b = -0.694, $P < 0.001$; M_1) and *change* in proxy status (b = -0.629, $P < 0.001$; M_1) were associated with a lower level of BMI over time; the difference due to attrition appeared to be explained by population heterogeneity (M_4).

III.4. DISCUSSION

Modification of health behaviors has long been advocated as a solution to the growing problem of overweight/obesity. This study assesses the effect of changes in smoking, physical activity, and alcohol use on the long-term *trajectory* of BMI starting in middle age. To our knowledge, this is the first study of its kind. Analyzing the trajectory of BMI is important for understanding intra- and inter-personal variations in body-weight over time. Because it is often the persistence or variability of lifestyle behaviors that influence health status (Eigenbrodt et al., 2001), trajectories are useful outcome measures, in that they allow for multiple transitions, each distinguished by a specific level, direction, and rate-of-change.

Our results show that smoking was associated with a downward BMI trajectory, while smoking cessation resulted in an increase in BMI over time. The magnitude of their effect is far from trivial. The regression coefficient for smoking (between 1.05 and 1.4 BMI units lower for smokers versus non-smokers) corresponds to a moderate effect of between 0.2 to 0.4 standard deviations in BMI (Olejnik & Algina, 2000) and translates into an actual difference of about 8 pounds for an average 5 feet 6 inches (5'6") tall individual. Further, a significant increase in BMI over time is observed with smoking cessation - BMI increases by 1.1 units after smoking cessation, an actual weight gain of 6 pounds for an average 5'6'' tall individual. These findings are consistent with previously reported correlations between body-weight and tobacco use in the elderly (Haapanen-Niemi et al., 2000; Kruger, Ham, & Prohaska, 2009). Yet, our specifications are more dynamic in that the trajectory of BMI was defined as a function of lagged measures of health behaviors and repeated changes between two points in time. It is important to consider multiple changes and subsequent reversals in health behaviors over time (i.e., initiation, cessation,

and relapse), to accurately represent the instability of actual lifestyles (Mulder et al., 1998), especially in older ages, when adverse life and health events have the potential to trigger sudden and compound changes in health behaviors (Keenan, 2009; Sachs-Ericsson et al., 2009).

Older adults engaged in vigorous physical activity and those who initiated physical activity had a lower BMI compared with those consistently inactive. Previous studies report that reductions in physical activity levels, especially when coupled with smoking cessation, result in weight gain and a metabolically-adverse body composition profile (Pisinger & Jorgensen, 2007). Our results show an analogous effect of smoking and physical activity, and of changes thereof, on long-term body-weight development, and substantiate previous findings suggesting that initiation or increase in physical activity levels attenuate the degree of weight gain after smoking cessation (Froom, Melamed, & Benbassat, 1998). Even infrequent (once per week) engagement in vigorous physical exercise, considerably lower than the amount currently recommended for older adults (Nelson et al., 2007), can yield weight control benefits. Nevertheless, the difference between smoking and physical activity coefficients (approximately three times higher in absolute values for smoking) implies that a considerable increase in activity levels is needed to counter the weight gaining effect of smoking cessation (Figure 7).

[Figure 7 about here]

Alcohol users and those who initiated drinking had significantly lower BMI over time. However, these effects appear to be confounded by differences in health status. Our findings are similar to prior observations that alcohol users have lower BMI compared with abstainers (A. A. Arif & Rohrer, 2005b) and that subjects with poorer health may be more likely to drink in the first place (O'Connell, Chin, Cunningham, & Lawlor, 2003). An alternative explanation is that alcohol consumption acts as a proxy for other deleterious health behaviors. We explored this alternative by examining additional models (results not shown; *provided in Supplementary Data Appendix*), which showed that smoking and physical activity,

but not socio-economic or health measures, render the effects of alcohol use non-significant. This supports our assertion that the observed clustering of risky health habits (Chiolero et al., 2006; Kruger et al., 2009) requires that multiple behaviors be considered simultaneously and underscores the need to account for pre-existing morbidity status in studies of health behaviors and body-weight in older populations.

An often-raised conjecture is that the observed declines in smoking and physical labor across all demographic groups over the last 40 years may explain the concomitant increase in the prevalence of obesity (Cutler, Glaeser, & Shapiro, 2003; Philipson & Posner, 2003; Rashad & Grossman, 2004). Yet, studies find little support for this association at the population level (K. M. Flegal, 2007; Gruber & Frakes, 2006). At the individual level, we show that the negative effects of smoking and smoking cessation on BMI increased with time, while the effects of physical activity and alcohol use remained constant. This suggests that individuals who quit smoking nowadays may expect to gain more weight than 14 years ago. While it is outside the scope of our study to explore the causes underlying these findings, they should not discourage efforts to reduce smoking among older individuals. Smoking cessation in older ages has multiple health benefits (Appel & Aldrich, 2003) and prevention of weight gain in ex-smokers is feasible (John et al., 2005). Especially among individuals of lower socio-economic status, tobacco consumption is clustered with other risk habits known to favor weight gain (e.g., poor diet) (Chiolero, Faeh, Paccaud, & Cornuz, 2008; Kruger et al., 2009). Currently, most smoking cessation programs do not encourage simultaneous attempts at weight control through dietary modifications or increased physical activities, partly because interventions aimed at concurrently changing several health behaviors have not been successful (Copeland, Martin, Geiselman, Rash, & Kendzor, 2006). Yet, as shown for type-2 diabetes (Tuomilehto, 2005) and hypertension (Elmer et al., 2006), effectively addressing these factors could counterbalance the weight gaining effect of smoking cessation.

Health behaviors are differentially distributed by socio-economic characteristics, with a pooling of unhealthy behaviors at the bottom of the socio-economic hierarchy (Lynch et al., 1997); there is also more resistance to changing health lifestyles among those in lower socio-economic positions (Honda, 2005). As such, we adjusted our analyses to control for socio-economic differentials. The coefficients for both smoking and physical activity measures were slightly modified (increased and respectively decreased) but remained significant, suggesting that, although social heterogeneity needs to be considered, the effects extend to all the groups considered in our study. To check whether the effects of health behaviors on BMI differ by race/ethnicity, gender or education, we tested the appropriate interaction effects and found them to be non-significant (results not shown; *provided in Supplementary Data Appendix*). Tempting as it may be to propose behavioral interventions as a way to reduce health inequalities in late life, the evidence is less than encouraging (Lantz et al., 2001). Nevertheless, even without a lessening of social health disparities, modifications of risky health behaviors are a worthy public health goal, as they may provide health and weight control benefits across the social divide.

Several limitations of our study should be noted. Given that individuals tend to over-estimate their height and under-estimate their weight (Nawaz, Chan, Abdulrahman, Larson, & Katz, 2001), BMI calculations based on self-reported measures are likely to understate the "true" BMI. However, if the bias in reporting is consistent over time, and there is no indication in the literature to the contrary, the analysis should yield valid estimates of changes in BMI. Further, we compared self-reported and interviewer-measured weight and height (available for 2004 and 2006), and, consistent with other studies (Weir, 2008), found only small differences (results not shown; *provided in Supplementary Data Appendix*). Second, BMI may not be an adequate measure of overall and abdominal fatness in elderly subjects (Prentice & Jebb, 2001; Seidell & Visscher, 2000). Differential effects of smoking (Canoy et al., 2005; Pisinger & Jorgensen, 2007; Simon, Seeley, Lipschutz, Vittinghoff, & Browner, 1997) and physical activity (Raguso et al., 2006) on body

composition measures have been identified. Further research is required to assess the trajectories of body composition indicators and their association with health behaviors. Finally, measures of health behaviors could be refined further. We undertook separate analyses using the number of cigarettes-per-day and the number of drinks-per-day and found that they do not improve upon the dichotomous measures presented here (results not shown). We are encouraged that even the crude dichotomous measure of physical activity predicted a considerable decrease in BMI over time. However, more refined measures of physical activity, such as type of activity, intensity, or duration, should be tested in their effect on body-weight trajectory.

The value in disease prevention among older adults is not sufficiently recognized. Reducing obesity back to the 1980s levels in people over age 65 may yield vast improvements in morbidity with cost savings of over $1 trillion by 2030 (Goldman, Cutler, Shang, & Joyce, 2006). Nonetheless, older age groups have largely been excluded from trials aimed at reducing health risk behaviors (Levy, Kosteas, Slade, & Myers, 2006). Hence, the impact of changes in behavioral profiles on various health outcomes, including body-weight trajectories, is not well understood. We expand current knowledge by quantifying the effects of health behaviors, and changes thereof, on the 14-year trajectory of BMI in middle and older age adults, offer support for prior observations that body-weight is a modifiable outcome, and suggest ways to achieve beneficial modifications.

Table 4: Stability and Change in Health Behaviors, 1992-2006

	Smoking	Physical Activity	Alcohol Use
Stable (no change)	92.3%	68.4%	86.9%
Cessation	5.0%	15.7%	7.4%
Initiation	2.7%	16.0%	5.7%

[a] Weighted distributions; case weights are respondent-level weights obtained from the Cross-Tracker 2006 HRS file.
[b] Sample N =10,314.

Table 5: Descriptive Statistics for Time-varying Covariates

	1992	1994	1996	1998	2000	2002	2004	2006
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
BODY-MASS INDEX (BMI)	26.98 (4.96)	27.11 (4.95)	27.23 (5.05)	27.30 (5.05)	27.47 (5.15)	27.59 (5.22)	27.63 (5.32)	27.97 (5.43)
PHYSICAL ACTIVITY (lag)	0.32 (0.47)	0.33 (0.47)	0.58 (0.41)	0.53 (0.50)	0.49 (0.50)	0.51 (0.50)	0.47 (.050)	0.42 (0.49)
Change[c] in Physical Activity		0.01 (0.29)	-0.17 (0.63)	-0.04 (0.54)	-0.01 (0.55)	-0.03 (0.54)	-0.06 (0.57)	-0.04 (0.53)
SMOKING STATUS	0.40 (0.49)	0.40 (0.49)	0.24 (0.43)	0.22 (0.41)	0.19 (0.39)	0.17 (0.38)	0.15 (0.36)	0.14 (0.35)
Change in Smoking Status		-0.16 (0.41)	-0.01 (0.25)	-0.03 (0.25)	-0.02 (0.23)	-0.02 (0.23)	-0.01 (0.23)	0.06 (0.35)
ALCOHOL USE	0.63 (0.48)	0.64 (0.48)	0.58 (0.49)	0.55 (0.50)	0.53 (0.50)	0.51(0.50)	0.52 (0.50)	0.51 (0.50)
Change in Alcohol Use		-0.06 (0.37)	-0.03 (0.39)	-0.02 (0.39)	-0.02 (0.39)	-0.01 (0.40)	-0.01 (0.40)	0.02 (0.39)
ASSETS (lag)	2.46 (1.21)	2.48 (1.19)	2.51 (1.19)	2.55 (1.20)	2.58 (1.21)	2.61 (1.21)	2.63 (1.20)	2.66 (1.18)
Change in Assets		0.03 (0.88)	0.01 (0.83)	0.02 (0.80)	0.02 (0.78)	0.01 (0.77)	0.01 (0.75)	0.01 (0.73)
INCOME (lag)	2.57 (1.11)	2.60 (1.11)	2.61 (1.11)	2.58 (1.11)	2.60 (1.10)	2.61 (1.09)	2.63 (1.09)	2.64 (1.10)
Change in Income		0.02 (0.88)	-0.02 (0.86)	0.01 (0.84)	0.01 (0.83)	0.01 (0.82)	-0.01 (0.83)	0.01 (0.81)
PROXY STATUS (lag)	0.05 (0.21)	0.04 (0.19)	0.07 (0.25)	0.07 (0.26)	0.08 (0.27)	0.09 (0.29)	0.09 (0.28)	0.07 (0.26)

Change in Proxy Status		0.02 (0.18)	-0.01 (0.24)	0.01 (0.22)	0.01 (0.22)	0.01 (0.24)	-0.01 (0.21)	-0.02 (0.02)
MARITAL STATUS (lag)	0.77 (0.42)	0.78 (0.42)	0.75 (0.43)	0.74 (0.44)	0.72 (0.45)	0.71 (0.45)	0.71 (0.46)	0.69 (0.46)
Change in Marital Status		-0.02 (0.18)	-0.02 (0.24)	-0.02 (0.21)	-0.01 (0.19)	-0.01 (0.22)	-0.01 (0.19)	-0.02 (0.20)
SELF-RATED HEALTH (lag)	2.55 (1.18)	2.55 (1.18)	2.72 (1.20)	2.69 (1.19)	2.83 (1.15)	2.79 (1.16)	2.81 (1.14)	2.86 (1.14)
Change in SRH		0.17 (1.01)	-0.03 (1.09)	0.15 (1.15)	-0.04 (1.13)	0.03 (1.16)	0.04 (1.17)	0.02 (1.17)
INDEX DISEASES (lag)	1.15 (1.11)	1.16 (1.12)	2.44 (1.47)	2.73 (1.62)	3.02 (1.77)	3.31 (1.88)	3.57 (1.94)	3.81 (1.95)
Change in Index Diseases		1.27 (1.27)	0.30 (0.89)	0.29 (0.96)	0.28 (0.94)	0.28 (0.90)	0.23 (0.79)	0.21 (0.76)
NAGI INDEX (lag)	1.44 (1.66)	1.42 (1.64)	1.19 (1.54)	1.26 (1.58)	1.28 (1.57)	1.32 (1.57)	1.43 (1.59)	1.46 (1.57)
Change in NAGI Index		-0.23 (1.32)	0.08 (1.24)	0.06 (1.21)	0.06 (1.23)	0.15 (1.25)	0.08 (1.25)	0.13 (1.25)
CES-D SCORE (lag)	3.77 (1.57)	3.75 (1.56)	3.22 (1.61)	3.49 (1.28)	3.61 (1.32)	3.59 (1.34)	3.57 (1.32)	3.51 (1.30)
Change in CES-D Score		-1.72 (1.76)	0.28 (1.59)	0.14 (1.46)	-0.01 (1.46)	0.02 (1.46)	-0.04 (1.43)	0.11 (1.40)

N (valid) = 82,512 observations; CES-D --- Center for Epidemiological Studies – Depression Scale.

[a] Time-varying covariates are those associated with repeated measures within individuals (1992-2006 HRS cohort data).

[b] Case weights for each wave are respondent-level weights obtained from the Cross-Tracker 2006 HRS file.

[c] All "*change*" variables represent the difference between current (t_i) and previous wave (t_{i-1}).

Table 6: Descriptive Statistics for Time-Constant Covariates

Covariates	Mean / % (SD)
Age (1992)	55.83 (3.17)
Female	52.3%
Education[c]	12.34 (3.05)
Non-Hispanic Black	10.3%
Hispanic	6.5%
Mortality[d]	18.7%
Attrition[d]	7.1%

N(valid) = 10,314 respondents.
[a]Time-constant covariates are those associated with each individual at baseline (1992).
[b]Case weights are respondent-level 1992 weights (2006 HRS Cross-Tracker File).
[c]Education is measured as "number of school-years completed".
[d] Mortality and attrition recorded between baseline (1992) and 2006.

Table 7: Intra-personal and Inter-personal Estimates of BMI Growth Curve Coefficients: Hierarchical Linear Modeling Results, 1992-2006

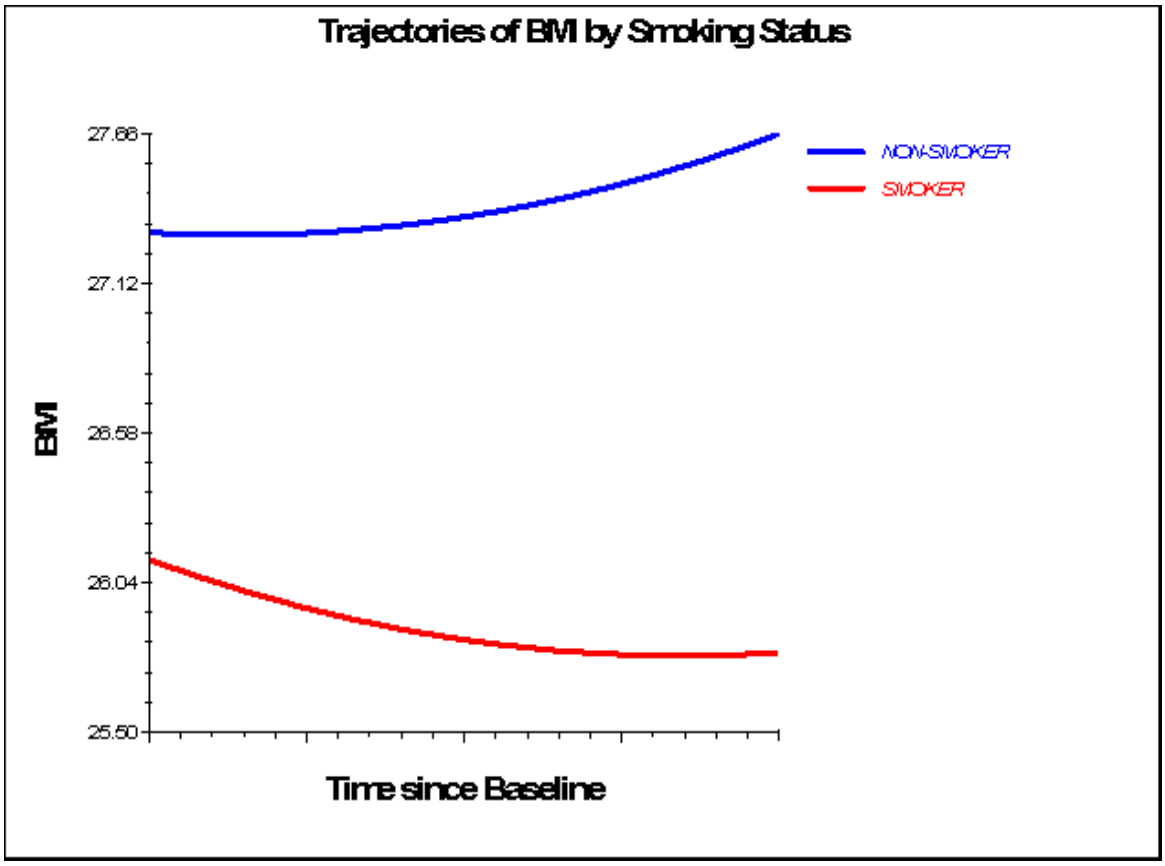
Measures	**M1**	**M2**	**M3**	**M4**
Fixed Effects				
Time-varying variables				
Smoker (lag)	-1.263***	-1.324***	-1.383***	-1.054***
Δ Smoker	-1.048***	-1.068***	-1.085***	-0.959***
Smoker*Time(T)		-0.047***	-0.051***	-0.097***
Δ Smoker*T		-0.038**	-0.040**	-0.060**
Drinker (lag)	-0.329**	-0.323**	-0.203*	-0.083
Δ Drinker	-0.208***	-0.192***	-0.126*	-0.074
Drink*T		0.016	0.012	-0.011
Δ Drinker*T		0.001	0.001	-0.011
Physical Activity (lag)	-0.549***	-0.561***	-0.437***	-0.417***
Δ Physical Activity	-0.381***	-0.389***	-0.314***	-0.315**
Physical Activity*T		-0.027	-0.028	-0.049*
Δ Physical Activity*T		-0.025	-0.024	-0.032
Proxy (lag)	-0.694***	-0.692***	-0.745***	-0.537***
Δ Proxy	-0.629***	-0.630***	-0.682***	-0.587***
Assets (lag)			-0.080	-0.043
Δ Assets			0.001	0.017
Income (lag)			-0.083*	-0.059
Δ Income			-0.073**	-0.059*
Marital Status (lag)			0.314***	0.170***
Δ Marital Status			0.250***	0.177**
Δ Self-Rated Health			-0.003	0.001
Index Disease (lag)			0.003	0.004
Δ Index Disease			-0.001	-0.002
NAGI (lag)			0.258***	0.135***
Δ NAGI			0.209***	0.156***
CES-D (lag)			-0.021	-0.029*
Δ CES-D			-0.014	-0.017
Intercept				
Intercept	27.569***	27.572***	27.614***	27.698***
Mortality	-0.323*	-0.318*	-0.612***	-0.611***
Attrition	-0.632***	-0.633**	-0.635**	-0.170
Black			1.290***	0.080

Hispanic			0.106	-0.275*
Female			-0.385***	-0.163**
Age (1992)			-0.089***	-0.063***
Education			-0.085***	-0.019
BMI (1992)				0.685***
Linear Slope				
Intercept	0.048***	0.046***	0.050***	0.051***
Mortality	-0.076***	-0.070***	-0.049**	-0.052***
Attrition	0.008	0.011	0.014	-0.006
Black			-0.060***	-0.012
Hispanic			-0.047**	-0.033*
Female			0.011	0.001
Age (1992)			-0.006***	-0.008***
Education			-0.001	-0.003*
BMI (1992)				-0.026***
Quadratic Slope[a]				
Intercept	0.002**	0.002**	-0.001	-0.001
Random Effect (Variance)				
Intercept1	19.105***	19.080***	18.099***	6.805***
Linear	0.070***	0.070***	0.068***	0.059***
Quadratic	0.001***	0.001***	0.001***	0.001***
Level1 R	7.477	7.475	7.451	7.023
AIC	444,322.4	444,201.3	443,555.6	433,092.7

*p <0.05; **P <0.01; ***p </= 0.001.

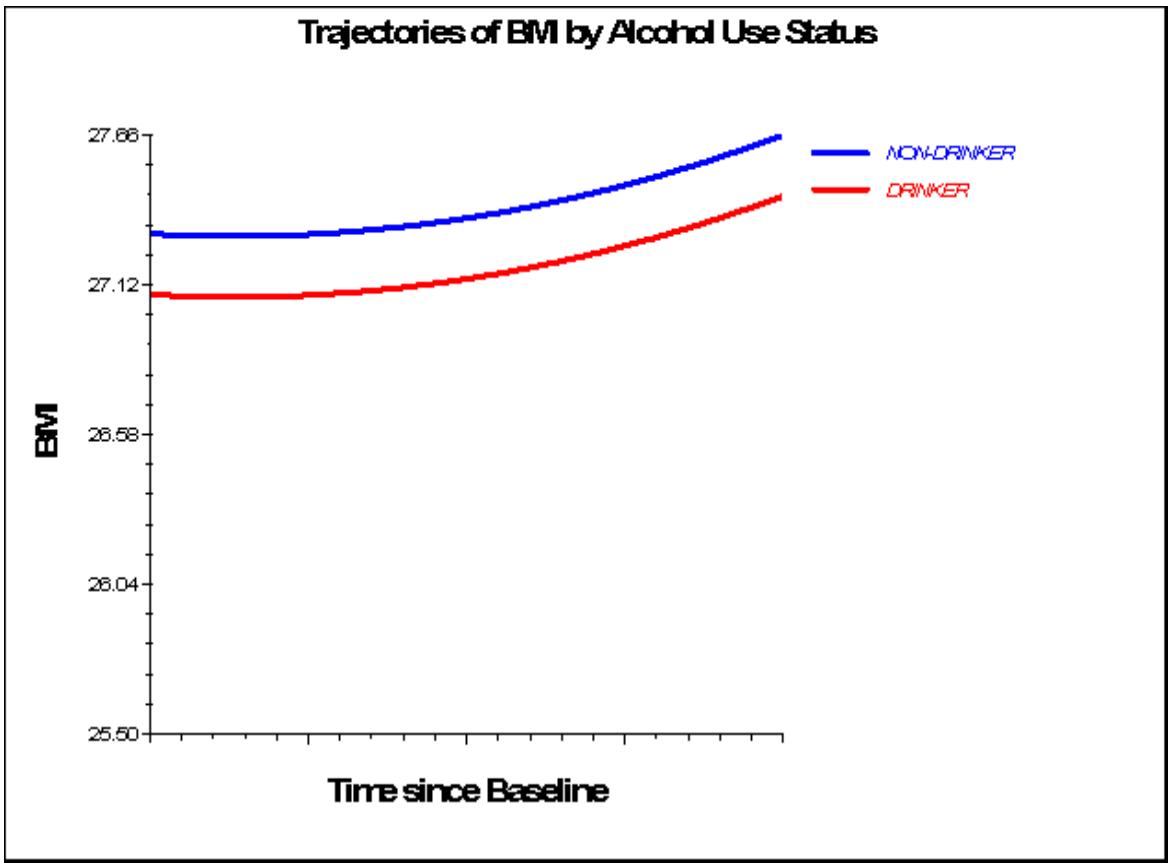
[a] Regression coefficients not shown; coefficients associated with all covariates are non-significant in models 1 through 4.

Figure 4: Model-based BMI Trajectories by Smoking Status, 1992-2006



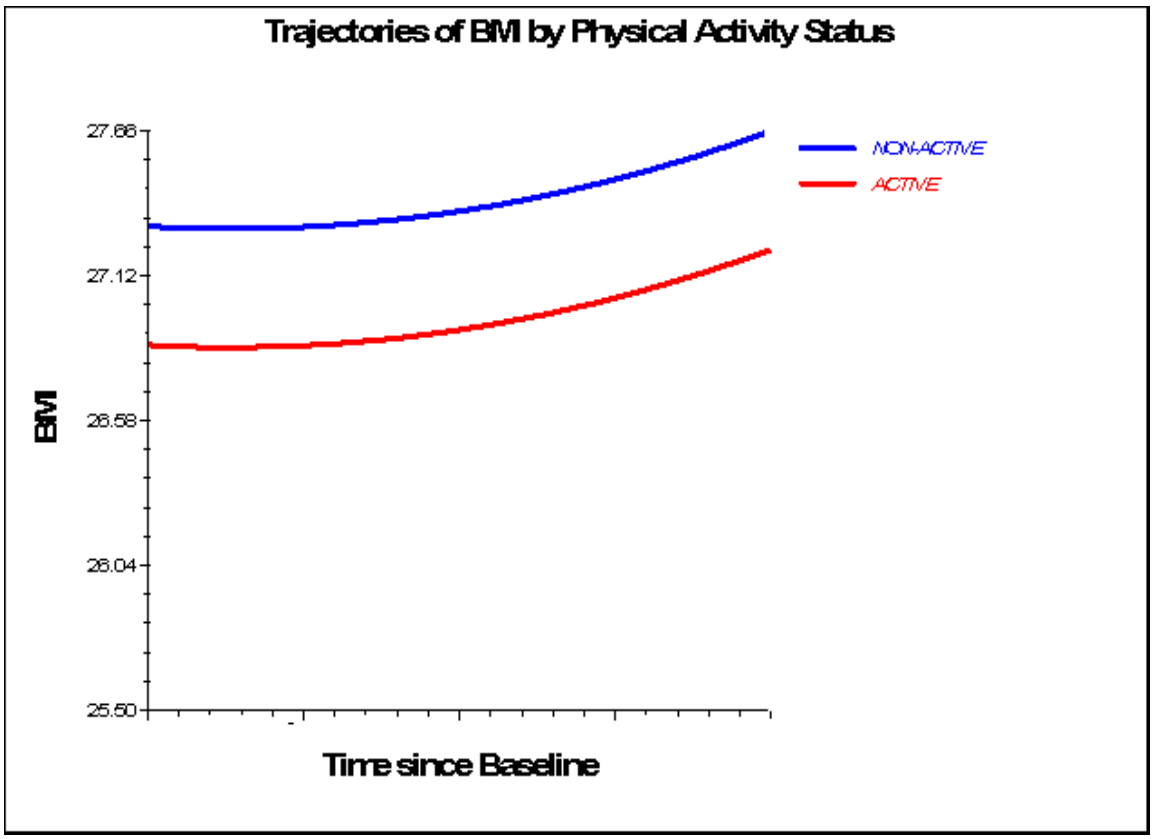
Graphs created based on M_3 with statistically-significant time-interactions (Table 4).

Figure 5: Model-based BMI Trajectories by Alcohol-Use Status, 1992-2006



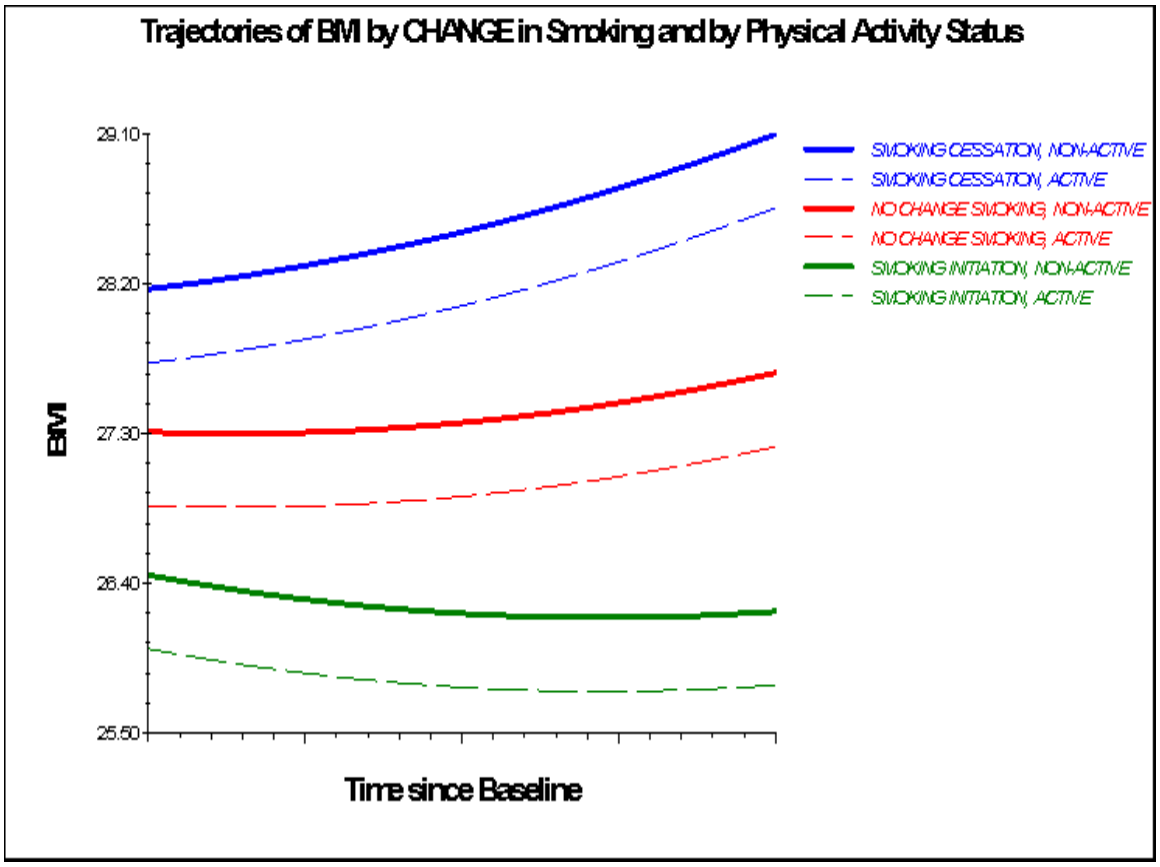
Graphs created based on M_3 with statistically-significant time-interactions (Table 4).

Figure 6: Model-based BMI Trajectories by Physical Activity Status, 1992-2006



Graphs created based on M_3 with statistically-significant time-interactions (Table 4).

Figure 7: Model-based BMI Trajectories by Change in Smoking and by Physical Activity Status, 1992-2006



Graphs created based on M_3 with statistically-significant time-interactions (Table 4).

Appendix 2: Chapter III

Table 8: Descriptive statistics for Total Household Assets and Total Household Income (weighted)

Descriptives		**Total Assets**	**Total Income**
Mean		325,877.84	52,698.75
Median		126,000.00	35,776.00
Std. Deviation		1,123,743.80	89,706.00
Skewness		32.56	25.21
Kurtosis		1,758.41	1,346.71
Percentiles	**25**	38,500.00	18,660.00
	50	126,000.00	35,776.00
	75	319,000.00	61,430.00

N (valid) = 82,512 observations; case weights are respondent-level weights obtained from the Cross-Tracker 2006 HRS file.

Table 9: Descriptive Statistics for Time-Constant Covariates (Level 2) - (weighted & unweighted)

	Unweighted		**Weighted[b]**	
Covariates[a]	Mean / %	SD	Mean / %	SD
Age (1992)	55.80	3.15	55.83	3.17
Female	52.8%		52.3%	
Education[c]	11.95	3.31	12.34	3.05
Non-Hispanic Black	17.2%		10.3%	
Hispanic	10.7%		6.5%	
Mortality[d]	19.7%		18.7%	
Attrition[d]	7.5%		7.1%	
Valid N (respondents)	10,314		10,314	

[a]Level 2 measures are time-constant associated with the individual at baseline (1992).
[b]Case weights, where used, are respondent-level 1992 weights from the Cross-Tracker 2006 HRS file.
[c]Education is measured as "number of school-years completed".
[d] Mortality and attrition recorded between baseline (1992) and 2006.

Table 10: Coefficient Estimates for Successive Models with Alcohol-only and All-Behaviors Measures

	M_{3A}	M_{3B}
Fixed Effect		
Time-varying variables		
Alcohol (Lagged)	-0.269 **	-0.203 *
Δ Alcohol	-0.178 ***	-0.126 *
Smoking (Lagged)		-1.383 ***
Δ Smoking		-1.085 ***
Physical Activity (Lagged)		-0.437 ***
Δ Physical Activity		-0.314 ***
Time-constant variables		
Intercept		
Intercept	27.521 ***	27.517 ***
Linear Slope		
Intercept	0.065 ***	0.049 ***
Quadratic		
Intercept	-0.001	0.000
Random Effect		
Intercept	18.791 ***	8.099 ***
Linear	0.071 ***	0.068 ***
Quadratic	0.001 ***	0.001 ***
Level 1	7.510	7.451

Significance p-value level set at p < 0.01; *p <0.05; **P <0.01; ***p </= 0.001.

Table 11: Coefficients Estimates for Models with Interactions Between Socio-Economic Measures and Health Behaviors

	M_{3_1} Education	M_{3_2} Gender	M_{3_3} Age-at-Baseline	M_{3_4} Ethnicity
Fixed Effects				
Time-Varying Variables				
Smoker (lagged)				
Intercept	-1.309***	-1.278***	-1.326***	-1.313***
Interaction Term(1)	0.034	-0.086	-0.026	-0.198
Interaction Term(2) [a]				0.241
Δ Smoker				
Intercept	-1.052***	-1.073***	-1.068***	-1.031***
Interaction Term(1)	0.033*	0.011	-0.008	-0.121
Interaction Term(2) [a]				-0.061
Alcohol (lagged)				
Intercept	-0.070	0.016	-0.073	-0.041
Interaction Term(1)	-0.001	-0.178*	0.008	-0.177
Interaction Term(2) [a]				0.031
Δ Alcohol				
Intercept	-0.048	-0.089	-0.052	0.014
Interaction Term(1)	0.019	0.079	0.009	-0.224
Interaction Term(2) [a]				-0.174
Physical Activity (lagged)				
Intercept	-0.426***	-0.375***	-0.427***	-0.442***

Interaction Term(1)	-0.005	-0.101	0.023	0.046
Interaction Term(2) [a]				0.065
Δ Physical Activity				
Intercept	-0.307***	-0.308*	-0.307***	-0.314*
Interaction Term(1)	-0.004	0.001	0.009	0.061
Interaction Term(2) [a]				-0.031
Time-Constant Variables				
Intercept				
Intercept	27.601***	27.584***	27.590***	27.594***
Linear Slope				
Intercept	0.053***	0.055***	0.053***	0.052***
Quadratic Slope				
Intercept	-0.000	-0.001	-0.001	-0.001
Random Effects				
Intercept	18.129***	18.111***	18.122***	18.122***
Linear Slope	0.068***	0.068***	0.068***	0.068***
Quadratic Slope	0.001***	0.001***	0.001***	0.001***
Level-1	7.452	7.454	7.452	7.449

[a] For *Race/Ethnicity* two interaction terms corresponding to "Black" [i.e., Interaction Term (1)] and "Hispanic" [i.e., Interaction Term (2)] respectively have been included. Significant p < 0.01; *p <0.05; **p <0.01; ***p </= 0.001.

Table 12: Mean values and differences between self-reported and interviewer-measured height and weight values

		Self-reported measure	Interviewer-obtained measure	
		Mean	Mean	Mean Difference
2004	WEIGHT (lb)	177.86	183.00	(5.14) ***
	HEIGHT (in.)	67.08	65.75	1.33 ***
2006	WEIGHT (lb)	177.90	180.52	(2.62) ***
	HEIGHT (in.)	66.99	66.90	0.09 ***

*** p-value < 0.05.

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CHAPTER IV: HETEROGENEITY IN LONG-TERM TRAJECTORIES OF BODY-MASS INDEX FROM MIDDLE TO OLDER AGE: RACIAL/ETHNIC, GENDER AND EDUCATIONAL PREDICTORS OF TRAJECTORY MEMBERSHIP

IV.1. BACKGROUND

Overweight and obesity are increasingly prevalent among middle-age and older adults (Flegal, Carroll, Ogden, & Curtin, 2010b). Excess weight in older ages is associated with unfavorable physical and cognitive aging outcomes (Field et al., 2001; Profenno, Porsteinsson, & Faraone, 2010) and poses an escalating economic burden for individuals and society (Finkelstein, Trogdon, Cohen, & Dietz, 2009; Lakdawalla, Goldman, & Shang, 2005).

Current knowledge on the progression of body-weight starting in middle-age rests on an assumption of population homogeneity with respect to changes in body-weight over time. In other words, it assumes that the population follows an "average" pattern of change, and that the observed deviation of individuals from the "average" can be explained in terms of distinctive characteristics, such as gender, race, age, or health status indicators (i.e., *variable-based approach*) (Botoseneanu & Liang, 2011; Kahng, Dunkle, & Jackson, 2004). It is conceivable though that identifiable heterogeneity in body-weight development exists within the larger population, reflecting qualitatively distinctive groups which vary in their patterns of growth. This alternative approach, i.e., *person-centered approach,* allows for *multiple distinctive trajectories* within a given population and defines the probability of individuals to belong to any such trajectory as a function of time-constant and/or time-varying individual characteristics (Nagin & Tremblay, 2005; Nagin, 2005).

Understanding heterogeneity in body-weight trajectories starting in middle-age may be useful in several respects. First, distinct clinical significance may be assigned to each trajectory, in accordance with the associated risk for specific physical or cognitive clinical outcomes. Second, it may assist in identifying social, behavioral, or economic individual characteristics associated with a higher propensity of experiencing "risky" trajectories. Third, the ability to identify body-weight trajectories at higher risk for health or mental decline may result in more efficient public health efforts aimed at reducing the long-term effects of obesity, by selectively focusing on those sub-groups most likely to benefit from such interventions.

This study has two specific aims. First, using group-based semi-parametric mixture models (Jones & Nagin, 2007; Nagin, 2005), we identify multiple distinct trajectories of body-mass index (BMI) over a 14-year period (1992-2006) in a nationally-representative sample of Americans age 51 to 61 years old at baseline. Second, we explore racial/ethnic and gender differences in trajectory-group membership probabilities.

IV.2. METHODS

Study Population

Data came from the Health and Retirement Study (HRS documentation at: http://hrsonline.isr.umich.edu/). Consistent with the purpose of the study and to minimize the potential for cohort effects on BMI (Reynolds & Himes, 2007), respondents from a single cohort (1931-1941 birth-years) were analyzed. Data were gathered every 2 years from 1992 to 2006, for up to 8 repeated observations. Of the 13,565 individuals in the HRS cohort, we excluded 3,116 (22.9%) cohort-ineligible spouses (i.e., born before 1931 or after 1941) and 135 (0.9%) participants who did not respond to the health survey sections. The final analytic sample consisted of 10,314 individuals (6.4 average number of interviews completed). Response rates ranged from 81.7% (1992) to 89.2% (1994); 55.7% of respondents

completed all 8 interviews. As of 2006, the cumulative mortality rate, validated through the National Death Index (NDI), was 18.7%. Proxy interviews were conducted when a respondent was unable to participate due to physical or cognitive limitations (range from 4.8% in 1992 to 9.0% in 2002).

Assessment of Weight, Height and BMI

Self-reported weight was recorded at each wave; height was recorded at the first interview and verified in the second wave. Body Mass Index (BMI) was calculated at each wave as: BMI = [Weight (lb)/Height (inches) ^{2}] x 703, using current weight and initial height.

Statistical Analysis

We conducted analyses in two stages. First, BMI trajectories were determined by fitting semi-parametric mixture models (SPMM) to the data using the PROC TRAJ procedure in SAS version 9.2 (Jones, Nagin, & Roeder, 2001). SPMMs identify distinct groups of individual trajectories within a population and, by use of posterior probabilities, assign individuals to the group to which they have the highest probability of belonging (probability of 0.9 or higher was considered excellent fit, a value of 0.7 or lower was considered poor fit) (Nagin, 2005). Maximum likelihood estimation was used to estimate the group trajectory parameters. We assumed a censored normal distribution to account for potential floor and ceiling effects in BMI and employed a time-based analysis approach (i.e., BMI growth curves plotted as a function of time) (Alwin, Hofer, & McCammon, 2006), with control for inter-personal age-at-baseline differences (Liang et al., 2008). Linear, quadratic and cubic time-functions for successive models with between two and ten trajectories were tested. Time was centered at its mean to minimize the possibility of multicollinearity when evaluating non-linear time functions. The best fitting model (i.e., the number of distinct trajectories) was specified using Bayesian Information Criterion (BIC) scores and examination of 95% CIs for each trajectory (Nagin, 2005).

In the second stage, multinomial logit models were used to examine predictors of trajectory membership. We derived the log-odds of the impact of each predictor on the likelihood of membership in each latent group or trajectory relative to a designated comparison group. Multiple sequential models were analyzed, in accordance with the classic recommendations for mediation testing (Baron & Kenny, 1986). Specifications similar to multinomial logistic regression analysis were used to capture the effects of time-constant covariates, such as race/ethnicity, gender, education, age-at-baseline) on the probability of membership in each group or trajectory (Nagin, 2005). The PROC TRAJ procedure in SAS estimated the equation for stage 2 simultaneously with equation for stage 1 (Jones et al., 2001; Jones & Nagin, 2007).

We chose not to weight the data because many of the variables used in the calculation of differential selection weights (e.g., race/ethnic group, gender, marital status) are explicitly included in the models, making unweighted ordinary least squares estimates less biased and preferable over weighted estimates (Winship & Radbill, 1994). Results from unweighted analyses are shown henceforth, except in Tables 1 and 2, where in accordance with the consensus on presentation of descriptive information we show weighted sample characteristics (Gelman, 2007).

To minimize the loss of participants due to item missing (Little & Rubin, 2002; Schafer & Graham, 2002), 3 complete data sets were imputed using the NORM software (Schafer & Olsen, 1998). We ran PROC TRAJ analyses using each of the 3 data sets. Parameter estimates and their standard errors were calculated by averaging across the 3 data sets and adjusting for their variance.

Finally, because of the huge HRS sample, a two-sided p-value of less than 0.01 was considered to indicate statistical significance.

Trajectory Predictors and Potential Confounders

Race/ethnicity (i.e., mutually-exclusive group designation - non-Hispanic White, non-Hispanic Black, and Hispanic) and gender (1 = female, 0 = male) were examined as predictors of BMI trajectory membership.

Socio-demographic characteristics related to BMI and/or potential predictors were included as control variables: education (years of education completed) (Ball & Crawford, 2005) and age (Clarke, O'Malley, Johnston, & Schulenberg, 2009) were measured at baseline and included as time-constant covariates; total household income (quartiles) (Chang & Lauderdale, 2005), total household assets (quartiles) (Fonda, Fultz, Jenkins, Wheeler, & Wray, 2004), and marital status (1 = married/living with a partner, 0 = single/ divorced/widowed/separated) (J. Sobal, Rauschenbach, & Frongillo, 2003) were also included.

To account for potential "healthy survivor bias" in BMI (Mehta & Chang, 2009), time-varying measures of physical and mental health were also included: *index of chronic diseases* (count of seven chronic conditions – heart disease, stroke, high-blood pressure, diabetes, arthritis, chronic lung disease, and cancer; range = 0 - 7), *self-rated health* (single-item rating; range = 1 (excellent) -5 (poor)), *Nagi index of functional limitations* (count of six items representing reported difficulties with common activities; range = 0 - 6) (Nagi, 1979), and *CES-D (depression) score* (count of nine items from the Center for Epidemiological Studies Depression Scale; range = 0 - 9) (Radloff, 1977).

Each of the socio-economic and health status time-varying covariates was represented by a *lagged measure* (i.e., observation from the previous wave) and a *change term* (i.e., difference between current and previous observation) to ensure clear time-precedence between these and the dependent variable.

Mortality and attrition in older populations are sources of non-random missing data (Little & Rubin, 2002) and need to be addressed as confounders (Harel, Hofer, Hoffman, & Pedersen, 2007; Mroczek & Spiro, 2005; Murphy et al., 2011). Similar to

pattern-mixture modeling (Hedeker & Gibbons, 2006), sub-groups were identified based on patterns of missing data and group membership indicators were included in the models (for mortality: 1 = died, 0 = alive at end of study; for attrition: 1 = dropped-out for reasons other than mortality and did not return, 0 = completed study). Time-varying proxy status was represented by a lagged measure (1 = proxy respondent, 0 = self) and a change term (i.e., difference between current and previous wave).

IV.3. RESULTS

Sample Description

Time-varying (Table 13) and time-constant (Table 14) characteristics of the study population are shown below.

[Tables 13, 14 about here]

Trajectories of BMI over Time

We estimated alternative order (i.e., intercept-only, linear, quadratic, and cubic) sequential SPMMs that allowed for two- to ten groups of BMI trajectories. In the unconditional (M_0) and mortality/attrition-controlled models (M_1), the cubic slope coefficients were non-significant. Hence, only quadratic time functions were further analyzed (Table 15). For both the unconditional and covariate-adjusted models, BIC revealed improved model fit from 2 to 5 trajectories, with a plateau in fit improvement evident at specifications greater than 5 trajectories (not shown; *see Supplementary Appendix*). The narrow, non-overlapping 95% confidence intervals (CI) (Figure 8) provided further support for the 5 trajectories model. Thus, the analyses presented hereafter are based on the 5 trajectories model.

[Table 15 about here]

[Figure 8 about here]

In the unconditional best-fitting model (M_0, Table 15), the intercepts for the 5 trajectories largely represent the major BMI categories: normal (b = 22.12, p<.001) (trajectory # 1; 19.9% of sample), overweight (b=26.06, p<.001) (trajectory # 2; 43.8% of sample), borderline-obese (b=29.87, p<.001) (trajectory # 3; 25.4% of sample), obese (b=34.90, p<.001) (trajectory # 4; 8.8% of sample) and morbidly-obese (b=43.44, p<.001) (trajectory #5; 2.1% of sample). In terms of rate-of-change in BMI, all 5 groups experienced significant increases in BMI over the period of observation. Trajectory # 1 *(Normal with accelerating increase in BMI)* is characterized by a normal intercept and positive linear (b= 0.069, p<.001) and quadratic (b= 0.011, p<.001) slopes; the intercept and slope coefficients for trajectory #1 are robust in subsequent models (Table 15). The quadratic slope coefficients for trajectories #2 *(Overweight with linear increase in BMI)*, #3 *(Borderline-obese with linear increase in BMI),* and #4 *(Obese with linear increase in BMI)* were or became non-significant in SES and health-adjusted models, suggesting that these groups follow a linear pattern of increase in BMI. In contrast, trajectory group #5 *(Morbidly-obese with slowing increase in BMI)* showed a decelerating increase in BMI over time; the positive linear and negative quadratic slopes remained robust after control for socioeconomic and health covariates (b= -0.033, p<.001 respectively in M_3, Table 15).

Table 16 shows the characteristics of individuals according to their propensity to follow each of the identified BMI trajectories.

[Table 16 about here]

Socio-Demographic Predictors of Trajectory Membership

Next, predictors of BMI trajectory groups were examined (trajectory # 1 as reference) in successive minimally-adjusted (i.e., adjustment for mortality, attrition and proxy-status) and fully-adjusted (i.e., adjustment for time-varying socio-economic and health status covariates, and baseline BMI) models. Coefficient estimates (not shown; *see Supplementary Appendix; positive coefficients indicate an*

increased likelihood that an individual possessing such characteristic will belong to the given trajectory as opposed to the reference trajectory; negative coefficients indicate the opposite) were used to derive relative risk ratios (RRR = e^b, where *b* is the logistic regression coefficient). Results (Table 17) indicated significant racial/ethnic and gender differences in the likelihood of trajectory membership.

[Table 17 about here]

Racial/Ethnic Differences in Trajectory Membership

In the minimally-adjusted model (M_2), as compared with Whites, Blacks and Hispanics had a significantly greater probability of belonging to the higher BMI trajectories relative to the normal BMI reference trajectory (Table 17). Specifically, Blacks had a two-fold increase in the risk of following overweight trajectories (RRR=1.86 for trajectory #2 and RRR=2.65 for trajectory # 3) and a three-fold increase in the risk of following obese BMI trajectories (RRR=3.04 for trajectory #4 and RRR=3.23 for trajectory # 5). On the other hand, differences in risk between Hispanics and Whites were greater for overweight trajectories (RRR=2.19 for trajectory #2 and RRR=2.30 for trajectory # 3) than for obese BMI trajectories (RRR=1.52 for trajectory #4 and non-significant for trajectory # 5). Further, differences between Whites and Blacks in the relative risk of trajectory membership disappeared after control for socio-economic differences and baseline BMI (fully-adjusted models, M_{2_1}, M_{2_2}), while those between Hispanics and Whites increased, mainly in the overweight and borderline-obese trajectories (M_{2_2}).

Gender Differences in Trajectory Membership

In all models, females showed a lower likelihood of following the higher BMI trajectories (Table 17) compared with males. Females had approximately half the risk of belonging to overweight and obese BMI trajectories (RRR=0.43, RRR=0.45, RRR=0.68 respectively, $p<.001$, for trajectories #2, #3 and #4 in M_2; RRR=0.64 for trajectory #4 and RRR=0.51 for trajectory # 5 respectively, $p<.01$ in M_{2_2}). Baseline BMI explained the difference in group membership risk between females and males

in overweight trajectories (trajectories #2, #3), but not in obese trajectory groups (trajectories #4, #5).

Effects of Other Covariates

Older respondents had a slightly lower risk of membership in the higher BMI trajectory groups as compared with the reference normal-weight trajectory (Table 17). In SES and health-controlled models (M_2, M_3), higher education was associated with a slightly lower risk of experiencing the higher BMI trajectories; this inverse association was fully explained by baseline BMI (M_{2_2}), with the exception of morbidly-obese trajectory (Trajectory # 5) risk, which increased after control for baseline BMI. Mortality, attrition and proxy status were included as confounders to account for possible selection bias. Respondents who died during the study period had substantially lower probabilities of experiencing the obese or morbidly-obese trajectories (trajectories #4 and #5, M_2, M_{2_1} and M_{2_2}).

Interestingly, when baseline BMI is accounted for, the probability of membership in the over-weight trajectory group (trajectory # 2, M_{2_2}) was increased more than two-folds for respondents who died, compared with those alive at the end of the study. Attrition was consistently associated with a higher likelihood of membership in the overweight trajectory group (trajectory #2) and substantially lower risk of following the obese or morbidly-obese trajectories (trajectories #4 and #5) (Table 17). The significant associations between mortality/attrition and the probability of group membership indicate that the coefficients for trajectories estimates would be incorrect if these sources of selection bias are not explicitly addressed in the models.

IV.4. DISCUSSION

In this study, we identified clusters of BMI growth trajectories in middle-aged and older adults. A major finding of our work is the underlying heterogeneity in the patterns of BMI change and the considerable racial/ethnic and gender differences in the propensity to experience each of the identified trajectories. The group-based approach used here differs from more conventional latent growth curve modeling in

that it assumes a number of distinct trajectories, each with a distinct intercept, time-slope and estimated population prevalence (Jones & Nagin, 2007). Because we suspected that individuals follow discrete BMI trajectories, rather than varying continuously on a latent trait, the semi-parametric mixed-modeling method is the most appropriate analytical procedure.

This approach identified five sub-groups and provided average patterns of change over 14 years for each sub-group. We offer two key observations from the identified trajectory groups: (1) only about 20 percent of this sample observes a "normal" BMI trajectory (i.e., normal BMI intercept and weight gain within the normal BMI range), with the rest divided between over-weight (43%) and obese or morbidly-obese (37%); and (2) all groups gain weight over the period of observation, with only the morbidly-obese sub-group experiencing a slight deceleration in weight gain over time. We believe these are troubling findings for two reasons. First, the 20% prevalence of normal BMI trajectory is even lower than recent cross-sectional estimates, which put the prevalence of normal BMI among middle-age and older adults at 22% for men and 32% for women (Flegal, Carroll, Ogden, & Curtin, 2010a). Second, for some of the groups, the rates of increase justify an upward change in categorization over the study period (from borderline-obese to overtly obese-category 1 for trajectory # 3 and from obese-category 1 to obese-category 2 for trajectory #4) and signify a substantial increase in obesity-associated disease risks. (NIH, 2010). While we expected a more pronounced dissimilarity between the trajectory sub-groups (e.g., raising vs. falling vs. stable BMI), the results are in line with observations from other population-average longitudinal studies (Barone et al., 2006; Dugravot et al., 2010; Jacobsen et al., 2001), which suggested that BMI increases into older age past the point previously believed to represent the age of peak body-weight (i.e., around age 65).

Racial/ethnic differences in weight status and weight change have been documented, primarily in young adults (Clarke et al., 2009; Mujahid, Diez Roux, Borrell, & Nieto, 2005; Sanchez-Vaznaugh, Kawachi, Subramanian, Sanchez, &

Acevedo-Garcia, 2009). The course of BMI also differed considerably between the racial/ethnic groups of older adults considered in our study. Notably, the relative distribution of trajectory membership risk by race/ethnicity was sensitive to variations in baseline BMI, but not to variations in socio-economic or health status indicators. Regardless of SES and health status, Blacks were more likely (compared with Whites) to follow each of the higher BMI trajectories, yet such differences were entirely explained by the BMI with which they enter middle-age. Conversely, trajectory risk differences between Hispanics and Whites increased substantially after accounting for baseline BMI. These findings suggest that differences in BMI trajectories between Blacks and Whites are established in early life, while those between Hispanics and Whites persist and even increase from middle to old age. It is quite possible that factors other than the conventional measures of SES (income, assets or education), such as early-life behavioral (Akresh, 2007), cultural (Abraido-Lanza, Chao, & Florez, 2005), and birthplace/immigration status (Sanchez-Vaznaugh et al., 2009) differences between the groups, partially explain the timing of weight disadvantage initiation. This is a consequential finding, suggesting that the critical periods for interventions to reduce racial/ethnic inequalities in weight status are prior to entering middle-age among Blacks, and continue into middle and older age among Hispanics.

The initial association between gender and trajectory risk - women showing a substantially lower propensity to follow the high BMI trajectories - was also partially explained by the baseline BMI, but not by educational or health status differences. It is somewhat difficult to place our results in the context of previous literature, mainly because studies on gender differences in BMI trajectories are scarce and the results from other studies (cross-sectional or 2-point transitions) are mixed. While some studies point to a higher prevalence of overweight in men (He & Baker, 2004; Novak, Ahlgren, & Hammarström, 2005; Wang & Beydoun, 2007) and a higher prevalence of obesity and higher variability in body-weight in women (Jenkins, Fultz, Fonda, & Wray, 2003; Williamson, 1993), others find no such differences (Ogden et al., 2006; J. Sobal & Rauschenbach, 2003). A recent

examination of the average long-term BMI trajectory in a sample of middle-age adults found no significant gender differences in either BMI intercept or rate-of-change (Botoseneanu & Liang, 2011). This, coupled with the present finding of differences favorable to women in the obese and morbidly-obese BMI trajectories, but not in the overweight trajectories, suggests that "averaging out " of significant effects occurs under the classic latent growth models. When such models are employed and distinct trajectories are collapsed into an "average" trajectory, substantial differences on particular trajectories not observed on others may be lost. In contrast, group-based modeling allows not only for discrete trajectories, but also for discrete predictors of each trajectory. Future studies linking gender heterogeneity in body-weight evolution to specific health outcomes are needed and may partially explain the observed gender-related disparities in morbidity and mortality in old age (Oksuzyan et al., 2008).

It is paramount to recognize that the results derived from group-based modeling are approximations of population differences in BMI trajectories, which are based on sub-group means over a period of time (Nagin, 2005). Thus, the results should not be construed to mean that individuals actually "belong" to a trajectory group, but rather that individual trajectories can be clustered into a finite number of sub-groups, which is not immutable, but arrived at through assessments of model-fitting indices, to approximate a continuous distribution within a population (Nagin & Tremblay, 2005). Further, while this technique allows for the identification of population risk-factors, one should refrain from predicting any particular individual's trajectory membership group based on ex-ante individual characteristics. This reflects the fact that even if a set of characteristics increase the probability of individuals following a particular trajectory, not all individuals with those characteristics will follow that trajectory (Nagin & Tremblay, 2005).

Some limitations of this study should be acknowledged. First, self-reported BMI was used for trajectory calculation. Individuals are known to over-report height and under-report weight (Nawaz, Chan, Abdulrahman, Larson, & Katz, 2001). As such,

self-reported BMI tends to reflect conservative estimates of the "true" BMI. We performed a comparison of self-reported and interviewer-measured height and weight (available in HRS only for 2004 and 2006) and, consistent with others studies (Fillenbaum et al., 2010; Weir, 2008), found only small differences (not shown; *see Table 3A in Supplementary Appendix*). Second, BMI may not be the optimal body-weight indicator in older age (Seidell & Visscher, 2000). Other body-composition measures (e.g., waist circumference or fat-free mass) offer more accurate assessments of disease risk (Janssen, Katzmarzyk, & Ross, 2004) and are not affected by the potential age-related loss of height. This limitation is partially mitigated by the observation that the age range of a majority of our respondents was below the age of accelerated height loss (i.e., starting in eighth decade of life) (Dey, Rothenberg, Sundh, Bosaeus, & Steen, 1999; Sorkin, Muller, & Andres, 1999). However, some respondents were followed up to age 75, so we cannot entirely rule-out the possibility of upward bias in BMI in later waves. We were unable to perform similar analyses of other body-composition indicators and to assess the potential for artifactual BMI changes due to loss of height because HRS does not collect such data. Similar studies on heterogeneity in trajectories of other body-composition indicators are needed, as they may prove more accurate predictors of associated disease risk in old age.

One of the main goals of identifying heterogeneous BMI trajectories within a given population is to assess whether such trajectories carry differential morbidity and mortality potential. While this kind of analysis is outside the scope of our present study, we can use the results to draw some inferences and suggestions for future research. For example, the female advantage in mortality in older age is well documented. Overweight and obesity have been associated with increased mortality in the same age group (Berrington de Gonzalez et al., 2010). In this context, our results showing that women have a substantially lower risk of following the obese BMI trajectories compared with men imply that the gender gap in mortality will potentially increase in the future. This may hold true for other health outcomes. Over time and barring effective population-level weight-preserving interventions,

detrimental health consequences associated with obesity will disproportionately accrue in those groups (i.e., racial minorities and men) more likely to follow the high-BMI trajectories and will result in an increase in obesity-related racial and gender health disparities. Additional research linking relevant health outcomes to various BMI trajectories is warranted; we suggest that priority be given to those conditions which carry a heavy morbidity burden for individuals and society, and for which racial/ethnic or gender disparities have been well documented, such as diabetes (Duru et al., 2009; Saydah, Cowie, Eberhardt, De Rekeneire, & Narayan, 2007), stroke (Glymour, Avendaño, Haas, & Berkman, 2008; Kleindorfer, 2009), or dementia (Glymour & Manly, 2008; Husaini et al., 2003).

This study identifies five distinct BMI trajectories in a middle-age and older population, and substantial racial/ethnic and gender differences in the propensity of following each trajectory. Yet this is only an initial step towards a better understanding of the risk factors associated with variability in body-weight course starting in middle-age. Awareness of discrete BMI trajectories may allow clinicians and policy professionals to tailor programs to specific groups who are at risk of poor aging outcomes due to obesity and to intervene at an early stage to alter the path of risky trajectories.

Table 13: Descriptive Statistics for Time-varying Covariates

	1992	1994	1996	1998	2000	2002	2004	2006
Body-Mass Index (BMI)	26.98±4.96	27.11±4.95	27.23±5.05	27.30±5.05	27.47±5.15	27.59±5.22	27.63±5.32	27.97±5.43
Attrition Status Indicators								
Proxy Status (lag)	0.05±0.21	0.04±0.19	0.07±0.25	0.07±0.26	0.08±0.27	0.09±0.29	0.09±0.28	0.07±0.26
Change in Proxy Status		0.02±0.18	-0.01±0.24	0.01±0.22	0.01±0.22	0.01±0.24	-0.01±0.21	-0.02±0.02
Socio-Economic Status Indicators								
Assets (lag)	2.46±1.21	2.48±1.19	2.51±1.19	2.55±1.20	2.58±1.21	2.61±1.21	2.63±1.20	2.66±1.18
Change in Assets		0.03 ±0.88	0.01±0.83	0.02±0.80	0.02±0.78	0.01±0.77	0.01±0.75	0.01±0.73
Income (lag)	2.57±1.11	2.60±1.11	2.61±1.11	2.58±1.11	2.60±1.10	2.61±1.09	2.63±1.09	2.64±1.10
Change in Income		0.02±0.88	-0.02±0.86	0.01±0.84	0.01±0.83	0.01±0.82	-0.01±0.83	0.01±0.81
Marital Status (lag)	0.77±0.42	0.78±0.42	0.75±0.43	0.74±0.44	0.72±0.45	0.71±0.45	0.71±0.46	0.69±0.46
Change in Marital Status		-0.02±0.18	-0.02±0.24	-0.02±0.21	-0.01±0.19	-0.01±0.22	-0.01±0.19	-0.02±0.20

	1992	1994	1996	1998	2000	2002	2004	2006
Health Status Indicators								
Self-Rated Health (lag)	2.55±1.18	2.55±1.18	2.72±1.20	2.69±1.19	2.83±1.15	2.79±1.16	2.81±1.14	2.86±1.14
Change SRH		0.17±1.01	-0.03±1.09	0.15±1.15	-0.04±1.13	0.03±1.16	0.04±1.17	0.02±1.17
Index Diseases (lag)	1.15±1.11	1.16±1.12	2.44±1.47	2.73±1.62	3.02±1.77	3.31±1.88	3.57±1.94	3.80±1.95
Change Index Diseases		1.27±1.27	0.3±0.89	0.29±0.96	0.28±0.94	0.28±0.90	0.23±0.79	0.21±0.76
Nagi Index (lag)	1.44±1.66	1.42±1.64	1.19±1.54	1.26±1.58	1.28±1.57	1.32±1.57	1.43±1.59	1.46±1.57
Change Nagi Index		-0.23±1.32	0.08±1.24	0.06±1.21	0.06±1.23	0.15±1.25	0.08±1.25	0.13±1.25
CES-D[3] Score (lag)	3.77±1.57	3.75±1.56	3.22±1.61	3.49±1.28	3.61±1.32	3.59±1.34	3.57±1.32	3.51±1.30
Change CES-D Score		-1.72±1.76	0.28±1.59	0.14±1.46	-0.01±1.46	0.02±1.46	-0.04±1.43	0.11±1.41

N(valid) = 82,512 observations
[1]Case weights for each wave are respondent-level weights obtained from the Cross-Tracker 2006 HRS file.
[2]All "*change*" variables represent the difference between current (t_i) and previous wave (t_{i-1}).
[3] CES-D = Center for Epidemiological Studies – Depression Scale.

Table 14: Descriptive Statistics for Time-Constant Covariates

Covariates	Mean ± SD/%
Age (1992)	55.83 ± 3.17
Female	52.3%
Education[3]	12.34 ± 3.05
Non-Hispanic Black	10.3%
Hispanic	6.5%
Mortality[4]	18.7%
Attrition[4]	7.1%

N(valid) = 10,314 respondents
[1]Time-constant covariates are those associated with the individual at baseline (1992).
[2]Case weights are respondent-level 1992 weights (2006 HRS Cross-Tracker File).
[3]Education is measured as "number of school-years completed".
[4] Mortality and attrition recorded between baseline (1992) and 2006.

Figure 8: BMI Trajectories for Five Groups Model with 95% Confidence Intervals

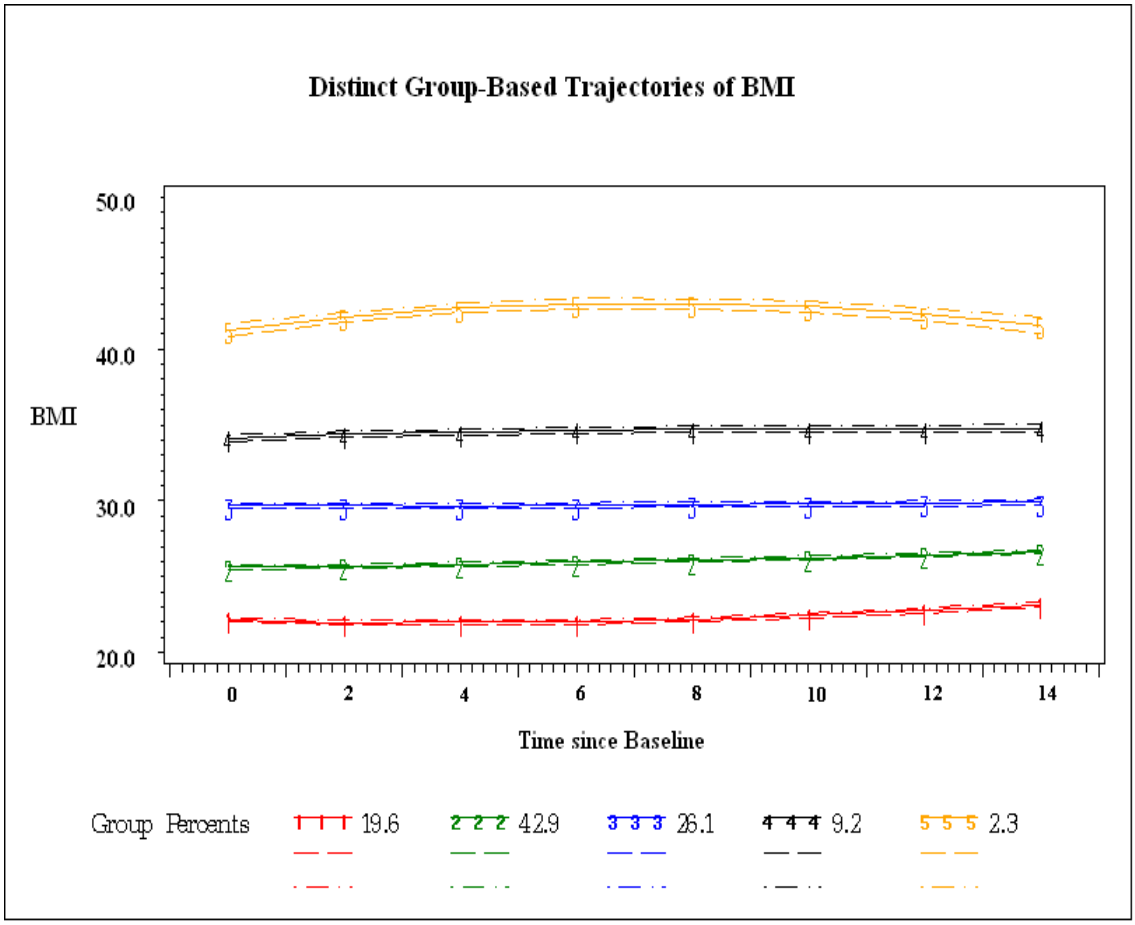

Table 15: Estimates of Growth Curves Parameters for Distinct Trajectories of BMI - 1992-2006

Trajectory		M_0	M_1	M_2	M_3
Trajectory 1 **Normal, Increasing (accelerating)**	Intercept	22.122***	22.015***	21.602***	21.505***
	Linear Slope	0.069***	0.064***	0.073***	0.073***
	Quadratic Slope	0.011***	0.011***	0.010***	0.012***
Trajectory 2 **Overweight, Increasing (linear)**	Intercept	26.067***	26.081***	26.208***	25.679***
	Linear Slope	0.070***	0.072***	0.073***	0.073***
	Quadratic Slope	0.003**	0.002	0.002	0.001
Trajectory 3 **Borderline Obese, Increasing (linear)**	Intercept	29.869***	29.974***	30.432***	29.484***
	Linear Slope	0.016*	0.028***	0.039***	0.033***
	Quadratic Slope	0.001	-0.001	0.000	-0.001
Trajectory 4 **Obese, Increasing (linear)**	Intercept	34.905***	35.000***	36.208***	33.745***
	Linear Slope	0.042***	0.069***	0.039***	0.011
	Quadratic Slope	-0.006*	-0.005*	-0.005*	-0.008***
Trajectory 5 **Morbidly Obese, Increasing (decelerating)**	Intercept	43.441***	43.504**	47.279***	42.239***
	Linear Slope	0.077***	0.073***	0.146***	0.097***
	Quadratic Slope	-0.033***	-0.030***	-0.292***	-0.033***

M_0 is the unconditional, time-only model; M_1 controls for mortality, attrition and proxy status; M_2 controls for socio-economic status measures; M3 controls for health status measures. *p <0.05; **P <0.01; ***p </= 0.001.

Table 16: Sub-Group Characteristics According to BMI Trajectory

	Trajectory 1	Trajectory 2	Trajectory 3	Trajectory 5	Trajectory 5
Body-Mass Index (BMI)	22.0 ± 2.25	25.7 ± 2.90	29.64 ± 3.23	34.27 ± 3.70	41.56 ± 5.51
Demographics					
Female (%)	64.4	47.4	49.1	58.5	74.4
Black (%)[2]	10.6	15.7	21.1	23.8	31.2
Hispanic (%)[2]	6.1	11.7	12.6	10.5	7.7
Age-at-Baseline	55.86 ± 3.12	55.92 ± 3.16	55.71 ± 3.14	55.44 ± 3.16	55.44 ± 3.21
Education	12.47 ± 3.07	11.98 ± 3.36	11.73 ± 3.33	11.41 ± 3.43	11.33 ± 3.05
Health Status					
Self-Rated Health (range 1-5)	2.61 ± 1.18	2.58 ± 1.20	2.57 ± 1.20	2.60 ± 1.21	2.70 ± 1.17
Nagi Index (range 0-6)	1.29 ± 1.62	1.42 ± 1.67	1.59 ± 1.69	1.96 ± 1.74	2.82 ± 1.69
CES-D Index (range 0-9)	4.88 ± 1.74	4.93 ± 1.77	4.99 ± 1.81	5.25 ± 1.80	5.57 ± 1.81
Index Chr. Diseases (range 0-7)	1.12 ± 1.07	1.10 ± 1.12	1.08 ± 1.12	1.07 ± 1.10	1.15 ± 1.12
Died (%)	17.3	22.1	19.6	14.3	15.8
Dropped-Out (%)	6.1	9.2	7.6	3.4	3.0
Overall % sample[3]	18.4	41.3	27.6	10.2	2.5

[1]Plus-minus represents *means ± SD*.
[2]Non-Hispanic White racial/ethnic group represent the default percentage up to 100%.
[3]Based on fully-adjusted model (i.e., model adjusted for time-varying covariates).
*p <0.05; **P <0.01; ***p < 0.001.

Table 17: Odds Ratios of Trajectory Membership (Before and After Adjustment for Time-Varying Covariates): PROCTRAJ Results

	M_2	M_{2_1}	M_{2_2}
Trajectory # 1			
Reference Group			
Trajectory # 2			
Mortality	1.185	1.162	2.479***
Attrition	1.579***	1.660***	2.416***
Black	1.863***	1.763***	1.342
Hispanic	2.188***	2.149***	3.438***
Female	0.427***	0.434***	0.836
Education	0.960***	0.958***	0.394
Age_1992	1.002	1.005	0.955**
BMI_1992			2.654***
Trajectory # 3			
Mortality	0.955	0.886	1.132
Attrition	1.179	1.269	2.250**
Black	2.651***	2.502***	1.412
Hispanic	2.300***	2.305***	3.117***
Female	0.450***	0.436***	0.847
Education	1.049***	1.041***	1.020
Age_1992	0.980	0.975*	0.878***
BMI_1992			9.786***
Trajectory # 4			
Mortality	0.758*	0.698**	0.238***
Attrition	0.623*	0.736	1.161
Black	3.037***	3.133***	1.300
Hispanic	1.520*	1.697**	1.921*
Female	0.683***	0.554***	0.641**
Education	0.919***	0.936***	1.043
Age_1992	0.955***	0.957**	0.856***
BMI_1992			17.236***
Trajectory # 5			
Mortality	0.450***	0.394***	0.037***
Attrition	0.392*	0.542	0.750
Black	3.228***	3.007**	0.701
Hispanic	0.933	0.925	0.872
Female	0.999	0.812	0.505**
Education	0.910***	0.927**	1.121**

Age_1992	0.933***	0.939**	0.815***
BMI_1992			25.103***
Group Membership			
Trajectory # 1	19.11%	18.39%	16.82%
Trajectory # 2	42.82%	41.32%	35.43%
Trajectory # 3	26.51%	27.55%	30.98%
Trajectory # 4	9.28%	10.22%	13.53%
Trajectory # 5	2.28%	2.52%	3.24%

M_2 is the model without adjustment for socio-economic and health covariates (i.e., minimally-adjusted model); M_{2_1} is the model adjusted for time-varying socioeconomic and health covariates; M_{2_2} is the model adjusted also for baseline BMI (i.e., fully-adjusted model).

*p <0.05; **P <0.01; ***p < 0.001.

Appendix 3: Chapter IV

Table 18: Bayesian Information Criterion (BIC) and Aikeke Information Criterion (AIC) Scores for Various Numbers of Groups

Number of Groups	BIC (N=82,512)	BIC (N=10,314)	AIC
2	-238,318.1	-238,309.8	-238,280.8
3	-231,338.8	-231,326.3	-231,282.9
4	-227,793.6	-227,777.0	-227,719.1
5	-226,227.3	-226,206.5	-226,134.1
6	-225,445.4	-225,420.6	-225,333.7
7	-224,767.3	-224,738.2	-224,636.8
8	-223,866.7	-223,833.4	-223,717.5
9	-223,355.1	-223,298.9	-223,103.4
10	-223,341.3	-223,178.2	-223,002.4

Figure 9: Bayesian Information Criterion (BIC) and Aikeke Information Criterion (AIC) Scores for Various Numbers of Groups

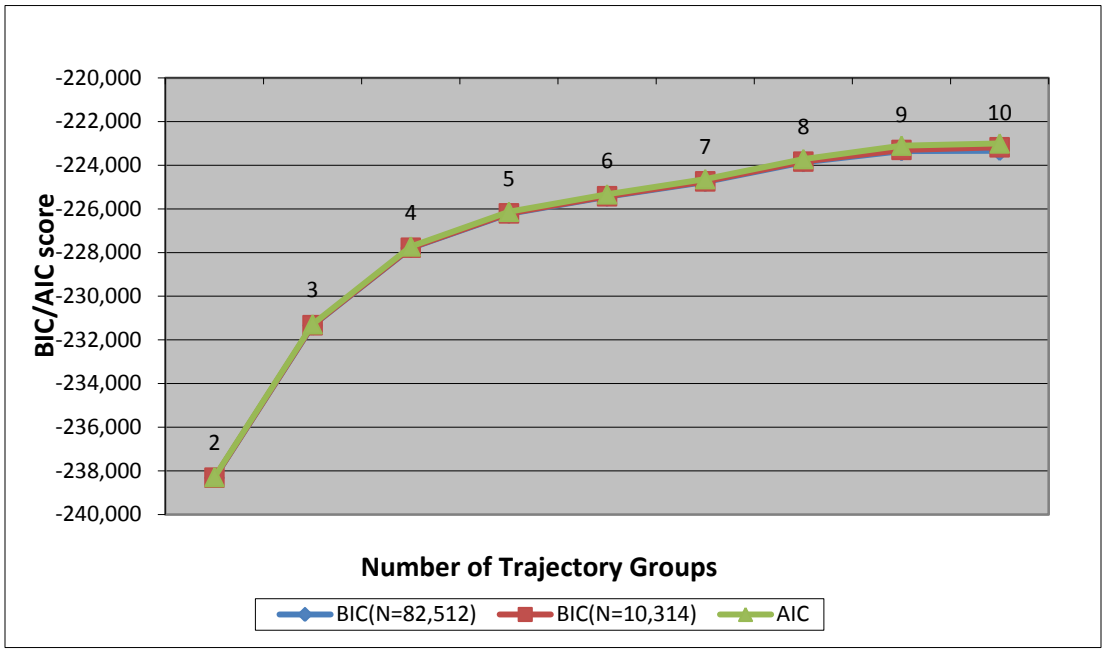

Table 19: Hierarchical Models with Time-constant and Time-varying covariates (PROC TRAJ results)

Parameters	**M_0**	**M_1**	**M_2**	**M_3**	**M_4**
Trajectory Parameters with Time-varying Covariates(β-coefficients)					
Trajectory # 1					
Intercept	22.121***	22.015***	21.602***	21.505***	21.538***
Linear Slope	0.069***	0.064***	0.073***	0.073***	0.111***
Quadratic Slope	0.011***	0.011***	0.010***	0.012***	0.008***
Proxy $_{(t-1)}$		0.913***	0.839***	0.892***	1.077***
Δ Proxy		-0.719***	-0.714***	-0.712***	-0.607***
Assets $_{(t-1)}$			0.016	0.002	0.002
Δ Assets			0.153***	0.145***	0.139***
Income $_{(t-1)}$			0.038	0.021	0.021
Δ Income			0.011	0.002	0.003
Marital Status $_{(t-1)}$			0.386***	0.397***	0.324***
Δ Marital Status			0.687***	0.691***	0.665***
Self-Rated Health $_{(t-1)}$				0.028	0.032
Δ Self-Rated Health				0.017	0.028
Index Disease $_{(t-1)}$				0.010	0.006
Δ Index Disease				0.050	0.056
NAGI $_{(t-1)}$				0.017	-0.029
Δ NAGI				-0.029	-0.072**

CES-D $_{(t-1)}$				-0.017	-0.016
Δ CES-D				-0.039	-0.038
Trajectory # 2					
Intercept	26.067***	26.081***	26.208***	25.679***	25.603***
Linear Slope	0.069***	0.072***	0.073***	0.073***	0.118***
Quadratic Slope	0.002**	0.002	0.002	0.001	-0.003**
Proxy $_{(t-1)}$		-0.327***	-0.447***	-0.406***	-0.116
Δ Proxy		-0.812***	-0.844***	-0.907***	-0.940***
Assets $_{(t-1)}$			-0.083***	-0.076**	-0.077**
Δ Assets			-0.044	-0.035	0.011
Income $_{(t-1)}$			-0.127***	-0.098***	-0.067*
Δ Income			-0.072**	-0.061*	-0.044
Marital Status $_{(t-1)}$			0.521***	0.560***	0.375***
Δ Marital Status			0.338***	0.346***	0.361***
Self-Rated Health $_{(t-1)}$				0.026	0.007
Δ Self-Rated Health				0.037	0.030
Index Disease $_{(t-1)}$				-0.018	-0.01
Δ Index Disease				-0.019	-0.019
NAGI $_{(t-1)}$				0.209***	0.073***
Δ NAGI				0.192***	0.115***
CES-D $_{(t-1)}$				0.002	0.019
Δ CES-D				-0.031*	-0.028
Trajectory # 3					
Intercept	29.869***	29.974***	30.432***	29.484***	28.593***
Linear Slope	0.015*	0.028***	0.039***	0.033***	-0.012

Quadratic Slope	0.001	-0.001	0.000	-0.001	0.006***
Proxy $_{(t-1)}$		-1.716***	-1.652***	-1.619***	-1.439***
Δ Proxy		-0.682	-0.730***	-0.847***	-0.826***
Assets $_{(t-1)}$			0.084**	-0.077*	-0.080**
Δ Assets			0.028	0.038	-0.02
Income $_{(t-1)}$			-0.215***	-0.207***	-0.096**
Δ Income			-0.137***	-0.121**	-0.065*
Marital Status $_{(t-1)}$			0.306***	0.504***	0.236***
Δ Marital Status			0.159	0.358***	0.158*
Self-Rated Health $_{(t-1)}$				-0.016	0.015
Δ Self-Rated Health				0.031	0.032
Index Disease $_{(t-1)}$				0.020	0.001
Δ Index Disease				-0.006	0.008
NAGI $_{(t-1)}$				0.496***	0.274***
Δ NAGI				0.340***	0.245***
CES-D $_{(t-1)}$				-0.049*	-0.034
Δ CES-D				-0.037*	-0.021
Trajectory # 4					
Intercept	34.905***	35.000***	36.208***	33.745***	32.800***
Linear Slope	0.042***	0.069***	0.039***	0.011	-0.011
Quadratic Slope	-0.006*	-0.005*	-0.005*	-0.008***	-0.001
Proxy $_{(t-1)}$		-3.056***	-3.439***	-3.360***	-2.910***
Δ Proxy		-0.208	-0.179	-0.261	-0.379**
Assets $_{(t-1)}$			-0.148**	-0.135**	-0.209***
Δ Assets			0.044	0.077	0.035

Income $_{(t-1)}$			-0.418***	-0.228***	-0.136**
Δ Income			-0.334***	-0.219***	-0.187***
Marital Status $_{(t-1)}$			0.128	0.237*	0.370***
Δ Marital Status			-0.572***	-0.532***	-0.351**
Self-Rated Health $_{(t-1)}$				0.845	-0.024
Δ Self-Rated Health				0.001	-0.035
Index Disease $_{(t-1)}$				-0.028	0.048
Δ Index Disease				-0.036	-0.022
NAGI $_{(t-1)}$				0.829***	0.627***
Δ NAGI				0.631***	0.522***
CES-D $_{(t-1)}$				0.000	0.024
Δ CES-D				0.065*	0.058*
Trajectory # 5					
Intercept	43.440***	43.504**	47.279***	42.239***	40.766***
Linear Slope	0.077***	0.073***	0.146***	0.097***	0.019
Quadratic Slope	-0.033***	-0.030***	-0.292***	-0.033***	-0.026***
Proxy $_{(t-1)}$		-6.364***	-5.620***	-4.688***	-5.259***
Δ Proxy		-0.974**	-1.186**	-1.553***	-1.196***
Assets $_{(t-1)}$			-0.496***	-0.588***	-0.468***
Δ Assets			0.310**	-0.218*	0.187
Income $_{(t-1)}$			-1.208***	-0.584***	-0.540***
Δ Income			-0.617***	-0.337**	-0.348***
Marital Status $_{(t-1)}$			-0.491	-0.146	0.174
Δ Marital Status			-0.758	-0.154	-0.254
Self-Rated Health $_{(t-1)}$				-0.189*	-0.186*

Δ Self-Rated Health				-0.044	-0.066
Index Disease $_{(t-1)}$				0.186***	0.202***
Δ Index Disease				0.039	0.101
NAGI $_{(t-1)}$				1.205***	1.314***
Δ NAGI				0.749***	0.818***
CES-D $_{(t-1)}$				0.108	0.029
Δ CES-D				0.137*	0.084
SIGMA	3.244***	3.245***	3.234***	3.203***	3.223***
Group Membership with Time-constant Variables (β-coefficients)					
Trajectory # 1					
Reference Group					
Trajectory # 2					
Constant		0.683***	1.46*	1.332*	-19.076***
Mortality		0.304***	1.19	0.150	0.908***
Attrition		0.532***	1.58***	0.507***	0.882***
Black			1.86***	0.567***	0.294
Hispanic			2.19***	0.765***	1.235***
Female			0.43***	-0.835***	-0.178
Education			0.96***	-0.043***	-0.032
Age_1992			1.00	0.005	-0.046**
BMI_1992					0.976***
Trajectory # 3					
Constant		0.239***	2.297***	2.574***	-49.849***

Mortality	0.151	-0.046	-0.121	0.124
Attrition	0.250	0.165	0.238	0.811**
Black		0.975***	0.917***	0.344
Hispanic		0.833***	0.835***	1.136***
Female		-0.799***	-0.830***	-0.166
Education		-0.048***	-0.040***	0.020
Age_1992		-0.021	-0.025*	-0.130***
BMI_1992				2.281***
Trajectory # 4				
Constant	-0.690***	2.964***	2.869***	-66.642***
Mortality	-0.275*	-0.277*	-0.359**	-1.435***
Attrition	-0.58**	-0.473*	-0.307	0.149
Black		1.111***	1.142***	0.261
Hispanic		0.419*	0.529**	0.653*
Female		-0.381***	-0.591***	-0.445**
Education		-0.085***	-0.066***	-0.155***
Age_1992		-0.046***	-0.044**	2.848***
BMI_1992				
Trajectory # 5				
Constant	-2.099***	2.783*	2.575*	-78.964***
Mortality	-0.192	-0.798***	-0.931***	-3.310***
Attrition	-0.742	-0.937*	-0.613	-0.288
Black		1.172***	1.101***	-0.355
Hispanic		-0.068	-0.078	-0.137
Female		-0.001	-0.209	-0.683**
Education		-0.094***	-0.076**	0.114**

Age_1992			-0.069**	-0.063**	-0.205***
BMI_1992					3.223***
Group Membership					
Trajectory # 1	19.89%	19.61%	19.11%	18.39%	16.82%
Trajectory # 2	43.80%	42.91%	42.82%	41.32%	35.43%
Trajectory # 3	25.39%	26.06%	26.51%	27.55%	30.98%
Trajectory # 4	8.85%	9.16%	9.28%	10.22%	13.53%
Trajectory # 5	2.07%	2.25%	2.28%	2.52%	3.24%
BIC (N=82,512)	-226,227.3	-225,658.7	-225,387.7	-224,898.2	-218,784.8
BIC (N=10,314)	-226,206.5	-225,619.2	-225,296.2	-224,765.1	-218,647.5
AIC	-226,134.2	-225,481.6	-224,977.6	-224,201.7	-218,169.6

M_0 is the unconditional, time-only model; M_1 controls for mortality, attrition and proxy status; M_2 controls for socio-economic status; M_3 controls for health status; M_4 controls for baseline (1992) BMI.
*p <0.05; **P <0.01; ***p < 0.001.

Table 20: Mean values and differences between self-reported and interviewer-measured height and weight values

		Self-reported measure	Interviewer-obtained measure	
		Mean	Mean	Mean Difference
2004	WEIGHT (lb)	177.86	183.00	(5.14) ***
	HEIGHT (in.)	67.08	65.75	1.33 ***
2006	WEIGHT (lb)	177.90	180.52	(2.62) ***
	HEIGHT (in.)	66.99	66.90	0.09 ***

*** p-value < 0.05.

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CHAPTER V: CONCLUSIONS

The fundamental question collectively addressed in the three dissertation essays is this: "*What is the trajectory of body-weight from middle to older age and what modifies its shape?*" Because of the strong association between body-weight and mortality (which eliminates sicker individuals from their cohort before they reach old age) on the one hand, and physical, functional and cognitive morbidities (which compound to define successful vs. usual vs. pathological aging) on the other, improving the health of the aging population and/or reducing the health disparities in old age call for an understanding of weight trajectories and their determinants.

The Discussion section of each essay elaborates on the essay's findings, places them within the existing literature, with an emphasis on how they extend current knowledge and contribute to elucidating some unresolved issues, and proposes a number of directions for future research. The following discussion will not reiterate what has already been said in each essay, but will attempt to compare the findings from the three essays and draw collective conclusions, to create a cohesive piece of research and direct attention to areas that may benefit from further investigation.

V.1. TRAJECTORIES OF BMI FROM MIDDLE TO OLD AGE: AVERAGE VS. GROUP-BASED HETEROGENEITY

The first essay quantitatively depicts the change in body-weight from mid-adulthood into older age, showing that, on average, BMI increases following a rather linear trajectory. The third essay validates the hypothesis that significant heterogeneity in body-weight development underlies the "average" trajectory of BMI in this age group and identifies five distinct trajectories. These trajectories

differ both in terms of intercept, with the five groups largely representing the five commonly accepted BMI categories – normal BMI, overweight, borderline-obese, overtly obese, and morbidly obese, and in terms of rate-of-change over time. Differences in the rate-of-change between the five trajectories are fairly small (yet significant), with two trajectories showing linear patterns of increase, while the other three follow a quadratic pattern. First impression aside, the two essays complement rather than contradict each other. This is because they illuminate important empirical regularities in the development of BMI across the life span, namely that BMI increases with time in older age, following a number of distinct ascending trajectories. It is worth mentioning that additional analyses (not included in the third essay write-up) of mixed-models with between two- and ten distinct BMI trajectories revealed only positive, either linear or quadratic, slope trajectories, further supporting this conclusion.

As stated in the respective essays, hierarchical linear modeling and group-based mixture modeling provide alternative statistical tools for measuring and explaining differences across population members in their developmental course. Because the two approaches share the common theoretical goal of modeling individual-level heterogeneity in BMI trajectories, each must make assumptions about the distribution of trajectories within the population. It is these assumptions that differentiate the two approaches and dictate the type of conclusions that can be drawn from each. Hierarchical linear modeling generally assumes that the trajectory parameters are normally distributed throughout the population and that covariates of interest (e.g., demographic, socio-economic, or health status characteristics) explain the deviation of individuals from the norm trajectory. As such, an average trajectory (i.e., intercept and slope) is depicted and the deviation from the average associated with each individual characteristic is quantified. Conversely, group-based modeling assumes that there may be clusters or groupings of developmental trajectories, reflecting qualitatively distinctive groups within the population. Using this approach, multiple distinct trajectories are identified, each with unique parameters, population prevalence and predictors. This observation is of particular

significance, because distinct trajectories may reflect distinctive etiologies and may have different clinical significance (i.e., different health consequences). It also suggests perhaps the most critical direction for future research, namely to evaluate whether each of the identified trajectories carries a differential disease risk. Linking each trajectory to an increased risk for specific morbidities may assist targeted interventions aimed at improving aging health outcomes within the population.

Both methods result in estimates of trajectory parameters and a mathematical formula that approximates each trajectory. This should not be construed to mean that inferences can be made about the shape of the trajectories outside the range of actual observations. In other words, no conclusions can be drawn about the trajectory of BMI in individuals younger or older than the group observed in these two studies. The participants in these studies represented the 1931-1941 birth years and were followed from 1992 to 2006. The youngest participants in the HRS cohort were enrolled in the study at age 50; the oldest respondents were followed up to age 75. As such, a second important direction for future research is to expand the analysis to include younger and older age-groups, such that a cohesive depiction of a life course trajectory of BMI is obtained.

V.2. RACIAL/ETHNIC, EDUCATIONAL AND GENDER DIFFERENCES IN TRAJECTORIES OF BMI

The first and the third essay find significant racial/ethnic and educational differences in the trajectories of BMI, but diverge in their findings on gender variations. While the first essay finds no gender differences in BMI trajectory parameters, the third essay finds subtle distinctions – men show an approximately two-fold increase in the risk of following the obese and morbidly-obese trajectories compared with women, while the propensity to follow the overweight trajectories is similar between genders. This raises an interesting possibility, namely that "averaging out" of significant population differences occurs under the classic growth curve modeling approach, and emphasizes the importance of using multiple complementary methods for the study of population heterogeneity.

In younger age groups, minorities (African-Americans and Hispanics) tend to exhibit similar trajectories of BMI, with a higher intercept and higher rates-of-increase compared with Whites. Our work presents a more complex picture of racial/ethnic differences in BMI trajectories in older adults and highlights the import of the BMI with which adults enter middle age in determining the subsequent trajectory of body-weight. To summarize, the first and the third essay concur in showing that African-Americans tend to follow higher BMI trajectories compared with Whites, with most differences explained by the baseline BMI. Conversely, the unadjusted differences between Hispanics and Whites are small and increase after adjustment for baseline BMI. This is an important observation, highlighting distinct "critical periods" for interventions aimed at reducing racial disparities in obesity-related aging outcomes (before entering middle age for Black-White differences and continuing into old age for Hispanic-White differences).

A shared limitation of the two studies lies in their rather under-refined specification of racial/ethnic groups and race-relevant moderators/mediators. Race groups do not indicate biological differences, but stand as proxies for distinct life experiences, exposures to protective and detrimental health risk factors, and interactions with social institutions. The diversity of such experiences mandates a more refined racial/ethnic sub-group classification within the three traditionally recognized groups, as well as the recognition of other factors (e.g., culturally-entrenched health behaviors, immigration status, social networks and social support) as intervening factors in the linkage between race and BMI trajectory.

V.3. THE EFFECT OF HEALTH BEHAVIORS ON THE TRAJECTORY OF BMI

The second essay attempts to quantify the effects of smoking, physical activity and alcohol use on the long-term trajectories of BMI and finds, not entirely unexpected, that smokers and individuals involved in vigorous physical activity have lower trajectories of BMI over time, and that physical activity can counter the weight-gaining effect of smoking cessation in older adults. The significance of the findings and their concordance or divergence from prior studies are noted in the essay

write-up. The following discussion will focus on work not included in the write-up, which tests a number of alternative explanations and refinements of the study findings.

Racial/ethnic differences in the effect of health behaviors

The racial/ethnic differences observed in the trajectories of BMI (essays #1 and 3) and previous reports of a moderating effect of health behaviors on the relationship between race/ethnicity and various health outcomes (Lantz et al., 2001) suggest a possible differential distribution of behavioral effects by race/ethnicity. This hypothesis was tested and revealed no significant interactions between race/ethnicity and health behaviors in their effect on the trajectory of BMI. The result indicates that although behavioral profiles and the propensity for behavioral modifications are differentially distributed across the socio-economic hierarchy (Honda, 2005; Lynch, Kaplan, & Salonen, 1997; Wray, Alwin, & McCammon, 2005), the effect of health risk behaviors on body-weight extends to all the groups under consideration in this study. Two important implications to be derived from these results are that: (1) the consequences of beneficial changes in health lifestyles (smoking cessation, increase in physical activity involvement) may extend along the socio-economic ladder, and (2) it is unlikely that interventions aimed at behavioral modifications will result in a mitigation of social disparities in obesity-related health outcomes.

Health behaviors measurement issues

The dichotomous categorization of participants according to their user status for each of the three behaviors was dictated by practical considerations (i.e., coding consistency between waves) and supported by exploratory analyses. Supplementary analyses using "number of cigarettes per day" and "number of drinks per day" as indicators of smoking and alcohol use did not significantly alter the results. Unfortunately, sensitivity analyses using other indicators of vigorous physical activity involvement were not possible, because the coding varied significantly

between waves and allowed for consistent coding only as a dichotomous variable (user/active vs. non-user/inactive).

Two considerations mandate further refinements in the assessment of health behaviors. First, a number of prior studies reported a J-shaped relationship between alcohol use and BMI (Arif & Rohrer, 2005; Colditz et al., 1991; Lukasiewicz et al., 2005). Others, including the supplementary analyses in our study, did not find evidence to support a non-linear relationship. Nevertheless, the inconsistent findings on the alcohol – BMI relationship highlight the importance of testing behavioral indicators that allow for a more sophisticated evaluation of non-linear patterns of association. Second, alternative indicators of health behaviors, for example type of alcoholic beverage and percent alcohol consumed (Kiefer & Spanagel, 2006; Lukasiewicz et al., 2005), or type (strength vs. cardio training) and intensity of physical activity (Nelson et al., 2007), may show different patterns of association with BMI. As such, a more discriminate understanding of the relationships between BMI and alternative measures of health behaviors may assist in the design of interventions aimed at maintaining health weight in older adults.

V.4. LIMITATIONS AND ISSUES FOR FURTHER INVESTIGATION

Despite the improvements offered by each of the three essays, they also share some important limitations. Two of the limitations, namely the potential for bias associated with the self-reporting of weight and height, and the imperfect nature of BMI as a measure of overweight/obesity in older adults, are discussed in each of the studies. When evaluating longitudinal studies of health in older populations, two additional issues are worth mentioning:

Left truncation and the potential for selection bias due to mortality

Death, either among eligible persons within the cohort of interest prior to entering the study (left truncation) or among enrolled participants during the study (right truncation), modifies the composition of the study cohort and challenges the representativeness of the results. If the event or mechanism leading to censoring

(truncation), such as death or attrition, is systematically related to the outcome of interest, it exerts a selection effect on the cohort over time. Overweight/obese adults are more likely to die at any stage in the life course compared with normal weight individuals (Berrington de Gonzalez et al., 2010), resulting in a "healthy survivor bias" attributable to healthier persons living longer and contributing more data to the study.

A recent review on the treatment of mortality in longitudinal studies of health in older adults provides guidelines for the use of alternative statistical methods for minimizing the potential for selection bias associated with right truncation (Murphy et al., 2011). The approach adopted in all three essays, similar to pattern-mixture modeling (Hedeker & Gibbons, 2006), consisted in the inclusion of mortality/attrition indicators in successively adjusted models and revealed considerable differences in BMI trajectories between individuals who died and those alive at the end of the study. The significant associations between mortality/attrition indicators and BMI trajectory estimates underscore the potential for bias due to non-random right truncation during the study period.

The treatment of left truncation is more problematic. It is reasonable to assume that overweight/obese adults died at a disproportionately higher rate prior to the start of the HRS in 1992 and that the cohort admitted into the study represented a "healthier" remnant of the original birth cohort. Adjustments for left truncation bias require assumptions about the differential rates of death within the specific cohort prior to entering the study. Such assumptions are difficult to formulate and substantiate, hence no suitable method for dealing with left censoring due to death is currently available for outcomes other than survival time (Singer & Willett, 2003). The limitations on the representativeness of findings from gerontological studies arising from death-related left truncation can be overcome only by extending the period of observation, ideally to include the entire life-course. Obvious impracticalities aside, studies of long-term BMI trajectories starting in young age can provide a more complete picture of body-weight development across the life

course and clarify the association between body-weight and potential confounders at every stage.

Cohort Definition and Time vs. Age Effects

Cohort analyses in which the joint effects of aging, time (historical) change and birth cohort membership are estimated for a specific outcome are often desirable on theoretical grounds. Even when only one of the variables is the focus of investigation, such analyses are complicated not only because age, time period and birth cohort are linearly dependent on each other, but also because of the variability in the boundaries of cohorts. In each of the three essays, I argue that a single cohort is analyzed in order to minimize the potential for bias due to cohort differences in BMI (Reynolds & Himes, 2007). This is technically correct according to the HRS specifications, yet a discussion on the meaning of "cohort" and on the implications for the interpretation of results is warranted. The HRS defines its original cohort on the basis of a 10-year birth interval (1931-1941). This definition is concordant with the accepted medical/epidemiological view of a cohort as *"the population born during a particular period and identified by period of birth so that its characteristics can be ascertained as it enters successive time and age periods"* (Stedman's Medical Dictionary, 28th edition, 2006). In contrast, sociological definitions emphasize the common characteristics of cohort members attained through shared life experiences (e.g., *"Successive cohorts are differentiated by the changing content of formal education, peer-group socialization and by idiosyncratic historical experience"*; Ryder, 1965). The two definitions highlight not only interdisciplinary differences, but also the inherent relativity in cohort analyses. Case in point, the 10-year birth interval used by the HRS in defining its original "cohort" may be interpreted as representing one or more actual cohorts (a brief review of epidemiological studies involving BMI revealed a wide-range of intervals used to identify cohorts, varying from one to ten years, most often five years; available upon request). As such, to exclude (at least to some extent) the possibility of residual cohort effects influencing the results, additional analyses were done by dividing the participants into an early-cohort

(1931-1935 birth years) and a late-cohort (1936-1941) group and separately modeling the trajectories of BMI for each group. The results showed only minimal differences in terms of intercept, with both groups following increasing BMI trajectories over time. Additional studies are needed to further refine the understanding of cohort differences in BMI trajectory, as they may suggest lifestyle, cultural, or societal factors responsible for the observed "epidemic" increase in the prevalence of obesity in recent cohorts.

Evaluating the effects of age on BMI trajectories is a desirable research goal. Yet, as explicitly stated in the essays, because the HRS data is not suitable for age-based analyses, I have chosen to undertake a time-based analysis of BMI in a single cohort. Consequently, the results reflect the evolution of BMI over time in a specific age-group, rather than the effect of age on the trajectory of BMI. Time-based models specify intra-personal changes as a function of time since a fixed benchmark (usually the beginning of the study) (Alwin, Hofer, & McCammon, 2006), while in age-based models age, rather than time, is used in estimating the growth parameters. Because in a time-based analysis age and time are perfectly collinear, we can only estimate time-related intra-personal changes while controlling for age-at-baseline. As such, inferences can be drawn about how the effect of time on BMI is modified by a one-year increase/decrease in age-at-baseline, but not on how BMI progresses as a function of age. A time-based analysis (in which age- and cohort effects are usually confounded) yields results different from those of an age-based analysis (see discussion in Liang et al., 2008). Given appropriate data (i.e., where members of different cohorts are observed at the same age points over extended periods of time), age-based analyses have the capacity to separate the effects of age and cohort (McArdle & Anderson, 1990). This is not accomplished in the present study.

V.5. DIRECTIONS FOR FUTURE RESEACH

I envision two key directions for future research: (1) investigating the effect of obesity on health outcomes in older age using alternative body-composition

indicators, and (2) elucidating the biological mechanisms linking social stratification patters, obesity and health outcomes in older age. The two topics were chosen not necessarily because they address some theoretical or methodological limitation of the work presented here, but because they naturally tap into the current medical and public health debate over the role of obesity in aging.

The first direction involves *the investigation of alternative body composition indicators* (e.g., % lean body mass ratio, % fat mass, visceral fat mass, mineral bone content). Understanding the "natural" time-based progression and social patterning of body-composition indicators other than BMI may refocus clinical and policy attention, by suggesting alternative targets for obesity-reducing interventions. In addition, "why do poor people behave poorly?" (Lynch et al, 1997) is still a question without definitive empirical answers. Better understanding the role of various health behaviors in shaping the trajectories of body-composition indicators may clarify some of the mechanisms underlying the observed socio-economic disparities in aging outcomes and assist in the design of policies and interventions aimed at reducing the excessive burden of bad health in disadvantaged elderly individuals.

The second direction targets *the biological mechanisms linking body-weight to morbidity and mortality* across the life course. Inflammatory, metabolic and pre-clinical vascular processes may contribute to the onset and progression of various health conditions, such as functional decline and disability, cardiovascular disease, diabetes and cognitive impairment. However, the empirical evidence on the longitudinal relationship between body-weight (or other body-composition indicators), inflammatory/metabolic/vascular biomarkers, and health outcomes is currently very limited. Studies are needed to evaluate the socio-economic patterning of biomarkers-health relationships and how distinct trajectories of body-weight relate to longitudinal changes in biomarkers. The results of such studies may add to the empirical evidence on the role of inflammation and metabolic processes in aging and help clarify the biological pathways linking obesity to age-related health outcomes. This is of particular import, given that many of the metabolic or

inflammatory pathologies assumed to be related to obesity (on one hand) and to detrimental health outcomes (on the other) are currently amenable to therapeutic interventions.

For practical purposes, this research project on the longitudinal linkage between body-weight and aging has been divided into three essays. The essays complement each other, and together and individually offer significant improvements over the existing literature on the role of obesity in aging. The work presented here provides the first, to our knowledge, depiction of the long-term trajectory of body-weight and its predictors in older adults, reveals significant population heterogeneity in body-weight development, and suggests that lifestyle modifications can achieve/maintain optimal body-weight profiles in old age. The fundamental assumption underlying this research is that aging is a mutable condition. Obesity is one of the life course injuries threatening the prospects of successful aging. Predicting whether we, as a society, will continue to compress the "inevitable" age-related morbidity towards the end of the possible biological life span, or whether we will witness an "extension of misery" due to obesity is one of the most significant questions facing the clinical and public health disciplines in our time. The answer is likely to carry profound scientific, political and ethical ramifications…

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