Original Article Noise exposure could increase the mortality of coronary heart disease: evidence from a meta-analysis

Wei Miao¹, Lei Wang¹, Ying Cui¹, Feng Zhang^{1,2}, Shuya Wang^{1,2}, Nan Liu^{1,2}, Guohai Su¹, Xiaojun Cai^{1,2}

¹Department of Cardiology, Jinan Central Hospital Affiliated to Shandong University, Jinan, China; ²Shandong University College of Medicine, Jinan, China

Received December 31, 2015; Accepted March 22, 2016; Epub June 15, 2016; Published June 30, 2016

Abstract: Purpose: Quantification of the association between noise exposure and risk of coronary heart disease (CHD) mortality is still conflicting. Thus, we conducted a meta-analysis summarizing the evidence from epidemiological studies to assess the association between them. Methods: Pertinent studies were identified by a search of PubMed and Web of Knowledge up to October 2015. The random effect model was used. Sensitivity analysis, subgroup analyses and publication bias were conducted. Results: Eight studies (6 cohort studies and 2 case-control studies) involving 23,749 CHD deaths were used in this meta-analysis. Pooled results suggested that highest noise exposure level versus lowest level was significantly associated with the increased risk of CHD deaths [summary RR=1.21, 95% CI=1.05-1.40, I²=37.5%], especially in European populations [summary RR=1.14, 95% CI=1.01-1.30]. The associations were also found in the cohort studies and in the subgroup analysis of the method to assess noise by modeling. Conclusions: Our analysis suggested that highest levels of noise exposure might increase the risk of CHD deaths, especially in European populations. As most of the current studies were from the Europe, additional studies are needed from other geographic locations and any further studies should adjust for more confounding factors.

Keywords: Noise, coronary heart disease, mortality, meta-analysis

Introduction

Epidemiologic studies have demonstrated that air pollution is associated with increased coronary heart disease (CHD) morbidity and mortality [1]. Meanwhile, accumulating evidence has suggested that community noise from road and air traffic is associated with an increased risk of CHD, especially on myocardial infarction (MI) [2-4]. In metropolitan areas, road traffic is a major contributor to ambient air pollution and the dominant source of community noise [5, 6]. Persons exposed to higher levels of air pollution might also be exposed to excessive traffic noise [5-7]. Therefore, it is possible that the observed associations between air pollution and adverse CHD outcomes disease and deaths could be confounded by community noise and vice versa [6]. Up to date, epidemiologic studies reporting the effect of noise exposure on CHD deaths risk conveyed conflicting results. Furthermore, due to small sample sizes, most studies were not adequately powered to detect the effect of noise exposure on CHD deaths risk. Thus, in order to provide the latest and more convincing evidence, we systematically reviewed the current available epidemiologic studies to investigate whether noise exposure was associated with increased CHD deaths risk.

Materials and methods

Literature search and study selection

PubMed and Web of Science were searched from their inception to October, 2015 by using a combination of Medical Subject Headings and related common keywords were used including "noise" AND "coronary heart disease" OR "CHD" OR "ischemic heart disease" OR "myocardial infarction" OR "coronary artery disease" AND "mortality" OR "death". No language restriction was applied. The search strategy is shown in **Figure 1**.

Studies meeting the following inclusion criteria were included: observational studies (prospective or retrospective cohort studies or casecontrol studies); investigated the association

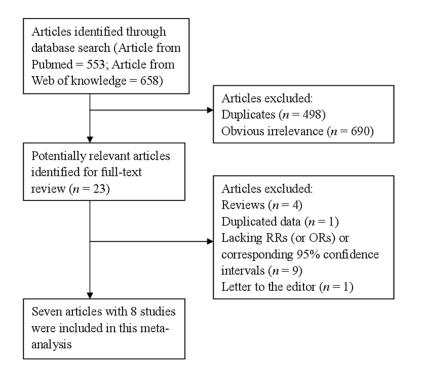


Figure 1. Flowchart of meta-analysis for exclusion/inclusion of studies.

between noise and the mortality of CHD; reported adjusted odds ratios (ORs) or relative risks (RRs) and its 95% confidence intervals (Cls). If articles were from the same study population, the largest study was included to avoid duplication of information.

Data extraction

Relevant data from each included study were extracted from each study: the first author's name, country, study design, no. of cases/controls, mean or median age, ethnicity, duration of follow-up, methods to assess noise, mostadjusted ORs or RRs with 95% Cls, and matched or adjusted factors in the design or data analysis. Since study-specific data can be obtained from original articles, no authors were contacted.

Statistical methods

We calculated summary RRs of highest level compared with the lowest level of noise exposure for the risk of CHD mortality using the random-effects model by DerSimonian and Laird [8], which takes into account both within and between study heterogeneity. Heterogeneity between studies was evaluated using Q test and l² statistics [9]. l² is a measure of how much of the heterogeneity that is due to between study variation rather than chance. I2values of 25%, 50% and 75% indicates low, moderate and high heterogeneity respectively. We conducted main analyses (all studies combined) and stratified by study characteristics such as study design, sex, geographic location and methods to assess noise. Sensitivity analysis was conducted to describe how robust the pooled estimator is to removal of individual studies. Publication bias was evaluated by means of Egger's regression test [10]. The statistical analyses were conducted using the software package Stata, version 10.1 software (StataCorp, Texas, US).

Results

Study identification and selection

Our systematic literature search yielded a total of 8 studies [3, 11-16] on noise exposure and CHD mortality risk in the final analysis. There are 1211 records were identified by searching the databases and hand-searching relevant bibliographies. Four hundred and ninety-eight records were excluded for duplicates and an additional 690 records were excluded based on the titles and abstracts. The remaining 23 full-text articles were assessed for eligibility, and 15 were further excluded due to the reasons present in **Figure 1**. The characteristics of these studies are presented in **Table 1**.

Highest versus lowest category

Data from 8 studies including 23749 CHD deaths were used in this meta-analysis. Two studies reported that highest noise exposure could increase the risk of CHD deaths, while no significant association was reported in 6 studies. Pooled results suggested that highest noise exposure level versus lowest level was significantly associated with the increased risk of CHD deaths [summary RR=1.21, 95% CI=1.05-1.40, I²=37.5%] (Figure 2).

Author, year	Country (year)	Age	Study design	Follow-up (years)	Participants (deaths)	Methods to assess noise	RR (95% CI)	Adjustment for covariates
Davies et al. 2005	Canada	30	Cohort	24	27464 (81)	Modeling	4.00 (1.80, 9.30)	Age, calendar year, and South Asian ethnicity
Mc Namee et al. 2006	England	37	Nested case-control	48	2202 (1101)	Modeling	1.13 (0.92, 1.39)	Systolic and diastolic BP, BMI, smoking, height and dura- tion of employment
Heinonen-Guzejev et al. 2007	Finland	31-88	Cohort	15	688 (111)	Questionnaire	Male: 1.52 (0.73, 3.18)	Age, noise sensitivity, hypertension, smoking and emphy-
							Female: 1.08 (0.48, 2.44)	sema
Beelen et al. 2009	Netherlands	55-69	Case-cohort	9	117528 (3521)	Modeling	1.15 (0.86, 1.53)	Age, sex, smoking status and area level indicators of socio-economic status
Huss et al. 2010	Switzerland	> 30	Cohort	5	4580331 (15532)	Modeling	1.30 (0.96, 1.76)	Sex and demographic, socioeconomic and geographical variables, air-pollution levels and distance to major roads
Gan et al. 2012	Canada	45-85	Cohort	4	445868 (3095)	Modeling	1.22 (1.04, 1.43)	Age, sex, preexisting comorbid conditions, and neighbor- hood SES were included as covariates and air pollution
Suadicani et al. 2012	Denmark	53-75	Cohort	16	2998 (197)	Questionnaire	0.97 (0.71, 1.33)	Age, hearing impairment, blood pressure, diabetes, fasting serum triglycerides and HDL cholesterol, LDL cholesterol, glucosuria, cancer, BMI, alcohol, tobacco, leisure-time physical activity, and social class

Table 1. Characteristics of the included studies

RR, Relative risk; CI, Confidence interval.

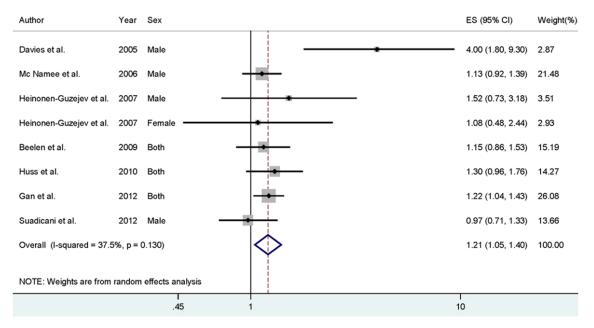


Figure 2. The forest plot of the relationship between noise exposure and risk of CHD mortality.

Sub-groups	Deaths	Studies	RR (95% CI)	l ² (%)	P					
	Beatho	Otdules	()		F heterogeneity					
All studies	23749	8	1.21 (1.05-1.40)	37.5	0.130					
Study design										
Case-control	4622	2	1.14 (0.96-1.34)	0.0	0.923					
Cohort	19127	6	1.29 (1.02-1.63)	53.2	0.058					
Sex										
Male	1490	4	1.36 (0.92-2.03)	71.6	0.014					
Both	22148	3	1.22 (1.08-1.38)	0.0	0.848					
Geographic locations										
North America	3176	2	2.06 (0.65-6.53)	87.1	0.005					
Europe	20573	6	1.14 (1.01-1.30)	0.0	0.799					
Methods to assess noise										
Modelling	23330	4	1.26 (1.05-1.51)	55.0	0.064					
Questionnaire	419	3	1.04 (0.80-1.37)	0.0	0.544					

 Table 2. Summary risk estimates of the association between

 noise exposure and CHD mortality risk

Table 2 reports the pooled RRs for noise exposure and CHD deaths in combined all results and selected subgroups. In the subgroup analyses, by study design, cohort studies showed an increased risk of noise exposure on CHD deaths with a RR of 1.29 (95% CI: 1.02-1.63), similar to the overall analysis. But, the association was not significant in the case-control studies. Regarding the type of geographic locations, the association was only significant in the European populations [summary RR=1.14, 95% CI=1.01-1.30], but not in the North American and other

populations else. When stratified by methods to assess noise, there was a statistically significant association in the studies assessed by modeling (summary RR=1.26, 95% CI= 1.05-1.51), but not in the studies assessed by questionnaire. Due to the limitation information of the reported studies on sex, we only combined the results for male, as no studies reported the association for female only. However, we did not find significant association in the male population.

In sensitivity analyses (**Figure 3**), no individual study had excessive influence on the asso-

ciation of noise exposure and CHD deaths risk. Egger's test (*P*=0.271) and Funnel plot (**Figure** 4) showed no evidence of significant publication bias between noise exposure and CHD deaths risk.

Discussion

The present meta-analysis identified 8 observational studies investigating noise exposure on CHD deaths risk. Our analysis showed that noise exposure was associated with a 21%

Noise exposure and CHD mortality

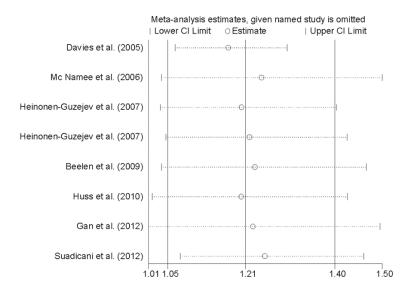


Figure 3. Sensitivity analysis between noise exposure and risk of CHD mortality.

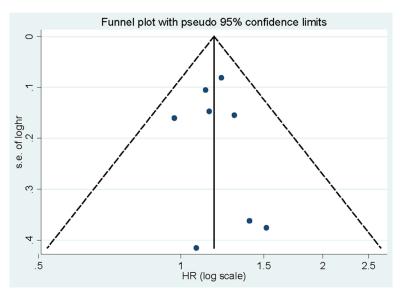


Figure 4. Funnel plot for the analysis of noise exposure and risk of CHD mortality.

increase in risk of CHD deaths when highest reported noise exposure was compared with lowest reported noise exposure. Moreover, this finding was consistent across sensitivity analyses and most subgroup analyses, and no publication bias was observed.

An association between noise and CHD is biologically plausible, and etiologic models of stress and heart disease have been proposed and extended to noise and heart disease [17]. Acute noise exposure activates the sympathetic nervous and neuroendocrine systems, causing the temporary increase of blood pressure and heart rate. Repeated and prolonged exposure to noise may lead to permanent effects, such as atherosclerosis, hypertension and ischemic heart disease [4]. In addition, exposure to high level of noise could increase the level of perceptive stress [18]. These promote the development of CHD and increase the CHD mortality.

As a meta-analysis of published studies, our findings showed some advantages. First, this is the first comprehensive meta-analysis of noise exposure and CHD deaths risk based on highest versus lowest analysis. Second, large number of participants was included, allowing a much greater possibility of reaching reasonable conclusions between noise exposure and CHD deaths risk. Third, no significant publication bias was found, indicating that our results are stable.

Some limitations in this meta-analysis should be concerned. First, a meta-analysis of observational studies is susceptible to potential bias inherent in the original studies, especially for case-con-

trol studies. Overstated association may be expected from the case-control studies because of recall or selection bias, and early symptoms in patients may have resulted in a change in dietary habits. In our meta-analysis, the significant association was only found in cohort studies, but not in the case-control studies, while only 2 studies included were casecontrol design. Second, the methods to assess noise are important, which can lead to overestimation of the range of noise and underestimation of the magnitude of the relationship between noise exposure and CHD deaths risk. In the present study, the association was only significant in the method of modeling, but not in the questionnaire. This may be caused by the bias of recall. Third, for the subgroups of geographic locations, the association was only significant in the European population, but not in the North America. Due to this limitation, the results are applicable to the Europe, but cannot be extended to populations elsewhere. More studies originating in other countries are required to investigate the association between noise exposure and CHD deaths risk.

In summary, results from this meta-analysis suggested that the highest levels of noise exposure might increase the risk of CHD deaths, especially in European populations. As most of the current studies were from the Europe, additional studies are needed from other geographic locations and any further studies should adjust for more confounding factors.

Acknowledgements

Shandong province natural science foundation of China (ZR2013HM003).

Disclosure of conflict of interest

None.

Address correspondence to: Dr. Xiaojun Cai, Department of Cardiology, Jinan Central Hospital Affiliated to Shandong University, 105 Jiefangroad, Jinan 250013, Shandong, China. E-mail: xjcai2016@ 163.com

References

- [1] Brook RD, Rajagopalan S, Pope CA 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F, Hong Y, Luepker RV, Mittleman MA, Peters A, Siscovick D, Smith SC Jr, Whitsel L, Kaufman JD; American Heart Association Council on E, Prevention CotKiCD, Council on Nutrition PA and Metabolism. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. Circulation 2010; 121: 2331-78.
- [2] Babisch W, Beule B, Schust M, Kersten N and Ising H. Traffic noise and risk of myocardial infarction. Epidemiology 2005; 16: 33-40.

- [3] Huss A, Spoerri A, Egger M, Roosli M; Swiss National Cohort Study G. Aircraft noise, air pollution, and mortality from myocardial infarction. Epidemiology 2010; 21: 829-36.
- [4] Babisch W. Cardiovascular effects of noise. Noise Health 2011; 13: 201-4.
- [5] Davies HW, Vlaanderen JJ, Henderson SB and Brauer M. Correlation between co-exposures to noise and air pollution from traffic sources. Occup Environ Med 2009; 66: 347-50.
- [6] Allen RW, Davies H, Cohen MA, Mallach G, Kaufman JD and Adar SD. The spatial relationship between traffic-generated air pollution and noise in 2 US cities. Environ Res 2009; 109: 334-42.
- [7] Foraster M, Deltell A, Basagana X, Medina-Ramon M, Aguilera I, Bouso L, Grau M, Phuleria HC, Rivera M, Slama R, Sunyer J, Targa J and Kunzli N. Local determinants of road traffic noise levels versus determinants of air pollution levels in a Mediterranean city. Environ Res 2011; 111: 177-83.
- [8] DerSimonian R and Laird N. Meta-analysis in clinical trials. Control Clin Trials 1986; 7: 177-88.
- [9] Higgins JP and Thompson SG. Quantifying heterogeneity in a meta-analysis. Stat Med 2002; 21: 1539-58.
- [10] Egger M, Davey Smith G, Schneider M and Minder C. Bias in meta-analysis detected by a simple, graphical test. BMJ 1997; 315: 629-34.
- [11] Davies HW, Teschke K, Kennedy SM, Hodgson MR, Hertzman C and Demers PA. Occupational exposure to noise and mortality from acute myocardial infarction. Epidemiology 2005; 16: 25-32.
- [12] McNamee R, Burgess G, Dippnall WM and Cherry N. Occupational noise exposure and ischaemic heart disease mortality. Occup Environ Med 2006; 63: 813-9.
- [13] Heinonen-Guzejev M, Vuorinen HS, Mussalo-Rauhamaa H, Heikkila K, Koskenvuo M and Kaprio J. The association of noise sensitivity with coronary heart and cardiovascular mortality among Finnish adults. Sci Total Environ 2007; 372: 406-12.
- [14] Beelen R, Hoek G, Houthuijs D, van den Brandt PA, Goldbohm RA, Fischer P, Schouten LJ, Armstrong B and Brunekreef B. The joint association of air pollution and noise from road traffic with cardiovascular mortality in a cohort study. Occup Environ Med 2009; 66: 243-50.
- [15] Gan WQ, Davies HW, Koehoorn M and Brauer M. Association of long-term exposure to community noise and traffic-related air pollution with coronary heart disease mortality. Am J Epidemiol 2012; 175: 898-906.

- [16] Suadicani P, Hein HO and Gyntelberg F. Occupational noise exposure, social class, and risk of ischemic heart disease and allcause mortality--a 16-year follow-up in the Copenhagen Male Study. Scand J Work Environ Health 2012; 38: 19-26.
- [17] Babisch W. Epidemiological Studies of the Cardiovascular effects of Occupational Noise -A Critical Appraisal. Noise Health 1998; 1: 24-39.
- [18] van Kempen EE, Kruize H, Boshuizen HC, Ameling CB, Staatsen BA and de Hollander AE. The association between noise exposure and blood pressure and ischemic heart disease: a meta-analysis. Environ Health Perspect 2002; 110: 307-17.