Developmental Trajectories of Overweight During Childhood: Role of Early Life Factors

Chaoyang Li,‖ Michael I. Goran,† Harsohena Kaur,‡ Nicole Nollen,§ and Jasjit S. Ahluwalia¶*

Abstract
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Objective: Our goal was to identify developmental trajectories of overweight in children and to assess early life influences on these trajectories.
Research Methods and Procedures: Participants consisted of 1739 white, black, and Hispanic children who were younger than 2 years at the first survey and were followed up to 12 years of age. Repeated measures of overweight, defined as BMI ≥95th percentile, were used to identify overweight trajectories with a latent growth mixture modeling approach.
Results: Three distinct overweight trajectories were identified: 1) early onset overweight (10.9%), 2) late onset overweight (5.2%), and 3) never overweight (83.9%). After adjustment for multiple potential risk factors, male gender [odds ratio (OR), 1.5; 95% confidence interval (CI), 1.0 to 2.2], black ethnicity (OR, 1.7; 95% CI, 1.1 to 2.6), maternal 25 ≤ BMI <30 kg/m^2 (OR, 2.2; 95% CI, 1.3 to 3.7) or ≥30 kg/m^2 (OR, 5.1; 95% CI, 2.9 to 9.1), maternal weight gain during pregnancy ≥20.43 kg (OR, 1.7; 95% CI, 1.0 to 2.9), and birth weight ≥4000 g (OR, 2.0; 95% CI, 1.2 to 3.4) were associated with an increased risk of early onset overweight. These risk factors, except maternal weight gain, exerted similar effects on late onset overweight. In addition, maternal smoking (OR, 1.6; 95% CI, 0.8 to 3.1) and birth order ≥3 (OR, 2.3; 95% CI, 1.0 to 5.2) were associated with an increased risk of late onset overweight only. Breastfeeding ≥4 months was associated with a decreased risk of both early (OR, 0.7; 95% CI, 0.3 to 1.3) and late onset overweight (OR, 0.7; 95% CI, 0.3 to 1.7).
Discussion: Two trajectories of overweight and one never overweight group were identified. Early life predictors may have a significant influence on the developmental trajectories of overweight in children.

Key words: trajectory, birth weight, maternal obesity, gestational weight gain, breastfeeding

Introduction

Overweight (≥95th BMI percentile) in childhood and adolescence may track into adulthood. Previous studies suggest that being overweight in childhood significantly predicts adult obesity independently of parental obesity status (1,2). Moreover, individuals who were overweight during childhood have an elevated risk of cardiovascular disease, type 2 diabetes, and all-cause mortality both in childhood (3) and in adulthood (4). Recent population-based surveys in the United States have shown a marked increase in overweight and obesity prevalence in both children and adults in the past two decades (5,6).

The developmental trajectories of overweight during childhood and adolescence are not well understood. A recent study has shown that ~15% of adolescents 9 to 16 years of age are chronically obese, while 8% have an onset of obesity late in adolescence (7). Little is known about the developmental trajectories of overweight in early childhood. Such research is important because it will enhance our understanding of the mechanisms underlying the increasing trends in overweight prevalence and help to identify different etiologic pathways of overweight onset and development during childhood.

As suggested by the fetal origins hypothesis (later referred to as the developmental origins hypothesis) (8–10),

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*University of Kansas Medical Center, Kansas City, Kansas; †Department of Preventive Medicine, University of Southern California, Los Angeles, California; Departments of ‡Pediatrics and ¶Medicine and Cancer Center, University of Minnesota, Minneapolis, Minnesota; and §Department of Preventive Medicine and Public Health, University of Kansas School of Medicine, Kansas City, Kansas.
‖Current affiliation: Division of Adult and Community Health, Centers for Disease Control and Prevention, Atlanta, Georgia.
Address correspondence to Chaoyang Li, Centers for Disease Control and Prevention, 4770 Buford Highway, MS K66, Atlanta, GA 30341.
E-mail: cli@cdc.gov

early exposure to undernutrition or overnutrition in utero, feeding practices in infancy, and growth in early childhood may play a role in the development of obesity and obesity-related diseases in adult life (11–17). However, no previous studies have examined whether certain early life factors predispose children to alternate trajectories in the development of overweight.

To address these issues, we attempted to identify possible trajectories in the development of overweight and to assess the associations of early life factors with overweight trajectory membership. We used data from the National Longitudinal Survey of Youth 1979 (NLSY79)[1] Child and Young Adult file, a nationally representative longitudinal cohort study of children 2 to 12 years of age. In addition, we used innovative growth modeling procedures to identify subgroups of children who displayed different patterns or trajectories toward the development of overweight.

Research Methods and Procedures

Design and Sample

The NLSY79 is a nationally representative sample of 12,686 young men and women who were 14 to 22 years of age when first surveyed in 1979. Beginning in 1986, the children of NLSY79 women (n = 11,205, age range, 0–20 years) were surveyed, and supplemental information on their health status and physical and behavioral development was collected biennially through 2000 (18). Sampling weights were constructed for all children to adjust for differential non-response and oversampling of blacks, Hispanics, and economically disadvantaged non-black/non-Hispanic whites.

In the current study, eligible children included those who were younger than 2 years of age at the their first survey, were born in 1984, 1986, 1988, or 1990, were younger than 13 years of age during follow-up surveys, and had at least three measured height and weight values between 1986 and 2000 (n = 1912 children; 7579 repeated observations; Figure 1).

We excluded the following children in the final analyses: 1) children whose mothers who reported diabetes or high blood pressure during pregnancy (n = 7) and 2) children with a gestational age <28 weeks or with a birth weight <0.5 or ≥6 kg (n = 166). The final analytic sample (n = 1739 children; 6872 observations; 54.3% boys; 20.5% Hispanic, 30.4% black, and 49.2% white) was similar in the proportions of gender and race/ethnicity to the total eligible sample (54.9% boys; 20.5% Hispanic, 31.2% black, and 48.3% white).

[1] Nonstandard abbreviations: NLSY79, National Longitudinal Survey of Youth 1979; OR, odds ratio; GMM, growth mixture model; CI, confidence interval; GDM, gestational diabetes mellitus.

Procedures

Mother-child assessments were conducted at the same time as the main NLSY79 interview of each mother (18). Maternal characteristics, including mother's height, weight before pregnancy, weight before the delivery of the child, frequency of alcohol use and smoking during pregnancy, and mother's age at the birth of the child were assessed by self-report within 24 months after the birth of their children. Children's characteristics, including their birth weight, birth length, gestational age, birth order, and duration of breastfeeding (measured as the weeks of age after birth when breastfeeding ended) were reported by mothers within 24 months after the birth of their children. Other characteristics of the children, such as child's date of birth, age, sex, and race/ethnicity, and descriptors of the child's immediate family, such as mother's highest grade completed, as well as annual family net income at the birth of the child, were self-reported by the mothers at the first survey. Details of the various prenatal and postnatal measures have been reported elsewhere (19).

Definition of Childhood Overweight and Maternal Prepregnancy Overweight

Children's height and weight were measured in person by interviewers using a portable scale and tape measure (18). BMI was calculated based on measured weight (kg) and height (m) using the formula: BMI = weight (kg)/height $(m)^2$. BMI percentiles for age and sex were derived using the CDC growth charts (20). At-risk-of-overweight was defined as 85th ≤ BMI <95th percentile. Overweight was defined as a BMI ≥95th percentile. Maternal prepregnancy BMI was calculated based on self-reported height and weight just before pregnancy. Maternal overweight was defined as 25 ≤ BMI <30 kg/m^2, and maternal obesity was defined as BMI ≥30 kg/m^2 (21). Childhood overweight (1 = overweight, 0 = non-overweight) was the major outcome variable for this analysis. Maternal and child characteristics and socioeconomic status (mother's highest education level and family net income) were originally recorded in continuous scales and were re-categorized in the analyses of weighted percentages and calculation of odds ratios (ORs).

Statistical Analyses

Childhood overweight trajectory subclasses were identified by fitting the latent growth mixture models (GMMs) of the repeated measures of binary overweight data over age using the Mplus (version 3.12) program (22). The GMM is a new technique that examines the heterogeneity in developmental trajectories in the population (23). Conventional linear growth curve analysis estimates the change in outcome variables over time or age by assuming that all individuals in the sample belong to one homogeneous population with the same developmental trajectory, i.e., a single

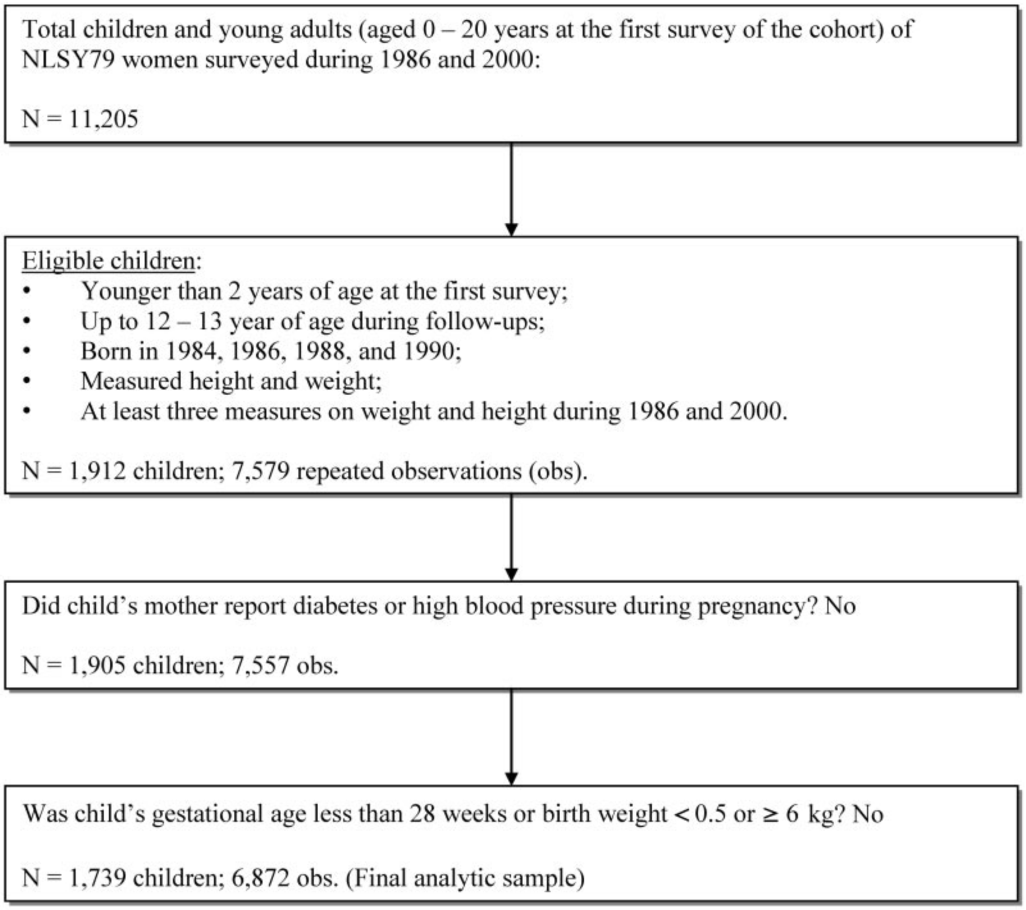

Figure 1: Flow chart of sample selection based on eligibility and exclusion criteria.

group (24). In contrast, the GMM extends the conventional growth model by assuming that individuals belong to two or more distinct populations with different developmental trajectories, i.e., two or more subgroups with different patterns of change over time. This assumption may be more realistic as we believe that children may have different pathways in the development of overweight. Thus, the GMM was selected as the most appropriate method for our data analyses.

The GMM was estimated by maximum likelihood using the expectation-maximization algorithm. Missing data were appropriately handled in the GMM with the assumption of missing at random (25). Logistic regression analysis was used for binary outcome variables. The categorical latent class variable captured the variations in continuous latent variables (intercept and slope). The number of latent classes was determined by the following model fit indices: Bayesian Information Criterion (26), probability of correct model (27), and Lo-Wendell-Rubin Likelihood Ratio Test (28). The model with the smallest Bayesian Information Criterion and largest probability of correct model indicates the specified model that best fits the data. A small *p* value (e.g., $p < 0.05$) for the Lo-Wendell-Rubin Likelihood Ratio Test indicates that a model with one less class ($c = k - 1$) has to be rejected in favor of a model with at least k classes. Each child in the sample was classified into a most probable trajectory class of overweight based on the posterior probability, which is the probability of each child belonging to each group (27). Therefore, all children in the sample were assigned to the group to which they had the highest posterior probability. Entropy, a weighted average of posterior probability, was used to measure the classification quality, with a value close to 1.0 or 0.0 indicating adequate classification.

Once the trajectory class membership was identified for each child, we conducted further analyses to examine the characteristics of children in each group and to determine the relative risk of being classified to high-risk groups in comparison to low-risk groups. First, we calculated weighted percentages and standard errors of categorical socio-demographic variables and early life predictors by overweight trajectory class. The overall differences in percentages were examined using χ^2 tests. Second, we calculated weighted means and standard errors of continuous socioeconomic and early life predictors. The overall differences in means were tested using Wald F-tests. Pairwise comparisons between each group were conducted using t tests with Bonferroni adjustment for p values. Finally, we conducted logistic regression analyses to calculate ORs and 95% confidence intervals (CIs) of trajectory class membership for socio-demographic and early life variables. To facilitate interpretation of the results, we created categorical scales for maternal prepregnancy BMI (1 = <25 kg/m^2, 2 = 25 to 29.9 kg/m^2, 3 = ≥30 kg/m^2), maternal weight gain (1 = <6.81 kg, 2 = 6.81 to 11.34 kg, 3 = 11.35 to15.88 kg, 4 = 15.89 to 20.42 kg, 5 = ≥20.43 kg), maternal age (1 = <25 years, 2 = 25 to 29 years, 3 = ≥30 years), birth weight (1 = <2500 g, 2 = 2500 to 3999 g, 3 = ≥4000 g), duration of breastfeeding (1 = never, 2 = 1–3 months, 3 = ≥4 months), mother's highest education level (1 = <high school, 2 = high school, 3 = some college, 4 = ≥college), and family net income (1 = <15,000 U.S. dollars, 2 = 15,000 to <25,000 U.S. dollars, 3 = 25,000 to <50,000 U.S. dollars, 4 = 50,000 to <75,000 U.S. dollars, 5 = ≥75,000 U.S. dollars) in logistic regression models.

All data management was conducted using SAS (version 9.12; SAS Institute, Inc., Cary, NC) (29). All weighted proportions, prevalence, means, and ORs were estimated using SUDAAN software (version 9.01; RTI International, Research Triangle Park, NC), a statistical package specifically designed to analyze data from complex surveys that incorporate multistage sampling and unequally weighted designs (30).

Results

Demographic, Maternal, and Early Life Characteristics of the Sample

The unweighted sample size and weighted percentages of the sample for each characteristic of children and mothers are shown in Table 1.

Prevalence of Childhood Overweight and Overweight by Age Groups

The overall weighted prevalence of at-risk-of-overweight (BMI ≥85th to 94.9th percentile) and overweight (BMI ≥95th percentile) are shown in Figure 2. The prevalence of at-risk-of-overweight and overweight at 2 years of age was 23.8% and 11.5%, respectively. The prevalence at age 4 and 6 years was lower than at age 2 years. After 6 years of age, the prevalence of both at-risk-of-overweight and overweight increased steadily with age and reached a peak at age 12.

Developmental Trajectories of Childhood Overweight

Using the latent growth mixture modeling approach, we estimated the developmental trajectories of overweight during childhood (age 2–12 years) as summarized in Table 2. Based on the smallest Bayesian Information Criterion (3946.3), largest probability of a correct model (≈1.0), sufficient entropy (≥ 0.90), and the Lo-Mendell-Rubin likelihood ratio test p value for three classes tested against four classes ($p > 0.05$), the model with three classes was determined as the best model for the developmental trajectories of overweight.

Based on the most likely latent class membership, the participants were classified in three classes ($n = 189$, 10.9% for the first class; $n = 91$, 5.2% for the second class; and $n = 1459$, 83.9% for the third class). The estimated probability of overweight for each of the three trajectory classes (Figure 3) suggests three distinct trajectories in the developmental progression of childhood overweight. The first trajectory represents children with an early onset of overweight that persisted throughout childhood. We characterized this trajectory as early-onset overweight (Early-onset). The second trajectory represents children who had a moderately high probability of overweight at age 2 years, low probability of overweight at age 4 and 6 years, but growing probability of overweight after age 8 years. This pattern was characterized as the late-onset overweight class (Late-onset). The third trajectory represents children who had a low probability of overweight throughout childhood and was characterized as the never overweight class (Normal).

Early Life Risk Factors and Overweight Trajectories During Childhood

We compared the weighted proportion of birth year, gender, race, and birth order in each overweight trajectory class using χ^2-tests and compared the weighted means of continuous early life predictors across the three overweight trajectory classes using Wald F-test as shown in Table 3. The proportion of black children was higher in the Late-onset (28.5%) and the Early-onset (24.8%) classes than in the Normal class (13.6%; $p < 0.001$). The proportion of first-born children was the highest in the Early-onset class (43.0%), whereas the proportion of second (42.0%) or third (39.2%) birth order children was highest in the Late-onset class ($p < 0.001$).

The mean maternal prepregnancy BMI was higher in the Late-onset and the Early-onset classes and exceeded the overweight level of BMI ≥25 kg/m^2 ($p < 0.0001$). The mean birth weights in the Early-onset class were heavier than in the Normal class ($p < 0.01$). The average duration of breastfeeding was shorter in the Late-onset and the Early-onset classes than that in the Normal class ($p < 0.001$). Mothers of children in the Late-onset class were slightly older and had a lower level of education ($p < 0.001$) and lower family net income compared with those in the Normal class.

Taking the Normal class as the referent group, the unadjusted ORs indicated the total effect of each predictor on the risk of being in the Early-onset or the Late-onset class, whereas the adjusted ORs indicated the direct or independent effect of each predictor after controlling for the possi-

Table 1. Sample and population-weighted characteristics of children and their mothers at baseline survey, NLSY79 child and young adults cohort (1986–2000), United States

Characteristic	Unweighted sample size (N = 1739) (N)	Population-weighted percentage (SE)
Sex		
Male	945	53.8 (1.4)
Female	794	46.2 (1.4)
Race/ethnicity		
Hispanic	356	6.9 (0.4)
Black	528	15.3 (0.7)
White	855	77.8 (0.8)
Birth order		
1	651	41.1 (1.4)
2	627	36.9 (1.4)
≥3	461	22.0 (1.1)
Gestational age (weeks)		
Pre-term (28 to 36)	179	9.3 (0.8)
Full term (≥37)	1560	90.7 (0.8)
Birth weight (grams)		
<2500	120	5.5 (0.6)
2500 to 3999	1422	81.6 (1.1)
≥4000	197	12.9 (1.0)
Breastfeeding (months)		
Never	873	44.7 (1.4)
1 to 3	487	32.6 (1.4)
≥4	291	22.7 (1.3)
Maternal prepregnancy BMI (kg/m^2)		
<25	1240	74.5 (1.2)
25 to 29.9	331	17.0 (1.1)
≥30	155	8.5 (0.8)
Maternal age at child birth (years)		
<25	801	40.9 (1.4)
25 to 29	819	50.5 (1.4)
≥30	119	8.6 (0.9)
Mother's highest education level (years)		
Less than high school (<12)	357	14.7 (0.9)
High school (12)	645	38.6 (1.4)
Some college (13 to 15)	481	28.8 (1.3)
College or above (≥16)	254	17.9 (1.1)
Family net income (U.S. $)		
<15,000	517	25.8 (1.2)
15,000 to 24,999	220	11.3 (0.9)
25,000 to 49,999	439	24.6 (1.2)
50,000 to 74,999	296	19.7 (1.2)
≥75,000	267	18.6 (1.2)

NLSY79, National Longitudinal Survey of Youth 1979; SE, standard error.

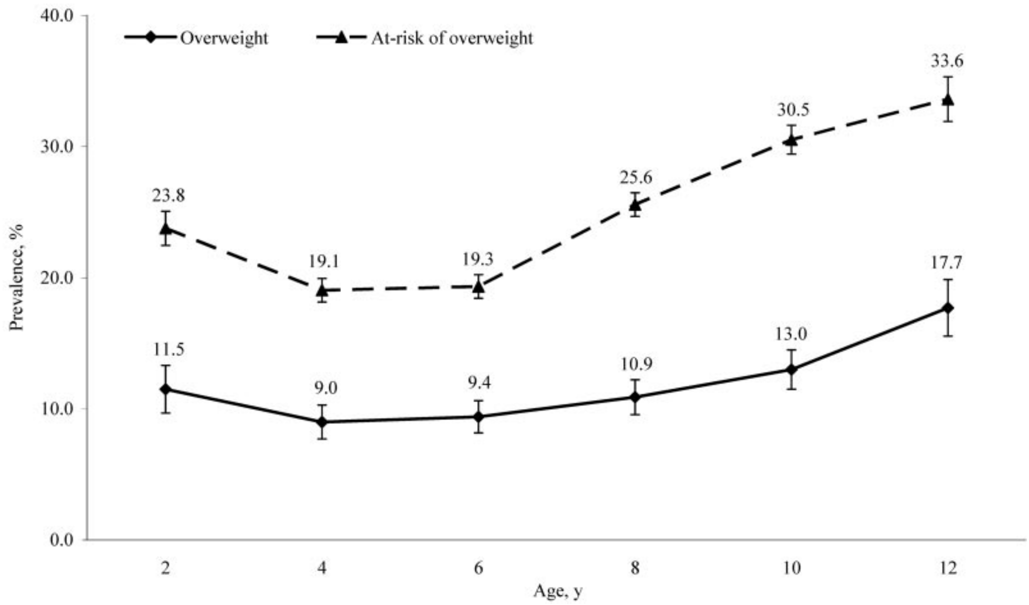

Figure 2: Weighted prevalence of overweight (BMI ≥ 95th percentile) and at-risk of overweight (BMI ≥ 85th percentile) over age.

ble confounding effects (Table 4). After adjusting for multiple potential risk factors, male gender (OR, 1.5; 95% CI, 1.0 to 2.2), black ethnicity (OR, 1.7; 95% CI, 1.1 to 2.6), maternal overweight (OR, 2.2; 95% CI, 1.3 to 3.7) or obesity (OR, 5.1; 95% CI, 2.9 to 9.1), maternal weight gain during pregnancy ≥20.43 kg (OR, 1.7; 95% CI, 1.0 to 2.9), maternal age ≥30 years (OR, 1.7; 95% CI, 0.7 to 4.0), and birth weight ≥4000 g (OR, 2.0; 95% CI, 1.2 to 3.4) were associated with an increased risk of early onset overweight (Early-onset). All of the above risk factors, except maternal weight gain, exerted similar effects on late onset overweight (Late-onset). In addition, maternal smoking (OR, 1.6; 95% CI, 0.8 to 3.1) and birth order ≥3 (OR, 2.3; 95% CI, 1.0 to 5.2) were associated with an increased risk of late onset overweight (Late-onset). Breastfeeding ≥4 months was associated with a decreased risk of both early (OR, 0.7; 95% CI, 0.3 to 1.3) and late onset overweight (OR, 0.7; 95% CI, 0.3 to 1.7).

Discussion

In this detailed longitudinal cohort study, we used advanced modeling techniques to identify three distinct subgroups of children who differed in their trajectory toward the development and progression of overweight status from the ages of 2 to 12 years. The largest group was characterized as never overweight (83.9%), where BMI percentile remained below the 95th percentile. For those children who developed overweight during childhood, two distinct subgroups emerged. One group of children (10.9%) had a high probability of being consistently overweight (termed early-onset overweight), and another group (5.2%) had a relatively high probability of being overweight initially, later lost weight, but then increased the probability of being overweight with increasing age (termed late-onset overweight). Both early- and late-onset subgroups appeared to be associated with black ethnicity, maternal prepregnancy

Table 2. Indices of goodness-of-fit of GMM

No. of latent classes	BIC	Probability of correct model	Entropy	LMR-LRT test Value (*df*)	LMR-LRT test *p*
1	5229.9	0.00	—	—	—
2	3959.6	1.7×10^{-6}	0.92	1300.2 (4)	<0.0001
3	3946.3	1.00	0.91	43.0 (4)	<0.0001
4	3969.6	7.6×10^{-11}	0.89	6.6 (4)	0.29

GMM, growth mixture model; BIC, Bayesian Information Criterion; LMR-LRT, Lo-Mendell-Rubin Likelihood Ratio.

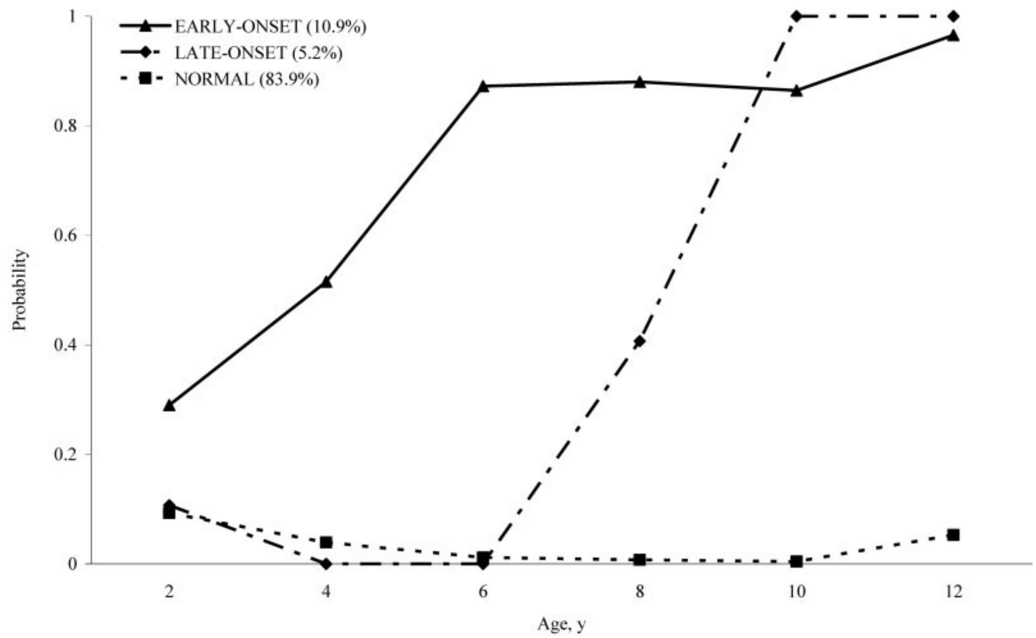

Figure 3: Weighted probabilities of childhood overweight (BMI ≥ 95th percentile) of 3 trajectory groups classified by the most likely class memberships.

overweight or obesity, maternal age ≥30 years, birth weight ≥4000 g, and lack of breastfeeding. Interestingly, maternal smoking during pregnancy and birth order ≥3 were specifically associated with the late onset overweight trajectory only.

To our knowledge, our analysis is the first to identify individual patterns of overweight trajectories during early childhood. Similar to a recent study conducted in children and adolescents 9 to 16 years of age (7), one subgroup of children with early-onset overweight was ascertained in our study. This group of children represents ~10% of the U.S. child population. Identification of children with early-onset overweight has important clinical and public health implications because these children possibly form the persistent layer of prevalent cases of overweight, and their trajectory suggests a high risk of their overweight persisting into adulthood (1).

According to the fetal origins hypothesis (8), which was later termed the developmental origins hypothesis (10), fetal and early life factors have influences on the development of overweight. There have been few longitudinal studies to test or provide direct support for this hypothesis in the research on childhood overweight. Our study sheds new light on the role of early life risk factors on the development of overweight in early childhood. We found that, of the 12 early life variables examined, male gender, black ethnicity, maternal prepregnancy overweight (25 kg/m^2 ≤ BMI <30 kg/m^2) or obesity (BMI ≥30 kg/m^2), first-born children, high birth weight (≥4000 g), mothers with high school education, and lower middle-class income were significantly associated with higher risk of early-onset overweight. In previous studies, high birth weight and excessive gestational weight gain were associated with increased risk of obesity in later life (31). Our research adds further evidence that overnutrition in utero may influence the development of early-onset overweight during childhood. Although the mechanism of influence is not well understood, it has been proposed that maternal obesity, gestational diabetes mellitus (GDM), and excessive weight gain in pregnancy may cause excess fatness in the fetus and newborn infant through an increased transfer of maternal fat fuels to the fetus (31–33). Future studies are needed to further examine and discern these mechanisms.

Numerous studies have shown that restrained growth in utero has been associated with increased risk of diabetes, metabolic syndrome, and cardiovascular disease in adult life (8). However, the effect of restrained growth in utero (e.g., low birth weight) on the development of obesity has been inconsistent. The majority of previous research has shown that low birth weight is associated only with central obesity or visceral fatness (9,34), and this effect is more pronounced among black children (13). The present study showed that maternal age >30 years at birth of child was associated with a 3-fold increased risk of late onset overweight. Higher birth order, independently of maternal age and birth weight, was associated with a >2-fold increased risk of late-onset overweight. Our finding is in contrast to that of a previous study, in which first-born status was associated with overweight in young black adults (35). The mechanism of the effect of advanced maternal age and parity is unknown. Very limited

Table 3. Weighted proportion of early life predictors by trajectory classes of overweight (BMI ≥ 95th percentile; 3-class GMM) ($N = 1739$)

Predictor	Trajectory classes of overweight: Normal	Early-onset	Late-onset	Significance tests	*p*
N (%)	1459 (83.9%)	189 (10.9%)	91 (5.2%)		
Categorical variables (% ± SE)				**χ^2 value (df)**	
Birth year (%)					
1984	20.3 ± 1.2	20.1 ± 3.5	16.6 ± 4.8	8.5 (6)	0.21
1986	30.9 ± 1.4	31.9 ± 4.1	23.4 ± 5.1		
1988	24.1 ± 1.3	17.0 ± 3.2	23.1 ± 5.4		
1990	24.7 ± 1.4	30.9 ± 4.1	36.8 ± 6.2		
Sex (%)					
Male	52.4 ± 1.6	62.7 ± 4.2	63.2 ± 6.0	7.3 (2)	0.03
Race (%)					
Hispanic	6.9 ± 0.5	6.9 ± 1.3	7.0 ± 1.9	24.1 (4)	<0.001
Black	13.6 ± 0.7	24.8 ± 3.0	28.5 ± 4.9		
White	79.5 ± 0.9	68.3 ± 3.4	64.6 ± 5.4		
Birth order (%)					
1	42.1 ± 1.5	43.0 ± 4.4	18.8 ± 4.9	20.5 (4)	0.001
2	37.3 ± 1.5	30.3 ± 4.0	42.0 ± 6.3		
3+	20.5 ± 1.2	26.7 ± 3.9	39.2 ± 6.3		
Maternal smoking during pregnancy (yes) (%)	30.7 ± 1.5	27.6 ± 4.0	40.6 ± 6.2	3.1 (2)	0.21
Maternal alcohol use during pregnancy (yes) (%)	52.4 ± 1.6	41.9 ± 4.4	44.4 ± 1.6	6.2 (2)	0.05
Continuous variables (mean ± SE)				**Wald F (df)**	
Maternal prepregnancy BMI (kg/m^2)	22.5 ± 0.1[a]	26.3 ± 0.6[b]	25.9 ± 0.6[b]	30.2 (2)	<0.001
Maternal weight gain (kg)	14.3 ± 0.2	15.0 ± 0.7	14.4 ± 0.6	0.5 (2)	0.61
Mother's age at birth of child (years)	25.1 ± 0.1[c]	25.2 ± 0.3	26.1 ± 0.4[d]	2.8 (2)	0.06
Gestational age (weeks)	38.8 ± 0.1	38.7 ± 0.2	38.8 ± 0.2	0.3 (2)	0.78
Birth weight (kg)	3.3 ± 0.02[a]	3.5 ± 0.05[b]	3.4 ± 0.08	4.6 (2)	0.01
Breast feeding (weeks)	11.9 ± 0.6[a]	8.1 ± 1.8[b]	7.4 ± 2.3[b]	8.6 (2)	<0.001
Mother's highest education level (years)	13.2 ± 0.1[a]	12.6 ± 0.2[b]	12.3 ± 0.3[b]	9.2 (2)	<0.001
Family net income (U.S. $)	60,118 ± 3317[a]	51,516 ± 4019	43,315 ± 3734[b]	5.7 (2)	0.003

GMM, growth mixture model; SE, standard error.
[a,b] Statistically significant at $\alpha = 0.01$ level; [c,d] statistically significant at $\alpha = 0.05$ level.

evidence suggests that, among older mothers, there may be some resistance to the anabolic effects of growth hormone and placental lactogen made by the placenta, which allows glucose to be transferred to the fetus (36) .

Maternal smoking during pregnancy has been related to childhood overweight (37), particularly among children 7 years of age or older (38,39). Our finding that maternal smoking had a stronger effect on late-onset overweight than

Table 4. Odds ratios of demographic, early life, and socioeconomic predictors for the trajectory classes of childhood overweight, NLSY79 Child and Young Adult cohort (1986–2000)

Characteristic	Early-onset		Late-onset	
	Unadjusted OR (95% CI)	Adjusted OR (95% CI)	Unadjusted OR (95% CI)	Adjusted OR (95% CI)
Sex				
Female	1.0	1.0	1.0	1.0
Male	1.5 (1.1, 2.2)	1.5 (1.0, 2.2)	1.6 (0.9, 2.6)	1.4 (0.8, 2.5)
Race				
White	1.0	1.0	1.0	1.0
Hispanic	1.2 (0.7, 1.9)	1.1 (0.6, 1.8)	1.3 (0.7, 2.4)	1.1 (0.5, 2.5)
Black	2.1 (1.5, 3.0)	1.7 (1.1, 2.6)	2.6 (1.5, 4.3)	2.1 (1.1, 4.2)
Maternal prepregnancy BMI (kg/m^2)				
<25	1.0	1.0	1.0	1.0
25 to 29.99	2.5 (1.6, 3.8)	2.2 (1.3, 3.7)	2.7 (1.4, 4.9)	2.4 (1.3, 4.7)
≥30	4.9 (3.0, 8.2)	5.1 (2.9, 9.1)	5.6 (2.8, 11.1)	5.8 (2.6, 13.0)
Maternal weight gains during pregnancy [kg (lb)]				
<6.81 (15)	2.6 (1.4, 5.0)	1.2 (0.6, 2.7)	1.1 (0.5, 2.8)	0.5 (0.2, 1.4)
6.81 (15) to 11.34	0.8 (0.5, 1.5)	0.6 (0.3, 1.1)	0.7 (0.3, 1.5)	0.5 (0.2, 1.3)
11.35 (25) to 15.88	1.0	1.0	1.0	1.0
15.89 (35) to 20.42	1.4 (0.8, 2.3)	1.3 (0.8, 2.2)	1.3 (0.7, 2.5)	1.4 (0.7, 2.8)
20.43 (45) +	1.9 (1.1, 3.2)	1.7 (1.0, 2.9)	0.8 (0.3, 1.7)	0.8 (0.3, 2.1)
Maternal smoking				
No	1.0	1.0	1.0	1.0
Yes	0.9 (0.6, 1.3)	1.0 (0.6, 1.6)	1.6 (0.9, 2.6)	1.6 (0.8, 3.1)
Maternal alcohol use				
No	1.0	1.0	1.0	1.0
Yes	0.7 (0.5, 0.9)	0.7 (0.5, 1.1)	0.7 (0.4, 1.2)	0.8 (0.5, 1.5)
Maternal age (years)				
<25	1.0	1.0	1.0	1.0
25 to 29	1.1 (0.7, 1.5)	1.3 (0.8, 2.0)	1.2 (0.7, 2.1)	1.2 (0.7, 2.3)
≥30	1.2 (0.6, 2.5)	1.7 (0.7, 4.0)	2.4 (1.0, 5.6)	2.7 (1.0, 7.2)
Birth order				
1	1.0	1.0	1.0	1.0
2	0.8 (0.5, 1.2)	0.6 (0.4, 1.0)	2.5 (1.3, 5.1)	1.7 (0.8, 3.4)
≥3	1.3 (0.8, 2.0)	0.8 (0.5, 1.3)	4.3 (2.1, 8.8)	2.3 (1.0, 5.2)
Birth weight (grams)				
<2500	0.9 (0.4, 2.2)	0.8 (0.3, 2.2)	1.0 (0.4, 2.5)	0.9 (0.3, 2.4)
2500 to 3999	1.0	1.0	1.0	1.0
≥4000	2.3 (1.4, 3.7)	2.0 (1.2, 3.4)	2.0 (1.0, 3.9)	1.6 (0.8, 3.5)
Breastfeeding (months)				
Never	1.0	1.0	1.0	1.0
1 to 3	0.9 (0.6, 1.3)	1.1 (0.7, 1.7)	0.4 (0.2, 0.8)	0.6 (0.3, 1.1)
4 or more	0.5 (0.3, 0.8)	0.7 (0.3, 1.3)	0.5 (0.2, 0.9)	0.7 (0.3, 1.7)

Table 4. Continued

Characteristic	Early-onset Unadjusted OR (95% CI)	Early-onset Adjusted OR (95% CI)	Late-onset Unadjusted OR (95% CI)	Late-onset Adjusted OR (95% CI)
Mother's education level				
Less than high school	1.6 (0.8, 3.3)	1.2 (0.5, 2.9)	3.4 (1.3, 8.8)	1.5 (0.5, 4.5)
High school	2.4 (1.3, 4.5)	2.0 (1.0, 4.2)	2.4 (1.0, 5.9)	1.5 (0.6, 4.0)
Some college	1.9 (1.0, 3.6)	1.6 (0.8, 3.4)	1.3 (0.5, 3.6)	1.0 (0.3, 2.9)
College or above	1.0	1.0	1.0	1.0
Family net income (U.S. $)				
<15,000	2.4 (1.3, 4.7)	1.8 (0.9, 3.9)	1.4 (0.6, 3.2)	1.1 (0.4, 2.8)
15,000 to 24,999	1.3 (0.5, 3.0)	0.9 (0.4, 2.2)	2.8 (1.1, 6.8)	2.1 (0.7, 6.6)
25,000 to 49,999	2.6 (1.4, 5.2)	1.9 (0.9, 3.8)	1.2 (0.5, 2.9)	1.1 (0.4, 2.7)
50,000 to 74,999	2.9 (1.5, 5.8)	2.0 (1.0, 4.1)	1.6 (0.7, 4.0)	1.5 (0.6, 3.8)
≥75,000	1.0	1.0	1.0	1.0

NLSY79, National Longitudinal Survey of Youth 1979; OR, odds ratio; CI, confidence interval. Normal trajectory class was set as the reference group. OR and 95% CI were adjusted for sex; race; birth order; gestational age; birth weight; breastfeeding; maternal prepregnancy BMI; maternal weight gains, smoking and alcohol use during pregnancy; maternal age at the birth of the child; mother's education level; and family net income.

on early-onset overweight adds further support to these observations. The underlying mechanism of the long-term effects of maternal smoking on childhood overweight is unknown. Potentially, maternal smoking may lead to fetal growth retardation due to poor appetite or nutrition induced by nicotine (40). Infants with low birth weight due to restrained growth in utero seem to gain weight more rapidly during the postnatal period than those with normal birth weight (41).

Breastfeeding has a small but consistent protective effect against the development of overweight in children (42). There is also an additive interaction between breastfeeding and maternal prepregnancy BMI on the risk of childhood overweight (15). Consistent with previous findings, the present study showed that breastfeeding was protective against both early-onset and late-onset overweight. However, after adjusting for potential confounders, this effect diminished, suggesting that the protective effect of breastfeeding is either small or may be partially explained by a combination of other early life factors.

Our findings have public health and clinical implications. First, the identification of the developmental trajectories of overweight is helpful to target high-risk groups and tailor interventions for children who are at a higher risk. Second, the assessment of early life factors in relation to different overweight trajectories may enhance our understanding of etiologic or causal pathways of overweight occurrence and development. Finally, assessment and identification of potentially modifiable risk factors may help make effective strategies for early prevention, intervention, or treatment of childhood overweight in public health services and clinical practice.

Our study is strengthened by the use of a nationally representative long-term cohort of white, black, and Hispanic children in the United States. Therefore, our findings may be generalizable to the population of U.S. children 2 to 12 years of age. Second, we used repeated measures of carefully measured weight and height for the same subjects to define the status of overweight over time. Our results are more reliable than those using self-reported weight and height. Third, the assessment of maternal weight status and behavioral risk factors before and during pregnancy and child characteristics at birth and infancy was comprehensive, which enabled us to examine the associations among these factors and the developmental trajectories of overweight during childhood. Lastly, we used state-of-the-art statistical methods to appropriately analyze the longitudinal data and yield reliable results.

There are some limitations in the present study. First, maternal weight and height before and after pregnancy, weight gain during pregnancy, gestational age, birth weight, and other prenatal and postnatal events were based on maternal self-report. Studies have shown that maternally reported neonatal information is sufficiently accurate for clinical and epidemiological research, especially when the information was obtained during the infancy of children (43). Second, our study lacked genetic biomarkers, making it difficult to differentiate the effects of early life environmental influences from genetic predisposition on overweight trajectories. Third, information on diabetes during pregnancy for the mothers was available only in the most recent surveys (i.e., in 1998 and 2000) in the NLSY data. Particularly, lack of information on GDM in the data prevented us from examining the effect of GDM on the overweight trajectories. Previous studies have found that offspring who were exposed to maternal GDM in utero had nearly 3-fold increased risk of being overweight at age 14 to 17 years (44) and had a higher BMI after age 9 years (45) than those unexposed to this condition. In particular, children of mothers with GDM had an apparent increase in BMI at birth and, more dramatically, after the age of 4 years, but not at the age of 1 to 3 years (46). This pattern was similar to the Late-onset group found in our study, suggesting that maternal GDM may exert a long-term influence in the development of overweight in adolescence. Nevertheless, the effects of GDM on childhood overweight appeared to be partially mediated through birth weight and maternal BMI (47), which were identified as two of the major risk factors of late-onset overweight in the present study.

In conclusion, to our knowledge, our analysis identifies, for the first time in a nationally representative cohort of children, two developmental trajectories of overweight and a normative group during childhood. While both early-onset and late-onset overweight trajectories may share some genetic predispositions, there may be distinct etiologic pathways for the development of overweight in children. Future research on the impact of the different overweight trajectories on long-term disease outcomes, such as diabetes, metabolic syndrome, and cardiovascular disease in later life, is warranted.

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