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Occupational exposures associated with severe exacerbation of asthma

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SUMMARY

BACKGROUND—The exacerbation of asthma by workplace conditions is common, but little is known about which agents pose a risk.

OBJECTIVE—We used data from an existing survey of adults with asthma to identify occupational exposures associated with severe exacerbation of asthma.

DESIGN—Questionnaires were completed by 557 working adults with asthma. Severe exacerbation of asthma in the past 12 months was defined as asthma-related hospitalization, or reports of both unplanned asthma care and treatment with a short course of oral corticosteroids. Occupational exposures for the same time period were assessed using an asthma-specific job exposure matrix. We modeled severe exacerbation to yield prevalence ratios (PRs) for exposures while controlling for potential confounders.

RESULTS—A total of 164 participants (29%) were positive for severe exacerbation, and 227 (40.8%) were assessed as being exposed to asthma agents at work. Elevated PRs were observed for several specific agents, notably the irritant subcategories of environmental tobacco smoke (PR 1.84, 95%CI 1.34–2.51) among all participants, inorganic dusts (PR 2.53, 95%CI 1.37– 4.67) among men, and the low molecular weight subcategory of other highly reactive agents (PR 1.97, 95%CI 1.08–3.60) among women.

CONCLUSION—Among working adults with asthma, severe exacerbation was associated with several occupational agents.

Keywords

work-exacerbated asthma; job-exposure matrix; occupational epidemiology

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WORK-RELATED ASTHMA comprises occupational asthma (OA) that is caused by conditions at work and work-exacerbated asthma (WEA), in which existing asthma is made worse by workplace conditions.[1] Work-related asthma is common, with work accounting for an average 16.9% of all new adult-onset asthma cases,[2] and with WEA detected in an estimated 21.5% of working adults with asthma.[3] While substantial costs and lost work time are associated with both WEA and OA,[4,5] WEA has received less attention in terms of research and prevention efforts.[1] WEA is potentially preventable by minimizing harmful workplace exposures,[3] but a better understanding of which agents pose a risk is needed to guide prevention efforts. While surveillance in the United States indicates that WEA is frequently caused by irritants, low molecular weight (e.g., acids, bases, aldehydes) and high molecular weight (e.g., latex) agents are also implicated.[6] The absence of denominators (i.e., the number of workers exposed) prohibits using surveillance data to estimate agent-specific risk.

We used data from a previous study to identify occupational risk factors for exacerbation of asthma.[7] In previous analyses of data from this study, we determined WEA status on a case-by-case basis. However, for the current investigation, we modeled severe exacerbation of asthma and determined which occupational exposures were associated with it, while simultaneously controlling for potential confounders. We assessed occupational exposures using a new asthma-specific job-exposure matrix (JEM).[8]

STUDY POPULATION AND METHODS

The details of the study are presented elsewhere.[9] The protocol was approved by the Human Subjects Review Board of the National Institute for Occupational Safety and Health (NIOSH, Washington DC, USA), and all participants provided informed consent. Study participants were patients at a health maintenance organization (HMO) in the state of Massachusetts, USA. The HMO electronic medical records were reviewed during 2000–2001 to identify cases of asthma aged 18–44 years. Invitees were required to have active asthma as indicated by a recorded asthma diagnosis and treatment for asthma in the past 12 months. With the goal of studying exacerbation rather than onset of asthma during the 12 months before interview, we reviewed paper medical records to confirm that asthma onset was at least 1 year before enrollment, and excluded those who reported their first asthma attack as occurring <14 months before interview. A total of 598 (61%) of the 978 invitees completed a telephone questionnaire during 2001–2002; 41 were excluded because they had been unemployed in the previous 12 months, leaving 557 participants for the current analysis.

Responses to questionnaire items provided information on occupations, severe exacerbation of asthma, and demographics. Each job worked in the past 12 months was coded using the 1988 International Standard Classification of Occupations (ISCO-88).[10] We assessed occupational exposures with a new asthma-specific JEM initially intended for use in Northern Europe, called the N-JEM.[8] Development of the N-JEM followed the same principles used to create an earlier asthma JEM.[11,12] Two industrial hygienists assessed each occupation as exposed, not exposed, or uncertain/low exposed. The ‘exposed’ label was assigned to occupations judged as having at least half of the workers with a high probability

of exposure. The raters judged exposure (no/yes) for each of several subcategories in four major categories of agents: high molecular weight (HMW), low molecular weight (LMW), irritants (IRR), and accidental peak exposures to irritants (PEAKS). Assessments were then discussed with two occupational medicine specialists until consensus was reached.[8]

We defined severe exacerbation of asthma based on self reports if in the past 12 months the participant had either been hospitalized for asthma, or received both unplanned care for an acute asthma attack (at a doctor's office, urgent care facility, or emergency department) and a short course of oral corticosteroids for asthma. This definition is consistent with recent recommendations for defining severe exacerbation of asthma.[13,14]

As the outcome was relatively common, we calculated prevalence ratios (PRs) rather than odds ratios,[15,16] using a Cox regression model with robust variance[17] and a constant follow-up time.[18] We fitted a base model for severe exacerbation using the following candidate variables: sex (female vs. male), age in years (30–38 and 39–44 vs. 18–29), highest education (college degree or more vs. some college or less), race (non-white vs. white), cigarette smoking (ever vs. never), age at asthma onset (≥18 vs. <18 years), history of allergies based on whether a doctor had ever said the participant had hay fever or skin allergies (yes vs. no), and asthma severity based on review of the medical records (persistent vs. intermittent).[19] We used forward selection and backward elimination, with $P \leq 0.15$ as the criterion for retention in the model. Occupational exposure variables developed using the N-JEM were then added to the base model, as detailed in the Results. Statistical tests were considered significant if $P \leq 0.05$. All data analyses were conducted using SAS® software version 9.2 (Statistical Analysis System, Cary, NC, USA).

RESULTS

The 557 participants included approximately twice as many women (68%) as men (32%), and were overwhelmingly White (94%). The median age was 34 years, the median age at asthma onset was 13, and 345 (62%) participants had experienced onset before the age of 18 years; 40% were smokers (18% current, 22% former), 74% had allergies, and 37% had completed at least college education. The distribution by level of asthma severity was 31% mild intermittent and 69% mild/moderate/severe persistent. The group had worked 771 jobs in the 12 months before interview, with two thirds (n = 377, 68%) working one job, one fourth (n = 151, 27%) two jobs, and only a few three (n = 25), four (n = 3), or five (n = 1) jobs. The participants worked primarily in white collar and service jobs (n = 480, 86%, in ISCO-88 Groups 1–5) and infrequently in blue collar jobs (n = 90, 16%, in ISCO-88 Groups 6–9). Women were more likely than men to have jobs as technicians and associate professionals and service workers, but were less likely to be employed in craft trades and as plant and machine operators and assemblers.

Approximately two in every five participants (n = 227, 40.8%) were assessed by the N-JEM to have experienced occupational exposure to asthma-related agents (i.e., HMW, LMW, IRR, and PEAKS) in the past 12 months (Table 1). IRR was the most common exposure, with about one fourth of all participants (27.1%), and environmental tobacco smoke (ETS) was the most common IRR subcategory (11.3%). The other major agent categories in

decreasing frequency were HMW (13.8%), LMW (4.8%), and PEAKS (1.1%). More men than women were exposed to asthma agents (48.3% vs. 37.1%, $P = 0.02$); men were more likely to have IRR and LMW exposures, and less likely to be exposed to HMW agents (Table 1). Many exposure subcategories had small sample sizes when the data were subdivided by sex.

Severe exacerbation was reported by 164 participants (29%). It was more common among women than men and those with persistent than intermittent severity, and was less common among the oldest participants (Table 2). Severe exacerbation showed little variation in frequency by education, race, smoking status, age at asthma onset, and history of allergies. The crude frequency was greater among participants with occupational exposure than among those with no exposure (35% vs. 25%, Table 3). By occupational exposure subcategories, crude values were notably high for the LMW agents epoxy (40%), other highly reactive agents (50%), and isocyanates (50%), and the IRR agents inorganic dusts (52%), metalworking fluids (42%), and ETS (48%).

The base regression model for severe exacerbation included covariates for sex, age (39–44 years vs. other ages), and asthma severity (persistent vs. intermittent). Participants with jobs that had no asthma-related exposures populated the common reference category for all occupational covariates. We did not estimate PRs for exposure categories with <5 exposed participants or no severe exacerbation cases. The PR for any exposure to asthma-related agents (other than uncertain or low exposure) was 1.43 (95% confidence interval [CI] 1.10–1.84, $P = 0.007$) (model not shown). We fitted separate models for each exposure category and subcategory (Table 4). All PRs for major exposure categories exceeded 1 and were statistically significant for LMW agents, IRR agents, and PEAKS. As all six participants with PEAKS also had IRR exposure and three had LMW exposure, it was impossible to evaluate the effect of PEAKS separately. With LMW and IRR in the same model (not shown), the PR (1.58, 95%CI 1.20–2.08, P=0.001) for IRR was similar to that observed when this exposure was in a model by itself, but the PR for LMW agents was no longer statistically significant (1.47, 95%CI 0.94–2.29, $P = 0.09$).

An association with severe¼exacerbation of asthma was observed for seven exposure subcategories (Table 4) when each agent was included in a regression model with the potential confounders: the LMW agents epoxy (PR 2.50, $P = 0.046$), other highly reactive agents (PR 1.93, $P = 0.03$), and isocyanates (PR 3.11, $P = 0.001$); and the IRR agents inorganic dusts (PR 3.61, $P < 0.0001$), metalworking fluids (PR 2.84, $P = 0.005$), combustion particles (PR 1.52, $P = 0.07$), and ETS (PR 1.88, $P = 0.0001$). Many participants experienced concurrent exposures and, in particular, exposure to inorganic dusts was related to all other implicated exposures except ETS. Epoxy and isocyanates overlapped so completely with inorganic dusts that it was impossible to test for their independent effects, while inorganic dusts had an effect that was separate from these two exposures (data not shown). In a model with the other five exposures, both inorganic dusts (PR 2.41) and ETS (PR 1.84) had strong positive effects (P<0.05), while the PR for other highly reactive agents was elevated but not statistically significant (PR 1.65, $P = 0.08$; Model A, Table 5).

Small sample sizes limited the exposure subcategories that we could examine separately for men and women. The model for men (Model B, Table 5) included covariates for four of the IRR agents implicated above, but excluded other highly reactive agents because only four men were exposed. The statistically significantly positive findings for men were inorganic dusts and ETS, similar to the findings for all participants. Women had sufficient sample sizes for only three of the seven exposures implicated, and this did not include inorganic dusts. The regression model specific to women (Model C, Table 5) had elevated PRs for other highly reactive agents (PR 1.97, $P = 0.03$) and ETS (PR 1.50, $P = 0.03$).

DISCUSSION

IRRs had the strongest association with severe exacerbation of asthma, and particularly the exposure subcategories of inorganic dusts and ETS. Based on surveillance conducted in the United States, mineral and inorganic dusts are frequently identified as the putative cause of WEA.[6,20] Exposure to ETS was the most common occupational exposure among the 16 exposure subcategories, and has frequently been reported as a cause of WEA in other studies.[6,21–24] Cigarette smoking and ETS in the workplace are less common now than when the interviews were conducted in 2001–2002.[25–28] Specifically, for the state of Massachusetts (the site of the current study), the 2010 Behavioral Risk Factor Surveillance System survey found that 5.4% of non-smoking adults reported being exposed to secondhand smoke at work during the past week, down from 8% in 2003.[29] However, the 2010 prevalence indicates that workplace ETS is still common, exceeding the prevalence of 13 of the 16 occupational exposure subcategories in the current investigation. The LMW subcategory of other highly reactive agents was a risk factor for severe exacerbation among women, and this category includes chemicals such as acids with irritant properties that may have contributed to exacerbation.

This investigation had several strengths: it was conducted in an HMO, a quasi-population-based setting, and participants were employed in different occupations and industries. Furthermore, by using regression to model severe exacerbation of asthma, we were able to control for potential confounders while estimating the strength of the association between the outcome and occupational exposures. The asthma-specific N-JEM has been used successfully in another study of asthma,[8] and provided objective assessments of occupational exposures, thus avoiding the bias that has been observed with self-reported exposures.[30] In addition, the N-JEM assigned participants to more specific subcategories of occupational exposures than was accomplished in other population-based studies that used either self reports and expert evaluation of workplace exposures[31] or another JEM[9] when investigating exacerbation of asthma.

The current study had several limitations. As the N-JEM was developed with a focus on the onset of asthma rather than exacerbation, exposures relevant to the latter but not the former may have been missed. When we limited the analysis to the 476 subjects with asthma onset before starting jobs held in the past 12 months, our findings remained unchanged (data not shown), suggesting that the results were not driven by exposure related to asthma onset. While the same jobs in northern Europe and the United States generally have similar occupational exposures, differences may have resulted in exposure misclassification.

Participants were required to have evidence in their medical records of treatment for asthma in the past 12 months. Very mild cases were therefore not included, and the findings are not necessarily relevant to them. While the participants' occupational exposures and severe exacerbation events occurred in the same 12-month period, it is uncertain whether exposure always preceded the outcome. This probably introduced non-differential misclassification of dichotomous exposure variables, thus biasing exposure PRs to the null.[32] As the maximum age at enrollment was 44 years, the results may not be relevant to older asthma patients. Excluding older adults probably did avoid confusing asthma with other respiratory conditions, notably chronic obstructive pulmonary disease.

Concurrent occupational exposures and small sample sizes limited our ability to test for exposure-response relationships. An extreme overlap with other exposures meant we were unable to assess the independent effects of PEAKS, epoxy, and isocyanates. Sample sizes were a particular problem when we divided the study group by sex. The small number of participants who had worked in manual labor jobs is indicative of Massachusetts, USA, which has fewer blue collar workers than most other states.[33] At the same time, the pattern of more white vs. blue collar jobs is indicative of the entire United States, and current findings are relevant beyond Massachusetts.

CONCLUSION

The current study identified several occupational exposures associated with exacerbation of asthma, providing an additional level of detail that was not available in other population-based studies that used a risk-set approach. The results suggest that minimizing exposure to the implicated agents might reduce the frequency of WEA. Additional studies with larger sample sizes are needed to refine our understanding of occupational risk factors for exacerbation of asthma, and to assess the impact of different interventions.

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Table 1

Frequency of asthma-related occupational exposures as assessed by the N-JEM for the 771 jobs worked by 557 adults with asthma

Occupational exposure assessed by the N-JEM	All (*n* = 557) *n* (%)	Men (*n* = 180) *n* (%)	Women (*n* = 337) *n* (%)
Any exposure to asthma-related agents (HMW+LMW+IRR+PEAKS)*	227 (40.8)	87 (48.3)	140 (37.1)
HMW agents	77 (13.8)	15 (8.3)	62 (16.4)
Animal antigens, mixed agricultural agents (animals)	4 (0.7)	0	4 (1.1)
Flour and plant antigens, mixed agricultural agents (not animals)	9 (1.6)	6 (3.3)	3 (0.8)
Mite and insect antigens, enzymes, molds, bioaerosols	9 (1.6)	7 (3.9)	2 (0.5)
Latex	56 (10.1)	3 (1.7)	53 (14.1)
Pharmaceutical products	16 (2.9)	0 (0)	16 (4.2)
LMW agents	27 (4.8)	16 (8.9)	11 (2.9)
Highly reactive agents, acrylates	9 (1.6)	5 (2.8)	4 (1.1)
Highly reactive agents, epoxy	10 (1.8)	8 (4.4)	2 (0.5)
Other highly reactive agents, such as amines, aldehydes, acids, anhydrides, chromates, curing agents, reactive gases and dyes	10 (1.8)	4 (2.2)	6 (1.6)
Highly reactive chemicals, isocyanates	12 (2.2)	10 (5.6)	2 (0.5)
IRR agents	151 (27.1)	78 (43.3)	73 (19.4)
Cleaning agents	12 (2.2)	6 (3.3)	6 (1.6)
Organic dust, textile industry	1 (0.2)	1 (0.6)	0
Organic dust, wood or paper	16 (2.9)	14 (7.8)	2 (0.5)
Inorganic dusts and fumes, mining and building construction workers, and others exposed to inorganic dusts	21 (3.8)	19 (10.6)	2 (0.5)
Metalworking fluids	12 (2.2)	11 (6.1)	1 (0.3)
Combustion particles/fumes: vehicle/motor exhaust	47 (8.4)	25 (13.9)	22 (5.8)
High probability of exposure to ETS	63 (11.3)	20 (11.1)	43 (11.4)
PEAKS	6 (1.1)	5 (2.8)	1 (0.3)
Uncertain or low exposure*	31 (5.6)	5 (2.8)	26 (6.9)
Unexposed	303 (54.4)	90 (50.0)	213 (56.5)

N-JEM = asthma-specific job-exposure matrix; HMW = high molecular weight; LMW = low molecular weight; IRR = irritant; PEAKS = accidental peak exposures to irritants; ETS = environmental tobacco smoke.

* As four of the 227 participants with HMW, LMW, IRR, or PEAKS exposure had another job with uncertain or low exposure, they were also counted among the 31 in this other category.

Table 2

Frequency of severe exacerbation among 557 working adults with asthma by selected characteristics

Characteristic	*N* in category	Severe exacerbation	
		n (%)	*P* value*
Sex			0.007
Male	180	39 (22)	
Female	377	125 (33)	
Age, years			
18–29	202	69 (34)	Reference
30–38	184	51 (28)	0.21
39–44	171	44 (26)	0.10
Highest educational level			
Some college or less	350	107 (31)	Reference
College degree or more	207	57 (28)	0.51
Race†			0.59
White	524	153 (29)	
Non-White	31	11 (35)	
Cigarette smoking status			
Never	334	96 (29)	Reference
Former	123	37 (30)	0.87
Current	100	31 (31)	0.76
Age at asthma onset, years			0.56
<18	345	98 (28)	
≥18	212	66 (31)	
History of allergies‡			0.64
No	147	46 (31)	
Yes	410	118 (29)	
Asthma severity			
Mild intermittent	171	35 (20)	Reference
Mild persistent	170	59 (35)	0.005
Moderate/severe persistent	216	70 (32)	0.01

*Based on continuity-corrected χ^2 statistic.

†Missing for two participants.

‡Doctor told participant s/he had hay fever or skin allergies.

Table 3

Frequency of severe exacerbation by different occupational exposures as assessed using the N-JEM for the 771 jobs worked by 557 adults with asthma

Occupational exposure assessed by the N-JEM	All *n/N* (%)	Men *n/N* (%)	Women *n/N* (%)
Any exposure to asthma-related agents (HMW+LMW+IRR+PEAKS)*	79/227 (35)	27/87 (31)	52/140 (37)
HMW agents	23/77 (30)	3/15 (20)	20/62 (32)
Animal antigens, mixed agricultural agents (animals)	1/4 (25)	0	1/4 (25)
Flour and plant antigens, mixed agricultural agents (not animals)	1/9 (11)	0/6 (0)	1/3 (33)
Mite and insect antigens, enzymes, molds, bioaerosols	2/9 (22)	1/7 (14)	1/2 (50)
Latex	20/56 (36)	2/3 (67)	18/53 (34)
Pharmaceutical products	5/16 (31)	0	5/16 (31)
LMW agents	11/27 (41)	6/16 (38)	5/11 (45)
Highly reactive agents, acrylates	2/9 (22)	2/5 (40)	0/4 (0)
Highly reactive agents, epoxy	4/10 (40)	4/8 (50)	0/2 (0)
Other highly reactive agents, such as amines, aldehydes, acids, anhydrides, chromates, curing agents, reactive gases and dyes	5/10 (50)	1/4 (25)	4/6 (67)
Highly reactive chemicals, isocyanates	6/12 (50)	5/10 (50)	1/2 (50)
IRR agents	55/151 (36)	25/78 (32)	30/73 (41)
Cleaning agents	2/12 (17)	0/6 (0)	2/6 (33)
Organic dust, textile industry	0/1 (0)	0/1 (0)	0
Organic dust, wood or paper	3/16 (19)	3/14 (21)	0/2 (0)
Inorganic dusts and fumes, mining and building construction workers, and others exposed to inorganic dusts	11/21 (52)	9/19 (47)	2/2 (100)
Metalworking fluids	5/12 (42)	4/11 (36)	1/1 (100)
Combustion particles/fumes: vehicle/motor exhaust	15/47 (32)	7/25 (28)	8/22 (36)
High probability of exposure to ETS	30/63 (48)	10/20 (50	20/43 (47)
PEAKS	3/6 (50)	2/5 (40)	1/1 (100)
Uncertain or low exposure*	9/31 (29)	2/5 (40)	7/26 (27)
Unexposed	77/303 (25)	11/90 (12)	66/213 (31)

N-JEM = asthma-specific job-exposure matrix; HMW = high molecular weight; LMW = low molecular weight; IRR = irritant; PEAKS = accidental peak exposures to irritants; ETS = environmental tobacco smoke.

Table 4

Prevalence ratios for occupational exposure from regression models of severe exacerbation of asthma

Occupational exposure assessed by the N-JEM	PR (95%CI)
Unexposed: reference category*	1.00
HMW agents	1.09 (0.74–1.60)
Mite and insect antigens, enzymes, molds, bioaerosols	1.27 (0.40–4.07)
Flour and plant antigens, mixed agricultural agents (not animals)	0.65 (0.1 1–3.82)
Latex	1.21 (0.81–1.82)
Pharmaceutical products	1.01 (0.48–2.13)
LMW agents	1.91 (1.19–3.09)†
Highly reactive agents, acrylates	1.14 (0.29–4.45)
Highly reactive agents, epoxy	2.50 (1.02–6.14)‡
Other highly reactive agents, e.g., amines, aldehydes, acids, anhydrides, chromates, curing agents, reactive gases, and dyes	1.93 (1.09–3.43)‡
Highly reactive chemicals, isocyanates	3.11 (1.56–6.20)†
IRR agents	1.61 (1.22–2.12)†
Cleaning agents	0.79 (0.24–2.69)
Organic dust, wood or paper	1.26 (0.42–3.79)
Inorganic dusts and fumes, mining and building construction workers, and others exposed to inorganic dusts	3.61 (2.18–5.97)†
Metalworking fluids	2.84 (1.38–5.84)†
Combustion particles/fumes: vehicle/motor exhaust	1.52 (0.96–2.39)§
High probability of exposure to ETS	1.88 (1.36–2.59)†
Accidental peak exposures to irritants	3.26 (1.46–7.29)†
Uncertain or low exposure	1.06 (0.60–1.89)

N-JEM = asthma-specific job-exposure matrix; PR = prevalence ratio; CI = confidence interval; HMW = high molecular weight; LMW = low molecular weight; IRR = irritant; ETS = environmental tobacco smoke.

*A separate regression model was fitted for each occupational exposure. The common reference category for occupational exposures comprised those who had jobs with no exposure to asthma-related agents. Each model also included three potential confounders: sex, the oldest of three age categories (39-44 years), and asthma severity (persistent vs. intermittent).

†$P < 0.01$.

‡$P \leq 0.05$.

§$0.05 < P < 0.10$.

Table 5

PRs from regression models of severe exacerbation of asthma with several occupational exposure subcategories*

Occupational exposure assessed by the N-JEM	PR (95%CI)	*P* value
Model A: all participants		
LMW agent		
Other highly reactive agents	1.65 (0.93–2.91)	0.08
IRR agents		
Inorganic dusts	2.41 (1.46–3.99)	0.0006
Metalworking fluids	1.34 (0.65–2.74)	0.43
Combustion particles	1.24 (0.80–1.91)	0.33
High probability of exposure to ETS	1.84 (1.34–2.51)	0.0001
Model B: men		
IRR agents		
Inorganic dusts	2.53 (1.37–4.67)	0.003
Metalworking fluids	1.51 (0.60–3.79)	0.38
Combustion particles	1.33 (0.63–2.80)	0.45
High probability of exposure to ETS	3.25 (1.72–6.14)	0.0003
Model C: women		
LMW agent		
Other highly reactive agents	1.97 (1.08–3.60)	0.03
IRR agents		
Combustion particles	1.16 (0.66–2.04)	0.60
High probability of exposure to ETS	1.50 (1.04–2.17)	0.03

PR = prevalence ratio; N-JEM = asthma-specific job-exposure matrix; CI = confidence interval; LMW = low molecular weight; IRR = irritant; ETS = environmental tobacco smoke.

*The common reference category for occupational exposures comprised those who had jobs with no exposure to asthma-related agents. Each model also included three potential confounders: sex, the oldest of three age categories (39-44 years), and asthma severity (persistent vs. intermittent).