ORIGINAL RESEARCH

High-Intensity Cigarette Smoking Is Associated With Incident Diabetes Mellitus In Black Adults: The Jackson Heart Study

Wendy B. White, PhD, MPH; Loretta R. Cain, PhD, MPH; Emelia J. Benjamin, MD, ScM; Andrew P. DeFilippis, MD, MSc; Michael J. Blaha, MD, MPH; Wei Wang, PhD; Victoria Okhomina, MS; Rachel J. Keith, PhD; Mahmoud Al Rifai, MD, MPH; Sina Kianoush, MD, MPH; Michael D. Winniford, MD; Rose M. Robertson, MD; Aruni Bhatnagar, PhD; Adolfo Correa, MD, MPH, PhD; Michael E. Hall, MD, MS, FAHA

***Background*—**Previous reports on whether smoking is associated with insulin resistance and diabetes mellitus have yielded inconsistent findings. We aimed to evaluate the relationship between cigarette smoking and incident diabetes mellitus in the Jackson Heart Study.

***Methods and Results*—**Jackson Heart Study participants enrolled at baseline without prevalent diabetes mellitus (n=2991) were classified by self-report as current smokers, past smokers (smoked ≥400 cigarettes/life and no longer smoking), or never smokers. We quantified smoking intensity by number of cigarettes smoked daily; we considered ≥20 cigarettes per day (1 pack) "high-intensity." We defined diabetes mellitus as fasting glucose ≥126 mg/dL, hemoglobin A1c ≥6.5% or International Federation of Clinical Chemistry units HbA1c 48 mmol/mol, or use of diabetes mellitus medication. We estimated the adjusted associations of smoking status, intensity, and dose (pack-years) with incident diabetes mellitus using Poisson regression models. At baseline there were 361 baseline current (1–10 cigarettes per day [n=242]; ≥20 [n=119]), 502 past, and 2128 never smokers. From Visit 1 to Visit 3 (mean 8.0±0.9 years), 479 participants developed incident diabetes mellitus. After adjustment for covariates, baseline current smokers who smoked less than a pack/d and past smokers had similar rates of incident diabetes mellitus compared with never smokers (incidence rate ratios 1.04, 95% confidence interval, 0.69–1.58 and 1.08, 95% confidence interval, 0.82–1.42, respectively). Baseline current high-intensity smokers had a 79% (95% confidence interval, 1.14–2.81) higher incidence of diabetes mellitus compared with never smokers. Smoking dose (per 10 pack-years) was also associated with a higher incidence of diabetes mellitus (incidence rate ratios 1.10, 95% confidence interval, 1.03–1.19) in adjusted models.

***Conclusions*—**High-intensity cigarette smoking and smoking pack-years are associated with an increased risk of developing diabetes mellitus in blacks. **(*J Am Heart Assoc*. 2018;7:e007413. DOI: 10.1161/JAHA.117.007413.)**

Key Words: diabetes mellitus • race and ethnicity • smoking

Cigarette smoking is a major cause of preventable disease and mortality worldwide, and the impacts of smoking on cancer and cardiovascular disease are well established.[1–3] Smoking also may be an independent risk factor for insulin resistance and diabetes mellitus; however, there have been discrepant findings from previous studies examining these relationships.[4–6] Furthermore, despite evidence that smoking is associated with incident diabetes mellitus in certain ethnic groups, little is known about the association of cigarette smoking with diabetes mellitus in blacks, who are disproportionately affected by obesity and diabetes mellitus.[7,8]

Racial/ethnic disparities in smoking behaviors have been described, including longer smoking durations for blacks compared with whites and lower cessation rates in blacks compared with both whites and Hispanics.[9] In the NHANES II (National Health and Nutrition Examination Study), smoking rates were significantly higher in newly diagnosed black versus white individuals with diabetes mellitus (42% versus 29%,

From the Tougaloo College, Tougaloo, MS (W.B.W.); Department of Data Sciences, University of Mississippi Medical Center, Jackson, MS (L.R.C., W.W., V.O.); Department of Medicine, Boston University School of Medicine, Boston, MA (E.J.B.); Department of Medicine, University of Louisville, Louisville, KY (A.P.D.); Ciccarone Center for the Prevention of Heart Disease, Johns Hopkins School of Medicine, Baltimore, MD (M.J.B., S.K.); Diabetes and Obesity Center, University of Louisville, Louisville, KY (R.J.K., A.B.); Department of Medicine, University of Kansas School of Medicine, Wichita, KS (M.A.R.); Department of Medicine, University of Mississippi Medical Center, Jackson, MS (M.D.W., A.C., M.E.H.); Department of Medicine, Vanderbilt University Medical Center, Nashville, TN (R.M.R.).

Correspondence to: Michael E. Hall, MD, MS, FAHA, Departments of Medicine and Physiology and Biophysics, University of Mississippi Medical Center, 2500 North State St, Jackson, MS 39216. E-mail: mehall@umc.edu

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Clinical Perspective

What Is New?

- Current high-intensity smoking (≥1 pack cigarettes per day) and smoking dose (pack-years) are associated with incident diabetes mellitus in blacks.
- Smoking intensity and dose were associated with impaired pancreatic beta cell function (homeostatic model assessment of pancreatic beta cell function).

What Are the Clinical Implications?

- Although smoking cessation should be encouraged for everyone, certain high-risk groups such as blacks who are disproportionately affected by diabetes mellitus should be targeted for cessation strategies.

respectively).[10] Behavioral smoking differences, coupled with a higher risk of other known diabetes mellitus risk factors, may augment the risk of diabetes mellitus in blacks who smoke.

Despite evidence suggesting a causal role for smoking predisposing to diabetes mellitus, the potential mechanisms that mediate this relationship are not well known. It has been hypothesized that smoking-induced inflammation or obesity may contribute to diabetes mellitus onset.[11–13] Although smoking has been associated with reduced body mass index (BMI), smoking is positively associated with visceral adiposity, a known risk factor for diabetes mellitus.[14] We aimed to examine the associations of cigarette smoking status, intensity, and dose with incident diabetes mellitus in a large, prospective black cohort, the Jackson Heart Study (JHS). We also evaluated the influence of measures of adiposity (body mass index [BMI] and waist circumference [WC]), and inflammation (high-sensitivity C-reactive protein [hs-CRP]) in this relationship.

Research Design and Methods

Study Cohort

The JHS is a large, prospective cohort study of 5301 blacks aged 21 to 84 years who were recruited from the tri-county area surrounding Jackson, MS from 2000 to 2004. We collected data at 3 participant examinations; we completed Visit 3 from 2009 to 2013. The 3 participating institutions (University of Mississippi Medical Center, Tougaloo College, and Jackson State University) approved the study and the University of Mississippi Medical Center Institutional Review Board approved the study. Each participant provided written, informed consent. The data, analytic methods, and study materials can be made available to other researchers for purposes of reproducing the results or replicating the procedure by following the JHS publications procedures and data use agreements.[15]

Inclusion/Exclusion Criteria

We included participants with available follow-up data including diabetes mellitus outcome measures and self-reported smoking status at Visit 3. We excluded participants with prevalent diabetes mellitus at baseline (Visit 1, n=733). We also excluded participants with relevant missing variables at Visit 3 (n=435).

Assessment of Smoking Status, Intensity, and Duration

We obtained self-reported cigarette smoking status via questionnaire at baseline. We defined participants who smoked >400 cigarettes in their lifetime as "ever smokers." We classified those who did not as "never smokers." We classified participants who gave a positive response to the question "Do you now smoke cigarettes" as "baseline current smokers." Participants who were classified as "ever" smokers who did not smoke for at least 30 days at the time of the examination were classified as "past" smokers. Further information related to number of cigarettes smoked daily also was collected. We defined smoking intensity as number of cigarettes smoked per day. We defined high-intensity smoking as smoking more than 20 cigarettes daily based on previous published literature.[16] We measured cumulative dose of smoking in "pack-years," which we calculated by multiplying the number of packs of cigarettes per day by the number of years smoking. One participant who identified as a current smoker did not report the number of cigarettes smoked daily and therefore was not included in analyses of smoking intensity or dose relationships.

Diabetes Mellitus Ascertainment

We defined diabetes mellitus as fasting plasma glucose ≥126 mg/dL, hemoglobin A1c (HbA1c) ≥6.5% or International Federation of Clinical Chemistry units HbA1c 48 mmol/mol, or use of diabetes mellitus medications within 2 weeks before the examination. We defined incident diabetes mellitus as presence of diabetes mellitus at Visit 3 in those participants who did not have diabetes mellitus at the baseline visit.

Baseline Covariates and Biomarker Measures Measured at Visit 1

The baseline examination included a home interview, self-administered questionnaires, and a clinic visit that included blood and urine collections. Other evaluated risk factors

included education level, physical activity, and alcohol consumption in the past 12 months. Participants self-reported the highest level of education attained (less than or greater than a high school diploma). Physical activity was defined as a summary score of the intensity, frequency, and duration of activities encountered daily, which has been previously validated.[17]

We recorded age, sex, and BMI at the baseline JHS examination. We averaged 2 measures of WC taken at the level of the umbilicus on each participant to determine baseline WC. Hypertension was defined as blood pressure ≥140/90 mm Hg or use of blood pressure–lowering medication. Additional information such as use of diuretics and statins was also collected at each visit. Psychosocial measures including global stress was measured using the Global Perceived Stress Scale, which is an 8-item measure of global perceptions of stressors associated with ongoing stressful conditions such as employment, relationships, neighborhood, meeting basic needs, and others.[18]

We asked each participant to fast overnight for at least 12 hours before their clinic visit. We collected blood and urine samples according to the National Committee for Clinical Laboratory Standards as previously reported.[19] We measured glucose and insulin concentrations using standard procedures that met the College of American Pathologists accreditation requirements. We used a high-performance liquid chromatography system (Tosoh Corporation, Tokyo, Japan) to measure glycosylated HbA1c concentrations. We measured total cholesterol from plasma with the cholesterol oxidase method (Roche Diagnostics, Indianapolis, IN) on a Roche COBAS FARA centrifugal analyzer. We measured hs-CRP using the immunoturbidimetric CRP-Latex assay (Kamiya Biomedical Company, Seattle, WA) with a Hitachi 911 analyzer (Roche Diagnostics).[20] The homeostatic model assessment for insulin resistance (HOMA-IR) was calculated using the equation HOMA-IR=(fasting plasma insulin [μU/mL])×(fasting plasma glucose [mmol/L])÷22.5. HOMA-B, a measure of pancreatic beta cell function, was measured using the equation: HOMA-B=20×insulin÷fasting plasma glucose−3.5.

Statistical Analyses

We compared baseline characteristics with χ^2 tests or 1-way ANOVA for differences among never, past, and baseline current smokers. We performed multivariable Poisson regression models to estimate the association between smoking status at Visit 1 and incident diabetes mellitus from Visit 1 to Visit 3 yielding incidence rate ratios (IRRs) and 95% confidence intervals (CI). Poisson regression models were utilized to accommodate different follow-up time intervals among participants between Visit 1 and Visit 3. In the Poisson regression models, the models were offset by log (years), which were calculated as the time between Visit 1 and Visit 3. Covariates included age, sex, BMI, hypertension, total cholesterol, education, physical activity, prevalent cardiovascular disease, diuretic use, statin use, perceived stress, menopause status, and alcohol consumption. Smoking dose (pack-years) was analyzed as a continuous variable (per 10 pack-years). We conducted additional secondary analyses adjusting for WC, and adjusting for hs-CRP. Multivariable linear regression models were used to assess the cross-sectional associations between smoking status and HOMA-IR and HOMA-B at Visit 1. All statistical analyses were performed using SAS 9.4 software (SAS Institute, Cary, NC). All *P* values were 2-tailed and *P* values <0.05 were considered statistically significant.

Results

We studied 2991 participants with follow-up data at Visit 3. At baseline (Visit 1) we identified 361 current smokers, 502 past smokers, and 2128 never smokers. Table 1 shows the baseline characteristics of the never smokers, past smokers, and current smokers. Compared with never smokers, baseline current and past smokers were more likely to be men (*P*<0.001). BMI was highest in never smokers and lowest in baseline current smokers (*P*<0.001). Similarly, WC was lowest in baseline current smokers (*P*<0.001). Baseline current smokers were younger, less educated, and were more likely to have consumed alcohol in the past year than never smokers and past smokers (all *P*<0.001). Table 2 shows the baseline characteristics of participants in each analyzed smoking intensity category. High-intensity cigarette smokers (>20 cigarettes per day) were more likely men, less educated, and more likely to have consumed alcohol in the past 12 months. There were no significant differences in unadjusted fasting glucose levels (*P*=0.71) or hemoglobin A1c (*P*=0.67); however, unadjusted plasma insulin levels were lower in high-intensity smokers (*P*<0.0001). BMI and WC were also significantly lower in high-intensity smokers (*P*<0.0001 and *P*=0.006, respectively).

Between baseline and Visit 3, 479 participants developed incident diabetes mellitus (mean follow-up time was 8.0±0.9 years). There were 320 incident diabetes mellitus cases in never smokers (unadjusted incidence rate 15%) compared with 99 in past smokers (unadjusted incidence rate 20%) and 60 in current smokers (unadjusted incidence rate 17%). After multivariable adjustment, smoking status was not significantly associated with risk of incident diabetes mellitus (Table 3). However, further analysis of smoking intensity showed that among baseline current smokers who smoked ≥20 cigarettes daily, there was a significantly higher incidence of diabetes mellitus compared with never smokers (*P*=0.01), which remained significant after further adjustment

Table 1. Characteristics of JHS Participants by Baseline Smoking Status

Variable	Never Smokers (n=2128)	Past Smokers (n=502)	Current Smokers (n=361)	*P* Value
Age, y	52±12	59±11	52±10	<0.001
Sex, % men	31.6	49.8	50.4	<0.001
At least high school diploma/GED, %	79.8	71.2	71.0	<0.001
Ideal health indicator via physical activity, %	21.9	24.5	17.2	0.04
Alcohol consumption in past 12 mo, %	45.0	54.3	75.6	<0.001
BMI, kg/m^2	31.7±7.2	30.74±6.1	29.0±6.6	<0.001
Waist circumference, cm	98.6±15.9	100.5±14.7	95.5±14.0	<0.001
Hypertension %	45.6	59.2	42.1	<0.001
Hemoglobin A1c, % (IFCC mmol/mol)	5.5±0.5, 37	5.6±0.5, 38	5.5±0.5, 37	<0.001
Fasting glucose, mg/dL	89.7±8.7	92.4±9.3	90.3±9.4	<0.001
Insulin (plasma), IU/mL	15.9±8.6	15.8±8.2	13.9±8.7	<0.001
High-sensitivity C-reactive protein, mg/L	0.5±0.6	0.5±0.8	0.5±0.8	0.14
Total cholesterol, mg/dL	199±39	202±42	195±41	0.02

BMI indicates body mass index; GED, general equivalency diploma; IFCC, International Federation of Clinical Chemistry units; JHS, Jackson Heart Study.

for WC (*P*=0.01). Smoking dose (pack-years) was also associated with a higher incidence of diabetes mellitus (*P*=0.02). We further adjusted these models to include hs-CRP to assess the role of inflammation in the increased risk of diabetes mellitus in smokers. Addition of hs-CRP only slightly attenuated the risk of incident diabetes mellitus (*P*=0.03). We also further adjusted for medications including diuretics and statins that have been implicated in hyperglycemia. The findings remained similar after adjusting for diuretic or statin use (IRR 1.73, 95% CI, 1.10–2.73 for high-intensity smoking and IRR 1.10, 95% CI, 1.01–1.19 for smoking dose). As mental stress has also been implicated in the pathogenesis of diabetes mellitus,[21] we additionally adjusted for perceived stress and there were no significant changes in the relationship between high-intensity smoking (IRR 1.76, 95% CI, 1.12–2.78) or dose (IRR 1.10, 95%, CI 1.01–1.20). The risk of diabetes mellitus has also been reported to increase in postmenopausal women.[22] After adjustment for menopause

Table 2. Baseline Participant Characteristics Among Smoking Intensity Groups

Variable	Never Smokers (n=2128)	1 to 19 Cigarettes/D (n=241)	≥20 Cigarettes/D (n=119)	*P* Value
Age, y	52±13	51±11	53±9	0.38
Sex, % men	31.6	44.4	62.2	<0.001
At least high school diploma/GED, %	79.8	74.1	64.6	<0.001
Ideal health indicator via physical activity, %	21.9	19.5	12.6	0.04
Alcohol consumption past 12 mo, %	45	78	72	<0.001
BMI, kg/m^2	31.7±7.2	29.7±6.8	27.6±6.1	<0.001
Waist circumference, cm	98.6±15.9	96.5±13.8	93.6±14.5	0.005
Hypertension %	45.6	41.9	42.9	0.48
Hemoglobin A1c, %	5.5±0.5	5.5±0.5	5.4±0.5	0.67
Fasting glucose, mg/dL	89.7±8.7	90.5±8.9	90.1±10.2	0.71
Insulin (plasma), IU/mL	15.9±8.6	14.4±9.4	12.7±7.1	<0.001
High-sensitivity C-reactive protein, mg/L	0.5±0.6	0.5±0.9	0.5±0.7	0.10
Total cholesterol, mg/dL	199±39	200±42	185±37	<0.001

BMI indicates body mass index; GED, general equivalency diploma.

Table 3. Associations Between Baseline Smoking Status at Visit 1 and Incident Diabetes Mellitus at Visit 3

Diabetes mellitus Incidence	Never Smokers	Past Smokers	Current Smokers	1 to 19 Cigarettes/D	≥20 Cigarettes/D
	320/2128 15.0%	99/502 19.7%	60/361 16.6%	34/241 14.1%	26/119 21.9%

	Smoking Status		Smoking Intensity		Smoking Dose
Model	Past vs Never Smokers	Current vs Never Smokers	Current (1–19 Cigarettes/D) vs Never Smokers	Current (≥20 Cigarettes/D) vs Never Smokers	Exposure Pack-Y/10 Pack-Y*
Model 1†	1.11 (0.84–1.46) 0.46	1.29 (0.93–1.77) 0.12	1.04 (0.69–1.58) 0.85	1.79 (1.14–2.82) 0.01	1.10 (1.03–1.19) 0.02
Model 2‡	1.08 (0.82–1.42) 0.59	1.28 (0.93–1.77) 0.13	1.04 (0.69–1.58) 0.85	1.79 (1.14–2.81) 0.01	1.10 (1.02–1.19) 0.02
Model 3§	1.06 (0.80–1.39) 0.69	1.21 (0.88–1.67) 0.24	0.99 (0.65–1.50) 0.95	1.67 (1.07, 2.63) 0.03	1.09 (1.00, 1.18) 0.04

Values are given as IRR (95% CI) *P* Value. CI indicates confidence interval; IRR, incidence rate ratio.
*Pack-y dose was analyzed as continuous variable and IRR corresponds to 10 cigarette pack-y effect. Only current smokers and never smokers are involved in the analysis.
†Adjusted for age, sex, education, physical activity, alcohol consumption in the past 12 mo, body mass index, hypertension, total cholesterol, and prevalent cardiovascular disease.
‡Model 1 plus waist circumference.
§Model 2 plus high-sensitivity C-reactive protein.

status, the risk for incident diabetes mellitus increased in high-intensity smokers (IRR 2.01, 95% CI, 1.00–4.06), although not quite statistically significant.

We also assessed insulin resistance (HOMA-IR) and pancreatic beta cell function (HOMA-B) to gain mechanistic insight into the relationship between smoking and incident diabetes mellitus. Smoking status and intensity were not significantly associated with HOMA-IR; however, smoking dose (pack-years) was associated with a slightly lower HOMA-IR, suggesting that insulin resistance is not the mechanism of the relationship between smoking and incident diabetes mellitus (Table 4). However, HOMA-B was lower in current compared with never smokers, and smoking intensity and smoking dose were associated with lower HOMA-B (Table 5). Therefore, reduced beta cell function is the likely factor leading to our findings.

Discussion

Our findings from a large cohort of blacks demonstrate high-intensity cigarette smoking (at least 1 pack per day) and higher pack-years of smoking were associated with incident diabetes mellitus. Additionally, adjusting for WC and hs-CRP minimally attenuated the incidence rate.

In 2014, the Surgeon General Report was published on the health consequences of smoking that stated there is sufficient evidence "to infer that cigarette smoking is a cause of diabetes" and that the "risk of developing diabetes is 30% to 40% higher in active smokers than nonsmokers."[23] Several large prospective cohorts have demonstrated increased risk of incident diabetes mellitus in current smokers compared with never smokers. The Nurses' Health Study, a prospective study of over 100 000 women with 24 years of follow-up,

Table 4. Associations Between Baseline Smoking Status at Visit 1 and Log-Transformed HOMA-IR Among Those Without Diabetes Mellitus at Baseline

	Smoking Status		Smoking Intensity		Smoking Dose
Model	Past vs Never Smokers	Current vs Never Smokers	Current (1–19 Cigarettes/D) vs Never Smokers	Current (≥20 Cigarettes/D) vs Never Smokers	Exposure Pack-Y/10 Pack-Y
Model 1*	0.03 (0.03) 0.36	−0.03 (0.03) 0.32	−0.04 (0.04) 0.29	−0.03 (0.06) 0.61	−0.01 (0.01) 0.21
Model 2†	0.004 (0.03) 0.88	−0.05 (0.03) 0.14	−0.06 (0.04) 0.16	−0.04 (0.06) 0.46	−0.02 (0.01) 0.11
Model 3‡	0.001 (0.03) 0.98	−0.06 (0.03) 0.06	−0.07 (0.04) 0.09	−0.05 (0.06) 0.28	−0.02 (0.01) 0.04

Values are given as beta (SE) *P* Value. HOMA-IR indicates homeostatic model assessment for insulin resistance; SE, standard error.
*Adjusted for age, sex, education, physical activity, alcohol consumption in the past 12 m, body mass index, hypertension, total cholesterol, and prevalent cardiovascular disease.
†Model 1 plus waist circumference.
‡Model 2 plus high-sensitivity C-reactive protein.

Table 5. Associations Between Baseline Smoking Status at Visit 1 and Log-Transformed HOMA-B Among Those Without Diabetes Mellitus at Baseline

	Smoking Status		Smoking Intensity		Smoking Dose
Model	Past vs Never Smokers	Current vs Never Smokers	Current (1–19 Cigarettes/D) vs Never Smokers	Current (≥20 Cigarettes/D) vs Never Smokers	Exposure Pack-Y/10-Pack-Y
Model 1*	−0.03 (0.04) 0.37	−0.13 (0.04) 0.00	−0.15 (0.05) 0.00	−0.10 (0.07) 0.17	−0.03 (0.01) 0.03
Model 2†	−0.06 (0.04) 0.14	−0.14 (0.04) 0.00	−0.16 (0.05) 0.00	−0.11 (0.07) 0.12	−0.03 (0.01) 0.01
Model 3‡	−0.06 (0.04) 0.09	−0.17 (0.04) 0.00	−0.19 (0.05) 0.00	−0.15 (0.07) 0.04	−0.04 (0.01) 0.00

Values are given as beta (SE) *P* Value. HOMA-B indicates homeostatic model assessment for pancreatic beta cell function; SE, standard error.
*Adjusted for age, sex, education, physical activity, alcohol consumption in the past 12 mo, body mass index, hypertension, total cholesterol, and prevalent cardiovascular disease.
†Model 1 plus waist circumference.
‡Model 2 plus high-sensitivity C-reactive protein.

showed a dose-dependent increased risk of incident type 2 diabetes mellitus in current smokers compared with nonsmokers (relative risk 1.39 for current smokers smoking 1–14 cigarettes per day and relative risk 1.98 for ≥25 cigarettes per day).[24] Whereas that study had a very large number of participants, over 93% of the participants were white and they did not analyze specific races separately. In the Insulin Resistance Atherosclerosis Study, current smokers exhibited a significantly higher incidence of diabetes mellitus compared with never smokers (odds ratio 2.66, P=0.001).[6] Although many of these studies demonstrated positive and dose-dependent associations with current cigarette smoking with incident diabetes mellitus, most included very limited numbers of black participants. Other multiethnic cohort studies such as the MESA (Multiethnic Study of Atherosclerosis) did not observe independent associations between cigarette smoking and insulin resistance.[5]

Given the discrepant published findings and limited data evaluating the link between smoking and incident diabetes mellitus in blacks, our findings have important implications. This is especially important given that almost 18% of blacks are current smokers and blacks have higher rates of diabetes mellitus compared with other ethnic groups.[25] Furthermore, many previous studies reporting that smoking was associated with incident diabetes mellitus[19] were from a period of time when smoking intensity was higher and composition of cigarettes differed from that of current smokers, including unfiltered cigarettes and higher tar content.[26,27]

Inflammation has been proposed as a precursor to the development of type 2 diabetes mellitus.[28] Previous studies, including analyses from the JHS, have observed a positive relationship between hs-CRP (biomarker of inflammation) and incident diabetes mellitus.[13,29] However, this association was largely explained by measures of obesity including BMI and WC. In our study, we further adjusted for hs-CRP, which only slightly attenuated the incidence rate, suggesting inflammation may play a minor role in the relationship between smoking and incident diabetes mellitus.

We further evaluated relationships of smoking status, intensity, and dose with HOMA-IR and HOMA-B, measures of insulin resistance and pancreatic beta cell function, respectively. Only smoking dose was slightly associated with HOMA-IR, suggesting insulin resistance is not the main contributor to the association of high-intensity smoking with incident diabetes mellitus. However, HOMA-B was lower in current smokers compared with never smokers and with increasing smoking dose (pack-years), suggesting reduced beta cell function is the likely factor leading to our findings.

Our report does have some limitations. First, the JHS is an all-black cohort, so our findings may not generalize to other ethnic groups. Second, our findings are based on a small number of incident cases of diabetes mellitus, particularly among current smokers. However, to our knowledge, our study is the largest study of the relationship between smoking and incident diabetes mellitus in blacks who have higher rates of diabetes mellitus. Most participants who developed diabetes mellitus were prediabetic (HbA1c 5.7–6.4 mmol/mol). Therefore, current high-intensity smoking may impose an additional risk in those with a propensity to diabetes mellitus. Cigarette smoking was self-reported in the JHS; therefore, there is potential for misclassification. Unfortunately, urine cotinine levels were not measured in the participants, which would have provided a more objective exposure level of cigarette smoking. Furthermore, the type of cigarettes that the participants smoked was not available for analysis. We did not account for change in smoking status over follow-up because of limited availability of longitudinal smoking data in the JHS. As our study was observational, we cannot exclude residual confounding, and cannot infer causality. It is also possible that genetic variation, which we did not assess, may play a role in the impact of smoking on incident diabetes mellitus.[30] Last, we cannot adequately

account for second-hand cigarette smoking since such data were not collected at Visit 1.

In conclusion, in a large prospective cohort of blacks, baseline current high-intensity cigarette smoking and smoking pack years were associated with incident diabetes mellitus in adjusted models. In general, people should be encouraged to stop smoking. Our data suggest that certain high-risk groups among blacks should be particularly identified for smoking cessation.

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Disclosures

None.

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