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HEALTH, ECONOMETRICS AND DATA GROUP

THE UNIVERSITY of York

WP 23/09

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August 2023

http://www.york.ac.uk/economics/postgrad/herc/hedg/wps/

Cognitive Decline and Dynamic Selection*

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November 2022

Abstract

The age profile of cognitive skill decline, and the investments that shape it, are potentially confounded by non-random mortality. I frame this dynamic selection problem in the context of health capital (Grossman, 1972), and I estimate a general model of health investment, health, and mortality. Consistent with theory, I find substantially wider gaps in cognitive health by education, race, and smoking behavior relative to cross-sectional comparisons, in some cases by 100%. Furthermore, these gaps grow in age, which suggests that theories of cognitive decline need to accommodate differential rates of change in cognitive health.

JEL Classification: I10; I12; J24

Keywords: Health Production; Dynamic Selection, Cognitive Skills; Dementia; Tobacco; Disparities

*This work was funded by a Hopkins' Economics of Alzheimer's Disease & Services Center grant. I appreciate valuable comments and suggestions from Edward Norton, John Mullahy, Daniel Polsky, Fabrizio Mazzonna, Andrew Ching, and participants of the Johns Hopkins Carey Business School Economics Seminar. Data and code used in the HRS analysis are available upon request. Code for the NHATS analysis are available upon request, but the data are restricted. Researchers interested in NHATS data may apply for access at https://nhats.org/researcher/nhats.

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1 Introduction

The question of how health (dis)investments affect health in older age is difficult to answer due to the problem of dynamic selection—non-random mortality and sample attrition that causes composition shifts in the distribution of health over the age profile. For example, because cigarette smoking causes premature mortality, it truncates the distribution of health, which generates bias because surviving smokers are relatively healthy. Because of dynamic selection, and because health itself is often difficult to measure, much of the economics literature focuses on either inputs to the health production function, assuming they matter for health, or on the determinants of mortality. This is surprising because the canonical Grossman (1972) framework jointly models both the health production function and the selection equation that dictates mortality. As that model makes clear, understanding the age profile of health has important implications for quality-of-life calculations, optimal treatment patterns, retirement planning, and a variety of other intertemporal problems.

In this paper, I study the dynamic selection problem in the context of cognitive health and cognitive decline among older Americans. Cognitive decline leads to dementia, a socially costly overarching condition that encompasses Alzheimer's disease (Hurd et al., 2013). Between 2015 and 2017, 11.7% of older Americans reported subjective cognitive decline (Taylor et al., 2018)—the admission of worsening memory and/or confusion — which likely understates its prevalence, as recent evidence suggests that many older individuals are unaware of, or in denial about, the state of their own cognition, especially with respect to financial decision-making (Finke et al., 2017; Nicholas et al., 2021; Mazzonna & Peracchi, 2020; Ameriks et al., 2022). Older workers, particularly women, face significant discrimination in hiring, which may be due to statistical discrimination on the part of cognitive skills and abilities (Neumark et al., 2019). Furthermore, the extent to which retirement causes cognitive decline has important policy implications (Fitzpatrick & Moore, 2018; Eibich, 2015; Rose, 2020). While the determinants of cognitive skill *formation* (which investments produce skills and when) in young children have been studied (Cunha & Heckman, 2008; Cunha et al., 2010; Agostinelli & Wiswall, 2020; Attanasio et al., 2020; Caucutt & Lochner, 2020), there is comparatively little work on the determinants of natural cognitive skill decline, partly because dynamic selection makes the

problem more difficult.[1] Understanding cognitive health, its decline, and the determinants of its age profile in later life are important in an aging society.

To address the selection problem, I formulate a simple Grossman model of health investment, cognitive and general health, and mortality. The model reveals that bias in the effect of a health investment or exogenous characteristic on health that is caused by dynamic selection depends on both the correlation between cognitive and general health and the correlation between the health investment or exogenous characteristic and sample exit. The solution to the model yields dynamic investment demand, health outcome, and selective mortality equations, and I estimate these dynamic empirical equations jointly using novel (to the economics literature) longitudinal data from the National Health and Aging Trends Study (NHATS).[2] The intuition is to jointly estimate theoretically founded dynamic equations and to flexibly allow for correlation across equations in the error structure. Doing so allows me to simulate the model under counterfactual scenarios that vary health (dis)investments and exogenous characteristics. Because mortality and attrition are explicitly modeled, simulating the model allows me to characterize the marginal individual and document the bias created by dynamic selection.

I consider three individual characteristics that may affect the age profile of cognitive decline. First, "cognitive reserve" theory (Stern, 2002), which stipulates that education builds reserve that buffers against age-related pathology, was developed, in part, because of epidemiological evidence that the age profiles of cognitive decline across education are different in levels but not slopes (Chapko et al., 2018; Lovden et al., 2020). The economic model of this paper shows that dynamic selection may mask differently sloped age profiles of cognitive health, especially if the well-known gradient between education and expected longevity (Lleras-Muney, 2005; Meara et al., 2008; Buckles et al., 2016; Savelyev, 2022; Lleras-Muney et al., 2022) implies that surviving individuals of lower educational attainment are relatively healthy. Second, I study the White/Black gap in cognitive health. A growing literature recognizes the significantly higher rates of dementia diagnoses for Blacks relative to Whites (Kornblith et al., 2022; Yeo, 2022), which may understate differences in cognitive health if Blacks are less likely to receive

[1] There is a recent literature on skill decline following unemployment (Dinerstein et al., 2022)

[2] See (Cameron & Heckman, 1998; Mroz, 1999; Darden, 2021) for similar empirical models.

a dementia diagnosis conditional on a given cognitive health level (Lin et al., 2021). As with education, Castora-Binkley et al. (2013) finds level, but not slope, differences in cognitive decline by race. Finally, I consider differences in cognitive health by smoking behavior, for which there is significant medical evidence that the vascular effects of smoking may accelerate cognitive decline (Peters et al., 2008; Qiu et al., 2009; Corley et al., 2012; Weuve et al., 2012; Durazzo et al., 2014; Baumgart et al., 2015).

In each case – education, race, and smoking – theory suggests that cross-sectional comparisons between groups should be biased because of dynamic selection as each individual characteristic is linked with mortality and because cognitive and physical health are positively correlated. Indeed, in NHATS data, pooled regressions of word recall scores (i.e., cross-sectional comparisons) suggest that this measure of cognitive health is 8.6% higher for college educated relative to non-college educated respondents; 8.1% lower for Black relative to White respondents; and 2% lower for current cigarette smokers relative to never-smokers. However, simulation of the theoretically founded empirical model suggests that these differences are 17.4%, 18.2%, and 4%.[3] I simulate that the marginal person—one who left the sample via death or attrition because of the respective characteristic—has significantly lower-than-average cognitive scores at a relatively young ages (50s and 60s). As a result, in the context of education and race, their importance in shaping cognitive decline grows in age, which is counter to the theory of cognitive reserve.

Emphasizing the distinction between the outcome of interest (i.e., cognitive health) and the mechanism driving selection (i.e., general health) differentiates this paper from the labor economics and econometrics literature on incidental truncation and selection (Hausman & Wise, 1979; Verbeek & Nijman, 1992; Kyriazidou, 1997; Lillard & Panis, 1998; Zabel, 1998; Ziliak & Kniesner, 1998; Kyriazidou, 2001), whose solution has settled on inverse probability weighting (IPW) (Wooldridge, 2002).[4] In IPW estimation, one estimates a selection equation via a probit model as a function of initial wave characteristics and uses the resulting predicted

[3]The results in HRS data are similar. Word recall scores are a standard proxy for underlying latent cognitive skills and health (Mazzonna & Peracchi, 2020), but in principle a dynamic factor model could be employed (Cunha et al., 2010; Agostinelli & Wiswall, 2020). Because the focus of this paper is dynamic selection, I leave a more general treatment of cognitive skills/health for future work.

[4]In the epidemiology literature, dynamic selection is sometimes referred to as survivorship bias. See Czeisler et al. (2021) for an example of survivorship bias generated by longitudinal panel attrition in the context of mental health and COVID-19.

probabilities to weight the structural outcome equation of interest. The key difference in my approach is to jointly estimate equations for the outcome of interest (i.e., cognitive health) and general health, along with equations for health investment and sample exit. Unlike IPW, the log-likelihood function is non-additive within individuals and correlation in the joint distribution of unobserved heterogeneity is captured in a semi-parametric way via the discrete factor method (Heckman & Singer, 1984; Wooldridge, 2002). Allowing for individual-level heterogeneity proves to really matter, as my simulation results are significantly larger than a) estimating the system separately, and thus not allowing for correlation across equations; and b) in the case of both education and the Black/White gap, results generated from an IPW estimator. While the notion of dynamic selection is separate from the *endogeneity* of health investment, flexibly modeling a dynamic system of equations lends itself to traditional solutions for endogeneity, including instrumental variables and dynamic panel data methods.

This paper also connects to the literature on cognitive skill formation and education. In seminal work, Cameron & Heckman (1998) discuss the potential for dynamic selection associated with modeling determinants (parental investments) of the probability of a student transiting to the next grade level. In that case, as students increasingly leave school over the age profile, the remaining students, and their parents, are selected. The authors find support for dynamic selection as an explanation for the finding that parental investments are less important at higher grades. They use a similar system estimator with unobserved heterogeneity and conclude that "research reporting piecemeal estimates of the schooling process tends to understate the true effects of family background on educational attainment as measured by the coefficients of (separate) logistic transition probabilities." By connecting empirical work with a rational decision-making framework (Grossman, 1972), this paper continues in the spirit of Cameron & Heckman (1998), yet applying this approach to investigate dynamic selection in the context of health and health investment is unique (Contoyannis et al., 2004; Jones et al., 2006).

The dynamic system of equations estimator is flexible, and its potential application to other dynamic selection problems is significant. For example, there exists a long running debate about the effects of health insurance on health (Card et al., 2009; Polsky et al., 2009; McWilliams et al., 2010; Polsky et al., 2010; Miller et al., 2021). In the context of Medicare, if the absence

of health insurance causes premature mortality as the uninsured approach age 65, then those gaining Medicare insurance at 65 will be relatively healthy. Similarly, dynamic selection may be important in the large literature on the health effects of retirement (Fitzpatrick & Moore, 2018; Eibich, 2015; Rose, 2020). Finally, in descriptive work, Darden & Kaestner (2022) study the effects of cigarette smoking on medical care expenditures using linked National Health Interview Survey/Medicare claims data. That paper shows that cross-sectional comparisons of smokers and non-smokers over the age profile may dramatically understate the effects of smoking on medical care expenditures at a given age, but other determinants of medical care expenditures may suffer from similar dynamic selection problems.

The rest of the paper proceeds as follows. In Section 2, I provide a simple Grossman-type framework that characterizes the selection problem. The model neatly emits estimating equations that can be taken to data, and Section 3 explains how to estimate the model via full-information maximum likelihood. Section 4 presents summary statistics from both the NHATS and the HRS. The section also demonstrates suggestive evidence of dynamic selection, and it documents how data are cleaned to feed into the dynamic empirical model. Section 5 presents estimates of the dynamic system of equations model along with simulation results on model fit and the average treatment effect on the treated estimates. Section 6 provides a discussion and concludes.

2 Theory

In this section I formalize a theory of cognitive decline and its determinants. The theory builds from a stylized Grossman (1972) model of health production in that it captures endogenous investments in health, the evolution of health over the life cycle, and the endogenous nature of death. I partition health into general health, which, as in Grossman (1972), determines death when health falls below a given value, and cognitive health, which is potentially correlated with general health and is valuable in a consumption sense. A richer model would include an investment rationale for cognitive health, but such a rationale is not needed to demonstrate the importance of dynamic selection.[5] The object of interest is the impact of a given investment

[5]Halliday et al. (2019) suggest health is predominantly a consumption good after the age of 62 and that a consumption motive for health investment is needed to explain the increase in health care expenditures

on the age profile of cognitive health. The model demonstrates how selective death causes an identification problem when comparing cognitive health across individuals with different levels of investment. At a given age, mean cognitive health is conditional on having survived to that age, and different patterns of investment generate different compositions of both cognitive and general health. The model demonstrates that the degree of dynamic selection depends on the correlation between cognitive and general health and on the extent to which (dis)investment causes changes in general health.

The model is of a cohort of individuals over the age profile. At age a, an individual observes their state vector, which includes health partitioned into general health H_a and cognitive health C_a. Conditional on these health values, the individual selects the optimal level of a vector of health investments I_a, the components of which may be specific to general and/or cognitive health. Conditional on these investments and the individual's state, general and cognitive health evolve to their age $a+1$ values. The individual survives ($\omega_{a+1} = 0$) to age $a+1$ if $H_{a+1} > \overline{H}$, and their optimization problem can be represented by the familiar Bellman equation:

$$V(H_a, C_a) = \max_{I_a} \Big[U(X_a, I_a; C_a, H_a) + \beta P(H_{a+1} > \overline{H}) EV(H_{a+1}, C_{a+1}) \Big], \tag{1}$$

where the expectation operator is taken over future general and cognitive health shocks.

In this model, utility is defined over general consumption X_a and investment I_a, which enters directly because some investments generate utility in unique ways (e.g., smoking and addiction). Both general health and cognitive health are allowed to shift the marginal utilities of consumption and investment (Finkelstein et al., 2012). Direct utility is constrained by a standard static budget constraint without borrowing or saving: $X_a = W_a - p_a I_a$, where income is given as W_a and p_a is the relevant investment price vector at age a. The second part of Equation 1 captures the present discounted expected value of life conditional on survival, where the probability of survival is dependent on health. The evolution equations for general

later in life.

and cognitive health are given, respectively, as follows:

$$H_{a+1} = \Big(1 - d^h(I_a, a+1)\Big)H_a + \epsilon^H_{a+1}, \tag{2}$$

and

$$C_{a+1} = \Big(1 - d^c(I_a, a+1)\Big)C_a + \epsilon^C_{a+1}. \tag{3}$$

The investment vector potentially shifts the respective rates of depreciation $d^{\cdot}$ on each form of health.[6] Depreciation is also a function of age, which captures natural aging and guarantees "optimal" death. Optimal health investment solves the familiar first-order condition in which the marginal benefit of investment equals the marginal cost. Included in the first-order condition is the change in both the probability of survival conditional on a given expected value of life and the change in the expected value of life conditional on a given probability of survival. Thus, both $d^h_{I_a}$ and $d^c_{I_a}$ are of empirical interest.

The main goal of this paper is to produce better estimates of $d^c_{I_a}$, the change in the rate of depreciation of cognitive health for a change in health investment, across the age profile. For simplicity, consider a scalar and binary investment such that $I \in \{0, 1\}$. According to the timing of the model, the comparison that sheds light on $d^c_{I=1} - d^c_{I=0}$ is between the cognitive health at age $a+1$ with and without investment (i.e., $C_{a+1|I_a=1}$- $C_{a+1|I_a=0}$), which nicely resembles the potential outcomes framework. However, the model makes clear that any comparison of cognitive health across investment groups at age $a+1$ requires individuals to *survive* to age $a+1$ such that C_{a+1} can be observed. To build intuition about the role of dynamic selection, I focus on the $\epsilon \in \{\epsilon^h_{a+1}, \epsilon^c_{a+1}\}$ terms, which generate individual-level heterogeneity in their respective health types. For the theoretical presentation of the model, I assume that the joint distribution of ϵ is bivariate normal, with zero mean and correlation coefficient ρ. Under this assumption, the problem is one of incidental truncation (Greene & Zhang, 2003), and the difference in mean cognitive health at age $a+1$ between those with $I_a = 1$ and $I_a = 0$ is given as

$$\underbrace{E(C_{a+1}|H_{a+1} > \overline{H}, I_a = 1) - E(C_{a+1}|H_{a+1} > \overline{H}, I_a = 0)}_{\text{Difference in Means at Age } a+1} = \underbrace{\Big(\mu_{c,I=1} - \mu_{c,I=0}\Big)}_{\text{ATT}} + \underbrace{\rho\sigma_c\Big(\frac{\phi(\alpha)}{1-\Phi(\alpha)} - \frac{\phi(\gamma)}{1-\Phi(\gamma)}\Big)}_{\text{Dynamic Selection Effect}}. \tag{4}$$

[6] I abstract away from questions regarding competing risk mechanisms with disease-specific investments. In this model, it is sufficient to think of investment as potentially having an impact on each form of health.

The left-hand side of the equation is the difference in the expected cognitive health conditional on survival across investment categories, which is observable in both cross-sectional and longitudinal data of cognitive health and investment. The right-hand side includes the average treatment effect on the treated (ATT) of investment on cognitive health at age a $\left(\mu_{c,I=1} - \mu_{c,I=0}\right)$ and the dynamic selection effect, where ρ is the correlation between general and cognitive health, σ_c is the standard deviation of cognitive health, and the remaining term is the difference in the inverse Mills ratios across investment groups relative to the threshold for death. Here, $\alpha = \frac{\overline{H} - \mu_{H,I=1}}{\sigma_H}$ and $\gamma = \frac{\overline{H} - \mu_{H,I=0}}{\sigma_H}$. While this difference is in "units" of health, the $\rho\sigma_c$ term coverts it to cognitive health units.

The model, and Equation 4 in particular, identifies a number of challenges for empirical work. First, at a given age, cross-sectional comparisons of cognitive health across different levels of investment will be confounded by dynamic selection if cognitive health and general health are correlated (i.e., $\rho \neq 0$) and if investment has a non-zero effect on mortality (i.e., $d_I^h \neq 0$). Second, while the dynamic selection effect is intuitive, its importance will vary over the age profile. The difference in the inverse Mills ratio reveals the magnitude of the margin of death due to investment, which depends on the means of health at different levels of investment. For example, the smoking literature suggests that the survival curves of smokers and non-smokers are roughly identical up to age 50, at which point they depart significantly (Doll et al., 2004; Darden et al., 2018). Thus, we should not expect the impact of smoking on cognitive health to be affected by dynamic selection (as implied by death) before age 50. As mean health declines naturally (due to aging), and as smoking causes premature mortality, the dynamic selective effect grows in importance to the extent that cognitive and general health are correlated. Particularly in the case of smoking, the dynamic effect of health on investment highlights the importance of modeling investment dynamics jointly with health dynamics.

The comparison in Equation 4 is not limited to endogenous health investment. In fact, similar comparisons can be made on the basis of any individual characteristic (i.e., exogenous or pre-determined characteristics), including those that are time invariant. Because the difference in means on the left side of Equation 4 is at a given age, the dynamic selection bias results from a cross-sectional comparison that is conditional on a dynamic process (i.e., health). Thus, researchers studying cross-sectional data cannot solve the selection problem because such data

do not reveal information on the path that led to being observed at a given age. However, longitudinal data are also problematic because, in addition to dynamic selection generated by death, attrition from longitudinal panels generates a second source of dynamic selection. Furthermore, with the exception of life-cycle longitudinal studies (e.g., the Framingham Heart Study), longitudinal surveys will suffer from an initial conditions problem. In what follows, I present an empirical model that is consistent with the theoretical model presented above and one that may be estimated with standard (i.e., NHATS and HRS) longitudinal surveys.

3 Econometrics

I propose a "quasi-structural" empirical approach that approximates the model presented above. In principle, the solution to the theoretical model yields demand (for investments) and outcome (health) equations. My strategy is to structure longitudinal data on cognitive health, general health, and health investments according to the model timing and to estimate these equations jointly while allowing for flexible correlation in the joint distribution of the error structure governing each equation. Because most longitudinal data of individuals over time include significant variation in age at the initial wave, I change notation in the empirical model to t, to indicate wave, although I argue that long panels of individuals over time, with variation in initial ages, may sufficiently capture the age profile. For each equation, I decompose the error structure into a permanent component and an i.i.d. term, and I allow the permanent components to be correlated across equations. Rather than assuming joint normality of the permanent components, I discretize their joint distribution and estimate mass points that are allowed to differ by equation. The error structure is designed to allow for types of individuals with certain, permanent patterns. I close the empirical model by allowing the initial conditions—initial wave cognitive and general health and health investments—to affect the *probability* of each type.

The solution of the dynamic model yields a demand equation for health investment:

$$I_{it} = f(I_{it-1}, H_{it}, C_{it}, Z_{it}, \nu^I_{it}), \tag{5}$$

where investment demand I_{it} for individual i at panel wave t is a function of lagged investment I_{it-1}; an individual's health state, H_{it} and C_{it}; exogenous characteristics Z_{it}; and unobserved, individual-level heterogeneity ν_{it}. Next, as a function of investment I_{it}, both general and cognitive health evolve:

$$h_{it+1} = h(h_{it}, I_{it}, C_{it}, Z_{it}, \nu_{it}^{H}), \tag{6}$$

and

$$c_{it+1} = h(h_{it}, I_{it}, C_{it}, Z_{it}, \nu_{it}^{C}). \tag{7}$$

And, finally, the binary selection equation dictates the probability that individual i exits as a function of both investment and health:

$$P(\omega_{it+1} = 1) = f(I_{it}, H_{it+1}, C_{it+1}, Z_{it}) + \nu_{it}^{\omega}. \tag{8}$$

In practice, I separate Equation 8 into separate equations for sample exit due to attrition and death.

As noted in Cameron & Heckman (1998), the non-linear nature of Equation 8 places significant emphasis on the distributional assumption made on ν, and joint estimation of Equations 5–8 is feasible only after making assumptions about the error structure $\{\nu^{I}, \nu^{C}, \nu^{H}, \nu^{\omega}\}$. To proceed, I decompose the structure of unobserved heterogeneity,

$$\nu_{it}^{\cdot} = \mu_{it}^{\cdot} + \epsilon_{it}^{\cdot}, \tag{9}$$

and I assume that $\mu_{it}^{\cdot} = \mu_{i}^{\cdot}$ in all equations such that μ represents time-invariant unobserved heterogeneity. Rather than making a parametric assumption on the joint distribution of the μ terms, I propose a discrete factor approach (Heckman & Singer, 1984), which uses a step function to approximate the joint distribution. This discrete factor random effects (DFRE) method allows for K unobserved types of individuals, where the researcher estimates both the points of support and the associated probabilities. The idea is to let $\mu_{i}^{\cdot}$ take one of K values $\{\mu_{i1}^{\cdot}, \ldots, \mu_{iK}^{\cdot}\}$ in each of the equations in the model. The probabilities of each type can be estimated subject to the normalization that $\mu_{i1}^{I} = \mu_{i1}^{C} = \mu_{i1}^{H} = \mu_{i1}^{\omega} = 0$. For example, if within-individual patterns in the data are consistent with a fraction of individuals for whom

a certain type of health investment is uncommon and excess mortality is common, then that type may yield a significantly negative value of μ_{ik}^I and a significantly positive value of μ_{ik}^ω.

In general, selective exits from a sample will cause μ_i and exogenous characteristics Z_{it} to be correlated over time because, for example, high values of μ may compensate for low values of Z in the mortality equation. The assumption that is needed is that μ_i and the initial condition of Z_t (i.e., $Z_{t=ic}$) are uncorrelated conditional on the endogenous variables at the initial condition. Following Keane & Wolpin (1997), one can model the probability of a given type k as a function of initial conditions with a logit specification:

$$\tau_k = ln\Big(\frac{P(\mu_i = \mu^k)}{P(\mu_i = \mu^1)}\Big) = \psi_0^k + \psi_1^k I_{i,t=ic} + \psi_2^k H_{i,t=ic} + \psi_3^k C_{i,t=ic}, \tag{10}$$

where $t = ic$ represents the initial wave from which the model occurs. In practice, because individuals enter typically enter longitudinal surveys at different ages, Equation 10 can be augmented to allow the initial age at the first wave of a panel to potentially affect the probability of each type.

To formulate a likelihood function, assumptions are required on the ϵ terms. For simplicity, and recognizing that the joint distribution of μ already captures cross-equation heterogeneity, I assume that both ϵ_{it}^H and ϵ_{it}^C are i.i.d. across equations and time and normally distributed. Furthermore, I consider a scalar, binary health investment such that $I \in \{0, 1\}$. Under these assumptions, the resulting likelihood function contribution from individual i is

$$L_i(\Theta) = \sum_{k=1}^{K} \tau_k \Bigg\{ \prod_{t=ic+1}^{T(i)} \Big\{ \prod_{I=0}^{1} P(I_t = I|\mu_{ik}^I)^{1[I_t=I]} \phi(h_t - (\hat{h}_t|\mu_{ik}^H)) \phi(c_t - (\hat{c}_t|\mu_{ik}^C)) \prod_{\omega=0}^{1} P(\omega_t = \omega|\mu_{ik}^\omega)^{1[\omega_t=\omega]} \Big\} \Bigg\}. \tag{11}$$

Equation 11 says that the likelihood contribution from individual i, as a function of the parameters to be estimated, Θ, is the weighted sum of likelihood contributions over the K discrete mass points, with weights given in Equation 10, where each probability or density is conditional on type μ_{ik}. Notice that the time horizon, $T(i)$ is dependent on individual i in the age product. The log-likelihood function can then be calculated over individuals: $L = \sum_{i=1}^{n} ln(L_i(\Theta))$.

3.1 Identification

Equation 4 highlights the identification problem with cross-sectional comparisons on the basis of health investments - the average treatment effect on the treated is confounded by the dynamic selection effect. The standard approach taken in the economics literature is to estimate some variant of Equation 7 in a single-equation model of the outcome of interest. The standard parameterization of this regression is:

$$C_{it+1} = \alpha_0 + \alpha_1 C_{it} + \alpha_2 I_{it} + \alpha_3 H_{it} + \alpha_4 Z_{it} + \epsilon_{it}, \tag{12}$$

where researchers study within-individual variation (either through individual fixed effects or through a dynamic model as presented here).

Wooldridge (2002) proposes a solution to the dynamic selection problem in which the researcher first estimates the probability of sample exit at each wave as a function of initial wave observable characteristics. The probabilities generated from this first stage probit equation then form weights for observations entering the structural outcome equation of interest (i.e. Equation 12). The inverse probability weighting (IPW) estimator is quite flexible and widely used. However, it is inconsistent with the theory in Section 2 in that it ignores correlation between the outcome of interest and the mechanism behind sample exits, in this case, the correlation between cognitive and general health, ρ. Theory suggests that H_{it} is endogenous in the model of cognitive health and cognitive health investments, so controlling for H_{it} while not explicitly modeling its dynamics and failing to allow for correlation between general health and cognitive health may generate biased estimates of the relationship between I_{it} and C_{it+1}.

Equations 5-11 provide a flexible way for researchers to model a wide variety of simultaneously chosen disease-specific and general health investments and subsequent health outcomes while controlling for dynamic selection. While the traditional Grossman (1972) framework focuses on endogenous health investments (e.g., medical care), the empirical model above can easily illuminate the impacts of exogenous characteristics on health. In the case of education, most research on the effect of education on health uses compulsory schooling laws for plausibly exogenous variation in educational attainment, but they focus on mortality as the health outcome (Lleras-Muney, 2005). Given the focus on cognitive health in later life, I assume that

education is pre-determined conditional on the factor μ; and similarly, I assume that race is exogenous. Under these assumptions the ATT in Equation 4 takes a causal interpretation. Cigarette smoking is a natural endogenous health investment a la Grossman (1972), and I include tobacco control laws in the smoking equation as natural exclusion restrictions - variables that only affect health outcomes through their effects on smoking behavior.

The system of dynamic equations model makes a number of simplifying assumptions. Most notably, correlated unobserved heterogeneity is assumed to be time-invariant and separable within each equation, and the time-varying shocks (i.e., the ϵ terms) are assumed to be i.i.d. over equations and time. These assumptions preclude dynamic unobserved heterogeneity and random coefficients. Additionally, all dynamics are assumed to work through one-period lags of other endogenous variables, which precludes richer lag structures or other capital stocks (e.g., addictive capital in the case of smoking). These assumptions — on the structure of the empirical model and the treatment of each individual characteristic — are maintained throughout the paper. The idea is to contrast ubiquitous, single equation estimators (e.g., IPW), which make equally strong assumptions, but which fail to account for the endogeneity of the selection mechanism (i.e., general health), with a simple dynamic system of health investments, health, and selective exits. Importantly, the empirical model is sufficiently flexible to relax these assumptions, and, in the case that the relevant tradeoff does not include intertemporal tradeoffs, the model allows for *ex ante* policy evaluation that is typically only possible in fully structural models.

4 Data

This section presents summary statistics and institutional details from both the National Health and Aging Trends Study (NHATS) and the Health and Retirement Study (HRS). Both data sources are nationally representative, but they emphasize different time periods and different parts of the age distribution. The NHATS sample, which began in 2011, is older by roughly 10 years, on average, so while selection into the NHATS may generate more of an initial conditions problem, it offers a much richer picture of individuals in their late 70s and 80s relative to the HRS. Meanwhile, because HRS waves are biennial (after 1996), 11 waves of

HRS data allow me to study individuals for over 20 years, providing a richer view of dynamic selection. Throughout the paper, I treat these data sources separately. Appendix Table 1 provides details on the sample construction for each panel.

The theory and empirical models presented above require consistently measured, continuous representations of cognitive and general health. Cognitive ability and cognitive health are not necessarily the same thing, but in clinical settings for the elderly, their measurement is strikingly similar.[7] In both data sources, respondents receive a cognitive skills assessment exam in which one component was a word recall test. After hearing a panel of 10 words, an individual was asked to recite as many words as they could remember (immediate recall); after five minutes, they were asked again to recite as many words as they could remember (delayed recall). Memory loss, particularly immediate recall, is a leading indicator of dementia and cognitive decline, and such tests are widely used to evaluate cognitive health. In this paper, I sum the immediate and delayed word recall scores to form a word recall measure that ranges from 0 to 20 words. This measure has the appeal of being relatively continuous and easily interpretable. A more general treatment of cognitive health would be to estimate the factor structure of several measures as they relate to latent cognitive health (Cunha et al., 2010), including observable measures of basic knowledge (e.g., correctly naming the President of the United States). However, for the purposes of documenting dynamic selection in the determinants of cognitive health and ability, word recall is sufficient. Similarly, I use the count of chronic conditions that an individual reports at a given wave as a quasi-continuous measure of general health.[8]

4.1 The National Health and Aging Trends Study

The NHATS began in 2011 as a nationally representative, longitudinal survey of Medicare beneficiaries aged 65 or older. The initial sampling procedure drew from Medicare enrollment files to produce a sample of 12,411 individuals, which over-sampled older and Black Americans. Subjects proceeded to receive annual interviews going forward, with information on physical,

[7] National Institute on Aging defines dementia as the "loss of cognitive functioning - thinking, remembering, and reasoning - to such an extent that it interferes with a person's daily life and activities."https://www.nia.nih.gov/health/what-is-dementia.

[8] Appendix Table 2 lists the conditions that contribute to general health in each data source.

mental, and emotional health, activities of daily living, the care environment, assets and socioeconomic characteristics, and interactions with the medical system. Because respondents were lost to attrition and death, in 2015, the sample was replenished to maintain the original sample sizes by age and race.

I use nine waves (2011–2019) on the original 2011 cohort. Of the 12,411 sampled individuals in 2011, 8,245 proceeded to complete wave 1. Of those 8,245, I consider a sample of 5,750 whose records included complete information on cognitive health, physical health, education, race, smoking behavior in all interviews in which the person remained alive and in the sample. The data are geocoded such that I am able to merge state of residence-by-year information on local area tobacco prices, consumption per capita, and tax revenue to the panel.[9] The data provide rich information on behavior and health at older ages of a recent cohort. To the best of my knowledge, this paper is the first in the economics literature to study these data.[10]

4.2 Health and Retirement Study

The data from NHATS nicely complement the more widely used panel information from the HRS, which is a household-level panel study that began in 1992 to address "important questions about the challenges and opportunities of aging." Like many researchers, I use data from the RAND HRS Longitudinal File, which has standardized many of the HRS data items across waves, including many of the derived variables such as cognitive health scores and chronic health conditions. Before 1996, the word recall questions used to proxy for cognitive health used 20-word panels. As a result, I use data starting in 1996, when HRS administered more standard 10-word recall panels, from both HRS and Study of Assets and Health Dynamics (AHEAD) cohorts for up to 11 waves through 2016. Respondents were interviewed every two years. At the 1996 wave, I have information on 12,402 individuals in the RAND file, and I consider longitudinal data on 11,262 of them, keeping those with complete records.[11]

[9] Local area characteristics on tobacco consumption and prices come from Orzechowski and Walker: The Tax Burden, 2019.

[10] More information is available at National Health and Aging Trends Study.

[11] See Appendix Table 1 for sample construction details.

4.3 Sample Statistics

Table 1 provides initial wave (wave 1 in the case of the NHATS, wave 3 in the case of the HRS) summary statistics for both the NHATS (2011) and the HRS (1996) samples. The top row of Table 1 presents the mean of the summed (immediate and delayed) word recall tests at wave 1 for the overall sample of each data source as well as for college graduates, Blacks, and current smokers. In general, HRS respondents have higher word recall scores overall (10.118 versus 8.140), and the patterns across data sources by education, race, and smoking status are similar — those with a college degree have higher word recall scores while Blacks have significantly lower word recall scores. Smokers have slightly higher word recall scores in both data sources, which itself is suggestive of dynamic selection.

To help explain differences in cognitive ability, Table 1 also reports the number of "chronic conditions" with which an individual has ever been diagnosed. Because different conditions contribute to the chronic condition count in each data source, they are not directly comparable across sources, but within each data source, college graduates have fewer chronic conditions and Black respondents had more; smokers have more conditions in the NHATS and fewer in the HRS, which again is suggestive of dynamic selection in that the smokers observed smoking at later ages must be relatively healthy to have not experienced premature mortality. At wave 1, 8.2% of NHATS respondents reported currently smoking, while 44.3% of NHATS respondents reported smoking in the past. Those statistics for the HRS (wave 3) are 17.0% and 25.3%, respectively.

The percentages of Black respondents, which is coded as any participant who identifies as "Black, non-Hispanic," are 21% and 13.1% for the NHATS and the HRS, respectively. The distributions of education are roughly similar between the two cohorts, with about 22.5% and 16.8% of respondents claiming a college degree or higher. The mean age in the NHATS sample at wave 1 was 76.4, whereas the mean age in the HRS was 66.2, and given the length of the panels and the variance in ages at wave 1, the differences in mean age make studying both the NHATS and the HRS appealing to get a wider coverage of the age profile. In both samples, roughly 58% of individuals are female.

Because the NHATS data include geocoded state of residence identifiers, I construct a mea-

Table 1: Initial Wave Summary Statistics

	NHATS				HRS			
	Overall	College	Black	Smoker	Overall	College	Black	Smoker
Word Recall Score	8.140	9.540	7.051	8.411	10.118	11.686	8.903	10.331
	3.283	3.124	2.994	3.227	3.804	3.564	4.000	3.783
Chronic Conditions	2.503	2.196	2.614	2.532	1.520	1.205	1.796	1.465
	1.555	1.442	1.522	1.687	1.291	1.130	1.328	1.342
Current Smoker	0.082	0.043	0.114	1.000	0.170	0.103	0.207	1.000
Former Smoker	0.443	0.457	0.402	0.000	0.253	0.311	0.256	0.000
Race								
White	0.707	0.802	0.000	0.637	0.843	0.900	0.000	0.810
Black	0.210	0.139	1.000	0.291	0.131	0.069	1.000	0.159
Other	0.083	0.059	0.000	0.072	0.026	0.031	0.000	0.031
College Education	0.225	1.000	0.149	0.116	0.168	1.000	0.088	0.101
Age/100	0.764	0.756	0.753	0.726	0.662	0.647	0.651	0.622
Female	0.571	0.428	0.596	0.578	0.588	0.456	0.649	0.567
Nonmetro Residence	0.190	0.135	0.109	0.232				
AHEAD					0.372	0.294	0.308	0.201
HRS					0.628	0.706	0.692	0.799
Obs. To Die	0.236	0.188	0.222	0.253	0.518	0.406	0.552	0.580
Obs. To Attrit	0.436	0.370	0.479	0.498	0.157	0.165	0.143	0.156
n	5,750	1,294	1,206	474	11,262	1889	1,474	1,913

Notes: The table presents initial wave (wave 1 in the NHATS, wave 3 in the HRS) summary statistics. The NHATS data include information on 5,750 individuals. Data labeled HRS are in fact the combined HRS and AHEAD cohorts, and they include information on 11,262 individuals. The HRS data were recorded in 1996, and the NHATS data were recorded in 2011.

sure of rurality based on rural-urban continuum codes. Roughly 19% of NHATS respondents lived in an area that was neither classified as metropolitan or metropolitan adjacent.[12] In addition, 62.8% of the HRS sample come from the main HRS survey, while the remaining 37.2% come from the AHEAD sample. Finally, Table 1 also hints at the potential for dynamic selection. Of all NHATS participants, 23.6% are observed to die over the ensuing nine waves, and 43.6% are observed to leave the sample through attrition. In the HRS, those percentages are 51.8% and 15.7%, respectively, and they make clear that sample compositions may potentially shift dramatically over time.

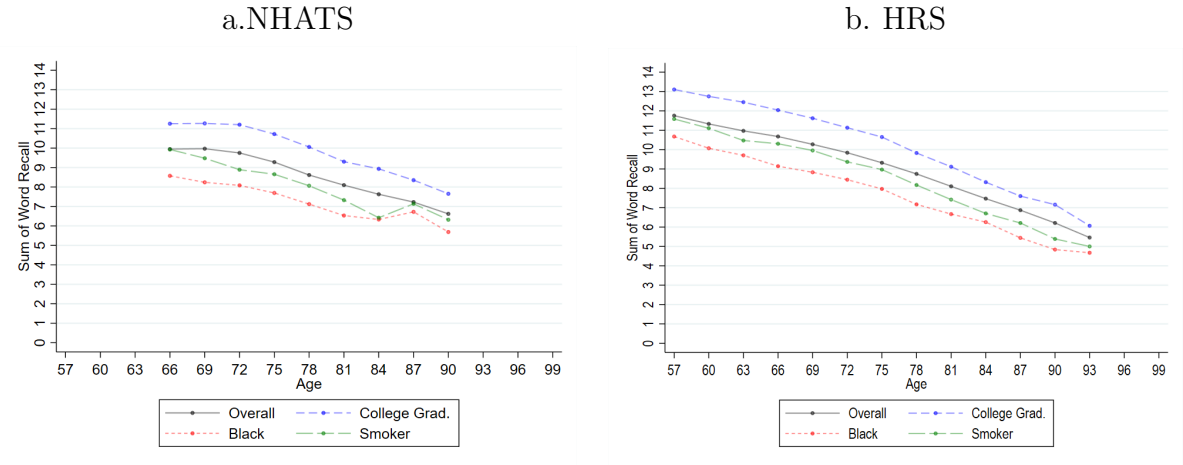
The theory in Section 2 says that cross-sectional comparisons of cognitive health at a given age reflect the ATT of a health investment or exogenous characteristic plus the dynamic selection effect. To investigate the left-hand side of Equation 4, Figure 1 presents the age profile of cognitive health overall and for those with a college education, Blacks, and smokers from each data source. In both NHATS and HRS data, word recall declines in age both overall and in each subgroup. Overall mean word recall falls from age 69 to age 90 by roughly 30-40%, and furthermore, with the exception of older Blacks and Smokers in the NHATS data, the age profiles are relatively parallel for the college educated, Black, and smoker groups, especially in the HRS. Figure 1 demonstrates the problem with cross-sectional comparisons over the age profile: in addition to the effects of individual characteristics on cognitive health, the health composition of the samples are potentially changing through non-random selective exits. Furthermore, in the case of smoking, smoking behavior itself is changing, often through non-random smoking cessation due to health changes.[13] Indeed, the literature has emphasized the dynamic nature of smoking and health, where both smoking affects health and health affects smoking (Arcidiacono et al., 2007; Darden, 2017).

One way to see the importance of dynamic selection is through Figure 2. In each figure, word recall is plotted over the age profile by the highest age of observation. For example, at age 81, the subset of the sample who live to at least age 90, as shown by the red line, has a mean word recall score that is 1.5 to 2 words higher than the mean word recall score for the

[12]The "Non-Metro" indicator here includes RUC codes 5, 7, and 9. Darden (2021) uses a similar rural definition in studying rural/urban smoking disparities.

[13]Darden & Kaestner (2022) use panel data on medical expenditures to demonstrate how cross-sectional comparisons understate the impact of smoking on health care usage, but their study only has a snapshot of smoking behavior and cannot decompose the effects of smoking cessation.

Figure 1: Age Profile of Cognitive Health by Smoking and Race.



Notes: The figures present the age profile of cognitive health (as measured by the sum of immediate and delayed word recall) both overall (Black solid line) and for college graduates (blue dashed line), Black respondents (red dotted line), and current smokers (green dashed line). Person/wave observations are 28,429 and 76,831 in the NHATS and the HRS, respectively.

subset of individuals who live to be at least 81, as shown by the blue line.[14] At a given age, the vertical distance between these lines makes clear that the sample composition of cognitive health changes through selective exits, and it demonstrates that the magnitude of the selection is not constant in age.

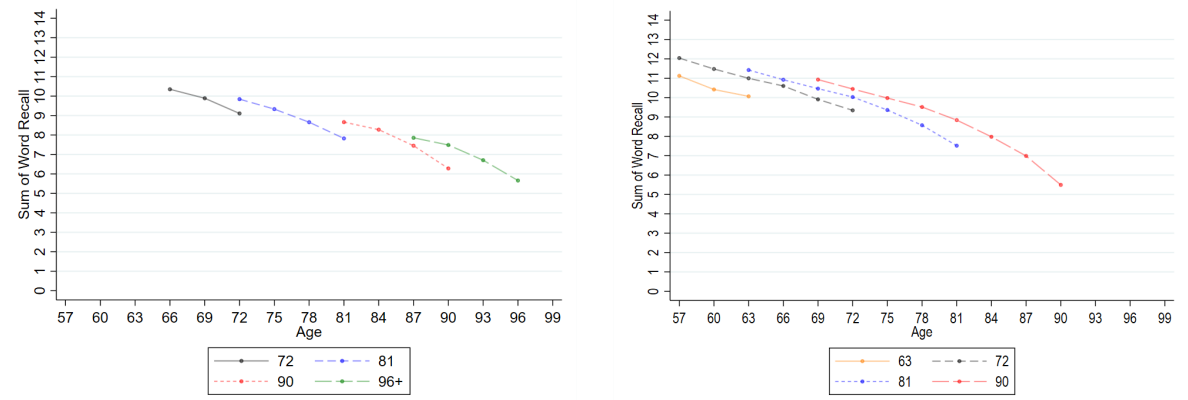
In Equation 4, the dynamic selection effect is the product of the correlation between general and cognitive health (i.e., ρ) and a term that depends on how the individual characteristic of interest affects general health. To investigate the correlation between physical and cognitive health, Figure 3 presents bar charts of word recall scores by the count of chronic health conditions for the NHATS and the HRS samples. The red lines represent the 95% confidence intervals for each count. In both data sources, there is a clear negative gradient between physical and cognitive health. For example, in the NHATS sample, word recall is one full word lower for individuals with four chronic health conditions relative to zero. Similar results exist in the HRS sample, and in all cases the confidence intervals reveal statistical differences across chronic health counts.

The second piece of the dynamic selection effect is the role of investments or characteristics on sample exit. Figure 4 presents a series of Kaplan-Meier survival curves, where survival is

[14]Confidence bands are omitted from the graph for clarity. Appendix Table 3 provides 95% confidence intervals for each age.

Figure 2: Age Profile of Cognitive Health by Highest Age of Observation.

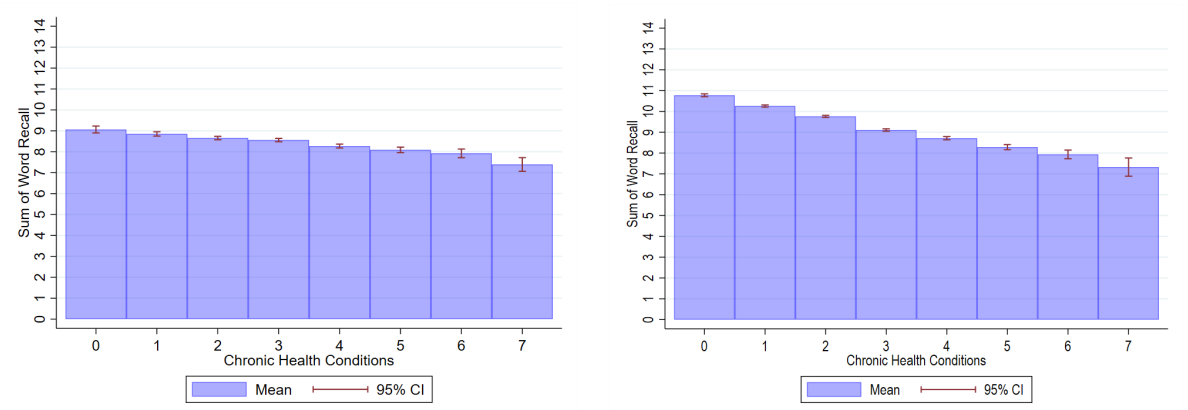
a. National Health and Aging Trends Study b. Health and Retirement Study



Notes: Each figure presents the age profile of the sum of immediate and delayed recall word scores for different maximum ages of observation. For example, the line labeled "81" in Figure 2a shows the observed age profile of word recall for NHATS respondents who lived to at least age 81. For a given age, the vertical distance between lines is suggestive of dynamic selection. The NHATS sample are of individuals over up to 9 years, and the HRS sample are of individuals over up to 21 years. Appendix Table 3 provides 95% confidence intervals for each figure. Person/wave observations are 28,429 and 76,831 for the NHATS and the HRS, respectively.

Figure 3: Cognitive Health by the Count of Chronic Health Conditions

a. NHATS b. HRS



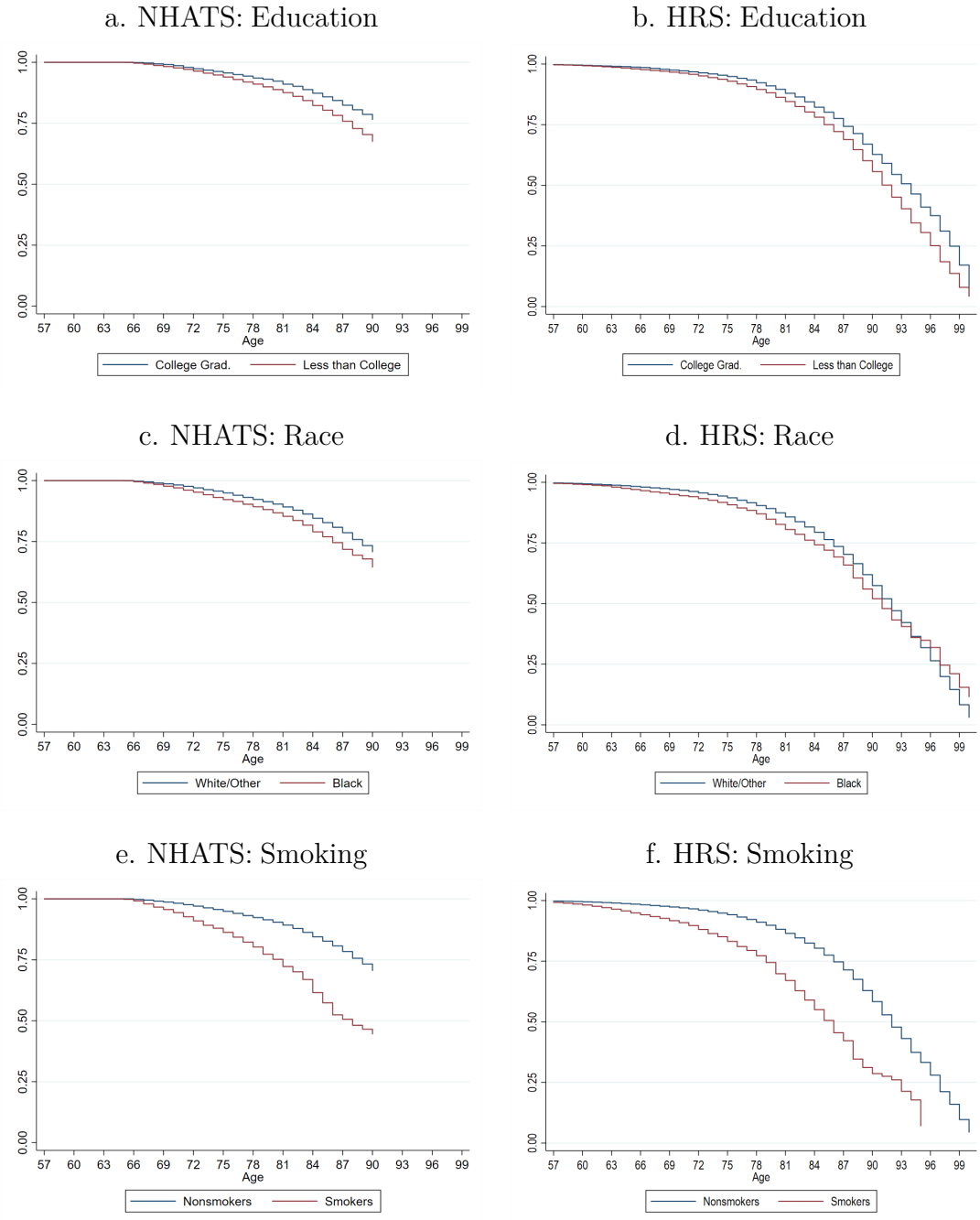
Notes: The figures present the sum of word recall scores by the count of chronic health conditions in the NHATS and the HRS. Person/wave observations are 28,429 and 76,831 in the NHATS and the HRS, respectively.

defined as participation in the sample such that a person does not die or leave through attrition. Figures 4a and 4b show the Kaplan-Meier survival curves for those with and without a college education in the NHATS and HRS, respectively, where a small gap in sample participation opens by the early seventies. Figures 4c and 4d show similarly small gaps between Blacks and

Whites/Other groups in the two data sets. Unsurprisingly, Figures 4e and 4f show much larger participation gaps between current smokers and non-smokers.[15]

[15]One concern is that, by focusing on the age profile with longitudinal data for 10 to 20 years, cohort effects may generate differential survival patterns. I find similar survival pictures over the wave profile, which suggests that gaps between groups are real.

Figure 4: Kaplan-Meier Survival Curves by Education, Race, and Smoking Status



Notes: Figures 4a and 4b show the Kaplan-Meier survival curves for college graduates and non-college graduates. Figures 4c and 4d show similar curves for White/other respondents and Black respondents. Figures 4e and 4f show survival curves for smokers and non-smokers. In all cases, survival is defined as remaining alive and actively participating (i.e., not leaving through attrition) in the sample. Person/wave observations are 28,429 and 76,831 in the NHATS and the HRS, respectively.

What can be inferred from Figures 1, 3, and 4? First, Figure 1 provides suggestive evidence

that education, race, and smoking are important factors in cognitive decline, but only in levels — the rate of decline is similar across subgroups, producing parallel age profiles. Second, the combination of results in Figures 3 and 4 suggest that the level differences in cognitive health by education, race, and smoking are potentially understated. Physical and cognitive health are positively correlated (Figure 3), and all three characteristics are related to sample participation probabilities(Figure 4). Finally, the dynamic selection effect is potentially not constant across the age profile. For example, Figure 4 suggests that the White/Black survival gap changes at older ages. To the extent that the correlation between physical and cognitive health changes over time, we would expect the bias induced by dynamic selection to change. In the following section, I present results from simple cross-sectional comparisons; the inverse probability weighting estimator; and my preferred discrete factor random effects system estimator.

5 Results

This section starts by presenting estimates from Equation 12, the single equation dynamic OLS estimator. I contrast results with and without inverse probability weights derived from a separately estimated selection equation. The remainder of the section describes the implementation, estimation, and simulation of the discrete factor random effects system estimator.

5.1 Regression Modeling

The first two columns in Table 2 report selected estimates of the parameters in Equation 12 for each data source, estimated on data from all waves except for the initial wave. In the NHATS, the mean, regression adjusted, difference in word recall scores between college graduates and those with less than a college education is 0.7 words. Relative to the initial wave mean of 8.14 words, this difference is roughly 8.6%. Relative to White respondents, Black respondents mean word recall score was 0.666 words lower, or roughly 8.2% lower. The mean difference between current smokers and non-smokers is -0.162 words, or 2% lower. In the HRS, the difference in mean word scores for college graduates is 0.828 words, or 8.2%; the White/Black gap is 0.776 words, or 7.7%; and the Smoking gap is 0.111 words, or 1.1%. In both NHATS and HRS, the education and race differences are statistically significant at all conventional levels

of significance, whereas the smoking results are marginally statistically significant.

An early test for the presence of dynamic selection comes from Verbeek & Nijman (1992), who proposed to re-estimate the OLS model with a control for the number of waves completed. The null hypothesis that the parameter on this variable is zero is consistent with there being no differences in mean word recall scores for those individuals with more frequent contributions to the likelihood function. In both NHATS and HRS, the p-value on this test is 0.000, which, consistent with Figure 2, provides evidence that those exiting the sample earlier are not random. To investigate, I estimate the inverse probability estimator of Wooldridge (2002), in which the researcher estimates a probit model of sample exit in waves $t \in \{2, \ldots, T\}$ as a function of initial wave $t = 1$ characteristics, and then uses the resulting predicted probabilities of sample exit as weights in the structural equation of interest. Identification comes from a "selection on observables" argument: conditional on observable characteristics in wave 1, sample exit is as good as "ignorable" in the structural equation. Table 2 presents estimates of the key α parameters from Equation 12 while weighting by the inverse of the predicted probability of sample exit in each wave generated from a probit model.[16]

The table shows that the inverse probability weights make little difference relative to the dynamic OLS model in either sample for education or race. In the NHATS, the White/Black gap increases in magnitude by 0.1 words while the smoking gap falls in magnitude by 0.05 words. The smoking gap, which was marginally statistically significant is no long statistically significant at normal significance thresholds. In the HRS, the college gap falls in magnitude by 0.1 words while the smoking gap increases in magnitude by 0.1 words. In neither NHATS nor HRS do the results with IPW move in uniform directions relative to the OLS estimates.

The IPW is limited in two ways. First, and most importantly, the IPW does not jointly model general health. Initial wave general health (i.e., $H_{it=1}$) influences the probability of selective exits later in the panel, and I have controlled for general health in the structural cognitive health equation, but there is no feedback from general health to cognitive health. Furthermore, in the absence of directly modeling general health, there is no correlation between determinants of general health and the determinants of cognitive health. Yet the theory suggests that this correlation helps to determine the sign of the dynamic selection bias. Second,

[16]See Appendix Table 4 for probit estimates.

Table 2: Cognitive Scores on Education, Race, and Smoking Behavior.

	OLS		IPW		DFRE	
NHATS n = 22,679 Person/Wave	Est.	St. Err.	Est.	St. Err.	Est.	St. Err.
College	0.700	0.035	0.708	0.042	1.419	0.059
Black	-0.666	0.041	-0.766	0.050	-1.484	0.057
Current Smoker	-0.162	0.078	-0.116	0.086	-0.340	0.101
HRS n = 65,569 Person/Wave						
College	0.828	0.035	0.732	0.036	-1.304	0.044
Black	-0.776	0.041	-0.754	0.068	1.345	0.047
Current Smoker	-0.111	0.044	-0.217	0.049	-0.175	0.043

Notes: The first two columns of the results report selected parameter estimates and standard errors from Equation 12. The last two columns report selected results from the inverse probability weighting estimator. The top panel reflects data from NHATS waves 2–9, and the bottom panel reflects data from HRS waves 2–11. All standard errors are clustered at the individual level. The p-values for the Verbeek & Nijman (1992) test on the coefficient on the number of completed waves are 0.000 for both samples. Person/wave observations are 22,679 and 65,569 in the NHATS and the HRS, respectively, which reflect the same sample above except for the initial wave, which is dropped to accommodate the dynamic model.

in the case of smoking, the IPW estimator offers no model of smoking behavior, which is itself clearly dynamic and subject to unobserved factors that shift prevalence. In fact, the dynamics of quitting are closely related to the dynamics of health as poor health often precedes smoking cessation.[17] The dynamic system of equations in Section 2 addresses these points, in addition to allowing for theoretically grounded counterfactual simulations. In the following subsection, I explain how I apply that estimator to both the NHATS and the HRS data.

5.2 Dynamic System Estimator

Applying the dynamic system estimator from Section 2 requires several assumptions and measurement decisions. To begin, consider the unobserved type equation (Equation 10) as a function of initial wave endogenous variables. The idea is to allow initial wave endogenous variables to shift the type probabilities such that these types reflect the endogenous histories leading to participation in a panel study. In this context, I include time-varying endogenous variables such as initial wave smoking, general health, and cognitive health. I also include an

[17] In the case of older individuals, virtually all smokers have a long history of smoking—there is little to no smoking initiation in this group.

indicator for former smoking (i.e., those with a smoking history but who have quit by the initial wave). Finally, given the focus on selection, the age that someone arrives at the panel is itself endogenous. Thus, I specify the probability that individual i is of type k as

$$\tau_k = ln\Big(\frac{P(\mu_i = \mu^k)}{P(\mu_i = \mu^1)}\Big) = \psi_0^k + \psi_1^k S_{i1} + \psi_2^k F_{i1} + \psi_3^k H_{i1} + \psi_4^k C_{i1} + \psi_5^k Age_{i1}. \tag{13}$$

Initial wave observed heterogeneity affects the probability of each unobserved type as in Equation 13. To implement the empirical model in waves 2 through 9 (in the case of the NHATS) or 11 (in the case of the HRS), the model proceeds as outlined in Section 2. I treat smoking behavior as a per-period endogenous health investment modeled as a function of existing health, lagged smoking behavior, and exogenous characteristics Z_{it}. For individuals who quit smoking during the panel, an indicator for former smoker turns on, and for individuals who relapse, the former smoker indicator turns off. In each data source, smoking is measured as recent smoking behavior prior to the interview wave, so I structure wave t smoking as affecting cognitive health as measured immediately at wave t. General health H_{it} is the sum of chronic conditions up to period t, so I assume that general health at t affects smoking behavior S_{it}, both of which shift cognitive health at t. Sample exits occur both from mortality and from attrition, and in practice, I models these types of exits separately as a function health and smoking and exogenous variables. Both education and race enter directly in all equations of the model. I treat race as an exogenous variable. In the case of education, the assumption is that the level of educational attainment is pre-determined relative to the time frame of the model and, conditional on μ and other characteristics in Z_{it}, exogenous.

In NHATS and HRS samples, I estimate parameters that dictate smoking behavior, cognitive health, general health, mortality, and attrition jointly following from the likelihood function in Equation 11. To arrive at the number of points of support of the distribution of unobserved heterogeneity (i.e., K), I progressively add points of support, re-estimating the entire model until the log-likelihood function fails to improve. For both the NHATS and the HRS model, I arrive at $K = 5$ points of support, which implies that I estimate 102 and 104 parameters in the NHATS and the HRS samples, respectively. The respective log likelihood values are -83,396.50 and -232471.99. Ultimately, to compare the system results to those in

Table 2, I rely on simulations that change education, race, and smoking information. However, to highlight the importance of unobserved heterogeneity, Table 3 presents estimates of the cognitive health equation when estimated separately ("Single Equation") and when estimated jointly with all other equations ("DFRE"). The cognitive health specification is very simple; it does not include polynomials in age or interaction terms. As the parameters capture level shifts, changes in the slope of the age profile of cognitive health will come through modeled composition changes due to selection.

Interpreting the parameters in Table 3 is challenging, especially in the case of the full system, but some patterns emerge. First, in both NHATS and HRS, the parameter estimates on college graduate and Black are significantly larger in magnitude in the system DFRE specification relative to the (unweighted) single equation estimator. In the case of education, these results are consistent with a significantly negative selection effect: Figure 3 shows a positive correlation between general and cognitive health, and education encourages sample participation, through either longer longevity and/or lower attrition. In the case of race, the dynamic selection effect is positive because Blacks leave the sample at a greater rate. For current cigarette smoking, in both NHATS and HRS, the negative effects grow in magnitude, although only in NHATS do the effects grow beyond those implied by the IPW estimator. In the NHATS sample, there is not sufficient variation in smoking behavior to identify former smoking separately from current smoking in the cognitive health equation *conditional* on former smoking entering in the determination of unobserved type. For this reason, I also omit former smoking from the single equation specification. Because the magnitude of these results is better presented through simulation, I present parameter estimates of the smoking, general health, mortality, attrition, and initial conditions equations in Appendix Tables 5–8.

To gain intuition on the estimated distribution of unobserved heterogeneity, Table 4 provides estimates of the μ intercept parameters and the type probabilities for each data source.[18] To identify the μ terms, in each data source, I normalize $\mu_{\text{i}} = 0$ in each equation. Thus, type one serves as the baseline to which other types are assessed. As an example, relative to this normalization in the NHATS data, type 4 individuals, who constitute roughly 10.3% of the sample in wave one, are significantly less likely to smoke; they have significantly lower word

[18] Appendix Table 8 provides the estimated ψ parameters from Equation 10.

Table 3: Cognitive Scores on Smoking Behavior: System Estimator

	NHATS				HRS			
	Single Equation		DFRE		Single Equation		DFRE	
	Est.	St. Err.	Est.	St. Err.	Est.	St. Err.	Est.	St. Err.
College Graduate	0.700	0.039	1.273	0.055	0.828	0.028	1.233	0.044
Race								
Black	-0.768	0.044	-1.337	0.058	-0.776	0.033	-1.223	0.050
Other	-0.639	0.068	-1.095	0.101	-0.595	0.066	-1.135	0.095
Current Smoker	-0.162	0.070	-0.311	0.097	-0.111	0.037	-0.193	0.053
Former Smoker					0.006	0.026	-0.028	0.040
L. Word Recall Score	0.560	0.006	0.213	0.007	0.462	0.003	0.184	0.004
Chronic Conditions	-0.046	0.011	-0.072	0.014	-0.131	0.008	-0.156	0.011
Age/100	-8.913	0.259	-14.433	0.347	-8.595	0.171	-12.942	0.202
Female	0.389	0.034	0.713	0.048	0.616	0.023	0.915	0.034
Nonmetro Residence	-0.110	0.042	-0.112	0.056				
AHEAD					-0.287	0.036	-0.362	0.048
Constant	10.738	0.229	18.931	0.306	11.124	0.133	13.919	0.153
Permanent Mass Points								
μ_2^c			-1.019	0.093			6.151	0.079
μ_3^c			2.174	0.063			4.068	0.060
μ_4^c			-4.250	0.091			2.519	0.142
μ_5^c			-2.024	0.051			2.183	0.047

Notes: The table presents estimates of the word recall equation for the NHATS and the HRS data. For each data source, the first two columns present estimates and standard errors when estimating this equation separately from other equations in the model. The second two columns present estimates and standard errors from my preferred system estimator in which all equations are estimated jointly. Samples sizes are 28,429 and 76,831 person/year observations for the NHATS and the HRS, respectively.

recall scores; and are significantly more likely to attrit from the sample. Furthermore, individuals with higher counts of chronic health conditions in wave 1 are significantly more likely to follow type 2 patterns (see Appendix Table 8), which constitute roughly 31.1% of the sample and are significantly more likely to attrit from the sample (but are not more likely to die). In the HRS, type 4 individuals, who make up 2.4% of the sample, have significantly higher word recall scores, but also have significantly greater numbers of chronic health conditions, and they are more likely to attrit from the sample and die.

Table 4: Estimated Unobserved Factors

		Smoking		Cognitive Health		General Health		Attrition		Death	
	Probability	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.
NHATS											
μ_1	0.289	0.000	.	0.000	.	0.000	.	0.000	.	0.000	.
μ_2	0.311	-0.114	0.345	-1.019	0.093	0.005	0.018	3.769	0.176	-10.283	0.000
μ_3	0.075	0.145	0.452	2.174	0.063	0.006	0.020	0.956	0.278	0.265	0.212
μ_4	0.103	-0.977	0.399	-4.250	0.091	-0.009	0.020	1.664	0.229	-0.343	0.164
μ_5	0.222	-0.337	0.265	-2.024	0.051	-0.009	0.014	-0.335	0.350	-0.314	0.127
HRS											
μ_1	0.109	0.000	.	0.000	.	0.000	.	0.000	.	0.000	.
μ_2	0.071	0.349	0.201	6.151	0.079	0.012	0.015	0.509	0.261	0.544	0.117
μ_3	0.372	0.190	0.145	4.068	0.060	0.000	0.010	0.465	0.177	-0.010	0.079
μ_4	0.024	-0.148	0.328	2.519	0.142	1.097	0.024	1.823	0.295	1.568	0.153
μ_5	0.425	0.207	0.136	2.183	0.047	-0.008	0.010	0.309	0.185	-0.233	0.076

Notes: The table presents the estimated μ factors and the associated standard errors from each equation in the DFRE system of equations model. These estimates are generated from estimating all equations jointly. Estimated factors are relative to the normalization that all factors associated with type 1 are zero.

Tables 3 and 4 provide evidence of the importance of jointly estimating health investment, health, and selection equations and allowing for unobserved heterogeneity, but parameters themselves are difficult to interpret because of the complexity of the system. In the next subsection, I simulate the estimated system model to both assess model fit and to demonstrate the role of dynamic selection bias over the age profile.

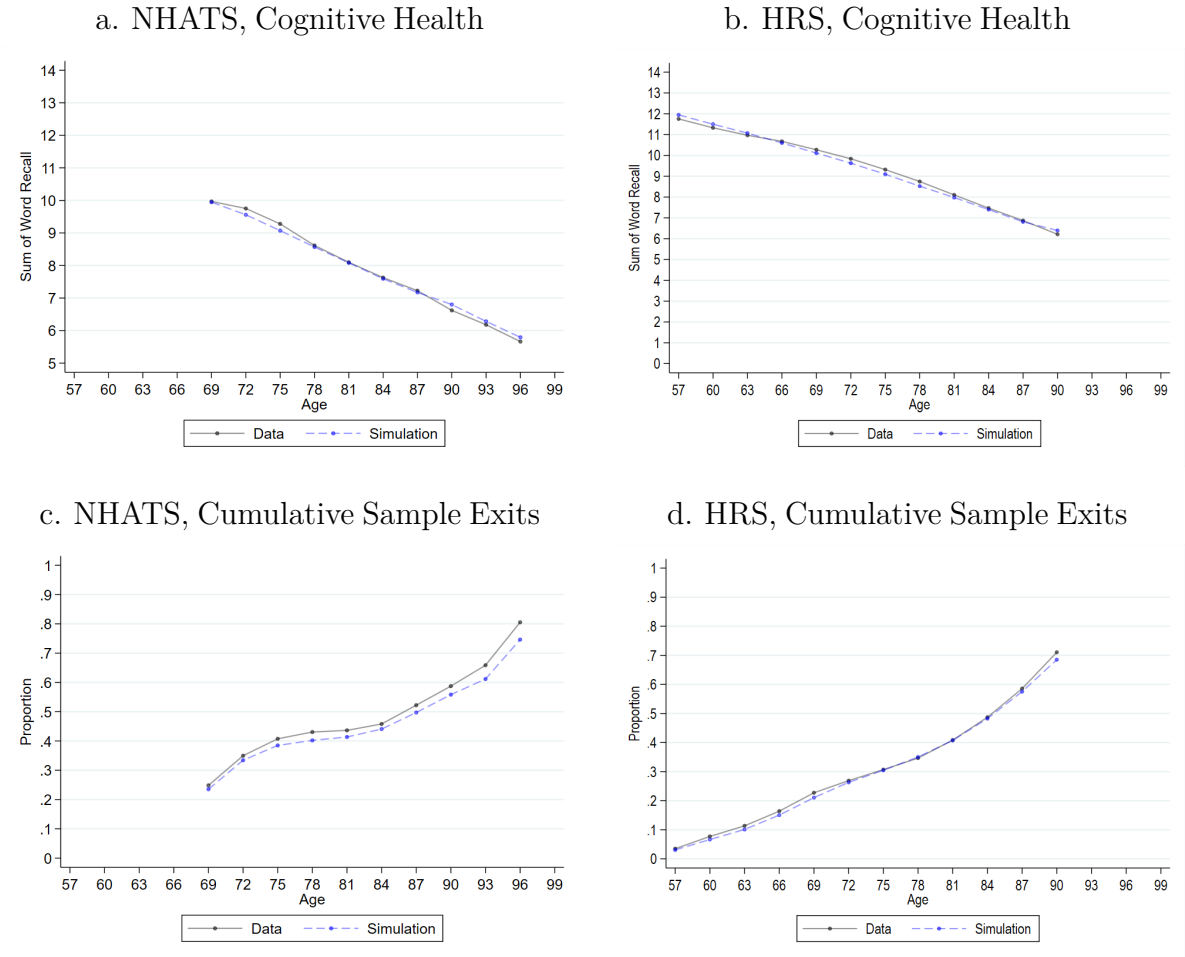
5.3 Simulation

To simulate the estimated model, I replicate each individual in each data source 50 times, and I endow each simulated individual with error draws from each equation. For a given individ-

ual, their data from the initial wave generates the probability of each unobserved type in the distribution of unobserved heterogeneity. From that probability distribution, a random draw determines a simulated individual's μ type, which remains fixed for the rest of the simulation. Thus, a given individual in the data may have multiple types over their 50 replications. Importantly, as I simulate forward, the endogenous characteristics (i.e., smoking, cognitive health, and general health) update based on the simulated behavior and outcomes. In the event that an individual is simulated to die or attrit, an indicator signals that the person is simulated to leave the sample. Because the exogenous characteristics are time invariant, in the case that a person dies or attrits in the data but is not simulated to leave, I continue to simulate the person's behavior and outcomes until they are simulated to leave.

I begin by simulating the sample under a baseline scenario in which all exogenous characteristics are kept as in the data and behavior (i.e., smoking), health, death, and attrition are all endogenous. From this simulation, I calculate the mean smoking, health, and sample exit probabilities conditional on remaining in the sample in the simulation. In all figures that follow, the results by age are grouped into three-year age bins for ease of presentation. Figure 5 demonstrates that over the age profile, the simulated cognitive health outcomes and cumulative sample exit proportions (from either mortality or attrition) closely mirror the data. Appendix Figures 1 and 2 demonstrate a similar model fit for smoking behavior, general health, and the separate outcomes of attrition and death.

Figure 5: Model Fit

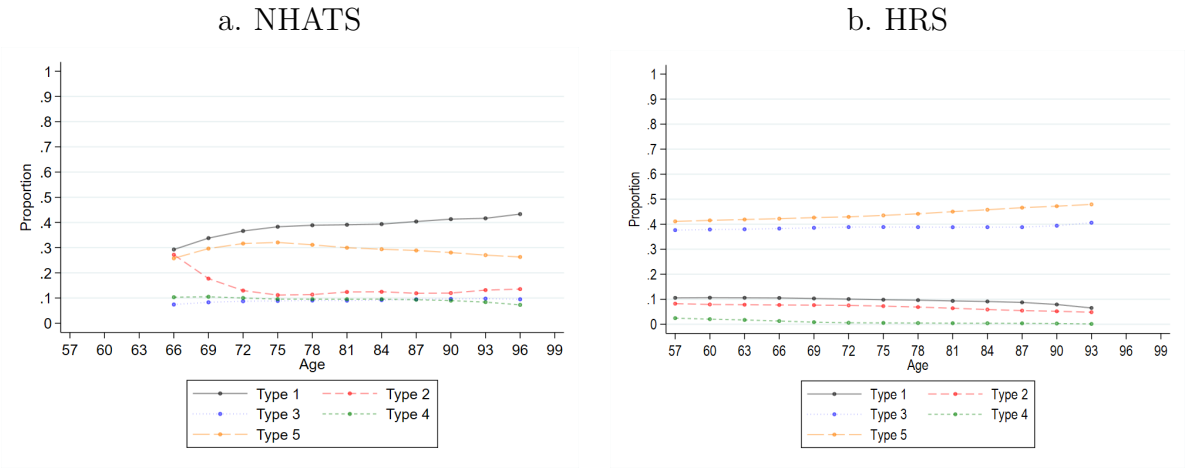


Notes: The figure presents cognitive health (a and c) and the cumulative sample exits (b and d) for the NHATS and HRS samples. The Black solid lines come directly from the data conditional on remaining in the sample data, and the blue dotted lines come from simulation conditional on remaining in the simulated sample.

Next, Figure 6 shows how the simulated distribution of unobserved heterogeneity changes over the age profile. Because types are estimated to die/attrit at different rates, the figure reveals how the sample composition on the basis of permanent type changes as some individuals selectively drop from the simulated sample. For example, in NHATS data in Figure 6a, type 2 individuals, who comprise roughly 30% of the sample at younger ages, comprise only 13% of the remaining sample by age 90 because they are much more likely to leave the study through attrition. Similarly, type 4 individuals are much more likely to leave the sample through attrition, and they have, on average, a 4.25 lower word recall score than type 1 individuals. In the HRS, Figure 6b shows that the most prominent type (type 5) becomes more so over the age

profile. These individuals are of significantly better cognitive health than type 1 individuals.

Figure 6: Age Profile of Unobserved Heterogeneity Distribution



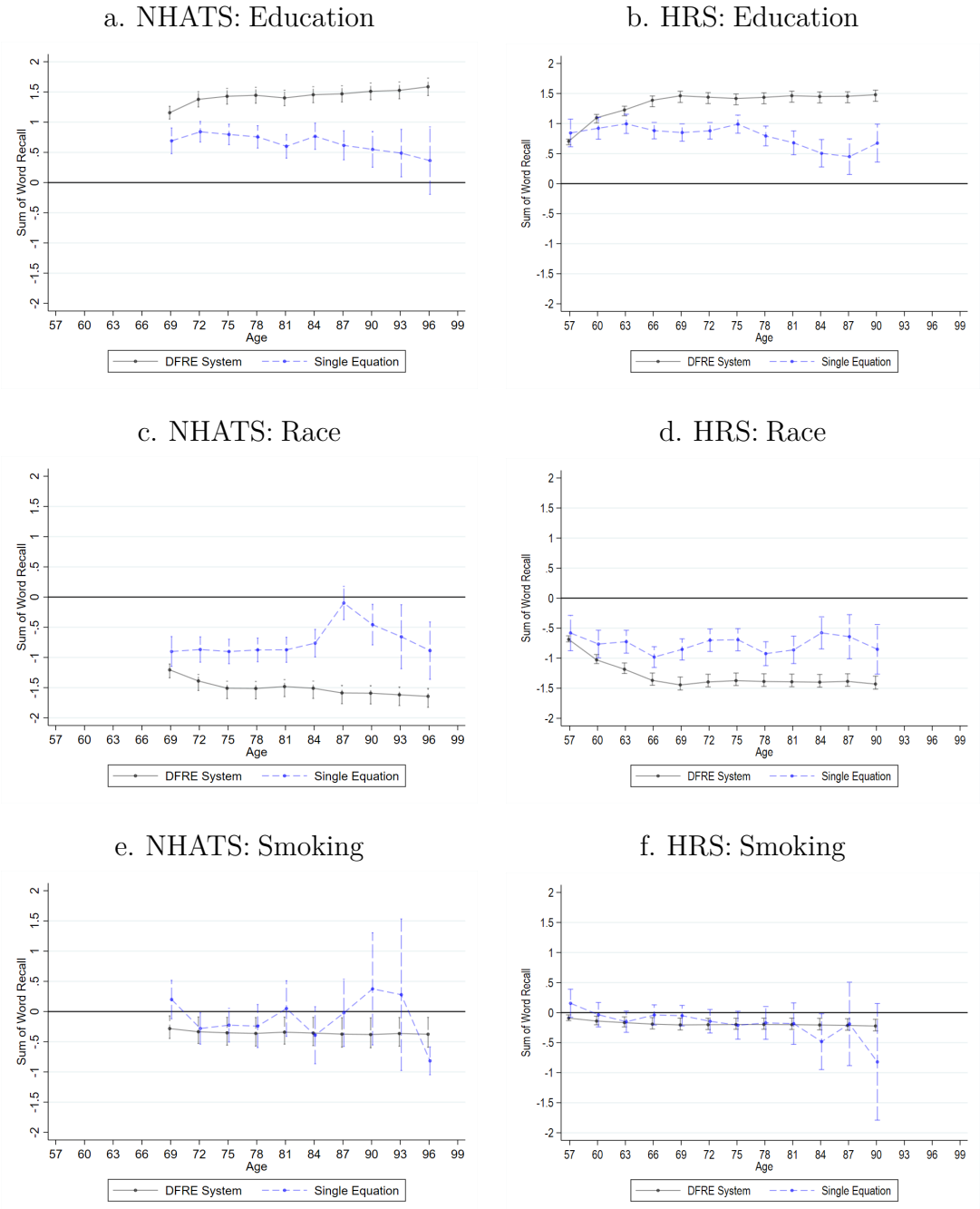
Notes: The figure presents the simulated proportions of each unobserved type over the age profile conditional on remaining in the simulated sample.

I focus on the average treatment effect on the treated (ATT) with respect to education, race, and smoking, which I define as the difference in mean cognitive health conditional on a given treatment relative to the mean cognitive health in a counterfactual scenario in which those same individuals did not have the treatment. To calculate the standard errors, I draw 50 sets of parameters from the full variance/covariance matrix and re-simulate the model under each draw. Table 2 presents estimates from simulation of the model under the column DFRE. As suggested by the point estimates in Table 3, in NHATS data, the simulated ATTs are all significantly larger in magnitude than either the OLS or IPW estimates. The college gap in mean word recall score is now 1.419 words, or 18.3%; the White/Black gap is now 1.484 words, or 18.2%, and the smoking gap is now 0.34 words, or 4.2% words. Similar results exist for HRS data. However, the average effects are larger in magnitude in ways that are consistent with theory and with results in Section 4.

Figures 7a and 7b show the effects of college graduation on word recall scores for those with a college education over the age profile. The blue dashed line presents regression adjusted estimates of these effects from the single equation (unweighted) estimator in Equation 12 in which I allow the effects to vary by three-year age bins. The black solid line presents similar estimates from the preferred DFRE system estimator. In both NHATS and HRS, Figures

7a and 7b show a.) significantly larger effects in the DFRE specification and b.) widening gaps between the estimates over the age profile. In both Figures, the DFRE effects grow in magnitude while the OLS estimates generally tend towards zero. Similarly, the effects of race for Blacks are a.) larger in magnitude than OLS and b.) grow in magnitude over the age profile. These effects, shown in Figures 7c and 7d, suggest that at nearly all ages, the dynamic selection effect is enough to statistically bias the implications of race towards zero. In the case of smoking in Figures 7e and 7f, the DFRE simulation results are larger in magnitude, but the confidence intervals overlap with those from the OLS estimator, and there are not significant trends in the gap between the estimates generated from each estimator.

Figure 7



Notes: The figure presents the simulated average treatment effect on the treated (ATT) over the age profile for education (a and b), race (c and d), and smoking (e and f). The blue, dashed line presents results from a modified version of Equation 12 in which the effects of the characteristics of interest are interacted with binary variables for 3-year age bins. The black, solid line presents simulated results from the DFRE estimator. 95% confidence intervals are clustered at the individual level for the OLS estimator and bootstrapped with 50 replications of the full variance-covariance matrix of parameter estimates.

Results in Figure 7 also shed light on the degree to which each individual characteristic affects just the level of cognitive health or also the slope. This issue highlights the value of

using both NHATS and HRS data, which have respectively better coverage at older and young ages. In the case of education, the gap in cognitive health between college graduates and non-graduates is fairly stable between ages 72 and 90 at roughly 1.5 words. However, at younger ages, particularly in the HRS, there is evidence that the gap in cognitive health is considerably smaller. For example, at age 57, the gap is only 0.6 words and not significantly different that the OLS estimate. Similarly, with respect to the White/Black gap, the DFRE emerges as statistically larger in magnitude at age 63 in HRS. In the case of smoking, the DFRE estimates are larger in magnitude relative to the OLS estimates, but the ATT of smoking on cognitive health is small, and the differences between estimators are not statistically significant.

6 Discussion

This paper shows that dynamic selection is an important source of bias in applied microeconomic settings in which researchers wish to understand how a treatment affects some outcome or behavior. The theory in Section 2 is general in that it demonstrates that dynamic selection will be a problem when the treatment in question affects the probability of sample composition changes (through the health implications for mortality and attrition) and when the outcome of interest is correlated with other drivers of sample exit. Importantly, dynamic selection is not limited to panel data settings—cross-sectional comparisons over the age profile are just as problematic.

In the context of cognitive decline, I find that three commonly thought of risk factors for decline—education, race, and smoking—are more important than previously believed, and, in the case of education and race, their importance grows at relatively young ages before leveling off. Simulating the estimated model yields effects of education and race that are roughly double the estimated effects from an IPW estimator in an older cohort; in a younger cohort, the effects of education and race are also significantly larger than IPW estimates. One possible explanation for these findings is that unobserved determinants of longevity—which the system of equations explicitly allow for—are important in a cohort whose initial mean age is 76.

The estimated model reveals that the overall age profile of cognitive health is also likely propped up by dynamic selection. To investigate, I compare the simulated age profiles of

cognitive health from the baseline simulation to a simulation in which no one is allowed to exit through either death or attrition. There is no significant difference in the mean of cognitive health at age 69 between simulations; but by age 90, mean cognitive health is 5.88% lower in the NHATS and 6.26% in the HRS in the simulation in which sample composition is not allowed to change. These results have implications for understanding how changes in life expectancy — through technological advancement, or, in the case of reductions, due to "deaths of despair" — change the prevalence of cognitive disease and dementia. As individuals live longer, failing to account for dynamic selection may suggest that elder cognitive health is getting worse, when in fact the marginal person is simply living longer.

Using panel data, this paper also shows that connecting economic theory with estimation in a quasi-structural way can illuminate empirical problems that may not be well-appreciated. Indeed, the motivation for the empirical model comes out of a standard (i.e., Grossman (1972)) economic theory of behavior, and yet the issue of dynamic selection is not often discussed in empirical economics. The quasi-structural approach is simple to estimate and simulate, and this estimator has the appealing property that correlation in the error structure is easy to allow for. Furthermore, researchers often wish to chart not only the average effect of an intervention, behavior, or policy but also how that effect varies over the age profile. Simulating the estimated model makes this exercise straightforward. Future research may apply this estimator to a wide variety of questions in which dynamic selection may be significant.

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