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Childhood air pollution exposure is related to cognitive, educational and mental health outcomes in childhood and adolescence: A longitudinal birth cohort study

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ABSTRACT

Background: A growing body of evidence supports an association between air pollution exposure and adverse mental health outcomes, especially in adulthood however, very little is known about the effects of early life air pollution exposure during childhood. We examined longitudinal associations between the extent and timing of children's annual air pollution exposure from conception to age 10 years and a wide range of cognitive, educational and mental health outcomes in childhood and adolescence that were assessed prospectively as part of a large birth cohort study.

Methods: We linked historical air pollution data ($\mu g.m^{-3}$) from pregnancy to age 10 years (1976–1987) using the addresses of all cohort members (n = 1265) of the Christchurch Health and Development Study (CHDS) who were born in New Zealand in mid-1977. Latent Class Growth Mixture Models were used to characterise different trajectories of air pollution exposure from the prenatal period to age 10 years. We then examined associations between these air pollution exposure trajectories and 16 outcomes in childhood and adolescence using R Studio and Stata V18.

Findings: Four air pollution exposure trajectories were identified: i) low, ii) persistently high, iii) high prenatal and postnatal, and iv) elevated pre-school exposure. While some associations were attenuated, after adjusting for a variety of covariates spanning childhood, family sociodemographic background and family functioning characteristics, several associations remained. Relative to the lowest exposure trajectory, persistently high and high prenatal and postnatal exposure were both related to attentional problems. High prenatal and postnatal was also related to higher risk of substance abuse. Elevated pre-school exposure was associated with conduct problems, lower educational attainment and substance abuse and persistently high childhood exposure increased risk of substance abuse.

Conclusions: Our study highlights potential adverse and longer-term impacts of air pollution exposure during childhood on subsequent development in later life.

1. Introduction

Outdoor air pollution is one of the most important environmental threats to human health (World Health Organisation, 2016; World Health Organization (WHO), 2006). It is estimated to cause 3 million premature deaths globally, with disadvantaged populations disproportionately affected (World Health Organisation, 2016). To date, most research has focused on the adverse impacts of air pollution on physical health (National Institute of Environmental Health Sciences, 2024). Disparities in pollution-related physical health outcomes include, but

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are not limited to, respiratory and cardiovascular diseases which have been consistently linked to variations in air pollution exposure (Pearce et al., 2006; Braithwaite et al., 2019; McGowan et al., 2002). While air pollution's harmful physical health sequelae are well known (Park et al., 2021; Khreis et al., 2017; Watts et al., 2021), associations between air pollution exposure and mental illness have not yet been established (Braithwaite et al., 2019) with results less consistent (Arseneault et al., 2011; Roberts et al., 2019; Theron et al., 2021). Moreover, even less evidence has considered the detrimental impact of air pollution exposure during childhood on developmentally important cognitive functioning and mental health in adolescence (Reuben et al., 2021). The causes of mental health are clearly multifaceted in aetiology however, given the already high (Vigo et al., 2016) and rising burden of mental illness worldwide (Wu et al., 2023), especially during childhood and adolescence (Shorey et al., 2022), understanding the potential effect of air pollution on a range of developmental outcomes to adolescence is especially crucial considering the human and societal cost of poor mental health and reduced human potential (Whiteford et al., 2015), the global shift toward urban living (World Health Organisation, 2016) and the backdrop of emissions-induced climate change (Newbury et al., 2024).

Childhood is critically important as a foundational phase in human development, characterised by heightened sensitivity to environmental influences which often shape lifelong health and development (Tooley et al., 2021; Desjardins et al., 2023; Hobbs and Atlas, 2019). This neuroplasticity is particularly marked during the prenatal period and early childhood years which form an important foundation for children's cognitive, emotional and behavioural growth/development (National Scientific Council on the Developing Child, 2023; Black et al., 2017; Barker, 2004). These periods of life represent windows of heightened opportunity but also vulnerability, where to environmental experiences and exposures. Thus, exposure to environmental factors such as air pollution may potentially exert significant and lasting effects on a young person's health, development and life course opportunities during this time (Braithwaite et al., 2019; Reuben et al., 2021). Similarly, early childhood, marked by rapid brain development and skill acquisition, is a crucial phase when exposure to pollutants can disrupt neurodevelopmental processes, potentially influencing later cognitive function, emotional regulation, and mental health (National Scientific Council on the Developing Child, 2023). Understanding the potential interplay between air pollution exposure and these sensitive developmental periods will better inform public health efforts aimed at mitigating the adverse impacts of air pollution on children, to prevent lasting harm and promote healthier developmental outcomes for future generations.

Developing evidence supports a possible biologically plausible aetiological link between air pollution and range of cognitive, educational and mental health outcomes in childhood and adolescence (Park et al., 2021; Lim et al., 2024). Indeed, there is mounting evidence for several tenable mechanisms by which air pollution might affect risk of multiple mental health outcomes and broader cognition (Braithwaite et al., 2019). Emerging evidence suggests air pollution exposure is related to inflammation (Liu et al., 2012), hypothalamic–pituitary–adrenal (HPA) axis dysregulation (Lopez-Duran et al., 2009) and increased stress hormone (cortisol) production (Li et al., 2017). Emerging evidence also suggests that air pollution can also impact directly for example, via translocation of ultrafine pollutant particles across the nasal olfactory nerve or indirectly for instance, via inflammatory signalling from other organ systems, particularly the lungs, harming the central nervous system (CNS) (Reuben et al., 2021). In addition, it is plausible that cumulative exposure to air pollution which starts at conception and continues throughout childhood may be of particular concern (Sunyer, 2008; Myhre et al., 2018). Research suggests that air pollution exposure in early life can result in lasting injury to cells and tissues that increases risk of disease in childhood, with effects persisting across the lifecourse (Barker, 2004).

A growing body of evidence suggests that air pollution exposure, especially during the prenatal period and early childhood may have negative impacts on brain development as well as other markers of childhood and adolescent functioning. This evidence has been further strengthened by recent neuroimaging research (Chandra et al., 2022; Calderón-Garcidueñas et al., 2011, 2012; Guxens et al., 2018). Higher air pollution exposure has been implicated in a greater risk of various deleterious outcomes in childhood and adolescence (Braithwaite et al., 2019; Newbury et al., 2024; McGuinn et al., 2020; Zhao et al., 2018; Oudin et al., 2016) including, but not limited to, attention-deficit/hyperactivity disorder (Rosi et al., 2023; Donzelli et al., 2019; Min and Min, 2017; Newman et al., 2013; Perera et al., 2012), conduct disorder (Roberts et al., 2019), intelligence quotient (IQ) (Harris et al., 2015; Porta et al., 2016), psychomotor development (Guxens et al., 2014), cognitive performance (Kerin et al., 2018), adaptive functioning, behavioural problems (Forns et al., 2016; Yorifuji et al., 2017) and behavioural indices (Chandra et al., 2022). Despite this, the evidence is not unequivocal and inconsistent results remain which limit confidence in drawing inferences from any associations identified. These inconsistencies likely reflect variations in study design, exposure assessment methods and definitions, diverse outcome definitions, as well as a lack of appropriate control for confounding factors (Braithwaite et al., 2019). While most studies support the hypothesis that prenatal and early life exposure to air pollution can have a lasting negative impact on brain development and function further research will be required to confirm any potential association.

Air pollution may also impact on other outcomes, including cognitive development, educational achievement and school performance (Lim et al., 2024). Potential mechanisms are not fully understood but are posited to include neurodevelopmental disruptions (Woodward et al., 2018; Tseng et al., 2019). For instance, while more research is required, air pollution may negatively impact neural connectivity in brain networks involved in language, executive functioning, learning, and creativity (Lim et al., 2024). Research linking early-life exposure to air pollution with an increased risk of other outcomes such as substance use disorders or alcohol use has shown more variability in findings. However, recent evidence has suggested that increases in ambient NO2 and PMs are associated with increased hospital admissions for substance abuse, possibly because of impacts of air quality on depression and other aspects of mental health (Braithwaite et al., 2019; Roberts et al., 2019; Woodward et al., 2018). However, evidence is sparse and not consistent in effect size or direction, highlighting the need for further investigation in this area.

Evidence linking air pollution exposure in childhood or adolescence to mental health outcomes in adolescence is emerging but is less clear than adult studies. In adulthood, meta-analyses are suggestive of an association between long-term (>6 months) particulate matter ($PM)^{2.5}$ exposure and depression, as well as possible associations between long-term $PM^{2.5}$ exposure and anxiety and between short-term (0–2 days) PM (Arseneault et al., 2011) exposure and suicide (Braithwaite et al., 2019). In contrast, the limited quality of existing studies (Perera et al., 2012) and increased potential for bias due to sample selection bias, measurement differences and inadequate control for confounding, serve to limit what can be meaningfully concluded about the links between air pollution exposure and mental health risk in earlier life (Theron et al., 2022). For example, many existing studies have not adequately controlled for pre-existing and/or socioeconomic factors earlier in life. Despite this, contemporary evidence has suggested $PM^{2.5}$ during critical periods of pregnancy and childhood is associated with elevated odds for psychotic experiences, with $PM^{2.5}$ exposure during pregnancy also associated with depression (Newbury et al., 2024). Other research has indicated that air pollution exposure is significantly positively associated with symptoms of depressed mood (Roberts et al., 2019), generalised anxiety disorder (Brunst et al., 2019), psychotic disorders (Newbury et al., 2019) and poorer general mental health (Sui et al., 2018). Notably, a study from the United Kingdom (UK) recently

demonstrated that age-12 pollution exposure was not associated with age-12 mental health problems but age-12 pollution exposure was significantly associated with age-18 depression (Roberts et al., 2019). This is important, as these findings highlight the importance of longer-term follow up periods to fully investigate potential associations between air pollution and youth mental health, and importantly understand not only concurrent effects, but also the potential cumulative or chronic effects of air pollution exposure on mental health outcomes (Roberts et al., 2019). It could be that air pollution exposure takes time to impact on the processes underlying such behavioural problems (Barker, 2004). Considering there is a plausible mechanism this preliminary evidence indicates a need for not only more, but higher quality research, to comprehensively understand the impact of childhood air pollution exposure on adolescent mental health.

This evidence base has several broad and notable limitations. As noted by a recent meta-analysis, most limitations reflect the considerable effort and resources needed to prospectively evaluate the long-term effects of air pollution on human health over time and age, as well as the specialised technology required to measure and estimate the level of exposure to air pollutants over a wide range of geographical areas and time (Braithwaite et al., 2019; Park et al., 2021). First, much previous work has been based on cross-sectional observations, limiting causal inference (Braithwaite et al., 2019). Second, previous work has often investigated associations between air pollution and individual psychiatric disorders, an approach that does not take into account the multidimensional nature of psychiatric problems or the high rate of comorbidity among disorders (Reuben et al., 2021). Third, studies that have repeated measures of historical air pollution seldom combine it with robust birth cohort data which has been collected prospectively, and for those that do have longitudinal data, seldom are individual-level data available across the lifecourse or from birth (Braithwaite et al., 2019). Fourth, other historical factors such as socioeconomic status at birth or parental mental health history are often not included to fully mitigate the risk of confounding (Braithwaite et al., 2019). Such data is essential to begin to more fully interrogate the nature and potential causality of any associations observed. Fifth, few studies can control for exposure at or pre-conception. Sixth, although more evidence using individual-level data is emerging, previous work has often relied on ecological or aggregated, i.e. city or census block level data, making findings prone to the ecological fallacy. These are notable limitations with few studies to date ruling out key threats to causal inference posed by unmeasured confounding (Braithwaite et al., 2019).

Seldom has historical air pollution been linked to repeated measures birth cohort data to examine the longer-term impacts of air pollution exposure on wide range of cognitive, educational and mental health outcomes in childhood and adolescence (Baranyi et al., 2023, 2024). This study uses prospectively collected observational data from the Christchurch Health and Development Study (CHDS), a birth cohort study established in Christchurch, Aotearoa New Zealand (NZ) in 1977 from birth to 18 years of age to test the hypothesis that greater air pollution exposure in childhood is associated with adverse consequences for development and mental health in adolescence. Christchurch, NZ has a well-documented and significant historical air pollution problem in which inequities are evident (Spronken-Smith et al., 2002; Hobbs et al., 2022). Disparities exist in the population exposed to different levels of both ambient and extreme air-pollution episodes (Pearce et al., 2006). Suspended particulate matter, particularly in the form of smoke from domestic heating and industrial processes, has long been a major environmental problem for the city (Sturman, 1982, 1985). Therefore, temporal and spatial variability of smoke concentration and intraurban variability in pollution levels (Kingham and Dorset, 2011) have been extensively and repeatedly assessed (Sturman, 1982, 1985) particularly because of their impact on respiratory disease (Kingham and Dorset, 2011). This unique combination of prospective birth cohort data and annual air pollution data for the prenatal period and the first 10 years of life creates a unique opportunity to better understand the longer-term impacts of air pollution exposure. Finally, to rule out confounding attributable to the selective effects of family socioeconomic and neighbourhood disadvantage, air pollution and outcome associations were adjusted using a series of high-quality measures of family and individual factors and adverse neighbourhood characteristics. We hypothesise that air pollution exposure will be more strongly associated with cognitive outcomes in childhood and adolescence than mental health outcomes.

2. Methods

2.1. Study design

A prospective longitudinal birth cohort study, representative of the Christchurch, NZ population in mid-1977 forms the basis of this work.

2.2. The Christchurch Health and Development Study

Data in this study were collected as part of the Christchurch Health and Development Study (CHDS), a longitudinal study of a cohort of 1265 children born in the Christchurch, NZ urban region during a 4-month period from April to August 1977. This cohort has been studied on 24 occasions from birth to 40 years using a combination of interviews with parents and participants, standardised testing, teacher reports, and medical record data (Fergusson et al., 1989; Fergusson and Horwood, 2001). Ethical approval was obtained for all aspects of data collection and all data collected with informed written consent.

2.3. Participants and sample

Residential address data were sourced from the interview sheets of 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.

2.4. Exposure measures - prenatal and childhood air pollution

We used a combination of data sources and methods to create an estimate of air pollution for the birth cohort in childhood from the prenatal period and then annually until age 10 years. The prenatal period was defined as the period the year before birth. Further details are provided in the online supplementary materials (Supplement 4). Briefly, we used local historical records of the (black) smoke that was commonly measured either in time-restricted campaigns (usually over Southern Hemisphere winters) or continually at a limited number of locations. The historical air pollution data (1971–1994) were obtained from the Canterbury Regional Council (now named Environment Canterbury or ECan) and included monthly (black) smoke estimates (micrograms (one-millionth of a gram) per cubic metre of air or μg/m- (National Institute of Environmental Health Sciences, 2024)) from up to 30 monitoring stations across the wider Christchurch area. Point estimates at monitoring locations were interpolated into both monthly and annual air pollution surfaces in a 100 × 100 m grid using spatiotemporal random forest regression kriging. Air pollution exposure of CHDS cohort members was then approximated by a spatial join of cohort members' residential addresses from prenatal to age 10 years and annual smoke/aggregated area-level estimates. Data from 1977 to 1987 were averaged over 1991 census area units to provide an air pollution metric for the first 10 years of life for CHDS cohort members including the prenatal period (Fig. 1). Census Area Units (CAUs) are geographic units used by Statistics New Zealand for statistical analysis during the national census. CAUs are designed to remain stable over time to facilitate reliable comparisons. However, they may be adjusted between census periods to reflect population changes or urban development.

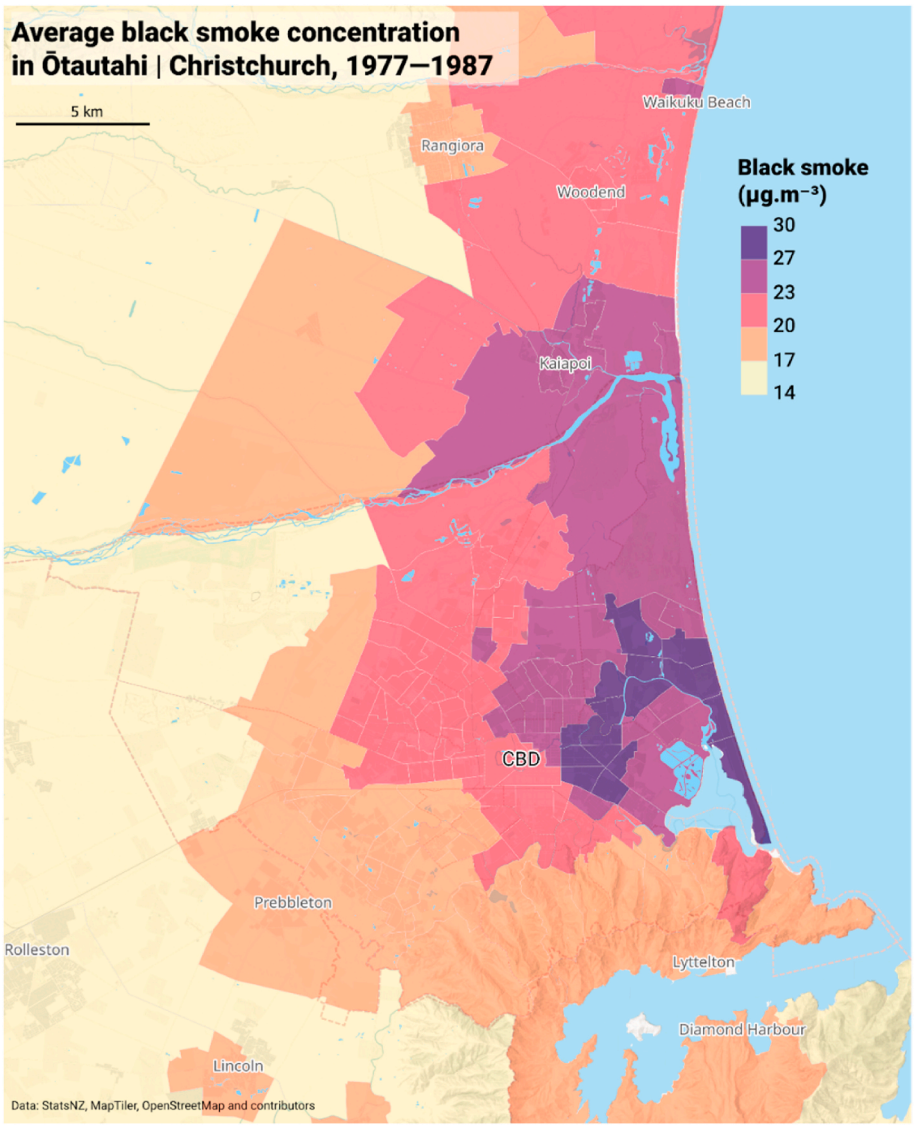

Fig. 1. Average black smoke concentrations in Christchurch, New Zealand from 1977 to 1987.

2.5. Outcomes - adolescent development and mental health

A comprehensive set of adolescent cognitive, educational and mental health outcomes were assessed as part of CHDS. A full description of outcomes selected for inclusion in this study based on previous research and theory, is provided in our online supplementary materials (Supplement 4). First, five cognitive indicators and standardised tests of educational achievement were selected for inclusion: cognitive ability (mean WISC IQ) (8–9 years), reading comprehension (age 10), mathematical reasoning (11 years), teacher rated GPA (11–13 years) and scholastic ability (13 years). Second, there were three education and learning outcomes which included: number of passing grades in School Certificate (15–18 years), Sixth Form Certificate (16–17 years) and university Bursary (17–18 years). There were three outcomes of adolescent disruptive behaviour disorders (14–16 years): attentional problems, conduct problems and oppositional defiant disorder. Finally, the five mental health outcomes (14–16 years) included: depression, anxiety, suicidal ideation, substance problems and alcohol problems). We also included a count measure of mental health outcomes as another variable to account for the multidimensional nature of mental health problems or the high rate of sequential comorbidity among disorders (see Supplement 9).

2.6. Family and individual-level covariates

A range of confounding factors potentially associated with air pollution trajectory assignment were selected from the CHDS database based on previous research and theory linking these to air pollution exposure. These factors spanned the following domains including: (i) *childhood characteristics*: sex at birth (male or female) and ethnicity at birth (Māori ethnicity or other); (ii) *family socio-demographic background*: family socioeconomic status at birth (1 = low; 2 = moderate; 3 = high), family type at birth (0 = two-parent family; 1 = single-parent family), family living standards from birth to age 10, and maternal age at birth (years) and; (iii) *family functioning and parental behaviour*: the number of changes of parental figures living in the home from birth to age 10 years, breastfeeding (number of months breastfed in the first year of life) (count in months), maternal smoking during pregnancy (0 = no; 1 = yes). Further details on the definitions and measurement of these covariates are provided in the online supplementary materials (Supplement 4).

2.7. Disadvantageous aspects of the neighbourhood environment

To capture neighbourhood disadvantage we used a recently

developed historic area-level deprivation metric (Deng et al., 2024) which was available for use to define the socioeconomic status of areas where cohort members resided from birth to age 10 years of age. The metric was constructed for the years 1981, 1986, and 1991 at the Census Area Unit (CAU) level which provided a temporal match with the air pollution data and to the CHDS birth cohort. These measures have been described in detail previously (Deng et al., 2024). We used area-level deprivation at birth to control for the potential impact of neighbourhood disadvantage. Area-level deprivation quintiles were further divided into two categories, with the fifth quintile categorised as the most deprived and the rest as less deprived. In the models we controlled for whether the cohort member lived in the most deprived areas at birth (0 = no; 1 = yes).

2.8. Statistical analysis

Latent Class Growth Mixture models (LCGMMs) were used to identify subgroups of trajectories of air pollution exposure from the prenatal period as well as from birth to age 10 years. The modelling process was conducted using the "lcmm" R package (Proust-Lima et al., 2017). The LCGMMs assume that there are multiple mixed effects models, each representing a subgroup of trajectories that share a common mean and shape with class-specific error variance structure (Herle et al., 2020). We fitted LCGMMs using black smoke as the dependent variable and age as the independent variable, while also incorporating random effects allowing both intercepts and slopes to vary randomly across subjects within each trajectory group. First, a single-class model was fitted to estimate the initial start values for subsequent multiple-class models. Second, two-to seven-class models with three polynomial forms (i.e., linear, quadratic, and cubic) were fitted using an automatic grid search procedure to avoid local maxima. Specifically, it involved a maximum of 1000 iterations using 100 random vectors of initial values from the estimates of the single-class model, yielding output results based on the initial values that yielded the optimal log-likelihood. Models of different numbers and polynomial forms of trajectories were compared using several criteria including Bayesian Information criterion (BIC), sample-adjusted BIC (SBIC), Akaike Information Criterion (AIC), entropy, and the sample proportion in the smallest group. We selected the model with a lower BIC, SBIC, AIC, and higher entropy as well as ensuring the interpretability of trajectories and reasonable sample size in the smallest group (see supplement 10 for further details of the posterior probabilities).

We then examined bivariate associations between trajectory classes and mental health outcomes using one-way analysis of variance (ANOVA) for continuous outcomes or $\chi 2$ for dichotomous outcomes. The strength of associations between childhood air pollution exposure trajectories classes and mental health outcomes were tested using logistic regression models for binary outcomes, linear regression for normally distributed continuous outcomes, and negative binomial regression for over-dispersed count outcomes. Specifically, teacher rated GPA, cognitive ability, and scholastic ability used linear regression, the number of school certificate passing grades, reading comprehension and mathematical reasoning used negative binomial regression. The coefficients are odds ratio for all logistic regression models, Exp(b) for all linear regression models, and incidence-rate ratios for all negative binomial models. The models were constructed with the classes of childhood air pollution exposure trajectories as the independent variable and the mental health outcomes as the dependent variables. A series of childhood covariates (see Section 2.6 and the online supplementary materials Supplement 4 for covariate details) were considered in adjusting the models, based on a) theoretically related to air pollution exposure based on previous literature and b) statistically correlated with air pollution exposure. A backward and forward stepwise selection method was applied in adjusting the models as consistent with previous method typically used in the CHDS (Boden et al., 2019, 2023). Sex and family socioeconomic status at birth were used as fixed variables in all models and not involved in the selection process. For variables for which imputed versions are available, the imputed version was used to avoid losing more samples; these included parental history of illicit substance use, family psychiatric history, parental alcohol problems, and parental criminal offending. We also repeated these analyses with the averaged air pollution exposure across childhood as opposed to the trajectory classes for sensitive analysis and to examine the impact of average air pollution exposure. Statistical analyses were conducted in R Studio and Stata V18.

3. Results

3.1. Classification of air pollution exposure trajectories

A series of Latent Class Growth Mixture Models (LCGMMs) were fitted to the repeated measures of air pollution exposure in childhood to identify cohort members with different air pollution exposure trajectories from conception (i.e. prenatal period) and over the first ten years of life (see the online supplementary materials supplement 1 and 2 for further model fit indices). A four-class quadratic LCGMM was defined as the best fit to the data. Fig. 2 shows the estimated response profile (mean frequency of air pollution exposure for each year) for the four-class quadratic model. For instance, the four trajectories all experienced some degree of decline in air pollution from prenatal to age 10 years. However, there were also several notable differences in the trajectories identified. Class 1 (*"Consistently Low"*, 52.7% of the birth cohort) was a group comprising the lowest relative air pollution exposure throughout childhood which steadily decreased over time. Class 2 (*"Elevated Pre-School""*, 6.2% of the birth cohort) was a group which experienced the highest exposure from around age three to six years and then a rapid decline in air pollution exposure in later childhood. Class 3 (*"Consistently High"*, 34.9% of the birth cohort) was a group which started high and remained high relative to the other groups but did decline over time. Class 4 (*"High Prenatal and Postnatal"*, 6.3% of the birth cohort) had the highest level of exposure to air pollution in the prenatal period and one of the highest in the first one to two years of life, which then declined greatly over time until around age eight when it increased again slightly.

3.2. Examining the association between air pollution exposure trajectories and adolescent development and mental health

3.2.1. Unadjusted analyses

The trajectory analyses were valuable for investigating individual-level changes and predicting future outcomes accurately by more dynamically accounting for heterogeneity within the population in terms of their air pollution exposure over time and age. The prevalence of cognitive, educational and mental health outcomes in childhood and adolescence by childhood air pollution trajectory are fully described in the online supplementary materials (Supplement 3). Fig. 3 visualises the unadjusted and adjusted associations between the air pollution trajectory classes and each outcome of interest (full model specifications are shown in the online supplementary materials Supplement 5). All odds ratios and beta coefficients were calculated with Class 1 as the reference category. Logistic regression models were used for depression, anxiety, suicidal ideation, alcohol problems, substance problems, attentional problems, conduct problems, oppositional defiant disorder, sixth form certificate, and university bursary (Odds Ratio [95% Confidence Interval (CI)]), negative binomial regression models were used for number of school certificate passing grades, reading comprehension and mathematical reasoning (Incidence Rate Ratio [95% CI]) and linear regression models were used for teacher rated GPA, cognitive ability and scholastic ability (exponentiation of the beta coefficient [95% CI]).

3.2.1.1. Cognitive indicators and standardised tests of academic achievement. In the unadjusted analyses, relative to Class 1 (*Consistently*

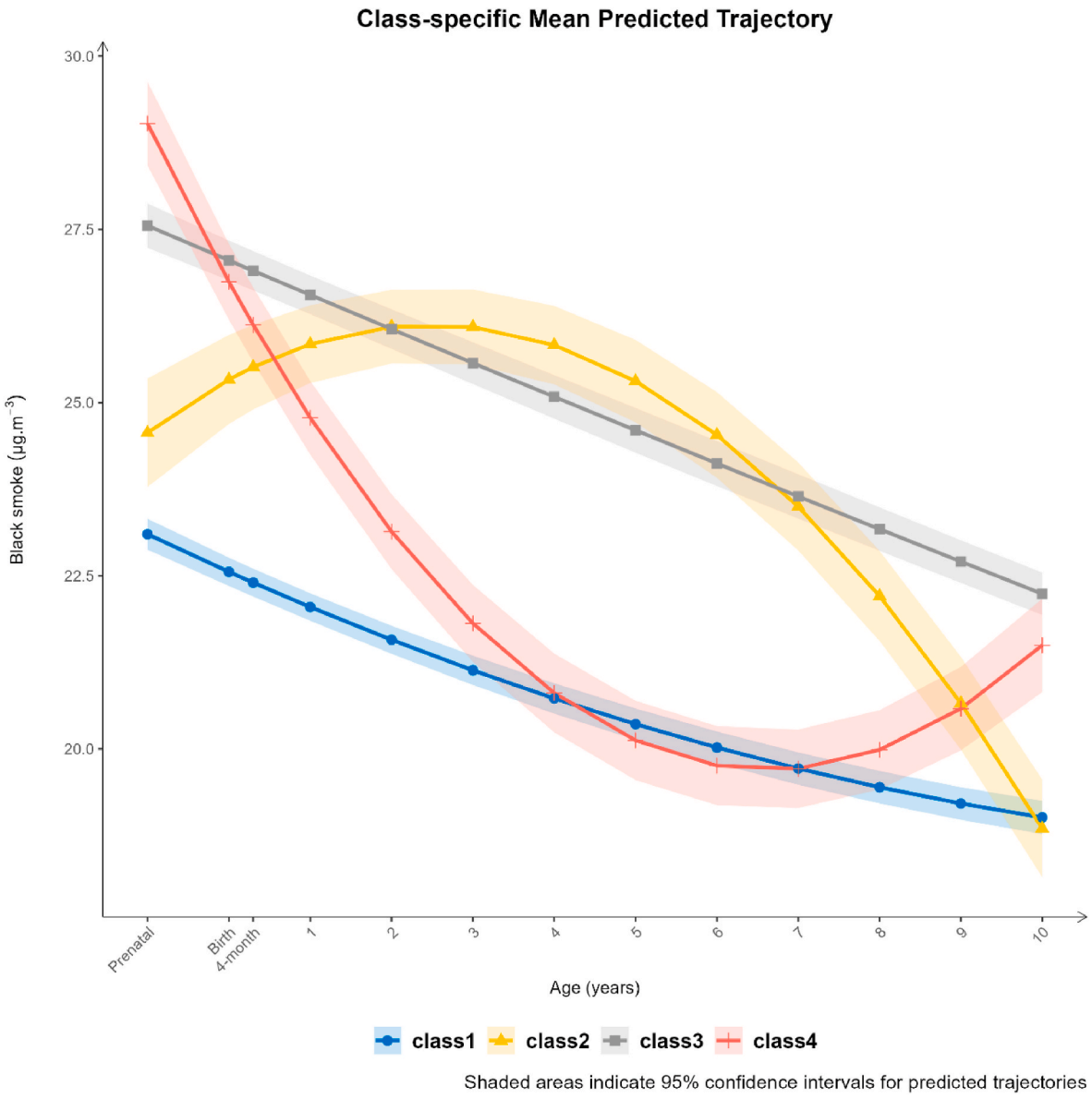

Fig. 2. Mean air pollution (black smoke) trajectories from prenatal and birth to 10 years of age by air pollution class trajectory (Note – Class 1: *Consistently Low*; Class 2*: Elevated Pre-School*; Class 3: *Consistently High*; Class 4: *High Prenatal and Postnatal*).

Low), there were no statistically significant associations for Class 2 (*Elevated Pre-School)*. Class 3 (*Persistently High)* was related to lower teacher rated GPA 7–13 years (Exp(b) = 0.86[0.78–0.96]), lower cognitive ability (Exp(b) = 0.06[0.01–0.33]), reading comprehension (IRR = 0.89[0.81–0.99)], mathematical reasoning (IRR = 0.95 [0.91–0.99]) and scholastic ability (Exp(b) = 0.02[0.00–0.20]). Class 4 (*High Prenatal and Postnatal)* was related to lower teacher rated average GPA 7–13 years (Exp(b) = 0.77[0.62–0.95]), cognitive ability (Exp(b) = 0.01[0.00–0.26]) and mathematical reasoning (IRR = 0.89[0.80–0.98]).

3.2.1.2. Educational and training. In the unadjusted analyses, relative to Class 1 (*Consistently Low*), Class 2 (*Elevated Pre-School)* was related to lower school certificate passing grades (IRR = 0.65[0.49–0.85]) and sixth form certificate (OR = 0.27[0.15–0.46]). Class 3 (*Consistently High)* was related to lower school certificate passing grades (IRR = 0.83 [0.75–0.92], lower odds of sixth form certificate (OR = 0.58 [0.43–0.78]) and university bursary (OR = 0.57[0.42–0.77]). Class 4 (*High Prenatal and Postnatal*) was related to lower school certificate passing grades (IRR = 0.76[0.61–0.95]), sixth form certificate (OR = 0.43[0.25–0.74]) and university bursary (OR = 0.35[0.17–0.72]).

3.2.1.3. Adolescent behaviour problems (14–16 years). In the unadjusted analyses, relative to Class 1 (*Consistently Low*), the Class 2 (*Elevated Pre-School*) trajectory group had higher odds of attentional problems (OR = 3.63[1.45–9.11]), conduct problems (OR = 3.52[1.91–6.50]) and Oppositional Defiant Disorder (OR = 3.30[1.78–6.13]). Class 3 (*Consistently High)* had higher odds of attentional problems (OR = 2.65 [1.45–4.87]), conduct problems (OR = 1.86 [1.27–2.73]) and Oppositional Defiant Disorder (OR = 1.49[1.00–2.23]). Class 4 (*High Prenatal and Postnatal*) had higher odds of attentional problems (OR = 4.32 [1.78–10.45]) and conduct problems (OR = 2.14[1.09–4.20]).

3.2.1.4. Mental health (14–16 years). In the unadjusted analyses, relative to Class 1 (*Consistently Low*), Class 2 (*Elevated Pre-School)* had a higher risk of depression (OR = 2.08[1.04–4.17]), anxiety (OR = 2.02 [1.11–3.69]), suicidal ideation (OR = 2.34[1.23–4.46]), alcohol problems (OR = 2.57[1.20–5.50]) and substance problems (OR = 4.14 [1.39–12.38]). Class 3 (*Consistently High*) was related to increased odds

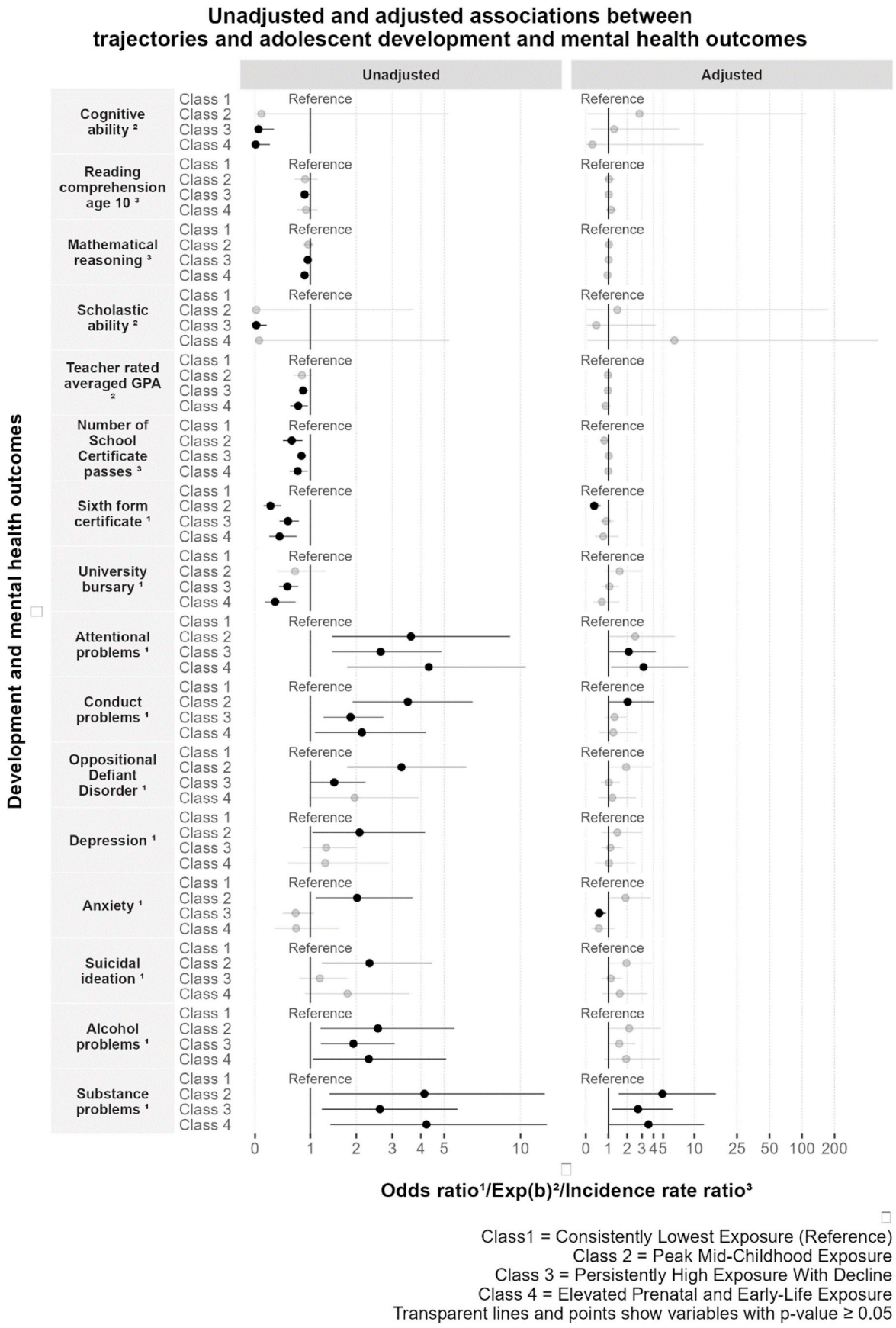

Fig. 3. Examining the association between air pollution trajectories classification and development and mental health in adolescence (please note that the x-axis is on a logarithmic scale).

of alcohol problems (OR = 1.93[1.21–3.07]) and substance problems (OR = 2.63[1.23–5.66]). Finally, Class 4 (*High Prenatal and Postnatal*) was related to an increased odds of alcohol problems (OR = 2.32 [1.05–5.09]) and substance problems (OR = 4.22[1.41–12.63]).

3.2.2. Fully adjusted analyses

As shown in Fig. 3, when models were adjusted for a comprehensive range of family and sociodemographic background factors, many of the associations seen in the unadjusted analyses were attenuated to null although a few remained. For Class 2 (*Elevated Pre-School*), there was an increased odds of conduct problems (OR = 2.03[1.02–4.06]) and lower odds of obtaining sixth form certificate (OR = 0.36[0.20–0.64]). Class 3 (*Consistently High*) also had higher odds of attentional problems (OR = 2.09[1.04–4.21]). Finally, Class 4 (*High Prenatal and Postnatal*) had higher odds of attentional problems (OR = 3.13[1.13–8.67]).

In terms of mental health outcomes, Class 2 (*Elevated Pre-School*) had close to a higher odds of suicidal ideation (OR = 1.96[1.00–3.80]) and substance abuse problems (OR = 4.92[1.52–15.95]), Class 3 (*Persistently High)* had a lower odds of anxiety (OR = 0.58[0.38–0.89]) and had higher odds of substance abuse problems (OR = 2.71[1.19–6.20]) and Class 4 (*High Prenatal and Postnatal*) also had a higher odds of substance abuse problems (OR = 3.54[1.02–12.31]). It should be noted that in the adjusted models, all statistically significant estimates had wide CIs around the coefficient or odds ratio. Full model specifications are shown in the online supplementary materials (Supplement 5). In line with our study aims, we also examined the impact of air pollution on development and mental health outcomes by age group (Supplement 6 and 7). However, due to a lack of statistical power there were few associations between exposure to average air pollution by age again highlighting the usefulness of trajectory analyses to capture inherent variability in exposure to air pollution over time.

4. Discussion

This study utilised prospectively collected birth cohort data and annual air pollution exposure data over the prenatal period and up to child age 10 years, to examine associations between air pollution exposure and a wide range of cognitive, educational and mental health outcomes in childhood and adolescence. It is well-recognised that the developing brain is much more vulnerable than the mature nervous system (Clifford et al., 2016) and our findings that after adjustment for covariates, persistently high and elevated prenatal and postnatal air pollution exposure were associated with attentional problems. Elevated pre-school exposure was linked to conduct problems, lower educational attainment and substance abuse. Persistently high exposure throughout childhood increased the odds of substance abuse. While it is important to be critical of the small yet statistically significant effect sizes in this study, the relatively consistent patterns observed, along with the direction of associations and the relatively modest prospective sample indicate that these findings should not be dismissed as inconsequential especially when considered alongside contemporary neuroimaging evidence (Chandra et al., 2022; Calderón-Garcidueñas et al., 2011, 2012; Guxens et al., 2018). It is also important to interpret the small effects in our study in the light of individual-versus population-level effects (Rose, 1985). For instance, 10-year exposure to air pollution may explain little of an individual's risk of mental health outcomes developing however, it may help to explain the shift in population-level outcomes which have gradually shifted upwards with the likelihood of increased burden of disease over time (Rose, 1985). This is one of many factors impacting on mental health, but also one we might expect in some regions of the world to worsen with climate change (Kumar et al., 2023).

Our findings support an emerging body of scientific study which tend to confirm the importance of air pollution exposure, especially during prenatal and postnatal periods and early childhood for later development. A recent systematic review concluded that air pollution exposure was potentially linked to disruptive, impulse control and conduct disorders among pollution-exposed adolescents (Theron et al., 2021, 2022; Clifford et al., 2016; Odo et al., 2023; Loftus et al., 2020). However, it is important to note that few of the included studies excluded the possibility of historical confounders available in our study. An exception being a prospective study demonstrating an association between prenatal and postnatal exposure to air pollution and higher risk of externalising behaviour problems in children (Reuben et al., 2021; Loftus et al., 2020). Less evidence has considered broader developmental outcomes such as educational attainment (Lim et al., 2024) or conduct disorder, despite these mental health issues often developing or worsening during adolescence (Theron et al., 2021, 2022). Supporting some of the findings in this study, 16 years of exposure data in a nationwide Danish cohort of adolescents showed that an increase of 5 $\mu g/m^3$ in $PM_{2.5}$ was linked to a one-point decrease in GPA (Lim et al., 2024). Our findings, considered alongside previous evidence, indicate that in children, poorer performance may be seen across a broad spectrum of developmental outcomes in those children exposed to higher levels of air pollution.

Evidence on the association between air pollution exposure and mental health is equivocal, with a dearth of evidence in childhood and adolescence. There is a distinct lack of consideration of how air pollution exposure relates to broader mental health difficulties that classically challenge adolescents such as substance use disorders or even broader mental health conditions such as anxiety (Theron et al., 2022). Despite this, most available evidence is at a high risk of bias thus limiting what can meaningfully be said about the links between air pollution exposure and mental health outcomes in childhood or adolescence (Theron et al., 2022; Ventriglio et al., 2020). Despite this, our findings showed that several trajectories were adversely related to risk of suicidal ideation and substance abuse albeit with relatively wide effect size confidence intervals. In our study while several associations were noted in unadjusted models, after adjustment, many of these were attenuated highlighting the importance of controlling for a wide set of family background and sociodemographic factors (Braithwaite et al., 2019; Park et al., 2021). A recent review suggested that air pollution exposure increased the risks of depressive symptoms (in 10 out of the 14 included papers) and suicide in the adolescents (in 4 out of 5 included papers) (Xie et al., 2023). Further robust epidemiological evidence is required to support the associations seen within this study relating air pollution trajectories to mental health in adolescence and to explore if the null associations seen for depressive symptoms and anxiety are confirmed.

Several scientific studies have demonstrated support for biological plausibility and mechanistic link in associations seen with plausible toxicological mechanisms also demonstrated (Clifford et al., 2016). For instance, neuroimaging studies have found air pollution-related brain structural and functional alterations with several neuroimaging studies revealing decreased grey-matter volume in the Cortico-Striato-Thalamo-Cortical neurocircuitry and others detecting white matter hyperintensities in the prefrontal lobe (Xie et al., 2023). In addition, a recent animal study indicated that after 10 months of exposure to air pollution at levels similar to those faced by many people daily, mice showed signs of depression, anxiety, and learning difficulties (Fonken et al., 2011). Theoretically, this also fits with a well-established body of literature which documents the importance of prenatal and early childhood development and it may be that exposure to air pollution within these sensitive windows of development may be important for potential disruptions in neurodevelopmental pathways (Black et al., 2017; Barker, 2004). Some studies have suggested that poorer cognitive function at 5–6 years among males followed increased maternal exposure to PM_{10}, $PM_{2.5}$ and NO_2 during mid-pregnancy and child exposure to $PM_{2.5}$ around 3–4 years. While more research is needed on any critical window or timing of exposure, evidence has posited that in utero exposure may be important for intelligence and neurodevelopment around ages 3–5 years (Clifford et al., 2016) whilst other evidence has suggested that the most time critical windows were in utero and infancy for PM_{10} exposure (Shi et al., 2024). Our findings support both an

'accumulation hypothesis' where persistently high exposure may be related to adverse outcomes and *'critical window hypothesis'* where time periods such as prenatal may be especially important. Further research with sufficient statistical power and repeated measures of air pollution over time is required to further explore this timing effect.

This study is unable to explain the differences in associations across exposure trajectories; however, they may reflect diverse neurodevelopmental sensitivities at different ages. The prenatal and postnatal periods are critical for brain development, which could explain why high prenatal and postnatal exposure was linked to slightly different outcomes to later childhood exposure which may have more influence on cognitive or emotional regulation, potentially contributing to conduct problems and lower educational attainment. However, further research is needed, especially as findings for the persistently high class do not consistently align with the hypothesis that chronic higher exposure would have the strongest effects. This is possibly due to unmeasured confounding. Further supporting this uncertainty, the lower odds of anxiety for the persistently high class may reflect residual confounding or selection effects. Given the wide confidence intervals and the complex interplay of individual and environmental factors, these associations should be interpreted with caution.

Our findings, if replicated, may have important implications for policy. First, even small elevations in risk may result in significant increases in the burden of disease at the population-level. Indeed, a recent World Health Organisation (WHO) estimate suggests that around 9 out of 10 of the global population is exposed to outdoor air pollutants in excess of current guidelines (World Health Organisation, 2016; World Health Organization (WHO), 2006). Given the ubiquity of air pollution exposure, as well as spatial and temporal variations resulting in high levels of air pollution even in countries with low average levels of air pollution, air pollution could represent a meaningful contributor to the global burden of mental illness and poorer child and adolescent development particularly in poor air-quality regions (Reuben et al., 2021). Associations with less susceptibility of residual confounding and selection bias in this study for air pollution trajectories further raise the possibility of the impact of sensitive periods of exposure to air pollution for children including the prenatal period of life and during early- or mid-childhood in a period of rapid growth, maturation and development. Further research is required to confirm our findings using different contemporary measures of air pollution. Moreover, we acknowledge that some of our outcomes are likely to be correlated and we have not explored the pathways by which such factors may be associated. Therefore, future research should explore the extent to which attentional and behavioural problems and/or cognitive ability may play a role in explaining the association between early life air pollution exposure and later poorer educational achievement as well as other plausible pathways not included in this study.

The findings in this study must be interpreted with several strengths and limitations in mind. First, the trajectory analyses enabled an examination of heterogenous patterns of exposure that would otherwise have been missed when using averaging methods over time which obscure dynamic patterns and only provide a static snapshot of the air pollution exposure (Campbell et al., 2021). Capturing the trajectory of exposure is important and reveals patterns and trends that may not be apparent when looking at averages of exposure over time (Gadd et al., 2019). In addition, examining trajectories helps identify critical periods of transition or stability within the air pollution exposure. However, while prospective in design, pollutant exposure estimates relied on modelled data rather than individual-level monitoring. The use of black smoke data from the 1970s–1980s may not fully represent contemporary PM exposure. Additionally, unaccounted factors such as traffic-related noise could confound results (Braithwaite et al., 2019; Park et al., 2021; Clark et al., 2020). Generalisability to regions with different pollution levels and types of particulate matter is uncertain, and findings may not fully translate to contemporary society. However, strengths include prenatal and preschool period measurement, multiple exposure and outcome measures, and robust covariate adjustment often missing in much contemporary literature. Yet we were unable to account for every residential move during the prenatal period relying only on one address in the year before birth. In addition, the observational nature and potential unmeasured confounders imply caution in interpreting any causality. Small sample sizes in some trajectories may also limit result confidence, often reflected in large confidence intervals around any statistically significant effects. Related to this, while a multilevel model is recognised as best practice, there were insufficient observations per area to complete such analyses in this study. Moreover, sample attrition impacts the CHDS study somewhat with the analysis sample to likely underrepresent children from socially disadvantaged families characterised by low maternal education, low socioeconomic status and single parenthood. Finally, residual confounding from green space and wider socioeconomic factors remains possible. Greenspace for instance, may buffer air pollution effects via stress reduction and physical activity, while unmeasured socioeconomic factors may introduce additional social and environmental stressors. Although we adjusted for wide-ranging factors future research should integrate these data better to disentangle these influences.

5. Conclusion

There are many factors that contribute to child and adolescent development and mental health. Our study addressed the lack of prospective evidence linking air pollution exposure in childhood to cognitive, educational and mental health outcomes in childhood and adolescence. Responding to calls for higher quality, prospectively collected data, we found statistically significant associations between childhood air pollution exposure trajectories and adverse outcomes in adolescence. Specifically, high prenatal and postnatal exposure, persistently elevated exposure, and elevated pre-school exposure were linked to several adverse developmental and mental health outcomes. The effect sizes and wide confidence intervals suggest that future research should confirm our findings and explore sensitive childhood periods in more detail. However, these findings, alongside other contemporary evidence of air pollution's harmful effects, underscore the importance of targeted interventions and policies to reduce exposure to air pollution, especially for children.

CRediT authorship contribution statement

Matthew Hobbs: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Bingyu Deng:** Writing – review & editing, Writing – original draft, Visualization, Software, Formal analysis, Data curation. **Lianne Woodward:** Writing – review & editing, Writing – original draft, Supervision, Methodology, Investigation, Funding acquisition, Conceptualization. **Lukas Marek:** Writing – review & editing, Visualization, Formal analysis, Data curation, Conceptualization. **Geri McLeod:** Writing – review & editing, Software, Resources, Project administration, Methodology, Investigation, Data curation, Conceptualization. **Andy Sturman:** Writing – review & editing, Validation, Supervision, Methodology, Conceptualization. **Simon Kingham:** Writing – review & editing, Supervision, Investigation, Conceptualization. **Annabel Ahuriri-Driscoll:** Writing – review & editing, Supervision, Methodology, Investigation, Funding acquisition, Conceptualization. **Phoebe Eggleton:** Writing – review & editing, Investigation, Data curation. **Malcolm Campbell:** Writing – review & editing, Methodology, Investigation, Conceptualization. **Joseph Boden:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Ethical approval

All phases of the CHDS were subject to ethical approval by the New Zealand Health and Disabilities Ethics Committee.

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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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 ASupplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envres.2025.121148.

Data availability

Data will be made available on request.

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