ORIGINAL ARTICLE

Current Smoking Raises Risk of Incident Hypertension: Hispanic Community Health Study–Study of Latinos

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BACKGROUND
Hypertension has been implicated as a smoking-related risk factor for cardiovascular disease but the dose–response relationship is incompletely described. Hispanics, who often have relatively light smoking exposures, have been understudied in this regard.

METHODS
We used data from a 6-year follow-up study of US Hispanic adults aged 18–76 to address the dose–response linking cigarette use with incident hypertension, which was defined by measured blood pressure above 140/90 mm Hg or initiation of antihypertensive medications. Adjustment was performed for potential confounders and mediators, including urinary albumin-to-creatinine ratio which worsened over time among smokers.

RESULTS
Current smoking was associated with incident hypertension, with a threshold effect above 5 cumulative pack-years of smoking (vs. never smokers, hazard ratio for hypertension [95% confidence interval] of 0.95 [0.67, 1.35] for 0–5 pack-years, 1.47 [1.05, 2.06] for 5–10 pack-years, 1.40 [1.00, 1.96] for 10–20 pack-years, and 1.34 [1.09, 1.66] for ≥20 pack-years, $P = 0.037$). In contrast to current smokers, former smokers did not appear to have increased risk of hypertension, even at the highest cumulative pack-years of past exposure.

CONCLUSIONS
The results confirm that smoking constitutes a hypertension risk factor in Hispanic adults. A relatively modest cumulative dose of smoking, above 5 pack-years of exposure, raises risk of hypertension by over 30%. The increased hypertension risk was confined to current smokers, and did not increase further with higher pack-year levels. The lack of a smoking–hypertension association in former smokers underscores the value of smoking cessation.

Keywords: blood pressure; chronic kidney disease; epidemiology; hypertension; longitudinal study; prospective cohort study; smoking

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The US Surgeon General's office recognizes cigarette smoking as a causal risk factor for nearly 2 dozen fatal conditions.[1] Hypertensive heart disease, essential hypertension, and hypertensive renal disease have been considered among the many causes of death that may be caused by smoking.[2] Epidemiological cohort data also suggest that as compared with nonsmokers, smokers have higher risk of developing incident hypertension.[3–6] While public health messages emphasize that "there is no safe level of smoking,"[7–9] the dose–response effect of smoking is not well known for hypertension. For example, risk of

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myocardial infarction rises rapidly across the lower end of the exposure spectrum, such that even low-intensity smoking[10] and exposure to secondhand tobacco smoke[11] increase the risks by 30%. Data are insufficient to define the dose–response curve linking tobacco smoke exposure with hypertension.[11,12]

The Hispanic Community Health Study–Study of Latinos (HCHS–SOL) cohort study obtained 6-year follow-up on over 7,000 individuals who were initially free of hypertension. This cohort study represents an understudied segment of the US population with high prevalence of metabolic disorders, among whom light and intermittent smoking patterns are common.[13] In this report, we characterized the association of smoking behaviors with incident hypertension in Hispanics/Latinos.

METHODS

Study population

HCHS–SOL in 2008–2011 recruited a 2-stage population-based sample of N = 16,415 Hispanic residents of Bronx (New York), Chicago (Illinois), Miami (Florida), and San Diego (California). Eligible participants aged 18–76 years old were identified from postal mailing lists and sampled randomly on the basis of geographic area (census tract) and household unit. Pregnant women were not studied, their enrollment being deferred until the postpartum period. Prior to recruitment of participants, the project received human subjects approval from all participating institutions. Upon providing informed consent, participants completed a baseline examination (Visit 1), and 81% of surviving enrollees completed a second in-person examination (Visit 2) after approximately 6 years.[14]

Data collection

In-person English or Spanish interviews elicited ever smoking (at least 100 cigarettes lifetime cigarettes), daily or nondaily (intermittent) current smoking, number of cigarettes/day, age at smoking initiation, periods of smoking cessation, and secondhand smoke exposure. We estimated lifetime pack-years based upon age of smoking initiation, periods of quitting, and average lifetime cigarettes smoked per day. Morning blood and urine samples were collected on site, processed in a standardized way and tested at a central laboratory for a variety of clinical and metabolic variables. Three measurements of seated systolic blood pressure and diastolic blood pressure were measured using an automated sphygmomanometer (OMRON HEM-907 XL, Omron Healthcare, Lake Forest, IL). Measurements were obtained at 30 second intervals after an initial 5-minute rest, and the average of the 3 measurements was used. All staff completed centralized training and certification. The reproducibility of sitting blood pressure (BP) measurements was assessed in a sample of 59 individuals through repeated visits and led to an intraclass correlation coefficient of 0.82 and 0.80 for systolic blood pressure and diastolic blood pressure, respectively. Potential confounders obtained at Visit 1 were age, sex, Hispanic/Latino background, educational attainment, annual household income, country of birth and years since migration to the United States, health insurance status, alcohol use, secondhand smoke exposure, alternative healthy eating index-2010 diet score derived from repeat 24-hour recalls,[15] self-reported physical activity, body mass index derived from measured height and weight as kg/m^2, serum C-reactive protein, urinary albumin-to-creatinine ratio, estimated glomerular filtration rate, white blood cell count, dyslipidemia defined as measured by a combination of low-density lipoprotein cholesterol level of 160 mg/dl or above, triglyceride level of 200 mg/dl or above, high-density lipoprotein cholesterol level below 40 mg/dl, or use of cholesterol-lowering medications. Also considered as confounders were diabetes, defined as either medical history or any abnormality in measured levels of fasting glucose (126 mg/dl or above), 2-hour post-oral glucose tolerance test (200 mg/dl or above), and hemoglobin A1c (6.5% or above), and prediabetes defined as fasting glucose between 100 and 125 mg/dl or post-oral glucose tolerance test glucose between 140 and 199 mg/dl or hemoglobin A1c level between 5.7% and 6.5%.

Outcomes

We excluded individuals who at baseline had either reported use of medications for hypertension, or hypertension by JNC-7 BP criteria[16] (systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg). Incident hypertension at Visit 2 was defined by reported use of medications for hypertension, or measured BP of 140/90 mm Hg or greater.

Statistical analysis approach

From among 11,623 individuals who attended HCHS–SOL Visit 2, we excluded 3,685 individuals who had hypertension at baseline, 9 who were missing hypertension-related variables, and 214 who lacked the requisite data on smoking history, leaving 7,715 individuals for analysis (Supplementary Table S1 online). In addition, 17% of individuals were missing 1 or more of the other covariates of interest, and missing values were imputed using multivariate imputation by chained equations based on a fully conditional specification with 5 imputations.[17] The exception was income (362/7,715 missing), which was modeled with a missing-value indicator rather than imputation because of the higher frequency of missing values. Analyses accounting for complex survey design, sampling probability and nonresponse used SAS 9.4 (SAS Institute) and SUDAAN 10.0.0 (RTI). Weighting accounted for the sampling design of HCHS/SOL, so that results could be applied to the underlying populations of the study communities.

Using Visit 1 data, smokers were subclassified into groups of never, former, and current smokers. Current smokers were subdivided into those who smoked on a nondaily basis, daily smokers consuming <20 cigarettes/day, and daily smokers consuming ≥20 cigarettes/day. We also divided former smokers and current smokers according to categories of lifetime pack-years of smoking exposure (less than 5 pack-years, 5–10 pack-years, 10–20 pack-years, and

20 or more pack-years). We then related incident hypertension with never smoking (reference group), former smoking, nondaily current smoking, daily current smoking <20 cigarettes/day, and daily current smoking ≥20 cigarettes/day. Effects were quantified as adjusted relative risks (RRs) using Poisson regression with time between Visits 1 and 2 as an offset variable with robust variance estimator. Smoking status was modeled according to baseline exposure although as expected, some participants changed their smoking status over time (Supplementary Table S2 online). To examine the associations of cumulative lifetime smoking with hypertension, we examined adjusted RRs and beta coefficients with 95% confidence intervals, relating categories of lifetime pack-years of smoking exposure with the outcome. *P* values to test associations with smoking were overall tests based on Wald statistics, using 4 (for former and current smoking groups) or 8 (for cumulative lifetime smoking exposure) degrees of freedom.

Covariate adjustment

Analyses of incident hypertension were adjusted for baseline BP levels. Other confounders in the "base" multivariable model were chosen on the basis of prior knowledge of relevant mechanisms and risk factor relationships, including age, sex, center, Hispanic/Latino background, education, income, birthplace, years living in the United States, and health insurance. Urinary albumin-to-creatinine ratio was also included as an adjustment variable, based upon further analyses demonstrating an association of smoking with change in kidney function (Supplementary Table S3 online). Additional adjustment variables, which were included because they met $P < 0.05$ criteria for their association with the outcome, included body mass index, diabetes/prediabetes, and use of angiotensin-converting enzyme inhibitor or angiotensin receptor blocker medications. Adjustment for C-reactive protein was examined on the basis of the potential role of inflammation as a mediator of the link between smoking and hypertension. Other variables that were considered for adjustment (Table 1), including diet and alcohol use, secondhand smoke exposure, and history of cardiovascular events, did not affect results appreciably.

Subgroup and sensitivity analyses

Subgroup analyses were performed by age, sex, body mass index >30, and diabetes, with first-order interaction terms evaluated by the $P < 0.05$ criteria. Although we conducted primary analyses using the definition of hypertension that was established at the time the study was conducted, we substituted the 2017 American College of Cardiology/American Heart Association criteria of >130/80 mm Hg in an alternative analysis.[18]

RESULTS

Former and current smokers were disproportionately men, while nondaily smokers were the youngest group at baseline (Table 1). All other demographic and clinical variables that were considered also differed across smoker and nonsmoker groups, in most cases to the disadvantage of the heaviest smokers.

Analyses of incident hypertension were performed among 7,715 individuals without prevalent hypertension at Visit 1 who attended Visit 2 after an average interval of 6.1 years. By the time of the second visit, 474 individuals had died and were excluded from analyses. During follow-up, incident hypertension was detected in 844 baseline never smokers (cumulative incidence, 17%), 340 baseline former smokers (cumulative incidence, 26%), and 302 baseline current smokers (cumulative incidence, 21%).

We identified an association between baseline smoking status and incidence of hypertension, indicating statistically significant variation in the RR of hypertension across the multilevel smoking exposure variable ($P = 0.008$, Table 2). The highest RR of incident hypertension was found among daily current smokers consuming <20 cigarettes/day; vs. never smokers, they had an RR = 1.42 for hypertension (95% confidence interval 1.16, 1.74). The RR among daily current smokers consuming >20 cigarettes/day overlapped the null value, but this was a small of group ($N = 191$) and the 95% confidence interval around the point estimate was compatible with an increased risk (vs. never smokers, RR = 1.20, 95% confidence interval 0.89, 1.62). The RRs for nondaily current smokers and former smokers were close to the null (vs. never smokers, RR = 0.90 among nondaily smokers, and RR = 1.10 among former smokers).

Further analyses of pack-years of exposure were consistent with a threshold effect of smoking on hypertension among those who were current smokers. The global statistical test relating cumulative pack-years of smoking exposure with incident hypertension was statistically significant ($P = 0.037$). Among current smokers, all but the lowest cumulative exposure group (<5 pack-years) had RR estimates consistent with increased risk of incident hypertension. When compared with never smokers, RRs (95% confidence intervals) were 1.47 (1.05, 2.06) for 5–10 pack-years of smoking, 1.40 (1.00, 1.96) for 10–20 pack-years of smoking, and 1.34 (1.09, 1.66) for 20 or more pack-years of smoking. In contrast, among former smokers there was no evidence of either a stepwise or a threshold pattern of increased hypertension risk with greater pack-years of smoking.

We found similar RRs for smoking when we compared the "base" and fully adjusted multivariable models, and adjustment for C-reactive protein level did not alter the RR (Supplementary Tables S4 and S5 online). The RR relating smoking with hypertension was comparable in magnitude to the RR of hypertension associated with diabetes (vs. normoglycemic, RR = 1.28) (Supplementary Tables S4 and S5 online).

Table 1. Characteristics of Hispanic Community Health Study–Study of Latino participants, by smoking status

	Never smoker	Former smoker	Nondaily current smoker	Daily current smoker, <20 cigarettes/day	Daily current smoker, ≥20 cigarettes/day	*P* value
N	4,996	1,303	473	752	191	
Women (%)	58.3 (56.4, 60.1)	37.9 (34.0, 41.8)	39.6 (33.2, 46.3)	42.0 (36.5, 47.6)	36.3 (27.7, 45.8)	<0.0001
Age (%)						<0.0001
18–35 years	53.7 (51.6, 55.7)	29.0 (24.4, 34.0)	55.4 (48.8, 61.7)	45.3 (39.6, 51.1)	24.9 (15.8, 36.9)	
35–55 years	38.0 (36.1, 40.0)	48.3 (43.9, 52.8)	36.0 (30.2, 42.3)	44.6 (39.3, 50.0)	59.1 (48.7, 68.7)	
55–76 years	8.3 (7.4, 9.3)	22.7 (19.6, 26.1)	8.6 (5.8, 12.6)	10.1 (7.9, 13.0)	16.0 (11.1, 22.6)	
Annual income ≥$30,000 (%)	35.6 (33.1, 38.2)	34.8 (30.5, 39.4)	29.5 (23.8, 35.8)	27.5 (22.8, 32.7)	17.3 (12.0, 24.3)	<0.0001
Education (%)						<0.0001
<9th grade	13.7 (12.4, 15.2)	16.9 (14.2, 20.1)	13.3 (9.8, 17.7)	14.1 (10.5, 18.7)	12.7 (8.3, 19.0)	
Some high school	14.0 (12.5, 15.7)	16.0 (12.9, 19.6)	20.3 (15.5, 26.2)	21.5 (17.1, 26.5)	24.8 (16.2, 36.1)	
High school graduate	28.9 (27.0, 30.9)	27.6 (23.8, 31.8)	32.0 (26.4, 38.2)	28.8 (24.0, 34.2)	26.9 (19.6, 35.8)	
College/higher education	41.2 (39.1, 43.3)	43.4 (41.0, 45.8)	39.5 (35.1, 44.0)	34.3 (28.1, 41.2)	35.6 (30.5, 41.1)	
Lacks health insurance (%)	52.9 (50.2, 55.7)	53.7 (49.3, 58.0)	58.4 (51.5, 65.1)	49.4 (44.2, 54.6)	57.2 (47.4, 66.5)	0.0219
Heavy alcohol use[a] (%)	3.5 (2.7, 4.5)	6.7 (4.7, 9.4)	16.7 (11.7, 23.3)	12.7 (9.0, 17.5)	12.0 (6.5, 20.9)	<0.0001
Alternative healthy eating index-2010, mean	47.1 (46.7, 47.4)	49.2 (48.5, 49.9)	48.2 (47.1, 49.3)	44.6 (43.9, 45.4)	42.9 (41.9, 44.0)	<0.0001
Second-hand smoking exposure, mean hours/week	4.1 (3.5, 4.7)	5.0 (4.0, 6.0)	9.6 (6.8, 12.5)	17.4 (14.7, 20.1)	21.8 (16.6, 27.1)	<0.0001
Overweight (BMI 25–30, %)	36.9 (35.0, 38.9)	43.9 (39.5, 48.4)	34.5 (28.4, 41.0)	31.8 (27.0, 37.0)	32.7 (25.2, 41.3)	<0.0001
Obese (BMI ≥30, %)	35.4 (33.4, 37.5)	38.1 (33.9, 42.4)	34.6 (28.6, 41.2)	37.7 (32.3, 43.4)	28.6 (20.1, 38.9)	<0.0001
Prediabetes (%)	30.3 (28.5, 32.1)	37.7 (33.8, 41.7)	32.3 (26.4, 38.9)	32.8 (28.6, 37.4)	39.2 (29.6, 49.7)	<0.0001
Diabetes (%)	8.1 (7.2, 9.1)	13.9 (11.5, 16.6)	9.1 (6.2, 13.0)	7.8 (5.9, 10.2)	8.4 (5.2, 13.3)	<0.0001
Dyslipidemia (%)	34.4 (32.5, 36.4)	46.1 (41.9, 50.4)	33.6 (28.6, 39.0)	48.3 (42.3, 54.4)	54.7 (45.2, 63.8)	<0.0001
ACE inhibitor/ARB use (%)	0.6 (0.4, 0.9)	2.2 (1.3, 3.7)	0.3 (0.1, 1.9)	0.7 (0.3, 1.5)	0.4 (0.1, 1.4)	<0.0001
Coronary heart disease (%)	0.8 (0.5, 1.1)	1.4 (0.8, 2.4)	3.9 (1.5, 9.8)	1.5 (0.8, 2.9)	4.1 (1.1, 14.5)	<0.0001
Stroke (%)	0.6 (0.4, 1.1)	0.6 (0.3, 1.2)	1.4 (0.5, 3.3)	0.4 (0.1, 1.0)	0.1 (0.0, 1.1)	<0.0001
SBP (mm Hg), mean	112.9 (112.4, 113.4)	116.9 (116.1, 117.8)	113.5 (112.3, 114.8)	114.6 (113.4, 115.9)	116.8 (114.4, 119.3)	<0.0001
DBP (mm Hg), mean	68.9 (68.4, 69.3)	71.2 (70.4, 71.9)	68.8 (67.4, 70.2)	69.4 (68.6, 70.3)	72.0 (70.3, 73.7)	<0.0001
eGFR (ml/min/1.73 m^2), mean	114.4 (113.6, 115.0)	106.9 (105.3, 108.4)	111.7 (109.6, 113.8)	104.9 (103.0, 106.7)	98.6 (95.2, 101.9)	<0.0001
log-UACR (mg/g), median	2.0 (1.9, 2.0)	1.8 (1.8, 1.9)	1.8 (1.7, 1.9)	1.8 (1.8, 1.9)	1.8 (1.7, 2.0)	<0.0001
log-WBC (×10e9), mean	1.8 (1.8, 1.8)	1.8 (1.8, 1.8)	1.8 (1.8, 1.9)	2.0 (1.9, 2.0)	2.0 (1.9, 2.1)	<0.0001
log-CRP (mg/l), mean	0.6 (0.5, 0.6)	0.6 (0.5, 0.7)	0.5 (0.3, 0.6)	0.7 (0.5, 0.8)	0.8 (0.7, 1.0)	<0.0001

Table 1. Continued

	Never smoker	Former smoker	Nondaily current smoker	Daily current smoker, <20 cigarettes/day	Daily current smoker, ≥20 cigarettes/day	*P* value
Foreign born, <10 years living in the United States (%)	31.5 (28.9, 34.2)	30.5 (26.6, 34.6)	26.4 (20.4, 33.3)	24.1 (19.9, 28.9)	31.2 (22.6, 41.2)	<0.0001
Foreign born, ≥10 years living in the United States (%)	43.4 (41.3, 45.7)	52.5 (48.3, 56.8)	40.4 (34.6, 46.4)	38.7 (33.6, 44.1)	41.1 (31.8, 51.0)	<0.0001
Hispanic/Latino background (%)						<0.0001
Dominican	12.1 (10.3, 14.1)	4.6 (3.2, 6.6)	2.7 (1.3, 5.4)	6.2 (3.8, 9.9)	5.3 (2.8, 10.0)	
Central American	9.1 (7.5, 11.0)	6.8 (5.1, 9.1)	6.4 (4.0, 10.2)	5.0 (3.4, 7.2)	2.6 (1.2, 5.6)	
Cuban	14.9 (12.1, 18.1)	18.2 (14.1, 23.2)	10.3 (6.8, 15.3)	23.6 (18.2, 30.1)	55.0 (45.2, 64.4)	
Mexican	42.8 (39.3, 46.4)	44.2 (39.1, 49.3)	55.9 (48.5, 63.1)	26.4 (21.5, 32.0)	5.7 (3.2, 9.8)	
Puerto Rican	10.8 (9.4, 12.5)	16.2 (13.2, 19.7)	13.0 (9.6, 17.3)	31.4 (26.2, 37.0)	24.5 (16.5, 34.9)	
South American	5.7 (4.7, 6.9)	6.6 (4.8, 8.9)	3.6 (2.3, 5.7)	2.4 (1.4, 4.2)	1.6 (0.6, 4.0)	
Other/>1	4.6 (3.7, 5.8)	3.4 (2.0, 5.9)	8.0 (4.6, 13.5)	5.0 (3.1, 7.9)	5.3 (2.0, 13.1)	
Bronx Field Center	29.3 (26.0, 32.8)	21.2 (17.3, 25.6)	20.4 (15.5, 26.4)	36.3 (30.2, 43.0)	30.3 (21.5, 40.9)	<0.0001
Chicago Field Center	17.5 (15.2, 20.1)	17.4 (14.4, 20.8)	22.2 (17.8, 27.4)	15.4 (12.0, 19.4)	2.4 (1.1, 5.1)	
Miami Field Center	25.0 (21.2, 29.2)	29.2 (24.3, 34.7)	18.8 (13.5, 25.6)	31.0 (24.8, 37.9)	62.6 (52.2, 72.0)	
San Diego Field Center	28.2 (24.7, 32.1)	32.3 (27.4, 37.6)	38.5 (31.2, 46.4)	17.3 (13.1, 22.5)	4.6 (2.5, 8.4)	

Data are weighted, except for *N*, to reflect the sampling design of the population-based cohort study. Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BMI, body mass index; CRP, C-reactive protein; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; SBP, systolic blood pressure; UACR, urinary albumin-to-creatinine ratio; WBC, white blood cell count.

[a]Heavy consumption of alcohol defined as 7+ drinks per week for women and 14+ drinks per week for men.

Table 2. Smoking status at baseline and cumulative pack-years of smoking in relation to 6-year incidence of hypertension

	N	Incident hypertension cases	Incident hypertension RR (95% CI)
Never smoker	4,996	844	Reference
Former smoker	1,303	340	1.00 (0.85, 1.17)
Nondaily current smoker	473	64	0.90 (0.65, 1.25)
Daily current smoker, <20 cigarettes/day	752	179	1.42 (1.16, 1.74)
Daily current smoker, ≥20 cigarettes/day	191	59	1.20 (0.89, 1.62)
P value			0.008
Never smoker	4,996	844	Reference
Former smoker			
<5 pack-years	659	141	0.97 (0.76, 1.24)
5–10 pack-years	212	63	1.27 (0.90, 1.79)
10–20 pack-years	174	52	0.97 (0.72, 1.29)
≥20 pack-years	258	84	1.02 (0.80, 1.29)
Current smoker			
<5 pack-years	561	66	0.95 (0.67, 1.35)
5–10 pack-years	259	62	1.47 (1.05, 2.06)
10–20 pack-years	271	72	1.40 (1.00, 1.96)
≥20 pack-years	325	102	1.34 (1.09, 1.66)
P value			0.037

Light daily smoker defined as current use of <20 cigarettes/day and heavy daily smoker defined as current use of ≥20 cigarettes/day. Incident hypertension defined according to measured SBP >140 mm Hg or DBP >90 mm Hg (37% of incident cases), antihypertensive medications (36% of cases), or both measured BP and medications (27%). Poisson regression models were adjusted for age, sex, center, background, education, income, place of birth–years in the United States, health insurance, systolic blood pressure, diastolic blood pressure, BMI, diabetes status, ACE inhibitor/ARB medication use, and log-UACR, with years between visits used as an offset. Missing values were handled using multiple imputation. Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BMI, body mass index; BP, blood pressure; CI, confidence interval; DBP, diastolic blood pressure; RR, relative risk; SBP, systolic blood pressure; UACR, urinary albumin-to-creatinine ratio.

We found no statistical evidence of effect modification of the smoking–hypertension relationship by age, sex, obesity, or diabetes (Supplementary Table S6 online). When analyses were repeated using the 2017 definition of hypertension, results were similar (Supplementary Table S7 online).

DISCUSSION

In a community-based cohort study among Hispanic adults, current smokers appeared to have an increased risk of incident hypertension over 6 years follow-up. Among current smokers, we did not find evidence for a dose–response pattern of increasingly higher hypertension risk with greater exposure to cigarettes. Rather, analyses of cumulative pack-years suggested a threshold effect among current smokers starting at 5 pack-years of cumulative exposure to cigarettes. In contrast, former smokers, even those with the highest cumulative pack-years of past exposure, did not appear to have increased risk of hypertension.

Our report is notable as a large study of smoking and hypertension from a population-based sample of Hispanic US residents. The prior literature has included few data on Hispanics, and moreover, results have been inconsistent, with some studies reporting unfavorable BP levels and increased risk of hypertension among smokers as compared with nonsmokers,[3–6] while others suggesting lower BP levels and reduced hypertension risk among smokers.[19–24] We found that current smokers had increased risk of hypertension, after controlling for potential differences in smokers and nonsmokers in body mass index, kidney function, diabetes, health behaviors, and socioeconomic variables. This observed association is consistent with mechanistic evidence linking cigarette smoke with renal injury, endothelial damage, arterial stiffening, and inflammatory responses.[25–32]

Hispanic smokers tend to have a relatively low intensity of cigarette consumption, which may tend to be overlooked in clinical tobacco exposure assessments and smoking cessation efforts. This supports the rationale of the present study to clarify the status of smoking as a hypertension risk factor in this population group. Those who smoked on a daily basis and consumed fewer than 20 cigarettes/day were the largest current smoker group in our study, and they had an over 40% increase in risk of hypertension above never smokers. Because of the relatively small number of heavy current smokers, we were unable to generate a precise estimate of the RR of hypertension associated with 20 or more cigarettes/day (RR = 1.20, 95% confidence interval = 0.89, 1.62). Among over 7,000 persons, only 191 (<2%) were current daily smokers consuming 20 or more cigarettes/day, of whom 59 developed hypertension during follow-up. Hypertension is among the most common modifiable risk factors for cardiovascular disease and dementia,[33,34] which underscores the clinical and public health implications of our findings, particularly as the relatively young Hispanic population will increasingly represent a higher fraction of US older adults over time.

The wide range of data available in the HCHS/SOL cohort allowed us to evaluate potential mediators of the smoking–hypertension association. For example, we found that results differed little before or after adjustment for C-reactive protein, suggesting that this clinically used biomarker of inflammation does not mediate the association. To explore additional mechanisms relating smoking to hypertension, we examined the association of smoking with adverse changes over time in urinary albumin-to-creatinine ratio. This association was statistically significant, but adjustment for the renal effects of smoking by including urinary albumin-to-creatinine ratio as a covariate in the model did not reduce the increased hypertension risk among smokers. Finally, we conclude that the effect of smoking on risk of hypertension was similar in strength to the effect of other established hypertension risk factors such as

diabetes.[35,36] Diabetics had a RR of 1.28 for incident hypertension, which is roughly comparable to the RR associated with smoking in our study.

A novel aspect of this study is the assessment of incident hypertension among over 400 people with a nondaily (intermittent) smoking pattern. This provided an opportunity to examine health risks of this behavior, which is often not captured because of the design of the tobacco use questionnaires used in other studies. This pattern of smoking behavior is particularly common among Hispanic adults of low income such as those studied here[37] but may be present as many as 20% of all US smokers.[38] Incident hypertension was not elevated in our study among nondaily smokers as compared with never smokers. We previously reported that in HCHS/SOL, nondaily smokers tend to initiate cigarette use at older ages than daily smokers,[13] and as compared with daily smokers this group also has less nicotine dependence and may be more likely to be influenced by social factors rather than withdrawal symptoms.[39,40] Although we have some understanding of the characteristics of people who have nondaily smoking behaviors, data are only starting to appear about health impacts of this smoking exposure.[41] Our study is among the first to conduct a long-term interval follow-up with repeated health assessments among nondaily smokers. While no adverse hypertension outcomes were found, prior studies have concluded that there is a substantial risk of relatively low levels of smoking exposure.[11] For example, analysis of 70,913 respondents to the US National Health Interview Surveys suggested a 72% increase in mortality among nondaily smokers as compared with never smokers.[41] Hispanics in the United States, who exhibit nondaily smoking behaviors more than other groups, have largely been excluded from prior long-term epidemiologic follow-up studies and our study is among the first to address this gap in the literature.

Study limitations include the potential for uncontrolled confounding. One-time BP measurements are not equivalent to clinical criteria for hypertension. However, these measures were obtained in a clinical visit using standardized protocols and we used a mean of 3 measures. We had incomplete longitudinal information about cigarette use, thus for example, we are unable to classify nondaily smokers according to whether they may have smoked on a daily basis prior to study induction, nor could we address the effect of long-term vs. short-term intermittent smoking on risks. We did not confirm self-reported information on smoking status using biomarkers such as cotinine. Although we believe our results to be generalizable to the general population, it is possible that particular features of smoking behavior or metabolism may differ in Hispanics vs. others. Finally, electronic cigarette use was not assessed.

Our study adds to a growing body of literature that points to hypertension as a smoking-related cardiovascular risk factor. We found that a relatively modest cumulative dose of smoking, above 5 pack-years of exposure, raises risk of hypertension by 30% or more. Because an apparent threshold effect of smoking pack-years on hypertension risk was seen, this seems to implicate acute, dose-limited hemodynamic effects of cigarettes,[42,43] rather than progressive structural vascular changes induced over the long term, as the mechanism for the hypertension risk. Although even low-level exposure to tobacco is associated with well-documented health risks, perhaps individuals who are relatively early in their smoking careers, or who have relatively sparse and sporadic smoking behaviors, might be spared from excess risk of hypertension if they avoid sustained exposure to cigarettes. Indeed, former smokers, including those who had accumulated many pack-years of exposure, had similar risk of hypertension as nonsmokers, which underscores the value of smoking cessation. Our cohort provided an opportunity to characterize health risks in the Hispanic population, among whom intensity of smoking is relatively light, and nondaily smoking is as commonly as daily smoking.[40] More generally, our study broadens the population representativeness of the data on health effects of smoking to include one of the largest race and ethnic groups in the United States.

SUPPLEMENTARY MATERIAL

Supplementary data are available at *American Journal of Hypertension* online.

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DISCLOSURE

The authors declared no conflict of interest.

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