Respiratory pathophysiologic responses

Effect of environmental molds on risk of death from asthma during the pollen season

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Objective: *Many studies have noted an association of ambient aeroallergen levels with exacerbation of asthma. This study was undertaken to examine the relationship of aeroallergen levels with asthma-related mortality in Chicago.*
Methods: *The association of environmental aeroallergen levels with death caused by asthma among 5- to 34-year-olds in Chicago was examined for the period of 1985 through 1989. Logistic regression analysis was used to compare the probability of a death caused by asthma occurring on the basis of environmental tree, grass, or ragweed pollen and mold spore levels.*
Results: *Mean mold spore levels but not tree, grass, or ragweed pollen levels were significantly higher for days on which asthma-related death occurred than for days on which no deaths occurred ($z = 2.80$, $p < 0.005$). The odds of a death caused by asthma occurring on days with mold spore counts of 1000 spores per cubic meter or greater was 2.16 times higher (95% confidence interval = 1.31, 3.56, $p = 0.003$) than on days on which mold spore counts were less than 1000 spores per cubic meter. The association with mold spore levels remained significant on multivariate logistic regression with mold spore counts measured as a continuous variable and controlling for pollens, with the odds of an asthma-related death occurring being 1.2 times higher (95% confidence interval = 1.07-1.34) for every increase of 1000 spores per cubic meter in daily mold spore levels.*
Conclusion: *Although death caused by asthma also involves personal, social, and medical access factors, these data suggest that exposure to environmental molds may play a role in asthma-related mortality and should be considered in prevention strategies. (J ALLERGY CLIN IMMUNOL 1995;95:955-61.)*

Key words: *Asthma mortality, mold, pollens*

Trends in asthma attacks have been related to total aeroallergens,[1, 2] grass pollen,[3] and fungal spores.[4] Although a large proportion of asthma symptoms in children and young adults has been attributed to extrinsic allergenic exposure[5, 6] and several previous studies have shown seasonal variations in asthma hospitalizations that are parallel to seasonal trends in environmental pollen and mold levels,[1, 3, 6, 7] literature relating asthma deaths to specific allergen exposure has been scarce.[8-11] Seasonal trends in asthma-related mortality have been noted,[12, 13] but they have been less striking than trends in hospitalizations. Peaks in deaths have been noted in August in Scotland,[10] from July to September in England and Wales,[11] and from September to October in Ontario, Canada.[12] Previous studies have noted that mold spore levels peak in the fall, parallel with peaks in asthma-related deaths.[10, 11] Similarly, seasonal variation in airway function further suggests that aeroallergen

Abbreviations used:
DOA: Dead on arrival
NWS: National Weather Service

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Supported in part by the Education Resource Center of the University of Illinois at Chicago Occupational Health and Safety Center, National Institute for Occupational Safety and Health grant no. T15 OH07104-14, and the Asthmatic Children's Aid Foundation.
Received for publication Sept. 29, 1993; revised Sept. 14, 1994; accepted for publication Oct. 3, 1994.
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0091-6749/95 $3.00 + 0 **1/1/61007**

exposure could contribute to asthma-related deaths.[14, 15] In the United States peaks in deaths among persons aged 5 to 34 years occur from June to August before peaks for hospitalizations from September to November.[13]

Chicago has a particularly high asthma-related mortality rate among persons aged 5 to 34 years, without a striking seasonal variation.[16] Lack of seasonality, however, does not preclude pollen and mold effects. Numbers of deaths per year in Chicago are relatively small and may not allow for identification of subtle trends. In addition, mortality may be more influenced than morbidity by nonseasonal socioeconomic factors relating to access to appropriate medical therapy. This study was undertaken to examine the relationship of asthma-related deaths in Chicago between 1985 and 1989 among 5- to 34-year-olds with selected pollen and mold spore counts in an effort to identify environmental factors associated with asthma-related deaths, which might be amenable to intervention.

METHODS

Data concerning daily tree, grass, and ragweed pollens and combined viable and nonviable mold spores were collected for the period of 1985 through 1989 from Grant Hospital in Chicago. Collection was performed by a rotorod method in which two greased glass rods are rotated by forced air at timed intervals for a 24-hour period ending at 7 AM. Because collection was not performed on Sundays, the 48-hour average value documented on Mondays was converted to an average for Saturdays and Sundays in this study. The yearly interval of March 21 to October 31 is used in this analysis because pollen counts are taken only from March 21 to the time of the first fall frost.

The 7-month periods examined during the years 1985 through 1989 in the mortality analysis included 1130 days, 1044 (92.4%) of which had data for at least one pollen count and 1030 of which had complete pollen data (91.2%). Missing values were due to technical difficulties or to tests not performed during weekends or holidays.

Death certificate information was obtained from the Illinois Department of Public Health for 124 persons aged 5 to 34 years, with asthma as any cause of death, who were residents of Chicago during the period from 1985 through 1989. Information relevant to this study included age, race, sex, date of death, underlying and other causes of death, and hospital status at death (inpatient, outpatient, dead on arrival [DOA], other).

Deaths were categorized as pollen-season deaths (March 21 to October 31) or non-pollen-season deaths (remainder of year). These groups were compared by chi square analysis from 1985 through 1989 to determine whether any differences in the distribution of race, sex, or hospital status existed; and mean age of the two groups was compared by a t test.

Daily values for pollen counts were compared between two groups for the period of 1985 through 1989 during which complete data were quantitated: days during which no asthma-related deaths occurred ($n = 963$) and days during which asthma deaths occurred ($n = 67$). A total of 71 deaths occurred on 69 days (two deaths occurred on 1 day on two separate occasions), and complete pollen data were available for 67 days (97.1% complete).

Differences in the distribution of mold and pollen levels were examined between death and non-death days by using the National Weather Service (NWS) classification for ambient allergen levels (0 to 99, 100 to 499, 500 to 999, 1000+ grains or spores per cubic meter) and a chi square test. Distributions of pollens were heavily skewed, with 81% of days for tree pollens, 97% of days for ragweed pollens, and 98% of days for grass pollens being in the lowest NWS category (0 to 99 grains per cubic meter). Differences in mean pollen grain and mold spore levels between days during which death caused by asthma occurred and days on which such deaths did not occur were therefore tested with nonparametric Wilcoxon scores. Pearson correlation coefficients were computed to examine the relationship between pollens and mold spore counts.

Mold spore levels were further examined by the calculation of the difference between consecutive 24-hour mold spore counts to assess the association of absolute and percent change in levels with death caused by asthma in the above statistical analyses. Rates of change in mold spore levels, defined as the absolute and percent difference between consecutive days, were normally distributed and were examined by Student's t tests. Relationships of asthma-related deaths with both absolute mold counts and percent change in mold counts were measured by using counts on the day preceding, as well as on, the day of death.

Univariate and multivariate logistic regression models, with and without interaction terms, were constructed to determine whether the odds of death caused by asthma were significantly higher on days with higher pollen or mold spore levels. These methods were also applied to the data with up to a 4-day lag between mold spore exposure and date of death and for the day-to-day change in mold spore levels.

RESULTS

Approximately 57% of the 124 asthma-related deaths among Chicago residents aged 5 to 34 years from 1985 through 1989 were recorded during the pollen seasons of those years. No clear seasonal pattern is apparent for asthma-related deaths (Fig. 1) when examined as a 3-week moving average throughout the pollen seasons, although numbers of deaths may be too small to allow for patterns to be delineated. The difference in mean age of the patients who died in the pollen season compared

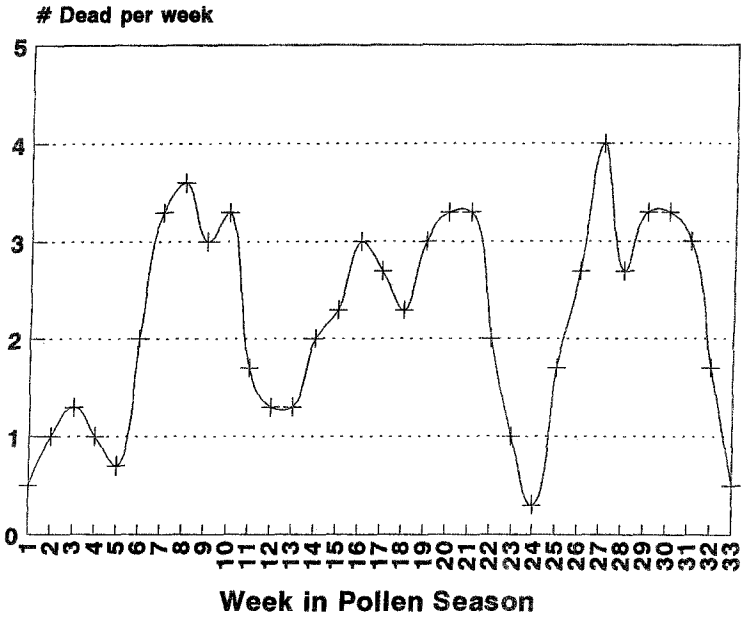

FIG. 1. Total weekly deaths caused by asthma among 5- to 34-year-olds in Chicago from March 21 to October 31, 1985 through 1989. Crosses represent 3-week moving average.

TABLE I. Comparison of death characteristics between pollen and non-pollen seasons, 1985 through 1989

	Season		chi square
	Pollen	Non-pollen	*p* value
Race			
White	15 (21.1%)	7 (13.2%)	0.37
Black	56 (78.9%)	46 (86.8%)	
Sex			
Male	32 (45.1%)	24 (45.3%)	1.00
Female	39 (54.9%)	29 (54.7%)	
Hospital status			
Inpatient	12 (17.1%)	12 (22.6%)	0.64
Outpatient	20 (28.6%)	17 (32.1%)	
DOA	34 (48.6%)	22 (41.5%)	
Other	4 (5.7%)	2 (3.8%)	
Autopsy			
Yes	52 (73.2%)	36 (67.9%)	0.45
No	19 (26.8%)	16 (30.2%)	

with those who died at other times of the year was insignificant (24.6 ± 7.5 and 22.8 ± 7.8, respectively; $t = 1.24$, $p = 0.22$), as were differences by season in the distribution of race, sex, autopsy status, and hospital status (Table I). A statistically insignificant decrease in the proportion of inpatient deaths and increase in patients who were DOA was observed for the pollen season when compared with the non-pollen season.

In Chicago, tree pollens are most prolific from March to June, grass pollens are present at relatively low levels from May through July, and ragweed pollens are prominent from August to the time of the first frost. Mold spores start to appear after snow and the ground thaw in spring and peak from July to October (Fig. 2). Pollen levels, in grains per cubic meter, ranged from 0 to 612 for grass, 0 to 792 for ragweed, 0 to 2502 for tree, and 0 to 18,735 spores per cubic meter for mold during the period of analysis.

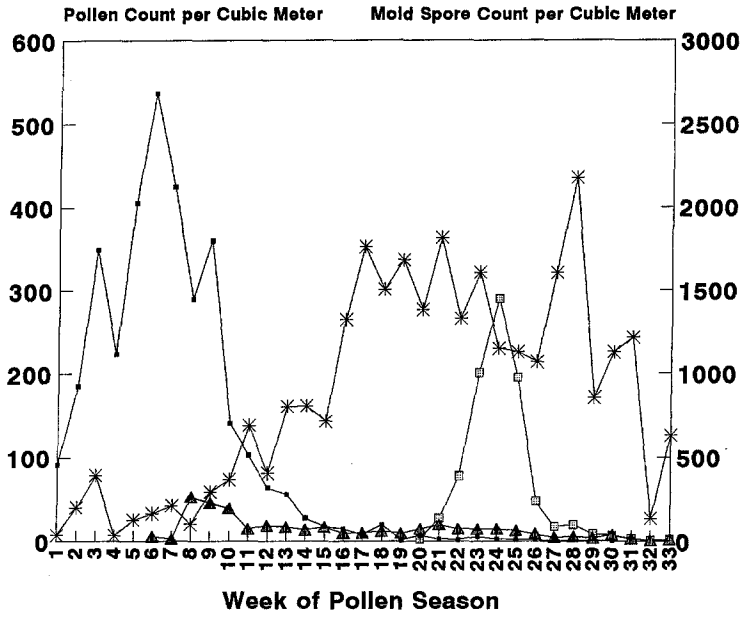

FIG. 2. Mean weekly pollen and mold spore levels from March 21 to October 31, 1985 through 1989. Tree (-■-); grass (-Δ-); mold (-*-); ragweed (-□-).

TABLE II. Mold spore and pollen grain Wilcoxon scores: March 21 to October 31, 1985 through 1989 comparing days with and without deaths

	Death (+)	Death (−)	Wilcoxon *p* value
Mold			
Mean	1533	905.0	0.005
n	67	965	
Tree			
Mean	92.11	99.79	0.42
n	67	970	
Grass			
Mean	19.00	11.93	0.08
n	67	967	
Ragweed			
Mean	14.48	31.12	0.37
n	67	969	

TABLE III. NWS categories of mold spore levels for death and non-death days, 1985 through 1989

Mold spore category	Period	
	Death (*n* = 67)	Non-death (*n* = 965)
0-99	15 (22.4%)	280 (29.0%)
100-499	11 (16.4%)	235 (24.4%)
500-999	9 (13.4%)	163 (16.9%)
1000+	32 (47.8%)	287 (29.7%)

Chi square = 9.65 (3 df); *p* value = 0.022.

Nonparametric analysis of mold spore and pollen levels for 1985 through 1989 was performed with Wilcoxon scores (Table II). Mean mold levels were significantly higher for days on which asthma-related deaths occurred than on days on which no asthma-related deaths occurred. Grass pollen, tree pollen, and ragweed pollen levels were not significantly different between days on which asthma-related deaths occurred and days on which such deaths did not occur. Similarly, when mold and pollen counts were compared between days preceding death and days preceding non-death days, mold levels ($z = 2.67$, $p = 0.008$) but not grass, tree, or ragweed pollen levels were significantly higher on days preceding deaths.

Comparison of the distributions of mold spore levels between days on which asthma-related deaths occurred and days on which such deaths did not occur were examined by a chi square analysis of NWS categories (Table III). The difference in the distribution of mold spore levels was statistically significant, with 47.8% and 29.7% of days on which asthma-related deaths did and did not occur, respectively, in the highest NWS mold category

TABLE IV. Logistic regression: Univariate and multivariate models assessing the association between mold and pollen levels and death caused by asthma, 1985 through 1989

	Univariate			Multivariate		
Exposure	**OR**	**95% CI**	***p* Value**	**OR**	**95% CI**	***p* Value**
Mold*	1.17	(1.05, 1.31)	<0.001	1.20	(1.07, 1.34)	0.001
Tree†	0.99	(0.90, 1.09)	0.82	1.00	(0.88, 1.11)	0.68
Grass†	1.34	(0.89, 2.02)	0.17	1.03	(0.67, 1,59)	0.13
Ragweed†	0.72	(0.45, 1.15)	0.18	0.63	(0.36, 1.08)	0.09

OR, Odds ratio; *CI,* confidence interval.
*Odds ratio reflects 1000-spore per cubic meter increase.
†Odds ratio reflects 100-grain per cubic meter increase.

(1000+ spores per cubic meter). Use of a 1-day lagged mold count for comparison between death and non-death days also revealed statistically significant differences, with 43.3% and 30.1% of days in the highest NWS mold category, respectively.

Analysis of dichotomous mold spore levels based on a cutoff at the highest NWS category (<1000, 1000+ spores per cubic meter) indicated an odds ratio of 2.16 (95% confidence interval = 1.31,3.56) for occurrence of asthma-related death on days with mold spore levels of 1000 spores per cubic meter or greater relative to all other days (chi square = 8.70, $p = 0.003$). Use of a 1-day lagged mold count in comparing the dichotomous distribution of mold spore levels on days preceding those with and without deaths caused by asthma produced a significant odds ratio of 1.77 for death caused by asthma the next day (95% confidence interval = 1.07,2.93: chi square = 4.48, $p = 0.034$). However, longer lags between high mold spore levels and the occurrence of a death caused by asthma were insignificant.

Pearson correlation coefficients were computed to examine the association between mold and pollen levels, primarily to determine whether the lack of significant univariate relationships between asthma-related deaths and pollen levels could have been confounded by inverse associations of mold levels with pollen counts. The magnitude of correlations ranged from 0.03 to 0.15, with only the mold-tree correlation being inverse ($r = -0.12$).

Results of the univariate logistic regressions produced results comparable to results of the Wilcoxon analysis and indicated a significantly greater odds of death caused by asthma on days with higher mold spore levels when measured continuously, with an odds ratio of 1.17 for each 1000-spore per cubic meter increase in mold spore levels (Table IV). Tree, grass, and ragweed pollens were not significant in univariate logistic models. Neither pollen levels nor mold levels were significantly associated with death caused by asthma when lagged relationships were examined with continuous data for the independent variables.

Multivariate main-effects models confirmed the univariate models, with mold levels but not pollen levels, remaining significantly related to deaths caused by asthma. Lagged mold spore and pollen levels were insignificant. The addition of interaction terms neither altered the direction of association between pollen or mold levels and probability of death caused by asthma nor provided a statistically significant effect on any of the main effect terms.

T tests for the difference in mean values for rates of change in mold spore levels between asthma-related death and non-death days were not significant for absolute change ($t = 1.21$, $p = 0.23$) or percent change ($t = 0.93$, $p = 0.35$). Similarly, logistic regression examining daily absolute and percent change in mean mold levels suggested that neither was significantly associated with death caused by asthma, although the odds of death caused by asthma with increasing magnitude of absolute change in mold spore level neared statistical significance.

DISCUSSION

Data from this study suggest that increased mold spore levels may contribute to deaths caused by asthma. The odds of dying of asthma on days with mold counts of 1000 spores per cubic meter or greater were 2.16 times higher than on days on which spore counts were less than 1000 per cubic meter. No consistent association was seen between asthma-related deaths and grass, ragweed, or tree pollens; and the increased risk of death associated with mold remained significant after control was applied for levels of these pollens.

The fact that the Midwest has particularly high levels of molds from early in spring through fall[17] may put residents in this region at increased risk of death caused by asthma during this time of year. The marked collinearity between mold counts and date renders separation of mold from other seasonally related risk factors—such as humidity, upper respiratory infection, temperature, and pollutants—difficult. Seasonal trends in asthma-related deaths (Fig. 2), however, were not very apparent in Chicago[16]; and trends in hospitalizations in the United States[13] more closely parallel ragweed counts than mold counts, suggesting that the association with mold seen in this study may be independent of other seasonal factors.

In addition to potential confounding by measured variables, this study is subject to limitations inherent in any ecologic study: confounding by other unknown factors related both to mold levels and deaths. Use of death certificate data did not permit assessment of other exposures, such as to animals or cigarette smoke, or to those in occupational settings, nor did it allow for the re-creation of events leading to the fatal attack. The fact that only one site was available for mold and pollen measurements and that all mold species were combined in a single daily value further limited the ability of this study to distinguish individual exposure levels. Approximately 6% of 6- to 24-year-old white subjects examined in NHANES II had positive skin test responses to *Alternaria* species, reactivity to which was associated with a fivefold increase in asthma prevalence.[18] Measurement of specific antibody levels to aeroallergens previously related to asthma, such as *Cladosporium* or *Alternaria,* which have been reported as among the most common molds in the Midwest,[17, 19, 20] would have been of interest but was not feasible retrospectively.

Exposure to indoor molds has also been related to asthma symptoms.[21-27] Studies of effects of indoor molds on asthma, however, have been limited by the large variability in levels over time and poor reproducibility in measurements.[28] Further, associations between indoor and outdoor mold levels have been inconsistent.[28]

The results of this study corroborate previous reports,[3, 8, 9, 18] which have shown associations of outdoor mold levels with asthma-related morbidity and mortality. A study of the relationship of hospital admissions for treatment of asthma with grass pollen in northern California noted a strong correlation with the intense grass pollination, which peaks in the spring, and a lesser but direct and significant correlation with mold levels.[3] A study of 256 asthma attacks among 169 children in Norway noted that more seasonal attacks occurred in children with *Cladosporium* allergy than in children without *Cladosporium* allergy.[21] Peaks in asthma-related mortality in the fall in Ontario,[12] Scotland,[10] and England and Wales[11] also parallel peaks in fungal spore counts. Increased asthma-related mortality in England during the summer months of the 1960s was significantly correlated with hyaline ascospore levels.[8] A more recent study of *Alternaria alternata* showed that 10 of 11 patients with asthma who had respiratory arrest in the fall and summer months had positive skin-puncture test results for sensitivity to this mold compared with only 31% of control subjects ($p < 0.001$).[9]

The mechanism of an effect of mold on asthma-related mortality is not clear. Some have suggested that mold spores may pass through the airways more easily than pollens because of their smaller size.[22] It is possible that molds interact with other allergens and environmental pollutants to increase risk. There did not appear to be significant interactions with the pollens measured in this study. Interactions with pollutants such as SO_2, ozone, and NO_2, however, were not measured.

The fact that a higher proportion of asthma-related deaths during the pollen season in this study were DOA, although statistically insignificant, suggests that a rapid change in exposure might result in fatal events. It is possible that in addition to high levels of mold spores, hypersensitivity to which would vary from person to person, relatively rapid temporal "spikes" in mold spore levels could lead to acute exacerbations of asthma and possibly death. The rapidity of change in pollen and mold counts is difficult to separate from absolute levels of the allergens, because absolute changes are higher on days with higher mold levels. Our examination of proportionate change in allergen levels on the day preceding death was an effort to control for absolute level of mold counts and did not reveal any significant associations with mortality, either on univariate or multivariate analysis. A larger number of events, more precise measurement of individual exposure, and more sophisticated methods such as time series analysis would be better suited to further explore this hypothesis.

The strength and independence of the relationship between mold level and asthma-related mortality in persons aged 5 to 34 years residing in Chicago and the consistency of this finding with previous reports suggests that mold may indeed be

a risk factor, not only for asthma symptoms but also for asthma-related deaths. Prevention of high indoor mold levels is feasible through reduction of humidity and dampness, adequate ventilation (particularly in the bathroom), and frequent cleaning. Prevention of exposure to outdoor levels is more complicated and generally involves avoidance procedures. Attention to NWS reports may be valuable in determining whether outdoor activity should be limited on high mold count days. Although death caused by asthma requires a complex interaction of host-intrinsic, social, and medical access factors, this study indicates that mold exposure also plays a role. If the results of this study are duplicated by other groups, it would behoove us to examine more closely the specific molds to which patients with asthma are exposed as potential targets of prevention strategies.

We are grateful for the time and efforts of Dr. Peter Orris and Dr. Patricia Kelleher of Cook County Hospital in reviewing this manuscript and for the epidemiologic advice of Dr. Daniel Hryhorczuk of the University of Illinois. We also thank Grant Hospital for providing aeroallergen data and Mr. Mark Peters of the Illinois Department of Public Health for his courteous assistance in obtaining mortality data.

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