Title

Traffic-related Noise Exposure and Late-life Dementia and Cognitive Impairment in Mexican-Americans.

Journal

Epidemiology, 31(6)

ISSN

1044-3983

Authors

Yu, Yu
Mayeda, Elizabeth Rose
Paul, Kimberly C
et al.

Publication Date

2020-11-01

DOI

10.1097/ede.0000000000001249

Peer reviewed

HHS Public Access

Author manuscript

Epidemiology. Author manuscript; available in PMC 2021 November 27.

Published in final edited form as:

Epidemiology. 2020 November 01; 31(6): 771–778. doi:10.1097/EDE.0000000000001249.

Traffic-Related Noise Exposure and Late-life Dementia and Cognitive Impairment in Mexican–Americans

Yu Yu[a], **Elizabeth Rose Mayeda**[a], **Kimberly C. Paul**[a], **Eunice Lee**[b], **Michael Jerrett**[c], **Jason Su**[b], **Jun Wu**[d], **I-Fan Shih**[a], **Mary Haan**[e], **Beate Ritz**[a,c,f]

[a]Department of Epidemiology, UCLA Fielding School of Public Health, Los Angeles, California, USA

[b]Division of Environmental Health Science, UCB School of Public Health, Berkeley, California, USA

[c]Department of Environmental Health Science, UCLA Fielding School of Public Health, Los Angeles, California, USA

[d]Program in Public Health, Susan and Henry Samueli College of Health Sciences, UCI, Irvine, USA

[e]Department of Epidemiology & Biostatistics, UCSF, San Francisco, California, USA

[f]Department of Neurology, David Geffen School of Medicine, Los Angeles, California, USA

Abstract

Background—Recently, it has been suggested that environmental exposures from traffic sources including noise may play a role in cognitive impairment in the elderly. The objective of the study was to investigate the association between local traffic-related noise pollution and incident dementia or cognitive impairment without dementia (CIND) during a 10-year follow-up period.

Methods—1,612 Mexican–American participants from the Sacramento Area Latino Study on Aging (SALSA) were followed every 12–15 months via home visits from 1998 to 2007. We used the SoundPLAN software package to estimate noise originating from local traffic with the input of Annual Average Daily Traffic (AADT) data from Metropolitan Planning Organizations (MPO) based on geocoded residential addresses at baseline (1998–99). We estimated the risks of incident dementia or CIND from 24-hour and nighttime noise exposure using Cox proportional hazard models.

Results—During the follow-up, we identified 159 incident dementia or CIND cases in total. Per 11.6 dB (interquartile range width, IQRw) increase in 24-hour noise, the hazard of developing dementia or CIND increased (HR = 1.2 [1.0, 1.5]) during follow-up; estimates were slightly

Corresponding author information: Address correspondence to Beate Ritz, Department of Epidemiology, Fielding School of Public Health, University of California at Los Angeles, Los Angeles, California, USA, 650 Charles E. Young Drive South, Los Angeles, CA 90095-1772, USA; Tel (310) 206 7458; Fax (310) 206 6039; britz@ucla.edu.

Conflicts of Interest: None declared.

Data Access: The data for this cohort are available on the server of Inter-university Consortium for Political and Social Research (ICPSR) at University of Michigan and the noise data will be available upon request to the authors. The analytical code used was standard SAS code (e.g., data steps, proc phreg).

lower (HR = 1.2 [0.95, 1.5]) when adjusting for modeled local air pollution exposure from traffic sources. Overall, the risk of dementia/CIND was elevated when 24-hour and nighttime noise were higher than 75dB and 65dB respectively.

Conclusions—In our study, traffic-related noise exposure was associated with increased risk of dementia or CIND in elderly Mexican–Americans. Future studies taking into account other noise sources and occupational noise exposure before retirement are needed.

Keywords

Noise; traffic-related; dementia; cognitive impairment; Mexican–Americans

INTRODUCTION

Cognitive impairment is a major concern for older adults due to its relationship with morbidity and mortality. It also reduces health related quality of life and increases caregiver burden. In societies with increasing life expectancy and aging populations, preventing these outcomes becomes ever more urgent[1]. According to the Alzheimer's Association in 2018, ~5.7 million people are living with dementia in the United States (US), and by 2060 it projects that the prevalence of Alzheimer disease (AD) will reach 13.9 million[2]. Possible or established risk factors for cognitive impairment include age, family history, apolipoprotein E (APOE) ε4, cardiovascular disease, diabetes, hypertension, and lifestyle factors such as smoking and alcohol consumption[3].

Recently, studies have indicated that environmental exposures including air pollution from traffic sources are consistently associated not only with cardiovascular and respiratory diseases and all-cause mortality[4,5], but also with cognitive impairment[6]. Most epidemiologic studies focused on investigating the association between air pollution and cognition function; however, the role of noise in relation to cognitive impairment is far less studied. Those studies that examined the influence of noise exposures mostly measured short-term effects, or used cross-sectional or case–control study designs[6]. To our knowledge, there are to date three longitudinal studies, in Switzerland[7], France,[8] and England,[9] investigating traffic-related noise exposure but none of them has explored the influence of long-term noise exposure on the incidence of dementia or CIND in Mexican–Americans, a fast-growing and vulnerable segment of the US elderly population.

The objective of our study was to investigate whether residential-based traffic-related noise exposure at baseline increases the risks of dementia or CIND in older Mexican–Americans over 10 years of follow-up.

METHODS

All procedures described here were approved by the Institutional Review Boards of the University of California San Francisco, Los Angeles, and Davis, University of North Carolina, and the University of Michigan.

Study Population

We relied on data from the Sacramento Area Latino Study on Aging (SALSA), a prospective cohort study of older Mexican–Americans that was originally designed to evaluate the effects of metabolic and cardiovascular risk factors for dementia and cognitive decline. Participants were eligible if **(i)** they were 60 years of age or older, **(ii)** resided in the six counties of the California Sacramento Valley (Sacramento, Yolo, Sutter, Solano, Yuba, and Placer counties), and **(iii)** self-identified as Mexican (78.4%), Latino (6%), Hispanic (10.8%), Anglo (1.6%), Chicano (1.16%) or other (1.8%). Of those eligible and contacted, 83.5% agreed to be in the study. 1789 participants were recruited from 1998 to 1999 and interviewed at their homes; they were re-contacted every 12–15 months for up to seven study visits, ending in December 2007. Between home visits, a 10-minute phone call was made every 6 months to update contact information, health status, and change in medication information. The average annual attrition rate from mortality and loss to follow-up was 2.6% and 2.3% respectively. The average length of follow-up was 6.5 years and the maximum was 10 years[10]. All participants provided written informed consent. Those who (1) did not participate in the interview at baseline (n=3), (2) lived too far away from traffic sources to generate noise measures (n=3), (3) already had CIND or dementia at baseline (n=114), (4) did not have a follow-up visit (n=57) were excluded, leaving 1,612 participants in total for this analysis (Figure 1).

Outcome Measurement

We administered two cognitive screening tests - the Modified Mini–Mental State Examination (3MSE) and a delayed word recall trial from the Spanish English Verbal Learning Test (SEVLT) – to each patient at baseline and follow-up visits. A geriatrician referred the participants for a neuropsychological test battery and a standard neuropsychological examination (Informant Questionnaire on Cognitive Decline in the Elderly) if their scores (1) were below the 20th percentile at baseline on the 3MSE or SEVLT, or (2) had decreased ≥ 8 points on the 3MSE or ≥ 3 points on the SEVLT between baseline and follow-up. These cases were reviewed by a team of neurologists and neuropsychologist and given a diagnosis of “cognitively normal,” “cognitively impaired but not dementia (CIND),” or “dementia” according to standard diagnostic criteria. Those diagnosed with dementia or CIND were also referred for a magnetic resonance imaging (MRI) examination[11]. Detailed procedures for dementia and CIND screening and classification are described elsewhere[10]. In this study, all-cause dementia and CIND were combined into one outcome, including incident dementia or CIND cases and those who were CIND at baseline and converted to dementia during the follow-up, to capture both cognitive decline prior to dementia and dementia to improve our statistical power.

Noise Exposure Assessment

We used the SoundPLAN (Version 8.0, NAVCON, Fullerton, CA, USA) software package to estimate ambient noise exposure levels during the baseline year based on AADT data we received from the local MPO. We implemented the noise prediction model — Federal Highway Administration (FHWA) Traffic Noise Model - in SoundPLAN. Each subject’s geocoded residential address at baseline was used as the receiver point, and the TNM

algorithm estimated the noise levels with the following information - speed of the vehicles, counts of different types of vehicles, ground classification (soft vs. hard ground), and distance from receptor points to the roadway [12]. More information about the Traffic Noise Model has been detailed elsewhere[13,14]. We calculated average diurnal traffic patterns using hourly traffic counts we obtained from the State Department of Transportation (DOT) in 2002; we also used these to adjust the MPO AADT values to generate hour-of-day specific traffic counts at each receptor point. We estimated the A-weighted measure (which is the most common weighting applied to noise measurements in order to account for differences in sensitivity of human sound perception at specific frequencies[15]) day–night average (Ldn) and nighttime (22:00–07:00, Leq,n) sound levels for each participant's residence. We added a constant penalty of 10dB for noise during the nighttime to allow for a potentially higher sensitivity to noise during nighttime hours, as has been done previously [16].

Only roadway traffic was considered a source of noise in our study. Also, we only counted the FHWA classified light- and heavy-duty vehicles and assumed that the average vehicle speed was 55 miles per hour when we generated noise estimates. We generated noise exposure metrics as 24-hour averages (A-weighted) and nighttime averages (22:00–07:00). We treated noise exposure estimates as both continuous and binary variables (24-hour average noise: < 65 dB, ≥ 65 dB; nighttime noise: <55 dB, ≥55 dB) following recommendations by the World Health Organization community noise guidelines (2009) comparable to noise studies conducted in the US and European countries[12,17]. Alternatively, we used a four-category scale according to (rounded) quartile values to generate categorical noise metrics.

Covariates

Considering that the noise exposure we modeled originates from traffic only, we addressed potential confounding by co-exposure to traffic-related air pollution. Estimates for traffic-related nitrogen oxides (NOx) were generated based on participants' residential addresses at baseline using the California Line Source Dispersion Model version 4 (CALINE4)[18–20], with traffic volume data from the California DOT in 2002 and meteorology data from the California Air Resources Board Air Quality and Meteorological Information System (https://www.arb.ca.gov/aqmis2/metselect.php). Details have been described elsewhere[21].

During cohort recruitment, we collected demographic information such as birthplace (Mexico, US, or other), years of education, and occupation held longest during the lifetime (non-manual labor, manual labor, or other). At each interview, participants also reported information regarding smoking, alcohol drinking, physical activity, medical diagnoses including cardiovascular diseases and stroke, and medication use. An indicator for urban or rural residential location was generated relying on Census tract 2000 information[22]. Neighborhood socioeconomic status (neighborhood SES) is represented as a score ranging from 1 to 5 (low−high neighborhood SES) depending on six census (2000) estimates: percentage of (1) individuals aged 25+ years without a high school diploma, (2) individuals under the poverty limit, (3) individuals aged 16+ who had been in the workforce at one time but are unemployed, (4) households owning their home, (5) vacant housing units, and (6) median number of rooms in a household[23]. We evaluated physical activity level

according to time spent performing 18 different activities that older adults commonly engage in during a regular week[24]. We created a modified Charlson index to indicate comorbidity at baseline by assigning a point each for a history of certain medical diagnoses including myocardial infarction, congestive heart failure, stroke, liver disease, diabetes, renal disease, any malignancy, and leukemia or lymphoma; we then generated an index score by summing across these items[25].

Statistical Methods

We used Cox proportional hazards regression models with calendar time as the underlying time scale to assess the impact of noise exposures on incident dementia or CIND. Participants were censored at their last date of contact if they missed follow-up examinations or at their time of death if they died before the end of 2007.

Ambient noise exposure was entered into Cox regression models as a continuous variable normalized by its interquartile range width (IQRw). We also repeated these models with dichtomized and a quartile-based scale for noise exposures, and stratified on a series of risk factors to further explore the association between noise exposure and dementia or CIND. We selected covariates for adjustment based on the prior literature mostly for air pollution but also noise exposures and cognitive function[6]. We also adjusted for NSES and residential county in the models, considering that our noise estimates are primarily varying spatially. When examining the impacts of noise eposures on dementia/CIND, we first adjusted for baseline age, gender and years of education, and then added, occupation, smoking, alcohol consumption, physical activity level, NSES and residential location; as an additional step, we co-adjusted traffic-related NOx. We also investigated associations between noise and air pollution exposures and all-cause mortality as it is widely accepted that air pollution affects mortality (eAppendix). Finally, we also used competing risk models considering death as a competing risk when estimating effects between noise and dementia or CIND[26]. We used SAS 9.4 (SAS Institute Inc., Cary, NC, USA) for Cox regression analyses.

RESULTS

The average age of SALSA participants at baseline was 70 years; 42% were men. Approximately 60% reported having held a manual labor job during most of their life. 87% lived in an urban area and more than 70% in Sacramento County. At baseline, about one-third of participants already had received a diagnosis of cardiovascular disease or diabetes, two-thirds had hypertension and ~8% reported a stroke. Around 20% of these elderly participants were considered physically active, while about 12% were current smokers and less than 10% were daily alcohol drinkers. Compared with those who did not develop dementia or CIND during active follow-up, participants who incurred adverse events were older and less educated, more often manual laborers, had experienced stroke or diabetes. and had a higher Charlson score at baseline (Table 1). Participants who were exposed to higher 24-hour (≥ 65 dB) or nighttime noise levels (≥ 55 dB) were more likely to live in an urban area and higher neighborhood SES areas (eTable 1). The annual average 24-hour and nighttime noise exposure levels ranged from 39 – 100 dB and 31 – 92 dB, with mean values of 68 and 60 dB respectively; these two noise measures were highly correlated (*Pearson r* =

0.99). The average estimated NOx exposure level was 2.6 ppb, and the correlation with noise exposures was 0.43 (eTable 2).

We identified a total of 159 incident dementia or CIND cases were identified from 1998 to 2007. For 24-hour noise exposure, the hazard ratio of developing dementia or CIND adjusting for personal characteristics and lifestyle factors was increased (per 12 dB increase, HR = 1.2 [1.0, 1.5]). In the model with both traffic-related exposures, the noise effect estimate was slightly attenuated (HR = 1.2 [0.95, 1.5]) when we further adjusted for traffic-related NOx. Further inclusion of baseline cognitive function or primary language used did not change results (Table 2 and eTable3). Relying on nighttime noise only generated the same results likely due to the perfect correlation between modeled 24-hour and nighttime noise exposures. Using cut-off thresholds, high 24-hour (≥ 65 dB) and nighttime (≥ 55 dB) noise exposures were also positively associated with incident dementia or CIND, but the 95% CIs were wider (eTable 4). Overall, the risk of incident dementia or CIND was positively associated with increasing noise exposure (eFigure1). The risk of dementia or CIND increased with each (rounded) noise quartile and was highest when 24-hour and nighttime noise were higher than 75dB or and 65 dB respectively (Figure 2).

In the stratified analyses, higher traffic-related exposures were consistently and positively associated with the occurrence of dementia/CIND in almost all categories; however, the number of events and subjects per stratum were much smaller and the confidence intervals were wider (Table 3 and eTable 5). The associations of noise exposure with dementia/CIND were similar but slightly decreased with wider 95% CI in the competing risk model (eTable 6).

DISCUSSION

Worldwide, a growing elderly population combined with strong urbanization trends fostering noise exposure from traffic sources raises concerns that noise may have adverse effects on chronic neurodegenerative diseases[27–30]. In this study of older Mexican–American residents living in the California Sacramento Valley, noise exposures were positively associated with incidence of dementia or CIND even after adjusting for a host of other risk factors including traffic-related air pollution.

Associations between noise exposure and cardio-metabolic diseases have been reported in previous epidemiologic studies[31–33], but investigations of noise effects on cognitive outcomes are still rare. A small cross-sectional study in Italy observed differences in logical reasoning (Raven's progressive matrices 1938 [Raven PM38]: t = 3.2, $p < 0.01$; arithmetic reasoning: t = 2.30, $p = 0.02$) between noise-exposed traffic police officers (n=39) and noise-unexposed office employees (n= 42) but not in their attention abilities, or state and trait anxiety[34]. A much larger cross-sectional study conducted within the Heinz Nixdorf Recall study in Germany consisting of 4,086 participants aged 50–80 years reported that for each 10 A-weighted decibel [dB(A)] increase in traffic noise modeled at the participants' residence the risk of mild cognitive impairment (MCI) (odds ratio [OR] = 1.4 [1.0, 1.9]) as well as amnestic MCI (aMCI) (OR = 1.5 [1.1, 2.2]) increased [35]. Most recently, a longitudinal cohort study in England of 130,978 adults aged 50–79 years observed a small

positive association between incident dementia and traffic nighttime noise at the postcode level (HR = 1.0 [1.0, 1.1] per 2.7 dB increase in nighttime noise)[9]. Other studies of noise did not find any associations[7,8,36,37], which might be explained by differences in study designs, methods of measuring noise exposure or sources of noise investigated (i.e. occupational-related noise), the time-frame for which noise was estimated (i.e. only nighttime), or how cognitive function was assessed[6].

Although the evidence from epidemiologic studies is still inconsistent, animal studies have linked noise exposure to decreased cognitive performance. Experimental studies indicated that noise is acting as a stressor that can influence brain structures such as reducing the brain volume in the medial prefrontal cortex (mPFC) area or cortical thickness in the hippocampus and amygdala area, which are essential components of the neural circuitry mediating stress responses[38,39]. Noise stressors could cause the amygdala to activate stress pathways in the hypothalamus and brainstem, followed by elevated release of noradrenaline and dopamine, and consequently lead to dysregulation of the prefrontal cortex responsible for cognitive abilities such as executive function[40–42]. Furthermore, noise might affect insulin resistance and endothelial dysfunction via activation of the hypothalamic–pituitary–adrenal (HPA) axis[43–46] that influences corticosterone and adrenocorticotropic hormone secretion followed by metabolic dysregulation[33,47–49] and cognition damage. A recent study reported increased catechol-O-methyltransferase (COMT) gene DNA methylation in the medulla oblongata of rats exposed to environmental noise (70–75 dB) for three days during nighttime and in the inferior colliculus after long-term exposure (70–75 dB for 21 days during nighttime). COMT serves as a key enzyme for the inactivation of prefrontal dopamine and is closely related to stress response and cognition. This experiment suggests one possible pathway through which noise exposure may influence cognitive function i.e. by modulating stress-responses[50].

When we stratified on several risk factors, the risk of having dementia or CIND seemed higher among those who held non-manual jobs and those who lived in high neighborhood SES areas; however, individuals within the same occupation or neighborhood SES category might still differ according to personal[23] and lifestyle characteristics (eTable7 and eTable8). Moreover, stratification reduces the numbers of events and subjects considerably resulting in much wider confidence intervals such that it is hard to draw firm conclusions from these analyses.

The SALSA study is one of few studies focusing on brain health in older Mexican–Americans and other Hispanics[10], and also one of few studies in North America exploring the long-term effect of noise on cognitive impairment. We estimated noise exposure at baseline residential addresses geocoded employing Global Positioning System (GPS) readings at the door step (performed during home visits), which guarantees high geo-location quality. During follow-up visits, incident dementia or CIND was diagnosed after repeated cognitive function testing and further confirmed by imaging examination (MRI), i.e. we did not have to rely on self-reports or records, thus ensuring a high accuracy of the dementia or CIND diagnosis in SALSA.

There are nevertheless several limitations. First, we did not have lifetime residential histories for the cohort participants, nor information regarding bedroom orientation, window insulation, or habits of opening windows or using noise protective equipment such as earplugs[51], all of which may have contributed to measurement error for noise exposures. However, participants had on average lived at their baseline residence for 22 years, and 90% remained in California throughout the study period with only 339 changing addresses between baseline and last follow-up visit. In our study, altogether 221 participants changed their addresses before the dementia or CIND events or last follow-up occurred, and among them only 96 moved out of the county. Excluding these participants did not change the results more than minimally (eTable9). Thus, the observed results suggested that the baseline address-based noise measurements are appropriate surrogates for long-term exposure. Additionally, study participants were mostly retired and consequently are expected to be at home during the day. Since our noise exposure was residential address-based, exposure misclassification should be expected to be smaller than in a working population. While the difference between ambient and personal-level exposure owing to individual behavior would be expected to cause exposure misclassification at the individual level, estimates of noise exposure at residences can be considered instrumental variables for personal exposures. That is, personal exposure is the common descendant of ambient exposure and individual behaviors, while individual behaviors are unlikely to influence ambient exposure[52]; therefore our results are less likely to be affected by confounding from personal behaviors. Additionally, we also have adjusted for personal demographic, lifestyle factors, health status, neighborhood SES and type of residential location related to personal health behaviors and brain health, but residual confounding can never be ruled out completely. Selection bias resulting from loss of follow-up was minimal in our study because the percentage of subjects lost to follow-up was 2.3% per year. Furthermore, environmental exposures and cognitive impairment status were not reported by the subjects themselves, making the differential loss-to-follow-up unlikely. Additionally, noise exposure is commonly considered to be highly related to traffic-related air pollution since they both originate from traffic and occur in time and space simultaneously, therefore, we also adjusted for air pollution. Although the 95% CIs became wider with such adjustments, the associations between noise and dementia or CIND remained similar, indicating an independent association of noise exposure with these conditions[9]. Our results for all-cause mortality and air pollution are consistent with what we would expect according to the literature, thus corroborating the validity of our exposure measures (eTable10 and eTable11). Lastly, we only took into account continuous roadway traffic as the source of residential noise exposures, we did not assess stop-and-go traffic, noise from the airport or railways, or occupational noise exposure before retirement, which likely contributed to non-differential exposure misclassification. Also, our noise model applied the same percentages for vehicle types (light or heavy) for daytime and nighttime to all roadways since we did not have sufficient information to model diurnal fluctuations. Thus, our 24-hour and nighttime noise estimates are by design highly correlated. Future noise studies taking into account diurnal traffic changes, additional major sources of noise as well as details about occupational exposures are needed.

Our findings among older Mexican–Americans are consistent with the hypothesis that noise exposure elevates the risk of cognitive impairment and affects brain health. Future studies taking into account other noise sources and occupational noise exposure before retirement - possibly using a life-course approach - are needed.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Sources of Funding:

This work was supported by grants R01ES023451 and F32ES028087 from National Institute of Environmental Health Sciences, AG012975and AG033751 from National Institute on Aging, and DK060753 from National Institute of Diabetes and Digestive and Kidney Diseases.

References

1. Paul KC, Haan M, Mayeda ER, Ritz BR. Ambient Air Pollution, Noise, and Late-Life Cognitive Decline and Dementia Risk. Annu Rev Public Health 2019;40:203–220. [PubMed: 30935305]
2. Matthews KA, Xu W, Gaglioti AH, et al. Racial and ethnic estimates of Alzheimer's disease and related dementias in the United States (2015–2060) in adults aged >/=65 years. Alzheimers Dement 2019;15(1):17–24. [PubMed: 30243772]
3. Alzheimer's_Society. Risk factors for dementia https://www.alzheimers.org.uk/sites/default/files/pdf/factsheet_risk_factors_for_dementia.pdf Accessed November 12, 2019.
4. Lim SS, Vos T, Flaxman AD, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. The Lancet 2012;380(9859):2224–2260.
5. Stieb DM, Chen L, Eshoul M, Judek S. Ambient air pollution, birth weight and preterm birth: a systematic review and meta-analysis. Environ Res 2012;117:100–111. [PubMed: 22726801]
6. Tzivian L, Winkler A, Dlugaj M, et al. Effect of long-term outdoor air pollution and noise on cognitive and psychological functions in adults. Int J Hyg Environ Health 2015;218(1):1–11. [PubMed: 25242804]
7. Brink M Parameters of well-being and subjective health and their relationship with residential traffic noise exposure--a representative evaluation in Switzerland. Environ Int 2011;37(4):723–733. [PubMed: 21419495]
8. Bocquier A, Cortaredona S, Boutin C, et al. Is exposure to night-time traffic noise a risk factor for purchase of anxiolytic-hypnotic medication? A cohort study. Eur J Public Health 2014;24(2):298–303. [PubMed: 23985724]
9. Carey IM, Anderson HR, Atkinson RW, et al. Are noise and air pollution related to the incidence of dementia? A cohort study in London, England. BMJ Open 2018;8(9):e022404.
10. Haan MN, Mungas DM, Gonzalez HM, Ortiz TA, Acharya A, Jagust WJ. Prevalence of dementia in older latinos: the influence of type 2 diabetes mellitus, stroke and genetic factors. J Am Geriatr Soc 2003;51(2):169–177. [PubMed: 12558712]
11. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Washington, DC: American Psychiatric Association; 2000.
12. Seong JC, Park TH, Ko JH, et al. Modeling of road traffic noise and estimated human exposure in Fulton County, Georgia, USA. Environ Int 2011;37(8):1336–1341. [PubMed: 21704376]
13. US_DOT. FHWA Traffic Noise Model (FHWA TNM) Technical Manual Federal Highway Administration, FHWA-PD-96-010, DOTVNTSC-FHWA-98-2. 1998;http://www.fhwa.dot.gov/environment/noise/tnm/ver10tm/tnm10techmanual.pdf. Accessed 8/14/2019.
14. US_DOT. Validation of FHWA's Traffic Noise Model (TNM): Phase 1 Federal Highway Administration, U.S. Department of Transportation, DOT-

VNTSC-FHWA-02-01. 2002;http://www.fhwa.dot.gov/environment/noise/traffic_noise_model/model_validation/phase_one/tnmph1va.pdf. Accessed 8/14/2019.

15. International_Electrotechnical_Commission. International Electrotechnical Commission Glossary A-weighting sound pressure level; A-weighted sound pressure level http://std.iec.ch/terms/terms.nsf/3385f156e728849bc1256e8c00278ad2/f2cdfd3b716912c9c12579eb004f55f6?OpenDocument. Accessed July 17, 2020.
16. Fecht D, Hansell AL, Morley D, et al. Spatial and temporal associations of road traffic noise and air pollution in London: Implications for epidemiological studies. Environ Int 2016;88:235–242. [PubMed: 26773394]
17. Lee EY, Jerrett M, Ross Z, Coogan PF, Seto EY. Assessment of traffic-related noise in three cities in the United States. Environ Res 2014;132:182–189. [PubMed: 24792415]
18. Wu J, Houston D, Lurmann F, Ong P, Winer A. Exposure of PM2.5 and EC from diesel and gasoline vehicles in communities near the Ports of Los Angeles and Long Beach, California. Atmospheric Environment 2009;43(12):1962–1971.
19. Wu J, Laurent O, Li L, Hu J, Kleeman M. Adverse Reproductive Health Outcomes and Exposure to Gaseous and Particulate-Matter Air Pollution in Pregnant Women. Res Rep Health Eff Inst 2016(188):1–58.
20. Benson PE, Pinkerman KO. CALINE4, a dispersion model for predicting air pollution concentration near roadways State of California, Department of Transportation, Division of Engineering Services, Office of Transportation Laboratory;1989.
21. Yu Y, Paul K, Arah OA, et al. Air pollution, noise exposure, and metabolic syndrome - A cohort study in elderly Mexican-Americans in Sacramento area. Environ Int 2019;134:105269. [PubMed: 31778933]
22. United_States_Department_of_Agriculture_Economic_Research_Service. Rural-Urban Commuting Area Codes https://www.ers.usda.gov/data-products/rural-urban-commuting-area-codes/ Accessed November 12, 2019.
23. Yost K, Perkins C, Cohen R, Morris C, Wright W. Socioeconomic status and breast cancer incidence in California for different race/ethnic groups. Cancer Causes Control 2001;12(8):703–711. [PubMed: 11562110]
24. Shih IF, Paul K, Haan M, Yu Y, Ritz B. Physical activity modifies the influence of apolipoprotein E epsilon4 allele and type 2 diabetes on dementia and cognitive impairment among older Mexican Americans. Alzheimers Dement 2018;14(1):1–9. [PubMed: 28692819]
25. Aiello AE, Haan MN, Pierce CM, Simanek AM, Liang J. Persistent infection, inflammation, and functional impairment in older Latinos. J Gerontol A Biol Sci Med Sci 2008;63(6):610–618. [PubMed: 18559636]
26. Fine JP, Gray RJ. A Proportional Hazards Model for the Subdistribution of a Competing Risk. Journal of the American Statistical Association 1999;94(446):496–509.
27. Eze IC, Schaffner E, Fischer E, et al. Long-term air pollution exposure and diabetes in a population-based Swiss cohort. Environ Int 2014;70:95–105. [PubMed: 24912113]
28. Eze IC, Foraster M, Schaffner E, et al. Long-term exposure to transportation noise and air pollution in relation to incident diabetes in the SAPALDIA study. Int J Epidemiol 2017;46(4):1115–1125. [PubMed: 28338949]
29. Cui B, Zhu L, She X, et al. Chronic noise exposure causes persistence of tau hyperphosphorylation and formation of NFT tau in the rat hippocampus and prefrontal cortex. Exp Neurol 2012;238(2):122–129. [PubMed: 22971273]
30. Cui B, Li K, Gai Z, et al. Chronic noise exposure acts cumulatively to exacerbate Alzheimer's disease-like amyloid-β pathology and neuroinflammation in the rat hippocampus. Scientific reports 2015;5:12943. [PubMed: 26251361]
31. de Souza TC, Perisse AR, Moura M. Noise exposure and hypertension: investigation of a silent relationship. BMC Public Health 2015;15(1):328. [PubMed: 25880499]
32. Arlien-Søborg MC, Schmedes AS, Stokholm Z, et al. Ambient and at-the-ear occupational noise exposure and serum lipid levels. International archives of occupational and environmental health 2016;89(7):1087–1093. [PubMed: 27319006]

33. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta-analysis. Diabetes Care 2010;33(2):414–420. [PubMed: 19910503]
34. Chiovenda P, Pasqualetti P, Zappasodi F, et al. Environmental noise-exposed workers: event-related potentials, neuropsychological and mood assessment. Int J Psychophysiol 2007;65(3):228–237. [PubMed: 17544162]
35. Tzivian L, Dlugaj M, Winkler A, et al. Long-Term Air Pollution and Traffic Noise Exposures and Mild Cognitive Impairment in Older Adults: A Cross-Sectional Analysis of the Heinz Nixdorf Recall Study. Environ Health Perspect 2016;124(9):1361–1368. [PubMed: 26863687]
36. Gomes L, Martinho Pimenta A, Castelo Branco N. Effects of occupational exposure to low frequency noise on cognition. Aviation, space, and environmental medicine 1999.
37. Hardoy MC, Carta MG, Marci AR, et al. Exposure to aircraft noise and risk of psychiatric disorders: the Elmas survey. Social Psychiatry and Psychiatric Epidemiology 2005;40(1):24–26. [PubMed: 15624071]
38. Czeh B, Muller-Keuker JI, Rygula R, et al. Chronic social stress inhibits cell proliferation in the adult medial prefrontal cortex: hemispheric asymmetry and reversal by fluoxetine treatment. Neuropsychopharmacology 2007;32(7):1490–1503. [PubMed: 17164819]
39. Jafari Z, Kolb BE, Mohajerani MH. Chronic traffic noise stress accelerates brain impairment and cognitive decline in mice. Exp Neurol 2018;308:1–12. [PubMed: 29936225]
40. Arnsten AFT, Goldman-Rakic PS. Noise Stress Impairs Prefrontal Cortical Cognitive Function in Monkeys: Evidence for a Hyperdopaminergic Mechanism. JAMA Psychiatry 1998;55(4):362–368.
41. Arnsten AF. Stress signalling pathways that impair prefrontal cortex structure and function. Nat Rev Neurosci 2009;10(6):410–422. [PubMed: 19455173]
42. Jafari Z, Kolb BE, Mohajerani MH. Noise Exposure Accelerates the Risk of Cognitive Impairment and Alzheimer's Disease: Adulthood, Gestational, and Prenatal Mechanistic Evidence from Animal Studies. Neurosci Biobehav Rev 2019.
43. Griefahn B, Robens S. Experimental studies on the effects of nocturnal noise on cortisol awakening response. Noise Health 2010;12(47):129–136. [PubMed: 20472958]
44. Schmidt FP, Basner M, Kroger G, et al. Effect of nighttime aircraft noise exposure on endothelial function and stress hormone release in healthy adults. Eur Heart J 2013;34(45):3508–3514a. [PubMed: 23821397]
45. 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. Scientific reports 2016;6:36693. [PubMed: 27811997]
46. Björntorp P, Rosmond R. Obesity and cortisol. Nutrition 2000;16(10):924–936. [PubMed: 11054598]
47. Passchier-Vermeer W, Passchier WF. Noise exposure and public health. Environ Health Perspect 2000;108 Suppl 1(Suppl 1):123–131. [PubMed: 10698728]
48. Chaput JP, Despres JP, Bouchard C, Tremblay A. Short sleep duration is associated with reduced leptin levels and increased adiposity: Results from the Quebec Family Study. Obesity 2007;15(1):253–261. [PubMed: 17228054]
49. Van Cauter E, Spiegel K, Tasali E, Leproult R. Metabolic consequences of sleep and sleep loss. Sleep Med 2008;9 Suppl 1:S23–28. [PubMed: 18929315]
50. Guo L, Li PH, Li H, et al. Effects of environmental noise exposure on DNA methylation in the brain and metabolic health. Environ Res 2017;153:73–82. [PubMed: 27914298]
51. Fuks KB, Weinmayr G, Basagana X, et al. Long-term exposure to ambient air pollution and traffic noise and incident hypertension in seven cohorts of the European study of cohorts for air pollution effects (ESCAPE). Eur Heart J 2017;38(13):983–990. [PubMed: 28417138]
52. Weisskopf MG, Kioumourtzoglou MA, Roberts AL. Air Pollution and Autism Spectrum Disorders: Causal or Confounded? Curr Environ Health Rep 2015;2(4):430–439. [PubMed: 26399256]

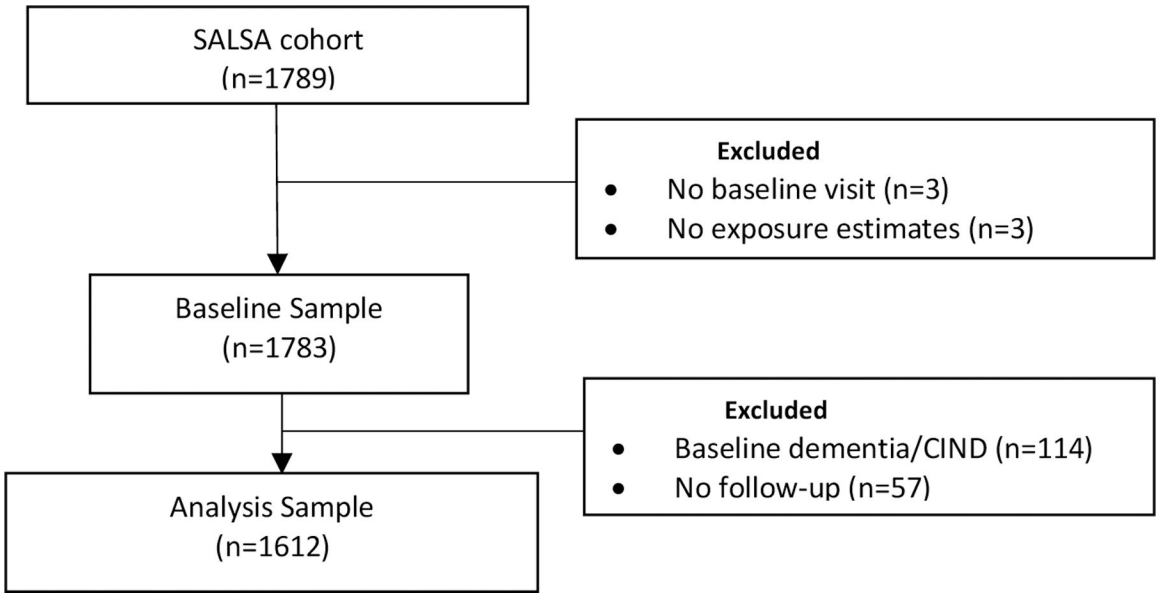

Figure 1.
Flow chart of study population, Sacramento Area Latino Study on Aging (SALSA), 1998–2008. Abbreviations: CIND, cognitive impaired without dementia.

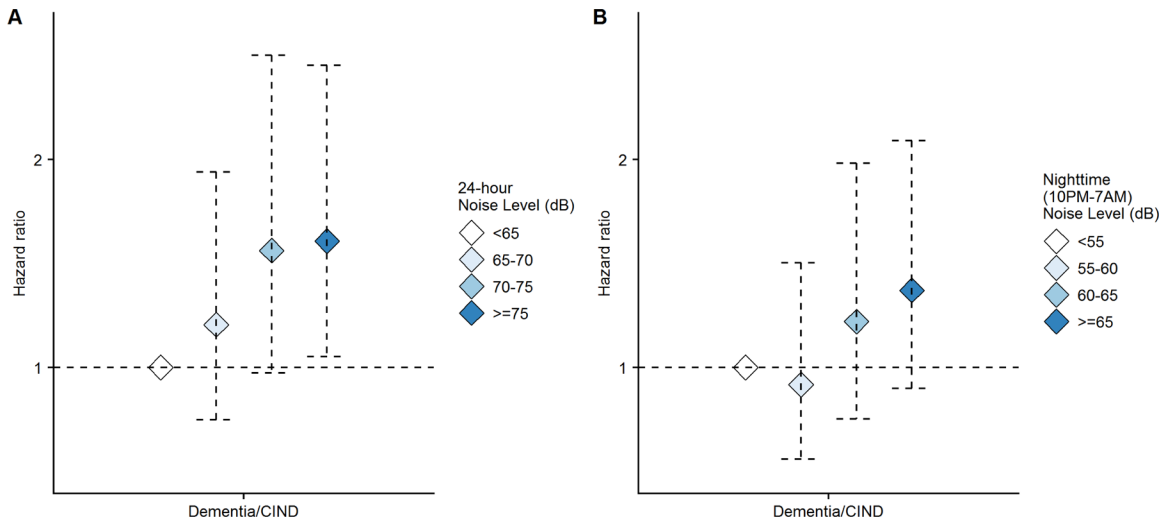

Figure 2.

Effect estimates (and 95% confidence intervals) from adjusted Cox models for annual average of 24-hour (A) or nighttime noise (B) exposure at a quartile-based scale levels and the risk of dementia/CIND. A: 24-hour noise level was divided into 4 categories (<65 dB, 65–70 dB, 70–75 dB, and ≥ 75 dB) according to (rounded) quartile values. The reference group included those with 24-hour average noise exposure <65 dB. B: Nighttime noise level was divided into 4 categories (<55 dB, 55–60 dB, 60–65 dB, and ≥65 dB), according to (rounded) quartile values. The reference group included those with nighttime noise exposure <55 dB. Models were adjusted for baseline age, gender, years of education, occupation during most of life, smoking status, alcohol consumption status, physical activity, neighborhood socioeconomic status indicator, residential county, traffic-related air pollution. CIND, cognitive impairment without dementia; dB, decibels. The dashed lines display the 95% confidence intervals.

Table 1.

Characteristics of the participants used for incidence analyses at baseline, Sacramento Area Latino Study of Aging, 1998–2007.

	Total	Dementia or CIND incidence	
Characteristics	**(n=1612)**	**Event (n=159)**	**Non-event (n=1453)**
Baseline Age, years, mean (SD)	70 (6.8)	75 (7.8)	70 (6.5)
Male, N(%)	680 (42)	58 (37)	622 (43)
Years of Education, years, mean (SD)	7.4 (5.3)	5.8 (5.2)	7.6 (5.3)
Sacramento County Residence, N(%)	1255 (78)	118 (74)	1137 (78)
Urban Residence, N(%)	1400 (87)	136 (86)	1264 (87)
Birth Country, N(%)			
Mexico	721 (45)	76 (48)	645 (45)
United States	797 (50)	75 (47)	722 (50)
Others (i.e. Central or South America)	88 (6)	8 (5)	80 (6)
Occupation Held During Most of Lifetime, N(%)			
Non-Manual	346 (22)	14 (9)	332 (23)
Manual	960 (61)	104 (67)	856 (60)
Other (Housewives and Unemployed)	282 (18)	38 (24)	244 (17)
Neighborhood Socio-economic Status (NSES), N(%)			
Lowest (NSES = 1)	544 (34)	64 (40)	480 (33)
Lower-middle/middle (NSES = 2 or 3)	912 (57)	80 (50)	832 (57)
High-middle/high (NSES = 4 or 5)	156 (10)	15 (9)	141 (10)
Baseline Smoking Status, N(%)			
Never/Non-Smoker	735 (46)	75 (47)	660 (46)
Former Smoker	681 (42)	64 (40)	617 (43)
Current Smoker	189 (11)	20 (17)	169 (12)
Baseline Alcohol Status, N(%)			
Frequent (Daily) Drinker	146 (9)	8 (5)	138 (10)
Moderate (Weekly) Drinker	172 (11)	11 (6)	161 (11)
Occasional (Monthly) Drinker	158 (10)	12 (8)	146 (10)
Yearly/Rarely/Never Drinker	1125 (70)	128 (81)	997 (69)
Baseline Physically Active, N(%)	341 (21)	27 (19)	314 (22)
Baseline Self-reported Cardiovascular Disease, N(%)	574 (36)	70 (44)	504 (35)
Baseline Self-reported Stroke, N(%)	126 (8)	26 (16)	100 (7)
Baseline Hypertension, N(%)	1093 (68)	115 (72)	978 (67)
Baseline Diabetes, N(%)	513 (32)	71 (45)	442 (31)
Baseline Charlson Index, mean (SD)	0.9 (1.2)	1.1 (1.2)	0.9 (1.2)
Baseline BMI, mean (SD)	30 (6.0)	29 (5.2)	30 (6.1)
Traffic-related NOx, ppb, mean (SD)	2.6 (2.2)	2.7 (2.3)	2.6 (2.5)
24hr Average Noise, dB, mean (SD)	69 (8.9)	70 (8.9)	68 (8.9)
Nighttime (10PM - 7AM) Noise, dB, mean (SD)	60 (8.9)	62 (8.9)	60 (8.9)

Note: CIND, cognitive impairment without dementia; dB, decibels; ppb, part per billion; BMI, body mass index; NOx, nitrogen oxides.

Table 2.

Effect estimates (and 95% CIs) from Cox models for 24-hour average noise exposure (per 11.6 dB increase) and the risk of incident dementia or CIND.

	Noise in single exposure model	Noise exposure additionally adjusted for traffic-related NOx
Models	**HR (95% CI)**	**HR (95% CI)**
Model 1[a]	1.2 (0.98, 1.5)	1.2 (0.93, 1.5)
Model 2[b]	1.3 (1.0, 1.6)	1.2 (0.97, 1.6)

Note: CIND, cognitive impairment without dementia; NOx, nitrogen oxides; dB, decibels; HR, hazard ratio; 95% CI, 95% confidence interval. For the noise exposure, we used 11.6 dB increase as the unit to estimate effects.

[a] Adjusted for baseline age, gender, years of education.

[b] Adjusted for baseline age, gender, years of education, occupation during most of life, smoking status, alcohol consumption status, physical activity level, neighborhood socioeconomic status indicator, residential county, baseline Charlson index, baseline cognition function and primary language.

Table 3.

Effect estimates (and 95% CIs) from Cox models[a] for 24-hour average noise exposure (per 11.6 dB increase) and the risk of incident dementia or CIND, stratified by other major risk factors.

	N (Total=1612)	Number of cases (Total = 159)	HR (95% CI)
Age			
60–80	1454	114	1.2 (0.95 −1.6)
>=80	143	44	1.4 (0.86 −2.1)
Gender			
Male	680	58	1.2 (0.87 −1.7)
Female	932	101	1.3 (0.97 −1.7)
Occupation held during most of life			
Non-Manual	346	14	1.6 (0.78 −3.1)
Manual	960	104	1.2 (0.91 −1.5)
Other (Housewives and Unemployed)	282	38	1.4 (0.87 −2.2)
Smoking Status			
Never	735	75	1.1 (0.79 −1.5)
Former	681	64	1.4 (0.98 −1.9)
Current	189	20	1.3 (0.69–2.3)
Neighborhood Socio-Economic Status (NSES)			
Lowest (NSES =1)	544	64	1.2 (0.83–1.6)
Lower-middle/middle (NSES =2 or 3)	912	80	1.2 (0.86 −1.6)
High-middle/high (NSES =4 or 5)	156	15	1.9 (0.89 −4.1)
Comorbidity			
No comorbidity (Charlson Index = 0)	819	56	1.3 (0.92 −1.9)
Comorbidity (Charlson Index > 0)	787	103	1.2 (0.90 −1.5)
County			
Sacramento	1255	118	1.3 (0.98 −1.6)
Non-Sacramento	357	41	1.1 (0.70 −1.6)
Living in Urban or Rural Area			
Urban	1400	136	1.2 (0.98 −1.6)
Rural	212	23	1.1 (0.63 −1.9)

Note: CIND, cognitive impairment without dementia; dB, decibels; HR, hazard ratio; 95% CI, 95% confidence interval. For the noise exposure, we used 11.6 dB increase as the unit to estimate effects.

[a]Adjusted for baseline age, gender, years of education, neighborhood socioeconomic status indicator, occupation during most of life, residential county, smoking status, alcohol consumption status, physical activity level.