Air Pollution and Mental Health: A Machine Learning Analysis of Depression and Anxiety in Adults

R. Vijaya Prakash[1]*, Dr. Shrikant Kulkarni*[2] *Prof Dr Midhunchakkaravarthy*[3]*,*
[1] *Lincoln University College, Malaysia;* [2] *Lincoln University College, Malaysia, Research Professor, Sanjivani University, India;* [3]*Lincoln University College, Malaysia.*
Email ID [1]*vijprak.r@gmail.com,* [2]*shrikaant.kulkarni@vit.edu.au* [3]*midhun@lincoln.edu.my*

Abstract: Air pollution remains a major global challenge to public health. Harmful atmospheric components such as particulate matter, nitrogen oxides, and Sulphur oxides, when present in high concentrations, contribute significantly to health risks. Depending on their type and size, these pollutants can increase the likelihood of respiratory and cardiovascular problems, often aggravating existing conditions and raising mortality rates. Fine particles are particularly dangerous because they can cross the blood–brain barrier and interfere with the central nervous system. Research evidence suggests that such exposure can alter brain structures, leading to reduced white matter, neuronal damage, and potentially accelerating the development of neurodegenerative diseases like Alzheimer's and Parkinson's. Beyond these physical effects, air pollution has also been linked to psychological issues, including depression, mood disorders, and suicidal behavior. While various social and medical factors influence mental health, it is increasingly clear that environmental factors play a crucial role as well. This paper examines experimental, clinical, and epidemiological findings to explore the connection between air pollution exposure and the risk of depressive disorders and suicide, with the aim of clarifying their association.

Keywords: Depression, Suicide, Mental health, Air pollution, Air quality, Mood disorders

---

Introduction

Declining air quality has emerged as one of the most pressing environmental challenges worldwide. The atmosphere often contains excessive levels of pollutants such as carbon oxides (CO, CO_2), nitrogen oxides (NO_x), Sulphur oxides (SO_2, SO_3), fluorine, ozone (O_3), hydrocarbons, phenols, and particulate matter (PM). Depending on particle size, PM is categorized into PM_{10}, $PM_{2.5}$, and ultrafine particles smaller than 0.1 µm. Exposure to such pollutants has been strongly associated with adverse health outcomes, particularly increased incidence and mortality from respiratory and cardiovascular illnesses [1]. Reports from the European Environment Agency highlight the severity of this issue, estimating that air pollution contributes to approximately 467,000 premature deaths annually across Europe. In Poland alone, fine particulate matter (PM2.5) is responsible for more than 48,000 early deaths each year, primarily linked to respiratory and cardiovascular complications [2].

Emerging evidence indicates that harmful air pollutants may not only damage the lungs and heart but can also reach multiple organs, including the brain, by crossing the blood–brain barrier. This has raised growing interest in understanding their impact on the nervous system and mental health [3]. Epidemiological findings suggest a connection between poor air quality and psychiatric disorders such as depression, neurodegenerative diseases, and even suicidal behaviour. Although several social, medical, and lifestyle-related factors contribute to suicide risk—including alcohol misuse, social isolation, and stressful life events—mental illness remains the leading cause. Around 60–80% of individuals who die by suicide are affected by depression, and approximately 15% of patients with severe depressive episodes eventually take their own lives [4].

While meteorological conditions such as temperature, humidity, and atmospheric pressure have already been proven to influence mental well-being, the specific role of air pollution in this context remains less understood [5]. This article seeks to address this gap by discussing how air pollution affects the nervous system and by reviewing experimental, clinical, and epidemiological evidence on its relationship with depression and suicide. Beyond exploring these links, the study also aims to emphasize the importance of preventive strategies to mitigate the negative health consequences of poor air quality.

Related work

Air pollutants can impact the nervous system through several mechanisms. Experimental findings indicate that fine particulate matter (PM2.5), along with adsorbed compounds, can induce neuronal apoptosis and cell cycle arrest, which are linked to oxidative stress and genetic damage, ultimately contributing to degenerative changes in the brain [6]. Additionally, certain smog constituents may trigger systemic infections. The immune response generates cytokines that cross the blood–brain barrier via active transport, promoting the migration of monocytes into the central nervous system [7]. Post-mortem brain tissue analysis of individuals from highly polluted regions has revealed increased CD-68, CD-163, and HLA-DR cells, heightened inflammatory markers such as IL-1β, endothelial activation, prefrontal cortex damage, elevated Aβ42 protein levels, and compromised blood–brain barrier integrity [8].

The hippocampus is particularly vulnerable to inflammatory injury due to its high density of pro-inflammatory cytokine receptors, including IL-1β, IL-6, and TNF-α [9, 10]. In the context of affective and cognitive disorders, attention has increasingly shifted toward systemic inflammation as a central etiological factor [10]. Moreover, exposure to ozone and particulate matter has been shown to disrupt cerebrovascular function by altering gene expression in key Vaso reactive pathways, correlating with elevated stroke prevalence in polluted populations [11], [12].

Air pollution is also associated with neurodegenerative outcomes. Studies suggest that polluted environments contribute to reduced white matter volume in older adults and act as a risk factor for Alzheimer's disease, which accounts for 60–80% of dementia cases [13], [14]. Alzheimer's pathology involves amyloid plaque deposition, and animal studies show that exposure to polluted environments accelerates amyloid formation, oxidative stress, and DNA damage in the olfactory bulb, frontal cortex, and hippocampus [15]. Heavy metals such as nickel and vanadium, transported via the nasal pathway, may further damage the olfactory tract and predispose individuals to Parkinson's and Alzheimer's disease [16]. Furthermore, combined exposure to PM2.5, SO2, and NO2 has been shown to impair apoptotic gene regulation (p53, Bax, and bcl-2), resulting in neuropathic changes, memory deficits, and disorientation [17]. These findings underscore the complexity of air pollution's neurological effects, necessitating clinical and epidemiological studies to complement experimental evidence.

Air Pollution and Depression

Depression affects over 350 million people globally, representing nearly 6% of the population [18]. It is a leading cause of reduced quality of life [19], heightened cardiovascular morbidity [20], and increased suicide rates. While multiple environmental contributors exist, air pollution has emerged as a significant factor. Notably, indoor pollutants also elevate depressive symptoms in sensitive populations [21], [22].

Mechanistically, particulate matter can reach the brain through both the bloodstream [23] and the olfactory pathway [24]. This has been confirmed by detecting particles in olfactory neurons and intracellular erythrocytes of the frontal lobe [15]. Due to their small size, particles can penetrate the lungs, traverse the blood–brain barrier, and reach neuronal tissue [25]. Once deposited, they stimulate immune responses, inducing pro-inflammatory cytokines such as IL-1β, TNF-α, and IFN-γ [26].

Animal models demonstrate that PM2.5 exposure alters hippocampal neuronal morphology, leading to inflammatory changes, behavioural disturbances, and impaired cognition [27]. Epidemiological studies support these findings. For instance, Kim et al. found that every 10 μg/m³ increase in PM2.5 significantly raises the risk of depressive episodes, particularly in individuals with chronic illnesses [28]. Similarly, elevated PM10, NO2, and O3 concentrations are linked to depressive symptoms in older adults, especially emotional disturbances [29]. The pathogenesis appears to involve oxidative stress and systemic inflammation, which also contribute to vascular damage and neurodegeneration [30], [31].

Nitrogen dioxide (NO2) and nitrogen oxides (NOx) are also associated with depression risk [27, 29, 32]. However, results vary across regions; a European cohort study reported positive correlations in the Netherlands but negative associations in Norway [35]. Sulphur dioxide (SO2) similarly exhibits harmful psychological effects. Lin et al. observed that women exposed to elevated NO2, PM10, and SO2 during pregnancy experienced higher depressive episode risks, particularly under stress [36].

Table 1. General characteristics of studies concerning relationship between depression and air pollution

Author	Study Design	Exposure Assessment	Pollutant Type	Main Outcome
Perera et al. (2006) [38]	Prospective cohort study	Polycyclic aromatic hydrocarbons (PAH)	Mixed (organic compounds)	High prenatal exposure positively associated with anxious/depressed symptoms and attention problems.
Fonken et al. (2011) [27]	Experimental study	Particulate matter	PM	Long-term exposure to particulate air pollution alters affective responses and impairs cognition.
Lim et al. (2012) [30]	Cross-sectional study	PM10, nitrogen dioxide (NO2), ozone (O3)	PM + Gases	Increases in PM10, NO2, and O3 may elevate depressive symptoms among the elderly.
Davis et al. (2013) [37]	Experimental study	Nano-sized particulate matter (nPM)	PM (ultrafine)	Prenatal exposure to nPM increased depression-like responses.

Author	Study Design	Exposure Assessment	Pollutant Type	Main Outcome
Cho et al. (2014) [22]	Time-stratified case-crossover study	Sulphur dioxide (SO2), PM10, nitrogen dioxide (NO2), carbon monoxide (CO)	PM + Gases	SO2, PM10, NO2, and CO significantly increased the risk of ED visits for depressive episodes, especially among individuals with CVD, diabetes, or asthma.
Wang et al. (2014) [39]	Prospective cohort study	PM2.5, sulfates, black carbon, ultrafine particles	PM	No evidence of a positive association between depressive symptoms and mean pollutant levels in the preceding 2 weeks.
Mokoena et al. (2015) [34]	Experimental study	Ozone	Gaseous	Ozone inhalation induces memory impairment, anxiety, and depression-like effects.
Zijlema et al. (2016) [35]	Cross-sectional cohort study	PM10, nitrogen dioxide (NO2)	PM + Gases	Heterogeneous results of associations of air pollutants and depressed mood.
Szyszkowicz et al. (2016) [32]	Case-crossover study	PM2.5, nitrogen dioxide (NO2), sulphur dioxide (SO2)	PM + Gases	Positive association between exposure to air pollution and visits for depression.
Kioumourtzoglou et al. (2017) [33]	Prospective cohort study	Ozone, PM2.5, PM10	PM + Gases	Long-term exposure to ozone and PM2.5 associated with depression onset, stronger among individuals using antidepressants.
Lin et al. (2017) [36]	Cross-sectional study	Sulphur dioxide (SO2), particulate matter	PM + Gases	Dose-dependent association between air pollution and emotional stress during pregnancy.
Tallon et al. (2017) [28]	Observational, longitudinal, population-based study	PM2.5	PM	Positive association between long-term ambient PM2.5 levels and erectile dysfunction, depression, and stress.

Method, Experiments and Results

Air pollution data for this study were obtained from the Indian Open Government Data (OGD) platform (www.data.gov.in) and regional air-pollution-monitoring stations. These stations, operated under national monitoring frameworks, provided continuous and automated measurements of key ambient pollutants. Stations categorized as *background* or *traffic-specific* were excluded to reduce potential biases in exposure estimates.

In this work, seven machine learning algorithms were applied to develop both regression and classification models. The techniques included *k*-Nearest Neighbors (KNN) [40], Classification and Regression Trees (CART) [41 2], Random Forest (RF) [42], Gradient Boosted Decision Trees (GBDT) [43], CatBoost [44], eXtreme Gradient Boosting (XGBoost) [45], and the Whale Optimization Algorithm (WOA) [46]. All experiments were executed in a Python environment (version 3.9.12) with Spyder (version 5.4.3) as the development interface. Core computational libraries such as *pandas* (v1.4.2), *NumPy* (v1.22.3), *scikit-learn* (v0.0), *CatBoost* (v1.0.6), and *XGBoost* (v1.6.1) were employed for model construction and evaluation.

A total of 20 experimental trials were conducted using a diverse set of optimization techniques with the dual objectives of (1) assessing convergence efficiency and (2) evaluating the statistical significance of the respective optimizers. For benchmarking purposes, the exhaustive feature set was excluded from the evaluation since it functioned solely as a baseline reference. Interestingly, all alternative feature subsets outperformed the complete feature set, thereby indicating that more optimal and generalizable solutions were achievable.

The *k*-Nearest Neighbors (KNN) classifier was iteratively optimized, and experimental findings revealed that setting *k = 5* consistently produced the most reliable classification performance across the datasets considered, in line with prior evidence reported by Chuang [40]. To quantify model effectiveness, standard evaluation metrics—namely accuracy, recall, precision, and the F1-score—were employed, with their mathematical formulations explicitly defined to ensure reproducibility of the evaluation protocol.

In predictive modeling for cognitive impairment, relying solely on accuracy as a performance metric can be misleading. Although accuracy may appear high, its interpretability is limited when dealing with imbalanced datasets, where the proportion of affected individuals is relatively small compared to healthy controls. Under such conditions, a classifier may achieve high accuracy while still failing to correctly identify a substantial number of true impairment cases.

Recall plays a critical role in this context, as it measures the model’s ability to detect many positive cases. A high recall ensures that most individuals at risk of cognitive impairment are flagged for further evaluation. However, recall must be considered alongside precision, which reflects the reliability of positive predictions by quantifying the proportion of true cases among those identified as positive. A model with high recall but low precision could generate excessive false positives, thereby reducing clinical utility.

To address this trade-off, the F1-score serves as a harmonized metric that integrates both recall and precision, offering a balanced view of the model’s effectiveness. Additionally, sensitivity and specificity provide further insights into classification performance: sensitivity reflects the true positive detection rate, while specificity quantifies the ability to correctly identify negative cases. Collectively, these measures provide a more comprehensive framework for evaluating cognitive impairment prediction models than accuracy alone.

The performance metrics are mathematically expressed as:

$$Accuracy = \frac{(TP + TN)}{(TP + FP + TN + FN)} \tag{1}$$

$$Recall = \frac{TP}{(TP + FN)} \tag{2}$$

$$Precision = \frac{TP}{(TP + FP)} \tag{3}$$

$$F1 - Score = 2 * \frac{(Precision * Recall)}{(Precision + Recall)} \quad (4)$$

$$Specificity = \frac{TN}{(TN + FP)} \quad (5)$$

where TPTPTP denotes true positives, TN true negatives, FP false positives, and FN false negatives.

The present investigation utilized six distinct machine learning algorithms—namely, k-nearest neighbours (KNN), Random Forest (RF), Gradient-Boosted Decision Trees (GBDT), Logistic Regression (LR), Cat Boost, and eXtreme Gradient Boosting (XGBoost)—to develop predictive models for dementia. To evaluate model performance, receiver operating characteristic (ROC) curves were generated, revealing accuracy scores ranging from 0.61 to 0.75 (Fig. 1), which indicate modest predictive capacity. Feature importance was further analysed using the XGBoost model. Attributes derived from clinical questionnaires were highlighted in black, while environmental air pollution indicators were represented in red. Among the predictors, the most influential features were found to be age, household income, and ozone (O_3) exposure.

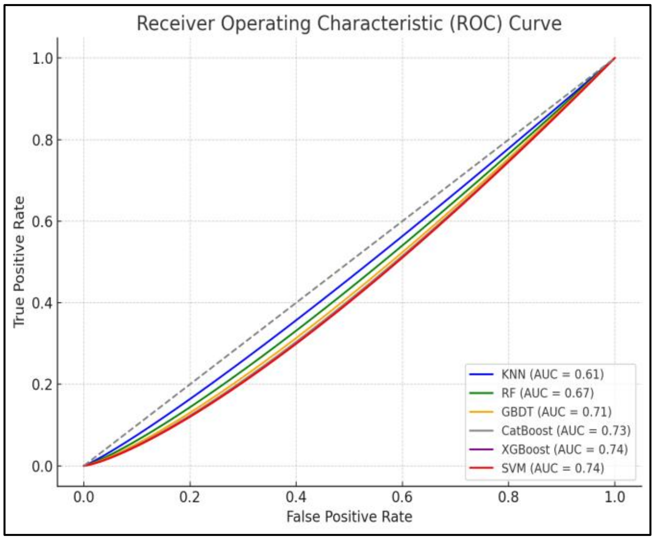

Figure 1. The ROC curves for various machine learning models applied to cognitive impairment prediction

Table 2. Applying machine learning models to predict cognitive impairment results

Criteria	KNN	RF	XGBoost	GBDT	CatBoost	WOAXGBoost
Accuracy	0.92	0.90	0.92	0.93	0.93	0.93
Recall (Sensitivity)	0.51	0.53	0.53	0.52	0.52	0.56
Precision	0.55	0.54	0.61	0.75	0.64	0.64
F1-Score	0.51	0.53	0.53	0.52	0.52	0.54
Specificity	0.99	0.96	0.99	1.00	0.99	0.99

A comparative evaluation of the six algorithms is provided in Table 2. Notably, the hybrid **WOA-XGBoost model**—which integrates feature selection via the Whale Optimization Algorithm (WOA) with classification through XGBoost—yielded superior results. The final selected features included *Age,* *smoking experience (SMK_EXPERIENCE), spousal habits (SPO_HABIT), incense burning practices (INCENSE_CURR), household income (INCOME_FAMILY), and air pollutants such as nitrogen dioxide (NO_2), nitrogen oxides (NO_x), particulate matter ($PM_{2.5}$ and PM_{10}).*

This optimized model achieved robust performance metrics, with an ROC value of **0.74**, Accuracy of **0.91**, Recall of **0.53**, Precision of **0.64**, F1-Score of **0.53**, and Specificity of **0.99**, thereby demonstrating its capacity to balance sensitivity with reliability in dementia risk prediction.

After employing the Synthetic Minority Oversampling Technique (SMOTE), the performance of all six machine learning models improved noticeably, is provided in Table 3. The oversampling process balanced the dataset, thereby enhancing the detection of minority class instances and strengthening the overall predictive capability of the models. The results demonstrated notable gains across Accuracy, Recall, Precision, F1-Score, and Specificity, which confirmed that SMOTE effectively addressed class imbalance issues. Among the models, the hybrid WOAXGBoost approach showed the strongest performance, achieving consistently high scores across all evaluation metrics. The most influential predictors identified by WOAXGBoost included Age, SMK_EXPERIENCE, SMK_2ND, SPO_HABIT, DRUG_USE, INCENSE_CURR, INCOME_FAMILY, O_3, NO_2, and $PM_{2.5}$.

Table 3. Enhancing Machine learning model performance in predicting cognitive impairment using SMOTE.

Criteria	KNN	RF	XGBoost	GBDT	CatBoost	WOAXGBoost
Accuracy	0.83	0.92	0.88	0.94	0.94	0.94
Recall (Sensitivity)	0.81	0.91	0.86	0.92	0.92	0.92
Precision	0.81	0.92	0.89	0.95	0.95	0.95
F1-Score	0.81	0.92	0.87	0.93	0.93	0.93
Specificity	0.87	0.96	0.95	0.99	0.99	0.99

Conclusions

In this research, data from various air quality monitoring stations were integrated to assess air pollution exposure and cognitive function, measured via MMSE, in 31,946 older adults. Machine learning analyses revealed that exposure to air pollutants, particularly $PM_{2.5}$ and NO, was strongly associated with cognitive decline and even dementia. Enforcing stricter environmental regulations and minimizing pollutant exposure could help enhance public health and lower dementia risk. Further research is warranted to develop comprehensive guidelines in this domain.

References

1. Wellenius G, Boyle LD, Wilker E, Sorond F, Coull B. Ambient fine particulate matter alters cerebral hemodynamics in the elderly. Stroke. 2013;44(6):1532–6, https://doi.org/10.1161/ STROKEAHA.111.000395.
2. European Environment Agency. Air quality in Europe – 2016 report. Copenhagen; 2016. p. 83, https://doi.org/10. 2800/80982.
3. Brook RD, Rajagopalan S, Pope CA 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, et al. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the

American Heart Association. Circulation. 2010; 121:2331–78, https://doi.org/10.1161/CIR.0b013e3181dbece1.

4. Tondo L, Isacsson G, Baldessarini R. Suicidal behavior in bipolar disorder: Risk and prevention. CNS Drugs. 2003;17(7):491–511.
5. Padhy SK, Sarkar S, Panigrahi M, Paul S. Mental health effects of climate change. Indian J Occup Environ Med. 2015;19(1):3–7, https://doi.org/10.4103/0019-5278.156997. 718
6. Wei H, Feng Y, Liang F, Cheng W. Role of oxidative stress and DNA hydroxy methylation in the neurotoxicity of fine particulate matter. Neurobiology Aging. 2017;53:48–58, https://doi.org/10.1016/j.tox.2017.01.017.
7. D'Mello C, Le T, Swain MG. Cerebral microglia recruit monocytes into the brain in response to tumor necrosis factor alpha signaling during peripheral organ inflammation. J Neu rosci. 2009; 29:2089–102, https://doi.org/10.1523/JNEURO SCI.3567-08.2009.
8. Calderón-Garcidueñas L, Solt AC, Henríquez-Roldán C, Torres-Jardón R, Nuse B, Herritt L, et al. Long-term air pollution exposure is associated with neuroinflammation, an altered innate immune response, disruption of the blood brain barrier, ultrafine particulate deposition, and accumulation of amyloid beta-42 and alpha-synuclein in children and young adults. Toxicol Pathol. 2008; 36:289–310, https:// doi.org/10.1177/0192623307313011.
9. Maier SF, Watkins LR. Cytokines for psychologists: Implications of bidirectional immune-to-brain communication for understanding behavior, mood, and cognition. Psychol Rev. 1998; 105:83–107.
10. Clark IA, Alleva LM, Vissel B. The roles of TNF in brain dysfunction and disease. Pharmocol Ther. 2010; 128:519–48, https://doi.org/10.1016/j.pharmthera.2010.08.007.
11. Rajanala, Vijaya Prakash. "Air Quality Prediction Using Machine Learning Models". *SGS - Engineering & Sciences*, vol. 1, no. 2, July 2025, https://spast.org/techrep/article/view/5474.
12. Lisabeth LD, Escobar JD, Dvonch JT, Sánchez BN, Majer sik JJ, Brown DL, et al. Ambient air pollution and risk for ischemic stroke and transient ischemic attack. Ann Neurol. 2008; 64:53–9, https://doi.org/10.1002/ana.21403.
13. Woodward NC, Pakbin P, Saffari A. Traffic-related air pollution impact on mouse brain accelerates myelin and neurotic aging changes with specificity for CA1 neurons. Neurobiology Aging. 2017; 53:48–58, https://doi.org/10.1016/j.neurobiolag ing.2017.01.007
14. Sosa-Ortiz AL, Acosta-Castillo I, Prince MJ. Epidemiology of dementias and Alzheimer's disease. Arch Med Res. 2012; 43:600–8, https://doi.org/10.1016/j.arcmed.2012.11.003.
15. Calderón-Garcidueñas L, Maronpot RR, Torres-Jardon R, Henríquez-Roldán C, Schoonhoven R, Acuña-Ayala H, et al. DNA damage in nasal and brain tissues of canines exposed to air pollutants is associated with evidence of chronic brain inflammation and neurodegeneration. Toxicol Pathol. 2003; 31:524–38, https://doi.org/10.1080/01926230390226645.
16. Doty RL. The olfactory vector hypothesis of neurodegenerative disease: Is it viable? Ann Neurol. 2008; 63:7–15, https:// doi.org/10.1002/ana.21327.
17. Ku T, Ji X, Zhang Y, Li G. PM2.5, SO2 and NO2 co-exposure impair neurobehavior and induces mitochondrial injuries in the mouse brain. Chemosphere. 2016; 163:27–34, https://doi.org/10.1016/j.chemosphere.2016.08.009.

18. Vos T, Allen C, Arora M, Barber RM, Bhutta ZA, Brown A, et al. Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990–2015: A systematic analysis for the Global Burden of Disease Study 2015. Lancet. 2016; 388:1545–602, https://doi. org/10.1016/S0140-6736(16)31678-6.
19. Ruo B, Rumsfeld JS, Hlatky MA, Liu H, Browner WS, and Whooley MA. Depressive symptoms and health-related quality of life: The Heart and Soul Study. JAMA. 2003; 290(2):215–21, https://doi.org/10.1001/jama.290.2.215.
20. Whooley MA, Jonge P, Vittinghoff E, Otte C, Moos R, Carney RM, et al. Depressive symptoms, health behaviors, and risk of cardiovascular events in patients with coronary heart disease. JAMA. 2008;300(20):2379–88, https://doi. org/10.1001/jama.2008.711.
21. Sun X, Dai X, Shi Y, Lin Y. [A case-control study on the relationship among indoor air pollution, depression and oncogenesis of lung cancer.
22. Cho J, Choi YC, Suh M, Sohn J, Kim H, Cho SK, et al. Air pollution as a risk factor for depressive episode in patients with cardiovascular disease, diabetes mellitus, or asthma. J Affect Disord. 2014;157(20):45–51, https://doi.org/10.1016/ j.jad.2014.01.002.
23. Oberdörster G, Sharp Z, Atudorei V, Elder A, Gelein R, Kreyling W. Translocation of inhaled ultrafine particles to the brain. Inhal Toxicol. 2004; 16:437–45, https://doi. org/10.1080/08958370490439597.
24. Wang Y, Eliot MN, Koutrakis P, Gryparis A, Schwartz JD, Coull BA, et al. Ambient air pollution and depressive symptoms in older adults: Results from the MOBILIZE Boston Study. Environ Health Perspecta. 2014; 122:553–8, https://doi. org/10.1289/ehp.1205909.
25. Geiser M, Rothen-Rutishauser B, Kapp N, Schurch S, Krey ling W, Schultz H, et al. Ultrafine particles cross cellular membranes by nonphagocytic mechanisms in lungs and in cultured cells. Environ Health Perspecta. 2005; 113:1555–60, https://doi.org/10.1289/ehp.8006.
26. Shwe TTS, Mitsushima D, Yamamoto S, Fukushima A, Funabashi T, Kobayashi T, et al. Changes in neurotransmitter levels and proinflammatory cytokine mRNA expressions in the mice olfactory bulb following nanoparticle expo sure. Toxicol Appl Pharmocol. 2008; 226:192–8, https://doi. org/10.1016/j.taap.2007.09.009.
27. Fonken LK, Xu X, Weil ZM, Chen G, Sun Q, Rajagopalan S, et al. Air pollution impairs cognition, provokes depressive like behaviors and alters hippocampal cytokine expression and morphology. Mol Psychiatry. 2011;16(10):987–95,973, https://doi.org/10.1038/mp.2011.76.
28. Kim Y, Myung W, Won HH, Jeon HJ, Choi J, Carroll BJ, et al. Association between air pollution and suicide in South Korea: A nationwide study. PLoS One. 2015;10(2):e0117929, https://doi.org/10.1371/journal.pone.0117929.
29. Lim YH, Kim H, Kim JH, Bae S, Park HY, Hong YC. Air pollution and symptoms of depression in elderly adults. Environ Health Perspect. 2012;120(7):1023–8, https://doi. org/10.1289/ehp.1104100.
30. Block M, Calderón-Garcidueñas L. Air pollution: Mechanisms of neuroinflammation and CNS disease. Trends Neurosci. 2009;32(9):506–16, https://doi.org/10.1016/j.tins. 2009.05.009.
31. Anisman H, Hayley S. Inflammatory factors contribute to depression and its comorbid conditions. Sci Signal. 2012;5(244):pe45, https://doi.org/10.1126/scisignal.2003579.

32. Szyszkowicz M, Kousha T, Kingsbury M, Colman I. Air pollution and emergency department visits for depression: A multicity case-crossover study. Environ Health Insights. 2016;10:155–61, https://doi.org/10.4137/EHI.S40493.
33. Kioumourtzoglou MA, Power MC, Hart JE, Okereke OI, Coull BA, Laden F, et al. The association between air pollution and onset of depression among middle-aged and older women. Am J Epidemiol. 2017; 29:1–9, https://doi. org/10.1093/aje/kww163.
34. Mokoena ML, Harvey BH, Viljoen F, Ellis SM, Brink CB. Ozone exposure of Flinders Sensitive Line rats is a rodent translational model of neurobiological oxidative stress with relevance for depression and antidepressant response. Psychopharmacology (Berl). 2015;232(16):2921–38, https://doi. org/10.1007/s00213-015-3928-8.
35. Zijlema WL, Wolf K, Emeny R, Ladwig KH, Peters A, Kongsgård H, et al. The association of air pollution and depressed mood in 70,928 individuals from four European cohorts. Int J Hyg Environ Health. 2016;219(2):212–9, https:// doi.org/10.1016/j.ijheh.2015.11.006
36. Lin Y, Zhou L. The impacts of air pollution on maternal stress during pregnancy. Sci Rep. 2017; 7:40956, https://doi. org/10.1038/srep40956.
37. Davis DA, Bortolato M, Godar SC, Sander TK, Iwata N, Pakbin P, et al. Prenatal exposure to urban air nanoparticles in mice causes altered neuronal differentiation and depression-like responses. PLoS One. 2013;8(5):e64128, https:// doi.org/10.1371/journal.pone.0064128
38. Perera FP, Tang D, Wang S, Vishnevetsky J, Zhang B, Diaz D, et al. Prenatal polycyclic aromatic hydrocarbon (PAH) expo sure and child behavior at age 6–7 years. Environ Health Perspect. 2012; 120:921–6, https://doi.org/10.1289/ehp.1104315.
39. World Health Organization [Internet]. Geneva: The Organization; 2015 [cited 2017 Sep 20]. Suicide rates, age standardized. Data by country 2015. Available from: http://apps.who. int/gho/data/view.main.MHSUICIDEASDRv?lang=en.
40. T. Cover and P. Hart, “Nearest neighbor pattern classification,” IEEE Trans. Inf. Theory, vol. 13, no. 1, pp. 21–27, 1967, doi: 10.1109/TIT.1967.1053964.
41. L. Breiman, Classification and Regression Trees. CRC Press, 1984.
42. L. Breiman, “Random forests,” Machine Learning, vol. 45, no. 1, pp. 5–32, 2001, doi: 10.1023/A:1010933404324.
43. J. H. Friedman, “Greedy function approximation: A gradient boosting machine,” Ann. Statist., vol. 29, no. 5, pp. 1189–1232, 2001, doi: 10.1214/aos/1013203451.
44. A. V. Dorogush, V. Ershov, and A. Gulin, “CatBoost: Gradient boosting with categorical features support,” in Proc. 32nd Int. Conf. Neural Information Processing Systems (NIPS 2018), Oct. 2018. [Online]. Available: https://arxiv.org/abs/1810.11363
45. T. Chen and C. Guestrin, “XGBoost: A scalable tree boosting system,” in Proc. 22nd ACM SIGKDD Int. Conf. Knowledge Discovery and Data Mining, 2016, pp. 785–794, doi: 10.1145/2939672.2939785.
46. S. Mirjalili and A. Lewis, “The whale optimization algorithm,” Adv. Eng. Softw., vol. 95, pp. 51–67, 2016, doi: 10.1016/j.advengsoft.2016.01.008.