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Research article

Ambient air pollution exposure and risk of depression: A systematic review and meta-analysis of observational studies

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ABSTRACT

Recent studies have reported an association between air pollution exposure and depression, with inconsistent results. To address this controversy, we conducted a systematic review and meta-analysis of published observational studies that investigated outdoor air pollution and depression. Five electronic databases were searched, and fifteen articles were finally identified. Pooled odds risks were calculated separately based on pollutant type, exposure duration and outcome. Subgroup analyses were conducted based on design, population, important potential confounders, and pollutants levels. We found a significantly increased risk of depression with long-term exposure to $PM_{2.5}$ and short-term exposure to PM_{10}, NO_2, SO_2, CO. No evidence was found in the association between exposure to O_3 and depression. Besides, exposure to high levels of pollutants indicates a higher risk of depression. Our results highlight the necessity of air pollution control for depression. However, further studies with standardized methods are still required to support the results due to the inconsistent results in stratified analyses and methodological limitations of the included studies.

1. Introduction

Depression is one of the most common mental health problems worldwide. Currently, more than 300 million people are living with depression, with an increase of 18% from 2005 to 2015 (World Health Organization, 2017). The prevalence of depressive disorders was estimated at 540.5 per 100,000, and this disease has become the third leading burden among non-fatal diseases globally (Kyu et al., 2018). Depression leads to various health-related damages such as decline in quality of life, social function impairment and increase of cancer mortality (Pinquart and Duberstein, 2010; Wariso et al., 2017). Furthermore, depression is a strong risk factor for suicide, and roughly 59% of those who commit suicide have a history of depression (Cavanagh et al., 2003, #10). It has been predicted that depression would become the second leading cause of disease by 2030 (Mathers and Loncar, 2006). As a significant health challenge throughout the world, and the management of risk factors is crucial to the prevention of depression.

Air pollution is a severe environmental issue which seriously threatens human health. Air pollution, mainly particulate pollutants ($PM_{2.5}$, particles with an aerodynamic diameter $\leq 2.5\,\mu m$; PM_{10}, particles with an aerodynamic diameter $\leq 10\,\mu m$) and gas pollutants (NO_2, nitrogen dioxide; SO_2, sulfur dioxide; O_3, Ozone; CO, carbon monoxide), leads to annually more than six million deaths and estimated loss of 5000 billion dollar related to health and well-being (World Health Organization, 2018b). Ambient air pollution is a major threat to health in both developed and developing countries. 91% of the population is living in areas where air pollution levels exceed WHO limits (proposed annual mean: $PM_{2.5} < 10\,\mu g/m^3$; $PM_{10} < 20\,\mu g/m^3$; $NO_2 < 40\,\mu g/m^3$), especially in South-East Asia and Western Pacific area (World Health Organization, 2018a). Mounting numbers of epidemiological studies have confirmed that depression would lead to severe health issues such as cardiovascular disease, asthma, diabetes and stroke (Eze et al., 2015; Guarnieri and Balmes, 2014; Kim et al., 2017). Studies also showed that air pollution exposure might be a risk factor for depression (Kim et al., 2016; Lim et al., 2012; Shin et al., 2018; Szyszkowicz et al., 2009).

Air pollution affects the central nervous system and brain function (Block and Calderon-Garciduenas, 2009). Air pollution caused systemic inflammation, neuroinflammation and oxidative stress of the central nervous system, and damage to the blood–brain barrier in animal studies (Block and Calderon-Garciduenas, 2009). All of these pathological characteristics had been reported in depression and were considered as possible biological mechanisms for depression (Benson et al., 2017; Jeon and Kim, 2016; Palta et al., 2014). Although the potential

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mechanism of the association between depression and air pollution have been proposed, the results of epidemiological studies remain controversial. Some studies reported that long-term exposure to $PM_{2.5}$ would significantly increase the risk of depressive disorder (Kim et al., 2016) and depressive symptoms (Lin et al., 2017; Shin et al., 2018). Similarly, short-term exposure to $PM_{2.5}$ would increase the emergency department visits for depression (Wang et al., 2018) and the risk of the depressive symptom (Pun et al., 2017). However, no evidence of the association between long-term exposure to $PM_{2.5}$ and depressive symptoms was observed in four European cohort studies {Zijlema et al., 2016, #42}. And a non-significant association was also found for short-term exposure to $PM_{2.5}$ in Canada (Zijlema et al., 2016). Short- and long-term exposure to PM_{10}, NO_2, SO_2, CO, O_3 can significantly increase the risk of depression (Cho et al., 2014; Shin et al., 2018). However, no association was found between these pollutants and depression in the USA and Canada (Szyszkowicz, 2007; Wang et al., 2014).

Several reviews have summarized investigations of air pollution and depression (Buoli et al., 2018; Gladka et al., 2018; Łopuszańska and Makara-Studzińska, 2017). However, the effect size remains inconclusive with the increased attention. Therefore, this comprehensive systematic review and meta-analysis of observational studies aims to investigate the association of depression with typical ambient air pollutant exposure and resolved divergences. Different exposure duration and different outcomes (depressive disorders and depressive symptoms) were also stratified analyzed.

2. Methods

2.1. Search strategy

Three authors (Y.Z., L.L., Y.L.) independently searched original studies in PubMed, Embase, Web of Science, the Cochrane Library and Clinical Key from establishment to September 20, 2018. Both quick search and Mesh term search were used. The terms were as follows: ("air pollution" OR "air pollutant*" OR "particulate matter" OR "nitrogen dioxide" OR "sulfur dioxide" OR "carbon monoxide" OR "ozone") AND ("depress*" OR "depressive disorder*" OR "mood disorder*" OR "affective disorder*" OR "mental health"). We also scanned the reference list of four related reviews (Buoli et al., 2018; Gladka et al., 2018; Łopuszańska and Makara-Studzińska, 2017; Zhao et al., 2018) and included studies.

2.2. Selection criteria and process

We only included articles published in English and met the following criteria: (1) the research participants were human; (2) original research; (3) exploring at least one of the most common outdoor air pollutants: $PM_{2.5}$, PM_{10}, NO_2, SO_2, O_3, CO; (4) observational study, including cohort study, case-crossover study, time-series study and cross-section study; (5) including a depression group; and (6) the studies providing numeric and figured effect sizes and their 95% confidence intervals (CIs), which included the odds ratio (OR), relative risk (RR), hazard ratio (HR) or the increased percentage of RRs, ORs, HRs. We excluded the study if it met: (1) the source of pollution was not from the outdoor atmosphere, such as household air pollution; (2) there was no clearly defined depression group, such as mental disorder, bipolar disorder; (3) conference papers or reviews.

Y.Z. and L.L. screened abstracts, then Y.Z. and R.L. strictly reviewed full-text based on the selection criteria. We excluded one study (marked in Table 1 by "a") for meta-analysis (Szyszkowicz, 2007) as the study sample was a part of another included study (Szyszkowicz et al., 2009). Besides, one study (marked in Table 1 by "b") reported effect sizes of $PM_{2.5}$, PM_{10}, NO_2, SO_2 during the warm season but O_3 during the whole years, so we only included the result of O_3 to avoid potential bias as we failed to receive response from the authors (Szyszkowicz et al., 2016). Y.L. was responsible for divergences. The selection process is showed in the flow chart (Fig. 1).

2.3. Data extraction and quality assessment

The following characteristics were independently collected for each eligible study by two authors (R.L. and L.L.): first author's name, published year, study design, study period and location, population characteristics (sample size, gender, age), outcome and definition, pollution characteristics (type, exposure durations, annual mean levels, pollutant assessment method), effect sizes and 95% CIs, control variables, main findings. We chose the data controlled the most potential confounders when different controlled models were reported. We defined short-term exposure as less than 2 weeks and long-term exposure as more than 1 year. If different exposure durations were reported, we chose the longest exposure duration for long-term exposure studies for meta-analysis. For short-term exposure, 0–2 lag or the closest one was selected because it was most frequently reported. After two authors completed the extraction, the first author (Y.Z.) extracted independently and checked all the data.

Two authors (Y.Z. and Y.L.) independently assessed the quality of each included studies by Newcastle–Ottawa Scale (NOS), which included three domains (Sakhvidi et al., 2018). For comparability domain, we gave two stars when study controlled for more than eight confounders and no star for less than two confounders. A score of 7 or higher was regarded as a high quality, a score of 3 or lower was considered as poor quality. Y.L. was responsible for addressing differences.

2.4. Statistical analyses

Because of the relatively low prevalence of depression, the estimates of odds ratio (OR) are similar to relative risk (RR). Therefore, we chose ORs for reporting of all of the results of meta-analysis. The increased percentage of morbidity was calculated into the OR by increasing the percentage of morbidity/100 + 1 (Szyszkowicz et al., 2009; Wang et al., 2018). We standardized the ORs into an increment of 5 $\mu g/m^3$ for $PM_{2.5}$; 10 $\mu g/m^3$ for PM_{10}; 5 ppb for NO_2, SO_2, O_3; 0.1 ppm for CO to pooled estimates (Nguyen Thi Trang et al., 2017). We separately performed meta-analysis according to pollutant type, exposure duration, and outcome. For each analysis, pooled ORs and 95% CIs were calculated using fixed or random effect model, and chi-square test and I^2 statistic was used to assess the heterogeneity (DerSimonian and Laird, 1986; Higgins et al., 2003). The random-effect model was used if the heterogeneity was substantial; otherwise, we use the fixed-effect model for analysis. Sensitivity analyses were performed to examine the consistency of the results by excluding each study and redo the analysis. Besides, subgroup analyses were performed based on study design, population, adjustment for important potential confounders (socioeconomic status, smoking, alcohol consumption, chronic diseases), pollutants levels. We conducted all analyses by using Stata 12.0 (StataCorp., College Station, Texas, USA).

3. Results

3.1. Study characteristics

Six thousand four hundred and thirty-one records were identified through our initially search. After reviewing, 15 papers for systematic review and 14 papers for meta-analysis were included finally (Fig. 1). Of the 14 papers for meta-analysis, nine were cohort studies (one article included four independent cohort studies) (Kim et al., 2016; Kioumourtzoglou et al., 2017; Lim et al., 2012; Pun et al., 2017; Wang et al., 2014; Zijlema et al., 2016), three were case-crossover studies (Cho et al., 2014; Szyszkowicz, 2011; Szyszkowicz et al., 2016; Wang et al., 2018), one was a time-series study (Szyszkowicz et al., 2009), and three were cross-section studies (Lin et al., 2017; Shin et al., 2018; Vert

Table 1
The basic overview of the included articles of systematic review.

Study	Design	Location	Study period	Outcome No. of case	Age Range/ *mean*	Gender Male%	Exposure characteristics	Pollution assessment method	Controlled variables	Main findings	*Q*
Kioumourtzoglou et al. (2017)	Prospective cohort	11 states, USA	1996–2000	DD *N* = 2405	≥50/66.6	0%	Type: $PM_{2.5}$, O_3 Long-term exposure	$PM_{2.5}$: GAM	Season, region, race, BMI, health behavior (a, b, c, d), SES(a, b), mental health,	− − (1) $PM_{2.5}$, O_3;	S = 3C = 1 O = 2T = 6
				DS *N* = 4516				O_3: Regional air quality data			
Kim et al. (2016)	Retrospective cohort	Seoul, Korea	2002–2010	DD *N* = 973	15–79/NA	54%	Type: $PM_{2.5}$ Long-term exposure	Regional air quality data	Gender, age, health behavior (a, b, c), SES (c), married ratio, social and economic satisfaction, social welfare	+ + (1) $PM_{2.5}$; (2) Susceptible factor: chronic disease	S = 4C = 1 O = 3T = 8
Pun et al. (2017)	Prospective cohort	USA	2005–2006	DS Wave 1: *N* = 730	57–85/69	48.4%	Type: $PM_{2.5}$ Long- and short-term exposure	GAM	Age, gender, race, year, season, day of week, SES(a, b, f, g and percentage of population below poverty level)	+ + (1) 7- and 30-days exposure to $PM_{2.5}$; (2) Susceptible factor: low SES level, chronic disease	S = 2C = 1 O = 3 T = 6
			2010–2011	Wave 2: *N* = 703	/72.4					− − (3) 180-days, 1- and 4-years exposure to $PM_{2.5}$.	
Zijlema et al. (2016) *Lifeline*	Retrospective cohort	The Netherland	2007–2013	DS *N* = 681	All age/ 43.8	43.1%	Type: $PM_{2.5}$, PM_{10}, NO_2 Long-term exposure	LUR	Age, gender, urbanity, SES(a, c), chronic disease (c, f, g), road traffic noise	− − (1) $PM_{2.5}$ + + (2) PM_{10}, NO_2	S = 3 C = 1 O = 2 T = 6
Zijlema et al. (2016) *KORA*	Retrospective cohort	Germany	2004–2008	DS *N* = 87	All age /55.3	48.5%	Type: $PM_{2.5}$, PM_{10}, NO_2 Long-term exposure	LUR	Age, gender, urbanity, SES(a, c), chronic disease (c, f, g), road traffic noise	− − $PM_{2.5}$, PM_{10} and NO_2	S = 3 C = 1 O = 2 T = 6
Zijlema et al. (2016) *HUNT*	Retrospective cohort	Norway	2006–2008	DS *N* = 1226	All age/ 54.7	44.3%	Type: PM_{10}, NO_2 Long-term exposure	LUR	Age, gender, chronic disease (c, f, g) road traffic noise	− − NO_2 and PM_{10}	S = 3 C = 1 O = 2 T = 6
Zijlema et al. (2016) *FINRISK*	Retrospective cohort	Finland	2007	DS *N* = 155	All age/ 51.9	43.6%	Type: $PM_{2.5}$, PM_{10}, NO_2 Long-term exposure	LUR	Age, gender, urbanity, SES(a, c), chronic disease (c, f, g), road traffic noise	− − $PM_{2.5}$, PM_{10} and NO_2	S = 3 C = 1O = 2 T = 6
Wang et al. (2014)	Prospective cohort	Boston, USA	2005–2008	DS *N* = 62	≥65/78.1	35.8%	Type: $PM_{2.5}$, NO_2, O_3, CO Short-term exposure	Regional air quality data	Age, gender, race, visit, T, barometric pressure, day of week, season, long-term temporal trends	− − $PM_{2.5}$, NO_2, O_3, CO	*S* = 3 *C* = 1 *O* = 2 T = 6
Lim et al. (2012)	Prospective cohort	Seoul, Korea	2008	DS	≥60/71	26%	Type: PM_{10}, NO_2, O_3, CO Short-term exposure	Regional air quality data	Age, gender, T, BMI, urine cotinine level, follow-up time, health behavior (b, c)	+ + PM_{10}, NO_2, and O_3	S = 3 C = 1 O = 2 T = 6
			2009 –2010	NA							
Wang et al. (2018)	Case-crossover	26 cities, China	2014–2015	DD *N* = 19,646	All age/46	33.1%	Type: $PM_{2.5}$, PM_{10} Short-term exposure	Regional air quality data	Sex, gender, T, humidity, day of week,	+ + (1) $PM_{2.5}$ and PM_{10} (2) Susceptible factor: the elderly	S = 4 C = 1

(*continued on next page*)

Table 1 (*continued*)

Study	Design	Location	Study period	Outcome No. of case	Age Range/ *mean*	Gender Male%	Exposure characteristics	Pollution assessment method	Controlled variables	Main findings	Q
									season, long-term trend		O = 2 T = 7
Szyszkowicz et al. (2016)[b]	Case-crossover	9 cities, Canada	2004–2011	DD *N* = 118,602	All age /NA	42.0%	Type: $PM_{2.5}$, NO_2, SO_2, O_3 Short-term exposure	Regional air quality data	T, relative humidity	++ (1) O_3; (2) stronger association in the warm season −− (3) $PM_{2.5}$, SO_2 and NO_2	S = 4 C = 1 O = 3 T = 7
Cho et al. (2014)	Case-crossover	Seoul, Korea	2005–2009	DD *N* = 4985	All age/44	26.7%	Type: PM_{10}, NO_2, SO_2, O_3, CO Short-term exposure	Regional air quality data	National holidays, T, sunlight hours, humidity, air pressure	++ (1) PM_{10}, NO_2, SO_2, CO	S = 4 C = 1 O = 3 T = 7
Szyszkowicz (2011)	Case-crossover	Toronto, Canada	1999–2002	DD *N* = 680	0–85/NA	0%	Type: O_3 Short-term exposure	Regional air quality data	Day of week, T, humidity	++ (1) O_3 in women; (2) Susceptible factor: the elderly	S = 4 C = 1 O = 2 T = 7
Szyszkowicz et al. (2009)	Time-series	8 cities, Canada	1992–2003	DD *N* = 27,047	All age/NA	NA	Type: PM_{10}, NO_2, SO_2, O_3, CO Short-term exposure	NA	T, humidity	++ (1) PM_{10}, NO_2, SO_2, CO; (2) Stronger association in the warm season	S = 4 C = 1 O = 2 T = 7
Szyszkowicz et al. (2007)[a]	Time-series	Edmonton, Canada	1992–2002	DD *N* = 15,556	All age /NA	40.6%	Type: PM_{10}, NO_2, SO_2, O_3, CO Short-term exposure	Regional air quality data	T, humidity	−− (3) $PM_{2.5}$ and O_3 ++ The significant association was only as follow: CO for all patients in warm season, NO_2, SO_2, O_3 for females in warm season, PM_{10}, $PM_{2.5}$ for females in cold season	S = 4 C = 1 O = 2 T = 7
Shin et al. (2018)	Cross-section	Korea	2013	DD *N* = 3105	≥19/48	49.9%	Type: PM_{10}, NO_2, SO_2, CO Long-term exposure	Regional air quality data	Age, sex, sleep, SES(a, b, c, d, e), health behavior (a, b, c), chronic disease (a, b, d, e, f, g, h)	++ (1) PM_{10}, NO_2, CO; (2) Susceptible factor: male	S = 2 C = 1O = 2 T = 5
Vert et al. (2017)	Cross-section	Spain	2013–2014	DS *N* = 9936 DD *N* = 13	44–74/56.5	36.1%	Type: $PM_{2.5}$, PM_{10}, NO_2 Long-term exposure	LUR	Age, sex, BMI, sleep, social support, SES(a, h), health behavior (a, c)	++ $PM_{2.5}$, PM_{10}, NO_2	S = 2 C = 1 O = 2 T = 5
Lin et al. (2017)	Cohort based cross-section	China, Ghana, India, Mexico, Russia, South Africa	2007–2010	DS *N* = 53 DS *N* = 3189	≥18/59.8	44.9%	Type: $PM_{2.5}$ Long-term exposure	Geos-Chem chemical transport model	Age, gender, BMI, health behavior (a, b), SES (a, b, c, d), domestic fuel type, ventilation	++ (1) $PM_{2.5}$; (2) Susceptible factor: smoking	S = 2 C = 1 O = 2 T = 5

NA, not available; DD, depressive disorders; DS, depressive symptom; ++, positive and significant association between pollutants and outcomes; −−, no association between pollutants and outcomes. *Pollution assessment method*: GAM, generalized additive model; LUR, land use regression model. *Controlled variables*: T, temperature; BMI, body mass index; SES, socioeconomic status (a, education, b, marital status, c, household income, d, residence, e, employment status, f, religion, g, residence, h, living alone); health behavior (a, smoking, b, alcohol consumption, c, exercise, d, dietary habits); chronic disease (a, dyslipidemia; b, diabetes mellitus; c, chronic obstructive pulmonary disease; d, hypertension; e, stroke; f, myocardial infarction, g, asthma, h, dyslipidemia). *Q*, quality evaluation. The Newcastle Ottawa quality assessment score was used. S, selection; C, comparability; E, exposure; T, total.

[a] Not included in the meta-analysis.

[b] Only O_3 were included in the meta-analysis.

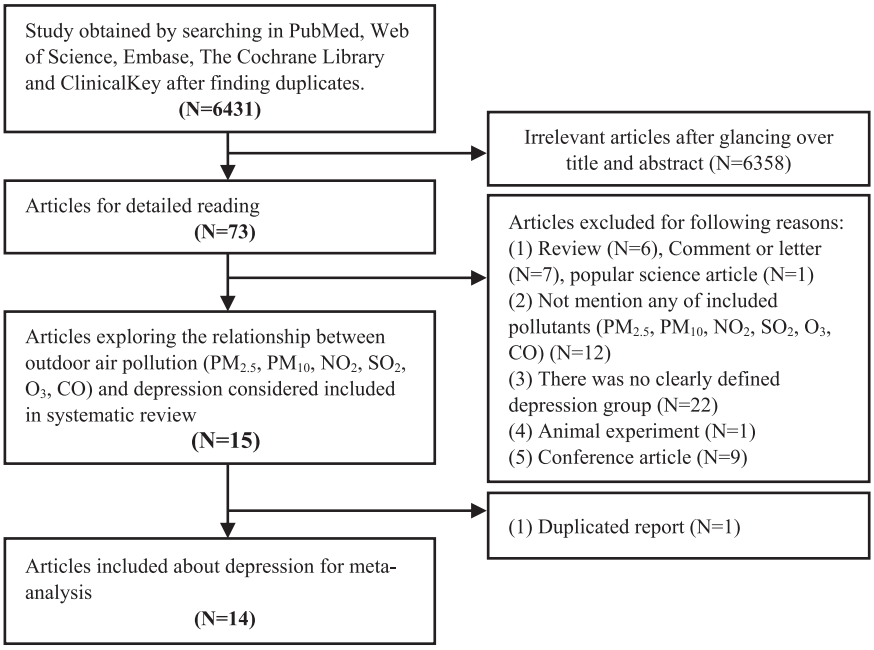

Fig. 1. Flow diagram of the study selection process.

et al., 2017). In sum, we identified 193,012 cases for depressive disorders and 21,338 cases for depressive symptom from 14 countries. Table 1 and Supplementary Table 1 summarized basic characteristics of the included studies for systematic review. For sampling, 11 studies were population-based; one study sampled female married registered nurses with age over 50 years (Kioumourtzoglou et al., 2017); three studies sampled community-dwelling old individuals (Lim et al., 2012; Pun et al., 2017; Wang et al., 2014); one study only sampled female population (Szyszkowicz, 2011) (Table S1). The duration of long-term exposure ranged from 1 year to 5 years (n = 10), and the duration of short-term exposure ranged from 0–2 lag to 0–14 lag (n = 7). Depressive disorders were identified according to electronic medical record in seven studies and self-report of doctor diagnoses in two studies. The majority of studies assessed the depressive symptom by widely used scales (n = 8), but two studies used self-report of antidepressants use (Table S1). Multiple methods were used to assess the pollution exposure, mainly using regional air quality data nearby (n = 9), land-use regression model (n = 6) and generalized additive model (n = 2) (Table S1). The results of the quality assessment were summarized in Table 1, and the details of rating were available in Table S2. The total quality score ranged from 5 to 8. Seven studies are rated as high quality (NOS ≥ 7).

3.2. $PM_{2.5}$ exposure and the risk of depression

In total, six cohort studies and two cross-sectional studies were finally included for meta-analysis for long-term exposure to $PM_{2.5}$, and 2 cohort studies and 2 case-crossover studies were included for short-term exposure. Statistical significant association between long-term exposure to $PM_{2.5}$ (OR = 1.06, 95% CI: 1.00, 1.13 per 5 μg/m^3 increase) and depression was found, with moderate heterogeneity (I^2 = 41.4%, p = 0.082). The summarized risk estimate was 1.01 (95% CI: 0.99, 1.03) per 5 μg/m^3 increase for short-term exposure, and the heterogeneity (I^2 = 69.6%, p = 0.020) was significant (Fig. 2(a)).

3.3. PM_{10} exposure and the risk of depression

Four cohort studies and two cross-sectional studies were included for meta-analysis for long-term exposure to PM_{10}. The summarized risk estimate for long-term exposure was 1.04 (95% CI: 0.85, 1.26) per 10 μg/m^3 increase, with substantial heterogeneity (I^2 = 75.2%, p < 0.001). Two cohort studies and one case-crossover studies study and one time-series study were included for short-term exposure to PM_{10}, and significant association (OR = 1.03, 95% CI: 1.01, 1.05 per 10 μg/m^3 increase) was found with heterogeneity (I^2 = 88.5%, p < 0.001) (Fig. 2(b)).

3.4. NO_2 exposure and the risk of depression

Fig. 2(c) shows the association between exposure to NO_2 and depression. In total, four cohort studies and two cross-sectional studies were pooled, the summarized risk per 5 ppb increase was 1.02 (95% CI: 0.96, 1.09). There was a statistically significant association between short-term exposure and depression (OR = 1.04, 95% CI: 1.01, 1.07 per 5 ppb increase), resulting from the two cohort, one case-crossover and one time-series studies. Substantial heterogeneity was observed for all analyses (Fig. 2(c)).

3.5. Other air pollutant exposure and the risk of depression

Table 2 summarized the association between of depression with exposure to SO_2, CO and O_3. The pooled estimates was significant for short-term exposure to SO_2 (OR = 1.03; 95% CI: 1.00, 1.06 per 5 ppb increase) and CO (OR = 1.01; 95% CI: 1.00, 1.01 per 0.1 ppm increase). The exposure to O_3 was not identified as the risk factor for depression.

3.6. Air pollution exposure and different depression outcomes

Studies were separately analyzed based on different outcomes measures (Table 3). A positive association was found for depressive symptom and long-term exposure to $PM_{2.5}$ (OR = 1.05, 95%: 1.02, 1.09 per 5 μg/m^3 increase) and short-term exposure to PM_{10} (OR = 1.07, 95%: 1.02, 1.12 per 10 μg/m^3 increase). Depressive disorder was related to short-term exposure to $PM_{2.5}$ (OR = 1.00, 95% CI: 1.00, 1.01 per 5 μg/m^3 increase), PM_{10} (OR = 1.02, 95% CI: 1.00, 1.05 per 10 μg/m^3 increase) and NO_2 (OR = 1.03, 95% CI: 1.02, 1.03 per 5 ppb increase). Nevertheless, other association was not statistically significant.

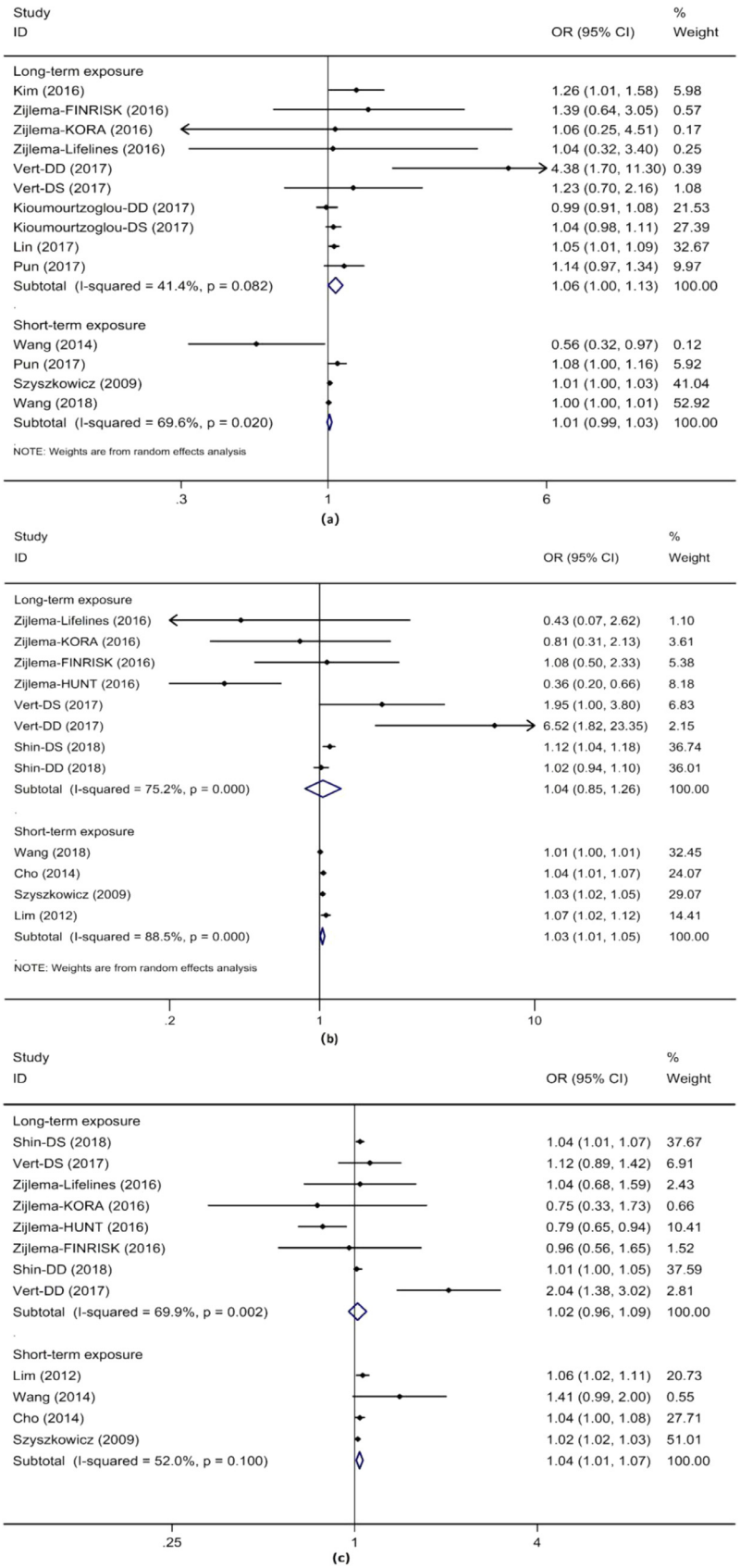

Fig. 2. Forest plot of the association between depression and exposure to air pollution. (a) $PM_{2.5}$; (b) PM_{10}; (c) NO_2. DS, depressive symptom; DD, depressive disorders. Odds ratios per 1 standard deviation for $PM_{2.5}$ (5 μg/m^3), PM_{10} (10 μg/m^3), NO_2 (5 ppb).

Table 2
The effect of exposure to air pollution on depression.

Pollutants	Long-term exposure				Short-term exposure			
	N	OR (95% CI)	*p*	I^2	*N*	OR (95% CI)	*p*	I^2
SO_2	1[a]	0.97 (0.66, 1.41)	0.869	89.3%	2	1.03 (1.00, 1.06)	0.029	76.8%
CO	1	1.05 (0.99, 1.12)	0.133	89.1%	3	1.01 (1.00, 1.01)	<0.001	70.2%
O_3	/	/	/	/	6	1.01 (0.99, 1.03)	0.384	81.3%

[a] , different two group from one study. N, number of included studies. Odds ratios per 1 standard deviation for SO_2 (5 ppb), CO (0.1 ppm) and O_3 (5 ppb).

3.7. Sensitivity analyses

To test the robustness of pooled estimates and explore the sources of heterogeneity, each study was removed one by one for meta-analysis. Besides, to avoid the effect of different outcome on heterogeneity, sensitivity analyses for each pollutant were conducted hierarchically based on the outcome. In the meta-analysis of long-term exposure to $PM_{2.5}$ and depression, the heterogeneity was decreased but the pooled estimate maintained unchanged when the Vert et al. study was excluded (OR = 1.05, 95% CI: 1.02, 1.08; I^2 = 0%) (Fig. S1a) (Vert et al., 2017). The stratified sensitivity analysis confirmed the positive association between long-term exposure to $PM_{2.5}$ and depressive symptoms (Fig. S1b).

For the effect of long-term exposure to PM_{10} and NO_2 on depression, heterogeneity was decreased when the Vert et al. study (Vert et al., 2017) and the HUNT cohort study (Zijlema et al., 2016) were excluded (Fig. S1c and S1d). The stratified sensitivity analyses also showed a decreased heterogeneity and an increased risk of depressive symptoms caused by the exposure to PM_{10} (OR = 1.12, 95% CI: 1.05, 1.19; I^2 = 4.3%; Fig. S1E) and NO_2 (OR = 1.04, 95% CI: 1.02, 1.07; I^2 = 0%; Fig. S1f), after excluding the HUNT cohort study (Zijlema et al., 2016).

3.8. Subgroup analysis

We also conducted subgroup analyses based on study design, population, control for potential confounders (socioeconomic factors, smoking and chronic diseases), pollutants levels. Subgroup analyses were conducted after studies were stratified based on exposure duration and outcome to avoid potential heterogeneity. Table 4 showed the results of the subgroup analyses of the association between long-term exposure to $PM_{2.5}$, PM_{10}, NO_2 and depressive symptom.

As a result, for the association between long-term exposure to $PM_{2.5}$ and depressive symptoms, the pooled estimate of cohort studies was 1.06 (95% CI: 0.99, 1.12), showed an insignificant association. However, the statistical significance was found for meta-analysis of population-based studies. We also found the ORs were higher among studies that adjusted for smoking (1.05, 95% CI: 1.02, 1.08), alcohol consumption (1.05, 95% CI: 1.01, 1.08) but lower among studies that adjusted for chronic disease (1.05, 95% CI: 1.02, 1.09). Heterogeneity remained low in all subgroup analyses.

For subgroup analyses of depressive symptoms and PM_{10} and NO_2 (Table 4). The pooled estimate of the cohort studies and population-based studies were consistent with the overall results. The results demonstrated that the subgroup controlled for socioeconomic, alcohol consumption and smoking showed significant risk. However, the heterogeneity was not decreased. We excluded the studies that contributed to the heterogeneity and re-conducted subgroup analysis (Table S3). The heterogeneity of was successfully decreased, and the pooled estimates were consistent with the original subgroup analyses.

For pollutants levels, the pooled ORs were more significant among the studies with higher median level of $PM_{2.5}$ (OR = 1.05, 95% CI: 1.02, 1.09), PM_{10} (OR = 1.12, 95% CI: 1.05, 1.19), and NO_2 (OR = 1.04, 95% CI: 1.02, 1.07), compared with the relatively low median pollutant levels.

4. Discussion

The environmental factors have been identified as risks for many diseases. Recently, the association of depression with exposure to air pollution has been extensively studied. Several previous review papers have preliminarily summarized studies on the correlations between air pollution and depression but has not quantitatively analyzed the effect size (Buoli et al., 2018; Gladka et al., 2018; Łopuszańska and Makara-Studzińska, 2017). This systematic review and meta-analysis respectively explored the association of depression with exposure to particular matters and gaseous pollutants. We found that air pollutants exposure was associated with increased risk of depression except for O_3. However, few studies have explored the effects of exposure to SO_2 and CO on depression. Therefore, the pooled estimate results were statistically significant but the evidence is weak. In stratified analyses based on exposure duration and depression outcomes, inconsistent results were obtained. However, long-term exposure to $PM_{2.5}$ and short-term exposure to PM_{10}, NO_2, SO_2, CO significantly increased the risk of depression. For different depression outcomes, long-term exposure of $PM_{2.5}$ and short-term exposure to PM_{10} were risk factors for depressive symptom. Moreover, short-term exposure to $PM_{2.5}$, PM_{10}, NO_2 increased the risk of depressive disorders. Overall, we found that an increased risk of depression with $PM_{2.5}$ exposure. However, the results of

Table 3
The effect of exposure of air pollutants on different outcomes.

Pollutants	Depressive symptom				Depressive disorders			
	N	OR (95% CI)	*p*	I^2	*N*	OR (95% CI)	*p*	I^2
$PM_{2.5}$								
Long-term	7	1.05 (1.02, 1.09)	0.002	0.0%	3	1.31 (0.89, 1.91)	0.479	84.9%
Short-term	2	0.88 (0.43, 1.56)	0.549	81.5%	2	1.00 (1.00, 1.01)	<0.001	4.7%
PM_{10}								
Long-term	6	0.70 (0.56, 1.44)	0.652	72.0%	2	2.30 (0.38, 14.01)	0.367	87.7%
Short-term	1	1.07 (1.02, 1.12)	0.004	/	3	1.02 (1.00, 1.05)	0.040	89.6%
NO_2								
Long-term	6	0.97 (0.85, 1.12)	0.710	51.4%	2	1.40 (0.71, 2.76)	0.335	91.8%
Short-term	2	1.16 (0.90, 1.48)	0.260	57.8%	2	1.03 (1.02, 1.03)	<0.001	0%
O_3								
Short-term	2	0.98 (0.83, 1.16)			4	1.00 (0.98, 1.02)		

N, number of included studies. Odds ratios per 1 standard deviation for $PM_{2.5}$ (5 μg/m^3), PM_{10} (10 μg/m^3), NO_2 (5 ppb) and O_3 (5 ppb).

Table 4
Subgroup analysis of long-term exposure to $PM_{2.5}$, PM_{10}, NO_2 and depressive symptom.

Study characteristics	$PM_{2.5}$			PM_{10}			NO_2		
	N	OR (95% CI)	I^2	*N*	OR (95% CI)	I^2	*N*	OR (95% CI)	I^2
Design									
Cohort	5	1.06 (0.99, 1.12)	0%	4	0.58 (0.38, 0.87)	45.%	4	0.83 (0.71, 0.97)	0%
Cross-section	2	1.05 (1.01, 1.09)	0%	2	1.33 (0.80, 2.21)	62.6%	2	1.04 (1.02, 1.07)	0%
Population based	5	1.05 (1.01, 1.09)	0%	6	0.70 (0.56, 1.44)	72.0%	6	0.97 (0.85, 1.18)	51.4%
Pollutant median levels									
High level	3	1.05 (1.01, 1,09)	0%	5	1.12 (1.05, 1.19)	4.3%	3	1.04 (1.02, 1.07)	0%
Low level	4	1.06 (0.99, 1.12)	0%	1	0.36 (0.20, 0.66)	/	3	0.83 (0.71, 0.98)	0%
Adjustment for SES									
Yes	7	1.05 (1.02, 1.08)	0%	5	1.12 (1.05, 1.19)	4.3%	5	1.04 (1.02, 1.07)	0%
No	NA	/	/	1	0.36 (0.20, 0.66)	/	1	0.79 (0.65, 0.94)	/
Adjustment for Smoking									
Yes	3	1.05 (1.02, 1.08)	0%	2	1.33 (0.80, 2.21)	62.6%	2	1.04 (1.02, 1.07)	0%
No	4	1.15 (0.98, 1.34)	0%	4	0.58 (0.38, 0.87)	45.%	4	0.83 (0.71, 0.97)	0%
Adjustment for alcohol consumption									
Yes	2	1.05 (1.01, 1.08)	0%	1	1.12 (1.05, 1.19)	/	1	1.04 (1.01, 1.07)	/
No	5	1.15 (0.99, 1.34)	0%	5	0.82 (0.39, 1.68)	74.9%	5	0.93 (0.77, 1.12)	36.1%
Adjustment for chronic disease									
Yes	3	1.23 (0.68, 2.24)	0%	5	0.75 (0.43, 1.31)	73.4%	5	0.94 (0.78, 1.12)	59.3%
No	4	1.05 (1.02, 1.09)	0%	1	1.95 (1.00, 3.80)	/	1	1.12 (0.89, 1.42)	/

SES, socioeconomic status; *N*, number of included studies; pollutant median levels: $PM_{2.5}$: high, more than 15 μg/m^3; low, less than 15 μg/m^3; PM_{10}: high, more than 15 μg/m^3; low, less than 15 μg/m^3; NO_2: high, more than 20 μg/m^3; low, less than 20 μg/m^3.

PM_{10} exposure were inconclusive.

There were several explanation for the inconsistent results in our stratified analysis. Firstly, the geographical are of the included studies was different, which may influence the concentration of exposure. For example, five out of six included studies explored the association between long-term exposure to PM_{10}, NO_2 and depression were conducted in Europe, where the air pollution was not severe. Hence, the subgroup analysis of different annual concentration was performed (Table 4) and the results showed that long-term exposure to high concentration of $PM_{2.5}$, PM_{10}, NO_2 were associated with higher risk of depressive symptoms. High concentration of air pollution was more likely to be linked to a variety of diseases and cause health damage such as cognitive impairment, psychiatric drug use (Ailshire and Clarke, 2015; Oudin et al., 2016). In addition, investigations on the risk of depressive disorders and short-term exposure to air pollution showed similar results. The study in South Korea with higher levels of PM_{10} reported a much higher risk of depression (OR = 1.155, 95% CI: 1.058, 1.262; PM_{10}: 54.15 μg/m^3) than in Canada (OR = 1.064, 95% CI: 1.038, 1.094; PM_{10}: 19.4 μg/m^3), suggested that different exposure concentrations may lead to different results. However, because of the limited number of included studies in each subgroup, the results need to be interpretated with caution. Second, most of the studies included in short-term exposure analysis were based on case-crossover study design, which used the same sample as the control group and case group to minimize the bias caused by the imbalance of socio-economic factors. This may also contribute to the difference between the long-term and short-term subgroup results. Third, various scales for depressive symptom assessment, with different cut-off scores, were used. However, the depression assessments were based on medical documents. This might have led to inconsistent results between depressive disorders and depressive symptoms.

In sensitive analyses, the data from the HUNT cohort study contributed significantly to the heterogeneity and affect the pooled estimates for the positive association between depressive symptom and long-term exposure to PM_{10} and NO_2 (Zijlema et al., 2016). There several reasons for altered results and heterogeneity caused by this study. First, the air pollution exposure in the HUNT study was predicted by land use regression model (LUR), with outcomes variables (pollutant concentration) from environment monitoring networks in 17 European countries (Zijlema et al., 2016). Nevertheless, in other European studies (Vert et al., 2017; Zijlema et al., 2016), they used specially established regional station data as the outcomes variables of LUR. Therefore, the predicted value of exposure from the HUNT study might be short of several important local predictors (e.g., traffic count), while a large proportion of the included studies were based on regional station data (Table S2). Second, the HUNT study failed to control important confounders (e.g., smoking, alcohol, socioeconomic), which may have resulted in bias. Third, the air pollution level may have caused inconsistent findings between studies. The studies, which concluded an association between air pollution and depression, were mostly located in areas with high air pollution, such as Asia (Cho et al., 2014; Lim et al., 2012). Taking the level of PM_{10} as an example, mean concentration in Norway from the HUNT cohort was 11.0 μg/m^3, which was a fifth in Korea (54.15 μg/m^3) (Cho et al., 2014), and a half in the Netherland (23.95 μg/m^3) (Zijlema et al., 2016). Based on the results of sensitivity analysis, the absence of correlation between long-term exposure to PM_{10}, NO_2 and depressive symptoms should also be interpreted with caution. The data from Vert et al. was also an important source of heterogeneity (Vert et al., 2017) due to few cases and self-report outcome assessment. Despite various air assessment methods, depression assessment method, and confounders controlling, our findings may be instructive for future research.

All of the included studies were observational studies with different study setting, population and potential confounders controlling. Therefore, we performed further subgroup analyses to explore whether these factors have an influenced the results. Pooled estimates from population-based studies were consistent with the total estimates, which showed the robustness of our main results. However, pooled estimates for subgroup of different study design were not consistent with the main results, this could be attribute to the limited number of included studies in each subgroup. Among potential confounders, a large number of previous studies had confirmed that smoking, alcohol consumption, chronic diseases comorbidity and low socioeconomic level (e.g., lower income and education) were risk factors for depression (Boden et al., 2010; Haynes et al., 2005; Huang et al., 2010; Korhonen et al., 2017; Messias et al., 2011). Moreover, we find the association of depressive symptoms with long-term exposure to $PM_{2.5}$ was more significant among the studies that have adjusted for smoking, chronic diseases. Similarly, for exposure to PM_{10} and NO_2, we observed a higher risk of depressive symptom for subgroups controlled for smoking, alcohol consumption and chronic disease, with decreased heterogeneity (Zijlema et al., 2016). These findings suggested that the control of

confounding factors was of importance for elucidation of the association.

Our findings were consistent with the experimental studies except for O_3. Mice exposed to particulate matters for a long time displayed more depressive-like responses (Fonken et al., 2011; Liu et al., 2018). Inhaling simulated air pollution containing nitrogen dioxide and carbon dioxide also increased the incidence of depressed behavior in mice (Salvi et al., 2017). The mechanism between air pollution and depression was not clearly understood, but several hypotheses have been proposed. Firstly, exposure to particulate matters such as nitrogen dioxide and ozone could lead to systemic inflammation (Block and Calderon-Garciduenas, 2009; Perret et al., 2017; Wigenstam et al., 2016). Systemic inflammation transfers inflammation to the brain through a systemic induced cytokine response and results in neuroinflammation and neurotoxicity (Block and Calderon-Garciduenas, 2009). It has been confirmed that systemic inflammation is closely related to sad mood and affective (Benson et al., 2017). Second, exposure to particulate matters could lead to the increase of hippocampal pro-inflammatory cytokine expression and the changes of the dendrites in the hippocampus, which increase the depressive-like behaviors in animal models (Fonken et al., 2011). Third, air pollution exposure also increases neurobiological oxidative stress (Calderon-Garciduenas et al., 2015), and the high level of oxidative stress was observed in brain of depressed patients (Wigner et al., 2017). Fourth, air pollution can damage human brain microvascular endothelial cell viability and decrease tight junction protein expression, which subsquently cause damage to the blood–brain barrier. Air pollution can also decrease the protective effect of the brain and induce inflammation in the brain (Block and Calderon-Garciduenas, 2009).

While ozone exposure can also lead to neurobiological oxidative stress, systemic inflammation, and the damage to the blood–brain barrier (Dantzer and Kelley, 2007; Mokoena et al., 2015), this meta-analysis did not evidence the link to depression. The morbidity of depression is higher in winter (Geoffroy et al., 2014). However, ozone levels are higher in summer with higher temperatures and sunlight (Tiwari et al., 2008). Therefore, there is a seasonal variation between the peak of depression prevalence and the peak of ozone exposure. Furthermore, ozone is a 'secondary' pollutant, it is hard to quantify and generated from primary air pollution (e.g., NO_2) (Chardon et al., 2007).

Our study has some limitations. While we have extensively retrieved studies for inclusion, the number of studies in each subgroup is limited. The total pooled sample size is relatively large, sample size is dramatically different in studies and further study is still required. While ICD was consistently used in the diagnosis of depression in all studies, screening method for depressive symptoms were various, and this may affect the identification of depressive symptoms. Publication bias was not tested in this meta-analysis because less than 10 studies were available for each subgroup. Also, we did not perform subgroup analyses based on age, gender and season due to insufficient number of studies.

In conclusion, this systematic review and meta-analysis provided evidence for the association between depression and exposure to $PM_{2.5}$, PM_{10}, NO_2, SO_2, CO. The management of air pollution, especially in areas with severe air pollution, is of great importance for potential prevention of depression. Further studies are warranted due to some inconsistence in the results. Further studies should pay more attention to study settings, including outcome assessment, population sampling, exposure assessment method and control for important confounders,.

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Conflicts of interest

The authors declare that they have no competing interests.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.psychres.2019.04.019.

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