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Does utilizing WHO's interim targets further reduce the risk - meta-analysis on ambient particulate matter pollution and mortality of cardiovascular diseases?☆

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ABSTRACT

Long-term exposed to ambient particulate matter (PM) has been recognized as a risk factor for cardiovascular diseases (CVDs) mortality but few studies examine the utility of WHO's interim targets (2006) concerning PM control on CVD mortality. This review aimed to synthesize the long-term exposed to ambient PM exposures on overall CVD mortality according to WHO's interim targets; meanwhile, subgroup analysis by ethnicity, smoking status and PM assessment method were also conducted. We systematically searched studies published between January 1974 and Jul 2017 in PubMed and Embase. Quality of each study was assessed using Critical Appraisal Skill Programme (CASP) checklist. Fixed-effects or random-effects model of meta-analysis was determined by the test of heterogeneity. Subgroup analyses were conducted according to ethnicity, smoking status, PM assessment method and interim PM targets of WHO guidelines. Overall, 16 eligible studies were included, covering 15,511,997 participants and 542,991 CVD deaths. Five studies concerning PM2.5 were rated as good quality. The pooled hazard ratio (HR) of every 10μg/m3 increment of PM2.5 exposure for CVD mortality was 1.12 (95% CI = 1.08−1.16), but it was not significant for PM10 (HR = 1.02, 95%CI = 0.89−1.16). Compared with the annual PM2.5 exposure level within WHO's interim targets (1.11−1.16), significantly smaller HR was observed for subjects with an exposure level below WHO's air quality guideline (HR = 1.03, 95%CI = 1.02 −1.04). The pooled HR was relatively higher for studies in Asian and with at least 11 years' follow-up and those adopting relatively poor methods (category 1) in assessing PM2.5, whilst the risk was similar regardless of smoking status. Egger and Begg's tests showed no evidence for publication bias. Long-term ambient PM2.5 exposure level was positively associated with the overall CVD mortality. Different interim targets above the WHO's Air Quality Guideline level exerted a similar scale of CVD risk, but there is no evidence for a threshold.

1. Introduction

Cardiovascular diseases (CVDs) are the leading causes of death that accounted for nearly 31% of global mortality in 2012 (World Health Organization, 2006). Particulate matter (PM) is a complex mixture with a number of components including acids, organic chemicals, and metals (Hildemann et al., 1991). Many of these components are highly toxic substances associated with chronic diseases including CVDs (Sacks et al., 2011). According to the

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research done by Schauer, the source of PM mainly came from diesel and gasoline exhaust, emission from cooking and heating, dust, cigarette smoke and natural gas combustion (Schauer et al., 1996). PM could be divided into PM2.5 (aerodynamic diameter <2.5 μm) and PM10 (aerodynamic diameter ≤10 μm), and the harmful effect of PM on CVDs was firstly reported in the Harvard 6 cities' study, in which an increased risk of cardiopulmonary disease mortality was observed among residents with long-term PM exposure ranged from 11.0 to 29.6 $\mu g/m^3$ (Dockery et al., 1993; Grahame and Schlesinger, 2010). This finding was further supported by several studies that were conducted in France, Netherland, and UK (Bentayeb et al., 2015; Brunekreef et al., 2009; Carey et al., 2013). Long-term exposure to elevated level of PM has posed serious public health problems worldwide, particularly in the areas with heavy PM pollution (Villeneuve et al., 2015).

The most recent meta-analysis regarding the long-term exposure to ambient PM air pollution and CVD mortality was conducted by Hoek et al., in 2013. They reported a 11% increased risk [95% confidence interval (95%CI) = 6-13%] of CVD mortality for per 10$\mu g/m^3$ increase of PM2.5 and 3.5% increased risk (95%CI = 0.4%–6.6%) for PM10 (Hoek et al., 2013). However, the review of Hoek et al. combined occupational and non-occupational studies and thus their results are probably entangled by occupational source of exposure to PM2.5 (Fang et al., 2010). World Health Organization (WHO) set a serial of interim targets for PM guideline (World Health Organization, 2006) based on the accumulative evidence on the harmful effects of PM2.5 aiming at reducing disease burden. Although this WHO's interim target for PM guideline quantitatively measures possible gradient relations with PM exposures, no review has been conducted to summarize the effect of PM on CVD mortality according to different interim targets of WHO. Also, there are possible variations in the effect of PM2.5 on CVD mortality amongst subgroups of ethnicity, smoking status, exposure period and PM assessing method. As vulnerable populations, such as, smokers or people with longer time PM exposure are more susceptible for developing cardiovascular diseases and premature death; however, these areas of evidence have never been assessed and summarized.

This review aimed to synthesize the long-term effect of ambient PM air pollution on overall CVD mortality according to WHO's interim targets (World Health Organization, 2006); meanwhile, subgroup analysis by ethnicity, smoking status and PM assessment method were also conducted.

2. Methods

This review follows the guidelines of the PRISMA statement (Moher et al., 2009).

2.1. Eligibility and search strategy

We searched PubMed and Embase databases for cohort studies that reported the association between CVD mortality and long-term exposure (study period equal or larger than 1 year) to outdoor PM10 or PM2.5. The search included papers published in English and between January 1974 and July 2017. Detailed searching strategy was summarized in Supplement 1. Two authors (ZL and FW) independently reviewed potential studies for inclusion, and any discrepancy encountered was resolved by consensus.

2.2. Inclusion and exclusion criteria

Duplicated studies were excluded. The initial screening was based on the title and abstract of searching results. The following inclusion criteria were used for the screening: (i) investigated the association between CVD mortality and long-term ambient PM exposure; (ii) individual studies provided sufficient exposure data on PM10 or PM2.5; and (iii) cohort study; and (iv) provided sufficient data to calculate hazards ratio (HR) with 95%CI. Studies that met the following items were excluded: (i) irrelevant to PM or CVD mortality; (ii) study period shorter than 1 year; (iii) indoor rather than outdoor PM exposure; (iv) non-cohort study. Full-text papers were reviewed to determine the eligibility of studies if their abstracts missed the key information. Studies were excluded if they did not report the quantitative estimation of effect size for the association between PM10 or PM2.5 and CVD mortality (or incidence). Studies were also excluded if they focused on CVD prognosis among patients or specific occupational groups. If two or more reports were generated from one cohort, the report that presented the largest sample or longest follow-up period was included in this review.

2.3. Data extraction

We extracted information from each eligible study regarding the study population/region, study period, exposure assessment (i.e., mean concentration and concentration range; data source and exposure assessment method), definition of outcome (ICD codes, 390–459 for ICD-9, or I00-I99 for ICD-10), number of CVD deaths, and hazard ratio (HR) (after adjustment) for each 10 $\mu g/m^3$ increment of exposure level and its 95% CI. We re-calculated the weighted arithmetic mean concentration for the Nishiwaki's study according to the population size and mean concentration of PM in each city due to lack of relevant exposure data (Nishiwaki et al., 2013). Two reviewers (ZL and YW) extracted the data independently.

2.4. Quality assessment

Quality assessment was conducted for each study using Critical Appraisal Skills Programme (CASP) checklist (Claydon, 2015). The checklist for assessing study quality provides an overall score for each study and subscales for three individual sections: study valid (8 items), result condition (3 items) and result applicability (3 items). Items regarding the cohort assessment were adopted for quality assessment, yielding a maximal score of 14 for each study based on the checklist (Supplement 2). We classified an included study as relatively low quality if the score of this study was below or equal to the median score; otherwise, the study was rated to have good quality. In CASP checklist, the Question 3 (Q3) "Was the exposure accurately measured to minimize bias?" was to evaluate the quality of exposure assessment methods (Supplement 2 & 3). According to PM assessment method described in the methodology studies (Hoek, 2017), we assigned "1" for the individual studies that retrieved PM exposure data in address level based on chemical transportation mode, land use regression modeling, or satellite modeling (category 3). We assigned "0.5" for those retrieving PM exposure data using interpolation concentration, atmospheric diffusion, or dispersion model in community level based on multiple fixed monitoring stations (category 2). We assigned "0" for the remaining studies that retrieved PM data at city level based on a single fixed monitoring station (category 1). Two authors (ZL and YW) independently scored each included study and any discrepancy encountered was resolved by consensus.

2.5. Statistical analysis

We conducted meta-analysis by using Stata v14.1 (Stata Corp, College station, Texas, USA) to obtain the pooled HRs for per 10 $\mu m/m^3$ increment of PM10 and PM2.5, respectively (Deeks et al., 2001). Random-effects model was used to combine HRs from individual