

# Childhood Socioeconomic Disadvantage and Prediabetes and Diabetes in Later Life: A Study of Biopsychosocial Pathways

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**Objective:** We examined the relationship between childhood socioeconomic status (SES) and gluoregulation in later life and used a life-course framework to examine critical periods and underlying pathways. **Methods:** Data came from the Midlife in the US (MIDUS) national study ( $n = 895$ ). Childhood SES indicators retrospectively reported at MIDUS I were used to create a childhood SES disadvantage index. Adult SES disadvantage and potential pathways were measured at MIDUS I and included waist circumference, depressive symptoms, and physical activity. Glucose and hemoglobin A1c, measured approximately 9 to 10 years later at MIDUS II, were used to create the ordinal outcome measure (no diabetes/prediabetes/diabetes). **Results:** Childhood SES disadvantage predicted increased odds of prediabetes and diabetes net of age, sex, race, and smoking (odds ratio = 1.11, 95% confidence interval = 1.01–1.22). Childhood SES disadvantage predicted adult SES disadvantage ( $\beta = .26, p = .001$ ) and the three key mediators: waist circumference ( $\beta = 0.10, p = .002$ ), physical activity ( $\beta = -0.11, p = .001$ ), and depressive symptoms ( $\beta = 0.07, p = .072$ ). When childhood and adult SES disadvantage were in the same model, only adult SES predicted gluoregulation (odds ratio = 1.07, 95% confidence interval = 1.01–1.13). The SES disadvantage measures were no longer significantly associated with gluoregulation after including waist circumference, physical activity, and depressive symptoms, all of which were significant predictors of gluoregulation. **Conclusions:** The consequences of childhood SES disadvantage are complex and include both critical period and pathway effects. The lack of a direct effect of childhood SES on gluoregulation does not negate the importance of early environment but suggests that early-life socioeconomic factors propel unequal life-course trajectories that ultimately influence health. **Key words:** childhood disadvantage, health disparities, socioeconomic status, diabetes, obesity, depressive symptoms.

SES = socioeconomic status; MIDUS = Midlife in the US.

## INTRODUCTION

More than one-third of American adults were obese in 2009 to 2010, up from 5% to 6% only three decades ago (1). Dramatic increases in the rates of Type 2 diabetes have paralleled the increases in obesity: diabetes affects 8.3% of the US population and 35% are at an increased risk for developing Type 2 diabetes due to prediabetes (2). The economic burden associated with Type 2 diabetes in the United States is staggering: the total cost of diagnosed diabetes in 2012 was US \$245 billion, and care for people with diagnosed diabetes accounted for more than 1 in 5 health care dollars (3).

Type 2 diabetes is a multifactorial disease with established risk factors such as obesity and physical inactivity that have traditionally been the main targets of diabetes prevention efforts. However, the social determinants of Type 2 diabetes and its risk factors are increasingly recognized: low socioeconomic status (SES), indexed by a variety of indicators across the life course, consistently predicts higher risk for obesity, the metabolic syndrome, and Type 2 diabetes (4–11). Understanding how the processes of social inequality affect chronic disease requires a life-course perspective, starting with early-life exposures (12). A life-course approach integrates biological, behavioral, and psychosocial pathways and, as such, is well suited to the study of complex disorders such as Type 2 diabetes.

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Within the life-course framework, we used the *critical period* model to investigate whether childhood SES disadvantage predicted health outcomes independently from adult SES and the *pathway* model to focus on mechanisms that linked childhood SES disadvantage to prediabetes and diabetes.

## The Critical Period Model

The critical period model suggests a biological imprinting mechanism and posits that early-life SES has long-lasting effects on biological and behavioral systems, and these effects are largely irreversible and permanent, albeit potentially modifiable under certain conditions (12). The critical period model emphasizes the importance of a specific time window during which exposures increase the risk of later disease. However, time windows have less specific boundaries for such complex and chronic exposures as socioeconomic disadvantage. Because childhood deprivation is a long-term exposure, it likely covers more than one critical period. The measures of early-life SES used in our study are comprehensive and comprise a long time span up to late adolescence. Consistent with the critical period model, some research suggests that low childhood SES is associated with incidence of Type 2 diabetes and metabolic abnormalities, and the relationship is attenuated but not eliminated with the inclusion of later exposures (8,11,13). Thus, we hypothesize that childhood SES disadvantage has an enduring impact on key biopsychosocial mediators and later-life gluoregulation that is unique to early-life adversity and independent of the impact of adult SES disadvantage.

The pathway model suggests that early-life environment is consequential mainly because it shapes life-course trajectories of beneficial or harmful exposures and experiences (12). The primary focus in this model is not the direct effect of early-life SES on health but the pathways connecting family background to health in later life. We focus on key biopsychosocial pathways that have been theoretically and empirically linked

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to both childhood SES and glucoregulation: adiposity, depressive symptoms, and vigorous physical activity.

Adiposity is an important pathway linking socioeconomic family background to later-life health: low childhood SES predicts higher obesity risk in adulthood (11), and obesity is etiologically implicated in diabetes (14,15). Furthermore, individuals from lower-SES families experience more depressive symptoms in later life than do their peers with higher-SES family background (16,17), and depressive symptoms have been implicated in cardiometabolic health (18–21). Similarly, physical inactivity is also patterned by early-life SES (22) and is widely accepted as one of the key risk factors for diabetes (15). Based on the pathway model, we hypothesize that early-life SES disadvantage is associated with increased odds of prediabetes and diabetes indirectly by shaping adult adiposity, depressive symptoms, and physical activity. We will elucidate whether childhood disadvantage affects biobehavioral mechanisms and glucoregulation independent of adult SES.

### METHODS

#### Sample

Data come from the original Midlife in the US (MIDUS) study and its longitudinal follow-up MIDUS II. Begun in 1995/1996, the overarching objective of MIDUS was to investigate the role of behavioral, psychological, and social factors in physical and mental health. All eligible participants were noninstitutionalized, English-speaking adults in the coterminous United States, initially 25 to 74 years of age. A national sample of 3487 individuals were surveyed via telephone using random digit dialing, with 3034 of the respondents completing an additional mail survey. Samples of siblings of randomly dialed respondents ( $n = 950$ ) and twins ( $n = 1914$ ) were also included in the baseline cohort. Approximately 9 to 10 years later, respondents were recontacted and invited to participate in MIDUS II, and the original cohort was resurveyed via telephone ( $n = 4474$ ) and mail ( $n = 3637$ ). The longitudinal retention rate was 75%, adjusted for mortality (23). One objective of MIDUS II was to extend the scientific scope of the study by adding comprehensive biological assessments on a subsample of respondents who had completed a telephone interview and self-administered questionnaires. Forty-three percent of the invited MIDUS II respondents participated in the Biomarker Substudy. Participants in the Biomarker Substudy were not significantly different from the main MIDUS II sample on most demographic variables (age, sex, race, marital status, income variables), although they were significantly more educated than the main sample (24). The MIDUS study was approved by the institutional review boards at UCLA, University of Wisconsin-Madison, Georgetown University, and Brandeis University.

The current study used data from the Biomarker Substudy. Of the 1054 participants from the baseline MIDUS cohort who participated in the biological data collection in MIDUS II, 159 cases were excluded because of partially missing data on any variable in the analysis, reporting diabetes at baseline, or race other than black or white, resulting in 895 participants with complete data. The variables with the most missing cases were waist circumference (missing  $n = 85$ ), race (missing  $n = 45$ ), and vigorous physical activity (missing  $n = 36$ ). Within-person mean substitution was used in 16 cases of missing data on childhood SES disadvantage where data were available on two of three variables used in calculating the childhood SES index and in 25 cases of missing data on adult SES disadvantage where data were available on 4 of 5 variables used in calculating the adult SES index.

#### Measures

Information on childhood SES disadvantage was collected retrospectively at MIDUS I and computed by summing values on three indicators: financial level growing up (2, worse off than others; 1, about the same as others; 0, better off than others), highest level of parental education (2, less than high school;

1, high school/GED; 0, some college or higher), and childhood welfare status (2, ever on welfare; 0, never on welfare) (25,26). MIDUS I adult SES disadvantage score was computed by summing values on five indicators: education level (2, high school/GED or less; 1, some college/associate arts degree; 0, bachelor's degree or higher), family size-adjusted income to poverty ratio (2, <300%; 1, 300%–599%; 0, ≥600%), current financial situation (2, worst possible; 1, average; 0, best possible), availability of money to meet basic needs (2, not enough; 1, just enough; 0, more than enough), and difficulty level of paying bills (2, very or somewhat difficult; 1, not very difficult; 0, not at all difficult) (25).

All mediators were measured during MIDUS I. Waist circumference was self-reported by respondents using a tape measure and diagram provided by the MIDUS staff. To test the reliability of the self-reported measure of waist circumference, we looked at the correlation between self-reported waist circumference at MIDUS II and waist circumference measured by MIDUS II staff in the same subset of participants used in our analyses, and the high correlation ( $r = 0.82$ ) suggested that the self-reported measure was a reasonable option. The underlying rationales for choosing waist circumference as the key measure of adiposity were, first, that stress activation has been implicated in the pathogenesis of abdominal obesity (27,28) and, second, that waist circumference is a stronger predictor of diabetes than body mass index (29). The depression assessment was defined according to the criteria in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (30). To assess depressive symptoms, participants were asked whether, during the previous 12 months, there was a period when they felt “sad, blue, or depressed” or “lost interest in most things” for 2 weeks or longer and lasted all day or most of the day. Participants who responded “yes” were asked follow-up questions, and sample items included “during two weeks in past 12 months, when you felt sad, blue, or depressed, did you feel down on yourself, no good, or worthless” and “during two weeks in past 12 months, when you lost interest in most things, did you feel more tired out or low on energy than is usual?” Depressive symptoms score was created by summing the number of affirmative responses. Vigorous activity was assessed by two questions (one for each season): “During the winter/summer, how often do you engage in vigorous physical activity (e.g., running or lifting heavy objects) long enough to work up a sweat?” Response choices were “several times a week or more,” “about once a week,” “several times a month,” “about once a month,” “less than once a month,” and “never.” The responses to each question were coded so that they represented estimated numbers of exercise sessions per month, and the two estimates were averaged to create a measure of vigorous activity. Demographic covariates included age (in years), sex (male or female), and race/ethnicity (white or black). Lifetime smoking history was assessed by the question: “Have you ever smoked cigarettes regularly—that is, at least a few cigarettes every day?” and coded as yes or no.

Fasting glucose and hemoglobin A1c (HbA1c) samples were obtained during an overnight stay in a General Clinical Research Center during MIDUS II. Fasting glucose was measured via an enzymatic assay photometrically on an automated analyzer (Roche Modular Analytics P). The HbA1c assay was a colorimetric total hemoglobin determination combined with an immunoturbidometric HbA1c assay, carried out using a Cobas Integra Systems instrument (Roche Diagnostics) (31). Criteria from the American Diabetes Association were used to define presence of prediabetes (HbA1c between 5.7% and 6.5% or glucose between 100 and 126 mg/dl, and NOT taking diabetes medications) and diabetes (HbA1c >6.5%, fasting glucose >126 mg/dl, or taking medications that lower glucose levels such as metformin) (15). The dependent variable was an ordered categorical variable with three levels: no diabetes, prediabetes, and diabetes.

#### Statistical Analyses

First, descriptive statistics were generated. Means, standard deviations, and ranges for all continuous variables and proportions for categorical variables were examined. A path model was built incrementally to test the associations between childhood SES disadvantage and glucoregulation via adult SES disadvantage, obesity, physical activity, and depressive symptoms. Multiple linear regressions were used in all analyses with continuous

TABLE 1. Descriptive Statistics for All Analytical Variables ( $n = 895$ )

	$n$ (%)	M (SD)	Range
SES disadvantage score			
Childhood SES score		1.83 (1.40)	0–6
Adult SES score		4.5 (2.59)	0–10
Biopsychosocial mediators			
Waist circumference		35.06 (5.37)	22–57
Depressive symptoms		0.74 (1.89)	0–7
Vigorous physical activity		6.77 (5.20)	0–13.5
Demographic and health covariates			
Age		46.25 (11.88)	25–74
Sex (1 = male)	406 (45.4)		
Race (1 = white)	869 (97)		
Ever smoked (1 = yes)	398 (44.5)		
Glucoregulation			
Normoglycemia	492 (55)		
Prediabetes	293 (32.7)		
Diabetes	110 (12.3)		

SES = socioeconomic status; M = mean; SD = standard deviation.

outcomes. Because glucoregulation, our focal outcome measure, is an ordered categorical variable, ordered logistic regression using a maximum likelihood estimator was estimated in all analyses that included glucoregulation. Ordered logistic regression based on the proportional odds model is a widely used technique that assumes that the effect of the independent variables is constant across all ordered categories of outcome variables. When the proportional odds assumption is met, the slope estimate in the regression model is interpreted to be constant across the entire range of the outcome. All analyses were conducted using MPlus 7.11 and IBM SPSS 20.

## RESULTS

Descriptive statistics for childhood SES disadvantage, the biopsychosocial mediators, demographic and health covariates, and glucoregulation are presented in Table 1. Slightly more than half of the sample had normal glycemic levels, whereas approximately 33% had prediabetes and 12% had diabetes. The sample was predominantly white, and only 3% were African American. Average childhood SES disadvantage score was moderate (mean = 1.83; range, 0–6) and fell on the

lower end of the scale of each of the three subcomponents; however, there was substantial variability in the range of scores.

### Childhood SES Disadvantage and Diabetes-Related Measures: Independence of Adult SES (the Critical Period Model)

Table 2 displays the incremental models that were estimated to evaluate the associations of childhood SES with proposed mediators. Childhood SES disadvantage predicted higher waist circumference ( $\beta = 0.10, p = .002$ ) and lower physical activity ( $\beta = -0.11, p = .001$ ) but was only marginally associated with higher depressive symptoms ( $\beta = 0.07, p = .072$ ). Furthermore, childhood SES disadvantage predicted higher adult SES disadvantage ( $\beta = 0.26, p = .001$ ). The relationships between childhood SES disadvantage and higher waist circumference ( $\beta = 0.07, p = .042$ ) and between childhood SES disadvantage and lower levels of vigorous physical activity ( $\beta = -0.08, p = .015$ ) were attenuated but remained significant after accounting for the influence of adult SES disadvantage, suggesting critical period effects of childhood SES disadvantage on at least some of the key risk factors for prediabetes and diabetes.

Childhood SES disadvantage predicted increased odds of prediabetes and diabetes net of age, sex, race, and smoking (odds ratio [OR] = 1.11, 95% confidence interval [CI] = 1.01–1.22) (Table 3). When childhood and adult SES disadvantage were in the same model, only adult SES disadvantage predicted glucoregulation (OR = 1.07, 95% CI = 1.01–1.13). In the fully adjusted model that included both SES disadvantage measures and all mediators, glucoregulation was significantly predicted by waist circumference (OR = 1.10, 95% CI = 1.07–1.14), vigorous physical activity (OR = 0.97, 95% CI = 0.94–0.99), and depressive symptoms (OR = 1.08, 95% CI = 1.01–1.16), but not by either SES measure (see Table 3, Model 7).

### Mediating Pathways Involved in the Association of Childhood SES Disadvantage with Prediabetes and Diabetes in Adulthood

Further estimates are presented in Table 4 and include the total effect, total direct effect, total indirect effect, and the indirect effects via specific mediating pathways (Fig. 1). The

TABLE 2. Path Model Results Predicting Mediators

Outcomes	Adult SES	Adult WC Models			Adult PA Models			Adult Depression Models		
	Model 1	2a	2b	2c	3a	3b	3c	4a	4b	4c
Childhood SES, $\beta$ (SE)	<b>0.26***</b> (0.03)	<b>0.10**</b> (0.03)		<b>0.07*</b> (0.03)	<b>-0.11**</b> (.03)		<b>-0.08*</b> (.03)	0.07 <sup>†</sup> (.04)		0.03 (.04)
Adult SES, $\beta$ (SE)			<b>0.14***</b> (0.03)	<b>0.13***</b> (0.03)		<b>-0.11**</b> (0.03)	<b>-0.09**</b> (0.03)		<b>0.16***</b> (0.04)	<b>0.15***</b> (0.04)
$R^2$	0.13	0.26	0.27	0.27	0.07	0.07	0.08	0.04	0.06	0.06

SES = socioeconomic status; SE = standard error; WC = waist circumference; PA = physical activity.

Standardized effects are shown. All models adjust for sex, age, race, and lifetime smoking history. Effects significant at  $p < .05$  are bolded.

<sup>†</sup>  $p < .10$ , \*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$ .

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TABLE 3. Path Model Results Predicting Prediabetes/Diabetes

	Model 5, OR (95% CI)	Model 6, OR (95% CI)	Model 7, OR (95% CI)
Childhood SES	<b>1.11* (1.01–1.22)</b>	1.07 (0.97–1.18)	1.03 (0.93–1.14)
Adult SES		<b>1.07* (1.01–1.13)</b>	1.03 (0.97–1.09)
Adult WC			<b>1.10*** (1.07–1.14)</b>
Adult PA			<b>0.97* (0.94–0.99)</b>
Adult Dep			<b>1.08* (1.01–1.16)</b>
Pseudo R <sup>2</sup>	0.12	0.13	0.19

SES = socioeconomic status; WC = waist circumference; PA = physical activity, Dep = depressive symptoms; OR = odds ratio; CI = confidence interval. All models adjust for sex, age, race, and lifetime smoking history. Effects significant at  $p < .05$  are bolded.

\*  $p < .05$ , \*\*\*  $p < .001$ .

total controlled effect of early-life SES was  $\beta = 0.097$  ( $p < .05$ ), net of sex, race, age, and smoking. The total indirect effect of childhood disadvantage through adult SES disadvantage, obesity, physical activity, and depressive symptoms was  $\beta = 0.070$  ( $p < .001$ ). Childhood SES disadvantage predicted gluco-regulation via waist circumference ( $\beta_{\text{indirect}} = 0.025$ ,  $p = .041$ ) and marginally via vigorous physical activity ( $\beta_{\text{indirect}} = 0.010$ ,  $p = .096$ ), but there was no evidence to support a pathway through depressive symptoms ( $p = .42$ ). Similarly, significant indirect pathways that included both childhood and adult SES disadvantage were conveyed via higher waist circumference ( $\beta_{\text{indirect}} = 0.012$ ,  $p < .001$ ) and marginally through depressive symptoms and vigorous activity ( $\beta_{\text{indirect}} = 0.004$  [ $p = .067$ ] and  $\beta_{\text{indirect}} = 0.003$  [ $p = .091$ ]).

## DISCUSSION

The prevalence of Type 2 diabetes has risen steadily over the last three decades and is associated with huge economic costs and human suffering. Socioeconomic disparities in Type 2 diabetes and its risk factors are well documented. Using longitudinal data from MIDUS, we tested life-course mechanisms linking early-life SES disadvantage and gluco-regulation

in midlife and later life. The overarching goal of our study was to look beyond individual behaviors assessed close in time to diagnosis and explore the long-term relationship between childhood SES disadvantage and adult gluco-regulation, and further, examine whether three of the key risk factors—waist circumference, depressive symptoms, and low levels of vigorous physical activity—were components of the pathway between early-life SES and gluco-regulation in later life. Importantly, we investigated whether the effect of childhood SES disadvantage was independent of the role of adult SES. We found support for the critical period model as well as for indirect effects. Childhood SES disadvantage predicted higher waist circumference and less physical activity net of adult SES, suggesting that there were critical periods for the effects of childhood SES disadvantage on some risk factors for gluco-regulation that were independent from adult SES. However, once the key mediators were in the model, none of the SES measures were significantly linked to gluco-regulation. Our results also supported a pathway model, with abdominal obesity emerging as a particularly strong mediator of the effect of early-life SES disadvantage on gluco-regulation. Thus, the effects of childhood SES disadvantage on adult gluco-regulation are complex, including effects of a critical period of childhood SES on waist

TABLE 4. The Indirect Effects of Childhood SES Disadvantage on Gluoregulation in Later Life Via Specific Mediating Pathways ( $n = 895$ )

Mediating Pathways	Indirect Effect	Indirect/Total
Childhood SES → Adult SES → no/pre/diabetes	0.013 (0.014)	0.134
Childhood SES → waist circumference → no/pre/diabetes	0.025* (0.012)	0.258
Childhood SES → Adult SES → Waist circumference → no/pre/diabetes	0.012*** (0.004)	0.124
Childhood SES → vigorous activity → no/pre/diabetes	0.010 <sup>#</sup> (0.006)	0.103
Childhood SES → Adult SES → vigorous activity → no/pre/diabetes	0.003 <sup>#</sup> (0.002)	0.031
Childhood SES → depression → no/pre/diabetes	0.003 (0.004)	0.031
Childhood SES → adult SES → depression → no/pre/diabetes	0.004 <sup>#</sup> (0.002)	0.041
Total effect: $\beta = 0.097^*$ (0.049)		
Total indirect effect: $\beta = 0.070^{***}$ (.020)		
Total direct effect: $\beta = 0.027$ (0.052)		

All paths in Table 3 are illustrated in Figure 1. Effects are presented as standardized logged odds ratios ( $\beta$ ) with standard errors in parentheses. All models are adjusted for age, race, sex, and smoking history.

<sup>#</sup> $p < .10$ , \* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .



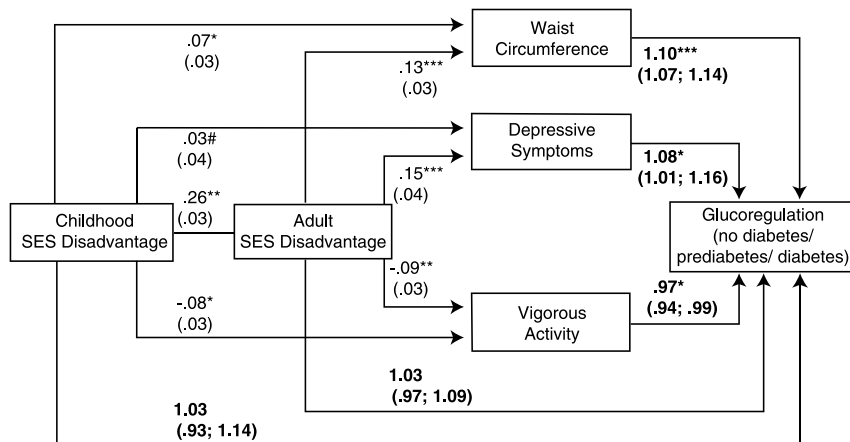


Figure 1. Results for mediational model linking childhood SES disadvantage and glucoregulation in later life ( $n = 895$ ). Our fully adjusted final model (Model 7, Table 2) is displayed by bolded paths leading to glucoregulation and shows ORs and 95% CI for ORs. All other paths lead to one of the continuous mediators (Models 1–4, Table 2) and show standardized path coefficients and standard errors. All models except Childhood SES→Adult SES include both SES measures as predictors and are further adjusted for age, race, sex, and smoking. OR = odds ratio; CI = confidence interval; SES = socioeconomic status.  $^{\#} p < .10$ ,  $^* p < .05$ ,  $^{**} p < .01$ ,  $^{***} p < .001$ .

circumference and physical activity, as well as indirect effects through adult SES and waist circumference, physical activity, and depressive symptoms. Taken together, these findings suggest that the lack of a direct effect of childhood SES on glucoregulation in our study does not negate the importance of early family environment but rather illustrates the idea that early-life social and economic factors are a powerful force propelling life-course trajectories of SES disadvantage and unhealthy behaviors in adulthood that ultimately influence health outcomes.

Previous research that has tested life-course models with respect to early-life SES and Type 2 diabetes has yielded mixed results, with some studies showing that the effects of childhood SES persisted over the life course and were generally independent from current SES, particularly for women (8,13,32), whereas other studies showing that childhood SES was not an independent predictor of diabetes in men or women (33,34). This inconsistency is not surprising given the differences across studies in countries of origin, adjustment for risk factors, years of follow-up, and measures of early-life SES and metabolic health. It is also important to note that some studies considered whether the effect of early-life SES persisted net of adult SES but did not include measures of adult adiposity, mental health, and physical activity. Consistent with our findings, studies that included adult SES as well as adult adiposity and other risk factors showed that the effect of early-life SES was explained largely (35) or at least partially (4,13) by these factors, although one study did not find evidence that the obesity mediated the relationship between childhood SES and diabetes in men or women (36).

One limitation of the current analyses is the lack of longitudinal data on childhood obesity, physical activity, depression, and glucoregulation, which prevents us from examining the longitudinal patterns that would best inform our understanding of the temporal and directional patterns of associations between SES and glucoregulation across the life course and could provide further insights into the unique contributions of the critical period and pathway models. Moreover, circumstances in later

life can attenuate or aggravate the effect of childhood SES disadvantage, and an important direction for future research is exploring life-course factors that moderate the effect of early-life deprivation on subsequent glucose metabolism. The proposed mediators were assessed at the same time point as adult SES disadvantage, which limits our ability to claim directionality of effects. Furthermore, these mediators have familial influences that may confound the estimates of childhood SES. Another potential limitation that is not unique to MIDUS is the retrospective and self-reported assessment of childhood SES. The multi-indicator SES disadvantage composites may provide a comprehensive assessment of SES disadvantage and have also already been linked to allostatic load (25) and bone strength (26), but the composite measures are broad and more research on their construct validity is needed. Furthermore, our physical activity and smoking measures are based on limited questions and provide somewhat crude assessments of these factors. Our sample composed primarily of white participants, and it is important for future research to look at diverse populations, as minority groups, especially African Americans, may derive fewer health benefits from higher SES compared with white adults (37).

Our final set of limitations pertains to our measures of diabetes: although we excluded participants who self-reported diabetes at MIDUS I, we do not have biological data that could help ascertain glycemic status at MIDUS I. Furthermore, our analyses were modeled to capture known risk influences for Type 2 diabetes, but we did not have information on whether participants in the diabetes category had Type 1 or Type 2 diabetes. Given that approximately 90% to 95% of people with diabetes have Type 2 diabetes (15), our results are not significantly affected by this imprecision. Despite these caveats, the findings that childhood SES disadvantage was associated with higher odds of prediabetes and diabetes and identifying the role of adult SES and other key mediators are novel and help advance understanding of the multifactorial roots of Type 2 diabetes. A notable strength of our study is that glycemic status

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was ascertained using biomarkers and allowed for investigating not only the odds of diabetes but also prediabetes, a clinically relevant outcome that is an important step on the progression from normoglycemia to Type 2 diabetes (15,38).

Continuing to elucidate the various processes that underlie glucoregulation is critical for developing effective preventive efforts that target prediabetes and Type 2 diabetes. Our central finding that early SES disadvantage propels individuals on unhealthy trajectories in adulthood suggests that policies addressing socioeconomic inequality among children may be an important route to alleviating socioeconomic health disparities in later life. Ultimately, successful prevention of Type 2 diabetes will depend on understanding its preclinical progression, both in terms of identifying predisease pathways to morbidity and how they are contoured by antecedent factors following from one's socioeconomic standing and psychosocial and behavioral strengths and vulnerabilities.

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