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## RESEARCH ARTICLE

# Association between PM<sub>10</sub> exposure and risk of myocardial infarction in adults: A systematic review and meta-analysis

Kleiton Strobl<sup>1\*</sup>, Syed Asad Irfan<sup>1</sup>, Hassan Masood<sup>1</sup>, Noor Latif<sup>2</sup>, Om Kurmi<sup>1,3</sup>

**1** Faculty of Health Sciences, McMaster University, Hamilton, Canada, **2** Faculty of Science, McMaster University, Hamilton, Canada, **3** Faculty Research Centre for Healthcare and Communities, Coventry University, Coventry, United Kingdom

\* [kstrobl1@gmail.com](mailto:kstrobl1@gmail.com)

## Abstract

### Background

Air pollution has several negative health effects. Particulate matter (PM) is a pollutant that is often linked to health adversities. PM<sub>2.5</sub> (PM with an aerodynamic diameter of  $\leq 2.5\mu\text{m}$ ) exposure has been associated with negative cardiovascular (CV) outcomes. However, the impact of PM<sub>10</sub> (PM with an aerodynamic diameter of  $\leq 10\mu\text{m}$ ) exposure is often overlooked due to its limited ability to pass the alveolar barrier. This study aims to assess the association between PM<sub>10</sub> exposure and risk of myocardial infarction (MI) amongst adults ( $\geq 18$  years of age) as this has been poorly studied.

### Methods

The study protocol was published on the International Prospective Register of Systematic Reviews (PROSPERO) (CRD42023409796) on March 31, 2023. Literature searches were conducted on 4 databases (Ovid Medline, Embase, CINAHL (Cumulative Index to Nursing and Allied Health Literature), and Web of Science) on January 17, 2023, for studies looking at associations between PM and MI. English studies from all time periods were assessed. Studies selected for review were time-series, case-crossover, and cohort studies which investigated the risk of MI as an outcome upon PM<sub>10</sub> exposure. The quality of evidence was assessed using Cochrane's Grading of Recommendations, Assessment, Development and Evaluation (GRADE) approach. Data for different risk outcomes (risk ratio (RR), odds ratio (OR), hazard ratio (HR)) and 3 lags was meta-analyzed using an inverse variance statistical analysis using a random effects model. The pooled effect sizes and the 95% confidence intervals (CIs) were reported in forest plots.

### Results

Among the 1,099 studies identified, 41 were included for review and 23 were deemed eligible for meta-analysis. Our analysis revealed that there is an increased risk (OR = 1.01; 95% CI: 1.00–1.02) of MI with a  $10\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> after a lag 0 and lag 1 delay.

## OPEN ACCESS

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## Conclusions

Our findings indicate that PM<sub>10</sub> exposure is associated with an increased risk of MI. This can aid in informing environmental policy-making, personal-level preventative measures, and global public health action.

## 1. Introduction

Air pollution is a complex combination of gaseous and particle constituents, which are hazardous to human health [1]. In air pollution, particulate matter (PM) comprises carbonaceous particles containing adsorbed organic compounds and reactive metals [1]. Nitrates, sulfates, polycyclic aromatic hydrocarbons, endotoxin, and metals such as iron, copper, nickel, zinc, and vanadium are all common constituents of PM [1]. PM can be further classified relative to the particle size: PM with a diameter <10 $\mu$ m (PM<sub>10</sub>), fine PM (PM<sub>2.5</sub>; diameter <2.5 $\mu$ m), and ultrafine PM (PM<sub>0.1</sub>; diameter <0.1 $\mu$ m) [1]. Earlier scientific research has reported that PM<sub>2.5</sub> is associated with adverse health outcomes, including poor cardiovascular (CV) health outcomes. PM<sub>10</sub> is present in dust from roads, farms, construction sites, and mines [2]. However, the chemical composition and size distribution of PM<sub>10</sub> varies widely depending on where it originates and how it forms in the environment [3]. Although PM<sub>10</sub> is also associated with adverse impact health as it is an irritant for the nose, throat, and eyes; however, in general, this is not a major focus airborne pollutant of study [2].

The short-term impacts of PM<sub>10</sub> exposure on respiratory pathologies, such as chronic obstructive pulmonary disease and asthma, are well known and studied [3]. However, in CV health research, the effects of PM<sub>2.5</sub> are more often researched as PM<sub>2.5</sub> has been shown to pass the alveolar barrier and cause an inflammatory response in blood vessels [4]. This exacerbates atherosclerosis and increases the risk of myocardial infarction (MI), ischemic heart disease, and thrombotic stroke [4]. The risk of MI was chosen as it is one of the largest causes of death worldwide, with approximately three million deaths annually [4]. Different populations are also exposed to varying levels of PM, and understanding its health adversities is important for understanding subsequent health disparities. Interestingly, there is a lack of up-to-date research regarding the impact of PM<sub>10</sub> on the risk of MI despite instances being reported where exposure to PM<sub>10</sub> has led to a surge in CV-related hospital admissions [3]. Although there is a higher probability of PM<sub>2.5</sub> passing the alveolar barrier, it is still possible for PM<sub>10</sub> to enter the bloodstream in smaller proportions [5]. When searching this topic on the International Prospective Register of Systematic Reviews (PROSPERO) database, only one matching systematic review and meta-analysis was found, and it solely assessed the impact of PM<sub>2.5</sub> and not PM<sub>10</sub> [6]. Past studies looked at MI risk due to short-term PM<sub>10</sub> exposure, or long-term PM<sub>10</sub> exposure, or both, but are missing up-to-date literature in their review [7–9]. This is the knowledge gap that this study aims to address. The primary aim of this systematic review and meta-analysis is to assess the effect of PM<sub>10</sub> on the risk of MI to better understand its burden of disease and update pre-existing literature looking at the CV impacts of PM exposure.

## 2. Methods

### 2.1. Search strategy

Studies were obtained from a systematic search of the following databases: OVID Medline, Embase, Cumulative Index to Nursing and Allied Health Literature (CINAHL), and Web of

Science. The literature searches were conducted on January 17, 2023, and included studies from all time periods. See the specific search terms utilized (S1 Table). The study protocol was published on PROSPERO (CRD42023409796) on March 31, 2023, after completing the literature search.

## 2.2. Inclusion/exclusion criteria

The inclusion criteria for this study were: (1) study design had to be time-series, case-cross-over, or cohort; (2) PM<sub>10</sub> had to be an exposure; (3) risk of myocardial infarction had to be an outcome (risk ratio/relative risk (RR), odds ratio (OR), hazard ratio (HR)); (4) population  $\geq 18$  years of age. Cohort studies were included after the protocol to increase the data pool.

The exclusion criteria for this study were: (1) Other inapplicable primary study designs (randomized controlled trials, clinical trials, protocols, pilot studies, etc.); (2) secondary studies (narrative reviews, systematic reviews, meta-analyses, scoping reviews); (3) Risk outcome(s) only reported in graph(s) (4) studies that only look at subset ranges of PM<sub>10</sub> (PM<sub>2.5</sub> or PM<sub>0.1</sub>); (5) population <18 years of age.

## 2.3. Lag periods

Lag intervals are a common tool used in air pollution literature to define the delay between exposure and disease onset. In this study, lag intervals were selected to accommodate various lag periods provided in the extracted studies, while ensuring that there was adequate data available to meta-analyze each lag interval effectively. This preserves data validity and reliability by maximizing the number of data points that can be meta-analyzed, considering that the studies acquired from the literature search exhibited a wide diversity of lag intervals. PM<sub>10</sub> exposure lags were based on the following definitions: lag 0 = same day (0–24 hours); lag 1 = 1–3 day delay (24–96 hours); lag 2 = 3 day delay or more (>96 hours). If there were multiple data points, the value closest to the midpoint of the range was selected for meta-analysis for lag 0 and 1 (lag 0 = 12 hours; lag 1 = 60 hours). For lag 2, the value closest to >96 hours was selected for meta-analysis.

## 2.4. Study selection

Covidence was used to manage the screening phase of this study [10]. For the abstract screening, two authors (HM & NL) independently screened abstracts based on the inclusion/exclusion criteria, and another two (KS & SI) resolved conflicts. For full-text screening, two authors independently (KS & SI) screened the full manuscripts, and the same authors (KS & SI) discussed any conflicts and reached a consensus. The study drafting process was recorded using “Preferred Reporting Items for Systematic Reviews and Meta-Analyses” (PRISMA) guidelines [11].

## 2.5. Data extraction

Two authors independently extracted data from one half of the included studies (KS & SI), while another two (HM & NL) independently extracted data from the other half of the included studies. Any discrepancies in data values were corrected cohesively amongst the two authors for their extracted data half, respectively.

Data extracted from studies included: study name, design, country, sample size, male/female ratio, participant characteristics, exposure increments for risk measure, risk scale, lag intervals, and effect size (risk outcomes).

For studies with multiple models, outcome data values adjusting for the greatest number of confounding variables were meta-analyzed to maintain data validity. For one study which reported data on both industrial and non-industrial locations, only risk outcome values from industrial locations were meta-analyzed. This was done because industrial locations are likely to have high PM<sub>10</sub> exposure and provide a better opportunity to study cardiovascular risks. For studies that solely reported data on multiple regions of a country, data was selected from the most central geographical region to represent the country as a whole. This was done because the risk measure value and their respective standard deviation values could not be averaged amongst the different regions.

## 2.6. Methodological risk of bias assessment

Risk of bias was assessed by two authors (KS & SI) using the National Heart, Lung, and Blood Institute (NHLBI) Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies [12] which assesses methodological risk of bias through 14 criteria: (1) clear research question/objective; (2) clearly defined study population; (3) 50% participation rate of eligible persons; (4) all participants selected from identical/similar populations in the same time period using pre-specified inclusion and exclusion criteria; (5) provision of sample size justification, power description, or variance and effect estimates; (6) exposure measured before outcome in analysis; (7) sufficient time frame to expect association; (8) examination of differing levels of exposure as related to the outcome; (9) clearly defined, valid, reliable, widely-applied exposure measures; (10) >1 exposure assessments; (11) clearly defined, valid, reliable, widely-applied outcome measures; (12) blinding of outcome assessors to participant exposure status; (13) <20% loss to follow-up after baseline; (14) measurement and adjustment of potential confounders affecting impact between exposure and outcome.

Each criterion was deemed to be satisfied or unsatisfied based on the author's rating. Overall quality ratings included: 'good', 'fair', and 'poor'. The overall quality of each study began at 'good', and got demoted by one level per unsatisfied criteria. Conflicts in the methodological risk of bias assessment were resolved by the same two authors (KS & SI) through consensus.

## 2.7. Meta-analysis

Meta-analysis was conducted using Review Manager 5.4.1.

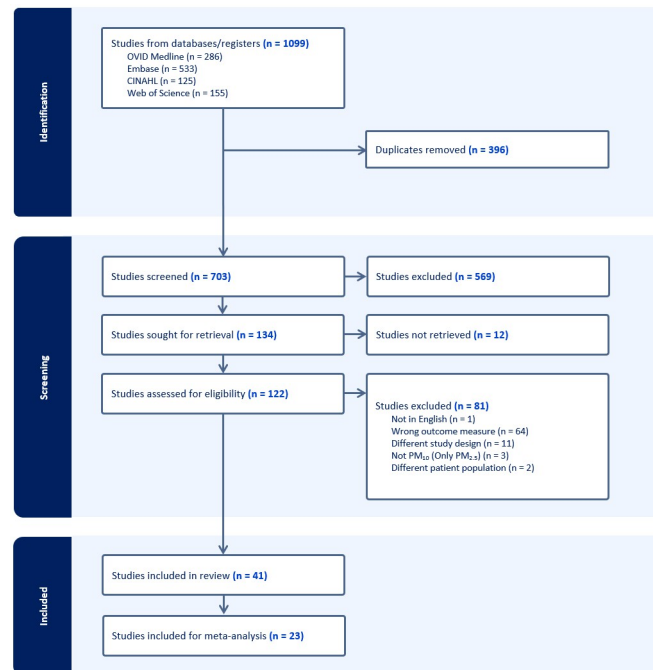
**2.7.1. MI risk outcome measures.** The primary outcome was MI risk. For the meta-analysis, data was included for studies reporting risk based on PM<sub>10</sub> concentration increments of 10µg/m<sup>3</sup>. MI risk outcome measures included RR, OR, and HR with 95% confidence intervals (CIs). A RR, OR, or HR >1 indicates a higher PM<sub>10</sub>-associated MI risk.

MI risk was stratified by MI risk outcome measure (RR, OR, HR) to preserve data validity because of the inherent distinctiveness of these outcome measures [13]. Data was also stratified by lags (0, 1, 2) as aforementioned.

**2.7.2. Data synthesis.** The pooled risk outcome was considered if the following criteria were met: two or more studies reported the same MI risk outcome measure, and two or more studies reported for the same lag interval.

An I<sup>2</sup> >60% was considered heterogeneous on a statistically significant level. An inverse variance statistical analysis was conducted with a random-effects model when creating the forest plots, as there was heterogeneity, differences in study design, setting, and adjustment models. Results with a P-value of <0.05 and I<sup>2</sup> ≤60% were considered statistically significant.

Publication bias was assessed through analysis of funnel plot symmetry. Publication bias was not assessed for quantitative analyses with <5 studies due to a lack of statistical power.



**Fig 1. PRISMA flow diagram.**

<https://doi.org/10.1371/journal.pone.0301374.g001>

**2.7.3. Quality of evidence evaluation.** Quality of evidence was evaluated by two authors (KS & SI) independently using Cochrane’s Grading of Recommendations, Assessment, Development and Evaluation (GRADE) approach, which assesses: (1) risk of bias; (2) inconsistency; (3) indirectness; (4) imprecision; (5) publication bias; (6) large magnitude of effect; (7) dose-response gradient; (8) residual confounding. Overall quality ratings include: ‘high’, ‘moderate’, ‘low’ and ‘very low’. All resulting outcomes began with a rating of ‘high’ and were demoted one level for each unsatisfied criteria 1–5; upgrading could occur for satisfying criteria 6–8. Any evaluation conflicts were resolved by the same two authors (KS & SI) through consensus.

## 3. Results

### 3.1. Study selection

A breakdown of study identification and screening can be found in the PRISMA flowchart (Fig 1). A systematic literature search from databases/registers identified 1,099 potential studies viable for inclusion. After 396 duplicates were removed, 703 total studies were available for abstract screening. Five hundred sixty-nine studies were excluded following abstract screening, and 12 studies were not retrieved due to a lack of full-text availability. One hundred twenty-two studies remained for full-text screening, and 81 studies were excluded after the full-text screening: 64 for having the wrong outcome measure, 11 for having different study design (not meeting the inclusion criteria), 3 for analyzing PM<sub>2.5</sub> exclusively and not PM<sub>10</sub>, 2 for having different patient population, and 1 study for not being in English. Forty-one studies were included for review, with 23 of 41 deemed eligible for meta-analysis.

**Table 1. Study characteristics for studies included in the meta-analysis.**

| #  | Author/publication year | Study Design   | Country        | Sample size (n) | # Male (%)     | Risk Measure | Lag Intervals  |
|----|-------------------------|----------------|----------------|-----------------|----------------|--------------|--|
| 1  | Argacha 2016 [14]       | Case-crossover | Belgium        | 11,428          | 8,607 (75.3)   | OR           | Lag 0: 24hrs post-exposure   |
| 2  | Bard 2014 [15]          | Case-crossover | France         | 2,134           | 1,642 (76.9)   | OR           | Lag 0: Same day<br>Lag 1: 1-day delay<br>Lag 0–1: Average of same day and 1 day previous   |
| 3  | Bhaskaran 2011 [16]     | Case-crossover | United Kingdom | 79,288          | 50,988 (64.8)  | RR           | Lag 0: 1-6hrs<br>Lag 1: 7-12hrs<br>Lag 2: 13-18hrs<br>Lag 3: 19-24hrs<br>Lag 4: 25-72hrs   |
| 4  | Buszman 2020 [17]       | Case-crossover | Poland         | 1,957           | NR             | OR           | Lag 0: Same day<br>Lag 1: 1-day delay  |
| 5  | Cheng 2021 [18]         | Case-crossover | Australia      | 3,307           | 2,162 (65.4)   | RR           | Lag 0: 1hr<br>Lag 1: 2-6hrs<br>Lag 2: 7-12hrs<br>Lag 3: 13-24hrs   |
| 6  | Claeys 2015 [19]        | Time-series    | Belgium        | 15,963          | 11,995 (75.1)  | RR           | 5-day delay  |
| 7  | Collart 2017 [20]       | Time-series    | Belgium        | 21,491          | 14,377 (66.9)  | RR           | Single-day lags: Lag 0, 1, 2, 3, 4, 5, 6   |
| 8  | Davoodabadi 2019 [21]   | Case-crossover | Iran           | 319             | 238 (74.6)     | OR           | Lag 0: 24h post-exposure<br>Lag 1: 48h post-exposure   |
| 9  | Downward 2018 [22]      | Cohort         | Netherlands    | 33,381          | 7,846 (23.5)   | HR           | 1-year delay   |
| 10 | Huss 2010 [23]          | Cohort         | Switzerland    | 4,580,311       | NR             | HR           | Average exposure in the year 2000  |
| 11 | Kim 2020 [24]           | Time-series    | South Korea    | 196,167         | 104,949 (53.5) | HR           | 5-year delay   |
| 12 | Konduracka 2019 [25]    | Time-series    | Poland         | 3,545           | 1,602 (45.2)   | OR           | Single-day lags: Lag 0, 1, 2, 3, 4, 5, 6   |
| 13 | Kuźma 2021 [26]         | Time-series    | Poland         | 9,046           | 5,692 (62.9)   | OR           | Single-day lags: Lag 0, 1, 2, 3, 4, 5, 6   |
| 14 | Lipsett 2011 [27]       | Cohort         | USA            | 124,614         | 0 (0)          | HR           | Median of 8.3-year delay   |
| 15 | Nuvolone 2011 [28]      | Case-crossover | Italy          | 11,450          | 6,985 (61.0)   | OR           | Single-day lags: Lag 0, 1, 2, 3, 4, 5<br>Multiple-day lags: Lag 0–2, 0–5, 3–5  |
| 16 | Puett 2008 [29]         | Cohort         | USA            | 66,250          | 0 (0)          | HR           | Lag 1: 1-month post-exposure average<br>Lag 2: 3-month post-exposure average<br>Lag 3: 12-month post-exposure average<br>Lag 4: 48-month post-exposure average |
| 17 | Royé 2019 [30]          | Time-series    | Spain          | 9,871           | 7,008 (71.0)   | RR           | 14-day delay   |
| 18 | Soleimani 2019 [31]     | Cohort         | Iran           | 6,425           | 3,652 (56.8)   | RR           | Single-day lags: Lag 0, 1, 2, 3, 4, 5, 6, 7, 8, 9  |
| 19 | Vidale 2017 [32]        | Cohort         | Italy          | 4,110           | 2,672 (65.0)   | HR           | Single-day lags: Lag 0, 1, 2, 3, 4   |
| 20 | Yang 2022 [33]          | Case-crossover | China          | 25,299          | 17,100 (67.6)  | RR           | Lag 1: 1-day delay   |
| 21 | Yen 2022 [34]           | Case-crossover | China          | 979,979         | 458,195 (46.8) | OR           | Lag 0: Same day<br>Lag 1: 1-day delay<br>Lag 0–1: Average of same day and 1 day previous   |
| 22 | Zhang 2016 [35]         | Case-crossover | China          | 2,749           | 1,612 (58.6)   | OR           | Single-day lags: Lag 0, 1, 2, 3, 4, 5  |
| 23 | Zhu 2019 [36]           | Cohort         | China          | 147,422         | NR             | RR           | Single-day lags: Lag 0, 1, 2, 3, 4, 5, 6   |

RR = Risk ratio; OR = Odds ratio; HR = Hazard ratio; NR = Not reported

<https://doi.org/10.1371/journal.pone.0301374.t001>

### 3.2. Study characteristics

Table 1 provides the characteristics of studies included in the meta-analysis [14–36], adjusted confounding variables can be found for these respective studies (S2 Table). See study characteristics for other studies included in the review (S3 Table) (reference).



**3.2.1. Study design characteristics.** Amongst the 41 studies included in the systematic review [14–54], 22 studies (53.7%) had a case-crossover design, 12 studies (29.3%) had an observational cohort design, and 7 studies (17.1%) had a time-series design.

For the meta-analysis, ten studies (43.5%) had a case-crossover design, seven studies (30.4%) had an observational cohort design, and six studies (26.1%) had a time-series design.

**3.2.2. Participant characteristics.** For qualitative synthesis, data was calculated from a total of 6,663,870 participants.

For the meta-analysis, data was calculated from a total of 6,336,506 participants. All but three studies reported a male/female participant ratio [17, 23, 36]. Of 1,606,816 participants from the remaining studies with a reported male/female participant ratio, there were 707,322 male participants (44.0%) and 899,494 (56.0%) female participants.

**3.2.3. MI risk outcome measures.** Amongst the 41 studies included in the review [14–54], 18 studies (43.9%) used OR as their effect size for MI risk outcome measure, 12 studies (29.3%) used RR, and 11 studies (26.8%) used HR.

For the meta-analysis, studies most often utilized OR (n = 9; 39.1%), followed by RR (n = 8; 34.8%) and HR (n = 6; 26.1%) for MI risk outcome measures.

### 3.3. Methodological risk of bias analysis for included studies

After the quality assessment was conducted (S4 Table), a large majority of studies were rated at a quality score of good (38/41), with the remainder of the studies rated at a quality of fair (3/41). No studies were rated at a poor quality score. All three studies demoted to a fair quality score were done so for the same reason: failure to adjust for confounders [17, 19, 42].

### 3.4. Primary MI risk outcomes—Meta-analysis

See the meta-analysis summary of primary MI risk outcomes (Table 2). For lag 0 and lag 1 HR outcomes, meta-analysis could not be conducted as there was only one associated study for each respective outcome [32] (S1 and S2 Figs). Evidence assessment could also not be conducted due to one study (S11 and S12 Tables).

**3.4.1. Risk ratio—Lag 0 (Same day [0–24hrs]).** Three studies with three outcomes (n = 89,020) were reported for RR. Soleimani et al. [31] and Cheng et al. [18] reported an increased risk of MI with PM<sub>10</sub> exposure, while Bhaskaran et al. [16] reported a decreased risk. Statistical pooling was appropriate due to statistical homogeneity ( $I^2 = 60\%$ ) (Fig 2). Although there was an overall increased risk of MI (RR = 1.02, 95% CI: 0.97–1.07) it was not statistically significant (P-value = 0.43). Evidence quality was rated high on the GRADE scale (S5 Table).

**3.4.2. Risk ratio—Lag 1 (1–3 days [24–96hrs]).** Four studies with four outcomes (n = 132,503) were reported for RR. Statistical pooling was inappropriate for the lag 1 RR

**Table 2. Summary of primary MI risk outcomes.**

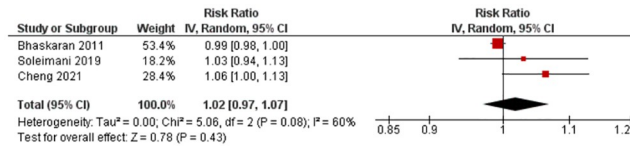
|              | Lag 0                | Lag 1                | Lag 2                |
|--------------|----------------------|----------------------|----------------------|
|              | Effect Size (95% CI) | Effect Size (95% CI) | Effect Size (95% CI) |
| Risk Ratio   | 1.02 (0.97,1.07)     | 1.01 (0.99,1.03)     | 1.01 (1.00,1.03)     |
| Odds Ratio   | 1.01 (1.00,1.02)*    | 1.01 (1.00,1.02)*    | 1.01 (0.99,1.02)     |
| Hazard Ratio | 1.00 (1.00,1.01)†    | 1.00 (1.00,1.01)†    | 1.00 (0.99,1.01)     |

\* P-value < 0.05 &  $I^2 \leq 60\%$ ;

† based on one study result

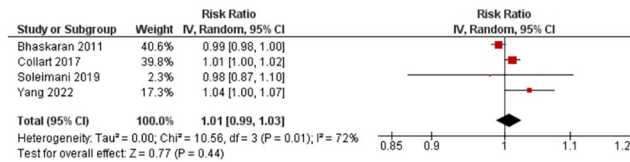
<https://doi.org/10.1371/journal.pone.0301374.t002>





**Fig 2. Lag 0 RR for MI after 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> exposure.**

<https://doi.org/10.1371/journal.pone.0301374.g002>



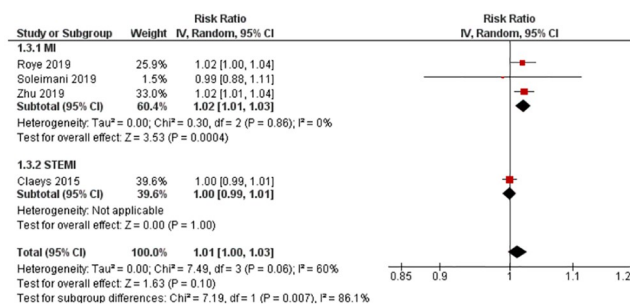
**Fig 3. Lag 1 RR for MI after 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> exposure.**

<https://doi.org/10.1371/journal.pone.0301374.g003>

values because of statistical heterogeneity (I<sup>2</sup> = 72%) (Fig 3). There was an overall increased risk of MI (RR = 1.01, 95% CI: 0.99,1.03) but it was not statistically significant (P-value = 0.44). Evidence quality was rated moderate on the GRADE scale due to inconsistency (S6 Table).

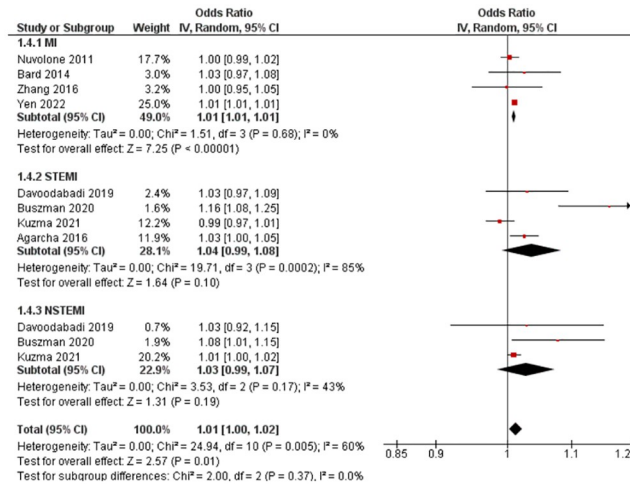
**3.4.3. Risk ratio—Lag 2 (3+ days [96hrs+]).** Four studies with four outcomes (n = 179,681) were reported for RR. Royé et al. [30] and Zhu et al. [36] reported an increased risk of MI with PM<sub>10</sub> exposure, while Soleimani et al. [31] reported a decreased risk. Claeys et al. [19] reported no change in risk. Statistical pooling was appropriate due to statistical homogeneity (I<sup>2</sup> = 60%) (Fig 4). Although there was an overall increased risk of MI (RR = 1.01, 95% CI: 1.00–1.03), it was not statistically significant (P-value = 0.10). Evidence quality was rated high on the GRADE scale (S7 Table).

**3.4.4. Odds ratio—Lag 0 (Same day [0-24hrs]).** Eight studies with eleven outcomes (n = 1,019,062) were reported for OR. Kuźma et al. [26] reported a reduced risk of ST-elevation myocardial infarction (STEMI) with PM<sub>10</sub> exposure, while all other studies reported an increased risk within their respective groups/subgroups (MI, STEMI, and Non-ST-elevation myocardial infarction (NSTEMI)). Statistical pooling was appropriate due to statistical homogeneity (I<sup>2</sup> = 60%) (Fig 5). There was an overall increased risk of MI (RR = 1.01, 95% CI: 1.00–1.02), and it was statistically significant (P-value = 0.01). Evidence quality was rated high on the GRADE scale (S8 Table).



**Fig 4. Lag 2 RR for MI after 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> exposure.**

<https://doi.org/10.1371/journal.pone.0301374.g004>

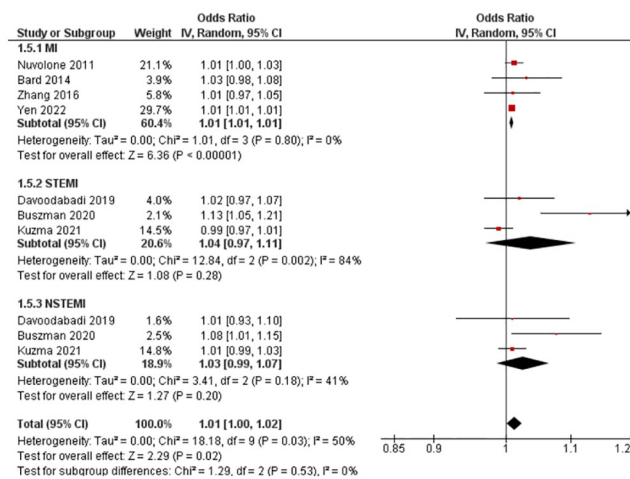


**Fig 5. Lag 0 OR for MI after 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> exposure.**

<https://doi.org/10.1371/journal.pone.0301374.g005>

**3.4.5. Odds ratio—Lag 1 (1–3 days [24–96hrs]).** Seven studies with ten outcomes (n = 1,007,634) were reported for OR. Kuźma et al. [26] reported a reduced risk of STEMI with PM<sub>10</sub> exposure, while all other studies reported an increased risk within their respective groups/subgroups (MI, STEMI, NSTEMI). Statistical pooling was appropriate due to statistical homogeneity (I<sup>2</sup> = 50%) (Fig 6). There was an overall increased risk of MI (RR = 1.01, 95% CI: 1.00–1.02), and it was statistically significant (P-value = 0.02). Evidence quality was rated high on the GRADE scale (S9 Table).

**3.4.6. Odds ratio—Lag 2 (3+ days [96hrs+]).** Five studies with seven outcomes (n = 27,109) were reported for OR. Zhang et al. [35] reported a reduced risk of MI with PM<sub>10</sub> exposure, while all other studies reported an increased risk within their respective group/subgroup (MI, STEMI, NSTEMI). Statistical pooling was appropriate due to statistical homogeneity (I<sup>2</sup> = 41%) (Fig 7). There was an overall increased risk of MI (RR = 1.01, 95% CI: 0.99–



**Fig 6. Lag 1 OR for MI after 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> exposure.**

<https://doi.org/10.1371/journal.pone.0301374.g006>

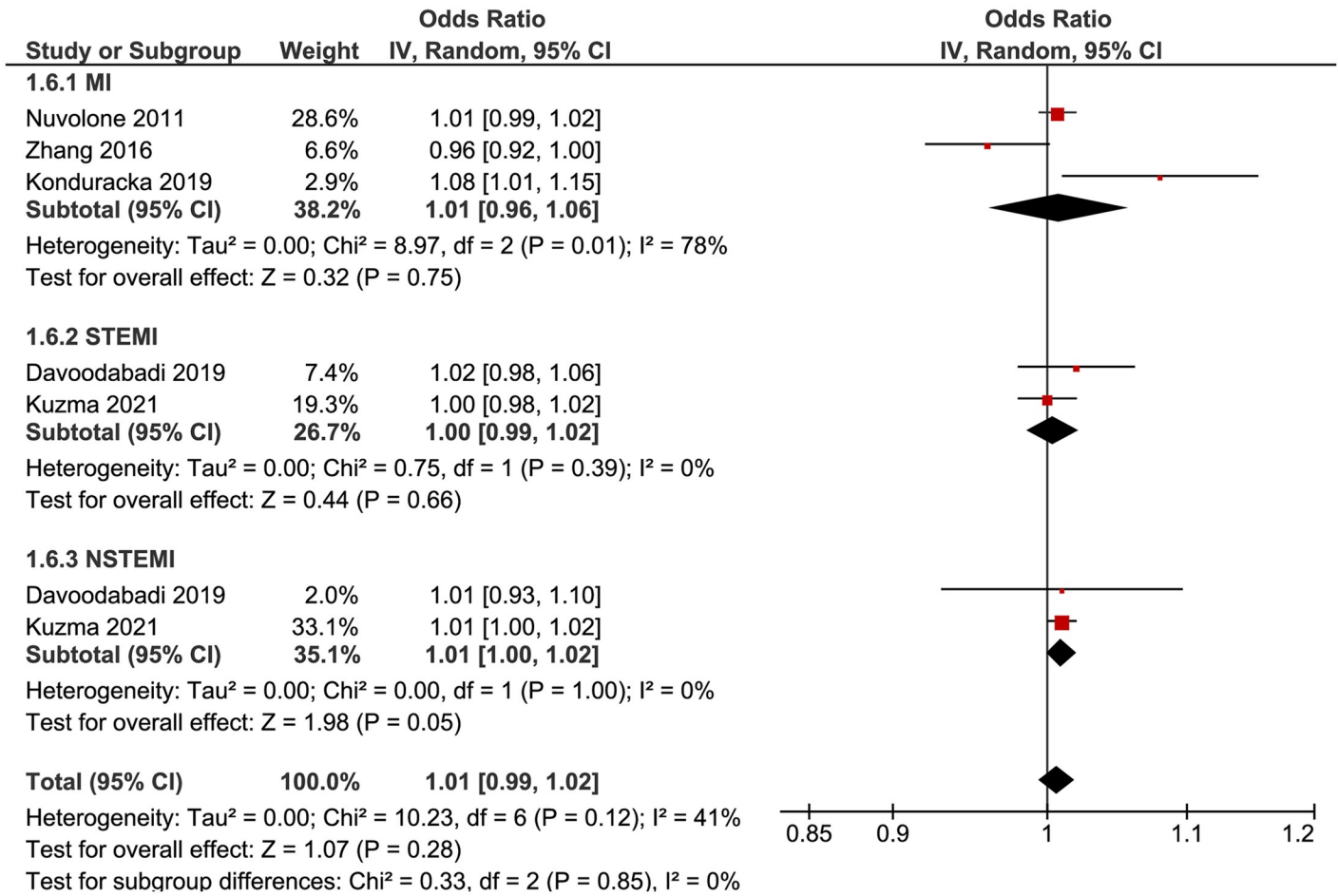


Fig 7. Lag 2 OR for MI after 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> exposure.

<https://doi.org/10.1371/journal.pone.0301374.g007>

1.02), but it was not statistically significant (P-value = 0.28). Evidence quality was rated high on the GRADE scale (S10 Table).

**3.4.7. Hazard ratio—Lag 2 (3+ days [96hrs+]).** Six studies with six outcomes (n = 4,913,615) were reported for HR. Nuvolone et al. [28] and Konduracka et al. [25] reported an increased risk of MI with PM<sub>10</sub> exposure, while Zhang et al. [35] reported a reduced risk. Statistical pooling was appropriate due to statistical homogeneity (I<sup>2</sup> = 6%) (Fig 8). There was no impact on risk of MI (RR = 1.00, 95% CI: 0.99–1.01), and the result was not

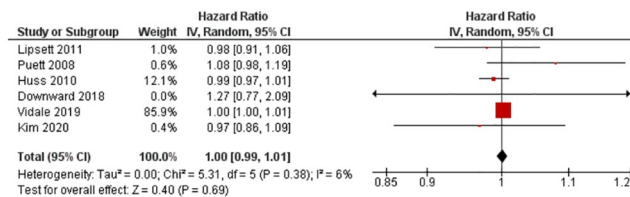


Fig 8. Lag 2 HR for MI after 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> exposure.

<https://doi.org/10.1371/journal.pone.0301374.g008>

statistically significant (P-value = 0.69). Evidence quality was rated high on the GRADE scale (S13 Table).

**3.4.8. Sensitivity analyses.** Two sensitivity analyses were performed. The first sensitivity analysis was the exclusion of risk outcome values which had more than a 7-day post-exposure delay. This was done in order to eliminate values which had relatively long post-exposure delays lasting multiple weeks, months, or years. This sensitivity was conducted to all lag 2 forest plots and consisted of six excluded values: 1 for RR [30] and 5 for HR [22–24, 27, 29]. However, no significant differences were found in this sensitivity analysis.

The second sensitivity analysis was the exclusion of Buszman et al. [17] from the OR forest plots for lag 0 and lag 1. This was done in order to evaluate whether this study was a detrimental outlier and played a significant role in the overall risk outcome. However, no significant differences were found in this sensitivity analysis.

## 4. Discussion

This study aimed to evaluate the risk of MI after exposure to PM<sub>10</sub> considering that small proportions of PM<sub>10</sub> can pass the alveolar barrier, enter the blood, and contribute to cardiovascular adversities as aforementioned [5]. Notably, our analysis showed an increased risk (OR = 1.01; 95% CI:1.00–1.02) of myocardial infarction with a 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> after a lag 0 and lag 1 delay. Considering the high prevalence of MI, the statistically significant risk increase of 1% indicates the absolute risk of PM<sub>10</sub> is substantial. This is an important finding considering that CV disease is the leading cause of death in the world [55]. Assessing potential exacerbators is crucial to reducing the burden of disease.

Farhadi et al. [6] investigated short-term exposure to PM<sub>2.5</sub> and its effects on the risk of MI. Our study and their study both looked at RR and OR as a risk outcome, and assessed studies with a case-crossover and time-series design. However, our study additionally looked at HR as a risk outcome, and assessed studies with a cohort design. Both studies also looked at the effects of lags, but lag definitions were different. Our study looked at lags with short delays (lag 0 = 0–24 hours, lag 1 = 24–96 hours, lag 2 = >96 hours), whereas Farhadi et al. [6] looked at long delays (short follow-up period = <4 years, long follow-up period = >4 years). The results of their study are comparable to our findings as the RR and OR in our meta-analysis were similar. Their meta-analysis reported a RR of 1.02 (95% CI: 1.01–1.03; P-value ≤ 0.0001) [6]. However, our results had more homogeneity, likely due to stratification of studies by risk outcome measures and smaller lag ranges. Our statistical analysis yielded ORs of 1.01 (95% CI 1.00–1.02; P-value <0.05) for lag 0 and lag 1. These results indicate that while the risk of MI from exposure to PM<sub>10</sub> is 1% less in comparison to PM<sub>2.5</sub> exposure, there is a potential impact of PM<sub>2.5–10</sub> pollutants on MI risk. This indicates that the risk of PM<sub>2.5–10</sub> entering the bloodstream through the alveolar barrier and causing CV adversities, is similar to that of PM<sub>2.5</sub>.

When assessing CV health concerns related to particulate matter, PM<sub>10</sub> should not be discredited—especially in communities of low socioeconomic status, as these populations are most vulnerable to the health effects of air pollution [56].

### 4.1. Strengths and limitations

This study had many strengths and some limitations. With regards to its strengths, the studies used in the systematic review and meta-analysis included data from a variety of population sizes and various geographical locations. This reduces the risk of bias and hence increases the reliability of the data. Another strength of this study is the use of strict screening protocols wherein two separate reviewers conducted the abstract and full-text screening, as well as the data-extraction. Furthermore, this study uses the NHLBI Quality Assessment Tool for Observational

Cohort and Cross-Sectional Studies for the risk of bias assessment, as well as the GRADE approach for quality of evidence assessment. These are robust tools used in advanced scientific literature, and further increase the reliability of the analyzed data. Another element which lends to this study's robustness is its stratification of MI risk based on time exposed to PM<sub>10</sub>. The implementation of lag intervals reduces the risk of time being a potential confounder.

This study also had limitations. The first one was the varying adjustment models utilized by included studies. There are multiple participant confounders (hypertension, diet, sleep, etc.) to consider since PM<sub>10</sub> association to MI risk is observational, which could explain potential differences in MI risk outcomes. Another factor to consider is the heterogeneity of the studies. All studies varied in study design, population and age characteristics, and the exposure assessment methods. We considered accounting for age as it is an important factor; however, we could not effectively conduct a meta-regression analysis of the outcomes due to insufficient data points per risk outcome.

## 4.2. Next steps

To enhance the precision and applicability of the findings, certain measures can be taken in the future. The compatibility and consistency of studies assessing MI risk can be increased by standardizing a specific risk outcome measure. While conversion algorithms exist for RR and OR, they depend on an estimated incidence variable (P0) which can constrain data synthesis validity. Compatibility and consistency of studies can also be improved by standardizing lag intervals. Currently, lag interval definitions are loosely defined from literature in the field. At times, studies define lags by day [26], sometimes by hour(s) [18], and other time intervals [16, 21]. As such, we based our lag intervals on what best preserves and applies to literature in the field, while also accommodating our dataset to ensure we had an adequate number of studies to meta-analyze each lag interval. Future research should seek to study the distinct risk of STEMI and NSTEMI. Understanding these MI subtypes' diverse relationships with PM<sub>10</sub> exposure can offer more specialized insights for prevention. Comprehensive adjustment models should also be more widely employed to ensure confounding variables are taken into account for all associative PM<sub>10</sub> studies. This will allow for more robust and reliable risk estimates. By taking these additional actions, researchers can improve the accuracy and application of the findings, and clarify the link between exposure to PM<sub>10</sub> and the risk of MI.

## 5. Conclusions

The results of this meta-analysis showed that there is an increased risk of MI with a 10 $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> exposure (OR = 1.01; 95% CI:1.00–1.02). Risk of MI is also marginal between PM<sub>10</sub> and PM<sub>2.5</sub> exposure. It is important to recognize and assess the CV impacts of PM<sub>10</sub> alongside other pollutants. This can help guide environmental policy, individual-level preventive measures, and global public health initiatives focused on lessening the disease burden of MI caused by PM<sub>10</sub> exposure.

## Supporting information

**S1 Fig. Lag 0 HR for MI after 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> exposure.**  
(PDF)

**S2 Fig. Lag 1 HR for MI after 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> exposure.**  
(PDF)

**S3 Fig. Protocol.**  
(PDF)

**S1 Table. Search strategy.**

(PDF)

**S2 Table. Adjusted confounding variables for studies included in the meta-analysis.**

(PDF)

**S3 Table. Study characteristics of remaining studies included in the review.**

(PDF)

**S4 Table. Methodological risk of bias assessment.**

(PDF)

**S5 Table. GRADE assessment for RR—Lag 1 (1–3 days [24–96hrs]).** O = Criteria Satisfied, -1 = Criteria Unsatisfied.

(PDF)

**S6 Table. GRADE assessment for RR—Lag 1 (1–3 days [24–96hrs]).** O = Criteria Satisfied, -1 = Criteria Unsatisfied.

(PDF)

**S7 Table. GRADE assessment for RR—Lag 2 (3+ days [96hrs+]).** O = Criteria Satisfied, -1 = Criteria Unsatisfied.

(PDF)

**S8 Table. GRADE assessment for OR—Lag 0 (Same day [0–24hrs]).** O = Criteria Satisfied, -1 = Criteria Unsatisfied.

(PDF)

**S9 Table. GRADE assessment for OR—Lag 1 (1–3 days [24–96hrs]).** O = Criteria Satisfied, -1 = Criteria Unsatisfied.

(PDF)

**S10 Table. GRADE assessment for OR—Lag 2 (3+ days [96hrs+]).** O = Criteria Satisfied, -1 = Criteria Unsatisfied.

(PDF)

**S11 Table. GRADE assessment for HR—Lag 0 (Same day [0–24hrs]).** O = Criteria Satisfied, -1 = Criteria Unsatisfied.

(PDF)

**S12 Table. GRADE assessment for HR—Lag 1 (1–3 days [24–96hrs]).** O = Criteria Satisfied, -1 = Criteria Unsatisfied.

(PDF)

**S13 Table. GRADE assessment for HR—Lag 2 (3+ days [96hrs+]).** O = Criteria Satisfied, -1 = Criteria Unsatisfied.

(PDF)

**S1 Checklist. PRISMA checklist.**

(PDF)

**Author Contributions****Conceptualization:** Kleiton Strobl, Syed Asad Irfan, Hassan Masood.



**Formal analysis:** Kleiton Strobl, Syed Asad Irfan.

**Investigation:** Kleiton Strobl, Syed Asad Irfan, Hassan Masood, Noor Latif.

**Methodology:** Kleiton Strobl, Syed Asad Irfan, Hassan Masood, Om Kurmi.

**Project administration:** Kleiton Strobl.

**Supervision:** Kleiton Strobl, Syed Asad Irfan, Om Kurmi.

**Validation:** Kleiton Strobl, Syed Asad Irfan, Hassan Masood, Om Kurmi.

**Visualization:** Kleiton Strobl, Syed Asad Irfan.

**Writing – original draft:** Kleiton Strobl, Syed Asad Irfan, Hassan Masood, Noor Latif.

**Writing – review & editing:** Kleiton Strobl, Syed Asad Irfan, Hassan Masood, Noor Latif, Om Kurmi.

## References

1. Hamanaka RB, Mutlu GM. Particulate matter air pollution: effects on the cardiovascular system. *Frontiers in endocrinology*. 2018 Nov 16; 9:680. <https://doi.org/10.3389/fendo.2018.00680> PMID: 30505291
2. Particle Pollution | Air | CDC [Internet]. [https://www.cdc.gov/air/particulate\\_matter.html](https://www.cdc.gov/air/particulate_matter.html)
3. Tornevi A, Olstrup H, Forsberg B. Short-term associations between PM10 and respiratory health effects in Visby, Sweden. *Toxics*. 2022 Jun 17; 10(6):333. <https://doi.org/10.3390/toxics10060333> PMID: 35736941
4. Thangavel P, Park D, Lee YC. Recent insights into particulate matter (PM<sub>2.5</sub>)-mediated toxicity in humans: an overview. *International journal of environmental research and public health*. 2022 Jun 19; 19(12):7511. <https://doi.org/10.3390/ijerph19127511> PMID: 35742761
5. Nemmar A, Hoet PM, Vanquickenborne B, Dinsdale D, Thomeer M, Hoylaerts MF, et al. Passage of inhaled particles into the blood circulation in humans. *Circulation*. 2002 Jan 29; 105(4):411–4. <https://doi.org/10.1161/hc0402.104118> PMID: 11815420
6. Farhadi Z, Abulghasem Gorgi H, Shabaninejad H, Aghajani Delavar M, Torani S. Association between PM 2.5 and risk of hospitalization for myocardial infarction: a systematic review and a meta-analysis. *BMC Public Health*. 2020 Dec; 20:1–2.
7. Luo C, Zhu X, Yao C, Hou L, Zhang J, Cao J, et al. Short-term exposure to particulate air pollution and risk of myocardial infarction: a systematic review and meta-analysis. *Environmental Science and Pollution Research*. 2015 Oct; 22:14651–62. <https://doi.org/10.1007/s11356-015-5188-x> PMID: 26298338
8. Zou L, Zong Q, Fu W, Zhang Z, Xu H, Yan S, et al. Long-term exposure to ambient air pollution and myocardial infarction: a systematic review and meta-analysis. *Frontiers in Medicine*. 2021 Mar 17; 8:616355. <https://doi.org/10.3389/fmed.2021.616355> PMID: 33816520
9. Mustafić H, Jabre P, Caussin C, Murad MH, Escolano S, Tafflet M, et al. Main air pollutants and myocardial infarction: a systematic review and meta-analysis. *Jama*. 2012 Feb 15; 307(7):713–21. <https://doi.org/10.1001/jama.2012.126> PMID: 22337682
10. Veritas Health Innovation. Covidence systematic review software [Internet]. Covidence. [cited 2023 Jul 03]. [www.covidence.org](http://www.covidence.org)
11. Moher D, Shamseer L, Clarke M, Ghersi D, Liberati A, Petticrew M, et al. Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. *Systematic reviews*. 2015 Dec; 4(1):1–9. <https://doi.org/10.1186/2046-4053-4-1> PMID: 25554246
12. Study quality assessment tools. National Heart, Lung, and Blood Institute. NHLBI [Internet]. <https://www.nhlbi.nih.gov/health-topics/study-qualityassessment-tools>
13. George A, Stead TS, Ganti L. What's the risk: differentiating risk ratios, odds ratios, and hazard ratios?. *Cureus*. 2020 Aug 26; 12(8). <https://doi.org/10.7759/cureus.10047> PMID: 32983737
14. Argacha JF, Collart P, Wauters A, Kayaert P, Lochy S, Schoors D, et al. Air pollution and ST-elevation myocardial infarction: a case-crossover study of the Belgian STEMI registry 2009–2013. *International journal of cardiology*. 2016 Nov 15; 223:300–5. <https://doi.org/10.1016/j.ijcard.2016.07.191> PMID: 27541680
15. Bard D, Kihal W, Schillinger C, Fermanian C, Segala C, Glorion S, et al. Traffic-related air pollution and the onset of myocardial infarction: disclosing benzene as a trigger? A small-area case-crossover study. *PLoS one*. 2014 Jun 16; 9(6):e100307. <https://doi.org/10.1371/journal.pone.0100307> PMID: 24932584



16. Bhaskaran K, Hajat S, Armstrong B, Haines A, Herrett E, Wilkinson P, et al. The effects of hourly differences in air pollution on the risk of myocardial infarction: case crossover analysis of the MINAP database. *Bmj*. 2011 Sep 20; 343. <https://doi.org/10.1136/bmj.d5531> PMID: 21933824
17. Buszman PE, Derbisz K, Kwasiborski P, Chrzęszcz P, Mularska M, Baron D, et al. Impact of air pollution on hospital patients admitted with ST-and non-ST-segment elevation myocardial infarction in heavily polluted cities within the European Union. *Cardiology Journal*. 2020; 27(5):541–7. <https://doi.org/10.5603/CJ.a2018.0156> PMID: 30566212
18. Cheng J, Tong S, Su H, Xu Z. Hourly air pollution exposure and emergency department visit for acute myocardial infarction: vulnerable populations and susceptible time window. *Environmental Pollution*. 2021 Nov 1; 288:117806. <https://doi.org/10.1016/j.envpol.2021.117806> PMID: 34329072
19. Claeys MJ, Coenen S, Colpaert C, Bilcke J, Beutels P, Wouters K, et al. Environmental triggers of acute myocardial infarction: results of a nationwide multiple-factorial population study. *Acta Cardiologica*. 2015 Dec 1; 70(6):693–701. <https://doi.org/10.2143/AC.70.6.3120182> PMID: 26717218
20. Collart P, Dramaix M, Levêque A, Coppieters Y. Short-term effects of air pollution on hospitalization for acute myocardial infarction: age effect on lag pattern. *International journal of environmental health research*. 2017 Jan 2; 27(1):68–81. <https://doi.org/10.1080/09603123.2016.1268678> PMID: 28002975
21. Davoodabadi Z, Soleimani A, Pourmoghaddas A, Hosseini SM, Jafari-Koshki T, Rahimi M, et al. Correlation between air pollution and hospitalization due to myocardial infarction. *ARYA atherosclerosis*. 2019 Jul; 15(4):161. PMID: 31819749
22. Downward GS, van Nunen EJ, Kerckhoffs J, Vineis P, Brunekreef B, Boer JM, et al. Long-term exposure to ultrafine particles and incidence of cardiovascular and cerebrovascular disease in a prospective study of a Dutch cohort. *Environmental health perspectives*. 2018 Dec 19; 126(12):127007. <https://doi.org/10.1289/EHP3047> PMID: 30566375
23. Huss A, Spoerri A, Egger M, Röösli M. Aircraft noise, air pollution, and mortality from myocardial infarction. *Epidemiology*. 2010 Nov 1:829–36. <https://doi.org/10.1097/EDE.0b013e3181f4e634> PMID: 20881600
24. Kim OJ, Lee SH, Kang SH, Kim SY. Incident cardiovascular disease and particulate matter air pollution in South Korea using a population-based and nationwide cohort of 0.2 million adults. *Environmental Health*. 2020 Dec; 19(1):1–2.
25. Konduracka E, Niewiara Ł, Guzik B, Kotynia M, Szolc P, Gajos G, et al. Effect of short-term fluctuations in outdoor air pollution on the number of hospital admissions due to acute myocardial infarction among inhabitants of Kraków, Poland. *Polskie Archiwum Medycyny Wewnętrznej = Polish Archives of Internal Medicine*. 2019; 129(2).
26. Kuźma Ł, Wańha W, Kralisz P, Kazmierski M, Bachórzewska-Gajewska H, Wojakowski W, et al. Impact of short-term air pollution exposure on acute coronary syndrome in two cohorts of industrial and non-industrial areas: A time series regression with 6,000,000 person-years of follow-up (ACS-Air Pollution Study). *Environmental Research*. 2021 Jun 1; 197:111154. <https://doi.org/10.1016/j.envres.2021.111154> PMID: 33872649
27. Lipsett MJ, Ostro BD, Reynolds P, Goldberg D, Hertz A, Jerrett M, et al. Long-term exposure to air pollution and cardiorespiratory disease in the California teachers study cohort. *American journal of respiratory and critical care medicine*. 2011 Oct 1; 184(7):828–35. <https://doi.org/10.1164/rccm.201012-2082OC> PMID: 21700913
28. Nuvolone D, Balzi D, Chini M, Scala D, Giovannini F, Barchielli A. Short-term association between ambient air pollution and risk of hospitalization for acute myocardial infarction: results of the cardiovascular risk and air pollution in Tuscany (RISCAT) study. *American journal of epidemiology*. 2011 Jul 1; 174(1):63–71. <https://doi.org/10.1093/aje/kwr046> PMID: 21597098
29. Puett RC, Schwartz J, Hart JE, Yanosky JD, Speizer FE, Suh H, et al. Chronic particulate exposure, mortality, and coronary heart disease in the nurses' health study. *American journal of epidemiology*. 2008 Nov 15; 168(10):1161–8. <https://doi.org/10.1093/aje/kwn232> PMID: 18835862
30. Royé D, Zarrabeitia MT, Fdez-Arroyabe P, Gutiérrez AA, Santurtún A. Role of apparent temperature and air pollutants in hospital admissions for acute myocardial infarction in the North of Spain. *Revista Española de Cardiología (English Edition)*. 2019 Aug 1; 72(8):634–40. <https://doi.org/10.1016/j.rec.2018.07.009> PMID: 30193931
31. Soleimani Z, Darvishi Bolorani A, Khalifeh R, Griffin DW, Mesdaghinia A. Short-term effects of ambient air pollution and cardiovascular events in Shiraz, Iran, 2009 to 2015. *Environmental Science and Pollution Research*. 2019 Mar 8; 26:6359–67. <https://doi.org/10.1007/s11356-018-3952-4> PMID: 30617889
32. Vidale S, Arnaboldi M, Bosio V, Corrado G, Guidotti M, Sterzi R, et al. Short-term air pollution exposure and cardiovascular events: a 10-year study in the urban area of Como, Italy. *International journal of cardiology*. 2017 Dec 1; 248:389–93. <https://doi.org/10.1016/j.ijcard.2017.06.037> PMID: 28711336

33. Yang J, Liu M, Cheng Q, Yang L, Sun X, Kan H, et al. Investigating the impact of air pollution on AMI and COPD hospital admissions in the coastal city of Qingdao, China. *Frontiers of Environmental Science & Engineering*. 2022 May; 16:1–3.
34. Yen CC, Chen PL. Effect of short-term exposure to fine particulate matter and particulate matter pollutants on triggering acute myocardial infarction and acute heart failure. *The American Journal of Cardiology*. 2022 Jul 15; 175:158–63. <https://doi.org/10.1016/j.amjcard.2022.03.062> PMID: 35595553
35. Zhang Q, Qi W, Yao W, Wang M, Chen Y, Zhou Y. Ambient particulate matter (PM<sub>2.5</sub>/PM<sub>10</sub>) exposure and emergency department visits for acute myocardial infarction in Chaoyang District, Beijing, China during 2014: a case-crossover study. *Journal of Epidemiology*. 2016 Oct 5; 26(10):538–45. <https://doi.org/10.2188/jea.JE20150209> PMID: 27064131
36. Zhu X, Qiu H, Wang L, Duan Z, Yu H, Deng R, et al. Risks of hospital admissions from a spectrum of causes associated with particulate matter pollution. *Science of The Total Environment*. 2019 Mar 15; 656:90–100. <https://doi.org/10.1016/j.scitotenv.2018.11.240> PMID: 30502738
37. Akbarzadeh MA, Khareshi I, Sharifi A, Yousefi N, Naderian M, Namazi MH, et al. The association between exposure to air pollutants including PM<sub>10</sub>, PM<sub>2.5</sub>, ozone, carbon monoxide, sulfur dioxide, and nitrogen dioxide concentration and the relative risk of developing STEMI: A case-crossover design. *Environmental research*. 2018 Feb 1; 161:299–303. <https://doi.org/10.1016/j.envres.2017.11.020> PMID: 29178978
38. Atkinson RW, Carey IM, Kent AJ, van Staa TP, Anderson HR, Cook DG. Long-term exposure to outdoor air pollution and incidence of cardiovascular diseases. *Epidemiology*. 2013 Jan 1:44–53. <https://doi.org/10.1097/EDE.0b013e318276ccb8> PMID: 23222514
39. Cheng MF, Tsai SS, Yang CY. Air pollution and hospital admissions for myocardial infarction in a tropical city: Kaohsiung, Taiwan. *Journal of Toxicology and Environmental Health, Part A*. 2009 Aug 31; 72(19):1135–40. <https://doi.org/10.1080/15287390903091756> PMID: 20077181
40. Cramer J, Jørgensen JT, Hoffmann B, Loft S, Bräuner EV, Prescott E, et al. Long-term exposure to air pollution and incidence of myocardial infarction: a Danish nurse cohort study. *Environmental health perspectives*. 2020 May 6; 128(5):057003. <https://doi.org/10.1289/EHP5818> PMID: 32438827
41. Kim H, Kim J, Kim S, Kang SH, Kim HJ, Kim H, et al. Cardiovascular effects of long-term exposure to air pollution: a population-based study with 900 845 person-years of follow-up. *Journal of the American Heart Association*. 2017 Nov 8; 6(11):e007170.
42. Kuźma Ł, Pogorzelski S, Struniawski K, Bachórzewska-Gajewska H, Dobrzycki S. Exposure to air pollution—a trigger for myocardial infarction? A nine-year study in Białystok—the capital of the Green Lungs of Poland (BIA-ACS registry). *International Journal of Hygiene and Environmental Health*. 2020 Aug 1; 229:113578. <https://doi.org/10.1016/j.ijheh.2020.113578> PMID: 32758862
43. Lee MW, Choi BG, Kim SW, Rha SW, Shim MS, Kim DJ, et al. Air pollution and short-term clinical outcomes of patients with acute myocardial infarction. *Clinical and Experimental Pharmacology and Physiology*. 2017 Jun; 44(6):631–8. <https://doi.org/10.1111/1440-1681.12755> PMID: 28345145
44. Liu X, Bertazzon S, Villeneuve PJ, Johnson M, Stieb D, Coward S, et al. Temporal and spatial effect of air pollution on hospital admissions for myocardial infarction: a case-crossover study. *Canadian Medical Association Open Access Journal*. 2020 Oct 1; 8(4):E619–26. <https://doi.org/10.9778/cmajo.20190160> PMID: 33037069
45. Pan HY, Cheung SM, Chen FC, Wu KH, Cheng SY, Chuang PC, et al. Short-term effects of ambient air pollution on ST-elevation myocardial infarction events: are there potentially susceptible groups?. *International journal of environmental research and public health*. 2019 Oct; 16(19):3760. <https://doi.org/10.3390/ijerph16193760> PMID: 31591299
46. Rasche M, Walther M, Schiffner R, Kroegel N, Rupprecht S, Schlattmann P, et al. Rapid increases in nitrogen oxides are associated with acute myocardial infarction: A case-crossover study. *European journal of preventive cardiology*. 2018 Nov 1; 25(16):1707–16. <https://doi.org/10.1177/2047487318755804> PMID: 29446990
47. Rodins V, Lucht S, Ohlwein S, Hennig F, Soppa V, Erbel R, et al. Long-term exposure to ambient source-specific particulate matter and its components and incidence of cardiovascular events—the Heinz Nixdorf Recall study. *Environment international*. 2020 Sep 1; 142:105854. <https://doi.org/10.1016/j.envint.2020.105854> PMID: 32590280
48. Sahlén A, Jungman P, Erlinge D, Chan MY, Yap J, Hausenloy DJ, et al. Air pollution in relation to very short-term risk of ST-segment elevation myocardial infarction: case-crossover analysis of SWEDE-HEART. *International Journal of Cardiology*. 2019 Jan 15; 275:26–30. <https://doi.org/10.1016/j.ijcard.2018.10.069> PMID: 30509372
49. Sen T, Astarcioglu MA, Asarcikli LD, Kilit C, Kafes H, Parspur A, et al. The effects of air pollution and weather conditions on the incidence of acute myocardial infarction. *The American Journal of*

- Emergency Medicine. 2016 Mar 1; 34(3):449–54. <https://doi.org/10.1016/j.ajem.2015.11.068> PMID: [26742457](https://pubmed.ncbi.nlm.nih.gov/26742457/)
50. Wang XD, Zhang XM, Zhuang SW, Luo Y, Kang S, Liu YL. Short-term effects of air pollution on acute myocardial infarctions in Shanghai, China, 2013–2014. *Journal of geriatric cardiology: JGC*. 2016 Feb; 13(2):132. <https://doi.org/10.11909/j.issn.1671-5411.2016.02.005> PMID: [27168738](https://pubmed.ncbi.nlm.nih.gov/27168738/)
  51. Wichmann J, Rosengren A, Sjöberg K, Barregard L, Sallsten G. Association between ambient temperature and acute myocardial infarction hospitalisations in Gothenburg, Sweden: 1985–2010. *PLoS one*. 2013 Apr 30; 8(4):e62059. <https://doi.org/10.1371/journal.pone.0062059> PMID: [23646115](https://pubmed.ncbi.nlm.nih.gov/23646115/)
  52. Wichmann J, Sjöberg K, Tang L, Haeger-Eugensson M, Rosengren A, Andersson EM, et al. The effect of secondary inorganic aerosols, soot and the geographical origin of air mass on acute myocardial infarction hospitalisations in Gothenburg, Sweden during 1985–2010: a case-crossover study. *Environmental Health*. 2014 Dec; 13(1):1–6.
  53. Wolf K, Schneider A, Breitner S, Meisinger C, Heier M, Cyrus J, et al. Associations between short-term exposure to particulate matter and ultrafine particles and myocardial infarction in Augsburg, Germany. *International journal of hygiene and environmental health*. 2015 Aug 1; 218(6):535–42. <https://doi.org/10.1016/j.ijheh.2015.05.002> PMID: [26013401](https://pubmed.ncbi.nlm.nih.gov/26013401/)
  54. Yu Y, Yao S, Dong H, Ji M, Chen Z, Li G, et al. Short-term effects of ambient air pollutants and myocardial infarction in Changzhou, China. *Environmental Science and Pollution Research*. 2018 Aug; 25:22285–93. <https://doi.org/10.1007/s11356-018-2250-5> PMID: [29808399](https://pubmed.ncbi.nlm.nih.gov/29808399/)
  55. Mc Namara K, Alzubaidi H, Jackson JK. Cardiovascular disease as a leading cause of death: how are pharmacists getting involved?. *Integrated pharmacy research and practice*. 2019 Feb 4:1–1. <https://doi.org/10.2147/IPRP.S133088> PMID: [30788283](https://pubmed.ncbi.nlm.nih.gov/30788283/)
  56. Hajat A, Hsia C, O'Neill MS. Socioeconomic disparities and air pollution exposure: a global review. *Current environmental health reports*. 2015 Dec; 2:440–50. <https://doi.org/10.1007/s40572-015-0069-5> PMID: [26381684](https://pubmed.ncbi.nlm.nih.gov/26381684/)