Impact of the Ultra Low Emission Zone on lung function growth in children in London, UK: a prospective parallel cohort study

Helen E Wood, Hajar Hajmohammadi*, Rosamund E Dove*, James Scales*, Ivelina Tsocheva, Jasmine Chavda, Harpal Singh Kalsi, Louise Cross, Grainne Colligan, Luke Sartori, Jessica E Mitchell, Jessica Moon, Esther Lie, Kristian Petrovic, Mia Keating, Jason Le, Bill Day, Amanda Keighley, Cheryll Crichlow, Janina Mallari, Andrew Beddows, Nosha Assareh, Gregor Stewart, David Dajnak, Sean Beevers, Veronica Toffolutti, Monica Fletcher, Borislava Mihaylova, Frances Balkwill, Jonathan Grigg, Rosalind Raine, Mark Mon-Williams, Esther van Sluijs, Frank J Kelly, Christopher Newby, Beth Stuart, W James Gauderman, Aziz Sheikh, Gurch Randhawa†, Ian S Mudway†, Chris J Griffiths†*

Summary

Background Traffic-related air pollution is a risk factor for lung disease and early mortality. Clean air zones are public health policy interventions used to reduce traffic-related air pollution in urban areas, but evidence of their health benefits is limited. We describe a natural experiment study evaluating the impact of the Ultra Low Emission Zone (ULEZ) in London, UK. The primary aim was to assess the impact of the ULEZ on children's lung function growth trajectories, by comparing forced expiratory volume in 1 s (FEV_1) measurements over 5 years between children in London and Luton, UK.

Methods The Children's Health in London and Luton (CHILL) study is a prospective, two-arm, parallel cohort study. We recruited children aged 6–9 years from primary schools in central London (the original area of the ULEZ implementation) and Luton (a comparator site with no clean air zone). Children were excluded if they had symptoms of lung disease (excluding asthma) or learning or physical disabilities preventing them from giving informed assent. Lung function was measured by spirometry at annual school visits at baseline (before ULEZ implementation) and over the following 4 years. Annual residential exposures to nitrogen dioxide (NO_2) and particulate matter with aerodynamic diameters of less than 10 µm (PM_{10}) and less than 2·5 µm ($PM_{2·5}$) were estimated at each child's home address at 20 m² resolution using a validated dispersion modelling system. The primary outcome was the annual rate of lung function growth, measured as post-bronchodilator FEV_1 over the 5-year study period. We assessed growth trajectories against individualised residential exposure to NO_2, PM_{10}, and $PM_{2·5}$. We used mixed-effects linear regression to compare lung function growth between London and Luton and to examine associations between air pollution exposures and lung growth.

Findings Of 122 schools approached, 84 (69%) agreed to participate (44 schools in London; 40 schools in Luton). Of 9419 children invited, we recruited 3414 children (1664 in London and 1750 in Luton) between June 5, 2018, and April 4, 2019, before ULEZ implementation. 3209 (94·0%) of 3414 children were included in the FEV_1 analysis (1557 children in London, 1652 children in Luton). Children were similar across the two sites in terms of age, height, and weight, with a higher proportion of girls in London (867 [56%] of 1557) than in Luton (809 [49%] of 1652). Baseline adjusted FEV_1 was lower in London than Luton (difference –38 mL, 95% CI –58 to –18; p=0·0002). At baseline, children's modelled annual exposures to NO_2, the pollutant most reflective of exhaust emissions, were 18·95 µg/m³ (95% CI 18·51 to 19·38; p<0·0001) higher in London than in Luton. Over the follow-up period, children's FEV_1 growth increased by 10 mL/year (95% CI 5 to 15; p=0·0002) more in London than in Luton (233 mL/year *vs* 223 mL/year), with modelled residential exposures to NO_2 decreasing faster in London than in Luton (decreases of –3·77 µg/m³ per year in London *vs* –1·77 µg/m³ per year in Luton; p<0·0001). After 4 years, mean FEV_1 reached parity across sites: 2283 mL (2207 to 2354) in London; 2282 mL (2185 to 2376) in Luton. The proportion of children with clinically impaired lung function fell from 184 (14%) of 1280 children to 51 (9%) of 585 children in London, and from 126 (9%) of 1339 children to 41 (7%) of 606 children in Luton.

Interpretation Introduction of the London ULEZ was associated with improved lung function growth trajectories in children in London compared with children in Luton, suggesting that previous deficits in lung development were restored. This evidence supports wider implementation of clean air zones as a public health intervention.

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*Contributed equally to this work

†Contributed equally to this work

Wolfson Institute of Population Health, Faculty of Medicine and Dentistry, Queen Mary University of London, London, UK (H E Wood DPhil, H Hajmohammadi PhD, R E Dove PhD, J Scales PhD, H S Kalsi PhD, L Cross SRN, G Colligan MRes, L Sartori BSc, J E Mitchell MSc, B Day, A Keighley MA, C Crichlow BEd(Hons), V Toffolutti PhD, Prof B Mihaylova DPhil, Prof B Stuart PhD, Prof C J Griffiths DPhil); **Institute for Health & Wellbeing Research, University of Bedfordshire, Luton, UK** (I Tsocheva PhD, J Chavda MSc, Prof G Randhawa PhD); **Centre of the Cell, Queen Mary University of London, London, UK** (J Moon MChem, E Lie BSc, K Petrovic PGCE, M Keating PGCE, J Le MDes, Prof F Balkwill PhD); **Royal London Hospital, Barts Health NHS Trust, London, UK** (J Mallari BSc); **Environmental Research Group and MRC Centre for Environment and Health, School of Public Health, Imperial College London, London, UK** (A Beddows PhD, N Assareh PhD, G Stewart PhD, D Dajnak PhD, S Beevers PhD, Prof F J Kelly PhD, I S Mudway PhD); **Nuffield Department of Primary Care Health Sciences, University of Oxford, Oxford, UK** (Prof M Fletcher MSc,

Prof A Sheikh MD, Prof C J Griffiths); **Usher Institute, University of Edinburgh, Edinburgh, UK** (Prof M Fletcher); **Nuffield Department of Population Health, University of Oxford, Oxford, UK** (Prof B Mihaylova); **Blizard Institute, Faculty of Medicine and Dentistry, Queen Mary University of London, London, UK** (Prof J Grigg MD); **Department of Primary Care and Population Health, University College London, London, UK** (Prof R Raine PhD); **Bradford Institute for Health Research, Bradford, UK** (Prof M Mon-Williams PhD); **MRC Epidemiology Unit, School of Clinical Medicine, University of Cambridge, Cambridge, UK** (Prof E van Sluijs PhD); **School of Medicine, University of Nottingham, Nottingham, UK** (C Newby PhD); **Division of Biostatistics and Health Data Science, Department of Population and Public Health Sciences, University of Southern California, Los Angeles, CA, USA** (Prof W J Gauderman PhD)

Correspondence to: Dr Helen E Wood, Wolfson Institute of Population Health, Faculty of Medicine and Dentistry, Queen Mary University of London, London E1 2AB, UK **helen.wood@qmul.ac.uk**

Research in context

Evidence before this study

Exposure to traffic-related air pollution is strongly linked to impaired lung development in children and adolescents. Failure to reach maximal lung growth in early adulthood is an established risk factor for asthma, chronic obstructive pulmonary disease, cardiometabolic disease, and earlier mortality. Clean air zones are the dominant public health intervention being considered globally to improve traffic-related air pollution in urban environments, but it remains unknown whether they improve poor lung growth. Systematic reviews addressing health effects of clean air zones have been published, the most recent in 2023, published in *The Lancet Public Health*, focusing on low-emission zones. These showed that no studies have addressed impacts of clean air zones on lung growth or development.

We searched MEDLINE, Web of Science, Cochrane, and Transport Research International Documentation to identify literature from Jan 23, 2023, the final search date of the most recent systematic review, to Aug 15, 2025. We used the same search terms, combining terms including "low emission? zon* or clean air zon*", "congestion or traffic or road?", and "charg* or payment? or tax* or zone? or zoning" with health outcomes, with no restrictions on language of publication. This provided a combined search with no language restrictions covering the period from 1946 to August, 2025. We found no studies or systematic reviews addressing impacts of clean air zones on lung growth or development. Our previous evaluation of the original London Low Emission Zone (implemented in 2008–12) established an inverse association between traffic-related pollution and lung volume in schoolchildren in London, UK. However, that 5-year study used a repeated cross-sectional design that assessed different groups of 8–9-year-olds each year. Although the study showed that children in higher pollution areas had smaller lungs, it did not include the longitudinal data needed to evaluate individual growth rates. The current study addresses these limitations by using a more robust prospective cohort design. We undertook baseline measurements before policy implementation, followed up the same children sequentially to establish lung function growth trajectories, and included a comparator cohort from a site without a clean air zone (ie, Luton).

Added value of this study

To our knowledge, we provide the first evidence that air quality improvements following introduction of a clean air zone are associated with improved lung growth trajectories in children. We established a prospective parallel cohort of 3414 school-children aged 6–9 years before the implementation of London's Ultra Low Emission Zone (ULEZ), the world's largest clean air zone. The cohort comprised children recruited from central London (where ULEZ was first implemented) and from Luton (a comparator site with no clean air zone). We measured lung function annually over 5 years, capturing data in the year preceding ULEZ implementation and for 4 subsequent years. We found that children's lung growth accelerated following ULEZ implementation and that improved air quality was strongly related to lung growth.

Implications of all the available evidence

Our findings address a critical gap in the literature, showing that air quality improvements following implementation of a clean air zone are associated with accelerated lung growth trajectories, a key parameter of children's development relating to risk of future morbidity and mortality. Children continue to be exposed to poor air quality in cities globally. Decision makers globally should consider accelerating the introduction of clean air zones to improve children's lung health.

See **Online** for appendix

Introduction

Studies from North America[1] and Europe,[2] and our own previous study in London (UK)[3] have shown that exposure to traffic-related air pollution harms lung development in children, causing suboptimal lung function and lung growth trajectories, a major risk factor for chronic lung disease and early mortality.[4,5] Improvements in air quality are associated with improved lung function trajectories in adolescents.[6,7] These studies raise the possibility that targeted public health policies to improve urban air quality might benefit lung development in children.

Clean air zones are the dominant public health intervention designed to cut pollution. They have been widely adopted across Europe.[8] Many other cities are implementing clean air zones through the C40 Cities Clean Air Accelerator programme.[9] However, evidence of their effectiveness on health outcomes is scant. In April, 2019, the Ultra Low Emission Zone (ULEZ) was introduced, initially in central London and expanding in 2021 and 2023 to cover an area of 1500 km² across London and a population of 9 million (appendix p 2). This initiative created the opportunity for a natural experimental evaluation of its health effects.[10] The aim of this study was to assess the impact of the ULEZ on children's lung function by comparing lung growth trajectories between children in London and children in the comparator site of Luton.

Methods

Study design

The Children's Health in London and Luton (CHILL) study was a prospective parallel cohort study. We established the CHILL cohort in the 12 months preceding ULEZ implementation, approaching all primary schools within or bordering the ULEZ in central London, as well as all those in Luton, a large town 32 miles northwest of London (population around 225 000).[11,12] We chose Luton as a comparator site because it met five criteria: (1) being geographically distant and therefore free from potential contamination (spillover effects) from the ULEZ;

(2) having a broadly similar population demographic to that of London; (3) having no plans to enact any form of low emission zone; (4) having a similar mixture of local pollutant sources to those in central London; and (5) having locations with established continuous air quality monitoring sites that provided freely available long-term trend data. These air quality monitoring sites had to demonstrate urban background nitrogen dioxide (NO_2) concentrations near 40 mg/m³, with confirmed exceedances of this threshold at roadside locations.[13] Although Luton had a similar pollutant mix to London, variables such as NO_2, particulate matter with an aerodynamic diameter less than 10 µm (PM_{10}), and particulate matter with an aerodynamic diameter less than 2·5 µm ($PM_{2·5}$) were lower in Luton than in our central London study area.

Children aged 6–9 years were invited to participate. All parents completed a consent form and demographic questionnaire, and all children provided verbal assent (appendix p 20). Children were excluded if they had symptoms of lung disease (excluding asthma) or learning or physical disabilities that prevented them from giving informed assent or performing study procedures.

The study was approved by Queen Mary University of London Research Ethics Committee (April 4, 2018, QMERC2018/08) and University of Bedfordshire Ethics Committee of the Institute for Health Research (April 13, 2018, IHREC842).

Procedures

Recruitment and baseline assessments began on June 5, 2018, and were completed before the ULEZ was implemented on April 8, 2019. Lung function was measured by spirometry (Vitalograph) according to American Thoracic Society/European Respiratory Society (ATS/ERS) guidelines[14] at annual school visits at baseline and over the following 4 years (appendix p 20). The COVID-19 pandemic disrupted school visits between March, 2020, and April, 2021. To compensate, we added a fifth year of data collection, recruiting secondary schools to which the older participants had moved (at age 11 years). Further details of COVID-19 restrictions and protocol changes are given in the appendix (p 20).

Annual residential exposures at each child's home address were estimated at 20 m² resolution using the validated London Toolkit/KCLurban dispersion modelling system for NO_2, PM_{10}, and $PM_{2·5}$. This system coupled WRF-CMAQ (version 5.3.1) for regional background with ADMS-Roads (version 6) for street-scale dispersion across five annual runs.[15] Model performance was validated against 162 monitoring sites, yielding correlation coefficients of 0·70–0·88 and a normalised mean bias of 4% or less (appendix pp 22–28). These individualised residential exposure assignments for NO_2, PM_{10}, and $PM_{2·5}$ were utilised as the primary independent variables within the health models to determine their impact on children's lung function growth trajectories.

Outcomes

The primary outcome was to assess the impact of the ULEZ on children's lung function growth trajectories, by comparing measurements of forced expiratory volume in 1 s (FEV_1) over 5 years between children in London and children in the comparator site of Luton.

Secondary outcomes,[15] including the impacts of the ULEZ on physical activity, cognitive function, and health-care utilisation, are beyond the scope of this paper and will be reported separately.

Statistical analysis

The sample size calculation, and revised calculations post-COVID-19, have been published[11] and are available in the appendix (p 19). We designed our study to achieve 90% power at a 0·05 significance level for detecting a difference in FEV_1 growth of 15 mL/year between the London and Luton cohorts. This design required a total of 3200 children, including 1600 children in London and 1600 children in Luton. We aimed to recruit 40 schools per site with 40 children aged 6–9 years from each school. We extended the study by 1 year to compensate for loss of data collection during the COVID-19 pandemic (follow-up years 1 and 2).

We used a mixed-effects linear regression model with random effects for child, age, and school to account for clustering. All models included adjustment for the following covariates: sex (categorical: male, female), height (cm), ethnicity (categorical: White, Asian or Asian British, Black or Black British, other), Index of Multiple Deprivation 2019 (IMD 2019, deciles)[16] and parent-reported asthma diagnosis (binary: yes or no). Because age and height were highly correlated, we first regressed height on age and used height residuals in the lung function models.

Our analysis followed a structured modelling approach, first addressing the primary hypothesis of whether lung function growth differed between sites, then examining whether time trends in air pollutants differed between sites, and finally examining the association between air pollution and lung function growth.

We developed three models. The main model (model 1) was lung function growth by site, which examined difference in lung function growth between London and Luton by including age, site, and their interaction, adjusting for covariates. The interaction term, site×age, was included to indicate any difference in lung function growth (slope) between London and Luton.

The second model (model 2) was air pollution changes by site. This model assessed changes in residential air pollution exposures over time in both London and Luton by regressing child-specific and time-specific air pollution assignments on age, site, and their interaction. The interaction term, site×age, was included to indicate any differences in the effect of time on each pollutant between London and Luton.

The final and third model (model 3) was the association between air pollution and lung function

growth, which was assessed using two variants. The first one (model 3a) was used to investigate the overall effect of air pollution on lung function growth across the entire cohort by including the interaction term pollutant × age, without site-specific effects. This model established the relationship between each pollutant and lung function growth for the whole cohort. The second one (model 3b) was used to examine site-specific differences in air pollution effects on growth by incorporating a three-way interaction (pollutant × age × site) and comparing London and Luton. This model established any differences in the effect of each pollutant on lung function growth between London and Luton (ie, effect modification by site). We also included two-way interactions of air pollution with age and air pollution with site in the third model.

For each of the three models, underlying assumptions were carefully checked, including linearity, homoscedasticity, and normality of residuals. Stratification analyses were conducted to further examine differences in lung function growth and the interaction between growth and air pollution by sex, ethnicity, socioeconomic deprivation (IMD <5 *vs* IMD ≥5), and parent-reported asthma diagnosis. No formal adjustment for multiplicity was made for secondary outcomes. We did sensitivity analyses to examine whether the associations between exposure to air pollution and lung function growth were affected by exposure to incomplete follow-up and second-hand smoke (appendix p 22).

The analysis for model 1 included all children who provided at least one acceptable lung function measurement (3209 of 3414 children for FEV_1 and 3094 of 3414 children for forced vital capacity [FVC]); for model 3, it included all children who additionally had air pollution exposure estimates (3073 of 3209 children for FEV_1 and 2960 of 3094 children for FVC; missing values resulted from residential address not being provided or being incomplete); model 2 included all children with at least one acceptable FEV_1 measurement and air pollution exposures (3073 children). FEV_1 and FVC measurements were deemed acceptable if they met the criteria defined in the 2005 ATS/ERS guidelines.[14] Manoeuvres were independently assessed for acceptability by HSK and JS.

Descriptive statistics are presented as means (SD) for normally distributed continuous variables, and as median (IQR) for those not normally distributed. Categorical variables are presented as counts and

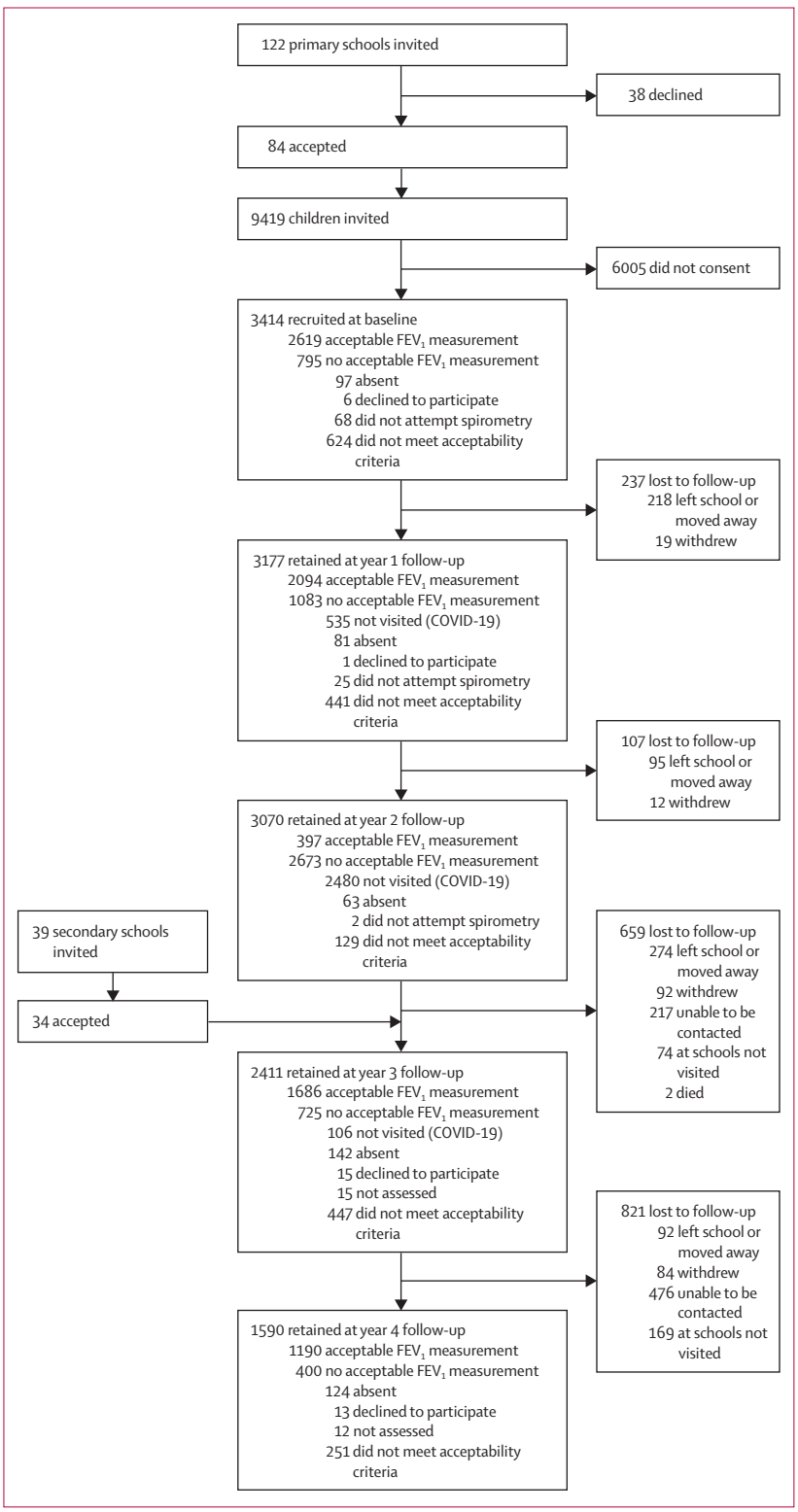

Figure 1: **Study profile**
Primary school refers to a school for children aged 5–11 years, and secondary school refers to a school for children aged 11–16 years. Acceptable FEV_1 measurements were those that met the criteria defined in the 2005 American Thoracic Society/European Respiratory Society guidelines.[14] Measurements that failed the acceptability criteria were not included in analyses. Children who declined to participate did not take part in any aspect of the health assessment visit. Children who did not attempt spirometry might have taken part in other aspects of the health assessment visit but declined to undertake spirometry or were unable to due to physical or learning disabilities. FEV_1=forced expiratory volume in 1 s.

	London	Luton
Number of participants included in analysis*	1557	1652
Age, years	8·0 (0·9)	7·8 (0·8)
Sex		
Female	867 (56%)	809 (49%)
Male	690 (44%)	843 (51%)
Height, cm	129·3 (8·1)	127·7 (7·4)
Weight, kg	29·1 (7·4)	28·1 (6·8)
BMI, kg/m²	17·2 (2·9)	17·0 (2·8)
Ethnicity		
White	476 (31%)	634 (38%)
Asian or Asian British	346 (22%)	644 (39%)
Black or Black British	294 (19%)	124 (8%)
Mixed	207 (13%)	130 (8%)
Other	135 (9%)	40 (2%)
Missing	99 (6%)	80 (5%)
Socioeconomic deprivation†	3·6 (1·7)	4·0 (2·1)
Missing	155 (10%)	130 (8%)
Smoking in the household	242 (16%)	383 (23%)
Missing	13 (1%)	11 (1%)
Parent-reported asthma diagnosis	140 (9%)	212 (19%)
Missing	82 (5%)	73 (4%)

Values are mean (SD) for continuous variables and n (%) for categorical variables. *Children were excluded from analysis if they did not provide a single acceptable measurement of post-bronchodilator forced expiratory volume in 1 s from any of the five annual health assessments. †Socioeconomic deprivation was assessed using deciles of the Index of Multiple Deprivation 2019,[16] in which decile 1 represents the most deprived 10% of areas, while decile 10 represents the least deprived 10%.

Table 1: **Participant characteristics at baseline by study site**

	Coefficient (95% CI)*	p value
FEV_1 (mL)		
Per year in Luton	223 (220 to 227)	<0·0001
Per site at baseline		
Luton	Ref	
London	–38 (–58 to –18)	<0·0001
Site × year		
Luton	Ref	
London	10 (5 to 15)	<0·0001
FVC (mL)		
Per year in Luton	252 (248 to 256)	<0·0001
Per site at baseline		
Luton	Ref	
London	–55 (–78 to –31)	<0·0001
Site × year		
Luton	Ref	
London	7 (2 to 13)	<0·0001

"Year" indicates the effect of time on lung function growth (whether FEV_1 and FVC differed over time across the 5-year study). "Site" indicates the effect of site at baseline on lung function growth (whether FEV_1 and FVC differed between sites [London, intervention site *vs* Luton, comparator site] before implementation of the Ultra Low Emission Zone). "Site × year" indicates the effect of any interaction between time and site (whether there was a difference in the effect of time on FEV_1 and FVC between sites). FEV_1=forced expiratory volume in 1 s. FVC=forced vital capacity. *Adjusted for sex, residuals of height, ethnicity, Index of Multiple Deprivation 2019, and parent-reported asthma diagnosis.

Table 2: **Model results for lung function growth**

percentages. All statistical analyses were undertaken using STATA (version 18).

Role of the funding source

The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

Of 122 schools approached, 84 (69%) agreed to participate (44 of 66 schools in London; 40 of 56 schools in Luton; figure 1). We invited 9419 children of whom 3414 (36·2%) provided consent to participate (1664 [36·7%] of 4533 in London; 1750 [35·8%] of 4886 in Luton). Children not providing at least one acceptable outcome measure of FEV_1 were excluded from all analyses, and those not providing at least one acceptable measure of FVC were excluded from the analyses for that outcome. For the FEV_1 analysis, 3209 (94·0%) of 3414 children were included (1557 children in London, 1652 children in Luton), providing 7993 individual measurements across 5 years. For the FVC analysis, 3094 (90·6%) children were included (1503 in London, 1591 in Luton).

Children were similar at recruitment across the two sites for age, height, and weight, with a higher proportion of girls in London (867 [56%] of 1557 children) than in Luton (809 [49%] of 1652 children; table 1, appendix p 6).

Both cohorts were ethnically diverse (table 1). The largest group in London was White children (476 [31%] of 1557 children), followed by Asian or Asian British children (346 [22%]), Black or Black British children (294 [19%]), and children of mixed ethnicity (207 [13%]). In Luton, the largest group was Asian or Asian British children (644 [39%] of 1652), with a similar proportion of White children (634 [38%]), and smaller proportions of children of mixed ethnicity (130 [8%]) and Black or Black British children (124 [8%]). Both cohorts lived in areas of moderate socioeconomic deprivation, according to IMD 2019 (appendix p 6).[17,18] The average annual loss to follow-up was 16·5% per year, with 1590 participants remaining at the end of follow-up (46·6% of those recruited; figure 1).[12] Retention was similar across sites (754 [45%] of 1664 children in London, 836 [48%] of 1750 children in Luton). The baseline characteristics of children retained throughout the study and those lost to follow-up were similar, except for a higher proportion of missing data at baseline among those lost to follow-up (reflecting lower parental questionnaire returns; appendix p 7).

At baseline, 184 (14%) of 1280 children with successful FEV_1 measurement in London had a clinically low FEV_1 (pre-bronchodilator FEV_1 <80% of predicted value) compared with 126 (9%) of 1339 children in Luton

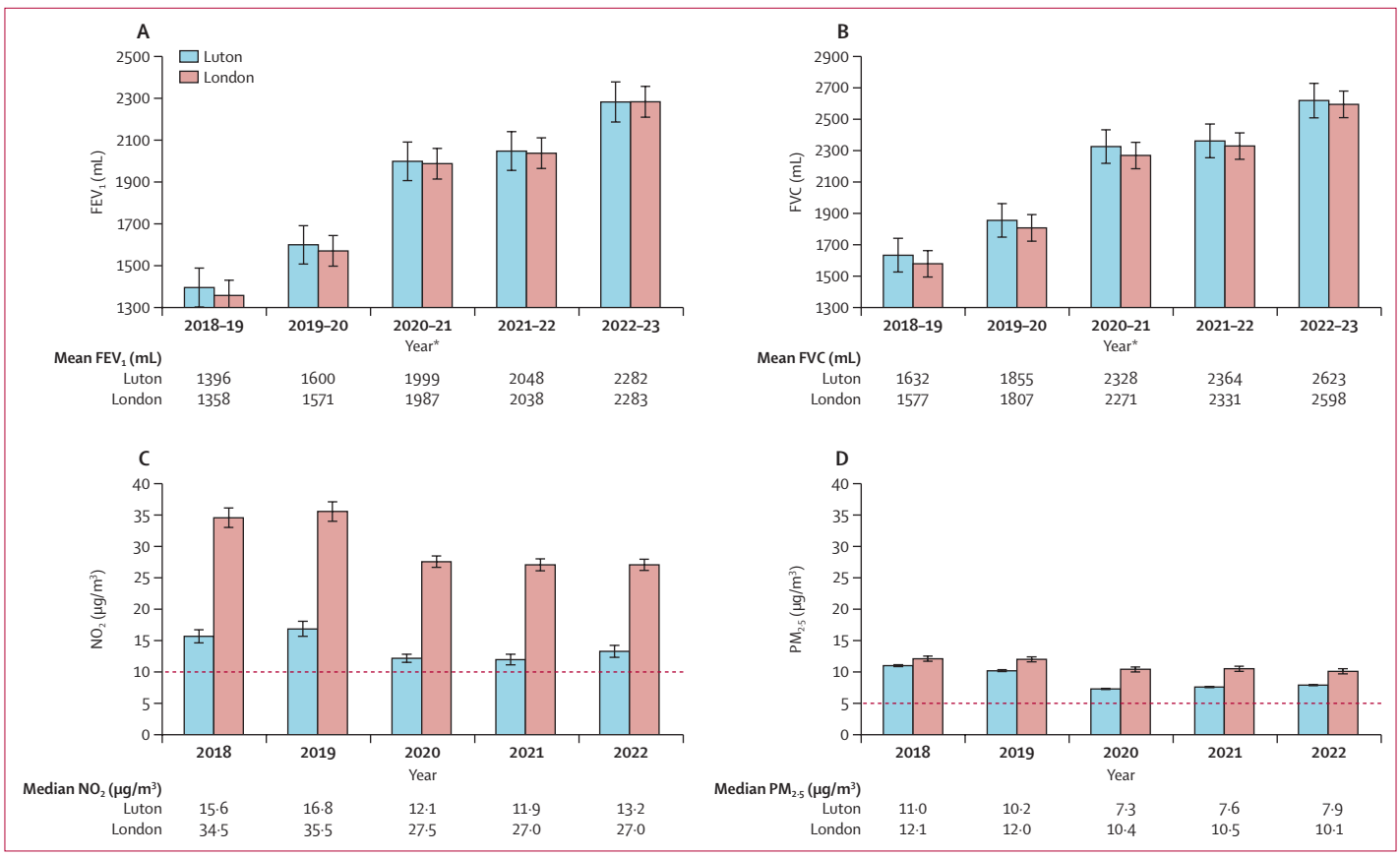

***Figure 2*: Lung function and air pollution by site and year**

Lung function values (FEV_1 [A] and FVC [B]) are adjusted means from our model examining lung function growth by site (adjusted for sex, residuals of height, ethnicity, Index of Multiple Deprivation 2019, and parent-reported asthma diagnosis). Air pollution values (NO_2 [C] and $PM_{2·5}$ [D]) are medians of individual estimated annual exposures, based on residential address. Error bars are 95% CIs (for FEV_1 and FVC) and IQR (for NO_2 and $PM_{2·5}$). Red dashed lines on panels C and D are exposure limits from the WHO Air Quality Guideline for NO_2 and $PM_{2·5}$, respectively. FEV_1=forced expiratory volume in 1 s. FVC=forced vital capacity. NO_2=nitrogen dioxide. $PM_{2·5}$=particulate matter with an aerodynamic diameter less than 2·5 µm. *Date ranges for the five visits are provided in the appendix (p 20).

(appendix p 8). At the end of the study, these values had dropped to 51 (9%) of 585 children in London and 41 (7%) of 606 children in Luton (appendix p 8). The FEV_1/FVC ratio was similar between children in London (87·8% [SD 6·3]) and Luton (87·2% [6·4]) at baseline and changed little across the 5-year study in either site (appendix p 8).

Our main model, examining lung function growth by site, showed that children's baseline adjusted mean FEV_1 (before ULEZ implementation) was significantly lower in London than in Luton (difference −38 mL, 95% CI −58 to −18; p=0·0002; table 2). Across the following 4 years, children's FEV_1 increased by 223 mL/year (95% CI 220 to 227) in Luton. In London, however, children's lung function growth was significantly greater, with an additional increase in FEV_1 of 10 mL/year (5 to 15; p=0·0002; 233 mL/year), such that by the end of the study, the adjusted mean FEV_1 reached parity (2283 mL [95% CI 2207 to 2354] in London; 2282 mL [2185 to 2376] in Luton; figure 2A). Similarly, FVC was significantly lower at baseline in London (difference −55 mL, −78 to −31; p<0·0001) and increased by a greater amount over follow-up (7 mL/year, 2 to 13; p=0·0071) compared with children in Luton (table 2). After 4 years, the difference in FVC across sites fell by more than half, with FVC only 25 mL lower in London (2598 mL, 2513 to 2683) than in Luton (2623 mL, 2513 to 2734; figure 2B).

Stratifying by sex indicated that estimated FEV_1 growth was significantly higher in girls than in boys (p=0·018), while for FVC growth, the difference between boys and girls was not significant. Stratification by ethnicity revealed that for both FEV_1 and FVC, lung function growth was significantly higher in White children than in children from other ethnic groups. Differences in growth rates between White and Asian or Asian British children and between White and Black or Black British children were significant (p<0·0001), based on the age×ethnicity interaction terms (appendix p 21). Across both sites, there was no difference in estimated lung function growth when stratified by deprivation. We additionally ran a stratification analysis using the main model to investigate any impact of parent-reported asthma diagnosis on lung function growth, finding no significant difference between the two groups (appendix p 20).

The appendix (p 9) shows the median estimated residential annual exposures to air pollutants by site and study year. Our second model, examining trends in exposure to air pollution estimates over time, showed that at baseline, children's estimated mean annual

	Coefficient (95% CI)*	p value
NO_2 (μg/m³)		
Per year in Luton	−1·77 (−1·91 to −1·62)	<0·0001
Per site at baseline		
Luton	Ref	
London	18·95 (18·51 to 19·38)	<0·0001
Site × year		
Luton	Ref	
London	−2·00 (−2·21 to −1·78)	<0·0001
PM_{10} (μg/m³)		
Per year in Luton	−1·26 (−1·35 to −1·17)	<0·0001
Per site at baseline		
Luton	Ref	
London	2·03 (1·76 to 2·30)	<0·0001
Site × year		
Luton	Ref	
London	0·61 (0·47 to 0·74)	<0·0001
$PM_{2·5}$ (μg/m³)		
Per year in Luton	−1·06 (−1·12 to −0·99)	<0·0001
Per site at baseline		
Luton	Ref	
London	1·38 (1·19 to 1·58)	<0·0001
Site × year		
Luton	Ref	
London	0·28 (0·18 to 0·38)	<0·0001

"Year" indicates the effect of time (whether NO_2, PM_{10}, and $PM_{2·5}$ differed over time across the 5-year study). "Site" indicates the effect of site at baseline (whether NO_2, PM_{10}, and $PM_{2·5}$ differed between sites [London, intervention site *vs* Luton, comparator site] before implementation of the Ultra Low Emission Zone). "Site × year" indicates the effect of any interaction between time and site (whether there is a difference in the effect of time on NO_2, PM_{10}, and $PM_{2·5}$ between sites). NO_2=nitrogen dioxide. PM_{10}=particulate matter with an aerodynamic diameter less than 10 μm. $PM_{2·5}$=particulate matter with an aerodynamic diameter less than 2·5 μm. *Adjusted for sex, residuals of height, ethnicity, Index of Multiple Deprivation 2019, and parent-reported asthma diagnosis.

Table 3: **Model results for air pollution changes**

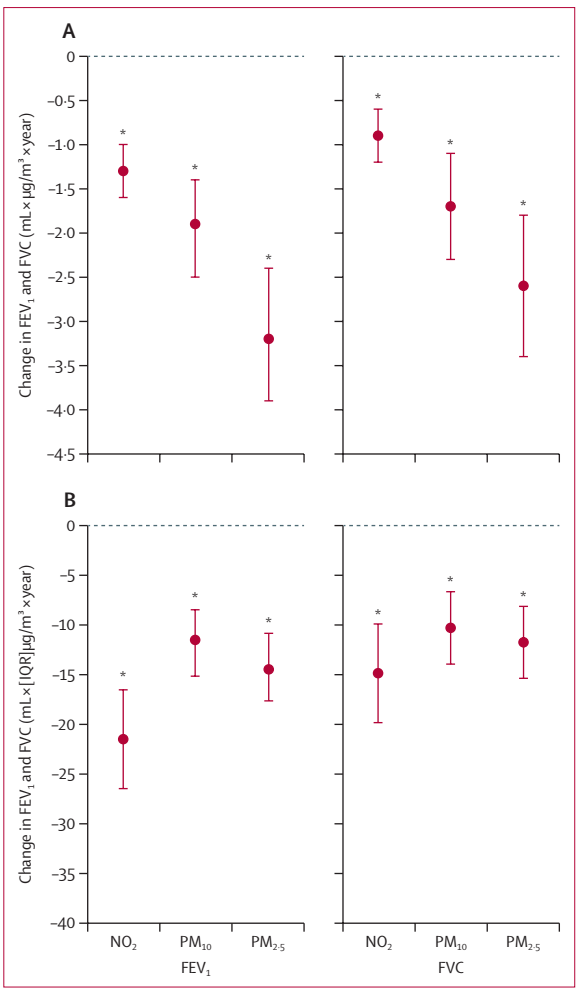

Figure 3: **Association (coefficient [95% CI]) between air pollution (μg/m³) and the annual rate of lung function growth (mL/year) across the entire cohort, presenting the exposure–growth association (not policy time trends)**

Data are illustrated per unit change in pollutant (A) and per IQR (B). Associations are adjusted for interaction of age and site, interaction of air pollution and age, interaction of air pollution and site, sex, residuals of height, ethnicity, Index of Multiple Deprivation 2019, and parent-reported asthma diagnosis. For both the FEV_1 and FVC analyses, the IQRs were 16·51 μg/m³ (NO_2), 6·06 μg/m³ (PM_{10}), and 4·52 μg/m³ ($PM_{2·5}$).FEV_1=forced expiratory volume in 1 s. FVC=forced vital capacity. NO_2=nitrogen dioxide. $PM_{2·5}$=particulate matter with an aerodynamic diameter less than 2·5 μm. PM_{10}=particulate matter with an aerodynamic diameter less than 10 μm. *p<0·0001.

exposures to air pollution were significantly higher in London than in Luton (by 18·95 μg/m³ for NO_2 [95% CI 18·51 to 19·38; p<0·0001], 2·03 μg/m³ for PM_{10} [1·76 to 2·30; p<0·0001], and 1·38 μg/m³ for $PM_{2·5}$ [1·19 to 1·58; p<0·0001]; table 3). In Luton, estimated mean annual exposures decreased significantly by an average of −1·77 μg/m³ per year (−1·91 to −1·62; p<0·0001) for NO_2, −1·26 μg/m³ per year (−1·35 to −1·17; p<0·0001) for PM_{10}, and −1·06 μg/m³ per year (−1·12 to −0·99; p<0·0001) for $PM_{2·5}$. However, in London, annual NO_2 exposures decreased significantly faster, by an additional decrease of −2·00 μg/m³ per year (−2·21 to −1·78; p<0·0001), while average PM exposures decreased significantly slower than in Luton (by 0·61 μg/m³ per year less [0·47 to 0·74; p<0·0001] for PM_{10} and 0·28 μg/m³ per year less [0·18 to 0·38; p<0·0001] for $PM_{2·5}$). The overall decrease in mean estimated NO_2 exposure in London during the study was more than double that in Luton (15·1 *vs* 7·1 μg/m³), while the decrease in mean estimated PM_{10} exposure was approximately half in London compared with Luton (2·6 *vs* 5·0 μg/m³), and the decrease in mean $PM_{2·5}$ was similar between sites (3·1 *vs* 4·2 μg/m³). Figure 2 shows the medians of individual estimated annual exposures for NO_2 and $PM_{2·5}$, along with WHO Air Quality Guidelines, illustrating that for both pollutants, estimated annual exposures remained above these guidelines by the end of the study in both London and Luton. The appendix (p 9) presents descriptive annual modelled residential exposure assignments for participants contributing lung function data; it should not be

interpreted as a measured policy time series. The 2019 estimates included both pre-ULEZ and post-ULEZ months; the largest observed NO_2 step change occurred between 2019 and 2020 during COVID-19-related travel disruption, and subsequent annual medians plateaued.

Long-term air pollution exposure (NO_2, PM_{10}, and $PM_{2·5}$) across both sites was associated with reductions in children's annual lung function growth rate. An IQR increase in estimated NO_2 exposure (16·51 μg/m³) was associated with decreases in FEV_1 of −21·46 mL/year (95% CI −26·41 to −16·51) and decreases in FVC of −14·86 mL/year (−19·81 to −9·91). IQR increases in estimated exposures to PM_{10} (6·06 μg/m³) and $PM_{2·5}$ (4·52 μg/m³) were associated with decreases in FEV_1 of −11·51 mL/year (−15·15 to −8·48) and −14·46 mL/year (−17·63 to −10·85), respectively, and with decreases in FVC of −10·30 mL/year (−13·94 to −6·67) and −11·75 mL/year (−15·37 to −8·14), respectively (figure 3; and appendix pp 15–16). Exploratory spline-based exposure–response analyses suggested some non-linearity for $PM_{2·5}$ and PM_{10} at the upper end of the exposure distribution (appendix p 17). However, these estimates in this range were based on sparse data and might be influenced by residual confounding, co-pollutant correlation, or exposure measurement error. The more reliable overall and quartile-based associations are reported in the appendix (p 16).

The appendix (p 12) shows results for the model with the effects of air pollutants stratified by site, including the three-way interaction (model 3b). Exploratory subgroup analyses by sex and deprivation index did not show any significant differences in the negative associations of air pollutants on estimated lung function growth. Differences in the impact of air pollution on annual lung function growth were significant for Asian and Black children compared with White children ($p<0·05$), indicating larger pollution-related growth deficits in these groups. In contrast, the estimated impact for children of Mixed ethnicity did not differ significantly from that observed in White children (appendix p 21).

We examined whether the association between air pollution exposure and lung function growth differed between children who completed all five annual assessments and those with incomplete follow-up. To assess this, we extended the primary mixed-effects model to allow the estimated impact of air pollution on annual lung function growth to vary by follow-up completion status, while retaining all original covariates and site terms. In single-pollutant models (NO_2, PM_{10}, and $PM_{2·5}$), there was no evidence that the pollution-related growth deficit differed between children with complete and incomplete follow-up. The estimated differences in annual growth associated with air pollution were very similar between the two groups and not significant, suggesting that incomplete follow-up did not materially bias the estimated association (appendix p 22).

Children's exposure to second-hand smoke was assessed via parental questionnaire. Sensitivity analysis revealed that exposure to second-hand smoke was not significantly associated with lung function growth (appendix p 22).

Discussion

In the CHILL study, the reduction in air pollution exposure, predominantly due to reduced NO_2 from vehicle exhaust emissions, following the introduction of London's ULEZ was associated with improvements in children's lung function growth trajectories.[19,20] Before ULEZ implementation, traffic-related pollution exposures were higher in London than Luton, and children in London had lower FEV_1 and FVC on average than those in Luton. Over the following 4 years, children's lung function growth rate in London exceeded that in Luton, resulting in average FEV_1 reaching parity across the two sites by the end of the study. We also observed a greater decrease in the proportion of children with clinically impaired lung function in London than in Luton. During the same period, modelled year-on-year residential exposures to NO_2 fell more rapidly in London than in Luton.

We have previously reported reduced lung function volumes in London schoolchildren associated with elevated exposures to traffic-related pollution.[3] The lung function deficit observed at the start of the present study in London children compared with Luton children is consistent with that work.

At the study's outset, exposure to NO_2 in London was more than double that in Luton. This finding reflected the well documented challenges posed by diesel vehicle emissions in central London, which prompted a series of policy interventions aimed at improving air quality,[3,19,21,22] culminating in the introduction and phased expansion of the ULEZ from April, 2019, to August, 2023.[23]

The ULEZ has been the most impactful strategy for air pollution reduction in London to date, with well documented city-wide changes in air pollution, demonstrating significant reductions in roadside NO_2 concentrations (although evidence for improvements in $PM_{2·5}$ has been less consistent).[23,24] In this study, the exposure trend model indicates that $PM_{2·5}$ and PM_{10} declined at both sites, with faster average declines in Luton than in London, whereas NO_2 declined more rapidly in London. This divergence is consistent with pollutant-specific behaviour and the primary focus of the ULEZ on exhaust emissions. NO_2 is closely linked to road-traffic exhaust and is therefore expected to be comparatively responsive to changes in vehicle fleet composition and compliance within the ULEZ. By contrast, $PM_{2·5}$ and PM_{10} mass comprise a heterogeneous mixture, with substantial contributions from regional background and non-exhaust sources (eg, brake and tyre wear, resuspension), which are less directly targeted by emissions standards and might be more weakly reflected

in annual residential-address estimates. Differences in PM trends between London and Luton cannot therefore be straightforwardly attributed to the ULEZ. Any policy-related PM signal is more readily attenuated by spatial averaging and by measurement and modelling uncertainty.

In Luton, during the period of our study, actions taken by the local council to improve air quality included development of cycling and walking networks and workplace travel plans, anti-idling campaigns and reduced speed limits, improvements in town centre car parks, and air quality workshops for schoolchildren.[13] London's ULEZ is, by comparison, a much more comprehensive air quality improvement policy, producing larger declines in traffic-related pollution.

Our findings accord with studies in North America and Sweden, which documented improving lung function growth in children and adolescents relating to improvements in air quality.[6,7]

Triangulation with other outcomes and qualitative enquiry can complement associations found in natural experiment studies to support potential causality.[25] Following ULEZ implementation, we found that children in London were four times as likely to switch from inactive to active travel to school, compared with children in Luton,[26] and that parents, children, and teachers described the air in London as cleaner, traffic less congested, and asthma less troublesome.[27] Furthermore, a recent parallel interrupted time series evaluation showed that ULEZ implementation was associated with reduced cardiovascular and all-cause hospitalisation in adults.[28] These complementary findings suggest the ULEZ might have a societal, systemic impact, extending beyond air quality improvement. Implementation of a clean air zone in Bradford, UK, was followed by reductions in primary care consultations for respiratory and cardiovascular disease and falls in NO_2 concentrations,[29] while a recent systematic review showed beneficial impacts of clean air zones on cardiovascular outcomes.[30]

Strengths of our study include its prospective parallel longitudinal design, with a distant comparator site to avoid spillover effects; the inclusion of comparably diverse populations with significant traffic-related pollution exposures; and a statistical power sufficient to detect clinically meaningful differences in lung function growth. Our study benefited from high-quality spirometry across the two sites, with external validation. We used high-resolution modelled annual air pollutant exposures at the level of residential address, estimated using a well validated model. Residential annual estimates smooth near-road microenvironments and exposures during commutes and while at school, and therefore likely underestimate ULEZ-relevant changes. Differences in baseline characteristics between sites were accounted for in our analyses by adjusting for covariates. Extending data collection compensated for school closures during the COVID-19 pandemic.

Our study has limitations. We could not ensure that either the participating schools or children were entirely representative of their local populations. However, participation rates for schools (over two-thirds) and children (over one-third) were high, and their demographic characteristics were broadly similar to local borough data available at the start of the study (appendix p 18). Representative sampling in a school setting would not be logistically or ethically practical, hence there might have been some self-selection bias that we were unable to control for. Other studies have used opt-out approaches, but this is generally not favoured by ethics committees where health-relevant data are being collected. We undertook extensive engagement with children and parents at the schools during recruitment to encourage participation.

Our exposure assessment used high-resolution (20 m × 20 m) modelled residential ambient concentrations to capture intra-urban spatial gradients. Residential modelled concentration does not explicitly account for individual time–activity patterns. Empirical studies in comparable London children's cohorts[3] demonstrate that incorporating activity weighting does not materially alter health effect estimates in children. Any measurement error in ambient surrogates is expected to be classical, attenuating associations towards the null.[31] Thus, our findings are likely conservative estimates of the true health impacts.

Total loss to follow-up was 53% (1824 of 3414 children, with 1590 [47%] remaining after 5 years); however, this number was within our predictions, and participants leaving the cohort were similar to those remaining with respect to key covariates (appendix p 7). Including additional sites with or without clean air zones would have been helpful, but we were unable to extend our data collection, and no existing datasets with comparable lung function data were available. There might be unmeasured reasons why children in London could have more rapid lung function growth than those in Luton. The COVID-19 pandemic saw travel and population restrictions, including school closures, but these applied nationally, hence children in London and Luton would have had comparable experiences during and after COVID-19 isolation restrictions. Descriptive annual medians in the appendix (p 9) should be interpreted cautiously because they represent modelled residential exposure assignments, not a measured policy time series. The 2019 estimates include both pre-ULEZ and post-ULEZ months, while the largest observed NO_2 reduction occurred between 2019 and 2020 during COVID-19-related travel disruption, after which annual values plateaued. Inference therefore rests on longitudinal growth models using repeated individual exposure assignments rather than on simple year-to-year comparisons of annual medians.

Although FEV_1 values reached parity across the two sites by the end of the study, for FVC there remained an average difference of 25 mL. Both FEV_1 and FVC are established

measures reflecting risk of long-term disease and mortality, with FVC more related to restrictive lung disorders.[32]

Both groups of children lived in highly polluted areas before the study, so both are likely to have traffic-related lung function impairment. It is unknown whether continued exposure to less polluted air can deliver further improvements in lung function across both groups. Extending assessment of the CHILL cohort into adolescence (under way) should address whether this is the case and permit analysis of air pollution exposure trends beyond 2022, which was beyond the scope of the current study.

In summary, we show that children in London had improved lung function growth trajectories following ULEZ implementation, associated with significant reductions in exhaust emissions. 4 years after implementation of the ULEZ, FEV_1 reached parity with a comparator population in Luton, with the proportion of children with clinically impaired lung function falling. Children showed similar improvements in FVC between London and Luton, but the FVC levels remained lower in London at the end of the study, implying there were still gains to be made. The observed improvements could potentially prevent long-term adverse health impacts in adulthood, including premature mortality. These findings support the wider use of effective clean air zones as a public health policy intervention to improve the lung health of children in cities.

Contributors

CJG, ISM, AS, JG, GR, WJG, HEW, RED, JS, HSK, IT, BD, AK, CC, SB, VT, BM, RR, MM-W, EvS, FJK, CN, FB, JMo, EL, KP, MK, and JL conceived and/or designed the study. HEW, HSK, RED, JS, IT, JC, GC, LC, LS, JEM, MM-W, EvS, CJG, and ISM supervised data collection. HEW, HSK, RED, JS, IT, JC, GC, LC, LS, JEM, and CJG collected data. HEW, HH, BS, CN, RED, JS, JEM, SB, AB, NA, GS, DD, VT, BM, JMa, WJG, CJG, and ISM analysed data. HEW, HH, WJG, ISM, CJG, BS, AS, RED, JS, BD, SB, VT, MF, BM, and CN interpreted data. HEW, CJG, and ISM drafted the manuscript. All authors revised the manuscript for important intellectual content and approved the final version. HEW, HH, BS, and CN accessed and verified the data. All authors vouch for the completeness and accuracy of the data and the fidelity of the study to the protocol, had access to all the included data, and had final responsibility for the decision to submit for publication.

Declaration of interests

We declare no competing interests.

Data sharing

De-identified participant data can be requested from CJG in accordance with the study data sharing policy. Any data requests will be subject to review by the authors.

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