Published in final edited form as:

J Health Soc Behav. 2015 June ; 56(2): 262–280. doi:10.1177/0022146515581857.

Poor Child Health, Family Capital and Cumulative Inequality in Academic Achievement

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Abstract

Our understanding of health and social stratification can be enriched by testing tenets of cumulative inequality theory that emphasize how the accumulation of inequality is dependent on the developmental stage being considered; the duration and stability of poor health; and the family resources available to children. I analyze longitudinal data from the British National Child Development Study (N=9,252) to ask: 1) if child health is a source of cumulative inequality in academic achievement; 2) whether this relationship depends on the timing and duration of poor health; and 3) whether trajectories are sensitive to levels of family capital. The results suggest that the relationship between health and academic achievement emerges very early in life and persists, and that whether we observe shrinking or widening inequality as children age depends on when we measure their health, and whether children have access to compensatory resources.

Scholars studying health and social stratification over the life course now recognize that the reproduction of intergenerational inequality begins at a very young age (Jonsson 2010). Socioeconomic inequalities in children's learning are present at the beginning of the school years, a troubling fact given strong correlations among achievement, completed schooling and economic status (Duncan, Ziol-Guest and Kalil 2010; Entwisle, Alexander and Olson 2005). Economic disadvantage and the risk of poor health go hand in hand, and socioeconomic inequality in child health is present at birth and increases throughout childhood (Adler et al. 1994; Finch 2003; Link and Phelan 1995). The appearance of health inequality so early in life has important implications for patterns over the life course and across generations. Child health is a strong determinant of both short-term opportunities for upward mobility in the form of skill development and academic progress, as well as longer-term risk of downward mobility in the form of job loss, declining income, and prohibitive health care costs (Conley, Strully and Bennett 2003; McLeod, Uemura and Rohrman 2012; Palloni 2006).

Despite increasing understanding of the effects of health on social processes, much existing research considers health and the skills that stem from it at isolated points in time. Such a perspective obscures a complete understanding of how the relationship between health and academic inequality emerges as children age, and how health acts as a pathway in the

intergenerational reproduction of disadvantage. Guided by cumulative inequality theory, this article advances research on health and social stratification by linking the dynamic nature of health in the early life course with trajectories of academic achievement (Ferraro, Shippee and Schafer 2009). Using longitudinal data from the United Kingdom, I test the following ideas: 1) child health is not only an early but also a *variable* and *accumulating* source of inequality in academic achievement—in other words, the timing, duration and stability of poor health are important in predicting *changes* in academic inequality through the school years; and 2) high levels of family capital may *buffer* the influence of poor health on academic trajectories. Ultimately, a cumulative inequality perspective reveals that the role of health in producing academic inequality depends on when and for how long children are in poor health, and whether children have access to compensatory resources.

Health and Academic Achievement: Insights from Cumulative Inequality Theory

The association between child health and social resources and relationships over the life course is striking. Child health, often measured by birth weight, affects youths' educational achievement/attainment and adults' earnings and labor force participation (Boardman et al. 2002; Currie and Stabile 2006; Haas and Fosse 2008; Jackson 2009). Though the majority of research on health and social stratification examines longer-term effects, there is growing attention among sociologists to the role of academic disparities in linking poor child health to adults' socioeconomic status (Crosnoe 2006; Jackson 2010). From this research we have learned that poor health in a sensitive period of human development affects children's readiness to learn and effectively participate in academic curricula, producing early inequalities in skill development and learning.

Few studies examine how health influences the *development* of academic inequality throughout the school years, with those that do suggesting an important cumulative process that begins early and grows over time (e.g., Cheadle and Goosby 2010). I use cumulative inequality theory as the framework for examining the role of children's health in enabling or compromising achievement trajectories. Cumulative disadvantage theory has long emphasized the persistent and compounding nature of intra-cohort inequality, whereby a particular social circumstance affects initial levels of a resource and produces widening gaps over time (Diprete and Eirich 2006; Merton 1968; Willson, Shuey and Elder 2007). Combining tenets of cumulative disadvantage and life course theories, cumulative inequality theory offers a framework for how the accumulation of inequality is sensitive to both developmental processes and the availability of compensatory resources (Ferraro and Shippee 2009).

A key principle of cumulative inequality theory is that circumstances during the early life course, as well as their timing, play a key role in producing compounding inequality into adulthood (Ferraro, Shippee and Schafer 2009). Despite this possibility, most research on health and academic trajectories considers birth weight as the sole indicator of child health. I consider the possibility that the accumulation of academic inequality depends on the timing, duration and stability of a child's state of poor health. Because the early life cycle, particularly the prenatal period through age three, is a highly sensitive period of brain

development, I expect that: 1) the influence of poor prenatal/infant health should become more negative—achievement disparities should compound—as children age (Knudsen 2004). Poor health very early in life, reflected by children's birth weight or by unhealthy exposures during the prenatal/infant period, may affect mechanisms responsible for skill development. Smoking during pregnancy reduces blood and oxygen flow to the placenta and exposes fetuses to nicotine (Wakschlag et al. 2002). Low birth weight is negatively related to both early academic achievement and achievement trajectories (Conley, Strully and Bennett 2003; Cheadle and Goosby 2010). Some research even suggests that exposures in utero can permanently “program” aspects of physical and cognitive development, despite compensatory behaviors by parents and schools (Barker 1994; Gluckman and Hansen 2006). Overall, existing evidence suggests a bio-social interaction linking early health to achievement, whereby large baseline inequalities in learning compound as children lag behind their peers in successive assessments.

After the “critical period” of early childhood, school-aged youth in poor health also exhibit delayed learning (Crosnoe 2006; Thies 1999). Children with school-age health problems may not participate as fully in the education system due to school absence or reduced educational expectations (McLeod and Fettes 2007; Jackson 2009). To the extent that age-specific health problems have short-term disruptive effects from which children can rebound, however, achievement disparities may not grow over time. This possibility suggests that: 2) poor school-age health should have a less negative relationship with achievement trajectories than poor prenatal/infant health.

In addition to its focus on early life course circumstances and their timing, cumulative inequality theory emphasizes the importance of duration and stability processes for understanding how inequality is generated over the life course (Ben-Schlomo and Kuh 2002; Diprete and Eirich 2006; Ferraro and Shippee 2009). In this vein, a long duration of obesity is related to health decline in adulthood, and the accumulation of adverse childhood circumstances influences individuals' own life evaluations (Ferraro and Kelley-Moore 2003; Schafer, Ferraro and Mustillo 2011). Continuously deteriorating health throughout childhood (the accumulation of health problems) may have a particularly detrimental impact on academic learning, as compared to an isolated period of poor health or even to a stable, chronic condition. To the extent that a longer duration of, or continual progression toward, poor health limits opportunities for academic participation and progress, I expect that: 3a) when the duration of poor health is longer, the influence of school-age health on academic trajectories should be more negative than when poor health is transitory, and 3b) declines in health during the school years should be associated with declines in achievement trajectories. This hypothesis focuses on the duration and stability of poor health during the school years, not particular duration/stability combinations of prenatal, infant and school-age health.

Finally, cumulative inequality theory emphasizes the importance of resource availability in shaping the accumulation of inequality (Ferraro, Shippee and Schafer 2009; O'Rand 2009). Among children, families are a central institution in which resources are redistributed, daily activities are managed and relationships are formed. The quantity and content of financial and time-related resources within families constitute “family capital” that is instrumental to

children's development and may offset other forms of disadvantage (Dufur, Parcel and McKune 2008; Parcel, Dufur and Zito 2010; Wagmiller et al. 2006). High levels of family capital can be mobilized to buffer against the negative academic effects of poor health.

Research examining the moderating influence of family capital on the relationship between health and achievement has yielded mixed findings. There is evidence that parental income blunts the negative educational effects of low birth weight, though other work suggests that the home environment—including family income and the frequency of mothers' reading to young children—does not reduce the negative academic effects of low birth weight (Cheadle and Goosby 2010, Conley and Bennett 2001, Power et al 2006). Little is known about whether family capital compensates for markers of health other than birth weight, and for poor health later in childhood. I will test the hypothesis that: 4) high levels of family capital weaken the negative influence of poor prenatal, infant and school-age health on early achievement disparities and achievement trajectories.

DATA AND METHODS

Data

The goal of this research is to use cumulative inequality theory as a framework for examining child health as a variable and accumulating predictor of academic trajectories, as well as the possible buffering influence of family capital. This effort requires longitudinal measures of child health coupled with longitudinal measures of academic learning, data that few prospective surveys of youth provide for the duration of childhood. I use data from the National Child Development Study (NCDS) in the United Kingdom, a survey of the 1958 birth cohort providing information at birth and ages 7, 11, 16, 23, 33, 42, 46 and 50. The ongoing survey follows every baby who was born in England, Scotland and Wales on a particular week in 1958 (almost 17,500 children). The NCDS includes information on health, cognitive and social development, educational progress, income, and family relationships. NCDS data have been used extensively to study the transition to adulthood (Cherlin et al. 1991).

In this analysis I use data from the prenatal period through age 16. Previous research with NCDS data documents the effects of child health on educational attainment and socioeconomic status in mid-adulthood (Case et al. 2005; Jackson 2010). I take the strong relationship between health and educational attainment in these data as understood, and focus on the early life course to reveal how the relationship between health and persistent academic inequality emerges. Though more contemporary surveys provide excellent data on shorter periods of the early life course (early childhood, adolescence), representative data with large enough samples to follow children from the prenatal period through the end of secondary schooling are rare. NCDS data provide an unparalleled resource for the questions considered here, in that they include medical exams at each age surveyed and permit observation of health, achievement and social environments at multiple points during childhood.

Mid-late twentieth century Great Britain provides a useful context in which to examine health as a source of cumulative achievement inequality. The structured educational system

during that period made learning assessments a particularly important gate keeping mechanism for eventual educational attainment. In the United States, educational opportunity has historically been open on the basis of ability and effort. In contrast, educational opportunity in the United Kingdom was determined at a young age through a tracking process. Though tracking has decreased substantially since the mid-1970s, the educational system was still deterministic during the time period considered here. After completing primary school at age eleven, students took “eleven plus” exams that determined (along with school performance and an interview process) entrance into an academically rigorous grammar school or a vocational secondary school focused on basic training (Kerckhoff, Haney and Glennie 2001). Students in grammar schools took “O-level” achievement exams at age sixteen and, depending on the result, continued until age eighteen, when they took “A-level” university entrance exams. Students in the non-university track generally left school at age 16. In this context, early learning differences had strong consequences for educational progression.

Measures

Health—NCDS children received a medical exam at each survey wave, permitting measurement of many specific health conditions. However, the small number of children with any particular health problem necessitates combining specific conditions into broader markers of health. This limitation should be balanced against the benefit gained from having comparable measures across multiple ages, however, which permits longitudinal measurement of child health. To measure prenatal and infant health I examine infants’ *low birth weight* status and whether the mother *smoked* after the fourth month of pregnancy. I differentiate among no, medium/variable and heavy levels of smoking, as reported by mothers.¹ Low birth weight is defined as weight below 5.5 lbs, a widely used threshold (Conley and Bennett 2001).

Table 1 shows that about 5% of children in the analytic sample had a low birth weight. About a third of mothers smoked after the fourth month of pregnancy, with 12% of mothers smoking heavily during this period. Despite the existence of the National Health Service (which began in 1948) at the time of this cohort’s birth, socioeconomic disparities in health and health behavior in these data are similar in size to patterns in the contemporary U.K., as well as in the United States (Marmot et al. 1978; Banks et al. 2003). One important exception to this pattern is a weak socioeconomic patterning to smoking. Beginning in the early 1970s, following the U.S. Surgeon General’s Report in 1969, women’s smoking prevalence declined steeply and became more strongly related to socioeconomic status (Townsend, Roderick and Cooper 1994). Prior to that decline, smoking was common among women in all socioeconomic groups, with only a weak class gradient and no media emphasis on communicating the dangers of smoking while pregnant (Fertig 2010, Graham 1994).

To measure school-age health I use physicians’ diagnoses of whether children have a *physical or mental/emotional* health condition at each age. Physical and mental conditions,

¹Models that additionally control for mothers’ and fathers’ smoking when children are age 16 produce virtually identical results for the prenatal smoking coefficients, suggesting that prenatal smoking behavior is not simply a proxy for maternal smoking during childhood.

diagnosed during a medical exam, reflect a slight, moderate or severe condition impeding normal functioning. Physical health conditions include systemic conditions (heart, respiratory, blood conditions), genetic conditions, and physical abnormalities (spinal or limb disfiguration). The most common physical health condition is asthma. A sensitivity analysis excluding children with physical abnormalities does not produce substantively different findings, so I include these children in the analytic sample. Mental health conditions include emotional and behavioral problems—I exclude children with mental retardation.² I use these measures in three ways: 1) to create age-specific measures of health; 2) to create a measure of the duration of poor health during the school years; and 3) to measure the duration and stability of poor health during the school years using a latent variable framework. First, age-specific measures of poor health indicate whether children have a physical or mental health condition at ages 7, 11, and 16. Second, I create a cumulative count measure of the duration of poor school-age health that differentiates among a health condition at no school ages; only one age; two ages; or all of ages 7, 11 and 16. The count measure ranges from 0-3 and uses information from ages 7, 11 and 16. Children with a condition at multiple ages have a higher count score, though they do not necessarily have the same condition at each age. Third, I use the age-specific measures of health in a latent variable framework to provide a more dynamic measure of duration and stability—I describe this approach in greater detail in the methods section.

Table 1 shows that about 6% of children in the analytic sample have an age 7 health condition, with this number increasing gradually during childhood to 9% at age 11, and 10% at age 16. Examining the count measure of duration reveals that, by age 16, 15% of children have experienced poor health during at least one age, 3% at two ages, and 2% at all ages. Sensitivity analyses separating physical and mental health conditions yield very similar patterns, though I do not present these results because of small sample sizes.

Academic Achievement—The NCDS administered standardized *reading and math achievement* assessments to all children at ages 7, 11, and 16. I examine reading and math scores to indicate achievement, rather than using assessments of cognitive ability. Achievement at age 7 is measured by scores on the Southgate Reading Test (word recognition and comprehension) and the Problem Arithmetic Test (Pringle, Butler and Davie 1966; Southgate 1962). At ages 11 and 16, reading and math assessments were constructed specifically for the NCDS by the National Foundation for Education Research. Because the age 7 reading test was designed to be able to identify “backward” readers, about 20% of children at this age attained perfect scores. The distribution of scores on the age 7 math test, and on the math and reading tests at ages 11 and 16, are very bell-shaped (also see Currie and Thomas 2001). Because of the unique design of the age 7 reading test, more attention should be paid to the results for math achievement. Analyses of both reading and math scores yield highly similar results, however.

²I explore the consistency of physician and parent reports of particular conditions during the childhood ages, and find that children whose parents report a particular condition are also less likely to be in the “no condition” category for the same condition (or family of conditions) as reported by physicians during the medical exam.

I use z-scores to measure performance relative to the sample mean at each age. Using standardized scores means that a flat trajectory reflects a child who consistently falls in the middle of the distribution, and that growth patterns will reflect changes in a child's position within the reading or math score distribution with age. This analytic approach is common (Cherlin, Chase-Lansdale and McRae 1998; Diprete and Jennings 2011).

Family Capital—To examine whether family capital moderates the influence of child health on academic achievement, I examine a measure of family social capital, specifically the frequency of mothers' time spent reading to children at age seven. I create two categories: hardly ever/never/occasionally and at least weekly.³ Table 1 shows that 47% of mothers read to their child at least once weekly at age 7. While parental education and family income indicate parents' ability to provide high-quality services and information to their children, parents' time use with children and their involvement in educational activities are markers of *family social capital* that may be more proximately related to children's academic performance and that provide parents with a way to transmit their human capital to children (Dufur, Parcel and McKune 2008). Family social capital is indicated by the time that parents spend interacting with and monitoring their children (Parcel, Dufur and Zito 2010). Children whose parents are able to spend time on educational activities benefit from constructive social relationships that provide an additional opportunity for learning (Crosnoe and Cavanaugh 2010). In supplementary analyses (not shown but available by request), I measure mothers' school-leaving age and family income as indicators of family financial and human capital, respectively. Because the substantive patterns using these measures do not differ from analyses using family social capital, I present the results from this more proximate measure of family behavior.

Other Childhood Characteristics—A benefit of the NCDS is its inclusion of rich markers of children's family and socioeconomic environments, enabling measurement of many factors correlated with both children's health and academic progress. I control for *sex* and *region* within the U.K. at birth (Wales, Scotland and England—the reference category). The sample is overwhelmingly white (over 98%), making it unnecessary to control for race/ethnicity. For questions asked in multiple waves, after numerous sensitivity analyses I create within-child averages that use information from multiple waves, through age 16.⁴ Table 1 lists the ages included in each within-child average, as well as the metric for each variable in the final models. *Father's social/occupational class* follows the registrar general's class scheme and indicates employment in professional, intermediate, skilled non-manual, skilled manual, partly skilled or unskilled professions. I include *maternal grandfather's social class* at birth as a measure of the father's family background, using the same measurement scheme. Because the results do not differ when father's and grandfather's social class is measured categorically vs. quantitatively, I measure both variables quantitatively in the final

³I also examine teachers' reports of each parent's interest in the child's educational progress at ages 7 and 16, separating very/over interested from some/little interest. Because results with this measure do not substantively differ from those using mothers' reading behavior, I do not report these findings.

⁴Because analyses that include age-specific sociodemographic measures (allowing these measures to perturb the latent achievement trajectory by only influencing achievement at the same age) produce virtually identical results for my research questions, I proceed with the within-child average measures in the final models. Similarly, picking one time point as representative of a child's history on a particular measure does not produce different findings, increasing my confidence in presenting results using within-child averages.

models. Children's *access to basic resources* in each year indicates sole access to hot water, a bathroom and indoor lavatory (higher score equals less access). Binary variables measure mothers' *paid work outside of the home* at birth and her *marital status* in 1958—the results are not sensitive to the inclusion of these measures at older ages, and sensitivity analyses including a more detailed measure of mothers' employment status do not produce different results. I measure the *number of children in the household* at each wave, and the number of times each child *moves* between birth and age 16 (measured at age 16). Finally, I measure whether children were *breastfed*, given the correlation of this maternal behavior with prenatal/infant health and cognitive development. Analyses with and without breastfeeding do not produce different results.

Table 1 shows that most mothers were married (96%) at the time of their child's birth. The average social class of children's fathers was a skilled manual position, and the average social class of maternal grandfathers at birth was a skilled manual/non-manual position. On average, mothers and fathers finished school between ages 15 and 16. About half of mothers worked outside of the home during childhood, and most children experienced a residentially stable childhood environment (average of 1.63 moves).

Attrition and Missing Data—Like all longitudinal surveys, the NCDS has experienced attrition. Response rates are high, however, especially given the length of the panel. Of the 17,415 children in 1958, 14,647—about 84%—participated in any module at age 16 (1974), and I limit the sample to those present in all waves between birth and age 16 (over 9,200 children). If the unhealthiest children drop out over time, the remaining sample could be positively selected on health and the observed influence of poor health on achievement may be downwardly biased. I examine differential attrition by health and socioeconomic status, and find little evidence of a systematic pattern. Low birth weight children are more likely to drop out before age seven but this pattern does not persist in subsequent waves, or for maternal smoking during pregnancy or school-aged health. Attrition is also not systematically higher among children in socioeconomically disadvantaged families—though those who drop out by age 16 are slightly more likely to be from disadvantaged backgrounds, measurable differences between the two groups are very small. These patterns of attrition are very similar to those reported in other research with these data (Case and Paxson 2005; Currie and Thomas 2001).

Rather than drop children missing information from a particular module within a wave, I use multiple imputation (five imputations, estimation via Stata) to replace missing values on independent and dependent variables, based on predictions from the independent variables described above (Allison 2002). Values are not imputed if a child is entirely missing from a wave.⁵

⁵Analyses using handling missing data in several other ways—including multiple imputation on only the independent variables, listwise deletion, and mean imputation with “missing” dummies—produce virtually identical findings. In addition, estimates obtained via full information maximum likelihood (FIML), which produces estimates based on the variables that are present for each individual, do not produce substantively different results.

Analysis

The main empirical approach relies on latent growth curve models to examine the association of poor child health with reading and math trajectories. Growth curve models capture not only initial variation in achievement based on health, but also variation in achievement growth or decline over time within individuals (Bollen and Curran 2006). An individual-specific (i) intercept (α), linear, time-specific (t) slope (β) and some error (ε) captures each child's trajectory:

$$y_{it} = \alpha_i + \beta_i t + \varepsilon_{it} \quad (1)$$

Growth models also allow children's trajectories to vary as a function of not only time, but of covariates that vary across individuals:

$$\alpha_i = \alpha_0 + \alpha_1 x_{i1} + \alpha_2 x_{i2} + \dots + \alpha_k x_{ik} + u_i \quad (2)$$

$$\beta_i = \beta_0 + \beta_1 x_{i1} + \beta_2 x_{i2} + \dots + \beta_k x_{ik} + v_i \quad (3)$$

In this study, x indicates children's health, sociodemographic characteristics and family capital. The metric for time is the survey wave. The intercepts and slopes for reading and math achievement are regressed on prenatal/infant health, school-age health and sociodemographic/family measures in order to reveal any group differences in the means of the growth factors. Table 1 lists the particular variables that predict the intercept and slope in each model. This framework permits a test of the first two hypotheses: 1) the influence of poor prenatal and infant health should become more negative—achievement disparities should compound—as children age, but 2) school-age health should have a less negative longer-term relationship with achievement than poor prenatal/infant health (Model 1).⁶

To test the third hypothesis—when the duration of poor health is longer, or when health declines, the influence of school-age health on persistent academic inequality should be more negative than when poor health is transitory—I extend the analysis described above to examine the duration and stability of poor health. First, to measure the duration of school-age health, I regress achievement intercepts and slopes on the count measure, whereby a condition at more ages increases the duration score (Model 2). Second, I use a latent variable framework to more dynamically capture the stability and ordering of poor health (Model 3). Here, latent school-age health trajectories become predictors of latent achievement trajectories. This multivariate latent growth curve model, or “parallel process” model, captures propensity rather than absolute presence/absence of health at a particular age. Modeling health trajectories as predictors is therefore equivalent to modeling a change in the propensity for poor health. Because the metric for the latent variable underlying the observed dichotomous measures for poor health at each school age is assumed to be z -distributed, the units on both sides of the equation are standardized units (Bollen and Curran

⁶In the final models I do not treat covariates as time-varying (except in Model 3, described below). I conducted several sensitivity analyses that extended this model to treat health and sociodemographic characteristics as having time-varying effects, whereby health or SES at a particular age “perturbed” the latent achievement trajectory. These models, which are more complex, did not change the substantive results.

2006). In order to provide an intuitive interpretation, I use the means and variances from each model's normal distribution to compute 95th percentile bounds $\left(\bar{x} \pm 1.96 \sqrt{\sigma^2}\right)$ and implied trajectories for three groups of children: those continuously healthy between ages 7-16 ("low illness propensity"), those continuously unhealthy ("high illness propensity"), and those who become increasingly unhealthy ("low to high illness propensity"). This method of measuring duration and stability permits health to be measured prospectively and reveals how changes in the direction of health are associated with achievement trajectories.

The final model tests the fourth hypothesis from cumulative inequality theory: 4) high levels of family capital weaken the negative influence of poor child health on early achievement disparities and achievement trajectories (Model 4). Here I interact health (prenatal/infant, age 7 health, duration at ages 11/16) with family social capital—mothers' time spent reading with young children—to test the moderating influence of family capital on academic trajectories. For ease of interpretation I present these results using the count measure of duration, though results using latent duration/stability do not differ substantively and are available by request. In all models, I rely on three common fit indices to guide model choice: the Comparative Fit Index (CFI), the Tucker-Lewis Index (TLI) and the Root-mean-square error of approximation (RMSEA). An ideal fit for the CFI and TLI is 1, and for the RMSEA, less than or equal to 0.05 (Bollen and Curran 2006).

FINDINGS

Early Health, Timing and Cumulative Inequality in Academic Achievement

Table 2, which disaggregates average achievement by health status and age, reveals clear variation in reading and math achievement across health categories. Respondents with no childhood health conditions score highest on reading and math assessments at ages 7, 11 and 16. In contrast, low-birth weight respondents, those exposed to heavy prenatal smoking late in utero, and those with early school-age health limitations perform more poorly, ranging from 0.10 to 0.5 of a standard deviation below average. Children with health conditions at all school ages perform nearly a full standard deviation lower in math and reading.⁷

Next, I examine these trajectories within a multivariate framework. Tables 3 and 4 show the influence of prenatal/infant and school-age health on reading and math achievement trajectories, respectively, controlling for the child, family and household characteristics in Table 1 (except for maternal reading behavior). Each panel shows the results for separate models, and primary independent variables are listed across the columns. Within each panel, the "Achievement Intercept" row shows the relationship between each predictor and age 7 achievement, while the "Achievement Slope" row shows each predictor's relationship to achievement trajectories between ages 7-16. The tables reveal, first, that prenatal/infant health is related to age 7 achievement: low birthweight is associated with significantly reduced reading and math achievement at age 7 (0.273 and 0.304 z-score units, respectively), as is exposure to late prenatal smoking in medium and heavy amounts.

⁷With the exception of maternal smoking, the categories in Table 2 are not mutually exclusive and are simply meant to demonstrate variation in achievement by health.

Second, poor health at age 7 is related to significantly lower age 7 reading and math performance (0.616 and 0.411 standardized units, respectively).

Providing evidence for Hypotheses 1 and 2—whether child health is associated with *persistent and accumulating* academic disadvantage—requires examination of not only the latent intercepts (differences at age 7) but also the slopes. Model 1 in Table 4 shows that the association between prenatal smoking exposure and math trajectories is negative between ages 7 and 16, at 0.006 z-score units/year for medium smoking exposure, and 0.007 units/year for heavy smoking exposure. Although the direction of the relationship between smoking and reading trajectories is similar, it is not significant. Math achievement gaps stemming from low birth weight compound over time, but this is not the case for reading. Turning to school-age health, Model 1 in Tables 3 and 4 shows that the relative academic disadvantage associated with poor health at age 7 is predicted to decline over the course of later childhood/adolescence—the reading and math achievement slopes become more positive over time (0.029 and 0.021 z-scores per year, respectively), revealing that these children partially catch up as they age. While the relative math and reading disadvantage of children exposed to heavy prenatal smoking widens with age, the achievement disadvantage among children with early school-age poor health declines with age. These patterns are consistent with Hypothesis 1, which predicts that achievement gaps stemming from prenatal/infant health should widen with age. The results are only partially consistent with the predictions of Hypothesis 2—while the influence of age 7 health on achievement trajectories is less negative than that of prenatal/infant health, it is actually positive and larger in absolute magnitude, whereby the initial achievement gap between children with and without a condition closes with age.

The magnitude of these relationships over time is easiest to visualize in the form of predicted values that depict trajectories for a child with otherwise average characteristics. Figures 1A and 1B display the predicted math and reading z-scores between ages 7 and 16, disaggregated by prenatal/infant health (prenatal smoking exposure and birth weight) and age 7 health—all other sample characteristics are held constant at their means. The graphs show the slightly widening inequalities in math achievement predicted from prenatal smoking exposure. A child not exposed to heavy smoking late in the mother's pregnancy, with otherwise average characteristics, is predicted to perform significantly higher than a child with heavy exposure at age 7, and to increase in his or her relative math performance with age, compared to a more stable predicted pattern of relative achievement for a similar peer exposed to heavy smoking. By age 16, the difference for this hypothetical child is expected to be almost 0.2 of a standard deviation.

In contrast, disaggregating by age 7 health reveals a significantly lower level of math achievement at age 7 for children in poor health (about 0.5 of a standard deviation), but a declining gap as children age. That is, an average child with a health condition is predicted to experience an improvement in relative math achievement compared to an otherwise similar, but healthier, peer. Overall, the findings in Tables 3 and 4, and Figure 1, provide some support for Hypotheses 1 and 2. An unhealthy prenatal health environment, indicated by exposure to smoking, is associated with early and growing achievement disadvantage, while early school-aged poor health is associated with early, but stable or declining,

achievement disadvantage. The findings for low birth weight are consistent with the predictions of Hypothesis 1 for math, but not reading.

The Duration and Stability of Poor Health

Next, I test a more nuanced prediction from cumulative inequality theory—academic trajectories are dependent on not only the timing of health disadvantage, but also its duration and stability. Model 2 in Tables 3 and 4 presents findings using the count measure of duration that increases with poor health at multiple ages. The findings demonstrate that measuring the duration of poor health offers a more complex picture of the association between health and persistent academic inequality. Model 2 and Figures 2A/B show how a longer duration of poor health can alter the achievement trajectory that would otherwise be predicted from shorter-term poor health. Figure 2A shows that, while children with a condition only at age 7 partially catch up to their healthier peers in reading achievement by age 16, this pattern is weaker for those with a health problem at both ages 7 and 11, and does not exist for those in poor health at ages 7, 11 and 16. Figure 2B shows a similar pattern for math achievement—children in poor health at all school ages do not catch up to their peers to the same degree as their peers with shorter-term poor health. By age 16, those in poor health at all ages perform about 0.7 and 0.5 standard deviations lower than their healthiest peers in reading and math achievement, respectively.

Model 3 in Tables 3 and 4 presents complementary findings from an alternative measure of duration that better measures the stability and ordering of poor health. I examine how latent school-age health trajectories predict latent reading and math trajectories—this approach captures a change in the propensity for poor health during the school years, rather than simply modeling the presence or absence of a condition at a particular age. Examining parallel trajectories of poor health and achievement reveals a strong relationship, whereby a standardized unit increase in the propensity for poor health between ages 7-16 (“poor health slope”) is related to a significant annual reduction in reading and math *z*-scores (−0.185 and −0.154 reading and math standardized units, respectively).

A more intuitive interpretation is again permitted by examining predicted values. Using the models’ implied trajectories, I take the 95th percentile bounds from the latent variable’s normal distribution and compute implied trajectories for three groups of children—those who are continuously healthy (“low illness propensity,” those at the bottom of the distribution at each age), continuously unhealthy (“high illness propensity,” those who remain the high end of the distribution), and increasingly unhealthy (“low to high illness propensity,” those moving from the low to the high end of the distribution). Children’s other characteristics are again held constant at their means. Figures 3A and 3B show these predicted values for reading and math achievement. Figure 3A shows that a standard unit increase in the propensity for poor health is related to about a 0.185 *z*-score decrease in reading achievement per year: by age 16 this is predicted to result in a substantial decline in achievement (almost a full standard deviation), relative to a peer who is continuously healthy. Those in continuously poor health (high illness propensity) are predicted to remain steadily disadvantaged in reading achievement, relative to their peers who experience the

onset of poor health or who are continuously healthy. Figure 3B presents similar patterns for math achievement.

The findings summarized in Figures 2 and 3 are generally consistent with the predictions of Hypothesis 3 and provide evidence for the importance of duration and stability—when poor health is experienced at multiple ages, or when health declines, its relationship with persistent academic inequality is more negative than when poor health is transitory. It is also worth noting that, after measuring duration, prenatal smoking continues to be associated with early and growing achievement gaps, and poor health at age 7 is associated with early but declining achievement disadvantage. In other words, the association of exposure to late prenatal smoking with academic trajectories may not simply reflect the tendency of unhealthy young children to become unhealthy adolescents.

The Moderating Influence of Family Capital

Finally, I examine the possibility that family capital buffers the negative influence of poor health on academic trajectories. I extend the findings in Tables 3 and 4 to test the moderating influence of family social capital—mothers' time spent reading with children—on reading and math trajectories. For ease of interpretation I present findings with the count measure of duration, though findings using the latent measure of poor health are substantively very similar. Table 5 summarizes these findings in the form of predicted values, with the full findings reported in Table A1. In line with the earlier analyses, children with no health conditions consistently achieve the highest reading and math scores. Moreover, consistent with the small body of existing research, there is little evidence that family capital moderates the influence of prenatal/infant health. Though children with unhealthy prenatal and infant environments have higher age 7 achievement when they are in higher-capital families, these differences are small and derive from insignificant regression coefficients.

There is stronger evidence, however, that family capital partially compensates for the negative influence of poor school-age health on achievement, and that this buffering is particularly pronounced in the early school years. Though they still lag behind their healthier peers, children in poor health at age 7 whose mothers frequently spend time reading with them have stronger age 7 reading and math achievement than their peers: 0.193 standard deviations (-0.333 vs. -0.526) for age 7 reading achievement, and 0.18 for math. These differences remain fairly stable during the remainder of the school years, suggesting that the compensatory influence of family capital may have a shorter-term impact on the degree of inequality in achievement. This is notable, though, since early academic learning provides a base for additional learning.

Additional Analyses

I perform several supplementary tests to address the possibility that the observed influence of health may reflect unobserved differences in the quality of children's environments as they age. These results are not shown but are available by request. First, I examine whether unhealthy parenting behaviors throughout childhood explain the academic influence of prenatal health environments, and find that they do not. Controlling for a number of health-

related parenting behaviors later in childhood, including preventive doctor's visits and parents' immunization decisions, does not explain the influence of prenatal/infant health environments. Second, I estimate models with child fixed effects to examine whether changes in health predict changes in achievement, net of unobserved, time-invariant differences between children. A significant relationship remains in this analysis, suggesting that unobserved, time-invariant differences between children are not the sole driver of the association between child health and academic trajectories. In a related approach, I estimate a model that centers time-varying health and sociodemographic variables around their child-specific means. This model, which produces both within and between-child estimates, generates coefficients for the cluster mean of school-age health (the proportion of ages at which a child has a health problem), and for the deviation of an age from the cluster mean of health. These two coefficients, respectively, compare different children with and without health problems, and the effect of changing health status within a child over time. This multilevel model yields very similar findings for the direction and significance of the relationship between health and achievement: a) for the "between-child" coefficient, children in poor health perform lower than their peers at age 16, and b) for the "within-child" coefficient, children who acquire a health problem perform more poorly at age 16 than those who do not.

DISCUSSION

Efforts to equalize educational opportunity among school-aged children have long been a priority in industrialized nations, motivated by the importance of educational attainment for social mobility. Adding complexity to this research and policy focus is the recognition that skill-based inequality emerges very early in the life course. From a convincing body of research we have learned how child health is an important determinant of early achievement inequality, as well as eventual educational attainment and income. Here I argue that our understanding of the role of health in the social stratification process is enriched by testing several tenets of cumulative inequality theory: 1) that inequality accumulates as children age; 2) that the degree to which achievement gaps accumulate is specific to the developmental stage being considered; 3) that this process depends on the duration and stability of poor health; and 4) that it is moderated by children's family resources.

Informing the study of health and social stratification with insights from cumulative inequality theory reveals several findings that deepen our understanding of when and how health is related to the emergence of academic inequality. I find some evidence for the first two hypotheses: adverse prenatal/infant health environments and poor school-aged health are negatively related to early academic achievement and, measured statically, poor school-age health is not associated with widening achievement inequality during the school years—its impact becomes less negative over time. In contrast, children exposed to late prenatal smoking increase their achievement at a slower pace relative to their otherwise similar peers who experience a healthier prenatal environment. These results are consistent with the idea that early life adversity negatively impacts life course processes (e.g., Conley, Strully and Bennett 2003; McLeod and Almazan 2003), and that the accumulation of inequality is tied to age-specific developmental processes. Health disadvantage occurring early in life, during

key developmental periods, may produce large baseline inequalities in learning that compound as children age, whereby children lag behind their peers in successive learning.

The results also reveal the importance of the duration and stability of poor health for achievement trajectories. Though a static conceptualization of school-age health suggested only a short-term negative impact on achievement, measuring the duration and stability of poor school-age health highlights the more persistent impact of the *accumulation* of poor health on achievement trajectories. Children with a longer duration of poor health are less likely to catch up in academic performance to their healthier peers than children with shorter-term poor health. Moreover, examining the stability of health in a latent variable framework reveals that the learning advantage that healthy children begin with early in the school years is predicted to decrease substantially by adolescence if they acquire conditions as they age. This finding suggests that children's cumulative experience of poor health is important for understanding how academic—and, subsequently, socioeconomic—inequality is generated.

Finally, testing whether family capital buffers the negative influence of poor child health on academic trajectories yields mixed findings. Children in poor health but with high levels of family capital—measured by mothers' time reading with children—have stronger reading and math achievement in the early school years than their peers in the lower-capital group. It is important to note, however, that all children in poor health lag behind their healthier peers in achievement, regardless of family capital levels. That these differences remain stable during the remainder of the school years suggests that family capital may have a shorter-term impact, but that family resources in the early school years may aid less healthy children in acquiring a stronger base for learning. It is notable that the compensatory role of family capital is weaker for children with unhealthy prenatal and infant exposures. On the surface, this finding suggests that it may be more difficult to compensate for adversity experienced very early in life. However, the magnitude of academic gaps stemming from unhealthy early exposures is also smaller than those related to poor school-age health.

The results from the measure of family capital used here should motivate future work examining a greater number of family resources that can be observed as children age. Parents may adjust their behaviors with children as they age, for example, in response to their performance and to the severity of their health conditions. When data permit, it will be useful to examine parents' economic and educational behaviors toward children at more time points, including in the years prior to school entry, and to examine how these behaviors are sensitive to the accumulation of health conditions. Forms of capital outside of the family also merit consideration, as children encounter institutions beyond the family as they age, and the resources within these other contexts—especially schools—may provide important resources that can help children maintain strong performance despite a health condition (Parcel, Dufur and Zito 2010).

The merits of the data and approach used here should be weighed against some limitations. First, these broad measures of school-age health do not isolate the influence of any particular condition. Error in the measurement of the true construct of “health” may produce an underestimated influence of health. Though I combine conditions to maximize sample size

and maintain measurement consistency over time, in supplementary analyses I separately analyze broad categories of mental and physical health, and find that both domains have strong associations with achievement trajectories. Greater detail in the measurement of school-age health is certainly an important task for future research. Despite imperfect measurement, examining health longitudinally permits observation throughout the early life course, which is often not possible when examining a particular condition, and affords examination of cumulative inequality processes. Relatedly, while the results in this research offer improved measurement of duration and stability, many additional questions will merit examination as life course data beginning in childhood become increasingly available. For example, is the accumulation of new health conditions equivalent to the experience of one continual chronic condition?

Second, as with any statistical model applied to observational data, the empirical approach used here does not permit causal claims. Though I observe a rich set of individual, family and household characteristics, these findings are upper-bound and potentially indirect estimates (though the imperfect measures of health used here may produce lower-bound estimates). For example, I cannot fully disentangle the effects of mothers' prenatal smoking from those of mothers' smoking during childhood. Though I control for mothers' smoking during children's adolescence, it is possible that the prenatal smoking coefficient partially reflects the influence of correlated behaviors during childhood that remain unmeasured in these data.

Finally, though not a limitation of the analysis, it is nonetheless useful to situate these findings in their historical context. A third of mothers in 1950s Britain smoked late in pregnancy, for example. This is not an anomaly, instead reflecting a particular historical context in which today's strong socioeconomic and parenting gradients in smoking and health behaviors were far weaker. Though it is possible to speculate about implications for contemporary patterns in different climates of "healthy parenting," these results may differ from what would be observed in contemporary Britain. The stronger socioeconomic gradients that are observed today in both health behaviors and parenting behavior related to cognitive development (e.g., Schaub 2010) suggest that, in the contemporary U.K. and U.S., parental behaviors are increasingly a marker for socioeconomic status. This could mean that a stronger compensatory role of family capital may be observed in more contemporary settings, as parents with high-achieving children are increasingly those with the highest levels of capital. This possibility merits consideration in future work, and will be increasingly possible as respondents in contemporary nationally representative surveys become old enough to be observed during the entirety of the school years. More generally, these results among members of the late "baby boom" generation in the U.K. may differ from what would be observed in other contexts and periods.

The study of child health and academic achievement is of central relevance to our understanding of health and social inequality, given increasing recognition of the early reproduction of educational and socioeconomic inequality, longstanding evidence documenting sensitive periods of child development for skill development and effective learning, and the demonstrated sensitivity of skill and achievement to unhealthy exposures and poor health. Models of cumulative inequality predict compounding group differences

over time, emphasizing the ways in which the accumulation of inequality should be sensitive to timing and duration, and the ways in which it may be reversible in favorable environmental circumstances. This analysis advances our understanding of health and social stratification by suggesting not only that the relationship between health and academic achievement emerges very early in life, but that whether we observe contraction or widening of inequality as children age depends on how and when we measure their health and environments. Revealing how this relationship unfolds during the school years is essential for developing an understanding of when and how to intervene.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Appendix

Table A1

Achievement Trajectories, Prenatal/Infant Health, Poor School-Age Health and Mothers' Reading Behavior at Age 7: NCDS, 1958-1974 (N=9,252)^{*}

	Reading		Math	
Model 4	Intercept (α)	Slope (β)	Intercept (α)	Slope (β)
Intercept	-1.785 ^{**} (0.10)	-0.020 (0.01)	-1.395 ^{**} (0.11)	-0.087 ^{**} (0.01)
Frequent Reading, Age 7	0.018 [*] (0.01)	0.010 ^{**} (0.00)	0.018 (0.04)	0.00 (0.01)
<i>Prenatal/Infant Health</i>				
Low BW	-0.251 ^{**} (0.01)	0.00 (0.01)	-0.252 [*] (0.06)	0.00 (0.01)
Low BW [*] Frequent Reading	0.031 (0.08)	0.01 (0.01)	0.098 (0.09)	0.013 (0.01)
Late Pregnancy Smoking: Variable/Medium	-0.086 ^{**} (0.03)	-0.01 (0.00)	-0.058 [*] (0.03)	-0.006 (0.00)
Variable/Medium Smoking [*] Frequent Reading	0.044 (0.05)	0.01 (0.01)	0.01 (0.05)	0.003 (0.01)
Late Pregnancy Smoking: Heavy	-0.104 ^{**} (0.04)	0.001 (0.00)	-0.084 ^{**} (0.04)	-0.010 [*] (0.01)
Heavy Smoking [*] Frequent Reading	0.065 (0.05)	-0.01 (0.01)	0.030 (0.06)	0.003 (0.01)
<i>School-Age Health</i>				
Poor Health, Age 7	-0.674 ^{**} (0.05)	0.061 ^{**} (0.01)	-0.512 ^{**} (0.05)	0.044 ^{**} (0.01)
Poor Health Age 7 [*] Frequent Reading	0.157 [*] (0.07)	-0.012 (0.01)	0.151 (0.07)	-0.010 (0.01)
Duration, Age 16		-0.021 ^{**} (0.00)		-0.010 ^{**} (0.00)
Duration Age 16 [*] Frequent Reading		0.004 (0.01)		0.01 (0.01)
<i>Fit Statistics</i>				
χ^2 (df)		376.6 (27)		346.3 (27)
RMSEA		0.03		0.03
TLI		0.97		0.97
CFI		0.95		0.94
N		9,252		9,252

* Models control for child characteristics listed in Table 1. Reference category for maternal reading frequency is occasionally/never.

[†] p<.10

^{*} p<.05

^{**} p <.01

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Note: Adjusted for individual and family characteristics.

Figure 1.
Predicted Achievement Trajectories by Prenatal/Infant and School-Age Health

Note: Adjusted for individual and family characteristics.

Figure 2.
Predicted Achievement Trajectories by Duration

Note: Adjusted for individual and family characteristics.

Figure 3.
Predicted Achievement Trajectories by Latent Duration/Stability

Table 1

Descriptive Characteristics of Birth-Age 16 Sample: NCDS, 1958-1974 (N=9,252)

	% unless mean indicated (SD)	Age of measurement	Metric used in final models	Predictor of intercept or slope
<i>Prenatal/Infant Health Environment</i>				
Low birthweight	5	Birth	Binary	Both, All Models
Mother smoked heavily	12	Birth	Binary	Both, All Models
Mother smoked "medium/variable" amount	22	Birth	Binary	Both, All Models
<i>School-Age Health</i>				
Condition, Age 7	6	7	Binary	Both, Model 1
Condition, Age 11	9	11	Binary	
Condition, Age 16	10	16	Binary	
<i>Duration, Age 16 Count (0-3)</i>		7, 11, 16	Interval	Slope, Model 2
Condition, One age	15			
Condition, Two ages	3			
Condition, Ages 7, 11, 16	2			
<i>Child Characteristics</i>				
Sex (male=1)	52	Birth	Binary	Both, All Models
Average childhood class	skilled manual	Birth, 7, 11, 16	Interval	Both, All Models
Mother married, birth	96	Birth	Binary	Both, All Models
Average maternal grandfather's social class, birth	skilled manual/non-manual	Birth	Interval	Both, All Models
Average monthly family income (pounds), age 16	180.6 (81.5)	16	Interval	Both, All Models
Average age mother finished school	15-16 years old	16	Interval	Both, All Models
Average number of children in household	1.77 (0.92)	7, 11, 16	Interval	Both, All Models
Average childhood access to basic resources	sole use of one facility	7, 11, 16	Interval	Both, All Models
Mother employed for pay, birth	0.55	Birth	Binary	Both, All Models
Average number of moves during childhood	1.63 (1.48)	16	Interval	Both, All Models
Breastfed as infant	69	Birth	Binary	Both, All Models
<i>Family Capital</i>				
Mothers' reading frequency to child, age 7		7	Categorical	Both, Model 4
At least weekly	47			
Occasionally	34			
Hardly ever	19			

Table 2

Mean Academic Achievement by Health, Birth-Age 16 Sample: NCDS, 1958-1974 (N=9,252)

	No Health Condition	Low BW	Medium/Variable Smoking	Heavy Prenatal Smoking	Condition at Age 7	Condition at Ages 7, 11 and 16
<i>Age 7</i>						
Mean Reading Z-Score	0.139	-0.304	-0.046	-0.084	-0.661	-1.561
Mean Math Z-Score	0.087	-0.321	-0.053	-0.085	-0.439	-1.026
<i>Age 11</i>						
Mean Reading Z-Score	0.102	-0.337	-0.101	-0.144	-0.454	-1.196
Mean Math Z-Score	0.101	-0.367	-0.081	-0.160	-0.411	-0.952
<i>Age 16</i>						
Mean Reading Z-Score	0.115	-0.317	-0.115	-0.160	-0.432	-1.206
Mean Math Z-Score	0.084	-0.321	-0.149	-0.196	-0.388	-0.706
N	7806	445	419	1080	586	126

Table 3

Regression of Reading Achievement Trajectories on Prenatal/Infant and School-Age Health: NCDS, 1958-1974 (N=9,252)^a

	Intercept	Medium Prenatal Smoking	Heavy Prenatal Smoking	Low BW	Poor Health, Age 7	Duration, Age 16	Poor Health Intercept	Poor Health Slope	Fit Statistics
Model 1: Prenatal/School-Age Health									
Achievement Intercept (α)	-1.653** (0.11)	-0.073** (0.02)	-0.081** (0.03)	-0.273** (0.040)	-0.616** (0.04)				72.31 (18)
Achievement Slope (β)	-0.021* (0.010)	-0.002 (0.003)	-0.001 (.004)	0.003 (0.005)	0.029** (0.005)				
χ ² (df)									
RMSEA									
TLI									0.02
CFI									0.99
Model 2: Duration									
Achievement Intercept (α)	-1.787** (0.11)	-0.074** (0.02)	-0.084** (0.03)	-0.274** (0.04)	-0.627** (0.04)				154.6 (19)
Achievement Slope (β)	-0.018* (0.01)	-0.002 (0.00)	-0.001 (0.00)	0.003 (0.01)	0.058** (0.01)	-0.019** (0.00)			
χ ² (df)									
RMSEA									
TLI									0.02
CFI									0.99
Model 3: Duration/Stability									
Achievement Intercept (α)	-1.793** (0.11)	-0.076** (0.02)	-0.086** (0.03)	-0.262** (0.040)			-0.192** (0.02)		264 (66)
Achievement Slope (β)	-0.021† (0.01)	-0.003 (0.003)	-0.002 (.004)	0.001 (0.005)			0.010** (0.00)	-0.185** (0.05)	
χ ² (df)									
RMSEA									
TLI									0.01
CFI									0.95

^aModels control for child characteristics listed in Table 1.

† p<.10

* p<.05

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Regression of Math Achievement Trajectories on Prenatal/Infant and School-Age Health: NCDS, 1958-1974 (N=9,252)^a

Table 4

	Intercept	Medium Prenatal Smoking	Heavy Prenatal Smoking	Low BW	Poor Health, Age 7	Duration, Age 16	Poor Health Intercept	Poor Health Slope	Fit Statistics
Model 1: Prenatal/School-Age Health									
Achievement Intercept (α)	-1.251** (0.12)	-0.076** (0.03)	-0.073** (0.03)	-0.304** (0.05)	-0.411** (0.04)				
Achievement Slope (β)	-0.084** (0.02)	-0.006* (0.00)	-0.007* (0.00)	-0.007* (0.00)	0.021** (0.00)				
χ^2 (df)									96.88 (18)
RMSEA									0.02
TLI									0.98
CFI									0.99
Model 2: Duration									
Achievement Intercept (α)	-1.397** (0.12)	-0.074** (0.02)	-0.077** (0.03)	-0.309** (0.04)	-0.451** (0.04)				
Achievement Slope (β)	-0.091** (0.01)	-0.008** (0.00)	-0.010** (0.00)	0.007 (0.00)	0.035** (0.01)	-0.010** (0.00)			
χ^2 (df)									143.2 (19)
RMSEA									0.02
TLI									0.98
CFI									0.99
Model 3: Duration/Stability									
Achievement Intercept (α)	-1.366** (0.12)	-0.079** (0.03)	-0.079** (0.03)	-0.296** (0.05)			-0.165** (0.02)		
Achievement Slope (β)	-0.095** (0.02)	-0.010* (0.00)	-0.010* (0.00)	0.007 (0.01)			0.009* (0.00)	-0.154* (0.06)	
χ^2 (df)									304.6 (66)
RMSEA									0.02
TLI									0.92
CFI									0.94

^aModels control for child characteristics listed in Table 1.

[†] p<.10

* p<.05

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Table 5

Predicted Achievement Z-Scores by Maternal Reading Behavior: NCDS, 1958-1974 *

	Age 7			Age 11			Age 16		
	Read Frequently			Read Frequently			Read Frequently		
	Read Frequently	Read Infrequently		Read Frequently	Read Infrequently		Read Frequently	Read Infrequently	
Reading									
No Condition	0.184	0.148		0.209	0.127		0.24	0.1	
Low Birthweight	-0.025	-0.091		0.037	-0.112		0.116	-0.138	
Heavy Late Prenatal Smoking	0.158	0.073		0.151	0.065		0.143	0.054	
Condition at Age 7	-0.333	-0.526		-0.18	-0.387		0.011	-0.213	
Condition at Ages 7, 11, 16	-0.333	-0.527		-0.316	-0.555		-0.295	-0.591	
Math									
No Condition	0.574	0.545		0.33	0.255		0.024	-0.107	
Low Birthweight	0.428	0.307		0.233	0.016		-0.01	-0.347	
Heavy Late Prenatal Smoking	0.532	0.484		0.27	0.163		-0.062	-0.238	
Condition at Age 7	0.213	0.033		0.105	-0.121		-0.03	-0.314	
Condition at Ages 7, 11, 16	0.213	0.033		0.105	-0.2		-0.03	-0.494	

* Values predicted from regression estimates in Table A1