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Neighborhood environmental exposures and incidence of attention deficit/hyperactivity disorder: A population-based cohort study

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ABSTRACT

Background: Emerging studies have associated low greenspace and high air pollution exposure with risk of child attention deficit/hyperactivity disorder (ADHD). Population-based studies are limited, however, and joint effects are rarely evaluated. We investigated associations of ADHD incidence with greenspace, air pollution, and noise in a population-based birth cohort.

Methods: We assembled a cohort from administrative data of births from 2000 to 2001 (N ~ 37,000) in Metro Vancouver, Canada. ADHD was identified by hospital records, physician visits, and prescriptions. Cox proportional hazards models were applied to assess associations between environmental exposures and ADHD incidence adjusting for available covariates. Greenspace was estimated using vegetation percentage derived from linear spectral unmixing of Landsat imagery. Fine particulate matter ($PM_{2.5}$) and nitrogen dioxide (NO_2) were estimated using land use regression models; noise was estimated using a deterministic model. Exposure period was from birth until the age of three. Joint effects of greenspace and $PM_{2.5}$ were analysed in two-exposure models and by categorizing values into quintiles.

Results: During seven-year follow-up, 1217 ADHD cases were diagnosed. Greenspace was associated with lower incidence of ADHD (hazard ratio, HR: 0.90 [0.81–0.99] per interquartile range increment), while $PM_{2.5}$ was associated with increased incidence (HR: 1.11 [1.06–1.17] per interquartile range increment). NO_2 (HR: 1.01 [0.96, 1.07]) and noise (HR: 1.00 [0.95, 1.05]) were not associated with ADHD. There was a 50% decrease in the HR for ADHD in locations with the lowest $PM_{2.5}$ and highest greenspace exposure, compared to a 62% increase in HR in locations with the highest $PM_{2.5}$ and lowest greenspace exposure. Effects of $PM_{2.5}$ were attenuated by greenspace in two-exposure models.

Conclusions: We found evidence suggesting environmental inequalities where children living in greener neighborhoods with low air pollution had substantially lower risk of ADHD compared to those with higher air pollution and lower greenspace exposure.

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1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) is characterized by neurobehavioral symptoms, such as difficulties with attention, impulsive behaviors, and hyperactivity (Sagvolden et al. 2005). Worldwide, ADHD is among the most prevalent neurodevelopmental disorders affecting up to approximately 5–10% of children and adolescents (Brikell et al. 2020; Polanczyk et al. 2014; Visser et al. 2010). Over the life course, ADHD is associated with impairments in social functioning, academic performance, educational attainment, work performance, and occupational stability (Carpenter Rich et al. 2009; Daley and Birchwood 2010; Loo et al. 2009; Pelham et al. 2007), with considerable impacts on individual wellbeing, health care, and the economy (Brook et al. 2013; Fletcher and Wolfe 2009; Hultman et al. 2007; Mick et al. 2002; Pelham et al. 2007; Willoughby 2003).Fig. 1

The full aetiology and pathophysiology of ADHD are unclear. Previous research suggested a genetic component as well as environmental risk factors, such as air pollution, and noise (Brockmeyer and d'Angiulli 2016; Froehlich et al. 2011; Oudin et al. 2019).

Air pollution may potentially be associated with child ADHD through induced systemic oxidative stress, which contributes to neuronal injury and disturbs brain development, leading to cognitive deficits (Genro et al. 2010; Grandjean and Landrigan 2006; Hagberg and Mallard 2005; Myhre et al. 2018). However, results from research on the impact of air pollution on ADHD are inconsistent (Fordyce et al. 2018). A number of epidemiological studies have shown associations between exposure to air pollution from road traffic or other sources and ADHD in children (Aghaei et al. 2019; Becerra et al. 2013; Dix-Cooper et al. 2012; Myhre et al. 2018; Vrijheid et al. 2012), but a few studies reported no associations (Gong et al. 2014; Oudin et al. 2019).

Noise exposure may increase stress, which in turn is associated with externalizing psychological disorders such as hyperactivity (Doan et al. 2012; Haines et al. 2001; Lim et al. 2018; Weyde et al. 2017). Recent reviews suggested, however, that evidence supporting a harmful effect of noise on children's mental health remain limited (Clark et al. 2020; Sakhvidi et al. 2018; Tzivian et al. 2015) and the underlying mechanisms are unclear.

Contrary to air pollution and noise, USGS. Landsat 8 annual greenest-pixel toa reflectance composite, 2013 exposure to greenness, such as urban parks, street trees, and domestic gardens, has been associated with lower risk of ADHD (Donovan et al. 2019; Markevych et al. 2018; Thygesen et al. 2020b). Various pathways linking greenness and ADHD have been suggested, such as mental restoration (Faber Taylor and Kuo 2009, 2011) and regulating ecosystem services, including reduction of heat (Pérez-Crespo et al. 2020), air pollution, and noise (Markevych et al. 2018; Zhao et al. 2021b).

Previous epidemiological studies linking environmental risk factors with ADHD vary greatly in terms of study designs (to a large extent, relying on cross-sectional designs), sample sizes, population representativeness, and definitions of ADHD. In addition, the potential spatial correlation between various exposures, such as air pollution, noise and greenness, have not always been considered. For example, green areas, such as parks, usually have fewer emission sources, resulting in lower levels of air pollution and noise (Bloemsma et al. 2019; Dzhambov and Dimitrova 2015; Markevych et al. 2018). Although empirical evidence is mixed, green areas with more trees and plants may also have a capacity to capture and/or filter air pollution (Beckett et al. 2000; Chen et al. 2017; Diener and Mudu 2021; Dzierżanowski and Gawroński 2011). The restorative impact of greenness may potentially also be stronger in areas with lower air pollution and noise levels than in areas with higher air pollution and noise levels. Finally, the magnitude of the impact of greenness on the risk of ADHD may differ depending on the level of air pollution or noise and vice versa.

In this study, we investigated associations between multiple environmental exposures (greenness, air pollution, and noise) in early life and later ADHD incidence, using a population-based birth cohort linked with administrative data, in combination with environmental exposure metrics.

We hypothesized that greenness is associated with lower risk of ADHD and vice versa for harmful exposures (air pollution and noise). We also hypothesised that there would be joint effects of the respective environmental exposures due to spatial correlation. Finally, we hypothesised that there could be a sex-dependent difference in the association because of the higher prevalence of ADHD among boys than girls (Mowlem et al. 2019; Pérez-Crespo et al. 2020; Sayal et al. 2018).

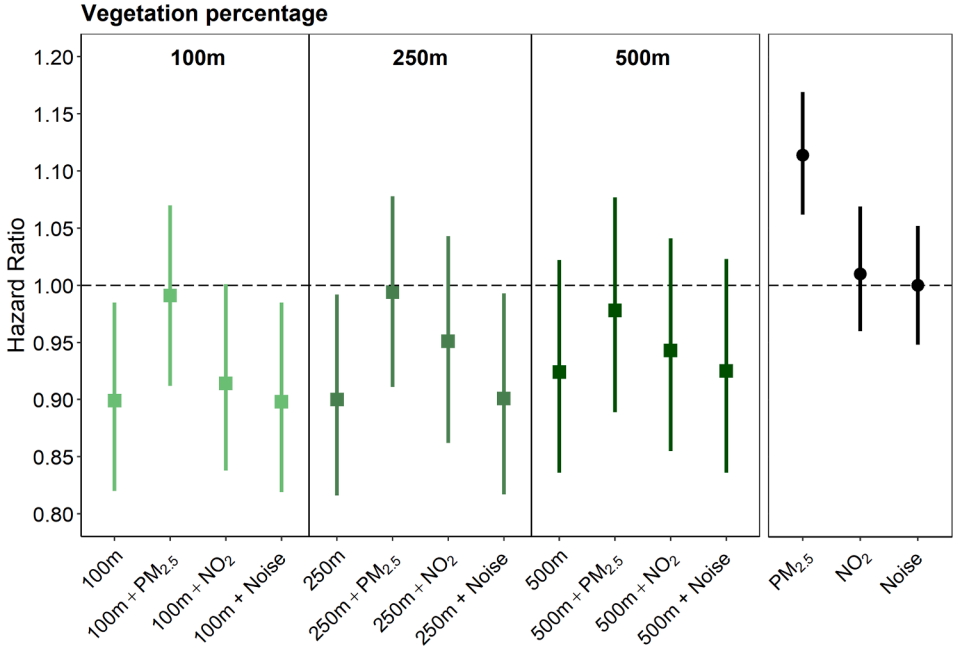

Fig. 1. Hazard ratios (95% confidence interval) associated with greenness (100-, 250- and 500-meter buffer size) from unmixed pixel dataset, air pollution and noise (per Interquartile range as indicated in Table 3) for Attention deficit hyperactivity disorder (ADHD). PM 2.5 = fine particulate matter, NO 2 = Nitrogen dioxide. Covariates included for ADHD: Maternal age, paternal age, maternal body mass index, maternal parity, birth weight, season of birth, gestational age, infant sex, education, household income, residential instability, material deprivation, dependency and ethnic concentration.

2. Methods

2.1. Study area and cohort

Our analysis was conducted in the urban area of Metro Vancouver, located in the southwest mainland of the province of British Columbia (BC) in western Canada. The region has a population of approximately 2.5 million and covers in total around 2,000 square kilometers (Statistics Canada 2017). The land use is characterised by densely built-up areas in the west and agricultural and suburban lands to the east. The study cohort comprised all 2000 and 2001 births identified by linking administrative datasets (Registration, Physician Visit, and Hospital Discharge data) from the BC Ministry of Health Services (British Columbia Ministry of Health 2011a, 2011b; Canadian Institute for Health Information 2011), vital statistics data from the BC Vital Statistics Agency (British Columbia Vital Statistics Agency 2011a, 2011b) and perinatal data from the Perinatal Services BC (Perinatal Services BCE 2011).

Our study focused on impact of postnatal exposures and the exposure period was from birth (2000–2001) until the age of three, with follow-up from age three until 2010. The exposure- and follow-up period was selected because children can be diagnosed with ADHD as young as age of three (Boyle et al. 2011; Visser et al. 2014; Xu et al. 2018) and data in this study were only available up to 2010. To be eligible, children and their mothers had to be registered for the provincial universal Medical Services Plan (MSP) plan and have lived in the study area for the duration of 2000 to 2010. The MSP is a mandatory health insurance program in the province of BC, covering nearly all residents (Chamberlayne et al. 1998Spatial, 2015). Still births were excluded from the cohort and only singletons were included. We only considered singleton births because multiple births pregnancies present higher rates of complications for fetal growth abnormalities compared with singleton births. Fetal growth abnormalities increase the prevalence of long-term neurodevelopmental outcomes (Oepkes and Sueters 2017; Santana et al. 2016). Exposures to greenness, air pollution, and noise were estimated at each individual's residential six-digit postal code, using vegetation percentage from satellite data (Czekajlo et al. 2020) as a measure of greenness, land-use regression models (Brauer et al. 2008; Henderson et al. 2007; Larson et al. 2009) and a deterministic noise model (Gan 2012), respectively. In the urban study area, a six-digit postal code typically refers to one side of a block or a single multi-unit building with a population of approximately 35 individuals (Khan et al. 2018). All linkages between exposures, outcome, and individual and area-level covariate data were on the six-digit postal code level and averaged (from annual values) over the exposure period while accounting for changes in addresses of participants who moved during this period. The data linkages were approved by the Behavioural Research Ethics Board of The University of British Columbia (#H18-00908).

2.2. Exposure assessment

2.2.1. Greenness

We used freely available satellite imagery to detect and map greenness across the region and applied a newly developed method for calculating greenness percentage in the area (Czekajlo et al. 2020). The same metric was used in a recently published study assessing the impact of greenness on childhood early development (Jarvis et al. 2021). In brief, the original dataset was a Landsat satellite-based time series dataset of three spectrally unmixed fractions or endmembers representing vegetation (e.g., grass), darkness (e.g., shadows of tall buildings) and high albedo (e.g., impervious surfaces). These three fractions datasets were temporally smoothed using LOESS (locally weighted smoothing) across the entire time series (i.e. years 1984–2016). This was conducted to reduce inter-annual noise (like exceptionally dry years causing a dip in vegetation greenness) (White et al. 2014). A sub-pixel linear spectral unmixing algorithm, using the three selected endmembers, was applied to every year and an annual greenness fraction of each pixel in the study area was extracted. Values for each of the endmembers ranged from 0 to 100, indicating the percentage of the pixel composed of each endmember. Based on this process, the proportion of vegetation was calculated per pixel and represents the area-based greenness per each year of our study period (see (Czekajlo et al. 2020Gan et al., 2011) for more information). From this layer, exposure to greenness was assessed as the average vegetation percentage within 250-meter radius (Euclidian distance) buffer zones around participants' residential postal codes (and 100- and 500-meter buffer zones in sensitivity analyses) (CanMap 2015), using the 'sp' and 'raster' packages (Hijmans and van Etten 2016; Pebesma and Bivand 2018) in R (version: 4.0.1) (R Core Team 2018). Buffer zones around 250-meters to 300-meters have typically been used in previous greenness exposure studies and are assumed to correspond to daily residential exposure within a short walking distance (Annerstedt van den Bosch, et al, 2016; Coombes et al. 2010; Ekkel and de Vries 2017; Jones et al. 2009; Lwin and Murayama 2011; Toftager et al. 2011). In sensitivity analysis, satellite derived normalized difference vegetation index (NDVI) from Landsat (spatial resolution 30 m) and Planet Scope (spatial resolution 3 m) (CanMap 2015; Gorelick et al. 2017; USGS; USGS; USGS; USGS) were used for the corresponding buffer zones. Water features were masked before exporting the band data that were used to calculate NDVI values (Gorelick et al. 2017; USGS). values. Annual mean NDVI values (2000–2004) from Landsat were used to calculate average greenness values over the exposure period.

2.2.2. Air pollution

National land use regression (LUR) models provided by CANUE (Canadian Urban Environmental Health Research Consortium, canue.ca) were applied to estimate exposures to fine particulate matter ($PM_{2.5}$) and nitrogen dioxide (NO_2), respectively (Boys et al. 2014; DMTI Spatial Inc. 2015; Hystad et al. 2011; Van Donkelaar et al. 2015; Weichenthal et al. 2017). Ground-level $PM_{2.5}$ over North America was estimated by combining a 0.01 × 0.01-degree (about 1 km) resolution optimal estimate-based Aerosol Optical Depth (AOD) with aerosol vertical profile and scattering properties stimulated by chemical transport models. Residual bias in the satellite-derived $PM_{2.5}$ estimates was adjusted using geographically weighted regression (GWR) with ground measurements. The LUR model ($R^2 = 0.78$) estimated $PM_{2.5}$ between 2000 and 2012 (Boys et al. 2014; Van Donkelaar et al. 2015). The national NO_2 land use regression model was initially developed from 2006 national air pollution surveillance (NAPS) monitoring data. The final model ($R^2 = 0.73$) included road length within 10 km, 2005–2011 satellite NO_2 estimates, area of industrial land use within 2 km, and summer rainfall. Local scale variation was modeled using a deterministic gradient and kernel density measures, which were added to the final model to produce NO_2 estimates (DMTI Spatial Inc. 2015; Hystad et al. 2011; Weichenthal et al. 2017).

2.2.3. Noise

A deterministic noise modelling software (CadnaA from DataKustik, Greifenberg, Germany) was applied to calculate annual average community noise levels at residential address. Details of the model are described elsewhere (Gan et al. 2012). In brief, noise exposures (annual day-evening-night A-weighted equivalent continuous noise level, L_{den} dB(A)) were calculated from descriptors such as street and rail layout, topography, traffic volume, road type, train frequency, building heights and footprints, and separate airport noise forecasts. On a 10 m × 10 m grid, annual weighted noise exposures were generated and averaged for each six-digit postal code (Gan et al. 2012Domínguez-Berjón et al., 2006). The L_{den} metric weights evening (5-dB(A) weighting) and night (10-dB(A) weighting) noise relative to daytime noise to account for increased noise sensitivity of residents to noise in the evening and night (Clark et al. 2017).

2.3. Covariates

Covariates that may potentially confound the relationships between the environmental exposures and ADHD were selected a priori from those available. The selection was based on findings from previous literature (Arnett et al. 2015; Hu et al. 2020; Lahti et al. 2006; Mick et al. 2002). Information on individual socio-economic status (SES) was not available in this study, but data on maternal age, paternal age, maternal body mass index (BMI), and maternal parity were included as proxies of socio-economic status, which is associated with exposures and ADHD (Emanuel et al. 2004; McDonald et al. 2009; Moore et al. 2009). Maternal BMI was classified into four categories: underweight (<18.5), normal (18.5–25), overweight (25–30), and obese (>30). Maternal parity consisted of four groups: 0-times, 1 time, 2 times and>3 times (Andersen et al. 2018; Chang et al. 2014; Chen et al. 2014; Heinonen et al. 2010; Hvolgaard Mikkelsen et al. 2017; Skoglund et al. 2014). Individual-level covariates for infants included sex (males, females), birth weight (<2500 g, ≥2500 g), season of birth, categorized into four seasons: winter (December to March), spring (April to May), summer (June to August), and fall (September to November), and gestational age (<28 weeks, 28–32 weeks, 32–37 weeks, >37 weeks) (Arnett et al. 2015; Gao et al. 2018; Heinonen et al. 2010; Hultman et al. 2007; Lahti et al. 2006; Mick et al. 2002; Zhang et al. 2018). Birth outcomes have been associated with both environmental exposures (Hu et al. 2020; Nieuwenhuijsen et al. 2019; Yuan et al. 2019) and risk of ADHD (Perapoch et al. 2021; Rahman et al. 2021). Equally, an association between season of birth and neurodevelopment has been suggested (Grootendorst-van Mil et al. 2017).

Residential postal codes were used to assign neighborhood (at dissemination area level) income quintiles and education information to study subjects, using data from the 2001 Statistics Canada Census. A dissemination area (DA) is the smallest geographic area at which all census data are disseminated and consists of 400–700 individuals (Wilkins 2002). Household size–adjusted average family income was used to rank all DAs, which were divided into quintiles (Gan et al. 2010). We used quintiles to create a relative census variable (e.g., income etc.) and compare people living in a particular geographic region together. Education was classified into three levels: without high school diploma, secondary, and post-secondary.

We also included a census- and geographically-based index of marginalization, the Canadian Marginalization (CAN-Marg) Index (Matheson et al. 2012a). CAN-Marg has been widely used in epidemiologic research in Canada and is developed based on factor analysis (Kim and Mueller 1978; Matheson et al. 2012) of several census variables and has been demonstrated to be stable across time periods and across different geographic areas (Centre for Research on Inner City Health 2006). Unlike traditional SES indicators such as income and education, the CAN-Marg attempts to describe differences in marginalization in urban and rural areas, and measure inequalities in multiple axes of health and social well-being (Supplemental Table S1).

In previous studies, the CAN-Marg index has been associated with various health outcomes including depression, alcohol consumption, and birthweight (Matheson et al. 2006; Matheson et al. 2008; Matheson et al. 2010, 2012b; Urquia et al. 2009; White et al. 2011). The index consists of four inequality dimensions: residential instability (e.g., proportion of dwellings that are not owned), material deprivation (e.g., proportion of households living in dwellings that are in need of major repair), dependency (e.g., proportion of the population not participating in labour force), and ethnic concentration (e.g., proportion of visible minorities) (Centre for Research on Inner City Health 2006; Matheson et al. 2012a). The respective indicators included in each of the four dimensions of CAN-Marg can be found in Supplemental Table S1. In our analysis, we used quintile scores of each dimension separately.

2.4. Case ascertainment

Diagnosis from hospital records, physician visits from MSP (British Columbia Ministry of Health 2011a, 2011b; British Columbia Vital Statistics Agency 2011a, 2011b; Canadian Institute for Health Information 2011) and prescriptions from PharmaNet (a provincial network linking all pharmacies to a central set of data systems) (British Columbia Ministry of Health 2011c) were applied to identify incident cases of ADHD during the follow-up period. The ADHD case definition was the same as that applied in previously published studies by the Manitoba Centre for Health Policy (Brownell et al. 2008; Martens et al. 2010) and is supported by published evaluations compared to electronic health records with sensitivity of 76–96% and specificity of 98–99% (Gruschow et al. 2019; Shi et al. 2020). Specifically, cases of ADHD were defined as participants having: (1) one or more hospitalizations with a diagnosis of hyperkinetic syndrome (International Classification of Disease, ICD 9th version diagnostic code: 314 or ICD 10th version diagnostic code: F90); (2) one or more physician visits with a diagnosis of hyperkinetic syndrome (code 314); (3) two or more prescriptions for ADHD drugs (e.g., stimulant drugs such as methylphenidate and amphetamine, Supplemental Materials Table S2) without a diagnosis of: conduct disorder (code 312 or F63, F91, F92), disturbance of emotions (code 313 or F93, F94), or cataplexy/narcolepsy (code 347 or G47.4); or (4) one prescription for ADHD drugs in one year and a diagnosis of hyperkinetic syndrome in the previous three years.

2.5. Statistical analysis

All statistical analyses were performed using the Statistical Analysis Software (SAS version 9.4, SAS Institute Inc, Cary, NC, the United States) (SAS Institute. 2015). Cox proportional hazards regression models were used to determine the associations between ADHD incidence and greenness, air pollution, or noise in single- and multi-exposure models. Julvez et al., 2021 Relevant model assumptions were examined. Two proportional hazard model assumptions were examined. One was proportional hazard assumption in which survival curves for different cases and non-cases had hazard functions that were proportional over the time. The other one was log-linearity assumption that the relationship between the log hazard and each variable was linear. All covariates listed above were included in the models USGS. Landsat 5 tm annual greenest-pixel toa reflectance composite, 1984 *a priori*. Cases without hospitalization records were treated as censored. Person-years were calculated for study subjects from birth to the date of the first diagnosis of ADHD, or end of follow-up.

We first assessed if greenness (measured by vegetation percentage within 250-meter buffer size), air pollution, and noise were respectively associated with ADHD incidence in single-exposure models. Next, we evaluated relationships to ADHD with vegetation percentage (buffer radius of 250 m), air pollution (level at postal code centroid), and noise (level at postal code centroid) in multi-exposure models. Given that ADHD is more prevalent among boys than among girls (Khushabi et al. 2006), we conducted stratified analyses to investigate whether the association between vegetation percentage and ADHD incidence differed between boys and girls. Both vegetation percentage (250-meter) and $PM_{2.5}$ were further stratified by neighbourhood material deprivation from CAN-Marg to assess potential residual confounding of socio-economic status.

An interaction test and stratified analysis were conducted separately between vegetation percentage (250-meter buffer size) and $PM_{2.5}$. A formal interaction test (multiplicative interaction) was intended to evaluate whether the effect of $PM_{2.5}$ on the risk of ADHD was dependent upon the level of vegetation percentage. Stratified analysis was conducted to generally describe, in an accessible approach, whether both vegetation percentage and $PM_{2.5}$ were associated with incidence of ADHD in a monotonically increasing or decreasing manner (where we refer to 'joint effects'). This type of stratified analysis along with the

formal interaction test has been used previously to test additive and multiplicative interaction, respectively (Gan et al. 2012).

In stratified analysis, we examined the joint effects between vegetation percentage (250-meter) and $PM_{2.5}$ by dividing study participants into twenty-five categories based on pairs of quintiles for each of the exposure. Wolch et al., 2014 Hazard ratios of ADHD were calculated for each quintile between vegetation percentage and $PM_{2.5}$ with the first quintile (lowest) being the reference group. Additionally, we estimated hazards ratios in single- and multi-exposure models using 100- and 500-meter buffer sizes of vegetation percentage. We also tested correlations (Pearson) between the respective environmental exposures.

In sensitivity analysis, we assessed the relationships between greenness and ADHD using NDVI within 250-meter buffer size adjusting for the same covariates. Given NO_2 estimates from a newly updated local land use regression model (Su et al. 2020) (Supplemental Materials Section 1) were made available to our study, we also conducted a sensitivity analysis using this model in comparison to the national model used for the main analyses. Sensitivity analysis was also conducted after excluding movers during follow-up period.

3. Results

We identified 1,217 incident cases (4.2%) of ADHD. Distributions of exposures, child and maternal characteristics are shown in Table 1. The median vegetation percentage within 250 m buffer in the cohort was 30 % with an interquartile range of 12%, ranging from 4% to 84%. The median $PM_{2.5}$, NO_2, and noise exposures in the cohort was 7.8 μg/m^3, 16.4 ppb and 62.2 Lden dB(A), with an interquartile range of 2.1 μg/m^3, 5.9 ppb and 6.9 Lden dB(A), respectively. Only half of mothers had normal BMI. The majority of infants had normal birth weight and gestational age over 37 weeks. USGS, 2013 Most mothers had post-secondary education and about two-thirds of them lived in neighborhoods with highest ethnic concentration (as defined by the CAN-Marg index). Characteristics by cases and non-cases can be found in Supplemental Material Table S3. Vegetation percentage values across buffers were highly correlated with each other. Moderate correlations were observed between vegetation percentage and other environmental exposures across buffer zones. Results from the correlation analysis are reported in Supplemental Materials Table S4. Unadjusted model results for each exposure can be found in Table S5 in Supplemental Materials

In fully adjusted single-exposure models, for every IQR (12%) increase in vegetation percentage within 250-meter buffer size, there was a 10% (HR: 0.90, 95% CI: 0.81, 0.99) reduction in the risk of ADHD (Table 2 and Figure S1). Results were similar for the 100- and 500-meter buffer sizes (Supplemental Materials Table S6 and Figure S1). After adjusting for $PM_{2.5}$, the protective association between vegetation percentage within 250-meter buffer size and the incidence of ADHD was reduced (HR: 0.99, 95% CI: 0.91, 1.08). Similar results were also observed between NDVI and ADHD after adjusting for $PM_{2.5}$ (Supplemental Materials Table S6 and Figure S1)

For every 2.1 μg/m^3 increase in $PM_{2.5}$, there was an 11% increase in the risk of ADHD (HR: 1.11, 95% CI: 1.06, 1.17). After adjustment for vegetation percentage, the adverse association between $PM_{2.5}$ and ADHD was reduced, decreasing from 1.11 (95 %CI: 1.06, 1.17) to 1.03 (95 %CI: 0.94, 1.12) (Table 2). No statistically significant associations were found between NO_2 or noise and ADHD incidence. Based on the magnitude and patterns of effect estimates, air pollution attenuated the association between vegetation percentage and incidence of ADHD.

In the sex-stratified analysis, based on both the magnitude and confidence intervals, the association between vegetation percentage within 250-meter buffer size and decreased ADHD incidence was similar between boys and girls. Doiron et al., 2020 Results were similar for the 100- and 500-meter buffer sizes (Table 3 and Supplemental Materials Table S7). $PM_{2.5}$ was associated with higher risk of ADHD among males (HR: 1.14, 95% CI: 1.08, 1.21) compared to females (HR: 1.03, 95% CI: 0.94, 1.13) (Table 3). NO_2 and noise were not associated with ADHD incidence in stratified models. While $PM_{2.5}$ was not associated with increased ADHD risk for those in the lowest quintile of deprivation (least deprived), associations were observed at the 2nd – 5th quintiles of deprivation (Supplemental Materials Table S8). The effects of vegetation percentage and $PM_{2.5}$ on ADHD were present across all levels of SES (measured by material deprivation), indicating results were not affected by residual confounding by SES.

There were joint effects of vegetation percentage (250-meter) and $PM_{2.5}$. There was a large difference in the risk of ADHD in the highest $PM_{2.5}$ quintile/lowest vegetation percentage quintile (HR: 1.62, 95% CI: 1.08, 2.42), compared to high vegetation percentage/low $PM_{2.5}$ quintile (HR: 0.50, 95% CI: 0.37, 0.66) (Table 4). Within the lowest quintile of $PM_{2.5}$ (<6.21 μg/m^3), as vegetation percentage (250 m) increased by quintiles (from 2nd to 5th), the risk of ADHD decreased from 0.71 to 0.50 compared to reference (1st quintile). Within the lowest quintile of vegetation percentage (<24.00), the risk of ADHD increased from 1.05 to 1.62. Such increasing trend in the risk of ADHD was also observed within other quintiles of vegetation percentage (within 2nd quintile of vegetation: HR increased from 0.71 to 1.54; within 3rd quintile: HR increased from 0.80 to 1.39 etc.) (Table 4). There was, however, no significant interaction (p-value: 0.23) between vegetation percentage and $PM_{2.5}$. To further evaluate potential joint impacts of these two exposures, we assessed the linear effects of vegetation percentage (250 m) in quintiles of $PM_{2.5}$ and the linear effect of $PM_{2.5}$ in quintiles of vegetation percentage (Table 5). We found that the effects of vegetation percentage on the risk of ADHD differed across the lowest (1st) to highest (5th) quintile of $PM_{2.5}$, and the effects of $PM_{2.5}$ on the risk of ADHD also differed across levels of vegetation percentage. In short, based on the magnitude and the patterns of hazard ratios from both Table 4 and Table 5, the associations between vegetation percentage and ADHD were attenuated by $PM_{2.5}$ and vice versa.

In the sensitivity analysis, USGS, 1984 NDVI derived from Planet Scope and Landsat was generally associated with lower risk of ADHD across all buffer sizes, although the associations were nonsignificant (Supplemental materials Table S6, Figure S2 and Figure S3). Results of NO_2 estimates from the local land use regression model (Su et al. 2020) were consistent with the results of NO_2 from the national model (Hystad et al. 2011Gan et al., 2013). Results were similar after excluding movers during follow-up period (Supplemental Materials Table S9). Cox proportional hazard model assumptions (proportional hazards for cases and non-cases, linear relationship between log hazard and each variable) were met.

4. Discussion

The results indicate that higher levels of residential greenness were associated with decreased risk of incident childhood ADHD, although the association was diminished after accounting for $PM_{2.5}$. $PM_{2.5}$ was associated with an increased risk of ADHD, but the effect of $PM_{2.5}$ was attenuated substantially and became insignificant after adjustment for vegetation percentage in two-exposure models. No associations were observed between incidence of ADHD and exposure to NO_2 or noise. While a formal test did not indicate a significant interaction, there appeared to be joint effects of $PM_{2.5}$ and greenness in stratified analysis. We observed that children living in greener neighborhoods with low air pollution had a substantially decreased risk of ADHD compared to children in neighbourhoods with high air pollution levels and little greenness.

In single-exposure models, both vegetation percentage and $PM_{2.5}$ were strongly associated with the incidence of ADHD. However, such strong effects were not observed in two-exposures models. Therefore, results from single-exposure models may overestimate the true effects of greenness and $PM_{2.5}$ on ADHD. One possible reason is that due to spatial correlations between greenness and air pollution where greenness may mitigate the effects of air pollution, the beneficial effects of vegetation percentage and the harmful effects of $PM_{2.5}$ on ADHD were neutralized

Table 1
Descriptive statistics showing exposures and covariates in birth cohort (2000–2001) of attention deficit hyperactivity disorder (ADHD).

		Total (N = 28797)			
Exposures		Minimum	Median	Maximum	Interquartile
	Greenness				
	Vegetation percentage (100 m) (%)	1.00	30.00	86.00	11.00
	Vegetation percentage (250 m) (%)	4.00	32.00	84.00	12.00
	Vegetation percentage (500 m) (%)	7.00	33.00	82.00	12.00
	NDVI (Planet Scope, 100 m)	0.08	0.34	0.68	0.10
	NDVI (Planet Scope, 250 m)	0.09	0.34	0.67	0.10
	NDVI (Planet Scope, 500 m)	0.13	0.34	0.66	0.09
	NDVI (Landsat, 100 m)	0.03	0.35	0.69	0.11
	NDVI (Landsat, 250 m)	0.02	0.36	0.70	0.11
	NDVI (Landsat, 500 m)	0.04	0.36	0.66	0.11
	Air pollution				
	$PM_{2.5}$ ($\mu g/m^3$)	2.48	7.80	10.52	2.13
	NO_2 (ppb)	5.08	16.40	44.32	5.96
	Noise (Lden dB(A))	33.00	62.23	98.50	6.91
Covariates		Minimum	Median	Maximum	Interquartile
Individual (Parents)	Maternal age (years)	14	32	52	7
	Paternal age (years)	15	34	72	8
		Count	%		
	Maternal body mass index				
	Underweight	944	3.28		
	Normal	18,516	64.30		
	Overweight	7602	26.40		
	Obese	1735	6.02		
	Maternal parity				
	0 time	9190	31.91		
	1 time	9852	34.31		
	2 times	5404	18.68		
	≥3 times	4351	15.10		
Individual (Infants)	Sex				
	Females	13,889	48.23		
	Males	14,908	51.77		
	Birth weight				
	≥2500 g	27,580	95.77		
	<2500 g	1217	4.23		
	Season of birth				
	Winter (Dec – Mar)	9085	31.54		
	Spring (Apr – May)	4994	17.34		
	Summer (Jun – Aug)	7478	25.96		
	Fall (Sep – Nov)	7238	25.16		
	Gestational age				
	<28 weeks	48	0.16		
	28 – 32 weeks	119	0.41		
	32 – 37 weeks	3378	11.75		
	>37 weeks	25,252	87.68		
Neighborhood	Education				
	Without high school	1760	6.11		
	Secondary	3666	12.74		
	Post-secondary	23,371	81.15		
	Household income				
	1st quintile - lowest	4298	14.92		
	2nd quintile	5635	19.56		
	3rd quintile	5591	19.44		
	4th quintile	5962	20.70		
	5th quintile - highest	7311	25.38		
	Residential Instability				
	1st quintile - lowest	5319	18.47		
	2nd quintile	7234	25.12		
	3rd quintile	7485	25.99		
	4th quintile	4838	16.80		
	5th quintile - highest	3921	13.62		
	Material Deprivation				
	1st quintile - lowest	5500	19.09		
	2nd quintile	6682	23.20		
	3rd quintile	6608	22.94		
	4th quintile	6433	22.36		
	5th quintile - highest	3574	12.41		
	Dependency				
	1st quintile - lowest	7052	24.48		
	2nd quintile	8697	30.20		
	3rd quintile	6912	24.00		
	4th quintile	4285	14.90		
	5th quintile - highest	1851	6.42		
	Ethnic Concentration				

(*continued on next page*)

Table 1 (*continued*)

	Total (N = 28797)	
1st quintile - lowest	740	2.57
2nd quintile	803	2.79
3rd quintile	2551	8.86
4th quintile	6811	23.65
5th quintile - highest	17,892	62.13

Note: Complete-case analysis (missing values were removed to create final study sample for analysis), IQR = Interquartile range

Table 2
Hazard ratios (per one Interquartile range) between exposures and attention deficit hyperactivity disorder (ADHD).

Single-exposure models [a]	Hazard ratio (95% CI)	Two-exposures models [a,b]	Hazard ratio (95% CI)	Multi-exposures models [a,b]	Hazard ratio (95% CI)
Vegetation percentage (250 m) [12.00][c]	0.90 (0.81, 0.99)*	Vegetation percentage (250 m) [b] + $PM_{2.5}$	0.99 (0.91, 1.08)	–	–
		Vegetation percentage (250 m) [b] + NO_2	0.95 (0.86, 1.04)	–	–
		Vegetation percentage (250 m) [b] + Noise	0.90 (0.82, 0.99)*	–	–
$PM_{2.5}$ (μg/m^3) [2.13] [c]	1.11 (1.06, 1.17)	$PM_{2.5}$ + vegetation percentage (250 m)	1.03 (0.94, 1.12)	–	–
		$PM_{2.5}$ + NO_2	1.13 (1.01, 1.26)*	$PM_{2.5}$ + NO_2 + vegetation percentage (250 m)	1.05 (0.92, 1.17)
		$PM_{2.5}$ + Noise	1.09 (0.99, 1.20)	$PM_{2.5}$ + Noise + vegetation percentage (250 m)	1.02 (0.90, 1.13)
NO_2 (ppb) [5.96] [c]	1.01 (0.96, 1.07)*	NO_2 + vegetation percentage (250 m)	1.00 (0.95, 1.06)	NO_2 + Noise + vegetation percentage (250 m)	0.97 (0.88, 1.05)
		NO_2 + Noise	0.99 (0.91, 1.07)	–	–
Noise (Lden dB(A)) [6.91] [c]	1.00 (0.95, 1.05)*	Noise + vegetation percentage (250 m)	0.99 (0.93, 1.04)	–	–

[a] All models adjusted for covariates including maternal age, paternal age, maternal body mass index, maternal parity, birth weight, season of birth, gestational age, infant sex, education, household income, residential instability, material deprivation, dependency and ethnic concentration
[b] In two- and multi-exposure models, hazard ratios were reported for the main exposure after adjusting for other exposures and all included covariates in the model
[c] per Interquartile Range [IQR]
* : statistically significant at 0.05 significance level

Table 3
Hazard ratios between exposures and attention deficit hyperactivity disorder (ADHD) stratified by sex (males, females).

Exposures [a]	Attention deficit hyperactivity disorder Hazard ratio (95% CI) per Interquartile Range [IQR]		
	Non-stratified	Males	Females
Vegetation percentage (250 m)	0.90 (0.81, 0.99) [12.00]	0.89 (0.74, 1.10) [12.00]	0.90 (0.81, 1.01) [11.00]
Air pollution			
$PM_{2.5}$ (μg/m^3)	1.11 (1.06, 1.17) [2.13]	1.14 (1.08, 1.21) [2.15]	1.03 (0.94, 1.13) [2.11]
NO_2 (ppb)	1.01 (0.96, 1.07) [5.96]	1.02 (0.98, 1.09) [6.30]	1.00 (0.95, 1.06) [6.02]
Noise (L_{den} dB(A))	1.00 (0.95, 1.05) [6.91]	1.00 (0.96, 1.06) [6.95]	0.99 (0.94, 1.04) [6.90]

[a] All models adjusted for covariates including maternal age, paternal age, maternal body mass index, maternal parity, birth weight, season of birth, gestational age, education, household income, residential instability, material deprivation, dependency, and ethnic concentration

when mutually adjusted in models. Notably, however, when we evaluated associations across exposure levels of both vegetation and $PM_{2.5}$, Sbihi et al., 2017 we observed gradients in which hazard ratios were highest for those exposed to low vegetation percentage and high $PM_{2.5}$, relative to those with high vegetation percentage and low $PM_{2.5}$ levels. The effects of $PM_{2.5}$ on the risk of ADHD varied a lot across quintiles of vegetation percentage. This may suggest that potential beneficial effects of vegetation percentage were more influential on ADHD in comparison to the harmful effects of $PM_{2.5}$ on ADHD. These findings indicate the importance of investigating joint effects of multiple environmental exposures in epidemiological studies.

There are several potential mechanistic pathways behind the associations between greenness and ADHD, including stress reduction or ecosystem services, such as reduction in air pollution or noise. Our study could, to some extent, provide support for an interrelation between greenness and air pollution in the association to ADHD and our findings suggest that reduction of air pollution may be one potential pathway behind the greenness-ADHD association. However, due to the study design, we are not able to draw any causal inference about the mechanisms, Zupancic et al., 2015 and we are also unable to determine if air pollution plays a role as mediator or confounder, i.e., it may be that the association is simply due to less emission sources in green areas.

Previous studies suggest that children of mothers experiencing stress or anxiety or are exposed to air pollution during pregnancy are at increased risk of developing ADHD symptoms later in preschool or grade-school and trimester specific vulnerability windows have also been identified, although the results are inconsistent (Aghaei et al. 2019; Clavarino et al. 2010; Suades-González et al. 2015). Previous studies have shown that early childhood (in utero to the first two years) is a critical period for brain development and neuroplasticity, in which human brains reach 80% of adult size by age of two and the size of the changes minimally after age five (Bryck and Fisher 2012; Giedd et al. 1999). Early childhood thus becomes a "window of susceptibility. However, further research is needed to explicitly identify specific window of postnatal susceptibility.

Our results were generally consistent with previous population-

Table 4
Hazard ratio of attention deficit hyperactivity disorder (ADHD) by quintiles of vegetation percentage (250 m) and quintiles of fine particulate matter ($PM_{2.5}$).

Quintiles of vegetation percentage (250 m)	Quintiles of $PM_{2.5}$ ($\mu g/m^3$)				
	1st quintile - lowest	2nd quintile	3rd quintile	4th quintile	5th quintile - highest
	<6.21	6.22–7.32	7.33–8.21	8.22–8.76	>8.77
1st quintile - lowest: <24.00	Reference	1.05 (0.64, 1.70)	1.14 (0.79, 1.65)	1.41 (0.84, 2.34)	1.62 (1.08, 2.42)*
2nd quintile: 25.00–30.00	0.71 (0.47, 1.06)	0.74 (0.44, 1.23)	1.08 (0.68, 1.73)	1.32 (0.91, 1.91)	1.54 (1.00, 2.36)*
3rd quintile: 31.00–34.00	0.80 (0.57, 1.11)	0.85 (0.51, 1.40)	0.87 (0.54, 1.41)	1.24 (0.78, 1.96)	1.39 (0.95, 2.04)
4th quintile: 35.00–39.00	0.62 (0.45, 0.85)*	0.73 (0.45, 1.16)	0.93 (0.58, 1.50)	1.12 (0.74, 1.69)	1.14 (0.75, 1.73)
5th quintile - highest: >40.00	0.50 (0.37, 0.66)*	0.67 (0.39, 1.15)	0.89 (0.66, 1.20)	1.05 (0.64, 1.62)	1.08 (0.67, 1.71)

Note: All models adjusted for including maternal age, paternal age, maternal body mass index, maternal parity, birth weight, season of birth, gestational age, infant sex, education, household income, residential instability, material deprivation, dependency and ethnic concentration
* : statistically significant at 0.05 significance level

Table 5
Hazard ratios of attention deficit hyperactivity disorder (ADHD) in associated with vegetation percentage (250 m) stratified by quintiles of and fine particulate matter ($PM_{2.5}$) and $PM_{2.5}$ stratified by quintiles of vegetation percentage (250 m).

	Exposures	Attention deficit hyperactivity disorder*
		Hazard ratio (95% CI) per Interquartile Range [IQR]
Non-stratified	Vegetation percentage (250 m)	0.90 (0.81, 0.99)* [12.00]
Stratified by $PM_{2.5}$	1st quintile - lowest	0.93 (0.77, 1.16) [11.00]
	2nd quintile	0.94 (0.78, 1.17) [9.00]
	3rd quintile	0.99 (0.81, 1.22) [9.00]
	4th quintile	0.95 (0.79, 1.20) [10.00]
	5th quintile - highest	1.03 (0.84, 1.26) [8.00]
Non-stratified	$PM_{2.5}$ ($\mu g/m^3$)	1.11 (1.06, 1.17)* [2.13]
Stratified by Vegetation percentage (250 m)	1st quintile - lowest	1.49 (1.19, 1.88)* [2.20]
	2nd quintile	1.42 (1.09, 1.84)* [2.12]
	3rd quintile	1.34 (1.01, 1.77)* [2.10]
	4th quintile	1.16 (0.88, 1.52) [2.07]
	5th quintile - highest	1.04 (0.77, 1.39) [2.17]

Note: Covariates included for ADHD: Maternal age, father's age, maternal body mass index, maternal parity, birth weight, season of birth, gestational age, infant sex, education, household income, residential instability, dependency, and ethnic concentration
* : statistically significant at 0.05 significance level

based cohort studies, which reported protective associations between greenness and ADHD. For example, a population-based cohort study (N = 66,823) in Germany found that 0.1 unit increase in NDVI was associated with 18% (95% CI: 0.68, 0.98) decrease in the relative risk of ADHD (Markevych et al. 2018Margaritis and Kang, 2017). A longitudinal study (N = 49,923) in New Zealand found that the odds of developing ADHD in the group of children with the highest NDVI exposure (after two years of age) was 0.66 (95% CI: 0.54, 0.80) times that in the group of children with the lowest NDVI exposure (Donovan et al. 2019), although greenness exposure before the age of two was not associated with a decreased risk of ADHD. A population-based cohort study in Denmark (N = 814,689) reported higher risk (Incidence rate ratio: 1.55, 95 %CI: 1.45, 65) of ADHD among subjects living in areas with lowest level of NDVI compared with subjects living in areas with the highest level of NDVI (Thygesen et al. 2020a).

Our findings were also consistent with previous cross-sectional studies that focused on symptoms of ADHD related to greenness. A study in China including 59,754 children reported a 13 % reduction (95 %CI: 0.83, 0.91) in the odds of ADHD symptoms among children with 0.1 unit increase in NDVI near schools (Yang et al. 2019). A cross-sectional study (N = 2,623) in Spain found that residential surrounding NDVI in 100-meter buffer had a positive impact on ADHD symptoms in school-age children (Amoly et al. 2014).

There were some difference and similarities between our studies and previous ones. Compared to previous studies, we used vegetation percentage as the metric of greenness, in addition to NDVI. Our study used multiple criteria such as hospital diagnosis, physician visits and prescription drugs to identify ADHD incident cases, instead of ADHD symptoms from questionnaires or survey. The follow-up period in our study is around seven years, while previous studies have a follow-up period ranging between 7 and 19 years. The relatively short follow-up period in our study, due to no data availability beyond 2010, may lead to a smaller number of incident ADHD cases being identified and likely underestimation of hazard ratios.

Mixed findings with respect to associations between air pollution and ADHD have been reported. A study (N = 3,426) conducted among Swedish twins suggested that there were no clear or consistent associations between air pollution exposure (NO_x and PM_{10}) and ADHD (Gong et al. 2014). Similarly, a study in the Netherlands (N = 553) indicated no association between NO_2 and ADHD (Van Kempen et al. 2012). However, the number of studies reporting an association between air pollution and increased risk of ADHD is relatively higher compared to the number of studies reporting no association (Aghaei et al. 2019; Zhang et al. 2020). Our results were in line with most previous studies, which reported associations between air pollution and ADHD. In Denmark, a large prospective cohort study (N = 809,654) reported relative risk of 1.38 (95% Cl: 1.35, 1.42) per 5 $\mu g/m^3$ increase in $PM_{2.5}$ (Thygesen et al. 2020b). In a population-based cohort study (N = 9,565) in Korea, with 1 $\mu g/m^3$ increase in PM_{10}, the hazard ratio of childhood ADHD was 1.18 (95% CI: 1.15, 1.21) (Min and Min 2017). In a German cohort study, relative risk of 1.97 (95% CI: 1.35, 2.86) in ADHD was associated with a 10 $\mu g/m^3$ increase of PM_{10} (Markevych et al. 2018).

We found no evidence for an association between noise and ADHD, similar with a cross-sectional study in the Netherlands (N = 2,230) incidence (Zijlema et al. 2020Sadeh et al., 2020). The null results maybe due to our relatively short exposure period. On the other hand, a large cohort study (N = 46,940) from Denmark found that a 10-decibel increase in noise exposure from birth to age seven was associated with a 9% increase in abnormal hyperactivity/inattention subscale scores (Hjortebjerg et al. 2016).

Our study has several strengths. First, case ascertainment of ADHD was based on validated criteria developed over time in Canada with high sensitivity and specificity (Brownell et al. 2018; Brownell and Yogendran 2001; Martens et al. 2007). Many previous studies relied on ADHD symptoms reported by teachers and parents via questionnaires or attention tests rather than doctor-validated diagnosis (Amoly et al.

2014; Faber Taylor and Kuo 2009, 2011; Kuo and Faber Taylor 2004; Mårtensson et al. 2009; Taylor et al. 2001; Wells 2000). Second, while previous studies have examined the effects of both greenness and air pollution on ADHD (Aghaei et al. 2019; Markevych et al. 2018; McCormick 2017), we are unaware of population-based cohort studies that have also included noise exposure. Third, most previous studies adopted cross-sectional designs with relatively small sample sizes (Amoly et al. 2014; Faber Taylor and Kuo 2009, 2011; Kuo and Faber Taylor 2004; Mårtensson et al. 2009; Taylor et al. 2001; Wells 2000), while only a few studies examined large population-based cohorts (Dadvand et al. 2017; Donovan et al. 2019; Markevych et al. 2018; Thygesen et al. 2020a).

Fourth, this is one of few epidemiological studies (Jarvis et al. 2021) to employ a novel and precise greenness exposure metric of vegetation percentage in association to health, in addition to the traditionally used NDVI. To date, the majority of epidemiological studies have relied on NDVI for exposure assessment in greenness and health research. Typically, it has been derived from satellite imagery, such as from the Landsat with a 30 m spatial resolution. While NDVI provides an objective measure of healthy vegetation, the use of 30 m resolution data introduces the risk of exposure misclassification due to, for example, the mixed pixel problem (Badgley et al. 2017), where disparate materials jointly occupy a single pixel. Mixed pixels also lead to difficulty in capturing small patches of greenness, for example, pocket parks or domestic gardens (Labib et al. 2020; Xu et al. 2017). The vegetation percentage we used was derived from a linear unmixing process, which identifies regionally calibrated pure pixels and likely provides a more accurate estimate of vegetation coverage compared to NDVI, especially for smaller green patches (Czekajlo et al. 2020, Davis et al. 2021). Given the pure pixels are directly sampled from the imagery, the vegetation percentage is relative to real values. NDVI, however, is not calibrated to a study area and can be insensitive when comparing between areas with different vegetation or other surfaces (Huete et al. 1997). Moreover, vegetation percentage was expressed in percent, ranging from 0 to 100, which provides higher variability (Sadeh et al. 2021b) and is more easily interpreted than NDVI (a ratio ranging from −1 to 1), which is usually expressed in quartiles or quintiles. In brief, NDVI is the overall level of greenness within a pixel, whereas vegetation percentage is more sensitive to vegetation within pixels that are mixed (not entirely greenness). Vegetation percentage is more accurate for urban areas where most of the land use is mixed (Sadeh et al. 2021b). Sadeh et al. 2021 reported that the dispersion of the greenness values detected was 60% higher when greenness was measured using the linear spectral unmixing in comparison to NDVI. Last, to our best knowledge, our study is the first to investigate joint effects of multiple environmental exposures on the incidence of ADHD.

Despite these strengths, our study has several limitations. First, there is a risk of residual confounding because data on some variables were not available in this population-based cohort. We did not have individual-level information on education and income for parents, instead we included neighborhood-level household income and education in the models. Due to high percentage of missing values (>75%), we were unable to account for potentially important parental behavioral risk factors, such as smoking or alcohol consumption. We were also unable to include breastfeeding as a covariate due to lack of information following hospital discharge. Although there may be a hereditary component of the risk of ADHD, we were unable to control for this because we did not have available data on family history of neurodevelopmental problem. Second, while cases were ascertained based on criteria with very high specificity (98–99%) and relatively high (76–96%) sensitivity (Gruschow et al. 2019; Shi et al. 2020), it is likely that not all incident cases were captured in our study because the case ascertainment depended on access to health care. Underreporting of incident ADHD cases may lead to underestimation of hazard ratios and bias towards null. Third, exposures to greenness, air pollution, and noise were assessed based on residential postal codes rather than individual linkages, likely resulting in some exposure misclassification, although in our densely populated study area, a residential postal code unit represents a relatively small spatial area (around half a city block) (Khan et al. 2018). As is common in large cohort analyses, these exposure estimates also do not fully represent personal exposures. Specifically, these estimated exposures do not account for individual factors (e.g., the time participants spent at home or activity or travelling patterns) or exposures encountered indoors (e.g., air pollution from cooking) (Helbich 2018). This limitation often results in non-differential exposure misclassification, likely biasing the results toward null (Yuchi et al. 2020). Thus, effect estimates may have been greater if we had been able to capture individual exposures. Fourth, there is a risk of selection bias because healthy parents may move to greener and healthier neighborhoods right after children were born. It is possible that the effects of greenness were overestimated. Another limitation was that our exposure period (from birth until the age of three for all children) may be relatively short. The three-year exposure period may not reflect the average exposures for children if they moved to a different neighborhood after the age of three. As a result, the exposure period may under- or overestimate the true exposures of children depending on where children moved. In our study, results were similar after excluding movers. However, future studies may have different findings depending on sample size and follow-up period. Another limitation is that the relatively short follow-up period in our study may lead to a smaller number of incident ADHD cases being identified and likely underestimation of hazard ratios.

We also used selected years (2000–2004) of annual greenness values which were smoothed using data outside exposure period. It is possible that there were exceptionally dry or wet years making a single year being more/less green. Therefore, these smoothed values may not represent true-ground conditions related greenness at the time. Additionally, it is possible that the impact on neurodevelopment is also influenced by the perception of environmental exposures, such as view of or interactions with greenness. Our database only included objectively measured environmental exposures and we were unable to address this aspect, something that could be considered in future studies. Last, it is possible that reverse causation may exist in this cohort study due to residential self-selection (Boone-Heinonen et al. 2011; Kaczynski and Mowen 2011; Pinjari et al. 2009).

5. Conclusions

In this population-based cohort study, greenness was associated with lower incidence of ADHD during childhood while $PM_{2.5}$ exposure was associated with a higher risk. However, greenness was no longer associated with incidence of ADHD in multi-exposure model in which we adjusted for $PM_{2.5}$. Joint effects between greenness and $PM_{2.5}$ on ADHD suggested that there were environmentally related inequalities in health outcome where children living in neighborhoods with the highest $PM_{2.5}$ exposure and the lowest greenness exposure were at significantly greater risk of ADHD. This suggests that future studies using similarly precise vegetation measures and addressing joint effects of multiple environmental exposures are warranted, in particular in other urban areas with different environmental conditions to test the generalisability of our results.

CRediT authorship contribution statement

Weiran Yuchi: Conceptualization, Investigation, Resources, Formal analysis, Writing – original draft, Writing – review & editing, Data curation, Visualization. **Michael Brauer:** Conceptualization, Investigation, Resources, Writing – review & editing, Supervision. **Agatha Czekajlo:** Investigation, Resources, Writing – review & editing, Data

curation. **Hugh W. Davies:** Investigation, Resources, Writing – review & editing. **Zoë Davis:** Investigation, Resources, Writing – review & editing. **Martin Guhn:** Investigation, Resources, Writing – review & editing. **Ingrid Jarvis:** Investigation, Resources, Writing – review & editing. **Michael Jerrett:** Investigation, Resources, Writing – review & editing. **Lorien Nesbitt:** Investigation, Resources, Writing – review & editing. **Tim F. Oberlander:** Investigation, Resources, Writing – review & editing. **Hind Sbihi:** Investigation, Resources, Writing – review & editing. **Jason Su:** Investigation, Resources, Writing – review & editing. **Matilda van den Bosch:** Conceptualization, Investigation, Resources, Writing – review & editing, Supervision, Project administration, Funding acquisition.

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 material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2022.107120.

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