Exercise or Extra Fries? Behavioral Drivers of Obesity Over the Life Cycle*

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Abstract

As America faces the obesity epidemic and its economic consequences, the factors driving weight variation across the U.S. population remain unclear, especially since these differences extend well beyond commonly studied channels like income or education. We leverage a unique thirty-year panel of U.S. registered nurses to examine the changes in body mass index (BMI) with age, the impact of lifestyle choices on body weight in adulthood, and the role of inherited metabolic traits and childhood preference formation. Our empirical findings reveal that among individuals who enter adulthood with a healthy body mass, those in the top diet quality or exercise deciles at age thirty are roughly 50% more likely to maintain a healthy BMI and avoid morbidities three decades later, compared to those in the bottom deciles. To better understand the drivers of weight gain, we propose a novel structural framework of endogenous BMI evolution where initial heterogeneity is amplified by behaviors to explain much of the observed divergence in weight. Counterfactual experiments using the model indicate that initial conditions, such as fixed *metabolic types*, account for at least one-third of the variance in weight at older ages. While course correction through behaviors can only partially compensate for poor initial conditions at the onset of adulthood, we find that promoting healthy behaviors in adults can reduce total healthcare expenses by at least $100 billion annually.

JEL classifications: I12, I14, H51, D15

Keywords: Obesity, diet, exercise, morbidities, BMI, health types, early childhood exposure, metabolism, medical spending, health production function

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1 Introduction

Data from the Centers for Disease Control and Prevention (CDC) shows that over 40% of Americans are classified as obese (Ward et al., 2021). This public health crisis poses significant macroeconomic challenges, with obesity-related medical expenses averaging $2,741 per person annually (in 2005 dollars) (Cawley and Meyerhoefer, 2012) and totaling $261 billion in 2016 (Cawley et al., 2021). While the economic costs of obesity are well-documented, less is understood about the drivers of weight gain, which is crucial for effective policy design. Existing literature attributes rising obesity rates to factors like technological changes in food production, but the reasons for weight variation among adults remain unclear.[1] Specifically, questions persist about why individuals with similar body weights in early adulthood experience different outcomes as they age, how lifestyle choices affect body mass index (BMI), and the role of inherited metabolism and childhood habits. We address these questions here by using a model of endogenous weight evolution which is informed by a unique longitudinal dataset of U.S. registered nurses, thereby filling a critical gap in the understanding of individual weight determination.

Using panel data from the Nurses' Health Study II (NHS II), a 30-year dataset of U.S. female registered nurses starting in 1989, we document key relationships between health behaviors and long-term changes in weight and morbidities. The dataset offers comprehensive information on medical conditions, health behaviors (physical activity and dietary intake), early childhood and adolescent circumstances, and family medical history, providing a unique overview of the progression of health over their life cycle between the ages of 25 and 60. We then develop a structural life cycle model of endogenous health, specifically focusing on the evolution of BMI and its impact on morbidity onset during adulthood. The model abstracts from economic resources, including income and wealth, as the drivers of healthy behaviors. Instead, it examines the role of initial conditions, habits, and preferences in explaining BMI and morbidity patterns. Finally, we use our model to conduct counterfactual experiments to understand the role of initial conditions and healthy behaviors in maintaining lower BMI—and, by implication, lower morbidities—over an individual's life, and consequently, on medical spending in the U.S.

While the focus of our paper is on understanding the drivers and implications of weight disparities in adulthood, it is influenced by and contributes to the existing literature in macro-health. In particular, it aligns well with the recent emphasis in the literature on the significance of fixed health heterogeneity in shaping health trajectories (De Nardi et al., 2022; Borella et al., 2024; Hosseini et al., 2021, 2022). At the same time, our paper distinguishes itself from exisiting studies in two key ways. First, while researchers have mostly focused on general health measures such as self-rated health and frailty, we focus specifically on BMI and its impact on morbidities due to the ongoing obesity crisis and the

[1]For example, Cutler et al. (2003) highlights how the "French fry became the dominant form of potato and America's favorite vegetable" following World War II, due to innovations that centralized French fry production.

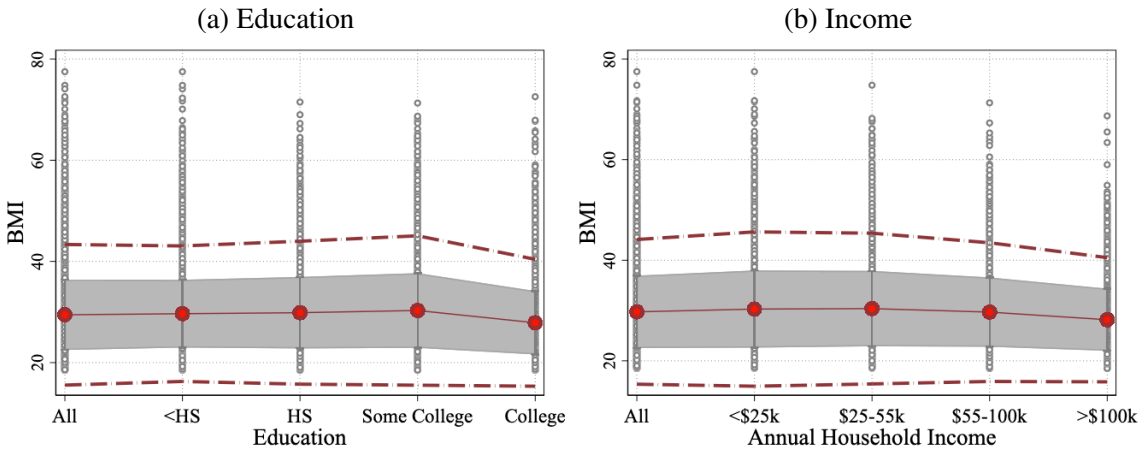


Figure 1: Within and across-group variation in BMI

Notes: Data is from the National Health and Nutrition Examination Survey. The figure plots means, standard deviation (shaded), and two standard deviations (dashed lines) of BMI across different education and income groups. The sample is restricted to individuals between the ages of 25 and 60.

significant macroeconomic challenges caused by this epidemic.[2] Second, our exclusion of economic resources from the structural model of endogenous weight determination is intentional and is distinct from how health is often modeled in this literature. For example, existing studies have shown that income can directly affect health outcomes by facilitating higher preventative medical spending or a greater ability to pay for both health shock treatments and health insurance coverage (Ozkan, 2014; Jung and Tran, 2016; Fonseca et al., 2021; Capatina and Keane, 2024; Chen et al., 2024), or indirectly by affecting incentives to engage in healthy behaviors (Cole et al., 2019; Mahler and Yum, 2022; Margaris and Wallenius, 2024). While these studies improve our understanding of the drivers of the large health disparities that exist in the U.S. population, there are also important limitations and gaps in this literature that our study aims to fill. In particular, our dataset offers unique opportunities to examine lesser-known factors influencing health variations beyond income or wealth, helping bridge the gap between existing theoretical models and empirical evidence (Baicker et al., 2013; Cesarini et al., 2016).

One of the most important gaps in the literature is that significant variations in health exist which cannot be explained solely by income differences. For example, Figure 1 shows the distribution of BMI, a key health indicator, across education groups (proxy for permanent income) and current income levels in the nationally representative National Health and Nutrition Examination Survey. As established in the literature, there is a gradient across groups. For example, average BMI is 28 among college graduates compared to 30 among high school dropouts, with corresponding obesity rates of 29% and 40%, respectively. However, the variance within each group remains substantial, with the standard

[2]Bolt (2021) and Margaris (2017) are among the few exceptions that also model BMI evolution.

deviation of BMI roughly a *quarter* of the group mean in each case. Note that the same holds in the case of other commonly used measures of health such as frailty, where the standard deviation within each education group is roughly as large as the group mean (refer to appendix Figure A1).

Our dataset comprises a highly homogeneous group of registered nurses in terms of socioeconomic differences. While fully controlling for economic differences, including parental, personal, or family income, is challenging, the dataset's design allows us to account for all major income variations and also identify how parental income may have impacted individuals' health. Specifically, all registered nurses in our sample largely have similar permanent incomes given that they have the same education, occupation, gender, and race.[3][4] Additionally in our empirical analysis, we are able to control for remaining variations in family budget sets by controlling for factors such as marital status, number of children, work shifts, spouse's education, and the average income in their residential census tract. Furthermore, given that all survey respondents have similar health insurance coverage due to their shared profession, additional variations in budget sets due to differences in medical spending is of limited concern. Their shared profession also means that the nurses have access to the same health-related information, which has been discussed by Deaton (2024) to be an important mechanism for explaining the education-health gradient. Finally, the potential impact of family wealth is captured in our dataset through key variables shaped by parental investments during early childhood, such as initial diet habits, adolescent weight, and physical activity preferences.

In summary, this survey gives us a clean setting for abstracting from income and wealth differences as causal drivers of health disparities and focusing instead on the extent to which factors like permanent health heterogeneity (or *metabolism*), habits, and preferences play a role in shaping health behaviors and outcomes. At the same time, the distribution of body weight in this sample aligns well with that of a nationally representative survey, providing assurance that insights from our analyses extend well beyond the unique population of registered nurses.

Our thirty year panel provides valuable insights on the joint dynamics of BMI, morbidities, diet, and physical activity over the life cycle. We find that despite improvements in dietary quality and physical activity over adulthood, both BMI and morbidity prevalence tend to rise, with obesity showing high persistence. Among nurses surveyed, 9% were obese (BMI $\geq$ 30) in the initial wave, and over 90% of them remained obese 30 years later. In contrast, health trajectories varied significantly among those with a healthy BMI (18.5–25) and no morbidities early on: 46% maintained a healthy BMI after 30 years (health maintainers or *stayers*), while 19% became obese (*gainers*). We find that diet and

[3] All registered nurses hold at least an associate or bachelor's degree, and 96% of the surveyed nurses are white. Russo et al. (2024) document significant health inequality by race, ethnicity, and gender.

[4] While we do not directly observe the current income or wealth of these nurses, it is not clear how much (or if at all) income or wealth shocks actually matter for the preservation of good health. For instance, using administrative data on Swedish lottery players, Cesarini et al. (2016) show that the causal effects of wealth are not a major source of the wealth-mortality gradient in rich countries.

exercise patterns are strong predictors of membership in these two groups. For example, individuals ranking in the top deciles for exercise or diet at age 30 have approximately 50–60% higher odds of being in the *stayers* group compared to those in the bottom deciles. Additionally, they have roughly 30–40% higher odds of remaining free from morbidities. Metabolic types, inferred from early-life exposures and family history, also played a significant role, with favorable metabolic types associated with 157% higher odds of maintaining a healthy BMI, thirty years later. Finally, we document that diet and exercise exhibit differing patterns of persistence across the life cycle. While diet quality displays high levels of short-run persistence across the entire spectrum, physical activity demonstrates similar persistence mainly at the lower and upper extremes of the distribution (for brevity, referred to as the *inactive* versus *more active* groups). Moreover, diet displays somewhat less persistence than exercise over the very long run.

We use the above stylized facts to discipline an endogenous health model of BMI and morbidity evolution where individuals exert effort to maintain good health. While the model is intentionally kept simple, it contains some key innovations in its design which help fit several important aspects of the data. At its core, the modeling framework posits that rational and forward-looking individuals face the following key trade-off: investing in costly health-maintaining behaviors now to enjoy the benefits of lower BMI, and therefore reduced morbidity prevalence in old age, *versus* higher flow utility today at the expense of poor health later in life. Individuals in the model differ in their permanent states in terms of their metabolic type and exercise preferences, evolving states including their past BMI, morbidity count, and diet habits, and the realization of their i.i.d. idiosyncratic preference shocks to both diet and exercise. We exploit our rich panel data to estimate BMI and morbidities laws of motion that depend on both metabolic type and health behaviors. Exercising and consuming a healthy diet both lead to lower weight and morbidities, but come with utility penalties. A key aspect of our model is to treat the habitual nature of diet and exercise differently, as suggested by our empirical observations. While the habitual nature of the former is captured through adjustment costs for any dietary improvements over past choices, the latter is modeled as permanent preference heterogeneity in attitudes toward physical activity, with the more active group incurring a lower disutility for the same levels of activity compared to the inactive group.

The model is estimated in two steps. First, health production functions for BMI and morbidities are directly estimated from longitudinal data, allowing BMI to influence morbidity evolution but not vice versa, consistent with empirical observations. Second, preference parameters are calibrated to match several model and data moments. Despite its simplicity, the model captures key relationships, including the life-cycle evolution of BMI, morbidities, diet, and exercise. It also aligns with our stylized facts and observed correlations between BMI, diet, and exercise. Alternative models highlight the importance of diet habits and exercise preferences, showing that excluding diet habits or using alternative frameworks, like heterogeneous discount factors, leads to significant mismatches with observed data, particularly in diet changes and exercise distribution.

Our model shows that initial conditions play an important role through a combination

of direct and indirect mechanisms. Individuals entering adulthood with a healthy BMI and no morbidities are more likely to sustain these favorable outcomes in the future due to the persistence in the associated laws of motion. Beyond these direct effects, however, there is an amplification effect through behaviors. A favorable BMI status facilitates higher levels of physical activity and healthier dietary choices because of the data-implied dependence of the disutility functions on current BMI. Similarly, starting adulthood with better diet habits and exercise preferences yield similar amplification effects. Consequently, equalizing initial conditions such as initial BMI, exercise preference types, and initial diet quality significantly affects the evolution of both effort and health outcomes in our model. For example, setting everyone's initial BMI to a healthy level reduces the rate of age-64 obesity by 13 percentage points (pp), while setting it to a class I obese level (BMI of 30–35) increases it by 30 pp. Overall, about one-third of BMI variance at age 50 can be attributed to initial conditions, rising to 60% when unobserved fixed effects are included in the model.

Finally, we utilize our model to investigate the impact of health-promoting behaviors among adults on BMI outcomes. Equalizing effort levels at low or high values has a significant effect on obesity and morbidities, underscoring the substantial influence of behaviors on health progression if individuals are able to maintain the effort over the life-cycle. For example, setting diet and exercise at the 90^{th} percentile of their respective distributions results in reductions in obesity rates by about 15 pp by age 64 and a 10 pp decline in the share of individuals with morbidities. We estimate that these health improvements could lead to an 8% reduction in aggregate annual healthcare expenditures, equivalent to approximately $100 billion based on 2022 national spending estimates. It is important to note that this is a conservative estimate of the total cost savings since the data is restricted to working-age individuals, and additional savings would occur among older individuals (age > 65) that are not captured in our model. At the same time, the estimate is a simple extrapolation from our sample of nurses, who might engage in healthier behaviors than Americans on average.

In summary, three insights emerge from our model about the role of initial conditions and behaviors:

INSIGHT 1: Because of the large effects of initial BMI on future BMI evolution, our paper highlights the potentially important role played by the increase in childhood obesity in the last few decades in causing high obesity prevalence among adult Americans (Ogden et al., 2012; Skinner and Skelton, 2014).

INSIGHT 2: In equalizing all initial conditions to *poor* levels, we find large implications for old-age obesity and the share of those with multiple morbidities. This highlights that course correction through behaviors in adulthood can only take individuals so far if their starting point is not favorable, especially when it applies to multiple factors. Policies that affect adolescent BMI, diet habits, and exercise preferences set early in life can therefore have large implications for adult health outcomes.

INSIGHT 3: Policies that promote healthy levels of diet and physical activity among adults

can have important implications for medical spending, a substantial share of which is tax-financed through Medicare or Medicaid in the U.S. The cost-savings of targeting healthy behaviors are likely larger than our estimates if the full impact on elderly health outcomes is considered.

Related literature and our contributions

Our paper contributes to several strands of the existing literature. First, it complements a growing body of work on the role of permanent or fixed heterogeneity in explaining the evolution of health, which lies beyond the control of economic agents.[5] Using general health measures such as self-rated health (De Nardi et al., 2022) and frailty (Hosseini et al., 2022), researchers have found that health fixed effects are crucial in explaining the variance in health dynamics. Hosseini et al. (2021) further demonstrates that these effects can also account for much of the observed earnings inequality. More recently, Borella et al. (2024) show that health types constructed using a clustering algorithm explain 84% of the variation in health trajectories, independent of observable characteristics.

Our study contributes to and complements this literature in two key ways. First, we specifically demonstrate the importance of permanent metabolic types in explaining BMI dynamics and obesity prevalence. This contribution emphasizes the significance of fixed characteristics in explaining the progression of body weight, distinguishing our work from studies using more general health measures. Second, our study provides new insights into the relationship between *observable* early-life characteristics and econometrically estimated *unobserved* fixed effects. We show that our metabolic index, constructed from early-life indicators and family medical history, correlates with the fixed effects estimated from our dynamic panel model. This correlation suggests that fixed effects partially capture aspects of pre-adulthood health heterogeneity, bridging the gap between observable measures and latent health characteristics. By aligning our metabolic index with estimated fixed effects, we enhance our understanding of how early-life conditions shape permanent health types to influence BMI and morbidities later in life.

Second, our paper is closely related to the literature using structural models to understand the evolution of health (Hall and Jones, 2007; Scholz and Seshadri, 2011; Ozkan, 2014; Jung and Tran, 2016; Cole et al., 2019; Fonseca et al., 2021; Bolt, 2021; Nygaard, 2021; Mahler and Yum, 2022; Chen et al., 2024; Capatina and Keane, 2024). Among these studies, the most closely related is Bolt (2021), which estimates an inter-generational model of parental health investments similar to the human capital investment literature (e.g., Lee and Seshadri (2019)), to explore drivers of BMI using UK data. Bolt's findings align with ours, showing that childhood health investments are more influential on obesity outcomes than investments made during adulthood. However, it is unclear how much of the variation in childhood *health investments* is attributed to parental liquidity constraints versus the transmission of dietary habits and physical activity preferences. In adulthood, our data sug-

[5]This parallels the income dynamics literature, where fixed heterogeneity is found to significantly influence the variance in life-cycle earnings (Guvenen, 2009).

gest that habits and preferences play a significant role in shaping health investments, which raises an important question: could these habits also be passed down intergenerationally, independent of liquidity constraints?

A key strength of our study is that by directly observing factors such as initial diet quality, early childhood factors surrounding birth, and adolescent weight, we are able to draw conclusions about the importance of parental investments, without having to resort to assumptions about their underlying drivers. Similarly, our data enables us to address key challenges in the endogenous health literature by providing insights on two important elasticities: (1) the income elasticity of health investments and (2) the investment elasticity of health. By analyzing a large, homogeneous sample of nurses and utilizing a thirty-year panel on both behaviors and health, we provide reliable estimates of the latter. Our estimated law of motion for BMI suggests that the short-run effects of healthy behaviors are significant, yet relatively small. However, sustained healthy behaviors over the life cycle compound to lead to substantial long-term improvements in BMI and reductions in morbidities. At the same time, by quantifying the importance of permanent health, habits and preference formation, our framework offers an important baseline for disciplining the income elasticity of health investments in quantitative health models with economic channels.

Third, by highlighting the importance of factors like adolescent obesity, physical activity preferences, and diet habits at the onset of adulthood in shaping the evolution of health outcomes over the life cycle, our paper contributes to an extensive literature on early childhood investments and the role of family in shaping economic outcomes of future generations (Becker and Tomes, 1986; Aiyagari et al., 2002; Cunha et al., 2006; Heckman, 2006; Miller, 2020; Caucutt and Lochner, 2020). Our work demonstrates that early childhood investments in healthy diet and exercise preferences can have far-reaching effects on future weight trajectories, which are strongly correlated with the onset of morbidities. These effects can be direct, by shaping preferences established by early adulthood, or indirect, by influencing BMI at the start of adulthood. We show that both types of initial conditions matter for the evolution of weight throughout adulthood.

Fourth and finally, our paper contributes to a growing body of work documenting the heterogeneity in population health outcomes (Deaton, 1999, 2007; Pijoan-Mas and Ríos-Rull, 2014; Miller and Bairoliya, 2023) and the impact of health on medical spending, economic outcomes, and welfare (De Nardi et al., 2010; Capatina, 2015; Conesa et al., 2018; Miller et al., 2019; Hosseini et al., 2021; De Nardi et al., 2022). We extend this literature by documenting significant divergence in BMI and morbidities even in socioeconomically homogeneous groups. We further show how BMI and morbidity trajectories are shaped by individuals' behaviors over the life cycle and quantify the implications of behavior for aggregate healthcare expenses.

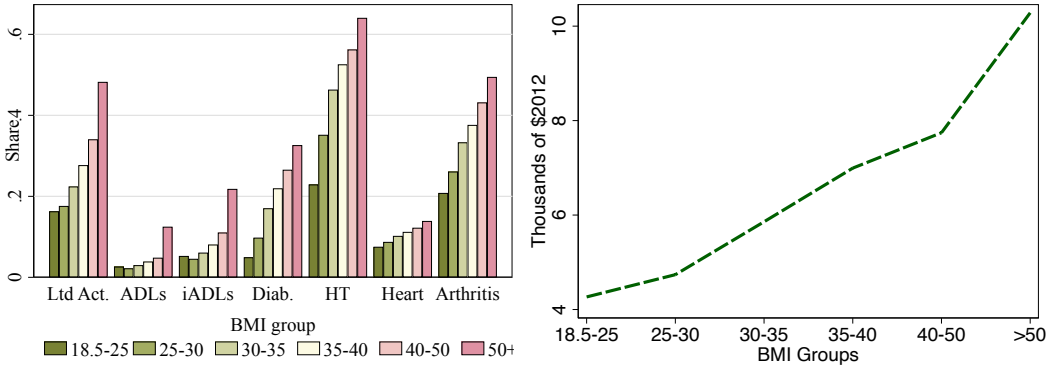
2 Background: Why obesity matters

Understanding obesity and its behavioral drivers is crucial to economists due its impact on chronic conditions and medical spending.[6] The first panel of Figure 2 plots the prevalence of activity limitations and selected morbidities by BMI category using data from the National Health and Nutrition Examination Survey (NHANES). Higher BMI is associated with higher reported incidence of having any activity limitation, greater need for help with (instrumental) activities of daily living (ADLs), and higher prevalence of diabetes, hypertension, heart disease, and arthritis.

Figure 2: Joint distribution of BMI, morbidities, and medical spending

(a) BMI and morbidities

(b) Medical expenditures



Notes: Panel (a) plots the share of those with specific diseases and physical limitations across different BMI groups in the 2015-18 NHANES surveys. Panel (b) plots the mean total medical expenditures for the same demographic across different BMI groups from the MEPS.

The second panel of Figure 2 uses data from the Medical Expenditure Panel Survey (MEPS) to demonstrate the steep BMI gradient in total medical spending. In the U.S., these obesity-induced medical costs are often tax-funded through public insurance programs like Medicare and Medicaid. Aggregate obesity-related medical spending reached an estimated $261 billion in 2016, accounting for about 8% of total annual medical expenditures in the U.S. (Cawley et al., 2021). Of this, an estimated $58 billion was covered by public health insurance programs. The study also found that each additional unit of BMI increases an individual's annual expenditures paid by public insurance by $240, or 7% of the annual mean predicted expenditures, substantially higher than the estimated increase accrued to private insurance. These figures highlight the financial strain on government-funded programs, underscoring the need to examine the factors contributing to obesity to develop effective policies to curb the epidemic.

[6]The drivers of obesity have received considerable attention in the medical and public health literature. We provide a summary of this literature in the supplementary material S1.

3 Data and key measures

We use data from the Nurses' Health Study II (NHS II), which is an ongoing longitudinal study of 116,429 female registered nurses from 15 US states who were between the ages of 25 and 42 in 1989. The goal of the study was to understand oral contraceptives, diet, and lifestyle risk factors. Every 2 years, a questionnaire was sent to each participant to assess lifestyle, health outcomes, and medication use. Every 4 years, a validated food frequency questionnaire (FFQ) was sent to assess usual dietary intake. The survey had a high response rate (85-90% for each two-year cycle). See appendix A2 for attrition analysis.

Registered nurses were selected due to their health knowledge and ability to provide complete and accurate information on a brief, technically worded questionnaire. Additionally, they were expected to be relatively easy to follow over time and more likely to maintain participation in a long-term study than the general U.S. population. This unique survey design has two important benefits for our study. First, having a homogeneous group of individuals like female registered nurses helps us disentangle life-cycle habit-driven behavioral changes from socioeconomic determinants of health, including income, educational attainment, and occupation. Second, having medical professionals as survey respondents reduces the likelihood of measurement error in the key variables of interest for this analysis.

We select our analytic sample by applying two restrictions on the full NHS II sample. First, to limit potentially large cohort effects, we select a narrow birth cohort of those aged 25 to 30 at the time of their first interview in 1989.[7] This cohort was followed over the majority of their adult working-age lives. This restriction reduces the sample size from 116,429 to 26,589 registered nurses. Second, we exclude those reporting a BMI less than 18.5 in any survey wave. An underweight BMI is relatively uncommon in the U.S. population and is often tied to unique circumstances such as morbidities, eating disorders, or chronic food shortage (Uzogara, 2016). This second restriction reduces our final sample to 24,380 registered nurses.

Dietary score

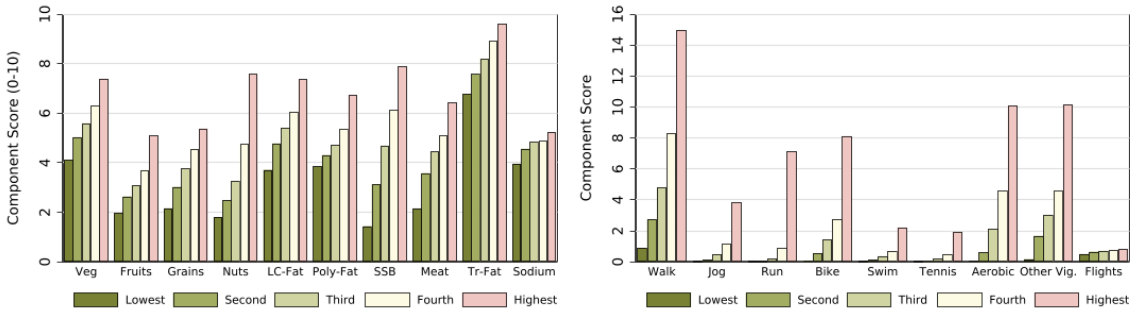
We use a version of the *Alternate Healthy Eating Index* (AHEI-2010) developed by Chiuve et al. (2012) as a measure of diet quality. It is a score that measures adherence to a diet pattern based on foods and nutrients most predictive of disease risk in the public health literature. There are a total of 10 components, each of which has a minimum score of 0 and a maximum score of 10 (see details in appendix Table A1). For example, fruit consumption scores range from 0 (no fruit) to 10 ($\geq$ 5 servings/day). Unhealthy consumption is reverse coded. For example, consuming one or more sugar-sweetened beverages a day receives a score of 0, while zero consumption receives a score of 10. A score between the minimum and maximum is assigned on a continuous basis (except for sodium). For example, if fruit intake is 2 servings/day, the score for this component would be 5. The sodium component was based on FFQ-specific deciles of sodium intake within the study population.

[7] Appendix Figure A2 shows the life-cycle evolution of BMI for different cohorts of nurses.

The AHEI-10 diet quality index is approximately normally distributed with a mean of 49 and standard deviation of 13 (see appendix Figure A3). The average diet quality in the top percentile is 81 while that of the lowest percentile is 24. Figure 3 further shows the composition of various food components within each quintile of the diet quality index. We find stark gradients in the consumption of fruits and vegetables, nuts and grains, as well as long chain and poly unsaturated fatty acids across quintiles of diet quality. For example, less than 1% of individuals in the top diet quintile consumed zero servings of nuts a day compared to almost 12% in the bottom quintile. Similarly, over 70% of individuals in the bottom quintile consumed at least one sugar-sweetened beverage (SSB) a day while this share was only about 7.2% for individuals in the top quintile.

Figure 3: Composition of diet quality and physical activity index

(a) Diet quality index (b) Physical activity index



Notes: Panel (a) shows average individual component score (between 0-10) for each food group within each quintile of diet quality. Panel (b) shows average individual component score (in MET-hours/week) for each activity within each quintile of physical activity index. Refer to appendix tables A1 and A3 for individual components for both diet and activity. Pooled data, for all nurses who were ages 25-30 in 1989, between the years 1991 and 2015 is used.

Physical activity

Physical activity is evaluated through survey questions about common leisure-time activities, which are then converted to metabolic equivalent (MET) hours per week. Specifically, survey participants were prompted on the biennial questionnaire to report their average weekly hours devoted to various physical activities.[8] For each respondent, survey wave, and activity i, a MET score is computed by multiplying (1) the participant's reported median number of hours per week for that cycle and (2) the MET value assigned to the

[8]Hours were selected from a discrete grid. Appendix Table A2 provides both the categorical responses for each activity and the corresponding median value in hours per week. This median value is used to generate a continuous measure of physical activity.

activity (refer to appendix Table A3).[9] The total weekly MET score is then derived as the sum of all MET scores, as shown below:

$$\text{METs per week}_i = \text{median hours per week on } i \times \text{MET value of } i.$$

$$\text{Total METs per week} = \sum_i \text{METs per week}_i.$$

To provide an illustrative example, consider a nurse who reported jogging for 2-3 hours per week; she would have a MET-hours/week from jogging calculated as $2.5 \times 7.0 = 17.5$. The total MET-hours/week is then obtained by summing over each activity. Non-activity variables like standing/sitting at work/home and time spent sleeping are not included in an individual's total MET score.

In our sample, the mean physical activity is 23.2 MET-hours/week, with a median of 14.5 and a standard deviation of 29.4 (see appendix Figure A4). This aligns with the CDC's recommendation of 23 MET-hours/week (150 minutes of brisk walking and two days of muscle-strengthening activity). However, the distribution has a long right tail, with the top 1% reaching 134.6 MET-hours/week, over five times the recommended level, while 15% of the sample engages in less than 3 MET-hours/week, indicating minimal activity. Panel (b) of Figure 3 shows the types of activities and their MET values across different quintiles of our physical activity index. Notably, sports like tennis, squash, racquetball, and swimming are less common among our sample of registered nurses. The highest quintiles are dominated by individuals who engage in extensive walking or vigorous aerobic exercises.

Body Mass Index

Every two years, the survey collects information on weight. Body mass index (BMI) is calculated as weight in kilograms divided by height (from the initial 1989 interview) in meters squared. Approximately half of our analytic sample (pooled across survey waves) falls within the healthy BMI range (between 18.5 and 25). However, the BMI distribution is substantially skewed to the right (see appendix Figure A5). For example, around 20% of the pooled sample is categorized as obese (BMI$\geq$ 30), with approximately 5% classified as class 3 obese according to the CDC definition (BMI$\geq$ 40).

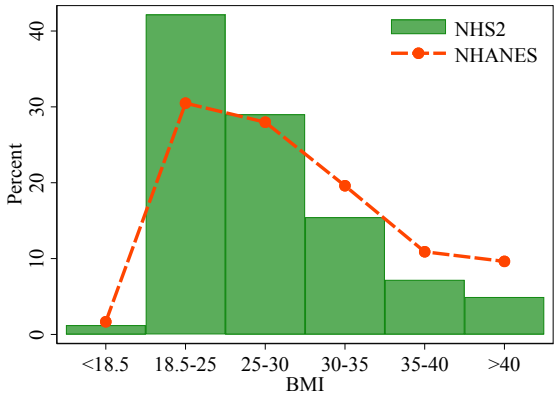
While the NHS II is not intended to be a nationally representative sample, it is valuable to examine how it compares with a more representative survey. Although comparable representative surveys are not available for diet and exercise, we draw a comparison between BMI reported in the NHS II and the widely used National Health and Nutrition Examination Survey (NHANES).[10] Figure 4 illustrates the BMI distribution for the pooled NHS II

[9] MET values for each activity were obtained from Ainsworth et al. (1993). One MET is the energy expended while sitting quietly. Therefore, an activity with a MET score of 5 requires five times the energy of sitting quietly.

[10] The NHANES program began in the early 1960s and collects data on a nationally representative sample of about 5,000 persons each year.

sample and NHANES for women aged 34-65. The shape of the BMI distributions generally align, although the NHS II exhibits a somewhat lower overall BMI. This difference is perhaps unsurprising given the average education and socioeconomic status of nurses compared to the general US population. Nonetheless, the significant distributional overlap provides assurance that insights from our analyses extend well beyond the unique population of registered nurses.

Figure 4: BMI distribution



Notes: Figure plots the BMI distribution in the NHS II and the NHANES surveys. Samples from both surveys are restricted to females between the ages of 34 and 65 pooled across survey years 1999 to 2015.

Morbidity index

Since BMI primarily influences healthcare spending through its impact on medical conditions, we also include a measure of these conditions in our analysis. We construct a morbidity index based on a count of the number of selected medical conditions each survey respondent exhibits. Specifically, we consider confirmed diagnoses for the following conditions: stroke, coronary artery bypass surgery, myocardial infarction, all types of cancers, Type 2 diabetes, Parkinsons's disease, rheumatoid arthritis, multiple sclerosis, inflammatory bowel disease (IBD)/Chron's disease, hypertension, thyroid and kidney stones. Note that several methods were employed by the survey team to validate disease diagnosis reports among survey respondents, including medical record reviews, supplemental questionnaires, and, in some cases, self-reports. For many conditions (e.g., cancer, coronary heart disease), permission was obtained from the respondent to contact their doctor or hospital for medical records, pathology reports, etc., pertaining to the relevant disease. Among our pooled analytic sample, 83% have no morbidities and 3% have two or more morbidities (refer to appendix Figure A6).

Our morbidity index is similar in spirit to a frailty index often employed in the gerontology literature to approximate an individual's health (see, for instance, Searle et al. (2008)). These indices have also become more common in the economics literature (e.g., Braun

et al. (2019); Nygaard (2021); Hosseini et al. (2022)). The gerontology literature constructs these indices by counting how many disabilities and diseases a person has, including functional limitations, cognitive impairments, and various morbidities. Because our goal is to understand how lifestyle choices affect BMI and related health outcomes, we depart from the gerontology literature by restricting the components of our morbidity index to conditions with known hereditary links and medically proven relationships with lifestyle choices, including physical activity and diet quality.

Metabolic index

We construct a measure of metabolic type that influences BMI development but is beyond an adult individual's control, shaped by early childhood exposures, birth circumstances, or genetics. Based on medical literature, factors like birth weight (Yu et al., 2011), breastfeeding (Victora et al., 2016), preterm birth (Johansson et al., 2005), and childhood obesity (Van Cleave et al., 2010) are linked to weight gain and morbidities in later life. Additionally, given the strong genetic ties to obesity, we consider family medical histories, as conditions such as diabetes and cardiovascular diseases have hereditary components.[11] Using 2013 survey data, 33% of nurses reported a family history of myocardial infarction, 19% reported stroke, and 40% reported diabetes.

We consolidate all the aforementioned information into a single measure, which we refer to as the individual's *metabolic index*, by tallying the count of "favorable" circumstances as follows:

$$\text{metabolic_index}_i = \mathbb{I}_{i,bweight} + \mathbb{I}_{i,breastfed} + \mathbb{I}_{i,full-term} + \mathbb{I}_{i,MI} + \mathbb{I}_{i,stroke} + \mathbb{I}_{i,diab} + \mathbb{I}_{i,bmi18},$$

where each indicator variable represents a favorable outcome. For birth weight, a favorable outcome is defined as being born within the normal weight range of 5.5 to 8.4 pounds. For gestational length, a favorable outcome is indicated by a full-term birth. For breastfeeding, the indicator reflects whether the individual was breastfed during childhood. The absence of adolescent obesity is approximated by an age-18 BMI within the healthy range of $[18.5, 25)$. Lastly, favorable outcomes for genetic links to obesity are indicated by the absence of any instances of the three related diseases in the family medical history. In this formulation, we expect a higher metabolic index to correspond to healthier BMI transitions.

Appendix Figure A8 displays the distribution of the metabolic index in our selected sample. Roughly 9% of registered nurses have all seven favorable metabolism outcomes, while approximately 3% have only one or two favorable outcomes (metabolic score ≤ 2). Additionally, appendix Table A4 shows how each variable in our metabolic index predicts future BMI outcomes in our pooled sample. Notably, adolescent BMI has the strongest effect on future BMI, followed by the incidence of diabetes in the biological family.

[11] Refer to (Friedlander et al., 1985; Seshadri et al., 2010; Grotto et al., 2003; Berentzen et al., 2016).

4 Stylized facts

This section presents key life-cycle patterns and relationships between variables used in the analysis. To ensure robustness against socioeconomic and behavioral disparities among nurses, we use three approaches:

1. We present the raw data from our full analytic sample without any controls.
2. We present the data for sub-samples based on marital status, fertility status, spousal education, smoking habits, mental health status, and working shifts.
3. We present the data after residualizing out differences in socioeconomic variables, including martial status, number of children, spousal education, and average pre-tax household income in the census tract of residence.

Examining results by sub-sample or after residualizing out differences serves to minimize the impact of remaining socioeconomic and behavioral disparities among nurses on the stylized facts. However, the facts are remarkably consistent across all approaches. Therefore, here we present our results from our preferred raw data approach (1) and provide additional details and results from approaches (2) and (3) in supplementary material S2 and S3, respectively.

Fact 1: **Average BMI, morbidities, and diet quality increased over adulthood while exercise fell and then recovered.**

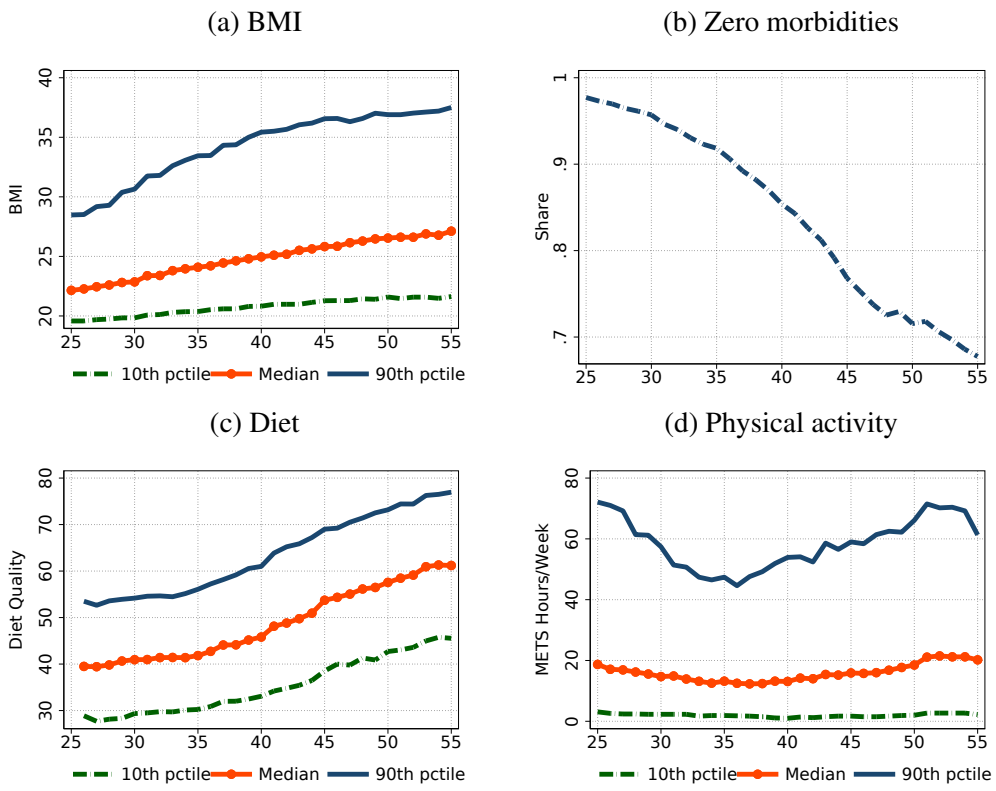
These observed trends are depicted in Figure 5. The first panel illustrates the evolution of BMI, showing the median, 10^{th}, and 90^{th} percentiles of the distribution by age. Each plotted percentile increases over the entire time horizon, indicating a continuous rightward shift of the BMI distribution throughout adulthood. The widening gap between the lines also reflects a substantial increase in the variance of BMI across the sample population. The second panel shows the steady decline in the share of nurses reporting no morbidities over adulthood. This mirrors the observed increase in BMI.

Shifting focus to trends in health behaviors, the third panel of Figure 5 demonstrates a similar rightward shift in diet quality as seen in BMI, suggesting that average diet quality improves over adulthood. Unlike BMI, the variance in diet quality remains relatively stable across the examined ages. The final panel shows life-cycle patterns for exercise. In contrast to BMI and diet quality, average exercise does not change monotonically over the examined ages. Specifically, average exercise decreases until the late thirties and then rises modestly until the early fifties.[12]

Fact 2: **BMI and morbidities diverged significantly among individuals with similarly healthy BMI and no morbidities in early adulthood.**

[12]These patterns qualitatively hold for older cohorts in the data as well (e.g., see appendix Figure A11).

Figure 5: Life-cycle patterns in BMI, morbidities, diet quality, and physical activity



Notes: Panels (a), (c), and (d) plot the distribution (median, 10th, and 90th percentile) of the evolution of BMI, diet quality, and physical activity index, respectively, over the life cycle for the sample of registered nurses in NHS II who were aged 25 to 30 in the initial 1989 wave. For morbidity measure, a total count of morbidities is used. Panel (b) plots the share of those with zero morbidity count by age.

Table 1 reports long-term transition probabilities for BMI categories. Column two reports the share of individuals by BMI category in the first survey wave (1989) and columns three to five report the transition probabilities to BMI category in the final survey wave (2015).[13] Note that 9% of individuals were classified as obese (BMI $\geq$ 30) in the first survey wave. Among this group, over 90% remained obese in the final survey wave, demonstrating exceptionally high obesity persistence over adulthood. In contrast, 75% of respondents in the sample had a healthy BMI ($18.5 - 25$) in the first survey wave. Among this group, 46% maintained a healthy BMI in 2015 while 19% became obese. The final column reports the share of respondents with zero morbidities in the final survey wave. Individuals that experienced weight gain were considerably more likely to experience an onset of morbidities. Among those that started with a healthy BMI in 1989, 77% remained free of

[13]We exclude the 3.3% of respondents with morbidities in 1989 when deriving Table 1 to maintain a comparable sample for the analysis discussed below and illustrated in Figure 6.

morbidities in 2015, compared to less than half, or 47%, of the individuals that were obese during the first survey wave.

Table 1: Long-term BMI transition probabilities

1989 BMI		2015 BMI & morbidities			
Groups	% share	Healthy	Overweight	Obese	Zero morbidities
Healthy	75.15	46.17	35.10	18.74	76.68
Overweight	16.81	6.86	25.61	67.53	60.76
Obese	8.04	1.85	7.75	90.40	46.60

Notes: The first column reports the share of individuals across three BMI groups in the first survey wave (1989): 1) healthy BMI $\in [18.5, 25)$, 2) overweight BMI $\in [25, 30)$ and 3) obese BMI ≥ 30. Columns 3, 4, and 5 provide the transition matrix of BMI groups between 1989 and 2015. Column 6 provides the share of those with zero morbidities in 2015 for each of the 1989 BMI groups. Note that the sample includes only those with zero morbidities in 1989.

For illustrative purposes, we label the 46% that maintained a healthy BMI as healthy weight maintainers, or *stayers*, and the 19% as weight *gainers*. Figure 6 illustrates the differences in BMI evolution and the probability of zero morbidities between these two groups by plotting average outcomes for weight gainers minus average outcomes for stayers. The first panel reveals that weight gainers consistently experienced widening average BMI gaps throughout adulthood, with the difference between the two groups growing by approximately 10 BMI points. The second panel illustrates a divergence in the probability of having zero morbidities between the two groups over the life cycle, with stayers having a roughly 15 pp higher probability of maintaining zero morbidities compared to weight gainers by their fifties. These facts demonstrate that a significant share of individuals that appear ex-ante homogeneous in weight and morbidities in early adulthood experience divergent weight and morbidity accrual as they age.

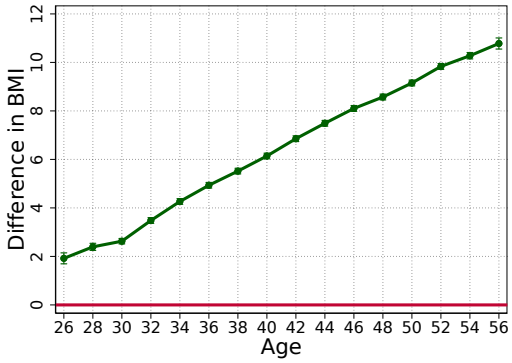
Fact 3: **Healthier diet and exercise behaviors, along with a favorable metabolic type, strongly predict long-run preservation of healthy weight.**

Our next objective is to examine whether patterns of health behaviors are significant predictors of weight change over a span of approximately 30 years. Continuing our illustrative example of healthy weight maintainers compared to weight gainers, Figure 7 shows the evolution of diet quality and exercise for the two groups.[14] Before age thirty, there is a statistically significant but relatively small gap for both health behaviors. However, these gaps begin to widen substantially in the early thirties. By the time individuals reach their fifties, the average diet quality difference is six to eight points. The gap is even more striking for exercise, with stayers approaching double the METs of weight gainers by age

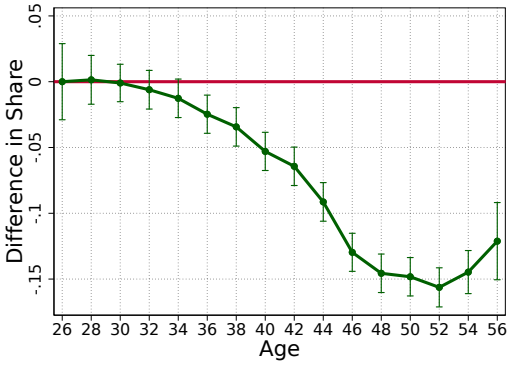
[14]Recall that both groups start with healthy BMI and zero morbidities at the beginning of their adulthood.

Figure 6: Life-cycle health gaps between BMI gainers and stayers

(a) BMI



(b) Zero morbidities

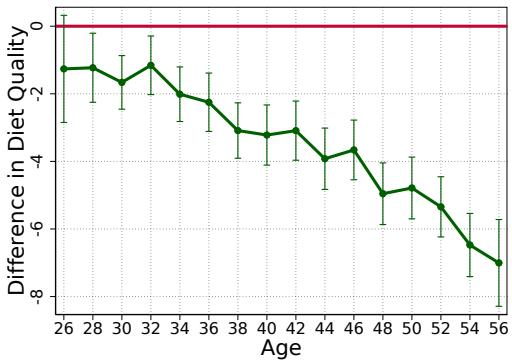


Notes: The figures plot average outcomes for those whose BMI increased from healthy ($18.5-25$) in 1989 to obese ($\geq$ 30) in 2015 minus average outcomes for those who maintained a healthy BMI between 1989 and 2015. Bars denote 95-percent confidence intervals. NHS II respondents aged 25-30 in 1989 are considered, excluding the 3.3% of respondents with non-zero morbidity in 1989.

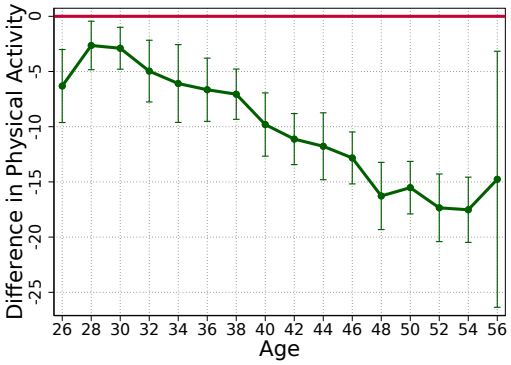
50 (a gap of nearly 20 METs per week between the two groups). This exercise surplus for stayers emerges through a combination of less reduction in exercise to the mid-thirties and a substantially larger increase in exercise thereafter.

Figure 7: Life-cycle behavior gaps between BMI gainers and stayers

(a) Diet quality



(b) Physical activity

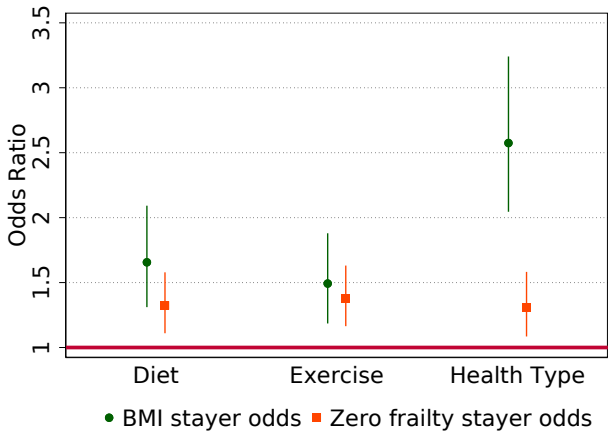


Notes: The figures plot average outcomes for those whose BMI increased from healthy ($18.5-25$) in 1989 to obese ($\geq$ 30) in 2015 minus average outcomes for those who maintained a healthy BMI between 1989 and 2015. Bars denote 95-percent confidence intervals. NHS II respondents aged 25-30 in 1989 are considered.

While health behaviors show comparatively modest gaps in early adulthood between stayers and gainers, early levels of diet and exercise are already strong predictors of long-

run preservation of healthy weight. For example, Figure 8 shows that those in the top exercise or diet deciles at age 30 have 49.2% and 65.6% (respectively) higher odds of belonging to the BMI stayers group compared to those in the bottom deciles. Additionally, the figure shows that individuals with a high (i.e., favorable) metabolic index ($\geq$ 6) had 157% higher odds of being in the stayers group than those with a low metabolic index ($\leq$ 3). This highlights that, in addition to healthy behaviors, initial conditions such as diet habits and exercise preferences established prior to adulthood are strong predictors of weight divergence across individuals in the sample.

Figure 8: Role of initial conditions



Notes: Sample includes NHS II respondents with healthy BMI and zero morbidities in 1989. The figure plots the odds ratio of being a BMI stayer (healthy BMI in 2015) and morbidity stayer (zero morbidities in 2015) for metabolic types (top vs bottom groups), diet (top vs bottom decile) and exercise (top vs bottom decile). Diet and exercise taken from the 1991 wave (roughly age 30). Bars denote 95-percent confidence interval.

It is important to note that this relationship between health behaviors and the long-run maintenance of healthy weight implies that cross-sectional relationships based on contemporaneous correlations can be very misleading. For example, compared to the long-run, the cross-sectional correlation between BMI, diet, and exercise are comparatively weak in our sample of nurses (see appendix Figure A7). Interpreting this as evidence of a limited role for behaviors in sustaining a healthy weight could be misleading, as it may not fully capture the long-term dynamics at play.

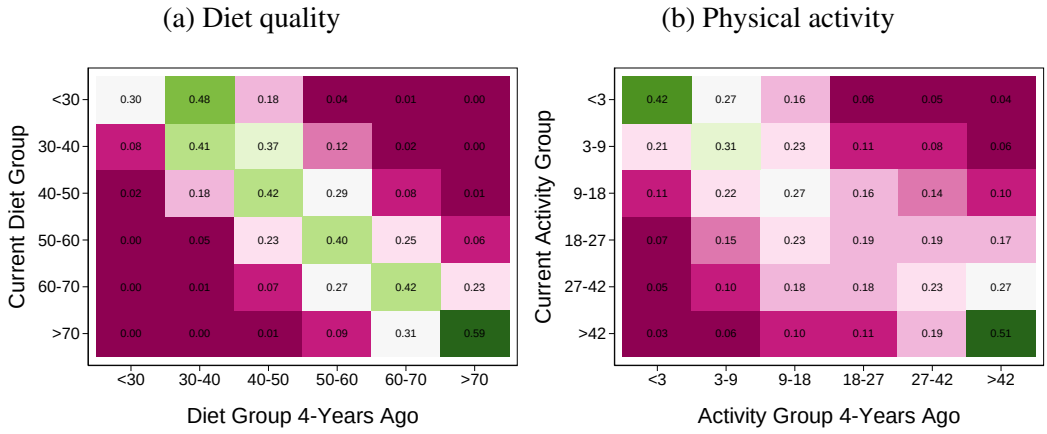
Mirroring the prior analysis contrasting weight maintainers with weight gainers, significant disparities were observed in health behaviors and metabolic types between individuals experiencing morbidity gain (i.e., those with at least one morbidity by 2015) and those maintaining zero morbidities (see appendix Figure A9). For instance, Figure 8 indicates that individuals ranking in the top deciles for diet and exercise at age 30 had 32% and 38% higher odds, respectively, of belonging to the zero morbidity maintainers group compared to those in the bottom deciles. Likewise, individuals with high metabolic index had 31%

higher odds of belonging to the zero morbidity stayers group compared to those with low metabolic index. This underscores that both health behaviors and initial conditions serve as strong predictors for disease development.

Fact 4: **Health behaviors were highly persistent over adulthood, although diet showed less long-run inertia than exercise.**

Figure 9 presents four-year transition probabilities for six discrete diet and physical activity groups to examine persistence over time.[15] The first panel reveals several noteworthy features regarding the persistence in diet choices. First, persistence is observed across the board, from very low levels of diet quality to the healthiest diet groups. For instance, about 40% of nurses with a diet quality between 30-70 maintained their specific diet group four years later. The lowest persistence was noted for the bottom diet group (30%) and the highest for the top group (59%). Second, there is a rightward shift in diet quality at the lower end of the distribution. For example, over half of nurses in the 30 to 40 group reported an improvement in diet quality four years later. Third, reporting a drastic change in diet over a four-year period was highly unlikely. For example, those in the 30 to 40 range had only a 2% chance of reaching a diet score above 60.

Figure 9: Persistence of diet and physical activity



Notes: Panels (a) and (b) plot the 4-year transition probability between different diet and physical activity groups, respectively. NHS II respondents aged 25-30 in 1989 are considered. Pooled data between 1991 and 2015 is used to construct the 4-year transition probabilities.

Persistence was also observed for exercise, though transition probabilities reveal a

[15] While the cut-offs for the six categories for each behavior are chosen somewhat arbitrarily, the top and bottom groups for each behavior are fixed from what is considered very low and high levels of diet and physical activity levels in the medical literature. Recall that the physical activity recommendation by the CDC for adults is 23 MET hours/week, equivalent to 150 minutes of brisk walking with two days of muscle-strengthening activity; thus, the top group with MET hours/week exceeding 42 reflects activity levels similar to those of some athletes.

somewhat different story compared to diet. Specifically, persistence was more concentrated at the tails of the distribution. For example, individuals reporting less than three METs of exercise per week had a 42% chance of remaining below this exercise threshold four years later. They also had only a 15% chance of moving above 18 METs per week (recall CDC guidelines recommend at least 23 METs per week). Conversely, individuals reporting more than 42 METs per week had a 51% chance of maintaining that high level of exercise four years later. Notably weaker evidence of persistence was observed in the middle of the distribution. For instance, an individual reporting 9-18 METs per week had at least a 10% chance of landing in each of the six exercise categories shown in the figure four years later.

The above facts on dietary persistence suggest that habitual consumption patterns may drive the consistent selection of certain types of food, thereby impacting overall diet quality. However, dietary habits may not be entirely rigid, as evidenced by a gradual long-run improvement in diet quality over adulthood. This improvement is indicated by the rightward shift observed in the diet transition probabilities as well as the changing distribution of diet quality over time depicted in Figure 5c. In fact, when looking over a period of twenty years, a majority of individuals improved their diet (see appendix Figure A10) and only 3% of those who started in the lowest diet group stayed there, suggesting that dietary habits can evolve with age and health considerations.

In contrast, exercise demonstrates considerably more long-term inertia. For instance, 47% of individuals who started with the lowest activity levels (< 3 MET hours/week) maintained levels below 9 MET hours/week even two decades later. Additionally, the changes in reported exercise hours over twenty years were noticeably smaller than changes in diet quality (see appendix Figure A10), indicating a relative stagnation in physical activity levels over time. The long-run inertia in physical activity is plausibly influenced less by flexible habits and more by individuals' inherent dispositions towards exercise, ranging from high aversion (*inactive* group) to a preference for it as leisure, resulting in consistent high activity levels (*active* group). This distinction in the persistence of diet and physical activity is important because policy interventions will likely have different effects on behaviors that are somewhat flexible compared to those characterized by permanent preferences.

5 Structural framework

Informed by our empirical findings, we next develop a structural model of endogenous health investments, in terms of health promoting behaviors, to analyze the life-cycle evolution of BMI and morbidities. This model posits that rational, forward-looking individuals face the following key trade-off: investing in costly health-maintaining behaviors now to enjoy the benefits of lower BMI, and therefore reduced morbidity prevalence in old age *versus* higher flow utility today at the expense of poor health later in life.

5.1 Idiosyncratic type and choice variables

The economy is populated by heterogeneous agents whose idiosyncratic type is given by $(j, b, h, q_{-1}, \xi, \varepsilon_q, \varepsilon_e, \alpha)$, where j is age, b is BMI, h is a count of morbidities, q_{-1} is last period's diet quality choice, ξ is a fixed exercise type, ε_q is an i.i.d. preference shock to diet quality, ε_e is an i.i.d. preference shock to exercise intensity, and α is the agent's metabolic type. We focus on the life cycle of agents from when they enter the workforce until they retire. Agents trade-off the benefits associated with a lower terminal (i.e., end-of-working-life) BMI with the disutility of leading a healthy life. In each period, they choose how much to exercise, e, and the quality of their food consumption, q; exercising more (higher e) or consuming a healthier diet (higher q) reduces the likelihood of experiencing an increase in BMI or in morbidities, both of which evolve stochastically over the life cycle. Following the findings in Section 4, we let the transition probabilities for BMI vary with the agent's current BMI and metabolic type, $\mathbb{P}^b\left(b'|b, \alpha, e, q\right)$, and let the transition probabilities for morbidities vary with the agent's current age, BMI, morbidities, and metabolic type, $\mathbb{P}^h\left(h'|j, b, h, \alpha, e, q\right)$.

The disutility of exercising depends on the agent's idiosyncratic type, $f\left(e|b, h, \xi, \varepsilon_e\right)$ (refer to Section 7.3 for further discussions regarding our modeling choices). First, we assume that the disutility of exercising is increasing in both BMI and morbidities. This is necessary to account for the negative cross-sectional relationship in the data between BMI and physical activity and between morbidities and physical activity. Next, we let the disutility vary with the agent's permanent exercise type to account for the high persistence at both the bottom and top of the exercise distribution documented in Figure 9. Lastly, we let the disutility vary with the realization of the agent's i.i.d. exercise preference shock. These sudden positive or negative "motivation shocks" enable us to account for variations in physical activity observed in the data that cannot be attributed to observable factors such as BMI or morbidities.

The agent's idiosyncratic type similarly affects the disutility of healthy eating, $r\left(q|b, q_{-1}, \varepsilon_q\right)$. As for exercising, we assume that the disutility of healthy eating is increasing in BMI. As discussed below in Section 6.2, this is necessary to match the observation that BMI and diet quality are nearly uncorrelated in the cross-section; in contrast, models without such BMI-specific disutility generate robust positive correlations because the return to healthy eating is higher for high-BMI agents in the model. Second, we allow for habit formation to account for the persistence in diet choices illustrated in Figure 9 by assuming that agents incur a utility loss from deviating from their past diet quality choice. Lastly, similarly to exercising, we let the disutility vary with the realization of the agent's diet quality preference shock to account for variations in diet quality observed in the data that cannot be attributed to observable factors.

5.2 Value function

The value function of an agent with idiosyncratic type $(j, b, h, q_{-1}, \xi, \varepsilon_q, \varepsilon_e, \alpha)$ who discounts the future at rate β is given by

$$\begin{aligned} V_j^{\alpha,\xi}(b, h, q_{-1}, \varepsilon_q, \varepsilon_e) &= \max_{e\geq 0, q\geq 0} -f(e|b, h, \xi, \varepsilon_e) - r(q|b, q_{-1}, \varepsilon_q) \\ &+\beta \mathbb{E}\left[b', h', \varepsilon_q', \varepsilon_e', |j, b, h, \alpha, e, q\right] V_{j+1}^{\alpha,\xi}\left(b', h', q, \varepsilon_q', \varepsilon_e'\right). \end{aligned} \tag{1}$$

Agents enter the model at age $j = 1$ and retire at age $j = J$. Age-J agents solve the same problem as described in Equation (1) but with a continuation value that depends on the BMI-specific terminal condition, $V_{J+1}(b)$.

Before we turn to the calibration section, we briefly discuss three modeling choices that we have made.

REMARK 1: We assume that the terminal condition depends on BMI but not on morbidities. We do so because the data-implied law of motion for morbidities depends on BMI, $\mathbb{P}(h'|j, b, h, \alpha, e, q)$, whereas the opposite is not the case, $\mathbb{P}(b'|b, \alpha, e, q)$. Therefore, a model that matches the evolution of BMI, exercise, and diet quality over the life cycle will also match the evolution of morbidities even if morbidities do not affect the terminal condition.

REMARK 2: We have solved alternative specifications of the model where flow utility depends on BMI or morbidities. Because the model specification without these flow utility terms is able to account for the targeted and non-targeted moments discussed in Section 7.1, we have opted for the simpler specification to reduce the number of parameters.

REMARK 3: Because the variance of BMI *within* socioeconomically homogeneous groups, like the NHS II sample used to calibrate our model, is considerably larger than the variance *across* socioeconomic groups, we have intentionally abstracted from certain features that are common in the macro-health literature—such as consumption, income uncertainty, wealth, educational attainment, and health insurance coverage—as these features cannot account for the variance of BMI for our data sample. Instead, we focus on the effects of behavior, metabolic types, dietary habits, and differential preferences towards physical exercise in explaining BMI and morbidity trajectories during adulthood.

6 Calibration

We parameterize the model in two stages. In the first stage, we choose functional forms for the various model components and assign values to parameters that can be estimated without solving the model. In the second stage, we calibrate the remaining parameters by minimizing the distance between target moments calculated using data and their model counterparts.

6.1 First-stage calibration and estimation

6.1.1 Life-cycle parameters and grids

A period in the model is six years.[16] Agents enter the model at age 28 and exit the model at age $J = 7$, which corresponds to a retirement age of 64. Following the quantitative macroeconomics literature, we choose β to match an annual discount factor of 0.96. We follow the CDC and split BMI into six categories, $b \in \{\underline{b}, \ldots, \overline{b}\}$, where the six categories correspond to a BMI of 18.5–25 (healthy weight), 25–30 (overweight), 30–35 (obese class I), 35–40 (obese class II), 40–50 (obese class III), and 50+.[17] We proxy the agent's morbidities by her count of the number of morbidities that she has ever been diagnosed with, $h \in \{0, \ldots, \overline{h}\}$, where $\overline{h} = 2$.[18] Lastly, we proxy the agent's metabolic type, $\alpha \in \{1, \ldots, \overline{\alpha}\}$, by her metabolic index as illustrated in Figure A8. We allow for $\overline{\alpha} = 3$ metabolic types, where $\alpha = 1$, $\alpha = 2$, and $\alpha = 3$ correspond to individuals with a metabolic index of 0–3, 4–5, and 6–7, respectively. As a robustness check, we also use an alternative strategy where metabolic types are calibrated using unobserved fixed effects from a dynamic panel data model to account for residual unobserved heterogeneity (results from the fixed effects model are discussed in appendix section A5.3).

6.1.2 Health production function

Law of motion for BMI: One of the contributions of our study is to estimate the law of motion for BMI directly from the rich data at our disposal while minimizing assumptions about the underlying structure of the BMI-caloric relationship. By integrating both diet and exercise into our model and allowing for randomness in BMI evolution, we capture the inherent variability and complexity in how different factors interact to influence long-term weight trajectories. This approach not only accommodates potential measurement errors but also reflects the nuanced effects of calorie sources and unobserved variables like Basal Metabolic Rate (BMR) on BMI, thereby providing a more robust and realistic estimation of the BMI law of motion.

Specifically, the evolution of BMI depends on the agent's current BMI, metabolic type, exercise level, and diet quality. We parameterize this stochastic process by means of an ordered logistic regression. The transition probabilities for each BMI-category $i \in \{\underline{b}, \ldots, \overline{b}\}$

[16] The length of each model period is chosen to coincide with the estimation of BMI and morbidity transition probabilities in the data. While BMI and morbidities are reported bi-annually in the survey, we estimate six-year transition probabilities for these variables because effort choices (diet and exercise) are reported every four years. As in Cole et al. (2019), long time periods also help ensure that efforts choices have robust effect on BMI and morbidity transitions.

[17] The CDC classifies individuals as underweight if their BMI is less than 18.5. We choose a lower BMI-bound of 18.5 in the model because only 1 percent of individuals are underweight in both the NHS II and NHANES datasets (see Figure 4).

[18] We top-code morbidities at 2 because less than 0.3 percent of our sample have more than 2 morbidities (see Figure A6).

are then given by the ordered logistic formula:

$$\mathbb{P}^b\left(b' = i|\boldsymbol{X}\right) = \frac{1}{1+\exp\left(-\kappa_i + \boldsymbol{X}\boldsymbol{\beta}\right)} - \frac{1}{1+\exp\left(-\kappa_{i-1} + \boldsymbol{X}\boldsymbol{\beta}\right)}, \tag{2}$$

where $\boldsymbol{X} = (b, \alpha, e, q)$, $\boldsymbol{\beta}$ is a vector of parameters, and the κ's are cutoffs for the ordered outcomes.

Appendix Figure A13 illustrates the impact of health behaviors on selected transition probabilities, highlighting the significance of diet and exercise in the BMI law of motion (see appendix Table A6 for the complete regression results). For instance, increasing diet quality from a score of 20-40 to 60-80 raises the probability of transitioning from overweight to a healthy BMI from just under 12% to over 16%. Similarly, increasing exercise from less than 3 MET-hours/week to 20-40 MET-hours/week raises this probability from about 11% to over 14%. Improving diet quality from a score of 20-40 to 60-80 reduces the probability of transitioning from overweight to obese from approximately 27% to 20%. Likewise, increasing exercise from less than 3 MET-hours/week to 20-40 MET-hours/week reduces this probability from about 28% to 23%. Overall, this shows that the short-run marginal effects of healthy behaviors are significant, yet relatively small. However, as shown in later counterfactual experiments, maintaining sustained healthy behaviors can have a substantial impact over the long-run.

Law of motion for morbidities: An individual's morbidity count is non-decreasing, as it reflects the total number of morbidities ever diagnosed. For over 99% of the observations in our sample, the morbidity count increases by increments of one.[19] Consequently, we model morbidity transition probabilities as the likelihood that the morbidity count remains constant or increases by one between two periods. This stochastic process is parameterized using a logistic regression. The likelihood of experiencing a morbidity increase is then given by the logistic formula:

$$\mathbb{P}^h\left(h' = h+1|\boldsymbol{X}\right) = \frac{\exp\left(\boldsymbol{X}\boldsymbol{\beta}\right)}{1+\exp\left(\boldsymbol{X}\boldsymbol{\beta}\right)}, \tag{3}$$

where $\boldsymbol{X} = (j, b, h, \alpha, e, q)$ and $\boldsymbol{\beta}$ is a vector of parameters. Recall that we assume that current BMI impacts the future evolution of morbidities, but that the latter does not impact the BMI law of motion. This choice is driven by empirical observations where we find a strong and statistically significant impact of current BMI on future morbidities but not vice versa (see appendix Table A7).

Appendix Figure A12 illustrates the significant impact of BMI on morbidity transitions as suggested by the law of motion (see appendix Table A8 for the complete regression results). For example, an increase in BMI from a healthy range (18-25) to an obese range (30-35) more than doubles the probability of developing an additional morbidity between

[19] We limit our estimation to cases where the morbidity count increases by at most one.

periods, from around 5% to over 10%.

6.2 Second-stage: simulated moment matching strategy

In the second stage, we use the parameters from the first stage and the joint initial distribution of diet quality, morbidities, BMI, metabolic index, and exercise group from the data to simulate the model. Appendix Table A10 provides the empirical joint distribution of the initial model states drawn from the data. Note that the data exhibits important correlations in these initial conditions. For example, individuals in the highest metabolic group have a 26 pp greater proportion of members within the healthy BMI category compared to those in the lowest group, while the corresponding figure for the highest versus lowest exercise type is 14.5 pp. The remaining parameters are then chosen to minimize the distance between model moments and their data counterparts. While these parameters are chosen jointly through the minimization procedure, each parameter is mostly informed by a specific targeted moment. A summary of the calibrated parameters, along with targets, data moments, and model moments, is given in Table 2.

Table 2: Calibrated parameters

Parameter	Value	Target	Data	Model
ψ_1	0.68	P90 exercise	54.5	55.1
ψ_2	3.51	Average exercise	23.5	25.3
λ_b	0.57	Exercise ratio (BMI < 25 vs. BMI ≥ 30)	1.66	1.68
λ_h	0.18	Exercise ratio ($h = 0$ vs. $h \geq 1$)	1.15	1.15
τ_1	0.66	P90 diet healthiness	64.1	63.1
τ_2	1.35	Average diet healthiness	47.2	48.4
υ_b	0.55	Diet ratio (BMI <25 vs. BMI ≥ 30)	1.00	1.02
υ_q	7.36	P90$-$P10 absolute gap in diet change ($\lvert q - q_{-1} \rvert$)	25.0	26.1
κ_b	0.77	Share of 58 year-olds with BMI < 25	31.2%	32.7%
σ_e	0.67	P10 exercise	1.9	1.6
σ_q	0.70	P10 diet healthiness	32.1	32.4
$\overline{\xi}$	4.81	Exercise persistence (prob. remain above 42 METs)	42.3%	39.9%
$\underline{\xi}$	0.27	Exercise persistence (prob. remain below 3 METs)	50.6%	51.7%

6.2.1 Disutility from exercising

We normalize exercise intensity, e, from 0 to 1, where a value of 0 corresponds to no exercising (equivalent to 0 MET-hours per week) and a value of 1 corresponds to 100+

MET-hours per week.[20] Following (Cole et al., 2019), the disutility of exercising is given by:

$$\begin{aligned} f\left(e|b,h,\xi,\varepsilon_e\right) &= p^e\left(b,h,\xi,\varepsilon_e\right)\left[\tfrac{1}{1-e}-(1+e)\right]^{\psi_1} \\ p^e\left(b,h,\xi,\varepsilon_e\right) &= \psi_2\xi\exp\left(\varepsilon_e\right)\left(b-\underline{b}+1\right)^{\lambda_b}\left(h+1\right)^{\lambda_h}, \end{aligned} \tag{4}$$

where ψ_1 governs the elasticity of the disutility function with respect to exercise, ψ_2 governs the average disutility, and $p^e\left(b,h,\xi,\varepsilon_e\right)$ determines the BMI-, morbidity-, exercise type-, and taste shock-specific slope of the function.

Because the first two parameters, ψ_1 and ψ_2, determine the average slope and the curvature of the disutility function, they can be identified by means of moments of the exercise distribution. We calibrate the elasticity parameter, ψ_1, to match the 90$^{\text{th}}$ percentile of the cross-sectional exercise distribution and calibrate ψ_2 to match the corresponding average exercise intensity.[21] The disutility of exercising must be increasing in BMI to account for the negative cross-sectional relationship in the data between BMI and physical activity; in the absence of such BMI-specific disutility, higher BMI would instead be associated with higher levels of physical activity because the return to exercising is higher for high-BMI agents in the model due to the BMI-specific terminal condition. Analogously, the disutility of exercising must be increasing in morbidities to match the negative cross-sectional relationship between morbidities and physical activity. We identify the parameters that govern the slope of the exercise disutility function with respect to BMI and morbidities by comparing average exercise levels across BMI groups and across morbidity groups, respectively. Given this, we calibrate the BMI-specific slope parameter, λ_b, to match the 66-percent higher average exercise intensity among healthy weight (BMI < 25) individuals compared to obese (BMI ≥ 30) individuals. Similarly, we calibrate the morbidity-specific slope parameter, λ_h, to match the 15-percent higher average exercise intensity among individuals with zero morbidities compared to those with at least one morbidity.

Section 4 demonstrated that there is considerable persistence at both the bottom and top of the exercise distribution. As illustrated in Figure 9b, 42% of individuals whose exercise intensity is less than 3 METs and 51% of individuals whose exercise intensity exceeds 42 METs remain in the same exercise category four years later; comparatively high persistence within exercise categories is also observed two decades later. We account for this persistence by incorporating three exercise types in the model: high distaste for exercising ($\bar{\xi} > 1$), low distaste for exercising ($\underline{\xi} < 1$), and a middle group whose exercise type is normalized to one ($\xi = 1$). The values for those with a high and low distaste of exercising are calibrated to match the probabilities discussed above of remaining, respectively, below 3 METs and above 42 METs between two model periods. Lastly, the agent's disutility of

[20] We top-code MET-hours per week at 100 due to the long tail of the physical activity distribution illustrated in appendix Figure A4. This top-coding applies to less than 2 percent of the sample.

[21] For consistency with the data sample that we use to estimate BMI and morbidity transition probabilities, we restrict both the data and model sample to individuals that are younger than 52 when calibrating parameters related to the disutility of exercising and healthy eating.

exercising depends on the realization of her i.i.d. preference shock, ε_e. This shock, which is drawn from a normal distribution with zero mean and standard deviation given by σ_e, enables us to account for sudden positive or negative "motivation shocks" that affects willingness to exercise but that cannot be attributed to the realization of other variables in the model. We calibrate the dispersion of this taste shock to match the 10^{th} percentile of the exercise distribution.

6.2.2 Disutility from healthy eating

Diet quality, q, is similarly normalized from 0 to 1, where a value of 0 corresponds to the least healthy diet (equivalent to an AHEI-score of 0) and a value of 1 corresponds to the healthiest diet (equivalent to an AHEI-score of 100). The disutility of healthy eating is given by

$$\begin{aligned} r\left(q|b,q_{-1},\varepsilon_q\right) &= p^q\left(b,\varepsilon_q\right)\left[\tfrac{1}{1-q}-(1+q)\right]^{\tau_1}+\upsilon_q\mathbb{I}_{q>q_{-1}}\left[q-q_{-1}\right] \\ p^q\left(b,\varepsilon_q\right) &= \tau_2\exp\left(\varepsilon\right)\left(b-\underline{b}+1\right)^{\upsilon_b}, \end{aligned} \tag{5}$$

where τ_1 governs the elasticity of the disutility with respect to diet quality, τ_2 determines the average disutility, and $p^q\left(b,\varepsilon_q\right)$ governs the BMI- and taste shock-specific slope of the function.

Similarly to the identification of the ψ_1 and ψ_2 above, τ_1 and τ_2 from Equation (5) can be identified by moments of the diet quality distribution. We calibrate τ_1 to match the 90^{th} percentile of the cross-sectional distribution and τ_2 to match the corresponding average value. The disutility of healthy eating must be increasing in BMI to match the observation that diet quality and BMI are cross-sectionally uncorrelated in the data. This follows because models without such BMI-specific disutility would generate a robust positive, and therefore inconsistent, cross-sectional relationship between BMI and diet quality because the return to healthy eating is higher for high-BMI agents in the model due to the BMI-specific terminal condition.[22] The parameter that governs the slope of the diet quality disutility function with respect to BMI, υ_b, can be identified by comparing average diet quality levels across BMI groups, and we therefore calibrate this parameter to match that average diet quality among healthy weight (BMI < 25) individuals is identical to that of obese (BMI ≥ 30) individuals.

The parameter υ_q governs the importance of habit formation. Consistent with the high degree of persistence in diet choices documented in Figure 9a, we assume that agents incur a utility loss from improving their diet quality relative to their last-period choice, $q >$

[22] Unhealthy foods tend to be more caloric, are absorbed fast, and generate spikes of insulin. Higher-BMI individuals might have stronger incentives to opt for unhealthy foods because the calorie requirement necessary for satiation (in terms of maintaining current weight) increases with BMI and unhealthy foods lead to a quick, albeit transitory, feeling of satiation.

q_{-1}, where the magnitude of the utility loss increases linearly with the size of the diet quality adjustment. Importantly, while agents in the model do not incur a direct utility loss from worsening their diet quality relative to last period's choice, $q < q_{-1}$, agents are forward-looking and thus internalize that eating less healthy this period makes it harder to eat healthy in future periods due to the adjustment cost. Hence, the higher the value of υ_q, the smaller are both the increases and the reductions in diet quality in the model and thus the lower is the absolute dispersion of $|\Delta(q)|$. We therefore calibrate υ_q to match the absolute gap between the 10$^{\text{th}}$ and 90$^{\text{th}}$ percentile of the diet quality change distribution.

Finally, we let the i.i.d. diet quality preference shock be drawn from a mean zero normal distribution with standard deviation given by σ_q. Similarly to our approach for the calibration of the exercise taste shock, we calibrate the dispersion of the diet quality taste shock to match the 10$^{\text{th}}$ percentile of the diet quality distribution.[23]

6.2.3 Terminal condition

Agents in the model trade-off the cost of exerting effort with the benefits associated with a lower terminal-period BMI. We let the terminal condition be given by

$$V_{J+1}(b) = -\kappa_b (b - \underline{b})^2, \tag{6}$$

where the parameter κ_b determines the utility loss associated with reaching retirement age with a high BMI (e.g., due to higher mortality risk). Given that this parameter governs the willingness to lead a healthy life, we calibrate its value to match the 31.2% share of individuals at age 58 with a BMI no greater than 25.

7 Results

7.1 Model validation

Although the theoretical framework discussed above is quite simple, it is able to capture key non-targeted features of the data related to changes in healthy diet and exercise behaviors, along with BMI and morbidities. Below, we discuss these properties of the model in detail.

7.1.1 Cross-sectional distributions

First, we examine how the benchmark model performs in matching the distribution of diet and physical activity. Note that some distributional moments were targeted in the cali-

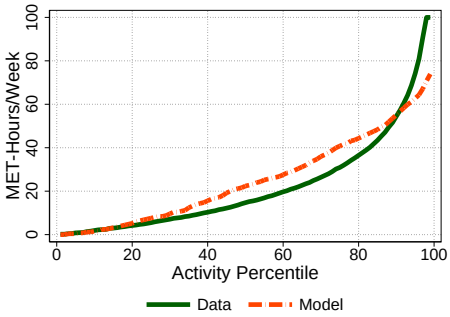
[23] We allow for two independent preference shocks, ε_e and ε_q, to match that diet quality and exercise are only weakly related in the data with a correlation of 0.23. A simplified version of our model with one common taste shock to both exercise and diet quality incorrectly generates a large positive relationship between the two effort variables.

bration, while others were not. Notably, we explicitly targeted the 10th and 90th percentiles as well as the mean for both diet quality and exercise.

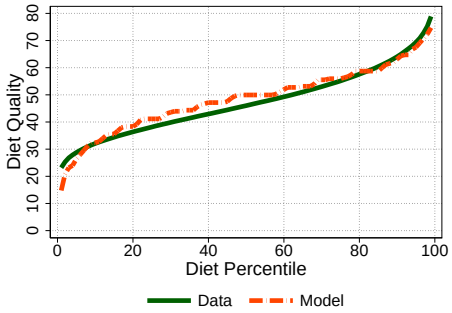
Panel (a) of Figure 10 displays the percentile distribution of physical activity levels for the benchmark model and our analytic sample of registered nurses. Due to the model's incorporation of fixed activity types, it closely aligns with the observed distribution, especially at very low activity levels. While it captures a substantial portion of the higher end of the activity distribution, it is important to note that the model's activity levels are capped at 100. This limits the model's ability to fully account for the upper extremes of the activity distribution observed in the data because the disutility of exercising in the model tends to infinity as exercise approaches 100 METs. Panel (b) illustrates the distribution of diet quality. The model effectively mirrors both the lower and upper extremes of the empirical diet distribution, despite not specifically targeting extreme diet levels in the calibration; for example, the 99th percentile is 76 in the data compared to 79 in the model.

Figure 10: Distribution of diet and physical activity

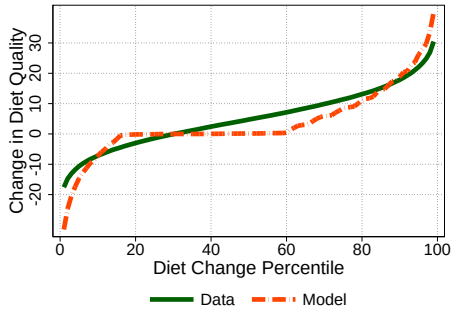
(a) Physical activity



(b) Diet quality



(c) Change in diet quality



Notes: Panels (a), (b) and (c) plot, respectively, the percentile distribution of exercise (e), diet quality (q) and change in diet quality ($q - q_{-1}$) in the benchmark model and the data. We top-code the physical activity METs-hours/weeks to one hundred in the data to make it comparable to the model.

In addition to examining the static distribution of diet quality, observing sequential changes provides valuable insights into the persistence of dietary habits over an individual's

life and the interplay between their current and past dietary choices. As individuals age, there is a growing motivation to improve their diet. In the absence of habitual influences, rational forward-looking individuals might be inclined to make significant dietary changes to improve their late-in-life health outcomes, even if they had previously maintained poor dietary habits. However, this contradicts empirical observations where the magnitude of diet changes over a six-year period is modest, with a median change of 4.7 in our analytic sample. This data characteristic poses a stringent test for the model, challenging it to offer a plausible representation of the evolution of healthy behaviors. Panel (c) of Figure 10 illustrates that our model effectively captures the range of 6-year dietary changes observed in the data, closely resembling both the lower and upper ends of the change distribution. Specifically, the model's mean change at the 10^{th} percentile of diet is -6.5, compared to -7.1 in the data, and at the 90^{th} percentile it is 19.2, compared to 17.8 in the data. While the P10-P90 *gap* in diet change was targeted in the calibration, other moments of the diet change distribution were not targeted. These results indicate a reasonable alignment of our model with observed dietary patterns.

In addition to replicating the distribution of healthy behaviors, the model also matches key patterns in the cross-sectional correlation between behaviors and BMI (see appendix Figure A14). Specifically, the model produces the weak correlation between diet quality and both BMI and physical activity levels observed in the data. Simultaneously, the model matches the stronger negative cross-sectional relationship between BMI and exercise.

7.1.2 Life-cycle evolution

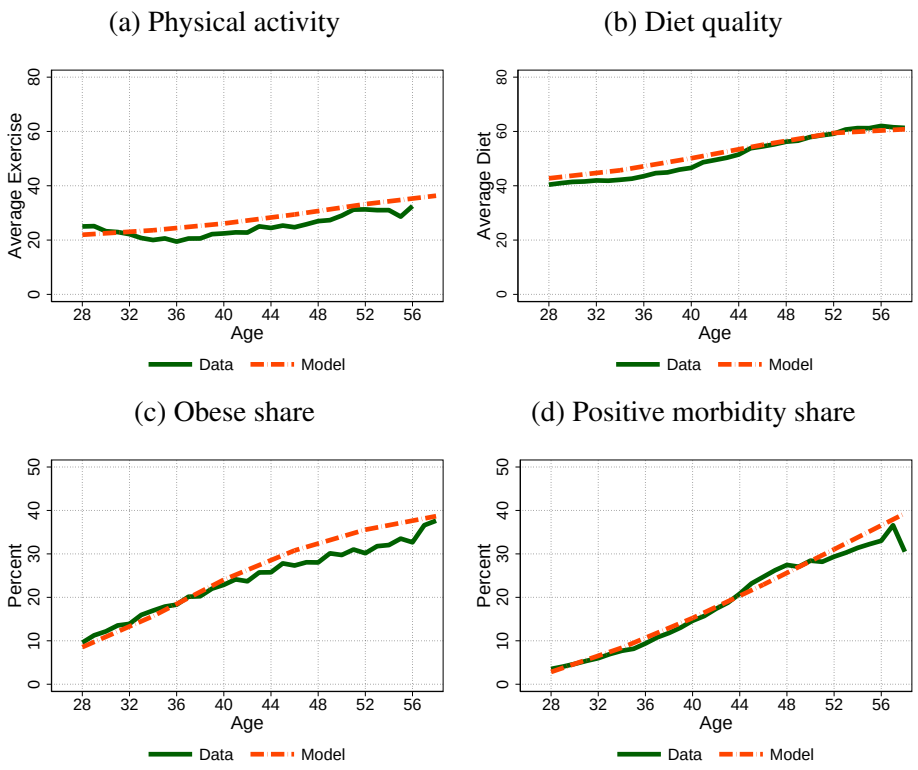
Next, we would like to see whether the model is able to capture the evolution of diet, exercise, morbidities, and BMI over the life cycle. Note that, besides terminal BMI, we do not target any of the age-specific moments in our calibration.

The first two panels of Figure 11 show average diet quality and physical activity levels over the life cycle for both our benchmark model and our analytic sample of registered nurses. The model does reasonably well in matching a key aspect of these behaviors: slow growth in both diet quality and physical activity levels over the life cycle. To reiterate, this is an important aspect of understanding how behaviors evolve. While simpler models without habits or exercise types might allow for a much faster growth in healthy behaviors, especially as individuals approach the terminal period, this is not the case in the data. Incorporating persistence through habits (diet) or permanent types (physical activity) is therefore key to capturing the evolution of healthy behaviors.[24]

Panel (c) of Figure 11 shows the age-specific share of obese individuals in the model and the data (see appendix Figure A15 for a further breakdown). The model tracks the evolution of BMI in the data. This is unsurprising given that (1) the model matches the evolution of healthy behaviors and (2) the BMI law of motion, conditional on current diet

[24]Note that, without additional age-specific factors in the disutility function with respect to exercising, our model cannot reproduce the initial decline in exercise observed in early adulthood. This limitation leads to a slight overestimation of the growth in exercise over the life cycle.

Figure 11: Effort and health over the life cycle: data vs. model



Notes: Figure plots age-specific averages for (a) exercise, (b) diet quality, (c) rate of obesity, and (d) share with at least one morbidity in the benchmark model and the data.

and exercise choices, was estimated directly from the data. Finally, panel (d) shows the age-specific share of individuals with at least one morbidity (see appendix Figure A16 for a further breakdown). Similar to the case of BMI, the model closely tracks the evolution of morbidities over the life cycle.

In addition to matching general life-cycle patterns across outcomes, the model is able to qualitatively capture several other important features observed in the data. For example, the model replicates the morbidity gap between individuals that maintained or gained weight over the life cycle (see appendix Figure A17). The model also produces the BMI and morbidity gaps between high and low metabolic types (see appendix Figure A18) and the high short-run persistence in the tails of the exercise distribution as well as the mildly increasing persistence across the diet quality distribution (see appendix Figure A19).

7.2 Counterfactual experiments

In this section, we conduct counterfactual experiments to understand the role of initial conditions and healthy behaviors in maintaining lower BMI and fewer morbidities over adult life. We start by examining the impact of four specific initial conditions: metabolic type (α), initial BMI (b_0), exercise type (ξ), and initial diet quality (q_0). Then, we explore how diet and exercise, done consistently across adult life, impact health outcomes. Our goal is to understand how weight is shaped by inherent individual characteristics and health-promoting behaviors. These experiments shed light on factors that are more important in determining health outcomes and can provide insights for potential policies to improve public health.

7.2.1 Role of initial conditions

Metabolic type: First, we examine a scenario where all agents in the model are assigned the same value for the metabolic index. Recall that metabolic type can take three values: $\alpha \in \{1, 2, 3\}$. We conduct two experiments, initially assigning all agents the lowest value and then the highest value. It is important to note that this experiment does not alter the outcomes for those who were already in these groups. For instance, approximately 13% of nurses were in the bottom metabolic group and 37% in the top group in our benchmark model.

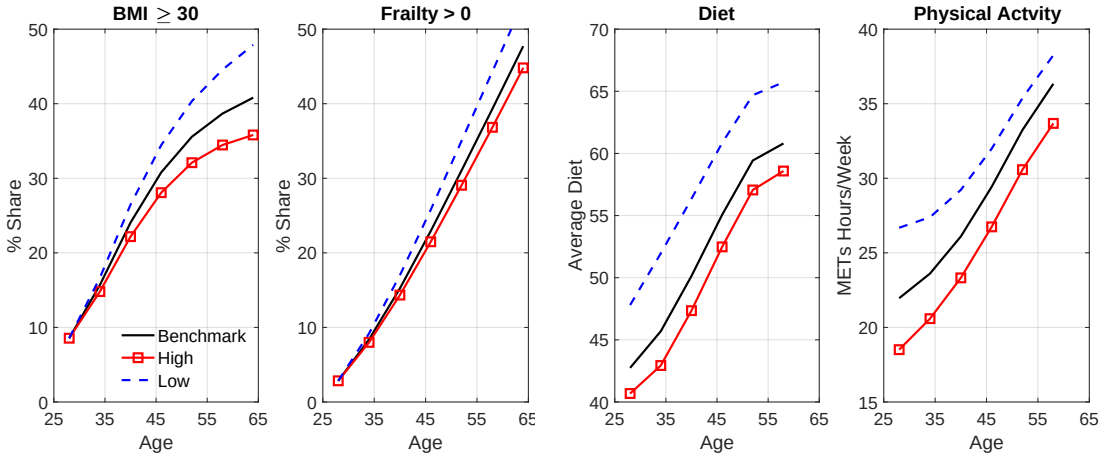
Figure 12 shows age-specific patterns of obesity, morbidities, and health behaviors in the benchmark model alongside the two counterfactual experiments. As shown in the first panel, there is about a 7 pp increase in the share of obese individuals (BMI$\geq$ 30) by age 64 when every agent is assigned the lowest metabolic type. Equalizing metabolism at the highest level leads to the opposite effect, with the share of obese individuals decreasing by 4.9 pp by age 64. Similar effects are observed in the second panel for morbidities. When assigned the highest metabolic type, the share of agents with at least one morbidity decreases by 2.7 pp by age 64 compared to the benchmark model. Meanwhile, when assigned the lowest metabolic type, the share of those with at least one morbidity increases by 6.1 pp at older ages.

To understand these results, it is important to recognize that metabolism plays a key role in the model both through its direct effect on the BMI and morbidity laws of motion (refer to section 6.1) as well as an indirect effect through behaviors. The pace of BMI change, driven by metabolic type, makes it easier or harder for agents to sustain a healthy weight. This is due to the dependence of disutility of exerting effort on BMI. Specifically, the disutility of both higher diet quality and physical activity levels increases with BMI. Additionally, the disutility of exerting effort through physical activity goes up with the number of morbidities. A final amplification effect occurs through the impact of BMI in the morbidity law of motion. Lower future BMI due to higher metabolism also results in fewer morbidities, which in turn makes it easier to maintain good health through behaviors.

However, having a higher metabolism partially distorts incentives to lead a healthy life in the model as forward-looking rational agents internalize that less effort is required to

Figure 12: Life-cycle evolution of health and effort: equalizing metabolism

(a) Health outcomes (b) Effort



Notes: Outcomes plotted for benchmark model and the experiments where metabolism is equalized at lowest value ($\alpha = 1$) and highest value ($\alpha = 3$) for all nurses.

achieve favorable weight transitions in this case. For example, mean exercise and diet quality levels decrease when we assign every agent to the highest metabolic group (see panel (b) of Figure 12). Conversely, assigning agents a lower metabolic group results in compensatory efforts to counter the losses from lower metabolism.

Initial BMI: Next, we consider a case where all agents are assigned the same BMI value in the initial model period (age 28). First, we assign all agents to the lowest BMI group ($b = 1$), representing a BMI within the range of 18.5–25.[25] Second, we assign all agents as class I obese ($b = 3$), corresponding to a BMI of 30–35. Again, this experiment does not change the outcomes for those who were already in these groups in the initial model period. For example, 74% of nurses were initially in the lowest BMI group and 6% in the class I obese group in our sample.

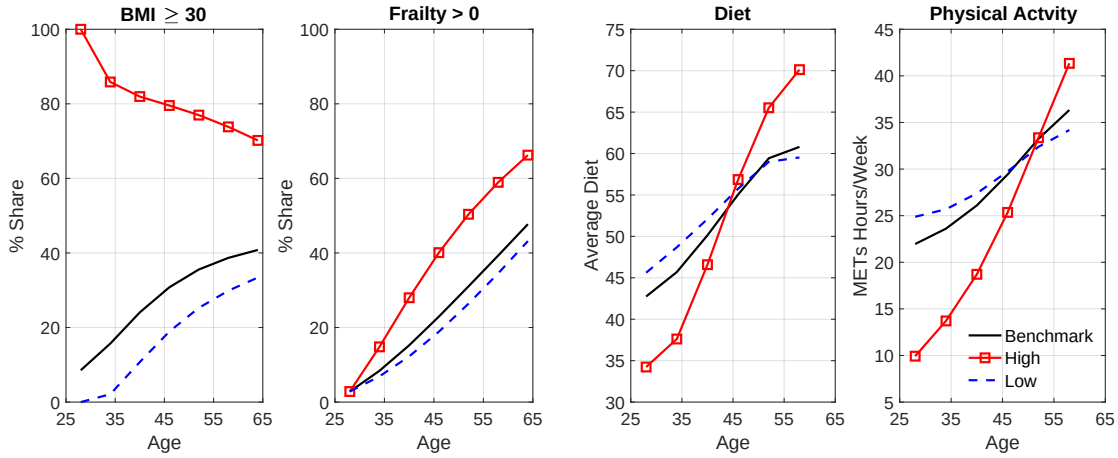
Figure 13 presents age-specific obesity, morbidities, and health behaviors in the benchmark model and the BMI experiments. The first panel shows that the share of obese individuals (BMI$\geq$ 30) decreases by 7-13 pp over the life cycle when every agent is assigned the lowest starting BMI value. Similarly, the share of agents with at least one morbidity decreases by 4.3 pp by age 64. Equalizing BMI at class I obese levels yields the opposite effect, with the obesity share remaining at least 30 pp higher than the benchmark across the life cycle. The share of those with at least one morbidity at age 64 also increases by 18.7 pp. This experiment has particularly important implications for the role played by

[25]Note that in the model, the group $b = 1$ is equal to the mean value of the healthy range (25–30) and corresponds to a BMI of 22.06, whereas the group $b = 3$ is equal to the mean value of the class I obese range (30–35) and corresponds to a BMI of 32.16.

the recent increase in childhood obesity in causing high obesity prevalence among adult Americans (Ogden et al., 2012; Skinner and Skelton, 2014).

Figure 13: Life-cycle evolution of health and effort: equalizing initial BMI

(a) Health outcomes (b) Effort



Notes: Outcomes plotted for benchmark model and the experiments where initial BMI is equalized at lowest value ($b = 1$, or BMI = 22.06) and highest value ($b = 3$, or BMI = 32.16) for all nurses.

Similar to the case of equalizing metabolic types, there are both direct and indirect effects operating through the BMI and morbidity laws of motion, with further amplification through behaviors. Equalizing initial BMI, however, has more substantial effects on the evolution of both BMI and morbidities than metabolism. This is primarily attributed to the stronger impact of lagged BMI in the BMI law of motion compared to metabolism. In terms of behavior, when BMI is equalized at the lowest (healthiest) levels, individuals initially increase effort in both diet and exercise relative to the benchmark model, as exerting effort is relatively less costly when BMI is lower (see panel (b) of Figure 13). However, having enjoyed the benefits of higher effort and a slower rise in obesity and morbidities, they subsequently reduce effort compared to the benchmark model upon reaching older ages. The opposite pattern emerges when all agents are given class I obese levels of BMI, with considerable reductions in effort relative to the benchmark model during the early stages of life, but with higher effort later in life to partially compensate for their worse initial BMI.

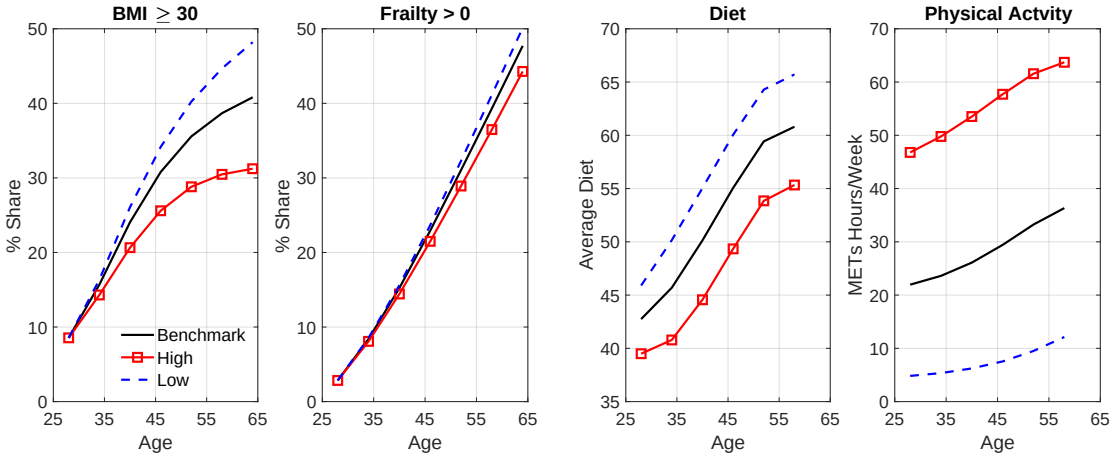
Exercise type: Recall that in the benchmark model, we incorporate permanent heterogeneity through exercise types: agents have either a low ($\xi > 1$), medium ($\xi = 1$), or high ($\xi < 1$) preference for exercise. This permanent type influences the agent's disutility from exercising each period, impacting the amount of effort exerted and, consequently, the evolution of BMI and morbidities. To assess the quantitative role of exercise type heterogeneity in determining weight evolution, we conduct two experiments where we assign all agents either the low or high exercise type. Similar to the above cases, this experiment does

not alter the outcomes for those who were already in these groups—16% of the nurses were in the high exercise type and 17% were in the low type in our benchmark model.

Figure 14 illustrates age-specific patterns of obesity, morbidities, and health behaviors in the benchmark model and the exercise type experiments. When all agents are assigned the high exercise type, the obesity share at age 64 decreases by 9.4 pp, and the share with morbidities experiences a more modest decline of 3.2 pp. Conversely, assigning all agents the low exercise type results in an increase in the obesity share by 7.5 pp and a rise in the share with a morbidity by 2.6 pp.

Figure 14: Life-cycle evolution of health and effort: equalizing exercise types

(a) Health outcomes (b) Effort



Notes: Outcomes plotted for benchmark model and the experiments where exercise type is equalized to low type ($\xi > 1$) and high type ($\xi < 1$) for all nurses.

There are notable differences between this experiment and those involving initial BMI and metabolic types that are worth highlighting. While equalizing exercise types has effects on BMI evolution similar in magnitude to equalizing metabolic types, the impact of the former on morbidities is significantly more subdued. This difference arises because metabolic type directly influences both the morbidity and BMI laws of motion, whereas exercise type has no direct impact on these processes. Even though acquiring a low exercise type leads to a substantial reduction in physical activity over the life cycle (65-77%), agents are able to counteract some of the detrimental effects of reduced physical activity by enhancing their diet quality by 6-8% (see panel (b) of Figure 14). Although this largely mitigates the adverse effects of lower activity levels on morbidity evolution, BMI growth remains higher compared to the benchmark.

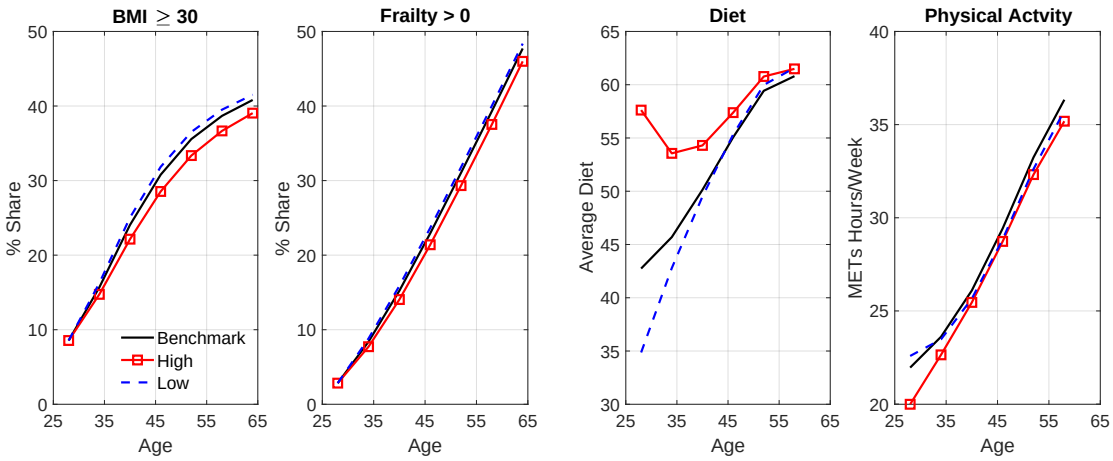
Initial diet quality: Diet persistence in the benchmark framework is modeled as habits, where individuals incur utility losses from improving their diet quality levels relative to the

previous period. This is in addition to the convex disutility costs associated with high levels of diet quality. Consequently, the initial diet quality, drawn from the data at the beginning of the life cycle, may play an important role. To assess its quantitative impact on health evolution, we conduct two experiments where we assign all agents at either low or high initial diet quality levels. Low levels are defined as those observed in the 10^{th} percentile of the pooled diet distribution in our analytic sample, while high diet corresponds to that observed in the 90^{th} percentile.

Figure 15 displays age-specific obesity, morbidity, and health behaviors in the benchmark model and the diet quality experiments. In sharp contrast to earlier experiments, equalizing initial diet at either high or low levels does not significantly impact the evolution of BMI, morbidities, or exercise. This is partly due to diet quality recovering towards benchmark levels relatively quickly. For example, agents starting with poor habits are willing to incur some early utility losses to improve diet quality and future BMI and morbidity transitions. This underscores that initial diet habits, while important, are somewhat malleable, and agents can offset most of the effects of poor diet by exerting additional effort early on.

Figure 15: Life-cycle evolution of health and effort: equalizing initial diet habits

(a) Health outcomes (b) Effort



Notes: Outcomes plotted for benchmark model and the experiments where initial diet quality is equalized at the 10^{th} (low) and 90^{th} (high) percentile of the pooled diet distribution in our analytic sample of nurses.

All initial conditions: Finally, we would like to gauge the overall importance of initial conditions in determining population health outcomes by equalizing all initial conditions explored above to either *poor* or *good* levels. For the former, we set all agents' starting BMI to class I obese levels ($b = 3$, or BMI = 32.16), one morbidity ($h = 1$), low metabolism ($\alpha = 1$), 10^{th} percentile for diet habits, and low exercise types ($\xi > 1$). For the latter, we set all agents' starting BMI to healthy weight ($b = 1$, or BMI = 22.06), zero morbidities

$(h = 0)$, 90^{th} percentile for diet habits, high metabolism $(\alpha = 3)$, and high exercise type $(\xi < 1)$. Appendix Figure A21 shows the impact of this for both behaviors and health outcomes. We find that equalizing all initial conditions at *good* levels results in a 21 pp reduction in the share of obese individuals and a 12 pp reduction in the share of agents with at least one morbidity by age 64. Conversely, setting all initial conditions at *poor* values results in a 45 pp increase in the share of obese individuals and a 51 pp increase in the share with at least one morbidity in old-age. Overall, by equalizing all initial conditions to their mean values (zero variance), we find that a third of the variance in BMI of agents in their fifties can be explained by the variance in initial conditions. These experiments highlight a critical finding: course correction through behaviors can only take individuals so far if their starting point is not favorable, especially when it applies to multiple factors. Policies that affect adolescent BMI and diet habits can therefore have large implications for adult weight and health outcomes.

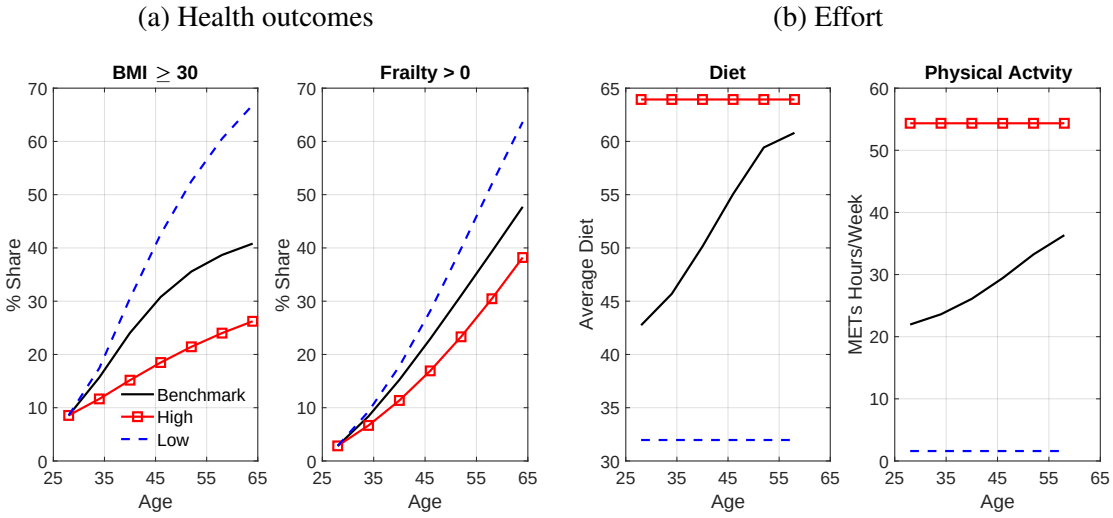
7.2.2 Role of behaviors

The above experiments highlight the significant role of initial conditions in determining both BMI and morbidities over adult life. Next, we aim to understand the importance of behaviors in shaping these outcomes. To achieve this, we conduct two experiments where we first set effort (both diet and exercise) at the 10^{th} percentile of their respective distribution and then at the 90^{th} percentile, across the entire life cycle. These experiments serve two main objectives: first, to understand the potential impact of any policy inducing individuals to exert higher effort on population health and, consequently, medical spending; and second, to disentangle stochastic noise from behavioral channels in determining health outcomes.

Figure 16 shows age-specific obesity, morbidity, and health behaviors in the benchmark model and the experiments where all effort is equalized at low or high values. The first panel indicates that the share of obese agents decreases by 14.4 pp by age 64 when every agent is assigned the 90^{th} percentile of effort. Equalizing effort at the 10^{th} percentile has the opposite effect, with the obesity share increasing by 26.02 pp by age 64. Similar effects on morbidities are observed in the second panel of the figure. When assigned high effort levels, the share of agents with at least one morbidity decreases by age 64 (9.5 pp) compared to the benchmark model. Conversely, when assigned low effort levels, the share of those with at least one morbidity increases by 15.9 pp.

Implications for medical spending: Next, we aim to understand the implications of these experiments for total medical spending in the U.S. Note that our model does not feature health improving expenditures and the evolution of health (BMI and morbidities) is completely governed by three factors: initial conditions, effort (diet and exercise), and stochastic shocks. To derive the medical expenditure implications, we first use data from MEPS to compute the distribution of total medical expenses by age, BMI, and morbidities (see

Figure 16: Life-cycle evolution of health and effort: equalizing effort



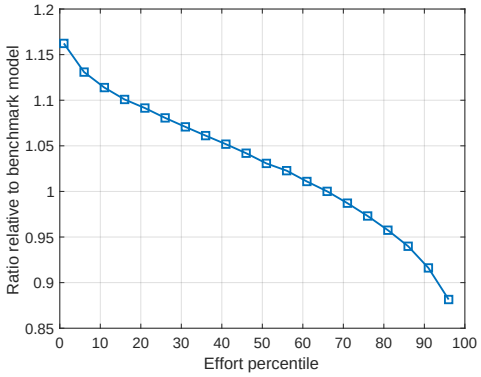
Notes: Outcomes plotted for benchmark model and the experiments where exercise and diet quality are equalized at the 10^{th} (low) and 90^{th} (high) percentile of the respective pooled effort distribution in our analytic sample of nurses.

appendix Section A3 for details.).[26] For each level of effort, we then combine the age-, BMI-, and morbidity-specific medical expenditure distribution with the model-implied joint distribution over age, BMI, and morbidities (which itself depends endogenously on the agents' effort choices over the life cycle) to calculate annual aggregate medical expenses for females aged 25-64.

The results are illustrated in Figure 17, which shows the ratio of total medical expenditures with counterfactual effort levels (both diet and exercise) relative to the benchmark model. In this experiment, we equalize agents' exercise and diet quality at a specific percentile of the respective distribution. For instance, the figure indicates that medical spending increased by 12% compared to the benchmark when both diet and exercise were permanently set at the 10^{th} percentile of their distributions (as in Figure 16). Conversely, spending decreased by 8% relative to the benchmark when exerted effort equalled the 90^{th} percentile of the distribution. To put these differences into perspective, total health spending in the working-age U.S. population surpassed $1.2 trillion in 2022 (MEPS). Therefore, an 8% reduction would translate to approximately $100 billion in aggregate annual savings, a substantial share of which is tax-financed through Medicare or Medicaid in the U.S. This value likely underestimates the scale of potential savings from higher effort because it does not consider any additional savings beyond age 64, where per-person expenditures are even higher; however, the estimate comes with the caveat that it is extrapolated from a specific cohort of registered nurses.

[26] Total medical expenses includes both expenses covered by private and/or public health insurance programs as well as expenses covered out-of-pocket.

Figure 17: Total medical spending relative to benchmark model: equalizing effort



Notes: The graph plots the ratio of medical expenditures in the model with counterfactual effort levels relative to the model with benchmark levels for exercise and diet quality.

7.3 Alternative model specifications

We conducted a series of experiments to assess the impact of excluding fixed exercise types and diet habits in our model. We briefly summarize results here with full details available in Appendices A5.1 and A5.2. Removing fixed exercise types leads to significant overestimation of physical activity levels and fails to capture exercise persistence, especially at the tail ends of the activity distribution. Similarly, eliminating dietary habits only marginally affects life-cycle and cross-sectional diet patterns, but fails to explain *changes* in diet quality over time, particularly underestimating the persistence in diet choices. These findings highlight the essential role of fixed exercise types and habitual dietary behaviors in accurately modeling health behaviors over the life cycle. Additionally, we explored a model with heterogeneous discount rates to account for varying time preferences, finding that while this modification improves some aspects of the model, it still struggles to replicate key features of the exercise data without incorporating exercise types. Specifically, the model overestimates the increase in physical activity over adulthood and continues to miss low-exercise individuals in the bottom third of the distribution.

In our benchmark model, we constructed a metabolic index based on observable early-life characteristics and family disease history, offering advantages such as using hereditary indicators and better external validity. This index is highly predictive of weight changes in later life and avoids potential overfitting issues. However, due to the limited number of indicators, we also explored an alternative approach by estimating unobserved individual fixed effects using a dynamic panel data model. The fixed effects served as proxies for metabolic types, allowing us to re-calibrate the model (see Appendix A5.3 for full details). While both approaches yielded similar estimates for the impact of health behaviors, the fixed effects model attributed a larger role to initial conditions, explaining around 60% of BMI variation in middle age, compared to one third under the benchmark model. These

estimates can be thought of as upper and lower bounds: the lower bound (one third) reflects the role of initial conditions when residual unobserved heterogeneity is not fully captured, while the upper bound (60%) assumes the fixed effects model fully accounts for all relevant variation, including persistent behaviors not explicitly modeled. This framing suggests that the true impact of initial conditions likely falls between these two estimates.

8 Conclusion

In summary, to understand adult body weight dispersion and related morbidities, we analyzed the long-term effects of diet and physical activity on BMI using a comprehensive longitudinal dataset of U.S. nurses. Obesity showed persistence throughout adulthood, but there was notable variation among those who started with a healthy weight. Our findings highlight that diet and exercise are key to maintaining long-term health and that both early-life conditions and lifestyle choices significantly influence health over the life cycle.

Using a simple life-cycle model of endogenous health behaviors, we quantified how various factors impact health outcomes and medical spending in the U.S. Our model highlights the importance of initial conditions and lifestyle choices in lowering BMI and reducing morbidities, which can significantly cut medical costs. For example, improving diet and exercise levels to the top 10% of the population could reduce annual healthcare expenses by at least 8% for women aged 25 to 65. This suggests that early lifestyle interventions could lead to substantial economic benefits by lowering obesity-related healthcare costs.

Our research contributes to a broader understanding of health evolution over the life cycle, emphasizing the interplay between diet, exercise, and inherited health predispositions. By focusing on a socioeconomically homogeneous sample of nurses, we have highlighted that significant health disparities can exist even within similar socioeconomic groups, driven by differences in lifestyle choices and early-life exposures. Our study opens several paths for future research in this direction that include embedding this framework within standard economic channels to achieve more comprehensive insights into health disparities both within and across socioeconomic groups.

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Online Appendix

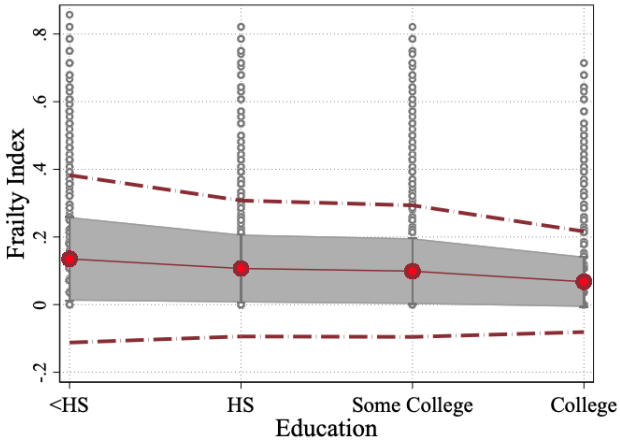


Figure A1: Within and across-group variation in frailty index

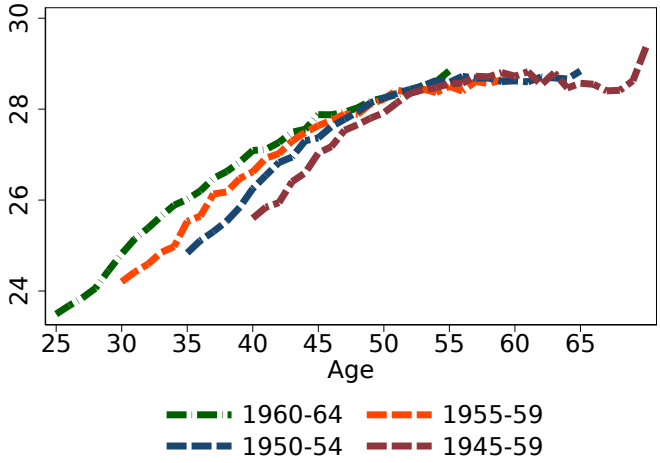
Notes: Data is from the Panel Study of Income Dynamics. The figure plots means, standard deviation (shaded), and two standard deviations (dashed lines) of frailty index (constructed using the methodology developed in Hosseini et al. (2022)) across different education groups. The sample is restricted to individuals between the ages of 25 and 60.

A1 Data and measures

Table A1: Components and score for AHEI-2010 dietary index

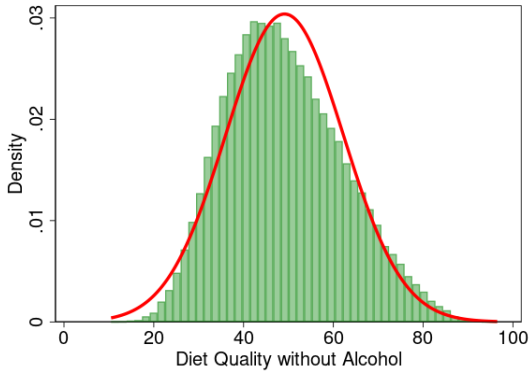
Components	Minimum score 0	Maximum score 10
Vegetables, servings/day	0	≥ 5
Fruit, servings/day	0	≥ 4
Whole grains, grams/day	0	75
Sugar-sweetened beverages and fruit juice, servings/day	≥ 1	0
Nuts and legumes, servings/day	0	≥ 1
Red meat and processed meat, servings/day	≥ 1.5	0
Trans fat, % of energy	≥ 4	≤ 0.5
Long-chain (n-3) fats (EPA + DHA), mg/day	0	250
Poly-unsaturated fatty acids, % of energy	≤ 2	≥ 10
Sodium, mg/day	highest decile	lowest decile
Total	0	110

Figure A2: BMI evolution of different birth cohorts



Notes: The figure plots the BMI-age profile of NHS II survey respondents of different birth cohorts. Pooled data between 1989 and 2015 is used and those with BMI less than 18.5 are dropped from the sample.

Figure A3: Distribution of diet quality index



Notes: The figure plots the distribution of diet quality in our selected sample of the NHS II survey respondents — those aged 25-30 in 1989. Pooled data between 1991 and 2015 is used. Note that even though the survey started in 1989, diet and physical activity variables were only available starting 1991.

Table A2: Categories of responses for the physical activity questionnaire

Categorical response	Median assigned value (hours/week)
1 = zero	0.0
2 = 1-4 minutes/week	0.03
3 = 5-19 minutes/week	0.20
4 = 20-59 minutes/week	0.67
5 = 1 hours/week	1.00
6 = 1-1.5 hour/week	1.25
7 = 2-3 hours/week	2.50
8 = 4-6 hours/week	5.00
9 = 7-10 hours/week	8.50
10 = 11+ hours/week	12.50

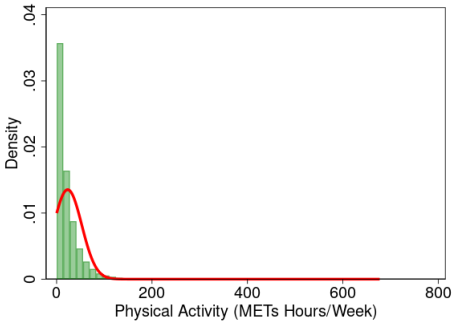
Table A3: Description of activities used in the computation of physical exercise index

Description	Assigned METs
Walking	
Easy pace	2.5
Average pace	3.0
Brisk pace	4.0
Very brisk pace	4.5
Unable to walk	1.0
Missing pace	3.0
Jogging ($\geq$ 10 minutes/mile)	7.0
Running (< 10 minutes/mile)	12.0
Bicycling	7.0
Lap swimming	7.0
Tennis, squash, or racquetball	7.0
Other aerobic exercise calisthenics, aerobics, rowing machine	6.0
Lower intensity exercise (e.g., yoga, stretching)	4.0
Other vigorous activities (e.g., lawn mowing)	6.0
Weight training - arms	4.0
Weight training - legs	4.0
Stair-climbing	8.0

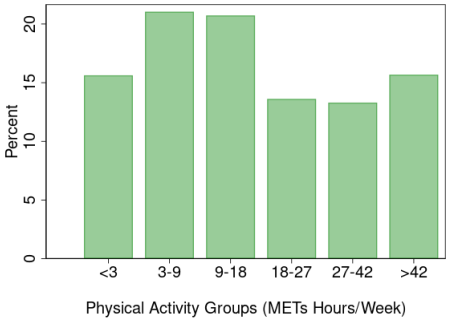
Notes: Activities related to weight training were added to the questionnaire starting 2001. Running and jogging activities were combined together starting 2013.

Figure A4: Distribution of physical activity

(a) Continuous measure



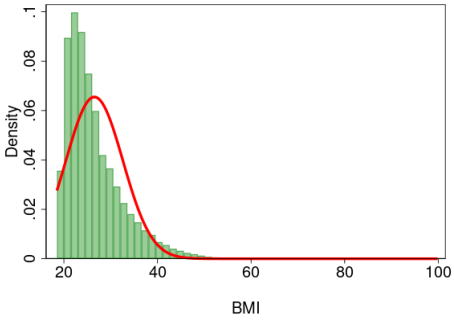
(b) Discrete groups



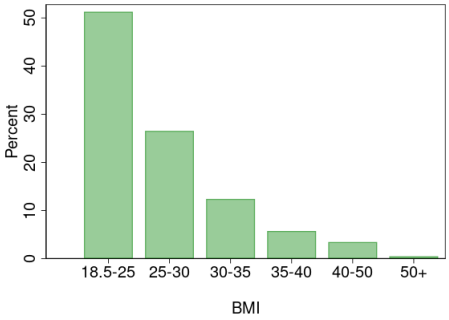
Notes: The figures plot the distribution of physical activity index (in METs hours/week) in our selected sample of the NHS II survey respondents — those aged 25-30 in 1989. Panel (a) plots the continuous distribution and panel (b) plots the distribution of select categories of physical activity. Pooled data between 1991 and 2015 is used. Note that even though the survey started in 1989, diet and physical activity variables were only available starting 1991.

Figure A5: Distribution of BMI

(a) Continuous index

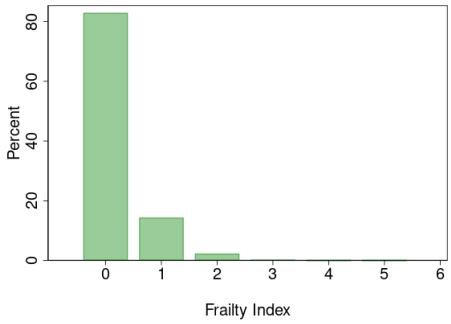


(b) Discrete groups



Notes: The figures plot the distribution of Body Mass Index (BMI) in our selected sample of the NHS II survey respondents — those aged 25-30 in 1989. Panel (a) plots the continuous distribution and panel (b) plots the distribution of select categories of BMI (as defined by the Centers for Disease Control and Prevention). Pooled data between 1989 and 2015 is used.

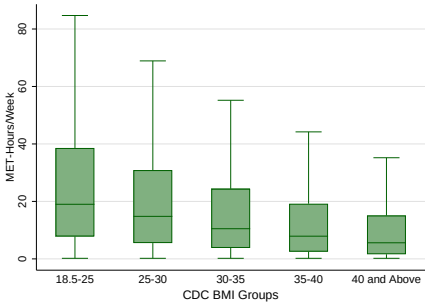
Figure A6: Distribution of morbidity count



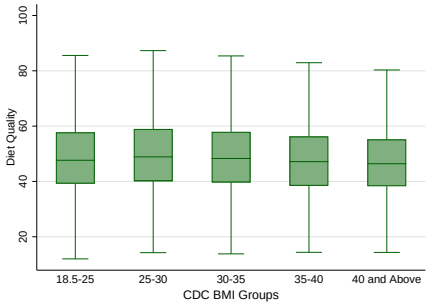
Notes: The figure plots the distribution of morbidity count (measured as a count of chronic conditions) in our selected sample of the NHS II survey respondents—those aged 25-30 in 1989. Pooled data between the years 1989 to 2015 is used.

Figure A7: Joint distribution of BMI, morbidities, diet quality, and physical activity

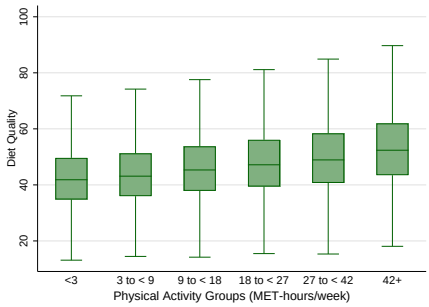
(a) Physical activity and BMI



(b) Diet quality and BMI

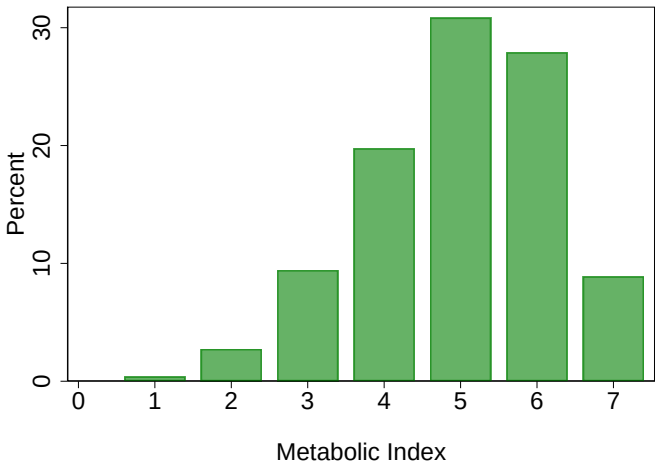


(c) Diet quality and physical activity



Notes: The figures plot joint distributions of a) physical activity and BMI, b) diet quality and BMI, and c) diet quality and physical activity in our selected sample of the NHS II survey respondents — those aged 25-30 in 1989. Pooled data between the years 1991 to 2015 is used. Note that even though the survey started in 1989, diet and physical activity variables were only available starting 1991.

Figure A8: Distribution of metabolic index



Notes: metabolic index is constructed as a total count of 7 indicator variables—(1) full-term gestational length, (2) breast fed in infancy, (3) normal birth weight, (4) healthy age-18 BMI ($[18.5, 25)$), and the absence of (5) diabetes, (6) stroke, and (7) myocardial infarction in the immediate biological family medical history.

Table A4: OLS estimates of metabolic index constituents on BMI

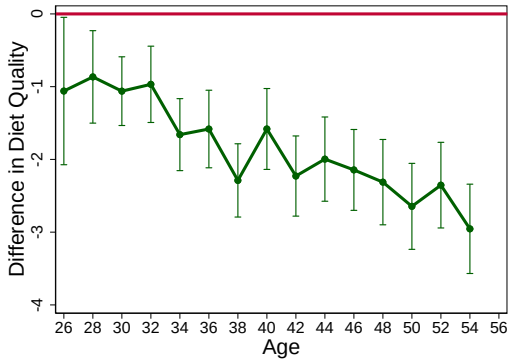
	BMI
Adolescent BMI	
Healthy age 8 BMI: $\mathbb{I}_{i,bmi18}$	-7.609***
	(-169.03)
Early Childhood/Neonatal Conditions	
Breastfed: $\mathbb{I}_{i,breastfed}$	-0.154***
	(-4.81)
Normal birth weight: $\mathbb{I}_{i,BW}$	-0.299***
	(-8.12)
Born full-term: $\mathbb{I}_{i,full-term}$	-0.462***
	(-8.69)
Family Medical History	
No Myocardial Infarction: $\mathbb{I}_{i,MI}$	-0.295***
	(-9.11)
No Stroke: $\mathbb{I}_{i,stroke}$	-0.121**
	(-3.08)
No Diabetes: $\mathbb{I}_{i,diab}$	-1.319***
	(-42.49)
Constant	35.17***
	(467.14)
N	135,176

t statistics in parentheses

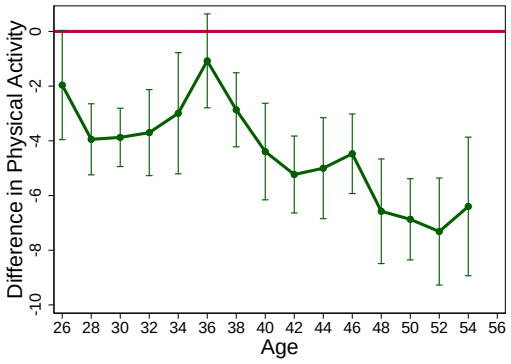
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Figure A9: Life-cycle gaps between morbidity gainers and stayers

(a) Diet quality



(b) Physical activity



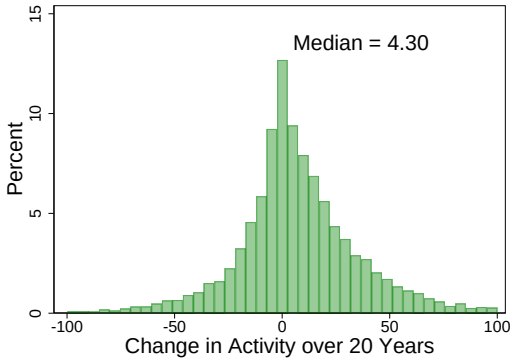
Notes: Panel (a) plots average diet quality for those who obtained at least one morbidity between 1989 and 2015 minus average diet quality for those who maintained a zero morbidity count between 1989 and 2015, conditional on both groups starting with zero morbidities in 1989. Panel (b) plots the corresponding statistic for physical activity. Bars denote 95-percent confidence intervals. NHS II respondents aged 25-30 in 1989 are considered.

Figure A10: Long-run persistence in diet and physical activity

(a) Diet

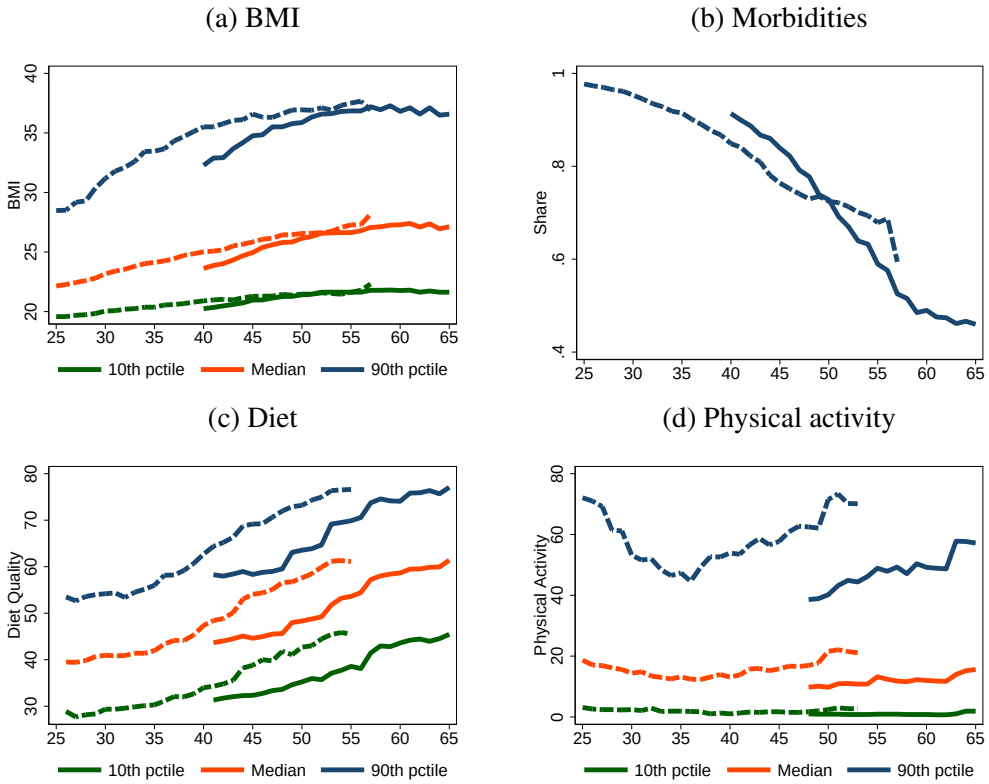


(b) Activity



Notes: Figure plots the distribution of diet and physical activity change over a period of 20 years. NHS II respondents aged 25-30 in 1989 are considered. Pooled data between the years 1991 to 2015 is used. Note that even though the survey started in 1989, diet and physical activity variables were only available starting 1991.

Figure A11: Life-cycle patterns in BMI, morbidities, diet quality, and physical activity by birth cohorts



Notes: Panels (a), (c), and (d) plot evolution (median, 10th and 90th percentile) of BMI, diet quality, and physical activity index, respectively, over the life cycle of registered nurses in NHS II. Solid lines indicate those who were ages 40 to 45 in 1989 and dashed lines are for the benchmark birth cohort (ages 25-30 in 1989). For morbidity measure, a total count of chronic conditions is used. Panel (b) plots the share of those with zero morbidity count over the life cycle.

A2 Sample attrition

Even though the survey has high response rate for each two-year cycle (85 to 90%), sample attrition could be a concern for the analysis, especially if it is systematically driven by those who are less healthy.[27] We find the attrition rates to be relatively low in the survey given that it is a long follow-up period. Specifically, roughly 70% of the registered nurses who were interviewed for the first time in 1989 also completed the survey in 2015. Table A5 shows the distribution of key baseline variables for nurses in our sample that completed a survey in 2015 (non-attrition sample) and those that did not (attrition sample). The attrition sample had a slightly higher, though statistically insignificant, baseline BMI, exercise score, and morbidity count, and a slightly lower diet quality score and metabolic index.

Table A5: Sample attrition

	Non-Attrition Sample			***Attrition Sample***		
	Mean	Std.Dev.	Obs	Mean	Std.Dev.	Obs
BMI in 1989	23.48	4.28	17262	24.11	4.82	6946
Diet Quality in 1991	41.47	9.89	14885	40.68	9.97	4555
Physical Activity in 1991	23.64	29.94	15658	24.69	33.33	4925
Metabolic Index	4.52	1.28	10801	4.32	1.27	506
Morbidity Count in 1989	0.03	0.17	17371	0.04	0.20	7009

Notes: Diet quality and physical activity were first reported in 1991. Attrition is defined as nurses who returned a filled questionnaire in 1989 but did not return their mailed questionnaire in 2015 (last period considered in the analysis).

A3 Medical expenditures

We use data from the Medical Expenditure Panel Survey (MEPS) to estimate age, BMI, and morbidity-specific medical expenditure risk. Following classifications by the CDC, we categorize BMI into six groups: $18.5-25$ (healthy weight), $25-30$ (overweight), $30-35$ (obese class I), $35-40$ (obese class II), $40-50$ (obese class III), and $50+$. Consistent with our construction of the morbidity index for the NHS II sample, we use data on whether the individual has ever been diagnosed with any of the following conditions: high blood pressure, diabetes, cancer, emphysema, angina, coronary heart disease, other heart disease, heart attack, stroke, asthma, arthritis, and high cholesterol. The morbidity index is then given by the sum of these indicator functions, top-coded at 2 for consistency with the NHS II sample as discussed in Section 6.1.

[27] In the survey, every cycle, questionnaires are sent to all surviving cohort members except those who have unforwardable addresses or who have refused further participation in the study.

To capture that the medical expenditure distribution has both a mass at zero and a long right tail, we categorize medical expenses into seven groups based on percentiles of the population distribution in the MEPS: $0-25$th percentile, $25-50$th percentile, $50-75$th percentile, $75-90$th percentile, $90-95$th percentile, $95-99$th percentile, and $99-100$th percentile. The medical spending level corresponding to each expenditure group is given by the weighted average spending level within that group in the MEPS. We then estimate the age, BMI, and morbidity-specific likelihood of drawing one of these seven medical expenditure levels by means of an ordered logistic regression (with the probabilities obtained from the ordered logistic formula), using 6-year age groups for consistency with the model periods and restricting the sample to women. The regression results are reported in Table A9. Finally, we combine these medical expenditure levels and probabilities with the joint distribution of age, BMI, and morbidities in the model, which itself is a function of the agents' specific effort levels (i.e., diet and exercise choices), to compute aggregate medical expenditures for the working-age population under different effort levels.

A4 Additional results

Table A6: Ordered logit estimates of BMI law of motion

	Estimates
BMI in $t-1$	
$b=2$	3.031***
$b=3$	5.385***
$b=4$	7.199***
$b=5$	8.977***
$b=6$	11.93***
Diet Quality Group	-0.209***
Physical Activity Group	-0.142***
Metabolic Group=2	-0.187***
Metabolic Group=3	-0.418***
cut 1	-0.0268
cut 2	2.760***
cut 3	4.986***
cut 4	6.998***
cut 5	10.40***
Observations	31102
Pseudo R^2	0.3667

$^{*}\ p<0.05$, $^{**}\ p<0.01$, $^{***}\ p<0.001$

Table A7: Ordered Logit Estimates of BMI Law of Motion with Morbidities

	Estimates
BMI in $t-1$	
$b=2$	3.027***
$b=3$	5.378***
$b=4$	7.191***
$b=5$	8.974***
$b=6$	11.94***
Morbidity count in $t-1$	
h=1	0.108**
h=2	-0.282**
h=3	0.103
h=4	-1.173
h=5	-0.586
Diet Quality Group	-0.210***
Physical Activity Group	-0.142***
Metabolic Group=2	-0.187***
Metabolic Group=3	-0.418***
cut 1	-0.0251
cut 2	2.763***
cut 3	4.990***
cut 4	7.004***
cut 5	10.42***
Observations	31102
$PseudoR^2$	0.367

$^{*}\ p<0.05$, $^{**}\ p<0.01$, $^{***}\ p<0.001$

Table A8: Logit estimates of morbidity law of motion

	Estimates
Morbidity count=1	0.627
Age	0.0305***
Morbidity count=1 $\times$ Age	-0.0181
Diet Quality Group	-0.176***
Physical Activity Group	-0.0546*
BMI in $t-1$	
b=2	0.545***
b=3	0.983***
b=4	1.342***
b=5	1.482***
b=6	1.902***
Metabolic Group=2	-0.200***
Metabolic Group=3	-0.324***
Constant	-3.174***
Observations	31942
R^2	0.0495

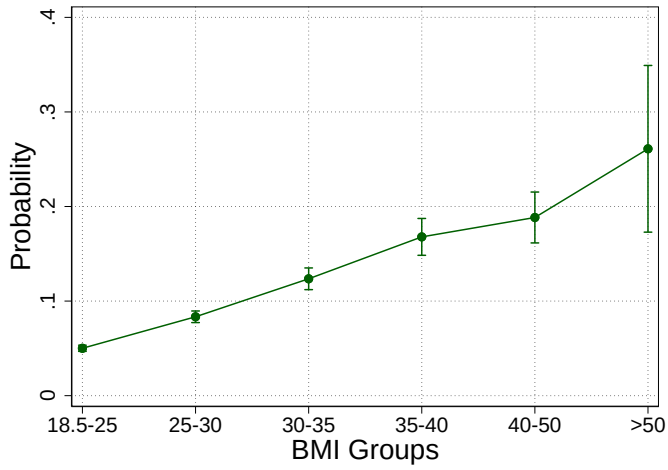
$^{*}\ p < 0.05$, $^{**}\ p < 0.01$, $^{***}\ p < 0.001$

Table A9: Ordered logit estimates of medical expenditure group

	Estimates
Age group	
$j = 2$	0.106**
$j = 3$	-0.000
$j = 4$	0.192***
$j = 5$	0.372***
$j = 6$	0.578***
$j = 7$	0.798***
BMI	
$b = 2$	0.057**
$b = 3$	0.167***
$b = 4$	0.318***
$b = 5$	0.406***
$b = 6$	0.702***
Morbidity count	
$h = 1$	0.803***
$h = 2$	1.563***
cut 1	-1.007
cut 2	0.104***
cut 3	1.384***
cut 4	2.601***
cut 5	3.452***
cut 6	5.346***
Observations	70519
Pseudo R^2	0.0335

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Figure A12: Impact of BMI on six-year morbidity transitions



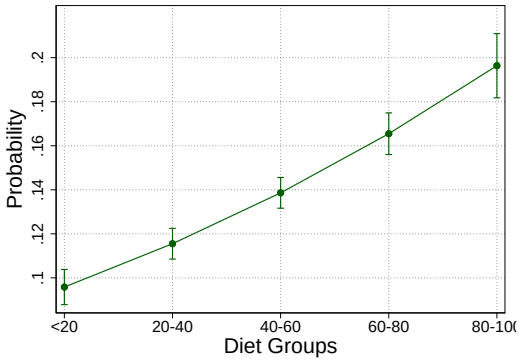
Notes: Figure plots the probability of obtaining an additional morbidity conditional on current BMI category. In all cases, remaining variables included in the morbidity law of motion are held at their overall sample mean. Error bounds display 95-percent confidence intervals.

Table A10: Distribution of subset of initial conditions

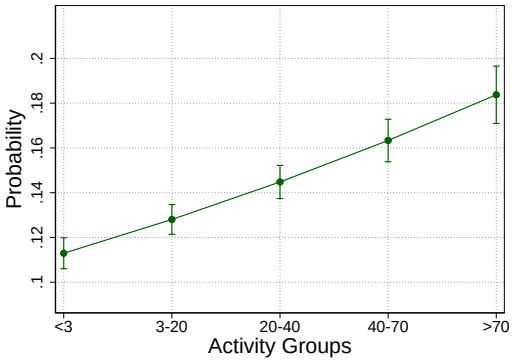
	Uncond. share (%)	Conditional share (%)					
		Metabolic group			Activity group		
		Bottom	Middle	Top	Inactive	Middle	Active
Unconditional share (%)		12.57	50.62	36.81	16.96	67.29	15.75
BMI groups							
18.5-24	74.10	55.50	71.37	81.52	66.54	74.42	81.01
25-29	17.12	24.86	18.81	15.10	19.76	17.26	13.75
30-34	5.64	11.56	6.39	2.63	7.77	5.52	3.76
35-39	2.14	4.91	2.51	0.59	3.50	2.02	1.12
40-49	0.90	2.93	0.84	0.13	2.08	0.73	0.37
50+	0.09	0.24	0.08	0.03	0.35	0.05	0
Morbidity groups							
Zero morbidities	96.7	94.52	97.01	98.26	96.76	96.79	96.88
One morbidity	3.3	5.48	2.99	1.74	3.24	3.21	3.12
Average diet quality	41.28	41.61	41.32	42.19	37.60	41.25	45.42

Figure A13: Impact of healthy behaviors on six-year BMI category transitions

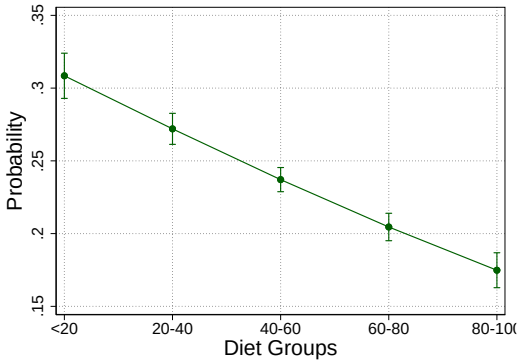
(a) Diet: overweight to healthy BMI



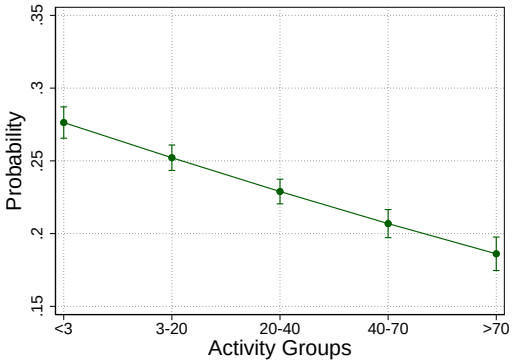
(b) Activity: overweight to healthy BMI



(c) Diet: overweight to obese



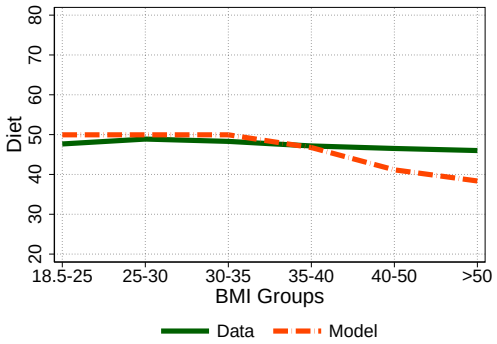
(d) Activity: overweight to obese



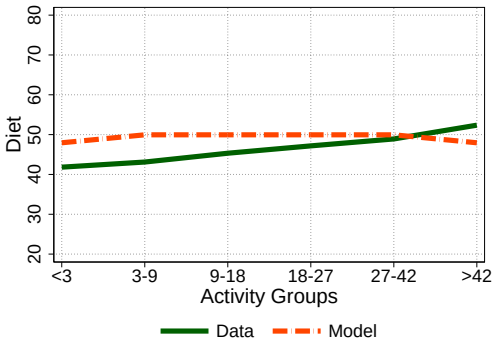
Notes: Figures (a) and (b) plot the six-year probability of transitioning from the overweight to healthy BMI category conditional on different levels of diet quality and physical activity (MET-hours/week) respectively. Figures (c) and (d) plot the probability of transitioning from the overweight to obese BMI category. In all cases, remaining variables included in the BMI law of motion are held at their overall sample mean. Error bounds display 95-percent confidence intervals.

Figure A14: Correlations: Model vs. data

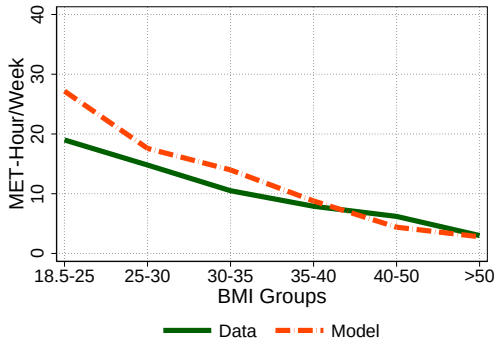
(a) Diet quality and BMI



(b) Diet quality and physical activity



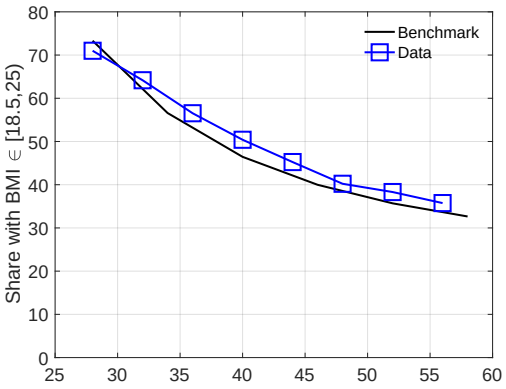
(c) Physical activity and BMI



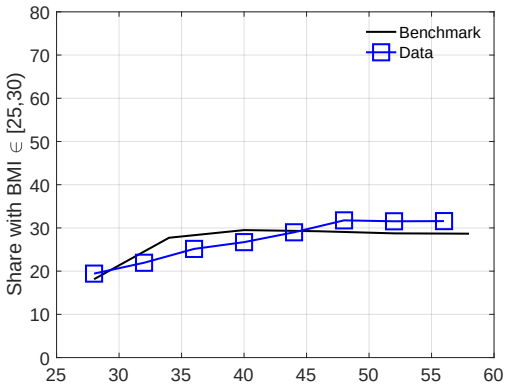
Notes: Panel (a) plots median diet quality for six BMI groups, panel (b) plots median diet quality for six exercise groups, and panel (c) plots median exercise for six BMI groups.

Figure A15: Life-cycle evolution of BMI: Data vs. model

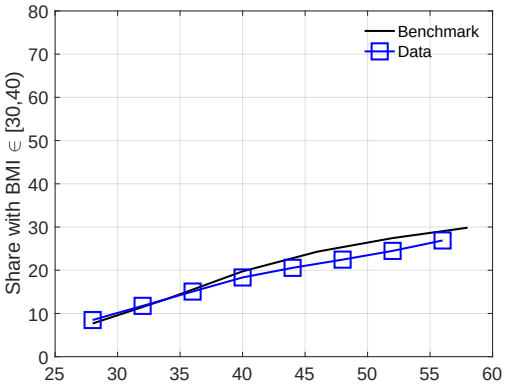
(a) Healthy weight



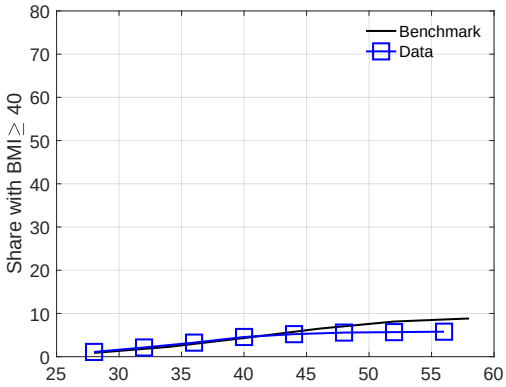
(b) Overweight



(c) Class I & II obese



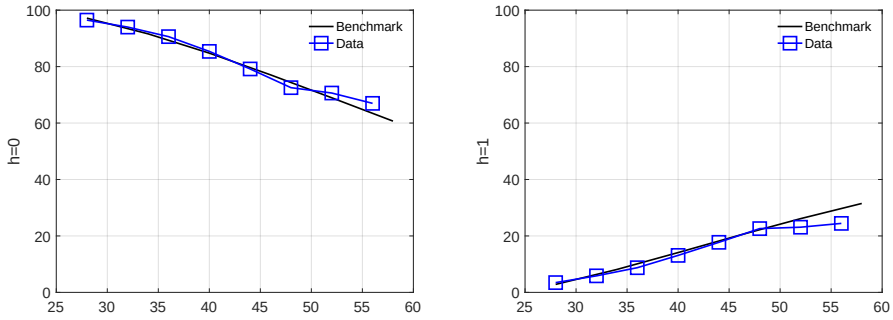
(d) Class III obese and higher



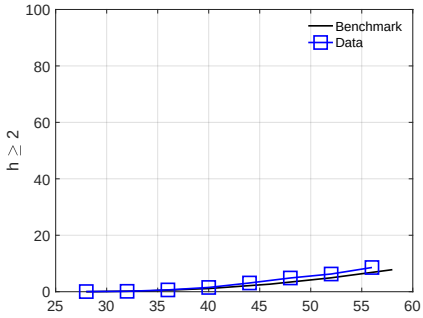
Notes: Panels (a), (b), (c), and (d) plot, respectively, the age-specific share of agents with a BMI between 18.5 and 25.0, between 25.0 and 30.0, between 30.0 and 40.0, and 40.0+ in the benchmark model (black) and the data (blue squares).

Figure A16: Life-cycle evolution of morbidity: Data vs. model

(a) $h = 0$ (b) $h = 1$



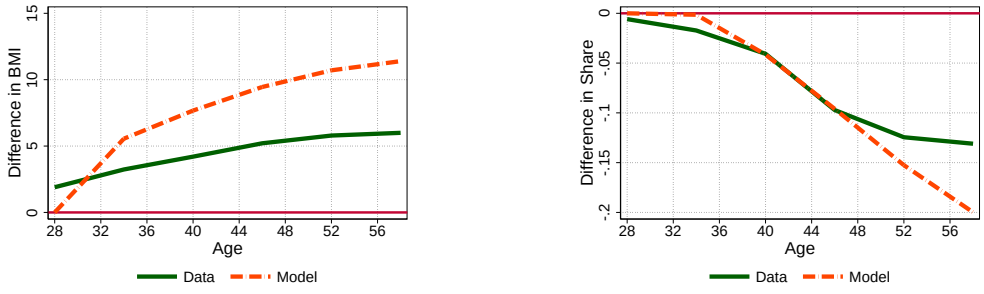
(c) $h \geq 2$



Notes: Panels (a), (b), and (c) plot, respectively, the age-specific share of agents with 0, 1, or 2+ morbidities in the benchmark model (black) and the data (blue squares).

Figure A17: BMI and morbidity divergence: Model vs. data

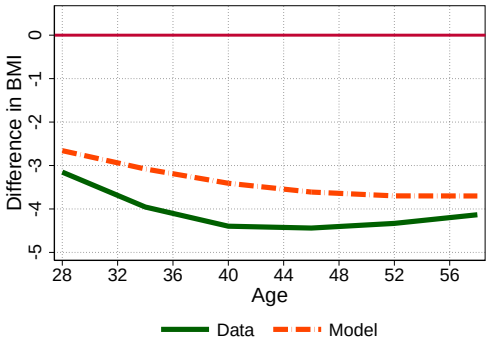
(a) BMI (b) Morbidities



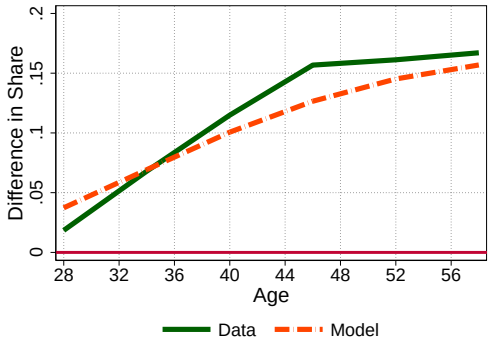
Notes: The figures plot the difference in average outcomes for those whose BMI increased from healthy (18.5-25) in 1989 to unhealthy (>25) at some point between 1989 and 2015 and average outcomes for those who consistently maintained a healthy BMI between 1989 and 2015 in both the data (solid line) and model (dashed line).

Figure A18: Metabolism: Model vs. data

(a) BMI



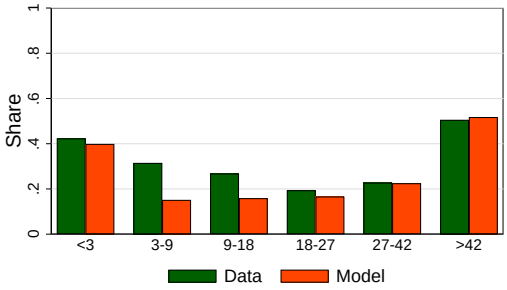
(b) Morbidities



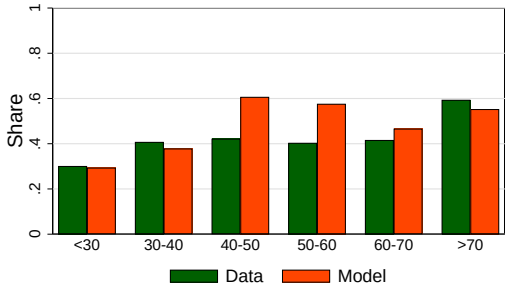
Notes: The figures plot the difference in average outcomes for those with a metabolic index $\geq$ 6 minus average outcomes for those with a metabolic index $\leq$ 3 both in the data (solid lines) and the model (dashed lines).

Figure A19: Persistence in diet and exercise: Model vs. data

(a) Exercise

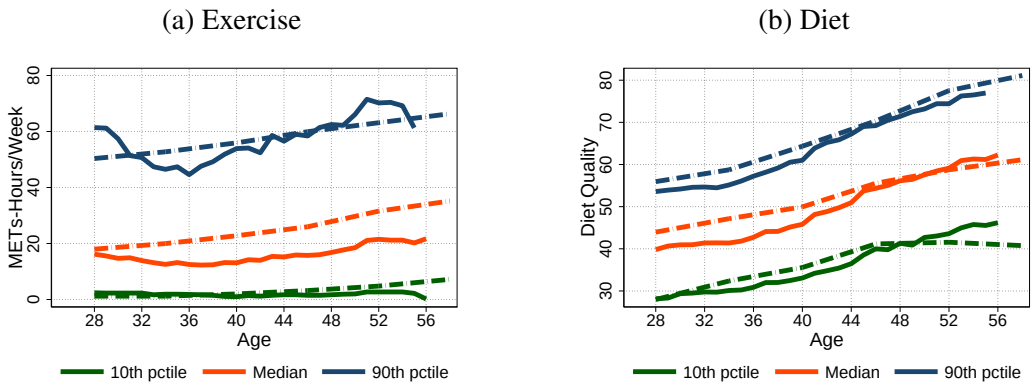


(b) Diet



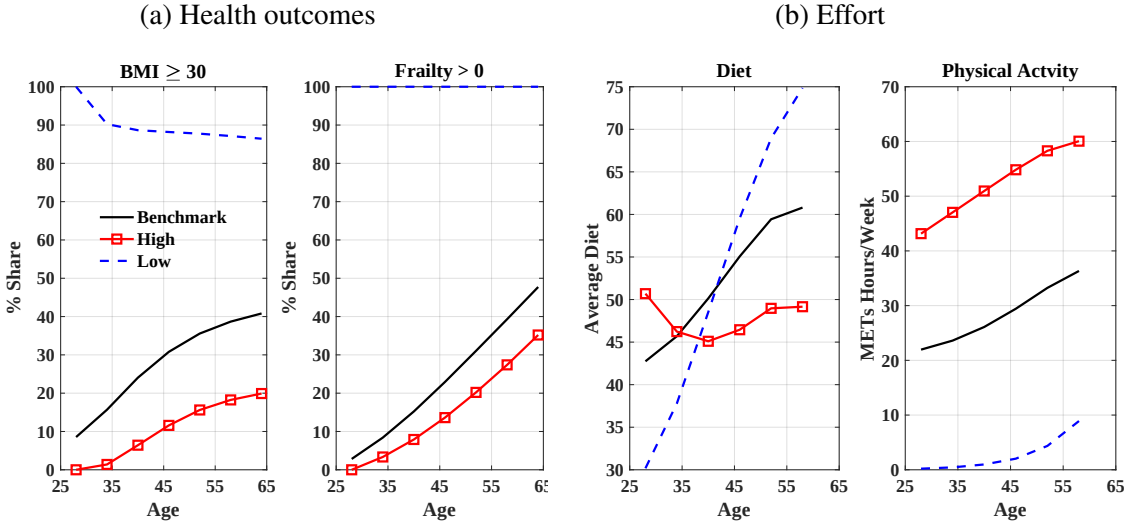
Notes: The figures plot the share of those who maintained their respective exercise and diet groups 4-years later, both in the model and the data.

Figure A20: Diet and exercise age distribution: Model vs. data



Notes: The figures plot the life-cycle evolution of the exercise and diet distribution, both in the model and the data.

Figure A21: Life-cycle evolution of health and effort: Equalizing all initial conditions



Notes: Outcomes plotted for benchmark model and the experiments where all initial conditions are equalized to their poor levels (dashed blue lines) and good levels (solid red lines) for all agents. The poor initial levels correspond to all agents being assigned obese class I ($b = 3$, or BMI = 32.16), one morbidity ($h = 1$), low metabolism ($\alpha = 1$), 10^{th} percentile for diet habits, and low exercise types ($\xi > 1$). For good levels, everyone is assigned healthy weight ($b = 1$, or BMI = 22.06), zero morbidities ($h = 0$), 90^{th} percentile for diet habits, high metabolism ($\alpha = 3$), and high exercise type ($\xi < 1$).

A5 Alternate models

To better understand the importance of some of the modeling choices in accounting for certain aspects of the data, we next simplify some features of our model. In each scenario, we re-calibrate the model to match the same targets (as discussed in Section 6.2) to give the alternate model the best chance of matching the data.

A5.1 Exercise types and diet habits

In this set of experiments, we examine the importance of incorporating fixed exercise types and diet habits into our modeling framework to match the data. Specifically, we start by removing the fixed exercise types in the model by setting $\xi = 1$ for all groups in Equation (4) as follows:

$$f\left(e|b,h,\varepsilon_e\right) \quad = \quad \psi_2 \exp\left(\varepsilon_e\right)\left(b-\underline{b}+1\right)^{\lambda_b}\left(h+1\right)^{\lambda_h}\left[\tfrac{1}{1-e}-\left(1+e\right)\right]^{\psi_1}. \tag{A1}$$

In the second experiment, we instead remove the dependence on previous diet quality by setting $\upsilon_q = 0$ in Equation (5), yielding the following disutility from diet:

$$r\left(q|b,\varepsilon_q\right) \quad = \quad \tau_2 \exp\left(\varepsilon\right)\left(b-\underline{b}+1\right)^{\upsilon_b}\left[\tfrac{1}{1-q}-\left(1+q\right)\right]^{\tau_1}. \tag{A2}$$

In each of the above cases, the remaining parameters of the model are re-calibrated to match the same targets as the benchmark model (see parameter values in Table A11).

The first panel of appendix Figure A22 shows that the model without fixed exercise types substantially overestimates the increase in physical activity over adulthood. Additionally, the second panel reveals a notable deficiency in explaining the low exercise levels among individuals in the bottom third of the distribution. The model also fails to match the persistence at the high end of the distribution. For example, the probability of remaining above 42 MET-hours/week is 31% in this version of the model compared to 42% in the data.

When focusing on diet, panels (c) and (d) show that ignoring dietary habits only marginally changes predicted average life-cycle and cross-sectional patterns of diet quality. However, the final panel demonstrates that such a model drastically fails to capture the distribution of *changes* in diet over successive periods. This discrepancy reveals that while models lacking habitual factors may capture some aspects of the diet distribution, they struggle to accurately represent changes in an individual's diet; in particular, it underestimates the persistence in diet choices. These limitations emphasize the complexity of exercise and dietary behavior and underscores the necessity of considering fixed and habitual influences in endogenous health models to achieve a more accurate understanding of the evolution of healthy behaviors like diet and exercise.

Table A11: Calibrated parameters: Alternate models

Parameter	Benchmark	No Diet Habits	No Exercise Types	Discount Rate Het.	Fixed Effects
ψ_1	0.68	0.68	0.51	0.51	0.80
ψ_2	3.51	3.51	2.70	2.60	1.90
λ_b	0.57	0.57	0.53	0.52	1.03
λ_h	0.18	0.18	0.18	0.17	0.19
τ_1	0.66	0.66	0.66	0.66	1.10
τ_2	1.35	2.00	1.35	1.61	0.55
υ_b	0.55	0.42	0.54	0.40	0.95
υ_q	7.36	-	7.36	4.36	5.93
κ_b	0.77	0.77	0.77	0.77	0.99
σ_e	0.67	0.67	0.45	0.48	0.66
σ_q	0.70	0.40	0.70	0.45	0.47
$\overline{\xi}$	4.81	5.12	-	-	2.52
$\underline{\xi}$	0.27	0.27	-	-	0.90
β	0.96	0.96	0.96	0.96	0.96
β_l	-	-	-	0.89	-
β_h	-	-	-	0.98	-

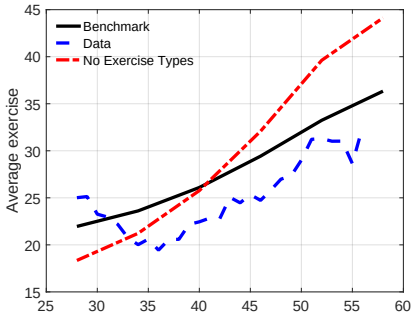
A5.2 Discount rate heterogeneity

As observed above, both permanent activity types and diet habits are crucial for understanding the evolution of behaviors over the life cycle. An alternative hypothesis is that the data can instead be accounted for by a model with heterogeneous time preferences. We therefore explore a framework where agents are heterogeneous in their discounting of the future. Given our socio-economically homogeneous sample, it is difficult to credibly identify discount rate heterogeneity over and above the existing channels in the benchmark model. However, one could argue that the observed tails in the activity distribution (inactive versus the physically active types) could be attributed to heterogeneous discount factors. Hence, we propose a framework where exercise types are replaced by discount rate types, while maintaining alignment with the same calibration targets as the benchmark model. Specifically, preferences for physical activity remain determined by Equation (A1), but we introduce three discount factor values across individuals (confer appendix Table A11 for values): low ($\underline{\beta} < \beta$), high ($\overline{\beta} > \beta$), and a middle group whose discount factor is given by the benchmark value, β (see Section 6.1).

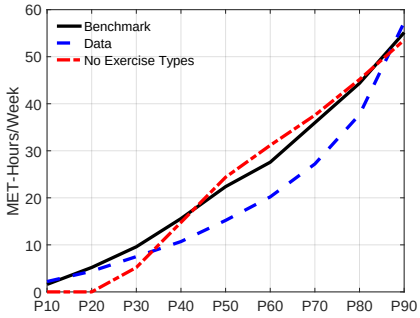
As this version of the model has both discount rate heterogeneity and diet habits, it is able to match the evolution of diet quality similarly well as the benchmark (see appendix Figure A23). Furthermore, compared to entirely excluding exercise types as in the previous section, integrating discount rate heterogeneity introduces additional free parameters, enhancing the model's capability to replicate the persistence of exercise across subsequent

Figure A22: Diet and physical activity: no habits and exercise types

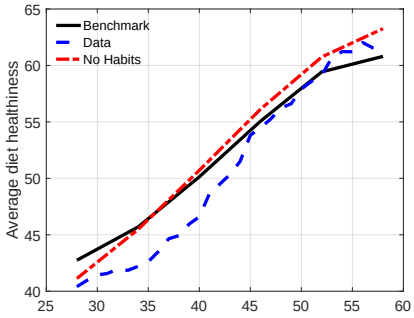
(a) Physical activity by age



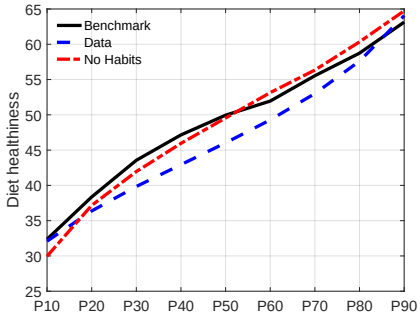
(b) Distribution of physical activity



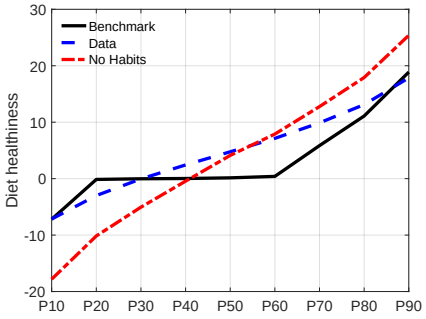
(c) Diet by age



(d) Distribution of diet



(e) Distribution of change in diet



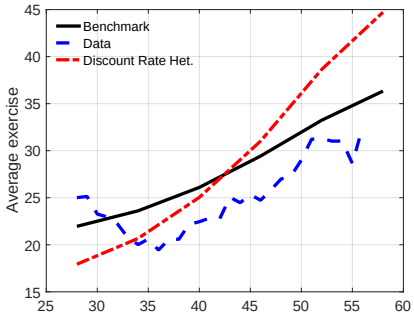
Notes: Panels (a) and (c) plot, respectively, the age-specific averages for exercise and diet quality in the benchmark model and the re-calibrated model. Panels (b), (d) and (e) plot, respectively, the percentile distribution of exercise, diet quality, and change in diet quality ($q - q_{-1}$) in the benchmark model and the re-calibrated model.

periods. However, compared to the benchmark with exercise types, discount rate heterogeneity still substantially overestimates the rise in physical activity over adulthood. The model also continues to miss the bottom third of the exercise distribution almost entirely.

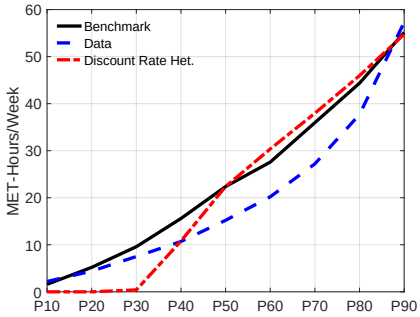
These patterns again highlight the role of exercise types in replicating important features of the data.

Figure A23: Diet and physical activity: Discount rate heterogeneity

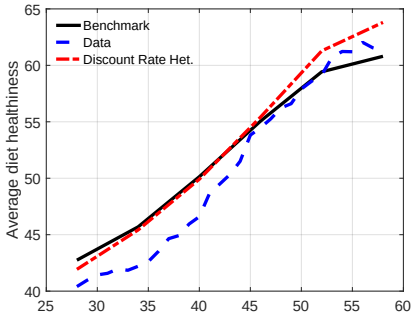
(a) Physical Activity by Age



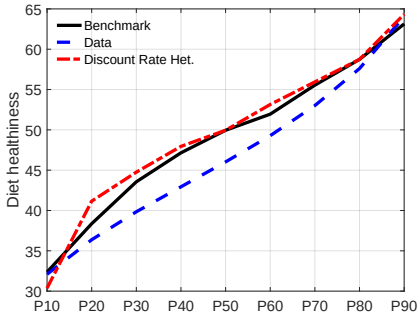
(b) Distribution of Physical Activity



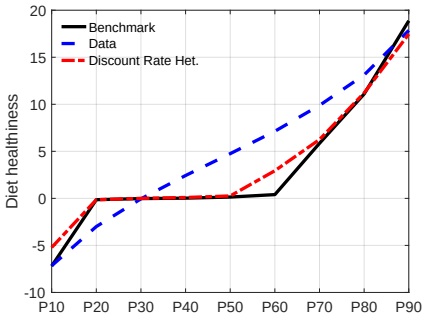
(c) Diet by Age



(d) Distribution of Diet



(e) Distribution of Change in Diet



Notes: Panels (a) and (c) plot, respectively, the age-specific averages for exercise and diet quality in the benchmark model and the re-calibrated model with discount rate heterogeneity replacing exercise types. Panels (b), (d), and (e) plot, respectively, the percentile distribution of exercise, diet quality, and change in diet quality ($q - q_{-1}$) in the benchmark model and the discount rate heterogeneity model.

A5.3 Fixed effects model of BMI

In our benchmark model, we constructed a metabolic index using observable early-life characteristics and family disease history. This approach offers several advantages. First, it relies on indicators that are established before adulthood and on diseases with known hereditary linkages. Second, as demonstrated in Figure 8, the index is highly predictive of weight changes later in life. Third, using observable data as our benchmark ensures better external validity, as the results are more likely to generalize to other populations. Finally, this approach helps to avoid potential problems of overfitting that can occur when using fixed effects.

Our benchmark metabolic index, however, is based on a limited number of available indicators. As an alternative, we therefore leveraged our long panel data to estimate unobserved individual fixed effects and then used these fixed effects as proxies for metabolic groups. We do so by using a linear dynamic panel data model with lagged dependent variables and individual-level fixed effects:

$$BMI_{it} = \beta_1 BMI_{it-1} + \beta_2 BMI_{it-2} + \beta_3 Diet_{it} + \beta_4 Exercise_{it} + \eta_i + \delta_t + \epsilon_{it}. \quad \text{(A3)}$$

The model was estimated using the system GMM estimator from Blundell and Bond (1998) (confer appendix Table A12 for results). After estimating the model, we predicted the individual fixed effect η_i for each nurse in the estimation sample. Appendix figure A24 compares our previously defined metabolic index with the predicted fixed effects from the dynamic panel model for BMI. Although there are some variations between the measures, a clear negative correlation (-0.167), as expected, is observed.

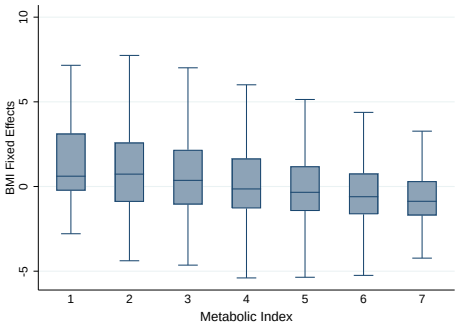
Table A12: Dynamic Panel Data Model

	Estimates
BMI in $t-1$	0.735***
BMI in $t-2$	0.001
Diet Quality Score	-0.035***
Physical Activity Score	-0.017***
Observations	30,589

Notes: Cluster-robust standard errors. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Includes time and individual fixed effects. Second lag of BMI and first lag of diet and exercise used as instruments in the equation in first differences. First lag of BMI and contemporaneous values for diet and exercise used as instruments for the levels equation. Instruments are collapsed and forward orthogonal deviations are used due to missing values in the data. Hansen test of overidentifying restrictions $p = 0.123$. Observations with BMI >70 or physical activity score >100 excluded.

We use the estimated fixed effects as an alternative approach to calibrate our model.

Figure A24: Distribution of BMI fixed effects across different metabolic groups



Notes: Correlation coefficient between estimated fixed effects and metabolic index is -0.167.

Specifically, we categorize individuals into metabolic types $\alpha = 1$, $\alpha = 2$, and $\alpha = 3$, corresponding to each tercile of the fixed effects distribution. We then re-estimate the laws of motion for BMI and morbidities (appendix Tables A13–A14) and re-calibrate the model accordingly (final column of appendix Table A11).

The estimated impact of health behaviors in adulthood remains quite similar under the fixed effects calibration compared to our benchmark. For instance, counterfactually setting both diet and exercise at the 10^{th} percentile of their distributions increased estimated medical spending by 9.5%, compared to 12% under the benchmark model. Similarly, medical spending decreased by 8% under both models when effort levels were set at the 90^{th} percentile of the distribution.

However, the shift to the fixed effects strategy had a more pronounced impact on the role of initial conditions. When equalizing all initial conditions to their mean values, we find that approximately 60% of the variation in BMI among individuals in their fifties can now be explained by the variance in initial conditions, compared to roughly a third under the benchmark model. These figures can be viewed as bounds on the role of initial conditions: the lower bound (one third) reflects the impact when initial conditions are not fully accounted for and include some residual unobserved heterogeneity, while the upper bound (60%) represents the role of initial conditions when fixed effects capture unobserved individual heterogeneity. The 60% estimate can be considered an upper bound because it assumes that all potential sources of variation related to initial conditions are fully captured by the fixed effects model, including, for example, other persistent (but not necessarily permanent) behaviors that are unaccounted for in the model.

Table A13: Ordered logit estimates of BMI law of motion using fixed effects model

	Estimates
BMI in $t-1$	
$b=2$	2.763***
$b=3$	4.930***
$b=4$	6.854***
$b=5$	8.725***
$b=6$	11.921***
Diet Quality Group	-0.239***
Physical Activity Group	-0.216***
Metabolic Group=2	1.495***
Metabolic Group=3	3.055***
cut 1	1.302***
cut 2	4.597***
cut 3	7.143***
cut 4	9.352***
cut 5	12.923***
Observations	43596
Pseudo R^2	0.4452

$^{*}\ p<0.05$, $^{**}\ p<0.01$, $^{***}\ p<0.001$

Table A14: Logit estimates of morbidity law of motion using fixed effects model

	Estimates
Morbidity=1	0.984***
Age	0.032***
Morbidity=1 $\times$ Age	-0.028***
Diet Quality Group	-0.164***
Physical Activity Group	-0.044**
BMI in $t-1$	
b=2	0.506***
b=3	0.868***
b=4	1.226***
b=5	1.513***
b=6	1.637***
Metabolic Group=2	0.107**
Metabolic Group=3	0.265***
Constant	-3.559***
Observations	44149
R^2	0.0481

$^{*}\ p < 0.05$, $^{**}\ p < 0.01$, $^{***}\ p < 0.001$

A Supplementary Materials, Not for Publication

S1 Drivers of obesity: Lessons from the public health literature

A key factor contributing to the current obesity epidemic among adult Americans is the sharp rise in childhood obesity over recent decades. Data from the CDC show that the prevalence of obesity among children and adolescents aged 2–19 increased from 5% in the 1970s to 19.3% in 2017–18 (Fryar et al., 2020). Obese children are much more likely to become obese adults, with the risk increasing if one or both parents are obese (Whitaker et al., 1997). More broadly, adult obesity has been traced back to various childhood circumstances, including causes as fundamental as genes and gene-environment interactions (Kilpeläinen et al., 2011). Prenatal factors influencing obesity include maternal smoking (Oken et al., 2008), weight gain (Ludwig and Currie, 2010), and blood sugar levels (Hillier et al., 2007) during pregnancy. Postnatal and childhood influences include breastfeeding (Harder et al., 2005), infant weight gain (Baird et al., 2005), sleep patterns (Landhuis et al., 2008), and the development of lasting food preferences (Gruber and Haldeman, 2009). However, research also suggests that genes are not destiny. For example, (Kilpeläinen et al., 2011) re-analyzed data from 54 studies and found that physical activity significantly reduced the risk of obesity in adults carrying an obesity-promoting gene.

S2 Stylized facts by sub-groups

We test the robustness of our stylized facts to differences in marital and fertility status, spousal education, smoking habits, mental health status, and working night shifts. We use the marital status information from the 2013 wave to categorize nurses as married if they reported being married or living with a domestic partner. Non-married nurses included those who were divorced, widowed, separated, or never married. Based on this definition, 81.6% of the nurses in our sample were married. While the nurses all have similar education (an associate or a bachelor's degree), they could be married to spouses with varying education levels. Education of the spouse was reported in the 1999 survey wave. We construct an indicator for college educated spouse if the reported education levels were equal to a two-year college or above. Based on this definition, 80% of the married nurses in our sample had a college educated spouse. To understand the role of fertility, we look at nurses with and without children. However, the survey does not include any direct questions on the total number of children. We infer the number of living children by looking at responses on *parity*: total number of pregnancies that resulted in delivery at $\geq$ 6 months gestation, of either a live birth or a stillbirth. Note that while this measure should be a good approximation of the number children, it has two special properties: (1) this measure will include any instance of stillbirth and (2) it does not include non-biological children who may be living with the survey respondents. Based on this classification, roughly 18.4% of nurses in our sample were classified as without children (zero parity) and rest with at least one child (positive parity).

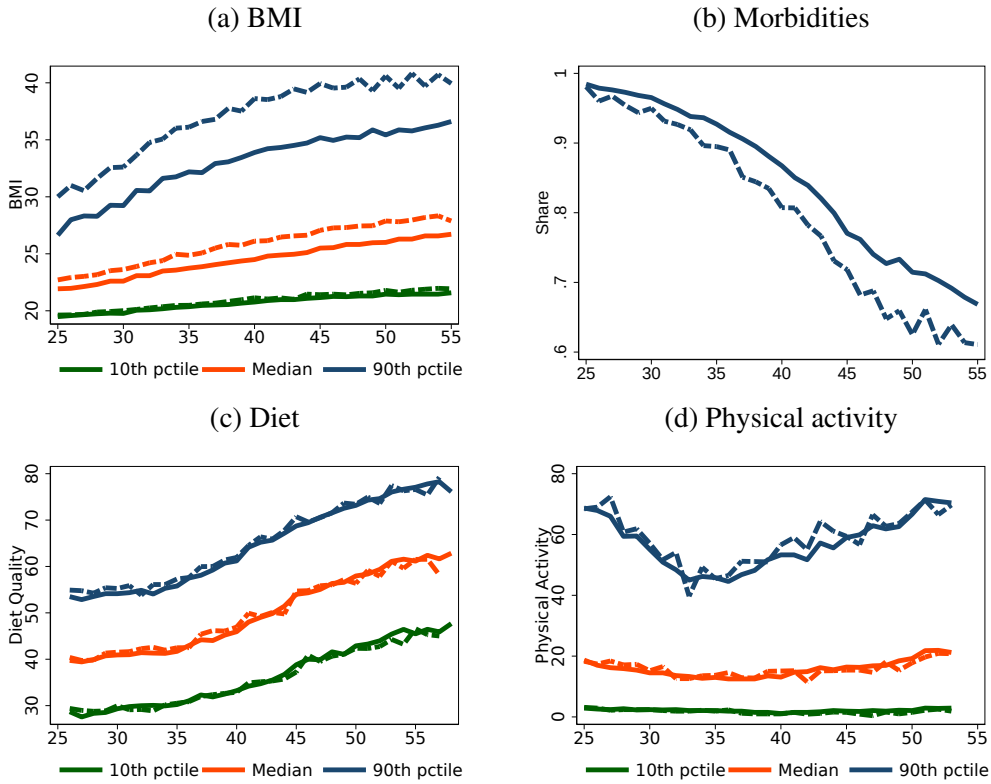
Smoking status was constructed from the 2015 survey wave. Nurses were designated as 0 if they never smoked or 1 if they had some smoking history. Based on this classification, 65% of the nurses in our sample were non-smokers. For mental health status, we use the SF-36 mental health index constructed from available information in the 1993 survey wave. This is a continuous index ranging from 0 to 100. We discretize the index into terciles and compare those in the top and bottom terciles. Finally, we use retrospective questions asked in the 2009 survey wave to categorize nurses who worked nights shifts versus those who did not. Nurses were asked to report their working shifts at various points in their life cycle: between ages 20-25, ages 26-35, ages 36-45, and after age 45. We categorize night shift nurses as those who only worked night-shifts during at least one of the four life phases mentioned above. Day shifters are then categorized as those who did not work night-shifts exclusively at any point during their lives up until the 2009 survey wave.

Figures in this appendix show that our stylized facts hold when broken down by the above sub-groups. For example, Figure S1 shows that while non-married nurses exhibited higher BMI and morbidities than married nurses, the life-cycle patterns stated in stylized fact 1 hold for both groups. Figure S2 shows life-cycle patterns for BMI, morbidities, and healthy behaviors by education of the spouse (college vs. non-college). Similar to non-married nurses, the BMI and morbidity levels are somewhat higher for nurses with non-college-educated spouses. In this case, we also observe somewhat poorer diet and exercise levels. However, we observe the same life-cycle improvements in behaviors and deterioration of health for both groups.

Concerns might arise about other factors influencing the observed life-cycle patterns highlighted in our stylized facts. For example, poor mental health and/or smoking could drive changes in BMI and morbidity and deteriorate health behaviors over adulthood. At the same time, the nature of the working shift (night versus day) could also impact diet and physical activity behaviors by impacting circadian rhythms. Figure S4 indicates some increase in morbidities and poorer health behaviors for nurses in the bottom tercile of the survey's available mental health index (SF-36). However, we once again observe life-cycle trends that align with stylized fact 1 for nurses with both high and low mental health scores. Similarly, Figure S5 shows some differences in outcomes for smokers, but the trends remain consistent between smokers and non-smokers. Finally, Figure S6 shows some differences in health outcomes for nurses who did and did not work night shifts between ages 20 and 46, but the trends remain consistent between the two groups.

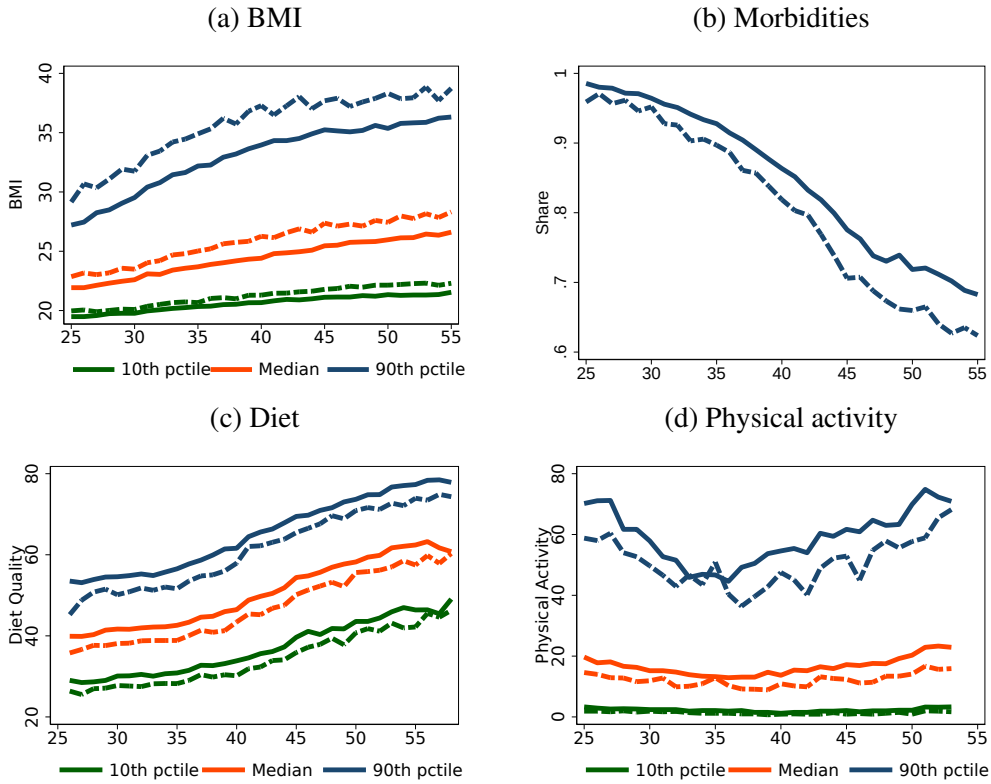
The remaining figures in this appendix show that the remaining stylized facts also hold for each of these sub-groups.

Figure S1: Life-cycle patterns in BMI, morbidities, diet quality, and physical activity by marital status



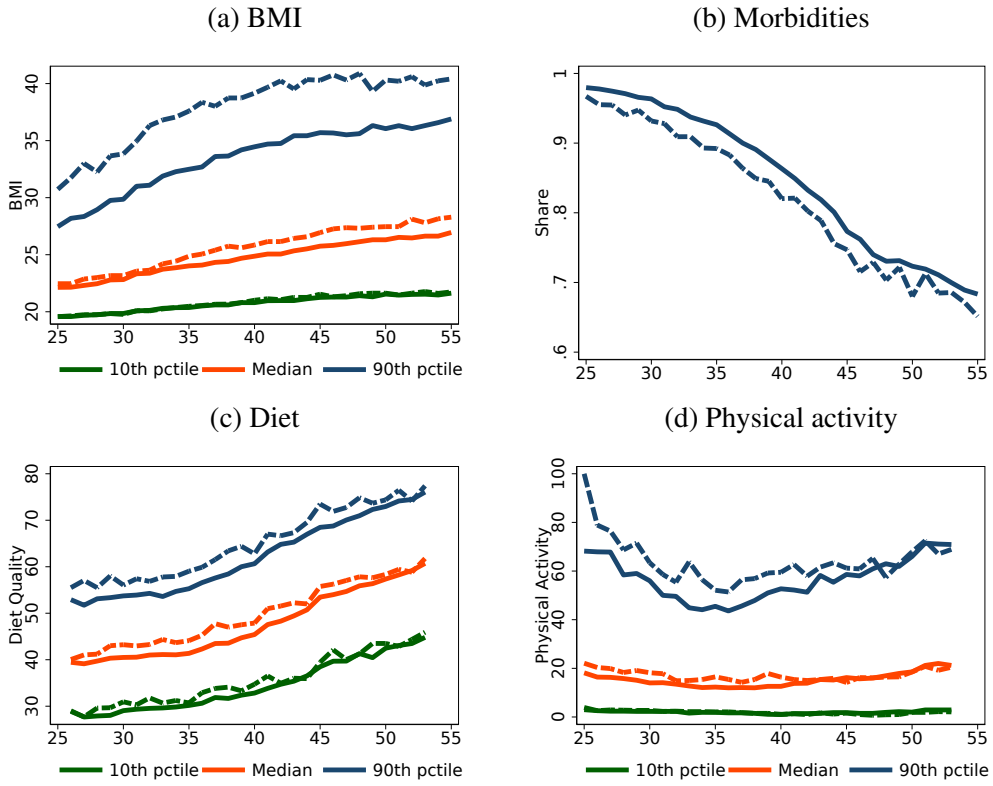
Notes: Panels (a), (c), and (d) plot evolution (median, 10th and 90th percentile) of BMI, diet quality, and physical activity index, respectively, over the life cycle of registered nurses in NHS II, who were aged 25 to 30 in the initial 1989 wave. Solid lines indicate those who were married and dashed lines are for non-married. For morbidity measure, a total count of chronic conditions is used. Panel (b) plots the share of those with zero morbidity count over the life cycle.

Figure S2: Life-cycle patterns in BMI, morbidities, diet quality, and physical activity by education status of the spouse



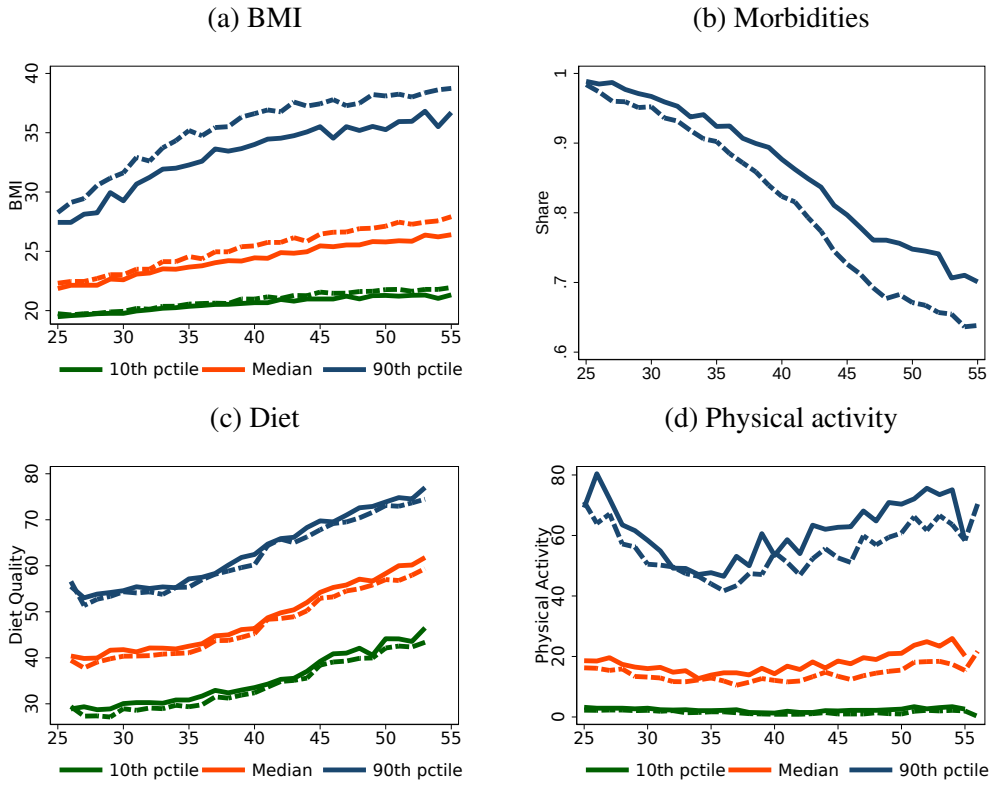
Notes: Panels (a), (c), and (d) plot evolution (median, 10th and 90th percentile) of BMI, diet quality, and physical activity index, respectively, over the life cycle of registered nurses in NHS II, who were aged 25 to 30 in the initial 1989 wave. Solid lines indicate those whose spouse had a college degree and dashed lines are for those whose spouse had less than college education. For morbidity measure, a total count of chronic conditions is used. Panel (b) plots the share of those with zero morbidity count over the life cycle.

Figure S3: Life-cycle patterns in BMI, morbidities, diet quality, and physical activity by fertility status



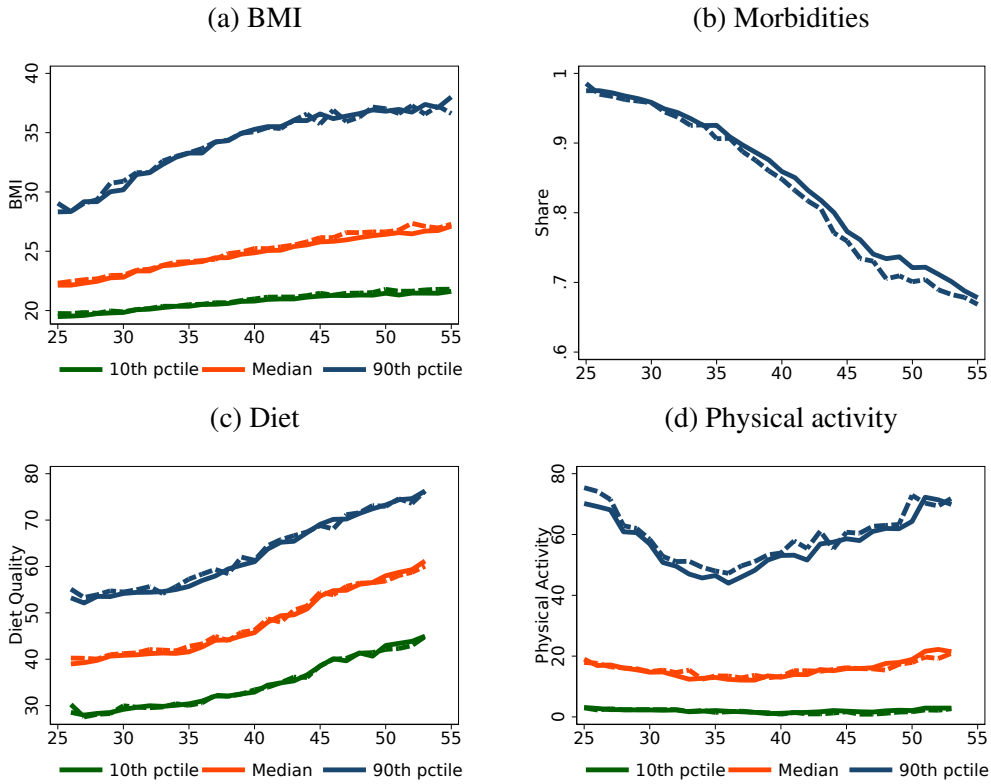
Notes: Panels (a), (c), and (d) plot evolution (median, 10th and 90th percentile) of BMI, diet quality, and physical activity index, respectively, over the life cycle of registered nurses in NHS II, who were aged 25 to 30 in the initial 1989 wave. Solid lines indicate those nurses who had at least one child (measured by pregnancies that resulted in the delivery at $\geq$6 months gestation) and dashed lines are for those with no biological children (zero parity). For morbidity measure, a total count of chronic conditions is used. Panel (b) plots the share of those with zero morbidity count over the life cycle.

Figure S4: Life-cycle patterns in BMI, morbidities, diet quality, and physical activity by mental health status



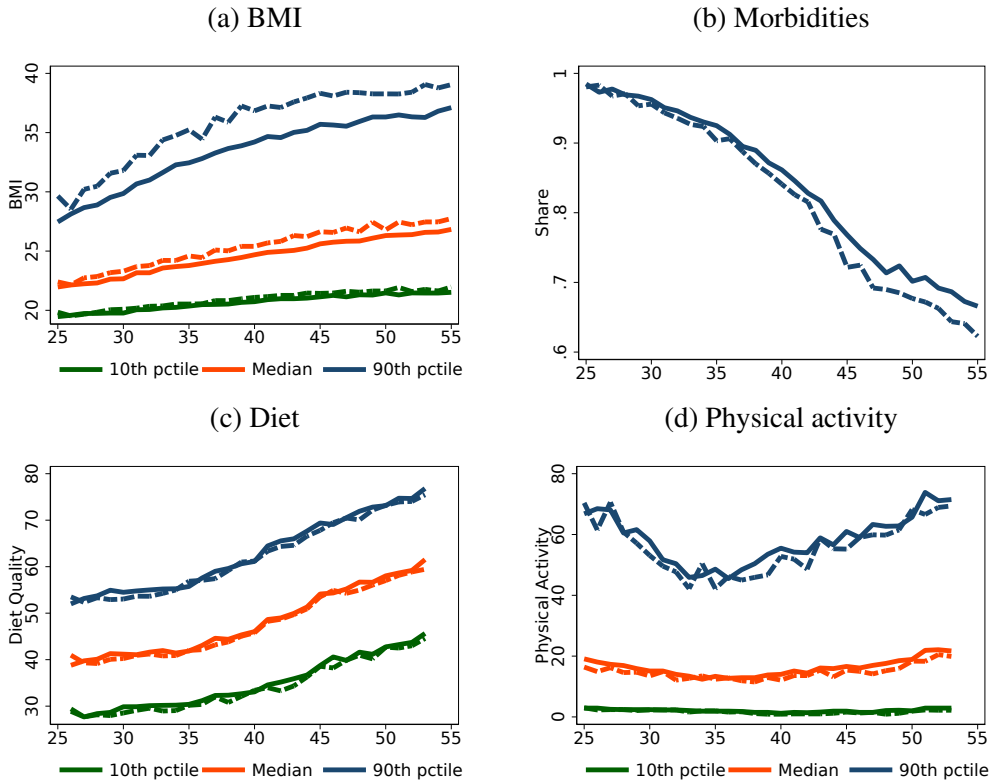
Notes: Panels (a), (c), and (d) plot evolution (median, 10th and 90th percentile) of BMI, diet quality, and physical activity index, respectively, over the life cycle of registered nurses in NHS II, who were aged 25 to 30 in the initial 1989 wave. Solid lines indicate those in the highest tercile of the mental health index and dashed lines are for those in lowest tercile. Mental health was measured in 1993 using SF-36 mental health index. For morbidity measure, a total count of chronic conditions is used. Panel (b) plots the share of those with zero morbidity count over the life cycle.

Figure S5: Life-cycle patterns in BMI, morbidities, diet quality, and physical activity by smoking status



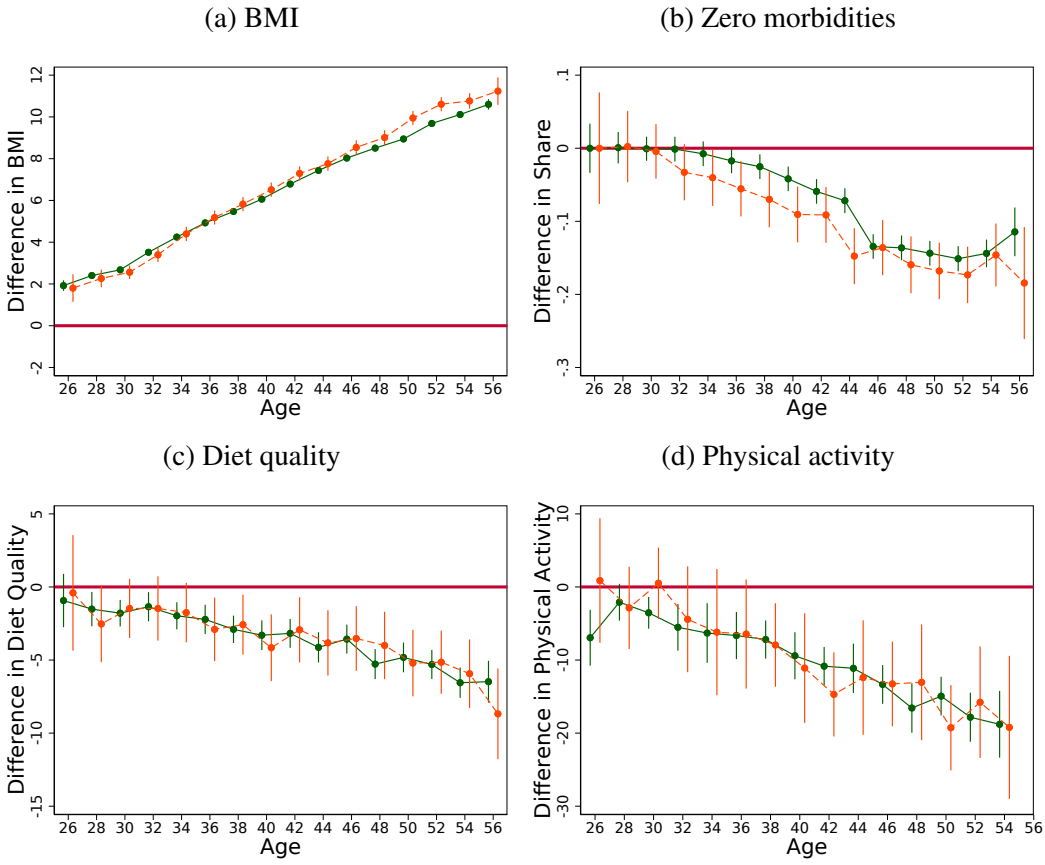
Notes: Panels (a), (c), and (d) plot evolution (median, 10th and 90th percentile) of BMI, diet quality, and physical activity index, respectively, over the life cycle of registered nurses in NHS II, who were aged 25 to 30 in the initial 1989 wave. Solid lines indicate those who never smoked and dashed lines are for those who smoked at some point. For morbidity measure, a total count of chronic conditions is used. Panel (b) plots the share of those with zero morbidity count over the life cycle.

Figure S6: Life-cycle patterns in BMI, morbidities, diet quality, and physical activity by working shift status



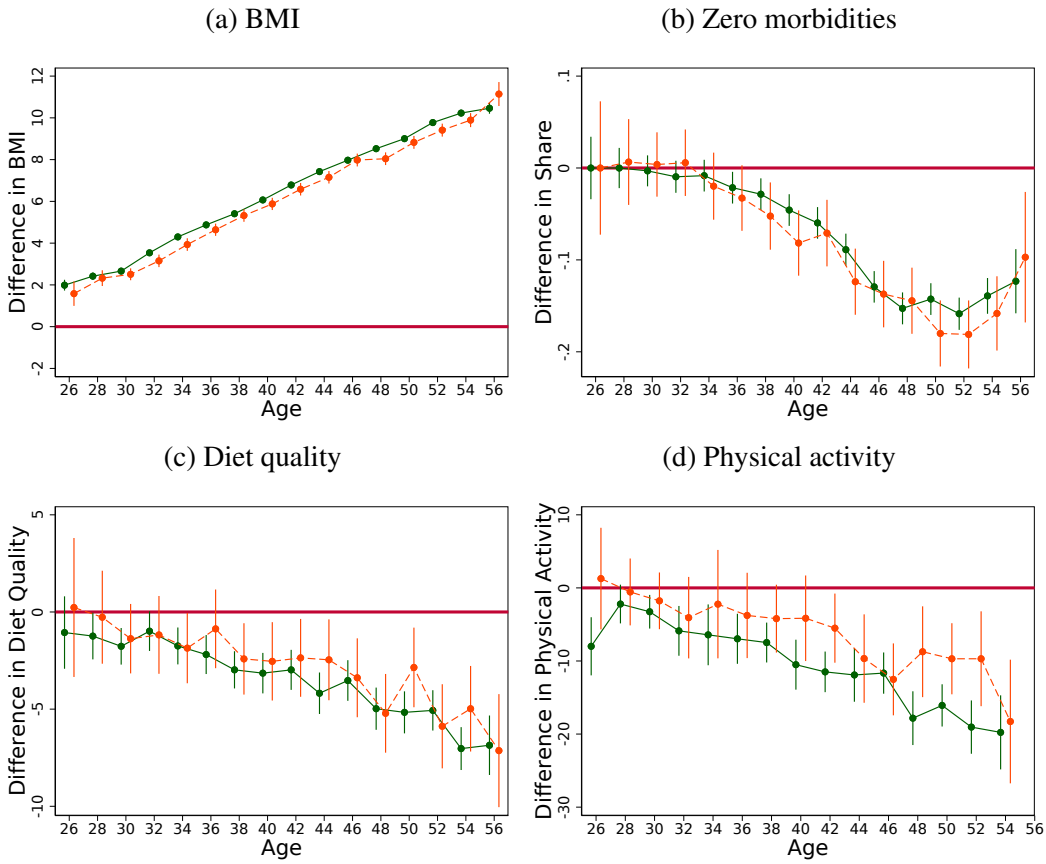
Notes: Panels (a), (c), and (d) plot evolution (median, 10th and 90th percentile) of BMI, diet quality, and physical activity index, respectively, over the life cycle of registered nurses in NHS II, who were aged 25 to 30 in the initial 1989 wave. Dashed lines indicate those who exclusively worked night shifts at some point between the ages of 20 and 46 and solid lines are for the rest. For morbidity measure, a total count of chronic conditions is used. Panel (b) plots the share of those with zero morbidity count over the life cycle.

Figure S7: Life-cycle gaps between BMI gainers and stayers by marital status



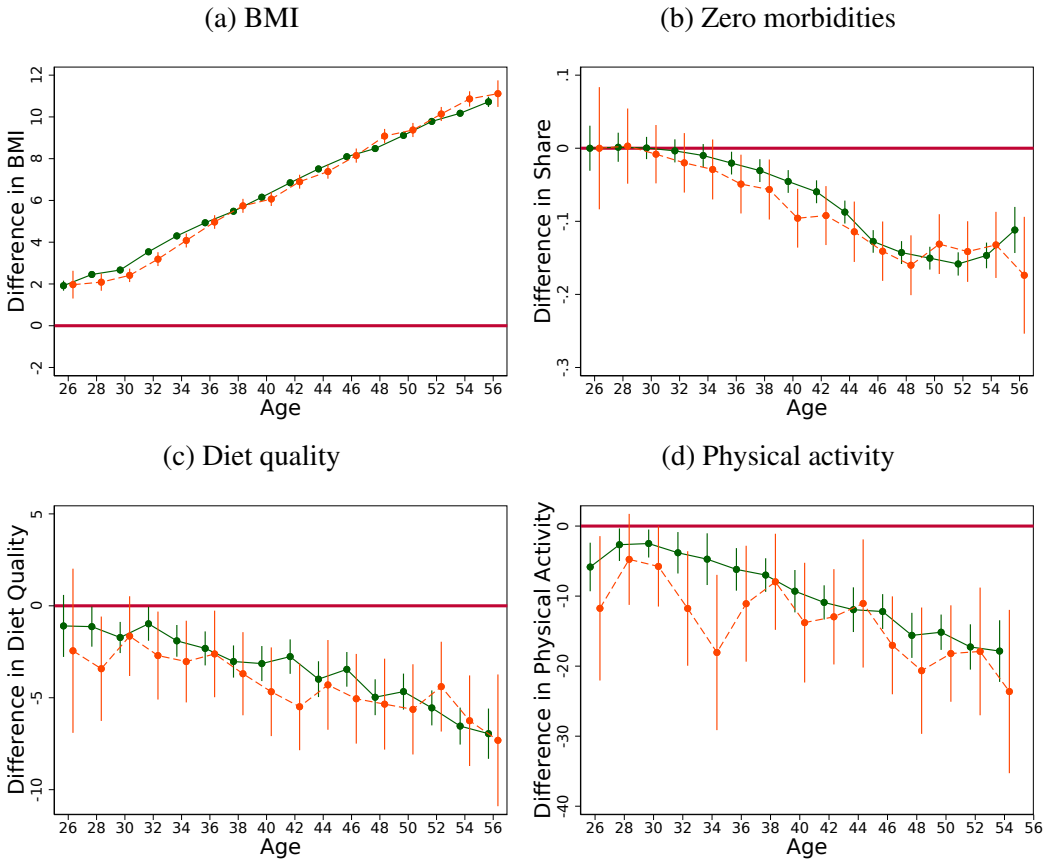
Notes: The figures plot difference in outcomes over the life cycle for those who maintained their healthy BMI (18.5-25) between the years 1989 and 2015 and those whose BMI increased from that in the healthy range in 1989 to BMI>30 in 2015 (last survey year). Solid green lines indicate these differences for married nurses and dashed orange lines for non-married nurses. NHS II respondents aged 25-30 in 1989 are considered. Panel (b) plots the difference in the share of those with zero morbidity count between the two BMI-change groups.

Figure S8: Life-cycle gaps between BMI gainers and stayers
by education status of spouse



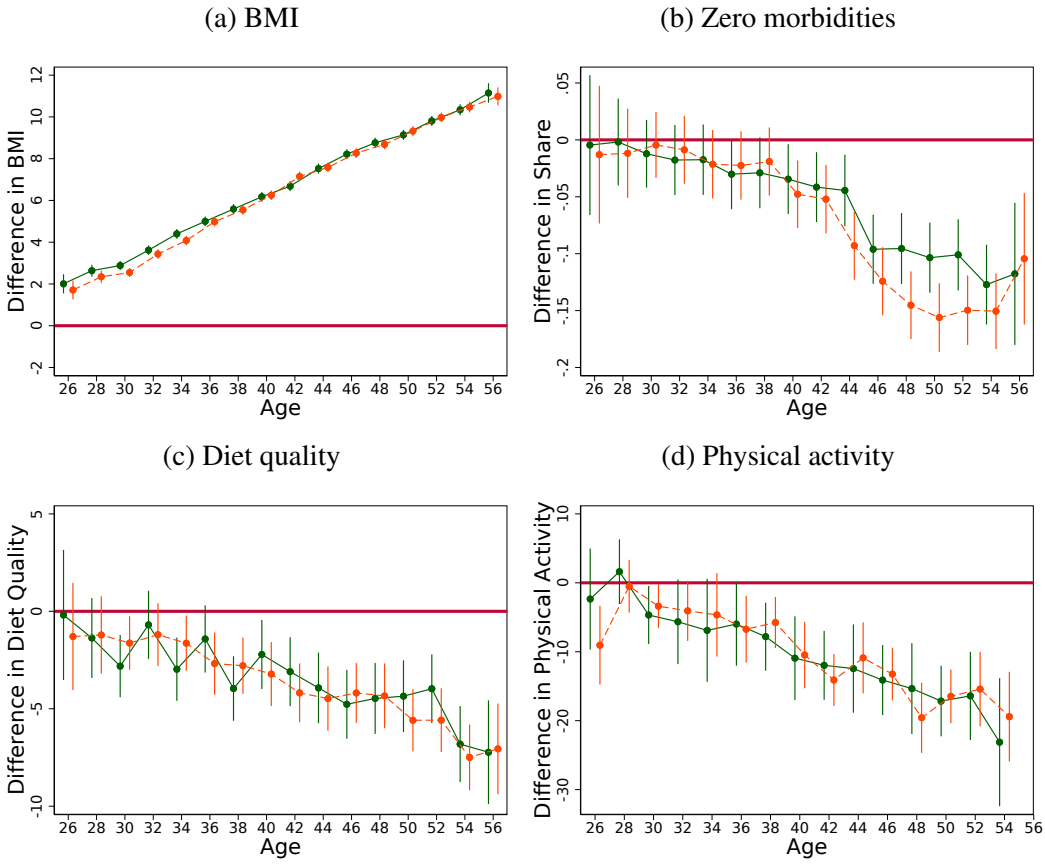
Notes: The figures plot average outcomes for those whose BMI increased from healthy (18.5-25) in 1989 to obese (>30) in 2015 minus average outcomes for those who maintained a healthy BMI between 1989 and 2015. Solid green lines indicate these differences for married nurses whose spouse have a college degree and dashed orange lines for those with non-college-educated spouses. NHS II respondents aged 25-30 in 1989 are considered.

Figure S9: Life-cycle gaps between BMI gainers and stayers by fertility status



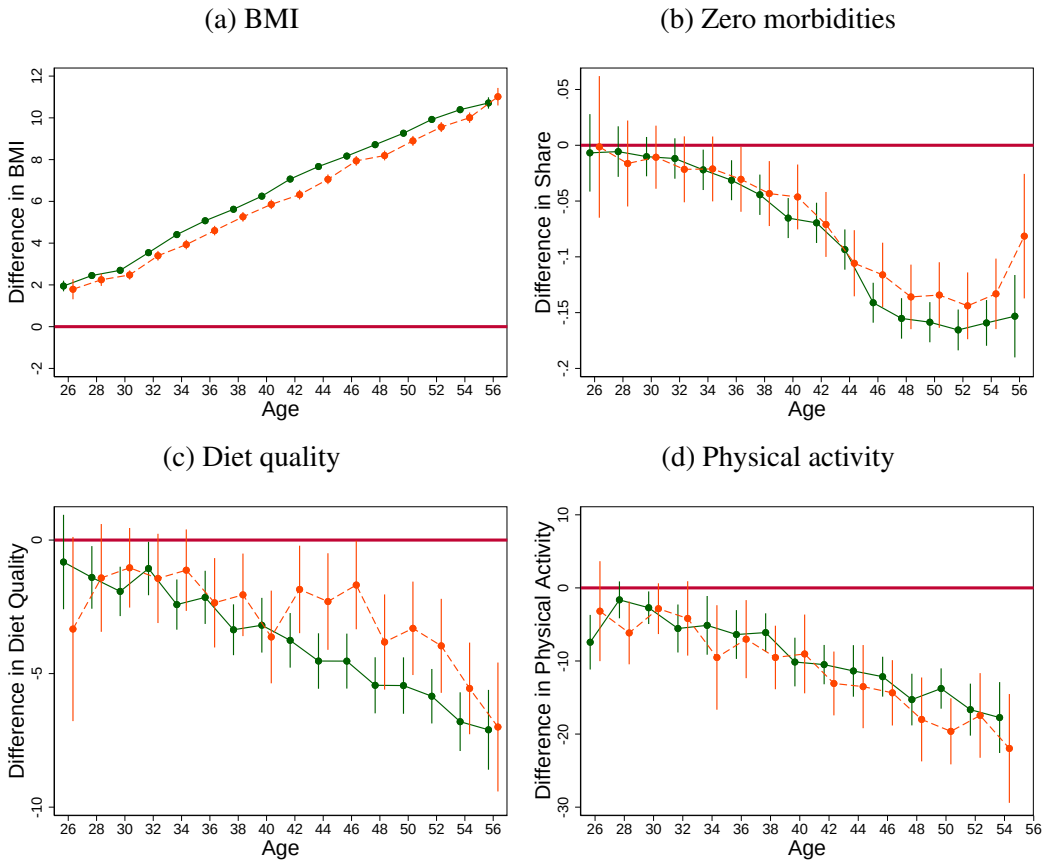
Notes: The figures plot average outcomes for those whose BMI increased from healthy (18.5-25) in 1989 to obese (>30) in 2015 minus average outcomes for those who maintained a healthy BMI between 1989 and 2015. Solid green lines indicate these differences for those nurses who had at least one child (measured by pregnancies that resulted in the delivery at ≥6 months gestation) and dashed orange lines are for those with no biological children (zero parity). NHS II respondents aged 25-30 in 1989 are considered.

Figure S10: Life-cycle gaps between BMI gainers and stayers by mental health status



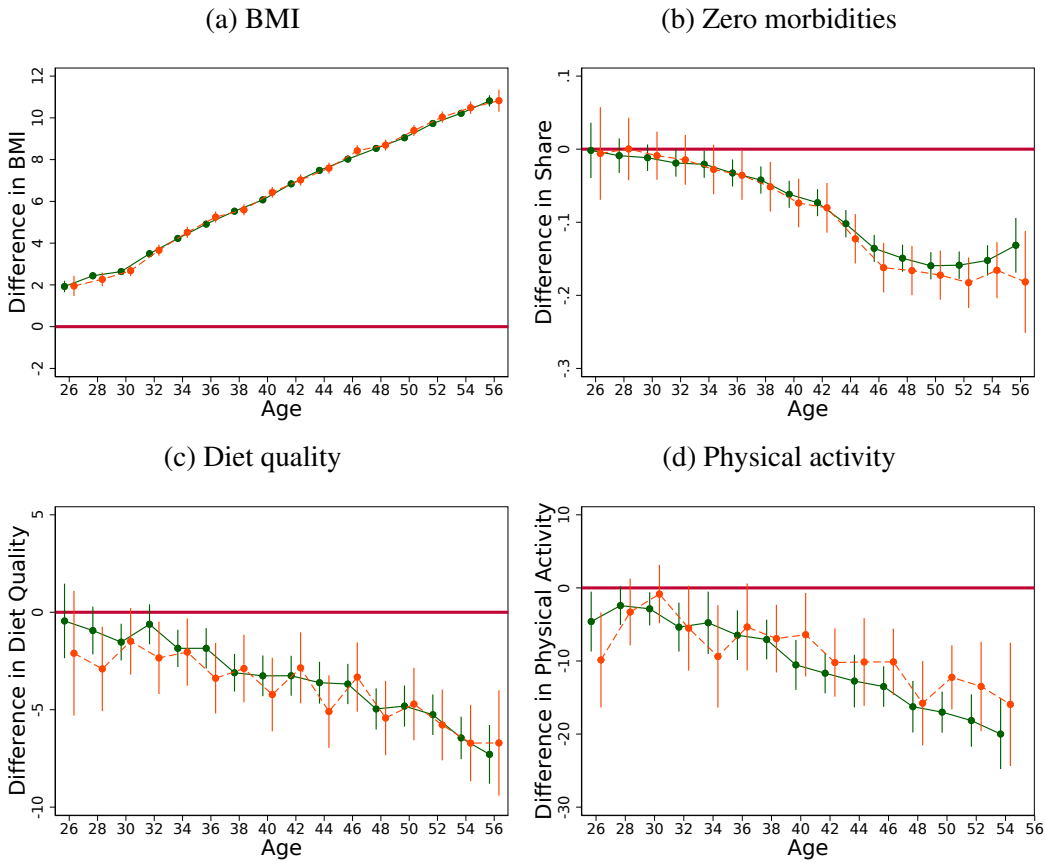
Notes: The figures plot average outcomes for those whose BMI increased from healthy (18.5-25) in 1989 to obese (>30) in 2015 minus average outcomes for those who maintained a healthy BMI between 1989 and 2015. Solid green lines indicate these differences for those in the highest tercile of the mental health index and dashed orange lines are for those in lowest tercile. Mental health was measured in 1993 using SF-36 mental health index. NHS II respondents aged 25-30 in 1989 are considered.

Figure S11: Life-cycle gaps between BMI gainers and stayers by smoking status



Notes: The figures plot average outcomes for those whose BMI increased from healthy (18.5-25) in 1989 to obese (>30) in 2015 minus average outcomes for those who maintained a healthy BMI between 1989 and 2015. Solid green lines indicate these differences for those who never smoked and dashed orange lines for those who smoked at some point. NHS II respondents aged 25-30 in 1989 are considered.

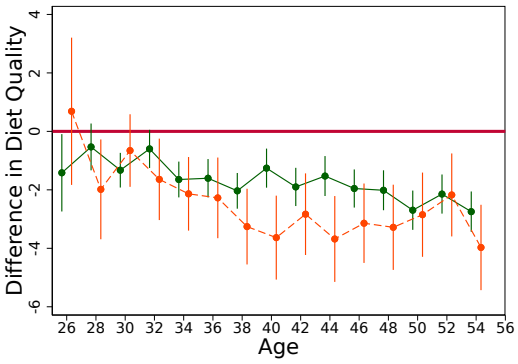
Figure S12: Life-cycle gaps between BMI gainers and stayers
by working shift status



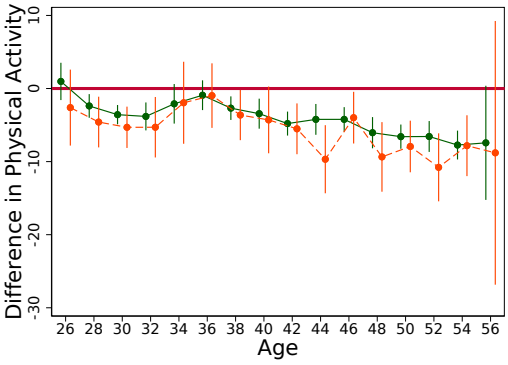
Notes: The figures plot average outcomes for those whose BMI increased from healthy (18.5-25) in 1989 to obese (>30) in 2015 minus average outcomes for those who maintained a healthy BMI between 1989 and 2015. Dashed orange lines indicate these differences for those who exclusively worked night shifts at some point between the ages of 20 and 46 and solid green lines are for the rest. NHS II respondents aged 25-30 in 1989 are considered.

Figure S13: Life-cycle gaps between morbidity gainers and stayers by marital status

(a) Diet Quality



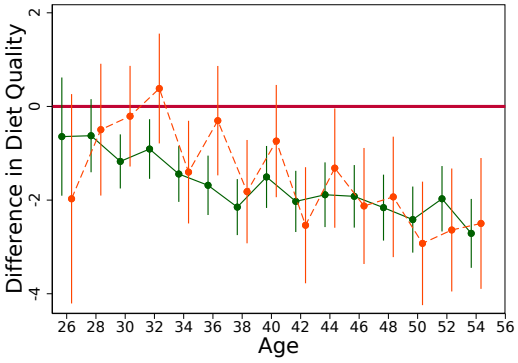
(b) Physical Activity



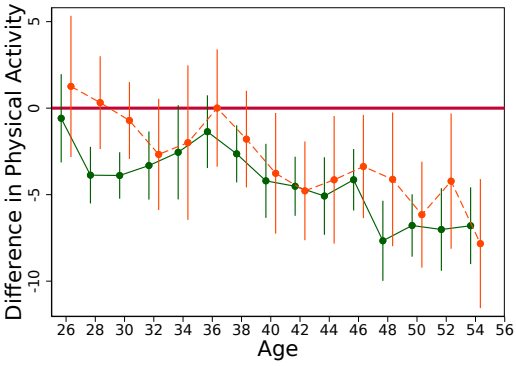
Notes: The figures plot average outcomes for those who obtained at least one morbidity between 1989 and 2015 minus average outcomes for those who maintained a zero morbidity count between 1989 and 2015. Solid green lines indicate these differences for married nurses and dashed orange lines for non-married nurses. NHS II respondents aged 25-30 in 1989 are considered.

Figure S14: Life-cycle gaps between morbidity gainers and stayers by education status of spouse

(a) Diet Quality



(b) Physical Activity

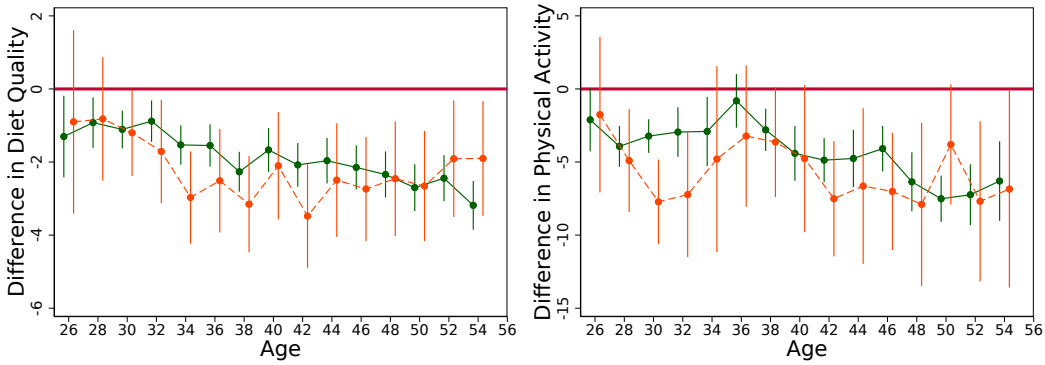


Notes: The figures plot average outcomes for those who obtained at least one morbidity between 1989 and 2015 minus average outcomes for those who maintained a zero morbidity count between 1989 and 2015. Solid green lines indicate these differences for married nurses whose spouse have a college degree and dashed orange lines for those with non-college-educated spouses. NHS II respondents aged 25-30 in 1989 are considered.

Figure S15: Life-cycle gaps between morbidity gainers and stayers by fertility status

(a) Diet Quality

(b) Physical Activity

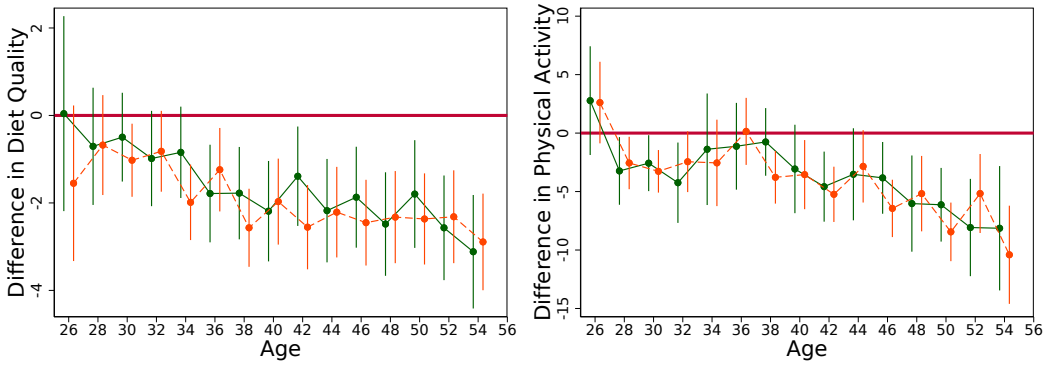


Notes: The figures plot average outcomes for those who obtained at least one morbidity between 1989 and 2015 minus average outcomes for those who maintained a zero morbidity count between 1989 and 2015. Solid green lines indicate these differences for those nurses who had at least one child (measured by pregnancies that resulted in the delivery at $\geq$6 months gestation) and dashed orange lines are for those with no biological children (zero parity). NHS II respondents aged 25-30 in 1989 are considered.

Figure S16: Life-cycle gaps between morbidity gainers and stayers by mental health status

(a) Diet Quality

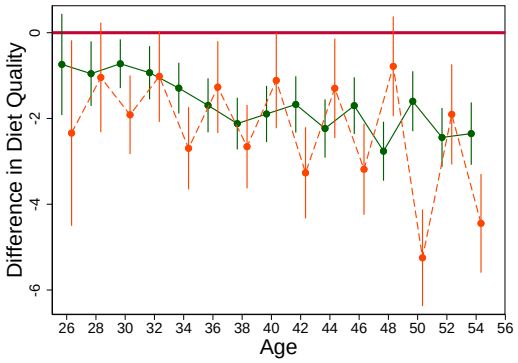
(b) Physical Activity



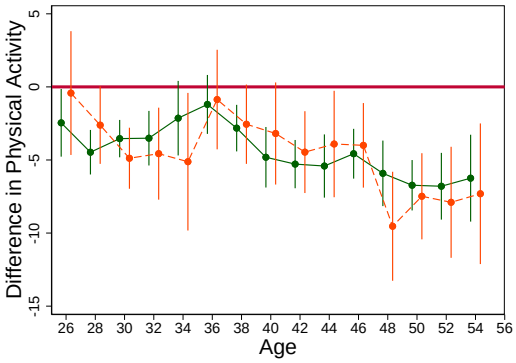
Notes: The figures plot average outcomes for those who obtained at least one morbidity between 1989 and 2015 minus average outcomes for those who maintained a zero morbidity count between 1989 and 2015. Solid green lines indicate these differences for those in the highest tercile of the mental health index and dashed orange lines are for those in lowest tercile. Mental health was measured in 1993 using SF-36 mental health index. NHS II respondents aged 25-30 in 1989 are considered.

Figure S17: Life-cycle gaps between morbidity gainers and stayers by smoking status

(a) Diet Quality



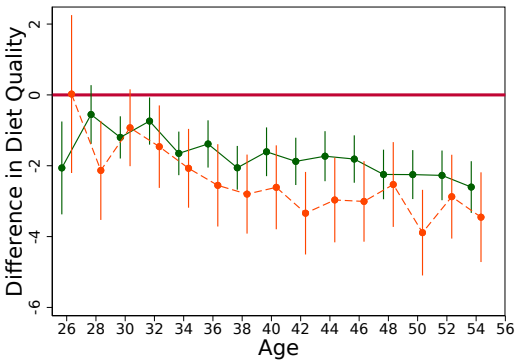
(b) Physical Activity



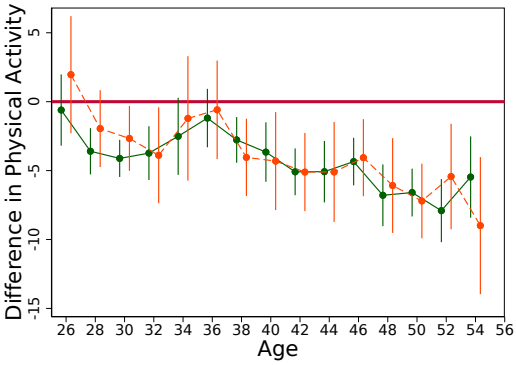
Notes: The figures plot average outcomes for those who obtained at least one morbidity between 1989 and 2015 minus average outcomes for those who maintained a zero morbidity count between 1989 and 2015. Solid green lines indicate these differences for those who never smoked and dashed orange lines for those who smoked at some point. NHS II respondents aged 25-30 in 1989 are considered.

Figure S18: Life-cycle gaps between morbidity gainers and stayers by working shift status

(a) Diet Quality

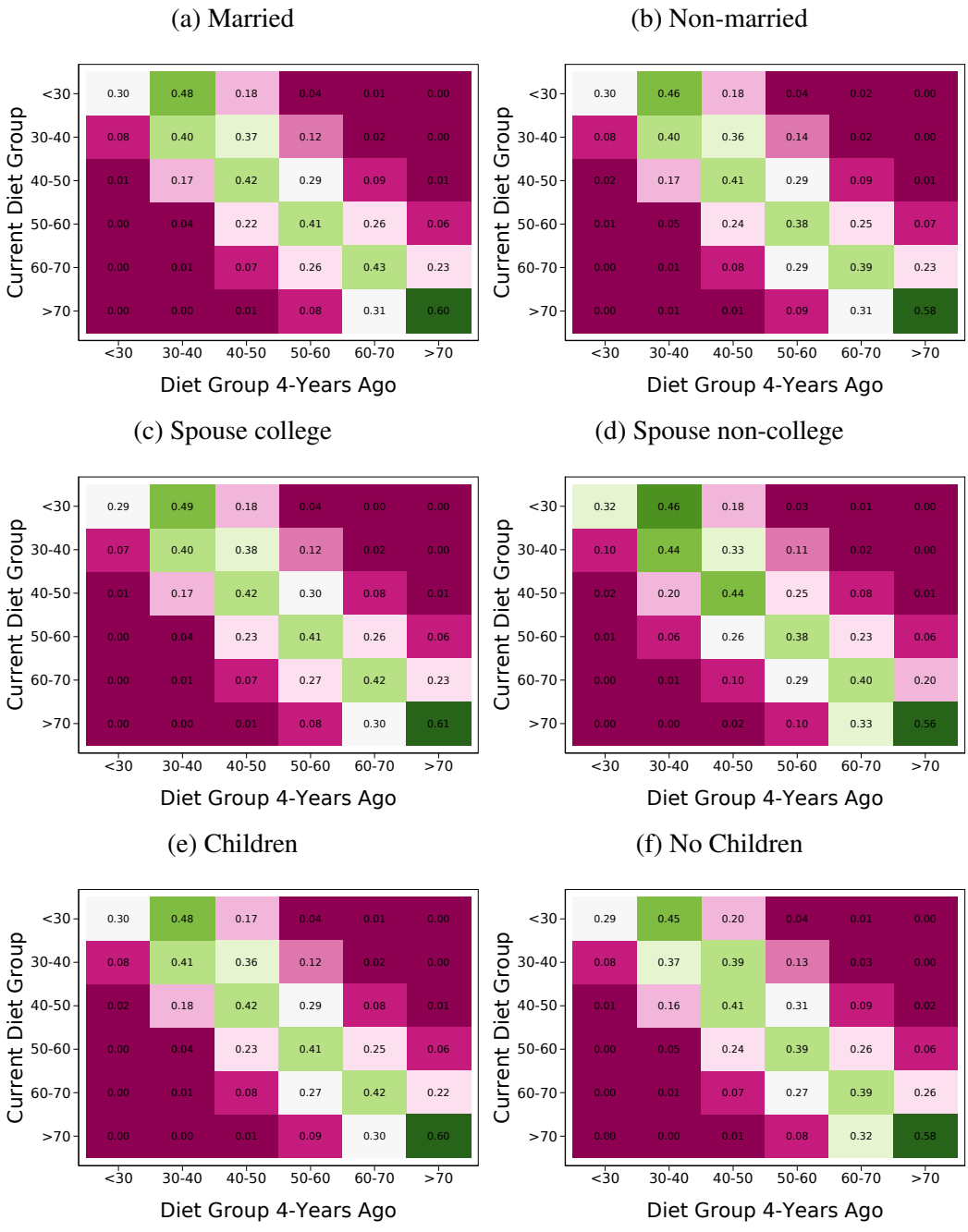


(b) Physical Activity



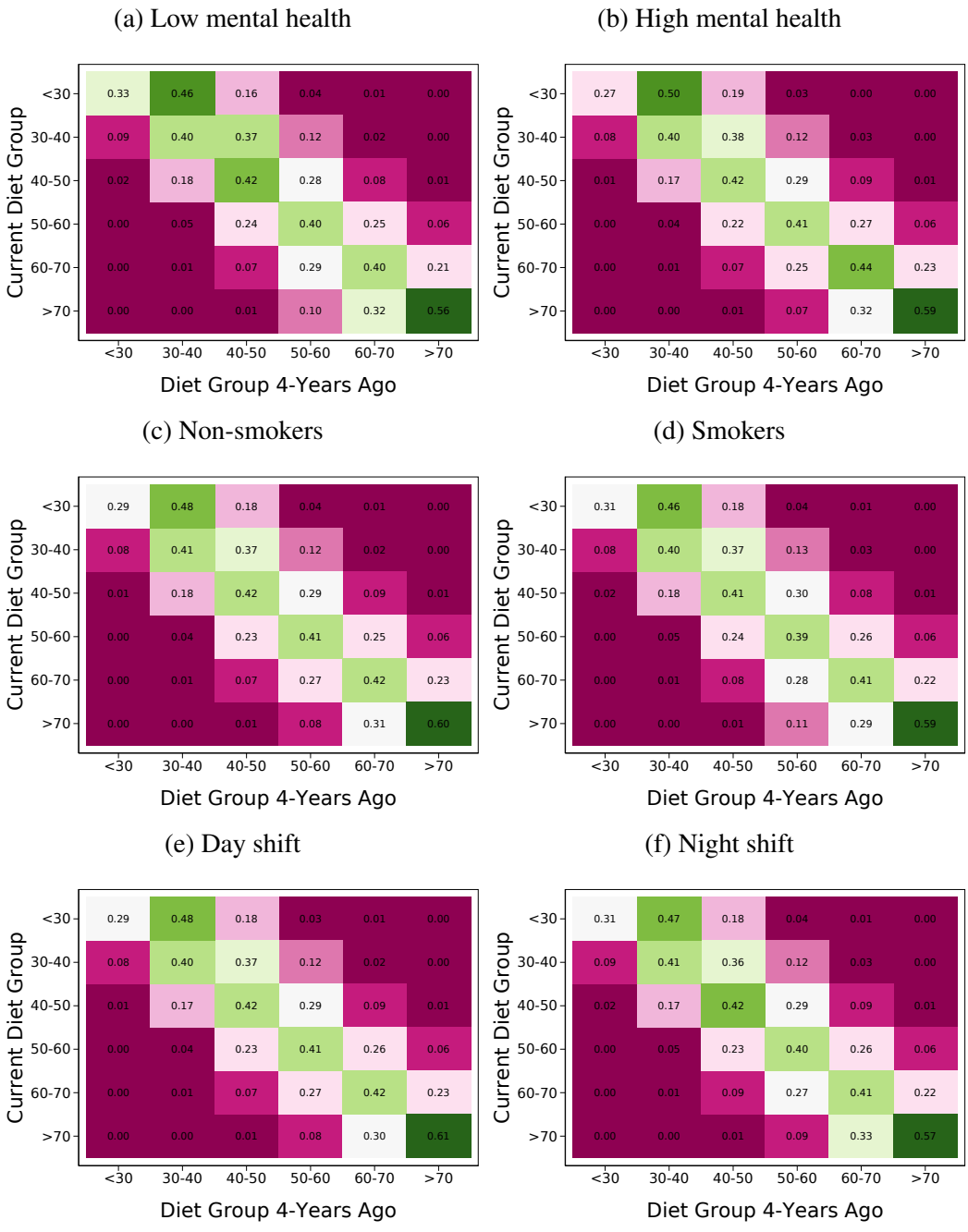
Notes: The figures plot average outcomes for those who obtained at least one morbidity between 1989 and 2015 minus average outcomes for those who maintained a zero morbidity count between 1989 and 2015. Dashed orange lines indicate these differences for those who exclusively worked night shifts at some point between the ages of 20 and 46 and solid green lines are for the rest. NHS II respondents aged 25-30 in 1989 are considered.

Figure S19: Persistence of diet
by marital status, education of spouse, and fertility status



Notes: Figure plots the 4-year transition probability between different diet groups by marital status, education status of spouse, and fertility status. Having children indicate those nurses who had at least one child (measured by pregnancies that resulted in the delivery at $\geq$6 months gestation) and no-children outcomes are for those with no biological children (zero parity). NHS II respondents aged 25-30 in 1989 are considered. Pooled data between 1991 and 2015 is used to construct the 4-year transition probabilities.

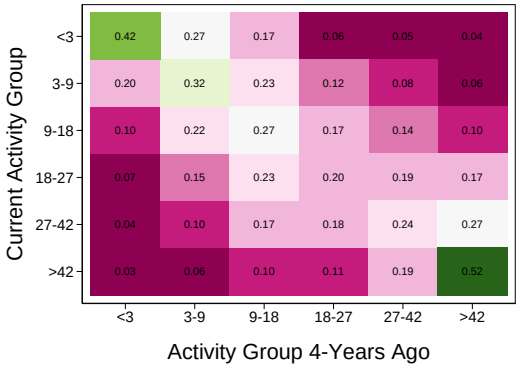
Figure S20: Persistence of diet
by mental health, smoking and working shift status



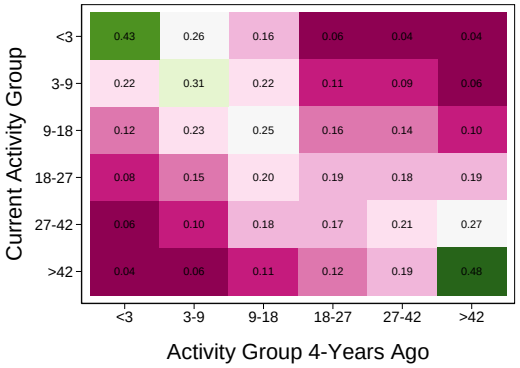
Notes: Figure plots the 4-year transition probability between different diet groups by mental health, smoking status and night shift working status. High mental health refers to those in the highest tercile of the mental health index and low refers to those in lowest tercile. Mental health was measured in 1993 using SF-36 mental health index. Smoking status was measured in 2015 as (1) smoked currently or in past and (2) never smoked. Working shift history was measured in 2009. We categorize night shift nurses as those who exclusively worked night-shifts during some point of their life as discussed in the text. NHS II respondents aged 25-30 in 1989 are considered. Pooled data between 1991 and 2015 is used to construct the 4-year transition probabilities.

Figure S21: Persistence of physical activity
by marital status, education of spouse, and fertility status

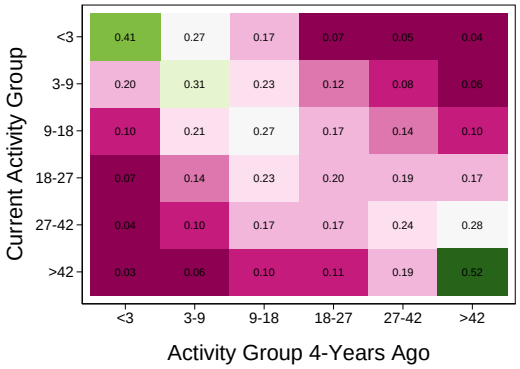
(a) Married



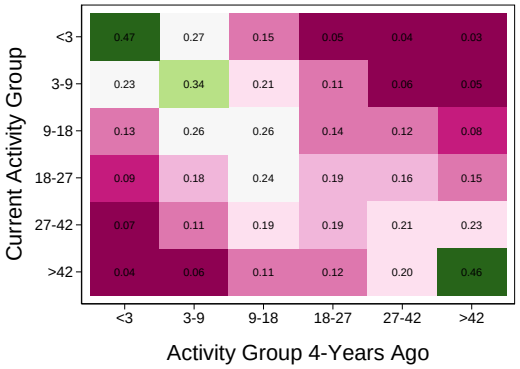
(b) Non-married



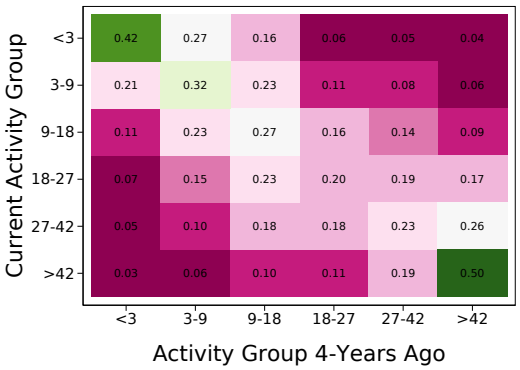
(c) Spouse college



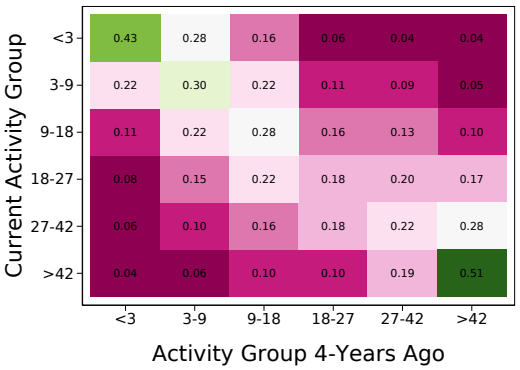
(d) Spouse non-college



(e) Children



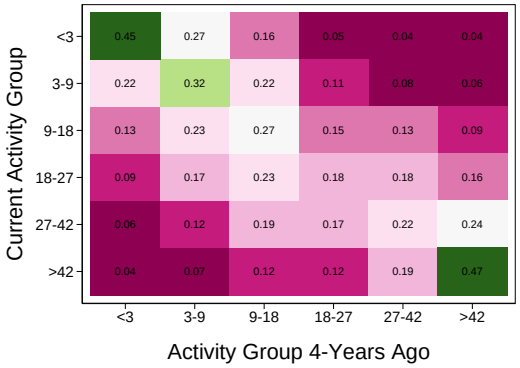
(f) No Children



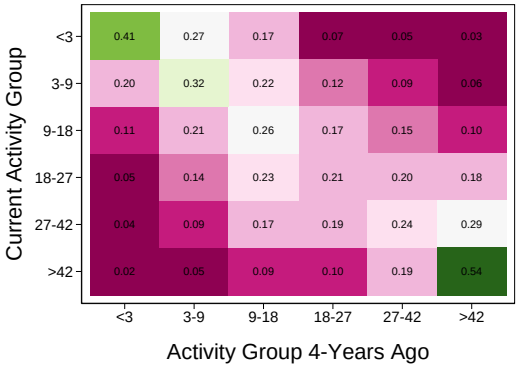
Notes: Figure plots the 4-year transition probability between different physical activity groups by marital status, education status of spouse, and fertility status. Having children indicate those nurses who had at least one child (measured by pregnancies that resulted in the delivery at $\geq$6 months gestation) and no-children outcomes are for those with no biological children (zero parity). NHS II respondents aged 25-30 in 1989 are considered. Pooled data between 1991 and 2015 is used to construct the 4-year transition probabilities.

Figure S22: Persistence of physical activity
by mental health, smoking and working shift status

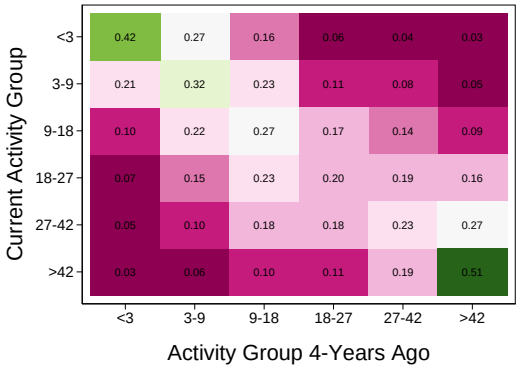
(a) Low mental health



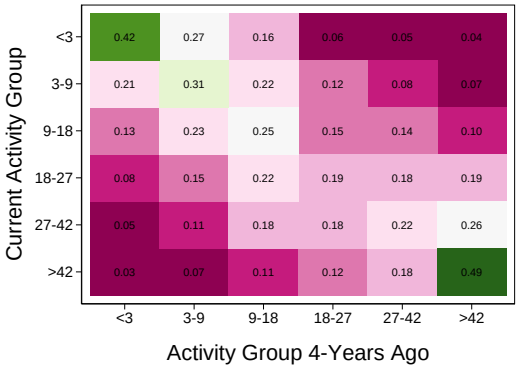
(b) High mental health



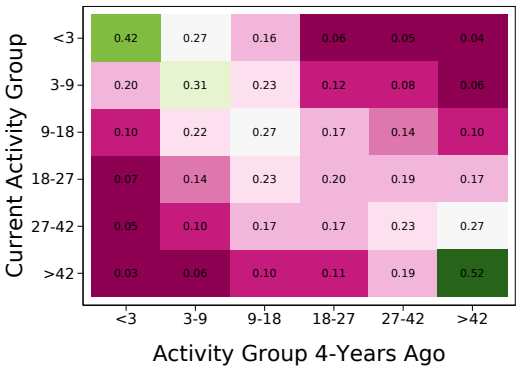
(c) Non-smokers



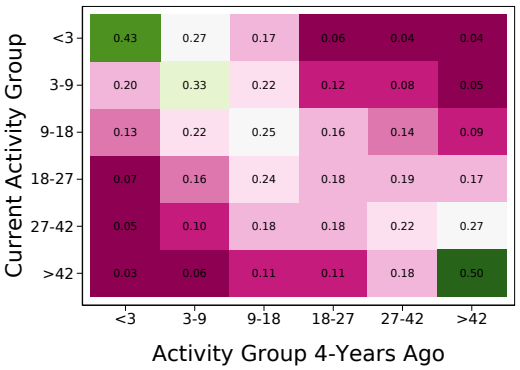
(d) Smokers



(e) Day shift

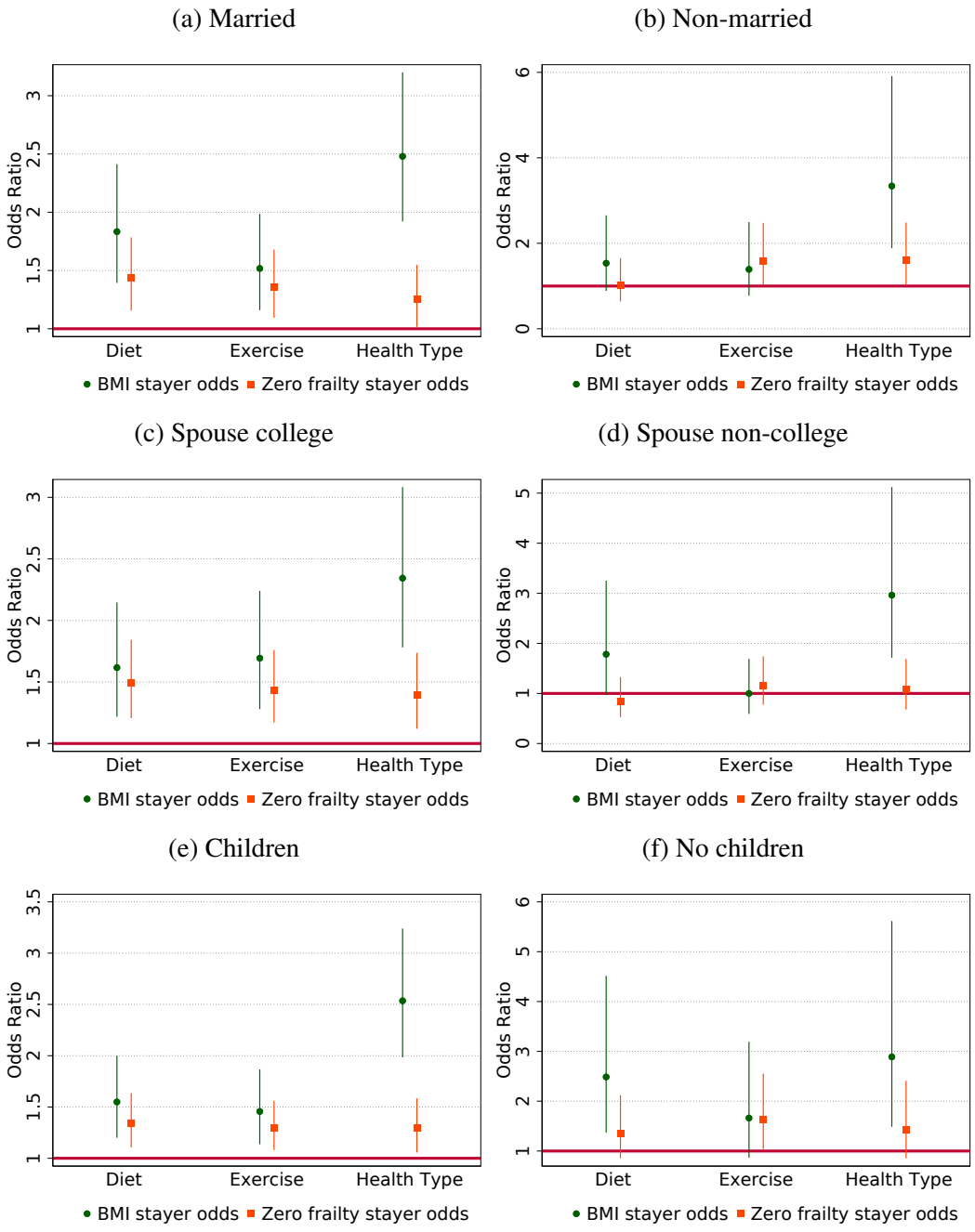


(f) Night shift



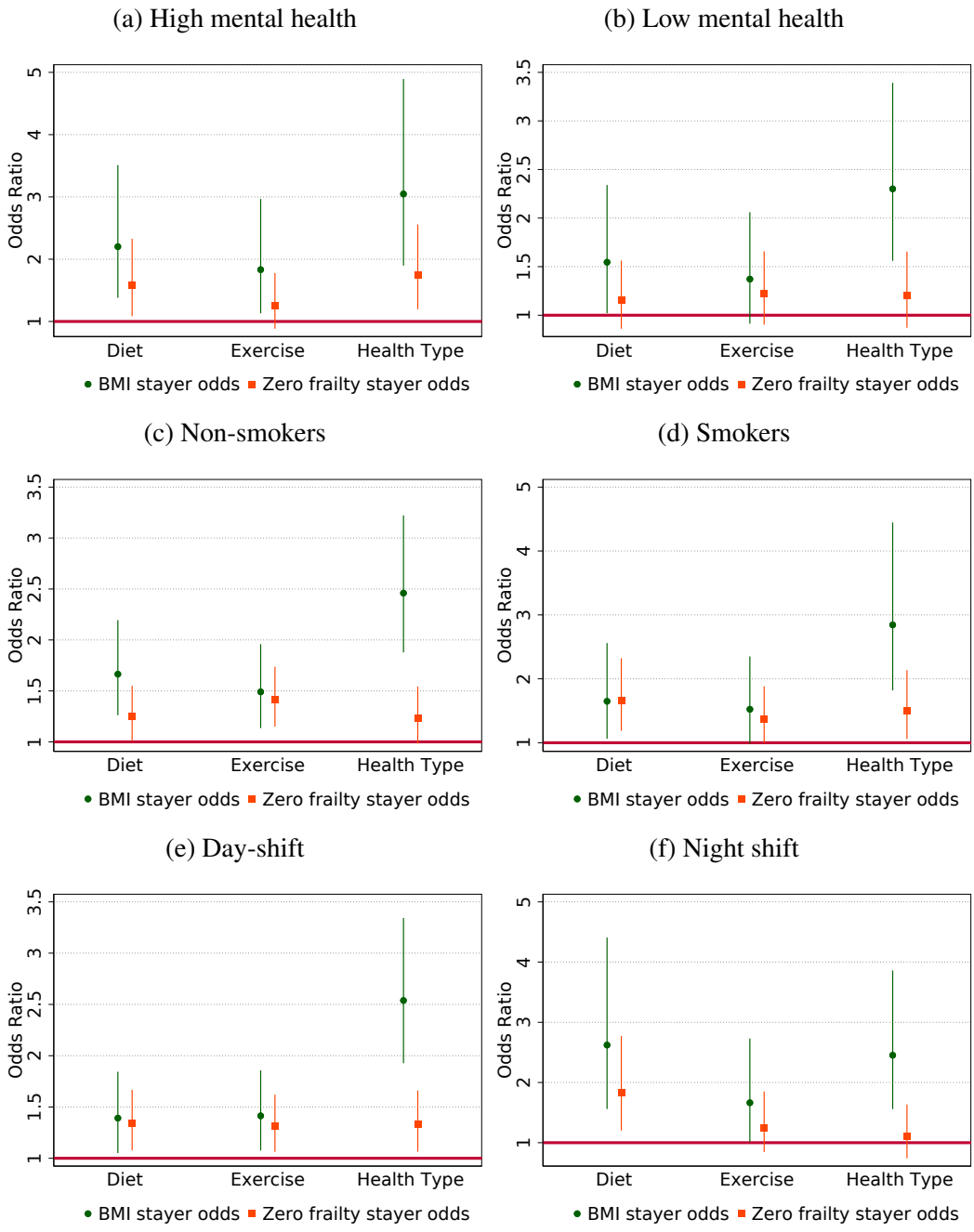
Notes: Figure plots the 4-year transition probability between different physical activity groups by mental health, smoking status and night shift working status. High mental health refers to those in the highest tercile of the mental health index and low refers to those in lowest tercile. Mental health was measured in 1993 using SF-36 mental health index. Smoking status was measured in 2015 as (1) smoked currently or in past and (2) never smoked. Working shift history was measured in 2009. We categorize night shift nurses as those who exclusively worked night-shifts during some point of their life as discussed in the text. NHS II respondents aged 25-30 in 1989 are considered. Pooled data between 1991 and 2015 is used to construct the 4-year transition probabilities.

Figure S23: Role of initial conditions
by marital status, education of spouse, and fertility status



Notes: Sample includes NHS II respondents with healthy BMI and zero morbidities in 1989. The figure plots the odds ratio of being a BMI stayer (healthy BMI in 2015) and morbidity stayer (zero morbidities in 2015) for metabolic types (top vs bottom groups), diet (top vs bottom decile) and exercise (top vs bottom decile) by marital status, education of husband and fertility. Diet and exercise taken from the 1991 wave (roughly age 30). Having children indicate those nurses who had at least one child (measured by pregnancies that resulted in the delivery at $\geq$6 months gestation) and no-children outcomes are for those with no biological children (zero parity).

Figure S24: Role of initial conditions
by mental health, smoking and working shift status



Notes: Sample includes NHS II respondents with healthy BMI and zero morbidities in 1989. The figure plots the odds ratio of being a BMI stayer (healthy BMI in 2015) and morbidity stayer (zero morbidities in 2015) for metabolic types (top vs bottom groups), diet (top vs bottom decile) and exercise (top vs bottom decile) by smoking status and night shift working status. Diet and exercise taken from the 1991 wave (roughly age 30). High mental health refers to those in the highest tercile of the mental health index and low refers to those in lowest tercile. Mental health was measured in 1993 using SF-36 mental health index. Smoking status was measured in 2015 as (1) smoked currently or in past and (2) never smoked. Working shift history was measured in 2009. We categorize night shift nurses as those who exclusively worked night-shifts during some point of their life as discussed in the text.

S3 Stylized facts by residualizing differences in socioeconomic status

As a more parsimonious check of the sensitivity of the stylized facts to socioeconomic differences, we also estimate these facts after residualizing out the influence of observable socioeconomic controls. Specifically, we regress outcomes (BMI, diet, morbidities, and exercise) on martial status, spousal education, average pre-tax household income in the census tract of residence (from 2001), and number of children in the following way:

$$y_{it} = \alpha_0 age_{it} + \alpha_1 \Big(age_{it} \times \mathbb{I}_{married} \Big) + \alpha_2 \Big(age_{it} \times \mathbb{I}_{college} \Big) + \alpha_3 \Big(age_{it} \times \mathbb{I}_{income} \Big) + \alpha_4 \Big(age_{it} \times \mathbb{I}_{children} \Big) + \epsilon_{it}$$

where y_{it} is our outcome of interest (BMI, diet, zero morbidities indicator, and exercise), $\mathbb{I}_x$ represent indicator variable for marital status, education of the spouse, pre-tax household income measured at census tract level, and number of children. We then predict the residual $\hat{\epsilon}$ for each nurse in our sample for each survey round. Finally, we predict outcomes for each nurse in the sample after assigning them the same set of controls——married, college-educated spouse, average pre-tax income, and average number of children. This approach serves to purge any socioeconomic differences across these variables that may be influencing life-cycle patterns in the raw data. Figures S25-S29 plot the stylized facts using these predicted outcomes. The patterns remain very similar to the benchmark results that used raw data without controls. Table S1 below provides our OLS coefficient estimates from the above linear regressions.

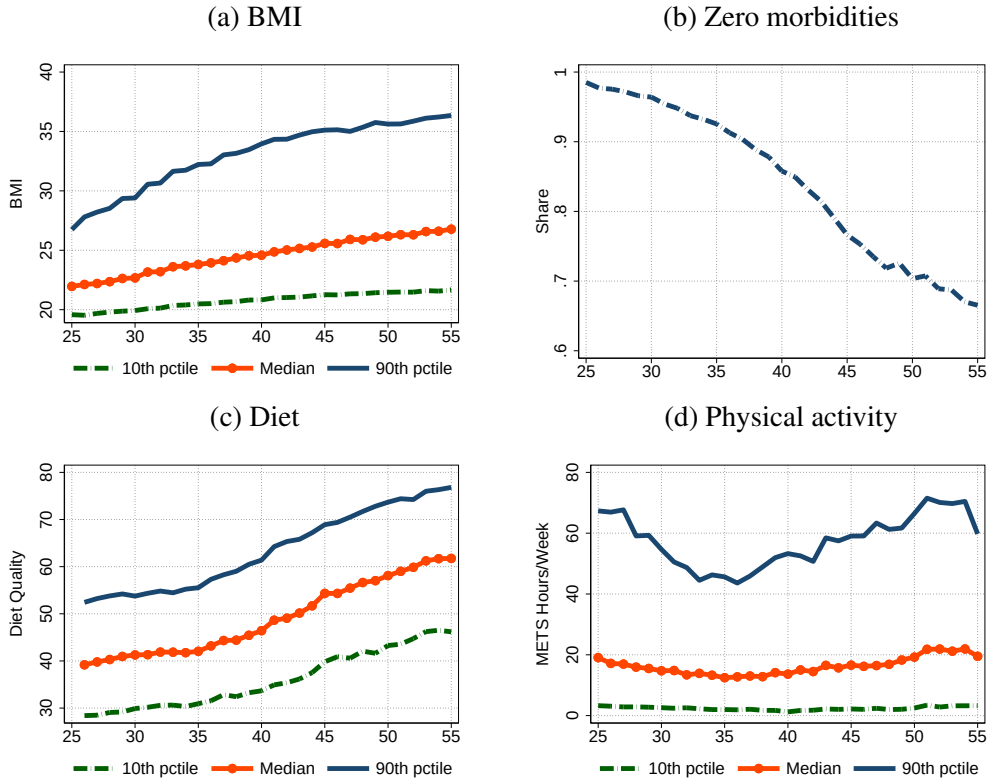
Table S1: OLS Regression Estimates

	Zero morbidity	BMI	Activity	Diet
Age	-0.0324***	0.651***	-1.205***	-0.338***
	(-121.39)	(169.32)	(-41.10)	(-29.40)
College=1 × Age	0.000699***	-0.0226***	0.0774***	0.0559***
	(30.66)	(-68.77)	(31.00)	(57.17)
Married=1 × Age	0.000564***	-0.0188***	-0.00268	0.00313**
	(24.02)	(-55.20)	(-1.04)	(3.07)
Children=1 × Age	-0.0000784*	-0.00124**	-0.0480***	-0.0155***
	(-2.37)	(-2.60)	(-13.29)	(-11.01)
Children=2 × Age	0.000235***	-0.00698***	-0.0629***	-0.0293***
	(8.72)	(-18.03)	(-21.44)	(-25.59)
Children=3 × Age	0.000468***	-0.00749***	-0.0588***	-0.0359***
	(15.73)	(-17.52)	(-18.10)	(-28.33)
children=4 × Age	0.000716***	-0.00698***	-0.0640***	-0.0485***
	(17.21)	(-11.65)	(-14.03)	(-27.07)
Children=5 × Age	0.000743***	-0.00745***	-0.0632***	-0.0622***
	(9.56)	(-6.65)	(-7.41)	(-18.57)
Children=6 × Age	0.00150***	-0.00747***	-0.110***	-0.0546***
	(10.71)	(-3.70)	(-7.17)	(-9.16)
Children=7 × Age	0.00114***	0.0134***	-0.125***	-0.0276*
	(4.31)	(3.51)	(-4.25)	(-2.39)
Children=8 × Age	0.00231***	-0.0187***	-0.0903*	-0.0227
	(6.06)	(-3.45)	(-2.12)	(-1.35)
Children=9 × Age	0.00481***	-0.0489***	-0.211*	-0.0311
	(6.25)	(-4.29)	(-2.30)	(-0.94)
Children=10 × Age	0.00337***	-0.00120	-0.202*	-0.176***
	(4.05)	(-0.10)	(-2.17)	(-4.78)
Children=11 × Age	0.00180*	-0.0259*	-0.258**	-0.0223
	(2.29)	(-2.29)	(-3.10)	(-0.70)
Age × log(income)	0.00134***	-0.0427***	0.111***	0.0906***
	(56.75)	(-125.60)	(42.72)	(89.09)
Constant	1.511***	20.49***	20.21***	18.02***
	(698.71)	(663.78)	(93.58)	(190.25)
Observations	934254	879057	423643	385756

t statistics in parentheses

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

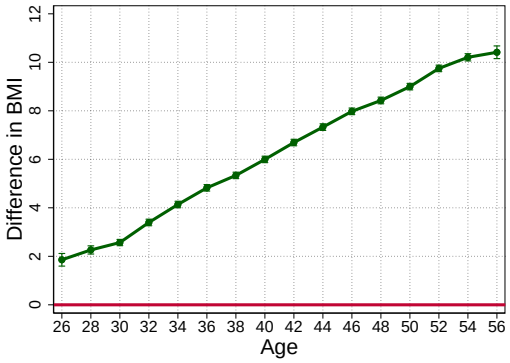
Figure S25: Life-cycle patterns in BMI, morbidities, diet quality, and physical activity



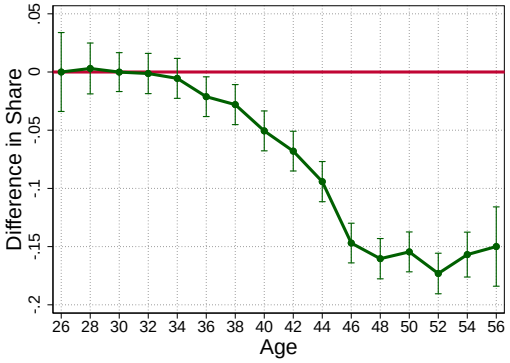
Notes: Panels (a), (c), and (d) plot the distribution (median, 10^{th}, and 90^{th} percentile) of the evolution of BMI, diet quality, and physical activity index, respectively, over the life cycle for the sample of registered nurses in NHS II who were aged 25 to 30 in the initial 1989 wave. For morbidities measure, a total count of morbidities is used. Panel (b) plots the share of those with zero morbidities count by age.

Figure S26: Life-cycle health gaps between BMI gainers and stayers

(a) BMI



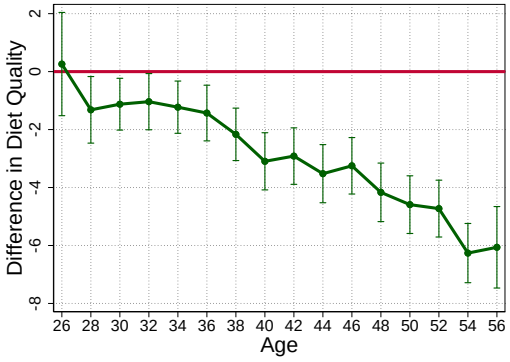
(b) Zero morbidities



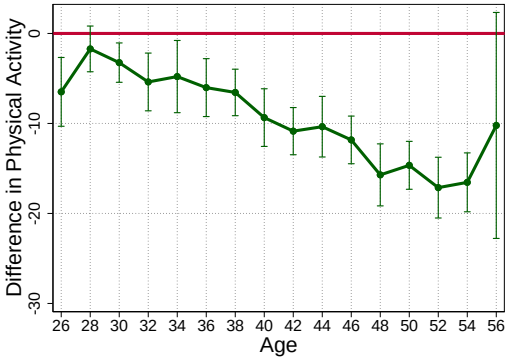
Notes: The figures plot average outcomes for those whose BMI increased from healthy ($18.5-25$) in 1989 to obese ($\geq$ 30) in 2015 minus average outcomes for those who maintained a healthy BMI between 1989 and 2015. Bars denote 95-percent confidence intervals. NHS II respondents aged 25-30 in 1989 are considered, excluding the 3.3% of respondents with non-zero morbidities in 1989.

Figure S27: Life-cycle behavior gaps between BMI gainers and stayers

(a) Diet quality

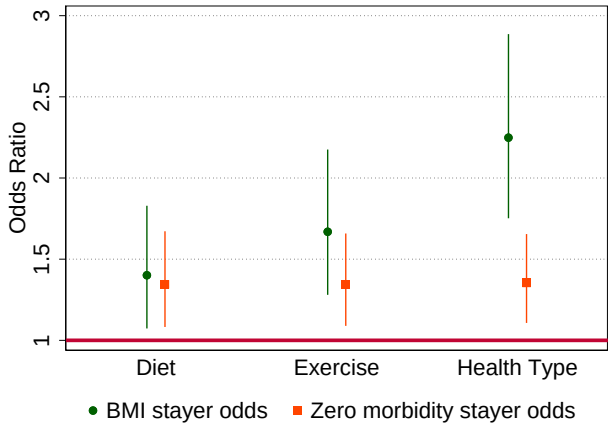


(b) Physical activity



Notes: The figures plot average outcomes for those whose BMI increased from healthy ($18.5-25$) in 1989 to obese ($\geq$ 30) in 2015 minus average outcomes for those who maintained a healthy BMI between 1989 and 2015. Bars denote 95-percent confidence intervals. NHS II respondents aged 25-30 in 1989 are considered.

Figure S28: Role of initial conditions

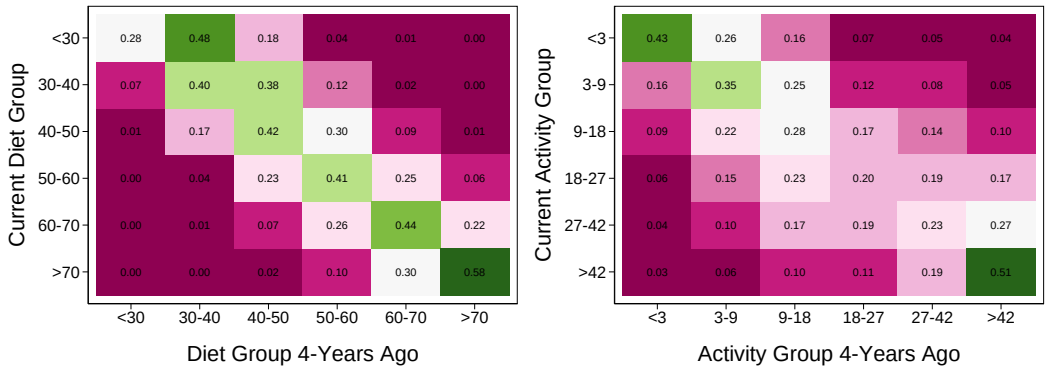


Notes: Sample includes NHS II respondents with healthy BMI and zero morbidities in 1989. The figure plots the odds ratio of being a BMI stayer (healthy BMI in 2015) and morbidity stayer (zero morbidities in 2015) for metabolic types (top vs bottom groups), diet (top vs bottom decile) and exercise (top vs bottom decile). Diet and exercise taken from the 1991 wave (roughly age 30). Bars denote 95-percent confidence interval.

Figure S29: Persistence of diet and physical activity

(a) Diet quality

(b) Physical activity



Notes: Panels (a) and (b) plot the 4-year transition probability between different diet and physical activity groups, respectively. NHS II respondents aged 25-30 in 1989 are considered. Pooled data between 1991 and 2015 is used to construct the 4-year transition probabilities.

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