

Draft Final Report

Air Pollution and Noise Exposures to Residents in San Joaquin Valley Communities

Agreement Number 20RD012

Principal Investigator: Elizabeth Noth, PhD, MPH
School of Public Health
University of California, Berkeley

Co-Principal Investigator: Asa Bradman, PhD, MS
Department of Public Health
University of California, Merced

January 20265

Prepared for the California Air Resources Board (CARB)
California Environmental Protection Agency

Report Authors

Rosemary Castorina, PhD; Asa Bradman, PhD; Kimberly Valle, PhD;
Aditya Simha, MPH; Marley Zalay, MPH; Neda Rafiee Jolodar, MSc;
Adriana Espinosa, Reykjavik Salvador, Elizabeth Noth, PhD

Berkeley Exposure Assessment Research (BEAR) Laboratory
Center for Environmental Research and Community Health (CERCH)
University of California, Berkeley
Berkeley, CA 94704

Department of Public Health
Health Sciences Research Institute
University of California, Merced
Merced, CA 95343

Disclaimer

The statements and conclusions in this report are those of the contractor and not necessarily those of the California Air Resources Board. The mention of commercial products, their source, or their use in connection with material reported herein is not to be construed as actual or implied endorsement of such products.

Acknowledgements

The authors wish to thank the California Air Resources Board (CARB) for sponsoring this study and especially CARB staff members Jeffery Williams and Pat Wong for ongoing technical and administrative support. We thank Drs. Kazukiyo Kumagai, Jeff Wagner, and Zhong Min-Wang and Ms. Flavia Wang from the California Department of Public Health, Environmental Health Laboratory Branch for guidance on the research and performing the air VOC sample analysis. We also thank Gary Casuccio and Roger West at RJ Lee for conducting the analyses of the UNC passive aerosol passive samples and Dr. Jeff Wagner for his valuable consultation and expertise. Dr. Chelsea Preble assisted with training and deployment of the Aerosol Black Carbon Detectors (ABCDs) for this study and provided guidance on black carbon data processing and results interpretation. We thank our community partners Tim Tyner, Anabelle Garza and Debra Manzo with the Central California Asthma Collaborative (Fresno) and Jermaine Reece, Jazmarie LaTour and Alexis Garcia with Little Manila Rising (Stockton) for their collaboration on the study, participant recruitment and conducting home visit study activities. We also thank Dr. Tracy Thatcher at Cal Poly, San Luis Obispo for advice on strategies to measure noise levels. Finally, we thank all the families that participated in this study, without whom this research would not have been possible.

This Report was submitted in fulfillment of ARB Contract No. 20RD012, “Total Exposures to Air Pollutants and Noise in Disadvantaged Communities”, by the Center for Environmental Research and Community Health (CERCH) and Berkeley Exposure Lab (BEAR), School of Public Health, UC Berkeley and UC Merced Health Sciences Research Institute, under the [partial] sponsorship of the California Air Resources Board. Work was completed as of **December XX, 2026.**

Table of Contents

Acknowledgements	III
Abstract	IX
Executive Summary	X
Background	X
Methods	X
Results	X
Policy Implications	XIII
Body of Report	1
1 Literature Review	1
1.1 Introduction: Disadvantaged Communities in California	1
1.2 Literature Overview	1
1.3 Air Pollution	2
1.4 Noise	7
1.4.1 Noise Exposure Regulatory Context and Mitigation	7
1.4.2 Noise Exposure in Disadvantaged Communities	9
1.4.3. Overview of noise-related health effects	10
1.4.4. Air and Noise Co-exposures	15
1.4.5. Discussion	16
2 Materials and Methods	16
2.1 Research Approach	16
2.1.1 Participant recruitment and consent	16
2.1.2 Overview of 24-hour Indoor and outdoor air monitoring and sampling in Fresno and Stockton	17
2.1.3 Personal Monitoring	18
2.1.4 Data Collection	18
2.2 Methods	20
2.2.1 Real-time Air Monitoring of Criteria Air Pollutants	20
2.2.2 Real-time Black Carbon Monitoring	21
2.2.3 VOCs	21
2.2.3 Formaldehyde	23
2.2.4 Polycyclic Aromatic Hydrocarbons (PAHs)	23
2.2.5 Noise	24
2.2.6 Passive PM sampling	24
2.2.7 Estimated Air Exchange Rates	25
2.3 Questionnaires and Health Outcomes	25
2.4 GeoSpatial Data and Information Sources	26
2.4.1. Geocoding house locations	26
2.4.2. CalEnviroScreen	26
2.4.3. Nearby Residential Traffic Density and Distance to SR-99	26
2.5 Statistical Analyses	27

2.6 Health Risk Characterization	27
2.6.1 Non-Cancer Risk	27
2.6.2 Cancer Risk	28
2.6.3 Noise Exposure	28
3 Results and Discussion.....	28
3.1 Cohort Characteristics	28
3.2 Geospatial Mapping of Study Area	33
3.3 Air pollutant concentrations and noise	34
3.3.1 Criteria Air Pollutants (PM _{2.5} , CO, NO ₂ and O ₃).....	34
3.3.2 Black Carbon	50
3.3.3 VOCs.....	61
3.3.4 Formaldehyde.....	69
3.3.5 PAHs.....	69
3.3.6 Noise.....	71
3.3.7 Passive PM Sample Results	80
3.4 Health Risk Characterization	85
3.4.1 Non-Cancer Risk Estimation.....	85
3.4.2 Cancer Risk Evaluation.....	86
3.4.3 Noise Exposure Hazard Evaluation.....	90
4 Opportunities and Challenges of Community Engaged Research	90
5 Strengths and Limitations.....	91
6 Summary and Conclusion.....	92
7 Recommendations	97
8 References	101
9. Glossary of Terms, Abbreviations, and Symbols.....	114

Tables

Table 1: Fresno County Municipal Code 8.40.040 - Exterior Noise Standards	9
Table 2: Summary of SPHERE Sample Collection from 64 Participating Households (Fresno and Stockton)	19
Table 3: Twenty-two VOCs measured in air (from 16 Fresno homes)	22
Table 4: Characteristics of adult participants (64 adult/child pairs)	30
Table 5: Characteristics of child participants (64 adult/child pairs).....	31
Table 6: Adult and child respiratory health characteristics (n=64 pairs).....	32
Table 7: Percentage of each day in each microenvironment or performing exposure-related activity for 21 participants	33
Table 8: Indoor and outdoor criteria pollutant concentrations and comparison to NAAQS standards.	35
Table 9: Association between indoor PM _{2.5} (µg/m ³) levels and CalEnviroScreen (CES) traffic indicators and residence in a AB 617 community.	36
Table 10: Association between outdoor PM _{2.5} (µg/m ³) levels and CalEnviroScreen (CES) traffic indicators and residence in an AB 617 community.	37
Table 11: Association between indoor and outdoor PM _{2.5} and Daily Vehicle Miles Traveled ^a (DVMT).....	38
Table 12: Correlation between Indoor PM _{2.5} and Residential Proximity (meters) to Highway SR-99.	38
Table 13: Correlation between outdoor PM _{2.5} and Residential Proximity (meters) to highway SR-99.....	39
Table 14: Indoor PM _{2.5} concentrations (µg/m ³) stratified by household characteristics (n=54).....	39
Table 15: Outdoor PM _{2.5} concentrations (µg/m ³) and household characteristics (n=54).....	40
Table 16: Summary of 24-hour average PM _{2.5} indoor, outdoor, and personal concentrations (µg/m ³).....	41
Table 17: Associations between indoor and outdoor PM _{2.5} concentrations and adult respiratory symptoms.	41
Table 18: Associations between indoor and outdoor PM _{2.5} concentrations and child respiratory symptoms.	42
Table 19: Association between indoor NO ₂ (ppb) levels and CalEnviroScreen (CES) traffic indicators and residence in a AB 617 community.	43
Table 20: Association between outdoor NO ₂ (ppb) levels and CalEnviroScreen (CES) traffic indicators and residence in a AB 617 community.	44
Table 21: Correlation between indoor and outdoor NO ₂ and residential proximity (meters) to highway SR-99.	44
Table 22: Indoor NO ₂ concentrations (µg/m ³) stratified by household characteristics (n=54).....	45
Table 23: Outdoor NO ₂ concentrations (µg/m ³) and potential determinants of exposure (n=54).....	46
Table 24: Associations Between Indoor and Outdoor NO ₂ Concentrations and Adult Respiratory Symptoms.	47
Table 25: Associations Between Indoor and Outdoor NO ₂ Concentrations and Child Respiratory Symptoms.	47
Table 26: Indoor O ₃ levels (ppm) and stratified by CalEnviroScreen (CES) traffic indicators and residence in an AB 617 community.	48
Table 27: Outdoor O ₃ levels (ppm) stratified by CalEnviroScreen (CES) traffic indicators and residence in an AB 617 community.	48
Table 28: Associations between indoor and outdoor O ₃ concentrations and adult respiratory symptoms.	49
Table 29: Child Respiratory Health Characteristics and O ₃ concentrations.	50
Table 30: Summary of 24-hour average indoor and outdoor black carbon levels (µg/m ³).	50
Table 31: Indoor black carbon concentrations (µg/m ³) and household characteristics (n=54).....	51
Table 32: Outdoor black carbon concentrations (µg/m ³) and household characteristics (n=44).....	52
Table 33: Summary of black carbon indoor-to-outdoor (I/O) ratios.	52

Table 34: Association between indoor 24-hour black carbon levels ($\mu\text{g}/\text{m}^3$) and CalEnviroScreen (CES) traffic indicators and residence in a AB 617 community.	53
Table 35: Association between outdoor 24-hour black carbon levels ($\mu\text{g}/\text{m}^3$) and CalEnviroScreen (CES) traffic indicator and residence in AB 617 community.	55
Table 36: Associations between indoor and outdoor black carbon and traffic (Daily Vehicle Miles Traveled (DVMT)).....	56
Table 37: Correlation between indoor black carbon and residential proximity (meters) to highway SR-99.	57
Table 38: Correlation between outdoor black carbon and residential proximity (meters) to highway SR-99.	57
Table 39: Spearman correlations between 24-hour average indoor air pollutant concentrations.	60
Table 40: Spearman correlations between 24-hour average outdoor air pollutant concentrations.	61
Table 41: Spearman correlations between 24-hour average indoor air pollutant concentrations and estimated air change rate (air changes/hour).	61
Table 42: Summary of indoor air VOC concentration measurements ($\mu\text{g}/\text{m}^3$) (n= 23 samples collected from 16 homes in Fresno, CA).	62
Table 43: Summary of outdoor air VOC concentration measurements ($\mu\text{g}/\text{m}^3$) (n= 8 samples collected from 8 homes Fresno, CA).	64
Table 44: OEHHA acute, 8-hour and chronic reference exposure levels (RELs) and U.S. EPA reference concentrations (RfCs).	65
Table 45: Correlations between outdoor BTEX levels and Residential Proximity (km) to Highway SR-99.	67
Table 46: Summary of indoor and outdoor TVOC concentrations (ppm) (n=46 SPHERE participant homes in Fresno, CA) ^a	68
Table 47: Summary of indoor formaldehyde concentrations ($\mu\text{g}/\text{m}^3$) from 24 homes	69
Table 48: Indoor air PAH concentration (ng/m ³) and indoor-to-outdoor (I/O) ratios.	70
Table 49: Outdoor Air PAH concentration (ng/m ³).	70
Table 50: Personal, indoor and outdoor noise measurements (dBA) averaged for day, night and over 24 hours.	72
Table 51: Associations between indoor and outdoor noise and nearby traffic (daily vehicle miles traveled (DVMT)).....	75
Table 52: Correlation matrix of indoor air pollutants and noise.	76
Table 53: Correlation matrix of outdoor air pollutants and noise.	77
Table 54: Reported Noise Sensitivity (n=64)	78
Table 55: Parent perception of environmental noise.	79
Table 56: Association Between Indoor Noise Exposure and Child Behavioral and Academic Outcomes.	80
Table 57: Comparison of indoor BTEX, formaldehyde, and naphthalene concentrations with health-based reference values.a.....	85
Table 58: Comparison of outdoor BTEX and naphthalene concentrations with health-based reference values (OEHHA RELs and U.S. EPA RfCs).a,b,c.....	86
Table 59: Indoor hazard ratios comparing adult women VOC exposure to OEHHA No Significant Risk Levels (NSRLs) (mean age: 42 years[XW)	87
Table 60: Outdoor hazard ratios comparing adult women VOC exposure to OEHHA No Significant Risk Levels (NSRLs) (mean age: 42 years).a, b.....	87
Table 61: Indoor hazard ratios comparing male child VOC exposure to OEHHA No Significant Risk Levels (NSRLs) (mean age: 9 years).a.....	88
Table 62: Outdoor hazard ratios comparing male child outdoor VOC exposures to OEHHA No Significant Risk Levels (NSRLs) (Mean Age: 9 years).a,b.....	88

Table 63: Indoor hazard ratios comparing female child VOC exposures to OEHHA No Significant Risk Levels (NSRLs) (mean age: 9 years).a.....	89
Table 64: Outdoor hazard ratios comparing female child VOC and naphthalene exposure to OEHHA No Significant Risk Levels (NSRLs) (mean Age: 9 years).a,b	89

Figures

Figure 1: CalEnviroScreen Community Scores for the San Joaquin Valley. Areas in red are at higher risk.	1
Figure 2: Department of Transportation (DOT) Noise Maps for Stockton, CA (Top) and Fresno, CA (Bottom).....	Error! Bookmark not defined.
Figure 3: The traditional definition of noise is unwanted or disturbing sound. “Sound becomes unwanted when it interferes with normal activities such as sleeping or conversation or generally diminishes one’s quality of life.”	10
Figure 4: Overview of SPHERE Environmental Sampling Platform	17
Figure 5: LEFT: Location of Fresno participant homes relative to the AB 617 boundary (in green), RIGHT: Location of Fresno participant homes relative to census tracts and CES sources.....	33
Figure 6: LEFT: Location of Stockton participant homes relative to the AB 617 boundary (in green), RIGHT: Location of Stockton participant homes relative to census tracts and CES scores.	34
Figure 7: Indoor 24-hour black carbon levels ($\mu\text{g}/\text{m}^3$) and CalEnviroScreen (CES) overall cumulative impact scores and AB 617 location.....	54
Figure 8: Outdoor 24-hour black carbon level ($\mu\text{g}/\text{m}^3$) and CalEnviroScreen (overall cumulative impact score) and AB 617 location.	56
Figure 9: Indoor black carbon concentrations by season (n=59 total measurements) (Fresno (n=48) and Stockton (n=11)). (Spring n=20; Summer n=25; Fall n=14).....	58
Figure 10: Indoor black carbon concentrations during the warm months (May - October) stratified by central air conditioning (AC) in the home (No (n=9); yes (n=35)).....	59
Figure 11: Box plot of outdoor black carbon concentrations by season (n=50 total measurements (Fresno (n=42) and Stockton (n=8))).....	60
Figure 12: Indoor and outdoor air concentrations of most frequently detected VOCs.	65
Figure 13: Ratios of BTEX Indoor-to-Outdoor concentrations (n=8 matched pairs).....	66
Figure 14: Comparison of summed indoor (n=23) and outdoor (n=8) air VOC concentrations (ppm)	68
Figure 15: Comparison of indoor and outdoor naphthalene concentrations (ng/m^3).....	71
Figure 16: Histograms of noise measurements collected during the day, night and 24-hours.....	74
Figure 17: Indoor passive PM sample results by particle composition category (20 samples collected from 8 Fresno homes).....	81
Figure 18: Outdoor passive PM sample results by particle composition category (16 samples collected from 8 Fresno homes).....	82
Figure 19: Indoor passive PM sample results by particle composition category (11 samples collected from 5 Merced homes).	83
Figure 20: Outdoor passive PM sample results by particle composition category (10 samples collected from 5 Merced homes).	84

Abstract

Many California residents in disadvantaged communities (DACs) experience disproportionate exposure to multiple environmental stressors. We assessed combined air pollution and environmental noise in 64 San Joaquin Valley households (each with at least one adult and a child aged 4-13 years) using 24-hour indoor and outdoor measurements of fine particulate matter (PM_{2.5}), nitrogen dioxide (NO₂), ozone (O₃), black carbon (a diesel-related pollutant), polyaromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), indoor formaldehyde, and noise. We also collected information about housing and participant activities and health information by questionnaire. Outdoor PM_{2.5} rarely exceeded U.S. EPA federal 24-hour standards, but the maximum 24-hour level (36.9 µg/m³) was higher than the 35 µg/m³ National Ambient Air Quality Standard (NAAQS) and the World Health Organization (WHO) 24-hour guideline (15 µg/m³). Indoor PM_{2.5} and in some cases NO₂ matched or exceeded outdoor levels, consistent with contributions from indoor sources (e.g., cooking) in addition to infiltration, underscoring the importance of indoor environments in determining overall PM_{2.5} exposure. Relative to other areas, Assembly Bill (AB) 617-designated communities exhibited higher average indoor PM_{2.5} (average=22.1 vs. 9.9 µg/m³), NO₂ (16.8 vs. 14.6 ppb), and black carbon (0.56 vs. 0.33 µg/m³) concentrations. Notably, homes located closer to State Route 99, a major transportation corridor, had significantly higher indoor PM_{2.5}, NO₂, black carbon, and VOC (BTEX: benzene, toluene, ethylbenzene and xylenes) levels; outdoor levels for all these pollutants were also higher, with statistically significant associations for NO₂ and most BTEX compounds, indicating strong traffic influences. Indoor formaldehyde, a respiratory irritant and carcinogen, was detected in 81% of homes, frequently exceeding state and federal reference levels. Noise exposures were consistently elevated, with 24-hour averages ~60 dBA, daytime peaks >65 dBA, and nighttime levels often >50 dBA, levels associated with sleep disturbance and stress. Together, these results underscore the cumulative environmental burdens and exposure disparities in DACs and will inform policies and practical measures to mitigate pollution and noise in vulnerable communities. By guiding interventions, such as accelerating clean transportation, improving home ventilation and filtration (e.g., MERV-13+ HVAC and effective range hoods), and integrating noise mitigation into housing renovation and community planning, this research supports efforts by California state agencies, including the Air Resources Board, the Department of Transportation, the Department of Public Health, Office of Environmental Health Hazard Assessment and the Department of Toxic Substances and Control, to protect public health and advance strategies to reduce pollution and environmental noise in vulnerable communities.

Executive Summary

Background

Many California communities face disproportionate exposure to air pollution and noise, contributing to poorer health outcomes. This study focused on disadvantaged communities (DACs) in the San Joaquin Valley (SJV) and aimed to assess indoor and outdoor air pollutant and noise exposures, understand exposure sources and potential health risks, and inform policies to reduce exposures. The project addressed knowledge gaps by examining how factors like household behaviors (cooking, cleaning, smoking), building characteristics, and proximity to traffic affect personal and total pollutant exposures in these overburdened communities. It is among the first studies in California to measure indoor and outdoor noise levels alongside air quality, enabling a broader look at cumulative environmental exposures.

Methods

Participants were recruited by community partners in Fresno (Central California Asthma Collaborative) and Stockton (Little Manila Rising). A total of 64 households participated, each with at least one adult and one child (4–13 years old). Field monitoring was conducted from February to November 2023 over single 24-hour periods in participants' homes to capture a snapshot of daily exposure. Indoor and outdoor air pollutant concentrations were measured using real-time sensors and integrated sampling methods. Key pollutants measured included fine particulate matter (PM_{2.5}), ozone (O₃), nitrogen dioxide (NO₂), total volatile organic compounds (TVOCs), black carbon (BC - a marker of diesel exhaust particles), and noise levels. Active sampling was used to collect air samples for laboratory analysis of specific volatile organic compounds (VOCs) and polycyclic aromatic hydrocarbons (PAHs). In addition, passive sampling methods that required no powered equipment were used to measure formaldehyde concentrations and obtain particulate matter concentration and particle composition information in selected homes. Participants also completed questionnaires on home characteristics, activities, and health, and a subset of adults carried personal monitors to track their individual exposure to PM_{2.5}, TVOCs, and noise during daily activities. This multi-faceted approach provided a rich dataset on indoor air quality (IAQ), outdoor pollution, and noise in DAC households.

Results

Determinants of Air Pollutants

Mean 24-hour PM_{2.5} concentrations were 14.8 µg/m³ indoors, 11.9 µg/m³ outdoors, and 12.1 µg/m³ for personal exposures. The indoor-to-outdoor ratios (I/O) for PM_{2.5} averaged 2.3, indicating that household activities such as cooking, heating, smoking, burning candles, or the use of other combustion appliances may have contributed more to indoor PM_{2.5} than outdoor infiltration. Mean (range) indoor black carbon concentrations were 0.42 µg/m³ (0.05 - 1.51), while outdoor concentrations averaged 0.65 (0.10 - 1.95) µg/m³. On average, indoor black carbon concentrations were approximately 80% of outdoor levels, suggesting that outdoor air is the primary source of indoor BC contamination, and that the home envelope provides a partial barrier to BC infiltration.

Homes in census tracts with high overall CalEnviroScreen (CES) scores (≥75th percentile) had significantly higher indoor PM_{2.5} concentrations compared with homes in lower CES score tracts (mean=17.9 versus 6.7 µg/m³, respectively). Similarly, homes in census tracts with high overall CES scores (≥75th percentile) had significantly higher indoor black carbon concentrations compared with homes in lower CES score tracts (mean=0.53 versus 0.21 µg/m³, respectively). We observed similar patterns when examining residence inside or outside AB 617-designated communities. Renters and lower-income households (< \$30,000 annual income) also experienced higher indoor particulate matter.

We found similar results for outdoor PM_{2.5} and black carbon (BC) concentrations. Homes in census tracts with high overall CES scores (≥75th percentile) compared with homes in lower CES score tracts had significantly higher outdoor PM_{2.5} concentrations (mean=12.7 versus 7.4 µg/m³, respectively). Similarly, homes in census tracts with high overall CES scores (≥75th percentile) had significantly higher outdoor black carbon concentrations compared with homes in lower CES score tracts (mean=0.74 versus 0.47 µg/m³, respectively). We observed similar patterns when examining residence inside or outside AB 617-designated communities. Black carbon (BC) concentrations were positively and significantly correlated with the concentrations of other combustion products, including PM_{2.5}, NO₂ and CO.

The findings for particulate matter and BC suggest that communities with higher CES environmental burden scores and/or AB 617 designation in our study region experience elevated particle exposures. These findings are notable because they provide on-the-ground air quality measurements that validate the CES burden scoring system, which are largely based on emission inventories and modeling, not direct measurement of pollutants, and underscore ongoing environmental justice concerns and the need for remediation of high air pollution exposures in targeted populations.

Overall, proximity to heavy traffic was linked to higher outdoor particulate matter exposure in these communities. Notably, indoor PM_{2.5}, NO₂, and BC concentrations were inversely correlated with proximity to State Route-99 (SR-99) (r=-0.47, -0.57, and -0.42, respectively), indicating concentrations were higher in homes closer to the highway. Outdoor BC and NO₂ were also higher, albeit not always statistically significantly, among homes closer to SR-99. Homes located closer to SR-99 also experienced higher outdoor concentrations of benzene, toluene, ethylbenzene, and xylenes (BTEX) compared to homes farther away. Overall,

these findings indicate traffic emissions are a continuing burden on DAC neighborhoods and aligns with community concerns about emissions from transportation corridors

Indoor air concentrations of nearly all measured volatile organic compounds (VOCs) were higher than outdoor levels, often by a factor of 2–3 or more. Frequently detected compounds such as toluene and xylenes showed substantially elevated indoor concentrations compared to outdoors (mean indoor-to-outdoor (I/O) ratios ranged from 2.4 to 6.3), indicating strong indoor sources like household products or activities.

Using passive 24-hour samplers, formaldehyde was detected in 81% of homes sampled, with a median indoor concentration of 18 $\mu\text{g}/\text{m}^3$. This median exceeds health-based reference levels established by OEHHA and U.S. EPA (9 $\mu\text{g}/\text{m}^3$), highlighting formaldehyde as a continuing indoor pollutant of concern.

Naphthalene was the most prevalent PAH measured, with mean concentrations of 45 ng/m^3 indoors and 26 ng/m^3 outdoors, highlighting additional indoor sources. Three of the four PAHs measured (fluorene, naphthalene and phenanthrene) had mean I/O ratios ≥ 1 , suggesting indoor sources.

Environmental Noise Exposure

Average 24-hour sound levels were generally around 60 dBA, with daytime outdoor peaks near 70 dBA and nighttime levels often above 50 dBA.¹ These values in some instances exceeded thresholds associated with sleep disturbance, stress, and cardiovascular effects. Noise measurements were not significantly correlated with any measures of air quality. Higher traffic density within 100 m of participant homes was significantly associated with higher outdoor noise; indoor noise did not show meaningful relationships with nearby traffic density.

Individual perception and sensitivity to noise is an important component of the impact of environmental noise on health. Noise was a common concern among households, with about one third of adult participants reporting annoyance from indoor noise and more than half disturbed by outdoor noise heard while indoors. Most adult participants characterized their neighborhoods as moderately or very quiet, although 23% reported them as moderately or very loud. The few households that had filed noise complaints complained about neighbors or, in one case, heavy duty trucks. Children living in homes with higher average indoor noise levels were more likely to have reported academic challenges or diagnosed learning disabilities. These analyses were limited by small sample size and short-term exposure assessment assessed against learning difficulties that may take years to develop but are suggestive and warrant future research.

Risk Evaluation

Formaldehyde was detected in 81% of homes, with a median concentration of 18 $\mu\text{g}/\text{m}^3$. These levels exceeded California and U.S. EPA health-based reference levels (9 $\mu\text{g}/\text{m}^3$). We also calculated formaldehyde, benzene, naphthalene, and ethylbenzene inhalation exposure estimates for women and children (4 to 13 yrs) and compared them to OEHHA Proposition 65

No Significant Risk Levels (NSRLs) (The NSRL represents a chronic exposure intake with potential cancer risks exceeding one in 100,000 (10^{-5})). Corresponding hazard ratios (HRs) were calculated as the ratio of estimated exposure to the NSRL and therefore represent potential cancer risk. Assuming concentrations are reflective of long-term averages, indoor formaldehyde exposure estimates exceeded NSRLs for all groups (median HRs ranged from 5.6 to 7.1; and 95th percentile HRs ranged from 16.2 to 21.2). If these single day measurements are reflective of long-term averages, estimated outdoor benzene exposures would exceed the NSRL at the 95th percentile for adults (HR = 1.5) and children (HR=1.2-1.3). Estimated naphthalene and ethylbenzene exposures, based on indoor and outdoor concentrations, were well below NSRLs for all groups (HRs \leq 0.5). Mitigation priorities include reducing indoor formaldehyde through low-emitting materials, source control, and ventilation/filtration, and possibly mitigating outdoor benzene via traffic-related controls and improved building envelope/filtration in highly impacted communities.

Policy Implications

This study generated new information on indoor and outdoor air quality in homes and the disadvantaged communities in the San Joaquin Valley. CARB can use this information in developing guidance or crafting regulations to better protect residents' health. For example, the strong role of indoor VOC sources points to the benefit of policies promoting healthier low-VOC building materials and household products and improving home ventilation and filtration systems. The elevated formaldehyde concentrations observed suggest a need for updated building codes or product standards to limit formaldehyde emissions in homes and for further evaluation of the CARB Composite Wood Products Airborne Toxic Control Measure (Section 93120-93120.12, Title 17, California Code of Regulations).

Our finding that exposures to traffic-related air pollution (TRAP) in communities were higher in AB 617 selected communities or in census tracts with high CalEnviroScreen scores indicates that more work and monitoring is needed to reduce exposure disparities. Recent studies have shown that despite overall reduction in traffic-related emissions, exposure disparities persist.² Additional steps to reduce vehicle emissions and tools such as buffer zones (setbacks) or barriers near highways may help reduce TRAP exposures. More broadly, strategies such as decarbonization and electrification of home appliances (to minimize indoor combustion), deployment of high-efficiency air filters, and community-level interventions under AB 617 (e.g., rerouting truck traffic, urban tree planting, or sound walls) are supported by this study's findings. By elucidating how and why indoor and outdoor air quality differs in overburdened communities, this research can help inform evidence-based policies and community actions to improve air quality and reduce environmental health disparities in the San Joaquin Valley.

Recommendations

Based on the study's findings, the following priority actions are recommended to reduce air pollution and noise exposures in disadvantaged communities (DACs):

- (i) Targeting traffic pollution in impacted areas by reducing emissions from heavy-duty traffic and freight operations in DAC regions. Traffic and diesel controls include accelerated zero-emission truck/bus deployment, strict anti-idling enforcement, freight rerouting, and “clean-air zones,” supplemented by roadside filtration barriers and strategic urban greenery to cut traffic related air pollutants and noise at the source.
- (ii) Improve indoor air quality by reducing indoor combustion and high-emitting materials (e.g., formaldehyde sources), and upgrading ventilation and air cleaning for particles and gases. Maintaining MERV-13+ HVAC filtration, using effective kitchen range hoods, and deploying portable air cleaners are potential strategies to reduce indoor particles. To address gaseous pollutants such as formaldehyde, benzene, and other VOCs, air cleaning technologies such as activated carbon or chemisorbent filters, should be considered in conjunction with HEPA filtration.
- (iii) Mitigate environmental noise exposure by constructing sound walls/earth berms along highways and freight routes, enforcing nighttime noise ordinances (restricting heavy trucks/construction), adopting quieter pavement technologies and vehicle designs, requiring buffer zones or setbacks for new housing developments near highways and industry, and expand weatherization programs including building insulation and double-paned windows to reduce noise infiltration and ensure benefits reach rental and low-income housing. Finally, incorporate noise as a CalEnviroScreen environmental indicator.
- (iv) Enhance community capacity & planning. Sustain community-engaged hyper-local monitoring (air and noise) and use the information to target interventions and assess progress of exposure-reduction policies and to strengthen environmental-health literacy through accessible guidance, training, and partnerships with local community-based organizations.

Implemented together, these measures will reduce cumulative air pollutant and noise exposures and improve indoor environmental quality across the San Joaquin Valley.

Future Research Needs

Additional research is warranted to expand on the findings of this study:

- Our findings indicate higher traffic-related air pollution exposure in AB 617 and DAC communities and underscores the continuing need to assess exposure disparities in these communities with field monitoring data. This information is essential to evaluate the success of ongoing community exposure-reduction plans. The emergence of low-cost sensors can provide hyper-local spatial resolution that can complement regulatory monitoring. CARB is already supporting community monitoring efforts, especially for

PM_{2.5}. Importantly, new low-cost (<\$500) monitors are being developed that can monitor NO₂ and ozone. Costs for black carbon monitors are also declining. Expanded community-based monitoring for these TRAP pollutants would provide additional information on health risks, the validity of the CalEnviroScreen pollutant burden ranking system, and the efficacy of exposure-reduction policies.

- Our findings for formaldehyde are consistent with other indoor air quality studies that identify formaldehyde as a contaminant of concern. Future research should further evaluate the efficacy of CARB's 2007 composite wood product regulations and evaluate other formaldehyde and VOC sources.
- This is the first California study to collect measurements and questionnaire-based information on community noise exposures. More work is needed to understand noise exposure and potential health impacts in our state, especially on children's development and school performance.
- More work is needed to understand exposure to BTEX VOCs. Specifically, wider geographic monitoring is needed to understand the spatial variability of BTEX exposures, including areas outside AB 617 and DAC communities and away from major transportation corridors.

Conclusions

In summary, this study demonstrates that SJV DAC households face elevated exposure to multiple harmful pollutants and noise, both indoors and outdoors. The combined influence of noise exposure, traffic-related air pollution, indoor air pollutant sources, and housing characteristics contributes to cumulative exposure burdens. These results support ongoing programs aiming to reduce exposures at both the household and community level to protect health and advance environmental equity.

Body of Report

1 Literature Review

1.1 Introduction: Disadvantaged Communities in California

California Senate Bill 535 (SB 535) designates vulnerable communities as “disadvantaged communities (DACs)” if they rank in the top 25% based on CalEnviroScreen (CES), a geographically-based mapping tool that scores communities by potential exposure, vulnerability, and social-economic indicators.^{3,4} Nearly all of the San Joaquin Valley (SJV) meets this threshold (**Figure 1**). The SJV has historically experienced some of the nation’s worst air quality, often exceeding health-based air quality standards for ozone (O₃) and particulate matter.⁵

In addition to air pollution, DACs are frequently exposed to environmental noise from traffic, aircraft, industrial operations, power generation, and construction.⁶ Anthropogenic noise, defined as “unwanted or disturbing sounds”, is one of the most common environmental exposures in the U.S.⁶ The health impacts of noise include sleep disturbance, cardiovascular disease, endocrine disruption, increased diabetes risk, impaired academic performance, heightened stress, and behavioral effects in children.⁶

1.2 Literature Overview

We reviewed literature examining air pollution and noise exposures and their health impacts, with a particular focus on California when information was available. Literature on air pollution and noise exposure focused on disparities related to socioeconomic status, CES scores, historical information on pollution trends by region, the impact of regulatory approaches to reduce disparities, and the impacts of personal behaviors and external factors on air pollution exposures. Literature was identified through searches of medical and public health databases including PubMed, as well as web searches to identify statements and other reports by governments or other authorities.

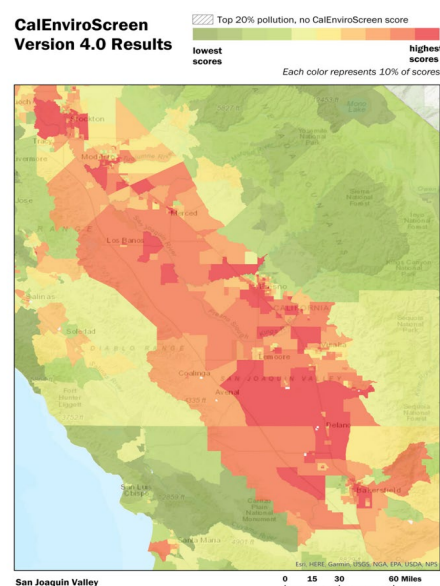


Figure 1: CalEnviroScreen Community Scores for the San Joaquin Valley. Areas in red are at higher risk.

1.3 Air Pollution

1.3.1. Disparities in Air Pollution Exposures and Health Impacts

The U.S. Environmental Protection Agency (EPA) established National Ambient Air Quality Standards (NAAQS) for six criteria pollutants known to harm human health and the environment: carbon monoxide (CO), lead (Pb), nitrogen dioxide (NO₂), ozone (O₃), particulate matter (PM_{2.5} and PM₁₀), and sulfur dioxide (SO₂).⁷ In California, particularly the San Joaquin Valley (SJV), the primary pollutants of concern are NO₂, O₃, and PM_{2.5}. Sources of these pollutants include traffic-related air pollution (TRAP), industrial facilities, agriculture, and rail, aviation and maritime activities, as well as community and household sources such as gas stations, restaurants, cooking, and fireplaces. Indoor pollutants that are not regulated under NAAQS—such as volatile organic compounds (VOCs) from building materials and furnishings—are often found at higher levels indoors than outdoors.⁸

Socioeconomic disadvantages and race are strong determinants of the disparities in air pollution exposure and health impacts observed in DACs in California and nationally. For example, Bennett et al. (2019) showed that counties with higher poverty experienced greater reductions in life expectancy for each unit increase in PM_{2.5}.⁹ Similarly, Perlin et al. (2001) found that African American children under age five in poor households were significantly more likely to live near industrial sources of air pollution compared with White children.¹⁰ Studies have also shown that Hispanics, particularly those with low income, are at increased risk of hazardous air pollutant exposures.¹¹ Analyses of VOC exposures (benzene, toluene, xylenes, chloroform, and others) indicate consistent associations between higher exposures and lower income or minority race/ethnicity.¹²

Residential segregation reinforces these disparities. Woo et al. (2019) found that ethnic minority communities are consistently exposed to higher concentrations of pollutants such as NO₂, PM₁₀, and PM_{2.5}.¹³ In California, the San Joaquin Valley is frequently ranked among the worst in the nation for air quality and often fails to meet federal and state standards.⁵ Other regions in the state show similar patterns: Molitor et al. (2011) demonstrated that higher NO₂, PM_{2.5}, and diesel exposures were associated with higher poverty counts; Winter et al. (2019) found that O₃ levels in Los Angeles parks were significantly higher in disadvantaged communities; and Stewart et al. (2020) reported that 14% of schools in Santa Clara County faced potentially high roadway pollution exposure, with economically disadvantaged students disproportionately attending those schools.^{14–16} In Worcester, MA, neighborhood-level NO₂ concentrations were inversely related to socioeconomic status, with poorer areas having significantly higher NO₂ levels.¹⁷ Even within homes, exposure can vary by housing type and associated demographics. For example, a study of children with asthma reported that in multifamily housing units (which are often more affordable and densely occupied), indoor NO₂ levels (even below 53 ppb, the federal standard) were associated with increased respiratory symptoms.¹⁸ These findings suggest that where and how people live (e.g., in high-density, lower-income housing) can lead to higher exposure to combustion pollutants like NO₂, exacerbating health problems in vulnerable groups.

Policy interventions have improved overall air quality in California. By 2013, all regions met state and federal standards for CO, NO₂, and SO₂.¹⁹ Significant progress has also been made in reducing O₃ and PM_{2.5} exposures, particularly through regulations on diesel and tailpipe emissions and restrictions on agricultural burning. Still, challenges persist in the South Coast and San Joaquin Valley. To address persistent disparities, the state established the Community Air Protection Program (CAPP) under AB 617 to reduce emissions and exposures in the most impacted communities.^{20,21} Additionally, CARB adopted the *Vision for Racial Equity* in 2023 to explicitly address environmental injustice.

Hazardous air toxics emitted from industries also disproportionately affect disadvantaged communities. A study in Maine found that lower-income census block groups faced higher estimated cancer and health risks from toxic air emissions, reinforcing income inequality through environmental exposure.²² Likewise, qualitative research in a Houston, Texas community documented that low-income Hispanic households tended to live closer to sources of hazardous air pollutants (HAPs), resulting in greater cancer risk from these toxic exposures.¹¹ Disproportionate exposure has been observed for volatile organic compounds (VOCs) and other organic pollutants in many regions. For example, a study assessing personal VOC exposures in three different study areas (Kanawha Valley in West Virginia, the Baton Rouge-New Orleans Corridor in Louisiana, greater Baltimore) found that factors such as race and income were significantly associated with higher exposure to certain VOCs, including chloroform, 1,4-dichlorobenzene, and tetrachloroethene,¹² indicating that people of color and low-income individuals often have greater contact with these harmful chemicals, likely due to a combination of environmental circumstances (e.g., proximity to chemical sources) and personal product use patterns. In another example, an investigation of several New Jersey communities with known air pollution issues showed that living in these hotspot areas poses heightened risks from ambient air toxics, contributing to health disparities in those disadvantaged neighborhoods.²³

Non-criteria pollutants such as polycyclic aromatic hydrocarbons (PAHs) have drawn attention for similar reasons. PAHs (a class of organic compounds from sources like vehicle exhaust, industrial processes, and wildfires) can deposit into soil and dust, later finding their way into the food chain. A recent literature review noted that long-term PAH exposures are associated with persistently elevated blood pressure and higher incidence of hypertension in exposed populations; moreover, PAH contamination in soil can taint locally grown vegetables and fruits, providing an additional exposure route in communities near pollution sources.²⁴ Health data also link PAH exposure to respiratory outcomes: an analysis of U.S. adults found a positive association between biomarkers of PAH exposure and the prevalence of current asthma.^{25,27} While PAH concentrations have decreased in the SJV over the last 25 years, they still remain elevated compared to other regions.²⁶

Diesel particulate matter and black carbon are also important pollutants of concern. Diesel PM emissions declined 37% statewide between 2000 and 2010 due to new controls and cleaner engines, and black carbon emissions have decreased by 90% from the 1960s to the 2010s.^{20,27} More recent studies show continuing reductions in emissions and exposures related to fossil fuel combustion.^{2,28} These improvements reflect decades of regulatory success, though exposure disparities persist and remain disproportionately high in communities near major transportation corridors.² Similarly, VOC emissions decreased

significantly from 2000 to 2015 but have since plateaued, with the San Joaquin Valley and Sacramento Metropolitan Area showing disproportionately high emissions from area-wide sources.¹⁹

1.3.2 Disparities in Air Pollution Exposures Based on CalEnviroScreen Scores

California studies consistently show that neighborhoods with high CalEnviroScreen scores, often low-income communities of color, experience disproportionately high air pollution exposures. For example, an analysis in Los Angeles County demonstrated that areas of greater poverty had higher estimated concentrations of ambient NO₂ and PM_{2.5}.¹⁴ Although the relationship was complex, the general trend aligns with CalEnviroScreen's identification of poverty as a factor associated with cumulative pollution burden. Another study in Southern California directly measured personal exposure: researchers tracked 24-hour PM_{2.5} exposures for individuals and found that those living in low-socioeconomic status neighborhoods had elevated exposure levels compared to people in more affluent areas.²⁹

In Santa Clara County (SF Bay Area), a spatial analysis of school sites found that many schools were located in socioeconomically disadvantaged and high-traffic areas.¹⁶ These schools, many of which serve lower-income, diverse student populations, are located near busy highways or trucking routes, a scenario common in high CalEnviroScreen-score census tracts. Thus, children in these communities are more likely to inhale traffic-related pollutants during the school day, reflecting geographic inequality tied to both pollution sources and demographic factors. On a statewide level, researchers modeled the effects of regulatory emission reductions on different California communities between 2000 and 2019 and projected future trends. The models indicate that continued emissions reductions (from cleaner technologies and stricter standards) will continue to reduce overall pollution levels and narrow the absolute gap in PM_{2.5} exposure between the most and least polluted areas.² However, relative disparities might persist, with the ratio of pollution between disadvantaged and advantaged areas continuing to be unequal.² Many high-scoring CalEnviroScreen communities have been chronically impacted by multiple sources of pollution (freeways, refineries, ports, etc.) and socio-economic stressors. Consequently, while California's air quality has improved overall, continuing exposure disparities underscore the need for continued, targeted efforts to alleviate pollution in the most impacted neighborhoods.

Despite improvements, disparities in exposure to air pollutants remain. Recent evidence indicates that “for people of color and overburdened community residents, relative exposure disparities persist and, in some cases, have increased”.² Studies consistently show that low-income and minority populations experience higher exposures and face more severe health consequences, including elevated risks of asthma, cardiovascular disease, and premature mortality.^{9,10,30–33} These inequities underscore that both *who you are* (race/ethnicity, socioeconomic status) and *where you live* (geographic and neighborhood context) profoundly shape air pollution exposures and associated health outcomes.

1.3.3 Regulatory Approaches to Reduce Disparities

Mitigating these inequities in air pollution exposure and health outcomes is a priority for California regulators and policymakers. One regulatory approach to address disparities includes targeted monitoring and enforcement in over-burdened areas. For example, a field study in Los Angeles observed significantly higher O₃ exposures in parks located in disadvantaged communities (with majority Latino visitors) compared with parks in wealthier areas, and recommended deploying additional air quality monitors in these neighborhoods to better inform community-level interventions.¹⁵ Enhanced monitoring can lead to more responsive regulation. For example, if pollution “hotspots” or high-exposure microenvironments are known (such as schools near highways), stricter controls on nearby emission sources or mitigation (e.g. air filtration in schools) can be implemented. Evidence from Houston and other cities, where many low-income and minority residents live closer to refineries or heavy traffic, underscores the need for such place-based regulatory focus.¹¹ Interventions can include tighter emission limits for facilities in environmental justice communities, enhanced permit conditions, or targeted enforcement sweeps to ensure compliance in areas that need relief the most. In California, the Community Air Protection Program, noted above, promotes the use of data on pollution levels and community vulnerability to develop local plans aimed at reducing disparities.^{20,21}

In summary, reducing air pollution disparities requires a combination of universal measures (to improve air quality overall) and equity-focused measures (to ensure improvements reach and benefit historically over-burdened populations). The literature supports this dual approach: without deliberate equity-driven policies, vulnerable groups may continue to face disproportionate pollution risks even as average air quality improves.²

1.3.4 Impacts of Personal Behaviors on Air Pollutant Exposure

Individual behaviors and daily activities can also influence personal exposure to air pollutants. Personal behavior patterns vary across different communities, thereby contributing to disparities in exposure. For example, higher exposures to chloroform (which can be emitted during bleach use) and dichlorobenzene (present in mothballs in some states) have been observed among non-white and lower-income individuals.¹² In a California study, use of “green” cleaning materials reduced many VOC exposures to residents in Salinas, CA.³⁴ For many low-income populations, access to more expensive green household or personal care products may not be feasible, resulting in exposure disparities. Similarly, use of “greener” personal care products have been associated with lower chemical exposure in Latina adolescents.³⁵

Mobility and time–activity patterns can also influence exposure. Research in New Jersey found that residents of disadvantaged, high-pollution neighborhoods had daily routines that increased their interaction with polluted environments, thereby increasing their exposure.²³ Children are often less mobile than adults, and if they live in a highly impacted community they may spend more time playing outside near traffic-dense streets and or attend schools adjacent to major highways (as noted in the Santa Clara County study), resulting in elevated exposures.¹⁶

Individual behaviors and housing conditions can further impact personal exposure levels. A study of asthmatic children in urban homes found that those living in multifamily apartment buildings, which often have shared ventilation systems and potential indoor sources such as gas

stoves, experienced higher indoor NO₂ concentrations, correlating with more frequent asthma symptoms.¹⁸ In this study, the “behavior” might simply be the necessity of living in a certain type of more affordable housing. Residents may also engage in cooking or heating practices (such as using gas appliances without ventilation) that increase indoor pollutants.³⁶ These everyday actions can disproportionately affect low-income families who are more likely to live in older, multi-unit housing with limited control over air quality.

Additionally, personal and cultural practices can create additional pathways of exposure. For example, growing food in backyard or community gardens is a beneficial activity, but if the soil is contaminated by nearby traffic or industry, it can lead to ingestion of pollutants. A review of PAH pollution noted that in communities with PAH-contaminated soil or dust, locally grown fruits and vegetables can accumulate these toxins; thus, residents who consume such produce might be exposed to more PAHs.²⁴ This pathway is a function of personal behavior (gardening and diet) interacting with environmental conditions.

In summary, while ambient air quality may characterize the neighborhood-level exposure and risk, individual behaviors and living circumstances can either mitigate or worsen actual individual exposure.

1.3.5 Air Pollutant Exposure and the Impacts of External Factors

Exposure to air pollution and its health effects are influenced by external factors beyond just the pollutants themselves. For example, community violence, psychosocial stress, neighborhood economic conditions, and broader structural inequalities can modify both the distribution of pollution and the susceptibility of populations. A study in Boston, MA found that children of women exposed to both air pollution and elevated community violence during pregnancy had a higher likelihood of repeated wheezing early in life.³⁷ In this case, an external social factor (violence and stress in the neighborhood) acted synergistically with pollution to harm children’s respiratory health. Similarly, research on older adults nationwide showed that associations between PM_{2.5} exposure and cognitive decline was stronger for those residing in neighborhoods with higher psychosocial stressors (e.g. crime, poverty, social disorder).³⁸ These findings support the concept that chronic stress and environmental pollution together impose a double jeopardy on health.

Structural societal factors also play a critical role in pollution exposure disparities. A national study by Woo et al. (2019) found that both an individual’s race/ethnicity and the level of segregation in their metropolitan area independently contributed to their pollution exposure. In segregated communities, minority communities often live in closer proximity to highways, industrial zones, or other pollution sources.¹³ Thus, differences in personal income and individual choices, as well as the layout of cities shaped by historical housing policies (e.g., redlining) and ongoing economic disparities, can lead to higher pollutant levels in neighborhoods of color. Another structural factor is income inequality and community poverty. A state-level analysis across the U.S. found that in states with greater income inequality, the adverse impact of PM_{2.5} on life expectancy was more severe.³¹ Thus, broader economic disparity can intensify the harm caused by pollution. At the community scale, a Washington State study showed how historical adverse childhood experiences (ACEs) and neighborhood

deprivation interacted with pollution: residents with high personal stress histories living in disadvantaged neighborhoods experienced stronger associations between air pollution (such as NO₂) and chronic health conditions (such as stroke and diabetes).³⁰ This finding underscores that social factors (trauma, poverty, lack of social support) can increase vulnerability to the adverse effects of air pollution.

In summary, the built environment and social factors influence exposure disparities and health impacts¹⁶ Features such as green space, urban design, regional climate impact, and socioeconomic status modify pollution exposure and health impacts. High pollution exposure is harmful, but high exposure in combination with social vulnerability (poverty, stress, isolation) can be more detrimental. Interventions that reduce community stress (e.g. violence prevention, economic development) or that reduce segregation and isolation may help diminish the compounding effect of external factors on pollution impact.^{31,37}

1.4 Noise

Many sources of air pollution also emit noise pollution, including transportation corridors with vehicles and rail, airports, ports, and energy production facilities and factories. Because noise and air pollution are often co-exposures, more information is needed to better understand how these two environmental factors interact.³⁹ To date, there is little direct information about noise exposure in California, although there is evidence that environmental noise is a serious public health concern. The World Health Organization (WHO) reports that transportation noise from road, rail, and air traffic is the second largest environmental contributor to disease burden in Europe, following air pollution.⁴⁰ WHO estimated there are 600 to 1,200 disability-adjusted life years (DALYs) lost per million people worldwide, largely due to cardiovascular disease, sleep disturbance, and other chronic outcomes.⁴¹ Even during sleep, noise activates the body's stress systems, raising cortisol levels, blood pressure, and heart rate.^{42,43}

1. EPA Identifies Noise Levels Affecting Health and Welfare.
<https://www.epa.gov/archive/epa/aboutepa/epa-identifies-noise-levels-affecting-health-and-welfare.html>

1.4.1 Noise Exposure Regulatory Context and Mitigation

Historically, noise exposure and control was under the purview of the U.S. EPA.¹

“Under the Clean Air Act, the EPA administrator established the Office of Noise Abatement and Control (ONAC) to carry out investigations and studies on noise and its effect on the public health and welfare. Through ONAC, the EPA coordinated all Federal noise control activities, but in 1981 the Administration concluded that noise issues were best handled at the State and local level. As a result, ONAC was closed and primary responsibility of addressing noise issues was transferred to State and local governments.”

Based on California Health and Safety Code 46000-46080, cities and municipalities regulate localized community noise sources, such as household activities. Typically, quiet hours, usually 10 PM to 7 AM, are designated. Nighttime and daytime noise limits vary depending on the time of day and the zoning regulations. Typical noise limits are 55 dB during the day and 45 dB during the night for residential zones. Commercial zones are typically allowed up to 70 dB during the day and 60 dB at night. Higher noise levels may be allowed during permitted events, such as fairs or concerts. In industrial zones, typical limits are a maximum of 85 dB during the day and 75 dB at night. The action threshold for hearing protection in an occupational environment is an 8-hour time-weighted-average of 85 dBA, and the Permissible Exposure Limit (PEL) for California is 90 dBA (Cal. Code regs., tit. 8, sections 1521, and 5095-5100, <https://www.dir.ca.gov/title8/sb7g15a105.html>).

Figures 2a and 2b show the U.S. Department of Transportation noise maps for Fresno and Stockton, California.⁴⁴ The maps on the left show estimated noise exposure from all sources (aviation, road, and rail). The maps on the right show estimated road noise exposure only. These maps provide context for local noise exposure. Aviation and rail are significant, episodic sources of noise. **Figures 2a and 2b** also show that major roadways (i.e., State Route 99) indicate high noise emission. In contrast to the airport-related noise, roadways are a source of continuous noise emissions that can impact health differently than the episodic noise from airports and rail. Both noise sources are often the focus of mitigation programs, including restrictions on flight paths and schedules, and sound walls along freeways (see below).

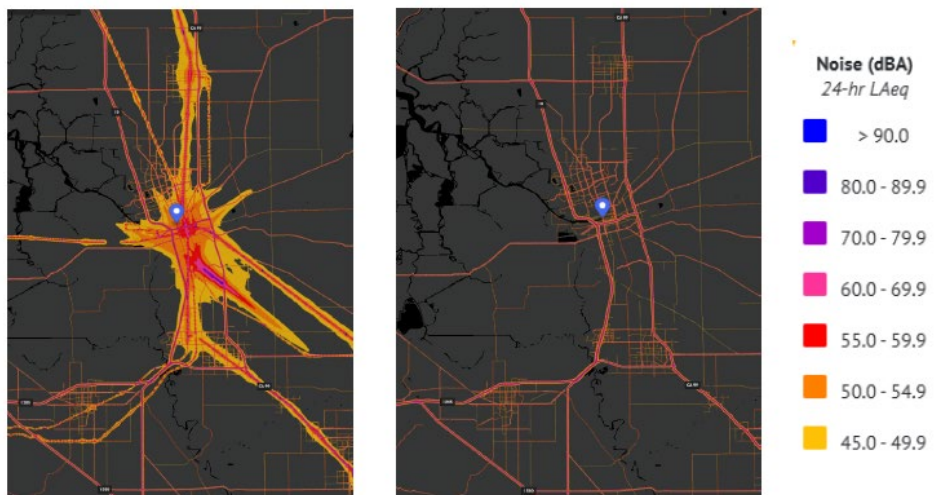


Figure 2a. Department of Transportation (DOT) Noise Maps for Stockton, CA. The map on the left shows estimated noise exposure from all sources (aviation, road, and rail). The map on the right shows estimated road noise.

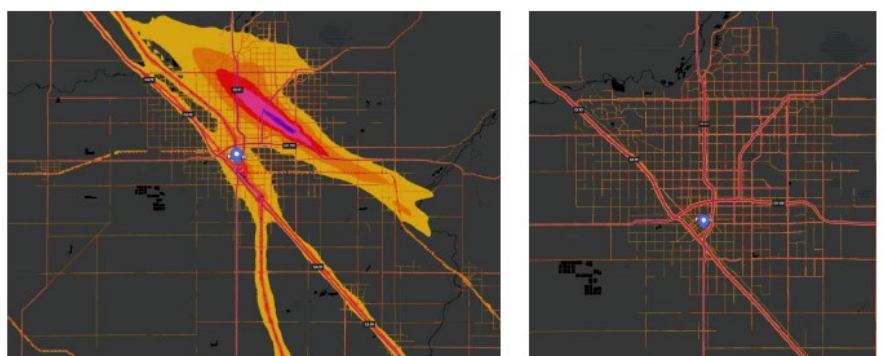


Figure 2b. Department of Transportation (DOT) Noise Maps for Fresno, CA. The map on the left shows estimated noise exposure from all sources (aviation, road, and rail). The map on the right shows estimated road noise.

The majority of community noise impacts on health risk are related to stress responses and sleep interruption related to non-damaging irritant noise levels (see below). There are no published scientific benchmarks established for irritant noise and psycho-social health impacts.

The Fresno County Municipal Health and Safety Noise Ordinance (Title 10, Chapter 10.24.020) is an example of exterior noise standards in a municipal setting, as seen in **Table 1**. However, these noise levels are not attainable, as 50 dBA is described as the noise level of moderate rain and 50-60 dBA is the average noise level from a regular conversation heard from 3 feet away. As such, it's unclear what the relationship is between the municipal code and health outcomes.

Table 1. Fresno County Municipal Code 8.40.040 - Exterior Noise Standards

Category	Cumulative Number of minutes in any one-hour time period	Noise Level Standards, dBA	
		Daytime 7 a.m. to 10 p.m.	Nighttime 10 p.m. to 7 a.m.
1	30	50	45
2	15	55	50
3	5	60	55
4	1	65	60
5	0	70	65

In California, several agencies have adopted strategies to mitigate environmental noise. Caltrans, in particular, has implemented important measures to reduce highway noise exposure, such as extensive sound wall construction.⁴⁵ Caltrans is also conducting research on road surfaces to reduce tire and pavement noise by identifying surface treatments, materials, design specifications, and construction methods that yield safe, durable, cost-effective roads with less noise production; early data suggests better construction quality is correlated with lower noise levels.⁴⁶ Before initiating construction projects, Caltrans and the Federal Highway Administration conduct noise studies during environmental reviews to determine where sound walls are recommended, and measuring existing and predicted traffic noise near sensitive locations such as homes, schools, churches and hospitals.⁴⁷ Engineers also use standardized templates and noise modeling protocols to assess noise impacts in planning and design processes. Sound wall feasibility and reasonableness are assessed based on topography, access, utility rights-of-way, other noise sources, safety, cost and community input.⁴⁷

Electrification of the state's rail systems is projected to reduce noise pollution from trains, although systematic assessments of these benefits remain limited. Airport-related noise is usually managed through land use compatibility guidance and regulatory oversight.

Overall, noise mitigation regulations and programs reflect substantial investments in noise reduction with likely public health benefits.

1.4.2 Noise Exposure in Disadvantaged Communities

Socially and economically disadvantaged communities may be disproportionately impacted by noise pollution because noise exposures increase with proximity to major roads,

which are often located near lower income communities. For example, in a study of noise exposures in 77 Chicago communities, higher noise levels were present in mid-to-low income communities.⁴⁸ Weuve et al. also reported higher noise exposures in neighborhoods on the south side of Chicago with lower socioeconomic status (2020).⁴⁹ In Minnesota, lower socioeconomic status or a higher proportion of minority race/ethnicities was also associated with higher aircraft noise exposures.⁵⁰ Casey et al. (2017) reported that higher model-based estimates of noise exposure in the United States occurred in neighborhoods with higher proportions of nonwhite and lower-socioeconomic status residents.⁴² In a similar U.S. nationwide study, “block groups with a higher Hispanic population had higher odds of being exposed to aircraft noise”.⁵¹ Finally, Huang and Seto (2024) estimated that “7.8 million (2.4%) individuals were highly annoyed by aviation noise, while 5.2 million (1.6%) and 7.9 million (2.4%) people were highly annoyed by rail and roadway noise, respectively, across the U.S.”, with Non-Hispanic Asian, Black, NHPI, and Hispanic populations disproportionately exposed to transportation noise nationwide.⁵²

These U.S.-based studies are consistent with published literature from other countries. For example, higher noise exposures were observed among lower socio-economic status neighborhoods in Hong Kong, Montreal, and London.^{53,54} The odds of living in a residence with a high risk of experiencing rail noise was higher among black compared with white residents, indicating inequalities in noise exposure based on race/ethnicity.

1.4.3. Overview of noise-related health effects

This section presents a review of noise-related health effects across several domains. It begins with evidence on birth outcomes, including low birth weight and preterm birth, followed by sections on cognitive outcomes, mental health, sleep disruption, metabolic disorders, and cardiovascular disease. While the strength of evidence varies, findings across all categories suggest that noise is a widespread and preventable public health risk. The implications are particularly relevant for California, where high traffic volumes and socioeconomic disparities may intensify health risks (Figure 3).⁵⁵

1.4.3.1. Birth Outcomes

Several studies have reported associations between prenatal noise exposure and low birth weight (LBW), preterm birth, and small for gestational age (SGA).⁵⁶⁻⁵⁸ Argys et al. (2020) found that residential exposure to aviation noise above 55 dB near airports increased the likelihood of LBW by 1.6 percentage points.⁵⁶ Similarly, Nieuwenhuijsen et al. (2017) and Ristovska et al. (2014)

Risk and warnings	Examples	dBA
Painful & dangerous: Use hearing protection or avoid	Fireworks, gun shots and stereos (at full volume)	130
Uncomfortable: Dangerous over 30 seconds	Jet planes (during take off)	125 120
Very Loud: Dangerous over 30 minutes	Concerts (any genre of music), car horns and sporting events	115 110 105 100 95 90
Over 85 dB for extended periods can cause permanent hearing loss		85
Loud	Alarm clocks	80 75
	Traffic and vacuums	70
Moderate	Normal conversation and dishwashers	65 60
	Moderate rainfall	55 50
	Quiet library	45 40
Soft	Whisper	35 30

Figure 2: The traditional definition of noise is unwanted or disturbing sound. “Sound becomes unwanted when it interferes with normal activities such as sleeping or conversation or generally diminishes one’s quality of life.”

reviewed multiple studies and concluded there was low-quality but suggestive evidence linking environmental noise, particularly from road and air traffic, to LBW, preterm birth, and SGA.^{57,59} Supporting this conclusion, Dzhambov and Lercher (2019) reported a small but statistically significant decrease in birth weight with increasing road traffic noise, although they found no consistent associations with LBW or preterm birth.⁶⁰ In contrast, Graafland et al. (2023) found no associations between residential noise exposure and fetal growth or adverse birth outcomes, though they observed a modest association with increased embryonic size.⁶¹ Likewise, Wallas et al. (2022) reported no link between maternal noise exposure during pregnancy and adverse birth outcomes, with an unexpected finding of reduced risk of preterm birth.⁶² Several reviews also suggest that occupational noise exposure may increase risks of SGA, preeclampsia, and congenital anomalies.^{58,60,63} In summary, there is growing but mixed evidence that both environmental and occupational noise exposure may adversely affect birth outcomes.⁴¹ While some studies suggest associations between transportation noise and risks such as low birth weight, preterm birth, and small for gestational age, others find no consistent effects. Similarly, reviews and meta-analyses indicate that occupational noise exposure, particularly at levels above 80 dB, may increase the risk of specific outcomes like SGA, preeclampsia, and congenital anomalies. Overall, although the evidence is not fully consistent, current findings support the need for further research and greater consideration of noise as a potential risk factor in maternal and fetal health, particularly in regions like California, where such studies remain limited.

1.4.3.2. Cognitive Function

Environmental and traffic noise are increasingly recognized as environmental stressors with measurable impacts on brain development and cognitive functioning, particularly in children and adolescents. Dohmen et al. (2022) conducted a systematic review and found that environmental noise negatively affects attention and may contribute to learned helplessness in children aged 8–13 due to cognitive fatigue.⁶⁴ Gheller et al. (2024) reviewed 26 studies and reported that environmental noise affects reading performance, with possible cognitive benefits only in children with low attentional control.⁶⁵ In an experimental study, Jafari et al. (2019) found that increasing noise was associated with significant declines in attention and mental performance in children, along with electroencephalogram changes indicating increased cognitive fatigue, particularly reduced Beta and increased Alpha activity in frontal and occipital brain regions.⁶⁶ Focusing specifically on traffic noise, López-Vicente et al. (2025) found that long-term exposure to road-traffic noise during childhood altered brain connectivity and was linked to mind-wandering, suggesting that traffic-related environmental exposures may alter brain network organization during a key developmental period, potentially affecting later cognition and mental health.⁶⁷ Similarly, in a meta-analysis, Liang et al. (2024) found low-frequency noise, often found with traffic, significantly impaired higher-order cognitive functions such as reasoning, math, and data processing.⁶⁸ Thompson et al. (2024) expanded on previous reviews and showed through meta-analyses that aircraft noise is associated with lower reading and language scores in children, and that general environmental noise correlates with cognitive decline in middle-to-older adults.⁶⁹

Other studies have also shown increased risk of cognitive decline and dementia associated with noise exposure.^{49,70,71} Belojević (2023) reported that traffic noise was associated with brain structure changes near the auditory cortex, potential early markers of Alzheimer's disease.⁷¹ In institutional settings, Janus et al. (2023) reviewed 35 studies and reported that high daytime noise levels in nursing homes (55–68 dB(A)) worsened sleep and agitation in residents with dementia.⁷² Large-scale epidemiological evidence supports these concerns: Meng et al. (2021) showed in a dose-response meta-analysis that every 25 dB increase in noise raised dementia risk by up to 19%.⁷⁰ Ritz and Yu (2021), citing Danish cohort data, estimated that 1,200 dementia cases in 2017 could be attributed to transport-related noise.⁷³ Similarly, Weuve et al. (2021) found in the Chicago Health and Aging Project that each 10 dBA rise in community noise exposure was linked to significantly higher odds of both mild cognitive impairment and Alzheimer's disease.⁴⁹ Together, these studies indicate that chronic exposure to environmental and traffic noise can lead to cognitive impairments and neurodevelopmental alterations.

1.4.3.3. Mental Health

Mental health is another area of concern. Gong et al. (2022) conducted a meta-analysis and found that individuals highly annoyed by environmental noise, particularly traffic and neighborhood noise, were significantly more likely to experience depression (23% increase), anxiety (55% increase), and general mental health issues (119% increase).⁷⁴ Similarly, Guha (2022) reviewed studies indicating that chronic traffic and aircraft noise exposure is associated with increased risks of depression, anxiety, and Alzheimer's disease, with particularly strong effects in vulnerable populations such as older adults.⁷⁵ Expanding on these findings, Hahad et al. (2025) highlighted both epidemiological and biological evidence showing that noise is associated with depression, anxiety, suicidal behavior, and emotional disorders in youth, potentially through mechanisms like neuroinflammation, oxidative stress, and circadian rhythm disruption.⁷⁶ Newbury et al. (2024), using UK cohort data, found that noise exposure during childhood and adolescence significantly raised the risk of anxiety by early adulthood.⁷⁷

1.4.3.4 Sleep Disruption

Sleep disruption is one of the most direct and consistent consequences of environmental noise and a growing body of evidence supports a strong association between environmental noise exposure and sleep disturbance. In Taiwan, Chan et al. (2024) found that lower acoustic comfort and frequent noise-induced sleep disruptions were linked to poor sleep quality and reduced overall quality of life, highlighting sleep as a key mediator in the noise–health relationship.⁷⁸ In Israel, Halperin (2014) emphasized that nocturnal transportation noise disrupts sleep architecture, triggers biological stress responses, and contributes to next-day fatigue, cognitive issues, and long-term cardiometabolic risks.⁷⁹ In the U.S., Rudolph et al. (2019) showed that adolescents living in high-noise urban areas went to bed significantly later, underscoring noise's impact on sleep timing during a vulnerable developmental period.⁸⁰ A global meta-analysis by Smith et al. (2022) reported that each 10 dB increase in nighttime transportation noise significantly raised the odds of high sleep disturbance, particularly from rail (OR = 2.97), road (OR = 2.52), and aircraft (OR = 2.18) noise.⁸¹ Consistent with these findings,

Yamagami et al. (2023) conducted a study in Japan and found that even small increases in indoor nighttime noise were linked to measurable declines in both objective and subjective sleep quality among older adults.⁸² In summary, higher nighttime noise is linked to poor sleep quality, delayed sleep timing, and long-term sleep disturbance across different populations and age groups and is a biologically plausible pathway through which noise exposure adversely affects health.

1.4.3.5. Metabolic Disorders

Several studies have found increased noise exposure associated with metabolic disorders, including higher risk of obesity. In the United States, Bozigar et al. (2023) analyzed data from 74,848 women in the Nurses' Health Study and National Health Study II, estimating aircraft noise exposure at residential addresses near 90 airports.⁸³ They found that exposure to aircraft noise ≥ 55 dB was associated with 11% higher odds of obesity and greater long-term BMI gain. In Switzerland, Foraster et al. (2018) used longitudinal data from 3,796 adults to estimate residential exposure to road, railway, and aircraft noise.⁸⁴ The study reported that increased road traffic noise was linked to higher BMI, larger waist circumference, and a 25% increased risk of obesity, while railway noise was associated with being overweight, especially poor sleepers. In China, Liang et al. (2022) conducted a cross-sectional study among 3,412 children, using perceived neighborhood noise levels to assess exposure.⁶⁸ Children living in very quiet areas had 50% lower odds of obesity, and higher noise was associated with increased BMI and blood pressure. In the Nordic region, Persson et al. (2024) pooled data from 11 cohorts including up to 162,639 participants, estimating long-term residential exposure to road, rail, and aircraft noise; every 10 dB increase in road and railway noise was associated with higher odds of both overall and central obesity.⁸⁵ Veber et al. (2023), using cross-sectional data from the RHINE cohort across Northern Europe, assessed self-reported traffic noise in bedrooms and measured BMI and waist circumference.⁸⁶ Among women, high nocturnal noise exposure was linked to higher BMI and waist size, partially explained by sleep disturbance. Finally, Yu et al. (2023) conducted a study among 3,427 factory workers in Guangzhou, China, and found that higher occupational noise exposure was associated with increased BMI and obesity risk, particularly among shift workers.⁸³

Growing evidence links long-term exposure to environmental noise, particularly from transportation sources, with an increased risk of type 2 diabetes mellitus (T2DM). Multiple cohort studies across different countries, including Canada (Shin et al. 2020, Basner et al., 2023; Clark et al., 2017), Denmark and Switzerland, consistently found that increases in average road traffic noise (typically 6–10 dB) were associated with a 6–8% rise in T2DM incidence or mortality.^{87–91} Experimental research in animals supports these findings by showing that chronic noise disrupts glucose metabolism and induces gut inflammation (Cui et al., 2016).⁹² Additional studies suggest that factors such as low neighborhood socioeconomic status (Letellier et al., 2023) and occupational noise exposure (Kim et al., 2021) may amplify this risk.^{93,94} While one cross-sectional study in Tunisia (Kacem et al., 2021) found no significant association, a comprehensive meta-analysis by Zare Sakhvidi et al., 2018 confirmed a 6% increased risk of diabetes per 5 dB increase in noise exposure, with stronger effects seen for air and road traffic

noise in Iran.^{95,96} Together, this evidence underscores transportation noise as an independent and under-recognized environmental risk factor for diabetes.

1.4.3.6. Cardiovascular Disease

Several studies suggest that long-term exposure to noise, particularly from transportation sources, can elevate the risk of cardiovascular disease such as hypertension, ischemic heart disease, heart failure, and stroke. For example, a meta-analysis of 14 studies reported a significant increase in relative risk of coronary heart disease with higher road traffic noise exposure.⁹⁷ In a cross-sectional study of 500 adults in Hong Kong, increased indoor nocturnal noise was associated with higher Body Mass Index (BMI) and blood pressure in women and higher blood pressure in men.⁹⁸ Similarly, adults in West India exposed to road traffic noise had increased risk of CHD and in Europe, traffic noise exposure was associated increased use of antihypertensive medication.⁹⁹ Similarly, antihypertensive medication use in the UK. Aircraft noise annoyance was significantly associated with use of antihypertensive medication, anxiolytics, hypnotics and sedatives. Correia et al. (2013) found that among over six million older adults, a 10 dB increase in aircraft noise was associated with a 3.5% rise in cardiovascular hospitalizations.¹⁰⁰ Similarly, Davies and van Kamp (2012) reviewed evidence across various noise types including road traffic, aircraft, railway, and occupational sources, and concluded that noise exposure, especially at higher intensities and during nighttime, was associated with elevated blood pressure, hypertension, and cardiovascular mortality.¹⁰¹ Roscoe et al. (2024), in a large U.S. cohort, reported a 4% increase in risk for CHD, CVD and stroke with higher daytime and nighttime anthropogenic noise levels.¹⁰² Supporting these associations, Balk et al. (2023) examined 390 adults in the Netherlands and found that higher levels of road traffic noise were linked to increased C-reactive protein, elevated triglycerides, and lower HDL cholesterol. These biomarkers reflect heightened systemic inflammation and metabolic disruption, pointing to a potential pathway by which noise promotes cardiovascular risk.

Mechanistic studies suggest a plausible biological mechanism for the impact of environmental noise on cardiovascular health by Hahad et al. (2019) and Münzel et al. (2021 and 2024).^{103–105} Chronic noise exposure likely triggers stress responses, elevating cortisol and other stress hormones, increasing blood pressure and heart rate, and promoting oxidative stress and inflammation. These effects can lead to endothelial dysfunction and vascular damage, which are key pathways in the development of hypertension and heart disease. Particularly, these biological responses are most noticeable at night and do not diminish with continued exposure, indicating that the body does not adapt to chronic noise stress. Importantly, studies by Foraster et al. (2014) and Sørensen et al. (2022) that noise has a distinct and independent effect on blood pressure and cardiovascular risk separate from traffic-related air pollution.^{90,106} Foraster's findings showed that indoor nighttime traffic noise raised systolic blood pressure and was linked to a borderline increase in hypertension risk, even after accounting for nitrogen dioxide exposure. Sørensen et al. reported that road traffic noise over a decade increased the risk of heart failure by 14%, and this association remained after adjusting for nitrogen dioxide. Women, older adults, and individuals with pre-existing conditions such as hypertension or diabetes were especially susceptible, as suggested by Jin et al. (2024) and Roscoe et al. (2024).^{25,102}

In summary, there is strong literature supporting an association of noise exposure and cardiovascular disease, but few studies have been conducted in the U.S. or California.

1.4.4. Air and Noise Co-exposures

Because noise and air pollution are often co-exposures, more information is needed to better understand whether these two environmental factors interact independently, additively or synergistically.³⁹ A population-based cohort study (n=445,868) with a 5-year exposure period and a 4-year follow-up period investigated the independent and joint influences of community noise and traffic-related air pollution on risk of coronary heart disease (CHD) mortality among adults in metropolitan Vancouver, Canada (Wen et al., 2012). Individual exposures to community noise and traffic-related air pollutants including black carbon, PM_{2.5}, NO₂, and nitric oxide were estimated at each residence using a noise prediction model and land-use regression models, respectively. Traffic-related air pollution and noise were independently associated with a 6% increase in CHD mortality. Elevations in noise and black carbon were associated with a 4% increase in CHD mortality. Study participants in the highest noise exposure category had a 22% increase in CHD mortality compared with the lowest exposure category, independent of traffic-related air pollution. This finding suggests that traffic-related air pollution and noise exposures have independent effects on CHD mortality.

In a population-based cohort study in British Columbia, Canada, the influence of modeled traffic noise exposure was positively associated with incident diabetes over a 5-year period. Air pollution exposures, including black carbon, PM_{2.5}, and nitrogen oxides, were also modeled. The independent association between noise exposure and incident diabetes remained after adjustment for the co-exposure of these traffic-related air pollutants.⁸⁹

Lim et al., 2021 investigated long-term exposure to air pollution and road traffic noise with incident heart failure using data on female nurses collected in the Danish Nurse Cohort Study.¹⁰⁷ Annual exposures to noise, NO₂ and PM_{2.5} were estimated based on location of residence. Noise, NO₂, and PM_{2.5} were each independently associated with increased hazard ratios for heart failure incidence. An enhanced risk of incident heart failure was observed for those exposed to combined high levels of noise and air pollutants, however the association was not statistically significant.¹⁰⁷ Sørensen et al. reported additive risks for traffic noise and air pollution for incident heart failure (IHF).¹⁰⁸ Despite these mixed findings, plausible biological mechanisms linking noise exposure and air pollution exposure to cardiovascular disease, suggesting that noise exposure could increase sensitivity to the hazardous effects of air pollution exposure and vice versa (Sørensen et al. 2017), but there is limited data available to examine this potential interaction.¹⁰⁸ In a retrospective cohort study of air and noise pollution on birth weight in the greater London area of the United Kingdom, PM_{2.5} was found to be associated with increased risk of low birth weight, but little evidence linked low birth weight and road traffic noise.¹⁰⁹

Overall, there is a limited literature examining the interaction of air pollution and noise exposure on health outcomes, and, to date, few or no studies focusing on California.

1.4.5. Discussion

Environmental noise is an important but often unexamined public health concern. Research consistently shows that long-term exposure to noise, particularly from transportation sources such as road traffic and aircraft, is linked to a wide range of potentially serious health conditions. These include cardiovascular disease, hypertension, diabetes, and obesity. Noise exposure likely affects the body by activating stress pathways, raising blood pressure, disrupting hormonal balance, and promoting inflammation.

The impact of noise may begin even before birth. While findings on birth outcomes are somewhat mixed, several studies suggest that noise exposure during pregnancy may increase the risk of low birth weight, preterm birth, or other developmental complications. Noise also has significant effects on sleep and brain function. Studies from several regions show that environmental noise, especially at night, disrupts sleep quality and timing. Poor sleep in turn increases the risk of heart disease, obesity, and mental health issues. In children, exposure to noise has been shown to reduce attention, impair reading and memory, and interfere with brain development. In older adults, chronic noise exposure is linked to cognitive decline and a higher risk of dementia. These effects are not just temporary disturbances but could result in long-term changes in brain structure and function. Existing evidence indicates that noise harms both mental and physical well-being, affecting people across the course of life.

These biological responses, especially when experienced repeatedly over time, contribute to the development and progression of chronic diseases. Vulnerable groups such as children, older adults, women, and individuals with preexisting health conditions appear to be especially sensitive to the effects of environmental noise. To date, little research on the distribution of noise exposure and health impacts has been conducted in California. Future research to assess exposures, health impacts, and benefits of noise mitigation policies should be evaluated. See below for our recommendations on research needs.

2 Materials and Methods

The following sections describe the approach used to understand air pollution and noise exposures and potential health risks among families living in the San Joaquin Valley, CA.

2.1 Research Approach

2.1.1 Participant recruitment and consent

Participants were invited to participate in the study by our community partners in Stockton, CA (Little Manila Rising - LMR) and Fresno, CA (Central California Asthma Collaborative - CCAC). Recruitment activities included outreach to families receiving services from LMR and CCAC and informational tabling at local clinics. Eligible participants included parents with a child aged 4 - 13 years old residing in Stockton, Fresno, or other areas in the SJV, California who spoke Spanish or English. Sixty-four parent-child pairs, including twelve

Stockton families and 52 Fresno area families, were enrolled. The study visits occurred between February and November of 2023. Given the small number of participating households from Stockton, results from this subgroup should not be considered representative of the broader Stockton community.

All study activities were reviewed and approved by the Institutional Review Board (IRB) at the University of California (UC), Berkeley. Parents provided written informed consent, and child participants 7 to 13 years old provided their assent to participate.

2.1.2 Overview of 24-hour Indoor and outdoor air monitoring and sampling in Fresno and Stockton

After consent was obtained, study staff visited each home to set up air and noise monitoring equipment, usually in the morning, on the first day of a two-day sampling period. Thus, air monitoring was conducted for approximately 24 hours during each two-day visit. Air monitoring equipment (described below) was set up inside and outside of participating family homes during the first day. The monitors were placed on a movable cart (**Figure 4**). Indoor sampling carts were deployed in a central living room location near bedroom entrances; outdoor sampling carts were placed outside near home entrances, often on a deck or rear-yard porch. Air monitoring included equipment to collect real-time measurements and, in some cases, integrated samples for laboratory measurements were deployed (**Figure 4**). The real-time measurements included: particulate matter with a diameter of 2.5 micrometers or less ($PM_{2.5}$); ozone (O_3); nitrogen dioxide (NO_2); carbon monoxide (CO); total VOCs (TVOCs), black carbon (BC), and noise levels.

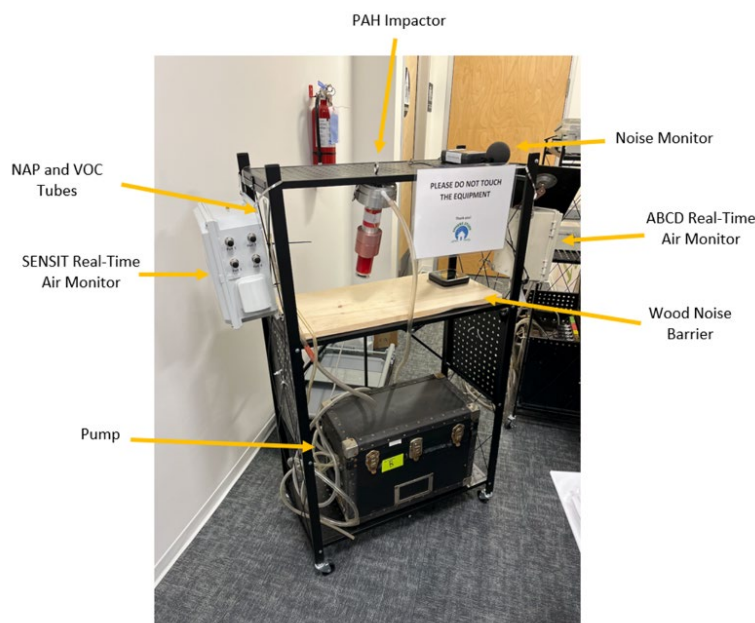


Figure 3: Overview of SPHERE Environmental Sampling Platform

Laboratory analyses were performed on indoor and outdoor actively collected samples for volatile organic compounds (VOCs) and polyaromatic hydrocarbons (PAHs), and passive samples for particulate matter and formaldehyde.

Study staff returned to participants' homes on day two of study activities to retrieve the 24-hour household-level monitoring devices and sampling equipment. Note, for eight families, a repeat study collection effort was completed 2-3 months after the first visit using the same protocol. **Table 2** summarizes SPHERE (San Joaquin Valley Household Environmental Research and Exposure Study) sample collection from 64 participating households in Fresno and Stockton.

2.1.3 Personal Monitoring

In addition to home-based monitoring, personal monitoring was conducted with the adult parent for PM_{2.5}, TVOCs, noise levels (see **Table 2**). Each parent was provided with a tote bag that included real-time monitoring devices for PM_{2.5}, TVOCs, and noise and could be easily held or kept nearby during normal daily activities. Study staff returned to the household the next day to retrieve the personal monitoring equipment and any related paper forms.

2.1.4 Data Collection

Table 2 summarizes environmental sample collection from the 64 participating households in the SPHERE study, including questionnaires, indoor, outdoor, and personal monitoring data, with repeat sampling conducted in 8 homes (resulting in total sample counts that may exceed the number of unique households). All 64 households completed the questionnaire and home inspection. (See **Appendix 1** for the pre- and post-sampling questionnaires and home inspection form.) Seventy-one real-time data sets were collected from homes for criteria pollutants and noise; 69 data sets for black carbon; and 46 data sets for total VOCs (numbers >64 includes repeat sampling visits). Thirty PM passive samples were collected from 8 homes in Fresno (16 samples collected during the initial SPHERE study visits (8 indoor and 8 outdoor samples); and 16 samples collected from the same homes one month later during repeat study visits. In Merced, 2 passive PM samples were twice collected from 5 homes (5 indoor and 5 outdoor samples) to increase the sample size. Additional field blanks were collected in Fresno and Merced. Additionally, 64 outdoor and 59 indoor integrated air samples were collected from Stockton and Fresno participant homes for polycyclic aromatic hydrocarbon (PAH) analysis as well as urine samples from 62 parent-child pairs for measurement of PAH and VOC metabolites and biomarkers of response (oxidative stress, inflammation, and lung injury). **Table 2** describes the distribution of real-time data sets and samples collected indoors and outdoors and for individual personal monitoring.

Table 2: Summary of SPHERE Sample Collection from 64 Participating Households (Fresno and Stockton)

	Questionnaire / Home Inspection	Active VOC samples	Active PAH filter samples	Active PAH Nap. gas tube	Criteria Pollutants (SENSIT)	Fine Particulate Matter	Total VOCs	Black Carbon (ABCDs)	Passive Formaldehyde Samples	UNC Passive PM Sample	Noise
Sampling events completed	64	23	108	109	71	71	46	69	24	28	71
Indoor		23	57	58	67	67	42	62	27	21	69
Outdoor		8	51	51	62	62	12	52		28	51
Personal monitoring						61	57				57

^aA total of 64 households and homes were enrolled; due to repeat sampling conducted in 8 homes, sample events may exceed 64.

***Note, additional passive PM samples were also collected from 5 homes in Merced.

2.2 Methods

See **Appendix 2** and **Appendix 3** for all standard operating procedures and instrument technical specification documents for air monitoring and sample collection.

2.2.1 Real-time Air Monitoring of Criteria Air Pollutants

We used the SENSIT® RAMP platform (Sensit Technologies, Valparaiso, Indiana) to collect real-time measurements of PM_{2.5}, NO₂, O₃, and carbon monoxide (CO) inside and outside homes (**Appendix 2 and 3**). The RAMP is a relatively low-cost (<\$4,000) and portable instrument modifiable to include sensors for a wide range of air pollutants. Measurements are uploaded to the cloud or transferred directly from a microSD card. The RAMP units were attached to the sampling cart with zip ties in a typical breathing zone (**Figure 4**).

2.2.1.1 Fine Particulate Matter (PM_{2.5})

We used the Sensit RAMP platform to collect real-time PM_{2.5} measurements inside and outside homes (See **Appendix 2** for the air monitoring protocol). The RAMP unit measures real-time PM using a laser-light based Plantower PMS5003 sensor (Nanchang, China). Light directed through air in the monitor is scattered by any particles present. An algorithm converts the degree of light scatter into an estimated PM concentration (these sensors are the same devices employed by the widely used Purple Air sensors). PM_{2.5} levels are reported from 1 to 1000 µg/m³ (± 10 µg min). Extensive literature has validated the validity of the Plantower sensors based on PM_{2.5} data reported by Purple Air (PurpleAir, Inc, Draper UT, USA). Recent comparisons between co-located Purple Air monitors and Beta Attenuation Mass Monitors (BAM) in the SJV showed strong correlations (r=0.86, A. Bradman, personal communication). We also collected co-located PM_{2.5} monitoring data from the Sensit RAMP monitors over six days and observed excellent consistency among the monitors (average correlation=0.99, coefficient of variation (CV) = 1.7%).

In addition, we deployed the Atmotube Pro (ATMO, San Francisco, California), a portable PM_{2.5} air monitor to monitor personal exposures for 24-hour monitoring periods. The Atmotube Pro characterizes PM_{2.5} using a Sensirion SPS30 particulate matter sensor, which also detects particles through laser light scattering. The Atmotube is MCERTS certified (MCERTS Performance Standards for Indicative Ambient Particulate Monitors and was evaluated by the AQ Spec South Coast Air Quality Management District.¹¹⁰ The Atmotube Pro PM_{2.5} sensors showed strong correlation with the corresponding federal equivalent method (FEM) (R²= 1) at 5-minute averaging. See **Appendix 3** for the Atmotube AQ Spec detailed evaluation report.

2.2.1.2 Ozone (O₃), Nitrogen Dioxide (NO₂), and Carbon Monoxide (CO)

We also used the Sensit RAMP platform to collect real-time O₃, nitrogen dioxide, and carbon monoxide measurements inside and outside homes. The sensors in the monitoring unit are manufactured by AlphaSense-Ametek (Braintree, United Kingdom). Ozone and nitrogen dioxide, both oxidizing gases, are measured with the AlphaSense OX-B431 electro-chemical sensor and carbon monoxide is measured with the AlphaSense CO-B4 sensor, both configured in the Sensit RAMP box. The target gases diffuse from the ambient air through a membrane into the sensor's internal cell. A small opening or capillary acts as a "diffusion barrier" to control the rate at which gas reaches the working electrode inside. This technology ensures a consistent, linear response. Once the gas molecules reach the electrode, they undergo an electrochemical reaction that produces an electrical signal proportional to the gas concentration. See **Appendix 3** for technical specifications for these sensors. We also collected co-located NO₂ and O₃ monitoring data from the Sensit RAMP monitors over six days and observed reasonable consistency for NO₂ (average correlation for NO₂=0.67, CV=1.0%) and good consistency for O₃ (average correlation O₃=0.82, CV=12.2%).

2.2.2 Real-time Black Carbon Monitoring

Black carbon is a component of particulate matter (PM) emitted during incomplete combustion of certain fuels, especially diesel, and is more commonly referred to as soot. The primary sources of BC include heavy-duty diesel trucks and other diesel engines, with minor sources from wood-burning stoves, agriculture burns, and charring food.¹⁹

We deployed Aerosol Black Carbon detectors (ABCDs) over twenty-four hours inside and outside of participant homes in Stockton and Fresno, CA on the SPHERE sampling cart (**Figure 4**) (**Appendix 2**). The ABCDs are aerosol absorption photometers that measure black carbon (BC) in units of micrograms per cubic meter (µg/m³).¹¹¹ The ABCD consists of a weatherproof enclosure that houses an optical cell, pump, Arduino and microSD card, and rechargeable battery. Ambient air is pulled through two filters in the optical cell. The instrument measures light absorption at 880 nm, where BC is the predominant species to absorb light, on a fibrous filter. As more particles are collected on the filter surface, the intensity of light transmitted through the filter is attenuated. The increase in filter attenuation (ATN) over time is proportional to the concentration of BC in the sampled air stream. The ABCDs have been validated previously in lab and field studies.^{111,112}

2.2.3 VOCs

2.2.3.1 Active Air Sampling

Volatile Organic Compounds (VOCs) sampling methods followed EPA TO-17 methods and were collected on the SPHERE sampling cart platform (**Figure 4**) (**Appendix 2**).¹¹³ Initially, we planned to collect 24-hour VOC samples; however, we encountered multiple problems with over-saturation of sorbent materials and ultimately reduced the sampling period to four hours. Airchek XR5000 pumps were deployed for four hours and calibrated to collect at 100 mL/min using a Chek-mate SKC flowmeter with a low-flow adapter on Markes TD Tenax tubes (Markes

International, Bridgend, United Kingdom). Air sample volumes ranged from 22.2 L to 25.8 L. VOC samples were kept in their sealed tubes and capped and shipped on ice to the CDPH Environmental Health Laboratory in Richmond, CA, where they were analyzed by thermal desorption and gas chromatography (TD-GS/MS). Repeat air sampling was performed at 7 of the homes after 30 days. In total, 23 indoor and 8 outdoor air samples were collected from 16 homes. In addition, QA/QC samples including 5 field blanks and 5 pairs of collocated duplicate samples were collected and analyzed.

Table 3 lists the 22 volatile organic compounds (VOCs) measured in the VOC samples. These VOCs represent a range of chemical classes, including aromatics, chlorinated solvents, aliphatic hydrocarbons, and methacrylates, reflecting both industrial and household sources of exposure. Commonly studied compounds such as benzene (CAS: 71-43-2), toluene (108-88-3), and methyl methacrylate (CAS: 80-62-6; a formaldehyde substitute) were included, along with chlorinated compounds such as chloroform (CAS: 67-66-3), methylene chloride (CAS: 75-09-2), and tetrachloroethene (CAS:P 127-18-4). Several xylenes (m-, o-, and p-xylene), trimethylbenzenes (1,2,4- and 1,3,5-), and other aromatic hydrocarbons such as ethylbenzene and naphthalene were also detected, indicating potential contributions from indoor sources including cleaning products, solvents, and off-gassing materials. The list also includes less frequently monitored compounds, such as tetrahydrofuran (CAS: 109-99-9), ethyl methacrylate (CAS: 97-63-2), and 4-isopropyltoluene (p-cymene, CAS: 99-87-6).

Table 3: Twenty-two VOCs measured in air (from 16 Fresno homes)

	VOC	CAS number
1	Acetonitrile	75-05-8
2	Benzene	71-43-2
3	Chloroform (Trichloromethane)	67-66-3
4	1,4-Dichlorobenzene	106-46-7
5	1,2-Dichloroethane	107-06-2
6	cis-1,3-Dichloropropene	10061-01-5
7	trans-1,3-Dichloropropene	10061-02-6
8	Ethylbenzene	100-41-4
9	Isobutyl Alcohol (2-methyl 1 propanol)	78-83-1
10	4-Isopropyltoluene (p-Cymene)	99-87-6
11	Methyl methacrylate	80-62-6
12	Methylene chloride	75-09-2
13	n-Propylbenzene	103-65-1
14	1,2,4-Trimethylbenzene	95-63-6
15	1,3,5-Trimethylbenzene	108-67-8
16	m-Xylene	108-38-3
17	o-Xylene	95-47-6
18	p-Xylene	106-42-3
19	Styrene	100-42-5

20	Tetrachloroethene	127-18-4
21	Tetrahydrofuran	109-99-9
22	Toluene	108-88-3

2.2.3.2 Real-time Total VOCs (TVOCs)

Instruments measuring total volatile organic compounds (TVOCs) characterize the total VOC concentration in air without targeting individual VOCs. Thus, the compounds measured may have different chemical structures, volatility, and toxicity; for example, benzene is a known carcinogen and toxic at higher concentrations, while others may be relatively less toxic or even non-toxic.

We used the Atmotube Pro (ATMO, San Francisco, California), a portable TVOC air monitor to supplement active VOC sampling and to monitor personal exposures (**Appendix 2**). Real-time indoor and outdoor TVOC measurements were collected by placing the Atmotube Pro on the sampling carts (**Figure 4**) for 24-hour monitoring periods. The Atmotube uses a Sensirion SGPC3 metal oxide sensing element (Sensirion, Zurich, Switzerland) to characterize TVOCs.¹² This sensor provides a TVOC output range from 0 to 60 ppm with a typical accuracy of 15% of the measured value. It operates with a PHE (photo-heating element) measurement interval of 2 second and the sensor offers a time resolution of 1 minute to allow fluctuations over short intervals. Technical specifications for the Atmotube and Sensirion sensor are included in **Appendix 3**.

2.2.3 Formaldehyde

We deployed SKC UMEx-100 (SKC Inc., Eighty Four, Pennsylvania) passive samplers to measure formaldehyde in air in selected homes (**Appendix 2**). The single-use UMEx-100 contains a tape treated with 2,4-dinitrophenylhydrazine (DNPH) which reacts with formaldehyde to form stable hydrazone derivatives, which are then analyzed using high pressure liquid chromatography (HPLC). Samples were collected and stored frozen until shipment to Atmospheric Analysis and Consulting, Inc. (AAC Lab) for analysis by EPA method TO-11A.^{114,115} Each passive sample includes a blank strip and additional field blanks were collected. Final concentrations were blank adjusted. See **Appendix 3** for UMEx-100 passive sample technical specifications.

2.2.4 Polycyclic Aromatic Hydrocarbons (PAHs)

We collected integrated 24-hour samples for PAHs with two methods: Harvard-type impactors with two 37mm filters and sorbent tubes with XAD4 (**Appendix 2**). Filters were pre-baked quartz fiber filters (PallFlex Tissue Quartz) impregnated with ground XAD-4 resin.^{116,117} The impactors had PM₁₀ inlets and airflow of 10 L/min.^{26,118,119} The sorbent tubes were collected with a flow rate of 200 cc/m. Both sample types were collected in the adult breathing range (**Figure 4**).

Filter samples were extracted in dichloromethane followed by vacuum filtration, then concentrated under nitrogen. Tube samples were extracted in dichloromethane and transferred to analysis vials without further concentration. Analyses were performed by gas chromatography/mass spectrometry (HP 6890/5972 or Agilent 7820/5977E) in the selected ion-monitoring mode with a 30m (5%-Phenyl)-methylpolysiloxane column (Agilent HP-5MS). We quantified and reported the following four PAHs: naphthalene, fluorene, phenanthrene and pyrene. Standard curves were run with the samples and sample results were blank corrected. The limit of quantitation (LQ) was calculated for individual PAH, media and day of laboratory analysis.

2.2.5 Noise

Indoor and outdoor noise levels were measured using Lutron DS-2013SD (Lutron Electronic Enterprise Co., Ltd., Taipei, Taiwan) sound level meters; these instruments quantify sound pressure levels in A-weighted decibels (dBA) and do not record sounds from the home, such as voices or private information (**Appendix 2 and 3**). The monitors were deployed on the sampling carts (**Figure 4**).

The Lutron DS-2013SD noise monitor is a Class 2 noise monitor, which is accurate to approximately ± 2 dB (OSHA Standard #1910.95). The noise monitors were calibrated before each deployment and set to log real-time noise levels every 15 seconds.

2.2.6 Passive PM sampling

Passive aerosol samplers (UNC-PAS, RJ Lee Group, Monroeville, PA, USA) were collected to characterize particulate matter concentrations and composition in 8 homes in Fresno, CA and 5 homes in Merced, CA (**Appendix 2**). (As noted above, the extra passive samples were collected in the Merced homes to increase the sample size for this substudy). The UNC-PAS samplers are about the size of a dime, and collect particles onto coated, 0.1 μm -pore polycarbonate filter substrates.¹²⁰ The sampler collects particles by gravitational settling and diffusion. Mass concentrations are calculated using a particle deposition velocity model that accounts for collection as a function of particle size.¹²¹ UNC-PAS mass size fractions and particle size distributions have shown good precision and correlation with active PM samplers.^{122,123}

The indoor samplers were deployed on the sampling cart approximately 1.5 m high. For outdoor sampling, each passive sampler was deployed inside a weather shelter.¹²⁴ After deployment, the samplers were shipped to RJ Lee group, Monroeville, PA and analyzed by computer-controlled scanning electron microscopy to determine particle characteristics.¹²⁵ Particle size, counts, and elemental chemistry were measured with a field emission scanning electron microscope (SEM) (Tescan MIRA3, Brno) with energy dispersive x-ray spectroscopy (EDS) (Bruker Quantax, Billerica, MA, USA) and automated analysis software (IntelliSEM v2, RJ Lee Group, Monroeville, PA, USA). Randomly selected 75 μm view fields were analyzed by the

IntelliSEM until 1,000 particles or 5 mm² were analyzed for each sample, and data were collected for each individual particle. CCSEM-EDS data from each sample were processed with a custom software algorithm that calculated PM size fractions (PM_{2.5}, PM_{10-2.5} (PM between 2.5 and 10 µm) and PM₁₀ (PM < 10 µm)), elemental size distributions, mass size distributions, and chemical types. Particle types were defined using a combination of the automated chemical types and a review of the acquired micro-images' particle size and morphology.

Agricultural particles were characterized by high potassium (>20%) or phosphorus (>20%), and lower aluminum and silicon (<20%) content. Metallic particles included heavy metals such as lead (Pb; >20%), chromium (Cr; >3%), copper (Cu; >3%) and titanium (Ti; >40%). Salt particle types were identified by the co-occurrence of sodium (Na; 20%) and chlorine (Cl; 20%).

The UNC passive aerosol samplers were deployed for four to five days in Fresno and Merced between October 2023 and January 2025. In Fresno, 32 total samples were collected from eight homes: 16 samples during the initial SPHERE study visits (8 indoor and 8 outdoor) and 16 additional samples from the same homes one month later during repeat visits. In Merced, 23 additional passive PM samples were collected from five homes (12 indoor and 11 outdoor) to increase the overall sample size. Repeat samples in Merced were collected sequentially, four days after the initial deployment. Field blanks were also collected for quality assurance.

As next steps, QA/QC results from these samples will be reviewed and summarized. In addition, results for larger particle fractions (i.e., PM₁₀ and coarse PM [PM_{10-2.5}]) will be evaluated. Additional SEM analyses are currently underway at the CDPH Environmental Health Laboratory Branch (EHLB) to further characterize a subset of Fresno and Merced PAS samples.

2.2.7 Estimated Air Exchange Rates

We estimated air exchange rates using indoor and outdoor carbon dioxide measurements recorded by the Sensit RAMP platform and estimates of indoor square footage obtain through county property data or the Zillow real estate website (Zillow Group, Seattle, WA; note, Zillow also obtains dwelling information from local county assessor data). We then used the Harvard CO₂ Concentration Calculator (<https://healthybuildings.hsph.harvard.edu/tools/co2-calculator/>) to estimate home air exchange rates.

2.3 Questionnaires and Health Outcomes

Pre- and post-sampling questionnaires were administered to assess sources of VOCs, PAHs and particulate matter exposure in the home, e.g., cooking behaviors, wood burning, hobbies, use of tobacco products and cleaning products, and proximity of the home to ambient sources of VOCs and noise pollution, e.g., auto, truck, buses, and other traffic. Study staff also collected information about sources and determinants of exposures in occupational settings,

including work Standard Industrial Classification (SIC) codes, specific tasks, chemicals used at work, use of tools and equipment, and whether work is inside or outside.

Additionally, the questionnaires collected health information about the adult parent and child health, and other members of the household. Information collected was used to assess respiratory health, stress, sleep habits, child behaviors, general health, awareness of noise and its impacts, as well as the participants' perspectives about environmental exposures. See **Appendix 1** for the pre- and post-sampling questionnaires used for this study.

2.4 GeoSpatial Data and Information Sources

2.4.1. Geocoding house locations

Participant addresses were geocoded using Google Earth (Google, Menlo Park, CA) and stored following approved IRB protocols.

2.4.2. CalEnviroScreen

Participant homes were assigned to census tracts and matched to the CalEnviroScreen 4.0 (CES) indicators, released in 2021, using ArcGIS Pro (Esri, Redlands, CA). CES indicator percentiles provide the relative rank of the census tract compared to the rest of the state, where the highest rank is the highest burdened census tract in the state. Indicators used in these analyses were:

- CalEnviroScreen Overall Score 4.0 (released in 2021) - Pollution Score multiplied by Population Characteristics Score;
- Diesel PM percentile score - diesel PM emissions from on-road and non-road sources
- Traffic Density percentile score - traffic density in vehicle-kilometers per hour per road length, within 150 meters of the census tract boundary.

2.4.3. Nearby Residential Traffic Density and Distance to SR-99

We also used the Tracking California Traffic Tool to quantify Daily Vehicle Miles Traveled (DVMT) near each home.¹²⁶ The tool uses Highway Performance Management System (HPMS) data from 2019 to compute the DVMT along monitored road segments within a given radius of residential locations. We used the tool to characterize daily traffic on road segments within buffers of 100, 250, 500, 750, and 1000 meters of each home, respectively.

Additionally, in Fresno and Stockton, we used Google Maps to compute the distance from each home to SR-99, the primary north-south heavy duty truck transportation corridor in the Fresno and Stockton regions.¹²⁷ Overall truck traffic on SR-99 has increased with increasing warehouse and distribution development, with 2023 traffic levels approaching pre-pandemic periods.¹²⁷

2.5 Statistical Analyses

For all survey instruments, the performing field technician reviewed questionnaire or inspection forms immediately after completing the forms to ensure all questions were answered. At the field office, an additional review was completed to ensure consistency and completeness. If any out-of-scale values were present, the forms were individually inspected to confirm recorded information. When needed, and approved by Drs. Noth or Bradman, participants were contacted to resolve any data problems.

Statistical analysis was performed with STATA statistical software Version 11.2 and R Statistical Software (v4.4.0; R Core Team 2024) to calculate the descriptive statistics and tests of association (e.g., Spearman Rank Correlation Coefficients, Mann-Whitney test, Wilcoxon test). Individual data sets were merged by participant identification numbers to create comprehensive data sets for statistical analysis. Only sampling periods with adequate data captured (at least 75% of the time frame) were included. Non-detectable values were imputed as $\text{LOD}/\sqrt{2}$, and out-of-range readings were capped at the instrument limits.

We used specific statistical methods for different types of comparisons described in the results, below. We examined exposure differences between Fresno and Stockton homes, as well as exposure determinants such as household characteristics, traffic burden, or proximity to major roads, using non-parametric tests and Spearman correlations. We also examined Spearman correlations pollutant co-occurrence and relationships between air and noise exposures.

2.6 Health Risk Characterization

2.6.1 Non-Cancer Risk

We evaluated non-cancer health risks from exposure to benzene, ethylbenzene, formaldehyde, and naphthalene using Hazard Quotients (HQs), based on Chronic Reference Exposure Levels (RELs) and Inhalation Reference Concentrations (RfCs). HQs were calculated for indoor and outdoor concentrations of benzene, ethylbenzene, toluene, xylenes, and naphthalene, as well as for formaldehyde (indoor only), using both median and 95th percentile concentrations.

To ensure consistent units, we obtained Chronic RELs from California's Office of Environmental Health Hazard Assessment (OEHHA) and RfCs from the U.S. EPA, then converted all values to $\mu\text{g}/\text{m}^3$. Hazard Quotients were calculated by dividing the measured exposure concentration ($\mu\text{g}/\text{m}^3$) by the corresponding Chronic REL or RfC ($\mu\text{g}/\text{m}^3$). We compared the concentrations to both RELs and RfCs, when available, to ensure the most health-protective standard was considered.

The Hazard Quotient (HQ) is a ratio that compares the estimated exposure to a chemical with a reference level considered unlikely to cause adverse health effects. An HQ greater than 1

suggests that the exposure exceeds the reference value and may pose a potential health concern.

2.6.2 Cancer Risk

Naphthalene, benzene, ethylbenzene, and formaldehyde are listed as carcinogens under Proposition 65.¹²⁸ For each VOC, daily inhalation exposures ($\mu\text{g}/\text{day}$) were estimated by multiplying measured concentrations by age- and sex-specific inhalation rates from the U.S. EPA 2011 *Exposure Factors Handbook* (adult women, mean age 42 years: $10.5 \text{ m}^3/\text{day}$; male children, mean age 9 years: $13.42 \text{ m}^3/\text{day}$; and female children, mean age 9 years: $12.41 \text{ m}^3/\text{day}$).¹²⁹ Median (50th percentile) and high-end (95th percentile) exposures were calculated for adult women, male children, and female children.¹²⁹ California OEHHA Proposition 65 No Significant Risk Levels (NSRLs) for each chemical were used as benchmarks, expressed in $\mu\text{g}/\text{day}$.¹²⁸ The NSRL represents a chronic exposure intake with potential cancer risks exceeding one in 100,000 (10^{-5}). Hazard ratios were computed by dividing estimated daily exposures by the respective NSRL values.

2.6.3 Noise Exposure

As noted above, the action threshold for hearing protection in an occupational environment is an 8-hour time-weighted-average of 85 dBA, and the Permissible Exposure Limit (PEL) for California is 90 dBA (Cal. Code regs., tit. 8, sections 1521, and 5095-5100).

The majority of community noise impacts on health risk are related to stress responses and sleep interruption related to non-damaging irritant noise levels. There are no published scientific benchmarks established for irritant noise and psycho-social health impacts. Fresno County Municipal Health and Safety Noise Ordinance (Title 10, Chapter 10.24.020) provides exterior noise standards in **Table 1**, above. However, these noise levels are not attainable, as 50 dBA is described as the noise level of moderate rain and 50-60 dBA is the average noise level from a regular conversation heard from 3 feet away. As such, it's unclear what the relationship is between the municipal code and health outcomes.

3 Results and Discussion

3.1 Cohort Characteristics

Sixty-four parent-child pairs living in the San Joaquin Valley (Stockton, Fresno and Clovis) were enrolled in the SPHERE study. Study participants spoke English (52%) and Spanish (48%) and were predominantly Hispanic (69%) (**Table 4**). Among adult participants, 95% were parents and 5% grandparents; 97% were female. The mean age was 42 years (range 26 to 66 years). Thirty-seven percent had not graduated from high school, and slightly more

than half of participating families had an annual household income of less than \$30,000 (56%). Fifty-two percent of children were male and 48% of children were female and their mean age was 9 years (range 4 to 13 years) (**Table 5**). Most participating children (57%) were in the overweight or obese BMI category (BMI \geq 85th percentile for age, sex, and height). (Note, some additional passive particulate air samples were collected from homes in Merced, CA; all participants in the study were from Stockton or Fresno, CA).

Table 4: Characteristics of adult participants (64 adult/child pairs)

Variable	Category	n (%) or Mean +/- SD
Language	English	33 (52%)
	Spanish	31 (48%)
Sex	Male	2 (3%)
	Female	62 (97%)
Gender Identity	Male	2 (3%)
	Female	62 (97%)
Race/Ethnicity	Asian	2 (3%)
	Black / African American	3 (5%)
	Hispanic / Latino	44 (69%)
	White	8 (12%)
	Two or more races	7 (11%)
Relationship to Child	Parent	61 (95%)
	Grandparent	3 (5%)
Age (years)		Mean (SD): 42 (+/- 8)
Education Level	Have not graduated from high school	23 (36%)
	High school graduate, GED, or equivalent	8 (13%)
	Some college or AA degree	15 (23%)
	College graduate or above	18 (28%)
Income	≤ \$15,000	11 (17%)
	>\$15,000 - \$30,000	25 (39%)
	>\$30,000 - \$50,000	9 (14%)
	>\$50,000 - \$75,000	4 (6%)
	≥ \$75,000	15 (23%)
Medical Insurance	No	6 (9%)
	Yes	58 (91%)
Married	No	24 (38%)
	Yes	40 (62%)
Home Ownership	Rent	35 (55%)
	Own	29 (45%)
Employed	No	26 (41%)
	Yes	37 (58%)
	Don't Know	1 (1%)
Home Type	Mobile Home	3 (5%)
	Single Family Detached Home	44 (69%)
	Residential Duplex	3 (5%)
	Apartment Building (w/ 3 or more units)	14 (22%)

Table 5: Characteristics of child participants (64 adult/child pairs)

Variable	Category	n (%) or Mean +/- SD
Sex	Male	33 (52%)
	Female	31 (48%)
Gender Identity	Male	33 (52%)
	Female	29 (45%)
	Non-binary	1 (2%)
	None of these	1 (2%)
Age (years)		Mean (SD): 9 (+/- 3)
Height (ft)		Mean (SD): 4 (+/- 1)
Weight (lbs)		Mean (SD): 88 (+/- 41)
BMI Category	Underweight	7 (11%)
	Normal Weight	20 (31%)
	Overweight	15 (23%)
	Obese	22 (34%)

Table 6 presents participating adult and child respiratory health characteristics. Respiratory symptoms were relatively common in both adults and children. Among adults, 27% reported that they had experienced wheezing or whistling in the chest, 14% reported a chronic cough not associated with illness, and 19% (n=12) had been diagnosed with asthma. Of those diagnosed with asthma, one-third (33%) reported regularly taking medication prescribed by a doctor to control their symptoms.

Among children, 23% had experienced wheezing or whistling in the chest, 14% had experienced a chronic cough unrelated to illness, and 27% had been diagnosed with asthma. Overall, the findings reflect a moderate burden of respiratory symptoms within these San Joaquin Valley households, consistent with elevated asthma prevalence previously documented in the region's disadvantaged communities.

Table 6: Adult and child respiratory health characteristics (n=64 pairs).

Variable	Category	n (%)
Parent		
Parent ever had wheezing or whistling in your chest at any time?	No	47 (73%)
	Yes	17 (27%)
Parent ever had chronic cough without cold or illness at any time?	No	55 (86%)
	Yes	9 (14%)
Parent ever diagnosed with asthma?	No	51 (80%)
	Yes	12 (19%)
	DK	1 (2%)
Parent regularly takes medicine prescribed by a doctor to control asthma? ^a	No	3 (25%)
	Yes	4 (33%)
	DK	5 (42%)
Child		
Child ever had wheezing or whistling in chest at any time?	No	49 (77%)
	Yes	15 (23%)
Child ever had chronic cough without cold or illness at any time?	No	54 (84%)
	Yes	9 (14%)
	DK	1 (2%)
Child ever diagnosed with asthma?	No	47 (73%)
	Yes	17 (27%)
Child regularly takes medicine prescribed by a doctor to control asthma? ^b	No	6 (35%)
	Yes	3 (18%)
	DK	8 (47%)

Note: DK = don't know / did not answer question.

^an=12 for parents diagnosed with asthma.

^bn=17 for children diagnosed with asthma.

For the 21 participants who completed all of the air sampling (personal, indoor, outdoor) and the questionnaires, we found that there was considerable diversity in the percentage of the day that participants spent indoors at home, ranging from a third to a full day (**Table 7**). For the 18 participants who spent time in vehicles, 17 were in automobiles and 1 used the bus.

Table 7: Percentage of each day in each microenvironment or performing exposure-related activity for 21 participants

Time spent in each location/activity	Mean ± SD	Min	Percentile				Max
			25th	50th	75th	90th	
Inside at home	72.2 ± 18.8	33.3	58.3	72.9	87.5	91.7	100.0
Work	18 ± 17.4	0.0	0.0	14.6	33.3	37.5	54.2
In Transit (traveling in vehicle)	1.7 ± 2.7	0.0	0.0	0.3	2.1	6.3	8.3
Outside at home	4.6 ± 6.7	0.0	0.0	2.1	8.3	12.3	8.3
Outside (not work or home)	6.9 ± 8.6	0.0	0.0	2.8	12.5	20.8	22.9

3.2 Geospatial Mapping of Study Area

Figure 5 (Left panel) shows the 52 Fresno participant home locations in relation to the AB 617 boundary (in green). 20 of the 52 homes were within the AB 617 boundary, but other homes were located in high vulnerability census tracts based on CES scores. **Figure 5 (Right panel)** shows the 52 Fresno participant home locations in relation to census tract boundaries and CES ranking. Most (46 of 52) of the Fresno participants resided in census tracts with CES scores over 75%.

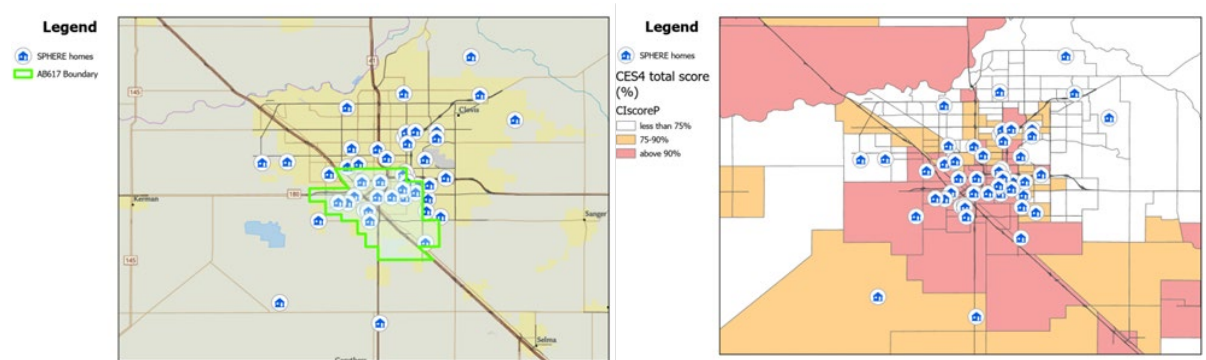


Figure 4: LEFT: Location of Fresno participant homes relative to the AB 617 boundary (in green), RIGHT: Location of Fresno participant homes relative to census tracts and CES sources.

Figure 6 (Left panel) shows the 12 Stockton participant home locations in relation to the AB 617 boundary (in green). Two of the 12 homes were within the AB 617 boundary, but other homes were located in high vulnerability census tracts based on overall CES scores (>75%). **Figure 6 (Right panel)** shows the 12 Stockton participant home locations in relation to census tract boundaries and CES ranking. Most (8 of 12) of the Stockton participants resided in census tracts with overall CES scores over 75%.



Figure 5: LEFT: Location of Stockton participant homes relative to the AB 617 boundary (in green), RIGHT: Location of Stockton participant homes relative to census tracts and CES scores.

3.3 Air pollutant concentrations and noise

3.3.1 Criteria Air Pollutants (PM_{2.5}, CO, NO₂ and O₃)

Table 8 summarizes indoor and outdoor concentrations of criteria air pollutants and corresponding U.S. EPA National Ambient Air Quality Standards (NAAQS) for comparison.⁷ Mean indoor air pollutant concentrations, except for O₃, were higher than outdoor concentrations (indoor PM_{2.5}=14.8 µg/m³, CO (8-hour)=2 ppm, CO (1-hour)=6 ppm, and NO₂ (1-hour)=23 ppb NO₂ versus outdoor PM_{2.5}=11.9 µg/m³, CO (8-hour)=1 ppm, 4 ppm CO (1-hour), and NO₂ (1-hour)=20 ppb, respectively). The outdoor mean O₃ concentration (0.030 ppm) exceeded the indoor mean (0.020 ppm), which is consistent with ozone's reactivity with indoor surfaces.¹³⁰ The mean indoor-to-outdoor (I/O) ratios for PM_{2.5}, NO₂ and O₃ were 2.3, 1.0 and 0.89, respectively.

In most cases, outdoor mean, 90th percentile, and maximum concentrations did not exceed the level of the NAAQS 24-hours standards. The maximum observed 24-hour PM_{2.5} outdoor concentration of 36.9 µg/m³ was higher than the level of the NAAQS 24-hour standard of 35 µg/m³ and the WHO 24-hour guideline of 15 µg/m³.¹³¹ (Note, the NAAQS standards are based on monitoring by equipment that meets the Federal Reference Method (FRM) standards that shows an exceedance of 35 µg/m³ at the 98th percentile, averaged over 3 years, thus, this

result cannot be interpreted as a violation).⁷ Overall, the SJV region is considered out of attainment for PM_{2.5} based on regulatory monitoring⁵.

Table 8: Indoor and outdoor criteria pollutant concentrations and comparison to NAAQS standards.

Criteria Pollutant	NAAQS Averaging Time	Primary NAAQS Standard	Unit	Indoor Mean	Indoor 90 th	Indoor (Max)	Outdoor Mean	Outdoor 90 th	Outdoor (Max)	Outdoor Exceedance (y/n)
PM _{2.5}	24 hr ^a	35.0	µg/m ³	14.8	31.1	84.9	11.9	26.4	36.9	See text ^a
CO	8 hr	9	ppm	2	4	7	1	2	2	N
CO	1 hr ^b	35	ppm	6	10	23	4	8	10	N
O ₃	8 hr	0.070	ppm	0.020	0.030	0.068	0.030	0.043	0.052	N
NO ₂	1 hr ^b	100	ppb	23	39	67	20	26	32	N

^a Note, the NAAQS standards are based on an exceedance of 35 µg/m³ at the 98th percentile, averaged over 3 years, using an FRM air monitor.⁷ ^bBased on hourly data.

3.3.1.1 Particulate matter < 2.5 micrometers (PM_{2.5})

Table 9 summarizes the distribution of indoor PM_{2.5} concentrations by key CES indicators and residence in an AB 617 community. Indoor PM_{2.5} concentrations were significantly higher in areas with a higher overall CES scores (≥75th percentile compared with locations <75th percentile (mean = 17.9 µg/m³ versus 6.7 µg/m³, p-value < 0.01) respectively. Similarly, indoor PM_{2.5} levels in homes in AB 617 communities were also higher (mean = 22.1 µg/m³ versus 9.9 µg/m³, p-value < 0.01, respectively). The mean PM_{2.5} concentrations were slightly elevated in homes with CES diesel indicator scores ≥75th percentile versus < 75th (15.6 µg/m³ and 13.2 µg/m³, respectively), albeit not statistically significant. Overall, the results suggest that communities with higher environmental burdens, especially those identified through CES scores and AB 617 designation, tended to have higher indoor PM_{2.5} concentrations.

Table 9: Association between indoor PM_{2.5} (µg/m³) levels and CalEnviroScreen (CES) traffic indicators and residence in a AB 617 community.

Variable	Category	Count	Mean	25 th	50 th	75 th	90 th	Min	Max	p-value ^a
CES Overall Score	< 75 th Percentile	23	6.7	2.2	6.1	8.8	14.6	0.7	18.9	<0.01
	≥ 75 th Percentile	41	17.9	5.3	14.4	23.2	32.3	0.7	84.9	
CES Diesel Emission Percentile	< 75 th Percentile	52	13.2	3.6	8.8	16.9	29.5	0.7	84.9	0.78
	≥ 75 th Percentile	12	15.6	5.9	21.1	23.2	32.1	3.6	32.1	
CES Traffic Density Indicator	< 75 th Percentile	56	14.3	4.9	9.9	18.0	29.4	0.7	84.9	0.26
	≥ 75 th Percentile	8	8.5	2.2	5.3	6.1	32.1	0.7	32.1	
AB 617	No	42	9.9	3.6	6.6	14.4	29.4	0.7	42.7	<0.01
	Yes	22	22.1	8.4	18.0	23.2	58.2	3.5	84.9	

^aKruskal-Wallis p-values.

Table 10 summarizes the distribution of outdoor PM_{2.5} concentrations stratified by CalEnviroScreen (CES) indicators and AB 617 designation. PM_{2.5} concentrations in neighborhoods with overall CES scores ≥75th percentile were significantly higher outdoors compared with neighborhoods with CES scores < 75th (mean: 12.7 µg/m³ versus 7.4 µg/m³; respectively, p < 0.01). A similar pattern was observed for the CES diesel indicator, with PM_{2.5} levels in neighborhoods with the overall CES score ≥75th percentile more than double than neighborhoods <75th percentile (20.2 µg/m³ versus 8.8 µg/m³; respectively, p < 0.01).

PM_{2.5} levels were slightly higher in neighborhoods with higher traffic density (mean: 12.7 µg/m³ versus 10.4 µg/m³), although these differences were not statistically significant. Significantly higher outdoor PM_{2.5} concentrations were found in AB 617–designated communities (mean: 13.9 µg/m³ versus 9.1 µg/m³; respectively, p < 0.05) (**Table 9**).

Overall, these findings suggest that communities with higher environmental burden scores and/or AB 617 designation experience elevated PM_{2.5} exposure, underscoring ongoing environmental justice concerns.

Table 10: Association between outdoor PM_{2.5} (µg/m³) levels and CalEnviroScreen (CES) traffic indicators and residence in an AB 617 community.

Variable	Category	Count	Mean	25 th	50 th	75 th	90 th	Min	Max	p-value ^a
CES Overall Score	< 75 th Percentile	23	7.4	2.5	6.6	7.6	18.4	0.7	34.5	<0.01
	≥ 75 th Percentile	41	12.7	5.4	9.4	18.7	26.0	0.7	36.9	
CES Diesel Emission Percentile	< 75 th Percentile	52	8.8	3.8	7.1	10.7	19.5	0.7	34.5	<0.01
	≥ 75 th Percentile	12	20.2	10.7	18.7	27.7	36.8	9.1	36.9	
CES Traffic Density Indicator	< 75 th Percentile	56	10.4	4.1	7.6	13.4	23.2	0.7	36.9	0.61
	≥ 75 th Percentile	8	12.7	5.4	9.1	18.4	32.1	0.9	32.1	
AB 617	No	42	9.1	3.7	7.5	9.4	19.5	0.7	34.5	0.04
	Yes	22	13.9	5.4	12.7	21.7	27.7	0.7	36.9	

^aKruskal-Wallis p-values.

Table 11 summarizes the relationships between 24-hour average indoor and outdoor PM_{2.5} concentrations and traffic volumes, measured by Daily Vehicle Miles Traveled (DVMT), across multiple concentric buffer zones ranging from 100 meters to 1000 meters.¹²⁶ As buffer size increased, the correlation between indoor PM_{2.5} and DVMT increased, from a coefficient of 0.04 at 100 meters to 0.21 at 1000 meters. Correspondingly, indoor p-values decreased from 0.75 to 0.09, indicating a weak but increasing association between indoor PM_{2.5} levels and nearby traffic volumes at larger spatial scales.

Outdoor PM_{2.5} showed a stronger relationship with traffic volume. The correlation with DVMT also increased with buffer size, increasing from 0.07 at 100 meters to 0.35 at 1000 meters. The associated p-values similarly decreased, from 0.62 at 100 meters to 0.05 at 1000 meters, reaching the threshold for statistical significance. This finding suggests a modest relationship between outdoor PM_{2.5} concentrations and traffic volumes, particularly at broader spatial scales. Overall, the data indicate that outdoor PM_{2.5} concentrations are more closely linked to traffic volumes than indoor concentrations. However, as buffer size increases, the correlation increases in both indoor and outdoor environments, highlighting the importance of considering broader spatial scales when evaluating the impact of traffic-related emissions on hyper-local air quality.

Table 11: Association between indoor and outdoor PM_{2.5} and Daily Vehicle Miles Traveled^a (DVMT).

Time	Buffer Size (meters)	Indoor average PM _{2.5} Correlation Coefficient ^b	Indoor p-value	DVMT Indoor	Buffer Size (meters)	Outdoor average PM _{2.5} Correlation Coefficient ^b	Outdoor p-value	DVMT Outdoor
24-hour	100	0.04	0.75	442	100	0.07	0.62	436
	250	0.07	0.57	4906	250	0.01	0.94	4847
	500	0.08	0.55	26019	500	0.14	0.28	25711
	750	0.19	0.12	69860	750	0.23	0.07	69036
	1000	0.21	0.09	128354	1000	0.35	0.05	126840

^aSource for traffic metrics: Tracking California traffic tool (see text).

^bSpearman correlation coefficient (rho) and p-values are presented.

Table 12 presents the correlation between indoor PM_{2.5} concentrations and residential proximity to Highway SR-99. We observed a statistically significant inverse correlation (rho = -0.47, p = 0.001), indicating that indoor PM_{2.5} concentrations tend to be higher in homes located closer to the highway. This finding suggests that proximity to a major traffic corridor like SR-99 may play a notable role in influencing indoor air quality through traffic-related pollutant infiltration.

Table 12: Correlation between Indoor PM_{2.5} and Residential Proximity (meters) to Highway SR-99.

N	Spearman rho	p-value
60	-0.47	0.001

Table 13 presents the correlation between outdoor PM_{2.5} concentrations and residential proximity to Highway SR-99. The correlation coefficient was -0.17, albeit not significant (p-value=0.23). We observed similar patterns for indoor and outdoor black carbon measurements (see black carbon discussion below). It is counterintuitive that indoor black carbon and PM_{2.5} would be more strongly correlated with the distance to Highway SR-99 when compared to the correlations for outdoor black carbon and PM_{2.5}. While we used a Spearman rank correlation to compare our pollution metrics with residential proximity, there may still be outliers that are biasing the relationship. The range of indoor PM_{2.5} is much wider than outdoor PM_{2.5}, lending support to this theory. Additionally, there are a different number of households in each subset, thus the two analyses are not entirely comparable.

Table 13: Correlation between outdoor PM_{2.5} and Residential Proximity (meters) to highway SR-99.

N	Spearman rho	p-value
54	-0.17	0.23

Table 13 presents indoor PM_{2.5} concentrations (µg/m³) in relation to household characteristics. Most household factors were not significantly associated with indoor PM_{2.5} levels. Indoor PM_{2.5} concentrations did not differ significantly by stove type, with mean levels of 13.8 µg/m³ in homes using gas stoves and 12.9 µg/m³ in homes using electric stoves (p = 0.1). Homes where smoking was reported had a higher indoor PM_{2.5} (mean: 28.9 µg/m³) compared with non-smoking households (12.1 µg/m³), although the difference was not statistically significant (p = 0.13), likely due to the small number of homes with smokers. Income level showed a trend of decreasing PM_{2.5} with higher income, with households earning <\$15,000 having the highest mean (19.7 µg/m³) and those earning >\$75,000 having the lowest (6.7 µg/m³), but this trend was not statistically significant (p = 0.10). Ethnicity and home size were also not significantly associated with PM_{2.5} levels. However, homes with higher density (≥1 resident per room) had slightly higher PM_{2.5} (mean: 13.8 µg/m³) compared with those with lower density (13.2 µg/m³, p = 0.05) but in absolute terms the difference was minor. Reported use of candle, incense, or sage was significantly associated with higher PM_{2.5} levels (p = 0.01), with the highest mean (23.3 µg/m³) observed in homes reporting use three or more times per week. Finally, renters experienced significantly higher indoor PM_{2.5} concentrations (18.0 µg/m³) compared with homeowners (8.3 µg/m³, p = 0.01). In summary, frequent use of combustion products and renting were significantly associated with elevated indoor PM_{2.5} concentrations, while other household factors showed less significance.

Table 14: Indoor PM_{2.5} concentrations (µg/m³) stratified by household characteristics (n=54).

Variable	Category	Count	Mean	SD	90 th	p-value*
Type of stove	Any Gas	39	13.8	16.1	27.6	0.1
	Electric	19	12.9	12.0	32.2	
Does anyone smoke?	No	53	12.1	11.5	28.2	0.1
	Yes	5	28.9	32.5	61.8	
Income	< \$15,000	10	19.7	24.8	35.0	0.1
	≥ \$15,000 - \$30,000	22	15.5	13.9	28.8	
	≥ \$30,000 - \$75,000	11	13.3	11.1	32.1	
	> \$75,000	15	6.7	5.8	15.1	
Race Category	Hispanic	39	13.9	12.0	29.4	0.2
	Non-Hispanic	19	12.9	19.6	28.2	
Size of Home	Larger Home	16	21.2	23.4	50.5	0.2
	Smaller Home	42	10.6	8.5	21.1	
Density (# of residents / # of rooms)	≥ 1	29	13.8	9.2	27.7	0.1
	< 1	29	13.2	19	32.1	

Table 14 (cont.). Indoor PM_{2.5} concentrations (µg/m³) stratified by household characteristics (n=54).

Variable	Category	Count	Mean	SD	90th	p-value*
Frequency of candle, incense, or sage use	3 or more times a week	13	23.3	21.9	40.6	0.01
	A few times a week	10	6.7	5.5	14.7	
	Less than once a week	35	11.8	11.6	26.1	
Rent or own home?	Rent	31	18.0	17.9	32.1	0.01
	Own	27	8.3	7.5	16.4	

*Wilcoxon rank sum or Kruskal Wallis test p-value.

Table 15 summarizes outdoor PM_{2.5} concentrations (µg/m³) across selected household characteristics. Income was the only factor significantly associated with outdoor PM_{2.5} levels (p = 0.02); households earning more than \$75,000 had significantly lower outdoor PM_{2.5} (5.3 µg/m³) compared to the other income groups. Ethnicity was not significantly associated with outdoor PM_{2.5} levels; although Hispanic households had somewhat higher levels (mean=11.4 µg/m³ versus 8.9 µg/m³, respectively). Renters also had somewhat higher average outdoor PM_{2.5} (12.2 µg/m³) compared with homeowners (9.1 µg/m³), but this difference was not statistically significant (p = 0.38).

Table 15: Outdoor PM_{2.5} concentrations (µg/m³) and household characteristics (n=54).

Variable	Category	Count	Mean	SD	90 th	p-value*
Has grill?	No	3	10.9	9.2	22.0	0.80
	Yes	5	9	3.3	12.3	
Income	< \$15,000	6	11.1	9.4	20.2	0.02
	≥ \$15,000 - \$30,000	22	12.6	8.7	23.1	
	≥ \$30,000 - \$75,000	12	12.8	11.6	31.5	
	> \$75,000	14	5.3	2.1	7.6	
Ethnicity	Hispanic	37	11.4	9.1	24.3	0.20
	Non-Hispanic	17	9	8.3	19.7	
Rent or own home?	Rent	27	12.2	9.8	25.0	0.34
	Own	27	9.1	7.5	14.9	

*Wilcoxon rank sum or Kruskal Wallis test p-value.

Personal daily PM_{2.5} exposures, as measured by the Atmotube Pro device (**Table 16**), were similar to both indoor and outdoor levels recorded by the Sensit instrument (**Tables 8 and 16**).

Table 16: Summary of 24-hour average PM_{2.5} indoor, outdoor, and personal concentrations (µg/m³).

	n	Mean	SD	Median	90 th	Min	Max	Instrument
Indoor	67	14.8	16.3	9.5	31.1	0.71	84.9	Sensit
Outdoor	62	11.9	9.5	8.4	26.4	0.71	36.9	Sensit
Personal	61	12.1	17.5	6.9	20.9	1.0	92.3	Atmotube

3.3.1.1.1 Fine Particulate Matter (PM_{2.5}) Concentrations and Respiratory Health Outcomes

Tables 17 and 18 present indoor and outdoor PM_{2.5} concentrations stratified by reported adult and child respiratory symptoms. Analysis of indoor and outdoor PM_{2.5} concentrations showed limited evidence of associations with self-reported respiratory symptoms among adults and children. For adults (**Table 17**), there were no significant differences among those reporting wheeze, chronic cough, or asthma compared to those without symptoms. A significant finding was observed among adults who reported regularly using asthma medication, whose homes had higher mean indoor PM_{2.5} concentrations (17.1 µg/m³) compared with those who did not use medication (4.3 µg/m³; p = 0.03), suggesting possible increased exposure among households managing more severe respiratory conditions (note, we observed a similar pattern for NO₂ (see below)).

Table 17: Associations between indoor and outdoor PM_{2.5} concentrations and adult respiratory symptoms.

Variable	Category	Indoor N	Indoor Mean (µg/m ³)	Indoor p-value*	Outdoor N	Outdoor Mean (µg/m ³)	Outdoor p-value*
Wheezing or whistling in your chest at any time	No	41	13.8	0.79	39	10.1	0.60
	Yes	17	12.8		15	12.0	
Wheezing or whistling in your chest without a cold or illness	No	7	13.9	0.70	7	11.7	0.54
	Yes	9	12.8		8	12.2	
	DK	1	4.9		0	NA	
Chronic cough without a cold or illness at any time	No	49	14.7	0.19	46	11.0	0.37
	Yes	9	7.2		8	8.35	
Doctor ever said that you have asthma	No	47	14.4	0.21	44	10.3	0.77
	Yes	10	10.7		10	11.9	
	DK	1	0.72		0	NA	
Regularly take asthma meds	No	5	4.3	0.03	4	4.3	0.17
	Yes	5	17.1		6	17.0	

Abbreviations: DK = Don't Know; NA= Not applicable.

* Wilcoxon rank sum or Kruskal-Wallis test p-value.

Among children (**Table 18**), fine particulate matter (PM_{2.5}) concentrations and child respiratory symptoms showed no statistically significant relationships in this study sample. Mean indoor PM_{2.5} concentrations were slightly higher in homes of children without reported respiratory symptoms compared to those with symptoms, though the differences were small. For example, indoor PM_{2.5} averaged 14.7 µg/m³ for children without wheezing compared to 10.2 µg/m³ for those who reported wheezing, and 14.4 µg/m³ for children without asthma compared to 11.5 µg/m³ for those diagnosed with asthma.

Outdoor PM_{2.5} concentrations were relatively consistent across all symptom categories, averaging around 10–11 µg/m³, with no meaningful variation between groups. Although the small sample size limited statistical power, these findings suggest that in this population, short-term differences in measured indoor or outdoor PM_{2.5} concentrations were not strongly associated with parent-reported child respiratory symptoms such as wheezing, coughing, or asthma diagnosis.

Table 18: Associations between indoor and outdoor PM_{2.5} concentrations and child respiratory symptoms.

Variable	Category	Indoor N	Indoor Mean (µg/m ³)	Indoor p-value*	Outdoor N	Outdoor Mean (µg/m ³)	Outdoor p-value*
Wheezing or whistling in your chest at any time	No	43	14.7	0.64	40	10.6	0.43
	Yes	15	10.2		14	10.6	
Wheezing or whistling in your chest without a cold or illness	No	6	9.7	0.95	5	8.1	0.44
	Yes	9	10.6		9	12.0	
Chronic cough without a cold or illness at any time	No	48	14.7	0.27	45	10.8	0.69
	Yes	9	8.3		8	10.5	
	DK	1	3.5		1	4.2	
Doctor ever said that you have asthma	No	41	14.4	0.93	40	10.3	0.80
	Yes	17	11.5		14	11.4	
Regularly take asthma medications	No	3	9.4	0.98	2	5.2	0.86
	Yes	12	12.1		10	12.7	
	DK	2	10.8		2	11.0	

Abbreviation: DK = Don't Know.

* Wilcoxon rank sum or Kruskal-Wallis test p-value.

3.3.1.2 Nitrogen Dioxide (NO₂)

Table 19 presents indoor NO₂ concentrations (ppb) stratified by CalEnviroScreen (CES) indicators and residence in an AB 617 community. Locations where the overall CES score was at or above the 75th percentile had a slightly higher mean concentration (15.7 ppb) compared with those below the 75th percentile (14.5 ppb) ($p < 0.01$). There was no association between location diesel emission ranking or traffic density and indoor NO₂. Residence in an AB 617 community was significantly associated with higher indoor NO₂ concentrations (16.8 ppb versus 14.6 ppb, $p < 0.01$).

Table 19: Association between indoor NO₂ (ppb) levels and CalEnviroScreen (CES) traffic indicators and residence in a AB 617 community.

Variable	Category	Count	Mean	25 th	Median	75 th	90 th	Min	Max
CES Overall Score	< 75 th Percentile	23	14.5	14.1	14.1	14.2	15.4	14.1	17.6
	≥ 75 th Percentile	41	15.7**	14.2	14.7	16.5	19.6	14.1	22.9
CES Diesel Emission Percentile	< 75 th Percentile	52	15.3	14.1	14.2	15.4	17.6	14.1	22.9
	≥ 75 th Percentile	12	15.1	14.1	14.4	15.6	16.5	14.1	18.0
CES Traffic Density Indicator	< 75 th Percentile	56	15.3	14.1	14.3	15.6	17.6	14.1	22.9
	≥ 75 th Percentile	8	14.5	14.1	14.2	15	15.5	14.1	15.5
AB 617	No	42	14.6	14.1	14.2	14.8	15.4	14.1	17.6
	Yes	22	16.8**	14.7	15.9	18.0	20.4	14.1	22.9

**Wilcoxon rank sum test p-value <0.01.

Table 20 presents outdoor NO₂ concentrations (ppb) stratified by CalEnviroScreen (CES) indicators and residence in an AB 617 community. Statistically significant differences were observed for the overall CES score, CES diesel emission ranking, and residence in an AB 617 community. Homes in areas at or above the 75th percentile for CES overall score had a somewhat higher mean NO₂ concentration (15.3 ppb) compared with those below the 75th percentile (14.5 ppb) ($p < 0.05$). A similar trend was seen for the CES diesel emission ranking (16.1 ppb versus 14.8 ppb, $p < 0.05$). In contrast, traffic density was not significantly associated with NO₂ levels. Residence in an AB 617 community was significantly associated with slightly higher NO₂ concentrations (mean=15.5 ppb versus 14.7 ppb). In summary, outdoor NO₂ concentrations tended to be higher in areas with higher overall CES score, diesel emission ranking, and residence in an AB 617 community.

Table 20: Association between outdoor NO₂ (ppb) levels and CalEnviroScreen (CES) traffic indicators and residence in a AB 617 community.

Variable	Category	Count	Mean	25 th	Median	75 th	90 th	Min	Max
CES Overall Score	< 75 th Percentile	23	14.5	14.2	14.4	14.8	14.9	14.1	15.4
	≥ 75 th Percentile	41	15.3*	14.3	14.9	15.8	16.9	14.1	19.1
CES Diesel Emission Percentile	< 75 th Percentile	52	14.8	14.2	14.5	15	16.2	14.1	17.4
	≥ 75 th Percentile	12	16.1*	14.9	15.3	16.9	19.1	14.1	19.1
CES Traffic Density Indicator	< 75 th Percentile	56	14.9	14.2	14.6	15.3	16.4	14.1	18.4
	≥ 75 th Percentile	8	15.4	14.5	14.8	15.3	19.1	14.1	19.1
AB 617	No	42	14.7	14.2	14.4	14.8	15.4	14.1	19.1
	Yes	22	15.5**	14.8	15.3	16.3	17.4	14.2	18.4

* Wilcoxon rank sum test p-value ≥0.01 & <0.05.

**p-value <0.01.

Table 21 presents the correlation between indoor and outdoor NO₂ concentrations and residential proximity to Highway SR-99. Statistically significant inverse correlations were observed (rho = -0.57 and -0.44, respectively, p < 0.001), indicating that homes located closer to the highway tended to have higher NO₂ levels. These inverse relationships suggest that traffic-related emissions from SR-99 may influence indoor air quality in nearby residences.

Table 21: Correlation between indoor and outdoor NO₂ and residential proximity (meters) to highway SR-99.

N	Spearman rho	p-value
Indoor (n=60)	-0.57	<0.001
Outdoor (n=54)	-0.44	<0.001

Table 22 summarizes indoor NO₂ concentrations stratified by household characteristics. Stove type was significantly associated with indoor NO₂ levels, with homes using gas stoves having higher concentrations (mean: 15.71 µg/m³) compared with those using electric stoves (mean: 14.30 µg/m³, p < 0.01). Reported smoking status was not significantly associated with NO₂. Households earning between \$15,000–\$30,000 had the highest mean NO₂ levels (16.1 µg/m³) compared with those earning over \$75,000 (14.4 µg/m³) (p<0.01). Hispanic households were exposed to slightly higher indoor NO₂ concentrations (mean: 15.6 versus 14.5 µg/m³, p=0.04). Size of home, occupancy density, frequency of burning incense or candles, garage status, and homeownership were not associated with indoor NO₂ levels. In summary, stove type (gas), income, and ethnicity were associated with higher indoor NO₂ concentrations.

Table 22: Indoor NO₂ concentrations (µg/m³) stratified by household characteristics (n=54).

Variable	Category	Count	Mean	SD	90 th	p-value*
Type of stove	Any Gas	39	15.7	2.0	18.3	<0.01
	Electric	19	14.3	0.4	14.5	
Does anyone smoke?	No	53	15.3	1.9	17.5	0.7
	Yes	5	15.0	1.1	16.3	
Income	< \$15,000	10	15.6	2.1	17.6	0.01
	≥ \$15,000 - \$30,000	22	16.1	2.3	19.4	
	≥ \$30,000 - \$75,000	11	14.3	0.4	14.4	
	> \$75,000	15	14.4	0.4	15.1	
Race Category	Hispanic	39	15.6	2.1	18.3	0.04
	Non-Hispanic	19	14.5	0.7	15.6	
Size of Home	Larger Home	16	15.6	2.5	18.4	0.9
	Smaller Home	42	15.1	1.5	17.3	
Resident density (number residents/ number rooms)	≥ 1	29	15.5	1.8	18.3	0.1
	< 1	29	15	1.8	17.2	
Frequency of candle, incense, or sage use	3 or more times a week	13	15.4	2.5	17.7	0.8
	A few times a week	10	14.8	1.1	15.5	
	Less than once a week	35	15.3	1.7	17.2	
Rent or own home?	Rent	31	15.7	2.3	19.6	0.2
	Own	27	14.8	1	15.6	

*Wilcoxon rank sum or Kruskal-Wallis test p-values.

Table 23 summarizes outdoor NO₂ concentrations (µg/m³) stratified by household characteristics. Households earning \$15,000–\$30,000 had somewhat higher levels (mean=15.3 µg/m³) compared with those earning over \$75,000 (14.3 µg/m³) (p=0.01). Other household characteristics were not significantly associated with NO₂ concentrations.

Table 23: Outdoor NO₂ concentrations (µg/m³) and potential determinants of exposure (n=54).

Variable	Category	Count	Mean	SD	90 th	p-value
Has grill	No	14	15.1	1.2	16.4	0.40
	Yes	40	14.9	1.1	16.3	
Income	< \$15,000	6	15.1	0.5	15.4	0.01
	≥ \$15,000 - \$30,000	22	15.3	1.2	16.9	
	≥ \$30,000 - \$75,000	12	15.1	1.5	16.3	
	> \$75,000	14	14.3	0.3	14.8	
Race Category	Hispanic	37	15.1	1.0	16.5	0.10
	Non-Hispanic	17	14.8	1.2	15.3	
Rent or own home?	Rent	27	15.1	1.2	16.5	0.30
	Own	27	14.8	0.9	16.3	

*Wilcoxon rank sum or Kruskal-Wallis test p-values.

3.3.1.2.1 Nitrogen Dioxide (NO₂) Concentrations and Respiratory Health Outcomes

Tables 24 and 25 present indoor and outdoor NO₂ concentrations stratified by reported adult and child respiratory symptoms. Measured indoor and outdoor NO₂ concentrations showed little variation across self-reported respiratory health outcomes among adults and children. For adults (Table 24), mean indoor NO₂ concentrations were similar among participants with and without respiratory symptoms, ranging from 14 - 16 ppb across categories. No statistically significant associations were observed for wheezing, chronic cough, or asthma diagnosis. The only variable approaching significance was asthma medication use, with adults who reported regular use of prescribed asthma medication exhibiting slightly higher mean indoor NO₂ levels (16.7 ppb) compared with those who did not (14.8 ppb; p = 0.05). (As noted above, we also observed significant associations between PM_{2.5} exposure and adult asthma medication use (Table 16)). Outdoor NO₂ concentrations followed a similar pattern, averaging 15 ppb, with no meaningful differences by respiratory status.

Among children (Table 25), mean indoor NO₂ levels ranged from 14.3 to 16.5 ppb, while outdoor concentrations averaged ~15 ppb across all symptom categories. Although children with wheezing or asthma tended to have marginally higher indoor NO₂ concentrations (15.9 ppb for asthma diagnosis vs. 15.0 ppb for no asthma), none of these differences were statistically significant. Overall, these results indicate that short-term indoor and outdoor NO₂ concentrations were not strongly associated with short-term self-reported respiratory symptoms in this sample of San Joaquin Valley households. Slightly higher indoor NO₂ among adults using asthma medication may reflect the presence of combustion-related indoor sources in homes with existing respiratory health concerns.

Table 24: Associations Between Indoor and Outdoor NO₂ Concentrations and Adult Respiratory Symptoms.

Variable	Category	Indoor N	Indoor Mean (ppb)	Indoor p-value*	Outdoor N	Outdoor Mean (ppb)	Outdoor p-value*
Wheezing or whistling in your chest at any time	No	41	15.3	0.89	39	14.8	0.27
	Yes	17	15.2		15	15.4	
Wheezing or whistling in your chest without a cold or illness	No	7	14.4	0.12	7	15.1	0.28
	Yes	9	15.9		8	15.8	
	DK	1	14.1		0	NA	
Chronic cough without a cold or illness at any time	No	49	15.2	0.20	46	14.9	0.17
	Yes	9	15.6		8	15.5	
Doctor ever said that you have asthma	No	47	15.2	0.46	44	14.8	0.13
	Yes	10	15.7		10	15.6	
	DK	1	14.1		0	NA	
Regularly takes asthma meds	No	5	14.8	0.05	4	14.6	0.11
	Yes	5	16.7		6	16.2	

Abbreviations: DK = Don't Know; NA = Not applicable.

*Wilcoxon rank sum or Kruskal-Wallis test p-values.

Table 25: Associations Between Indoor and Outdoor NO₂ Concentrations and Child Respiratory Symptoms.

Variable	Category	Indoor N	Indoor Mean (ppb)	Indoor p-value	Outdoor N	Outdoor Mean (ppb)	Outdoor p-value
Wheezing or whistling in your chest at any time	No	43	15.0	0.07	40	14.9	0.51
	Yes	15	15.9		14	15.2	
Wheezing or whistling in your chest without a cold or illness	No	6	15.6	0.63	5	14.5	0.26
	Yes	9	16.0		9	15.6	
Chronic cough without a cold or illness at any time	No	48	15.2	0.24	45	15.0	0.47
	Yes	9	15.4		8	14.9	
	DK	1	17.3		1	15.3	
Doctor ever said that you have asthma	No	41	15.0	0.13	40	14.9	0.69
	Yes	17	15.9		14	15.1	
Regularly takes asthma meds	No	3	14.3	0.57	2	14.2	0.06
	Yes	12	16.5		10	15.5	
	DK	2	14.7		2	14.1	

Abbreviations: DK = Don't Know.

*Wilcoxon rank sum or Kruskal-Wallis test p-values.

3.3.1.3 Ozone (O₃)

Table 26 summarizes indoor O₃ concentrations (ppm) stratified by CalEnviroScreen (CES) indicators and residence in an AB 617 community. Overall, there were no significant differences observed.

Table 26: Indoor O₃ levels (ppm) and stratified by CalEnviroScreen (CES) traffic indicators and residence in an AB 617 community.

Variable	Category	Count	Mean	25 th	Median	75 th	90 th	Max
CES Overall Score	< 75 th Percentile	23	0.024	0.014	0.018	0.029	0.036	0.068
	≥ 75 th Percentile	41	0.018	0.015	0.016	0.020	0.022	0.039
CES Diesel Emission Percentile	< 75 th Percentile	52	0.021	0.014	0.016	0.022	0.031	0.068
	≥ 75 th Percentile	12	0.018	0.015	0.017	0.019	0.021	0.026
CES Traffic Density Indicator	< 75 th Percentile	56	0.020	0.014	0.016	0.021	0.031	0.068
	≥ 75 th Percentile	8	0.020	0.016	0.017	0.022	0.027	0.030
AB 617	No	42	0.021	0.014	0.015	0.023	0.033	0.068
	Yes	22	0.019	0.016	0.018	0.020	0.022	0.030

Table 27 summarizes outdoor O₃ concentrations (ppm) by CalEnviroScreen (CES) indicators and AB 617 program status. Similar to the trends for indoor O₃ concentrations, overall, there were no significant differences observed.

Table 27: Outdoor O₃ levels (ppm) stratified by CalEnviroScreen (CES) traffic indicators and residence in an AB 617 community.

Variable	Category	Count	Mean	25 th	Median	75 th	90 th	Max
CES Overall Score	< 75 th Percentile	23	0.029	0.016	0.031	0.039	0.041	0.046
	≥ 75 th Percentile	41	0.033	0.026	0.034	0.040	0.045	0.052
CES Diesel Emission Percentile	< 75 th Percentile	52	0.032	0.023	0.034	0.040	0.045	0.052
	≥ 75 th Percentile	12	0.028	0.023	0.026	0.034	0.038	0.042
CES Traffic Density Indicator	< 75 th Percentile	56	0.032	0.025	0.034	0.040	0.044	0.052
	≥ 75 th Percentile	8	0.026	0.020	0.023	0.031	0.039	0.041
AB 617	No	42	0.030	0.022	0.033	0.039	0.041	0.046
	Yes	22	0.034	0.027	0.035	0.043	0.050	0.052

3.3.1.3.1 Ozone (O₃) Concentrations and Respiratory Health Outcomes

Associations between indoor and outdoor O₃ concentrations and self-reported respiratory outcomes were generally weak and not statistically significant for most variables. For adults (**Table 28**), participants who reported ever having wheezing or whistling in the chest, cough, or asthma had mean indoor O₃ concentrations similar to those who did not report these conditions (range = 18–25 ppb; $p \geq 0.11$). Outdoor concentrations averaged roughly 30 ppb across categories, with no meaningful differences by respiratory status.

Among children (**Table 29**), a modest pattern emerged for cough: children with a history of coughing without having a cold had higher mean indoor O₃ levels (31 ppb) than those without (18 ppb), a difference that reached statistical significance ($p = 0.02$). Similar but non-significant trends were observed for wheeze and asthma medication use. Outdoor O₃ levels averaged 30–33 ppb and did not differ significantly by symptom category.

Overall, these results suggest that short-term O₃ concentrations measured indoors and outdoors were not strongly associated with most self-reported respiratory outcomes in this small sample, although elevated indoor O₃ may contribute to cough or airway irritation among sensitive children.

Table 28: Associations between indoor and outdoor O₃ concentrations and adult respiratory symptoms.

Variable	Category	Indoor N	Indoor Mean (ppb)	Indoor p-value*	Outdoor N	Outdoor Mean (ppb)	Outdoor p-value*
Wheezing or whistling in your chest at any time	No	43	18.4	0.18	39	33.0	0.18
	Yes	17	24.5		15	27.7	
Wheezing or whistling in your chest without a cold or illness	No	7	29.0	0.61	7	26.2	0.61
	Yes	9	22.0		8	29.1	
	DK	1	14.8		NA	NA	
Chronic cough without a cold or illness at any time	No	51	19.9	0.11	46	31.1	0.60
	Yes	9	21.2	0.11	8	33.6	
Doctor ever said that you have asthma	No	49	20.6	0.49	44	31.6	0.96
	Yes	10	18.3		10	30.9	
	DK	1	14.2		NA	NA	
Regularly take asthma meds	No	5	16.3	0.22	4	33.0	0.61
	Yes	5	20.2	0.22	6	29.6	

Abbreviation: DK = Don't Know.

*Kruskal-Wallis test p-value.

Table 29: Child Respiratory Health Characteristics and O₃ concentrations.

Variable	Category	Indoor N	Indoor Mean (ppb)	Indoor p-value*	Outdoor N	Outdoor Mean (ppb)	Outdoor p-value*
Wheezing or whistling in your chest at any time	No	45	18.2	0.10	40	32.2	0.37
	Yes	15	25.9		14	29.4	
Wheezing or whistling in your chest without a cold or illness	No	6	18.8	0.11	5	34.5	0.29
	Yes	9	30.7		9	26.6	
Chronic cough without a cold or illness at any time	No	50	18.2	0.02	45	32.6	0.02
	Yes	9	31.1		8	22.9	
	DK	1	14.6		1	52.1	
Doctor ever said that you have asthma	No	43	19.4	0.46	40	31.5	0.98
	Yes	17	22.0		14	31.5	
Regularly take asthma meds	No	3	14.6	0.08	2	34.9	0.68
	Yes	12	23.9		10	29.5	
	DK	2	21.6		2	38.3	

Abbreviation: DK = Don't Know

*Kruskal-Wallis test p-value.

3.3.2 Black Carbon

Table 30 presents 24-hour average black carbon concentrations ($\mu\text{g}/\text{m}^3$) measured in indoor and outdoor environments. Indoor samples were collected from 54 homes, while outdoor samples were collected from 44 homes. Across all statistical metrics, outdoor black carbon concentrations exceeded indoor levels.

For indoor samples (N = 59), the mean concentration was $0.42 \mu\text{g}/\text{m}^3$ (SD: 0.33), with a median of $0.34 \mu\text{g}/\text{m}^3$, a 90th percentile value of $0.90 \mu\text{g}/\text{m}^3$, and a range from 0.05 to $1.51 \mu\text{g}/\text{m}^3$. Outdoor samples (N = 50) showed a higher mean concentration of $0.65 \mu\text{g}/\text{m}^3$ (SD: 0.43), a median of $0.58 \mu\text{g}/\text{m}^3$, a 90th percentile of $1.15 \mu\text{g}/\text{m}^3$, and a range from 0.10 to $1.95 \mu\text{g}/\text{m}^3$.

These results indicate that participants experienced consistently higher black carbon exposure outdoors compared with indoor environments.

Table 30: Summary of 24-hour average indoor and outdoor black carbon levels ($\mu\text{g}/\text{m}^3$).

Location ^a	N ^b	Mean ^c	SD	Median	90 th	Min	Max
Indoor	59	0.42	0.33	0.34	0.90	0.05	1.51
Outdoor	50	0.65	0.43	0.58	1.15	0.10	1.95

^a Indoor sample measurements from 54 homes; outdoor samples measurements from 44 homes.

^b Missing information due to timestamp error/equipment error. ^c Repeat samples were collected from five homes after 30 days. Values for repeat samples were treated as independent measurements.

Table 31 summarizes indoor black carbon concentrations ($\mu\text{g}/\text{m}^3$) by household characteristics ($n = 54$), with p-values based on Wilcoxon rank sum or Kruskal–Wallis tests. Among household characteristics, income, race, home density, and homeownership showed several statistically significant differences in indoor black carbon levels. Homes with annual incomes $< \$15,000$ had higher indoor black carbon (mean (SD): $0.39 (0.18) \mu\text{g}/\text{m}^3$) than those in the highest income group ($> \$75,000$, mean: $0.17 (0.06) \mu\text{g}/\text{m}^3$; $p < 0.01$). Hispanic households had higher mean levels ($0.38 \mu\text{g}/\text{m}^3$, SD: 0.19) than non-Hispanic households ($0.31 \mu\text{g}/\text{m}^3$, SD: 0.35 ; $p = 0.01$). Households with higher density (≥ 1 resident per room) had greater indoor black carbon (mean (SD): $0.42 (0.29) \mu\text{g}/\text{m}^3$) than those with lower density (< 1 resident per room) (mean (SD): $0.29 (0.20) \mu\text{g}/\text{m}^3$; $p = 0.01$). Similarly, renters had higher concentrations (mean: $0.43 \mu\text{g}/\text{m}^3$, SD: 0.30) compared with owners (mean (SD): $0.26 (0.16) \mu\text{g}/\text{m}^3$; $p = 0.01$). Other characteristics such as type of stove, smoking status, home size, candle/incense use, presence of attached garage showed no statistically significant associations with indoor black carbon levels.

Table 31: Indoor black carbon concentrations ($\mu\text{g}/\text{m}^3$) and household characteristics (n=54).

Variable	Category	Count	Mean	SD	90 th	p-value*
Type of stove	Any Gas	37	0.34	0.17	0.55	0.71
	Electric	17	0.40	0.38	0.93	
Does anyone smoke?	No	50	0.34	0.25	0.54	0.17
	Yes	4	0.50	0.27	0.73	
Income	$< \$15,000$	8	0.39	0.18	0.56	< 0.01
	$\geq \$15,000 - \$30,000$	22	0.44	0.22	0.69	
	$\geq \$30,000 - \$75,000$	11	0.37	0.39	0.42	
	$> \$75,000$	13	0.17	0.06	0.24	
Ethnicity	Hispanic	36	0.38	0.19	0.55	0.01
	Non-Hispanic	18	0.31	0.35	0.71	
Resident density (number residents/ number rooms)	≥ 1	27	0.42	0.29	0.62	0.01
	< 1	27	0.29	0.20	0.54	
Frequency of candle, incense, or sage use	≥ 3 times per week	12	0.51	0.44	0.94	0.31
	A few times per week	8	0.24	0.13	0.39	
	< 1 time per week	34	0.33	0.16	0.49	
Rent or own home?	Rent	30	0.43	0.30	0.76	0.01
	Own	24	0.26	0.16	0.42	

*Wilcoxon rank sum or Kruskal-Wallis test p-value.

Table 32 summarizes outdoor black carbon concentrations ($\mu\text{g}/\text{m}^3$) by household characteristics ($n = 44$). Outdoor black carbon levels varied significantly by income and homeownership. Households earning $< \$15,000$ had the highest mean concentration ($0.73 \mu\text{g}/\text{m}^3$, SD: 0.20), while those earning $> \$75,000$ had the lowest ($0.28 \mu\text{g}/\text{m}^3$, SD: 0.10 ;

p < 0.01). Similarly, renters experienced significantly higher outdoor levels (mean (SD): 0.75 (0.44) $\mu\text{g}/\text{m}^3$) compared to owners (mean (SD): 0.46 (0.31) $\mu\text{g}/\text{m}^3$; p = 0.01). Other variables, including grill ownership and race, showed differences in mean black carbon levels, but they were not statistically significant.

Table 32: Outdoor black carbon concentrations ($\mu\text{g}/\text{m}^3$) and household characteristics (n=44).

Variable	Category	Count	Mean	SD	90 th	p-value ^a
Has grill?	No	13	0.7	0.5	1.0	0.20
	Yes	31	0.6	0.4	0.9	
Income	< \$15,000	6	0.7	0.2	1	<0.01
	\geq \$15,000 - \$30,000	18	0.7	0.4	0.9	
	\geq \$30,000 - \$75,000	10	0.7	0.6	1.6	
	> \$75,000	10	0.3	0.1	0.4	
Race/Ethnicity	Hispanic	30	0.6	0.4	1	0.07
	Non-Hispanic	14	0.5	0.5	1	
Rent or own home?	Rent	22	0.8	0.4	1.0	0.01
	Own	22	0.5	0.3	0.8	

^a Wilcoxon rank sum or Kruskal-Wallis test p-value.

Table 33 summarizes indoor-to-outdoor (I/O) ratios for black carbon concentrations. The dataset includes 42 measurements from 40 unique homes. The I/O ratios had a mean of 0.80 (SD: 0.34), with values ranging from 0.31 to 2.00. In three instances, the I/O ratio substantially exceeded 1.0 (~1.5-2.0), suggesting the potential for indoor sources, but we could not explain these higher ratios based on questionnaire or home inspection information (i.e., BBQ use near the home).

On average, indoor black carbon concentrations were approximately 80% of outdoor levels, suggesting that outdoor air is the primary source of indoor BC contamination, and that the home envelope provides a partial barrier to BC infiltration.

Table 33: Summary of black carbon indoor-to-outdoor (I/O) ratios.

n ^a	Mean	SD	Min	Max
42	0.80	0.34	0.31	2.00

^a Sample measurements are from 40 unique homes.

Table 34 summarizes the associations between 24-hour indoor black carbon concentrations ($\mu\text{g}/\text{m}^3$) and CalEnviroScreen (CES) traffic-related indicators, as well as residence in an AB 617–designated community. Indoor black carbon concentrations were significantly higher in homes located in areas with CES Overall Scores above the 75th percentile (mean (SD): 0.53 (0.37) $\mu\text{g}/\text{m}^3$) compared to homes in areas at or below the 75th percentile (mean (SD): 0.21 (0.10) $\mu\text{g}/\text{m}^3$; $p < 0.001$). For the CES Diesel Emissions indicator, homes in the >75th percentile group had higher mean concentrations (0.55 $\mu\text{g}/\text{m}^3$, SD: 0.40) than those in the ≤ 75 th percentile group (mean: 0.39 $\mu\text{g}/\text{m}^3$, SD: 0.31), although this difference was not statistically significant ($p = 0.10$).

Indoor black carbon levels were similar across CES Traffic Density scores, with a mean (SD) of 0.46 (0.45) $\mu\text{g}/\text{m}^3$ for the >75th percentile group and 0.41 (0.32) $\mu\text{g}/\text{m}^3$ for the ≤ 75 th percentile group.

Homes located within AB 617–designated communities had significantly higher indoor black carbon concentrations (mean (SD): 0.56 (0.30) $\mu\text{g}/\text{m}^3$) compared to homes outside these communities (mean (SD): 0.33 (0.32) $\mu\text{g}/\text{m}^3$; $p < 0.001$), suggesting higher environmental burden in AB 617 areas.

Table 34: Association between indoor 24-hour black carbon levels ($\mu\text{g}/\text{m}^3$) and CalEnviroScreen (CES) traffic indicators and residence in a AB 617 community.

Variable	Category	Mean	SD	Median	75 th	90 th	Min	Max	p-value*
CES Overall Score	< 75 th Percentile	0.21	0.10	0.18	0.25	0.37	0.11	0.48	< 0.001
	≥ 75 th Percentile	0.53	0.37	0.42	0.64	0.94	0.05	1.51	
CES Diesel Emission Percentile	< 75 th Percentile	0.39	0.31	0.3	0.47	0.77	0.05	1.48	0.10
	≥ 75 th Percentile	0.55	0.40	0.42	0.56	1.00	0.17	1.51	
CES Traffic Density Indicator	< 75 th Percentile	0.41	0.32	0.34	0.48	0.90	0.05	1.48	0.89
	≥ 75 th Percentile	0.46	0.45	0.32	0.47	0.85	0.16	1.51	
AB 617 ^a	No	0.33	0.32	0.22	0.37	0.51	0.05	1.51	< 0.001
	Yes	0.56	0.30	0.47	0.7	0.94	0.16	1.47	

*Wilcoxon rank sum test p-value.

^a AB 617 CARB designated community.

Figure 7 presents indoor 24-hour black carbon concentrations ($\mu\text{g}/\text{m}^3$) stratified by CalEnviroScreen (CES) overall scores and residence within an AB 617 community. Indoor levels were significantly higher in homes located in areas with CES scores > 75th percentile compared to those ≤ 75 th percentile (also see Table 26). Similarly, homes in AB 617-designated communities showed significantly higher median and overall indoor concentrations than those outside these areas (also see Table 26). These results suggest that communities with greater environmental vulnerability experienced increased indoor black carbon exposure.

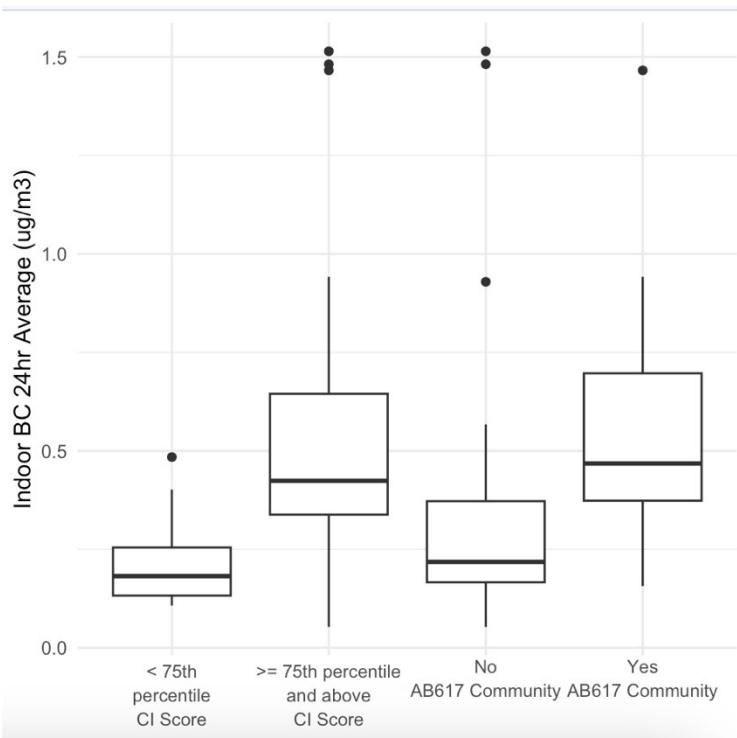


Figure 6: Indoor 24-hour black carbon levels ($\mu\text{g}/\text{m}^3$) and CalEnviroScreen (CES) overall cumulative impact scores and AB 617 location.

Table 35 summarizes outdoor 24-hour black carbon concentrations ($\mu\text{g}/\text{m}^3$) stratified by CalEnviroScreen (CES) traffic-related indicators and AB 617 community designation. Outdoor black carbon levels were significantly higher in areas with CES Overall Scores > 75th percentile (mean (SD): 0.74 (0.46) $\mu\text{g}/\text{m}^3$) than in those \leq 75th percentile (mean (SD): 0.47 (0.30) $\mu\text{g}/\text{m}^3$; $p = 0.04$). Higher concentrations also appeared in areas with CES Diesel Emission Percentiles > 75th (mean (SD): 1.01 (0.49) $\mu\text{g}/\text{m}^3$) compared to \leq 75th (mean (SD): 0.59 (0.37) $\mu\text{g}/\text{m}^3$, though not statistically significant ($p = 0.13$). For CES traffic density scores, mean concentrations were 0.65 $\mu\text{g}/\text{m}^3$ for \leq 75th and 0.60 $\mu\text{g}/\text{m}^3$ for > 75th percentile ($p = 0.30$). Homes within AB 617 communities had significantly higher outdoor black carbon (mean (SD): 0.79 (0.45) $\mu\text{g}/\text{m}^3$) than those outside these areas (mean (SD): 0.56 (0.40) $\mu\text{g}/\text{m}^3$; $p = 0.02$).

Table 35: Association between outdoor 24-hour black carbon levels ($\mu\text{g}/\text{m}^3$) and CalEnviroScreen (CES) traffic indicator and residence in AB 617 community.

Variable	Category	Mean	SD	Median	75 th	90 th	Min	Max	p-value*
CES Overall Score	< 75 th Percentile	0.47	0.30	0.39	0.58	0.82	0.10	1.24	0.04
	\geq 75 th Percentile	0.74	0.46	0.66	0.88	1.42	0.17	1.95	
CES Diesel Emission Percentile	< 75 th Percentile	0.59	0.37	0.55	0.81	1.02	0.10	1.75	0.13
	\geq 75 th Percentile	1.01	0.49	0.70	1.60	1.89	0.41	1.95	
CES Traffic Density Indicator	< 75 th Percentile	0.65	0.35	0.59	0.85	1.03	0.17	1.95	0.30
	\geq 75 th Percentile	0.60	0.21	0.37	0.65	1.27	0.10	1.84	
AB 617 ^a	No	0.56	0.40	0.45	0.73	1.01	0.10	1.84	0.02
	Yes	0.79	0.45	0.72	0.89	1.25	0.26	1.95	

*Wilcoxon rank sum test p-value.

^a AB 617 CARB designated community.

Figure 8 presents outdoor 24-hour black carbon concentrations ($\mu\text{g}/\text{m}^3$) stratified by CalEnviroScreen (CES) overall score and residence within an AB 617 community. Outdoor black carbon levels were significantly higher in areas with CES CI Scores > 75th percentile compared to those \leq 75th percentile (also see Table 27). Similarly, homes in AB 617-designated communities showed significantly higher outdoor concentrations than those outside these areas (also see Table 27). These findings indicate that communities facing greater environmental burdens also experienced elevated ambient black carbon exposure.

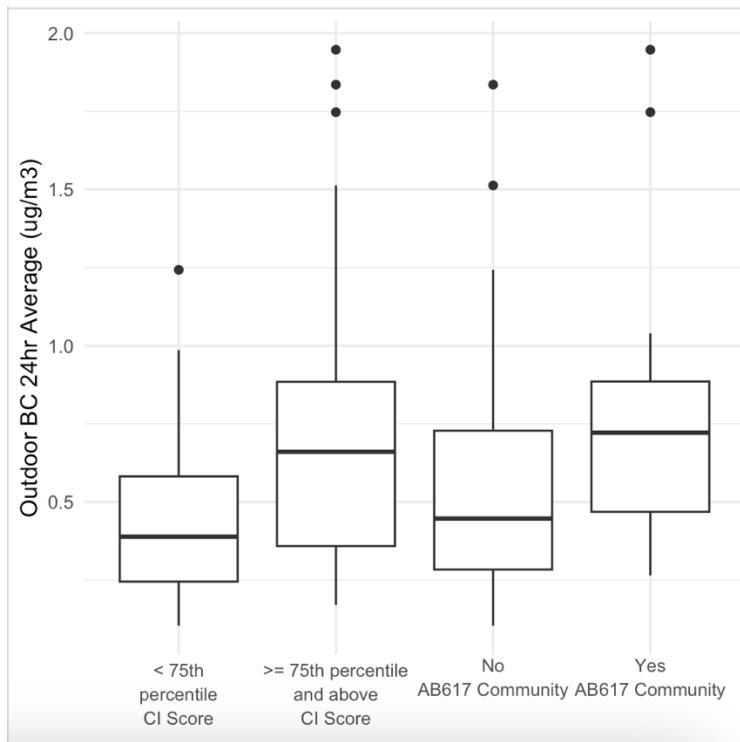


Figure 7: Outdoor 24-hour black carbon level ($\mu\text{g}/\text{m}^3$) and CalEnviroScreen (overall cumulative impact score) and AB 617 location.

Table 36 summarizes associations between indoor and outdoor 24-hour black carbon concentrations and Daily Vehicle Miles Traveled (DVMT) across buffer sizes from 100 to 1,000 meters. Spearman correlation coefficients for indoor black carbon increased with buffer size. The strongest indoor association occurred at the 1,000-meter buffer ($\rho = 0.29$, $p = 0.03$), while smaller buffers such as 100 m ($\rho = 0.05$, $p = 0.73$) and 250 m ($\rho = 0.20$, $p = 0.12$) showed weaker, non-significant results. In contrast, surprisingly, outdoor black carbon showed weak and non-significant correlations with DVMT across all buffer sizes, with coefficients ranging from $\rho = 0.02$ to 0.11 and p -values above 0.45 . We observed similar trends when we examined the correlation of indoor and outdoor black carbon concentrations with distance to SR-99 (see below).

Table 36: Associations between indoor and outdoor black carbon and traffic (Daily Vehicle Miles Traveled (DVMT)).

Time	Buffer Size (m)	Mean DVMT ^a	Indoor Correlation ^b	Indoor p-value	Outdoor Correlation ^b	Outdoor p-value
24-hour	100	417	0.05	0.73	0.11	0.45
	250	4906	0.20	0.12	0.07	0.62
	500	26315	0.18	0.18	0.03	0.84
	750	69577	0.25	0.06	0.06	0.68
	1000	126871	0.29	0.03	0.02	0.88

^a DVMT from Tracking California traffic tool.¹²⁶ ^b Spearman rank correlation between black carbon and traffic.

Table 37 presents the correlation between indoor black carbon concentrations and residential proximity to Highway SR-99. Based on data from 54 homes, the analysis showed a statistically significant negative correlation (Spearman rho = -0.42 , $p < 0.001$). This result indicates that homes located closer to the highway tended to have higher indoor black carbon levels, suggesting proximity to major roadways may contribute to indoor air pollution.

Table 37: Correlation between indoor black carbon and residential proximity (meters) to highway SR-99.

N	Spearman rho	p-value
54	-0.42	<0.001

Table 38 presents the correlation between outdoor black carbon concentrations and residential proximity to Highway SR-99. Based on 44 outdoor measurements, we observed a negative but non-significant correlation (Spearman rho = -0.24 , $p = 0.12$). This suggests a potential trend of higher outdoor black carbon levels closer to the highway, though the association was not statistically significant and it appears that indoor black carbon levels are more strongly influenced by nearby traffic. It is possible the building envelope traps outdoor air pollutants once they enter the building. In future analyses, we will determine whether concurrent meteorological conditions interact with distance to SR-99 as determinants of residential black carbon exposure.

Table 38: Correlation between outdoor black carbon and residential proximity (meters) to highway SR-99.

N	Spearman rho	p-value
44	-0.24	0.12

Figure 9 shows indoor black carbon concentrations ($\mu\text{g}/\text{m}^3$) by season across 59 total measurements collected in Fresno. Indoor levels were lowest during spring, slightly higher in summer, and highest in fall. These results suggest seasonal variation in indoor black carbon exposure, with higher levels during the fall months and lowest during spring, consistent with general patterns of $\text{PM}_{2.5}$ exposure.¹³²

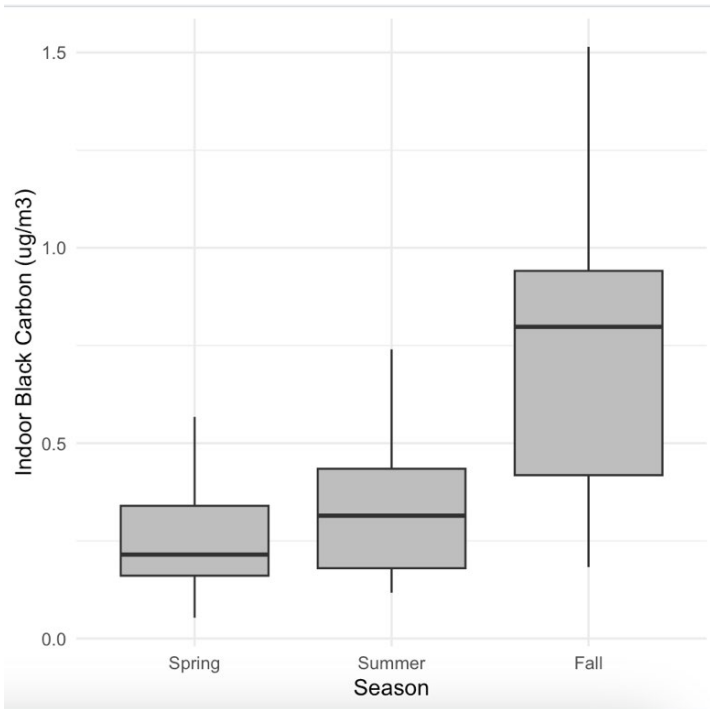


Figure 8: Indoor black carbon concentrations by season (n=59 total measurements) (Fresno (n=48) and Stockton (n=11)). (Spring n=20; Summer n=25; Fall n=14).

Figure 10 presents indoor black carbon concentrations ($\mu\text{g}/\text{m}^3$) during the warm months (May–October), stratified by the presence of central air conditioning (AC) in the home. Homes without central AC showed higher median black carbon levels compared to those with AC. These results suggest that central AC may be associated with lower indoor black carbon exposure during warmer seasons, perhaps due to filtration which is built into central AC.

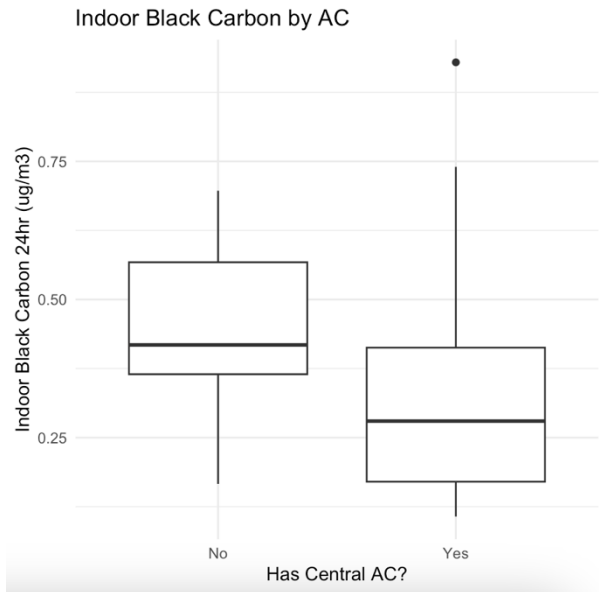


Figure 9: Indoor black carbon concentrations during the warm months (May - October) stratified by central air conditioning (AC) in the home (No (n=9); yes (n=35)).

Figure 11 presents boxplots of outdoor black carbon concentrations ($\mu\text{g}/\text{m}^3$) by season, based on samples collected in Fresno (n=42) and Stockton (n=8). Outdoor concentrations were lowest in spring (April 1 to June 21; n=12), increased in summer (June 22 to Sept 21; n=21), and peaked in fall (Sept 21-Nov 30; n= 17). These findings suggest a seasonal pattern, with outdoor black carbon concentrations higher in the fall months (Kruskal-Wallis p-values <0.01), consistent with general patterns of $\text{PM}_{2.5}$ exposure.¹³²

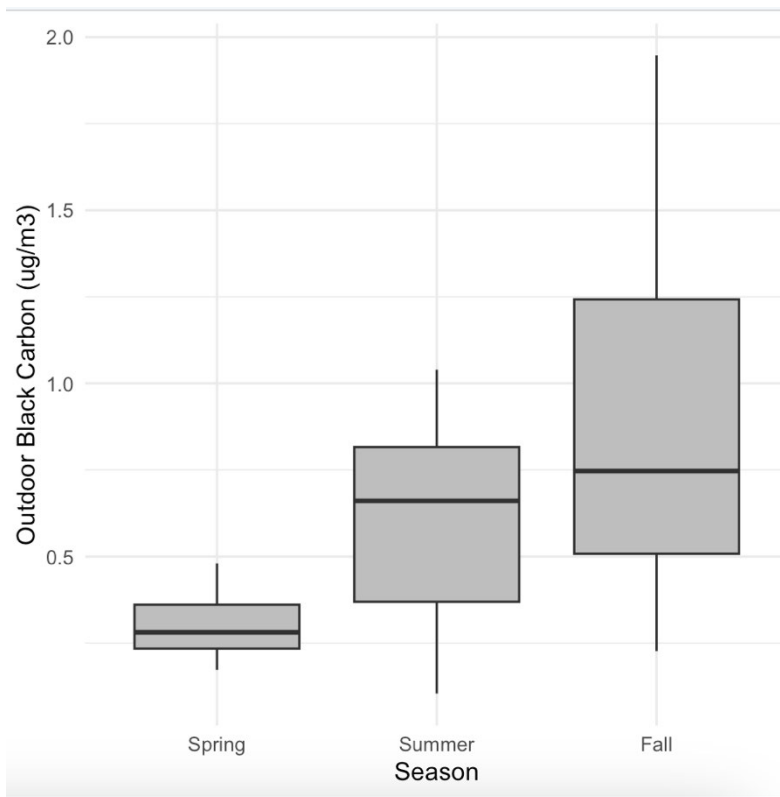


Figure 10: Box plot of outdoor black carbon concentrations by season (n=50 total measurements (Fresno (n=42) and Stockton (n=8))).

Table 39 presents Spearman correlations between 24-hour average indoor air pollutant concentrations of black carbon, PM_{2.5}, and NO₂. Black carbon showed a strong positive correlation with PM_{2.5} (rho = 0.69, p < 0.001; n = 55) and a moderate correlation with NO₂ (rho = 0.40, p < 0.01; n = 55). PM_{2.5} and NO₂ were also moderately correlated (rho = 0.37, p < 0.01; n = 65). These results indicate that indoor levels of these pollutants tend to co-vary, reflecting possible shared sources or similar infiltration pathways.

Table 39: Spearman correlations between 24-hour average indoor air pollutant concentrations.

Pollutant	Black carbon (n)	PM _{2.5} (n)	NO ₂
Black carbon	1		
PM _{2.5}	0.69* (55)	1	
NO ₂	0.40* (55)	0.37* (64)	1

*p-value < 0.01.

Table 40 presents Spearman correlations between 24-hour average outdoor air pollutant concentrations of black carbon, PM_{2.5}, and NO₂. Outdoor black carbon levels were strongly correlated with PM_{2.5} (rho= 0.76, p < 0.001; n = 50) and moderately correlated with NO₂ (rho = 0.53, p < 0.01; n = 50). PM_{2.5} and NO₂ were also moderately correlated (rho = 0.44, p < 0.001; n = 62). These results indicate consistent co-occurrence of outdoor air pollutants, likely driven by common emission sources and regional atmospheric patterns.

Table 40: Spearman correlations between 24-hour average outdoor air pollutant concentrations.

Pollutant	Black carbon (n)	PM _{2.5} (n)	NO ₂
Black carbon	1		
PM _{2.5}	0.76 (50)	1	
NO ₂	0.53 (50)	0.44 (62)	1

*p-value <0.01.

Table 41 presents Spearman correlations between estimated 24-hour average indoor air pollutant concentrations and air change rate (ACH; air changes per hour). Among the pollutants, only O₃ showed a statistically significant correlation with air change rate (rho = 0.61, p < 0.01; n = 37), suggesting increased ventilation may be associated with elevated indoor O₃ levels. Other pollutants (black carbon (rho = 0.15, p = 0.41; n = 31), PM_{2.5} (rho = -0.24, p = 0.16; n = 37), and NO₂ (rho = -0.02, p = 0.92; n = 37) did not show significant correlations with ventilation rate. These findings highlight the distinct behavior of O₃ compared to particulate and combustion-related pollutants in indoor environments.

Table 41: Spearman correlations between 24-hour average indoor air pollutant concentrations and estimated air change rate (air changes/hour).

Pollutant (n)	Correlation (p-value)
Black carbon (n=31)	0.15 (0.41)
PM _{2.5} (n=37)	-0.24 (0.16)
NO ₂ (n=37)	-0.02 (0.92)
O ₃ (n=37)	0.61 (<0.01)

3.3.3 VOCs

3.3.3.1 Integrated Individual VOCs

Indoor air VOC concentrations from the homes of 16 SPHERE study participants living in Fresno, CA (n=23 samples) are presented in **Table 42**. The nine most frequently detected compounds (DF>50%) in indoor air were toluene, para-xylene, 2-isopropyltoluene, styrene,

ethylbenzene, 1,2,4-trimethylbenzene, ortho- & meta-xylene and benzene. Toluene and para-xylene were detected in all indoor air samples (DF=100%). The median (95th percentile) indoor concentrations for the BTEX compounds benzene, toluene, ethylbenzene and xylenes were 0.26 (0.84) $\mu\text{g}/\text{m}^3$; 2.1 (5.1) $\mu\text{g}/\text{m}^3$; 0.56 (1.30) $\mu\text{g}/\text{m}^3$; and 2.1 (4.9) $\mu\text{g}/\text{m}^3$, respectively. "Xylenes" refers to the sum of all three xylene isomers (ortho-, meta-, and para-).

Concentrations of VOCs are typically higher indoors compared with outdoor air due to the predominance of indoor sources such as building materials, furniture, cleaning products, and personal care items that release VOCs into the air. The average and range of indoor-to-outdoor concentration (I/O) ratios for the eight SPHERE homes with both indoor and outdoor VOC measurements are presented in **Table 42**. The average I/O ratios for the nine most frequently detected compounds (DF>50%) ranged from 0.7 to 20.4. Benzene was the only compound with an average I/O ratio <1, suggesting indoor sources predominated for the other VOCs. The highest I/O ratio (38.5) was found for 4-isopropyltoluene, also known as p-cymene. 4-isopropyltoluene can be found in many household products, including air fresheners, cleaning products, personal care items (soaps, lotions), and laundry products.

Table 42: Summary of indoor air VOC concentration measurements ($\mu\text{g}/\text{m}^3$) (n= 23 samples collected from 16 homes in Fresno, CA).

VOC	DF (%)	Mean	SD	25th	50th	75th	95th	Max	I/O mean	I/O range
Toluene	100	2.7	2.6	1.1	2.1	3.3	5.1	13.2	2.4	0.7, 7.1
p-Xylene	100	1.4	1.4	0.4	1.2	1.8	2.7	6.6	3.8	0.5, 9.5
4-Isopropyltoluene	95.7	1.9	2.1	0.7	1.0	1.5	5.7	7.9	20.4	2.5, 38.5
Styrene	82.6	0.7	0.4	0.32	0.5	0.8	1.6	1.8	4.7	1.0, 10.9
Ethylbenzene	73.9	0.8	0.5	--	0.6	0.8	1.3	2.4	3.1	0.6, 5.2
1,2,4-Trimethylbenzene	73.9	1.0	0.9	--	0.4	0.9	2.7	3.4	4.8	0.6, 23.1
o-Xylene	73.9	0.9	0.6	--	0.6	0.9	1.5	2.7	3.1	0.6, 6.3
m-Xylene	65.2	0.7	0.5	--	0.4	0.6	0.9	2.5	2.6	0.7, 5.8
Benzene	56.5	0.5	0.3	--	0.3	0.6	0.8	1.0	0.7	0.4, 1.2
Tetrahydrofuran	39.1	0.3	0.1	--	--	0.2	0.4	0.5	--	--
1,3,5-Trimethylbenzene	34.8	0.4	0.2	--	--	0.3	0.4	0.8	--	--
Chloroform	21.7	0.7	0.5	--	--	--	0.5	2.4	--	--
Isobutyl alcohol	21.7	0.6	0.2	--	--	--	0.7	0.8	--	--
n-Propylbenzene	17.4	0.4	0.1	--	--	--	0.5	0.5	--	--
Tetrachloroethene	17.4	0.4	0.1	--	--	--	0.4	0.6	--	--
Methyl methacrylate	13.0	8.7	4.0	--	--	--	3.4	19.3	--	--
cis-1,3-Dichloropropene	8.7	0.3	<0.1	--	--	--	--	0.3	--	--
1,4-Dichlorobenzene	8.7	0.4	<0.1	--	--	--	--	0.5	--	--
trans-1,3-Dichloropropene	8.7	0.4	<0.1	--	--	--	--	0.5	--	--

Table 42 (cont). Summary of indoor air VOC concentration measurements ($\mu\text{g}/\text{m}^3$) (n= 23 samples collected from 16 homes in Fresno, CA).

VOC	DF (%)	Mean	SD	25th	50th	75th	95th	Max	I/O mean	I/O range
Acetonitrile	4.4	--	--	--	--	--	--	4.6	--	--
1,2-Dichloroethane	4.4	--	--	--	--	--	--	0.2	--	--
Methylene chloride	4.4	--	--	--	--	--	--	0.2	--	--
Xylenes ^b	100	2.6	2.5	0.8	2.1	3.3	4.9	11.9	3.2	0.5, 5.9
Sum of VOCs (ppm)	100	0.004	0.003	0.002	0.003	0.004	0.009	0.013	3.1	0.7, 11.4

Abbreviations: DF=detection frequency; I/O=indoor-to-outdoor concentration ratio.

LOD= Limit of detection = 5.0 ng; VOC concentrations <LOD were imputed as LOD/square root of 2.

^aMean VOC concentrations were calculated using values >LOD.

^bXylenes are the sum of p-, m- and o-xylene concentrations.

Note: Five sets of collocated indoor replicates were averaged for this summary table; I/O ratios were calculated when both indoor and outdoor concentration data were available (n=8 homes) and when indoor VOC detection frequencies were >50%.

Outdoor VOC concentrations from the homes of eight SPHERE study participants living in Fresno, CA are presented in **Table 43**. The five most frequently detected compounds (DF \geq 50%) in outdoor air were toluene, benzene, para-xylene, 1,2,4-trimethylbenzene and ortho-xylene. Toluene was detected in all outdoor air samples (DF=100%) and benzene was detected in 87.5% of outdoor air samples. The median (95th percentile) outdoor concentrations for the BTEX compounds (benzene, toluene, ethylbenzene and xylenes) were 0.39 (1.22) $\mu\text{g}/\text{m}^3$, 0.89 (3.47) $\mu\text{g}/\text{m}^3$, <LOD (0.73) $\mu\text{g}/\text{m}^3$, and 0.83 (3.60) $\mu\text{g}/\text{m}^3$, respectively.

Table 43: Summary of outdoor air VOC concentration measurements ($\mu\text{g}/\text{m}^3$) (n= 8 samples collected from 8 homes Fresno, CA).

Target VOC	DF (%)	Mean ^a	SD	Median	75th	95th	Max
Toluene	100	1.4	1.3	0.9	1.4	3.5	4.5
Benzene	87.5	0.6	0.5	0.4	0.5	1.2	1.6
p-Xylene	75.0	0.9	0.8	0.5	0.6	2.0	2.6
1,2,4-Trimethylbenzene	62.5	0.5	0.4	0.2	0.3	0.9	1.2
o-Xylene	50.0	0.5	0.4	0.2	0.3	0.9	1.3
Ethylbenzene	37.5	0.5	0.3	--	0.3	0.7	1.0
m-Xylene	37.5	0.5	0.3	--	0.2	0.7	0.9
Acetonitrile	25.0	4.7	3.2	--	0.2	6.0	9.1
4-Isopropyltoluene	12.5	--	--	--	--	--	0.2
Tetrachloroethene	12.5			--	--	--	0.3
Tetrahydrofuran	12.5	--	--	--	--	--	0.3
trans-1,3-Dichloropropene	12.5	--	--	--	--	--	0.2
1,3,5-Trimethylbenzene	12.5	--	--	--	--	--	0.4
Sum of Xylene isomers ^b	75.0	1.3	1.5	0.8	1.1	3.6	4.8
Sum of VOCs (ppm)	100	0.002	0.002	0.001	0.002	0.006	0.007

Abbreviations: DF=detection frequency; I/O=indoor-to-outdoor concentration ratio; LOD=limit of detection. LOD= 5.0 ng; VOC concentrations <LOD were imputed as LOD/sq rt 2.

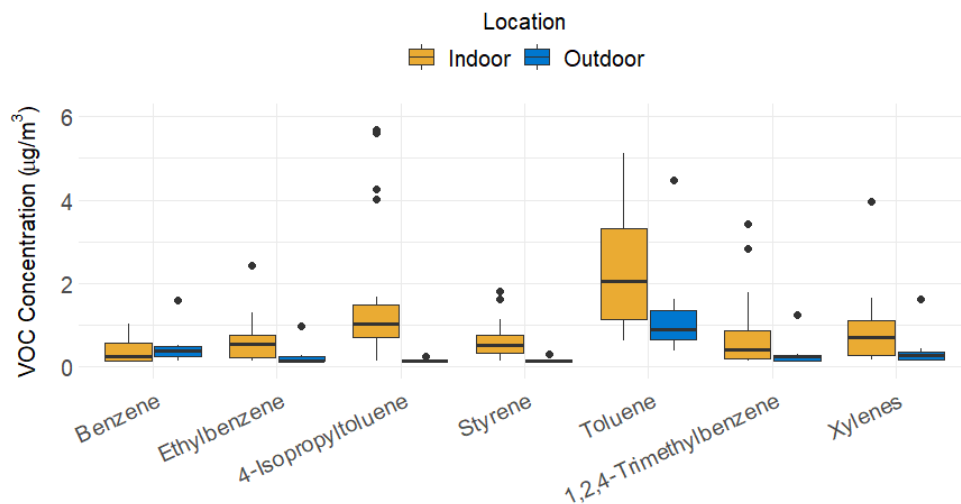
^aMean VOC concentrations were calculated using values >LOD only.

^bXylenes: sum of p-, m- and o-xylene concentrations.

Notes, n=8 VOC samples.

Figure 12 compares indoor and outdoor air concentrations ($\mu\text{g}/\text{m}^3$) of the most frequently detected volatile organic compounds (VOCs). Indoor air concentrations were consistently higher than outdoor levels for nearly all VOCs, with the exception of benzene, which showed similar indoor and outdoor values. Toluene had the highest indoor concentration (mean: $2.7 \mu\text{g}/\text{m}^3$), followed by isopropyl toluene (mean: $2.1 \mu\text{g}/\text{m}^3$), and p-xylene (mean: $1.5 \mu\text{g}/\text{m}^3$). For these compounds, indoor concentrations were approximately 2 to 3 times greater than outdoor levels. Other VOCs, including ethylbenzene, m-xylene, o-xylene, and styrene, also showed elevated indoor levels compared to outdoors, though the absolute concentrations were lower. 1, 2, 4-Trimethylbenzene showed relatively modest concentrations in both environments, with indoor levels still slightly higher.

Overall, these findings indicate elevated indoor VOC concentrations for most compounds, suggesting that indoor sources contribute substantially to overall exposure.



A toluene concentration outlier (13.19 µg/m³) was excluded

Figure 11: Indoor and outdoor air concentrations of most frequently detected VOCs.

Available health-based noncancer reference values for BTEX compounds established by the U.S. EPA (RfC) and OEHHA (Relative Exposure Levels (RELs)) are presented in **Table 44**. Indoor and outdoor BTEX concentrations measured for this study were relatively low (**Tables 42 and 43**), and none exceeded U.S. EPA RfCs or OEHHA RELs.

Table 44: OEHHA acute, 8-hour and chronic reference exposure levels (RELs) and U.S. EPA reference concentrations (RfCs).

BTEX	Acute REL (µg/m ³)	8-hour REL (µg/m ³)	Chronic REL (µg/m ³)	Inhalation RfC (µg/m ³)
Benzene	27	3	3	30
Ethylbenzene	--	--	2,000	1,000
Toluene	5,000	830	420	5,000
Xylenes (o-, m- and p-)	22,000	--	700	100

OEHHA RELs¹³³; Xylenes RfC¹³⁴; Benzene RfC¹³⁵; Ethylbenzene RfC¹³⁶ Toluene RfC¹³⁷.

Indoor and outdoor air concentrations for the BTEX compounds (benzene, toluene, ethylbenzene, and xylenes) are presented in **Tables 42 and 43** and **Figure 12** above. Indoor concentrations of toluene and xylenes were substantially higher than outdoor concentrations, with mean indoor-to-outdoor (I/O) ratios of 2.4 and 6.3, respectively. Toluene exhibited the widest interquartile range indoors, with several high-end outliers exceeding 10 µg/m³. Xylenes followed a similar pattern, with consistently elevated indoor levels and greater variability relative to outdoors.

Ethylbenzene concentrations were also higher indoors, though the difference was less pronounced. In contrast, benzene showed minimal differences between indoor and outdoor environments, consistent with previous findings. These results highlight the contribution of indoor sources, particularly household products and indoor activities, to elevated BTEX concentrations, most notably for toluene and xylenes.

These findings should be interpreted cautiously given the relatively small sample size, which included 23 indoor and 8 outdoor samples collected from 16 homes. The limited number of measurements may constrain the generalizability of the observed patterns.

Figure 13 presents box plots of indoor-to-outdoor (I/O) ratios for BTEX compounds based on eight matched indoor and outdoor air sample pairs. The I/O ratio for benzene was consistently below 1, indicating that outdoor sources likely contributed more to indoor benzene concentrations. In contrast, mean I/O ratios for toluene, ethylbenzene, and xylenes ranged from 2.4 to 3.2, indicating the presence of indoor sources for these compounds.

Xylenes exhibited the highest I/O ratio, with a median around 3 and upper values exceeding 5 in some homes. Ethylbenzene and toluene had more moderate ratios but still showed consistent indoor elevation across samples.

These results support the conclusion that toluene, ethylbenzene, and xylenes are predominantly influenced by indoor emission sources, while benzene appears more strongly influenced by outdoor air.

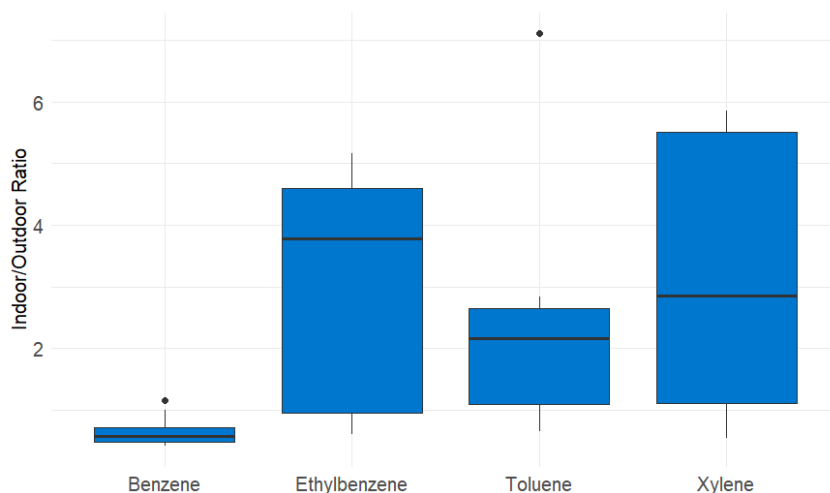


Figure 12: Ratios of BTEX Indoor-to-Outdoor concentrations (n=8 matched pairs)

Vehicular traffic is a potentially significant contributor to VOC air pollution. State Route 99 (SR-99) is a major north–south state highway with heavy truck traffic stretching almost the entire length of the SJV. **Table 45** summarizes Spearman correlations between outdoor BTEX levels and residential proximity (in kilometers) to Highway SR-99. Despite the relatively small sample size, results showed that concentrations of several volatile organic compounds (VOCs)

were significantly inversely correlated with proximity to the highway, indicating higher pollutant levels closer to SR-99.

Ethylbenzene showed the strongest inverse correlation ($\rho = -0.83$, $p = 0.01$), followed by xylenes ($\rho = -0.76$, $p = 0.03$), and the combined BTEX measure ($\rho = -0.75$, $p = 0.03$). Toluene also showed a moderately strong negative correlation ($\rho = -0.70$) that was marginally significant ($p = 0.05$). Although benzene levels were negatively correlated with proximity ($\rho = -0.47$), this association was not statistically significant ($p = 0.24$). Overall, these findings suggest that residents living closer to SR-99 may be exposed to higher levels of BTEX pollutants.

Table 45: Correlations between outdoor BTEX levels and Residential Proximity (km) to Highway SR-99.

VOC	N	Spearman rho	p-value
Benzene	8	-0.47	0.24
Toluene	8	-0.70	0.05
Ethylbenzene	8	-0.83	0.01
Xylenes	8	-0.76	0.03
BTEX	8	-0.75	0.03

To calculate summed VOC concentrations, we converted air concentrations for each VOC from $\mu\text{g}/\text{m}^3$ to ppm using compound-specific molecular weights. The VOC concentrations (ppm) were then summed across all measured compounds. **Figure 14** presents the comparison of the summed indoor ($n=23$) and outdoor ($n=8$) air VOC concentrations. Summed VOC concentrations were higher indoors compared to outdoors (mean (sd) were 0.004 (0.003) ppm and 0.002 (0.002) ppm, respectively). The average I/O ratio for summed VOC concentrations was 3.1, with a range of 0.65 to 11.4.

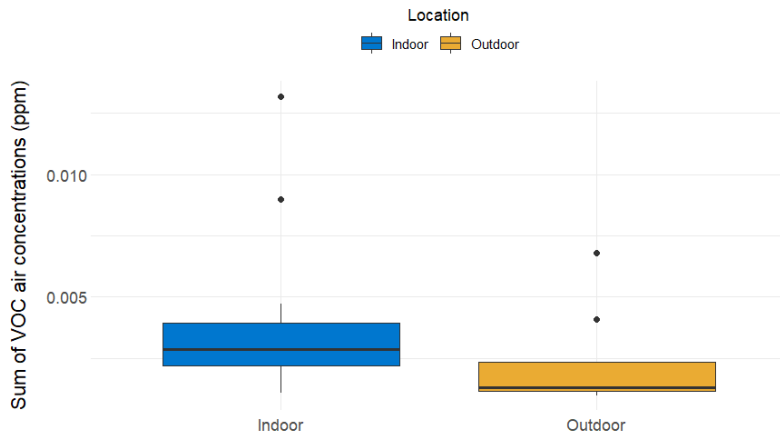


Figure 13: Comparison of summed indoor (n=23) and outdoor (n=8) air VOC concentrations (ppm)

3.3.3.2 Real-Time Total Volatile Organic Compounds (TVOCs)

We monitored indoor and outdoor TVOC concentrations over 24-hour periods in 46 participant homes in Fresno, CA using the Atmotube Pro (42 indoor and 12 outdoor measurements). Personal monitoring for TVOCs was also performed. **Table 46** presents summary statistics for total volatile organic compound (TVOC) concentrations measured from indoor, outdoor, and personal air monitoring in 46 SPHERE participant homes in Fresno, CA. Indoor TVOC concentrations (n = 42) had a mean of 0.51 ppm, with values ranging up to a maximum of 4.52 ppm. The median indoor concentration was 0.33 ppm, and the 95th percentile was 0.95 ppm, indicating a skewed distribution driven by high exposures in a few homes. Outdoor samples (n = 12) had lower concentrations, with a mean of 0.23 ppm and a maximum of 0.46 ppm. Personal samples (n = 57) showed a mean of 0.48 ppm and a maximum of 3.87 ppm. Indoor-to-outdoor (I/O) ratios, calculated from eight homes, had a mean of 3.62, with a wide range from 0.32 to 14.7, suggesting that in many cases, indoor sources were the dominant contributors to personal TVOC exposure. All measurements reflect 24-hour average concentrations.

Table 46: Summary of indoor and outdoor TVOC concentrations (ppm) (n=46 SPHERE participant homes in Fresno, CA)^a

TVOC	n	Mean	SD	Median	75th	95th	Max	I/O mean ^b	I/O range
Indoor	42	0.5	0.7	0.3	0.5	1.0	4.5	3.6	0.3, 14.7
Outdoor	12	0.2	0.1	0.2	0.3	0.4	0.5	--	--
Personal ²	57	0.5	0.6	0.3	0.5	1.7	3.9	--	--

^aSummary statistics are based on 24-hour average TVOC concentrations.

^bIndoor-to-outdoor (I/O) ratios are based on measurements from eight homes.

3.3.4 Formaldehyde

Indoor air formaldehyde concentrations were collected from 24 SPHERE homes in Stockton and Fresno, CA using 24-hour passive sampling methods. **Table 47** presents a summary of the indoor formaldehyde concentrations measured. Formaldehyde was frequently detected (DF=81%) and the median (range) concentration was 18.1 (3.3, 69.5) $\mu\text{g}/\text{m}^3$. The median indoor formaldehyde concentrations exceeded the U.S. EPA RfC for formaldehyde (0.007 mg/m^3 (7 $\mu\text{g}/\text{m}^3$)) and the OEHHA 8-hour REL (0.009 mg/m^3 (9 $\mu\text{g}/\text{m}^3$)).^{133,138}

Table 47: Summary of indoor formaldehyde concentrations ($\mu\text{g}/\text{m}^3$) from 24 homes

N	DF	Mean	SD	Median	90 th	Min	Max
27	81%	22.5	15.8	18.1	46	3.3	69.5

Field Blank Corrected LOD = 10.7 $\mu\text{g}/\text{m}^3$.

Field blank corrected concentrations are presented in the table; Average field blank concentration (n=3) = 5.3 $\mu\text{g}/\text{m}^3$.

3.3.5 PAHs

Indoor and outdoor samples were collected from the homes of 61 study participants living in Stockton and Fresno. The four PAHs we focused on were naphthalene, fluorene, phenanthrene and pyrene.

Tables 48 and **49** present PAH air concentrations from 59 indoor and 64 outdoor air samples collected from 61 participant homes. Thirteen pairs of co-located outdoor duplicates were averaged for the data summary. Among the four PAHs examined for indoor concentrations, naphthalene (NAP) had the highest mean concentration at 45 ng/m^3 (standard deviation (SD) = 57), with levels ranging up to 261 ng/m^3 across 58 samples (**Table 48**). Naphthalene was detected in 47% of indoor samples. Other measured PAHs, including fluorene (FLU), phenanthrene (PHE), and pyrene (PYR), were detected less frequently, with detection frequencies ranging from 5% to 18%. Their mean concentrations were relatively low (2.2–4.6 ng/m^3) in the quantifiable samples, and a number of values were below the limits of detection (LOD). The indoor-to-outdoor (I/O) ratios for these compounds ranged from 0.9 to 1.95, with naphthalene showing the highest mean I/O ratio (1.95) and a wide range up to 12.3, suggesting potential indoor sources or accumulation in some homes.

Table 48: Indoor air PAH concentration (ng/m³) and indoor-to-outdoor (I/O) ratios.

PAH	# Samples ^a	DF (%)	Mean (SD) ^b	50 th	75 th	95 th	Max	Average I/O ratio ^b	Range I/O ratio ^b
NAP	58	47%	45 (57)	16	58	164	261	2.0	0.1 – 12.3
FLU	57	5%	2.2 (0.4)	<LOD	<LOD	2.6	4.0	1.0	0.4 – 1.8
PHE	57	18%	4.6 (2.0)	<LOD	<LOD	8.9	10.6	1.0	0.4 – 2.3
PYR ^c	32	16%	3.6 (1.4)	<LOD	<LOD	6.7	8.6	0.9	0.3 – 2.6

Abbreviations: detection frequency (DF); naphthalene (NAP); fluorene (FLU); phenanthrene (PHE); and pyrene (PYR).

Limit of detection (LOD): NAP = 5.79 ng; FLU = 44.29 ng; PHE = 77.33 ng; PYR = 55.14 ng.

^a Concentrations include averaging of 13 co-located duplicate sample measurements.

^b Values below LOD have been imputed as LOD/square root 2.

^c Instrument error resulted in fewer quantifiable PYR samples than other PAHs.

Outdoor air concentrations of PAHs followed a similar pattern but with generally lower levels and detection frequencies (**Table 49**). Naphthalene was again the most frequently detected compound, quantifiable in 33% of outdoor samples, with a mean concentration of 26 ng/m³ (SD = 31) and a maximum of 200 ng/m³. Other PAHs (FLU, PHE, PYR) were detected in 8% to 20% of samples, with mean concentrations comparable to those observed indoors (2.4–4.6 ng/m³). Several outdoor measurements fell below detection thresholds, particularly for FLU and PYR.

Table 49: Outdoor Air PAH concentration (ng/m³).

PAH	# Samples ^a	DF (%)	Mean (SD) ^b	Median	75 th	95 th	Max
NAP	51	33%	26 (31)	15	26	71	200
FLU	51	8%	2.4 (1.0)	<LOD	<LOD	4.2	7.6
PHE	51	20%	4.6 (1.9)	<LOD	<LOD	8.6	9.2
PYR ^c	30	18%	3.3 (1.1)	<LOD	<LOD	4.5	9.1

Abbreviations: detection frequency (DF); naphthalene (NAP); fluorene (FLU); phenanthrene (PHE); and pyrene (PYR)

Limit of detection (LOD): NAP = 5.79 ng; FLU = 44.29 ng; PHE = 77.33 ng; PYR = 55.14 ng

^a Concentrations include average of duplicate sample measurements. ^b Values below LOD imputed as LOD/square root 2. ^c Several runs of PYR analyses were not quantifiable due to GC/MS error.

Figure 15 presents naphthalene air concentration results from 59 indoor and 64 outdoor air samples collected from 61 participant homes. Thirteen pairs of co-located outdoor duplicates were averaged for the data summary. The naphthalene I/O ratio was 2.3. **Figure 15** visually compares indoor and outdoor naphthalene concentrations, reinforcing the observation that indoor concentrations are often higher than outdoor levels. This is further supported by the average I/O ratio of 1.9 for naphthalene, suggesting indoor accumulation or sources such as building materials, consumer products, or tobacco smoke. The distribution of concentrations across homes highlights substantial variability, with some homes exhibiting markedly elevated indoor naphthalene levels. These findings point to the importance of investigating and mitigating indoor sources of PAHs, particularly in environments with vulnerable populations.

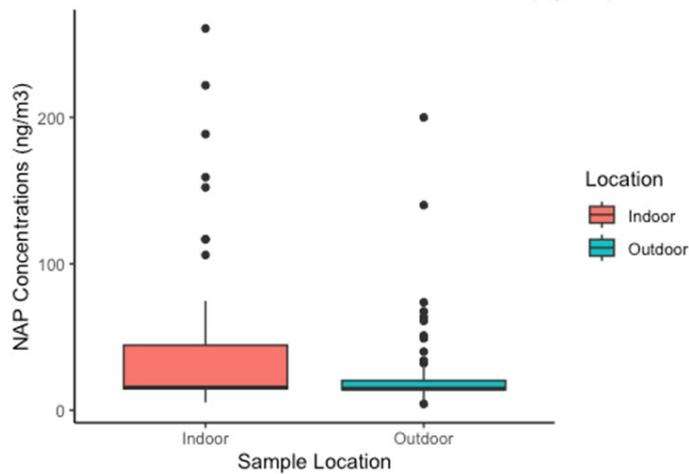


Figure 14: Comparison of indoor and outdoor naphthalene concentrations (ng/m³).

3.3.6 Noise

Table 50 presents summary statistics for personal, indoor, and outdoor noise measurements (dBA) collected over 24-hour, daytime (7 a.m.–10 p.m.), and nighttime (10 p.m.–7 a.m.) periods. Across all time periods and locations, average noise levels ranged from 61 to 68 dBA. Personal noise measurements showed a mean of 66 dBA over 24 hours, with 68 dBA during the day and 60 dBA at night, with a range of 37-72 dBA. Indoor noise levels were relatively stable, with a 24-hour mean of 64 dBA and slightly higher levels during the day (65 dBA) compared to night (63 dBA), with a range of 43-77 dBA. Outdoor noise had a 24-hour mean of 63 dBA, with 63 dBA during the day while nighttime levels were 62 dBA, with a range of 49 to 73 dBA.

Indoor/outdoor noise ratios were consistently near 1.0, with mean values of 1.0 for all three time periods, suggesting comparable noise exposure indoors and outdoors. The range of these ratios varied from 0.8 to 1.79, indicating individual variability in some cases. However, no significant correlations were observed between indoor and outdoor noise measurements based on Spearman correlation tests (p -values > 0.10).

Personal noise ratios to indoor and outdoor noise levels were centered around 1.0, with a range from 0.9-1.1. During the night, personal noise measurements were significantly correlated with indoor noise measurements ($\rho=0.36$). Other correlations of personal noise measurements and indoor noise were weak and non-significant. Correlations between personal and outdoor noise levels were weak and insignificant during all three time periods.

Table 50: Personal, indoor and outdoor noise measurements (dBA) averaged for day, night and over 24 hours.

Time	Location	Number of samples	Mean (dBA)	Median	90th	Min	Max
24-hour	Personal	37	66	69	66	52	73
	Indoor	65	64	61	67	54	76
	Outdoor	46	63	66	61	52	71
Day (7am-10pm)	Personal	39	68	67	70	54	75
	Indoor	65	65	62	68	55	75
	Outdoor	47	63	62	66	52	73
Night (10pm-7am)	Personal	37	60	53	64	37	72
	Indoor	65	63	59	63	43	77
	Outdoor	47	62	60	66	49	67

Note: No significant correlations were found between indoor and outdoor noise measurement (Spearman correlation P-values>0.10).

Figure 16 presents histograms of noise measurements collected during the day, night, and 24-hour periods across three different settings: personal, indoor, and outdoor. Each row of histograms represents a specific environment (personal, indoor, or outdoor), while columns correspond to the time period (day, night, and 24-hour). The x-axis of each histogram denotes noise levels in decibels (dB), and the y-axis indicates the count of minutes during which each noise level was observed.

Personal noise levels during the day are broadly distributed, with a peak between 55–60 dB and a long tail extending beyond 80 dB. At night, the distribution is centered around 50 dB with lower counts of higher decibel events. The 24-hour distribution integrates both periods, with a noticeable central peak around 55 dB and reduced extreme values compared to the daytime distribution. Indoor environments show tightly clustered noise levels across all time periods. During the day, indoor noise peaks sharply between 55–60 dB. At night, the pattern remains consistent but slightly narrower and shifts to the left, suggesting quieter conditions. The 24-hour indoor histogram remains highly concentrated, reinforcing the stability and lower variability of indoor noise. Outdoor noise during the day exhibits a broader range, peaking around 55 dB but with substantial counts above and below this level. At night, outdoor noise levels decline, with a central peak near 50 dB and less spread. The 24-hour outdoor histogram shows a bimodal tendency with a dominant peak between 50–55 dB, reflecting differences between day and night conditions.

Overall, noise levels are highest and most variable in personal and outdoor environments during the day, while indoor environments display more consistent and moderate noise levels across all time frames. Nighttime measurements consistently show reduced noise levels across all settings.

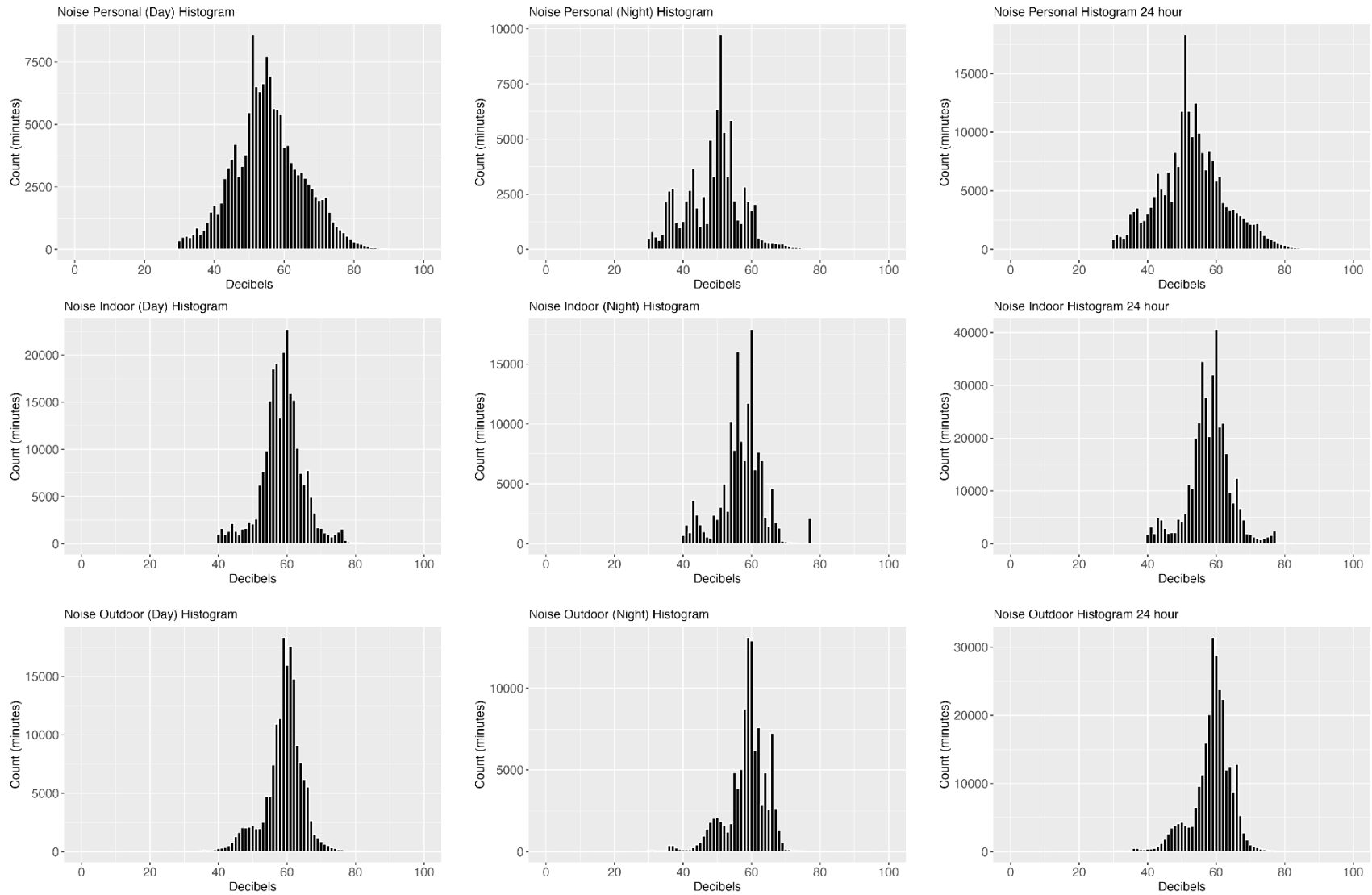


Figure 15: Histograms of noise measurements collected during the day, night and 24-hours.

Table 51 presents the associations between indoor and outdoor noise levels and nearby traffic, measured by daily vehicle miles traveled (DVMT), across varying buffer sizes (100–1000 meters). Spearman rank correlation coefficients (rho) were calculated to examine the relationship between traffic volume and noise levels both indoors and outdoors. Correlations were computed for multiple spatial buffer sizes, ranging from 100 m to 1000 m, during daytime, nighttime, and over 24-hour periods. Correlation coefficients between indoor noise and DVMT were consistently low across all buffer distances (rho ranging from 0.10 to 0.22), and none of the associations were statistically significant (p-values > 0.10). This suggests a weak and non-significant relationship between indoor noise levels and nearby traffic, regardless of buffer size or time of day. In contrast, outdoor noise levels showed stronger and more statistically significant correlations with DVMT, particularly at smaller buffer distances. The strongest correlations were observed at the 100 m buffer during all time periods (rho = 0.36 for daytime, 0.42 for nighttime, and 0.37 for 24-hour), with p-values ≤ 0.02, indicating a statistically significant relationship between outdoor noise and nearby traffic. Correlations were lower and became non-significant as buffer size increased. These results indicate that outdoor noise is more sensitive to nearby traffic activity, especially at smaller spatial scales, while indoor noise does not show a consistent relationship with surrounding traffic volume.

Table 51: Associations between indoor and outdoor noise and nearby traffic (daily vehicle miles traveled (DVMT))

Time	Buffer Size (m)	Mean DVMT	Indoor Noise Correlation ^a	p-value (indoor)	Outdoor Noise Correlation ^a	p-value (outdoor)
Daytime	100	447	0.2	0.2	0.4	0.02
	250	5093	0.1	0.5	0.3	0.06
	500	26,932	0.2	0.1	0.3	0.08
	750	71,335	0.1	0.4	0.3	0.06
	1000	131,018	0.1	0.5	0.2	0.16
Nighttime	100	447	0.2	0.2	0.4	0.006
	250	5093	0.1	0.5	0.3	0.05
	500	26,932	0.2	0.1	0.2	0.31
	750	71,335	0.1	0.5	0.3	0.08
	1000	131,018	0.1	0.3	0.3	0.07
24-hour	100	447	0.2	0.2	0.4	0.02
	250	5093	0.1	0.5	0.3	0.07
	500	26,932	0.2	0.1	0.2	0.21
	750	71,335	0.1	0.5	0.3	0.10
	1000	131,018	0.1	0.3	0.2	0.20

^aSpearman rank correlation coefficient (rho).

3.3.6.1 Correlations between air pollutant concentrations and noise

Table 52 presents Spearman correlations between indoor air pollutants and noise. PM_{2.5} showed strong positive correlations with both black carbon ($\rho = 0.70$, $p < 0.01$) and NO₂ ($\rho = 0.41$, $p < 0.01$), suggesting common indoor sources. PM_{2.5} was also moderately correlated with CO ($\rho = 0.37$, $p < 0.01$), and black carbon was moderately correlated with NO₂ ($\rho = 0.41$, $p < 0.01$). O₃ showed weak or non-significant relationships with other pollutants. BTEX and formaldehyde (CH₂O) generally had weak correlations with other pollutants, though CH₂O showed a moderate positive correlation with NO₂ ($\rho = 0.33$). Naphthalene was negatively correlated with PM_{2.5} and CO, indicating potentially different sources or removal mechanisms. Noise showed weak non-significant correlations to all other indoor pollutants.

Table 52: Correlation matrix of indoor air pollutants and noise.

Indoor	PM _{2.5}	NO ₂	O ₃	CO	Noise	Black Carbon	BTEX Total	CH ₂ O
NO ₂	0.41** (68)							
O ₃	0.06 (68)	-0.01 (68)						
CO	0.37** (68)	0.28* (68)	0.43** (70)					
Noise	-0.03 (63)	0.08 (63)	0.17 (65)	-0.1 (65)				
Black Carbon	0.70** (58)	0.41** (58)	-0.03 (60)	0.19 (60)	0.01 (56)			
BTEX total	-0.14 (21)	0.07 (21)	-0.21 (21)	-0.04 (21)	0.06 (22)	0.34 (15)		
CH ₂ O	-0.09 (26)	0.33 (26)	-0.32 (27)	0.09 (27)	-0.15 (26)	-0.39 (21)	0.19 (8)	
Naphthalene	-0.43* (35)	-0.3 (35)	-0.1 (36)	-0.45** (36)	0.25 (35)	-0.34 (30)	0.26 (8)	0.23 (27)

Abbreviations: CH₂O = formaldehyde.

* $p < 0.05$; ** $p < 0.01$; (Sample size in parenthesis).

Table 53 presents the Spearman correlations between outdoor air pollutants and noise. Outdoor pollutant correlations mirrored some indoor patterns but also revealed unique relationships. PM_{2.5} and black carbon were strongly correlated ($\rho = 0.77$, $p < 0.01$), indicating shared outdoor sources such as vehicle emissions. NO₂ was positively correlated with PM_{2.5} ($\rho = 0.41$, $p < 0.01$), black carbon ($\rho = 0.52$, $p < 0.01$), and CO ($\rho = 0.40$, $p < 0.01$). CO also correlated strongly with PM_{2.5} ($\rho = 0.47$, $p < 0.01$) but negatively with O₃ ($\rho = -0.35$, $p < 0.05$), reflecting the differing formation processes of these pollutants. Noise showed a modest negative correlation with NO₂ outdoors ($\rho = -0.33$, $p < 0.05$). While BTEX and naphthalene

showed strong associations with certain pollutants, these findings were based on small sample sizes and should be interpreted with caution.

Across both indoor and outdoor environments, consistent positive correlations among PM_{2.5}, black carbon, NO₂, and CO suggest combustion-related sources, particularly from vehicles and possibly indoor activities such as cooking. In contrast, O₃ tended to be weakly or negatively correlated with other pollutants, consistent with its distinct photochemical formation and sensitivity to environmental conditions. Naphthalene’s negative correlations with several pollutants suggest different emission or removal pathways. BTEX and formaldehyde correlations were generally weak, and their limited sample sizes limit firm conclusions. Noise was largely uncorrelated with air pollutants, though modest links were found outdoors. Overall, the data emphasize the value of multipollutant monitoring to understand source profiles and potential health risks in low-income residential settings.

Table 53: Correlation matrix of outdoor air pollutants and noise.

Outdoor	PM _{2.5}	NO ₂	O ₃	CO	Noise	Black Carbon
NO ₂	0.41** (76)					
O ₃	0.06 (76)	-0.19 (76)				
CO	0.47** (76)	0.40** (76)	-0.35* (76)			
Noise	-0.10 (54)	-0.33* (54)	0.33 (54)	-0.1 (54)		
Black Carbon	0.77** (62)	0.52** (62)	0.24 (62)	0.36 (62)	-0.19 (42)	
BTEX total	-0.05 (8)	0.88* (8)	-0.86* (8)	0.17 (8)	-0.48 (8)	0.49 (6)
Naphthalene	-0.37* (22)	-0.4 (22)	-0.24 (22)	-0.49* (22)	-0.18 (17)	-0.53* (18)

*p<0.05 ; **p<0.01; (sample size in parenthesis).

3.3.6.2 Reported Sensitivity and Perception of Environmental Noise

Average noise sensitivity scores were nearly identical between children and adults, indicating similar levels of perceived sensitivity across age groups (**Table 54**). Mean scores were 29.9 (± 9.3) for children and 29.8 (± 8.9) for adults, based on a 10–50 scale where higher scores reflect greater sensitivity to noise. Scores ranged from 10 to 45 for children and 10 to 48 for adults, with interquartile ranges spanning roughly 22–37 for both groups. These findings

suggest that, within participating households, both children and adults reported moderate sensitivity to environmental noise.

Table 54: Reported Noise Sensitivity (n=64)

	Mean	SD	25 th	75 th	Max
Child	29.9	9.3	24.8	37.0	45.0
Adult	29.8	8.9	22.0	37.0	48.0

*Notes: Noise sensitivity scores are computed from a Likert scale from questions based on a modified Weinstein Noise Sensitivity Scale (see questions 96-105 in the questionnaire (**Appendix 1**)).¹³⁹ A higher score indicates greater sensitivity to noise.

Noise was a frequent concern among participating households. About one-third of parents (32%) reported that indoor noise bothered them when they were inside their homes, while a majority (55%) said they were disturbed by outdoor noise heard while indoors (**Table 55**). In contrast, two-thirds (68%) stated that outdoor noise did not bother them when they were outside, suggesting that residents may be more sensitive to outdoor noise when they are inside their homes. When asked to characterize neighborhood sound levels, most respondents described their surroundings as moderately quiet (47%), with smaller proportions reporting very quiet (18%) or moderately loud (18%) conditions; only 5% perceived their neighborhoods as very loud. Only 7% of respondents said they had filed a formal noise complaint, most often citing disruptive neighbors, loud music, or, in one case, diesel trucks. Overall, these findings suggest that while most residents perceive their communities as relatively quiet, a substantial share experience annoyance from outdoor noise penetrating indoors, highlighting the role of housing characteristics and proximity to traffic in shaping perceived noise exposure.

Table 55: Parent perception of environmental noise.

Question	Response	Count and Percent
Does indoor noise bother you when you are inside?	No	44 (69%)
	Yes	20 (32%)
Are you bothered by outdoor noise when inside?	No	29 (46%)
	Yes	35 (55%)
Does outdoor noise bother you when you are outdoors?	No	43 (68%)
	Yes	21 (33%)
How would you generally describe sound in your neighborhood?	Very quiet	11 (18%)
	Moderately quiet	30 (47%)
	Not quiet or loud	9 (15%)
	Moderately loud	11 (18%)
	Very loud	3 (5%)
Did noise change during COVID-19?	No	38 (60%)
	Yes	25 (40%)
	Don't know	1 (2%)
If yes, how	More noise	7 (27%)
	Less noise	17 (66%)
	Don't know	2 (8%)
Have you ever filed a noise complaint ^a .	No	60 (94%)
	Yes	4 (7%)

^a Respondents who answered “Yes” to filing a noise complaint gave the following reasons: “Called the police for serious situation”; “Called about the diesel trucks”; “Called sheriff due to loud music next door”; “neighbor’s being noisy”.

We also investigated the association of noise exposure and adult sleep quality. Overall, many participants reported poor sleep quality, with 55% of adults reporting “fairly bad” or “very bad” sleep quality during the last month. However, there were no significant associations between sleep quality and noise exposure. Fifty-three percent of adult participants reported “ok” to “very good” sleep quality. Note, we did not ask parents to report on child sleep quality.

3.3.6.3 Child noise exposure and academic challenges

Analysis of indoor noise exposure indicated modest but noteworthy associations with child academic and behavioral outcomes. Children living in homes with higher average indoor noise levels were more likely to have reported academic challenges or diagnosed learning conditions such as ADHD or dyslexia (**Table 56**). Mean indoor noise exposure was significantly higher among children with academic challenges (66 dBA vs. 62 dBA, $p = 0.01$) and borderline significant among those with a diagnosed learning challenge (67 dBA vs. 62 dBA, $p = 0.05$). No statistically significant relationship was observed between indoor noise and reported behavioral problems at school (**Table 56**).

Table 56: Association Between Indoor Noise Exposure and Child Behavioral and Academic Outcomes.

Question	Category	N	Mean dBA	90 th	p-value
Child has academic challenges?	No	36	62	64	0.01
	Yes	21	66	67	
Child diagnosed with ADHD, dyslexia, or other learning challenge?	No	42	62	64	0.05
	Yes	15	67	68	
Child has behavioral problems or challenges at school?	No	48	64	66	0.33
	Yes	9	63	67	

3.3.7 Passive PM Sample Results

3.3.7.1 PAS results from Fresno homes

Twenty indoor and 16 outdoor UNC passive aerosol samples (PAS) were collected from 8 homes in Fresno, CA. The PAS results show clear differences in indoor and outdoor particle composition among the Fresno homes (**Figures 17 and 18**). Indoor samples indicated that carbonaceous material accounted for a large share of total particle mass, consistent with contributions from common household sources such as cooking, cleaning, and occupant activities. Smaller fractions of crustal material and trace metals were observed, suggesting limited infiltration of outdoor dust and soil. The predominance of carbon-rich material indoors highlights the influence of occupant behaviors and the relatively enclosed nature of indoor environments, which can concentrate particles from internal sources. In contrast, the outdoor PAS results (n = 16 from the same 8 homes) showed a larger contribution from crustal and elemental components, reflecting the influence of ambient dust and regional sources such as vehicular traffic and agricultural activity.

Outdoor samples contained a higher fraction of inorganic and metallic particles compared to indoor air, with more variability across sampling sites. Detected metallic particles included heavy metals such as lead (Pb) (>20 µm), chromium (Cr) (>3 µm), and copper (Cu) (>3 µm). Overall, these findings suggest that while outdoor air in Fresno contributes to particle infiltration, indoor emissions and activity patterns are dominant drivers of indoor aerosol composition, reinforcing the need to address both infiltration and source control in exposure mitigation strategies.

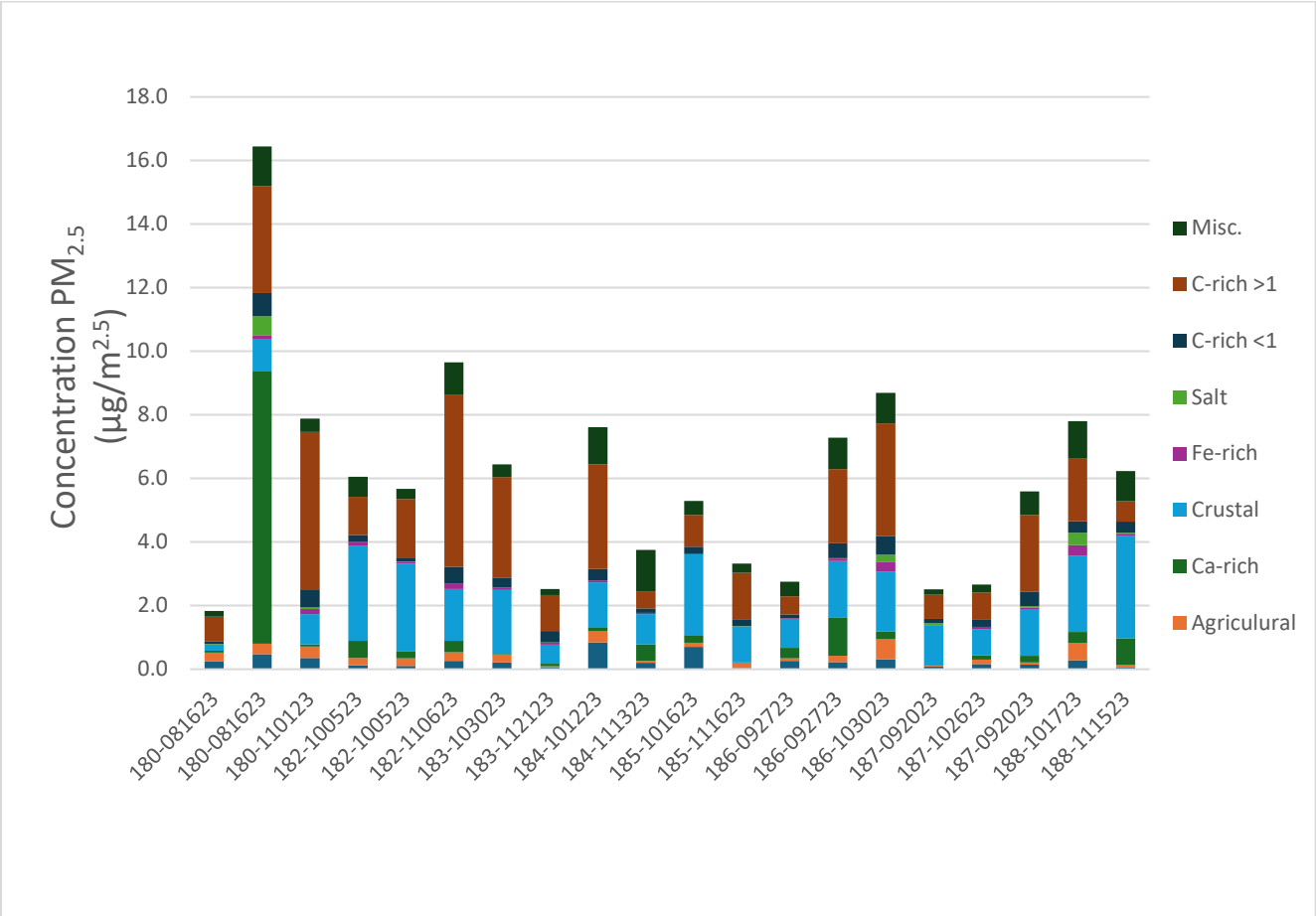


Figure 16: Indoor passive PM sample results by particle composition category (20 samples collected from 8 Fresno homes).

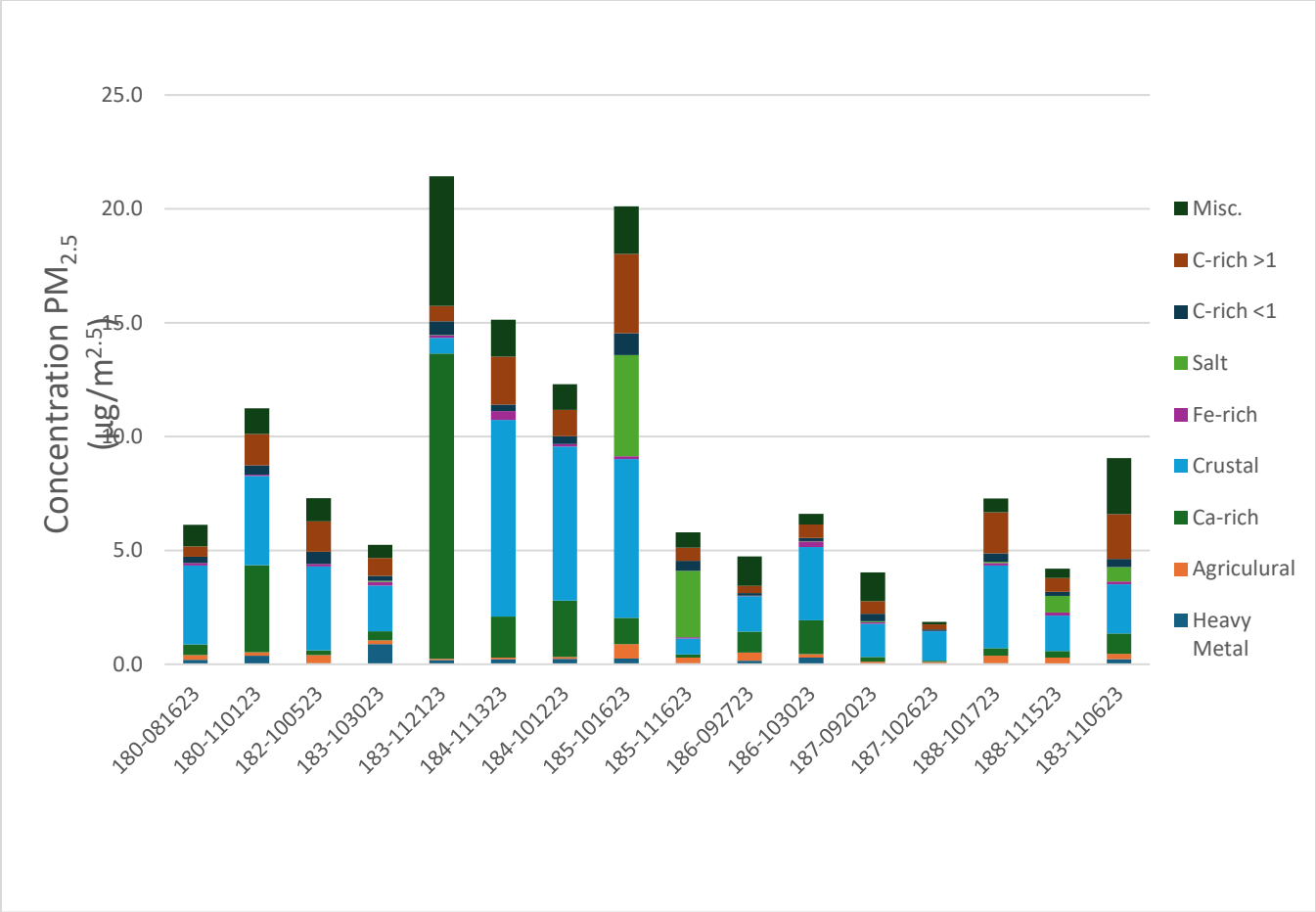


Figure 17: Outdoor passive PM sample results by particle composition category (16 samples collected from 8 Fresno homes).

3.3.7.2 PAS Results from Merced homes

Eleven indoor and 10 outdoor UNC passive aerosol samples (PAS) were collected from 5 homes in Merced, CA. The PAS results show distinct differences in the composition of indoor and outdoor particulate matter among Merced homes (**Figures 19 and 20**). Indoors samples were primarily composed of carbonaceous material, consistent with indoor activities such as cooking, heating, and cleaning. Minor contributions from crustal and metallic particles indicate limited infiltration of outdoor dust and soil. The relative uniformity in indoor composition across homes suggests that indoor sources and occupant behaviors, rather than outdoor influences, play the dominant role in shaping indoor aerosol characteristics.

The 10 outdoor PAS samples collected from the same homes exhibited a greater proportion of crustal and inorganic material compared with the indoor samples. This pattern reflects contributions from regional outdoor sources such as traffic, agricultural activity, and resuspended soil. Outdoor particle composition was more variable across sites, likely due to differences in proximity to major roads and local emission sources. Together, these results

underscore that while outdoor aerosols contribute to overall particulate levels, indoor-generated emissions remain the primary determinant of exposure composition within Merced residences.

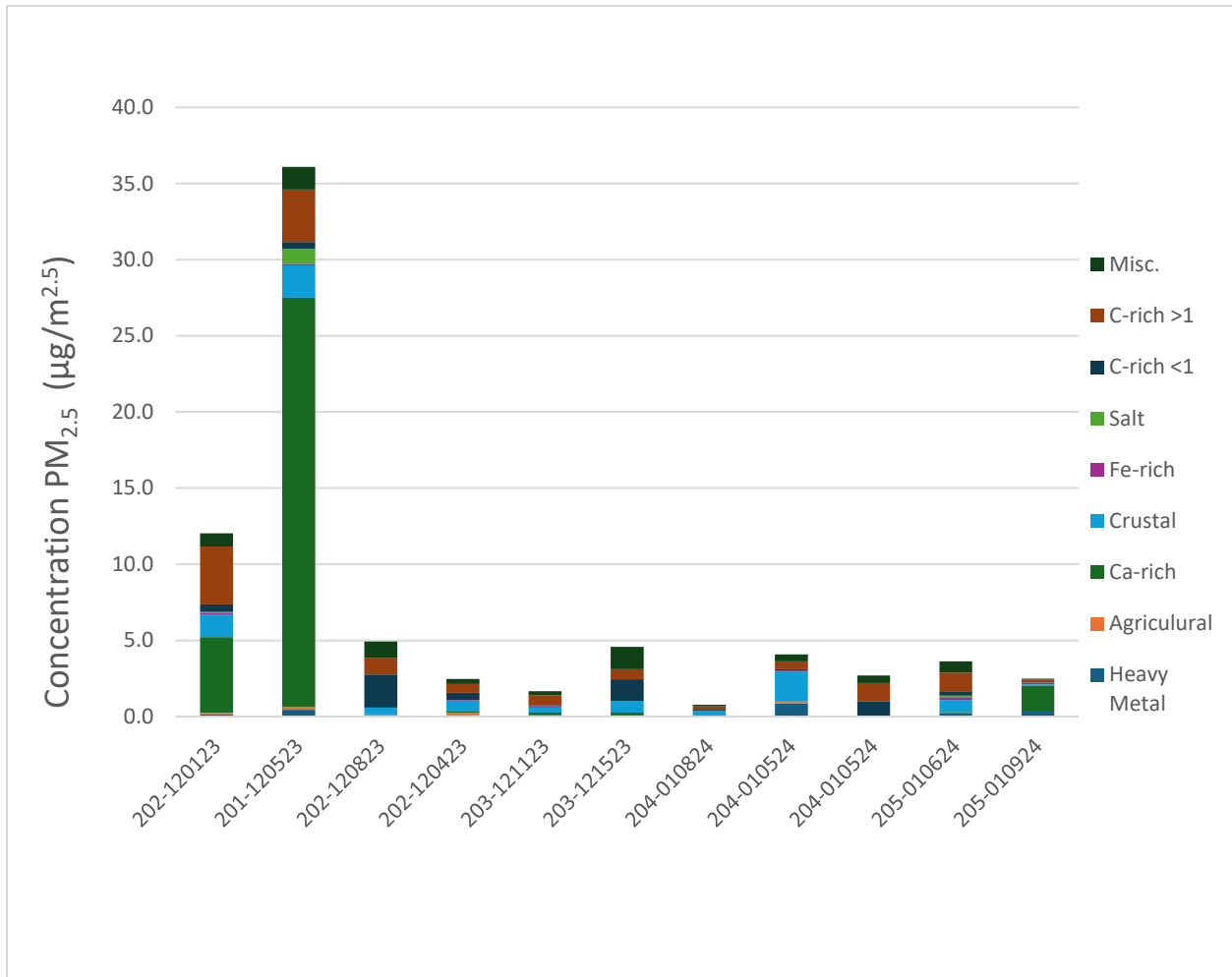


Figure 18: Indoor passive PM sample results by particle composition category (11 samples collected from 5 Merced homes).

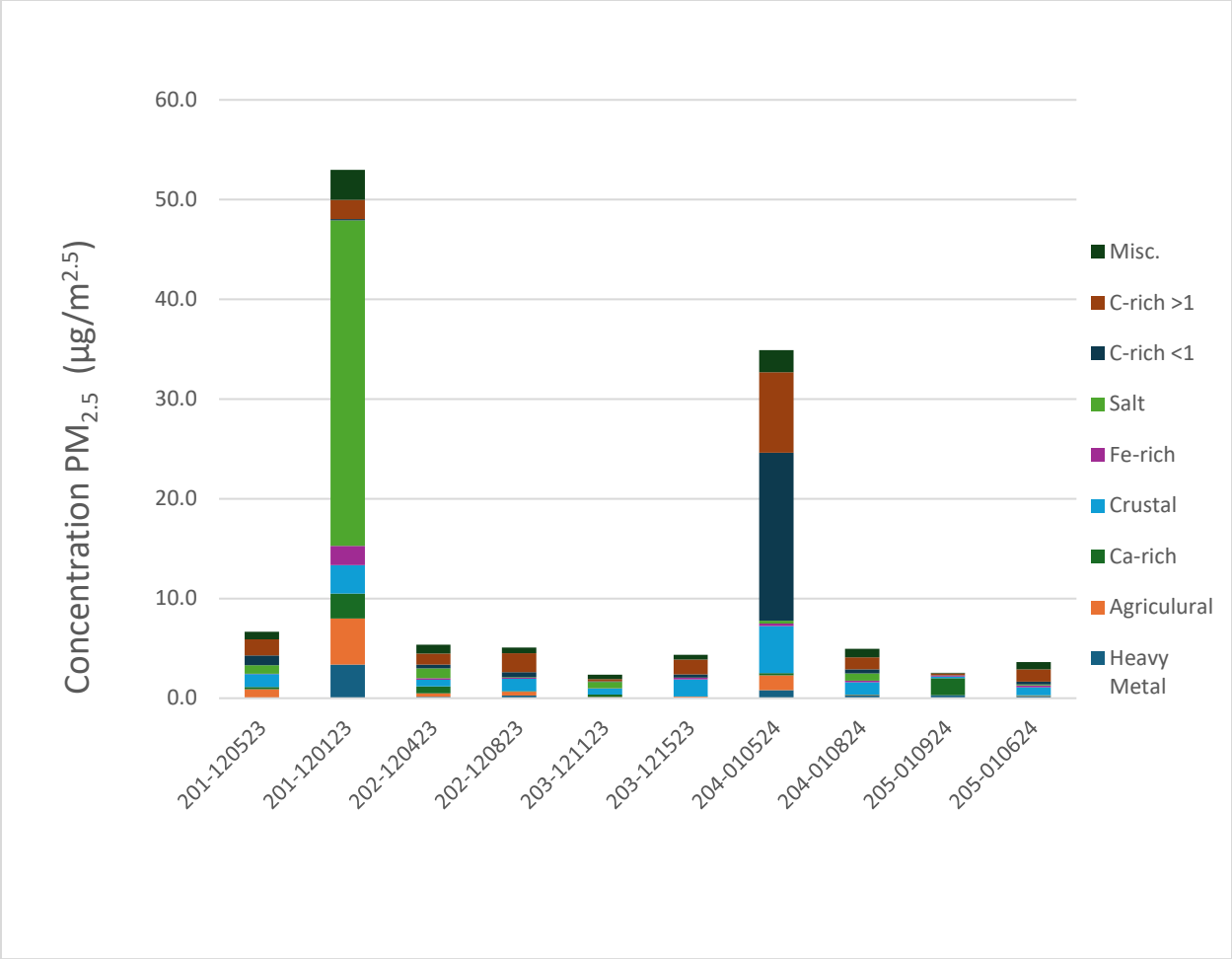


Figure 19: Outdoor passive PM sample results by particle composition category (10 samples collected from 5 Merced homes).

3.4 Health Risk Characterization

3.4.1 Non-Cancer Risk Estimation

Comparison of indoor SPHERE BTEX measurements with OEHHA and U.S. EPA health-based benchmarks showed that most compounds posed minimal non-cancer risk, with hazard quotients (HQs) well below 1 (**Table 57**). For benzene, HQs were low relative to both the chronic REL (50th percentile HQ = 0.086; 95th percentile = 0.28) and the RfC (<0.01–0.03). Ethylbenzene, toluene, and xylenes all showed HQs <0.05, indicating exposures far below concern levels. Naphthalene similarly had very low HQs (<0.05). Summing the HQs in cases where the health benchmarks are based on similar outcomes does not change these results. In contrast, formaldehyde exceeded health-based thresholds, with HQs above 1 for both the REL and RfC. The HQs based on the 95th percentile formaldehyde concentration and chronic REL and RfC were 5.8 and 7.5, respectively. These results indicate that, among the measured compounds, only formaldehyde concentrations exceeded reference values, suggesting a potential non-cancer health risk that warrants further attention. Indoor formaldehyde concentrations measured in 24 SPHERE homes are summarized in **Table 47** above.

Table 57: Comparison of indoor BTEX, formaldehyde, and naphthalene concentrations with health-based reference values.^a

BTEX	8-hour REL (µg/m ³)	Chronic REL (µg/m ³)	Inhalation RfC (µg/m ³)	Hazard Quotient Chronic (50th)	Hazard Quotient Chronic (95th)	Hazard Quotient RfC (50th)	Hazard Quotient RfC (95th)
Benzene	3	3	30	0.086	0.28	<0.01	0.03
Ethylbenzene	--	2,000	1,000	<0.01	<0.01	<0.01	<0.01
Toluene	830	420	5,000	<0.01	0.01	<0.01	<0.01
Xylenes (o-, m- and p-)	--	700	100	<0.01	<0.01	0.02	0.05
Formaldehyde	9	9	7	2.011	5.81	2.59	7.46
Naphthalene	--	9	3	<0.01	0.02	<0.01	0.05

^aOEHHA Reference Exposure Levels (RELs)¹³³; benzene and xylenes RfCs^{134,135}; ethylbenzene RfC¹³⁶; toluene RfC¹³⁷; formaldehyde RfC¹³³; naphthalene RfC¹⁴⁰.

Outdoor concentrations of BTEX compounds (benzene, ethylbenzene, toluene, and xylenes) and naphthalene were generally well below health-based reference values established by OEHHA and the U.S. EPA (**Table 58**). The indoor and outdoor concentrations of BTEX are presented in **Tables 42 and 43** and naphthalene (NAP) in **Tables 48 and 49**, respectively. Hazard quotients for most compounds at both the 50th and 95th percentiles were less than 0.1,

indicating exposures did not exceed health-based thresholds. The highest relative values were observed for benzene, with chronic REL hazard quotients of 0.13 (50th) and 0.41 (95th percentile), though still below 1, suggesting no exceedance of reference values. Naphthalene and xylenes showed very low hazard quotients across all comparisons, and ethylbenzene and toluene were consistently negligible relative to their reference concentrations. Formaldehyde is not included in this table because it was measured only indoors and not outdoors.

Table 58: Comparison of outdoor BTEX and naphthalene concentrations with health-based reference values (OEHHA RELs and U.S. EPA RfCs).a,b,c

Pollutant	8-hour REL (µg/m ³)	Chronic REL (µg/m ³)	Inhalation RfC (µg/m ³)	Hazard Quotient Chronic REL (50th)	Hazard Quotient Chronic REL (95th)	Hazard Quotient RfC (50th)	Hazard Quotient RfC (95th)
Benzene	3	3	30	0.13	0.41	0.01	0.04
Ethylbenzene	--	2,000	1,000	–	<0.01	–	<0.01
Toluene	830	420	5,000	<0.01	<0.01	<0.01	<0.01
Xylenes (o-, m-, p-)	--	700	100	<0.01	<0.01	<0.01	0.04
Naphthalene	–	9	3	<0.01	<0.01	<0.01	0.02

^aFormaldehyde is not included because it was measured indoors only.

^bOEHHA RELs¹³³; xylenes and benzene RfC^{134,135}; ethylbenzene RfC¹³⁶; toluene RfC¹³⁷ and naphthalene RfC¹⁴⁰.

^cThe PAHs fluorene, phenanthrene and pyrene do not have established RELs or RfCs^{140,142,143}.

3.4.2 Cancer Risk Evaluation

We evaluated potential inhalation cancer risk concerns for formaldehyde, naphthalene, benzene, and ethylbenzene by computing ratios of potential exposure divided by OEHHA's Proposition 65 No Significant Risk Levels (NSRLs) for adult women, male children, and female children. (The NSRL represents a chronic exposure intake with potential cancer risks exceeding one in 100,000 (10⁻⁵)). If the VOC measurements we conducted are reflective of long-term average concentrations and exposures, hazard ratios (HRs) >1 indicate exposure exceeding the NSRL. **Tables 59–64** present these estimates separately for indoor and outdoor exposure scenarios and for median (50th percentile) and high-end (95th percentile) exposure levels.

Table 59 shows that adult women experienced the highest potential cancer risk from indoor formaldehyde exposure, with a hazard ratio of 7.3 at the median level and 21.2 at the 95th percentile—well above the NSRL of 40 µg/day. Indoor exposure to benzene approached the NSRL at the 95th percentile (HR = 1.1), while naphthalene and ethylbenzene remained well below 1 (HRs ≤ 0.5).

Table 59: Indoor hazard ratios comparing adult women VOC exposure to OEHHA No Significant Risk Levels (NSRLs) (mean age: 42 years)[XW

Pollutant	Exposure Estimates (µg/day) 50th%	Exposure Estimates (µg/day) 95th%	NSRL (µg/day)	Hazard Ratio 50th%	Hazard Ratio 95th%
Formaldehyde	293.2	745.2	40	7.3	21.2
Naphthalene	0.3	2.7	5.8	0.04	0.5
Benzene	4.2	13.6	13	0.3	1.1
Ethylbenzene	9.1	21.1	54	0.2	0.4

^aInhalation rate based on U.S. EPA 2011 Exposure Factors Handbook.¹²⁴

Table 60 presents cancer risk concerns for adult women exposed to outdoor air. Outdoor benzene levels exceeded the NSRL at the 95th percentile (HR = 1.5) and approached it at the median (HR = 0.5), indicating a potential cancer concern. Naphthalene and ethylbenzene again had low hazard ratios (HR ≤ 0.2). Formaldehyde was not measured outdoors.

Table 60: Outdoor hazard ratios comparing adult women VOC exposure to OEHHA No Significant Risk Levels (NSRLs) (mean age: 42 years).a, b

Pollutant	Exposure Estimates (µg/day) 50th%	Exposure Estimates (µg/day) 95th%	NSRL (µg/day)	Hazard Ratio 50th%	Hazard Ratio 95th%
Naphthalene	0.2	1.2	5.8	0.04	0.2
Benzene	6.3	19.8	13	0.5	1.5
Ethylbenzene	NC	11.8	54	NC	0.2

^aInhalation rate based on the U.S. EPA 2011 Exposure Factors Handbook.¹²⁹

^bOutdoor formaldehyde was not measured.

NC = Not calculated because detection frequency for ethylbenzene < 50%.

For male children, **Table 61** indicates that indoor formaldehyde exposure also posed the greatest cancer risk, with HRs of 6.1 (median) and 17.5 (95th percentile). Benzene remained below 1.0 at both exposure levels (HR = 0.3 and 0.9), and the naphthalene and ethylbenzene 95th percentile HRs were even lower (HR ≤ 0.4).

Table 61: Indoor hazard ratios comparing male child VOC exposure to OEHHA No Significant Risk Levels (NSRLs) (mean age: 9 years).a

Pollutant	Exposure Estimates (µg/day) 50th%	Exposure Estimates (µg/day) 95th%	NSRL (µg/day)	Hazard Ratio 50th%	Hazard Ratio 95th%
Formaldehyde	294.9	617.3	40	6.1	17.5
Naphthalene	0.2	2.2	5.8	0.04	0.4
Benzene	3.5	11.3	13	0.3	0.9
Ethylbenzene	7.5	17.5	54	0.1	0.3

^aInhalation rate based on U.S. EPA 2011 Exposure Factors Handbook.¹²⁹

Table 62 shows that for male children outdoors, benzene again exceeded the NSRL at the 95th percentile (HR = 1.3) and approached it at the median (HR = 0.4). Naphthalene and ethylbenzene were below concern thresholds (HR ≤ 0.2), and outdoor formaldehyde was not measured.

Table 62: Outdoor hazard ratios comparing male child outdoor VOC exposures to OEHHA No Significant Risk Levels (NSRLs) (Mean Age: 9 years).a,b

Pollutant	Exposure Estimates (µg/day) 50th%	Exposure Estimates (µg/day) 95th%	NSRL (µg/day)	Hazard Ratio 50th%	Hazard Ratio 95th%
Naphthalene	0.2	1	5.8	0.03	0.2
Benzene	5.2	16.4	13	0.4	1.3
Ethylbenzene	NC	9.8	54	NC	0.2

^aInhalation rate based on U.S. EPA 2011 Exposure Factors Handbook.¹²⁵

^bOutdoor formaldehyde was not measured.

NC = Not calculated because detection frequency for ethylbenzene < 50%.

For female children, **Table 63** indicates indoor formaldehyde exposure resulted in hazard ratios of 5.6 (median) and 16.2 (95th percentile), confirming consistent elevated risk across age and sex. Benzene HRs were 0.3 and 0.8, while naphthalene and ethylbenzene remained below 0.4.

Table 63: Indoor hazard ratios comparing female child VOC exposures to OEHHA No Significant Risk Levels (NSRLs) (mean age: 9 years).a

Pollutant	Exposure Estimates (µg/day) 50th%	Exposure Estimates (µg/day) 95th%	NSRL (µg/day)	Hazard Ratio 50th%	Hazard Ratio 95th%
Formaldehyde	224.6	570.9	40	5.6	16.2
Naphthalene	0.2	2.0	5.8	0.03	0.4
Benzene	3.2	10.4	13	0.3	0.8
Ethylbenzene	7	16.1	54	0.1	0.3

^aInhalation rate based on EPA 2011 Exposure Factors Handbook.¹²⁹

Finally, **Table 64** shows that outdoor benzene exposure for female children exceeded the NSRL at the 95th percentile (HR = 1.2) and was close to it at the median (HR = 0.4). Hazard ratios for naphthalene and ethylbenzene were well below 1.0, and formaldehyde was not measured in outdoor air.

Table 64: Outdoor hazard ratios comparing female child VOC and naphthalene exposure to OEHHA No Significant Risk Levels (NSRLs) (mean Age: 9 years).a,b

Pollutant	Exposure Estimates (µg/day) 50th%	Exposure Estimates (µg/day) 95th%	NSRL (µg/day)	Hazard Ratio 50th%	Hazard Ratio 95th%
Naphthalene	0.2	0.9	5.8	0.03	0.2
Benzene	4.8	15.1	13	0.4	1.2
Ethylbenzene	NC	9.1	54	NC	0.2

^aInhalation rate based on EPA 2011 Exposure Factors Handbook.¹²⁹

^bOutdoor formaldehyde was not measured.

NC = Not calculated because detection frequency for ethylbenzene < 50%.

In summary, indoor formaldehyde and outdoor benzene exposures represented the most concerning cancer risks across populations. Formaldehyde posed elevated indoor risks for all groups, while benzene exceeded the NSRL in outdoor scenarios for adults and children at the 95th percentile.

3.4.3 Noise Exposure Hazard Evaluation

For all three time periods (24-hours, daytime, and nighttime) and settings (indoor, outdoor, and personal) observed noise exposure were well below all recommended thresholds for hearing protection. () The highest 10-hour average we observed was 70 dBA for daytime outdoor noise levels. We observed a single 1-minute maximum noise exposure of 85 dBA, but this was an outlier. Conversely, many homes were higher than the outdoor Fresno Municipal Standards (**Table 1**).

4 Opportunities and Challenges of Community Engaged Research

Partnering with local community organizations was central to this project and provided valuable opportunities for building relationships between researchers and community partners and strengthening local capacity. At the same time, these partnerships introduced practical challenges that influenced the study's implementation. For example, in Stockton, unexpected staffing changes within the community organization reduced field work capacity. As a small organization, this transition had a proportionally larger impact on their ability to carry out tasks such as scheduling home visits and managing the monitoring equipment.

These experiences highlight how differences in community partner organizational capacity and resources affect capacity to conduct community-engaged research. Overall, the project was a success, but the challenges underscored the need to complete a thorough workload assessment and staffing plan with each partner and, importantly, contingencies, as part of the implementation plan. In Stockton, expertise acquired during the study strengthened one key staff member's successful application to an environmental health-related local government position which has broadened continuing and long-term collaboration between the community and academic partners, but temporarily reduced the capacity of our Stockton community partner to continue the research. Thus, while the community engagement yielded clear capacity-building benefits across the region, it also temporarily reduced capacity to sustain our shorter-term research collaboration. Similar community-capacity building occurred with our Fresno community partners, where one staff member is now completing an MPH program at UC Berkeley. Additionally, a UC Merced graduate student helped coordinate the study with both community partners and is now working as a key scientist with the Fresno community partner. In summary, the net effect of the collaborative, community engaged research strengthened relationships between participants, community partners, and researchers and significantly increased region-wide community capacity to conduct air quality and other environmental health research and education across the San Joaquin Valley and resulted in long term partnerships that continue today.

5 Strengths and Limitations

One strength of this study is its comprehensive, multi-faceted exposure monitoring. The project conducted an approximately nine-month field campaign spanning both warm and cool seasons. Real-time sensors measured key pollutants – including PM_{2.5}, O₃, NO₂, and black carbon – at each home, while a dedicated noise monitor recorded continuous sound levels. Indoor and outdoor samples were collected simultaneously at each residence, enabling direct comparisons of infiltration and exposure patterns. Time integrated samples were also collected at each residence and analyzed in the laboratory for VOCs, formaldehyde, and PAHs, providing a rich chemical profile of many home environments. Notably, this was the first California study to pair concurrent indoor/outdoor air monitoring with simultaneous noise measurements and collection of questionnaire-based information in disadvantaged communities, demonstrating an innovative multi-exposure assessment approach.

The study also emphasized strong partnerships with community-based organizations and inclusive participant recruitment and community relevance. All study materials (questionnaires, consent forms, etc.) were offered in both English and Spanish. By focusing on neighborhoods at high risk of poor air quality in the San Joaquin Valley, the study responded to community concerns about disproportionate environmental burdens. These strengths, including robust, matched indoor/outdoor pollutant and noise measurements, seasonal coverage, in-depth questionnaires, and culturally inclusive recruitment, combine to create a rich dataset for understanding environmental exposures in disadvantaged communities.

One limitation was the smaller sample size recruited in Stockton. Twelve families (versus ~45 originally targeted) completed the full protocol in Stockton. To partially offset this reduction, 52 families were enrolled in Fresno (instead of 45), for a total of 64 families. The smaller sample size limited opportunities to compare exposures between the two locations and reduced overall statistical power of the study. The reduced sample size also meant that repeated visits or longitudinal sampling in Stockton, originally intended to capture short-term variability, could not be performed.

A limitation that is common to all cross-sectional cohort studies is the inability to assign causality to the observed relationships. In this study, we examined several cross-sectional associations of air pollutant and noise exposure with selected health outcomes (parent-reported respiratory symptoms, asthma, and child academic and behavior challenges). These health outcomes likely developed over months or years and any observed associations, while suggestive, do not indicate causal relationships. We also did not adjust for potential covariates, such as wind direction, land use, or other factors, due to the limited sample size.

The timing of this overall project was particularly challenging due to the ongoing COVID-19 pandemic. Compliance with changing COVID-19 incidence and exposure prevention guidelines also contributed to administrative and logistical challenges that delayed operationalization of the field work. Other challenges arose during study recruitment when, in some cases, fear of COVID-19 exposure and infection reduced potential participant's interest in

enrolling in the study. This bias may have led to fewer participants with chronic respiratory illnesses, such as asthma, from participating.

In summary, despite several limitations and challenges, this study has produced new information and a rich data set examining air pollution and noise exposure in the San Joaquin Valley.

6 Summary and Conclusion

This study provides new information about cumulative environmental exposures experienced by households in disadvantaged communities (DACs) of California's San Joaquin Valley (SJV). By integrating indoor, outdoor, and personal monitoring data for multiple pollutants, including fine particulate matter (PM_{2.5}), black carbon (BC), volatile organic compounds (VOCs), formaldehyde, polycyclic aromatic hydrocarbons (PAHs), and environmental noise, alongside household questionnaire data, the results provide important information about the magnitude of exposure and the implications for health and environmental equity in overburdened communities.

Overview of Literature

Air Quality: Air pollution in California, especially in the San Joaquin Valley (SJV), remains a major public health concern despite progress under federal and state regulations. The U.S. Environmental Protection Agency (EPA) regulates six “criteria pollutants,” but California communities—particularly disadvantaged and low-income neighborhoods—continue to experience high exposures to nitrogen dioxide (NO₂), ozone (O₃), fine particulate matter (PM_{2.5}), and volatile organic compounds (VOCs) from traffic, industry, agriculture, and household sources. Many studies indicate that exposure and health impacts are unequally distributed, with lower-income populations living closer to major pollution sources, especially traffic-related air pollution, experiencing higher rates of exposure and asthma, cardiovascular disease, and premature mortality. In the SJV and other regions, air quality disparities are exacerbated by historical development patterns and residential proximity to major transportation corridors. Personal and behavioral factors, such as the use of unventilated gas stoves or the use of in-home filtration, can also increase or decrease exposure risks, but cannot fully mitigate high regional or local emissions. In California, while California's air quality has generally improved through policies such as the Community Air Protection Program (AB 617), persistent inequities remain.

Noise: Many sources of air pollution also generate noise pollution, particularly from transportation corridors, airports, ports, and industrial or energy facilities. Although data on noise exposure in California are limited, noise, especially from transportation, is likely to be a major health burden. In the United States, regulation of environmental noise shifted from federal to state and local authority after the closure of the EPA's Office of Noise Abatement and Control in 1981. California cities and counties set their own noise standards. Statewide, Caltrans has invested in substantial mitigation efforts such as constructing sound walls and developing

quieter road surfaces. Disadvantaged and lower-income communities are often disproportionately exposed to noise due to their proximity to major roadways and industrial zones.

A growing literature demonstrates that chronic noise exposure is linked to several adverse outcomes, including sleep disruption, cardiovascular disease, obesity, diabetes, and mental health issues such as anxiety and depression. Evidence also suggests potential impacts on birth outcomes, child cognitive development, and cognitive decline in older adults. Noise may act through stress-related biological pathways involving hormonal, inflammatory, and metabolic dysregulation. When combined with air pollution, noise can have independent or additive health effects; however, few studies have examined this interaction, particularly in California. Overall, environmental noise is an underrecognized but significant public health issue, and more research is needed to assess exposures, evaluate mitigation policies, and address the disproportionate burdens faced by vulnerable and disadvantaged populations.

Criteria Pollutants: Fine Particulate Matter (PM_{2.5}), NO₂, and O₃

Mean 24-hour PM_{2.5} concentrations were 14.8 µg/m³ indoors, 11.9 µg/m³ outdoors, and 12.1 µg/m³ for personal exposures. The average I/O ratio of 2.23 indicates that household activities, such as cooking, heating, smoking, and use of combustion appliances, may have contributed more to indoor PM_{2.5} levels than outdoor infiltration alone. Questionnaire data supported these patterns, with homes reporting frequent cooking, smoking, or use of combustion appliances showing higher indoor PM_{2.5} concentrations. These results suggest that everyday behaviors and housing characteristics play a central role in shaping personal exposure. In disadvantaged communities with older housing stock, limited ventilation, and higher reliance on combustion-based appliances, these factors can significantly elevate indoor pollution levels and exacerbate exposure and health risks.

Published studies of California homes generally report lower indoor PM_{2.5} concentrations than those observed in Table 8, which reflects measurements from SPHERE homes located in the San Joaquin Valley, a region out of compliance with federal and state ambient air quality standards.¹¹⁵ In statewide or multi-region studies of newer and code-compliant homes, indoor PM_{2.5} median values typically range from 5 to 10 µg/m³ (Chan et al., 2020¹¹¹; Zhao et al., 2021¹¹²), well below the 14.8 µg/m³ indoor mean and the 31.1 µg/m³ 90th percentile reported in Table 8. Although indoor PM_{2.5} can temporarily increase due to cooking or infiltration of wildfire smoke (Less et al., 2015¹¹³; Zhao et al., 2021¹¹²), most studies do not report sustained indoor concentrations exceeding 30 µg/m³ under normal conditions (Chan et al., 2020¹¹¹). These comparisons suggest that long-term indoor PM_{2.5} exposures in many California residences, particularly those outside the San Joaquin Valley, are lower than the levels we observed in SPHERE. Elevated SPHERE indoor values possibly reflect higher ambient PM_{2.5} in the San Joaquin Valley,¹¹⁵ proximity to traffic corridors, regional meteorology, and potential differences in housing characteristics such as filtration and ventilation.

Similarly, indoor NO₂ levels reported in other California home studies are generally lower than those presented in Table 8. Multi-day indoor NO₂ concentrations in homes with gas stoves are typically between 15–20 ppb (Mullen et al., 2016¹¹⁴; Zhao et al., 2021¹¹²), and homes with

electric cooking often report values below 10 ppb (Chan et al., 2020¹¹¹; Less et al., 2015¹¹³), both lower than the 23 ppb indoor mean and 39 ppb 90th percentile in Table 8. While homes with heavy gas appliance use or limited ventilation can experience short-term NO₂ peaks during cooking (Less et al., 2015¹¹³; Mullen et al., 2016¹¹⁴), the longer-term levels reported in previous studies remain below the peak values observed in SPHERE. This difference may reflect the SPHERE study's setting in the San Joaquin Valley, where ambient NO₂ is locally higher due to traffic and industrial sources, and where infiltration into homes may contribute more substantially to indoor concentrations than in other parts of the state.

Notably, indoor PM_{2.5}, NO₂, and black carbon (see below) concentrations were inversely correlated with proximity to State Route-99, which indicates that homes closer to SR-99 had higher levels, thus linking residence near major transportation corridors with significant traffic-related air pollution exposure, including diesel exhaust.

Although the SJV is considered out of attainment for O₃,¹¹⁵ the concentrations we observed were generally lower than air quality standards and were not associated with potential exposure determinants.

Black Carbon

Black carbon, an indicator of diesel exhaust exposure, was consistently detected indoors and outdoors, reflecting infiltration of traffic-related emissions into homes. Elevated indoor BC levels in homes located in AB 617 communities and census tracts with high CalEnviroScreen (CES) ranking underscore the persistent environmental inequities experienced by SJV residents. In combination with measured criteria pollutants such as NO₂ and PM_{2.5}, these data confirm the dual burden of outdoor infiltration and indoor sources. While regulatory measures have reduced regional diesel exhaust emissions, concentrations in SJV DAC households remain elevated compared with households in census tracts with lower CES environmental burden scores or outside AB 617 communities.²

Volatile Organic Compounds (VOCs), Formaldehyde, and Polycyclic Aromatic Hydrocarbons (PAHs)

VOCs: Indoor VOC concentrations measured in 16 Fresno homes were consistently higher than outdoor levels, with indoor-to-outdoor (I/O) ratios often ranging from 2 to 6 across compounds such as toluene, xylenes, and ethylbenzene. Benzene was an exception, with similar indoor and outdoor levels, suggesting the main source to be infiltration from outdoor sources such as traffic emissions, as opposed to indoor sources. Elevated indoor VOCs indicate that household products, cleaning agents, and building materials may be significant contributors to total exposure. Combined with PAH and formaldehyde findings, the VOC data highlight the important role of indoor sources in contributing to total exposures. Despite the small sample size, outdoor concentrations of BTEX compounds showed a strong correlation with proximity to the SR-99, indicating higher ambient pollutant levels nearer this major transportation corridor.

Formaldehyde: Indoor formaldehyde was measured in 27 samples from 24 homes in Fresno using passive samplers. Formaldehyde was detected in 81% of homes, with a median concentration of 18 µg/m³. These levels exceeded California and U.S. EPA health-based

reference levels ($9 \mu\text{g}/\text{m}^3$), underscoring formaldehyde as a priority indoor pollutant. Emissions are likely related to building materials, furnishings, and other consumer products.^{34,144,145} The relatively high proportion of the homes in this study with formaldehyde concentrations above health-based benchmarks is typical of other California studies.^{111,146}

Polyaromatic hydrocarbons (PAHs): We collected 59 indoor and 64 outdoor air samples from 61 homes in Stockton and Fresno to measure four PAHs (naphthalene, fluorene, phenanthrene, and pyrene). Naphthalene levels were highest, with mean concentrations of $45 \text{ ng}/\text{m}^3$ indoors and $26 \text{ ng}/\text{m}^3$ outdoors, and higher detection frequency indoors (47% vs. 33%, respectively). Other PAHs (fluorene, phenanthrene, pyrene) were detected infrequently at lower concentrations ($2\text{--}5 \text{ ng}/\text{m}^3$). Indoor-to-outdoor ratios highlighted that naphthalene was often elevated indoors (mean I/O $\sim 1.9\text{--}2.3$, maximum 12.3), suggesting indoor sources, while other PAHs showed little difference between these environments. Separately, naphthalene metabolites were elevated in urine samples collected from participating SPHERE children compared to national reference levels. Together with the environmental measurements, these findings indicate the need for future research to understand exposure naphthalene exposure variability, health risks, and mitigation approaches.

Comparison of exposure to health benchmarks: Health-based reference levels established by OEHHA and/or U.S. EPA were available for BTEX compounds, formaldehyde, and naphthalene concentrations. We computed the ratio of estimated exposures to adults and children to these benchmarks and computed hazard quotients. Most hazard quotients were below 1, indicating exposures did not exceed health-based thresholds. However, formaldehyde hazard quotients, based on non-cancer risks, exceeded 1 at both median and 95th percentile concentrations, suggesting potential concerns about chronic exposure. We also compared exposures to OEHHA No Significant Risk Levels (NSRLs) when they were available. (The NSRL represents a chronic exposure intake with potential cancer risks exceeding one in 100,000 (10^{-5})). If the single-day monitoring we conducted reflects long-term average concentrations in the homes, formaldehyde exposures in some homes would result in exposures that exceed the NSRL (up to 21 times the NSRL). In several instances the highest levels of the single-day benzene levels, if reflective of long-term averages in the homes, would result in exposures that exceed the NSRL by a small fraction (hazard quotient=1.5).

Environmental Noise

We monitored noise in 65 homes indoors, 46 homes outdoors, including 44 matched pairs, and one-day personal monitoring for 37 adults. Average noise levels ranged from 60–70 dBA, with minimum levels from 33 - 55 dBA, and one short-term peak up to 85 dBA. Personal exposures were highest over the 24-hour period (66 dBA). Levels at night tended to be lower. Indoor and outdoor noise levels tended to be similar and showed individual variability (indoor/outdoor ratios averaged ~ 1.0 , range = 0.70 - 1.8), but no significant correlations were observed between indoor and outdoor noise measurements. Overall, noise levels were highest and most variable in personal and outdoor environments during the day, while indoor

environments display more consistent and moderate noise levels across all time frames. Nighttime measurements consistently showed reduced noise levels across all settings.

Noise measurements were not significantly correlated with any measures of air quality. Traffic was significantly associated with outdoor noise at close distances (≤ 100 m) but showed no meaningful relationship with indoor noise. Noise was a common concern among households, with 32% of parents reporting annoyance from indoor noise and more than half (55%) disturbed by outdoor noise heard while indoors. Most residents described their neighborhoods as moderately or very quiet, though a subset (23%) perceived their neighborhoods as moderately or very loud. The few households that had filed noise complaints complained about neighbors or, in one case, heavy duty trucks.

Children living in homes with higher average indoor noise levels were more likely to have reported academic challenges or diagnosed learning disabilities. These analyses were limited by small sample size and short-term exposure assessment evaluated against learning difficulties that may take years to develop but are suggestive and warrant future research.

Conclusions

Households in disadvantaged San Joaquin Valley communities are exposed to multiple environmental pollutants, including fine particulate matter (PM_{2.5}), black carbon, VOCs (including BTEX compounds), PAHs, formaldehyde, and environmental noise. These exposures result from both traffic-related air pollution, agriculture, industrial activity, and other outdoor sources and indoor sources modified by housing conditions and behaviors. We found that exposures to fine particulate matter, black carbon, and VOCs (BTEX) were often higher in AB 617 designated communities and census tracts with high-CalEnviroScreen scores, and in homes nearer major traffic corridors. These findings are notable because they provide on-the-ground air quality measurements that validate the CES burden scoring system, which are largely based on emission inventories, not direct measurement of pollutants, and underscore ongoing environmental justice concerns and the need for remediation of high air pollution exposures in targeted populations.

The sample size was relatively small in this study; thus, our findings may not be generalizable to other areas. Exposures may be higher or lower in other communities that were not studied. While most concentrations were below health-based benchmarks, indoor formaldehyde levels frequently exceeded non-cancer reference values; also, in several instances, the highest levels of the single-day benzene levels, if reflective of long-term averages in the homes, would result in exposures that exceed the NSRL by a small fraction. These findings support targeted mitigation at the household level, such as reducing indoor combustion, the use of high-emitting materials and personal care products in homes, and improved ventilation and filtration (e.g., well-maintained HVAC with MERV-13+ or portable HEPA, and effective range hoods). At the community level, diesel/traffic emission controls and rerouting and integrated noise mitigation (e.g., sound walls, quieter pavement, and buffer zones) will also mitigate exposures. Additionally, incorporating noise indicators into CalEnviroScreen would expand the value of CES as a metric for characterizing communities with higher environmental

burdens. Continued community-engaged monitoring and evaluation of interventions are essential to track progress and the success of community exposure reduction plans (CERPs). Implemented together, these actions can lower cumulative exposures, protect vulnerable residents, especially children, and advance environmental justice across the Valley.

7 Recommendations

Targeted mitigation strategies are necessary to protect community health and advance environmental justice. Priority actions include: (1) reducing heavy-duty traffic emissions and rerouting freight corridors where feasible; (2) improving ventilation and filtration systems in homes; (3) promoting electrification to eliminate indoor combustion sources; (4) evaluate the efficacy of CARB polices to reduce formaldehyde and other VOCs from furnishings and consumer products; and (5) incorporating noise mitigation and monitoring into community air protection initiatives under AB 617. Strengthening local capacity for environmental monitoring and integrating combined air and noise exposure indicators into CalEnviroScreen will further enable equitable, data-driven policy responses.

Based on the study's findings, the following priority actions are recommended to reduce air pollution and noise exposures in disadvantaged communities (DACs):

1. **Target Traffic Pollution in Impacted Areas:** *Reduce emissions from heavy-duty traffic and freight operations in DAC regions.*

For several pollutants we found significantly higher levels in homes located near major roadways and trucking corridors. Black carbon, PM_{2.5}, and NO₂ concentrations were inversely associated with distance to State Route 99, a major truck route, with strong and statistically significant inverse associations for indoor concentrations. Further, black carbon and PM_{2.5} concentrations were highly correlated with each other, indicating that diesel truck traffic is a major contributor to particulate matter exposure. Communities designated for air quality action under AB 617 also showed approximately 70% higher indoor black carbon (diesel exhaust particulate) concentrations compared with other areas. Residents living closer to State Route 99 also experienced elevated VOC concentrations from traffic sources. Prioritizing emission reductions in these transportation “hotspots” will deliver substantial health benefits to the most affected residents.

Strategies to reduce these traffic-related pollution and noise exposures include:

- Targeted acceleration of the transition to zero-emission trucks and buses to prioritize DAC communities, strengthening enforcement of diesel emissions standards and anti-idling regulations, and rerouting high-volume freight corridors away from residential neighborhoods.
- Installing or upgrading roadside filtration barriers,
- Implementing “clean air zones” restricting older diesel vehicles,
- Expanding urban greenery along highways

2. Improve Indoor Air Quality through Ventilation and Filtration. *Enhance the indoor environments where residents spend the majority of their time.*

Many homes in this study had indoor pollutant levels similar to or higher than outdoor levels, such as PM_{2.5}, NO₂, and VOCs, reflecting a combination of indoor sources and outdoor pollutant infiltration. We also observed that black carbon concentrations were higher in homes without central air conditioning, likely due to the presence of air filters in these systems, underscoring the role of mechanical ventilation in reducing particulate exposures. Improved ventilation and filtration also help lower concentrations of indoor toxicants such as formaldehyde, which exceeded health guidelines in a majority of sampled homes.

Strategies to reduce these indoor exposures include:

- For homeowners, provide or incentivize the installation of kitchen range hoods or exhaust fans for use when cooking.
- Require all rental properties to have functioning kitchen range hoods or exhaust fans.
- Encourage the use of exhaust fans.
- Maintain HVAC systems with high-efficiency (MERV 13 or HEPA) filters.
- Provide portable air cleaners to households in high-pollution areas.
- State and local programs can expand weatherization and home-upgrade initiatives in DACs to include improved airflow, heat-recovery ventilators, and furnace filter upgrades to reduce indoor pollutant accumulation and promote overall respiratory health.

3. Mitigate Environmental Noise Exposure. *Implement community-level measures to reduce chronic noise exposure, particularly from transportation sources, and incentivize noise-reducing residential construction and practices.*

Policymakers and urban planners should prioritize noise reduction strategies, including:

- Constructing sound barriers or earth berms along highways and freight routes not already protected.
- Enforcing noise ordinances, such as restricting heavy truck traffic or construction activities during nighttime hours.
- Promoting quieter pavement technologies and vehicle designs.
- Add environmental noise to CalEnviroScreen as an environmental indicator.
- Require buffer zones or setbacks separating new housing developments from highways or industrial facilities.

At the state level, Caltrans has invested in substantial mitigation efforts such as constructing sound walls and developing quieter road surfaces along major roadways. These efforts should be bolstered and supported by an interagency effort involving CARB, OEHHA, and other agencies. Building on the AB 617 framework, future efforts should explicitly incorporate noise exposure as a co-factor in emission reduction and community health plans.

At the household level, to reduce noise intrusion:

- For homeowners, provide or incentivize the installation of double-pane windows, added wall insulation and vegetative buffers (e.g., trees and shrubs).
- Require all rental properties to have double-pane windows and added wall insulation.

These interventions, combined with expanded community green spaces, will help improve sleep, reduce stress, and enhance the quality of life for residents in DACs.

4. Strengthen Community-Based Planning and Community Capacity in Environmental Health Literacy. *Empower and integrate community members into planning for the reduction of cumulative air and noise pollution burdens.*

Enhanced community participation in decision-making ensures that interventions—such as traffic rerouting, facility permitting, or air-monitor placement—address local priorities and lived experiences. It is also important to support community members in understanding and managing environmental risks through education, outreach, and technical assistance. CARB has developed laudable programs as part of the AB 617 Community Air Protection Program with an emphasis on selected communicating. Extending these programs to other environmentally burdened areas will extend the benefits of air quality interventions and help reduce persisting exposure disparities.

CARB and partner agencies should continue to develop and distribute accessible materials on indoor and outdoor air quality, filtration, and noise mitigation; fund training workshops for residents and community leaders; and expand partnerships with local community-based organizations already engaged in environmental justice and public health advocacy. Improving environmental health literacy builds community resilience, promotes behavior change, and ensures that residents can fully participate in identifying and implementing solutions that benefit their neighborhoods.

5. Support continuing research. *Conduct studies to increase our understanding of environmental exposures and their health impacts and evaluate the success of exposure mitigation policies.*

Additional research is warranted to expand on the findings of this study:

- Our findings indicate higher traffic-related air pollution exposure in AB 617 and DAC communities and underscores the continuing need to assess exposure disparities in these communities with field monitoring data. This information is essential to evaluate the success of ongoing community exposure-reduction plans (CERPs). The emergence of low-cost sensors can provide hyper-local spatial resolution that can complement regulatory monitoring.¹⁴⁶ CARB is already supporting community monitoring efforts, especially for PM_{2.5}. Importantly, new low-cost (<\$500) monitors can now monitor NO₂, TVOCs, and carbon monoxide, and future low-cost devices may also be able to

accurately test for O₃. Costs for black carbon monitors are also declining. Expanded community-based monitoring for these TRAP pollutants would provide additional information on health risks and the success of exposure-reduction policies.

- This is the first California study to collect measurements and questionnaire-based information on community noise exposures. More work is needed to understand noise exposure and potential health impacts in California, especially on children's development and school performance.
- More work is needed to understand exposure to BTEX VOCs. Specifically, wider geographic monitoring is needed to understand the spatial variability of BTEX exposures, including areas outside AB 617 and DAC communities and away from major transportation corridors.

Summary

Collectively, these recommendations provide a roadmap for reducing cumulative air and noise exposures in California's most overburdened communities. Continued investment in community air and noise monitoring networks will help track progress and maintain accountability. Empowering residents with accessible data and participation opportunities strengthens the effectiveness and legitimacy of environmental health policies. By combining emission reductions, housing improvements, clean energy transitions, and community empowerment, CARB and its partners can meaningfully advance environmental justice and protect public health in the San Joaquin Valley and across the state.

8 References

1. U.S. EPA. EPA Identifies Noise Levels Affecting Health and Welfare. April 2, 1974. <https://www.epa.gov/archive/epa/aboutepa/epa-identifies-noise-levels-affecting-health-and-welfare.html>
2. Koolik,L., Alvarado,A., Budahn,A., Plummer,L., Marshall,D. J., Apte,S. J. PM2.5 exposure disparities persist despite strict vehicle emissions controls in California. *Science Advances*. 2024;Vol. 10,No.37. doi:10.1126/sciadv.adn8544
3. de Leon K. *California Senate Bill 535. Global Warming Solutions Act: Greenhouse Gas Reduction Fund.*; 2012. https://leginfo.ca.gov/faces/billNavClient.xhtml?bill_id=201120120SB53
4. OEHHA. SB 535 Disadvantaged Communities. [https://oehha.ca.gov/calenviroscreen/sb535#:~:text=Senate%20Bill%20535%20\(De%20Le%20C3%B3n,projects%20located%20within%20those%20communities](https://oehha.ca.gov/calenviroscreen/sb535#:~:text=Senate%20Bill%20535%20(De%20Le%20C3%B3n,projects%20located%20within%20those%20communities)
5. San Joaquin Valley Air Pollution Control District. Valley Air District. Homepage. June 17, 2025. <https://ww2.valleyair.org/>
6. Hammer MS, Swinburn TK, Neitzel RL. Environmental Noise Pollution in the United States: Developing an Effective Public Health Response. *Environmental Health Perspectives*. 2014;122(2):115-119. doi:10.1289/ehp.1307272
7. U.S. EPA. National Ambient Air Quality Standards (NAAQS) Table. EPA. December 16, 2024. <https://www.epa.gov/criteria-air-pollutants/naaqs-table>
8. California Air Resource Board. *Indoor Air Pollution in California.*; 2005. <https://ww2.arb.ca.gov/sites/default/files/classic/research/apr/reports/l3041.pdf>
9. Bennett JE, Tamura-Wicks H, Parks RM, et al. Particulate matter air pollution and national and county life expectancy loss in the USA: A spatiotemporal analysis. *PLoS Med*. 2019;16(7):e1002856. doi:10.1371/journal.pmed.1002856
10. Perlin SA, Wong D, Sexton K. Residential Proximity to Industrial Sources of Air Pollution: Interrelationships among Race, Poverty, and Age. *Journal of the Air & Waste Management Association*. 2001;51(3):406-421. doi:10.1080/10473289.2001.10464271
11. Hernandez M, Collins TW, Grineski SE. Immigration, mobility, and environmental injustice: A comparative study of Hispanic people's residential decision-making and exposure to hazardous air pollutants in Greater Houston, Texas. *Geoforum*. 2015;60:83-94. doi:10.1016/j.geoforum.2015.01.013
12. Wang SW, Majeed MA, Chu PL, Lin HC. Characterizing relationships between personal exposures to VOCs and socioeconomic, demographic, behavioral variables. *Atmospheric Environment*. 2009;43(14):2296-2302. doi:10.1016/j.atmosenv.2009.01.032

13. Woo B, Kravitz-Wirtz N, Sass V, Crowder K, Teixeira S, Takeuchi DT. Residential Segregation and Racial/Ethnic Disparities in Ambient Air Pollution. *Race Soc Probl.* 2019;11(1):60-67. doi:10.1007/s12552-018-9254-0
14. Molitor J, Su JG, Molitor NT, et al. Identifying Vulnerable Populations through an Examination of the Association Between Multipollutant Profiles and Poverty. *Environ Sci Technol.* 2011;45(18):7754-7760. doi:10.1021/es104017x
15. Winter PL, Padgett PE, Milburn LAS, Li W. Neighborhood Parks and Recreationists' Exposure to Ozone: A Comparison of Disadvantaged and Affluent Communities in Los Angeles, California. *Environmental Management.* 2019;63(3):379-395. doi:10.1007/s00267-019-01140-3
16. Stewart IT, Clow GL, Graham AE, Bacon CM. Disparate air quality impacts from roadway emissions on schools in Santa Clara County (CA). *Applied Geography.* 2020;125:102354. doi:10.1016/j.apgeog.2020.102354
17. Yanosky J, Schwartz J, Suh H. Associations Between Measures of Socioeconomic Position and Chronic Nitrogen Dioxide Exposure in Worcester, Massachusetts. *Journal of toxicology and environmental health Part A.* 2008;71:1593-1602. doi:10.1080/15287390802414307
18. Belanger K, Gent JF, Triche EW, Bracken MB, Leaderer BP. Association of Indoor Nitrogen Dioxide Exposure with Respiratory Symptoms in Children with Asthma. *Am J Respir Crit Care Med.* 2006;173(3):297-303. doi:10.1164/rccm.200408-1123OC
19. California Air Resource Board. *Air Quality and Emissions Data Almanac.*; 2013. <https://ww2.arb.ca.gov/our-work/programs/resource-center/technical-assistance/air-quality-and-emissions-data/almanac>
20. California Air Resources Board. *Research Synthesis #17-01 "How California Is Giving Soot the Boot!"*; 2017. <https://ww2.arb.ca.gov/resources/documents/research-synthesis-17-01-how-california-giving-soot-boot>
21. California Air Resource Board. *Community Air Protection Program Blueprint 2.0.*; 2024. <https://ww2.arb.ca.gov/capp/mdc/bp2/community-air-protection-program-blueprint-20#:~:text=California%20Air%20Resources%20Board,-Main%20navigation&text=Approved%20in%20October%202023%2C%20the,impacted%20by%20poor%20air%20quality.>
22. Bouvier R. Distribution of income and toxic emissions in Maine, United States: Inequality in two dimensions. *Ecological Economics.* 2014;102:39-47. doi:10.1016/j.ecolecon.2014.03.005
23. Wu X (May), Fan Z (Tina), Ohman-Strickland P. Time-Location Patterns of a Population Living in an Air Pollution Hotspot. *Journal of Environmental and Public Health.* 2010;2010:e625461. doi:10.1155/2010/625461
24. Mallah MA, Changxing L, Mallah MA, et al. Polycyclic aromatic hydrocarbon and its effects on human health: An overview. *Chemosphere.* 2022;296:133948. doi:10.1016/j.chemosphere.2022.133948

25. Jin Z, Sun W, Wang G. Association between polycyclic aromatic hydrocarbons exposure and current asthma: a population-based study. *BMC Public Health*. 2025;25(1):53. doi:10.1186/s12889-024-21015-5
26. Noth EM, Lurmann F, Perrino C, Vaughn D, Minor HA, Hammond SK. Decrease in ambient polycyclic aromatic hydrocarbon concentrations in California's San Joaquin Valley 2000–2019. *Atmospheric Environment*. 2020;242:117818. doi:10.1016/j.atmosenv.2020.117818
27. Propper R, Wong P, Bui S, et al. Ambient and Emission Trends of Toxic Air Contaminants in California. *Environ Sci Technol*. 2015;49(19):11329-11339. doi:10.1021/acs.est.5b02766
28. World-leading economy and climate solutions: California's emissions drop in 2023, driven by clean transportation. November 6, 2025. <https://www.gov.ca.gov/2025/11/06/world-leading-economy-and-climate-solutions-californias-emissions-drop-in-2023-driven-by-clean-transportation/>
29. Do K, Yu H, Velasquez J, Grell-Brisk M, Smith H, Ivey CE. A data-driven approach for characterizing community scale air pollution exposure disparities in inland Southern California. *Journal of Aerosol Science*. 2021;152:105704. doi:10.1016/j.jaerosci.2020.105704
30. Hazlehurst M, Nurius P, Hajat A. Individual and Neighborhood Stressors, Air Pollution and Cardiovascular Disease. *IJERPH*. 2018;15(3):472. doi:10.3390/ijerph15030472
31. Hill TD, Jorgenson AK, Ore P, Balistreri KS, Clark B. Air quality and life expectancy in the United States: An analysis of the moderating effect of income inequality. *SSM - Population Health*. 2019;7:100346. doi:10.1016/j.ssmph.2018.100346
32. Brochu PJ, Yanosky JD, Paciorek CJ, et al. Particulate Air Pollution and Socioeconomic Position in Rural and Urban Areas of the Northeastern United States. *Am J Public Health*. 2011;101(S1):S224-S230. doi:10.2105/AJPH.2011.300232
33. Chi GC, Hajat A, Bird CE, et al. Individual and Neighborhood Socioeconomic Status and the Association between Air Pollution and Cardiovascular Disease. *Environmental Health Perspectives*. 2016;124(12):1840-1847. doi:10.1289/EHP199
34. Harley KG, Calderon L, Nolan JES, et al. Changes in Latina Women's Exposure to Cleaning Chemicals Associated with Switching from Conventional to "Green" Household Cleaning Products: The LUCIR Intervention Study. *Environ Health Perspect*. 2021;129(9):097001. doi:10.1289/EHP8831
35. Harley KG, Kogut K, Madrigal DS, et al. Reducing Phthalate, Paraben, and Phenol Exposure from Personal Care Products in Adolescent Girls: Findings from the HERMOSA Intervention Study. *Environ Health Perspect*. 2016;124(10):1600-1607. doi:10.1289/ehp.1510514
36. Belanger K, Gent JF, Triche EW, Bracken MB, Leaderer BP. Association of Indoor Nitrogen Dioxide Exposure with Respiratory Symptoms in Children with Asthma. *Am J Respir Crit Care Med*. 2006;173(3):297-303. doi:10.1164/rccm.200408-1123OC

37. Chiu YHM, Coull BA, Sternthal MJ, et al. Effects of prenatal community violence and ambient air pollution on childhood wheeze in an urban population. *Journal of Allergy and Clinical Immunology*. 2014;133(3):713-722.e4. doi:10.1016/j.jaci.2013.09.023
38. Ailshire J, Karraker A, Clarke P. Neighborhood social stressors, fine particulate matter air pollution, and cognitive function among older U.S. adults. *Social Science & Medicine*. 2017;172:56-63. doi:10.1016/j.socscimed.2016.11.019
39. Roswall N, Raaschou-Nielsen O, Ketznel M, et al. Long-term residential road traffic noise and NO₂ exposure in relation to risk of incident myocardial infarction - A Danish cohort study. *Environmental research*. 2017;156:80-. doi:10.1016/j.envres.2017.03.019
40. World Health Organization. *Burden of Disease from Environmental Noise: Quantification of Healthy Life Years Lost in Europe*. WHO Regional Office for Europe.; 2011. Accessed August 18, 2025. <https://www.who.int/publications-detail-redirect/burden-of-disease-from-environmental-noise-quantification-of-healthy-life-years-lost-in-europe>
41. Hänninen O, Knol AB, Jantunen M, et al. Environmental Burden of Disease in Europe: Assessing Nine Risk Factors in Six Countries. *Environmental Health Perspectives*. 2014;122(5):439-446. doi:10.1289/ehp.1206154
42. Casey JA, Morello-Frosch R, Mennitt DJ, Frstrup K, Ogburn EL, James P. Race/Ethnicity, Socioeconomic Status, Residential Segregation, and Spatial Variation in Noise Exposure in the Contiguous United States. *Environ Health Perspect*. 2017;125(7):077017. doi:10.1289/EHP898
43. World Health Organization. Environmental Noise Guidelines for the European Region (2018). Accessed February 18, 2022. <https://www.euro.who.int/en/health-topics/environment-and-health/noise/publications/2018/environmental-noise-guidelines-for-the-european-region-2018>
44. US Department of Transport. National Transportation Noise Map. <https://maps.dot.gov/BTS/NationalTransportationNoiseMap/>
45. Paul Donavan. *Design and Acoustic Evaluation of Optimal Sinusoidal Muble Strips Versys Conventional Ground-in Rumble Strips*. Caltrans; 2018. <https://dot.ca.gov/programs/environmental-analysis/noise-vibration/rumble-strips>
46. California Department of Transport (CalTrans). Caltrans Quieter Pavements. Quieter Pavements. 2025. <https://dot.ca.gov/programs/maintenance/pavement/concrete-pavement-and-pavement-foundations/quieter-pavement>
47. California Department of Transport (CalTrans). Sound Wall FAQ's. Sound Wall FAQ's. 2025. <https://dot.ca.gov/caltrans-near-me/district-11/current-projects/i805sr15-transitionly-managedlanes/soundwallsfaq>
48. Yu-Kai H, Mitchell UA, Conroy LM, Jones RM. Community daytime noise pollution and socioeconomic differences in Chicago, IL. *PLoS One*. 2021;16(8):e0254762. doi:http://dx.doi.org/10.1371/journal.pone.0254762

49. Weuve J, D'Souza J, Beck T, et al. Long-term community noise exposure in relation to dementia, cognition, and cognitive decline in older adults. *Alzheimer's & dementia*. 2021;17(3):525-533. doi:10.1002/alz.12191
50. Nega TH, Chihara L, Smith K, Jayaraman M. Traffic Noise and Inequality in the Twin Cities, Minnesota. *Human and Ecological Risk Assessment: An International Journal*. 2013;19(3):601-619. doi:10.1080/10807039.2012.691409
51. Simon MC, Hart JE, Levy JI, et al. Sociodemographic Patterns of Exposure to Civil Aircraft Noise in the United States. *Environmental Health Perspectives*. 2022;130(2):027009. doi:10.1289/EHP9307
52. Huang CH, Seto E. Estimates of population highly annoyed from transportation noise in the United States: An unfair share of the burden by race and ethnicity. *Environmental Impact Assessment Review*. 2024;104:107338. doi:10.1016/j.eiar.2023.107338
53. Dale LM, Goudreau S, Perron S, Ragetti MS, Hatzopoulou M, Smargiassi A. Socioeconomic status and environmental noise exposure in Montreal, Canada. *BMC public health*. 2015;15:205. doi:http://dx.doi.org/10.1186/s12889-015-1571-2
54. Lam K che, Chan PK. Socio-economic status and inequalities in exposure to transportation noise in Hong Kong. *Open environmental sciences*. 2008;2(1):107-113. doi:10.2174/1876325100802010107
55. U.S. EPA. Clean Air Act Title IV-Noise Pollution. Clean Air Act Title IV-Noise Pollution. 1990. <https://www.epa.gov/clean-air-act-overview/clean-air-act-title-iv-noise-pollution>
56. Argys LM, Averett SL, Yang M. Residential noise exposure and health: Evidence from aviation noise and birth outcomes. *Journal of Environmental Economics and Management*. 2020;103:102343. doi:10.1016/j.jeem.2020.102343
57. Nieuwenhuijsen M, Ristovska G, Dadvand P. WHO Environmental Noise Guidelines for the European Region: A Systematic Review on Environmental Noise and Adverse Birth Outcomes. *IJERPH*. 2017;14(10):1252. doi:10.3390/ijerph14101252
58. Wang Z, Qian R, Xiang W, et al. Association between noise exposure during pregnancy and pregnancy complications: A meta-analysis. *Front Psychol*. 2022;13:1026996. doi:10.3389/fpsyg.2022.1026996
59. Ristovska G, Laszlo H, Hansell A. Reproductive Outcomes Associated with Noise Exposure — A Systematic Review of the Literature. *IJERPH*. 2014;11(8):7931-7952. doi:10.3390/ijerph110807931
60. Dzhambov AM, Lercher P. Road Traffic Noise Exposure and Birth Outcomes: An Updated Systematic Review and Meta-Analysis. *IJERPH*. 2019;16(14):2522. doi:10.3390/ijerph16142522
61. Graafland N, Essers E, Posthumus A, et al. Exposure to outdoor residential noise during pregnancy, embryonic size, fetal growth, and birth outcomes. *Environment International*. 2023;171:107730. doi:10.1016/j.envint.2023.107730

62. Wallas A, Ekström S, Bergström A, et al. Traffic noise exposure in relation to adverse birth outcomes and body mass between birth and adolescence. *Environmental Research*. 2019;169:362-367. doi:10.1016/j.envres.2018.11.039
63. Vincens N, Persson Waye K. Occupational and environmental noise exposure during pregnancy and rare health outcomes of offspring: a scoping review focusing on congenital anomalies and perinatal mortality. *Reviews on Environmental Health*. 2023;38(3):423-438. doi:10.1515/reveh-2021-0166
64. Dohmen M, Braat-Eggen E, Kemperman A, Hornikx M. The Effects of Noise on Cognitive Performance and Helplessness in Childhood: A Review. *IJERPH*. 2022;20(1):288. doi:10.3390/ijerph20010288
65. Gheller F, Spicciarelli G, Scimemi P, Arfé B. The Effects of Noise on Children's Cognitive Performance: A Systematic Review. *Environment and Behavior*. 2024;55(8-10):698-734. doi:10.1177/00139165241245823
66. Jafari MJ, Khosrowabadi R, Khodakarim S, Mohammadian F. The Effect of Noise Exposure on Cognitive Performance and Brain Activity Patterns. *Open Access Maced J Med Sci*. 2019;7(17):2924-2931. doi:10.3889/oamjms.2019.742
67. López-Vicente M, Kusters M, Binter AC, et al. Long-Term Exposure to Traffic-Related Air Pollution and Noise and Dynamic Brain Connectivity across Adolescence. *Environ Health Perspect*. 2025;133(5). doi:10.1289/ehp14525
68. Liang P, Li J, Li Z, et al. Effect of low-frequency noise exposure on cognitive function: a systematic review and meta-analysis. *BMC Public Health*. 2024;24(1). doi:10.1186/s12889-023-17593-5
69. Thompson, McKenna, Castorina, Rosemary, Chen, Wenhao, Moore, David, Peerless, Kyle, Hurley, Susan. Effectiveness of Air Filtration in Reducing PM2.5 Exposures at a School in a Community Heavily Impacted by Air Pollution. *Atmosphere*. 2024;15(8):901. doi:https://doi.org/10.3390/atmos15080901
70. Meng L, Zhang Y, Zhang S, et al. Chronic Noise Exposure and Risk of Dementia: A Systematic Review and Dose-Response Meta-Analysis. *Front Public Health*. 2022;10. doi:10.3389/fpubh.2022.832881
71. Belojević G. Noise and Alzheimer's disease. *Ann Nursing*. 2023;1(4):10-33. doi:10.58424/annnurs.y9e.cdx.n73
72. Janus SIM, Kusters J, Van Den Bosch KA, Andringa TC, Zuidema SU, Luijendijk HJ. Sounds in nursing homes and their effect on health in dementia: a systematic review. *International Psychogeriatrics*. 2021;33(6):627-644. doi:10.1017/s1041610220000952
73. Ritz B, Yu Y. Noise exposure and dementia: a rising concern in ageing populations. *BMJ*. Published online September 8, 2021:n2120. doi:10.1136/bmj.n2120
74. Gong X, Fenech B, Blackmore C, et al. Association between Noise Annoyance and Mental Health Outcomes: A Systematic Review and Meta-Analysis. *IJERPH*. 2022;19(5):2696. doi:10.3390/ijerph19052696

75. Guha M. Noise pollution and mental health. *Journal of Mental Health*. 2022;31(5):605-606. doi:10.1080/09638237.2022.2118694
76. Hahad O, Kuntic M, Al-Kindi S, et al. Noise and mental health: evidence, mechanisms, and consequences. *J Expo Sci Environ Epidemiol*. 2025;35(1):16-23. doi:10.1038/s41370-024-00642-5
77. Newbury JB, Heron J, Kirkbride JB, et al. Association of early-life exposure to air and noise pollution with youth mental health: findings from the ALSPAC cohort. *The Lancet Planetary Health*. 2024;8:S11. doi:10.1016/S2542-5196(24)00076-7
78. Chan TC, Wu BS, Lee YT, Lee PH. Effects of personal noise exposure, sleep quality, and burnout on quality of life: An online participation cohort study in Taiwan. *Science of The Total Environment*. 2024;915:169985. doi:10.1016/j.scitotenv.2024.169985
79. Halperin D. Environmental noise and sleep disturbances: A threat to health? *Sleep Science*. 2014;7(4):209-212. doi:10.1016/j.slsci.2014.11.003
80. Rudolph KE, Shev A, Paksarian D, et al. Environmental noise and sleep and mental health outcomes in a nationally representative sample of urban US adolescents. *Environmental Epidemiology*. 2019;3(4):e056. doi:10.1097/ee9.000000000000056
81. Smith MG, Cordoza M, Basner M. Environmental Noise and Effects on Sleep: An Update to the WHO Systematic Review and Meta-Analysis. *Environ Health Perspect*. 2022;130(7):076001. doi:10.1289/EHP10197
82. Yamagami Y, Obayashi K, Tai Y, Saeki K. Association between indoor noise level at night and objective/subjective sleep quality in the older population: a cross-sectional study of the HEIJO-KYO cohort. *SLEEP*. 2023;46(5):zsac197. doi:10.1093/sleep/zsac197
83. Bozigar M, Huang T, Redline S, et al. Associations between Aircraft Noise Exposure and Self-Reported Sleep Duration and Quality in the United States-Based Prospective Nurses' Health Study Cohort. *Environ Health Perspect*. 2023;131(4):047010. doi:10.1289/EHP10959
84. Foraster M, Eze IC, Vienneau D, et al. Long-term exposure to transportation noise and its association with adiposity markers and development of obesity. *Environment international*. 2018;121(Pt 1):879-889. doi:10.1016/j.envint.2018.09.057
85. Persson Å, Pyko A, Stucki L, et al. Long-term exposure to transportation noise and obesity: A pooled analysis of eleven Nordic cohorts. *Environmental Epidemiology*. 2024;8(4):e319. doi:10.1097/EE9.0000000000000319
86. Veber T, Pyko A, Carlsen HK, et al. Traffic noise in the bedroom in association with markers of obesity: a cross-sectional study and mediation analysis of the respiratory health in Northern Europe cohort. *BMC Public Health*. 2023;23(1):1246. doi:10.1186/s12889-023-16128-2
87. Shin S, Bai L, Oiamo TH, et al. Association Between Road Traffic Noise and Incidence of Diabetes Mellitus and Hypertension in Toronto, Canada: A Population-Based Cohort Study. *JAHA*. 2020;9(6):e013021. doi:10.1161/JAHA.119.013021

88. Basner M, Smith MG. The effects on sleep play a critical role in the long-term health consequences of noise exposure. *Sleep*. 2024;47(2):zsad314. doi:10.1093/sleep/zsad314
89. Clark C, Sbihi H, Tamburic L, Brauer M, Frank LD, Davies HW. Association of Long-Term Exposure to Transportation Noise and Traffic-Related Air Pollution with the Incidence of Diabetes: A Prospective Cohort Study. *Environmental health perspectives*. 2017;125(8):087025. doi:http://dx.doi.org/10.1289/EHP1279
90. Sørensen M, Hvidtfeldt UA, Poulsen AH, et al. Long-term exposure to transportation noise and risk of type 2 diabetes: A cohort study. *Environmental Research*. 2023;217:114795. doi:10.1016/j.envres.2022.114795
91. Vienneau D, Stafoggia M, Rodopoulou S, et al. Association between exposure to multiple air pollutants, transportation noise and cause-specific mortality in adults in Switzerland. *Environ Health*. 2023;22(1):29. doi:10.1186/s12940-023-00983-y
92. Cui B, Gai Z, She X, Wang R, Xi Z. Effects of chronic noise on glucose metabolism and gut microbiota–host inflammatory homeostasis in rats. *Sci Rep*. 2016;6(1):36693. doi:10.1038/srep36693
93. Letellier N, Yang JA, Cavallès C, et al. Aircraft and road traffic noise, insulin resistance, and diabetes: The role of neighborhood socioeconomic status in San Diego County. *Environmental Pollution*. 2023;335:122277. doi:10.1016/j.envpol.2023.122277
94. Kim J, Kwan MP. Assessment of sociodemographic disparities in environmental exposure might be erroneous due to neighborhood effect averaging: Implications for environmental inequality research. *Environmental Research*. 2021;195:110519. doi:10.1016/j.envres.2020.110519
95. Kacem I, Kahloul M, Maoua M, et al. Occupational Noise Exposure and Diabetes Risk. Houghton J, ed. *Journal of Environmental and Public Health*. 2021;2021:1-7. doi:10.1155/2021/1804616
96. Zare Sakhvidi MJ, Zare Sakhvidi F, Mehrparvar AH, Foraster M, Dadvand P. Association between noise exposure and diabetes: A systematic review and meta-analysis. *Environmental Research*. 2018;166:647-657. doi:10.1016/j.envres.2018.05.011
97. Babisch W. Updated exposure-response relationship between road traffic noise and coronary heart diseases: A meta-analysis. *Noise & Health*. 2014;16(68):1-9. doi:10.4103/1463-1741.127847
98. Li S, Fong DYT, Wong JYH, et al. Noise sensitivity associated with nonrestorative sleep in Chinese adults: a cross-sectional study. *BMC Public Health*. 2021;21(1):643. doi:10.1186/s12889-021-10667-2
99. Banerjee D, Das PP, Foujdar A. Association between road traffic noise and prevalence of coronary heart disease. *Environ Monit Assess*. 2014;186(5):2885-2893. doi:10.1007/s10661-013-3587-3

100. Correia AW, Peters JL, Levy JI, Melly S, Dominici F. Residential exposure to aircraft noise and hospital admissions for cardiovascular diseases: multi-airport retrospective study. *BMJ*. 2013;347(oct08 3):f5561-f5561. doi:10.1136/bmj.f5561
101. Davies H, Kamp I. Noise and cardiovascular disease: A review of the literature 2008-2011. *Noise Health*. 2012;14(61):287. doi:10.4103/1463-1741.104895
102. Roscoe C, Grady ST, Hart JE, et al. Association between Noise and Cardiovascular Disease in a Nationwide U.S. Prospective Cohort Study of Women Followed from 1988 to 2018. *Environ Health Perspect*. 2023;131(12):127005. doi:10.1289/EHP12906
103. Hahad O, Kröller-Schön S, Daiber A, Münzel T. The Cardiovascular Effects of Noise. *Deutsches Ärzteblatt international*. Published online April 5, 2019. doi:10.3238/arztebl.2019.0245
104. Münzel T, Sørensen M, Daiber A. Transportation noise pollution and cardiovascular disease. *Nat Rev Cardiol*. 2021;18(9):619-636. doi:10.1038/s41569-021-00532-5
105. Münzel T, Molitor M, Kuntic M, et al. Transportation Noise Pollution and Cardiovascular Health. *Circulation Research*. 2024;134(9):1113-1135. doi:10.1161/CIRCRESAHA.123.323584
106. Foraster M, Künzli N, Aguilera I, et al. High Blood Pressure and Long-Term Exposure to Indoor Noise and Air Pollution from Road Traffic. *Environ Health Perspect*. 2014;122(11):1193-1200. doi:10.1289/ehp.1307156
107. Lim YH, Jørgensen JT, So R, et al. Long-Term Exposure to Air Pollution, Road Traffic Noise, and Heart Failure Incidence: The Danish Nurse Cohort. *J Am Heart Assoc*. 2021;10(20):e021436. doi:10.1161/JAHA.121.021436
108. Sørensen M, Wendelboe Nielsen O, Sajadieh A, et al. Long-Term Exposure to Road Traffic Noise and Nitrogen Dioxide and Risk of Heart Failure: A Cohort Study. *Environmental health perspectives*. 2017;125(9):097021-097021. doi:10.1289/EHP1272
109. Smith RB, Fecht D, Gulliver J, et al. Impact of London's road traffic air and noise pollution on birth weight: retrospective population based cohort study. *BMJ*. 2017;359:j5299. doi:10.1136/bmj.j5299
110. MCERTS Performance Standards for Indicative Ambient Particulate Monitors. Published online August 4, 2017. <https://www.csagroup.org/wp-content/uploads/MC-20035001.pdf>
111. Singer B. C., Chan, W. R., Kim, Y.S., Offerman, F. J., & Walker, I. S. (2020). Indoor air quality in California homes with code-required in California homes with code-required mechanical ventilation. *Indoor air*, 30(5), 885-899. <https://doi.org/10.1111/ina.12676>
112. Zhao, H., Chan, W. R., Cohn, S., Delp, W. W., Walker, I. S., & Singer, B. C. (2021). Indoor air quality in new and renovated low-income apartments with mechanical ventilation and natural gas cooking in California. *Indoor air*, 31(3), 717-729. <https://doi.org/10.1111/ina.12764>

113. Less, B., Mullen N. A., Singer, B. C., Walker, I. S. (2015). Indoor air quality in 24 California residences designed as high-performance homes. *Science and Technology for the Built Environment*, 21(1), 14-24. <https://doi.org/10.1080/10789669.2014.961850114>.
Mullen, N. A., Li, J., Russel, M. K., Spears, M., Less, B. D., & Singer, B. C. (2016). Results of the California Healthy Homes Indoor Air Quality Study of 2011-2013: impact of natural gas appliances on air pollutant concentrations. *Indoor air*, 26(2), 231-245. <https://doi.org/10.1111/ina.12190>
115. San Joaquin Valley Air Pollution Control District. Ambient air quality standards & valley attainment status. Accessed January 23, 2026. <https://www.valleyair.org/air-quality-information/ambient-air-quality-standards-valley-attainmnet-status> 116. Caubel J, Cados T, Kirchstetter T. A New Black Carbon Sensor for Dense Air Quality Monitoring Networks. *Sensors*. 2018;18(3):738. doi:10.3390/s18030738
117. Caubel JJ, Cados TE, Preble CV, Kirchstetter TW. A Distributed Network of 100 Black Carbon Sensors for 100 Days of Air Quality Monitoring in West Oakland, California. *Environ Sci Technol*. 2019;53(13):7564-7573. doi:10.1021/acs.est.9b00282
118. U.S. EPA. *Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air Second Edition Compendium Method TO-17 Determination of Volatile Organic Compounds in Ambient Air Using Active Sampling Onto Sorbent Tubes.*; 1999. <https://www.epa.gov/sites/default/files/2019-11/documents/to-17r.pdf>
119. U.S. EPA. *Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air Second Edition Compendium Method TO-11A Determination of Formaldehyde in Ambient Air Using Adsorbent Cartridge Followed by High Performance Liquid Chromatography (HPLC) [Active Sampling Methodology].*; 1999. <https://www.epa.gov/sites/default/files/2019-11/documents/to-11ar.pdf>
120. Atmospheric Analysis and Consulting, Inc. Atmospheric Analysis and Consulting, Inc. Full Service Air Quality Laboratory.
121. Gundel LA, Lee VC, Mahanama KRR, Stevens RK, Daisey JM. Direct determination of the phase distributions of semivolatile polycyclic aromatic hydrocarbons using annual denuders. *Atmospheric Environment*. 1995;29(14):1719-1733. doi:10.1016/1352-2310(94)00366-s
122. Lee JJ, Huang KL, Yu YY, Chen MS. Laboratory retention of vapor-phase PAHs using XAD adsorbents. *Atmospheric Environment*. 2004;38:6185-6193. doi:doi:10.1016/j.atmosenv.2004.07.024
123. Noth EM, Hammond SK, Biging GS, Tager IB. A spatial-temporal regression model to predict daily outdoor residential PAH concentrations in an epidemiologic study in Fresno, CA. *Atmospheric Environment*. 2011;45(14):2394-2403. doi:10.1016/j.atmosenv.2011.02.014
124. Noth EM, Lurmann F, Northcross A, Perrino C, Vaughn D, Hammond SK. Spatial and temporal distribution of polycyclic aromatic hydrocarbons and elemental carbon in Bakersfield, California. *Air Quality Atmosphere and Health*. 2016;9(8):899-908. doi:10.1007/s11869-016-0399-y

125. Wagner J, Macher JM. Comparison of a Passive Aerosol Sampler to Size-Selective Pump Samplers in Indoor Environments. *AIHA Journal*. 2003;64(5):630-639. doi:10.1080/15428110308984856
126. Wagner J, Leith D. Passive Aerosol Sampler. Part I: Principle of Operation. *Aerosol Science and Technology*. 2001;34(2):186-192. doi:10.1080/027868201300034808
127. Castillo MD, Wagner J, Casuccio GS, et al. Field testing a low-cost passive aerosol sampler for long-term measurement of ambient PM_{2.5} concentrations and particle composition. *Atmospheric Environment*. 2019;216:116905. doi:10.1016/j.atmosenv.2019.116905
128. Peters JM, Colavin A, Shi H, et al. A Comprehensive, CRISPR-based Functional Analysis of Essential Genes in Bacteria. *Cell*. 2016;165(6):1493-1506. doi:10.1016/j.cell.2016.05.003
129. Ott DK, Peters TM. A Shelter to Protect a Passive Sampler for Coarse Particulate Matter, PM_{10-2.5}. *Aerosol Science and Technology*. 2008;42(4):299-309. doi:10.1080/02786820802054236
130. Arashiro M, Leith D. Precision of PM measurements with the UNC passive aerosol sampler. 2013;57:181-184. doi:https://doi.org/10.1016/j.jaerosci.2012.09.001
131. Tracking California. Tracking California Traffic Tool. Tracking California informing action for healthier communities. <https://ext.trackingcalifornia.org/traffic/#/map/explore>
132. California Department of Transport (CalTrans). *California Public Road Data: Statistical Information Derived from the Highway Performance Monitoring System.*; 2023. <https://dot.ca.gov/-/media/dot-media/programs/research-innovation-system-information/documents/hpms2023-prd-final.pdf>
133. OEHHA. Proposition 65 No Significant Risk Levels (NSRLs) and Maximum Allowable Dose Levels (MADLs). Proposition 65 No Significant Risk Levels (NSRLs) and Maximum Allowable Dose Levels (MADLs). October 27, 2023. Accessed July 10, 2025. <https://oehha.ca.gov/proposition-65/general-info/proposition-65-no-significant-risk-levels-nsrls-and-maximum-allowable-dose-levels-madls>
134. U.S. EPA. U.S. EPA. Highlights of the Exposure Factors Handbook (Final Report). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-10/030, 2011. Published online 2011. <https://assessments.epa.gov/risk/document/&deid=221023>
135. Weschler CJ. Ozone in Indoor Environments: Concentration and Chemistry: Ozone in Indoor Environments. *Indoor Air*. 2000;10(4):269-288. doi:10.1034/j.1600-0668.2000.010004269.x
136. WHO *Global Air Quality Guidelines: Particulate Matter (PM_{2.5} and PM₁₀), Ozone, Nitrogen Dioxide, Sulfur Dioxide and Carbon Monoxide*. 1st ed. World Health Organization; 2021.

137. Cisneros R, Schweizer D, Amiri M, Zarate-Gonzalez G, Gharibi H. Long-Term Fine Particulate Matter (PM_{2.5}) Trends and Exposure Patterns in the San Joaquin Valley of California. *Atmosphere*. 2025;16(6):721. doi:10.3390/atmos16060721
138. OEHHA. *OEHHA Acute, 8-Hour and Chronic Reference Exposure Level (REL) Summary*.; 2023. <https://oehha.ca.gov/air/general-info/oehha-acute-8-hour-and-chronic-reference-exposure-level-rel-summary>
139. U.S. EPA. Xylenes; CASRN 1330-20-7. Published online February 21, 2003. https://iris.epa.gov/static/pdfs/0270_summary.pdf
140. U.S. EPA. Benzene; CASRN 71-43-2. Published online April 17, 2003. https://iris.epa.gov/static/pdfs/0276_summary.pdf
141. U.S. EPA. Ethylbenzene; CASRN 100-41-4. Published online March 1, 1991. https://iris.epa.gov/static/pdfs/0051_summary.pdf
142. U.S. EPA. Toluene; CASRN 108-88-3. Published online September 23, 2005. https://iris.epa.gov/static/pdfs/0118_summary.pdf
143. US EPA. IRIS Toxicological Review of Formaldehyde (Inhalation). <https://www.epa.gov/iris>
144. Weinstein ND. Individual differences in reactions to noise: a longitudinal study in a college dormitory. *J Appl Psychol*. 1978;63(4):458-466.
145. EPA. Naphthalene; CASRN 91-20-3. Published online September 17, 1998. https://iris.epa.gov/static/pdfs/0436_summary.pdf
146. Bradman, A., Gaspar, F., Castorina, R., Williams, J., Hoang, T., Jenkins, P. L., McKone, T. E., & Maddalena, R. (2017). Formaldehyde and acetaldehyde exposure and risk characterization in California early childhood education environments. *Indoor air*, 27(1), 104–113. <https://doi.org/10.1111/ina.12283>
147. EPA. Fluorene; CASRN 86-73-7. Published online December 1, 1990. https://iris.epa.gov/static/pdfs/0435_summary.pdf
148. EPA. Phenanthrene; CASRN 85-01-8. Published online December 1, 1990. https://iris.epa.gov/static/pdfs/0459_summary.pdf
149. Pyrene; CASRN 129-00-0. Published online September 1, 1990. https://iris.epa.gov/static/pdfs/0445_summary.pdf
150. Dodson RE, Franklin ET, Zota AR, et al. Formaldehyde and Formaldehyde Releasing Preservatives in Personal Care Products Used by Black Women and Latinas. *Environ Sci Technol Lett*. 2025;12(9):1205-1210. doi:10.1021/acs.estlett.5c00242
151. Johnson PI, Favela K, Jarin J, et al. Chemicals of concern in personal care products used by women of color in three communities of California. *J Expo Sci Environ Epidemiol*. 2022;32(6):864-876. doi:10.1038/s41370-022-00485-y

152. California Air Resource Board. Low-Cost Sensors for Healthier Indoor Air Quality in Impacted Communities. March 2025. Accessed November 14, 2025.
<https://ww2.arb.ca.gov/low-cost-sensors-healthier-indoor-air-quality-impacted-communities>

9. Glossary of Terms, Abbreviations, and Symbols

Acronym	Description
#	Number
AAC Lab	Atmospheric Analysis and Consulting, Inc.
AB 617	California Assembly Bill 617
ABCD	Aerosol Black Carbon Detector
AC	Air Conditioning
ACE	Adverse Childhood Experiences
ACH	Air Changes per Hour
aREL	Acute Reference Exposure Level (OEHHA)
ATN	Attenuation
BC	Black Carbon
BEAR	Berkeley Exposure Assessment Research Laboratory
BMI	Body Mass Index
BTEX	Benzene, Toluene, Ethylbenzene, and Xylenes
CA-4	California State Route 4
CAPP	Community Air Protection Program
CARB	California Air Resources Board
CAS	Chemical Abstracts Service
CCAC	Central California Asthma Collaborative
cc/m	Cubic Centimeter per meter
CDPH	California Department of Public Health
CERCH	Center for Environmental Research and Community Health, UC Berkeley School of Public Health
CES	CalEnviroScreen
CH ₂ O	Formaldehyde
CHD	Coronary Heart Disease
CI	Cumulative Impact
CO	Carbon Monoxide
cREL	Chronic Reference Exposure Level (OEHHA)
CVD	Cardiovascular Disease
DAC	Disadvantaged Community
DALY	Disability-Adjusted Life Year
dB	Decibel
dBA	A-weighted decibels; unit to measure sound levels
DF	Detection Frequency
DNPH	2,4-Dinitrophenylhydrazine
DVMT	Daily Vehicle Miles Traveled

Acronym	Description
EDS	Energy Dispersive X-ray Spectroscopy
EPA	U.S. Environmental Protection Agency
EtBz	Ethylbenzene
FLU	Fluorene
ft	Foot
GC/MS	Gas chromatography / Mass spectrometry
HAP	Hazardous Air Pollutant
HDL	High-Density Lipoprotein
HEPA	High Efficiency Particulate Air
HPLC	High Pressure Liquid Chromatography
HPMS	Highway Performance Management System
HQ	Hazard Quotient
HR	Hazard Ratio
HVAC	Heating, ventilation and air conditioning
IAQ	Indoor Air Quality
IHF	Incident Heart Failure
I/O	Indoor-to-Outdoor Ratio
IRB	Institutional Review Board
km	Kilometer
L	Liter
lbs	Pounds
LBW	Low Birth Weight
L/min	Liters per minute
LMR	Little Manila Rising
LOD	Limit of Detection
LQ	Limit of Quantitation
m	Meter
Max	Maximum
MDL	Method Detection Limit
MERV	Minimum Efficiency Reporting Value
Min	Minimum
mL/min	Milliliters per minute
mm	Millimeter
µg/m ³	Micrograms per cubic meter
µg min	Microgram minute
µg/day	Micrograms per day
µm	Micrometer
NAAQS	National Ambient Air Quality Standard
NAP	Naphthalene
ng	Nanogram

Acronym	Description
ng/m ³	Nanograms per cubic meter
NHANES	National Health and Nutrition Examination Survey
nm	Nanometer
NO ₂	Nitrogen dioxide
NSRL	No Significant Risk Level (OEHHA)
O ₃	Ozone
OEHHA	California Office of Environmental Health Hazard Assessment
ONAC	Office of Noise Abatement and Control
OR	Odds Ratio
PAH	Polyaromatic Hydrocarbon
Pb	Lead
PEL	Permissible Exposure Limit
PHE	Phenanthrene
ppb	Parts per billion
ppm	Parts per million
PM	Particulate Matter
PM _{2.5}	Particulate Matter < 2.5 µm diameter
PM ₁₀	Particulate Matter < 10 µm diameter
PM _{10-2.5}	Particulate Matter between 2.5 and 10 µm diameter
PYR	Pyrene
QA/QC	Quality Assurance and Quality Control
REL	Reference Exposure Level (OEHHA)
RfC	Reference Concentration (U.S. EPA)
RHINE	Respiratory Health in Northern Europe
rho	Spearman Rho test statistic
SB 535	California Senate Bill 535
SD	Standard Deviation
SEM	Scanning Electron Microscope
SGA	Small for Gestational Age
SIC	Standard Industrial Classification
SJV	San Joaquin Valley
SO ₂	Sulfur dioxide
SPHERE	San Joaquin Valley Household Environmental Research and Exposure Study
SR-99	State Route 99
T2DM	Type 2 Diabetes Mellitus
TRAP	Traffic-Related Air Pollution
TVOC	Total Volatile Organic Compounds
UC	University of California

Acronym	Description
U.S. EPA	United States Environmental Protection Agency
VOC	Volatile Organic Compound
WHO	World Health Organization