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Does air pollution trigger suicide? A case-crossover analysis of suicide deaths over the life span.

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Abstract

Background: In addition to underlying health disorders and socio-economic or community factors, air pollution may trigger suicide mortality. This study evaluates the association between short-term variation in air pollution and 10 years of suicide mortality in Belgium.

Methods: In a bidirectional time-stratified case-crossover design, 20,533 suicide deaths registered between January 1st 2002 and December 31st 2011 were matched by temperature with control days from the same month and year. We used municipality-level air pollution [particulate matter (PM₁₀) and O₃ concentrations] data and meteorology data. We applied conditional logistic regression models adjusted for duration of sunshine and day of the week to obtain odds ratios (OR) and their 95% CI for an increase of 10µg/m³ in pollutant concentrations over different lag periods (lag 0, 0-1, 0-2, 0-3, 0-4, 0-5, and 0-6 days). Effect modification by season and age was investigated by including interaction terms.

Results: We observed significant associations of PM₁₀ and O₃ with suicide during summer (OR ranging from 1.02 to 1.07, p-values<0.05). For O₃, significant associations were also observed during spring and autumn. Age significantly modified the associations with PM₁₀, with statistically significant associations observed only among 5 to 14 year old children (lag 0-6: OR=1.45; 95%CI: 1.03-2.04) and ≥85 years old (e.g. lag 0-4: OR=1.17; 95%CI: 1.06-1.29).

Conclusions: Recent increases in outdoor air pollutants such as PM₁₀ or O₃ can trigger suicide, particularly during warm periods, even at concentrations below the European thresholds. Furthermore, PM₁₀ may have strong trigger effects among children and elderly population.

Key words: air pollution; suicide; case-crossover; epidemiology.

Introduction

According to the World Health Organization, 804,000 suicide deaths occurred worldwide in 2012, representing an annual global age-standardized suicide rate of 11.4 per 100,000 population [1]. In 2012, Eurostat estimated for Belgium an age-standardized suicide rate of 18.8 per 100,000 inhabitants, the highest suicide rate in Western Europe [2]. Physical and mental disorders as well as socio-demographic and community factors are well established factors for suicide mortality [1], however, environmental factors may also have an impact on suicide. Elements of diesel exhaust particles can accumulate in the brain [3,4] and particulate matter and ozone inhalation rapidly increase plasma levels of the hypothalamic –pituitary-adrenal axis stress hormones [5]. Results from animal studies show alterations in the serotonin system after exposure to traffic related pollutants and O₃, which may lead to aggressive and depressive behaviours [6–8]. So far, studies in humans investigating the relationship between long-term exposure to air pollution and depression were not conclusive [9–11]. However, short-term exposure was associated with emergency department visits for depression [12] and depression symptoms [11]. To date, only a few recent studies have considered outdoor air pollution as a potential trigger for suicide attempts [13] or suicide mortality [14–18]. These studies performed in Asia and North America have reported consistent positive associations between short-term exposure to outdoor air pollution and suicide attempts and mortality.

In Belgium, the levels of ambient air pollutants in the last decade have been below or close to the European standards and, as for suicide rates, among the highest in Europe [19]. Here, we present a case-crossover study that evaluates the association between recent changes in exposure to PM₁₀ and O₃ and suicide mortality registered in Belgium between 2002 and 2011, considering potential effect modifications by season, age, sex and mode of suicide.

Methods

Suicide mortality

The studied at risk population consists of all persons included in the Belgian 2001 census (n=10,295,724). Mortality follow-up for the period between January 1st 2002 and December 31st 2011 is based on the National Population Register linked to the registers of cause-specific mortality with an exact match for 90% of the records and probability linkage for the remaining records [20]. Data management and linkage were executed according to the Belgian privacy legislation and have been approved by the Belgian Commission for the Protection of Privacy. The database contains information on the cause, date and municipality of death, age at death, and sex. Suicide events were defined as mortality causes with ICD-10 codes X60 to X84 (intentional self-harm) or Y10 to Y34 (event of undetermined intent, i.e. uncertainty as to the intention of death). During the study period, 21,231 suicide events were registered among the population aged 5 years or more.

Outdoor air pollutants

In Belgium, the air concentrations of various pollutants are continuously measured by a dense network of monitoring sites (www.irceline.be). In a spatial-temporal (kriging) interpolation model, data collected by the monitoring stations are combined with land cover data from satellite images to account for the local variations of pollutant concentrations at locations where no monitoring stations are available [21]. This provides estimates for the measured pollutants on a 4x4 km grid, which are then used to calculate population-weighted averages per municipality. In the present study, we included daily average PM₁₀ concentrations ($\mu\text{g}/\text{m}^3$) and daily maximum 8-hour average O₃ concentrations for the study period (2002 – 2011). The pollutant concentrations were linked to the mortality data using the date and municipality of death.

Potential confounders and effect modifiers

Because meteorological variables may confound the association between air pollution and suicide mortality [22,23], we obtained daily data on average temperature and duration of sunshine from the Belgian Royal Meteorological Institute (Uccle measurement station, Belgium). We also considered day of the week as a potential confounder [24] and season as effect modifier. Previous studies showed

seasonal patterns in the effects of air pollution on mortality [25], in Belgium being stronger during summer[26]. Seasons were defined as 4 groups of 3 full months (winter: December to February, spring: March to May, summer: June to August, and autumn: September to November). Demographic variables such as sex and age may modify the association between air pollution and suicide [14,27]. In particular, there is consistent evidence that exposure to traffic-related pollutants has negative effects among the young and the elderly [27]. Age at death was categorized into 5 categories: childhood (5 to 14 y), adolescence (15 to 19 y), adulthood (20 to 64 y), young elderly (65 to 84 y), and elderly (≥ 85 y). Finally, the method to commit suicide may determine the time lag between the suicide act and the moment of death. Therefore, we distinguished between non-violent (e.g. self-poisoning) and violent (related to physical impacts or strangulation) mode of death (X60 to X69 and Y10 to Y19 for non-violent and X70 to X84 and Y20 to Y34 for violent).

Statistical analyses

We used a bidirectional time-stratified case-crossover design [28]. This design combines features of the crossover design and the matched case-control design where each subject serves as his/her own control and the inference is based on a comparison of exposure distribution rather than the risk of disease. Thus, known and unknown time-invariant confounders are inherently adjusted for.

To create the database, event days (i.e. the day of death) were matched with control days based on three criteria. First, control days were taken from the same month and year as the event days, both before and after the event, thus controlling for possible seasonality and long-term trends by design [28]. Second, control days and event days had to be at least 3 days apart from each other to avoid short-term autocorrelation [29]. Third, since suicide mortality and air pollution are both associated with temperature [23,26], only control days with a daily average temperature within 2°C from that on the event day were selected. The number of control days per event ranged from zero to 23, with an average of 9. The 675 events (3.2%) without control days were not included.

To evaluate the associations between daily concentrations of air pollutants and suicide mortality we used conditional logistic regression models adjusted for duration of sunshine and day of the week. We used separate models for each of the air pollutants and for different lag periods: single-day lag for the day of the suicide event (lag 0) and average exposures up to one week before the event (lag 0-1, lag 0-2, lag 0-3, lag 0-4, lag 0-5, lag 0-6). We estimated the odds ratios (OR) and their 95% CI for an increase in pollutant concentrations of 10 $\mu\text{g}/\text{m}^3$. Furthermore, to examine effect modification we included the interaction terms for each pollutant and modifier (season, age category, sex, and mode of death) in separate models. To check potential misclassification of suicide events within the “event of undetermined intent” category (codes Y10 to Y34) we performed sensitivity analyses excluding these codes. Statistical analyses were performed with SAS software (version 9.4; SAS Institute Inc., Cary, NC, USA).

Results

From January 1st 2002 to December 31st 2011, a total of 21,231 suicides were registered among the population aged 5 years or more. Overall, the number of suicides registered per year ranged from 2,006 to 2,275, with a slightly higher number of suicides registered during spring and summer seasons (52%). After excluding 675 cases without control days and 23 cases without meteorological information, our study included 20,533 suicide events (71 events registered among children (5 to 14 y), 482 among adolescents (15 to 19 y), 15,278 among adults (20 to 64 y), 4,081 among young elderly population (65 to 84 y) and 621 among elderly population (≥ 85 years old). Altogether, 72% (n=14,708) of suicides were committed by males and 86% (n=17,671) were classified as violent. Table 1 presents the distribution of the daily number of suicide deaths and of air pollutant concentrations and duration of sunshine on event days, and the absolute differences in air pollutant concentrations and duration of sunshine between event and control days. The absolute difference demonstrates the existence of sufficient variation around a non-zero mean value. The daily average concentrations of PM₁₀ were

strongly negatively correlated with O₃ concentrations during winter (Spearman correlation coefficient (r) = -0.67), but positively correlated during summer (r=0.46).

Figure 1 shows the adjusted ORs for 10 µg/m³ increase in PM₁₀ or O₃ during the whole study period and by season, considering average exposures up-to one week before the suicide event. Suicide mortality was not significantly associated with ambient PM₁₀ concentrations when considering the whole study period. However, during summer a 6 to 7% statistically significant increase in the odds of suicide was observed for 10 µg/m³ increases of PM₁₀, for average concentrations in lags 0-3, 0-4 and 0-5 (interaction p-value<0.1 for lags 0-4). Instead, ambient O₃ concentrations were statistically significantly associated with suicide mortality throughout the whole study period, with positive associations in all seasons (interaction p-values<0.05 for lags 0-2 to 0-6) except winter, for which significant inverse associations with O₃ were observed for lags 0-5 and 0-6.

Figure 2 shows the associations of suicide mortality with PM₁₀ and O₃ by age group. Each 10 µg/m³ increment in PM₁₀ was significantly associated with a 45% increase in the odds of suicide among children (95% CI 1.03-2.04; interaction p-values<0.05 for lags 0-4 to 0-6), and with a 7 to 12% increase in suicide among the elderly (≥85 years old). For O₃ exposure, a statistically significant 1 to 2% increase in the odds of suicide mortality was observed for all the studied lags among the adult population. Among adolescents, a 10 µg/m³ increase in lag0-1 O₃ was associated with an 8% increased odds of suicide (95% CI: 1%-16%). However, the interaction terms between O₃ and age group were not statistically significant.

Associations by sex and mode of death did not show statistically significant differences between groups (interaction p-values≥0.05). For mode of death, the ORs were only statistically significant for the associations with O₃ among individuals who committed suicide using violent methods (Figure 3). Finally, sensitivity analyses excluding deaths categorized as “undetermined intent” (n of events=1857) did not modify the results (data not shown).

Discussion

We show that ambient levels of PM₁₀ and O₃ may trigger suicide mortality in Belgium, a country with the highest air pollution levels and suicide rate in Western Europe.[2] In particular, we observed triggering effects of both pollutants during summer and, for O₃, also during spring and autumn. Age significantly modified the trigger effect of PM₁₀ on suicide mortality regardless of the season, with significant trigger effects among extreme age groups (children and elderly). We do not claim that air pollution causes people to commit suicide, however, our findings suggest that when people are to commit suicide, they are more likely to do so when air pollution is high.

Experimental and epidemiological studies provide plausible potential biological mechanisms to explain the impact of traffic-related air pollution on the brain [27]. In animal models, exposure to diesel exhaust particles during pregnancy increases testosterone and dopamine levels, and decreases serotonin in mice, which may lead to aggressive and depressive behaviours [6]. Furthermore, exposure to O₃ can have an adverse effect on the serotonin system [7], and may precipitate, among others, adverse neurobehavioural outcomes [8]. In humans, in utero exposure to high concentrations of PM_{2.5} is associated with low transcription of Brain Neurotrophic Derived Factor, which plays a role in the development of the nervous system [30]. Recent studies using neuroimaging techniques suggest that air pollution is associated with lower functional integration and segregation in key brain networks, relevant to both inner mental processes and stimulus-driven mental operations [31]. Furthermore, results from recent epidemiological studies show that outdoor air pollution is associated with early neurovascular damage in children [32], dementia [33] and Parkinson disease [34].

So far, epidemiological studies focusing on depression have not shown conclusive results in relation with long-term exposure to air pollution [9–11]. However, recent high levels of air pollution may trigger emergency department visits for depression [12] and depression symptoms [11]. To date, few studies have investigated the effects of air pollution on suicide [13–18], and only four of them investigated the trigger effect of air pollution on suicide mortality [14,16–18]. Kim et al[16] included suicide mortality events that occurred during 2004 in seven South Korean cities. A recently published

study included suicide deaths that occurred in Tokyo between 2001 and 2011 [17]. In the USA, Bakian et al [14] investigated suicide events in Salt Lake City between 2000 and 2010. Finally, a smaller study included 1550 suicide cases that occurred in the Chinese region of Guangzhou [18]. The four studies showed increased risk of suicide mortality after up-to 3 days exposure to elevated levels of pollutants, however, except for the Chinese study, the observed effects varied across studies depending on demographic and seasonal factors. The Chinese study [18] reported significant effects among all the included population and seasons. Bakian et al [14] and Kim et al [16] reported stronger effects among adults aged 35 to 64, males, and during transition seasons (i.e. spring and fall). Consistently with our results, the study performed in Tokyo showed significant associations during summer and among younger ages. However, none of these studies tested effect modification by the mentioned factors, and they did not study extreme ages (i.e. childhood and elderly).

Our study shows significant associations between suicide mortality and PM₁₀ in Belgium, however, only during summer and at extreme ages. The differences in the trigger effects across season between our study and the studies by Bakian et al and Kim et al [14,16] may be explained by the meteorological differences between regions. Peng et al [25] compared air pollutant effects on mortality across different climatic regions and seasons in the USA and concluded that seasonal patterns vary across regions. In Belgium, stronger effects of PM₁₀ on mortality during summer have been described [26]. The seasonal patterns in the PM₁₀ effects suggest that this pollutant is not equally harmful under different meteorological conditions. First, the composition of PM changes across the year with the highest pro-inflammatory content being measured during summer [35]. Second, the pattern of personal exposure changes across seasons, the amount of time spent indoors in cold periods is higher than during warm periods leading to differential personal exposure to air pollution [26,25,36]. Third, the PM₁₀ effect may be swamped by more powerful triggers of suicide, such as recent stressful life events at school or work [37].

In addition to season, age may also modify the relationship between air pollution and suicide. Previous research reported quantifiable impairments of brain development in the young, and cognitive decline in the elderly in association with exposure to traffic-related pollutants [27]. In contrast to the two previous studies on trigger effects of air pollution on suicide [14,16], our study includes more than 20,000 registered suicide events thus allowing us to explore the associations in five age groups. Consistently with previous studies reporting effects on the central nervous system in children and elderly [27], we observed statistically significant trigger effects of PM₁₀ among children aged 5 to 14 years and among the elderly population (≥ 85 years old). Caution needs to be exercised when interpreting our results for children, since the suicide rates among children are low [1]. Our study included only 71 events of child suicide mortality. Nevertheless, despite the relatively low number of events – and, hence, the wide confidence intervals – we observed a statistically significant association with PM₁₀.

Other than PM₁₀, O₃ has also been hypothesized as potential trigger of suicide mortality. In line with our results, an ecological study recently reported that cumulative exposure to O₃ during 5 weeks was associated with suicide rates in South Korea [15]. In our study, we evaluated the acute (up to one week) associations between O₃ concentrations and suicide mortality. As for PM₁₀, O₃ trigger effects also exhibited a seasonal pattern with trigger effects observed during all seasons except winter, as reported for other mortality causes [38]. In winter, we observed statistically significant inverse associations between O₃ and suicide which may be explained by the well-known strong negative correlation between this pollutant and PM₁₀ during winter, as well as with other common pollutants such as PM_{2.5} or NO₂ not included in this study [39]. For O₃, we did not observe any statistically significant effect modification by age, although the effect estimates appeared generally stronger for younger and older populations.

The method employed to commit suicide (violent vs non-violent) has been associated with sex and the concentrations of inflammatory biomarkers [40]. Moreover, the mode of suicide may determine the

time lag between the suicide act and the moment of death. Suicides committed using non-violent methods such as self-poisoning may result in delayed deaths as compared with violent methods, such as jumping from a high place. We assessed the potential effect modification by sex and mode of suicide for both investigated pollutants. Our results were in line with those reported in previous research [14,16], we observed no significant differences between violent and non-violent methods of suicide, and significant trigger effects were only evident among men and violent methods, in the case of O₃ exposure.

A major strength of our study is its population wide character. Our study is among the largest studies performed to date focusing on recent trigger effects of environmental pollution on suicide and the first performed in Europe. It includes more than 20,000 completed suicides registered in a whole country during a period of one decade. In addition, it is the first study analysing effect modification by age groups including childhood and old age. Nevertheless, some limitations have to be considered when interpreting our results: first, our study is based on registry data which is limited in terms of specificity of the information available and accuracy; second, we used interpolated information on ambient air pollutants from monitoring stations instead of using actual personal exposure data, and third the high correlation between pollutants does not allow to disentangle the difference in the effects between pollutants.

Issues to be considered when working with registry based data are the availability of information on relevant confounders of the studied associations. Nonetheless, the characteristics of the case-crossover design limits the potential confounders to variables that are time varying within a month. Therefore, only acute events that are also related to the exposure, such as the loss of a family member, may have confounded our results, in particular when considering the loss of the partner among the elderly. Although the mortality dataset used in this study contains information on cause-specific mortality for all the registered Belgian population, but this data cannot be linked with ours due to privacy legislation. In addition, the accuracy of suicide mortality codification is not optimal [41].

It is based on death certificates and may be influenced by the cultural or religious beliefs of the practitioner who fills in the death certificate or to lack of recognition of the suicide event due to its characteristics. Yet, potential errors in the suicide code are unlikely to be linked with the exposure to air pollution, so they would not confound our results.

Regarding exposure assessment, the use of air pollution measurements from local monitoring stations lacks precision regarding the actual personal exposure. We assumed that the relevant exposure would be the outdoor exposure at the municipality of death, although changes in exposure may occur during commuting to work or school. However, studies comparing personal and ambient exposure have reported good correlations [42], and spatial variability in Belgium is small when compared with the temporal variability studied here, which is driven by meteorological fluctuations [43]. Yet, our study design allowed to rule out confounding by temperature as case days were matched to control days with similar temperature. Finally, the well-known strong correlation that exists between specific pollutants [39] makes it difficult to disentangle the effects of separate pollutants, as already discussed for the inverse associations observed in the present study for O₃. Nevertheless, models including both pollutants did not show differences in the effect estimates, suggesting potential independent effects.

In conclusion, high concentrations of common outdoor air pollutants such as PM₁₀ or O₃ can trigger suicide, particularly during warm seasons. The novelty of our study is that we were able to evaluate these associations among young and old age groups (childhood and elderly) in an affluent society, these age segments are considered most vulnerable to the effects of air pollution on the central nervous system [27]. We observed especially strong trigger effects of PM₁₀ for children aged 5 to 14 years and elderly population aged 85 years or more during the warmer period of the year.

Ethical approval: for this type of study formal consent is not required.

Conflict of Interest: The authors declare that they have no conflict of interest.

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Tables

Table 1. Daily numbers of suicide events, air pollution levels and duration of sunshine on event days, and absolute differences between the daily average levels of each pollutant and duration of sunshine on event days and the average exposure on control days, Belgium 2002-2011.

	Mean	SD	Min	p25	p50	p75	Max
Daily number of suicide events	5.8	2.5	0.0	4.0	6.0	7.0	19.0
Exposure on event days							
PM ₁₀ (µg/m ³)	29.0	15.2	1.0	18.6	25.4	35.7	140.2
O ₃ (µg/m ³)	61.8	30.3	1.0	42.2	59.9	77.9	221.2
Sunshine (minutes)	236.6	243.2	0.0	23.0	163.0	389.0	942.0
Exposure difference between event days and average of control days							
PM ₁₀ (µg/m ³)	9.9	9.9	0.0	3.2	7.0	13.3	116.9
O ₃ (µg/m ³)	14.0	11.3	0.0	5.5	11.4	19.5	84.8
Sunshine (minutes)	161.0	135.3	0.0	55.5	123.1	238.3	703.0

SD: standard deviation; Min: minimum; p25: 25 percentile; p50: 50 percentile or median; p75: 75

percentile; Max: maximum.

Figures

Fig 1. Odds ratios (OR) and 95% confidence interval for suicide mortality (n events=20,533) associated with a 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} (a) and O_3 (b) concentrations for different exposure windows, over the whole study period and by season. OR were adjusted for day of the week and average duration of sunshine at the same lags as for the air pollutants.

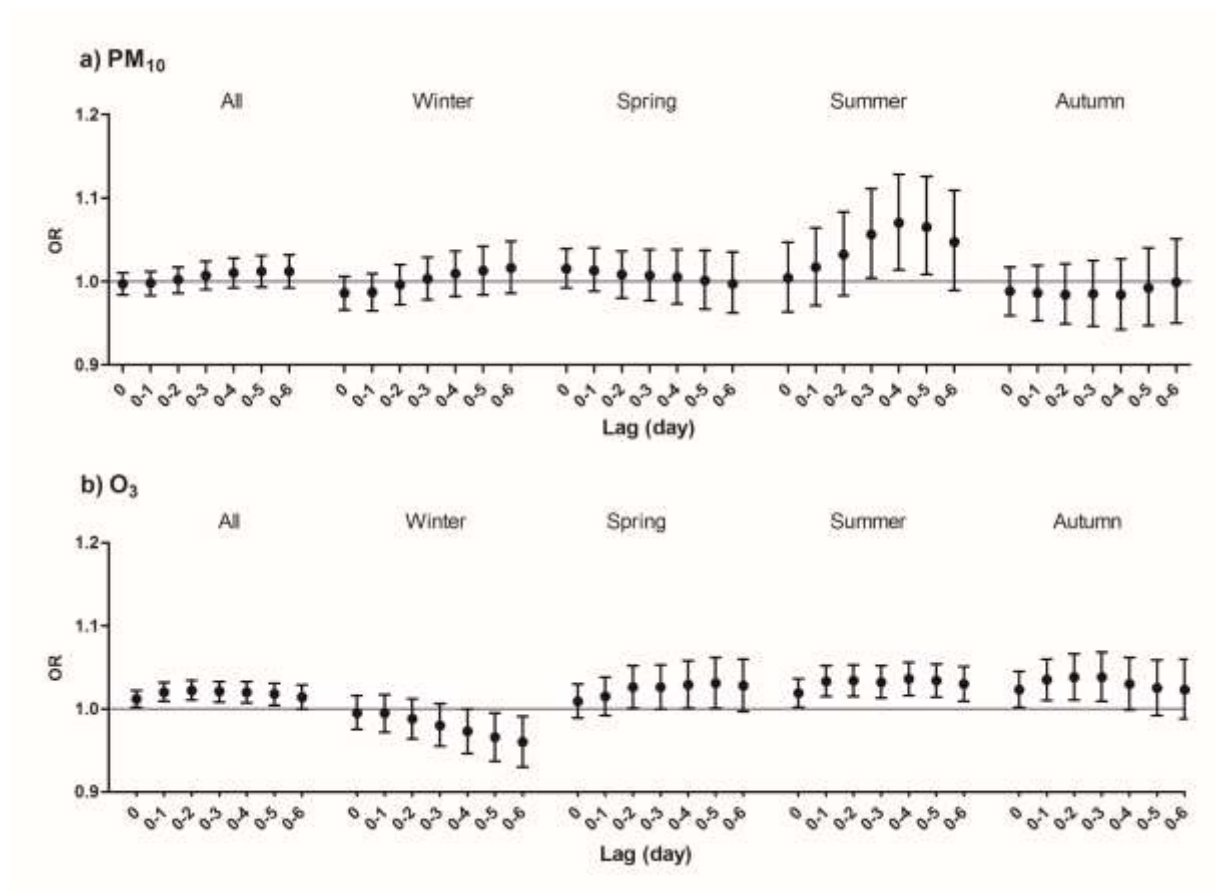


Fig 2. Odds ratios (OR) and 95% confidence interval for suicide mortality (n events=20,533) associated with a 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} (a) and O_3 (b) concentrations for different exposure windows, by age group. OR were adjusted for day of the week and average duration of sunshine at the lags considered for the air pollutants.

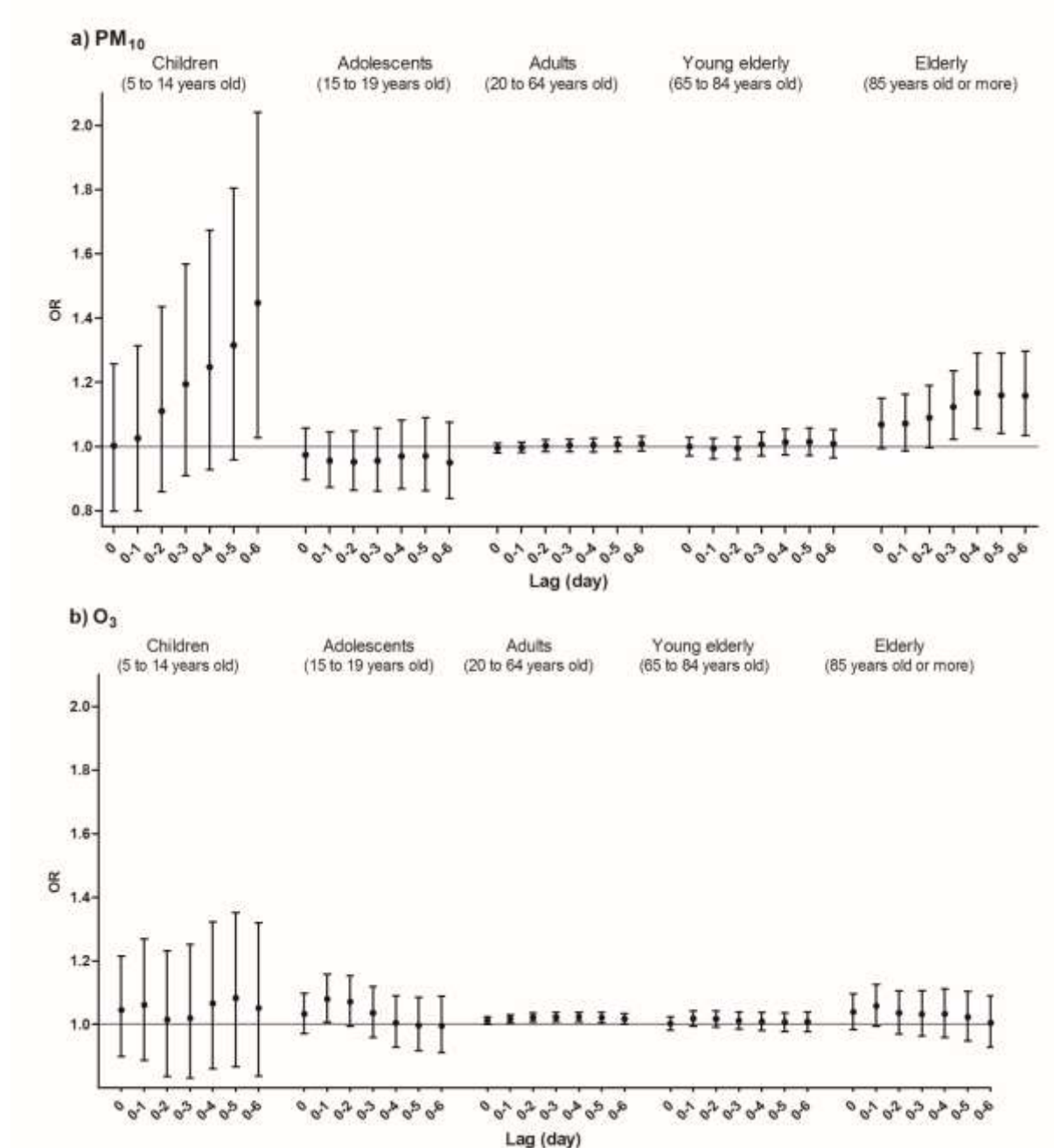


Fig 3. Odds ratios (OR) and 95% confidence interval for suicide mortality (n events=20,533) associated with a 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} and O_3 concentrations for different exposure windows, by sex (a) and cause of death (violent and non-violent) (b). OR were adjusted for day of the week and average duration of sunshine at the lags considered for the air pollutants.

