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Enhancing the Recognition of Mental Health Risks Related to Environmental Hazards

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ABSTRACT **Commentary**

Background: Atmospheric pollution is widely recognised as a determinant of respiratory and cardiovascular morbidity; however, its contribution to psychiatric burden remains underappreciated in both clinical and regulatory contexts. This asymmetry in recognition risks may underestimate the mental health toll of environmental hazard exposure.

Purpose: This commentary synthesises the available evidence linking ambient air pollutant exposure primarily particulate matter ($PM_{2.5}$, PM_{10}) and nitrogen oxides to psychiatric outcomes including depression, anxiety, psychological stress, and suicidal behaviour, and examines the implications for clinical practice and environmental policy.

Evidence summary: Reviewed studies spanning longitudinal cohorts, quasi-experimental designs, systematic reviews, and predictive modelling consistently demonstrate associations between air pollution and psychiatric symptomatology across diverse populations. A non-linear dose-response pattern is observed, with mental health risks emerging at pollutant concentrations within or below established regulatory thresholds. Stress response to inhaled pollutants with neuroendocrine activation including hypothalamus-pituitary-adrenal axis and sympathetic-adrenal-medullary stimulation offer a plausible mechanistic basis. Decontamination interventions have shown prospective benefits on depressive burden.

Conclusions: The mental health burden associated with ambient air pollution could be underestimated by current diagnostic frameworks and may not be adequately prevented by existing air quality standards. Clinicians should consider environmental exposures in the differential assessment of psychiatric symptoms, and policymakers are urged to revise regulatory thresholds to reflect the non-linear risk profiles identified for vulnerable populations.

Keywords: Air Pollution, Particulate Matter, Mental Health, Depression, Anxiety, Neuroendocrine Response, Public Health Policy.

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Introduction

When atmospheric toxicity reaches hazardous levels, the link to respiratory alterations tends to be evident, contrary to the effect on psychiatric disorders that can be manifested in these circumstances, which are not always considered as being related to environmental conditions. This approach could reflect an underestimation of the influence these factors may have in the identification of mental health burden, which could lead to a limited diagnosis and treatment of psychiatric disorders. In fact, the relevance of mental health complications related to particulate matter (PM) exposure has been comparable to the impact verified in physical health diseases.

Previous studies found that PM_{10} contamination was related to intensified depressive symptomatology by 17% (95% CI, 4.9-30.5%) for each 24.2 μg/m³ three-day collective increment (Lim et al., 2012). In turn, the morbimortality associated with PM_{10} pollution, according to a review from the American Heart Association (Brook et al., 2010), revealed an increase in cardiovascular diseases requiring hospital admission by 0.8-2.6% each day, at any 20 μg/m³ rise in pollutant levels. It is believed that if the association between psychosocial and environmental factors is unraveled, the diagnosis accuracy could be improved, as well as the therapeutic alternatives, which might cover a spectrum beyond the pharmacological treatment to include also air purification measures, along with concomitant strategies that would minimize the combined detrimental effect on health outcomes.

The Psychosomatic Overlap: Physical Symptoms as Psychiatric Indicators

Turner et al. (2022) carried out a study with healthy and clinical (asthma) adolescents, verifying that short-term inhalation of ultrafine particles (UFP), mostly indoors, was positively related to physical symptomatology of stress without gender differences. These authors have argued that these symptoms could play a role in the development of anxiety and depression, whereas also assuming that physical stress might be a somatic symptom irrespective of the levels identified in the self-reported measurements for psychiatric symptomatology. This was verified by the lack of significant effects, at this time-frame, on anxiety, depression or psychological stress perceptions (Turner et al., 2022).

Even though not often acknowledged by the individuals themselves, the simultaneous manifestation of physical and psychiatric symptoms is common (Kroenke et al., 1997) and could be underestimated by disclosure concerns regarding mental health illnesses (Clement et al., 2015; Conner et al., 2010). Kroenke et al. (1997) identified self-perceived stress as an independent predictor of psychiatric disorders, being patients with anxiety or depression more prone to express physical complaints. In this study, almost 30% of the patients reporting physical symptoms were subsequently diagnosed with depression and/or anxiety suggesting an underlying psychiatric genesis. There were also cases where these diseases could not be clinically recognized due to a borderline level (e.g., minor depression or anxiety), in spite of lasting for long periods of time (Kroenke et al., 1997). Several studies corroborate this assumption assuming anxiety and depression disorders as a predisposition for psychosomatization (Creed et al., 2012; Janssens et al., 2010).

Environmental Determinants and Vulnerable Populations

The risks of ambient pollution for mental health motivated the need to assess this relationship in individuals exposed to high levels of harmful hazards, including dwellers from areas with increased aerosol contamination due to combustion, traffic pollution, wildfires, among other adverse conditions (Chen et al., 2008; To et al., 2021). Previous studies have demonstrated that standard levels for air quality do not always prevent psychiatric burden (Casas et al., 2017; Chen et al., 2008; Lyons et al., 2024; Yang et al., 2023), suggesting that there are groups at a higher risk of suffering unwanted outcomes, as well as conditions that may favour this nefarious impact. Therefore, it is considered that the repercussions of inhaled pollutants on mental health should be addressed accounting for the influence of psychosocial aspects, behavioural choices and environmental hazards, being one of the theoretical explanations referring to the allostatic load (McEwen, 1998).

Pre-clinical research (Henriquez et al., 2018; Miller et al., 2016;Snow et al., 2018)has elucidated mechanisms underlying physiological responses to the inhalation of atmospheric pollutants, including the neuroendocrine activation, which is still scarcely represented in interventional studies with human participants (Li et al., 2017). Table 1 intends to review and summarize scientific literature demonstrating the relationship between psychiatric symptomatology and ambient toxicity, mainly focused on stress, depression, anxiety and suicidal behaviour. Among the several chemical harmful substances, particulate matter and its components were primordially analysed, along with the combined effect of the most toxic pollutants. As less severe symptoms could be of considerable relevance when analysing mental health burden related to ambient pollution, studies using emergency room visits or medical consultations as main outcomes were not included. In line with the explanation provided by Gao et al. (2023), there might be an underestimation of psychiatric risks, as these outcomes only cover the cases when psychological suffering motivates the use of mental health services.

Evidence Synthesis: Decontamination, Dose–Response, and Neuroendocrine Pathways

Several areas of knowledge may be unraveled by the body of evidence presented in Table 1. As a starting point, the effectiveness of atmospheric decontamination measures demonstrated by quasi-experimental studies, predictive analysis and longitudinal research (Braithwaite et al., 2019; Chen et al., 2023; Wang et al., 2014; Xue et al., 2021) have revealed the potential of targeting not only psychiatric symptomatology, but also the mitigation of health-risks associated with mental illnesses (Xue et al., 2021).

These strategies, however, have identified vulnerable groups to decontamination intervention ineffectiveness, such as the oldest populations (≥ 75 years old; Chen et al., 2023). Considering this age-group, in individuals with prior cardiorespiratory diseases and with no such clinical pre-conditions, air pollution was found to be a deterrent of the beneficial effects from physical activity in cardiovascular diseases burden (Sinharay et al., 2018). Distinct health risks werealso observed among diverse pollutant(s) concentrations/contamination conditions (Gao et al., 2023; Mehta et al., 2015; Yang et al., 2023) whereas length of exposure was a determinant factor for the most harmful atmospheric hazards (Borroni et al., 2022; Park et al., 2024).Mental health burden related to air pollution showed a non-linear relationship with increasing atmospheric toxicity, demonstrating an increased risk for depression and anxiety at levels that do not tend to be harmful, as highlighted in the findings of Gao et al. (2023) and Yang et al. (2023), and further supported by other studies (Chen et al., 2008; Lyons et al., 2024; Park et al., 2024).

While the latter assumption still applies, some research pointed out the combined impact of inhaled hazards to explain related mental health burden with distinct weights for each pollutant (e.g., Borroni et al., 2022; Gao et al., 2023; Mehta et al., 2015; Min et al., 2024; Wu et al., 2024). Emphasizing gaps in knowledge in need of further elucidation, Gao et al. (2023) verified mental health risks associated with ambient pollution at baseline and within a follow-up of seven years (Gao et al., 2023). Neuroendocrine activation in human stress response related to atmospheric pollutants involving hypothalamus-pituitary-adrenal and sympathetic-adrenal-medullary has been mostly corroborated by Li et al. (2017), although also strengthened by Zundel et al.'s systematic review (Zundel et al., 2022), with the findings of Turner et al. (2022) embedded in the accumulated evidence in this field suggesting that physical symptoms of stress related to atmospheric contamination might be psychosomatic manifestations of anxiety/depression or factors contributing to the development of these clinical entities.

Policy and Clinical Implications

Based on the argument sustained, the observed impact of air pollution, along with the predictive benefits from decontamination policies on stress, depression, anxiety and suicidal symptomatology sets the ground for the design of interventional studies aiming to provide further evidence on the conjectured hypotheses. Furthermore, the highlighted research areas impel the analysis of current environmental law and regulations on air pollution, as the standards defined for healthy levels of atmospheric pollutants might be challenged by a non-linear pollutant(s)-exposure relationship in certain groups and medical conditions at risk for harmful effects derived from atmospheric contamination.

Limitations

Several limitations qualify the argument sustained in this commentary. First, this article relies on a narrative (rather than systematic) literature review; studies were selected to illustrate the breadth of evidence rather than through a pre-registered, exhaustive search strategy, which introduces the possibility of selection bias and limits the reproducibility of the evidence synthesis. Second, the body of literature summarised in Table 1 is predominantly derived from high-income countries (United States, United Kingdom, China, South Korea, Canada), restricting the generalisability of findings to populations in low- and middle-income settings where ambient pollution levels frequently exceed those examined and access to psychiatric services is more constrained. Third, the methods applied and heterogeneity in study design, which spans randomised crossover trial, prospective cohort studies, quasi-experimental analyses, and predictive modelling, precludes direct comparison of effect sizes across studies, and meta-analytic pooling was not performed. Fourth, the predominantly self-reported nature of psychiatric outcome measurement across reviewed studies introduces response and recall biases, while underreporting driven by mental health stigma may further attenuate observed associations (Clement et al., 2015; Conner et al., 2010). Fifth, residual confounding by socioeconomic status, housing quality, green space access, and psychosocial stressors cannot be excluded in observational studies, even when adjusted. Finally, the evidence sustaining physiological stress response to air pollutants involving neuroendocrine activation remains largely pre-clinical, with limited confirmatory interventional data in human participants (Li et al., 2017), warranting cautious interpretation of proposed pathways.

Table 1. Evidence supporting psychiatric burden related to air pollutants toxicity

STUDY ID	STUDY DESIGN/ TYPE OF ANALYSIS	COUNTRY/ REGION	POPULATION	LENGTH OF EXPOSURE	ENVIRONMENTAL HAZARD(S)	POLLUTANTS CONCENTRATION LEVELS (µg/m³)[a]/ INTERVENTION	OUTCOMES AND/OR PREDICTIONS
Chen et al. (2023)	Predictions for 2100 based on deep learning tools	Global	< 75 yo	Until 2100	$PM_{2.5}$	Reaching 15.7 in 2100	• PM-related mortality would decline by 16.3% in 60 years.
			≥ 75 yo			Estimations between 15.7-34.5 for 2100	• Higher PM-related mortality by 2090 irrespective of the healthiest air contamination prediction.

Braithwaite et al. (2019)	Predictions based on a systematic review with meta-analysis	Global	Estimations for the world's population		$PM_{2.5}$	25 µg/m³ standard compared to 44 µg/m³ in previous estimations	• Decline of 15.2% in depression risk (95% CI, 3.85-25.9%).
		United Kingdom	Estimations for UK urban areas			10 µg/m³ standard compared to 13 µg/m³ in 2014 in the UK	• Reduction of depression incidences by 2.5% (95% CI, 0.58-4.34%).
Xue et al. (2021)	Quasi-Experimental	China	Retired citizens with a mean age of 61 yo	4 years	$PM_{2.5}$	Prior 2011 vs. at the end of Clean Air Policy intervention (2015), per each decontamination of 10 µg/m³	• Depressive symptoms decreased by 4.14% (95% CI, 0.41-8.00%); • Retardation of expecting aging-related repercussion on depression by 0.25% (95% CI, -0.78-1.30%) between 2011-2015.
Braithwaite et al. (2019)	Systematic review with meta-analysis	Mostly Asia and North America	People aged 18 yo or over	Short- and long-term	$PM_{2.5}$ PM_{10}	At each $PM_{2.5}$ 5 (Pun et al., 2017) and 10µg/m³ increase (Power et al., 2015)	• Anxiety – one-year exposure: OR: 1.15 (95% CI, 1.06-1.25, *p*=.001; Power et al., 2015); OR: 1.39 (95% CI, 1.15-1.69; Pun et al., 2017); • Anxiety – four-year exposure: OR: 1.34 (95% CI, 1.12-1.61; Pun et al., 2017); • Anxiety – fifteen-year exposure: OR: 1.09 (95% CI, 1.01-1.18, *p*=.03; Power et al., 2017).
						Per $PM_{2.5}$ 10µg/m³ increment, long-term (> 6 months) exposure	• Depression risk (MA): OR: 1.10 (95% CI, 1.02-1.19, *p*=.011); • For PM_{10}, the effects on depression did not reach significant levels.
						Per PM_{10} 10µg/m³ increment, at 2 days exposure	• Suicide health risk (MA): 1.02 (95% CI, 1.00-1.03, *p*=.031)
Borroni et al. (2022)	Systematic review with meta-analysis	Data not available	Data not available	Data not available	$PM_{2.5}$ PM_{10} NO_2 SO_2 O_3 CO	Long-term (≥ 30 days) and short-term (< 30 days) exposure to atmospheric hazards	• Relative Risks for depression of long-term exposure to air pollutants: $PM_{2.5}$ (RR: 1.07, 95% CI, 1.02-1.13); NO_2 (RR: 1.04, 95% CI, 1.01-1.06); • Short-term effects: PM_{10} (RR: 1.01, 95% CI, 1.01-1.01); $PM_{2.5}$ (RR: 1.01, 95% CI, 1.01-1.01); NO_2 (RR: 1.02, 95% CI, 1.01-1.03); SO_2 (RR: 1.02, 95% CI, 1.01-1.04); O_3 (RR: 1.01, 95% CI, 1.00-1.03); CO (RR: 1.06, 95% CI, 1.02-1.11).
Mehta et al. (2015)	Longitudinal	United States of America	Male older Veterans (mean age of 69 yo)	12 years of follow up with 4-week assessment of exposure to ambient pollutants	$PM_{2.5}$ Black Carbon NO_2 Particle Number Counts (PNC) O_3 Sulfate	Daily means: $PM_{2.5}$ (11) Black Carbon (1) NO_2 (0.02ppm) PNC (24 counts/cm³)	• $PM_{2.5}$, Black Carbon, NO_2 and PNC showed statistically significant associations with psychological stress perceptions within a time-frame of up to 4 weeks, while sulfate and ozone did not reveal significant associations; • PNC was the most relevant hazard related to subjective stress scores with 3.2 point deterioration in perceived stress ratings (95% CI, 2.1-4.3), per each 15.997 counts/cm³ increment in pollutant atmospheric toxicity within the first week.
Yang et al. (2023)	Prospective, longitudinal study	United Kingdom	National cohort from UK Biobank with a mean age of 57 yo and no previous depression or anxiety disorders	11 years of follow up	Combined effect $PM_{2.5}$ $PM_{2.5-10}$ NO_2 NO,	Lowest levels: $PM_{2.5}$ (~8-10) NO_2 (~13-28) NO, (~0-20) Highest levels: $PM_{2.5}$ (~10-13) NO_2 (~28-48) NO, (~20-48)	• Aggravated depression and anxiety risks within the lowest concentrations range with steady effects at incremented levels of ambient pollutants, including the combined effect, $PM_{2.5}$, NO_2, and NO,, but no $PM_{2.5-10}$ (no change at ~6-9µg/m³ exposure).
						Highest ambient toxicity index ($PM_{2.5}$, NO_2, and NO,) vs. healthiest pollutants atmospheric concentrations score	• Health risk for depression of 1.16 (95% CI, 1.09-1.23, *p*<.001) and for anxiety of 1.11 (95% CI, 1.05-1.17, *p*<.001).
Gao et al. (2023)	Prospective, longitudinal study	United Kingdom	National cohort from UK Biobank (excluding participants from Northern England and Scotland in PM analysis) with a mean age of 57 yo and no previous depression or anxiety disorders	7 years of follow up	Combined effect $PM_{2.5}$ $PM_{2.5-10}$ PM_{10} NO_2 NO_x	$PM_{2.5}$ $PM_{2.5-10}$ PM_{10} NO_2 NO_x	• Ambient pollution at baseline was associated with depression burden at this time-point by 8% for $PM_{2.5}$ and NO_2, as well as by 6% and 5% for NO_x and PM_{10}, respectively (all *p*<.0001); • Higher risk for anxiety incidences at baseline was also verified in association with environmental pollution at the same time-point assessment, increasing by 7% for $PM_{2.5}$, by 4% for NO_x, by 3% for NO_2 (all *p*<.0001), and by 2% for PM_{10} (*p*=.010); • Neither depression, nor anxiety incidences at baseline were significantly affected by PM coarse contamination; • The odds of depression incidences at follow up, per each rise in pollutants levels at baseline, incremented by 1% (95% CI, 1.00-1.04, *p*=.047) for $PM_{2.5}$, by 5% for both PM_{10} (95% CI, 1.02-1.07, *p*=.0009) and NO_2 (95% CI, 1.02-1.08, *p*=.0002), and by 3% for NO_x (95% CI, 1.00-1.05, *p*=.031); • Regarding anxiety, environmental hazards increased toxicity at baseline was associated with aggravated health risks by 3% for $PM_{2.5}$ (95% CI, 1.00-1.06, *p*=.031), by 7% for PM_{10} and NO_2 (both 95% CI, 1.04-1.10, *p*<.0001), and by 4% for NO_x (95% CI, 1.01-1.06, *p*=.0047); • No significant effects on mental health related to PM coarse in either

							psychiatric outcomes; • In this selected sample of the UK population, a positive relationship was observed between ambient contamination index and psychiatric disorders for all the pollutants, except PM coarse, which increased at lower levels and decreased within the most toxic range (~7-18); • Sensitive analyses for prolonged exposure, excluding the influence of PM coarse on mental disorders, revealed significant associations for both NO_2 and NO_x pollutants, whereas $PM_{2.5}$ did not reach significant levels and PM_{10} had a reduced impact.
						Highest ambient toxicity index ($PM_{2.5}$, PM_{10}, NO_2, and NO,) vs. healthiest pollutants atmospheric concentrations score	• Baseline measurements showed similar or higher chances of psychiatric burden than that at follow up with incremented odds of depression and anxiety incidences by 24% and 14%, respectively (both $p<.0001$); • Increased odds, at follow up, for depression with an hazard ratio of 1.10 (95% CI, 1.03-1.17, $p=.0067$) and of 1.15 (95% CI, 1.06-1.24, $p=.0009$) for anxiety.
Lyons et al. (2024)	Cross-sectional analysis from a prospective, longitudinal study	Republic of Ireland	National cohort aged 50 yo or older with a mean age of 66 yo [54-95]	Data from the third wave of the longitudinal study were analysed (2014-2015) matched with long-term $PM_{2.5}$ measurements (1998-2014)	$PM_{2.5}$	Highest levels did not exceed 12	• Pollutant concentrations above 7 µg/m³ were considered a health risk for depression and anxiety; • Regarding anxiety, PM effects on mental health were clinically significant with an odds ratio of 1.22 (95% CI, 1.09-1.36, $p=.0008$), however, binary analysis did not show a clinically relevant risk for depressive symptoms; • There were no statistically significant effects on perceived stress, worry, and quality of life; • From 1998 to 2014, air pollution considerably improved.
Chen et al. (2008)	Cross-sectional	Canada	Clinical sample of adolescents (9-18 yo) diagnosed with asthma, with no other comorbidities	6 months followup	Long-term exposure to NO_2 contamination from traffic	Mean exposure: 17ppb Groups divided by median NO_2 exposure: • Mean for high-pollution group: 19ppb • Mean for low-pollution group: 14ppb	• Perceived chronic stress related to traffic pollution was associated with interleukin-5 (ß=-0.31, $p=.02$), Immunoglobulin E (ß=-0.29, $p=.02$), and eosinophil counts (ß=-0.24, $p=.04$); • When the levels of inhaled ambient hazards toxicity declines, aggravated stress was related to the increment of inflammatory biomarkers; • In a long-term exposure to atmospheric pollution, intensified chronic stress was associated with lower peak expiratory flow rate (interaction ß=0.30, $p=.03$), as pollutant concentrations declined.
Wu et al. (2024)	Prospective, longitudinal study	China	National cohort aged 45yo or above	5 years of follow up	$PM_{2.5}$ constituents: Sulfate; Nitrate; Ammonium; Organic elements; Black carbon.	Data not available	• From all constituents, depressive symptoms related to $PM_{2.5}$ atmospheric toxicity were affected by nitrate, sulfate, and black carbon core components.
Min et al. (2024)	Prospective, longitudinal study	China	National cohort aged 45yo or above	7 years of follow up	$PM_{2.5}$ constituents: Sulfate; Nitrate; Ammonium; Organic elements; Black carbon.	$PM_{2.5}$ (57-39); Sulfate (11-7); Nitrate (12-9); Ammonium (8-6); Organic elements (14-10); Black carbon (3-2).	• Higher odds of depressive symptomatology were associated with $PM_{2.5}$ increment by 2.6% (95% CI, 1.3-4.0%). In terms of PM components, the aggravated health risk increased by 2.2% for both nitrate (95% CI, 0.6-3.9%) and organic elements (95% CI, 1.0-3.4%), and by 2.0% for ammonium (95% CI, 0.6-3.4%), which were the main contributors for psychiatric burden.
Wang et al. (2014)	Prospective, longitudinal study	United States of America	Older citizens aged 65yo or over from urban areas	Long-term follow up with short term assessment of ambient toxicity prior to each assessment	$PM_{2.5}$ UFP Sulfate NO NO_2 CO O_3 Black carbon	24-hour average: $PM_{2.5}$ (9); UFP (15); Sulfate (3); NO (13 ppb); NO_2 (17 ppb); CO (0.3 ppm); O_3 (25 ppb); Black carbon (0.6).	• None of the pollutants derived from traffic contamination had a significant effect on depressive symptomatology in older dwellers from urban areas complying with regulated health standards for air quality.
Park et al. (2024)	Prospective, longitudinal study	South Korea	National cohort of adult citizens aged 45 yo or above	4 years of follow up (2016-2020)	Long-term exposure to: $PM_{2.5}$ PM_{10} (NO_2 and O_3)	Per each 10 µg/m³ increase in $PM_{2.5}$ and in PM_{10}. Annual average: $PM_{2.5}$ (18-25) PM_{10} (32-45)	• Higher depressive symptoms were positively associated with pollutants toxicity; • Controlling for the influence of NO_2 and O_3, the inhalation of harmful levels of $PM_{2.5}$ and PM_{10} for long periods of time increased the odds of experiencing depressive symptoms by 1.36 (95% CI, 1.20-1.56) and by 1.19 (95% CI, 1.10-1.29), respectively; • OR adding NO_2 of 1.41 (95% CI, 1.22-1.63) for $PM_{2.5}$ and of 1.18 (95% CI, 1.09-1.28) for PM_{10}; • OR adding O_3 of 1.44 (95% CI, 1.25-1.65) for $PM_{2.5}$ and of 1.20 (95% CI, 1.11-1.29) for PM_{10}; • There was a decrease in pollutants toxicity over time.
Turner et al. (2022)		United States of America	Healthy adolescents and diagnosed with asthma (43%)	Longitudinal cohort (2017-2019)	One week pollutant measurement:	Per each 10-fold increment. Mean UFP: 6792 particles/cm³ (6716 for the	Inhaled UFP was associated with physical symptoms of stress (ß=5.92, 95% CI, 0.72-11.13), but not with self-reported psychological stress, depression or anxiety.

			aged between 13-17 yo		UFP (<0.01µm of diameter)	asthma subgroup vs. 6898 for the healthy subgroup); Median individual exposure was not significantly different among clinical (4,660 [584-26,800]) and non-clinical subgroups (4,210 [351-58,300]; Turner et al., 2021).	
Zundel et al. (2022)	Systematic review	Mostly United States of America, China and South Korea	Mainly adult populations	Short- and long-term follow up	Predominantly assessed: $PM_{2.5}$ NO_2 PM_{10}	Not specified	• Ambient toxicity was associated with oxidative stress, inflammatory responses, release of neurotransmitters and metabolites, among other physiological alterations; • Psychiatric disorders, such as depression and anxiety, were related to atmospheric pollution and consistent with changes in brain functioning.
Li et al. (2017)	Randomized Crossover Trial	China	University students with no cardiovascular, respiratory or allergic pre-morbid conditions	4 weeks during November-December	PM $_{2.5}$	Increase of 10 µg/m³ in decontaminated vs. non-purified rooms	• 'Fold change' of 1.18 for cortisone and 1.33 for cortisol levels; • Increment in cortisone by 3.76% (95% CI, 1.84-5.71) and by 7.79% (95% CI, 4.75-10.91) in cortisol levels; • Increase in norepinephrine of 11.70% (95% CI, 7.36-16.22) and in epinephrine of 5.37 % (95% CI, 3.30-7.48); • Increment in systolic blood pressure of 0.86% (95% CI, 0.10-1.62); • Increase of corticotropin releasing hormone of 6.96% (95% CI, 0.01-13.96).

Note. [a]: unless specified otherwise; Yo: years old; PM: particulate matter; NO_2: nitrogen dioxide; NO,: nitric oxide; NO_x: nitrogen oxides; UFP: ultrafine particles; MA: meta-analysis.

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