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Pathways of Health and Human Capital from Adolescence into Young Adulthood

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Abstract

Social inequalities in health and human capital are core concerns of sociologists, but little research examines the developmental stage when such inequalities are likely to emerge—the transition to adulthood. With new data and innovative statistical methods we conceptually develop, and empirically operationalize, pathways of physical health and human capital accumulation from adolescence into young adulthood, using an autoregressive cross-lagged structural equation model. Results reveal that pathways of health and human capital accumulate at differential rates across the transition to adulthood; evidence of cross-lagged effects lend support for both social causation and health selection hypotheses. We then apply this model to assess the presence of social inequality in metabolic syndrome—the leading risk factor of cardiovascular disease in the U.S. Findings document social stratification of cardiovascular health that is robust to both observed and unobserved social and health selection mechanisms. We speculate that this social stratification will only increase as this cohort ages.

INTRODUCTION

Social inequality is a fundamental sociological concern. Decades of research have documented the trends, prevalence, and correlates of social stratification (Blau and Duncan 1967, Featherman and Hauser 1978, Hout and DiPrete 2006, Hout 2007, Grusky 2008). However, we still have much to learn about how inequalities develop across the early life course, from adolescence into young adulthood. Most social stratification research focuses on the quantity, quality, and access to human capital. Less attention has been paid to the development of physical health inequalities across the life course [Palloni (2006) excepted] and their relation to human capital development.

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Using rich longitudinal data, this study conceptually develops, and empirically operationalizes, intra-generational pathways of physical health and human capital accumulation from adolescence into young adulthood. This is the first major contribution of our study. We then gauge the impact of these pathways on cardiovascular health in young adulthood—our study’s second major contribution—as this is highly predictive of health and wealth in old age.

Health and Human Capital across the Transition to Adulthood

According to Blau and Duncan’s (1967) seminal work, social stratification has its roots in both inter- and intra-generational processes: father’s education and occupation (‘social origins’) influences child’s education, and each contributes to child’s occupation (‘social destination’). Subsequent conceptualizations affirmed this model (Featherman and Hauser 1978), spawning a generation of social stratification research.

A common approach, and the one we take here, is to focus specifically on intra-generational processes, whereby early investments in human capital demonstrate lifelong gains for individuals by maximizing physical health as well as psychological and economic wellbeing (Ross and Wu 1996, Sewell and Hauser 1975, Aneshensel 1992). A separate literature takes a longitudinal approach, noting a pattern of path dependence in which poor physical health is sequentially contingent from one life stage to the next (O’Rand 2009, Willson, Shuey, and Elder Jr 2007). However, neither literature has examined how human capital and health pathways develop and interact across the transition to adulthood. This agenda is challenging in multiple ways.

Concerning human capital, there are conceptual and measurement difficulties in estimating pathways linking income potential across early life stages (Furstenberg 2000, Furstenberg, Brooks-Gunn, and Morgan 1989). Most adolescents live with their parents, are in the midst of their educational trajectories, and do not work full-time; thus, current income, educational level, or occupation are poor proxies for their eventual level of human capital. Similarly, individual income is an unreliable indicator of human capital in young adulthood when some youth attend college while others move quickly into the labor market. We contribute new conceptual and measurement work in this area by estimating a pathway of human capital accumulation across the transition to adulthood using latent constructs that capture human capital at different life stages.

Concerning physical health, much past work either constructs trajectories of health only across adulthood, or builds trajectories of health spanning childhood and adulthood by asking adults to retrospectively report childhood illness and general health (Chen, Yang, and Liu 2010, Warren 2009, Willson, Shuey, and Elder Jr 2007, Palloni 2006, Haas 2006, 2008, Marmot et al. 2001). The latter approach suffers from recall bias, measurement error (Looker 1989), and mortality selection (Pollitt, Rose, and Kaufman 2005) as only individuals surviving to mid- to late- adulthood are surveyed. Other studies use longitudinal data to identify health pathways from adolescence through young adulthood (Bauldry et al. 2012) or links between adolescent health and key educational outcomes (Jackson 2009), but employ a single, subjective indicator of physical health (self-reported health). Moreover, these studies face the difficulty of estimating causal effects of social factors on health, aside

from key unmeasured factors (Goldman 2001, Palloni 2006, Kawachi, Adler, and Dow 2010, Adler, Bush, and Pantell 2012). All in all, we know relatively little about how early life health habits and behavior manifest within individuals over the early life course. We conceptually develop and estimate pathways of physical health, based on multiple objective and subjective measures of health, across the transition to adulthood. We also address the endogeneity of health (and human capital) by using multiple time-varying instrumental variables to identify the system of equations in our model.

In addition, health and human capital development are likely interdependent. In adulthood, poor physical health is associated with lower levels of educational attainment, lower income, fewer hours worked in the labor market, and ultimately less accumulation of wealth over time (Haas 2006, 2008, Currie and Madrian 1999, Jackson 2009), pointing to health as a primary driver of social stratification. In turn, socioeconomic disadvantage is associated with health detriments through greater exposure to stress and poor physical environments (Adler and Ostrove 1999, Adler, Bush, and Pantell 2012)—in neighborhoods and schools, within families, and in the labor market through work in low-wage jobs. Furthermore, these processes are likely deeply intertwined, reflecting a mutual interdependence of health and human capital with strong path dependent components (O’Rand 2001) that makes it difficult to produce unbiased estimates of either one (Kawachi, Adler, and Dow 2010, Adler, Bush, and Pantell 2012). We model these interdependencies and estimate their causal impacts.

Our study takes the additional step of estimating the effects of accumulated human capital and physical health on an important, objective marker of cardiovascular health in young adulthood, metabolic syndrome, that is measured using anthropometric measures and physiological markers of health. Metabolic syndrome is the leading risk factor for adult-onset cardiovascular disease and Type II diabetes—both of which encompass significant morbidity and mortality risk in the U.S. (Beltrán-Sánchez et al. 2013).

THEORY AND RESEARCH ON INTRA-GENERATIONAL DEVELOPMENT OF HEALTH AND HUMAN CAPITAL

The life course perspective is a useful organizing framework for understanding intra-generational development of human capital and physical health. While acknowledging the import of human agency (Hitlin and Elder 2007), events during childhood, adolescence, and adulthood are cumulative and are connected to broader structural context and social change (Elder, Johnson, and Crosnoe 2003, Elder Jr 1995). The cumulative disadvantage hypothesis articulates how later-life inequalities are anchored in early life experiences (Merton 1968, Ross and Wu 1996). These early life experiences have been broadly defined to include differential exposures to persistent poverty, social support, and stressful neighborhood environments (DiPrete and Eirich 2006).

The Importance of the Early Life Course

Striking distinctions in economic wellbeing are drawn between individuals who complete high school and those who do not (Hallinan 1988, Hogan and Astone 1986). High school completion is an important credential for the low-wage labor force and opens many

employment opportunities. Early returns to high school success are also evident in college attendance and completion; in turn, those who complete college benefit from more advantageous economic and employment opportunities in adulthood. Thus, adolescence and the transition to adulthood are critically formative for lifelong human capital.

Furthermore, as family-, employment-, and schooling-related transitions have become less structured, human agency has played an increasingly important role in the transition to adulthood (Shanahan 2000, Mortimer 1994). This is a time when individuals try on new roles and navigate a myriad of choices in different realms (labor market, dating market, etc.); behavior during this stage forms the basis for later-observed economic behavior (Zimmer-Gembeck and Mortimer 2006, Macmillan 2006).

Social inequalities in adult health are rooted in childhood experiences (Blackwell, Hayward, and Crimmins 2001, Hayward and Gorman 2004, Lynch, Kaplan, and Shema 1997). Childhood socioeconomic status (SES) has persistent effects on adult health outcomes, net of adult SES (Poulton et al. 2002). Identifying early life determinants of adult/old age health are active research questions (George 2005, Kawachi, Adler, and Dow 2010, Adler, Bush, and Pantell 2012). Furthermore, poor health in early life can constrain subsequent socioeconomic attainment and health maintenance (Elman and O’Rand 2007, O’Rand 2001). Yet, most studies rely on retrospective measures of early life events. Studies such as Add Health are advantageous because early life markers of social wellbeing and health are measured prospectively, and, in the case of health, with multiple subjective and objective indicators.

Much remains to be learned about the pathways linking early life exposures to adult health outcomes (Power and Hertzman 1997), although a growing literature proposes and tests potential pathways [see for example, (Almond and Currie 2011, Hertzman 2006, Hertzman and Power 2006, Kuh and Shlomo 2004, Ben-Shlomo and Kuh 2002)]. Most such studies however have insufficient data describing exposures during adolescence—a stage in which young people assert their independence and begin to manage lifestyle choices and health behaviors that often persist (Harris 2010, Macmillan 2006)—and the transition to adulthood—a life stage dense with critical transitions (e.g., leaving home, attending college, entering full time employment, entering a marriage or cohabitation, transitioning to parenthood) (Elder Jr 1995, Elder Jr, Johnson, and Crosnoe 2003, Shanahan 2000). These are periods when behavioral and health trajectories gain momentum and inequalities become entrenched.

Social Causation, Health Selection, and Mutual Interdependence

Physical health and socioeconomic status are tightly linked across the life course. *Social causation* suggests that early and sustained social and economic hardship leads to self-reinforcing cycles of poor health, including health problems such as obesity (Ferraro and Kelley-Moore 2003), hypertension, heart disease (Dupre 2007, 2008), cognitive decline (Rodgers, Ofstedal, and Herzog 2003), and higher rates of disability and mortality in older adulthood (Lynch, Kaplan, and Shema 1997, Smith and Kington 1997). *Health selection* posits that poor health decreases labor force participation [for a review, see Currie and Madrian (1999)], in turn, reducing financial capital and socioeconomic status (Haas 2006).

These interrelated processes (social causation and health selection) can induce upward or downward mobility across socioeconomic strata. Past studies have focused on which mechanism dominates. The bulk of evidence and argument suggests social causation (Chandola et al. 2003, Elstad and Krokstad 2003, Mulatu and Schooler 2002), which resonates with Link and Phelan's (1995) argument that social conditions determine access to health-related resources, thereby producing social inequalities in health. Lower levels of human or financial capital lead to lower access to, or utilization of, healthcare (Ross and Wu 1996), and are associated with lower levels of social support (Thoits 1995) and greater exposure to cumulative stress (McEwen 1998). Social causation processes are also supported by Mirowsky and Ross' (2003) notion of education as learned effectiveness: education improves health by increasing self-efficacy and problem-solving capacity. Others (Fox 1990, Haas 2006) have argued that both processes are at work (particularly in early life). However, other scholars place emphasis not on social causation *or* health selection, but rather on the mutual interdependence between health and human capital through which (dis)advantages accumulate (O'Rand 2001).

Conceptualizing Pathways of Physical Health and Human Capital across the Transition to Adulthood

We now introduce our conceptualization of physical health and human capital across the transition to adulthood. Physical health is a complex construct (Sartorius 2006); thus multiple indicators are useful and their selection depends on life stage. But generally, key indicators include: physical activity (or, activities of daily living, among older adults), body composition (e.g., adult body mass index or nutritional intake; infant birth weight), and/or health conditions (e.g., self-reported health, medical examinations, presence of chronic health conditions) (Bircher 2005, Huber et al. 2011). We strive for parsimony and replicability: we choose one indicator with face validity from each domain—level of physical activity, body mass index, and self-reported health.

For human capital, we follow past theoretical work in economics that defines investment in human capital as “activities that influence future real income through the imbedding of resources in people” [Becker (1962), page 9]. Thus, we conceptualize human capital as one's *potential for income* and seek to identify activities that embed resources within individuals. Of course, these activities will differ by life stage as the key institutions with which individuals interact change dramatically across the transition to adulthood.

During adolescence, key resource-embedding activities relate to education. Schools are crucial in the production of training; they transmit knowledge, skills, and resources to youth that influence their potential income (Becker 1962, Coleman 1988). The success of this transmission can be captured, at least in part, by markers of aptitude (e.g., cognitive ability) and school performance (e.g., assigned grades, being on track with age-related peers). These markers are also linked with college attendance—an important factor stratifying future employment opportunities and thereby future income.

Pathways through the transition to adulthood are less structured now than in the past (Shanahan 2000, Hogan and Astone 1986, Furstenberg, Rumbaut, and Settersten 2005). Still, most individuals enroll in college or enter the labor market in at least a part-time

capacity during the transition to adulthood (Mouw 2005, Sandefur, Eggerling-Boeck, and Park 2005)—decisions that are likely informed by youths' structural advantage, school performance, and anticipated socioeconomic gains of employment (Mortimer, Staff, and Lee 2005, Mortimer 2003). Both college and early full-time employment involve resource-embedding activities such as gaining credentials or employment. Conversely, remaining idle during this time is defined by exclusion—not working and not attending school (Shanahan 2000). More than a third of idle youth suffer from cognitive impairments (Amato et al. 2008), but, idleness outside of such impairment can be problematic as it restricts youths' investment in future income. Additionally, financial decisions in young adulthood are influential determinants of net worth later in life. Individuals holding low-risk financial assets (e.g., a savings or checking account) in early adulthood accumulate more assets by mid- to late-adulthood compared with those who are permanently asset-poor—meaning, never holding a checking account, savings account, bonds or stock, or never owning a home (Keister 2003). More generally, holding such assets within the transition to adulthood likely signals a foundation for longer-term advantageous behaviors (Zimmer-Gembeck and Mortimer 2006, Macmillan 2006, Shanahan 2000). In sum, activities reflecting potential for income during the transition to adulthood include education and employment (versus being idle), cognitive ability, and financial asset activity.

By young adulthood, many individuals have exited schooling and have entered the labor force (or engaged in caretaking) (Mouw 2005, Sandefur, Eggerling-Boeck, and Park 2005). At this stage, we conceptualize human capital as past literature focusing on mid- to late-adulthood—via employment behavior, educational attainment, and socioeconomic position, whether objective or subjective (Teachman, Paasch, and Carver 1997).

Current Study

This is the first study to prospectively estimate the development of physical health and human capital pathways from adolescence into young adulthood for a contemporary, nationally representative young adult cohort (Add Health). We use maximum likelihood estimation (MLE) for a structural, multi-equation model to estimate causal pathways of health and human capital development and their cross-lagged effects on young adult cardiovascular health. Throughout, we employ multiple measures of health and human capital at each time point to track developmental change.

Our study addresses three questions. *First*, how does physical health and human capital develop within individuals across the transition to adulthood? Does one life stage seem to be more critical than another? *Second*, is there evidence of persistent social inequality in adult health, above and beyond underlying selection mechanisms? *Third*, through what pathways do young adult health inequalities manifest?

DATA, MEASURES, AND ANALYTIC STRATEGY

Data

We use data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a school-based, nationally representative sample of 20,745 7th–12th graders in

1994–1995. Respondents were re-interviewed in 1996 (Wave II), 2001–02 (Wave III), and 2008–09 (Wave IV) (Harris 2010, Harris et al. 2009). Our sample includes male and (non-pregnant) female respondents participating in Waves I, III, and IV (as anthropometric measures of health are not comparable across pregnant and non-pregnant women; $n = 12,311$). We select native-born respondents ($n = 11,499$) based on complex differences in health status for foreign-born versus native-born individuals (Hummer et al. 1999, Singh and Miller 2003), particularly in early life (Crosnoe 2006). Respondents with a valid sampling weight are retained to produce nationally-representative estimates ($n = 9,033$). Missing data is minimal (<4% for most variables). We use a single imputation procedure in Stata to replace missing data on all independent variables. Results based on listwise deletion and full information maximum likelihood (in Mplus) were largely unchanged. Analyses are weighted and stratified to account for the sampling design.

Measures

As depicted in Figure 1, we constructed a latent variable reflecting poor health for each life stage represented in our data (Poor Health, WI, adolescence; Poor Health WIII, emerging adulthood; and Poor Health WIV, young adulthood) using the same three variables: self-reported health (1 = *excellent*, 4 = *fair or poor*), body mass index (range = 11.21 – 80.40)¹, and physical inactivity [bouts of physical inactivity in the last 7 days; range (WI) = 0–3; range (Waves III and IV) = 0–5].

Likewise, we constructed a latent variable reflecting human capital in each life stage (see Figure 1) using age- and developmental stage-appropriate measures. In adolescence, human capital is indicated by GPA (range = 1–4), Add Health Picture Vocabulary Test (AH PVT) score—a measure of cognitive ability (range = 14–146), and if the respondent was ever held back a grade (1 = *not retained*). In emerging adulthood, human capital is indicated by years of schooling (range = 6–22), AH PVT score (range = 7–122), if the respondent was employed or in school (1 = *yes*), and if the respondent had a checking and/or savings account (1 = *yes*). In young adulthood, years of schooling (range = 8–26), household income (range = \$0–\$150,000), employment status (1 = *employed*), and self-rated socioeconomic status (range = 1–10) characterize human capital.²

Similar to the clinical definition of metabolic syndrome (Grundy et al. 2005),³ a latent variable reflecting metabolic syndrome (shown in Figure 1) was constructed using five

¹We top-coded weight for 9 respondents whose weight exceeded the scale maximum (200kg/440lbs). Results were unchanged when these individuals were excluded.

²Pairwise correlations between latent variables:

	1	2	3	4	5	6
1. Poor Health–WI	1					
2. Poor Health–WIII	0.85	1				
3. Poor Health–WIV	0.79	0.93	1			
4. Human Capital–WI	-0.15	-0.11	-0.16	1		
5. Human Capital–WIII	-0.08	-0.09	-0.14	0.91	1	
6. Human Capital–WIV	-0.11	-0.13	-0.16	0.87	0.95	1

³Our measure diverges slightly from the clinical definition. Add Health participants were not asked to fast pre-interview. Rather than combine fasting and non-fasting glucose, we use HbA1c, a more stable measure of metabolic dysregulation. We also use membership in the highest decile for triglycerides; the clinical definition is indicated by 150mg/dL or drug treatment for elevated triglycerides. For HDL-C, clinical cutoffs are <50mg/dL for women and <40mg/dL for men, or drug treatment for reduced HDL-C. The laboratory assaying Add Health specimens for lipids used two different assays—a common occurrence. The two should be directly comparable

indicators: elevated waist circumference (*88cm for women and 102cm for men*), elevated blood pressure (*130mm Hg systolic blood pressure, 85mm Hg diastolic blood pressure, or antihypertensive drug treatment*), elevated triglycerides (*membership in highest decile of measured triglycerides*), reduced high-density lipoprotein cholesterol (HDL-C) (*membership in lowest 2 deciles of HDL-C, based on sex*), and pre-diabetic value of glycosylated hemoglobin (HbA1c) (*>5.6% of hemoglobin molecules are glycosylated*).

WI control variables include family structure (*1 = two biological parent family, 0 = all other family structures*), parent education (range = 0–9)⁴, (logged) household income-needs ratio, race-ethnicity, age, and respondent sex (*1 = female*). Additionally, we control for WIII residence in urban/non-urban census tract and WIV household composition (reside with parents, married or cohabiting, live alone).

Consistent with past research linking structural and economic features of the neighborhood environment to individuals' health and human capital [e.g., Kawachi and Berkman (2003)], we include multiple, contextual, time-varying instrumental variables for both: subjective indicators of neighborhood safety (if s/he knows most people in their neighborhood, stopped to talk to any neighbors in the past month, usually feels safe in his/her neighborhood), an objective indicator of neighborhood safety (juvenile violent crime rate), indicators of the economic landscape (Bureau of Labor Statistics cost price index⁵, per capita income, male unemployment rate, percent of female-headed households with children), and indicators of housing quality (percent of homes with insufficient plumbing, median year housing was built—before or after 1978). Additional instrumental variables of poor health include: (Euclidean) distance between the respondent's home and nearest park, age-specific mortality rate of 15–24 year olds, and the number of inactivity resources (i.e., movie theaters, arcades) within 1km of their home. In order to be plausible instruments, these factors must be correlated with health (or human capital) at a given time, but may not predict future human capital or health except through the lagged value.

The proposed instruments are plausible on conceptual grounds. Perceived neighborhood safety or Euclidian distance to the nearest park, for example, can regulate one's ability to engage in physical activity locally, with important implications for overweight status (Gordon-Larsen et al. 2006); however, these factors would not directly impact time₂ physical health, except through time₁ health behaviors and/or weight status. We also tested the assumptions of instrumental variables. Results based on *ivregress* post-estimation commands reject the null hypothesis of exogeneity (Wu-Hausman test: $p < .001$ for each instance) while the identification tests fail to reject the null hypothesis of correlation between the error terms in the structural equations and the instrumental variables this indicating that the exclusion restrictions are valid (WIII health: $\chi^2 = 6.11$, $p = .41$; WIV health: $\chi^2 = 1.18$, $p = .76$; WIII

using a simple algebraic transformation. After extensive data cleaning and checking efforts were performed, only the rank-ordering (by deciles) was released as it was deemed to be a more reliable measure. Thus, we indicate reduced HDL-C as membership in the lowest category for women (HDL-C=1; 7.0%) and the lowest two categories for men (HDL-C<3; 26.2%), based on evidence suggesting 11.9% of women and 31.4% of men exhibit reduced HDL-C (Carroll, Kit, and Lacher 2012). Results did not change when we defined reduced HDL-C by membership in the lowest two categories for women (HDL-C<3; 15.6%) and the lowest three categories for men (HDL-C<4; 37.2%).

⁴When this was missing, respondent's report of parental education was used.

⁵The BLS cost price index is only available for urban areas; thus, we control for Wave III residence in an urban census tract.

human capital: $\chi^2 = 4.24$, $p = .52$; WIV human capital: $\chi^2 = 6.19$, $p = .11$) (Sargan 1958). Furthermore, the instruments are sufficient in strength (WIII health: $F = 21.14$; 10% value for 2SLS = 10.27; WIV health: $F = 10.71$; 10% value for 2SLS = 9.08; WIII human capital: $F = 34.42$; 10% value for 2SLS = 10.27; WIV human capital: $F = 95.12$; 10% value for 2SLS = 9.08) (Stock, Wright, and Yogo 2002).

Analytic Strategy

We employ an autoregressive structural equation model (SEM) to estimate pathways of physical health and human capital, and cross-lagged effects; we also allow each pathway to have a direct effect on metabolic syndrome. From this base model, we relax a series of parameter constraints and compare five nested models to select a preferred model (see Table 1). For example, in the first column, the likelihood ratio test compares Model 1 (including none of the instruments, error covariances for latent variables, or cross-lags) to Model 2 (including instruments, but not error covariances or cross-lags); the statistical significance of the likelihood ratio test statistic indicates Model 2 is a better fit of the data. This was repeated until all parameter constraints noted were relaxed. The final column indicates Model 5 was the best fit overall. Thus, our preferred model includes cross-lags and error covariances across the system of equations jointly along with model parameters, and uses instrumental variables within the SEM to statistically identify causal effects for health and human capital at each life course stage. This strategy produces consistent estimates of the effect of health (or human capital) at one point in time on health (or human capital) at a subsequent time point, and effectively allows us to address the endogeneity of both health and human capital across time.

We estimate this preferred model using maximum likelihood estimation in Mplus 7. MLE produces efficient parameter estimates, assuming error terms follow a multivariate normal distribution. This is a tenuous assumption however, particularly when the true distribution is non-normal (Guilkey and Lance 2014, Mroz 1999), although very few studies compare how their parameter estimates differ with and without the normality assumption. We assess the extent to which our results are robust to this assumption by comparing MLE results to those produced by a two-stage least squares instrumental variables regression model, estimated in Stata 13.0 using the *ivregress* command—a statistical specification that does not assume normality but is less efficient. If model results are similar across both specifications, this would suggest joint normality is not violated and the more efficient estimation (MLE) is preferred. If model results are not similar, joint normality may be violated and the IV results are preferred.

We then estimate total, direct, and indirect effects to identify pathways through which health and human capital operate directly and indirectly on metabolic syndrome. Finally, we assess whether or not the endogeneity corrections we employ are necessary. Because the system of equations in our model is jointly estimated using full information MLE, this approach requires IVs for statistical identification. Therefore, to assess whether or not correcting for endogeneity makes a difference, we calculate OLS regression estimates, where we adjust for controls, lagged values of health and human capital, and cross-lagged values, but not for IVs or correlated error terms between latent variables. We refer to this as the uncorrected model.

RESULTS

Table 2 presents descriptive statistics of all study variables, weighted and adjusted for clustering of the sample design. The mean and variance of self-reported health remained somewhat stable over time, but rose steadily for BMI. Physical inactivity was lowest during high school, increasing within emerging and young adulthood. Average GPA in the current or past academic year was 2.79; 79% of respondents never repeated a grade. Average AHPVT score remained similar over time. Eighty-three percent of respondents were not idle in emerging adulthood; many had a checking (71%) or savings (63%) account. In this life stage, the average level of completed education was 13.1 years; by young adulthood it was 14.2 years. In young adulthood, two thirds (66%) were employed; average household income was \$60,125. Over half of sample members were classified with elevated waist circumference (52%) and just under half with elevated blood pressure (42%). Nearly a third met or surpassed the pre-diabetic cutoff for glycosylated hemoglobin (31%).

Selected parameters from the SEM are presented in Figure 2; all parameters are presented in Appendix A.⁶ Unstandardized (not italicized) and standardized (italicized) beta coefficients are presented with standard errors in parentheses.

Our first key question is: how does physical health and human capital develop within individuals across the transition to adulthood? Results demonstrate strong path dependence: all path coefficients linking poor health over time and those linking human capital over time are positive and statistically significant ($p < .001$). Comparing the standardized coefficients, first for poor health, shows that the path coefficient linking poor health in WI and WIII (standardized $b = .92$) is more than twice as large as the path coefficient linking poor health in WIII and WIV (standardized $b = .40$). Thus, path dependence appears to be stronger during later (versus earlier) stages of the transition to adulthood. Results from a Wald test suggest these two path coefficients cannot be constrained to be equal ($PH_1 \rightarrow PH_3$ $PH_3 \rightarrow PH_4$; test statistic = 24.46, $df = 1$, $p < .001$). Substantively, this indicates poor health appears to develop at a differential rate, rather than a constant rate, as individuals transition into adulthood.

Estimates of path dependence for human capital present a different pattern. The standardized path coefficient linking WI and WIII human capital (standardized $b = 1.01$) is slightly larger than the corollary path coefficient between WIII and WIV (standardized $b = .99$). A Wald test suggests the null hypothesis ($HC_1 \rightarrow HC_3 = HC_3 \rightarrow HC_4$) can be rejected (160.47, $df = 1$, $p < .001$), indicating that the human capital pathway crystalizes quickly in adolescence and is further solidified in young adulthood.

Next we address our second research question: is there evidence of persistent social inequality in adult health, above and beyond underlying selection mechanisms. The key estimate is the effect of young adult human capital on metabolic syndrome. This path

⁶Model fit indices presented indicate acceptable fit (RMSEA=.026; SRMR=.050). Other model fit indices (CFI, TLI) are below recommended levels. However, residual variances of latent variables are low (range, .01–.17) and R-squared values of latent variables are high (all but one is between .50 and .97), lending further evidence of adequate model fit. In accordance with recommendations for testing structural equation models (Bollen and Long 1993), we balanced these indications of model fit with indications based on theory and substantive experience when developing our model.

coefficient (unstandardized $b = -.02$, $p < .001$) indicates higher levels of young adult human capital are associated with lower risk of metabolic syndrome, net of all other pathways in the model, including the endogeneity corrections for underlying social selection and health selection mechanisms. Thus, we observe salient social stratification of metabolic syndrome in this contemporary cohort.

The statistical specification used to produce Figure 2 assumed the scaling indicator (elevated waist circumference) for metabolic syndrome was continuous; therefore, the path coefficients linking young adult health and human capital to metabolic syndrome are approximated by a linear probability model. To examine if study results were sensitive to this specification, we replicated the SEM scaling metabolic syndrome by the linear form of waist circumference, triglycerides, and hemoglobin A1c (one at a time). Results presented in Appendix B suggest study findings are extremely robust across different specifications of metabolic syndrome.

We also examined the extent to which the normality assumption imposed by MLE was upheld. Results (presented in Appendix C) indicate that a two-stage least squares instrumental variable regression specification produces similar, and at times, identical point estimates compared to the MLE. The standard errors produced in the IV model are wider, reflecting the decreased efficiency of this approach relative to MLE. The similarity across specifications provides strong evidence that the normality assumption imposed by MLE is not likely violated in this model. Therefore, the MLE results are preferred due to increased efficiency.

We now return to the full model to examine our final research question: through what pathways do young adult health inequalities manifest? We address this in two ways: we examine the cross-lags and then assess total, direct, and indirect effects. As depicted in Figure 2, cross-lags linking factors between WI and WIII are not statistically significant. However, the cross-lags between WIII and WIV are statistically significant at the $p < .001$ level. Both are negative, indicating that poor health and human capital are inversely related. The standardized coefficient for the path reflecting social causation is $-.04$; that reflecting health selection is $-.05$. Results from a Wald test suggest the null hypothesis ($HC_3 \rightarrow PH_4 = PH_3 \rightarrow HC_4$) can be rejected (7.161 , $df = 1$, $p = .008$). Thus, we conclude that both social causation and health selection are present during this life stage and appear to differ modestly in strength.

Table 3 presents a summary of total, direct, and indirect effects (poor health in Panel A, human capital in Panel B). The first coefficient shown represents the direct effect of WIV poor health on metabolic syndrome (unstandardized $b = .42$); this also appears in Figure 3. The second coefficient represents the total effect of WIII poor health on metabolic syndrome ($b = .45$). (With no direct effect here, the sum of the indirect effects are equal to the total effect.) The two indirect effects are: $Health_3 \rightarrow Health_4 \rightarrow Metabolic\ syndrome$ ($b = .45$), and $Health_3 \rightarrow Human\ Capital_4 \rightarrow Metabolic\ syndrome$ ($b = .002$).⁷ Even though

⁷Supplementary analyses (not shown) suggested that adding pathways from each WI latent variable to metabolic syndrome did not significantly improve model fit.

unstandardized betas are presented, the difference exhibited makes the relative association fairly clear. The last set of results in Panel A mirror this finding: the total effect of WI poor health on metabolic syndrome (unstandardized $b = .26$) is comprised of four indirect effects; the one largest in magnitude does not involve a cross-lag ($\text{Health}_1 \rightarrow \text{Health}_3 \rightarrow \text{Health}_4 \rightarrow \text{Metabolic syndrome}$; $b = .26$). Panel B depicts a very similar pattern of results for human capital. Thus overall, the key causal pathways reflect path dependence, a finding to which we return in the discussion below.

Finally, the supplementary analyses exploring the necessity of correcting for endogeneity show a markedly different pattern of results (see Appendix D). Relative to the SEM, the uncorrected OLS model tends to underestimate the path coefficients reflecting path dependence and social inequality in health (net of health selection). In most cases, the uncorrected model estimate is roughly half the magnitude of the SEM estimate. Moreover, the uncorrected model overestimates the cross-lags (in most cases, by more than double) and all four cross-lags are statistically significant—suggesting a starkly different substantive result. In other words, had we not corrected for endogeneity, we would have mistakenly placed less emphasis on the role of path dependence, more emphasis on the role of social causation and health selection, and we would have inaccurately concluded that social causation and health selection were operating throughout each stage across the transition to adulthood. Thus, it is necessary, on both statistical and theoretical grounds, to account for the endogeneity of poor health and human capital and include error covariances.

DISCUSSION

A vast literature documents social inequalities in health and human capital, but less is known about how these inequalities develop across the early life course. We trace the intra-generational development of these emerging pathways as adolescents transition into adulthood. In addition, little is known about the interdependence of human capital and poor physical health during the transition to adulthood. As we detail below, understanding more about inequality in both domains informs a range of sociology sub-disciplines, including social stratification, medical sociology and health inequalities. Furthermore, no research has yet examined the contributions of intra-generational development of poor health and accumulation of human capital on metabolic syndrome—an important marker for cardiovascular disease and Type II diabetes, two conditions that predict significant morbidity and mortality risks for U.S. adults.

A primary contribution of our study is the conceptualization and operationalization of pathways of health and human capital across the transition to adulthood. An empirical test of these pathways showed powerful evidence of path dependence: poor health in adolescence is strongly related to poor health in emerging adulthood, which in turn is related to poor health in young adulthood. The same holds for human capital. This in and of itself is not surprising, but results also show these pathways develop at differential rates over the transition to adulthood. The health pathway crystalizes more slowly between adolescence (WI) and emerging adulthood (WIII), but more rapidly between emerging and young adulthood. Substantively, this suggests that adolescence is a life stage in which health behaviors, such as food choices and levels of physical activity, are still being formulated, whereas, by

emerging adulthood, these behaviors are more entrenched—a finding consistent with past research (Harris et al. 2006). Conversely, the human capital pathway crystalizes strongly in adolescence and solidifies further in adulthood. This provides evidence of the notion proposed by life course scholars, that human agency, along with structural distributions of opportunity, together play an important role in the transition to adulthood as individuals enact human agency (e.g., open a savings/checking account) and begin to formulate health behaviors, both of which can become the basis of longer-term health and economic behaviors and outcomes in adulthood (Zimmer-Gembeck and Mortimer 2006, Macmillan 2006, Shanahan 2000, Hitlin and Kirkpatrick Johnson 2015). Additionally, this finding buttresses support for Heckman's (2000) call for greater emphasis to be placed on early intervention programs and policies, as human capital pathways are taking shape well before our study first measures them. Moreover, supplementary analyses showed that failing to account for endogeneity would have led us to inaccurately place less emphasis on path dependency within the model, as estimates from uncorrected OLS models were roughly half the magnitude of SEM estimates (that included endogeneity corrections and error covariances).

A second key contribution is strong evidence of emerging social stratification in metabolic syndrome for this cohort. Our study extends past research by showing that the estimated effect of young adult human capital on metabolic syndrome is statistically significant ($p < .001$), and robust to the inclusion of parental socioeconomic status, family background characteristics, and underlying social and health selection mechanisms (which we account for using numerous instrumental variables identifying poor health and human capital at each time point).

We speculate that the social stratification of metabolic syndrome signals the interplay between opportunity structures and human agency is already taking hold in the lives of this cohort, influencing a key marker of adult health. It may also reflect learned effectiveness—in that education, even in young adulthood, provides individuals with knowledge and skills that marshal personal control and enact human agency to produce healthy behaviors and a healthy lifestyle (Mirowsky and Ross 2003). Similarly, higher education provides access to advantageous employment opportunities, relative to low-wage work or unemployment, that accommodate/facilitate healthier lifestyles (Ross and Wu 1996).

Based on past studies describing rather large social inequalities in health, we anticipated finding a substantial direct effect of human capital on metabolic syndrome. Instead we observed a relatively small but statistically significant effect ($p < .001$); we speculate this stems from two sources. Metabolic syndrome is relatively rare early in life, becoming increasingly common with age [prevalence is 3% in children (Friend, Craig, and Turner 2013), 23% in adults age 19+ (Beltrán-Sánchez et al. 2013), and 44% in adults age 50+ (Alexander et al. 2003).] Therefore, detecting any degree of social stratification in metabolic syndrome within young adulthood, above and beyond endogeneity and selection bias, is a strong signal of things to come. Second, we measured metabolic syndrome objectively, using biomarker data, which are independent of self-reflection bias or social circumstances that are often correlated with subjective measures of health. For this reason, estimated effects of

objective health indicators are often smaller in magnitude than those based on subjective indicators (Wu et al. 2013).

We suspect that the social stratification we document will only increase as cohort members age. Specifically, mid- to late-adulthood is a time when morbidity and mortality risks due to health conditions linked with metabolic syndrome, such as cardiovascular disease, stroke, and Type II diabetes, become more pronounced. It is also a time when these conditions become increasingly concentrated among individuals within lower socioeconomic strata. This is influential as cardiovascular diseases are among the most expensive health conditions: one study estimated that 17% of all medical expenditures each year (totaling \$149 billion) can be attributed to cardiovascular diseases alone (Trogon et al. 2007). As these costs place an undue economic burden on the least advantaged, this will further stratify the health and wealth of this cohort as they age. Recent trends related to the prevalence of metabolic syndrome also suggest increased concentration among minority populations, and particularly among minority women (Mozumdar and Liguori 2011, Beltrán-Sánchez et al. 2013). As such, these trends may reinforce race/ethnic social stratification of adult health.

Furthermore, given recent trends pointing to an earlier onset of chronic diseases, such as obesity (Van Cleave, Gortmaker, and Perrin 2010), and rising prevalence of metabolic syndrome across the population (Mozumdar and Liguori 2011), the long-term implications of social inequalities in cardiovascular disease may far exceed what we have observed in the past. Observing these developmental trends now, among a contemporary population at the forefront of the obesity epidemic (Harris 2010), affords time to plan and implement prevention and intervention efforts among vulnerable populations. Our work suggests that interventions would be more impactful during these critical early life stages if they can curb metabolic dysregulation before it manifests in more harmful conditions.

The strength of the health and human capital pathways, and the importance of early and sustained investments in human capital permeate our results. But we also show important (both substantively and statistically) cross-lagged effects between poor health and human capital between WIII and WIV (as respondents move into young adulthood). Thus, our study is the first to pinpoint the stage within the transition to adulthood in which social causation and health selection operate. This is the third contribution of our study. Supplementary analyses showed that failing to account for endogeneity would have led to markedly different substantive conclusions, including inaccurately concluding that social causation and health selection operate throughout the entire span of the transition to adulthood, buttressing the importance of correcting for endogeneity in terms of accurately depicting these cross-lagged associations.

The standardized coefficients of the social causation ($-.04$) and health selection ($-.05$) cross-lags were statistically significant at the $p < .01$ level, and could not be constrained to be equal, according to a Wald test. In contrast, other studies demonstrate stronger effects of social causation for mental health in mid-adulthood (Chandola et al. 2003, Elstad and Krokstad 2003, Mulatu and Schooler 2002) and physical health in late adulthood (Warren 2009). Observing both social causation and health selection processes at work in our study is consistent with past scholarship emphasizing the mutual interdependence between the two

that operates in conjunction with strong path dependent components (O'Rand 2001), and resonates with a developmental perspective identifying the transition to adulthood as one in which youth create, form, and maintain social identities, health habits, and economic preferences simultaneously (Macmillan 2006). In this way, human capital and health in the early life course are substantively distinct from human capital and health in mid- to late-adulthood—two life stages in which social causation and health selection signal a distinct underlying process. In mid- to late-adulthood, poor health directly impacts labor market participation, earnings, and wealth accumulation (health selection). In turn, participation in the labor market affects health (social causation), partially through employment stability (ensuring stable earnings), access to health insurance through one's job, or through education itself via learned effectiveness (Mirowsky and Ross 2003). In contrast, aptitude and educational achievement are the important drivers of human capital development in the early stage of the life course (as opposed to labor market activity and income) and this development is both dependent upon and predictive of early life health pathways.

In sum, this study provides a general model that traces the development and persistence of health and human capital between adolescence and young adulthood. We focused on measures readily available in other datasets both within (e.g., NLSY97) and outside the U.S. (e.g., China Health and Nutrition Survey) to encourage future research. Future social stratification studies for example could trace the pathways through which ascribed characteristics (race-ethnicity, sex) shape inequalities at each time point, through constraints imposed by social structures and social systems. Other model extensions could chart variations in the accumulation of health and human capital by parental characteristics (education, income, occupation) or by interactions with social structures in childhood (family of origin, neighborhood, school), contributing a broader understanding of the development of the intergenerational transmissions of inequality. Exploring nuances in this model as individuals interact with health institutions or health professionals may contribute to the medical sociology literature.

We chose to apply the model to understanding social inequalities in cardiovascular health, but future work could apply the model to any number of social well-being and/or health markers in young adulthood, contributing to a number of sociological sub-disciplines. For instance, future research could also address how these pathways operate differentially across geographic location (contributing to urban and rural sociology), or how these pathways are disrupted by engagement in criminal activity (contributing to sociology of crime and deviance) or timing of family formation behavior (contributing to family sociology). We have in mind here the development of the Blau-Duncan model, where solving a measurement issue (operationalizing socioeconomic status) and laying out a causal framework facilitated a generation of comparable research findings. Extensions of this study's framework across numerous subareas of sociology could enrich our understanding of the longer-term social processes associated with the development of physical health and human capital development.

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Figure 1.

Measurement Model of the Accumulation of Poor Health and Human Capital across the Transition to Adulthood

Note: Path dependence is indicated by paths linking poor health over time and paths linking human capital over time. Social causation is indicated by paths linking human capital to poor health. Health selection is indicated by paths linking poor health to human capital. Social inequality in health net of health selection is indicated by the path linking WIV human capital to metabolic syndrome.

Table 1

Results from a Nested Model Comparison Test to Select a Preferred Model

Model 1: Covariances = 0, cross-lags = 0								
Model 2: Covariances = 0, cross-lags = 0, + instruments								
Model 3: Covariances = 0, cross-lags = 0, + instruments								
Model 4: Covariances = 0, cross-lags = 0, + instruments								
Model 5: Covariances = 0, cross-lags = 0, + instruments								
	Model 1 vs. Model 2		Model 2 vs. Model 3		Model 2 vs. Model 4		Model 2 vs. Model 5	
LL _x	94,500.11		94,148.71		94,148.71		94,148.71	
DF _x	76		105		105		105	
LL _{x+1}	94,148.71		93,699.98		93,992.82		93,558.29	
DF _{x+1}	105		111		109		115	
Difference in deviance statistic	351.40		448.73		155.89		590.42	
Difference in <i>df</i>	29		6		4		10	
LR test	.000		.000		.000		.000	
Best fitting model	Model 2		Model 3		Model 4		Model 5	

Table 2

Descriptive Statistics

	Mean or Percent	Std. Dev.	Minimum	Maximum
<i>Poor Health – Adolescence (Wave I)</i>				
Self-Reported Health	2.14	0.90	1	4
BMI	22.56	4.46	11.21	49.12
Physical Inactivity	1.04	0.85	0	3
<i>Poor Health – Emerging Adulthood (Wave III)</i>				
Self-Reported Health	2.02	0.86	1	4
BMI	26.91	6.44	13.80	73.17
Physical Inactivity	2.98	1.57	0	5
<i>Poor Health – Young adulthood (Wave IV)</i>				
Self-Reported Health	2.36	0.88	1	4
BMI	29.17	7.62	14.40	80.40
Physical Inactivity	2.86	1.50	0	5
<i>Human Capital - Adolescence (Wave I)</i>				
GPA	2.79	0.72	1	4
Add Health Picture Vocabulary Test	102.47	13.36	14	146
No grade retention	78.96%		0	1
<i>Human Capital - Emerging Adulthood (Wave III)</i>				
Years of Completed Schooling	13.05	1.97	6	22
Add Health Picture Vocabulary Test	100.56	14.12	7	122
Employed or In School	82.96%		0	1
Have Checking Account	71.40%		0	1
Have Savings Account	62.54%		0	1
<i>Human Capital - Young adulthood (Wave IV)</i>				
Years of Completed Schooling	14.23	2.59	8	26
Household Income	60,125	37,057	0	150,000
Currently Employed	66.49%		0	1
Self-Rated Socioeconomic Status	4.95	1.74	1	10
<i>Metabolic Syndrome</i>				
Elevated Waist Circumference	51.80%		0	1
Elevated Blood Pressure	41.84%		0	1
Elevated Triglycerides	9.89%		0	1
Reduced HDL Cholesterol	16.25%		0	1
Pre-Diabetic Value, Hemoglobin A1c	31.24%		0	1
<i>Sociodemographic Controls - Adolescence (Wave I)</i>				
Two Parent Family Structure	56.40%		0	1
Parental Education	5.50	2.13	0	9
Household Income-Needs Ratio	2.98	3.00	0.00	97.80
Race-Ethnicity				
Non-Hispanic White (reference)	72.14%		0	1

	Mean or Percent	Std. Dev.	Minimum	Maximum
Non-Hispanic Black	14.75%		0	1
Hispanic	3.58%		0	1
Non-Hispanic Other	9.52%		0	1
Age at Wave I	15.43	1.82	11	21
Sex (1 = female)	51.02%		0	1
<i>Wave IV Household Composition^a</i>				
Live alone	11.44%		0	1
Live with parents	15.85%		0	1
Married or cohabiting	57.50%		0	1
<i>Wave III Urbanicity</i>				
Living in urban census tract	58.25%		0	1
<i>Instrumental Variables</i>				
Euclidean Distance to Nearest Park (meters), WI	11,830.92	11,386.65	0	81,562.27
Know Most People in Neighborhood, WI	74.08%		0	1
Stopped to Talk to Neighbors in Past Month, WI	79.23%		0	1
Usually Feel Safe in Neighborhood, WI	91.00%		0	1
Juvenile Violent Crime Rate (per 100,000), WI	48.60	36.47	0	214.94
Bureau of Labor Statistics Cost Price Index, WIII	1.78	0.01	1.75	1.80
Inactivity Resources, Count, WIII	0.18	0.75	0	26
Median Year Housing Built was Before 1978, WIII	74.40%		0	1
Per Capita Income, WIII	19,493	8,095	2,700	108,600
Age-Specific Mortality Rate (per 1,000), WIII	0.83	0.37	0	3.48
Juvenile Violent Crime Rate (per 100,000), WIV	27.68	21.43	0	90.00
Male Unemployment Rate, WIV	0.08	0.06	0	0.68
Percent Female-Headed Households with Children, WIV	7.90%	5.28	0	49.08
Percent of Homes with Insufficient Plumbing, WIV	1.85%	3.04	0	31.23

Note: Sample size is 9,033 men and women. Statistics are weighted and adjusted for clustering.

^aPercentages do not total 100 as these are not mutually exclusive categories of the same variable. Age-specific mortality rate indicates a smoothed rate across 1999 – 2001 of the number of deaths among persons age 15–24 per 1,000 residents at the county level.

Source: National Longitudinal Study of Adolescent Health

Table 3

Total, Direct, and Indirect Effects of Poor Health and Human Capital on Metabolic Syndrome

Panel A. Effects of Poor Health at Waves I, III, and IV	Unstandardized β (SE)	β / SE
Poor Health (WIV) $\rightarrow$ Metabolic Syndrome		
Total Effect = Direct Effect	.42 (.01) ***	35.42
Poor Health (WIII) $\rightarrow$ Metabolic Syndrome		
Total Effect = Indirect Effect	.45 (.01) ***	31.75
<i>Specific Indirect Paths</i>		
Health ₃ $\rightarrow$ Health ₄ $\rightarrow$ Metabolic Syndrome	.45 (.01) ***	31.89
Health ₃ $\rightarrow$ Human Capital ₄ $\rightarrow$ Metabolic Syndrome	.002 (.001) *	2.46
Poor Health (WI) $\rightarrow$ Metabolic Syndrome		
Total effect = Indirect Effect	.26 (.04) ***	6.26
<i>Specific Indirect Paths</i>		
Health ₁ $\rightarrow$ Health ₃ $\rightarrow$ Health ₄ $\rightarrow$ Metabolic Syndrome	.26 (.04) ***	6.23
Health ₁ $\rightarrow$ Health ₃ $\rightarrow$ Human Capital ₄ $\rightarrow$ Metabolic Syndrome	.001 (.001) *	2.37
Health ₁ $\rightarrow$ Human Capital ₃ $\rightarrow$ Health ₄ $\rightarrow$ Metabolic Syndrome	.000 (.000)	1.16
Health ₁ $\rightarrow$ Human Capital ₃ $\rightarrow$ Human Capital ₄ $\rightarrow$ Metabolic Syndrome	.001 (.001)	1.13
Panel B. Effects of Human Capital at Waves I, II, and IV		
Human Capital (WIV) $\rightarrow$ Metabolic Syndrome		
Total Effect = Direct Effect	-.02 (.005) ***	-3.76
Human Capital (WIII) $\rightarrow$ Metabolic Syndrome		
Total Effect = Indirect Effect	-.03 (.006) ***	-4.83
<i>Specific Indirect Paths</i>		
Human Capital ₃ $\rightarrow$ Human Capital ₄ $\rightarrow$ Metabolic Syndrome	-.02 (.006) ***	-3.61
Human Capital ₃ $\rightarrow$ Health ₄ $\rightarrow$ Metabolic Syndrome	-.01 (.002) ***	-3.84
Human Capital (WI) $\rightarrow$ Metabolic Syndrome		
Total effect = Indirect Effect	-.12 (.03) ***	-4.57
<i>Specific Indirect Paths</i>		
Human Capital ₁ $\rightarrow$ Human Capital ₃ $\rightarrow$ Human Capital ₄ $\rightarrow$ Metabolic Syndrome	-.08 (.02) ***	-3.59
Human Capital ₁ $\rightarrow$ Human Capital ₃ $\rightarrow$ Health ₄ $\rightarrow$ Metabolic Syndrome	-.03 (.01) ***	-3.81
Human Capital ₁ $\rightarrow$ Health ₃ $\rightarrow$ Human Capital ₄ $\rightarrow$ Metabolic Syndrome	-.00 (.00)	-.26
Human Capital ₁ $\rightarrow$ Health ₃ $\rightarrow$ Health ₄ $\rightarrow$ Metabolic Syndrome	-.01 (.02)	-.26

Appendix Table A

Factor Loadings and Parameter Estimates from the Structural Equation Model of Poor Health, Human Capital, and Metabolic Syndrome

	β (SE)
Factor Loadings	
<i>Poor Health - WI</i>	
BMI	1
Self-Reported Health	.54 (.04) ***
Physical Inactivity	.24 (.04) ***
<i>Poor Health - WIII</i>	
BMI	1
Self-Reported Health	.32 (.02) ***
Physical Inactivity	.04 (.04)
<i>Poor Health - WIV</i>	
BMI	1
Self-Reported Health	.43 (.03) ***
Physical Inactivity	.22 (.03) ***
<i>Human Capital - WI</i>	
GPA	1
Add Health Picture Vocabulary Test	2.19 (.18) ***
No grade retention	.50 (.03) ***
<i>Human Capital - WIII</i>	
Years of Completed Schooling	1
Add Health Picture Vocabulary Test	.56 (.04) ***
Employed or In School	.10 (.01) ***
Have Checking Account	.17 (.01) ***
Have Savings Account	.11 (.01) ***
<i>Human Capital - WIV</i>	
Years of Completed Schooling	1
Household Income	.22 (.02) ***
Currently Employed	.04 (.01) ***
Self-Rated Socioeconomic Status	.44 (.02) ***
<i>Metabolic Syndrome</i>	
Elevated Waist Circumference	1
Elevated Blood Pressure	.41 (.02) ***
Elevated Triglycerides	.15 (.01) ***
Reduced HDL Cholesterol	.18 (.02) ***
Pre-Diabetic Value, Hemoglobin A1c	.30 (.03) ***

	β (SE)
Parameter Estimates	
Poor Health, WIV $\leftarrow$ Poor Health, WIII	1.06 (.02) ***
Poor Health, WIV $\leftarrow$ Human Capital, WIII	-.02 (.01) ***
Poor Health, WIII $\leftarrow$ Poor Health, WI	.58 (.10) ***
Poor Health, WIII $\leftarrow$ Human Capital, WI	-.01 (.03)
Human Capital, WIV $\leftarrow$ Human Capital, WIII	1.19 (.04) ***
Human Capital, WIV $\leftarrow$ Poor Health, WIII	-.13 (.04) ***
Human Capital, WIII $\leftarrow$ Human Capital, WI	3.84 (.20) ***
Human Capital, WIII $\leftarrow$ Poor Health, WI	-.06 (.05)
Metabolic Syndrome $\leftarrow$ Poor Health, WIV	.42 (.01) ***
Metabolic Syndrome $\leftarrow$ Human Capital, WIV	-.02 (.005) ***
Metabolic Syndrome $\leftarrow$ Parent Education	-.003 (.003) *
Metabolic Syndrome $\leftarrow$ (log) Income-Needs	.01 (.004)
Metabolic Syndrome $\leftarrow$ nH Black	.04 (.02) *
Metabolic Syndrome $\leftarrow$ Hispanic	-.02 (.03)
Metabolic Syndrome $\leftarrow$ nH other	-.03 (.03)
Metabolic Syndrome $\leftarrow$ WI Age	.01 (.003) ***
Metabolic Syndrome $\leftarrow$ Female	-.61 (.04) ***
Poor Health, WIV $\leftarrow$ Family Structure	.01 (.01)
Poor Health, WIV $\leftarrow$ (log) Income-Needs	-.01 (.01)
Poor Health, WIV $\leftarrow$ nH Black	.06 (.03) *
Poor Health, WIV $\leftarrow$ Hispanic	.04 (.02)
Poor Health, WIV $\leftarrow$ nH other	.02 (.04)
Poor Health, WIV $\leftarrow$ Female	-.01 (.01)
Poor Health, WIV $\leftarrow$ Juvenile Violent Crime, WIV	-.01 (.004) *
Poor Health, WIV $\leftarrow$ Male Unemployment, WIV	.02 (.01)
Poor Health, WIV $\leftarrow$ Insufficient Plumbing, WIV	-.004 (.002) $\dagger$
Poor Health, WIV $\leftarrow$ Female-Headed HH with Kids, WIV	-.01 (.01)
Poor Health, WIII $\leftarrow$ Family Structure	.01 (.02)
Poor Health, WIII $\leftarrow$ Parent Education	-.02 (.005) ***
Poor Health, WIII $\leftarrow$ (log) Income-Needs	-.01 (.01)
Poor Health, WIII $\leftarrow$ nH Black	.02 (.02)
Poor Health, WIII $\leftarrow$ Hispanic	.01 (.02)
Poor Health, WIII $\leftarrow$ nH other	.02 (.05)
Poor Health, WIII $\leftarrow$ Female	.04 (.01) **
Poor Health, WIII $\leftarrow$ BLS Cost Price Index, WIII	-.10 (.09)
Poor Health, WIII $\leftarrow$ Live in Urban tract, WIII	.004 (.02)
Poor Health, WIII $\leftarrow$ Inactivity Resources, WIII	-.02 (.005) ***

	β (SE)
Poor Health, WIII $\leftarrow$ Per Capita Income, WIII	-.02 (.01) *
Poor Health, WIII $\leftarrow$ Age-Specific Mortality Rate, WIII	.02 (.02)
Poor Health, WI $\leftarrow$ Family Structure	-.003 (.01)
Poor Health, WI $\leftarrow$ Parent Education	-.02 (.004) ***
Poor Health, WI $\leftarrow$ (log) Income-Needs	-.01 (.01)
Poor Health, WI $\leftarrow$ nH Black	.06 (.02) **
Poor Health, WI $\leftarrow$ Hispanic	.04 (.03)
Poor Health, WI $\leftarrow$ nH other	.04 (.07)
Poor Health, WI $\leftarrow$ WI Age	.05 (.01) ***
Poor Health, WI $\leftarrow$ Female	-.02 (.01)
Poor Health, WI $\leftarrow$ Distance to Nearest Park, WI	.006 (.007)
Poor Health, WI $\leftarrow$ Know Most Neighbors, WI	-.01 (.01)
Poor Health, WI $\leftarrow$ Stopped to Talk to Neighbors, WI	-.01 (.01)
Poor Health, WI $\leftarrow$ Juvenile Violent Crime Rate, WI	-.001 (.002)
Human Capital, WIV $\leftarrow$ nH Black	.99 (.21) ***
Human Capital, WIV $\leftarrow$ Hispanic	.002 (.16) ***
Human Capital, WIV $\leftarrow$ nH other	.002 (.16)
Human Capital, WIV $\leftarrow$ WI Age	-.05 (.03) $\dagger$
Human Capital, WIV $\leftarrow$ Female	-.28 (.08) ***
Human Capital, WIV $\leftarrow$ Juvenile Violent Crime, WIV	.07 (.02) ***
Human Capital, WIV $\leftarrow$ Male Unemployment, WIV	-.08 (.04) *
Human Capital, WIV $\leftarrow$ Insufficient Plumbing, WIV	-.02 (.01) *
Human Capital, WIV $\leftarrow$ Female-Headed HH with Kids, WIV	-.23 (.05) ***
Human Capital, WIV $\leftarrow$ Live with Parents, WIV	.13 (.10)
Human Capital, WIV $\leftarrow$ Married or Cohabiting, WIV	.29 (.07) ***
Human Capital, WIV $\leftarrow$ Live Alone, WIV	.18 (.11)
Human Capital, WIII $\leftarrow$ Family Structure	.09 (.05) $\dagger$
Human Capital, WIII $\leftarrow$ nH Black	.03 (.17)
Human Capital, WIII $\leftarrow$ Hispanic	.28 (.08) ***
Human Capital, WIII $\leftarrow$ nH other	.15 (.13)
Human Capital, WIII $\leftarrow$ WI Age	.31 (.04) ***
Human Capital, WIII $\leftarrow$ Housing Built Before 1978, WIII	.04 (.05)
Human Capital, WIII $\leftarrow$ BLS Cost Price Index, WIII	-.37 (.30)
Human Capital, WI $\leftarrow$ Family Structure	.12 (.02) ***
Human Capital, WI $\leftarrow$ Parent Education	.08 (.01) ***
Human Capital, WI $\leftarrow$ (log) Income-Needs	.05 (.01) ***
Human Capital, WI $\leftarrow$ nH Black	-.26 (.03) ***
Human Capital, WI $\leftarrow$ Hispanic	-.10 (.03) **

	β (SE)
Human Capital, WI ← nH other	.02 (.04)
Human Capital, WI ← WI Age	-.06 (.01) ***
Human Capital, WI ← Female	.05 (.02) ***
Human Capital, WI ← Feel Safe in Neighborhood, WI	.12 (.02) ***
Human Capital, WI ← Stopped to Talk to Neighbors, WI	.001 (.01)
Human Capital, WI ← Juvenile Violent Crime Rate, WI	.001 (.003)
Residual Variances of Latent Variables	
Poor Health, WIV	.07 (.01) ***
Poor Health, WIH	.17 (.02) ***
Poor Health, WI	.17 (.01) ***
Human Capital, WIV	.09 (.10)
Human Capital, WIH	.07 (.04) **
Human Capital, WI	.07 (.01) ***
Metabolic Syndrome	.01 (.01)
R-Square of Latent Variables	
Poor Health, WIV	.87 (.02) ***
Poor Health, WIH	.53 (.06) ***
Poor Health, WI	.07 (.01) ***
Human Capital, WIV	.97 (.04) ***
Human Capital, WIH	.97 (.02) ***
Human Capital, WI	.5 (.02) ***
Metabolic Syndrome	.96 (.02) ***
Sample size	9,033
Chi-Square	6,277.34 ***
Degrees of Freedom	897
Fit Indices	
RMSEA	.026
SRMR	.05
TLI	.837
CFI	.826

Notes:

p < .001,

**
p < .01,

*
p < .05,

†
p < .10 (two-tailed).

Correlations between error variances of latent variables included but not shown.

Source: National Longitudinal Study of Adolescent Health

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Appendix Table B

Parameter Estimates from a Series of Structural Equation Models Specifying Metabolic Syndrome by Varying Indicators

All MetS indicators are binary		MetS scaled by linear waist circumference		MetS scaled by linear triglycerides		MetS scaled by linear hemoglobin A1c	
		Standardized <i>b</i>	<i>z</i>	Standardized <i>b</i>	<i>z</i>	Standardized <i>b</i>	<i>z</i>
MetS ← PH4	.64	.67	35.13	.61	42.13	.75	31.88
PH4 ← PH3	.93	.91	123.18	.92	114.07	.92	112.69
PH3 ← PH1	.42	.43	9.88	.43	9.81	.42	9.22
HC4 ← HC3	.98	.98	88.24	.98	88.09	.94	73.49
HC3 ← HC1	1.01	1.02	83.38	1.02	82.96	1.02	76.38

Appendix Table C

Parameter Estimates from Instrumental Variables Regression Models

	First-Stage Equations Predicting:		Second-Stage Equation Predicting:
	Poor Health, WIV	Human Capital, WIV	MetS, WIV
Poor Health, Wave IV	-	-	.44 *** (.12)
Human Capital, Wave IV	-	-	-.01 (.01)
<i>Sociodemographic Controls</i>			
Two Parent Family Structure	.03 * (.01)	.65 *** (.02)	0 (.01)
Parental Education	-.04 *** (.00)	.37 *** (.01)	-.001 *** (.00)
Household Income-Needs Ratio	-.06 *** (.01)	.21 *** (.01)	.02 *** (.00)
Race-Ethnicity			
nH Black	.18 *** (.02)	-.08 * (.03)	.04 [†] (.02)
Hispanic	.09 *** (.02)	.33 *** (.04)	.01 (.02)
nH Other	.08 * (.03)	.04 (.05)	-.05 *** (.01)
Age at Wave I	.03 *** (.00)	.05 *** (.01)	0 (.00)
Sex (1 = female)	.00 (.01)	-.23 *** (.02)	-.50 *** (.00)
<i>Instruments</i>			
Male Unemployment Rate, WIV	.04 ** (.01)	-.24 *** (.02)	-
Percent Female-Headed Households with Children, WIV	.03 * (.02)	-.38 *** (.02)	-
Percent of Homes with Insufficient Plumbing, WIV	.01 * (.00)	-.04 *** (.00)	-
Juvenile Violent Crime Rate (per 100,000), WIV	-.01 *** (.00)	.10 *** (.01)	-
Intercept	-1.84 *** (.07)	.68 *** (.12)	.11 (.21)
Sargan's test			$\chi^2(2) = .298$ ($p = .86$)

	First-Stage Equations Predicting:		Second-Stage Equation Predicting:
	Poor Health, WI	Human Capital, WI	Poor Health, WIII
Poor Health, Wave I	-	-	.49 (.45)
Human Capital, Wave I	-	-	-.42 *** (.12)
Two Parent Family Structure	.01 (.01)	.12 *** (.00)	.07 *** (.02)
Parental Education	-.02 *** (.00)	.08 *** (.00)	.01 (.01)
Household Income-Needs Ratio	-.02 *** (.00)	.04 *** (.00)	.00 (.01)
Race-Ethnicity			
nH Black	.09 *** (.01)	-.26 *** (.01)	-.05 (.04)
Hispanic	.05 *** (.01)	-.12 *** (.01)	-.01 (.03)
nH Other	.06 ** (.02)	-.04 *** (.01)	.01 (.03)
Age at Wave I	.05 *** (.00)	-.06 *** (.00)	-.02 (.02)
Sex (1 = female)	-.02 ** (.01)	.06 *** (.00)	.06 *** (.01)
<i>Instruments</i>			
(Lg) Euclidean Distance to Nearest Park, WI	.00 (.00)	.00 (.00)	-
Know Most People in Neighborhood, WI	-.01 (.01)	-.04 *** (.01)	-
Stopped to Talk to Neighbors in Past Month, WI	.00 (.01)	-.01 † (.01)	-
Juvenile Violent Crime Rate (per 100,000), WI	.00 (.00)	.00 *** (.00)	-
Usually Feel Safe in Neighborhood, WI	-.03 * (.01)	.12 *** (.01)	-
Intercept	.01	.02	-1.64 ***
Sargan's test			$\chi^2(3) = 4.46$ ($p = .22$)
	First-Stage Equations Predicting:		Second-Stage Equation Predicting:
	Poor Health, WIII	Human Capital, WIII	Poor Health, WIV
Poor Health, Wave III	-	-	1.17 ***

	First-Stage Equations Predicting:		Second-Stage Equation Predicting:
	Poor Health, WIII	Human Capital, WIII	Poor Health, WIV
			(.16)
Human Capital, Wave III	-	-	0
			(.05)
<i>Sociodemographic Controls</i>			
Two Parent Family Structure	.02 [†]	.54 ^{***}	.01
	(.01)	(.02)	(.03)
Parental Education	-.03 ^{***}	.28 ^{***}	0
	(.00)	(.00)	(.01)
Household Income-Needs Ratio	-.02 ^{***}	.16 ^{***}	-.03 ^{***}
	(.01)	(.01)	(.01)
Race-Ethnicity			
nH Black	.08 ^{***}	-.70 ^{***}	.07 ^{**}
	(.02)	(.02)	(.03)
Hispanic	.07 ^{***}	.05 [†]	0
	(.02)	(.03)	(.01)
nH Other	.06 [*]	-.05	0
	(.03)	(.04)	(.01)
Age at Wave I	.03 ^{***}	.07 ^{***}	-.01
	(.00)	(.01)	(.01)
Sex (1 = female)	.03 [*]	.21 ^{***}	-.03 [†]
	(.01)	(.02)	(.02)
<i>Instruments</i>			
BLS Cost Price Index, WIII	-.06	-.12	-
	(.09)	(.14)	
Inactivity Resources, Count, WIII	-.03 ^{***}	.05 ^{***}	-
	(.01)	(.01)	
Median Year Housing Built was Before 1978, WIII	.02	-.03	-
	(.01)	(.02)	
Per Capita Income, WIII	-.04 ^{***}	.14 ^{***}	-
	(.01)	(.01)	
Age-Specific Mortality Rate (per 1,000), WIII	.09 ^{***}	-.27 ^{***}	-
	(.02)	(.03)	
Intercept	-.62	2.33	.17
	(1.68)	(2.45)	(.26)
Sargan's test			$\chi^2 (3) = 2.11$ ($p = .55$)

Notes:

 $p < .001$,**
 $p < .01$,

*
 $p < .05$,

[†]
 $p < .10$ (two-tailed).

Analyses are unweighted. N=9,033.

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Appendix Table D

Standardized Parameter Estimates from the Structural Equation Model and the Uncorrected OLS Regression Model

Dependent Variable ← Independent Variable	SEM	OLS
<i>Path Coefficients Reflecting Path Dependence</i>		
Poor Health, WIV ← Poor Health, WIII	.92***	.59***
Poor Health, WIII ← Poor Health, WI	.40***	.50***
Human Capital, WIV ← Human Capital, WIII	.99***	.53***
Human Capital, WIII ← Human Capital, WI	1.01***	.56***
<i>Cross-Lagged Path Coefficients Representing Social Causation</i>		
Poor Health, WIV ← Human Capital, WIII	-.04**	-.08***
Poor Health, WIII ← Human Capital, WI	-.005	-.03*
<i>Cross-Lagged Path Coefficients Representing Health Selection</i>		
Human Capital, WIV ← Poor Health, WIII	-.05**	-.16***
Human Capital, WIII ← Poor Health, WI	-.02	-.08***
<i>Path Coefficient Representing Social Inequality in Health, Net of Health Selection</i>		
Metabolic Syndrome ← Human Capital, WIV	-.07***	-.03*

Notes: The uncorrected OLS model includes only lagged and cross-lagged values of health and human capital, and controls.