

# Local NO<sub>2</sub> concentrations and asthma among over-50s in Ireland: a microdata analysis

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## Abstract

**Background** Links between air pollution and asthma are less well established for older adults than some younger groups. Nitrogen dioxide (NO<sub>2</sub>) concentrations are widely used as an indicator of transport-related air pollution, and some literature suggests NO<sub>2</sub> may directly affect asthma.

**Methods** This study used data on 8162 adults over 50 years old in the Republic of Ireland to model associations between estimated annual outdoor concentration of NO<sub>2</sub> and the probability of having asthma. Individual-level geo-coded survey data from The Irish Longitudinal Study on Ageing (TILDA) were linked to model-based estimates of annual average NO<sub>2</sub> at 50m resolution. Asthma was identified using two methods: self-reported diagnoses and respondents' use of medications related to obstructive airway diseases. Logistic regressions were used to model the relationships.

**Results** NO<sub>2</sub> concentrations were positively associated with the probability of asthma (marginal effect (ME) per 1 ppb of airborne NO<sub>2</sub> = 0.24 percentage points asthma self-report, 95% CI 0.06-0.42, mean asthma prevalence 0.09; for use of relevant medications ME = 0.21 percentage points, 95% CI 0.049-0.37, mean prevalence 0.069). Results were robust to varying model specification and time period. Respondents in the top 5<sup>th</sup> percentile of NO<sub>2</sub> exposure had a larger effect size but also greater standard error (ME = 2.4 percentage points asthma self-report, 95% CI -0.49- 5.3).

**Conclusions** Associations between local air pollution and asthma among older adults were found at relatively low concentrations. To illustrate this: the marginal effect of an increase in annual average NO<sub>2</sub> concentration from sample minimum to median (2.5 ppb ) represented about 7-8% of the sample average prevalence of asthma.

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**Keywords:** asthma, nitrogen dioxide, older adults, Ireland

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### **Key messages**

- Regressions on individual-level data for a large sample of older people in Ireland showed a strong association between asthma risk and local annual average NO<sub>2</sub> concentrations.
- Models using self-reported and medication-based identification of asthma gave consistent results.
- Associations are present at lower levels of pollutant exposure than current regulatory thresholds imply.
- Further research is needed into the effects of pollution on asthma in older populations.

## **INTRODUCTION**

Asthma is the most prevalent chronic respiratory condition worldwide, affecting over 300 million people.[1] While the exact causes of the condition are not fully understood,[2] a growing body of evidence suggests that environmental factors, in particular ambient air pollution, are involved. The possibility that sustained exposure to air pollution affects the onset and exacerbation of asthma cases is biologically plausible. Indeed, the UK Committee on the Medical Effects of Air Pollution has proposed a set of pathways through which such effects may operate in [3]. Potential mechanisms include oxidative stress and damage, airway remodelling, inflammatory pathways and immunological effects, and enhancing respiratory sensitisation to allergens.[4] While it is likely that individual pollutants bear some direct responsibility in the operation of these mechanistic pathways, it remains methodologically challenging to separate independent effects from those caused by other constituents of air pollution which may be contemporaneously emitted [3]. Nonetheless, nitrogen dioxide (NO<sub>2</sub>) concentration is often used as a marker for local transport-related air pollution in epidemiological studies.[5]

Much of the existing observational evidence linking various pollutants to cases of asthma focuses on paediatric cases. A recent survey by Bowatte et al. suggests that early childhood exposure to traffic-related air pollution is related to increased incidence of asthma and,

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3 indeed, that some of this effect is directly attributable to NO<sub>2</sub> [6]. Globally, effects of NO<sub>2</sub>  
4 exposure on asthma are sizeable, with Achakulwisut et al. estimating that 4 million new  
5 paediatric cases could be attributable to the pollutant annually [5]. While the biological  
6 framework above does not preclude an effect on asthma onset or exacerbation later in life,  
7 findings from the literature on adult populations remain inconsistent [7,8,9,10,11].  
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15 There are relatively few past studies focusing on the potential health effects of ambient  
16 pollution on the older population. Yet co-morbidities in older groups make management of  
17 asthma more difficult, and about two-thirds of asthma-related deaths occur among people  
18 over 65 years old [12]. Our study is probably most similar to research by Lindgren et al. of  
19 adults aged 18-77 in southern Sweden [13]. However, the present study focuses on over-50  
20 year olds and introduces a medication-based indicator of asthma in addition to self-reported  
21 diagnoses. We use rich survey microdata that allows us to relate estimated levels of ambient  
22 pollution at each respondent's residential address to asthma as well as a range of potentially  
23 confounding socioeconomic and health-related factors. The ability to observe such variables  
24 at individual respondent level is particularly valuable, because exposure to pollutants and  
25 any associated health impacts may be affected by socioeconomic characteristics and  
26 individual behaviours [14]. Studies using area-based averages find it difficult to examine  
27 such relationships.  
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45 The present study was carried out using data from the Republic of Ireland, where the  
46 national ambient air quality is relatively high. The EU Directive which governs the legal  
47 limits above which ambient air pollutants should not rise (2008/50/EC) limits annual mean  
48 concentrations of NO<sub>2</sub> at 40ug/mg (21 parts per billion). An exceedance in this dimension  
49 of the directive has only been observed once in Ireland (in 2009) between the years 2007-  
50 2017.[15] Although longitudinal data on health outcomes and many socioeconomic  
51 characteristics are available in the dataset, longitudinal pollution exposure data are not yet  
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3 available for the relevant years. In addition, changes in the structure of questions across  
4 survey waves made it difficult to reliably identify the timing of asthma incidence. These  
5 data limitations meant that this study estimated cross-sectional models of asthma prevalence  
6 rather than the incidence of asthma among older age groups and prevented us from  
7 examining causal mechanisms.  
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## 14 15 16 **METHODS**

17 Health information was drawn from The Irish Longitudinal Study on Ageing (TILDA) and  
18 linked to estimates of local NO<sub>2</sub> concentrations from Naughton *et al.*, 2018 [16].  
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### 23 **Sample**

24 TILDA is a nationally representative study of those aged over 50 in the Republic of Ireland.  
25 Data collection for Wave 1 (W1) of the study occurred between October 2009 and July  
26 2011, and follow-up data has been subsequently collected at two-year intervals: Wave 2  
27 (W2) in 2012 and Wave 3 (W3) in 2014-15 [17,18]. In W1, 8175 individuals over the age  
28 of 50 from a sample of 6279 households participated in the study. Including some spouses  
29 and partners of respondents, the total W1 sample size was 8504. Figure 1 shows how our  
30 final sample for each wave of the TILDA data was constructed.  
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50 The TILDA data used in this study were collected using Computer Assisted Personal  
51 Interviewing (CAPI) carried out by trained interviewers, face-to-face at each individual's  
52 home. Sensitive questions were included in a supplemental self-completed questionnaire  
53 (SCQ), which the respondents returned by mail, and a nurse-administered health assessment  
54 (SCQ), which the respondents returned by mail, and a nurse-administered health assessment  
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3 was collected in every second wave. However, SCQ and health assessment data were not  
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5 used in the current study.  
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8 The TILDA sample was recruited using the RANSAM protocol [19], which samples  
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10 households from the population of residential addresses in the Republic of Ireland. As a  
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12 result, the residential geo-location of each household is known.  
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## 15 16 **Measures**

### 17 18 **Outcome variables: self-report of asthma diagnosis and use of relevant** 19 **medications**

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21 We identified respondents with asthma in two ways. First, we used self-reported doctor  
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23 diagnoses of the condition (*asthma*). As part of the CAPI interview, respondents were shown  
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25 a card with a list of various chronic conditions, of which one is asthma, and are asked: “Has  
26  
27 your doctor ever told you that you have any of the following conditions?”. The binary  
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29 responses to this question were used as a dependent variable in the econometric models in  
30  
31 this study. However, since self-reported data can be subject to recall bias, we also used an  
32  
33 alternative identification method based on whether respondents regularly used medications  
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35 with a therapeutic purpose in the management of respiratory conditions (*medications*). The  
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37 set of medications used by each respondent was recorded by interviewers at the CAPI  
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39 interview. These medications were subsequently classified by their World Health  
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41 Organisation Anatomical Therapeutic Chemical Classification System codes. Class R03 in  
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43 this system comprises medications for use in the treatment of obstructive airway diseases  
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45 (OAD), including adrenergic agents such as salbutamol, inhaled corticosteroids,  
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47 anticholinergics, and leukotriene receptor antagonists. A binary indicator of whether or not  
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49 respondents were taking any such medications was used in our analysis as a second indicator  
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51 for asthma.  
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### Air pollution proxy variable: local NO<sub>2</sub> concentration

Estimates of NO<sub>2</sub> exposure were obtained from Naughton *et al.* 2018, which used a wind-sector land use regression (WS-LUR) to produce a high-resolution map from which estimated mean annual NO<sub>2</sub> concentrations at any given location across the Republic of Ireland can be extracted.[16] A spatial join in QGIS 2.18 was used to assign the estimated local pollutant concentration to each TILDA residence. The map used monitoring data from the Environmental Protection Agency's (EPA) national ambient air quality network, which records hourly NO<sub>2</sub> concentrations at various fixed locations throughout the state. The underlying model used air pollution data from 2010 to 2012, when the network consisted of 15 monitoring stations. There is a small offset between the timeline of the two datasets but insufficient local air pollution data were available for years prior to 2010. Figure 2 shows that national NO<sub>2</sub> trends were reasonably static from 2009 to 2013 [20]. These data were linked with hourly wind speed and wind direction data for each monitoring station as recorded by Met Éireann (the Irish meteorological service). The spatial variation in NO<sub>2</sub> was explained as a function of various land use and traffic-related variables. NO<sub>2</sub> model validation revealed a good explanation of variation at all sites. Following the approach taken by Beelen *et al.* [21], a leave-one-out cross validation method was employed in which the final model was fitted to N-1 sites and the predicted concentration compared to the actual concentration at the omitted site. This was repeated for all N sites and the overall level of fit between the predicted and measured concentration assessed. The final model explained over 78% of the spatial variability in NO<sub>2</sub>, while the cross validation R<sup>2</sup> was found to be slightly lower at 77.4%. The spread of data was well captured and the relationship between modelled and measured values close to linear [16]. The model was subsequently employed to predict NO<sub>2</sub> concentrations in other locations. More details of the methodology are included in Appendix A in the Supplementary File.

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11 Since there are very few observations at the upper end of the observed NO<sub>2</sub> distribution,  
12 some top-coding is necessary to protect the anonymity of individual TILDA respondents.  
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14 All respondents for whom the observed concentration is above the 95<sup>th</sup> percentile of the NO<sub>2</sub>  
15 distribution are assigned to a single “high exposure” category. To further protect participant  
16 anonymity, NO<sub>2</sub> concentration estimates for all other respondents were rounded to the  
17 nearest integer parts per billion (ppb). Setting up the data in this way means that we have  
18 two variables that together capture the full range of NO<sub>2</sub> exposures in the sample: an ordinal  
19 variable rounded to integer level that contains the exposures for each respondent whose  
20 exposure was below a ceiling of 13.1 ppb and a second categorical variable set to 1 for all  
21 respondents with values above 13.1 ppb and zero otherwise. Including both of these  
22 variables in regression models allows us to observe a linear relationship between exposures  
23 and outcomes for most respondents and an average marginal effect for those with exposures  
24 above the ceiling.  
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#### 42 Other explanatory variables

43 Asthma risk may be associated with socioeconomic characteristics. The environmental  
44 quality around each respondent’s residence could also be associated with his or her  
45 economic circumstances, with better-off households able to self-select into more attractive  
46 and potentially healthier neighbourhoods. While it is not possible to be certain all such  
47 factors were captured in our modelling, the TILDA dataset allowed us to control for many  
48 socioeconomic, demographic, and health-related variables which may jointly affect  
49 exposure to NO<sub>2</sub> and the probability of suffering from asthma. In particular, we controlled  
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for age, gender, income category, employment status, educational attainment, marital status, whether or not a respondent has medical insurance, whether or not they were (or had ever been) a smoker, and as a proxy for mobility limitations, whether or not they reported having difficulty walking 100m.

### Analysis

Both of our outcome variables of interest are binary:  $asthma_i$  takes on a value of 1 if a respondent reports ever having been diagnosed with asthma and is 0 otherwise. Similarly,  $medications_i$  takes on a value of 1 if a respondent is found to be taking medications for obstructive airway diseases. Logistic regressions were used to model the factors associated with asthma. Specifically, we estimate:

$$P(asthma_i = 1 | NO_2, X) = \Lambda(\alpha + \beta_0 NO_{2i} + \beta_1 HighNO_{2i} + \sum \beta_k X_{ki}) + \epsilon_i \quad ()$$

$$P(medications_i = 1 | NO_2, X) = \Lambda(\alpha + \beta_0 NO_{2i} + \beta_1 HighNO_{2i} + \sum \beta_k X_{ki}) + \epsilon_i \quad ()$$

where  $\Lambda(z) = \frac{e^z}{1+e^z}$ , the cumulative distribution of the logistic function,  $NO_{2i}$  is the estimated concentration of  $NO_2$  at the residential address of each TILDA respondent rounded to the nearest integer value (or zero for those in the high  $NO_2$  category),  $HighNO_{2i}$  is the dummy variable indicating those whose estimated  $NO_2$  exposure is greater than the 95<sup>th</sup> percentile,  $X_{ki}$  is a matrix of individual level explanatory variables and  $\epsilon_i$  is an error term such that  $\epsilon_i \sim IID(0, \sigma_\epsilon^2)$ .

We also conduct several robustness checks aimed at testing the stability of the observed association between  $NO_2$  and the prevalence of asthma under various model specifications. To see if the observed associations are sensitive to the timing of the TILDA survey, we repeat the analysis using data from Wave 2 and Wave 3 of the TILDA data. This is done both for prevalence of self-reported asthma (Model 1 above) and for the medication use outcome (Model 2). In addition, we test a combined outcome variable requiring that



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3 respondents both self-report asthma and use relevant medication before being classified as  
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5 having asthma. Finally, we check the effect of dropping the explanatory variable for  
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7 mobility limitation, i.e. whether or not respondents report having difficulty walking 100m.  
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9 This might be seen as an outcome of ill health, and thus arguably should be omitted as an  
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11 explanatory variable.  
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## 14 15 **RESULTS**

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17 Table 1 provides descriptive statistics for the two primary outcome variables of interest in  
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19 our analysis. The first shows the prevalence of self-reported asthma. 9% of individuals in  
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21 our sample indicated that a doctor had previously diagnosed them with the condition. The  
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23 share of respondents who were taking medications for the treatment of obstructive airway  
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25 diseases was smaller, at just 6.9%.  
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32 [Insert Table 1 about here]  
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38 It is helpful to compare the two asthma indicators. If both self-reported and medication-  
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40 based metrics were fully accurate and referred exclusively to the identical underlying  
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42 disease process, then respondents taking medications would correspond with those who  
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44 report having had an asthma diagnosis. Any differences between the two groups would arise  
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46 only from the subset of diagnosed cases that do not require regular medication (e.g. less  
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48 severe cases).  
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52 There were some differences in what the two metrics measured (see Table 2). Just over  
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54 51% of those who reported an asthma diagnosis were using OAD medications at the time of  
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56 interview (n=378). In contrast, 32.6% (n=183) of all those using medications did not report  
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58 a diagnosis of asthma. It is possible that some respondents made errors when reporting  
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3 whether they had an asthma diagnosis. In addition, some respondents could have reported  
4 accurately that they were not diagnosed with asthma but were taking OAD medications to  
5 manage other chronic respiratory conditions such as chronic obstructive pulmonary disease  
6 (COPD).  
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16 [Insert Table 2 about here]  
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21 The distribution of estimated exposure to NO<sub>2</sub> among TILDA respondents is illustrated in  
22 Figure 3. The figure shows a positive skew in the data. Much of the TILDA sample was  
23 exposed to relatively low levels of the pollutant, which is in line with expectations given the  
24 overall favourable status of air quality in Ireland.  
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33 [Insert Figure 3 about here]  
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40 Tables A1 and A2 in the Supplementary File describe the distribution of NO<sub>2</sub> concentrations  
41 among TILDA respondents as used in our analysis. Among those respondents outside the  
42 high exposure category, the mean concentration was 4.8 ppb with a maximum estimated  
43 concentration of 13 ppb. This is well below the EU limit for ambient mean annual  
44 concentrations of NO<sub>2</sub>, which is approximately equivalent to 21 ppb. The high exposure  
45 group had a mean concentration of 14.1ppb.  
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53 Table 3 presents descriptive statistics for other explanatory variables in our sample. 54% of  
54 our sample were female and 46% were male. This is consistent with the full TILDA cohort  
55 at W1.[22] For a sample of older people the observed sample was relatively young, with  
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3 57% in the 50-64 age bracket. This age profile was also reflected in the employment status  
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5 variable, which indicated that 36% of the sample were in employment. The sample covers  
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7 a broad spectrum of educational attainment. While 29% had attained a third-level  
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9 qualification, 31% had either no formal education or primary level only. The remaining 40%  
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11 reported having a secondary-level education. Smoking was quite prevalent in the sample,  
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13 with 56% of respondents reporting having smoked at some point in their lives. Since the  
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15 relationship between smoking habits and asthma may be different between past and current  
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17 smokers, we further subdivided this group: 18% reported being current smokers. Mobility-  
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19 limiting disability appeared relatively uncommon in the sample with just over 7% reporting  
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21 difficulty walking 100m due to some physical or mental health condition.  
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30 **[Insert Table 3 about here]**  
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35 Table 4 presents the logistic regression results for the NO<sub>2</sub> variables. Full regression results  
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37 are provided in Table A3 in the Supplementary File. The numbers reported are average  
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39 marginal effects of a given variable on the probability of suffering from asthma. Marginal  
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41 effects show the variation in the dependent variable associated with a 1 unit change in each  
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43 explanatory variable.<sup>1</sup> Model 1 suggests that, controlling for a wide range of socioeconomic  
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45 and health-related factors, a 1ppb increase in NO<sub>2</sub> concentration was associated with a 0.24  
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54 <sup>1</sup> This way of expressing the strength of association can be especially useful for expressing results from  
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56 pollution-related models, because regulatory authorities normally express limits on emissions or exposures in  
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58 absolute terms.  
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3 percentage point (95% CI 0.06-0.422) increase in the probability of reporting an asthma  
4 diagnosis for those with annual average exposures below 13.1 ppb. The increase in average  
5 prevalence of reported asthma for for those with exposures in the high NO<sub>2</sub> category has an  
6 effect size an order of magnitude higher but also a much larger standard error.  
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21 Model 2 explains the probability of taking OAD medications. With this outcome variable,  
22 the marginal effect of a 1ppb increase in local NO<sub>2</sub> concentrations was around 0.21  
23 percentage points (95% CI 0.0492-0.367). The marginal effect of being in the high NO<sub>2</sub>  
24 category is very imprecisely estimated in this model, which may be due to the limited  
25 number of observations in this segment of the NO<sub>2</sub> distribution.  
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33 We conducted several robustness checks aimed at testing the stability of the observed  
34 association between NO<sub>2</sub> and the prevalence of asthma under various model specifications  
35 (see Table 5). In W2 and W3, the associations between the same NO<sub>2</sub> concentrations and  
36 asthma were broadly similar to W1 for both the self-report and medication models.  
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51 A further robustness check narrowed down the assignment of asthma to cases where a  
52 reported diagnosis is corroborated by medication use. The observed association between  
53 this definition of asthma and NO<sub>2</sub> was broadly consistent with our previous results for W1  
54 and W2. In W3, there was a smaller effect size that is less precisely estimated. This might  
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3 reflect a lack of statistical power as attrition reduced the W3 sample size: relatively fewer  
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5 respondents had asthma under this narrow definition.  
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8 As one additional check, we dropped the limited mobility regressor to allow for the  
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10 possibility that one's perception of having difficulty walking 100m might be affected by  
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12 having asthma. If this were the case, including mobility difficulties as a control variable  
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14 might obscure the true association between NO<sub>2</sub> and the condition. The results were robust  
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16 to this exclusion.  
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## 20 21 **DISCUSSION**

22 This study found a positive association between local air pollution and the probability of  
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24 suffering from asthma for a large representative sample of older adults in Ireland.  
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26 Specifically, our results indicated that a 1ppb increase in local NO<sub>2</sub> was associated with a  
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28 0.15-0.25 percentage point increase in the probability of suffering from asthma, depending  
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30 on the metric used to identify asthma, the sample period and the model specification. The  
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32 magnitude of this association is large. We can illustrate this by observing that the overall  
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34 observed probability of self-reporting asthma in our data was about 9% and the share of  
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36 respondents taking relevant medications was 6.9%. Based on the models in Table 4, the  
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38 marginal effect of a 2.5ppb increase in annual average NO<sub>2</sub> concentration (equivalent to  
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40 someone moving from a place with sample minimum NO<sub>2</sub> to the median) represented about  
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42 7-8% of the sample average prevalence of asthma.  
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48 These results differ from the findings of Lindgren et al. [13], who reported associations  
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50 between asthma prevalence and living close to a major road (OR=1.40, 95% CI=1.04–1.89),  
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52 but found weaker evidence for associations with NO<sub>2</sub> exposure. They refer to effects only  
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54 in one of the cities within their sample and at exposure levels greater than 19 µg/m<sup>3</sup> of NO<sub>2</sub>.  
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3 Our study made several contributions. First, it found evidence of an association between  
4 NO<sub>2</sub> exposure and asthma among older adults, a group that is underrepresented in the  
5 literature. Second, it used both self-reports and use of medication to identify asthma cases,  
6 with consistent results. Third, the findings relate to a geographic area where levels of air  
7 pollution are relatively low, with exposures for many in the sample below standard  
8 regulatory thresholds. Finally, the individual-level data used in this study allowed us to  
9 control for a wealth of socioeconomic factors and health-related factors that may confound  
10 the relationship between NO<sub>2</sub> and asthma.  
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22 The study was subject to limitations. First, since our measure of local NO<sub>2</sub> concentrations  
23 was available only for a single point in time, we could not relate changes in exposure to  
24 changes in outcomes. In addition, variations in how the health status questions were asked  
25 in different survey waves made it hard to be certain about the timing of incidence for some  
26 individuals. The number of new cases reported in later waves was also small. Taken  
27 together, these problems limited us to analysing associations with the prevalence of asthma  
28 rather than its incidence among older people. If longitudinal data on a sufficiently large  
29 sample were available, it would allow researchers to control for unobserved individual-level  
30 heterogeneity and perhaps to test a possible channel of reverse causation such as respondents  
31 reacting to local air pollution when choosing where to live.[23]  
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46 Second, the indicator used for local air pollution, NO<sub>2</sub>, is only one of several pollutants  
47 implicated in respiratory disease. For policy purposes, it would be useful to examine the full  
48 range of air pollutants (e.g. adding particulates and ozone) simultaneously so that the  
49 relative contributions of different pollutants to disease burdens could be measured and any  
50 interactions among the pollutants might be identified. As a result of these limitations, it  
51 remains for future work to establish whether the association between NO<sub>2</sub> and the incidence  
52 of asthma in an older age group can be considered causal.  
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**Competing interests** None declared.

**Patient consent for publication** Not required.

**Ethics approval** Ethical approval was not required for this secondary data analysis. Ethical approval for each wave of TILDA data collection was obtained from the Trinity College Dublin Faculty of Health Sciences Research Ethics Committee.

**Data availability statement** Data may be obtained from a third party and are not publicly available. The linked data file can be accessed on site via the TILDA hot desk system (contact [tilda@tcd.ie](mailto:tilda@tcd.ie) for details). The unlinked data file can be accessed from the Irish Social Science Data Archive ([www.ucd.ie/issda/](http://www.ucd.ie/issda/)) and other sources, e.g. the Gateway to Globing Aging ([www.g2aging.org/](http://www.g2aging.org/)) and the Interuniversity Consortium for Political and Social Research ([www.icpsr.umich.edu/icpsrweb/](http://www.icpsr.umich.edu/icpsrweb/)).

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3 **Tables**  
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10 **Table 1: Descriptive statistics for dependent variables, TILDA Wave 1**

		Frequency	Percent
Self-reported Asthma	No Asthma	7424	91.0
	Asthma	738	9.0
OAD Medication Use	No OAD medications used	7601	93.1
	Some OAD medications used	561	6.9
Total		8162	100

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21 **Table 2: Relationship between dependent variables**

Medication use	Self-reported asthma		
	Yes	No	Total
Yes	378	183	561
No	360	7241	7601
Total	738	7424	8162

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32 **Table 3: Descriptive statistics for other explanatory variables**

	Frequency	Percent
<b>Gender</b>		
Male	3,736	45.77
Female	4,426	54.23
<b>Age category</b>		
50-64	4,662	57.12
65-74	2,160	26.46
≥ 75	1,340	16.42
<b>Income category</b>		
0 - 9,999	647	7.93
10,000 - 19,999	1,658	20.31
20,000 - 39,999	2,702	33.1
40,000 - 69,999	1,560	19.11
≥ 70,000	701	8.59
Not reported	894	10.95
<b>Marital status</b>		
Married	5,629	68.97
Never married	791	9.69
Sep/divorced	551	6.75
Widowed	1,191	14.59
<b>Employment status</b>		
Employed	2,930	35.9

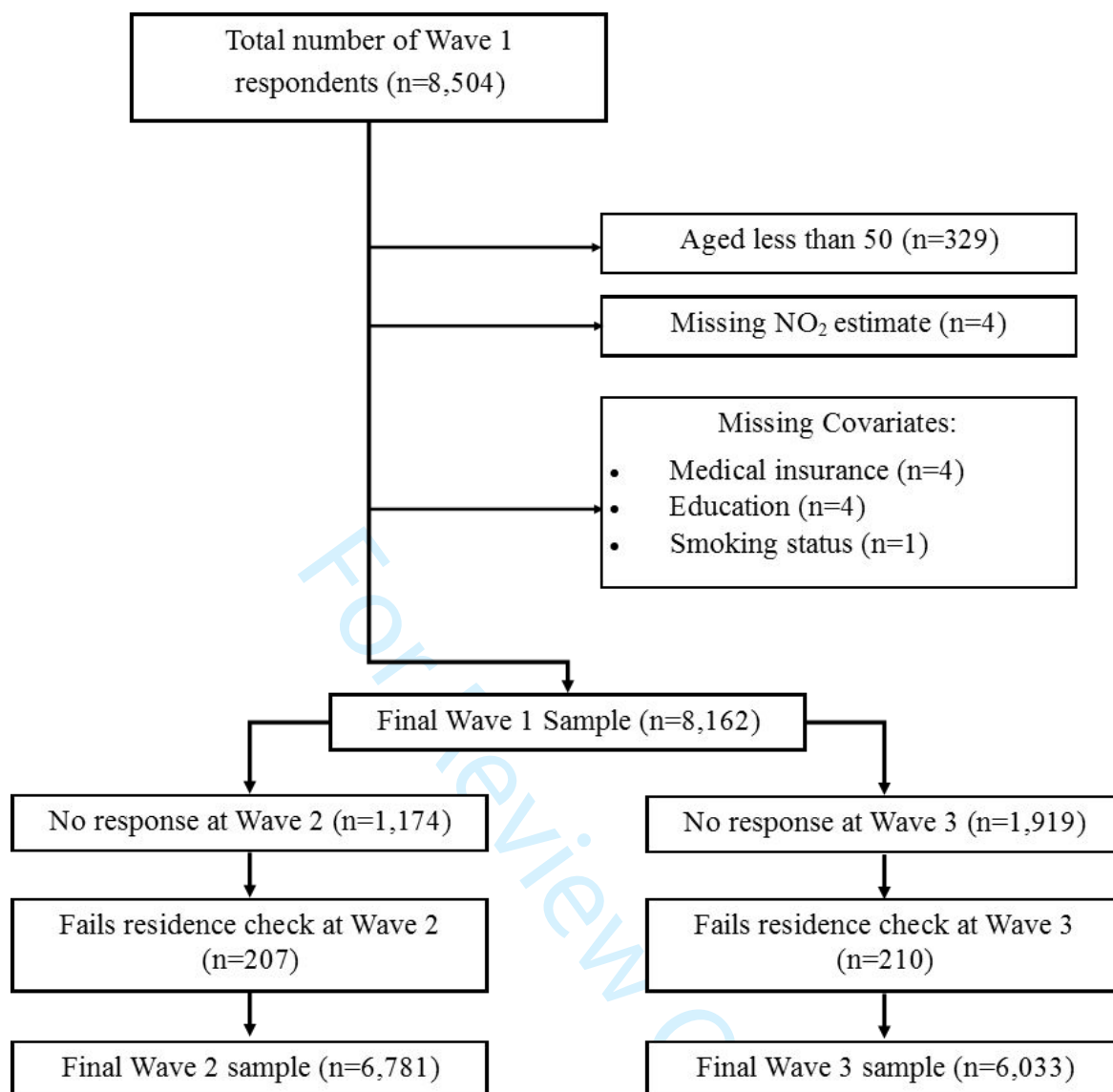
	Frequency	Percent
Retired	3,039	37.23
Other	2,193	26.87
<b>Smoking status</b>		
Never	3,561	43.63
Past	3,112	38.13
Current	1,489	18.24
<b>Educational attainment</b>		
Primary/none	2,502	30.65
Secondary	3,258	39.92
Third/higher	2,402	29.43
<b>Medical cover</b>		
Not covered	844	10.34
Medical insurance	3,282	40.21
Medical card	4,036	49.45
<b>Mobility</b>		
No difficulty walking 100m	7,562	92.65
Difficulty walking 100m	600	7.35
<b>Total</b>	<b>8,162</b>	<b>100</b>

**Table 4: Logistic regressions estimating the relationship between NO<sub>2</sub> and asthma**

	Model 1: Self-reported Asthma			Model 2: OAD Medication Use		
	$\delta y/\delta x$	95% CI	P-value	$\delta y/\delta x$	95% CI	P-value
<b>NO<sub>2</sub> Exposure</b>						
NO <sub>2</sub> level (PPB)	0.00241	[0.0006, 0.00422]	0.009	0.00208	[0.000492, 0.00367]	0.01
High NO <sub>2</sub> (NO <sub>2</sub> > P <sub>95</sub> )	0.0241	[-0.00493, 0.0531]	0.104	0.000926	[-0.0264, 0.0282]	0.947
N	8162			8162		
AIC	4898.5			3977.7		
BIC	5059.7			4138.9		

**Table 5: Robustness checks**

	NO <sub>2</sub> level			High NO <sub>2</sub> indicator			N
	$\delta y/\delta x$	95% CI	P-value	$\delta y/\delta x$	95% CI	P-value	
<b>Prevalence of self-reported asthma at other waves</b>							
Wave 1	0.00241	[0.0006, 0.00422]	0.009	0.0241	[-0.00493, 0.0531]	0.104	8162
Wave 2	0.00191	[-0.000654, 0.00389]	0.058	0.0125	[-0.0203, 0.0453]	0.454	6781
Wave 3	0.00246	[0.000464, 0.00446]	0.016	0.0204	[-0.0139, 0.0548]	0.244	6033
<b>Medication use at other waves</b>							
Wave 1	0.00208	[0.000492, 0.00367]	0.01	0.000926	[-0.0264, 0.0282]	0.947	8162
Wave 2	0.0019	[0.000048, 0.00376]	0.044	0.0164	[-0.0135, 0.0463]	0.282	6781
Wave 3	0.00233	[0.000397, 0.00426]	0.018	0.0104	[-0.0227, 0.0435]	0.538	6033
<b>Dependent variable based on both self-reported asthma &amp; medication use</b>							
Wave 1	0.00204	[0.000724, 0.00335]	0.002	0.0104	[-0.0114, 0.0321]	0.35	8162
Wave 2	0.0017	[0.00016, 0.00323]	0.03	0.024	[0.000481, 0.0476]	0.045	6781
Wave 3	0.00149	[-0.000969, 0.00309]	0.066	0.00324	[-0.0254, 0.0319]	0.825	6033
<b>Drop limited mobilityvariable</b>							
Wave 1	0.00248	[0.000667, 0.00429]	0.007	0.0251	[-0.00386, 0.054]	0.089	8162
Wave 2	0.00202	[0.0000502, 0.004]	0.044	0.0134	[-0.0194, 0.0462]	0.424	6781
Wave 3	0.00254	[0.000545, 0.00454]	0.013	0.0208	[-0.0136, 0.0553]	0.237	6033



**Figure 1: Construction of the final sample**

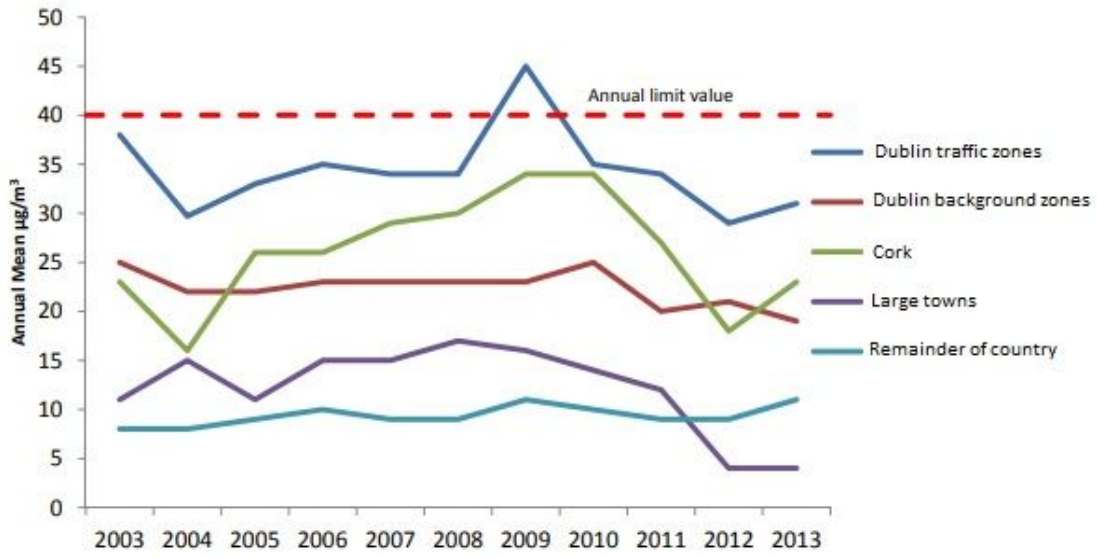
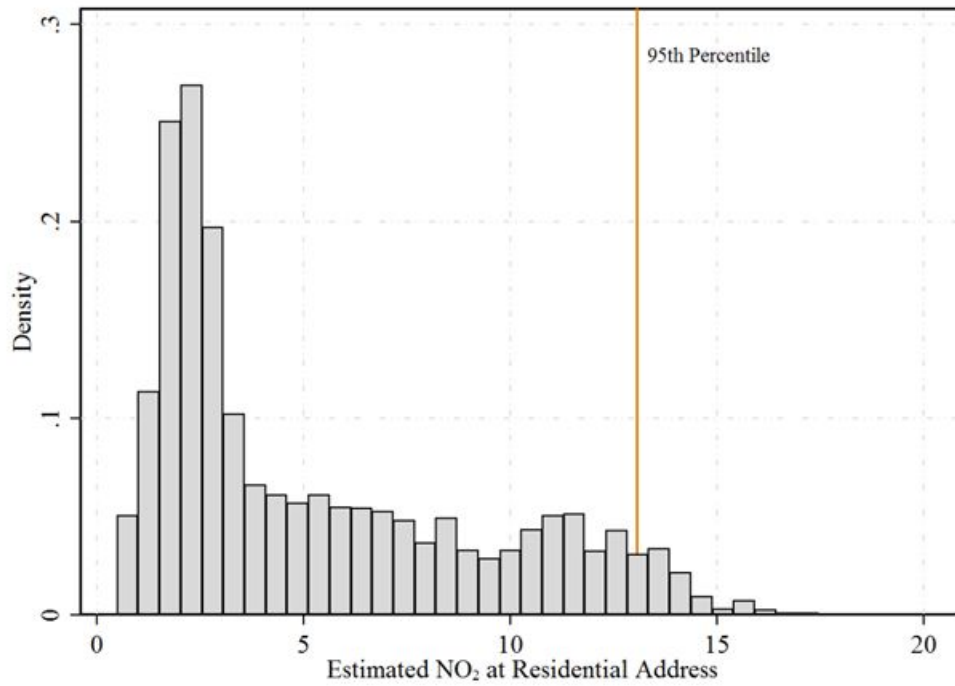


Figure 2. Trend in NO2 concentrations for zones in Ireland from 2003-2013 [17]

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**Figure 3: Frequency Distribution of NO<sub>2</sub> among TILDA Respondents**

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