Childhood Socioeconomic Position, Gender, Adult Body Mass Index, and Incidence of Type 2 Diabetes Mellitus Over 34 Years in the Alameda County Study

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Objectives. We examined the association between childhood socioeconomic position and incidence of type 2 diabetes and the effects of gender and adult body mass index (BMI).

Methods. We studied 5913 participants in the Alameda County Study from 1965 to 1999 who were diabetes free at baseline (1965). Cox proportional hazards models estimated diabetes risk associated with childhood socioeconomic position and combined childhood socioeconomic position–adult BMI categories in pooled and gender-stratified samples. Demographic confounders and potential pathway components (physical inactivity, smoking, alcohol consumption, hypertension, depression, health care access) were included as covariates.

Results. Low childhood socioeconomic position was associated with excess diabetes risk, especially among women. Race and body composition accounted for some of this excess risk. The association between childhood socioeconomic position and diabetes incidence differed by adult BMI category in the pooled and women-only groups. Adjustment for race and behaviors attenuated the risk attributable to low childhood socioeconomic position among the obese group only.

Conclusions. Childhood socioeconomic position was a robust predictor of incident diabetes, especially among women. A cumulative risk effect was observed for both childhood socioeconomic position and adult BMI, especially among women. (*Am J Public Health.* 2008;98:1486–1494. doi:10.2105/AJPH.2007.123653)

In recent years, much effort has gone into characterizing biological and social exposures during gestation and childhood that may lead to adult chronic diseases. Childhood socioeconomic disadvantage has been associated with mortality[1–4] and several adult physical[5–7] and mental health[5,7–9] outcomes.

Studies investigating the relationship between childhood socioeconomic disadvantage and diabetes have shown inconsistent results. Childhood socioeconomic position (SEP) was linked to prevalent type 2 diabetes,[10–14] insulin resistance,[15] higher glucose levels,[16,17] and metabolic syndrome[18,19] in some studies, yet showed no association with impaired glucose tolerance[20,21] or metabolic syndrome[22] in others. Three studies investigated the association between childhood SEP and incident diabetes in adulthood and reported either modest[11,23] or no effects.[12]

Although the evidence thus far is insufficient to establish a causal link between childhood SEP and incident type 2 diabetes, the hypothesis is plausible. Childhood disadvantage has been linked to illnesses, such as cardiovascular diseases,[24] that have overlapping pathologies with diabetes. Persons exposed to socioeconomic disadvantage in childhood are more likely to be of lower socioeconomic means as adults.[25,26] Several studies have shown inverse, graded associations between different measures of adult SEP and the prevalence[11,13,22,27,28] and incidence[11,12,23,29–34] of type 2 diabetes. Childhood SEP also influences adult body composition[35–41] and several behaviors[20,42–45] that are risk factors for type 2 diabetes.

Obesity is a strong predictor of type 2 diabetes.[46–48] Therefore, the effect of childhood SEP on diabetes incidence may differ by body mass index (BMI; weight in kilograms divided my height in meters squared) in adulthood. For example, low childhood SEP and adult obesity together may impart a greater risk of type 2 diabetes than the risk imparted by low childhood SEP alone. Such exposure patterns may represent an accumulation of risk over time or a risk pathway. In addition, several studies have shown that the effects of childhood circumstances on adult health and risk behaviors differ by gender.[37,38,40,49–52] The question remains whether childhood SEP differentially influences diabetes risk for women and men.

Previous studies of childhood SEP and incident diabetes had short follow-up periods,[11,12,23] and one was limited to women.[23] Our approach complemented these studies by using 5 waves of data collected in a population-based sample from 1965 to 1999 to examine the association between childhood SEP and the incidence of type 2 diabetes and how this association may differ by gender or adult BMI.

METHODS

Study Population

Data were drawn from the Alameda County Study, a longitudinal, population-based study of a random, stratified, closed sample of 6928 noninstitutionalized adults aged 17 to 94 years residing in Alameda County, California, in 1965. Comprehensive, self-administered questionnaires were distributed by mail to participants in each of 5 study waves: 1965 (baseline), 1974, 1983, 1994, and 1999. Data was collected on measures of health and physical functioning and their risk factors. Response rates for the 5 surveys were between 85% and 95% of eligible respondents.[53–55]

Of the 6928 eligible participants at baseline, we excluded those who reported previously diagnosed diabetes (n=157; 2.3%), whose diabetes status was unknown (n=5; 0.07%), or who had inconsistencies across

waves in their reported date of diagnosis (n=89, 1.3%). Participants with missing data for any final model covariates (n=764; 11.0%) were also removed. Excluded respondents were more likely to be older, non-White, female, overweight or obese, and of lower SEP. Thus, any association between diabetes incidence and childhood SEP, gender, or adult BMI in the remaining sample would likely be biased toward the null. The final sample consisted of 5913 participants (53.4% women).

Measures

Self-reported diabetes status was assessed at each study wave with 2 questions: "Have you had any of these conditions [e.g., diabetes] during the past 12 months (yes/no)?" and "When did it start (year)?" Incident cases were those reported at one wave but not reported at the previous wave and whose reported year of diagnosis occurred between those 2 waves. Cumulative incidence was the summed total of new cases that occurred between 1965 and 1999. Year of diagnosis was the censoring variable.

Childhood SEP was derived from respondents' fathers' occupation or fathers' education when occupation data were not available (6.5% of sample). Childhood SEP was classified as low (manual occupation or ≤12 years of education) or high (nonmanual occupation or >12 years of education). Analyses were adjusted for respondents' height at baseline. Components of adult height have been suggested as markers of fetal exposures,[56–58] malnutrition,[56,59,60] and other childhood socioeconomic circumstances[56,59–62] that may not be captured by parental SEP. Leg length has been associated with insulin resistance and type 2 diabetes in adulthood.[63–65] In our data, the correlation between baseline height and BMI was small (Pearson $r=0.0587$; $P<.001$).

Demographic risk factors included age, race/ethnicity (White or non-White) and marital status (single; married; or separated, divorced, or widowed). Two dichotomous (yes or no) variables measured access to health care services: possessing health insurance and having a regular health care provider. The presence of high blood pressure was assessed with the question, "Have you had any of these conditions [e.g., high blood pressure] during the past 12 months?" Depression was defined as a score of 5 or higher on a valid and reliable 18-item scale used in other studies to assess depressive symptoms.[66,67]

Data on the type and frequency of 4 activities (physical exercise, long walks, swimming, and participation in active sports) were used to create a physical activity scale that was collapsed into 3 categories: no or low, moderate, and high activity. This scale was used previously and was related to all-cause mortality.[68] Smoking status was defined as current, former, or never smoked. Alcohol use was measured with a scale that combined alcohol type (beer, wine, or liquor), drinking frequency (never, <1 time per week, 1–2 times per week, >2 times per week), and intake at each sitting (none, 1–2 drinks, 3–4 drinks, ≥5 drinks). Three categories of consumption were identified: abstinence (0 drinks per month), light to moderate (1–45 drinks per month), and heavy (≥46 drinks per month). This alcohol consumption scale has been used in other studies to predict mortality.[69,70]

We created continuous values of BMI from self-reported weight and height and further sorted these data into 3 groups: obese (≥30 kg/m^2), overweight (25.0–29.9 kg/m^2), and normal weight (18.5–24.9 kg/m^2).[71] Waist circumference was measured in inches and converted to millimeters. We combined childhood SEP (low, high) with each BMI group to create 6 joint-exposure categories: low childhood SEP–obese, low childhood SEP–overweight, low childhood SEP–normal weight, high childhood SEP–obese, high childhood SEP–overweight, and high childhood SEP–normal weight.

Total years of education was assessed at each study wave and categorized as less than 12, 12, or more than 12 years. At each wave, household income data were collected in bounded categories. A multiple imputation procedure accounted for missing data and assigned a continuous income quantity at each wave.[72] This imputation process was described in detail elsewhere.[30] In these analyses, the continuous imputed household income variable was standardized to 1999 dollars to allow for direct comparison across study waves, adjusted for household size, and log transformed, normalizing the distribution. Three income classifications (low, moderate, and high) were formed from tertiles of the imputed income distribution.

We used US census criteria to code self-reported current, most recent, or (for retirees) primary lifetime occupation and categorized these as white collar (nonmanual occupations: professionals; semiprofessional or technical; managerial, preprietors, or officials; clerical; and sales), blue collar (manual occupations: foremen or craftsmen; operatives; service workers; and laborers), homemaker, or other. The category other included students, the unemployed, and unclassifiable participants (n=425). Results shown are limited to white-collar and blue-collar occupation categories.

Statistical Analyses

Incidence density (new cases per 1000 person-years at risk) was calculated for all covariates for the total population and by gender. We assessed the degree of association between diabetes and childhood SEP, adult BMI, and other covariates for the pooled and gender-stratified samples with the χ^2 test.

We estimated hazard ratios (HRs) and 95% confidence intervals (CIs) for pooled and gender-stratified associations between diabetes incidence and childhood SEP independently and in combination with adult BMI categories with Cox proportional hazards regression models.[73] All analyses modeled baseline covariates. Cox model assumptions and sensitivity were tested and met with Kaplan–Meier curves and SEP–time interactions. We assessed model fit with the likelihood ratio χ^2 test after introduction of each set of covariates.

Participants who died (n=2494; 49.4% women) by the end of 1999 were censored in the year of their death. Living participants who dropped out between 2 waves of data collection were censored at the interval midpoint. Analyses were performed with SAS version 9.1 software (SAS Institute, Cary, North Carolina).

RESULTS

Of 5913 participants at baseline, 307 (5.2%) reported developing diabetes during the 34-year study period. The crude diabetes incidence rate was 3.0 per 1000 person-years for participants with low childhood SEP and 1.7 per 1000 person-years for those with high childhood SEP.

TABLE 1—Baseline Distribution of Study Covariates and Crude Incidence Density (Incidence per 1000 Person-Years at Risk) for Type 2 Diabetes Mellitus Over 34 Years, by Gender: Alameda County Study, Alameda, CA, 1965–1999

	Total		Women		Men	
	No. (%)	Incidence Density (No. of Cases)	No. (%)	Incidence Density (No. of Cases)	No. (%)	Incidence Density (No. of Cases)
Total	5913	2.4 (307)	3157 (53.4)	2.4 (167)	2756 (46.6)	2.4 (140)
Age, y						
<40	2720 (46.0)	2.4 (159)**	1468 (46.5)	2.4 (87)**	1252 (45.4)	2.5 (72)
≥40	3193 (54.0)	2.3 (148)	1689 (53.5)	2.3 (80)	1504 (54.6)	2.3 (68)
Racial group						
Non-White	1139 (19.3)	4.2 (96)**	612 (19.4)	4.8 (59)**	527 (19.1)	3.5 (37)**
White	4774 (80.7)	2.0 (211)	2545 (80.6)	1.9 (108)	2229 (80.9)	2.1 (103)
Marital status						
Single	608 (10.3)	1.6 (22)*	287 (9.1)	1.1 (7)**	321 (11.7)	2.1 (15)
Married (Ref)	4463 (75.5)	2.3 (236)	2238 (70.9)	2.2 (118)	2225 (80.7)	2.4 (118)
Widowed/separated/divorced	842 (14.2)	3.4 (49)	632 (20.0)	3.8 (42)*	210 (7.6)	2.2 (7)
Height						
Below mean	2597 (43.9)	2.7 (146)	1337 (42.4)	2.9 (82)**	1260 (45.7)	2.5 (64)**
Above mean	3316 (56.1)	2.1 (161)	1820 (57.6)	2.0 (85)	1496 (54.3)	2.3 (76)
Childhood SEP[a]						
Low	3082 (52.1)	3.0 (198)**	1604 (50.8)	3.2 (113)**	1478 (53.6)	2.7 (85)*
High (Ref)	2831 (47.9)	1.7 (109)	1553 (49.2)	1.5 (54)	1278 (46.4)	1.9 (55)
Education, y						
<12	1966 (33.3)	3.0 (110)*	1066 (33.8)	3.2 (65)	900 (32.7)	2.8 (45)**
12	1828 (30.9)	2.5 (104)*	1051 (33.3)	2.1 (53)	777 (28.2)	3.0 (51)**
>12 (Ref)	2119 (35.8)	1.8 (93)	1040 (32.9)	1.9 (49)	1079 (39.1)	1.7 (44)
Income, tertile						
Low	1969 (33.3)	2.9 (117)**	1094 (34.7)	3.0 (69)**	875 (31.7)	2.7 (48)
Moderate	1971 (33.3)	2.4 (107)*	1035 (32.8)	2.3 (55)	936 (34.0)	2.5 (52)
High (Ref)	1973 (33.4)	1.9 (83)	1028 (32.5)	1.8 (43)	945 (34.3)	1.9 (40)
Occupation						
White collar	2271 (38.4)	2.2 (116)	1065 (33.7)	2.3 (57)	1206 (43.8)	2.1 (59)
Blue collar	1684 (28.5)	2.9 (99)	391 (12.4)	3.5 (28)	1293 (46.9)	2.8 (71)
Regular health care provider						
No	1327 (22.4)	2.3 (62)	548 (17.4)	2.7 (30)	779 (28.3)	1.9 (32)
Yes	4586 (77.6)	2.4 (245)	2609 (82.6)	2.3 (137)	1977 (71.7)	2.5 (108)
Health Insurance						
No	864 (14.6)	2.5 (40)	501 (15.9)	2.8 (26)	363 (13.2)	2.1 (14)
Yes	5049 (85.4)	2.3 (267)	2656 (84.1)	2.3 (141)	2393 (86.8)	2.4 (126)
Depression						
Yes	841 (14.2)	3.0 (47)	527 (16.7)	3.1 (32)	314 (11.4)	2.7 (15)
No	5072 (85.8)	2.2 (260)	2630 (83.3)	2.2 (135)	2442 (88.6)	2.3 (125)
High blood pressure						
Yes	569 (9.6)	4.3 (41)**	361 (11.4)	4.5 (28)**	208 (7.6)	3.9 (13)
No	5344 (90.4)	2.2 (266)	2796 (88.6)	2.2 (139)	2548 (92.4)	2.3 (127)
BMI category						
Obese (≥30 kg/m^2)	326 (5.5)	9.1 (58)**	186 (5.9)	10.5 (38)**	140 (5.1)	7.3 (20)**
Overweight (25.0–29.9 kg/m^2)	1597 (27.0)	2.9 (100)**	571 (18.1)	2.9 (34)**	1026 (37.2)	3.0 (66)**
Normal (18.5–24.9 kg/m^2; Ref)	3990 (67.5)	1.7 (149)	2400 (76.0)	1.7 (95)	1590 (57.7)	1.6 (54)

Continued

TABLE 1—*Continued*

Waist circumference						
Large[b]	335 (5.7)	7.6 (42)**	227 (7.2)	8.4 (31)	108 (3.9)	5.8 (11)
Normal	5578 (94.3)	2.1 (262)	2930 (92.8)	2.0 (136)	2648 (96.1)	2.3 (129)
Physical activity						
Inactive/low	1858 (31.4)	2.8 (97)	1123 (35.6)	3.0 (65)	735 (26.7)	2.4 (32)
Moderate	2648 (44.8)	2.4 (145)	1388 (44.0)	2.1 (69)	1260 (45.7)	2.7 (76)*
High (Ref)	1407 (23.8)	1.9 (65)	646 (20.4)	2.0 (33)	761 (27.6)	1.8 (32)
Tobacco use						
Current smoker	2655 (44.9)	2.7 (151)**	1298 (41.1)	2.8 (81)**	1357 (49.2)	2.5 (70)
Past smoker	951 (16.1)	2.5 (53)	368 (11.7)	2.8 (24)**	583 (21.2)	2.3 (29)
Never smoked (Ref)	2307 (39.0)	2.0 (103)	1491 (47.2)	1.9 (62)	816 (29.6)	2.2 (41)
Alcohol use, drinks/mo						
None	1207 (20.4)	2.6 (63)	813 (25.8)	2.6 (42)	394 (14.3)	2.7 (21)
1–45	3822 (64.6)	2.3 (201)	2085 (66.0)	2.4 (116)	1737 (63.0)	2.2 (85)
≥46 (Ref)	884 (15.0)	2.2 (43)	259 (8.2)	1.5 (9)	625 (22.7)	2.6 (34)

Note. SEP = socioeconomic position; BMI = body mass index.
[a]Derived from respondents' fathers' occupation (or education when occupation data was not available [6.5% of total]). Low childhood SEP: father with manual occupation (blue-collar occupations: craftsmen and operatives, service workers, and laborers) or 12 years or fewer of education; high childhood SEP: father with non-manual occupation (white-collar occupations: professionals, technical, proprietors, clerical, and sales) or more than 12 years of education (reference category).
[b]Defined as more than 880 mm for women and more than 1020 mm for men.
*$P < .10$; **$P \leq .05$ (χ^2 test for proportional comparison of incident diabetes cases between different variable categories).

TABLE 2—Hazard Ratios (HRs) and 95% Confidence Intervals (95% CIs) for the 34-Year Incidence of Type 2 Diabetes Associated with Childhood Socioeconomic Position, by Gender: Alameda County Study, Alameda, CA, 1965–1999

Model	Total (n = 5913), HR (95% CI)	Women (n = 3157), HR (95% CI)	Men (n = 2756), HR (95% CI)
1	1.8 (1.4, 2.2)	2.1 (1.5, 2.9)	1.5 (1.1, 2.1)
2	1.8 (1.4, 2.3)	2.1 (1.5, 2.9)	1.5 (1.1, 2.1)
3	1.6 (1.3, 2.1)	1.8 (1.3, 2.6)	1.4 (1.0, 2.0)
4	1.6 (1.3, 2.0)	1.8 (1.3, 2.5)	1.4 (1.0, 1.9)
5	1.5 (1.2, 1.9)	1.7 (1.2, 2.4)	1.3 (0.9, 1.9)
6	1.5 (1.1, 1.9)	1.7 (1.2, 2.4)	1.2 (0.8, 1.7)
7	1.5 (1.1, 1.9)	1.7 (1.2, 2.4)	1.2 (0.8, 1.7)

Note. All covariates were measured at baseline (1965). Model 1 was unadjusted; model 2 was adjusted for age; model 3 was adjusted for age, height, race (White or non-White), and marital status; model 4 was adjusted for model 3 covariates plus physical activity, alcohol intake, and smoking; model 5 was adjusted for model 4 covariates plus body mass index and waist circumference; model 6 was adjusted for model 5 covariates plus education, income, and occupation; model 7 was adjusted for model 6 covariates plus high blood pressure, depression, regular access to a medical doctor, and health insurance status.

Table 1 summarizes the distribution and 34-year crude incidence rates for select characteristics at baseline for the total study population and by gender. Differences in diabetes incidence were observed for racial/ethnic group, childhood SEP, obesity, and overweight in the pooled and gender-stratified samples. Among women, but not men, we observed differences for age, marital status, income, high blood pressure, and smoking status. We found differences among men by education and moderate activity level.

Table 2 presents proportional hazards model results for the association between childhood SEP and diabetes incidence for the total population and by gender. Low childhood SEP was associated with an increased risk of diabetes in unadjusted models for all groups, although the relative hazard was largest among women (model 1). These associations did not change after we controlled for age (model 2). Adjustment for height and demographic confounders, especially racial/ethnic group, improved model fit and slightly attenuated the relationship for all groups (likelihood ratio χ^2 $P<.001$; pooled, low childhood SEP, HR=1.6; 95% CI=1.3, 2.1; women, low childhood SEP, HR=1.8; 95% CI=1.3, 2.6; men, low childhood SEP, HR=1.4; 95% CI=1.0, 2.0).

Subsequent models added potential pathway components between childhood SEP and incident diabetes. Behavioral covariates did not change the risk of low childhood SEP associated with diabetes incidence that we observed after adjustment for demographic factors (model 4). Inclusion of body composition variables improved the fit of the model (likelihood ratio χ^2 $P<.001$), but with negligible change in effect size (model 5; pooled, low childhood SEP, HR=1.5; 95% CI=1.1, 1.9; women, low childhood SEP, HR=1.1; 95% CI=1.2, 2.4; men, low childhood SEP, HR=1.2; 95% CI=0.8, 1.7). Inclusion of other SEP measures (model 6) or full adjustment (model 7) did not improve model fit in the pooled or gender-stratified data.

Results from analyses of diabetes risk attributable to the combined effect of childhood

TABLE 3—Hazard Ratios (HRs) and 95% Confidence Intervals (95% CIs) for 34-Year Incidence of Type 2 Diabetes, by Childhood Socioeconomic Position (SEP), Adult Body Mass Index Category, and Gender: Alameda County Study, Alameda, CA, 1965–1999

	Model 1		Model 2		Model 3		Model 4	
	Low Childhood SEP, HR (95% CI)	High Childhood SEP, HR (95% CI)	Low Childhood SEP, HR (95% CI)	High Childhood SEP, HR (95% CI)	Low Childhood SEP, HR (95% CI)	High Childhood SEP, HR (95% CI)	Low Childhood SEP, HR (95% CI)	High Childhood SEP, HR (95% CI)
Total sample (n = 5913)								
Normal weight	1.6 (1.2, 2.2)	1.00	1.5 (1.1, 2.1)	1.00	1.4 (1.0, 2.0)	1.00	1.4 (1.0, 1.9)	1.00
Overweight	3.00 (2.1, 4.2)	1.6 (1.1, 2.6)	2.7 (1.9, 3.9)	1.6 (1.0, 2.5)	2.7 (1.9, 3.8)	1.6 (1.00, 2.5)	2.5 (1.7, 3.6)	1.5 (1.0, 2.4)
Obese	8.6 (5.8, 12.8)	6.3 (3.7, 10.8)	7.0 (4.7, 10.7)	5.5 (3.2, 9.4)	5.5 (3.5, 8.8)	4.0 (2.2, 7.1)	5.0 (3.1, 8.1)	3.6 (2.0, 6.5)
Women (n = 3157)								
Normal weight	1.7 (1.1, 2.6)	1.00	1.5 (1.0, 2.3)	1.00	1.5 (1.0, 2.2)	1.00	1.5 (1.0, 2.2)	1.00
Overweight	3.4 (2.1, 5.5)	1.00 (0.4, 2.4)	3.0 (1.8, 4.9)	0.9 (0.4, 2.2)	2.9 (1.7, 4.8)	0.9 (0.4, 2.2)	2.8 (1.6, 4.7)	0.9 (0.4, 2.2)
Obese	10.2 (6.2, 16.8)	6.6 (3.4, 13.0)	7.8 (4.6, 13.3)	5.4 (3.1, 10.9)	5.8 (3.1, 10.9)	3.5 (1.6, 7.5)	5.4 (2.8, 10.3)	3.1 (1.4, 6.9)
Men (n = 2756)								
Normal weight	1.4 (0.8, 2.5)	1.00	1.4 (0.8, 2.3)	1.00	1.3 (0.8, 2.3)	1.00	1.2 (0.7, 2.1)	1.00
Overweight	2.7 (1.6, 4.6)	1.9 (1.1, 3.4)	2.5 (1.5, 4.2)	1.9 (1.1, 3.3)	2.5 (1.5, 4.2)	1.9 (1.1, 3.3)	2.2 (1.3, 3.8)	1.8 (1.0, 3.2)
Obese	6.3 (3.2, 12.5)	5.9 (2.9, 11.8)	5.9 (2.9, 11.8)	5.6 (2.4, 13.1)	5.1 (2.5, 10.6)	4.6 (1.9, 11.5)	4.4 (2.1, 9.3)	4.3 (1.7, 10.7)

Note. All covariates were measured at baseline (1965). Childhood socioeconomic position was derived from respondents' fathers' occupation (or education when occupation data was not available; 6.5% of total). Low childhood SEP: father with manual occupation (blue-collar occupations: craftsmen and operatives, service workers, and laborers) or 12 years or fewer of education; high childhood SEP: father with non-manual occupation (white-collar occupations: professionals, technical, proprietors, clerical, and sales) or more than 12 years of education (reference category). Model 1 was unadjusted; model 2 was adjusted for age, height, race (White or non-White), and marital status; model 3 was adjusted for model 2 covariates plus waist circumference, physical activity, alcohol consumption, and smoking status; model 4 was adjusted for model 3 covariates plus education, income, occupation, high blood pressure, depression, regular access to a medical doctor, and health insurance status. Obese was defined as a body mass index (BMI) of 30 kg/m^2 or more. Overweight was a BMI of 25.0 to 29.9 kg/m^2. Normal was a BMI of 18.5 to 24.9 kg/m^2.

SEP and adult BMI category for the pooled and gender-stratified samples are presented in Table 3. In unadjusted models (model 1), we observed an excess risk of incident diabetes for each joint childhood SEP–adult BMI category compared with the referent group (high childhood SEP–normal weight) for the pooled sample and in both women and men, except for overweight women who had high childhood SEP. In the pooled sample, the risk of diabetes associated with low and high childhood SEP differed by adult BMI category. Among women, only those with low childhood SEP showed different HRs and distinct CIs for each adult BMI category. Although the effect sizes differed among men, the CIs for each adult BMI category overlapped regardless of childhood SEP (model 1).

The risk and CI patterns observed in unadjusted models remained after adjustment for demographic confounders and height (model 2). Although effect sizes remained elevated for most joint-exposure categories, they were reduced for the low childhood SEP–obese category in all samples. CIs included 1 for overweight women who had high childhood SEP and for normal-weight men and women who had low childhood SEP (model 2).

Including waist circumference and behaviors in the model (model 3) or full adjustment (model 4) did not change effect sizes in any category except the low childhood SEP–obese group. CIs associated with each adult BMI category no longer were distinct for any category in the pooled and gender-stratified groups. These changes may have reflected a reduction in statistical power attributable to diminished sample size in each joint-exposure category after multivariate adjustment (models 3 and 4).

DISCUSSION

Our data identified low childhood SEP as a robust, independent predictor of incident type 2 diabetes in adulthood, especially among women. Adjustment for race/ethnicity and body composition (i.e., BMI and waist circumference) partially explained this association. The association of low childhood SEP and diabetes incidence was independent of education in women and of income or occupation in both women and men. Adjustment for education, although it produced no change in effect size or model fit in these data, may have been overadjustment, because education is often considered a component of childhood SEP.

We observed a cumulative risk effect, especially among women, with childhood disadvantage and adult overweight or obesity. For example, the risk associated with joint exposure to low childhood SEP and overweight or obesity in later life was greater than the risk associated with each factor independently. Diabetes risk factors (physical activity, waist circumference, etc.) and race/ethnicity minimally reduced the effect size associated with the joint low childhood SEP–obese groups in pooled and gender-stratified models.

Limitations and Strengths

Limitations constrained any inferences we could draw from our findings. The foremost of these was that all data were self-reported, which could have resulted in misclassification of exposure and disease status. However, self-reported disease status has been correlated with diagnostically confirmed diabetes.[74]

Several studies have shown that adult recall of childhood SEP factors are likely to underestimate their effects on adult health outcomes[24,75,76]; another study produced similar effects for adult recall and actual SEP measurement during childhood.[77] The observed associations between childhood SEP and adult diabetes in our data, therefore, may underestimate the true effect. Moreover, all covariates were measured at baseline, which may not have captured their total influence on diabetes incidence. Given the time-dependent nature of most covariates, some misclassification could have resulted from using only 1 measurement.

The association between childhood SEP and diabetes incidence was confounded by age and race in these data. Statistical adjustment is appropriate when a variable is not an exposure of interest.[78] In these data, adjustment averaged risk across demographic groups and likely controlled for unmeasured factors, such as discrimination, that are associated with race and age and possibly are predictive of diabetes risk.

We could not distinguish between diabetes types 1 and 2. Approximately 93% of cases diagnosed in persons 30 years or older are type 2 diabetes.[79] All participants who reported diabetes after 1965 were counted as incident cases, regardless of age at diagnosis. Covariate distributions did not differ by age at diagnosis. However, age-stratified diabetes risk attributable to childhood SEP differed by gender. We observed no difference in risk for men when we compared analyses of the full sample with a sample restricted to persons aged 40 years or older at baseline. Among women, the effect in the all-ages sample was smaller than that in the age-restricted sample (results not shown). Consequently, models that used the all-ages sample likely attenuated the association between childhood SEP and incident diabetes for women but led to minimal bias for men.

Survival bias also may have affected our findings. Compared with participants without diabetes, case participant may have been more likely to die or drop out before being counted. If those participants were disadvantaged in childhood, the relationship between low childhood SEP and incident disease would be reduced. Notwithstanding selective participation or survival, the incidence proportion (5.2%) over the 34-year study period was similar to national self-reported estimates (5.1%).[80]

Finally, in these data, the small sample of incident cases, especially when stratified by gender or BMI category, resulted in wide CIs for many associations, despite elevated effect sizes. Similar patterns were observed between childhood SEP and incident diabetes, and subsequent covariate adjustment, in a sample of 100 330 women from the Nurses Health Study.[23] A study combining data from the Health and Retirement Study and the Study of Asset and Health Dynamics Among the Oldest Old found that higher SEP measures were protective for women.[11] Conversely, childhood SEP had no effect on diabetes incidence in Health and Retirement Study data from a later period.[12] BMI was an explanatory factor for the childhood SEP–diabetes incidence relationship in 2 of these studies.[11,23]

This study had several strengths. First, data were collected over 34 years. Second, longitudinal data allowed study of multiple determinants of incident disease. Third, these data permitted simultaneous examination of a variety of sociodemographic confounders and potential components of the causal pathway(s) from childhood SEP to incident diabetes in adulthood. Finally, this is one of the few studies to investigate the effect of childhood SEP on incident type 2 diabetes and to consider how the association may differ by gender or adult BMI.

Childhood SEP and Diabetes Risk

Childhood SEP affects development or expression of diabetes risk factors, such as physical inactivity, poor eating habits, and limited socioeconomic opportunities, that persist into adulthood[41,81–84] and that may help explain the relationship between childhood SEP, adult obesity, and adult diabetes in our data. Parental SEP is associated with parental health behaviors and body composition, which influence these characteristics in children.[41,81,85] Other, unmeasured childhood exposures, such as environmental hazards, social instability, or other stressors, also may contribute to the relationship observed between childhood SEP and diabetes.[25,26,86–88] Finally, diabetes and its precursors have been linked to altered nutrition or other exposures during critical periods of fetal and childhood growth and development.[10,56,89–93]

Height is used as a marker of early-life circumstances.[56–62] In our data, the association between childhood SEP and incident diabetes persisted after adjustment for height, suggesting that the childhood SEP measure did not act solely as a proxy for developmental processes. Adult height, however, would not measure fetal insults that do not alter growth in early life but manifest as metabolic and other abnormalities in adulthood.[94] Therefore, these unmeasured factors, and mechanisms other than impaired growth or development, also contributed to disease incidence.

Our data did not include childhood SEP measures, such as parental income or maternal education, or information about fetal, neonatal, or childhood growth and development, childhood BMI, or components of adult height (e.g., leg and trunk length). Comprehensive data on these and related early-life characteristics, especially during critical periods of development, are necessary to elucidate the relationship between childhood SEP, its biological correlates, and the incidence of adult type 2 diabetes.

Disparate social opportunities also help explain gender differences in the childhood SEP–diabetes relationship. Childhood disadvantage contributes to gender discrimination in education and occupation opportunities throughout adolescence and adulthood.[25,87,95] In our study, the distribution of life-course SEP measures differed by gender, with women less likely than men to have had a high SEP in childhood, to have higher education, or to have a white-collar occupation. Consequences of social disadvantage across the life course, including poor nutrition, unhealthy behaviors, and limited access to material goods, all factors related to diabetes, may have been stronger for women in our study because of their limited social mobility.[41]

The lack of an education or adult SEP effect for women in this study may be attributable to their underrepresentation in the workforce. More than 50% of female participants did not work outside the home at baseline. Childbearing and other family responsibilities contribute to sporadic labor force participation, ultimately limiting adult SEP mobility for women.[96] Regardless of the mechanism,

our results support other findings that childhood SEP influences metabolic characteristics in women, independent of education or adult SEP.[18,20,23,43,83,97]

The relationship between childhood SEP and incident diabetes differed by adult BMI category in our data. Although low childhood SEP was independently associated with increased risk of diabetes, we found that the combined effect of childhood disadvantage and adult overweight or obesity imparted an even greater risk of type 2 diabetes.

The differences in the association between childhood SEP and incident diabetes by adult BMI category were significant for the pooled and women-only samples, although effect sizes were elevated for all groups, including men. These gender differences are not surprising. Obesity is a known risk factor for type 2 diabetes.[46–48] The prevalence of adult measures of body composition differ by gender,[98] disproportionately affecting women.[97,99] In addition, adult body composition is associated with social disadvantage during childhood,[35–41,97] especially among women.[37,38,97]

Conclusions

Our results add to the literature showing an increased risk of type 2 diabetes[11,23] and diabetes markers[10,15,16,18,19] in persons socially disadvantaged in early life. We also demonstrated the effect of an accumulation of harmful exposures over the life course on the development of diabetes in adulthood. These associations suggest that the relationships between childhood disadvantage and later disease may differ by gender, disproportionately affecting women.

The consequences of early-life exposure to damaging risk factors, including low SEP, persist into adulthood. Perpetuation of childhood poverty, combined with increasing obesity, leads to exaggerated rates of diabetes and related diseases, particularly among women. Therefore, it is vital to elucidate the association between SEP and other risk factors across the life course and the development of type 2 diabetes and other conditions in later life. Clear understanding of these pathways would inform the design of prevention programs and social policies to reduce the burden of disease linked to social disadvantage across the life course. ■

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Contributors

S.C. Maty originated the study, completed data analysis and interpretation, and wrote the article. J.W. Lynch and G.A. Kaplan contributed to concept development, study design, interpretation of results, and critical revision of the article. T.E. Ragunathan provided assistance with statistical methods and interpretation of results.

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Human Participant Protection

This study was approved by the institutional review board of the University of Michigan.

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