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Air pollution and metabolic disorders: Dynamic versus static measures of exposure among Hispanics/Latinos and non-Hispanics

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Abstract

Introduction: Exposure to air pollution disproportionately affects racial/ethnic minorities that could contribute to health inequalities including metabolic disorders. However, most existing studies used a static assessment of air pollution exposure (mostly using the residential address) and do not account for activity space when modelling exposure to air pollution. The aim of this study is to understand how exposure to air pollution impacts metabolic disorders biomarkers, how this effect differs according to ethnicity, and for the first time compare these findings with two methods of exposure assessment: dynamic and static measures.

Methods: Among the Community of Mine study, a cross-sectional study conducted in San Diego County, insulin resistance, diabetes, hypertension, obesity, dyslipidemia, and metabolic syndrome (MetS) were assessed. Exposure to air pollution ($PM_{2.5}$, NO_2, traffic) was calculated using static

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envres.2022.112846.

measures around the home, and dynamic measures of mobility derived from Global Positioning Systems (GPS) traces using kernel density estimators to account for exposure variability across space and time. Associations of air pollution with metabolic disorders were quantified using generalized estimating equation models to account for the clustered nature of the data.

Results: Among 552 participants (mean age 58.7 years, 42% Hispanic/Latino), Hispanics/Latinos had a higher exposure to $PM_{2.5}$ compared to non-Hispanics using static measures. In contrast, Hispanics/Latinos had less exposure to $PM_{2.5}$ using dynamic measures. For all participants, higher dynamic exposure to $PM_{2.5}$ and NO_2 was associated with increased insulin resistance and cholesterol levels, and increased risk of obesity, dyslipidemia and MetS (RR 1.17, 95% CI: 1.07–1.28; RR 1.21, 95% CI: 1.12–1.30, respectively). The association between dynamic $PM_{2.5}$ exposure and MetS differed by Hispanic/Latino ethnicity.

Conclusion: These results highlight the importance of considering people's daily mobility in assessing the impact of air pollution on health.

Keywords

Biomarkers; Pollution inequity; Ethnic inequalities; Geographic information systems (GIS); Kernel density estimators (KDE); Mobility

1. Introduction

In the US, it has been established that inequalities regarding detrimental environmental exposures across racial/ethnic groups exist (Hajat et al., 2015). Exposure inequalities could be even more pronounced between racial/ethnic groups compared to income and educational attainment (Mikati et al., 2018). Racial and ethnic minorities are often exposed to higher levels of air pollution compared to non-Hispanic Whites (Woo et al., 2019; Li et al., 2019; Bell and Ebisu, 2012; Rosofsky et al., 2018; Pratt et al., 2015; Clark et al., 2014; Demetillo et al., 2021; Chambliss et al., 2021). Such differences are exacerbated by residential segregation in metropolitan areas (Woo et al., 2019) and linked to racially discriminatory housing policies during the twentieth century (Chambliss et al., 2021). For example, in a study by Jones et al. across six US cities, neighborhoods with higher Hispanic composition have higher air pollution exposures (Jones et al., 2014). Such documented exposure inequalities may lead to a higher health burden attributable to air pollution among racial and ethnic minorities (Di et al., 2017). While air pollution is decreasing overall across the US, minority populations are not equally benefiting from pollutant reductions. In fact, inequalities in exposure to particulate matter among racial/ethnic communities have increased between 2010 and 2016 (Jbaily et al., 2022), highlighting the importance for air pollution regulations to focus on the reduction of environmental injustice across the US.

Most studies documenting inequalities and related health burdens regarding exposure to air pollution use static measures drawn from the residential neighborhood. Static measures do not consider total exposure throughout the day or time spent in other locations, limiting the ability to assess daily variations in exposure and potentially under-estimating pollution effects on health (Shareck et al., 2014). Some studies relied on individual pollution sensors to directly measure time-varying exposure to air pollutants (Liu et al., 2013; Yarza et

al., 2020; Chaix, 2018) and related health outcomes such as blood pressure or heart rate variability (Ren et al., 2019; Smargiassi et al., 2014; Buteau and Goldberg, 2016). However, such studies require a dedicated data collection and can be difficult to implement logistically (Languille et al., 2020). When such data collection is not feasible because of costs, or simply because the study was not directly designed to answer question related to dynamic exposure to air pollution specifically via sensors, it is possible to capitalize on studies that collected Global Positioning Systems (GPS) data. GPS data can be used to model daily mobility and indirectly estimate dynamic exposure to air pollution. In the past decade, many studies collected individual GPS data to characterize activity spaces and various exposures such as tobacco retailers (Shareck et al., 2016) or active transportation (Chaix et al., 2019). With such data, it becomes possible to combine spatio-temporal estimates of air pollutants such as fine particulate matter ($PM_{2.5}$) or nitrogen dioxide (NO_2) or alternative metrics such as distance to major highways, and methods to calculate dynamic or time-weighted exposures such as Kernel Density Estimators (KDE) (Smith et al., 2019), which provide spatially varying exposures weighted on the time spent in each location. Comparison between dynamic and static exposure approaches is important as it may document potential exposure misclassification when using residential air pollution, which is the common approach in environmental epidemiological studies. To the best of our knowledge, no study has evaluated disparities in exposure to air pollution between Hispanics/Latinos and non-Hispanics considering both static and dynamic methods.

In parallel, ethnic inequalities in metabolic disorders are well established (Schneiderman et al., 2014; Shiels et al., 2017; Heiss et al., 2014). Metabolic syndrome (MetS) is a suite of conditions that contribute to cardiovascular morbidity and premature mortality. It has been shown that the highest prevalence of MetS in the US was observed among Hispanics/Latinos (Aguilar et al., 2015). Recent studies have also shown that traffic-related air pollution might play a role in MetS incidence mostly through inflammatory pathways (Ursula et al., 2010; UP, 2015). Among Mexican-Americans, air pollution effects on metabolic disorders have been previously reported (Yu et al., 2020; Chen et al., 2016). Exposure to higher air pollution is associated with several metabolic outcomes in Hispanics/Latinos including insulin resistance, poor HDL-to-LDL cholesterol ratio, and elevations in fasting glucose, total cholesterol and blood pressure (Chen et al., 2016; Choi et al., 2019). Increased exposure to air pollution can lead to insulin resistance, associated with risk for type 2 diabetes, cancer and cardiometabolic disease (Dang et al., 2018). Understanding the role of air pollution on metabolic disorders biomarkers across ethnic groups while considering daily variation in air pollution exposure can help us understand the role of air pollution on ethnic disparities and formulate targeted interventions while accounting for heterogenous activity spaces.

The aim of this study is to understand how exposure to air pollution impacts biomarkers of metabolic disorders, how this effect differs according to ethnicity, and for the first time compare findings using two methods of exposure assessment: dynamic and static measures. We utilize data from the Community of Mine study, involving an ethnically diverse population in San Diego, CA, where both static measures of exposure around participants' home locations, and dynamic measures of exposure across multiple days were collected (Jankowska et al., 2019). Our objective can be divided into 3 steps. We first

investigated whether exposure to $PM_{2.5}$, NO_2 and traffic differed by ethnicity (Hispanics/Latinos vs non-Hispanics), and we compared findings derived from static and dynamic methods. We then studied the relationship between air pollution (through both static and dynamic exposure) and metabolic disorders outcomes. Finally, we examined whether this relationship is modified by Hispanic/Latino ethnicity.

2. Methods

2.1. Study population

The Community of Mine study was an observational study completed between 2014 and 2017 in San Diego County, California. The full protocol is described elsewhere (Jankowska et al., 2019). Briefly, 602 adults aged 35–80 years old living for at least 6 months in a selected census block groups completed the study. About 40% of the participants were Hispanics/Latinos. The main objective of the Community of Mine study is to advance methods of cancer risk exposure assessment by measuring both neighborhood access and total exposure to healthy and unhealthy environments by integrating GPS data with Geographical Information System (GIS) data across the full day. Participants wore GPS and accelerometer sensors for up to two weeks and attended a clinical visit where they completed a medical history interview that included any current medications. At the visit, blood pressure, height, weight, hip, and waist circumference were recorded, and blood and urine samples were collected. Fasting plasma insulin, fasting plasma glucose, total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), systolic blood pressure (SDB), diastolic blood pressure (DBP), triglycerides (TG), and hemoglobin A1C (HbA1c) were measured. Demographic characteristics (e.g., age, gender, race/ethnicity) were collected via self-report survey.

Study ethics approval was obtained from UCSD IRB protocol #140510. Signed informed consent was obtained from all participants who enrolled in the study.

2.2. Biological outcomes

We focused on six major biological outcomes: insulin resistance, diabetes, hypertension, obesity, dyslipidemia, and metabolic syndrome (MetS). Insulin resistance is gauged by the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR). The HOMA-IR index was calculated according to the formula: fasting plasma insulin (mlU/L) x fasting plasma glucose (mg/dL)/22.5. Diabetes was defined as a blood glucose level higher than 126 mg/dl. Hypertension was defined as blood pressure of at least 140/90 mm Hg. Obesity was defined as body mass index (BMI) over 30 kg/m2. Dyslipidemia was defined as the presence of one of more of the following conditions: TC≥200 mg/dL, TG≥150 mg/dL, LDL-C ≥ 130 mg/dL or HDL-C < 40 mg/dL. Using the NCEP ATP III definition (Huang, 2009), the presence of MetS (yes/no) was indicated based on having at least three of the following risk determinants: increased waist circumference (>102 cm [>40 in] for men, >88 cm [>35 in] for women); elevated triglycerides (≥150 mg/dl); low HDL cholesterol (<40 mg/dl in men, <50 mg/dl in women); hypertension (≥130/≥80 mmHg); and impaired fasting glucose (≥110 mg/dl).

2.3. Air pollution modelling

Based on daily observations of $PM_{2.5}$ and NO_2 in ug/m3 from eight Environmental Protection Agency Air Quality System (EPA AQS) monitoring stations in San Diego County, we used an inverse distance weighting approach to calculate yearly averages of air pollution across the study area for years 2014–2017. Interpolated values at each population-weighted zip code centroid were then assigned to each zip code. To validate interpolated estimates, each monitoring station data point was removed and then predicted at that location using remaining data points. Correlations between predicted and actual values at the location of the omitted point were used to assess validity of modeled estimates (with a minimum r of 0.55). The year a participant completed the study was matched to the same year of $PM_{2.5}$ and NO_2 air pollution if air pollution data was available in the census tract where the participant lived. If air pollution data from the year a participant completed the study was missing in the home census tract, air pollution data from the next subsequent year with available data was used. Traffic data was utilized as an additional proxy for air pollution. Average daily traffic volumes were pulled from ESRI Traffic Counts (Version 19.3; Q4 2019) data for years 2013–2017. Updated quarterly, ESRI Traffic Counts data are produced from Kalibrate TrafficMetrix®, and are an aggregation of traffic volumes from multiple sources including city governments, engineering firms, highway and transportation departments, and field verifications.

2.4. Exposure measures

2.4.1. Static—Static measures of exposure were derived from buffers around participant homes using circular buffers. Static exposure was measured using the average pollution (for years 2014–2017) value for a 1600 m buffer around the participant's home. We performed sensitivity analyses using additional buffer sizes for static exposure assessment (ranging from 1000 m to 3000 m).

2.4.2. Dynamic—Participants were asked to wear a GT3X ActiGraph accelerometer (ActiGraph, LLC; Pensacola, FL) and Qstarz GPS device (Qstarz International Co. Ltd, Taipei, Taiwan) on a belt around the waist for 14 days. Data was cleaned and processed through the Personal Activity and Location Measurement System (PALMS) and aggregated to the minute level (Carlson et al., 2015). Valid days were defined as having at least 10 h of accelerometer measured wear time after non-wear time was excluded using the validated Choi algorithm (Choi et al., 2012); missing GPS data was imputed per a previously validated algorithm (Meseck et al., 2016). It is important to note that participants do not wear devices during sleep, and thus wear time represents times of the day the participant is active and awake. Participants without at least four valid days were asked to re-wear devices for an additional seven days. Participants wore devices for an average (minimum - maximum) of 11.8 valid days (Hajat et al., 2015; Mikati et al., 2018; Woo et al., 2019; Li et al., 2019; Bell and Ebisu, 2012; Rosofsky et al., 2018; Pratt et al., 2015; Clark et al., 2014; Demetillo et al., 2021; Chambliss et al., 2021; Jones et al., 2014; Di et al., 2017; Jbaily et al., 2022; Shareck et al., 2014, 2016; Liu et al., 2013; Yarza et al., 2020; Chaix, 2018; Ren et al., 2019; Smargiassi et al., 2014; Buteau and Goldberg, 2016; Languille et al., 2020).

All valid-wear GPS points for the entire participant's wear time were used to create a time-weighted activity space using the Kernel Density Equation (KDE) model in ArcGIS with a 200 m bandwidth, which has been shown in a previous study to be a reliable distance for exposure estimation (Lee and Kwan, 2019). To account for variation in amount of time participants wore devices, we normalized the time-weighted density measure values for each participant on a scale of 0–1 where the greatest amount of time spent by a participant in a location was equal to 1, and the least equal to 0 (Jankowska et al., 2021). The time-weighted activity space was then multiplied by each pollution layer using raster map algebra. The average pollution score across the entire time-weighted activity space was calculated to derive a total exposure measure that accounts for locations visited and the time spent in all locations. This dynamic exposure, similarly, to the static exposure, aimed at capturing long-term exposures and was based on data from years 2014–2017.

2.5. Covariates

Covariates included age, sex, ethnicity (Hispanic/Latino vs. non-Hispanic), income (categorized into 3 categories: less then 30 k, 30 k - 55 k, more than 55 k), current smoking status (yes, no) and Body Mass Index (BMI) (categorized into lean/normal (<25 kg/m^2), overweight (25–30 kg/m^2), and obese (>30 kg/m^2)).

2.6. Statistical analyses

The baseline characteristics of the study population were described with mean (SD), median (interquartile range) or frequencies (%) according to ethnic status. Hispanics/Latinos' and non-Hispanics' static and dynamic exposures to the three measures of air pollution ($PM_{2.5}$, NO_2, and traffic) were compared using Wilcoxon test. Because the distribution of HOMA-IR was not normal, we chose to log transform this continuous variable. For the three exposures to air pollution generalized estimating equations (GEE) models with exchangeable matrixes were performed (accounting for participants clustering within census tracts) to quantify pollution exposure effects on six biological outcomes (one continuous outcome: log transformed HOMA-IR, and five binary outcomes: diabetes, hypertension, obesity, dyslipidemia, and presence of MetS). We used modified Poisson regressions to account for the high prevalence of the binary outcomes of interest. The GEE models were adjusted for age, sex, ethnicity, income, smoking status, and BMI (except for obesity ad MetS models). To evaluate whether the effect of air pollution on biological biomarkers was modified by ethnicity, we used stratified analyses. Moreover, to test if regression coefficients between air pollution and outcomes differed for Hispanic/Latino ethnicity, we used Cochran Q-tests, as recommended for assessing race/ethnicity as an effect measure modifier (Benmarhnia et al., 2021).

In supplementary analysis, we quantified the association between air pollution levels and eight continuous biomarkers of metabolic disorders: cholesterol (mg/dl), LDL cholesterol (mg/dl), SDB (mm Hg), DBP (mm Hg), triglycerides (mg/dl), HbA1c (%), fasting plasma glucose (mg/dl) and BMI (kg/m^2).

Analyses were performed with R Version 4.1.

3. Results

3.1. Study population

Among the 602 participants, we restricted the population to participants with HOMA-IR, diabetes, hypertension, obesity, dyslipidemia, and MetS data. Participants with missing information on covariates (income and smoking status) and without air pollution exposure data ($PM_{2.5}$, NO_2, or traffic data) were also excluded (Fig. 1).

Among 552 participants included, 312 were female (56.5%) and 231 were Hispanic/Latino (42.2%). The mean age was 58.7 years (SD 11.0). In comparison to non-Hispanics, Hispanic/Latino participants were younger (median age: 55.9 vs. 60.7), more often women (62.3 vs 52.3%), poorer, and they had worse cardiovascular health (Table 1). Hispanics/Latinos had higher levels of insulin resistance (HOMA-IR median: 2.8 vs 1.8). Diabetes (11.3 vs 5.6%) and obesity (44.5 vs 28.7%) were also more frequent in Hispanic/Latino participants. However, no differences between Non-Hispanic and Hispanic participants were found for hypertension, dyslipidemia and MetS.

3.2. Air pollution exposures among Hispanics/Latinos and non-Hispanics

Using static air pollution measures, Hispanics/Latinos had higher exposure to $PM_{2.5}$ compared to non-Hispanics (9.36 vs 9.12 μg/m3, p-value<0.0001) (Table 2). However, with dynamic measures, they had lower exposure to $PM_{2.5}$ (0.066 vs 0.073, p-value = 0.07). Using static and dynamic measures, Hispanics/Latinos had lower exposure to NO_2 compared to non-Hispanics (9.77 vs 11.28 μg/m3 and 0.080 vs 0.092, respectively). For traffic-related exposures, we did not find any differential exposure between ethnic groups using both static and dynamic exposures.

3.3. Air pollution and biological outcomes

For all participants, higher dynamic exposure to NO_2 was found to be associated with increased HOMA-IR (β 0.08, 95%CI: 0.03–0.13) (Table 3). Higher dynamic exposure to $PM_{2.5}$ and NO_2 was associated with obesity (RR 1.23, 95%CI: 1.10–1.37; RR 1.21, 95%CI: 1.10–1.33, respectively), dyslipidemia (RR 1.12, 95%CI: 1.03–1.22; RR 1.14, 95% CI: 1.06–1.22, respectively), and MetS (RR 1.17, 95%CI: 1.07–1.28; RR 1.21, 95%CI: 1.12–1.30, respectively). No associations were observed with static measures for all outcomes. Static and dynamic traffic exposures were not associated with HOMA-IR, diabetes, hypertension, obesity, dyslipidemia or MetS.

Regarding the associations between air pollution and continuous cardiometabolic outcomes, no association between air pollution was observed with SDB, DBP, HbA1c and fasting glucose (Table S1). However, dynamic air pollution measures ($PM_{2.5}$ and NO_2) was associated with the level of total cholesterol, LDL cholesterol, triglycerides, and BMI. Only one association was observed with static air pollution measures. Higher static exposure to NO_2 was associated with higher triglycerides (β 0.05, 95%CI: 0.02–0.09).

3.4. Association between air pollution and biological outcomes according to racial/ethnicity status

No significant interactions were found between static or dynamic exposure and racial/ethnicity status for all six outcomes. Among non-Hispanic participants, we found that dynamic exposure to $PM_{2.5}$ and NO_2 was associated with higher HOMA-IR level (β 0.13, 95%CI: 0.03–0.24, β 0.06, 95%CI: 0.00–0.11, respectively) (Fig. 2). Dynamic exposure to $PM_{2.5}$ was associated with higher risk of obesity (RR 1.21, 95%CI: 1.04–1.40), dyslipidemia (RR 1.16, 95%CI: 1.05–1.28), and MetS (RR 1.23, 95%CI: 1.12–1.34). Dynamic NO_2 exposure was also found to be associated with obesity (RR 1.18, 95%CI: 1.03–1.35), dyslipidemia (RR 1.21, 95%CI: 1.04–1.40) and MetS (RR 1.23, 95%CI: 1.12–1.33). Among Hispanic/Latino participants, greater dynamic exposure to NO_2 was associated with increased HOMA-IR (β 0.13, 95% CI: 0.03–0.24) and higher risk of obesity (RR 1.22, 95%CI: 1.05–1.42). Based on Cochran Q-test, the association between dynamic $PM_{2.5}$ exposure and MetS was significantly different according to ethnicity status (p-value 0.07) (Table S2).

The findings obtained through sensitivity analysis for static exposure (conducted for buffer sizes ranging from 1000 m to 3000 m) were in line with the 1600 m buffer findings. We did not find any association with static measures regardless of the buffer size used (see Table S3 and Table S4). Moreover, when we removed non-Hispanic non-White participants (n = 50) (i.e., confined the analysis to Hispanics/Latinos vs non-Hispanic Whites), the results did not change (data not shown).

4. Discussion

Using data from the Community of Mine study, which includes 552 Hispanic and non-Hispanic individuals in San Diego County, we observed major difference in air pollution health effects based on the type of air pollution assessment, static or dynamic measures. Air pollution exposure according to ethnicity was different based on static or dynamic assessment, and on which air pollution measure was used. Hispanics/Latinos were most exposed to $PM_{2.5}$ compared to non-Hispanics using static measures, in contrast they were least exposed to $PM_{2.5}$ using dynamic measures. For all participants, greater dynamic exposure to $PM_{2.5}$ and NO_2 was associated with increased insulin resistance, cholesterol, LDL-C, and triglycerides levels, and higher risk of obesity, dyslipidemia and MetS. The relationship between dynamic exposure to $PM_{2.5}$ and MetS is greater among non-Hispanic participants. All these patterns were not identified when the exposure was evaluated using static methods more traditionally utilized in the existing literature (except for the level of triglycerides).

In this study, we highlighted the importance of considering dynamic exposure to air pollution in relation to air pollution exposure in-equalities and various metabolic disorders outcomes. We also proposed an approach to capitalize on studies that collected GPS data to indirectly estimate dynamic exposure to air pollution considering spatio-temporal mobility. While some studies were specifically designed to measure dynamic exposure to air pollution using sensors (Chaix, 2018; Buteau and Goldberg, 2016), we offer an alternative approach

to investigate analogous research questions relying on secondary data in which GPS data is available, which is becoming more common in the past decade.

These results have important implications for future studies as they highlight the importance of considering dynamic measures when assessing health effects of air pollution exposure. Activity space has been defined as “the subset of all locations within which an individual has direct contact as a result of his or her day-to-day activities” (Golledge, 1997). It is a measure of daily mobility (Sherman et al., 2005) that better reflects the individual exposure to the environment by capturing the spatial range of daily experience, and thus may provide a more complete picture of the inequalities induced by residential environmental exposure (Vallée, 2017; Basta et al., 2010). Daily mobility and activity space are critical in the evaluation of inequalities in health (Shareck et al., 2014). Previous research showed substantial bias in neighborhood-limited measures of exposure. For example, exposure measures limited to the residential neighborhood may underestimate true exposure to certain food store types (Kestens et al., 2012) or even exposure to ambient NO_2 (Setton et al., 2011). Bias may also become more strong as the geographic mobility (time and distance away from home) increases (Setton et al., 2011). Then, it was previously highlighted that considering mobility could exacerbated social inequalities (Shareck et al., 2014). However, few studies have directly examined the relationship between daily mobility and ethnic inequalities in health.

We highlighted that dynamic exposures to $PM_{2.5}$ and NO_2 were associated with insulin resistance, cholesterol levels, obesity, dyslipidemia and MetS, while static measures were not. However, we did not identified association neither with hypertension (including continuous SDB and DBP) nor with diabetes (including continuous fasting plasma glucose). It was previously shown that increased exposure to air pollution is associated with insulin resistance, increasing risk for type 2 diabetes, cancer and cardiometabolic diseases (Dang et al., 2018; Zhang et al., 2021). However, only two studies focused on Mexican-Americans (a growing portion of the US population with a high occurrence of MetS), and reported associations with insulin resistance, elevated fasting glucose and total cholesterol using static measures of air pollution (Yu et al., 2020; Chen et al., 2016).

In the US, Hispanics/Latinos have a higher prevalence of several cardiovascular risk factors such as obesity, diabetes mellitus, and dyslipidemia (Cortes-Bergoderi et al., 2013). In our study, Hispanic/Latino participants had poorer metabolic and cardiovascular health outcomes. However, we found one difference in health impact of dynamic air pollution exposure depending on Hispanic/Latino ethnicity, and paradoxically this impact seemed more important in non-Hispanic participants. The stronger impact of dynamically measured air pollution exposure on MetS among non-Hispanics than Hispanics/Latinos remains unclear. It could be due to some demographic differences (e.g., different prevalence of gender, important age difference) as well as difference in air pollution exposure. While Hispanics/Latinos tended to live in areas of higher $PM_{2.5}$ pollution (static exposure), their dynamic exposure (which also accounts for exposure at home) was lower for both $PM_{2.5}$ and NO_2. The number of Hispanics/Latinos in our study were fewer compared to non-Hispanics, which reduces statistical power in stratified analyses (maximum 231 Hispanic/Latino participants vs. 321 non-Hispanics). It can also be due to difference in personal mobility such as active travel (i.e., biking and walking to get from place to place).

Previously, it was shown that Non-Hispanics had more device-based active travel minutes, derived from accelerometer and GPS devices, than Hispanics/Latinos (Crist et al., 2021). This unexpected finding is not consistent with previous literature. Such paradox has been identified for other outcomes previously (Grineski et al., 2015; Parker et al., 2018; Mehta et al., 2021), and could be explained by underlying socio-economic determinant. Yet, it is also possible than in our sample we may have recruited healthier Hispanic participants as compared to the target population. Therefore, studies that would reproduce such analyses in other geographical contexts are crucial to unveil the role of air pollution on ethnic disparities in regards to metabolic disorders.

Our study has some limitations. First, our sample size is relatively small, and participants were recruited in the San Diego County limiting the generalization of these results. Second, the assessment of dynamic air pollution was carried out over a relatively short period of time (over a two-week period) and the health indicators were assessed at a single point in time. Third, some residual confounding may be present. While we controlled for several individual-level confounders such as income or age, and accounted for participants clustering within census tracts, some variables were not controlled for such as the type of occupation for example. Finally, individual-level data of air pollution exposure (from sensors for example) were not available which could lead to possible misclassification, and it would be interesting to compare individually measured dynamical exposure to air pollution (via sensors) and our approach to assess the amplitude of such bias. In the same vein, we used interpolated estimates from IDW models that have been traditionally used in epidemiological studies. Yet, more advanced interpolation (e.g. land use regression models based on ensemble models) have been developed in the past few years and may provide different estimates. It would be interesting for future studies having access to various air pollution estimates and GPS data to investigate to which extent the type of air pollution model influences the discrepancies between static and dynamic exposures.

Our findings highlighted the relevance of investigating both static and dynamic exposure when studying ethnic inequalities in health-relevant exposures. Our study showed that disregarding daily mobility and spatial variability of air pollution could lead to differential exposure misclassification and thus to biased health risk assessment. We found different health impact of exposure to air pollution according to the type of air pollution assessment, static or dynamic measures. Only dynamic exposure to air pollution was associated with insulin resistance, cholesterol, LDL-C, obesity, dyslipidemia and MetS.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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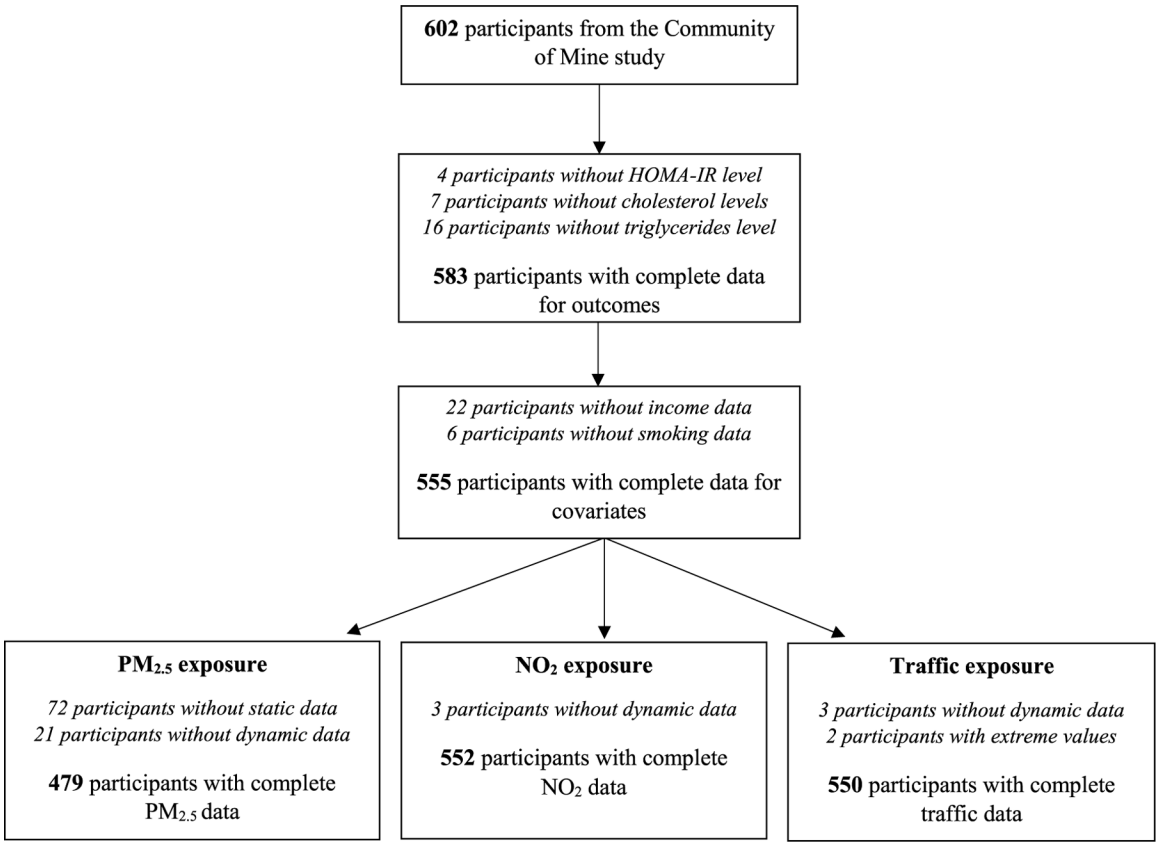

Fig. 1.

Flowchart showing how participants from the Community of Mine study were selected for this study.

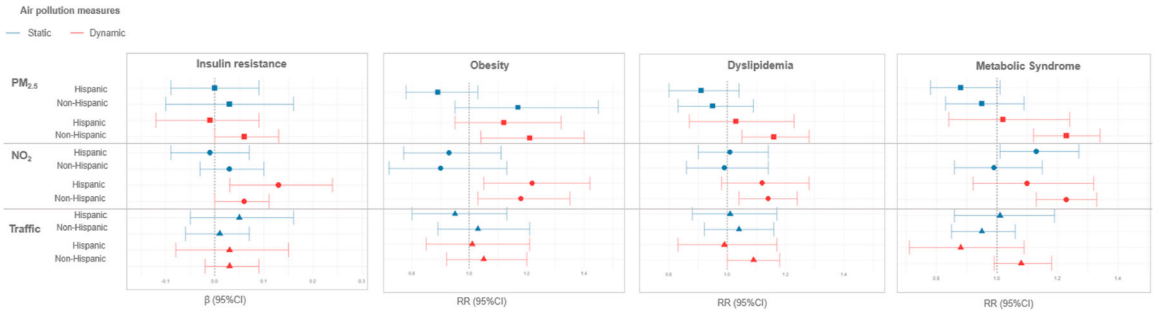

Fig. 2.

Association between air pollution measures (both static and dynamic) and metabolic disorders according to Hispanic/Latino ethnicity. $PM_{2.5}$: fine particulate matter, NO_2: nitrogen dioxide. Models were adjusted for sex, age, income, smoking status, and BMI (except for models with obesity and MetS as outcome).

Table 1

Distribution of participants characteristics from the Community of Mine study according to racial/ethnicity status (N = 552)

n, (%)	Non-Hispanic (n = 321)	Hispanic/Latino (n = 231)	p-value[c]
Female	168 (52.3)	144 (62.3)	0.02
Age[a]	60.7 (10.8)	55.9 (10.7)	<.0001
Income			<.0001
30 k or les	66 (20.6)	88 (38.1)	
30 k to 55 k	63 (19.6)	68 (29.4)	
55 k+	192 (59.8)	75 (32.5)	
Smoking	26 (8.1)	16 (6.9)	0.73
Presence of metabolic disorders			
HOMA-IR[b]	1.79 (1.06–2.86)	2.78 (1.55–4.66)	<.0001
Diabetes	18 (5.6)	26 (11.3)	0.02
Hypertension	82 (25.5)	44 (19.0)	0.09
Obesity	91 (28.3)	101 (43.7)	0.0003
Dyslipidemia	150 (46.7)	119 (51.5)	0.31
Metabolic Syndrome	127 (39.6)	100 (43.3)	0.43

HOMA-IR: Homeostatic Model Assessment of Insulin Resistance.

[a]Mean (SD).

[b]median (interquartile range).

[c]Student or Wilcoxon test for quantitative variable. Chi-square or Fisher test for qualitative variable.

Table 2

Air pollution measures among non-Hispanic and Hispanic participants.

Median, IQR	Non-Hispanic	Hispanic/Latino	*p*-value
Static exposure (1600m buffer)			
$PM_{2.5}$	9.12 (8.87–9.29)	9.36 (8.87–9.67)	<.0001
NO_2	11.28 (10.70–11.87)	9.77 (9.19–11.10)	<.0001
Traffic[a]	16,264 (13,587–18705)	16,159 (14,367–18108)	0.94
Dynamic exposure (KDE)			
$PM_{2.5}$	0.073 (0.054–0.104)	0.066 (0.048–0.100)	0.07
NO_2	0.092 (0.071–0.129)	0.080 (0.063–0.108)	<.0001
Traffic[a]	68.7 (45.4–109.0)	65.1 (44.7–99.1)	0.44

$PM_{2.5}$: fine particulate matter (ug/m^3), NO2: nitrogen dioxide (ug/m^3). Median, Interquartile Range (IQR), Wilcoxon test for comparison.

[a]Measured with average daily traffic volumes.

Table 3

Association between air pollution measures (static and dynamic) and metabolic disorders.

	HOMA-IR	Diabetes	Hypertension	Obesity	Dyslipidemia	MetS
	β (95%CI)	RR (95%CI)	RR (95%CI)	RR (95%CI)	RR (95%CI)	RR (95%CI)
Static exposure (1600m buffer)						
$PM_{2.5}$	0.02 (−0.06–0.10)	1.13 (0.81–1.57)	0.90 (0.77–1.05)	1.01 (0.89–1.15)	0.93 (0.84–1.02)	0.92 (0.84–1.02)
NO_2	0.01 (−0.04–0.06)	0.86 (0.67–1.11)	0.97 (0.81–1.15)	0.90 (0.79–1.02)	1.01 (0.92–1.11)	1.07 (0.98–1.18)
Traffic[a]	0.02 (−0.03–0.07)	1.09 (0.86–1.39)	0.87 (0.75–1.02)	1.00 (0.89–1.13)	1.03 (0.94–1.12)	0.97 (0.88–1.07)
Dynamic exposure (KDE)						
$PM_{2.5}$	0.04 (—0.01–0.10)	1.02 (0.77–1.35)	1.06 (0.91–1.24)	**1.23 (1.10–1.37)**	**1.12 (1.03–1.22)**	**1.17 (1.07–1.28)**
NO_2	**0.08 (0.03–0.13)**	1.05 (0.83–1.31)	1.05 (0.90–1.22)	**1.21 (1.10–1.33)**	**1.14 (1.06–1.22)**	**1.21 (1.12–1.30)**
Traffic[a]	0.03 (—0.02–0.08)	1.10 (0.88–1.39)	0.96 (0.81–1.15)	1.05 (0.95–1.16)	1.06 (0.98–1.13)	1.02 (0.92–1.12)

$PM_{2.5}$: fine particulate matter (ug/m^3), NO2: nitrogen dioxide (ug/m^3).

HOMA-IR: Homeostatic Model Assessment of Insulin Resistance; MetS: Metabolic Syndrome.

Models were adjusted for sex, age, Hispanic/Latino ethnicity, income, smoking status, and BMI (except for models with obesity and MetS as outcome).

[a]Measured with average daily traffic volumes.