7-1-2017

The Origins of Early Childhood Anthropometric Persistence

Augustine Denteh
Georgia State University

Daniel L. Millimet
Southern Methodist University & IZA

Rusty Tchernis
Georgia State University, rtchernis@gsu.edu

W. J. Usery Workplace Research Group Paper Series

Working Paper 2017-7-1
July 2017

The Origins of Early Childhood Anthropometric Persistence

Augustine Denteh
Georgia State University

Daniel L. Millimet
Southern Methodist University and IZA

Rusty Tchernis
Georgia State University, NBER and IZA

Georgia State University
Andrew Young School of Policy Studies

The Origins of Early Childhood Anthropometric Persistence

Augustine Denteh

Georgia State University

Daniel L. Millimet

Southern Methodist University & IZA

Rusty Tchernis*

Georgia State University, NBER & IZA

July 5, 2017

Abstract
The rates of childhood obesity have increased dramatically in the last few decades. Non-causal evidence suggests that childhood obesity is highly persistent over the life cycle. However, little is known about the origins of this persistence. This paper examines the evolution of anthropometric measures from birth through primary school. We provide estimates of the causal effect of past anthropometric outcomes on future anthropometric outcomes (state dependence) and investigate the importance of time varying and time invariant factors in the dynamics of childhood anthropometric measures. We find that anthropometric measures are highly persistent from infancy through primary school. Moreover, most of this persistence is driven by time invariant, unobserved factors that are determined prior to birth, consistent with the so-called fetal origins hypothesis. Thus, policy interventions designed to improve childhood obesity will only have meaningful, long-run effects if these time invariant, unobserved factors are altered. Future research is needed to identify such factors, although evidence suggests that maternal nutrition may play an important role.

JEL: C23, I12, I18
Keywords: Childhood Obesity, Persistence, Fetal Origins Hypothesis

* This study was conducted by Georgia State University and Southern Methodist University under a cooperative agreement with the U.S. Department of Agriculture, Economic Research Service, Food and Nutrition Assistance Research Program (agreement no. 58-5000-0-0080). The views expressed here are those of the authors and do not necessarily reflect those of the USDA or ERS. The authors are grateful to Chris Ruhm for helpful comments and Lorenzo Almada for excellent research assistance. Corresponding author: Rusty Tchernis, Department of Economics, Andrew Young School of Policy Studies, Georgia State University, P.O. Box 3992, Atlanta, GA 30302-3992, USA.

1 Introduction

The rise in childhood obesity in the U.S. is well chronicled (Ogden et al. 2002, 2008, Skinner et al. 2016). Moreover, non-causal evidence suggests that childhood obesity is highly persistent over the life cycle (Serdula et al. 1993, Power et al. 1997). However, little is known about the origins of this persistence. In this paper we attempt to answer three questions. First, how do weight, height, and body mass index (BMI) evolve from birth through primary school? Second, what is the *causal* effect of past weight and height status on the future weight and height of children? In other words, how important is *state dependence* in the evolution of anthropometric measures during the early part of the life cycle. Third, how important are time varying and time invariant factors in the dynamics of childhood anthropometric measures? Our analysis is fundamentally important for researchers and policy makers involved in formulating policies and interventions to address the obesity epidemic. For instance, if obesity originates early in life and is persistent over time, then early childhood intervention is preferable to later in adolescence or beyond.[1] However, and perhaps more importantly, if persistence is due to time invariant underlying factors rather than state dependence, then only by altering these factors can children be moved onto a different trajectory.

The prevalence of obese adolescents has tripled over the last thirty years; it has more than doubled for younger children. Defined as having an age- and sex-adjusted BMI above the 95^{th} percentile of the reference distribution, the prevalence of obese children increased from 5% to 12.4% for 2-5 year old children and from 5% to 17.6% for 12 to 19 year-olds between 1976 and 2006 (Ogden et al. 2008). In addition, vast differences in the time trends of BMI increases have been documented: the incidence of obesity among white girls aged 12-19 has increased from 7.4% to 14.5% between 1988 and 2006, whereas the corresponding figures for African-American girls are 13.2% and 27.7% (Ogden et al. 2002, 2008). Epidemic increases in childhood obesity have also been observed among pre-school children (Deckelbaum & Williams 2001, p.242S).

While there exists some evidence that childhood obesity rates may have begun to recede or at least stabilized in the U.S. (Ogden et al. 2014, 2016), public health concerns over childhood obesity remains high due to the well-documented consequences of obesity and the lack of understanding pertaining to the turnaround.[2] Obesity burdens

[1] For instance, Eriksson et al. (2001, p. 735) conclude that "obesity is initiated early in life." Also, an article in the *New York Times* on March 22, 2010 discusses some evidence suggesting that children may become entrenched "on an obesity trajectory" even before kindergarten; however, the evidence is not "ironclad" (`http://www.nytimes.com/2010/03/23/health/23obese.html?ref=health`.). While public health officials tend to advocate school-based reforms in light of the near universal enrollment, others stress the importance of preschool interventions (e.g., Frisvold & Giri (2014), Dietz & Gortmaker (2001), Davis & Christoffel (1994)).

[2] See, for instance, `http://www.nytimes.com/2013/08/07/health/broad-decline-in-obesity-rate-seen-in-poor-young-children.html?pagewanted=all`.

individuals with severe physical, economic, and emotional suffering, and puts children and adolescents at risk for a number of health problems such as those affecting cardiovascular health, the endocrine system, and mental health (Deckelbaum & Williams 2001, Krebs et al. 2003). For instance, Dietz & Gortmaker (2001) note that 60% of overweight children aged five to ten years old have at least one associated cardiovascular disease risk factor.

Perhaps the most crippling consequence of childhood obesity stems from its impact on adult obesity (Serdula et al. 1993, Power et al. 1997), with its concomitant effects. Currently, 60% of the total U.S. population is overweight or obese and 50% is expected to be obese in 2030 at the current rate (Dor et al. 2010). Walpole et al. (2012) calculate that, due to obesity, North America accounts for 34% of the total human biomass in the world despite containing only 6% of the world population. Moreover, the authors estimate that, if the entire world had the same age-sex BMI distribution as the U.S., total human biomass will increase by 58 million tonnes which would be equivalent to 935 million people of world average BMI. Finkelstein & Zuckerman (2010) report that if the childhood obesity epidemic continues unabated at the current rate, as many as 30-40% of the US population will develop Type 2 Diabetes during their lifetime. Mocan & Tekin (2011) document the links between adult obesity and lower wages, productivity, and self-esteem. In the U.S., the total cost attributable to obesity was over $75 billion in 2000 according to Finkelstein et al. (2004); more recent estimates put the cost over $200 billion (Cawley & Meyerhoefer 2012).

While the changes in childhood obesity rates across cohorts, as well as the consequences of these increases, are well-documented, much less is known about how anthropometric measures of children evolve over the life cycle. A growing literature has investigated persistence in anthropometric outcomes in a non-causal framework, stressing the correlation in outcomes over time.[3] Whitaker et al. (1997) found that the probability of an overweight six year-old child becoming an obese adult is 50% compared to 10% for a non-overweight child. In addition, the risk of becoming obese in adulthood is exacerbated by having an obese parent (Whitaker et al. 1997). Eriksson et al. (2001) found that individuals with BMI greater than 16 as opposed to below 14.5 at age seven were three times more likely to be obese as an adult. Nader et al. (2006) find that children who were overweight prior to the age of 5 are five times as likely to be overweight at 12 relative to children who were not overweight prior to the age of 5.

Freedman et al. (2001) also report a strong relationship between overweight status in childhood and adult BMI. However, they find a striking result that obese adults who were overweight prior to age eight have a much higher BMI than individuals suffering from adult onset obesity (41 versus 35). Van Cleave et al. (2010) analyze changes in

[3]Iughetti et al. (2008) provide an excellent review.

the prevalence of obesity and other chronic conditions (e.g., asthma, other physical and learning conditions). The authors find that prevalence of obesity is increasing and is highly persistent over time. Conversely, many children with chronic conditions at ages two through eight did not have the condition six years later. Finally, Millimet & Tchernis (2013*a*) assess persistence of anthropometric outcomes during infancy and primary school, documenting a significant increase in persistence upon entry into primary school. Deckelbaum & Williams (2001, p. 239S) conclude: "Disturbingly, obesity in childhood, particularly in adolescence is a key predictor for obesity in adulthood." Similarly, Dietz & Gortmaker (2001, p. 340) state: "The best evidence suggests that the majority of overweight adolescents go on to be overweight adults."

In addition to the literature already discussed pertaining to the correlations between childhood weight status and adult obesity, a related literature investigates the persistence in health among adolescents and adults in a *causal* framework. For example, Halliday (2008) investigates persistence in self-reported health status among white adults aged 22-60 using data from the Panel Study of Income Dynamics (PSID) and allows the parameters of the model to vary. The results suggest that the degree of state dependence – the causal effect of past states on one's current state – in health is modest for half the population, yet it explains much of the observed persistence in health for the other half. Ham et al. (2013) analyze persistence in bulimia nervosa in young women. The authors find a substantial role for state dependence in the persistence of bulimia nervosa. Our analysis follows the logic of these studies.

This paper contributes to the existing literature by attempting to understand the origins of the persistence in childhood anthropometric measures. We revisit the question of persistence in early childhood anthropometric outcomes, investigating the relative importance of state dependence (i.e., a causal effect of past anthropometric status on future anthropometric status), unobserved heterogeneity (i.e., unobserved genetic or environmental risk factors), and observed heterogeneity (i.e., commonly measured risk factors) on this persistence. We do so by estimating dynamic regression models using data from two samples: the Early Childhood Longitudinal Survey – Kindergarten Cohort (ECLS-K) and the Early Childhood Longitudinal Survey – Birth Cohort (ECLS-B). The ECLS-K is a nationally representative longitudinal survey of children entering kindergarten in Fall 1998. In addition to providing information on birthweight, anthropometric data is collected at several points in time between kindergarten and eighth grade. The ECLS-B is also a nationally representative longitudinal survey of children born in the U.S. in 2001. Information is provided on these children at ages 9 months, two years, four years, and five years. These two samples are complementary because while the ECLS-K sample permits an examination of anthropometric persistence from

kindergarten through primary school, the ECLS-B sample allows for a more refined examination of anthropometric trajectories prior to kindergarten entry.

Two salient conclusions derive from the analysis in this paper. First, weight, height, and BMI are highly persistent starting in early infancy. Second, majority of this persistence is attributable to time invariant characteristics of children. This finding is crucial as it suggests that only interventions that alter these time invariant attributes will have substantive, long-run effects on a child's anthropometric status. Thus, current policy interventions may, at best, have a marginal impact in the short-run and, at worst, be destined to fail (see, e.g., Davis & Gebremariam (2010)). Moreover, while it is difficult to say what these critical, time invariant attributes are given the data at hand, we find some evidence that fetal nutrition – with mother's pre-pregnancy weight and weight gain during pregnancy, gestation age, birth status (singleton, twin, or higher order birth), and birthweight as proxies – impacts the evolution of anthropometric measures over the early life cycle. However, time invariant, *unobserved* attributes play a much more prominent role.

The remainder of the paper is organized as follows. Section 2 presents the methodology. Section 3 presents the ECLS-K sample analysis. Section 4 presents the ECLS-B sample analysis. Section 5 discusses the results of the analyses and Section 6 concludes.

2 Methodology

We assess the extent and origins of persistence using a dynamic regression framework. This approach allows for the decomposition of persistence into various components reflecting state dependence, observed heterogeneity, and unobserved heterogeneity.

The simplest estimating equation is

$$y_{it} = \gamma y_{it-1} + \varepsilon_{it}, \qquad i = 1, ..., N;\ t = 1, ..., T, \tag{1}$$

where y_{it} is a measure of anthropometric status for child i at time t, ε_{it} is a mean zero error term, and T must be at least two (given observability of the initial observation, y_{i0}). Here, γ reflects the overall level of persistence as it captures the entire association between past and current anthropometric status.

To decompose the overall persistence, we next incorporate *observed* heterogeneity into the model as follows:

$$y_{it} = \gamma y_{it-1} + x_{it}\beta + w_i\delta + \varepsilon_{it}, \qquad i = 1, ..., N;\ t = 1, ..., T, \tag{2}$$

where x_{it} is a vector of observed, time varying attributes of child i at time t and w_i is a vector of observed, time invariant attributes of child i. The change in the estimate of γ from (1) to (2) reflects the portion of persistence attributable to observed heterogeneity.

Finally, we include observed time-varying heterogeneity and all sources (observed and unobserved) of time-invariant heterogeneity into the model as follows

$$y_{it} = \gamma y_{it-1} + x_{it}\beta + \alpha_i + \varepsilon_{it}, \qquad i = 1, ..., N;\ t = 1, ..., T, \tag{3}$$

where α_i is a child-specific fixed effect. In (3) γ reflects the degree of *state dependence* as it captures the causal effect of past weight status on current weight status. The child-specific fixed effect, α, reflects persistence in child anthropometric measures due to persistent observed and unobserved, child-specific heterogeneity (such as time invariant environmental and genetic factors). In such models, β represents the contemporaneous effects of the observed, time varying regressors, whereas $\beta/(1-\gamma)$ represents the long-run effects of a permanent unit change in these variables.

Estimation of (3) is straightforward (assuming the model is correctly specified). Following Anderson & Hsiao (1981), (3) is first-differenced to eliminate α_i. The first-differenced model is then estimated via instrumental variables since the first-differenced lagged dependent variable is necessarily correlated with the first-differenced error term. However, y_{it-2} represents a valid instrument if ε is serially uncorrelated. The models are estimated by Generalized Method of Moments (GMM).

In addition to simply examining the coefficient estimates, we follow the logic in Ulrick (2008) and simulate probabilities (given estimates of the regression model) such as the following:

$$\Pr(y_{iT} \geq y^* | y_{i0} \geq y_0). \tag{4}$$

Here, (4) represents the probability of a child having an anthropometric measure above y^* in the terminal period conditional on an initial measure greater than or equal to some value y_0. For example, one might be interested

in the probability of a child having a BMI above the 85^{th} percentile in period T conditional on being above the 85^{th} percentile in the initial period, $t = 0$. These probabilities incorporate not just the coefficient directly related to persistence, γ, but also reflect persistence due to persistence in observed and unobserved determinants of child weight. Moreover, we can simulate counterfactual probabilities by altering the attributes of individuals.

Before detailing the simulations undertaken, note that upon estimating (3), the estimates of the child-specific fixed effects, α_i, are given by

$$\widehat{\alpha}_i = \frac{1}{T}\sum_{t=1}^{T}\left[y_{it} - \widehat{\gamma}y_{it-1} - x_{it}\widehat{\beta}\right], \qquad i = 1, ..., N. \tag{5}$$

These child-specific fixed effects estimates can then be decomposed into observed and unobserved time invariant factors by estimating the following model using ordinary least squares:

$$\widehat{\alpha}_i = w_i\delta + \eta_i, \tag{6}$$

where w_i now includes an intercept and η_i is a mean zero error term.[4] Finally, given estimates of γ, β, and α, we can obtain estimates of the idiosyncratic errors, ε and η, using (3) and (6).

We now turn to the simulation of probabilities such as those given by (4) under the following counterfactual scenarios:

1. Own y_{i0}, own x_{it}, set $\varepsilon_{it} = 0$, and
   (a) replace $\alpha_i = \overline{\widehat{\alpha}}$, or
   (b) draw $\alpha_i \sim F(\alpha)$ where $F(\cdot)$ is the empirical distribution of α_i.
2. Own y_{i0}, own x_{it}, set $\eta_i = 0$, set $\varepsilon_{it} = 0$, and
   (a) own w_i, or
   (b) draw $w_i \sim F(w)$ where $F(\cdot)$ is the empirical distribution of w_i.
3. Own y_{i0}, own x_{it}, own w_i, set $\varepsilon_{it} = 0$, and draw $\eta_i \sim F(\eta)$ where $F(\cdot)$ is the empirical distribution of η_i.

[4] See for e.g., Hornstein & Greene (2012) and references therein for examples of two-step estimation approaches using a vector of estimated coefficients as a dependent variable.

4. Own y_{i0}, own α_i, set $\varepsilon_{it} = 0$, and

   (a) replace $x_{it} = \overline{x}_t$, or

   (b) draw $x_{i\cdot} \sim F(x_1, ..., x_T)$ where $F(\cdot)$ is the empirical joint distribution of $x_1, ..., x_T$.

5. Own y_{i0}, own x_{it}, own α_i, and draw $\varepsilon_{i\cdot} \sim F(\varepsilon_1, ..., \varepsilon_T)$ where $F(\cdot)$ is the empirical distribution of $\varepsilon_{i\cdot}$.

6. Own y_{i0}, own α_i, and draw $x_{i\cdot}, \varepsilon_{i\cdot} \sim F(x_1, ..., x_T, \varepsilon_1, ..., \varepsilon_T)$ where $F(\cdot)$ is the empirical joint distribution of $x_1, ..., x_T$, and $\varepsilon_1, ..., \varepsilon_T$.

7. Own x_{it}, own w_i, own α_i, own ε_{it}, and

   (a) replace $y_{i0} = \overline{y}_{i0}$, or

   (b) draw $y_{i0} \sim F(y_{i0})$ where $F(\cdot)$ is the empirical distribution of y_{i0}.

Probabilities are obtained using 500 simulations. See Appendix A for further details.

Case 1 eliminates time varying, unobserved heterogeneity, ε_{it}, and assesses the impact of altering the distribution of time invariant heterogeneity, α_i. Case 1a eliminates all time invariant heterogeneity by setting it to the sample mean of $\hat{\alpha}_i$. Cases 1b replaces actual time invariant heterogeneity with a random draw from the empirical distribution of α_i. Case 1b entirely breaks any correlation between the initial condition, y_{i0}, x_{it}, and time invariant heterogeneity, α_i. Put together, these scenarios highlight the relative importance of time invariant heterogeneity in the origin of persistence in child anthropometric measures.

Case 2 continues to eliminate time varying, unobserved heterogeneity, ε_{it}. However, time invariant, unobserved heterogeneity, η_i, is now also eliminated; the observed component of time invariant heterogeneity is then altered. Case 2a uses each observation's own time invariant heterogeneity, w_i. Case 2b draws w_i from the empirical distribution. Case 3 is similar to Case 2, but has individuals retain their time invariant, observed heterogeneity, w_i, and alters the distribution of time invariant, unobserved heterogeneity, η_i. Case 3a draws η_i from the population empirical distribution. Altogether, Cases 2 and 3 permit the assessment of the relative importance of the observed and unobserved components of time invariant heterogeneity in the persistence of child anthropometric outcomes.

Case 4 continues to eliminate time varying, unobserved heterogeneity, ε_{it}, and assesses the impact of altering the distribution of time varying, observed heterogeneity, x_{it}. Case 4a eliminates all time varying heterogeneity. Case 4b replaces actual time varying, observed heterogeneity with a random draw from the empirical distribution

and succeeds in entirely breaking any correlation between the initial condition, y_{i0}, α_i, and time varying, observed heterogeneity, x_{it}. The scenarios in Case 4 complement the simulations performed in Case 1 as they speak to the relative importance of time varying, observed heterogeneity in the persistence of child anthropometric outcomes.

Case 5 has individuals retain their time varying, observed attributes, x_{it}, time invariant attributes, α_i and y_{i0}, but alters the distribution of time varying, unobserved heterogeneity, ε_{it} by drawing $\varepsilon_{i\cdot}$ from the empirical distribution. Case 6 has individuals retain only their time invariant attributes, α_i and y_{i0}. All time varying heterogeneity is sampled. In particular, $x_{i\cdot}$ and $\varepsilon_{i\cdot}$ are drawn from the population empirical distribution. Thus, Cases 5 and 6 address the relative importance of the observed and unobserved components of time-varying heterogeneity in the persistence of childhood anthropometric outcomes.

Finally, Case 7 has individuals retain their time varying, observed attributes, x_{it}, time invariant, observed heterogeneity, w_i, and time invariant, unobserved heterogeneity, α_i, and assesses the importance of altering the initial condition, y_{i0}. While Case 7a replaces the initial condition with the sample mean of y_{i0}, Case 7b replaces the actual initial condition with a random draw from the empirical distribution of y_{i0}.

3 ECLS-K Sample Analysis

3.1 Data

We use data from the restricted version of the ECLS-K. Collected by the U.S. Department of Education, the ECLS-K surveys a nationally representative cohort of children throughout the U.S. in fall and spring kindergarten, fall and spring first grade, spring third grade, spring fifth grade, and spring eighth grade. The sample includes data on over 20,000 students who entered kindergarten in one of roughly 1,000 schools during the 1998-99 school year. In addition to family background information, height and weight measures are available for children in each round, as well as information on birth weight.

Our final sample consists of children for whom we have valid measures of age, gender, height, and weight.[5] From the information on height and weight of the children, we obtain z-scores for weight, height, and BMI. Note that z-scores and percentiles are based on CDC 2000 growth charts; these are age- and gender-specific, are adjusted for

[5]The initial sample size of the ECLS-K is 21,260. After cleaning age, weight, and height as described in Millimet & Tchernis (2013*b*, Appendix C), and due to sample attrition, the sample size falls to 9,360 in the final wave of the data. Restricting the sample to a balanced panel reduces the sample size to approximately 9,160. This is the final sample size per wave in the analysis.

normal growth, and percentiles are based on the underlying reference population.[6] The estimation utilizes data from five waves: fall kindergarten, spring first grade, spring third grade, spring fifth grade, and spring eighth grade.[7] The sample is a balanced a panel of roughly 9,160 children.[8]

The following time invariant covariates are included in the regression model: gender, race/ethnicity (white, black, Hispanic, Asian, and other), birthweight, indicator for premature birth, indicator for being born in the U.S., indicator for being a native English speaker, city type (urban, suburban, or rural), region (northeast, midwest, south, and west), mother's education (less than high school, high school/GED, some college, four-year college degree, and more than four years of college), mother's age at first birth, mother's marital status at birth, mother's height, mothers pre-pregnancy weight, mother's weight gain during pregnancy, gestation age, birth status (singleton, twin, or higher order birth), indicator for attending nonparental pre-kindergarten, indicator for mother's labor force participation during infancy, indicator for mother's participation in WIC (Women's, Infants, and Children) during pregnancy, indicator for mother's participation in WIC during infancy, indicator for mother's participation in TANF (Temporary Assistance for Needy Families) during infancy, indicator for participation in FSP (Food Stamp Program) during infancy, and indicator for attending full day kindergarten.[9]

The following time varying covariates are included in the regression model: an index of socioeconomic status (SES), indicator for the household being in poverty, number of children's books in the household, household size, family type (two parents plus siblings, two parents and no siblings, one parent and siblings, one parent and no siblings, and other), mother's labor force participation status (full-time, part-time, and not working), mother's weight, indicator for mother absent from the household, indicator of current TANF participation, indicator of current FSP participation, indicator for health insurance, hours spent watching television during the school week, hours spent watching television during the weekend, indicator for household rules regarding television watching, days per week household eats breakfast together, days per week household eats dinner together, indicator for household food security (household never worried about running out of food), neighborhood safety (very safe, somewhat safe, and not safe), and percent of minority students in class at school. For all covariates (except gender, age, height, and weight), we include dummy variables for missing observations.

[6] z-scores and their percentiles are obtained using the -*zanthro*- command in Stata.

[7] The survey design is troublesome in that the ECLS-K contains irregularly spaced waves. To minimize the issue, we omit the spring kindergarten wave and thus each period conceptually represents roughly a two-year window.

[8] Note, all sample sizes are rounded to the nearest 10 per NCES restricted data regulations.

[9] FSP was renamed the Supplemental Nutrition Assistance Program (SNAP) in October 2008. Since the data pre-dates this change, we refer to the program as FSP.

Table 1 presents descriptive statistics of the above time varying and time invariant covariates. Although we use data from five waves of the ECLS-K, Table 1 presents means and standard deviations (in parenthesis) for the final sample after restrictions for the first wave (see first column of Table 1) and the initial sample before restrictions (see the second column of Table 1). Although we do not discuss the summary statistics in detail for brevity, the sample restrictions we implement, as well as attrition do not lead to a drastically different sample, at least at the mean, except a few cases such as mother's marital status at birth, number of children's books in household, indicator for child's health insurance status, indicator for neighborhood being very safe, and the indicator for the household never been worried about running out of food.

3.2 Results

Table 3 displays the results from estimation of equations (1), (2), and (3) for weight, height, and BMI z–scores, respectively. In addition to reporting estimates of the coefficient on the lagged outcome, γ, we report the first-stage Kleibergen & Paap (2006) Wald rk F-statistic, the Kleibergen & Paap (2006) rk test of underidentification, and a test of endogeneity. The first two tests are designed to detect any issues associated with weak instruments. Finally, recall that the estimate of γ from (1) reflects the overall level of persistence, the change in the estimate of γ moving from (1) to (2) captures the portion of persistence explained by the observable covariates, and the change moving from (2) to (3) reflects the portion of persistence explained by unobserved, time invariant factors.

Table 4 presents the dynamic simulations based on (4), which affords further analysis of the sources of persistence, especially in the upper tail of the distribution of anthropometric measures, and the role of time varying and time invariant observed attributes. As noted earlier, the simulations are based on the estimates of the fixed effects specification given in (3), along with the subsequent estimates of the fixed effects and their decomposition given in (5) and (6). From the regression results For each outcome, we simulate three sets of probabilities:

1. $\Pr(y_{iT} \geq 85^{\text{th}} \text{ percentile } | y_{i0} \geq 85^{\text{th}} \text{ percentile})$,
2. $\Pr(y_{iT} \geq 95^{\text{th}} \text{ percentile } | y_{i0} \geq 95^{\text{th}} \text{ percentile})$, and
3. $\Pr(y_{iT} \geq 85^{\text{th}} \text{ percentile } | y_{i0} \leq 50^{\text{th}} \text{ percentile})$,

where period T denotes spring eighth grade and period 0 corresponds to fall kindergarten. Note, the percentile outcomes are based on the underlying reference population used in the CDC 2000 growth charts, *not* the current

sample. Thus, the 85th and 95th percentiles correspond to usual cutoffs for overweight and obese when examining BMI. Finally, Table 4 also presents the *benchmark* probability, which is the empirical probability observed in the data (i.e., the sample probability as opposed to an estimate), for comparison.

3.2.1 Weight

Panel I of Table 3 displays the regression results for weight z-scores. The estimates of γ across the three specifications are 0.931, 0.932, and 0.775 (standard errors are 0.003, 0.003, and 0.067, respectively) in Columns (1), (2), and (3) respectively. Each coefficient estimate is statistically significant at the 1% confidence level and all three specifications are strongly identified. The estimates of γ above 0.9 indicate a substantial degree of childhood anthropometric persistence. Moreover, the fact that the estimate of γ does not change moving from Column (1) to Column (2) implies that our lengthy vector of time varying and time invariant observed factors explain *none* of the persistence in weight status for primary–school–aged children. Thus, while we find extreme persistence from one period to the next, this persistence is not attributable to or explained by characteristics typically observed by policy makers or health practitioners.

Moving to the specification in Column (3), which replaces the time invariant observed factors with child-level fixed effects and thereby accounts for all time invariant attributes of the child, the estimate of γ falls to 0.775, a decline of roughly 17% from 0.93 in Columns (1) and (2). This implies that time invariant, unobserved factors explain about 17% of the observed persistence in weight z–scores. Examples of such factors include genetic endowments, prior health shocks determined *in utero* or during infancy, time invariant environmental factors such as the presence of grocery stores or outdoor amenities, etc.

In sum, we find that overall persistence is fairly extreme as a one standard deviation increase in weight is associated with roughly nine-tenth of a standard deviation increase in the subsequent period. However, time-varying and time invariant observed attributes explain none of this persistence. Furthermore, time invariant unobserved factors also explain very little of the persistence (less than one-fifth). Thus, much of the persistence in child weight is attributable to state dependence, which implies that early interventions that are successful in reducing child weight will have long-run effects. Unfortunately, since our covariates explain little of the variation in weight, identifying such early interventions may be difficult.[10]

[10]The full set of results are available upon request. While some estimated coefficients are statistically significant at conventional levels, the magnitudes are quite small; even the long-run effects of permanent changes in the covariates, given by $\beta/(1-\gamma)$, are quite small. That said, while our covariate set does include a wide array of the usual family background variables, we do not have information on

Table 4 displays the simulation results for $\Pr(y_{iT} \geq 85^{\text{th}}$ percentile $|y_{i0} \geq 85^{\text{th}}$ percentile), $\Pr(y_{iT} \geq 95^{\text{th}}$ percentile $|y_{i0} \geq 95^{\text{th}}$ percentile), and $\Pr(y_{iT} \geq 85^{\text{th}}$ percentile $|y_{i0} \leq 50^{\text{th}}$ percentile) for weight, height, and BMI z–scores in Columns (1)-(3), (4)-(6), and (7)-(8) respectively.

Simulated Probabilities: $Pr(y_{iT} \geq 85^{th}$ percentile $|y_{i0} \geq 85^{th}$ percentile)

Focusing on Column (1) of Table 4, the benchmark probability is 0.84. In other words, 84% of children above the 85^{th} percentile in the initial period remain above the 85^{th} percentile in the terminal period in our sample. This is consistent with a high degree of persistence in weight in the upper tail of the distribution. We turn to the simulations to explore the sources of this persistence.

Panel I in Table 4 contains the simulated probabilities when time varying unobservables are ignored (i.e., $\varepsilon_{it} = 0$ for all i, t) and time invariant heterogeneity is altered first by removing it entirely (i.e., setting α to the sample mean of $\widehat{\alpha}$) and then by retaining the heterogeneity in α, but breaking its correlation with x and y_0 by giving each child a random draw from the empirical distribution of $\widehat{\alpha}$. In the first case, the conditional probability of staying above the 85^{th} percentile falls to about 0.753 while it falls to about 0.576 in the second case. The fact that the conditional staying probability drops noticeably from the benchmark in the second case, but only marginally in the first case, indicates that it is not the *variation* in α across children that influences persistence, but rather the *correlation* between α and the time varying covariates. In fact, the correlation between time invariant heterogeneity and the time varying covariates explain a little over 30% of total persistence in weight status (i.e., $1 - (0.576/0.84) = 0.314$). Moreover, since the results in Table 1 indicate that the time varying, observed covariates, x, have little explanatory power, it is the correlation between α and the initial condition, y_0, that explains nearly one-third of the total persistence in weight status. In other words, children with high initial conditions – measured by weight z–scores upon kindergarten entry – also have high values of α, and this combination is responsible for one-third of the conditional staying probability over the span of kindergarten through eighth grade.

Panels II and III in Table 4 assess whether the importance of α is driven by time invariant observed factors, w, or unobserved factors, η. The first simulation in Panel II sets η equal to zero and keeps w at its actual value. The result is very similar to the first case in Panel I, where α is set equal to its sample mean. In this case, the conditional staying probability is 0.727, implying that the results in Panel I were driven by having set η to its sample mean.

many recent interventions designed to combat obesity, such as education efforts, healthy food programs, and efforts to promote physical activity. We also do not have data on parents' height or weight. We return to the issue of parental anthropometric status later.

When children were instead given a random draw for w from its empirical distribution, the probability changes only modestly to 0.703. Again, this is consistent with the results in Table 3 where we found little explanatory power for the time invariant, observed covariates. In Panel III, however, when children retain their own observed factors, x and w, but receive a random draw for η from its empirical distribution, the conditional staying probability falls to 0.591. As such, it is the correlation between time invariant, *unobserved* factors and the initial condition, y_0, that is responsible for roughly one-third of the conditional staying probability. In other words, children with high initial conditions also have high values of η, and this combination is responsible for one-third of the persistence in weight from kindergarten through eighth grade.

Panels IV, V, and VI in Table 4 report the simulated probabilities obtained when children retain their time invariant heterogeneity, α, but receive draws of either time varying, observed covariates, x, or unobserved attributes, ε, or both from their respective empirical distributions. The results indicate no impact from altering either, which is consistent with the prior results in Table 3.

Lastly, Panel VII in Table 4 presents the simulated probabilities when the initial condition, y_0, is altered by first, setting it equal to the sample mean for all i, t and second, by giving each child a random draw from the empirical distribution of y_0. In the first case, by removing all heterogeneity in the initial condition (and thus, entirely breaking its correlation with α and x), the conditional probability of staying above the 85^{th} percentile falls from 0.840 to about 0.337. When children are instead given a random draw from the empirical distribution of y_0, the conditional staying probability falls only to 0.583.

In sum, the simulations indicate that about one-third of the conditional probability of staying above the 85^{th} percentile for weight is due to *time invariant*, *unobserved* risk factors such as genetic endowments, early life health shocks, time invariant environmental factors, etc. In spite of the increased role of time invariant, unobserved heterogeneity, a greater portion of the persistence in weight status is still attributable to state dependence as evidenced by the regressions in Table 1. The fact that about two-thirds of persistence in weight status is due to state dependence is encouraging in that early interventions, to the extent that they are successful in reducing weight prior to kindergarten, can have long-run effects on weight during middle school.

Simulated Probabilities: $Pr(y_{iT} \geq 95^{th}$ *percentile* $|y_{i0} \geq 95^{th}$ *percentile*)

Column (2) of Table 4 displays the analogous results for $\Pr(y_{iT} \geq 95^{\text{th}}$ percentile $|y_{i0} \geq 95^{\text{th}}$ percentile). Com-

pared to the results in Column (1) of Table 4, three primary differences emerge. First, the benchmark probability of 0.762 is lower for the full sample. Second, the vast majority of the persistence is due to variations in time invariant heterogeneity, α, across children even more so than in Column (1). In fact, the conditional probability of staying above the 95$^{\text{th}}$ percentile falls to about 0.134 when the time invariant heterogeneity is altered by removing it entirely (i.e., setting α to the sample mean of $\widehat{\alpha}$) in Panel I of Column (2). State dependence, as well as time varying factors, x and ε, do not play much of a role in explaining persistence in the extreme upper tail. Third, unlike in Column (1) of Table 4, we find that setting η to zero in Panel II results in a relatively greater reduction in the conditional staying probability than in Panel III when η is replaced by random draws from different empirical distributions (i.e., about 45% vs. about 30%). This indicates that giving children initially above the 95$^{\text{th}}$ percentile an average draw from the distribution of η (i.e., setting η to zero) is sufficient to bump most of these children below the 95$^{\text{th}}$ percentile by the terminal period, whereas this is not sufficient when using the 85$^{\text{th}}$ percentile as the threshold.

Simulated Probabilities: $Pr(y_{iT} \geq 85^{th}$ percentile $|y_{i0} \leq 50^{th}$ percentile)

Finally, Column (3) of Table 4 presents the results for Pr($y_{iT} \geq 85^{\text{th}}$ percentile $|y_{i0} \leq 50^{\text{th}}$ percentile). This case illuminates factors associated with relatively extreme weight gain during early childhood (i.e., sizable upward mobility as opposed to persistence). In terms of the benchmark case, the probability of moving from below the median at kindergarten entry to above the 85$^{\text{th}}$ percentile by the end of eighth grade is roughly 12%.

Turning to the simulations, we obtain a few noteworthy findings. First, time varying factors, x and ε, continue not to play any meaningful role. Second, replacing α with the sample mean reduces the probability of crossing the 85$^{\text{th}}$ percentile conditional on starting below the median to zero. Replacing α with a random draw from different empirical distributions roughly doubles the probability of crossing the 85$^{\text{th}}$ percentile relative to the benchmark. Similarly, replacing the initial condition with a random draw from the empirical distribution of y_0 roughly doubles the conditional probability of crossing the 85$^{\text{th}}$ percentile relative to the benchmark. Put together, these results imply that children initially below the median tend to have favorable values of α. Specifically, α is not randomly distributed in the population, but rather has a positive (partial) correlation with the initial condition, y_0. Only the few children with extremely unfavorable draws of α, despite being below the median in the initial period, experience extreme upward mobility. Moreover, if α were randomly assigned, the probability of moving from below the median to above the 85$^{\text{th}}$ percentile would roughly double. This is a testament to the importance of time invariant factors

(not state dependence), in determining the probability of extreme upward mobility in weight status.

Third, the effect of randomly assigning α is due to randomly assigning time invariant, unobserved factors, η. Randomly assigning the time invariant, observed factors, w, has little impact on the probability of extreme upward mobility. Moreover, removing time invariant, unobserved factors by setting η to zero reduces the probability of extreme upward mobility to nearly zero. The implication is that children below the median tend to have favorable draws of α, which really means favorable draws of time invariant, unobserved factors, η.

Overall, the evidence presented thus far points to an important role for state dependence in explaining persistence in weight status on average but less so in the upper right tail of the weight distribution, where time invariant, unobserved heterogeneity assumes a greater role. Next we turn to the analysis of height

3.2.2 Height

While height *per se* is not a policy concern in the U.S., it is interesting to compare the dynamics of height with those of weight. In addition, it is useful to examine the individual components of BMI prior to assessing BMI z–scores in the next section.

Panel II of Table 1 displays the results for height z–scores. The estimates of γ across the first two specifications are very similar to those using weight z-scores; namely, 0.937 and 0.936 (standard errors are 0.004 and 0.004, respectively). However, the estimate of γ falls to 0.603 (standard error is 0.048) in the fixed effect specification (compared to 0.775 in Table 1). Similar to Panel I of Table 1, the estimate of γ is statistically significant at the $p < 0.01$ confidence level, and all three specifications are strongly identified. The estimate of γ barely changes when we include time varying and time invariant observed attributes, and the estimates of γ above 0.9 in the first two specifications indicate a substantial degree of persistence in height. Thus, as for weight in Panel I of Table 1, while height is quite persistent from one period to the next, this is not attributable to or explained by observed characteristics.

However, in contrast to weight z–scores, the inclusion of child-level fixed effects explains about 36% (i.e., $1 - (0.603/0.936) = 0.356$) of the overall persistence in child height (versus only 17% for weight z–scores). This is perhaps not surprising as unobserved biological factors – most noticeably, parental height – are not included in our set of observed covariates. The fact that time invariant, unobserved attributes account for a greater share of the persistence in height implies that state dependence, and thus the long-run impact of successful, early interventions – that do not alter relevant, time invariant, unobserved attributes – is diminished.

Columns (4)-(6) of Table 4 present the analogous set of simulation results for height z–scores. In terms of the benchmark probabilities, a few differences emerge relative to the prior results for weight. First, the benchmark probabilities are lower for height than the corresponding probabilities for weight. For example, $\Pr(y_{iT} \geq 85^{\text{th}}$ percentile $|y_{i0} \geq 85^{\text{th}}$ percentile) and $\Pr(y_{iT} \geq 95^{\text{th}}$ percentile $|y_{i0} \geq 95^{\text{th}}$ percentile) are 0.606 and 0.467, respectively, for height; these probabilities are 0.840 and 0.762, respectively, for weight. Thus, persistence in the upper half of the distribution is lower, albeit still high, for height. Second, while there may exist more mobility in terms of height, extreme upward mobility for height is less common than for weight – $\Pr(y_{iT} \geq 85^{\text{th}}$ percentile $|y_{i0} \leq 50^{\text{th}}$ percentile) is 0.030 for height and 0.118 for weight.

Turning to the simulations, a few patterns emerge. First, while the time varying factors, x and ε, have a bit more impact on height than weight, their combined effect is still modest. In Columns (4)-(6) of Table 4, replacing x and/or ε with different values increases the conditional staying probabilities in all cases (See Panels IV through VI). This indicates that, on average, children initially above the median tend to have less favorable (in terms of raising height) time varying attributes, partially offsetting the child's height in the initial period.

Second, most of the persistence in height is attributable to time invariant factors captured by α, but with different patterns when compared to prior results for weight. For instance, in Columns (4)-(6) of Table 4, we find that replacing α with the sample mean drops the conditional staying probabilities above the 85^{th} and 95^{th} percentiles to zero. Further analysis reveals that this stems from the unobserved component captured by η; varying the time invariant, observed component, w, has little effect. This implies that children in the upper tail of the height distribution upon entry to kindergarten possess time invariant, unobserved attributes that tend to keep them in the upper tail. Replacing these attributes with the sample mean or a random draw of α essentially guarantees these children will fall out of the upper tail by the end of eighth grade. Replacing the unobserved component of the fixed effects, η, with a random draw similarly reduces the conditional staying probabilities, but not as much; the probabilities fall to around 0.256 and 0.108 in Columns (4) and (5) of Table 4, respectively. In addition, replacing the initial condition with a random draw from the empirical distribution reduces the conditional probabilities of staying above the 85^{th} and 95^{th} percentiles to 0.241 and 0.435. This is perhaps not surprising as genetics and early biological factors presumably play a large role in determining child height.

Third, Column (6) of Table 4 suggests that extreme upward mobility in height is rare since children initially below the median have unfavorable draws of time invariant, unobserved heterogeneity, η. Replacing η with its

sample average would eliminate extreme upward mobility entirely as the few cases of observed extreme upward mobility is due to a handful of children having very favorable values of η despite being below the median upon entry into kindergarten. On the other hand, replacing η with a random draw would increase extreme upward mobility by almost four–fold. This pattern is similar to the impact of replacing the initial condition with a random draw where the conditional probability increases from 0.030 to 0.153. In sum, although on average, state dependence is still important in explaining persistence in height (i.e., almost two-thirds), persistence in the upper tail of the height distribution is almost entirely attributable to time invariant, unobserved heterogeneity. We now turn to the analysis of BMI.

3.2.3 BMI

Panel III of Table 3 resents the regression results for BMI z–scores. For the full sample, the estimates of γ across the first two specifications are very similar to those in Panels I and II of Table 3; namely, 0.912 and 0.911 (standard errors are 0.004 and 0.005, respectively). However, the estimate of γ now falls to 0.217 (standard error is 0.015) in the fixed effect specification (compared to 0.775 and 0.603 in Panels I and II of Table 3, respectively). As in Panels I and II of Table 3, the estimate of γ is statistically significant at the $p < 0.01$ confidence level and all three specifications are strongly identified. Also, the estimate of γ barely changes when we include time varying and time invariant observed attributes, and the estimates of γ above 0.9 in the first two specifications indicate a substantial degree of persistence. Thus, as with weight and height z–scores, while persistence from one period to the next in BMI z–scores is high, it is not attributable to or explained by observed characteristics.

While the first two specifications differ little across Panels I, II, and III of Table 3, the results from the fixed effect specification have more pronounced differences. As noted above, time invariant, unobserved factors account for roughly 17% of the total persistence in weight z–scores and 36% for height z–scores. In sharp contrast and perhaps, surprisingly, the fixed effects account for nearly 80% of total persistence in BMI. The economically and statistically meaningful drop in the estimate of γ implies a substantially smaller role for state dependence in the persistence of child BMI. Consequently, the long-run impact of early interventions – that do not alter relevant, time invariant, unobserved attributes – on BMI is quite small. For example, a *one-time* intervention that reduces a child's BMI prior to kindergarten entry by one standard deviation, *ceteris paribus*, is expected to have essentially no impact on BMI in spring eighth grade. A *permanent* intervention that reduces a child's BMI by 0.10 standard deviations *every period*,

will only result in a long-run decrease in the child's BMI of roughly 0.13 standard deviations. This has profound implications for the types of policies one should pursue if the objective is to reverse the obesity epidemic.

Columns (7)-(9) of Table 4 displays the simulation results for BMI z–scores. In Columns (7) and (8), the benchmark probabilities lie in between the conditional staying probabilities for weight and height reported in the corresponding Columns (1)-(2) and Columns (4)-(5). Furthermore, the benchmark probabilities are consistent with the high degree of persistence in BMI documented earlier. For example, the conditional probability of staying above the 85^{th} percentile is 0.746 (see Column (7) of Table 4) and 0.715 for staying above the 95^{th} percentile (see Column (8) of Table 4).

Turning to the simulated probabilities, a few findings stand out. First, altering the values of the time invariant components in Panels I, II, and III of Columns (7) and (8) in Table 4 yields results that are qualitatively similar to those reported in Columns (4) and (5) for height. In particular, in Panel I, we find that replacing α with the sample mean reduces the conditional probability of staying above the 85^{th} and 95^{th} percentiles to zero in nearly every case. Moreover, this is predominantly due to the salient role of time invariant, unobserved factors, η. However, the impact of replacing the initial condition with the mean of y_0 as well as a random draw from the empirical distribution is similar to the reduction in the conditional probability that results from replacing α with a random draw. Variation in time invariant, observed factors, w, explain a modest amount of variation in the conditional probability of staying above the 85^{th} percentile (see Column (7)), but not when using the 95^{th} percentile as the threshold (see Column (8)). Thus, the results are consistent with children in the upper part of the BMI distribution possessing less favorable time invariant factors, particularly those unobserved. Second, as in all the analysis of weight and height, we find very little role for variation in time varying factors, either observed or unobserved.

Column (9) of Table 4 presents the results for $\Pr(y_{iT} \geq 85^{\text{th}} \text{ percentile } | y_{i0} \leq 50^{\text{th}} \text{ percentile})$. In terms of the benchmark probabilities for extreme upward mobility, we obtain higher probabilities for BMI than either weight or height. For example, the probability of having a BMI above the 85^{th} percentile in the terminal period conditional on entering kindergarten below the median is 0.142 (see Column (9)) while the corresponding figures are 0.118 and 0.003 for weight and height, respectively.

Turning to the simulations in Column (9) of Table 4, we obtain a few noteworthy findings. First, time varying factors, x and ε, continue to not play an important role. Second, replacing α with the sample mean reduces the probability of crossing the 85^{th} percentile conditional on starting below the median to zero, just as in Columns (3)

and (6) of Table 4. Replacing α with a random draw from different empirical distributions increases the probability of crossing the 85^{th} percentile by roughly two–and–a–half fold relative to the benchmark (see Column (9) of Panel I in Table 4). Again, a similar increase in the conditional probability obtains for replacing the initial condition with a random draw from the empirical distribution. Together, these results continue to imply that children initially below the median tend to have favorable values of α. Only a few children with extremely unfavorable draws of α, despite being initially below the median, experience extreme upward mobility. Moreover, if α were randomly assigned, the probability of moving from below the median to above the 85^{th} percentile would increase substantially.

Third, the effect of altering α is due to altering the time invariant, unobserved factors, η. However, as in Column (7), the time invariant, observed factors, w, explain a modest amount of the variation in the probability of extreme upward mobility. Specifically, whereas removing time invariant, unobserved factors by setting η to zero reduces the probability of extreme upward mobility to nearly zero for weight and height, this is not the case for BMI as the probability varies from roughly 4% to 5%.

Summarizing, while persistence in BMI, on average, is largely attributable to time invariant, unobserved heterogeneity, it is almost entirely driven by the same in the upper tail of the BMI distribution. State dependence plays the least role in explaining persistence in BMI compared to height and weight. An interesting insight that follows from the ECLS-K sample analysis is that examining persistence in BMI alone can potentially mask the importance of state dependence relative to time invariant, unobserved heterogeneity as the former plays a bigger role in the persistence of the components of BMI (i.e., weight and height). The remainder of this paper undertakes a complementary analysis of the ECLS-B sample, comparing and contrasting its results to those of ECLS-K sample analysis.

4 ECLS-B Sample Analysis

4.1 Data

To explore the early life origins of childhood anthropometric persistence, we use data from the restricted version of the ECLS-B. Collected by the US Department of Education, the ECLS-B collects information on a nationally representative cohort of children born in 2001 at 9 months of age, two years, four years, and five years. As with the ECLS-K, our final sample consists of a balanced sample of children for whom we have valid measures of age, gender,

height, and weight.[11] Given the age of the sample, we convert weight into z–scores; height is however measured in centimeters.

The following time invariant covariates are included in the regression model: gender, race/ethnicity (white, black, Hispanic, Asian, and other), mother's age at first birth, birthweight indicators (normal or low), indicator for intrauterine growth retardation (less than 10%, 10-24%, 25-49%, 50-75%, 76-89%, and 90% and above)[12], indicator for premature birth, indicator for birth status (singleton, twin, or higher order birth), mother's height, mother's weight prior to pregnancy, mother's weight gain during pregnancy, indicator for prenatal care (inadequate, intermediate, adequate, or adequate plus), indicator for maternal prenatal vitamin consumption within the three months preceding conception, indicator for maternal prenatal vitamin consumption during the first trimester, indicator for maternal smoking within the three months preceding conception, indicator for maternal smoking within the third trimester, indicator for whether mother has smoked more than 100 cigarettes in her lifetime, indicator for maternal alcohol consumption within the three months preceding conception, number of current smokers in the household, region (northeast, midwest, south, and west), city type (urban, suburban, or rural), indicator for mother's participation in WIC during pregnancy, indicator for mother's participation in WIC during infancy, and scores on infant mental and motor assessments administered at 9 months.

The following time varying covariates are also included: age, mother's education (less than high school, high school/GED, some college, four-year college degree, and more than four years of college), an index of SES status, indicator for the household being in poverty, number of children's books in the household, household size, family type (two parents plus siblings, two parents and no siblings, one parent and siblings, one parent and no siblings, and other), indicator for biological mother present, indicator for biological father present, indicator for no father present, indicator for no mother present, indicator for parental respondent's marital status, indicator of current TANF participation, indicator of current FSP participation, indicator for health insurance, indicator for current medicaid participation, indicator for current WIC participation, indicator for household food security (household never worried about running out of food), hours per day spent watching television during the school week, indicator for household rules regarding television watching, neighborhood safety (very safe, somewhat safe, and not safe), mother's labor force status (full-time, part-time, and not working), indicators for primary child care arrangement

[11] The possible sample size is roughly 6,950; the initial sample size in the first wave is about 10,700. After restricting the sample to those with valid data on age, gender, height, and weight, the sample size reduces to approximately 5,450. This is the final sample size per wave used in the regression analysis. Note that all sample sizes are rounded to the nearest 50 per NCES restricted data regulations for the ECLS-B.

[12] Intrauterine growth retardation measures the ratio of birthweight to predicted weight based on gestation age.

(parents, other relatives, non-relatives, center-based care, or Head Start), indicator for school enrollment, indicator if English is the primary home language, and mother's weight. For all covariates (except gender, age, height, and weight), we include dummy variables for missing observations.

Similarly, Table 2 presents descriptive statistics of the time varying and time invariant covariates in the ECLS-B sample. Although we use data from four waves of the ECLS-B, Table 2 presents means and standard deviations (in parenthesis) for the final sample after restrictions for the first wave (see first column of Table 2) and the initial sample before restrictions (see the second column of Table 2). Again, at least at the mean, the summary statistics do not suggest that the sample restrictions we implement result in a significantly different sample.

Also, by comparing the first columns of Tables 1 and 2, there are no significant observed differences between the ECLS-K and ECLS-B samples' covariates at the mean, except for a few attributes such as the percent living in urban, suburban, and rural areas, the percent of households with two parents and siblings, the percent of households with two parents and no siblings, the percent of mothers that do not work, and the percent of households that never worried about running out of food.

4.2 Results

The results for the ECLS-B sample are presented in Tables 5 and 6. Table 5 displays the regression estimates while Table 6 presents the simulation results.

In terms of the coefficient estimates, the results in Panel I of Table 5 using weight z–scores are fairly similar to those obtained using the ECLS-K when child-specific fixed effects are omitted. Specifically, the estimates of γ are statistically significant and range from 0.870 to 0.873. The fact that the estimate of γ does not change moving from (1) to (2) implies that our lengthy vector of time varying and time invariant observed factors explain none of the persistence in weight status for infants and young children. Given the additional time invariant controls available in the ECLS-B, this result is striking. Moreover, the estimates of γ near 0.9 indicate a substantial degree of persistence even prior to kindergarten. However, unlike in the ECLS-K sample, the inclusion of child-level fixed effects explains the majority of this persistence. Here, the estimate of γ falls to 0.124 (standard error of 0.013). This implies that time invariant, unobserved factors explain about 85% of the observed persistence in weight z–scores during early childhood. Recall that, in contrast, only 17% of observed persistence in weight z–scores during primary school (ECLS-K sample) is due to time invariant, unobserved heterogeneity. Again, given that we observe many more time

invariant attributes of children in the ECLS-B, this is a startling result.

Panel II of Table 5 displays the corresponding regression results for height. Four interesting patterns emerge. First, persistence in height in the models not controlling for any other covariates – based on the specification in (1) – is of a much smaller magnitude than found in the ECLS-K when assessing height for older children or in the ECLS-B when assessing weight. Second, when controlling for observed heterogeneity – based on the specification in (2) – persistence actually increases by about 15%. This is consistent with a negative correlation between the initial condition for height, y_0, which is really "length" at nine months of age, and observed heterogeneity associated with greater height. Finally, when child-level fixed effects are included, the estimates of γ become *negative* and statistically insignificant. Thus, *all* of the persistence in child height up to age five is attributable to *time invariant*, *unobserved* heterogeneity.

Columns (1) - (3) and (4) - (6) of Table 6 report the results of the same simulations performed using the ECLS-K. In the interest of brevity, we only briefly summarize the results of the simulations. First, time varying attributes, both observed and unobserved have no effect on persistence. Given the lengthy vector of attributes, as well as the plethora of time varying, unobserved attributes captured by ε, this continues to be a noteworthy finding.

Second, time invariant heterogeneity continues to play a prominent role in understanding persistence in child weight and height. For weight, replacing α with its sample mean explains virtually all persistence through age five. Moreover, replacing the fixed effect of a child initially below the median with the sample mean roughly doubles the probability that the child's weight will exceed the 85^{th} percentile at age five. However, replacing the fixed effect of a child initially below the median with the sample mean does not alter the probability that the child's height will exceed the 85^{th} percentile at age five. In addition, for weight, replacing the initial condition with either the mean of y_0 or a random draw from the empirical distribution reduces the conditional probabilities of staying above the 85^{th} and 95^{th} percentiles with sizes comparable to the impact of altering the time invariant, unobserved heterogeneity. Similar patterns in the reduction of conditional probabilities due to altering the initial condition are observed for height.

Third, time invariant, *observed* attributes play a more prominent role, particularly for height, in explaining persistence up to age five than in the ECLS-K sample of primary school children. This could be attributed to two sources. On the one hand, the time invariant, observed attributes may play a more important role in the determination of child weight and height prior to age five. On the other hand, the vector of controls is not identical across the

two data sources. Examining the results of (6) for the ECLS-B sample, the most important covariates relate to birthweight, birth status (i.e., singleton, twin, or higher order birth), intrauterine growth retardation, breastfeeding duration, mother's height, and mother's weight gain during pregnancy. That said, as measured by the R^2, only 19% (22%) of the variation in $\widehat{\alpha}$ is explained by the covariates included in (6) when examining weight (height) for the ECLS-B sample and even smaller for the ECLS-K sample (i.e., 4.5%, 5%, and 6% for weight, height and BMI, respectively).[13]

5 Discussion

This section compares and contrasts the analyses of the ECLS-K and ECLS-B samples, and summarizes the salient findings of this paper.

First, the results from the sample of children aged five and younger in the ECLS-B are consistent with the sample of primary school children in the ECLS-K: persistence in weight and height is substantially high, and this persistence is generally driven by time invariant, unobserved heterogeneity.

Second, for the ECLS-K sample on the one hand, while persistence in BMI is high over the period spanning kindergarten through eighth grade, predominantly driven by time invariant, unobserved heterogeneity, focusing separately on weight and height reveals a nontrivial role for state dependence. In other words, in the ECLS-K sample, a seemingly counter-intuitive finding is that examining persistence in BMI masks the importance of state dependence in childhood weight and height, at least on average (as evidenced by results in Table 3). For instance, while over 80% and 64% of the persistence in weight and height (on average), respectively, is attributable to state dependence, nearly 80% of the persistence in BMI (on average), is explained by time invariant heterogeneity. Nonetheless, it is worth reiterating that persistence in the upper tail of the distribution of height and BMI is almost entirely driven by time invariant, unobserved heterogeneity. Thus, policy makers seeking to address the childhood obesity epidemic can potentially do more if the early childhood interventions also alter relevant, time invariant, unobserved attributes. For the ECLS-B sample on the other hand, persistence in weight and height is predominantly driven time invariant, unobserved heterogeneity; state dependence plays very little role.

Third, time invariant, observed heterogeneity plays little role in both samples, albeit its role is slightly more pronounced in the ECLS-B for reasons mentioned in the previous section. In the ECLS-K sample, children who are

[13]The full regressions for $\widehat{\alpha}$ are available upon request from the authors.

male or black, rural or northeast residents, non-native English speakers, had a high birthweight, and have a mother with low education, a low age at first birth, or who participated in the labor force during the child's infancy tend to have higher BMI (as evidenced by inspection of the estimation results of (6)). In the ECLS-B sample, while the associations between birthweight, gestation age, maternal height and weight, and single versus multiple birth and fetal development are not strong, perhaps due to the complexities involving these relationships that are only currently beginning to be understood in the medical literature, these controls do play a small role in explaining persistence. Nonetheless, the primary determinants of fetal and infant development that may be critical in placing children on an "obesity trajectory" remain unobserved, even in the ECLS-B.

Lastly, a noteworthy finding that persists across the two samples is the almost negligible effect of time varying – observed and unobserved – factors on persistence in childhood anthropometric measures. This result is noteworthy in light of the lengthy vector of time varying, observed factors in both samples as well as the time varying, unobserved attributes captured by the idiosyncratic error terms in the regression specifications.

The foregoing discussion naturally raises questions or concerns about the attributes captured by or reflected in η (i.e., the time invariant, unobserved heterogeneity), at the minimum. From the analysis presented here, one can only conclude that they are not contained in our set of covariates available in the ECLS-K and ECLS-B samples and they do not at least vary during the primary school years. Nonetheless, prior literature posits some possibilities – prenatal attributes such as maternal BMI, maternal weight gain, maternal smoking, periods of undernutrition during pregnancy, gestational diabetes requiring insulin, and post-natal attributes such as breastfeeding, and transitions to solid foods. While we do control for birthweight in both analyses, birthweight alone is not a sufficient proxy for these early influences on fetal development as noted earlier.

Furthermore, a related line of research has documented genetic associations with BMI over the life cycle. For instance, variants of the fat mass and obesity associated (FTO) gene was associated with increased odds of being overweight or obese in adults and children (Frayling et al. 2007, Dina et al. 2007). Fawcett & Barroso (2010) reviews studies documenting associations of single nucleotide polymorphisms (SNPs) in the FTO gene region with BMI in multiple populations as well as approaches aimed at understanding other obesity-associated genes. Genetic abnormalities have also been found to be associated with associated with obesity (e.g., Asai et al. (2013), Ramachandrappa et al. (2013)). An active area of research is deepening our understanding of obesity-associated genes and the mechanisms or pathways through which they impact BMI with the hope of developing therapeutic interventions.

Genetic associations with BMI may be moderated by environment factors. While prior evidence suggests that time invariant, environmental factors *per se* are not likely to play a significant role, gene-by-environment interactions may play a nontrivial role. For example, prior studies using twins that are raised apart conclude that familial environment does not play an important role (Eriksson et al. 2001). However, recent research finds genetic associations with BMI vary by cohort and may be contingent on the environmental context. Using longitudinal data from the Framingham Heart Study, Rosenquist et al. (2015) finds that the association between the rs993609 variant of the FTO gene and BMI varies by birth cohorts and across time. Walter et al. (2016) also reports that a polygenic risk score (GRS-BMI) had a greater association with BMI with more recent birth cohorts. Other studies have found similar genetic associations with BMI that reflect cohort effects (see for e.g., Rokholm et al. (2011), Demerath et al. (2013), Guo et al. (2015)). In spite of the empirical evidence supporting genetic associations with BMI, which reflects the role of time invariant, unobserved heterogeneity, much less is known about the particular environmental factors that interact with genetic risks. Depending on the importance of cohort-specific genetic associations with BMI, our findings may not apply to other cohorts. Further research is needed to shed light on these environmental factors and provide guidance to formulate policies that address gene-by-environment interactions.

The findings in this paper imply that, while earlier intervention is preferred to later interventions, *only* interventions that alter the crucial, time invariant, unobserved risk factors captured by η are likely to be effective in combating obesity in the long-run. Interventions that leave the attributes captured by η unaltered are likely to have, at best, minimal short-run effects and little to no long-run effects. This is entirely consistent with the findings in Davis & Gebremariam (2010), where the authors document that community-based interventions designed to combat childhood obesity that were deemed as successful according to the analysis of data collected via randomized control trials did not produce lasting effects. Eventually, children returned to their "natural state" (Davis & Gebremariam 2010, p. 22). The results are also consistent with Figlio et al. (2014) who document constant effects of birthweight (conditional on gestation length) on cognitive outcomes throughout primary school.

The notion that attributes determined at or shortly after birth, and thus time invariant over the life of an individual, play a dominant role in the evolution of obesity is *consistent* with the strong evidence in economics and elsewhere on the so-called fetal origins hypothesis (see, e.g., Almond & Currie (2011)), with quite profound implications.[14] The fetal origins hypothesis, also referred to as the thrifty phenotype hypothesis or Barker's hypothesis (due to Barker's

[14]While the findings in this paper are consistent with the fetal origins hypothesis, we cannot eliminate other possible explanations for what these salient, unobserved attributes entail.

original publication in 1992), posits long-run effects of conditions *in utero* during critical periods of development through "programmed" changes in the physiology and metabolism of individuals (Barker 1997).

Beginning with Barker's work (Barker 1992), there is a strong belief that *in utero* events may determine whether a fetus ends up on an "obesity trajectory." Deckelbaum & Williams (2001, p. 239S) note that "emerging data suggest associations between the influence of maternal and fetal factors during intrauterine growth and growth during the first year of life, on risk of later development of adult obesity and its comorbidities." More recently, Brisbois et al. (2012) state: "Based on recent evidence, early-life experiences in utero and postnatal influences may induce permanent changes in physiologic function that programme the long-term regulation of energy balance. This subsequently may adversely impact obesity risk in later life."

Factors that may induce such permanent changes in order to set a fetus upon an "obesity trajectory" is the subject of on-going research. While initial hypotheses focused on undernutrition and oxygen supply, additional factors such as maternal BMI, maternal weight gain, maternal smoking, gestational diabetes requiring insulin, and postnatal characteristics such as breastfeeding and the timing of introduction to solid foods are also found to be important (Dietz 1997, Deckelbaum & Williams 2001, Brisbois et al. 2012).

Other research closely related to the literature on the fetal origins hypothesis focuses on the identification of early life physical indicators of predisposition to future obesity. Preliminary results suggest that birthweight, length, and gestation age at birth alone are not strong predictors. Instead, there are complex interactions between these measures, along with other measures such as head circumference, that matter. For example, a fetus born prematurely and, as a result, with low birthweight and length is not likely to be at greater risk of future obesity as long as the fetus' measurements are in proportion and within "normal" ranges given its gestation age. On the other hand, a fetus born with disproportionate physical measurements suggests a greater risk of future obesity (Barker 1997, Godfrey & Barker 2001, Brisbois et al. 2012).

6 Conclusion

This paper investigates the origins of persistence in early childhood anthropometric outcomes in a dynamic regression framework, which permits the decomposition of persistence into components reflecting state dependence, observed heterogeneity, and unobserved heterogeneity. While childhood and adult obesity remain important public health

concerns in the United States, our understanding that interventions earlier in life are likely to have greater impact has deepened over the past two decades. This understanding follows from well-chronicled evidence that obesity is highly persistent; as such, children may become trapped on an "obesity trajectory" early in life. However, little is known about the origins of these correlations in anthropometric measures over the life cycle. Specifically, whether this correlation reflects state dependence, observed heterogeneity, or unobserved heterogeneity is unknown. Moreover, when this persistence in weight status begins – adolescence, early childhood, postnatally, or prenatally – is also unknown.

Better understanding of the dynamics of weight status is crucial for sound policymaking. If weight is highly persistent and the source of this persistence is state dependence, then small (permanent) changes will have large, long-run effects even if the contemporaneous effects are small. However, if persistence is due to biological or environmental factors that are time invariant, then the only changes that will have long-run effects are those that alter these underlying factors.

The evidence presented in this paper indicates, that there is significant persistence in childhood anthropometric measures – weight, height, and BMI – starting during infancy. Moreover, the persistence in BMI is predominantly due to time invariant heterogeneity across individuals determined at birth or shortly thereafter, not state dependence. Moreover, little variation in this time invariant heterogeneity is explained by attributes observed in the data analyzed here.

If the finding that time invariant, unobserved attributes play a dominant role in the evolution of obesity is correct, then the most effective interventions to curb obesity may need to start *prior* to childbirth. Similar policy prescriptions to consider preventive measures preconception, during pregnancy, and throughout early childhood have been advocated elsewhere (Brisbois et al. 2012, Deckelbaum & Williams 2001). These findings suggest that strategies to reverse the current childhood obesity epidemic may need to start even earlier than previously thought, namely *in utero*. Examples of such measures may include altering institutional rules concerning federal nutrition programs such as SNAP or WIC, or education provided under these programs, as they relate to pregnant women (e.g., Baum (2012)).

References

Almond, D. & Currie, J. (2011), 'Killing Me Softly: The Fetal Origins Hypothesis', *The Journal of Economic Perspectives* **25**(3), 153–172.

Anderson, T. W. & Hsiao, C. (1981), 'Estimation of Dynamic Models With Error Components', *Journal of the American Statistical Association* **76**(375), 598–606.

Asai, M., Ramachandrappa, S., Joachim, M., Shen, Y., Zhang, R., Nuthalapati, N., Ramanathan, V., Strochlic, D. E., Ferket, P., Linhart, K., Ho, C., Novoselova, T. V., Garg, S., Ridderstrale, M., Marcus, C., Hirschhorn, J. N., Keogh, J. M., O'Rahilly, S., Chan, L. F., Clark, A. J., Farooqi, S. I. & Majzoub, J. A. (2013), 'Loss of Function of the Melanocortin 2 Receptor Accessory Protein 2 is Associated with Mammalian Obesity', *Science* **341**(6143), 275–278.

Barker, D. J. (1997), 'Maternal Nutrition, Fetal Nutrition, and Disease in Later Life', *Nutrition* **13**(9), 807–813.

Barker, D. J. P. (1992), *Fetal and Infant Origins of Adult Disease*, London:British Medical Journal Publishing.

Baum, C. L. (2012), 'The Effects of Food Stamps on Weight Gained by Expectant Mothers', *Journal of Population Economics* **25**(4), 1307–1340.

Brisbois, T., Farmer, A. & McCargar, L. (2012), 'Early Markers of Adult Obesity: A Review', *Obesity Reviews* **13**(4), 347–367.

Cawley, J. & Meyerhoefer, C. (2012), 'The Medical Care Costs of Obesity: An Instrumental Variables Approach', *Journal of Health Economics* **31**(1), 219–230.

Davis, K. & Christoffel, K. K. (1994), 'Obesity in Preschool and School-age Children: Treatment Early and Often May Be Best', *Archives of Pediatrics & Adolescent Medicine* **148**(12), 1257–1261.

Davis, M. M. & Gebremariam, A. (2010), *Economic Long-Term Impacts of Interventions Aimed at Preventing or Reducing Obesity Among Children*, University of Michigan.

Deckelbaum, R. J. & Williams, C. L. (2001), 'Childhood Obesity: The Health Issue', *Obesity Research* **9**(S11), 239S–243S.

Demerath, E. W., Choh, A. C., Johnson, W., Curran, J. E., Lee, M., Bellis, C., Dyer, T. D., Czerwinski, S. A., Blangero, J. & Towne, B. (2013), 'The positive association of obesity variants with adulthood adiposity strengthens over an 80-year period: a gene-by-birth year interaction', *Human heredity* **75**(2-4), 175–185.

Dietz, W. H. (1997), 'Periods of Risk in Childhood for the Development of Adult Obesity - What Do We Need to Learn?', *The Journal of Nutrition* **127**(9), 1884S–1886S.

Dietz, W. H. & Gortmaker, S. L. (2001), 'Preventing Obesity in Children and Adolescents 1', *Annual Review of Public Health* **22**(1), 337–353.

Dina, C., Meyre, D., Gallina, S., Durand, E., Körner, A., Jacobson, P., Carlsson, L. M., Kiess, W., Vatin, V., Lecoeur, C. et al. (2007), 'Variation in fto contributes to childhood obesity and severe adult obesity', *Nature genetics* **39**(6), 724–726.

Dor, A., Ferguson, C., Langwith, C. & Tan, E. (2010), *A Heavy Burden: The Individual Costs of Being Overweight and Obese in the United States*, Washington, D.C.: Department of Health Policy, School of Public Health and Health Services, The George Washington University.

Eriksson, J., Forsén, T., Tuomilehto, J., Osmond, C. & Barker, D. (2001), 'Size at Birth, Childhood Growth and Obesity in Adult Life', *International Journal of Obesity & Related Metabolic Disorders* **25**(5).

Fawcett, K. A. & Barroso, I. (2010), 'The genetics of obesity: Fto leads the way', *Trends in Genetics* **26**(6), 266–274.

Figlio, D., Guryan, J., Karbownik, K. & Roth, J. (2014), 'The Effects of Poor Neonatal Health on Children's Cognitive Development', *The American Economic Review* **104**(12), 3921–3955.

Finkelstein, E. A., Fiebelkorn, I. C. & Wang, G. (2004), 'State-level Estimates of Annual Medical Expenditures Attributable to Obesity', *Obesity Research* **12**(1), 18–24.

Finkelstein, E. A. & Zuckerman, L. (2010), *The Fattening of America: How the Economy Makes Us Fat, If It Matters, And What To Do About It*, John Wiley & Sons.

Frayling, T. M., Timpson, N. J., Weedon, M. N., Zeggini, E., Freathy, R. M., Lindgren, C. M., Perry, J. R., Elliott, K. S., Lango, H., Rayner, N. W. et al. (2007), 'A common variant in the fto gene is associated with body mass index and predisposes to childhood and adult obesity', *Science* **316**(5826), 889–894.

Freedman, D. S., Khan, L. K., Dietz, W. H., Srinivasan, S. R. & Berenson, G. S. (2001), 'Relationship of Childhood Obesity to Coronary Heart Disease Risk Factors in Adulthood: The Bogalusa Heart Study', *Pediatrics* **108**(3), 712–718.

Frisvold, D. E. & Giri, A. (2014), 'The Potential of Early Childhood Education as a Successful Obesity Intervention', *Obesity Interventions in Underserved Communities: Evidence and Directions* p. 25.

Godfrey, K. M. & Barker, D. J. (2001), 'Fetal Programming and Adult Health', *Public Health Nutrition* **4**(2b), 611–624.

Guo, G., Liu, H., Wang, L., Shen, H. & Hu, W. (2015), 'The genome-wide influence on human bmi depends on physical activity, life course, and historical period', *Demography* **52**(5), 1651–1670.

Halliday, T. J. (2008), 'Heterogeneity, State Dependence and Health', *The Econometrics Journal* **11**(3), 499–516.

Ham, J. C., Iorio, D. & Sovinsky, M. (2013), 'Caught in the Bulimic Trap? Persistence and State Dependence of Bulimia Among Young Women', *Journal of Human Resources* **48**(3), 736–767.

Hornstein, A. S. & Greene, W. H. (2012), 'Usage of an estimated coefficient as a dependent variable', *Economics Letters* **116**(3), 316–318.

Iughetti, L., De Simone, M., Verrotti, A., Iezzi, M. L., Predieri, B., Bruzzi, P., Bernasconi, S., Balli, F. & Bedogni, G. (2008), 'Thirty-year Persistence of Obesity after Presentation to a Pediatric Obesity Clinic', *Annals of Human Biology* **35**(4), 439–448.

Kleibergen, F. & Paap, R. (2006), 'Generalized Reduced Rank Tests Using the Singular Value Decomposition', *Journal of Econometrics* **133**(1), 97–126.

Krebs, N. F., Jacobson, M. S., of Pediatrics Committee on Nutrition, A. A. et al. (2003), 'Prevention of Pediatric Overweight and Obesity', *Pediatrics* **112**(2), 424.

Millimet, D. L. & Tchernis, R. (2013*a*), 'Anthropometric Mobility During Childhood', *Andrew Young School of Policy Studies Research Paper Series* (13-11).

Millimet, D. L. & Tchernis, R. (2013*b*), 'Estimation of Treatment Effects Without an Exclusion Restriction: With an Application to the Analysis of the School Breakfast Program', *Journal of Applied Econometrics* **28**(6), 982–1017.

Mocan, N. & Tekin, E. (2011), Obesity, Self-esteem, and Wages, *in* N. Grossman & N. Mocan, eds, 'Economic Aspects of Obesity', University of Chicago Press.

Nader, P. R., O'Brien, M., Houts, R., Bradley, R., Belsky, J., Crosnoe, R., Friedman, S., Mei, Z., Susman, E. J. et al. (2006), 'Identifying Risk for Obesity in Early Childhood', *Pediatrics* **118**(3), e594–e601.

Ogden, C. L., Carroll, M. D. & Flegal, K. M. (2008), 'High Body Mass Index for Age Among US Children and Adolescents, 2003-2006', *Journal of the American Medical Association* **299**(20), 2401–2405.

Ogden, C. L., Carroll, M. D., Kit, B. K. & Flegal, K. M. (2014), 'Prevalence of Childhood and Adult Obesity in the United States, 2011-2012', *Journal of American Medical Association* **311**(8), 806–814.

Ogden, C. L., Carroll, M. D., Lawman, H. G., Fryar, C. D., Kruszon-Moran, D., Kit, B. K. & Flegal, K. M. (2016), 'Trends in obesity prevalence among children and adolescents in the united states, 1988-1994 through 2013-2014', *Jama* **315**(21), 2292–2299.

Ogden, C. L., Flegal, K. M., Carroll, M. D. & Johnson, C. L. (2002), 'Prevalence and Trends in Overweight Among US Children and Adolescents, 1999-2000', *Journal of American Medical Association* **288**(14), 1728–1732.

Power, C., Lake, J. & Cole, T. J. (1997), 'Review: Measurement and Long-term Health Risks of Child and Adolescent Fatness', *International Journal of Obesity & Related Metabolic Disorders* **21**(7).

Ramachandrappa, S., Raimondo, A., Cali, A. M., Keogh, J. M., Henning, E., Saeed, S., Thompson, A., Garg, S., Bochukova, E. G., Brage, S. et al. (2013), 'Rare Variants in Single-minded 1 (SIM1) Are Associated with Severe Obesity', *The Journal of Clinical Investigation* **123**(7), 3042.

Rokholm, B., Silventoinen, K., Tynelius, P., Gamborg, M., Sørensen, T. I. & Rasmussen, F. (2011), 'Increasing genetic variance of body mass index during the swedish obesity epidemic', *PloS one* **6**(11), e27135.

Rosenquist, J. N., Lehrer, S. F., OMalley, A. J., Zaslavsky, A. M., Smoller, J. W. & Christakis, N. A. (2015), 'Cohort of birth modifies the association between fto genotype and bmi', *Proceedings of the National Academy of Sciences* **112**(2), 354–359.

Serdula, M. K., Ivery, D., Coates, R. J., Freedman, D. S., Williamson, D. F. & Byers, T. (1993), 'Do Obese Children Become Obese Adults? a Review of the Literature', *Preventive Medicine* **22**(2), 167–177.

Skinner, A. C., Perrin, E. M. & Skelton, J. A. (2016), 'Prevalence of Obesity and Severe Obesity in US Children, 1999-2014', *Obesity* **24**(5), 1116–1123.

Ulrick, S. W. (2008), 'Using Semi-parametric Methods in an Analysis of Earnings Mobility', *The Econometrics Journal* **11**(3), 478–498.

Van Cleave, J., Gortmaker, S. L. & Perrin, J. M. (2010), 'Dynamics of Obesity and Chronic Health Conditions Among Children and Youth', *Journal of American Medical Association* **303**(7), 623–630.

Walpole, S. C., Prieto-Merino, D., Edwards, P., Cleland, J., Stevens, G. & Roberts, I. (2012), 'The Weight of Nations: An Estimation of Adult Human Biomass', *BMC Public Health* **12**(1), 1.

Walter, S., Mejía-Guevara, I., Estrada, K., Liu, S. Y. & Glymour, M. M. (2016), 'Association of a genetic risk score with body mass index across different birth cohorts', *Jama* **316**(1), 63–69.

Whitaker, R. C., Wright, J. A., Pepe, M. S., Seidel, K. D. & Dietz, W. H. (1997), 'Predicting Obesity in Young Adulthood from Childhood and Parental Obesity', *New England Journal of Medicine* **337**(13), 869–873.

Appendix A. Simulation Details.

1. Case I. Own y_{i0}, own x_{it}, replace $\alpha_i = \overline{\widehat{\alpha}}$, set $\varepsilon_{it} = 0$. This eliminates heterogeneity due to time invariant and time-varying unobserved factors.

   (a) Compute

   $$\widehat{y}_{it} = \widehat{\gamma}\widehat{y}_{it-1} + x_{it}\widehat{\beta} + \overline{\widehat{\alpha}}, \qquad t = 1, ..., T$$

   where $\widehat{y}_{i0} = y_{i0}$.

   (b) Compute

   $$\Pr(y_{iT} > y^* | y_{i0} \geq y_0) = \frac{\sum_i I(\widehat{y}_{iT} > y^*) I(y_{i0} > y_0)}{\sum_i I(y_{i0} \geq y_0)}.$$

2. Case II. Own y_{i0}, own x_{it}, draw $\alpha_i \sim F(\alpha)$ where $F(\cdot)$ is the empirical distribution of α_i, set $\varepsilon_{it} = 0$. This allows for time invariant unobserved heterogeneity, but breaks the correlation between x and α.

   (a) Draw $\widetilde{\alpha}_i(r) \sim F(\widehat{\alpha})$, $r = 1, ..., R$, where R is the number of simulations.

   (b) Compute

   $$\widehat{y}_{it}(r) = \widehat{\gamma}\widehat{y}_{it-1}(r) + x_{it}\widehat{\beta} + \widetilde{\alpha}_i(r), \qquad t = 1, ..., T$$

   where $\widehat{y}_{i0}(r) = y_{i0}$ $\forall r$.

   (c) Compute

   $$\Pr(y_{iT} > y^* | y_{i0} \geq y_0) = \frac{1}{R} \sum\nolimits_r \left[\frac{\sum_i I(\widehat{y}_{iT}(r) > y^*) I(y_{i0} > y_0)}{\sum_i I(y_{i0} \geq y_0)} \right].$$

3. Case III. Own y_{i0}, own x_{it}, own w_i, set $\eta_i = 0$, set $\varepsilon_{it} = 0$. This eliminates heterogeneity due to time invariant and time-varying unobserved factors.

   (a) Compute

   $$\widehat{y}_{it}(r) = \widehat{\gamma}\widehat{y}_{it-1} + x_{it}\widehat{\beta} + w_i\widehat{\delta}, \qquad t = 1, ..., T$$

   where $\widehat{y}_{i0} = y_{i0}$.

   (b) Compute

   $$\Pr(y_{iT} > y^* | y_{i0} \geq y_0) = \frac{\sum_i I(\widehat{y}_{iT} > y^*) I(y_{i0} > y_0)}{\sum_i I(y_{i0} \geq y_0)}.$$

4. Case IV. Own y_{i0}, own x_{it}, draw $w_i \sim F(w)$ where $F(\cdot)$ is the empirical distribution of w_i, set $\eta_i = 0$, set $\varepsilon_{it} = 0$. This eliminates heterogeneity due to time invariant and time-varying unobserved factors and breaks the correlation between x and w.

   (a) Draw $\widetilde{w}_i(r) \sim F(w)$, $r = 1, ..., R$, where R is the number of simulations.

   (b) Compute

$$\widehat{y}_{it}(r) = \widehat{\gamma}\widehat{y}_{it-1}(r) + x_{it}\widehat{\beta} + \widetilde{w}_i(r)\widehat{\delta}, \qquad t = 1, ..., T$$

   where $\widehat{y}_{i0}(r) = y_{i0}$ $\forall r$.

   (c) Compute

$$\Pr(y_{iT} > y^* | y_{i0} \geq y_0) = \frac{1}{R}\sum\nolimits_r \left[\frac{\sum_i I(\widehat{y}_{iT}(r) > y^*) I(y_{i0} > y_0)}{\sum_i I(y_{i0} \geq y_0)}\right].$$

5. Case V. Own y_{i0}, own x_{it}, own w_i, draw $\eta_i \sim F(\eta)$ where $F(\cdot)$ is the empirical distribution of η_i, set $\varepsilon_{it} = 0$. This breaks the correlation between x, w and η and eliminates heterogeneity due to time-varying unobserved factors.

   (a) Draw $\widetilde{\eta}_i(r) \sim F(\widehat{\eta})$, $r = 1, ..., R$, where R is the number of simulations.

   (b) Compute

$$\widehat{y}_{it}(r) = \widehat{\gamma}\widehat{y}_{it-1}(r) + x_{it}\widehat{\beta} + w_i\widehat{\delta} + \widetilde{\eta}_i(r), \qquad t = 1, ..., T$$

   where $\widehat{y}_{i0}(r) = y_{i0}$ $\forall r$.

   (c) Compute

$$\Pr(y_{iT} > y^* | y_{i0} \geq y_0) = \frac{1}{R}\sum\nolimits_r \left[\frac{\sum_i I(\widehat{y}_{iT}(r) > y^*) I(y_{i0} > y_0)}{\sum_i I(y_{i0} \geq y_0)}\right].$$

6. Case VI. Own y_{i0}, replace $x_{it} = \overline{x}_t$, own α_i, set $\varepsilon_{it} = 0$. The eliminates heterogeneity due to time-varying factors.

   (a) Compute

$$\widehat{y}_{it} = \widehat{\gamma}\widehat{y}_{it-1} + \overline{x}_t\widehat{\beta} + \widehat{\alpha}_i, \qquad t = 1, ..., T$$

   where $\widehat{y}_{i0} = y_{i0}$.

(b) Compute

$$\Pr(y_{iT} > y^* | y_{i0} \geq y_0) = \frac{\sum_i I(\widehat{y}_{iT} > y^*) I(y_{i0} > y_0)}{\sum_i I(y_{i0} \geq y_0)}.$$

7. Case VII. Own y_{i0}, draw $x_{i\cdot} \sim F(x_1, ..., x_T)$ where $F(\cdot)$ is the empirical joint distribution of $x_1, ..., x_T$, own α_i, set $\varepsilon_{it} = 0$. This breaks the correlation between x and α and eliminates heterogeneity due to time-varying unobserved factors.

   (a) Draw $\widetilde{x}_{i\cdot}(r) \sim F(x_1, ..., x_T)$, $r = 1, ..., R$, where R is the number of simulations.

   (b) Compute

$$\widehat{y}_{it}(r) = \widehat{\gamma}\widehat{y}_{it-1}(r) + \widetilde{x}_{it}(r)\widehat{\beta} + \alpha_i, \qquad t = 1, ..., T$$

   where $\widehat{y}_{i0}(r) = y_{i0}$ $\forall r$.

   (c) Compute

$$\Pr(y_{iT} > y^* | y_{i0} \geq y_0) = \frac{1}{R} \sum\nolimits_r \left[\frac{\sum_i I(\widehat{y}_{iT}(r) > y^*) I(y_{i0} > y_0)}{\sum_i I(y_{i0} \geq y_0)} \right].$$

8. Case VIII. Own y_{i0}, own x_{it}, own α_i, draw $\varepsilon_{i\cdot} \sim F(\varepsilon_1, ..., \varepsilon_T)$ where $F(\cdot)$ is the empirical distribution of $\varepsilon_{i\cdot}$. This breaks the correlation between α and ε.

   (a) Draw $\widetilde{\varepsilon}_{i\cdot}(r) \sim F(\widehat{\varepsilon}_1, ..., \widehat{\varepsilon}_T)$, $r = 1, ..., R$, where R is the number of simulations.

   (b) Compute

$$\widehat{y}_{it}(r) = \widehat{\gamma}\widehat{y}_{it-1}(r) + x_{it}\widehat{\beta} + \widehat{\alpha}_i + \widetilde{\varepsilon}_{it}(r), \qquad t = 1, ..., T$$

   where $\widehat{y}_{i0}(r) = y_{i0}$ $\forall r$.

   (c) Compute

$$\Pr(y_{iT} > y^* | y_{i0} \geq y_0) = \frac{1}{R} \sum\nolimits_r \left[\frac{\sum_i I(\widehat{y}_{iT}(r) > y^*) I(y_{i0} > y_0)}{\sum_i I(y_{i0} \geq y_0)} \right].$$

9. Case IX. Own y_{i0}, own α_i, draw $x_{i\cdot}, \varepsilon_{i\cdot} \sim F(x_1, ..., x_T, \varepsilon_1, ..., \varepsilon_T)$ where $F(\cdot)$ is the empirical joint distribution of $x_1, ..., x_T, \varepsilon_1, ..., \varepsilon_T$. This breaks the correlation between α and x, ε.

   (a) Draw $\widetilde{x}_{i\cdot}(r), \widetilde{\varepsilon}_{i\cdot}(r) \sim F(x_1, ..., x_T, \widehat{\varepsilon}_1, ..., \widehat{\varepsilon}_T)$, $r = 1, ..., R$, where R is the number of simulations.

   (b) Compute

$$\widehat{y}_{it}(r) = \widehat{\gamma}\widehat{y}_{it-1}(r) + \widetilde{x}_{it}(r)\widehat{\beta} + \widehat{\alpha}_i + \widetilde{\varepsilon}_{it}(r), \qquad t = 1, ..., T$$

where $\widehat{y}_{i0}(r) = y_{i0}\ \forall r$.

(c) Compute

$$\Pr(y_{iT} > y^* | y_{i0} \geq y_0) = \frac{1}{R} \sum\nolimits_r \left[\frac{\sum_i I(\widehat{y}_{iT}(r) > y^*) I(y_{i0} > y_0)}{\sum_i I(y_{i0} \geq y_0)} \right].$$

Appendix B. Tables.

Table 1. Summary Statistics for ECLS-K Sample

Variable	Final Sample	Initial Sample
Panel I. Time varying and time invariant characteristics for wave 1		
Male (=1 if Male)	0.505	0.517
	(0.499)	(0.499)
White (=1 if White)	0.615	0.509
	(0.487)	(0.500)
Black (=1 if Black)	0.101	0.187
	(0.301)	(0.390)
Hispanic (=1 if Hispanic)	0.172	0.178
	(0.377)	(0.383)
Asian (=1 if Asian)	0.056	0.071
	(0.231)	(0.257)
Birthweight (ounces)	119.466	117.626
	(23.154)	(23.944)
Premature (=1 if more than 2 weeks premature)	0.155	0.156
	(0.362)	(0.363)
Northeast (=1 if live in Northeast)	0.190	0.181
	(0.392)	(0.385)
Midwest (=1 if live in Midwest)	0.282	0.222
	(0.450)	(0.415)
South (=1 if live in South)	0.314	0.347
	(0.464)	(0.476)
West (=1 if live in West)	0.215	0.251
	(0.411)	(0.433)
Urban	0.364	0.449
	(0.481)	(0.497)
Suburb	0.380	0.394
	(0.485)	(0.489)
Rural	0.255	0.157
	(0.436)	(0.363)
WIC recipient during pregnancy	0.321	0.409
	(0.467)	(0.492)
WIC recipient as infant/child	0.365	0.464
	(0.481)	(0.499)
AFDC recipient as infant/child	0.135	0.218
	(0.341)	(0.413)
Food Stamps recipient as infant/child	0.209	0.302
	(0.406)	(0.459)
Mother's AFB	24.434	23.001
	(5.119)	(4.828)
Mother's marital status at birth (=1 if married)	0.711	0.556
	(0.453)	(0.497)
Attended Full Day Kindergarten (=1 if attended)	0.499	0.536
	(0.5000)	(0.499)
Born in the USA	0.929	0.821
	(0.256)	(0.383)
Native English Speaker	0.839	.791
	(0.368)	(0.406)
Attended Non-parental Pre-kindergarten	0.799	0.738
	(0.400)	(0.439)
Mother worked during child's infancy	0.695	0.643
	(0.461)	(0.479)
Mother's education less than high school	0.112	0.147
	(0.315)	(0.354)
Mother's education is high school/GED	0.270	0.291
	(0.444)	(0.454)
Mother's education is some college	0.313	0.288
	(0.464)	(0.453)
Mother's education is 4-year college degree	0.172	0.124
	(0.377)	(0.330)
Mother's education is more than 4-year college	0.093	0.061
	(0.291)	(0.239)
Index of Socioeconomic status	0.098	-0.059
	(0.793)	(0.766)
Poverty (=1, if household in poverty)	0.156	0.200
	(0.363)	(0.400)
Number of children's books in household	79.106	68.409
	(57.007)	(53.194)
Household received AFDC	0.069	0.116
	(0.253)	(0.320)
Household received Food Stamps	0.120	0.185
	(.325)	(0.388)

Notes. See notes at the end of Table 1 on the next page.

Table 1 (Continued). Summary Statistics for ECLS-K Sample

Variable	Final Sample	Initial Sample
Household size	4.530	4.510
	(1.265)	(1.319)
Family Type (Two parents and siblings)	0.633	0.513
	(0.482)	(0.500)
Family Type (Two parents and no siblings)	0.085	0.080
	(0.280)	(0.272)
Family Type (One parent and siblings)	0.108	0.153
	(0.310)	(0.360)
Family Type (One parents and no siblings)	0.046	0.068
	(0.210)	(0.252)
Family Type (Other)	0.014	0.017
	(0.119)	(0.129)
Mother works full time	0.392	0.371
	(0.488)	(0.483)
Mother works part time	0.207	0.160
	(0.405)	(0.366)
Mother does not work	0.270	0.276
	(0.444)	(0.447)
No mother in household	0.027	0.033
	(0.161)	(0.180)
Child has health insurance	0.877	0.757
	(0.328)	(0.429)
Number of hours watched TV in school week	1.798	1.873
	(1.201)	(1.193)
Number of hours watched TV during weekend	2.422	2.500
	(1.463)	(1.491)
Household has rules regarding watching TV	0.843	0.739
	(0.364)	(0.439)
Number of days Household eats breakfast together per weekly	4.542	4.362
	(2.414)	(2.285)
Number of days Household eats dinner together per weekly	5.702	5.739
	(1.719)	(1.633)
Percent of minority students in class at school	35.922	43.931
	(32.301)	(32.787)
Neighborhood not safe	0.029	0.035
	(0.167)	(0.185)
Neighborhood somewhat safe	0.224	0.247
	(0.417)	(0.431)
Neighborhood very safe	0.700	0.565
	(0.458)	(0.496)
Household never worried about running out of food	0.840	0.710
	(0.366)	(0.453)
Panel II. Anthropometric Outcomes by Wave		
Weight z-scores, (wave 1)	0.264	0.261
	(1.094)	(1.059)
Weight z-scores, (wave 2)	0.393	
	(1.080)	
Weight z-scores, (wave 3)	0.558	
	(1.101)	
Weight z-scores, (wave 4)	0.609	
	(1.132)	
Weight z-scores, (wave 5)	0.673	
	(1.118)	
Height z-scores, (wave 1)	0.182	0.126
	(1.026)	(1.030)
Height z-scores, (wave 2)	0.076	
	(1.016)	
Height z-scores, (wave 3)	0.127	
	(1.012)	
Height z-scores, (wave 4)	0.296	
	(1.020)	
Height z-scores, (wave 5)	0.166	
	(1.077)	
BMI z-scores, (wave 1)	0.294	0.360
	(1.297)	(1.159)
BMI z-scores, (wave 2)	0.440	
	(1.103)	
BMI z-scores, (wave 3)	.613	
	(1.064)	
BMI z-scores, (wave 4)	0.669	
	(1.081)	
BMI z-scores, (wave 5)	0.600	
	(1.107)	
Number of Observations	9,150	11,050

Notes. Summary statistics (means and standard deviations in parenthesis) are based on the ECLS-K sample. While we use data from five waves of the ECLS-B, we report summary statistics for the first wave before and after our sample restrictions described in the text. In Panel I, the first column presents summary statistics for the final sample for wave 1 used in the regression analysis after restrictions while the second column presents those for the initial sample before any restrictions. Panel II presents the summary statistics for the anthropometric measures for all waves before and after sample restrictions. Note that all sample sizes are rounded to the nearest 50 per NCES restricted data regulations for the ECLS-K.

Table 2. Summary Statistics for ECLS-B Sample

Variable	Final Sample	Initial Sample
Panel I. Time varying and time invariant characteristics for wave 1		
Male (=1 if Male)	0.502	0.520
	(0.500)	(0.500)
White (=1 if White)	0.413	0.414
	(0.492)	(0.493)
Black (=1 if Black)	0.154	0.163
	(0.361)	(0.369)
Hispanic (=1 if Hispanic)	0.200	0.211
	(0.400)	(0.408)
Asian (=1 if Asian)	0.109	0.116
	(0.312)	(0.320)
Northeast (=1 if live in Northeast)	0.137	0.171
	(0.344)	(0.376)
Midwest (=1 if live in Midwest)	0.247	0.217
	(0.431)	(0.412)
South (=1 if live in South)	0.344	0.351
	(0.475)	(0.477)
West (=1 if live in West)	0.272	0.260
	(0.445)	(0.440)
Urban	0.714	0.740
	(0.452)	(0.440)
Suburban	0.130	0.115
	(0.336)	(0.320)
Rural	0.155	0.145
	(0.362)	(0.352)
Low Birthweight	0.252	0.303
	(0.434)	(0.459)
Normal Birthweight	0.683	0.635
	(0.465)	(0.481)
High Birthweight	0.065	0.062
	(0.246)	(0.241)
Premature (=1 if more than 2 weeks premature)	0.147	0.177
	(0.354)	(0.382)
Birth status (singleton)	0.807	0.854
	(0.394)	(0.3531)
Birth status (twin)	0.176	0.133
	(0.381)	(0.340)
Birth status (higher order)	0.007	0.008
	(0.081)	(0.088)
Mother's weight before pregnancy (in kilograms)	66.448	65.227
	(16.473)	(15.724)
Mother's weight gain during pregnancy	13.998	13.713
	(5.802)	(5.698)
WIC recipient during pregnancy	0.400	0.423
	(0.490)	(0.494)
WIC recipient as infant/child	0.397	0.413
	(0.489)	(0.492)
Mother's AFB	24.186	23.498
	(5.862)	(5.485)
Child's age (in months)	10.477	10.570
	(1.8740)	(1.884)
Food Stamps recipient as infant/child	0.204	0.227
	(0.403)	(0.419)
Index of Socioeconomic status	0.012	-0.118
	(0.865)	(0.852)
Poverty (=1, if household in poverty)	0.241	0.277
	(0.428)	(0.448)
Household size	4.478	4.434
	(1.485)	(1.564)
Child lives with biological mother	0.991	0.989
	(0.094)	(0.102)
Child lives with biological father	0.794	0.759
	(0.404)	(0.428)
Child does not live with father	0.192	0.227
	(0.394)	(0.419)
Mother's current marital status (=1 if married)	0.677	0.623
	(0.467)	(0.484)
Mother's education less than high school	0.173	0.210
	(0.378)	(0.458)
Mother's education is high school/GED	0.255	0.298
	(0.436)	(0.458)
Mother's education is some college	0.280	0.253
	(0.449)	(0.435)
Mother's education is 4-year college degree	0.172	0.144
	(0.378)	(0.351)
Mother's education is more than 4-year college	0.118	0.0921
	(0.323)	(0.289)

Notes. See notes at the end of Table 2 on the next page.

Table 2 (Continued). Summary Statistics for ECLS-B Sample

Variable	Final Sample	Initial Sample
Child's primary care with parents	0.491	0.510
	(0.499)	(0.500)
Child's primary care with relatives	0.261	0.272
	(0.439)	(0.445)
Child's primary care with non-relatives	0.159	0.130
	(0.366)	(0.336)
Child's primary care with care center	0.078	0.079
	(0.269)	(0.269)
Native English Speaker	0.798	0.775
	(0.401)	(0.417)
Mother's weight (in kilograms)	71.464	70.525
	(17.388)	(15.831)
Family Type (Two parents and siblings)	0.545	0.492
	(0.498)	(0.499)
Family Type (Two parents and no siblings)	0.257	0.275
	(0.437)	(0.447)
Family Type (One parent and siblings)	0.102	0.118
	(0.303)	(0.323)
Family Type (One parents and no siblings)	0.089	0.107
	(0.285)	(0.309)
Mother works full time	0.331	0.316
	(0.470)	(0.465)
Mother works part time	0.188	0.163
	(0.391)	(0.369)
Mother does not work	0.478	0.513
	(0.499)	(0.500)
No mother in household	0.001	0.002
	(0.033)	(0.041)
Household received Food Stamps	0.204	0.227
	(0.403)	(0.419)
Household received Welfare	0.087	0.102
	(0.282)	(0.303)
Child has health insurance	0.969	0.957
	(0.172)	(0.202)
Number of months on medicaid	3.372	3.669
	(4.671)	(4.834)
Child received WIC	0.520	0.558
	(0.500)	(0.497)
Household never worried about running out of food	0.559	0.589
	(0.496)	(0.492)
Panel II. Anthropometric Outcomes by Wave		
Weight z-scores, (wave 1)	-0.175	-0.263
	(1.474)	(1.601)
Weight z-scores, (wave 2)	-0.023	
	(1.369)	
Weight z-scores, (wave 3)	0.296	
	(1.120)	
Weight z-scores, (wave 4)	0.316	
	(1.119)	
Height (centimeters), (wave 1)	72.466	
	(4.130)	
Height (centimeters), (wave 2)	85.182	
	(3.676)	
Height (centimeters), (wave 3)	104.434	
	(5.365)	
Height (centimeters), (wave 4)	111.560	
	(5.367)	
Number of Observations	5,450	6,950

Notes. Summary statistics (means and standard deviations in parenthesis) are based on the ECLS-B sample. While we use data from four waves of the ECLS-B, we report summary statistics for the first wave before and after our sample restrictions described in the text. In Panel I, the first column presents summary statistics for the final sample for wave 1 used in the regression analysis after restrictions while the second column presents those for the initial sample before any restrictions. Panel II presents the summary statistics for the anthropometric measures for all waves before and after sample restrictions. Note that all sample sizes are rounded to the nearest 50 per NCES restricted data regulations for the ECLS-B.

Table 3. Dynamic Panel Data Estimation Results (ECLS-K)

	Model Specification		
	(1)	(2)	(3)
Panel I. Weight z–scores			
Lag Weight	0.931*	0.932*	0.775*
	(0.003)	(0.003)	(0.067)
Time-Varying Covariates	No	Yes	Yes
Time Invariant Covariates	No	Yes	No
Fixed Effects	No	No	Yes
Observations	27470	27470	27470
Underidentification	p = 0.000	p = 0.000	p = 0.000
Endogeneity	p = 0.000	p = 0.000	p = 0.000
First-Stage F-stat	88146.9	84006.9	269.0
Panel II. Height z–scores			
Lag Height	0.937*	0.936*	0.603*
	(0.004)	(0.004)	(0.048)
Time-Varying Covariates	No	Yes	Yes
Time Invariant Covariates	No	Yes	No
Fixed Effects	No	No	Yes
Observations	27470	27470	27470
Underidentification	p = 0.000	p = 0.000	p = 0.000
Endogeneity	p = 0.000	p = 0.000	p = 0.000
First-Stage F-stat	68631.4	64548.2	673.1
Panel III. BMI z–scores			
Lag BMI	0.912*	0.911*	0.217*
	(0.004)	(0.005)	(0.015)
Time-Varying Covariates	No	Yes	Yes
Time Invariant Covariates	No	Yes	No
Fixed Effects	No	No	Yes
Observations	27470	27470	27470
Underidentification	p = 0.000	p = 0.000	p = 0.000
Endogeneity	p = 0.000	p = 0.000	p = 0.000
First-Stage F-stat	8637.8	8518.0	820.8

Notes: ‡ $p < 0.10$, † $p < 0.05$, $*$ $p < 0.01$. Robust standard errors in parentheses. Estimation by GMM. Excluded instrument is the dependent variable twice-lagged. Sample sizes rounded to the nearest 10 per NCES restricted data regulations. Sample includes data from fall kindergarten, spring first, spring third, spring fifth grades, and spring eighth grade. See text for the list of covariates and further details.

Table 4. Dynamic Simulation Results (ECLS - K Sample)

	Weight z**-scores**			**Height** z**-scores**			**BMI** z**-scores**		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	P1	P2	P3	P1	P2	P3	P1	P2	P3
Benchmark	0.840	0.762	0.118	0.606	0.467	0.030	0.746	0.715	0.142
Panel I. Own Xs, $\varepsilon = 0$, and									
$\alpha = E[\alpha]$	0.753	0.134	0.000	0.000	0.000	0.000	0.000	0.000	0.000
$\alpha \sim f(\alpha)$	0.576	0.396	0.213	0.235	0.092	0.144	0.347	0.179	0.346
Panel II. Own Xs, $\eta = 0, \varepsilon = 0$, and									
$W = W_i$	0.727	0.246	0.006	0.006	0.000	0.000	0.070	0.000	0.044
$W \sim f(W)$	0.703	0.196	0.006	0.003	0.000	0.000	0.055	0.000	0.054
Panel III. Own Xs, own Ws, $\varepsilon = 0$, and									
$\eta \sim f(\eta)$	0.591	0.418	0.209	0.256	0.108	0.133	0.368	0.195	0.335
Panel IV. Own $\alpha, \varepsilon = 0$, and									
$X = E[X]$	0.844	0.780	0.116	0.669	0.523	0.019	0.797	0.792	0.105
$X \sim f(X)$	0.849	0.784	0.120	0.663	0.523	0.018	0.795	0.792	0.107
Panel V. Own Xs, own α, and									
$\varepsilon \sim f(\varepsilon)$	0.846	0.786	0.121	0.635	0.506	0.021	0.766	0.734	0.125
Panel VI. Own α and									
$X, \varepsilon \sim f(X, \varepsilon)$	0.843	0.779	0.124	0.635	0.509	0.022	0.765	0.735	0.125
Panel VII. Own Xs, Own Ws, Own α and									
$y_{i0} = E[y_{i0}]$	0.337	0.468	-	0.182	0.383	-	0.358	0.189	-
$y_{i0} \sim f(y_{i0})$	0.583	0.590	0.212	0.241	0.435	0.153	0.359	0.189	0.356

Notes: P1 denotes $\Pr(y_{iT} \geq 85^{\text{th}}$ percentile $|y_{i0} \geq 85^{\text{th}}$ percentile), P2 denotes $\Pr(y_{iT} \geq 95^{\text{th}}$ percentile $|y_{i0} \geq 95^{\text{th}}$ percentile), and P3 denotes $\Pr(y_{iT} \geq 85^{\text{th}}$ percentile $|y_{i0} \geq 50^{\text{th}}$ percentile). Benchmark case denotes the observed probability in the sample. Simulations (based on 500 draws) are obtained after estimating the dynamic model $y_{it} = \alpha_i + \gamma y_{it} - 1 + X_{it}\beta + \varepsilon_{it}$, where $\alpha_i = w_i\delta + \eta_i$. f() denotes the empirical distribution of the argument. Sample includes data from fall kindergarten, spring first, spring third, spring fifth grades, and spring eighth grade. See text for the list of covariates and further details. "-" indicates failed convergence.

Table 5. Dynamic Panel Data Estimation Results (ECLS-B)

	Model Specification		
	(1)	(2)	(3)
Panel I. Weight z–scores			
Lag Weight	0.873*	0.870*	0.124*
	(0.010)	(0.012)	(0.013)
Time-Varying Covariates	No	Yes	Yes
Time Invariant Covariates	No	Yes	No
Fixed Effects	No	No	Yes
Observations	10900	10900	10900
Underidentification	p = 0.000	p = 0.000	p = 0.000
Endogeneity	p = 0.000	p = 0.000	p = 0.000
First-Stage F-stat	112.0	1398.4	1929.3
Panel II. Height z–scores			
Lag Height	0.480*	0.506*	-0.002
	(0.004)	(0.010)	(0.007)
Time-Varying Covariates	No	Yes	Yes
Time Invariant Covariates	No	Yes	No
Fixed Effects	No	No	Yes
Observations	10900	10900	10900
Underidentification	p = 0.000	p = 0.000	p = 0.000
Endogeneity	p = 0.000	p = 0.000	p = 0.000
First-Stage F-stat	770.2	6940.2	10250.2

Notes: ‡ $p < 0.10$, † $p < 0.05$, $*$ $p < 0.01$. Robust standard errors in parentheses. Estimation by GMM. Excluded instrument is the dependent variable twice-lagged. Sample sizes rounded to the nearest 50 per NCES restricted data regulations. Sample includes data from waves 1-4 in the ECLS-B. See text for the list of covariates and further details.

Table 6. Dynamic Simulation Results (ECLS-B Sample)

	Weight z-Scores			**Height (cm)**		
	(1)	(2)	(3)	(4)	(5)	(6)
	P1	P2	P3	P1	P2	P3
Benchmark	0.541	0.366	0.431	0.600	0.365	0.179
Panel I. Own Xs, $\varepsilon = 0$, and						
$\alpha = E[\alpha]$	0.007	0.000	0.837	0.190	0.000	0.183
$\alpha \sim f(\alpha)$	0.255	0.118	0.617	0.380	0.092	0.383
Panel II. Own Xs, $\eta = 0, \varepsilon = 0$, and						
$W = W_i$	0.169	0.031	0.613	0.396	0.020	0.148
$W \sim f(W)$	0.101	0.007	0.719	0.292	0.007	0.296
Panel III. Own Xs, own Ws, $\varepsilon = 0$, and						
$\eta \sim f(\eta)$	0.332	0.166	0.556	0.444	0.136	0.297
Panel IV. Own $\alpha, \varepsilon = 0$, and						
$X = E[X]$	0.605	0.419	0.435	0.607	0.350	0.176
$X \sim f(X)$	0.596	0.416	0.440	0.597	0.353	0.204
Panel V. Own Xs, own α, and						
$\varepsilon \sim f(\varepsilon)$	0.581	0.405	0.431	0.597	0.304	0.158
Panel VI. Own α and						
$X, \varepsilon \sim f(X, \varepsilon)$	0.582	0.413	0.441	0.589	0.357	0.214
Panel VII. Own Xs, Own Ws, Own α and						
$y_{i0} = E[y_{i0}]$	0.240	0.115	-	0.396	0.123	-
$y_{i0} \sim f(y_{i0})$	0.240	0.117	0.612	0.404	0.127	0.386

Notes: P1 denotes $\Pr(y_{iT} \geq 85^{\text{th}}$ percentile $|y_{i0} \geq 85^{\text{th}}$ percentile), P2 denotes $\Pr(y_{iT} \geq 95^{\text{th}}$ percentile $|y_{i0} \geq 95^{\text{th}}$ percentile), and P3 denotes $\Pr(y_{iT} \geq 85^{\text{th}}$ percentile $|y_{i0} \leq 50^{\text{th}}$ percentile). Benchmark case denotes the observed probability in the sample. Simulations (based on 500 draws) are obtained after estimating the dynamic model $y_{it} = \alpha_i + \gamma y_{it-1} + X_{it}\beta + \varepsilon_{it}$, where $\alpha_i = w_i\delta + \eta_i$. f() denotes the empirical distribution of the argument. Sample includes data from waves 1-4 of the ECLS-B. See text for the list of covariates and further details. "-" indicates failed convergence.