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Traffic related air pollution and development and persistence of asthma and low lung function

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ABSTRACT

Background and aims: Traffic Related Air Pollution (TRAP) exposure is known to exacerbate existing respiratory diseases. We investigated longer term effects of TRAP exposure for individuals with or without existing asthma, and with or without lower lung function.

Methods: Associations between TRAP exposure and asthma (n = 689) and lung function (n = 599) were investigated in the prospective Tasmanian Longitudinal Health Study (TAHS). TRAP exposure at age 45 years was measured using two methods based on residential address: mean annual NO_2 exposure; and distance to nearest major road. Adjusted multinomial logistic regression was used to model the association between exposure to TRAP at 45 years and changes in asthma and lung function, using three follow ups of TAHS (45, 50 and 53 years).

Results: For those who never had asthma by 45, living < 200 m from a major road was associated with increased odds of new asthma that persisted from 50 to 53 years (adjusted Odds Ratio [aOR] 5.20; 95% CI 1.07, 25.4). Asthmatic participants at 45 had an increased risk of persistent asthma up to 53 years if they were living < 200 m from a major road, compared with asthmatic participants living > 200 m from a major road (aOR = 5.21; 95% CI 1.54, 17.6).

Conclusion: For middle aged adults, living < 200 m for a major road (a marker of TRAP exposure) influences both the development and persistence of asthma. These findings have public health implications for asthma prevention strategies in primary and secondary settings.

Abbreviations: TRAP, Traffic Related Air Pollution; TAHS, Tasmanian Longitudinal Health Study; RHINE, Respiratory Health in Northern Europe; ECRHS, European Community Respiratory Health Survey; ESCAPE, European Study of Cohorts for Air Pollution Effects; PM_{10}, particulate matter < 10 μm in diameter; $PM_{2.5}$, particulate matter < 2.5 μm in diameter; COPD, Chronic Obstructive Pulmonary Disease; LUR, Land Use Regression; ATS, American Thoracic Society; ERS, European Respiratory Society; FEV_1, Forced Expiratory Volume in 1 s; FVC, Forced Vital Capacity; DAG, Directed Acyclic Graph; UFP, Ultrafine Particles

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1. Introduction

There is substantial evidence that short term Traffic Related Air Pollution (TRAP) exposure exacerbates existing adult asthma (Guarnieri and Balmes, 2014; Braback and Forsberg, 2009), but evidence is lacking on whether TRAP affects subsequent asthma and lung function for those with or without asthma, as well as for those with or without impaired lung function. Studies of birth cohorts provide evidence for an association between early life TRAP exposure and incidence and persistence of asthma in children (Brandt et al., 2015; Bowatte et al., 2015), but it is not clear whether the same relationship exists in adults. This is a critical knowledge gap. Further investigations on the health effects of air pollution are required to develop better targeted public health preventive strategies and health promotion messages.

A small number of longitudinal studies have investigated the effects of TRAP exposure on the incidence of asthma, but not its persistence (Jacquemin et al., 2015; Jacquemin et al., 2009a; Modig et al., 2009; Young et al., 2014; Paulin and Hansel, 2016). Three longitudinal studies, the Respiratory Health in Northern Europe (RHINE), European Community Respiratory Health Survey (ECRHS) and European Study of Cohorts for Air Pollution Effects (ESCAPE) found that exposure to NO_2 was associated with adult asthma incidence over 9 years. However, a nationwide cohort of US women followed up for three years reported only a modest association with NO_2 exposure and incidence of asthma (Young et al., 2014). Few studies have been conducted on the association between asthma incidence in adults and particulate matter < 10 μm in diameter (PM_{10}), particulate matter < 2.5 μm in diameter ($PM_{2.5}$) or proximity to roads, and their findings have been inconsistent (Jacquemin et al., 2015; Modig et al., 2009; Young et al., 2014; Schindler et al., 2009; Modig et al., 2006). As all of these studies which investigated air pollution exposure and incidence of asthma began as adult cohorts, the potential for misclassification of incident asthma, i.e. the inability to recall early life asthma, may have contributed to the inconsistent findings.

Furthermore the evidence for longitudinal effects of TRAP exposure on adult lung function are not comparable for different TRAP proxies (NO_2, $PM_{2.5}$, PM_{10} or distance to major roads) and different measures of lung function (FEV_1, FVC or FEV_1/FVC) (Jacquemin et al., 2015; Lubinski et al., 2005; Ackermann-Liebrich et al., 1997). Recent studies provide modest evidence that in individuals with established Chronic Obstructive Pulmonary Disease (COPD) or asthma, longitudinal exposure to TRAP is associated with lung function decline (Carlsen et al., 2015; Lagorio et al., 2006; Qian et al., 2009; Kariisa et al., 2015). To our knowledge, no study has investigated effects of TRAP in subgroups with impaired lung function.

Given the above critical knowledge gaps, we used data from a longitudinal cohort study to investigate associations between adult TRAP exposure and subsequent change in asthma status and lung function over eight years.

2. Methods

2.1. Study population

The study sample comprised of participants of the 45, 50 and 53 year follow ups of the Tasmanian Longitudinal Health Study (TAHS) (Wharton et al., 2006; Gibson et al., 1969; Bowatte et al., 2017a). TAHS is a population based cohort that commenced in 1968, by recruiting (98.7%, n = 8583) of 7 year old children attending schools in the state of Tasmania, Australia. A number of follow-up surveys have been conducted since its inception. The majority of participants (67%) were resurveyed in 2002–05, when their mean age was 45 years. Respondents to this survey who had either participated in past follow up studies and/or reported symptoms of asthma or cough in the 2002–05 survey were invited to participate in a laboratory study in 2005–08 (n = 2387). Of those invited, 1397 (58.6%) participated in a full laboratory visit, including lung function testing and questionnaire. Only a telephone questionnaire or laboratory visit was completed by a further 354 (14.8%).

In 2010–12, when participants were aged around 50 years, those who took part in the 2005–08 laboratory study (n = 1397) were again invited for another laboratory study. Of those invited, 794 (56.8%) performed a full laboratory visit and completed a questionnaire (n = 837) (Bowatte et al., 2017a). In 2012–16, those who participated at the 2002–2005 survey were invited to complete a further survey and laboratory study. Of those, 3609 completed the survey and 2689 performed laboratory testing, including spirometry. The mean (± SD) ages of participants were 44.8 (± 1) (referred to as 45 year follow up), 49.6 (± 0.6) years (referred to as 50 year follow up) and 53.0 (± 1.4) (referred to as 53 year follow up) years, at the 2005–08, 2010–12 and 2012–16 follow ups, respectively. Addresses were geocoded for 705 of 723 (97.5%) participants who completed all three follow ups and who are included in this analysis (Fig. 1).

2.2. TRAP exposure assessment

2.2.1. Living < 200 m from a major road

Distance from each participant's residence to the nearest major road at the 45 year follow up was calculated using ArcGIS 10.1 software (Environmental Systems Research Institute, Redlands, CA). Major roads were defined using public sector mapping agencies (PSMA), Australia transport hierarchy codes 301 and 302. This mainly includes freeways, highways and arterial roads defined as “whose main function is to form the principal avenue of communication for movements: Between capital city and adjoining States and their capital cities; or Between a capital city and key towns; or Between key towns” (The Intergovernmental Committee of Surveying and Mapping, 2006). Participants were categorised into two groups: (i) living < 200 m; and (ii) living > 200 m from a major road. This cut-off was chosen based on the decay observed in levels of major traffic pollutants, given that the sharp decay in pollutant concentrations means that most TRAP components approach background concentrations at approximately 200 m (Karner et al., 2010).

2.3. NO_2 exposure

A satellite based land use regression (LUR) model was used to assign mean annual NO_2 exposures at the 45 year follow up (Knibbs et al., 2014). Briefly, this LUR model predicted mean annual NO_2 levels based on tropospheric NO_2 columns derived from satellite observations in combination with other predictors, such as land use and roads, to estimate ground level NO_2 across Australia. During the study period, 81% of spatial variation in annual NO_2 levels was captured by the model, with a cross-validated prediction error of 19% (Knibbs et al., 2014). An external validation of this LUR model was conducted; it captured 66% of annual NO_2 at a completely independent set of 98 urban background and near-traffic validation sites across Australia. Mean prediction bias in validation study was low (−0.2 ppb) and prediction error comparable to the initial cross-validation results (19% vs 25% in the original and validated results, respectively) (Knibbs et al., 2016). Mean annual residential exposures to outdoor NO_2 were estimated and assigned based on participants' geocoded addresses at 45 years.

2.4. Lung function

Pre-bronchodilator spirometry was performed using the EasyOne™ Ultrasonic Spirometer (Ndd, Medizintechnik, AG, Switzerland) according to American Thoracic Society (ATS) and European Respiratory Society (ERS) guidelines (Miller et al., 2005). Global Lung Initiative 2012 (GLI 2012) reference values were used to derive z-scores (Quanjer et al., 2012) for lung function variables. The z-scores were expressed as