Self-Reported Occupational Noise may be Associated With Prevalent Chronic obstructive pulmonary disease in the US General Population

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Abstract

Introduction: Occupational noise exposure and chronic obstructive pulmonary disease (COPD) are common in the United States, but so far their association has not been explored. Given the neuroimmunological effects of noise, such an association seems plausible. Thus, the present study aimed to explore the association of occupational noise exposure with prevalent COPD in the US general population.

Materials and Methods: We used data from the population-based National Health Interview Survey (NHIS) 2014. The cross-sectional association of self-reported duration of exposure to very loud noise during participants’ occupational lifetime with self-reported COPD and emphysema was explored using weighted logistic regression.

Results and Discussion: The fully adjusted model yielded odds ratio (OR) ≥15 years = 1.68 [95% confidence interval (CI): 1.28, 2.21] for COPD and OR ≥15 years = 1.61 (95% CI: 1.13, 2.30) for emphysema. Race/ethnicity was a significant effect modifier. In sensitivity analysis with cumulative noise exposure based on a job exposure matrix, we found no effect.

Conclusion: In conclusion, we found a relationship between self-reported occupational noise exposure and the risk of prevalent COPD in the US general population, but none with objective noise levels. Being the first study on the subject matter, and given the design limitations, these findings are tentative and should be treated with caution.

Keywords: COPD, emphysema, noise exposure, occupational noise, respiratory disease

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a lung “disease state characterized by airflow limitation that is not fully reversible.”¹¹ Data from the National Health and Nutrition Examination Survey (NHANES) 2007–2010, based on spirometric examinations, showed that the prevalence of COPD among people aged 40–79 years in the US varies from 10.2 to 20.9%, depending on the diagnostic criteria.²² Since 1990, the number of years of life lost due to COPD in the US has increased with 34.7%, and now it ranks fourth, just behind ischemic heart disease, lung cancer, and stroke.³³ This burden translates into some $50 billion in direct and indirect health expenditures.⁴⁴ Cigarette-smoking is considered the most prominent risk factor for developing COPD, but there is substantial proportion of the variance in COPD that cannot be accounted for by smoking.⁵⁵ Evidence has accumulated of some novel risk factors such as asthma, tuberculosis, genetics, and indoor/outdoor air pollution.⁶⁶ As regards occupational environment, self-reported exposure to dust, fumes, and vapors has been associated with elevated risk of COPD,⁵⁷ and a recent meta-analysis estimated odds ratio (OR) = 1.43 [95% confidence interval (CI): 1.19, 1.73] for COPD among workers exposed to vapors, gases, dusts, or fumes, in comparison to not exposed workers.⁶⁷ However, no study has so far explored whether occupational noise exposure is associated with COPD. Such an association seems counterintuitive, at first, but there is a plausible mechanistic hypothesis behind it.

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Noise is a socio-acoustic stressor, which may alter the immune system through a cascade of neurochemical processes. It activates the hypothalamic–pituitary–adrenal axis and sympathetic–adrenal axis, leading to increase in catecholamine and cortisol. In turn, stress hormones may be responsible for reduction in lymphocyte count, migration, and adhesion, and suppressed natural killer-cell activity, which may predispose the respiratory system to infections. Sleep disturbance due to noise may hamper the division and migration of lymphocytes, promote systemic inflammation, and suppress the antiviral immune response. Finally, noise stress may generate reactive oxygen species and pro-inflammatory cells, involved in pulmonary inflammation, degradation of the extracellular matrix, and fibrotic changes in connective tissue. Similar neuroimmunological mechanisms are involved in the pathogenesis of COPD.

Studues have reported associations between traffic noise and respiratory diseases such as bronchitis, pneumonia, and asthma. To our knowledge, there are no occupational studies on the topic, although some authors reported an adverse impact of workplace noise on lung function. On the basis of the same pathophysiological rationale described for noise, authors have reported both cross-sectional and longitudinal associations between higher psychological stress and COPD.

Workplace noise is typically higher than residential noise, and about 22 million workers in the US are exposed to dangerous levels of noise above 85 dB. Therefore, an insight into this alleged risk factor is needed to gain a better understanding of COPD prevention. The present study aimed to explore the association of occupational noise exposure with prevalent COPD in the US general population.

**Material and Methods**

**Study population**

For this study, we re-analyzed the National Health Interview Survey (NHIS) 2014 dataset. NHIS is a population-based cross-sectional survey conducted in the US by the Centers for Disease Control and Prevention (CDC)/National Center for Health Statistics (NCHS). NHIS 2014 employed a multistage stratified random sampling, ensuring nationally representative estimates. All collected data were based on a questionnaire administered during a personal household interview. The final response rate was 58.9%.

The CDC/NCHS grants researchers an academic access to primary data from its surveys through public-use data files for statistical analysis or reporting purposes. All information used in the present study was obtained anonymized from the website of the CDC/NCHS (http://www.cdc.gov/nchs/nhis/). Participation in the NHIS 2014 was voluntary and participants’ confidentiality was assured under Section 308 (d) of the Public Health Service Act. All information collected in the survey is kept strictly confidential, and privacy is protected by public laws.

The “Sample Adult” file contained data on 36,697 civilian noninstitutionalized people aged ≥18 years, who were living in the US at the time of the interview. Annual family income was extracted from another data file – the “Family” file. Only complete-case analyses were conducted; therefore, sample-sizes vary due to missing data. The multivariate models for self-reported COPD and emphysema were based on 29,867 and 29,882 participants, respectively.

**Assessment of chronic obstructive pulmonary disease and emphysema**

Cases of prevalent COPD and emphysema in NHIS 2014 were defined as self-reported health care professional-diagnoses: “Have you EVER been told by a doctor or other health professional that you had chronic obstructive pulmonary disease, also called COPD?” and “Have you EVER been told by a doctor or other health professional that you had...Emphysema?” Diagnosis with emphysema was used in addition to COPD to minimize the self-report bias (i.e., COPD cases may report having emphysema, but not COPD).

**Assessment of occupational lifetime noise exposure**

Participants in NHIS 2014 were asked “Have you ever had a job, or combination of jobs, where you were exposed to very loud sounds or noise for 4 or more hours a day, several days a week?”, and, if they answered affirmatively, “In your work, how many months or years have you been exposed at work to very loud sounds or noise for 4 or more hours a day, several days a week?” “Very loud” noise referred to a noise so loud such that one must shout to be understood by someone standing 3 feet (arm’s length) away from them. This question has been validated in several studies and is a good proxy for >80–85 dB. Participants were allocated in one of the following durations of exposure categories: “never exposed” (worked but not exposed, or never worked) (reference category), “<5 years”, “5–9 years”, “10–14 years”, or “≥15 years.”

In addition to the questions above, participants answered two other questions, which referred to “loud noise,” but were otherwise similar in wording. “Loud” was defined as noise so loud that one must speak in a raised voice to be heard. For sensitivity analysis, we constructed another exposure variable representing different thresholds of noise exposure. This threshold variable had three categories: “never exposed” (reference category), “ever exposed to loud noise” (including loud but not very loud noise exposure), and “ever exposed to very loud noise” (including loud and very loud noise exposure).

In addition to self-reported exposure, job-specific noise exposure levels were obtained from a US/Canada job-exposure matrix (JEM) for noise. This database was...
recently published online. Owing to some technical issues, it was used only for sensitivity analysis to give us some idea of the effect on COPD if objective noise exposure was used instead of self-reported. Briefly, the developers collected and compiled occupational noise exposure data from government, academic, and industry sources, and coded each measurement using a standardized occupational coding scheme. The version of the JEM we used is available at http://noisejem.sph.umich.edu/. It reports time weighted average sound level in dB(A) accumulated for any time period but with an average level computed over an 8 h time period. To link these exposure measurements to participants, we transcribed the detailed occupation codes in NHIS 2014 to corresponding 4-digit Standard Occupation Classification (SOC 2010) codes. Because the JEM reports 6-digit SOC codes, and NHIS 2014 reports 4-digit SOC codes, we averaged all 6-digit SOC code measurements within the respective 4-digit SOC code. To occupations with no measurements at the 4-digit SOC level, we assigned the average noise level at the 2-digit SOC level. To account for the length of participants’ exposure, we calculated cumulative noise exposure (CNE) in dB(A)-years according to the formula:

\[
\text{CNE} = 10 \times \log_{10} \left[ 10^{\text{noise level}/10} \times \text{exposure in years} \right]
\]

For exposure duration, we used the overall number of years on the current job. For <1 year on the job, we assumed 1 year of exposure, and for more than 35 years, we assumed 35 years of exposure. Thus, the analysis with CNE is limited to participants employed at the time of the interview and with a known length of service.

**Other covariates**

The NHIS 2014 dataset supplied the following sociodemographic variables: age, gender, race/ethnicity (white, black/African American, American Indian/Chinese/Filipino/Asian Indian, multiple race/other) and annual family income (<$35,000, $35,000–74,999, $75,000–99,999, ≥$100,000).

We also elicited information on cigarette-smoking status (never smoker, former smoker, current some day smoker, and current every day smoker), ever smoked other tobacco products (yes/no), alcohol-drinking status (lifetime abstainer, former drinker, and current drinker), frequency of light/moderate physical activity per week (never, unable to do it, <3 times/week, 4–7 times/week, and >7 times/week), and frequency of vigorous physical activity per week (never, unable to do it, <3 times/week, 4–7 times/week, and >7 times/week).

Body mass index (BMI) was calculated from self-reported weight (in kilograms) divided by height squared (in meters). It was categorized as “< 25.00 kg/m^2” (normal or underweight), “25.00–29.99 kg/m^2” (overweight) or “≥30.00 kg/m^2” (obese).

Participants’ occupations were clustered into 23 major occupation groups, based on Census Occupation Codes as reported by SOC codes. They were further clustered into white-collar (0010–3540 and 4700–5930), service (3600–3950 and 4000–4650), and blue-collar workers (6200–6940 and 7000–9750) for some of the analyses. Another question asked was whether participants’ current job was also their longest held job.

Self-reported diagnosis with asthma was ascertained by the question: “Have you EVER been told by a doctor or other health professional that you had asthma?” Participants’ self-rated hearing (without hearing aids/listening devices) was elicited from the question “Is your hearing excellent, good, a little trouble hearing, moderate trouble, a lot of trouble, or are you deaf?” It was recoded as “excellent hearing,” “good/little trouble hearing” or “moderate/lot of trouble hearing.”

Another question asked about hearing protection use at work: “BEFORE THE LAST 12 MONTHS, when exposed at work to VERY LOUD sounds or noise, how often DID you wear hearing protection, such as ear plugs or ear muffs?”

**Data analysis**

The complex design of the NHIS 2014 was incorporated into all individual-level analyses to obtain nationally representative estimates. This was made possible using strata, cluster, and weight variables.

The univariate associations between the variables were examined with Pearson chi-square test and t-test. Weighted logistic regression was employed to explore the association of occupational lifetime exposure to very loud noise with COPD and emphysema; the prevalence risk was approximated by the OR. The multivariate models for COPD and emphysema were adjusted for gender, age, race/ethnicity, annual family income, cigarette-smoking status, smoking other tobacco products, alcohol-drinking status, light/moderate physical activity, vigorous physical activity, BMI, asthma, and for the major SOC group. This covariate set was determined a priori based on theoretical rationale and directed acyclic graphs using DAGitty v. 2.3 (http://dagitty.net/).

In sensitivity analysis, the sample was stratified by sociodemographics, co-morbidity, and work-related factors. Potential effect modifiers were tested using the Wald test to determine the overall statistical significance (at the P < 0.05 level) of their interactions with very loud noise exposure for ≥15 years (as a dummy variable with a reference category “never exposed”). Some of the covariates were re-categorized (categories were combined) when they were tested as potential effect modifiers.

In another sensitivity analysis, the model for COPD was restricted to participants who had never used hearing protection at work. In another scenario, it was restricted to participants aged ≥40 years because COPD mostly affects
Further, the model for COPD was re-run using the threshold noise variable as the noise exposure indicator. As a final sensitivity analysis, the multivariate logistic regression for COPD was repeated with CNE instead of self-reported very loud noise exposure. The adjustment set was the same, except that we dropped the major SOC group variable, because CNE was already based on SOC taxonomy. Results were considered statistically significant at $P < 0.05$.

Analyses were conducted with Statistical Package for the Social Sciences.

Figure 1: Analysis of potential effect modifiers of the association of occupational lifetime exposure to very loud for $\geq 15$ years and prevalent chronic obstructive pulmonary disease (COPD). The scale of the graph is logarithmic. P-values stand for statistical significance of interaction terms. Models are based on weighted logistic regression to obtain nationally representative estimates. Models are adjusted for gender, age, race/ethnicity (white, black/African American, American Indian/Chinese/Filipino/Asian Indian, and multiple race/other), annual family income ($<$ $35,000, $35,000–$74,999, $75,000–$99,999, and $\geq$ $100,000), cigarette-smoking status (never smoker, former smoker, current some day smoker, and current every day smoker), smoking other tobacco products, alcohol-drinking status (lifetime abstainer, former drinker, and current drinker), light/moderate physical activity (never, unable to do it, $< 3$ times/week, $4–7$ times/week, and $> 7$ times/week), vigorous physical activity (never, unable to do it, $< 3$ times/week, $4–7$ times/week, and $> 7$ times/week), body mass index ($< 25$ kg/m², $25.00–29.99$ kg/m², and $\geq 30.00$ kg/m²), asthma and Standard Occupational Classification major occupation group (unless stratified by the respective factor). Some of the covariates are re-categorized when they were tested as effect modifiers. Data source: CDC/NCHS, National Health Interview Survey, 2014.
Table 1: Participants’ characteristics according to their self-reported chronic obstructive pulmonary disease (COPD) status

| Characteristic                                      | No (n = 35,284, 96.9%) | Yes (n = 1371, 3.1%) | P-value |
|-----------------------------------------------------|------------------------|----------------------|---------|
| **Sociodemographics**                               |                        |                      |         |
| Men (n, %)                                          | 15,778 (48.3)          | 593 (44.7)           | 0.060   |
| Age (mean, SE)                                      | 46.44 (0.17)           | 63.31 (0.56)         | <0.001  |
| Age ≥ 40 years (n, %)                               | 22,041 (59.1)          | 1312 (94.6)          | <0.001  |
| **Race/ethnicity (n, %)**                           |                        |                      |         |
| White                                               | 25,367 (74.6)          | 1139 (87.5)          |         |
| Black/African American                              | 5004 (12.4)            | 144 (7.4)            |         |
| American Indian/Chinese/Filipino/Asian Indian       | 1725 (4.6)             | 39 (2.4)             |         |
| Multiple race/other                                 | 3188 (8.3)             | 49 (2.7)             |         |
| **Annual family income (n, %)**                     |                        |                      | <0.001  |
| <$35,000                                            | 13,340 (31.4)          | 800 (54.8)           |         |
| $35,000–74,999                                      | 9758 (26.7)            | 323 (31.8)           |         |
| $75,000–99,999                                      | 3456 (12.7)            | 67 (5.5)             |         |
| ≥$100,000                                           | 5880 (25.2)            | 68 (7.8)             |         |
| **Lifestyle**                                       |                        |                      | <0.001  |
| Cigarette-smoking status (n, %)                     |                        |                      |         |
| Never smoker                                        | 21,550 (62.6)          | 275 (21.0)           |         |
| Former smoker                                       | 7654 (21.2)            | 615 (43.8)           |         |
| Current some day smoker                             | 1446 (3.9)             | 71 (4.4)             |         |
| Current every day smoker                            | 4454 (12.3)            | 398 (30.8)           |         |
| Ever smoked other tobacco products (n, %)           | 7754 (22.7)            | 356 (25.9)           |         |
| **Alcohol-drinking status (n, %)**                  |                        |                      | <0.001  |
| Lifetime abstainer                                  | 7404 (21.4)            | 223 (16.0)           |         |
| Former drinker                                      | 5099 (12.8)            | 477 (36.5)           |         |
| Current drinker                                     | 22,248 (65.8)          | 649 (47.4)           |         |
| Light/moderate physical activity (n, %)             |                        |                      | <0.001  |
| Never                                               | 13,472 (38.5)          | 744 (56.8)           |         |
| Unable to do it                                     | 404 (0.9)              | 92 (6.8)             |         |
| <3 times/week                                       | 11,339 (33.8)          | 263 (18.8)           |         |
| 4–7 times/week                                      | 8327 (23.6)            | 201 (13.9)           |         |
| >7 times/week                                       | 1112 (3.3)             | 40 (3.7)             |         |
| Vigorous physical activity (n, %)                   |                        |                      | <0.001  |
| Never                                               | 18,522 (51.2)          | 1012 (74.4)          |         |
| Unable to do it                                     | 615 (1.8)              | 116 (8.6)            |         |
| <3 times/week                                       | 10,549 (31.9)          | 146 (11.0)           |         |
| 4–7 times/week                                      | 4534 (13.6)            | 70 (5.1)             |         |
| >7 times/week                                       | 645 (2.0)              | 14 (0.9)             |         |
| Body mass index (n, %)                              |                        |                      | <0.001  |
| <25.00 kg/m²                                        | 12,147 (36.3)          | 444 (30.9)           |         |
| 25.00–29.99 kg/m²                                   | 11,789 (34.6)          | 387 (30.1)           |         |
| ≥30.00 kg/m²                                        | 10,076 (29.1)          | 493 (37.6)           |         |
| **Co-morbidity**                                    |                        |                      | <0.001  |
| Asthma (n, %)                                       | 4213 (12.0)            | 549 (37.3)           |         |
| Emphysema (n, %)                                    | 230 (0.5)              | 446 (29.9)           |         |
| Hearing (n, %)                                      | 17,194 (51.5)          | 295 (21.9)           |         |
| Excellent hearing                                   | 15,689 (42.9)          | 775 (57.3)           |         |
| Good/little trouble hearing                         | 2295 (6.5)             | 294 (20.8)           |         |
| Moderate trouble/lot of trouble hearing             |                        |                      |         |
| **Occupation**                                      |                        |                      | <0.001  |
| White-collar workers                                | 19,031 (60.3)          | 588 (47.3)           |         |
| Service workers                                     | 6155 (18.2)            | 259 (19.7)           |         |
| Blue-collar workers                                 | 7014 (21.4)            | 407 (33.0)           |         |
| Exposure to very loud noise (n, %)                  |                        |                      | <0.001  |

(Continued')
RESULTS

Table 1 reports participants’ characteristics by their COPD status. Overall, 3.1% had self-reported COPD. In comparison to those without COPD, they were significantly older, 94.6% were 40 years old or older. COPD cases were more often white, with low income, former smokers, and alcohol-drinkers; they also exercised less often and were more likely to be obese. In terms of co-morbidity, participant with COPD reported asthma, emphysema, and hearing impairment more often. They were also more often blue-collar workers and had been exposed to very loud noise longer.

Table 2 shows the association between occupational noise exposure and other factors.

Next, we looked at the association of very loud noise exposure with COPD. First, we estimated gender and age-adjusted effects. In comparison to never exposed participants, exposed participants had OR_{<5\ years} = 2.04 (95% CI: 1.54, 2.72), OR_{5–9\ years} = 1.95 (95% CI: 1.33, 2.85), OR_{10–14\ years} = 2.21 (95% CI: 1.47, 3.33), and OR_{15\ years} = 2.75 (95% CI: 2.20, 3.43). For emphysema, the gender and age-adjusted model yielded OR_{<5\ years} = 1.66 (95% CI: 1.07, 2.57), OR_{5–9\ years} = 1.73 (95% CI: 0.89, 3.37), OR_{10–14\ years} = 1.93 (95% CI: 1.17, 3.17), and OR_{15\ years} = 2.92 (95% CI: 2.18, 3.91).

Table 3 gives results from the fully adjusted multivariate logistic regressions. The additional adjustments decreased the magnitude of the effect. For COPD, it reached statistical significance, OR_{15\ years} = 1.68 (95% CI: 1.28, 2.21). For emphysema, the only significant risk above 1.00 was OR_{15\ years} = 1.61 (95% CI: 1.13, 2.30).

To explore the dataset for significant effect modifiers, we stratified the multivariate model for COPD by participants’ characteristics. From Figure 1, the only significant effect modifier at the \( P < 0.05 \) level was race/ethnicity. The OR was statistically significant among whites (OR = 1.80, 95% CI: 1.34, 2.41); among blacks/African Americans it was still elevated, but not significant (OR = 1.72, 95% CI: 0.77, 3.84); whereas among other minorities, there was no elevated risk (OR = 0.15, 95% CI: 0.03, 0.63).

As sensitivity analysis, we restricted the multivariate model for COPD to participants who had never used hearing protection for very loud noise at work, which yielded OR_{15\ years} = 1.97 (95% CI: 1.42, 2.73). Restricting the model for COPD to participants aged \( \geq 40 \) years yielded OR_{15\ years} = 1.56 (95% CI: 1.19, 2.05).

In another sensitivity analysis, the multivariate model for COPD was re-run using the threshold self-reported noise variable as noise exposure indicator [Figure 2]. In comparison to never exposed participants, those who had ever been exposed had OR_{loud\ noise} = 1.24 (95% CI: 0.77, 2.00) and OR_{very\ loud\ noise} = 1.53 (95% CI: 1.22, 1.91).

The final sensitivity analysis, using CNE as an exposure variable, yielded the following association with self-reported COPD (adjustments were the same as those in the multivariate model shown in Table 3, minus the major...
Table 2: Participants’ characteristics according to their occupational lifetime exposure to very loud noise

| Duration of exposure | Never exposed | <5 years | 5–9 years | 10–14 years | ≥15 years | P-value |
|----------------------|---------------|----------|-----------|-------------|-----------|---------|
| Sociodemographics    |               |          |           |             |           |         |
| Men (%)              | 41.59         | 70.88    | 72.45     | 74.66       | 84.09     | <0.001  |
| Age (mean, SE)       | 46.83 (0.18)  | 42.02 (0.50) | 45.50 (0.63) | 50.08 (0.59) | 59.11 (0.38) | <0.001  |
| Age ≥40 years (%)    | 59.96         | 47.71    | 56.60     | 69.31       | 92.10     | <0.001  |
| Race/ethnicity (%)   |               |          |           |             |           | <0.001  |
| White                | 75.27         | 75.81    | 76.45     | 74.93       | 85.66     |         |
| Black/African American| 12.04        | 13.81    | 11.92     | 14.19       | 8.57      |         |
| American Indian/Chinese/Filipino/Asian Indian | 4.77 | 2.87 | 3.74 | 1.80 | 1.74 | |
| Multiple race/other  | 7.92          | 7.51     | 7.89      | 9.08        | 4.03      |         |
| Annual family income (%) |         |          |           |             |           | <0.001  |
| <$35,000             | 29.84         | 35.70    | 36.18     | 34.30       | 31.22     |         |
| $35,000–74,999       | 29.90         | 30.94    | 36.14     | 35.44       | 38.32     |         |
| $75,000–99,999       | 12.96         | 11.61    | 13.01     | 12.59       | 13.24     |         |
| ≥$100,000            | 27.31         | 21.75    | 14.67     | 17.68       | 17.22     |         |
| Lifestyle            |               |          |           |             |           | <0.001  |
| Cigarette-smoking status (%) |       |          |           |             |           |         |
| Never smoker         | 64.48         | 48.29    | 42.45     | 44.77       | 40.66     |         |
| Former smoker        | 20.69         | 24.18    | 27.58     | 29.49       | 37.93     |         |
| Current some day smoker | 3.61        | 6.83     | 5.42      | 3.36        | 3.56      |         |
| Current every day smoker | 11.22       | 20.70    | 24.55     | 22.39       | 17.85     |         |
| Ever smoked other tobacco products (%) | 19.64 | 40.80 | 38.37 | 34.60 | 36.20 | <0.001 |
| Alcohol-drinking status (%) |        |          |           |             |           | <0.001  |
| Lifetime abstainer   | 20.88         | 10.71    | 8.98      | 13.65       | 10.71     |         |
| Former drinker       | 12.56         | 14.17    | 14.05     | 18.61       | 23.83     |         |
| Current drinker      | 66.56         | 75.12    | 76.97     | 67.74       | 65.46     |         |
| Light/moderate physical activity (%) |        |          |           |             |           | <0.001  |
| Never                | 38.24         | 33.21    | 40.48     | 38.64       | 41.74     |         |
| Unable to do it      | 0.83          | 0.68     | 2.03      | 1.79        | 2.26      |         |
| <3 times/week        | 34.41         | 36.28    | 33.68     | 32.07       | 27.83     |         |
| 4–7 times/week       | 23.31         | 26.16    | 20.03     | 23.77       | 24.18     |         |
| >7 times/week        | 3.21          | 3.68     | 3.78      | 3.72        | 3.99      |         |
| Vigorous physical activity (%) |         |          |           |             |           | <0.001  |
| Never                | 50.90         | 45.38    | 50.77     | 53.79       | 59.56     |         |
| Unable to do it      | 1.29          | 1.28     | 2.75      | 2.78        | 2.82      |         |
| <3 times/week        | 32.69         | 35.57    | 30.05     | 28.48       | 22.68     |         |
| 4–7 times/week       | 13.11         | 15.41    | 14.23     | 11.28       | 13.85     |         |
| >7 times/week        | 2.01          | 2.36     | 2.20      | 3.67        | 1.09      |         |
| Body mass index (%)  |               |          |           |             |           | <0.001  |
| <25.00 kg/m²         | 37.98         | 29.94    | 27.42     | 23.32       | 22.37     |         |
| 25.00–29.99 kg/m²    | 34.23         | 37.30    | 38.42     | 36.83       | 38.43     |         |
| ≥30.00 kg/m²         | 27.79         | 32.75    | 34.16     | 39.85       | 39.21     |         |
| Co-morbidity         |               |          |           |             |           |         |
| Asthma (%)           | 12.56         | 15.90    | 14.07     | 13.27       | 12.71     | 0.005   |
| Emphysema (%)        | 1.12          | 1.27     | 1.45      | 2.07        | 4.49      | <0.001  |
| Hearing (%)          |               |          |           |             |           | <0.001  |
| Excellent hearing    | 54.80         | 42.66    | 33.99     | 33.20       | 20.62     |         |
| Good/little trouble hearing | 41.07 | 49.89 | 55.41 | 55.20 | 57.70 | |
| Moderate trouble/lot of trouble hearing | 4.13 | 7.45 | 10.59 | 11.60 | 21.68 | |
| Occupation           |               |          |           |             |           | <0.001  |
| Occupational group (%) |           |          |           |             |           | <0.001  |
| White-collar workers | 66.12         | 46.32    | 31.52     | 33.35       | 30.64     |         |
| Service workers      | 18.70         | 20.40    | 18.45     | 15.43       | 11.69     |         |
| Blue-collar workers  | 15.17         | 33.27    | 50.03     | 51.22       | 57.66     |         |
| Hearing protection use (%) |         |          |           |             |           | <0.001  |

(Continued)
SOC group): <85 dB(A)-year (reference), 85–90 dB(A)-year (OR = 1.04, 95% CI: 0.75, 1.43), 90–95 dB(A)-year (OR = 1.11, 95% CI: 0.82, 1.51), and >95 dB(A)-year (OR = 1.01, 95% CI: 0.74, 1.39); and for self-reported emphysema: 85–90 dB(A)-year (OR = 1.13, 95% CI: 0.72, 1.76), 90–95 dB(A)-year (OR = 0.92, 95% CI: 0.60, 1.41), and >95 dB(A)-year (OR = 0.85, 95% CI: 0.57, 1.25).

When we used CNE in the multivariate model, there was no effect on self-reported COPD and emphysema; this could be due to one of two reasons: (1) some exposure misclassification, because we used group-level noise data, or (2) a true null effect. Overall, there is limited evidence to suggest an association between occupational noise and COPD, which calls for further research into this area.

Nevertheless, several pathways are surmised to mediate the alleged effect of noise on the respiratory system; taken together, they are a plausible explanation for the significant associations observed in this study. One of these pathways is neuroimmunological. It involves bronchomotor tone dysregulation, airway inflammation, and increased susceptibility to infection. Cardoso et al. [12] examined the lung function of 28 women working for more than 10 years in the textile industry (versus 30 controls) and evidenced “small airways aggression by noise,” although residual confounding could not be ruled-out. Few other studies reported an effect of environmental noise on respiratory health. Niemann et al. [9] observed significantly higher risk of doctor-diagnosed bronchitis in adults and children with severe noise annoyance; Ising et al. [11] associated nighttime traffic noise with aggravation of bronchitis in children; and Tobías et al. linked traffic noise to respiratory disease admissions [27] and mortality [10] in

| Duration of exposure | COPD (n = 29,867) | Emphysema (n = 29,882) |
|----------------------|------------------|-----------------------|
|                      | Cases (n)        | OR (95% CI)           | Cases (n)        | OR (95% CI)           |
| Never exposed        | 797              | 1.00                  | 386              | 1.00                  |
| <5 years             | 109              | 1.42 (0.98, 2.04)     | 51               | 0.83 (0.50, 1.40)     |
| 5–9 years            | 54               | 1.18 (0.74, 1.87)     | 23               | 0.89 (0.44, 1.80)     |
| 10–14 years          | 54               | 1.20 (0.75, 1.94)     | 24               | 0.93 (0.50, 1.72)     |
| ≥15 years            | 231              | 1.68 (1.28, 2.21)     | 137              | 1.61 (1.13, 2.30)     |

DisProportionately significant associations observed in this study. One of these pathways is neuroimmunological. It involves bronchomotor tone dysregulation, airway inflammation, and increased susceptibility to infection. Cardoso et al. [12] examined the lung function of 28 women working for more than 10 years in the textile industry (versus 30 controls) and evidenced “small airways aggression by noise,” although residual confounding could not be ruled-out. Few other studies reported an effect of environmental noise on respiratory health. Niemann et al. [9] observed significantly higher risk of doctor-diagnosed bronchitis in adults and children with severe noise annoyance; Ising et al. [11] associated nighttime traffic noise with aggravation of bronchitis in children; and Tobías et al. linked traffic noise to respiratory disease admissions [27] and mortality [10] in

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Madrid. Recio et al.\textsuperscript{[28]} also reported a higher risk of COPD mortality due to traffic noise in Madrid.

However, no study focused on the relationship occupational noise – COPD; therefore, we have to make an analogy with studies on psychosocial stress and COPD, since noise and psychosocial stress are hypothesized to share common biological mechanisms. One example is the study of Pembroke et al. who followed nearly 4000 participants for 3 years and found that psychological distress, measured with the General Health Questionnaire, was associated with an elevated risk of chronic bronchitis and forced expiratory volume in 1 s (FEV1) ≤ 75% in both men (OR = 1.49, 95% CI: 0.83, 2.71 and OR = 2.00, 95% CI: 1.16, 3.46, respectively) and women (OR = 2.00, 95% CI: 1.16, 3.46 and OR = 1.62, 95% CI: 1.13, 2.32, respectively).\textsuperscript{[15]} In another 16-year cohort study, including 8728 participants, Clark et al.\textsuperscript{[16]} found that accumulated psychosocial risk factors (adverse life events, adverse aspects of social network, economic hardship) were associated with incident COPD – hazard ratio (HR) = 1.93 (95% CI: 1.33, 2.80) in men and HR = 2.40 (95% CI: 1.78, 3.22) in women. The described pathophysiological underpinnings might also explain the increased risk of exacerbations among patients with COPD suffering from anxiety/depression,\textsuperscript{[29]} and the risk of asthma due to psychological stress.\textsuperscript{[30]}

We can also speculate on another indirect pathway. Occupational noise has been associated with increased levels of smoking,\textsuperscript{[31,32]} which is the major risk factor for COPD.\textsuperscript{[5]} Likewise, psychosocial stress might increase the risk of COPD through increased smoking behavior.\textsuperscript{[33]} Moreover, according to one study, people’s noise sensitivity was associated with smoking behavior and self-reported emphysema independently from noise itself – those with emphysema and former smokers had OR = 4.43 (95% CI: 1.51, 12.99) and OR = 1.48 (95% CI: 1.02, 2.15), respectively, for high noise sensitivity.\textsuperscript{[34]}

Strengths and limitations

One of the main strengths of this study is its large nationally representative sample, along with the fact that it was drawn from the general population. Nevertheless, there are several limitations.

First, a cross-sectional study cannot disentangle the causality between noise exposure and COPD. Given that there are no previous epidemiological studies to ascertain their association, we cannot rule out the possibility of it being spurious. In favor of our results, however, we used information on occupational lifetime exposure, meaning that for people exposed ≥15 years the exposure most likely preceded the diagnosis with COPD; further, it is not very likely that once diagnosed with COPD, people would move into a noisy job. In fact, is much more likely for COPD cases to have left a noisy job, which would have flattened the association with noise. Healthy worker/survivor effect cannot be ruled-out, and such an effect is expected to bias the results toward the null, making them conservative.

Second, self-reported diagnosis underestimates the true prevalence of COPD.\textsuperscript{[35]} While objective spirometric examinations of lung function in the NHANES 2007–2010 yielded 10.2–20.9% prevalence of COPD,\textsuperscript{[32]} the prevalence of COPD, based on self-reported physician diagnoses of emphysema and chronic bronchitis in the NHIS 2004–2011, was 4.2%.\textsuperscript{[26]} However, “in analytical epidemiological studies, especially when the aim is to evaluate a risk factor, it is preferable to have a test/question with very high specificity and lower sensitivity, to avoid false positive findings and, consequently, bias in risk estimates.”\textsuperscript{[35,36]} Thus, our results are probably biased toward the null, suggesting that the lacking effect in some models might actually be due to the definition of COPD.

Reassuringly, self-reported exposure to very loud noise in the workplace has been found to be a good indicator for exposure to >80–85 dB, when cross-validated with objectively measured noise via dosimetry or sound level meters.\textsuperscript{[19–25]} Nevertheless, we also used a JEM for noise to assign CNE. The measurements included in the JEM covered the period 1963–2015 and were averaged at the 4-digit SOC level which leads to exposure misclassification.

Third, in NHIS 2014, we had no information on occupational exposure to smoke, fumes, dust, etc., which are known risk factors for COPD.\textsuperscript{[6]} To account for this and other occupational factors, we adjusted the multivariate models for the 23 SOC major occupation groups in the NHIS 2014. Notwithstanding, we cannot rule out the possibility of noise being highly correlated with other occupational exposures and reflecting some of their effect on COPD.

Conclusions

In conclusion, we found a relationship between self-reported occupational noise exposure and the risk of prevalent COPD in the US general population, but none with objective noise levels. These results warrant further research. Being the first study on occupational noise and COPD, and given the design limitations, these findings are tentative and should be treated with caution.

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Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Pauwels RA, Buist AS, Calverley PM, Jenkins CR, Hurd SS. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. NHLBI/WHO Global Initiative for Chronic Obstructive Lung Disease (GOLD) Workshop summary. Am J Respir Crit Care Med 2001;163:1256-76.

2. Tillet T, Dillon C, Paulose-Ram R, Hnizdo E, Doney B. Estimating the U.S. prevalence of chronic obstructive pulmonary disease using pre- and post-bronchodilator spirometry. The National Health and Nutrition Examination Survey (NHANES) 2007–2010. Respir Res 2013;14:103.

3. Murray CJ, Atkinson C, Bhalla K, Birbeck G, Burstein R, Chou D, et al. The state of US health, 1990-2010: Burden of diseases, injuries, and risk factors. JAMA 2013;310:591-608.

4. Guarascio AJ, Ray SM, Finch CK, Selh TH. The clinical and economic burden of chronic obstructive pulmonary disease in the USA. Clinicoecon Outcomes Res 2013;5:235-45.

5. Eisner MD, Anthonisen N, Coultas D, Kuenzli N, Perez-Padilla R, Postma D, et al. An official American Thoracic Society public policy statement: Novel risk factors and the global burden of chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2010;182:693-718.

6. Ryu JY, Sunwoo YE, Lee SY, Lee CK, Kim JH, Lee JT, et al. Chronic obstructive pulmonary disease (COPD) and vapors, gases, dusts, or fumes (VGDF): A meta-analysis. COPD 2015;12:374–80.

7. Recio A, Linares C, Banegas JR, Díaz J. Road traffic noise effects on cardiovascular, respiratory, and metabolic health: An integrative model of biological mechanisms. Environ Res 2016;146:359-70.

8. Barnes PJ. Cellular and molecular mechanisms of chronic obstructive pulmonary disease. Clin Chest Med 2014;35:71-86.

9. Niemann H, Bonnefoy X, Braubach M, Hecht K, Maschke C, Rodrigues C, et al. Noise-induced annoyance and morbidity results from the pan-European LARES study. Noise Health 2006;8:63-79.

10. Tobias A, Recio A, Díaz J, Linares C. Does traffic noise influence respiratory mortality? Eur Respir J 2014;44:797-9.

11. Ising H, Lange-Asschenfeldt H, Moriske HJ, Born J, Eilts M. Low frequency noise and stress: Bronchitis and cortisol in children exposed chronically to traffic noise and exhaust fumes. Noise Health 2004;6:21-8.

12. Cardoso AP, Oliveira MJ, Silva AM, Aguas AP, Pereira AS. Effects of long term exposure to occupational noise on textile industry worker’s lung function. Rev Port Pneumol 2006;12:45-59.

13. Hynynen KM, Breitve MH, Wiborg AB, Pallesen S, Nordhus IH. Psychological characteristics of patients with chronic obstructive pulmonary disease: A review. J Psychosom Res 2005;59:429-43.

14. Andenaes R, Kalfoss MH. Psychological distress in hospitalized patients with chronic obstructive pulmonary disease. Eur J Epidemiol 2004;19:851-9.

15. Pembroke TP, Rasul F, Hart CL, Davey Smith G, Stansfeld SA. Psychological distress and chronic obstructive pulmonary disease in the Renfrew and Paisley (MIDSPAN) study. J Epidemiol Community Health 2006;60:789-92.

16. Clark AJ, Strandberg-Larsen K, Masters Pedersen JL, Lange P, Prescott E, Rod NH. Psychosocial risk factors for hospitalisation and death from chronic obstructive pulmonary disease: A prospective cohort study. COPD 2015;12:190-8.

17. Masterson EA, Tak S, Themann CL, Wall DK, Groenewold MR, Deddens JA, et al. Prevalence of hearing loss in the United States by industry. Am J Ind Med 2013;56:670-81.

18. National Center for Health Statistics. Survey Description, National Health Interview Survey, 2014. Hyattsville, Maryland: 2015.

19. Ahmed HO, Dennis JH, Ballal SG. The accuracy of self-reported high noise exposure level and hearing loss in a working population in Eastern Saudi Arabia. Int J Hyg Environ Health 2004;207:227-34.

20. Neitzel RL, Daniell WE, Sheppard L, Davies HW, Seixas NS. Evaluation and comparison of three exposure assessment techniques. J Occup Env Hyg 2011;8:310-23.

21. Neitzel RL, Svensson EB, Sayler SK, Ann-Chrisitin J. A comparison of occupational and nonoccupational noise exposures in Sweden. Noise Health 2014;16:270-8.

22. Neitzel RL, Andersson M, Andersson E. Comparison of multiple measures of noise exposure in paper mills. Ann Occup Hyg 2016;60:581-96. pii: mew001.

23. Schlaefer K, Schlehofer B, Schuz J. Validity of self-reported occupational noise exposure. Eur J Epideimiol 2009;24:469-75.

24. University of Michigan Department of Environmental Health Sciences. A US/Canada Job Exposure Matrix for Noise. Available from: http://noisejem.org/ [Last accessed on 2016 Aug].

25. Song C. Occupational noise exposure and the risk of diabetes, rheumatoid arthritis, and cardiovascular disease [Master thesis]. Vancouver: The University of British Columbia; 2013. Available from: http://www.rdc-cdr.ca/occupational-noise-exposure-and-risk-diabetes-rheumatoid-arthritis-and-cardiovascular-disease. [Last accessed on 2016 Apr].

26. Doney B, Hnizdo E, Sanyamlal G, Kullman G, Burchfield C, Martin CJ, et al. Prevalence of chronic obstructive pulmonary disease among US working adults aged 40 to 70 years. National Health Interview Survey data 2004 to 2011. J Occup Environ Med 2014;56:1088-93.

27. Tobias A, Diaz J, Saee M, Alberdi JC. Use of poisson regression and box-jenkins models to evaluate the short-term effects of environmental noise levels on daily emergency admissions in Madrid, Spain. Eur J Epidemiol 2001;17:765-71.

28. Recio A, Linares C, Banegas JR, Diaz J. The short-term association of road traffic noise with cardiovascular, respiratory, and diabetes-related mortality. Environ Res 2016;150:383-90.

29. Laurin C, Moullec G, Bacon SL, Lavoie KL. Impact of anxiety and depression on chronic obstructive pulmonary disease exacerbation risk. Am J Respir Crit Care Med 2012;185:918-23.

30. Wright RJ, Rodriguez M, Cohen S. Review of psychosocial stress and asthma: An integrated biopsychosocial approach. Thorax 1998;53:1066-74.

31. Cherek DR. Effects of acute exposure to increased levels of background industrial noise on cigarette smoking behavior. Int Arch Occup Environ Health 1985;56:23-30.

32. Kim YJ. Impact of work environments and occupational hazards on smoking intensity in Korean workers. Workplace Health Saf 2016;64:103-13.

33. Rod NH, Grombaek M, Schnoor P, Prescott E, Kristensen TS. Perceived stress as a risk factor for changes in health behaviour and cardiac risk profile: A longitudinal study. J Intern Med 2009;266:467-75.

34. Heinonen-Guzejev M, Vuorinen HS, Mussalo-Rauhamaa H, Heikklä K, Koskenvuo M, Kaprio J. Somatic and psychological characteristics of noise-sensitive adults in Finland. Arch Environ Health 2004;59:410-7.

35. Murgia N, Brisman J, Claesson A, Muzi G, Olin AC, Torén K. Validity of a questionnaire-based diagnosis of chronic obstructive pulmonary disease in a general population-based study. BMC Pulm Med 2014;14:49. doi: 10.1186/1471-2466-14-49.

36. Copeland KT, Checkoway H, McMichael AJ, Holbrook RH. Bias due to misclassification in the estimation of relative risk. Am J Epidemiol 1977;105:488-95.