Investigating the interaction of diet quality on the association between air pollution exposure and diabetes incidence in the Hispanic Community Health Study/Study Of Latinos

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Abstract

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Background
Type 2 Diabetes (T2D) is a chronic disease linked to increased morbidity and mortality. Hispanic and Latino groups have a disproportionately high prevalence of diagnosed T2D cases among ethnic and racial groups in the United States. Dietary patterns that feature the consumption of processed meats and sugar-sweetened beverages have been linked to development of chronic disease, including T2D. Increased exposure to ambient air pollutants have also been linked to the development of T2D. It remains uncertain whether diet may modify the risk of diabetes associated with air pollution.

Methods
We investigated the association between air pollution, diet, and incident T2D between visit 1 and visit 2 of the Hispanic Community Health Study/Study of Latinos (HCHS/SOL). We utilized a spatio-temporal air pollution model to estimate outdoor residential exposure to particulate matter with an aerodynamic diameter of 2.5 microns ($PM_{2.5}$) and nitrogen dioxide (NO_2) over the follow-up period at each participant's residential address. Diet was assessed at baseline using the Alternative Healthy Eating Index-2010 (AHEI-2010). Using covariate adjusted Poisson regression models, we investigated the association with air pollution and T2D, diet and T2D, and the potential effect modification of diet on the association between air pollution and T2D, adjusting for age, sex, income, education, family history of diabetes, heritage-study center, and waist-to-hip ratio.

Results
In our analytic sample of 7,534 study participants without diabetes at baseline and with complete data available, 876 incident cases of diabetes were observed. Primary models of $PM_{2.5}$ (IRR: 1.21; 95% CI: 1.01, 1.44) and NO_2 (IRR: 1.11; 95% CI: 1.02, 1.22) demonstrated an association with incidence of diabetes, which was attenuated with adjustment for adiposity and study site. Primary models between AHEI-2010 and incidence of diabetes demonstrate a suggestive but not statistically significant association between healthy eating and diabetes (IRR: 0.93; 95% CI: 0.83, 1.04). We did not find evidence that diet modified the association between ambient air pollution and incident diabetes.

Conclusion
Though initial models demonstrate the association we expected, further adjustment attenuated the estimates we observed. Additional research, including different approaches to dietary quality ascertainment, may be necessary to better understand these relationships.

Introduction

Type 2 Diabetes (T2D) is a chronic disease impairing regulation of the body's blood glucose levels and characterized by insulin resistance.[1] Globally, there are an estimated 830 million people living with diabetes, 95% of whom have T2D.[2] T2D is a concern for Hispanic/Latino groups due to the prevalence of diagnosed and undiagnosed cases within this population consistently ranked highest among race and ethnicity groups.[3] Known risk factors for T2D include family history of diabetes, a sedentary lifestyle, and an individual's dietary pattern. While not all risk factors can be changed, T2D prevention emphasizes the modifiable risk factors such as physical activity and dietary patterns, thus the need to better understand how they may interact is of great importance.

Dietary patterns and high or excessive caloric intake are established risk factors for T2D. The World Health Organization (WHO) and others emphasize the role of a healthy diet such as limiting overconsumption of sugar and saturated fat, maintenance of a healthy body weight, and physical activity to prevent diabetes.[2] The content of the diet is an important consideration for long-term risk of development of T2D. For example, plant-based diets, when compared to meat based diets, are suggested to present a lower risk in incidence of T2D.[3] A diet high in calories from fats and sugars combined with a sedentary lifestyle facilitate the growth of adipose tissue. While high BMI is considered a risk factor, visceral obesity and ectopic fat have been shown to be more strongly associated with risk of T2D.[3] Specific dietary patterns that consider these and other factors that have been associated with chronic disease development have been linked to better health outcomes.[4–6] Chiuve et al. demonstrate two well established dietary pattern indices that are associated with chronic disease prevention.[7] The Healthy Eating Index-2005 and Alternative Healthy Eating Index-2010 (AHEI-2010) were both found to be inversely correlated with chronic disease risk. Of these two, the AHEI-2010 was shown to be a stronger predictor.[7] Thus, it is important to mitigate the risk of T2D by managing this dietary component.

Environmental factors are yet another factor of importance when it comes to the development of T2D. Short- and long-term exposure to environmental pollutants particulate matter 2.5 and 10 microns in diameter ($PM_{2.5,}$ PM_{10}), nitrogen oxides (NO_2, NO_x), and ozone (O_3) [8–11] have been linked to increased morbidity and mortality, and more recently also linked to risk of development of T2D.[12–17] Links to other harmful health effects include inflammation, oxidative stress, and other chronic diseases such as cardiovascular disease.[8,15–17] A systematic review conducted by Yang and colleagues provides varying levels of evidence for air pollutants' association to these outcomes.[18] Of the 21 studies that investigated incidence of diabetes, 15 found an association between some air pollutant. Of these 15 studies, 12 included either $PM_{2.5}$, NO_2, or both. Weinmayr et al.,[5] investigated the association between PM and incidence of T2D and found that individuals who lived closer than 100 meters to a busy road had a 30% higher risk than those who lived 200 meters away.[5] These findings point to an association between increased exposure to traffic-related air pollutants and the risk of T2D.

With such evidence suggesting exposure to environmental air pollutants fulfilling a role in the development of T2D, assessing the interplay between the dietary component and air

pollutants is an important question, as both are potentially modifiable risk factors. A few other investigators have studied this, though many questions remain. [10,19,20] [17]

This study aimed to determine if diet quality modifies the risk of air pollutant exposure on T2D incidence. Using the Hispanic Community Health Study/Study of Latinos, we investigated the relationship of these risk factors within the context of the Hispanic/Latino population, a population with high incidence of T2D.

Methods

Study Design and Population

The Hispanic Community Health Study/Study of Latinos (HCHS/SOL) is a multi-center cohort study focused on the Hispanic/Latino community. A total of 16,415 participants were recruited beginning in 2006. The goal of this study was to assess various factors that could be predictors of health within the context of this population.[21] HCHS/SOL established study centers around the US at four major cities; 1) Bronx, NY, 2) Chicago, IL, 3) Miami, FL, and 4) San Diego, CA. These locations were strategically selected to capture the diversity of heritages within the U.S. Hispanic/Latino population. Each of these study centers recruited at least 4,000 individuals who identify as Hispanic or Latino.

The HCHS/SOL cohort follow-up comes in two forms: in-person examinations with several years in between visits and annual phone calls. Baseline examination of the HCHS/SOL took place between 2008 and 2011. Participants aged from 18-74 years old were invited to participate and undergo examination. Baseline examination lasted approximately 7 hours conducted in either English or Spanish, dependent on participant preference, conducted by bilingual staff and used questionnaires. The exam began in the morning to best accommodate fasting blood draws. Relevant data collected at this visit included anthropometry (including height, weight, and both waist and hip circumferences), diabetes data (fasting glucose, blood draws, medication use), 24-hour dietary recall, and sociodemographics. Visit 2 took place between 2014 and 2017. Relevant data collection during this visit consisted of anthropometry, diabetes data, and sociodemographics. Average time between baseline and visit 2 was 6 years. Annual follow-up via phone calls collected general health updates as reported by the participant. Topics covered in the phone follow-up include hospitalizations, health screenings, medication use, changes in health symptoms, and up-to-date address and contact information. A wide variety of participant characteristics and health measures have been assessed repeatedly in this population, including sociodemographic characteristics, anthropometric measurements, laboratory measurements of blood and urine, and outcomes including cardiovascular diseases.

Diet Quality

Diet quality was measured using the Alternative Healthy Index-2010 (AHEI-2010). The AHEI-2010 is a ranking system used to score diet quality on eleven separate categories (whole fruits, vegetables, whole grains, sugar-sweetened beverages and fruit juice, nuts and legumes, fresh red meat, processed meat, fish, unsaturated fatty acid rich oils, alcohol, and sodium), scored to reflect the direction of association of each factor with chronic disease risk. Each of the

eleven factors is scaled from 1-10; these are summed to create an AHEI score which can range from 0-110. A score closer to the maximum 110 points indicates better adherence to the AHEI diet, and thus a higher diet quality. Validation of the AHEI-2010 scoring system has been conducted in a meta-analysis by Franziska et al., with 21% decreased risk of T2D for those who adhered to the AHEI compared to those who did not within the US population.[4] Diet quality assessed using the AHEI-2010 system also factors in caloric intake into the overall score.

AHEI-2010 scores in the HCHS/SOL study were calculated using two 24-hour dietary recalls. All dietary interviewers were required to be bilingual in written and spoken Spanish and English, have knowledge of different names of foods and preparations for each background represented in the HCHS/SOL study, and be certified in the Nutrition Data System for Research (NDSR) database or program. Interviewers used the multiple-pass approach during their interviews. This entails four passes through the data collection step using the NDSR system to improve collection of intake content, timing, and portion sizes. Additionally, interviewees were provided with a booklet to aid in the estimation of portion sizes during both interviews. A total of four passes were conducted guided by the NDSR system. Pass 1 is an initial outline of the participant's intake during the previous day, likely only getting foods and beverages with no timing. Pass 2 reviews data collected during pass 1 and notes major gaps in time. Pass 3 probes for further details of the foods and beverages collected in the previous two passes. Time, location, and names of foods are clarified during this pass with the aid of the NDSR's food search feature. Pass 4 is a review of the information collected, as well as inquiry into the major gaps in time noted during pass 2. This pass will address any flagged items the NDSR displayed, such as missing time window or amount eaten. The first dietary recall was administered in-person during the baseline visit. NDSR software contained portion size tools for food and beverages to assist in measurement recall. The combined time to finish all baseline dietary interviews was estimated to be about 45 minutes. The second dietary recall took place between 5 and 45 days after the initial dietary interview over a phone follow-up call. Participants were asked to schedule a time best suited for their schedule to facilitate a second interview. Both interviews followed a standardized script ensuring consistency and all information collected by the interviewer was kept confidential.

Air Pollution Modeling

*HCHS/*SOL participant exposure predictions were generated from validated fine-scale national spatio-temporal models of ambient $PM_{2.5}$ and NO_2.[15,22] Concentrations of $PM_{2.5}$ and NO_2 were predicted by a hierarchical model in a universal kriging framework which accounts for temporal and spatial trends in air pollution. The spatio-temporal models incorporates agency (Environmental Protection Agency Air Quality System and New York City Community Air Survey [NYCCAS]) and researcher-deployed air pollution monitoring data, geographic covariates, and spatial smoothing to characterize pollutant concentrations on a two-week time scale at point locations over the full follow-up period.[20,21] The hierarchical model uses long-term data, defined as having 18 years of 95% or better coverage and accounts for unbalanced data. Spatial variation in the model was accounted for by using geographic covariates such as proximity to major roadways, intersections, and bodies of water.[24]

Cross-validation for the NO_2 and $PM_{2.5}$ models used long-term averages and one of three techniques: 10-fold cross-validation leaving 10% of the total data out for home and NYCCAS distributed sites, leave-one-out cross-validation for AQS and NYCCAS reference sites, or 10-fold cross-validation with monitors of the same cluster left out together for snapshot sites.[24] Performance was evaluated using root mean-squared error (RMSE) and cross-validation R^2 (R^2_{CV}). Predictive accuracy of the models ranged from good ($R^2_{CV} > 0.6$) to excellent ($R^2_{CV} > 0.8$) for each pollutant in almost all monitoring areas. If a participant obtained a new address during the study period, we were able to account for the change in location. The primary exposures in this study were average $PM_{2.5}$ or NO_2 concentration over the follow-up period for each participant.

Diabetes Ascertainment

Diabetes status was ascertained at the in-person visits at baseline and visit 2 and during the yearly follow-up phone calls between the in-person visits. For the purpose of blood tests, participants were asked to fast for 12 hours prior to their in-person visits. Participants were first screened in order to take the oral-glucose tolerance test. Fasting blood samples were drawn via venipuncture. Individuals without a fasting plasma glucose greater than 150 mg/dL or a previously reported diagnosis of diabetes were invited to have a two-hour oral glucose tolerance test. Participants eligible for the oral-glucose tolerance test were given a glucose solution containing 75 grams of glucose.[25] Participants were instructed to consume the drink within 5 minutes for best results.

Two definitions of the outcome, primary and secondary, were used during analysis. The primary definition of diabetes used is based on the American Diabetes Association criteria,[26] as well as self-reported antihyperglycemic medication use. Study participants were considered to have diabetes if they had a fasting plasma glucose greater than or equal to 126 milligrams per deciliter, glycosylated hemoglobin (HbA1c) greater than or equal to 6.5%, post-oral glucose tolerance test with serum glucose greater than 200 milligrams per deciliter at two-hours, or use of any antihyperglycemic drugs. A secondary definition of diabetes added to this primary definition by including those with self-reported diabetes. This secondary definition was used in a sensitivity analysis. These definitions of diabetes status within the HCHS/SOL cohort did not distinguish type 1 and type 2 diabetes.

Covariates Assessed

Hispanic/Latino heritage and study site used a combined, validated variable. This combined variable was created to ensure sufficient sample size of at least 100 individuals in each category for adequate comparison. Groups with an insufficient n were pooled, combining into an “Other” category specific to a study site. Heritage was collected at baseline using a seven-level categorical variable. Participants were asked to self-identify as either Cuban, Dominican, Central American, South American, Mexican, Puerto Rican, or other. Study site was collected in accordance with the location of the HCHS/SOL center. Together, 17 heritage-site levels were identified: 1) Dominicans - Bronx, 2) Central American - Bronx, 3) Central American - Chicago, 4) Central American - Miami, 5) Cubans - Miami, 6) Mexicans - Bronx, 7) Mexicans - Chicago, 8) Mexicans - San Diego, 9) Puerto Ricans - Bronx, 10) Puerto Ricans - Chicago, 11)

South American - Bronx, 12) South American - Chicago, 13) South American - Miami, 14) Other - Bronx, 15) Other - Chicago, 16) Other - Miami, 17) Other - San Diego.

Several covariates, assessed at the baseline visit, were used in analysis, including age in years, sex, household income, education, waist-to-hip ratio, and family history of diabetes. Sex was collected at baseline as female or male. Household yearly income was categorized as: less than $10,000, $10,001-$20,000, $20,001-$40,000, $40,001-$75,000, and more than $75,000. Education was based on self-report into one of four categories: 1) No high school diploma or GED, 2) At most a high school diploma or GED, 3) High school (or GED) education, 4) University/college education. Waist to hip ratio is a continuous measure of participant adiposity calculated by dividing waist circumference by hip circumference. Family history of diabetes was ascertained by asking the participant if any immediate family member had diabetes.

Inclusion/Exclusion Criteria

The total population at baseline for the HCHS/SOL study was 16,415 individuals. We excluded individuals that had a positive diabetes status at baseline (n = 3,245) and participants with unreliable or missing dietary intakes from the study (e.g., impossible calorie intakes, less than two dietary recalls) (n = 149). Similarly, we excluded individuals with missing adiposity measures (n = 32). Those without education attainment (n = 26) and income data (n = 1,082) were excluded as well. Study participants needed at least one geocoded residential address for at least 95% of the follow-up period. Without this geocoded address, we could not generate reliable air pollution estimates, thus we excluded these participants from analysis. 835 participants were excluded due to missing air pollutant data. Our final study population was a total of n = 7,534.

Missing Data

Data within this dataset had a non-monotonic missingness pattern. Most variables had <1% missingness, with study site, age, and sex having 0% missing. AHEI-2010 score had a missingness proportion between 1 and 2%, being 1.2%. The variable with the highest proportion of missingness was baseline income, with 8.4% missingness. We used complete case analysis for this study.

Statistical Analysis

Descriptive analysis was conducted to assess the baseline characteristics and the study population at the second in-person visit. Some variables were collected only at baseline (sex, heritage, education, study center, AHEI-2010), while others were collected at baseline and visit 2 (age, waist-to-hip ratio, and diabetes status). Air pollution estimates were calculated as an average for the entire follow-up period, so it was assigned to the visit 2 column. To evaluate differences between baseline and visit 2, a third column was added to account for changes in characteristics due to follow-up time and attrition.

Poisson regression was used to estimate the association between average $PM_{2.5}$ and NO_2 concentrations during follow-up and incident diabetes at visit 2. The poisson regression

model used a log-link and participant's follow-up time as the offset to estimate the incident rate ratio (IRR) of diabetes per one-IQR increase in $PM_{2.5}$ or NO_2. Four sets of models were created: (1) to assess the effects of air pollution without an AHEI-2010 effect, (2) the effect of AHEI-2010 without an air pollution effect, (3) mutually adjusted effects of air pollutant and AHEI-2010, and (4) interaction of AHEI-2010 between air pollutant and diabetes status. Each set contained three models with varying levels of adjustment. The minimally adjusted model adjusted for age, income, sex, and education. The second model included adjusting for the heritage and study site cross-classification variable. The fully adjusted model added waist-to-hip ratio, a measure of adiposity considered a possible mediator of the overall relationship. Sensitivity analysis included expanding the definition of the outcome to include self-reported diabetes diagnosis. All models used sampling weights calculated through a multi-stage probability sampling design, which are routinely used in HCHS/SOL analysis to permit population-level conclusions about the recruitment areas. These weights account for census-tract level characteristics, and can also partially account for loss to follow-up from baseline to visit 2. All analyses were conducted using R version 4.3.2.

Results

In our sample of 7,534 study participants, we observed a total of 876 incident cases of diabetes. The age-adjusted incidence rate of diabetes in our sample was roughly 175 new cases per 10,000 person-years when standardized to the 2010 US census data. Table 1 compares the participants who returned for exam 2 to the overall HCHS/SOL cohort at baseline. Returning participants were an average of 1.4 years older and disproportionately female. Slightly higher differences in income and educational attainment within the returning population compared to the overall population are also of note. Baseline AHEI-2010 score was normally distributed in the population. Inclusion and exclusion criteria were applied later for complete case analysis.

Table 1: Baseline population characteristics of HCHS/SOL cohort and those returning for second visit.

	Mean (SD) or Percentage	
Variables	Baseline Population of HCHS/SOL (N = 16,415)	Baseline Characteristics of Returning Population (N = 11,623)
Female	59.9	63.2
Age, years	45.9 (13.9)	47.3 (13.2)
Hispanic/Latino Heritage		
Central American	10.6	10.4
Cuban	14.4	14.2
Dominican	9.0	8.8
Mexican	39.6	41.5
Puerto Rican	16.7	15.5
South American	6.6	6.9
More than one heritage	3.1	2.7

Education		
No High School Diploma or GED	38.0	37.6
At most a High school diploma or GED	25.6	25.0
High school (or GED) education	12.6	13.1
University/college education	23.8	24.2
Income		
Less than $10,000	15.7	15.4
$10,001-$20,000	32.6	32.3
$20,001-$40,000	33.9	34.4
$40,001-$75,000	13.5	13.7
More than $75,000	4.3	4.4
Waist-to-Hip Ratio	0.93 (0.07)	0.93 (0.07)
Study Center		
Bronx	25.1	22.8
Chicago	25.2	26.6
Miami	24.8	24.5
San Diego	24.9	26.1
Family History of Diabetes	44.7	46.1
Air Pollutant		
$PM_{2.5}$, $\mu g/m^3$	-	9.3 (1.3)
NO_2, ppb	-	14.3 (5.3)
Alternative Healthy Eating Index-2010 Score	49.0 (7.6)	49.5 (7.6)
Diabetes Prevalence	19.7	20.6

In the minimally adjusted model (see table 2), a 2.48 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 21% higher incidence of diabetes when adjusting for age, income, sex, education, and family history of diabetes (IRR: 1.21; 95% CI: 1.01, 1.44). This association was not observed in models which included an adjustment term for study center-heritage (IRR: 0.88; 95% CI: 0.54, 1.42). In the fully adjusted model, exposure to a 2.48 $\mu g/m^3$ increase in $PM_{2.5}$ was also not associated with an increased incidence of diabetes (IRR: 0.89; 95% CI: 0.54, 1.45) when adjusting for age, income, sex, education, family history of diabetes, study center-heritage variable, and adiposity.

A 5.97 ppb increase in NO_2 was associated with an 11% higher incidence of diabetes when adjusting for age, income, sex, education, and family history of diabetes (IRR: 1.11; 95% CI: 1.02, 1.22) (Table 3). As with $PM_{2.5}$, including the study center-heritage variable for NO_2 led

to no observation of increased diabetes risk (IRR: 0.94; 95% CI: 0.74, 1.26). The association was also not observed when additionally adjusting for waist-to-hip ratio (IRR: 0.99; 95% CI: 0.75, 1.30).

Table 2. Single Pollutant Model Exposure and Diabetes Risk from Baseline to Visit 2 Among Study Participants in the Hispanic Community Health Study/Study of Latinos (n = 7,534).

	Air Pollutant and Diabetes Risk			
	$PM_{2.5}$[d]		NO_2[e]	
Model	IRR	95% CI	IRR	95% CI
Model 1[a]	**1.21**	**1.01, 1.44**	**1.11**	**1.02, 1.22**
Model 2[b]	0.88	0.54, 1.42	0.96	0.74, 1.26
Model 3[c]	0.89	0.54, 1.45	0.99	0.75, 1.30

Abbreviations: IQR, Interquartile Range; IRR, Incidence Rate Ratio; CI, Confidence Interval; ppb, Parts per billion; $PM_{2.5}$, particulate matter 2.5 microns in diameter or less.

[a] Model adjusted for age, income, sex, education, and family history of diabetes.

[b] Model adjusted for age, income, sex, education, family history of diabetes, and study center-heritage cross-classification variable.

[c] Model adjusted for age, income, sex, education, family history of diabetes, study center-heritage cross-classification variable, and waist-to-hip ratio.

[d] $PM_{2.5}$ estimates scaled to IQR (2.48 $\mu g/m^3$). $PM_{2.5}$ models had n = 7,534 and 876 incident cases of diabetes.

[e] NO_2 estimates scaled to IQR (5.97 ppb). NO_2 models had n = 7,534 and 876 incident cases of diabetes.

We report the results for AHEI-2010 score and diabetes incidence using the same staged analysis as for air pollutants; for this dietary scale we used the standard deviation from our sample as our increment for assessing the association. When adjusting for age, income, sex, education, and family history of diabetes, we found a 7.6 point higher AHEI-2010 score was associated with a 7% lower incidence of diabetes (IRR: 0.93; 95% CI: 0.83, 1.04) (Table 3). Including the study center-heritage variable in the adjustment set, a 7.6 point higher AHEI-2010 score was associated with a 12% lower incidence of diabetes (IRR: 0.88; 95% CI: 0.75, 1.02). Results from the fully adjusted model indicate that a 7.6 point higher AHEI-2010 score was associated with a 7% lower incidence of diabetes adjusting for age, income, sex, education, family history of diabetes, study center-heritage variable, and adiposity (IRR: 0.93; 95% CI: 0.80, 1.09). None of these associations achieved statistical significance.

Table 3. AHEI-2010 Score and Diabetes Risk from Baseline to Visit 2 Among Study Participants in the Hispanic Community Health Study/Study of Latinos (n = 7,534).

	AHEI-2010 Score and Diabetes Risk	
AHEI-2010 Score	IRR (SD - 7.6 points)	95% CI
Model 1[a]	0.93	0.83, 1.04
Model 2[b]	0.88	0.75, 1.02
Model 3[c]	0.93	0.80, 1.09

Abbreviations: AHEI, Alternative Healthy Eating Index; SD, Standard Deviation; IRR, Incidence Rate Ratio; CI, Confidence Interval.

[a] Model adjusted for age, income, sex, education, and family history of diabetes.

[b] Model adjusted for age, income, sex, education, family history of diabetes, and study center-heritage cross-classification variable.

[c] Model adjusted for age, income, sex, education, family history of diabetes, study center-heritage cross-classification variable, and waist-to-hip ratio.

For our mutually adjusted models shown in Table 4, a 2.48 μg/m^3 increase in $PM_{2.5}$ was associated with a 25% higher incidence of diabetes (IRR: 1.25; 95% CI: 1.04, 1.49) when adjusting for age, income, sex, family history of diabetes, and AHEI-2010 score. A 7.6 point higher AHEI-2010 score was associated with a 10% lower incidence of diabetes (IRR: 0.90; 95% CI: 0.80, 1.01) when adjusting for age, income, sex, family history of diabetes, and $PM_{2.5}$. As with the pollutant-focused models, these associations with $PM_{2.5}$ were not observed in models which included the center-heritage variable without or with adjustment for waist-to-hip ratio (IRR: 0.87; 95% CI: 0.53, 1.41; and IRR: 0.88; 95% CI: 0.54, 1.45, respectively). The association between AHEI-2010 scores and incidence of diabetes were less affected by incorporation of these variables, demonstrating a reduction in risk with higher healthy eating score, but this association was not statistically significant.

In the comparable mutually adjusted models including both NO_2 and AHEI-2010, our minimally adjusted model estimates that an increase od 5.97 ppb of NO_2 was associated with an 11% increase in diabetes incidence (IRR: 1.11; 95% CI: 1.02, 1.22) when adjusting for age, income, sex, family history of diabetes, and AHEI-2010 score. A 7.6 point higher AHEI-2010 score was associated with a 7% lower incidence of diabetes (IRR: 0.93; 95% CI: 0.83, 1.04) when adjusting for age, income, sex, family history of diabetes, and NO_2. This association was not observed in models Including the study center-heritage variable without or with waist-to-hip ratio (Table 4).

Table 4. Associations between Pollutant and Diabetes Risk and AHEI-2010 Score and Diabetes Risk, with Mutual Adjustment, from Baseline to Visit 2 Among Study Participants in the Hispanic Community Health Study/Study of Latinos.

	Air Pollutant and Diabetes Risk	
	Pollutant	AHEI-2010[f]
$PM_{2.5}$[d]	IRR (95% CI)	

Model 1[a]	**1.25 (1.04, 1.49)**	0.90 (0.80, 1.01)
Model 2[b]	0.87 (0.53, 1.41)	0.87 (0.75, 1.02)
Model 3[c]	0.88 (0.54, 1.45)	0.93 (0.80, 1.09)
NO_2[e]		
Model 1[a]	**1.11 (1.02, 1.22)**	0.93 (0.83, 1.04)
Model 2[b]	0.97 (0.74, 1.27)	0.87 (0.75, 1.02)
Model 3[c]	1.00 (0.76, 1.31)	0.93 (0.80, 1.09)

Abbreviations: IQR, Interquartile Range; IRR, Incidence Rate Ratio; CI, Confidence Interval; ppb, Parts per billion; $PM_{2.5}$, particulate matter 2.5 microns in diameter or less; AHEI, Alternative Healthy Eating Index.

[a] Model adjusted for age, income, sex, family history of diabetes, and AHEI-2010 score.

[b] Model adjusted for age, income, sex, family history of diabetes, AHEI-2010 score, education, and study center-heritage cross-classification variable.

[c] Model adjusted for age, income, sex, family history of diabetes, AHEI-2010 score, education, study center-heritage cross-classification variable, and waist-to-hip ratio.

[d] $PM_{2.5}$ estimates scaled to IQR (2.48 μg/m^3). $PM_{2.5}$ models had n = 7,534 and 876 incident cases of diabetes.

[e] NO_2 estimates scaled to IQR (5.97 ppb). NO_2 models had n = 7,534 and 876 incident cases of diabetes.

[f] AHEI-2010 estimates scaled to SD (7.6 points).

Models with an interaction term are shown in Table 5. In all three models for $PM_{2.5}$ and NO_2, there was no evidence that AHEI-2010 score modified the association between ambient air pollutant and incidence of diabetes. In the $PM_{2.5}$ models, confidence intervals of the air pollutant and AHEI-2010 score are very wide. The NO_2 models have comparably narrower confidence intervals, but there is still no evidence of any association between these variables.

Table 5. Associations between Pollutant and Diabetes Risk and AHEI-2010 Score and Diabetes Risk, with Mutual Adjustment and Assessment of Interaction, from Baseline to Visit 2 Among Study Participants in the Hispanic Community Health Study/Study of Latinos.

	Air Pollutant and Diabetes Risk		
	Pollutant	AHEI-2010[f]	Interaction
$PM_{2.5}$[d]	IRR (95%CI)		Ratio
Model 1[a]	2.40 (0.59, 9.75)	1.35 (0.57, 3.22)	0.90 (0.72, 1.12)
Model 2[b]	1.56 (0.30, 8.19)	1.21 (0.48, 3.07)	0.92 (0.72, 1.17)
Model 3[c]	1.46 (0.29, 7.30)	1.23 (0.50, 3.03)	0.93 (0.73, 1.17)
NO_2[e]			
Model 1[a]	1.42 (0.70, 2.87)	1.02 (0.76, 1.38)	0.96 (0.86, 1.07)

Model 2[b]	1.10 (0.47, 2.59)	0.92 (0.64, 1.31)	0.98 (0.86, 1.11)
Model 3[c]	1.12 (0.48, 2.64)	0.97 (0.68,1.39)	0.98 (0.87, 1.11)

Abbreviations: IQR, Interquartile Range; IRR, Incidence Rate Ratio; CI, Confidence Interval; ppb, Parts per billion; $PM_{2.5}$, particulate matter 2.5 microns in diameter or less; AHEI, Alternative Healthy Eating Index.

[a] Model adjusted for age, income, sex, family history of diabetes, and AHEI-2010 score.

[b] Model adjusted for age, income, sex, family history of diabetes, AHEI-2010 score, education, and study center-heritage cross-classification variable.

[c] Model adjusted for age, income, sex, family history of diabetes, AHEI-2010 score, education, study center-heritage cross-classification variable, and waist-to-hip ratio.

[d] $PM_{2.5}$ estimates scaled to IQR (2.48 μg/m^3). PM2.5 models had n = 7,534 and 876 incident cases of diabetes.

[e] NO_2 estimates scaled to IQR (5.97 ppb). NO_2 models had n = 7,534 and 876 incident cases.

[f] AHEI-2010 estimates scaled to SD (7.6 points).

Tables 6-9 are the sensitivity analyses we conducted using the secondary definition of diabetes. The three model structure used in previous tables was maintained: model 1 adjusted for age, income, sex, education, and family history of diabetes, model 2 added the study center-heritage cross-classification variable, and the fully adjusted model 3 included waist-to-hip ratio. When compared to the primary definition models, there was not an observable difference in outcomes.

Discussion

We assessed the relationship between air pollution, health diet and T2DM risk in an urban Latino population, and did find convincing evidence that diet quality modifies the relationship between air pollution and incident diabetes. Rather, our results suggested a complex relationship between diet and air pollution exposure that is sensitive to choice of covariate selection, and that needs further investigation.

Both dietary patterns and long-term air pollution exposure can affect chronic inflammation and oxidative stress.[27,28] A diet high in calories from saturated fats, sugars, and overconsumption of meats has been linked to incidence of T2D. Ambient air pollutants have demonstrated higher incidence of T2D as well. Hence, our expectation was that a healthy diet would mitigate the adverse effects of air pollution on inflammation and oxidative stress and reduce the pollution-related impact on T2DM risk. Our findings did not support this expectation.

Other researchers have assessed the potential interaction between diet and air pollution on this outcome.[10,19,20] A noteworthy article investigating specific nutrients by Shin and Kim chose $PM_{2.5}$ and NO_2 as the air pollutants associated with incident diabetes.[17] Various antioxidant and antiinflammatory nutrients were chosen for being potential modifiers in the relationship between air pollutants and incident diabetes. Of these pollutants, only NO_2 showed evidence for a specific nutrient intake to be a modifier.[17]

Our minimally adjusted point estimates for both $PM_{2.5}$ (IRR: 1.21; 95% CI: 1.01, 1.44) and NO_2 (IRR: 1.11; 95% CI: 1.02, 1.22) were similar to estimates from a recent umbrella

review by Jiang et al. Estimates for studies that looked at the association between $PM_{2.5}$ and incidence of T2D ranged from 1.08 (95% CI: 1.04, 1.12) to 1.12 (95% CI: 1.07, 1.12).[29] NO2 pooled estimates were less consistent, ranging from 1.13 (95% CI: 1.04, 1.22) to 1.01 (0.99, 1.04).[29] We found no evidence of an association with incidence of diabetes (IRR: 0.93; 95% CI: 0.83, 1.04). Compared to meta-analysis results assessing different diet quality measures, AHEI was associated with a reduction in risk of incident diabetes (RR: 0.79; 95% CI: 0.69, 0.90), as well as the Dietary Approaches to Stop Hypertension (DASH) diet (RR: 0.81; 95% CI: 0.72, 0.92) and the Mediterranean diet (RR: 0.87; 95% CI: 0.82, 0.93).[4] Fewer studies looked at the interaction between pollution and diet on diabetes incidence so these results are difficult to compare. Importantly, we found dramatic attenuation of our findings when we adjusted for the heritage-study center variable, suggesting a potential confounding relationship which was possible to discern given the approach to cohort development in HCHS/SOL.

Strengths

This study leveraged the diversity within the Hispanic/Latino community often overlooked by conventional literature. Common practice in previous studies would dictate a clumping of the different Hispanic/Latino groups into one category. However, this population is a diverse group with prevalence of diabetes that differ by background.[30] The spatiotemporal air pollution models used state-of-the-art methods to generate fine-scale estimates of $PM_{2.5}$ and NO_2 for each study participant's residential address over the entire follow-up period. Ascertainment of diabetes at baseline and visit 2 using serial glucose tolerance tests is a reliable method of collecting diabetes status. Aside from the many confounders used in this study, the HCHS/SOL dataset contains a large number of potential confounders and residential addresses, allowing for robust analysis. Lastly, this study investigates a growing body of literature focused on bridging the gap between long-term environmental air pollutant exposure and an individual's diet quality.

Limitations

Air pollution estimates are based on models using residential addresses of participants. While we accurately estimate the outdoor concentrations at the precise home location, participants are unlikely to remain solely at these locations, which can result in a varying exposure of air pollutants that are not reflected in the models. The timing of our air pollution estimates are also of note. Accurately capturing the induction period of a chronic disease such as T2D may require a longer period of follow-up. Exposure data is limited to the study period, with an average follow-up of six years. Exposure to ambient air pollutants before study enrollment are unavailable because residential addresses prior to baseline visit were not collected. While we anticipate that the biologically relevant period of exposure and elicitation of inflammation and oxidative stress is during the observed period of follow-up, it is possible that earlier periods not assessed here could also be important in these relationships.

Dietary ascertainment data is collected with a 24-hour recall multiple-pass method. While commonly done, the extrapolation to long term diet quality is challenging.[31,32] Dietary data, when collected in a patient self-report manner, may be prone to recall bias with omission of specific food groups occurring more frequently than others.[32] Another major limitation specific to this dataset is dietary data using the 24-hour recall was only collected at baseline. An

individual's diet is very dynamic. Collection at a single point in time may not accurately reflect the changes within the individual's diet over the six year follow-up period we are proposing this initial measurement to represent.[4,30] Thus, misclassification of an individual's reported diet may be an issue due to the dynamic nature of dietary patterns over time.

We cannot precisely determine that the cases of incident diabetes observed all represent Type 2 DM. Given the age of the participants, however, the assumption that those who developed diabetes during follow-up did indeed develop T2D is considered safe. Development of Type 1 Diabetes past the age of 18 is considered rare and typically occurs during childhood.[33]

Implications

Our analysis added to the growing literature on chronic health impacts associated with air pollution exposure and diet quality. Additionally, this study draws further attention to the environmental and lifestyle factors that affect individuals within the HCHS/SOL cohort. While our results confirmed our prior finding that air pollution was modestly associated with diabetes incidence, we found no strong evidence that diet quality modified that effect. While our dietary measurement method and operationalization precludes interpreting this lack of evidence as a true lack of effect modification, our results suggest a different approach to measuring diet quality be taken, whether that be more granular or another established dietary pattern.

Next Steps

The HCHS/SOL continues to follow up participants, currently wrapping up data collection for visit 3. Our study considered including data from baseline up until visit 3. Due to a variety of reasons, we chose to focus only on baseline and visit 2. First is the uncertainty of when data would become available for analysis. Second, a different analytical approach, such as time-to-event analysis would have been necessary. At the time of analysis planning, HCHS/SOL study analysts had not yet published their recommended baseline to visit 3 analytical methods document. Lastly, the covariates we used were collected at baseline, with many of them being dynamic variables that can change over time. Additional time between visits 2 and 3 would likely render baseline variables no longer representative of participant exposure (i.e. baseline diet quality). Furthermore, due to data availability we were only able to include visits 1 and 2 in our analysis. Visit 3 data became available during analysis, but including it would require a different analysis procedure. We plan to include this new data in future analyses.

When implementing further studies, it may be worthwhile to explore other dietary patterns such as the DASH diet, which is also measured in the HCHS/SOL cohort. Articles commonly use more than one dietary pattern measure during their analysis. Conversely, it may also be useful to consider a more refined diet quality measure, such as selecting specific food groups that are most closely related to T2D instead of a holistic diet measure such as AHEI-2010. Another aspect of diet quality and Hispanic/Latino health that may be worth considering is how an individual's diet may change over the years as they immigrate to the US. Latino populations in the US are a heterogenous population with a variety of heritages, ancestry, and time in the US. Changes to a participant's dietary pattern to a western diet is a phenomenon observed *a posteriori* in the HCHS/SOL study with many HCHS/SOL investigators looking

further into the implications on one's health.[13,14] Changes in one's diet patterns can occur over many years, even spanning generations in immigrant populations. Changes observed in eating patterns of individuals may be dependent on the heritage group, with traditional diets replaced by the host country.[30] Satia[34] reports that years living in the US may be associated with relatively less healthy diets, possibly increasing the risk of diet-related chronic disease. Less healthy diets were specified to be more calorie dense, sometimes partnered with a reduction in physical activity.[3,34]

Conclusion

We observed associations between ambient air pollutant concentrations of $PM_{2.5}$ and NO_2 and diabetes incidence, without accounting for AHEI-2010 in the HCHS/SOL study, but these results were sensitive to adjustment for potential confounding by clinic site and heritage. We did not find convincing evidence for an interaction between healthy eating and pollution on risk of T2DM. There are many reasons which could explain our findings, including lack of characterization of other individual characteristics, residual confounding (e.g., by acculturation and dietary changes), quality or focus of the dietary assessment, limited duration of follow-up, or inadequate sample size. Additionally, future studies may consider design features to address these issues. Air pollution and dietary factors are two predictors of health at the individual and community level, each of which can be considered modifiable risk factors, and which require vastly different approaches to address. Should future work demonstrate an association and interaction between diet, ambient air pollutants, and incidence of diabetes, it could serve to recontextualize the steps taken to mitigate the development of chronic disease.

References

1. CDC. Type 2 Diabetes. Centers for Disease Control and Prevention. April 18, 2023. Accessed November 20, 2023. https://www.cdc.gov/diabetes/basics/type2.html

2. WHO. Diabetes. 2023. Accessed November 20, 2023. https://www.who.int/news-room/fact-sheets/detail/diabetes

3. Menke A, Casagrande S, Geiss L, Cowie CC. Prevalence of and Trends in Diabetes Among Adults in the United States, 1988-2012. *JAMA*. 2015;314(10):1021. doi:10.1001/jama.2015.10029

4. Franziska J, Janine K, Matthias B S. Dietary Patterns and Type 2 Diabetes: A Systematic Literature Review and Meta-Analysis of Prospective Studies. *J Nutr*. 2017;147(6):1174-1182. doi:10.3945/jn.116.242552

5. Ley SH, Pan A, Li Y, et al. Changes in Overall Diet Quality and Subsequent Type 2 Diabetes Risk: Three U.S. Prospective Cohorts. *Diabetes Care*. 2016;39(11):2011-2018. doi:10.2337/dc16-0574

6. Takahashi F, Hashimoto Y, Kondo Y, Hamaguchi M, Yamazaki M, Fukui M. 1382-P: Relationship between Diabetes Diet–Related Quality of Life and Dietary Fiber Intake among People with Type 2 Diabetes—A Cross-Sectional Study. *Diabetes*. 2023;72(Supplement_1):1382-P. doi:10.2337/db23-1382-P

7. Chiuve SE, Fung TT, Rimm EB, et al. Alternative Dietary Indices Both Strongly Predict Risk of Chronic Disease. *J Nutr*. 2012;142(6):1009-1018. doi:10.3945/jn.111.157222

8. Li Y, Xu L, Shan Z, Teng W, Han C. Association between air pollution and type 2 diabetes: an updated review of the literature. *Ther Adv Endocrinol Metab*. 2019;10:204201881989704. doi:10.1177/2042018819897046

9. Krämer U, Herder C, Sugiri D, et al. Traffic-Related Air Pollution and Incident Type 2 Diabetes: Results from the SALIA Cohort Study. *Environ Health Perspect*. 2010;118(9):1273-1279. doi:10.1289/ehp.0901689

10. Hansen AB, Ravnskjær L, Loft S, et al. Long-term exposure to fine particulate matter and incidence of diabetes in the Danish Nurse Cohort. *Environ Int*. 2016;91:243-250. doi:10.1016/j.envint.2016.02.036

11. Eze IC, Schaffner E, Fischer E, et al. Long-term air pollution exposure and diabetes in a population-based Swiss cohort. *Environ Int*. 2014;70:95-105. doi:10.1016/j.envint.2014.05.014

12. Kolb H, Martin S. Environmental/lifestyle factors in the pathogenesis and prevention of type 2 diabetes. *BMC Med*. 2017;15(1):131. doi:10.1186/s12916-017-0901-x

13. Brunekreef B, Holgate ST. Air pollution and health. *The Lancet*. 2002;360(9341):1233-1242. doi:10.1016/S0140-6736(02)11274-8

14. Weinmayr G, Hennig F, Fuks K, et al. Long-term exposure to fine particulate matter and incidence of type 2 diabetes mellitus in a cohort study: effects of total and traffic-specific air pollution. *Environ Health*. 2015;14(1):53. doi:10.1186/s12940-015-0031-x

15. Kirwa K, Szpiro AA, Sheppard L, et al. Fine-Scale Air Pollution Models for Epidemiologic Research: Insights From Approaches Developed in the Multi-ethnic Study of Atherosclerosis and Air Pollution (MESA Air). *Curr Environ Health Rep*. 2021;8(2):113-126. doi:10.1007/s40572-021-00310-y

16. Pope CA, Dockery DW. Health Effects of Fine Particulate Air Pollution: Lines that Connect. 2006;56.

17. Shin MK, Kim KN. Association between long-term air pollution exposure and development of diabetes among community-dwelling adults: Modification of the associations by dietary nutrients. *Environ Int*. 2023;174:107908. doi:10.1016/j.envint.2023.107908

18. Yang BY, Fan S, Thiering E, et al. Ambient air pollution and diabetes: A systematic review and meta-analysis. *Environ Res*. 2020;180:108817. doi:10.1016/j.envres.2019.108817

19. Han YY, Jerschow E, Forno E, et al. Dietary Patterns, Asthma, and Lung Function in the Hispanic Community Health Study/Study of Latinos. *Ann Am Thorac Soc*. 2020;17(3):293-301. doi:10.1513/AnnalsATS.201908-629OC

20. Chen C, Hayden KM, Kaufman JD, et al. Adherence to a MIND-Like Dietary Pattern, Long-Term Exposure to Fine Particulate Matter Air Pollution, and MRI-Based Measures of Brain Volume: The Women's Health Initiative Memory Study-MRI. *Environ Health Perspect*. 2021;129(12):127008. doi:10.1289/EHP8036

21. Sorlie PD, Avilés-Santa LM, Wassertheil-Smoller S, et al. Design and Implementation of the Hispanic Community Health Study/Study of Latinos. *Ann Epidemiol*. 2010;20(8):629-641. doi:10.1016/j.annepidem.2010.03.015

22. Wang M, Keller JP, Adar SD, et al. Development of long-term spatiotemporal models for ambient ozone in six metropolitan regions of the United States: The MESA Air study. *Atmos Environ*. 2015;123:79-87. doi:10.1016/j.atmosenv.2015.10.042

23. Kirwa K, Gassett AJ, Sack C, et al. Estimating ambient air pollutant concentrations outside and inside homes in the Subpopulations and Intermediate outcomes in COPD air pollution (SPIROMICS air) cohort. *Environ Res*. 2024;259:119512. doi:10.1016/j.envres.2024.119512

24. Keller JP, Olives C, Kim SY, et al. A Unified Spatiotemporal Modeling Approach for Predicting Concentrations of Multiple Air Pollutants in the Multi-Ethnic Study of Atherosclerosis and Air Pollution. *Environ Health Perspect*. 2015;123(4):301-309. doi:10.1289/ehp.1408145

25. Diaz KM, Goldsmith J, Greenlee H, et al. Prolonged, Uninterrupted Sedentary Behavior and Glycemic Biomarkers Among US Hispanic/Latino Adults: The HCHS/SOL (Hispanic Community Health Study/Study of Latinos). *Circulation*. 2017;136(15):1362-1373. doi:10.1161/CIRCULATIONAHA.116.026858

26. American Diabetes Association Professional Practice Committee, ElSayed NA, Aleppo G, et al. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes—2024. *Diabetes Care*. 2024;47(Supplement_1):S20-S42. doi:10.2337/dc24-S002

27. Gomes EC, Florida-James G. Lung Inflammation, Oxidative Stress and Air Pollution. In: Ong KC, ed. *Lung Inflammation*. InTech; 2014. doi:10.5772/58252

28. Barthelemy J, Sanchez K, Miller MR, Khreis H. New Opportunities to Mitigate the Burden of Disease Caused by Traffic Related Air Pollution: Antioxidant-Rich Diets and Supplements. *Int J Environ Res Public Health*. 2020;17(2):630. doi:10.3390/ijerph17020630

29. Jiang W, Zhou H, Xu G, Zhang M, Tung TH, Luo C. The association between air pollution and three types of diabetes: An umbrella review of systematic reviews and meta-analyses. *Ecotoxicol Environ Saf*. 2025;294:118080. doi:10.1016/j.ecoenv.2025.118080

30. Maldonado LE, Adair LS, Sotres-Alvarez D, et al. Dietary Patterns and Years Living in the United States by Hispanic/Latino Heritage in the Hispanic Community Health Study/Study of Latinos (HCHS/SOL). *J Nutr*. 2021;151(9):2749-2759. doi:10.1093/jn/nxab165

31. Chan V, Davies A, Wellard-Cole L, et al. Using Wearable Cameras to Assess Foods and Beverages Omitted in 24 Hour Dietary Recalls and a Text Entry Food Record App. *Nutrients*. 2021;13(6):1806. doi:10.3390/nu13061806

32. Whitton C, Ramos-García C, Kirkpatrick SI, et al. A Systematic Review Examining Contributors to Misestimation of Food and Beverage Intake Based on Short-Term Self-Report Dietary Assessment Instruments Administered to Adults. *Adv Nutr*. 2022;13(6):2620-2665. doi:10.1093/advances/nmac085

33. Dahlquist GG, Nyström L, Patterson CC, the Swedish Childhood Diabetes Study Group, the Diabetes Incidence in Sweden Study Group. Incidence of Type 1 Diabetes in Sweden Among Individuals Aged 0–34 Years, 1983–2007. *Diabetes Care*. 2011;34(8):1754-1759. doi:10.2337/dc11-0056

34. Satia JA. Dietary acculturation and the nutrition transition: an overviewThis is one of a selection of papers published in the CSCN–CSNS 2009 Conference, entitled Can we identify culture-specific healthful dietary patterns among diverse populations undergoing nutrition transition?This paper is being published without benefit of author's corrections. *Appl Physiol Nutr Metab*. 2010;35(2):219-223. doi:10.1139/H10-007

Appendix

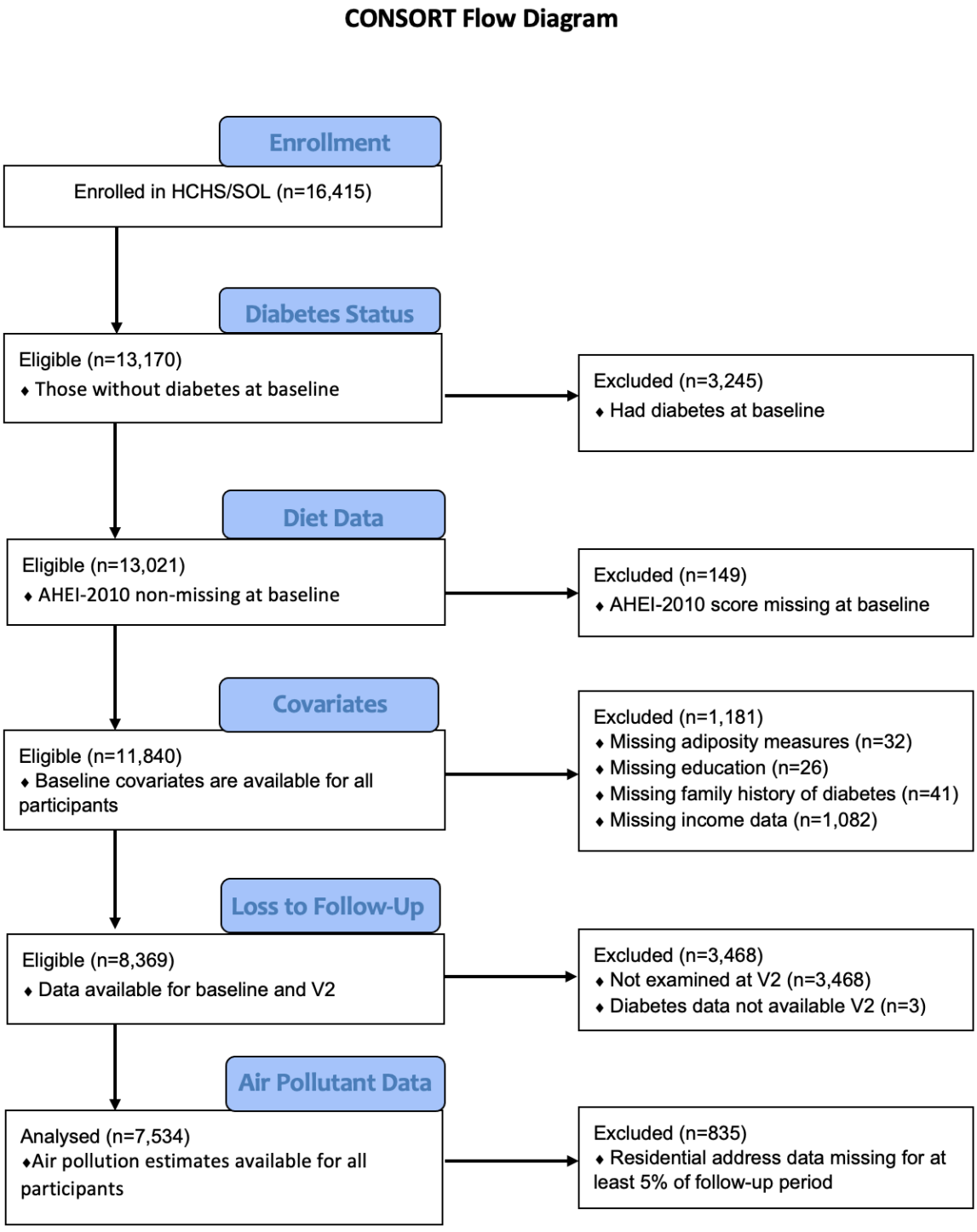
CONSORT Flow Diagram
Enrollment
Enrolled in HCHS/SOL (n=16,415)
Diabetes Status
Eligible (n=13,170)
♦ Those without diabetes at baseline
Excluded (n=3,245)
♦ Had diabetes at baseline
Diet Data
Eligible (n=13,021)
♦ AHEI-2010 non-missing at baseline
Excluded (n=149)
♦ AHEI-2010 score missing at baseline
Covariates
Eligible (n=11,840)
♦ Baseline covariates are available for all participants
Excluded (n=1,181)
♦ Missing adiposity measures (n=32)
♦ Missing education (n=26)
♦ Missing family history of diabetes (n=41)
♦ Missing income data (n=1,082)
Loss to Follow-Up
Eligible (n=8,369)
♦ Data available for baseline and V2
Excluded (n=3,468)
♦ Not examined at V2 (n=3,468)
♦ Diabetes data not available V2 (n=3)
Air Pollutant Data
Analysed (n=7,534)
♦Air pollution estimates available for all participants
Excluded (n=835)
♦ Residential address data missing for at least 5% of follow-up period

Table 6. Sensitivity Analysis: Single Pollutant Model Exposure and Diabetes Risk, Using Secondary Outcome Definition, from Baseline to Visit 2 Among Study Participants in the Hispanic Community Health Study/Study of Latinos.

	Air Pollutant and Diabetes Risk			
	$PM_{2.5}$[d]		NO_2[e]	
Model	IRR	95% CI	IRR	95% CI
Model 1[a]	**1.26**	**1.16, 1.36**	**1.03**	**1.01, 1.05**
Model 2[b]	1.22	0.96, 1.55	1.01	0.95, 1.06
Model 3[c]	1.23	0.98, 1.55	1.02	0.97, 1.07

Abbreviations: IQR, Interquartile Range; IRR, Incidence Rate Ratio; CI, Confidence Interval; ppb, Parts per billion; $PM_{2.5}$, particulate matter 2.5 microns in diameter or less.

[a] Model adjusted for age, income, sex, education, and family history of diabetes.

[b] Model adjusted for age, income, sex, education, family history of diabetes, and study center-heritage cross-classification variable.

[c] Model adjusted for age, income, sex, education, family history of diabetes, study center-heritage cross-classification variable, and waist-to-hip ratio.

[d] $PM_{2.5}$ estimates scaled to IQR (2.48 $\mu g/m^3$). $PM_{2.5}$ models had n = 7,540. And 876 incident cases of diabetes.

[e] NO_2 estimates scaled to IQR (5.97 ppb). NO_2 models had n = 7,540 and 876 incident cases of diabetes.

Table 7. Sensitivity Analysis: AHEI-2010 Score and Diabetes Risk, Using Secondary Outcome Definition, from Baseline to Visit 2 Among Study Participants in the Hispanic Community Health Study/Study of Latinos (n = 7,540).

	AHEI-2010 Score and Diabetes Risk	
AHEI-2010 Score	IRR (SD - 7.6 points)	95% CI
Model 1[a]	1.05	0.99, 1.10
Model 2[b]	0.98	0.92, 1.05
Model 3[c]	1.01	0.95, 1.08

Abbreviations: AHEI, Alternative Healthy Eating Index; SD, Standard Deviation; IRR, Incidence Rate Ratio; CI, Confidence Interval.

[a] Model adjusted for age, income, sex, education, and family history of diabetes,.

[b] Model adjusted for age, income, sex, education, family history of diabetes, and study center-heritage cross-classification variable.

[c] Model adjusted for age, income, sex, education, family history of diabetes, study center-heritage cross-classification variable, and waist-to-hip ratio.

Table 8. Sensitivity Analysis: Single Pollutant Model Exposure and Diabetes Risk, Using Secondary Outcome Definition, Adjusted for AHEI-2010 Score and Baseline Characteristics

from Baseline to Visit 2 Among Study Participants in the Hispanic Community Health Study/Study of Latinos.

	Air Pollutant and Diabetes Risk	
	Pollutant	AHEI-2010[f]
$PM_{2.5}$[d]	IRR (95% CI)	
Model 1[a]	**1.25 (1.15, 1.36)**	1.02 (0.97, 1.07)
Model 2[b]	1.22 (0.96, 1.54)	0.98 (0.92, 1.05)
Model 3[c]	1.23 (0.98, 1.55)	1.01 (0.95, 1.08)
NO_2[e]		
Model 1[a]	**1.03 (1.01, 1.05)**	1.05 (0.99, 1.10)
Model 2[b]	1.01 (0.96, 1.06)	0.98 (0.92, 1.05)
Model 3[c]	1.02 (0.97, 1.07)	1.01 (0.95, 1.08)

Abbreviations: IQR, Interquartile Range; IRR, Incidence Rate Ratio; CI, Confidence Interval; ppb, Parts per billion; $PM_{2.5}$, particulate matter 2.5 microns in diameter or less; AHEI, Alternative Healthy Eating Index.

[a] Model adjusted for age, income, sex, education, family history of diabetes, and AHEI-2010 score.

[b] Model adjusted for age, income, sex, education, family history of diabetes, AHEI-2010 score, and study center-heritage cross-classification variable.

[c] Model adjusted for age, income, sex,education, family history of diabetes, AHEI-2010 score, study center-heritage cross-classification variable, and waist-to-hip ratio.

[d] $PM_{2.5}$ estimates scaled to IQR (2.48 μg/m^3). $PM_{2.5}$ models had n = 7,540 and 876 incident cases of diabetes.

[e] NO_2 estimates scaled to IQR (5.97 ppb). NO2 models had n = 7,540 and 876 incident cases of diabetes.

[f] AHEI-2010 estimates scaled to SD (7.6 points).

Table 9. Sensitivity Analysis: Single Pollutant Model Exposure and Diabetes Risk, Using Secondary Outcome Definition, with AHEI-2010 Score Interaction from Baseline to Visit 2 Among Study Participants in the Hispanic Community Health Study/Study of Latinos.

	Air Pollutant and Diabetes Risk		
	Pollutant	AHEI-2010[f]	Interaction
$PM_{2.5}$[d]	IRR (95%CI)		Ratio
Model 1[a]	2.76 (1.48, 5.17)	1.65 (1.14, 2.38)	0.88 (0.81, 0.97)
Model 2[b]	3.36 (1.52, 7.41)	1.72 (1.15, 2.58)	0.86 (0.77, 0.96)
Model 3[c]	3.28 (1.50, 7.16)	1.74 (1.16, 2.60)	0.87 (0.78, 0.96)

NO_2[e]			
Model 1[a]	**1.19 (1.04, 1.35)**	**1.20 (1.05, 1.36)**	**0.98 (0.96, 0.99)**
Model 2[b]	1.13 (0.94, 1.36)	1.08 (0.92, 1.27)	0.98 (0.96, 1.01)
Model 3[c]	1.15 (0.96, 1.37)	1.12 (0.95,1.32)	0.98 (0.96, 1.01)

Abbreviations: IQR, Interquartile Range; IRR, Incidence Rate Ratio; CI, Confidence Interval; ppb, Parts per billion; $PM_{2.5}$, particulate matter 2.5 microns in diameter or less; AHEI, Alternative Healthy Eating Index.

[a] Model adjusted for age, income, sex, education, family history of diabetes, and AHEI-2010 score.

[b] Model adjusted for age, income, sex, education, family history of diabetes, AHEI-2010 score, and study center-heritage cross-classification variable.

[c] Model adjusted for age, income, sex, education, family history of diabetes, AHEI-2010 score, study center-heritage cross-classification variable, and waist-to-hip ratio.

[d] $PM_{2.5}$ estimates scaled to IQR (2.48 μg/m^3). $PM_{2.5}$ models had n = 7,540 and 876 incident cases of diabetes.

[e] NO_2 estimates scaled to IQR (5.97 ppb). NO_2 models had n = 7,540 and 876 incident cases of diabetes.

[f] AHEI-2010 estimates scaled to SD (7.6 points).