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The Role of Physical Environmental Characteristics and Intellectual Disability in Conduct Problem Trajectories Across Childhood: A Population-Based Cohort Study

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Abstract:

Background:

The paucity of research investigating the role of the physical environment in the developmental progression of conduct problems and the potential moderating effects of intellectual disability (ID) is surprising, given the clinical relevance of elucidating environmental determinants of disruptive behaviours.

Aims:

To use data from a large UK cohort study to assess associations between physical environmental exposures, ID, and conduct problem trajectories.

Method:

The sample included 8,168 Millennium Cohort Study children (1.9% with ID). Multilevel growth curve modelling was used to examine the role of physical environment characteristics in the developmental trajectories of conduct problems after adjustments for ID status.

Results:

Exposure to external environmental domains was not associated with differences in children's conduct problems across development. Alternatively, internal aspects of the household environment: spatial density ($b = 0.40, p < .001$) and damp problems ($b = 0.14, p < .001$) were both significantly associated with increased trajectories. Various individual and familial covariates were positively associated with conduct problems over time, including: presence of ID ($b = 0.96, p < .001$), autism spectrum disorder ($b = 1.18, p < .001$), male sex ($b = 0.26, p < .001$), poverty ($b = 0.19, p < .001$), maternal depression ($b = 0.65, p < .001$), and non-nuclear family structure ($b = 0.35, p < .001$). Positive ID status appeared to moderate the effects of internal household spatial density, reporting a non-linear negative association with spatial density and conduct problems across development ($b = -1.08, p < .01$).

Conclusions

Our findings highlight the potential harmful consequences of poor internal residential conditions on children's development of disruptive behaviours.

Keywords: Intellectual Disability, Conduct Problems, Physical Environment, Millennium Cohort Study, Longitudinal Birth Cohort

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Introduction

Interest in the role of children's early physical environment on neurodevelopmental and socio-cognitive outcomes has increased in recent decades, evidenced by reviews examining its effects on well-being^{1,2,3}, mental health^{4,5}, and development⁶. Physical domains explored are diverse, ranging from nature exposure and meteorology, to architectural design. Previous studies have highlighted the valence of physical environmental aspects such as exposure to natural greenspaces⁷ and air particulate matter⁸ on childhood psychological and neurophysiological outcomes. Considering that conduct problems have been associated with a wide range of negative long-term consequences, ranging from high societal economic costs⁹, to reduced life satisfaction and disintegration of social connections¹⁰, it is surprising that there is a paucity of current literature examining the role of the physical environment in the developmental progression of childhood conduct problems. Moreover, it is yet to be examined whether the influence of the physical environment on conduct problem symptom trajectories is more severe for children with intellectual disability (ID) than typically developing children. The global prevalence of ID has been estimated at 1.37%¹¹, with frequency of reported comorbid challenging behaviors inconsistent, varying between 10-22%^{12,13,14,15,16} in population studies, and between 50 – 60% in observation studies^{17,18}. Additionally, children with ID have been shown to exhibit significantly more conduct problems than their typically developing peers¹⁹.

Physical environmental characteristics such as air pollution and urbanicity^{20,21} have been previously positively associated with conduct problems, whilst exposure to greenspaces has been negatively associated with these problems in both typically developing children^{22,23}, and children with autism spectrum disorder²⁴ (ASD). Theories explicating these associations increasingly cite (neuro)physiological paradigms, for example, neuroinflammation via direct exposure to air particulate matter in the brain²⁵, or nature exposure conveying protection (maintaining homeostasis via regulation of physiological stress and attentional resources) from potentially harmful physical environmental influences²⁶⁻²⁹. Although previous epidemiological work has explored the varied nature of conduct problems in children with cognitive difficulties³⁰, few systematic reviews examine environmental effects on the trajectories of these behaviours in children with ID and neurodevelopmental disorders (NDDs) more broadly. For these children, escape from external environmental stressors (negative reinforcement) may constitute a significant proportion of the antecedent motivation behind conduct

problems³¹. Additionally, possible limitations in ability to communicate distress and/or regulate their environment, may increase the salience of negative physical environmental characteristics in comparison to typically developing children^{14,15}. Considering that the impact of the physical environment may be more potent in early neurodevelopmental stages³², and that consequences of childhood conduct problems are severe and persistent across the lifespan³³⁻³⁹, this study has the potential to illuminate the roles of both the external and interior physical environment in the developmental trajectories of conduct problems in children with and without ID.

Method

Sample

The sample was drawn from the Millennium Cohort Study (MCS: <https://cls.ucl.ac.uk/cls-studies/millennium-cohort-study/>⁴⁰), a UK population-based longitudinal birth cohort study. We used data collected from participants at MCS waves 2, 3, 4 and 5, with children aged, 3, 5, 7, and 11 years, respectively. In total 19,243 families have participated in the MCS to date. The MCS employed a stratified sampling protocol which disproportionally recruited disadvantaged or ethnic minority families (see Plewis⁴¹). The analytic sample of this study (n = 8,168; 49% male) included children (singletons and first-born twins or triplets) with valid data across all included sweeps (2-5) on cognitive ability and the behavioral outcome measure (conduct problems).

Intellectual disability ascertainment

To identify children with intellectual disability, first we ran principal components analysis (PCA) on standardised cognitive ability assessments available at each wave to derive summary cognitive scores. Such synthesis of multiple well-validated assessments has been shown to produce an adequate metric capturing general cognitive ability⁴². Previous research has adopted PCA to ascertain ID frequency within the MCS⁴³⁻⁴⁸. These studies predominantly use PCA at sweep 4 (age 7) to identify children with ID, only using scores at sweep 3 (age 5) if children had missing cognitive information at sweep 4, and using parental reports of child special educational needs (SEN) as a proxy for ID, only if cognitive information was not available at any sweep.

Our strategy was divergent in several key regards: We used PCA across cognitive measures at all sweeps (2-5) to classify children, requiring children to score two standard deviations below the mean score for cognitive ability at two or more waves to be classified with ID status. Parental confirmation of SEN statement due to learning disability was also used to provide a positive ID classification for

children across sweeps. Children who received a SEN statement due to being classified as 'gifted', or 'high IQ' were not classified as ID but still included in the analytic sample. Our rationale for using a modified ID ascertainment methodology was the concern that classifying children based on cognitive performance at a singular time point may facilitate miss-identification. Additionally, as we are using cognitive scores as a proxy measure for ID and not direct clinical diagnostic information, we wanted to ensure that classification was accurate and stable over time.

The cognitive assessments used for PCA were: the Bracken School Readiness Assessment-Revised⁴⁹ (conceptual knowledge) (BSRA-R) and the BAS-II Naming Vocabulary test⁵⁰ (expressive language ability) at MCS wave 2 (child age 3). For MCS wave 3 (child age 5) cognition was assessed using the: BAS Naming Vocabulary, BAS Pattern Construction (spatial problem-solving ability) and BAS Picture Similarities (non-verbal reasoning). For MCS wave 4 (children aged 7) the age standardised pattern construction and word reading scores from the BAS-II were used as well as a shortened version of the Progress in Maths tests⁵¹ (National Foundation for education Research). At wave 5 (child age 11) cognitive ability was assessed with the BAS Verbal Similarities (verbal knowledge and reasoning capability) and the error scores on the spatial working memory (SWM) task (representative of executive functioning⁵²) from the Cambridge Neuropsychological Test Automated Battery⁵³ (CANTAB). Our ascertainment methodology resulted in a prevalence rate for ID of 1.9% (N = 155) in our analytic sample.

Conduct problems

Conduct problems at ages 3, 5, 7, and 11 years were measured using the parent-reported Strengths and Difficulties Questionnaire⁵⁴ (SDQ). In MCS, the conduct problems subscale of the SDQ has been shown to have satisfactory internal reliability with Cronbach's alphas ranging from 0.77 and 0.82 for ages 3 – 7 years⁵⁵, and 0.68 for children aged 11⁵⁶. In the analytic sample, Cronbach's alpha values ranged from 0.53 to 0.60 across assessments. Importantly, the SDQ has been shown to be comparably valid across populations with and without ID^{57,58}.

External physical environmental exposures

Greenspace was assessed in the MCS using ward-level greenspace data from the UK, estimated with data from the Generalized Land Use Database⁵⁹ (GLUD) and from the Coordination of information on the Environment⁶⁰ (CORINE; EEA). Regression models predicted GLUD percentage greenspace per

English ward which was adapted for use on the whole of the UK⁶¹ resulting in a linear greenspace metric reporting the deciles of percentage of greenspace within wards, with higher scores reflective of more greenspace. Information on children's *access to private garden space* was also available. Data on *pathogenic air particulate matter* in the UK was obtained from the Multiple Environmental Deprivation Index (MEDIX) and linked to children's residential addresses. Annual population weighted mean concentrations of nitrogen dioxide (NO₂) were taken between 1999 – 2003 from 2001 UK Census Areas Statistics (CAS) ward with annual means converted into deciles (higher scores representative of increased NO₂) before being linked to MCS waves⁶². Data from the National Office of Statistics (ONS) was used to assess *urbanicity or rurality* of children's residential geography⁶³ at each included wave.

Interior physical environmental exposures

Home spatial density was measured by extracting parent reported data on the total number of people residing in each household at each wave, divided by the total number of rooms. Parents reported information on *residential damp problems* per wave and this was transformed into a binary variable indicating presence or not of household damp.

Covariates

Adjustment for a variety of time-invariant and time-varying covariates were made in our analyses. Time-invariant covariates included *ethnicity* (classified as white or non-white due to the granularity of ethnicity captured within the MCS), *sex*, and *autism spectrum disorder (ASD) status*. ASD confirmation was obtained from parental and teacher questionnaires specifying whether the child has a previous ASD diagnosis across at least 2 assessment waves. The time-varying covariates measured at each assessment wave included, *age (in years)*, *poverty* (defined as household income - equivalised for household size - below 60% median national income obtained from the Organisation for Economic Co-operation and Development: OECD), *maternal depression*, and *family structure*. Maternal depression was measured using scores from the Kessler K6 depression scale⁶⁴, a 6-item Likert response questionnaire, measuring psychological distress related to symptoms of depression and anxiety. We used previously validated diagnostic cut-off values greater than 13 as representative of serious mental illness (SMI) and depression⁶⁴. Nuclear family structure was defined as the presence of both biological parents in the household.

Statistical analysis

We initially examined whether children in the analytic sample differed from the remaining MCS sample in terms of their sociodemographic characteristics, exposure to external and interior physical environmental exposures, and conduct problem levels. Next, growth curve models (GCM) were fitted as random intercept multilevel models (MLM) to investigate the impact of external (greenspace, access to private garden space, air pollution, and urbanicity) and interior (home spatial density and residential damp problems) environmental measures on the developmental trajectories of conduct problems. The MLM had three levels; repeatedly measured data points for individuals (Level 1) were treated as being nested within children (Level 2), which were in turn, nested within electoral wards (Level 3). Children's age was measured in years and was centred around the grand mean (6.71 years). Parameterised in this way, the effect of the predictor variables reflect mean differences at approximately age 7 years. We included an additional quadratic term for age in all models to explore the temporal linearity of conduct problems. Missing data in covariates were multiply imputed (MI) using chained equations (MICE) under the assumption that data is missing at random (MAR). Percentages of missing data ranged between 0.02 – 3.38% and a total of 20 datasets were imputed⁶⁵.

Our rationale for imputing data for covariates but not ID or our primary outcome was to avoid risking misclassifying typically developing children as intellectually disabled or exhibiting conduct problems, particularly considering the stringency of our non-time varying ID classification (i.e., requiring children to meet cognitive thresholds or parental-reported SEN diagnosis at two or more waves). In an exploratory manner we replicated the main analysis after imputing data for all variables in the model, including ID and SDQ conduct problem scores (N=19,243). In this model ID diagnosis was used as a time-varying variable as it was not possible to reliably impute the original model (B) using the original time-invariant ID diagnosis variable (=>2 waves) across the whole sample. These results (presented in the supplementary material Table S1) were comparable with the analysis presented below (Table 3) which are based on cohort members with complete available information on ID.

The main part of the analyses included two MLM: The first model (Model A) sought to examine the impact of external and interior physical environmental exposures on the trajectories of conduct problems after adjustments for ID status. The second model (Model B) examined the effects of the exposure variables on the outcome after adjustments for the time-varying (maternal psychological distress, socioeconomic disadvantage, family structure) and time-invariant cofounders (sex, ethnicity,

ASD status). For both models we calculated the associated intra-class correlation coefficients (ICC) to estimate the variance in the trajectories that is attributable to the clustering of the variables within higher levels (Levels 2 and 3). For the environmental exposure variables that emerged as significant in the fully adjusted analytic model (B) we estimated their associated E-values. E-values are a recently introduced measure of the hypothetical strength that an unmeasured confounder would need to have with both the exposure and outcome variables, conditional on the measured covariates, to fully explain the association between the exposure and the outcome^{66(p.8)}. Additional models including all two-way and three-way interaction terms between ID status, physical environmental measures, and age, were run to assess if the environmental influences on trajectories conduct problems varied dependent on children's ID classification (Supplementary tables S2 – S6). We applied a stringent p-value significance criterion ($* = p < 0.01$) to all models to adjust for multiple comparisons.

Because the MCS sample is disproportionately stratified due to the intentional over-sampling of sub-groups of the population all models were accordingly adjusted by including appropriate stratification variables. Each country had two strata: advantaged and disadvantaged. England had an additional one for areas with high percentage of ethnic minorities. We also adjusted for the clustered MCS sample at electoral ward level but also for attrition and non-response rates by incorporating study-specific weights into our models. All analyses were run in Stata SE 16.1⁶⁷.

Results

Bias analysis

First, we explored differences between children in the analytic (N = 8,168) and non-analytic sample (N = 11,075, Table 1). Children in the analytic sample lived in greener, less air polluted neighbourhoods and were more likely to have access to a private garden and to reside in more rural environments. The profile of their indoor home environments also differed, with residential homes being less spatially dense and with fewer damp problems. Significantly higher proportions of the non-analytic sample had income below the 60% medium national level, psychologically distressed mothers, ASD, lower cognitive scores. and lived in non-nuclear family structures.

“INSERT TABLE 1 HERE”

Physical environment and intellectual disability

Next, MLMs were run to examine the impact of physical environmental exposures and ID on trajectories of conduct problems across childhood. Model A included physical environmental exposures and ID status as the only explanatory variables. The ICC estimates suggested that individual child differences (Level 2) explained 51.5% of the variance in conduct problem trajectories, while commonalities of children residing within the same electoral wards (level 3) explained a mere 1.7% of the variation in the outcome. The unstandardised regression estimates of the model are summarised in Table 2. The results showed that children with ID ($b = 1.270$, $SE = 0.176$, $p < .001$), and those residing in houses with higher residential spatial density ($b = 0.356$, $SE = 0.062$, $p < .001$) and with household damp ($b = 0.163$, $SE = 0.033$, $p < .001$) had more conduct problems at around 7 years (the centred intercept). In contrast, none of the exterior environmental characteristics considered were significantly associated with the conduct problem trajectories. The regression estimates of the linear ($b = -0.217$, $SE = 0.005$, $p < .001$) and quadratic ($b = 0.052$, $SE = 0.001$, $p < .001$) age terms indicated that, across childhood, conduct problems decreased in a nonlinear fashion.

“INSERT TABLE 2 HERE”.

After adjustments for the covariates (Model B), the ICC values associated with clustering within the second and third level were reduced to 48.2% and 1.3%, respectively, suggesting that a significant amount of variance in conduct problems trajectories was still accounted for by individual-child differences. Table 3 summarises the results of this MLM. Overall, after adjustments for ASD status, ethnicity, sex, family structure, poverty, and maternal depression, children with ID still had more conduct problem scores at around age 7 ($b = 0.963$, $SE = 0.175$, $p < .001$). Presence of damp ($b = 0.143$, $SE = 0.031$, $p < .001$) and high spatial density ($b = 0.400$, $SE = 0.062$, $p < .001$) were significantly associated with elevated levels of conduct problem after adjustments for confounding. As in Model A, we did not find evidence for an impact of greenspace, NO₂ particulate matter, access to private gardens or urbanicity on conduct problem trajectories.

Regarding the effect of covariates, those classified as ASD ($b = 1.182$, $SE = 0.311$, $p < .001$), males ($b = 0.263$, $SE = 0.035$, $p < .001$), those whose mothers were psychologically distressed ($b = 0.650$, $SE =$

0.098, $p < .001$) and those living in non-nuclear (b = 0.346, SE = 0.036, $p < .001$) or poorer families (b = 0.190, SE = 0.037, $p < .001$) had, on average, more conduct problems at around 7 years.

We calculated E-values for those environmental exposures that emerged as statistically significant in the fully adjusted model. The association between presence of damp problems and conduct problem trajectories corresponded with an E-value of 1.369 (lower bound of 95% CI = 1.272). As spatial density was measured on a continuous scale, we used three distribution-based cut-offs (Lower quartile, median-split, and upper quartile of the distribution of spatial density in the sample) to reclassify spatial density as a categorical variable with scores above or below the distribution-based thresholds. The respective E-values for the three analyses were 1.404 (lower bound of 95%CI= 1.335), 1.493 (lower bound of 95%CI= 1.433), and 1.426 (lower bound of 95%CI=1.349). E-values in the range of 1.369 to 1.493 have been suggested to indicate magnitudes of unmeasured confounding which are unlikely to affect the associations found⁶⁸, suggestive that our model estimates are robust.

“INSERT TABLE 3 HERE”.

Interactions between physical environment and intellectual disability

To assess if exposure to physical environmental aspects was differentially associated with conduct problem trajectories for children with or without ID, additional GCM models, each including all 2-way interaction terms and the 3-way interaction term between environmental exposures, ID status and age, were run (results summarised in Tables S2-S6 in the supplementary material). ID did not appear to modify associations between external physical environment exposures and conduct problems. However, the effect of the interaction term between ID and spatial density on conduct problems was statistically significant (b = -1.075, SE = 0.346, $p < .01$), suggesting that positive ID status and increased household spatial density was associated with decreases in conduct problems over time (Table 4).

“INSERT TABLE 4 HERE”.

Sensitivity analysis

We investigated this association between spatial density, ID, and conduct problems further by conducting a sensitivity analysis to examine if the ethnicity of children with ID influenced conduct problem trajectories. We found that white ethnicity and increased spatial density interacted significantly to predict elevated SDQ conduct scores ($b = 0.419$, $SE = 0.113$, $p < .001$), however the 3-way interaction including ID did not ($b = 0.906$, $SE = 0.670$, $p = 0.176$), suggesting that whilst ethnicity (white / non-white) interacted with home crowdedness in predicting conduct problem development, ID diagnosis did not appear to moderate this relationship (Supplementary Table 7).

Discussion

The role of physical environmental exposures on children's socioemotional and neurodevelopment is currently not well understood, with previous calls for large scale multilevel interdisciplinary approaches to investigate its impacts being issued⁶⁹. In this study, we used data from UK's largest recent birth cohort to examine associations between various interior and external environmental aspects with conduct problems across childhood. Additionally, the role of ID as a potential moderator of these associations was examined to explore if associations between physical environmental characteristics and conduct problems were more salient for children with ID.

In this sample, external physical environmental domains were not associated with conduct problems, whilst both home crowdedness and damp were both related to increased conduct problem scores across development. In-line with previous literature, multiple socio-economic and familial covariates were also associated with behavioural problems, including: maternal psychological distress⁷⁰, poverty⁷¹, living within a non-nuclear family structure⁷², ethnicity⁷³, and male sex⁷⁴.

Our lack of significant associations between greenspace and conduct problems is incongruous with literature examining its effects on children's psychobehavioural outcomes. For example, some of the available studies suggest advantageous effects of access to greenspace on children's conduct problems⁷⁵⁻⁷⁹. Whilst our findings may be indicative of a true null association between greenspace exposure and child conduct problems, it may also be an artefact of the longitudinal nature of this sample, as previous studies have been primarily cross-sectional. It may also be attributable to inadequacies in the ward-level greenspace data available within the MCS, its reliance on geographical location and inability to assess the quality and frequency of children's greenspace

exposure has been highlighted previously⁸⁰. These are critical components of greenspace contact⁸¹ and their omission may draw into question the validity of this metric. Whilst children's access to private gardens did trend towards reductions in conduct problem trajectories in this study, it did not reach significance. Again, these results are discordant with previous studies which reported beneficial associations between access to private gardens and childhood conduct problems^{82,83}. Due to safety concerns, children's opportunity for autonomous play has diminished in recent decades⁸⁴⁻⁸⁷, potentially limiting their access to neighbourhood greenspaces and making private garden access a more salient measure of greenspace exposure.

Similarly, NO₂ exposure was not correlated with conduct problems in our sample. This mirrors previous findings which found no correlation between NO₂ particulate matter⁸⁸ or elemental carbon attributed to traffic pollution (ECAT) on childhood conduct problems. Nonetheless, the available evidence on the associations between air pollution and behavioural outcomes is mixed, with a previous study reporting²⁰ harmful associations between particulate matter less than 2.5 microns (PM_{2.5}) and NO₂ exposure, and increased risk of conduct disorders. This may suggest that cumulative exposure to air pollution grows during childhood, resulting in negative behavioural outcomes manifesting later in development, postliminary to the latest assessment used in our analysis (age 11). Recent research on the effects of ambient air pollution on children with ID - also using the MCS⁴⁷ - reported that they are between 17% - 33% more likely to live in areas of high air pollution dependent on the particulate matter exposure measured. Neurotoxicity related to air pollutants has also been linked to the aetiology of NDDs^{89,90}, and in a recent review of the current epidemiological evidence, Xu, Ha, and Basnet⁹¹ reported the harmful effects of exposure to various air pollutants (H₂S, ozone, PM₁₀, PM_{2.5}, and NO₂, among others) on neurodevelopment and psychobehavioural functioning in both adults and children. This is in accordance with recent research linking the presence of ultra-fine air particulate matter in the brain to neuroinflammatory and intrathecal inflammatory responses⁹², highlighting valid neurophysiological mechanisms for the aetiology of air pollution in childhood aggression.

We report no association between urbanicity or rurality of residential geography and conduct problems. Previous studies that have examined the relationship between urban/rural residence and aggression outcomes in children, reporting contradictory findings⁹³⁻⁹⁷. Urban environments have been shown to increase exposure to potentially harmful environmental influences (noise pollution, air

pollution, overcrowding) that may place additional strain on cognitive and self-regulatory processes⁹⁹, contributing to the aetiology of violent behaviour¹⁰⁰. Urban inhabitants also have limited access to greenspaces¹⁰¹, which have been purported to operate as a protective buffer against harmful environmental exposures^{102,103}, potentially compounding the negative effects of urban stimuli. It is worth considering if the possible therapeutic efficacy of greenspace exposure may be attributable to reduced exposure to harmful urban environmental stimuli¹⁰³ rather than a direct benefit of nature experience itself.

Presence of damp in children's homes was associated with increased SDQ conduct problems scores across development. Previous work using MCS data reported negative effects of children's indoor environment on cognitive and behavioural processes such as self-regulation and conduct problems^{104,105,80}. Increased damp can lead to toxic mould and poor air quality¹⁰⁶ to which children are especially vulnerable¹⁰⁷ and which may cause neuroinflammatory and/or neurotoxic responses^{108,109}. Financial circumstances and poor interior physical conditions of residential homes are highly correlated¹¹⁰, it is therefore likely that homes with multiple structural deficits exacerbate their negative effects on children's mental health. Future research should elucidate the direct toxic neurophysiological effects of household damp and mould and attempt to separate these from the influences of comorbid low socioeconomic status that so often accompanies occupation of deficient home environments.

Increased spatial density was associated with decreased conduct problem trajectories in children with a positive ID status. This finding is rather counterintuitive; previous research on spatial density and aggressive behaviour has primarily examined the influence of low-density vs high-density playground or classroom conditions in typically developing children, reporting disparate associations¹¹¹⁻¹¹⁴. One contemporary study¹¹⁵ reported that residential overcrowding was correlated with increased teacher reported externalising behaviors. Parents in crowded households have been shown to be less attentive and patient¹¹⁶, potentially indicative of a habituation effect leading to inaccurate caregiver reported conduct problems. The paucity of research exploring the impact of crowding on onset and/or maintenance of conduct problems in children with ID makes it impossible to embed our findings within the wider literature. This association between spatial density and conduct problems in children with ID may be attributable to several causes: first, this analysis was underpowered due to the relatively small sample of children with ID in this cohort. Second, households with an elevated density of family

members may convey additional benefits to children with ID, for example increased availability of support from proximal family members. Third, due to limited availability of complex spatial density metrics within the MCS, we adopted a one-dimensional measure of parental reported residential crowding, which may fail to capture the whole range of the spatial dynamics in the home. Finally, children with ID may spend a higher proportion of time in indoor environments due to the additional logistical complexity of undertaking activities outside of the home. This is especially relevant given contextual circumstances related to the COVID-19 pandemic pertaining to the additional risks and difficulties of complying to infection control measures¹¹⁷.

Strengths and limitations

To our knowledge, this is the first study exploring the influences of children's physical environment on conduct problem trajectories whilst examining moderating effects of ID diagnosis. A strength of this work is the large and diverse sample size facilitated by the MCS, even after exclusion of participants due to missing data. The longitudinal nature of this study also allows the examination of conduct problems across children's early neurodevelopmental periods.

Several limitations are also present in the current study, and the results should be interpreted with these caveats in mind. One is the dependence on the accuracy of parental reported SDQ conduct scores, a metric likely to contain inherent biases²². Another is the stringency in how we derived our ID classification, which may have resulted in under-ascertainment, however due to inability to certify clinical ID diagnosis this was deemed appropriate, and the resulting ID prevalence in our sample is comparative to previous reported global prevalence rates¹¹. It is also worth drawing attention to our bias analysis which evidenced consistent differences between our analytic and non-analytic sample across variables. The lack of diversity in physical environmental aspects that were included in the MCS data collection protocol may be considered a limitation of this work. Ambient road traffic noise for example, has been associated with elevated parent reported conduct problems in children^{118,119}, therefore the inability to include potential additional confounding physical environmental influences in analysis may limit the external validity of findings.

Conclusion

In conclusion, external physical environmental measures were not associated with childhood trajectories in this population; moreover, classification of ID did not appear to mediate these

relationships. Residential crowdedness and damp problems were both associated with increased conduct problems during development. Investigation of the moderating influence of ID status reported significant interaction effects for home crowding only, reporting a negative non-linear association with children's conduct problems trajectories. A dearth of research on the influence of spatial density on children with ID inhibits extrapolation of this finding, and caution in interpretation is warranted.

Additional individual, sociodemographic, and familial covariates such as: ID and ASD diagnosis, maternal psychological distress, poverty, living in a non-nuclear familial structure, and male sex were also significant predictors of increased conduct problems trajectories.

This work highlights the scarcity of contemporary research investigating the influence of physical environmental factors on the pathogenesis and progression of conduct problems in childhood NDDs. Previous work has outlined how NDD populations are disproportionately affected by health inequalities largely attributable to preventable environmental determinants¹²⁰. Despite calls to improve mental ill-health of children over the previous decade¹²¹, systemic study into the harmful effects of children's physical environment, and strategies to mitigate these preventable risks have not been conducted. Understanding the harmful and therapeutic influences of physical environmental domains can inform special educational policy and facilitate additional tools for clinicians and caregivers to alleviate a range of detrimental neurobehavioral outcomes, including conduct problems.

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Author contribution

Alister C. Baird: Original manuscript formation, Statistical analysis, writing.

Angela Hassiotis: Conceptualisation, Review and editing, Supervision.

Efstathios Papachristou: Statistical analysis, Supervision, Review and editing.

Eirini Flouri: Conceptualization, Methodology, Supervision, Review and editing.

All authors: Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Data availability

The data that support the findings of this study are available from the corresponding author, [A.B.], upon reasonable request and approval from relevant research authorities.

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Table and figures document

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Table1: Bias analysis

Table 1
Bias analysis between analytic and non-analytic samples.

Continuous variables	Analytic sample (n = 8168)		Non-analytic sample (n = 11,075)		Test
	n	M (SD)	n	M (SD)	F
SDQ conduct scores Sweep 2	8168	2.70 (2.01)	6602	2.99 (2.13)	75.05**
SDQ conduct scores Sweep 3	8168	1.40 (1.42)	6577	1.67 (1.61)	121.82**
SDQ conduct scores Sweep 4	8168	1.27 (1.47)	5304	1.59 (1.65)	132.99**
SDQ conduct scores Sweep 5	8168	1.29 (1.50)	4630	1.56 (1.69)	87.22**
Cognitive scores Sweep 2	8168	0.202 (1.204)	5389	-0.307 (1.279)	551.79**
Cognitive scores Sweep 3	8168	0.256 (1.197)	6695	-0.312 (1.344)	741.37**
Cognitive scores Sweep 4	8148	0.205 (1.310)	5124	-0.325 (1.398)	488.45**
Cognitive scores Sweep 5	8147	0.082 (1.006)	4329	-0.155 (1.065)	151.47**
Greenspace Sweep 2	8167	4.49 (2.67)	7422	4.07 (2.67)	206.44**
Greenspace Sweep 3	8167	4.78 (2.69)	7078	4.15 (2.70)	206.46**
Greenspace Sweep 4	8166	4.84 (2.69)	5689	4.17 (2.73)	202.55**
Greenspace Sweep 5	8164	4.91 (2.69)	5116	4.15 (2.72)	249.94**
Air pollution (NO ₂) Sweep 2	8167	6.15 (2.91)	7422	6.77 (3.06)	166.79**
Air pollution (NO ₂) Sweep 3	8167	6.66 (3.08)	7078	6.66 (3.08)	132.34**
Air pollution (NO ₂) Sweep 4	8166	6.05 (2.91)	5689	6.65 (3.09)	132.66**
Air pollution (NO ₂) Sweep 5	8164	5.99 (2.90)	5116	6.79 (3.06)	227.77**

Household spatial density Sweep 2	8166	0.72 (0.24)	7255	0.82 (0.32)	448.91**
Household spatial density Sweep 3	8157	0.73 (0.24)	6967	0.82 (0.31)	449.96**
Household spatial density Sweep 4	8154	0.74 (0.26)	5573	0.85 (0.32)	467.08**
Household spatial density Sweep 5	8120	0.88 (0.28)	4814	1.02 (0.35)	594.22**
Child Age (years) Sweep 2	8168	3.17 (0.19)	7422	3.20 (0.30)	55.81**
Child Age (years) Sweep 3	8168	5.28 (0.24)	7077	5.30 (0.26)	24.68**
Child Age (years) Sweep 4	8168	7.22 (0.24)	5676	7.26 (0.27)	87.82**
Child Age (years) Sweep 5	8168	11.16 (0.32)	5119	11.19 (0.34)	27.37**
Categorical variables	n	%	n	%	Chi ²
Access to private garden Sweep 2	8107	94.99	7198	89.25	176.85**
Access to private garden Sweep 3	8096	95.81	6051	90.38	167.82**
Access to private garden Sweep 4	8095	95.96	4497	90.95	132.24**
Access to private garden Sweep 5	8083	96.67	3736	91.70	135.45**
Urban residence Sweep 2	8167	77.01	7422	81.92	57.28**
Urban residence Sweep 3	8167	75.84	7078	81.63	75.43**
Urban residence Sweep 4	8166	75.25	5689	81.28	70.39**
Urban residence Sweep 5	8164	74.31	5116	81.43	90.10**
Household damp problems Sweep 2	8168	12.82	7279	16.06	32.91**
Household damp problems Sweep 3	8165	11.87	6989	14.48	22.61**
Household damp problems Sweep 4	8166	13.38	5596	16.24	21.83**
Household damp problems Sweep 5	8142	16.20	4872	18.45	10.94**
Below poverty line Sweep 2	8151	25.09	7248	41.96	493.47**
Below poverty line Sweep 3	8164	25.12	6971	44.33	618.27**
Below poverty line Sweep 4	8165	22.79	5672	40.57	502.82**
Below poverty line Sweep 5	8168	16.12	5119	38.97	877.89**
Maternal psychological distress Sweep 2	7670	2.71	5920	4.53	32.57**
Maternal psychological distress Sweep 3	8070	2.86	6262	4.50	27.51**
Maternal psychological distress Sweep 4	8097	3.11	5066	4.46	16.20**
Maternal psychological distress Sweep 5	7732	4.45	3973	8.38	74.48**
Atypical family structure Sweep 2	8153	15.80	7323	25.00	203.16**
Atypical family structure Sweep 3	8164	20.22	7071	30.39	209.29**

Atypical family structure Sweep 4	8164	23.80	5683	33.71	163.86**
Atypical family structure Sweep 5	8164	30.56	5115	39.47	111.29**
Male	8168	48.98	11075	53.18	33.18**
Autism Spectrum Disorder	7958	0.44	7539	1.25	30.55**
Ethnicity White	8168	90.45	11050	75.61	698.22**
England-Advantaged	2698	33.03	2130	19.23	476.25**
England-Disadvantaged	1971	24.13	2834	25.59	5.34*
England-Ethnic Minority	557	6.82	2034	18.37	537.89**
Wales-Advantaged	381	4.66	451	4.07	3.99*
Wales-Disadvantaged	792	9.70	1136	10.26	1.64
Scotland-Advantaged	562	6.44	619	5.59	6.08*
Scotland-Disadvantaged	407	4.98	784	7.08	35.57**
Northern Ireland-Advantaged	350	4.29	373	3.37	10.93**
Northern Ireland-Disadvantaged	486	5.95	714	6.45	1.99

Note. *p < .05, **p < .01

Table 2: Model A (Minimally adjusted)Table 2: Model A (minimally adjusted GCM predicting SDQ conduct scores, $N = 8168$).

Fixed effects	Coefficient (SE)	95% CI
Age	-0.217 (0.005)**	[-0.226, -0.208]
Age ²	0.052 (0.001)**	[0.049, 0.054]
Intellectual disability	1.270 (0.176)**	[0.925, 1.614]
Greenspace (ward decile)	-0.003 (0.009)	[-0.021, 0.015]
Air pollution (NO ₂)	-0.021 (0.010)	[-0.040, -0.002]
Access to private garden	-0.236 (0.146)	[-0.523, 0.051]
Urban residence	0.107 (0.057)	[-0.005, 0.219]
Home spatial density	0.356 (0.062)**	[0.234, 0.477]
Damp / condensation	0.163 (0.033)**	[0.098, 0.227]
Constant	1.148 (0.191)**	[0.774, 1.523]
Random effects	Estimate (SE)	95% CI
Level 3 (ward-level)		
Intercept variance	0.218 (0.025)	[0.174, 0.273]
Level 2 (Child level)		
Slope (age) variance	0.140 (0.005)	[0.131, 0.149]
Intercept variance	1.120 (0.019)	[1.083, 1.159]
Intercept-slope variance	-0.329 (0.036)	[-0.398, -0.256]

Note. Age was measured in years and grand mean centered (6.71 yrs).
For fixed effects: * $p < .01$, ** $p < .001$

Table 3: Model B (Fully adjusted)Table 3: Model B (fully adjusted GCM predicting SDQ conduct scores, $N = 8168$).

Fixed effects	Coefficient (SE)	95% CI
Age	-0.224 (.005)**	[-0.234, -0.215]
Age ²	0.052 (.001)**	[0.049, 0.054]
Intellectual disability	0.963 (.175)**	[0.620, 1.307]
Greenspace (ward decile)	-0.000 (0.009)	[-0.018, 0.017]
Air pollution (NO ₂)	-0.014 (0.009)	[-0.031, 0.004]
Access to private garden	-0.164 (0.130)	[-0.419, 0.091]
Urban residence	0.071 (0.056)	[-0.039, 0.181]
Home spatial density	0.400 (0.062)**	[0.277, 0.523]
Damp / condensation	0.143 (0.031)**	[0.083, 0.203]
Maternal psychological distress	0.650 (0.098)**	[0.458, 0.842]
Below poverty line	0.190 (0.037)**	[0.117, 0.262]
Atypical family structure	0.346 (0.036)**	[0.275, 0.416]
Male	0.263 (0.035)**	[0.195, 0.331]
Autism Spectrum Disorder	1.182 (0.311)**	[0.572, 1.793]
White ethnicity	0.170 (0.068)	[0.036, 0.304]
Constant	0.605 (0.181)*	[0.251, 0.959]
Random effects	Estimate (SE)	95% CI
Level 3 (ward-level)		
Intercept variance	0.176 (0.022)	[0.138, 0.224]
Level 2 (Child level)		
Slope (age) variance	0.139 (0.005)	[0.131, 0.148]
Intercept variance	1.058 (0.018)	[1.024, 1.094]
Intercept-slope variance	-0.356 (0.034)	[-0.420, -0.288]

Note. Age was measured in years and grand mean centered (6.71 yrs).
For fixed effects: * $p < .01$, ** $p < .001$

Table 4: spatial density x intellectual disability interaction model

Table 4: Spatial density x ID diagnosis growth curve interaction model predicting SDQ conduct scores (N = 8168).

Fixed effects	Coefficient (SE)	95% CI
Age	-0.195 (0.010)**	[-0.215, -0.170]
Age ²	0.053 (0.001)**	[0.050, 0.055]
ID diagnosis	1.882 (0.340)**	[1.118, 2.646]
Greenspace (ward decile)	-0.000, (0.009)	[-0.017, 0.017]
Air pollution (NO ₂)	-0.013 (0.009)	[-0.031, 0.004]
ID diagnosis x spatial density x Age	-0.055 (0.063)	[-0.178, 0.068]
Spatial density x Age	-0.040 (0.012)*	[-0.063, -0.017]
ID diagnosis x Age	0.123 (0.061)	[0.006, 0.251]
ID diagnosis x Spatial density	-1.075 (0.346)*	[-1.753, -0.397]
Urban residence	0.072 (0.056)	[-0.038, 0.182]
Access to private garden	-0.153 (0.130)	[-0.408, 0.102]
Home spatial density	0.460 (0.063)**	[0.257, 0.495]
Damp / condensation	0.145 (0.031)**	[0.085, 0.205]
Maternal psychological distress	0.654 (0.098)**	[0.461, 0.846]
Below poverty line	0.192 (0.037)**	[0.119, 0.264]
Atypical family structure	0.348 (0.036)**	[0.278, 0.419]
Male	0.263 (0.035)**	[0.195, 0.331]
Autism Spectrum Disorder	1.156 (0.312)**	[0.543, 1.768]
White ethnicity	0.165 (0.069)*	[0.031, 0.299]
Constant	0.549 (0.182)*	[0.193, 0.905]
Random effects	Estimate (SE)	95% CI
Level 3 (ward-level)		
Intercept variance	0.178 (0.022)	[0.139, 0.225]
Level 2 (Child level)		
Slope (age) variance	0.138 (0.005)	[0.129, 0.147]
Intercept variance	1.058 (0.018)	[1.023, 1.093]
Intercept-slope variance	-0.351 (0.034)	[-0.417, -0.283]

Note. Age was measured in years and grand mean centered (6.71 yrs).
For fixed effects: * $p < .01$, ** $p < .001$

Supplementary Table 1: Sensitivity analysis: ID as time-invariant (non-analytic sample)Table 1: Fully adjusted model across whole non-analytic sample ($N = 19,243$).

Fixed effects	Coefficient (SE)	95% CI
Age	-0.247 (0.005)**	[-0.256, -0.238]
Age ²	0.053 (0.001)**	[0.051, 0.056]
ID diagnosis	0.514 (0.064)**	[0.389, 0.638]
Greenspace (ward decile)	-0.010 (0.008)	[-0.025, 0.005]
Air pollution (NO ²)	-0.018 (0.009)	[-0.036, 0.001]
Damp	0.147 (0.028)**	[0.093, 0.201]
Urban	0.074 (0.046)	[-0.017, 0.165]
Access to garden	-0.133 (0.086)	[-0.302, 0.036]
Home spatial density	0.289 (0.046)**	[0.199, 0.380]
Maternal psychological distress	0.731 (0.068)**	[0.597, 0.864]
Below poverty line	0.273 (0.030)**	[0.216, 0.331]
Atypical family structure	0.088 (0.020)**	[0.049, 0.126]
Male	0.301 (0.027)**	[0.248, 0.355]
ASD	0.829 (0.157)**	[0.522, 1.137]
White ethnicity	0.143 (0.066)	[0.014, 0.273]
Constant	0.839 (0.141)**	[0.563, 1.116]
Random effects	Estimate (SE)	95% CI
Level 3 (ward-level)		
Intercept variance	0.197 (0.021)	[0.160, 0.244]
Level 2 (Child level)		
Slope (age) variance	0.145 (0.004)	[0.137, 0.154]
Intercept variance	1.132 (0.016)	[1.102, 1.163]
Intercept-slope variance	-0.336 (0.030)	[-0.393, -0.276]

Note. Age was measured in years and grand mean centered (6.71 yrs).
For fixed effects: * $p < .01$, ** $p < .001$

S2 – S6:**Supplementary Table 2: Greenspace interaction model**

Table 2: Fully adjusted greenspace model predicting SDQ conduct scores (N = 8168).

Fixed effects	Coefficient (SE)	95% CI
Age	-0.238 (0.008)**	[-0.253, -0.223]
Age ²	0.052 (0.001)**	[0.049, 0.054]
ID diagnosis	0.780 (0.327)	[0.140, 1.421]
Greenspace (ward decile)	-0.002 (0.009)	[-0.020, 0.015]
ID diagnosis x Greenspace x Age	0.007 (0.010)	[-0.013, 0.027]
Greenspace x Age	0.003 (0.001)	[0.000, 0.005]
ID diagnosis x Age	0.004 (0.050)	[-0.094, 0.102]
ID diagnosis x Greenspace	0.034 (0.059)	[-0.081, 0.150]
Air pollution (NO ²)	-0.014 (0.009)	[-0.031, 0.004]
Damp	0.144 (0.031)**	[0.084, 0.205]
Urban	0.073 (0.056)	[-0.037, 0.183]
Access to garden	-0.162 (0.131)	[-0.418, 0.094]
Home spatial density	0.401 (0.063)**	[0.279, 0.525]
Maternal psychological distress	0.650 (0.098)**	[0.457, 0.842]
Below poverty line	0.190 (0.037)**	[0.118, 0.263]
Atypical family structure	0.346 (0.040)**	[0.275, 0.416]
Male	0.263 (0.035)**	[0.195, 0.331]
ASD	1.193 (0.312)**	[0.581, 1.804]
White ethnicity	0.169 (0.069)	[0.033, 0.302]
Constant	0.614 (0.180)*	[0.261, 0.970]
Random effects	Estimate (SE)	95% CI
Level 3 (ward-level)		
Intercept variance	0.177 (0.022)	[0.139, 0.224]
Level 2 (Child level)		
Slope (age) variance	0.139 (0.005)	[0.130, 0.148]
Intercept variance	1.058 (0.018)	[1.024, 1.094]
Intercept-slope variance	-0.355 (0.034)	[-0.420, -0.287]

Note. Age was measured in years and grand mean centered (6.71 yrs).

For fixed effects: * $p < .01$, ** $p < .001$

Supplementary Table 3: NO₂ interaction model

Table 3: Fully adjusted greenspace model predicting SDQ conduct scores (N = 8168).

Fixed effects	Coefficient (SE)	95% CI
Age	-0.219 (0.007)**	[-0.234, -0.205]
Age ²	0.052 (0.001)**	[0.049, 0.054]
ID diagnosis	0.892 (0.303)*	[0.299, 1.485]
Greenspace (ward decile)	-0.000 (0.009)	[-0.018, 0.017]
Air pollution (NO ²)	-0.013 (0.009)	[-0.031, 0.005]
ID diagnosis x Air pollution (NO ²) x Age	0.009 (0.009)	[-0.008, 0.026]
Air pollution (NO ²) x Age	-0.001 (0.001)	[-0.003, 0.001]
ID diagnosis x Age	-0.027 (0.058)	[-0.142, 0.087]
ID diagnosis x Air pollution (NO ²)	0.006 (0.044)	[-0.081, 0.093]
Damp	0.144 (0.031)**	[0.084, 0.204]
Urban	0.072 (0.056)	[-0.038, 0.182]
Access to garden	-0.164 (0.130)	[-0.419, 0.091]
Home spatial density	0.400 (0.063)**	[0.276, 0.522]
Maternal psychological distress	0.651 (0.098)**	[0.459, 0.848]
Below poverty line	0.190 (0.037)**	[0.118, 0.263]
Atypical family structure	0.346 (0.036)**	[0.275, 0.417]
Male	0.263 (0.035)**	[0.195, 0.331]
ASD	1.175 (0.313)**	[0.561, 1.789]
White ethnicity	0.170 (0.068)	[0.037, 0.304]
Constant	0.602 (0.181)	[0.247, 0.967]
Random effects	Estimate (SE)	95% CI
Level 3 (ward-level)		
Intercept variance	0.176 (0.022)	[0.138, 224]
Level 2 (Child level)		
Slope (age) variance	0.139 (0.005)	[0.131, 0.148]
Intercept variance	1.058 (0.018)	[1.024, 1.094]
Intercept-slope variance	-0.356 (0.034)	[-0.420, -0.288]

Note. Age was measured in years and grand mean centered (6.71 yrs).

For fixed effects: * $p < .01$, ** $p < .001$

Supplementary Table 4: Garden access interaction model

Table 4: Fully adjusted greenspace model predicting SDQ conduct scores (N = 8168).

Fixed effects	Coefficient (SE)	95% CI
Age	-0.241 (0.017)**	[-0.274, -0.207]
Age ²	0.052 (0.001)**	[0.049, 0.054]
ID diagnosis	0.825 (0.496)	[-0.148, 1.798]
Greenspace (ward decile)	-0.000 (0.009)	[-0.148, 1.798]
Air pollution (NO ²)	-0.014 (0.009)	[-0.031, 0.004]
ID diagnosis x Access to garden x Age	-0.085 (0.117)	[-0.315, 0.145]
Access to garden x Age	0.016 (0.017)	[-0.016, 0.049]
ID diagnosis x Age	0.115 (0.114)	[-0.108, 0.339]
ID diagnosis x Access to garden	0.113 (0.493)	[-0.853, 1.079]
Access to garden	-0.168 (0.134)	[-0.430, 0.094]
Damp	0.144 (0.031)	[0.084, 0.205]
Urban	0.072 (0.056)	[-0.038, 0.182]
Home spatial density	0.400 (0.063)	[0.276, 0.523]
Maternal psychological distress	0.651 (0.068)**	[0.458, 0.844]
Below poverty line	0.189 (0.037)**	[0.116, 0.262]
Atypical family structure	0.346 (0.036)**	[0.276, 0.417]
Male	0.263 (0.035)**	[0.195, 0.331]
ASD	1.183 (0.311)**	[0.574, 1.793]
White ethnicity	0.170 (0.068)	[0.036, 0.304]
Constant	0.611 (0.182)*	[0.255, 0.968]
Random effects	Estimate (SE)	95% CI
Level 3 (ward-level)		
Intercept variance	0.176 (0.022)	[0.138, 0.224]
Level 2 (Child level)		
Slope (age) variance	0.139 (0.005)	[0.131, 0.148]
Intercept variance	1.058 (0.018)	[1.024, 1.094]
Intercept-slope variance	-0.355 (0.034)	[-0.420, -0.287]

Note. Age was measured in years and grand mean centered (6.71 yrs).
For fixed effects: * $p < .01$, ** $p < .001$

Supplementary Table 5: Urbanicity and Rurality interaction model

Table 5: Fully adjusted greenspace model predicting SDQ conduct scores (N = 8168).

Fixed effects	Coefficient (SE)	95% CI
Age	-0.218 (0.007)**	[-0.231, -0.205]
Age ²	0.052 (0.001)**	[0.049, 0.054]
ID diagnosis	1.230 (0.361)*	[0.523, 1.938]
Greenspace (ward decile)	-0.000 (0.009)	[-0.018, 0.017]
Air pollution (NO ²)	-0.013 (0.009)	[-0.031, 0.004]
ID diagnosis x Urban x Age	0.032 (0.074)	[-0.113, 0.178]
Urban x Age	-0.009 (0.008)	[-0.024, 0.006]
ID diagnosis x Age	0.007 (0.066)	[-0.123, 0.136]
ID diagnosis x Urban	-0.368 (0.386)	[-1.125, 0.390]
Urban	0.083 (0.057)	[-0.028, 0.194]
Damp	0.144 (0.031)**	[0.084, 0.204]
Access to garden	-0.163 (0.131)	[-0.419, 0.093]
Home spatial density	0.400 (0.063)**	[0.277, 0.523]
Maternal psychological distress	0.651 (0.098)**	[0.458, 0.843]
Below poverty line	0.190 (0.037)**	[0.117, 0.263]
Atypical family structure	0.345 (0.036)**	[0.275, 0.416]
Male	0.263 (0.035)**	[0.195, 0.331]
ASD	1.205 (0.313)**	[0.592, 1.818]
White ethnicity	0.169 (0.069)	[0.035, 0.303]
Constant	0.596 (0.183)*	[0.238, 0.953]
Random effects	Estimate (SE)	95% CI
Level 3 (ward-level)		
Intercept variance	0.177 (0.022)	[0.139, 0.224]
Level 2 (Child level)		
Slope (age) variance	0.139 (0.005)	[0.130, 0.148]
Intercept variance	1.058 (0.018)	[1.024, 1.094]
Intercept-slope variance	-0.356 (0.034)	[-0.420, -0.288]

Note. Age was measured in years and grand mean centered (6.71 yrs).

For fixed effects: * $p < .01$, ** $p < .001$

Supplementary Table 6: Damp interaction model

Table 6: Fully adjusted damp interaction model predicting SDQ conduct scores (N = 8168).

Fixed effects	Coefficient (SE)	95% CI
Age	-0.225 (0.005)**	[-0.235, -0.216]
Age ²	0.052 (0.001)**	[0.049, 0.054]
ID diagnosis	1.013 (0.177)**	[0.666, 1.361]
Greenspace (ward decile)	-0.000 (0.009)	[-0.018, 0.017]
Air pollution (NO ²)	-0.014 (0.009)	[-0.031, 0.004]
ID diagnosis x Damp x Age	-0.060 (0.077)	[-0.211, 0.091]
Damp x Age	0.002 (0.011)	[-0.019, 0.022]
ID diagnosis x Age	0.040 (0.029)	[-0.016, 0.096]
ID diagnosis x Damp	-0.531 (0.246)	[-1.014, -0.048]
Damp	0.154 (0.033)**	[0.090, 0.218]
Urban	0.071 (0.056)	[-0.039, 0.180]
Access to garden	-0.166 (0.130)	[-0.421, 0.089]
Home spatial density	0.399 (0.063)**	[0.278, 0.522]
Maternal psychological distress	0.650 (0.098)**	[0.458, 0.843]
Below poverty line	0.191 (0.037)**	[0.163, 0.303]
Atypical family structure	0.345 (0.036)**	[0.274, 0.416]
Male	0.263 (0.035)**	[0.195, 0.331]
ASD	1.196 (0.313)**	[0.583, 1.810]
White ethnicity	0.171 (0.068)	[0.037, 0.304]
Constant	0.607 (0.180)*	[0.254, 0.960]
Random effects	Estimate (SE)	95% CI
Level 3 (ward-level)		
Intercept variance	0.176 (0.022)	[0.138, 0.224]
Level 2 (Child level)		
Slope (age) variance	0.139 (0.005)	[0.130, 0.148]
Intercept variance	1.058 (0.018)	[1.023, 1.093]
Intercept-slope variance	-0.357 (0.034)	[-0.421, -0.289]

Note. Age was measured in years and grand mean centered (6.71 yrs).

For fixed effects: * $p < .01$, ** $p < .001$

Supplementary Table 7: Sensitivity analysis: ethnicity x spatial density x Intellectual disability

Table 7: ethnicity x spatial density x intellectual disability sensitivity analysis (N = 8168).

Fixed effects	Coefficient (SE)	95% CI
Age	-0.225 (0.005)**	[-0.234, -0.215]
Age ²	0.052 (0.001) **	[0.049, 0.054]
ID diagnosis	2.656 (0.754)**	[1.178, 4.134]
Greenspace (ward decile)	0.000 (0.008)	[-0.017, 0.017]
Air pollution (NO ²)	-0.013 (0.009)	[-0.030, 0.005]
ID diagnosis x ethnicity x spatial density	0.906 (0.670)	[-0.407, 2.219]
Ethnicity x spatial density	0.419 (0.113)**	[0.197, 0.641]
ID diagnosis x ethnicity	-1.061 (0.841)	[-2.710, 0.587]
ID diagnosis x spatial density	-1.623 (0.580)*	[-2.760, -0.486]
Damp	0.143 (0.031)**	[0.083, 0.203]
Urban	0.067 (0.056)	[-0.042, 0.176]
Access to garden	-0.166 (0.130)	[-0.420, 0.089]
Home spatial density	0.0751 (0.102)	[-0.124, 0.274]
Maternal psychological distress	0.653 (0.098)**	[0.461, 0.845]
Below poverty line	0.190 (0.037)**	[0.118, 0.263]
Atypical family structure	0.343 (0.036)**	[0.272, 0.414]
Male	0.262 (0.035)**	[0.194, 0.330]
ASD	1.173 (0.312)**	[0.561, 1.786]
White ethnicity	-0.199 (0.122)	[-0.438, 0.041]
Constant	0.903 (0.202)**	[0.508, 1.299]
Random effects	Estimate (SE)	95% CI
Level 3 (ward-level)		
Intercept variance	0.173 (0.022)	[0.136, 0.222]
Level 2 (Child level)		
Slope (age) variance	0.139 (0.005)	[0.130, 0.148]
Intercept variance	1.056 (0.018)	[1.022, 1.092]
Intercept-slope variance	-0.357 (0.034)	[-0.422, -0.289]

Note. Age was measured in years and grand mean centered (6.71 yrs).
For fixed effects: * $p < .01$, ** $p < .001$