Joint Effects of Ethnic Enclave Residence and Air Pollution Exposure on Risk of Gestational Diabetes Mellitus Among Asian/Pacific Islander Women in the United States

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Abstract

Background: Asian/Pacific Islander (API) communities in the United States often reside in metropolitan areas with distinct social and environmental attributes. Residence in an ethnic enclave, a socially distinct area, is associated with lower gestational diabetes mellitus (GDM) risk, yet exposure to air pollution, which is often elevated in urban areas, is associated with increased GDM risk. We examined the joint effects of ethnic enclaves and air pollution to better understand GDM risk among API women, the group with the highest prevalence of GDM.

Methods: We examined 9,069 API births in the Consortium on Safe Labor (19 hospitals, 2002-2008). API ethnic enclaves were defined as areas ≥66^{th} percentile for percent API residents, dissimilarity (distribution of API and White residents), and isolation (degree that API individuals interact with another API individual). High levels of 14 volatile organic compounds (VOC) were defined as ≥75^{th} percentile. Four joint categories were created for each VOC: Low VOC/Enclave (reference group), Low VOC/No Enclave, High VOC/Enclave, High VOC/No Enclave. GDM was reported in medical records. Hierarchical logistic regression estimated odds ratios (OR) and 95% confidence intervals (95%CI) between joint exposures and GDM, adjusted for maternal factors and area-level poverty. Risk was estimated for 3-months preconception and first trimester exposures.

Results: Enclave residence was associated with lower GDM risk regardless of VOC exposure. Preconception benzene exposure was associated with increased risk when women resided outside enclaves [High VOC/No Enclave (OR:3.45, 95%CI:1.77,6.72)], and the effect was somewhat mitigated within enclaves, [High VOC/Enclave (OR:2.07, 95%:1.09,3.94)]. Risks were similar for 12 of 14 VOCs during preconception and 10 of 14 during the first trimester.

Conclusions: API residence in non-enclave areas is associated with higher GDM risk, regardless of VOC level. Ethnic enclave residence may mitigate effects of VOC exposure, perhaps due to lower stress levels. The potential benefit of ethnic enclaves warrants further study.

Background

In the United States (U.S.), the Asian/Pacific Islander (API) population increased 72% between 2000 and 2015, more than any other racial/ethnic group(1). Approximately 95% of the U.S. API population is concentrated in metropolitan areas (2), contributing to distinct social and environmental attributes of these areas (3–8). Despite the concentration of API communities in metropolitan areas, these populations are underrepresented in the environmental health literature, in part due to the 'model minority' myth.(6,9,10) The purpose of this study is to build on previous investigations of contextual health determinants (11,12) among pregnant API women to provide additional data to better understand the health implications of joint social and environmental exposures among U.S. API communities.

Ethnic enclaves are socially and geographically distinct areas with relatively high concentrations of residents of a similar racial/ethnic ancestry within a metropolitan area(3–5). Evidence suggests that residence in an ethnic enclave may contribute to better health outcomes among members of the prominent group in that area. Compared to API residents of non-enclave areas, API residents of ethnic enclaves live longer (13) and have lower cancer risk.(14). Evidence among pregnant API women is limited, yet initial findings suggest API women residing in ethnic enclaves seek prenatal care earlier (15) and smoke and use alcohol at lower rates (11,15,16). However, the potentially healthy effect of residence in an ethnic enclave may not be uniform, and may differ by ancestry of the API population, evidenced by studies of low birth weight, preterm birth and gestational diabetes mellitus (11,17,18) among API women residing in ethnic enclaves. The potentially healthy effect observed among residents of ethnic enclaves, compared to residents in non-enclave areas is hypothesized to be due to low exposure to discrimination(19,20) and stress(19), which are among the key determinants of health (21–23). The reduced exposure to discrimination and stress among ethnic enclave residents is likely due to residents' high levels of political representation and civic participation, as well as greater access to culturally-relevant goods and services that maintains the resident population's connection to their cultural identity. (3–5,15,24)

On average, U.S. API communities are exposed to higher levels of air pollution in comparison to all other racial/ethnic groups in the U.S.,(6–8,25) with pregnant API women nearly three times as likely to live in areas with high levels of air pollution compared to pregnant white women.(25) Exposure to air pollution may contribute to poor health outcomes through systemic inflammation and oxidative stress (26–31). In animal studies, increasing exposure to benzene, a volatile organic compound (VOC), has a linear association with oxidative stress, pancreatic β-cell dysfunction, and greater insulin resistance (32). High levels of oxidative stress have been linked with pancreatic β-cell dysfunction and insulin resistance among humans (30,31). Furthermore, API populations have a higher prevalence of genetic variations associated with pancreatic β-cell dysfunction and insulin resistance than other racial/ethnic groups (33,34). Thus, the potential interaction between air pollution exposure and social context merits further attention given the potential genetic susceptibility for adverse metabolic outcomes among API populations.

As high exposure to psychosocial stress is associated with immune system dysfunction(35), low exposure to stress among residents of ethnic enclaves suggests more normative immune function. As part of the normative immune response, cells exposed to an insulting agent release pro-inflammatory cytokines and become inflamed in order to isolate damage and protect healthy cells and tissue; as the insult is eliminated, anti-inflammatory cytokines are released and inflammation is contained, thus mitigating development of disease.(36) In contrast, air pollution exposure signals an inflammatory response(27,32,37–40), and among those with a compromised immune system, an excessive inflammatory response to air pollution may increase risk for metabolic disease.(41,42) Considering joint social and environmental exposures among API communities in the U.S. will provide new insights into health outcomes among these understudied populations.(43) To the best of our knowledge, joint exposure to residence in ethnic enclaves and air pollution has not yet been examined in pregnant women. Given evidence suggesting residence in ethnic enclaves may be less stressful residential contexts than other areas,(19,20), residents of ethnic enclaves more normative immune function may better mitigate the negative health consequences associated with exposure to air pollution compared to those residing elsewhere.

Gestational diabetes mellitus (GDM) presents a unique opportunity to examine joint exposures to ethnic enclaves and air pollution among U.S. API women. In the U.S., API women have the highest prevalence of GDM compared to other racial/ethnic groups.(44–52) GDM is associated with an increased risk of maternal, fetal and neonatal complications, including an increased risk of developing type 2 diabetes mellitus among mothers, and increased risk of obesity and diabetes among offspring.(53) In separate studies among API women within the Consortium on Safe Labor (CSL), we observed API women residing in ethnic enclaves had lower risk of GDM compared to API women residing in non-enclave areas(11) and that exposure to VOCs early in pregnancy was associated with increased risk of GDM among API women.(12) Thus, we hypothesized that pregnant API women residing in an ethnic enclaves were less susceptible to negative consequences of air pollution than pregnant API women residing elsewhere.

Methods

Data and participants

The Consortium on Safe Labor (CSL) was an electronic medical record-based retrospective cohort study from 2002–2008 which included 19 hospitals (8 university teaching hospitals, 9 community teaching hospitals, 2 community hospitals) in 15 Hospital Referral Regions (HRR), catchment areas for tertiary care hospitals.(54) Hospitals were selected based on availability of electronic medical records, and for representation of the 9 American College of Obstetricians and Gynecologists districts.(55) Data were extracted for deliveries ≥ 23 weeks gestation and include maternal sociodemographic characteristics; medical, reproductive and prenatal history; labor and delivery, and newborn data. A total of 228,438 deliveries were included in the CSL. We excluded multifetal pregnancies (n = 5,053; 2.21%), mothers with pre-existing diabetes (n = 3,309; 1.44%), and those with missing air pollution exposure information (n = 10; .004%). Including only Asian/Pacific Islander mothers resulted in an analytic sample of 9,069 births to 8,350 mothers. Institutional Review Boards at all sites approved the CSL, and data are anonymous.

Outcome variable

GDM was drawn from medical record data or in discharge summaries using ICD-9 code 648.8. During the CSL study period (2002–2008), the American Diabetes Associations recommended screening for GDM between 24–28 weeks gestation using the Carpenter and Coustan criteria.(56)

Ethnic enclave exposure

In the CSL, area of residence was estimated using the HRR in which the birth occurred. HRR is the only geographic unit of analysis available in the CSL.(57) HRRs are regional geographies (average miles^2: 13,065) comparable to Metropolitan Statistical Areas,(58) with large enough populations (average population size in thousands: 2,026) for observable residential sorting.(54, 58)

We aggregated sociodemographic data at the zip code tabulation area (ZCTA) level to provide estimates at the HRR level. As HRR are partially defined by ZCTA, we aggregated ZCTA data to the corresponding HRR using year-specific ZCTA to HRR crosswalk from the Dartmouth Atlas of Health Care. (54, 58) ZCTA data was accessed from the National Historical Geographic Information System for the 2000 decennial census, and the 2007–2011 5-year average of the American Community Survey (ACS).(59) We linked CSL data with year-specific sociodemographic data: births between 2002–2004 were linked with 2000 Census data, and births between 2005–2008 were linked with 2007–2011 ACS data.(11, 58)

We identified ethnic enclaves at the HRR level.(11) HRRs are centered on urban areas, where the majority of U.S. API populations reside,(2) yet the regional coverage of HRRs allows for inclusion of potential ethnic enclaves outside of urban centers.(60)

Described in Table 1, the distinct social and geographic attributes of an ethnic enclave are represented by API population density and racial/ethnic segregation, defined using three variables.(5, 11) First, API population density, is measured by the percent of API individuals residing in an HRR. Second, API-White dissimilarity index, is the differential distribution of API and White populations within a geographic area,(61, 62). Lastly, the API isolation index, is the probability that an API individual will interact with another API individual.(61, 62) API population density, API-white dissimilarity index, and API isolation index were calculated separately for Census data and ACS data.

Table 1

Area-level measures used to identify ethnic enclaves (also described in Williams et al., 2020)(11)

Measure	Formula	Description
API population density (social attribute)	$(A_T/P_T)*100$	Percentage of API residents within an HRR. Range 0–100; 100 suggests HRR consists of only API residents
Dissimilarity Index (geographic attribute)	$\frac{1}{2}\sum_{i=1}^{n}\left\|\frac{w_i}{W_T}-\frac{a_i}{A_T}\right\|$	Differential distribution of API and White populations within an HRR. Range 0–1; score of 1 suggests absolute geographic separation of API and White populations within HRR.
Isolation Index (geographic attribute)	$\sum_{i=1}^{n}\left(\frac{a_i}{A_T}\right)*\left(\frac{a_i}{P_T}\right)$	Probability that API residents of an HRR will interaction with another API individual. Range 0–1; score of 1 suggests an API resident in an HRR will only interact with other API residents.
Components	**Description**	
a_i	Number of API in the Zip code	
A_T	Number of API in the HRR	
n	Number of Zip codes	
P_T	Total population of the HRR	
w_i	Number of whites in the Zip code	
W_T	Number of white in the HRR	

We used population-based percentiles(4, 5, 11, 18) to identify tertiles (low, medium, high) for API population density, API-white dissimilarity, and API isolation. An HRR was considered an ethnic enclave if it was in the upper third of the distribution for all three variables: API population density, API-white dissimilarity, and API isolation.(11)

Air pollution exposure

The Air Quality and Reproductive Health study estimated air pollution exposure in the CSL using a modified version of the Community Multiscale Air Quality Model, a 3-dimensional, multipollutant air quality model used to predict ambient pollutant levels using National Emission Inventory emissions data and Weather Research Forecasting Model meteorological data.(57) Exposure was based on predicted hourly ambient pollutant concentrations within HRRs, fused with local air monitoring data to improve accuracy, and weighted to reflect population concentration and non-residential areas, as previously described.(57)

As GDM screening is recommended between 24–28 weeks gestation,(56) we averaged the predicted hourly ambient pollutant concentration across preconception (3 months preconception) and first trimester (through 13 weeks gestation) exposure windows. Ambient concentrations (parts per billion; ppb) were estimated for 14 VOCs: benzene, 1,3-butadiene, ethylbenzene, cyclohexane, methyl-tertiary-butyl ether, *N*-hexane, ethyl-methyl ketone, m-xylene, o-xylene, p-xylene, propene, sesquiterpene, styrene, and toluene for each exposure window. Exposure to ≥ 75th percentile in ppb was considered high exposure.

Joint exposure categories

Using the categorical ethnic enclave (yes/no) variable, and the categorical VOC (high/low) variable, we created joint exposure categories: Low VOC/Enclave (reference), Low VOC/No Enclave, High VOC/Enclave, High VOC/No Enclave. The

joint exposure variables were created for each of the 14 VOC in both the preconception and first trimester exposure windows.

Covariates

Individual-level covariates included maternal age, marital status (married, single, other), health insurance (public, private, other), pre-pregnancy body mass index (BMI, < 18.5, 18.5-<224.9, 25-<29.9, ≥ 30), season of conception (winter, spring, summer, fall) and parity (nulliparous or multiparous). As income is not available in the CSL, health insurance(63) and marital status(64) were used as proxies for socioeconomic status. BMI was imputed using multiple imputations (10 iterations) due to a high degree of missingness.

Area-level poverty (continuous proportion of residents in the HRR living below federal poverty thresholds), hospital type (university teaching hospital, community teaching hospital, and community non-teaching hospital) were included as HRR-level covariates. Covariates included in analysis were informed by previous studies.(11, 12)

Statistical analysis

Prevalence of GDM was reported for ethnic enclave residence and maternal characteristics, and by joint enclave-VOC exposure.

Hierarchical logistic regression models were used to estimate the odds ratio (OR) and 95% confidence intervals for the association between joint VOC/Ethnic Enclave exposure and GDM, with robust standard errors to account for repeat births to the same mother while adjusting for mothers nested within HRR (n = 731, 7.9% of births). Low VOC/Enclave exposure category served as reference group as we anticipated this is the lowest risk category. Separate models were run for each of the 14 VOCs for the preconception and first trimester exposure windows, using PROC GLIMMIX (SAS 9.4) (65). Benjamini-Hochberg false discovery rate adjustment procedure was used to account for multiple testing (66) (false discovery rate = 10%). Analyses were performed using PROC MULTTEST (SAS 9.4) (65).

Results

Of the 9069 pregnancies among API women in the CSL, there were 899 (9.9%) cases of GDM. Table 2 includes distribution of GDM by ethnic enclave residence, maternal characteristics, and area-level covariates. There were 1891 (20.8%) API women within ethnic enclaves, and 7178 (79.2%) API women in non-enclave areas. The prevalence of GDM was significantly lower among women in ethnic enclaves (7.5%) compared to women in non-enclave areas (10.5%). GDM was more prevalent as BMI and age increased, as well as among multiparous women. GDM was more prevalent among women with private (10.6%) versus public (9.7%), self pay (9.3%) or other (6.5%) insurance coverage. GDM prevalence differed by season of conception, with warmer months having lower prevalence of GDM compared to colder months. Of note, GDM prevalence did not greatly differ by area-level poverty.

Table 2

Frequency (and percent) of gestational diabetes mellitus status by ethnic enclave residence and maternal characteristics among Asian/Pacific Islander women in the Consortium on Safe Labor among Asian/Pacific Islander women (n = 9069)

	Gestational Diabetes Mellitus		p-values[a]
	Yes (n = 899)	No (n = 8170)	
Ethnic enclave			
Yes (1891)	142 (7.5)	1749 (92.5)	*p < .01*
No (7178)	757 (10.5)	6421 (89.5)	
Maternal Age			
< 20 years (168)	4 (2.34)	164 (97.4)	*p < .01*
20–24 years (1289)	74 (5.7)	1215 (94.3)	
25–29 years (2797)	226 (8.1)	2571 (91.9)	
30–34 years (2958)	318 (10.8)	2640 (89.2)	
35 + years (1851)	277 (15.0)	1574 (85.0)	
Unknown/Missing (6)	0 (0.0)	6 (100.0)	
Body Mass Index			
≥30 (425)	75 (17.7)	350 (82.3)	*p < .01*
25-29.9 (744)	111 (14.9)	633 (85.1)	
18.5–24.9 (3466)	282 (8.1)	3184 (91.9)	
11.2-18.49 (621)	31 (5.0)	590 (95.0)	
Unknown (3813)	400 (10.5)	3413 (89.5)	
Insurance Type			
Private (6374)	677 (10.6)	5697 (89.4)	*p < .01*
Public (1280)	124 (9.7)	1156 (90.3)	
Self Pay (193)	18 (9.3)	175 (90.7)	
Other (1222)	80 (6.5)	1142 (93.5)	

a. P-values obtain using generalized estimating equations to account for women who had more than one pregnancy in the study

Distribution of GDM by joint VOC/Enclave exposure categories is included in Table 3. For preconception VOC exposure, prevalence of GDM was lowest in Low VOC/Enclave areas for 7 of 14 VOCs, as anticipated, but was lowest in 6 of 14 High VOC/Enclave areas. For preconception exposure to sesquiterpene, Low VOC/Enclave areas and High VOC/Enclave areas, had the same GDM prevalence (7.5%). Prevalence of GDM was similar across categories of first trimester VOC exposure. For both preconception and first trimester exposures, non-enclave areas had higher GDM prevalence than enclave areas, regardless of VOC exposure levels.

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Insert Table 3 here

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	Gestational Diabetes Mellitus		p-values[a]
Marital Status			
Married (7642)	800 (10.5)	6842 (89.5)	*p < .01*
Single (1241)	78 (6.3)	1163 (93.7)	
Divorced (186)	21 (11.3)	165 (88.7)	
Parity			
0 (4433)	395 (8.9)	4038 (91.1)	*p < .01*
≥ 1 (4636)	504 (10.9)	4132 (89.1)	
Hospital Type			
University Affiliated (3716)	329 (8.9)	3387 (91.1)	*p < .01*
Community: Teaching (4948)	541 (10.9)	4407 (89.1)	
Community: Non-teaching (405)	29 (7.2)	376 (92.8)	
Season of Conception			
March-May (2140)	203 (9.5)	1937 (90.5)	*p = .05*
June-August (2363)	208 (8.8)	2155 (91.2)	
September-November (2437)	250 (10.3)	2187 (89.7)	
December-February (2129)	238 (11.2)	1891 (88.8)	
Area-Level Poverty			
≥ 15.9% (3323)	348 (10.5)	2975 (89.5)	*p = .17*
< 15.9% (5746)	551 (9.6)	5195 (90.4)	

a. P-values obtain using generalized estimating equations to account for women who had more than one pregnancy in the study

Distribution of GDM by joint VOC/Enclave exposure categories is included in Table 3. For preconception VOC exposure, prevalence of GDM was lowest in Low VOC/Enclave areas for 7 of 14 VOCs, as anticipated, but was lowest in 6 of 14 High VOC/Enclave areas. For preconception exposure to sesquiterpene, Low VOC/Enclave areas and High VOC/Enclave areas, had the same GDM prevalence (7.5%). Prevalence of GDM was similar across categories of first trimester VOC exposure. For both preconception and first trimester exposures, non-enclave areas had higher GDM prevalence than enclave areas, regardless of VOC exposure levels.

Insert Table 3 here

Table 3
Frequency (and percent) of gestational diabetes mellitus status by joint preconception VOC-Enclave category among Asian/Pacific Islander women in the Consortium on Safe Labor (2002–2008)

			Preconception				First Trimester			
VOC (High = ≥ 75th percentile)		Enclave	n (N = 9069)	GDM (N = 899)	No GDM (n = 8170)	p value[a]	n (N = 9069)	GDM (N = 899)	No GDM (n = 8170)	p value[a]
Benzene	Low	Yes	242	13 (5.3)	229 (94.6)	*p < .01*	245	15 (6.1)	230 (93.9)	*p < .01*
		No	4115	415 (10.0)	3700 (90.0)		4179	420 (10.0)	3759 (90.0)	
	High	Yes	1649	129 (7.8)	1520 (92.2)		1646	127 (7.7)	1519 (92.3)	
		No	3063	342 (11.1)	2721 (88.9)		2999	337 (11.2)	2662 (88.8)	
Ethylbenzene	Low	Yes	245	15 (6.1)	230 (93.9)	*p < .01*	245	15 (6.1)	230 (93.9)	*p < .01*
		No	3916	406 (10.3)	3510 (89.7)		3943	412 (10.4)	3531 (89.6)	
	High	Yes	1646	127 (7.7)	1519 (92.3)		1646	127 (7.7)	1519 (92.3)	
		No	3262	351 (10.7)	2911 (89.3)		3235	345 (10.6)	2890 (89.4)	
MTB Ether	Low	Yes	738	63 (8.5)	675 (91.5)	*p < .01*	596	50 (8.4)	546 (91.6)	*p < .01*
		No	4578	437 (9.5)	4141 (90.5)		4436	419 (9.4)	4017 (90.6)	
	High	Yes	1153	79 (6.9)	1074 (93.1)		1295	92 (7.1)	1203 (92.9)	
		No	2600	320 (12.3)	2280 (87.7)		2742	338 (12.3)	2404 (87.7)	
N-hexane	Low	Yes	535	44 (8.22)	491 (91.8)	*p < .01*	410	32 (7.8)	378 (92.2)	*p < .01*
		No	3834	373 (9.7)	3461 (90.3)		3984	388 (9.7)	3596 (90.3)	
	High	Yes	1356	98 (7.2)	1258 (92.8)		1481	110 (7.4)	1371 (92.6)	
		No	3344	384 (11.5)	2960 (88.5)		3194	369 (11.5)	2825 (88.5)	

a. P-values obtain using generalized estimating equations to account for women who had more than one pregnancy in the study

			Preconception				First Trimester			
EMK	Low	Yes	739	58 (7.8)	681 (92.2)	*p < .01*	627	54 (8.6)	573 (91.4)	*p < .01*
		No	4827	490 (10.1)	4337 (89.9)		4615	460 (9.9)	4155 (90.1)	
	High	Yes	1152	84 (7.3)	1068 (92.7)		1264	88 (6.9)	1176 (93.1)	
		No	2351	267 (11.3)	2084 (88.7)		2563	297 (11.6)	2266 (88.4)	
m-xylene	Low	Yes	245	15 (6.1)	230 (93.9)	*p < .01*	245	15(6.1)	230 (93.9)	*p < .01*
		No	3909	408 (10.4)	3501 (89.6)		3926	410 (10.4)	3516 (89.6)	
	High	Yes	1646	127 (7.7)	1519 (92.3)		1646	127 (7.7)	1519 (92.3)	
		No	3269	349 (10.7)	2920 (89.3)		3252	347 (10.7)	2905 (89.3)	
o-xylene	Low	Yes	304	18 (5.9)	286 (94.1)	*p < .01*	246	15(6.1)	231 (93.9)	*p < .01*
		No	3897	405 (10.4)	3492 (89.6)		3936	410 (10.4)	3526 (89.6)	
	High	Yes	1587	124 (7.8)	1463 (92.2)		1645	127 (7.7)	1518 (92.3)	
		No	3281	352 (10.7)	2929 (89.3)		3242	347 (10.7)	2895 (89.3)	
p-xylene	Low	Yes	438	31 (7.1)	407 (92.9)	*p < .01*	338	24 (7.1)	314 (92.9)	*p < .01*
		No	3868	399 (10.3)	3469 (89.7)		3900	406 (10.4)	3494 (89.6)	
	High	Yes	1453	111 (7.6)	1342 (92.4)		1553	118 (7.6)	1435 (92.4)	
		No	3310	358 (10.8)	2952 (89.2)		3278	351 (10.7)	2927 (89.3)	
Propene	Low	Yes	827	67 (8.1)	760 (91.9)	*p < .01*	945	74 (7.8)	871 (92.2)	*p < .01*
		No	4728	449 (9.5)	4279 (90.5)		4471	420 (9.4)	4051 (90.6)	
	High	Yes	1064	75 (7.1)	989 (92.9)		946	68 (7.1)	878 (92.9)	
		No	2450	308 (12.6)	2142 (87.4)		2707	337 (12.5)	2370 (87.5)	
Sesquiterpene	Low	Yes	818	62 (7.5)	756 (92.5)	*p < .01*	741	62 (8.4)	679 (91.6)	*p < .01*

a. P-values obtain using generalized estimating equations to account for women who had more than one pregnancy in the study

			Preconception				First Trimester			
		No	3753	355 (9.5)	3398 (90.5)		3841	360 (9.4)	3481 (90.6)	
	High	Yes	1073	80 (7.5)	993 (92.5)		1150	80 (6.9)	1070 (93.1)	
		No	3425	402 (11.7)	3023 (88.2)		3337	397 (11.9)	2940 (88.1)	
Toluene	Low	Yes	334	19 (5.7)	315 (94.3)	*p < .01*	245	15 (6.1)	230 (93.9)	*p < .01*
		No	3891	407 (10.5)	3484 (89.5)		3934	411 (10.5)	3523 (89.5)	
	High	Yes	1557	123 (7.9)	1434 (92.1)		1646	127 (7.7)	1519 (92.3)	
		No	3287	350 (10.6)	2937 (89.4)		3244	346 (10.7)	2898 (89.3)	
Styrene	Low	Yes	736	64 (8.7)	672(91.3)	*p < .01*	619	52 (8.4)	567 (91.6)	*p < .01*
		No	6268	657 (10.5)	5611 (89.5)		6266	661 (10.6)	5605 (89.4)	
	High	Yes	1155	78 (6.7)	1077 (93.3)		1272	90 (7.1)	1182 (92.9)	
		No	910	100 (11.0)	810 (89.0)		912	96 (10.5)	816 (89.5)	
1,3 butadiene	Low	Yes	732	64 (8.7)	668 (91.3)	*p < .01*	595	51 (8.6)	544 (91.4)	*p < .01*
		No	6197	642 (10.4)	5555 (89.6)		6354	673 (10.6)	5681 (89.4)	
	High	Yes	1159	78 (6.7)	1081 (92.3)		1296	91 (7.0)	1205 (93.0)	
		No	981	115 (11.7)	866 (88.3)		824	84 (10.1)	740 (89.9)	
Cyclohexane	Low	Yes	1022	66(6.5)	956(93.5)	*p < .01*	1016	74 (7.3)	942 (92.7)	*p < .01*
		No	3747	391 (10.4)	3356 (89.6)		3794	400 (10.5)	3394 (89.5)	
	High	Yes	869	76 (8.7)	793 (91.3)		875	68 (7.8)	807 (92.3)	
		No	3431	366 (10.7)	3065 (89.3)		3384	357 (10.6)	3027 (89.5)	

a. P-values obtain using generalized estimating equations to account for women who had more than one pregnancy in the study

Hierarchical regression results for the association between VOC/Enclave joint exposure and GDM are reported in Table 4. Compared to Low VOC/Enclave areas, non-enclave areas were generally associated with higher risk of GDM, regardless of VOC exposure levels. For example, preconception benzene exposure was associated with elevated risk for High VOC/No

Enclave (OR:3.45, 95%CI:1.77, 6.72) and for Low VOC/No Enclave (OR:2.85, 95%CI:1.57, 5.17), while the risk for High VOC/Enclave (OR:2.07, 95%:1.09, 3.94) was elevated but somewhat mitigated. There was a similar pattern for 12 of 14 VOC during preconception and 10 of 14 during the first trimester. For example, for propene exposure, risks were similar for both preconception High VOC/No Enclave (OR:1.99, 95%CI: 1.46, 2.72) and first trimester High VOC/No Enclave (OR:1.96, 95%CI: 1.44, 2.67).

Table 4
Joint associations between exposure to ambient volatile organic compounds, ethnic enclaves, and gestational diabetes mellitus among Asian/Pacific Islander women in the Consortium on Safe Labor (2002–2008)

			Preconception		First Trimester	
VOC (High = ≥ 75th percentile)		Enclave	n (N = 9069)	Odds Ratio (95% CI)	n (N = 9069)	Odds Ratio (95% CI)
Benzene	Low	Yes	242	Ref	245	Ref.
		No	4115	2.85 (1.57, 5.17)*	4179	2.38 (1.36, 4.16)*
	High	Yes	1649	2.07 (1.09, 3.94)*	1646	1.65 (0.90, 3.03)
		No	3063	3.45 (1.77, 6.72)*	2999	2.70 (1.45, 5.03)*
Ethylbenzene	Low	Yes	245	Ref.	245	Ref.
		No	3916	2.43 (1.37, 4.32)*	3943	2.30 (1.29, 4.09)*
	High	Yes	1646	1.70 (0.90, 3.22)	1646	1.56 (0.82, 2.95)
		No	3262	2.76 (1.32, 5.75)*	3235	2.29 (1.10, 4.77)*
MTB Ether	Low	Yes	738	Ref.	596	Ref.
		No	4578	1.38 (1.02, 1.87)*	4436	1.40 (1.01, 1.95)
	High	Yes	1153	0.87 (0.58, 1.31)	1295	0.90 (0.60, 1.36)
		No	2600	1.84 (1.34, 2.52)*	2742	1.88 (1.33, 2.66)*
N-hexane	Low	Yes	535	Ref.	410	Ref.
		No	3834	1.56 (1.11, 2.20)*	3984	1.72 (1.16, 2.55)*
	High	Yes	1356	1.03 (0.68, 1.57)	1481	1.16 (0.74, 1.83)
		No	3344	2.09 (1.39, 3.15)*	3194	2.25 (1.43, 3.53)*
EMK	Low	Yes	739	Ref.	627	Ref.
		No	4827	1.62 (1.19, 2.20)*	4615	1.43 (1.04, 1.96)*
	High	Yes	1152	1.09 (0.72, 1.63)	1264	0.85 (0.57, 1.28)
		No	2351	1.84 (1.32, 2.58)*	2563	1.67 (1.18, 2.36)*
m-xylene	Low	Yes	245	Ref.	245	Ref.
		No	3909	2.33 (1.31, 4.15)*	3926	2.30 (1.29, 4.08)*
	High	Yes	1646	1.59 (0.84, 3.01)	1646	1.55 (0.82, 2.94)
		No	3269	2.40 (1.15, 5.00)*	3252	2.27 (1.08, 4.76)*
o-xylene	Low	Yes	304	Ref.	246	Ref.
		No	3897	2.37 (1.42, 3.98)*	3936	2.35 (1.32, 4.17)*
	High	Yes	1587	1.66 (0.94, 2.94)	1645	1.60 (0.85, 3.04)

Analytic sample restricted to Asian/Pacific Islander women without diagnosed preconception diabetes. Hierarchical logistic regression, women nested within hospital referral region. Models adjusted for maternal age, preconception BMI, parity, insurance status, hospital, marital status, area-level poverty, season of birth. *statistically significant after Benjamini-Hochberg procedure (false discovery rate = 10%).

			Preconception		First Trimester	
		No	3281	2.52 (1.30, 4.87)*	3242	2.42 (1.15, 5.09)*
p-xylene	Low	Yes	438	Ref.	338	Ref.
		No	3868	1.87 (1.25, 2.78)*	3900	1.93 (1.23, 3.03)*
	High	Yes	1453	1.27 (0.80, 2.03)	1553	1.29 (0.77, 2.16)
		No	3310	2.02 (1.20, 3.40)*	3278	1.93 (1.06, 3.53)*
Propene	Low	Yes	827	Ref.	945	Ref.
		No	4728	1.44 (1.07, 1.93)*	4471	1.45 (1.09, 1.93)*
	High	Yes	1064	0.93 (0.64, 1.36)	946	0.94 (0.66, 1.34)
		No	2450	1.99 (1.46, 2.72)*	2707	1.96 (1.44, 2.67)*
Sesquiterpene	Low	Yes	818	Ref.	741	Ref.
		No	3753	1.55 (1.15, 2.10)*	3841	1.38 (1.02, 1.87)*
	High	Yes	1073	1.13 (0.77, 1.65)	1150	0.87 (0.59, 1.28)
		No	3425	2.23 (1.59, 3.14)*	3337	1.91 (1.36, 2.69)*
Toluene	Low	Yes	334	Ref.	245	Ref.
		No	3891	2.39 (1.45, 3.95)*	3934	2.31 (1.30, 4.11)*
	High	Yes	1557	1.68 (0.96, 2.91)	1646	1.57 (0.83, 2.97)
		No	3287	2.38 (1.25, 4.52)*	3244	2.31 (1.09, 4.89)*
Styrene	Low	Yes	736	Ref.	619	Ref.
		No	6268	1.41 (1.04, 1.93)*	6266	1.47 (1.05, 2.06)*
	High	Yes	1155	0.84 (0.56, 1.26)	1272	0.89 (0.59, 1.34)
		No	910	1.59 (1.13, 2.24)*	912	1.56 (1.08, 2.25)*
1,3 butadiene	Low	Yes	732	Ref.	595	Ref.
		No	6197	1.39 (1.02, 1.88)*	6354	1.48 (1.06, 2.06)*
	High	Yes	1159	0.83 (0.55, 1.25)	1296	0.86 (0.57, 1.29)
		No	981	1.67 (1.19, 2.33)*	824	1.46 (1.01, 2.13)*
Cyclohexane	Low	Yes	1022	Ref.	1016	Ref.
		No	3747	1.86 (1.37, 2.53)*	3794	1.70 (1.26, 2.28)*

Analytic sample restricted to Asian/Pacific Islander women without diagnosed preconception diabetes. Hierarchical logistic regression, women nested within hospital referral region. Models adjusted for maternal age, preconception BMI, parity, insurance status, hospital, marital status, area-level poverty, season of birth. *statistically significant after Benjamini-Hochberg procedure (false discovery rate = 10%).

			Preconception		First Trimester	
	High	Yes	869	1.31 (0.92, 1.87)	875	1.08 (0.76, 1.54)
		No	3431	1.73 (1.15, 2.59)*	3384	1.46 (0.98, 2.18)

Analytic sample restricted to Asian/Pacific Islander women without diagnosed preconception diabetes. Hierarchical logistic regression, women nested within hospital referral region. Models adjusted for maternal age, preconception BMI, parity, insurance status, hospital, marital status, area-level poverty, season of birth. *statistically significant after Benjamini-Hochberg procedure (false discovery rate = 10%).

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Insert Table 4 here

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Discussion

In this first investigation of the association between joint exposure to air pollution and residence in an ethnic enclave and GDM risk, we found evidence that residence within an ethnic enclave may mitigate negative consequences of environmental exposures, particularly VOC. In line with evidence of an association between preconception and first trimester exposure to air pollution and increased risk of GDM(12, 28, 67–69) as well as evidence of lower risk of GDM among women residing within ethnic enclaves(11, 17, 18) we found evidence that residence in enclaves is associated with lower GDM risk, regardless of VOC level.

The observations suggest chronic exposure to residence outside of ethnic enclaves and VOCs are associated with increased GDM risk for API mothers, as risks appear consistent across preconception and first trimester exposure windows. Previously among women in the CSL, we have observed consistent increases in GDM risk across preconception and first trimester exposure windows for criteria air pollutants such as nitrogen oxides and sulfur dioxide(28), as well as VOCs.(12) Similar observations of chronic exposure to criteria air pollutants and GDM were observed among women in Denmark, Sweden, and Taiwan.(67–69) Given that air pollution and ethnic enclave exposures are likely chronic, the development of GDM is likely not due to an acute exposure in pregnancy.

As ethnic enclave residence appears to mitigate the negative consequences of VOC exposure, these observations suggest immunologic response to air pollution may be an important factor. The normative immunologic response to air pollution, including during pregnancy(27), induces pro-inflammatory responses evidenced by heightened cytokine production and serum c-reactive protein levels. (27, 32, 37–40) Exposure to chronic stress leads to excessive release of stress hormones resulting in physiologic dysregulation, including impaired immune function, and consequent excessive inflammation.(42, 43) Evidence of potential physiologic dysregulation and impaired immune function in regards to ethnic enclaves is seen among Hispanic women, as those residing in non-enclave areas have higher risk of increased allostatic load, which incorporates immune function, compared to those residing in ethnic enclaves.(20) Impaired immunologic function may respond to air pollution exposure with excessive inflammation, resulting in excessive release of pro-inflammatory cytokines and damage to healthy cells, which in turn can lead to insulin resistance, a precursor to metabolic disease.(41, 42) Thus, the similar systemic inflammatory and oxidative stress responses between exposure to chronic stress and exposure to air pollution may explain the synergic effects between residence in non-enclave areas and exposure to high levels of VOCs.

Our findings are also in line with evidence suggesting that the deleterious effect of air pollution on health is stronger among those residing in more stressful contexts. For instance, air pollution exposure during the first year of life is associated with increased risk of childhood asthma, but only among children in high poverty areas.(70) Additionally, exposure to high levels of air pollution is more strongly associated with poor cardiometabolic health among adolescents residing in high-poverty

areas compared to those residing in low-poverty areas.(71) It is noteworthy that the observed GDM risks were independent of individual-level proxies of health insurance and marital status, suggesting residence in an ethnic enclave may buffer the negative consequences of exposure to high levels of air pollution.

Our observations highlight the importance of focusing on API communities in environmental health research. API communities are often identified as 'model minorities' due to higher socioeconomic status compared to other non-white racial/ethnic groups in the U.S., suggesting API communities have favorable health outcomes compared to other racial/ethnic groups.(6, 9) Reliance on the 'model minority' label, in addition to API encompassing approximately 6 percent of the U.S. population, contributes to limited representation of API populations in national datasets, poor recognition of disparities among API populations, and a lack of environmental justice research targeting API communities.(6–9) The lack of relevant data excludes API communities from environmental health policy and health promotion planning when they may be an at-risk group.(7, 9) Given known health disparities and adverse environmental exposures among API communities, environmental justice research should increase efforts to better address disparities impacting API communities.

In order to address persistent racial/ethnic health disparities in the U.S. API communities should lead culturally-specific efforts to jointly improve social and environmental conditions. Previous attempts to improve environmental conditions have failed when a community's cultural considerations have not been taken into account, resulting in worse environmental conditions and rapid displacement and gentrification.(72–74) API communities in California have been successful in community-led efforts to assemble multisector coalitions to implement environmentally friendly transportation and infrastructure improvements, affordable housing developments, and economic vitalization that reflect cultural values of communities.(72) However, further research is warranted to better understand the population-health benefits of these community-led efforts.

Our findings are notable for several reasons. First, to the best of our knowledge, this is the initial investigation of joint exposure to air pollution and residence in an ethnic enclave among pregnant women. The observations that residence within an ethnic enclave mitigates air pollution suggest chronic exposure to low or high stress prior to pregnancy has important physiologic implications during pregnancy. Secondly, this study expands our understanding of complex socioenvironmental exposures among an understudied minority population. API communities are at greater risk for high air pollution exposure, and are typically concentrated within urban areas in the U.S. Lastly, this study benefits from a large amount of clinical data for a large sample of API women in the CSL. This allows for a robust examination of community-level risk factors for GDM, a condition that disproportionately affects U.S. API women.

These findings are best considered in the context of the study's limitations. Our measure of ethnic enclaves has not been validated in studies outside of the CSL,(11) yet was informed by attempts to capture geographic and social distinctions of ethnic enclaves. API women in the CSL are aggregated into a single category, not allowing us to examine API women by ancestry. Due to this, we used the aggregated API census data to measure ethnic enclaves. This limits our observations as API ancestry may be related to GDM risk,(18) and air pollution exposure,(6, 75) and effect of ethnic enclave residence may differ by API ancestry.(17, 18) However, previous analyses suggest the API population of metropolitan areas represented in the CSL is over 93% women of Asian ancestry with relatively few Pacific Islander women.(12) The CSL lacks maternal residential history, limiting our understanding of length of exposure to ethnic enclaves. However, most residential relocation during pregnancy occurs with a similar geographic area, and cross-sectional data allows for an approximate understanding of chronic exposures to community-level factors.(76)

VOC exposure was averaged over HRRs in which the birth occurred and was not based on participant residence. Exposure misclassification may occur if mothers resided outside the HRR for all or part of their pregnancy. However, while 10–30% of pregnant women change residence during pregnancy, most move to an area of similar level of air pollution.(77, 78) Misclassification may also be a function of local mobility and activity patterns of pregnant women. While the CSL does not

have local mobility or daily activity data, current evidence suggests pregnant women and a general population comparison group both spent approximately 15 hours per day indoors.(79) Additionally, during the first trimester of pregnancy, exposure estimates based on residential address are strongly correlated with exposure estimates accounting for daily activities ($r = 0.98$, $p < 0.01$).(80)

Conclusions

In conclusion, we observed that API women residing in non-enclave areas have higher risk for GDM, regardless of VOC level. Residence in an ethnic enclave may mitigate the negative health effects of VOC exposure, potentially due to lower stress levels. Lower levels of stress among residents of ethnic enclaves may be related to greater access to culturally-relevant goods and services, and greater political representation.(3, 4, 15, 24) API communities should lead culturally-relevant efforts to promote health through improved social and environmental conditions. Additional research is warranted to better understand the effects of joint exposures to air pollution and ethnic enclave across diverse ancestry groups within the broader U.S. API population.

Abbreviations

95% CI
95 percent confidence intervals
ACS
American Community Survey
API
Asian/Pacific Islander
BMI
body mass index
CSL
Consortium on Safe Labor
GDM
gestational diabetes mellitus
HRR
Hospital referral region
OR
odds ratio
U.S.
United States
VOC
volatile organic compounds
ZCTA
zip code tabulation area

Declarations

Ethics Approval: Institutional Review Boards at all study sites approved the CSL. The University of North Dakota Institutional Review Board waived need for approval.

Availability of data and materials: Consortium on Safe Labor data is publicly available at

https://dash.nichd.nih.gov/. Geographic identifying information is not publicly available, please

see http://grants.nih.gov/grants/policy/data_sharing/ for National Institutes of Health data

sharing policy.

Dartmouth Atlas of Healthcare data is available at https://www.darthmouthatlas.org/data

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Author contributions: AW contributed to study design, conducted statistical analyses, interpreted the data, and wrote the first draft of the manuscript. LM, JK, SH, ES, and PM each contributed to the concept of the study, interpretation of the data, and provided extensive comments and critiques of the manuscript. All authors read and approved the final manuscript.

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