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Dynamics of Early Childhood Overweight

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Abstract

Objective—To study the dynamic processes that drive development of childhood overweight by examining the effects of prenatal characteristics and early-life feeding (breastfeeding versus bottle feeding) on weight states through age 7 years. We test a model to determine whether prenatal characteristics and early-life feeding influence the development of a persistent early tendency toward overweight and/or whether prenatal characteristics and early-life feeding factors influence the likelihood that children will change weight states as they get older.

Methods—Data from the National Longitudinal Survey of Youth's Child-Mother file were used to implement these analyses. A total of 3022 children were included in this sample. For inclusion in this sample, valid information on height and weight during 3 consecutive interviews when the child was aged 24 to 95 months as well as valid data on prenatal and birth characteristics were needed. The primary outcome measure was childhood overweight (BMI > 95th percentile). Multivariate logistic models and first-order Markov models were estimated.

Results—Early development of childhood overweight was associated with race, ethnicity, maternal prepregnancy obesity, maternal smoking during pregnancy, and later birth years. In later years, the factor that contributed the most to being overweight was having been overweight in the previous observation period. However, with conditioning on the child's having been overweight in the previous observation period, the prenatal factors that contributed to early childhood overweight, except for birth cohort, were also associated with development of overweight among children who had previously been normal weight and perpetuated the persistence of overweight over time.

Conclusions—This research suggests that prenatal characteristics, particularly race, ethnicity, maternal smoking during pregnancy, and maternal prepregnancy obesity, exert influence on the child's weight states through an early tendency toward overweight, which then is perpetuated as the child ages. These findings are intriguing as they provide additional clues to the genesis of childhood overweight and suggest that overweight prevention may need to begin before pregnancy and in early childhood.

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The Social Life of Paper (Part 2)

“Why do we pile documents instead of filing them? Because piles represent the process of active, ongoing thinking. The psychologist Alison Kidd argues that ‘knowledge workers’ use the physical space of the desktop to hold ‘ideas which they cannot yet categorize or even decide how they might use.’ The messy desk is not necessarily a sign of disorganization. It may be a sign of complexity: those who deal with many unresolved ideas simultaneously cannot sort and file the papers on their desks, because they haven’t yet sorted and filed the ideas in their head. Kidd writes that many of the people she talked to use the papers on their desks as contextual cures to ‘recover a complex set of threads without difficulty and delay’ when they come in on a Monday morning, or after their work has been interrupted by a phone call. What we see when we look at the piles on our desks is, in a sense, the contents of our brains.”

The New Yorker. March 25, 2002

Noted by JFL, MD

During the past decade, life-course studies have provided significant evidence for a link between prenatal and early-life exposures and later development of a range of chronic diseases.¹⁻³ The life-course approach conceptualizes health development as a lifelong dynamic process in which genetic, biological, social, and environmental factors interact to produce health states.⁴⁻⁷ The life-course approach is well suited for the study of childhood overweight because it is widely acknowledged that the development of childhood overweight is a multifactor disorder involving genetic, biological, social, and environmental pathways.⁸ Prenatal characteristics and early-life feeding factors, including maternal weight before and during pregnancy, smoking during pregnancy, infant feeding, and birth weight, have been associated with overweight during childhood.⁹⁻¹²

Maternal nutrition is widely recognized as critical to the developing fetus.¹³⁻¹⁶ The availability of macronutrients and micronutrients to the fetus is associated with the mother's prepregnancy nutritional status, as well as her food intake during pregnancy.^{10,17-19} Evidence suggests that the development of obesity in adolescents and adults may be related to nutritional programming that occurs very early in life.^{19,20} For example, maternal undernutrition during the Dutch Famine of 1944 and 1945 has been associated with adult obesity, although the timing and the mechanisms are not clear.^{2,21} Male children of mothers who were exposed to severe malnutrition during the first trimester were at greater risk for obesity at 18 years but not at 50 years. Female children who were exposed to severe malnutrition during the first trimester were at greater risk for obesity at age 50.²² In addition, children of mothers with diabetes have been the subject of numerous studies linking the intrauterine environment with fetal growth and development.^{14,19,23-27} These studies suggest that the altered glucose-insulin metabolism of the mother may affect a lifelong change in insulin production and sensitivity in her offspring, increasing the likelihood that these offspring will become overweight and develop type 2 diabetes later in life.^{14,24,28,29} Furthermore, recent studies using animal models have suggested a hyperinsulinemia effect with maternal diets high in carbohydrates and overnutrition.^{30,31} Contemporaneous maternal overweight or obesity is consistently related to childhood overweight,³²⁻³⁸ but to our knowledge, only 1 study linked maternal weight during early pregnancy to child overweight among preschoolers.¹¹

Other exposures in utero may also have a lasting effect on overweight development. Several studies have documented an association between maternal smoking during pregnancy and childhood overweight.^{11,39-45} Fetal physiologic changes in response to maternal smoking during pregnancy have been hypothesized. These include maternal poor nutrition influenced by a suppression of appetite by nicotine, a nicotine-induced vasoconstriction that compromises the uteroplacental vasculature, and an increased exposure to carbon monoxide that decreases the fetal uploading of oxygen, affecting fetal growth and development.⁴⁶ Impaired fetal growth has been strongly associated with smoking during pregnancy, and a dose-dependent relationship has been found between serum cotinine and restricted growth.⁴⁷

Birth weight is thought to be a crude measure of these exposures in utero. The attained birth weight is a result of the interactive effects of length of gestation, rate of fetal growth, adequacy of the intrauterine environment, uteroplacental function, and genetic potential.^{29,46,48} There has been a positive relationship between birth weight and later weight outcomes in most studies,^{11,35,36,38,49-51} with a reported U- or J-shaped relationship noted.⁵²

After birth, influences within the early-life period are also linked to later overweight. In particular, the method of infant feeding, breast- or bottle-feeding, has been studied with mixed results.⁵³⁻⁵⁶ Some studies have found a dose-response effect, with an increased protective effect associated with increased duration of breastfeeding.^{35,53,57,58} One of the mechanisms posited for a protective effect of breastfeeding is that breastfeeding may permit the infant to establish self-regulation in feeding that is not available to the bottle-fed infant.⁵⁵

Although these studies have tested for an effect between prenatal characteristics and early-life feeding on overweight development at various ages, they have not tracked the dynamics of the process of overweight development. The purpose of this article is to develop and test a model of the extent, timing, and persistence of prenatal characteristics and early-life feeding on the development of childhood overweight across time.

To study the dynamic effects, we propose a model linking prenatal characteristics and early feeding with overweight development in children from birth to 7 years of age. We chose this period because we wanted to examine prenatal characteristics and early feeding practices on overweight among young children but wanted to extend the observation period beyond the period of adiposity rebound. Although childhood overweight is a probabilistic process, a child's weight status displays persistence over time. In other words, the probability that a child is observed to be overweight across any 2 successive observation periods is a function of whether the child was overweight in the previous observation period. It therefore is natural to conceptualize the dynamic process of childhood overweight as a first-order Markov process.⁵⁹ Figure 1 presents our model for a child who is observed at 4 points during their life cycle. The initial conditions, observed in period T_0 , capture the maternal factors of prepregnancy weight, maternal smoking during pregnancy, race, ethnicity, and the early-life influence of feeding method. These factors are primarily either biological, such as intrauterine exposure to nicotine, or social, such as race and access to material resources. Some ways in which social factors influence biological processes are through the quality of diet and lower immunity brought on by exposure to stress.⁶⁰

We hypothesize that the initial conditions can have 2 effects on development of childhood overweight. On the one hand, these initial conditions can “program” structural or functional changes that lead to differences in the child's lifetime tendency toward overweight. The initial conditions affect the probability of overweight at T_1 , which through persistence of weight states will result in a lifetime tendency to overweight. The thicker arrows in Fig 1 illustrate the “programmed” lifetime tendency toward overweight hypothesized because of these initial conditions. We also hypothesize that initial conditions may have an independent effect on the dynamics of the process that leads to the development of overweight at T_2 and T_3 by changing the probability that the child moves between weight states at successive periods. We call this phenomenon the dynamic effect of the initial conditions. The dotted arrows in Fig 1 illustrate dynamic effects. A dynamic effect exerts a direct and independent influence on the probability of transition between weight states across successive time periods conditional on weight in the previous state. Contemporaneous risk factors, represented by the upward arrows in Fig 1, can influence the overweight development process.

Previous studies that analyzed the effects of initial conditions on weight of cross-sections of children at different ages cannot isolate the persistent effects from the dynamic effects of the initial conditions hypothesized by our model. For example, maternal prepregnancy type 2 diabetes, known to occur with increasing frequency in obese women, may affect a lifelong change in insulin production and sensitivity in her offspring, increasing the likelihood that these offspring will become overweight. This represents a persistent effect. Maternal prepregnancy obesity may also be an indicator for a shared postnatal nutritional environment that places children at risk for becoming overweight if he or she was normal weight in the previous observation period or at risk for remaining overweight if he or she was overweight in the previous period. This represents the dynamic effects of the initial conditions. These 2 effects can be identified separately only with longitudinal data over the child's life course and a dynamic modeling framework in which the child's weight state is a function of observed weight state in the preceding observation period. The purpose of this article is to use a life-course approach to examine the dynamic processes that drive childhood overweight, examining

the effects of prenatal characteristics and early-life feeding practices on childhood overweight through age 7 years.

METHODS

Study Population

We used data from the National Longitudinal Survey of Youth's Child-Mother file. This data set was based on the National Longitudinal Survey of Youth 1979, which began in 1979 with 6283 young women aged 14 to 21. These women were followed annually until 1994 and biennially thereafter. The military sample ($N = 456$) was dropped in 1985. The poor white oversample ($N = 195$) was dropped in 1991. We focus on the 5729 women in the cross-section sample and the oversamples of black and Hispanic individuals. In 1986, the National Longitudinal Survey of Youth's Child-Mother began biennially surveying the children who were born to these women. As of 2002, the latest round of survey data available, 9595 children were born to the cross-section sample, which included a nationally representative cross-section of women who lived in the United States in 1978 and were born between 1957 and 1964 and the oversamples of black and Hispanic individuals of the comparable birth cohorts.

Our sample is composed of children who had valid information on height and weight and were observed during 3 consecutive biennial interviews when they were aged 24 to 95 months. We required 3 observations on each child. The first observation period, T_1 , occurred when the child was between 24 and 47 months (age 2–3 years). The second observation period, T_2 , occurred when the child was between 48 and 71 months (age 4–5 years). The third observation period, T_3 , occurred when the child was between 72 and 95 months (age 6–7 years). Because the data are collected biennially only beginning in 1986, children who were born before 1982 and after 1996 could not be included in the study. Likewise, children of the 1982 to 1996 birth cohorts who missed an interview between the ages of 2 and 7 years were excluded. Because we are concerned about the effects of prenatal and birth characteristics on subsequent child overweight, we also required valid information on these characteristics.

Of the 6615 children who were born between 1982 and 1996, 4197 had 3 consecutive interviews during the appropriate age ranges. Height and weight were observed in each period on 3331 children. The final sample consists of 3022 children for whom we also observed all of the prenatal characteristics and early feeding practices in the analysis. Because of the loss in subjects as a result of the inclusion criteria, we tested (using t tests) for differences in demographics and outcomes between the eligible sample (6615 children) and the study sample (3022). The study sample overrepresented white individuals by 6 percentage points and underrepresented black and Hispanic individuals by 3 percentage points each. Within age and race/ethnicity groups, the prevalence of overweight for white children was no different between the eligible sample and the study sample. However, the prevalence of overweight for black children aged 72 to 95 months was lower in the study sample than in the eligible sample. The prevalence of overweight for Hispanic children in both of the groups aged 48 to 71 months and aged 72 to 95 months was lower than in the eligible sample.

Data Construction and Measures

The primary outcome of interest was childhood overweight, defined as BMI >95th percentile, as determined using the 2000 Centers for Disease Control and Prevention (CDC) Growth Charts for the United States. The CDC has published gender-specific growth charts of BMI for age for children aged 2 to 20 years.⁶¹ The 95th percentile of the 1977 National Center for Health Statistics Growth Charts has been used for many years to screen children who are at increased overall health risk. The upper percentiles of the revised 2000 curves are higher than the corresponding percentiles of the 1977 curves and result in fewer children's being classified

as overweight. The BMI percentile scores were determined using the Nutrition program within the CDC's Epi Info software program. The program calculates a BMI percentile using the child's height, weight, age in months, and gender. Height and weight were obtained on each child at the interview. The interviewer measured height and used a scale to obtain the weight of the child. Interviewers were trained to conduct these measurements by the National Opinion Research Center at the University of Chicago. Mothers were given the option of reporting the height and weight. The method that was used to obtain the height and weight measures (measured or reported) was recorded as part of the interview in all years except 1986. In 1986, the interviewers measured the children, but the method to obtain height and weight was not recorded. In preliminary analyses, we found that the reported mean values for height were always less than the measured values in each age category. The reported mean value of weight was greater than the measured value at T_1 and T_2 . However, the reported mean value of weight was less than the measured value at T_3 . Because of this, we controlled for the method of data collection in the analyses.

Explanatory variables included both time-invariant and time-varying characteristics and were chosen because they have been shown in previous studies to be associated with childhood overweight. The time-invariant characteristics included the following:

- Maternal age at the time of the birth of the child, reported in years
- Race/ethnicity (reported as white, black, or Hispanic)
- Maternal smoking during pregnancy (reported as yes/no in response to a questions asking the mother whether she ever smoked cigarettes during pregnancy)
- Breastfeeding (reported as yes/no in response to the question was child ever breastfed; if no, then bottle feeding = 1)
- Percentile birth weight for gestational age based on mother-reported data (a nearly continuous measure developed and reported by Oken et al⁶²)
- Gender (male = 1)
- Parity (collapsed into either first child or not first child)
- Maternal prepregnancy weight (reported in response to a question asking for the woman's weight immediately before pregnancy, and, using mother's reported height, BMI was determined and classified as underweight, BMI <18.5 kg/m²; normal weight, BMI 18.5–24.9 kg/m²; overweight, BMI 25–29.9 kg/m²; and obese, BMI ≥30 kg/m²)
- Birth cohort, determined by year of birth of child and used as continuous variable in the logistic models and categorized into 5-year periods for descriptive reporting.

Time varying characteristics included

- Maternal education, collected at each interview and reported in number of years of completed schooling
- Mother's marital status (collected at each interview in response to the question, married with spouse present = 1; otherwise = 0)
- Child's current age in months was included to control for the wide age span (24 months) necessitated because of the timing of the data collection
- Child's weight status (normal, <95th percentile or overweight ≥95th percentile) during the previous observation period

Data Analysis

The analyses were conducted separately on children at T_1 , and T_2 , T_3 . The sample was restricted to children with observations in each of the age ranges, because in some of the analysis, we condition on child overweight during the previous observation period, eg, the probability of a child's being overweight at age 4 years (6 years) is conditioned on the child's overweight status at age 2 years (4 years). There were 2070 mothers in the sample, 1307 of whom have a single child in the sample and 763 of whom have multiple children, with an average of 2.25 children per multiparous mother.

The analysis proceeded first with χ^2 tests of the bivariate association between each of the independent factors and child overweight at the 3 ages. To study the relationship between child overweight and several independent variables, we estimated multivariate logistic regressions at the 3 ages. These models were run unconditioned on overweight during the previous observation period. Next, we estimated first-order Markov models of the transition probabilities between 2 observation periods. To do so, we conditioned the logistic models at T_2 and T_3 on the child's being overweight during the previous period. By including the child's previous weight, we controlled for the effect of factors up to the previous period capitalized into the child's previous weight state and tested for independent dynamic effects of the initial conditions during the current period. These analyses provided information about the persistence of independent risks across time, the timing of the influence, and the magnitude of the risk. Estimates were obtained using Stata (Stata Corp., College Station, TX). A robust Huber/White/sandwich estimator of the variance of the logistic odds ratios was used to account for the nonindependence in the errors between siblings who were born to the same mother.

RESULTS

Table 1 presents the variables used in this analysis. Of the 3022 children, 53% were white, 27% were black, and 20% were Hispanic. The mean age of the child at T_1 was 35 months, at T_2 was 60 months, and at T_3 was 84 months. Thirty-eight percent of the children were first-born. Approximately half of the children were ever breastfed. The mean age of the mother at birth was 26.9 years, and 29% smoked during pregnancy. Sixty-three percent of the mothers were normal weight before pregnancy, 8% were underweight, 19% were overweight, and 10% were obese before pregnancy. The percentage of mothers who were married decreased slightly over time, from 70% at T_1 to 67% at T_3 . Mean number of years of education was ~13 years at each of the time points. The mean percentage of weight for gestational age was 51%. Measurement of height was recorded in 55% of children at T_1 and ~80% at T_2 and T_3 . Measurement of weight was recorded in 47% of children at T_1 and ~76% at T_2 and T_3 . The lower prevalence of recorded measurement in T_1 was driven by the fact that in 1986 it was not recorded whether height and weight were measured or mother reported.

Table 2 details the percentage of overweight children by subgroup. At each of the 3 times, a greater percentage of black and Hispanic children were overweight, compared with white children. The percentage of children who were overweight decreased in all race/ethnic groups as the children aged. This is consistent with the fact that we study children over their life cycle up to a point just past their adiposity rebound. There was little difference in overweight between boys and girls or by parity. Method of feeding (breast or bottle) and maternal smoking during pregnancy showed differences as expected. As the mother's prepregnancy weight increased, so did the percentage of children who were overweight. Marital status was significant in the 2 earliest periods, with a higher percentage of overweight children in households without a married couple. There were significant differences by child's birth year at T_1 but not at T_2 or T_3 .

Table 3 presents the results of the 3 cross-sectional logistic models run at 3 different time points. Several of the bivariate relationships were no longer significant in the multivariate models. At the earliest time, T_1 , black (odds ratio [OR]: 1.61; 95% confidence interval [CI]: 1.24–2.07) and Hispanic (OR: 1.65; 95% CI: 1.26–2.15) individuals were more likely to be overweight than white individuals. Children whose mothers smoked during pregnancy were more likely to be overweight (OR: 1.37; 95% CI: 1.08–1.73). They were also more likely to be overweight if their mother's prepregnancy BMI was ≥ 30 (OR: 1.37; 95% CI: 1.02–1.84). Age of the mother at birth, breastfeeding, percentile weight for gestational age, gender of the child, age of child in months, parity, and socioeconomic status (SES; marital status and education) were not significant. When the height (OR: 0.63; 95% CI: 0.47–0.84) and weight (OR: 0.53; 95% CI: 0.39–0.71) were recorded to have been measured, the child was less likely to be overweight. Children who were born in a later birth cohort were at greater risk for overweight (OR: 1.17; 95% CI: 1.11–1.23).

At time T_2 , when the child was between 48 and 71 months, race/ethnicity (black: OR: 1.57; 95% CI: 1.17–2.10; Hispanic: OR: 1.43; 95% CI: 1.05–1.96), smoking during pregnancy (OR: 1.43; 95% CI: 1.11–1.84), and the birth cohort (OR: 1.07; 95% CI: 1.01–1.13) remained statistically significant. When the mother was overweight (OR: 1.40; 95% CI: 1.07–1.83) or obese (OR: 1.69; 95% CI: 1.22–2.34) before pregnancy, the child was more likely to be overweight. Percentile birth weight for gestational age was significant, with increasing weights associated with an increased risk for overweight (OR: 1.50; 95% CI: 1.02–2.19). Older mothers were less likely to have an overweight child (OR: 0.95; 95% CI: 0.90–0.99). Breastfeeding, gender of the child, child age in months, parity, and SES (marital status and education) remained insignificant. When the height (OR: 0.63; 95% CI: 0.44–0.91) or weight (OR: 0.71; 95% CI: 0.52–0.97) was measured, the child was less likely to be overweight.

At time T_3 , when the child was between 72 and 95 months, race was significantly associated with over-weight for black (OR: 1.73; 95% CI: 1.28–2.35) and Hispanic (OR: 1.64; 95% CI: 1.18–2.28) individuals. Smoking during pregnancy (OR: 1.74; 95% CI: 1.32–2.29), maternal prepregnancy weight (overweight OR: 2.11; 95% CI: 1.60–2.78; obese OR: 2.91; 95% CI: 2.09–4.03), percentage weight for gestational age (OR: 1.61; 95% CI: 1.07–1.74), and birth cohort (OR: 1.07; 95% CI: 1.01–1.14) remained significant. In addition, parity (OR: 1.36; 95% CI: 1.07–1.74) and child's age in months (OR: 1.02; 95% CI: 1.01–1.04) now exerted significant influences. Age of the mother at birth, gender of the child, and SES (marital status and education) continued to be statistically insignificant. When height (OR: 0.55; 95% CI: 0.37–0.81) was recorded to have been measured, the child was less likely to be overweight, but there were no statistical differences between children whose weight was recorded as measured.

Table 4 presents results of the first-order Markov models at times T_2 and T_3 . The greatest risk for overweight is the weight state in the previous period. At T_2 , the child was approximately twice as likely to be overweight when he or she was overweight at T_1 (OR: 2.21; 95% CI: 1.68–2.92 when not measured and OR: 2.70; 95% CI: 1.83–3.99 when measured). At T_3 , the child was 6 to 16 times more likely to be overweight (OR: 6.22; 95% CI: 4.33–8.92 when not measured and OR: 16.47; 95% CI: 12.04–22.54 when measured) when he or she was overweight at T_2 . When conditioned on the previous observation state, percentile birth weight for gestational age and birth cohort were no longer significant at either period. Parity was no longer significant at T_3 . At T_2 , Hispanic ethnicity was no longer significantly associated with overweight in the conditioned models but remained significant in the conditioned model at T_3 (OR: 1.60; 95% CI: 1.13–2.25). Black children were at higher risk for overweight than white children in the conditioned models at T_2 (OR: 1.46; 95% CI: 1.09–1.96) and T_3 (OR: 1.59; 95% CI: 1.14–2.23). Maternal smoking during pregnancy at T_2 (OR: 1.37; 95% CI: 1.05–1.78) and T_3 (OR: 1.71; 95% CI: 1.28–2.31) remained significant and of similar magnitude to that found in the cross-section models. Maternal prepregnancy overweight at T_2 (OR: 1.40; 95%

CI: 1.07–1.85) and T_3 (OR: 2.13; 95% CI: 1.57–2.88) remained significant and of similar magnitude to that found in the cross-section models. Maternal prepregnancy obesity at T_2 (OR: 1.64; 95% CI: 1.18–2.28) and T_3 (OR: 2.89; 95% CI: 2.02–4.15) remained significant and of similar magnitude to that found in the cross-section models. Gender of the child, maternal SES, and maternal age at birth remained insignificant.

DISCUSSION

The findings from this study suggest that a dynamic model can yield insights into the effects of the initial conditions on the development of childhood overweight. Such a model allows separation of the persistent effects from the dynamic effects of the initial conditions. Referring to Fig 1, we found that the variables that had a persistent effect on childhood overweight, ie, those represented by the heavy arrows that affected the probability that the child was overweight between 24 and 47 months, were black race, Hispanic ethnicity, maternal smoking during pregnancy, and maternal obesity before pregnancy.

Estimates from the Markov models of overweight in subsequent periods suggest that the greatest risk for overweight is the child's having been overweight in the previous observation period. Thus, risk factors for overweight between the ages of 24 and 47 months have a persistent effect consistent with the heavy arrows in the model described by Fig 1. In a separate analysis, we examined the previous weight states for children who were overweight and of normal weight at T_3 . Two thirds of the children who were overweight at T_3 had been overweight during at least 1 previous period, whereas three fourths of the normal-weight children had always been of normal weight. This illustrates a strong tendency for weight states to track across time. Although there is movement across weight states, there is also a pattern established at a very young age that can be used to identify children who are at risk and target specify preventive strategies.

The variables that had dynamic effects in estimates of the Markov models, ie, variables that had a significant effect of weight at T_2 and T_3 conditional on overweight in the previous period, included black race, Hispanic ethnicity (in T_3 only), maternal smoking during pregnancy, maternal prepregnancy overweight, and maternal prepregnancy obesity. These variables increase the probability that a child will transition into overweight or remains overweight conditional on overweight in the previous observation period. These variables correspond to the dynamic effects signified by the dashed arrows in Fig 1.

At T_1 , the only maternal prepregnancy weight category that had a statistically significant effect on child overweight was maternal obesity. In each of the subsequent periods, the influence of maternal prepregnancy obesity increased substantially. By T_2 , children of mothers who were overweight before pregnancy were also at greater risk for being overweight. There was a similar time pattern for the effects of maternal overweight in that the magnitude of this risk factor increased in T_3 . The magnitude of the risk for childhood overweight increased with the weight category of the mother. Conditioning on previous weight resulted in only small changes in the point estimates of the ORs of maternal prepregnancy weight. That the ORs in the cross-sectional models and in the Markov models of transition probabilities have similar magnitudes suggest that the effect of maternal prepregnancy weight not only is the result of an early, persistent propensity to overweight but also affects the dynamics of the process of the development of childhood overweight. Whether these findings result from an intrauterine programming process that affects the dynamics of childhood overweight or that maternal prepregnancy overweight is a proxy for an obesogenic home environment high in caloric foods and not conducive to exercise remains an open question.

Smoking during pregnancy was related to childhood overweight at T_1 and had independent effects after conditioning on the weight state during the previous period at T_2 and T_3 . The ORs

were similar in the cross-sectional models and the Markov models. These findings add to the growing body of evidence that smoking during pregnancy may be a significant risk factor for overweight development during childhood. However, we do not have data on postnatal maternal smoking and cannot test for an effect of prenatal smoking while controlling for postnatal maternal smoking. A third alternative is that smoking during pregnancy is not itself a risk factor for childhood overweight but is instead a marker for an obesogenic home environment. Thus, it is preliminary to state that smoking may have an effect in utero.

Race and ethnicity were related to childhood overweight at T_1 and had independent effects after conditioning on the weight state during the previous period at T_2 and T_3 for black children. For Hispanic children, ethnicity was significant in the conditioned model only at T_3 . The influences of race and ethnicity are laid down early in life and operate for black individuals both through early development that persists and an independent risk for development at T_2 or T_3 . Black children are at greater risk for overweight than white children because overweight is more likely to develop at the youngest age, and that has a persistent effect over time. In addition, at older ages, being black is a risk factor for becoming or remaining overweight, even conditional on past weight status. The findings show that at the youngest ages, Hispanic children are more likely than white children to develop overweight. At T_2 , Hispanic children are more likely to be overweight because of the persistence of overweight from T_1 , but there is no increased risk for Hispanics to become or remain overweight. By T_3 , Hispanic individuals are more likely to be overweight as a result of the persistence of previous overweight, and there is also an independent influence of entering or remaining overweight.

We included the birth cohort in our analysis and found that in cross-sectional models, children who were born in later cohorts were more likely to be overweight than those who were born in earlier birth cohorts. In the dynamic models, birth cohort was no longer significant. This raises questions about why children, especially very young children aged 24 to 47 months, who were born in the mid-1990s were more likely to become overweight at very young ages than children who were born 10 years earlier. The rapidity of this change argues against genetics and for environmental conditions. One clear difference is the weight status of women. The prevalence of overweight has been increasing in adults as well as in children. In our sample, the mean prepregnancy weight of the mothers has increased over time (a mean increase of 14 lb, mean weight of 133 lb, BMI = 23 during the period from 1982 through 1986 to a mean weight of 147 lb, mean BMI = 25 a decade later, 1992–1996). The increase in adult BMI has been related to a nutrition transition that occurred in the last 1 or 2 decades of the 20th century. The nutrition transition is characterized by diets high in saturated fats, sugar, and refined foods but low in fiber and on lifestyles characterized by lower levels of activity.⁶³

Another possible contributing factor to this time trend is household changes that have occurred during this period. One of the most notable trends is the rise in maternal employment over that past 30 years as more women have either elected to enter and remain in the workforce or been encouraged to enter the workforce because of welfare reform. From 1970 to 1999, the fraction of women who had children who were younger than 6 years and were in the workforce has doubled, from 30% to 62%. Anderson et al⁶⁴ found a small association between childhood overweight and the number of maternal hours per week worked over the life of the child, thus providing some evidence for an employment effect, but it does not explain a large portion of this time trend difference, and more needs to be done to understand why young children are at greater risk for overweight now as compared with a decade ago.

A limitation of the study is that height and weight were not measured for all children. Results from the Markov model at T_3 suggested that measurement error was a problem because of the large difference in the ORs on weight status at T_2 depending on whether it was recorded as being measured. A second major limitation of the study is that the study sample is not

representative of the eligible sample. There are more white individuals and fewer minorities in the study sample than in the eligible population. The use of race and ethnic control variables in the statistical analyses ameliorates but does not entirely solve this problem. A more serious sample selection problem is that the prevalence of overweight in black children aged 72 to 95 months is lower in the study sample than in the eligible population. This leads to an underestimation of the effect of black race in both the cross-section and Markov models for children aged 72 to 95 months. The prevalence of overweight among Hispanic children is lower in the study sample than in the eligible population for both ages 48 to 71 months and 72 to 95 months. This leads to an underestimation of the effect of Hispanic ethnicity in the cross-section models for these 2 age groups. It leads to an underestimation of the effect of Hispanic ethnicity in the Markov model for ages 48 to 71 months and has an indeterminate effect on the estimate of Hispanic ethnicity in the Markov model for ages 72 to 95 months. This study is limited further because we cannot shed light on the specific biological mechanisms whereby these initial conditions affect the dynamics of childhood overweight. Other limitations include lack of information on duration of breastfeeding, which may explain the insignificance of the breastfeeding variable in the childhood overweight equations, and mother reports of birth weight and length of gestation.

These findings are clinically relevant in that they suggest 3 areas of emphasis. First, they highlight the importance of very early monitoring (before the child's second birthday) of healthy weight maintenance. Because weight states have persistence across time, a child who reaches his or her second birthday at a healthy weight is less likely to become overweight at a later age. For children who are already overweight at these young ages, interventions should begin immediately. Women who are overweight or obese and considering becoming pregnant should be counseled to achieve a healthy weight before becoming pregnant. Women who smoke should also be counseled to quit as these results provide additional evidence that smoking during pregnancy may be harmful to the fetus. The mechanisms by which maternal smoking during pregnancy and prepregnancy obesity may influence child weight states are not fully understood. These risk factors may be operating through biological processes and/or as markers for obesogenic environments. From a clinical perspective, however, these findings suggest factors that can be used to identify children who are at high risk for the development of childhood overweight at very young ages, thus providing an opportunity to target intensive preventive strategies before the establishment of an unhealthy weight pattern.

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ABBREVIATIONS

CDC, Centers for Disease Control and Prevention; OR, odds ratio; CI, confidence interval; SES, socioeconomic status.

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Fig 1.
Dynamic model of the development of childhood overweight.

TABLE 1

Sample Characteristics

	n	% or Mean
Race/ethnicity		
White	1590	53%
Black	826	27%
Hispanic	606	20%
Child age, mo		
At T_1	3022	35
At T_2	3022	60
At T_3	3022	84
Gender		
Male	1553	0.51
Female	1469	49%
Parity		
First born	1153	38%
Not first born	1869	62%
% Weight for gestational age		
Mean % weight for gestational age	3022	51%
Feeding method		
Breast	1596	53%
Bottle	1426	47%
Smoking during pregnancy		
No	865	29%
Yes	2157	81%
Prepregnancy BMI		
Underweight, <18.5 kg/m ²	228	8%
Normal weight, 18.5–24.9 kg/m ²	1919	63%
Overweight, 25–29.9 kg/m ²	580	19%
Obese, ≥30 kg/m ²	295	10%
Highest grade completed (mother)		
At T_1	3022	12.88
At T_2	3022	12.96
At T_3	3022	13.02
Maternal age at birth	3022	26.9
Marital status: married spouse present		
At T_1	2127	70%
At T_2	2082	69%
At T_3	2036	67%
Child birth year		
1982–1986	1219	40%
1987–1991	1399	46%
1992–1996	404	14%
Weight measured		
At T_1	1415	47%
At T_2	2164	72%
At T_3	2273	75%
Height measured		
At T_1	1664	55%
At T_2	2419	80%
At T_3	2465	82%
Child overweight		
At T_1	580	19%
At T_2	406	13%
At T_3	372	12%

TABLE 2

Bivariate Comparisons of the Percentage of Children Who Were Overweight at Each Time Period According to Major Risk Factors

	Overweight		Overweight		Overweight	
	% at T_1	P	% at T_2	P	% at T_3	P
Race/ethnicity						
White	0.16	<.001	0.11	<.001	0.10	.001
Black	0.22		0.17		0.15	
Hispanic	0.22		0.15		0.14	
Gender						
Male	0.20	.20	0.14	.80	0.13	.67
Female	0.18		0.13		0.12	
Parity						
First born	0.19	.90	0.14	.15	0.13	.49
Not first born	0.19		0.13		0.12	
Feeding method						
Breast	0.17	.002	0.12	.009	0.11	.05
Bottle	0.22		0.15		0.14	
Smoking during pregnancy						
No	0.18	0.05	0.13	.01	0.11	.003
Yes	0.22		0.16		0.15	
Prepregnancy BMI						
Underweight, <18.5 kg/m ²	0.18	0.06	0.09	<.001	0.07	<.001
Normal weight, 18.5–24.9 kg/m ²	0.18		0.12		0.09	
Overweight, 25–29.9 kg/m ²	0.20		0.16		0.19	
Obese, ≥30 kg/m ²	0.26		0.19		0.24	
Marital status: married spouse present						
No	0.22	.006	0.16	.003	0.13	.51
Yes	0.18		0.12		0.12	
Child birth year						
1982–1986	0.13	<.001	0.16	.70	0.11	.21
1987–1991	0.23		0.13		0.13	
1992–1996	0.24		0.15		0.15	

TABLE 3
Multivariate Logistic Regressions of Childhood Overweight

	Overweight at T_1 , OR (95% CI)	Overweight at T_2 , OR (95% CI)	Overweight at T_3 , OR (95% CI)
Race/ethnicity			
White	1.00	1.00	1.00
Black	1.61 (1.24–2.07)	1.57 (1.17–2.10)	1.73 (1.28–2.35)
Hispanic	1.65 (1.26–2.15)	1.43 (1.05–1.96)	1.64 (1.18–2.28)
Gender			
Female	1.00	1.00	1.00
Male	1.15 (0.95–1.38)	1.05 (0.84–1.31)	1.06 (0.84–1.33)
Child age, mo	1.00 (0.98–1.01)	1.00 (0.98–1.01)	1.02 (1.01–1.04)
Parity			
Not first born	1.00	1.00	1.00
First born	0.97 (0.78–1.21)	1.24 (0.98–1.58)	1.36 (1.07–1.74)
% Weight for gestational age	1.40 (0.99–1.97)	1.50 (1.02–2.19)	1.61 (1.07–1.74)
Feeding			
Breast	0.81 (0.66–1.01)	0.90 (0.71–1.15)	0.94 (0.73–1.22)
Bottle	1.00	1.00	1.00
Smoking during pregnancy			
Yes	1.37 (1.08–1.73)	1.43 (1.11–1.84)	1.74 (1.32–2.29)
No	1.00	1.00	1.00
Prepregnancy weight			
Underweight, <18.5 kg/m ²	0.97 (0.65–1.44)	0.75 (0.47–1.19)	0.63 (0.36–1.11)
Normal weight, 18.5–24.9 kg/m ²	1.00	1.00	1.00
Overweight, 25–29.9 kg/m ²	1.04 (0.81–1.33)	1.40 (1.07–1.83)	2.11 (1.60–2.78)
Obese, ≥30 kg/m ²	1.37 (1.02–1.84)	1.69 (1.22–2.34)	2.91 (2.09–4.03)
Maternal age at birth	0.96 (0.92–1.01)	0.95 (0.90–0.99)	0.98 (0.93–1.04)
Maternal education: highest grade completed	1.00 (0.95–1.05)	0.98 (0.93–1.04)	0.97 (0.92–1.03)
Marital status: married spouse present			
Yes	0.87 (0.70–1.09)	0.89 (0.68–1.15)	1.17 (0.90–1.53)
No	1.00	1.00	1.00
Height measured	0.63 (0.47–0.84)	0.63 (0.44–0.91)	0.55 (0.37–0.81)
Weight measured	0.53 (0.39–0.71)	0.71 (0.52–0.98)	1.36 (0.94–1.97)
Birth cohort	1.17 (1.11–1.23)	1.07 (1.01–1.13)	1.07 (1.01–1.14)

TABLE 4

Comparison Between Multivariate Logistic Models of Childhood Overweight Unconditioned and Conditioned at T_2 and T_3 on Overweight in Previous Time Period

	T_2		T_3	
	Unconditioned, OR (95% CI)	Conditioned, OR (95% CI)	Unconditioned, OR (95% CI)	Conditioned, OR (95% CI)
Race/ethnicity				
White	1.00	1.00	1.00	1.00
Black	1.57 (1.17–2.10)	1.46 (1.09–1.96)	1.73 (1.28–2.35)	1.59 (1.14–2.23)
Hispanic	1.43 (1.05–1.96)	1.33 (0.98–1.82)	1.64 (1.18–2.28)	1.60 (1.13–2.25)
Gender				
Female	1.00	1.00	1.00	1.00
Male	1.05 (0.84–1.31)	1.04 (0.83–1.30)	1.06 (0.84–1.33)	1.08 (0.84–1.38)
Child age, mo	1.00 (0.98–1.01)	1.00 (0.98–1.02)	1.02 (1.01–1.04)	1.02 (1.01–1.05)
Parity				
Not first born	1.00	1.00	1.00	1.00
First born	1.24 (0.98–1.58)	1.22 (0.96–1.56)	1.36 (1.07–1.74)	1.22 (0.93–1.60)
% Weight for gestational age	1.50 (1.02–2.19)	1.43 (0.98–2.09)	1.61 (1.07–1.74)	1.43 (0.90–2.27)
Feeding				
Breast	0.90 (0.71–1.15)	0.92 (0.72–1.18)	0.94 (0.73–1.22)	0.99 (0.77–1.30)
Bottle	1.00	1.00	1.00	1.00
Smoking during pregnancy				
Yes	1.43 (1.11–1.84)	1.37 (1.06–1.78)	1.74 (1.32–2.29)	1.71 (1.28–2.31)
No	1.00	1.00	1.00	1.00
Prepregnancy weight				
Underweight, <18.5 kg/m ²	0.75 (0.47–1.19)	0.75 (0.47–1.19)	0.63 (0.36–1.11)	0.72 (0.40–1.30)
Normal weight, 18.5–24.9 kg/m ²	1.00	1.00	1.00	1.00
Overweight, 25–29.9 kg/m ²	1.40 (1.07–1.83)	1.40 (1.07–1.85)	2.11 (1.60–2.78)	2.13 (1.57–2.88)
Obese, ≥30 kg/m ²	1.69 (1.22–2.34)	1.64 (1.18–2.28)	2.91 (2.09–4.03)	2.89 (2.02–4.15)
Maternal age at birth	0.95 (0.90–99)	0.95 (0.90–1.00)	0.98 (0.93–1.04)	1.00 (0.94–1.06)
Maternal education: highest grade completed	0.98 (0.93–1.04)	0.98 (0.93–1.04)	0.97 (0.92–1.03)	0.98 (0.92–1.04)
Marital status: married spouse present				
Yes	0.89 (0.68–1.15)	0.91 (0.70–1.18)	1.17 (0.90–1.53)	1.32 (0.99–1.77)
No	1.00	1.00	1.00	1.00
Height measured	0.63 (0.44–0.91)	0.63 (0.46–0.86)	0.55 (0.37–0.81)	0.60 (0.37–0.87)
Weight measured	0.71 (0.52–0.98)	0.77 (0.57–1.05)	1.36 (0.94–1.97)	1.34 (0.92–2.02)
Birth cohort	1.07 (1.01–1.13)	1.06 (0.99–1.12)	1.07 (1.01–1.14)	1.05 (0.98–1.12)
Child overweight at T_1 (measured)				
No		2.21 (1.68–2.92)		
Yes		2.70 (1.83–3.99)		
Child overweight at T_2 (measured)				
No				6.22 (4.33–8.92)
Yes				16.47 (12.04–22.54)