Life Course Socioeconomic Position, Allostatic Load, and Incidence of Type 2 Diabetes among African American Adults: The Jackson Heart Study, 2000-04 to 2012

Gloria L. Beckles, MD, MSc[1]; Kai McKeever Bullard, PhD, MPH[1]; Sharon Saydah, PhD[1]; Giuseppina Imperatore, MD, PhD[1]; Fleetwood Loustalot, PhD, MPH[2]; Adolfo Correa, MD, MPH[3]

Objective: We examined whether life course socioeconomic position (SEP) was associated with incidence of type 2 diabetes (t2DM) among African Americans.

Design: Secondary analysis of data from the Jackson Heart Study, 2000-04 to 2012, using Cox proportional hazard regression to estimate hazard ratios (HR) with 95% CI for t2DM incidence by measures of life course SEP.

Participants: Sample of 4,012 nondiabetic adults aged 25-84 years at baseline.

Outcome Measure: Incident t2DM identified by self-report, hemoglobin A1c ≥6.5%, fasting plasma glucose ≥126 mg/dL, or use of diabetes medication.

Results: During 7.9 years of follow-up, 486 participants developed t2DM (incidence rate 15.2/1000 person-years, 95% CI: 13.9-16.6). Among women, but not men, childhood SEP was inversely associated with t2DM incidence (HR=.97, 95% CI: .94-.99) but was no longer associated with adjustment for adult SEP or t2DM risk factors. Upward SEP mobility increased the hazard for t2DM incidence (adjusted HR=1.52, 95% CI: 1.05-2.21) among women only. Life course allostatic load (AL) did not explain the SEP-t2DM association in either sex.

Conclusions: Childhood SEP and upward social mobility may influence t2DM incidence in African American women but not in men. *Ethn Dis*. 2019;29(1):39-46; doi:10.18865/ed.29.1.39

Keywords: Life Course Socioeconomic Position; Type 2 Diabetes; Allostatic Load; African Americans

Introduction

In the United States, minority racial-ethnic and socioeconomically disadvantaged groups are disproportionately affected by type 2 diabetes mellitus (t2DM).[1,2] As elsewhere, risks for t2DM increase with decreasing socioeconomic position (SEP).[3] Several conceptual models propose how SEP across the life course can influence health in adulthood.[4,5] The critical/sensitive period model specifies that, during specific periods of development adverse physical and social exposures may have long-lasting effects on the structure and function of systems, organs, and tissues. The effect of this biological programming on risk may be modified by exposures in adulthood. The accumulation of risk model proposes that effects of exposures at different life stages may accumulate over time resulting in increasing cumulative damage to health. The pathways effects model proposes that early life socioeconomic circumstances track social trajectories into adulthood which, in turn, influence health. Studies using the life course approach have shown that the timing (critical/sensitive periods), frequency and duration of exposure to social stressors influence incidence of t2DM.[6-13] To date, these models have been tested among White adults, and in three studies the relationships were demonstrated in women but not in men.[6-7, 9,12] One study reported that low childhood SEP increased t2DM incidence regardless of race but did not report interaction with sex.[13]

Complementary to the accumulation of risk hypothesis is the concept of allostatic load (AL) or dysregulation of multiple physiologic systems that may arise from repeated or chronic exposure to social stressors.[14] Compared with Whites, African Americans have higher levels of

[1] Division of Diabetes Translation, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, Atlanta, GA
[2] Division for Heart Disease & Stroke Prevention, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, Atlanta, GA
[3] University of Mississippi Medical Center, Jackson, MS

Address correspondence to Gloria Beckles, MD, MSC; Centers for Disease Control and Prevention, Division of Diabetes Translation, 4770 Buford Hwy NE, MS F-75, Atlanta, GA 30341; glb4@cdc.gov

AL and are more likely to experience chronic exposure to socioeconomic stressors.[15] To our knowledge, no study has examined the contribution that AL may make to the life course SEP-t2DM association among African Americans. Therefore, we aimed to examine whether: 1) life course SEP was associated with t2DM incidence among African Americans; 2) the relationship was modified by sex; and 3) AL explained the life course SEP-t2DM association.

We aimed to examine whether: 1) life course SEP was associated with t2DM incidence among African Americans; 2) the relationship was modified by sex; and 3) AL explained the life course SEP-t2DM association.

Methods

Data Source and Study Population

We used data from the Jackson Heart Study (JHS), a population-based prospective study of cardiovascular disease among African American residents of Jackson, Mississippi.[16] Of the 5,306 residents aged 21-94 years who participated at baseline (2000-2004), we identified 4,012 participants aged 25 to 84 years with no evidence of diabetes who were followed through December 31, 2012.

Variables

Incident cases were identified during follow-up if a participant reported physician-diagnosed diabetes, use of diabetes medication, or had a fasting plasma glucose ≥126 mg/dL or HbA1c ≥6.5%.

Life-course (SEP) was conceptualized to represent three life stages during which individuals experienced different timing and levels of SEP exposure.[5] Childhood SEP (C-SEP) was measured using number of years of schooling or highest degree completed by parents or most important caretakers when participant was up to age 16 years. Young adulthood SEP (YA-SEP) was measured by participants' educational attainment at baseline (<high school, high school/GED, >high school). Levels of C-SEP and YA-SEP exposures were categorized as low (<12 years of schooling or <high school); medium (12 years of schooling or high school/GED); high (>12 years of schooling or >high school).[17] Mature adulthood SEP (MA-SEP) was measured using the Olin Wright social class typology which describes a managerial, supervisory and worker hierarchy based on job autonomy in the workplace.[17] Participants were classified as managers if they reported that in the workplace, they: a) made decisions about such things as the products or services offered, number of people employed, budgets; and b) supervised the work of other employees, had responsibility for what work other employees did. Those who reported that they only supervised other employees were classified as supervisors. Otherwise, participants were classified as neither. Level of MA-SEP exposure was categorized as low (neither), medium (supervisor), and high (manager). Based on a social mobility framework which recognizes that SEP may vary across the life span,[4] each SEP measure was re-categorized as a binary variable (less than high, high) to define 3 SEP trajectories: 1) stable if the level of SEP exposure in childhood remained the same in young adulthood; 2) downward if the level of SEP exposure fell from high in childhood to less than high in young adulthood; 3) upward if the level of SEP exposure rose from less than high in childhood to high in young adulthood. Trajectories from childhood to mature adulthood were defined similarly.

Traditional t2DM risk factors selected were age, sex, parental history of diabetes, physical activity and dietary consumption (poor, intermediate, ideal),[18] and smoking status (current, former, never); height was selected as a biological marker of cumulative nutritional, socioeconomic, and health deprivation.[19] (Table 1). Based on previous research and availability in the JHS dataset, we selected a total of 11 biomarkers to reflect responses to: a) the neuroendocrine system (serum cortisol); b) the cardiovascular system (systolic blood pressure, diastolic blood pressure, heart rate, homocysteine); c) the metabolic system (total cholesterol, HDL cholesterol, serum creatinine, waist circumference); and d) the immune system (high sensitivity C-reactive protein,

Table 1. Baseline characteristics of nondiabetic African Americans by sex—the Jackson Heart Study, 2000-2004

Characteristics	Women, N = 2,518		Men, N = 1,494	
	% or mean	95% CI	% or mean	95% CI
Childhood SEP				
Mother's educational attainment, %				
<12 years	62.7	(60.6-64.8)	52.8	(50.2-55.5)
12 years	17.8	(16.2-19.4)	23.4	(21.2-25.6)
>12 years	19.5	(17.8-21.2)	23.8	(21.6-26.0)
Father's educational attainment, %				
<12 years	68.4	(66.1-70.8)	64.6	(61.7-67.7)
12 years	16.3	(14.8-17.9)	17.6	(15.0-20.3)
>12 years	15.2	(13.1-17.4)	17.7	(15.1-20.4)
Young adulthood SEP				
Own educational attainment, %				
Less than high school	17.0	(15.5-18.4)	19.1	(17.1-21.1)
High school/GED	25.0	(23.3-26.7)	23.4	(21.3-25.6)
More than high school	58.0	(56.1-60.0)	57.4	(54.9-60.0)
Mature adulthood SEP				
Occupational social class, %				
Neither	54.7	(52.7-57.0)	40.7	(38.0-43.3)
Supervisory	13.3	(11.9-14.7)	14.4	(12.5-16.3)
Managerial	32.0	(30.1-33.9)	44.9	(42.2-47.7)
t2DM risk factors				
Age, years, mean	54.7	(54.2-55.2)	53.6	(53.0-54.3)
Height, cm, mean	164.0	(163.8-164.3)	177.5	(177.1-177.8)
Family history of diabetes, %	35.5	(33.7-37.4)	30.5	(28.2-32.9)
Physical activity, %				
Poor	47.8	(45.9-49.8)	45.3	(42.7-47.8)
Intermediate	34.1	(32.3-36.0)	29.9	(27.5-32.2)
Ideal	18.0	(16.5-19.5)	24.9	(22.7-27.1)
Healthy diet, %				
Poor	61.6	(59.7-63.5)	68.5	(66.1-70.8)
Intermediate	37.5	(35.6-39.3)	31.1	(28.7-33.4)
Ideal	.9	(.1-.8)	.4	(.1-.8)
Smoking status, %				
Current	10.9	(9.7-12.2)	19.2	(17.2-21.3)
Former	1.2	(.7-1.6)	1.3	(.8-1.9)
Never	87.9	(86.6-89.2)	79.4	(77.3-81.5)
AL global risk score, mean	2.5	(2.5-2.6)	3.5	(3.5-3.6)

SEP, socioeconomic position; AL, allostatic load.
All percentages do not sum to 100 because of rounding.

white blood cells). Biomarkers were stratified into quartiles and (except for HDL cholesterol) values above the 75th percentile of each biomarker were considered high risk; otherwise, values were not high risk.[13] For HDL cholesterol, values below the 25th percentile were considered high risk; otherwise, values were not high risk. Then, we calculated an AL global risk score for each participant by summing the total number of biomarkers with high risk levels; not high risk levels received a score=0 (overall range: 0-11).

Statistical Analyses

We used the iterated chained equations approach to perform multiple imputations of all variables needed for the analysis.[20] The mi impute chained and the mi estimate commands in Stata version 13 (StataCorp LP; College Station, Texas) were used to create 5 imputed datasets to calculate pooled estimates.[21] Descriptive analyses examined the distributions of baseline covariates. Behavioral covariates were recategorized as binary variables (poor, not poor; current/former, never) for use in the regression analyses. Survival

analysis was used to estimate time (in years) from baseline examination to first occurrence of t2DM, with survival times censored at dates of death, loss to follow-up, or December 31, 2012. Incidence rates (cases per 1000 person-years) were calculated for each life course SEP measure. Cox proportional hazards regression models were fitted to estimate unadjusted and adjusted hazard ratios for incident t2DM by life-course SEP measures. All analyses were stratified by sex. Differences were considered significant at P<.05.

Results

Table 1 presents the characteristics of nondiabetic participants by sex at baseline examination. Participants reported more years of schooling for their mothers than fathers (men, 10.9 years vs 9.3 years; women, 10.2 years vs 8.9 years). We found no sex difference in YA-SEP but for MA-SEP, more men than women were in the managerial class (44.9% vs 32.0%). Men and women were of similar age. Women were more likely than men to report a family history of diabetes but they were less likely to report ideal physical activity (18.0% vs 24.9%), a poor diet (61.6% vs 68.5%), or current smoking (10.9% vs 19.2%). Women also had a lower mean AL global risk score than men (2.6 vs 3.6).

Association of Life Course SEP with T2DM Incidence

During a mean follow-up of 7.9 years, 486 of the 4,012 nondiabetic participants developed t2DM: overall crude incidence rate was 15.2/1000 person-years (women 15.4/1000 person-years; men 14.8/1000 person-years) (Table 2). Because father's years of schooling were not associated with t2DM incidence in either sex, C-SEP was measured by mother's years of schooling in all further analyses (Table 3). C-SEP was inversely associated with t2DM among women but not men. C-SEP was barely associated with t2DM (HR=.97; P=.05) in a model adjusted only for the traditional risk factors and AL (Model 2). In the fully adjusted model, no SEP measures were associated with t2DM (Model 3). We repeated all analyses using the full sample and confirmed the sex interaction (P=.02) in the C-SEP-t2DM association.

Table 2. Incidence rate of type 2 diabetes by life course socioeconomic position and sex—the Jackson Heart Study, 2000-04 to 2012

	Women, N=2,518				Men, N=1,494			
	Person-years (p-y)	Cases (n)	Incidence rate per 1000 p-y	(95% CI)	Person-years (p-y)	Cases (n)	Incidence rate per 1000 p-y	(95% CI)
Total	20,378	314	15.4	(13.8-17.2)	11,590	172	14.8	(12.8-17.2)
Childhood SEP								
Mother's educational attainment								
<12 years	1281	216	16.9	(14.7-19.3)	6201	88	14.2	(11.5-17.5)
12 years	7553	55	15.5	(11.9-20.2)	2669	45	16.9	(12.6-22.6)
>12 years	4008	43	10.7	(8.0-14.5)	2761	39	14.1	(10.3-19.3)
Father's educational attainment								
<12 years	14175	211	14.9	(13.0-17.0)	7580	103	13.6	(11.2-16.5)
12 years	3254	59	18.1	(14.0-23.4)	2049	45	22.0	(16.4-29.4)
>12 years	2949	44	14.9	(11.1-20.1)	2002	24	12.0	(8.0-17.9)
Young adulthood SEP								
Own educational attainment								
Less than high school	3366	58	17.2	(13.3-22.3)	2164	27	12.5	(8.6-18.2)
High school/GED	5080	75	14.8	(11.8-18.5)	2670	37	13.9	(10.0-19.1)
More than high school	11862	180	15.2	(13.1-17.6)	6756	108	16.0	(13.2-19.3)
Mature adulthood SEP								
Occupational social class								
Neither	10141	167	16.5	(14.2-19.2)	4125	74	17.9	(14.3-22.5)
Supervisory	2496	37	14.8	(10.7-20.5)	1517	24	15.8	(10.6-23.6)
Managerial	6003	96	16.0	(13.1-19.5)	4696	65	13.8	(10.9-17.7)

SEP, socioeconomic position.

Table 3. Hazard ratios (95% CI) for incidence of type 2 diabetes by life course socioeconomic position and sex—the Jackson Heart Study, 2000-04 to 2012

	Women, N=2,518			Men, N=1,494		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Childhood SEP	.97 (.94-.99)	.97 (.94-1.00)	.97 (.94-1.04)	1.02 (.98-1.06)	1.02 (.98-1.06)	1.02 (.98-1.06)
Age, years		.99 (.99-1.01)	1.00 (.99-1.01)		1.00 (.98-1.01)	1.00 (.98-1.02)
Height, cm		1.02 (.99-1.02)	1.01 (.99-1.03)		1.02 (1.00-1.04)	1.02 (1.00-1.04)
Family history of diabetes						
Yes		1.43 (1.14-1.80)[b]	1.40 (1.10-1.76)[b]		1.81 (1.33-2.45)[c]	1.77 (1.29-2.41)[b]
No (ref.)		1.00	1.00		1.00	1.00
Physical activity						
Poor		1.00 (.86-1.17)	.97 (.83-1.10)		.84 (.69-1.02)	.81 (.66-.99)[a]
Not poor (ref.)		1.00	1.00		1.00	1.00
Healthy diet						
Poor		1.17 (.94-1.45)	1.20 (.96-1.50)		1.25 (.91-1.71)	1.29 (.93-1.79)
Not poor (ref.)		1.00	1.00		1.00	1.00
Smoking status						
Current or former		1.21 (.99-1.50)	1.19 (.96-1.47)		1.12 (.91-1.38)	1.07 (.86-1.34)
Never (ref.)		1.00	1.00		1.00	1.00
AL global risk score		1.17 (1.10-1.25)[c]	1.19 (1.11-1.28)[c]		1.16 (1.06-1.27)[b]	1.16 (1.06-1.27)[b]
High						
Not high						
Young adulthood SEP						
Less than high school (ref.)			1.00			1.00
High school or GED			.91 (.63-1.31)			1.11 (.64-1.92)
More than high school			1.02 (.72-1.44)			1.21 (.74-1.98)
Mature adulthood SEP						
Manager			.97 (.75-1.25)			.75 (.54-1.06)
Supervisor			.91 (.64-1.30)			.89 (.56-1.41)
Neither (ref.)			1.00			1.00

SEP, socioeconomic position; AL, allostatic load.
a. P<.05.
b. P<.01.
c. P<.001.
Model 1 = unadjusted; Model 2 = controls for traditional diabetes risk factors and AL global risk score; Model 3 = additional control for Young adulthood SEP and Mature adulthood SEP.

Association of Life Course SEP Trajectories with Incidence of T2DM

The SEP trajectory from childhood to adulthood was associated with incidence of t2DM among women but not among men (Table 4). Women exposed to low SEP in childhood and young adulthood experienced a higher unadjusted hazard ratio incidence (HR=1.61) compared with women exposed to a stable high SEP. With adjustment, this effect was attenuated (HR=1.41) and the association was no longer significant. Among women whose SEP status rose from low/medium in childhood to high in young adulthood, the hazard was 1.64 times that for those with stable high SEP. With adjustment for traditional risk factors and AL the association was attenuated (HR=1.52) but remained significant. Decline in SEP status from high in childhood to low/medium in young adulthood was not associated with t2DM incidence (HR=1.68). Childhood to mature adulthood SEP trajectories were not associated with t2DM incidence.

Contribution of AL to the Life Course SEP-T2DM Association

Adjustment for the AL global risk score did not reduce or completely explain the association between any life course SEP measure and t2DM incidence (Table 4). However, the score was positively associated with t2DM incidence in

Table 4. Hazard ratios (95% CI) for incidence of type 2 diabetes by change in socioeconomic position among women—the Jackson Heart Study, 2000-04 to 2012

Social mobility indicator	Model 1		Model 2		Model 3		Model 4	
	OR	95% CI	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Stable low SEP	1.61[a]	(1.03-2.51)	1.46	(.90-2.37)	1.50	(.92-2.44)	1.45	(.89-2.36)
Downward mobility	1.83	(.9-3.52)	1.72	(.88-3.35)	1.78	(.91-3.46)	1.68	(.86-3.26)
Upward mobility	1.64[b]	(1.14-2.36)	1.56[a]	(1.08-2.27)	1.56[a]	(1.08-2.27)	1.52[a]	(1.05-2.20)
Stable high SEP (ref.)	1.00		1.00		1.00		1.00	

SEP, socioeconomic position.
a. P<.05.
b. P<.01.
Model 1=unadjusted; Model 2= adjusted for demographic diabetes risk factors; Model 3= additional adjustment for behavioral diabetes risk factors; Model 4= additional adjustment for allostatic load global risk score.

the C-SEP and SEP trajectory models in both sexes [data not shown].

Discussion

In this study of African American adults, we found that life course SEP may influence the development of t2DM in women but not in men. The risk of developing t2DM in later life was inversely related to the level of SEP exposure in childhood, but the association was not independent of either SEP or traditional t2DM risk factors in adulthood. We also found that women who experienced upward SEP mobility from childhood to young adulthood compared with those with stable high SEP had an increased risk of developing t2DM, and that this association was independent of adult SEP and t2DM risk factors. However, AL did not explain the effect of life course SEP on t2DM incidence in either sex.

Our finding that the critical/sensitive period hypothesis did not support an effect of childhood SEP on incidence of t2DM among African American women in later life was consistent with results from studies conducted among White Americans and elsewhere.[8,10,11] However, the current result is not strictly comparable for reasons such as differences in the populations, duration of follow-up, measures of early-life SEP, t2DM risk factors and analytic methods. To date, only one study has reported race-specific results: at 34 years of follow-up of the Alameda County cohort, low childhood SEP increased t2DM incidence among Black and White participants but no sex interaction was reported.[7]

With regard to social mobility across the life course, our findings are consistent with earlier research.[6,7,10] Studies in the United States and United Kingdom all reported increased incidence with downward SEP mobility. In contrast, the current study found that increased incidence among African American women was not associated with decline in SEP but was associated with upward SEP mobility.

People who develop t2DM grow differently in early life from those who do not develop the disease.[22] Exposure to adverse environmental influences during development is associated with slow growth in utero, low birthweights, small size throughout infancy, and rapid gain in weight and body mass when no longer exposed to the adverse influences. High rates of such adverse outcomes among African Americans are well-documented.[23] Most members of the JHS cohort were born before the middle of the 20th century; therefore, many of their mothers could have experienced the intergenerational economic and nutritional deprivation prevalent in the southern states until the late 1970s.[24,25] Research also shows that early life exposure to socioeconomic

In this study of African American adults, we found that life course SEP may influence the development of t2DM in women but not in men.

stressors may also set in motion long-term trajectories of metabolic risk factors for t2DM but only in women.[26]

Kaplan et al showed how, after 1964, Black women proved to be the greatest beneficiaries of the occupational and economic improvements that increased in the southern states in response to Civil Rights policies.[25] However, the striving to escape persistent poverty could itself have proven to be a chronic stressor. Goal-striving stress, the discrepancy between socially derived aspiration and achievement, is associated with poor physical and mental health among adult Americans.[27,28] Specifically, among African Americans, this type of stress was strongly associated with psychological distress, a condition more common and more severe among women than men.[28] Upward social mobility may also be strongly associated with reduced psychological well-being among African Americans, more so in women than men.[29] Recent evidence indicates that psychological distress is associated with incident t2DM independent of traditional behavioral risk factors.[30] The studies cited above suggest plausible explanations for the unexpected effect of upward social mobility on t2DM observed among African American women.

Limitations

The current study is subject to several limitations. First, the JHS sample is not a nationally representative sample; consequently, the findings are not generalizable to the total adult African American population. Second, few studies have assessed the accuracy with which adults recall parental SEP.[31-33] One recent study found poor agreement between young African American women and their mothers about SEP in early (kappa=.14) and late childhood/adolescence (kappa=.20).[33] If such inaccuracy is typical, we may have underestimated the effect of C-SEP on t2DM in women. Third, the sample size for men could have resulted in the null findings we observed; however, our findings are consistent with those from several earlier studies.[6,7,9,12] Finally, we used imputed models to reduce bias due to missing values but we are uncertain about the extent to which values were missing at random.

Conclusions

Despite the limitations, this study has several strengths. We analyzed data from a large cohort and a prospective design which allowed examination of the effect of SEP on future risk of t2DM among African Americans. The life course approach yielded support for the social mobility hypotheses suggesting that the duration of exposure to social stressors may influence t2DM incidence, at least, among African American women. Future research is necessary to ascertain replicability of our findings and provide further insights into how socioeconomic stressors increase risk for t2DM in later life.

Acknowledgements

The Jackson Heart Study is supported by contracts HHSN268201300046C, HHSN268201300047C, HSN268201300048C, HHSN268201300049C, HHSN268201300050C from the National Heart, Lung, and Blood Institute and the National Institute on Minority Health and Health Disparities. The authors thank the participants and data collection staff of the Jackson Heart Study.

All procedures were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000. Informed consent was obtained from all participants included in the study.

Disclaimer

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention, or the US Department of Health and Human Services.

Conflict of Interest

No conflicts of interest to report.

Author Contributions

Research concept and design: Beckles, Bullard, Saydah, Imperatore, Correa; Acquisition of data: Beckles, Bullard, Imperatore, Correa; Data analysis and interpretation: Beckles, Bullard, Imperatore, Loustalot, Correa; Manuscript draft: Beckles, Saydah, Loustalot; Statistical expertise: Beckles, Bullard, Saydah, Correa; Acquisition of funding: Correa; Administrative: Beckles, Saydah, Loustalot; Supervision: Imperatore

References

1. CDC. National Diabetes Statistics Report, 2017. Atlanta, GA: U.S. Dept. of Health and Human Services; CDC, 2017. Last accessed October 11, 2018 from: https://www.cdc.gov/diabetes/pdfs/data/statistics/national-diabetes-statistics-report.pdf
2. Beckles GL, Zhu J, Moonesinghe R. Diabetes — United States, 2004 and 2008. *MMWR*. 2011;60(suppl):90-93. Last accessed October 11, 2018 from: https://www.cdc.gov/mmwr/preview/mmwrhtml/su6001a20.htm
3. Agardh E, Allebeck P, Hallqvist J, Moradi T, Sidorchuk A. Type 2 diabetes incidence and socio-economic position: a systematic review and meta-analysis. *Int J Epidemiol*. 2011;40(3):804-818. https://doi.org/10.1093/ije/dyr029 PMID:21335614
4. Kuh D, Ben-Shlomo Y. *A life course approach to chronic disease epidemiology*. Oxford: University Press; 1997.
5. Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C. Life course epidemiology. *J Epidemiol Community Health*. 2003;57(10):778-783. https://doi.org/10.1136/jech.57.10.778 PMID:14573579
6. Lidfeldt J, Li TY, Hu FB, Manson JE, Kawachi I. A prospective study of childhood and adult socioeconomic status and incidence of type 2 diabetes in women. *Am J*

Epidemiol. 2007;165(8):882-889. https://doi.org/10.1093/aje/kwk078 PMID:17284723
7. Maty SC, Lynch JW, Raghunathan TE, Kaplan GA. Childhood socioeconomic position, gender, adult body mass index, and incidence of type 2 diabetes mellitus over 34 years in the Alameda County Study. *Am J Public Health*. 2008;98(8):1486-1494. https://doi.org/10.2105/AJPH.2007.123653 PMID:18556612
8. Smith BT, Lynch JW, Fox CS, et al. Life-course socioeconomic position and type 2 diabetes mellitus: The Framingham Offspring Study. *Am J Epidemiol*. 2011;173(4):438-447. https://doi.org/10.1093/aje/kwq379 PMID:21242301
9. Demakakos P, Marmot M, Steptoe A. Socioeconomic position and the incidence of type 2 diabetes: the ELSA study. *Eur J Epidemiol*. 2012;27(5):367-378. https://doi.org/10.1007/s10654-012-9688-4 PMID:22539241
10. Stringhini S, Batty GD, Bovet P, et al. Association of lifecourse socioeconomic status with chronic inflammation and type 2 diabetes risk: the Whitehall II prospective cohort study. *PLoS Med*. 2013;10(7):e1001479. https://doi.org/10.1371/journal.pmed.1001479 PMID:23843750
11. Tsenkova V, Pudrovska T, Karlamangla A. Childhood socioeconomic disadvantage and prediabetes and diabetes in later life: a study of biopsychosocial pathways. *Psychosom Med*. 2014;76(8):622-628. https://doi.org/10.1097/PSY.0000000000000106 PMID:25272201
12. Cirera L, Huerta JM, Chirlaque MD, et al. Life-course social position, obesity and diabetes risk in the EPIC-Spain Cohort. *Eur J Public Health*. 2016;26(3):439-445. https://doi.org/10.1093/eurpub/ckv218 PMID:26635012
13. Maty SC, James SA, Kaplan GA. Life-course socioeconomic position and incidence of diabetes mellitus among blacks and whites: the Alameda County Study, 1965-1999. *Am J Public Health*. 2010;100(1):137-145. https://doi.org/10.2105/AJPH.2008.133892 PMID:19197084
14. McEwen BS, Gianaros PJ. Central role of the brain in stress and adaptation: links to socioeconomic status, health, and disease. *Ann N Y Acad Sci*. 2010;1186(1):190-222. https://doi.org/10.1111/j.1749-6632.2009.05331.x PMID:20201874
15. Geronimus AT, Hicken M, Keene D, Bound J. "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *Am J Public Health*. 2006;96(5):826-833. https://doi.org/10.2105/AJPH.2004.060749 PMID:16380565
16. Fuqua SR, Wyatt SB, Andrew ME, et al. Recruiting African-American research participation in the Jackson Heart Study: methods, response rates, and sample description. *Ethn Dis*. 2005;15(4)(suppl 6):S6-S18-S6-29. PMID:16317982
17. Krieger N, Williams DR, Moss NE. Measuring social class in US public health research: concepts, methodologies, and guidelines. *Annu Rev Public Health*. 1997;18(1):341-378. https://doi.org/10.1146/annurev.publhealth.18.1.341 PMID:9143723
18. Lloyd-Jones DM, Hong Y, Labarthe D, et al; American Heart Association Strategic Planning Task Force and Statistics Committee. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic Impact Goal through 2020 and beyond. *Circulation*. 2010;121(4):586-613. https://doi.org/10.1161/CIRCULATIONAHA.109.192703 PMID:20089546
19. Perkins JM, Subramanian SV, Davey Smith G, Özaltin E. Adult height, nutrition, and population health. *Nutr Rev*. 2016;74(3):149-165. https://doi.org/10.1093/nutrit/nuv105 PMID:26928678
20. van Buuren S, Boshuizen HC, Knook DL. Multiple imputation of missing blood pressure covariates in survival analysis. *Stat Med*. 1999;18(6):681-694. https://doi.org/10.1002/(SICI)1097-0258(19990330)18:63.0.CO;2-R PMID:10204197
21. StataCorp. *Stata Statistical Software: Release 13.* College Station, TX: StataCorp. LP; 2013.
22. Barker DJP. Sir Richard Doll Lecture. Developmental origins of chronic disease. *Public Health*. 2012;126(3):185-189. https://doi.org/10.1016/j.puhe.2011.11.014 PMID:22325676
23. Dominguez TP. Adverse birth outcomes in African American women: the social context of persistent reproductive disadvantage. *Soc Work Public Health*. 2011;26(1):3-16. https://doi.org/10.1080/10911350902986880 PMID:21213184
24. Steckel RH. The hidden cost of moving up: type 2 diabetes and the escape from persistent poverty in the American South. *Am J Hum Biol*. 2013;25(4):508-515. https://doi.org/10.1002/ajhb.22399 PMID:23784720
25. Kaplan GA, Ranjit N, Burgard SA. Lifting gates, lengthening lives: did Civil Rights policies improve the health of African American women in the 1960s and 1970s? In: Schoeni RF, House JSR, Kaplan GA, eds. *Making Americans Healthier: Social and Economic Policy as Health Policy.* New York: Russell Sage; 2008:145-169.
26. Liu H, Umberson D. Gender, stress in childhood and adulthood, and trajectories of change in body mass. *Soc Sci Med*. 2015;139:61-69. https://doi.org/10.1016/j.socscimed.2015.06.026 PMID:26151391
27. Sellers SL, Neighbors HW, Zhang R, Jackson JS. The impact of goal-striving stress on physical health of white Americans, African Americans, and Caribbean blacks. *Ethn Dis*. 2012;22(1):21-28. PMID:22774305
28. Sellers SL, Neighbors HW. Effects of goal-striving stress on the mental health of black Americans. *J Health Soc Behav*. 2008;49(1):92-103. https://doi.org/10.1177/002214650804900107 PMID:18418987
29. Cole ER, Omari SR. Race, class and the dilemmas of upward mobility for African Americans. *J Soc Issues*. 2003;59(4):785-802. https://doi.org/10.1046/j.0022-4537.2003.00090.x
30. Virtanen M, Ferrie JE, Tabak AG, et al. Psychological distress and incidence of type 2 diabetes in high-risk and low-risk populations: the Whitehall II Cohort Study. *Diabetes Care*. 2014;37(8):2091-2097. https://doi.org/10.2337/dc13-2725 PMID:24784831
31. Krieger N, Okamoto A, Selby JV. Adult female twins' recall of childhood social class and father's education: a validation study for public health research. *Am J Epidemiol*. 1998;147(7):704-708. https://doi.org/10.1093/oxfordjournals.aje.a009512 PMID:9554610
32. Batty GD, Lawlor DA, Macintyre S, Clark H, Leon DA. Accuracy of adults' recall of childhood social class: findings from the Aberdeen children of the 1950s study. *J Epidemiol Community Health*. 2005;59(10):898-903. https://doi.org/10.1136/jech.2004.030932 PMID:16166367
33. Straughen JK, Caldwell CH, Osypuk TL, Helmkamp L, Misra DP. Direct and proxy recall of childhood socio-economic position and health. *Paediatr Perinat Epidemiol*. 2013;27(3):294-302. https://doi.org/10.1111/ppe.12045 PMID:23574418