Home exposure to moisture and mold is associated with poorer asthma control in children: CHAMPIASTHMA study

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Background: Deleterious indoor environment is a risk factor for poor asthma control in children.

Objective: We assessed the association between exposure to moisture and/or mold (EMM) and asthma control in children.

Methods: The CHAMPIASTHMA study is a multicenter cross-sectional observational study. Children with recurrent wheeze/asthma aged 1 to 17 years were stratified by EMM, as assessed by a standardized questionnaire administered to their parents. The primary outcome was asthma control according to Global Initiative on Asthma guidelines. Secondary outcomes were: control according to asthma control test or pediatric asthma control test score, exacerbations (unscheduled visits, oral corticosteroid receipt, and hospitalization), asthma treatment step, and mold sensitization. Clinical trial registration: NCT04918394.

Results: Four hundred twenty-four patients were included, among whom 146 (34%) noted EMM. Patients with EMM more frequently had disease that was not controlled according to Global Initiative on Asthma guidelines (64 [45%] vs 90 [33%]; $P = .03$), and had lower asthma control test scores (22 [19-25] vs 24 [21-25]; $P = .02$), more frequent unscheduled visits (65 [45%] vs 86 [32%]; $P = .02$), and a trend for higher oral corticosteroids receipt in the past year (53 [37%] vs 74 [27%]; $P = .09$). There was no difference in asthma treatment step and hospitalization for exacerbations. Forty-two (12%) of 341 children were sensitized to molds, with no difference between the EMM and non-EMM groups ($P = .85$).

Conclusion: The CHAMPIASTHMA study highlights that EMM is associated with poorer disease control and asthma outcomes in children. The search for EMM during scheduled visits should be systematic, especially in cases of uncontrolled asthma. (J Allergy Clin Immunol Global 2025;4:100415.)

Key words: *Asthma, children, mold, moisture, home exposure, asthma control, sensitization*

Numerous studies have established the link between asthma and the environment. According to the World Health Organization, dampness affects between 10% and 50% of homes in Europe, North America, Australia, India, and Japan.[1] In Europe, a 2012 study estimated the prevalence of dampness and mold at 10% to 15% of the housing stock.[2]

Dampness promotes the proliferation of mold, among other microbial agents.[1] Many studies have shown an association between this exposure and various diseases, such as rhinitis, allergic rhinitis, respiratory infections, and bronchitis, in both adults and children.[3-5] Studies have also shown that exposure to dampness and mold at home increases the risk of developing asthma, especially early-onset asthma, and can be associated with asthma symptoms, such as coughing and wheezing.[4-8] Nevertheless, data on the impact of this indoor exposure on asthma control in children are lacking.

Because asthmatic children may be particularly vulnerable to their home environment, the CHAMPIASTHMA study aimed to report asthma outcomes in a large multicenter population of well-characterized children aged 1 to 17 years, stratified by their exposure to moisture and/or mold (EMM), as assessed by a parental questionnaire. Our primary objective was to assess the association of EMM with asthma control in children. We hypothesized that children with EMM would be less likely to have controlled asthma. Exacerbations, asthma treatment step, and mold sensitization were also assessed.

METHODS

The CHAMPIASTHMA study is a multicenter, prospective, cross-sectional, observational study conducted in 11 French hospitals from the Nord and Pas-de-Calais departments between June 2020 and August 2021. Institutional ethical approval (Comité de Protection des Personnes Ile de France XI, approval 20023-65432) and written informed consent were obtained

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Abbreviations used
ACT: Asthma control test
EMM: Exposure to moisture and/or mold
GINA: Global Initiative for Asthma
OCS: Oral corticosteroids
pACT: Pediatric ACT

from all participating parents or caregivers. The study was registered on ClinicalTrials.gov under NCT04918394.

Study participants

Patients were included if they met the following criteria: (1) children aged 1-17 years; (2) diagnosis of recurrent wheezing (<5 years) or asthma and no other chronic respiratory disease; and (3) seen for a scheduled visit by a pediatric pulmonologist or hospitalized for an asthma exacerbation. Children whose parents or guardians did not consent to the study or could not complete the questionnaire were not included.

Exclusion criteria were the following: (1) patients living less than 50% of the time or for less than 6 months at the home of the responding parent; (2) incomplete responses to the indoor environment assessment; and (3) patients living outside of the Nord and Pas-de-Calais departments.

Questionnaire

The parents completed a questionnaire with the help of the investigating physician during a scheduled visit or hospitalization. Data collected included demographic characteristics, atopic comorbidities (ie, eczema, allergic rhinitis, and food allergy), allergic sensitization (ie, at least one 3 mm skin prick test result and/or specific IgE of 0.35 kU/L in the most recent allergy test), including mold sensitization when available (ie, *Alternaria alternata* and *Aspergillus fumigatus*), and asthma outcomes (ie, asthma control; number of asthma exacerbations in the past year; number of exacerbations requiring an unscheduled visit and/or use of oral corticosteroids [OCS] and/or hospitalization; and maintenance treatment including inhaled corticosteroid doses).

In addition, the following data were recorded: demographic characteristics of the responding parent, including education level (no diploma, other degree before high school, high school degree, bachelor's degree or higher), housing characteristics (house/apartment), location of the home in the Nord and Pas de Calais departments (coastline, former mining area, or Lille metropolitan area), heating and ventilation systems of the home, parental smoking, and presence of pets.

EMM assessment

A previously published questionnaire was used to assess the children with EMM.[9,10] Exposure was defined if at least one of the following 5 items was positive: (1) perception of moisture in the home; (2) apparent moisture in the home; (3) history of water damage in the home within the past year; (4) musty smell in the home; and (5) presence of mold in the home. When parents reported the presence of mold, they specified the surface area (ie, <1 m^2, 1-5 m^2, or >5 m^2) and the room involved. The study population was stratified into exposed (EMM group) and unexposed (non-EMM group).

Outcomes of interest

The primary outcome was asthma control defined according to Global Initiative for Asthma (GINA) guidelines (ie, well-controlled, partly controlled, or uncontrolled).[11] Asthma was classified as well controlled or not controlled (ie, partially controlled or uncontrolled).

The secondary outcomes were control according to asthma control test (ACT) score for children aged ≥12 years or the pediatric ACT (pACT) score for children aged 4 to 11 years.[12,13] ACT was not assessed in children aged 1 to 3 years. Asthma was considered controlled if ACT/pACT was ≥20. Other secondary outcomes included unscheduled visits for asthma in the previous year, severe exacerbations (OCS treatment for ≥3 days or hospitalization) in the previous year, and asthma maintenance treatment step according to the GINA guidelines (step 1 to step 5).[11] The exploratory results were mold sensitizations according to EMM exposure.

Covariate assessment

Exposure to tobacco and pets potentially affecting asthma control were considered covariates.[14,15] Home location, with a potential impact on EMM, was also included as a confounder in the final analysis.[16]

Statistical analysis

Categorical variables were described as frequencies and percentages. Gaussian numerical variables were described as means and standard deviations, and non-Gaussian numerical variables as medians and interquartile ranges. The normality of the numerical variables was checked graphically and tested by the Shapiro-Wilk test. The patients were divided into 2 groups according to whether they had EMM.

Comparisons of demographic and housing characteristics between the EMM and non-EMM groups were made by chi-square or Fisher exact test (eg, when the validity conditions of the chi-square test were not met) for categorical variables, a Student *t* test for Gaussian numerical variables, and a Mann-Whitney *U* test for non-Gaussian numerical variables.

The asthma control end points were compared between the EMM and non-EMM groups by linear regression models for the numerical end points or logistic regression (ie, binary or multinomial) for the qualitative end points. Comparisons were made before and after adjustment for the *a priori*–defined confounding covariates.

Two-tailed tests were performed at a significance level of 5%. Statistical analyses were performed by SAS v9.4 software (SAS Institute, Cary, NC).

RESULTS

Population and household characteristics

In total, 451 questionnaires were filled out. We included 424 patients with complete data for EMM. A total of 146 patients were assigned to the EMM group. The characteristics of patients with and without EMM are listed in Table I. Eczema was more frequent in the EMM group (37% vs 25%; $P = .01$), and the level of

TABLE I. Population characteristics

Characteristic	Total population (N = 424)	No EMM (n = 278)	EMM (n = 146)	*P*
Male sex (n′ = 423)	279 (66)	186 (67)	93 (64)	.57
Age (years) (n′ = 415)	8.8 (5.8-12.5)	8.8 (5.9-12.8)	8.6 (5.7-12.2)	.27
1-5 years	112 (27)	71 (26)	41 (29)	.73
6-11 years	182 (43)	119 (43)	63 (44)	
12-17 years	126 (30)	86 (31)	40 (28)	
Atopic comorbidities	248 (59)	155 (56)	93 (64)	.11
Eczema	124 (29)	70 (25)	54 (37)	.01
Allergic rhinitis	108 (26)	64 (23)	44 (30)	.11
Food allergy	72 (17)	49 (18)	23 (16)	.63
Atopy (n′ = 415)	286 (69)	195 (71)	91 (65)	.92
Home location (n′ = 420)				
Lille and suburbs	294 (69)	193 (69)	101 (69)	
Coastline	78 (18)	51 (19)	27 (19)	
Former mining area	38 (9)	24 (9)	14 (10)	
Responder's education level (n′ = 411)				.02
No diploma	29 (7)	15 (6)	14 (10)	
Other degree before high school	77 (19)	43 (16)	34 (25)	
High school degree	107 (26)	74 (27)	33 (24)	
Bachelor's degree	107 (26)	81 (30)	26 (19)	
Higher degree	91 (22)	59 (22)	32 (23)	

Results for quantitative variables are expressed as medians (interquartile ranges). Qualitative variables are expressed by nos. (%) of patients, excluding missing data. *P* value is significant if $P < .05$. *n′*, Counts of nonmissing data.

education of the responding parent was lower ($P = .02$). There were no other significant differences between the EMM and non-EMM groups.

Regarding their home environment, children with EMM were significantly more likely to live in apartments (36% vs 11%; $P < .001$) and were more often exposed to tobacco smoke (49% vs 35%; $P = .007$) (Table II).

EMM

Of the 146 patients in the EMM group, 47 (32%) reported water damage, 70 (49%) reported a feeling of dampness, and 88 (60%) reported an apparent dampness. Only 20 (14%) reported a musty odor. Of the 94 patients (65%) who reported visible mold, the majority of affected rooms/sites were the bathroom and window seals (50%), with the child's bedroom affected in 14% of cases. Visible mold did not exceed an estimated 1 m^2 in 47% of cases, but the surface area exceeded 5 m^2 in 12% of cases (Table III).

Primary outcome: Control according to GINA guidelines

According to the GINA criteria, asthma was not controlled in 154 patients (37%). This was significantly more frequently observed in patients from the EMM group versus the non-EMM group (45% vs 33%; $P = .02$) (Table IV). This difference remained significant after adjustment for exposure to tobacco, pets, and the living environment ($P = .03$).

Secondary outcomes

According to the ACT/pACT score, asthma was not controlled in 81 (22%) of all patients, which was more frequent in the EMM group than in the non-EMM group (39 [31%] vs 42 [17%]; $P = .002$). The difference remained significant after adjustment for exposure to tobacco, pets, and the living environment ($P = .005$). Moreover, the EMM group showed a significantly lower score than the non-EMM group (23 [20-25] vs 24 [21-25]; $P = .01$) (Table IV). These differences were also found in the age group of 4 to 11 years: asthma was more frequently not controlled, based on pACT of ≤20 in the EMM group (33% vs 16%, $P = .002$), and their pACT score was significantly lower (22 [19-25] vs 24 [21-26], $P = .004$) than in the non-EMM group. These differences based on ACT were not found in the 12-to-17–year age group.

There were significantly more unscheduled visits in the previous year for asthma symptoms in patients with EMM compared to patients without EMM (45% vs 32%; $P = .007$). There was also a trend toward more exacerbations in the previous year in patients with EMM compared to patients without EMM (59% vs 49%; $P = .06$), and significantly more severe exacerbations treated with OCS (37% vs 27%; $P = .046$) (Table V). The 2 groups did not show significant differences in hospitalization for asthma exacerbation ($P = .13$) or asthma maintenance treatment step ($P = .48$).

Exploratory outcomes

Two hundred eighty-six patients (67%) had at least one allergic sensitization to common airborne allergens (eg, pollen, dust mites, dog and cat dander, and molds). Three hundred forty-one patients (80%) had a mold sensitization assessment. Of these, 42 patients (12%) were sensitized, 34 (10%) to *A alternata* and 14 (4%) to *A fumigatus*. In the EMM group, 15 patients (10%) were sensitized to *A fumigatus* and/or *A alternata* versus 27 (10%) in the non-EMM group ($P = .85$) (Table VI). Moreover, there was no difference between EMM and non-EMM groups for the other sensitizations we explored: house dust mite ($P = .73$), pollens ($P = .51$), and cat and dog dander ($P = .39$).

DISCUSSION

In our large population of 424 asthmatic children aged 1 to 17 years included in the CHAMPIASTHMA study, EMM, as

TABLE II. Housing characteristics

Characteristic	Total population (N = 424)	No EMM (n = 278)	EMM (n = 146)	*P*
Housing type				
Apartment (n′ = 418)	81 (19)	29 (11)	52 (36)	<.001
Built before year 2000 (n′ = 380)	244 (64)	158 (63)	86 (67)	.21
Housing tenure (years) (n′ = 405)	6.3 (3.3-10.8)	7.2 (3.4-11.8)	5.7 (3.3-8.9)	.02
Ventilation (n′ = 409)				
Natural ventilation	344 (84)	223 (83)	121 (86)	.35
Mechanical supply and exhaust ventilation	313 (77)	204 (76)	109 (78)	.65
Mechanical exhaust ventilation	51 (13)	35 (13)	16 (11)	.65
Heating system (n′ = 416)				
Electric heating	114 (27)	75 (27)	39 (28)	.93
Fuel/gas	261 (63)	165 (60)	96 (68)	.11
Wood-burning stove	75 (18)	63 (23)	12 (9)	<.001
Smoker in home	168 (40)	97 (35)	71 (49)	.007
Pet in home	256 (60)	169 (61)	87 (60)	.81

Results for quantitative variables are expressed as medians (interquartile ranges). Qualitative variables are expressed by nos. (%) of patients, excluding missing data. *P* value is significant if $P < .05$. *n′*, Counts of nonmissing data.

TABLE III. EMM characteristics

Characteristic	No. (%) (n = 146)
Positive answers to questionnaire	
Feeling of dampness	70 (49)
Apparent dampness	88 (61)
Water damage in the last year	47 (32)
Odor of mold	20 (14)
Apparent mold	94 (65)
Mold location	
Damp areas (bathroom, window seals)	73 (50)
Child's bedroom	21 (14)
Other rooms	57 (39)
Estimated apparent mold surface	
<1 m^2	44 (47)
1 to 3 m^2	16 (17)
3 to 5 m^2	10 (11)
>5 m^2	11 (12)

Results for qualitative variables are expressed by nos. (%) of total number of patients, excluding missing data.

assessed by questionnaire, was frequent, reported in one third of the population. We observed the positive association of this exposure with poor asthma control and with asthma symptoms requiring unscheduled medical visits.

In our study, the reported rate of EMM was 34%, with 21% apparent dampness and 22% apparent mold in homes, which is higher than the national average and the estimated prevalence in Europe of 10% to 15%.[2] This result can be explained by the geographical location of the region in which the study was conducted, as Northern France has a humid oceanic climate. Furthermore, compared to the general French population, this region is characterized by older housing stock and a lower socioeconomic level—factors associated with home moisture and mold proliferation.[16,17]

Our study showed a significant association between reported home EMM and poor asthma control according to GINA criteria and ACT/pACT scores. This association was maintained after adjustment for exposure to tobacco, pets, and area of residence, suggesting an independent association. Our study was not designed for an age group analysis, with a possible lack of power, but EMM may have a different effect according to age, as suggested by the different results for the ACT and pACT scores. On mold exposure, the mechanisms leading to asthma symptoms are multiple and include the specific characteristics of the fungal species, the presence of mold proteases that alter the integrity of the respiratory epithelial barrier, and the type 2–biased inflammation induced by the host immune response.[18] One possible explanation for the age difference that we observed is that these mechanisms intervene differently in preschoolers, who have smaller airways and display an immature immune response compared to older children.[19]

Previous studies have shown a link between EMM, asthma symptoms, and other respiratory diseases. A 2007 meta-analysis found an association between EMM and cough/wheezing in children and adults, and with upper respiratory tract symptoms and ever-diagnosed asthma in the general population.[8] Another meta-analysis by Fisk et al found an association between EMM and respiratory infections in children and infants.[3] However, to our knowledge, these studies did not specifically evaluate the impact on asthma control itself. A study conducted in Montreal, Canada, analyzed the environmental factors associated with poor asthma control (ie, GINA criteria and absence from school or day care due to asthma during the past 3 months) in 980 children aged <12 years.[20] EMM, assessed by the same 5 humidity indicators as in our study, was one of the main environmental factors associated with poor control, increasing the risk by 20%. Another study conducted in Puerto Rico among 177 children found a threefold increased risk of EMM in uncontrolled asthma patients (according to GINA criteria) compared to patients with controlled disease.[21]

Several literature reviews have reported an association between EMM and asthma exacerbations,[5,22] with that of Caillaud et al[5] noting the existence of sufficient evidence of a causal relationship between EMM and asthma development and exacerbation in children. In our study, patients with EMM were more likely to have higher health care utilization, with significantly more unscheduled visits for asthma symptoms and a trend toward more frequent asthma exacerbations requiring OCS.

We found no significant association between EMM and asthma maintenance treatment step, which is an indicator for assessing the level of asthma severity. Other studies have shown conflicting results. In the French COBRAPed cohort, which

TABLE IV. Asthma control

Characteristic	Total population (n = 424)	No EMM (n = 278)	EMM (n = 146)	Unadjusted analysis		Adjusted analysis	
				P	Het-*P*	P_a	Het-P_a
GINA score (n′ = 414)				.02		.03	
Patients well controlled	260 (63)	181 (67)	79 (55)				
Patients not controlled	154 (37)	90 (33)	64 (45)				
Patients partly controlled	102 (25)	57 (21)	45 (32)				
Patients uncontrolled	52 (13)	33 (12)	19 (13)				
According to ACT or pACT score (n′ = 372)							
Median score					.11		.09
Overall	23 (20-25)	24 (21-25)	23 (20-25)	.01		.02	
Children aged 4-11 years (pACT score)	23 (20-25)	24 (21-26)	22 (19-25)	.003		.004	
Children aged >11 years (ACT score)	22 (20-25)	23 (21-25)	22 (19-25)	.78		.99	
ACT or pACT < 20					.47		.44
Overall	81 (22)	42 (17)	39 (31)	.002		.005	
Children aged 4-11 years (pACT score)	52 (22)	25 (16)	27 (33)	.004		.006	
Children aged >11 years (ACT score)	29 (21)	17 (18)	12 (27)	.21		.31	

Results for quantitative variables are expressed as medians (interquartile ranges). Qualitative variables are expressed by nos. (%), excluding missing data. *P* value was adjusted (P_a) for tobacco, pet exposure, and location. *P* value is significant if $P < .05$. Het-*P* refers to *P* value for heterogeneity. *n′,* Counts of nonmissing data.

TABLE V. Asthma maintenance treatment step, exacerbations and health care utilization

Characteristic	Total population (n = 424)	No EMM (n = 278)	EMM (n = 146)	*P*	P_a
Treatment step according to GINA (n′ = 419)				.48	.38
Step 1	75 (18)	55 (20)	20 (14)		
Step 2	32 (8)	21 (8)	11 (8)		
Step 3	123 (29)	76 (28)	47 (32)		
Step 4	168 (40)	106 (39)	62 (43)		
Step 5	21 (5)	15 (6)	6 (4)		
Daily ICS dose (μg) (n′ = 416)	200 (100-250)	200 (100-200)	200 (100-200)	.11	.16
At least one asthma exacerbation in past year	202 (53)	124 (49)	78 (59)	.06	.07
At least one unscheduled visit for asthma in past year (n′ = 416)	151 (36)	86 (32)	65 (45)	.007	.02
At least one hospitalization for asthma in past year (n′ = 417)	55 (13)	31 (11)	24 (17)	.13	.18
At least one course of OCS in past year (n′ = 415)	127 (31)	74 (27)	53 (37)	.04	.09

Results for quantitative variables are expressed as medians (interquartile ranges). Qualitative variables are expressed by nos. (%), excluding missing data. *P* value was adjusted (P_a) for tobacco, pet exposure, and location. *P* value is significant if $P < .05$. *ICS,* Inhaled corticosteroid; *n′,* counts of nonmissing data.

assessed the factors associated with a diagnosis of severe asthma according to European Respiratory Society/American Thoracic Society criteria in 131 preschool children (3-6 years old) and 207 school-age children (7-11 years old), the authors found that this diagnosis in preschool children was significantly associated with EMM compared to children with nonsevere asthma (odds ratio [95% CI], 4.22 [1.25-18.2]). In contrast, this factor was not observed in school-aged children.[23] This result suggests that the effect of mold and moisture may differ by patient age.

These data raise the question of the effectiveness of home mold and moisture remediation in improving asthma control. EMM might be considered a treatable trait that should be considered in a personalized approach. Kalayci et al summarized the various measures that can reduce mold in homes, such as proper ventilation, basement dehumidifiers, and fungicides.[24] Among other studies, the results of a randomized trial by Burr et al suggested that mold remediation reduces asthma symptoms, medication, and rhinitis.[25] In addition, these interventions could reduce health care costs, as suggested by Kattan et al, with a decrease in symptoms, unscheduled visits, and receipt of inhaled β-agonists.[26] Indoor dampness and mold remediation to decrease asthma symptoms and medication use are now strongly recommended by the GINA guidelines.[11]

Finally, we observed mold sensitization in 42 patients (12%), which is consistent with some studies including asthmatic children but more than in the French COBRAPed cohort (7-9%).[27] There was no significant difference in mold sensitization between the EMM and non-EMM groups. Furthermore, there were no differences regarding asthma control and exacerbations between children sensitized to *A alternata* or *A fumigatus* and those nonsensitized (data not shown).

The strength of our study was its assessment of the effect of EMM on asthma control in a multicenter study with a large number of subjects and few missing data. We assessed control using the GINA criteria for the primary end point and the validated ACT/pACT questionnaire for the secondary end point. Using two different tools strengthens the association between EMM and asthma control. Additionally, the demographic characteristics were overall comparable between the exposed and nonexposed populations, except for the frequency of atopic dermatitis, which may also be favored by EMM,[28,29] and the frequency of exposure to smoking, which was considered in the adjusted analysis.

TABLE VI. Airborne sensitization

Sensitization to:	Total population (N = 424)	No EMM (n = 278)	EMM (n = 146)	*P*
Mold (n′ = 341)*	42 (12)	27/225 (12)	15/116 (13)	.86
House dust mite (n′ = 386)†	240 (62)	162/255 (64)	78/131 (60)	.73
Pollen (n′ = 376)‡	123 (33)	85/247 (34)	38/129 (29)	.51
Cat or dog dander (n′ = 374)	131 (35)	93/248 (38)	38/126 (30)	.39

Airborne sensitization was defined in case of at least one 3 mm skin prick test result and/or specific IgE ≥ 0.35 kU/L in most recent allergy test. Qualitative variables are expressed by nos. (%), excluding missing data. *P* value is significant if $P < .05$. *n′*, Counts of nonmissing data.
**Aspergillus fumigatus* and *Alternaria alternata*.
†*Dermatophagoides pteronyssinus* and *Dermatophagoides farinae*.
‡Grass and birch pollens.

A main limitations of our work is the self-reported assessment of EMM by parents. The assessment of EMM is difficult because of the lack of a reference standard or standardized assessment, which has led to differences in the definitions used in the literature. Self-report questionnaires are the easiest and most cost-effective way of assessing the presence of moisture or mold in the home, but they have certain limitations. The general population is untrained and may either fail to report certain sources of dampness or mold or overestimate them, especially if the subjects are children, ill (eg, asthma, allergies), or symptomatic.[2,6,30] However, the questions used here have often been used in the literature and are the basis for real-life assessment.[6,31] Finally, recruitment in hospital settings might select patients with more severe disease than those of the general asthmatic population, limiting the study's external validity.

In conclusion, this study adds to the limited data on the deleterious association of moisture and molds with asthma control and other important asthma outcomes. EMM can be easily assessed during a scheduled visit using the 5 questions of the questionnaire, and a diagnosis of EMM exposure should lead clinicians to educate parents and seek mold remediation in their home, although its impact on asthma outcomes remains to be determined. Further studies are required to understand the pathogenic mechanisms underpinning their effect on asthma pathologic processes, considering the lifelong course of asthma.

DISCLOSURE STATEMENT

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Clinical implication: Exposure to moisture and/or mold may alter asthma control in children. The search for this exposure during scheduled visits should be systematic, especially in cases of uncontrolled wheeze/asthma.

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