

ORIGINAL ARTICLE

Ambient wood smoke, traffic pollution and adult asthma prevalence and severity

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ABSTRACT

Background and objective: The impact of ambient wood smoke and traffic-related air pollution on adult asthma has not been well studied. This paper aims to investigate associations between exposure to ambient wood smoke, traffic-related air pollution and current asthma/asthma severity in middle age, and whether any associations are modified by atopic status.

Methods: Using data from the Tasmanian Longitudinal Health Study, associations between ambient wood smoke and two indices of traffic-related air pollution (frequency of heavy vehicles near the home and frequency of intense traffic noise) and current asthma/asthma severity were investigated. Unconditional logistic regression to examine current asthma and ordinal logistic regression to examine asthma severity was used.

Results: For asthmatics, both exposure to ambient wood smoke (odds ratio 1.11; 95% confidence interval 1.02–1.20) and being frequently exposed to heavy vehicles (odds ratio 1.80; 95% confidence interval 1.09–2.96) were associated with increased asthma severity. Neither association varied by atopic status.

Conclusions: In middle-aged adults, ambient wood smoke and traffic pollution were associated with increased asthma severity. These findings suggest that avoiding or limiting exposure to traffic pollution and wood smoke may help to reduce asthma. Future studies to replicate this finding are recommended and should examine specific biological mechanisms for this effect.

Key words: adult asthma, ambient wood smoke, atopic status, severity, traffic pollution.

SUMMARY AT A GLANCE

We investigated associations between ambient wood smoke and traffic-related air pollution exposure and current asthma severity in middle age, and whether any associations are modified by atopic status. There were associations between wood smoke and traffic pollution, and asthma severity. This study adds evidence of adverse impact of the exposures on adult asthma.

Abbreviation: TAHS, Tasmanian Longitudinal Health Study.

INTRODUCTION

Asthma affects more than 300 million people worldwide and is one of the most common chronic health conditions, imposing a significant burden on patients, their families and society.¹ Asthma accounts for 0.9% of the total global disease burden, corresponding to an annual loss of 22.5 million disability-adjusted life years.² During recent decades, asthma prevalence has increased in most countries.³ More recently, there has been a slight decline in asthma prevalence in children in the developed world, but it continues to rise in the developing world.⁴ Although its prevalence is lower in adults than in children, asthma in adults remains a public health concern.

Increasing attention has been given to ambient air pollution, particularly traffic-related pollution and wood smoke, as risk factors for asthma. Effects of traffic-related pollution on asthma have been well studied in children with consistent findings that such exposure contributes to both asthma onset and asthma exacerbations.^{5,6} In contrast, there are fewer studies in adults, and the results are less consistent. While some studies have reported associations

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between markers of traffic pollution and asthma prevalence, severity or incidence,^{7–13} others have contradicted these findings.^{14–17} There have also been a limited number of ecological studies that investigated effects of ambient wood smoke exposure on adult asthma. Some report no association between ambient wood smoke and adult respiratory disease,^{18,19} while others report a detrimental impact of ambient particulate matter on asthma hospital admissions.^{20,21}

These conflicting reports may be related to inaccurate asthma phenotyping. There is increasing recognition that adult asthma comprises multiple phenotypes,^{22,23} with some appearing more related to environmental, occupational or other health factors rather than 'classic' allergic inflammation. We postulated that the contribution of ambient air pollution to adult asthma might be clarified by phenotyping adult asthma with respect to atopic status.

Using data from the Tasmanian Longitudinal Health Study (TAHS), we aimed to investigate for middle-aged adults associations between exposure to ambient wood smoke or traffic-related air pollution, and (i) current asthma and (ii) asthma severity, and possible differential effects by atopic status.

METHODS

Study population and data collection

The data for this analysis were obtained from the TAHS, which has been described in detail elsewhere.^{24,25} In brief, a population-based cohort study, was initiated in 1968 to examine the prevalence and natural history of asthma in childhood. A total of 8583 eligible school students were recruited, representing 98.8% of all 7-year-old school students in Tasmania. There were follow-up surveys in 1974, 1981, 1992 and 2003–2005. In the 2003–2005 survey, 7312 (85.2%) of the participants from the original 1968 cohort were successfully traced to an address and sent a respiratory questionnaire. Responses were received from 5729 (78.4%) participants. Subsequently, 2373 respondents were invited to attend a more detailed laboratory study. These 2373 were selected based on their previous participation in either the 1974 clinical examination or the 1992 postal survey. Respondents who had asthma from 1968 to 2004 were also included. Of those invited, 1383 (58.3%) completed an additional detailed questionnaire. This laboratory questionnaire also gathered information about outdoor air pollution exposure. Data from those 1383 participants form the basis of this analysis.

Ethical approval

The 2003–2005 follow-up survey of the TAHS was approved by the Human Research Ethics Committee of the University of Melbourne (Approval number 010626). The survey was conducted according to the principles expressed in the World Medical Association Declaration of Helsinki. All participants provided written informed consent for participation in the study.

Definitions of exposures, outcomes and covariates

The outcomes of interest were current asthma and asthma severity. The exposures of interests were traffic pollution (defined as *frequency of heavy vehicles near home* and *frequency of intense traffic noise*) and levels of ambient wood smoke in winter. These exposures and outcomes were defined using questionnaire responses.

Frequency of heavy vehicles near home and *frequency of intense traffic noise* included four categories: never, seldom, frequent and constant.

Levels of ambient wood smoke in winter were defined by the question 'On a scale of 0 to 10, how much is the area where you live subjected to wood smoke in winter?'

Current asthma was defined by an affirmative answer to the question 'Have you ever had asthma' and reporting asthma symptoms in last 12 months.

Asthma severity rating used the National Asthma Council Australia classification for untreated, newly diagnosed asthma.²⁶ Based on self-reported symptoms and the number of flare-ups (exacerbations), asthma severity was graded into four levels:

- Intermittent asthma: symptoms manifest in the last 12 months but not the last month, and three or less flare-ups in the last 12 months
- Mild persistent asthma: symptoms manifest in the last month but less than weekly, or four or more flare-ups in the last 12 months but less than monthly
- Moderate persistent asthma: symptoms manifest more than weekly but not daily, or flare-ups more than monthly in the last 12 months
- Severe persistent asthma: symptoms manifest daily or flare-ups more than weekly in the last 12 months

Atopic status was defined by a positive skin prick test to at least one of eight aeroallergens: house dust mite, cat, *Hormodendrum*, *Alternaria tenuis*, *Penicillium* mix, *Aspergillus fumigatus*, Perennial Rye grass and mixed grass pollen. Positive skin prick test was defined by a wheal of diameter 3 mm or more to each allergen extract with a positive skin prick test to histamine and a negative control.

Statistical methods

The characteristics of participants were compared using Pearson's chi-squared tests. Multivariable logistic regression²⁷ examined associations between exposures and current asthma. Ordinal logistic regression models were used to examine associations between exposures and asthma severity. Proportional odds assumption was tested and was not violated.

Analyses were performed separately for different exposures. Wood smoke was treated as a continuous variable. For the two traffic variables, we collapsed some of the exposure categories due to small cell sizes. The baseline model was constructed with the inclusion of a set of *a priori* confounders: sex, socio-economic status and smoking status. Other potential confounders were examined by being added to the baseline model one by one. These potential confounders included family history of asthma, history of allergic diseases (hay fever and eczema), atopic

status, presence of mould in the home, type of energy used for cooking and heating, and passive smoking. The confounding variable was retained in the model if its inclusion altered the point estimate by 10% or more. Interaction terms were tested and included in the regression models if the interaction term was statistically significant. Sensitivity analysis was performed for associations between exposures and current asthma by excluding from the control group participants with previously recorded but now remitted asthma. All analyses were performed using STATA version 11.0 (Stata Corp, College Station, TX, USA).

RESULTS

Characteristics of participants

Personal and environmental characteristics of participants by current asthma status are summarized in Table 1. Approximately 18.9% ($n = 262$) of the study population had current asthma. Participants with and without current asthma did not differ significantly by smoking status. Participants with current asthma had higher socio-economic status and included a higher proportion of females, atopic status and history of allergic diseases (hay fever and eczema). Residential duration was not significantly different between those with and without current asthma (9.2 vs 9.7 years, $P = 0.34$). In addition, current and non-current asthmatics did not differ significantly by education level, heating and cooking appliances, family history of asthma, or presence of domestic moulds (data not shown).

Table 2 shows characteristics of the 262 current asthmatics by asthma severity. Across the levels of asthma severity, subjects did not differ significantly in baseline characteristics, except for the frequency of heavy vehicle exposure ($P = 0.02$) and frequency of intense traffic noise exposure ($P = 0.05$).

Association between traffic pollution, ambient wood smoke and asthma

Associations between exposures and asthma are shown in Table 3. There was no association between either of the two markers of traffic pollution or ambient wood smoke and the prevalence of current asthma. The results of the sensitivity analysis performed by omitting participants with remitted asthma from the control group did not materially differ to those from the main analysis.

There was a statistically significant but modest association observed between wood smoke exposure and asthma severity (odds ratio 1.11; 95% confidence interval 1.02–1.20). This association was observed both for users and non-users of wood combustion (data not shown). Of the indices of traffic pollution, the frequency of heavy vehicular traffic was also significantly associated with asthma severity (odds ratio 1.80; 95% confidence interval 1.09–2.96), but the association with the frequency of intense traffic noise was not statistically significant (odds ratio 2.27; 95% confidence interval 0.91–5.62).

We stratified associations between exposure to wood smoke and to frequency of heavy vehicles and

Table 1 Characteristics of participants stratified by current asthma status

	Non-current asthma ($n = 1121$)		Current asthma ($n = 262$)		<i>P</i> -value
	<i>n</i>	%	<i>n</i>	%	
Age (mean, SD)	42.6 (0.76)		42.7 (0.82)		0.19
Sex: Female	523	46.7	156	59.5	<0.001
Smoking status					0.76
Non-smoker	465	41.6	115	44.1	
Past smoker	335	29.9	74	28.4	
Current smoker	317	28.4	72	27.6	
Socio-economic status (quintiles)					0.06
1st	319	28.6	75	28.9	
2nd	144	12.9	22	8.5	
3rd	234	21.0	44	17.0	
4th	195	17.5	52	20.1	
5th	222	19.9	66	25.5	
Atopic status	567	50.6	198	75.6	<0.001
Passive smoker	160	14.5	41	15.9	0.58
History of ever hay fever	622	55.8	226	86.3	<0.001
History of ever eczema	448	40.4	157	60.6	<0.001
Frequency of heavy vehicles					0.44
Never or seldom	746	67.4	183	69.8	
Frequent or constant	362	32.6	79	30.2	
Frequency of intense traffic noise					0.96
Never or seldom	1038	93.4	245	93.5	
Frequent or constant	73	6.6	17	6.5	
Levels of ambient wood smoke (mean, SD)	3.42 (2.65)		3.62 (2.88)		0.85

SD, standard deviation.

Table 2 Characteristics of participants with current asthma stratified by asthma severity

	Asthma severity				<i>P</i> -value
	Intermittent asthma, <i>n</i> (%)	Mild persistent asthma, <i>n</i> (%)	Moderate persistent asthma, <i>n</i> (%)	Severe persistent asthma, <i>n</i> (%)	
Sex: Female	47 (56.6)	42 (59.1)	43 (64.2)	24 (58.5)	0.82
Smoking status					0.18
Non-smoker	39 (46.9)	34 (47.9)	27 (40.9)	15 (36.6)	
Past smoker	28 (33.7)	20 (28.2)	17 (25.7)	9 (21.9)	
Current smoker	16 (19.3)	17 (23.9)	22 (33.3)	17 (41.5)	
Socio-economic status (quintiles)					0.33
1st	25 (30.5)	19 (26.7)	16 (24.2)	15 (37.5)	
2nd	8 (9.8)	6 (8.5)	5 (7.6)	3 (7.5)	
3rd	14 (17.1)	18 (25.3)	7 (10.6)	5 (12.5)	
4th	15 (18.3)	16 (22.5)	13 (19.6)	8 (20.0)	
5th	20 (24.4)	12 (16.9)	25 (37.8)	9 (22.5)	
Atopic status	61 (73.6)	51 (71.8)	56 (83.6)	30 (73.2)	0.36
Passive smoker	8 (9.7)	11 (15.7)	12 (18.2)	10 (25.0)	0.16
Residence time					0.16
>5 years	53 (63.8)	49 (69.0)	34 (50.7)	25 (60.9)	
<5 years	30 (36.1)	22 (30.9)	33 (49.2)	16 (39.0)	
History of ever hay fever	74 (89.2)	61 (85.9)	57 (85.1)	34 (82.9)	0.78
History of ever eczema	51 (62.9)	38 (53.5)	46 (69.7)	22 (53.6)	0.18
Medication use	65 (78.3)	57 (80.3)	59 (88.0)	39 (95.1)	0.06
Late asthma onset (after 21 years old)	37 (44.6)	37 (52.1)	32 (47.7)	18 (43.9)	0.77
Frequency of heavy vehicles					0.02
Never or seldom	67 (80.7)	51 (71.8)	40 (59.7)	25 (60.9)	
Frequent or constant	16 (19.3)	20 (28.2)	27 (40.3)	16 (39.0)	
Frequency of intense traffic noise					0.05
Never or seldom	80 (96.4)	69 (97.2)	58 (86.6)	38 (92.7)	
Frequent or constant	3 (3.6)	2 (2.8)	9 (13.4)	3 (7.3)	
Levels of ambient wood smoke (mean, SD)	3.09 (2.67)	3.60 (2.85)	3.55 (2.90)	4.85 (3.04)	0.07

SD, standard deviation.

Table 3 Association between ambient wood smoke, traffic pollution and asthma

	Current asthma		Asthma severity	
	Adjusted OR (95% CI)	<i>P</i> -value	Adjusted OR [†] (95% CI)	<i>P</i> -value
Frequency of heavy vehicles [‡]				
Never or seldom	1	0.20	1	0.02
Frequent or constant	0.82 (0.60–1.11)		1.80 (1.09–2.96)	
Frequency of intense traffic noise [§]				
Never or seldom	1	0.28	1	0.08
Frequent or constant	0.72 (0.39–1.31)		2.27 (0.91–5.62)	
Levels of ambient wood smoke	1.01 (0.96–1.06)	0.55	1.11 (1.02–1.20)	0.01

[†] Odds for severe persistent asthma compared with all lower categories of persistent asthma combined for a one unit increase in exposure.[‡] Adjusted for sex, smoking status and socio-economic status for both current asthma and asthma severity.[§] Adjusted for sex, smoking status and socio-economic status for asthma severity, and additionally adjusted for atopy, history of eczema and hay fever for current asthma.

CI, confidence interval; OR, odds ratio.

asthma severity by atopic status, but neither of the associations varied across strata. Similarly, there were no differential associations by gender, age at asthma onset or family history of asthma.

DISCUSSION

We report that exposure to ambient wood smoke and to traffic pollution are both associated with more

severe asthma. To our knowledge, ours is the first study to investigate the impact of ambient wood smoke on adult asthma using individual exposure assessment.

The mechanism underlying any association between wood smoke exposure and asthma is not fully understood but might be due to airway inflammation secondary to an 'irritant effect' from wood smoke inducing activation of the innate immune system.²⁸ Studies of firefighters have shown an effect of wood smoke on bronchial hyperresponsiveness and neutrophil/macrophage activation.²⁹ An intervention trial in Guatemala reported that exposure to high levels of biomass smoke was associated with an increase in gene and/or protein expression in sputum for molecules involved in innate airway inflammation and remodelling.³⁰

Earlier studies of asthmatic adults have reported associations between traffic-related air pollution and decreased lung function, and poorly controlled asthma.^{11–13} Similarly, our study supports the detrimental effect of such pollution on adults with asthma. Traffic exhaust, a complex mixture of particulate matter and gases (e.g. nitric oxide, nitrogen dioxide, carbon monoxide and benzene), is thought to exacerbate asthma through airway inflammation.³¹ Particles from heavy vehicles' exhaust have been shown to enhance allergic inflammatory responses in sensitized persons.³² Traffic pollutants are also likely to cause oxidant injury, which in turn might lead to airway remodelling.³³ This is supported by a recent randomized study that showed reductions in lung function and increases in biomarker of neutrophilic inflammation after short-term traffic exposure in asthmatics.¹² Interestingly, a synergy between diesel exhaust particles and natural allergen exposure has been reported and is considered to be a key feature in increasing allergen-induced respiratory allergic disease.³⁴

We report an association between frequency of heavy vehicles as an index of traffic air pollution and a modest association between frequency of intense traffic noise and asthma severity. To some extent, this finding might be related to subjective exposure assessments, but the lack of an association between markers of traffic exposure and current asthma suggests otherwise. Only 6.5% of participants reported, frequent or constant exposure to intense traffic noise and small numbers might have contributed to the non-significant odds ratio over 2 estimated for this exposure.

There was no association between any of the exposure variables and current asthma prevalence. As traffic air pollution is well known to trigger asthma,^{35,36} persons with severe asthma may have migrated away from busy roads, so contributing to the absence of association between traffic pollution and asthma prevalence. On the other hand, residential duration at the current house did not differ significantly between those with or without current asthma, so it is unlikely that migration bias is important.

In contrast with our null findings for associations between traffic exposures and current asthma, a cross-sectional study in Germany that used the same

measures of exposure as the present study reported a significant association between traffic pollution and current asthma in adolescents.³⁷ Browning *et al.*¹⁸ also reported an association between ambient wood smoke and current asthma for children but not adults. These contrasting findings suggest that the impact of air pollution on the prevalence of asthma might be age-dependent.

Users of indoor wood combustion have been shown to suffer adverse health impacts such as chronic obstructive pulmonary disease.³⁸ The association we observed between ambient wood smoke exposure and asthma severity provides evidence that the use of wood or coal for heating has a detrimental impact in a community where domestic use of wood burning is common. Exposure to emissions from wood burners would vary with the operating conditions. For example, combustion of damp wood generates significantly higher emissions than dry wood. Clean burning practices and the replacement of old polluting wood stoves by new ones are likely to minimize both indoor and outdoor wood smoke pollution.

There are strengths and limitations to our study. Being nested in the TAHS provides a major strength because it uses a well-defined population-based study sample that has been followed from childhood allowing detailed phenotyping of asthma within the cohort. There is also complete information for a wide range of potential confounders and effect modifiers. An additional strength is that the definition of asthma used in our study has been validated against a respiratory physician's diagnosis of asthma.³⁹

As adults spend a considerable amount of time outside their homes, the lack of information about traffic pollution at the workplace and commuting time in traffic is a limitation. Also, the lack of objective measurements of exposure is also a limitation possibly resulting in exposure misclassification. Any non-differential misclassification of the exposure status might have contributed to the null results. Persons with and without asthma may have different perceptions of the impact of air pollution so that those with asthma could conceivably overreport their exposure levels. Such differential reporting could produce spurious associations, but the absence of any association in our study does not support the likelihood of exposure misclassification.

Finally, we suggest that avoiding or limiting exposure to traffic pollution and wood smoke could be important to reduce disease burden for asthmatics. We suggest that people with asthma should avoid living close to major roads, although Tasmania is largely a rural state with lower traffic density compared with other Australian states. In Tasmania, where around 30% of households used wood-burning devices for heating in winter in 2004, the replacement of wood burners and open fireplaces with cleaner forms of heating has been promoted and has shown some success in reducing ambient wood smoke pollution.⁴⁰ It is likely that people with asthma may have already benefited from these interventions, but our results suggest there could be room for further improvement.

In conclusion, we report associations between ambient wood smoke, traffic pollution and asthma severity for middle-aged adults. Neither association varied by atopic status. Neither wood smoke nor traffic-related air pollution was associated with asthma prevalence. Due to the small number of studies that have examined the association between ambient air pollution, particularly from wood smoke, and adult asthma, these associations merit further research first to corroborate them and second to investigate underlying biological mechanisms.

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