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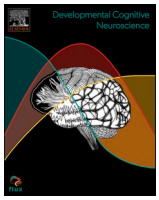

Air pollution and cortical myelin T1w/T2w ratio estimates in school-age children from the ABCD and NeuroSmog studies.

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ABSTRACT

Air pollution affects human health and may disrupt brain maturation, including axon myelination, critical for efficient neural signaling. Here, we assess the impact of prenatal and current long-term particulate matter (PM) and nitrogen dioxide (NO_2) exposure on cortical T1w/T2w ratios – a proxy for myelin content – in school-age children from the Adolescent Brain Cognitive Development (ABCD) Study (United States; N = 2021) and NeuroSmog study (Poland; N = 577), using Siemens scanners. Across both samples, we found that NO_2 and PM were not significantly associated with cortical T1w/T2w except for one association of PM_{10} with lower T1w/T2w in the precuneus in NeuroSmog. Superficially, ABCD Study analyses including data from all scanner types (Siemens, GE, Philips; N = 3089) revealed a negative association between NO_2 exposure and T1w/T2w ratios. However, this finding could be an artifact of between-site sociodemographic differences and large scanner-type-related measurement differences. While significant associations between air pollution and cortical myelin were largely absent, these findings do not rule out the possibility that air pollution affects cortical myelin during other exposure periods/stages of neurodevelopment. Future research should examine these relationships across diverse populations and developmental periods using unified analysis methods to better understand the potential neurotoxic effects of air pollution.

1. Introduction

Air pollution is known to adversely affect human health, with well-known damage to cardiovascular and pulmonary systems (Boogaard et al., 2022; Schraufnagel et al., 2019) and an emergent literature on neurological impacts across the lifespan (Cory-Slechta et al., 2023). Its effects might be particularly deleterious during early years, when they can disturb critical and delicate developmental processes (Cory-Slechta et al., 2023; Herting et al., 2024). As such, in the last decade, a number of neuroimaging studies have investigated how particulate matter (PM)

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and nitrogen dioxide (NO_2) exposure through breathing polluted air may influence brain development in children, particularly its structure (Cory-Slechta et al., 2023; Herting et al., 2019, 2024; Polemiti et al., 2024).

A substantial amount of evidence now shows that inhaled pollutant particles can directly access the brain by crossing the blood-brain barrier, which can induce blood-brain barrier and neuronal damage, cell death, and a neuroinflammation cascade (Kang et al., 2021). Emerging evidence also suggests that inhalation of metals within PM plays a key role in neurotoxicity of airborne particles, causing neuronal cell death, oxidative stress, and inducing dyshomeostasis in the brain (Cory-Slechta et al., 2020; Fang et al., 2017). Myelination of axons, critical for efficient transduction of neural signals, is thought to be a key target for these adverse impacts (reviewed in Cory-Slechta et al., 2023; Herting et al., 2024). Human and animal model studies suggest that myelination may be altered by both PM and NO_2 exposure. A human post-mortem/magnetic resonance imaging (MRI) study found a greater prevalence of white matter hyperintensities in both children and dogs from Mexico City as compared to those from cities in Mexico with lower air pollution concentrations (Calderón-Garcidueñas et al., 2008). Several cross-sectional and longitudinal diffusion MRI studies have also consistently shown prenatal and/or childhood air pollution exposure is associated with altered white matter microstructure (Binter et al., 2022; Burnor et al., 2021; Cotter et al., 2024; Kusters et al., 2024; Lubczynska et al., 2020; Peterson et al., 2022; Pujol et al., 2016). In accordance with this literature, experimental animal models have also shown that adult PM pollution exposure impacts microglial function (Levesque et al., 2011, 2013). Similarly, PM exposure has been directly linked to impaired myelin repair and aberrant myelination in an adult mouse model (Parolisi et al., 2021).

Further investigation of air pollution effects on myelin, however, might be particularly enlightening, given inconsistent human structural and diffusion MRI findings in the field regarding brain areas affected and effect directions among studies (Herting et al., 2019; Morrel et al., 2024). Prior inconsistencies may stem from differences in air pollution concentrations (e.g., Strak et al., 2021), different pollutant types, and/or differences in imaging methods and analysis pipelines. Moreover, the demographic composition of these previous studies are also variable (Morrel et al., 2024). For example, while the Columbia Center for Children's Environmental Health (CCCEH) study mostly included families from disadvantaged socioeconomic backgrounds from the Bronx in New York City (Peterson et al., 2022), the HCP-D study included families with median annual incomes of $100,000, with a large fraction of mothers with Master's, PhD or professional degrees (Bos et al., 2023). As more air pollution results emerge, it becomes increasingly important to compare findings between cohorts using unified data analysis approaches and to seek new neuroimaging modalities that may illuminate how ambient air quality is linked to white matter development.

In this regard, the T1w/T2w ratios in white matter may potentially decipher how air pollution impacts myelination during human brain development. T1w and T2w acquisitions are perhaps the most used sequences in all brain imaging research. They provide high-quality structural images of the brain, emphasizing complementary (either T1 or T2) brain relaxation tissue properties. The T1w/T2w ratio is the ratio of these two property types and provides a quantitative metric that correlates with the amount of myelination within the cortex (Glasser et al., 2013). The T1w/T2w approach has been used in studies of psychiatric disorders (Ganzetti et al., 2015), aging (Rokicki et al., 2021), and recently, the impact of socioeconomic status on the brain (Weissman et al., 2023). Yet, to our knowledge, no study has examined the impact of air pollution exposure on T1w/T2w ratio outcomes.

Here, we investigate the associations between air pollution and cortical myelin using T1w/T2w ratios in school-age children from two independent study samples: **The Adolescent Brain Cognitive Development℠ Study (ABCD Study®)** in the United States and NeuroSmog in Poland. The populations were exposed to similar NO_2 levels, but divergent PM levels, which were approximately three-fold higher in the Polish population (Table 1) as southern Poland has some of the highest air pollution levels in the European Union (Horálek et al., 2023). While these two cohorts are exposed to different PM levels and comprise of varying sample sizes, they nonetheless have similar participant ages and identical MRI acquisition protocols. This allowed us to apply a unified data analysis approach to both datasets. We hypothesized that higher air pollution exposure would be related to decreases in cortical myelin, as indexed by the T1w/T2w ratios, in both the ABCD and NeuroSmog samples. Given the differences in sample sizes, we also hypothesized effects, if present, would be more widespread in the ABCD study given increased statistical power to detect smaller effects.

2. Methods

2.1. Study populations

We examined two independent cohorts: the *Adolescent Brain Cognitive Development (ABCD Study)* and *NeuroSmog*. An overview of these cohorts is presented in Table 1, with additional details for each provided below.

Table 1
Comparison of ABCD and NeuroSmog studies.

	ABCD	NeuroSmog
Location	United States	Southern Poland
Age range	9–10 years old	10–13 years old
Participants with T1w/T2w ratio/total number of participants in study cohort	4713/11,868	602/741
Sampling	Multi-stage probability sampling	Random stratified (Typically developing group) / Convenience (ADHD diagnosis group)
Race and ethnicity	Diverse*	Homogenous
Number of study sites with a Siemens MRI scanner	13	1
Scanner manufacturer	General Electric Medical Systems, Philips Medical Systems, Siemens	Siemens
City size	Predominantly urban metropolitan regions	Large (>90,000 pop.) and small cities
Air pollution exposures	Prenatal and Current $PM_{2.5}$, NO_2	PM_{10}* *, NO_2
Current particulate matter (PM) Median (Range)	$PM_{2.5}$: 7.74 μg/m^3 (1.72–15.90)	PM_{10}: 37.29 μg/m^3 (11.55–56.59)
Prenatal particulate matter (PM) Median (Range)	$PM_{2.5}$: 11.06 μg/m^3 (2.04–23.65)	PM_{10}: 44.89 μg/m^3 (16.04–99.55)
Current nitrogen dioxide (NO_2) Median (Range)	18.85 ppb (0.62–37.94)	19.32 ppb (3.34–58.6)
Prenatal nitrogen dioxide (NO_2) Median (Range)	25.96 ppb (1.03–63.89)	23.18 ppb (7.74–73.70)

Summary of characteristics of ABCD (n = 11,868) and NeuroSmog (n = 741) study cohorts. Air pollution data for particulate matter (PM) and nitrogen dioxide (NO2) included prenatal estimates (averaged across 9 months of pregnancy based on the child's birthdate) and estimates from the initial year of the studies (2016 for ABCD, and 2018 for NeuroSmog). *) Description of race and ethnicity provided in Table 2 **) PM10 and PM2.5 levels are highly correlated (Pearson R = 0.9) for current PM levels in the NeuroSmog study area, with PM2.5 concentrations equal to 74.8% of PM10 concentrations (see Methods for details). Abbreviations: ABCD, Adolescent Brain and Cognitive Development; ADHD, attention deficit hyperactivity disorder; MRI, magnetic resonance imaging.

2.1.1. The Adolescent Brain Cognitive Development (ABCD) study

The ABCD Study® is the largest long-term study of brain development and child health in the United States (https://abcdstudy.org). The study enrolled 11,880 9- and 10-year-old participants in 2016–2018 from 21 study sites across the United States (see Fig. A1; Garavan et al., 2018; Jernigan et al., 2018; Volkow et al., 2018). Across all sites, inclusion of children was based on required age range, whereas exclusion criteria included lack of English proficiency or presence of severe sensory, intellectual, medical, or neurological issues that would impact valid data collection or compliance with the study protocol. The study implemented an identical, harmonized protocol for recruitment and neuroimaging of all participants at each site (Casey et al., 2018; Feldstein Ewing et al., 2018; Garavan et al., 2018; Hagler et al., 2019; Luciana et al., 2018). Centralized Institutional Review Board (IRB) approval was obtained from the University of California, San Diego. Study sites obtained approval from their local IRBs. Written informed consent was provided by each parent or caregiver; each child provided verbal assent.

Data for the current analyses were acquired from the National Institute of Mental Health Data Archive (NDA) 5.0 data release (https://doi.org/10.15154/8873-zj65) although attention-deficit/hyperactivity disorder (ADHD) status information was acquired from the 4.0 release (https://doi.org/10.15154/1523041) due to missing data in the 5.0 release.

Variables specific to the current analyses included ambient air pollution concentrations for $PM_{2.5}$ and NO_2 during the prenatal period (entire period of pregnancy) and at the first timepoint of the study (baseline, i.e., ages 9–10 years) at the residential address, as well as valid T1w/T2w MRI data and additional confounders (see Appendix Methods & Table A1). From the larger ABCD study sample, we applied a more stringent set of exclusionary criteria in line with Neurosmog exclusionary criteria (see details below) to reduce potential confounding due to preterm birth and/or birth complications (Fig. A2). We excluded participants with a gestational age of less than 35 weeks, a birthweight < 2500 g, or if they had ≥ 2 birth complications that served as proxies for several APGAR indicators (i.e., activity, appearance, pulse, and respiration but not grimace). Furthermore, given that the NeuroSmog cohort was collected on a Siemens scanner, and the large effect sizes of scanner manufacturer on brain outcomes (Chen et al., 2014) including in ABCD (Dudley et al., 2023), we conducted two sets of analyses. First, our a priori set of analyses included ABCD participants collected only on Siemens MRI scanners ($N = 2021$). However, to determine if these findings generalized to other scanner types, we also conducted an additional analysis using ABCD participant data from all scanners ($N = 3089$; participant selection flowchart in Fig. A2). Moreover, we chose one participant per family to reduce nonindependence and improve the model fit.

2.1.2. NeuroSmog

The NeuroSmog study is a study of brain and cognitive development based in 18 towns in southern Poland, a region characterized by high levels of air pollution (Fig. A1). The study was initiated to assess whether long-term exposure to outdoor air pollution affects brain function, structure, and connectivity in children diagnosed with ADHD and those without ADHD (i.e., typically developing, TD) (Compa et al., 2023; Markevych et al., 2022). Between October 2020 and July 2022, 741 children aged 10–13 were enrolled. Eighteen study towns classified as either large (>90,000 inhabitants, including Kraków, Częstochowa and the Silesian agglomeration) or small (e.g., Skawina, Zakopane, and Kłobuck) and were selected based on their levels of particulate air pollution and location within two hours' drive to the MRI scanning center in Kraków. TD children were randomly selected from randomly selected schools in the 18 study towns (stratified random sampling). Children with ADHD were recruited using convenience sampling among children living in the same 18 towns and attending the same schools as the selected TD children. They were referred to the study by local psychological counseling centers, school psychologists, medical doctors, or they applied for participation through their parents. ADHD was diagnosed according to the 11th revision of the International Classification of Diseases and Related Health Problems (ICD-11) (World Health Organization, 2019) and based on a test battery including the Continuous Performance Test (CPT), a variant of the Go/NoGo task (Conners, Sitarenios, et al., 2018), the Third Edition of Conners' Rating Scales (Conners, Wujcik, et al., 2018), the Child Behaviour Checklist (CBCL) for Ages 6–18 (Achenbach and Rescorla, 2001; Wolańczyk, 2002), and Youth Self-Report (YSR) (Wolańczyk, 2002). Polish versions of these tests were used. Diagnosis was independently verified by project psychologists. Inclusion of children was based on school year, whereas exclusion criteria were intellectual disability, neurological or comorbid psychiatric disorders, or serious medical conditions. Children with gestational age outside of the 35th and 42nd week range, with an APGAR (Appearance, Pulse, Grimace, Activity and Respiration) score lower than 8, or birth weight below 2500 g were also excluded, as were children who did not live in Poland for at least the latest year, were not fluent in Polish, or had parents who were not fluent (see subject exclusion flowchart in Fig. A3). The final NeuroSmog analytic sample included 577 participants.

Although NeuroSmog was designed to test the hypothesis that individuals with ADHD may be more susceptible to the effects of air pollution, the ABCD Study does not have clinician-based ADHD diagnosis information. Thus, to harmonize the two studies, the current set of analyses accounted for ADHD status as a fixed effect, rather than aiming to test modification of exposure by ADHD diagnosis.

2.2. Air pollution exposure

Both studies included PM measures, although different PM fractions were used across the studies: PM_{10} for NeuroSmog and $PM_{2.5}$ for ABCD. Distributions of current air pollution exposure in both samples are in the plotted in the appendix (Fig. A4).

2.2.1. ABCD study

Briefly, ambient air pollution estimates $PM_{2.5}$ ($\mu g/m^3$) and NO_2 (ppb) were calculated at the home address of the study participants during the prenatal period (9-month average) as well as for a one-year annual average exposure when the participants were ages 9–10 years using hybrid spatiotemporal models. These models utilized satellite-based aerosol optical depth models, land-use regression, and chemical transport models to derive daily estimates across the continental United States at a 1-km^2 resolution (Di et al., 2019, 2020; Requia et al., 2020). In the ABCD Study, 91 % ($N = 7837$) of participants' caregivers of the study sample reported valid residential information dating back to the child's birth year. For these participants, prenatal exposure was assigned by averaging across the daily exposure estimates for 9 months of pregnancy based on the child's birthdate and linked to the address congruent with the child's date of birth. One-year annual averages were estimated across the 2016 calendar year based on the primary residential address reported at the time of study entry when the children were 9–10 years of age (Fan et al., 2021).

2.2.2. NeuroSmog

Ambient exposure estimates were calculated for all months covering the children's prenatal period as well as for one-year annual exposure during the year preceding the onset of the study (2018). Raster maps of NO_2 and PM_{10} were developed using hybrid land use regression (LUR) models. For NeuroSmog, $PM_{2.5}$ modeling was attempted, but there were too few $PM_{2.5}$ monitoring stations in the study area to allow for adequate modeling. Nonetheless, one can roughly gauge the $PM_{2.5}$ exposures of the NeuroSmog population from the fact that $PM_{2.5}$ and PM_{10} levels at monitoring stations in the NeuroSmog study area were highly correlated (Pearson $R = 0.91$ and 0.90 for current ($p < 0.001$) and prenatal ($p < 0.001$) periods, respectively), with $PM_{2.5}$ levels equal to 74.8 % and

76.7 % of PM_{10} levels for current and prenatal periods, respectively (range 65.9 %-84.9 % and 69.4 %-85.6 %, respectively). Detailed methods for modeling and exposure assignment are provided in the Appendix Methods.

2.3. MRI Acquisition and T1w/T2w Preprocessing

2.3.1. ABCD Study

In terms of structural MRI, a harmonized data protocol was utilized across sites (Casey et al., 2018). Motion compliance training, as well as real-time, prospective motion correction, was used to reduce motion distortion. T1w images were acquired using a magnetization-prepared rapid acquisition gradient echo (MP-RAGE) sequence and T2w images were obtained with fast spin echo sequence with variable flip angle (Casey et al., 2018). Both T1w and T2w images consisted of 176 slices with 1 mm^3 isotropic resolution. We obtained pre-processed whole--brain vertex-wise T1w/T2w ratio data from the NIMH Data Archive (N = 4713) (Feczko et al., 2021). In the ABCD T1w/T2w dataset from the NIMH Data Archive, a quality control procedure was already implemented, and only data that passed the Data Analysis, Informatics, and Resource Center (DAIRC) quality control were included (Hagler et al., 2019), including the removal of negative T1w/T2w ratio values. Furthermore, only images without clinically significant incidental findings (mrif_score = 1 or 2) that passed all ABCD quality-control parameters were included in the analysis (imgincl_t1w_include and imgincl_t2w_include = 1).

2.3.2. NeuroSmog

All NeuroSmog neuroimaging data were acquired at the Małopolska Centre of Biotechnology, Jagiellonian University in Kraków, Poland, on a Siemens MAGNETOM Skyra 3 T MRI scanner using a 64-channel head coil. Participants were familiarized with the scanner and trained to remain still using a mock scanner (pstnet.com). The protocol includes T1-weighted (T1w) and T2-weighted (T2w) structural images identical to those used in the Adolescent Brain Child Development study (Casey et al., 2018). NeuroSmog data were preprocessed in-house using the same tools as the ABCD data, i.e., using scripts from the ABCD Community MRI Collection and Utilities (https://collection3165.readthedocs.io). The preprocessing steps included distortion correction, brain extraction, alignment, surface reconstruction, and registration to standard templates. The "MyelinMap_BC" bias field correction method included in the HCP minimal preprocessing pipeline was applied (Glasser et al., 2013). T1w/T2w ratios were then mapped on the cortex using the HCP method (create_ribbon.sh), which comprises smoothing data with a cylindrical weighting function.

2.3.3. Extraction of atlas-based data – both datasets

Subsequently, for both datasets, we employed the Human Connectome Workbench utility (wb_command) to extract atlas-based tabulated data from whole-brain data. The T1w/T2w ratio values were mapped to a normalized 32k_FS_LR surface and averaged for regions of interest (ROIs) from the Desikan-Killiany template-based parcellation in a shared "fsaverage" (32k_FS_LR) space. In NeuroSmog, the quality of all structural data was assessed with the mriqc (ver. 22.0.6) pipeline. We excluded all subjects with a T1 Signal to Noise Ratio (SNR) lesser than or equal to 8.9. This threshold was determined empirically as the minimal SNR value necessary for sufficient Freesurfer parcellation quality by manually inspecting Freesurfer parcellations for subjects with SNR values is between 8 and 9. After this thresholding, the Neurosmog data contained some biologically implausible (negative or very high) T1w/T2w ratios in some brain areas. Therefore, for the Neurosmog data, after obtaining tabulated atlas data, we additionally filtered individual data points (i.e., individual subjects' atlas ROI averages) using the interquartile range (IQR) method, with a threshold of ± 3 IQR.

2.4. Statistical analyses

2.4.1. ABCD analysis

Analyses were performed using R software (version 4.3.1; R Core Team 2023) with the lme4 package and glmer() command (Bates et al., 2015). We ran four sets of analyses to examine the effect of each air pollutant ($PM_{2.5}$ and NO_2 measured prenatally and currently, one-year average at address corresponding to the baseline study visit, at ages 9–10 years) on T1w/T2w myelin ratios (34 cortical regions per hemisphere). NO_2 was scaled to 10 ppb for all models to improve model convergence. We have applied a minimally sufficient adjustment set of confounders and covariates based on our extensive air pollution and brain research in the ABCD Study to date (Burnor et al., 2021; Cotter et al., 2024; Sukumaran et al., 2023). Specifically, we aimed to minimize bias when estimating the association between annualized exposure and T1w/T2w myelin ratios at ages 9–10 years old. Since the exposure is based on the primary residential address of the child, the potential confounder (c) must predict both the residential location of the child (X) and their brain outcome (Y). Pollution levels are higher in minority communities and for those from disadvantaged social status backgrounds in the United States (Hajat et al., 2015). Thus, as a function of racialization in the U.S., a child's primary caregiver's race/ethnicity (Asian/Other, Black, Hispanic, or White) may influence parental education (<High School Diploma, High School Diploma or a General Educational Development (GED) equivalent, Some College, Bachelor Degree, or Post Graduate Degree) and total household income (see Appendix Methods), which are likely to influence both the geolocation of the child's primary residence as well as neurodevelopmental outcomes, and thus are considered potential confounders. Although not confounders, other key demographic characteristics of the child such as their age, sex (Female or Male), pubertal stage (Pre-puberty, Early puberty, Mid-puberty, or Late/Post-Puberty via the pubertal development scale (Carskadon and Acebo, 1993; Petersen et al., 1988), and handedness (Left, Mixed, or Right) are also likely to influence the outcome, and thus are considered precision variables. Since NeuroSmog was designed to include typically developing children and children with ADHD diagnosis, for greater parsimony between the studies, we also included a confounder for ADHD in the ABCD analysis. Children were considered to have ADHD status in the dataset if they were diagnosed with ADHD in partial remission, unspecified ADHD, or present ADHD according to the computerized Kiddie Schedule for Affective Disorders and Schizophrenia (Townsend et al., 2020) or if they were taking ADHD medications (amphetamine, methylphenidate, or non-stimulants).

In addition to adjusting for each confounder and precision variable as a fixed effect, we implemented a random effect of participants nested within the ABCD study sites to account for the nested structure of the ABCD data. Residual diagnostics were performed to ensure all model assumptions were met and model convergence issues were resolved by adapting an optimization strategy that was confirmed to ensure precision and accuracy via a series of sensitivity analyses (see Appendix Methods). Analyses were corrected for multiple comparisons using a false discovery rate (FDR) correction at a 0.05 level of significance. In the secondary, sensitivity analysis conducted using data from all three MRI scanners, we also included scanner manufacturer (Siemens, General Electric Medical Systems, and Philips Medical Systems) as a precision variable.

2.4.2. NeuroSmog analysis

For the NeuroSmog analysis, we implemented four separate sets of linear regressions for each exposure (NO_2/PM_{10}) at each time point of interest (prenatal/current), while adjusting for key confounders of age, sex, town size, and ADHD diagnosis. Since air pollution was not significantly correlated with indicators of socioeconomic status (parental education), we did not include SES measures in our initial air pollution linear regressions.

3. Results

3.1. Study population and exposure distribution

The analytical ABCD and NeuroSmog samples are described in Table 2.

The ABCD sample analyzed here was more likely to be White, report higher caregiver education, have slightly lower $PM_{2.5}$ exposure, and have greater NO_2 exposure as compared to the full ABCD study cohort (Table A2). Median current air pollution exposure levels for ABCD participants were 7.50 μg/m^3 for $PM_{2.5}$ and 19.26 ppb for NO_2. Median prenatal air pollution exposure levels were 10.31 μg/m^3 for $PM_{2.5}$ and 25.51 ppb for NO_2. For ABCD participants, correlations between prenatal and current exposures for each pollutant were moderate to high, whereas NO_2 - $PM_{2.5}$ exposure correlations were weak to moderate (Fig. 1A).

Median current air pollution exposure levels for NeuroSmog participants were 37.29 μg/m^3 for PM_{10} and 19.32 ppb for NO_2. For pregnancy, they were 44.89 μg/m^3 for PM_{10} and 23.18 ppb for NO_2. For NeuroSmog, correlations between the prenatal and current exposures and NO_2 - PM_{10} exposure correlations were all moderate (Fig. 1B). Note that for both study populations, pollutant concentrations have decreased between the time when the children were *in utero* and the time when the children were scanned.

In the ABCD study analytical sample, the mean air pollution exposure was significantly lower than the United States Environmental Protection Agency primary standard for $PM_{2.5}$ (9 μg/m^3; $p < 2 \times 10^{-16}$) and for NO_2 (53 ppb; $p < 2 \times 10^{-16}$). The World Health Organization (WHO) recommended annual standard for $PM_{2.5}$ (5 μg/m^3) was significantly lower than the mean air pollution exposure in the ABCD study analytical sample ($p < 2 \times 10^{-16}$) and the Neurosmog analytical sample ($p < 2 \times 10^{-16}$). Similarly, the WHO recommended annual standard for NO_2 (5.32 ppb) was significantly lower than the mean air pollution exposure in the ABCD study analytical sample ($p < 2 \times 10^{-16}$) and the Neurosmog analytical sample ($p < 2 \times 10^{-16}$).

3.2. Effects of exposure to air pollution on T1w/T2w ratios

Effect estimates of current particulate matter (PM) exposure (μg/m^3) and NO_2 (ppb) on cortical myelin T1w/T2w ratios in the ABCD and NeuroSmog samples are presented in Figs. 2–3. After FDR correction for multiple comparisons, NO_2 and PM were not significantly associated with cortical myelin content in either study sample, except for an association between PM_{10} and lower myelin content in the left precuneus in NeuroSmog (FDR-corrected $p = 0.022$) (Fig. 2). This effect size was small and corresponded to a 0.05 % decrease in myelin content for each additional 1 μg/m^3 of PM_{10} concentration. For prenatal exposure, like current exposure, no significant effects were seen for PM (Figure A5) or NO_2 (Figure A6) on T1w/T2w cortical myelin estimates in either ABCD or NeuroSmog.

In our primary a priori models examining NO_2 and PM, we did, however, observe main effects of age and sex for most T1w/T2w myelin measures after FDR correction. In ABCD, age effects had the expected (positive) relationship between age and myelin content (Fig. 4, Figure A7). A significant age effect was observed for 41/68 ROIs in the $PM_{2.5}$ models and 44/68 ROI in the NO_2 models. In NeuroSmog, a significant age effect was observed in 6/68 ROIs in the NO_2 models and in 6/68 ROIs in the PM_{10} models.

In terms of sex effects, in ABCD, 66/68 ROI in the NO_2 and $PM_{2.5}$ models showed females had less cortical myelin, as indexed by larger T1w/T2w ratios, as compared to males (Fig. 5, Figure A8). For NeuroSmog, a significant sex effect was observed for 9/68 ROIs and 6/68 ROIs in PM_{10} and NO_2 models, respectively. Their direction was also more variable, with some regions showing more cortical myelin in one sex, and some in another (Fig. 5). Age and sex parameter estimates were on average larger in the ABCD Study compared to NeuroSmog.

Table 2
Sample characteristics of the ABCD study and NeuroSmog analytical samples.

ABCD analytical sample		NeuroSmog analytical sample	
Number of subjects	2021	Number of subjects	577
Age (years)		Age (years)	
Mean (SD)	9.93 (0.62)	Mean (SD)	11.57 (0.81)
Range	8.9–11.0	Range	10.6–14.0
Missing	0	Missing	16
Sex at birth		Sex at birth	
Female	928 (45.9 %)	Female	263 (43.8 %)
Male	1093 (54.1 %)	Male	338 (56.2 %)
Missing	0	Missing	1
Race/ethnicity		Race/ethnicity	
Asian/Other*	220 (10.9 %)	Asian/Other	0 (0.0 %)
Black	287 (14.2 %)	Black	0 (0.0 %)
Hispanic	400 (19.8 %)	Hispanic	0 (0.0 %)
White	1114 (55.1 %)	White	602 (100 %)
Caregiver education level^		Caregiver education level^	
< HS Diploma	61 (3.0 %)	< HS Diploma or Vocational School	109 (18.1 %)
HS Diploma/GED	186 (9.2 %)	HS Diploma or Some	240
Some College	542 (26.8 %)	College	(39.9 %)
Bachelor Degree	545 (27.0 %)	Bachelor Degree or higher	248
Post Graduate Degree	687 (34.0 %)		(41.2 %)
Missing	0	Missing	5
Site adjusted income[§]		Site adjusted income[§]	
Income group 1	684 (33.8 %)	Very bad or bad	55 (10.1 %)
Income group 2	539 (26.7 %)	Striving	87 (15.9 %)
Income group 3	644 (31.9 %)	Good	337 (61.6 %)
		Very good	102 (18.6 %)
Don't know/ Refused	154 (7.6 %)	Don't know/Refused	21 (3.8 %)
Puberty		Puberty	
Pre Puberty	1032 (51.1 %)	Pre Puberty	NA
Early Puberty	495 (24.5 %)	Early Puberty	NA
Mid Puberty	468 (23.2 %)	Mid Puberty	NA
Late/Post Puberty	26 (1.3 %)	Late/Post Puberty	NA
Handedness		Handedness	
Right	1629 (80.6 %)	Right	550 (91.4 %)
Left	141 (7.0 %)	Left	45 (7.5 %)
Mixed	251 (12.4 %)	Mixed	7 (1.2 %)
ADHD status[†]		ADHD	
No	1693 (83.8 %)	No	437 (72.6 %)
Yes	328 (16.2 %)	Yes	165 (27.4 %)

Participants in the ABCD Siemens scanner sample were selected to match the exclusionary criteria for NeuroSmog and had structural MRI data collected from a Siemens scanner. Values reflect numbers (percentage) unless otherwise noted.
*The "Other" race/ethnicity category includes subjects who were parent-identified as American Indian/Native American, Alaska Native, Native Hawaiian, Guamanian, Samoan, Other Pacific Islander, Asian Indian, Chinese, Filipino, Japanese, Korean, Vietnamese, Other Asian, or Other Race.
^Note, ABCD study analysis reports on highest parental education obtained whereas NeuroSmog reports on lowest parental education obtained. See Methods for rationale and additional information.
[§]Income group 1 indicates participants whose total combined income is $91,000 to $820 below their study's site median income. Income group 2 indicates participants whose total combined income is $2,760 to $78,500 above their study's site median income. Income group 3 indicates participants whose total combined income is $81,000 to $154,000 above their study's site median income.
[†]ADHD (attention-deficit/hyperactivity disorder) was considered present if adolescents had unspecified ADHD, present ADHD, or ADHD in partial remission – as determined by the parent-report Kiddie Schedule for Affective Disorders and Schizophrenia – or if adolescents were taking amphetamine, methylphenidate, or nonstimulant ADHD medication. The latter decision was made based on

reports of ADHD underdiagnosis in the ABCD study.
Abbreviations: ABCD, Adolescent Brain and Cognitive Development; HS, High School; GED, General Educational Development.

Additionally, no effects were found for ADHD status in both ABCD and NeuroSmog studies.

3.2.1. Sensitivity analyses in ABCD using data from all MRI scanners

Based on previous reports of scanner differences in brain outcomes (Dudley et al., 2023), we found noticeable differences in T1w/T2w cortical myelin estimates. Differences in myelin estimates were most pronounced for Philips scanners (Figure A9; median: 0.28; mean: 0.42; IQR: 0.18), which gave radically lower T1w/T2w values as compared to Siemens (median: 1.71; mean: 1.76; IQR: 0.32) and GE scanners (median: 2.18; mean: 2.36; IQR: 0.52). Of note, sample demographics and air pollution estimates also notably differ by study site, and thus, by scanner manufacturer in those with usable T1w/T2w myelin ratios (Table A3).

A sensitivity analysis using data from all MRI scanners revealed widespread associations between childhood NO_2 exposure and cortical myelin for all 68 ROIs (Figure A10). A similar pattern was seen with prenatal NO_2 exposure albeit this did not survive multiple comparison correction (Figure A11). However, the widespread age and sex effects previously found in the ABCD study sample became totally absent in this larger study sample (data not shown).

4. Discussion

In this paper, we investigated the impact of particulate matter (PM) and nitrogen dioxide (NO_2) on T1w/T2w ratios, a proxy measure for cortical myelin, in school-age children from two independent study samples. Using Siemens only data, we failed to find associations between PM or NO_2 and T1w/T2w ratio in participants aged 9–13 years, apart from a negative association between PM_{10} and myelin content in the precuneus in the NeuroSmog sample. Although they were not the focus of this study, we did observe the expected widespread effects of age on myelin content, with older children having higher myelin content. We also found notable sex differences, albeit directionality of effects varied in the two samples.

Subsequently, in a sensitivity analysis on a larger ABCD study subsample that included data from GE and Philips scanners, we found an NO_2 effect on myelin T1w/T2w ratio estimates across all cortical regions examined. Worryingly, however, both age and sex effects disappeared in that larger sub-sample. The secondary sensitivity models used there attempted to statistically adjust for confounding of sociodemographic and scanner effects (i.e., fixed effects) by site (i.e., random effect). However, it is still possible that the findings of the sensitivity analysis might stem from residual confounding caused by a combination of sociodemographic differences (in SES, race/ethnicity, and/or air pollution exposure, see Figure A4 and Table A3) with notably discrepant T1w/T2w measures coming from the three scanner types (see Figure A9). Below we discuss these findings in the context of the larger literature.

4.1. T1w/T2w ratio, myelin, age and sex

Neither T1w nor T2w images are direct measures of cortical myelin, their ratio merely correlates with myelin content. The relationship of T1w/T2w ratio to ex vivo cortical myelin measures is indirect: T1w/T2w ratio measures correlate with quantitative relaxometry measurements like R1 and R2 * maps (Shams et al., 2019), which, in turn, have been shown to directly correlate with myelin content measured directly in post-mortem histological studies (Geyer et al., 2011). Childhood and adolescence are a time of complex changes in the cortex, including reductions in thickness and increases in the T1w/T2w ratio myelin estimates. These patterns are typically observed from ages 1–24 years-old (Amlien et al., 2016; Khundrakpam et al., 2019; Tamnes et al., 2017). These changes tend to occur at different speeds based on age and in varying brain regions. Specifically, Deoni et al. (2015) found logarithmic increases in myelin water volume fraction in children aged 1–6 years, while Whitaker et al. (2016) demonstrated linear cortical myelin growth in participants aged 14–24 years, suggesting distinct myelination trajectories during these developmental periods. These effects also differ by brain region: a study based on the HCP Developmental sample (ages 8–21 years) found that in sensorimotor areas, T1w/T2w ratios are high in early childhood, increase rapidly, then decrease in later ages (18–21 years), whereas in intermediate multimodal areas, T1w/T2w ratios begin at intermediate levels and increase linearly at an intermediate pace (Baum et al., 2022). In multimodal/paralimbic association areas, T1w/T2w ratios start low and increase linearly at the slowest pace (Baum et al., 2022). Most of these previous developmental studies did not report sex effects. In our study, we found significant main effects of

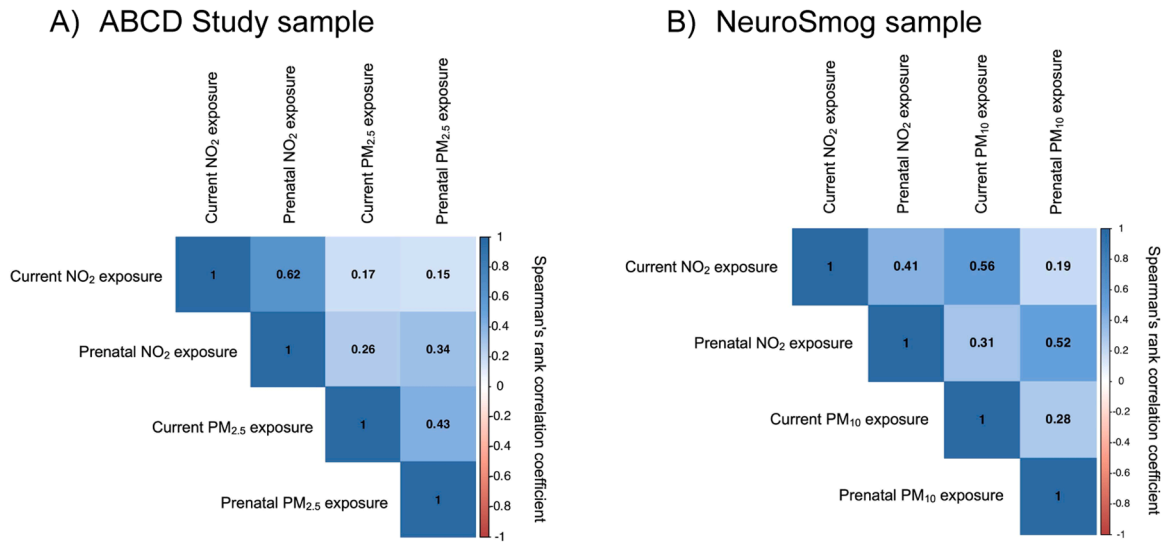

Fig. 1. Air pollution exposure correlations in the ABCD Study analytical sample and NeuroSmog cohort. Spearman's rank correlation coefficients for the associations between the air pollution variables within the ABCD study Siemens only analytical sample (N = 2,021) and NeuroSmog analytical sample (N = 577). Air pollution data for particulate matter (PM) and nitrogen dioxide (NO_2) included prenatal estimates (averaged across 9 months of pregnancy based on the child's birthdate) and estimates from the initial year of the studies (2016 for ABCD, and 2018 for NeuroSmog). Abbreviations: ABCD, Adolescent Brain and Cognitive Development.

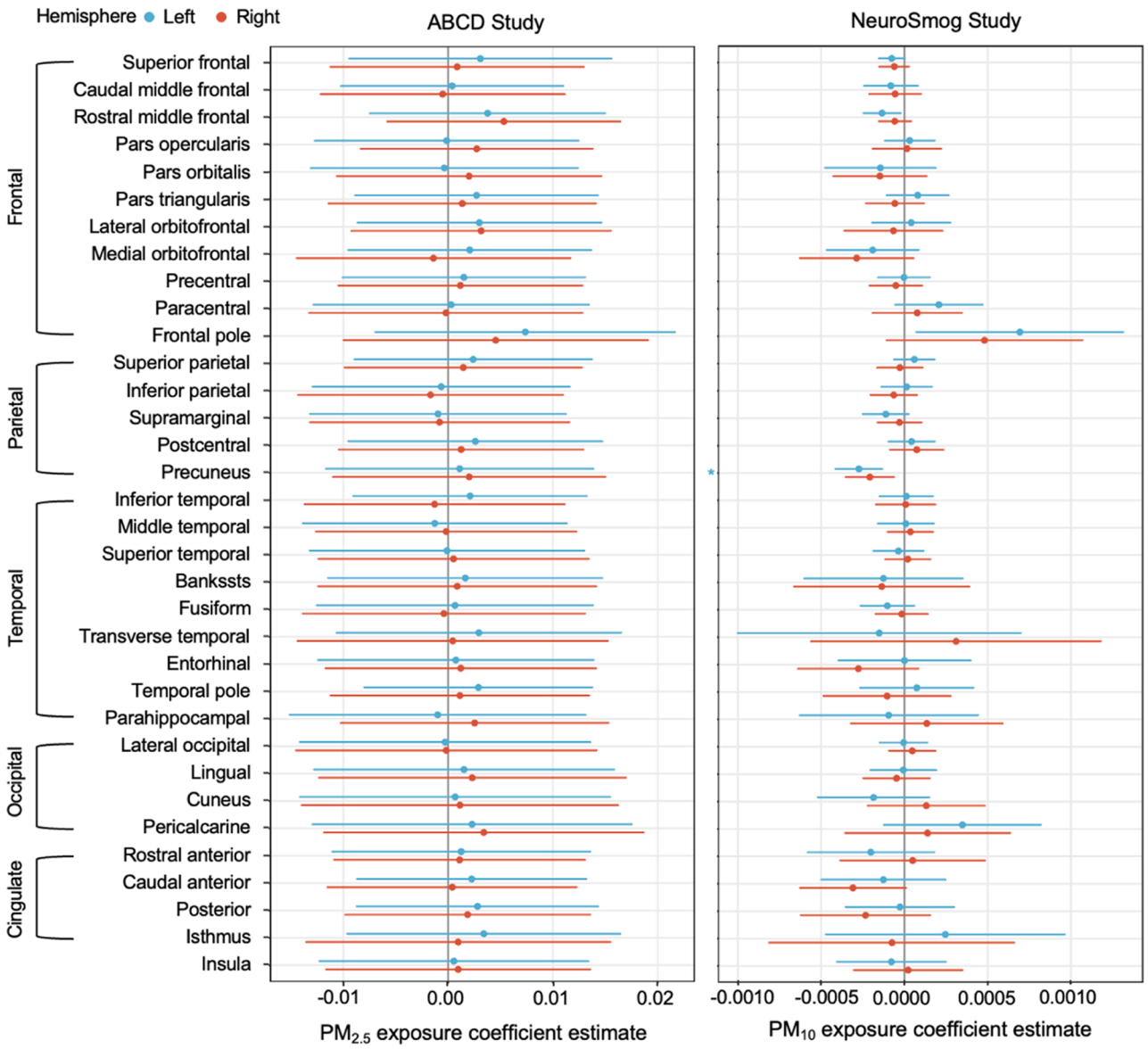

Fig. 2. Current particulate matter (PM) exposure (μg/m^3) effect estimates on cortical myelin T1w/T2w ratios in the ABCD Study (Siemens only) and NeuroSmog analytical samples. Unstandardized beta coefficients (95 % confidence interval) for $PM_{2.5}$ and PM_{10} exposure effects associated with hemisphere-specific cortical myelin T1w/T2w ratios in the ABCD Study Siemens scanner sample (n = 2021) and NeuroSmog analytical sample (n = 577), respectively. Results are presented with '* ' for passing False Discovery Rate correction ($p < 0.05$), where asterisks are colored according to hemisphere. Note the x-axis scale differences between the plots. Abbreviations: ABCD, Adolescent Brain and Cognitive Development; Bankssts, Banks of the superior temporal sulcus.

both age and sex in both the Siemens only ABCD and NeuroSmog samples, despite a relatively narrow age range. In particular, similar to other studies (Baum et al., 2022), age effects in both our study samples show increases of T1w/T2w cortical myelin estimates. Regarding sex effects, they were partially divergent between our two study samples, with ABCD showing higher T1w/T2w myelin ratios in males (male > female) and with NeuroSmog showing bidirectional effects (male > female and male < female) depending on brain region. Future studies are needed to clarify the age and sex effects observed in T1w/T2w ratios during childhood and adolescence. These studies must also account for scanner acquisition methods to accurately capture normative neurodevelopmental milestones and trajectories.

4.2. Possible interpretations of air pollution effects on T1w/T2w ratio myelin estimates

The current study focused on associations between prenatal and current childhood PM and NO_2 exposures and T1w/T2w ratios measured between ages 9–13 years old using cross-sectional data from two study samples. Regardless of study sample, prenatal and current PM exposures were not found to be associated with T1w/T2w cortical myelin estimates. In terms of NO_2, our findings highlight that the relationship between childhood NO_2 exposure and T1w/T2w cortical myelin estimates at ages 9–10 years may be absent or highly dependent on the characteristics of the study sample.

The lack of the expected association between PM and T1w/T2w needs to be first considered in the context of both the window(s) of exposure and the timing of MRI assessment. In other words, it is still possible that PM exposure during development may contribute towards differences in cortical myelin as indexed by T1w/T2w mapping when examined at other windows of development. Myelination does not follow a linear trajectory, with large changes in myelination as measured by MRI from birth to 5 years old (Baum et al., 2022). Thus, it is possible that prenatal and current exposure may have differential

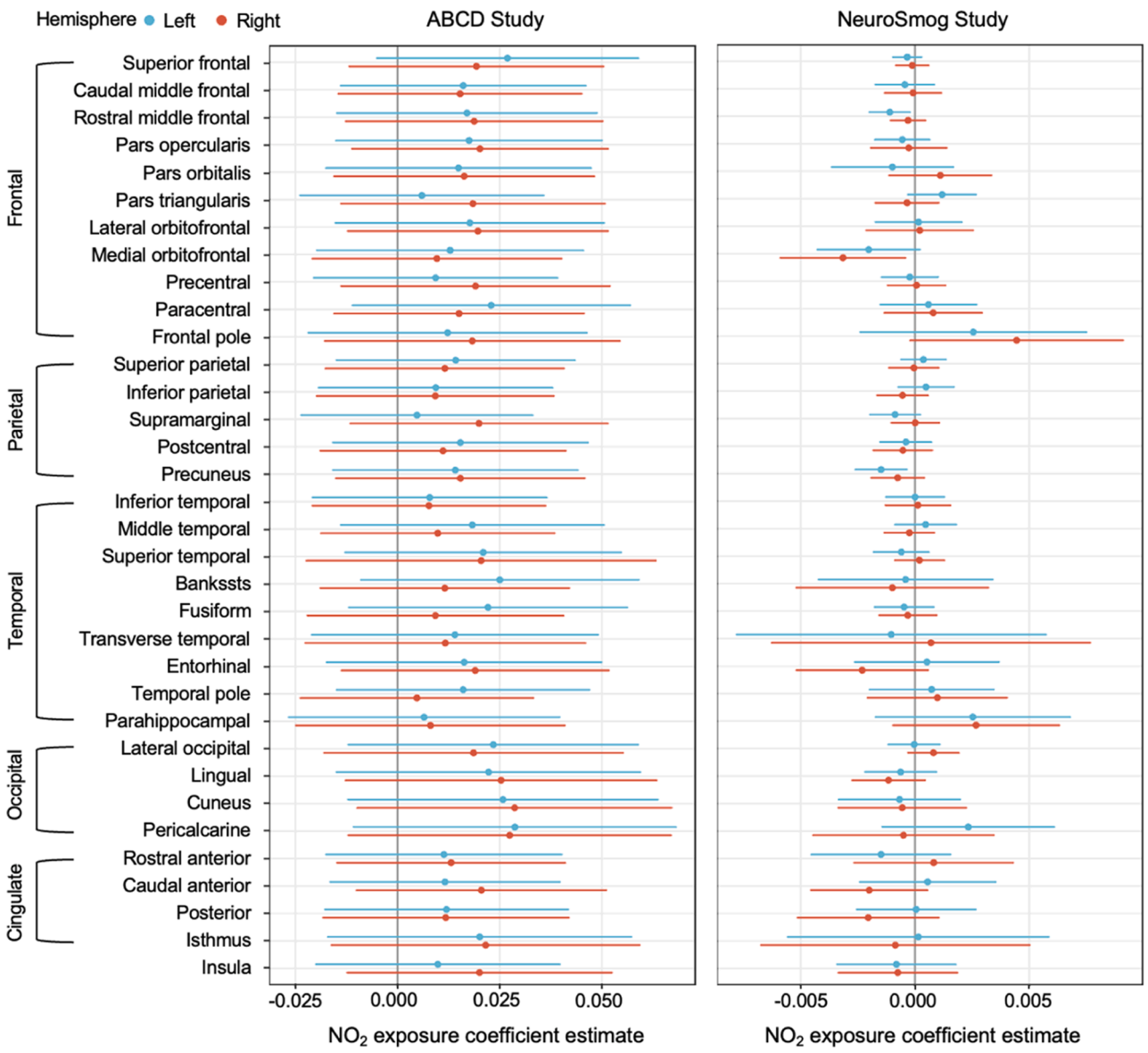

Fig. 3. Current nitrogen dioxide (NO_2) exposure (10 ppb) effect estimates on cortical myelin T1w/T2w ratios in the ABCD Study (Siemens only) and NeuroSmog analytical samples. Unstandardized beta coefficients (95 % confidence interval) for NO_2 exposure effects associated with hemisphere-specific cortical myelin T1w/T2w ratios in the ABCD Study Siemens scanner sample (n = 2021) and NeuroSmog analytical sample (n = 577), respectively. Results are presented with '* ' for passing False Discovery Rate correction ($p < 0.05$), where asterisks are colored according to hemisphere. Note the x-axis scale differences between the plots. Abbreviations: ABCD, Adolescent Brain and Cognitive Development; Bankssts, Banks of the superior temporal sulcus.

impacts on myelination earlier in development as compared to the age ranges studied here. In addition, the current study examined these associations cross-sectionally. Given the dynamic nature of cortical myelination noted by others, it is feasible that cumulative exposure(s) across the lifespan may lead to differences in cortical myelination that may emerge in later adolescence or early adulthood. Future, longitudinal studies that include both lifetime exposures and repeated MRI outcomes are necessary.

Another important factor is that PM contains complex mixtures of chemicals, the composition and physicochemical characteristics of which exhibit enormous variation by geography and other factors. For example, whereas components of PM may be dominated by anthropogenic emissions from combustion processes in one place, they may be dominated by compounds emitted from natural processes in another (Snider et al., 2016). Gross PM includes not only the larger PM_{10} and finer particles (i.e., $PM_{2.5}$), but also much finer particles, which display different properties and behaviors both in the environment and in the human body (Diociaiuti et al., 2001; Li et al., 2013), potentially leading to different human health effects (e.g. Schraufnagel, 2020; Schraufnagel et al., 2019). These effects include disruption to a variety of organ systems, including cardiovascular disease, respiratory disease and lung cancer (Chen and Hoek, 2020) through inflammation pathways (Arias-Pérez et al., 2020). All these can cause a variety of downstream effects relating to the endocrine system (e.g., Jung et al., 2018), immune system (e.g. Prunicki et al., 2021), and other systems. In the current study, we examined an overall exposure to PM. Yet, it is feasible that specific components in PM at different study sites – for example, combustion emissions from light industrial sites (e.g., furniture manufacturing/leather tanning in Nowy Targ in Neurosmog) or burning forest emissions in California and Oregon sites in ABCD – may have differential effects than other PM source locations in both NeuroSmog and the ABCD study. Future studies should consider assessing how specific sources and components found within total PM mass may influence cortical myelin, as previous studies have done with cortical microarchitecture

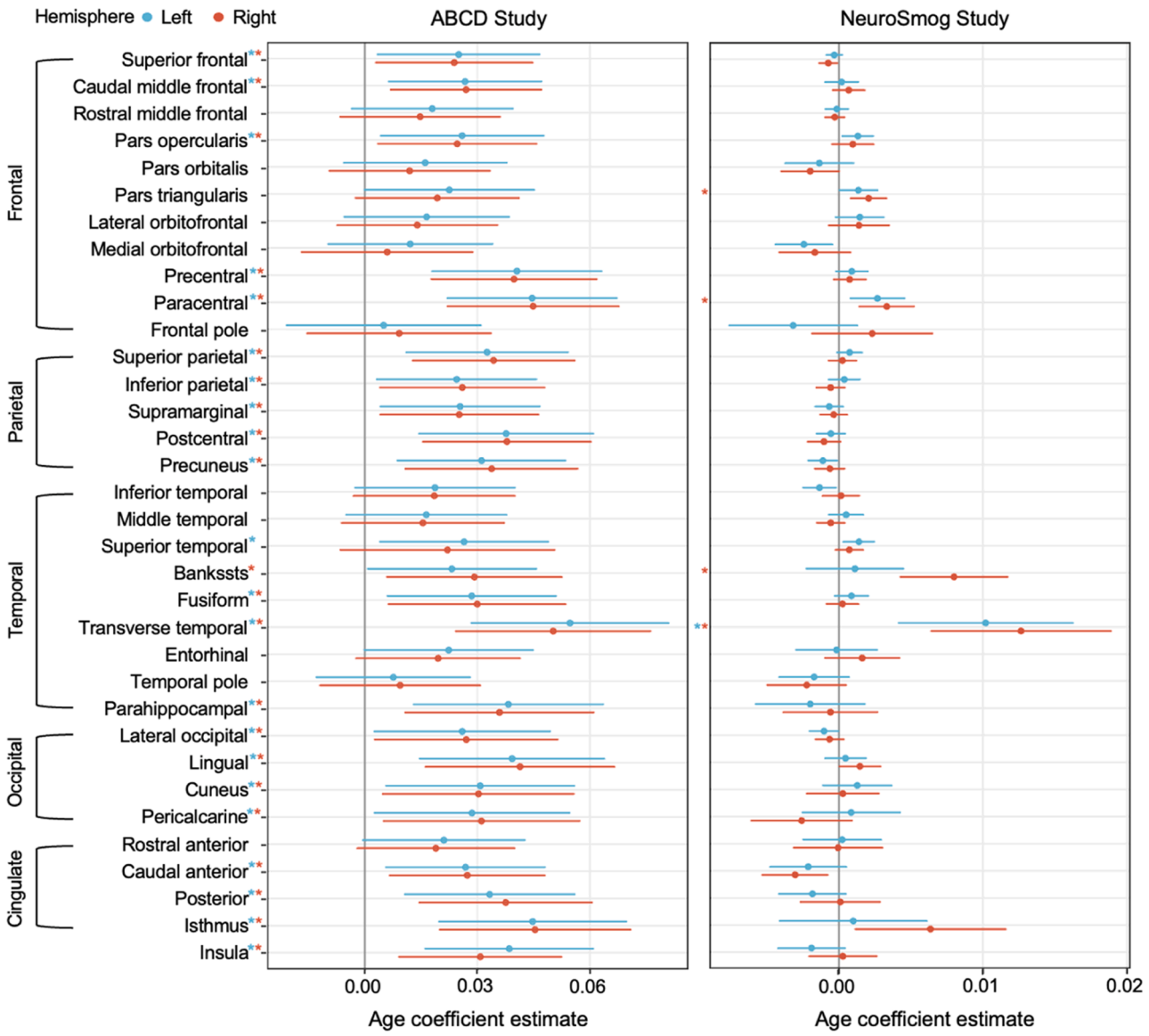

Fig. 4. Age (years) effect estimates on cortical myelin T1w/T2w ratios from NO_2 models in the ABCD Study (Siemens only) and NeuroSmog analytical samples. Unstandardized beta coefficients (95 % confidence interval) for age effects associated with hemisphere-specific cortical myelin T1w/T2w ratios in the ABCD Study Siemens scanner sample (n = 2021) and NeuroSmog analytical sample (n = 577), respectively. Results are presented with '* ' for passing False Discovery Rate correction ($p < 0.05$), where asterisks are colored according to hemisphere. Note the x-axis scale differences between the plots. Abbreviations: ABCD, Adolescent Brain and Cognitive Development; Bankssts, Banks of the superior temporal sulcus.

(Bottenhorn et al., 2024). Additionally, personal air pollution measurement devices, which measure air pollution exposure at home and other locations frequented by study participants, may have given more precise air pollution estimates and provided estimates for other pollutants of interest such as polyaromatic hydrocarbons. More accurate and precise measures of air pollution may reflect relationships between air pollution and myelin. However, these measurements are specific to relatively short periods (weeks/months) and may have been infeasible in larger-scale studies like ABCD and Neurosmog.

In terms of NO_2, we observed that prenatal and current NO_2 exposure levels were not associated with differences in T1w/T2w ratios neither in the NeuroSmog sample nor in the Siemens only ABCD sample. In contrast, the expanded ABCD sample including T1w/T2w ratios from all three MRI scanners showed a widespread, global negative association between current levels of NO_2 and myelin. Although the secondary analysis using all MRI scanner data had greater power to detect potential effects, it was unexpected to see that the larger sample found effects across all regions of interest and in the opposite direction as compared to the Siemens only subsample. Suspiciously, age and sex effects, both expected and very visible in the smaller ABCD sample, entirely disappeared in this larger sample.

Coupled together with the notably discrepant T1w/T2w ratios coming from different scanner types (Figure A9) and the notable differences in demographics inherent to each of the study sites within the ABCD cohort (Figure A4 and Table A3), it seems that NO_2 effects on myelin estimates are at best heterogenous and dependent on population characteristics. However, another possibility is that despite statistically controlling for scanner/site differences and demographic factors, residual confounding artifacts are responsible for the observed significant association noted in the larger sample. Overall, the association between annual NO_2 exposure and cortical myelination should be interpreted with caution, and it remains to be determined whether it can be replicated in an independent sample. Replication of findings on NO_2 exposure will be critical to determining whether T1w/T2w ratios are a sufficiently

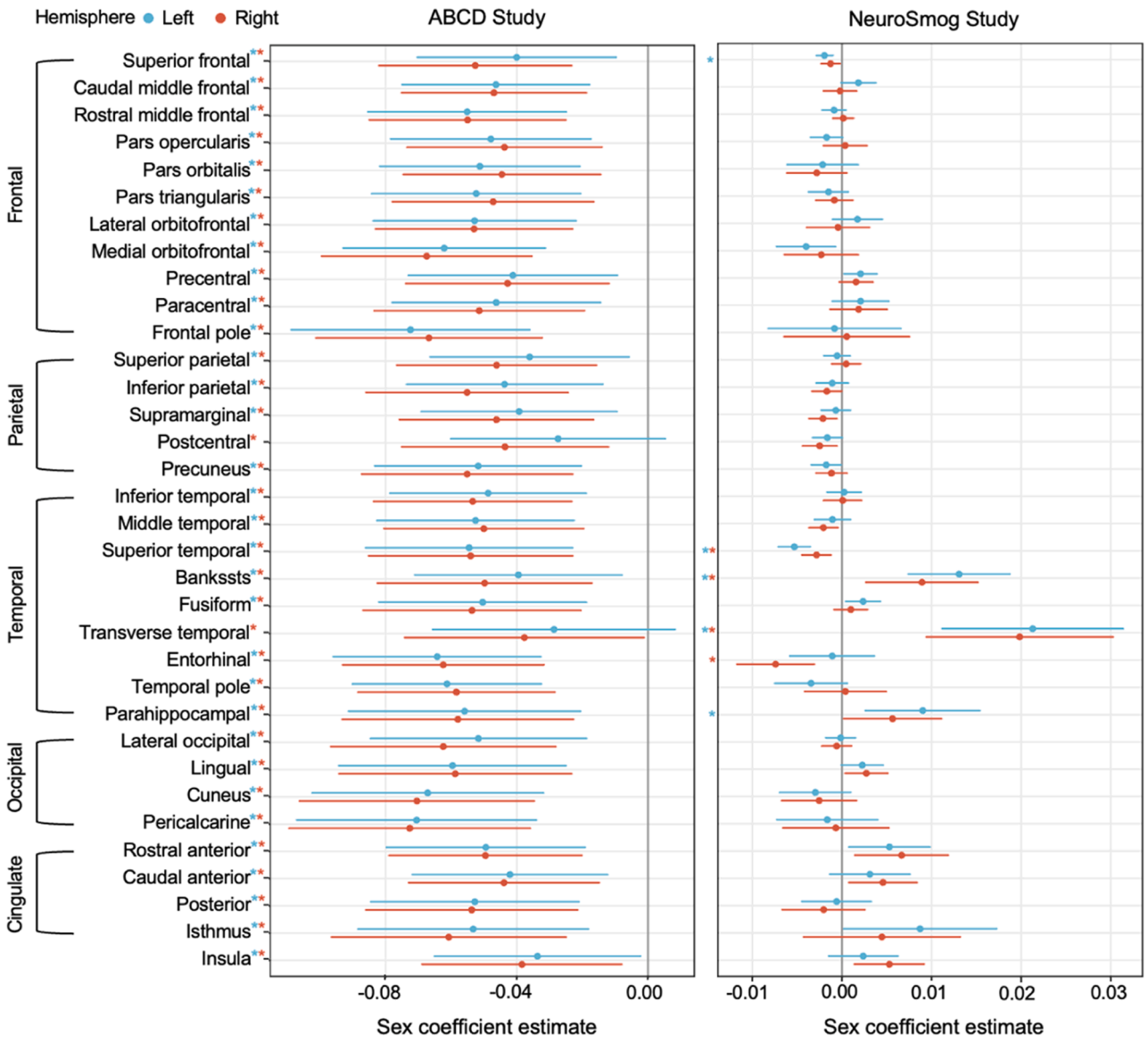

Fig. 5. Sex effect estimates on cortical myelin T1w/T2w ratios from NO_2 models in the ABCD Study (Siemens only) and NeuroSmog analytical samples. Unstandardized beta coefficients (95 % confidence interval) for sex effects associated with hemisphere-specific cortical myelin T1w/T2w ratios in the ABCD Study Siemens scanner sample (n = 2021) and NeuroSmog analytical sample (n = 577), respectively. Results are presented with '* ' for passing False Discovery Rate correction ($p < 0.05$), where asterisks are colored according to hemisphere. The reference category for sex is male. Note the x-axis scale differences between the plots. Abbreviations: ABCD, Adolescent Brain and Cognitive Development; Bankssts, Banks of the superior temporal sulcus.

sensitive MRI metric to detect the neurotoxic effects of air pollution on the brain during childhood and adolescence.

Further research is also needed to explore whether T1w/T2w ratios might serve as a useful marker for studying the neurological impacts of ambient pollution across other stages of the lifespan. Notably, alternative MRI techniques, such as Diffusion Weighted Imaging, which focus on white matter microstructure, may prove more sensitive for detecting the neurotoxic effects of air pollution during this developmental period. Indeed, previous studies using a biophysical model of diffusion, known as restriction spectrum imaging (RSI), have found that one-year annual estimates of ambient pollutants (i.e., $PM_{2.5}$, NO_2, and O_3) are related to differences in intracellular gray matter and white matter microstructure at ages 9–10 years-old in the ABCD Study cohort (Burnor et al., 2021; Cotter et al., 2023; Sukumaran et al., 2023). This includes markers representing increases in axonal density, axonal caliber, myelination, as well as number and size of support cells. Similarly, exposure to combinations of air pollutants (i.e., NO_2, $PM_{2.5}$ and its components) was found to relate to decreased white matter tract integrity, as indexed by lower fractional anisotropy (FA) and higher mean diffusivity (MD) in two cohorts from Spain (Pujol et al., 2016) and the Netherlands (Binter et al., 2022; Kusters et al., 2024; Lubczyńska et al., 2020). A significant microstructural effect, albeit in the opposite direction (i.e., higher FA and lower MD), was also reported in a New York City birth cohort (Peterson et al., 2022). Future studies should thus explore the application of T1w/T2w ratios across different developmental periods, while also incorporating known diffusion metrics sensitive to microstructural changes, to provide a more comprehensive understanding of the neurotoxic effects of air pollution on white matter development.

5. Summary

Our findings suggest that prenatal and one-year annual exposure to PM and NO_2 long-term air pollution exposure is not associated with T1w/T2w ratio myelin estimates at ages 9–13 years in two cross-

sectional samples in the United States and Poland. While for NO_2, a discrepancy in findings among ABCD subsamples collected on different scanners/sites suggest that associations between NO_2 exposure and T1w/T2w myelin estimates could possibly exist in some contexts, these findings should be interpreted with caution.

This being said, our findings do not preclude the existence of links between PM, NO_2 and T1w/T2w ratio myelin measures at other period (s) of exposure and other period(s) of brain development. Finally, one should note that different PM emissions also contain different mixtures of multiple chemicals that may cause different disruptions – inflammatory, endocrine or other. Methods that take into account differences in sources of air pollution as well as longitudinal study designs might unveil effects not captured by the current study.

CRediT authorship contribution statement

Kaczmarek-Majer Katarzyna: Methodology, Investigation. **Degórska Anna:** Methodology, Investigation. **Skotak Krzysztof:** Methodology, Investigation. **Sitnik-Warchulska Katarzyna:** Investigation, Data curation. **Lipowska Małgorzata:** Investigation, Data curation, Conceptualization. **Szwed Marcin:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Grellier James:** Writing – review & editing, Conceptualization. **de Jesus Alethea V.:** Writing – review & editing, Writing – original draft, Visualization, Formal analysis. **Markevych Iana:** Writing – review & editing, Visualization, Data curation, Conceptualization. **Kossowski Bartosz:** Formal analysis, Data curation. **Herting Megan M.:** Writing – review & editing, Supervision, Funding acquisition, Conceptualization. **Ahmadi Hedyeh:** Writing – review & editing, Formal analysis. **Rutkowska Emilia:** Software, Formal analysis. **Mysak Yarema:** Data curation. **Baumbach Clemens:** Software, Data curation.

Declaration of Competing Interest

The authors declare no conflict of interest.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.dcn.2025.101538.

Data availability

The ABCD Study anonymized data including all assessment domains is released annually to the research community. Information on how to access ABCD data through the NIMH Data Archive (NDA) is available on the ABCD study data sharing webpage: http://abcdstudy.org/scientists_data_sharing.html. We welcome individual data inquiries for NeuroSmog data and engage to provide data on a case-to-case basis. Due to European Union General Data Protection Rules on sharing of sensitive data, NeuroSmog data sharing is possible on the basis of bilateral data sharing agreements ratified by the Jagiellonian University and the inquirer's institution. Please direct inquiries to m.szwed@uj.edu.pl. NeuroSmog data sharing guidelines are expected to be published online in 2025–26 at http://neurosmog.psychologia.uj.edu.pl/dla-naukowcow/for-scientists

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