

Specific Molds Associated With Asthma in Water-Damaged Homes

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Objective: We sought to determine if specific molds were found in significantly higher concentrations in the water-damaged homes of asthmatic children compared with homes with no visible water damage. **Methods:** The mold concentrations in the dust in asthmatic children's bedrooms in water-damaged homes ($N = 60$) and control homes ($N = 22$) were measured by mold-specific quantitative polymerase chain reaction. **Results:** Two molds, *Scopulariopsis brevicaulis* and *Trichoderma viride*, had significantly ($P < 0.05$) higher concentrations in asthmatics' homes compared with control homes and three other molds (*Penicillium crustosum* group, *Stachybotrys chartarum*, and *Wallemia sebi*) had P values < 0.1 . **Conclusions:** A relative moldiness index was developed to predict the likely development of asthma in water-damaged homes in Cleveland. (J Occup Environ Med. 2006;48:852–858)

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Asthma afflicts an ever increasing number of children in the United States.¹ Growing evidence blames this increase on the combination of many environmental and social factors. Childhood exposure to molds may be one such factor.^{2–4} The Institute of Medicine's report on dampness and health expressed the opinion that there was scientific evidence linking molds and damp environments with asthma symptoms.⁵ However, the report does not suggest whether it is all molds or specific molds that might be important in the etiology of asthma symptoms. Because there are mold cells in every environment, it would be nearly impossible to completely avoid exposures.

In earlier work, we established that there were some molds that were statistically more common in water-damaged homes, which we called group 1 molds.⁶ On the other hand, there were other molds that are found in every home, the group 2 molds. In this study, these mold groupings were used to evaluate mold populations in asthmatics' water-damaged homes. Mold populations were measured in dust samples and air samples by mold-specific quantitative polymerase chain reaction (MSQPCR) from water-damaged homes of asthmatics children in Cleveland, Ohio, and results compared with a set of control homes.

Materials and Methods

Cleveland Asthma Study and Selection of Homes and Dust Sampling

This study was conducted through the Center for Chronic Conditions of Childhood, a clinical research facil-

ity at Rainbow Babies and Children's Hospital. The study protocol was approved by the Institutional Review Board of University Hospitals of Cleveland and written informed consent was obtained from parents.

Water-damaged homes of asthmatic children were identified as described earlier.⁷ The mold score was calculated by adding mold scales across all rooms, which included the basement, kitchen, TV/living room, child's bedroom, attic, and other bedroom. The visible mold scales in each area had four categories, that is, 0 = no visible mold, 1 = <4 sq ft, 2 = 4 to 32 sq ft, and 3 = >32 sq ft.

Dust samples were taken from 82 homes, 60 where an asthmatic child lived and 22 controls. Dust was collected by vacuuming 1 to 3 m² of carpeted or hard surface floors for 5 minutes using a Eureka (Bloomington, IL) "THE BOSS" Mighty Mite 9 amp vacuum cleaner, model no. 3670. Dust was collected with a Hysurf insert (Midwest Filtration Co., Fairfield, OH) located in the vacuum hose in-line between the extension hose inlet and the crack and crevice tool. The Hysurf is a 37-mm diameter, 0.8- μ m pore size polycarbonate filter. While vacuuming, the crack and crevice tool was positioned at a 45° angle so that the opening of the wand made full contact with the surface to be vacuumed. The wand was moved across the template area in an S-pattern at an approximate rate of 0.3 m/s. On completion of the initial direction, the sampling was repeated in a direction perpendicular (90°) to the original direction. This process was repeated until time expired. Dust was processed by the IBT Reference Laboratory (Lenexa, KS). The samples were each sieved (pore size 300 μ m) before sending a portion to the U.S. Environmental Protection Agency Laboratory for MSQPCR analysis and a similar portion to P&K Microbiology Services Inc. (Cherry Hill, NJ) for analysis by culturing.

Mold-Specific Polymerase Chain Reaction Analysis of Dust

Methods have been reported previously for preparing conidial suspensions from fungal cultures, extracting DNA, performing MSQPCR analyses, and preparing standard calibration curves for target conidia versus delta cycle threshold values ($\Delta C_T = C_{T,target} - C_{T,reference}$) using coextracted DNA from *Geotrichum candidum* as an exogenous reference.⁸⁻¹⁰ All primer and probe sequences, as well as known species comprising the assay groups, were published at the web site: <http://www.epa.gov/microbes/moldtech.htm>. Primers and probes were synthesized commercially (Applied Biosystems, Foster City, CA; Integrated DNA Technologies, Coralville, IA; Sigma Genosys, Woodlands, TX).

Culture-Based Analysis of Dust

Part of each dust sample was sent to P&K Microbiology Services for analysis. Dust samples were weighed, extracted in sterile distilled water with 0.1% Tween-80 wetting agent, diluted serially, and inoculated onto 2% malt extract agar (MEA), dichloren glycerol 18% agar (DG18), and cornmeal agar (CMA). Ingredients of MEA and CMA were obtained from DIFCO (Detroit, MI) and DG18 from Oxoid (Remel Inc., Lenexa, KS). All plates were incubated at 25°C $\pm$ 0.5°C and examined in 7 to 10 days. Colonies were counted under a stereomicroscope and fungal species were identified by morphologic characters referencing mycology keys. For each home and mold species, the plate with the largest population count among the three media was used in the statistical analysis.

Air Sampling

The air sampling pumps were Gillian Aircon2 (Sensidyne, Clearwater, FL) calibrated twice a month using a primary standard calibrator DryCal DC-1 Flow Calibrator as a primary standard and calibrated before every new sampling event using the

built-in rotameter as a secondary standard. A cassette (37 mm) holding a polycarbonate filter (Osmonics, Minnetonka, MN) with pore size of 8 μ m was used "open-faced" by removing the top (inlet) ring. Tygon tubing with a metal male adapter was inserted into this outlet side of the cassette.

The cassette was attached to the adjustable Gillian AirCon 2 stand and positioned approximately 1 m above the floor. The "open face" is positioned away from the wall, furnace supplies, cold air returns, open windows, and doorways to the greatest extent possible in an effort to obtain a representative sample. The samples were collected on the same day as dust samples with the heating/cooling/ventilation system operating under normal conditions.

The flow rate was adjusted for 10.0 L per minute (LPM) and the sampling time was 6 to 8 hours. The flow rate was checked after 15 minutes and before leaving the house. At the conclusion of the sampling period, the flow rate and the end run time were recorded. The inlet ring with blue cap and red outlet cap were replaced and the cassette stored at approximately 5°C before the filters were analyzed, as previously described.⁴

Statistical Analyses

In general, mold concentration data analyzed with MSQPCR having a minimum detection limit of 1 CFU mg⁻¹ were treated as left-censored data with appropriate statistical methods applied.¹¹ Standard non-parametric statistical methods are sufficient for analyses of MSQPCR data having a single minimum detection limit presented in this article. Procedurally, nondetections were set at half the minimum detection limit.

Two types of analyses were performed: 1) two-group comparisons in mold spore concentrations between asthma study and control homes, and 2) correlation analysis comparing sampling methods and quantification methods. Two-group comparisons

were made on the basis of the Wilcoxon rank sum test. Correlation coefficient analyses also used a non-parametric approach based on Spearman's rho statistic, which measures the strength of monotonic correlation on paired samples (x, y). In each case, the null hypothesis tested for the correlation coefficient (ρ) states that x and y have correlation $\rho = 0$ versus the alternative hypothesis, $\rho \neq 0$.

Comparison of Mold Populations in Dust From Water-Damaged Asthmatic Child's Bedroom and Dust From Bedroom of Control Homes

The two-sided Wilcoxon rank sum test was used to compare the populations of 33 molds in homes of asthmatic children versus control homes.

Comparison of Dust Sample Analysis by Culture-Based and Mold-Specific Quantitative Polymerase Chain Reaction Analyses

Quantification results for samples in the MSQPCR and culture-based data sets were paired only for the molds that were identified to species in both sets. In cases in which the MSQPCR assays target a group of organisms, for example, *Penicillium crustosum* group, then the populations of the members of that group, as identified by culture analysis, were added together. In addition, when MSQPCR analysis divides a species into two assays, for example, *Cladosporium cladosporioides* types 1 and 2, for this comparison to live culture statistical analysis, these values were added together. Correlations were computed on nine mold species in 60 homes with at least nine samples above detection limits.

Mold in Dust in Child's Room Compared With Air Samples

The mold populations in dust samples from the asthmatic's bedroom were compared with air samples taken by cassette filters in 52 of the

same homes. Correlations in species-specific mold concentrations were analyzed using Spearman's estimate of correlation.

Relative Moldiness Index

The relative moldiness index (RMI) is a summary metric that ranks the relative moldiness of homes and is computed for each home as the sum of logs of the concentrations of the group 2 species subtracted from the sum of the logs of the concentrations of the group 1 species (Table 1).

Results

Cleveland Home Types and Demographics

The mean age of the asthmatic children was 6.8 years and the majority was black (66%). Over 75% of

the children had mild persistent to severe persistent asthma. One third of the families had an annual income <\$20,000 and 83% of families resided in traditional, single-family housing. Most of the housing stock in the study dated from the first half of the 20th century.⁷ The average mold score for the water damaged homes was 3.03 ± 1.59 .⁷

Molds in Dust Based on Mold-Specific Quantitative Polymerase Chain Reaction Analysis

Notched box plots are shown in Figure 1 comparing the distribution of RMI values between the 60 homes with children who developed asthma and the 22 control homes. The median RMI value is higher among the 60 homes of asthmatics versus the 23 control homes ($T = 1.9107$, $df = 58$,

TABLE 1

Odds Ratios for Predicting Illness Based on the Relative Moldiness Index (RMI)

RMI	True-Negative	False-Positive	False-Negative	True-Positive	No. Correct	No. Wrong	Odds Ratio
-9	2	20	1	59	61	21	5.90
-8	2	20	1	59	61	21	5.90
-7	2	20	1	59	61	21	5.90
-6	2	20	1	59	61	21	5.90
-5	3	19	1	59	62	20	9.32
-4	5	17	2	58	63	19	8.53
-3	5	17	4	56	61	21	4.12
-2	6	16	6	54	60	22	3.38
-1	6	16	6	54	60	22	3.38
0	9	13	8	52	61	21	4.50
1	11	11	10	50	61	21	5.00
2	11	11	11	49	60	22	4.45
3	12	10	14	46	58	24	3.94
4	12	10	17	43	55	27	3.04
5	12	10	18	42	54	28	2.80
6	12	10	20	40	52	30	2.40
7	14	8	25	35	49	33	2.45
8	15	7	27	33	48	34	2.62
9	16	6	35	25	41	41	1.90
10	17	5	38	22	39	43	1.97
11	18	4	41	19	37	45	2.09
12	19	3	45	15	34	48	2.11
13	19	3	46	14	33	49	1.93
14	19	3	48	12	31	51	1.58
15	20	2	49	11	31	51	2.24
16	20	2	52	8	28	54	1.54
17	20	2	54	6	26	56	1.11
18	21	1	54	6	27	55	2.33
19	21	1	56	4	25	57	1.50
20	21	1	58	2	23	59	0.72
21	21	1	58	2	23	59	0.72

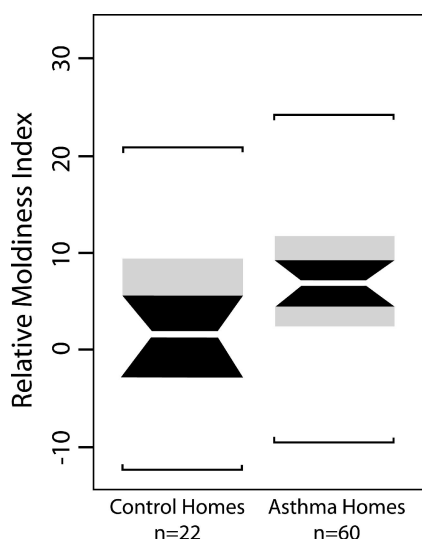


Fig. 1. Notched box plot of relative moldiness index values for homes with asthmatic child versus control homes. The notches form approximate 95% confidence intervals around the median. *t* test results support inference of larger mean among asthmatic child's homes ($P = 0.06$).

$P = 0.06$) equal variance assumption supported (*F* test, $P = 0.96$).

Table 2 shows the differences in the mold populations of specific molds between asthmatics' homes and control homes. Two molds, *Scopulariopsis brevicaulis* and *Trichoderma viride*, had significantly ($P < 0.05$) higher concentrations in the asthmatics' homes compared with control homes and three others (*Penicillium* group 2, *Stachybotrys chartarum*, and *Wallemia sebi*) had P values < 0.1 .

There were seven molds (*Alternaria alternata*, *Aureobasidium pullulans*, *Cladosporium cladosporioides* types 1 and 2, *Cladosporium sphaerospermum*, *Epicoccum nigrum*, and *Penicillium spinulosum*) that were significantly lower ($P < 0.05$) in concentration in asthmatics' homes compared with control homes and one other (*Acremonium strictum*) with a P value < 0.1 (Table 2).

Mold-Specific Quantitative Polymerase Chain Reaction Results Compared With Live Culturing Analyses of Dust Samples

Correlation results between MSQPCR and culture-based analy-

ses are shown in Table 3 along with the number of paired samples measured above minimum detection limits for both quantification methods. The concentration data for only nine mold species were comparable to the MSQPCR data, because most mold concentrations measured by culturing were below the detection limit.

Comparison of Mold Concentrations From Bedroom Dust Sample Analysis and Air Samples

Table 4 summarizes the results for concentrations of 36 mold species from the 52 study homes. Overall, correlations were 0.34 or below and three were weakly negative. For 13 species, correlations were not estimable because one or both samples reported fewer than 10 detections. Collectively, these results suggest a lack of real correlation between the two sampling techniques.

Predictive Value of the Relative Moldiness Index

Table 1 shows the odds ratios for each of the RMI values. For example, an RMI of 1 has an odds ratio of 5.0. In this context, the odds ratio would quantify the relative proportion (relative risk) for the population of study homes of developing asthma. Therefore, 61 of 82 homes would be correctly assessed, that is, 50 true-positives and 11 true-negatives.

Discussion

The Institute of Medicine report on the role of "Damp Indoor Spaces and Health" concluded that "factors" in damp environments, probably including molds, are involved in asthma symptoms.⁵ If so, what are the specific molds involved? This is an important question because total elimination of childhood mold exposures is not practical. Therefore, it was important to determine if there are certain molds that are likely associated or involved with

asthma pathology and target those for elimination.

Using the RMI values, we were able to show that the mold populations in homes of children with asthma were different than the controls (Fig. 1). However, only members of the group 1 molds were in higher concentrations in the bedroom dust from asthmatics' homes. This is demonstrated by the ratios greater than one of the geometric means (GM) of the mold dust population in asthmatics' over control home dust (Table 2). Some of these populations were significantly greater, including *Scopulariopsis brevicaulis* and *Trichoderma viride*, or nearly significant like *Penicillium crustosum* group, *Stachybotrys chartarum*, and *Wallemia sebi*. All of the group 2 molds had GM ratios less than 1 and nearly half were statistically significantly lower. Thus, populations of some specific molds were directly associated with water-damaged homes of asthmatic children and some clearly were not.

Clinically, moderately severe asthmatic children had a significant decrease in symptom score ($P < 0.006$) and symptom days ($P < 0.003$) after remediation of their water-damaged homes.⁷ Asthmatic children living in homes not receiving the remediation did not significantly change in symptom score or symptom days. During the period from 6 months postrandomization to the end of the study, which was primarily postremediation, asthmatics receiving home interventions had a lower rate of asthma exacerbations compared with control asthmatics (one of 29 vs 11 of 33, respectively, $P = 0.003$).⁷ This suggests that the way to estimate the potential for mold-related asthma health problems in a water-damaged asthmatic's home is to measure the group 1 molds in a bedroom dust sample.

Sample analysis and collection limitations of previous studies prevented linkage of specific molds and asthma symptoms. Most studies of childhood mold exposures have re-

TABLE 2

Comparison of Mold Populations in Cell Equivalents per Gram of Dust From Asthma and Control Homes

	Geometric Means		GM Ratio	Wilcoxon Statistic	P Value
	Asthma Homes (n = 60)	Control Homes (n = 22)			
Group 1					
<i>Aspergillus fumigatus</i>	493.98	733.76	0.673	−0.823	0.411
<i>Aspergillus ochraceus</i>	1895.46	2117.95	0.895	−0.262	0.794
<i>Aspergillus penicillioides</i>	103,285.40	72,823.67	1.418	0.173	0.863
<i>Aureobasidium pullulans</i>	417,991.00	727,917.30	0.574	−2.329	0.020*
<i>Aspergillus restrictus</i>	227.79	298.52	0.763	−0.332	0.740
<i>Aspergillus sclerotiorum</i>	474.12	429.75	1.103	0.238	0.812
<i>Aspergillus unguis</i>	3831.60	1881.66	2.036	1.002	0.316
<i>Aspergillus versicolor</i>	4261.87	1948.05	2.188	0.839	0.402
<i>Chaetomium globosum</i>	1135.01	1438.13	0.789	−0.417	0.677
<i>Cladosporium sphaerospermum</i>	4714.39	8172.98	0.577	−2.204	0.028*
<i>Eurotium amstelodami</i>	149,314.50	128,746.90	1.160	0.392	0.695
<i>Penicillium brevicompactum</i>	3652.60	2353.54	1.552	0.483	0.629
<i>Penicillium corylophilum</i>	2317.31	1328.69	1.744	0.777	0.437
<i>Penicillium group 2</i>	2604.09	654.49	3.979	1.764	0.078*
<i>Penicillium purpurogenum</i>	478.79	474.68	1.009	−0.051	0.959
<i>Penicillium spinulosum</i>	710.90	3600.06	0.197	−2.508	0.012*
<i>Penicillium variable</i>	1050.69	1033.93	1.016	−0.101	0.920
<i>Paecilomyces variotii</i>	1718.81	1575.08	1.091	−0.090	0.929
<i>Scopulariopsis brevicaulis</i>	1179.00	480.64	2.453	2.112	0.035*
<i>Scopulariopsis chartarum</i>	446.12	577.68	0.772	−0.452	0.651
<i>Stachybotrys chartarum</i>	648.07	334.71	1.936	1.690	0.091*
<i>Trichoderma viride</i>	1602.96	284.82	5.628	2.601	0.009†
<i>Wallemia sebi</i>	18,954.01	8442.97	2.245	1.952	0.051*
Group 2					
<i>Alternaria alternata</i>	16,452.45	55,594.45	0.296	−3.459	0.001‡
<i>Acremonium strictum</i>	946.14	2177.42	0.435	−1.729	0.084*
<i>Aspergillus ustus</i>	1039.10	1794.22	0.579	−1.229	0.219
<i>Cladosporium cladosporioides 1</i>	177,704.30	544,160.00	0.327	−3.773	<0.001‡
<i>Cladosporium cladosporioides 2</i>	16,155.37	50,671.42	0.319	−2.507	0.012*
<i>Cladosporium herbarum</i>	33,532.34	48,206.32	0.696	−0.947	0.344
<i>Epicoccum nigrum</i>	407,868.70	920,578.10	0.443	−3.093	0.002†
<i>Mucor amphibiorum</i>	12,028.50	20,292.92	0.593	−1.031	0.303
<i>Penicillium chrysogenum</i>	11,362.78	11,222.07	1.013	0.215	0.830
<i>Rhizopus stolonifer</i>	571.72	724.86	0.789	−0.818	0.413

* $P \leq 0.1$; † $P \leq 0.01$; ‡ $P \leq 0.001$.

GM ratio indicates geometric means of asthmatic home/GM of control home.

TABLE 3

Correlations in Mold Spore Density Estimates for MSQPCR versus Live Culturing in 60 Study Homes

	Homes With Live Culture Detections	Homes With MSQPCR Detections	Correlation Estimate (Spearman's rho)	P Value
Group 1				
<i>Aspergillus fumigatus</i>	9	33	0.189	0.146
<i>Aspergillus ochraceus</i>	9	40	0.221	0.089
<i>Aureobasidium pullulans</i>	43	60	−0.045	0.727
<i>Eurotium chevalieri</i>	36	60	0.311	0.017
<i>Penicillium brevicompactum</i>	14	43	0.152	0.242
<i>Penicillium chrysogenum</i>	14	54	0.384	0.003
Group 2				
<i>Alternaria alternata</i>	44	57	0.433	0.001
<i>Aspergillus versicolor</i>	29	38	0.387	0.003
<i>Mucor racemosus</i>	30	57	0.437	0.001

MSQPCR indicates mold-specific quantitative polymerase chain reaction.

lied on culturing or spore counting to identify and quantify indoor molds. These methods do not allow easy identification to the species level, especially for *Penicillium* and *Aspergillus*. The Institute of Medicine report⁵ recommended “more rapid development of measurement methods for specific microorganisms that use DNA-based and other technology.” MSQPCR was developed to identify and quantify molds in a highly standardized and objective way.

Culture-based analysis of the dust samples did not give comparable results to the MSQPCR data (Table 3).

TABLE 4

Dust Samples Compared With Air Samples From 52 Study Homes After Analysis by Mold-Specific Quantitative Polymerase Chain Reaction

Mold Species	Dust Sample Detections	Air Sample Detections	Correlation Estimate (Spearman's rho)	P Value
Group 1				
<i>Aspergillus fumigatus</i>	0	7	NE	—
<i>Aspergillus penicillioides</i>	32	24	−0.167	0.233
<i>Aureobasidium pullulans</i>	0	37	NE	—
<i>Aspergillus sclerotiorum</i>	37	0	NE	—
<i>Aspergillus unguis</i>	52	45	0.212	0.130
<i>Aspergillus versicolor</i>	52	43	0.153	0.275
<i>Chaetomium globosum</i>	12	0	NE	—
<i>Cladosporium sphaerospermum</i>	24	13	0.340	0.015
<i>Eurotium amstelodami</i>	0	0	NE	—
<i>Penicillium brevicompactum</i>	47	10	0.200	0.153
<i>Penicillium corylophilum</i>	34	3	NE	—
<i>Penicillium group 2</i>	40	11	0.242	0.083
<i>Penicillium purpurogenum</i>	48	32	0.119	0.395
<i>Penicillium spinulosum</i>	52	46	0.183	0.190
<i>Penicillium variable</i>	40	10	0.067	0.632
<i>Paecilomyces variotii</i>	35	2	NE	—
<i>Scopulariopsis brevicaulis</i>	29	7	NE	—
<i>Scopulariopsis chartarum</i>	24	3	NE	—
<i>Stachybotrys chartarum</i>	24	1	NE	—
<i>Trichoderma viride</i>	37	1	NE	—
<i>Wallemia sebi</i>	43	18	0.173	0.216
Group 2				
<i>Alternaria alternate</i>	51	3	NE	—
<i>Acremonium strictum</i>	34	6	NE	—
<i>Aspergillus ustus</i>	31	14	0.111	0.428
<i>Cladosporium cladosporioides</i> 1	51	52	−0.073	0.602
<i>Cladosporium cladosporioides</i> 2	51	49	0.165	0.238
<i>Cladosporium herbarum</i>	52	47	0.125	0.372
<i>Epicoccum nigrum</i>	52	44	−0.153	0.273
<i>Mucor amphibiorum</i>	50	29	0.215	0.126
<i>Penicillium chrysogenum</i>	46	16	0.351	0.012
<i>Rhizopus stolonifer</i>	40	12	0.249	0.076

NE indicates not estimable.

Culture-based methods are limited because not all species grow on the media used and because fast-growing species can overtake the slow-growing species. In this study, only nine of the 33 species tested by MSQPCR could be compared because the others were not recorded in the culture data (Table 3). In addition, the concentrations were reported as generally much lower in the culture-based results. This is consistent with our earlier reports that culture-based analyses resulted in population estimates that were much lower than those estimates for the same samples as determined by MSQPCR analysis.¹² This may be because an aggregate of cells would be

individually counted with MSQPCR, but such aggregates would only yield a single colony on culturing. Also, cells may lose viability over time and because MSQPCR can measure fungal fragments that lack viability but still have DNA associated with them. However, even nonviable molds are still potentially allergenic and thus relevant to the study of asthma, that is, genomic DNA may reflect allergenic load better than viability. Another limitation of earlier studies was the types of samples collected.

Many studies of the mold populations indoors used short-term air sampling, usually 5 minutes or less.¹³ In this study, we used 6- to 8-hour sampling times. Even with these longer

sampling times, we found little correlation between the dust data and the air data, even when both samples were analyzed by MSQPCR.

Molds may be just one of many factors in asthma induction and exacerbation.¹⁴ In fact, the measurement of high populations of group 1 mold species in water-damaged homes may merely be an indicator for many other agents (bacteria, dust mites, and so on) and molds may not be linked to causality. However, molds do produce proteins that are allergens in experimental animals,^{15,16} so it may be that some molds are directly involved in asthma. The observation that *Scopulariopsis brevicaulis*, *Trichoderma viride*, *Penicillium crustosum* group, *Stachybotrys chartarum*, or *Wallemia sebi* are higher in asthmatics' homes may be coincidental. However, a closer look at the allergens produced by these species may be warranted.

The group 2 molds (*Alternaria alternata*, *Cladosporium cladosporioides* types 1 and 2, and *Epicoccum nigrum*) that were significantly lower in concentration in these water-damaged homes of asthmatic children in Cleveland have been described as "slightly xerophilic" species.¹⁸ Therefore, these group 2 species were likely displaced when their environment goes to a water-damaged condition. When that happens, their generally high concentrations in dust¹⁸ would go down by comparison to undamaged homes.

Asthma prevention measures like dust mite reduction and cockroach eradication have a proven value in reducing asthma symptoms.¹⁷ Water damage is not always obvious in a home, but testing dust for molds is a way to find out if there is a potential problem. The RMI values may be a useful tool for predicting the mold condition of a home. If group 1 molds are discovered, water-damage remediation and mold removal might be considered as part of the total prevention plan in an asthmatic child's home.

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