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The impact of the Hazelwood coal mine fire smoke exposure on asthma

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ABSTRACT

Objective: In 2014, a fire at an open cut coal mine in South-eastern Australia burned for about 6 weeks. Residents of the adjacent town were exposed to high levels of fine particulate matter (PM_{2.5}) during this period. Three and a half years after the event, this study aimed to investigate potential long-term impacts of short-term exposure to coal mine fire smoke on asthma.

Methods: A cross-sectional analysis was undertaken in a group of exposed participants with asthma from Morwell ($n = 165$) and a group of unexposed participants with asthma from the control town of Sale ($n = 64$). Exposure was determined by modeled PM_{2.5} data for the mine fire period. Respiratory symptoms were assessed with a validated respiratory health questionnaire and symptom severity score. Asthma control was assessed with a validated questionnaire. Lung function testing included spirometry, bronchodilator response, and fraction of exhaled nitric oxide.

Results: There was no evidence that exposed participants had more severe asthma symptoms, worse lung function, or more eosinophilic airway inflammation than unexposed participants. However, there was some evidence that Morwell participants had more uncontrolled than well-controlled asthma, compared to the participants from Sale (adjusted relative risk ratio 2.71 95% CI: 1.02, 7.21, $p = .046$).

Conclusion: Three and a half years after exposure, coal mine fire smoke did not appear to be associated with more severe asthma symptoms or worse lung function but might be associated with poorer asthma control.

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Introduction

Air pollution is a well-recognized environmental asthma trigger (1–3). Airborne particulate matter (PM), specifically respirable particles with a median aerodynamic diameter less than 10 μm (PM₁₀), can cause asthma exacerbations, impair lung function, and possibly contribute to the development of asthma (1,3–10). Fine particles less than 2.5 μm (PM_{2.5}) have been of particular interest as they enter the lung periphery and may cause greater respiratory effects, including more severe asthma symptoms (3,7,8,11).

In February 2014, a fire started in the Hazelwood open cut brown coal mine in the Latrobe Valley of South-eastern Australia. For 6 weeks, smoke from the fire affected residents in the adjacent town of Morwell (population approximately 14 000 (12)), resulting in

community concerns about potential long-term respiratory and other health effects of the smoke exposure.

To the best of our knowledge, there was no evidence in the literature about the impact of short-term coal mine fire smoke exposure on asthma in the long term (5). However, there was evidence for the impact of analogous exposures on respiratory health. For example, exposures to wildfire smoke have been associated with a concomitant increase in the severity of asthma symptoms, medication use, emergency presentations, and hospital admissions (13–19). Smoke from domestic coal combustion, which is of similar composition to coal mine fire smoke, has been associated with increased asthma symptoms, childhood asthma and reduced lung function (20–23).

The Hazelwood Health Study (HHS; www.monash.edu/hazelwood-health-study) was established to

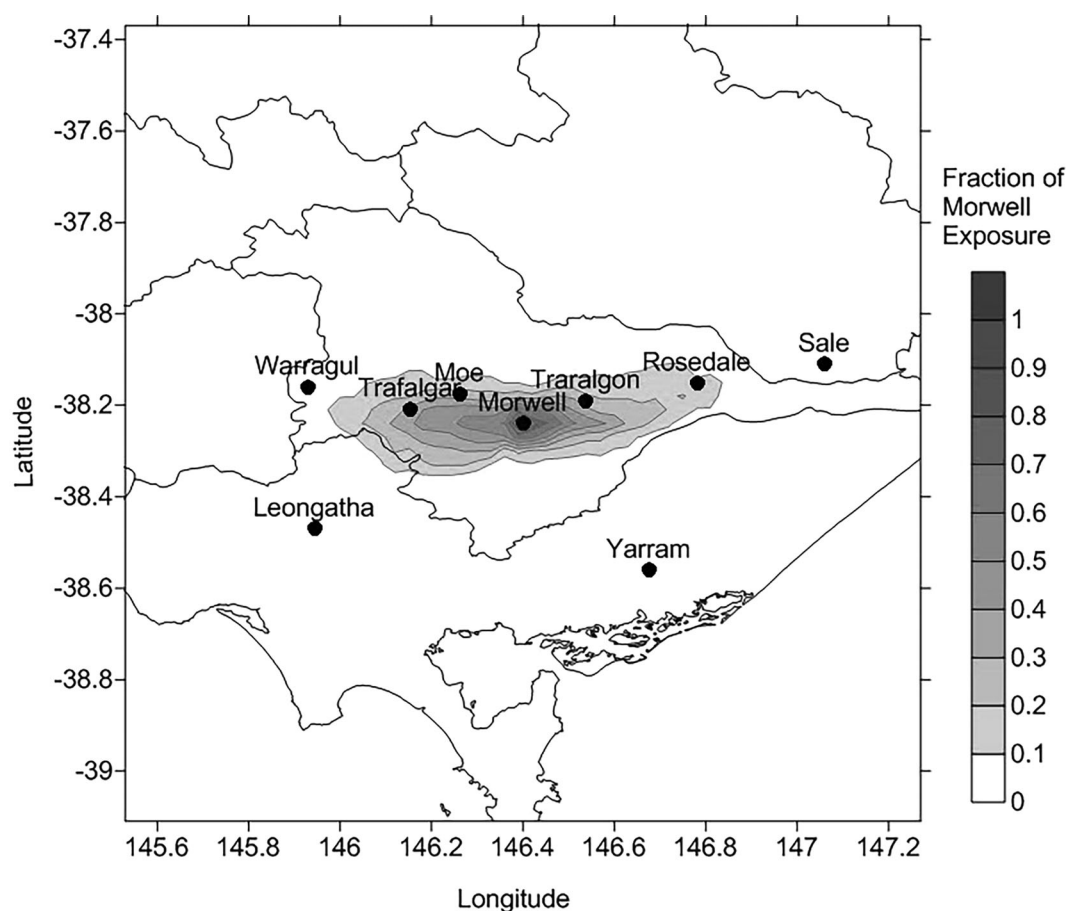


Figure 1. Modeled smoke exposure during the Hazelwood mine fire for towns in the Latrobe Valley including Morwell and Sale. Source: Emmerson et al. [24].

investigate potential long-term health outcomes in people exposed to the mine fire smoke. In 2016, the Adult Survey demonstrated that, compared to a control sample from the unexposed town of Sale (24), the exposed sample from Morwell had a significant increase in self-reported asthma symptoms, asthma diagnoses since 2014, and a higher mean symptom severity score amongst asthmatics (25,26).

This analysis aimed to further investigate findings in relation to asthma. Specifically, 3.5 years after the fire, we examined whether a group of people with asthma exposed to the coal mine fire smoke had more severe asthma symptoms, worse lung function or poorer control, compared to a group of an unexposed people with asthma.

Materials and methods

Study design and setting

This cross-sectional analysis examined data from the longitudinal HHS Respiratory Stream. Residents of Morwell ('exposed') and Sale ('unexposed') had previously been selected based upon mine fire-related

hourly $\text{PM}_{2.5}$ exposure levels modeled by the Commonwealth Scientific and Industrial Research Organization (CSIRO) Oceans and Atmosphere (Figure 1) (24,27). Modeling showed $\text{PM}_{2.5}$ levels exceeded Australian air quality standards ($25 \mu\text{g m}^{-3}$ as a 24-h average) on 23 days in southern Morwell and 12 days in eastern Morwell, reaching up to $3700 \mu\text{g m}^{-3}$ in southern Morwell in the first 2 days of the fire (24). There were no air quality breaches in Sale, a town with selected areas of comparable socio-economic status (SES), age distribution, household size, and population stability as Morwell (25).

Clinical testing occurred in Morwell between August and December 2017 and in Sale between January and March 2018. Ethics approval was obtained from the Monash University Human Research Ethics Committee (approval number 1078). All participants provided written informed consent.

Participants

Potential participants for the Respiratory Stream were drawn from the Adult Survey. Methodology for the

Adult Survey has been outlined previously (26). In summary, participants were aged at least 18 years and lived in Morwell or in selected areas of Sale during the fire. Participants were excluded from the Respiratory Stream if they had requested no further contact after the Adult Survey, had unknown age or sex, or were aged over 90 years.

From 3854 eligible Adult Survey participants, a weighted random sample of 1346 people was invited to the Respiratory Stream. Participants who had self-reported asthma were intentionally oversampled so they represented 40% of invitees. It was estimated that a sample size of 339 Morwell and 170 Sale participants was required to observe a 5 ml/year greater FEV₁ decline in exposed compared with non-exposed residents using a two-sample *t*-test with a two-sided *p* values of .05 and 80% power. We also assumed a 23.1 (±17.1) ml/year FEV₁ decline (28) among Sale residents and 18% loss to follow up rate.

Post-hoc power calculations were conducted for the asthma sub-sample with 64 participants from Sale and 165 participants from Morwell. With a two-sample *t*-test (two-sided *p* values of .05), the study would be able to detect a 0.413 difference in baseline FEV₁ *z*-score with 80% power (assuming a standard deviation (SD) of 1) as well as a 2.9% difference in post bronchodilator (BD) FEV₁%change with 80% power (assuming the mean post BD FEV₁%change was 7 and SD also 7).

Recruitment was via mailed invitation with follow-up of non-responders. Upon contact, potential participants were excluded if they had a contraindication to spirometry (29).

Demographics

Age, sex, ethnicity, employment status, educational background, and occupational exposure (working in dusty or polluted environments, such as coal mines, farms, or driving diesel trucks) had been previously collected by self-report for the Adult Survey (25).

Medication use and health risk factors

Trained respiratory scientists collected data in the Respiratory Stream clinic. Study data were managed using REDCap (Research Electronic Data Capture) (30) electronic data capture tools (Monash University, Victoria, Australia).

An interviewer-administered, standardized questionnaire recorded asthma medication use in the previous 3 months, including type and dosage regimen (31). Corticosteroid doses were calculated as

beclomethasone daily dose equivalents (μg), and categorized into low (100–200 μg), medium (>200–400 μg), or high (>400 μg) (2). Smoking history was obtained. Participants were classified as current smokers, former smokers (current nonsmokers with >100 cigarettes in their lifetimes), or never smoked (<100 cigarettes in their lifetimes) (32). Duration of smoking (years) was also calculated for current and former smokers (33). A self-reported history of nasal allergies or hay fever was recorded as an indicator of atopy. Height and weight were measured for body mass index (BMI) calculation (34).

Self-perceived respiratory health

Self-perceived respiratory health was assessed via an interviewer-administered validated questionnaire adapted from the European Community Respiratory Health Survey II (31). Diagnosis of asthma was by self-report. A validated asthma symptom severity score (35) was calculated from eight questions in the questionnaire. Asthma control was assessed with the validated, self-administered, 7-item Asthma Control Questionnaire (ACQ-7) (36). Responses were given on a 7-point scale with the score being a mean of the responses. Scores were categorized into well-controlled asthma (ACQ < 0.75), borderline control (0.75 ≤ ACQ ≤ 1.5) and uncontrolled asthma (ACQ > 1.5) (37).

Respiratory function tests

Standardization between staff and testing sites was achieved via Standard Operating Procedures and validated equipment. Participants received preparation instructions before testing. Medication was not withheld for ethical reasons, and the time inhaled medication that was last taken was recorded and adjusted for at the analysis stage.

Respiratory function was determined with spirometry (EasyOne Pro™ LAB Respiratory Analysis System; and Medical Technologies AG, Zurich, Switzerland) performed according to American Thoracic Society/European Respiratory Society (ATS/ERS) criteria (38). FEV₁, forced vital capacity (FVC) and FEV₁/FVC were measured. *Z*-scores for FEV₁, FVC and FEV₁/FVC were calculated using the Global Lung Initiative 2012 spirometry reference equations (39). The measurements were repeated 10 min after administration of three puffs of Salbutamol pressurized Metered Dose Inhaler (100 μg per activation) (40). Mean percentage change in FEV₁ for each group was calculated.

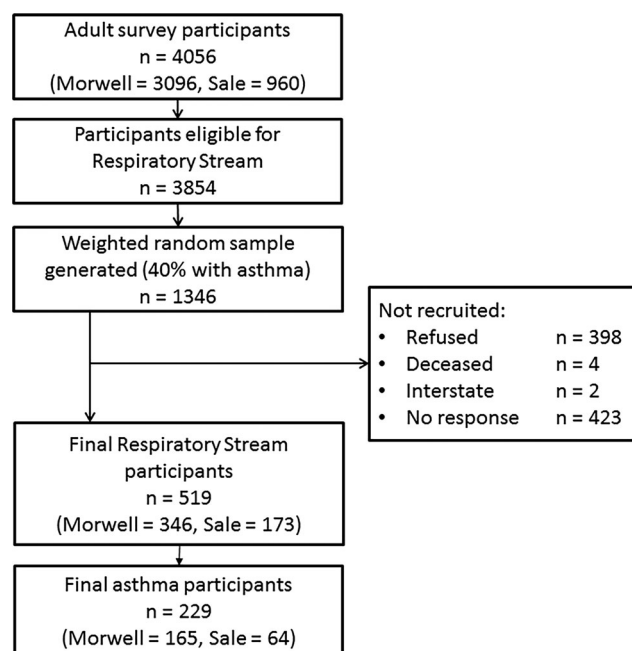


Figure 2. Participant recruitment flow diagram.

Fraction of exhaled nitric oxide (FeNO) was measured (Niox Vero FeNO device; Aerocrine, Solna, Sweden) according to ATS/ERS recommendations (41). Mean FeNO was calculated for each group.

Statistical methods

Statistical analysis and data transformations were performed using Stata version 15 (Stata Corporation, College Station, Texas 2017). Simple descriptive statistics summarized characteristics and clinical outcomes for the exposed and non-exposed participants, with crude differences between the groups assessed using Pearson chi-squared tests for categorical measures and *t*-tests for continuous measures. Linear regression models were used to compare the differences controlling for key confounders in continuous outcomes between the groups. Although the Sale comparison group was drawn from Level 1 Statistical Areas (42) with comparable socioeconomic indices to Morwell, there were still potential individual-level SES differences between these sampled groups. Hence, individual-level SES indicators such as education and employment were controlled in the model. FeNO was transformed to log scale due to its skewed distribution. Logistic regression models were used for binary outcomes, and multinomial models were used for categorical outcomes. Multiple-imputation procedures using chained equations were used in regression models to obtain more accurate estimates, and to control for non-response bias.

Results

The Respiratory Stream recruited 346 Morwell participants and 173 Sale participants, totaling 519 (39% of those eligible) (Figure 2). For this cross-sectional analysis, only data from the 229 asthmatic participants were analyzed: 165 from Morwell and 64 from Sale.

Table 1 shows the demographic characteristics of the participants. Morwell participants were more likely to be using medium to high doses of inhaled corticosteroids. In other respects, participants were similar.

The primary (unadjusted) outcomes are reported in Table 2. A higher proportion of Morwell participants reported asthma symptoms compared with Sale, but there were no statistical differences between the two groups. Other unadjusted outcomes are comparable between the groups, except for a slightly higher proportion of uncontrolled asthma in Morwell.

Tables 3 and 4 show univariate and multivariate regression analyses investigating the association between exposure (Morwell versus Sale) and a selection of the primary outcomes. After adjustment for confounders, there was no association with Morwell or Sale for any of the self-reported asthma symptoms or objective measures of lung function and airway inflammation.

When the ACQ-7 results were compared (Table 5), there was no effect of exposure (Morwell versus Sale) when those with borderline control ($0.75 \leq \text{ACQ} \leq 1.5$) were compared to those who were well-controlled ($\text{ACQ} < 0.75$). However, after adjustment for confounders, compared to Sale participants, Morwell participants had comparatively more uncontrolled asthma ($\text{ACQ} > 1.5$) than well-controlled asthma ($p = .046$). Being a current smoker and being on any type of asthma medication was positively associated with being borderline or uncontrolled compared to being well-controlled.

Discussion

Nearly 4 years after the Hazelwood mine fire, there was no evidence that exposed adult participants have significantly more severe asthma symptoms, worse lung function, or more eosinophilic airway inflammation compared to unexposed participants. However, compared to the Sale participants, there was evidence that Morwell participants have more uncontrolled than well-controlled asthma when assessed with a validated questionnaire.

One possible reason for this finding is that particulates in the smoke caused long-lasting respiratory damage in some exposed participants, reducing their

Table 1. Demographic characteristics for participants from Morwell and Sale.

Participant characteristics	Morwell N = 165		Sale N = 64		p-value
	n	%	n	%	
<i>Age/years</i>					.376
18–29	13	7.9	3	4.7	
30–44	45	27.3	13	20.3	
45–59	52	31.5	20	31.3	
60–74	39	23.6	23	35.9	
75+	16	9.7	5	7.8	
<i>Gender</i>					.948
Male	56	33.9	22	34.4	
Female	109	66.1	42	65.6	
<i>Ethnicity</i>					.532
Caucasian/White	164	99.4	64	100	
Other	1	0.6	0	0	
<i>Employment status</i>					.320
Employed	64	38.8	31	48.4	
Retired	44	26.7	19	29.7	
Not working due to poor health	20	12.1	6	9.4	
Other (unemployed, studying, home duties etc.)	37	22.4	8	12.5	
<i>Highest educational qualification</i>					.195
Secondary up to year 10	40	24.5	13	20.6	
Secondary year 11–12	34	20.9	10	15.9	
Certificate (trade/apprenticeship/technicians)	53	32.5	30	47.6	
University or other Tertiary Institute degree	36	22.1	10	15.9	
<i>Body mass index (BMI)</i>					.671
Underweight/normal (BMI < 25kg/m ²)	27	16.4	11	17.2	
Overweight (25 ≤ BMI < 30)	43	26.1	20	31.3	
Obese (BMI ≥ 30)	95	57.6	33	51.6	
<i>Smoking status</i>					.129
Never	80	48.5	22	34.4	
Former smoker	50	30.3	28	43.8	
Current smoker	35	21.2	14	21.9	
<i>Nasal allergies/hay fever</i>					.105
Yes	102	61.8	32	50	
No	63	38.2	32	50	
<i>Asthma medication</i>					.834
No inhaled medications	66	40	26	40.6	
Other asthma medications ^a	34	20.6	11	17.2	
Inhaled corticosteroids	65	39.4	27	42.2	
<i>Inhaled corticosteroid use^b</i>					.001
No use	101	61.2	40	62.5	
Low dose (100–200 µg)	16	9.7	17	26.6	
Medium dose (>200–400 µg)	21	12.7	2	3.1	
High dose (>400 µg)	27	16.4	5	7.8	
<i>Occupational exposure^c</i>					
Exposed	57	34.5	26	40.6	.394
	Mean	SD	Mean	SD	p-value
<i>Years of smoking among current and former smokers (n = 127)</i>	20.7	12.5	24.2	13.8	.212
<i>Age</i>	52.1	16.3	55.7	14.8	.108

^aOther asthma medications including short-acting beta agonists, short-acting muscarinic antagonists, long-acting beta agonists (LABA), long-acting muscarinic antagonists (LAMA), LAMA/LABA combinations.

^bDoses were calculated as beclomethasone equivalents (µg).

^cOccupational exposure from working in dusty or polluted environments, such as coal mines, farms, and driving diesel trucks.

ability to achieve good control. However, prior to the HHS, there had been no published evidence about the long-term respiratory impacts of short-term coal mine fire smoke exposure (5). Ambient air pollution studies have shown that acute exposures to PM damage the respiratory system through oxidative stress and inflammation, affecting respiratory function and causing asthma exacerbations (3,6,43). Furthermore, long-term exposures to PM from ambient pollution cause respiratory morbidity from chronic inflammation and airway remodeling (3,6,43). However, whether coal

mine fire smoke exposure causes similar long-term respiratory damage is unknown. The Early Life Follow-up stream of the HHS has recently reported that mothers whose children were exposed *in utero* were more likely to report respiratory symptoms such as runny nose, cough or wheeze in their children than those not exposed (44). There might be a critical time window for exposure to coal mine fire smoke to affect symptoms or lung function.

Other environmental factors at the time of testing might explain the difference in asthma control

Table 2. Respiratory questionnaires and lung function outcomes for participants from Morwell and Sale.

Outcome	Morwell N = 165		Sale N = 64		p-value
	n	%	n	%	
First asthma attack post-2014	18	11	8	12.9	.688
Most recent asthma attack post-2014	117	70.9	41	66.1	.478
Asthma-related respiratory symptoms					
Wheeze in the last 12 months	113	68.5	43	67.2	.852
Wheeze and breathlessness	90	54.5	33	51.6	.691
Wheeze without URTI	94	57	35	54.7	.754
Chest tightness in last 12 months	61	37	15	23.4	.057
Dyspnea at rest in last 12 months	47	28.5	15	23.4	.427
Dyspnea after exercise in last 12 months	108	65.9	35	54.7	.121
Woken with dyspnea in last 12 months	37	22.4	9	14.1	.162
Woken with cough in last 12 months	91	55.2	28	43.8	.145
Asthma attack in last 12 months	76	46.1	28	43.8	.754
	Mean	SD	Mean	SD	p-value
<i>Spirometry^a</i>					
FEV ₁ L	2.7	0.8	2.5	0.7	.164
FEV ₁ (z-score)	-0.8	1.1	-0.9	1.2	.665
FEV ₁ % predicted	88.5	16.8	86.2	18.4	.465
FVC L	3.5	1.0	3.5	0.8	.616
FVC (z-score)	-0.4	1.0	-0.3	1.0	.514
FVC % predicted	94.8	14.4	96.0	14.9	.609
FEV ₁ /FVC	74.6	10.1	71.3	11.7	.072
FEV ₁ /FVC (z-score)	-0.7	1.1	-1.0	1.2	.141
FEV ₁ /FVC % predicted	92.8	11.8	89.4	13.2	.100
<i>Post BD response^a</i>					
% change FEV ₁	6.4	7.2	8.0	8.0	.154
FeNO ^a	21.4	17.8	27.0	22.2	.066
Asthma symptom severity score	4.1	2.2	3.7	2.0	.140
	n	%	n	%	p-value
<i>Asthma control (ACQ-7)</i>					.255
Well-controlled asthma (ACQ < 0.75)	90	56.3	42	65.6	
Borderline controlled asthma (ACQ 0.75–1.5)	35	21.9	14	21.9	
Uncontrolled asthma (ACQ > 1.5)	35	21.9	8	12.5	

Abbreviations: URTI = Upper respiratory tract infection; FEV₁ = forced expiratory volume in 1 s; FVC = forced vital capacity; BD = bronchodilator; FeNO = fraction of exhaled nitric oxide; ACQ-7 = 7-item asthma control questionnaire(36).

^aResults were missing for five spirometry, nine post-BD response, and one FeNO test result due to difficulty in conducting the test or low test quality.

Table 3. Linear regression analysis of the association between exposure and primary outcomes.

	Univariate regression	Multivariate regression	
	Mean diff. (95% CI)	Adj mean diff. (95% CI)	p-value
FEV ₁ (z-score)	0.07 (-0.31, 0.46)	0.06 (-0.29, 0.40) ^a	.741
FEV ₁ /FVC (z-score)	0.28 (-0.11, 0.67)	0.25 (-0.12, 0.62) ^a	.180
% change FEV ₁ (BD response)	-1.66 (-3.98, 0.67)	-1.50 (-3.72, 0.73) ^b	.186
Log FeNO	-0.18 (-0.40, 0.04)	-0.20 (-0.42, 0.03) ^c	.084
Asthma symptom severity score	0.44 (-0.14, 1.02)	0.31 (-0.20, 0.81) ^d	.229

Abbreviations: FEV₁ = Forced expiratory volume in 1 s; FVC = forced vital capacity; BD = bronchodilator; FeNO = fraction of exhaled nitric oxide.

^aAdjusted for employment status, education, smoking status, years of smoking, occupational exposure, nasal allergies/hay fever, and whether the patient took asthma medication prior to lung function testing.

^bAdjusted for age, gender, body mass index, height, employment status, education, smoking status, years of smoking, occupational exposure, nasal allergies/hay fever, and whether the patient took asthma medication prior to lung function testing.

^cAdjusted for age, gender, body mass index, height, employment status, education, current smoking, inhaled corticosteroid dose, nasal allergies/hay fever, and whether the patient followed the FeNO preparation guidelines.

^dAdjusted for age, gender, body mass index, employment status, education, smoking status, occupational exposure, nasal allergies/hay fever, and asthma medication use.

findings. The ACQ only assessed asthma control in the week before the questionnaire and testing of Morwell subjects coincided with the grass pollen season in Victoria (October to December). We attempted to adjust for pollen with nasal allergies and hay fever; however, it still may have impacted on control. We also could not exclude the role of other unmeasured environmental factors during the testing period.

The lack of significant differences between exposed and unexposed participants for self-reported asthma symptoms and the symptom severity score was inconsistent with the Adult Survey results (25). However, the Adult Survey examined the prevalence of respiratory symptoms in the general population, including not only asthmatics but also people with chronic obstructive pulmonary disease and other respiratory

Table 4. Logistic regression analysis of the association between exposure and primary outcomes.

	Univariate regression	Multivariate regression	
	Crude OR (95% CI)	Adj OR ^a (95% CI)	p-value
Wheeze in the last 12 months	1.06 (0.57, 1.99)	0.90 (0.41, 1.97)	.798
Wheeze and breathlessness	1.13 (0.62, 2.04)	0.95 (0.46, 1.96)	.886
Wheeze without URTI	1.10 (0.61, 1.96)	1.07 (0.55, 2.09)	.838
Chest tightness in last 12 months	1.92 (0.98, 3.76)	2.11 (0.93, 4.77)	.073
Dyspnea at rest in last 12 months	1.30 (0.68, 2.50)	1.04 (0.47, 2.29)	.920
Dyspnea after exercise in last 12 months	1.60 (0.88, 2.90)	1.66 (0.83, 3.30)	.148
Woken with dyspnea in last 12 months	1.77 (0.79, 3.96)	1.77 (0.71, 4.41)	.217
Woken with cough in last 12 months	1.58 (0.85, 2.94)	1.66 (0.84, 3.25)	.141
Asthma attack in last 12 months	1.10 (0.61, 1.98)	1.05 (0.49, 2.26)	.897

Abbreviations: OR = Odds ratio; URTI = upper respiratory tract infection.

^aAdjusted for age, gender, body mass index, education, employment status, smoking status, occupational exposure, nasal allergies/hay fever, and asthma medication use.

Table 5. Multinomial logistic regression analysis of borderline versus well-controlled asthma and uncontrolled versus well-controlled asthma for ACQ-7.^a

	Borderline vs well-controlled			Uncontrolled vs well-controlled		
	Crude RRR	Adj. RRR ^b (95% CI)	p-value	Crude RRR	Adj. RRR ^b (95% CI)	p-value
<i>Morwell versus Sale</i>	1.16	1.28 (0.53, 3.13)	.585	2.25	2.71 (1.02, 7.21)	.046
<i>Smoking status</i>						
Never	Ref	Ref		Ref	Ref	
Former smoker	2.05	1.75 (0.73, 4.22)	.209	0.84	0.58 (0.21, 1.59)	.288
Current smoker	2.59	5.82 (1.74, 19.5)	.004	2.00	3.70 (1.19, 11.5)	.024
<i>Asthma medication</i>						
No	Ref	Ref		Ref	Ref	
Other asthma medications	4.92	5.66 (1.69, 18.9)	.005	8.91	9.75 (3.11, 30.6)	<.001
Inhaled corticosteroids	7.10	9.08 (3.20, 25.8)	<.001	13.76	17.44 (5.81, 52.4)	<.001

Abbreviations: RRR = Relative risk ratio.

^aACQ-7: 7-item asthma control questionnaire, where well-controlled asthma (ACQ < 0.75), borderline controlled asthma (ACQ 0.75–1.5) and uncontrolled asthma (ACQ > 1.5).

^bAdjusted for age, gender, body mass index, employment status, education, smoking status, asthma medication use, and nasal allergies/hay fever.

conditions. The inconsistent findings may be because the Adult Survey studied a larger number of people, including more asthmatics overall, giving it greater power to detect symptom differences.

Interestingly, we found a higher proportion of Morwell residents were on medium to high dose inhaled corticosteroids. Morwell participants may have been prescribed higher doses because they had more severe asthma, potentially due to smoke exposure. We might not have seen a difference in the self-reported asthma symptoms, symptom severity score or lung function results because the participants were adherent with their medication and had good inhaler technique.

Strengths and limitations

This analysis provides novel evidence that short-term coal mine fire smoke exposure has limited long-term impacts on asthma. To the best of our knowledge, there are no similar studies in the literature. We used validated questionnaires and objective respiratory function tests following standard guidelines, which minimized the risks associated with self-reported measures of health, such as differential reporting and recall bias.

However, our study also has limitations. In this cross-sectional analysis, we could only examine possible associations between coal mine fire smoke and asthma; we cannot infer causation. We did not collect information on all individual-level SES indicators (e.g. individual and family income) which also possibly influenced health service and medication usage. Other potential confounders not collected included medication adherence and inhaler technique, potentially impacting on asthma severity and control. Follow up of these participants over time through the longitudinal HHS will provide further evidence regarding the impact of the fire on Morwell asthmatics and facilitate evaluation of additional confounders.

Our study may be subject to other biases. Asthma was self-reported. Our study sample was drawn from the Adult Survey participants; by chance we may have sampled more mild asthmatics from Morwell than Sale. Furthermore, selection bias due to volunteer effects and non-response bias cannot be excluded. It is difficult to say which way these may have biased our results, but the results should only be generalized to other rural populations with caution.

Finally, our participants were drawn from a study population determined by sample size calculations powered to detect a change in FEV₁ over time for the

Respiratory Stream. Therefore, our study may have been underpowered to detect small short-term differences in asthma symptoms or lung function between the groups.

Nearly 4 years after the Hazelwood mine fire, our study has found some evidence of poorer asthma control in adults with asthma exposed to the mine fire smoke, but no significant difference in severity of asthma symptoms or lung function. Further research is needed to determine the clinical importance of our findings and whether coal mine smoke exposure has a causal role in long-term asthma control. The planned longitudinal study will examine this question for both asthma control and severity. Investigating the association between individual PM_{2.5} exposure and asthma-related outcomes would help characterize the degree of risk more comprehensively.

The risk of coal mine fires is increasing due to climate change (5), with potential impact on the burden of disease. Evidence for the impact of coal mine fire smoke on health will allow governments and public health policy makers to plan for future similar events to minimize harm to populations at risk of exposure.

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Disclosure statement

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Data availability statement

Restrictions apply to the availability of these data, which were used under license for this study. Data are available from the authors with the permission of the Victorian Department of Health and Human Services.

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