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Risk of all-cause mortality and cardiovascular disease associated with secondhand smoke exposure: A systematic review and meta-analysis

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ABSTRACT

Background: Emerging studies have assessed the association between secondhand smoke (SHS) exposure and cardiovascular disease (CVD) as well as all-cause mortality. However, findings were not consistent due to the heterogeneity of study characteristics.

Methods: PubMed and Embase were searched through May 2014 for prospective cohort and case–control studies investigating the associations of SHS exposure in never smokers with all-cause mortality and the risk of CVD. The main analysis was performed in studies using self-reported SHS exposure and secondary analysis was performed in studies using objectively measured SHS exposure. Summary estimates were calculated using random-effects models.

Results: Twenty-three prospective and 17 case–control studies were included. The pooled relative risks (RR) for never smokers exposed to SHS in comparison with those unexposed were 1.18 [95% confidence interval (CI): 1.10–1.27] for all-cause mortality (12 studies), and 1.23 (1.16–1.31) for CVD (38 studies). The association of SHS exposure with CVD was markedly stronger among studies conducted in China (RR = 1.65, 95% CI 1.27–2.13) than that in the US (RR = 1.09, 95% CI 1.03–1.16). Studies using objectively measured SHS exposure demonstrated a slightly higher risk for CVD compared with those using self-reported SHS exposure.

Conclusions: Exposure to SHS significantly increased the risk for all-cause mortality and CVD. The risk associated with SHS exposure was large in China while the risk was only modest in the US. Studies using objectively measured SHS exposure may yield a higher risk of CVD than those using self-reported SHS exposure.

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1. Introduction

Cardiovascular disease (CVD) remains the leading cause of death worldwide [1]. Among various modifiable risk factors of CVD, smoking is one of the most significant. Secondhand smoke (SHS), also known as passive smoking, was associated with 25% to 30% increased risk of coronary heart disease (CHD), as suggested by previous systematic reviews and meta-analyses [2–5]. With over 30% of the world's non-smokers exposed to SHS [6], the worldwide burden of deaths from CVD attributable to SHS is immense.

A number of new studies assessing the association between SHS and CVD have been published following the last comprehensive meta-analysis in 1999 [2]. Most of these studies demonstrated an increased risk of CVD associated with SHS, while others found little or no relationship, presumably due to the heterogeneity of study characteristics (e.g. gender distribution, ethnicity, or source of exposure). For example, Gallo et al. reported a significantly increased risk of CVD mortality related to SHS exposure at home but not to exposure at workplaces [7]. In a large prospective study in Norway, Iversen et al. found that the risk of myocardial infarction was increased in passive smoking women but not in passive smoking men [8]. Additionally, by analyzing data from the American Cancer Society's Cancer Prevention Study I, Enstrom et al. failed to detect a significant association of exposure to SHS with CHD mortality [9], while in a cohort study in China, He et al. showed that never smokers exposed to SHS had a 2-fold risk of CHD death [10]. Therefore, the question as to what extent SHS is associated with CVD risk and whether the association varied by important study characteristics warrants an in-depth reassessment of the relationship between SHS exposure and CVD.

Early studies investigating the relationship between SHS and CVD mainly relied on participants' self-reported SHS exposure at home or workplaces. However, this questionnaire-based assessment of SHS

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might be subject to misclassification. In 2004, for the first time, Whincup et al. evaluated the risk of CHD associated with SHS using an objective assessment of SHS exposure by measuring concentrations of serum cotinine, showing that the risk of CHD might be increased by 54% to 89% in never smokers with different levels of SHS exposure [11]. Since the objective measurement was able to capture the total SHS exposure from all sources, researchers argued that the cardiovascular impact of SHS exposure may be as large as that of active smoking and that previous assessment of SHS by self-reported exposure could have underestimated the impact of SHS [3]. Following Whincup, three studies were conducted using this objective cotinine-based measurement to examine the risk of CVD related with SHS. Available evidence needs to be synthesized to generate a more precise risk estimate of CVD associated with objectively assessed SHS exposure and to determine whether self-reported measurement of SHS did bias the risk of CVD downward.

SHS exposure was not only related to risk of cardiovascular morbidity and mortality, but also increased the risk of other causes of death, such as death due to breast cancer, lung cancer, or chronic obstructive pulmonary disease [12–15]. Literatures have suggested an association between SHS and all-cause mortality. However, the magnitude of the association has not been systematically assessed.

Therefore, we conducted the current meta-analysis to 1) estimate the relative risk (RR) of CVD associated with self-reported SHS exposure as well as objectively measured SHS exposure, and find out whether the association varied by important study characteristics, and 2) estimate the RR of all-cause mortality associated with SHS exposure.

2. Methods

2.1. Search strategy

We performed this meta-analysis following the proposed MOOSE (Meta-Analysis of Observational Studies in Epidemiology) statement.

Two qualified investigators (X.L. and J.S.) conducted database search and discrepancies were resolved by discussion and consensus with a senior investigator (Y.X.). We searched PubMed and Embase for prospective cohort and case–control studies published from inception date to May 2014, which investigated the association between SHS exposure and the risk of all-cause mortality or cardiovascular disease in never smokers. We used the following terms in the search for titles/abstracts: 1) study design: prospective, cohort, follow-up, longitudinal, case–control, inciden*, hazard ratio*, odds ratio*, and relative risk*; 2) exposure: secondhand/second-hand smok*, passive smok*, and environmental tobacco smok*; and 3) outcomes: death, mortality, cardiovascular, coronary heart/artery disease, ischemic/ischaemic heart disease, myocardial infarction, acute coronary syndrome, and stroke.

For each of the three themes, corresponding MESH (medical subject heading) terms in their exploded versions were also used in combination with free term search. Then the three themes were combined using the Boolean operator "AND". No restrictions were imposed. The details of search syntax in PubMed and Embase are shown in Supplementary Tables 1 and 2, respectively. Important relevant reviews and reference lists of included studies were manually examined for additional publications.

2.2. Study selection and eligibility criteria

Following database search, two authors (X.L. and J.S.) independently assessed the study eligibility. Any discrepancies were resolved by discussion. A preliminary screening was based on titles or abstracts to discard studies clearly irrelevant. Then potentially eligible studies were evaluated based on full-text review. Studies were included if they met the following criteria: 1) studies were prospective cohort studies or case–control studies in humans aged ≥18 years; 2) the exposure was SHS or passive smoking in never smokers, and both self-reported SHS exposure and objectively measured SHS exposure were eligible. For self-reported SHS, detailed questionnaire-based descriptions confirming the regular exposure to another person's tobacco smoke at home or out of home should be available; 3) the outcomes of interest were all-cause mortality or CVD (including CHD and stroke); 4) unexposed subjects were used as the reference group, and quantitative estimates such as RR, hazard ratio, or odds ratio and corresponding variance (or information to calculate these measures) were reported; and 5) published in English.

We did not include unpublished studies or abstracts because they had not been formally peer reviewed or published and concerns exist that such studies may be of inferior quality and will bias the meta-analyses findings.

2.3. Data extraction and synthesis

The primary exposure variable was SHS exposure in never smokers, and the reference group was never smokers unexposed to SHS. The assessment of SHS could be based on self-report or measured cotinine levels. For self-reported SHS exposure, the source of exposure could be home, out of home, or both according to the description of SHS exposure in original studies. Home exposure was defined as exposure from family members such as spouse, parents, children or cohabited members. Out-of-home exposure was defined as exposure at workplaces or public places such as bars and restaurants. Where possible, we extracted information for overall sources and specific sources. For cotinine-based SHS exposure, we defined participants with the lowest cotinine levels, or undetectable cotinine concentration as the unexposed group, and other cotinine levels as low to high SHS exposed groups. A single estimate was derived by pooling the RRs of low to high SHS exposed groups as referenced to the unexposed group using a fixed-effects model. This approach was able to distinguish the true SHS exposed group from the true SHS unexposed group and gave a more objective RR estimate.

The primary outcomes were all-cause mortality and CVD (fatal and nonfatal), including CHD (fatal and nonfatal) and stroke (fatal and nonfatal). CHD was defined as myocardial infarction, acute coronary syndrome, or other ischemic heart diseases. The secondary outcomes were CHD and stroke considered separately.

Two authors (X.L. and J.S.) independently extracted data from included studies using a standard form and disagreements were resolved by consensus. The extracted information included first author, year of publication, study period, country of origin, general characteristics of study population (age and gender distribution, and number of never smokers), source of exposure, measurement of outcomes, follow-up duration (for cohort studies), risk estimates and corresponding 95% confidence intervals (95% CI), and confounders adjusted.

Most studies presented results with various degrees of confounding adjustment. RRs with different degrees of adjustment were extracted. In the primary meta-analysis we used RRs calculated from the most extensively adjusted model. For studies reporting risk estimates with different durations of follow-up, we retrieved data with a moderate follow-up period (neither too long nor too short).

If a single risk estimate was not available for a study in which risk estimates were only presented as dose–response ones, or stratified by gender or sources of exposure, the estimates were pooled using a fixed-effects model to calculate a combined risk estimate, and the combined estimate was used for the study.

The quality of the included studies was evaluated according to the Newcastle–Ottawa quality assessment scale (NOS) only for descriptive purposes.

2.4. Statistical analyses

Because the incidence of CVD was low, HR and OR were considered approximations of RR. Individual RRs and corresponding 95% CIs were transformed to their natural logarithms, or lnRR, to stabilize variance and normalize the distribution [16]. We then used the "metan" command in Stata to pool the lnRRs across studies using the DerSimonian and Laird random-effects model [17].

The majority of studies assessed SHS exposure by self-reported questionnaires, thus for the main analysis, we summarized risk estimates from studies using self-reported SHS exposure. If the results on total CVD were not available, we used CHD or stroke (in sequential order) as a surrogate for CVD. We then calculated pooled risk estimates separately for CHD and stroke as two specific categories of CVD. A secondary analysis was performed by summarizing the RRs of CVD associated with objectively measured SHS exposure.

For the main analysis, we conducted stratified analysis according to the following study characteristics: study design (cohort and case–control), gender (male and female), source of exposure (home and out of home), country (US, Europe, China, and other areas), mean age (<65 and ≥65 years), and duration of follow-up for cohort studies (<10 and ≥10 years). The potential modifying effects of these study characteristics on the risk estimates were also tested using meta-regression. Furthermore, to examine whether socioeconomic or other important cardiovascular risk factors had substantial influence on the risk effects, we performed additional meta-analysis using RRs from models with different levels of adjustment separately. Level 1 was unadjusted or minimally adjusted for age and gender. Level 2 was adjusted for age, gender, socioeconomic and lifestyle factors. Level 3 was adjusted for age, gender, socioeconomic and lifestyle factors, and important cardiovascular risk factors including hypertension, diabetes, dyslipidemia, or levels of blood pressure, glucose, and lipids.

Heterogeneity across the studies was tested by the Cochrane Q statistic and the I^2 statistic. Publication bias was evaluated by visual inspection of the asymmetry of funnel plots, and Egger linear regression test at the $P < 0.10$ level of significance. If publication bias was indicated, we further performed the trim-and-fill method to assess the possible effect of publication bias [18].

All statistical analyses were done on Stata version 12.0 (StataCorp, College Station, TX, USA).

3. Results

3.1. Identification of studies

Our initial electronic database search yielded 752 publications: 521 from PubMed and 231 from Embase. After screening titles and abstracts, we identified 58 articles for further full-text review. Among those, we identified two studies which met our inclusion criteria but the required

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