

Early Childhood Poverty, Cumulative Risk Exposure, and Body Mass Index Trajectories Through Young Adulthood

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One of the most rapid and startling epidemics confronting contemporary society is obesity. The prevalence of overweight and obesity among US adults increased from 56% in 1988 to 1994 to 66.3% in 2003 to 2004. The rate of obesity alone rose from 22.9% to 32.2% among adults,¹ and the incidence of overweight among adolescents aged 12 to 19 years increased from 11% to 17% during the same period.² Obesity has been linked to elevated rates of diabetes, cancer, coronary heart disease, and other ailments.³ Being obese in late adolescence is associated with an adult mortality risk comparable to heavy smoking (>10 cigarettes/day), and being overweight in late adolescence is comparable to the adult mortality risk associated with light smoking (1–10 cigarettes/day).⁴ Because of the obesity epidemic, future life expectancy in the United States may actually drop in this century for the first time ever.⁵

An important predictor of adult obesity is early childhood socioeconomic disadvantage.^{6–13} This longitudinal association is even more consistent than are concurrent associations of socioeconomic status (SES) and obesity in adulthood.^{14,15} For example, Poulton et al. found that as childhood SES decreased, body mass index (BMI; defined as weight in kilograms divided by height in meters squared) and waist-to-hip ratio at age 26 years increased.¹⁰ Eighty percent of women who grew up in low-SES households were overweight or obese in adulthood; only 40% of women raised in higher-SES households were subsequently overweight or obese.¹⁶

The association between childhood poverty and later obesity has been documented in a variety of contexts. We assessed whether early childhood poverty leads to adult obesity because of cumulative risk exposure during childhood. We built on the childhood and obesity literature by using linear growth curve (LGC) modeling to examine whether individual life course trajectories in BMI are also affected by early childhood poverty.

Objectives. We assessed whether cumulative risk exposure underlies the relation between early childhood poverty and body mass index (BMI) trajectories.

Methods. We interviewed youths and their mothers in rural upstate New York (168 boys and 158 girls) from 1995 to 2006 when the youths were aged 9, 13, and 17 years. At each interview, we calculated their BMI-for-age percentile.

Results. Early childhood poverty predicted BMI growth trajectories from ages 9 to 17 years ($b=3.64$; $SE=1.39$; $P<.01$). Early childhood poverty also predicted changes in cumulative risk ($b=0.31$; $SE=0.08$; $P<.001$). Cumulative risk, in turn, predicted BMI trajectories ($b=2.41$; $SE=0.75$; $P<.01$). Finally, after we controlled for cumulative risk, the effect of early childhood poverty on BMI trajectories was no longer significant, indicating that cumulative risk exposure mediated the relation between early childhood poverty and BMI trajectories ($b=2.01$; $SE=0.94$).

Conclusions. We show for the first time that early childhood poverty leads to accelerated weight gain over the course of childhood into early adulthood. Cumulative risk exposure during childhood accounts for much of this accelerated weight gain. (*Am J Public Health.* 2010;100:2507–2512. doi:10.2105/AJPH.2009.184291)

Risk factors associated with poverty, such as poor housing quality or family turmoil, often do not occur in isolation. Cumulative risk captures the extent of ecological covariation in risk exposures by generating an index that additively models exposure to multiple sources of risk. Across multiple physical and psychological health outcomes, cumulative risk exposure predicts morbidity significantly better than does exposure to any single risk factor.^{17,18} Among adult women, less physical activity, greater likelihood of skipping breakfast, and inadequate sleep function as intervening variables between poverty and the likelihood of remaining obese.¹⁹

We employed a life course approach to examine the relation between early childhood poverty and the trajectory of obesity as well as the potential mediating role of cumulative risk. We tested 4 hypotheses in longitudinal data collected at 3 time points. First, we hypothesized that early childhood poverty predicts a life course trajectory of elevated BMI into young adulthood. Specifically, we theorized that the greater the proportion of a child's life

that is spent in poverty from birth to age 9 years, the more likely that child is to follow a trajectory toward obesity from childhood to early adulthood. Second, we hypothesized that early childhood poverty predicts cumulative risk exposure. Third, we examined how cumulative risk and obesity covary over time. We theorized that changes in cumulative risk predict changes in BMI over time. Finally, we hypothesized that the relation between early poverty and the BMI trajectory is mediated by cumulative risk exposure.

METHODS

We applied LGC analysis^{20,21} to examine changes in obesity from childhood to young adulthood. Our level 1 model estimated the association between time (represented by 3 waves of data collection) and the outcome variable, BMI percentile. The level 2 model introduced additional variables to explain individual differences in any statistical associations found at level 1. At level 2, we examined whether the predictor variable, childhood poverty, was

associated with observed individual differences in BMI trajectories. Last, we examined whether cumulative risk functioned as an underlying explanatory mechanism, linking early experiences of poverty with obesity.

Participants and Procedure

Participants were 329 youths born between 1983 and 1990 and interviewed when they were aged 9, 13, and 17 years, from 1995 to 2006. Three youths were omitted because their BMIs were outliers, leaving a sample of 326 youths. At initial recruitment, the children were in grades 3 through 5, and 53% lived in households with incomes at or below the federal poverty line. Low-income families were oversampled because our study was part of a larger research program focused on rural poverty.¹⁷

We recruited the poverty sample from participants in various programs in rural New York State, such as Head Start, Section 8 federal housing, and the New York State Co-operative Extension, and from public schools. We recruited middle-income children from Co-operative Extension programs and public schools. The refusal rate was less than 5%.

The overall mean income-to-needs ratio was 1.66 at wave 1, 2.36 at wave 2, and 2.84 at wave 3. Forty-nine percent of the participants were female, and 91% were White. In each of the 3 waves of data collection, youths and their mothers were interviewed independently in their homes by 2 trained assistants, who used a standard protocol to measure weight and height and to collect sociodemographic information and data on cumulative risk exposure.

Constructs and Measures

Childhood poverty. We measured childhood poverty as the proportion of the youth's life spent living in poverty from birth to wave 1, approximately age 9 years. Poverty was defined as an income-to-needs ratio of less than or equal to 1.0. This ratio was the annually adjusted, per capita index of poverty for the United States. We recorded this information for every 6-month period of life from birth through wave 3 (age 17 years) on the basis of household income and family composition.

Obesity—overweight status. Height and weight were measured by research assistants at each wave of data collection. To calculate BMI

variables that took into account a youth's age and gender, we used the SAS program for Centers for Disease Control and Prevention growth charts,²² yielding a percentile for BMI for age. This growth curve across 3 waves was the dependent variable.

Cumulative risk. We measured cumulative risk at each wave of data collection by summing 6 dichotomous items related to 3 physical risks (crowding, noise, and substandard housing) and 3 social risks (family turmoil, child's separation from parents, and exposure to violence). Crowding was measured by the total number of people living in a household divided by the total number of rooms in the house (people per room). Noise (equivalent constant decibel level) was measured with a decibel meter (Burel and Kjaer, Naerum, Denmark, model 2239A) placed in the main social area of the home for 2 periods of 2 hours each. Substandard housing was measured with a standardized, rater-based instrument.²³ Exposure to physical risks was calculated identically at each wave of the data collection.

For social risks, at wave 1 we determined exposure from mothers' reports on the Life Events and Circumstances checklist.²⁴ At wave 2, youths' self-reports on the Adolescent Perceived Events Scale²⁵ were also included (an event was counted as a single time if it was reported by the youth, the mother, or both). Each social risk factor was determined by multiple items (answered yes or no).

For each of the 6 individual risk domains, we coded risk dichotomously at each wave. A score of 1 indicated that the youth's exposure at a given wave was in the upper quartile for the entire sample's distribution of continuous exposure at that wave, and a score of zero was recorded in all other cases. We then calculated cumulative risk exposure (0–6) for each wave of data collection by summing across the 6 single risk factors.

Linear Growth Curve Analysis

We tested our hypotheses through LGC analysis.^{20,26,27} In this method, the individual LGCs, or trajectories, are estimated. The flexibility of LGCs provides several advantages. First, LGC analysis is appropriate for longitudinal data such as ours, which had a hierarchical structure with up to 3 within-person observations nested within each of 326 participants.

Second, the LGC method does not require that all individuals be measured at all occasions: we were able to include participants who had missing data for some time points. Third, in LGC analyses, reliable units of observation contribute more to the estimation of parameters than less reliable units, a process known as precision weighting (for a discussion, see Raudenbush and Bryk²⁰[p32–57]). By separating true and error variance, LGC analysis thus provides more accurate and robust estimates of parameters than does ordinary least squares analysis.

Finally, the LGC approach allowed us to estimate between- and within-person effects simultaneously. Between-person effects addressed the relation between intraindividual coefficients and interindividual variables (e.g., over time, is obesity, on average, higher among children who spend a greater proportion of time living in poverty?). Within-person effects addressed links between variables at the intra-individual level and yielded slope and intercept coefficients to index these relations (e.g., on occasions in which children experience greater cumulative risk, do they also evidence higher levels of obesity?). We also assessed the extent of within-person mediation across between-person effects (e.g., is the between-person association between early poverty and later obesity mediated by changes in cumulative risk?).

Linear Growth Curve Equations

We developed LGC equations predicting BMI percentiles to test our hypotheses. Following recommendations by Raudenbush and Bryk,²⁰ we centered all within-person variables on the individuals' means and all between-person variables on sample means. Equation 1 described the simplest within-person (i.e., level 1) model, specifying that the BMI percentile score for person j on occasion i was a function of his or her mean BMI percentile (β_{0j}) and a random residual component (e_{ij}):

$$(1) \text{ bmi}_{ij} = \beta_{0j} + e_{ij}.$$

This unconditional means model fits an overall mean and variance across all persons and measurement occasions.²⁶ The unconditional model provided a benchmark of within-person variance that we used to judge successive models.²⁰

We augmented equation 1 to include time. This was the basic LGC, illustrated in equation 2:

$$(2) \text{bmi}_{ij} = \beta_{0j} + \beta_{1j} \text{Time}_{ij} + e_{ij},$$

where bmi_{ij} represented BMI percentile on occasion i for person j , β_{0j} represented person j 's predicted average BMI percentile at the start of the study, β_{1j} was the linear coefficient or the rate of change (slope), and e_{ij} was a within-person error or residual term.

We expanded the basic LGC further to include between-person (i.e., level 2) predictors. More formally, the model was expressed as

$$(3) \text{B}_{1j} = \gamma_{10} + \gamma_{11} \text{Poverty} + u_{1j}.$$

Here the level 1 linear coefficient (β_{1j} , or average BMI slope) was regressed on the between-person variable poverty (γ_{11}), and a between-persons error term (u_{1j}). Thus, in equation 3, (γ_{11}) can be interpreted as the between-person effect of poverty status on the linear rate of change in BMI.

Finally, to assess within-person associations, we augmented the level 1 model (in equation 1) with time-varying predictors. Equation 4 showed BMI as a function of cumulative risk:

$$(4) \text{bmi}_{ij} = \beta_{0j} + \beta_{1j} \text{Cumulative Risk}_{ij} + e_{ij},$$

where bmi_{ij} represented the amount of BMI on occasion i for person j . Because we centered our within-person variables, β_{0j} represented person j 's predicted level of BMI on an occasion of average childhood cumulative risk, β_{1j} was the within-person slope of the cumulative risk–BMI relationship for person j , and e_{ij} was a within-person error or residual term.

We first examined an unconditional means model (equation 1), which allowed intercepts (mean levels) to vary randomly. The ratio of between- to within-person variance is called the intraclass correlation, and the rule of thumb is that the correlation should be above 0.10 to permit moving on to growth curve models.²⁰ Anything less than 0.10 indicates very little within-person variability. We next examined how average levels of BMI percentile varied over time (equation 2). This coefficient is functionally equivalent to an unstandardized regression coefficient and can be interpreted as such.

RESULTS

Our unconditional means model (equation 1) yielded an estimate of the mean BMI percentile score of 64.93. The estimated intraclass correlation was 0.62. These data thus indicated that there was sufficient variability at the within-person level to allow for the possibility of modeling intraindividual relationships in BMI.

The linear effect or overall rate of change in BMI over time (equation 2) was significant ($b=5.96$; $SE=0.90$; $P<.001$). For every unit increase in time (i.e., each approximate 4-year interval between waves of data) mean levels of BMI percentile increased 5.96 units.

Poverty

The longitudinal association between childhood poverty and BMI percentile is illustrated in Figure 1. The cross-sectional relation between proportion of life spent in poverty and BMI percentile at waves 1, 2, and 3 is shown in Table 1. The figure and table categorize the proportion of life spent in poverty from birth to age 9 years for descriptive purposes only; all

inferential analyses maintained the continuous nature of the childhood poverty index.

Our first hypothesis was that BMI trajectories would vary as a function of childhood poverty (birth to wave 1; equation 3). We regressed BMI percentile scores on childhood poverty. The main effect of childhood poverty was significant ($b=3.64$; $SE=1.39$; $P<.01$). We examined the strength of this relationship by comparing random parameter estimates, and strength was quantified as the between-person variance in BMI accounted for by poverty, a procedure discussed in Raudenbush and Bryk.^{20(p65)} The residual variance of β_{1j} (BMI) from an analysis in which poverty was not included at the person level was 67.24, and the residual variance from a second analysis in which poverty was included was 64.21, a reduction of 5%. This corresponds to an effect size correlation of 0.22 between poverty and linear changes in BMI percentile.

Cumulative Risk

The relation between poverty and cumulative risk is illustrated in Figure 2. To test the

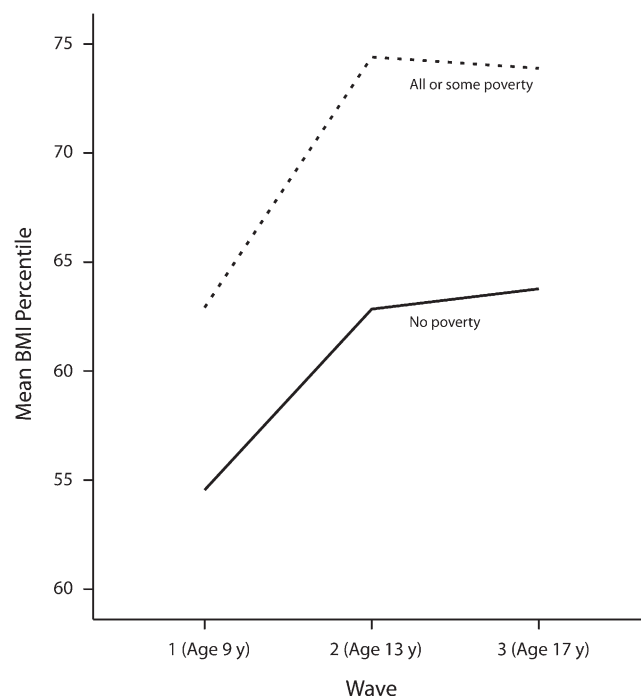


FIGURE 1—Body mass index (BMI) percentile at age 9 years (wave 1), age 13 years (wave 2), and age 17 years (wave 3) years, by time spent in poverty from birth to age 9 years: 1995–2006.

TABLE 1—Mean BMI Percentile by Proportion of Life Spent in Poverty at Waves 1, 2, and 3, cross-sectionally: 1995–2006

Proportion of Life Spent in Poverty	Wave 1 (Age 9 Years; n=261)		Wave 2 (Age 13 Years; n=203)		Wave 3 (Age 17 Years; n=198)	
	BMI percentile (SD)	No.	BMI percentile (SD)	No.	BMI percentile (SD)	No.
None	55.07 (32)	141	64.00 (28)	112	64.78 (28)	114
Some	48.99 (27)	8	73.58 (27)	52	74.78 (28)	67
All	66.47 (27)	112	76.06 (19)	39	64.94 (27)	17

Note. BMI = body mass index. BMI is defined as weight in kilograms divided by height in meters squared.

hypothesis that early poverty would predict changes in cumulative risk, we regressed cumulative risk scores on poverty. As predicted, there was a main effect of poverty ($b=0.31$; $SE=0.08$; $P<.001$): for every unit increase in poverty, mean levels of cumulative risk increased by 0.31 units. Examination of the random parameter estimates indicated that poverty accounted for 52% of the intra-individual variance in cumulative risk, corresponding to an effect size correlation of 0.72.

To examine the effect of cumulative risk on BMI, we regressed BMI scores on ratings of cumulative risk. We found a main effect of cumulative risk ($b=2.41$; $SE=0.75$; $P<.01$). Thus, for every unit increase in cumulative risk, mean BMI percentile increased 2.41 units. Examination of the random parameter estimates indicated that cumulative risk accounted for 21% of the intra-individual variance in BMI percentile, corresponding to a within-person correlation of 0.46.

Mediation

Our main hypothesis was that cumulative risk would serve as an explanatory mechanism,

linking early experiences of poverty with BMI trajectories into adulthood. The statistical analysis framework²⁸ entailed 3 separate equations. Step 1 was to find a significant main effect of the predictor (poverty) on the outcome (BMI) supported by hypothesis 1. Step 2 was to find a significant main effect of the predictor (poverty) on the mediator (cumulative risk) supported by hypothesis 2. Step 3 was to find a significant effect between the mediator (cumulative risk) and the outcome (BMI) supported by hypothesis 3. Statistical mediation was supported if the predictor (poverty) had no effect on the outcome (BMI) when the mediator (cumulative risk) was controlled.²⁸

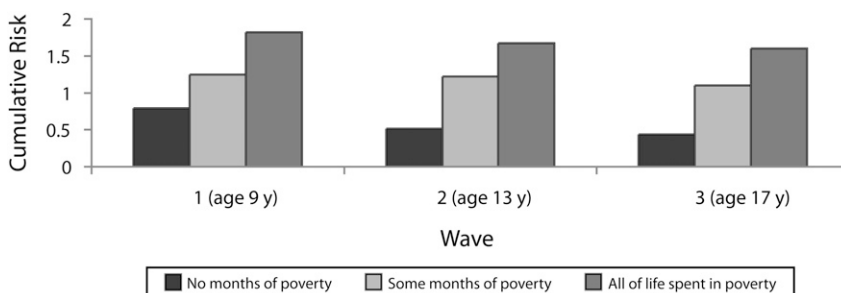
The data indicated that the effect of early childhood poverty on BMI trajectories was no longer significant when we controlled for changes in cumulative risk ($b=2.01$; $SE=0.94$; Figure 3). As an additional check on the mediation effect, we examined 95% confidence intervals (CIs); if zero was not within the upper and lower limits of the CI, then the mediation effect was statistically significant.²⁹ In these data, the lower and upper limits were

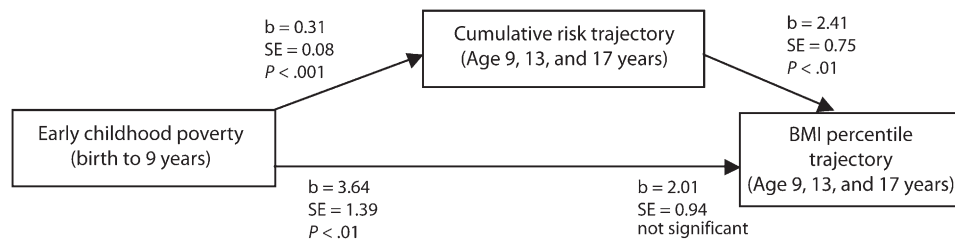
–3.621 and –1.248, respectively, indicating significant mediation. Taken together, these results supported our main hypothesis (hypothesis 4), that cumulative risk represents a mediation path linking early poverty to subsequent elevated BMI.

To further examine whether cumulative risk constitutes a unique intervening pathway that links childhood poverty to BMI trajectories, we also tested a plausible alternative model of mediation. The alternative model tested whether BMI might in fact mediate the association between childhood poverty (as predictor) and cumulative risk (as outcome). This alternative model located changes in BMI as the key mediator accounting for the association between childhood poverty and cumulative risk. We tested the model with a regression equation in which cumulative risk was the dependent variable, with BMI and childhood poverty entered simultaneously as level 1 and level 2 predictors, respectively. We found no evidence of mediation (95% CI=–0.03, 0.08). Childhood poverty remained a strong predictor of cumulative risks even when BMI was controlled ($b=0.28$; $SE=0.06$; $P<.001$). The null effects of this alternative model provided support for cumulative risk as a mediator of the pathway from childhood poverty to BMI trajectories.

DISCUSSION

For the first time, we showed that early childhood poverty predicted individual trajectories of BMI into young adulthood. This finding substantially strengthens earlier evidence for an association between childhood SES and weight.^{6–16} We also showed that accumulated exposure to multiple social and physical risk factors served as a mediator, largely explaining the association between early poverty and subsequent obesity. Specifically, family turmoil, violence, child separation from family, density, noise, and housing quality together provided an underlying explanatory mechanism linking early childhood poverty to obesity in young adulthood. Poorer children become overweight adults, at least in part, because they are confronted with a greater array of risk factors over their life course.

**FIGURE 2—Cumulative risk by proportion of life spent in poverty between birth and age 9 years at waves 1, 2, and 3: 1995–2006.**



Note. The nonsignificant path, after taking into account cumulative risk as a mediator, has no corresponding P value.

FIGURE 3—Cumulative risk as the mediator of relation between early childhood poverty and body mass index (BMI) percentile trajectory: 1995–2006.

Our results make 2 important contributions to the literature. To our knowledge, ours was the first study to examine the relation between early childhood poverty and individual trajectories in weight gain from childhood to adulthood. In addition, we examined a plausible underlying explanatory mechanism to account for the well-established link between early childhood poverty and adult weight gain. Many studies have revealed health inequalities in adulthood in relation to childhood SES.^{30–32} Our data help to elucidate long-established pathways between childhood SES and adult health outcomes associated with obesity, such as cardiovascular disease,³ by suggesting that a key aspect of the environment of childhood poverty, the confluence of risk exposure, may play an essential role in setting life course trajectories for health.^{33,34}

These findings also have important implications for obesity prevention intervention strategies. Identification of mechanisms that link poverty to later obesity reveals a variety of possible leverage points. In addition to efforts to reduce poverty and improve early detection of obesity tendencies, interventions might address the mediating cumulative risk factors of family turmoil, violence, child separation from family, density, noise, and housing quality directly.

Limitations

Because we did not randomly select our sample, our findings may not be generalizable to the broader population. Although the longitudinal design of this study strengthened our ability to draw conclusions regarding causal order, a true experiment employing random assignment would enable more confident conclusions. A spurious variable could possibly

drive the relations observed among variables; however, we attempted to rule out this possibility through our supplemental analysis with an alternative model of mediation.

Finally, our study would have been strengthened by more waves of data collection across more of the life course. Replication among more ethnically diverse, urban children would also be valuable.

Conclusions

Early childhood poverty is a well-documented risk factor for obesity. Our results show that this relationship is established early in life and linked to trajectories in weight gain from childhood through young adulthood. We also provide evidence that income inequalities in BMI are attributable, at least in part, to the accumulation of multiple risk exposures accompanying childhood poverty.

Future research might examine more explicitly how and why a life course trajectory characterized by cumulative risk leads to obesity in late adolescence. What are the proximal processes that underlie such associations?³⁵ For example, are SES-related differences in diet and physical activity directly linked to stressor exposure or to parenting practices in more chaotic, stressful settings? Chronic stress itself influences biological mechanisms that make the body less efficient in metabolizing fat.³⁶ Research might examine the role of body image, which has been found to partially mediate the association between obesity and psychological distress.³⁷

Researchers might also examine whether there is a critical period linking cumulative risk exposure to obesity. In other words, at what ages or stages of development are children more or less vulnerable to risk exposure and weight gain? Moreover, from a life course

perspective, it would be useful to understand what life events or intervention strategies alter life course trajectories in weight gain such that those at risk avoid adult obesity. ■

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Contributors

N.M. Wells originated the study, led the writing, and contributed to the analysis. G.W. Evans originated the study, supervised the data collection, and contributed to writing the article. A. Beavis assisted with data collection, data management, and data analysis. A.D. Ong led the data analysis and contributed to writing the article. All authors reviewed drafts of the article.

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Human Participant Protection

This study was approved by Cornell University's institutional review board.

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