Original Contribution

Chronic Exposure of Grandparents to Poverty and Body Mass Index Trajectories of Grandchildren: A Prospective Intergenerational Study

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In this study, I used the growth curve model to examine the association between grandparents' (first generation (G1)) life-course exposure to chronic poverty and grandchildren's (third generation (G3)) body mass index (BMI; weight (kg)/height (m)^2) growth trajectories. This association was estimated separately for male and female grandchildren. Analyses were based on prospective data from a US longitudinal survey, the Panel Study of Income Dynamics (1968–2011), and 2 of its supplemental studies: the Child Development Supplement (1997–2011) and the Transition into Adulthood Study (1997–2011). A prospectively enrolled nationally representative cohort of 2,613 G3 youth (1,323 male, 1,290 female) sampled in the 2 supplemental studies was linked to 1,719 grandparents from the Panel Study of Income Dynamics core sample. Chronic exposure to poverty among grandparents was prospectively ascertained annually over a 30-year period prior to the collection of data on grandchildren. Findings suggested that grandparents' chronic poverty exposure was positively associated with the slope of the BMI trajectory among granddaughters ($\beta = 0.10$, 95% confidence interval: 0.03, 0.17) but not among grandsons ($\beta = 0.02$, 95% confidence interval: −0.04, 0.08). The association between grandparents' chronic poverty exposure and granddaughters' BMI growth slope remained even after controlling for parental (second generation (G2)) socioeconomic status and BMI.

body mass index; chronic poverty; intergenerational transmission; life course; obesity; socioeconomic status

Obesity is an important risk factor for multiple chronic conditions and mortality (1–5). It also exacts a significant social and psychological toll on the individual due to the stigma associated with obesity in modern societies (6, 7). The literature has documented an inverse association between socioeconomic status (SES) and the risk of obesity (8, 9). Recent life-course studies have suggested that the SES gradient in obesity travels across generations, in the sense that people with poor parents are more likely to gain excess body weight in the long run (2, 4, 5, 10–13). Such an intergenerational SES-obesity association is particularly consistent among females (12, 14).

However, still absent is a study that extends the life-course model of the SES-obesity gradient to cover 3 generations. A 3-generation study of the SES-obesity association has both theoretical and empirical implications. Theoretically, it

more explicitly incorporates the concept of family lineage into the life-course model. Current applications of the life-course model have been heavily influenced by the nuclear family structure that is prevalent in modern societies. Research on SES exposure has largely focused on a participant's own life course within the nuclear family (i.e., from birth and childhood to adulthood). However, this focus on the nuclear family may have excluded the impacts of SES exposure that are felt beyond this setting. Parents' SES may not fully reflect the socioeconomic dis/advantages that have built up along the family lineage (15). People with similar parental SES might experience considerable heterogeneity in access to health resources, depending on their grandparents' social position (15). For instance, compared with parents from intergenerationally stable middle-class families, parents that have moved up into the middle class from poverty may have fewer

resources available for their children. Similarly, parents who have moved from the middle class to the lower class (the so-called "sunken middle class") are still better able to provide important health resources to their children than parents in second-generation lower-SES families (16).

A few intergenerational studies recently demonstrated the importance of family lineage for understanding the health consequences of SES. For instance, Schreier and Chen (17) found that grandparents' SES is inversely associated with their grandchildren's level of blood pressure and C-reactive protein. Another 3-generation study based on data collected from mothers and grandmothers of 987 singleton infants found that grandparents' poverty status predicts low birth weight for grandchildren, even after controlling for parental SES (18). These studies indicate that researchers need to cover more generations along the family lineage in order to fully evaluate the health impact of SES.

Empirically, a 3-generation study of the SES-obesity association represents an "upstream" approach to obesity, which identifies distal social risk factors before they cause measurable harm. Although the above-mentioned studies revealed the long-range health impact of grandparental SES, very little is known about its implications for obesity, which is now a distinct public health concern. By focusing on grandparental SES and its relationship to body mass index (BMI), we can explore the following questions: How far do the obesity impacts of SES travel along the family lineage? What intermediate factors help transmit those impacts across multiple generations? Finally, does grandparental SES predict grandchildren's BMI independent of the parents' SES? Answering these questions will help us make projections about the population that will be at risk in the next several decades and to design more effective strategies for mitigating the "generational curse" experienced by the socioeconomically disadvantaged population.

Using data on a nationally representative sample of children from the Child Development Supplement (CDS) of the Panel Study of Income Dynamics (PSID), a US prospective cohort study, I examined the association between chronic poverty experienced by grandparents and the developmental trajectories of BMI among their grandchildren during childhood and early adulthood. I focused on chronic poverty instead of transient poverty exposure, because the literature has suggested that the former is theoretically distinct from the latter and has a much greater impact on life outcomes (19–24). Moreover, because of the sex difference in the SES-obesity association documented in previous literature, I examined the association separately for male and female grandchildren. I hypothesized that grandchildren with chronically poor grandparents would have faster weight gain than grandchildren whose grandparents were not exposed to chronic poverty. Further, I expected to find a stronger pattern among granddaughters than among grandsons.

METHODS

Data

Analyses were based on an intergenerationally linked data set constructed from the PSID and 2 of its supplemental studies: the CDS and the Transition into Adulthood (TA) Study.

The PSID is an ongoing longitudinal study of a nationally representative sample of approximately 5,000 US households. Started in 1968, it followed the initial sample of households annually until 1997 and has followed them biannually since then. Patterned on a genealogical design, the PSID follows every individual born or adopted into the original sample families and therefore yields a continuously representative sample of children born into US families (25, 26).

In 1997, a total of 3,563 children aged 12 years or less from 2,394 PSID families were selected to create a nationally representative sample of children in that age range (known as CDS wave 1, or CDS I). The CDS children were followed up in 2002 when they were 5–<18 years of age (in CDS wave 2, or CDS II). In 2007, a third wave of CDS data (CDS III) were collected specifically for children between the ages of 10 and <18 years. All CDS children who turned 18 years of age were instead followed in the TA Study, which administered questionnaires in 2005, 2007, 2009, and 2011. Together, the CDS and the TA Study yielded 3–6 waves of data for each CDS child.

I linked the CDS-TA participants (third generation (G3)) with their parents (second generation (G2)) and grandparents (first generation (G1)) from the PSID core sample. CDS-TA participants who were offspring of the PSID immigrant sample were excluded because only limited information was collected for the immigrant sample. A total of 3,167 CDS-TA participants remained after the exclusion, among whom 3,023 were linked to a total of 5,324 G1 figures (879 paternal grandfathers, 1,141 paternal grandmothers, 1,387 maternal grandfathers, and 1,917 maternal grandmothers). Most participants were linked to only a patrilineal or matrilineal grandparent; only 99 participants were linked to both paternal and maternal grandparents.

Dependent variable: BMI trajectory among grandchildren

Grandchildren's BMI (weight (kg)/height (m)^2) was ascertained on the basis of body weight and height information collected in 3 waves of CDS studies (1997, 2002, and 2007) and 4 waves of TA studies (2005, 2007, 2009, and 2011). In each wave of the CDS, the body weights of all children aged 5 years or older were reported by the primary caregiver, and height was measured with a rafter square and tape measure by the interviewer. In each wave of the TA Study, information about body weight and height was self-reported by the respondents. Because of the age eligibility requirement for body weight and height data collection, each wave of the survey collected data for a different subset of the original 1997 sample. In total, more than 80% of all participants had data from at least 2 waves of data collection.

Independent variable: chronic poverty among grandparents

From 1968 to 1997, the PSID investigators measured previous-year poverty status annually for each sampled household. Poverty status in each year was coded as an indicator variable, with 1 indicating that the income-to-need ratio of

the respondent's household fell below 125% of the official US poverty threshold (0 otherwise). The 25% inflation of the poverty threshold has been generally adopted by studies using PSID data to accommodate the fact that the PSID has consistently found higher annual incomes than the US Census (27–29). Numbers of poverty spells, defined as any year in which the respondent's household fell below 125% of the official US poverty threshold, were calculated over the 30-year period from 1967 to 1996 for all linked G1 figures (range, 0–30). Any G3 participant linked to 1 or more G1 participants who had experienced 8 or more poverty spells during the 30-year study period was coded as having a chronically poor grandparent. It was rather arbitrary to set 8 as the cutpoint in coding the variable, so I experimented with a series of alternative cutpoints.

Control variables

G2 SES. Parental SES was captured by 2 variables: G2 poverty status and G2 education. Parents' poverty status was measured as the number of poverty spells experienced by the household before the child turned 10 years of age. Because of the small sample size for G3 participants whose parents had more than 1 poverty spell during the study period, the poverty status variable was dichotomously recoded such that 0 represented no poverty spells while 1 represented 1 or more poverty spells. Parental education was measured as the highest number of years of education attained by either of the 2 parents of the CDS child. The variable was then recoded into a dichotomous variable such that 1 represented a G2 education above high school and 0 represented a G2 education of high school or lower.

G2 BMI. Parental BMI was ascertained as the father's BMI if the chronic poverty data were from the patrilineal grandparent and as the mother's BMI if the chronic poverty measure was from the matrilineal grandparent. Information used for calculation of G2 BMI was collected in 1999. For G2 participants for whom the 1999 BMI measure was missing, BMI calculated from data collected in 2001 was used instead.

G3 demographic characteristics. The analyses controlled for 2 G3 demographic variables, including race (coded as "white," "black," or "other," with white being the reference category) and cohort (measured as the year of birth).

After exclusion of participants who were not linked with any grandparental figure and participants with missing data on any variables included in the final model, the final analytical sample consisted of 2,613 G3 youth (1,323 male, 1,290 female) nested within 1,719 G1 figures. A detailed description of the analytical data can be found in Table 1.

Table 1. Weighted Characteristics of a 3-Generation Sample of Participants From the Panel Study of Income Dynamics (1968–2011), Child Development Supplement (1997–2011), and Transition into Adulthood Study (1997–2011), United States

Variable	Total (n = 2,613)		Female Grandchildren (n = 1,290)		Male Grandchildren (n = 1,323)	
	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%
Child's BMI[a]						
1997	17.91 (3.74)		17.78 (3.89)		18.04 (3.58)	
2002	20.75 (5.35)		20.71 (5.39)		20.79 (5.31)	
2005	23.48 (4.16)		22.8 (4.38)		24.26 (3.72)	
2007	22.94 (5.80)		22.85 (5.86)		23.04 (5.73)	
2009	24.60 (4.78)		24.15 (4.98)		25.12 (4.49)	
2011	24.98 (5.21)		24.68 (5.62)		25.29 (4.72)	
Chronically poor G1 (≥8 poverty spells[b])		21.8		21.8		21.8
G2 poverty status (≥1 poverty spell)		11.1		11.2		11.0
G2 education above high school		63.1		62.8		63.3
G2 BMI	26.41 (5.54)		26.37 (5.36)		26.45 (5.72)	
G3 baseline (1997) age, years	6.26 (3.59)		6.28 (3.65)		6.24 (3.54)	
G3 birth year	1990 (3.73)		1990 (3.79)		1990 (3.68)	
G3 race						
White		76.8		77.4		76.2
Black		15.9		13.8		17.9
Other		7.3		8.8		5.9

Abbreviations: BMI, body mass index; G1, first generation (grandparents); G2, second generation (parents); G3, third generation (grandchildren); SD, standard deviation.

[a] Weight (kg)/height (m)^2.

[b] A poverty spell was defined as any year in which the respondent's household fell below 125% of the official US poverty threshold.

Statistical analysis

Considering the hierarchical structure of the data, I used the 3-level growth curve model and the STATA 12 (StataCorp LP, College Station, Texas) *xtmixed* procedure (30) to estimate the age-based BMI trajectories for the CDS children from 1997 to 2011. The analyses were conducted with G3 repeated measurements (i.e., within-G3 effects) forming the first level, grandchildren (i.e., between-G3 and within-G1 effects) forming the second level, and grandparents (i.e., between-G1 effects) forming the third level. The growth curve model allows us to examine how the intercepts and slopes of the BMI trajectories covary with the independent variables across individuals. It also allows us to obtain robust

Table 2. Estimates of Grandchildren's Body Mass Index[a] Trajectory (Growth Curve Model) According to Grandparental Chronic Poverty Status, Panel Study of Income Dynamics (1968–2011), Child Development Supplement (1997–2011), and Transition into Adulthood Study (1997–2011), United States[b]

| | Female Grandchildren (*n* = 1,290) | | | | Male Grandchildren (*n* = 1,323) | | | |
| | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
	β	95% CI	β	95% CI	β	95% CI	β	95% CI
	Fixed Effects							
Initial BMI status								
Intercept	22.31	21.89, 22.72	22.71	22.16, 23.26	22.98	22.60, 23.36	23.31	22.80, 23.82
≥8 G1 poverty spells[c,d]	0.50	−0.22, 1.22	0.25	−0.43, 0.93	−0.02	−0.69, 0.65	−0.21	−0.86, 0.44
≥1 G2 poverty spell[e]			0.02	−0.72, 0.76			−0.50	−1.18, 0.18
G2 education above high school[f]			−0.19	−0.75, 0.37			−0.08	−0.59, 0.43
G2 BMI (centered)			0.28	0.23, 0.32			0.21	0.17, 0.25
Linear BMI growth rate								
Intercept (age-centered)	0.45	0.39, 0.50	0.45	0.39, 0.52	0.56	0.52, 0.60	0.59	0.54, 0.64
≥8 G1 poverty spells[d]	0.12	0.05, 0.19	0.10	0.03, 0.17	0.03	−0.03, 0.09	0.02	−0.04, 0.08
≥1 G2 poverty spell[e]			0.06	−0.01, 0.13			0.01	−0.05, 0.07
G2 education above high school[f]			0.01	−0.04, 0.07			−0.03	−0.07, 0.02
G2 BMI (centered)			0.02	0.01, 0.02			0.01	0.01, 0.02
Quadratic BMI growth rate								
Intercept (age-centered[2])	−0.03	−0.04, −0.03	−0.03	−0.04, −0.03	−0.03	−0.03, −0.02	−0.03	−0.03, −0.02
Cubic BMI growth rate								
Intercept (age-centered[3])	0.00	0.00, 0.00	0.00	0.00, 0.00				
	Random-Effects Variance Components							
Level 3: between-G1 effects								
Linear slope	0.04	0.02, 0.06	0.04	0.02, 0.06	0.02	0.01, 0.04	0.01	0.00, 0.05
Initial status	4.10	2.33, 7.23	2.00	0.75, 5.32	4.38	2.77, 6.93	2.56	1.25, 5.27
Cov(linear slope, initial status)	0.29	0.12, 0.45	0.210	0.06, 0.36	0.14	0.01, 0.28	0.04	−0.09. 0.16
Level 2: between-G3 and within-G1 effects								
Linear slope	0.03	0.02, 0.06	0.03	0.01, 0.05	0.03	0.02, 0.06	0.03	0.02, 0.06
Initial status	15.42	13.14, 18.09	15.02	12.89, 17.50	12.36	10.52, 14.54	12.64	10.78, 14.83
Cov(linear slope, initial status)	0.60	0.43, 0.77	0.55	0.39, 0.71	0.34	0.20, 0.48	0.36	0.21, 0.50
Level 1: within-G3 effects	8.27	7.77, 8.81	8.26	7.75, 8.79	6.49	6.06, 6.94	6.49	6.07, 6.95
No. of G1 observations	848		848		871		871	
Bayesian Information Criterion	23,601.02		23,504.84		22,109.99		22,065.65	

Abbreviations: BMI, body mass index; CI, confidence interval; Cov, covariance; G1, first generation (grandparents); G2, second generation (parents); G3, third generation (grandchildren).

[a] Weight (kg)/height (m)^2.

[b] All models controlled for G3 race and birth cohort (centered). G2 BMI was also centered to facilitate parameter estimates. The quadratic and cubic terms for age were constrained to be fixed, because the log likelihood ratio tests showed that they did not vary significantly across individuals.

[c] A poverty spell was defined as any year in which the respondent's household fell below 125% of the official US poverty threshold.

[d] Reference group: <8 G1 poverty spells.

[e] Reference group: 0 G2 poverty spells.

[f] Reference group: G2 education of high school or lower.

standard error estimates for the G1-level variables by taking into account the higher-order clusters in the data (i.e., grandchildren nested within grandparents). Finally, it does not require the data to be balanced (i.e., for all youth to have the same number of data points). In the current analyses, age was centered at the grand mean (14 years) in order to facilitate interpretation of the estimated model intercept.

Unconditional growth curve models suggested that a quadratic model best fitted the BMI growth trajectories of the male G3 youth (i.e., grandsons), while a cubic model best fitted the BMI growth trajectories for female G3 youth (i.e., granddaughters). Therefore, I fitted quadratic models and cubic models separately for each of the male and female G3 groups in the conditional growth curve models. Model specification is shown in the Web Appendix (available at http://aje.oxfordjournals.org/). Web Figure 1 presents the observed BMI trajectories from a randomly selected sample (n = 200) to provide a flavor of what the observed BMI trajectories looked like.

RESULTS

Table 1 presents the weighted characteristics of the analytical sample by sex. The mean age at baseline (1997) was 6 years (standard deviation, 4). The majority of the sample (76.8%) was white. Twenty-two percent of the G3 sample had G1 figure(s) who were exposed to chronic poverty during the 30-year period from 1967 to 1996. Approximately 11% of the CDS-TA sample (grandchildren) had experienced at least 1 poverty spell before the age of 10 years. Web Table 1 presents a detailed description of the mean BMIs for male and female grandchildren by age and grandparents' chronic poverty status.

Table 2 shows the estimates from the conditional growth curve models on the BMI trajectories from 1997 to 2011. The first model for each sex group (model 1 for females and model 3 for males) controlled for G3 demographic characteristics (race and birth cohort), while the second model for each sex group (model 2 for females and model 4 for males) additionally controlled for G2 SES factors and G2 BMI.

Across all models, the significant positive coefficients for the linear term of age suggested that BMI grows with age, and the significant negative coefficients for the quadratic term of age suggested that such growth follows a convex downward-facing curvilinear pattern, with the rate of BMI increase declining with age. Grandparents' chronic poverty exposure was not significantly associated with the initial status of BMI trajectories.

Grandparents' chronic poverty exposure was significantly and positively associated with the linear growth rate of BMI for female grandchildren but not for male grandchildren. For females, the linear rate of BMI growth increased if the granddaughters came from a family with a chronically poor grandparent. Models 2 and 4 showed that, among G3 groups of both sexes, G2 poverty status and educational level were not significantly associated with either initial BMI status or the rate of BMI growth. G2 BMI was positively and significantly associated with the linear rates of BMI growth for both sexes. In the female G3 group, the association was somewhat attenuated, but this did not render nonsignificant the negative association found between the linear BMI slope and grandparents' chronic poverty exposure.

To test whether the results were robust to the coding for G1 chronic poverty, I fitted a series of models with grandparents' chronic poverty defined at an alternative cutpoint. The model results did not vary significantly as the cutpoint was set at any point between 6 and 10 poverty spells. Web Table 2 provides a detailed comparison of model results based on different ways of coding G1 chronic poverty.

Model-predicted trajectories and observed trajectories of BMI from age 6 years to age 26 years are plotted in Figure 1, which shows that female grandchildren (Figure 1A) with a
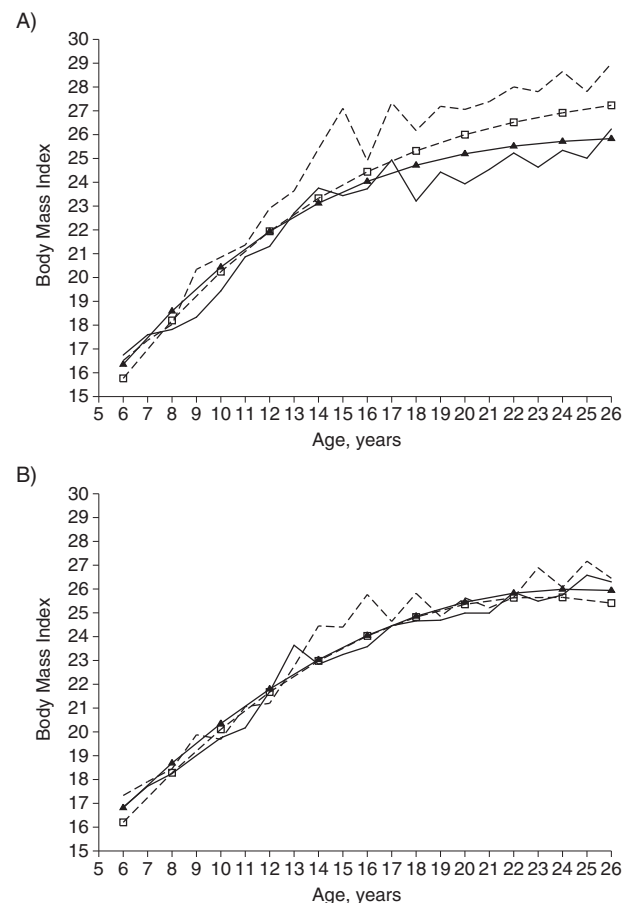

Figure 1. Predicted and observed body mass index (BMI) trajectories among female (A) and male (B) grandchildren (third generation (G3)) according to grandparental (first generation (G1)) chronic poverty status, Panel Study of Income Dynamics (1968–2011), Child Development Supplement (1997–2011), and Transition into Adulthood Study (1997–2011). BMI was calculated as weight (kg)/height (m)². The predicted BMI trajectory for female grandchildren was based on model 2, while that for male grandchildren was based on model 4. Both models controlled for race, birth year, parental poverty exposure, parental education, and parental BMI. The solid black line represents the mean observed BMI trajectory for grandchildren whose grandparents were not chronically poor; the dashed line represents the mean observed BMI trajectory for grandchildren whose grandparents were chronically poor; the solid line with black triangles represents the predicted BMI trajectory for grandchildren whose grandparents were not chronically poor; and the dashed line with white squares represents the predicted BMI trajectory for grandchildren whose grandparents were chronically poor.

chronically poor grandparent had a faster BMI growth rate than those whose grandparents were not chronically poor. However, Figure 1B shows that male grandchildren were not significantly different in terms of BMI growth rates based on their grandparent's chronic poverty status. Note that the predicted trajectories are sandwiched between the observed trajectories (which is more obvious in Figure 1A). This is due to the fact that the variances of the intercept and slope were partly explained by additional variables included in the model.

DISCUSSION

Few studies have explicitly examined the intergenerational SES-BMI association in a 3-generation model. Accordingly, little is known about how family background beyond the nuclear family setting affects one's body weight growth, as well as how far the impact of SES could reach along the family line. Results from the present study showed that grandparents' lifetime exposure to chronic poverty was associated with faster BMI increase for female grandchildren but not for male grandchildren. In females, this association remained significant even after controlling for parents' education, poverty exposure, and BMI. However, the attenuated coefficient estimate for grandparents' chronic poverty after inclusion of the G2 controls suggests that its association with female grandchildren's BMI growth rate was only partially explained by parental BMI.

These findings echo those of a handful of other studies suggesting that low SES can have a negative health impact across 3 generations (17, 18). The gendered pattern of the intergenerational SES-BMI association is consistent with previous prospective studies (31–34). This study also supports previous findings about a sociobiological pathway for the intergenerational transmission of health inequality, whereby grandparents' SES could have a negative imprint on grandchildren's health through parents' health status (35).

The remaining unexplained association between grandparents' chronic poverty and female grandchildren's BMI trajectories could possibly be attributed to some mediating mechanisms that were not specified in the model. Based on the previous literature, several important mechanisms can be suggested for future study: ecological mediators, social/psychological mediators, and biological mediators.

Ecologically, exposure to the obesogenic environment associated with poverty can be passed down to successive generations. Studies on the intergenerational transmission of neighborhood poverty found not only that the chronically poor tend to live in neighborhoods with a high concentration of poverty but also that their offspring have limited mobility out of such neighborhoods (36, 37). The inherited obesogenic environment would set the individual on a disadvantaged trajectory of weight gain.

Socially and psychologically, stresses associated with chronic poverty could trigger a chain of risk events within the family system, such as marital conflict, risky health behaviors, impaired quality of intergenerational relationships, and the social, psychological, and/or biological maladaptation of the next generation (38). Through modeling and familial socialization, this process can repeat itself in subsequent gener-

ations, accumulating disadvantages that greatly increase the risk of obesity for later generations.

Biologically, long-term exposure to a poverty-associated obesogenic environment may lead to metabolic adaptations that produce epigenetic phenotypes that are more susceptible to obesity, such as insulin resistance and leptin resistance (39). The adaptive phenotypes are inheritable by subsequent generations and therefore could put them onto risky trajectories of weight gain (40).

Regardless of what the potential mechanism(s) might be, the findings of this study point to the limitations of the current life-course model, with its focus on nuclear families. To fully evaluate the health impact of economic dis/advantages, future life-course studies should consider the social positions of family members beyond the nuclear family setting. Two recent social trends make it even more urgent to do so. First, the multigenerational "linked lives" become longer as the population ages (41). Second, the number of multigenerational households has increased in the United States over the past 3 decades; in 2012, the number of people living in multigenerational households reached 57 million (42). Together these trends indicate greater grandparental involvement in grandchildren's lives and greater social influences of the family lineage beyond the nuclear family setting, especially for children whose parents experience economic difficulties or marriage disruption.

This study informs interventional strategies by revealing the longevity and the gendered pattern of the "generational curse" of chronic poverty. These findings not only help us to identify the population currently at risk of obesity but also enable us to project the population that will be at risk in the future: namely, descendants or future descendants of persons currently living in chronic poverty. Furthermore, this study implies that future interventions should direct more resources toward those youth whose parents are not poor enough to qualify for government assistance but whose grandparents are chronically poor. According to the PSID, 18% of youth with parents that have never lived below the poverty line have a chronically poor grandparent. Although these youth face a higher risk of obesity, they can barely receive any assistance from current governmental programs, most of which allocate funds on the basis of parents' income level.

The major strength of this study is that it was based on a nationally representative sample with intergenerationally linked poverty data that were prospectively measured over 30 years. The prospective design enabled me to avoid the biased estimation that is often found with recalled SES data (43, 44).

Several limitations, however, should be mentioned. First, I had only a limited number of control variables included in the model. The lack of data on other potentially mediating factors mentioned above prevented me from examining these possible links between grandparents' poverty and grandchildren's BMI growth pattern. Second, the analyses excluded the immigrant sample because of limited information for this subsample. Therefore, the generalizability of this study to immigrants is limited. Third, the last 4 waves of BMI data (collected in the TA Study) were based on respondents' self-reports, which tend to be lower than technician-measured BMI due to underreporting of weight and overreporting of height (45). However, self-reported BMI is highly correlated

with technician-measured BMI and is generally accepted as a valid biomarker for diseases in epidemiologic studies (46). Further, previous studies have suggested that people of low SES are more likely to underreport BMI than people of high SES (47); therefore, the gap I found between the chronically poor G1 group and the non-chronically poor G1 group is likely to have been an underestimation.

Finally, the PSID did not collect information about women's pregnancy status during the interview. Thus, I could not follow the common practice of excluding from the model maternal BMI values that were ascertained during pregnancy. However, even with the presence of potential measurement error, parental BMI was still significantly associated with the BMI intercept and linear slope. In reality, the measurement error due to women's pregnancy status may be expected to have been small, and removing such error would be unlikely to greatly alter the association between our key independent variable and the intercept/slope of the BMI trajectories.

With all of its limitations, this study is among the first to have evaluated the SES-BMI association within a 3-generation framework. Its findings suggest the importance of moving our attention beyond the nuclear family, and hopefully it will trigger more studies investigating the intermediate links that explain the extremely long-term intergenerational health impact of socioeconomic disadvantage.

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