Racial and Ethnic Disparities in Functional Health Trajectories and Their Life Course Antecedents: Results from a Two-Part Latent Growth Mixture Model

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Racial and Ethnic Disparities in Functional Health Trajectories

Abstract

This paper investigates racial/ethnic disparities in functional health trajectories and the life course determinants of those disparities. We further differentiate between trajectories of functional limitation onset and severity using two-part latent class growth mixture models. We find significant racial and ethnic inequalities in functional health trajectories. Non-Hispanic whites have lower odds of having any limitation at baseline than blacks and Hispanics. Among those with any limitations whites also have fewer initial limitations than blacks and Hispanics. However, whites tend to have larger increases in the severity (number) of limitations over time than racial/ethnic minorities. These inequalities result from both differences in processes of limitation onset as well as processes related to severity. We further find that racial/ethnic inequalities in functional health trajectories result from both childhood and adult factors, though the latter largely mediate the impact of the former. Finally, we find that racial/ethnic inequalities in health trajectories derive from both differential exposure to risk as well as the group differences in the impacts of those risks

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INTRODUCTION

In this paper we examine racial and ethnic disparities in functional health trajectories among older Americans with particular attention paid to their life course antecedents. An enduring aspect of the US health and mortality regime is that African Americans suffer disproportionately from disease, illness, and premature mortality relative to the non-Hispanic white population (Rogers 1992; Fingerhut and Makuc 1992; NCHS 2001). Yet the black-white health gap must also be juxtaposed against the broader landscape of substantial racial/ethnic heterogeneity in health outcomes (Braithwaite and Taylor 1992; Furino 1992; Livingston 1994; Zane, Takeuchi, and Young 1994). The ongoing debate about the Hispanic-Latino health paradox illustrates but a small slice of that heterogeneity (Franzini, Ribble, and Keddie 2002; Palloni and Arias 2004).

BACKGROUND

Racial and Ethnic Disparities in Functional Health

Much of the literature on racial and ethnic differentials in health has historically focused on either early life outcomes (birth outcomes, infant and child mortality) or summary measures of population health (life expectancy). The past decade has seen increased interest on the other end of the life course. For example, using data from the Health and Retirement Study (HRS), Kington and Smith (1997) have shown that among those aged 51-61 blacks experience significantly higher prevalence of chronic disease compared to non-Hispanic whites including hypertension and diabetes, while Hispanics had higher prevalence of diabetes than non-Hispanic whites but lower prevalence of heart conditions and arthritis. Accordingly, a growing body of research has begun documenting racial and

ethnic disparities in later life health outcomes including functional impairment and disability and the mechanisms that underlie them.

Previous research has found that there are important racial and ethnic differences in older adult functional health status, measured by functional/mobility limitations, and activities of daily living (ADLs) (Cho et al. 2004; Ostchega et al. 2000; Himes 2000; Ferraro, Farmer, and Wybraniec 1997; Kington and Smith 1997; Smith and Kington 1997; Jette, Crawford, and Tennstedt 1996). Racial and ethnic differences in functional health are also age dependent (Markides and Mindel 1987; Gibson 1991). For example, Clark, Maddox, and Steinhauser (1993) have found that over the age of 70 blacks have consistently higher levels of activity of daily living (ADL), upper body, and lower body limitation than do whites. However, they also found substantial differences across age groups in racial inequalities in the decline of functional health over a 6-year follow-up such that blacks under the age of 80 were more likely to experience a decline, while those 85 and older were significantly less likely.

Most studies of racial and ethnic disparities in age-related patterns of functional health have not investigated group differences in individual-level trajectories. Instead they have documented group differences in functional status at different ages and then inferred differences in trajectories based on those cross-sectional observations (Clark et al. 1993). For example, Zsembik and colleagues (2000) have investigated how racial and ethnic differences in disability result from differences in the underlying disablement process. They find that differences in disability between whites and blacks is due largely to differences in rates of chronic disease and cognitive impairment, while differences between whites and Mexican Americans result from impairments to both physical and

cognitive functioning. Unfortunately, their cross sectional analysis is unable to model ethnoracial heterogeneity in the disablement process as it unfolds over time at the individual level. Those that have modeled ethnoracial differences in functional health trajectories have typically only looked at onset of disability (Dunlop et al. 2007).

Another consistent finding is that racial and ethnic differentials in functional health are substantially reduced when controls for adult socioeconomic position are included (Cho et al. 2004; Kelley-Moore and Ferraro 2004; Clark and Maddox 1992; Rogers 1992). In cross-sectional analysis of data from the HRS, Kington and Smith (1997) found that blacks and Hispanics experienced significantly greater functional impairment than non-Hispanic whites without adjustment for adult socioeconomic attainment. After controlling for household income and wealth racial and ethnic disparities largely disappear. This would suggest that a large portion of the racial and ethnic inequality in functional health outcomes is likely due to the larger racialized nature of social stratification processes.

Life Course Influences on Racial and Ethnic Disparities in Health

Two general theoretical perspectives have been offered to describe how health unfolds over the life course. The *critical period* approach posits that negative events occurring during developmentally salient periods may permanently alter the trajectory of health over the life course (Kuh and Ben-Shlomo 1997). While health insults may occur at very early ages (even in-utero), it is not until much later that these effects manifest themselves in disease pathologies. The most well-known and controversial example of critical period effects are the fetal-origins of diabetes and cardiovascular disease proposed by Barker (1994). Barker hypothesizes that poor maternal nutrition at critical periods during

gestation results in fetal growth retardation which alters the structure and function of important tissues associated with insulin, blood pressure, and lipid regulation. In turn, this increases the risk of adult chronic disease, most notably cardiovascular disease and diabetes.

Alternatively, the *cumulative insult* model suggests that exposures accumulate over the life course and that it is this lifetime accumulation that is important. The cumulative insults approach posits that there are social, environmental, and behavioral exposures over the life course which alters an individual's risk of disease in addition to any critical period effects (Kuh and Ben-Shlomo 1997). Under this conception, poor health and socioeconomic disadvantage in childhood represent two of many possible health-related insults over the life course, the effects of which may be either compounded by continued social, economic, and physical deprivation or partially or wholly ameliorated by upward social mobility and or healthy adult lifestyle. A growing body of research documents the role of health insults and inputs at various points in the life course in influencing adult health.

In addition, previous research has found that early childhood characteristics can play a significant role in determining later life health outcomes. For example, previous research has found that those from disadvantaged backgrounds have worse self-rated health (Rahkonen, Lahelma, and Huuhka 1997), more health-related risk factors (Blane, Davey Smith, Gillis, Hole, and Hawthorne 1996), increased risk of chronic diseases (Gilman, Kawachi, Fitzmaurice, and Buka 2002; Wannamethee, Whincup, Sharper, and Walker 1996; Hart, Hole, and Davey Smith 2000) and higher mortality rates (Davey Smith, Hart, Blane, Gillis, and Hawthorne 1997). Childhood socioeconomic disadvantage

has also been found to be associated with low physical functioning at midlife (Lou and Waite 2005; Guralnik, Butterworth, Wadsworth, and Kuh 2006) as well as functional health trajectories (Haas 2008).

However, not all studies have confirmed an important role for childhood SES in determining adult health (Lynch et al. 1994). There is also debate as to the relative influence of early life and adult socioeconomic circumstances on adult health. Despite the above evidence, some researchers suggest its role is limited to that of an upstream determinant of more proximal adult SES (Marmot, Shipley, Brunner, & Hemingway 2001). Other research suggests that the relative contribution of childhood and adult SES may vary by the underlying disease process (Davey Smith et al. 1998).

There is an extensive body of research documenting that childhood health insults also have long-term impacts on health that persists into late adulthood. Those who experience poor childhood health have increased risk of numerous adverse adult health outcomes including chronic disease, poor self-rated health status, and work-limiting disability (Colley, Douglas, and Reid 1973; Kuh and Wadsworth 1993; Blackwell, Hayward, and Crimmins 2001; Lou and Waite 2005; Haas 2007). There is evidence from the 1946 British cohort study linking childhood SES, birth weight, physical growth, and cognitive development to physical performance in midlife (Kuh et al. 2002; Kuh et al. 2006). Similarly, early life health has been shown to have significant impacts on trajectories of functional limitation in the HRS (Haas 2008).

Given the large and persistent racial and ethnic disparities in early life socioeconomic opportunities (Eggebeen and Lichter 1991) and health insults (Singh and Yu 1995; Hummer et al. 1999), there is reason to expect that such differentials in early

life exposures may also play a role in determining racial/ethnic differentials in the onset and progression of functional limitation. It is also unclear how much of the impact of early life insults is mediated by later life health and socioeconomic status. Little research in racial disparities in functional health have examined the whether impact of such life course factors vary across racial and ethnic group or the extent to which such differences explain group differences in health trajectories.

Research Questions

Based on the preceding discussion we pose the following research questions. First, how do trajectories of functional health status vary between racial and ethnic groups and what are the life course determinants of that variation? We take advantage of 6 waves of longitudinal observation covering 10 years to model individual growth trajectories in functional health. Second, to what extent are racial and ethnic differences in functional health trajectories tied to differential onset of limitations or to the degree of severity conditional on onset? No studies have estimated the extent to which such disparities result from differential onset or differential severity conditional on onset. We employ a two-part latent growth curve model for semi-continuous data which allows us to disaggregate racial and ethnic differences in longitudinal functional health trajectories into their onset and severity components. Third, do various life course factors have differential impacts on functional health trajectories across racial and ethnic groups? Towards this aim we investigate the relative contribution made by childhood and adult health and socioeconomic factors towards explaining racial and ethnic differentials in functional health trajectories.

METHODS

Racial and Ethnic Disparities in Functional Health Trajectories

The Health and Retirement Study (HRS) is an ongoing panel study of Americans begun in 1992 and designed to investigate economic and health transitions associated with retirement (Juster & Suzman 1995). Follow-up takes place every second year. The original HRS cohort was composed of 12,652 individuals. Respondents were selected from a sample of housing units generated using a multi-stage, clustered area probability sample. Face-to-face, in-home interviews were conducted at baseline and follow-up telephone interviews occur every second year, with proxy interviews after death. The HRS includes over-sampling of Hispanics, Blacks, and Florida residents. Cases that were missing information on ethnoracial group were dropped from the analysis. Due to their small number of cases we also dropped cases that were not either non-Hispanic white, non-Hispanic black, or Hispanic. The final analytic sample consists of 9,860 respondents

Dependent Variable

Functional limitations for the HRS cohort come from six waves of data (1994-2004). The wording of the questions assessing functional limitations among the HRS cohort changed from 1992 to 1994 making the 1992 data not comparable with later waves. Respondents were asked if they had any difficulty with the following tasks: walking several blocks, walking one block, sitting for about 2 hours, getting up from a chair after sitting for long periods, climbing several flights of stairs without resting, climbing one flight of stairs without resting, lifting or carrying weights over 10 lbs, stooping kneeling, or crouching, reaching arms above shoulder level, pushing or pulling large objects, and picking up a dime from the table. Answers were coded to be 0 for no limitation in the task and 1 for limitation in the task. The above indicators are combined into an additive scale with values ranging from 0 to 11. The reliability for the functional limitation scale has been

assessed with Chronbach's alpha and is .86 for the HRS 1994 (Fonda and Herzog 2004). The Chronbach's alpha for the functional limitation scales in later waves are all .87 or higher.

Independent Variables

The primary variable of interest in this analysis is respondents' race and ethnicity, which is coded into the following categories: White Non-Hispanic (reference), Black Non-Hispanic, and Hispanic. Other independent variables for this analysis include gender, age, marital status, region of residence, morbidity, smoking behavior, BMI, education, household income, and net wealth. Childhood predictors include childhood health, parental education, childhood SES, if a child ever moved for financial reasons, and if the father was unemployed.

Gender is coded so that 0 indicates males and 1 indicates females. Age at baseline is a continuous variable. Marital status at baseline is coded as a dummy variable with the following categories: married (reference), separated/divorced, widowed, and never married.

Morbidity is indicated by the number of chronic health conditions ever diagnosed. The respondents were asked whether or not a doctor has ever told them they had the following conditions: high blood pressure or hypertension, diabetes or high blood sugar, cancer or a malignant tumor of any kind except skin cancer, chronic lung disease except asthma such as chronic bronchitis or emphysema, heart attack, coronary heart disease, angina, congestive heart failure, or other heart problems, stroke or transient ischemic attack, arthritis or rheumatism, and emotional, nervous, or psychiatric problems. The number of conditions one has been diagnosed with is summed.

Smoking behavior is assessed with two questions from wave 1 (1992). The first question assesses if the respondent ever smoked cigarettes and the second assesses if the respondent currently smokes cigarettes. Smoking behavior is coded as a dummy variable with the following categories: never smoked (reference), former smoker, and current smoker. BMI is calculated by dividing weight by the square of height (weight/height^2). Wave 1 BMI is coded as a dummy variable and separated into normal weight (BMI of 18.5 to 25; reference), underweight (BMI of less than 18.5), overweight (BMI of 25 to 30), or obese (BMI of above 30).

The following measures of socioeconomic status are taken from wave 1. Education is coded as a continuous variable indicating number of years of school completed. Household income indicates total household income during the last calendar year and is logged to correct for a skewed distribution. Net wealth is net household assets and is also logged.

In 1998, several questions were asked which assess childhood factors. To assess childhood health, the respondents were asked, "Consider your health while you were growing up, from birth to age 16. Would you say that your health during that time was excellent, very good, good, fair, or poor?" Following Haas (2007) the variable is coded 0 for fair/poor and 1 for good/very good/excellent. Unfortunately, this measure does not allow for the precise linking of specific childhood conditions to health trajectories. However, there is some information available that provides insight into what types of childhood conditions are captured by this measure. This measure is strongly associated with the experience of infectious (e.g. tuberculosis, polio), autoimmune (e.g. asthma, allergies), and non-infectious conditions (e.g. cancer, injuries) that resulted in substantial

physical limitation during childhood. Infectious and autoimmune conditions were most correlated with the probability of reporting poor childhood health.[1] However, infectious conditions were the most prevalent type of childhood condition reported in the HRS, representing half of all conditions reported (Blackwell et al. 2001).

Previous research has found that retrospective measures of childhood health perform reasonably well. It has been shown using data from the PSID and the HRS that this retrospective measure of overall childhood health is reliably reported over time (polychoric correlation =0.6; Goodman-Kruskal gamma=0.6), especially when the measure was dichotomized into a good/very good/excellent vs. fair/poor comparison (tetrachoric correlation=0.7; Goodman-Kruskal gamma=0.9) (Haas 2007). Quality of measurement did not vary substantially by gender or age. There is also no evidence that retrospective reports are subject to anchoring, by which current health status contaminates reports of health in childhood. Retrospective reports are also correlated with birth weight (Haas 2007). Elo (1998) has further demonstrated a high level of internal consistency between this measure and reports of specific long-term health limitations in childhood.

Krall and colleagues (1988) compared retrospective self reports of childhood communicable diseases, accidents, hospitalizations, surgeries, and other illnesses against a series of physical exams and parental interviews in a birth cohort. Retrospective childhood health questionnaires administered at ages 30, 40, and 50 showed a very high

[1] Analysis of the 1996 HRS experimental module by the author found that compared to those who did not experience a limiting childhood health condition, those who experienced a non-infectious, infectious, or autoimmune condition were 4.0 ($p<.0001$), 5.0 ($p<.0001$), and 13.2 ($p<.0002$) times more likely to report having poor health in childhood (good, fair, or poor), respectively. Unfortunately, the experimental module is only available for 733 respondents and does not permit a full analysis of the impact of specific childhood conditions on functional health trajectories.

level of accuracy (averaging 85% at age 50). Accidents and surgeries were recalled correctly 75% and 89% of the time at age 50, respectively. Reliability did not change much between ages 30 and 50, nor was recall accuracy correlated with education (Krall, Valadin, Dwyer, and Gardner 1988).

Parental education is measured as two continuous variables indicating number of years of school the respondent's mother and father completed. Questions assessing childhood SES indicate if the respondent's family was poor (0 = no, 1 = yes), if the respondent ever moved as a child due to financial difficulties (0 = no, 1 = yes), and if the father ever experienced a period of unemployment during the respondents' childhood (0 = no, 1 = yes). Parental education and the measure of overall family SES are designed to capture more permanent long-standing socioeconomic conditions in childhood while the later two measures are designed to asses the impact of specific economic shocks. Previous research has confirmed the quality of retrospective childhood SES reports (Krieger, Okamoto, and Selby 1998). Descriptive statistics for the variables used in the analysis are presented in table 1.

[Table 1]

Statistical Analysis

This study utilizes a two-part latent class growth mixture model to investigate racial and ethnic differences in trajectories of both the onset on any functional limitation and the number of limitations conditional on onset. A common problem with analyses of functional limitation and disability data is that the distribution of the outcome often takes on non-normal, semi-continuous characteristics in which there is disproportionate heaping at zero due to large numbers of individuals without any limitation. The

underlying assumption of the two-part model is that values of the outcome variable derive from two processes. The model splits the outcome into two random variables; a binary part representing the probability of onset of any functional limitation, and a continuous part which captures the magnitude of the nonzero values (if any limitations, how many). In addition to adjusting for the non-normality of the data due to over dispersion of zeros, conceptually, the model also provides an efficient way of simultaneously modeling the onset and severity of functional limitation. While a cross-sectional version of the two-part model has been in use for decades (Manning et al. 1981; Duan et al. 1983), it was not until recently that this has been adapted to longitudinal data applications (Olsen and Shafer 2001; Tooze, Grunwald, and Jones 2002).

This model has a number of important advantages over other classes of models designed to deal with the potential bias of zeros such as Tobit regression and Heckman selection models. Perhaps most important is that the underlying assumption about the process that generates zeros in the two part model fits better with the theoretical understanding of the disablement process. The two-part model treats zeros as representing true values (those without limitations) and not as either proxies for unobserved values (Heckman) or as censored or truncated (Tobit). In addition, unlike the Heckman model it does not require exclusion criteria (variables that predict the probability of having a zero response but are not otherwise associated with the number of limitations) to get proper estimation (Leung and Lu 1996; Puhani 2000).

In the current application, the observed response variable Y_{ij} represents the number of functional limitations for individual i at moment j. This variable can be recoded into two different random variables,

$$U_{ij} = \begin{cases} 1 & \text{if } Y_{ij} > 0, \\ 0 & \text{if } Y_{ij} = 0, \end{cases}$$

and

$$V_{ij} = \begin{cases} \ln(Y_{ij}) & \text{if } Y_{ij} > 0, \\ \text{missing} & \text{if } Y_{ij} = 0, \end{cases}$$

where $j = 1,\ldots, n_i$ indexes time points for individual $i = 1,\ldots, m$. For each individual i two correlated random effects growth curves are then estimated. The first growth curve, for the probability that the outcome takes on nonzero values (logit P(U_{ij}=1)), is

$$\eta_i = \text{logit}\,(\pi_i) = X_i^T\,\beta + Z_i^T\,c_i,$$

where $U_{it} \sim$ Bernoulli(π_{it}), π_{it} = P(U_{it}=1), $\pi_i = (\pi_{i1}, \pi_{i2},\ldots)^T$, and X_i^T and Z_i^T are matrices of covariates for the fixed and random effects. The second growth curve, for the mean response value when the outcome takes on non-zero values (E(V_{it} | U_{it}=1)), is expressed as a linear model

$$V_i = X_i^{*T}\,\gamma + Z_i^{*T}\,d_i + \varepsilon_i,$$

where $\varepsilon_i \sim N(0,\sigma^2)$ and V_i is a vector of V_{ij} for all j such that U_{ij}=1. A final important feature of the model is that the two random effects c_i and d_i are assumed to be jointly normal and can be correlated,

$$b_i = \begin{pmatrix} c_i \\ d_i \end{pmatrix} \sim N\left(0\,,\quad \Psi = \begin{pmatrix} \Psi cc & \Psi cd \\ \Psi dc & \Psi dd \end{pmatrix}\right).$$

Estimation is accomplished in Mplus (5) using Full Information Maximum Likelihood (FIML). See Olsen and Schafer (2001) for a detailed description of the model and its estimation.

A path diagram for the model is presented in figure 1. U_1-U_6 represent the binary responses corresponding to the time-specific onset variables (U_{ij}). V_1-V_6 represent the continuous responses for the natural log of number of limitations at each observation conditional on having any limitation (V_{ij}). IU (intercept) and SU (slope) represent the latent growth factors for the binary part of the model, while IV (intercept) and SV (slope) represent the latent growth factors for the continuous part of the model. Finally, the known latent class, C, identifies race-ethnic subpopulation groups in which functional health trajectories are allowed to vary.

[Figure 1]

The analysis proceeds in three steps. We first conduct omnibus tests for racial and ethnic differences in growth trajectories by comparing nested unconditional two-part growth mixture models under various constraints. The initial model allows the means and variances of the latent variables to vary across racial/ethnic group. To this we compare models in which means and then variances are constrained to be invariant across race/ethnicity.

Second, to investigate the determinants of racial/ethnic disparities in functional health trajectories we estimate a series of models with race/ethnic groups as a covariate (without latent classes) and include various childhood and adult adjustment factors to see what impact they have on ethnoracial differences in growth parameters. Model 1 adjusts only for race/ethnicity and demographic background. Model 2 adjusts for demographic background, childhood health, and socioeconomic status. Model 3 adjusts for demographic background and adult SES. Model 4 for adjusts for demographics and adult

health factors. Model 5 adjusts for demographics, childhood health and SES, and adult SES. Model 6 (full model) adjusts for all covariates.

The final step of the analysis investigates the differential impact of life course factors on race and ethnic group trajectories of functional limitation. This is accomplished by estimating a series of nested conditional growth mixture models (again treating ethnoracial group as latent classes) that allow the impact of covariates to vary by ethnic group. All conditional models control for gender, birth cohort, marital status, and geographic region. Model 1 additionally accounts for childhood health and socioeconomic status. Model 2 controls for adult SES and health characteristics. Finally, the full model (model 3) includes all covariates.

RESULTS

Ethnoracial Differences in Functional Health Trajectories

Table 2 presents results from the omnibus tests for racial/ethnic differences in growth trajectories of functional limitation onset and severity. This analysis reveals significant racial and ethnic differentials in functional health trajectories. We first fit a model in which the means and variances of the latent constructs are allowed to vary across race/ethnicity. Imposing the constraint that the means of the latent growth factors be equal across racial and ethnic group results in a substantial deterioration in model fit based on both the Satorra-Bentler scaled-likelihood ratio test ($\Delta \chi^2$ (df)= 622.51 (10)) (Satorra and Bentler 2001) and the Bayesian Information Criterion (Δ BIC=473). Constraining the variances of the latent growth factors to be equal across race/ethnic group results in a significant decline in fit based on the scaled-chi-square test ($\Delta \chi^2$ (df)= 16.59 (8)). However, BIC, which strongly favors parsimony, is reduced by 34, suggesting

that the constrained model is preferred. In the conditional models below, we present the parameter estimates from the unconstrained model. However, we recognize that a model with variances constrained across race/ethnic group is also plausible.

[Table 2]

Growth parameters by race/ethnicity based on model 1 are presented in table 2. Relative to Hispanics, non-Hispanic whites are significantly less likely to have any baseline functional limitation. Baseline risk of any limitation for blacks is not significantly different than that of Hispanics. Whites have slight higher average growth in limitation onset than Hispanics and blacks. Among those who have any baseline limitation, Blacks and Hispanics have more than whites, however, the later has higher rates of increase over time suggesting that disparities in functional limitations would tend to decline over time.

Life Course Determinants of Ethnoracial Disparities in Functional Health Trajectories

Having documented significant ethnoracial variation in functional health trajectories, we now turn to investigating the determinants of that variation. Table 3 presents estimates of racial and ethnic differences in latent growth trajectories of functional limitations with adjustment for various life course health and socioeconomic factors. Conditional on demographic background (model 1), both blacks and Hispanics have significantly elevated risk of functional limitation onset and severity compared to non-Hispanic whites. This echoes the results from table 2. After adjusting for both childhood health and SES (model 2), racial and ethnic differences in the baseline odds of having a functional limitation go away. However, the estimated racial and ethnic differences in severity, though reduced, remain large and significant. Adjusting for adult SES (model 3) results

in a complete reversal of the association between race/ethnicity and the baseline probability of having any limitation. Net of adult SES, blacks and Hispanics are less likely to have any limitation at baseline. There is also substantial attenuation of the association between race/ethnicity and baseline severity. The black-white differential in the number of baseline limitations is reduced by 75%. However, the average black respondent with any limitation continues to have significantly more limitations than the average white respondent who has limitations. Controlling adult health characteristics (model 4) completely attenuates the black white differential in the baseline odds of having and limitation. However, the difference between whites and Hispanics is not affected. Adult health status also attenuates the black-white differential in the baseline severity of limitation (by 45%), however a large and significant racial disparity is still present.

[Table 3]

Model 5 adjusts for demographic background, childhood health and childhood and adult SES. In this model blacks are no longer have increased odds of having baseline limitation, however, among those who have limitations, blacks continue to be more limited than whites. Adjusting for these factors Hispanics are significantly less likely to have any limitation at baseline. Otherwise the growth trajectory for Hispanics is not significantly different from that of non-Hispanic whites. Finally, after adjusting for all covariates (model 6) blacks are not significantly less likely to have any functional limitation at baseline. In addition, there are no longer any significant racial and ethnic differentials in the trajectories of limitation severity conditional on onset.

Ethnoracial Differences in Predictors of Functional Health Trajectories

Racial and Ethnic Disparities in Functional Health Trajectories

The final part of the analysis investigates racial/ethnic differences in the impact of life course factors on functional health trajectories. Maximum likelihood parameter estimates derived from the two-part growth model are presented in tables 4-6 for non-Hispanic whites, non-Hispanic blacks, and Hispanics, respectively. The parameter estimate for intercept of the binary part is interpreted in the same fashion as a traditional logistic regression coefficient. Positive values are associated with factors that increase the log odds of having any functional limitation while negative values are associated with factors that lower the log odds of having a functional limitation. The slope coefficients of the binary part are interpreted as reflecting the impact of the variable on the probability of onset over one-time unit (in this case 1 year). Similarly, estimates from the continuous part of the model are interpreted as the effect on the mean number of limitations conditional on having any. The intercept reflects the conditional mean number of limitations at baseline (1994) and the slope coefficients reflect deviations from the average rate of change in the number of limitations over time.

[Table 4]

Model 1 of tables 4-6 (columns 1-4) present the association between childhood health and socioeconomic status and the latent growth factors of functional limitation controlling for age, gender, geographic region and marital status. White women have 3.4 times greater odds of having any functional limitation at baseline and among those who already have limitation, they have 25% more than their male peers. These gender differences are even more pronounced for racial and ethnic minorities. Black women are 5.7 times more likely to report any baseline limitation and overall report 34% mobility

limitations than their male peers. Hispanic women are 6.8 times more likely to be limited at wave 1 and have 32% more limitation than Hispanic men.

[Table 5]

For non-Hispanic whites and blacks childhood SES is strongly associated with adult functional health trajectories. Among whites, a year of maternal and paternal education is associated with a 6% and 8% reduction in the odds of baseline limitation respectively, and with 2% fewer limitations among those who have any. Similarly, those who came from poor families, or who experienced childhood economic shocks experienced more functional limitations at baseline. Among blacks, each additional year of mother's education reduces the number of baseline limitations by 3% while each additional year of father's education reduces the probability of having any limitation at baseline by 12%. Blacks whose father experienced unemployment were also 2.3 times more likely to have any functional limitations at baseline. Blacks whose families were forced to move due to financial problems also experienced larger growth in limitations over time. However, among Hispanics there is no evidence that disadvantaged childhood SES is associated with functional health trajectories. For whites and blacks the experience of good childhood health is also associated with 64% and 72% decreased odds of having any functional limitation at baseline, respectively. Good childhood health is also 31% and 39% fewer limitations among whites and blacks who have any limitations.

[Table 6]

Model 2 (columns 5-8) investigates the affect of adult socioeconomic and health factors on trajectories of functional limitation onset and severity. For whites each additional year of education reduces baseline risk of functional limitation by 16% and the

number of baseline limitations by 4%. For blacks and Hispanics the effects of education were slightly smaller. The marginal impact of each year of ones own education also has larger impact than an additional year of parental education. For whites a 1% increase in household income and wealth is associated with a 14% and 7% decrease in baseline risk of any functional limitation and with 7% and 2% fewer limitations, respectively. Adult health status was also associated with functional health trajectories. Among whites those who are smokers, overweight, or obese are 1.73, 1.48, and 4.26, more likely to have any baseline limitation, respectively. Each additional chronic health condition increases baseline risk by 325% and the number of baseline limitations by 26%. Smokers have faster rising risk of limitation onset relative to non-smokers. Whites who are overweight or obese also experienced greater rates of limitation onset and larger growth in the number of limitations over time. Among blacks, current smokers, the obese, and those with more chronic health conditions have increased risk of baseline limitation. Current smokers and those with greater chronic disease also have a larger number of baseline limitations. Among Hispanics, current smokers, the obese and those with more chronic health conditions are more likely to have any limitation at baseline. However, only the later is associated with increased number of baseline limitations.

Model 3 (columns 9-12) presents the full models that control for both childhood and adult health and SES. Among whites, controlling for adult health and SES completely attenuates the effect of childhood SES on functional health trajectories. The effect of childhood health is reduced by about half though remains significant. Whites who report good childhood health had 41% reduced risk of baseline limitation and 17% fewer limitations than their unhealthy peers. Among blacks, the impacts of childhood

SES are also attenuated though significant effects of father's education and childhood economic shocks remain. The impact of childhood health on functional health trajectories is also no longer significant for blacks. This would suggest that for whites, childhood SES and health impacts functional health trajectories largely through their effects on adult health and SES. However, among blacks, childhood socioeconomic disadvantage is not mediated by subsequent adult socioeconomic attainment.

DISCUSSION

The above analysis reveals large racial and ethnic disparities in functional health trajectories. Non-Hispanic whites have lower risk of having any baseline limitation than blacks and Hispanics. Among those with some limitation at baseline, whites report fewer limitations than their black and Hispanic peers. The results also show that for whites and blacks, though not Hispanics, both childhood and adult factors are important determinants of adult functional health trajectories. This confirms previous research that has found childhood health and SES to be significant predictors of a wide variety of adult health outcomes (Haas 2007; Lou and Waite 2005; Blackwell et al. 2001; Hart et al. 2000). For blacks and whites poor childhood health significant adverse impacts on adult functional health trajectories. However, for both groups the impact of childhood health was strongly mediated through subsequent health and socioeconomic attainment in adulthood. That is, poor childhood health adversely impacts adult functional health trajectories in part by selecting individuals into lower socioeconomic strata (Haas 2006) and by increasing the risk of disabling adult health conditions (Haas 2007; Blackwell et al. 2001).

Racial and Ethnic Disparities in Functional Health Trajectories

The analysis also sheds light on other notable aspects of racial and ethnic disparities in these trajectories. Racial and ethnic disparities in functional health trajectories result from both differences in the processes related to limitation onset as well processes that determine the severity of limitation over time. That is racial and ethnic inequalities in functional health result both from inequality in the probability of experience the onset of mobility limitations and from differential growth in their severity over time.

In addition, racial and ethnic disparities in functional health trajectories result from both differential risk exposures as well as from differential impact of the risks themselves by race/ethnicity. There are notable differences in the effects of covariates across racial and ethnic group. For example, gender differentials in health trajectories are much more pronounced among racial and ethnic minorities. While the marginal impact of chronic health conditions was fairly consistent across race/ethnicity, blacks have 40% more chronic conditions at baseline than do whites and Hispanics. In the case of marital status, not only did the marginal effects of marital status vary across racial and ethnic group, there are significant group differences in the distribution of marital statuses. Similarly, while the effect of childhood SES for whites was completely attenuated with the inclusion of adult health and SES, this was not the case for blacks, where it continued to have independent effects on trajectories. The opposite was true for childhood health. Childhood health and socioeconomic conditions were not strong predictors of functional health trajectories at all for Hispanics.

Finally, among the covariates investigated here, adult health factors were most associated with onset and severity of functional limitations over time, especially among

whites. Childhood health was never associated with differential rates of onset or severity over time and childhood SES was associated with the change components of onset and severity only rarely. This conflicts with previous analysis that has shown that childhood factors were more strongly associated with trajectories of change in mobility limitations over time than were adult health and SES (Haas 2008). However, the previous analysis utilized standard growth models and thus did not account for differences in growth patterns of onset and severity.

This study highlights the complexity of racial and ethnic disparities in functional health trajectories. By documenting racial and ethnic differences in trajectories of onset and severity of functional limitation it affirms the importance of contextualizing ethno-racial inequalities in functional health within the large disablement process. This echoes the findings of Zsembik and colleagues (2000) who observe important racial and ethnic differences in the underlying disablement process. It further suggests that substantial understanding of ethno-racial health inequalities be understood as embedded within the life course with attention paid to both differences in exposures that elevate health risks but also to the understanding how the impacts of those exposures may vary between groups.

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Table 1. Weighted Descriptive Statistics By Race/Ethnicity (HRS 1992-2004)

	Non-Hispanic Whites			Non-Hispanic Blacks			Hispanics		
	%	Mean	SD	%	Mean	SD	%	Mean	SD
Functional Limiations									
1994		1.90	2.49		2.80	3.19		2.80	2.03
1996		1.93	2.61		2.83	3.28		2.52	3.03
1998		1.99	2.61		2.93	3.31		2.55	2.99
2000		2.10	2.69		3.09	3.39		2.74	3.17
2002		2.30	2.72		3.26	3.32		2.90	3.14
2004		2.48	1.79		3.19	3.16		3.21	3.18
Childhood Health and SES									
Childhood Health (Good-Excellent)	94.0			91.2			90.8		
Mother's Education		10.00	3.16		8.33	3.36		5.02	4.06
Father's Education		9.68	3.64		7.74	3.71		5.49	4.44
Family SES (Poor)	27.9			41.8			41.7		
Family Moved Due to Financial Problems	16.6			16.8			20.0		
Father Ever Unemployed	19.3			15.6			14.4		
Current SES									
Education (years)		12.73	2.63		11.31	3.19		8.75	4.48
Household Income (log)		10.56	1.23		9.88	1.77		9.84	1.75
Household Wealth (log)		11.07	3.84		8.01	5.79		8.96	4.63
Demographic									
Male	48.1			44.3			47.2		
Age		55.97	2.19		55.96	3.15		55.63	3.16
Married	78.1			49.1			67.3		
Northeast	22.6			20.6			13.9		
Midwest	26.7			19.2			5.2		
South	32.3			50.7			40.4		
West	18.4			9.5			40.5		
Health Factors									
Current Smoker	26.5			31.3			25.2		
Former Smoker	38.2			32.3			32.7		
Body Mass Index									
Underweight	1.3			1.6			1.0		
Overweight	33.9			22.0			23.4		
Obese	41.3			40.2			45.0		
# Chronic Conditions		1.02	1.09		1.39	1.24		0.99	1.12
N		7,196			1,723			941	

Table 2. Model Fit Iindices and Curve Parameters for Unconditional Two-Part Latent Growth Model of Functional Limitations (HRS 1994-2002).

	Model Comparison			
	log-likelihood	χ^2 (df)	P-Value	BIC
1. Free Means and Variances	-48885	—		97954
2. Model 1 Plus Equality of Means	-49151	622.51 (10)	<.0001	98427
3. Model 1 Plus Equality of Variances	-48891	16.59 (8)	0.0347	97920

	Growth Parameters[1]		
	Non-Hispanic White	Non-Hispanic Black	Hispanic
Binary Intercept Mean	-.393*	.067	—
Binary Intercept Variance	9.06***	8.18***	8.04***
Binary Slope Mean	.108***	.096***	.092***
Binary Slope Variance	.015***	.003	.010
Continuous Intercept Mean (log)	.522***	.836***	.831***
Continuous Intercept Variance	.545***	.620***	.575***
Continuous Slope Mean (log)	.030***	.025***	.016***
Continuous Slope Variance	.002***	.002***	.002***

Notes

χ^2=Satorra-Bentler scaled chi-square difference test

[1] Growth Parameters from Model 1

** p< .05; ** p< .01; *** p< .001*

Table 3. Racial/Ethnic disaparities in Latent Growth Trajectories of Functional Limitation (HRS 1994-2004)

	Non-Hispanic Black[1]				Hispanic[1]			
	Binary (Onset)		Continuous (Severity)		Binary (Onset)		Continuous (Severity)	
Model Adjustment Factors	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope
1. Demographics	.348**	0.003	.805***	-0.004	.556***	-0.017	1.020***	-.034*
2. Demographics, Childhood Health, Childhood SES	0.105	0.018	.568**	-0.007	-0.053	-0.015	.400**	-0.028
3. Demographics & Adult SES	-.329**	0.007	.200*	-0.002	-.683***	-0.01	-0.085	-0.026
4. Demographics & Adult Health	-0.195	-0.002	.448***	-0.009	.581***	-0.02	1.056***	-.037**
5. Demographics, Childhood Health, Childhood SES, Adult SES	-0.276	0.021	.231*	-0.007	-.729***	-0.01	-0.176	-0.024
6. Demographics, Childhood Health, Childhood SES, Adult SES, Adult Health	-.573***	0.018	0.083	-0.013	-0.262	-0.009	0.142	-0.021

[1] Parameter Estimates are Relative to Non-Hispanic Whites

** p< .05; ** p< .01; *** p< .001*

Table 4. Parameter Estimates from Two-part Growth Mixture Model of Functional Limitation Non-Hispanic White (HRS 1994-2004)

	Model 1				Model 2				Model 3			
	Binary (Onset)		Continuous (Severity)		Binary (Onset)		Continuous (Severity)		Binary (Onset)		Continuous (Severity)	
	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope
Demographic												
Age at Baseline	0.05**	0.00	0.00	0.00*	0.04**	0.00	0.00	0.00*	0.03	0.00	0.00	0.00**
Gender	1.21***	-0.01	0.25***	0.00	1.07***	0.00	0.23***	0.00	1.14***	0.00	0.23*	0.00
Marital Status												
Divorced	-0.01	0.06*	0.14**	0.00	-0.31	0.05*	0.01	0.00	-0.46*	0.06*	-0.01	0.00
Widow	0.26	0.03	0.14*	0.00	-0.40	0.03	-0.11*	0.01	-0.51	0.02	-0.07	0.00
Never Married	0.56*	-0.03	0.15*	0.00	0.17	-0.02	0.05	0.00	0.28	-0.02	0.07	0.00
Region												
Midwest	0.28	-0.01	0.01	0.01	0.29*	-0.02	-0.01	0.01	0.23	-0.02	-0.02	0.01
South	0.07	0.01	-0.03	0.01*	0.19	0.02	-0.01	0.01**	0.16	0.01	-0.02	0.01*
West	-0.20	-0.01	-0.05	0.00	0.22	-0.01	0.03	0.01	0.14	-0.02	0.02	0.01
Childhood Factors												
Mother's Education	-0.06**	0.00	-0.02**	0.00					-0.01	0.00	-0.01	0.00
Father's Education	-0.08***	0.00	-0.02***	0.00					-0.03	0.00	0.00	0.00
Family SES Poor	0.20	0.00	0.08*	0.00					0.02	0.00	0.03	0.00
Moved Due to Financial Probs.	0.31	0.03	0.10*	0.01					0.15	0.03	0.06	0.00
Father Ever Unemployed	0.33*	0.01	0.07*	-0.01					0.26	0.01	0.05	0.00
Childhood Health	-1.01***	0.02	-0.31***	0.00					-0.52*	0.02	-0.17***	0.00
Adult SES												
Education (years)					-0.17***	0.00	-0.04***	0.00	-0.14***	0.00	-0.03***	0.00
Household Income (log $)					-0.15***	0.00	-0.07***	0.00	-0.13*	-0.01	-0.06***	0.00
Household Wealth (log $)					-0.07***	0.00	-0.02***	0.00	-0.08***	0.01*	-0.02***	0.00
Adult Health												
Body Mass Index												
Underweight					0.47	-0.07	0.27**	-0.03*	0.51	-0.11	0.24*	-0.02
Overweight					0.39***	0.01	0.06*	0.01	0.47***	0.01	0.08**	0.01
Obese					1.45***	0.05**	0.24***	0.01***	1.56***	0.05*	0.25***	0.01***
Current Smoker					0.55***	0.06***	0.16***	0.01*	0.66***	0.04*	0.15*	0.001*
Former Smoker					0.05	0.03	0.04	0.00	0.13	0.01	0.05	0.00
# Chronic Conditions					1.18***	0.00	0.26***	-0.00*	1.14***	0.01	0.26***	0.00

** p< .05; ** p< .01; *** p< .001*

Table 5. Parameter Estimates from Two-part Growth Mixture Model of Functional Limitation Non-Hispanic Black (HRS 1994-2004)

	Model 1				Model 2				Model 3			
	Binary (Onset)		Continuous (Severity)		Binary (Onset)		Continuous (Severity)		Binary (Onset)		Continuous (Severity)	
	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope
Demographic												
Age at Baseline	0.03	0.00	-0.01	0.00	0.00	0.00	-0.01	0.00	-0.01	0.00	-0.02	0.00
Gender	1.74***	-0.06	0.34***	-0.01	1.66***	-0.06*	0.25***	-0.01	1.53***	-0.07*	0.32***	-0.01
Marital Status												
Divorced	0.93**	-0.07	0.10	0.00	0.14	-0.01	-0.02	0.00	0.49	-0.04	-0.08	0.01
Widow	0.04	0.00	0.17	-0.01	-0.18	0.03	0.01	0.00	-0.32	0.02	0.00	0.00
Never Married	0.96*	-0.05	0.16	0.01	0.45	-0.01	-0.03	0.02	0.72	-0.03	0.00	0.02
Region												
Midwest	-0.30	-0.02	0.05	-0.01	-0.20	-0.02	0.13	-0.01	-0.27	-0.01	0.10	-0.01
South	-0.45	0.00	-0.01	0.00	-0.59*	0.03	-0.01	0.01	-0.61*	0.01	-0.04	0.01
West	0.04	-0.04	0.17	-0.01	-0.10	0.03	0.23*	-0.02	-0.10	-0.02	0.21	-0.02
Childhood Factors												
Mother's Education	-0.03	0.00	-0.03*	0.00					-0.03	0.00	-0.02	0.00
Father's Education	-0.12*	0.00	-0.01	0.00					-0.09*	0.00	0.00	0.00
Family SES Poor	-0.02	0.03	0.03	0.00					-0.21	0.02	-0.02	0.00
Moved Due to Financial Probs.	0.29	-0.06	-0.17	0.02*					0.22	-0.07	-0.21**	0.02*
Father Ever Unemployed	0.82*	0.02	0.18	0.00					0.80*	0.01	0.13	0.00
Childhood Health	-1.44**	0.13	-0.39**	0.01					-0.81	0.11	-0.17	0.01
Adult SES												
Education (years)					-0.09**	0.00	-0.04***	0.00**	-0.06	0.00	-0.05***	0.00*
Household Income (log $)					-0.15*	0.01	-0.05***	0.01*	-0.22**	0.00	-0.07***	0.01*
Household Wealth (log $)					-0.03	0.00	-0.01***	0.00	-0.01	0.00	-0.02**	0.00
Adult Health												
Body Mass Index												
Underweight					1.41	-0.14	0.27	0.01	7.44***	-0.79*	0.48	0.01
Overweight					-0.04	0.07	-0.05	0.01	-0.09	0.07	-0.04	0.01
Obese					0.55**	0.07	0.11	0.00	0.71*	0.08	0.13	0.00
Current Smoker					0.55**	-0.04	0.14*	0.01	0.73*	-0.04	0.15	0.01
Former Smoker					0.23	-0.07*	-0.01	0.00	0.29	-0.06	0.01	0.00
# Chronic Conditions					1.24***	-0.01	0.29***	0.00	1.20***	-0.02	0.25***	0.00

** p< .05; ** p< .01; *** p< .001*

Table 6. Parameter Estimates from Two-part Growth Mixture Model of Functional Limitation Hispanic (HRS 1994-2004)

	Model 1				Model 2				Model 3			
	Binary (Onset)		Continuous (Severity)		Binary (Onset)		Continuous (Severity)		Binary (Onset)		Continuous (Severity)	
	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope
Demographic												
Age at Baseline	0.06**	0.00	0.02	0.00	-0.01	0.00	0.01	0.00	0.00	0.01	0.01	0.00
Gender	1.92***	-0.08	0.32***	0.00	1.30***	-0.05	0.19**	0.00	1.54***	-0.06	0.23**	0.00
Marital Status												
Divorced	0.87	-0.09	0.35***	-0.01	-0.09	-0.08	0.09	-0.01	-0.34	-0.09	0.07	-0.01
Widow	1.33	-0.18*	0.11	0.01	0.41	-0.15	0.06	0.01	0.60	-0.18*	-0.02	0.01
Never Married	0.49	0.15	0.24	0.00	0.05	0.16	0.10	0.01	-0.12	0.16	0.09	0.00
Region												
Midwest	-2.71***	0.17	-0.36*	0.02	-1.96***	0.09	-0.39**	0.03	-2.46***	0.14	-0.27	0.02
South	-0.94	0.10	-0.06	0.03	-1.21**	0.09	-0.14	0.02	-1.48**	0.11	-0.12	0.02
West	-0.58	0.02	-0.05	0.02	-0.72	0.03	-0.15	0.03*	-0.93	0.02	-0.08	0.01
Childhood Factors												
Mother's Education	0.01	0.00	-0.02	0.00					0.03	-0.01	-0.02	0.00
Father's Education	-0.04	0.00	0.00	0.00					0.03	-0.01	0.01	0.00
Family SES Poor	0.43	0.00	0.09	-0.01					0.23	0.00	0.01	0.00
Moved Due to Financial Probs.	0.20	-0.07	-0.03	0.01					-0.28	-0.08	-0.12	0.00
Father Ever Unemployed	0.12	-0.02	-0.02	0.00					0.56	-0.04	0.06	0.00
Childhood Health	-0.30	-0.12	-0.17	0.01					0.03	-0.10	-0.09	0.01
Adult SES												
Education (years)					-0.09**	0.01	-0.02***	0.00	-0.10*	0.01	-0.02*	0.00
Household Income (log $)					-0.18*	0.01	-0.03	0.00	-0.28*	0.03*	-0.02	0.00
Household Wealth (log $)					-0.07*	0.00	-0.02***	0.00	-0.06	0.00	-0.02**	0.00
Adult Health												
Body Mass Index												
Underweight					0.23	0.02	0.05	0.00	0.73	-0.13	0.30	0.01
Overweight					0.38	-0.05	0.10	0.01	0.39	0.03	0.14	0.02
Obese					0.88*	0.00	0.16	0.02	1.18**	0.03	0.20*	0.02
Current Smoker					0.86*	0.04	0.15	0.00	1.17**	0.02	0.18	0.01
Former Smoker					0.02	0.04	-0.02	0.00	-0.15	0.04	-0.01	-0.01
# Chronic Conditions					1.39***	0.01	0.28***	0.00	1.37***	0.02	0.29***	0.00

** p< .05; ** p< .01; *** p< .001*

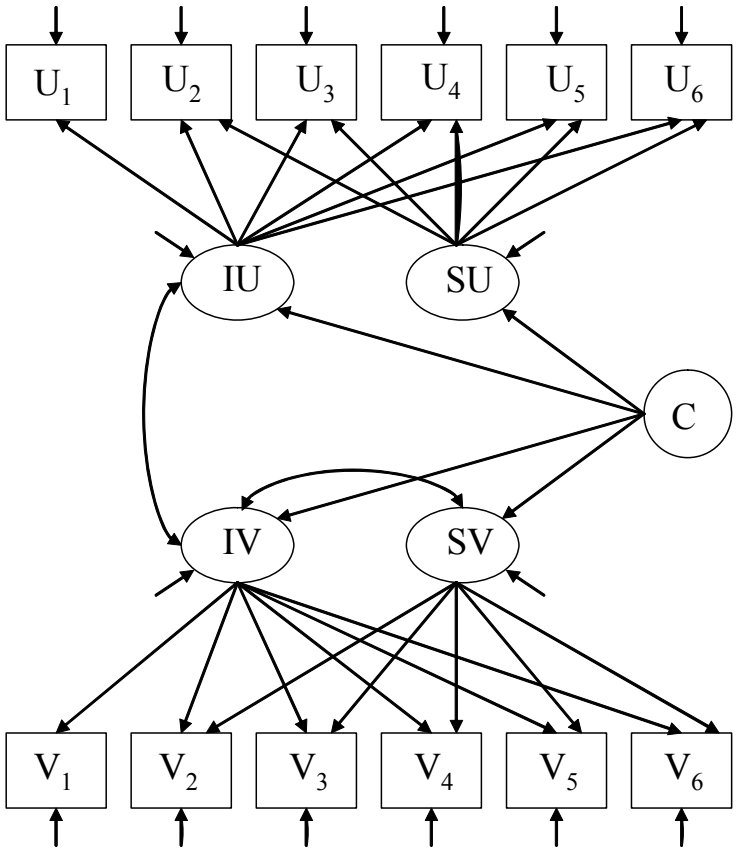

Figure 1. Path Diagram of Two-part Latent Class Growth Mixture Model.