Original Article

MEDICINE PALLIATIVE CARE

DOI: 10.47582/jompac.1649428

J Med Palliat Care. 2025;6(3):177-182

Assessment of demographic characteristics and non-occupational exposures in occupational asthma: a single-center experience

Bilge Akgündüz[1], Muhittin Akarsu[2]

[1]*Department of Occupational Diseases, Eskişehir City Hospital, Eskişehir, Turkiye*

[2]*Department of Allergy and Immunology, Faculty of Medicine, Ankara University, Ankara, Turkiye*

Cite this article as: Akgündüz B, Akarsu M. Assessment of demographic characteristics and non-occupational exposures in occupational asthma: a single-center experience. *J Med Palliat Care*. 2025;6(3):177-182.

Received: 01.03.2025 ◆ **Accepted**: 26.03.2025 ◆ **Published**: 18.06.2025

ABSTRACT

Aims: This study aimed to evaluate differences in demographic characteristics, occupational, and non-occupational exposures (NOE) between patients diagnosed with occupational asthma (OA) and non-occupational asthma (NOA).

Methods: A total of 114 patients with suspected work-related asthma were evaluated, and 82 asthma-diagnosed patients were included in the study.

Results: Among the 82 patients, 29 (35.4%) were diagnosed with OA. Seventy-two (87.8%) asthma patients were exposed to low molecular weight agents. The most common sector was ceramics (OA group n: 6 [7.3%]; NOA group n: 6 [7.3%]). The NOA group had a higher likelihood of being exposed to non-occupational agents that could trigger asthma. A higher frequency of pet bird ownership (OA group n: 4 [4.9%]; NOA group n: 12 [14.6%]) and current humidity or moisture exposure at home (OA group n: 3 [3.7%]; NOA group n: 7 [8.5%]) was observed in the NOA group. A negative correlation was found between the duration of asthma symptoms and both FEV1 and the FEV1/FVC ratio in OA patients with a history of COVID-19. Additionally, total IgE levels were significantly higher in immunological asthmatics with OA compared to those with NOA. Among OA patients exposed to NOE, FEV1 levels were 1.33 times higher in those without NOE.

Conclusion: NOE that may cause asthma can coexist with occupational exposures in OA cases. A comprehensive history, including environmental, indoor, and individual risk factors, as well as previous COVID-19 infection, is crucial for accurately identifying multiple asthma-causing agents and improving disease management by eliminating triggers.

Keywords: Non-occupational exposures, sector, occupational asthma, occupational exposures

INTRODUCTION

Occupational exposures are responsible for 15-25% of asthma cases in adults.[1] A detailed history of occupational exposure during patient examination is essential to prevent misdiagnosis of work-related asthma.[2] Work-related asthma is classified into two categories: occupational asthma (OA), which is directly caused by occupational exposures, and work-exacerbated asthma (WEA), which occurs when asthma, previously under control, is triggered by workplace-related exposures.[3,4] The prevalence of WEA in adults is 21.5%, while the prevalence of OA is 16%.[5,6]

OA-causing exposures are generally classified into agents of high molecular weight (HMW) and low molecular weight (LMW).[1,7,8] In 2018, a new OA-specific occupational exposure matrix (OAsJEM) was developed by Moual et al.[9,10] which added eight additional exposures to known irritants and sensitizers that directly cause OA. Specific irritants such as household cleaners, pesticides, endotoxins, aliphatic amines, acrylates, epoxy resins, persulfates/henna, and organic solvents were incorporated into subgroups in the updated OAsJEM.[10] Although certain agents known to cause asthma in the workplace have been identified, the effects of exposures such as humidity or animal proteins—known to trigger non-occupational asthma (NOA)—are not fully understood when they coexist with occupational exposures. The role of non-occupational exposures (NOE) in OA remains unclear, and research on this subject is limited.

In this study, our primary objective was to investigate whether there were differences in demographics, asthma symptom duration, smoking habits, occupational and NOE, exposure duration, total IgE levels, and pulmonary function test (PFT) results between patients diagnosed with OA and NOA. The secondary aim was to explore the coexistence of NOE agents with occupational exposures.

Corresponding Author: Bilge Akgündüz, bilgeuzmezoglu@hotmail.com

METHODS

Ethics

The study protocol was approved by the Non-interventional Clinical Researches Ethics Committee of Eskişehir City Hospital (Date: 18.01.2023, Decision No: ESH/GOEK 2022/19SK). This study was conducted in accordance with the principles outlined in the Declaration of Helsinki.

Study Population

This retrospective descriptive cross-sectional study was conducted between January 2021 and January 2022. A total of 114 patients with suspected OA who were admitted or referred to the Occupational Diseases Clinic at Eskişehir City Hospital were evaluated. As a retrospective study, the research did not require direct informed consent from participants. Patient data was anonymized and handled in strict accordance with ethical guidelines to protect the privacy and confidentiality of all participants.

Evaluations included demographics, symptoms, duration of asthma symptoms, host history, occupational and NOE, duration of exposures, smoking habits, lung function test results, laboratory and radiological tests, reversibility tests, and peak expiratory flow (PEF) measurements.

Eighty two patients diagnosed with asthma were included in the study. Asthma patients were divided into OA and NOA groups (**Figure**).

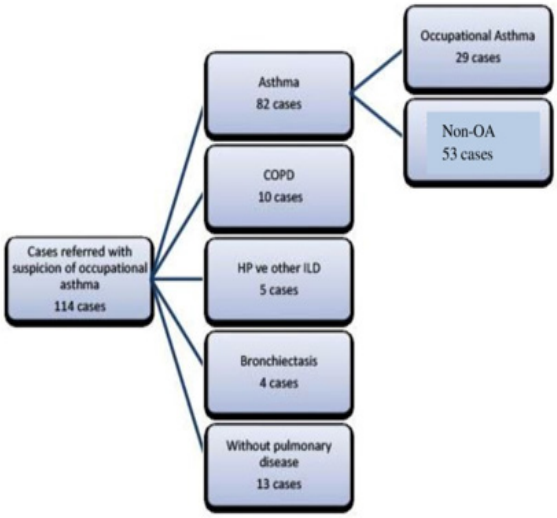

Figure. 114 cases with differential diagnosis
COPD: Chronic obstructive pulmonary disease, HP: *Helicobacter pylori*, OA: Occupational asthma

Diagnosis of Asthma

Asthma was diagnosed in patients with one or more respiratory symptoms, including shortness of breath related to work or non-work related factors, cough, wheezing, chest fullness, air hunger, and a positive reversibility on the PFT or a positive nonspecific bronchial provocation test (NSBPT). In cases where patients exhibited symptoms but negative early or late reversibility, a positive result from the PEF meter in follow-up testing, with a forced expiratory volume in the first second/forced vital capacity (FEV_1/FVC) ratio of less than 70%, was used to confirm asthma diagnosis.[11]

One patient underwent a specific bronchial provocation test (SBPT) at a outpatient university hospital. SBPT was performed when workers were suspected to have OA due to exposure to specific agents, mimicking actual workplace exposures under clinically monitored conditions. A positive SBPT was defined by a significant decrease in FEV1 after exposure, indicating an asthmatic response.[1,12]

Groups Definition

Occupational asthma: Diagnosed by a positive SBPT in patients who had no prior respiratory symptoms or known history of asthma, but developed work-related respiratory symptoms and/or signs after workplace exposure, or by positive variability of the PEF at work versus away from work.

Non-occupational asthma: Included patients with asthma who did not meet the criteria for OA.

Specific Bronchial Provocation Test (SBPT)

The SBPT aims to simulate workplace exposure to a suspected causative agent of asthma.[1,12] SBPT was performed on a patient exposed to isocyanates, which was later confirmed as OA with a positive SBPT result. Only one patient underwent SBPT at a university hospital.

PEF Meter Monitoring and Method

A PEF meter was used to monitor the variability of PEF. The test was considered positive if PEF variability was detected on more than two-thirds of the total measurement days, with at least four PEF measurements per day over a period of at least five weeks, including three weeks at work and two weeks without work interruption.[13,14] PEF measurements were made using a mini peak flow meter, known for its reliability and precision in asthma management. The formula for PEF variability calculation was as follows:

PEF variability %=(PEF-lowest PEF)/(2x[(PEF+lowest PEF)/2])x100. This formula was used to assess fluctuations in PEF over time.[13,14]

Non-Occupational Exposure (NOE)

The occupational history of lung disease, diagnosis, and follow-up form was used in the clinic to document both occupational and NOE. In this region, keeping pet birds (budgerigar, pigeon, canary, cockatoo-parrot), pigeon cultivation, and environmental asbestos exposure are common. Detailed records were kept regarding smoking habits, pet bird ownership, pigeon cultivation, and other NOE such as environmental mould, humidity, and hobbies related to asthma development (e.g., animal husbandry, farming, painting). Additionally, history of allergy, family allergy, and COVID-19 were also recorded. NOE was defined within this context.

Immunologic Occupational Asthma (Immunologic OA)

Cases with total IgE levels greater than 100 U/ml were classified as immunologic OA.[15]

Exclusion Criteria

Patients with respiratory symptoms and/or signs that did not meet asthma diagnostic criteria (e.g., chronic

obstructive pulmonary disease with irreversible airway obstruction on respiratory function tests), patients with asthma-related symptoms but radiologic findings consistent with hypersensitivity pneumonitis or other interstitial lung diseases, and patients without work-related complaints or abnormalities on pulmonary function or bronchial provocation tests were excluded from the study.

Statistical Analysis

Data analysis was conducted using the SPSS V22 software (SPSS Inc., Chicago, IL, USA). The frequencies and percentages of categorical variables and the mean, median, and standard deviation of numerical variables were calculated. The t-test was used for normally distributed numerical variables, while categorical variables were analyzed using the Chi-square test. Nonparametric tests were applied for variables without a normal distribution. The Pearson correlation test was used for correlation analysis. A p-value of <0.05 was considered statistically significant.

RESULTS

OA was diagnosed in 29 patients (35.4%) among the 82 asthma patients evaluated. The average age of the participants was 40.8±7.5 years, with a predominance of male patients (75.6%, n=62). Dyspnea was the most common symptom (n=64). The mean duration of asthma symptoms was 3.6±3.8 years. More than half of the patients were smokers (54.9%, n=45), with an average of 5 pack-years (8.6±8.6 cigarette pack-years in total). No statistically significant differences were found in terms of demographic and clinical characteristics between OA and NOA patients, suggesting that differentiating between OA and NOA based solely on these factors is challenging (Table 1).

Occupational and Non-Occupational Exposures

When examining occupational exposures, several industries were associated with both OA and NOA. The ceramic industry had an equal distribution of OA and NOA cases, with 7.3% of both groups originating from this sector, totaling 14.6%. Similarly, food manufacturing and painting industries had relatively higher proportions of OA, each contributing 11.0% of the total cases. The metal industry also represented a significant portion of the cases, with metalworking comprising 9.8% and welding 8.5%.

In contrast, NOE played a significant role in the NOA group. The NOA patients had a notably higher frequency of exposure to NOA triggers. For example, 12 (14.6%) patients in the NOA group reported exposure to humidity or mould in their homes, compared to only 3 (3.7%) in the OA group. Additionally, 12 (14.6%) patients in the NOA group kept pet birds at home, while only 4 (4.9%) patients in the OA group had this exposure. This highlights the importance of environmental and domestic factors as potential triggers for asthma in the NOA group, which can overlap with occupational exposures in patients diagnosed with OA (Table 2).

Specific IgE positivity results have been added. RAST was performed in 33 cases. In the OA group, six cases had RAST positivity for latex, inhalant allergens (two cases), grass pollen mix, mold mix, and tree mix. In the NOA group, specific IgE positivity was detected for bee venom, *Aspergillus*, mold mix, weed mix, *Dermatophagoides*, inhalant allergens, and budgerigar dander.

Duration of Symptoms, Work Duration, and Immunological Findings

We found significant correlations between the duration of symptoms, work exposure duration, and levels of Total IgE with clinical factors. Specifically, there was a relationship between the duration of asthma symptoms and both FEV_1 and FEV_1/FVC ratios in patients with OA (p=0.05 and p=0.001), showing a decline in lung function as symptoms persisted over time. Furthermore, a longer duration of dyspnea was observed in patients with OA who had prolonged exposure to work-related allergens (p=0.007), indicating that work-related exposures might exacerbate asthma symptoms over time (Table 3).

Importantly, a positive relationship was found between the duration of symptoms and total IgE levels in both OA (p=0.012) and NOA (p=0.019) patients, emphasizing the role of immune responses in the progression of asthma. Moreover, the NOE were linked to higher IgE levels, further suggesting that these environmental factors contribute to asthma exacerbation in both OA and NOA patients (Table 3).

DISCUSSION

In our study, we observed that individuals diagnosed with OA, when exposed to NOE agents, experienced a significant decrease in FEV_1 levels compared to those diagnosed with NOA. A thorough evaluation of environmental exposures was conducted through detailed patient histories, environmental surveys, and tracking of exposures to both occupational and non-occupational agents. This comprehensive approach highlights the necessity of considering both environmental and occupational exposures when diagnosing asthma, even in patients with a history of occupational exposure. However, due to the limited sample size, our findings should be interpreted with caution, and further research with larger sample sizes is needed to clarify the complex relationships among environmental, occupational, and individual factors in asthma pathogenesis.

The importance of obtaining a detailed environmental exposure history was reinforced by our findings. Structured questionnaires that address both occupational and NOE should be utilized to improve diagnostic accuracy and treatment efficacy. These should include inquiries about home environments, hobbies, and lifestyle factors that could contribute to asthma-related exposures. Additionally, family history should be considered, particularly in relation to any environmental changes that coincide with symptom onset or worsening. Incorporating these detailed investigations into routine clinical evaluations can enhance diagnostic precision and improve the effectiveness of subsequent treatments, including those targeting removal from exposures in OA.[16,17]

The increasing diversity of non-occupational respiratory environmental agents complicates the establishment of a direct causal relationship between asthma and exposure in occupational diseases. The complex pathogenesis of asthma, variability in individual responses to allergens, and the

Table 1. Comparison of demographics, symptoms, exposures, smoking habits, allergy status, and pulmonary function test parameters in OA and NOA cases

Parameters	OA (n: 29)	NOA (n: 53)	Total (n: 82)	p
Age (mean±SD)	40.7±7.7	40.9±7.4	40.8±7.5	0.933
Gender (n, %)				
Male	21 (25.6)	41 (50.0)	62 (75.6)	0.404
Female	8 (9.8)	12 (14.6)	20 (24.4)	
Symptoms (n, %)				
Cough	6 (7.4)	7 (8.6)	13 (16.0)	0.602
Dyspnea	13 (16.0)	31 (38.3)	44 (54.3)	
Cough & dyspnea	8 (9.9)	12 (14.8)	20 (24.4)	
Duration of asthma symptoms (mean±SD)	3.7±4.4	3.4±3.6	3.6±3.8	0.762
Package-year (mean±SD)	8.7±8.4	8.5±8.7	8.6±8.6	0.765
Smoking habits (n, %)				
Nonsmoker	8 (9.8)	19 (23.2)	27 (32.9)	0.309
Current smoker	19 (23.2)	26 (31.7)	45 (54.9)	
Ex-smoker	2 (2.4)	8 (9.8)	10 (12.2)	
Non-occupational exposures and host history				
Present history of COVID-19 (n, %)	8 (9.8)	17 (20.7)	25 (30.5)	
Present COVID-19 vaccine (n, %)	24 (29.3)	43 (52.4)	67 (81.7)	0.758
Pet bird at home	4 (4.9)	12 (14.6)	16 (19.5)	0.323
Present history of humidity or moisture (n, %)	3 (3.7)	7 (8.5)	10 (12.2)	0.702
Present history of pet animal at home (n, %)	1 (1.2)	2 (2.4)	3 (3.7)	-
Present history of farming (n, %)	2 (2.4)	4 (4.9)	6 (7.3)	-
Present history of pneumonia	1 (1.2)	1 (1.2)	2 (2.4)	-
Hobbies (painting, pigeon cultivation and wood working) (n, %)	0	3 (3.7)	3 (3.7)	-
Present history of allergy (n, %)	5 (6.1)	8 (9.8)	13 (15.9)	0.800
Occupational exposures (n, %)				
HMW	2 (2.4)	7 (8.5)	9 (11.0)	0.283
LMW	26 (31.7)	46 (56.1)	72 (87.8)	
Mix	1 (1.2)	0 (0.0)	1 (1.2)	
Present peripheral eosinophilia	4 (4.9)	2 (2.4)	6 (7.4)	0.269
Present total IgE ≥100 U/ml (n, %)	7 (8.8)	18 (22.5)	25 (31.3)	0.329
Total IgE, U/ml, (mean±SD)	190.3±614.8	181.1±380.9	184±475.2	0.935
Present spesifik IgE positive (n: 33)	6 (18.2)	9 (27.3)	15 (45.5)	0.614
Pulmonary function test findings				
Present FEV_1/FVC<%70, (n, %)	12 (14.6)	21 (25.6)	33 (40.2)	0.877
FEV_1,L (mean±SD)	2.9±0.9	3.1±0.9	3.0±0.9	0.253
FEV_1% (mean±SD)	86.1±21.2	89.5±20.8	88.3±20.9	0.484
FEV1/FVC% (mean±SD)	75.9±7.9	74.9±8.7	75.3±8.4	0.623

OA: Occupational asthma, NOA: Non-occupational asthma, SD: Standard deviation, IU: International unit, L: Liters, HMW: High molecular weight, LMW: Low molecular weight, IgE: Immunoglobulin E, FEV_1: Forced expiratory volume in the first second, FVC: Forced vital capacity, statistical significance p<0.05

diversity of agents that trigger asthma make distinguishing OA challenging.[18,19] Factors such as clinicians' neglect of occupational exposure histories, failure to assess the relationship between symptoms and work, and the use of multiple diagnostic tests can delay the diagnosis of OA. A study at the Ontario Occupational Lung Disease Clinic found that the average time to diagnosis was over 3 years.[20] Similarly, our study found that the average diagnostic delay was 3.7 years. This underlines the importance of symptom screening questionnaires in workplaces to evaluate symptoms following exposure, which may facilitate earlier diagnosis of OA.

In our investigation, NOE, such as home humidity, mold, and the feeding of domestic birds, were also considered as potential contributors to asthma. While no significant differences in NOE were observed between OA and NOA patients, those with NOE were more likely to be diagnosed with asthma. A survey by Rollins et al.[21] demonstrated that home renovations

Table 2. Distribution of OA and NOA cases according to their respective work industries

Sectors or jobs	OA n (%)	NOA n (%)	Total n (%)
Duration of occupational exposure, year, (mean±SD)	7.9±6.1	10.4±8.8	9.5±8.0
Metal	2 (2.4)	6 (7.3)	8 (9.8)
Ceramic	6 (7.3)	6 (7.3)	12 (14.6)
Food	3 (3.7)	6 (7.3)	9 (11.0)
Non-domestic cleaners	3 (3.7)	5 (6.1)	8 (9.8)
Manufacture	2 (2.4)	3 (3.7)	5 (6.1)
Animal husbandry	0 (0.0)	1 (1.2)	1 (1.2)
Painter	5 (6.1)	4 (4.9)	9 (11.0)
Mining	2 (2.4)	4 (4.9)	6 (7.3)
Glass industry	0 (0.0)	1 (1.2)	1 (1.2)
Hairdresser	0 (0.0)	1 (1.2)	1 (1.2)
Welder	3 (3.7)	4 (4.9)	7 (8.5)
Foundry	0 (0)	4 (4.9)	4 (4.9)
Artificial marble	0 (0.0)	2 (2.4)	2 (2.4)
Other	2 (2.4)	4 (4.9)	6 (7.3)

This table shows how many cases of occupational asthma (OA) and non occupational asthma (NOA) are reported in industry sectors. OA cases are related to workplace exposures while NOA cases involve asthma diagnoses not connected to work conditions but are included for an industry overview. SD: Standard deviation

needed to investigate the effect of these exposures on asthma and OA specifically.

In terms of exposure to LMW and HMW agents, no significant differences in asthma development were noted in our study. However, a higher number of cases were exposed to LMW agents, likely due to the industrial focus of the region, including ceramics, casting, and metal industries. This finding may also reflect the higher prevalence of LMW agents in workplaces, especially in industrial settings.[7]

Post-COVID-19 disease has also been associated with asthma-like symptoms.[23,24] In our study, 20.7% of patients with a history of COVID-19 were diagnosed with NOA, indicating that patients with ongoing respiratory symptoms after COVID-19 should not solely be considered to have prolonged COVID symptoms but should also be evaluated for asthma.

Cigarette smoking is a well-known risk factor for chronic diseases, including coronary heart disease and chronic obstructive pulmonary disease. The relationship between smoking and OA remains controversial, with insufficient and contradictory findings regarding its role in increasing OA risk.[25] In our study, 54.9% of asthmatic patients were smokers, and no significant differences in smoking habits were found

Table 3. Correlation between duration of symptoms, duration of exposure, and total IgE levels with pulmonary function and IgE levels

Relationship type	Parameters	Group	Correlation type	p-value
Symptom duration vs. FEV_1 & FEV_1/FVC ratios	FEV1 & FEV1/FVC	NOA	Negative	0.05 & 0.001
Duration of exposure vs. duration of asthma symptoms	Dyspnea duration	OA	Positive	0.007
Symptom duration vs. total IgE levels	Total IgE levels	OA & NOA	Positive	0.012 & 0.019

Table 3 illustrates correlations and their significance (p-value) across groups, negative values indicate inverse relationships, positive values indicate direct relationships. FEV1: Forced expiratory volume in the first second, FVC: Forced vital capacity, IgE: Immunoglobulin E, OA: Occupational asthma, NOA: Non-occupational asthma, statistical significance $p<0.05$

and humidity, particularly related to work environments, can be risk factors for asthma symptoms. While occupational exposures are more prominent in OA, ongoing exposure to such environments, combined with the cessation of other non-occupational environmental exposures, indicates that controlling asthma is complex.

In the OA group, six cases had specific IgE positivity for specific allergens, including latex, inhalant allergens, grass pollen mix, mold mix, and tree mix. However, no significant differences were found between the occupational and NOA groups regarding household environmental factors such as mold exposure and bird keeping. This suggests that occupational exposure plays a key role in OA development, while individual sensitivities and environmental factors may contribute to asthma pathogenesis. Larger studies are needed to further evaluate these influences.

Our region is characterized by common household pet ownership, including parrots and budgerigars, as well as pigeon breeding. Although no significant differences between OA and NOA were found in relation to these exposures, it may be necessary to address non-occupational environmental agents in asthma management, as they are known to cause asthma and extrinsic allergic alveolitis.[22] Further studies are

between OA and NOA patients. Nevertheless, the higher degree of airway obstruction in smokers with OA suggests that both smoking and occupational exposures may have a compounded effect on airway function.

Although no significant differences in airway obstruction or FEV1 levels were observed between asthmatic patients, the lower FEV1 levels in OA patients exposed to non-occupational agents suggest that multiple exposure factors play a role. The combination of non-occupational environmental and occupational exposures may have a synergistic effect on the loss of airway function. These results remain hypothetical and should be confirmed through further studies. The positive correlation between the duration of symptoms and loss of function in OA supports the idea that prolonged exposure exacerbates airway limitation and can lead to permanent airway damage.[26]

CONCLUSION

The etiology of OA is complex, and the interaction between occupational and NOE requires further investigation. While our study's limited sample size and regional factors should be considered, our findings emphasize that asthma cannot be attributed to a single cause, and a comprehensive evaluation

of environmental, occupational, and personal factors is essential. Due to the small sample size, the results should be interpreted as preliminary and require validation through larger-scale studies.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study protocol was approved by the Non-interventional Clinical Researches Ethics Committee of Eskişehir City Hospital (Date: 18.01.2023, Decision No: ESH/GOEK 2022/19SK).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Akgündüz ÜB. Inhalation challenge tests in occupational asthma: why are multiple tests needed? *Turk Thorac J*. 2021;22(2):154-162. doi:10.5152/TurkThoracJ.2021.20007
2. MacKinnon M, To T, Ramsey C, Lemière C, Lougheed MD. Improving detection of work-related asthma: a review of gaps in awareness, reporting and knowledge translation. *Allergy Asthma Clin Immunol*. 2020;16(1):73. doi:10.1186/s13223-020-00470-w
3. Vandenplas O, Malo JL. Definitions and types of work-related asthma: a nosological approach. *Eur Respir J*. 2003;21(4):706-712. doi:10.1183/09031936.03.00113303
4. Hoy R, Burdon J, Chen L, et al. Work-related asthma: a position paper from the Thoracic Society of Australia and New Zealand and the National Asthma Council Australia. *Respirology*. 2020;25(11):1183-1192. doi:10.1111/resp.13951
5. Maestrelli P, Henneberger PK, Tarlo S, Mason P, Boschetto P. Causes and phenotypes of work-related asthma. *Int J Environ Res Public Health*. 2020;17(13):4713. doi:10.3390/ijerph17134713
6. Roio LCD, Mizutani RF, Pinto RC, Terra-Filho M, Santos UP. Work-related asthma. *J Bras Pneumol*. 2021;47(4):e20200577. doi:10.36416/1806-3756/e20200577
7. Meca O, Cruz MJ, Sánchez-Ortiz M, González-Barcala FJ, Ojanguren I, Munoz X. Do low molecular weight agents cause more severe asthma than high molecular weight agents? *PLoS One*. 2016;11(6):e0156141. doi:10.1371/journal.pone.0156141
8. Vandenplas O, Godet J, Hurdubaea L, et al. Are high- and low-molecular-weight sensitizing agents associated with different clinical phenotypes of occupational asthma? *Allergy*. 2019;74(2):261-272. doi:10.1111/all.13542
9. Akgündüz B, Pierre S, Saab L, et al. Application of the new asthma-specific job exposure matrix: a study in quebec apprentice cohort exposed to isocyanates. *IJCOM*. 2022;2(2):83-88. doi:10.53773/ijcom.v2i2.61.83-8
10. Le Moual N, Zock JP, Dumas O, et al. Update of an occupational asthma-specific job exposure matrix to assess exposure to 30 specific agents. *Occup Environ Med*. 2018;75(7):507-514. doi:10.1136/oemed-2017-104866
11. Coates AL, Wanger J, Cockcroft DW, et al. ERS technical standard on bronchial challenge testing: general considerations and performance of methacholine challenge tests. *Eur Respir J*. 2017;49(5):1601526. doi:10.1183/13993003.01526-2016
12. Vandenplas O, Suojalehto H, Aasen TB, et al. ERS task force on specific inhalation challenges with occupational agents. Specific inhalation challenge in the diagnosis of occupational asthma: consensus statement. *Eur Respir J*. 2014;43(6):1573-1587. doi:10.1183/09031936.00180313
13. Gannon PF, Burge PS. Serial peak expiratory flow measurement in the diagnosis of occupational asthma. *Eur Respir J Suppl*. 1997;24:57-63.
14. Şimşek C, Akgündüz B, Sarı G. Comparison of specific inhalation challenge test results in occupational and non-occupational asthma patients: is monitoring peak expiratory flow rate sufficient for diagnosis? *Int J Pul Res Sci*. 2021;5(4):555666. doi:10.1183/13993003.congress-2020.3171
15. Baur X, Degens PO, Sander I. Baker's asthma: still among the most frequent occupational respiratory disorders. *J Allergy Clin Immunol*. 1998;102(6):984-997. doi:10.1016/s0091-6749(98)70337-9
16. Sauvé JF, Friesen MC. Using decision rules to assess occupational exposure in population-based studies. *Curr Environ Health Rep*. 2019;6(3):148-159. doi:10.1007/s40572-019-00240-w
17. Dopart PJ, Locke SJ, Cocco P, et al. Estimation of source-specific occupational benzene exposure in a population-based case-control study of non-hodgkin lymphoma. *Ann Work Expo Health*. 2019;63(8):842-855. doi:10.1093/annweh/wxz063
18. Vandenplas O. Occupational asthma: etiologies and risk factors. *Allergy Asthma Immunol Res*. 2011;3(3):157-167. doi:10.4168/aair.2011.3.3.157
19. Castro-Giner F, Kauffmann F, de Cid R, Kogevinas M. Education: gene-environment interactions in asthma. *Occup Environ Med*. 2006;63(11):776-761. doi:10.1136/oem.2004.019216
20. Tarlo SM. Critical aspects of the history of occupational asthma. *Allergy Asthma Clin Immunol*. 2006;2(2):74-77. doi:10.1186/1710-1492-2-2-74
21. Rollins SM, Su FC, Liang X, et al. Workplace indoor environmental quality and asthma-related outcomes in healthcare workers. *Am J Ind Med*. 2020;63(5):417-428. doi:10.1002/ajim.23101
22. Enomoto T, Sekiya R, Sugimoto H, et al. Hypersensitivity pneumonitis caused by exposure to a gray parrot (*Psittacus erithacus*). *Intern Med*. 2022;61(14):2197-2202. doi:10.2169/internalmedicine.8679-21
23. Esmaeilzadeh H, Sanaei Dashti A, Mortazavi N, Fatemian H, Vali M. Persistent cough and asthma-like symptoms post COVID-19 hospitalization in children. *BMC Infect Dis*. 2022;22(1):244. doi:10.1186/s12879-022-07252-2
24. Guijon OL, Morphew T, Ehwerhemuepha L, Galant SP. Evaluating the impact of coronavirus disease 2019 on asthma morbidity: a comprehensive analysis of potential influencing factors. *Ann Allergy Asthma Immunol*. 2021;127(1):91-99. doi:10.1016/j.anai.2021.03.018
25. Siracusa A, Marabini A, Folletti I, Moscato G. Smoking and occupational asthma. *Clin Exp Allergy*. 2006;36(5):577-584. doi:10.1111/j.1365-2222.2006.02486.x
26. Tiotiu AI, Novakova S, Labor M, et al. Progress in occupational asthma. *Int J Environ Res Public Health*. 2020;17(12):4553. doi:10.3390/ijerph17124553