ORIGINAL RESEARCH

Associations of Smoke-Free Policies in Restaurants, Bars, and Workplaces With Blood Pressure Changes in the CARDIA Study

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***Background*—**Smoke-free legislation has been associated with reductions in secondhand smoke exposure and cardiovascular disease. However, it remains unknown whether smoke-free policies are associated with reductions in blood pressure (BP).

***Methods and Results*—**Longitudinal data from 2606 nonsmoking adult participants of the CARDIA (Coronary Artery Risk Development in Young Adults) Study (1995–2011) were linked to state, county, and local-level 100% smoke-free policies in bars, restaurants, and/or nonhospitality workplaces based on participants' census tract of residence. Mixed-effects models estimated associations of policies with BP and hypertension trajectories over 15 years of follow-up. Fixed-effects regression estimated associations of smoke-free policies with within-person changes in systolic and diastolic BP and hypertension. Models were adjusted for sociodemographic, health-related, and policy/geographic covariates. Smoke-free policies were associated with between-person differences and within-person changes in systolic BP. Participants living in areas with smoke-free policies had lower systolic BP on average at the end of follow-up compared with those in areas without policies (adjusted predicted mean differences [in mm Hg]: restaurant: −1.14 [95% confidence interval: −2.15, −0.12]; bar: −1.52 [−2.48, −0.57]; workplace: −1.41 [−2.32, −0.50]). Smoke-free policies in restaurants and bars were associated with mean within-person reductions in systolic BP of −0.85 (−1.61, −0.09) and −1.08 (−1.82, −0.34), respectively. Only restaurant policies were associated with a significant within-person reduction in diastolic BP, of −0.58 (−1.15, −0.01).

***Conclusions*—**While the magnitude of associations was small at the individual level, results suggest a potential mechanism through which reductions in secondhand smoke because of smoke-free policies may improve population-level cardiovascular health. **(*J Am Heart Assoc*. 2018;7:e009829. DOI: 10.1161/JAHA.118.009829.)**

Key Words: blood pressure • epidemiology • health policy • hypertension, tobacco control • smoking

Tobacco smoke exposure results in nearly 100 000 deaths per year in the United States because of coronary heart disease.[1] Many cities, states, and countries have passed legislation banning smoking in public places including restaurants, bars, and workplaces in an effort to minimize population-level exposure to tobacco smoke. Smoke-free policies are intended to decrease secondhand smoke (SHS) exposure and encourage current smokers to quit or reduce the amount they smoke. Systematic reviews indicate that smoke-free legislation is associated with reductions in SHS.[2] Results for smoking are somewhat less conclusive, although many studies have found smoke-free policies to be associated with reduced smoking prevalence and intensity.[2]

Compared with other smoking-related diseases such as lung cancer, the risk that cigarette smoking and SHS exposure imparts on cardiovascular health is relatively transient, as is evidenced by the rapid decline in risk of a recurrent heart attack after quitting smoking.[3] Prior ecologic studies have shown smoke-free laws, in a short timeframe, to be associated with reductions in hospital admissions for cardiovascular disease (CVD), especially acute myocardial infarction.[2,4–6] As high blood pressure (BP) significantly increases the risk of coronary heart disease, including

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Accompanying Data S1, S2, Tables S1 through S8 and Figure S1 are available at https://www.ahajournals.org/doi/suppl/10.1161/JAHA.118.009829

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Clinical Perspective

What Is New?

- Smoke-free policies are associated with reduced rates of hospitalization for cardiovascular disease, but prior studies have not examined associations with blood pressure changes.
- We linked longitudinal data from the CARDIA (Coronary Artery Risk Development in Young Adults) Study to state, county, and local smoke-free policies in restaurants, bars, and workplaces to examine associations with BP.

What Are the Clinical Implications?

- Smoke-free policies were associated with small but significant between-person differences and within-person reductions in systolic blood pressure.
- While the magnitude of associations was small on an individual level, systolic blood pressure reduction is a potential mechanism through which smoke-free policies may reduce cardiovascular risk at the population level.

myocardial infarction,[7] and SHS exposure has been linked to hypertension across a variety of settings,[8–12] BP changes are one potential mechanism through which smoke-free policies may reduce the risk of adverse cardiovascular outcomes in nonsmokers. Although the association of smoke-free policies on BP would be expected to be small at the level of the individual, even small reductions in BP may meaningfully reduce cardiovascular risk at the population level.[13] However, little is known regarding whether smoke-free policies are associated with reductions in BP in nonsmokers. As such, our objective was to evaluate associations of smoke-free policies in restaurants, bars, and workplaces with changes in BP and hypertension among a large, geographically diverse cohort of nonsmoking adults.

Methods

Study Population

The data, analytic methods, and study materials will not be made available to other researchers for the purpose of reproducing the results or replicating the procedure. Researchers interested in the data, methods, or analysis can contact the corresponding author for more information. The CARDIA (Coronary Artery Risk Development in Young Adults) study is a longitudinal, multicenter cohort study that enrolled 5115 black and white adults aged 18 to 30 years old in 1985 to 1986 from 4 US cities: Birmingham, Alabama; Chicago, Illinois, Minneapolis, Minnesota; and Oakland, California.[14] Follow-up examinations were conducted after 2, 5, 7, 10, 15, 20, 25, and 30 years (retention rates of 91%, 86%, 81%, 79%, 74%, 72%, 72%, and 71% among the surviving cohort). The Institutional Review Board at each participating institution approved the study and all participants provided written informed consent. Participant home addresses were geocoded in years 0, 7, 10, 15, 20, and 25. We included years 10 to 25 (1995–2011) in this analysis in order to align with the timing of smoke-free policies. We excluded participants who did not have at least 2 BP measurements during follow-up (493 participants, 9.5%). Also, in order to focus on the potential for smoke-free policies to influence BP through reductions in SHS, we restricted to participants who reported *not* actively smoking (defined as smoking at least 5 cigarettes per week for the past 3 months) at any of the year 10, 15, 20, and 25 examinations (N=2606 participants).

Smoke-Free Policies

Smoke-free laws were extracted from the American Non-Smokers' Rights Foundation's Local Ordinance Database,[15] which compiled dates of all state, county, and city-level laws that prohibited smoking in restaurants, bars, or nonhospitality workplaces. Smoke-free policies from the American Non-Smokers' Rights Foundation database were linked to CARDIA participants at each examination based on the census tract they lived in. Participant addresses were converted to state-county-census tract Federal Information Processing Standard codes. State and county Federal Information Processing Standard codes were used to determine whether each census tract was located in a state or county covered by a smoke-free policy in restaurants, bars, and/or workplaces. Then, census tracts were converted to census place names (approximately equivalent to city boundaries) using the Missouri Census Data Center's MABLE/Geocorr Geographic Correspondence Engine[16] in order to determine whether each tract was located in a municipality with local smoke-free policies. Policies were linked back to participants at each examination based on their census tract and examination date. Participants were considered exposed if they had a smoke-free policy in a given venue (restaurant, bar, workplace) at either the state, county, or local level. Policies were treated as time-varying variables in all models, and smoke-free policy exposure was successfully linked for all participants at each examination.

Outcomes

Systolic and diastolic BP (SBP, DBP) were measured at each examination. Trained technicians used a random-zero mercury sphygmomanometer (years 10–15) or an oscillometer (years 20–25) to measure brachial artery BP. Resting SBP and DBP were measured 3 times at 1-minute intervals after the

participant had sat in a quiet room for 5 minutes, and the mean of the second and third measurements were calculated. Details on BP measurement and quality control have been published previously.[17] In order to account for changes in observed BP because of treatment with BP medications, we added 10 mm Hg to the observed SBP and 5 mm Hg to the observed DBP in patients reporting BP medication use.[18] In a sensitivity analysis, we used the original BP values and adjusted for whether or not participants reported BP medication use. Hypertension was defined at each examination as SBP≥130 mm Hg, DBP≥80 mm Hg, or self-reported BP medication use.

Covariates

Participant age, sex, and race were recorded at baseline. All other covariates were time-varying and were assessed at each examination. Educational attainment (in years), current marital status (married/cohabitating versus not), and alcohol consumption (heavy versus not heavy) were ascertained via standardized questionnaires. Alcohol use was dichotomized as heavy (>7 alcoholic beverages per week for women and >14 for men) versus not heavy based on the recommended consumption in the American College of Cardiology/American Heart Association's 2017 Hypertension Guidelines.[19] Household income was collected as a 9-level ordinal variable, converted to a continuous variable using the average dollar value for each category, and adjusted for inflation using the Consumer Price Index.[20] Depressive symptoms were assessed using the 20-item Center for Epidemiologic Studies-Depression questionnaire.[21] Participants were considered to have depressive symptoms if their score was greater than or equal to the validated cut point for clinical depression of 16 (on a scale of 0–60).[22]

Physical activity was assessed at each examination using a validated questionnaire assessing frequency of participation in 13 categories of exercise during the past 12 months.[23] A total physical activity score was calculated by multiplying frequency of participation by intensity of the activity and summing across activities to create a continuous exercise unit score. Participant height and weight were measured in the clinic at each examination, and body mass index was calculated as weight in kilograms divided by height in meters squared. Diabetes mellitus was defined using the following criteria: fasting glucose ≥126 mg/dL, 2-hour glucose tolerance test ≥200 mg/dL, hemoglobin A1c ≥6.5%, or self-reported use of diabetes mellitus medication.

Dietary quality was assessed at the year 7 and year 20 examinations using the CARDIA dietary history,[24] which ascertained usual dietary practices and food frequency over the past month. A Dietary Approaches to Stop Hypertension (DASH) diet score was calculated based on the 8 components of DASH (increased consumption of fruits, vegetables, whole grains, nuts and legumes, and low-fat dairy products; and reduced consumption of sodium, sugar-sweetened beverages, and red and processed meats).[25] For each component, participants were classified into sex-specific quintiles according to their intake. Quintiles were assigned scores from 1 (worst) to 5 (best) for each component, and then summed to create a summary DASH diet score ranging from 8 to 40, with higher scores reflecting better diet. The year 7 value was used for examinations 10 and 15, and the year 20 value was used for years 20 and 25. Fast-food habits were assessed at each examination using the question: "How often do you eat breakfast, lunch, or dinner at places such as McDonald's, Burger King, Wendy's, Arby's, Pizza Hut, or Kentucky Fried Chicken?"

Participants who worked outside the home in an indoor setting were asked to report whether their workplace had a policy banning smoking (either partially or completely). We compared those with a self-reported total workplace ban to all others in order to account for workplaces that had voluntarily banned smoking without laws mandating them to do so. At each examination, participants self-reported the average number of hours per week that they were exposed to tobacco smoke because of smoking by others in their home, in small spaces other than their home (eg, office, car) or large indoor spaces other than their home (eg, restaurant, hotel lobby). State cigarette taxes from *The Tax Burden on Tobacco*[26] were adjusted for inflation using the Consumer Price Index[20] and linked to participants at each examination. Metropolitan statistical area–level percent of the population below the US Census Bureau–defined poverty threshold was calculated at each examination to account for socioeconomic differences that might be associated with smoke-free policy implementation. Finally, state of residence was included as a covariate to account for additional unmeasured area-level differences in BP.

Statistical Analysis

We conducted multiple imputation using chained equations[27] to impute missing covariate values (percent missing ranged from 0% to 8.8% across variables) (Data S1). To examine associations of smoke-free policies with BP and hypertension, we used 2 analytic strategies: mixed-effects and fixed-effects models. We used mixed-effects models to examine between-person differences in BP and hypertension trajectories associated with smoke-free policies and to calculate average between-person differences at each examination. Fixed-effects modeling was used to examine associations of smoke-free policies with within-person changes in BP. Fixed-effects models include only within-person variation when estimating associations and treat each individual as his/her

own control. This process inherently controls for all time-invariant characteristics, both measured and unmeasured, by conditioning these characteristics out of the estimation process.[28] This is advantageous as fixed-effects models provide improved control for residual confounding by unmeasured variables that are stable over time. However, fixed-effects models may lead to wide confidence intervals (CIs) because between-person differences are discarded. In both sets of models, we calculated robust standard errors using Stata's vce(robust) option (Stata version 15.1; StataCorp, College Station, TX).

We first used mixed-effects models (linear regression for SBP and DBP, logistic regression for hypertension) with subject random intercepts in order to account for the correlation of repeated measures within subjects. We ran separate models for each type of policy (restaurant, bar, workplace) because the policies were highly correlated (r=0.70–0.87). Models included time-varying smoke-free policy exposure status, time since baseline in 5-year intervals (treated as continuous for SBP models because of an approximately linear relationship with time, and as dummy variables for DBP and hypertension models because of substantial departures from linearity; Figure S1), and a cross-product interaction between policy status and time. Models were adjusted for baseline age, sex, race, and the following time-varying covariates: education, marital status, income, body mass index, physical activity, alcohol consumption, dietary quality, fast-food consumption, diabetes mellitus, depressive symptoms, state of residence, state cigarette tax, metropolitan statistical area–level poverty, and self-reported workplace smoking prohibition (workplace policy models only). All continuous variables were centered at the mean. We presented the mixed-effects model findings as adjusted predicted average BP values and predicted probability of hypertension at each examination among participants who were and were not living in areas with smoke-free policies at that examination. Differences in average BP values and difference in predicted probability of hypertension between participants living in areas with and without smoke-free policies at each examination were calculated, along with 95% CIs.

We then used fixed-effects models to estimate associations of smoke-free policies with within-person changes in BP (linear regression) and within-person changes in the odds of hypertension (logistic regression). As time-invariant characteristics are conditioned out of the model, we adjusted only for the time-varying covariates listed above in order to control for confounding by changes in these characteristics over time. As above, time was modeled as a continuous variable for SBP and as dummy variables for DBP and hypertension. Additionally, in a supplemental analysis, we examined the potential mediating role of self-reported SHS exposure. We used fixed-effects models to examine associations of (1) smoke-free policies with change in self-reported SHS exposure; (2) change in self-reported SHS exposure with change in SBP and DBP; and (3) smoke-free policies with change in SBP and DBP after adjusting for self-reported SHS exposure.

As a sensitivity analysis, we used methods described by VanderWeele and Ding[29] to calculate the e-value, or the amount of residual confounding because of an omitted covariate that would be needed to shift the magnitude of association between smoke-free policies and BP to the null. Details of this sensitivity analysis are presented in Data S2. Author SLM had full access to all the data in the study and takes responsibility for its integrity and the data analysis.

Results

A total of 2606 CARDIA participants were included in the analysis. Among the whole sample, SBP increased from 109.7 to 121.0 mm Hg, and DBP increased from 72.5 to 75.2 mm Hg (Table 1). The percent of participants with hypertension increased from 22.6% to 44.4%.

At baseline (year 10 examination), 1.5% of participants lived in areas with smoke-free policies in restaurants, 0.8% in bars, and 7.3% in workplaces (Figure 1). Smoke-free policy exposure increased substantially over follow-up, and by year 25, 88% of participants lived in areas with smoke-free policies in restaurants, 75% in bars, and 73% in workplaces (Figure 1). Participants had dispersed geographically from the 4 original study centers by the year 10 examination and lived in 46 different states. Table S1 presents the proportion of participants covered by smoke-free policies passed at each geographic level (state, county, and local).

At each examination, participants living in areas with restaurant policies had lower SBP on average than those in areas without restaurant policies, and the difference increased over time (Figure 2A). By the year 25 examination, participants in areas with restaurant policies had SBP values on average 1.14 mm Hg lower than those in areas without restaurant policies (95% CI, −2.15, −0.12). Patterns were similar for bar policies, with a slightly larger difference by year 25 (difference of −1.52 mm Hg, 95% CI, −2.48, −0.57). For workplace policies, participants living in areas with policies had slightly higher SBP in years 10 and 15, which reversed in years 20 and 25 (final difference in year 25 of −1.41 mm Hg, 95% CI, −2.32, −0.50). Table S2 presents the regression coefficients from the mixed-effects models that were used to calculate the predicted values. For all 3 types of policies, the slope of the SBP curve increased at a slower rate among those exposed to smoke-free policies (5-year differences of

Table 1. Characteristics of Study Participants at Baseline and Follow-Up Examinations

Characteristics	Year 10	Year 15	Year 20	Year 25
N	2606	2427	2332	2247
Blood pressure variables*				
SBP, mean (SD), mm Hg	109.7 (12.5)	113.1 (15.0)	117.3 (15.9)	121.0 (17.1)
DBP, mean (SD), mm Hg	72.5 (10.0)	74.6 (11.6)	73.2 (11.9)	75.2 (11.8)
Hypertension, N (%)†	588 (22.6)	805 (33.2)	806 (34.6)	997 (44.4)
Antihypertensive medication use, N (%)	80 (3.1)	191 (7.9)	359 (15.4)	546 (24.3)
Sociodemographic characteristics				
Age, y, mean (SD)	35.1 (3.6)	40.2 (3.6)	45.2 (3.6)	50.3 (3.6)
Sex, N (%)				
Female	1483 (56.9)	1373 (56.6)	1335 (57.3)	1287 (57.3)
Male	1123 (43.1)	1054 (43.4)	997 (42.7)	960 (42.7)
Race, N (%)				
White	1476 (56.6)	1407 (58.0)	1355 (58.1)	1311 (58.3)
Black	1130 (43.4)	1020 (42.0)	977 (41.9)	936 (41.7)
Educational attainment, y, mean (SD)	15.2 (2.5)	15.4 (2.4)	15.5 (2.5)	15.6 (2.6)
Inflation-adjusted household income, mean (SD), per $10 000	5.5 (2.9)	7.6 (4.6)	7.5 (4.3)	7.0 (3.9)
Married/living as married, N (%)	1451 (55.9)	1583 (65.4)	1563 (67.2)	1484 (66.3)
Health-related covariates				
Heavy alcohol consumption, N (%)	654 (25.2)	630 (26.0)	674 (29.5)	682 (30.6)
Body mass index, mean (SD), kg/m^2	27.4 (6.3)	28.7 (6.7)	29.4 (7.3)	30.1 (7.2)
Total units of physical activity‡, mean (SD)	333.1 (269.3)	347.4 (281.6)	348.2 (277.8)	349.4 (277.8)
Dietary quality, mean (SD)	24.1 (5.2)	24.1 (5.2)	24.2 (5.1)	24.0 (5.0)
Fast-food frequency (times per wk), mean (SD)	1.7 (2.0)	1.8 (2.3)	1.7 (2.3)	1.2 (2.0)
Diabetes mellitus, N (%)	90 (3.5)	120 (5.0)	224 (9.7)	299 (13.4)
Depressive symptoms, N (%)	382 (14.9)	287 (12.0)	295 (12.9)	292 (13.1)
Policy/geographic covariates				
State cigarette tax in $, mean (SD)	0.25 (0.10)	0.29 (0.15)	0.36 (0.15)	0.33 (0.17)
Self-report of ban on smoking in their workplace§, N (%)	1451 (55.7)	1405 (57.9)	1291 (55.4)	1211 (53.9)
MSA-level percent of population below the poverty threshold, mean (SD)	11.4 (2.8)	10.9 (2.8)	11.0 (2.8)	13.1 (2.7)
Hours per week of self-reported SHS exposure‖				
At home, mean (SD)	2.0 (7.8)	1.4 (7.0)	1.3 (8.6)	0.8 (5.8)
In small spaces other than home, mean (SD)	1.8 (6.3)	1.4 (6.3)	1.3 (9.4)	0.5 (2.7)
In large spaces other than home, mean (SD)	1.8 (4.4)	1.5 (5.7)	1.5 (11.5)	0.6 (3.3)

DBP indicates diastolic blood pressure; MSA, metropolitan statistical area; SBP, systolic blood pressure; SHS, secondhand smoke.
*Blood pressure values were adjusted for participants who reported use of blood pressure medications. 10 mm Hg was added to the measured value of SBP, and 5 mm Hg to the value of DBP, for years in which participants reported blood pressure medication use.
†Hypertension defined as SBP≥130 mm Hg, DBP≥80 mm Hg, or self-reported blood pressure medication use.
‡Measured in self-reported exercise units that accounted for the frequency and intensity of each activity.
§Participants who worked indoors were asked to self-report whether their workplace had a policy banning smoking.
‖Participants were asked to report how many hours per week on average they were exposed to tobacco smoke in their home, in a small space other than their home (eg, office), or a large space other than their home (eg, restaurant) because of smoking by others.

−0.32 mm Hg for restaurant policies, −0.53 mm Hg for bar policies, and −0.82 mm Hg for workplace policies; *P* interaction: 0.3 for restaurant policies, 0.08 for bar policies, and 0.002 for workplace policies).

For DBP and hypertension, patterns were less clear. Participants living in areas with smoke-free policies had higher DBP on average in year 10, lower in years 15 and 20, and higher again in year 25 compared with those living in

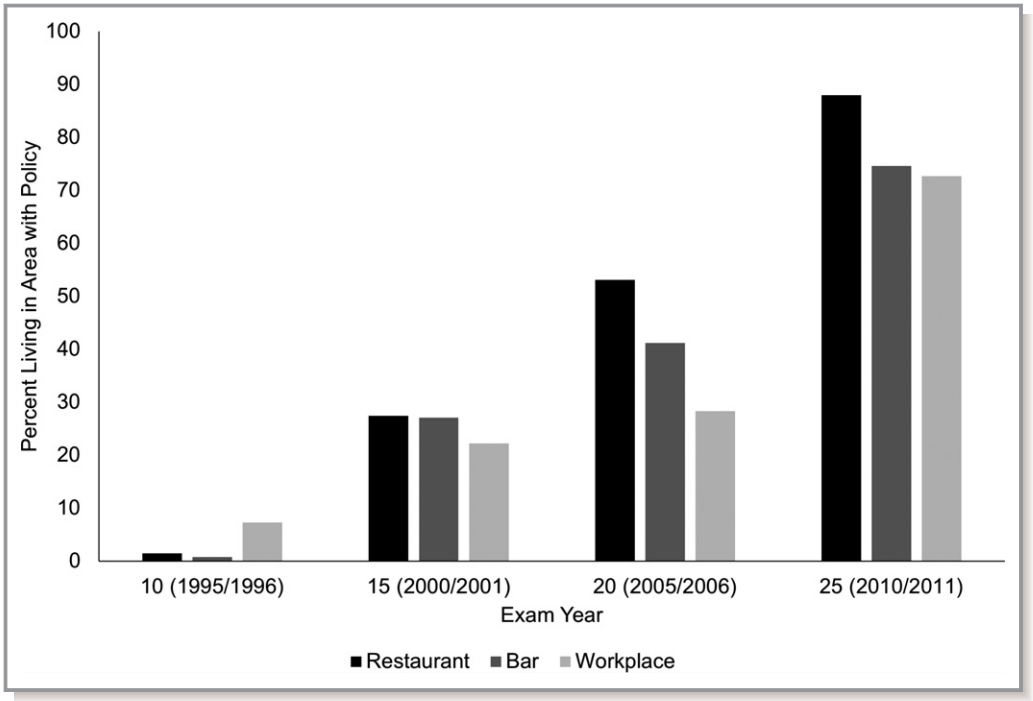


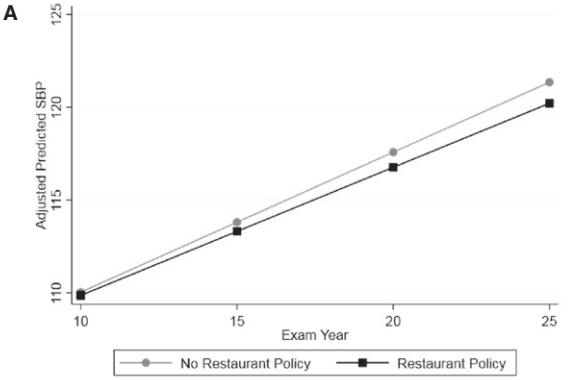
Figure 1. Prevalence of smoke-free policies in restaurants, bars, and workplaces over follow-up. Participant exposure to smoke-free policies was defined as living in a census tract in a state, county, or locality that implemented a 100% ban on smoking in restaurants, bars, or workplaces.

areas without smoke-free policies (Figure 3A through 3C, Table S3). A higher proportion of participants living in areas with smoke-free policies in year 10 had prevalent hypertension compared with those in areas without smoke-free policies, but there was no difference in the predicted probability of hypertension between those living in areas with and without smoke-free policies in later years (Figure 4A through 4C, Table S4). Results for both SBP and DBP were similar in sensitivity analyses that accounted for BP medication use by using the original recorded BP values and adjusting for BP medication use in the model (Tables S5 through S6).

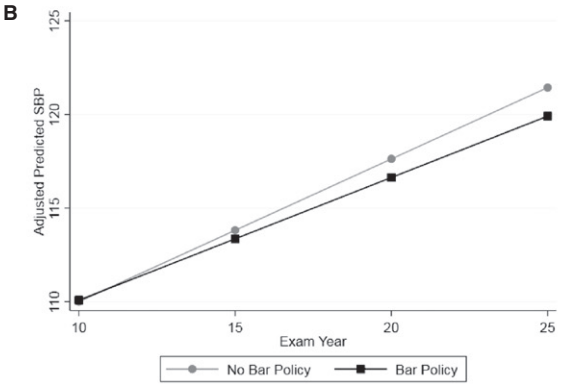
Table 2 presents the results of the fixed-effects models, which estimated the average change in SBP and DBP, and average change in the odds of hypertension, associated with becoming exposed to a smoke-free policy in restaurants, bars, or workplaces. Living in an area with a restaurant smoke-free policy was associated with a mean SBP change of −0.85 mm Hg (95% CI, −1.61, −0.09) after controlling for time and time-varying sociodemographic, clinical, and policy/geographic covariates (Table 2). Associations were slightly stronger for bar policies (−1.08, 95% CI, −1.82, −0.34) and weaker for workplace policies with a CI crossing the null (−0.60, 95% CI, −1.33, 0.14). For DBP, patterns differed. Restaurant policies were associated with a reduction in DBP of −0.58 (95% CI, −1.15, −0.01), while point estimates were in the opposite direction and CIs overlapped the null for bar and workplace policies (estimated mean change of 0.26, 95% CI, −0.32, 0.83 for bar policies and 0.22, 95% CI, −0.37, 0.80 for workplace policies). Results were similar in models that used the original BP values and adjusted for BP medication use (Table S7). For hypertension, point estimates were in the hypothesized direction but CIs were wide (restaurant policies: odds ratio: 0.93, 95% CI, 0.71, 1.23; bar policies: odds ratio: 0.82, 95% CI, 0.61, 1.10; workplace policies: odds ratio: 0.91, 95% CI, 0.68, 1.20; Table 2).

In a supplemental analysis, we found smoke-free policies in restaurants and bars to be associated with within-person reductions in average hours/wk of SHS exposure in large indoor spaces (Table S8). Smoke-free policies were not associated with changes in SHS exposure in the home. Changes in self-reported SHS exposure were not associated with changes in SBP or DBP, and associations between smoke-free policies and blood pressure were unchanged upon adjustment for self-reported SHS exposure (Table S8). In a sensitivity analysis to assess the potential influence of unmeasured confounding, we estimated that an unmeasured confounder would need to have an association equivalent to a relative risk of ≈1.3 to 1.4 with both the exposure and outcome in order to shift the magnitude of the predicted difference in SBP between participants living in areas with and without smoke-free policies to the null. As this was estimated from fully adjusted models, this strength of association would

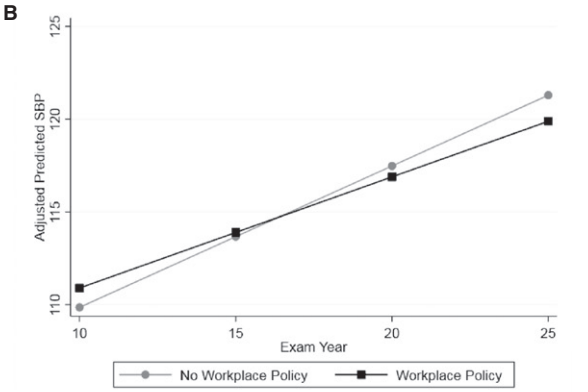
need to persist after adjustment for all of the measured covariates including individual-level sociodemographics, health behaviors, other health risk factors, area-level covariates, state of residence, and the time trend (Data S2).



	Year 10	Year 15	Year 20	Year 25
Between-Group Difference in SBP (95% CI)	-0.18 (-1.45, 1.10)	-0.50 (-1.36, 0.37)	-0.82 (-1.56, -0.08)	-1.14 (-2.15, -0.12)



	Year 10	Year 15	Year 20	Year 25
Between-Group Difference in SBP (95% CI)	0.08 (-1.25, 1.41)	-0.46 (-1.34, 0.43)	-0.99 (-1.70, -0.28)	-1.52 (-2.48, -0.57)



	Year 10	Year 15	Year 20	Year 25
Between-Group Difference in SBP (95% CI)	1.04 (-0.11, 2.19)	0.23 (-0.56, 1.02)	-0.59 (-1.27, 0.09)	-1.41 (-2.32, -0.50)

Discussion

Smoke-free policies have been associated with reduced risk of hospitalization for CVD,[2,4–6] but prior studies have not examined associations of these policies with BP changes. Among a geographically diverse cohort of black and white nonsmoking adults followed for 15 years, we found that participants living in areas with smoke-free policies in restaurants, bars, and workplaces had lower SBP at the end of follow-up compared with participants living in areas without smoke-free policies. In addition, smoke-free policies in bars and restaurants were associated with within-person reductions in SBP. For DBP and hypertension, patterns were inconsistent and CIs were generally wide.

Smoke-free policies have been shown to reduce environmental tobacco smoke exposure among hospitality workers[30–33] and the general population.[34] Smoke-free policies have also been associated with reductions in hospital admissions for CVD, particularly for acute myocardial infarction.[2,4–6] We previously found smoke-free policies to be associated with reduced risk of incident CVD in CARDIA.[35] Although not all studies have found associations between smoke-free policies and CVD,[36,37] systematic reviews have concluded the evidence is strong.[2] While no prior studies have examined associations of smoke-free policies with changes to BP, our results are consistent in the context of prior findings for SHS and CVD. High BP significantly increases the risk of coronary heart disease[7] and SHS exposure has been linked to hypertension,[8–12] likely because of adverse effects of harmful components of SHS (nicotine, carbon monoxide, methane, formaldehyde, and others) on vasoconstriction or vasodilation,[38] as well as vascular dysfunction.[39] The results of our study suggest BP reduction

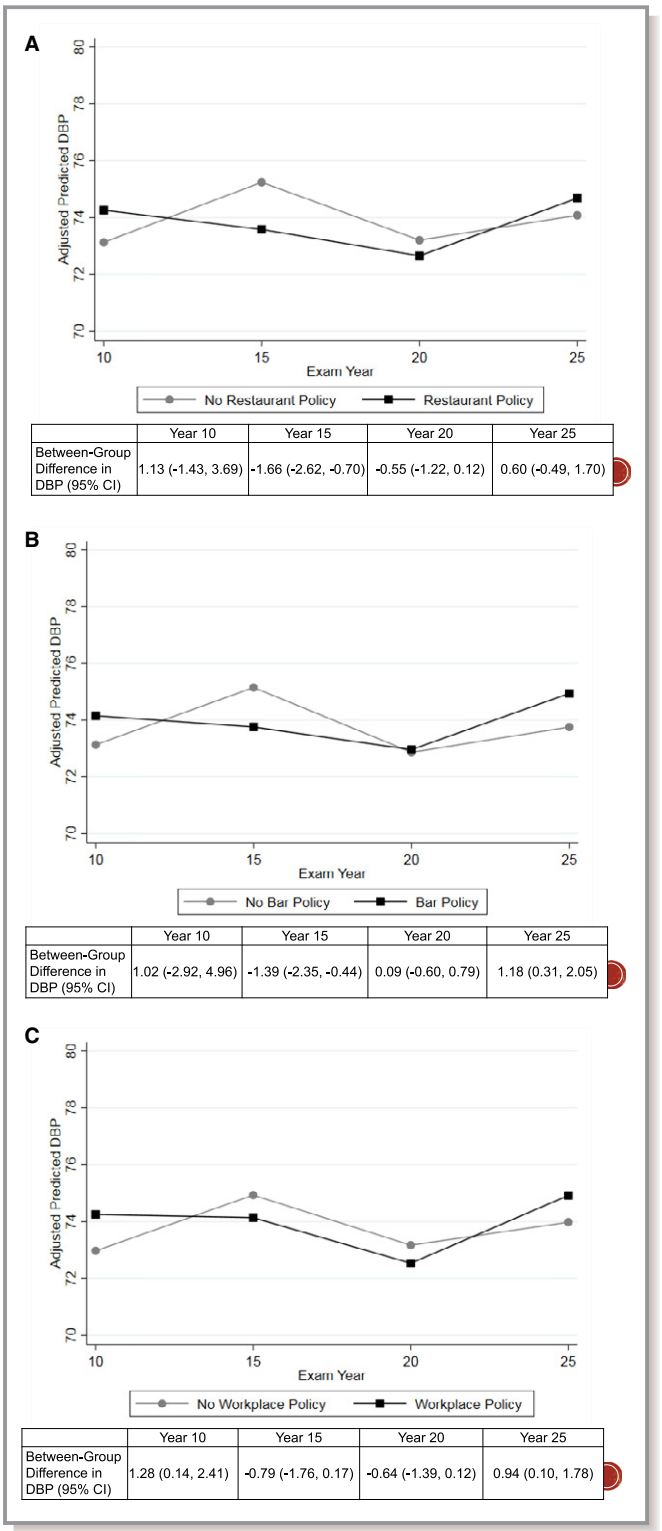
Figure 2. Longitudinal changes in SBP over 15 y by smoke-free policy exposure status in (**A**) restaurants, (**B**) bars, and (**C**) workplaces. Results are predicted mean SBP values at each examination among participants living in areas with and without smoke-free policies (time-varying), adjusted for participant sex, race, baseline age, education, marital status, income, alcohol consumption, diet quality, fast-food consumption, depressive symptoms, body mass index, physical activity, diabetes mellitus, state cigarette taxes, state of residence, metropolitan statistical area–level poverty, self-reported workplace smoking prohibition (workplace policy models only), and interactions between mean-centered baseline age×time and race×time. Estimated using linear mixed-effects models with subject random intercepts using the "mixed" package in Stata. The "margins" package was used to calculate covariate-adjusted predicted values at each examination for both policy exposure groups, and the between-group difference with confidence intervals. CI indicates confidence interval; SBP, systolic blood pressure.

as a potential mechanism through which smoke-free policies may reduce rates of CVD at the population level.

We found smoke-free policies to be consistently associated with reduced SBP, but not DBP or hypertension. SBP elevation in isolation is often a result of age-related atherosclerosis, and occurs more frequently than isolated diastolic elevation after age 50.[40] Recent work in CARDIA indicated that SBP was more strongly associated with CVD risk than DBP among participants in middle age,[41] which corresponds with the time period of our analysis. Increasing BP values below the threshold for hypertension have been found to be associated with worse CVD outcomes.[7,42] In addition, even small reductions in SBP may result in meaningful reductions in CVD risk across a population—for example, a study that estimated CVD incidence rates for a hypothetical population-wide intervention estimated that a 1 mm Hg reduction was associated with ≈10 fewer coronary heart disease events per 100 000 people per year.[13] Thus, changes to SBP of the magnitude seen in our study have important implications for cardiovascular public health. For hypertension, the use of a cruder dichotomous outcome measure reflecting the same underlying process as SBP may have limited statistical power to detect small associations.

Our finding that self-reported SHS was not associated with changes in BP and did not appear to mediate associations of smoke-free policies with SBP may reflect challenges in measuring actual SHS exposure via a crude self-reported measure (asking participants to report the number of hours per week on average they were exposed). SHS exposure intensity may vary significantly from place to place and at different times of the day. In addition, the measures required participants not only to recollect their exposure but also to synthesize this information across a variety of possible venues. Finally, these measures may be overly broad to reflect exposures in the venues covered by smoke-free policies (for example, SHS exposure in small indoor spaces may include exposure in cars as well as workplaces).

Strengths of this study include use of data from a large, geographically diverse cohort with 15 years of follow-up,

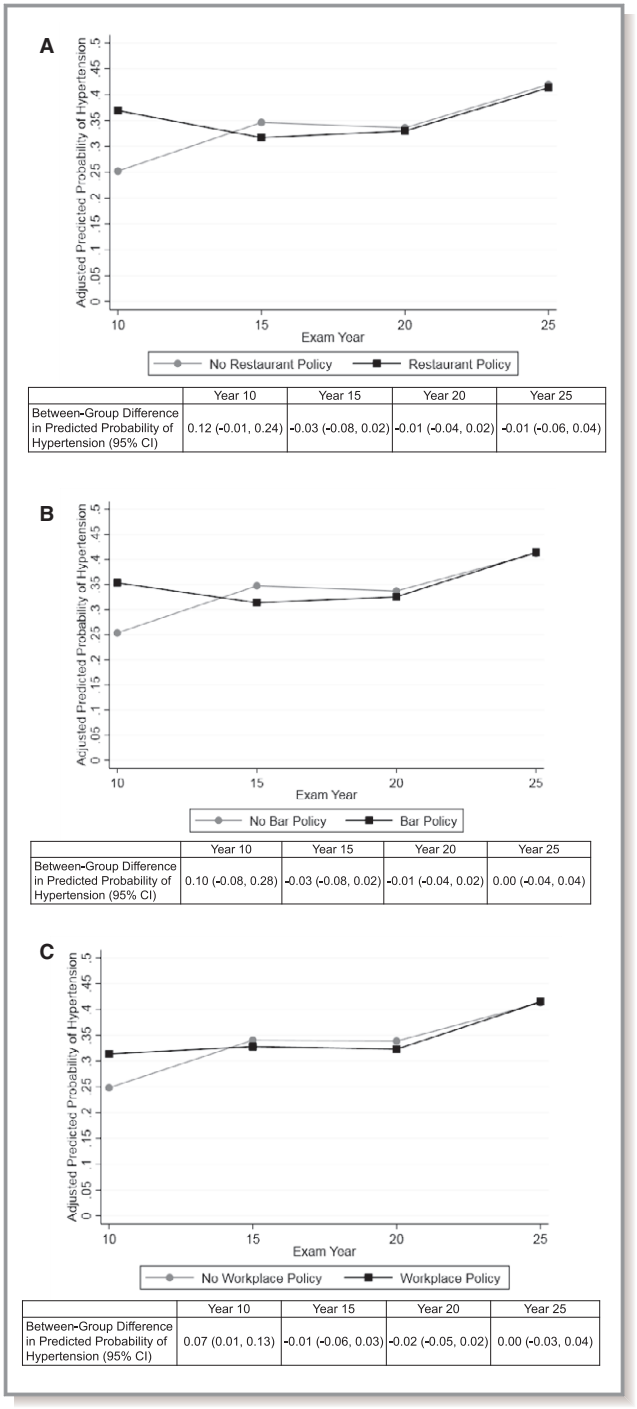


	Year 10	Year 15	Year 20	Year 25
Between-Group Difference in DBP (95% CI)	1.13 (-1.43, 3.69)	-1.66 (-2.62, -0.70)	-0.55 (-1.22, 0.12)	0.60 (-0.49, 1.70)

	Year 10	Year 15	Year 20	Year 25
Between-Group Difference in DBP (95% CI)	1.02 (-2.92, 4.96)	-1.39 (-2.35, -0.44)	0.09 (-0.60, 0.79)	1.18 (0.31, 2.05)

	Year 10	Year 15	Year 20	Year 25
Between-Group Difference in DBP (95% CI)	1.28 (0.14, 2.41)	-0.79 (-1.76, 0.17)	-0.64 (-1.39, 0.12)	0.94 (0.10, 1.78)

Figure 3. Longitudinal changes in DBP over 20 y by smoke-free policy exposure status in (**A**) restaurants, (**B**) bars, and (**C**) workplaces. Results are predicted mean DBP values at each examination among participants living in areas with and without smoke-free policies (time-varying), adjusted for participant sex, race, baseline age, education, marital status, income, alcohol consumption, diet quality, fast-food consumption, depressive symptoms, body mass index, physical activity, diabetes mellitus, state cigarette taxes, state of residence, metropolitan statistical area–level poverty, self-reported workplace smoking prohibition (workplace policy models only), and interactions between race×examination year and sex×examination year. Estimated using linear mixed-effects models with subject random intercepts using the "mixed" package in Stata. The "margins" package was used to calculate covariate-adjusted predicted values at each examination for both policy exposure groups, and the between-group difference with confidence intervals. Because of nonlinearity in patterns of DBP over time, examination year was treated as categorical. CI indicates confidence interval; DBP, diastolic blood pressure.



	Year 10	Year 15	Year 20	Year 25
Between-Group Difference in Predicted Probability of Hypertension (95% CI)	0.12 (-0.01, 0.24)	-0.03 (-0.08, 0.02)	-0.01 (-0.04, 0.02)	-0.01 (-0.06, 0.04)

	Year 10	Year 15	Year 20	Year 25
Between-Group Difference in Predicted Probability of Hypertension (95% CI)	0.10 (-0.08, 0.28)	-0.03 (-0.08, 0.02)	-0.01 (-0.04, 0.02)	0.00 (-0.04, 0.04)

	Year 10	Year 15	Year 20	Year 25
Between-Group Difference in Predicted Probability of Hypertension (95% CI)	0.07 (0.01, 0.13)	-0.01 (-0.06, 0.03)	-0.02 (-0.05, 0.02)	0.00 (-0.03, 0.04)

Figure 4. Longitudinal changes in hypertension prevalence over 15 y by smoke-free policy exposure status in (**A**) restaurants, (**B**) bars, and (**C**) workplaces. Results are predicted probabilities of hypertension at each examination among participants living in areas with and without smoke-free policies (time-varying), adjusted for participant sex, race, baseline age, education, marital status, income, alcohol consumption, diet quality, fast food consumption, depressive symptoms, body mass index, physical activity, diabetes mellitus, state cigarette taxes, state of residence, metropolitan statistical area-level poverty, self-reported workplace smoking prohibition (workplace policy models only), and interactions between sex and examination year, and race and examination year. Estimated using logistic mixed-effects models with subject random intercepts. The "margins" package was used to calculate covariate-adjusted predicted probabilities at each examination for both policy exposure groups, and the between-group difference with confidence intervals. Because of nonlinearity in patterns of hypertension over time, examination year was treated as categorical. CI indicates confidence interval.

and use of fixed-effects models to tightly control for both measured and unmeasured time-invariant characteristics of participants. However, this study was also subject to several limitations. First, our results may be subject to residual confounding. Although fixed-effects models provide tight control for unmeasured confounding by time-invariant characteristics, they do not control for unmeasured confounders that vary over time. For example, we were unable to control for anti-smoking marketing campaigns because no comprehensive database of these campaigns is available. Our sensitivity analysis using the e-value indicated an unmeasured confounder with an association equivalent to a risk ratio of 1.3 to 1.4 with both the exposure and outcome might explain results. However, this level of association would need to persist after adjustment for all of the measured covariates that were included in our analysis, which included the SBP time trend, state of residence, metropolitan-level poverty, cigarette taxes, and individual-level sociodemographics, health behaviors, and hypertension risk factors.

In addition, although CARDIA had high retention rates, it is possible that loss to follow-up could bias results if loss to follow-up is patterned by both smoke-free policy exposure status and BP. Because BP values were measured by technicians, it is possible that measured values were elevated relative to what would be seen in 24-hour ambulatory BP monitoring because of "white coat hypertension." However, prior work in CARDIA that compared in-clinic to ambulatory BP measurements among a subset of participants found the prevalence of white coat hypertension to be small (3% for white and 4% for black participants).[43] Also, we restricted to participants who reported that they were never regular smokers during the study period (defined as smoking at least 5 cigarettes per week). However, we did not ask participants whether they smoked at less frequent intervals, so it is possible that some participants were occasional smokers rather than true nonsmokers. Finally, smoke-free policies were linked to participants based on their residence. However, if participants worked or spent time in bars and restaurants in different cities from their home, the policies exposures attributed to them in this study may not reflect their actual exposure.

Table 2. Mean Within-Person Changes in SBP and DBP Associated With Exposure to Smoke-Free Policies in Restaurants, Bars, and Workplaces*,†,‡,§

	Restaurant Policy	Bar Policy	Workplace Policy
Adjusted mean change (95% CI), mm Hg			
SBP	−0.85 (−1.61, −0.09)	−1.08 (−1.82, −0.34)	−0.60 (−1.33, 0.14)
DBP	−0.58 (−1.15, −0.01)	0.26 (−0.32, 0.83)	0.22 (−0.37, 0.80)
Odds ratio (95% CI)			
Hypertension	0.93 (0.71, 1.23)	0.82 (0.61, 1.10)	0.91 (0.68, 1.20)

CI indicates confidence interval; DBP, diastolic blood pressure; SBP, systolic blood pressure.

*Participant exposure to smoke-free policies was defined as living in a census tract in a state, county, or locality that implemented a 100% ban on smoking in restaurants, bars, or workplaces.

†Results for SBP and DBP are from linear fixed-effects models and can be interpreted as the mean difference in blood pressure in years where participants lived in areas with smoke-free policies compared with years where they did not, controlling for time and the covariates listed below. Results for hypertension are from logistic fixed-effects models and can be interpreted as the ratio of the odds of hypertension in years where participants lived in areas with smoke-free policies compared with years where they did not, adjusted for covariates.

‡Models adjusted for time since baseline (5-year increments), time-varying covariates (education, marital status, income, body mass index, physical activity, alcohol consumption, diet quality, fast-food consumption, diabetes mellitus, depressive symptoms, state cigarette tax, state of residence, metropolitan statistical area–level poverty, and whether participants reported that their workplace had a prohibition on smoking [workplace policy models only]) and interaction between time-invariant characteristics and time that were significant at the $P<0.05$ level (race×time and baseline age×time).

§As longitudinal patterns of DBP and hypertension were not well approximated by a linear relationship with follow-up time, models for DBP and hypertension included examination year as a categorical variable. For DBP, models included interactions between race×examination year and sex×examination year (P interaction<0.05).

Conclusion

Smoke-free policies in bars, restaurants, and workplaces were associated with significantly lower SBP at the end of follow-up, and with small within-person reductions in SBP, among a cohort of black and white US nonsmokers. Results underscore the potential benefit at a population level of smoke-free policies as a prevention measure for high SBP.

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Disclosures

None.

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SUPPLEMENTAL MATERIAL

Data S1.

Supplemental Methods 1. Description of the Multiple Imputation Model.

The multiple imputation model used to create the 10 imputed datasets used for analysis was fitted using the MI Impute Chained package in Stata version 15.1 (StataCorp, College Station, Texas). This package uses chained equations, a method with fully conditional specification of prediction equations that allows for arbitrary missingness-value patterns that can accommodate multiple types of variables.[1]

The analytic dataset was converted from a long format (up to 4 rows per participant reflecting the 4 included exams, years 10, 15, 20, and 25) to a wide format. This was done following guidance for the imputation of longitudinal data[2,3] in order to simultaneously include data from all exams, so that participants' responses for a given variable from other exams as well as other variables could be used to impute missing. Following imputation, the dataset was converted back to a long (person-period) format and imputed variables from exams that participants did not actually attend were dropped.

All analytic variables were included in the model as predictors (systolic blood pressure, diastolic blood pressure, blood pressure medication use, baseline age, study center, state of residence, sex, race, education, income, marital status, alcohol use, body mass index, physical activity, fast food consumption, dietary quality, diabetes status, depressive symptoms, smoke-free policies, state cigarette taxes, secondhand smoke exposures in the home, small indoor spaces, and large indoor spaces. Logit models were used to predict dichotomous variables with missing data (marital status, alcohol use, depressive symptoms, diabetes) and predictive mean matching was used to impute continuous variables with missing data (education, income, body mass index, physical activity, dietary quality, fast food consumption, state cigarette tax, secondhand smoke exposure).

Data S2.

Supplemental Methods 2. Sensitivity to Omitted Variable Residual Confounding.

We conducted a sensitivity analysis to assess the potential influence of an unmeasured confounder on the associations between smoke-free policies and systolic blood pressure (SBP) observed in this study. We focus this section on SBP since associations for diastolic blood pressure and prevalent hypertension were inconsistent and confidence intervals were generally wide. We calculated the e-value, as described by VanderWeele and Ding[4], which quantifies the extent of residual confounding from an omitted covariate that would be required to shift associations between smoke-free policies and SBP to the null. While the e-value is often used for risk ratios, extensions to other types of measures are available. We calculated Cohen's *d* effect sizes using the predicted mean differences between participants living in areas with and without smoke-free policies in restaurants, bars, and workplaces in year 25, as well as the pooled standard deviation (SD) of SBP across the exposed and unexposed groups. We then used the formula published by VanderWeele and Ding[4] to calculate the e-value based on standardized effect size.

Steps:

1) We calculated pooled standard errors for the exposed and unexposed groups:

$$SD_{pooled} = \sqrt{\frac{(SD^2_{exposed} + SD^2_{unexposed}}{2}})$$

2) We calculated standardized effect size measure (Cohen's *d*) using the mean difference between exposure groups and the pooled standard deviation, calculated as:

$$\mathrm{d} = \frac{(Mean_{exposed} - Mean_{unexposed})}{SD_{pooled}}$$

3) We converted the effect size into a risk ratio (RR) using the equation provided in Vanderwheele and Ding: $RR = e^{(0.91*d)}$

4) As the RR is <1, we used the formula: $RR^* = \frac{1}{RR}$

5) We then used RR* to calculate the e-value using the formula:

$$e - value = RR^* + \sqrt{RR^* * (RR^* - 1)}$$

This was done for each type of smoke-free policy (restaurant, bar, workplace). The e-values, and the values used to calculate them, are shown below

Policy Type	Adjusted mean difference in SBP between exposed and unexposed groups in year 25	SD_{pooled}	Cohen's *d*	RR	1/RR (RR*)	e-value
Restaurant	-1.14	17.32	-0.066	0.942	1.062	1.32
Bar	-1.52	17.42	-0.087	0.924	1.083	1.38
Workplace	-1.41	17.45	-0.081	0.929	1.076	1.36

The results suggest that a potential unmeasured confounder would be required to have an association equivalent to a relative risk between 1.3 and 1.4 with both the exposure and the outcome. Because we adjusted for the time trend in SBP, state of residence, state cigarette taxes, and individual-level sociodemographics, health behaviors, and other health risk factors, this degree of confounding would need to persist after these adjustments.

Table S1. Participant Residence in Areas with Smoke-Free Policies in Restaurants, Bars, and Workplaces, by Geographic Level, the CARDIA Study (N=2,606 Non-Smokers, 1995-2011).*

	Year 10 N (%)	Year 15 N (%)	Year 20 N (%)	Year 25 N (%)
Total N	2606	2427	2332	2247
Restaurant Policy				
State	6 (0.2)	664 (27.4)	723 (31.0)	1670 (74.3)
County	16 (0.6)	376 (15.5)	370 (15.9)	1098 (48.9)
Local	19 (0.7)	88 (3.6)	605 (25.9)	1023 (45.5)
Bar Policy				
State	0 (0.0)	657 (27.1)	695 (29.8)	1626 (72.4)
County	14 (0.5)	375 (15.5)	367 (15.7)	1090 (48.5)
Local	6 (0.2)	73 (3.0)	337 (14.5)	728 (32.4)
Workplace Policy				
State	0 (0.0)	0 (0.0)	79 (3.4)	1025 (45.6)
County	109 (4.2)	472 (19.5)	469 (20.1)	902 (40.1)
Local	137 (5.3)	175 (7.2)	228 (9.8)	702 (31.2)

CARDIA=Coronary Artery Risk Development in Young Adults

*Categories are not mutually exclusive.

Table S2. Mean Difference in Systolic Blood Pressure in Year 10 and Mean Differences in 5-Year Change in Systolic Blood Pressure by Smoke-Free Policy Exposure in Restaurants, Bars, and Workplaces From Mixed Effects Models, The CARDIA Study (N=2,606 Non-smokers, 1995-2011)*†

	Mean Difference in Systolic Blood Pressure (95% Confidence Interval), mm Hg		
	Restaurant Policy	**Bar Policy**	**Workplace Policy**
Mean Difference in Year 10 (Policy versus No Policy)	-0.18 (-1.45, 1.10)	0.08 (-1.25, 1.41)	1.04 (-0.11, 2.19)
Mean 5-year Change among those Living in Areas with No Policy (Time Main Effect)	3.11 (2.71, 3.50)	3.17 (2.81, 3.54)	3.18 (2.84, 3.53)
Deviation in 5-year Change Associated with Policy Exposure‡	-0.32 (-0.90, 0.26)	-0.53 (-1.12, 0.06)	-0.82 (-1.33, -0.30)
P-interaction‡	0.3	0.08	0.002

CARDIA=the Coronary Artery Risk Development in Young Adults Study

*Participant exposure to smoke-free policies was defined as living in a census tract in a state, county, or locality that implemented a 100% ban on smoking in restaurants, bars, or workplaces.
†Results are from linear mixed-effects models with participant-specific random intercepts. Models included the time-varying smoke-free policy exposure (coded “exposed” versus “unexposed” at a given exam), time since the year 5 exam (in 5-year increments), and a policy*time interaction. Models were adjusted for baseline age, sex, race, education, marital status, income, body mass index, physical activity, diabetes, alcohol consumption, diet quality, fast food consumption, depressive symptoms, state cigarette tax, state of residence, metropolitan statistical area-level poverty, participants self-report that their workplace had a prohibition on smoking (workplace policy models only), and race*time and mean-centered baseline age*time interactions.
‡Estimated from interaction of time-varying smoke-free policy exposure status and time.

Table S3. Mean Difference in Diastolic Blood Pressure at Baseline and Mean Differences Between Exams by Exposure to Smoke-Free Policies in Restaurants, Bars, and Workplaces, The CARDIA Study (N=2,606 Non-smokers, 1995-2011)*†

	Mean Difference In Diastolic Blood Pressure (95% Confidence Interval), mm Hg		
	Restaurant Policy	**Bar Policy**	**Workplace Policy**
Mean Difference in Year 10 (Policy versus No Policy)	1.13 (-1.43, 3.69)	1.02 (-2.92, 4.96)	1.28 (0.14, 2.41)
Mean Difference Between Year 15 and Year 10 Among those Living in Areas with No Policy	1.88 (1.23, 2.53)	1.78 (1.13, 2.43)	1.73 (1.08, 2.37)
Deviation in Year 15-Year 10 Difference Associated with Policy Exposure‡	-2.79 (-5.45, -0.13)	-2.41 (-6.43, 1.60)	-2.07 (-3.42, -0.72)
P-interaction‡	0.04	0.2	0.003
Mean Difference Between Year 20 and Year 10 Among those Living in Areas with No Policy	-0.29 (-1.10, 0.51)	-0.62 (-1.41, 0.16)	-0.13 (-0.88, 0.63)
Deviation in Year 20-Year 10 Difference Associated with Policy Exposure‡	-1.68 (-4.32, 0.96)	-0.93 (-4.92, 3.07)	-1.91 (-3.20, -0.62)
P-interaction‡	0.2	0.6	0.004
Mean Difference Between Year 25 and Year 10 Among those Living in Areas with No Policy	0.60 (-0.65, 1.86)	0.22 (-0.84, 1.27)	0.64 (-0.35, 1.63)
Deviation in Year 25-Year 10 Difference Associated with Policy Exposure‡	-0.53 (-3.31, 2.25)	0.16 (-3.86, 4.19)	-0.34 (-1.70, 1.03)
P-interaction‡	0.7	0.9	0.6
Global Test of Interaction Between Policy Exposure Status and Exam Year (testing that all = 0)	0.007	0.0008	0.0003

CARDIA=the Coronary Artery Risk Development in Young Adults Study

*Participant exposure to smoke-free policies was defined as living in a census tract in a state, county, or locality that implemented a 100% ban on smoking in restaurants, bars, or workplaces.
†Results are from linear mixed-effects models with participant-specific random intercepts. Models included the time-varying smoke-free policy exposure (coded “exposed” versus “unexposed” at a given exam), categorical exam year (15, 20, or 25 versus 10), and a policy*exam year interaction. Models adjusted for categorical exam year (year 10 as reference), baseline age, sex, race, education, marital status, income, body mass index, physical activity, diabetes, alcohol consumption, diet quality, fast food consumption, depressive symptoms, state cigarette tax, state of residence, metropolitan statistical area-level poverty, participants self-report that their workplace had a prohibition on smoking (workplace policy models only), and interactions between race*exam year and sex*exam year.
‡Estimated from interaction of time-varying smoke-free policy exposure status and categorical exam year.

Table S4. Odds Ratios of Hypertension at Baseline and Differences Between Exams by Exposure to Smoke-Free Policies in Restaurants, Bars, and Workplaces, The CARDIA Study (N=2,606 Non-smokers, 1995-2011)*†

	Odds Ratio for Hypertension (95% Confidence Interval)		
	Restaurant Policy	**Bar Policy**	**Workplace Policy**
Mean Difference in Year 10 (Policy versus No Policy)	3.39 (1.01, 11.41)	2.86 (0.48, 17.11)	2.04 (1.09, 3.85)
Mean Difference Between Year 15 and Year 10 Among those Living in Areas with No Policy	3.93 (2.63, 5.88)	3.87 (2.60, 5.78)	3.88 (2.62, 5.74)
Deviation in Year 15-Year 10 Difference Associated with Policy Exposure‡	0.22 (0.06, 0.79)	0.25 (0.04, 1.58)	0.43 (0.21, 0.90)
P-interaction‡	0.02	0.1	0.03
Mean Difference Between Year 20 and Year 10 Among those Living in Areas with No Policy	3.19 (2.06, 4.92)	3.15 (2.07, 4.81)	3.44 (2.28, 5.20)
Deviation in Year 20-Year 10 Difference Associated with Policy Exposure‡	0.28 (0.08, 0.95)	0.31 (0.05, 1.89)	0.42 (0.21, 0.81)
P-interaction‡	0.04	0.2	0.01
Mean Difference Between Year 25 and Year 10 Among those Living in Areas with No Policy	7.08 (4.05, 12.38)	6.43 (3.92, 10.55)	7.00 (4.34, 11.28)
Deviation in Year 25-Year 10 Difference Associated with Policy Exposure‡	0.28 (0.08, 1.00)	0.36 (0.06, 2.20)	0.50 (0.25, 0.99)
P-interaction‡	0.05	0.3	0.05
Global Test of Interaction Between Policy Exposure Status and Exam Year (testing that all = 0)	0.1	0.4	0.08

CARDIA=the Coronary Artery Risk Development in Young Adults Study

*Participant exposure to smoke-free policies was defined as living in a census tract in a state, county, or locality that implemented a 100% ban on smoking in restaurants, bars, or workplaces.
†Results are from logistic mixed-effects models with participant-specific random intercepts. Models included the time-varying smoke-free policy exposure (coded “exposed” versus “unexposed” at a given exam), categorical exam year (15, 20, or 25 versus 10), and a policy*exam year interaction. Models adjusted for categorical exam year (year 10 as reference), baseline age, sex, race, education, marital status, income, body mass index, physical activity, diabetes, alcohol consumption, diet quality, fast food consumption, depressive symptoms, state cigarette tax, state of residence, metropolitan statistical area-level poverty, participants self-report that their workplace had a prohibition on smoking (workplace policy models only), and interactions between race*exam year and sex*exam year.
‡Estimated from interaction of time-varying smoke-free policy exposure status and categorical exam year.

Table S5. Sensitivity Analysis: Using Original Recorded BPs and Controlling for BP Medication Use. Mean Difference in Systolic Blood Pressure in Year 10 and Mean Differences in 5-Year Change in Systolic Blood Pressure by Smoke-Free Policy Exposure in Restaurants, Bars, and Workplaces From Mixed Effects Models, The CARDIA Study (N=2,606 Non-smokers, 1995-2011)*†

	Mean Difference in Systolic Blood Pressure (95% Confidence Interval), mm Hg		
	Restaurant Policy	**Bar Policy**	**Workplace Policy**
Mean Difference in Year 10 (Policy versus No Policy)	0.01 (-1.27, 1.29)	0.29 (-1.04, 1.62)	1.03 (-0.10, 2.16)
Mean 5-year Change among those Living in Areas with No Policy (Time Main Effect)	2.88 (2.50, 3.27)	2.86 (2.51, 3.22)	2.88 (2.54, 3.21)
Deviation in 5-year Change Associated with Policy Exposure‡	-0.45 (-1.03, 0.14)	-0.55 (-1.14, 0.04)	-0.78 (-1.28, -0.27)
P-interaction‡	0.1	0.07	0.003

BP=blood pressure, CARDIA=the Coronary Artery Risk Development in Young Adults Study

*Participant exposure to smoke-free policies was defined as living in a census tract in a state, county, or locality that implemented a 100% ban on smoking in restaurants, bars, or workplaces.
†Results are from linear mixed-effects models with participant-specific random intercepts. Models included the time-varying smoke-free policy exposure (coded “exposed” versus “unexposed” at a given exam), time since the year 5 exam (in 5-year increments), and a policy*time interaction. Models were adjusted for baseline age, sex, race, education, marital status, income, body mass index, physical activity, diabetes, alcohol consumption, diet quality, fast food consumption, depressive symptoms, blood pressure medication use, state cigarette tax, state of residence, metropolitan statistical area-level poverty, participants self-report that their workplace had a prohibition on smoking (workplace policy models only), and race*time and mean-centered baseline age*time interactions.
‡Estimated from interaction of time-varying smoke-free policy exposure status and time.

Table S6. Sensitivity Analysis: Using Original Recorded BPs and Controlling for BP Medication Use. Mean Difference in Diastolic Blood Pressure at Baseline and Mean Differences Between Exams by Exposure to Smoke-Free Policies in Restaurants, Bars, and Workplaces, The CARDIA Study (N=2,606 Non-smokers, 1995-2011)*†

	Mean Difference In Diastolic Blood Pressure (95% Confidence Interval), mm Hg		
	Restaurant Policy	**Bar Policy**	**Workplace Policy**
Mean Difference in Year 10 (Policy versus No Policy)	1.09 (-1.46, 3.64)	0.88 (-2.99, 4.76)	1.24 (0.13, 2.36)
Mean Difference Between Year 15 and Year 10 Among those Living in Areas with No Policy	1.86 (1.21, 2.51)	1.75 (1.09, 2.40)	1.70 (1.05, 2.34)
Deviation in Year 15-Year 10 Difference Associated with Policy Exposure‡	-2.77 (-5.43, -0.12)	-2.25 (-6.21, 1.71)	-2.02 (-3.37, -0.68)
P-interaction‡	0.04	0.3	0.003
Mean Difference Between Year 20 and Year 10 Among those Living in Areas with No Policy	-0.47 (-1.27, 0.33)	-0.84 (-1.62, -0.06)	-0.32 (-1.06, 0.43)
Deviation in Year 20-Year 10 Difference Associated with Policy Exposure‡	-1.70 (-4.32, 0.93)	-0.76 (-4.69, 3.17)	-1.92 (-3.18, -0.65)
P-interaction‡	0.2	0.7	0.003
Mean Difference Between Year 25 and Year 10 Among those Living in Areas with No Policy	0.21 (-1.05, 1.47)	-0.31 (-1.36, 0.73)	0.12 (-0.86, 1.09)
Deviation in Year 25-Year 10 Difference Associated with Policy Exposure‡	-0.47 (-3.24, 2.31)	0.49 (-3.47, 4.46)	-0.11 (-1.46, 1.24)
P-interaction‡	0.7	0.8	0.9
Global Test of Interaction Between Policy Exposure Status and Exam Year (testing that all = 0)	0.006	0.0003	0.0001

BP=blood pressure, CARDIA=the Coronary Artery Risk Development in Young Adults Study

*Participant exposure to smoke-free policies was defined as living in a census tract in a state, county, or locality that implemented a 100% ban on smoking in restaurants, bars, or workplaces.
†Results are from linear mixed-effects models with participant-specific random intercepts. Models included the time-varying smoke-free policy exposure (coded “exposed” versus “unexposed” at a given exam), categorical exam year (15, 20, or 25 versus 10), and a policy*exam year interaction. Models adjusted for categorical exam year (year 10 as reference), baseline age, sex, race, education, marital status, income, body mass index, physical activity, diabetes, alcohol use, blood pressure medication use, state cigarette tax, state of residence, metropolitan statistical area-level poverty, participants self-report that their workplace had a prohibition on smoking (workplace policy models only), and interactions between race*exam year and sex*exam year.
‡Estimated from interaction of time-varying smoke-free policy exposure status and categorical exam year.

Table S7. Sensitivity Analysis: Using Original Recorded BPs and Controlling for BP Medication Use. Mean Within-Person Changes in Systolic and Diastolic Blood Pressure Associated with Exposure to Smoke-free Policies in Restaurants, Bars, and Workplaces, From Fixed Effects Models, The CARDIA Study (N=2,606 Non-Smokers, 1995-2011)*†

	Adjusted Mean Change (95% Confidence Interval), mm HG		
	Restaurant Policy	Bar Policy	Workplace Policy
Systolic Blood Pressure‡	-0.87 (-1.63, -0.12)	-0.99 (-1.73, -0.26)	-0.56 (-1.29, 0.18)
Diastolic Blood Pressure[4]	-0.58 (-1.15, -0.01)	0.26 (-0.31, 0.84)	0.22 (-0.36, 0.80)

BP=blood pressure, CARDIA=the Coronary Artery Risk Development in Young Adults Study

*Participant exposure to smoke-free policies was defined as living in a census tract in a state, county, or locality that implemented a 100% ban on smoking in restaurants, bars, or workplaces.
†Results are from linear fixed-effects models that use only within-person variation, and can be interpreted as the mean difference in blood pressure in years where participants lived in areas with smoke-free policies compared to years where they did not, controlling for time and the covariates listed below.
‡Models adjusted for time since baseline (5-year increments), time-varying covariates (education, marital status, income, body mass index, physical activity, alcohol use, diabetes, state cigarette tax, state of residence, metropolitan statistical area-level poverty, and for whether participants reported that their workplace had a prohibition on smoking (workplace policy models only)) and interaction between time-invariant characteristics and time that were significant at the $p<0.05$ level (race*time and baseline age*time).
§As longitudinal patterns of diastolic blood pressure were not well approximated by a linear relationship with time since baseline, models for diastolic blood pressure included exam year as a categorical variable (and interactions between race*exam year and sex*exam year).

Table S8. Supplemental Analysis Examining Potential Mediating Role of Self-Reported Secondhand Smoke (SHS) Exposure*†‡§||

Association of Smoke-Free Policies in Restaurants, Bars, and Workplaces with Within-Person Changes in Self-Reported SHS Exposure

	Adjusted Mean Change in Hours/Week of Self-Reported SHS Exposure (95% CI):		
	Restaurant Policy	Bar Policy	Workplace Policy
SHS in Large Indoor Spaces	-0.79 (-1.32, -0.26)	-1.51 (-1.05, 0.03)	-0.41 (-0.94, 0.13)
SHS in Small Indoor Spaces	--	--	-0.34 (-0.83, 0.14)
SHS in the Home	-0.04 (-0.54, 0.46)	0.05 (-0.46, 0.57)	-0.26 (-0.76, 0.23)

Association of Change in Self-Reported SHS Exposure with Within-Person Changes in SBP and DBP

	Adjusted Mean Change (95% CI), mm HG	
	SHS in Large Indoor Spaces	SHS in Small Indoor Spaces
SBP	0.01 (-0.02, 0.05)	0.03 (-0.01, 0.07)
DBP	0.01 (-0.02, 0.03)	0.01 (-0.01, 0.04)

A) Mean Within-Person Changes in SBP and DBP Associated with Exposure to Smoke-free Policies in Restaurants, Bars, and Workplaces After Adjusting for Hours per Week of Self-Reported SHS Exposure

	Adjusted Mean Change (95% CI), mm HG		
	Restaurant Policy	Bar Policy	Workplace Policy
SBP	-0.84 (-1.57, -0.11)	-1.07 (-1.80, -0.34)	-0.58 (-1.29, 0.12)
DBP	-0.57 (-1.15, 0.00)	0.26 (-0.32, 0.84)	0.22 (-0.35, 0.79)

SHS=secondhand smoke, CI=confidence interval, SBP=systolic blood pressure, DBP=diastolic blood pressure

*Self-reported SHS exposure was ascertained at each exam by asking participants to report the average number of hours per week that they were exposed to tobacco smoke due to smoking by others in their home, in small spaces other than their home (e.g. office, car) or large indoor spaces other than their home (e.g. restaurant, hotel lobby). We examined associations of SHS in small spaces with workplace smoke-free policies, and SHS in large spaces with bar and restaurant smoke-free policies.

†Participant exposure to smoke-free policies was defined as living in a census tract in a state, county, or locality that implemented a 100% ban on smoking in restaurants, bars, or workplaces.

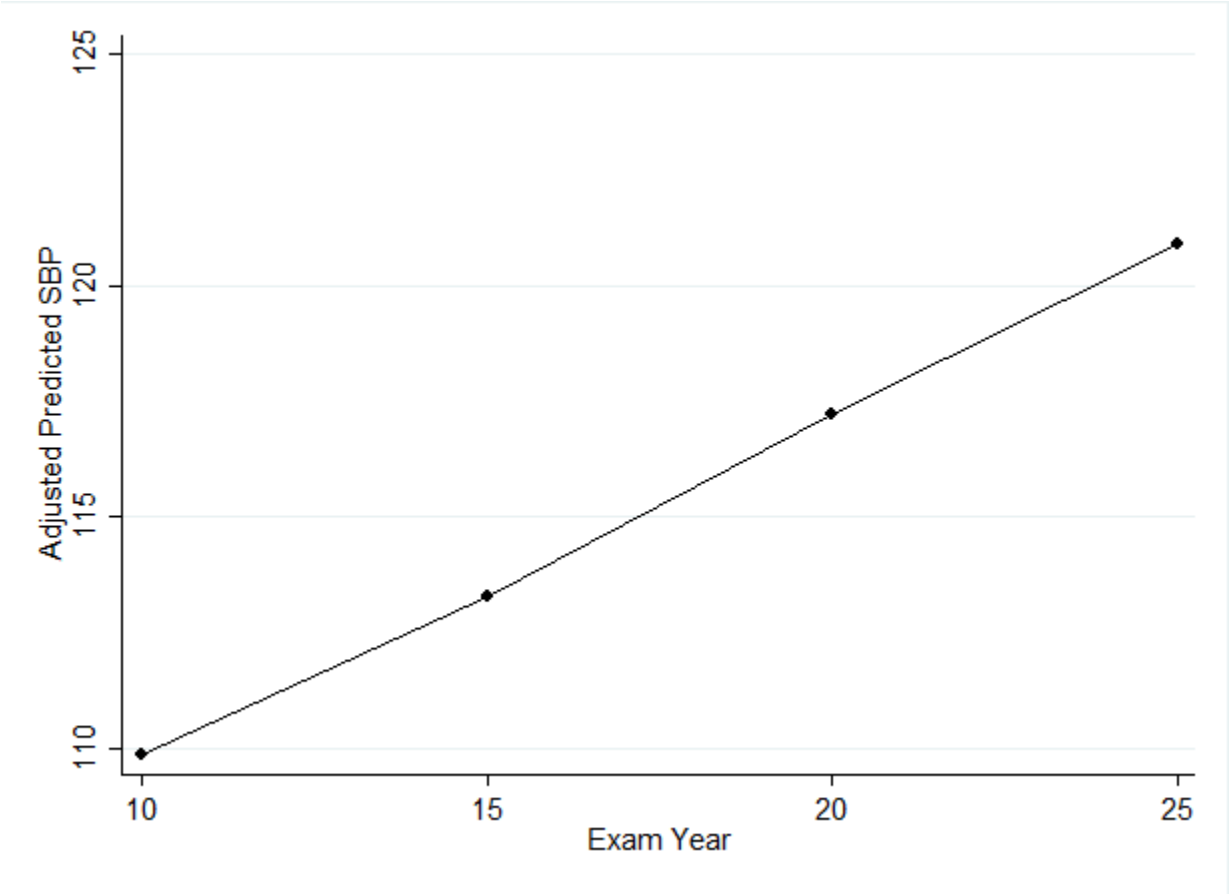
‡Statistical modeling was as follows: A) Results are from linear fixed-effects models and can be interpreted as the difference in hours per week of SHS exposure comparing years where participants lived in areas with smoke-free policies compared to years where they did not. B) Results are from linear fixed-effects models and can be interpreted as the mean difference in BP associated with a 1 hour per week increase in self-reported SHS exposure. C) Results are from linear fixed-effects models and can be interpreted as the mean difference in BP in years where participants lived in areas with smoke-free policies compared to years where they did not, controlling for SHS and other covariates.

§All models were adjusted for time since baseline (5-year increments), time-varying covariates (education, marital status, income, body mass index, physical activity, alcohol use, diabetes, state cigarette tax, state of residence, metropolitan statistical area-level poverty, and whether participants reported that their workplace had a prohibition on smoking (workplace policy models only)), and in part C, self-reported secondhand smoke exposure in large (bar, restaurant) or small (workplace) indoor places outside the home, and interaction between time-invariant characteristics and time that were significant at the $p<0.05$ level (age*time and race*time for SBP, sex*time and race*time for DBP).

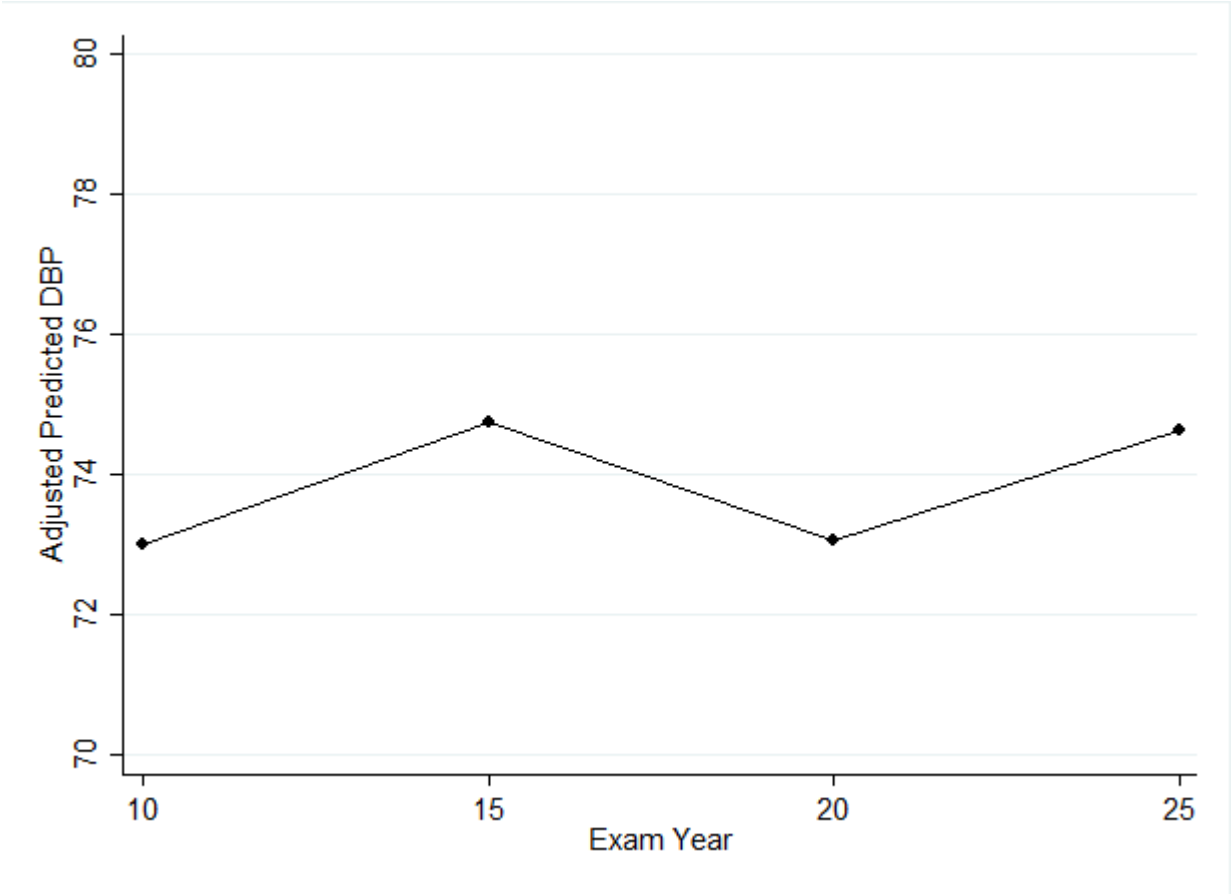
||As longitudinal patterns of DBP were not well approximated by a linear relationship with follow-up time, models for DBP included exam year as a categorical variable.

Figure S1. Adjusted Predicted Mean Systolic (A) and Diastolic (B) Blood Pressure, and Adjusted Predicted Probability of (C) Hypertension, at Each Exam (Time Modeled as Categorical to Determine Shape of Time Trends). Estimated using mixed effects linear (A, B) and logistic (C) models with subject random intercepts using the "mixed" package in Stata. Models adjusted for participant sex, race, baseline age, education, marital status, income, alcohol consumption, diet quality, fast food consumption, depressive symptoms, body mass index, physical activity, diabetes, state cigarette taxes, state of residence, and metropolitan statistical area-level poverty.

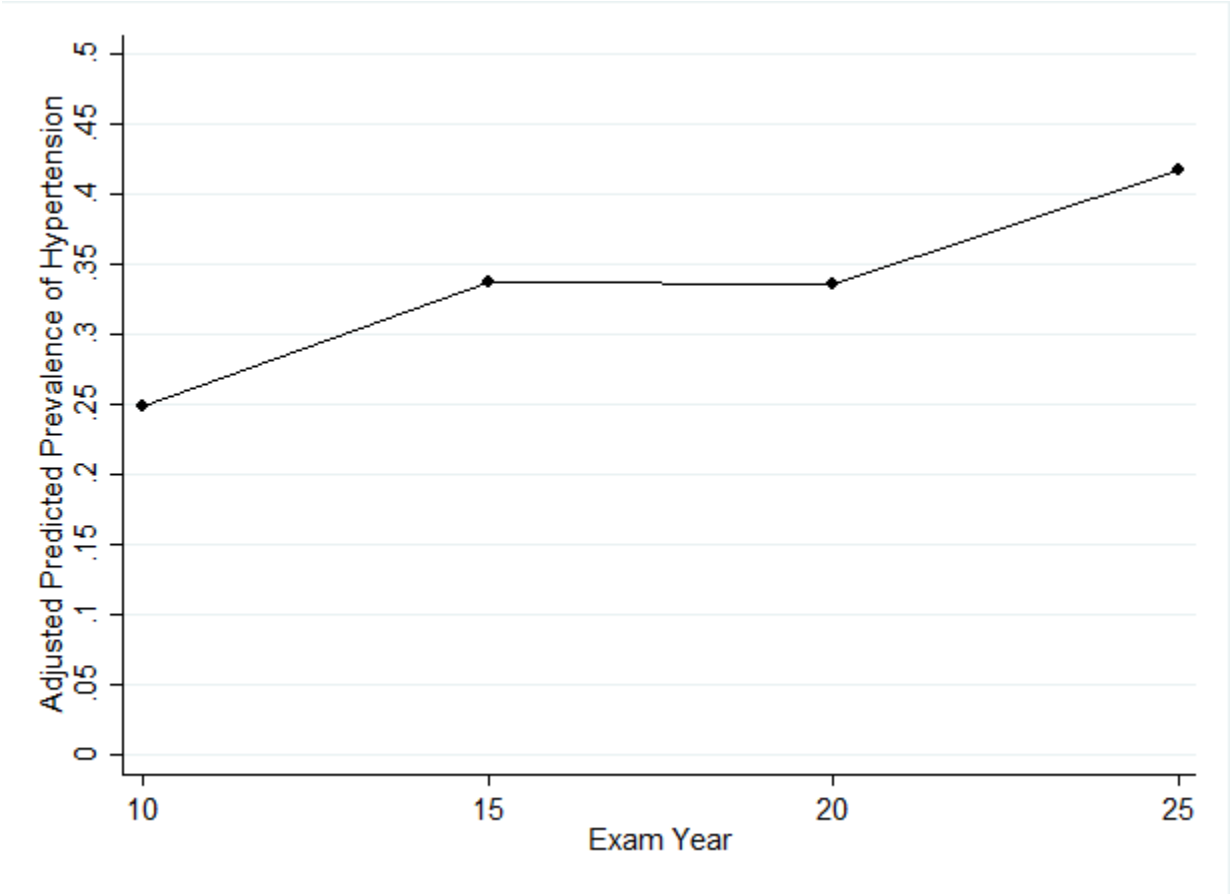
A) Systolic Blood Pressure



B) Diastolic Blood Pressure



C) Hypertension



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