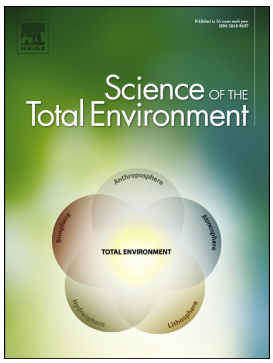

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PII: S0048-9697(20)38415-1

DOI: https://doi.org/10.1016/j.scitotenv.2020.144882

Reference: STOTEN 144882

To appear in: *Science of the Total Environment*

Received date: 18 August 2020

Revised date: 26 December 2020

Accepted date: 26 December 2020

Please cite this article as: M. Davoudi, F. Barjasteh-Askari, H. Amini, et al., Association of Suicide with Short-term Exposure to Air Pollution at Different Lag Times: A Systematic Review and Meta-analysis, *Science of the Total Environment* (2021), https://doi.org/10.1016/j.scitotenv.2020.144882

Association of Suicide with Short-term Exposure to Air Pollution at Different Lag Times: A Systematic Review and Meta-analysis

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Abstract

Background: Suicide is a major public health problem, with some environmental risk factors.

Objectives: This meta-analysis study explored the association between short-term exposure to air pollution and suicide mortality, with an emphasis on different lag times.

Methods: A systematic search was used to find relevant studies in databases including Scopus, Web of Knowledge, Pubmed, and Embase published up to 19 May 2020. The inclusion criteria included case-crossover or time-series studies assessing the association of criteria air pollutants with suicide mortality at different Lag Days of 0-7 (LD0 to LD7) and Cumulative Lags of 1-7 days (CL1 to CL7). Odds ratios (OR) were calculated with 95% confidence intervals (CI).

Results: Of 1,436 retrieved articles, 11 were eligible for data extraction, representing data on 283,550 suicides published between 2010 to 2019. The odds of suicide death increase with each 10 μg/m^3 increase in the mean concentrations of NO_2 at CL1 (1.013: 1.006-1.021), CL2 (1.028: 1.003-1.053), CL3 (1.035: 1.021-1.070), and LD2 (1.011: 1.001-1.022), SO_2 at CL1 (1.024: 1.014-1.034), CL2 (1.030: 1.012-1.048), CL3 (1.029: 1.009-1.049), and CL4 (1.027: 1.005-1.049), O_3 at CL6 (1.008: 1.000-1.016), PM_{10} at CL1 (1.004: 1.000-1.008), and $PM_{2.5}$ at CL1 (1.017: 1.003-1.031). Besides, the odds of suicide death increases with each 0.5 mg/m^3 increase in the mean concentration of CO at LD6 (1.005: 1.000-1.011). However, it decreased with increased O_3 exposure at LD3 (0.997: 0.994-1.000).

Conclusion: The study supports a positive association between air pollution and suicide mortality. No immediate risk was elucidated but the possible effects seem to be exerted

cumulatively.

Keywords: Air Pollutant; Air Pollution; Cumulative Exposure; Lag Time; Suicide

1. Introduction

Suicide is a serious public health problem. It is estimated that about 10 million people each year attempt suicide but survive. However, there are more than 800,000 documented suicide deaths annually worldwide (Bilén et al., 2013). The search for factors that increase the risk for suicidal behavior has looked at both personal and social variables that can impact the individual. The most common personal variable is a psychiatric disorder. It is found that the presence of a depressive disorder (both unipolar and bipolar depression) is a risk factor for suicide. At the social level, the role of social support (from family and friends) has been proven so that the absence of social support increases the risk of suicide. The role of the physical environment, however, has been relatively neglected.

The association of season with suicide was noted by Durkheim (1897) over one hundred years ago, with a peak of suicide found in the spring in most countries (Lester, 1979). Another environmental factor is a change in the weather. A recent research study by Bozsonyi et al. (2020) in a region of Hungary found that the amount of daily sunshine had an immediate, significant positive impact on the number of suicides. However, some environmental factors may be protective. For example, Barjasteh-Askari et al. (2020) found an association between the level of lithium in drinking water and lower suicide rates.

Atmospheric pollution is an environmental risk factor for many health problems (Dehghani et al., 2018; Dobaradaran et al., 2009). A wide variety of air pollutants, including gaseous and particulate compounds with organic and inorganic origins, can be emitted from industries, vehicles, power plants, etc. (Keramati et al., 2016; Sarkhosh et al., 2013; Sarkhosh et al., 2012).

Hospitals experience significantly more visits for cardiovascular and respiratory diseases shortly after exposure to air pollution (Dominici et al., 2006). Atmospheric air pollution may also trigger psychiatric disorders such as depression and suicide. Research has shown that the odds of emergency department visits for depression increase after exposure to ambient ozone (O_3) (Szyszkowicz et al., 2016). In another study, carbon monoxide (CO), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and particulate matter <10 μm (PM_{10}) significantly increased emergency department visits for suicide attempts (Szyszkowicz et al., 2010). Respirable particulate matter (PM_{10} and $PM_{2.5}$) can find its way to the brain from the lungs through the circulatory system or from the nose via the olfactory tract (Chen and Samet, 2013). Air pollutants can induce oxidative stress and generalized inflammation, leading to mental illness exacerbations including depression and self-harm behaviors (Gładka et al., 2018).

Several epidemiological studies have assessed the suicide death resulting from long-term or short-term exposure to air pollution. Long-term exposure studies usually assess the association between air pollution and suicide incidence over months or years. On the other hand, in short-term exposure, the event of air pollution can occur on the same day of suicide or a day in a short period (usually two weeks) preceding the suicide, representing respectively immediate and lagged risk of air pollution. Besides, it is plausible that air pollution acts cumulatively over a few consecutive days. To estimate the cumulative risk of short-term exposure to air pollution, epidemiological studies calculate the moving average concentrations of air pollutants measured on the same day of suicide and several days before the suicide. Associations in short terms can be quantified in time-series or case-crossover designs, while long-term exposure is assessed using cohort, cross-sectional, or case-control designs (Braithwaite et al., 2019).

Air pollution has been shown to predict suicide in the general population in both short-term and

long-term exposure studies. In a cohort study, an interquartile range increase in PM_{10}, NO_2, and SO_2 significantly increased the risk of suicide death (Min et al., 2018). A case-crossover study in 26 cities of South Korea (Lee et al., 2018) showed significant positive associations between some air pollutants and completed suicide at various lag days and cumulative lags. However, contrary results were observed in a recently published time-series analysis from Mexico (Astudillo-García et al., 2019). The associations between air pollution exposure and suicide may be affected by the concentrations of air pollutants, their variations in the study locations, and data periods (Lin et al., 2016). Therefore, a meta-analysis of various studies may be useful to estimate the pooled effect from the published studies. In a search of the literature, only two meta-analysis studies concerning the relationship between air pollution exposure and suicide were found, both of which focused only on the risk associated with particulate matter (Braithwaite et al., 2019; Gu et al., 2019). The study by Braithwaite et al. (2019) could locate only four eligible studies to estimate the pooled effect and focused on cumulative lags 0-1 days and 0-2 days. Although another study by Gu et al. (2019) pooled the results of seven studies, they combined the results of long-term and short-term evaluation studies and, thereby, failed to calculate the acute risk of air pollution exposure at various lag times. Therefore, the present study utilized a meta-analysis approach to explore the association between short-term exposure to both gaseous and particulate pollutants and suicide death at various lag times and cumulative lags.

2. Methods

2.1 Study Protocol

We followed the Cochrane Handbook for Systematic Reviews of Interventions (Higgins and Green, 2008) for the conduct of this study. We submitted the study protocol in the International Prospective Register of Systematic Reviews ((PROSPERO ID CRD42020173506). The present

report is organized according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement (Moher et al., 2009).

2.2 Search Strategy

We carried out a systematic search for studies of the relationship between air pollution exposure and suicide mortality in the general population. The search was conducted in Web of Knowledge (ISI), PubMed, Scopus, and Embase by MeSH and non-MeSH terms in the full text of journal articles published until 19 May 2020. The search strategy included the following keywords: suicide, air pollution, air pollutant, and atmospheric pollutant

2.3 Eligibility Criteria

The inclusion criteria included original articles assessing the association between short-term exposure to criteria air pollutants and suicide mortality in the general population. Based on the United States Environmental Protection Agency (USEPA), the criteria air pollutants include NO_2, SO_2, O_3, CO, PM_{10}, and $PM_{2.5}$ and are known for their global adverse effects on public health and the environment (Guxens et al., 2012). We focused on short-term exposure to assess the immediate, delayed, and cumulative risks of air pollution in short periods. Therefore, we selected only studies with time-stratified case-crossover designs or those using time-series analyses. Based on Lu and Zeger (2007), the case-crossover design can be considered equal to time-series analysis when both refer to a common exposure, such as in air pollution epidemiologic studies.

In this paper, suicide refers to the act of intentional self-poisoning and self-harm based on the International Statistical Classification of Diseases and Related Health Problems (ICD). Eligible studies were those that measured suicide mortality under ICD-9 codes E950.0-E958.9, ICD-10 codes X60-X69 (intentional self-poisoning), ICD-10 codes X70-X84 (intentional self-harm),

ICD-10 codes Y10-Y10.34 (undetermined intent), and ICD-10 code Y87.0 (sequelae of intentional self-harm). Studies that did not report on ICD codes but asserted "suicide death" or "complete suicide" in their methodology were also included in the study. However, we excluded studies assessing suicide attempts, suicidality, or self-harm without suicide intent, as well as studies without intended/extractable data.

2.4 Study Selection

The steps of study selection included the removal of duplicate records, the examination of titles/abstracts for possibly relevant studies, the examination of full texts for eligibility criteria, cross-checking between researchers to reach a consensus on study selection, quality assessment of select studies, and data extraction. Two authors (M. D and F. BA) independently examined the retrieved papers for the eligibility criteria. Disagreements were resolved by a third author (AH. M). Furthermore, we manually screened the reference lists of the relevant studies to find possibly missing papers from the electronic search.

2.5 Quality Assessment

The Newcastle-Ottawa Quality Assessment Scale (NOS) for cross-sectional studies (Aibibula et al., 2017) was used for the qualitative assessment of the studies selected. The NOS assesses the methodology of cross-sectional studies in the domains of selection, comparability, and outcome. It includes eight items, with a maximum attainable score of 10. A score of 50% or more suggests that the study has enough quality to be included in the data synthesis (Halpin and Ford, 2012).

2.6 Data Extraction

The data were gathered from the selected articles by two authors independently. They included authors' names, publication year, study place, study design, statistical models, data periods, air pollutants, lag times, suicide death numbers, and effect measure (Incidence Rate Ratio (IRR),

Relative Risk (RR), or Odds Ratio (RR) and Confidence Interval (CI)).

2.7 Statistical Analysis

The comprehensive meta-analysis software (CMA; version 2.2.064) was used to calculate an effect size for each study, expressed as OR and 95%CI. The pooled estimate was computed based on the random-effects model to account for the heterogeneity between the studies. The publication bias was assessed based on the symmetry/asymmetry of funnel plots in Duval and Tweedie's Trim and Fill method. In this method, based on the funnel plot of the study measure as a function of effect size, first, the missing studies were trimmed on the plot and then they were imputed to have a symmetric funnel plot. Finally, the method re-computed the effect size in a new, symmetric funnel plot. A leave-one-out sensitivity analysis was performed to check for the robustness of the pooled estimate. The analysis failed if the p-value of the pooled effect changed, from significant to insignificant and vice versa, by removing any study. P-values of <0.05 were acknowledged as statistically significant associations.

3. Results

3.1 Flow and Characteristics of Included Studies

Figure 1 shows the PRISMA 2009 flow diagram for the process of study selection. Briefly, the search retrieved 1,436 journal articles (Web of Science, n=118; Pubmed, n=202; Scopus, n=730; Embase, n=386); however, no additional studies were found in the manual search. Of the retrieved studies, 11 studies (Astudillo-García et al., 2019; Bakian et al., 2015; Casas et al., 2017; Fernández-Niño et al., 2018; Kim et al., 2010; Kim et al., 2018; Lee et al., 2018; Li et al., 2018; Lin et al., 2016; Ng et al., 2016; Yang et al., 2019b) were eligible for qualitative assessment. Table 1 summarizes the characteristics of the studies.

Insert Figure 1 and Table 1 here

The studies were published between 2010 and 2019 and they collected data on 283,550 complete suicides. There were seven studies from Asia, two from North America, one from South America, and one from Europe. The data periods ranged from one year (Kim et al., 2010) to 31 years (Kim et al., 2018). Nine out of 11 studies utilized time-stratified case-crossover designs and two studies used time-series analyses. In all studies, the exposure was criteria air pollutants (NO_2, SO_2, O_3, CO, PM_{10}, and $PM_{2.5}$) and the outcome was suicide mortality. The quality score for the articles, obtained using the NOS, ranged from 60% to 90%. Therefore, all the studies were used for the quantitative analysis.

3.2 Lag times

All the selected studies assessed the associated risk of suicide due to short-term exposure to air pollution at different lag times. The lag times referred to the time intervals from exposure to the outcome; that is, how many days elapsed from an air pollution event to the suicide. In the studies, Lag Days (LD) ranged from 0 to 7 days, assessing the suicide risk from air pollution on the day of suicide (LD0) to seven days preceding the suicide (LD7). Thus, LD0 represents the immediate risk, and LD1 to LD7 indicates the delayed risk. Besides, the risks from Cumulative Lag (CL) exposures were assessed based on the moving average of air pollutant concentrations on the suicide day and one to seven days before the suicide (CL1 to CL7, respectively). The studies assessed the association of air pollution with suicide at LD0 up to LD7 and/or CL0 up to CL7. One study (Kim et al., 2018) extended the cumulative lag period to CL9.

3.3 Overlap Resolving and Effect Measure

There were overlaps in data between studies that shared the same locations, the same data periods, the same air pollutants, and the same lag times. This was a matter of concern between Kim et al. (2010) and Lee et al. (2018) studies, Kim et al. (2018) and Lee et al. (2018) studies,

Kim et al. (2010) and Kim et al. (2018) studies, and between Kim et al. (2018) and Ng et al. (2016) studies. In each paired comparison, we gave an advantage to the study that was extended over a larger period and removed the other one. The detailed procedure is presented in Table S1 the Supplemental File.

Among 11 selected studies, the effect measure was OR in seven studies, calculated with conditional logistic regression models. Two studies reported RR and two other studies reported IRR, all of which from conditional Poisson regression models. As suicide mortality can be considered a rare event, we assumed OR, RR, and IRR to be equal in calculations. The suicide risk was assessed in association with an IQR increase in air pollutant concentrations in seven studies, 20% increases in the mean concentration in two studies, and 10 $\mu g/m^3$ increases in the mean concentration in one study. Before calculating a pooled effect, we converted the measures so as to indicate the odds of suicide per each 10 $\mu g/m^3$ increase in the mean concentration of pollutants NO_2, SO_2, O_3, PM_{10}, and $PM_{2.5}$ and 0.5 mg/m^3 for CO.

3.4 Association Between Air Pollution and Suicide

The associations of suicide with air pollutants including NO_2, SO_2, O_3, CO, PM_{10}, and $PM_{2.5}$ were assessed separately based on lag days (LD) and cumulative lags (CL). They were LD0 to LD7 and CL1 to CL7 (for NO_2, SO_2, O_3, and PM10), LD0 to LD7 and CL7 (for CO), and LD0 to LD7 and CL1 to CL3, as well as CL7 (for $PM_{2.5}$). Figure 2 shows only the significant associations ($P<0.05$). The detailed results are presented in Figures S1 to S6 in the Supplemental File.

As shown, the odds of suicide increase with each 10 $\mu g/m^3$ increase in the mean atmospheric concentration of NO_2 at CL1 (OR: 1.013; 95%CI: 1.006-1.021), CL2 (OR: 1.028; 95%CI: 1.003-1.053), CL3 (OR: 1.035; 95%CI: 1.001-1.070), and LD2 (OR: 1.011; 95%CI: 1.001-1.022).

Besides, suicide death increases with exposure to each 10 μg/m^3 increase in the mean concentration of SO_2 at CL1 (OR: 1.024; 95 %CI: 1.014-1.034), CL2 (OR: 1.030; 95%CI: 1.012-1.048), CL3 (OR: 1.029; 95%CI: 1.009-1.049), and CL4 (OR: 1.027; 95%CI: 1.005-1.049). Concerning the risk of suicide death due to exposure to ground-level O_3, there was a significant positive association at CL6 (OR: 1.008; 95%CI: 1.000-1.016) and a significant negative association at LD3 (OR: 0.997; 95%CI: 0.994-1.000). The odds of suicide increase with every 0.5 mg/m^3 increase in the mean CO concentration in the atmosphere at LD6 (OR: 1.005; 95%CI: 1.000-1.011). Finally, exposure to each 10 μg/m^3 increase in the concentration of PM_{10} at CL1 (OR: 1.004; 95%CI: 1.000-1.008) and $PM_{2.5}$ at CL1 (OR: 1.017; 95%CI: 1.003-1.031) increased the risk of suicide death in the general population.

Insert Figure 2 here

3.5 Sensitivity Analysis

The one-study remove sensitivity analysis was applied to investigate the robustness of the results. The findings are presented in Figures S7 to S12 in the Supplemental File. Concerning NO_2 exposure, significant associations with suicide are not robust at CL1, CL2, CL3, and LD2, as the removal of any study changes the associations into insignificant. The association at LD0 turns significant if the study by Astudillo-García et al. (2019) is removed. However, the insignificant associations at CL4, CL5, CL6, CL7, LD1, LD3, LD4, LD5, LD6, and LD7 are robust. Concerning SO_2 exposure, significant associations were robust at CL1 and CL2 but not CL3 and CL4 in the sensitivity analysis. On the other hand, the insignificant associations are made significant by removing the study by Astudillo-García et al. (2019) from LD0 and LD1. Besides, removing the study by Lin et al. (2016) from LD4 leads to a significant negative association between SO_2 and suicide. The insignificant associations at CL5, CL6, CL7, LD2,

LD3, LD5, LD6, and LD7 are robust. Regarding O_3 exposure, none of the significant associations at CL6 and LD3 are robust. However, insignificant associations at CL1 to CL5 turn significant if the study by Lee et al. (2018) is removed. The same is true if the study by Astudillo-García et al. (2019) is removed from LD0. The insignificant associations at CL7, LD1, LD2, and LD4 to LD7 are robust. Regarding CO exposure, the only significant association at LD6 is not robust. Besides, the insignificant associations at CL7, LD0, LD1, and LD2 turn significant by the removal of the study by Fernández-Niño et al. (2018). The same is true if the study by Lee et al. (2018) is removed from LD4. However, the insignificant associations at LD3, LD5, and LD7 are robust. Concerning PM_{10} exposure, the only significant association at CL1 is not robust in the sensitivity analysis. However, the removal of the study by Lee et al. (2018) from CL3, LD2, and LD7 and the study by Lin et al. (2016) from LD0 turns the insignificant associations into significant ones. However, the insignificant associations are robust at CL2, CL4 to CL7, LD1, and LD3 to LD6. Regarding $PM_{2.5}$ exposure, the only significant association at CL1 is not robust. Besides, the insignificant association at LD4 turns to significant by removing the study by Astudillo-García et al. (2019). However, the insignificant associations at CL2 to CL4, LD0 to LD3, and LD5 to LD7 are robust.

3.6 Publication Bias

The results of the publication bias assessment are shown in Table S2 in the Supplemental File. The test detected the number of trimmed studies ranging from 0 to 3. Publication bias existed if trimmed studies >0. This condition was fulfilled in the majority of the assessments. However, just a small number of the assessments (n=4) showed a substantial change in the 95% confidence interval width (to encompass or exclude one) when the imputed studies were included. This shows that if trimmed studies are added, insignificant associations will be made significant, or

vice versa. NO_2 exposure at CL1, SO_2 exposure at CL1, CL2, and CL3, O_3 exposure at LD3, and $PM_{2.5}$ exposure at CL1 remained significantly associated with suicide death even if trimmed studies, if any, were imputed. The publication bias with the Duval and Tweedie's Trim and Fill test was not applicable to lag times that had fewer than three studies for meta-analysis.

4. Discussion

To our knowledge, this is the most comprehensive analysis of the association of air pollution exposure with suicide mortality risk, focusing on all criteria air pollutants at different lag times. The analysis showed that the odds of suicide death increase with each 10 $\mu g/m^3$ increase in the mean atmospheric concentrations of NO_2 at cumulative lags of 0-1 days (1.013: 1.006-1.021), 0-2 days (1.028: 1.003-1.053), 0-3 days (1.035: 1.001-1.070), and lag day of 2 (1.011: 1.001-1.022), SO_2 at cumulative lags of 0-1 days (1.024: 1.014-1.034), 0-2 days (1.030: 1.012-1.048), 0-3 days (1.029: 1.009-1.049), and 0-4 days (1.027: 1.005-1.049), O_3 at cumulative lag of 0-6 days (1.008: 1.000-1.016), PM_{10} at cumulative lag of 0-1 days (1.004: 1.000-1.008), and $PM_{2.5}$ at cumulative lag of 0-1 days (1.017: 1.003-1.031). Besides, the odds of suicide death increases with each 0.5 mg/m^3 increase in the mean concentration of CO at lag day of 6 (1.005: 1.000-1.011). The only significant inverse association was observed for O_3 at lag day of 3 (0.997: 0.994-1.000). Therefore, it seems that air pollutants can exert their acute effects cumulatively on complete suicide in the general population. This finding emphasizes the crucial need for measures to avoid air pollution exposure on several consecutive days. However, we found no immediate risk, as suicide death was not correlated with the increased concentrations of air pollutants on the same day of suicide (LD0). The possible lagged effects may provide the chance of intervening in periods of air pollution to prevent the associated risk of suicide incidence. The lagged effects can also be used to explain the involved mechanisms.

Air pollution exposure can lead people to commit suicide by deteriorating their physical and mental health. Air pollutants can penetrate the thoracic airways and reduce respiratory function (Roy and Goh, 2019). This, in turn, decreases the level of oxygen saturation and induces oxidative stress and hypoxia (Lee et al., 2019). Hypoxia can result in a deficit in serotonin, a neurotransmitter with a possible role in the neurobiology of suicide. In a cohort study, Riblet et al. (2019 showed that patients who had the markers for hypoxia were four times more likely to die of suicide. The central nervous system may also be affected by air pollution. This is especially the case for neurotoxic particles such as lead, mercury, and manganese. In this mechanism, air pollutants exert neuroinflammatory effects due to the release of proinflammatory cytokines, which, in turn, leads to changes in the brain, for example, altered levels of neurotransmitter cytokines (Zhao et al., 2019). Therefore, some mental and behavioral changes may appear, including depression, aggressive behaviors, and suicide. However, neurotoxicity usually requires long-term exposures and may not explain the acute effects of air pollutants as measured in the current study. Therefore, the exact mechanisms remain to be elucidated. Studies have also shown associations between air pollution exposure and the worsening of chronic diseases such as cardiovascular and respiratory disease, in addition to the increased risk of suicide (Kye and Park, 2017; Liu et al., 2017). Kim et al. (2010) reported an 18.9% increase in suicide risk among cardiovascular disease patients, while such an association was not found among their healthy peers. Some depression-inducing effects are also plausible for air pollutants while depression is a strong predictor of suicide (Cummins et al., 2015). Research has shown associations between ambient air pollution and emergency room visits for depressive symptoms (Braithwaite et al., 2019; Brokamp et al., 2019). Depression can also develop as a reaction to the adverse effect of air pollution on underlying diseases including cardiovascular and respiratory

disease (Novak et al., 2016).

Although we found several significant associations between atmospheric air pollutants and suicide death, only two of them (SO_2 at CL1 and CL2) were robust in the sensitivity analysis. Besides, there was significant bias in the publication of studies. These show the need for more reports based on well-designed studies. A limitation of the current research is that the majority of the studies included in the meta-analysis were from non-temperate zone Asian countries. Therefore, further research is needed to conduct in other regions of the world. The small number of studies restricted us to carry out subgroup analyses for possible causes of heterogeneity between the studies. Besides, the examined studies made some unique adjustments mostly for meteorological factors including air pressure, temperature, humidity, precipitation, duration of sunlight, and dew point temperature. These adjustments are very important, as meteorological factors seem to be significant predictors of suicide (Kim et al., 2019; Qian et al., 2008). Fernández-Niño et al. (2018) showed that controlling for temperature, precipitation, humidity, and holidays turned significant negative associations between air pollution exposure and suicide into non-significant associations. Air pollutants can also play a confounding or modifying role in the effect of other pollutants. To remove this effect, some studies (Kim et al., 2018; Lee et al., 2018; Ng et al., 2016; Yang et al., 2019a) utilized two-pollutant models versus single-pollutant models. They showed that some associations weakened after adjusting for a second air pollutant. For example, the significant positive association for PM_{10} disappeared after adding SO_2 and NO_2 into the model (Kim et al., 2018). Therefore, the interactive effects of air pollutants merit more investigation. Finally, as correlational studies are not appropriate to elucidate cause and effect, we were limited in the adequate explanation of the results. It is also noteworthy that the possible effects of air pollution on mental health aspects may be due to contaminants that emerge in the

atmosphere concurrently with criteria air pollutants but are not measured routinely.

Of the strengths of the study, we only selected case-crossover and time-series studies. This allowed adjustments for confounders including day-of-week, public holidays, seasonality, and long-term time trends. Therefore, the studies were homogenous in terms of some important confounders. We also resolved the overlap of data that existed in some studies from the same locations, including South Korea and Japan. We suggest exploring the relationship between suicide and long-term exposure to air pollution in future meta-analyses. Besides, future studies are encouraged to assessing the combinatory effects of air pollutants and aeroallergens, as evidence supports that suicide is also triggered by aeroallergens (Ragguett et al., 2017).

5. Conclusions

Overall, our meta-analysis supports the positive association between transient increases in air pollutants and suicide mortality in the general population. As an important finding, a 10 $\mu g/m^3$ increase in the average concentration of air pollutants NO_2, SO_2, PM_{10}, and $PM_{2.5}$ on the same day of suicide and one day preceding the suicide (CL1) well explained the rates of suicide death. These results are valuable as they represent the data of completed suicides from various locations with different geographic, socioeconomic, environmental, and cultural characteristics, as well as emission sources. The findings could be used for designing epidemiologic studies in the future. Moreover, the study highlights the need for policies and programs to avoid consecutive days of air pollution exposure to reduce the risk of suicide.

Acknowledgment

The authors would like to thank the authors of relevant papers who responded to emails and provided us with additional information on their work.

Authors' Contribution

AmirHossein Mahvi: Conceptualization, Critical revision, and editing of the manuscript

Fateme Barjasteh-Askari: Initial search, data extraction, manuscript drafting, data analysis, and interpretation

Mojtaba Davoudi: Initial search, data extraction, manuscript drafting, data analysis, and interpretation

Homayoun Amini: Critical revision and editing of the manuscript

David Lester: Critical revision and editing of the manuscript

Vahid Ghavami: Data analysis, interpretation, and critical revision of the manuscript
Mohammad Rezvani Ghalhari: Manuscript preparation

Funding/Support

The study was funded by the Institute for Environmental Research, Tehran University of Medical Sciences, Tehran, Iran (Grant# 48683). The funder had no role in study design, data collection, and analysis, decision to publish, or preparation of the manuscript.

Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Table 1. Characteristics of Studies Assessing Association between Suicide Death and Air Pollution in Total Population

No.	**Author, Year**	**Study Place**	**Study design/model**	**Data Period**	**Criteria Air Pollutants**	**Lag Times**	**Suicide Death Number**	**Effect measure**	**Significant Associations (p<0.05)**	**Quality Scores**
1	Kim et al. 2010	Seven cities, South Korea	Case-crossover / Conditio	2004	PM_{10}, $PM_{2.5}$	LD0 to LD3;	4341	OR per IQR increa	PM_{10} at LD1, LD2, CL1,	80%

			nal logistic regressio n			CL1 to CL3		ses in Mean	CL2, CL3 $PM_{2.5}$ at LD1	
2	Bakian et al. 2015	Utah, USA	Case-crossover / Conditio nal logistic regressio n	2000 - 2010	NO_2, SO_2, PM_{10}, $PM_{2.5}$	LD0 to LD3 ; CL1 to CL3	1546	OR per IQR increa ses in Mean	NO_2 at LD2, LD3, CL3 $PM_{2.5}$ at LD2	90%
3	Lin et al. 2016	Guangz hou, China	Case-crossover / Conditio nal logistic regressio n	2003 - 2012	NO_2, SO_2, PM_{10}	LD0 to LD7 ; CL1 to CL7	1550	OR per IQR increa ses in Mean	NO_2 at LD0, CL1, CL2, CL3 SO_2 at LD0, CL1 PM_{10} at LD0, CL1, CL2	90%
4	Ng et al. 2016	Tokyo, Japan	Case-crossover / Conditio nal logistic regressio n	2001 - 2011	NO_2, SO_2, $PM_{2.5}$	LD0 to LD3 ; CL1 to CL3	29939	OR per IQR increa ses in Mean	None	80%

5	Casas et al. 2017	Belgium	Case-crossover / Conditional logistic regression	2002 - 2011	O_3, PM_{10}	LD0; CL1 to CL6	20533	OR per 10 µg/m3 increase in mean	O_3 at LD0, CL1, CL2, CL3, CL4, CL5	90%
6	Fernández-Niño et al. 2018	Four cities, Colombia	Time-series/ Conditional Poisson regression	2011 - 2014	NO_2, SO_2, O_3, CO, PM_{10}, $PM_{2.5}$	LD0 to LD7; CL7	1942	IRR per 20% increase in mean; 10 µg/m3 for PM_{10}; 5 µg/m3 for $PM_{2.5}$	NO_2 at LD4 CO at LD4 PM_{10} at LD4 $PM_{2.5}$ at LD4	70%
7	Kim et al. 2018	10 Cities, Northeast Asia	Case-crossover / Conditional Poisson regression	Korea 2001 - 2010; Japan	NO_2, SO_2, PM_{10}, $PM_{2.5}$	LD0; CL1 to CL9	134811	RR per IQR increases in Mean	NO_2 at LD0 SO_2 at LD0, CL1, CL2, CL3, CL4	90%

				1979 - 2009 ; Taiwan 1994 - 2007					PM_{10} at LD0, CL1, CL2, CL3	
8	Lee et al. 2018	26 cities, South Korea	Case-crossover / Conditional logistic regression	2002 - 2013	NO_2, SO_2, O_3, CO, PM_{10}	LD0 to LD7 ; CL1 to CL7	73445	OR per IQR increases in Mean	NO_2 at LD0, LD1, CL1, CL2, CL3 SO_2 at LD0, CL1, CL2, CL3 O_3 at LD0, LD5 CO at LD0, LD1, CL1, CL2, CL3, CL4, CL6,	80%

									CL7	
9	Li et al. 2018	Beijing, China	Case-crossover / Conditional Poisson regression	2009 - 2011	$PM_{2.5}$	CL1, CL7	1148	RR per 10 µg/m3 increase in mean	$PM_{2.5}$ at CL1	60%
10	Astudillo-García et al. 2019	Mexico City, Mexico	Time-series/ Conditional Poisson regression	2000 - 2016	NO_2, SO_2, O_3, PM_{10}, $PM_{2.5}$	LD0 to LD7	12294	IRR per 20% increase in mean; 10 µg/m3 for PM_{10}; 5 µg/m3 for $PM_{2.5}$	NO_2 at LD0*, LD7* SO_2 at LD3*, LD4, LD7* O_3 at LD3*, LD6*, LD7* PM_{10} at LD7*	70%
11	Yang et al. 2019A	Taipei, Taiwan	Case-crossover / Conditional logistic regression	2004 - 2008	O_3	CL2	2001	OR per IQR increases in Mean	O_3 at CL2[#]	60%

* significant negative associations (otherwise, significant positive associations); # calculated in top groups by the current authors; NO_2: Nitrogen dioxide; SO_2: Sulfur dioxide; O_3: Ozone; CO: Carbon monoxide; PM_{10}: Particulate matter <10 μm; $PM_{2.5}$: Particulate matter <2.5 μm; LD: Lag day; CL: Cumulative lag; OR: Odds ratio; IRR: Incidence rate ratio; RR: Risk ratio; IQR: Interquartile range

Fig. 1. PRISMA 2009 flow diagram of the study

Fig. 2. Suicide risks significantly associated with criteria air pollutants at various lag times in the general population. The values are OR with 95%CI per 10 μg/m^3 increase in NO_2, SO_2, O_3, PM_{10}, and $PM_{2.5}$ and 0.5 mg/m^3 increase in CO; NO_2: Nitrogen dioxide; SO_2: Sulfur dioxide; O_3: Ozone; CO: Carbon monoxide; PM_{10}: Particulate matter <10 μm; $PM_{2.5}$: Particulate matter <2.5 μm; LD: Lag day; CL: Cumulative lag

Graphical abstract

Highlights

- Air pollution is associated with an increased risk of suicide death in the general population.
- The possible effects of air pollutants seem to be exerted on suicide cumulatively.
- NO_2, SO_2, PM_{10}, and $PM_{2.5}$ at the cumulative lag of 0-1 days increase the risk of suicide.
- It seems that air pollution possesses no immediate risk of suicide mortality.

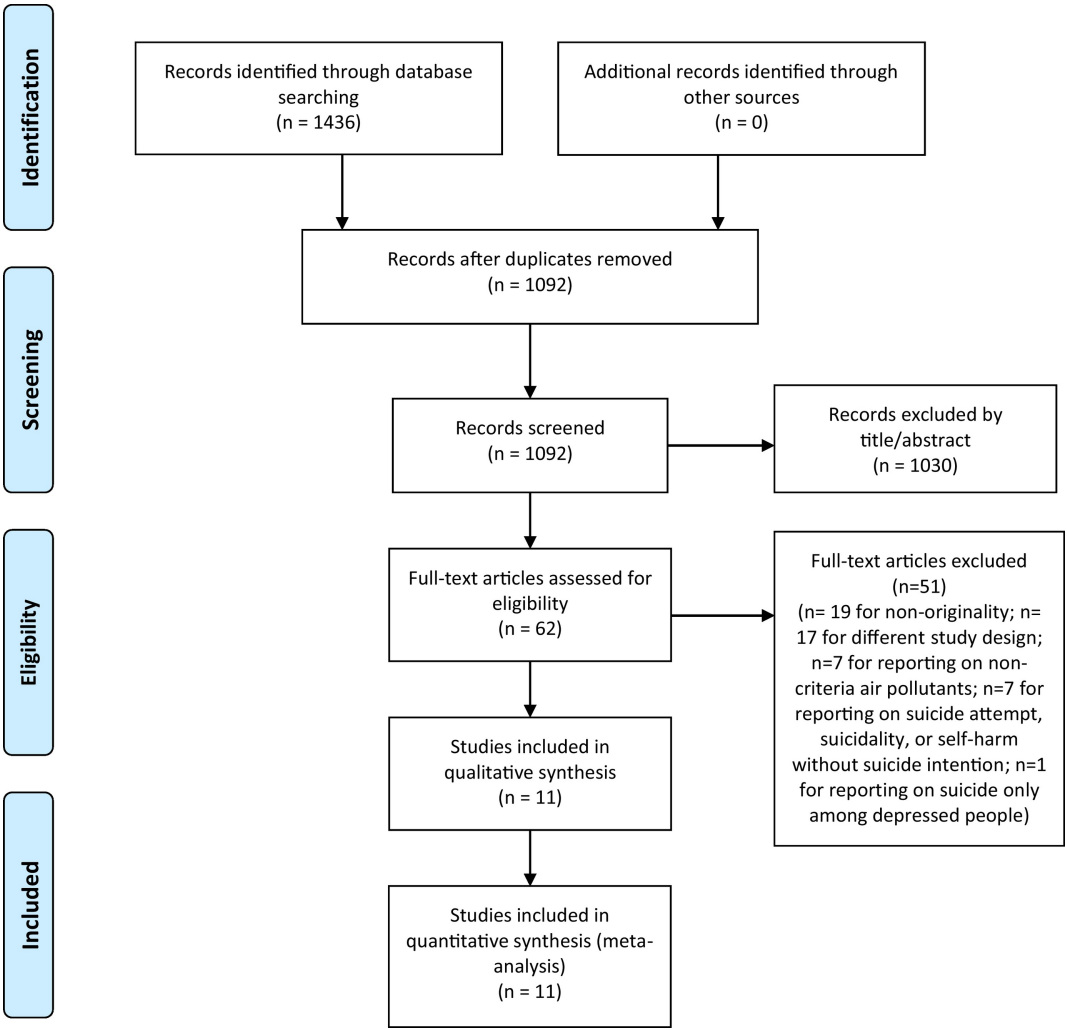
Identification
Screening
Eligibility
Included
Records identified through database searching (n = 1436)
Additional records identified through other sources (n = 0)
Records after duplicates removed (n = 1092)
Records screened (n = 1092)
Records excluded by title/abstract (n = 1030)
Full-text articles assessed for eligibility (n = 62)
Full-text articles excluded (n=51) (n= 19 for non-originality; n= 17 for different study design; n=7 for reporting on non-criteria air pollutants; n=7 for reporting on suicide attempt, suicidality, or self-harm without suicide intention; n=1 for reporting on suicide only among depressed people)
Studies included in qualitative synthesis (n = 11)
Studies included in quantitative synthesis (meta-analysis) (n = 11)

Figure 1

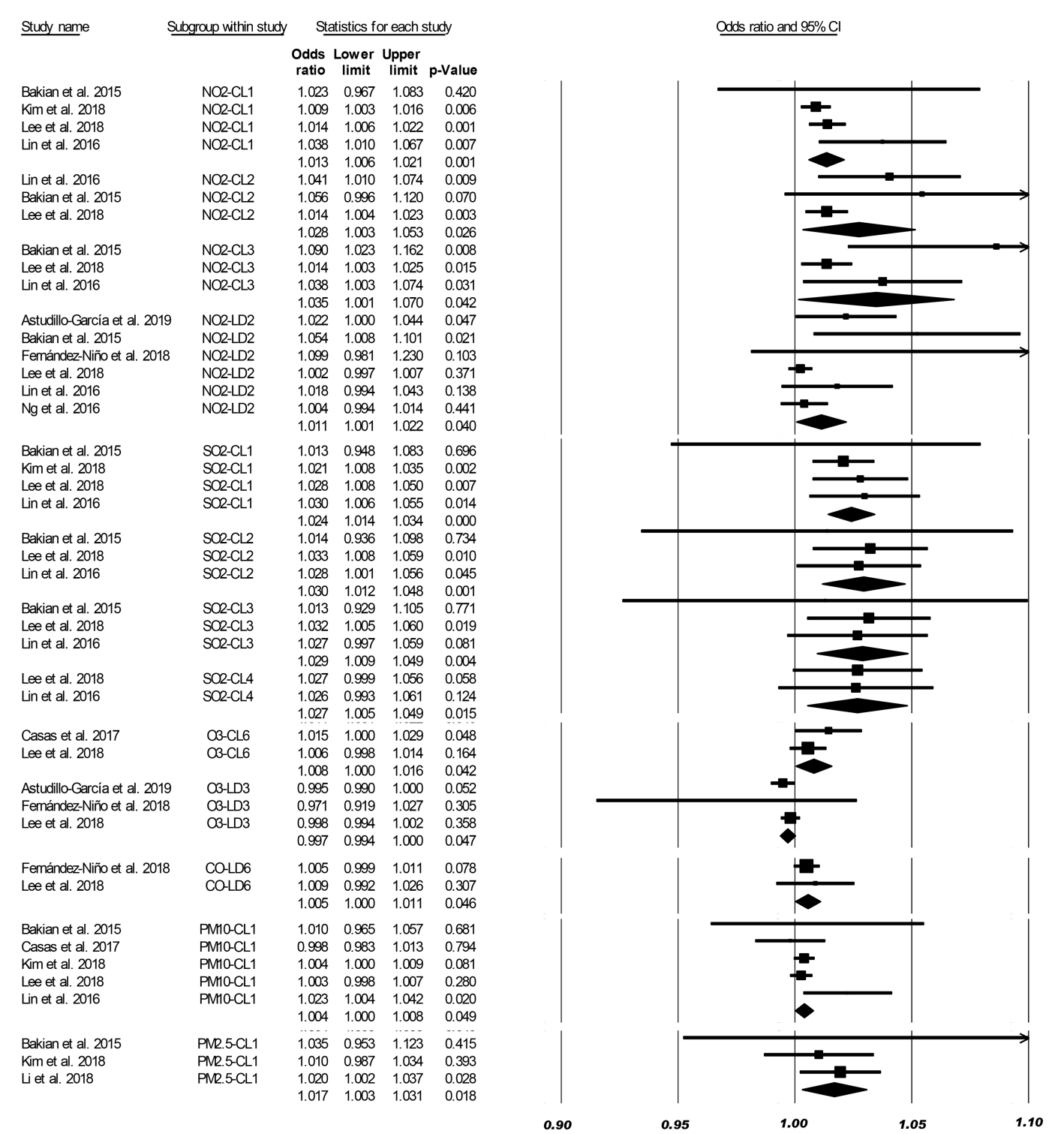
Study name
Subgroup within study
Statistics for each study
Odds ratio
Lower limit
Upper limit
p-Value
Odds ratio and 95% CI
Bakian et al. 2015 NO2-CL1 1.023 0.967 1.083 0.420
Kim et al. 2018 NO2-CL1 1.009 1.003 1.016 0.006
Lee et al. 2018 NO2-CL1 1.014 1.006 1.022 0.001
Lin et al. 2016 NO2-CL1 1.038 1.010 1.067 0.007
1.013 1.006 1.021 0.001
Lin et al. 2016 NO2-CL2 1.041 1.010 1.074 0.009
Bakian et al. 2015 NO2-CL2 1.056 0.996 1.120 0.070
Lee et al. 2018 NO2-CL2 1.014 1.004 1.023 0.003
1.028 1.003 1.053 0.026
Bakian et al. 2015 NO2-CL3 1.090 1.023 1.162 0.008
Lee et al. 2018 NO2-CL3 1.014 1.003 1.025 0.015
Lin et al. 2016 NO2-CL3 1.038 1.003 1.074 0.031
1.035 1.001 1.070 0.042
Astudillo-García et al. 2019 NO2-LD2 1.022 1.000 1.044 0.047
Bakian et al. 2015 NO2-LD2 1.054 1.008 1.101 0.021
Fernández-Niño et al. 2018 NO2-LD2 1.099 0.981 1.230 0.103
Lee et al. 2018 NO2-LD2 1.002 0.997 1.007 0.371
Lin et al. 2016 NO2-LD2 1.018 0.994 1.043 0.138
Ng et al. 2016 NO2-LD2 1.004 0.994 1.014 0.441
1.011 1.001 1.022 0.040
Bakian et al. 2015 SO2-CL1 1.013 0.948 1.083 0.696
Kim et al. 2018 SO2-CL1 1.021 1.008 1.035 0.002
Lee et al. 2018 SO2-CL1 1.028 1.008 1.050 0.007
Lin et al. 2016 SO2-CL1 1.030 1.006 1.055 0.014
1.024 1.014 1.034 0.000
Bakian et al. 2015 SO2-CL2 1.014 0.936 1.098 0.734
Lee et al. 2018 SO2-CL2 1.033 1.008 1.059 0.010
Lin et al. 2016 SO2-CL2 1.028 1.001 1.056 0.045
1.030 1.012 1.048 0.001
Bakian et al. 2015 SO2-CL3 1.013 0.929 1.105 0.771
Lee et al. 2018 SO2-CL3 1.032 1.005 1.060 0.019
Lin et al. 2016 SO2-CL3 1.027 0.997 1.059 0.081
1.029 1.009 1.049 0.004
Lee et al. 2018 SO2-CL4 1.027 0.999 1.056 0.058
Lin et al. 2016 SO2-CL4 1.026 0.993 1.061 0.124
1.027 1.005 1.049 0.015
Casas et al. 2017 O3-CL6 1.015 1.000 1.029 0.048
Lee et al. 2018 O3-CL6 1.006 0.998 1.014 0.164
1.008 1.000 1.016 0.042
Astudillo-García et al. 2019 O3-LD3 0.995 0.990 1.000 0.052
Fernández-Niño et al. 2018 O3-LD3 0.971 0.919 1.027 0.305
Lee et al. 2018 O3-LD3 0.998 0.994 1.002 0.358
0.997 0.994 1.000 0.047
Fernández-Niño et al. 2018 CO-LD6 1.005 0.999 1.011 0.078
Lee et al. 2018 CO-LD6 1.009 0.992 1.026 0.307
1.005 1.000 1.011 0.046
Bakian et al. 2015 PM10-CL1 1.010 0.965 1.057 0.681
Casas et al. 2017 PM10-CL1 0.998 0.983 1.013 0.794
Kim et al. 2018 PM10-CL1 1.004 1.000 1.009 0.081
Lee et al. 2018 PM10-CL1 1.003 0.998 1.007 0.280
Lin et al. 2016 PM10-CL1 1.023 1.004 1.042 0.020
1.004 1.000 1.008 0.049
Bakian et al. 2015 PM2.5-CL1 1.035 0.953 1.123 0.415
Kim et al. 2018 PM2.5-CL1 1.010 0.987 1.034 0.393
Li et al. 2018 PM2.5-CL1 1.020 1.002 1.037 0.028
1.017 1.003 1.031 0.018
0.90
0.95
1.00
1.05
1.10

Figure 2