The quality of air outside and inside the home: associations with emotional and behavioural problem scores in early childhood

Emily Midouhas*, Theodora Kokosi and Eirini Flouri

Abstract

Background: This study explored the role of outdoor air pollution [nitrogen dioxide (NO₂) and sulphur dioxide (SO₂)] and indoor air quality (measured with damp or condensation and secondhand smoke exposures) at age 9 months in emotional, conduct and hyperactivity problems at age 3 years.

Method: Data from 11,625 Millennium Cohort Study children living in England and Wales were modelled using multilevel regression.

Results: After adjusting for a host of confounders, having a damp or condensation problem at home was related to both emotional and conduct problems. Secondhand smoke exposure was associated with all three problem types. Associations with outdoor air pollution were less consistent.

Conclusions: Exposures to damp or condensation and secondhand smoke in the home are likely to be risk factors for child emotional and behavioural problems. Parents should continue to be educated about the dangers of exposing their children to poor air quality at home.

Keywords: Air pollution, Damp, Emotional and behavioural problems, Millennium Cohort Study, Secondhand smoke

Background

It has been recently estimated that outdoor air pollution results in around 40,000 deaths per year in the UK, predominantly caused by harmful toxins emitted by diesel engines such as nitrogen dioxide (NO₂) and particulate matter (PM) [1]. Air pollution has been associated with a number of adverse respiratory and cardiovascular health outcomes in UK adults and children [2, 3]. Yet little is known about its impact on child mental health in the UK. About 10% of UK children are estimated to have severe mental health problems [4]. Such problems are associated with increased risk of mental disorders and crime in adulthood [5].

Exposure to air pollutants may increase children’s risk of poor mental health through its impact on the developing brain and by promoting inflammation. Evidence, from mainly animal but some human studies too, suggests that air pollution can lead to systemic inflammation that triggers endothelial activation and breakdown of the blood-brain barrier (BBB) [6]. Weakening of the BBB facilitates the passage of air pollutants into the brain via the BBB leading to neuroinflammation, the elevation of pro-inflammatory cytokines and reactive oxygen species. Knowledge of the exact mechanisms by which pollutants in the brain bring about neuroinflammation is still developing, although microglia, the immune response cells of the central nervous system, are likely to be integral to this process [7]. Also developing is research on the specific areas of the brain that may be affected. Studies by Calderón-Garcidueñas and colleagues [8–10] with children in Mexico City where air pollution levels are extremely high have found evidence that suggests that the prefrontal cortex, a region important for behavioural regulation and which...
undergoes considerable development throughout childhood and adolescence [11], may be particularly sensitive to neurological effects of air pollution. Other research studies have found elevated neuroinflammation in the hippocampus due to air pollutants [12].

The pollution-child mental health link via inflammation is as yet unexplored, despite much evidence for the link between pollution and inflammation and for the one between inflammation and mental health problems (although mainly in adult populations) [13, 14]. For example, studies have found higher levels of inflammatory markers including interleukin 6 (IL-6), a multifunctional cytokine, and C-reactive protein (CRP), an acute phase protein, among clinical patients with psychiatric disorders [15, 16] compared with individuals without these disorders. Possible mechanisms linking inflammatory responses in the central nervous system to mental health problems including depression and anxiety are hyperactivity and hypoaclivity of the hypothalamic-pituitary-adrenal axis, oxidative stress and altered monoamine and glutamate neurotransmission and hippocampal neurogenesis (evidenced in adults, but unexplored in children) [13, 17–19].

What research has established is that children exposed to air pollutants, either in the prenatal or in the postnatal period, are at greater risk of behavioural problems, a diagnosis of attention deficit/hyperactivity disorder and being prescribed medication for psychiatric disorders [17, 20–28]. For example, one study in the U.S. found a relationship between exposures to black carbon attributed to traffic in the first year of life and children's symptoms of hyperactivity at age 7 in the Cincinnati Childhood Allergy and Air Pollution Study birth cohort [29]. However, the relationship applied only to children exposed to the highest tertile of traffic-related pollution and who had a mother with more than a secondary school degree.

Another, by Harris et al. [21], using data from a small pregnancy cohort in Massachusetts, U.S., identified a link between black carbon exposure and behavioural problems as reported by teachers. A third, by Oudin et al. [23] in Sweden, found effects of NO₂ and PM on dispensing of medication for child and adolescent mental health problems. More recently, Min and Min [22] identified an association between NO₂ and a diagnosis of attention deficit/hyperactivity disorder in a large sample of Korean children tracked from birth until age 10. Studies on non-human subjects also show adverse effects. Experimental studies exposing laboratory animals (rats and mice) to polycyclic aromatic hydro-carbon during the postnatal period have reported anxiety and depression-like symptoms [30, 31].

Estimating the genuine impact of outdoor air pollution on child mental health outcomes is not straightforward. The majority of studies do not account for sources of indoor air pollution that may be confounders [29, 32]. Also, children spend most of their time indoors and, as such, exposure to indoor pollutants generated from activities such as heating, cooking, smoking and use of cleaning products, varnishes and paints may be more important for young children than exposure to outdoor pollutants. Moreover, outdoor air quality may affect indoor air quality as pollutants from outdoors (e.g., produced by traffic emissions and industrial processes) may enter the home through windows or other types of ventilation. Therefore, alongside indicators of outdoor air quality two measures of indoor air quality were considered in this study: damp or condensation and second-hand smoke.

Damp or condensation can cause growth of moulds and bacteria that generate microscopic airborne particles [33], some of which contain allergens or chemicals that have the potential to cause neuroinflammation. Whereas many epidemiological studies have uncovered associations between indoor mould and respiratory problems in children [34–38], studies addressing relationships between mould and mental health in childhood are rare [17, 39]. In fact, this is the first study, to our knowledge, that explores damp or condensation problems in the home and young children's behaviour in a general population sample.

Secondhand smoke, the other indicator of indoor air quality we explored, has high concentrations of many toxic chemicals that are harmful to the brain [40]. For example, carbon monoxide in the bloodstream can reduce oxygen in the brain [41] and nicotine can impact the cholinergic system that regulates higher-order processing and memory [42, 43]. There is indeed evidence that preschool children exposed to secondhand smoke, either prenatally or postnatally, show more externalising (behavioural) problems than those unexposed [44–51], and that effects persist into adolescence [45, 49, 51].

There is therefore evidence supporting the link between poor air quality and child behaviour, but the existing research has several limitations that this study was carried out to address. First, although recent research has explored outdoor and indoor air quality and cognitive ability in early childhood [52], no study has explored simultaneously the roles of outdoor and indoor air quality in emotional and behavioural problems during this developmental phase. Second, no study has yet explored the associations between air quality and child emotional and behavioural problems in the general population (the research to date has targeted urban samples). Last, to our knowledge, no study on outdoor air pollution effects on child behaviour has adjusted for the amount of greenery in the child's neighbourhood. Green space may confound the association between outdoor air pollution and child behaviour since lower pollution is likely to be found in greener neighbourhoods. With
more land devoted to greenery, there is less land available for pollution-generating processes and combustion. Additionally, trees (one of the two key components of urban vegetation) capture large amounts of airborne gaseous, particulate and aerosol pollutants, thereby reducing their concentration in the air [53–57]. Therefore, woodlands and the presence of trees and, in general, amount of green space in the environment can improve air quality, and are, at the same time, negatively related to emotional problems among children [58, 59].

The present study aimed to address these gaps in the literature through a secondary analysis of data from the UK’s Millennium Cohort Study (MCS), a longitudinal study of 19,519 children, born around 2000, and their families. It explored the association between neighbourhood-level (i.e. ward) NO2 and sulphur dioxide (SO2) measured at the beginning of MCS (at age 9 months) and child emotional and behavioural problems at age 3 (when first measured in MCS) in England and Wales. Although brain development continues well into adulthood, the first 2–3 years of life is a key period for brain development when children may be particularly vulnerable to the impact of environmental toxins [60].

NO2 and SO2 are gaseous pollutants mainly caused by fossil fuel combustion [61]. SO2 is an acid gas resulting from the combustion of fossil fuels that contain sulphur such as coal and heavy oils. NO2 results from nitrogen oxides reacting with oxygen or ozone. Road traffic (mainly from diesel emissions) and energy production processes are the main sources of NO2. The EU has published air quality standards indicating the legal levels of pollutants, including those outlined in the EU Ambient Air Quality Directive and the fourth Daughter Directive. In the UK, the level of NO2 has regularly exceeded the legal level. In 2013, around one-third of licensed cars in the UK were diesel [62]. As a result, air pollution exposures have become an important concern for policymakers, urban planners and citizens, especially in large urban areas (e.g., London) where pollution levels are highest. Hence, understanding the effects of air pollution on children may have important implications for UK transport and environmental policy as well as urban planning. Unfortunately, UK air quality standards presently apply to outdoor air only and not to indoor air in residential homes. With regard to indoor air quality, the Committee on the Medical Effects of Air Pollutants (COMEAP) have published guidelines [63] and the National Institute for Health and Care Excellence (NICE) and Public Health England (PHE) are presently developing guidelines to be published in 2019 (https://www.nice.org.uk/guidance/inddevelopment/gid-ng10022). It is important that we continue to further our understanding of the risks posed by both outdoor and indoor air pollutants, to inform policies and practices that affect outdoor air quality as well as the development of standards for indoor air quality in homes.

**Methods**

**Study sample**

The Millennium Cohort Study (MCS) is a longitudinal survey taking its sample from all births in the UK over a year, starting on 1 September 2000 [64]. There have been six sweeps of data thus far with 19,519 children participating in at least one of these sweeps. The sample is disproportionately stratified to allow for sufficient numbers in the four UK countries and electoral wards with disadvantaged or (in England) ethnic minority populations [64]. Ethical approval was gained from National Health Service Research Ethics Committees and parents gave written informed consent for themselves and their children before interviews took place, at each sweep.

We used data2 from Sweeps 1 (at age 9 months, dates ranging June 2001 to January 2003) and 2 (at 3 years, dates ranging September 2003 to April 2005). Initially, we retained singleton children and first born twins/triplets (19,244 out of 19,519), making the number of children equal to the number of families. Our ‘analytic’ sample (those included in our analyses) included children living in England or Wales and who had data on emotional and behavioural problems, at Sweep 2 (n = 11,625). Our ‘non-analytic’ sample (those not included in our analyses) comprised children who did not live in England and Wales and/or did not have data on emotional and behavioural problems, at Sweep 2, i.e., at age 3 (n = 7619).

**Measures**

Emotional and behavioural problems at age 3 were measured with 15 items (on 3-point response scales) of the parent-reported Strengths and Difficulties Questionnaire (SDQ) [65] measuring hyperactivity (α = .71 across sweeps), emotional symptoms (α = .52) and conduct problems (α = .69). Each of the three scales has 5 items. Examples of items from the hyperactivity scale are ‘restless, overactive, cannot stay still for long’ and ‘constantly fidgeting or squirming’. Emotional symptoms items include ‘many worries, often seems worried’ and ‘often unhappy, down-hearted and tearful’, and examples of conduct problems items are ‘often fights with other children and bullies them’ and ‘often has temper tantrums or hot tempers’. Each SDQ item is scored 0 if the response is ‘not true’, 1 for ‘somewhat true’ and 2 for ‘certainly true’. Scores for each scale may range 0–10. In our sample, internal consistency (see above) was in line with other research using the SDQ [66].

Outdoor air pollutants, SO2 and NO2, were measured at age 9 months with data from the Multiple Environmental Deprivation Index (MEDIx). MEDIx is a measure of physical environmental deprivation that represents both pathogenic and salutogenic characteristics in each
UK ward [67, 68]. Air pollution was measured with annual mean concentrations [in micrograms (one-millionth of a gram) per cubic meter air or μg/m³] within each ward. Means are population weighted using output area (OA) units, and cover the years 1999–2003. Data were taken from 1 km grids, modelled from National Atmospheric Emissions Inventory data. The annual mean values were converted to deciles across all wards in the UK. In the bottom decile the annual mean concentration of NO₂ is less than 9.26 μg/m³ and in the top decile it is greater than 33.89. Note that the annual mean legal limit for NO₂ is 40 μg/m³ [69]. With regard to SO₂, these values are 1.78 and 7.24, respectively. The annual mean legal limit for SO₂ is 20 μg/m³.

Indoor air quality was measured by the mother’s report of whether there was a damp or condensation problem at home and whether the baby was exposed to secondhand smoke, both at age 9 months. For the latter, mothers were asked whether anyone smokes in the same room as the baby.

We also measured two additional neighbourhood factors at age 9 months – urbanicity and the percentage of green space. Urbanicity was measured with a variable that indicated if the family resided in an urban area (i.e., a settlement with a population greater than 10,000) or not. The percentage of green space, covering all green spaces larger than 5 m² (excluding private gardens) for each ward, was measured with data from the MEDIX. Green spaces include neighbourhood greenery, parks, playing fields and forests/woodlands. Estimates are based on land use data from the Generalised Land Use Database [70] and the Coordination of Information on the Environment [71]. In MCS, the percentages of ward-level greenery using the MEDIX data are available in deciles across all wards in the UK. The lowest decile corresponds to wards with 0–21% green space and the top decile refers to wards with 94–95% green space.

A range of key family and child covariates were included. Family-level variables (measured at age 3) were household income (equivalised weekly income, adjusting for household size and composition), maternal education (University degree or not), maternal psychological distress, measured with the Kessler Psychological Distress scale (K6), a 6-item screener of psychological distress [72] and maternal general health (self-reported). For the latter, a binary variable was used where ‘good’ or ‘excellent’ general health was compared to ‘fair’ or ‘poor’. The child-level covariates were gender, age (in years), ethnicity (White, Black, Indian, Pakistani/Bangladeshi, Mixed and Other) and low birth weight (< 2.5 kg).

Statistical analysis
The main aim of the study was to examine the relationship between outdoor and indoor air quality at age 9 months and emotional and behavioural problems at age 3, controlling for neighbourhood greenery and urbanicity, family-level variables related to neighbourhood selective sorting and emotional and behavioural problems and child covariates. To meet this aim, we first carried out descriptive analyses, including correlations among the main variables of the study. As MCS data have a hierarchical structure due to the sampling design (with children/families nested within areas), we fitted 2-level linear regression models for each SDQ scale we considered (emotional problems, conduct problems and hyperactivity problems). We accounted for MCS area clustering at the ward-level and, as explained, the environmental variables (outdoor air pollution, green space and urbanicity) were measured at ward-level, thus aligning with the clustering of cohort families in wards in MCS.

In our models, we adjusted for the MCS strata to reflect the stratified sample design: in each of the four UK countries, families were oversampled from areas with high child poverty (‘disadvantaged’) and (in England only) from areas with high proportions of ethnic minorities (‘ethnic’). Since our sample included only children from England and Wales, we adjusted for five strata: 1) England-advantaged, 2) England-disadvantaged, 3) England-ethnic, 4) Wales-advantaged and 5) Wales-disadvantaged. Listwise deletion was used in all models.

Results
On average, children in the analytic sample lived in wards in England and Wales with NO₂ and SO₂ annual concentrations at the higher end of the distribution of wards in the UK (Table 1). Around 14% and 12% families in the analytic sample reported damp or condensation and secondhand smoke in the home, respectively. Additionally, they resided in comparatively less green neighbourhoods across the distribution of wards in the UK. The majority of the sample lived in urban areas (83%). With regard to emotional/behavioural problems, SDQ scores ranged 1–4 depending on the domain, on average, which is at the lower end of the 0–10 range. There were statistically significant correlations (Table 2), as expected, across the main study variables. The associations between air quality (both indoor and outdoor) and emotional and behavioural problems, however, were weak.

With respect to selection bias, the children in the analytic sample (n = 11,625) differed from those in the non-analytic sample (n = 7619) on all variables except for gender, maternal education and birth weight. For example, the children in the analytic sample were more likely to have higher household incomes (p < .001) and to have mothers with more psychological distress (p < .001). The analytic sample was additionally more likely to live in areas with higher levels of SO₂ (p < .001) and NO₂ (p < .001) and less green space (p < .001). It also had proportionately fewer cases of secondhand smoking exposure (p < .001) but more cases of
Table 1 Descriptives of study variables in the analytic sample (n = 11,625)

| Child | n  | %  |
|-------|----|----|
| Female | 5706 | 49.26 |

| Ethnicity | n  | %  |
|-----------|----|----|
| White     | 9550 | 86.65 |
| Black     | 424  | 2.90 |
| Indian    | 333  | 1.89 |
| Pakistani/Bangladeshi | 720  | 3.67 |
| Mixed     | 420  | 3.62 |
| Other     | 173  | 1.26 |
| Low birth weight | 794  | 6.83 |

| Parent/household (S2) | n  | %  |
|-----------------------|----|----|
| Mother is university-educated | 3748 | 33.69 |
| Mother has good general health | 9371 | 81.52 |

| Indoor air quality (S1) | n  | %  |
|-------------------------|----|----|
| Damp or condensation problem | 1598 | 14.22 |
| Secondhand smoke        | 1397 | 12.36 |

| Neighbourhood (S1) | n  | %  |
|-------------------|----|----|
| Urbanicity        | 9336 | 83.01 |

| Child (S2) | n  | M (SD) |
|------------|----|--------|
| Emotional problems | 11,549 | 1.36 (1.41) |
| Conduct problems | 11,569 | 2.84 (1.98) |
| Hyperactivity problems | 11,463 | 3.97 (2.26) |
| Age (years) | 11,625 | 3.13 (0.19) |

| Parent/household (S2) | n  | M (SD) |
|-----------------------|----|--------|
| Household income      | 10,924 | 324.01 (196.36) |
| Maternal psychological distress | 9660 | 3.28 (3.50) |

| Neighbourhood (S1) | n  | M (SD) |
|-------------------|----|--------|
| NO2 deciles       | 11,035 | 7.27 (2.27) |
| SO2 deciles       | 11,035 | 6.51 (2.34) |
| Green space deciles | 11,035 | 4.27 (2.33) |

Note: S1 Sweep 1 (age 9 months), S2 Sweep 2 (age 3 years). Estimates are weighted and Ns are unweighted.

The two measures of indoor air quality in infancy were significantly related to both emotional and conduct problems but only secondhand smoke exposure was related to hyperactivity problems (b = 0.37; Table 3). With regard to conduct problems, damp/condensation in the home and exposure to secondhand smoke were significantly associated with an increase of 0.16 and 0.59 problems, respectively. Damp/condensation in the home and exposure to secondhand smoke were related positively to emotional problems (b = 0.09 and b = 0.16, correspondingly).

With respect to the other variables in the model, urbanicity was not significantly associated with any problem type. The amount of green space was significantly related to conduct problems (b = 0.04) but not to emotional problems or hyperactivity. Household income and mother’s university education and good general health were significantly associated with fewer problems in all three domains. With regard to the child factors, Indian and Pakistani/Bangladeshi (relative to White) children as well those with low birth weight had more emotional problems. Girls and children of Black ethnic origin (relative to White) had fewer conduct problems. Girls were less likely to have hyperactivity problems and children from Pakistani/Bangladeshi backgrounds as well as those with low birth weight were more likely to have such problems. Random effects for all models showed that the between-child variance in emotional, conduct and hyperactivity problems was significant, but the between-ward variance in children’s problems was not.4

Discussion
This study assessed the role of outdoor neighbourhood exposure to NO2 and SO2 and indoor residential exposure to damp/condensation and secondhand smoke at age 9 months in three domains of child behaviour – emotional symptoms, conduct problems and hyperactivity problems (measured at age 3 years) – in a nationally representative sample in England and Wales. A range of important confounders were included such as neighbourhood greenery and urbanicity as well as family background characteristics related to selective sorting into neighbourhoods.

We found that our indoor air quality indicators were associated with multiple child behaviour outcomes. Living in a home with a damp or condensation problem at age 9 months was related to both emotional symptoms and conduct problems at age 3. Effects were robust to adjustment for socio-economic status including maternal education and household income as well as measures of outdoor air quality including green space and air pollution. This is the first study we are aware of to link indoor damp/condensation to child behaviour and mental health in a general population sample. Moreover, secondhand smoke exposure at age 9 months was related to
all three child outcomes at age 3, with the largest effect seen for conduct problems (equivalent to an increase in .59 points on the conduct problems scale). Most of the extant literature has examined the relationship between prenatal rather than postnatal exposure to smoking and child behaviour [44–46, 49, 50]. However, reflecting this study’s finding, a fairly recent study in Scotland found that exposure to secondhand smoke (objectively-measured) was associated with poorer child behaviour, particularly externalising problems, also measured with the SDQ [47].

Table 3: Effects of indoor and outdoor air quality at age 9 months on emotional, conduct and hyperactivity problems at age 3 years

| Variables | Emotional symptoms | Conduct problems | Hyperactivity problems |
|-----------|--------------------|------------------|----------------------|
|           | Coeff. (SE)        | 95% CI           | Coeff. (SE)         | 95% CI            | Coeff. (SE)         | 95% CI |
| Fixed effects |                        |                  |                      |                                      |                      |       |
| Constant  | 0.90**(0.26)       | [0.39, 1.41]     | 3.70***(0.356)      | [3.00, 4.39]      | 3.96***(0.41)      | [3.15, 4.77] |
| SO2 (deciles) | 0.004(0.01)     | [−0.01, 0.02]    | 0.01(0.01)         | [−0.01, 0.03]    | 0.02(0.01)         | [−0.001, 0.05] |
| NO2 (deciles) | 0.01(0.01)       | [−0.01, 0.02]    | 0.03(0.01)         | [0.004, 0.06]    | 0.02(0.02)         | [−0.001, 0.05] |
| Damp or condensation | 0.09*(0.04) | [0.002, 0.17]   | 0.16***(0.06)      | [0.04, 0.27]     | 0.03(0.07)         | [−0.10, 0.17] |
| Secondhand smoke | 0.16**(0.05) | [0.07, 0.25]     | 0.59***(0.06)      | [0.47, 0.71]     | 0.37**(0.07)       | [0.23, 0.52] |
| Green space | 0.01(0.01)       | [−0.01, 0.02]    | 0.04*(0.01)        | [0.02, 0.07]     | 0.02(0.02)         | [−0.001, 0.05] |
| Urban     | 0.07(0.05)        | [−0.04, 0.18]    | 0.08(0.08)         | [−0.07, 0.23]    | 0.03(0.09)         | [−0.17, 0.18] |
| Age       | 0.08(0.07)        | [−0.06, 0.22]    | −0.36**(0.10)      | [−0.53, −0.16]   | 0.19(0.12)         | [−0.04, 0.41] |
| Female    | 0.003(0.03)       | [−0.06, 0.06]    | −0.22**(0.04)      | [−0.30, −0.15]   | −0.60**(0.05)      | [−0.70, −0.51] |
| Ethnicity (Ref: White) |            |                  |                      |                                      |                      |       |
| Mixed     | −0.04(0.08)       | [−0.20, 0.13]    | 0.03(0.11)         | [−0.19, 0.26]    | −0.01(0.13)        | [−0.27, 0.25] |
| Indian    | 0.26*(0.11)       | [0.04, 0.48]     | −0.02(0.16)        | [−0.32, 0.29]    | 0.16(0.18)         | [−0.20, 0.51] |
| Pakistani/Bangladesi | 0.81****(0.09) | [0.63, 0.99]     | −0.03(0.13)        | [−0.28, 0.21]    | 0.32**(0.15)       | [0.03, 0.61] |
| Black     | −0.11(0.10)       | [−0.31, 0.08]    | −0.32*(0.13)       | [−0.58, −0.06]   | −0.28(0.16)        | [−0.58, 0.03] |
| Other     | 0.23(0.18)        | [−0.12, 0.58]    | −0.32(0.24)        | [−0.79, 0.15]    | −0.32(0.29)        | [−0.88, 0.24] |
| Low birth weight | 0.18**(0.06) | [0.06, 0.30]     | 0.04(0.09)         | [−0.13, 0.21]    | 0.53**(0.10)       | [0.33, 0.72] |
| Mother is university-educated | −0.16****(0.03) | [−0.23, −0.09]  | −0.31****(0.05)   | [−0.40, −0.22]   | −0.48**(0.06)      | [−0.59, −0.37] |
| Household income | −0.00****(0.00) | [−0.00, −0.00]  | −0.001****(0.00)  | [−0.001, −0.001] | −0.001****(0.00)  | [−0.001, −0.001] |
| Maternal psychological distress | 0.06**(0.00) | [0.07, 0.09]     | 0.13****(0.01)    | [0.12, 0.14]     | 0.11****(0.01)    | [0.10, 0.12] |
| Mother has good general health | −0.17****(0.04) | [−0.25, −0.10]  | −0.25****(0.06)   | [−0.36, −0.15]   | −0.22**(0.06)      | [−0.34, −0.09] |
| Random effects |                        |                  |                      |                                      |                      |       |
| Ward-level intercept variance (Level 2) | 0.00(0.00)       | [0.00, 0.00]     | 0.01(0.01)         | [0.00, 0.09]     | 0.003(0.01)        | [0.00, 0.35] |
| Child-level intercept variance (Level 1) | 1.93****(0.03) | [1.87, 1.99]     | 3.58****(0.05)    | [3.48, 3.69]     | 4.87****(0.07)    | [4.73, 5.02] |

Notes: *p < .05; **p < .01; ***p < .001. Coefficients are unstandardised. All models are adjusted for the area strata.
The probability of incorrectly rejecting the null hypotheses that there are no differences in behaviour for children living in homes with secondhand smoke and a damp or condensation problem is less than 5%, given we set our significance level at .05. However, at the same time, having such a large sample (n = 11,625) raises the likelihood of detecting significant effects in the sample. Thus, it is especially important to consider the practical significance of the effects detected. For example, considering the weaker effects we found for a damp or condensation problem (relative to secondhand smoke), the estimated differences in problems for a child with and without a damp or condensation problem were .09 and .16 points on the emotional symptoms and conduct problems scales, respectively. Note that each scale score ranges 0 to 10 and an increase in one point is the difference between a response of ‘not at all true’ and ‘somewhat true’ or a response of ‘somewhat true’ and ‘certainly true’. Therefore, an increase in .09 and .16 points is not even close to reaching the endorsement of a problem. On the other hand, the stronger effects on externalising problems for secondhand smoke were .37 (hyperactivity) and .59 (conduct problems). These reflect a difference of roughly a third and over a half of a problem respectively for those exposed and those unexposed to secondhand smoke. Still, explanatory variables in general were found to have small to moderate effect sizes. Nevertheless, our indoor air quality effects were akin to or greater than those found for other child and family factors known to be important for child behaviour. For example, in our models, a greater increase in conduct problems was found for children who were exposed to secondhand smoke than children whose mothers did not have a university degree. Therefore, we believe it is important to consider indoor air quality as another risk factor for child behaviour, alongside family background and key child characteristics, assuming the differences we have found are not due to sampling error and are true in the population.

Conversely, we did not find evidence for a reliable association between child behaviour and exposure to outdoor NO₂ or SO₂. The level of NO₂ (but not SO₂) in the neighbourhood at age 9 months had a very small but significant linear association with conduct problems at age 3. This finding reflects those of Min and Min [22], Oudin et al. [23] and Newman et al. [29]. However, two of these studies [22, 23] found associations with clinical outcomes - diagnoses of attention deficit/hyperactivity disorder and dispensing of medication, respectively - rather than the continuum of problem behaviour explored in this study. Newman et al. [29] did find an association between exposures to high levels of traffic-related pollution in the first year of life (when we measured NO₂) and symptoms of hyperactivity symptoms at age 7 (we examined child behaviour at age 3) but only for children whose mothers had attended higher or further education. That study also examined, but did not find, an association with conduct problems. Our study shows that even changes in externalising behaviour outside of a clinical range may result from air pollution exposures. Nonetheless, we must be cautious about the implications given the lack of effects for emotional problems and hyperactivity and the small effect size for conduct problems. The study’s findings may have implications for environmental policy with regard to indoor air quality, however. They may offer support for the need to develop UK health-based indoor air quality standards, especially about damp, condensation, mould and tobacco use.

Nonetheless, this study has several limitations. First, our findings regarding neighbourhood-level pollution may be due to traffic noise, neglected in other similar studies as well [22, 23, 29], which we could not adjust for in this study. Second, we could not consider exposures to pollution, smoking and alcohol prenatally [73] as MCS does not have data on prenatal exposures. Hence, the relationships we found between postnatal exposures and child behaviour may be at least partly due to exposures in pregnancy. Third, our measure of air pollution for each neighbourhood was an average of the annual mean concentrations across a 4-year period. The 4-year average may overlook annual changes in pollutant levels. Fourth, a smaller spatial resolution would have captured the more immediate outdoor air pollutants near children’s homes. A more appropriate neighbourhood geography might have been Lower layer Super Output Area (LSOA) or Output Area (OA) rather than ward. OAs are built from groups of adjacent postcodes in the UK (with an average of 309 residents) and are the base unit for census data releases. LSOAs are built from clusters of OAs (typically 4–6) with a population averaging 1500. Fifth, we were also unable to account for outdoor pollutants that may come from adjacent neighbourhoods. Sixth, we could not measure directly the amount of pollution indoors (e.g., NO₂, PM) including those resulting from secondhand smoke and damp or condensation or from activities such as heating, cooking, and use of cleaning products, paints and varnishes. Relatedly, our measure of secondhand smoke in the home did not consider other sources such as parents or other residents wearing smoke-laden clothes inside the home. Finally, we were unable to measure whether reports of damp or condensation were provided during the UK condensation season (March to October) which may affect the extent of dampness or condensation in the home. Future research studies should endeavour to address these limitations.

Conclusions

In a large UK general population sample, we found that exposures to damp or condensation and secondhand
smoke in the home are likely to be risk factors for emotional and behavioural problems in young children, measured using a well-validated measure of emotional and behavioural problem scores. Thus, this study offers reasons to continue to educate parents about the health risks of exposing their children to poor air quality, particularly within the home, and may give further evidence for the need for health-related UK residential air quality standards. Future research should continue to explore the proposed mechanisms linking air pollution to child behaviour and mental health [74] including neuroinflammation via oxidative stress, changes in serotonin and glutamate neurotransmission and hyperactivity and hypovascularity of the hypothalamic-pituitary-adrenal axis [13, 18, 19] as well as identify which regions of the brain, that are implicated in mental health problems, are most affected by these consequences of neuroinflammation.

Endnotes
1Electoral wards are the key building block of UK electoral geography (http://www.ons.gov.uk/ons/guide-method/geography/beginner-s-guide/administrative/england/electoral-wards-divisions/index.html). The average population is around 5500, though counts can vary substantially. Census area statistical (CAS) wards in particular are used in this study. They are a type of ward created for the 2001 census output produced by aggregating smaller output areas (http://webarchive.nationalarchives.gov.uk/20160108225108/http://www.ons.gov.uk/ons/guide-method/geography/beginner-s-guide/administrative/england/electoral-wards-divisions/statistical-wards%2D%2Dcas-wards-and-st-wards/index.html). In England and Wales, there were a total of 8850 CAS wards.
2Note that all measures and exclusions for our study are presented in this paper.
3Initially, we explored the correlations of all four pollutants available in MEDix – particulate matter 10 μm or less in diameter (PM10), NO2, SO2 and carbon monoxide (CO) - in our sample. Correlations among PM10, NO2 and CO were high enough (.83-.91) to warrant including only one of these in a regression model to avoid problems with multicollinearity. SO2 was only moderately correlated with the other three pollutants (with correlations ranging .42-.46). Therefore, we decided to retain NO2 and SO2 only for the analysis.
4We also considered whether the child’s health status might explain these differences in emotional and behavioural problems according to air pollution exposures. At age 3, parents were asked to report on whether their child had a longstanding illness or disability. We added this covariate to the regression models and the significance of the coefficients did not change.

Abbreviations
BBB: Blood-brain barrier; CO: Carbon monoxide; CRP: C-reactive protein; IL-6: Interleukin 6; K6: Kessler Psychological Distress 6-item scale; MCS: Millennium Cohort Study; MEDIx: Multiple Environmental Deprivation Index; NO2: Nitrogen dioxide; OA: Output area; PM: Particulate matter; SDQ: Strengths and Difficulties Questionnaire; SO2: Sulphur dioxide

Acknowledgements
We would like to thank Richard Mitchell, Elizabeth Richardson and their colleagues for developing and providing the Multiple Environmental Deprivation Index (MEDIx) air pollutant and green space data that we linked with the MCS for this project. We would also like to thank David Church at the Centre for Longitudinal Studies for his support in linking these data with the MCS.

Funding
This research was funded by the UCL Institute of Education Seed Funding Scheme, project 539323. The UCL Institute of Education had no part in the design, analysis, interpretation of data or manuscript write-up.

Availability of data and materials
The Millennium Cohort Study data analysed in this study are publicly available here: https://beta.ukdataservice.ac.uk/datacatalogue/series/series?id=2000031. To access these data, we had to register with the U.K. Data Archive and agree to an End User License. The data from the Multiple Environmental Deprivation Index (MEDIx) that we linked with the MCS are also publicly available as a result of this project (with registration and agreement to End User License required for users); https://discover.ukdataservice.ac.uk/catalogue/?sn=8153&type=data%20catalogue.

Authors’ contributions
EM, EF and TK designed the study, TK analysed the data and EM, EF and TK wrote the paper. All authors read and approved the final manuscript.

Ethics approval and consent to participate
Ethical approval was gained from National Health Service Research Ethics Committees (RECs) and parents gave written informed consent for themselves and their children (all children were under age 16 during sweeps 1–6) before interviews took place, at each sweep.

Consent for publication
N/A

Competing interests
The authors declare that they have no competing interests.

Publisher’s Note
Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Received: 5 October 2018 Accepted: 31 March 2019
Published online: 15 April 2019

References
1. Royal College of Physicians. Every breath we take: The lifelong impact of air pollution. Report of a working party. London: RCP; 2016.
2. Guarneri M, Balmes JR. Outdoor air pollution and asthma. Lancet. 2014;383(9928):1581–92.
3. Schwartz J, Litonjua A, Suh H, Verrier M, Zanobetti A, Syring M, Nearing B, Verrier R, Stone P, MacCallum G, et al. Traffic related pollution and heart rate variability in a panel of elderly subjects. Thorax. 2005;60(6):455–61.
4. Gutman L, Joshi H, Parsonage M, Schoon I. Children of the new century. Mental health findings from the Millennium Cohort Study London. London: Centre for Mental Health; 2015.
5. Goodman A, Joyce R, Smith JP. The long shadow cast by childhood physical and mental problems on adult life. Proc Natl Acad Sci U S A. 2011;108(15):6032–7.
6. Brockmeyer S, D’Angiulli A. How air pollution alters brain development: the role of neuroinflammation. Transl Neurosci. 2016;7(1):24–30.
7. Calderon-Garciduenas L, Leray E, Heydarpour P, Torres-Jardon R, Reis J. Air pollution, a rising environmental risk factor for cognition,
neuroinflammation and neurodegeneration: the clinical impact on children and beyond. Rev Neurol (Paris). 2016;172(1):69–80.

8. Calderon-Garciduenas L, Cross JV, Franco-Lira M, Aragon-Flores M, Kavamura M, Torres-Jardon R, Chao CK, Thompson C, Chang J, Zhu HT, et al. Brain immune interactions and air pollution: macrophage inhibitory factor (MIF), prion cellular protein (PrPC), interleukin-6 (IL-6), interleukin 1 receptor antagonist (IL-1Ra), and interleukin-2 (IL-2) in cerebrospinal fluid and MIF in serum differentiate urban children exposed to severe vs. low air pollution. Front Neurol. 2013;7:183.

9. Calderon-Garciduenas L, Macias-Parra M, Hoffmann H, Valencia-Salazar G, Henriquez-Roldan C, Osnaya N, Monte OC, Barragan-Neja G, Villareal-Calderon R, Romero L, et al. Immunotoxicity and environmental immunodepression and systemic inflammation in children. Toxicol Pathol. 2009;37(2):161–9.

10. Calderon-Garciduenas L, Mora-Tiscareno A, Styner M, Gomez-Gaiza G, Zhu H, Torres-Jardon R, Carlos E, Solorio-Lopez E, Medina-Cortina H, Kavamura M, et al. White matter hyperintensities, systemic inflammation, brain growth, and cognitive functions in children exposed to air pollution. J Alzheimers Dis. 2012;31(1):183–91.

11. Tsujimoto S. The prefrontal cortex: functional neural development during early childhood. Neuroscientist. 2008;14(4):345–58.

12. Folken LK, Xu X, Weil ZM, Chen G, Sun Q, Rajagopalan S, Nelson RJ. Air pollution impairs cognition, provokes depressive-like behaviors and alters hippocampal cytokine expression and morphology. Mol Psychiatry. 2011;16(10):987–95.

13. Krishnadas R, Cavanagh J. Depression: an inflammatory illness? J Neurol Neurosurg Psychiatry. 2012;83(5):495–502.

14. Smith LL. Acute inflammation: the underlying mechanism in delayed onset muscle soreness? Med Sci Sports Exerc. 1991;23(5):242–11.

15. Howren MB, Lamkin DM, Suls J. Associations of depression with C-reactive protein, IL-1, and IL-6-a meta-analysis. Psychosom Med. 2009;71(2):171–86.

16. Kiecolt-Glaser JK, Derry HM, Fagundes CP. Inflammation: depression fans the flames and feasts on the heat. Am J Psychiatry. 2015;172(11):1075–83.

17. Anyanwu EC, Campbell AW, Vojdani A. Neurophysiological effects of chronic indoor environmental toxic mold exposure on children. ScientificWorldJournal. 2003;3:281–90.

18. Leonard BE. Inflammation and depression: a causal or coincidental link to the pathophysiology? Acta Neuropsychiatr. 2018;30(1):1–16.

19. Pariente CM. Why are depressed patients inflamed? A reflection on 20 years of research on depression, glucocorticoid resistance and inflammation. Eur Neuropsychopharmacol. 2017;27(6):534–9.

20. Cheng D, Schwarz EB, Douglas E, Horon I. Unintended pregnancy and delinquency of the offspring: an association without the definition of the syndrome. J Neural Transm. 2016;123(7):785–90.

21. Min JY, Min KB. Exposure to ambient PM10 and NO2 and the incidence of behavior. Neurotoxicol Teratol. 2016;57:60–73.

22. Yokota S, Mizu K, Moriya N, Oshio S, Sugavara I, Takeda K. Effect of prenatal exposure to diesel exhaust on dopaminergic system in mice. Neurosci Lett. 2009;469(1):38–41.

23. Clifford A, Leng L, Chen R, Anstey KJ, Seaton A. Exposure to air pollution and cognitive functioning across the life course—a systematic literature review. Environ Res. 2016;147:383–98.

24. Gomzy RL, Reponen T, Willette K, Schmeichel D, Robine E, Boissier M, Grinshpun SA. Fungal fragments as indoor air biocorrosions. Appl Environ Microbiol. 2002;68(7):3522–31.

25. Gent JF, Ren P, Belanger K, Triche E, Bracken MB, Hoford TR, Leaderer BP. Levels of household mold associated with respiratory symptoms in the first year of life in a cohort at risk for asthma. Environ Health Perspect. 2002;110(2):A781–A86.

26. Hyens HP, Brugge D, Osgood N-D, Snell J, Vallarino J, Spengler J. Investigations into the indoor environment and respiratory health in Boston public housing. Rev Environ Health. 2004;19(3):271–89.

27. Peat JK, Dickerson J, Li J. Effects of damp and mould in the home on respiratory health: a review of the literature. Allergy. 1998;53(2):120–8.

28. Perzanowski MS, Spork R, Squallace SP, Kelber LE, Call R, Carter M, Platts-Mills TAE. Association of sensitization to Alternaria allergens with asthma among school-age children. J Allergy Clin Immun. 1998;101(5):S62–3.

29. Zock JP, Jarvis D, Luczynska C, Sunyer J, Burney P. European Community respiratory health survey: Housing characteristics, reported mold exposure, and asthma in the European Community respiratory health survey. J Allergy Clin Immun. 2002;110(2):285–92.

30. Jedrychowsky W, Mauger E, Perera F, Stigter L, Jankowski J, Butscher M, Mroz E, Flak E, Skarupa A, Sowa A. Cognitive function of 6-year old children exposed to mold-contaminated homes in early postnatal period. Prospective birth cohort study in Poland. Physiol Behav. 2011;104(5):899–95.

31. Chen R, Clifford A, Lang L, Anstey KJ. Is exposure to secondhand smoke associated with cognitive parameters of children and adolescents?-a systematic literature review. Ann Epidemiol. 2013;23(10):652–61.

32. Mezzacappa E, Buckner JC, Earls F. Prenatal cigarette exposure and infant learning stimulation as predictors of cognitive control in childhood. Developmental Sci. 2011;14(4):881–91.

33. Siotkin TA. Cholinergic systems in brain development and disruption by neurotoxictants: nicotine, environmental tobacco smoke, organophosphates. Toxicol Appl Pharmacol. 2004;198(2):132–51.

34. Siotkin TA. Developmental cholinotoxicants: nicotine and chlorpyrifos. Environ Health Persp. 1999;107(Suppl 1):71–80.

35. Day NL, Richardson GA, Goldschmidt L, Cornelius MD. Effects of prenatal exposure to tobacco smoke exposure on preschooler' behavior. J Dev Behav Pediatr. 2000;21(3):180–8.

36. Fergusson DM, Horwood LJ, Lynsey MT. Maternal smoking before and after pregnancy - effects on behavioral outcomes in middle childhood. Pediatrics. 1993;92(5):S15–22.

37. Fergusson DM, Woodward LJ, Horwood LJ. Maternal smoking during pregnancy and psychiatric adjustment in late adolescence. Arch Gen Psychiatry. 1998;55(8):721–7.

38. Harner M, Ford T, Stannatakas E, Dockray S, Batty GD. Objectively measured secondhand smoke exposure and mental health in children: evidence from the Scottish health survey. Arch Pediatr Adolesc Med. 2011;165(4):326–31.

39. Kahn RS, Zuckerman B, Bauchner H, Homer CJ, Wise PH. Women’s health after pregnancy and child outcomes at age 3 years: a prospective cohort study. Am J Public Health. 2002;92(8):1312–8.

40. Rantakallio P, Läärä E, Isohanni M, MOILANEN I. Maternal smoking during pregnancy and delinquency of the offspring: an association without causation? Int J Epidemiol. 1992;21(6):1063–1063.

41. Weitzman M, Byrd RS, Aligne CA, Moss M. The effects of tobacco exposure on children's behavioral and cognitive functioning: implications for clinical and public health policy and future research. Neurotoxicol Teratol. 2002;24(3):397–406.

42. Zhou S, Rosenthal DG, Sherman S, Zelkoff J, Gordon T, Weitzman M. Physical, behavioral, and cognitive effects of prenatal tobacco and postnatal secondhand smoke exposure. Curr Probl Pediatr Adolesc Health Care. 2014;44(8):219–41.
52. Midouhas E, Kokosi T, Flouri E. Outdoor and indoor air quality and cognitive ability in young children. Environ Res. 2018;161:321–8.
53. Beckett KP, Freer-Smith PH, Taylor G. Urban woodlands: their role in reducing the effects of particulate pollution. Environ Pollut. 1998;99(3):347–60.
54. Freer-Smith P, Broadmeadow M. The improvement of urban air quality by trees. Arboriculture Research and Information Note-Department of the Environment (United Kingdom); 1996.
55. Freer-Smith PH, Holloway S, Goodman A. The uptake of particulates by an urban woodland: site description and particulate composition. Environ Pollut. 1997;95(1):27–35.
56. Nowak D, Jones R, Berger J, Claussen M, Magnussen H. Airway responsiveness to sulfur dioxide in an adult population sample. Am J Respir Crit Care Med. 1997;156(4 Pt 1):1151–6.
57. Powe NA, Willis KG. Mortality and morbidity benefits of air pollution (SO2 and PM10) absorption attributable to woodland in Britain. J Environ Manag. 2004;70(2):119–28.
58. Feng X, Astell-Burt T. The relationship between neighbourhood green space and child mental wellbeing depends upon whom you ask: multilevel evidence from 3083 children aged 12–13 years. Int J Environ Res Public Health. 2017;14(3):235.
59. Flouri E, Midouhas E, Joshi H. The role of urban neighbourhood green space in children’s emotional and behavioural resilience. J Environ Psychol. 2014;40:179–86.
60. Block ML, Elder A, Auten RL, Bilbo SD, Chen H, Chen JC, Cory-Slechta DA, Costa D, Diaz-Sanchez D, Dorman DC, et al. The outdoor air pollution and brain health workshop. Neurotoxicology. 2012;33(5):972–84.
61. Katsouyanni K. Ambient air pollution and health. Br Med Bull. 2003;68(1):143–56.
62. Transport DF. National Travel Survey 2014: Statistical release. In: http://www.gov.uk/government/statistics/national-travel-survey-2014. Accessed 14 Jul 2017.
63. Committee on the Medical Effects of Air Pollutants. Guidance on the effects on health of indoor air pollutants. 2004. http://www.dh.gov.uk/products/consum_dh/groups/dh_digitalassets/@dh/@ab/documents/digitalasset/dh_096801.pdf. Accessed 1 Dec 2018.
64. Plewis I. Non-response in a birth cohort study: the case of the millennium cohort study. Int J Soc Res Methodol. 2007;10(5):325–34.
65. Goodman R. The strengths and difficulties questionnaire: a research note. J Child Psychol Psychiatry. 1997;38(5):581–6.
66. Stone LL, Otten R, Engels RC, Vermulst AA, Janssens JM. Psychometric properties of the parent and teacher versions of the strengths and difficulties questionnaire for 4- to 12-year-olds: a review. Clin Child Fam Psychol Rev. 2010;13(3):254–74.
67. Richardson EA, Mitchell R, Shortt NK, Pearce J, Dawson TP. Developing summary measures of health-related multiple physical environmental deprivation for epidemiological research. Environ Plann A. 2010;42(5):1650–68. https://cresh.org.uk/cresh-themes/environmental-deprivation/medix-and-medclass/. Accessed 10 July 2017.
68. Shortt NK, Richardson EA, Mitchell R, Pearce J. Re-engaging with the physical environment: a health-related environmental classification of the UK. Area. 2011;43(1):76–87. https://cresh.org.uk/cresh-themes/environmental-deprivation/medix-and-medclass/. Accessed 10 July 2017.
69. EEA. Air quality in Europe 2017: European Environment Agency. p. 2017. Accessed 15 July 2017.
70. Office of the Deputy Prime Minister. Generalised Land Use Database statistics for England. London: ODPM Publications; 2001.
71. EEA. CORINE land cover 2000. Copenhagen: European Environment Agency. p. 2000. http://www.eea.europa.eu/publications/CORD-landcover. Accessed 15 July 2017.
72. Kessler RC, Barker PR, Colpe LJ, Epstein JF, Gfroerer JC, Hiripi E, Howes MJ, Normand SL, Mersandichle RD, Walters EE, et al. Screening for serious mental illness in the general population. Arch Gen Psychiatry. 2003;60(2):184–9.
73. Perera FP, Rauh V, Whyatt RM, Tsai WY, Tang DL, Diaz D, Hoepner L, Barr D, Tu YH, Camann D, et al. Effect of prenatal exposure to airborne polycyclic aromatic hydrocarbons on neurodevelopment in the first 3 years of life among inner-city children. Environ Health Persp. 2006;114(8):1287–92.
74. Tzivian L, Winkler A, Dlugaj M, Schikowski T, Vossoughi M, Fuks K, Weinmayr G, Hoffmann B. Effect of long-term outdoor air pollution and noise on cognitive and psychological functions in adults. Int J Hyg Environ Health. 2015;218(1):1–11.