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Original Contribution

Home Dampness and Molds as Determinants of Allergic Rhinitis in Childhood: A 6-Year, Population-based Cohort Study

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The authors assessed the relation between exposure to dampness and molds in dwellings and the development of allergic rhinitis in childhood in a 6-year, population-based prospective cohort study of 1,863 children aged 1–7 years at baseline in 1991 (follow-up rate, 77%) from Espoo, Finland. The studied exposures were history of water damage, presence of moisture and visible mold, and perceived mold odor in the home, based on parent-administered questionnaire. A total of 246 (13.2%) children developed physician-diagnosed allergic rhinitis during the study period, resulting in an incidence rate of 440 cases per 10,000 person-years (95% confidence interval (CI): 387, 499). In logistic regression adjusting for confounding, any mold or dampness exposure indicator at baseline (adjusted odds ratio = 1.55, 95% CI: 1.10, 2.18), at follow-up (adjusted odds ratio = 1.62, 95% CI: 1.21, 2.18), or both (adjusted odds ratio = 1.96, 95% CI: 1.29, 2.98) was an important independent determinant of the risk of allergic rhinitis. Of the individual indicators, water damage and moisture on the surfaces were consistent determinants of allergic rhinitis. The results of this cohort study, which assessed exposure before the onset of allergic rhinitis, strengthen considerably the evidence of the role of indoor dampness problems as determinants of allergic rhinitis in children.

air pollution, indoor; fungi; housing; rhinitis, allergic, perennial

Abbreviation: CI, confidence interval.

There is substantial epidemiologic evidence of an association between dampness and mold problems in the home and various respiratory problems, including upper respiratory tract symptoms, chronic wheezing and cough, and development of asthma in children (1–6). Epidemiologic studies conducted in different climates have suggested that home dampness and molds may also increase the risk of allergic rhinitis in children (7–16), but this evidence is based mainly on cross-sectional studies (7–12, 15, 16), where selection bias and information bias, as well as establishment of temporality between exposure and outcome, constitute problems. In our systematic Medline search, we identified only 2 cohort studies (13, 14) on the role of home dampness and mold exposure in the development of allergic rhinitis, both of which provided suggestive, but inconclusive results. As dampness and mold problems are relatively common indoor problems in many parts of the world, further studies are needed on their potential role in the development of allergic rhinitis. For example, the report from the Institute of Medicine Committee on Damp Indoor Spaces and Health estimated that at least 20% of homes in the United States have dampness problems or visible mold (2).

We assessed longitudinally the relations between exposure to molds and dampness in dwellings and the risk of developing allergic rhinitis in childhood up to 14 years of age. Further, we explored the role of different dampness and mold exposure indicators as determinants of allergic rhinitis. In addition, we tested a hypothesis that the joint effect of genetic propensity to allergic rhinitis and indoor exposures to dampness and molds is greater than expected on the basis of their independent effects. We used parental history of allergic diseases as a measure of genetic propensity to allergic rhinitis.

MATERIALS AND METHODS

Study population

The source population included all children living in the city of Espoo in Finland who were born between January 1, 1984, and December 31, 1989. Espoo is an urban-suburban municipality with a population of 244,353 (December 31, 2009), located across the western border of the capital city, Helsinki. A parent-administered baseline questionnaire was distributed in March 1991 to a random sample of children of the source population (17). The baseline study population included a total of 2,568 children whose parents completed the questionnaire (response rate, 80.3%). In March 1997, we conducted a 6-year follow-up survey directed at all the members of the cohort (3). A completed questionnaire was received from parents of 1,984 children (77.3% of the baseline study population). A total of 1,863 children free of allergic rhinitis at baseline constituted the study population.

Data collection

In the baseline survey, parents or other guardians were asked about the child's personal characteristics, health, details of the environment, and other relevant factors (Table 1). The questions on respiratory health were partly from the 1978 American Thoracic Society–Division of Lung Disease questionnaire for children translated into Finnish and Swedish, the 2 official languages of Finland (18). Rather than making a direct translation, the questions were modified with the aid of 2 pulmonary physicians to correspond to the everyday use of the languages (17). The follow-up survey included questions about health and environment identical to those at baseline, as well as some more detailed questions about the environment.

Health outcome

The outcome of interest was the development of allergic rhinitis (incident cases) during the study period. We included only children who did not have physician-diagnosed rhinitis at baseline ($n = 1{,}863$) for these analyses. Among these, we identified those who indicated a history of or current physician-diagnosed allergic rhinitis in the 6-year follow-up survey. The allergic rhinitis could have been diagnosed as the result of any allergies and was not limited to allergies to fungal allergens only.

Genetic and environmental determinants of interest

Parental atopy was defined as a history of maternal or paternal allergic rhinitis or asthma. Information on parental allergic rhinitis and asthma was collected in the baseline questionnaire.

We used 4 indicators of exposure defined from the answers to the following structured questions at baseline and at follow-up.

Mold odor. "Have you perceived mold odor in your dwelling during the past 12 months?" (No; yes, almost daily; yes, 1–3 days a week; yes, 1–3 days a month; yes, less often.)

Visible mold. "Have you ever had visible mold in your dwelling?" (No; yes, during the past 12 months; yes, only earlier.)

Moisture. "Have you ever had wet spots on the ceilings, floors, or walls of the occupied rooms in your dwelling?" (No; yes, during the past 12 months; yes, only earlier.)

Water damage. "Have you ever had water damage in your dwelling?" (No; yes, during the past 12 months; yes, only earlier.)

Any exposure indicator. The presence of any of the 4 exposure indicators defined above.

The follow-up survey included similar questions about the presence of the 4 exposure indicators.

From these data, we constructed dichotomous exposure indicators (yes, exposure during the past 12 months or earlier, vs. no) measured at baseline, at follow-up, or at both baseline and follow-up. Exposures during the past 12 months and earlier were combined in the present analyses.

Statistical methods

First, we estimated the occurrence of allergic rhinitis during the 6-year study period and indicators of exposure to dampness and molds. In the crude analysis, the cumulative incidence ratio of the occurrence of allergic rhinitis in relation to the exposure indicators was estimated. Then, we estimated adjusted odds ratios applying multivariate logistic regression analysis. The effect estimates were adjusted for the covariates described in Table 1.

Second, we studied the joint effects of parental atopy and the 2 most important exposures, namely, "water damage" and "moisture on the surfaces," on the risk of allergic rhinitis. We compared the risk (R) of developing allergic rhinitis in 4 exposure categories: 1) no parental atopy and no exposure (R_{00}, reference category); 2) parental atopy and no exposure (R_{10}); 3) no parental atopy and exposure (R_{01}); and 4) parental atopy and exposure (R_{11}). On an additive scale, the interaction (IA) of 2 factors was quantified by calculating whether the risk is more than expected on the basis of the independent effects of these factors:

$$\mathrm{IA} = (R_{11} - R_{00}) - (R_{10} - R_{00}) - (R_{01} - R_{00}).$$

Then, we used the odds ratio as a measure of effect. To assess the joint effect of parental atopy and exposure, we calculated odds ratios contrasting each of the 3 exposure categories to the reference category (R_{00}). Estimates for the independent effects of parental atopy and exposure and for their joint effect were derived from the same logistic regression model adjusting for the covariates.

RESULTS

Study population

Characteristics of the baseline study population, those lost to follow-up, and the 6-year cohort are provided in

Table 1. Personal and Environmental Characteristics of the Baseline Study Population, Those Lost to Follow-up, and the 6-Year Cohort, The Espoo Cohort Study, 1991–1997

Characteristic at Baseline	Baseline		Lost to Follow-up		6-Year Cohort	
	No.	%	No.	%	No.	%
No. of children	2,568	100	584	22.7	1,984	77.3
Age, years						
1	424	16.5	100	17.1	324	16.3
2	405	15.8	104	17.8	301	15.2
3	410	16.0	92	15.8	318	16.0
4	400	15.6	67	11.5	333	16.8
5	415	16.2	101	17.3	314	15.8
6–7	514	20.0	120	20.6	394	19.9
Gender						
Boy	1,258	49.0	275	47.1	983	49.6
Girl	1,310	51.0	309	52.9	1,001	50.5
Single parent or guardian						
Yes	183	7.1	53	9.1	130	6.6
No	2,385	92.9	531	90.9	1,854	93.5
Highest level of parental education						
Nonprofessional	498	19.5	129	22.3	369	18.7
Trade school	663	25.9	140	24.2	523	26.5
College or university	1,395	54.6	310	53.5	1,085	54.9
Breastfeeding						
<4 months	555	21.6	158	27.1	397	20.0
4–<8 months	670	26.1	159	27.2	511	25.8
8 months	1,343	52.3	310	45.7	1,076	54.2
Maternal smoking in pregnancy						
Yes	349	13.6	100	17.1	249	12.6
No	2,219	86.4	484	82.9	1,735	87.5
Exposure to secondhand tobacco smoke						
Yes	277	10.8	80	13.7	197	9.9
No	2,291	89.2	504	86.3	1,787	90.1
Gas stove						
Yes	86	3.4	24	4.1	62	3.1
No	2,469	96.6	556	95.9	1,913	96.9
Hairy/feathery pets						
Yes	480	18.7	113	19.4	367	18.5
No	2,088	81.3	471	80.7	1,617	81.5
Type of day care						
100% home	940	36.6	210	36.0	730	36.8
100% family	513	20.0	119	20.4	394	19.9
100% center	252	9.8	56	9.6	196	9.9
Combinations	863	33.6	139	34.1	664	33.5

Table 1. The 6-year cohort did not differ substantially from the baseline study population, as none of the differences was statistically significant (chi-square and Fisher's exact test). A total of 246 children (13.2%) developed allergic rhinitis during the follow-up period. The estimated incidence rate was 440 per 10,000 person-years (95% confidence interval (CI): 387, 499). Table 2 compares the characteristics of the exposed group and the reference

Table 2. Comparison of Personal and Environmental Characteristics of the Exposed and Reference Groups ($N = 1,863$), The Espoo Cohort Study, 1991–1997

Characteristic at Baseline	Exposed Group		Reference Group		Statistical Significance
	No.	%	No.	%	
No. of children	366	19.6	1,497	80.4	
Age, years					$\chi^2_5 = 4.05$; $P = 0.54$
1	52	14.2	266	17.8	
2	60	16.4	229	15.3	
3	68	18.6	236	15.7	
4	61	16.7	240	16.0	
5	56	15.3	242	16.2	
6	69	18.8	284	19.0	
Gender					$\chi^2_1 = 0.003$; $P = 0.95$
Boy	183	50.0	746	49.8	
Girl	183	50.0	751	50.2	
Single parent or guardian					$\chi^2_1 = 0.108$; $P = 0.74$
Yes	22	6.0	97	6.5	
No	344	94.0	1,400	93.5	
Highest level of parental education					$\chi^2_2 = 2.45$; $P = 0.29$
Nonprofessional	75	20.6	274	18.4	
Trade school	102	28.0	384	25.7	
College or university	187	51.4	834	55.9	
Breastfeeding					$\chi^2_2 = 1.70$; $P = 0.43$
<4 months	73	20.0	295	19.7	
4–<8 months	104	28.4	379	25.3	
8 months	189	51.6	823	55.0	
Maternal smoking in pregnancy					$\chi^2_1 = 8.41$; $P = 0.04$
Yes	62	16.9	170	11.4	
No	304	83.1	1,327	88.6	
Exposure to secondhand tobacco smoke					$\chi^2_1 = 3.08$; $P = 0.08$
Yes	46	12.6	142	9.5	
No	320	87.4	1,355	90.5	
Gas stove					$\chi^2_1 = 1.87$; $P = 0.17$
Yes	15	4.1	41	2.8	
No	349	95.9	1,449	97.2	
Hairy/feathery pets					$\chi^2_1 = 4.39$; $P = 0.04$
Yes	83	22.7	268	17.9	
No	283	77.3	1,229	82.1	
Type of day care					$\chi^2_3 = 1.04$; $P = 0.79$
100% home	129	35.3	567	37.9	
100% family	74	20.2	299	20.0	
100% center	39	10.7	144	9.6	
Combinations	124	33.8	487	32.5	

group. The exposed group comprised children with any reported indicator of home dampness or mold at baseline (19.6%). The exposed children slightly more often had maternal smoking in pregnancy (16.9% vs. 11.4%; $\chi^2_1 = 8.41$; $P = 0.04$) and exposures to secondhand tobacco smoke (12.6% vs. 9.5%; $\chi^2_1 = 3.08$; $P = 0.08$) and hairy or feathery pets (22.7% vs. 17.9%; $\chi^2_1 = 4.39$; $P = 0.04$) in the home compared with the reference group.

Table 3. Cumulative Incidence of Allergic Rhinitis in Different Exposure Categories and Odds Ratios Contrasted to the Reference Category of No Exposure and Adjusted for Other Exposures and Confounding in Logistic Regression Analysis, The Espoo Cohort Study, 1991–1997

Exposure	Size of the Group, no.	No. of New Allergic Rhinitis Cases	Cumulative Incidence	Crude Odds Ratio	95% Confidence Interval	Adjusted Odds Ratio[a]	95% Confidence Interval
Total no.	1,863	246	0.1320				
No exposure (reference)	1,139	132	0.1159	1.00		1.00	
Water damage							
At baseline	100	23	0.2300	2.28	1.32, 3.82	2.40	1.43, 4.03
At follow-up	172	36	0.2093	2.02	1.30, 3.08	2.06	1.35, 3.13
Both	39	13	0.3333	3.81	1.75, 7.91	3.83	1.87, 7.83
Moisture on the surfaces							
At baseline	280	44	0.1571	1.42	0.96, 2.08	1.57	1.08, 2.30
At follow-up	432	76	0.1759	1.63	1.18, 2.23	1.73	1.27, 2.38
Both	130	22	0.1692	1.55	0.90, 2.58	1.76	1.06, 2.92
Visible mold							
At baseline	80	9	0.1125	0.97	0.41, 2.00	1.06	0.51, 2.21
At follow-up	204	38	0.1863	1.75	1.14, 2.63	1.98	1.32, 2.99
Both	37	1	0.0270	0.21	0.01, 1.28	0.24	0.03, 1.79
Mold odor							
At baseline	51	5	0.0980	0.83	0.25, 2.13	0.94	0.36, 2.45
At follow-up	160	23	0.1438	1.28	0.76, 2.09	1.45	0.89, 2.37
Both	18	3	0.1667	1.53	0.28, 5.49	1.88	0.52, 6.78
Any exposure							
At baseline	366	58	0.1585	1.44	1.01, 2.03	1.55	1.10, 2.18
At follow-up	546	92	0.1685	1.55	1.14, 2.08	1.62	1.21, 2.18
Both	188	36	0.1915	1.81	1.17, 2.74	1.96	1.29, 2.98

[a] Logistic regression controlling for other exposures, age, gender, duration of breastfeeding, parents' highest education, single parent or guardian, maternal smoking in pregnancy, exposure to secondhand tobacco smoke, gas cooking, the presence of hairy or feathery pets at home, and type of day care.

Effects of exposure to dampness and mold problems

Table 3 presents the cumulative incidence of developing allergic rhinitis during the 6-year study period according to the 4 exposure indicators at baseline, the follow-up survey, and at both baseline and follow-up, as well as the odds ratios contrasted to the reference category of no exposure. The overall risk of developing allergic rhinitis during the 6 years was 0.1320. The risk was substantially elevated among children living in a home with water damage measured at baseline (incidence, 0.2300) or follow-up (incidence, 0.2093), with adjusted odds ratios of 2.40 (95% CI: 1.43, 4.03) and 2.06 (95% CI: 1.35, 3.13), respectively. The risk was highest (incidence, 0.3333) in children living in a home with water damage at both the baseline and follow-up, with an adjusted odds ratio of 3.83 (95% CI: 1.87, 7.83). A similar pattern was observed for moisture on the surfaces. Visible mold at the baseline did not predict development of allergic rhinitis during the study period, but when measured at the follow-up, it increased the risk of developing allergic rhinitis with an adjusted odds ratio of 1.98 (95% CI: 1.32, 2.99). The risk of allergic rhinitis was not related to perceived mold odor at the baseline, but the risk estimates were elevated related to exposure at the follow-up and in relation to exposure at both the baseline and follow-up, although the confidence intervals were wide because of relatively low prevalence of this exposure indicator. The effect estimate for any dampness or mold exposure indicator at the baseline was 1.55 (95% CI: 1.10, 2.18), at the follow-up 1.62 (95% CI: 1.21, 2.18), and at both the baseline and follow-up 1.96 (95% CI: 1.29, 2.98).

Joint effects of parental atopy and exposure to water damage and moist surfaces

Table 4 shows the risks (cumulative incidences) of asthma in 4 categories, representing the reference category, the independent effects of parental atopy and exposure to water damage, and their joint effect. In logistic regression adjusting for confounding, the risk of allergic rhinitis was related to parental atopy alone with an adjusted odds ratio of 2.23 (95% CI: 1.63, 3.05). The effect estimate for exposure to water damage among children of nonatopic parents was 1.34 (95% CI: 0.55, 3.29). In children with both atopic heredity and exposure to water damage, the adjusted odds ratio of allergic rhinitis was 5.35 (95% CI: 2.22, 12.85). The adjustment did not change the odds ratios substantially, and therefore it was reasonable to assess interaction on an additive scale on the basis of crude cumulative incidences. The

Table 4. Independent and Joint Effects of Parental Atopy and Exposure to Water Damage at Baseline on the Incidence of Allergic Rhinitis Between 1 and 14 Years of Age, The Espoo Cohort Study, 1991–1997

Exposure	Size of the Group, no.	No. of New Allergic Rhinitis Cases	Cumulative Incidence	Crude Odds Ratio	95% Confidence Interval	Adjusted Odds Ratio[a]	95% Confidence Interval
No parental atopy, no exposure	991	92	0.0928	1.00		1.00	
Parental atopy, no exposure	506	96	0.1897	2.29	1.68, 3.12	2.23	1.63, 3.05
No parental atopy, water damage	48	6	0.1250	1.40	0.58, 3.37	1.34	0.55, 3.29
Parental atopy, water damage	25	9	0.3600	5.50	2.36, 12.79	5.35	2.22, 12.85

[a] Logistic regression controlling for other exposures, age, gender, duration of breastfeeding, parents' highest education, single parent or guardian, maternal smoking in pregnancy, exposure to secondhand tobacco smoke, gas cooking, presence of hairy or feathery pets at home, and type of day care.

excess risk of allergic rhinitis was substantially greater among exposed children of parents with atopy ($0.2672 = 0.3600 - 0.0928$) than expected from the excess risk of unexposed children of parents with atopy ($0.0969 = 0.1897 - 0.0928$) and exposed children of nonatopic parents ($0.0322 = 0.1250 - 0.0928$). Thus, the interaction on an additive scale was $0.1381 = [(0.3600 - 0.0928) - (0.1897 - 0.0928) - (0.1250 - 0.0928)]$.

Table 5 shows that the joint excess risk from parental atopy and exposure to moisture on the surfaces ($0.1092 = 0.2020 - 0.0928$) was of similar magnitude as the sum (0.1367) of the independent excess risks for parental atopy ($0.0969 = 0.1897 - 0.0928$) and exposure to moisture on the surfaces ($0.0398 = 0.1326 - 0.0928$) and, thus, there was no evidence of interaction between parental atopy and moisture on surfaces.

DISCUSSION

In our population-based, prospective cohort study, children living in homes with any dampness or mold exposure problem at baseline had close to a 50% increased risk of developing allergic rhinitis in the following 6 years and an almost 100% increased risk if there was also such exposure at follow-up. Thus, there was evidence suggesting a dose-dependent increase in the risk, with a higher risk of allergic rhinitis related to a longer duration of exposure to indoor dampness and mold problems. Water damage was the strongest determinant of allergic rhinitis: For water damage measured at baseline, the effect estimate showed over 100% increased risk and, for water damage at both baseline and follow-up, the excess risk was over 200%. There was also evidence that the relation between water damage exposure and the risk of allergic rhinitis was stronger among the children of atopic parents. Moisture on the surfaces had a similar exposure-effect pattern, but there was no indication of any interaction between exposure and parental atopy. The risk of developing allergic rhinitis was related to the presence of visible mold and mold odor reported at the follow-up only. Lack of effect of visible mold or mold odor at baseline was somewhat surprising but could be explained by the fact that such exposure is probably an indicator of a more advanced problem than damp spots on surfaces and, thus, the problem is more likely to have been repaired already in the beginning of the follow-up period, thus not affecting development of rhinitis during the follow-up. Another explanation could be that other dampness-related causal agents than molds underlie allergic rhinitis at an early age; for example, factors such as mites and chemicals emitted from the damp surfaces may play a role. In addition, the number of cases was rather small in the groups with visible mold and mold odor at baseline, which makes these risk estimates less precise than the estimates for water damage and moisture on the surfaces.

Validity of results

In a prospective cohort study, several threats to validity are weaker compared with cross-sectional and retrospective cohort studies. The relatively high follow-up rate of 77%, as

Table 5. Independent and Joint Effects of Parental Atopy and Exposure to Moisture on the Surfaces at Baseline on the Incidence of Allergic Rhinitis Between 1 and 14 Years of Age, The Espoo Cohort Study, 1991–1997

Exposure	Size of the Group, no.	No. of New Allergic Rhinitis Cases	Cumulative Incidence	Crude Odds Ratio	95% Confidence Interval	Adjusted Odds Ratio[a]	95% Confidence Interval
No parental atopy, no exposure	991	92	0.0928	1.00		1.00	
Parental atopy, no exposure	506	96	0.1897	2.29	1.68, 3.12	2.23	1.63, 3.05
No parental atopy, moisture on surfaces	181	24	0.1326	1.49	0.92, 2.31	1.64	1.00, 2.67
Parental atopy, moisture on surfaces	99	20	0.2020	2.47	1.45, 4.23	2.51	1.45, 4.34

[a] Logistic regression controlling for other exposures, age, gender, duration of breastfeeding, parents' highest education, single parent or guardian, maternal smoking in pregnancy, exposure to secondhand tobacco smoke, gas cooking, presence of hairy or feathery pets at home, and type of day care.

well as similar distributions of exposure indicators and other characteristics of the study population at baseline and in the 6-year cohort, minimized the role of bias related to losses to follow-up. The prospective study design minimizes information bias, especially when focusing on the effects of exposures measured at baseline before the onset of the studied outcome.

A limitation of the present study was that exposure assessment was based on parental reporting rather than objective measurements. On the other hand, even today there is no consensus on any one exposure measure that would be the best from the health effects point of view. Thus, an approach commonly used in health effect studies of dampness and mold problems is to ask about the sources of dampness-related exposures, that is, water damage, moist spots on surfaces, visible mold, and mold odor (19). There is some evidence suggesting that occupants themselves tend to underestimate exposure to dampness and molds compared with a visual observation by a trained person (19–22). Such a tendency in reporting would lead to underestimating the risk. On the other hand, some strengths of exposure assessment in our study balance the limitations due to the lack of objective measurements. First, in the main analyses, the exposure information was collected at baseline, that is, before the onset of allergic rhinitis, and therefore any bias due to awareness of the disease was avoided. Second, when the baseline data collection took place in 1991, there was no general awareness of potential adverse health effects related to indoor dampness and mold problems. Thus, any error in our exposure assessment at baseline is likely to be random, so it would lead to underestimation of the effect. There was more knowledge and awareness of the potential health effects of indoor dampness and mold problems during the follow-up data collection (23), but the media coverage in Finland focused largely on workplace and public building dampness and mold problems.

We focused on reported physician-diagnosed allergic rhinitis rather than performing clinical examinations for the purposes of the study. This is a potential source of misclassification that is likely to be random, that is, not related to the exposure of interest at baseline, and thus it could also lead to underestimation of the effect. It should be noted that the allergic rhinitis could be due to any allergies and was not limited to only fungal allergies. Although in a study of adult-onset asthma immunoglobulin E antibodies to molds or mites were related to highly increased odds ratios of asthma, such specific antibodies were detected only in a rather small proportion (1%–15%) of cases (24). Thus, other mechanisms are also likely to be involved. For example, an inflammatory reaction in the airways due to irritant or toxic agents related to indoor dampness and mold problems (19, 20, 25) could increase permeability of the airway epithelium to any allergen.

We were able to take into account most of the known potential confounders related to individual characteristics and other environmental exposures in the logistic regression analysis, where most of the known determinants were included. However, dampness problems may also be related to other indoor environmental factors than molds, such as dust mites. Although the occurrence of dust mites is rather small in the Finnish climate, part of the allergic rhinitis related to water damage and moisture on the surfaces may be explained by dust mites rather than by molds. Dampness problems may also indicate a low ventilation rate and consequently increased levels of indoor pollutants from interior surfaces or human activities, as discussed later in more detail. Dampness and mold problems were common also in Finnish day-care centers (26). In a random sample of 30 day-care centers in the city of Espoo in 1991, dampness was found to be common: Water damage had taken place in 70% of the centers, and workers perceived mold odor in 17% of them. In our study, we were able to adjust for the type of day care, but not for dampness and mold problems in the day-care centers, as the parents might not be aware of such problems in day-care centers. However, any exposure to dampness and mold problems in the day-care center was not likely to be related to such problems in the home and, thus, could not be responsible for any systematic error.

Synthesis with the previous knowledge

The Institute of Medicine Committee on Damp Indoor Spaces and Health published a comprehensive review of publications by the end of 2003 (2). The Committee judged separately the effects of ''damp indoor environments'' and those of ''mold or other agents.'' The Committee found no sufficient evidence for a causal relation between either of these 2 exposures and health effects. The Committee found sufficient evidence for an association between dampness and upper respiratory tract (nasal and throat) symptoms, cough, wheeze, and asthma symptoms in sensitized persons. There was sufficient evidence of an association between exposure to molds and upper respiratory symptoms, cough, wheeze, and asthma symptoms in sensitized asthmatic persons, as well as hypersensitivity pneumonitis. Evidence on the development of asthma was deemed limited or suggestive for both dampness and mold exposures at the time. The role of dampness and molds in the development of allergic rhinitis was not included, apparently because of an insufficient number of studies on this outcome at the time. A recent meta-analysis on the respiratory health effects of dampness and mold in homes assessed the risk of upper respiratory tract symptoms based on 13 studies including children or adults, the central odds ratio estimate being 1.70 (95% CI: 1.44, 2.00), but they did not provide any risk estimate for rhinitis specifically (5).

The present study strengthens the evidence that home dampness due to water damage or other moisture problems increases the risk of allergic rhinitis (7–16). The history of water damage and moisture on the surfaces at baseline predicted future development of allergic rhinitis. Water damage and moisture in combination with perceived molds (i.e., any dampness or mold exposure at baseline) increased the risk further, as well as the presence of these exposure indicators at both the baseline and the follow-up. Use of baseline exposure indicators is likely to provide valid effect estimates, whereas use of follow-up exposure indicators is more susceptible to similar types of limitations to validity as present in cross-sectional studies. On the other hand, also analyzing

exposure at both baseline and follow-up gives an estimate of effect related to exposure of longer duration.

Home dampness increases exposure to allergens in several ways that may play a role in the development of allergic rhinitis as well as asthma. Home dampness enhances the growth of dust mites and fungi, promotes bacterial growth, and increases the number of cockroaches (19, 24, 25). The latter are extremely rare in Finland. In addition to fungi and their spores, toxins emitted by molds or bacteria, as well as their cell wall components, including beta-glucan, may induce an inflammatory reaction in the airways, which could enhance allergic responses to other allergens (19, 25). Moisture may also result in increased chemical emissions from building materials, which may play a role in the causation of allergies. Recently, a combination of dampness problems and the presence of polyvinyl chloride flooring was shown to exert synergistic effects on the risk of asthma and allergic rhinitis, which were explained by harmful degradation product emissions (27).

New experimental research results support the epidemiologic evidence on the relation between exposure to dampness and molds and development of allergic rhinitis. Stark et al. (28) showed in controlled human experiments that exposure to *Aspergillus fumigatus* induces airways inflammation in the upper respiratory tract. Inflammation in the upper airways was measured as increased cytokine levels in nasal lavage fluid, the most distinct response seen in interleukin-1β levels.

Conclusions

The results of this population-based cohort study, which assessed exposure before the onset of allergic rhinitis, considerably strengthen the evidence of the effects of exposure to indoor dampness problems on the development of allergic rhinitis in children. There is some support for the role of molds as determinants of allergic rhinitis. Such indoor exposures should be remembered as potential causal agents when preventing, diagnosing, and treating allergic rhinitis.

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