A Mixed-Methods Study to Examine the Role of Psychosocial Stress and Air Pollution on Hypertension in Mexican-Origin Hispanics

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Abstract
Purpose Independent and combined effects of air pollution and psychosocial stressors on hypertension, a risk factor for cardiovascular disease, among Hispanics are not well studied.

Methods We administered a pilot-tested questionnaire on individual- and neighborhood-level psychosocial stressors, developed with community input, to nearly 2500 individuals from the MD Anderson Cancer Center cohort of Mexican-Americans. We used data from local air quality monitors to estimate individual exposures to ozone (O₃) and fine particulate matter (PM₂.₅) for the 12-month period preceding enrollment using inverse distance interpolation. We applied logistic regression models to examine relationships between exposures to psychosocial stressors and air pollution with prevalent hypertension and used stratified analyses to examine the interacting effects of these two exposures on hypertension.

Results There was a positive association between prevalent hypertension and a high frequency of feeling anxious or depressed (prevalence odds ratio (POR) = 1.36, 95% CI [1.06–1.75]) and experiencing aches and pains (POR = 1.29, 95% CI [1.01–1.64]). The odds of having hypertension were also elevated among those worrying about their own health (POR = 1.65, 95% CI [1.30–2.06]) or about not having enough money (POR = 1.27, 95% CI [1.01–1.6]). We observed an inverse association between O₃ and hypertension. There was no interaction between psychosocial stressors and O₃ on hypertension.

Conclusion Our findings add to the evidence of a positive association between individual and family stressors on hypertension among Hispanics and other racial/ethnic groups. Contrary to previous studies reporting positive associations, our results suggest that long-term exposure to O₃ may be inversely related to prevalent hypertension.

Keywords Air pollution · Psychosocial stress · Hypertension · Mexican-origin Hispanics
Introduction

A relatively large body of literature has examined associations between psychosocial stressors and hypertension, one of the leading risk factors for cardiovascular disease (CVD). Making comparisons between investigations, however, is challenging. Not only do the specific domains of psychosocial stress that have been evaluated vary between studies, but the indicators selected to measure these domains and the duration (e.g., acute versus chronic) and context (e.g., at work) in which stress occurs also differ [1]. Further, most of the research has focused on non-Hispanic whites [2–5].

Relatively less is known about the impact of psychosocial stress on the risk of hypertension among Hispanics in the United States (U.S.), for whom CVD is the leading cause of death [6]. Gallo et al. found that self-reported chronic stress was positively associated with increased odds of hypertension (OR = 1.10, 95% CI [1.02–1.19]) whereas traumatic stress was associated with a lower odds of hypertension (OR = 0.88, 95%, CI [0.82–0.93]) among adult Hispanics largely from Mexico, Cuba, and Central America [1]. Among postmenopausal Hispanic women, Zambrana et al. reported a positive association between depression and hypertension at baseline (OR = 1.25, 95% CI [1.04–1.51]), as well as between history of depression and pre-hypertensive status (OR = 1.27, 95% CI [1.01–1.61]) [7]. Acculturation has also been studied as a psychosocial risk factor for hypertension among Hispanics with conflicting findings [8–11]. Additionally, perceived race-based discrimination has been associated with hypertension among racial and ethnic minorities [12]. Hicken et al. found that racism-related vigilance, a source of chronic stress, is associated with hypertension among Hispanics (OR = 1.05, 95% CI [0.99–1.12]) [13]. Further, LeBron et al. report that Latino immigrants are more likely to experience increases in blood pressure associated with individual or institutional discrimination compared to US-born Latinos [14].

Beyond psychosocial stressors, there are ample studies underscoring the putative role of exposures to outdoor air pollutants on risk of hypertension [15–20]. Of particular concern is fine particulate matter (particulate matter with aerodynamic diameter less than 2.5 μm; PM2.5) [18, 21–24]. In contrast, fewer studies have examined the risks of hypertension associated with exposure to ozone (O3) [16, 18], a secondary air pollutant formed when oxides of nitrogen and volatile organic compounds interact in the presence of sunlight. Thus far, the relation between O3 exposure and hypertension is equivocal [25–30] and unlike PM2.5, no studies have investigated the association between O3 and hypertension among U.S. Hispanics.

Proinflammatory and oxidative stress pathways have been posited as underlying biological mechanisms for CVD. Potential pathways linking psychosocial stressors and CVD involve neuroendocrine activity of the autonomic nervous system (ANS) and the hypothalamus-pituitary adrenal (HPA) axis [31]. Ambient air pollutants are capable of mediating adverse cardiovascular responses through several mechanisms, such as impacting endothelial and other hemodynamic function, triggering acute autonomic imbalance and oxidative stress in the lungs with systematic inflammatory responses [15–20].

We designed a study to address the paucity of literature informing the role of air pollution and psychosocial stress on hypertension among individuals of Mexican-origin in Houston, Texas. In addition to being a busy seaport and home to the largest petrochemical complex in the country, Houston’s heavy traffic contributes to its poor air quality and the city’s diverse residents face documented health disparities [32–35].

In the present analysis, we recruited 2481 participants aged 20 years or older who enrolled in Mano a Mano Mexican-American cohort study in Houston, Harris County, Texas (n = 23,606) [38]. At baseline, participants complete an interview in the language of their choice (either English or Spanish) and provide information about health status, demographic characteristics, access to healthcare, degree of acculturation, lifestyle behaviors, and occupational and residential histories. As part of the Mano a Mano study, additional follow-up telephone interviews occur every 6 months.

For the present analysis, we recruited 2481 participants aged 20 years or older who enrolled in Mano a Mano between 2007 and 2014, based on responses to the question: “have you been told by a health professional that you have high blood pressure, also called hypertension?” After excluding participants (n = 13) without valid geographic coordinates for their residential address, the final sample size was 2468: 1135 cases with hypertension and 1333 controls without hypertension. There were 87 households with two participants and six households with more than two participants.

Methods

Study Population

Participants were randomly selected from The University of Texas MD Anderson Cancer Center (MDACC) Mano a Mano Mexican-American cohort study in Houston, Harris County, Texas (n = 23,606) [38]. At baseline, participants complete an interview in the language of their choice (either English or Spanish) and provide information about health status, demographic characteristics, access to healthcare, degree of acculturation, lifestyle behaviors, and occupational and residential histories. As part of the Mano a Mano study, additional follow-up telephone interviews occur every 6 months.

For the present analysis, we recruited 2481 participants aged 20 years or older who enrolled in Mano a Mano between 2007 and 2014, based on responses to the question: “have you been told by a health professional that you have high blood pressure, also called hypertension?” After excluding participants (n = 13) without valid geographic coordinates for their residential address, the final sample size was 2468: 1135 cases with hypertension and 1333 controls without hypertension. There were 87 households with two participants and six households with more than two participants.

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Psychosocial Stressors

We collected primary data in 2014–2015 during regularly scheduled Mano a Mano follow-up interviews. Following development of a questionnaire [39], trained interviewers administered a 32-item survey in the language preference of participants (English or Spanish) on psychosocial stressors in the home, neighborhood, and at work that they may have experienced at the time they enrolled. Questions were also asked about certain behaviors and lifestyle preferences that might affect exposure to air pollutants. Participants responded to each question using a five-point Likert scale: Not at all; Yes, a little bit; Yes, sometimes; Yes, a lot of the time; Yes, Most of the time. For analyses, responses were collapsed into three categories: low (no, not at all and yes, a little bit), medium (yes, sometimes) and high (yes, a lot of the time and yes, most of the time). Large proportions of participants (66% percent of cases and 63% of controls) did not answer the questions about work-related stressors (e.g., occupational exposure to chemicals, unsafe work conditions, working too hard). Hence, these questions were not further analyzed.

Long-Term Exposures to PM$_{2.5}$ and O$_3$

We obtained validated hourly air pollution data for O$_3$ and PM$_{2.5}$ from the Texas Commission on Environmental Quality (TCEQ), the environmental agency for the state. We used data from all active monitoring stations that continuously measured hourly O$_3$ ($n = 49$) and PM$_{2.5}$ ($n = 15$) concentrations in the 8-county greater Houston area (i.e., Brazoria, Chambers, Fort Bend, Galveston, Harris, Liberty, Montgomery, and Waller Counties) for 2006 through 2014 (Fig. 1). We excluded one O$_3$ monitoring station and one PM$_{2.5}$ monitoring station reporting ≥25% missing observations over the entire study period. Ozone and PM$_{2.5}$ concentrations were reported in parts per billion and micrograms per cubic meter, respectively.

Exposure estimates were constructed using SAS (Version 9.4, SAS Institute, Cary, North Carolina) and ArcGIS Desktop (Release 10.2.2., Environmental Systems Research Institute (ESRI), Redlands, California). We calculated the maximum average eight-hour O$_3$ concentration within a 24-h period and the daily average PM$_{2.5}$ concentration for each monitoring station from January 1, 2006 to December 31, 2014. Using
this daily time series of O$_3$ and PM$_{2.5}$ concentrations, we assigned individual exposure estimates based on the average concentration for the 12-month period preceding baseline enrollment using inverse distance weighting (IDW; $p=2$) [40] for the three monitoring stations nearest to the participant’s geocoded residential address.

**Covariates**

Demographic characteristics were obtained during the baseline Mano a Mano interview. Age at baseline was categorized into four strata (<30, 30–39, 40–39, 50+). Education was assessed in terms of highest level completed and collapsed into three levels (< High School, High School/General Education Development (GED), and > High School education). Nativity status (U.S.- or Mexico-born) was used to measure acculturation. Annual household income in the year preceding baseline was broken down into four levels ($\leq$ $24,999, $25,000 to $44,999, $45,000 to $74,999, $\geq$ $75,000). Smoking and alcohol use were categorized as current, former, or never. Body Mass Index (BMI) was calculated and categorized as underweight/normal weight ($< 25.0$ kg/m$^2$), overweight ($25.0 < 30.0$ kg/m$^2$), extremely obese II ($35.0 < 40.0$ kg/m$^2$), and extremely obese III ($\geq 40.0$ kg/m$^2$). Having asthma at baseline as diagnosed by a healthcare provider was reported as “yes” or “no”.

**Statistical Analyses**

All statistical analyses were conducted using SAS software (Version 9.4, SAS Institute, Cary, North Carolina). We used logistic regression and computed prevalence odds ratios (POR) and 95% confidence intervals (CI) to examine associations between psychosocial stressors or air pollution and hypertension. We examined air pollution exposures as continuous or categorical (quartiles) variables, in separate models. The following variables were identified a priori as risk factors and included in all adjusted models: age, sex, nativity, smoking, alcohol, BMI, and having asthma. We also evaluated education and employment using the change-in-estimate approach [41] but their inclusion did not change the effect estimate by more than 10% and thus, they were excluded from the final models. We additionally used stratified analysis to examine potential interaction between air pollution and those psychosocial stressors that were independently associated with hypertension ($p < 0.05$).

**Sensitivity Analyses**

We conducted sensitivity analyses using air pollution exposure estimates constructed with a single (i.e., the closest) monitor and applied mixed-effects logistic regression models with household specified as random effect to account for the correlation among individuals living together in the same household.

The Institutional Review Boards at MDACC and the University of Texas Health Science Center at Houston (UTHHealth) approved the study and oral informed consent during phone interviews was obtained from all participants.

**Results**

The majority of participants lived within the Houston city limits (66.25%). The next largest proportion of participants lived in Pasadena (22.11%), a community located east of Houston, near the Houston Ship Channel and numerous industrial facilities. Individuals ranged in age from 20 to 60 years at baseline; the mean age was 53 years (SD 11.15) among cases and 40 years (SD 10.77) among controls (Table 1). Over 85% of cases and controls were women and most had less than 12 years of education (64% of cases and 57% of controls) and were born in Mexico (72% of cases and 85% of controls). A large proportion of cases (42%) and controls (39%) did not report on income (data not shown).

Selected percentiles of the distribution of O$_3$ and PM$_{2.5}$ exposures appear in Table 2. The median (IQR) O$_3$ exposure was 35.41 ppb (4.78) among cases and 36.07 ppb (2.69) among controls. The median (IQR) PM$_{2.5}$ exposure was 11.44 μg/m$^3$ (1.12) among cases and 11.58 μg/m$^3$ (0.85) among controls. Due to the lack of variability in estimated exposure to PM$_{2.5}$, we excluded this pollutant from subsequent analyses.

Table 3 presents adjusted associations between hypertension and sources of psychosocial stress. There was a positive association between both reporting high frequency of stress due to unfair or disrespectful treatment based on race, ethnicity, or immigration status (POR = 1.55, 95% CI [1.04–2.32]) as well as stress due to too much litter or trash in the neighborhood (POR = 1.48, 95% CI [1.06–2.07]) and hypertension. Lower odds of prevalent hypertension were observed among individuals experiencing medium (POR = 0.60, 95% CI [0.40–0.90]) and high (POR = 0.87, 95% CI [0.55–1.38]) levels of stress due to domestic violence.

Table 4 presents adjusted associations between stress-related conditions and hypertension. There was a positive association between both a high frequency of feeling anxious or depressed (POR = 1.36, 95% CI [1.06–1.75]) and experiencing aches, pains or nausea (POR = 1.29, 95% CI [1.0–1.64]) and hypertension. The odds of having hypertension were also elevated among those with concerns about health (POR = 1.65, 95% CI [1.30–2.06]) or not having enough money (POR = 1.27, 95% CI [1.01–1.6]).

Adjusted PORs (95% CIs) for the association between O$_3$ exposure and hypertension were 0.89 (0.69–1.15), 0.44 (0.33–0.58), and 0.55 (0.42–0.72) for the second, third, and fourth quartiles of O$_3$, respectively, as compared to the lowest
**Table 1** Sociodemographic characteristics and self-reported hypertension among Mexican-origin Hispanics (N=2468) (missing observations are not shown), Houston, Texas, 2007–2014

|                                | Cases (n=1135) | Controls (n=1333) | OR  | 95% CI   |
|--------------------------------|----------------|-------------------|-----|----------|
| **Age (years)**                |                |                   |     |          |
| < 30                           | 22             | 219               | Ref |          |
| 30–39                          | 131            | 503               | 2.59*| 1.61     | 4.18     |
| 40–49                          | 271            | 359               | 7.51*| 4.72     | 11.97    |
| 50+                            | 711            | 252               | 28.09*| 17.71    | 44.55    |
| **Gender**                     |                |                   |     |          |
| Men                            | 163            | 99                | Ref |          |
| Women                          | 972            | 1234              | 0.48*| 0.37     | 0.62     |
| **Nativity**                   |                |                   |     |          |
| Mexico                         | 820            | 1131              | Ref |          |
| USA                            | 314            | 201               | 2.16*| 1.77     | 2.63     |
| **Education (years)**          |                |                   |     |          |
| 13+                            | 209            | 264               | Ref |          |
| < 12                           | 722            | 764               | 1.19 | 0.97     | 1.47     |
| High school graduate/GED       | 203            | 305               | 0.84 | 0.65     | 1.08     |
| **Employment**                 |                |                   |     |          |
| Never                          | 192            | 210               | Ref |          |
| Ever                           | 936            | 1103              | 0.93 | 0.75     | 1.15     |
| **Smoking**                    |                |                   |     |          |
| Never                          | 842            | 1113              | Ref |          |
| Current                        | 81             | 88                | 1.22 | 0.89     | 1.67     |
| Former                         | 211            | 132               | 2.11*| 1.67     | 2.67     |
| **Alcohol Consumption**        |                |                   |     |          |
| Never                          | 837            | 1049              | Ref |          |
| Current                        | 169            | 200               | 1.06 | 0.85     | 1.33     |
| Former                         | 129            | 79                | 2.05*| 1.53     | 2.75     |
| **Body mass index (kg/m^2)**   |                |                   |     |          |
| Underweight/normal weight (< 24.9) | 103           | 244               | Ref |          |
| Overweight (25.0 to 29.9)      | 303            | 455               | 1.58*| 1.20     | 2.07     |
| Obese I (30.0 to 34.9)         | 309            | 350               | 2.09*| 1.59     | 2.76     |
| Obese II (35.0 to 39.9)        | 199            | 168               | 2.81*| 2.06     | 3.82     |
| Obese III (≥ 40.0)             | 190            | 75                | 6.00*| 4.22     | 8.54     |
| **Asthma**                     |                |                   |     |          |
| No                             | 451            | 342               | Ref |          |
| Yes                            | 684            | 991               | 0.52*| 0.44     | 0.62     |

OR: odds ratio; CI: confidence interval

* p < 0.05 for associations between covariates and hypertension

**Table 2** Distribution of annual 8-h maximum O<sub>3</sub> and 24-h average PM<sub>2.5</sub> exposure estimates among Mexican-origin Hispanics (N=2468), Houston, Texas, 2007–2014

|                                | Mean ± SD | 25th percentile | 50th percentile | 75th percentile | Range   |
|--------------------------------|-----------|------------------|-----------------|-----------------|---------|
| **O<sub>3</sub> (ppb)**        |           |                  |                 |                 |         |
| Cases                          | 35.45 ± 1.95 | 34.06            | 35.41           | 38.84           | 27.54–42.11 |
| Controls                        | 35.88 ± 2.04 | 34.56            | 36.07           | 37.25           | 23.77–43.97 |
| **PM<sub>2.5</sub> (µg/m<sup>3</sup>)** |        |                  |                 |                 |         |
| Cases                          | 11.60 ± 0.92 | 10.94            | 11.44           | 12.06           | 9.51–14.88 |
| Controls                        | 11.77 ± 0.92 | 11.24            | 11.58           | 12.09           | 9.36–14.92 |
quartile. Odds of hypertension decreased by a factor of 0.90 for each parts per billion increase in exposure to $O_3$ (adjusted POR = 0.90, 95% CI [0.86–0.95]). There was no evidence of effect measure modification by psychosocial stress in the association between ozone and hypertension. These results did not change when using data from a single monitor to construct air pollution exposure estimates (data not shown). Further, the results from the mixed-effects models accounting for the correlation among individuals living in the same household were similar as well (Adjusted PORs (95% CIs) were 0.89 (0.68–1.15), 0.44 (0.33–0.59), and 0.55 (0.41–0.73) for the second, third and fourth quartiles of $O_3$, respectively).

**Discussion**

We examined co-exposures to air pollution and psychosocial stress among an overburdened population, i.e., Mexican-origin Hispanics living in Houston, Texas. We observed elevated odds of prevalent hypertension with several conditions resulting from stress including feeling anxious or depressed, experiencing aches, pains, or nausea and having concerns about poor health and not having enough money. Additionally, we detected associations between hypertension and being unfairly treated or disrespected because of race, ethnicity or immigration status and having too much litter and trash in the neighborhood. While we could not examine associations with PM$_{2.5}$ because of too little variability in our exposure estimates, we found inverse associations between ozone exposure and hypertension.

In our study, experiencing a high level of stress-induced anxiety or depression was associated with a 36% increase in the odds of prevalent hypertension. Zambrana et al. reported similar associations between depression and prevalent hypertension among postmenopausal Hispanic women ages 50 and older [7]. In a meta-analysis, Meng et al. reported elevated risks of hypertension with depression, which increased with longer follow-up time [42]. We also found that a high level of stress from unfair or disrespectful treatment based on race, ethnicity or immigration status was positively associated with hypertension, which is consistent with the literature on

### Table 3

Association between sources of psychosocial stressors and prevalent hypertension among Mexican-origin Hispanics ($N = 2468$) (missing observations are not shown) Houston, Texas, 2007–2014

| Stressor                                      | Cases  | Controls | POR*  | 95% CI |
|----------------------------------------------|--------|----------|-------|--------|
| Domestic violence                            |        |          |       |        |
| Low                                          | 1023   | 1196     | Ref   |        |
| Medium                                       | 58     | 85       | 0.60* | 0.40   | 0.90   |
| High                                         | 54     | 52       | 0.87  | 0.55   | 1.38   |
| Problems with children                       |        |          |       |        |
| Low                                          | 582    | 728      | Ref   |        |
| Medium                                       | 249    | 352      | 1.03  | 0.82   | 1.31   |
| High                                         | 279    | 249      | 1.17  | 0.92   | 1.49   |
| Caring for a sick family member              |        |          |       |        |
| Low                                          | 788    | 1005     | Ref   |        |
| Medium                                       | 145    | 156      | 1.03  | 0.77   | 1.37   |
| High                                         | 202    | 172      | 0.99  | 0.76   | 1.30   |
| Separated from family living elsewhere       |        |          |       |        |
| Low                                          | 866    | 890      | Ref   |        |
| Medium                                       | 123    | 226      | 0.83  | 0.63   | 1.10   |
| High                                         | 146    | 217      | 0.90  | 0.68   | 1.19   |
| Contact with authorities/law enforcement     |        |          |       |        |
| Low                                          | 975    | 1112     | Ref   |        |
| Medium                                       | 99     | 147      | 0.80  | 0.59   | 1.10   |
| High                                         | 61     | 74       | 0.73  | 0.49   | 1.10   |
| Unfair treatment/disrespect based on race, ethnicity or immigration status | | | | |
| Low                                          | 179    | 357      | Ref   |        |
| Medium                                       | 119    | 213      | 1.13  | 0.80   | 1.59   |
| High                                         | 100    | 89       | 1.55* | 1.04   | 2.32   |
| Neighborhood noise                           |        |          |       |        |
| Low                                          | 898    | 1106     | Ref   |        |
| Medium                                       | 141    | 151      | 1.14  | 0.85   | 1.53   |
| High                                         | 96     | 76       | 1.19  | 0.81   | 1.73   |
| Neighborhood traffic/construction            |        |          |       |        |
| Low                                          | 749    | 884      | Ref   |        |
| Medium                                       | 208    | 272      | 0.99  | 0.77   | 1.26   |
| High                                         | 178    | 177      | 1.09  | 0.82   | 1.43   |
| Neighborhood litter/trash                    |        |          |       |        |
| Low                                          | 871    | 1070     | Ref   |        |
| Medium                                       | 138    | 168      | 0.98  | 0.73   | 1.31   |
| High                                         | 126    | 95       | 1.48* | 1.06   | 2.07   |
| Being safe in home or neighborhood           |        |          |       |        |
| Low                                          | 716    | 850      | Ref   |        |
| Medium                                       | 255    | 340      | 0.99  | 0.79   | 1.24   |
| High                                         | 164    | 143      | 1.21  | 0.90   | 1.62   |
| Unknown people hanging around the neighborhood |        |          |       |        |
| Low                                          | 788    | 922      | Ref   |        |
| Medium                                       | 209    | 289      | 0.92  | 0.72   | 1.17   |
| High                                         | 138    | 122      | 1.17  | 0.85   | 1.61   |
| Violence at children’s school                |        |          |       |        |
| Low                                          | 845    | 956      | Ref   |        |

**Table 3 (continued)**

| Stressor                                      | Cases  | Controls | POR*  | 95% CI |
|----------------------------------------------|--------|----------|-------|--------|
| Medium                                       | 152    | 235      | 0.82  | 0.63   | 1.07   |
| High                                         | 138    | 142      | 1.13  | 0.84   | 1.52   |

*POR prevalent odds ratio; CI confidence interval

*p < 0.05 for associations between covariates and hypertension status

1 Adjusted for age, sex, nativity, smoking, alcohol consumption, BMI and asthma
perceived racial discrimination and hypertension [12–14]. In contrast, we observed that experiencing stress from domestic violence in the home resulted in lower odds of prevalent hypertension, which is similar to the inverse association reported previously between traumatic stressors (including physical or sexual assault) and hypertension [1].

We found an inverse association between 12-month averaged ozone exposure and prevalent hypertension. This finding is consistent with previous studies of short-term [29, 30] but not long-term exposure. Chuang et al. reported a 21.51-mmHg (95% CI [16.90–26.13]) change in systolic blood pressure and a 20.56-mmHg (95% CI [18.14–22.97]) change in diastolic blood pressure with an IQR increase of 8.95 ppb in 1-year averaged \(O_3\) concentrations among Taiwanese men and women ages 54 and older [27]. A study conducted in China found that an IQR increase of 22 µg/m³ (approximately 11 ppb) in 3-year averaged \(O_3\) concentrations increased the odds of prevalent hypertension (OR = 1.13, 95% CI [1.06–1.20]) [28]. In a study of black women in the U.S., Coogan et al. reported elevated risks of hypertension per IQR increase of 6.7 ppb of averaged \(O_3\) levels over 2 years (hazard ratio (HR) = 2.09, 95% CI [1.00–1.18]) [43].

Our study relied on prevalent cases of hypertension and was therefore unable to establish a temporal relationship between the exposures and the outcome. Further, the validity and accuracy of using self-reported hypertension have been evaluated with inconsistent results [44–49]. Hence, it will be important to evaluate the association of psychosocial stress and air pollution with incident hypertension when follow-up data become available in the Mexican-American Mano a Mano cohort. We constructed long-term estimates of \(O_3\) exposure based on the

| Stress-related condition | Cases \((n = 1135)\) | Controls \((n = 1333)\) | \(\text{POR}^1\) | 95% CI |
|-------------------------|-------------------|-------------------|----------------|--------|
| Anxiety/depression due to stress | Low 596 | 758 | Ref | |
| Medium 277 | 361 | 1.00 | 0.80 | 1.26 |
| High 262 | 214 | 1.36* | 1.06 | 1.75 |
| Aches/pains/nausea due to stress | Low 594 | 754 | Ref | |
| Medium 255 | 333 | 1.05 | 0.83 | 1.33 |
| High 286 | 246 | 1.29* | 1.01 | 1.64 |
| Trouble sleeping due to stress | Low 552 | 740 | Ref | |
| Medium 228 | 320 | 0.93 | 0.73 | 1.19 |
| High 355 | 273 | 1.22 | 0.96 | 1.53 |
| Worrying about not having enough time for oneself | Low 566 | 594 | Ref | |
| Medium 226 | 360 | 0.80 | 0.63 | 1.02 |
| High 343 | 379 | 0.96 | 0.77 | 1.20 |
| Worrying about one’s own health | Low 430 | 658 | Ref | |
| Medium 257 | 372 | 1.05 | 0.82 | 1.33 |
| High 448 | 303 | 1.65* | 1.30 | 2.06 |
| Worrying about not having enough money | Low 450 | 518 | Ref | |
| Medium 302 | 451 | 1.06 | 0.84 | 1.35 |
| High 383 | 364 | 1.27* | 1.01 | 1.60 |

\(\text{POR}\) prevalent odds ratio; CI confidence interval

* \(p < 0.05\) for associations between covariates and hypertension status

1 Adjusted for age, sex, nativity, smoking, alcohol consumption, BMI and asthma

Table 4 Associations between stress-related conditions and prevalent hypertension among Mexican-origin Hispanics \((N = 2468)\), Houston, Texas, 2007–2014
residential address of each participant using a relatively large air pollution database from stationary monitors in the study region. Thus, our exposure assessment likely captured spatial and temporal influences on outdoor air levels of ozone. Yet, the lack of equally distributed monitors in the study area may have introduced some error in our exposure assessment. Notwithstanding the complex and multi-dimensional aspects of stress that make it difficult to study, a strength of our study was in the use of mixed methods to assess exposure to psychosocial stressors.

Our study provides evidence of positive associations between multiple indicators of psychosocial stress in the family, social and neighborhood environments and hypertension in an ethnically homogenous population of Mexican-origin Hispanics. As CVD remains the leading cause of death among U.S. Hispanics, developing interventions that target some of these potentially modifiable sources of psychosocial stress may lead to improvements in cardiovascular health among this population.

Author Contributions Elaine Symanski designed the study and provided oversight for all aspects of data collection, analysis and interpretation. Melissa Bondy and Sara Strom provided access to the Mano a Mano cohort. Sara Strom oversaw data collection via phone interviews and Maria Jimenez served as the liaison between the MD Anderson Cancer Center interviewers and UTHealth School of Public Health researchers. Wenyaw Chan provided oversight of the statistical analysis; Amal Rammah performed all analyses. Amal Rammah and Elaine Symanski drafted the manuscript and Kristina Whitworth, Inkyu Han, and Sara Strom offered revisions of the manuscript. All authors contributed to and approved the final version.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval The Institutional Review Boards at MDACC and the University of Texas Health Science Center at Houston (UTHealth) approved the study and oral informed consent during phone interviews was obtained from all participants. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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