RESEARCH REPORT

Respiratory diseases and pesticide exposure: a case-control study in Lebanon

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Study objective: To evaluate the odds of being exposed to pesticides in asthmatic adults.

Design: A case-control study was performed in Lebanon.

Setting: People were approached when consulting physicians as outpatients.

Patients: Asthmatic patients and non-asthmatic controls in several Lebanese hospitals were interviewed.

Main results: The study included 407 subjects from 10 medical centres. Any exposure to pesticides was associated to asthma (OR=2.11 (1.47 to 3.02); $p<10^{-4}$). Occupational use presented the highest association (OR=4.98 (1.07 to 23.28); $p=0.02$), followed by regional exposure (OR 3.51 (2.11 to 5.85); $p<10^{-4}$). Results were confirmed by multivariate analysis, particularly for regional exposure (OR_a=2.78; $p=0.02$) and house exposure (OR_o=2.17; $p=0.001$).

Conclusions: Results are comparable to those found in other studies; especially for occupational exposure. Pesticides toxicological effects may explain chronic respiratory symptoms and asthma associations found with all exposure types. Pesticide exposure was associated with asthma in Lebanese adults.

Asthma is a chronic inflammatory disease associated with reversible airway obstruction. Low concentrations of irritants common to the agricultural environment, such as pesticides, may aggravate underlying asthma; whether pesticides cause asthma themselves has yet to be more investigated and proved.¹

Occupational use of pesticides may be involved with respiratory diseases. In an epidemiological study on grapes and apple trees workers, excessive chronic respiratory symptoms have been found: dyspnea and suffocation crisis (74.1% v.5.2%), cough (27.6% v.15.7%), expectoration (25.3% v.13.8%), and chronic bronchitis (20.7% v.13.9%), and also acute symptoms in smokers of more than 10 years employment duration. Low respiratory volumes (0.54 v.0.73 in FVC measured-predicted difference, and 0.12 v.0.35 in FEV₁ measured-predicted difference) were noted even after adjustment for smoking, suggesting obstructive and restrictive effects.² In a study by Gamsky and colleagues, reduced FVC was seen in grape workers; the mean cross-shift decrement (150 ml) in FVC was significantly greater than in the comparison group.³ Farming and agricultural work were later associated with increased asthma and respiratory symptoms prevalence (cough and phlegm)⁴ and decreased respiratory function.⁵ In Lebanon, we have undertaken a study on agricultural workers and found that they reported more upper respiratory tract infections (PR = 9.39; $p<10^{-4}$) and allergic (PR = 1.58; $p=0.01$) symptoms than non-agricultural ones.⁶ Furthermore, pesticide workers have been shown to have higher mortality rate than the general population, especially the one linked to asthma: standardised mortality ratio = 3.45; 95% confidence interval 1.39 to 7.10.⁷

Pesticides are among involved agents in the appearance of these effects,⁸ probably not attributable to direct local effects, but to effects on the lungs from the systemic circulation after inhalation and absorption; they could occur at doses several fold inferior to those causing similar responses after dermal or oral exposure.⁹ In fact, inhalation of pesticides on the worksite occurs, provided that one or more of the following conditions exist: the chemical in use is quite volatile, application is in an enclosed poorly ventilated area, or the

manner of application leads to an aerosol cloud of finely dispersed droplets that do not readily settle.¹

Many indirect and direct exposure opportunities also exist for farmers' next of kin who live in places where pesticides are used or brought home by lack of decontamination measures.^{10–11} Subjects living in heavily treated regions are further exposed to pesticide air pulverisation and contaminated food and water.^{12–13} The general population is also exposed to pesticides by domestic use or accidental exposure. Non-occupational chronic respiratory indoor exposure can result in high annual cumulative doses.¹⁴

Several accidents have occurred, where people living next to fumigant treated surface (most toxic pesticides used in agriculture)¹² experienced laryngeal irritation, headache, or even acute pulmonary irritation, cough, and death.¹⁵ With paraquat, several symptoms were reported such as cough, rhinitis, dyspnea, wheezing, etc.¹⁶

Few studies have investigated the health effects of non-occupational pesticide exposure, but living in areas where pesticides are used on crops may represent an environmental health concern, particularly acute intoxication symptoms affecting respiratory, digestive, and neurological systems.^{17–18} We have conducted a study on Lebanese children, and found out that any exposure to pesticides was associated with asthma and chronic respiratory symptoms (chronic phlegm, chronic wheezing, ever wheezing).¹⁹ Thus conducting research regarding pesticides and their relation with asthma in adults is necessary, especially in the Lebanese context.

The objective of this study is to evaluate the odds of occupational, domestic, and environmental exposures to pesticides in adult asthmatic patients in Lebanon.

METHODS

Study design

We conducted a case-control study, comparing pesticide exposure in cases of asthma, in comparison with controls without respiratory problems. The study protocol was approved by an independent review board from the Lebanese Council for Scientific Research, permission number 3750/o.

Population of the study

Cases and controls were recruited in Lebanese hospital centres, chosen from the list of hospitals according to convenience in all Lebanese regions (Beyrouth, Mount Lebanon, North Lebanon, South Lebanon, and Bekaa).

Procedure

One pulmonologist was contacted within every hospital. After they agreed to contribute to the study, they were asked to recruit cases of newly diagnosed asthma, and equal numbers of controls from other divisions. Asthma was defined according to current guidelines for clinical definition of asthma: recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning.²⁰ Any other chronic pulmonary pathology diagnosed by pulmonologists was an exclusion criterion (tuberculosis, fibrosis, cancer, etc). Chronic bronchitis patients were included in the study, but not in this analysis (article in press). Age categories between 12 and 99 years were included in the study.

Controls were taken in a source population of persons accompanying cases (parents, friends) or seeking advice for another problem in the same hospital: gastroenterology, endocrinology, orthopaedics, cardiology, surgery, neurology, nephrology, and urology. Any chronic respiratory symptom or problem reported by controls before filling in the questionnaire was an exclusion criterion.

Subjects were not aware of the exact objective of the study; information they were given consisted of "a questionnaire regarding their health, the results of which will be useful for science", and an oral consent to give the information was required, to respect the ethical issue regarding medical information use in research.

Questionnaire and variables

A pretested self administered questionnaire,²¹ adapted to local Arabic language from the standardised and validated American Thoracic Society chronic respiratory disease questionnaire,²² was given to pulmonologists to administer to any subject fulfilling inclusion criteria. Questionnaires were delivered to eligible subjects by local inquirers, independently of exposure status. The same conditions were applied for questionnaires in both cases and controls, to evaluate the diagnosis of asthma and respiratory symptoms, in addition to occupational, regional, local, and domestic pesticides exposures.

Reported symptoms in the self administered questionnaire permitted the study of the declared asthma issue. This primary diagnosis of declared asthma was defined as a recurrent wheezing during the day, evening, night, the whole day or at exercise, a recurrent cough during the day, evening, night, the whole day or at exercise, a history of more than one dyspnea plus wheezing episode treated by a doctor, or a history of asthma previously confirmed by a doctor. The primary diagnosis had then to be reconfirmed by an independent pulmonologist, by re-examining the answers to the questionnaire.

The presence of symptoms was indicated by affirmative responses to the questions, "Have you noticed any wheezing in the chest when you do not have a cold?" "Have you had an episode of dyspnea and wheezing?" "Do you usually have cough when you do not have a cold?"

For pesticide exposure, questions were the following ones: "Have you ever used pesticides in your work?" "Have you ever used pesticides out of your work (for house or garden treatment...)" "Do you live in a region heavily treated by pesticides?" "Do you live in the proximity of a heavily treated field by pesticides?"... An expert in agronomy, who assigned to residency regions a score from 0 to 9 according to yearly consumption of pesticides, provided complementary evaluation of regional exposure. Personal occupational pesticide exposure, occupational use by a family member, regional exposure because of residence in a heavily treated region (score > 4), local exposure by proximity to a heavily treated field, non-occupational exposure by personal house or garden treatment, and indirect exposure by house treatment by someone else, were evaluated. A variable named "ever exposed" is considered positive if the subject declared any exposure type.

Cumulated exposure index were also calculated: for regional exposure, the product of the region's score by the number of years of residence in that region was calculated; for other uses, the product of the number of yearly uses by the number of years of use was calculated when applicable. A unique general exposure index could not be calculated as single cumulative exposures are measured by different units (lifetime number of applications and score×years).

Active smoking was determined by several questions, categorising subjects in non-smokers (smoking history of less than 20 packs in a lifetime),²² current smokers, and former smokers. The number of pack years was determined by multiplying the average number of packs of 20 cigarettes smoked per day by the number of years of smoking. Passive smoking was characterised by the number of smokers at home.

Table 1 Social and demographic characteristics

Variable	Controls (n = 262) (%)	Cases (n = 245) (%)	p Value
Department			0.09
Beirut	34 (13.0)	37 (15.1)	
Mount Lebanon	149 (57.1)	106 (43.3)	
South	11 (4.2)	26 (10.6)	
North	47 (18.0)	54 (22.0)	
Bekaa	20 (7.7)	22 (9.0)	
Age (y) mean (SD)	37.6 (14.9)	36.2 (17.4)	0.32
Sex distribution			0.05
Male	116 (44.3)	129 (52.9)	
Female	146 (55.7)	192 (47.1)	
Education level			<10 ⁻⁶
<9 years at school	41 (16.0)	76 (31.4)	
9–15 years at school	95 (37.0)	115 (46.5)	
College graduate	121 (47.1)	51 (21.1)	
Nationality			0.04
Non-Lebanese	2 (0.8)	8 (3.3)	
Lebanese	259 (99.2)	237 (96.7)	
BMI mean (SD)	24.4 (3.8)	25.0 (4.3)	0.13

BMI, body mass index, calculated by BMI=weight in kg/(height in metres)².

Table 2 Smoking status, toxic exposure, personal disease, and family history

Variable	Controls (n = 262) (%)	Cases (n = 245) (%)	p Value
Smoking status			0.08
Non-smoker	175 (67.1)	157 (64.1)	
Previous smoker	34 (13.0)	49 (20.0)	
Actual smoker	52 (19.9)	39 (15.9)	
Pack years (SD)	14.1 (13.3)	27.9 (57.0)	0.16
Smokers at home			0.12
No smoker	123 (47.3)	92 (38.2)	
One smoker	59 (22.7)	62 (25.7)	
More than one	78 (30.0)	87 (36.1)	
Works in a toxic gases environment	4 (1.5)	20 (8.3)	<10 ⁻⁴
Works in a dusty environment	24 (9.2)	54 (22.3)	<10 ⁻⁴
Chest trauma	7 (2.7)	10 (4.1)	0.37
Cardiac disease	10 (3.8)	11 (4.5)	0.70
Paternal respiratory problem	9 (3.4)	21 (8.6)	0.014
Maternal respiratory problem	21 (8.0)	23 (9.4)	0.58

Sample size calculation

Sample size calculation was done with an α risk of 5%, a β risk of 20%, and a minimal exposure probability of 20%, representing the percentage of Lebanese residents working in the agricultural sector.²³ The minimal sample size necessary to show a twofold increase in risk consisted of 372 subjects, divided into 186 cases and 186 controls.

Statistical analysis

Data analysis was performed on SPSS software, version 12. Two sided statistical tests were used; χ^2 test for dichotomous or multinomial qualitative variables, Wilcoxon test for quantitative variables with non-homogeneous variances or non-normal distribution, and Student's *t* test for quantitative variables of normal distribution and homogeneous variances.

Regarding multivariate analysis, an ascending stepwise logistic regression was performed, and adjustment over potential confounding variables was done: residency department, number of pack years, number of smokers at home, sex, age, body mass index, nationality, education, working in dusty and toxic fumes environments, cardiac and parental respiratory problem history were included in the analysis.

A Mantel-Haenszel secondary analysis was performed to account for the effect of education level, the surrogate measure for socioeconomic status, on the effect of pesticides exposure over asthma.

RESULTS

In 10 Lebanese hospital centres, 10 pulmonologists participated from several areas of the country (three at Beirut, four in Mount Lebanon, one in Northern Lebanon, one in the Bekaa, and one in Southern Lebanon), with the average ratio of one pneumonologist/4×10⁵ Lebanese inhabitants.

Altogether 790 questionnaires (53%) were filled in from 1500 distributed; 33 were then eliminated because of inadequate completion; 757 were accepted (50%).

Altogether 140 (27.8%) subjects reported symptoms not in accordance with medical diagnosis and presented unclassifiable respiratory problems, and 110 (7.3%) presented a chronic bronchitis: these categories were not included in the analysis. A total of 507 questionnaires were finally analysed.

Results are presented for these asthma patients 245 (48.3%) and 262 controls (51.7%).

Social and demographic characteristics

Table 1 summarises social and demographic characteristics. There were significant differences regarding sex, education, and nationality between cases of asthma and controls ($p < 0.05$). Male sex, lower educational level, and non-Lebanese nationality were more common in cases than in controls (table 1).

Smoking, work environment, medical and familial history

There were no differences regarding smoking history of cases and controls. However, more cases declared working in dusty and toxic fumes environments. In addition, a paternal respiratory history was significantly more prevalent in cases than in controls ($p < 0.05$) (table 2).

Exposure to pesticides and asthma

Table 3 reports the types of pesticides exposure. All associations were statistically significant, except for occupational use of pesticides by a family member. Asthmatic patients had higher odds of being exposed to pesticides

Table 3 Pesticides exposure types and asthma in Lebanon

Exposure type	Controls (n = 262) (%)	Cases (n = 245) (%)	p Value; OR (95%CI)
Any exposure	123 (47.3)	159 (65.4)	<10 ⁻⁴ ; 2.11 (1.47 to 3.02)
Occupational exposure	2 (0.8)	9 (3.7)	0.02; 4.98 (1.07 to 23.28)
Non-occupational exposure*	68 (26.0)	82 (33.6)	0.06; 1.44 (0.98 to 2.12)
House exposure†	73 (27.9)	98 (40.3)	0.003; 1.75 [1.21 to 2.54]
Regional exposure‡	24 (9.2)	64 (26.2)	<10 ⁻⁴ ; 3.51 (2.11 to 5.83)
Local exposure§	34 (13.0)	69 (28.3)	<10 ⁻⁴ ; 2.63 (1.67 to 4.15)
Paracoccupational exposure¶	9 (3.4)	12 (4.9)	0.40; 1.45 (0.60 to 3.51)

* Personal use of pesticides; †house treated by pesticides; ‡lives in a region heavily treated by pesticides; §lives near a field heavily treated by pesticides; ¶has a family member occupationally exposed to pesticides.

Table 4 Pesticides exposure types and asthma in Lebanon: multivariate analysis

Exposure type	p Value	Adjusted OR (95%CI)	Significant factors retained in the analysis
Any exposure	$<10^{-4}$	2.32 (1.47 to 3.68)	Age, education, nationality, paternal lung problem, working in dust or toxic fumes
Occupational exposure	0.056	5.68 (0.96 to 33.33)	Age, education, nationality, paternal lung problem, working in dust or toxic fumes
Non-occupational exposure*	0.005	2.28 (1.28 to 4.08)	Age, education, nationality, paternal lung problem, working in dust or toxic fumes
House exposure†	$<10^{-4}$	2.59 (1.58 to 4.26)	Age, education, nationality, paternal lung problem, working in dust or toxic fumes
Regional exposure‡	0.002	2.91 (1.48 to 5.71)	Age, education, nationality, paternal lung problem, working in dust or toxic fumes
Local exposure§	0.047	1.83 (1.01 to 3.31)	Age, education, nationality, paternal lung problem, working in dust or toxic fumes
Multiple exposure model			
Occupational exposure	0.21	2.86 (0.56 to 14.28)	Age, education, nationality, working in dust or toxic fumes
Non-occupational exposure*	0.25	1.35 (0.81 to 2.27)	
House exposure†	10^{-3}	2.17 (1.37 to 3.50)	
Regional exposure‡	0.02	2.78 (1.18 to 6.67)	
Local exposure§	0.93	1.04 (0.45 to 2.37)	

*Personal use of pesticides; †house treated by pesticides; ‡lives in a region heavily treated by pesticides; §lives near a field heavily treated by pesticides.

(OR = 2.11 (1.47 to 3.02); $p < 10^{-4}$). Occupational use presented the highest association (OR = 4.98 (1.07 to 23.28); $p = 0.02$), followed by regional exposure (OR = 3.51 (2.11 to 5.83); $p < 10^{-4}$) and by living near a field treated by pesticides (OR = 2.63 (1.67 to 4.15); $p < 10^{-4}$). House treatment (OR = 1.75 (1.21 to 2.54); $p = 0.003$) was also declared by asthmatic patients and personal exposure from non-occupational use (OR = 1.44 (0.98–2.12); $p = 0.06$) gave borderline results, while paraoccupational exposure did not seem to be more common in asthmatic patients.

Multivariate analysis

Stepwise ascending logistic regression was performed. Adjusted ORs still show significant association of exposure to pesticides with asthma. Any exposure was associated with asthma, $OR_a = 2.32$ (1.47 to 3.68) (table 4): occupational use ($OR_a = 5.68$), regional exposure ($OR_a = 2.91$), house treatment ($OR_a = 2.59$), non-occupational use ($OR_a = 2.28$), and local exposure ($OR_a = 1.83$) were also all associated with asthma.

Taking all types of exposure in the same model, the following results were obtained: professional use ($OR_a = 2.86$; $p = 0.21$), personal non-professional use ($OR_a = 1.35$; $p = 0.25$), regional exposure ($OR_a = 2.78$; $p = 0.02$), local exposure ($OR_a = 1.04$; $p = 0.93$), and house exposure ($OR_a = 2.17$; $p = 0.001$).

In the Mantel-Haenszel analysis, education level showed an interaction on the effect of pesticides exposure over asthma. In the three subsequent categories of education, pesticides exposure was associated with asthma, with a lower effect in the low education category that could not reach significance: $OR_1 = 1.45$ ($p = 0.34$), $OR_2 = 2.60$ ($p = 0.001$), and $OR_3 = 2.21$ ($p = 0.02$). The Mantel-Haenszel common OR estimate was 2.15 (1.52 to 3.11); $p < 10^{-4}$. Moreover, the low education level subjects smoked more than others ($p = 0.002$).

Quantitative analysis

Analysis of cumulative exposure index correlation to asthma, gave also statistically significant results (table 5). Asthmatic patients had higher cumulative exposure: $p < 0.05$ for any exposure type, except for occupational use that did not reach statistical significance.

DISCUSSION

In this Lebanese case-control study on asthma, asthmatic patients had higher odds of exposures to pesticides. Dose effects relation and multivariate analysis confirmed the results obtained in bivariate analysis, particularly professional, regional, and house exposure. The relative imprecise estimates and borderline significance for the adjusted OR of occupational exposure association to asthma is probably attributable to the small number of people in that category. The same applies for cumulative exposure of occupational exposure and house treatment.

Despite an overall visible effect of pesticides on asthma, the lower education category subjects seemed not to present this association, and their presence in the study diluted the global

What this paper adds

- Pesticides are toxic substances known to cause professional diseases, including asthma; little is known about the effect of non-professional exposure
- Adult asthmatic patients in Lebanon have higher odds of being professionally and non-professionally exposed to pesticides
- Pesticides seem to cause respiratory diseases, particularly asthma, in non-professional settings

Table 5 Mean cumulative exposure to pesticides and asthma in Lebanon

Cumulative exposure mean (SEM)	Controls (n = 262)	Cases (n = 245)	p Value
Occupational exposure	0 applications	1.31 (1.19) applications	0.25
Non-occupational exposure†	1.10 (0.32) applications	6.00 (2.06) applications	0.02
House exposure‡	10.61 (1.17) applications	14.15 (1.32) applications	0.045
Cumulative regional exposure§	87.45 (7.64) years	152.51 (23.95) years	0.01
Local exposure¶	2.19 (0.53) years	5.73 (0.94) years	$<10^{-4}$

†Personal use of pesticides: lifetime number of applications; ‡house treated by pesticides: lifetime number of house applications; §region score × duration of living in that region; ¶duration of living near a field heavily treated by pesticides.

Policy implications

- Cases of asthma can be prevented with adequate preventive measures, such as eviction of exposure to pesticides
- Policies to educate the general population about the risks of pesticides should be implemented in Lebanon
- Pest control with non-pesticides used should also be proposed (integrated pest management, biological means, etc)

results. The possible reasons for this finding may be that people with low socioeconomic status may live in environmental conditions other than pesticides exposure that may affect their asthma, or may have behaviour such as smoking different from higher education level. In fact, in this study, lower education persons smoked more, and smoking is known to induce hepatic enzymes, and by that to decrease xenobiotics toxicity, pesticides in particular.²⁴ This is why multivariate analysis was necessary. However, residual confounding attributable to socioeconomic status is still possible, as education level may not completely account for socioeconomic status. This issue remains to be explained with more specific studies. On the other hand, environmental allergens other than pesticides and passive smoking at work were difficult to assess in this study, and may thus also account for residual confounding.

It was not possible to get detailed information on non-responders. However, as subjects did not know the exact objective of the study, answer refusals are expected to be mainly because of non-motivation or illiteracy, but not because of exposure status: all other factors being equivalent, exposed and unexposed people have the same probability of responding. Persons with lower education level have generally lower probability of responding, and they also have higher odds of regional and local exposure to pesticides (results not shown). This may introduce a selection bias of more educated persons, but we do not expect this bias to be differential. Pulmonologists recruiting subjects were also unaware of their exposure status, especially when considering various types of exposure (regional, occupational, domestic). This may decrease selection bias, which however cannot be excluded.

We cannot exclude the fact that cases are likely to present different degrees of diagnosis. On the other hand, controls had mixed non-respiratory diagnosis and are non-comparable to the general population; this could induce an under-estimation of the expected associations.

The geographical comparability of cases and controls and the fact that family members were sometimes taken as controls may cause them to have similar non-occupational exposures to pesticides or common familial allergies. This may further bias the results towards the null. Recall and subjectivity bias may also be possible because information on some types of exposure (except regional) was based on self reporting without further evaluation, but it is most probably a non-differential one between the two groups of comparison, as the subjects were not aware of the study objectives.

Several epidemiological and toxicological studies have shown results comparable to those of this study. A cross-sectional study was conducted in Canada on 1939 farmers for carbamates and their respiratory effects: carbamates use was associated with isolated asthma and pulmonary function change; prevalence odds ratio was 1.8 for asthma ($p = 0.02$) and there was a decrease in farmers' respiratory functions

($p < 0.001$).²⁵ In the Iowa farm family health and hazard surveillance project, among farmers, applying pesticides to livestock was associated with significantly increased odds of phlegm (OR = 1.91; 95% CI 1.20 to 3.57), chest ever wheezy (OR = 3.92, 95% CI 1.76 to 8.72), and flu-like symptoms (OR = 2.93, 95% CI 1.69 to 5.12) in models adjusting for age and smoking.²⁶ Hoppin and colleagues examined chemical use in the past year and self reports of wheeze in the past year among the agricultural health study (AHS) cohort. By using a large, heterogeneous cohort, they were able to control for other asthma related risk factors and to explore the associations of individual pesticides with wheeze. They found an independent role for specific pesticides in respiratory symptoms of farmers.²⁷

On the other hand, in Durban, South Africa, an association between asthma among adults and regional use of insecticides was found.²⁸ In addition, the results of our study are comparable to those found in Lebanese children, where asthma was associated with any exposure (OR_a = 1.73; $p < 0.01$), regional or local exposure (OR_a = 2.10; $p < 0.05$), and domestic exposure (OR_a = 1.99; $p < 0.05$). The only notable difference is that regarding para-occupational exposure: children of pesticide professional users have more asthma than other children (OR_a = 4.61, $p < 0.001$),¹⁹ while this association type was not found in adults. Possible explanations for this would be the inverse correlation that exists between allergy expression and age,²⁹ or another concomitant exposure (regional for example) in children not seen in adults. This issue remains to be established by other trials.

Biological plausibility is confirmed by multiple experimental toxicological studies on animals, in addition to known clinical effects of high level exposure. Effects on the respiratory system, linked to the local and systemic toxic actions (especially immune, allergic, and neurological) have sometimes been studied. Most of pesticides being small molecules, they can exacerbate symptoms of atopic patients, asthma, and contact dermatitis by hapten formation.³⁰ Besides type I and IV hypersensitivity, the development of the respiratory problem can be attributable an oxidative stress that may cause detoxification or reparation capacity overwhelming of cells.⁹ Pesticides can cause laryngeal and bronchial constriction.³¹ Specifically, organophosphates and organochlorates have anticholinesterase activity and are known to cause asthma episodes.³² Furthermore, some data showed that organophosphate insecticides can cause airway hyperactivity in the absence of AChE inhibition by decreasing neuronal M2 receptor function.³³ Pyrethroid derivatives are also allergens, causing asthma-like episodes.¹⁵

In conclusion, pesticide exposure was more common in asthmatic adults, compared with non-asthmatic controls, particularly occupational, regional, and house exposure. The observed effects cannot be related to a specific pesticide as several products are used concomitantly or vary with time, season, crops type, and personal preference. Typical pesticides' use evaluation and monitoring of different pesticides and other pollutants in the air would be necessary in future research to eliminate the effects of residual biases and confirm our results.

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