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RESEARCH ARTICLE

The outdoor physical environment, inflammation and adult psychological distress in a UK general population sample

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Abstract

1. Inflammation is one of the suggested pathways linking exposure to neighbourhood air pollution and greenspace to psychological distress. Yet, inflammation has not been tested formally as a mediating factor in an adult human sample.
2. Using data from 6813 adults participating in Understanding Society, the present study examines longitudinally the joint role of the two neighbourhood-level exposures in psychological distress and the mediating role of inflammation. Annual concentrations of nitrogen dioxide and availability of greenspace in participants' neighbourhoods across England and Wales were examined. Psychological distress was measured with the General Health Questionnaire. Inflammation was assessed with C-reactive protein and fibrinogen.
3. A structural equation model path analysis showed that, in fully adjusted models, an increase in the amount of greenspace in one's neighbourhood was related to lower psychological distress 4 years later, but inflammation did not mediate this relationship.
4. Neighbourhood-level air pollution, specifically nitrogen dioxide, was not associated with psychological distress.

KEYWORDS
air pollution, greenspace, inflammation, psychological distress, Understanding Society

1 | INTRODUCTION

The neighbourhood effects literature has paid much attention to the link between the neighbourhood *social* environment and adult psychological distress, with most studies exploring the role of neighbourhood deprivation or disadvantage (Mair et al., 2008; Paczkowski & Galea, 2010). A small but growing number of longitudinal studies shows that the accumulation of deprivation exposure across the life course has adverse effects on later mental health (Jivraj et al., 2020). In the past two decades, researchers have also been interested in the role that *physical* factors of the residential neighbourhood play in mental health and well-being. Air pollution and greenspace are two aspects of the neighbourhood physical environment that have been associated with *elevated* and *lower* adult psychological distress respectively (Braithwaite et al., 2019; Buoli et al., 2018; Collins et al., 2020; Feng & Astell-Burt, 2018; Pinault et al., 2020; Sass et al., 2017; Wang et al., 2019; Wendelboe-Nelson et al., 2019). Several systematic review and meta-analysis studies on the relationship between air pollution and mental health problems (including depression, anxiety and suicidal ideation) have been published (Braithwaite et al., 2019; Fan et al., 2020; Liu et al., 2021; Trushna et al., 2021; Zeng et al., 2019). Overall, these reviews have

purported mixed findings, with the most consistent evidence for the role of exposure to particulate matter (i.e. $PM_{2.5}$ and PM_{10}) in depressive symptoms. There also is some evidence for a link between particulate matter and suicidal ideation (Braithwaite et al., 2019; Liu et al., 2021), and for an association between nitrogen dioxide (NO_2) and depression (Braithwaite et al., 2019; Liu et al., 2021). The evidence for a link between air pollution and anxiety is mixed; some studies suggest a positive link (Braithwaite et al., 2019) and some studies suggest a negative relationship (Trushna et al., 2021). With regard to greenspace, cross-sectional and longitudinal studies have found associations with several aspects of mental health and well-being in adults, including higher life satisfaction, reduced mental distress, better self-rated mental health, lower perceived stress and lower levels of depression and anxiety (Collins et al., 2020; Crouse et al., 2021; Gascon et al., 2015; Houlden et al., 2018; Wendelboe-Nelson et al., 2019). However, although air pollution and greenspace both seem to be associated with psychological distress, they have rarely been measured simultaneously.

Greenspace and air pollution tend to be spatially, inversely related (Hystad et al., 2014; Thiering et al., 2016). Higher levels of greenspace are typically related to lower levels of air pollution because where there is more greenspace, there is less traffic which, in turn, is associated with lower levels of traffic-related air pollution. Similarly, where there are more roads and more traffic, there is less greenspace but more traffic-related air pollution. This means that there is a systematic, inverse relationship between greenspace and air pollution. Nonetheless, most studies to date investigated the role of *either* greenspace *or* air pollution but not of both exposures together. This means that findings for each exposure may be biased because studies did not account for potential confounding by the other exposure (Klompmaker et al., 2019; Tzivian et al., 2015). A positive effect of greenspace may be explained by lower levels of air pollution; similarly, a negative effect of air pollution may be explained by lower levels of greenspace. This problem has also been identified by Klompmaker et al. (2019) who investigated the associations of greenspace, air pollution and traffic noise with mental health in a large sample of Dutch adults, using single- and multi-exposure models. Indicators of mental health were psychological distress, and prescriptions of anxiolytics, hypnotics and sedatives, and antidepressants. In single-exposure models, the researchers found associations of both greenspace and air pollution with mental health. In multi-exposure models, these associations remained but were attenuated. The researchers concluded that single-exposure models may overestimate the influence of a given exposure on mental health, highlighting the importance of simultaneously investigating the role of spatially correlated exposures. In a recent study, Crouse et al. (2021) assessed the association of residential greenness with mental health in a large Canadian sample. Indicators of mental health were self-reported perceptions of life stress, psychological distress and self-rated mental health. They found that, across all participants, more greenness was associated with lower odds of poor self-rated mental health. However, the association of greenness with psychological distress depended on the level of urbanicity. A negative association of greenness with psychological distress was only found for adults who lived in the active core of cities (not in suburban areas). Although not part of the main analysis, the researchers assessed the role of air pollution in a sensitivity analysis. Adjusting for air pollution did not change their results substantially. Additionally, Mueller et al. (2019) investigated the role of air pollution and greenspace, simultaneously and alongside subjective area perceptions, in the mental health of more than 3000 adolescents from the United Kingdom. The researchers did not find an association of either greenspace or air pollution with mental health. Although this study used data on adolescents, not adults, it assessed the simultaneous impact of air pollution and greenspace on mental health, and it used the same data sources as our study (i.e. Understanding Society, the Multiple Environmental Deprivation Index and air pollution estimates from Mukhopadhyay and Sahu (2018)).

Studying the relationship between physical environment and health comes with the question about underlying mechanisms. Both air pollution and greenspace are linked to health via complex and intertwined pathways. The main way in which air pollution is meant to play a role in mental health is by its effect on the central nervous system (CNS; Buoli et al., 2018). Indeed, air pollution not only affects human respiratory and cardiovascular systems but virtually every cell in the human body. Pollutants enter the lung, enter the blood stream and travel through the whole of the human body, affecting every organ, including the CNS (Schraufnagel et al., 2019). Here they cause inflammation, oxidative stress and changes in the blood–brain barrier, and these effects are thought to be related to neurodevelopmental and neurodegenerative diseases (Block & Calderón-Garcidueñas, 2009; Brockmeyer & D'Angiulli, 2016; Costa et al., 2020; Genc et al., 2012). It has been suggested that the effect of air pollution on the CNS mediates the effect of air pollution on mental health. However, although the link between air pollution and inflammation is supported by the evidence (Chaparro et al., 2018; Chuang et al., 2007; Dadvand et al., 2014; Midouhas et al., 2019; Mostafavi et al., 2015; Pekkanen et al., 2000), the subsequent link between inflammation and mental health is not well studied. Although inflammation has already been linked to depression (Gimeno et al., 2009; Howren et al., 2009; Rook et al., 2013), to the best of our knowledge, this is the first study to investigate the mediating role of inflammation in the link between air pollution and psychological distress in adults.

Several pathways have been suggested that may link greenspace to mental health. These can be distinguished into 'reducing harm' (mitigation), 'restoring capacities' (restoration) and 'building capacities' (instoration), as proposed by Markevych et al. (2017). For example, greenspace may 'reduce harm' by lessening exposure to environmental stressors; 'restore capacities' by helping people to replenish their cognitive resources and to recover from stress; and 'build capacities' by promoting physical activity and social interactions. All these effects of greenspace are, in turn, associated positively with health (Hartig et al., 2014; Markevych et al., 2017). Although the evidence is not yet conclusive, the three pathways above have been tested repeatedly (Zhang et al., 2021). Another way in which greenspace may have a positive effect on mental health is via reduced levels of inflammation.

This is a plausible biological pathway that complements the psychological pathways described above. Exposure to a variety of microbes (including bacteria and fungi) that are found in natural areas is thought to reduce inflammation and to have immunoregulatory benefits (Rook et al., 2013, 2014). In addition to this *direct* effect, inflammation may also be reduced *indirectly* through reduced stress and increased physical activity (two of the pathways mentioned above). Both acute and chronic psychological stress have been linked to increased levels of inflammation (Black, 2002; Marsland et al., 2017; Rohleder, 2014; Steptoe et al., 2007), whereas regular engagement in physical activity has been linked to decreased levels of inflammation (Hamer et al., 2012; Kasapis & Thompson, 2005). This makes neighbourhood greenspace a promising factor for reducing levels of inflammation, both directly and indirectly. Yet, as for air pollution, the mediating role of inflammation in the link between greenspace and mental health is not well studied. However, Midouhas et al. (2019), also using data from Understanding Society, as in the present paper, examined the joint contributions of the amount of air pollution and greenspace in a resident's neighbourhood and later inflammation in adults. They found a relationship between air pollution levels (specifically nitrogen dioxide) and one inflammatory marker, fibrinogen (but not C-reactive protein), adjusting for greenspace. Greenspace availability did not predict inflammation levels in that study, suggesting that inflammation may not mediate its effect on mental health which is shown to be a consistent association in the literature. Moreover, there is research studying the link between greenspace and cortisol, a biological marker of stress (Jones et al., 2021; Roe et al., 2013; Ward Thompson et al., 2012). However, this is the first study to test the mediating role of inflammation (marked by C-reactive protein and fibrinogen) in the link between greenspace and psychological distress in adults.

1.1 | The present study

In the present study, we used longitudinal, multi-exposure models to investigate the joint associations of neighbourhood air pollution and greenspace with psychological distress in 6813 adults participating in Understanding Society (a UK longitudinal household study). We also assessed the mediating role of inflammation in these associations. This study is an important contribution to the literature, investigating a novel, biological pathway between the physical environment and mental health.

2 | METHODS

2.1 | Study sample

Understanding Society is a longitudinal study tracking annually around 40,000 households (at wave 1) in the United Kingdom. Its sample comprises the larger general population sample (GPS; a stratified clustered random household sample recruited in 2009–2011 [wave 1]) and three further samples: the Ethnic Minority Boost Sample, the former British Household Panel Survey (BHPS) sample and the Immigrant and Ethnic Minority Boost Sample. Data are available for 12 waves of interviews thus far (2009–2020) and we used data from waves 1–4 (spanning 2009–2014). We include biomarker measures in our study including C-reactive protein (CRP), fibrinogen and body mass index. These were taken during a nurse visit after either the main wave 2 interview (2010–2012; GPS participants) or the main wave 3 interview (2011–2013; BHPS participants; McFall et al., 2014). To be eligible to take part in the nurse visit, individuals had to have: (1) taken part in the main interview, (2) been aged 16+ years, (3) lived in any of the four UK countries (England, Wales, Scotland and Northern Ireland) and (4) not been pregnant. More information about the sampling and survey timeline are found here: https://www.understandingsociety.ac.uk/documentation/.

Our analytic sample ($n = 6813$) included individuals aged 21+ years (ages ranged 21–102) who took part in the biomarker measurement at wave 2 or 3, had both fibrinogen and CRP data, were living in England/Wales at both wave 1 and wave 2 or 3, had data on air pollution at both wave 1 and wave 2 or 3, had valid data on psychological distress at wave 4 and had complete data on all covariates ($n = 6813$). As our air pollution data for small areas was for England and Wales, our sample comprised individuals living in those countries. In our analytic sample, 2962 (44%) participants were male and 3851 (56%) were female.

2.2 | Ethics

Understanding Society gained ethical approved from the University of Essex Ethics Committee and the National Research Ethics Service. Participants gave oral or written consent to participate.

2.3 | Measures

2.3.1 | Psychological distress at wave 4 (2012–2014)

The General Health Questionnaire-12 (GHQ; Goldberg, 1972), a 12-item self-report measure for minor psychiatric disorders, was used to measure *psychological distress*. The GHQ is able to identify changes in typical functioning and caseness, the likelihood of the individual having a minor psychiatric disorder. The items measure the inability to carry out everyday activities and the appearance of new and distressing symptoms. Additionally, they capture depression, feelings of strain, inability to cope, anxiety-driven insomnia and low confidence. Each frequency item (with a 4-point rating scale) asks whether the participant has recently experienced a certain symptom or behaviour. We converted answers to the 12 items to a scale by recoding[1] the scale for individual items (0 to 3 instead of 1 to 4), and then summing the 12

[1]The GHQ-12 scale recoding from 1–4 to 0–3 respectively is a standardized approach used by Understanding Society to aid in interpretation by allowing participants to have a score of 0. This is a rescaling along a continuum. Therefore, no information is lost and the scale maintains its validity.

answers, giving a scale ranging from 0 (the least distressed) to 36 (the most distressed; Cox et al., 1987).

2.3.2 | Inflammatory markers at wave 2 (2010–2012) or 3 (2011–2013)

Fibrinogen was analysed from citrate plasma samples using a modification of the Clauss thrombin clotting method on the IL-ACS-TOPS analyser. Intra- and inter-assay coefficients of variation were <7%. Fibrinogen was treated as a continuous variable given there are no established clinical thresholds. It was log-transformed for analyses due to its skewed distribution.

C-reactive protein was analysed from serum using the N latex CRP mono assay on the Behring Nephelometer II Analyzer (Dade Behring, Milton Keynes, UK). Intra- and inter-assay coefficients of variation were less than 2%. Systemic inflammation is defined as CRP > 3 mg/L levels. Participants with CRP levels higher than 10 mg/L (5.6% of the analytic sample), typically due to infection, were left out of the sample. CRP was log-transformed and used as a continuous variable.

2.3.3 | Neighbourhood air pollution and greenspace at wave 1 (2009–2011)

Neighbourhood air pollution was measured with modelled estimates of annual concentrations of nitrogen dioxide (NO_2) in micrograms per cubic metre of air ($\mu g/m^3$). Estimates were for Lower Layer Super Output Areas (LSOAs) where study members lived in England and Wales (Mukhopadhyay & Sahu, 2018) from 2009 to 2011. LSOAs are built from Output Areas (the smallest standard areas of UK geography) and, on average, include about 600 homes and 1500 residents. Mukhopadhyay and Sahu (2018) modelled air pollution data collected from the 144 active Automatic Urban and Rural Network (AURN) stations in England and Wales. AURN is the UK's largest automatic monitoring network and is the main network used for compliance reporting against the Ambient Air Quality Directives (http://uk-air.defra.gov.uk/networks). The best geostatistical model obtained by Mukhopadhyay and Sahu (2018) was used to predict NO_2 at the corners of 1-km grid-squares covering England and Wales. These data were used to obtain NO_2 concentration estimates at LSOA level.[2] A 1-unit increase in NO_2 is an increase of $1\,\mu g/m^3$. Note that neighbourhood air pollution was fixed for a given location over the time observed but could vary between waves if the cohort member moved.[3] This is true for all of our neighbourhood variables.

Neighbourhood greenspace was captured using data from the Multiple Environmental Deprivation Index (MEDIx; http://cresh.org.uk/cresh-themes/environmental-deprivation/medix-and-medclass/). The MEDIx is an ordered measure of physical environment deprivation which represents the balance of pathogenic and salutogenic characteristics in a ward (Richardson et al., 2010). The MEDIx measures the amount of greenspace across the United Kingdom at ward level. Electoral wards are the key building block of UK electoral geography and typically have around 5500 residents (http://www.ons.gov.uk/ons/guide-method/geography/beginner-s-guide/administrative/england/electoral-wards-divisions/index.html). The amount of greenspace was measured by combining data from the Coordination of Information on the Environment (CORINE; EEA, 2000) and the 2001 Generalised Land Use Database (GLUD; Office of the Deputy Prime Minister, 2001). CORINE is a land cover dataset from 2000 for the whole of the United Kingdom, derived from remotely sensed satellite imagery. It does not capture small green spaces (the smallest area mapped in the United Kingdom was roughly 1 ha). Thus, it is only sensitive to larger green spaces (e.g. parks). GLUD categorises land use at high geographical resolution (England only) into nine categories: greenspace, domestic gardens, fresh water, domestic buildings, non-domestic buildings, roads, paths, railways and other. GLUD estimates include all vegetated areas larger than $5\,m^2$ in an area (except for domestic gardens), regardless of their accessibility (public or private). CORINE and GLUD were combined to create estimates of the amount of greenspace for each ward in the United Kingdom (Richardson & Mitchell, 2010; https://cresh.org.uk/cresh-themes/green-spaces-and-health/ward-level-green-space-estimates/). An increase in 1 unit of greenspace is equivalent to an increase in one percentage point of greenspace. In our analytic sample in England and Wales (covering both urban and rural areas), the percentage of greenspace per ward ranges 11%–97%.

2.3.4 | Covariates

We adjusted for a range of covariates to reduce possible confounding as well as biases associated with neighbourhood selective sorting. These were neighbourhood deprivation, urbanicity and noise from neighbours, age in years when inflammation was measured (at waves 2/3), sex, individual and household socioeconomic factors (i.e. degree qualification, marital status, housing tenure and household composition), and health-related behaviours and conditions.

Neighbourhood deprivation at wave 1 was measured with the Carstairs index from 2011 capturing material deprivation in LSOAs (Carstairs et al., 1989; Wheeler, 2014). Scores comprise an unweighted combination of four Census variables: car ownership, unemployment, low social class and overcrowding (Carstairs et al., 1989). Each variable was standardised (z-scored). The z-scores for each variable were summed to form a single deprivation score for a given LSOA. A higher Carstairs score represents more deprivation.

[2]For each LSOA, Mukhopadhyay and Sahu (2018) identified the grid cells' corners that fall inside that LSOA. They then took simple averages to arrive at the LSOA level estimates. To obtain uncertainty of the estimates they used the Markov chain Monte Carlo (MCMC) output at each of the grid corners falling within a particular LSOA. MCMC methods, being iterative, produce LSOA aggregated estimates of a particular pollution level at each of its iteration. Those iterative estimates were then used to obtain an estimate of the uncertainty of each LSOA-level estimate of exposure to air pollution.
[3]NO_2 estimates at waves 1 and 2/3 are correlated at 0.82. Therefore this suggests that the level of air pollution exposure is stable across the first 3 years of the study.

Urbanicity at wave 1 was measured with a household-level indicator of whether the address lies within urban settlements (i.e. with a population of 10,000 or more). *Noise* at wave 3 was measured with a question to the respondents about whether they experience noise from neighbours.

Household composition was measured by whether respondents were living alone or with others and *housing tenure* was captured by whether the respondent owned a home or not. *Degree status* was measured by whether participants had obtained a university degree or not and *marital status* was measured by whether participants were partnered (married or in civil partnership) or not.

We measured a number of health-related behaviours and conditions. These included *body mass index* (BMI), *alcohol consumption*, *smoking status*, having a *cardiovascular disease* (CVD) or *diabetes*, having *asthma*, having *bronchitis* and having *cancer*. (Less) *alcohol consumption* was measured with the question, 'How often have you had an alcoholic drink during the last 12 months?' with responses ranging from 1 (*weekly*) to 4 (*never*). *Smoking status* was based on whether the participant reported being a current, regular smoker (at least one cigarette per week) rather than a former regular smoker or having never been a regular smoker. Participants with a *history of cardiovascular diseases or diabetes* (self-reported) had a cardiovascular disease (i.e. angina, heart failure, coronary heart disease, stroke, heart attack and/or emphysema) or diabetes. We also controlled for having a history of another chronic disease (*asthma*, *bronchitis* or *cancer*).

2.4 | Analytic strategy

Analyses were performed in Stata 15.0 (StataCorp, 2017) and Mplus version 8 (Muthén & Muthén, 2017). First, we examined descriptive statistics of the analytic sample and the correlations between the main variables of the study. Following that, we conducted a structural equation model (SEM) path analysis to test the relationship between the physical environment variables (NO_2 and greenspace) and psychological distress. We specified paths for NO_2 and psychological distress and greenspace and psychological distress. We then adjusted for neighbourhood, family and child covariates and, finally, examined the extent to which the observed associations between NO_2 and greenspace and psychological distress were mediated by inflammation. Two models were run—one for each inflammatory marker, fibrinogen and CRP—testing longitudinal associations (where the physical environment variables were measured at wave 1, inflammation at wave 2 or 3 and psychological distress at wave 4). The pathway from NO_2 and greenspace to inflammation was also adjusted for neighbourhood deprivation in order to rule out confounding[4] by the social environment. All models accounted for the complex sampling design of Understanding Society. Stratification by Government Office region, population density and minority ethnic density and non-independence of observations resulting from cluster sampling (households within postal sectors; Asparouhov & Muthen, 2006) were taken into account. To test for mediation in a path analysis framework, we fitted an explicit test for the indirect effects of the predictors (NO_2 and greenspace) via the mediating variable (inflammatory markers) with bootstrapped bias-corrected confidence intervals, recommended by MacKinnon (2012) and Hayes (2017). Bootstrapping confidence intervals involves resampling a single dataset to create multiple simulated samples to calculate the intervals. Additionally, we modelled residual covariances among independent variables and used full information maximum likelihood estimation. Given the complex design of Understanding Society, the Chi-square test of model fit was not available with replicate weights. The value (0.001) of the standardized root mean squared residual (SRMR), a fit index, was less than the recommended cut-off value indicating a good fit to the data (≤0.08) (Hu & Bentler, 1999).

[4]Urbanicity and noise from neighbours were not significantly correlated with inflammation (as reported in the results section); otherwise they would have also been adjusted for.

3 | RESULTS

3.1 | Descriptive statistics

Table 1 displays the descriptive statistics of all study variables. Participants in the analytic sample lived in areas with, on average, 54% greenspace. They were exposed to annual mean concentrations of NO_2 (37 μg/m^3) well above the recently revised legal levels set by World Health Organisation (reduced to 10 μg/m^3 from 40 μg/m^3). The majority lived in urban areas (78%) and did not experience noise from their neighbours (80%).

The correlations among area-level variables, inflammatory markers and psychological distress are shown in Table 2. Higher levels of NO_2 were positively related to psychological distress and fibrinogen and negatively related to greenspace. Greenspace was negatively related to CRP and psychological distress. Finally, both fibrinogen and CRP were related to psychological distress. Noise from neighbours and urbanicity were unrelated to the inflammatory markers and therefore were not adjusted for in the pathways from NO_2 and greenspace to inflammation (only in that from NO_2 and greenspace to psychological distress). Area deprivation *was* significantly associated with both inflammatory markers and therefore was controlled for in these associations.

3.2 | SEM results

3.2.1 | Results for fibrinogen

Results from the fully adjusted SEM for fibrinogen (Table 3) show that NO_2 at wave 1 did not significantly predict psychological distress at wave 4. However, NO_2 predicted increased levels of fibrinogen ($b = 0.01$, SE = 0.006, $p < 0.05$, $\beta = 0.031$). Greenspace

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TABLE 1 Descriptive statistics of the main variables of the study ($n = 6813$)—(NO_2 as continuous raw variable & GHQ caseness)

Continuous		
	Mean	**SD**
Fibrinogen log (g/L)	1.00	0.21
CRP log (mg/L)	0.29	0.91
Psychological distress at wave 4	1.71	3.01
NO_2 ($\mu g/m^3$) at wave 1	37.00	7.29
Greenspace (%) at wave 1	54.29	26.37
Area deprivation wave 1 (*z*-scores)	−0.30	2.91
Higher BMI	24.61	5.78
Age	50.33	16.14
Categorical		
	n	**%**
Noise from neighbours at wave 3		
Yes	1245	20.0
No	5453	80.0
Urbanicity at wave 1		
Urban	5122	77.9
Rural	1688	22.1
Gender		
Male	2962	44.6
Female	3851	55.4
Education		
Degree	2602	37.8
Other	4207	62.2
Marital status		
Partnered	4120	57.3
Not partnered	2693	42.7
Housing tenure		
Owns house/flat	5301	72.9
Does not own	1504	27.1
Household composition		
Living alone	1503	19.6
Living with people	5310	80.4
Current smoker		
Yes	1283	20.7
No	5529	79.3
Alcohol consumption		
Weekly	3787	59.1
Monthly	900	15.0
Less often than month	1082	18.4
Never	429	7.5
Cardiovascular disease or diabetes		
Yes	756	10.0
No	6057	90.0
Asthma		
Yes	723	10.5
No	6090	89.5
Bronchitis		
Yes	96	1.3
No	6717	98.7
Cancer		
Yes	148	1.9
No	6665	98.1

Note: Fibrinogen and CRP are log-transformed. Means and %s are weighted. Ns are unweighted.

Abbreviations: BMI, body mass index; CRP, C-reactive protein; GHQ, General Health Questionnaire.

was a significant predictor of psychological distress ($b = 0.0.01$, SE = 0.004, $p < 0.01$, $\beta = 0.05$) even after adjusting for covariates. However, greenspace did not predict increased levels of fibrinogen. Furthermore, fibrinogen was not related to psychological distress.

3.2.2 | Results for CRP

With regard to CRP, in the fully adjusted SEM (Table 4) greenspace (but not NO_2) at wave 1 significantly predicted psychological distress at wave 4 ($b = 0.01$, SE = 0.004, $p < 0.01$, $\beta = 0.05$). Both area NO_2 and greenspace were unrelated to CRP. In contrast to the results for fibrinogen, an increase in CRP was associated with an increase in psychological distress ($b = 0.15$, SE = 0.074, $p < 0.05$, $\beta = 0.02$).

3.3 | Mediation analysis

3.3.1 | Results for fibrinogen

Fibrinogen did not mediate the relationship between NO_2 and psychological distress (indirect effect: $b = 0.000$, SE = 0.000, 95% CI = −0.001, 0.001, $\beta = 0.000$; total effect: $b = 0.006$, SE = 0.006, 95% CI = −0.006, 0.018, $\beta = 0.014$; direct effect: $b = 0.006$, SE = 0.006, 95% CI = −0.006, 0.018, $\beta = 0.014$). Similarly, fibrinogen did not mediate the relationship between greenspace and psychological distress (indirect effect: $b = 0.000$, SE = 0.000, 95% CI = 0.000, 0.000, $\beta = 0.000$; total effect: $b = 0.004$, SE = 0.002, $p < 0.05$, 95% CI = 0.000, 0.008, $\beta = 0.037$; direct effect: $b = 0.004$, SE = 0.002, $p < 0.05$, 95% CI = 0.000, 0.008, $\beta = 0.037$). Figure 1 shows all paths that were tested in this model.

3.3.2 | Results for CRP

CRP did not mediate the relationship between NO_2 and psychological distress (indirect effect: $b = 0.000$, SE = 0.000, 95% CI = −0.002,

TABLE 2 Correlations among the main study variables.

	1.	2.	3.	4.	5.	6.	7.	8.
1. Fibrinogen log (g/L) at wave 2/3	1							
2. CRP log (mg/L) at wave 2/3	0.39***	1						
3. Psychological distress at wave 4	0.03*	0.06***	1					
4. NO_2 (μg/m^3) at wave 1	0.04***	0.02	0.04**	1				
5. Greenspace (%) at wave 1	−0.01	−0.04**	−0.03*	−0.57***	1			
6. Area deprivation at wave 1	0.06***	0.11***	0.11***	0.36***	−0.42***	1		
7. Noise from neighbours at wave 3	−0.02	−0.01	0.08***	0.09***	−0.11***	0.15***	1	
8. Urban area at wave 1	0.00	0.02	0.03**	0.48***	−0.66***	0.34***	−0.10***	1

Abbreviation: CRP, C-reactive protein.

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

TABLE 3 Fully adjusted model for psychological distress through fibrinogen ($N = 6813$)—(only neighbourhood covariates shown).

	Coefficient (SE)	Beta	95% CI
Direct paths to psychological distress			
1. NO_2 at wave 1 → psychological distress	0.006 (0.006)	0.014	[−0.006, 0.018]
2. Greenspace at wave 1 → psychological distress	0.004* (0.002)	0.037	[0.000, 0.008]
3. Deprivation at wave 1 → psychological distress	0.026 (0.017)	0.026	[−0.007, 0.60]
4. Noise → psychological distress	0.253* (0.106)	0.034	[0.045, 0.462]
5. Urban area at wave 1 → psychological distress	0.106 (0.105)	0.015	[−0.100, 0.311]
6. Fibrinogen → psychological distress	0.045 (0.189)	0.003	[−0.325, 0.415]
Paths to fibrinogen			
1. NO_2 at wave 1 → fibrinogen	0.001*** (0.000)	0.050	[0.001, 0.002]
2. Greenspace at wave 1 → fibrinogen	0.000 (0.000)	0.017	[0.000, 0.000]
3. Deprivation at wave 1 → fibrinogen	0.002* (0.001)	0.030	[0.000, 0.004]

*$p < 0.05$; ***$p < 0.001$.

0.000, $\beta = -0.001$; total effect: $b = 0.006$, SE = 0.006, 95% CI = −0.015, 0.044, $\beta = 0.014$; direct effect: $b = 0.006$, SE = 0.006, 95% CI = −0.015, 0.044, $\beta = 0.015$). Furthermore, there was no mediation by CRP for the relationship between greenspace and psychological distress (indirect effect: $b = 0.000$, SE = 0.000, 95% CI = 0.001, 0.074, $\beta = 0.000$; total effect: $b = 0.004$, SE = 0.002, $p < 0.01$, 95% CI = 0.001, 0.073, $\beta = 0.037$; direct effect: $b = 0.004$, SE = 0.002, $p < 0.05$, 95% CI = 0.001, 0.068, $\beta = 0.037$). Figure 2 shows all paths that were tested in this model.

4 | DISCUSSION

Using a UK GPS, this study examined the roles of neighbourhood air pollution and greenspace in adult psychological distress and the mediating role of inflammation—a key theorised pathway linking the built environment to mental health. Adjusting for a range of possible confounders, including area social deprivation, urbanicity and self-reported noise from neighbours, we found that the amount of air pollution in one's neighbourhood was *unrelated*, but that the amount of greenspace was *related* to psychological distress. The association of greenspace with psychological distress was not mediated by inflammation. These findings suggest that higher levels of greenspace in the neighbourhood may be more important for adult mental health than lower levels of air pollution, and that the link between greenspace and psychological distress may be explained by pathways other than inflammation. However, we need to consider characteristics and limitations of our study that may contribute to our (null-)findings.

4.1 | Air pollution and psychological distress

Overall, our findings are only partly in line with the existing literature. The null association of air pollution with psychological distress was especially unexpected. Both short- and long-term exposures of air pollution have been shown to be associated with psychological distress symptoms and disorders including NO_2 but there are few looking across several years of exposure as we do (across 4 years). Zeng et al.'s (2019) systematic review and meta-analysis found weaker evidence for long-term (more than a year) NO_2 exposures being detrimental than shorter-term ones (less than a month). However, a more recent systematic review and meta-analysis (Borroni et al., 2022) found evidence for both longer-term (more than 30 days) and shorter-term effects of NO_2.

TABLE 4 Fully adjusted model for psychological distress through CRP ($N = 6813$)—(only neighbourhood covariates shown).

	Coefficient (SE)	Beta	95% CI
Direct paths to psychological distress			
1. NO_2 at wave 1 → psychological distress	0.006 (0.006)	0.015	[−0.006, 0.018]
2. Greenspace at wave 1 → psychological distress	0.004* (0.002)	0.037	[0.000, 0.008]
3. Deprivation at wave 1 → psychological distress	0.024 (0.017)	0.024	[−0.009, 0.058]
4. Noise → psychological distress	0.256* (0.106)	0.034	[0.048, 0.465]
5. Urban area at wave 1 → psychological distress	0.106 (0.105)	0.015	[−0.099, 0.312]
6. CRP → psychological distress	0.093* (0.040)	0.028	[0.015, 0.170]
Paths to CRP			
1. NO_2 at wave 1 → CRP	−0.003 (0.002)	−0.020	[−0.006, 0.001]
2. Greenspace at wave 1 → CRP	0.000 (0.000)	−0.010	[−0.001, 0.001]
3. Deprivation at wave 1 → CRP	0.022*** (0.004)	0.071	[0.013, 0.031]

Abbreviation: CRP, C-reactive protein.
*$p < 0.05$; ***$p < 0.001$.

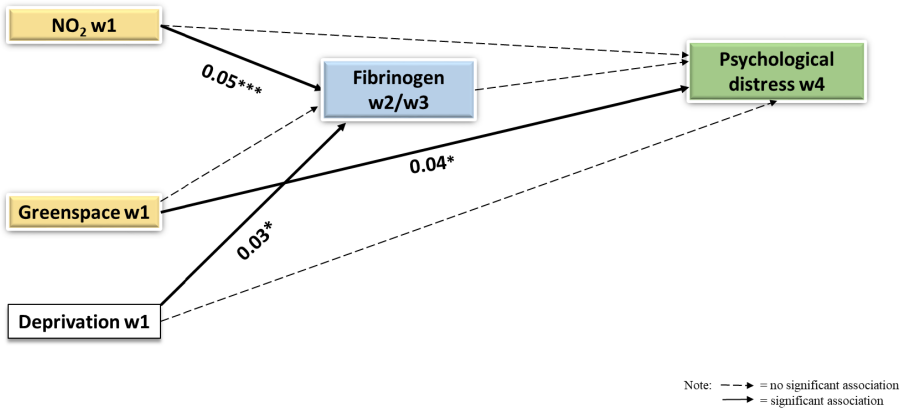

FIGURE 1 SEM and path analysis for Fibrinogen.

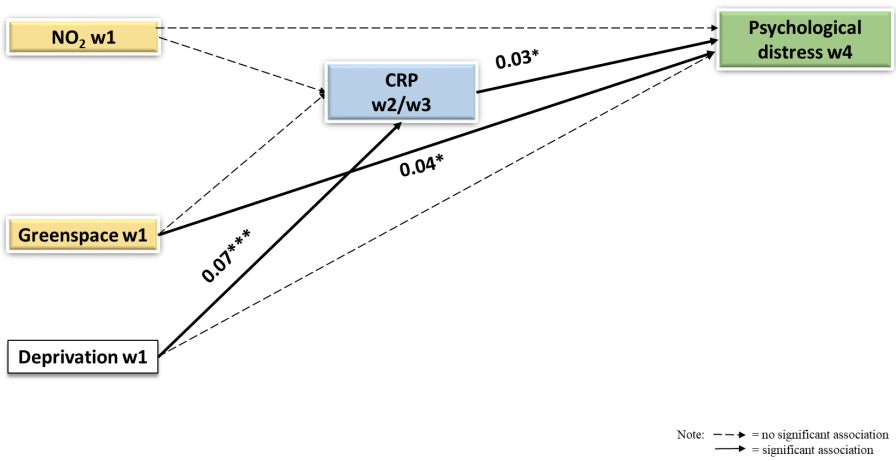

FIGURE 2 SEM and path analysis for C-reactive protein.

Nevertheless, studies have found stronger evidence that PM, another primary air pollutant, has long-term impacts. In Li et al.'s (2021) systematic review, the authors identified a 2% increase in risk of depression as a result of 'short-term' exposure (defined as exposure within 1 month) to PM compared to an 18% increase in risk following 'long-term' exposure (defined as exposure over a period equal to or greater than 1 year).

Additionally, although area deprivation was related to more psychological distress in the correlations matrix, in the fully adjusted model it was not, unexpectedly. Therefore, the deprivation coefficient was likely attenuated by the additional neighbourhood exposures including air pollution and greenspace given the moderate correlations among these variables presented in the matrix. Moreover, the direct relationship between deprivation and psychological distress may have been absorbed by the indirect effect via the inflammatory markers.

Second, although we found an association of air pollution with one marker of inflammation—fibrinogen—it may take much longer for the impact of inflammation to be seen on psychological distress symptoms. In other words, although exposure to air pollution can have an effect on inflammation after a relatively short period of time—after a year (Dadvand et al., 2014; Gao et al., 2020) or even a week (Li et al., 2017)—this may not directly or immediately translate into an effect on mental health. Those participants who develop inflammation as a result of air pollution could exhibit symptoms of depression and anxiety not after 4 years but at a later point in their lives, not captured in the present study. Future research might look at psychological distress several years beyond the 4 years examined here.

Fibrinogen and CRP are both considered to be important for capturing systemic inflammation. CRP has been linked with psychological distress in several studies but fibrinogen shows mixed results in relation to psychological distress (Hamilton et al., 2021). There is evidence to support CRP as a stable marker of inflammation (Lassale et al., 2018) and some research shows that fibrinogen may be more dependent than CRP on individuals' risk profiles including socio-economic status and related factors (Hamilton et al., 2021; Ndrepepa et al., 2014). This supports the fairly weak correlation we found between the two markers in our study, indicating they are picking up on different processes and may explain partly the differential findings in the CRP and fibrinogen models.

Finally, one could suspect that levels of air pollution in our study were too low to affect mental health. Many studies finding links between air pollution and depression have been conducted in countries where pollution levels are especially high, such as Korea (Cho et al., 2014; Lim et al., 2012). The average annual mean concentration of NO_2 in our UK sample was 38 $\mu g/m^3$. This is much higher than the WHO guideline of an NO_2 annual mean concentration of 10 $\mu g/m^3$ (https://www.who.int/news-room/fact-sheets/detail/ambient-(outdoor)-air-quality-and-health), so we can assume that the null association of air pollution with psychological distress is *not* due to too low levels of air pollution. In other words, in theory, levels of air pollution in our sample would have been high enough to affect mental health. Future research might explore this relationship in a sample living in an urban setting with higher levels of pollution.

4.2 | Greenspace and psychological distress

Although air pollution was unrelated to psychological distress, the amount of greenspace in the neighbourhood *was* associated with psychological distress 4 years later (in models adjusted for air pollution). This finding is in line with a large body of literature suggesting an association of (neighbourhood) greenspace with (adult) mental health. Inflammation, our hypothesised pathway, did *not* mediate this association. This suggests that other pathways from greenspace to psychological distress may be more important, such as reducing ambient noise, restoring directed attention capacities or promoting physical activity and social interactions (Hartig et al., 2014; Markevych et al., 2017; Zhang et al., 2021). It is also possible that a longer period of exposure to greenspace may be needed to reduce inflammation levels before they can lower depressive symptoms. We also have no knowledge of the extent to which our participants use greenspace—we only know the amount of greenspace in their immediate surroundings. There is likely to be wide variability in views of and experiences with nature, particularly in urban settings, and these variations may affect the link between the presence of greenspace and inflammation. For example, incidental or passive exposure to greenspace may affect psychological pathways, such as restoration. However, a different level of exposure or experience may be necessary to result in effects on inflammation. One could imagine that a more immersed experience of greenspace may have a greater physiological effect (e.g. via changes in the human microbiome), which, in turn, could reduce inflammation. As a result, future research should aim to understand whether the amount of time and level of immersion in greenspace is important. Similarly, different types of greenspace or greenery are likely to have a different effect on inflammation. As we used a generic measure of greenspace quantity, we could not distinguish between different types of greenspace such as parks, private gardens and greenways. Future research could assess whether different types of greenspace have different effects on inflammation. Moreover, understanding whether there are differences in the exposure effect depending on the amount of greenery is needed. For example, there may be open spaces (e.g. for sports) with little or no greenery that can still reduce stress and benefit psychological health given opportunities for physical activity and social interaction. Future research should also continue to examine the reasons why greenspaces can benefit individuals as they are likely to be complex and multifaceted. In summary, although we did find a relationship between greenspace and psychological distress, which may be explained by pathways not assessed in this study, we did not find a relationship between greenspace and inflammation. It is possible that more time, a certain level of engagement or experience, or particular types of greenery are necessary for greenspace to affect levels of inflammation.

4.3 | Limitations

Our study has a range of limitations. Many of these pertain to our measures of air pollution and greenspace. First, the greenspace measure was based on data from 2000 and 2001 and there is a chance that there were small shifts in greenery between 2000 and

2009. For example, the Committee on Climate Change reported a decrease in urban greenspace in England from 63% to 55% (Committee on Climate Change, 2019) suggesting a small drop in greenspace in cities due to urbanisation. However, despite a possible overestimation of the amount of greenery in residential (urban) areas, a significant association between greenspace and psychological distress was found. Additionally, an LSOA is a unit of UK geography that varies in size and shape. As we do not know where exactly the individual lived in an LSOA, and because the LSOA may not have captured the individual's actual activity space or subjective conception of their neighbourhood, exposure misclassification must be considered as one of the main limitations of this study and could explain any null findings. In other words, inferring the individual's air pollution and greenspace exposure from the LSOA in which they live may not accurately capture their *true* exposure. Relatedly, because long-term exposure may be relevant for the associations of exposures with inflammation and mental health, we should note that we did not have information about our sample's neighbourhood history (prior to 2009). Without these data, we must assume that individuals of our sample had lived in similar neighbourhoods with similar levels of air pollution and greenspace prior to 2009, but this may not always be true. Another limitation (i.e. common in observational studies of neighbourhood effects) is that we cannot make definite inferences about causality. Although the causal effect of greenspace on psychological distress is plausible, and although we accounted for several neighbourhood- and individual-level confounders, we cannot fully rule out reverse causality or residual confounding. Finally, we should also note the drawback of our measures of inflammation only being available at one timepoint limiting our ability to test how *changes* in inflammation over time may mediate the relationship between greenspace and mental health problems.

5 | CONCLUSION

In this study, we used data on a UK GPS to assess the combined associations of neighbourhood air pollution and greenspace with psychological distress, and the mediating role of inflammation in these relationships. We found an association of greenspace with psychological distress, but this was not mediated by inflammation. We did not find an association of air pollution with psychological distress; however, air pollution was associated with a marker of inflammation (fibrinogen). Together these results do not provide evidence for a mediating role of inflammation in physical environment effects on health. However, we have discussed several limitations regarding our measures of air pollution and greenspace, and the longitudinal aspects of our study, which may contribute to our null findings regarding the mediating role of inflammation. Despite the null findings regarding inflammation as a mediator, our findings suggest that greenery promotes good mental health in adults and thus area policies should continue to work on ensuring greenspace provision for all regardless of residential locale. Future research should investigate this novel, biological pathway (inflammation) further, addressing the problem of exposure misclassification and assessing the mediating role of inflammation in the link between short-, mid- and long-term exposure to air pollution, greenspace and other physical environment factors, and mental health.

AUTHOR CONTRIBUTIONS

Emily Midouhas and Eirini Flouri conceived the ideas and designed methodology; Theodora Kokosi prepared and analysed the data; Emily Midouhas led the writing of the manuscript and Marie A. E. Mueller contributed to the literature review. All authors contributed critically to the drafts and gave final approval for publication.

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CONFLICT OF INTEREST STATEMENT

We have no conflict of interest to declare.

DATA AVAILABILITY STATEMENT

The modelling syntax is archived here: https://doi.org/10.5281/zenodo.10034138.

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REFERENCES

Asparouhov, T., & Muthen, B. (2006). *Multilevel modeling of complex survey data*. Paper presented at the Proceedings of the Joint Statistical Meeting in Seattle, WA.

Black, P. H. (2002). Stress and the inflammatory response: A review of neurogenic inflammation. *Brain, Behavior, and Immunity*, *16*, 622–653. https://doi.org/10.1016/S0889-1591(02)00021-1

Block, M. L., & Calderón-Garcidueñas, L. (2009). Air pollution: Mechanisms of neuroinflammation and CNS disease. *Trends in Neurosciences*, *32*, 506–516. https://doi.org/10.1016/j.tins.2009.05.009

Borroni, E., Pesatori, A. C., Bollati, V., Buoli, M., & Carugno, M. (2022). Air pollution exposure and depression: A comprehensive updated systematic review and meta-analysis. *Environmental Pollution*, *292*, 118245. https://doi.org/10.1016/j.envpol.2021.118245

Braithwaite, I., Zhang, S., Kirkbride, J. B., Osborn, D. P. J., & Hayes, J. F. (2019). Air pollution (particulate matter) exposure and associations with depression, anxiety, bipolar, psychosis and suicide

risk: A systematic review and meta-analysis. *Environmental Health Perspectives*, *127*, 126002. https://doi.org/10.1289/EHP4595

Brockmeyer, S., & D'Angiulli, A. (2016). How air pollution alters brain development: The role of neuroinflammation. *Translational Neuroscience*, *7*, 24–30. https://doi.org/10.1515/tnsci-2016-0005

Buoli, M., Grassi, S., Caldiroli, A., Carnevali, G. S., Mucci, F., Iodice, S., Cantone, L., Pergoli, L., & Bollati, V. (2018). Is there a link between air pollution and mental disorders? *Environment International*, *118*, 154–168. https://doi.org/10.1016/j.envint.2018.05.044

Carstairs, V., Morris, R., & Carstairs, V. (1989). Deprivation, mortality and resource allocation. *Journal of Public Health (United Kingdom)*, *11*(4), 364–372. https://doi.org/10.1093/oxfordjournals.pubmed.a042491

Chaparro, M. P., Benzeval, M., Richardson, E., & Mitchell, R. (2018). Neighborhood deprivation and biomarkers of health in Britain: The mediating role of the physical environment. *BMC Public Health*, *18*(1), 801. https://doi.org/10.1186/s12889-018-5667-3

Cho, J., Choi, Y. J., Suh, M., Sohn, J., Kim, H., Cho, S. K., Ha, K. H., Kim, C., & Shin, D. C. (2014). Air pollution as a risk factor for depressive episode in patients with cardiovascular disease, diabetes mellitus, or asthma. *Journal of Affective Disorders*, *157*, 45–51. https://doi.org/10.1016/j.jad.2014.01.002

Chuang, K. J., Chan, C. C., Su, T. C., Te Lee, C., & Tang, C. S. (2007). The effect of urban air pollution on inflammation, oxidative stress, coagulation, and autonomic dysfunction in young adults. *American Journal of Respiratory and Critical Care Medicine*, *176*(4), 370–376. https://doi.org/10.1164/rccm.200611-1627OC

Collins, R. M., Spake, R., Brown, K. A., Ogutu, B. O., Smith, D., & Eigenbrod, F. (2020). A systematic map of research exploring the effect of greenspace on mental health. *Landscape and Urban Planning*, *201*, 103823. https://doi.org/10.1016/j.landurbplan.2020.103823

Committee on Climate Change. (2019). Progress in preparing for climate change – 2019 Progress Report to Parliament.

Costa, L. G., Cole, T. B., Dao, K., Chang, Y. C., Coburn, J., & Garrick, J. M. (2020). Effects of air pollution on the nervous system and its possible role in neurodevelopmental and neurodegenerative disorders. *Pharmacology and Therapeutics*, *210*, 107523. https://doi.org/10.1016/j.pharmthera.2020.107523

Cox, B. D., Blaxter, M., Buckle, A. L. J., Fenner, N. P., Golding, J. F., Gore, M., Huppert, F., Nickson, J., Roth, S. M., Stark, J., Wadsworth, M., & Whichelow, M. (1987). *The health and lifestyle survey. Preliminary report of a nationwide survey of the physical and mental health, attitudes and lifestyle of a random sample of 9003 British adults*. Health Promotion Research Trust.

Crouse, D. L., Pinault, L., Christidis, T., Lavigne, E., Thomson, E. M., & Villeneuve, P. J. (2021). Residential greenness and indicators of stress and mental well-being in a Canadian national-level survey. *Environmental Research*, *192*, 110267. https://doi.org/10.1016/j.envres.2020.110267

Dadvand, P., Nieuwenhuijsen, M. J., Agustí, À., de Batlle, J., Benet, M., Beelen, R., Cirach, M., Martinez, D., Hoek, G., Basagaña, X., Ferrer, A., Ferrer, J., Rodriguez-Roisin, R., Sauleda, J., Guerra, S., Antó, J. M., & Garcia-Aymerich, J. (2014). Air pollution and biomarkers of systemic inflammation and tissue repair in COPD patients. *European Respiratory Journal*, *44*(3), 603–613. https://doi.org/10.1183/09031936.00168813

EEA. (2000). *CORINE land cover 2000*. http://www.eea.europa.eu/publications/COR0-landcover

Fan, S. J., Heinrich, J., Bloom, M. S., Zhao, T. Y., Shi, T. X., Feng, W. R., Sun, Y., Shen, J.-C., Yang, Z.-C., Yang, B.-Y., & Dong, G. H. (2020). Ambient air pollution and depression: A systematic review with meta-analysis up to 2019. *Science of the Total Environment*, *701*, 134721. https://doi.org/10.1016/j.scitotenv.2019.134721

Feng, X., & Astell-Burt, T. (2018). Residential green space quantity and quality and symptoms of psychological distress: A 15-year longitudinal study of 3897 women in postpartum. *BMC Psychiatry*, *18*(1), 348. https://doi.org/10.1186/s12888-018-1926-1

Gao, N., Xu, W., Ji, J., Yang, Y., Wang, S. T., Wang, J., Chen, X., Meng, S., Tian, X., & Xu, K. F. (2020). Lung function and systemic inflammation associated with short-term air pollution exposure in chronic obstructive pulmonary disease patients in Beijing, China. *Environmental Health*, *19*(1), 1–10.

Gascon, M., Mas, M. T., Martínez, D., Dadvand, P., Forns, J., Plasència, A., & Nieuwenhuijsen, M. J. (2015). Mental health benefits of long-term exposure to residential green and blue spaces: A systematic review. *International Journal of Environmental Research and Public Health*, *12*, 4354–4379. https://doi.org/10.3390/ijerph120404354

Genc, S., Zadeoglulari, Z., Fuss, S. H., & Genc, K. (2012). The adverse effects of air pollution on the nervous system. *Journal of Toxicology*, *2012*, 1–23. https://doi.org/10.1155/2012/782462

Gimeno, D., Kivimäki, M., Brunner, E. J., Elovainio, M., de Vogli, R., Steptoe, A., Kumari, M., Lowe, G. D., Rumley, A., Marmot, M. G., & Ferrie, J. E. (2009). Associations of C-reactive protein and interleukin-6 with cognitive symptoms of depression: 12-year follow-up of the Whitehall II study. *Psychological Medicine*, *39*(3), 413–423. https://doi.org/10.1017/S0033291708003723

Goldberg, D. P. (1972). *The detection of psychiatric illness by questionnaire: A technique for the Identification and assessment of non-psychotic illness*. London: Oxford University Press.

Hamer, M., Sabia, S., Batty, G. D., Shipley, M. J., Tabák, A. G., Singh-Manoux, A., & Kivimaki, M. (2012). Physical activity and inflammatory markers over 10 years: Follow-up in men and women from the Whitehall II cohort study. *Circulation*, *126*(8), 928–933. https://doi.org/10.1161/CIRCULATIONAHA.112.103879

Hamilton, O. S., Cadar, D., & Steptoe, A. (2021). Systemic inflammation and emotional responses during the COVID-19 pandemic. *Translational Psychiatry*, *11*(1), 626. https://doi.org/10.1038/s41398-021-01753-5

Hartig, T., Mitchell, R., de Vries, S., & Frumkin, H. (2014). Nature and health. *Annual Review of Public Health*, *35*, 207–228. https://doi.org/10.1146/annurev-publhealth-032013-182443

Hayes, A. F. (2017). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. Guilford Publications.

Houlden, V., Weich, S., de Albuquerque, J. P., Jarvis, S., & Rees, K. (2018). The relationship between greenspace and the mental wellbeing of adults: A systematic review. *PLoS ONE*, *13*, e0203000. https://doi.org/10.1371/journal.pone.0203000

Howren, M. B., Lamkin, D. M., & Suls, J. (2009). Associations of depression with c-reactive protein, IL-1, and IL-6: A meta-analysis. *Psychosomatic Medicine*, *71*(2), 171–186. https://doi.org/10.1097/PSY.0b013e3181907c1b

Hu, L. T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*, *6*(1), 1–55. https://doi.org/10.1080/10705519909540118

Hystad, P., Davies, H. W., Frank, L., Van Loon, J., Gehring, U., Tamburic, L., & Brauer, M. (2014). Residential greenness and birth outcomes: Evaluating the influence of spatially correlated built-environment factors. *Environmental Health Perspectives*, *122*(10), 1095–1102. https://doi.org/10.1289/ehp.1308049

Jivraj, S., Murray, E. T., Norman, P., & Nicholas, O. (2020). The impact of life course exposures to neighbourhood deprivation on health and well-being: A review of the long-term neighbourhood effects literature. *European Journal of Public Health*, *30*, 922–928. https://doi.org/10.1093/EURPUB/CKZ153

Jones, R., Tarter, R., & Ross, A. M. (2021). Greenspace interventions, stress and cortisol: A scoping review. *International Journal of Environmental Research and Public Health*, *18*, 2802. https://doi.org/10.3390/ijerph18062802

Kasapis, C., & Thompson, P. D. (2005). The effects of physical activity on serum C-reactive protein and inflammatory markers: A systematic review. *Journal of the American College of Cardiology*, *45*, 1563–1569. https://doi.org/10.1016/j.jacc.2004.12.077

Klompmaker, J. O., Hoek, G., Bloemsma, L. D., Wijga, A. H., van den Brink, C., Brunekreef, B., Lebret, E., Gehring, U., & Janssen, N. A. H. (2019). Associations of combined exposures to surrounding green, air pollution and traffic noise on mental health. *Environment International*, *129*, 525–537. https://doi.org/10.1016/j.envint.2019.05.040

Lassale, C., Batty, G. D., Steptoe, A., Cadar, D., Akbaraly, T. N., Kivimäki, M., & Zaninotto, P. (2018). Association of 10-year C-reactive protein trajectories with markers of healthy aging: Findings from the English Longitudinal Study of Ageing. *The Journals of Gerontology.*, *74*, 195–203. https://doi.org/10.1093/gerona/gly028

Li, C., Zhou, Y., & Ding, L. (2021). Effects of long-term household air pollution exposure from solid fuel use on depression: Evidence from national longitudinal surveys from 2011 to 2018. *Environmental Pollution*, *283*, 117350. https://doi.org/10.1016/j.envpol.2021.117350

Li, W., Dorans, K. S., Wilker, E. H., Rice, M. B., Ljungman, P. L., Schwartz, J. D., Coull, B. A., Koutrakis, P., Gold, D. R., Keaney, J. F., Jr., Vasan, R. S., Benjamin, E. J., & Mittleman, M. A. (2017). Short-term exposure to ambient air pollution and biomarkers of systemic inflammation: The Framingham Heart Study. *Arteriosclerosis, Thrombosis, and Vascular Biology*, *37*(9), 1793–1800.

Lim, Y. H., Kim, H., Kim, J. H., Bae, S., Park, H. Y., & Hong, Y. C. (2012). Air pollution and symptoms of depression in elderly adults. *Environmental Health Perspectives*, *120*(7), 1023–1028.

Liu, Q., Wang, W., Gu, X., Deng, F., Wang, X., Lin, H., Guo, X., & Wu, S. (2021). Association between particulate matter air pollution and risk of depression and suicide: A systematic review and meta-analysis. *Environmental Science and Pollution Research*, *28*, 9029–9049. https://doi.org/10.1007/s11356-021-12357-3

MacKinnon, D. P. (2012). *Introduction to statistical mediation analysis*. Routledge.

Mair, C., Diez Roux, A. V., & Galea, S. (2008). Are neighbourhood characteristics associated with depressive symptoms? A review of evidence. *Journal of Epidemiology and Community Health*. https://doi.org/10.1136/jech.2007.066605

Markevych, I., Schoierer, J., Hartig, T., Chudnovsky, A., Hystad, P., Dzhambov, A. M., de Vries, S., Triguero-Mas, M., Brauer, M., Nieuwenhuijsen, M. J., Lupp, G., Richardson, E. A., Astell-Burt, T., Dimitrova, D., Feng, X., Sadeh, M., Standl, M., Heinrich, J., & Fuertes, E. (2017). Exploring pathways linking greenspace to health: Theoretical and methodological guidance. *Environmental Research*, *158*, 301–317. https://doi.org/10.1016/j.envres.2017.06.028

Marsland, A. L., Walsh, C., Lockwood, K., & John-Henderson, N. A. (2017). The effects of acute psychological stress on circulating and stimulated inflammatory markers: A systematic review and meta-analysis. *Brain, Behavior, and Immunity*, *64*, 208–219. https://doi.org/10.1016/j.bbi.2017.01.011

McFall, S. L., Conolly, A., & Burton, J. (2014). Collecting biomarkers and biological samples using trained interviewers. Lessons from a pilot study. *Survey Research Methods*, *8*(1). https://doi.org/10.18148/srm/2014.v8i1.5471

Midouhas, E., Kokosi, T., & Flouri, E. (2019). Neighbourhood-level air pollution and greenspace and inflammation in adults. *Health and Place*, *58*, 102167. https://doi.org/10.1016/j.healthplace.2019.102167

Mostafavi, N., Vlaanderen, J., Chadeau-Hyam, M., Beelen, R., Modig, L., Palli, D., Bergdahl, I. A., Vineis, P., Hoek, G., Kyrtopoulos, S. A., & Vermeulen, R. (2015). Inflammatory markers in relation to long-term air pollution. *Environment International*, *81*, 1–7. https://doi.org/10.1016/j.envint.2015.04.003

Mueller, M. A. E., Flouri, E., & Kokosi, T. (2019). The role of the physical environment in adolescent mental health. *Health and Place*, *58*, 102153. https://doi.org/10.1016/j.healthplace.2019.102153

Mukhopadhyay, S., & Sahu, S. K. (2018). A Bayesian spatiotemporal model to estimate long-term exposure to outdoor air pollution at coarser administrative geographies in England and Wales. *Journal of the Royal Statistical Society: Series A (Statistics in Society)*, *181*(2), 465–486. https://doi.org/10.1111/rssa.12299

Muthén, L. K., & Muthén, B. O. (2017). *Mplus user's guide* (8th ed.). Muthén & Muthén. https://doi.org/10.1111/j.1600-0447.2011.01711.x

Ndrepepa, G., Braun, S., Tada, T., King, L., Cassese, S., Fusaro, M., Keta, D., Kastrati, A., & Schmidt, R. (2014). Comparative prognostic value of C-reactive protein & fibrinogen in patients with coronary artery disease. *The Indian Journal of Medical Research*, *140*(3), 392–400.

Office of the Deputy Prime Minister. (2001). *Generalised land use database statistics for England*. ODPM Publications.

Paczkowski, M. M., & Galea, S. (2010). Sociodemographic characteristics of the neighborhood and depressive symptoms. *Current Opinion in Psychiatry*, *23*, 337–341. https://doi.org/10.1097/YCO.0b013e32833ad70b

Pekkanen, J., Brunner, E. J., Anderson, H. R., Tiittanen, P., & Atkinson, R. W. (2000). Daily concentrations of air pollution and plasma fibrinogen in London. *Occupational and Environmental Medicine*, *57*(12), 818–822. https://doi.org/10.1136/oem.57.12.818

Pinault, L., Thomson, E. M., Christidis, T., Colman, I., Tjepkema, M., van Donkelaar, A., Martin, R. V., Hystad, P., Shin, H., Crouse, D. L., & Burnett, R. T. (2020). The association between ambient air pollution concentrations and psychological distress. *Health Reports*, *31*(7), 3–11. https://doi.org/10.25318/82-003-x202000700001-eng

Richardson, E. A., & Mitchell, R. (2010). Gender differences in relationships between urban green space and health in the United Kingdom. *Social Science and Medicine*, *71*, 568–575. https://doi.org/10.1016/j.socscimed.2010.04.015

Richardson, E. A., Mitchell, R., Shortt, N. K., Pearce, J., & Dawson, T. P. (2010). Developing summary measures of health-related multiple physical environmental deprivation for epidemiological research. *Environment and Planning A*, *42*(7), 1650–1668. https://doi.org/10.1068/a42459

Roe, J. J., Thompson, C. W., Aspinall, P. A., Brewer, M. J., Duff, E. I., Miller, D., Mitchell, R., & Clow, A. (2013). Green space and stress: Evidence from cortisol measures in deprived urban communities. *International Journal of Environmental Research and Public Health*, *10*(9), 4086–4103. https://doi.org/10.3390/ijerph10094086

Rohleder, N. (2014). Stimulation of systemic low-grade inflammation by psychosocial stress. *Psychosomatic Medicine*, *76*, 181–189. https://doi.org/10.1097/PSY.0000000000000049

Rook, G. A. W., Raison, C. L., & Lowry, C. A. (2014). Microbial "old friends", immunoregulation and socioeconomic status. *Clinical and Experimental Immunology*, *177*(1), 1–12. https://doi.org/10.1111/cei.12269

Rook, G. A. W., Lowry, C. A., & Raison, C. L. (2013). Microbial "old friends", immunoregulation and stress resilience. *Evolution, Medicine and Public Health.*, *2013*, 46–64. https://doi.org/10.1093/emph/eot004

Sass, V., Kravitz-Wirtz, N., Karceski, S. M., Hajat, A., Crowder, K., & Takeuchi, D. (2017). The effects of air pollution on individual psychological distress. *Health and Place*, *48*, 72–79. https://doi.org/10.1016/j.healthplace.2017.09.006

Schraufnagel, D. E., Balmes, J. R., Cowl, C. T., de Matteis, S., Jung, S. H., Mortimer, K., Perez-Padilla, R., Rice, M. B., Riojas-Rodriguez, H., Sood, A., Thurston, G. D., To, T., Vanker, A., & Wuebbles, D. J. (2019). Air pollution and noncommunicable diseases: A review by the forum of international respiratory societies' environmental committee, part 1: The damaging effects of air pollution. *Chest*, *155*, 417–426. https://doi.org/10.1016/j.chest.2018.10.042

StataCorp. (2017). *Stata statistical software: Release 15*. StataCorp LLC.

Steptoe, A., Hamer, M., & Chida, Y. (2007). The effects of acute psychological stress on circulating inflammatory factors in humans: A review and meta-analysis. *Brain, Behavior, and Immunity*, *21*, 901–912. https://doi.org/10.1016/j.bbi.2007.03.011

Thiering, E., Markevych, I., Brüske, I., Fuertes, E., Kratzsch, J., Sugiri, D., Hoffmann, B., von Berg, A., Bauer, C. P., Koletzko, S., Berdel, D., & Heinrich, J. (2016). Associations of residential long-term air pollution exposures and satellite-derived greenness with insulin resistance in German adolescents. *Environmental Health Perspectives*, *124*(8), 1291–1298. https://doi.org/10.1289/ehp.1509967

Trushna, T., Dhiman, V., Raj, D., & Tiwari, R. R. (2021). Effects of ambient air pollution on psychological stress and anxiety disorder: A systematic review and meta-analysis of epidemiological evidence. *Reviews on Environmental Health*, *36*, 501–521. https://doi.org/10.1515/reveh-2020-0125

Tzivian, L., Winkler, A., Dlugaj, M., Schikowski, T., Vossoughi, M., Fuks, K., Weinmayr, G., & Hoffmann, B. (2015). Effect of long-term outdoor air pollution and noise on cognitive and psychological functions in adults. *International Journal of Hygiene and Environmental Health*, *218*, 1–11. https://doi.org/10.1016/j.ijheh.2014.08.002

Wang, P., Meng, Y. Y., Lam, V., & Ponce, N. (2019). Green space and serious psychological distress among adults and teens: A population-based study in California. *Health and Place*, *56*, 184–190. https://doi.org/10.1016/j.healthplace.2019.02.002

Ward Thompson, C., Roe, J., Aspinall, P., Mitchell, R., Clow, A., & Miller, D. (2012). More green space is linked to less stress in deprived communities: Evidence from salivary cortisol patterns. *Landscape and Urban Planning*, *105*(3), 221–229. https://doi.org/10.1016/j.landurbplan.2011.12.015

Wendelboe-Nelson, C., Kelly, S., Kennedy, M., & Cherrie, J. W. (2019). A scoping review of mapping research on green space and associated mental health benefits. *International Journal of Environmental Research and Public Health*, *16*, 2081. https://doi.org/10.3390/ijerph16122081

Wheeler, B. (2014). *Carstairs Index 2011 for LSOAs*. UK Data Service.

Zeng, Y., Lin, R., Liu, L., Liu, Y., & Li, Y. (2019). Ambient air pollution exposure and risk of depression: A systematic review and meta-analysis of observational studies. *Psychiatry Research*, *276*, 69–78. https://doi.org/10.1016/j.psychres.2019.04.019

Zhang, R., Zhang, C. Q., & Rhodes, R. E. (2021). The pathways linking objectively-measured greenspace exposure and mental health: A systematic review of observational studies. *Environmental Research*, *198*, 111233. https://doi.org/10.1016/j.envres.2021.111233

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