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# Effect of air pollution and racism on ethnic differences in respiratory health among adolescents living in an urban environment $\stackrel{\text{\tiny $\%$}}{=}$

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## ABSTRACT

Recent studies suggest that stress can amplify the harm of air pollution. We examined whether experience of racism and exposure to particulate matter with an aerodynamic diameter of less than  $2.5 \,\mu\text{m}$  and  $10 \,\mu\text{m}$  (PM<sub>2.5</sub> and PM<sub>10</sub>) had a synergistic influence on ethnic differences in asthma and lung function across adolescence. Analyses using multilevel models showed lower forced expiratory volume (FEV<sub>1</sub>), forced vital capacity (FVC) and lower rates of asthma among some ethnic minorities compared to Whites, but higher exposure to PM<sub>2.5</sub>, PM<sub>10</sub> and racism. Racism appeared to amplify the relationship between asthma and air pollution for all ethnic groups, but did not explain ethnic differences in respiratory health.

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### 1. Introduction

The global burden of asthma is rising (Asher et al., 2006), with 4.2 million adults and 1.1 million children in the United Kingdom (UK) alone costing over £2.3 billion a year to treat (Asthma, 2011). In the UK (Whitrow and Harding, 2008), ethnic minorities have lower lung function, which is an early predictor of cardiovascular mortality. Asthma is less commonly reported among ethnic minorities than Whites in the UK (Netuveli et al., 2005; Whitrow and Harding, 2010). While some of the determinants are known (Dezateux and Stocks, 1997), ethnic differences in respiratory health remain unexplained.

Environmental factors, such as air pollution (Pope, 2000; Samet et al., 2000), may play a key role in determining ethnic differences in respiratory health (Clougherty and Kubzansky, 2009; Gee and Payne-Sturges, 2004). Some ethnic minority adolescents in the UK are more likely than their White peers to reside in deprived neighbourhoods (Astell-Burt et al., 2012), which experience higher levels of air pollution as measured by a number of different indicators (Wheeler and Ben-Shlomo, 2005). There is increasing evidence that air pollution has a harmful impact, in particular, on lung function development during childhood. Studies have reported negative association

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between Forced Expiratory Volume in the first second (FEV<sub>1</sub>) and Forced Vital Capacity (FVC) with pollutants (Rojas-Martinez et al., 2007), especially particulate matter with an aerodynamic diameter of less than 2.5 µm (PM<sub>2.5</sub>) (Gauderman et al., 2004) and particulate matter with an aerodynamic diameter of less than  $10 \,\mu m \,(PM_{10})$ (Kulkarni et al., 2006). There is also increasing evidence for positive association between these pollutants and asthma (Anderson et al., 2011). However few studies have examined the role of air pollution on ethnic differences in respiratory health in childhood.

Psychosocial stress may also contribute to ethnic differences in respiratory health either as a direct cause or via enhancing neuroimmune and hypersensitivity response to air pollution (O'Neill et al., 2003). For example, stress related to low socioeconomic circumstances has been reported to increase the risk of asthma (Chen et al., 2006). Studies have also found a higher risk of asthma and lower lung function associated with traffic-related air pollution among children reporting stress in the home environment (Islam et al., 2011; Shankardass et al., 2009) and exposure to violence or domestic verbal abuse (Clougherty et al., 2007). Racism, more commonly experienced by ethnic minority than majority groups and a significant psychosocial stressor regardless of ethnicity (Astell-Burt et al., 2012; Krieger and Sidney, 1996), may have a similar modifying influence on pollution driven ethnic differences in respiratory health (Clougherty and Kubzansky, 2009; Gee and Payne-Sturges, 2004).

While some studies in the UK (Netuveli et al., 2005; Whitrow and Harding, 2008, 2010) have reported ethnic differences in asthma and lung function, and an extensive literature concerning adverse associations between respiratory health and air pollution has developed (Anderson et al., 2011; Dezateux and Stocks, 1997;





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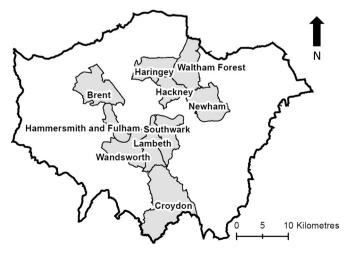
Gauderman et al., 2004; McConnell et al., 2006; Pope, 2000; Samet et al., 2000), very few studies have investigated the role of air pollution on ethnic differences in lung function and asthma, nor has this been extended to examine the potentially modifying role of psychosocial stress. Using a UK-based multiethnic cohort study of adolescents, we investigate the influence of air pollution on ethnic differences in lung function and asthma and the extent to which racism modifies these associations.

## 2. Methods

# 2.1. Data and sampling

The Determinants of Adolescent Social wellbeing and Health (DASH) study received approval from the Multicentre Research Ethics Committee and local education authorities (Harding et al., 2007). Pupils (N=6645) aged 11–13 years old were recruited at Wave 1 in 2003/04 from 52 schools that spanned above and below the national average for academic performance and contained at least 5% of pupils of Black Caribbean ethnicity according to the Department for Education (known then as the 'Department of Education and Skills'). These schools were located within 10 of London's 32 boroughs known to have at least 5% of ethnic minority groups. These boroughs were Brent, Croydon, Hackney, Hammersmith & Fulham, Haringey, Lambeth, Newham, Southwark, Waltham Forest and Wandsworth (see Fig. 1).

A self-complete questionnaire was administered in classrooms asking a range of questions on socioeconomic circumstances, health status, lifestyles and parenting. A substantive part of the survey was attributed to the identification of ethnic identity, which facilitated the cross-checking of participants' responses against parental ethnicity and the parental and grandparental countries of birth. The Wave 1 response rate was 83%, with generally better response rates for ethnic minority pupils than White UK pupils. In the Wave 2 survey during 2005 and 2006, 4775 adolescents aged 14-16 years participated (approximately 72% of the Wave 1 sample). The main source of attrition was due to non-participation by two schools because of construction work. The Wave 2 survey repeated the majority of selfreported questions from Wave 1, though physical measures did not include lung function. Survey assistants were trained for a week prior to the survey and recertified at regular intervals. DASH fieldwork protocols can be found at http://dash.sphsu.mrc.ac.uk/. Anthropometry was measured according to World Health Organisation recommendations (Report of a WHO Expert Committee, 1995) and lung function according to American Thoracic Society/European Respiratory Society



**Fig. 1.** Map of the London boroughs (shaded in grey and labelled) in which schools were sampled for the Determinants of Adolescent Social wellbeing and Health study.

(ATS/ERS) guidelines (Miller et al. 2005), including training by respiratory physiologists, daily checks and regular calibration of spirometers, and at least 3 satisfactory attempts by pupils.

The longitudinal sample used in this study included White UK (n=873), Black Caribbean (778), Nigerian and Ghanaian (504), Other African (386), Indian (419), Pakistani and Bangladeshi (446), and mixed White/Black Caribbean (262) boys and girls. Adolescents identifying with an ethnic group numerically too small for individual analysis were aggregated into an 'Other' category (n=1107). Pupils classified as 'Other' included those of several ethnic groups which makes interpretation difficult. We report the results for this group in the text rather than in the tables. Missing data for independent variables was resolved via single imputation of the ethnic- and gender-group specific mean.

#### 2.2. Dependent variables: asthma and lung function

Affirmative asthma status ('asthma' hereafter) was identified among adolescents self-reporting to the questions "have you ever had asthma?", and "in the last month, have you had breathing difficulties or wheeze?", in line with guidelines set out by the International Study of Asthma and Allergies in Childhood (ISAAC) (Asher et al., 2006). Each of these questions on asthma was asked at 11–13 yr and 14–16 yr.

Forced Expiratory Volume in one second (FEV<sub>1</sub>) and Forced Vital Capacity (FVC) were measured at 11-13 yr only. FEV1 and FVC are sensitive indices of lung growth, and strongly related to anthropometry (height, sitting height and chest shape) and age. FEV1 is the volume exhaled during the first second of a forced expiratory maneuver started from the level of total lung capacity. FEV1 is by far the most frequently used index for assessing airway obstruction, bronchoconstriction or bronchodilatation. FVC is the volume change of the lung between a full inspiration to total lung capacity and a maximal expiration to residual volume.

The best FEV<sub>1</sub> and FVC for each child were selected for analysis, according to ATS/ERS guidelines. Repeatability of the measures was assured by specifying that the difference between the largest and next largest FEV<sub>1</sub> and FVC be less than 0.15 l (L); or less than 0.1 L if the FVC is less than 1 L. Of 4775 children at Wave 1, 3768 had a satisfactory measure of FEV<sub>1</sub> and 3097 for FVC. We found no ethnic differences in satisfactory measures of FEV<sub>1</sub>. Compared to Whites, Black Caribbeans (odds ratio (OR): 1.27, (95% confidence interval (95%Cl) 1.04, 1.56), Nigerians and Ghanaians (OR: 1.56, 95% Cl 1.24, 1.95) and Other Africans (OR: 1.33, 95%Cl 1.04, 1.71) were significantly more likely to have unsatisfactory measures of FVC. For the statistical analyses involving lung function measures as the dependent variables, pupils with missing FEV<sub>1</sub> or FVC data were omitted. Adolescents with cystic fibrosis (n=8) were also omitted from the analysis.

### 2.3. Independent variables

Our main focus of investigation was the level of objectivelymeasured air pollution within urban neighbourhoods of residence. Pupils' residential addresses were obtained at the time of each survey and coded to Output Areas (OA) using look-up tables provided by

EDINA UKBORDERS, with the support of the Economic and Social Research Council (ESRC) and the Joint Information Systems Committee (JISC), and using boundary material which is copyright of the Crown and the Post Office. OA boundaries (approximately 297 residents on average) were used as proxies for neighbourhoods at 11–13y and 14–16y. Measures of annual mean air pollution per km<sup>2</sup>, including PM<sub>2.5</sub> and PM<sub>10</sub>, were provided by AEA Technology for 2003, 2004, 2005 and 2006 (http://www.aeat.co.uk/cms/). PM<sub>2.5</sub> and PM<sub>10</sub> were measured in micrograms per cubic metre ( $\mu$ g/m<sup>3</sup>). Measures were

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