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## Late life socioeconomic status and hypertension in an aging cohort: the Atherosclerosis Risk in Communities Study

M. Maya McDoom<sup>a</sup>, Priya Palta<sup>b</sup>, Priya Vart<sup>a</sup>, Stephen P. Juraschek<sup>c</sup>, Anna Kucharska-Newton<sup>b</sup>, Ana V. Diez Roux<sup>d</sup>, and Josef Coresh<sup>a</sup>

<sup>a</sup>Johns Hopkins University Bloomberg School of Public Health and Welch Center for Prevention, Epidemiology and Clinical Research, Baltimore, Maryland <sup>b</sup>University of North Carolina at Chapel Hill Gillings School of Global Public Health, Chapel Hill, North Carolina <sup>c</sup>Beth Israel Deaconess Medical Center, Boston, Massachusetts <sup>d</sup>Drexel University Dornsife School of Public Health, Philadelphia, Pennsylvania, USA

### Abstract

**Objective:** To investigate the association between individual and area-level socioeconomic status and hypertension risk among individuals later in life.

**Methods:** We used Cox proportional hazards models to examine the association of socioeconomic status with incident hypertension using race-specific neighborhood socioeconomic status, median household income, and education among 3372 participants (mean age, 61 years) from the Atherosclerosis Risk in Communities Study at Visit 4 (1996–1998). Incident hypertension was defined as self-reported diagnosis or reported use of antihypertensive medications.

**Results:** Over a median follow-up time of 9.4 years, there were 1874 new cases of hypertension (62.1 per 1000 person-years). Overall, being in high as compared with low socioeconomic status categories was associated with a lower risk of developing hypertension in late life, with hazard ratios (95% confidence intervals) of 0.87 (0.77–0.98) for high neighborhood socioeconomic status tertile, 0.79 (0.69–0.90) for high individual income, and 0.75 (0.63–0.89) for college education after adjustment for traditional risk factors. These findings were consistent and robust whenever accounting for competing risks of all-cause mortality. No significant interactions by race and age (dichotomized at age 65) were observed.

**Conclusion:** Among participants free of hypertension in midlife, high neighborhood and individual socioeconomic status are associated with a decreased risk of incident hypertension. Our findings support population-level interventions, such as blood pressure screening at senior centers

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Correspondence to M. Maya McDoom, PhD, MPH, Johns Hopkins University Bloomberg School of Public Health and Welch Center for Prevention, Epidemiology and Clinical Research, 2024 E. Monument Street, Suite 2-600, Baltimore, MD 21207, USA. Tel: +1 410 955 4156; mmcdoom1@jh.edu.

Conflicts of interest

There are no conflicts of interest.

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and faith-based organizations, that are tailored to shift the distribution of blood pressure and reduce hypertension health inequalities among older adults.

### Keywords

aging; area level characteristics; blood pressure; hypertension; social environment; socioeconomic status

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## INTRODUCTION

Hypertension is a key determinant of morbidity and mortality in the United States population at both mid-life and older ages [1]. The risk of developing hypertension over the remaining lifetime of nonhypertensive adults aged 55 and 65 years is between 70 and 90% [2]. Moreover, the economic burden of hypertension, and the suboptimal effects on cardiovascular outcomes, such as stroke and heart failure, represents a major public health problem [1]. The risk of hypertension among this rapidly growing aging population is an increasingly important issue, given that by the year 2030 nearly 20% of the United States population will be of age 65 years or older [3] and the prevalence of hypertension at this age is high (63.6% using the older high-blood pressure guidelines and 75.6% using the new guidelines) [4]. Thus, there is a need to identify what factors contribute to the onset of hypertension in late life.

Socioeconomic status (SES) has been well established as a predictor of hypertension [5,6]. SES affects health throughout the lifespan [6]; however, its impact among older adults may be dependent on whether multiple dimensions of SES are examined. Findings in the National Health Interview Survey and National Health and Nutrition Examination Survey show that health disparities by individual income or education are not as strongly related to poorer health at older age compared with younger age [7,8]. Yet, other research has shown that higher income and net worth are associated with better health outcomes in older age [9]. Therefore, several different dimensions of SES may be relevant to the development of hypertension in late life. In addition, prospective analyses of SES and hypertension risk among individuals in late life are limited [10]. Much of the literature has examined how SES influences hypertension risk and onset at midlife [11–13], yet documented associations of SES and hypertension may not accurately represent similar patterns among older populations. Furthermore, factors such as retirement, death of a spouse, and deteriorating health can adversely affect the SES of aging adults [14]. Thus, the association between multiple SES dimensions and hypertension in late life could provide evidence for whether the effects of SES are relevant at older age and even among those who have yet to develop hypertension.

In an effort to examine these issues, we used data from the Atherosclerosis Risk in Communities Study (ARIC) to prospectively examine whether SES, both neighborhood-level and individual-level SES, is associated with greater incidence of hypertension in late life. We hypothesized that higher levels of neighborhood and individual SES will be associated with a lower incidence of hypertension in late life. We also hypothesized that,

although absolute levels of hypertension risks differ by SES, the associations between neighborhood, individual SES, and hypertension incidence will be similar by age.

## METHODS

### Study population and design

The ARIC study is a population-based cohort study of 15 972 participants aged 45–64 years, recruited in 1987–1989 from four United States communities (Washington County, Maryland; Forsyth County, North Carolina; Jackson, Mississippi; Minneapolis, Minnesota). Participants at two of the study sites, Washington County and Minneapolis are nearly all white; whereas the Forsyth County and Jackson sites recruited 15 and 100% black participants, respectively. A detailed description of the study has been published previously [15]. Briefly, visits 1–4 were conducted 3 years apart beginning in 1987–1989 and visit 5 occurred 14 years after visit 4 from 2011 to 2013. Clinical, social, and demographic data were collected at each visit. Semi-annual follow-up telephone calls are ongoing to maintain contact and assess the health status of the cohort. The fourth visit (1996–1998) served as the baseline for the present analysis and includes 11 656 participants. We define age at least 65 years as late life (40% of visit 4 participants were in late life).

Participants with prevalent cases of hypertension at visit 4 ( $n = 6507$ ) were excluded from analysis. Further exclusions from our analytic sample included: missing exposure or covariate data ( $n = 766$ ); incident hypertension prior to visit 4 (doctor diagnosis, medication use, or elevated measured blood pressure), but not currently hypertensive ( $n = 445$ ); or not contributing follow-up time because of nonresponse or death ( $n = 566$ ; Table S1, <http://links.lww.com/HJH/A903>). Prevalent cases of diagnosed hypertension were excluded and defined as elevated average of last two readings of measured blood pressure (SBP  $\geq 140$  mmHg or DBP  $\geq 90$  mmHg), self-reported antihypertensive medication use during the past 2 weeks, or self-report based on recalling a physician diagnosis. The institutional review boards of participating institutions approved the ARIC study protocol, and study participants provided written informed consent.

### Neighborhood and individual socioeconomic status

The exposures were neighborhood-level and individual-level SES. As described previously, neighborhood SES (nSES) was defined using a summary score that sums six z-score indicators of area characteristics at the census-tract level, based on the geocoded address at visit 4 of each participant [11]. Briefly the six domains included: median household income; median housing value, percentage of households with interest or rental income; proportion of adults greater than 25 years with a high school education; proportion of adults greater than 25 years with a college education; and proportion of adults greater than 16 years with executive, managerial, or professional occupations. A higher summary score represents a less deprived neighborhood environment. Race-specific tertiles were calculated because we observed large differences in the distribution of nSES by race.

Two domains of individual-level SES were measured: income and education. Individual-level household income was ascertained at visit 4 and categorized as less than \$25000, \$25

000 to less than \$50000, and at least \$50000 in 1996–1998 (\$50 000 in 1996 corresponds to \$77386 in 2017 based on a cumulative inflation of approximately 1.5-fold) [16]. Individual-level educational attainment was ascertained at visit 1 as education typically does not increase after midlife and was categorized as less than high school, high school graduate, and college or greater.

### Incident hypertension

Incident hypertension was the primary outcome of this analysis. Measured blood pressure was assessed only at visit 4 and not during follow-up. Thus, we defined incident hypertension as self-reported diagnosis or reported use of antihypertensive medications occurring after baseline (visit 4). Incident hypertension was ascertained from the annual telephone questionnaires (semi-annual starting 2012) through December 31, 2013 (the last follow-up date available). Response rates averaged approximately greater than 80% for each telephone follow-up throughout the ARIC study. Participants were classified as incident cases if they reported the use of antihypertensive medications or physician diagnoses of hypertension. Similar to previous ARIC analyses [17,18], we considered the date the participant reported hypertension as the date of diagnosis. Participants who did not develop hypertension were administratively censored at the date of last telephone response.

### Covariates

Age, sex, race, physical activity, smoking status, alcohol consumption, and BMI were based on self-reported data available at visit 4. Physical activity during leisure time (range 0–5; higher score indicates more activity) was assessed using the modified Baecke Physical Activity questionnaire [19]. Smoking status and alcohol consumption were characterized as current, former, or never. Standing height (meters) and weight (kilograms) were used to calculate BMI.

### Statistical analysis

Baseline characteristics of the study population were examined as means for continuous variables and proportions for categorical variables by age. ANOVA was used to compare continuous variables and chi-square tests were used for categorical variables. Kaplan–Meier plots were created to show the cumulative hazard of incident hypertension by race-specific tertiles of nSES summary score. Differences across categories were evaluated by the log-rank test. Cox proportional hazards models were used to examine the association between neighborhood-level and individual-level SES at baseline and incident hypertension. Previous analyses in the ARIC study have found that Cox proportional hazards results were similar to discrete proportional hazards results [11]. To account for within-census tract correlation, all models included clustering at census tract through robust standard errors estimation using a clustered sandwich estimator [20].

There were large differences in the distribution of neighborhood SES by race. Furthermore, all black participants were sampled from two study sites: the Jackson and Forsyth sites. Therefore, we used race-specific neighborhood SES categories. Extant literature has established that age is a key predictor of blood pressure [21,22], thus we present multivariable results stratified by age categories, and tested the interaction between SES

measures and age to assess for effect modification. Prior research has suggested that controlling for individual-level variables that determine neighborhood selection, such as education or income, results in the reduction of meaningful neighborhood variation [23,24]. Therefore, separate multivariable models were constructed for neighborhood and individual SES measures. Sequential models were created to progressively adjust for variables more proximal to SES and incident hypertension. In the overall analyses, two models were evaluated: Model 1 adjusted for demographic factors (sex and age); Model 2 further adjusted for lifestyle factors (alcohol consumption, smoking, physical activity, BMI). In the age-stratified analysis, Model 1 was adjusted for sex and race; Model 2 further adjusted for lifestyle factors. We performed a test for trend across categories of neighborhood SES, individual income, and years of education, using the median values of each variable.

Supplemental analyses were conducted to address potential misclassification of hypertension, race-stratified multivariable analyses, competing risk of death, and the independent neighborhood effect. At baseline, self-reported hypertension was compared with measured blood pressure and checking medication bottles. Elevated measured blood pressure was classified as SBP greater than 140 mmHg or DBP greater than 90 mmHg. The sensitivity, specificity, and positive predictive value were assessed for all participants with blood pressure measurements, medication, and self-reported hypertension at visit 4. We conducted a competing risk analysis using the Fine and Gray competing-risks analysis [25] by simultaneously evaluating hazards for incident hypertension while accounting for death. As there were also large differences in individual income, we conducted analyses stratified by race and tested the interaction between SES measures and race. Additionally, we used multilevel Weibull survival models including a neighborhood-level random intercept to explore the independent effect of neighborhood SES, apart from individual SES by controlling for individual income and education [26].

All statistical analyses were performed using Stata, version 14.0 (StataCorp LP, College Station, Texas, USA). Statistical significance was defined a priori as a two-sided *P* value less than 0.05.

## RESULTS

### Baseline characteristics

In our study population of 3372 participants, the mean age was  $61 \pm 5.5$  years and 12% were black. Compared with whites, blacks were more likely to live in disadvantaged neighborhoods and have lower individual SES (Table 1). Whites age at least 65 were more likely to be men, never drinkers, never or former smokers, and have lower BMI compared with whites age less than 65. Leisure index and smoking status did not differ among white participants. No significant difference was observed by sex, leisure index, drinking status, or smoking status among black participants. On average, the distribution of nSES (Fig. 1) shifted towards lower values among black participants than white participants. At baseline we examined the validity of the outcome, self-reported hypertension. Compared with hypertension based on blood pressure measurement or of checking medication bottles, self-reported hypertension has 72.9% sensitivity, 94.2% specificity, 75.6% negative predictive value, and 93.4% positive predictive value (Table S2, <http://links.lww.com/HJH/A903>).

Similar patterns were observed for specificity and sensitivity whenever comparing hypertension based on blood pressure measurement or of checking medication bottles to self-reported hypertension by individual-level and area-level SES (Tables S3-S5, <http://links.lww.com/HJH/A903>).

### Overall associations between socioeconomic status and incident hypertension

Over a median follow-up time of 9.4 years, there were 1874 incident cases of hypertension (62.1 per 1000 person-years). The Kaplan–Meier survival curves according to race-specific nSES tertiles (Fig. 2) suggest that the cumulative incidence of hypertension in late life is high; after 10 years of follow-up, the cumulative incidence was approximately 45% among whites and 55% among the blacks who were free of hypertension at baseline. Overall, participants in the highest nSES tertile had a significantly lower cumulative incidence of hypertension than those in the intermediate or low nSES tertile groups (log-rank test,  $P = <0.01$ ). A similar relationship was seen among the white participants ( $P = <0.01$ ); however, the associations were not statistically significant among the smaller number of black participants ( $P = 0.48$ ). Incidence rates demonstrated a lower absolute risk of hypertension among those in higher SES (Table 2). In a Cox proportional hazards survival analysis, higher area-level and individual-level SES categories were associated with lower risk of developing hypertension in late life for the overall sample in Model 1 and in the fully adjusted models (Model 2, Table 2).

### Age-stratified associations between socioeconomic status and incident hypertension

Age-stratified results (Table 2) also demonstrated a decreased risk of incident hypertension in later life among those with higher SES. Among those age at least 65 years, high nSES compared with low nSES was inversely associated with incident hypertension, but nonsignificant in both Model 1 and Model 2. However, among those younger than age 65 years, high nSES as compared with low nSES was significantly associated with a 14% lower risk of incident hypertension even in the adjusted model. In both age groups, high vs. low individual-level income was significantly associated with a decreased risk of incident hypertension in late life (Model 1;  $P_{\text{trend}} = 0.003$  and  $<0.001$ , respectively). The results for individual income showed similar results but the association remained significant only for those age at least 65 years [hazard ratio 0.72, 95% confidence interval (CI) 0.55–0.94] whereas it was not statistically significant among those age less than 65 (hazard ratio 0.85; 95% CI 0.72–1.01). High and intermediate vs. low-individual education categories were associated with a lower risk of developing late life hypertension among those age less than 65 (Model 1:  $P_{\text{trend}} <0.001$ ); these associations were moderately attenuated but remained significant after adjustment for key covariates in Model 2 ( $P_{\text{trend}} = 0.001$ ). Similar, but nonsignificant associations were observed among those age at least 65. No statistical interaction was observed between age and SES for the risk of incident hypertension ( $P$  value for interactions: nSES = 0.96, education = 0.18; and income = 0.42 respectively, Model 2).

### Supplemental analyses

In race-stratified analysis, we observed that membership in high-SES categories was associated with a decreased relative risk of developing hypertension in late life at least for white participants (Table S6, <http://links.lww.com/HJH/A903>). However, incidence rates of

hypertension were substantially higher for blacks compared with whites. Differences in hazard ratios for nSES or individual-level income and education attainment in blacks were in the same direction as whites, but weaker and not statistically significant. Interactions by race were not statistically significant. Associations were also similar in men and women (Table S7, <http://links.lww.com/HJH/A903>). Competing risk analyses showed that inferences remained unchanged and were consistent with the traditional Cox models results, suggesting that mortality occurring during the study did not bias the results (Table S8, <http://links.lww.com/HJH/A903>). In multilevel models, whenever area SES was adjusted for individual-level income and education, it did not carry additional statistically independent risk (Table S9, <http://links.lww.com/HJH/A903>). Further adjustment for baseline SBP within the normotensive range did not materially change the results (not shown).

## DISCUSSION

In the present population-based sample, our results suggest that higher area-level and individual-level SES indicators were associated with lower hypertension incidence in later life. The inclusion of clinical and behavioral factors attenuated these associations but did not fully explain them. Comparable with prior research, our findings suggest a gradient of hypertension risk across tertiles of area-level and individual-level SES [21,27–29] extending the findings to those who have not developed hypertension by mid-life. Specifically, those in the most advantaged socioeconomic neighborhoods (high nSES), highest incomes, and highest educational attainment had a lower incidence and absolute risk of hypertension compared with their less advantaged (intermediate and low nSES) counterparts at older age.

Although contemporary research has focused on social and physical environment characteristics, which might be associated with hypertension, such as safety or cohesion [13,27,30], relatively few prospective studies have been able to examine whether both area-level and individual-level SES gradients continue into late life. Prior studies report cross-sectional associations between poor neighborhood SES or characteristics and sub-optimal health outcomes among older adults [10,27]; prospective associations between neighborhood and individual-level SES disadvantage in midlife [11]; and associations between limited measures of SES (most commonly individual income or education) and hypertension among older adults [31]. Although nSES is a more general measure of neighborhood context, our findings have extended the understanding of how multiple dimensions of SES are associated with hypertension in late-life. First, at least among this aging cohort and among whites, consistent results in both individual-level as well as area-level measures of SES suggest the robust contribution of SES to hypertension incidence in late life. These findings are consistent with recent mortality trends among middle-age white adults, and underscore the importance of cumulative socioeconomic disadvantage and the development of chronic disease [32]. Second, we observed that the association between SES and incident hypertension was independent of traditional risk factors. Population-based interventions partnered with civic or senior organizations focused on the adoption of healthy behaviors can reduce the burden of high blood pressure [33] and may be tailored to those in disadvantaged SES groups in late life. In addition, optimizing hypertension treatment in disadvantaged SES groups at older age, whenever risk of complications is high, could be helpful.

A number of considerations are also relevant to understanding the independent effects of neighborhoods on hypertension. In our main analyses, we did not include neighborhood and individual SES in the same models to avoid over-adjustment. In sensitivity analyses, we found that whenever nSES was adjusted for individual-level SES, the association with incident hypertension at older age was mostly explained by the individual-level SES measures. Inclusion of individual-level SES could underestimate the nSES relationship with incident hypertension because individual SES may determine neighborhood selection and classification of neighborhood SES is subject to error [23,24,34]. Methodologically, this may limit the interpretation of ‘independent’ effects of neighborhoods on health outcomes [34].

The survivor effects phenomenon might also be relevant to our findings of a gradient hypertension risk across tertiles of area-level and individual-level SES. Survivor effects occur whenever individuals who have a poorer SES profile and who have more biological risk may be more vulnerable to risk of disease or mortality at a younger age, whereas those with less risk and a better SES profile are more likely to survive or be disease free at old age – even among the poor [35,36]. Individuals who have survived or are disease free may be more resilient or have adapted to their social position and environments than those who did not survive or are not disease free [37,38]. Our findings suggest that even if survivor effects occur, those who are more resilient in the lower SES groups still suffer from a continued disadvantage for disease.

Several mechanisms may explain our findings of a SES gradient relating to health in late life. These relationships might be mediated by individual risk factors, psychosocial, or physiological processes. Individual risk factors for hypertension (e.g. obesity, smoking, physical inactivity, diet) are influenced by individual behaviors as well as the neighborhood environment, which may mediate the relationships between SES and hypertension in later life. Those living in disadvantaged neighborhoods may have limited access to social and economic resources, recreational facilities, and healthy foods [34,39,40]. which may directly or indirectly affect individuals’ ability to engage in healthy behaviors. Poor SES among older adults may also reflect the cumulative history of low SES, cumulative exposure to adverse conditions associated with low SES, or a recent shift in SES because of retirement [9,41]. Psychosocial or physiological processes, such as chronic inflammation and stress, which are known to have an adverse impact on hypertension and subsequent cardiovascular disease [42–44]. may also be additional pathways by which SES affects hypertension [45,46]. We were unable to determine whether this occurred in our study and future inquiries should examine this more closely among older cohorts.

There are several limitations of this study to be considered in the interpretation of our results. The neighborhood SES measure was defined at the census tract level, which may not correspond to the actual geographic boundaries of a neighborhood as perceived by participants. In addition, both area-level and individual-level SES were assessed at mid-life and may not accurately capture SES changes over the life course. More specific neighborhood features, such as neighborhood cohesion or change in neighborhood characteristics over time, may be more relevant mechanisms that are associated with hypertension. We studied only four communities with most blacks coming from Jackson



Mississippi limiting generalizability. Although we defined hypertension using the best data available, the imperfect sensitivity of self-reported hypertension could have biased the results. Diagnosis of hypertension could also be related to frequency of medical care and quality of blood pressure measurement which could vary by SES; however, we were unable to assess these factors. The exclusion of a large proportion of black participants because of prevalent hypertension at study baseline reduced the number of blacks susceptible to hypertension in late life and made our estimates for this group underpowered. Limited SES variability among the black participants could impact our ability to detect consistent results in this group. Finally, wealth measured as total value of assets rather than personal income may be a better indicator of personal SES in late life, as income declines considerably during retirement.

The current study has several strengths. It is a large multicenter, community-based prospective cohort study with more than a decade of follow-up and rigorous measurement of exposures and confounders, measurement of SES, clinical indicators, and risk factors. Furthermore, comprehensive data collection protocols afford the opportunity to assess SES on individual and neighborhood levels.

From a clinical viewpoint, the present findings suggest that incident hypertension is associated with an SES gradient among older adults who had not developed hypertension by mid-life even after adjustment for classic behavioral and lifestyle factors. An increased focus on determining the processes through which SES over the life course shapes the development of hypertension at older ages is critical to reducing disease burden. In addition, testing strategies to manage blood pressure using the new stricter blood pressure guidelines as the population ages has the potential to shift or delay hypertension onset and reduce risk among aging populations.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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## Abbreviations:

<b>ARIC</b>	Atherosclerosis Risk in Communities Study
<b>CI</b>	confidence interval
<b>nSES</b>	neighborhood socioeconomic status

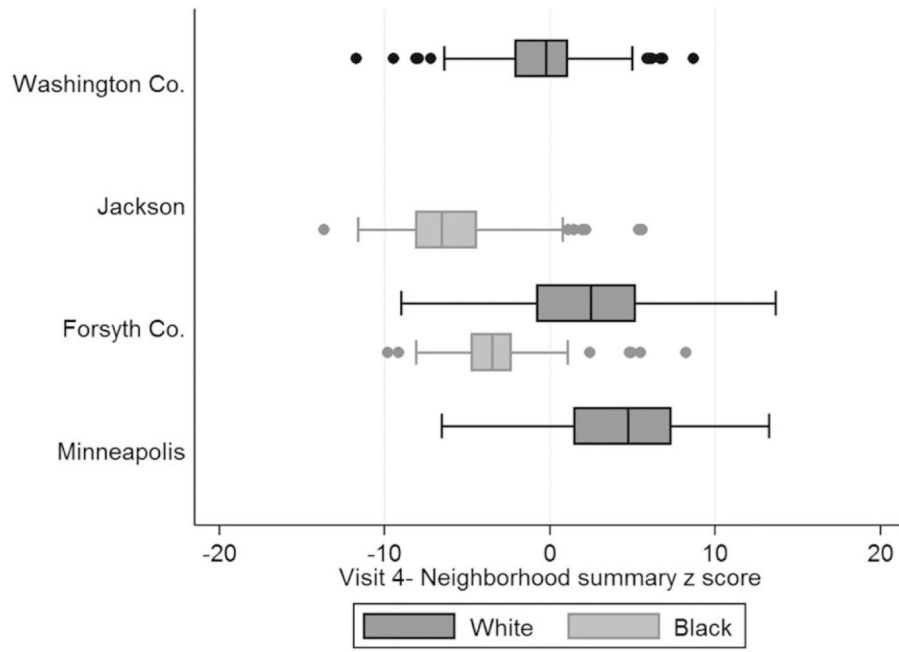
SES socioeconomic status

## REFERENCES

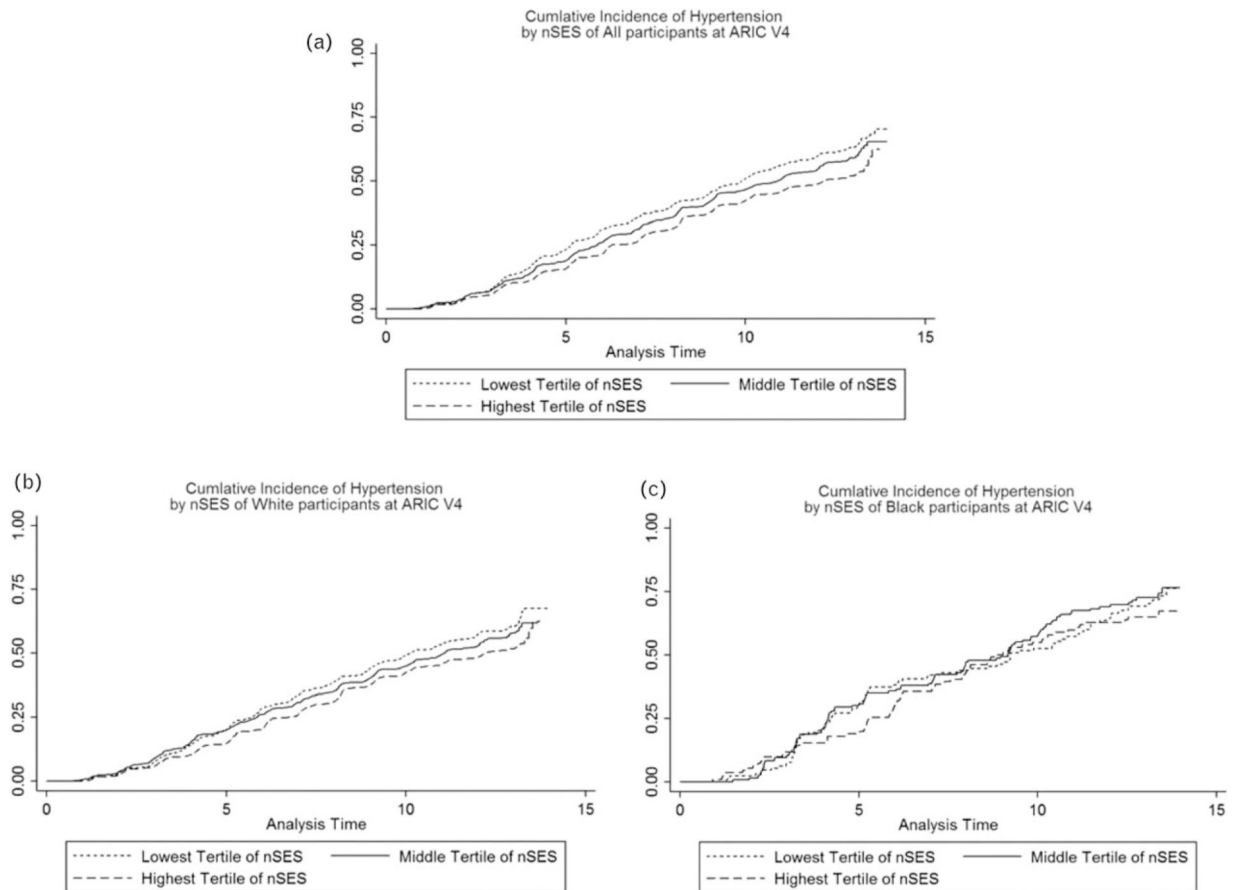
1. Benjamin EJ, Blaha MJ, Chiuve SE, Cushman M, Das SR, Deo R, et al., American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics—2017 update: a report from the American Heart Association. *Circulation* 2017; 135:e146–e603. [PubMed: 28122885]
2. Vasan RS, Beiser A, Seshadri S, Larson MG, Kannel WB, D'Agostino RB, Levy D. Residual lifetime risk for developing hypertension in middle-aged women and men: The Framingham Heart Study. *JAMA* 2002; 287:1003–1010. [PubMed: 11866648]
3. Centers for Disease Control, Prevention. Public health and aging: trends in aging—United States and worldwide. *JAMA* 2003; 289: 1371–1373. [PubMed: 12636453]
4. Muntner P, Carey RM, Gidding S, Jones DW, Taler SJ, Wright JT Jr, Whelton PK. Potential U. S. population impact of the 2017 American College of Cardiology/American Heart Association High Blood Pressure Guideline. *Circulation* 2018; 71:109–118.
5. Colhoun H, Hemingway H, Poulter N. Socio-economic status and blood pressure: an overview analysis. *J Hum Hypertens* 1998; 12: 91–110. [PubMed: 9504351]
6. James SA, Van Hoewyk J, Belli RF, Strogatz DS, Williams DR, Raghunathan TE. Life-course socioeconomic position and hypertension in African American men: the Pitt County study. *Am J Public Health* 2006; 96:812–817. [PubMed: 16571689]
7. Smith JP. Unraveling the SES: health connection. *Popul Dev Rev* 2004; 30:108–132.
8. Seeman T, Merkin SS, Crimmins E, Koretz B, Charette S, Karlamangla A. Education, income and ethnic differences in cumulative biological risk profiles in a national sample of US adults: NHANESIII (1988–1994). *Soc Sci Med* 2008; 66:72–87. [PubMed: 17920177]
9. Xu X, Liang J, Bennett JM, Botosaneanu A, Allore HG. Socioeconomic stratification and multidimensional health trajectories: evidence of convergence in later old age. *J Gerontol B Psychol Sci Soc Sci* 2015; 70:661–671. [PubMed: 25161216]
10. Menec VH, Shooshtari S, Nowicki S, Fournier S. Does the relationship between neighborhood socioeconomic status and health outcomes persist into very old age? A population-based study. *J Aging Health* 2010; 22:27–47. [PubMed: 20048126]
11. Diez Roux AV, Chambless L, Merkin SS, Arnett D, Eigenbrodt M, Nieto FJ, et al. Socioeconomic disadvantage and change in blood pressure associated with aging. *Circulation* 2002; 106:703–710. [PubMed: 12163431]
12. Rose KM, Newman B, Tyroler HA, Szklo M, Arnett D, Srivastava N. Women, employment status, and hypertension: cross-sectional and prospective findings from the Atherosclerosis Risk in Communities (ARIC) Study. *Ann Epidemiol* 1999; 9:374–382. [PubMed: 10475537]
13. Morenoff JD, House JS, Hansen BB, Williams DR, Kaplan GA, Hunte HE. Understanding social disparities in hypertension prevalence, awareness, treatment, and control: the role of neighborhood context. *Soc Sci Med* 2007; 65:1853–1866. [PubMed: 17640788]
14. Saeed BI, Xicang Z, Yawson AE, Nguah SB, Nsawah-Nuamah NN. Impact of socioeconomic status and medical conditions on health and healthcare utilization among aging Ghanaians. *BMC Public Health* 2015; 15:276. [PubMed: 25884931]
15. The ARIC Investigators. The Atherosclerosis Risk in Communities (ARIC) Study: design and objectives. *Am J Epidemiol* 1989; 129:687–702. [PubMed: 2646917]
16. Bureau of Labor Statistics, United States Department of Labor CPI Inflation Calculator. Available at: [https://www.bls.gov/data/inflation\\_calculator.htm](https://www.bls.gov/data/inflation_calculator.htm). [Accessed 17 February 2017].
17. Juraschek SP, Bower JK, Selvin E, Shantha S, Palamaner G, Hoogeveen RC, et al. Plasma lactate and incident hypertension in the Atherosclerosis Risk in Communities Study. *Am J Hypertens* 2015; 28:216–224. [PubMed: 24994607]
18. Bower JK, Lazo M, Matsushita K, Rubin J, Hoogeveen RC, Ballantyne CM, Selvin E. N-terminal pro-brain natriuretic peptide (NT-proBNP) and risk of hypertension in the Atherosclerosis Risk in Communities (ARIC) Study. *Am J Hypertens* 2015; 28:1262–1266. [PubMed: 25783741]

19. Baecke JA, Burema J, Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. *Am J Clin Nutr* 1982; 36:936–942. [PubMed: 7137077]
20. Williams RL. A note on robust variance estimation for cluster-correlated data. *Biometrics* 2000; 56:645–646. [PubMed: 10877330]
21. Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, Cushman M, et al. Heart disease and stroke statistics—2015 update A report from the American Heart Association. *Circulation* 2015; 131:e29–e322. [PubMed: 25520374]
22. Whelton PK. Epidemiology and the prevention of hypertension. *J Clin Hypertens* 2004; 6:636–642.
23. Oakes JM. The (mis)estimation of neighborhood effects: causal inference for a practicable social epidemiology. *Soc Sci Med* 2004; 58: 1929–1952. [PubMed: 15020009]
24. Glass TA, Bilal U. Are neighborhoods causal? Complications arising from the ‘stickiness’ of ZNA. *Soc Sci Med* 2016; 166:244–253. [PubMed: 26830654]
25. Fine JP, Gray RJ. A proportional hazards model for the subdistribution of a competing risk. *J Am Stat Assoc* 1999; 94:496–509.
26. Banerjee S, Carlin B, Gelfand AE. Hierarchical modeling and analysis for spatial data, 2nd ed. Florida: CRC Press; 2014.
27. Mujahid MS, Diez Roux AV, Morenoff JD, Raghunathan TE, Cooper RS, Ni H, Shea S. Neighborhood characteristics and hypertension. *Epidemiology* 2008; 19:590–598. [PubMed: 18480733]
28. Conen D, Glynn RJ, Ridker PM, Buring JE, Albert MA. Socioeconomic status, blood pressure progression, and incident hypertension in a prospective cohort of female health professionals. *Eur Heart J* 2009; 30:1378–1384. [PubMed: 19297384]
29. He J, Muntner P, Chen J, Roccella EJ, Streiffer RH, Whelton PK. Factors associated with hypertension control in the general population of the United States. *Arch Intern Med* 2002; 162:1051–1058. [PubMed: 11996617]
30. Fan AZ, Strasser SM, Zhang X, Fang J, Crawford CG. State socioeconomic indicators and self-reported hypertension among US Adults, 2011 Behavioral Risk Factor Surveillance System. *Prev Chronic Dis* 2015; 12:E27. [PubMed: 25719217]
31. He J, Klag MJ, Appel LJ, Charleston J, Whelton PK. Seven-year incidence of hypertension in a cohort of middle-aged African Americans and Whites. *Hypertension* 1998; 31:1130–1135. [PubMed: 9576125]
32. Case A, Deaton SA. Mortality and Morbidity in the 21st Century. Brookings Institute; 2017:63 <https://www.brookings.edu/bpea-articles/mortality-and-morbidity-in-the-21st-century/>. [Accessed 24 August 2017].
33. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. Seventh Report of the Joint National Committee on Prevention, Detection, and Evaluation, and Treatment of High Blood Pressure. *Hypertension* 2003; 42:1206–1252. [PubMed: 14656957]
34. Diez Roux AV. Investigating neighborhood and area effects on health. *Am J Public Health* 2001; 91:1783–1789. [PubMed: 11684601]
35. Manton KG, Stallard E. Methods for evaluating the heterogeneity of aging processes in human populations using vital statistics data: explaining the black/white mortality crossover by a model of mortality selection. *Hum Biol* 1981; 53:47–67. [PubMed: 7239492]
36. Crimmins EM, Kim JK, Seeman TE. Poverty and biological risk: the earlier ‘aging’ of the poor. *Gerontol A Biol Sci Med Sci* 2009; 64:286–292.
37. Roepke SK, Grant I. Toward a more complete understanding of the effects of personal mastery on cardiometabolic health. *Health Psychol Off J Div Health Psychol Am Psychol Assoc* 2011; 30:615–632.
38. Anderson NB, Armstead CA. Toward understanding the association of socioeconomic status and health: a new challenge for the biopsychosocial approach. *Psychosom Med* 1995; 57:213–225. [PubMed: 7652122]
39. Diez Roux AV. Residential environments and cardiovascular risk. *J Urban Health Bull N Y Acad Med* 2003; 80:569–589.

40. Pickett KE, Pearl M. Multilevel analyses of neighbourhood socioeconomic context and health outcomes: a critical review. *J Epidemiol Community Health* 2001; 55:111–122. [PubMed: 11154250]
41. Galobardes B, Smith GD, Lynch JW. Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. *Ann Epidemiol* 2006; 16: 91–104. [PubMed: 16257232]
42. Solak Y, Afsar B, Vaziri ND, Aslan G, Yalcin CE, Covic A, Kanbay M. Hypertension as an autoimmune and inflammatory disease. *Hypertens Res* 2016; 39:567–573. [PubMed: 27053010]
43. Doan SN, Dich N, Evans GW. Stress of stoicism: low emotionality and high control lead to increases in allostatic load. *Appl Dev Sci* 2016; 20:310–317.
44. Agyemang C, van Hooijdonk C, Wendel-Vos W, Ujcic-Voortman JK, Lindeman E, Stronks K, Droomers M. Ethnic differences in the effect of environmental stressors on blood pressure and hypertension in the Netherlands. *BMC Public Health* 2007; 7:118. [PubMed: 17587458]
45. Black PH, Garbutt LD. Stress, inflammation and cardiovascular disease. *J Psychosom Res* 2002; 52:1–23. [PubMed: 11801260]
46. Pickering T Cardiovascular pathways: socioeconomic status and stress effects on hypertension and cardiovascular function. *Ann N Y Acad Sci* 1999; 896:262–277. [PubMed: 10681903]



**FIGURE 1.** Distribution of neighborhood socioeconomic status score by study site and race.



**FIGURE 2.**

Kaplan-Meier plots examining cumulative incidence of hypertension by neighborhood socioeconomic status tertile for (a) all participants ( $P=0.0001$ ), (b) whites ( $P=0.0002$ ), and (c) blacks ( $P=0.48$ ). nSES, neighborhood socioeconomic status.

**TABLE 1.** Baseline characteristics of Atherosclerosis Risk in Communities Study participants at visit 4 (1996–1998) by race and age

	White participants		Black participants		Overall
	Age less than 65 years	Age at least 65 years	Age less than 65 years	Age at least 65 years	
N	2037	941	307	87	3372
nSES median (Q1, Q3)	2.0 (−0.2, 5.4)	1.3 (−0.7, 4.7)	−5.6 (−8.1, −2.3)	−6.6 (−9.6, −4.5)	1.3 (−1.7, 4.8)
Income in 1996–1998 <sup>a</sup> , n (%)					
<\$25000, \$25000	260 (12.8)	296 (31.5)	120 (39.1)	55 (63.2)	731 (21.7)
\$25000 to less than \$50 000	653 (32.1)	406 (43.1)	104 (33.9)	19 (21.8)	1 182 (35.1)
\$50000	1124 (55.2)	239 (25.4)	83 (27.0)	13 (14.9)	1459 (43.3)
Education, n (%)					
Less than high school	166 (8.1)	157 (16.7)	56 (18.2)	31 (35.6)	410 (12.2)
High school graduate	726 (35.6)	311 (33.0)	64 (20.8)	16 (18.4)	1117 (33.1)
College or greater	1145 (56.2)	473 (50.3)	187 (60.9)	40 (46.0)	1845 (54.7)
Female sex, n (%)	1132 (55.6)	485 (51.5)	186 (60.6)	50 (57.5)	1853 (55.0)
Drinker, n (%)					
Current drinker	1302 (63.9)	492 (52.3)	117 (38.1)	22 (25.3)	1933 (57.3)
Former drinker	461 (22.6)	273 (29.0)	107 (34.9)	38 (43.7)	879 (26.1)
Never drinker	274 (13.5)	176 (18.7)	83 (27.0)	27 (31.0)	560 (16.6)
Physical Activity Index, mean ± SD	2.5 ± 0.5	2.5 ± 0.5	2.2 ± 0.6	2.1 ± 0.6	2.5 ± 0.5
Smoking, n (%)					
Current smoker	342 (16.8)	127 (13.5)	54 (17.6)	14 (16.1)	537 (15.9)
Former smoker	874 (42.9)	435 (46.2)	113 (36.8)	30 (34.5)	1452 (43.1)
Never smoker	821 (40.3)	379 (40.3)	140 (45.6)	43 (49.4)	1383 (41.0)
BMI, median (Q1, Q3)	26.6 (24.0, 29.7)	26.1 (23.7, 28.8)	28.1 (25.5, 31.3)	26.4 (23.8, 28.7)	26.6 (24.0, 29.7)

All age comparisons were significant at  $P < 0.05$  with the exception for whites: Physical Activity Index,  $P = 0.36$ ; smoking,  $P = 0.05$ . Exceptions for blacks: female sex,  $P = 0.60$ ; drinker  $P = 0.08$ ; Physical Activity Index,  $P = 0.13$ ; smoking,  $P = 0.82$ . Q1, quartile 1; Q3, quartile 3. nSES, neighborhood SES; SD, standard deviation; SES, socioeconomic status.

<sup>a</sup> \$50 000 in 1996 is \$77 386 in 2017; ~1.5 times the 1996 dollar.

TABLE 2.

Risk for developing incident hypertension in late-life for total sample and age by socioeconomic status

	Total sample (1874 cases/3372 participants)			Age at least 65 at baseline (568 cases/1028 participants)			Age less than 65 at baseline (1306 cases/2344 participants)		
	Incidence rate <sup>a</sup>	Model 1, <sup>b</sup> hazard ratio (95% CI)	Model 2, <sup>c</sup> hazard ratio (95% CI)	Incidence rate <sup>a</sup>	Model 1, <sup>b</sup> hazard ratio (95% CI)	Model 2, <sup>c</sup> hazard ratio (95% CI)	Incidence rate <sup>a</sup>	Model 1, <sup>b</sup> hazard ratio (95% CI)	Model 2, <sup>c</sup> hazard ratio (95% CI)
nSES <sup>d</sup>									
Highest Tertile	55.00	0.79 (0.70–0.90)**	0.87 (0.77–0.98)*	57.78	0.82 (0.66–1.01)	0.85 (0.69–1.05)	54.07	0.79 (0.68–0.91)**	0.86 (0.74–0.99)*
Middle Tertile	64.24	0.94 (0.84–1.06)	0.97 (0.86–1.09)	65.63	0.93 (0.78–1.12)	0.94 (0.79–1.13)	63.65	0.94 (0.82–1.08)	0.97 (0.85–1.11)
Lowest Tertile	67.58	REF	REF	68.87	REF	REF	66.94	REF	REF
<i>P</i> trend <sup>e</sup>	–	<0.001	0.001	–	0.308	0.713	–	<0.001	0.005
Individual-level income									
\$50 000+	54.38	0.72 (0.64–0.82)**	0.79 (0.69–0.90)**	53.35	0.65 (0.49–0.86)**	0.72 (0.55–0.94)*	54.59	0.76 (0.65–0.89)**	0.85 (0.72–1.01)
\$25 000–\$50 000	66.70	0.91 (0.81–1.02)	0.95 (0.84–1.06)	62.96	0.79 (0.66–0.95)*	0.83 (0.69–1.01)	68.78	0.99 (0.84–1.17)	1.06 (0.89–1.26)
<\$25 000	72.17	REF	REF	76.04	REF	REF	68.90	REF	REF
<i>P</i> trend <sup>e</sup>	–	<0.001	<0.001	–	0.003	0.01	–	<0.001	0.001
Individual-level education									
College graduate	56.81	0.69 (0.58–0.81)**	0.75 (0.63–0.89)**	58.16	0.77 (0.57–1.03)	0.80 (0.60–1.06)	56.32	0.65 (0.55–0.78)**	0.71 (0.59–0.86)**
High school graduate	65.53	0.82 (0.69–0.97)*	0.87 (0.73–1.03)	70.07	0.98 (0.73–1.32)	0.99 (0.75–1.31)	63.78	0.78 (0.64–0.94)**	0.82 (0.67–0.99)*
Less than high school	80.29	REF	REF	74.14	REF	REF	85.38	REF	REF
<i>P</i> trend <sup>e</sup>	–	<0.001	<0.001	–	0.016	0.034	–	<0.001	0.001

<sup>f</sup> *P* value for interactions: age and nSES, 0.96; age and income, 0.18; age and education, 0.42. CI, confidence interval. nSES, neighborhood socioeconomic status.

<sup>a</sup> Incidence rate reported per 1000 person-years.

<sup>b</sup> Adjusted for sex, race.

<sup>c</sup> Model 1 with adjustments for covariates: alcohol use, leisure activity, smoking status, BMI.

<sup>d</sup> Race-specific nSES cutoffs were: highest tertile (white: 4.07 to 13.64; blacks: –4.15 to 8.20), middle tertile (white: 0.40 to 4.06; black –7.33 to –4.47), lowest tertile (white: –11.73 to 0.39; black: –13.67 to –7.67).

<sup>e</sup> *P* value for trend evaluated using the category median for nSES, individual income, and year of education.

\* *P*<0.05.

\*\* *P*<0.001.