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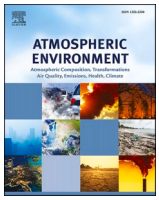

Examining the association between air pollution exposure (PM_{10}, $PM_{2.5}$ and NO_2) across the lifecourse on behavioural and mental health outcomes in adulthood in the Christchurch Health and Development study: a prospective birth cohort study

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HIGHLIGHTS

- Employed a spatial lifecourse epidemiological framework.
- Historical and contemporary air pollution were estimated for a large prospective birth cohort study.
- While air pollution exposure was related to some outcomes, any effects were often small.
- Associations were largely attenuated in fully adjusted models.
- More robust data are needed to confirm our findings.

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ABSTRACT

The adverse effects of air pollution on human physical health are well established, but potential links with behavioural and mental health problems are also suggested. However, evidence is limited, with few studies employing spatial life course methods or considering the effects of unmeasured and residual confounding factors. This study addresses the identified gap by using prospective longitudinal data from the Christchurch Health and Development Study to examine associations between cumulative air pollution exposure and subsequent behaviour and mental health outcomes in adulthood. The study sample consists of a birth cohort of 1265 individuals born during 1977 in Christchurch, New Zealand. Individual-level residential address data were linked to air pollution estimates, including annual black smoke levels (micrograms per cubic meter of air or $\mu g/m^3$) from during pregnancy to age 10 years (1976–1987), and PM_{10} (21–40 years), $PM_{2.5}$ (30–40 years), and NO_2 (40 years) in adulthood. Behavioural (i.e. nicotine, alcohol, cannabis, and other illicit substance use disorders) and mental health outcomes (i.e. depressive symptoms, anxiety disorders, and suicidal ideation) were prospectively assessed from age 21–40 years. Findings from unadjusted population-averaged GEE logistic regression models indicated only weak associations between cumulative air pollution exposure and some behavioural and mental health outcomes. In fully adjusted models, associations were fully attenuated, other than $PM_{2.5}$ exposure and nicotine abuse/dependence (OR = 1.074 [1.0156, 1.136]). While further research is needed to confirm our findings, air pollution exposure may be less important than more proximal factors for behaviour and mental health outcomes in adulthood.

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1. Introduction

Mental health disorders are a leading global health challenge, affecting 13 % of people worldwide (Vigo et al., 2016) and contributing to 4.9 % of global disability-adjusted life years in 2019 (Global and regional, 2022). In New Zealand (NZ), high or very high levels of psychological distress nearly tripled from 4.6 % in 2011/12 to 13.0 % in 2023/24 (New Zealand Ministry of Health, 2024), with notable recent increases in depression and anxiety (New Zealand Ministry of Health, 2024). Although mental illness has multifactorial causes, air pollution is increasingly recognised as an additional risk factor that may also place individuals at increased mental health risk during childhood (Hobbs et al., 2025a) and potentially into the adult years (Braithwaite et al., 2019).

Air pollution is the leading environmental contributor to global disease burden (Kan et al., 2019), causing 4.2 million premature deaths annually (World Health Organisation, 2016). Even low levels of particulate pollution can harm human health (World Health Organisation, 2016). While cardiovascular (de Bont et al., 2022; Khoshakhlagh et al., 2024) and respiratory impacts are well established (Arca-Lafuente et al., 2025; Keleb et al., 2025), emerging evidence links fine particulate matter ($PM_{2.5}$ and NO_2) to cognition (Thompson et al., 2023) and mental health (van der Wal et al., 2021). However, current findings are largely mixed (Braithwaite et al., 2019; Hobbs et al., 2025a. Understanding these relationships is critical given the rising burden of poor mental health (New Zealand Ministry of Health, 2024), urbanisation (van der Wal et al., 2021) and increasing concerns about emissions-induced climate change (Ebi et al., 2021).

Proposed mechanisms include inflammation (Liu et al., 2012), epigenetic changes, hypothalamic–pituitary–adrenal axis dysregulation (Lopez-Duran et al., 2009), and associated cortisol elevations (Li et al., 2017). Inflammation involving the central nervous system has been implicated as having an important role in the pathophysiology of depression (Liu et al., 2012) as well as psychosis (Barron et al., 2017). Several studies have linked fine PM exposure with markers of neuroinflammation such as glial activation and oxidative stress in both human and animal models (Block and Calderón-Garcidueñas, 2009; Calderón-Garcidueñas et al., 2004; Fonken et al., 2011; Levesque et al., 2011). Modifications in DNA methylation patterns (Isaevska et al., 2021), altered lung (Bettiol et al., 2021) and brain development (Lubczyńska et al., 2021) are more common among young children living in polluted areas. These are key predictors of healthy lifecourse development and longevity. Cumulative exposure to air pollution, which starts at conception and continues throughout childhood and into adulthood, may be of particular concern given that this is an extended period of developmental plasticity. As a result, early life exposure may result in cascading physiological changes that increase disease risk not only in childhood (Steinle et al., 2020), but across the lifecourse (Baranyi et al., 2023).

Emerging evidence also links air pollution exposure to several adult mental health outcomes, but high-quality evidence is limited and inconsistent in effect direction and magnitude (Roberts et al., 2019). Two commonly investigated pollutants include $PM_{2.5}$ and PM_{10} (fine and coarse particulate matter) originating from sources such as combustion (fossil fuel burning, residential wood fires), dust (construction, windblown soil), and natural processes (sea spray, volcanic ash, pollen). A recent meta-analysis found small associations between long-term (>6 months) $PM_{2.5}$ exposure and depression (OR = 1.102 (per 10 $\mu g/m^3$) (95 % CI: 1.023, 1.189; p = 0.011), as well as between long-term $PM_{2.5}$ exposure and anxiety, and between short-term PM_{10} exposure and suicide (Braithwaite et al., 2019). Another meta-analysis focusing on depression, reported an increased risk of depression associated with long-term (defined as ≥30 days) exposure to $PM_{2.5}$ (Relative Risk 1.074, 95 % CI: 1.021–1.129) and short-term (<30 days) exposure to PM_{10} (1.009, 1.006–1.012), $PM_{2.5}$ (1.009, 1.007–1.011), NO_2 (1.022, 1.012–1.033) (Borroni et al., 2022). $PM_{2.5}$ is of particular concern due to its small size, which enables deep penetration into the respiratory system and potential passage across the blood–brain barrier (Li et al., 2022; Sangkham et al., 2024), possibly triggering neuroinflammation and exacerbating mental health disorders (Kim et al., 2020; Li et al., 2022). This may account for the stronger and more consistent associations for $PM_{2.5}$ (Kim et al., 2020).

Nitrogen dioxide (NO_2), primarily produced by vehicle emissions and industrial activity has also been linked to mental health. A meta-analysis found a statistically significant association between short-term (<30 days) NO_2 exposure (per 10 μg/m3 increase) and depression (OR: 1.02, 95 % CI: 1.00–1.04) but not in the long-term. However, the credibility of this evidence was low with extreme heterogeneity in effect sizes and direction (Fan et al., 2020). Many reviews include few prospective studies, and even these often suffer from potential bias due to sample selection, exposure misclassification, and inadequate control for confounding—limiting firm conclusions and highlighting the need for robust research (Theron et al., 2022).

Several methodological gaps limit confidence in existing evidence (Fan et al., 2020). Long-term studies are rare, with most following participants for several months or years rather than decades (Baranyi et al., 2023). Few adopt a spatial lifecourse approach, despite cumulative exposure and known sensitive periods of neurodevelopment. For example, pregnancy and the early childhood years (Deng et al., 2025), which have been shown to be important for both brain development and longer-term child cognitive and behavioural outcomes (Barker, 2004). Lifecourse studies have been further hindered by difficulties in reconstructing historical air pollution data over multiple decades using consistent measures (Deng et al., 2024a). Evidence linking air pollution to a range of mental health conditions is scarce (Hobbs et al., 2024), and important time-varying area-level covariates, such as socioeconomic status or greenspace, are often missing (Deng et al., 2024b). Heterogeneous outcome definitions, exposure assessment methods, and reliance on ecological data further limit comparability and risk ecological fallacy. Small sample sizes also reduce statistical power and exposure variability. These limitations underscore the need for high-quality, long-term, individual-level studies (Braithwaite et al., 2019).

In Christchurch, NZ, an air pollution hotspot with historical exposure inequities (Hobbs et al., 2024) we supplemented 40-year prospective birth cohort data with lifecourse air pollution exposure estimates. This study aims to investigate associations between exposure from ages 21–40 and three adult mental health outcomes (depression, anxiety, suicidal ideation) plus four adverse behavioural outcomes (nicotine dependence, alcohol use dependence, cannabis use disorder, other illicit substance use disorder). Using a spatial lifecourse epidemiological framework, we hypothesised that higher exposure would be associated with increased risk of these outcomes after adjustment for relevant covariates.

2. Methods

2.1. Study design

A prospective longitudinal birth cohort study, representative of the Christchurch, New Zealand (NZ) population in mid-1977.

2.2. The Christchurch health and development study

The Christchurch Health and Development Study (CHDS) is a prospective birth cohort study that has collected data on 1265 individuals (630 females) born in 1977 in Christchurch, the largest city in the South Island of NZ (Hobbs et al., 2022). As outlined elsewhere (Deng et al., 2025) the CHDS includes 97% of all births occurring over the four months from April to August 1977. The CHDS cohort has been assessed at 24 age time points since birth, including birth, four months, yearly until age 16 years, and then at age 18, 21, 25, 30, 35 and 40 years (Fergusson and Horwood, 2001). The CHDS gathered data through

various methods, such as parental interviews (birth to age 16); direct interviews with cohort members (from age 8 onwards); teacher reports, standardised tests, and official medical and other records (Deng et al., 2025). All aspects of the CHDS data collection were approved through ethics (#16_STH_144), and all data were collected with the explicit written consent of study participants.

2.3. Participants and settings

Residential address data were sourced from the interview records of all 1265 cohort members, with 1220 (96.4%) successfully geocoded to a NZ address at birth (1977). Specifically, 1075 were resident in Christchurch, 140 in the rest of the South Island and 5 in the North Island. At age 4 years (1981), 1103 of the 1265 original cohort members (80.1%) were successfully geocoded to a NZ address, including: 795 in Christchurch, 197 in the rest of the South Island, and 111 in the North Island (see online supplementary materials Supplement 3 for more details on geocoding processes).

2.4. Exposure: air pollution ($PM_{2.5}$, PM_{10} and NO_2) in adulthood

Air pollution exposure in adulthood was defined using a variety of data sources. Nationwide (census) area estimates of PM_{10} (Fig. 1) were the most consistent over time available from the Health and Air Pollution in New Zealand (HAPINZ) studies from 2001 (HAPINZ), 2006 (HAPINZ 2.0) and 2016 (HAPINZ 3.0) (Ministry for the Environmenta). Nationwide (census) area estimates of $PM_{2.5}$ were available from 2006 to 2016 and NO_2 for 2016 only. HAPINZ employs a comprehensive methodology to assess the health and economic impacts of air pollution across the country (Ministry for the Environmenta). The most recent iteration, HAPINZ 3.0, refines previous assessments by incorporating updated air quality data, epidemiological evidence, and economic modelling approaches. To estimate population exposure, HAPINZ utilises a combination of regulatory air quality monitoring data and atmospheric dispersion modelling. Concentrations of key pollutants have included PM_{10}, $PM_{2.5}$, and NO_2, and are mapped across different regions, accounting for seasonal and geographic variability. Where direct monitoring is unavailable, exposure estimates are derived from satellite observations and land-use regression models. The study also employs source apportionment techniques to quantify the contribution of different pollution sources, including transport, domestic heating, industry, agriculture, and natural emissions such as wildfires and sea salt. The methodological framework employed in HAPINZ aligns with international best practices in air pollution epidemiology and health impact assessment (Ministry for the Environmenta; Ministry for the Environmentb).

Air pollution exposure of CHDS cohort members was then approximated by spatial join of members' based on the exact residential address location. Air pollution data from 2001, 2006 and 2016 were aligned with appropriate data collection periods from 21 to age 40 years. Specifically, CHDS data at age 21 years (1998) and 25 years (2002) were joined using CAU (Census Area Unit) 2001 to HAPINZ 1.0. Then, 30-years (2007) were joined using CAU 2006 to HAPINZ 2.0, and 35-years (2012) was joined with CAU 2013 to average of HAPINZ 1.0 and 2.0, and lastly 40-years (2017) were joined using CAU 2013 to HAPINZ 3.0. Our final air pollution exposure estimates, therefore, covered cohort members annually from each wave of data collection from 21 to 40 years of age for PM_{10}. Data on $PM_{2.5}$ and NO_2 were unavailable for the whole study period, but were included for the available age 30–40 years for $PM_{2.5}$ and for NO_2 for the age 40 years only. Further details on how the CHDS birth cohort childhood air pollution exposure are provided in Online Supplementary Materials Supplement 6. As only black smoke estimates were available for childhood, the main analyses from here onwards focus on air pollution exposure in adulthood only, with sensitivity analyses carried out, which included childhood air pollution (Supplement 11). Including childhood air pollution also resulted in a significant loss of statistical power.

2.5. Outcomes

The CHDS assessed cohort members for behavioural disorders and mental health conditions since the previous interview at ages 18, 21, 25, 30, 35 and 40 years, generating mental health measures for this study during the five intervals: 18–21, 21–25, 25–30, 30–35, and 35–40 years. Data from 21 to 40 years were used to temporally match the maximum extent (PM_{10}) of the air pollution exposure data. The four behavioural outcomes included i) illicit substance abuse disorder, excluding cannabis, ii) alcohol abuse/dependence, iii) cannabis abuse/dependence, iv) nicotine abuse/dependence. The four mental health outcomes in adulthood included: i) depressive symptoms, ii) anxiety, and iii) suicidal ideation, and iv) any of the three aforementioned mental health conditions.

2.5.1. Substance use disorder (age 21–40 years)

At each assessment of the cohort during the period 21–40 years (at ages 21, 25, 30, 35, and 40 years) participants were asked a series of questions based on the CIDI (Composite International Diagnostic Interview) to assess DSM-IV criteria for abuse/dependence on illicit drugs other than cannabis (including amphetamines, opioids, cocaine, hallucinogens, and other drugs). On the basis of this questioning, participants who met criteria for either a) other illicit drug abuse or b) other illicit drug dependence in any assessment period were classified using a dichotomous measure as having other illicit drug abuse/dependence during that period. Participants were then further classified as follows: if there were one or more assessment periods in which participants met criteria for other illicit drug abuse or other illicit drug dependence, then participants were classified as having other illicit drug abuse/dependence during the period 21–40 years.

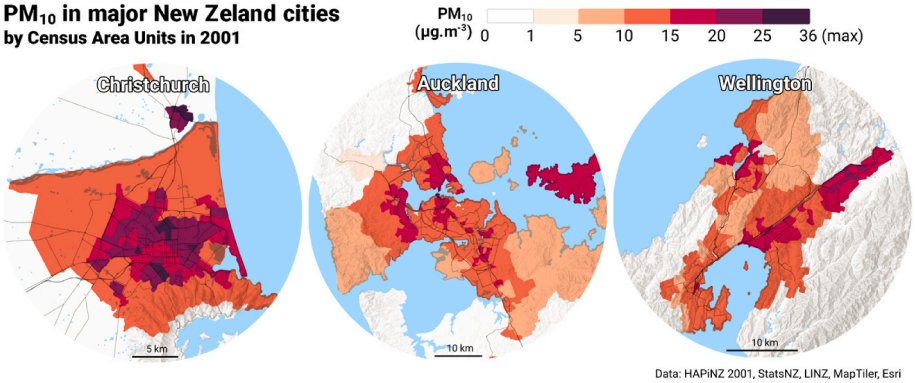

Fig. 1. Map of air pollution (PM_{10}) at age 21 years in New Zealand with a focus on Christchurch, Auckland and Wellington.

2.5.2. Alcohol abuse/dependence (age 21–40 years)

At each assessment of the cohort during the period 21–40 years (at ages 21, 25, 30, 35, and 40 years) participants were asked a series of questions based on the CIDI (World Health Organization, 1993) to assess DSM-IV (American Psychiatric Association, 1994) criteria for alcohol abuse/dependence. On the basis of this questioning, participants who met criteria for either a) alcohol abuse or b) alcohol dependence in any assessment period were classified using a dichotomous measure as having alcohol abuse/dependence during that period. Participants were then further classified as follows: if there were one or more assessment periods in which participants met criteria for alcohol abuse or alcohol dependence, then participants were classified as having alcohol abuse/dependence during the period 21–40 years.

2.5.3. Cannabis abuse/dependence (age 21–40 years)

At each assessment of the cohort during the period 21–40 years (at ages 21, 25, 30, 35, and 40 years) participants were asked a series of questions based on the CIDI (World Health Organization, 1993) to assess DSM-IV (American Psychiatric Association, 1994) criteria for cannabis abuse/dependence. From this questioning, participants who met criteria for either a) cannabis abuse or b) cannabis dependence in any assessment period were classified using a dichotomous measure as having cannabis abuse/dependence during that period. Participants were then further classified as follows: if there were one or more assessment periods in which participants met criteria for cannabis abuse or cannabis dependence, then participants were classified as having cannabis abuse/dependence during the period 21–40 years.

2.5.4. Nicotine abuse/dependence (age 21–40 years)

At each assessment from 21 to 40 years, cohort members were asked a series of questions that combined relevant components of the Composite International Diagnostic Interview (CIDI) and DSM-IV symptom criteria to assess past 30-day symptom criteria for Nicotine abuse/dependence. Using this information, a dichotomous measure reflecting whether the cohort member had ever met diagnostic criteria for a diagnosis of Nicotine abuse/dependence over the period 21–40 years was constructed.

2.5.5. Major depression (age 21–40 years)

From age 18 onwards, symptoms of major depression were assessed using CIDI items and DSM-IV diagnostic criteria. For the purposes of the present analysis sample, members who met DSM diagnostic criteria for experiencing a major depressive episode at any point within an assessment interval were classified as having depressive symptoms during that interval.

2.5.6. Anxiety disorders (age 21–40 years)

The CHDS assessed cohort members using CIDI and DSM-III-R for a range of anxiety disorders (including generalised anxiety disorder, panic disorder, agoraphobia, social phobia, and specific phobia). From age 18 onwards, these anxiety disorders were assessed using CIDI items and DSM-IV diagnostic criteria. Sample members who met DSM diagnostic criteria for one or more anxiety disorders during the assessment interval were classified as having an anxiety disorder during that interval.

2.5.7. Suicidal ideation/attempt (age 21–40 years)

The CHDS asked cohort members about their suicidal ideation through custom-written survey items about whether they had ever considered killing themselves or had attempted suicide during the assessment intervals (Fergusson and Horwood, 2001). Based on their responses, we categorised cohort members as having experienced suicidal ideation during that time interval.

2.5.8. Any mental health problem (age 21–40 years)

To provide an overall measure of the burden of mental health problems, the above three measures of mental disorder (depression, anxiety and suicidal ideation/attempt) were combined to classify participants as to whether they had experienced any internalising mental health problems over the period 21–40 years.

2.6. Covariates

2.6.1. Individual- and family-level covariates

Based on previous CHDS and geospatial research (Deng et al., 2024a, 2024b, 2025; Hobbs et al., 2024, Hobbs et al., 2025a) a number of individual and family covariates were selected from the CHDS database based on their theoretical relevance to air pollution. These covariates were divided into two parts based on the time of measurement, including childhood covariates and adulthood covariates. Childhood covariates included two domains: i) individual characteristics (e.g. sex and ethnicity), b) parents' characteristics (e.g. maternal age, parental relationship stability from birth to age 16 years and parental mental health history). All childhood covariates were time-invariant, as they were either measured at a single time point or summarised over a certain period to construct an overall variable. Adulthood covariates also spanned two domains, including individual circumstances (e.g. the highest educational attainment and welfare dependency) and family circumstances (e.g. whether living with a partner, number of dependent children and residential moves). As outlined in previous research (Deng et al., 2025), all adulthood covariates were time-variant as they were measured and modelled at the same time as the mental health outcomes. We provided a list of all covariates and their full descriptions in the Online Supplementary Materials Supplement 5 (i.e. Table S5) and a Directed Acyclic Graph (DAG) in Supplement 8.

2.6.2. Area-level covariates

We also included three area-level covariates, which were area-level socioeconomic deprivation, population density, and greenspace availability. The selected covariates were theoretically or empirically verified to correlate with air pollution exposure and mental health (Deng et al., 2024a, 2025; Fergusson and Horwood, 2001). All area-level covariates were time-variant and were measured at the time when outcomes were measured. Area-level deprivation from age 18–40 years was measured using NZ Deprivation Index (NZDep) from 1996, 2001, 2006, 2013 and 2018 (Atkinson et al., 2019). NZDep is a widely used composite index measuring socioeconomic status at the small area level across NZ. Population density was defined from age 18–40 years using census data from 1996–2018 and it was calculated by the number of privately occupied dwellings divided by the area of the geographical units (Deng et al., 2025). We further divided the calculated results into time-specific quartiles, with quartile one consisting of the least populated areas and quartile 4 representing the most populated areas. Greenspace availability was measured as the proportion of vegetated areas within a 1500 m buffer area around members' homes using a time-series impervious surfaces data (Deng et al., 2025). The 1500 m buffer area was chosen as previous evidence showed greenspace within this range had the largest mental health benefits for this cohort (Deng et al., 2025). All joins between area-level data and CHDS data followed the closest in time principle. The temporal alignments between CHDS data and area-level covariates are presented in the Online supplementary Materials Table S4 (Supplement 4).

2.7. Statistical analysis

2.7.1. Descriptive analysis

The mean (standard deviation (SD)) of air pollution exposure, stratified by cohort members' characteristics and key outcomes, were used for initial descriptive statistics. We treated all characteristics and outcomes as categorical variables in descriptive analysis. We kept air pollution exposure in the original format in descriptive analyses.

2.7.2. Examining associations between air pollution exposure and outcomes in adulthood

We examined population-averaged associations between air pollution exposure and outcomes in adulthood using a Generalised Estimating Equation (GEE) logistic regression modelling framework. For PM_{10}, we modelled mental health conditions (depressive symptoms, anxiety disorders, and suicidal ideation) at ages 21, 25, 30, 35 and 40 years for the intervals 18–21, 21–25, 25–30, 30–35, and 35–40 years as a function of each air pollution measure using the five waves of repeated-measures data simultaneously. We applied an unstructured correlation matrix to handle within-subject correlations over the five waves of repeated measures. We fully adjusted the models using both childhood and adulthood potential covariates (see online supplementary materials for further details). For $PM_{2.5}$, we used the same approach but included CHDS data from 30 to 40 years to temporally match the available air pollution data for these three waves of data collection. For NO_2, which was only available at age 40 years, we included a binary logistic regression model. Analyses were conducted in STATA V18.0, and a significance level of $\alpha = 0.05$ was set for statistical significance.

2.8. Sample size and bias

The sample included in the GEE model comprised those with at least one outcome measure from age 21–40 years (for PM_{10}) and from age 30–40 years (for $PM_{2.5}$), at least one air pollution exposure measured during this period, and at least one measure available for each covariate during this period. The sample size in the GEE model for PM_{10} was n = 1003 after adjustment, representing 82.1% of the cohort members surviving to 40 years (n = 1221). The sample size in the GEE model for $PM_{2.5}$ was n = 793, representing 64.9% of the cohort members surviving to 40 years. The sample included in the logistic regression model for NO_2 was those with all air pollution exposure data, outcome data, and covariate data available. The sample size of the model was 662, representing 54.2% of the cohort members surviving to 40 years. To examine potential selection bias, we compared the studied samples with the surviving members of the cohort assessed at age 40 in terms of their childhood characteristics. Online Supplementary Material Section 6 Table S5 shows that no statistically significant differences were found across childhood characteristics between cohort members surviving to 40 years and the analytic samples for PM_{10}, $PM_{2.5}$, and NO_2.

Online Supplement Tables S1 and S2 show the sample size and percentage of the surviving cohort for each CHDS assessment from birth to 40 years. Online Supplement Table S1 and Table S2 show modest but statistically significant tendencies for the analysis sample to underrepresent females and cohort members from disadvantaged backgrounds, including, for instance, those of single-parent families, low socioeconomic status families and with maternal smoking. Analyses were conducted using SAS 9.4 and STATA 18. Statistical significance was set at $\alpha = 0.05$. Comparison of the analysis sample with surviving members of the cohort not assessed at age 40 was conducted to examine whether selection bias due to the processes of sample attrition influenced the findings.

3. Results

3.1. Descriptive statistics

Table 1 describes the study sample characteristics of CHDS cohort members at age 21 years, which equates to the baseline adult air pollution exposure of PM_{10}. We summarised air pollution exposure for PM_{10} at age 21 years and key CHDS sociodemographic characteristics, stratified by the presence or absence of any behavioural and any mental health condition at age 21 years. Data are presented as median (interquartile range [IQR]) for continuous variables and frequency (percentage [%]) for categorical variables. We repeated the analysis for $PM_{2.5}$ exposure at age 30 and NO_2 exposure at age 40 (see Online

Table 1
Study sample characteristics of Christchurch Health and Development Study (CHDS) at age 21 years (baseline air pollution exposure of PM_{10}).

Characteristic	Overall (n = 1011)	Any behaviour problems at age 21		Any mental health problems at age 21	
		No (n = 596)	Yes (n = 415)	No (n = 680)	Yes (n = 331)
PM_{10} at age 21 years ($\mu g/m^3$)	19.07 (13.98, 22.54)	18.74 (13.84, 22.53)	19.38 (14.28, 22.66)	19.41 (14.01, 22.82)	18.57 (13.94, 22.08)
Missing	71	44	27	50	21
Sex					
Male	496 (49 %)	262 (44 %)	234 (56 %)	369 (54 %)	127 (38 %)
Female	515 (51 %)	334 (56 %)	181 (44 %)	311 (46 %)	204 (62 %)
Ethnicity at birth					
European or other	874 (86 %)	523 (88 %)	351 (85 %)	590 (87 %)	284 (86 %)
Māori	137 (14 %)	73 (12 %)	64 (15 %)	90 (13 %)	47 (14 %)
Maternal age at birth	26.0 (22.0, 29.0)	26.0 (23.0, 29.0)	25.0 (22.0, 28.0)	26.0 (23.0, 29.0)	26.0 (22.0, 28.0)
Parental relationship stability (age 0–16 years)					
No parental relationship change	635 (63 %)	402 (67 %)	233 (56 %)	450 (66 %)	185 (56 %)
At least one parental relationship change	376 (37 %)	194 (33 %)	182 (44 %)	230 (34 %)	146 (44 %)
Parental mental health history					
No	657 (70 %)	385 (70 %)	272 (70 %)	456 (72 %)	201 (66 %)
Yes	284 (30 %)	167 (30 %)	117 (30 %)	180 (28 %)	104 (34 %)
Missing	70	44	26	44	26
Highest educational attainment					
No formal qualifications	139 (14 %)	49 (8.2 %)	90 (22 %)	81 (12 %)	58 (18 %)
High school qualifications	804 (80 %)	500 (84 %)	304 (73 %)	556 (82 %)	248 (75 %)
Tertiary qualifications below degree level	28 (2.8 %)	18 (3.0 %)	10 (2.4 %)	16 (2.4 %)	12 (3.6 %)
Tertiary qualifications above degree level	40 (4.0 %)	29 (4.9 %)	11 (2.7 %)	27 (4.0 %)	13 (3.9 %)
Whether living with partner					
No	788 (78 %)	481 (81 %)	307 (74 %)	536 (79 %)	252 (76 %)
Yes	223 (22 %)	115 (19 %)	108 (26 %)	144 (21 %)	79 (24 %)
Number of dependent children					
0	927 (92 %)	563 (94 %)	364 (88 %)	633 (93 %)	294 (89 %)
1	69 (6.8 %)	25 (4.2 %)	44 (11 %)	37 (5.4 %)	32 (9.7 %)
2	15 (1.5 %)	8 (1.3 %)	7 (1.7 %)	10 (1.5 %)	5 (1.5 %)
Welfare dependency					
No	605 (60 %)	417 (70 %)	188 (45 %)	428 (63 %)	177 (53 %)
Yes	406 (40 %)	179 (30 %)	227 (55 %)	252 (37 %)	154 (47 %)
Residential moves					
Not moved since last wave	344 (38 %)	236 (44 %)	108 (29 %)	258 (42 %)	86 (29 %)
Moved since last wave	559 (62 %)	298 (56 %)	261 (71 %)	351 (58 %)	208 (71 %)
Missing	108	62	46	71	37
Area-level deprivation	5.00 (3.00, 8.00)	4.00 (2.00, 7.00)	6.00 (3.00, 8.00)	5.00 (2.00, 7.00)	5.00 (3.00, 8.00)

(continued on next page)

Table 1 (*continued*)

Characteristic	Overall (n = 1011)	Any behaviour problems at age 21		Any mental health problems at age 21	
		No (n = 596)	Yes (n = 415)	No (n = 680)	Yes (n = 331)
Missing	71	44	27	50	21
Greenspace availability within 1500m	0.34 (0.22, 0.53)	0.37 (0.23, 0.58)	0.30 (0.20, 0.50)	0.35 (0.22, 0.55)	0.34 (0.21, 0.51)
Missing	71	44	27	50	21
Population density					
Q1 - the least populated	54 (5.8 %)	38 (6.9 %)	16 (4.1 %)	40 (6.3 %)	14 (4.5 %)
Q2	114 (12 %)	67 (12 %)	47 (12 %)	79 (13 %)	35 (11 %)
Q3	237 (25 %)	139 (25 %)	98 (25 %)	153 (24 %)	84 (27 %)
Q4 - the most populated	534 (57 %)	308 (56 %)	226 (58 %)	358 (57 %)	176 (57 %)
Missing	72	44	28	50	22

Data are presented as n (%) for continuous variables, Median (Q1, Q3) for categorical variables.

Supplementary Materials 9 for more details).

Overall, median PM_{10} exposure was higher among members with any behavioural problems and those without mental health problems at age 21 years. Behaviour and mental health problems were more prevalent among members from less stable family backgrounds, with lower educational attainment, those having one or more dependent children, those reporting welfare dependency, and greater residential mobility. In addition, higher prevalence of any behavioural problems is also found among males and those living in areas of higher area-level deprivation and lower greenspace availability. Furthermore, higher prevalence of any mental health problems is found in females, members whose parents had mental health history and those living in areas of higher population density.

3.2. Examining the association between air pollution exposure and behavioral disorder and mental health outcomes in adulthood

We examined associations between PM_{10} exposure in adulthood from age 21 to age 40 years, $PM_{2.5}$ from age 30–40 years and NO_2 at age 40 only, four behavioural disorder outcomes of alcohol abuse/dependence, cannabis abuse/dependence, nicotine abuse/dependence and other illicit substance abuse disorder and four mental health outcomes of depressive symptoms, anxiety disorder and suicidal ideation/attempt, and any mental health condition. Fully adjusted models were adjusted for cohort member sex, ethnicity, as well as maternal age, parental relationship stability, parental mental health history, highest educational attainment, whether parent lives with partner, number of dependent children, welfare dependency, residential moves in adulthood, area-level deprivation, greenspace and population density. Unadjusted and fully adjusted models are shown in Fig. 2 , and the fully adjusted models are shown in online supplementary materials (Supplement 7).

3.2.1. Unadjusted models

The unadjusted models showed several notable associations between adult air pollution exposure and both behavioural disorders as well as mental health outcomes. First, for PM_{10} exposure, associations were noted for alcohol abuse/dependence, nicotine abuse/dependence, depressive symptoms and suicidal ideation/attempt. More specifically, greater PM_{10} air pollution exposure was related to a higher odds of alcohol abuse/dependence (OR = 1.023 [95 % CI 1.006, 1.041], nicotine abuse/dependence (OR = 1.016 [1.001, 1.031]), depression symptoms (OR = 1.017 [1.002, 1.033]) and suicidal ideation/attempt (OR = 1.024 [1.001, 1.048]). Second, for $PM_{2.5}$ exposure from age 30–40 years, associations were noted for cannabis abuse/dependence, nicotine abuse/dependence and any mental health problem. More specifically, greater PM2.5 exposure was related to increased odds of cannabis abuse/dependence (OR = 1.097 [1.005, 1.198]), nicotine abuse/dependence OR = 1.077 [1.033, 1.124] and any mental health problem (OR = 1.036 [1.001, 1.074]). Finally, for NO_2 at age 40 only, several associations were noted with Other illicit substance use disorder, depressive symptoms, anxiety disorders and any mental health problem. More specifically, increased exposure to NO_2 was related to increased odds of other illicit substance use disorder (OR = 1.166 [1.039, 1.308]), depression symptoms (OR = 1.053 [1.011, 1.098]), anxiety disorders (OR = 1.073 [1.023, 1.126]) and any mental health problem (OR = 1.071 [1.031, 1.113]).

3.2.2. Fully adjusted models

In the fully adjusted models, all associations between PM_{10} exposure and NO_2 and any outcome were fully attenuated, with no statistically significant associations detected. Only one association remained between $PM_{2.5}$ and any outcome, showing that an increased $PM_{2.5}$ air pollution exposure was related to increased odds of nicotine abuse/dependence (OR = 1.074 [1.016, 1.136]). Models adjusted also for childhood air pollution exposure showed substantively the same findings (see Online Supplementary Materials - Supplement 11).

4. Discussion

This study is among the first to apply a spatial lifecourse approach to examine the effects of cumulative air pollution exposure on behavioural and mental health outcomes in adulthood. This approach allowed us to examine the role of childhood confounding factors in explaining any observed associations (Desjardins et al., 2023), an issue often overlooked in air pollution research (Theron et al., 2022). This study generated two key findings. First, the associations between air pollution exposure (between age 21 and 40 years) and behavioural disorders and mental health conditions in adulthood were inconclusive due to the small magnitude of any observed associations and the often wide confidence intervals. Second, while several associations were noted in unadjusted models, these statistically significant relationships were largely attenuated in fully adjusted models, highlighting the potential importance of accounting for influencing confounding variables across the lifecourse.

Consistent with several other studies, including longitudinal evidence (Lyons et al., 2024) and meta-analysis (Braithwaite et al., 2019), we observed statistically significant but small associations between the extent of air pollution exposure and a range of adult behaviour problems in unadjusted models. Specifically, greater PM_{10} exposure from 21 to 40 years was associated with an increased risk of alcohol abuse/dependence (OR = 1.023 [1.006,1.041]), nicotine abuse/dependence (OR = 1.016 [1.001,1.031]), depressive symptoms (1.017 [1.002,1.033]) and suicidal ideation/attempt (OR = 1.024 [1.001,1.048]). In addition, higher levels of $PM_{2.5}$ exposure from age 30–40 years were related to a greater odds of cannabis abuse/dependence (OR = 1.097 [1.005, 1.198]), nicotine abuse/dependence (OR = 1.077 [1.033,1.124]) and any mental health problem (OR = 1.036 [1.001,1.074]). However, after adjusting for key confounders across childhood, family background, and at the area-level (i.e. greenspace exposure), these associations were fully attenuated, except for $PM_{2.5}$ exposure and nicotine abuse/dependence (OR = 1.074 [1.0156, 1.136]). This tends to suggest that the observed effects are explained by other factors (i.e. sociodemographic or lifestyle) as opposed to a direct causal relationship with air pollution. However, several other explanations for the null associations in this study are also possible as the mechanistic evidence linking air pollution, especially $PM_{2.5}$ to brain health has been widely demonstrated (Barron et al., 2017; Block and Calderón-Garcidueñas, 2009; Calderón-Garcidueñas et al., 2004; Levesque et al., 2011; Li et al., 2017; Lopez-Duran et al., 2009).

This study used several sensitivity analyses and a directed acyclic

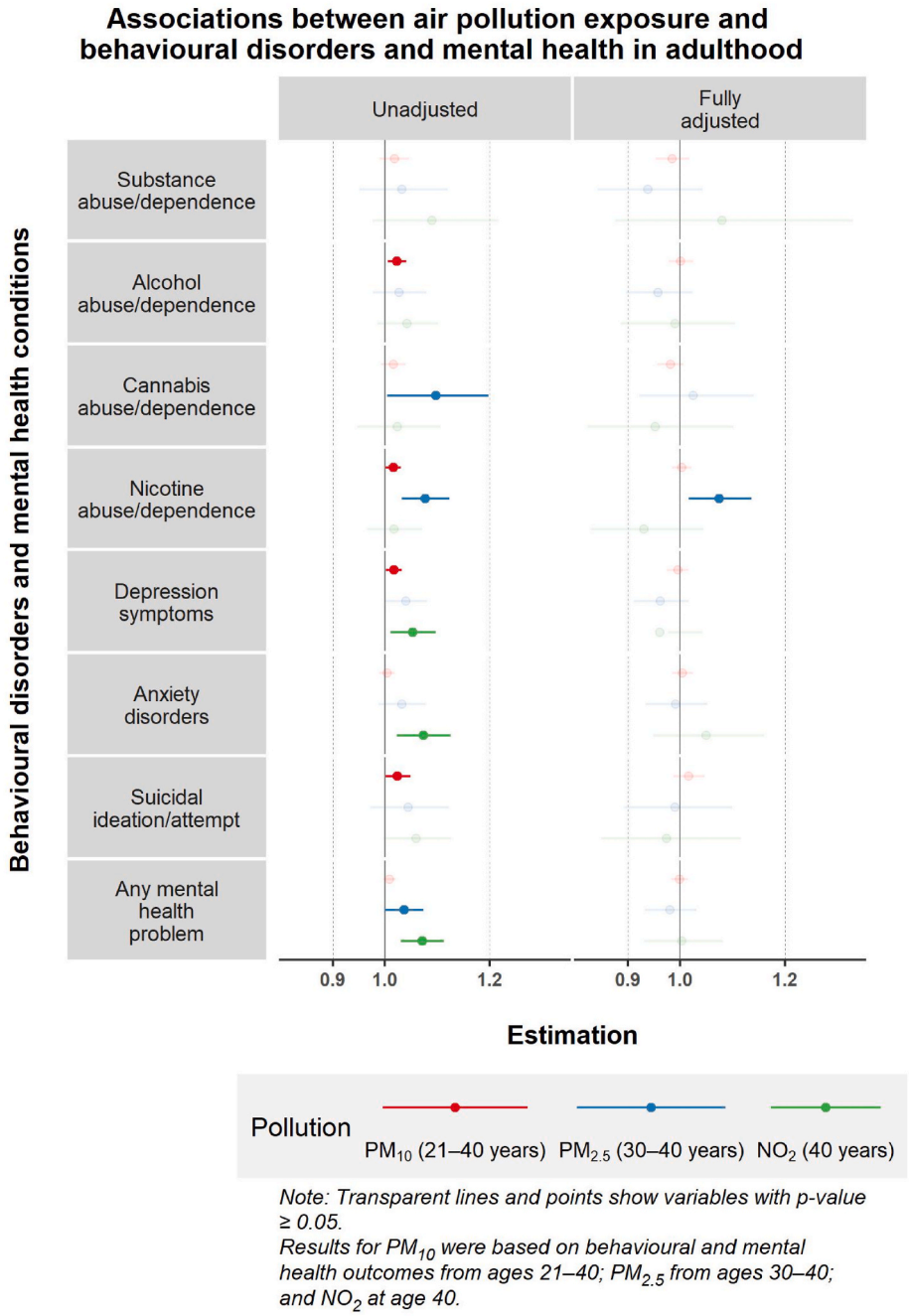

Fig. 2. Examining the association between average air pollution exposure and behaviour and mental health outcomes in adulthood for unadjusted and fully adjusted models.

graph (DAG) to inform covariate selection and minimise confounding bias, and residual and unmeasured confounding, however, some potential concerns remain. For instance, DAGs rely on existing knowledge and assumptions about causal relationships, but if key variables are missing or misclassified, bias can still persist. Early-life socioeconomic conditions, psychosocial stressors, or genetic predispositions may not be fully captured or adequately adjusted for, potentially influencing both air pollution exposure and mental health outcomes (Deng et al., 2024a). In addition, while this study design inherently limits this concern, measurement error in exposure assessment or covariates can also introduce residual confounding if variables are imprecisely recorded or subject to misclassification (Deng et al., 2025). Overadjustment is another important consideration, as adjusting for variables that may be on the causal pathway, such as respiratory health conditions linked to both air pollution and mental health, could lead to a distortion of true effects. IThe variables that are truly causal are still yet to be confirmed (Schisterman et al., 2009). These limitations highlight the need for careful sensitivity analyses, improved data collection strategies, and complementary methodological approaches, such as negative control analyses or instrumental variable methods, to strengthen causal inference in future research.

To assess the robustness of our findings, we conducted sensitivity analyses examining varying levels of covariate adjustment and different modelling approaches. While results remained largely consistent, some associations became stronger or weaker depending on the included covariates. While this may highlight the importance of precise adjustment in air pollution studies, it did not substantively change the findings of this study. Additionally, effect modification by factors such as

socioeconomic status or pre-existing health conditions were unable to be explored due to limitations in sample size limiting statistical power; however, interactions may be significant in future research with stronger effects noted for some population groups over others. Given the potential exposure misclassification and potential for residual confounding, future studies could consider approaches to account for this or more detailed geospatial exposure models to improve causal inference.

This study has several notable strengths which improve the current state of science. First, this study attempted to integrate air pollution data into a prospective birth cohort study design that reconstructed individuals' residential air pollution exposure at the address-level from birth to age 40 years. While not all pollutants were available over this whole study period, the longitudinal component ensures a clearer temporal relationship between exposure and outcome than is possible with a cross-sectional study design. In addition, this study examines multiple mental health conditions, extending our understanding of how air pollution may be associated with a range of outcomes, including some rarely investigated in longitudinal research. Due to the birth cohort study design, we were able to use a range of individual, family, and area-level covariates to adjust the associations between air pollution and our key study outcomes, for example parental mental health history. Accounting for these confounders is crucial for understanding the life-course impacts and reduces the likelihood of residual or unmeasured confounding.

The largely null associations observed in this study may partly stem from limitations in the air pollution metrics available. For instance, PM_{10}, had the most complete temporal coverage from ages 21 to 40, however, it is thought to be unable to cross the blood-brain barrier, making it a less relevant exposure for direct neurological effects (Li et al., 2022). While $PM_{2.5}$ can penetrate the blood-brain barrier, data for this pollutant were only available from ages 30 to 40, restricting our ability to conduct comprehensive lifecourse analyses. Despite this, a ten-year exposure window is an important addition to most current evidence. Similarly, NO_2 exposure was only measured at age 40, limiting analyses to a cross-sectional design and preventing assessment of cumulative or long-term exposure effects. Furthermore, childhood air pollution data were only available in the form of black smoke, a pollutant with limited comparability to more recent exposure measures, further complicating our efforts to examine early-life contributions to mental health outcomes (Hobbs et al., 2025a).

Data on residential locations in the CHDS birth cohort childhood (0–16 years) is available annually, however, from age 21 onwards, the data for address history is only available around every five years. Therefore, it is possible that addresses during this period are missed, and if participants went overseas or are lost for a period of time and then re-enter the study based with a NZ address, there is no way of knowing if they were overseas in a polluted area, which introduces further bias. This may lead to additional exposure misclassification, particularly for subgroups with higher mobility. Indeed, to provide some quantitative indication of the scale of mobility, the recorded number of cohort members overseas at age 21, 25, 30, 35, and 40 was 3.8%, 12.9%, 18.1%, 17.3%, 13.8% respectively. While we were not able to test the impact of exposure misclassification, future studies with more granular spatiotemporal exposure estimates and occupational histories are needed to address this limitation (Lopez-Duran et al., 2009). This further emphasises the need for future studies with more consistent, long-term air pollution data spanning early life to adulthood (Zundel et al., 2022).

In addition, this study only considered air pollution exposure at residential locations, while individuals also spend time outside their homes, such as near schools or at workplaces, and this leads to exposure misclassification (Campbell et al. 2021). Despite this, it is difficult to follow the birth cohort over 40 years with individualised activity spaces through, for instance, GPS that may be more feasible in the shorter-term but was not available at the time of data collection for CHDS. Perhaps most importantly, due to the time frame of the CHDS birth cohort, we were limited by what pollutants were measured. While estimates could have been made to convert black smoke estimations in the 1970's and 1980's to more contemporary $PM_{2.5}$, these would have been made on several large assumptions which were difficult to overlook. In addition, while birth cohort sample sizes are small by contemporary cohort methods, other possible data linkage projects in NZ, such as the Integrated Data Infrastructure, seldom have the richness of data availability from childhood. While sample attrition was notable, we found no statistically significant differences in childhood characteristics between the modelling samples and cohort members who survived to age 40. The attenuation noted in this study could reflect the influence of confounding by individual-, family-, or area-level factors, which may themselves be strong predictors of mental health outcomes. However, it is also possible that adjusting for multiple correlated covariates may have led to over-adjustment, especially if some of these covariates lie on the causal pathway between early-life disadvantage and later health outcomes (Hobbs et al., 2025b). Further research using causal modelling approaches may help to clarify these complex relationships; however, this requires further work to disentangle.

5. Conclusion

In summary, the study's findings revealed a complexity in the association between air pollution and adverse mental health and developmental outcomes. While some evidence suggests a concerning association between air pollution and adverse mental health outcomes, this study identified few associations; however, further research is needed to confirm these findings. It remains to be established to what extent cumulative air pollution exposure is associated with adverse mental health and behavioural disorders in adulthood.

CRediT authorship contribution statement

M. Hobbs: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **B. Deng:** Writing – review & editing, Writing – original draft, Visualization, Methodology, Formal analysis, Data curation. **L.J. Woodward:** Writing – review & editing, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **L. Marek:** Writing – review & editing, Visualization, Methodology, Formal analysis, Data curation. **P. Eggleton:** Writing – review & editing, Data curation. **J.M. Boden:** Writing – review & editing, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.atmosenv.2025.121553.

Data availability

The Christchurch Health and Development Study data are not freely available as we do not currently have ethical approval to upload these data to any repository and this prevents us from sharing this data in this way. However, the data are available on request subject to approval by the CHDS Director at: chds.uoc@otago.ac.nz.

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