

Mental health impacts of air pollution, chemicals, and noise



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Cover design: EEA

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Layout: EEA / ETC HE European Topic Centre on Human Health and the Environment.

Publication Date: 28 January 2026

DOI: 10.5281/zenodo.18374150

Version: 1

EEA activity: Human health and the environment

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Preparation of this report has been co-funded by the European Environment Agency as part of a grant with the European Topic Centre on Human health and the environment (ETC HE) and expresses the views of the authors. The contents of this publication does not necessarily reflect the position or opinion of the European Commission or other institutions of the European Union. Neither the European Environment Agency nor the European Topic Centre on Human health and the environment is liable for any consequences stemming from the reuse of the information contained in this publication.

How to cite this report:

Jiang, X., Joss, M. K., Santjer, C., Bathow, M., Plass, D., & Rösli, M. (2026). Mental health impacts of air pollution, chemicals, and noise. ETC HE Report 2025/13. Zenodo. <https://doi.org/10.5281/zenodo.18374150>

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ETC HE coordinator: Stiftelsen NILU, Kjeller, Norway (<https://www.nilu.com/>)

ETC HE consortium partners: Federal Environment Agency/Umweltbundesamt (UBA), Aether Limited, Czech Hydrometeorological Institute (CHMI), Institut National de l'Environnement Industriel et des Risques (INERIS), Swiss Tropical and Public Health Institute (Swiss TPH), Universitat Autònoma de Barcelona (UAB), Vlaamse Instelling voor Technologisch Onderzoek (VITO), 4sfera Innova S.L.U., klarFAKT e.U.

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Acknowledgements

The EEA task manager was Gerardo Sanchez. Many thanks for the inspiring discussions and helpful feedback to draft reports. We are also grateful for the help with data extraction of meta-analyses from Jasmin Tröhler.

1 Summary

Mental, behavioural and neurodevelopment disorders such as depression, anxiety and mood disorders, substance-related disorders, schizophrenia, personality disorders, suicide and trauma emerge from a complex interplay between intrinsic factors like genetics, social and economic determinants, psychological, lifestyle, and environmental factors, many of which are not well understood. The aim of this report is to provide an evidence review covering the impact of environmental determinants on mental health with a focus on ambient air pollution, chemicals and transportation noise.

The evidence is captured using an umbrella+ review approach. This means that the primary focus is well-conducted systematic reviews complemented with narrative reviews if they provide additional information. For exposure-outcome combinations with lack of good quality and recent reviews, original European studies of high-quality are also considered. High-quality studies are generally large prospective cohort studies with high-quality exposure and outcome assessment although cross-sectional studies are also considered to be suitable for transient or recurrent diseases.

1.1 Air pollution and mental health

Air pollution can affect the brain through several pathways, including direct entry of particles via the olfactory system, transfer of gaseous and ultrafine pollutants across the blood–brain barrier, and inflammatory responses originating in the lungs. These mechanisms contribute to neuroinflammation and oxidative stress, which are considered central to brain damage. Neuroimaging studies have linked air pollution to structural and functional alterations in brain regions like the cerebellum, corpus callosum, and caudate, as well as changes in connectivity and cerebral blood flow. Pollutants also interfere with neurotransmitter systems, stress hormone regulation, and epigenetic processes, which may increase risks for cognitive impairment, depression, and other mental health disorders.

The existing epidemiological research points towards increased risk of depression with long-term air pollution with most evidence for PM_{2.5} and NO₂. It is also shown that short-term peaks of air pollution exposure are consistently associated with worsening of symptoms of depression or schizophrenia and some evidence exists for increased numbers of suicides or suicide attempts. For other mental health diseases, evidence for an association with long term air pollution is limited or inconsistent.

Table 1.1 Summary of evidence levels for air pollution exposure–outcome pairs

Exposure	Outcome	Level of evidence
Long-term		
PM _{2.5} and PM ₁₀	Depression	Moderate
	Anxiety	Low
	Schizophrenia	Low
NO ₂	Depression	Moderate
	Anxiety	Low
	Schizophrenia	Low
Ozone	Depression	Low
	Anxiety	Low

Short-term		
PM _{2.5} and PM ₁₀	Depression	Moderate
	Suicide	Low
	Exacerbation of schizophrenia	Moderate
NO ₂	Depression	Moderate
	Suicide	Low
	Exacerbation of schizophrenia	Moderate
Ozone	Depression	Low
	Suicide	Low
SO ₂	Depression	Moderate
	Suicide	Low
	Exacerbation of schizophrenia	Moderate
CO	Depression	Moderate
	Suicide	Low

1.2 Environmental chemicals and mental health

For various types of chemicals many potential mechanisms of action are mentioned in the literature, but few of them have been definitively confirmed. A key mechanistic pathway is the disruption of biochemical processes through direct cellular damage or interference with intracellular signalling pathways. As a consequence, oxidative stress is triggered, which initiates multiple downstream processes leading to neuroinflammation. Chronic neuroinflammation and long-term impairments in the neurotransmitter systems contribute to the manifestation of mental health outcomes such as depression, schizophrenia and anxiety. Two of these critical neurotransmitter systems are the dopaminergic and serotonergic systems, both of which were frequently cited in the reviewed literature as potential mechanistic targets of environmental chemicals.

In epidemiological studies, *second-hand smoke (SHS)* is consistently linked with depression and schizophrenia, with stronger effects reported in vulnerable groups such as pregnant individuals and children. Findings for anxiety remain somewhat less consistent. Most literature on *heavy metals* focussed on *lead* indicating an association between prenatal or childhood lead exposure and both, depression and schizophrenia. For other heavy metals such as *cadmium*, *mercury*, *tin*, *copper* and *manganese* the results of the reviews are not consistent with some sporadic associations observed for prenatal and childhood exposure in relation to anxiety. In terms of *endocrine-disrupting chemicals*, a potential link between prenatal exposure to bisphenol A and depression or anxiety in childhood has repeatedly been observed. Further, there is a generally consistent link between exposure to *pesticides* and various mental health outcomes, primarily depression, but also schizophrenia and anxiety. Evidence for effects of other environmental exposures such as *phthalates* and *PFAS* on mental health outcomes is inconsistent or absent and consistency varies by chemical.

Table 1.2 Summary of evidence levels for chemical exposure–outcome pairs

Exposure	Outcome	Level of evidence
SHS (pre- and postnatal)	Depression	Moderate
	Schizophrenia	Moderate
	Anxiety	Low
Lead (pre- and postnatal)	Depression	Moderate
	Schizophrenia	Moderate
Other metals (Cd, Hg, Sn, Cu, Mn, Mg)	Depression, anxiety, schizophrenia	Low
BPA (prenatal)	Depression in children and adolescents	Moderate
	Anxiety in children and adolescents	Moderate
Phthalates	Depression	Low
Pesticides	Depression	Moderate
	Anxiety	Low
	Schizophrenia	Low
PFAS (prenatal)	Depression	Low
Mixtures (prenatal)	Depression in children and adolescents	Low

Abbreviations: SHS = Second-hand Smoke, Cd = cadmium, Hg = mercury, Sn = tin, Cu = copper, Mn = manganese, Mg = magnesium, BPA = Bisphenol A, PFAS = per- and polyfluoroalkyl substances.

1.3 Noise and mental health

Transportation noise may affect mental health through a direct pathway, in which very high noise levels can cause auditory damage, and a more relevant indirect pathway, where long-term noise exposure interferes with sleep and activates the sympathetic nervous system and the hypothalamic–pituitary–adrenal (HPA) axis, leading to repeated release of stress hormones such as cortisol, adrenaline, and noradrenaline. Chronic activation of the stress systems and disturbed sleep are well established risk factors for depression, anxiety, and suicidal behaviour most likely via inducing oxidative stress and subsequent neuroinflammation.

Relatively few research addressed mental health problems in relation to transportation noise exposure directly, but evidence from prospective cohort studies is emerging that transportation noise is a risk factor to develop depression. It may also exacerbate existing depressive and anxiety disorders and trigger suicides. In children, behavioural problems including attention deficit hyperactivity disorder (ADHD) are repeatedly found to be related to transportation noise exposure. For other mental health outcomes, research is mostly absent.

Table 1.3 Summary of evidence levels for noise exposure–outcome pairs

Exposure	Outcome	Level of evidence
Road traffic noise	Depression (adults)	Moderate
	Anxiety (adults)	Low
	Mental health problems (children/adolescents)	Moderate
	Behavioural problems (children/adolescents)	Moderate
Railway noise	Depression (adults)	Low
	Anxiety (adults)	Low
	Mental health and behavioural problems (children/adolescents)	Low
Aircraft noise	Depression (adults)	Moderate
	Anxiety (adults)	Low
	Behavioural problems (children/adolescents)	Low

1.4 Conclusion

Although many questions remain open in terms of magnitude of the risk, current evidence indicates that air pollution, various chemicals and transportation noise are relevant risk factors for mental health disorders, in particular for depression and depressive disorders. Studies find that the developing brain, from early prenatal phase up to adolescence, is especially vulnerable, because of ongoing processes such as synaptogenesis, myelination, and blood–brain barrier maturation, which may be disrupted by environmental exposures.

In Europe, mental health problems are on the rise. In 2023, more than 11 million disability-adjusted life years (DALYs) were lost in the European Union due to mental health disorders according to the Global Burden of Disease 2023 study (IHME, 2025). This implies that even small individual risks from air pollution, chemicals and transportation noise are likely to make significant population-level contributions to mental health burden given these widespread exposures. Future research should focus on characterizing long-term consequences and lifetime cumulative effects of exposure in longitudinal studies with rigorous outcome and exposure assessment, that include other environmental exposures to disentangle pollutant mixture effects. Susceptible windows of exposure as well as individual differences in susceptibility should be assessed. The contribution of poor socioeconomic status, poor living conditions, poor nutrition or individual health risk behaviours should be better understood and considered in all policies as expressed in the health in all policies approach (HiAP).

The environment is not only a risk factor but certain environmental exposures such as contact with nature in various form have been observed to provide a certain degree of protection against some mental disorders, facilitating stress reduction, mental recovery, or symptom reduction. Thus, beside reduction in environmental pollution, prevention should also address the positive potential of the environment for interventions to reduce the mental health burden in the society.

2 Introduction

Mental health emerges from a complex interplay between intrinsic factors like genetics, social and economic determinants, and psychological, lifestyle, and other factors, many of them not well understood. Emerging evidence points at an influence of environmental exposures such as noise, air pollution, drinking water, diet, weather conditions, housing conditions or lack of green space. Environmental factors may trigger mental disorders, aggravate symptoms or contribute to the aetiology of diseases by means of long-term exposure (Cao et al., 2024; Hahad et al., 2025; Rückle et al., 2025). Mental health conditions include mood disorders (such as depressive disorders and bipolar disorders), anxiety and fear-related disorders, schizophrenia and other primary psychotic disorders, substance use and addictive disorders, personality disorders, trauma- and stressor-related disorders, feeding and eating disorders, sexual dysfunctions, and neurodevelopmental disorders (such as attention-deficit/hyperactivity disorder and autism spectrum disorder) (WHO, 2024). Suicide is a possible outcome or behaviour associated with several mental disorders.

In European Union, more than 11 million disability-adjusted life years (DALYs) were lost in 2023 due to mental health disorders, according to the Global Burden of Disease study (IHME, 2025). Mental health disorders ranked sixth overall, following cancer, cardiovascular diseases, musculoskeletal disorders, neurological disorders, and unintentional injuries. Multiple studies show a significant rise in the prevalence and incidence of mental health disorders across Europe, particularly since the late 2000s. For example, in the Netherlands, the 12-month prevalence of any mental disorder increased from 17.4% (2007–2009) to 26.1% (2019–2022), with sharper increases among young adults and urban populations (Lozano-Sánchez et al., 2024; Momen et al., 2025; ten Have et al., 2023). Anxiety and depressive disorders are the most common diseases, with pooled prevalence rates among youth at 7.9% and 1.7%, respectively. Eating disorders and ADHD have shown the largest relative increases (Sacco et al., 2024). On the other hand, the incidence of self-harm has decreased in the last decades (Castel Pietra et al., 2022).

Environmental factors are not only risk factors but certain environmental exposures or contact with nature in various forms have been observed to provide a certain degree of protection against some mental disorders, facilitating stress reduction, mental recovery, or symptom reduction. Thus, research on environmental interventions is of interest for public health to reduce the mental health burden in the society (Bratman et al., 2012).

The evidence on the causal links between mental health and some environmental factors is far from conclusive, whereas for some diseases a link with specific exposures is increasingly solidly established. For example, the effects of lead exposure on behavioural and cognitive outcomes are well established (EEA, 2024). A particular challenge of the research on air pollution, noise, housing conditions, neighbourhood social capital, and several other environmental and sociodemographic variables is their mutual correlation, hampering the causal interpretation.

3 Aims

The overall aim of this report is to provide an overview of the evidence on the impact of environmental determinants on mental health with a focus on ambient air pollution, chemicals and noise. In particular, the following objectives are defined:

- To provide a list of known and suspected mental health outcomes caused or influenced by each of these three risk factors.
- To summarize the most relevant evidence on the effect of air pollution, chemicals and noise on mental health.
- Where available, exposure-response functions for relevant outcomes derived from high-quality studies, recent systematic reviews or meta-analyses are presented.

4 Methods

4.1 Umbrella+ review

An umbrella+ review approach is used to summarize evidence on mental health outcomes in Europe with respect to ambient air pollution, chemicals and noise. With umbrella+ review, we mean that for selected health outcomes, where there is a lot of evidence and/or the scope is huge (i.e. chemicals) we limit the review to key reports and systematic reviews. In addition, narrative reviews are included if they provided additional information on top of the systematic reviews. For exposure-outcome combinations with lack of good quality reviews or lack of solid study base from Europe, original European studies of high-quality were considered. High-quality studies are generally large prospective cohort studies with high-quality exposure and outcome assessment.

4.1.1 PECOS approach

The umbrella+ review process follows the PECOS (Participants, Exposures, Comparators, Outcomes and Study design) scheme. The study question was “In the general population or in population subgroups, including pre-diseased or diseased participants (P), what is the increase in risk of health effects (O) per unit increase (C) of long-term or short-term exposure to ambient air pollution, environmental chemicals or noise (E), identified by in reviews of epidemiological studies or original studies (S)?”

Population

The review focusses on studies in the general population and in vulnerable populations. The latter refers mainly on exacerbations of diseases or symptoms in patients with existing mental health disorders or high-risk groups, such as children or pregnant individuals.

Exposures

This review focuses on ambient air pollution, and exposure to environmental chemicals and noise. Eligible studies had to study the environmental exposures of the general population. We did not consider occupational exposure and exposure from accidents. Active smoking was also not of interest for this review.

Ambient air pollution was defined as objectively measured and/or modelled ambient air pollutants of particulate matter, oxides of nitrogen, ozone, SO₂, soot/black carbon, carbon monoxide benzene, the metals (in PM) Lead, Nickel, Zinc, Vanadium, Iron, Copper, Cadmium, Arsenic, traffic related noise pollution as done in a previous report (EEA, 2024). See Table 4.1, Table 4.2, and Table 4.3 for the definition of inclusion and exclusion criteria for the exposures.

Table 4.1 Definition of inclusion and exclusion criteria for the exposure with ambient air pollution

P(E)C(O)S	Inclusion	Exclusion
Exposure	Ambient air pollution with particulate matter air pollution including various measures of ultrafine particles, measures for oxides of nitrogen, ozone, SO ₂ , Soot/black carbon, benzene, the metals (in PM) Lead, Nickel, Zinc, Vanadium, Iron, Copper, Cadmium, Arsenic, measures of traffic related air pollution	Indoor and occupational settings and pollutants such as tobacco smoke, other outdoor air pollutants which are not of interest, indirect or not objective measures of exposure such as urinary metabolites or felt exposure levels/annoyance

Table 4.2 Definition of inclusion and exclusion criteria for the exposure with environmental chemicals

P(E)C(O)S	Inclusion	Exclusion
Exposure	Environmental chemicals that the general population experiences/can experience through the occurrence of chemicals in environmental media such as soil, (drinking) water, but also dietary occurrences, children exposed in the womb	Occupational settings, exposure after (natural) disasters/accidents, individual case studies, self-poisoning with suicidal intent, active smoking, air pollution

Table 4.3 Definition of inclusion and exclusion criteria for the exposure with noise

P(E)C(O)S	Inclusion	Exclusion
Exposure	Environmental noise exposure from transportation sources including road traffic, railway, and aircraft noise; exposure assessed using objective measures such as modelled or measured noise levels in decibels (e.g., L _{den} , L _{Aeq}) typically derived from noise mapping or monitoring systems	Noise exposure from indoor, occupational, or recreational sources; studies estimating exposure based on distance to the noise source; studies using subjective exposure measures only, such as noise annoyance, self-reported disturbance, or perceived noise levels, without accompanying objective noise level data

Outcomes

The following health outcomes are of priority for this review:

- Depression, depressive disorders etc.
- Anxiety disorders
 - Generalized Anxiety Disorder (GAS)
 - Obsessive-Compulsive Disorder (OCD)
 - Panic Disorder
 - Phobic Disorders
- Mood disorders
 - Bipolar
 - Depressive
- Substance-related disorders
- Schizophrenia
- Personality disorders
- Psychological sexual dysfunctions
- Feeding and Eating Disorders
- Suicide
- Trauma and Stressor Related Disorders

Depending on the outcome assessment methods considered to be eligible are doctor/clinical diagnosis, hospital records (incl. admission), death certificates (if applicable), self-reports, scores of established/validated mental health symptom questionnaires.

Eligible Studies

This review focuses on epidemiological research. We include systematic reviews, meta-analyses and narrative reviews of epidemiological studies. Further, original studies were included for ambient air pollution and noise. For incidence, only cohort studies are considered to be eligible. For prevalence, cross-sectional studies are eligible. We do not include human experimental research, in-vitro or in-vivo studies.

4.2 Literature search

4.2.1 Databases and time frame

Depending on the PECOS scheme, different databases and sources were searched to optimise adequate and efficient coverage of locating relevant reviews/studies for the umbrella+ review. We focused on the most recent evidence studies, and the literature search focused primarily on the last 10 years (January 2015-May 2025).

For ambient air pollution, we searched publications from 1 January 2015 to 11 April 2025 in PubMed and PsycINFO. Meanwhile, the special topic database LUDOK (Kutlar Joss and Probst-Hensch, 2023) was also used to specifically identify high-quality European studies to reflect on evidence from European populations. Research from European populations has only been published in the last few years. Therefore, the included reviews often only rely on studies from other world regions, which might be less representative for the European context.

For environmental chemicals, we searched publications from 1 January 2015 to 28 April 2025 in the databases PubMed and SCOPUS and from 1 January 2015 to 12 May 2025 in PsycINFO.

For noise, the literature search was designed to build on the evidence base compiled for the World Health Organization Environmental Noise Guidelines for the European Region (WHO ENG) on noise and mental health (Clark and Paunovic, 2018), which synthesised 29 studies published from January 2005 and October 2015. We searched publications from 1 October 2015 to 4 April 2025 in PubMed and from 1 October 2015 to 8 April 2025 in PsycINFO.

The search was conducted by one person per exposure. As most of the peer-reviewed literature is published in English, our search was restricted to the English language.

4.2.2 Search terms

Outcome search terms were decided to be the same for all exposures and the specific search terms each exposure domain were developed separately for each research area (see Annex).

4.2.3 Study selection

Search results were imported into Rayyan (Ouzzani et al., 2016), a web-based systematic review management tool, for organization and screening of articles. The software was used to identify and remove duplicate records automatically, followed by manual verification of duplicate detection. The title and abstract screening process was conducted within Rayyan, with one reviewer against the predetermined inclusion and exclusion criteria.

4.2.4 Data extraction

Data from each study was extracted by one researcher. From systematic and narrative reviews we extracted information on author, publication year, type of review, end of search date, range of publication years of included studies, quality assessment method, pollutants studied, outcomes studied, results.

From original studies the following information was extracted for air pollution/noise studies: author, publication year, country, study name, study design, population under study, number of participants, exposure assessment method, effect measure including 95 % confidence interval, qualitative results for the pollutants under study, and results of analyses with noise/air pollution as a confounder or in co-pollutant models.

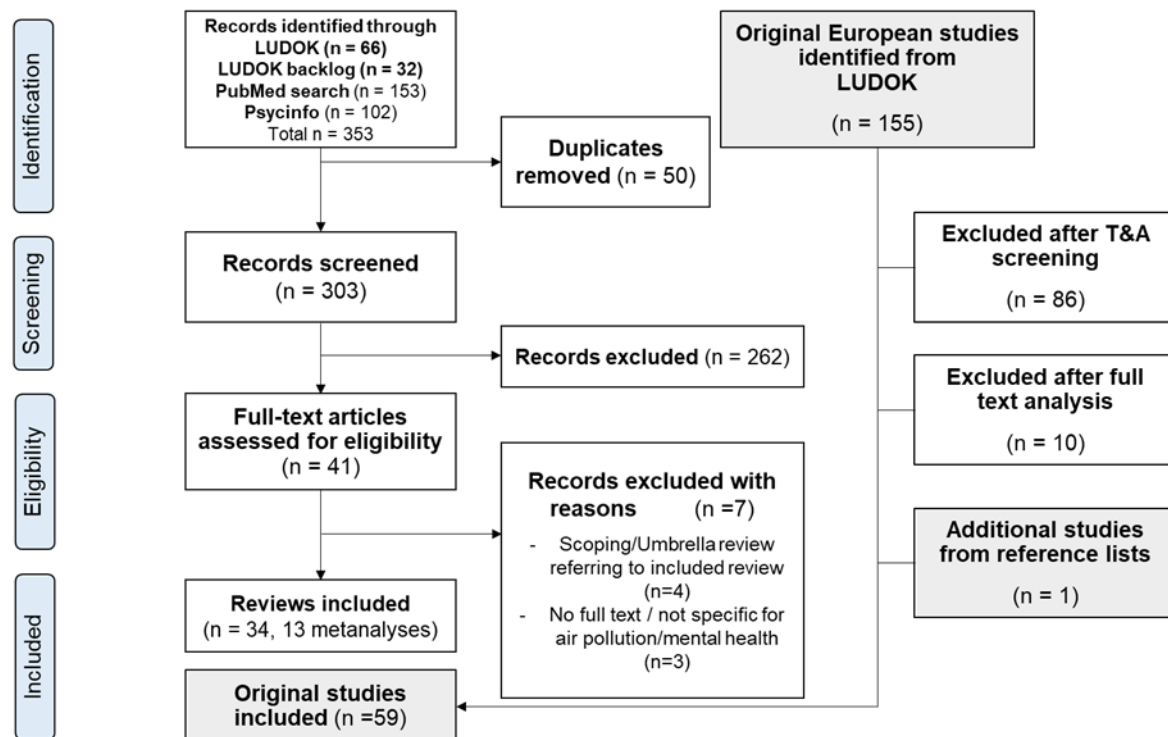
5 Results

5.1 Air pollution and mental health

5.1.1 Study characteristics

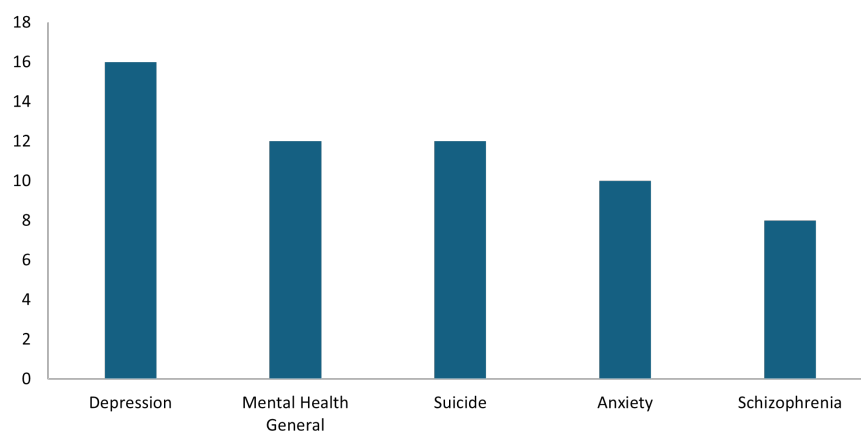
A total of 353 reviews were identified in the database searches, leaving 303 original articles for screening after duplicate removal. 41 reviews entered the full text review, of which 7 articles were excluded due to their design as umbrella reviews containing reviews that were already included without giving additional insights into the matter ($n = 4$), due to their unspecificity regarding the role of air pollution in mental health ($n = 2$) and non-availability of full text ($n = 1$). Of the included 34 reviews, 13 were meta-analyses. The LUDOK database identified 99 European original studies of which 59 were selected to supplement the findings from the reviews with European data. An overview of the screening process is provided in the PRISMA flow diagram (Figure 5.1).

Figure 5.1 PRISMA flow chart for the systematic search and identification of (1) reviews studying mental health outcomes in association with ambient air pollution and (2) original studies conducted in Europe on health effects of air pollution on mental health



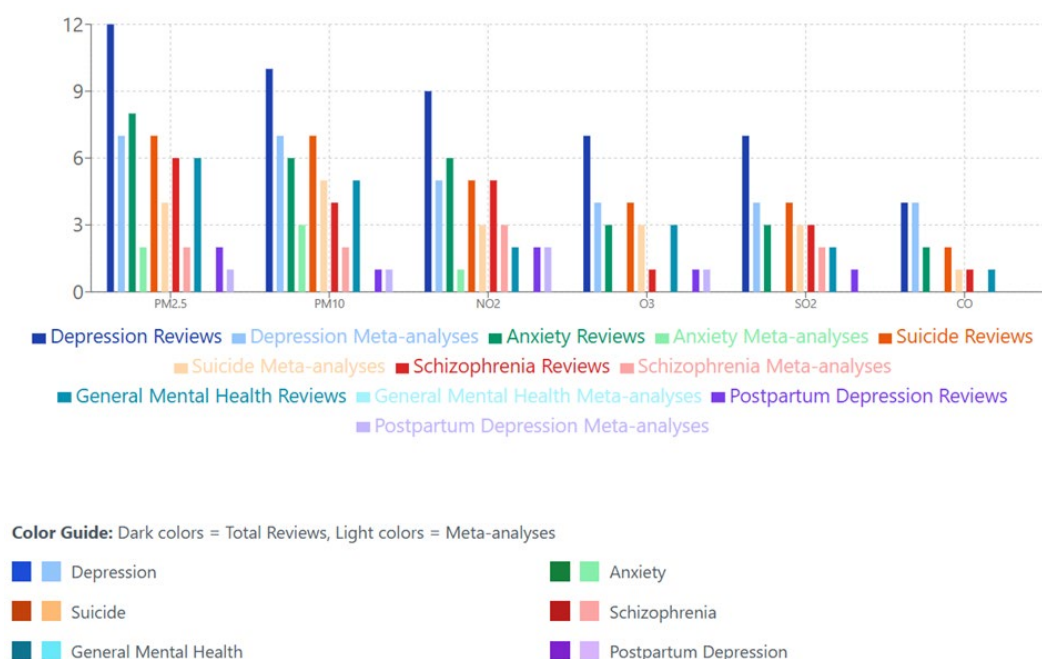
The most reviewed mental health outcome was depression (16 reviews), followed by reviews on suicide ($n = 12$), anxiety ($n = 10$), more general or combined description of mental health outcomes (9 reviews), and Schizophrenia ($n = 8$) (Figure 5.2). We were not able to identify dedicated reviews on outcomes such as bipolar or personality disorders, psychological sexual dysfunctions, feeding and eating disorders or substance-related disorders. Effects of air pollution on mental health in children, adolescents or over the life course were studied in five reviews (Cardenas-Iniguez et al., 2022; Herting et al., 2024; Morrel et al., 2025; Trombley, 2023; Xie et al., 2023).

Figure 5.2 Number of reviews included per mental health outcome. No reviews identified on bipolar or personality disorders, psychological sexual dysfunctions, feeding and eating disorders or substance-related disorders



From the pollutants, particulate matter was the most addressed pollutant in reviews (PM_{2.5} in 21 reviews, PM₁₀ in 16 reviews). NO₂ was studied in 15 reviews, SO₂ in eleven, ozone in ten, with one review exclusively reviewing ozone effects (Zhao et al., 2018), and CO in five reviews. Data from 13 meta-analyses were extracted. The distribution of reviews per pollutant-outcome pair (dark colour) and meta-analyses per pollutant-outcome pair (lighter colour) can be found in Figure 5.3.

Figure 5.3 Number of studies by pollutant and mental health outcome



Note: Darker colours indicate the overall number of reviews for the specific pollutant outcome pair. Lighter colours indicate the number of meta-analyses.

Source: Figure created with assistance from Claude (Anthropic, 2025).

As supporting evidence from Europe, 59 studies were selected from the LUDOK database (see Annex). 37 studies informed on European study results regarding depression endpoints including symptoms of depression and one study on postpartum depression (Latham et al., 2021; Jin et al., 2024; Newbury et al., 2024b; Nobile et al., 2023; Pan et al., 2024; Roberts et al., 2019; Yang et al., 2023b; Yao et al., 2025; Yuan et al., 2025; Li et al., 2023a; Gao et al., 2023; Motoc et al., 2025; Brehm et al., 2024; Altuğ et al., 2020; Hao et al., 2022; Allaouat et al., 2021; Ma et al., 2024; Wu et al., 2022; Petrowski et al., 2021; Meng et al., 2024; Generaal et al., 2019; Lyons et al., 2024; Pelgrims et al., 2021; Vert et al., 2017; Zare Sakhvidi et al., 2022; Hautekiet et al., 2022; Bai et al., 2024c; Thompson et al., 2024; Zhao et al., 2019, 2020; Fan et al., 2024; Borroni et al., 2024; Pignon et al., 2022; Muhsin et al., 2022; Cadman et al., 2024; Wu et al., 2023b; Zijlema et al., 2016), 22 studies informed on anxiety endpoints (Bai et al., 2024a; Brehm et al., 2024; Fan et al., 2024; Gao et al., 2023; Hautekiet et al., 2022; Jin et al., 2024; Lyons et al., 2024; Ma et al., 2024; Meng et al., 2024; Muhsin et al., 2022; Newbury et al., 2024a; Nobile et al., 2023; Pan et al., 2024; Pelgrims et al., 2021; Petrowski et al., 2021; Roberts et al., 2019; Thompson et al., 2024; Vert et al., 2017; Yang et al., 2023b; Yao et al., 2025; Yuan et al., 2025; Zhao et al., 2020), 18 articles analysed combined or more general mental health endpoints (Abed Al Ahad et al., 2022; Andersen et al., 2022; Bakolis et al., 2021; Bernardini et al., 2019; Bloemsma et al., 2022a, 2022b; Dzhambov et al., 2018; Hautekiet et al., 2022; Muhsin et al., 2022; Pan et al., 2024; Pelgrims et al., 2021; Pinheiro Guedes et al., 2024; Reuben et al., 2021; Ronaldson et al., 2023; So et al., 2022; Yao et al., 2025; Yuan et al., 2025; So et al., 2023), including symptoms, emergencies, mortality or self-rated health. 17 studies investigated schizophrenia endpoints or psychotic experiences (Antonsen et al., 2020; Bai et al., 2024a; Bakolis et al., 2021; Fan et al., 2024; Horsdal et al., 2019; Liu et al., 2024; Ma et al., 2024; Newbury et al., 2019, 2021; Nobile et al., 2023; Pan et al., 2024; Pignon et al., 2022; Reuben et al., 2021; Zhang et al., 2024; Yao et al., 2025; Yuan et al., 2025; Newbury et al., 2024b).

Ten studies investigated endpoints related to bipolar disorder (Carugno et al., 2021; Fan et al., 2024; Hao et al., 2022; Li et al., 2023b; Ma et al., 2024; Nobile et al., 2023; Pan et al., 2024; Pignon et al., 2022; Yuan et al., 2025; Bai et al., 2024c). Four studies analysed mortality endpoints related to suicides or attempted suicides or self-harm (Andersen et al., 2022; Casas et al., 2017; Hautekiet et al., 2022; Mok et al., 2021). Finally, three studies investigated various endpoints such as anorexia nervosa, OCD obsessive compulsive disorder or post-traumatic stress disorder, substance abuse, overall personality disorders (Ma et al., 2024; Muhsin et al., 2022; Nobile et al., 2023). A large number of publications relied on data of the UK biobank (15) (Fan et al., 2024; Gao et al., 2023; Hao et al., 2022; Jin et al., 2024; Liu et al., 2024; Ma et al., 2024; Meng et al., 2024; Pan et al., 2024; Zhang et al., 2024; Li et al., 2023b; Wu et al., 2022; Yang et al., 2023b; Yao et al., 2025; Yuan et al., 2025).

Data of the UK twin study E-RISK Twin was analysed in four publications (Latham et al., 2021; Newbury et al., 2019; Reuben et al., 2021; Roberts et al., 2019). Other cohorts and studies were analysed in one or two papers at maximum.

UK studies dominated with 26 publications from six cohorts (UK biobank, SLAM, SELCoH, SCAMP, E-Risk Twin, ALSPAC), followed by five publications each from Denmark (DNR, DNBC, administrative birth registry, iPSYCH) and Germany (USUMA, SALIA, GINIplus, LISA, AOK PLUS, register study), four pan-European studies, three each from Belgium, the Netherlands, and Sweden, two from France, and one each from Bulgaria, Finland, Ireland, Portugal, and Spain.

28 studies used a cohort design, 25 articles analysed their data cross-sectionally, one publication analysed both longitudinal and cross-sectional data, 4 studies used a time-series design and one a case-control study design. Table 5.1 provides the overview of the reviews identified from our search.

Table 5.1 Overview of reviews

Author (Year)	Type of review	Publication range	End search date	Quality assessment	Pollutants studied	Outcomes studied	Results / Conclusions by study authors
Trombley (2023)	NR	2011-2022	May 2022	JHNEBP	PM	General mental health	Numerous negative mental health outcomes among children and adolescents have been potentially identified in relation to PM _{2.5} exposure. The strongest evidence supports a possible correlation between PM _{2.5} exposure and adolescent mental health outcomes, including probable depression, probable anxiety, probable conduct disorders, and emergency psychiatric issues, although there were still some studies that contradicted these associations.
Bai (2024)	NR	2024	Apr 2024	none	Air Pollution	General mental health, mechanisms	The existing evidence for air pollution-related mental health loss is relatively insufficient compared to e.g. respiratory diseases. Available studies provide preliminary information for early warning biomarkers that can characterize the adverse effects of air pollution on mental health. These biomarkers are related to oxidative stress and inflammation, and epigenetic and genetic mechanisms.
Cardenas-Iniguez (2022)	NR	not available	NA	none	PM _{2.5}	General mental health, mechanisms	Emerging data suggests a link between outdoor PM and mental health. Prenatal and childhood air pollution exposure has been associated with poor neurodevelopmental and emotional outcomes, including emotional behavioural problems, impaired emotional regulation, anxiety/depression [...], psychotic experience, and increased likelihood of being prescribed psychiatric medication. At the neural level, exposure to small particles [...] during development alters dendritic spine density and neurogenesis and affects microglial cells – processes underlying synaptic proliferation and “pruning”. They are essential hallmarks of childhood and adolescent neurodevelopment. Moreover, distinct neuronal cell types have different biochemical sensitivity to oxidative stress, with key emotional regions, including the prefrontal cortex and amygdala.
Bazyar (2019)	NR	2000-2018	2018	STROBE >20 points	PM, O ₃ , SO ₂	General mental health	PM, SO ₂ , and CO increase the risk of mental disease incidence.
Bernardini (2020)	SR	1979-2020	March 2019	none	Air Pollution	General mental health	Air pollution may contribute either to worsening of psychiatric symptoms or to the decision to seek treatment for a large group of mental diseases and conditions, including severe psychiatric disorders such as schizophrenia and depression.

Author (Year)	Type of review	Publication range	End search date	Quality assessment	Pollutants studied	Outcomes studied	Results / Conclusions by study authors
Bhui (2023)	NR	2017-2021	NA	none	PM, NO ₂ , PAHs	all endpoints of interest	There is emerging evidence of associations between poor air quality and poor mental health more generally, as well as specific mental disorders. Preexisting long-term conditions appear to deteriorate, requiring more healthcare. - Similar findings exist for environmental noise. It is difficult to disentangle the effects of noise and air pollution. These and other different potential causal factors need to be considered in future studies.
Braithwaite (2019)	MA	2007-2017	1974-Sep 2017	EPHPP: RoB	PM	all endpoints of interest	Long-term PM _{2.5} exposure was significantly associated with depression risk (robust in 2/3 sensitivity analyses). Long-term PM ₁₀ exposure and depression not statistically significant associated. Results appear to support the hypothesis of an association with multiple adverse mental health outcomes, most clearly depression.
King (2022)	NR	2021-2021	last 12 months 2021	none	Air Pollution	all endpoints of interest	Depression: Within current methodological limits a small but increased risk of depressive symptoms exists among individuals more exposed to PM _{2.5} in the long-term, and evidence of a positive association with PM ₁₀ in the short term. Outcomes for other air pollutants were variably reviewed. Anxiety: inconclusive or nonsignificant findings linking exposure to air pollutants and anxiety disorders. Of interest only five studies reporting sulfur dioxide (SO ₂) data were identified. SO ₂ exposure has been linked with anxiety for many years Psychosis: limited evidence about the role of air pollutants and risk of relapse in chronic psychotic disorders has come from hospital attendance data. Suicide: Increasing short-term exposure to NO ₂ , PM _{2.5} and PM ₁₀ is associated with small but significant increased incidence of suicide. Some supporting evidence that depressive symptoms may mediate the association between air pollutants and suicidal ideation.
Radua (2024)	MA	not listed nicely	Jun 2023	AMSTAR-2	PM, NO ₂ , O ₃ , SO ₂ , CO	all endpoints of interest	Air pollution and climate change can exert mental health.
Pourhoseini (2024)	MA	2021-2022	May 2022	NOS, RoB (OHAT)	PM _{2.5} , NO ₂	Depression during/after pregnancy	Air pollutants could be associated with an increased risk of postpartum depression.

Author (Year)	Type of review	Publication range	End search date	Quality assessment	Pollutants studied	Outcomes studied	Results / Conclusions by study authors
Surace (2023)	SR	2011-2022	June 2022	Armijo-Olivo et al.	Air Pollution	Depression during/after pregnancy	Two studies showed an association between, respectively, only PM _{2.5} and both PM _{2.5} and NO ₂ exposure and postpartum depression PPD onset 12 months after childbirth, while another study found a significant association between NO ₂ exposure and PPD occurrence 6 months after childbirth. One study observed a link between stressful symptoms and exposure to PM _{2.5} , PM ₁₀ during pregnancy. Positive and preliminary findings also regard PM ₁₀ and SO ₂ , while available data are contradictory for O ₃ and CO.
Borroni (2022)	MA	2009-2021	May 2021	RoB/GRADE	PM, NO ₂ , O ₃ , SO ₂ , CO	Depression	Increase in long-term exposure to PM _{2.5} and NO ₂ , and in short-term PM ₁₀ , PM _{2.5} , NO ₂ , SO ₂ , O ₃ (although less robustly), and CO exposure is associated with an augmented risk of depression. Publication bias (half of associations) and high heterogeneity in MA results partly prevent to draw very firm conclusion
Buoli (2018)	SR	2004-2018	1982-2018	EPHPP	Air Pollution	Depression	Different air pollutants (PM and NO _x) have been associated with poor mental health; long-term PM _{2.5} has been associated with an increased risk of new onset of depressive symptoms, while increased concentration of NO _x in summer with worsening of existing depressive conditions. However, interpretation of these finding should take into account the retrospective design of most of studies, different periods of observations, confounding factors such as advanced age or medical comorbidity
Fan (2020)	MA	2009-2019	Aug 2019	NOS	PM, NO ₂ , O ₃ , SO ₃	Depression	Short-term exposure to NO ₂ , but not other air pollutants, was significantly associated with depression. Given the limitations, a larger meta-analysis incorporating future well-designed longitudinal studies will be necessary for a more definitive result.
Zeng (2019)	MA	2007-2018	Sep 2018	NOS	PM, NO ₂ , O ₃ , SO ₂ , CO	Depression	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. Results for long-term PM ₁₀ were inconclusive. No evidence was found in the association between exposure to O ₃ and depression. Some inconsistencies were found in stratified analyses. Methodological limitations included confounder control, exposure and outcome assessment.

Author (Year)	Type of review	Publication range	End search date	Quality assessment	Pollutants studied	Outcomes studied	Results / Conclusions by study authors
Zhao (2018)	SR	1960-2017	Dec 2017	OHAT	O ₃	Depression, anxiety, general mental health	Current evidence for an association between ambient ozone exposure and mental health outcomes is inconclusive and further high-quality studies are needed to assess any potential links given the strong biologic plausibility.
Zundel (2022)	SR	2011-2021	Sep 2021	none	PM, NO ₂ , O ₃	Depression, anxiety	The literature suggests that air pollution is associated with increased depressive and anxiety symptoms and behaviours, and alterations in brain regions implicated in risk of psychopathology. However, gaps include limited number of studies and a comprehensive developmental approach to examine windows of susceptibility.
Cao (2024)	MA	2015-2022	Oct 2022	NOS	PM, NO ₂ , O ₃ , SO ₂ , CO	Depression, anxiety	PM _{2.5} and NO ₂ exposure, especially long-term exposure, may be associated with the onset of depression, and no association was found for the time being between PM ₁₀ , CO, O ₃ , SO ₂ exposure and depression and PM _{2.5} exposure and anxiety disorders.
Chauhan (2025)	SR	2004-2023	Jan 2000-Dec 2023	none	PM _{2.5}	Depression, anxiety, mechanisms	Equivocal evidence was identified from pre-clinical (animal model) and human studies that PM _{2.5} exposure contributes to depression. In addition, there was substantial evidence from human studies that PM _{2.5} also was associated with anxiety.
Ragguett (2017)	SR	not listed nicely	May 2017	none	Air Pollution	Depression, anxiety, mechanisms	Available evidence suggests that exposure to harmful air quality may be associated with suicidality. In addition, those with atopic sensitivity may represent a specific subgroup that is at risk.
Xie (2023)	SR	2008-2022	June 2022	NIH	Air Pollution	Depression, anxiety, suicide, mechanisms	The results of epidemiological studies in children/youth consistently showed that air pollution increases the risk of depression and suicide-related events. In addition, neuroimaging studies revealed that exposure to air pollution is associated with the alterations in brain structure, function, and metabolism.
Trushna (2021)	MA	1988-2020	Mar 2020	JB1	PM, NO ₂	Anxiety	The meta-analyses reveal that exposure to PM ₁₀ , PM _{2.5} , and NO ₂ showed a negative [protective] association with anxiety disorder whereas exposure to PM ₁₀ increased the odds of developing psychological stress. However, scarcity of studies and methodological limitations prevalent in the published studies restrict the generation of unequivocal conclusions.

Author (Year)	Type of review	Publication range	End search date	Quality assessment	Pollutants studied	Outcomes studied	Results / Conclusions by study authors
Lu (2020)	SR	1983-2019	2019	none	Air Pollution	Anxiety, suicide	Air pollution is also associated with increased annoyance and anxiety. Psychologically, perceived air pollution can trigger existential anxiety about one's health and future. More devastatingly, air pollution is associated with increased mental disorders, such as depression, schizophrenia. In addition to self-report measures of depression, several studies have leveraged objective measures from hospitals (more emergencies on higher polluted days). Even worse, air pollution may be a risk factor for substance abuse, non-suicidal self-harm, and suicide.
Cuijpers (2023)	SR	2016-2020	Jun 2021	AMSTAR	PM, NO ₂ , SO ₂	Depression, suicide	The meta-analyses on pollution suggested that there may be a small but significant association between PM _{2.5} , PM ₁₀ , NO ₂ , SO ₂ , CO and mental health, especially depression and suicide. All associations were small and because unmeasured confounders may further reduce the strength and level of significance of the association, these findings should be considered with caution.
Liu (2021)	MA	2010-2020	Sep 2020	EPHPP	PM	Depression, suicide	The meta-analysis found that an increase in ambient PM concentration was strongly associated with an increased risk of depression and suicide, and the associations for depression appeared stronger for smaller particles (PM _{2.5}) and at a long-term time pattern.
Davoudi (2021)	MA	2010-2019	May 2020	NOS	PM, NO ₂ , O ₃ , SO ₂	Suicide	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.
Heo (2021)	MA	2010-2021	Aug 2020	OHAT	PM, NO ₂ , O ₃ , SO ₂ , CO	Suicide	RRs of suicide per IQR increase in PM _{2.5} , PM ₁₀ , and NO ₂ were 1.02 (95% CI: 1.00–1.05), 1.01 (95% CI: 1.00–1.03), and 1.03 (95% CI: 1.00–1.07). O ₃ , SO ₂ , and CO were not associated with suicide. Suicide risks associated with air pollution did not significantly differ by income level, national suicide rates, or average exposure levels. Research gaps were found for interactions between air pollution and temperature on suicide risks.

Author (Year)	Type of review	Publication range	End search date	Quality assessment	Pollutants studied	Outcomes studied	Results / Conclusions by study authors
Tota (2024)	NR	not listed nicely	Nov 2023	none	PM _{2.5} , NO ₂ , SO ₂	Depression, anxiety, schizophrenia, mechanisms	In air pollution studies, increased concentrations of PM _{2.5} , NO ₂ , and SO ₂ were the most strongly associated with the exacerbation of anxiety, schizophrenia, and depression symptoms. The pathogenesis of environmental pollution-related diseases is multifactorial, including increased oxidative stress, systematic inflammation, disruption of the blood-brain barrier, and epigenetic dysregulation.
Song (2023)	MA	2017-2022	Feb 2022	JBI, Mustafic	PM, NO ₂ , SO ₂	Schizophrenia	The current meta-analysis and systematic review suggested that short-term exposure to particulate matter (PM _{2.5} , PM _{10-2.5} , PM ₁₀) and gaseous pollutants (SO ₂ , NO ₂) may be positively associated with schizophrenia-related risk, while no significant effect was observed with CO.
Attademo (2017)	SR	1964-2016	Feb 2016	none	PM _{2.5} , metals	Schizophrenia	Among air pollutants, particulates, PM appear to play an influential role recently Also, oxides of nitrogen (NO _x), particularly NO ₂
Xu (2023)	MA	2017-2022	Dec 2022	NOS, RoB (Cochrane)	NO ₂	Schizophrenia	Evidence that NO ₂ exposure significantly increases the risk of hospital admission for schizophrenia. However, the heterogeneity was high for the summary estimates and attenuated estimates in two-pollutant models reduce the credibility of the evidence.
Thomson (2019)	NR	narrative (none)	narrative (none)	none	Air Pollution	Mechanisms	Observations of similar systemic responses to particulate and gaseous pollutants suggest that the endocrine stress response could be a common mechanism contributing to extrapulmonary effects of pollutant exposure, including impacts on the brain. Further research is warranted to investigate the relative importance of stress responses and HPA axis dysregulation in contributing to air pollutant-induced disease progression.
Herting (2024)	NR	NA	NA	none	Air Pollution	Mechanisms	The developmental inhalation toxicology literature provides strong evidence that air pollution exposure directly impacts cellular structure and function during brain development. Higher levels of air pollution during childhood and adolescence is not associated with concurrent behavioural differences, but rather predicts later onset of major depressive and other internalizing, externalizing, and thought disorder symptoms at age 18 years.

Author (Year)	Type of review	Publication range	End search date	Quality assessment	Pollutants studied	Outcomes studied	Results / Conclusions by study authors
Morrel (2024)	SR	2015-2023	Jan 2025	RoB (WHO)	Air Pollution	Mechanisms	Prenatal and childhood exposure to outdoor air pollution is associated with structural and functional brain variations. Further research is needed to clarify the effects of developmental timing, along with the downstream implications of outdoor air pollution exposure on children's cognitive and mental health.

Abbreviations:

NR = Narrative review, SR = Scoping review, MA = Meta-analysis.

NA = Not available.

5.1.2 Biological mechanism

The precise mechanisms by which air pollution affects mental health are not fully understood. However, various pathways and modes of action have been described, that shed some light on possible causal pathways.

How air pollution reaches the brain

The brain is exposed to air pollution by different ways. Firstly, gaseous pollutants enter the blood stream in the lungs and pass the blood-brain barrier. Secondly, particles can enter directly via the olfactory system. Nasal inhalation allows the smaller particles to contact with olfactory receptors or trigeminal nerve, by which air pollutants are transported to the brain. The third way is via inflammatory processes in the lungs. The air pollutants that reach the alveolars during breathing, provoke inflammation in lung tissue. As a consequence, particles are transported to the systemic circulation and subsequently pass through the blood-brain barrier. Finally, chronic respiratory and systemic inflammation affects the brain indirectly and induces neuroinflammation and an increased production of reactive oxygen species, that can lead to damage in the brain (Tota et al., 2024). Neuroinflammation and oxidative stress are considered the most important mechanisms affecting the brain.

Brain development and disruption of various processes by air pollution

Ongoing brain development may render children and adolescents especially vulnerable to neurotoxic effects of air pollution. Hertig et al. (2024) describe detailed the brain development as a prolonged process that begins in utero and continues into young adulthood (Figure 5.4). This ongoing cellular and system-level changes during childhood and adolescence, possibly make the developing brain more sensitive to pollutants' neurotoxic effects, as a function of both timing and duration, with relevance to cognition and mental health. In early life, there is an increase in network segregation followed by a period of integration that persists through adolescence, as network refinement takes place (Morrel et al., 2025). Hertig et al. (2024) conclude that air pollution exposure from conception through adolescence could disrupt microglia driven synaptogenesis and later synaptic pruning functions, which, depending on the timing, could lead to inefficient and/or aberrant neural circuitry for sensorimotor, language, or higher order cognitive brain circuits. Further impacts on both myelination and oligodendrocyte precursor cell differentiation may disrupt white matter development, maintenance, and repair during childhood and adolescence, additionally compromising the development of efficient neural circuitry. Damage from prenatal air pollution exposure could impact blood-brain barrier development in utero, while blood-brain barrier damage from postnatal exposure may include astrocyte disruption and altered blood-brain barrier permeability (Herting et al., 2024). Additionally, distinct neuronal cell types have different biochemical sensitivity to oxidative stress, with key emotional regions, including the prefrontal cortex and amygdala, purported to be most susceptible (Cardenas-Iniguez et al., 2022).

Figure 5.4 Sensitive periods in human brain development during childhood and adolescents

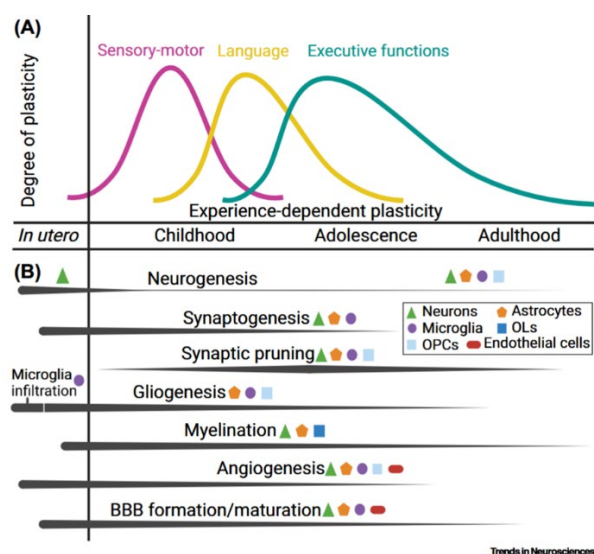


Figure 2. Sensitive periods in human brain development during childhood and adolescence. (A) Neuroplasticity of different functional systems in humans, with approximate timelines adapted from [141]. Sensory-motor development peaks in

Source: Figure 2 from (Herting et al., 2024).

Alteration of brain structures, connectivity, functionality in association with air pollution

Brain imaging studies have shown that outdoor air pollution exposure during critical windows of brain development such as pregnancy, childhood, and early adolescence has been associated with widespread structural and functional brain outcomes. However, results are not entirely consistent across studies. Brain regions most identified by Structural Magnetic Resonance Imaging to be affected by air pollution in the literature include the cerebellum, corpus callosum, and caudate. Significant associations between air pollution and Diffusion Magnetic Resonance Imaging outcomes were detected in all studies indicating that the microarchitecture of the developing brain might be particularly susceptible to damage from PM_{2.5} and its components. Furthermore, most studies looking into functional connectivity with functional MRI found that air pollution exposure was associated with greater (i.e., in cross-sectional studies) or increasing (i.e., in longitudinal studies) functional connectivity and initial studies suggest air pollution may influence both cerebral blood flow and metabolite levels during development (Morrel et al., 2025; Zundel et al., 2022).

Changes to neurotransmitters, neuromodulators and their metabolites in association with air pollution and mental health

Changes to neurotransmitters, neuromodulators and their metabolites have also been described in association with air pollution and mental health outcomes (Bhui et al., 2023).

One such early biological response triggered by exposure to air pollutants is a stress response that includes activation of the hypothalamic-pituitary-adrenal (HPA) axis and release of stress hormones. Dysregulation of the HPA axis is a feature of many disease processes common to both chronic stress and long-term exposure to air pollution, including cardiovascular disease, metabolic diseases such as type 2 diabetes, cognitive disorders, and depression (Thomson, 2019).

Particulate matter and ozone have been found to alter the expression of glucocorticoid-regulated genes in the brain of exposed rats resulting in increased concentrations of glucocorticoids impairing regulation of hippocampal neurogenesis, thought to be a key cause of morphological and functional deficits observed in depression. Because the hippocampus contributes to regulation of the HPA axis, this effect can contribute to HPA axis dysregulation (Thomson, 2019).

Epigenetic changes by hyper-and hypomethylation of genes regulating neurotransmitters and hormones have been observed in association with air pollution. Such disbalance include changes in the dopamine, serotonin, glutamate levels; all associated with mental health and air pollution (Zundel et al., 2022). Modification brain-derived neurotrophic factor (BDNF) methylation has been associated with different mental disorders. BDNF is a neurotrophic factor crucial for neuronal survival, development, and synaptic plasticity (Bai et al., 2024b). Dopaminergic or glutaminergic toxicity, increased glucocorticoid activity, increased microglial activation, and reduced neurogenesis are processes leading to disturbance in neurotransmitters (Tota et al., 2024).

Overall, air pollution can reach and affect the brain by various pathways. Air pollution can thereby have immediate effects following exposure, and/or lasting effects on developmental process(es) that can persist into adulthood or even follow a protracted onset (Herting et al., 2024). The detrimental effect of air pollutants on mental health is multifactorial (Tota et al., 2024) and various processes affecting brain development, brain structures, connectivity or functionality and changes to neurotransmitters or epigenetic changes to regulation of such transmitters have been described in association with air pollution.

5.1.3 Depression

Long-term associations

All systematic or narrative reviews looking into effects of air pollution on depression or depressive symptoms agree that poor air quality is associated with prevalence or new onset of depression or exacerbation of depressive symptoms (Bhui et al., 2023; Buoli et al., 2018; Chauhan et al., 2025; Cuijpers et al., 2023; King et al., 2022; Ragguett et al., 2017; Tota et al., 2024; Xie et al., 2023; Zhao et al., 2018; Zundel et al., 2022). The exacerbation of depressive mood might even lead to suicidality (Ragguett et al., 2017). The characteristics of the identified meta-analyses are summarised in Table 5.2.

Table 5.2 Results of meta-analyses of the association of depression with long-term air pollution

Author (year)	End of search	Duration	Pollutant	Increment	# of European studies	# of studies or EE	Result	I ²
Liu (2021)	Sep 2020	LT	PM _{2.5}	10 µg/m ³	6	15	OR = 1.18, (1.05–1.32)	46%
Zeng (2019)	Sep 2018	LT	PM _{2.5}	5 µg/m ³	2	10	OR = 1.06, (1.00–1.13)	41.40%
Borroni (2022)	May 2021	LT	PM _{2.5}	10 µg/m ³	7	16	RR = 1.074, (1.021–1.129)	19.80%
Cao (2024)	Oct 2022	LT	PM _{2.5}	5 µg/m ³	3	19	RR = 1.12, (1.03–1.21)	86%
Fan (2020)	Aug 2019	LT	PM _{2.5}	10 µg/m ³	5	12	<i>OR = 1.12, (0.97–1.29)</i>	51.60%
Braithwaite (2019)	1974-Sep 2017	LT	PM _{2.5}	10 µg/m ³	1	5	OR = 1.1, (1.02–1.19)	0.00%
Radua (2024)	Jun 2023	LT	PM _{2.5}	1 µg/m ³	NA	n = 25066	RR = 1.007, (1.002–1.012)	56%
Radua (2024)	Jun 2023	LT	PM _{2.5}	1 µg/m ³	NA	n = 5512	OR = 1.01, (1.004–1.016)	52%
Radua (2024)	Jun 2023	LT	PM _{2.5}	1 µg/m ³	AN	n = 6650	OR = 1.01, (1.004–1.017)	0%
Liu (2021)	Sep 2020	LT	PM ₁₀	10 µg/m ³	5	11	<i>OR = 1.08, (0.98–1.19)</i>	67.20%
Zeng (2019)	Sep 2018	LT	PM ₁₀	10 µg/m ³	2	8	<i>OR = 1.04, (0.85–1.26)</i>	75.20%
Borroni (2022)	May 2021	LT	PM ₁₀	10 µg/m ³	8	17	<i>RR = 1.092, (0.988–1.206)</i>	96.90%
Cao (2024)	Oct 2022	LT	PM ₁₀	10 µg/m ³	2	13	<i>RR = 1.06, (0.83–1.35)</i>	91%
Fan (2020)	Aug 2019	LT	PM ₁₀	10 µg/m ³	4	8	<i>OR = 1.04, (0.88–1.25)</i>	85.70%
Braithwaite (2019)	1974-Sep 2017	LT	PM ₁₀	10 µg/m ³	1	3	<i>OR = 0.89, (0.5–1.58)</i>	0.00%
Zeng (2019)	Sep 2018	LT	NO ₂	5 ppb	2	8	<i>OR = 1.0, 2 (0.96–1.09)</i>	69.90%
Borroni (2022)	May 2021	LT	NO ₂	10 µg/m ³	7	14	RR = 1.037, (1.011–1.064)	62.70%
Cao (2024)	Oct 2022	LT	NO ₂	10 µg/m ³	3	15	RR = 1.17, (1.04–1.32)	87%
Fan (2020)	Aug 2019	LT	NO ₂	10 µg/m ³	5	7	<i>OR = 1.05, (0.83–1.34)</i>	83.60%
Radua (2024)	Jun 2023	LT	NO ₂	1 µg/m ³	NA	n = 40199	RR = 1.004, (1.001–1.006)	74%
Borroni (2022)	May 2021	LT	SO ₂	10 µg/m ³	0	3	<u><i>RR = 0.917, (0.847–0.992)</i></u>	84.30%
Cao (2024)	Oct 2022	LT	SO ₂	1 ppb	0	4	<i>RR = 1.09, (0.97–1.24)</i>	65%
Borroni (2022)	May 2021	LT	O ₃	10 µg/m ³	2	4	<i>RR = 0.965, (0.896–1.039)</i>	89.10%
Cao (2024)	Oct 2022	LT	O ₃	5 ppb	1	9	<i>RR = 1.05, (0.98–1.12)</i>	87%
Borroni (2022)	May 2021	LT	CO	1 µg/m ³	0	3	RR = 1.143, (1.034–1.263)	0.00%

Author (year)	End of search	Duration	Pollutant	Increment	# of European studies	# of studies or EE	Result	I ²
Cao (2024)	Oct 2022	LT	CO	0.1 ppm	0	3	RR = 1.32, (0.73–2.40)	97%
Radua (2024)	Jun 2023	LT	CO	1 µg/m ³	NA	n = 28697	RR = 1.144, (1.035–1.264)	0%

Abbreviations/Symbols:

LT = long-term exposure, NA = no data available, EE = effect estimates, # = number, n = number, RR = Relative Risk, OR = Odds Ratio.

bold = statistically significant results; italic = non-statistically elevated risks/results; underlined = statistically significant negative risk/result indicative of a protective effect.

Most solid evidence seems to exist for particulate matter (Buoli et al., 2018; Chauhan et al., 2025; Cuijpers et al., 2023; King et al., 2022). Looking into results from the compiled meta-analyses, all analyses except one (Fan et al., 2020) find significantly increased risks for depression diagnosis or symptoms with increased long-term PM_{2.5} with risk estimates ranging from 1.074 to 1.18 per 10 µg/m³ (Borroni et al., 2022; Braithwaite et al., 2019; Cao et al., 2024; Fan et al., 2020; Liu et al., 2021; Radua et al., 2024; Zeng et al., 2019). Some reviews indicated the presence of publication bias. However, in Borroni et al. (2022) the corrected effect estimates after trim and fill still indicated a small but significantly increased risk with PM_{2.5}. The latest and most comprehensive umbrella review calculating new effect estimates from meta-analyses by Borroni et al. (2022), Zeng et al. (2019) and a review on indoor air pollution found significantly increased risks, e.g. a 0.7% (95% CI: 0.2–1.3%) increased risk of depression diagnosis per 1 µg/m³ increased long-term PM_{2.5}. However, the credibility of the evidence was classified only as suggestive, since the combination of the number of cases, p-value, prediction interval, heterogeneity, evidence of small-study effects or excess of significance bias was not sufficient for a higher rating by the authors.

European studies also find quite consistent increases in depression incidence in children/adolescents (Latham et al., 2021; Roberts et al., 2019; Newbury et al., 2024b) (all studies in the UK) and adults in association with elevated PM_{2.5} exposure. In the prevalence analyses more null or non-significantly elevated results with PM_{2.5} can be found. However, most effect estimates are still significantly elevated. Study characteristics of the original European studies can be found in Table 5.3.

Table 5.3 Original studies from Europe studying incidence of depression or symptoms in association with air pollution

Author (year)	Country	Study design	Study name	Population	# participants	Depression outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Incidence																
Latham (2021)	UK	CH	E-Risk Twin	Adolescents	1879	Depression	(+)	0		0	+					not
Roberts (2019)	UK	CH	E-Risk Twin	Adolescents	284	Depression	+			+						not
Newbury (2024)	UK	CH	ALSPAC	Young adults	9065	Depression	+			(+)					noise 0	not
Nobile (2023)	IT	CH	RoLS	Adults	1733331	Depression	+		+	+					UFP +, noise +	x
Wu (2023)	SE	CH	SNAC-K	Adults	2812	Depression	+	(+)			+					(x) =
Jin (2024)	UK	CH	UK Biobank	Adults	257534	Depression							+			not
Pan (2024)	UK	CH	UK Biobank	Adults	170369	Depression	+			+					PM _{10-2.5} 0, NO +	x
Yang (2023)	UK	CH	UK Biobank	Adults	389185	Depression	+			+					PM _{10-2.5} 0, NO +	not
Yao (2025)	UK	CH	UK Biobank	Adults	252376	Depression	+	0	0	+	+				PM _{10-2.5} 0	not
Yuan (2025)	UK	CH	UK Biobank	Adults	245820	Depression						+			noise +	x
Li (2023)	UK	CH	UK Biobank	Adults	354897	Major depressive disorder	+	0		0	+					not
Jin (2024)	UK	CH	UK Biobank	Adults	257534	Depression or anxiety							+			not
Gao (2023)	UK	CH, CS	UK Biobank	Adults	345876	Depression	+	+		+	+				PM _{10-2.5} 0	not
Gao (2023)	UK	CH, CS	UK Biobank	Adults	345876	Depression or anxiety	+	+		+	+				PM _{10-2.5} 0	not

Author (year)	Country	Study design	Study name	Population	# participants	Depression outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Roberts (2019)	UK	CH	E-Risk Twin	Adolescents	284	Symptoms CDI	(+)			(+)						not
Brehm 2024	DE	CS	x	Adolescents, adults	2029359	Depression									LEZ - PM NO ₂ reduced	not
Motoc 2025	NL	CH	RS-I	Adults	840-17100	Symptoms CESD	+		0	0					NDVI 0	not
Motoc 2025	NL	CH	RS-II	Adults	840-17100	Symptoms CESD	0		0	0					NDVI 0	not
Motoc 2025	NL	CH	DFBC	Adults	840-17100	Symptoms HADSD	0		0	0					NDVI -	not
Motoc 2025	NL	CH	ALSPAC	Adults	840-17100	Symptoms EPSD	+		0	0					NDVI 0	not

Abbreviations/Symbols:

UK = United Kingdom; IT = Italy; DE =Germany; SE = Sweden; NL = the Netherlands.

CH = cohort study; CS = cross-sectional study.

E-Risk Twin = Environmental Risk Longitudinal Twin Study; ALSPAC = The Avon Longitudinal Study of Children and Parents; RoLS = Rome longitudinal study; SNAC-K = Swedish National study on Aging and Care in Kungsholmen; UK Biobank (italic) = UK Biobank Research Analysis Platform; RS-I/II = Rotterdam study I/II; ALSPAC = The Avon Longitudinal Study of Children and Parents; DFBC = Dutch Famine Birth Cohort.

CDI = Children's Depression Inventory; CES-D = Center for Epidemiologic Studies Depression Scale; EPSD = Edinburgh Postnatal Depression Scale; HADSD =Hospital Anxiety and Depression Scale for depression.

+ = significant adverse association; (+) = non-significant elevated risk = when $p > 0.05$ and < 0.10 ; 0 = no association / effect; (-) = non-significant reduced risk; - = significant protective association.

LEZ = low emission zone; NDVI = Normalized Difference Vegetation Index.

Noise as confounder: + = included in analysis; (+) = included in sensitivity analysis/multi-pollutant models with similar results; not = not measured/included in analysis.

Risk estimates for an association of depression or depressive symptoms with PM₁₀ were in two (Borroni et al., 2022; Liu et al., 2021) of the six meta-analyses increased though not significantly. Radua et al. (2024) also considered the association to be suggestive. European studies find mixed results for PM₁₀ with two analyses of UK Biobank data with null findings regarding depression incidence (Yao et al., 2025; Li et al., 2023a) and one with significantly elevated risks (Gao et al., 2023) and a Swedish study with non-significantly elevated risks (Wu et al., 2023a). Results of analyses of prevalence data are inconsistent as well: Altuğ et al. (2020) found significantly elevated depressive symptom scores but not depression prevalence with PM₁₀ in an elderly women's cohort in Germany, significantly increased prevalence risks in Spanish (Vert et al., 2017) and Belgian (Pelgrims et al., 2021) adult cohort studies and a German insurance claims analysis (Zhao et al., 2019), mixed results in the UK Biobank data (Fan et al., 2024; Ma et al., 2024; Wu et al., 2022) and null associations in another German cross-sectional study (Petrovski et al., 2021), a UK adolescent study (Thompson et al., 2024), and the analysis of the European cohorts FINRISK, KORA with additionally significantly reduced risks in the HUNT cohort and non-significantly elevated risks in the LifeLines cohort.

Even though three of the five meta-analyses with NO₂ find significantly elevated risks of NO₂ with depression, Radua et al. (2024) considered the evidence to be weak to date. European studies show mixed evidence for incidence of depression with long-term exposure to NO₂. The analysis of the UK E-Risk Twin study using chemical transport models find null associations (Latham et al., 2021) in adolescents, whereas the earlier analysis studying only 284 twins of that sample find significantly elevated risks for depression and a borderline association with depressive symptoms (Roberts et al., 2019). Another UK adolescent study also finds null associations (Newbury et al., 2024a). Studies in adults find null associations in a Dutch analysis of four cohorts (Motoc et al., 2025), positive and null associations in the UK Biobank analyses (Gao et al., 2023; Pan et al., 2024; Yang et al., 2023a; Yao et al., 2025; Li et al., 2023b) and a significantly increased risk with NO₂ in the Rome longitudinal study (Nobile et al., 2023). Out of 5 cohorts being analysed for depression prevalence, the majority showed significantly elevated risks with NO₂ (SALIA, ALFA, CONSTANCES, BHIS – depending on the definition, and UK Biobank depending on the analysis). The score for depressive symptoms was increased in SALIA, CONSTANCES and LifeLines, but not in the adolescent study SCAMP, KORA, FINRISK and mixed findings in the UK Biobank study (Fan et al., 2024; Meng et al., 2024). Notably, exposure to NO_x was associated with an increase of depression incidence and prevalence in all European studies (except the UK biobank data).

The number of studies included in meta-analyses of other pollutants was rather low, with no evidence from European long-term studies for SO₂ or CO exposure. Except for one UK biobank analysis with significantly elevated risk for depression incidence with SO₂ (Yuan et al., 2025), none of the other European studies report on results with SO₂ or CO.

The meta-analysis by Cao et al. (2024) including 9 studies and one study from Europe found only non-significantly elevated risks with depression and long-term exposure to elevated ozone levels. Increased risks were reported with UK biobank data (Jin et al., 2024), null associations in adolescent data of SCAMP (Thompson et al., 2024) and GINIPlus and LISA (Zhao et al., 2019) and increased prevalence risk in the administrative AOK PLUS cohort (Zhao et al., 2019). See Table 5.4 for the extracted study characteristics from original European studies on depression prevalence.

Table 5.4 Original studies from Europe studying prevalence of depression or symptoms in association with air pollution

Author (year)	Country	Study design	Study name	Population	# participants	Depression outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Prevalence																
Pelgrims (2021)	BE	CS	BHIS	Adolescents, adults	1325	Depression	(+)	+	+	(+)			-		NDVI 0, noise (+), street 0	x
Altuğ (2020)	DE	CH	SALIA	Adults/Women	821	Depression	+	(+)	+	+	(+)				PM _{10-2.5} +, Traffic +	(x) =
Hao (2022)	UK	CS	UK Biobank	Adults	334986	Broad Depression NATD	+								noise +	not
Allaouat (2021)	FI	CS	x	Adults	5895	Depressive Disorder	(+)								Traffic PM _{2.5}	x
Allaouat (2021)	FI	CS	x	Adults	5895	Depressive Disorder	0								Wood	x
Hao (2022)	UK	CS	UK Biobank	Adults	334986	Major depressive disorder	+								noise 0	not
Ma (2024)	UK	CS	UK Biobank	Adults	NA	Major depressive disorder	+	(+)		+	+				PM _{10-2.5} 0	not
Wu (2022)	UK	CS	UK Biobank	Adults	39168	Major depressive disorder	+	0								not
Petrowski (2021)	DE	CS	USUMA	Adults	3020	Depression PHQ-2		0								not
Meng (2024)	UK	CS	UK Biobank	Adults high genetic risk vs low	104385	Depression PHQ-9	+			+					NO +, PM _{10-2.5} +	not
Generaal (2019)	NL	CS	Dutch Cohorts	Adults	32487	Depression			+						noise (+), NDVI -	x

Author (year)	Country	Study design	Study name	Population	# participants	Depression outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Lyons (2024)	IE	CS	TILDA	Adults	3407	Depression	(+)									not
Fan (2024)	UK	CS	UK Biobank	Adults	42000-45000	Depression	+	(+)		0	0				PM _{10-2.5} 0	not
Vert (2017)	ES	CS	ALFA	Adults	958	Depression	+	+	+	+	+				PM _{10-2.5} 0	not
Zare Sakhvidi (2022)	FR	CS	CONSTANCES	Adults	123754	Depression CES-D	+		+	+						not
Hautekiet (2022)	BE	CS	BHIS	Adolescents, adults	16455	Depression SCL-90-R	+		(+)	0						not
Bai (2024)	Europe	CS	UK Biobank IEU OPEN GWAS	Adults	NA	Depression, causal	+	0		0	0				PM _{10-2.5} 0	not
Altuğ (2020)	DE	CH	SALIA	Adults/Women	821	Symptoms CESD-R %	+	+	(+)	+	+				PM _{10-2.5} (+), Prox Road +	(x) =
Thompson (2024)	UK	CH	SCAMP	Adolescents	7555	Symptoms PHQ-9	0	0		0			0		noise 0	x
Zhao (2019)	DE	CS	GINIplus, LISA	Adolescents	2827	LT Symptoms							0			not
Lyons (2024)	IR	CS	TILDA	Adults	3407	Symptoms	+									not
Zare Sakhvidi (2022)	FR	CS	CONSTANCES	Adults	123754	Symptoms CESD	+		+	+						not
Zijlema (2016)	Europe	CS	FINRISK	Adults	70928	Symptoms CESD	0	0	0	0						x
Zijlema (2016)	Europe	CS	HUNT	Adults	70928	Symptoms HADSD		-		-						x

Author (year)	Country	Study design	Study name	Population	# participants	Depression outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Zijlema (2016)	Europe	CS	LifeLines	Adults	70928	Symptoms MINI	0	(+)	0	(+)						x
Meng (2024)	UK	CS	<i>UK Biobank</i>	<i>Adults high genetic risk vs low</i>	104385	<i>Symptoms PHQ-9</i>	+			+					<i>NO +, PM_{10-2.5} +</i>	<i>not</i>
Zijlema (2016)	Europe	CS	KORA	Adults	70928	Symptoms PHQ-9	0	0	0	0						x
Generaal (2019)	NL	CS	Dutch Cohorts	Adults	32487	Symptoms/Severity			0						in 1/8 cohorts significant, noise 0, NDVI 0	x
Brehm (2024)	DE	CS	x	Adolescents, adults	2029359	Severity (Medication) Depression Anxiety									LEZ - PM NO ₂ reduced	not

Abbreviations/Symbols:

BE = Belgium; DE = Germany; UK = United Kingdom; ES = Spain; IR = Ireland; NL = the Netherlands; FR = France.

CH = cohort study; CS = cross-sectional study.

BHIS = Belgian Health Interview Surveys; SALIA = Study on the influence of Air pollution on Lung function, Inflammation and Aging; UK Biobank (italic) = UK Biobank Research Analysis Platform; USUMA = Random, representative sample of German population by USUMA, an independent service for surveys, methods, and analyses; TILDA = The Irish Longitudinal Study on Ageing; ALFA = Early Identification of Markers in Alzheimer's Families; CONSTANCES = CONSULTANTS des Centres d'Examens de Santé; SCAMP = Study of Cognition, Adolescents and Mobile Phones; GINIplus, LISA = German Infant Nutritional Intervention plus environmental and genetic influences on allergy development, Influence of Life-style factors on the development of the Immune System and Allergies in East and West Germany; FINRISK = FINRISK = National FINnish MONitoring of Risk factors for cardiovascular disease and diabetes; HUNT = Helseundersøkelsen i Nord-Trøndelag: Nord-Trøndelag Health Study, LifeLines = Lifelines prospective cohort study and biobank from the northern population of the Netherlands; KORA = Kooperative Gesundheitsforschung in der Region Augsburg.

NATD = symptoms of nerves, anxiety, tension, or depression; PHQ -2/9 = Patient Health Questionnaire; CES-D = Center for Epidemiologic Studies Depression Scale; HADSD = Hospital Anxiety and Depression Scale for depression; MINI = Mini-International Neuropsychiatric Interview.

+ = significant adverse association; (+) = non-significant elevated risk = when $p > 0.05$ and < 0.10 ; 0 = no association / effect; (-) = non-significant reduced risk; - = significant protective association.

LEZ = low emission zone; NDVI = Normalized Difference Vegetation Index.

Noise as confounder: + = included in analysis; (+) = included in sensitivity analysis/multi-pollutant models with similar results; not = not measured/included in analysis.

Three studies have included noise as a confounder in their models and find independent effects for the studied pollutants PM_{2.5}, NO₂, SO₂ and EC (Pan et al., 2024; Yuan et al., 2025; Wu et al., 2023b). One study found consistent effects for PM_{2.5} and NO₂ in the multi-pollutant analyses with noise (Wu et al., 2023a).

Short-term associations

Worsening of depressive symptoms or emergency or hospital admissions due to depressive symptoms or diagnosis seem to be consistently associated with short-term elevated exposures to PM₁₀, PM_{2.5}, SO₂ and CO. However, these associations are mostly based on non-European studies (Table 5.5).

European studies overall do not find associations of short-term elevated exposures to PM_{2.5} or PM₁₀ with severity of depressive symptoms or daily emergencies due to depression (and anxiety) (Borroni et al., 2024; Muhsin et al., 2022; Pignon et al., 2022), except for a times-series in France, that found higher emergencies due to depression or anxiety with PM₁₀ but not PM_{2.5} (Pignon et al., 2022). In the analysis of the German GINIplus and LISA adolescent data, there also was no association with increased depressive symptoms with ozone exposure two weeks before the assessment (Zhao et al., 2019). Finally, an Italian study found mixed and contradicting results with NO₂ depending on the instrument that measured severity of depression (Borroni et al., 2024) (Table 5.6).

Table 5.5 Results of meta-analyses of the association of depression with short-term air pollution

Author (year)	End of search	Duration	Pollutant	Increment	# of European studies	# of studies or EE	Result	I ²
Zeng (2019)	Sep 2018	ST	PM _{2.5}	5 µg/m ³	0	4	OR = 1.01, (0.99–1.03)	69.60%
Borroni (2022)	May 2021	ST	PM _{2.5}	10 µg/m ³	0	19	RR = 1.009, (1.007–1.011)	0.02%
Fan (2020)	Aug 2019	ST	PM _{2.5}	10 µg/m ³	0	6	<i>OR = 1.01, (0.99–1.04)</i>	51.60%
Liu (2021)	Sep 2020	ST	PM _{2.5}	10 µg/m ³	0	9	OR = 1.02, (1.01–1.03)	61.90%
Radua (2024)	Jun 2023	ST	PM _{2.5}	1 µg/m ³	NA	n = 351094	RR = 1.002, (1.001–1.004)	59%
Zeng (2019)	Sep 2018	ST	PM ₁₀	10 µg/m ³	0	4	OR = 1.03, (1.01–1.05)	88.50%
Borroni (2022)	May 2021	ST	PM ₁₀	10 µg/m ³	0	16	RR = 1.009, (1.006–1.012)	79.10%
Fan (2020)	Aug 2019	ST	PM ₁₀	10 µg/m ³	0	5	<i>OR = 1.01, (0.98–1.04)</i>	86.70%
Liu (2021)	Sep 2020	ST	PM ₁₀	10 µg/m ³	0	5	OR = 1.02, (1.01–1.03)	88.20%
Radua (2024)	Jun 2023	ST	PM ₁₀	1 µg/m ³	NA	n = 216449	RR = 1.001, (1.001–1.002)	77%
Radua (2024)	Jun 2023	ST	PM ₁₀	1 µg/m ³	NA	n = 51924	OR = 1.003, (1.001–1.005)	84%
Zeng (2019)	Sep 2018	ST	NO ₂	5 ppb	0	4	OR = 1.04, (1.01–1.07)	52.00%
Borroni (2022)	May 2021	ST	NO ₂	10 µg/m ³	0	19	RR = 1.022, (1.012–1.033)	87.80%
Fan (2020)	Aug 2019	ST	NO ₂	10 µg/m ³	0	7	OR = 1.02, (1.00–1.04)	65.40%
Radua (2024)	Jun 2023	ST	NO ₂	1 µg/m ³	NA	n = 307618	RR = 1.002, (1.001–1.003)	84%
Radua (2024)	Jun 2023	ST	NO ₂	1 µg/m ³	NA	n = 32522	OR = 1.003, (1.001–1.006)	57%
Borroni (2022)	May 2021	ST	SO ₂	10 µg/m ³	0	18	RR = 1.024, (1.010–1.037)	76.30%
Fan (2020)	Aug 2019	ST	SO ₂	10 µg/m ³	0	6	<i>OR = 1.03, (0.99–1.07)</i>	71.20%
Radua (2024)	Jun 2023	ST	SO ₂	1 µg/m ³	NA	n = 105887	RR = 1.002, (1.001–1.003)	69%
Zeng (2019)	Sep 2018	ST	O ₃	5 ppb	0	6	OR = 1.01, (0.99–1.03)	81.30%
Borroni (2022)	May 2021	ST	O ₃	10 µg/m ³	0	17	<i>RR = 1.011, (0.997–1.026)</i>	97.20%
Fan (2020)	Aug 2019	ST	O ₃	10 µg/m ³	1	7	<i>OR = 1.01, (0.99–1.03)</i>	82.20%
Zeng (2019)	Sep 2018	ST	CO	0.1 ppm	0	3	OR = 1.01, (1.00–1.01)	70.20%
Borroni (2022)	May 2021	ST	CO	1 µg/m ³	0	12	RR = 1.062, (1.020–1.105)	69.90%
Radua (2024)	Jun 2023	ST	CO	1 µg/m ³	NA	n = 166021	RR = 1.06, (1.014–1.108)	67%

Abbreviations/Symbols: ST = short-term exposure; NA = no data available; EE = effect estimates; # = umber; n = number; RR = relative risk; OR = odds ratio.

bold = statistically significant results; italic = non-statistically elevated risks/results; underlined = statistically significant negative risk/result indicative of a protective effect.

Table 5.6 Original studies from Europe studying depression or symptoms in association with short-term air pollution

Author (year)	Country	Study design	Study name	Population	# participants	Depression outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other Pollutants	Noise as confounder
Short-term associations depression																
Zhao (2020)	DE	CS	AOK PLUS	Adolescents, adults	1.13 Million	ST Depression diagnosis		+					+			not
Zhao (2019)	DE	CS	GINIplus, LISA	Adolescents	2827	ST Symptoms							0			not
Borroni (2024)	IT	CS	Policlinico	Adult Patients	416	ST Severity CGI	0	0		0					PM _{2.5} effect in hypersusceptible (chronically ill) subjects	not
Borroni (2024)	IT	CS	Policlinico	Adult Patients	416	ST Severity GAF	0	0		- daily activities					0	not
Borroni (2024)	IT	CS	Policlinico	Adult Patients	416	ST Severity HAMD	0	0		+					PM _{2.5} effect in hypersusceptible (chronically ill) subjects	not
Borroni (2024)	IT	CS	Policlinico	Adult Patients	416	ST Severity MADRS	0	0		0					PM _{2.5} effect in hypersusceptible (chronically ill) subjects	not
Borroni (2024)	IT	CS	Policlinico	Adult Patients	416	ST Severity SDS perceived support	0	0		-					0	not

Author (year)	Country	Study design	Study name	Population	# participants	Depression outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other Pollutants	Noise as confounder
Pignon (2022)	FR	TS	x	Emergencies	20727	Daily Emergencies	0	+								not
Muhsin (2022)	SE	TS	x	Adults	81548	Daily emergencies Depression or anxiety	0	0								not

Abbreviations/Symbols:

DE = Germany; IT = Italy; FR = France; SE = Sweden.

CS = cross-sectional study; TS = time series.

AOK PLUS = Study with administrative health records of health insurer AOK; GINIplus, LISA = German Infant Nutritional Intervention plus environmental and genetic influences on allergy development, Influence of Life-style factors on the development of the Immune System and Allergies in East and West Germany; Policlinico = Persons with diagnosis of major depressive disorder at the Psychiatry Unit of the Policlinico Hospital in Milan; x = no study name.

CGI = Clinical Global Impression-severity of illness; GAF = Global Assessment of Functioning; HAMD = Hamilton Depression Rating Scale; MADRS = Montgomery–Asberg Depression Rating Scale; SDS = Sheehan Disability Scale.

+ = significant adverse association; (+) = non-significant elevated risk = when $p > 0.05$ and < 0.10 ; 0 = no association / effect; (-) = non-significant reduced risk; - = significant protective association.

LEZ = low emission zone; NDVI = Normalized Difference Vegetation Index.

Noise as confounder: + = included in analysis; (+) = included in sensitivity analysis/multi-pollutant models with similar results; not = not measured/included in analysis.

Postpartum depression

The evidence for an association of postpartum depression with exposure to PM₁₀ during the second trimester of the pregnancy was deemed to be highly suggestive by Radua et al. (2024). Associations with other pollutants were less certain (Figure 5.7).

This seems also to be reflected in the analysis of over 30'000 pregnant women in the Netherlands, where postpartum depression was borderline associated with PM₁₀ exposure but not PM_{2.5} or NO₂ (Cadman et al., 2024) (Figure 5.8).

Overall, even though some reviews are cautious of causally attributing air pollution to depression or depressive symptoms due to unmeasured confounding, e.g. by co-occurring noise exposure (Bhui et al., 2023; Buoli et al., 2018; Cuijpers et al., 2023), others see supporting evidence from animal models and studies that relate brain alterations or brain function and metabolism to such behaviour (Chauhan et al., 2025; Xie et al., 2023). In the few European studies considering noise in their analyses, air pollution effects seemed independent. However, research gaps include reliable long-term studies with rigorous outcome and exposure assessments, taking a developmental approach to examine windows of susceptibility (Zundel et al., 2022).

Table 5.7 Results of meta-analyses of the association of postpartum depression with air pollution exposure during pregnancy

Author (year)	End of search	Duration	Pollutant	Increment	# of European studies	# of studies or EE	Result	I ²
Pourhoseini (2024)	May 2022	Whole pregnancy	PM _{2.5}	10 µg/m ³	0	3	OR = 1.03, (0.95–1.11)	9.65%
Radua (2024)	Jun 2023	Pregnancy: 2nd trimester	PM ₁₀	1 µg/m ³	NA	n = 2211	OR = 1.023, (1.014–1.033)	36%
Pourhoseini (2024)	May 2022	Whole pregnancy	NO ₂	10 µg/m ³	0	3	OR = 1.27, (0.93–1.72)	96.1%
Radua (2024)	Jun 2023	Pregnancy: 2nd trimester	NO ₂	1 µg/m ³	NA	n = 5859	OR = 1.017, (1.00–1.034)	93%
Radua (2024)	Jun 2023	Pregnancy: 2nd trimester	O ₃	1 µg/m ³	NA	n = 2211	<u>OR = 0.985, (0.972–0.999)</u>	75%

Abbreviations/Symbols:

NA = no data available; EE = effect estimates; # = number; n = number; RR = relative risk; OR = odds ratio.

Table 5.8 Original study from Europe studying postpartum depression in association with air pollution

Author (year)	Country	Study design	Study name	Population	# participants	Depression outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	O	Other pollutants	Noise as confounder
Cadman (2024)	NL	CH	ESCAPE/ELAPSE	Pregnant women	30772	Postpartum depression	0	(+)		0					noise +, NDVI 0, walkability, built environment	x

Abbreviations/Symbols:

NL = the Netherlands; CH = cohort study; ESCAPE/ELAPSE = European Study of Cohorts for Air Pollution Effects; NDVI = Normalized Difference Vegetation Index.

+ = significant adverse association; (+) = non-significant elevated risk = when p > 0.05 and < 0.10; 0 = no association / effect; (-) = non-significant reduced risk; - = significant protective association.

Noise as confounder: + = included in analysis; (+) = included in sensitivity analysis/multi-pollutant models with similar results; not = not measured/included in analysis.

5.1.4 Suicide and ambient air pollution

Suicide in association with ambient air pollution was studied in relation to short-term elevated exposures and not long-term exposures in the included reviews, only. We identified four systematic reviews (Braithwaite et al., 2019; Davoudi et al., 2021; Heo et al., 2021; Liu et al., 2021) and two umbrella reviews (King et al., 2022; Radua et al., 2024). The included systematic reviews had end-search dates between 2017 (Braithwaite et al., 2019) to 2020 and included zero to one European study in their analyses, while the umbrella review by Radua et al. (2024) included all identified reviews except for Liu et al. (2021).

Overall, results indicated increased risk for mortality due to suicide or suicide attempts or hospitalization risks with elevated exposures to air pollutants in the days before the event (Table 5.9X). Risks were significantly (Davoudi et al., 2021; Liu et al., 2021) and non-significantly (Heo et al., 2021) elevated with PM_{2.5} with risk estimates ranging between 1.017 and 1.02 per 10 µg/m³. The same pattern was found for PM₁₀ with the significantly elevated risk estimates ranging between 1.004 and 1.02 per 10 µg/m³ from three analyses (Braithwaite et al., 2019; Davoudi et al., 2021; Liu et al., 2021) and non-significantly elevated risks in two analyses (Braithwaite et al., 2019; Heo et al., 2021). Braithwaite only found a higher suicide risk with increased exposures over the previous 2 days and not with the previous day.

Suicide risk and mortality were also significantly elevated with short-term NO₂ exposure (Davoudi et al., 2021; Heo et al., 2021). Ozone and SO₂ showed possibly elevated but non-significant effect estimates (Davoudi et al., 2021; Heo et al., 2021), whereas CO exposure seemed not to be associated with the outcome (Heo et al., 2021). The umbrella review by King et al. (2022) based on the other reviews (Davoudi et al., 2021; Heo et al., 2021; Liu et al., 2021) concluded that suicide risk was elevated with short-term increases of PM₁₀, PM_{2.5}, and NO₂. The umbrella review by Radua et al. (2024), however, deemed the associations for suicide mortality to be suggestive for shorter lags with NO₂ with a pooled estimate of 1.001 (95% CI: 1.001–1.002) per 1 µg/m³ and SO₂ (1.002, 95% CI: 1.001–1.003 per 1 µg/m³) and only weak with ozone and PM_{2.5} and PM₁₀.

Table 5.9 Results of meta-analyses of the association of suicide with short-term air pollution

Author (year)	End of search	Suicide outcome	Duration of exposure	Pollutant	Increment	# of European studies	# of studies or EE	Result	I ²
Davoudi (2021)	May 2020	Mortality	ST: lag 0-1	PM _{2.5}	10 µg/m ³	0	3	OR = 1.017 (1.003–1.031)	n/a
Heo (2021)	Aug 2020	Risk	ST	PM _{2.5}	13.1 µg/m ³	0	7	<i>RR = 1.02 (1.00–1.05)</i>	61%
Liu (2021)	Sep 2020	Suicide	ST	PM _{2.5}	10 µg/m ³	0	6	OR = 1.02 (1.01–1.03)	0.00%
Radua (2024)	Jun 2023	Mortality	ST: lag 0-1	PM _{2.5}	1 µg/m ³		n = 1323866	OR = 1.002 (1.00–1.003)	0%
Braithwaite (2019)	1974-Sep 2017	Completed	ST: lag 0-1	PM ₁₀	10 µg/m ³	1	3	<i>RR = 1.01 (0.99–1.03)</i>	44.60%
Braithwaite (2019)	1974-Sep 2017	Completed	ST: lag 0-2	PM ₁₀	10 µg/m ³	1	4	RR = 1.02 (1.00–1.03)	43.50%
Davoudi (2021)	May 2020	Mortality	ST: lag 0-1	PM ₁₀	10 µg/m ³	1	5	OR = 1.004 (1.000–1.008)	n/a
Heo (2021)	Aug 2020	Risk	ST	PM ₁₀	7.3 µg/m ³	1	7	<i>RR = 1.01 (1.00–1.03)</i>	62.30%
Liu (2021)	Sep 2020	Suicide	ST	PM ₁₀	10 µg/m ³	1	8	OR = 1.01 (1.00–1.01)	38.80%
Radua (2024)	Jun 2023	Mortality	ST: lag 0-1	PM ₁₀	1 µg/m ³		n = 1397117	OR = 1.00 (1.00–1.001)	19%
Davoudi (2021)	May 2020	Mortality	ST: lag 0-1	NO ₂	10 µg/m ³	0	4	OR = 1.013 (1.006–1.021)	n/a
Heo (2021)	Aug 2020	Risk	ST	NO ₂	17.7 ppm	0	6	RR = 1.03 (1.00–1.07)	64.10%
Radua (2024)	Jun 2023	Mortality	ST: lag 0-1	NO ₂	1 µg/m ³		n = 1396689	OR = 1.001 (1.001–1.002)	30%
Radua (2024)	Jun 2023	Mortality	ST: lag 0-2	NO ₂	1 µg/m ³		n = 76541	OR = 1.003 (1.00–1.005)	53%
Radua (2024)	Jun 2023	Mortality	ST: lag 0-3	NO ₂	1 µg/m ³		n = 76541	OR = 1.004 (1.00–1.007)	68%
Davoudi (2021)	May 2020	Mortality	ST: lag 0-1	SO ₂	10 µg/m ³	0	4	OR = 1.024 (1.014–1.034)	n/a
Heo (2021)	Aug 2020	Risk	ST	SO ₂	3.7 ppb	0	5	<i>RR = 1.02 (1.00–1.04)</i>	71.50%
Radua (2024)	Jun 2023	Mortality	ST: lag 0-1	SO ₂	1 µg/m ³		n = 1396689	OR = 1.002 (1.001–1.003)	0%
Radua (2024)	Jun 2023	Mortality	ST: lag 0-2	SO ₂	1 µg/m ³		n = 76541	OR = 1.003 (1.001– 1.005)	0%
Radua (2024)	Jun 2023	Mortality	ST: lag 0-3	SO ₂	1 µg/m ³		n = 76541	OR = 1.003 (1.001–1.005)	0%
Radua (2024)	Jun 2023	Mortality	ST: lag 0-4	SO ₂	1 µg/m ³		n = 74995	OR = 1.003 (1.001–1.005)	0%
Davoudi (2021)	May 2020	Mortality	ST: lag 0-2	O ₃	10 µg/m ³	1	3	<i>OR = 1.018 (0.997–1.039)</i>	n/a
Heo (2021)	Aug 2020	Risk	ST	O ₃	21.3 ppb	1	5	<i>RR = 1.02 (0.96–1.10)</i>	54.10%
Radua (2024)	Jun 2023	Mortality	ST: lag 0-3	O ₃	1 µg/m ³		n = 75815	<i>OR = 1.00 (0.999–1.00)</i>	0%
Radua (2024)	Jun 2023	Mortality	ST: lag 0-6	O ₃	1 µg/m ³		n = 73873	OR = 1.001 (1.00–1.002)	7%
Heo (2021)	Aug 2020	Risk	ST	CO	1.4 ppm	0	4	<i>RR = 1.02 (0.95–1.08)</i>	61.50%

Abbreviations/Symbols:

Risk = suicide attempt, complete, self-harm, ideation; ST = short-term exposure days (to weeks); lag 0-x = exposure averaged over days 0 and x; RR = Relative Risk, OR = Odds Ratio.

bold = statistically significant results; *italic* = non-statistically elevated risks/results.

A European time-series study from Belgium found increased risks for suicide in adults and adolescents with short-term increases of exposure to ozone (e.g. OR = 1.08, 95% CI: 1.01–1.16 per 10 µg/m³ lag 0–1 for adolescents and 1–2% in adults depending on lag studied) (Table 5.10). Results for PM₁₀ were significant for exposures during the summer or in either young or very old people, only (risk estimates 1.03–2.04 in 5–14 years old and 7–12% in adults over 84 years of age). Other pollutants were not analysed (Casas et al., 2017). Another Belgian study found that suicidal ideation in adolescents was not associated with long-term PM_{2.5}, EC or NO₂ exposure (Hautekiet et al., 2022). Whereas self-harm in children was associated in the Danish DNBC study with long-term exposure to PM_{2.5} and NO₂ (Mok et al., 2021). Finally, the pan-European ELAPSE study in adults, only found non-significantly elevated risks for suicide mortality with long-term exposure to PM_{2.5} (HR = 1.19, 0.76–1.87 per 5 µg/m³), EC and NO₂ (HR = 1.13, 0.92–1.38 per 10 µg/m³) with null or reduced risks with ozone (Andersen et al., 2022). None of these studies controlled for noise.

Overall, reviews suggest increased risk for suicides with short-term increased air pollution exposure with most rather weak evidence for PM, NO₂ and SO₂. The one European study found some association with ozone and PM₁₀. Reviews on long-term exposure to air pollution in association with suicide are lacking. The few European studies that were identified found some associations though confounding by other factors such as noise could not be ruled out.

Table 5.10 Original studies from Europe studying suicide in association with air pollution

Author (year)	Country	Study design	Study name	Population	# participants	Suicide outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	No _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Andersen (2022)	SE	CH	ELAPSE	Adults	271720	Mortality	(+)		(+)	(+)			(-)		Components, Sources	not
Hautekiet (2022)	BE	CS	BHIS	Adolescents, adults	16455	Ideation	0		0	0						not
Mok (2021)	DK	CH	DNBC	Children	1424670	Self-Harm hospitalization	+			+						not
Casas (2017)	BE	TS	x	Adolescents, adults	20533	ST Suicide		+ summer only					+		Age: PM ₁₀ + 5-14; +> 85 years	not

Abbreviations/Symbols:

SE = Sweden; BE = Belgium; DK = Denmark.

CH = cohort study; CS = cross-sectional study; TS = time-series study; NA = data not available.

ELAPSE = Effects of Low-Level Air Pollution: A study in Europe; BHIS = Belgian Health Interview Surveys; DNBC = Danish National Birth Cohort.

+ = significant adverse association; (+) = non-significant elevated risk = when $p > 0.05$ and < 0.10 ; 0 = no association / effect; (-) = non-significant reduced risk; - = significant protective association.

Noise as confounder: + = included in analysis; (+) = included in sensitivity analysis/multi-pollutant models with similar results; not = not measured/included in analysis.

5.1.5 Anxiety

Associations of anxiety disorders or symptoms of anxiety with air pollution were analysed in two meta-analyses (Cao et al., 2024; Trushna et al., 2021) and the umbrella review by Radua et al. (2024), which lists the review by Trushna et al. (2021) and finds weak evidence for PM₁₀ exposure with psychological distress (OR = 1.002, 95% CI: 1.00–1.005 per 10 µg/m³) (Table 5.11).

Cao et al. (2024) combined 6 effect estimates from five studies including 1 European study. The included study population was mixed ranging from children to elderly and pregnant women. The authors reported no “significant correlation” between PM_{2.5} exposure and anxiety disorder onset, however the meta-analytic estimate was RR = 1.10 (95% CI: 0.99–1.22). The other meta-analysis combined eleven effect estimates from five studies, with three overlapping studies from the review by Cao et al. (2024). They additionally included results from a pan-European children’s study analysis on symptoms and a Spanish cross-sectional analysis in adults and found a non-significantly reduced anxiety risk with higher PM_{2.5} exposure (OR = 0.88, 95% CI: 0.72–1.06 per 5 µg/m³). Results for PM₁₀ and NO₂ indicated even a significantly reduced risk (OR = 0.88, 95% CI: 0.78–0.98 per 10 µg PM₁₀/m³ and OR = 0.98, 95% CI: 0.89–0.97 per 10 µg NO₂/m³).

Table 5.11 Results of meta-analyses of the association of anxiety with long-term air pollution

Author (year)	End of search	Outcome	Duration of exposure	Pollutant	Increment	# of European studies	# of studies or EE	Result	I ²
Cao (2024)	Oct 2022	Anxiety (onset)	LT	PM _{2.5}	5 µg/m ³	1	6	<i>RR = 1.1, (0.99–1.22)</i>	71 %
Trushna (2021)	Mar 2020	Anxiety disorder	LT	PM _{2.5}	5 µg/m ³	3	11	OR = 0.88, (0.72–1.06)	80 %
Radua (2024)	Jun 2023	Psychological distress	LT	PM ₁₀	1 µg/m ³	NA	n = 1712	OR = 1.002, (1.00–1.005)	49 %
Trushna (2021)	Mar 2020	Anxiety disorder	LT	PM ₁₀	10 µg/m ³	2	10	<u>OR = 0.88, (0.78–0.98)</u>	59 %
Trushna (2021)	Mar 2020	Psychological distress	LT	PM ₁₀	10 µg/m ³	1	4	OR = 1.03, (1.00–1.05)	41 %
Trushna (2021)	Mar 2020	Anxiety disorder	LT	NO ₂	10 µg/m ³	11	11	<u>OR = 0.93, (0.89–0.97)</u>	0%

Abbreviations/Symbols:

LT = long-term exposure; NA = no data available, EE = effect estimates; # = number; n = number.

RR = relative risk; OR = odds ratio.

bold, statistically significant results; italic, non-statistically elevated risks/results; underlined, statistically significant negative risk/result indicative of a protective effect.

Other reviews without meta-analysis either conclude that there is “substantial evidence” for PM_{2.5} and anxiety (Chauhan et al., 2025), associations of annoyance and anxiety with air pollution (Lu, 2020), or that PM_{2.5}, NO₂ and SO₂ were triggers for exacerbation of anxiety (Tota et al., 2024). However, other authors are more cautious describing a “possible association” with increased PM exposure at both shorter and long-term exposure with anxiety (Braithwaite et al., 2019). Or they find the results

inconclusive or non-significant (King et al., 2022) or criticize the mixed results and limited number of studies and conclude that at least for children there is not greater risk of anxiety or depression with prenatal air pollution (Zundel et al., 2022). Finally, Bhui et al. (2023) describe studies that show associations for anxiety and air pollution, noise and increased temperatures and find it in general difficult to disentangle the effects of noise and air pollution.

European cohort studies in children, adolescents and young adults show no associations of air pollution with anxiety incidence (Newbury et al., 2024a; Roberts et al., 2019) (Table 5.12). Likewise, prevalence of anxiety or symptoms of anxiety were not associated with air pollutants in adolescents or young adults (Pelgrims et al., 2021; Roberts et al., 2019; Thompson et al., 2024). However, a Belgian study found non-significantly increased risks for generalized anxiety disorder and psychological distress with PM_{2.5} and EC exposure in adolescents and adults (Hautekiet et al., 2022) and a health impact assessment saw less anxiety cases and less medication sales against depression and anxiety in regions with low emission zones (LEZ) with reduced NO₂ and PM levels after introduction of LEZ compared to those without LEZ (Brehm et al., 2024).

Studies in adults generally show increased risks of anxiety incidence with various air pollutants, though except for one Italian study (Nobile et al., 2023) all eight other studies rely on UK Biobank data (Table 5.12). Results on prevalence are more mixed. Increased risks were found for PM_{2.5} in a European wide gene-wide association study (non-significant) (Bai et al., 2024c) and the Irish TILDA study (Lyons et al., 2024), whereas the Spanish ALFA study found null associations of PM_{2.5} with anxiety medication prescriptions (Vert et al., 2017) and various papers on the UK biobank data either find increased risks (Fan et al., 2024; Meng et al., 2024) or null results (Ma et al., 2024). Associations with PM₁₀ and incidence or prevalence were either significantly or non-significantly elevated in only three out of eleven analyses, with EC in 4 out of 5, with NO₂ in three out of seven analyses (additionally in all 9 UK biobank analyses positive), with NO_x in 1 out of two analyses (and 4 out of 5 UK biobank analyses) and with ozone in 3 out of 5 analyses positive. Depression or anxiety incidence was significantly elevated with long-term SO₂ exposure in a UK biobank analysis (Yuan et al., 2025).

Table 5.12 Original studies from Europe studying anxiety in association with air pollution

Author (year)	Country	Study name	Study design	Population	Number of participants	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Anxiety incidence															
Roberts (2019)	UK	E-Risk Twin	CH	Adolescents	284	0			0						not
Newbury (2024)	UK	ALSPAC	CH	Young adults	9065	0			0					noise +	not
Brehm (2024)	DE	x	CS	Adolescents, adults	2029359									LEZ - PM NO ₂ reduced	not
Gao (2023)	UK	UK Biobank	CH, CS	Adults	345876	+	+		+	+				PM _{10-2.5} 0	not
Jin (2024)	UK	UK Biobank	CH	Adults	257534							+			not
Nobile (2023)	IT	RoLS	CH	Adults	1733331	+		+	+					UFP(+), noise (+)	x
Pan (2024)	UK	UK Biobank	CH	Adults	170369	+			+					PM _{10-2.5} 0, NO +	x
Yang (2023)	UK	UK Biobank	CH	Adults	389185	+			+					PM _{10-2.5} 0, NO +	not
Yao (2025)	UK	UK Biobank	CH	Adults	252376	+	0	(+)	+	+				PM _{10-2.5} 0	not
Yuan (2025)	UK	UK Biobank	CH	Adults	245820						+			noise +	x
Gao (2023)	UK	UK Biobank	CH, CS	Adults	345876	+	+		+	+				PM _{10-2.5} 0	not
Jin (2024)	UK	UK Biobank	CH	Adults	257534							+			not
Anxiety prevalence															
Pelgrims (2021)	BE	BHIS	CS	Adolescents, adults	1325	0	0	0	0			0		NDVI 0, noise 0, street 0	x
Hautekiet (2022)	BE	BHIS	CS	Adolescents, adults	16455	(+)		(+)	0						not
Thompson (2024)	UK	SCAMP	CH	Adolescents	7555	0	0		0			0		noise +	x
Roberts (2019)	UK	E-Risk Twin	CH	Adolescents	284	0	-	-	0	-	-	-	-		not
Brehm (2024)	DE	x	CS	Adolescents, adults	2029359	-	-	-	-	-	-	-	-	LEZ - PM NO ₂ reduced	not
Zhao (2020)	DE	AOK PLUS	CS	Adolescents, adults	1.13 Million		+					+			not
Bai (2024)	Europe	UK Biobank IEU OPEN GWAS	CS	Adults	NA	(+)	0		0	0				PM _{10-2.5} 0	not
Fan (2024)	UK	UK Biobank	CS	Adults	42000-45000	+	0		(+)	+				PM _{10-2.5} 0	not

Author (year)	Country	Study name	Study design	Population	Number of participants	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Lyons (2024)	IE	TILDA	CS	Adults	3407	+	-	-	-	-	-	-	-		not
Ma (2024)	UK	UK Biobank	CS	Adults	NA	0	0		0	0				PM _{10-2.5} 0	not
Vert (2017)	ES	ALFA	CS	Adults	958	0	0	(+)	(+)	(+)	-	-	-	PM _{10-2.5} 0	not
Petrowski (2021)	DE	USUMA	CS	Adults	3020	-	0	-	-	-	-	-	-		not
Meng (2024)	UK	UK Biobank	CS	Adults high genetic risk vs low	104385	+			+					NO +, PM _{10-2.5} +	not
Symptoms of anxiety															
Lyons (2024)	IE	TILDA	CS	Adults	3407	+	-	-	-	-	-	-	-		not
Meng (2024)	UK	UK Biobank	CS	Adults high genetic risk vs low	104385	+			+					NO +, PM _{10-2.5} +	not
Emergencies															
Muhsin (2022)	SE	x	TS	Adults	81548	0	0								not

Abbreviations/Symbols:

UK = United Kingdom, DE = Germany, IT = Italy, BE = Belgium, IE = Ireland, ES = Spain, SE = Sweden.

E-Risk Twin = Environmental Risk Longitudinal Twin Study; ALSPAC = The Avon Longitudinal Study of Children and Parents; UK Biobank (italic) = UK Biobank Research Analysis Platform; RoLS = Rome longitudinal study; BHIS = Belgian Health Interview Surveys; SCAMP = Study of Cognition, Adolescents and Mobile Phones; AOK PLUS = Study with administrative health records of health insurer AOK; TILDA = The Irish Longitudinal Study on Ageing; ALFA = Early Identification of Markers in Alzheimer's Families; USUMA = Random, representative sample of German population by USUMA, an independent service for surveys, methods, and analyses.

CH = cohort study; CS = cross-sectional study; TS = time-series study; NA = data not available.

+ = significant adverse association; (+) = non-significant elevated risk = when $p > 0.05$ and < 0.10 ; 0 = no association / effect; (-) = non-significant reduced risk; - = significant protective association.

Noise as confounder: + = included in analysis; (+) = included in sensitivity analysis/multi-pollutant models with similar results; not = not measured/included in analysis.

Noise was included as a confounder in five studies (Nobile et al., 2023; Pan et al., 2024; Pelgrims et al., 2021; Thompson et al., 2024; Yuan et al., 2025). Results were positive for PM_{2.5}, EC and NO₂ in the Rome longitudinal incidence study independent from noise (risks were non-significantly elevated with noise) (Nobile et al., 2023), as were the results for PM_{2.5}, NO₂ and SO₂ in the UK biobank analyses (Pan et al., 2024; Yuan et al., 2025). The Belgian study BHIS did not find any associations with air pollution or noise (Pelgrims et al., 2021). Finally, the UK analysis of ALSPAC data in adolescents found increased incidence of anxiety with noise but not air pollutants and the results remained robust for noise in the multi-pollutant analysis with air pollutants (Newbury et al., 2024a).

Overall, the study base seems limited and results from reviews and meta-analyses show mixed results regarding associations of air pollution with anxiety incidence or prevalence. European studies in adolescents also do not support an association of anxiety symptoms or disorders with early childhood exposure and low support for an association comes from adult studies to date (except for UK Biobank analyses). Some authors point out that perceived air pollution can trigger existential anxiety about one's health and future (Lu, 2020) and according to King et al. (2022), SO₂ exposure has been linked with anxiety for many years, as one of the only perceptibly foul-smelling pollutants. Awareness of one's exposure is more likely at lower levels and perception of poor physical environment and awareness of poor-quality air have been proposed as a psychological pathway linking air pollution to distress. However, the data found do not readily support this hypothesis.

5.1.6 Schizophrenia

Out of eight selected reviews three conducted meta-analysis (Radua et al., 2024; Song et al., 2023; Xu et al., 2023) of short-term associations of air pollution with hospital admissions for or relapses of schizophrenia (Table 5.13). No meta-analysis studied long-term effects. The umbrella review by Radua et al. (2024) based their assessment on results of the review by Song et al. (2023), that included studies up to December 2022. Radua et al. (2024) classified the association of short-term elevated SO₂ exposure 5 and 7 days before a relapse as suggestive (RR = 1.005; 95% CI: 1.004–1.006; and RR = 1.004; 95% CI: 1.003–1.005 per 1 µg SO₂/m³, respectively 5 and 7 days after exposure). Associations with other time lags or PM_{2.5} were classified as weak (RR = 1.001; 95% CI: 1.0–1.002 per 1 µg PM_{2.5}/m³) (Radua et al. 2024). Song et al. (2023) additionally found significantly elevated risk for hospital admissions or emergency department / outpatient visits with short-term increases of PM₁₀, PM_{10-2.5} and NO₂, but not with CO. The review by Xu et al. (2023) focused on associations with short-term increases of NO₂ with hospital admissions due to schizophrenia and found significantly elevated risks (RR = 1.03, 95% CI: 1.02–1.04 per 10 µg/m³), that were robust in 2-pollutant models with PM_{2.5}, PM₁₀ and SO₂. Notably, none of the reviews included European studies in their analysis and only one to two US studies, that showed null associations.

The reviews without meta-analysis find that air pollution such as PM_{2.5}, NO₂ and SO₂ seem detrimental to patients with schizophrenia (Tota et al., 2024) with some limited evidence (King et al., 2022). However, various other reviews point out the limitations by the studies, which are often cross-sectional and small in size and cannot exclude risks by residual confounding, especially regarding traffic noise (Attademo et al., 2017; Bhui et al., 2023; Braithwaite et al., 2019).

One time-series studies from France found an increase of hospital admissions with short-term exposure to PM_{2.5} and PM₁₀ (Pignon et al., 2022) (Table 5.14). The other identified European studies showed generally increased incidence and prevalence of schizophrenia or psychotic experiences with long-term air pollution.

Table 5.13 Results of meta-analyses of the association of schizophrenia with short-term air pollution

Author (year)	End of search	Duration of exposure	Pollutant	Increment	# of European studies	# of studies or EE	Result (RR, 95% CI)	I ²
Radua (2024)	Jun 2023	ST	PM _{2.5}	1 µg/m ³	NA	n = 33755	RR = 1.001, (1.00–1.001)	61%
Song (2023)	Feb 2022	ST	PM _{2.5}	10 µg/m ³	0	10	RR = 1.005, (1.0017–1.0083)	57.40%
Radua (2024)	Jun 2023	ST: lag2	PM _{2.5}	1 µg/m ³	NA	n = 20732	RR = 1.001, (1.00–1.002)	0%
Radua (2024)	Jun 2023	ST: lag5	PM _{2.5}	1 µg/m ³	NA	n = 12050	RR = 1.001, (1.00–1.002)	0%
Radua (2024)	Jun 2023	ST	PM ₁₀	1 µg/m ³	NA	n = 33755	RR = 1.00, (1.00–1.001)	79%
Song (2023)	Feb 2022	ST	PM ₁₀	10 µg/m ³	0	10	RR = 1.0047, (1.0025–1.007)	77%
Radua (2024)	Jun 2023	ST: lag3	PM ₁₀	1 µg/m ³	NA	n = 25073	RR = 1.001, (1.00–1.001)	76%
Radua (2024)	Jun 2023	ST: lag5	PM ₁₀	1 µg/m ³	NA	n = 12050	RR = 1.001, (1.00–1.001)	0%
Radua (2024)	Jun 2023	ST	PM _{10-2.5}	1 µg/m ³	NA	n = 12050	RR = 1.002, (1.001–1.003)	21%
Song (2023)	Feb 2022	ST	PM _{10-2.5}	10 µg/m ³	0	3	RR = 1.0117, (1.0023–1.0211)	69.40%
Radua (2024)	Jun 2023	ST: lag3	PM _{10-2.5}	1 µg/m ³	NA	n = 7709	RR = 1.001, (1.00–1.003)	0%
Radua (2024)	Jun 2023	ST: lag4	PM _{10-2.5}	1 µg/m ³	NA	n = 7709	RR = 1.001, (1.00–1.002)	0%
Radua (2024)	Jun 2023	ST	NO ₂	1 µg/m ³		n = 42154	RR = 1.002, (1.001–1.004)	83%
Song (2023)	Feb 2022	ST	NO ₂	10 µg/m ³	0	9	RR = 1.0275, (1.0132–1.042)	90.70%
Xu (2023)	Dec 2022	ST	NO ₂	10 µg/m ³	0	12	RR = 1.03, (1.02–1.04)	89.60%
Radua (2024)	Jun 2023	ST: lag1	NO ₂	1 µg/m ³		n = 17364	RR = 1.002, (1.001–1.004)	79%
Radua (2024)	Jun 2023	ST	SO ₂	1 µg/m ³	NA	n = 33472	RR = 1.003, (1.001–1.005)	85%
Song (2023)	Feb 2022	ST	SO ₂	10 µg/m ³	0	7	RR = 1.0288, (1.0146–1.0432)	79.10%
Radua (2024)	Jun 2023	ST: lag5	SO ₂	1 µg/m ³	NA	n = 8682	RR = 1.005, (1.004–1.006)	0%
Radua (2024)	Jun 2023	ST: lag7	SO ₂	1 µg/m ³	NA	n = 8682	RR = 1.004, (1.003–1.005)	0%
Radua (2024)	Jun 2023	ST: lag1	SO ₂	1 µg/m ³	NA	n = 17364	RR = 1.001, (1.00–1.001)	0%
Radua (2024)	Jun 2023	ST: lag2	SO ₂	1 µg/m ³	NA	n = 17364	RR = 1.001, (1.00–1.003)	70%
Radua (2024)	Jun 2023	ST: lag3	SO ₂	1 µg/m ³	NA	n = 21705	RR = 1.002, (1.00–1.004)	86%
Radua (2024)	Jun 2023	ST: lag6	SO ₂	1 µg/m ³	NA	n = 8682	RR = 1.006, (1.002–1.009)	56%

Abbreviations/Symbols:

ST = short-term exposure; lagx = exposure at day x before event; NA = no data available, EE = effect estimates; # = number; n = number; RR = Relative Risk; OR = Odds Ratio; bold = statistically significant results; italic = non-statistically elevated risks/results; underlined = statistically significant negative risk/result indicative of a protective effect.

Table 5.14 Original studies from Europe studying schizophrenia in association with air pollution

Author (year)	Country	Study design	Study name	Population	# participants	Schizophrenia outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	No _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Incidence																
Antonsen (2020)	DK	CH	x (register)	Young adults	230844	Incidence	(+)	(+) to +		+	+				No _x did not contribute beyond NO ₂ in two-pollutant models	not
Newbury (2024)	UK	CH	ALSPAC	Young adults	9065	Psychotic experience Incidence	+			0					noise 0	not
Newbury (2021)	England	CH	SLaM	Adolescent , adult patients	13887	Consultations	+	+		+	+					not
Newbury (2021)	England	CH	SLaM	Adolescent , adult patients	13887	Hospital admissions	+	+		+	+					not
Nobile (2023)	IT	CH	RoLS	Adults	1733331	Incidence	+		+	(+)					UFP +, noise +	(x) = for UFP
Liu (2024)	UK	CH	UK Biobank	Adults	485288	Incidence	+	+		+	+					not
Pan (2024)	UK	CH	UK Biobank	Adults	170369	Incidence	(+)			(+)					PM _{10-2.5} 0, NO 0	x
Yao (2025)	UK	CH	UK Biobank	Adults	252376	Incidence	(+)	0	+	+	+				PM _{10-2.5} 0	not

Author (year)	Country	Study design	Study name	Population	# participants	Schizophrenia outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Yuan (2025)	UK	CH	UK Biobank	Adults	245820	Incidence						0			noise 0	x
Zhang (2024)	UK	CH	UK Biobank	Adults	437802	Incidence	+	+		+	+					not
Prevalence																
Newbury (2019)	England, Wales	CS	E-Risk Twin	Adolescents	2063	Psychotic experience	+	(+)		+	+					not
Reuben (2021)	England, Wales	CS	E-Risk Twin	Adolescents	2039	Mental Disorder (thought/psychosis)	(+)				+					not
Bakolis (2021)	UK	CS	SELCoH	Adolescents, adults	1052 adolescents, 1655 adults	Psychotic Experiences Prevalence	(-)	+		(+)	(+)		-			x
Horsdal (2019)	DK	CC	iPSYCH register	(Young) Adults	23355	Prevalence				+						not
Bai (2024)	Europe	CS	UK Biobank IEU OPEN GWAS	Adults	NA	Prevalence (causal)	0	+		0	+				PM _{10-2.5} 0	not
Fan (2024)	UK	CS	UK Biobank	Adults	42000-45000 adults	Prevalence	+	0		+	+				PM _{10-2.5} 0	not
Ma (2024)	UK	CS	UK Biobank	Adults	NA	Prevalence	+	0		+	(+)				PM _{10-2.5} 0	not
Emergencies																
Pignon (2022)	FR	TS	x	Emergencies	20727	Daily Emergencies	+	+								not

Abbreviations/Symbols:

DK = Denmark; UK = United Kingdom; IT = Italy; FR = France.

CH = cohort study; CS = cross-sectional study; TS = time-series study; NA = data not available.

ALSPAC, The Avon Longitudinal Study of Children and Parents; SLaM, South London and Maudsley NHS Foundation Trust for psychotic and mood disorders; RoLS, Rome longitudinal study; UK Biobank, UK Biobank Research Analysis Platform; E-Risk Twin, Environmental Risk Longitudinal Twin Study; SELCoH, South East London Community Health study; iPSYCH register, The Lundbeck Foundation Initiative for Integrative Psychiatric Research.

+ = significant adverse association; (+) = non-significant elevated risk = when $p > 0.05$ and < 0.10 ; 0 = no association / effect; (-) = non-significant reduced risk; - = significant protective association.

Noise as confounder: + = included in analysis; (+) = included in sensitivity analysis/multi-pollutant models with similar results; not = not measured/included in analysis.

PM_{2.5} was consistently associated with an increased risk of developing schizophrenia or psychotic experiences in adolescents and young adults in a Danish register study (borderline) (Antonsen et al., 2020) and the UK ALSPAC cohort study (Newbury et al., 2024a), as well as in adults in Italy (Nobile et al., 2023). Mental health consultations and hospital admissions were also significantly increased with PM_{2.5} in Southeast London in adolescents and adults (Newbury et al., 2021). The UK biobank analyses found either significantly increased risks with PM_{2.5} or borderline significant results (Liu et al., 2024; Pan et al., 2024; Zhang et al., 2024; Yao et al., 2025; Yuan et al., 2025). Results of prevalence data were not that consistent. Four of the six studies looking into schizophrenia prevalence showed positive associations with PM_{2.5} (Fan et al., 2024; Ma et al., 2024; Newbury et al., 2019; Reuben et al., 2021), a UK study found borderline preventive association with psychotic experiences (Bakolis et al., 2021) and null associations in a causal modelling genome wide associations study with UK biobank data (Yuan et al., 2025; Bai et al., 2024c). PM₁₀ showed a similar pattern with more consistent positive association with schizophrenia incidence and mixed results with prevalence. The risk to develop schizophrenia was increased with BC and UFP exposure in two out of two studies in Italy (Nobile et al., 2023) and with EC in a UK biobank analysis (Yao et al., 2025). Schizophrenia incidence was generally increased with NO₂ and NO_x, as was prevalence. The null finding in the ALSPAC cohort (Newbury et al., 2024a) and the null findings in the causal analysis by Bai et al. (2024c) were an exception to this generally consistent picture. SO₂ was not associated with schizophrenia incidence in one UK biobank analysis (Yuan et al., 2025). Schizophrenia outcomes in association with ozone or CO exposure were not assessed in any of the European studies.

Regarding independent effects from noise exposure, the Rome longitudinal study found increased risk for schizophrenia incidence with PM_{2.5}, BC and UFP, as well as noise. In two- and multipollutant models only the association with UFP remained significantly elevated (Nobile et al., 2023). The Southeast London study on mental health consultations and hospital admissions included noise as a confounder and found elevated risk with PM₁₀ and borderline associations with NO₂ and NO_x (Newbury et al., 2021). Two UK biobank analysis included noise as a confounder in their analysis with null results for noise and SO₂ (Yuan et al., 2025), null results for NO and PM_{10-2.5} but non-significantly elevated risks with PM_{2.5} and NO₂ (Pan et al., 2024). Finally, the ALSPAC analysis by Newbury et al. (2024a) only considered noise in separate models and found null effects for noise exposure.

Overall, exacerbation of pre-existing disease could be triggered by short-term increases of air pollution. We believe that noise exposure is not highly correlated with air pollution in short-term studies, thus not a relevant confounder. The study base for long-term associations of schizophrenia incidence and prevalence with air pollution is rather small with no reviews identified. Results regarding schizophrenia incidence from European studies with PM_{2.5}, NO₂, NO_x, EC and UFP seem to point towards the direction of some associations with a few studies considering noise as a co-pollutant. However, even though there exists some biological plausibility for effects from mechanistic (animal) studies, this needs to be followed up with research on specific mechanisms, and caution is needed before inferring causality. There are numerous potential confounders and covariates that need to be taken into account and there does not yet exist a strong case from animal studies to support the hypothesis, according to Attademo et al. (2017).

5.1.7 Bipolar disorder

The reviews also including bipolar disorder into the scope of their work reported, that they did not identify studies on the outcome (Braithwaite et al., 2019; Buoli et al., 2018). We identified ten papers, of which seven based their results on UK biobank data and two assessed short-term exposures with emergencies or severity of bipolar disorder (Carugno et al., 2021; Pignon et al., 2022) (Table 5.15).

The Rome longitudinal study found no consistent associations of bipolar disorder incidence with environmental variables; non significantly increased associations with EC and UFP exposure, significantly decreased risks with PM_{2.5} and NO₂ exposure and a null association with noise (Nobile et al., 2023). The results of the UK biobank data on bipolar disorder incidence and prevalence also showed mixed results for the studied pollutants (PM_{2.5}, PM₁₀, NO₂, NO_x, SO₂) (Hao et al., 2022; Ma et al., 2024; Pan et al., 2024; Yuan et al., 2025; Li et al., 2023b; Bai et al., 2024c; Fan et al., 2024). The French time series study did not find associations of PM_{2.5} or PM₁₀ with daily emergencies (Pignon et al., 2022). A small panel study following 186 patients with manic episodes in Milan found an increased risk for hospitalization due to manic episodes with mixed features with elevated levels of PM₁₀ on and before the days of the event (OR = 2.50; 95% CI 1.03–6.08). Severity of the manic episode measured with the Young Mania Rating Scale was reduced (Carugno et al., 2021).

Overall, there is very limited evidence for an association of bipolar disorders with air pollution due to missing reviews and only few studies from Europe with mixed results. There could be indications of air pollution triggering episodes of disease, but this was only found in a small study.

Table 5.15 Original studies from Europe studying bipolar disorders in association with air pollution

Author (year)	Country	Study design	Study name	Population	# participants	Bipolar disorder outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other Pollutants / Comment	Noise as confounder
Incidence																
Nobile (2023)	IT	CH	RoLS	Adults	1733331	Incidence	-		(+)	-					UFP (+), noise 0	x
Pan (2024)	UK	CH	UK Biobank	Adults	170369	Incidence	0			0					PM _{10-2.5} 0, NO 0	x
Yuan (2025)	UK	CH	UK Biobank	Adults	245820	Incidence						0			noise 0	x
Li (2023)	UK	CH	UK Biobank	Adults	482726	Incidence	+	+		+	+					not
Prevalence																
Bai (2024)	Europe	CS	UK Biobank IEU OPEN GWAS	Adults	NA	Prevalence	0	0		0	(+)				PM _{10-2.5} 0	not
Fan (2024)	UK	CS	UK Biobank	Adults	42000-45000	Prevalence	+	0		+	0				PM _{10-2.5} 0	not
Hao (2022)	UK	CS	UK Biobank	Adults	90706	Prevalence	+								noise +	not
Ma (2024)	UK	CS	UK Biobank	Adults	NA	Prevalence	+	0		+	0				PM _{10-2.5} 0	not
Short-term Emergencies																
Pignon (2022)	FR	TS	x	All	20727	Daily Emergencies	0	0								not
Carugno (2021)	IT	CS	Policlinico	Adult Bipolar Patients	186	Severity: Charac. Manic Episode		+							more mixed features incl. psychotic characteristics	not
Carugno 2021	IT	CS	Policlinico	Adult Bipolar Patients	186	Severity: YMRS Mania Rating		-							less severe manic symptoms	not

Abbreviations/Symbols:

IT = Italy; UK = United Kingdom; FR = France.

RoLS = Rome longitudinal study; UK Biobank (*italic*) = UK Biobank Research Analysis Platform; Policlinico = Persons with diagnosis of major depressive disorder at the Psychiatry Unit of the Policlinico Hospital in Milan.

CH = cohort study; CS = cross-sectional study; TS = time-series study; NA = data not available.

+ = significant adverse association; (+) = non-significant elevated risk = when $p > 0.05$ and < 0.10 ; 0 = no association / effect; (-) = non-significant reduced risk; - = significant protective association.

Noise as confounder: + = included in analysis; (+) = included in sensitivity analysis/multi-pollutant models with similar results; not = not measured/included in analysis.

5.1.8 General mental health and ambient air pollution

Reviews (see Table 5.1) looking beyond specific mental health outcomes regarding associations of air pollution with more general mental health or combined mental health outcomes, conclude that there is emerging evidence for worsening of psychiatric symptoms (Bernardini et al., 2019) or poor mental health incidence or prevalence in association with air pollution (Bhui et al., 2023; Braithwaite et al., 2019; Cardenas-Iniguez et al., 2022; Trombley, 2023) or specific air pollutants such as PM, SO₂ and CO (Bazyar et al., 2019; Radua et al., 2024), except for ozone (Zhao et al., 2018). Additionally, there is emerging evidence on early warning biomarkers that are related to oxidative stress and inflammation, and epigenetic and genetic mechanisms that could be on the pathway to symptoms and disease (Bai et al., 2024b). However, the same authors caution, that the existing studies are still insufficient, and heterogeneity in the study population and study design may weaken the reliability and generalizability of the existing study results. In addition, Bhui et al. (2023) stress that results for noise exposure in association with mental health are similar, which make it difficult to disentangle the results. There was not meta-analysis on these more general endpoints.

European studies generally find positive association of diminished mental health with air pollution (Table 5.16). Mental health in these studies was measured with various instruments like the general health questionnaire GHQ, the mental health inventory MHI, or the clinical interview schedule CIS-R, clinical diagnoses, as incidence or prevalence of mental health consultations or emergencies or mortality with mental health diagnoses. Due to these different outcomes and instruments applied comparison is difficult. The outcome of symptom scores of the questionnaire instruments was either analysed as the continuous change in score or dichotomous with specific cut-offs.

Cohort studies in the UK, Sweden and the Netherlands found that long-term air pollution to PM_{2.5}, PM₁₀ and NO₂ was associated with diminished mental health scores or symptoms of mental health that needed treatment in children, adolescents or young adults (Abed Al Ahad et al., 2022; Bloemsma et al., 2022b; Oudin et al., 2016). UK biobank data also indicated an increased incidence of mental disorder diagnoses in adults with various air pollutants and noise (Pan et al., 2024; Yuan et al., 2025; Yao et al., 2025). Additionally, mortality due to or with mental health disorders was associated with long-term PM_{2.5}, EC, NO₂ exposure but not ozone in the analysis of the pan-European ELAPSE data (Andersen et al., 2022) and the Danish national register cohort (So et al., 2023).

Results of prevalence data were not as consistent as the incidence analyses. In adolescents the Belgian BHIS data found no associations regarding mental health measure with GHQ, but poor rated general health (Hautekiet et al., 2022; Pelgrims et al., 2021). The UK E-Risk Twin study found borderline significant results of PM_{2.5} with symptoms of psychopathological behaviour in adolescents (Reuben et al., 2021), whereas another analysis with SELCoH data found positive associations (Bakolis et al., 2021). Studies in adults and young adults from Bulgaria and Portugal find null results with NO₂ and noise (Dzhambov et al., 2018) or borderline significant results with PM₁₀ (Pinheiro Guedes et al., 2024). In an Italian and Swedish study daily emergencies due to psychiatric emergencies were increased with PM_{2.5}. The Swedish study additionally found an association with PM₁₀ (Muhsin et al., 2022), but the Italian analysis found paradoxical borderline decreased emergencies with PM₁₀ and NO₂ (Bernardini et al., 2019).

European studies, that included noise in their analyses generally find robust results with various air pollutants. Effect estimates for NO₂ and EC became stronger upon inclusion of greenness and noise in the models in the Dutch analysis of PIAMA data (Bloemsma et al., 2022b) with no associations for noise. The two UK biobank analyses including noise in their models found independent effects of PM_{2.5}, NO₂ or SO₂ with overall mental disorder incidence (Pan et al., 2024; Yuan et al., 2025). Likewise, the prevalence study in SELCoH found independent air pollutant effects (Bakolis et al., 2021), while the

Bulgarian analysis neither find an association of NO₂ with mental health nor with noise (Dzhambov et al., 2018).

Overall, the described emerging evidence for mental health outcomes in general with air pollution in various reviews seems to be backed by European study results, that indicated independent effects from noise.

Table 5.16 Original studies from Europe studying general mental health outcomes in association with air pollution

Author (year)	Country	Study design	Study name	Population	# participants	Outcome (measure)	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Incidence																
Abed Al Ahad (2022)	UK	CH	UKHLS	Adolescents, adults	60146	Mental Health GHQ-12	+	+		+		+				not
Oudin (2016)	SE	CH	x	Children, Adolescents	552221	Symptoms Mental Health (Medication)	+	+		+					Two-pollutant model NO ₂ +, PM ₁₀ -	not
Bloemsm a (2022)	NL	CH	PIAMA	Children, Adolescents	3059	Poor mental wellbeing MHI-5	+	(+)	+	+					OP +, noise 0, NDVI -	(x) attenuated, strong for NO ₂ & PM _{2.5} abs
Ronaldson (2023)	UK	CH	SLaM	Patients with dementia	5024	Mental health consultations	+	+		+						not
Yao (2025)	UK	CH	UK Biobank	Adults	252376	Mental Disorder	+	0	+	+	+				PM _{10-2.5} 0	not
Yuan (2025)	UK	CH	UK Biobank	Adults	245820	Mental Disorder						+			noise +	x
Pan (2024)	UK	CH	UK Biobank	Adults	170369	Mental Disorder	+			+					PM _{10-2.5} (+), NO +	x
Mortality																
Andersen (2022)	SE	CH	ELAPSE	Adults	271720	Mortality Psychiatric Disorders	(+)		+	+			-		Components, Sources	not
So (2022)	DK	CH	DNR	Adults	3 Million	Mortality Psychiatric Disorders	+		+	+			-			not

Author (year)	Country	Study design	Study name	Population	# participants	Outcome (measure)	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
So (2023)	DK	CH	DNR	Adults	3 Million	Mortality Psychiatric Disorders	+								PM _{2.5} Components Cu + , Fe + , Zn+	not
Prevalence																
Pelgrims (2021)	BE	CS	BHIS	Adolescents, adults	1325	Mental Health GHQ-4	0	0	0	0			0		NDVI 0, noise 0, street canyon 0	x
Hautekiet (2022)	BE	CS	BHIS	Adolescents, adults	16455	Mental Health GHQ	0		0	(+)						not (but urbanisation)
Hautekiet (2022)	BE	CS	BHIS	Adolescents, adults	16455	Poor self-rated health	+		+	+						not (but urbanisation)
Dzhambov (2018)	BE	CS	Plovdiv Study	Young adults	720	Mental Health GHQ				0					noise 0 only + in crude	x
Reuben (2021)	England, Wales	CS	E-Risk Twin	Adolescents	2039	Symptoms Psychopathological Behaviour	(+)				+					not
Bakolis (2021)	UK	CS	SELCoH	Adolescents, adults	2700	Mental Disorder CIS-R Prevalence	+	(+)		+	+		(-)			x
Pinheiro Guedes (2024)	PT	CS	INSEF	Adults	2398	Mental Disorders MHI-5		(+)								not
Pinheiro Guedes (2024)	PT	CS	INSEF	Adults	2398	Symptoms Mental Disorders MHI-5		(+)								not
Short-term Emergencies																
Bernardini 2019	IT	TS	x	Emergencies	1860	Daily psychiatric Emergencies	(+)	(-)		(-)			+	(+)	results of multipollutant model	not

Author (year)	Country	Study design	Study name	Population	# participants	Outcome (measure)	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Muhsin 2022	SE	TS	x	Adults	81548	Daily psychiatric Emergencies	+	+								not

Abbreviations/Symbols:

UK = United Kingdom; NL = the Netherlands; SE = Sweden; BE = Belgium; IT = Italy; PT = Portugal.

CH = cohort study; CS = cross-sectional study; TS = time-series study.

Study name: UKHLS, UK Household Longitudinal Study; PIAMA, Prevention and Incidence of Asthma and Mite Allergy; SLaM, South London and Maudsley NHS Foundation Trust for psychotic and mood disorders; UK Biobank, UK Biobank Research Analysis Platform; ELAPSE, Effects of Low-Level Air Pollution: A study in Europe; DNR, Danish National Register; BHIS, Belgian Health Interview Surveys; E-Risk Twin, Environmental Risk Longitudinal Twin Study; SELCoH, South East London Community Health study; INSEF, Portuguese National Health Examination Survey.

Effects: +, significant adverse association; (+), non-significant elevated risk, when $p > 0.05$ and < 0.10 ; 0, no association / effect; (-), non-significant reduced risk; -, significant protective association.

Noise as confounder: +, included in analysis; (+), included in sensitivity analysis/multi-pollutant models with similar results; not, not measured/included in analysis.

5.1.9 Other endpoints of mental health disease

A few European studies were identified for less well studied mental health outcome, for which we could not identify any reviews (Table 5.17). The mental health endpoints of eating disorders (Anorexia nervosa), obsessive compulsive disorder and posttraumatic stress disorder were not associated with long-term air pollution in a UK biobank analysis (Ma et al., 2024), as well as personality disorders in the Rome longitudinal cohort study (Nobile et al., 2023). However, substance abuse incidence was associated with PM_{2.5} (but not EC, NO₂, UFP or noise) in this analysis (Nobile et al., 2023). A Swedish time-series analysis found no association of substance abuse emergencies with short-term exposure to PM_{2.5} or PM₁₀ (Muhsin et al., 2022).

To date, there is no sufficient evidence for associations of eating disorders, substance abuse disorder or personality disorders with air pollution.

Table 5.17 Original studies from Europe studying other mental health endpoints in association with air pollution

Author (year)	Country	Study design	Study name	Population	# participants	Outcome	PM _{2.5}	PM ₁₀	EC	NO ₂	NO _x	SO ₂	O ₃	CO	Other pollutants	Noise as confounder
Ma (2024)	UK	CS	UK Biobank	Adults	NA	Anorexia nervosa	0	0			0				PM _{10-2.5} 0	not
Ma (2024)	UK	CS	UK Biobank	Adults	NA	OCD obsessive compulsive disorder	0	0 PM _{10-2.5} 0		0	0				PM _{10-2.5} 0	not
Ma (2024)	UK	CS	UK Biobank	Adults	NA	PTSD	0	0		0	(+)				PM _{10-2.5} 0	not
Nobile (2023)	IT	CH	RoLS	Adults	1733331	Personality Disorder	0		0	0					UFP 0, noise 0	x
Nobile (2023)	IT	CH	RoLS	Adults	1733331	Substance Abuse Incidence	+		0	0					UFP 0, noise 0	x
Muhsin (2022)	SE	TS	x	Adults	81548	Daily emergencies substance abuse	0	0								not

Abbreviations/Symbols:

UK = United Kingdom; IT = Italy; SE = Sweden.

CH = cohort study; CS = cross-sectional study; TS = time-series study; NA = data not available.

RoLS = Rome longitudinal study; UK Biobank (italic) = UK Biobank Research Analysis Platform; x = no name.

+ = significant adverse association; (+) = non-significant elevated risk = when $p > 0.05$ and < 0.10 ; 0 = no association / effect; (-) = non-significant reduced risk; - = significant protective association.

Noise as confounder: + = included in analysis; (+) = included in sensitivity analysis/multi-pollutant models with similar results; not = not measured/included in analysis.

5.1.10 Secondary health outcomes

We did not evaluate associations of air pollution with autism spectrum disorder and attention-deficit disorder, since we evaluated these endpoints in a dedicated previous report on children's health in 2022 (EEA, 2022a). The review included studies until June 2022 – in the meantime many more studies have been published that might alter or strengthen the conclusions made here. However, this was out of the scope of this review and we describe our conclusion from 2022 here briefly.

Autism spectrum disorder

Autism is a condition that includes a spectrum of impairments affecting social interaction, language development, and communication skills and that often involves rigid and repetitive behaviours (US EPA, 2019). Overall, there was emerging evidence on associations of autism spectrum disorder with strongest results for PM_{2.5} and NO₂.

Studies were mainly conducted in North America or Europe. Uncertainties remain regarding co-pollutant exposures like noise or other air pollutants, exposure time windows, with evidence pointing towards effects with post-natal exposure. The review by the health effects institute rated the evidence level moderate-high for associations with TRAP. Oxidative stress and neuroinflammation are among proposed mechanisms, but this remains to be elucidated in future studies. The evidence was considered as moderate with emerging evidence.

Attention-Deficit/Hyperactivity Disorder – ADD/ADHD

Attention-deficit disorder, with or without hyperactivity (ADD/ADHD) is marked by an ongoing pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development. ADHD is one of the most common neurodevelopmental disorders of childhood. Overall, results of epidemiological studies regarding associations of ambient air pollution with ADHD and related behaviours showed mixed results. The number of studies is low, and the inconsistent findings result in insufficient or low evidence ratings for an association with air pollution. The qualitative literature review by the HEI panel concluded that there is low evidence of an association of TRAP with ADHD and related behaviours. Currently, the evidence for an association of air pollution with ADHD and related behaviours is low.

5.1.11 Discussion/Synthesis

This review synthesized the existing evidence on the association of air pollution with mental health outcomes based on reviews, meta-analyses and results from original studies from Europe, which often were not included in the reviews due to their more recent publication.

There are clear mechanistic pathways that can explain observed health effects with air pollution. However, the processes involved in adverse mental health outcomes are multifactorial. Various processes affecting brain development, brain structures, connectivity, functionality, changes to neurotransmitters and epigenetic changes to the regulation of such processes in association with exposure to air pollution have been described. Their direct link to clinical mental health outcomes needs to be established more firmly.

The existing studies point towards increased risk of depression with long-term air pollution with most evidence for PM_{2.5} and NO₂. The few studies including noise as a co-pollutant additionally found independent effects of air pollution. The evidence for development of other mental health problems in association with long-term air pollution is more limited or inconsistent. However, it was shown that short-term peaks of exposure were consistently associated with worsening of symptoms of depression

or schizophrenia. Some evidence was also found for increased numbers of suicides or suicide attempts with short-term air pollution.

Despite these not firmly established causal attributions of air pollution to mental health disease, it is clear that even small reductions in pollution associated with often clinically small decreases in depressive symptoms or other mental health outcomes, are likely to make significant population-level contributions to mental health burden given the coverage of exposure.

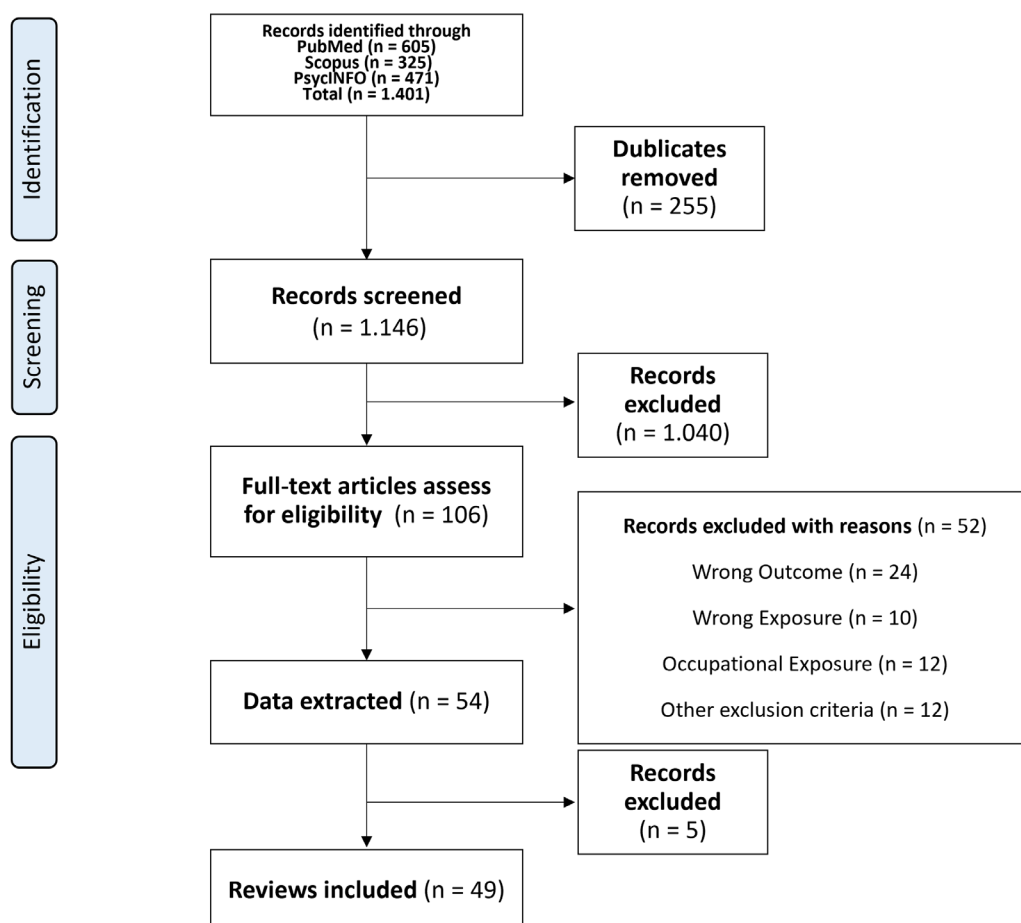
Future research should focus on characterizing long-term consequences and lifetime cumulative effects of exposure in longitudinal studies with rigorous outcome and exposure assessment, that include other environmental exposures to disentangle pollutant mixture effects. Susceptible windows of exposure as well as individual differences in susceptibility should be assessed.

5.2 Environmental chemicals and mental health

5.2.1 Review characteristics

A total of 1,401 review articles were identified in the database searches (PubMed: 605, Scopus: 325, PsycINFO: 471). After removing 255 duplicates, 1,146 reviews were eligible for title and abstract screening. 106 reviews entered the full text review, of which 52 articles were excluded. The main reasons for exclusion were that mental health outcomes were either only briefly addressed or not mentioned at all ($n = 24$), the exposure did not align with the defined criteria for environmental chemicals ($n = 10$), the exposure was assessed exclusively in an occupational context ($n = 12$), or other inclusion criteria were not met ($n = 14$), such as the requirement for human studies or English-language publications. During the data extraction process five reviews were subsequently removed because the link between environmental chemicals and mental health outcome was addressed only insufficiently ($n = 3$), the cited reference did not in fact include any mental health outcomes, despite being presented as such ($n = 1$) or the study population did not meet the inclusion criterion of representing the general population ($n = 1$). No further reviews were identified by other methods (e.g. cross-referencing). This resulted in 49 reviews being included in the analysis. A detailed overview of the screening process is provided in the PRISMA flow diagram (Figure 5.5).

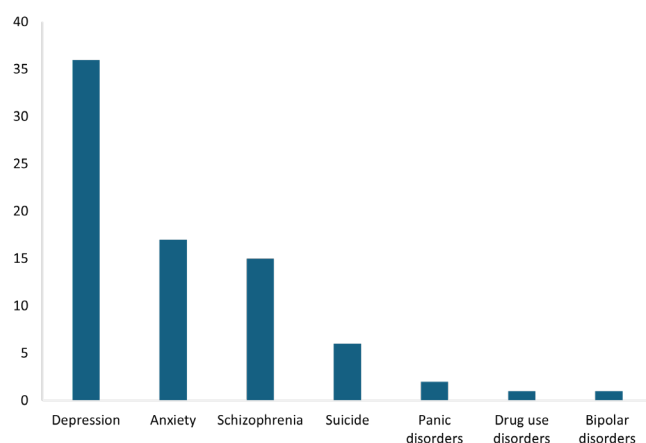
Figure 5.5 PRISMA flow chart for the systematic search and identification of reviews with focus on mental health outcomes in association with environmental chemicals



Notes: Records excluded in full-text screening captured in different criteria do not sum up to 52, because some reviews were excluded due to multiple exclusion criteria.

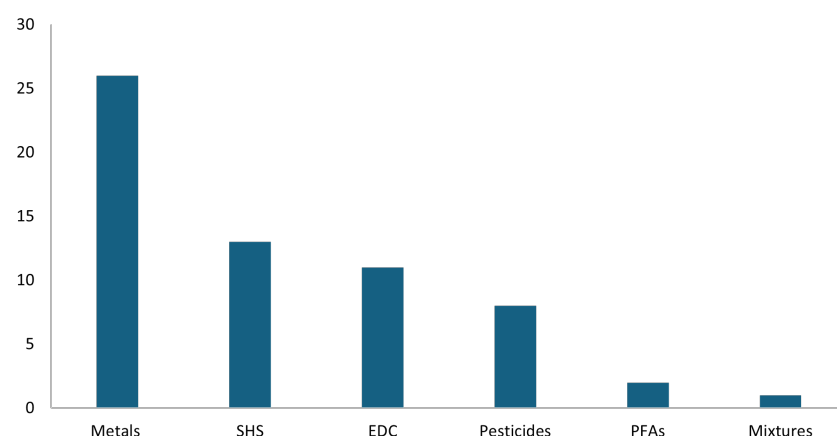
The most frequently examined mental health outcome across the included reviews was depression (n = 36), followed by anxiety (n = 17) and schizophrenia/psychosis (n = 15). Panic disorder (Neuwirth et al., 2020; Vorvolakos et al., 2016) was addressed in two reviews, while bipolar disorder (Vorvolakos et al., 2016) and substance use disorder (Albores-Garcia et al., 2021) were mentioned in one review each. Suicide as an indicator for mental health outcomes was addressed in six reviews (Figure 5.6). We were not able to identify reviews on mental health outcomes such as personality disorder, psychological sexual dysfunction or feeding and eating disorder.

Figure 5.6 Number of reviews included per mental health outcome



With regard to environmental exposures, metals (incl. heavy metals, transition metals, semimetals and alkali metals) were addressed in 26 reviews. Lead, in particular, was addressed in 15 of these, either as a central focus or as one of several exposures examined. Second-hand smoke (SHS) was the subject of 13 reviews, and endocrine-disrupting chemicals (EDCs) were investigated in 11 reviews, among which bisphenol A was the most frequently studied compound (n = 5), followed by phthalates (n = 2). Pesticide exposure was included in 8 reviews. Some exposures were examined only in one or two reviews: PFAS (n = 2) (Jacobson et al., 2022; Suthar et al., 2024) and chemical mixtures (n = 1) (Constantinescu et al., 2025). An overview of exposure distribution is provided in Figure 5.7.

Figure 5.7 Number of reviews included per exposure



Abbreviations:

SHS: Second-hand Smoke; EDC: Endocrine-disrupting chemicals; PFAS: Per- and polyfluoroalkyl substance.

The total number of reviews listed above do not correspond exactly to the number of studies shown in Figure 5.6 and Figure 5.7, as some reviews addressed multiple outcomes and/or exposures.

The included reviews covered the entire search period (2015-2025), and various types of reviews were included: Narrative reviews (n = 25), systematic reviews (n = 9), systematic reviews and meta-analyses (n = 8), meta-analysis (n = 3), scoping reviews (n = 2) and critical reviews (n = 2). When examining the geographic scope reported in the included reviews, it becomes evident that the majority do not specify particular regions, which is partly attributable to their narrative nature. Among those that do, the United States and Japan are the most frequently cited countries. However, European regions — either as a collective entity or as individual countries such as the Netherlands, Spain, Denmark, Sweden, Italy, and France — are also represented across the included reviews.

Most reviews focused on the general population (n = 27), which in some cases encompasses children, adolescents, and adults. Ten reviews specifically addressed children and adolescents, while seven reviews examined environmental chemical exposure in women during sensitive life stages such as pregnancy, postpartum, and menopause.

5.2.2 Second-hand smoke

Biological mechanisms

The scientific literature outlines several potential mechanisms through which SHS may impact mental health, which are discussed in more detail below. The first mechanism involves neurotransmitter and receptor dysregulation. Han et al. (2019) highlighted that nicotine, a primary component of SHS, activates the nicotinic acetylcholine receptors (nAChRs). Because of its role in the central nervous system, this receptor is linked to psychiatric disorders, such as depression or anxiety. In contrast some articles proposed that SHS may inhibit these receptors, potentially disrupting normal neurotransmission (Suzuki et al., 2019). Additionally, SHS has been suspected to alter dopaminergic signalling, resulting in imbalances that are implicated in both depression and anxiety (van der Eijk and Woh, 2023). Christian and Kim (2022) expanded on this by suggesting that chronic exposure to environmental smoke or SHS could reduce the blood concentrations of dopamine and gamma-aminobutyric acid (GABA). These two neurotransmitters are central to mood regulation. These findings collectively point to disrupted neurochemical homeostasis as a plausible mechanism linking SHS to mental health disorders.

The second mechanism of action involves the dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis and the alteration in stress hormone levels. Al-abri et al. (2023) referenced general stress-related hormonal dysregulation as a potential mediator between SHS and adverse psychological outcomes. Zeng and Li (2016) provided more specific insights, reporting that nicotine in SHS activates nAChRs and affects HPA axis function, leading to potentially elevated levels of corticotropin-releasing hormone (CRH) and adrenocorticotrophic hormone (ACTH). Both of which play a central role in the body's response to stress and may contribute to the development of depression and anxiety when chronically dysregulated.

Inflammatory responses triggered by SHS exposure have also been identified as a possible mechanism in the onset of depression. Zhang et al. (2024) described inflammation-related processes wherein SHS may stimulate the production of inflammatory cytokines. These proinflammatory molecules are increasingly recognized as key contributors to the pathophysiology of depression.

The association between SHS exposure and schizophrenia may be partly explained by its effects on neurotransmitter regulation, particularly within the dopaminergic system. Modabbernia et al. (2016) proposed that cadmium, a heavy metal commonly found in tobacco smoke, can release

neurotransmitters, including dopamine and serotonin by altering the metabolism. Disruptions in these systems are widely recognized as contributing factors in the pathophysiology of schizophrenia. Similarly, Hunter et al. (2020) suggested that nicotine, another key compound in SHS, may increase dopamine release, thereby enhancing dopaminergic activity in brain regions implicated in schizophrenia.

Depression

The associations between depression and environmental tobacco smoke (ETS) or SHS were discussed in 13 reviews, mostly as the primary focus, sometimes as part of a broader set of investigated exposures (Al-Abri et al., 2023; Alkhateeb, 2023; Christian and Kim, 2022; Han et al., 2019; Hunter et al., 2020; Jacobson et al., 2022; Modabbernia et al., 2016; Padhani et al., 2024; Suzuki et al., 2019; Theron et al., 2022; van der Eijk and Woh, 2023; Zeng and Li, 2016; Zhang et al., 2024). Ten reviews examined depression, with additional outcomes including anxiety (n = 4) and schizophrenia (n = 2). The reviews synthesized evidence from a range of study designs, mostly cross-sectional studies and cohort-studies. Some reviews focused on the general population (Han et al., 2019; Zeng and Li, 2016; Zhang et al., 2024). Ten reviews addressed specific vulnerable groups, such as pregnant and postpartum women (Al-Abri et al., 2023; Jacobson et al., 2022; Padhani et al., 2024; Suzuki et al., 2019) children and adolescences (Alkhateeb, 2023; Christian and Kim, 2022; Theron et al., 2022; van der Eijk and Woh, 2023) and fetuses in the womb (Hunter et al., 2020; Modabbernia et al., 2016). While most of the reviews regarding metals, EDC and pesticides have a narrative character, for SHS most reviews focusing on depression are systematic reviews and meta-analyses (n = 10). Furthermore, the included literature comprised two narrative reviews and one scoping review. Details of the studies reporting on depression can be found in Table 5.18 (meta-analyses) and Table 5.19 (reviews).

Table 5.18 Included meta-analyses for second-hand smoke exposure and the outcome depression

Author (year)	Outcome	Population	# of studies included in meta-analysis	# of relevant studies ^(a)	# of relevant European studies ^(a)	Results	I ²
Han (2019)	Depressive symptoms	General population	24	24	1	OR = 1.32, (1.25–1.3)	72.6%
Suzuki (2019)	Depression symptoms (prenatal and postnatal)	Pregnant woman	2	2	0	OR = 1.77, (1.12–2.79), p = 0.01	28%
Zeng (2016)	Depressive symptoms	General population	7	7	0	OR = 1.60, (1.35–1.90), p < 0.001	89.6%
Zhang (2024)	Depression	General population	12	7 ^(b)	1	RR = 1.11, (0.87–1.41)	80%

^(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

^(b) 7 studies were relevant for a subgroup analysis regarding indoor air pollution (IAP) from Second-hand smoke. The other 5 studies included in the overall meta-analysis focused on IAP from solid fuel.

Table 5.19 Included reviews for second-hand smoke exposure and the outcome depression

Author (year)	Outcome	Population	Review type	# of studies included in review	# of relevant studies ^(a,b)	Results / Conclusion by the study authors
Al-abri (2023)	Perinatal depression	Pregnant woman	Umbrella	128	1	One study reports significant association between SHS during pregnancy and perinatal depression (OR = 1.77, 95% CI: 1.12–2.79, p = 0.01)
Christian (2022)	Depression	Children and adolescents (4-18 years)	Systematic	8	8	Positive association between depression and SHS at home supported by majority of studies (6/8); more frequent exposure is associated with higher odds of developing depression Inconsistent results for the link between SHS at school and depressive symptoms in the population
Han et al. (2019)	Depressive symptoms	General population	Systematic	24	24	Significant link between SHS and depressive symptoms found; increasing odds for outcome associated with increasing days of exposure; SHS sources make significant differences in odds
Jacobson (2022)	Depression	Pregnant woman	Scoping	15	6	Various studies support link between SHS exposure and depression during and after pregnancy
Padhani (2024)	Antenatal depression	Pregnant woman	Systematic	61	1	One study reports significant association between SHS during pregnancy and antenatal depression (OR = 2.68, 95% CI: 1.73–4.17)
Theron (2022)	Depressive symptoms	Adolescents (10-24 years)	Systematic	17	11	Link between SHS and increased symptoms of depressed mood are supported by several studies; more frequent exposure is associated with more depressive symptoms
Suzuki (2019)	Depression symptoms	Pregnant woman	Systematic	6	2	Association found for SHS exposure and depressive symptoms during and after pregnancy
Van der Eijk (2023)	Depressive symptoms	General population	Narrative	77	32	Positive association between SHS and depressive symptoms in adults and vulnerable groups (children and adolescents, pregnant woman)
Zhang (2024)	Depression	General population	Systematic	12	7	When considering indoor air pollution from SHS evidence is inconsistent regarding significant relationship (previous meta-analyses reported significance)

^(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

^(b) For narrative reviews, the reported number of included studies may vary, as it is mostly not always clearly specified how many studies were considered.

Across all reviews, SHS exposure was consistently associated with depression, anxiety and schizophrenia. Several reviews (n = 9) reported significant positive associations, while others (n = 4) described positive associations or indicative trends.

Han et al. (2019), Zhang and colleagues (2024) and Zeng and Li (2016) identified positive associations between SHS exposure and depression for the general population. The meta-analysis by Zhang et al. (2024) focussed on indoor air pollution (IAP), including SHS. However, since their results did not differentiate between IAP from SHS and that from fuel combustion, the findings are of limited relevance for the specific scope of this review. Still, when isolating the SHS data, the authors reported an 11% increased risk of depression associated with SHS exposure. This result, however, was not statistically significant (RR = 1.11, 95% CI: 0.87–1.41, I^2 = 80%). In contrast Han et al. (2019) found a statistically significant association between SHS exposure and depressive symptoms in the general population (OR = 1.32, 95% CI: 1.25–1.3). They also identified a dose-response relationship, reporting that each additional day of SHS exposure was associated with 57 % increased odds for depressive symptoms (OR = 1.57, 95% CI: 1.26–1.87). Regarding exposure measurement quantified by hours, the authors described a linear relationship (p-value for linear trend < 0.001).

Han et al. (2019) further conducted several subgroup analyses. These showed no statistically significant differences by sex (males: OR = 1.00, 95% CI: 0.97–1.03; females: OR = 1.22, 95% CI: 0.87–1.56, p-value for differences between subgroups = 0.21) or age-group (adults: OR = 1.39, 95% CI: 1.21–1.56; adolescents: OR = 1.24, 95% CI: 0.74–1.75, p-value = 0.58). However, significant differences emerged based on the source of SHS exposure. Specifically, exposure on campuses, in public places, and the workplace showed significant associations with depressive symptoms, whereas exposure at home did not (p-value for differences between subgroups = 0.019). Additionally, Han et al. (2019) addressed potential confounders and emphasized the importance of adjusting for the variables such as social support and negative life events. Their findings indicated that not accounting for these factors could lead to underestimation of the true relationship between SHS exposure and depressive symptoms. Zeng and Li (2016) also reported a significant association between SHS exposure and depressive symptoms, with an odds ratio of 1.60 (95% CI: 1.35–1.90), which was also cited by van der Eijk and Woh (2023).

Jacobson et al. (2022), Suzuki et al. (2019), Padhani et al. (2024) and Al-abri et al. (2023) reported findings on the association between SHS exposure during pregnancy and perinatal depression in pregnant women. In their scoping review, Jacobson and colleagues (2022) reported that pregnant woman who did not actively smoke but were exposed to SHS had a significantly higher chance for developing postpartum depression (OR = 1.30, 95 % CI: 1.03–1.64). Another study reported that pregnant women exposed to SHS had more than twice the odds of developing severe depressive symptoms compared to those not exposed (OR = 2.22, 95% CI: 1.12–4.39), whereas no significant association was found for moderate depressive symptoms (OR = 1.05, 95% CI: 0.77–1.43). Similarly, Suzuki et al. (2019) reported findings on the associations between perinatal depression and SHS exposure, differentiating between the pregnancy and postpartum periods. Their meta-analysis of two studies found a significant positive association between postpartum depressive symptoms and SHS exposure (OR = 1.77, 95% CI: 1.12–2.79, p-value = 0.01, I^2 = 28%). Al-abri et al. (2023) also cited this meta-analysis in their review. In addition to depression, Suzuki et al. (2019) also examined suicidal ideation as a predictor of adverse mental health outcomes following childbirth, reporting a significant positive association with SHS exposure (OR = 1.75, 95% CI: 1.14–2.70, p-value = 0.01, I^2 = 51%). Since the perinatal period includes the postpartum phase, the authors also looked at SHS exposure during pregnancy among non-smoking women and its relationship with postpartum depression (PPD). Compared to individuals with no SHS exposure, a significant association was observed (OR = 1.30, 95 % CI: 1.03–1.64). Furthermore, a longitudinal study revealed that depressive symptoms increased up to

one month postpartum; however, at the six-month postpartum follow-up, no significant association between prenatal SHS exposure and depressive symptoms was observed (regression coefficient (RC) = 0.9, 95% CI: -0.4–2.2). Padhani et al. (2024) also reported SHS during pregnancy as a significant contributor to antenatal depression (OR = 2.68, 95% CI: 1.73–4.17) based on one study in their review.

Three reviews focussed on children and adolescences and the effect of SHS on their mental health (Christian and Kim, 2022; Theron et al., 2022; van der Eijk and Woh, 2023). In the review by Theron et al. (2022), SHS exposure was positively associated with an increase in depressed mood among adolescents. Additionally, a dose-response relationship was reported, with more frequent exposure associated with increased depressive symptoms. van der Eijk and Woh (2023) also reported positive associations between SHS exposure and depressive symptoms, not only in children, but also in the general population. Among the studies cited a longitudinal study indicated that SHS exposure among children and adults is associated with depression and panic attacks. Specifically for children, van der Eijk and Woh (2023) referenced a follow-up study in which the adjusted hazard ratio for suicide was 2.83 among students aged eleven to 16, who were exposed to SHS of more than 20 cigarettes per day. In this context, suicide may be considered an outcome and also a potential indicator of mental health disorders.

Another review focusing on children and adolescents is by Christian and Kim (2022). The relevant population included individuals aged four to 18 years. The majority of the studies cited in this systematic review (six out of eight) reported a positive association between SHS exposure at home and depression. One study reported that children exposed to SHS for more than one hour per day had 2.73 times higher odds of experiencing mental disorders, while those exposed for less than one hour per day had 1.49 times higher odds compared to no exposure. Another study found a significant positive association between depression and SHS exposure for one to four days per week (OR = 1.22, 95% CI: 1.17–1.27), with an even stronger association for exposure on higher or equal to five days per week (OR = 1.36, 95% CI: 1.29–1.43). A further study reported that students exposed to SHS had 1.66 times higher odds of experiencing depressive symptoms (95% CI: 1.26–2.18). However, Christian and Kim (2022) also reported inconsistent findings. One study found a significant association only among male adolescents (OR = 4.43, 95% CI: 1.52–2.90), but not among females (OR = 1.70, 95% CI: 0.67–4.43). Another study conducted among Korean high school students found no significant relationship between depressive symptoms and SHS exposure at school. Overall, the majority of reviews included report a positive and often significant association between SHS exposure and depression, both in adults and in children and adolescents.

Other mental health outcomes

Anxiety

In addition to depression, several reviews also examined the evidence regarding SHS exposure and anxiety. However, considerably fewer reviews are available. van der Eijk and Woh (2023) as well as Alkhateeb (2023) primarily focused on children, in some cases also on the general population, whereas Suzuki et al. (2019) and Jacobson et al. (2022) centre their analysis (as with their focus on depression) on pregnant women. Details of the studies reporting on anxiety can be found in Table 5.20.

Alkhateeb (2023) based on findings from Sabbagh et al. (2021), reported a significant association between SHS exposure and anxiety in children ($B = 12.16$, $SE = 3.82$, $\beta = 0.172$, $t = 3.18$, $p\text{-value} = 0.002$). Similarly, van der Eijk and Woh (2023) cited a study showing that children aged six to eleven, who were exposed to SHS at home had 80 % higher odds of being diagnosed with anxiety ($OR = 1.80$). They also reported an association between SHS exposure and increased likelihood of clinically diagnosed anxiety disorders among non-smoking adults.

As already noted in the section on depression, Suzuki et al. (2019) and Jacobson et al. (2022) focus on women during and after pregnancy in their reviews. Regarding anxiety in the perinatal period, the findings are inconsistent. One study in Suzuki et al. (2019)'s review showed a significant association between perinatal anxiety and SHS exposure (perinatal period: regression coefficient (RC) = 2.2, 95% CI: 0.3–4.2), while another study found a positive trend but no statistically significant association ($OR = 0.88$, 95% CI: 0.25–3.14). Jacobson et al. (2022) also cited studies linking SHS exposure to an increased risk of anxiety.

Overall, the literature demonstrates a consistent association between SHS exposure and anxiety in children, whereas findings for pregnant women are statistically inconsistent, although they generally indicate a trend toward an association.

Schizophrenia

Two reviews (Hunter et al., 2020; Modabbernia et al., 2016) examined the mental health endpoint schizophrenia and specifically focused on SHS exposure in utero, i.e., the effects of maternal smoking on the unborn child. Details of the studies reporting on schizophrenia can be found in Table 5.21 (meta-analyses) and Table 5.20 (reviews). Both reviews consistently reported a positive association between prenatal SHS exposure and an increased risk of developing schizophrenia later in life. The pooled analysis by Hunter et al. (2020) reported a relative risk of 1.29 (95% CI: 1.10–1.51, $I^2 = 71\%$) based on follow-up periods ranging from eleven to 31 years. Similarly, Modabbernia et al. (2016) cited a study (Stathopoulou et al., 2013), that found a statistically significant association between maternal smoking during pregnancy and risk of developing schizophrenia ($B = 0.78$, $SE = 0.26$, $OR = 2.19$, 95%CI: 1.32–3.64, $p\text{-value} = 0.003$) and additionally reported that maternal smoking was significantly associated with more severe schizophrenia symptoms in affected individuals ($B = 0.199$, 95% CI: 0.26–3.73, $p\text{-value} = 0.024$). Although this outcome was only addressed in a limited number of reviews, it remains noteworthy due to the potential link between maternal smoking during pregnancy and the development of schizophrenia later in life.

Table 5.20 Included reviews for second-hand smoke exposure and the outcomes anxiety, schizophrenia, suicide and panic attack

Author (year)	Outcome	Population	Review type	# of studies included in review	# of relevant studies ^(a,b)	Results / Conclusion by the study authors
Alkhateeb (2023)	Anxiety	Children and adolescents (1-15 years)	Systematic	13	1	One study reported significant positive link between SHS exposure and anxiety in children ($p = 0.002$)
Jacobson (2022)	Anxiety	Pregnant woman	Scoping	15	2	Various studies support link between SHS exposure and anxiety during and after pregnancy
Suzuki (2019)	Anxiety	Pregnant woman	Systematic	6	2	Studies found inconsistent data on the link between SHS exposure and perinatal anxiety (RC = 2.2, 95% CI: 0.3–4.2; AORs = 0.88, 95% CI: 0.25–3.14)
Van der Eijk (2023)	Anxiety	General population	Narrative	77	6	Positive association between SHS and anxiety supported by multiple studies
Hunter (2020)	Schizophrenia	Prenatal exposure	Systematic	12	7	Prenatal tobacco smoke exposure as a risk factor for schizophrenia
Modabberniaa (2016)	Schizophrenia	Children (Prenatal exposure)	Narrative	nan	1	Exposure in prenatal phase associated with increased risk of schizophrenia for the child
Suzuki (2019)	Suicide ideation	Pregnant woman	Systematic	6	2	Higher odds for antenatal suicidal ideation from SHS exposure found in non-smokers compared to active smokers
Van der Eijk (2023)	Suicide	Adolescents (11-16 years)	Narrative	12	9	Heavy SHS exposure increases the risk to commit suicide in adolescents (HR = 2.83)
Van der Eijk (2023)	Panic attack	General population	Narrative	12	1	One study reported positive link between chronic exposure to SHS and panic attacks

^(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

^(b) For narrative reviews, the reported number of included studies may vary, as it is mostly not always clearly specified how many studies were considered.

Table 5.21 Included meta-analyses for second-hand smoke exposure and the outcomes schizophrenia and suicide ideation

Author (year)	Outcome	Population	# of studies included in meta-analysis	# of relevant studies ^(a)	# of relevant European studies ^(a)	Results	I ²
Hunter (2020)	Schizophrenia	Prenatal exposure	7	7	7	RR = 1.29, (1.10–1.51)	71%
Suzuki (2019)	Antenatal suicide ideation	Pregnant woman	2	2	0	OR = 1.75, (1.14– 2.70), p = 0.01	51%

^(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

During the analysis of the reviews, several limitations repeatedly occurred, which were acknowledged by the authors. The authors criticized the lack of evidence for causal relationships and emphasized the need for more longitudinal studies. This is also accompanied by the frequent use of cross-sectional study designs, which inherently limits the ability to establish causality (Christian and Kim, 2022; Han et al., 2019; Hunter et al., 2020; Theron et al., 2022; van der Eijk and Woh, 2023).

Another frequently mentioned limitation is the heterogeneity of methodological approaches in the reviewed human epidemiological studies, for example in the assessment of mental health outcomes (Han et al., 2019; Jacobson et al., 2022). Especially for SHS the exposure is often assessed by self-reporting questionnaires, which are susceptible to bias. Such methods may not accurately capture the actual level of exposure (Christian and Kim, 2022; Han et al., 2019; Hunter et al., 2020; Jacobson et al., 2022; Theron et al., 2022; van der Eijk and Woh, 2023). Han et al. (2019) addressed this issue in their review and demonstrated a significant discrepancy between self-reported SHS exposure and exposure measured using biomarkers, with associations being stronger for self-reported data, thereby potentially overestimating the adverse relationship. The quality and quantity of the available studies have also been repeatedly criticized (Alkhateeb, 2023; Hunter et al., 2020; Modabbernia et al., 2016; Padhani et al., 2024; Suzuki et al., 2019; Theron et al., 2022). Finally, confounding remains a major concern. Several authors noted that consistent adjustment for confounders is difficult, especially when not all included studies report them. Moreover, some reviews emphasized the possibility of residual or unmeasured confounding (Christian and Kim, 2022; Hunter et al., 2020; Modabbernia et al., 2016; van der Eijk and Woh, 2023).

5.2.3 Metals

Biological mechanisms

Heavy Metals

In the scientific literature, several biological mechanisms involving metals and their effect on mental health have been reported. This section provides an overview of the most frequently described pathways, as well as mechanisms that have been reported only infrequently. For heavy metals, three main mechanisms were consistently mentioned, alongside a small number of additional, less commonly described processes. Heavy metals, such as lead (Pb), cadmium (Cd) and mercury (Hg) can interfere with neurotransmitter systems and receptors functions. Both Hg and Pb can affect the glutamatergic system by inducing either overactivation or hypofunction of the N-methyl-D-aspartate (NMDA) receptor. This can contribute to mental health outcomes, such as depression and schizophrenia (Albores-Garcia et al., 2021; Attademo et al., 2017; Hollander et al., 2020; Luz et al., 2021; Modabbernia et al., 2016; Ordemann and Austin, 2016; Zaks et al., 2021). For Pb specifically, exposure has been suggested to result in the loss of GABAergic interneurons (Albores-Garcia et al., 2021; Hollander et al., 2020; Zaks et al., 2021). The dopaminergic and serotonergic systems play a major role in mood disorders. Therefore, their disruption can cause severe adverse mental health outcomes. Both Pb and Cd have been shown to disturb these systems and their metabolism, contributing to schizophrenia, depression and anxiety (Albores-Garcia et al., 2021; Hollander et al., 2020; Modabbernia et al., 2016; Vorvolakos et al., 2016; Zaks et al., 2021). The second frequently cited mechanism involves oxidative stress and neuroinflammation. All three heavy metals mentioned in this umbrella+ review (Pb, Cd and Hg) can cause an increase in oxidative stress, neuronal damage, mitochondrial dysfunction or neuroinflammation (Luz et al., 2021; Tota et al., 2024; Van Den Bosch and Meyer-Lindenberg, 2019; Ventriglio et al., 2021).

Another mechanism mentioned involves structural changes of the brain, after exposure to Pb. Tota et al. (2024) and Vorvolakos et al. (2016) reported a reduction of cortical volume and white matter, specifically in regions involved in emotional and cognitive processing. In addition to the more frequently discussed pathways, several other mechanisms were sporadically reported in the literature

linking heavy metal exposure to mental health outcomes. Some authors noted that Pb may disrupt stress-regulatory systems in the brain, interfering with hormonal feedback loops that control the body's stress response (Vorvolakos et al., 2016). In the case of methylmercury (MeHg), the literature suggests a reduction in proteins essential for neuron maintenance and the adaptability of neural connections, which could undermine brain resilience (Luz et al., 2021). Other findings point to alterations in the gut microbiota caused by environmental contaminants (Hollander et al., 2020). Finally, Pb exposure has been linked in one review to changes in the brain's reward circuitry that could increase the vulnerability to develop substance use problems (Albores-Garcia et al., 2021).

Other metals

Similar mechanisms of action have been reported for other metals discussed in the literature. In these cases, neurotransmitter systems appear to play a central role. Copper (Cu), manganese (Mn), zinc (Zn), and magnesium (Mg) may interfere with glutamatergic, dopaminergic, serotonergic, monoaminergic, and GABAergic pathways, all of which are involved in the potential development of mental health outcomes such as depression, schizophrenia, and anxiety (Młyniec et al., 2015; Modabbernia et al., 2016; Ni et al., 2018; Phelan et al., 2018; Scassellati et al., 2020; Tarnacka et al., 2021; Zaks et al., 2021). Examples include dysregulation of the N-methyl-D-aspartate (NMDA) receptor within the glutamatergic system or disruption of hypothalamic–pituitary–adrenal (HPA) axis function (Ni et al., 2018; Zaks et al., 2021). Oxidative stress and neuroinflammation are recurrently involved in the mechanisms of action for metals. Cu, Mn, Fe and Zn have all been reported to induce oxidative stress, which may subsequently trigger neuroinflammatory processes and contribute to neurodegeneration. These pathophysiological cascades are also involved in the onset and progression of mental health disorders (Młyniec et al., 2015; Modabbernia et al., 2016; Ni et al., 2018; Scassellati et al., 2020; Tarnacka et al., 2021; Zaks et al., 2021). Additionally, Cu exposure has been identified as a potential driver of cytokine dysregulation, which can further disrupt neurotransmitter homeostasis (Ni et al., 2018). In addition to the more frequently discussed pathways, several other mechanisms were sporadically reported in the literature linking metal exposure to mental health outcomes. For example, Cu has been suspected to play a role in the dysregulation of neurotrophic factors, particularly brain-derived neurotrophic factor and nerve growth factor, contributing to the pathophysiology of mental health disorders (Ni et al., 2018). Exposure to Mn and Fe has been linked to neuronal loss and apoptosis, potentially through oxidative stress–mediated pathways. In the case of Mn, excessive glutamatergic activity can cause abnormal cortical organization and excitotoxic injury, contributing to cognitive and emotional dysfunction (Modabbernia et al., 2016; Zaks et al., 2021).

Depression, anxiety and panic disorder

Mental Health effects of metals in general were discussed in 22 reviews, either as the primary focus or as part of a broader set of investigated exposures (Albores-Garcia et al., 2021; Attademo et al., 2017; Heyer and Meredith, 2017; Hollander et al., 2020; Jacobson et al., 2022; Luz et al., 2021; Mesnil et al., 2020; Młyniec et al., 2015; Modabbernia et al., 2016; Neuwirth et al., 2020; Ni et al., 2018; Ordemann and Austin, 2016; Phelan et al., 2018; Scassellati et al., 2020; Schmitt et al., 2021; Tarnacka et al., 2021; Theron et al., 2022; Tota et al., 2024; Van Den Bosch and Meyer-Lindenberg, 2019; Ventriglio et al., 2021; Vorvolakos et al., 2016; Zaks et al., 2021). The difference between the 26 reviews on metals mentioned in Section X is due to the postponement of the reviews on lithium and selenium. These two metals are discussed in more detail in the discussion section. The 22 reviews include insights on heavy metals (Lead (Pb): n = 15; Cadmium (Cd): n = 3; Mercury (Hg): n = 3; Tin (Sn): n = 1), transition metals (Manganese (Mn): n = 7; Copper (Cu): n = 6; Iron (Fe): n = 2; Zinc (Zn): n = 2), semimetals (Arsenic (As): n = 3) and alkali metals (Magnesium (Mg): n = 2; Nitrate (NO₃): n = 1). Depression was the most frequently discussed outcome in relation to metal exposure across the included reviews (n = 24), followed by schizophrenia (n = 23). Less commonly reported outcomes included anxiety (n = 2: Hollander et al., 2020; Luz et al., 2021), panic disorder (n = 2: Neuwirth et al. (2020); Vorvolakos et al. (2016)), bipolar disorder and manic disorder (n = 1: Vorvolakos et al. (2016)), and suicide as an indicator

for mental illness (n = 1: Tota et al. (2024)). As noted previously in section 5.2.1, these numbers may differ from those reported above, as many reviews examined multiple exposures and outcomes simultaneously. The majority of reviews on the topic of metals were narrative in nature (n = 15). Only a few systematic reviews (n = 3), systematic reviews combined with a meta-analysis (n = 2), scoping reviews (n = 1), or critical reviews (n = 1) were identified. Providing exact numbers regarding the populations studied is challenging, as many reviews refer broadly to the general population while also explicitly highlighting vulnerable subgroups. In general, most reviews focused on the general population, occasionally including children and adolescents. There was also a notable focus on pregnant women and early-life exposure. The reviews synthesized evidence from a range of study designs, including mostly cross-sectional studies, cohort-studies and some longitudinal studies. Details of the studies reporting on depression, anxiety and panic disorder can be found in Table 5.22 (meta-analyses) and Table 5.23 (reviews).

Table 5.22 Included meta-analyses for Cu and Mg²⁺ exposure and the outcome depression

Author (year)	Pollutant	Outcome	Population	# of studies included in meta-analysis	# of relevant studies ^(a)	# of relevant European studies ^(a)	Results	I ²
Ni (2018)	Cu	Depression	General population	21	21	6	Blood copper: WMD = 1.985, (0.642–3.329), p = 0.004 Hair copper: WMD = -0.46, (-3.97–3.04), p = 0.795	96.4% 97.9%
Phelan (2018)	Mg ²⁺	Depression	General population	58	14 ^(b)	6	Cross-sectional (highest versus lowest category): OR = 0.66, (0.51–0.81), p < 0.01, k = 12 Longitudinal (incidence new onset): OR = 0.71, (0.40–1.02), p = 0.10, k = 2	95.2% 4.2%

Abbreviations: Cu = Copper, Mg = Magnesium.

^(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

^(b) The 44 additional studies addressed general mood disorders rather than depression.

Table 5.23 Included reviews for metal exposure and the outcomes depression, anxiety and panic disorder

Author (year)	Pollutant	Outcome	Population	Review type	# of studies included in review	# of relevant studies ^(a,b)	Results / Conclusion by the study authors
Hollander (2020)	Pb	Depressive symptoms	General population	Narrative	n.a.	1	Each 1 µg/dL increase in childhood blood lead linked to 0.9-point increase in depression symptom scale (Adolescents)
Jacobson (2022)	Pb, Cd, Mn, As	Depression	Pregnant woman	Scoping	15	4	Mn blood levels in late trimesters significantly positive associated with PPD No association found for Pb and Cd Arsenic urine concentrations during pregnancy associated with PPD in women >25 years, showing an inverse link without, but a positive link with, a history of depression
Luz (2021)	Hg	Depressive Symptoms	General population	Narrative	n.a.	7	Inconsistent evidence for link between Hg exposure and depression
Mesnil (2020)	Mn, Sn, Cd, Pb, Hg	Depression	General population	Narrative	n.a.	7	Exposure to metals linked to increased risk of depression or depressive symptoms
Młyniec (2015)	Cu, Mn	Depression	General population	Narrative	n.a.	16	Significant increase in Cu blood levels in depressed patients (some inconsistencies)
Neuwirth (2020)	Pb	Major depressive disorder	Children and young adults	Narrative	79	2	Increased risk for MDD (+2.3x) in children and young adults for BLL of 1.24 µg/dL, Pb exposure in children linked to greater frequency of MDD
Ni (2018)	Cu	Depression	General population	Systematic	21	21	Significant association between copper blood level and depressed patients, significant for mean age < 50, non-significant for mean age > 50, copper in hair samples may not reflect short-term exposure
Phelan (2018)	Mg	Depression	General population	Systematic	58	58	Contradicting results: Protective effect of dietary Mg ²⁺ intake on depression reported, average higher Mg blood concentration in depressed patients, antidepressants potential driver
Schmitt (2021)	As	Depression	General population	Systematic	41	1	Association between higher levels of arsenic in private well water and higher levels of depression (r = 0.26), more reported depression in residence living in areas with more arsenic in water

Author (year)	Pollutant	Outcome	Population	Review type	# of studies included in review	# of relevant studies ^(a,b)	Results / Conclusion by the study authors
Tarnacka (2021)	Cu, Fe, Mn	Depression	General population	Narrative	n.a.	6	Suggestive link between Cu and depression, one study reported negative correlation between Fe and depression, contradicting results for Mn regarding association with depression (positive/negative link)
Theron (2022)	Pb, As	Depression	Adolescents (10-24 years)	Systematic	17	2	Pb and As exposure through water intake associated with higher depressive symptoms
Van den Bosch (2019)	Pb, Cd, Hg	Depression	General population	Narrative	193	16	Lead and mercury exposure linked to depression, evidence for cadmium inconclusive
Vorvolakos (2016)	Pb	Depression	General population	Narrative	n.a.	4	Lead was found to be increased in depressed patients ($p < 0.01$) Significant increase in risk of MDD found for higher BLL, but no association between BLL and depression
Hollander (2020)	Pb	Anxiety	General population	Narrative	n.a.	1	Each 1 µg/dL increase in childhood blood lead linked to 0.9-point increase in anxiety symptom scale (Adolescents)
Luz (2021)	Hg	Anxiety	General population	Narrative	n.a.	4	Evidence from prospective pregnancy/birth cohort study linking Hg exposure to anxiety in children
Neuwirth (2020)	Pb	Panic disorder	Children and young adults	Narrative	79	2	Increased risk for panic disorder (+4.9x) in children and young adults for BLL of 1.24 µg/dL, Pb exposure in children linked to greater frequency of panic attacks
Vorvolakos (2016)	Pb	Panic disorder	General population	Narrative	n.a.	1	One significant association between increased BLL and higher risk of panic disorder in young adults

Abbreviations: As = Arsenic, Cd = cadmium; Cu = Copper, Hg = Mercury, Fe = Iron, Mg = Magnesium, Mn = Manganese, Sn = Tin, Pb = Lead.

^(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

^(b) For narrative reviews, the reported number of included studies may vary, as it is mostly not always clearly specified how many studies were considered.

Heavy Metals

Findings related to heavy metals vary in consistency depending on the specific metal. The largest number of studies is available on lead (Pb) exposure, particularly in relation to depression, anxiety, and panic disorder. Neuwirth et al. (2020), and Hollander et al. (2020), to some extent, van den Bosch and Meyer-Lindenberg (2019), as well as Vorvolakos et al. (2016) focused on children, adolescents, and young adults as vulnerable populations.

Neuwirth et al. (2020) and Hollander et al. (2020) described an increased risk of developing major depressive disorder or depressive symptoms in adulthood following childhood exposure to lead. Hollander et al. (2020) reported a 0.9-point increase in depression symptom scores for every 1 µg/dL increase in childhood blood lead levels (BLL). In addition, Neuwirth et al. (2020) reported a 4.9-fold increased risk of panic disorder associated with a BLL of 1.24 µg/dL, while Hollander et al. (2020) described a positive association with anxiety symptoms (Mesnil et al., 2020) following childhood lead exposure. Neuwirth et al. (2020) also reported a 2.3-fold increased risk of major depressive disorder at a BLL of 1.24 µg/dL in young adults (20 to 39 years old). Similarly, van den Bosch and Meyer-Lindenberg (2019) noted a positive association of lead exposure on depression, particularly during vulnerable developmental periods such as childhood. However, this review also presented some of the first inconsistent findings. While several studies supported the assumed association, one longitudinal study included in the review found no significant relationship between lead exposure and later mental health outcomes (Van Den Bosch and Meyer-Lindenberg, 2019). Vorvolakos et al. (2016) also cited studies that confirmed a positive association between long-term lead exposure and an increased risk of depression. However, they also included one study that did not confirm the significant association when measured via blood lead levels (BLL). Theron et al. (2022) reported a positive association between Pb exposure via water contamination and depressive symptoms. Albores-Garcia et al. (2021) and Mesnil et al. (2020) also briefly mentioned a positive association between lead exposure and depression, but did not discuss the findings in detail.

Mercury (Hg), cadmium (Cd) and tin (Sn) as other heavy metals were also examined in some reviews. With a few exceptions, the overall findings are relatively consistent. Mesnil et al. (2020) reported in their review a positive association between urinary Hg, Cd and Sn concentrations and depression. van den Bosch and Meyer-Lindenberg (2019) referred to Hg as having 'neurotoxic effects' (Van Den Bosch and Meyer-Lindenberg, 2019) that can be linked to depression. However, they take a more cautious stance regarding Cd, reporting inconsistent findings. With regard to Hg, Luz et al. (2021) reported one study that also support a positive association between Hg levels in hair and depressive symptoms, in contrast to another study that found no significant difference between depressive symptoms in people with higher ($\geq 10 \mu\text{g g}^{-1}$) or lower ($< 10 \mu\text{g g}^{-1}$) total mercury concentrations in hair. Based on these mixed findings, the authors concluded that depressive symptoms are likely not directly caused by mercury exposure. As mentioned frequently, another suggestion is that exposure to Hg (here specifically MeHg) in early childhood leads to an increased risk of depressive disorders in adulthood (Luz et al., 2021). Luz et al. (2021) also addressed anxiety as an outcome in their review and reported a positive correlation between maternal Hg exposure during pregnancy and anxiety in children. This association was identified in a prospective pregnancy/birth cohort study and was supported by additional studies cited in the review.

Overall, the literature on heavy metal exposure and depression indicates a particularly strong association for Pb, with childhood and prenatal exposure linked to an increased risk of depression later in life. However, some reviews have reported contradictory findings. For Cd, the evidence remains limited. While some reviews suggest a potential association between Hg exposure and depression, for instance based on findings from a pregnancy/birth cohort study, other authors did not find evidence supporting this link.

Transition Metals

Transition metals, including manganese (Mn), copper (Cu) and iron (Fe), were also discussed in some reviews. Tarnacka et al. (2021) report findings on all three elements. Regarding Fe, only one result was cited, indicating a negative correlation between Fe blood levels and depression. Suggesting that lower Fe concentrations may be associated with an increased risk of depression. For Mn and Cu the findings across reviews are inconclusive. Tarnacka et al. (2021) described a negative association between dietary Mn intake and depressive symptoms in children and pregnant women, while Mesnil et al. (2020) reported a positive association between urinary Mn levels and depressive symptoms in the general population. In addition, Jacobson et al. (2022) found a positive relationship between blood Mn levels in the third trimester ($\beta = 0.13$, 95% CI: 0.04–0.21) as well as the average across second and third trimester ($\beta = 0.14$, 95% CI: 0.02–0.26) and postpartum depressive symptoms, measured with the Edinburgh Postnatal Depression Scale (EPDS), 12 months postpartum. But, when PPD was analysed as a binary variable (EPDS ≥ 13), the association was not statistically significant. In terms of Cu exposure, both Tarnacka et al. (2021) and Młyniec et al. (2015) suggested that a positive association between Cu and depression may exist. Młyniec et al. (2015) referred to several studies reporting elevated Cu levels in individuals with depression, although other findings fail to confirm such a relationship. Ni et al. (2018) further supported the positive association reported by Młyniec et al. (2015), as they found a statistically significant relationship between elevated blood Cu levels and depression (weighted mean difference (WMD) = 1.985, 95% CI: 0.642–3.329, p-value = 0.004). Additionally, the authors identified age as a relevant mediating factor. In populations with a mean age below 50 years, the association was stronger and statistically significant (WMD = 2.941, 95% CI: 1.603–4.279, p < 0.001), whereas in populations over 50 years of age, the effect was not statistically significant (WMD = 0.956, 95% CI: -0.292–2.203, p-value = 0.133). The authors hypothesized that with increasing age, blood Cu concentrations may decline, potentially influencing the observed association. Ni et al. (2018) reported another noteworthy finding: while blood Cu concentrations showed a significant positive association with depression, Cu exposure measured in hair samples did not show a statistically significant association (WMD = -0.463, 95% CI: -3.966–3.039, p-value = 0.795). The authors offered a possible explanation for this discrepancy, suggesting that Cu levels in hair primarily reflect long-term exposure, whereas short-term changes are not adequately captured through hair analysis.

Overall, the literature on transition metals remains inconclusive. For Fe, the evidence is very limited. Findings regarding Mn are inconsistent both in terms of the presence and the direction of the association with depression. Similarly, results for Cu are contradictory: while some studies reported an association with depression, others did not. These discrepancies may partly be explained by differences in the biological matrices analysed.

Semimetals and Alkali Metals

The only semimetal discussed in the reviewed literature is arsenic. Schmitt et al. (2021) cited a study by Zierold et al. (2004) that found higher arsenic concentrations in private well water to be associated with increased rates of depression ($r = 0.26$). A significant difference in depression prevalence was observed between individuals exposed to arsenic concentrations between 2 $\mu\text{g/L}$ and 10 $\mu\text{g/L}$ and those exposed to levels below 2 $\mu\text{g/L}$ (OR = 2.74, 95% CI: 1.14–6.63). Theron et al. (2022) reported similar findings for both arsenic and nitrates. Here, too, a positive association between arsenic- and nitrate-contaminated water and depressive symptoms is reported.

Phelan et al. (2018) examined the role of magnesium (Mg^{2+}), with a particular focus on dietary intake. In contrast to the other alkali metal NO_3 , they reported a significant negative association between dietary Mg^{2+} intake and the prevalence of depression based on cross-sectional data (p-value < 0.01). However, this association did not remain statistically significant in longitudinal studies, although the trend persisted (OR = 0.71, 95% CI: 0.40–1.02, p-value = 0.10). When examining Mg^{2+} concentrations in body fluids, patients with mood disorders were found to have significantly higher Mg^{2+} levels than

individuals without such disorders. The authors, however, noted that medication use may be a relevant confounding factor, as no significant differences in Mg^{2+} levels were observed in patients who were not taking psychotropic medication in contrast to non-patients.

Schizophrenia and psychosis

Heavy Metals

Several reviews identified heavy metals, especially lead (Pb) and cadmium (Cd), as potential contributing factors for schizophrenia and psychosis. Ventriglio et al. (2021), Attademo et al. (2017) and Tota et al. (2024) briefly reported a potential positive association between heavy metal exposure and the risk of developing schizophrenia and psychosis. Especially early life exposure to lead was highlighted as a factor for psychosis risk and a disruption of the dopaminergic and glutamatergic neurotransmission as a mechanism of action (Ventriglio et al., 2021). Among the few reviews addressing cadmium, Modabbernia et al. (2016) reported an association between Cd and schizophrenia, citing studies that found elevated cadmium concentrations in hair samples of individuals with schizophrenia. However, the majority of the literature focused on lead (Pb) exposure and its association with schizophrenia and psychosis. Two key studies Opler et al. by (2004, 2008) (Opler et al., 2004, 2008) were cited in six different reviews. Modabbernia et al. (2016), Vorvolakos et al. (2016), Heyer and Meredith (2017), Ordemann and Austin (2016), Zaks et al. (2021) and Hollander et al. (2020) highlighted one or both studies by Opler et al. in their reviews (2004, 2008). The studies (Opler et al., 2004, 2008) examined the association between prenatal lead exposure measured via δ -aminolevulinic acid (δ -ALA) levels in archived maternal serum samples and the risk of developing schizophrenia in adulthood. Both studies found positive associations between maternal δ -ALA levels and later schizophrenia diagnosis, with reported odds ratios of 1.83 (95% CI: 0.87–3.87) (Opler et al., 2004) and 1.92 (95% CI: 1.05–3.87, $p = 0.03$) (Opler et al., 2008), when the Pb concentration in the maternal serum found to be $>15 \mu g dL^{-1}$. Hollander et al. (2020) additionally reported elevated plasma Pb concentrations in individuals newly diagnosed with schizophrenia. Furthermore, both Zaks et al. (2021) and Hollander et al. (2020) referenced a study that identified a positive association between early-life lead exposure and schizophrenia by analysing lead levels in childhood-shed teeth of adult patients. However, the findings across studies remain inconsistent, as Zaks et al. (2021) also cited a subsequent investigation that failed to replicate this association. Furthermore, one review specifically focused on chronic Pb exposure during childhood and linked it to a significantly increased risk of developing schizophrenia later in life (Albores-Garcia et al., 2021).

Overall, the reviewed literature supports a link between early-life lead exposure and an increased risk of developing schizophrenia or experiencing psychotic disorders later in life. Details of the studies reporting on schizophrenia and psychosis can be found in Table 5.24.

Table 5.24 Included reviews for metal exposure and the outcomes schizophrenia and psychosis

Author (year)	Pollutant	Outcome	Population	Review type	# of studies included in review	# of relevant (a,b)	Results / Conclusion by the study authors
Albores-Garcia (2021)	Pb	Schizophrenia	General population (early-life exposure)	Narrative	n.a.	9	Significant association described between chronic early-life exposure and schizophrenia in adulthood
Attademo (2017)	Pb, Cd	Schizophrenia	General population	Narrative	19	9	Authors suggest a link between heavy metal exposure, especially lead, and an increased risk of getting schizophrenia
Heyer (2017)	Pb	Schizophrenia	General population	Narrative	210	1	Study by Opler et al. reported association between second-trimester lead exposure and schizophrenia later in life
Hollander (2020)	Pb	Schizophrenia	General population	Narrative	n.a.	10	Findings suggest significant association between early-life and prenatal Pb exposure and schizophrenia, increased plasma Pb concentration found in newly diagnosed schizophrenia patients
Modabbernia (2016)	Pb, Cd, Cu, Mn	Schizophrenia	General population	Narrative	n.a.	8	Higher Mn blood concentration found in schizophrenia patients, association between Pb and Cd schizophrenia reported, mixed findings regarding link between Cu and schizophrenia
Ordemann (2016)	Pb	Schizophrenia	General population	Critical	n.a.	4	Evidence suggests an association between early life Pb exposure (especially prenatal) and Schizophrenia later in life Double the risk for developing schizophrenia, when maternal blood lead levels > 15 mg dL ⁻¹
Scassellati (2020)	Cu, Zn, Fe, Mn	Schizophrenia	General population	Narrative	n.a.	24	Conflicting results for Cu concentration in schizophrenia patients (some higher, some lower), antipsychotics lower Cu to control levels Mixed findings for FE, low ferritin linked to severe negative symptoms; antipsychotics decrease Fe concentration Often reduced MN concentration in schizophrenia patients, antipsychotics significantly reduce Mn levels

Author (year)	Pollutant	Outcome	Population	Review type	# of studies included in review	# of relevant ^(a,b)	Results / Conclusion by the study authors
							Meta-analysis showed reduced serum Zn in schizophrenia patients, strongest in drug-naive patients
Tota (2024)	Pb	Schizophrenia Psychosis	General population	Narrative	180	4	Positive association between lead exposure and schizophrenia and psychosis was found
Ventriglio (2021)	Pb, Cd	Psychosis	General population	Narrative	134	8	Lead and cadmium exposure linked to development of psychosis, prenatal lead exposure associated with onset of psychosis
Vorvolakos (2016)	Pb	Schizophrenia	General population	Narrative	n.a.	1	Study by Opler et al. reported association between second-trimester lead exposure and schizophrenia later in life
Zaks (2021)	Pb, Cu, Mg, Mn, Zn	Schizophrenia	General population	Systematic	38	38	Perinatal and adult Pb exposure linked to schizophrenia risk/severity; longitudinal data suggest nearly doubled risk, but findings mixed for perinatal studies Lower perinatal Cu in teeth of future schizophrenia patients; inconsistent results in serum, plasma, and hair Perinatal Mg in teeth higher in future schizophrenia cases, mixed evidence in perinatal and patient-level studies Lower perinatal Mn in teeth of schizophrenia patients; inconsistent findings in blood-based measures Consistently lower perinatal Zn in teeth of future schizophrenia patients, mixed evidence in serum/plasma

Abbreviations: Cd = Cadmium; Cu = Copper, Fe = Iron, Mg = Magnesium, Mn = Manganese, Pb = Lead, Zn = Zinc.

^(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

^(b) For narrative reviews, the reported number of included studies may vary, as it is mostly not always clearly specified how many studies were considered.

Transition Metals

Scassellati et al. (2020), Modabbernia et al. (2016), and Zaks et al. (2021) reported findings on the transition metals manganese (Mn), copper (Cu), zinc (Zn) and Iron (Fe). The evidence regarding Mn is inconsistent both within and across reviews. Modabbernia et al. (2016) higher levels of Mn in schizophrenia patients, whereas Scassellati et al. (2020) found lower Mn concentration in biological specimens of schizophrenia patients. Zaks et al. (2021) reported mixed results: on the one hand, they found lower Mn^{2+} concentrations in the teeth of adults diagnosed with schizophrenia; on the other hand, when examining biological matrices such as serum and plasma, five studies reported increased Mn levels in schizophrenia patients, while four studies found reduced levels. Findings related to Cu are similarly inconclusive (Modabbernia et al., 2016; Scassellati et al., 2020; Zaks et al., 2021). For example, a study looked at the shed teeth from individuals who were later diagnosed with schizophrenia and found a 41% lower Cu^{2+} concentration six months postnatally, compared to controls (Zaks et al., 2021). However, results based on biological specimen, like plasma, scalp hair and serum, were mixed and inconsistent (Modabbernia et al., 2016; Scassellati et al., 2020; Zaks et al., 2021). Modabbernia et al. (2016) reported a positive association between Cu levels in hair and schizophrenia, while also highlighting a negative association for Cu levels in blood. Importantly, Scassellati et al. (2020) noted that patients treated with antipsychotic medications exhibited reduced Cu concentrations, suggesting that pharmacological treatment may influence Cu levels in biological matrices and thus complicate the interpretation of findings. Findings related to zinc (Zn) exposure appear to be more consistent across studies. Zaks et al. (2021) and Scassellati et al. (2020) reported reduced levels of Zn in both shed teeth of individuals, who later experienced schizophrenia, and serum Zn levels of schizophrenia patients. Scassellati et al. (2020) highlighted medication use as a potential confounding factor, noting that several studies have reported altered zinc levels in schizophrenia patients undergoing pharmacological treatment. This suggests that antipsychotic medication may influence Zn concentrations in biological specimens, complicating the interpretation of observed associations. For Fe, one review by Scassellati et al. (2020) reported inconsistent findings. While some studies found elevated blood iron levels in individuals with schizophrenia, others reported lower levels compared to control groups. One study cited in the review identified a significant positive association between serum ferritin (indicator for iron stores) and the severity of psychotic symptoms.

Overall, the literature on transition metals remains inconclusive, with findings for Mn and Cu in particular being inconsistent regarding both the existence and the direction of their association with schizophrenia

Alkali Metals

Magnesium (Mg), classified as an alkali metal, was the only such element reviewed in relation to schizophrenia. Zaks et al. (2021) reported non-significantly elevated Mg levels in shed teeth of later schizophrenia patients. For an Mg assessment in biological matrices (serum, plasma, scalp hair) the results were inconsistent, with five studies reporting higher levels in schizophrenia patients, while eight studies could not report any association.

Overall, the findings concerning exposure to metals and schizophrenia remain mostly, apart from Pb, inconclusive, with substantial variability and contradictory results across studies and biological matrices.

Other mental health outcomes

In addition to depression, anxiety, panic disorder, schizophrenia, and psychosis, a few other potential mental endpoints of metal exposure were identified in the reviewed literature. Albores-Garcia et al. (2021) reported a positive association between chronic early-life Pb exposure and substance use disorder later in life. Vorvolkos et al. (2016) examined the effect of Pb exposure on bipolar disorder and manic episodes. Although elevated serum Pb concentrations were not observed in patients experiencing mania, the authors emphasized that lead-induced body burden may be positively associated with bipolar disorder and manic episodes. Tota et al. (2024) reported a positive association between arsenic-contaminated drinking water and suicide rates. The study analysed 1639 settlements in Hungary and the age-standardized suicide rate. The towns and villages were then classified according to the arsenic-concentrations in the drinking water into low ($\leq 10 \mu\text{g/l}$), intermediate ($11\text{--}30 \mu\text{g/l}$), high ($31\text{--}50 \mu\text{g/l}$) and very high ($\geq 51 \mu\text{g/l}$) contaminations. The cited study by Rihmer et al. (2015) found a significant difference in suicide rates between the groups low and intermediate (p-value < 0.001) and low and high (p-value < 0.001). Details of the studies reporting on the forementioned other mental health outcomes can be found in Table 5.25.

Table 5.25 Included reviews for metal exposure and the outcomes suicide and bipolar disorder

Author (year)	Pollutant	Outcome	Population	Review type	# of studies included in review	# of relevant studies ^(a,b)	Results / Conclusion by the study authors
Albores-Garcia (2021)	Pb	Substance use disorder	Adolescents and Adults	Narrative	n.a.	8	Significant association described between (early-life) lead exposure and increased risk of substance use disorder in adolescents and adults
Tota (2024)	As	Suicide rates	General population	Narrative	180	4	Association between arsenic exposure through drinking water and higher suicide rates was found
Vorvolakos (2016)	Pb	Bipolar disorder	General population	Narrative	n.a.	1	One study reported high body lead burden linked to bipolar disorder

Abbreviations: As = Arsenic, Pb = Lead.

^(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

^(b) For narrative reviews, the reported number of included studies may vary, as it is mostly not always clearly specified how many studies were considered.

During the analysis of the reviews, several limitations repeatedly occurred, which were acknowledged by the authors. The authors criticized the lack of evidence for causal relationships and emphasized the need for more long-term studies (Attademo et al., 2017; Młyniec et al., 2015; Theron et al., 2022; Van Den Bosch and Meyer-Lindenberg, 2019; Ventriglio et al., 2021; Zaks et al., 2021) or a greater emphasis on human-based research (Flores-Gutierrez et al., 2023). This is also accompanied by the frequent use of cross-sectional study designs, which inherently limits the ability to establish causality (Luz et al., 2021; Tarnacka et al., 2021; Theron et al., 2022; Van Den Bosch and Meyer-Lindenberg, 2019; Vorvolakos et al., 2016). Another frequently mentioned limitation is the heterogeneity of methodological approaches in the reviewed human epidemiological studies, for example in the variety in assessment tools for mental health outcomes or the diversity in exposure matrices (Jacobson et al., 2022; Modabbernia et al., 2016; Ni et al., 2018; Phelan et al., 2018; Tota et al., 2024; Zaks et al., 2021). Some authors also mention the limited study pool as a challenge (Luz et al., 2021; Modabbernia et al., 2016; Scassellati et al., 2020; Tarnacka et al., 2021; Zaks et al., 2021). As mentioned in the section above some reviews highlighted medication or psychopharmaceuticals as potential confounders. Several authors noted that consistent adjustment for confounders is difficult, especially when not all included studies report them. Moreover, some reviews emphasized the possibility of residual or unmeasured confounding, resulting in a difficult interpretation of the findings itself (Modabbernia et al., 2016; Ni et al., 2018; Ordemann and Austin, 2016; Phelan et al., 2018; Scassellati et al., 2020; Vorvolakos et al., 2016). In addition, Phelan et al. (2018) highlighted limitations related to exposure assessment. They criticised the reliance on single-point dietary measurements, questioning the validity of drawing conclusions about real-life exposure based on such data. Lastly, Tota et al. (2024) and Schmitt et al. (2021) mentioned the potential risk of bias as a limitation.

5.2.4 Endocrine disruptors

Biological mechanisms

Endocrine-disrupting chemicals (EDCs) can affect mental health in various ways. Four mechanisms of action are explained in more detail below, as they are mentioned as potential processes in the included reviews.

Bisphenol A (BPA), for example, is considered an estrogen-mimicking compound that binds to estrogen receptors and interferes with the hormonal balance between estrogen and androgens. Particularly during sensitive periods of brain development such as the in-utero phase, this disruption may alter the formation of sex-specific brain structures and behaviours. It therefore affects both development and mood. Over time, this can contribute to conditions such as depression or anxiety disorders (Costa and Cairrao, 2024; Ejaredar et al., 2017; Mustieles et al., 2015; Wiersielis et al., 2020).

A second mechanism is EDC-induced neuroinflammation. Several reviews suggest that exposure to EDCs can lead to oxidative stress and inflammatory responses in key brain regions that are involved in emotional regulation. These processes are known to play a role in the development of both depression and anxiety (Costa and Cairrao, 2024; Jacobson et al., 2022; Liu et al., 2023; Van Den Bosch and Meyer-Lindenberg, 2019; Mesnil et al., 2020).

The third mechanism explains an association via epigenetic changes. Two reviews highlighted epigenetic modifications as a potential mechanism (Raja et al., 2022; Van Den Bosch and Meyer-Lindenberg, 2019). BPA exposure has been linked to methylation changes in relevant regions of hippocampal genes, a brain region involved in emotion regulation. Furthermore, during the neurodevelopmental phase, the brain undergoes epigenetic reprogramming. These processes are highly sensitive to environmental exposures, and interference by EDCs may lead to lasting changes in gene expression, increasing the risk of depression or anxiety later in life.

Lastly, a review points to the disruption of neurotransmitter systems as a potential pathway. BPA has been shown to disrupt serotonin signalling in brain regions involved in mood regulation, which may contribute to depression and anxiety. It may also dysregulate the norepinephrine system, a neurotransmitter linked to stress and arousal, leading to increased anxiety-like behaviours. Furthermore, there is evidence suggesting BPA interferes with the development of the GABAergic system, which normally helps to calm brain activity and reduce anxiety (Wiersielis et al., 2020).

Depression and anxiety

EDC were discussed in 11 reviews, either as the primary focus or as part of a broader set of investigated exposures (Arfianti Wiraagni et al., 2020; Costa and Cairrao, 2024; Ejaredar et al., 2017; Jacobson et al., 2022; Liu et al., 2023; Mikołajewska et al., 2015; Mustieles et al., 2015; Raja et al., 2022; Van Den Bosch and Meyer-Lindenberg, 2019; Webb et al., 2018; Wiersielis et al., 2020; Mesnil et al., 2020). All 11 reviews examined depression as an outcome, sometimes with the additional outcome anxiety (n = 6). Eight reviews examined either the endocrine disruptor BPA or phthalates individually (BPA: (Arfianti Wiraagni et al., 2020; Costa and Cairrao, 2024; Ejaredar et al., 2017; Mikołajewska et al., 2015; Mustieles et al., 2015; Wiersielis et al., 2020); Phthalates: (Jacobson et al., 2022; Liu et al., 2023)), while four reviews (Raja et al., 2022; Van Den Bosch and Meyer-Lindenberg, 2019; Webb et al., 2018; Mesnil et al., 2020) examined both substances or did not distinguish between different EDCs. The reviews synthesized evidence from a range of study designs, including mostly cross-sectional studies and cohort-studies. Most reviews focused on the prenatal exposure (n = 10) and the general population, occasionally including children and adolescents. Across all reviews, EDC exposure was linked to depression and anxiety. Several reviews reported positive associations, and others described indicative trends. Details of the studies reporting on the outcomes depression and anxiety can be found in Table 5.26.

Table 5.26 Included reviews for EDC exposure and the outcomes depression and anxiety

Author (year)	Pollutant	Outcome	Population	Review type	# of studies included in review	# of relevant studies ^(a,b)	Results / Conclusion by the study authors
Costa (2024)	BPA	Depression	Children (Prenatal exposure)	Narrative	41	3	Findings of association between depression and prenatal BPA exposure found only in boys (7 and 2-4 years)
Ejaredar (2017)	BPA	Depression	Children (prenatal exposure & up to 12 years)	Systematic	11	5	Various studies support a link between childhood and prenatal BPA exposure and depression, potential sex-specific effect (effects only in girls / only in boys - explained by socio-economic status and ethnicity)
Jacobson (2022)	Phthalates	Depression	Pregnant woman	Scoping	15	2	Inconsistent evidence for phthalate exposure and postpartum depression (1 study found increased odds for outcome, one did not)
Liu (2023)	EDC	Depression	Children (Prenatal exposure)	Critical	44	5	Narratively reported a link between childhood and maternal exposure (pregnancy) and depression
Mesnil (2021)	BPA Phthalates	Depressive symptoms	General population	Narrative	n.a.	6	Several studies reported link between prenatal and childhood BPA exposure and depressive symptoms (stronger evidence for boys), Association for phthalate exposure and depressive symptoms are found in adults and elderly
Mikołajewska (2015)	BPA	Depression	Children (3-5 years)	Narrative	29	2	Link between maternal urine BPA concentration during pregnancy to increased depression in 3-year-old-children was found, significant association in girls between depressive behaviour and prenatal BPA exposure
Mustieles (2015)	BPA	Depression	Children & adolescents	Narrative	12	4	Several human studies found significant associations between prenatal or childhood BPA exposure and increased depressive in children, potential sex-specific effect
Raja (2022)	Phthalates	Depression	Children (prenatal exposure)	Narrative	n.a.	3	For girls (3-5 years) positive association between prenatal phthalate exposure and depression was reported

Author (year)	Pollutant	Outcome	Population	Review type	# of studies included in review	# of relevant studies ^(a,b)	Results / Conclusion by the study authors
Van den Bosch (2019)	BPA Phthalates	Depression	General population	Narrative	193	13	Several studies reported link between prenatal and childhood BPA exposure and depressive symptoms (stronger evidence for boys), Association for phthalate exposure and depressive symptoms are found in adults and elderly
Webb (2022)	EDC	Depression	Children (7 years)	Narrative	49	3	Link between perinatal EDC exposure and depression in children
Wiersielis (2020)	BPA	Depressive symptoms	Children	Narrative	n.a.	8	Especially peri/prenatal exposure to BPA is linked to depressive symptoms, potential sex-specific effect (explained by time and age of testing)
Costa (2024)	BPA	Anxiety	Children (Prenatal exposure)	Narrative	41	3	Findings of association between anxiety and prenatal BPA exposure found only in boys (7 and 2-4 years)
Ejaredar (2017)	BPA	Anxiety	Children (prenatal exposure & up to 12 years)	Systematic	11	5	Various studies support a link between childhood and prenatal BPA exposure and anxiety, potential sex-specific effect (effects only in girls / only in boys - explained by socio-economic status and ethnicity)
Liu (2023)	EDC	Anxiety	General population	Critical	44	5	Narratively reported a link between childhood and maternal exposure (pregnancy) and anxiety
Mikołajew ska (2015)	BPA	Anxiety	Children (3-7 years)	Narrative	29	2	Link between maternal urine BPA concentration during pregnancy to increased anxiety in 3-year-old-children was found, significant association in girls between anxious behaviour and prenatal BPA exposure
Mustieles (2015)	BPA	Anxiety	Children & adolescents	Narrative	12	4	Several human studies found significant associations between prenatal or childhood BPA exposure and increased anxiety-like behaviour in children, potential sex-specific effect
Raja (2022)	Phthalates	Anxiety	Children (prenatal exposure & 3-5 years)	Narrative	n.a.	2	Inconsistent results regarding direction of the link between prenatal phthalate exposure an anxiety in girls (positive vs. negative findings)

Author (year)	Pollutant	Outcome	Population	Review type	# of studies included in review	# of relevant studies ^(a,b)	Results / Conclusion by the study authors
Wiersielis (2020)	BPA	Anxiety symptoms	Children	Narrative	n.a.	4	Especially peri/prenatal exposure to BPA is linked to anxiety symptoms, potential sex-specific effect (explained by time and age of testing)

Abbreviations: EDC = endocrine-disrupting chemicals, BPA = Bisphenol A.

^(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

^(b) For narrative reviews, the reported number of included studies may vary, as it is mostly not always clearly specified how many studies were considered.

van den Bosch and Meyer-Lindenberg (2019), Mesnil et al. (2020), Raja and colleagues (2022) all report a positive association between prenatal BPA exposure and depressive symptoms in children. In the studies included in these reviews, maternal biological samples (e.g., urine, blood) were analysed, and behavioural outcomes in children aged three to ten years were assessed. They also examined the effect of phthalate exposure and came to similar conclusions, with one notable exception. Two reviews (Van Den Bosch and Meyer-Lindenberg, 2019; Mesnil et al., 2020) reported a positive association between phthalate exposure and depressive symptoms in adults and older individuals. In contrast, Raja and colleagues (2022) found a negative association between prenatal phthalate exposure and anxiety symptoms in girls. Only one other review on EDCs (Costa and Cairrao, 2024) reported inverse associations for girls. Webb et al. (2018) examined EDCs in general without distinguishing between specific substances. Although the findings remained non-specific, the authors hypothesized that EDC exposure may contribute to the development of depression.

Five reviews specifically examined BPA in relation with depression and anxiety. These reviews present a coherent picture: all identified a positive effect of prenatal BPA exposure on the risk of depressive and/or anxiety symptoms during childhood. Costa and Cairrao (2024) reported this association in boys aged seven years (anxiety: $\beta = 1.9$, 95% CI: 0.0–3.7, p-value < 0.05; depression: $\beta = 3.2$, 95% CI: 1.4–5.1, p-value < 0.01 (Harley et al., 2013)) as well as between two and four years ($RR_{middle} = 1.83$, 95% CI: 1.01–3.30; $RR_{highest} = 2.59$, 95% CI: 1.52–4.42) (Li et al., 2020). Similarly, Ejaredar et al. (2017) found increased depressive symptoms in boys aged seven to nine (CBCL-Score increase for anxious/depressed: 0.48, 95% CI: 0.23–0.74, p-value = 0.0002) (Roien et al., 2015) and girls aged three (BASC-2 Score increase for anxiety = 11, 95% CI: 3.6–18; for depression: 12, 95% CI: 4.7–20) (Braun et al., 2011) and aged 2 (BSI score (incl. Items on anxiety and depression) increase: $\beta = 5.5$, 95% CI: 0.3–10.7, p-value > 0.005) (Braun et al., 2009). Mikołajewska et al., (2015) also cited Braun et al. (2011) reporting the effect being more prominent in girls. Wiersielis et al. (2020) identified associations for boys up to 12 years (children's depression rating scale increase for depression: $\beta = 2.47$, p-value = 0.04) (Perera et al., 2016). And Mustieles et al. (2015) for three-year-old girls. To statistically support this association, particular attention can be drawn to the review by Mustieles et al. (2015), which reported significant associations between maternal urinary BPA levels and both anxiety and depression in three-year-old girls. Using the Behaviour Assessment System for Children, Second Edition (BASC-2), the authors found elevated anxiety symptoms ($\beta = 12$, 95% CI: 4.7–20, p-value < 0.05) and depressive symptoms ($\beta = 11$, 95% CI: 3.6–18, p-value < 0.05). Even beyond the context of prenatal exposure, associations between elevated urinary BPA levels and increased symptoms of depression and anxiety have been observed. For example, Ejaredar et al. (2017) reported such associations in children between the ages of eight and ten years. Mustieles et al. (2015) also reported a significant association between urinary BPA levels and combined anxiety and depression scores in children, as measured by the Child Behaviour Checklist (CBCL). The authors found that higher BPA concentrations were associated with increased symptom scores ($\beta = 1.07$, 95% CI: 0.57–1.58, p-value = 0.001), further supporting the link between BPA exposure and adverse emotional outcomes in early childhood.

The results suggest a potential sex-specific effect, that can be observed across the reviews, although the findings are inconsistent. Some studies included in the review only showed an association between prenatal BPA exposure and depression/anxiety in girls (Mikołajewska et al., 2015; Raja et al., 2022). Other showed the same association but mostly in boys (Van Den Bosch and Meyer-Lindenberg, 2019; Mesnil et al., 2020). The reviews by Ejaredar et al. (2017), Mustieles et al. (2015) and Wiersielis et al. (2020) summarized the inconsistencies and provided possible explanations for the observed heterogeneous findings. It is suggested that the inconsistencies may be due to the timing of the outcome measurements (age difference) or the timing of exposure (trimester difference) (Mustieles et al., 2015; Wiersielis et al., 2020). Furthermore, one possible explanation is that humans are often exposed to a mixture of EDCs rather than individual substances. Such mixtures can disrupt the balance between male and female sex hormones during the neurodevelopment phase, which may interfere

with sex-specific brain development. Depending on the specific compound, dose, and timing of exposure, either boys or girls may be particularly vulnerable to these effects, potentially leading to different mental health outcomes later in life (Mustieles et al., 2015).

Two reviews focused specifically on phthalates as EDC (Jacobson et al., 2022; Liu et al., 2023). Liu and colleagues (2023) reported a positive association between maternal and early childhood exposure to the phthalates DEHP and MEHP and depression-like problems. Jacobson et al. (2022) cited two studies regarding phthalates and mental health, yielding inconsistent results. One study analysed urinary phthalate (DnOP) levels during pregnancy and found an increased 48 % risk of PPD (OR = 1.48, 95% CI: 1.04–2.11). In contrast, another study found no association between phthalate exposure measured via breast milk and PPD symptoms.

Across all reviews, bisphenol A (BPA) exposure was most consistently associated with depression and anxiety. Several reviews reported positive associations, while others described indicative but non-significant trends. Another noteworthy observation is the potential for sex-specific effects, which warrants further investigation. In contrast, findings regarding phthalates were more inconsistent and less conclusive.

During the analysis of the reviews, several limitations repeatedly occurred, which were acknowledged by the authors. The authors criticized the lack of evidence for causal relationships and emphasized the need for more long-term studies (Costa and Cairrao, 2024; Raja et al., 2022; Van Den Bosch and Meyer-Lindenberg, 2019; Webb et al., 2018). This is also accompanied by the frequent use of cross-sectional study designs, which inherently limits the ability to establish causality (Van Den Bosch and Meyer-Lindenberg, 2019). Another frequently mentioned limitation is the heterogeneity of methodological approaches in the reviewed human epidemiological studies, for example in the assessment of mental health outcomes (Ejaredar et al., 2017; Jacobson et al., 2022; Mustieles et al., 2015; Raja et al., 2022; Webb et al., 2018). In addition, some authors highlight limitations related to exposure assessment. Raja et al. (2022) criticised the reliance on single-point measurements, questioning the validity of drawing conclusions about real-life exposure based on such data. Costa and Cairrao (2024) noted that the exposure often is assessed through self-reports, which may introduce bias.

5.2.5 Pesticides

Biological mechanisms

The reviews included in the analysis identified three major biological mechanisms that may explain how pesticide exposure affects mental health. The first is pesticide-induced dysregulation of the cholinergic system. Pesticides interfere with the cholinergic system by disrupting the enzyme acetylcholinesterase (AChE). Pesticides can inhibit AChE, leading to an imbalance of acetylcholine. This imbalance has been associated with symptoms such as anxiety, depression, and cognitive impairments (Stallones and Beseler, 2016; Tota et al., 2024; Van Den Bosch and Meyer-Lindenberg, 2019; Ventriglio et al., 2021).

A second mechanism is pesticide-induced neuroinflammation. Pesticides can activate inflammatory processes in the brain and central nervous system, which is associated with many mental illnesses, especially depression. In the process, the messenger substance cytokines are released, which is also linked to depression and other mental disorders (Jacobson et al., 2022; Stallones and Beseler, 2016; Ventriglio et al., 2021; Mesnil et al., 2020). A specific form of inflammatory response is described by Mesnil et al. (2020): pesticides can disrupt cell communication by impairing the gap junction intercellular communication (GJIC), leading to the activation of hemichannels, potentially leading to cellular stress and inflammation.

A third cluster of findings refer to the multimodal neurotoxic effects of pesticides. Flores-Gutierrez and colleagues (2023), for example, describe the inhibition of the enzyme neuropathic target esterase (NTE), which can lead to delayed nerve damage. This nerve damage can contribute to outcomes such as depression or anxiety disorders. van den Bosch and Meyer-Lindenberg (2019) additionally highlighted the interference of pesticides with the serotonergic and dopaminergic systems, both of which are central to mood regulation and implicated in disorders like depression and schizophrenia. Jacobson et al. (2022) also discussed some hypothetical mechanisms of action, including dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and the promotion of oxidative stress.

Mental health outcomes

Pesticides were discussed in eight reviews, either as the primary focus or as part of a broader set of investigated exposures (Flores-Gutierrez et al., 2023; Jacobson et al., 2022; Stallones and Beseler, 2016; Tota et al., 2024; Van Den Bosch and Meyer-Lindenberg, 2019; Ventriglio et al., 2021; Wu et al., 2023a; Mesnil et al., 2020). Eight reviews examined depression, with additional outcomes including anxiety (n = 3) and schizophrenia (n = 1). The reviews synthesized evidence from a range of study designs, including medical studies (e.g. postmortem studies), longitudinal and cross-sectional studies, as well as case studies. Most reviews focused on the general population, occasionally including children and adolescents. One review specifically addressed pregnant and postpartum women (Jacobson et al., 2022). Details of the studies reporting on mental health outcomes (depression, anxiety, suicide, schizophrenia) from pesticide exposure can be found in Table 5.27 (meta-analyses) and Table 5.28 (reviews).

Table 5.27 Included meta-analyses for pesticide exposure and the outcome suicide risk

Author (year)	Outcome	Population	# of studies included in meta-analysis	# of relevant studies ^(a)	# of relevant European studies ^(a)	Results	I ²
Wu (2023)	Suicide risk	General population	20	3 ^(b)	1	Reference group (general population): OR = 1.23, (0.64–2.38) Exposure type (environmental exposure): OR = 1.72, (1.23–2.40)	90.6% 50.9%

^(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

^(b) 3 studies were relevant for a subgroup analysis regarding environmental pesticide exposure. The other 17 studies included in the overall meta-analysis either focused on occupational context or did not distinguish between occupational and environmental exposure.

Table 5.28 Included reviews for pesticide exposure and the outcomes depression, anxiety, suicide risk and schizophrenia

Author (year)	Outcome	Population	Review type	# of studies included in review	# of relevant studies ^(a,b)	Results / Conclusion by the study authors
Flores-Gutierrez (2023)	Depression	General population	Narrative	n.a.	3	Cited studies supported the link between long-term and acute pesticide exposure and increased risk of depression
Jacobson (2022)	Depression	Postpartum woman	Scoping	15	1	One cited study found positive association between heptachlor epoxide and depression
Mesnil (2020)	Major depressive disorder	General population	Narrative	n.a.	3	Prenatal and adult exposure associated with higher risk of depression
Stallones (2016)	Depression	General population	Narrative	24	15	Positive association of organophosphate poisoning and postpartum depression was found
Tota (2024)	Depression	General population	Narrative	180	4	Studies from Thailand, Brazil and Canada supported positive link between pesticide exposure and depression
Van den Bosch (2019)	Depression	General population	Narrative	193	17	Authors generally reported moderate evidence for an associated between pesticides and an increased risk of depression (specifically prenatal exposure with increased risk of depression in adulthood)
Ventriglio (2021)	Depression	General population	Narrative	134	19	Chronic exposure to organophosphate was associated with depression through inhibition of the AChE
Jacobson (2022)	Anxiety	Postpartum woman	Scoping	15	1	One cited study found positive association between heptachlor epoxide and anxiety
Tota (2024)	Anxiety	General population	Narrative	180	4	Studies from Thailand, Brazil and Canada supported positive link between pesticide exposure and anxiety
Ventriglio (2021)	Anxiety	General population	Narrative	134	19	Chronic exposure to organophosphate was associated with anxiety through inhibition of the AChE
Ventriglio (2021)	Suicide risk	General population	Narrative	134	19	Chronic exposure to organophosphate was associated with suicide risk through inhibition of the AChE
Stallones (2016)	Schizophrenia	General population	Narrative	24	2	Association of organophosphate poisoning and schizophrenia was reported by two studies

^(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

^(b) For narrative reviews, the reported number of included studies may vary, as it is mostly not always clearly specified how many studies were considered.

Across all reviews, pesticide exposure was consistently linked to trends indicating an increased risk of depression. While only a minority of findings were statistically significant, several reviews reported positive associations, and others described indicative trends. Positive associations were also reported for schizophrenia and anxiety, although these outcomes were examined less frequently.

Tota et al. (2024) based their findings on studies conducted in Thailand, Canada, and Brazil, where pesticides are primarily suspected to act as agrochemicals present in soil. Another review (Flores-Gutierrez et al., 2023) focused specifically on organophosphates (OPs), a class of pesticides, that is toxic to humans. This review was primarily based on case-control- and cross-sectional-studies and reported positive associations between chronic OP exposure and depression. Furthermore, acute OP poisoning was also linked to depression. Ventriglio and colleagues (2021) also specifically examined the class of OP as pesticides and found (based on 19 studies) a positive association between chronic OP exposure and depression, anxiety and suicidality. A meta-analysis (Wu et al., 2023a) examined exposure to the pesticides groups OPs and organochlorines (OCPs) in relation to suicide risk. The authors conducted the only meta-analysis on pesticide exposure in our systematic umbrella+ review, concluding a non-significant trend towards increased risk (OR = 1.23, 95% CI: 0.64–2.38, I^2 = 90.6%, p -value < 0.001) for the general population (excluding occupational exposure). When stratified by type of pesticide exposure, the analysis found a statistically significant association between environmental exposure and increased suicide risk (OR = 1.72, 95% CI: 1.23–2.40, I^2 = 50.9%, p -value = 0.154). Stallones and Beseler (2016) also specifically dealt with OPs in their review. They also found a positive association between OP poisoning and depression, but also for schizophrenia. van den Bosch and Meyer-Lindenberg (2019), examined a range of environmental chemicals, including pesticides. Their conclusions were based on 17 studies. In addition to reporting a positive association between pesticide exposure and depression risk, the authors noted that the evidence was stronger in cases of high-dose exposures. They also addressed the effects of prenatal pesticide exposure on mental health outcomes later in life and found a positive association in that context as well. Similarly, Mesnil et al. (2020) reviewed both prenatal and adulthood exposure to pesticides and reported an increased risk of depression for both exposure periods. Notably, one of the two underlying studies referenced in Mesnil et al. (2020) was the review by van den Bosch and Meyer-Lindenberg (2019). The last review (Jacobson et al., 2022) found a positive association between pesticides and depression and anxiety, but only for the insecticide heptachlor epoxide. The population examined in the cited study consisted specifically of postpartum women (8 months postpartum), whose breast milk was analysed for OCP residues

Overall, the findings regarding pesticide exposure and mental health endpoints like depression, schizophrenia, anxiety and suicidality are very consistent across all studies. While some reviews provided statistically significant data, the non-significant results still supported the trend of a positive association.

During the analysis of the reviews, several limitations repeatedly occurred, which were acknowledged by the authors. The overall pattern appears relatively coherent; however, some of the reported findings remain speculative or are based on indirect effects (Flores-Gutierrez et al., 2023; Stallones and Beseler, 2016; Ventriglio et al., 2021), thereby necessitating further empirical investigation. The authors also criticized the lack of evidence for causal relationships and emphasized the need for more long-term studies (especially low-dose exposure) (Van Den Bosch and Meyer-Lindenberg, 2019) or a greater emphasis on human-based research (Flores-Gutierrez et al., 2023). This is also accompanied by the frequent use of cross-sectional study designs, which inherently limits the ability to establish causality (Stallones and Beseler, 2016; Van Den Bosch and Meyer-Lindenberg, 2019). Additionally, Stallones and Beseler (2016) highlighted the challenge of disentangling the effects of pesticides, especially OPs, from other life stressors, further complicating causal interpretations.

5.2.6 *Other chemicals*

In addition to the four categories of environmental chemicals SHS, metals, EDCs, and pesticides, the reviews discussed three other ECs that do not clearly fit into any of these categories. These include per- and polyfluoroalkyl substances (PFAS) and mixtures. PFAS are addressed in two reviews (Jacobson et al., 2022; Suthar et al., 2024) and mixtures in one review (Mixtures: Constantinescu et al. (2025)). Details of the studies reporting on PFAS and Mixture exposure and the mental health outcomes depression and schizophrenia can be found in Table 5.29.

Table 5.29 Included reviews for PFAS and mixture exposure and the outcomes depression and schizophrenia

Author (year)	Pollutant	Outcome	Population	Review type	# of studies included in review	# of relevant studies ^(a,b)	Results / Conclusion by the study authors
Constant inescu (2025)	Mixtures	Depression	Children and Adolescents	Narrative	14	1	One study supported link between prenatal exposure of mixtures (metals and organochlorine compounds) and depression
Constant inescu (2025)	Mixtures	Schizophrenia	Children and Adolescents	Narrative	14	1	One study supported link between prenatal exposure of mixtures (metals and organochlorine compounds) and schizophrenia
Jacobson (2022)	PFAS	Depression	Pregnant woman	Scoping	15	1	No association between PFAS and depression was found
Suthar (2024)	PFAS	Depression	Pregnant woman	Scoping	11	11	Alanine metabolism most consistently reported significant pathway regarding perinatal and antenatal depression, followed by glutamate metabolism and tyrosine metabolism

Abbreviations: PFAS = Per- and polyfluoroalkyl substances.

(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

(b) For narrative reviews, the reported number of included studies may vary, as it is mostly not always clearly specified how many studies were considered.

Per- and polyfluoroalkyl substances

Per- and polyfluoroalkyl substances (PFAS) were considered as the primary exposure of interest in the review by Suthar et al. (2024), whereas in Jacobson et al. (2022), they were considered as part of a broader category of persistent organic pollutants (POPs). Both reviews focused specifically on perinatal depression in pregnant women. The objective of the review by Suthar et al. (2024) was to identify the potential metabolic mechanisms between PFAS exposure and perinatal or antenatal depression in pregnant individuals. The scoping review was based on 11 studies, from China (n = 5), the USA (n = 4), Canada (n = 1) and Japan (n = 1), all of which were cross-sectional or case studies with one longitudinal study. The authors identified alanine metabolism as the most consistently reported significant pathway in studies on the metabolomics of perinatal and antenatal depression. This was followed by glutamate metabolism and tyrosine metabolism. With regard to PFAS-associated metabolic changes, fatty acid metabolism emerged as the most frequently reported significant pathway, suggesting it may play a central role in the biological response to PFAS exposure.

These findings suggest that alterations in specific metabolic pathways—particularly those involving alanine, glutamate, tyrosine, and fatty acids—may serve as biological explanations, linking environmental PFAS exposure to depressive symptoms during pregnancy. However, the authors emphasize that this does not confirm causality but rather provides initial evidence of common molecular pathways that should be specifically investigated in future studies.

Jacobson and colleagues (2022) examined the relationship between POPs, which include PFAS, and prenatal depressive symptoms. The authors referenced a cohort study in which maternal serum concentrations of PFAS were measured and depressive symptoms were assessed multiple times during and after pregnancy (up to 8 years postpartum). No significant association between PFAS and perinatal depression was found. In contrast to the review by Suthar et al. (2024), the potential mechanisms of action are not explained in detail here.

Both reviews (Jacobson et al., 2022; Suthar et al., 2024) acknowledged limitations. Suthar et al. (2024) described the challenge of pathway analysis due to the use of diverse computational tools across studies. The generalizability of the results was also addressed by the authors. Largely due to heterogeneity in biospecimen collection methods and the measurement tools used to assess depressive symptoms.

In summary, the available reviews provided inconclusive evidence regarding a link between PFAS exposure and perinatal depression. While Suthar et al. (2024) presented initial insights into potential shared metabolic pathways, Jacobson et al. (2022) reported no significant associations. Further longitudinal and mechanistically focused studies are required to clarify the relationship between PFAS exposure and mental health outcomes during and after pregnancy.

Mixtures

Constantinescu et al. (2025) examined exposure to chemical mixtures, specifically combinations of organochlorine compounds and heavy metals, as a potential risk factor for the development of depression. Drawing on findings from the cohort study conducted by Rokoff et al. (2022) the review highlighted a positive association between mixture exposure during the prenatal phase and depression and schizophrenia in childhood and adolescence. The cohort study included follow-up periods of eight and 15 years and also incorporated analysis of umbilical cord blood samples. The authors suggested that the combined exposure to multiple environmental chemicals may exert synergistic effects on mental health. The proposed mechanisms of action primarily involve inflammation and oxidative stress. Chronic exposure to chemical mixtures is suggested to have long-term detrimental effects on the brain, potentially contributing to neurodegenerative processes. Both oxidative stress and

inflammation are recognized as predictors of neurological disorders, including depression and schizophrenia (Constantinescu et al., 2025).

5.2.7 Discussion

This section of the umbrella+ review focus on environmental chemicals and their potential impact on human mental health. The aim is to address the research question: ‘In the general population or in population subgroups, including children and pregnant individuals (P), what is the increase in risk of health effects (O) per unit increase (C) of environmental chemicals (E), observed in reviews of epidemiological studies (S)?’.

For this purpose, an umbrella+ review was conducted. PsycINFO, SCOPUS and PubMed were searched systematically for any type of reviews (narrative, systematic, scoping, critical and meta-analyses) investigating the association between environmental chemical exposure and mental health outcomes. The findings of this search are presented in Section 5.2. During the search process, numerous articles were excluded due to predefined criteria. To provide an overview of the most common reasons for exclusion during the initial title and abstract screening the reasoning for exclusions for a proportion of the reviews are presented in the following. The largest number of exclusions occurred because reviews focused on neurodegenerative or neurodevelopmental disorders such as ADHD, Parkinson’s disease, dementia, or Autism Spectrum Disorder, which were defined as outside the scope of the present review. Physical health outcomes, such as psoriasis, endometriosis, or structural brain changes, were also beyond the study focus. Furthermore, self-poisoning with environmental chemicals was not considered as a suitable exposure following our research question. Additional reasons were the absence of any environmental chemical or a focus on chemicals in medication, which does not fit the criteria of exposure in the general population. Overall, the developed search strategy yielded relevant results; however, it frequently retrieved reviews on neurodegenerative outcomes, which were systematically excluded during the screening process.

In total, 49 reviews met the inclusion criteria. Pb emerged as the most prominently studied exposure, with 15 reviews specifically addressing its relationship to mental health outcomes. Likewise, depression was by far the most frequently examined outcome, reported in 36 reviews. Overall, both the spectrum of environmental chemicals investigated, and the range of mental health outcomes assessed were comparatively narrow. The evidence base clustered around four major exposure groups (metals, SHS, EDCs, pesticides) supplemented by two smaller categories (PFAS and chemical mixtures). With respect to outcomes, the identified reviews predominantly focused on depression, anxiety, schizophrenia, and psychosis, with bipolar disorders, manic episodes, and substance use disorders showing only limited coverage. This concentration of research topics underscores a clear gap in the literature: the need to expand investigations to a wider variety of mental health outcomes and to environmental chemicals beyond those most frequently studied.

Central findings

Second-hand smoke

SHS exposure has been studied primarily in relation to depression, with additional emphasis on vulnerable groups such as children and pregnant individuals. The available evidence indicates a reasonably consistent positive association between SHS exposure and both depression and schizophrenia. The literature comprises systematic reviews and meta-analyses in which either the meta-analyses, or the primary studies they synthesized, frequently reported significant associations. For example, Han et al. (2019) reported a significant odds ratio of 1.32 (95% CI: 1.20–1.30) for depression in the general population; Jacobson et al. (2022) and Suzuki et al. (2019) found ORs of 1.30 (95% CI: 1.03–1.64) and 1.77 (95% CI: 1.12–1.79), respectively, for PPD. In the case of antenatal depression, significantly increased odds of up to 2.68 (95% CI: 1.73–4.17) (Padhani et al., 2024) have been reported. Multiple dose-response relationships have also been documented, indicating that

more frequent SHS exposure is associated with either a greater increase in depressive symptoms or a higher risk of developing depression (Han et al., 2019; Theron et al., 2022). The literature also presented a fairly consistent picture for schizophrenia as an outcome, reporting a positive association between the development of schizophrenia and prenatal SHS exposure (e.g. OR = 1.29, 95% CI: 1.10–1.51) (Hunter et al., 2020). For the vulnerable group of children, findings from the included reviews were also broadly consistent: most reviews reported a positive, sometimes statistically significant, association between either prenatal SHS exposure and risk of depression or childhood SHS exposure (e.g., at home) and an increased likelihood of depressive symptoms (Theron et al., 2022; van der Eijk and Woh, 2023). However, there were also studies that found no association or gender-specific effects only, as reported by Christian and Kim (2022). In contrast, the evidence for anxiety as an outcome was inconsistent, with no clear pattern of significant associations between SHS exposure and anxiety-related symptoms in the literature.

According to the EEA (2022b), SHS exposure affects approximately 31% of the European population. Sources of SHS include the home, workplace or school, public spaces, and leisure environments. The WHO (2023a, 2023b) highlights children as a particularly vulnerable group, noting that in some European countries (e.g., Croatia and Serbia), up to six out of ten children aged 13–15 are exposed to SHS at home. For the general European population, SHS was estimated to account for 4.3 million DALYs and 171,000 deaths in 2019 (WHO, 2023b). Ma et al. (2021), summarised data from the Global Youth Tobacco Survey across 33 European countries, reported SHS prevalence rates among adolescents aged 12–16 of 73% (70.8–75.0) for ≥ 1 day in the past 7 days, 60.8% (58.5–63.0) for ≥ 3 days, 47.5% (45.5–49.6) for ≥ 5 days, and 38.2% (36.5–40.0) for daily exposure.

Given these high levels of exposure, particularly among children and adolescents, the potential contribution of SHS to adverse mental health outcomes warrants careful consideration and further research.

Metals

Most of the literature focused on heavy metals, especially lead. The most consistent findings concerned the positive association between prenatal or childhood Pb exposure and both depression and schizophrenia, exemplified by an increased odds ratio of 2.3 for developing major depressive disorder in young adulthood following childhood exposure (Neuwirth et al., 2020). Evidence on prenatal Pb exposure and schizophrenia was primarily derived from two studies by Opler et al. (2004, 2008), which were cited as key references in six different reviews. For other heavy metals such as Cd, Hg or Sn, the results of the reviews were somewhat consistent. Correlations between exposure and mental outcomes were frequently found, e.g. between Hg and anxiety in a pregnancy/birth cohort study (Luz et al., 2021). However, some studies found no evidence of an association.

The literature is also inconsistent on the relationship between copper (Cu) and manganese (Mn) exposure and depression. While some reports suggested the existence of a link, the direction of association varies. For example, Tarnacka et al. (2021) and Mesnil et al. (2020) reported contradictory directions of a potential link (positive or negative). On the other hand, significant positive associations are also described, such as between Cu levels in blood and depression (Ni et al., 2018). In contrast, Mg²⁺ exposure was negatively associated with depression, although this finding was not supported by any longitudinal study (OR = 0.71, 95% CI: 0.40–1.02, p-value = 0.10) (Phelan et al., 2018). For schizophrenia, the results were very inconsistent.

It is not possible to provide a general statement regarding population exposure to metals, as each element requires separate consideration. For heavy metals such as Cd and Pb, human biomonitoring (HBM4EU, 2020c) data are available that provide insight into exposure levels within Europe. Given the multiple pathways of exposure, HBM represents the most appropriate method of assessment. Blood

lead levels (BLLs) in the general population have been measured in several European countries and show a steady decline over the past decade. This trend is likely attributable, at least in part, to regulatory bans on lead in drinking water pipes, paint, and petrol in many European countries. Declines in BLLs have also been observed in children in recent years. However, the HBM4EU initiative highlights the lack of current data, noting that only seven countries (Belgium, Germany, Denmark, Kosovo, Poland, Slovenia, and Sweden) have conducted BLL measurements within the last five years (HBM4EU, 2020c). HBM4EU data are also available for Cd, indicating that dietary intake (including drinking water) and tobacco smoke represent the main exposure routes. On average, adults in Europe consume 10–20 µg Cd per day through food, reflected in urinary concentrations of 0.5–1.0 µg Cd/day and blood concentrations of 0.5–1.0 µg Cd/L (HBM4EU, 2020b). For mercury, the European Food Safety Authority (EFSA) (2015) assessed exposure primarily through fish consumption. The report concluded that it is not possible to make generalized statements for Europe, given substantial variation in consumption patterns, fish and seafood types, and age groups. Nevertheless, young children and children aged three to ten years, were identified as especially vulnerable, as they may reach the tolerable weekly intake of methylmercury before the nutritional benefits of fish consumption can be realized.

Another example is arsenic. EFSA reported on arsenic exposure through food and drinking water in 2014 and 2021 (EFSA, 2014; EFSA et al., 2021). In 2014, nearly 100,000 samples were analysed, of which 66% were below the limit of detection or quantification (EFSA, 2014). For inorganic arsenic specifically, 98% of drinking water samples in the EU were found to be below the regulatory limit. In the 2021 report, EFSA analysed approximately 13,600 water and food samples, identifying infants, toddlers, and children as particularly vulnerable groups (EFSA et al., 2021). Compared with 2014, arsenic levels were markedly reduced, with maximum mean and 95th percentile estimates being 1.5–3 times lower. Overall, exposure to most metals appears to have decreased in recent years. However, chronic exposure continues to pose risks, underscoring the importance of future research into the long-term health effects of metal exposure.

Endocrine-disrupting chemicals

The reviews on EDCs primarily focused on BPA and phthalates. There were many similar findings, particularly for BPA, all of which found a positive association between prenatal BPA exposure and depression as well as anxiety in childhood (Raja et al., 2022; Van Den Bosch and Meyer-Lindenberg, 2019; Mesnil et al., 2020). There is substantial evidence suggesting sex-specific effects of BPA; however, these effects are not consistent across reviews. In some cases, significant positive associations were observed only in girls, as reported by Mustieles et al. (2015) for anxiety symptoms ($\beta = 12$, 95% CI: 4.7–20, p -value < 0.05) and depressive symptoms ($\beta = 11$, 95% CI: 3.6–18, p -value < 0.05). In other instances, positive associations were observed only in boys (Van Den Bosch and Meyer-Lindenberg, 2019; Mesnil et al., 2020). Some reviews also reported non-sex-specific positive associations between BPA exposure and depressive symptoms in children (Mikołajewska et al., 2015). Authors have hypothesized that these differences may result from BPA-induced disruption of brain development, leading to altered formation of sex-specific brain structures and behaviours, which in turn could produce sex-specific effects.

While BPA is generally positively associated with mental health, the situation is different for phthalates. Here, the evidence for phthalates is more inconsistent, with mixed directions of association and varying strength of evidence. A few reviews reported a positive association between phthalate exposure and depressive symptoms (Van Den Bosch and Meyer-Lindenberg, 2019; Mesnil et al., 2020), whereas others have found evidence suggesting negative associations (Raja et al., 2022). Findings for PPD are similarly inconsistent: some studies reported significant associations (OR = 1.48, 95% CI: 1.04–2.11), while others found no evidence for a relationship (Jacobson et al., 2022).

The exposure pathways for EDCs are diverse, including dermal absorption, inhalation, and dietary intake. Consequently, human biomonitoring (HBM), which analyses urine, blood, or other body materials for phenols or phthalates, is frequently employed to assess exposure. For BPA in particular, there is broad consensus in the literature that the majority of the population is exposed (HBM4EU, 2020a). Several studies have analysed HBM data with respect to BPA and phthalates. For example, Casas et al. (2013) summarised six European cohort studies and reported elevated phenol concentrations across samples, with pregnant women in Spain showing higher levels than those in France. BPA levels were also highest in Spanish children compared to children from France and Germany.

Phthalate exposure has been investigated in nine European cohort studies and similarly demonstrated variability depending on country and population group. Again, Spanish children showed the highest phthalate-metabolite levels compared to Spanish mothers and German children. Evidence from Duisburg, Germany, also indicated higher phthalate concentrations in children than in their mothers, suggesting that children may be particularly vulnerable to exposure. Such geographical and population-specific differences may reflect variations in dietary habits (e.g., food packaging and container use), cosmetic use (e.g., body care products), and other lifestyle factors (Casas et al., 2013).

Pesticides

Across the eight reviews focusing on pesticide exposure, a generally consistent link was reported between exposure and various mental health outcomes, primarily depression, but also schizophrenia and anxiety. Both Ventriglio et al. (2021) and Wu et al. (2023a) examined suicide risk as an outcome and found some significant associations (OR = 1.72, 95% CI: 1.23–2.40, I² = 50.9%, p-value = 0.154 (Wu et al., 2023a)). Although suicide is not a mental disorder per se, it can be considered an indicator of underlying mental illness (Wu et al., 2023a). Several reviews investigated the relationship between prenatal pesticide exposure and depressive symptoms later in life, identifying positive associations in their included studies (Van Den Bosch and Meyer-Lindenberg, 2019; Mesnil et al., 2020). One review also examined the postpartum phase of woman and found a positive association between the pesticide heptachlor epoxide and depression and anxiety (Jacobson et al., 2022). While only a minority of findings reached statistical significance, multiple reviews nonetheless reported positive associations between pesticide exposure and mental health outcomes, and others described indicative, though nonsignificant, trends.

Unlike SHS, data on pesticide exposure are more complex, as multiple exposure pathways need to be considered. Dietary intake represents one of the most important sources. In its 2025 report, EFSA presented the results of the EU-coordinated multiannual control programme (EU MACP) for 2023 (EFSA et al., 2025). More than 13000 food samples from EU Member States, Iceland, and Norway (including carrots, oranges, potatoes, and dried beans) were analysed. The vast majority (98%) complied with EU standards for pesticide residues, while 2% exceeded maximum residue levels, with 1% classified as non-compliant after accounting for measurement uncertainties. Overall, the EFSA (2025) assessed both the acute and chronic risks of pesticide exposure as very low. It should also be noted that dietary intake is only one pathway of pesticide exposure; however, there is only limited data on alternative pathways. Nevertheless, this umbrella review indicates a potential harmful link to mental health, highlighting the need for continued surveillance and research.

PFAS and Mixtures

The two reviews focusing on PFAS both examined perinatal depression in pregnant women but adopted markedly different approaches. Jacobson et al. (2022) cited a study assessing PPD up to eight years after childbirth and PFAS exposure, reporting no evidence of an association. In contrast, Suthar et al. (2024) investigated potential biological pathways, identifying alterations, particularly in those

involving alanine, glutamate, tyrosine, and fatty acids, that may act as marks linking environmental PFAS exposure to depressive symptoms during pregnancy.

One environmental chemical category that appeared only once in the captured literature was mixtures, specifically those comprising heavy metals and the pesticide organochlorine. Constantinescu et al. (2025) referenced a cohort study with follow-up periods of eight and 15 years, which found a positive association between mixture exposure and depression.

Core biological mechanisms

This umbrella+ review focused on environmental chemicals and their effects on mental health. In order to understand the effects, the biological mechanisms of action concerning environmental chemicals were also examined. Some of these stood out because they were mentioned multiple times in relation to several chemicals. However, it should be noted that many potential mechanisms of action were mentioned in the reviews, but few of them have been confirmed. Often, there are only assumptions or hypotheses about how the chemicals actually affect the brain and why they can have negative effects on mental health. It was particularly emphasized that the developmental phase is a very fragile one and that environmental chemicals can have a particularly strong influence during this period (prenatal or early childhood), which can then manifest later in life as depression, schizophrenia, etc. Overall, two processes were mentioned across all chemicals, which are now briefly summarized here.

A large proportion of reviews identified chemical-induced oxidative stress as a key mechanistic pathway. Environmental chemicals can disrupt biochemical processes through direct cellular damage or interference with intracellular signalling pathways. Such disruptions can trigger oxidative stress, which in turn may initiate multiple downstream processes leading to neuroinflammation. Chronic neuroinflammation can cause long-term impairments in neurotransmitter systems and thereby contribute to the manifestation of mental health outcomes such as depression, schizophrenia and anxiety (Constantinescu et al., 2025; Costa and Cairrao, 2024; Jacobson et al., 2022; Liu et al., 2023; Luz et al., 2021; Młyniec et al., 2015; Modabbernia et al., 2016; Ni et al., 2018; Scassellati et al., 2020; Tarnacka et al., 2021; Tota et al., 2024; Van Den Bosch and Meyer-Lindenberg, 2019; Ventriglio et al., 2021; Zaks et al., 2021; Mesnil et al., 2020). Two of these neurotransmitter systems are the dopaminergic and serotonergic systems, both of which were frequently cited in the reviewed literature as potential mechanistic targets of environmental chemicals. The dopaminergic system is primarily involved in motivation and reward processes, but also mood regulation, whereas the serotonergic system regulates, among other functions, mood, sleep, and anxiety. Disruption of either system, e.g. through a reduction of the production of the neurotransmitters dopamine or serotonin, may contribute to the development of mental health outcomes (Albores-Garcia et al., 2021; Hollander et al., 2020; Hunter et al., 2020; Młyniec et al., 2015; Modabbernia et al., 2016; Ni et al., 2018; Phelan et al., 2018; Scassellati et al., 2020; Tarnacka et al., 2021; Van Den Bosch and Meyer-Lindenberg, 2019; Vorvolakos et al., 2016; Zaks et al., 2021).

Further findings

In some reviews, negative associations between environmental chemicals and mental health outcomes are mentioned sporadically, for example for Fe (Tarnacka et al., 2021) or dietary intake of Mg^{2+} (Phelan et al., 2018). However, in most cases, additional studies reported either the opposite effect or no association at all. In the present systematic search, selenium as an example was associated with a protective effect on depressive symptoms in several studies (Młyniec et al., 2015; Scassellati et al., 2020). While there was one environmental chemical identified for which the majority of studies suggested a negative association with mental health outcomes: the alkali metal lithium, with four reviews addressing this substance. Details of the studies reporting on lithium and selenium exposure and depression, anxiety, suicide rate, bipolar disorder and schizophrenia can be found in Table 5.30

(reviews). The three meta-analyses regarding lithium exposure and suicide rates are shown in Table 5.31.

Table 5.30 Included reviews for lithium and selenium exposure and the outcomes depression, suicide rate, anxiety, bipolar disorder and schizophrenia

Author (year)	Pollutant	Outcome	Population	Review type	# of studies included in review	# of relevant studies ^(a,b)	Results / Conclusion by the study authors
Eyre-Watt (2021)	Li	Depression	General Population	Systematic	27	4	Mixed results for link between depressive symptoms and lithium in drinking water, no significant association between lithium in drinking water and MDD was found
Memon (2020)	Li	Suicide rate	General population	Systematic	15	15	Majority of studies (both genders combined) (7/9) showed protective association between lithium in drinking water and suicide rates (5 significant), potential modifier: lithium prescription in population
Vita (2015)	Li	Suicide rate	General population	Systematic	9	9	Overall consistent evidence for a protective effect of lithium in drinking water and suicide rates
Eyre-Watt (2021)	Li	Anxiety	General Population	Systematic	27	1	After drinking lithium rich drinking water one cited study found improvement in anxiety scales
Eyre-Watt (2021)	Li	Bipolar disorder	General Population	Systematic	27	3	No association found between lithium in tap water and bipolar disorder
Eyre-Watt (2021)	Li	Schizophrenia	General Population	Systematic	27	1	One study linked highest concentration of lithium in drinking water to higher risk of developing schizophrenia (compared to lowest concentration)
Młyniec (2015)	Se	Depression	General Population	Narrative	n.a.	4	Inconsistent findings regarding selenium intake and depressive symptoms, MDD or PDD

Abbreviations: Li = Lithium, Se = Selenium, MDD = Major depressive disorder, PDD = Postpartum depression.

^(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

^(b) For narrative reviews, the reported number of included studies may vary, as it is mostly not always clearly specified how many studies were considered.

Table 5.31 Included meta-analyses for lithium exposure and the outcome suicide rate

Author (year)	Outcome	Population	# of studies included in meta-analysis	# of relevant studies ^(a)	# of relevant European studies ^(a)	Results	I ²
Barjasteh-Askari (2020)	Suicide rate	General population	14	14	7	OR = 0.42, (0.27–0.67), p < 0.01 Europe: OR = 0.65, (0.40–1.04), p = 0.071 Male: OR = 0.54, (0.35–0.84), p < 0.01 Female: OR = 0.70, (0.48–1.01), p = 0.057	85.51% 77.26% n.a. n.a.
Eyre-Watt (2021)	Suicide rate	General population	16	16	7	r = -0.191 (-0.287– -0.090), p < 0.001	80.7%
Memon (2020)	Suicide rate	General population	9	9	4	Overall: β = -0.27, (-0.47– -0.08), p = 0.006 Male: β = -0.26, (-0.56–0.03), p = 0.08 Female: β = -0.13, (-0.24– -0.02), p = 0.03	83.3% 91.9% 28.5%

(a) Studies identified as directly relevant to the review topic (in contrast to the full set of included studies).

Lithium as a drug is mainly used for treating manic episodes, uni- and bipolar depression, as well as bipolar disorders (Kibirige et al., 2013). Interestingly multiple meta-analyses report a protective effect on mental health of naturally occurring low-dose lithium occurring in drinking water (tap water) (Barjasteh-Askari et al., 2020; Eyre-Watt et al., 2021; Memon et al., 2020; Vita et al., 2015). A large proportion of these reviews synthesized studies examining suicide rates as the primary outcome. Several of these analyses found highly significant reductions in suicide rates associated with higher lithium concentrations in drinking water. For example, Vita et al. (2015) reported an odds ratio of 0.26 (95% CI: 0.09–0.77). Nearly all studies (8 out of 9) cited in Vita et al. (2015) showed an association between suicide prevention and lithium in drinking water ($\beta = -0.65$, p -value < 0.004), with the effect being more prominent in males ($\beta = -0.61$, p -value < 0.008) than in females ($\beta = -0.46$, $0.05 < p$ -value < 0.06). Another study cited by Vita et al. (2015) observed a negative association between lithium levels in water and the standardized mortality ratio for suicide in females ($\beta = -0.37$, p -value < 0.10), but not in males ($\beta = 0.12$, p -value = 0.597). A Japanese study also found a significant negative association with suicide rates ($R^2 = 0.15$, $\beta = 70.39$, $t = 74.14$, p -value > 0.001). Only one study reported non-significant results for the association between lithium levels in tap water and suicide mortality ($r = -0.03$, p -value = 0.838), however, the authors criticized methodological limitations and suggested that the null finding may have been biased as a result. Barjasteh-Askari et al. (2020) also reported a significant protective association of lithium in drinking water with reduced suicide rates (OR = 0.42, 95% CI: 0.27–0.67, p -value < 0.01). Subgroup analyses indicated that this association remained statistically significant in studies conducted in Japan, but not in a European sample (OR = 0.65, 95% CI: 0.40–1.04, p -value = 0.071). Moreover, Barjasteh-Askari et al. (2020) observed a potential sex-specific effect: the odds for suicide reduction were significant among men (OR = 0.54, 95% CI: 0.35–0.84, p -value < 0.01), but not among women (OR = 0.70, 95% CI: 0.48–1.01, p -value = 0.057). The authors suggested that men may be more responsive to the protective effects of lithium (Barjasteh-Askari et al., 2020). Memon et al. (2020) also investigated lithium concentrations in tap water, measured through water samples, as in previous reviews, and the association with suicide mortality rates. Of the nine studies included, seven reported a protective association, five of which were statistically significant. The meta-analysis conducted by the authors likewise yielded significant results ($\beta = -0.27$, 95% CI: -0.47–0.08, p -value = 0.006, $I^2 = 83.3\%$). Sex-specific analyses were performed by examining male and female suicide mortality rates separately. For men, the meta-analysis found a negative but non-significant association ($\beta = -0.26$, 95% CI: -0.56–0.03, p -value = 0.08, $I^2 = 91.9\%$), while the female suicide rate significantly reduced with higher lithium levels in drinking water (tap water) ($\beta = -0.13$, 95% CI: -0.24–0.02, p -value = 0.03, $I^2 = 28.5\%$). The authors further suggested that lithium medication use within the population may act as a potential effect modifier of these associations. The last review focusing on lithium is by Eyre-Watt et al. (2021). While individual studies reported either non-significant protective trends or no association at all between lithium exposure and e.g. major depressive disorder or bipolar disorder, the meta-analysis conducted by the authors, comprising predominantly longitudinal studies (20 out of 27), found a significant protective association between lithium concentrations in drinking water and suicide rates ($r = -0.191$, 95% CI: -0.287–0.090, p -value < 0.001). Overall, the reviews indicate a robust and statistically significant trend suggesting that lithium in tap water may be associated with a reduction of suicide rates, with the effect being more pronounced in men in some studies and in women in others. The mechanism of action of lithium in drinking water remains incompletely understood. Several authors have proposed that the mood-stabilising, anti-aggressive, and anti-violent properties may underlie its potential to reduce suicide rates (Barjasteh-Askari et al., 2020; Memon et al., 2020; Vita et al., 2015). Eyre-Watt et al. (2021) further suggest that lithium may affect multiple neurobiological processes, such as increasing the availability of vitamin B12, which could contribute to mood improvement, and exhibiting anti-inflammatory properties. Memon et al. (2020) further

proposed that prolonged low-dose exposure may exert cumulative effects, which could, in turn, contribute to lithium's potential protective role against suicide.

This umbrella+ review showed that suicide has been referred to several times as a mental health outcome. However, since suicide is not a mental illness per se, a brief explanation is needed as to why suicide was nevertheless included as an important mental health outcome that should not be underestimated. Suicide is widely recognized as a significant indicator of mental health problems, given its strong association with a range of psychiatric disorders. Evidence from this umbrella+ reviews also suggests that suicide can be linked to depression, schizophrenia, and substance use disorders (Eyre-Watt et al., 2021). Mood disorders, in particular, are associated with a substantially elevated suicide risk, estimated to be 10-20 times higher than in the general population (Vita et al., 2015). Lifetime suicide mortality has been estimated at 2.2 to 4% for unipolar depression and around 5% for schizophrenia, with elevated prevalence also observed in substance use disorders (Vita et al., 2015). Barjasteh-Askari et al. (2020) indicate that there is approximately ten times the risk to commit suicide in individuals with mood disorder (6-10%) in comparison to the non-psychiatric populations. Suicide is a "multifactorial event caused by a complex interaction between biological, genetic, psychological, social and environmental factors" (Vita et al., 2015). Given these associations, suicide is often considered a meaningful indicator of underlying mental health conditions in epidemiological research, even though causality cannot be assumed.

During the analysis of the reviews, several limitations repeatedly occurred, which were acknowledged by the authors. The authors criticized the lack of evidence for causal relationships and thereby emphasize the necessity for further empirical investigation (Barjasteh-Askari et al., 2020; Vita et al., 2015). Another frequently mentioned limitation is the heterogeneity of methodological approaches in the reviewed human epidemiological studies, such as differences in the assessment of mental health outcomes and limitations related to exposure measurement (Eyre-Watt et al., 2021; Memon et al., 2020). Barjasteh-Askari et al. (2020) and Memon et al. (2020) noted that in some studies, the outcome was measured prior to the exposure, or that the time interval between exposure and outcome assessment was quite long, which raises concerns regarding the validity of the findings. A further methodological concern, highlighted by Vita et al. (2015) and Barjasteh-Askari et al. (2020), relates to lithium intake: tap water is not the single dietary source of lithium, and other potential sources, such as bottled water, were not considered in some analyses. Another limitation is the various confounders. Nearly all reviews reported to consider several potential confounders, such as residence region (Barjasteh-Askari et al., 2020), local climate, healthcare provision (Eyre-Watt et al., 2021; Memon et al., 2020), as well as age, gender, population size and density and urbanicity (Eyre-Watt et al., 2021). These factors could all influence both exposure levels and mental health outcomes, thereby complicating the interpretation of the observed associations.

One important point warrants consideration. While the studies presented may portray lithium as a near "miracle agent" in suicide prevention, it is important to acknowledge that low-dose lithium exposure may also entail adverse effects, also in healthy individuals. For example, Vita et al. (2015) noted that lithium exposure may impair thyroid function, especially in pregnant women and in children exposed prenatally. Other literature, that was not included in this systematic search also emphasised the potential adverse effects of low-dose lithium exposure on health. Both Broberg et al. (2011) and Harari et al. (2015) each conducted cohort studies on the association between lithium concentration in tap water and impaired thyroid function in pregnant women, with exposure assessed via urinary or blood measurements. Both studies found a link between impaired thyroid function and elevated lithium concentration. Harari et al. (2015) in particular emphasised the relevance of the results in relation to the unborn child, noting that the thyroid gland plays a critical role in numerous developmental processes through hormone production. Impaired thyroid function can lead to placental abruption, premature birth, lower birth weight and foetal loss, as well as impaired neurological function in the child. Broberg et al. (2011) additionally observed a non-significant trend

towards higher body mass index (BMI) with increasing urinary lithium concentrations. By contrast, Liew et al. (2023) investigated the effects of low-dose lithium exposure in pregnant women via drinking water on the risk of autism spectrum disorder (ASD) in offspring. This population-based case-control study in Denmark found a moderate increase in ASD risk associated with higher maternal lithium exposure.

Strengths and limitations

This umbrella+ review is the first to compile the existing literature on the effects of exposure to ambient air pollution, noise and environmental chemicals on mental health. This section on environmental chemicals discusses the strengths and weaknesses. The specific selection of reviews in the search process allows for a focus on the general population. By excluding occupational contexts, active smoking and chemical exposure with suicidal intent, the target group is precisely defined. It is important to include vulnerable groups, especially children, adolescents and pregnant women (including prenatal exposure), in order to investigate the effects of environmental chemicals during such fragile phases.

However, several limitations must be acknowledged. First, due to the exclusion criteria and the focus on reviews addressing environmental chemicals and mental health, while excluding neurodegenerative diseases, the pool of eligible studies was limited. Furthermore, many included reviews primarily synthesized evidence from cross-sectional or case-control studies. Cross-sectional designs are limited by their single time-point measurement of both the exposure and the outcome, which does not allow determining the directionality of associations and therefore constrains causal inference. In case-control studies, exposure is typically assessed retrospectively and often relies on self-reports, while the sample selection is often not random and not representative. This can lead to recall and selection biases of the study results. Furthermore, a clear statement about causal associations is often not possible due to the mostly retrospective reconstruction of exposure. A further limitation is that the included reviews rarely stratify results by underlying study design, making it difficult to draw robust conclusions about causality. This is an inherent drawback of the umbrella+ review approach: unlike systematic reviews of primary studies, which may exclude certain designs such as cross-sectional studies, such restrictions are generally not possible in umbrella+ reviews. A considerable proportion of the included reviews were narrative in nature. Unlike systematic reviews or meta-analyses, narrative reviews often rely on non-systematic and potentially subjective literature selection and typically do not incorporate quantitative analyses. Consequently, narrative reviews are particularly prone to selection and confirmation biases, while publication bias frequently remains undetected.

Another limitation, which was already mentioned by several authors of the reviews is the considerable heterogeneity across the reviewed studies. Both exposure assessment and outcome measurement varied substantially, not only between reviews but also among the primary studies they included. Such variation can contribute to divergent findings. For example, Ni et al. (2018) reported a significant positive association between blood copper concentrations and depression whereas copper exposure assessed via hair samples showed no significant association. Similarly, differences in the operationalisation of SHS exposure, such as coding home exposure as a binary variable versus measuring it on a continuous scale, may influence results, as could variations in the temporal frame (e.g., cigarettes per day versus days per week). Moreover, the tools used to assess mental health outcomes ranged from self-reported symptom questionnaires to clinical diagnoses, introducing additional variability and potential bias. This raises the question of how conclusive the evidence can be when methodological approaches differ markedly across studies. The issue of terminology is also pertinent. In the case of depression in particular, a wide range of terms is used across studies, including 'depression', 'depressive symptoms' and 'major depressive disorder.' Some of these terms, e.g. major

depressive disorder, most likely refer to clinical diagnoses, whereas broader terms such as depressive symptoms allow for greater interpretation and may not indicate a clinical condition. This ambiguity is especially relevant when identifying depression in children, where it remains uncertain whether depressive behaviours and symptoms necessarily reflect a depressive disorder. Since this is often not explained in detail in the reviews, this umbrella+ review on environmental chemicals reflected the terminology used in each included review. It should be noted, however, that ‘depression’ as referred to in this umbrella+ review does not generally define a clinical diagnosis. If a confirmed clinical diagnosis would have been required an inclusion criterion, the number of eligible reviews would likely have approached zero.

Like the limitation that the reviews often do not differentiate between study designs in the results section, there is also the possibility that certain exclusion criteria are not explicitly stated, leading to the inadvertent inclusion of studies that should have been excluded. This is particularly the case for ambient air pollution and occupational exposure, especially for pesticides. In some cases, studies fail to specify that the exposure is occupational in nature or that the environmental chemicals are solely airborne, whereas in other cases such details are explicitly mentioned. In the latter case, this information led to the exclusion of two meta-analyses (Wu et al., 2023a; Zhang et al., 2024) from the present synthesis, as one focused on air pollution and the other on occupational exposure.

Another limitation of this umbrella+ review concerns potential biases and the overall quality of the included studies. The reviews that included quality assessment often found a high potential for publication bias, a moderate to low quality of studies or low certainty of the evidence. This is partly attributable to the commonly used study designs discussed above. Additionally, social desirability as a bias, particularly for SHS exposure, must be considered. SHS exposure was often assessed based on self-reports, which is prone to underreporting due to social desirability, especially during pregnancy. Previous research has demonstrated that pregnant women are likely to underreport cigarette consumption (Ford et al., 1997). The issue is compounded when smoking behaviour is assessed retrospectively, in some cases decades later (e.g., up to 31 years) (Hunter et al., 2020), further increasing the potential for recall bias.

Another limitation, stemming from the heterogeneity of the included reviews, concerns the handling of confounders. While some reviews did not address this issue at all, others criticised that the primary studies assessed potential confounders only sporadically. Among the reviews that did report on this, a wide range of possible confounding variables was mentioned, including among others urbanisation, maternal alcohol consumption, low birth weight, socioeconomic status, medication use, population density, and genetic factors. Overall, no single confounder emerged as a consistently emphasised factor across multiple reviews.

One final methodological consideration is that the title and abstract screening was carried out by one reviewer, while the full-text screening and data extraction were performed by another reviewer. This means that each screening stage involved only a single reviewer rather than both reviewers conducting all stages. Nevertheless, both reviewers maintained communication throughout the process, and unclear cases during the full-text screening were discussed jointly. In instances where uncertainties remained, a third reviewer was consulted to reach consensus.

Implications for practice and science

This umbrella+ review on environmental chemicals urges the need for further research. For several substances, such as phthalates or certain metals (e.g., copper and manganese), the available evidence remains highly inconclusive. More research is needed to clarify the effects of these pollutants on mental health, enabling the identification of potential risks and the development of appropriate policy

measures. This umbrella+ review also revealed an imbalance in the range of both exposures and outcomes investigated. In particular, most research to date has focused on depression as a mental health outcome, with far fewer studies on anxiety, schizophrenia, or substance use disorders. Entirely different mental health outcomes, such as phobic disorders, personality disorders, psychosexual dysfunctions, post-traumatic stress disorder, or eating disorders, have to our knowledge not yet been addressed in the existing research. A similar concentration is observed for exposures, where only a few substance groups dominate the literature.

Limitations identified by the included reviews, as well as those inherent to this umbrella+ review, highlight the need for study designs capable of addressing causal relationships, particularly longitudinal designs. Given the large proportion of cross-sectional studies, causal statements remain limited, even in cases where associations appear strong, e.g. in the case of SHS. Greater use of systematic reviews and meta-analyses would further strengthen the evidence base. Additionally, a standardisation of the methodology within this research field is essential. Uniform, validated exposure measurements and outcome assessments would enhance comparability across studies and reviews. One additional research topic for the future should be the research on gender-specific effects. In particular for the EDC bisphenol A and the metal lithium this umbrella+ reviews indicated such effects, although the findings were not consistent across all reviews and were also contradictory, sometimes indicating greater effects in men and sometimes in women. Furthermore, multidisciplinary collaboration, with researchers from environmental sciences, neurobiology, psychology and psychiatry, would be necessary to identify the biological mechanisms of action of the pollutants.

With regard to study populations, the included reviews showed a representation of the general population but also vulnerable groups such as children, adolescents and pregnant individuals. However, people with pre-existing conditions or older adults were only mentioned sporadically in the research so far. Explicit analyses of the European population are also largely absent. While some primary studies were conducted in Europe or the EEA regions, there was only one detailed analysis for the European region, finding no evidence for a protective association between lithium in drinking water and reduced suicide rates (Barjasteh-Askari et al., 2020).

In this umbrella+ review, the mental health outcomes attention deficit hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) were not considered. Previous research by Bessems et al. (2023) has already examined potential associations between chemical exposures and ADHD or ASD. Their umbrella+ review reported strong evidence for a link between pesticide exposure, particularly pyrethroids, and ADHD, as well as suggestive evidence for associations between pesticide exposure and ASD, and between lead exposure and ADHD. Future research within the ETC-HE framework will also aim to provide an updated assessment of the relationships between environmental chemical exposures and mental health outcomes such as ADHD and ASD.

Several policy implications can be drawn from this umbrella+ review. Vulnerable groups, particularly pregnant women, should be protected, as exposure to pollutants during pregnancy affects both the mother and the unborn child during a critical developmental period. Public awareness initiatives should aim not only to promote smoking cessation during pregnancy but also to raise awareness about pesticide exposure, with the goal of reducing such exposures through targeted education on their potential mental health effects, particularly when they occur early in life. Furthermore, expanded monitoring and surveillance of both environmental chemical exposures and mental health outcomes would facilitate longitudinal research.

Overall, this umbrella+ review highlights the multifaceted impact of environmental chemicals on mental health and calls for longitudinal studies, as well as a broader scope in both chemicals and mental health outcomes investigated.

5.2.8 Conclusion

This umbrella+ review synthesised the existing evidence on the association between environmental chemical exposure and mental health outcomes. While certain exposures, such as SHS, Pb and pesticides, demonstrated more consistent associations with outcomes like depression, anxiety and schizophrenia, the evidence for many other chemicals, including phthalates and several metals, remain inconclusive. The findings of this umbrella+ review remain nonetheless highly relevant to the scientific community, as they not only identify significant research gaps but also provide valuable evidence on vulnerable populations, particularly regarding the effects of environmental chemicals and prenatal exposure.

The research on the topic of exposure to environmental chemicals and mental health outcome so far focused mostly on depression, with far less attention given to other mental disorders. Additionally, methodological heterogeneity of the reviews, but also the included studies and limited causal evidence, constrain the strength of the conclusions. Future research should prioritise longitudinal study designs, adopt standardised exposure and outcome assessments, investigate a broader range of both environmental chemicals and mental health outcomes, and studying the European population.

5.3 Noise and mental health

5.3.1 Study characteristics

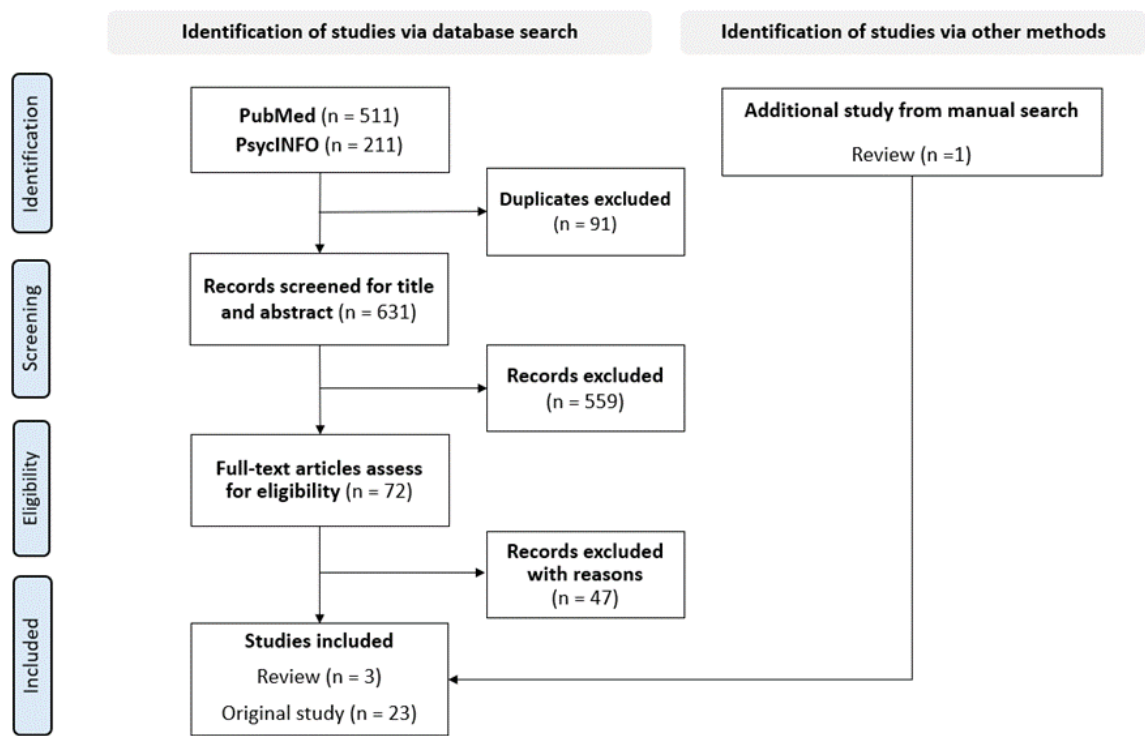
In total, 722 articles published between 2015 and 2025 were identified. After removing duplicates, 631 records remained for screening. Following title and abstract screening, 72 articles underwent full-text assessment, comprising 62 original studies and 10 reviews.

The reviews mainly addressed depression, anxiety, and poor mental health, often drawing on overlapping sets of original studies. To avoid redundancy while capturing the most up-to-date evidence, we selected the most recent high-quality review for each outcome and supplemented it with original studies published thereafter.

For depression and anxiety, three non-systematic reviews (Chen et al., 2023; Hahad et al., 2025; Ordóñez-Iriarte, 2020) and five systematic reviews without meta-analyses (Tortorella et al., 2022; Zaman et al., 2022; Dickerson et al., 2020; Zare Sakhvidi et al., 2018; Clark et al., 2020) were excluded. The most recent high-quality review was Hegewald et al. (2020). One older review (Dzhambov and Lercher, 2019) was thus excluded to avoid overlap. For other mental health disorders, we relied on the WHO ENG review (Clark and Paunovic, 2018) and its updated systematic review (Clark et al., 2020). Although it does not provide meta-analyses, it offers the most comprehensive synthesis of non-depression and non-anxiety outcomes. Children's mental health outcomes and behavioural or neurodevelopmental disorders were not a primary focus of this review, as these were recently synthesised in an umbrella+ review and burden of disease assessment (EEA, 2025).

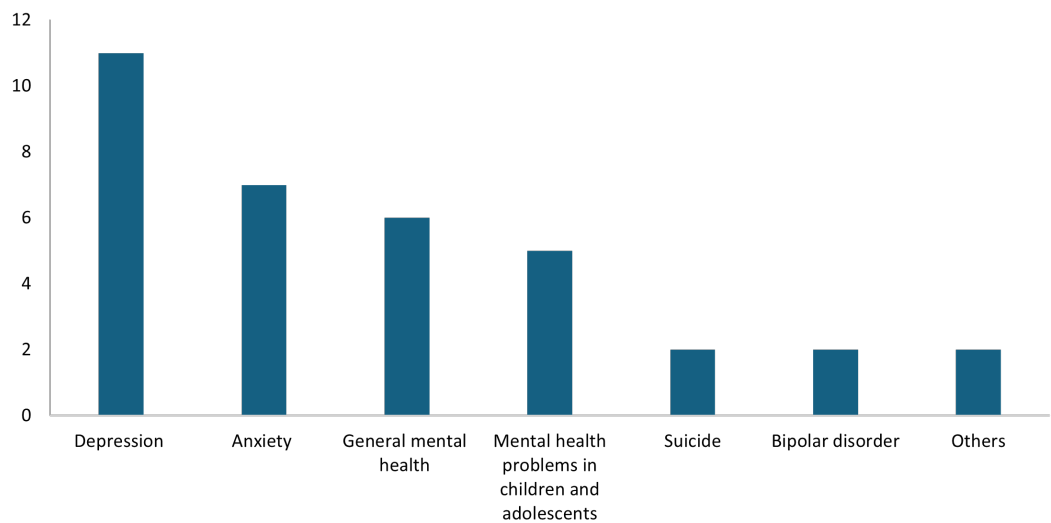
Among the 62 original studies retrieved, 23 were excluded due to subjective noise assessment ($n = 5$), proxy exposure measures ($n = 6$), ineligible outcomes ($n = 2$), non-European study populations ($n = 4$), non-eligible study designs ($n = 5$), or non-English language ($n = 1$). A further 16 studies were excluded because they were already included in one of the retained reviews (Hegewald et al., 2020; Clark et al., 2020; EEA, 2025). This process resulted in three reviews (Hegewald et al., 2020; Clark et al., 2020; EEA, 2025) and 23 original studies (Cadman et al., 2024; Tsimpida and Tsakiridi, 2024; Wicki et al., 2023; Motoc et al., 2023; Gómez González et al., 2023; Díaz et al., 2020; Stansfeld et al., 2021; Dzhambov et al., 2019; Roberts and Helbich, 2021; Nobile et al., 2023; Eze et al., 2020; Lercher et al., 2025; Lan et al., 2022; Zijlema et al., 2024; Hao et al., 2022; Bouter et al., 2023; Pelgrims et al., 2021; Dédelé et al., 2025; Gomm and Bernauer, 2023; Li et al., 2021; Thompson et al., 2024; Wicki et al., 2024; Clark et al., 2021) being included in the analysis. The selection process is summarised in the PRISMA diagram (Figure 5.8).

Figure 5.8 PRISMA flow chart for the systematic search and identification of reviews and original studies with focus on mental health outcomes in association with noise



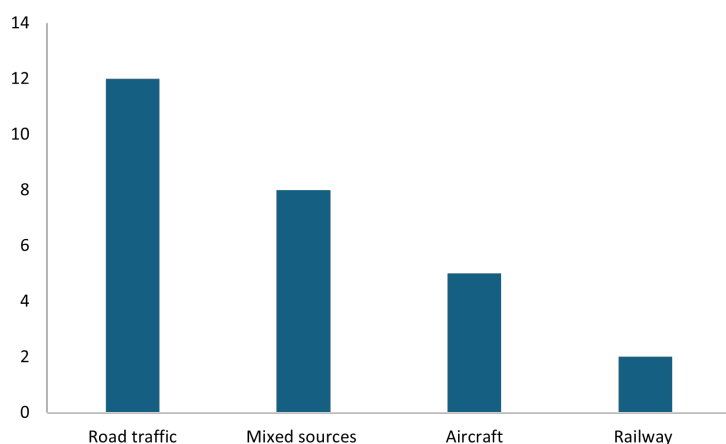
In the 23 original studies identified in our updated search, five focus on children and adolescents, two on the general population, and the remaining 16 on adults. Some studies assess more than one noise source or mental health outcome. For adults and the general population, the most frequently investigated outcome is depression (n = 11), followed by anxiety (n = 7), general mental health (n = 6), suicide (n = 2), bipolar disorder (n = 2), and several other psychiatric disorders and psychotropic drug prescriptions (n = 2). For children and adolescents, the five studies address general mental health (n = 2), depression (n = 1), anxiety (n = 1), and behavioural problems (n = 3). An overview of outcome distribution is provided in Figure 5.9.

Figure 5.9 Distribution of included original studies by mental health outcome category



Road traffic noise was the most commonly assessed exposure ($n = 12$), followed by combined transportation sources ($n = 8$), aircraft noise ($n = 5$), and railway noise ($n = 2$) (Figure 5.10).

Figure 5.10 Distribution of included original studies by transportation noise source



5.3.2 Biological mechanisms

Transportation noise may affect mental health through multiple, interconnected biological and psychological pathways. According to the “noise–stress concept” (Babisch, 2002), two routes are distinguished: a direct pathway, in which very high noise levels (> 100 dB(A)) can cause auditory damage (e.g. hearing loss, tinnitus) and a more relevant indirect pathway, where lower-level noise (typically 50–70 dB(A)) interferes with communication, daily activities, and especially sleep (Hahad et al., 2025).

The direct pathway refers to the physiological effects of noise, particularly at high intensities, which not only cause physical damage to the inner ear that leading to hearing loss and sleep deprivation, but also disrupt normal brain signalling. This disruption can result in altered neural activity and stress responses, further contributing to neuroinflammation and oxidative stress in the brain (Münzel et al., 2018). The resulting inflammation and oxidative damage can impair neuronal function and promote the development and progression of neurodegenerative and mental health disorders (Kuntić et al., 2024; Beurel et al., 2020; Hahad et al., 2025).

Most people are exposed to noise through the indirect pathway. This involves psychological and emotional responses to noise exposure, even at lower intensities (Hahad et al., 2025). Chronic or moderate noise can lead to annoyance, sleep disturbance, and increased stress, which in turn activate the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system (SNS), leading to increased stress hormone levels (Ulrich-Lai and Herman, 2009). This stress response drives the production of reactive oxygen species (ROS) and pro-inflammatory cytokines, which can contribute to neuroinflammation by activating microglia and astrocytes in the brain (Beurel et al., 2020; Galea, 2021). This neuroinflammatory state disrupts neuronal function and is implicated in the pathogenesis of depression, anxiety, cognitive impairment, and neurodegenerative disorders (Kuntić et al., 2024; Hahad et al., 2025).

Both pathways may ultimately converge on stress systems and activate the HPA axis and the SNS, contributing to neuroinflammation and oxidative stress in the brain. The effects from the direct pathway are most relevant at very high noise exposures, but may interact with indirect pathways at lower exposures, and these effects are further amplified by stress system activation. Chronic activation of these stress systems also contributes to dysregulation of circadian rhythms, reduced coping capacity

(Hahad et al., 2019), and maladaptive behavioural responses such as alcohol and tobacco use (Roswall et al., 2017, 2018; Foraster et al., 2016). These processes provide biological plausibility for observed associations between transportation noise and a range of mental health outcomes, although the relative contribution of sleep disturbance versus direct stress and inflammatory pathways remains to be clarified (Hahad et al., 2025)

5.3.3 Depression

Hegewald et al. (2020) included 20 studies examining depression in relation to transportation noise, 10 of which were conducted in Europe. Outcomes were assessed by clinical diagnosis, antidepressant medication use, or depressive symptoms measured with validated screening tools, with most studies using self-reported questionnaires. Most studies were cross-sectional, though several cohort and case-control studies were also included. The review covers noise from road traffic, railway, and aircraft sources. For road traffic noise (11 out of 12 studies) and railway noise (3 studies identified), pooled estimates suggest small, non-significant risk increases of around 2–3% per 10 dB L_{den} increase. A meta-analysis of five out of seven identified studies on aircraft noise indicates a pooled relative risk of 1.12 (95% CI: 1.02–1.23) for depression per 10 dB increase in L_{den} . Studies not included in the meta-analysis generally use different outcome or exposure assessments or have insufficient data for pooling and generally report weak or non-significant associations. Two studies concerning mixed noise sources from road traffic, railway, and aircraft combined are included. Both studies find weak or non-significant associations between mixed-source transportation noise and depression.

Since the end of the search period of this review (December 2019), 11 further original studies on depression in adults have been identified (Motoc et al., 2023; Li et al., 2021; Pelgrims et al., 2021; Hao et al., 2022; Eze et al., 2020; Nobile et al., 2023; Roberts and Helbich, 2021; Dzhambov et al., 2019; Díaz et al., 2020; Motoc et al., 2023; Tsimpida and Tsakiridi, 2024; Cadman et al., 2024), including one earlier study by Dzhambov et al. (2019) that meets our inclusion criteria but was not included in Hegewald et al. (2020).

These studies vary in outcome measurement, with seven assessing self-reported depressive symptoms and four assessing clinically diagnosed depression or antidepressant prescription. Study populations range from several hundred to 3.2 million participants, with designs including cross-sectional surveys, longitudinal cohorts with up to 10 years of follow-up, one ecological time-series, and one natural experiment assessing the effect of a noise control policy at Schiphol Airport (Li et al., 2021). Road traffic noise is the most common exposure, followed by combined sources (road, rail, aircraft, and also additional industry and wind turbine noise), and aircraft noise alone. Most studies used modelled façade levels expressed as L_{den} or L_{Aeq} . Most analyses adjusted for a broad range of sociodemographic, lifestyle, and contextual variables, and several additionally account for area-level deprivation, neighbourhood characteristics, or greenness. Seven of the studies accounted for air pollution exposures. Study characteristics are summarised in Table 5.32.

Table 5.32 Characteristics of the identified original studies for noise and depression

First author, year	Country	Study design (follow-up)	Study population (N, age)			Outcome (Prevalence/Incidence, tool)	Noise source(s), place	Adjustment for air pollution	Confounders adjusted for in analyses	Exposure metric, increment †	Results (Risk estimates, 95% CI) †‡
Self-reported depression											
Cadman, 2024	NL, UK, DK, FR, NL, ES, NO, IT, GR	Pooled analysis of 12 European cohorts	N = 23,956	Women, 96% aged 21-40 yrs	P	Postpartum depression (11/12 cohorts using validated questionnaires)	Road, at home	PM _{2.5}	Maternal education, disposable family income, area-specific deprivation, parity, maternal age, year and season of birth, population density, and NDVI	L _{den} , ≥ 65 dB vs. < 65 dB	OR = 1.12, (0.94–1.33)
Motoc, 2023	NL	Cohort (10-yr)	N = 1,365	Mean 70	I	Depressive symptoms (CES-D)	Road, rail, and aircraft combined, at home	PM _{2.5}	Age, sex, education, income, and years in neighbourhood	L _{den} , 3.3 dB	HR = 1.09, (0.95–1.26)
Hao, 2022	UK	Cross-sectional	N = 90,706	Mean 56	P	Depressive symptoms (PHQ, SCID-I, help seeking for mental health)	Road, at home	-	Age, sex, ethnicity, education, assessment centre	L _{den} , ≥ 57.8 dB vs. < 52.1 dB	OR = 1.00, (0.94–1.06)
Roberts, 2021	NL	Cross-sectional	N = 393	Mean 45	P	Depressive symptoms (PHQ-9)	Road, rail, aircraft, industry, and wind	PM _{2.5}	Age, sex, employment, education, marital status, household	L _{den} , 5.53 dB	β = 0.057, SE: 0.049

First author, year	Country	Study design (follow-up)	Study population (N, age)		Outcome (Prevalence/Incidence, tool)		Noise source(s), place	Adjustment for air pollution	Confounders adjusted for in analyses	Exposure metric, increment †	Results (Risk estimates, 95% CI) †‡
							turbine combined, at home and along GPS-tracked mobility paths (7 days)		type, origin, income, population density, social fragmentation and deprivation		
Pelgrims, 2021	BE	Cross-sectional	N = 1,325	Median 47	P	Depressive disorder (SCL-90-R)	Road, rail, and aircraft combined, at home	BC	Age, sex, family composition, household income, highest educational level in the household, year, social support, physical activity, and chronic condition	L _{den} , 1 dB	OR = 1.03, (0.99–1.06)
Li, 2021	NL	Natural experiment	N = 1,746	Mean 73	P	Depressive symptoms (CES-D)	Aircraft, at home	-	Age, marital status, employment, retirement, income, physical functioning, and year	Pre and post policy comparison	Change in score = 0.044, SE: 0.704

First author, year	Country	Study design (follow-up)	Study population (N, age)		Outcome (Prevalence/Incidence, tool)		Noise source(s), place	Adjustment for air pollution	Confounders adjusted for in analyses	Exposure metric, increment †	Results (Risk estimates, 95% CI) †‡
Dzhambov, 2019	BG	Cross-sectional	N = 437	Mean 21	P	Depressive disorder (PHQ-9)	Road, at home	-	Age, sex, nationality, income adequacy, population density, and university faculty	L _{Aeq} , 5 dB	OR = 0.71, (0.31–1.61)
Clinical diagnosis or drug prescription of depression											
Eze, 2020	CH	Cohort (9-yr)	N = 4,581	Mean 52	I	Depression (physician diagnosis/antidepressant use/short form-36 mental health score)	Road, rail, and aircraft, at home	NO ₂	Age, sex, education, neighbourhood socioeconomic position, study area, smoking, alcohol use, fruit and vegetable intake, family history of depression, and residential greenness	L _{den} , 10 dB	IRR, road = 1.07, (0.93–1.22) IRR, rail = 0.88, (0.76–1.03) IRR, aircraft = 1.20, (0.92–1.55)
Nobile, 2023	IT	Cohort (8-yr)	N = 1.7 mio	Mean 56	I	Hospitalizations of depression, prescriptions of antidepressants	Road, at home	PM _{2.5} , BC, UFP	Age, sex, marital status, place of birth, citizenship, education level, employment status, socioeconomic	L _{den} , 9.43 dB	HR, depression = 1.057, (0.995–1.123) HR, antidepressant

First author, year	Country	Study design (follow-up)	Study population (N, age)		Outcome (Prevalence/Incidence, tool)		Noise source(s), place	Adjustment for air pollution	Confounders adjusted for in analyses	Exposure metric, increment †	Results (Risk estimates, 95% CI) †‡
									deprivation index, and area level variables (unemployment rate, percentage of graduates, and house prices)		t = 1.008, (0.999–1.016)
Díaz, 2020	ES	Ecological time-series	N = 3.2 mio	All ages	I	Number of emergency hospital admissions for depression and others	Road, city monitoring network	NO ₂ , PM ₁₀ , PM _{2.5} , O ₃	Long-term trends, seasonal patterns, and day of the week	L _{Aeq} , 1 dB	RR = 1.11*, (1.06–1.16)
Tsimpida, 2025	UK	Cross-sectional	N = 2.7 mio	Adults	P	Depression from medical records	Road and rail combined, area-level (LSOA) based	-	Area-level deprivation: income, employment, education, health, crime, housing, and living environment deprivation	1% ↑ in noise coverage above 55 dB in the study region	β = 0.015926*, SE: 0.002496, p = 0.00000*

Abbreviations:

BG = Bulgaria; NL = the Netherlands; BE = Belgium; UK = United Kingdom; ES = Spain; DK = Denmark; FR = France; NO = Norway; IT = Italy; GR = Greece; CH = Switzerland.

PHQ = Patient Health Questionnaire; CES-D = Center for Epidemiologic Studies Depression Scale; SCL-90-R = Symptom Checklist-90-Revised; SCID-I = help-seeking for mental health

P = Prevalence; I = Incidence; LSOA: Lower Super Output Area, unit of analysis; L_{Aeq} = Equivalent Continuous Sound Level; L_{den} = Day-Evening-Night Equivalent Level; BC = Black carbon; UFP= Ultrafine Particles; NDVI = Normalized Difference Vegetation Index; CI = Confidence Interval; OR = Odds Ratio; SE = Standard Error; HR = Hazard Ratio; RR = Relative Risk; IRR = Incidence Rate Ratio.

* Statistically significant results.

† Results are presented as reported in the original studies, with varying exposure increments or comparison groups.

‡ Risk estimates are air pollution-adjusted when available; unadjusted estimates are noted where applicable.

Self-reported depression

Seven studies assessed depression using validated screening instruments or self-reported measures, including the Patient Health Questionnaire-9 (PHQ-9), Symptom Checklist-90-R (SCL-90R), Center for Epidemiological Studies Depression Scale (CES-D), and Structured Clinical Interview for DSM-IV Disorders (SCID) (Dzhambov et al., 2019; Li et al., 2021; Pelgrims et al., 2021; Roberts and Helbich, 2021; Hao et al., 2022; Motoc et al., 2023; Cadman et al., 2024), one of which focused specifically on postpartum depression (Cadman et al., 2024). All studies report weak associations that are not statistically significant.

Two studies with longitudinal or pooled design have been identified (Motoc et al., 2023; Cadman et al., 2024). In a nationally representative Dutch cohort of 1,365 older adults, Motoc et al. (2023) tracked participants over ten years and report a 9% increase in depressive symptoms per 3.3 dB increase in combined transportation noise (road, rail, and aircraft), though this association is not statistically significant. Similarly, in a pooled analysis of 12 European birth cohorts, Cadman et al. (2024) analysed 23,956 women with available residential road traffic noise exposure data and observe a 12% increased odds of postpartum depression among those exposed to high levels (≥ 65 dB) during pregnancy, but again the association does not reach statistical significance.

Four cross-sectional studies investigate transportation noise in relation to depression, but none provide convincing evidence of an effect (Pelgrims et al., 2021; Roberts and Helbich, 2021; Dzhambov et al., 2019; Hao et al., 2022). In Brussels, Pelgrims et al. (2021) analysed combined road, rail, and aircraft noise exposure among 1,325 adults and observed a small, non-significant increase in the odds of depressive symptoms with higher residential noise exposure (around 3% per 1dB L_{den} increase). Roberts and Helbich (2021) assessed a nationwide Dutch sample of 393 adults, estimating noise exposure at both residence and along daily mobility paths using GPS tracking, and found that higher exposure to combined noise from transportation, industry, and wind turbines is associated with a small, non-significant increase in depressive symptoms. Two further studies focused specifically on road traffic noise: Dzhambov et al. (2019) reported null findings among 437 Bulgarian university students, while Hao et al. (2022), analysing over 90,000 UK Biobank participants, also found no evidence of elevated depression risk at higher exposure levels.

The only natural experiment evaluated the impact of a noise control policy implemented at Schiphol Airport, Amsterdam, in 2008–2009 (Li et al., 2021). The study compared depressive symptoms before (2005/2006) and after policy implementation (2011/2012) among a treated group of older adults living close to the airport and a control group living farther away. In line with the study's finding of no reduction in aircraft noise levels following the policy, no difference in depressive symptom scores was observed among 1,746 older adults post-policy.

Clinically diagnosis depression or drug for depression prescription

Four studies assessed depression using clinical diagnoses from medical records, antidepressant prescription data, or hospital admissions (Eze et al., 2020; Nobile et al., 2023; Tsimpida and Tsakiridi, 2024; Díaz et al., 2020).

Two large cohort studies in Switzerland (Eze et al., 2020) and Italy (Nobile et al., 2023) addressed the associations between transportation noise and depression by adjusting for sociodemographic, lifestyle, and environmental confounders, including air pollution (Table 5.32). The Swiss SAPALDIA cohort (Eze et al., 2020) followed 4,581 adults aged 29–73 years between 2001 and 2010/2011. Depression was defined as a composite outcome of doctor diagnosis, antidepressant use, or low mental health score at follow-up. Analyses suggested weak, non-significant positive associations for road traffic and aircraft noise, and no evidence of an effect for railway noise. The Italian population-

based cohort (Nobile et al., 2023) followed adults aged 30 years and older from 2011 to 2019 using census data in Rome. Outcomes were defined as new cases of mental disorders identified from hospital discharge records (ICD-9 codes) or by at least two prescriptions of psychiatric drugs (antipsychotics, antidepressants, or mood stabilizers) within a six-month period. In over 1.7 million participants, small increases in depression risk were also observed in relation to road traffic noise, but these associations did not reach statistical significance for either hospitalisations or antidepressant prescriptions.

By contrast, the other two studies reported statistically significant increased risks. Tsimpida and Tsakiridi (2024) conducted a spatial analysis of neighbourhoods in two English counties using 2019 primary care data. They found that greater neighbourhood-level exposure to road traffic and railway noise was linked to higher depression prevalence, and for every 1% increase in the proportion of a neighbourhood's area exposed to noise above 55 dB, depression prevalence increased by 0.016%. Similarly, Díaz et al. (2020) investigated acute effects in an ecological time-series of more than 3.2 million residents in Madrid. They found that short-term increases in road traffic noise were linked to elevated risk of emergency admissions for depression, with a 1 dB increase in daytime L_{Aeq} associated with an 11% rise in admissions, after adjustment for long-term trends, seasonality, meteorology, and air pollution. This association was observed for daytime L_{Aeq} on the same day as exposure, after adjustment for long-term trends, seasonality, meteorology, and air pollution. While a limitation is that both studies used aggregated data and could not control for individual-level factors such as socioeconomic status, age, sex, or comorbidities.

Overall, the emerging evidence on depression aligns in part with the findings of Hegewald et al. (2020). While many recent studies reported null or weak associations between transportation noise and depression, the direction of effect estimates is more often positive than negative, and no study indicates a protective effect of noise exposure. Several recent cohort studies (Eze et al., 2020; Nobile et al., 2023; Hao et al., 2022; Motoc et al., 2023) reported small, non-significant increases in risk after adjustment for major confounders. Importantly, two studies observed statistically significant associations in specific contexts, such as short-term effects (Díaz et al., 2020) or neighbourhood-level exposures (Tsimpida and Tsakiridi, 2024). Building on this, a recent pooled analysis of cohort evidence (Hegewald et al., 2020; Eze et al., 2020; Nobile et al., 2023) reported a summary effect of RR 1.038 (95% CI: 1.008–1.069) (Röösli et al., 2025), indicating a small but statistically significant increase in depression risk associated with road traffic noise. Further support comes from Motoc et al. (2023), who observed similar adverse associations for combined transportation noise in a large cohort. Taken together, these findings suggest a possible increased risk of depression linked to transportation noise, particularly for road traffic and in certain settings, although the evidence remains limited by heterogeneity in exposure and outcome assessment. Railway noise and aircraft noise are still understudied.

5.3.4 Anxiety

Hegewald et al. (2020) reviewed 11 studies examining the association between transportation noise and anxiety disorders, with ten conducted in Europe. Anxiety outcomes were assessed using validated screening instruments, prescription or health insurance data for anxiolytic medication use or diagnoses, or self-reported use of anxiolytics. The majority of studies are cross-sectional, with three cohort studies and two case–control studies also included. The review covered noise from road traffic, railway, and aircraft sources. A meta-analysis of six out of nine identified studies on road traffic noise found a non-significant 2% increase in the risk of anxiety per 10 dB L_{den} increase (Effect Size = 1.02, 95% CI: 0.98–1.06). For railway noise (2 studies) and aircraft noise (1 study), there were too few studies to conduct meta-analyses, and the available evidence was limited and inconclusive. Studies not included in the meta-analysis generally used different outcome or exposure assessments or had insufficient data for pooling, and also reported weak or non-significant associations. One study concerning mixed noise sources from road, railway, and aircraft combined was included and found a weak or non-significant association between mixed-source transportation noise and anxiety.

Seven additional original studies on anxiety in adults were identified (Motoc et al., 2023; Díaz et al., 2020; Dzhambov et al., 2019; Nobile et al., 2023; Lan et al., 2022; Pelgrims et al., 2021; Li et al., 2021): three cross-sectional, two cohort, one ecological time-series, and one natural experiment. Two examined combined road, rail, and aircraft sources; one additionally considered industry and wind turbines; three focused on road traffic noise only; and one on aircraft noise. Exposures were typically modelled and linked to residential addresses, with one exception (Lan et al., 2022), which used GPS-tracking to estimate personal exposures. Five studies measured anxiety symptoms via questionnaires, while two relied on hospital admissions, clinical diagnoses, or prescriptions. Most studies adjusted for a broad set of sociodemographic, lifestyle, and contextual variables, and four additionally accounted for air pollution exposures (Table 5.33).

Table 5.33 Characteristics of the identified original studies for noise and anxiety

Author , year	Country	Study design (follow- up)	Study population (N, age)		Outcome (Prevalence/Incidence, tool)		Noise source	Adjustm ent for air pollution	Confounders adjusted for in analyses	Exposure metric, increment [†]	Results (Risk estimates, 95% CI) ^{†‡}
Self-reported anxiety											
Motoc, 2023	NL	Cohort (10-yr)	N = 1,365	Mean 70	I	Anxiety symptoms (HADS-A)	Road, rail, and aircraft combined, at home	PM _{2.5}	Age, sex, education, income, years in neighbourhood	L _{den} , 3.1 dB	HR = 1.10, (0.93– 1.31)
Lan, 2022	NL	Cross- sectional	N = 358	Mean 44	P	Anxiety symptoms (GAD-7)	Road, rail, aircraft, industry, and wind turbine combined, along GPS- tracked mobility paths (7 days)	PM _{2.5}	Age, sex, origin, education, employment, marital status, household type, income, perceived neighbourhood quality, neighbourhood population density & deprivation	L _{den} , 1 dB	β, unstandardised = 0.016, (-0.111– 0.143)
Li, 2021	NL	Natural experim ent	N = 1,746	Mean 73	P	Anxiety symptom (HADS-A)	Aircraft, at home	-	Age, marital status, employment, retirement, income, physical functioning, and year	Pre and post policy comparison	Change in anxiety scores: - 0.259 (SE 0.351) Change in likelihood of having elevated anxiety

Author , year	Country	Study design (follow- up)	Study population (N, age)		Outcome (Prevalence/Incidence, tool)		Noise source	Adjustm ent for air pollution	Confounders adjusted for in analyses	Exposure metric, increment †	Results (Risk estimates, 95% CI) †‡
											symptoms: - 0.016 (SE: 0.022)
Pelgrim s, 2021	BE	Cross- sectional	N = 1,325	Median 47	P	Anxiety disorder (SCL-90R)	Road, rail, and aircraft combined, at home	BC	Age, sex, family composition, reported household income, highest educational level in the household, year, social support, physical activity, and chronic condition	L _{den} , 1 dB	OR = 1.00 (0.97– 1.04)
Dzham bov, 2019	BG	Cross- sectional	N = 437	Mean 21	P	Anxiety symptoms (GAD-7)	Road, at home	-	Age, sex, nationality, income adequacy, population density, and university faculty	L _{Aeq} , 5 dB	OR = 0.93 (0.35– 2.46)
Clinical diagnosis or drug prescription of anxiety											
Nobile, 2023	IT	Cohort (8-yr)	N = 1.7 mio	Mean 56	I	Hospitalizations of anxiety, stress- related and somatoform disorder	Road, at home	PM _{2.5} , BC, UFP	Age, sex, marital status, place of birth, citizenship, education level, employment status, socioeconomic deprivation index, area level variables (unemployment rate, percentage of graduates, and house prices)	L _{den} , 9.43 dB	HR = 1.000 (0.917–1.090)

Author , year	Country	Study design (follow- up)	Study population (N, age)		Outcome (Prevalence/Incidence, tool)		Noise source	Adjustm ent for air pollution	Confounders adjusted for in analyses	Exposure metric, increment †	Results (Risk estimates, 95% CI) †‡
Díaz, 2020	ES	Ecologic al time- series	N = 3.2 mio	All ages	I	Number of emergency hospital admissions for anxiety	Road, city monitoring network	NO ₂ , PM ₁₀ , PM _{2.5} , O ₃	Long-term trends, seasonal patterns, day of the week	L _{Aeq, day} , 1 dB	RR = 1.20* (1.14– 1.26)

Abbreviations:

GAD = Generalized Anxiety Disorder; HADS-A = Anxiety subscale of the Hospital Anxiety and Depression Scale; SCL-90R = Symptom Checklist-90-R.

BG = Bulgaria; NL = the Netherlands; BE = Belgium; ES = Spain; IT = Italy; P = Prevalence; I = Incidence; L_{Aeq} = Equivalent continuous sound level; L_{den} = Day-evening-night equivalent level; BC = Black carbon; UFP = Ultrafine particles; CI = Confidence Interval; OR = Odds Ratio; SE = Standard Error; HR = Hazard Ratio; RR = Relative Risk.

* Statistically significant results.

† Results are presented as reported in the original studies, with varying exposure increments or comparison groups.

‡ Risk estimates are air pollution-adjusted when available; unadjusted estimates are noted where applicable.

Self-reported anxiety

Five studies assessed anxiety symptoms with validated instruments, including Patient Health Questionnaire-9 (PHQ-9), Centre for Epidemiologic Studies Depression Scale (CES-D), Symptom Checklist 90-Revised (SCL-90-R), and Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) (Dzhambov et al., 2019; Li et al., 2021; Pelgrims et al., 2021; Lan et al., 2022; Motoc et al., 2023). Overall, findings suggest weak and non-significant associations across noise sources.

In a Dutch cohort of older adults followed for 10 years, Motoc et al. (2023) observed a non-significant 10% increase in anxiety risk with higher combined transportation noise from road traffic, railway, and aircraft. Three cross-sectional studies reported no associations between transportation noise and anxiety. Dzhambov et al. (2019) analysed 437 university students in Bulgaria and found no association between residential road traffic noise and anxiety symptoms. Regarding combined sources, Pelgrims et al. (2021) examined 1,325 adults in Brussels and reported no associations with transportation noise from road, rail, and aircraft. Similarly, Lan et al. (2022) used a GPS-based approach to estimate exposure to road, rail, aircraft, industrial, and wind turbine noise along daily mobility paths among 358 Dutch adults and observed no associations with anxiety symptoms.

For aircraft noise, in the Schiphol Airport natural experiment, Li et al. (2021) found no post-policy differences in anxiety symptoms among older adults, consistent with the lack of change in aircraft noise levels.

Clinically diagnosis anxiety or drug for anxiety prescription

Two large-scale studies evaluated clinically diagnosed anxiety or related hospital admissions (Díaz et al., 2020; Nobile et al., 2023). Findings here are mixed.

In Madrid, Díaz et al. (2020) analysed over 3.2 million residents in an ecological time-series and reported acute effects of road traffic noise: each 1 dB increase in daytime L_{Aeq} was associated with a 20% rise in same-day emergency hospital admissions for anxiety. In contrast, Nobile et al. (2023) followed 1.7 million adults in Rome, based on 1,922 incident cases, found no evidence that long-term road traffic noise is linked to hospital diagnoses of anxiety, stress-related, or somatoform disorders.

Overall, findings from recent studies are heterogeneous and broadly consistent with the earlier review by Hegewald et al. (2020): there is still no strong or consistent association between transportation noise and anxiety. Questionnaire-based studies largely reported null results, and registry-based analyses or ecological studies suggested possible associations, particularly for acute exposures but their design is limited for the research question. However, heterogeneity in exposure assessment (residential vs. mobility-based, single vs. combined sources), outcome definitions (symptom scales vs. clinical diagnoses), and study designs continues to limit comparability. As with depression, the current body of evidence does not support a strong or consistent association between transportation noise and anxiety.

5.3.5 General mental health

Evidence on general mental health outcomes in adults was synthesised in the WHO Environmental Noise Guidelines (ENG) review (Clark and Paunovic, 2018) and an updated systematic review by Clark et al. (2020), together covering studies published between January 2005 and March 2019. Both reviews evaluated environmental noise in relation to wellbeing, quality of life, psychological distress, and self-reported mental health, and concluded that the overall evidence was of very low quality and insufficient to establish clear associations. The WHO ENG review included 17 studies on self-reported quality of life, wellbeing, and mental health, while Clark et al. (2020) included 24 studies, most of which are European studies. Specifically, for road traffic and aircraft noise, the evidence was rated as very

low quality and suggested no clear effects, whereas for railway noise, there was some indication of an effect, but the evidence remained of low-quality. Both reviews highlighted inconsistencies across study designs, exposure metrics, and outcome definitions, and called for more longitudinal research using standardised measures.

Our updated search identified six additional original studies (Gómez González et al., 2023; Stansfeld et al., 2021; Zijlema et al., 2024; Hao et al., 2022; Pelgrims et al., 2021; Gomm and Bernauer, 2023). Four used the self-reported General Health Questionnaire (GHQ), one applied a measure of “nerves, anxiety, tension, or depression” (NATD), and one relied on administrative data on hospital admissions for a broad range of mental health disorders. Populations range from approximately 1,300 to over 330,000 participants. Most studies are cross-sectional, with one longitudinal cohort and one ecological time-series. Four focused specifically on road traffic noise, one assessed combined road, rail, and aircraft noise, and another employed noise data from urban environmental and airport monitoring networks. These studies generally adjusted for a wide range of sociodemographic, lifestyle, and contextual factors, and three additionally controlled for air pollution. Study characteristics are summarised in Table 5.34.

Table 5.34 Characteristics of the identified original studies for noise and general mental health

First author, year	Country	Study design (follow-up)	Study population (N, age)		Outcome (Prevalence/ Incidence, tool)		Noise source	Adjustment for air pollution	Confounders adjusted for in analyses	Exposure metric, increment †	Results (Risk estimate, 95% CI) †‡
Self-reported poor mental health/psychological distress											
Stansfeld, 2021	UK	Cohort (10-yr)	N = 2,398 men	45+	I	Psychological ill-health (GHQ-30)	Road, at home	-	Age, social class, marital status, employment status, smoking status, BMI, alcohol consumption, physical activity at leisure, and noise at work	L _{Aeq} , 16hrs, 56–60 vs. 51-55 dB	First follow-up OR = 1.54, (0.91, 2.59); Final follow-up OR = 1.82*, (1.07, 3.07)
Zijlema, 2024	ES	Cross-sectional	N = 3,145	Mean 50	P	Mental health status, at risk of psychiatric problems (GHQ-12)	Road	-	Age, sex, marital status, place of birth, education, perceived income situation, social class, length of residence, and area-level unemployment rate	L _{den} , 1 dB	OR = 0.998, (0.981–1.015)
Gomm, 2023	CH	Cross-sectional	N = 5,729	Median 52	P	Mental distress (GHQ-12)	Street traffic broadly, at home	PM ₁₀	Age, sex, education, income, urbanization,	L _{Aeq} , 8.6-8.7 dB	β = -0.02, (SE 0.01)

First author, year	Country	Study design (follow-up)	Study population (N, age)		Outcome (Prevalence/ Incidence, tool)		Noise source	Adjustment for air pollution	Confounders adjusted for in analyses	Exposure metric, increment †	Results (Risk estimate, 95% CI) †‡
									chronic disease, area-level SES		
Hao, 2022	UK	Cross-sectional	N = 334,986	Mean 56	P	NATD symptoms	Road, at home	-	Age, sex, ethnicity, education, assessment centre	L _{den} ≥57.8 dB vs. <52.1 dB	OR = 1.04*, (1.01–1.07)
Pelgrims, 2021	BE	Cross-sectional	N = 1,325	Median 47	P	Psychological distress (GHQ-12)	Road, rail, and aircraft combined, at home	BC	Age, sex, family composition, reported household income, highest educational level in the household, year, social support, physical activity, and chronic condition	L _{den} , 1 dB	OR = 1.01, (0.98 –1.05)
Clinical diagnosis of mental disorders											
Gomez Gonzalez, 2023	ES	Ecological time series (retrospective)	67,225 admissions	All ages	I	Daily emergency hospital admissions for mental, behavioural and developmental	Environmental noise from urban and airport monitoring networks	PM _{2.5} , PM ₁₀ , NO ₂ , O ₃	Seasonality, trend, day of week, holidays, autoregressive effects, and meteorological variables	L _{Aeq7-23h} , 1 dB	RR = 1.008*, (1.003–1.013)

First author, year	Country	Study design (follow-up)	Study population (N, age)		Outcome (Prevalence/ Incidence, tool)	Noise source	Adjustment for air pollution	Confounders adjusted for in analyses	Exposure metric, increment †	Results (Risk estimate, 95% CI) †‡
					neurological disorders					

Abbreviations:

CH = Switzerland; ES = Spain; NATD = symptoms of nerves, anxiety, tension, or depression; L_{Aeq} = Equivalent continuous sound level; L_{den} = Day-evening-night equivalent level; SEP = Socioeconomic position; CI = Confidence Interval; RR = Relative Risk; HR = Hazard Ratio.

* Statistically significant results.

† Results are presented as reported in the original studies, with varying exposure increments or comparison groups.

‡ Risk estimates are air pollution-adjusted when available; unadjusted estimates are noted where applicable.

For road traffic noise, in a longitudinal cohort of 2,398 middle-aged men from South Wales, Stansfeld (2021) found higher risk of psychological ill-health with increasing residential road traffic noise, with associations strengthening over time and reaching statistical significance at the final follow-up (OR = 1.82, 95% CI: 1.07–3.07). This finding aligns with results from the large UK Biobank sample, where Hao et al., (2022) reported significant higher odds of NATD (symptoms of nerves, anxiety, tension, or depression) among participants exposed to higher residential road traffic noise levels (OR = 1.04, 95% CI: 1.01–1.07). By contrast, Gomm et al. (2023) observed a small, non-significant negative association between street traffic noise and mental health distress score in Switzerland, and Zijlema et al., (2024) found no direct effect of residential road traffic noise on poor mental health in Barcelona.

For combined transportation noise, Pelgrims et al. (2021) examined 1,325 adults in Brussels and found no associations between road, rail, and aircraft noise combined and mental distress scores. An ecological time-series study from Madrid (Gomm and Bernauer, 2023) shows that higher daytime environmental noise from urban and airport monitoring networks was associated with a 0.8% increase in daily emergency hospital mental health admissions for mental, behavioural, and developmental neurological disorders per 1 dB (RR = 1.008, 95% CI: 1.003–1.013).

To sum up, the new evidence identified since the WHO ENG review (Clark and Paunovic, 2018) provide a somewhat more nuanced picture but largely confirm the earlier assessment. For road traffic noise, findings remain inconsistent: while large scale analyses from the UK biobank and a longitudinal cohort of men in the UK suggested adverse associations, other studies reported null results. Evidence for combined sources is mixed: Pelgrims et al. (2021) reported no long-term effects of noise on mental distress, and Gómez González et al. (2023) suggested that short-term increases in environmental noise may trigger or worsen mental health symptoms, leading to hospital admissions. Taken together, the new evidence strengthens the suggestion of possible adverse associations with road traffic noise, but results remain heterogeneous and of limited quality. For aircraft and railway noise, the evidence base remains scarce and insufficient to draw firm conclusions.

5.3.6 Suicide

No previous reviews have identified studies on transportation noise and suicide. Our updated search yielded two original investigations focusing on road traffic noise (Wicki et al., 2023; Díaz et al., 2020) (Table 5.35).

In Switzerland, Wicki et al. (2023) examined long-term exposure in a nationwide cohort of 5.1 million adults. Road, rail, and aircraft noise were modelled at residential addresses. After adjustment for air pollution, green space, and socioeconomic factors, the study reported a 3.9% higher risk of suicide mortality per 10 dB L_{den} increase in road traffic noise (HR = 1.039, 95% CI: 1.015–1.065) and a 2.2% higher risk per 10 dB L_{den} increase in railway noise (HR = 1.022, 95% CI: 1.004–1.041), while no association was observed for aircraft noise. In Madrid, Díaz et al. (2020) assessed the acute effect of daily road traffic noise on suicide-related hospital admissions in an ecological time-series. After adjusting for long-term trends, season, day of the week, and multiple air pollutants, each 1 dB increase in daytime L_{Aeq} on the previous day was linked to a 17% higher risk of admissions (RR = 1.17, 95% CI: 1.05–1.30).

Taken together, the evidence on transportation noise and suicide remains limited with only two studies. The ecological study suggested short-term increases in suicide-related hospital admissions following higher daily road traffic noise, but such designs are vulnerable to residual confounding and do not establish causality. The large Swiss cohort provided stronger evidence, indicating modest but robust associations of long-term road and rail noise exposure with suicide mortality, independent of major confounders.

Table 5.35 Characteristics of the identified original studies for noise and suicide

First author, year	Country	Study design (follow-up)	Study population (N, age)		Outcome (Prevalence/Incidence, tool)	Noise source	Adjustment for air pollution	Confounders adjusted for in analyses	Exposure metric, increment †	Results (Risk estimate, 95% CI) ‡
Wicki, 2023	CH	Cohort (15-yr)	N = 5.1 mio	Mean 45	I Suicide mortality (national mortality records, ICD-10)	Road, rail, and aircraft, at home	PM _{2.5}	Individual level: education, civil status, nationality, mother tongue, SEP; area-level SEP, unemployment, degree of urbanization	L _{den} , 10 dB	HR, road = 1.040* (1.015–1.065) HR, railway = 1.022* (1.004–1.041) HR, aircraft = 0.997 (0.965–1.029)
Díaz, 2020	ES	Ecological time-series	N = 3.2 mio	All ages	I Number of emergency hospital admissions for depression and others	Road, city monitoring network	NO ₂ , PM ₁₀ , PM _{2.5} , O ₃	Long-term trends, seasonal patterns, and day of the week	L _{Aeq} , 1 dB	RR = 1.17* (1.05–1.30)

Abbreviation:

CH = Switzerland; ES = Spain; ICD-10 = International Classification of Diseases, 10th Revision; LAeq = Equivalent continuous sound level; L_{den} = Day-evening-night equivalent level; SEP = Socioeconomic position; CI = Confidence Interval; RR = Relative Risk; HR = Hazard Ratio.

* Statistically significant results.

† Results are presented as reported in the original studies, with varying exposure increments or comparison groups.

‡ Risk estimates are air pollution-adjusted when available; unadjusted estimates are noted where applicable.

5.3.7 *Bipolar disorder*

Previous systematic reviews did not identify any studies on transportation noise and bipolar disorder. Our updated search yielded two original investigations focusing on road traffic noise (Nobile et al., 2023; Hao et al., 2022) (Table 5.36).

In a nationwide cohort of 1.7 million adults in Italy, Nobile et al. (2023) followed 3,082 incident cases of bipolar disorder and found no evidence of an association with road traffic noise exposure (HR = 0.98, 95% CI: 0.93–1.02) after adjusting for various sociodemographic and area-level factors. By contrast, in the UK Biobank analysis, Hao et al. (2022) assessed bipolar disorder using a questionnaire using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I). Based on 334,986 adults, they reported that residential road traffic noise exposure was associated with higher odds of bipolar disorder, with risks significantly increasing by 30% to 54% across higher exposure deciles. Both results were not adjusted for air pollution.

Taken together, the current evidence on bipolar disorder and road traffic noise remains very limited and inconsistent. While one large cross-sectional study suggested elevated risks at higher exposure levels, another cohort study reported no effect.

Table 5.36 Characteristics of the identified original studies for noise and bipolar disorder

First author, year	Country	Study design (follow-up)	Study population (N, age)		Outcome (Prevalence/Incidence, tool)		Noise source	Adjustment for air pollution	Confounders adjusted for in analyses	Exposure metric, increment †	Results (Risk estimate, 95% CI) †‡
Nobile, 2023	IT	Cohort (8-yrs)	N = 1.7 mio	Mean 56	I	Hospitalization of bipolar disorder	Road, at home	-	Age, sex, marital status, place of birth, citizenship, education level, employment status, socioeconomic deprivation index, and area level variables (unemployment rate, percentage of graduates, and house prices)	L _{den} , 9.43 dB	HR = 0.98, (0.93–1.02) §
Hao, 2022	UK	Cross-sectional	N = 334,986	Mean 56	P	Bipolar disorder (SCID-I)	Road, at home	-	Age, sex, ethnicity, education, assessment centre	Reference: L _{den} < 52.1 dB	54.9–57.8 dB vs. reference: OR = 1.30*, (1.02–1.65) ≥ 57.8 dB vs. reference: OR = 1.54* (1.21–1.97)

Abbreviations:

UK = United Kingdom; IT = Italy; SCID = Structured Clinical Interview for DSM-IV Disorders; L_{den} = Day-evening-night equivalent level; CI = Confidence Interval; OR = Odds Ratio; HR = Hazard Ratio.

* Statistically significant results.

† Results are presented as reported in the original studies, with varying exposure increments or comparison groups.

‡ Risk estimates are air pollution-adjusted when available; unadjusted estimates are noted where applicable.

§ HR not adjusted for air pollution reported.

5.3.8 Other mental health outcomes

In addition to the outcomes reviewed above, our search identified two studies that investigate mental health outcomes that do not clearly fit into these categories (Table 5.37). These include several other psychiatric disorders and psychotropic drug prescriptions examined by Nobile et al. (2023), as well as acute psychiatric responses to military aircraft noise (Wicki et al., 2024).

In the nationwide cohort from Rome, Nobile et al. (2023) found no clear associations between road traffic noise and schizophrenia spectrum disorders, substance use disorders, personality and behavioural disorders, or prescriptions of antipsychotic drugs. However, small but statistically significant associations were observed for mood stabiliser prescriptions, with a 1% increase in risk per 9.43 dB increase in road traffic noise.

Another study from Switzerland applied a quasi-experimental case-time series design to assess short-term impacts of military aircraft noise among psychiatric inpatients near a military airbase (Wicki et al., 2024). Among 5,968 inpatient stays, fighter jet noise exposure was associated with increased use of rescue medication: within three hours following exposure, the probability of sedative administration increased by 1.6% (95% CI: 0.6%–2.6%) and analgesic use by 3.2% (95% CI: 1.6%–4.8%) per 10dB L_{Aeq} increase. Effects were larger among patients with multiple comorbidities.

A recent follow-up analysis published after our search refines exposure assessment using long-term noise measurements, background noise correction, and indoor attenuation (Wicki et al., 2025). This study confirmed the earlier findings by Wicki et al., (2024), again observing increased sedative use at psychiatric clinic associated with military aircraft noise events. Effect estimates remained of similar magnitude (≈ 1 –6% increase per 10 dB L_{Aeq}), with slightly higher effect estimates for certain exposure metrics, although confidence intervals were wider.

Overall, evidence for these outcomes remains scarce. However, while general population evidence is limited, the time-series study from Switzerland suggests that individuals with pre-existing psychiatric conditions may be especially vulnerable to the acute effects of noise exposure, leading to exacerbation of symptoms and adverse events, with larger effects observed among patients with multiple comorbidities.

Table 5.37 Characteristics of the identified original studies for noise and other mental health outcomes

First author, year	Country	Study design (follow-up)	Study population (N, age)		Outcome (Prevalence/Incidence, tool)	Noise source	Adjustment for air pollution	Confounders adjusted for in analyses	Exposure metric, increment †	Results (Risk estimate, 95% CI) †‡
Nobile, 2023	IT	Cohort (8-yrs)	N = 1.7 mio	Mean 56	I Incident hospitalization of schizophrenia spectrum disorders, substance use disorders, personality, and behavioural disorders, new prescriptions of antipsychotics and mood stabilizers	Road, at home	PM _{2.5} , BC, UFP	Age, sex, marital status, place of birth, citizenship, education level, employment status, socioeconomic deprivation index, and area level variables (unemployment rate, percentage of graduates, and house prices)	L _{den} , 9.43 dB	Schizophrenia spectrum disorders HR = 1.026, (0.956–1.102); antipsychotic drug HR = 1.011, (0.998–1.025); mood stabilizer drug HR 1.010*, (1.001–1.018); substance use disorder HR = 1.002, (0.929–1.081) §; personality and behaviour disorders HR = 0.994, (0.922–1.072) §
Wicki, 2024	CH	Case-time series	N = 5,968, psychiatric patients	18+	P Sedative and analgesic PRN (as-needed) drug administrations from clinic records	Military aircraft, at psychiatric clinic	-	Time-varying weather, day of week, hour of day, week of stay, long-term and seasonal trends	L _{Aeq} , hourly, 10 dB	Sedatives OR = 1.016*, (1.006, 1.026); analgesics OR = 1.032*, (1.016–1.048)

Abbreviations:

IT = Italy; CH = Switzerland; LAeq = Equivalent continuous sound level; L_{den} = Day-evening-night equivalent level; BC = Black carbon; UFP = Ultrafine particles; CI = Confidence Interval; OR = Odds Ratio; HR = Hazard Ratio.

† Results are presented as reported in the original studies, with varying exposure increments.

‡ Risk estimates are air pollution-adjusted when available; unadjusted estimates are noted where applicable.

* Statistically significant results.

§ HR not adjusted for air pollution reported.

5.3.9 Mental health problems in children and adolescents

Children's mental health outcomes and behavioural problems or neurodevelopmental disorders (e.g. ADHD) were not a primary focus of this current review, as a recent umbrella+ review and burden of disease assessment specifically addressed this topic (EEA, 2025). It covered evidence published until March 2024. Here, we summarised the main findings of that report and supplemented them with additional original studies identified in our literature search.

General mental health/mental distress in children and adolescents.

Evidence comes from two systematic reviews (Clark et al., 2020; Zare Sakhvidi et al., 2018) and three original studies (Zeng et al., 2023; Bloemsma et al., 2022b; Newbury et al., 2024a), as summarised in the previous ETC report (EEA, 2025). While one review reported no associations (Zare Sakhvidi et al., 2018), the other suggested potential adverse effects of transportation noise on mental health in children (Clark et al., 2020). Findings from the original studies are mixed: Zeng et al. (2023) reported increased internalizing and externalizing problems, whereas Bloemsma et al. (2022b) and Newbury et al. (2024a) largely reported null associations, except for anxiety. Overall, the evidence pointed to possible adverse effects of noise on mental health in children and adolescents, but results remained inconsistent.

Our updated search identified three original studies (Thompson et al., 2024; Lercher et al., 2025; Bouter et al., 2023). All outcomes were measured using self-reported scales, including the Patient Health Questionnaire (PHQ-9), Generalized Anxiety Disorder (GAD-7), Prodromal Questionnaire-16 (PQ-16), and Index of children's quality of life (KINDL). One study examined road traffic noise and two assessed mixed transportation and environmental noise. Two have cross-sectional designs, and one is a cohort study.

A large longitudinal SCAMP cohort from London (Thompson et al., 2024), followed 7,555 adolescents aged 11–15 years and found that higher road traffic noise at home and school was associated with increased anxiety symptoms ($\beta = 0.28$, $p = 0.035$ for the highest vs. lowest quartile of 24-h L_{den}), but not with depression after adjustment for air pollution. In Rotterdam, a cross-sectional analysis of 815 adolescents (Bouter et al., 2023) reported that high residential exposure to mixed environmental noise from road, rail, air, industry, and wind power was associated with more psychotic experiences in boys ($\beta = 0.23$, 95% CI: 0.01–0.46, not adjusted for air pollution), but not in girls, as assessed by the PQ-16. Finally, a cross-sectional study of 1,251 schoolchildren aged 8–12 years in Austria and Northern Italy (Lercher et al., 2025) observed a small but statistically significant inverse association between exposure to road traffic and railway noise at home and mental health problems, measured with the KINDL questionnaire ($\beta = -0.03$, 95% CI: -0.05, -0.003 per 1 dB L_{den} , after adjustment for air pollution). The authors interpreted this counterintuitive finding as likely reflecting indirect pathways through neighbourhood perceptions and coping factors.

Overall, emerging evidence suggests possible adverse effects of transportation noise on mental health in children and adolescents, though findings remain inconsistent.

Behavioural problems in children and adolescents

Evidence was synthesised from one systematic review (Schubert et al., 2019) and three European studies (Tangermann et al., 2022; Essers et al., 2022; Zijlema et al., 2021), most of which assessed behavioural problems with the Strengths and Difficulties Questionnaire (SDQ) and assigned noise exposure at home or school. The 2025 report conducted meta-analyses of the available European studies (EEA, 2025). These meta-analyses showed small but consistent associations for residential exposure: across three studies, road traffic noise was linked to higher total behavioural difficulties (RR = 1.07, 95% CI: 1.01–1.14, per 10 dB increase in L_{den}), and five studies on hyperactivity/inattention

suggested a weaker, non-significant increase (RR = 1.0, 95% CI: 0.95–1.16, per 10 dB increase in L_{den}). When both road traffic and railway noise were pooled, results were similar (RR = 1.09, 95% CI: 1.03–1.15 and RR = 1.06, 95% CI: 0.99–1.31, respectively, for total behavioural difficulty and hyperactivity/inattention). By contrast, analyses of two to three studies on road or aircraft noise at school provided pooled RRs close to unity. Overall, the evidence suggested modest associations for residential exposure, but little consistent evidence for school settings or non-road sources.

In addition to the studies summarised by the recent report (EEA, 2025), our updated search identified three further studies (Clark et al., 2021; Thompson et al., 2024; Dédélé et al., 2025). Two focused on road traffic noise and one on aircraft noise, spanning different study designs.

A cross-sectional study from Lithuania (Dédélé et al., 2025) including 1,457 preschool children, with outcomes assessed via mother-reported questionnaires, found that high residential night-time road traffic noise (≥ 50 dB L_{night}) was associated with increased odds of hyperactivity (OR = 1.79, 95% CI: 1.06–3.02) and total difficulties (OR = 1.51, 95% CI: 1.06–2.33), but not with emotional symptoms, conduct problems, or peer problems. In the longitudinal SCAMP cohort from London (Thompson et al., 2024), higher road traffic noise exposure at home and school was linked to more emotional and behavioural problems (total SDQ score) in adolescents, although these associations were not statistically significant. Similar adverse patterns were also observed for externalising and internalising problems across noise exposure quantiles. Finally, a meta-analysis of three aircraft noise studies at schools (Clark et al., 2021) reported a small but statistically significant increase in continuous hyperactivity scores in primary school children aged 8 to 10 years, with a 0.017 increase in SDQ hyperactivity score per 1 dB increase in aircraft noise, while no associations were observed with total difficulties, emotional symptoms, or conduct problems.

Taken together, the new studies provide some support for adverse associations of road traffic noise with behavioural difficulties in younger children, while findings for adolescents and for aircraft noise exposures remain inconsistent.

5.3.10 Discussion

This umbrella+ review synthesised the evidence on transportation noise and mental health outcomes in Europe, building on existing reviews and incorporating 23 additional original studies. Compared with other health outcomes such as cardiovascular disease, the evidence on mental health remains relatively limited, heterogeneous, and fragmented. Below we summarise the findings by major noise source.

Road traffic noise

Road traffic noise is by far the most extensively studied source of transportation noise in relation to mental health. Evidence from the previous systematic review and meta-analyses has suggested a small but directionally consistent association with increased risk of depression and anxiety, corresponding to 2–3% increase in risk per 10 dB L_{den} increase in noise (Hegewald et al., 2020), though these findings are not statistically significant and the overall quality of evidence is low due to the dominance of cross-sectional studies, heterogeneity in exposure and outcome assessment, and potential confounding.

With 11 original studies identified in our review covering depression, anxiety, general mental health, bipolar disorder, suicide, and other mental health outcomes, the evidence base has expanded. Large cohort studies that adjust for sociodemographic and environmental confounders frequently reported small, non-significant risk increase for depression and anxiety (Nobile et al., 2023; Eze et al., 2020; Motoc et al., 2023)

Two additional large cohort studies, published after our search period and both relying on clinically diagnosed outcomes, provide further insights into this pattern. Based on a 8-year cohort from 114,353 individuals, He et al. (2025) reported a significant 5% increase in depression risk (HR = 1.05; 95% CI 1.02–1.09) and 4% increase in anxiety risk (HR = 1.04, 95% CI: 1.01–1.07) per 10 dB increase in road traffic (L_{den}) among 114,353 Finnish children, adolescents, and young adults (baseline age 8–21 years), after adjustment for parental mental health history, socioeconomic deprivation, air pollution, and green space exposure. Lu et al. (2025) found no overall association between residential road traffic noise and depression among 18,373 middle-aged and older Swedish adults controlling for sociodemographic, lifestyle, and health-related factors, as well as air pollution, but observed weak associations in women, primarily driven by dispensed antidepressants rather than clinical diagnoses. This suggests that case definitions focusing on medication use may capture milder depressive symptoms that are more sensitive to noise exposure. Differences in case definitions also influence findings in other cohorts. In the Italian study by Nobile et al. (2023), effect sizes were larger for hospitalizations of depression than for antidepressant prescriptions, suggesting stronger associations for more severe cases. Similarly, in the Swiss SAPALDIA cohort (Eze et al., 2020), associations appeared stronger when outcomes were restricted to clinical diagnoses or antidepressant use, compared with a broader composite measure that also included mental health scores.

Indications for an association appear somewhat stronger for depression than for anxiety. Signals has also emerged for other outcomes: higher odds of bipolar disorder and symptoms of nerves, anxiety, tension, or depression in the UK Biobank cohort (Hao et al., 2022); increased risk of psychological ill-health in a longitudinal study of men in South Wales (Stansfeld et al., 2021); and small but statistically significant associations with suicide mortality in a Swiss national cohort (Wicki et al., 2023). Acute effects, such as increased same-day emergency admissions for depression, anxiety, and suicide, have been linked to daily road noise fluctuations in ecological studies (Díaz et al., 2020), although the extent to which these reflect causal effects remains unclear. In children, some evidence suggests an increased risk of hyperactivity and behavioural problems, though findings are not entirely consistent (EEA, 2025).

Although the empirical evidence for a link between transportation noise and depression as well as other mental health outcomes has not yet resolved, it is plausible from a biological point of view. Mechanistic research indicates that chronic exposure to road traffic noise may contribute to mental health problems through stress-related pathways, neuroinflammation, and circadian disruption, but direct evidence in humans is still limited (Hahad et al., 2025). Noise annoyance and sleep disturbance are important mediators, with individuals who are more noise sensitive or have poor sleep quality showing greater psychological distress (Beutel et al., 2020). Based on the findings from the Swiss SAPALDIA cohort, where mediation analysis demonstrates that the association between road traffic noise and aircraft noise exposure and depression risk was significantly mediated by noise annoyance, while no significant mediation was observed for other factors such as BMI and physical activity (Eze et al., 2020).

Railway noise

The evidence for railway noise is more limited and less consistent than for road traffic noise. Hegewald et al. (2020) reported a small and non-significant 2–3% increase in depression risk per 10 dB L_{den} . Evidence for anxiety and other mental health disorders has remained insufficient, as only a few studies have been available, and their findings are inconsistent. The strongest evidence currently comes from the Swiss cohort study (Wicki et al., 2023), which shows modest but statistically significant associations between long-term railway noise exposure and suicide mortality. In contrast, another cohort study (Eze et al., 2020) from Switzerland found no direct effect of railway noise on depression. Annoyance and sleep disturbance are recognized as important mediators between railway noise and mental health. Co-exposure to vibration is rarely quantified in epidemiological studies, but field and laboratory research has demonstrated that combined exposure to railway noise and vibration leads to greater

annoyance and sleep disturbance than noise alone. These effects appear additive, and vibration can be as disruptive as noise for sleep and annoyance (Smith et al., 2013, 2017; Seidler et al., 2023).

Of note, residential exposure to railway noise is generally less prevalent than road traffic noise, which may partly explain why fewer studies have been conducted. In the existing studies, it may also be masked by other sources, particularly road traffic noise, contributing to less consistent findings. Overall, while some evidence links railway noise to depression and suicide risk, these associations are generally weaker and less consistent than for road or aircraft noise. The field is limited by a lack of high-quality longitudinal studies, and further research is needed to clarify these relationships.

Aircraft noise

Aircraft noise has emerged as a particularly notable source of environmental noise with respect to mental health outcomes. Meta-analyses indicate that exposure to aircraft noise is associated with a significant increase in depression risk, with the systematic review by Hegewald et al. (2020) reporting a 12% rise in depression risk per 10 dB L_{den} increase, an association that appears stronger than that observed for road or railway noise. Since then, recent studies have suggested elevated risk of depression and poor mental health (Wright et al., 2018; Eze et al., 2020), although associations are not statistically significant. Evidence for other mental health outcomes remains limited and inconsistent. In children, chronic aircraft noise exposure at school is associated with increased hyperactivity and impaired reading comprehension.

Aircraft noise is also notable for causing higher levels of annoyance compared to other transportation sources at similar noise levels (Brink et al., 2019), and noise annoyance is strongly linked to both depression and anxiety in the general population (Gong et al., 2022). Large population-based studies show that individuals reporting extreme noise annoyance have nearly double the prevalence of depression and anxiety, with aircraft noise being the leading source of annoyance (Beutel et al., 2016). Longitudinal studies show that noise annoyance predicts new onset of depressive and anxiety symptoms and mediates the effect of noise exposure on mental health (Beutel et al., 2020). Noise sensitivity further amplifies these associations, particularly in vulnerable groups (Baudin et al., 2018, 2021). In fact, psychological factors such as noise annoyance and sensitivity often show stronger associations with mental health symptoms than measured noise exposure itself (Baudin et al., 2018).

Vulnerable populations, particularly those with pre-existing psychiatric conditions, high noise sensitivity, or those in psychiatric care, show increased susceptibility to noise exposure. As evident in the studies from Switzerland (Wicki et al., 2024, 2025), the observed increase in rescue medication use suggests that noise acts as an acute stressor, exacerbating psychiatric symptoms and necessitating pharmacological intervention. This aligns with mechanistic research indicating that noise exposure can include neuroinflammation, dysregulate stress hormone pathways, and disrupt circadian rhythms, all of which may contribute to acute mental health deterioration in susceptible individuals (Hahad et al., 2025; Xue et al., 2024).

Overall, while there is consistent evidence that aircraft noise contributes to annoyance and increases the risk of depression, the evidence for other mental health outcomes remains limited and sometimes inconsistent. The role of individual susceptibility, particularly noise sensitivity and pre-existing mental health conditions, appears important in mediating these effects. More longitudinal and mechanistic studies are needed to clarify causal pathways and to better understand the impact of aircraft noise on diverse mental health outcomes across different populations.

5.3.11 Conclusion

In conclusion, the current empirical evidence cannot firmly solve the question whether transportation noise affects mental health outcomes such as depression and anxiety. Nonetheless, the high biological

plausibility as well as signals for other outcomes, including suicide, bipolar disorder, and acute psychiatric exacerbations, suggest potentially important effects that merit further investigation. Further, individuals with mental health problems may be particularly vulnerable to noise exposure in their daily life. Road traffic noise remains the priority source given its ubiquity and the relatively stronger evidence base, while aircraft and rail noise require substantially more research, particularly using longitudinal cohorts with robust exposure and outcome assessment. Future work should aim to harmonise exposure metrics, incorporate personal and perceived noise measures, disentangle noise from correlated environmental exposures such as air pollution, and explore mechanistic pathways through sleep disruption, stress biology, and neuroinflammation.

Box 1 Nature-based solutions and interventions

In a recent review published by WHO Europe it was emphasized that there is growing recognition of the potential of holistic approaches, which jointly address both human health and a healthy natural environment (WHO, 2025). In this report nature-based solutions are defined as actions that protect, restore, and sustainably manage ecosystems to tackle issues such as climate change, pollution, and biodiversity loss, which - when well-designed, have “co-benefits” for health and social equity. Examples include river restoration to improve water quality, urban greening to reduce air pollution and heat, and coastal wetlands that protect communities from floods. Such nature-based solutions can also promote mental health, physical activity, and social cohesion through access to green and blue spaces. However, poorly planned interventions can have unintended effects, such as increasing disease vectors or driving “green gentrification.”

Nature-based solutions have relevance to multiple levels of prevention including primordial prevention (protecting the fundamentals of health and well-being for entire populations), primary, secondary and tertiary preventions. In terms of tertiary prevention, various nature-based therapeutic approaches or interventions have been developed in the recent decades (Coventry et al., 2021). The general principle of such interventions are structured activities and environmental changes designed to engage individuals with natural settings for mental health benefits. This includes:

- **Gardening and Horticultural Therapy:** Involvement in gardening activities, often in community or therapeutic settings, has shown significant improvements in mood, reduction in depression and anxiety, and increased well-being (Menhas et al., 2024; Vujcic et al., 2017).
- **Green Exercise:** Physical activities like walking, running, or group exercise in parks, forests, or other green spaces are linked to reduced depressive symptoms and anxiety, and improved positive affect (Bloomfield, 2017).
- **Forest-Based Interventions (Forest Bathing):** Immersive experiences in forest environments, such as “forest bathing,” have demonstrated large, significant effects on anxiety, depression, and overall mental health (Kang et al., 2022).
- **Nature-Based Social Prescribing:** Programs where healthcare providers prescribe nature-based activities (e.g., community gardening, park visits, group walks) as complementary or alternative therapy to traditional mental health treatments (Lavelle Sachs et al., 2024).
- **Blue Space Interventions:** Activities in or near water bodies (e.g., wetlands, rivers) such as wetland walks or outdoor swimming, which can reduce stress and improve emotional well-being (Coventry et al., 2021).
- **Nature-Based Meditation and Mindfulness:** Mindful walking, meditation, and guided imagery in natural settings can decrease rumination and depressive symptoms, especially in young people (Owens and Bunce, 2022).
- **Therapeutic Environmental Changes:** Creating or improving access to green spaces in urban environments, such as hospital gardens or urban parks, to facilitate passive or active engagement with nature (Nejade et al., 2022).

Nature-based interventions are often found to be effective for a range of mental health outcomes, including reducing depression, anxiety, stress, and loneliness, and improving positive affect and social connectedness (Gawrych and Romaniuk, 2025). Reviews conclude that most effective interventions are typically structured, last 8–12 weeks, and involve 20–90 minutes of nature contact per session (Coventry et al., 2021). It seems that both, individual and group-based NBIs are beneficial, with group activities often enhancing social support and belonging (Lavelle Sachs et al., 2024). Although many of nature-based intervention studies report beneficial effects on a variety of mental health outcomes, it should be considered that such trials are inherently not placebo controlled and not double blind and most rely on self-reported outcomes, which makes them vulnerable to bias. Further, if and to what extent beneficial effects persist in the long run, has been rarely addressed.

6 Conclusions

In conclusion, this review found plausible links between exposure to ambient air pollutants, chemicals and noise and various mental, behavioural and neurodevelopmental disorders, although empirical data are often scarce for specific exposure-outcomes pairs. In urban settings many environmental exposures are increased, but also socioeconomic factors and lifestyle may differ from non-urban areas, which poses a challenge for deriving causality for specific environmental exposures. Future research should focus on the causal pathways using innovative research methods to elucidate which environmental conditions are most detrimental for mental health. A better understanding of the combined effects of various exposures is also desirable. Such information will help to design effective interventions to reduce mental health burden from environmental exposure. Nevertheless, even if there are knowledge gaps, improving environmental conditions will be of benefit for the population's mental health status. Well-designed nature-based solutions to tackle climate change, pollution, and biodiversity loss have “co-benefits” for health and social equity if well-designed. Thus, our environment should not only be seen as a risk factor for mental health but also as a resource to improve mental health in the population.

List of abbreviations

Abbreviation	Name	Reference
95% CI	95% confidence interval	
ADHD	Attention deficit/hyperactivity disorder	
AMSTAR	A MeaSurement Tool to Assess systematic Reviews	
DALY	Disability-adjusted life years	
GRADE	Grading of Recommendations Assessment, Development and Evaluation	
EEA	European Environment Agency	www.eea.europa.eu
EFSA	European Food Safety Authority	https://www.efsa.europa.eu/en/efsajournal/pub/9398
dB(A)	A-weighted decibels	
HiAP	health in all policies approach	
IHME	Institute For Health Metrics and Evaluation	https://www.healthdata.org/
L _{Aeq}	Equivalent continuous sound level	
L _{Aeq,24h}	L _{Aeq} averaged over 24 hours	
L _{den}	L _{Aeq} averaged over 8 hours (during nighttime)	
L _{day} , L _{Aeq,16h}	L _{Aeq} averaged over 16 hours (during daytime)	
L _{night} , L _{Aeq,8h}	Time-weighted L _{Aeq,24h} with a penalty of 5 dB(A) for evening-time and 10 dB(A) for night-time	
NDVI	Normalized Difference Vegetation Index.	
NO _x	Nitrogen oxides	
OR	Odds ratio	
PM	Particulate matter	
PM _{2.5}	Particulate matter with a diameter of 2.5 µm or less	
PM ₁₀	Particulate matter with a diameter of 10 µm or less	
PECOS	Population, Exposures, Comparators, Outcomes and Study design	
RoB	Risk of Bias	
RR	Relative risk	
SDQ	Strength and Difficulties Questionnaire	
SO _x	Sulphur oxides	

WHO	World Health Organization	https://www.who.int/
WHO ENG	Environmental Noise Guidelines from the World Health Organization	https://www.who.int/europe/ publications/i/item/9789289053563

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Annex 1 Mental health outcomes search terms in PubMed and PsycINFO

A. 1 Mental health outcomes search terms in PubMed

Mental health condition and search term entries	PubMed search
Mental health	"Mental Health"[MeSH Terms] OR "Mental Disorders"[MeSH Terms] OR "mental health"[Title/Abstract] OR "mental disorder*"[Title/Abstract] OR "mental illness"[Title/Abstract] OR "mental ill health" [Title/Abstract] OR "psychological disorder*"[Title/Abstract] OR "psychiatric disorder*"[Title/Abstract]
Anxiety disorders	"Anxiety Disorders"[MeSH Terms] OR "anxiety"[Title/Abstract] OR "anxiety disorder*"[Title/Abstract] OR "anxiety symptom*"[Title/Abstract] OR "anxiety state*"[Title/Abstract] OR "generalized anxiety disorder*"[Title/Abstract] OR "generalised anxiety disorder*"[Title/Abstract] OR "generalized anxiet*"[Title/Abstract] OR "generalised anxiet*"[Title/Abstract] OR "general anxiety disorder*"[Title/Abstract] OR "overanxious disorder*"[Title/Abstract] OR "GAD"[Title/Abstract] OR "obsessive-compulsive"[Title/Abstract] OR "obsessive compulsive disorder*"[Title/Abstract] OR "OCD"[Title/Abstract] OR "panic disorder*"[Title/Abstract] OR "panic attack*"[Title/Abstract] OR "phobic disorder*"[Title/Abstract] OR "phobia*"[Title/Abstract]
Mood disorders	"Mood Disorders"[MeSH Terms] OR "Depression"[MeSH Terms] OR "mood disorder*"[Title/Abstract] OR "affective disorder*"[Title/Abstract] OR "bipolar disorder*"[Title/Abstract] OR "bipolar"[Title/Abstract] OR "unipolar disorder*"[Title/Abstract] OR "unipolar depression"[Title/Abstract] OR "Depression"[Title/Abstract] OR "depress*"[Title/Abstract] OR "depressive disorder*"[Title/Abstract] OR "depressive episode*"[Title/Abstract] OR "depressive symptom*"[Title/Abstract] OR "manic episode*"[Title/Abstract] OR "manic disorder*"[Title/Abstract] OR "hypomania"[Title/Abstract]
Substance-related disorders	"Substance-Related Disorders"[MeSH Terms] OR "substance related disorder*"[Title/Abstract] OR "substance related disorder*"[Title/Abstract] OR "substance use disorder*"[Title/Abstract] OR "drug use disorder*"[Title/Abstract] OR "substance abuse*"[Title/Abstract] OR "substance addiction"[Title/Abstract] OR "substance dependence"[Title/Abstract] OR "drug abuse*"[Title/Abstract] OR "drug addiction"[Title/Abstract] OR "drug dependence"[Title/Abstract] OR "alcoholism"[Title/Abstract] OR "alcohol abuse"[Title/Abstract] OR "alcohol addiction"[Title/Abstract] OR "alcohol dependence"[Title/Abstract]
Personality disorders	"Personality Disorders"[MeSH Terms] OR "personality disorder*"[Title/Abstract]
Schizophrenia	"Schizophrenia"[MeSH Terms] OR "Psychotic Disorders"[MeSH Terms] OR "schizophreni*"[Title/Abstract] OR "schizotypal"[Title/Abstract] OR "schizoaffective"[Title/Abstract] OR "psychotic disorder*"[Title/Abstract] OR "psychosis"[Title/Abstract] OR "psychotic*"[Title/Abstract]

Psychological sexual dysfunctions	"sexual dysfunctions, psychological"[MeSH Terms] OR "psychological sexual dysfunction*"[Title/Abstract] OR "psychosexual disorder*"[Title/Abstract] OR "psychosexual dysfunction*"[Title/Abstract]
Feeding and eating disorders	"Feeding and Eating Disorders"[MeSH Terms] OR ("eating"[Title/Abstract] AND "feeding disorder*"[Title/Abstract]) OR "appetite disorder*"[Title/Abstract] OR "eating disorder*"[Title/Abstract] OR "anorexia"[Title/Abstract] OR "anorexia nervosa"[Title/Abstract] OR "binge eating disorder*"[Title/Abstract] OR "bulimia"[Title/Abstract] OR "bulimia nervosa"[Title/Abstract]
Trauma and Stressor Related Disorders	"Trauma and Stressor Related Disorders"[MeSH Terms] OR ("trauma"[Title/Abstract] AND "stress related disorder*"[Title/Abstract]) OR ("trauma"[Title/Abstract] AND "stressor related disorder*"[Title/Abstract]) OR "post traumatic stress disorder*"[Title/Abstract] OR "post traumatic stress disorder*"[Title/Abstract] OR "posttraumatic stress disorder*"[Title/Abstract] OR "adjustment disorder*"[Title/Abstract] OR "acute stress disorder*"[Title/Abstract] OR "acute stress reaction"[Title/Abstract] OR "stressor related disorder*"[Title/Abstract] OR "stress related disorder*"[Title/Abstract] OR ("stressor-related"[All Fields] AND "illness*"[Title/Abstract]) OR "stress related illness*"[Title/Abstract] OR "PTSD"[Title/Abstract]
Suicidality and self-injurious behaviour	"Suicide"[MeSH Terms] OR "Self-Injurious Behavior"[MeSH Terms] OR "suicid*"[Title/Abstract] OR "Self-Injurious Behavior"[Title/Abstract] OR "self injurious behaviour"[Title/Abstract] OR "self harm*"[Title/Abstract] OR "parasuicide*"[Title/Abstract]
Hospital admissions related to mental health conditions mentioned above	"psychiatric admission*"[Title/Abstract] OR "psychiatric diagnos*"[Title/Abstract] OR "psychiatric hospitalization"[Title/Abstract]

A. 2 Mental health outcomes search terms in PsycINFO

exp "Mental Health"/ or exp "Mental Disorders"/ or "mental health".ti,ab. or "mental disorder*".ti,ab. or "mental illness".ti,ab. or "mental ill health".ti,ab. or "psychological disorder*".ti,ab. or "psychiatric disorder*".ti,ab. or ("anxiety" or "anxiety disorder*" or "anxiety symptom*" or "anxiety state*" or "generalized anxiety disorder*" or "generalised anxiety disorder*" or "generalized anxiet*" or "generalised anxiet*" or "general anxiety disorder*" or "overanxious disorder*" or "GAD" or "obsessive compulsive" or "obsessive-compulsive disorder*" or "OCD" or "panic disorder*" or "panic attack*" or "phobic disorder*" or "phobia*").ti,ab. or (exp "Depression (Emotion)"/ or "mood disorder*".ti,ab. or "affective disorder*".ti,ab. or "bipolar disorder*".ti,ab. or "bipolar".ti,ab. or "unipolar disorder*".ti,ab. or "unipolar depression".ti,ab. or "depression".ti,ab. or "depress*".ti,ab. or "depressive disorder*".ti,ab. or "depressive episode*".ti,ab. or "depressive symptom*".ti,ab. or "manic episode*".ti,ab. or "manic disorder*".ti,ab. or "hypomania".ti,ab.) or ("substance related disorder*" or "substance-related disorder*" or "substance use disorder*" or "drug use disorder*" or "substance abuse*" or "substance addiction" or "substance dependence" or "drug abuse*" or "drug addiction" or "drug dependence" or "alcoholism" or "alcohol abuse" or "alcohol addiction" or "alcohol dependence").ti,ab. or "personality disorder*".ti,ab. or ("schizophreni*" or "schizotypal" or "schizoaffective" or "psychotic disorder*" or "psychosis" or "psychotic*").ti,ab. or (exp "Psychosexual Behavior"/ or "psychological sexual dysfunction*".ti,ab. or "psychosexual disorder*".ti,ab. or "psychosexual dysfunction*".ti,ab.) or (("eating" adj3 "feeding disorder*") or "appetite disorder*" or "eating disorder*" or "anorexia" or "anorexia nervosa" or "binge-eating disorder*" or "bulimia" or "bulimia nervosa").ti,ab. or (("trauma" adj3 "stress related disorder*") or ("trauma" adj3 "stressor related disorder*") or "post-traumatic stress disorder*" or "post traumatic stress disorder*" or "posttraumatic stress disorder*" or "adjustment disorder*" or "acute stress disorder*" or "acute stress reaction" or "stressor-related disorder*" or "stress-related disorder*" or "stressor-related illness*" or "stress-related illness*" or "PTSD").ti,ab. or ("suicid*" or "self injurious behavior" or "self injurious behaviour" or "self-harm*" or "parasuicide*").ti,ab. or (exp "Psychiatric Hospital Admission"/ or exp "Psychiatric Hospitalization"/ or "psychiatric admission*".ti,ab. or "psychiatric diagnos*".ti,ab. or "psychiatric hospitalization".ti,ab.)

Annex 2 Search strategy in LUDOK database and LUDOK backlog for air pollution and mental health outcomes

The strategy to check the LUDOK resources was two pronged. First the LUDOK database LINK was searched with translated keywords from the PubMed search since the LUDOK entries are written in German (Table A.2. 1). Additionally, the original abstract of studies was searched with English keywords. The results were then restricted to review studies using the LUDOK code 5U or for the European study analysis to the Code 2R for studies conducted in Europe.

Table A.2. 1 LUDOK search terms

Topic	LUDOK Search term
Mental Health	Title: Mental Health OR abstract: "mental health"
	Methods: Mentale Gesundheit
	Methods: Psychisch
Mood / Depression	Methods: Depression Abstract: "mood disorder" OR "affective disorder" OR unipolar OR manic
Anxiety disorders	Methods: Angst Abstract: phobi
Bipolar	Methods: Bipolar OR abstract: bipolar
Substance-related disorders	Methods: Drogen (Methods: Missbrauch)
Schizophrenia	Methods: Schizophren OR Psychos (Methods: psychot)
Personality disorders	Methods: Persönlichkeits OR abstract: Personality
Psychological sexual dysfunctions	Abstract: Sexual dysfuntion
Feeding and Eating Disorders	Methods: Essstörung Abstract: Eating disorder OR bulimia
Trauma and Stressor Related Disorders	Abstract: PTSD OR traumatic stress OR "stress disorder"
Suicidality and self-injurious behaviour	Methods: Suizid OR Selbsttötung Abstract: "self harm" OR "self injur"
Hospital admissions related to mental health conditions mentioned above	Included in general search for mental health

Second, the backlog of LUDOK (an EndNote file) was searched for studies that have been selected but not yet extracted and summarized in the LUDOK database (see figure below).

Title

Contains

anxiety

+

x

Or

Abstract

Contains

anxiety

+

x

And

Added to Library

Is greater than

01.05.2024

+

x

And

Rating

Is greater than or equal to

★ ★ . . .

+

x

Clear search

Simple search

Search options

Search

Mental health condition and search term entries	PubMed search	Search terms Endnote

Mental health	"Mental Health"[MeSH Terms] OR "Mental Disorders"[MeSH Terms] OR "mental health"[Title/Abstract] OR "mental disorder*"[Title/Abstract] OR "mental illness"[Title/Abstract] OR "mental ill health"[Title/Abstract] OR "psychological disorder*"[Title/Abstract] OR "psychiatric disorder*"[Title/Abstract]	Mental health (52) OR mental ill (2) OR psychiatric (4)
Anxiety disorders	"Anxiety Disorders"[MeSH Terms] OR "anxiety"[Title/Abstract] OR "anxiety disorder*"[Title/Abstract] OR "anxiety symptom*"[Title/Abstract] OR "anxiety state*"[Title/Abstract] OR "generalized anxiety disorder*"[Title/Abstract] OR "generalised anxiety disorder*"[Title/Abstract] OR "generalized anxiet*"[Title/Abstract] OR "generalised anxiet*"[Title/Abstract] OR "general anxiety disorder*"[Title/Abstract] OR "overanxious disorder*"[Title/Abstract] OR "GAD"[Title/Abstract] OR "obsessive-compulsive"[Title/Abstract] OR "obsessive compulsive disorder*"[Title/Abstract] OR "OCD"[Title/Abstract] OR "panic disorder*"[Title/Abstract] OR "panic attack*"[Title/Abstract] OR "phobic disorder*"[Title/Abstract] OR "phobia*"[Title/Abstract]	Title/Abstract: anxiety OR anxious OR obsessive OR panic OR phobia OR affective OR mood OR phobic (0)
		Abstract: anxiety OR anxious OR obsessive disorder OR panic OR phobic OR phobia OR mood OR affective
Mood disorders	"Mood Disorders"[MeSH Terms] OR "Depression"[MeSH Terms] OR "mood disorder*"[Title/Abstract] OR "affective disorder*"[Title/Abstract] OR "bipolar disorder*"[Title/Abstract] OR "bipolar"[Title/Abstract] OR "unipolar disorder*"[Title/Abstract] OR "unipolar depression"[Title/Abstract] OR "Depression"[Title/Abstract] OR "depress*"[Title/Abstract] OR "depressive disorder*"[Title/Abstract] OR "depressive episode*"[Title/Abstract] OR "depressive symptom*"[Title/Abstract] OR "manic episode*"[Title/Abstract] OR "manic disorder*"[Title/Abstract] OR "hypomania"[Title/Abstract]	Title/Abstract: depress OR manic OR unipolar
		Title/Abstract: bipolar
Substance-related disorders	"Substance-Related Disorders"[MeSH Terms] OR "substance related disorder*"[Title/Abstract] OR "substance related disorder*"[Title/Abstract] OR "substance use disorder*"[Title/Abstract] OR "drug use disorder*"[Title/Abstract] OR "substance abuse*"[Title/Abstract] OR "substance addiction"[Title/Abstract] OR "substance dependence"[Title/Abstract] OR "drug abuse*"[Title/Abstract] OR "drug addiction"[Title/Abstract] OR "drug dependence"[Title/Abstract] OR "alcoholism"[Title/Abstract] OR "alcohol abuse"[Title/Abstract] OR "alcohol addiction"[Title/Abstract] OR "alcohol dependence"[Title/Abstract]	Title: drug OR substance related OR substance-related OR substance abuse OR substance dependence OR addiction

Personality disorders	"Personality Disorders"[MeSH Terms] OR "personality disorder*"[Title/Abstract]	Title/abstract: personality
Schizophrenia	"Schizophrenia"[MeSH Terms] OR "Psychotic Disorders"[MeSH Terms] OR "schizophreni*"[Title/Abstract] OR "schizotypal"[Title/Abstract] OR "schizoaffective"[Title/Abstract] OR "psychotic disorder*"[Title/Abstract] OR "psychosis"[Title/Abstract] OR "psychotic*"[Title/Abstract]	Title/Abstract: schizophren OR psychotic
Psychological sexual dysfunctions	"sexual dysfunctions, psychological"[MeSH Terms] OR "psychological sexual dysfunction*"[Title/Abstract] OR "psychosexual disorder*"[Title/Abstract] OR "psychosexual dysfunction*"[Title/Abstract]	Title: sexual
		(tested dysfunction – too much hits regarding hormonal/metabolics)
Feeding and eating disorders	"Feeding and Eating Disorders"[MeSH Terms] OR ("eating"[Title/Abstract] AND "feeding disorder*"[Title/Abstract]) OR "appetite disorder*"[Title/Abstract] OR "eating disorder*"[Title/Abstract] OR "anorexia"[Title/Abstract] OR "anorexia nervosa"[Title/Abstract] OR "binge eating disorder*"[Title/Abstract] OR "bulimia"[Title/Abstract] OR "bulimia nervosa"[Title/Abstract]	Title/Abstract: Eating disorder OR feeding OR appetite OR anorexia (2) OR binge (0) OR bulimia (0)
		Notes: eating à heating / treating = no relevant hits, only 1 with eating disorder
Trauma and Stressor Related Disorders	"Trauma and Stressor Related Disorders"[MeSH Terms] OR ("trauma"[Title/Abstract] AND "stress related disorder*"[Title/Abstract]) OR ("trauma"[Title/Abstract] AND "stressor related disorder*"[Title/Abstract]) OR "post traumatic stress disorder*"[Title/Abstract] OR "post traumatic stress disorder*"[Title/Abstract] OR "posttraumatic stress disorder*"[Title/Abstract] OR "adjustment disorder*"[Title/Abstract] OR "acute stress disorder*"[Title/Abstract] OR "acute stress reaction"[Title/Abstract] OR "stressor related disorder*"[Title/Abstract] OR "stress related disorder*"[Title/Abstract] OR ("stressor-related"[All Fields] AND "illness*"[Title/Abstract]) OR "stress related illness*"[Title/Abstract] OR "PTSD"[Title/Abstract]	Title/Abstract: PTSD (2) OR traumatic stress (2) OR stress related OR stress-related OR stress disorder
Suicidality and self-injurious behaviour	"Suicide"[MeSH Terms] OR "Self-Injurious Behavior"[MeSH Terms] OR "suicid*"[Title/Abstract] OR "Self-Injurious Behavior"[Title/Abstract] OR "self injurious behaviour"[Title/Abstract] OR "self harm*"[Title/Abstract] OR "parasuicide*"[Title/Abstract]	Title/Abstract: suicide OR self harm OR self injur OR self-injur
Hospital admissions related to mental health conditions mentioned above	"psychiatric admission*"[Title/Abstract] OR "psychiatric diagnos*"[Title/Abstract] OR "psychiatric hospitalization"[Title/Abstract]	included with general search for mental health see above

List of excluded studies from the LUDOK search regarding European studies

The LUDOK search on European non-review studies resulted in 155 hits. Thereof, 86 were excluded. Reasons for exclusion included topics like sick building syndrome, spills / accidents, neurodegenerative and cognitive outcomes, cardio-respiratory endpoints studying for example ST-segment “depression” myocardial infarction, occupational, indoor or tobacco smoke exposures.

In the full-text stage the following 10 studies were excluded with reasons:

(Newbury et al., 2022) – identical data without gene analysis in Newbury 2019
(Newbury et al., 2024b) (Association of early-life exposure to air and noise pollution with youth mental health: findings from the ALSPAC cohort.) – no full text available, similar with potentially less long follow-up as (Newbury et al., 2024a) (Air and Noise Pollution Exposure in Early Life and Mental Health From Adolescence to Young Adulthood.)
(Gładka et al., 2022) – geographical correlation analysis
(Khan et al., 2019) – geographical correlation analysis
(Hautekiet et al., 2020) – methods paper without useful results
(Feng et al., 2023) – analysis of diabetes patients - not general population (UK biobank)
(Payne et al., 1994) – no objective pollution measurement
(Petkus et al., 2019) – study in the USA, not Europe (wrong code in database)
(Sheffield et al., 2018) – study in the USA, not Europe (wrong code in database)
(Strahilevitz et al., 1979) – correlation analysis with no confounder control

From the reference lists of reviews one additional European study was identified (Oudin et al., 2016)

List of excluded studies from the screening stage for all exposures (without reasons)

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Ayres, J. G., Semple, S., MacCalman, L., Dempsey, S., Hilton, S., Hurley, J. F., Miller, B. G., Naji, A., & Petticrew, M. (2009). Bar workers’ health and environmental tobacco smoke exposure (BHETSE): symptomatic improvement in bar staff following smoke-free legislation in Scotland. *Occup Environ Med*, 66, 339-346. <https://doi.org/10.1136/oem.2008.040311>

Barath, S., Mills, N. L., Adelroth, E., Olin, A. C., & Blomberg, A. (2013). Diesel exhaust but not ozone increases fraction of exhaled nitric oxide in a randomized controlled experimental exposure study of healthy human subjects. *Environ Health*, 12, 36. <https://doi.org/10.1186/1476-069X-12-36>

Barregard, L., Saellsten, G., Gustafson, P., Andersson, L., Johansson, L., Basu, S., & Stigendal, L. (2006). Experimental exposure to wood-smoke particles in healthy humans: effects on markers of inflammation, coagulation, and lipid peroxidation. *Inhal Toxicol*, 18(11), 845-853. <https://www.ncbi.nlm.nih.gov/pubmed/16864402>
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In the full-text stage the following 9 studies were excluded

(Newbury et al., 2022)– identical data without gene analysis in Newbury 2019
 (Gładka et al., 2022) – geographical correlation analysis
 (Khan et al., 2019) – geographical correlation analysis
 (Hautekiet et al., 2020) – methods paper without useful results
 (Feng et al., 2023) – analysis of diabetes patients - not general population (UK biobank)
 (Payne et al., 1994) – no objective pollution measurement
 (Petkus et al., 2019) – study in the USA, not Europe (wrong code in database)
 (Sheffield et al., 2018) – study in the USA, not Europe (wrong code in database)
 (Strahilevitz et al., 1979) – correlation analysis with no confounder control

Annex 3 Search strategy for environmental chemicals and mental health outcomes in PubMed, Scopus, and PsycINFO

Mental health condition and search term entries	Search terms
Mental health	"Mental Health"[MeSH Terms] OR "Mental Disorders"[MeSH Terms] OR "mental health"[Title/Abstract] OR "mental disorder*"[Title/Abstract] OR "mental illness"[Title/Abstract] OR "mental ill health" [Title/Abstract] OR "psychological disorder*"[Title/Abstract] OR "psychiatric disorder*"[Title/Abstract]
Anxiety disorders	"Anxiety Disorders"[MeSH Terms] OR "anxiety"[Title/Abstract] OR "anxiety disorder*"[Title/Abstract] OR "anxiety symptom*"[Title/Abstract] OR "anxiety state*"[Title/Abstract] OR "generalized anxiety disorder*"[Title/Abstract] OR "generalised anxiety disorder*"[Title/Abstract] OR "generalized anxiet*"[Title/Abstract] OR "generalised anxiet*"[Title/Abstract] OR "general anxiety disorder*"[Title/Abstract] OR "overanxious disorder*"[Title/Abstract] OR "GAD"[Title/Abstract] OR "obsessive-compulsive"[Title/Abstract] OR "obsessive compulsive disorder*"[Title/Abstract] OR "OCD"[Title/Abstract] OR "panic disorder*"[Title/Abstract] OR "panic attack*"[Title/Abstract] OR "phobic disorder*"[Title/Abstract] OR "phobia*"[Title/Abstract]
Mood disorders	"Mood Disorders"[MeSH Terms] OR "Depression"[MeSH Terms] OR "mood disorder*"[Title/Abstract] OR "affective disorder*"[Title/Abstract] OR "bipolar disorder*"[Title/Abstract] OR "bipolar"[Title/Abstract] OR "unipolar disorder*"[Title/Abstract] OR "unipolar depression"[Title/Abstract] OR "Depression"[Title/Abstract] OR "depress*"[Title/Abstract] OR "depressive disorder*"[Title/Abstract] OR "depressive episode*"[Title/Abstract] OR "depressive symptom*"[Title/Abstract] OR "manic episode*"[Title/Abstract] OR "manic disorder*"[Title/Abstract] OR "hypomania"[Title/Abstract]
Substance-related disorders	"Substance-Related Disorders"[MeSH Terms] OR "substance related disorder*"[Title/Abstract] OR "substance related disorder*"[Title/Abstract] OR "substance use disorder*"[Title/Abstract] OR "drug use disorder*"[Title/Abstract] OR "substance abuse*"[Title/Abstract] OR "substance addiction"[Title/Abstract] OR "substance dependence"[Title/Abstract] OR "drug abuse*"[Title/Abstract] OR "drug addiction"[Title/Abstract] OR "drug dependence"[Title/Abstract] OR "alcoholism"[Title/Abstract] OR "alcohol abuse"[Title/Abstract] OR "alcohol addiction"[Title/Abstract] OR "alcohol dependence"[Title/Abstract]
Personality disorders	"Personality Disorders"[MeSH Terms] OR "personality disorder*"[Title/Abstract]
Schizophrenia	"Schizophrenia"[MeSH Terms] OR "Psychotic Disorders"[MeSH Terms] OR "schizophreni*"[Title/Abstract] OR "schizotypal"[Title/Abstract] OR "schizoaffective"[Title/Abstract] OR "psychotic"

	disorder*[Title/Abstract] OR "psychosis"[Title/Abstract] OR "psychotic"[Title/Abstract]
Psychological sexual dysfunctions	"sexual dysfunctions, psychological"[MeSH Terms] OR "psychological sexual dysfunction*[Title/Abstract] OR "psychosexual disorder*[Title/Abstract] OR "psychosexual dysfunction*[Title/Abstract]
Feeding and eating disorders	"Feeding and Eating Disorders"[MeSH Terms] OR ("eating"[Title/Abstract] AND "feeding disorder*[Title/Abstract]) OR "appetite disorder*[Title/Abstract] OR "eating disorder*[Title/Abstract] OR "anorexia"[Title/Abstract] OR "anorexia nervosa"[Title/Abstract] OR "binge eating disorder*[Title/Abstract] OR "bulimia"[Title/Abstract] OR "bulimia nervosa"[Title/Abstract]
Trauma and Stressor Related Disorders	"Trauma and Stressor Related Disorders"[MeSH Terms] OR ("trauma"[Title/Abstract] AND "stress related disorder*[Title/Abstract]) OR ("trauma"[Title/Abstract] AND "stressor related disorder*[Title/Abstract]) OR "post traumatic stress disorder*[Title/Abstract] OR "post traumatic stress disorder*[Title/Abstract] OR "posttraumatic stress disorder*[Title/Abstract] OR "adjustment disorder*[Title/Abstract] OR "acute stress disorder*[Title/Abstract] OR "acute stress reaction"[Title/Abstract] OR "stressor related disorder*[Title/Abstract] OR "stress related disorder*[Title/Abstract] OR ("stressor-related"[All Fields] AND "illness*[Title/Abstract]) OR "stress related illness*[Title/Abstract] OR "PTSD"[Title/Abstract]
Suicidality and self-injurious behaviour	"Suicide"[MeSH Terms] OR "Self-Injurious Behavior"[MeSH Terms] OR "suicid*[Title/Abstract] OR "Self-Injurious Behavior"[Title/Abstract] OR "self injurious behaviour"[Title/Abstract] OR "self harm*[Title/Abstract] OR "parasuicide*[Title/Abstract]
Hospital admissions related to mental health conditions mentioned above	"psychiatric admission*[Title/Abstract] OR "psychiatric diagnos*[Title/Abstract] OR "psychiatric hospitalization"[Title/Abstract]
Chemicals and search term entries	
Heavy metals	"heavy metal*[Title/Abstract] OR "cadmium"[Title/Abstract] OR "mercury"[Title/Abstract] OR "quicksilver"[Title/Abstract] OR "arsenic"[Title/Abstract] OR "manganese"[Title/Abstract]
Environmental tobacco smoke	"environmental tobacco smoke"[Title/Abstract] OR "tobacco smoke pollution*[Title/Abstract] OR "secondhand smok*[Title/Abstract] OR "second hand smok*[Title/Abstract] OR "shs"[Title/Abstract] OR "passive smok*[Title/Abstract] OR "passive tobacco smok*[Title/Abstract] OR "involuntary smok*[Title/Abstract]
Endocrine disruptors	"endocrine disrupting chemical*[Title/Abstract] OR "endocrine disrupting chemical*[Title/Abstract] OR "endocrine disruptor*[Title/Abstract] OR "phthalat*[Title/Abstract] OR

	"phthalic"[Title/Abstract] OR "polybrominated diphenyl ether*"[Title/Abstract] OR "bisphenol*"[Title/Abstract] OR "per and polyfluoroalkyl substance*"[Title/Abstract] OR "polyfluoroalkyl*"[Title/Abstract] OR "perfluoroalkyl*"[Title/Abstract] OR "polychlorinated biphenyl*"[Title/Abstract] OR "PFAS"[Title/Abstract] OR "PFOA"[Title/Abstract]
Volatile organic compound	"volatile organic compound*"[Title/Abstract]
Pesticides	"pesticide*"[Title/Abstract] OR "organophosphat*"[Title/Abstract]
MeSH Terms	"Metals, Heavy/adverse effects"[Mesh] OR "Lead"[Mesh] OR "Environmental Pollutants/chemistry"[Mesh] OR "Hazardous Substances/chemistry"[Mesh] OR "Pesticide Residues"[Mesh] OR "Drug Residues"[Mesh]

Search string in PsycINFO for chemicals

#	Query Chemicals
1	exp "Mental Health"/ or exp "Mental Disorders"/ or "mental health".ti,ab. or "mental disorder*".ti,ab. or "mental illness".ti,ab. or "mental ill health".ti,ab. or "psychological disorder*".ti,ab. or "psychiatric disorder*".ti,ab. or ("anxiety" or "anxiety disorder*" or "anxiety symptom*" or "anxiety state*" or "generalized anxiety disorder*" or "generalised anxiety disorder*" or "generalized anxiet*" or "generalised anxiet*" or "general anxiety disorder*" or "overanxious disorder*" or "GAD" or "obsessive compulsive" or "obsessive-compulsive disorder*" or "OCD" or "panic disorder*" or "panic attack*" or "phobic disorder*" or phobia*).ti,ab. or (exp "Depression (Emotion)"/ or "mood disorder*".ti,ab. or "affective disorder*".ti,ab. or "bipolar disorder*".ti,ab. or "bipolar".ti,ab. or "unipolar disorder*".ti,ab. or "unipolar depression".ti,ab. or "depression".ti,ab. or "depress*".ti,ab. or "depressive disorder*".ti,ab. or "depressive episode*".ti,ab. or "depressive symptom*".ti,ab. or "manic episode*".ti,ab. or "manic disorder*".ti,ab. or "hypomania".ti,ab.) or ("substance related disorder*" or "substance-related disorder*" or "substance use disorder*" or "drug use disorder*" or "substance abuse*" or "substance addiction" or "substance dependence" or "drug abuse*" or "drug addiction" or "drug dependence" or "alcoholism" or "alcohol abuse" or "alcohol addiction" or "alcohol dependence").ti,ab. or "personality disorder*".ti,ab. or ("schizophreni*" or "schizotypal" or "schizoaffective" or "psychotic disorder*" or "psychosis" or "psychotic*).ti,ab. or (exp "Psychosexual Behavior"/ or "psychological sexual dysfunction*".ti,ab. or "psychosexual disorder*".ti,ab. or "psychosexual dysfunction*".ti,ab.) or (("eating" adj3 "feeding disorder*") or "appetite disorder*" or "eating disorder*" or "anorexia" or "anorexia nervosa" or "binge-eating disorder*" or "bulimia" or "bulimia nervosa").ti,ab. or (("trauma" adj3 "stress related disorder*") or ("trauma" adj3 "stressor related disorder*") or "post-traumatic stress disorder*" or "post traumatic stress disorder*" or "posttraumatic stress disorder*" or "adjustment disorder*" or "acute stress disorder*" or "acute stress reaction" or "stressor-related disorder*" or "stress-related disorder*" or "stressor-related illness*" or "stress-related illness*" or "PTSD").ti,ab. or ("suicid*" or "self injurious behavior" or "self injurious behaviour" or "self-harm*" or "parasuicide*).ti,ab. or (exp "Psychiatric Hospital Admission"/ or exp "Psychiatric Hospitalization"/ or "psychiatric admission*".ti,ab. or "psychiatric diagnos*.ti,ab. or "psychiatric hospitalization".ti,ab.) AND "heavy metal*".ti,ab. or "cadmium".ti,ab. or "mercury".ti,ab. "quicksilver".ti,ab. or "arsenic".ti,ab. or "manganese".ti,ab. or "environmental tobacco smoke".ti,ab. or "tobacco smoke pollution*".ti,ab. or "secondhand smok*".ti,ab. or or

"second hand smok*".ti,ab. or "second-hand smok*".ti,ab. or "shs".ti,ab. or "passive smok*".ti,ab. or "passive tobacco smok*".ti,ab. or "involuntary smok*".ti,ab. or "endocrine-disrupting chemical*".ti,ab. or "endocrine disrupting chemical*".ti,ab. or "endocrine disruptor*".ti,ab. or "phthalat*".ti,ab. or "phthalic".ti,ab. or "polybrominated diphenyl ether*".ti,ab. or "bisphenol*".ti,ab. or "per- and polyfluoroalkyl substance*".ti,ab. or "polyfluoroalkyl*".ti,ab. or "perfluoroalkyl*".ti,ab. or "polychlorinated biphenyl*".ti,ab. or "volatile organic compound*".ti,ab. or "pesticide*".ti,ab. or "organophosphat*".ti,ab. or "PFAS".ti,ab. or "PFOA".ti,ab. or exp "Lead (Metal)"/ or exp „Lead Poisoning“/ or exp “chemical exposure”/ or exp “metals”/ or exp “metallic elements”/ or exp “pesticides”/ or exp “insecticides”/ or exp “toxins”/
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In the full-text stage on environmental chemicals 52 articles were excluded. Exclusion criteria primarily included wrong outcome, wrong population, occupational exposure, wrong exposure, and wrong study design. List of excluded articles in the full-text screening with reasons:

Aaseth et al., 2018 – occupational exposure
Allaouat et al., 2020 – occupational exposure
Arab & Mostafalou, 2022 – occupational exposure
Avram et al., 2019 – wrong outcome
Bakoyiannis et al., 2021 – wrong population
Barrett & Padula, 2019 – wrong outcome
Berghuis & Roze, 2019 – wrong outcome
Błazewicz & Grabrucker, 2023 – wrong outcome
Bochynska et al., 2024 – wrong outcome
Brandt et al., 2022 – wrong exposure
Brown et al., 2024 – occupational exposure
Calderòn-Garciduenas et al., 2020 – wrong exposure
Cassereau et al., 2017 – occupational exposure, wrong population
Cheng et al., 2024 – wrong outcome
Comanescu & Racovita, 2024 – wrong exposure, wrong outcome
Cornelius et al., 2021 – occupational exposure, wrong outcome, wrong population
Costa, 2017 – occupational exposure, wrong outcome
Dinocourt et al., 2015 – occupational exposure, wrong population
Escudero et al., 2021 – wrong population, wrong outcome

Frengidou et al., 2024 – occupational exposure, wrong population
 Garza-Lombó et al., 2019 – wrong outcome
 Goldberg & Gould, 2019 – wrong exposure
 Hector et al., 2018 – wrong exposure
 Hertz-Picciotto et al., 2018 – wrong outcome
 Hogan & Freeman, 2016 – wrong outcome
 Jung et al., 2015 – wrong study design
 Lanphear, 2015 – wrong outcome
 Lini et al., 2024 – occupational exposure
 Malone Rubright et al., 2017 – wrong outcome
 Maqbool et al., 2016 – wrong population
 Marshall et al., 2019 – occupational exposure, wrong population
 Młyniec et al., 2017 – wrong outcome
 Okoli & Kodet, 2015 – wrong outcome
 Park & Lee, 2021 – wrong exposure
 Patwa & Flora, 2020 – wrong outcome
 Peres et al., 2016 – wrong outcome
 Pessah et al., 2019 – occupational exposure, wrong outcome
 Polemiti et al., 2024 – wrong exposure
 Rauh & Margolis, 2016 – wrong outcome
 Reddy et al., 2022 – wrong exposure
 Reed & Claunch, 2020 – occupational exposure
 Rock & Patisaul, 2018 – wrong outcome
 Rueda-Ruzafa et al., 2019 – wrong population
 Sánchez et al., 2024 – wrong outcome
 Sinyor et al., 2017 – wrong outcome
 Soyer-Gobillard et al., 2022 – wrong exposure
 Soyer-Gobillard et al., 2025 – wrong exposure
 Underner et al., 2023 – not in english language
 Vellingiri, 2023 – wrong outcome
 Wadhwa et al., 2018 – wrong exposure
 Yesayan et al., 2024 – wrong outcome
 Zwolak, 2020 – wrong outcome

In the data-extraction stage on environmental chemicals the following 5 articles were excluded with reasons:

Arfianti Wiraagni et al., 2020 – wrong population
 Dickerson et al., 2020 – background article, wrong population
 Hernández-Coro et al., 2021 – background article
 Hyun & Ka, 2024 – wrong outcome
 Manley et al., 2018 – background article

Annex 4 Search strategy for noise and mental health outcomes in PubMed and PsycINFO

#	PubMed query	Results
1	((((((((((((((((((((("Anxiety Disorders"[MeSH Terms]) OR (anxiety[Title/Abstract])) OR (anxiety disorder*[Title/Abstract])) OR (anxiety symptom*[Title/Abstract])) OR (anxiety state*[Title/Abstract])) OR (generalized anxiety disorder*[Title/Abstract])) OR (generalised anxiety disorder*[Title/Abstract])) OR (generalized anxiet*[Title/Abstract])) OR (generalised anxiet*[Title/Abstract])) OR (general anxiety disorder*[Title/Abstract])) OR (overanxious disorder*[Title/Abstract])) OR (GAD[Title/Abstract])) OR (obsessive compulsive[Title/Abstract])) OR (obsessive-compulsive disorder*[Title/Abstract])) OR (OCD[Title/Abstract])) OR (panic disorder*[Title/Abstract])) OR (panic attack*[Title/Abstract])) OR (phobic disorder*[Title/Abstract])) OR (phobia*[Title/Abstract])) OR ("Mental Health"[MeSH Terms] OR "Mental Disorders"[MeSH Terms] OR "mental health"[Title/Abstract] OR "mental disorder*[Title/Abstract] OR "mental illness"[Title/Abstract] OR "mental ill health"[Title/Abstract] OR "psychological disorder*[Title/Abstract] OR "psychiatric disorder*[Title/Abstract] OR (((((((((((((((("Mood Disorders"[MeSH Terms]) OR ("Depression"[MeSH Terms])) OR (mood disorder*[Title/Abstract])) OR (affective disorder*[Title/Abstract])) OR (bipolar disorder*[Title/Abstract])) OR (bipolar[Title/Abstract])) OR (unipolar disorder*[Title/Abstract])) OR (unipolar depression[Title/Abstract])) OR (depression[Title/Abstract])) OR (depress*[Title/Abstract])) OR (depressive disorder*[Title/Abstract])) OR (depressive episode*[Title/Abstract])) OR (depressive symptom*[Title/Abstract])) OR (manic episode*[Title/Abstract])) OR (manic disorder*[Title/Abstract])) OR (hypomania[Title/Abstract])) OR (((((((((((((((("Substance-Related Disorders"[MeSH Terms]) OR (substance related disorder*[Title/Abstract])) OR (substance-related disorder*[Title/Abstract])) OR (substance use disorder*[Title/Abstract])) OR (drug use disorder*[Title/Abstract])) OR (substance abuse*[Title/Abstract])) OR (substance addiction[Title/Abstract])) OR (substance dependence[Title/Abstract])) OR (drug abuse*[Title/Abstract])) OR (drug addiction[Title/Abstract])) OR (drug dependence[Title/Abstract])) OR (alcoholism[Title/Abstract])) OR (alcohol abuse[Title/Abstract])) OR (alcohol addiction[Title/Abstract])) OR (alcohol dependence[Title/Abstract])) OR (("Personality Disorders"[MeSH Terms]) OR (personality disorder*[Title/Abstract])) OR (((((((((((("Schizophrenia"[MeSH Terms]) OR ("Psychotic Disorders"[MeSH Terms])) OR (schizophreni*[Title/Abstract])) OR (schizotypal[Title/Abstract])) OR (schizoaffective[Title/Abstract])) OR (psychotic disorder*[Title/Abstract])) OR (psychosis[Title/Abstract])) OR (psychotic*[Title/Abstract])) OR (((("Sexual Dysfunctions, Psychological"[MeSH Terms]) OR (psychological sexual dysfunction*[Title/Abstract])) OR (psychosexual disorder*[Title/Abstract])) OR (psychosexual dysfunction*[Title/Abstract])) OR (((((((((((("Feeding and Eating Disorders"[MeSH Terms]) OR (eating[Title/Abstract] AND feeding disorder*[Title/Abstract])) OR (appetite disorder*[Title/Abstract])) OR (eating disorder*[Title/Abstract])) OR (anorexia[Title/Abstract])) OR (anorexia nervosa[Title/Abstract])) OR (binge-eating disorder*[Title/Abstract])) OR (bulimia[Title/Abstract])) OR (bulimia nervosa[Title/Abstract])) OR (((((((((((("Trauma and Stressor Related Disorders"[MeSH Terms]) OR (trauma[Title/Abstract] AND stress related disorder*[Title/Abstract])) OR (trauma[Title/Abstract] AND stressor related disorder*[Title/Abstract])) OR (post-traumatic stress disorder*[Title/Abstract])) OR (post traumatic stress disorder*[Title/Abstract])) OR (posttraumatic stress disorder*[Title/Abstract])) OR (adjustment disorder*[Title/Abstract])) OR (acute stress disorder*[Title/Abstract])) OR (acute stress reaction[Title/Abstract])) OR (stressor-related disorder*[Title/Abstract])) OR (stress-related disorder*[Title/Abstract])) OR (stressor-related illness*[Title/Abstract])) OR (stress-related illness*[Title/Abstract])) OR (PTSD[Title/Abstract])) OR (((((((("Suicide"[MeSH Terms]) OR ("Self-Injurious Behavior"[MeSH Terms])) OR (suicid*[Title/Abstract])) OR (self injurious behavior[Title/Abstract])) OR (self injurious behaviour[Title/Abstract])) OR (self-harm*[Title/Abstract])) OR	2,451,493

	(parasuicide*[Title/Abstract])) OR (((psychiatric admission*[Title/Abstract]) OR (psychiatric diagnos*[Title/Abstract])) OR (psychiatric hospitalization[Title/Abstract]))	
2	("Noise, Transportation" [MeSH] OR (noise [tiab] AND traffic[tiab]) OR (noise [tiab] AND transportation [tiab]) OR (noise [tiab] AND road [tiab]) OR (noise [tiab] AND (airplane [tiab] OR aircraft [tiab])) OR (noise [tiab] AND rail* [tiab]) OR (noise[tiab] AND environmental[tiab]) OR (noise[tiab] AND community[tiab]))	13,019
3	2015/10/01:2025/04/04[dp]	13,103,979
4	#1 AND #2 AND #3	665
5	#1 AND #2 AND #3	Filter: Humans 511

#	PsycINFO query	Results from 8 Apr 2025
1	exp "Mental Health"/ or exp "Mental Disorders"/ or "mental health".ti,ab. or "mental disorder".ti,ab. or "mental illness".ti,ab. or "mental ill health".ti,ab. or "psychological disorder".ti,ab. or "psychiatric disorder".ti,ab. or ("anxiety" or "anxiety disorder*" or "anxiety symptom*" or "anxiety state*" or "generalized anxiety disorder*" or "generalised anxiety disorder*" or "generalized anxiet*" or "generalised anxiet*" or "general anxiety disorder*" or "overanxious disorder*" or "GAD" or "obsessive compulsive" or "obsessive-compulsive disorder*" or "OCD" or "panic disorder*" or "panic attack*" or "phobic disorder*" or phobia*).ti,ab. or (exp "Depression (Emotion)"/ or "mood disorder".ti,ab. or "affective disorder".ti,ab. or "bipolar disorder".ti,ab. or "bipolar".ti,ab. or "unipolar disorder".ti,ab. or "unipolar depression".ti,ab. or "depression".ti,ab. or "depress*".ti,ab. or "depressive disorder".ti,ab. or "depressive episode".ti,ab. or "depressive symptom".ti,ab. or "manic episode".ti,ab. or "manic disorder".ti,ab. or "hypomania".ti,ab.) or ("substance related disorder*" or "substance-related disorder*" or "substance use disorder*" or "drug use disorder*" or "substance abuse*" or "substance addiction" or "substance dependence" or "drug abuse*" or "drug addiction" or "drug dependence" or "alcoholism" or "alcohol abuse" or "alcohol addiction" or "alcohol dependence").ti,ab. or "personality disorder".ti,ab. or ("schizophreni*" or "schizotypal" or "schizoaffective" or "psychotic disorder*" or "psychosis" or "psychotic").ti,ab. or (exp "Psychosexual Behavior"/ or "psychological sexual dysfunction".ti,ab. or "psychosexual disorder".ti,ab. or "psychosexual dysfunction".ti,ab.) or (("eating" adj3 "feeding disorder*") or "appetite disorder*" or "eating disorder*" or "anorexia" or "anorexia nervosa" or "binge-eating disorder*" or "bulimia" or "bulimia nervosa").ti,ab. or (("trauma" adj3 "stress related disorder*") or ("trauma" adj3 "stressor related disorder*") or "post-traumatic stress disorder*" or "post traumatic stress disorder*" or "posttraumatic stress disorder*" or "adjustment disorder*" or "acute stress disorder*" or "acute stress reaction" or "stressor-related disorder*" or "stress-related disorder*" or "stressor-related illness*" or "stress-related illness*" or "PTSD").ti,ab. or ("suicid*" or "self injurious behavior" or "self injurious behaviour" or "self-harm*" or "parasuicide*").ti,ab. or (exp "Psychiatric Hospital Admission"/ or exp "Psychiatric Hospitalization"/ or "psychiatric admission".ti,ab. or "psychiatric diagnos".ti,ab. or "psychiatric hospitalization".ti,ab.)	1,774,161
2	(exp Transportation/ or (rail* or aircraft or airport* or airplane or road* or traffic or transportation or automobile* or vehicle* or environmental or community).ti,ab.) and (noise*.ti,ab. or "Noise Levels (Work Areas)"/ or exp Noise Effects/)	2,940
3	1 and 2	523
4	limit 3 to human	451
5	limit 4 to yr="2015-2025"	211

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