

# Prolonged exposure to damp and moldy workplaces and new-onset asthma

Kirsi Karvala · Elina Toskala · Ritva Luukkonen ·  
Jukka Uitti · Sanna Lappalainen · Henrik Nordman

Received: 26 May 2010 / Accepted: 1 July 2011  
© Springer-Verlag 2011

## Abstract

**Purpose** Epidemiological evidence shows that indoor dampness is associated with respiratory symptoms, the aggravation of preexisting asthma, and the development of new-onset asthma. Follow-up studies indicate that symptoms compatible with asthma constitute risk factors for the future development of asthma. The aims of the study were (1) to assess whether asthma-like symptoms (cough, dyspnea, and wheeze) that occur in relation to exposure to damp and moldy work environments lead to the later development of asthma and (2) to assess the importance of continued exposure to indoor dampness and molds at work in the development of asthma.

---

K. Karvala (✉)  
Occupational Medicine Team, Finnish Institute of Occupational Health (FIOH), Topeliuksenkatu 41 a A, 00250 Helsinki, Finland  
e-mail: kirsi.karvala@ttl.fi

E. Toskala  
Control of Hypersensitivity Diseases Team, Finnish Institute of Occupational Health (FIOH), Helsinki, Finland

R. Luukkonen  
Statistics and Health Economics Team, Finnish Institute of Occupational Health (FIOH), Helsinki, Finland

J. Uitti  
Occupational Medicine Team, Finnish Institute of Occupational Health (FIOH), Tampere, Finland

S. Lappalainen  
Developing Indoor Environments Team, Finnish Institute of Occupational Health (FIOH), Helsinki, Finland

H. Nordman  
Occupational Medicine Team, Finnish Institute of Occupational Health (FIOH), Helsinki, Finland

**Methods** We followed 483 patients with asthma-like symptoms related to damp workplaces but without objective evidence of asthma in baseline examinations. The development of asthma and present work conditions were established with the use of a questionnaire 3–12 years later. **Results** A total of 62 patients (13%) reported having developed asthma during the study period. Continued exposure to a damp or moldy environment was associated with a more than fourfold increase in the risk of asthma (odds ratio 4.6, 95% confidence interval 1.8–11.6). Working in a non-remediated environment at follow-up was the strongest risk factor for developing asthma. The remediation of damp buildings seemed to be associated with a decrease in the risk of asthma.

**Conclusions** The results indicate that exposure at work to dampness and molds is associated with the occurrence of new-onset asthma. Exposed workers suffering from asthma-like symptoms represent a risk group for the development of asthma. The risk appears especially high if the exposure continues. Due to inherent weaknesses of patient series, the findings need corroborative studies.

**Keywords** Asthma · Asthma-like symptoms · Dampness · Mold · Occupational exposure · Remediation

## Introduction

International expert groups have recently concluded that there is sufficient epidemiological evidence indicating an association between factors related to indoor dampness and several respiratory health effects, including the development of asthma, the exacerbation of preexisting asthma, and current asthma (Bornehag et al. 2001, 2004; IOM 2004; WHO 2009). Dampness and mold in the living area

of houses has been found to have a dose–response relationship to the development of asthma in infants and children (Pekkanen et al. 2007). In Finland, indoor dampness has been a particularly common indoor health problem since the 1980s (Nevalainen et al. 1998). Occupational asthma has been attributed to exposure to indoor dampness and molds since the early 1990s. Since 2002, such exposure has been the most frequently reported cause of occupational asthma in the national register of occupational diseases (Piipari and Keskinen 2005; Karvala et al. 2010).

The epidemiological evidence also indicates that exposure to indoor dampness and molds is associated with respiratory symptoms: cough, wheeze, and dyspnea (Bornehag et al. 2001, 2004; IOM 2004; WHO 2009). These symptoms, as well as chest tightness in the absence of lung function disturbances compatible with asthma, have been called asthma-like inflammation and even pre-asthma in some studies (Haahtela 1999; Ryttilä et al. 2008). Patients suffering from this condition run a markedly increased risk of developing asthma (Puolijoki and Lahdensuo 1987; Remes et al. 1998). In particular, recurrent or persistent cough has been considered a harbinger of future asthma (Puolijoki and Lahdensuo 1987; Dicpinigaitis 2006).

Clinics investigating occupational asthma have often found that workers referred for suspected work-related asthma turn out to have respiratory symptoms only, without asthma. In a Canadian study, 57.5% of the patients with respiratory symptoms did not have asthma (Chirý et al. 2009); in a British study, the corresponding figure was 40.4% (Fishwick et al. 2007). Workers who have been examined for occupational asthma but who did not have the disease confirmed may often continue in their previous work without their exposure conditions being improved. In our present study, we hypothesized that workers who display symptoms suggestive of asthma but whose lung function is normal may run an increased risk of developing clinical asthma. In addition, we assessed the importance of continued exposure to indoor workplace dampness and molds in the development of asthma.

The study was approved by the Ethics Committee of the Hospital District of Helsinki and Uusimaa.

## Materials and methods

### Source population

The study population was derived from a source population consisting of 2,200 workers who had been examined at the Finnish Institute of Occupational Health (FIOH) in the period 1995–2004 because of symptoms related to work in

damp and moldy indoor environments as a consequence of workplace moisture damages. The workers were referred to FIOH from all over Finland. The symptoms consisted of upper or lower airway symptoms, or both, and the patients had been examined by a physician because of a suspicion of an occupational disease. Those patients having complained of work-related symptoms compatible with asthma (cough, dyspnea, and wheeze) had been clinically assessed with respect to possible occupational asthma. The investigations included flow-volume spirometry according to the guidelines of the European Respiratory Society (Quanjer et al. 1993), as well as a bronchodilation test and a histamine challenge test (Sovijarvi et al. 1993). Diurnal variations in peak expiratory flow (PEF) had been assessed from either serial PEF monitoring at and away from work (Burge 1993) or PEF measurements carried out in the morning and in the evening, both over a period of 2 weeks. Altogether 912 patients (41%) had been diagnosed with asthma (either occupational or non-occupational asthma) in the initial examinations. The diagnostic criteria for asthma were (1) significant improvement ( $\geq 15\%$  and at least 200 mL) in the forced expiratory volume in 1 s (FEV1) or forced vital capacity (FVC) in response to short-acting bronchodilating medication in a bronchodilator test or (2) a repeated daily variation of  $\geq 20\%$  or improvement of  $\geq 15\%$  (and at least 60 L/min) in response to short-acting bronchodilating medication in a 2-week diurnal PEF follow-up.

This study was part of a larger questionnaire study of the source population (Fig. 1). Of the 2,200 patients, we excluded 24 persons whose symptoms were assessed as being caused by factors other than building dampness. During the follow-up, 20 patients died. Thus, at the beginning of 2007, 3–12 years after the initial examinations, 2,156 patients were invited to participate in a follow-up study comprised of a questionnaire on current symptoms, asthma medication, smoking habits, work status, work conditions, and the presence of dampness and molds in the current work environment. The development of new-onset asthma during the follow-up period was established by the question “Have you been diagnosed with asthma by a physician after the examinations at FIOH?”. Current symptoms were given a score by rating each symptom (cough, dyspnea, and wheeze) on a scale of severity ranging from 1 (not at all) to 5 (extremely). Atopic history was defined as a history of infantile eczema or atopic dermatitis, hay fever, or other allergic rhinitis or conjunctivitis. The questionnaire was answered by 1,295 (60.1%) of the 2,156 patients.

### Study group

The study group comprised 483 respondents who fulfilled the following inclusion criteria: (1) work-related

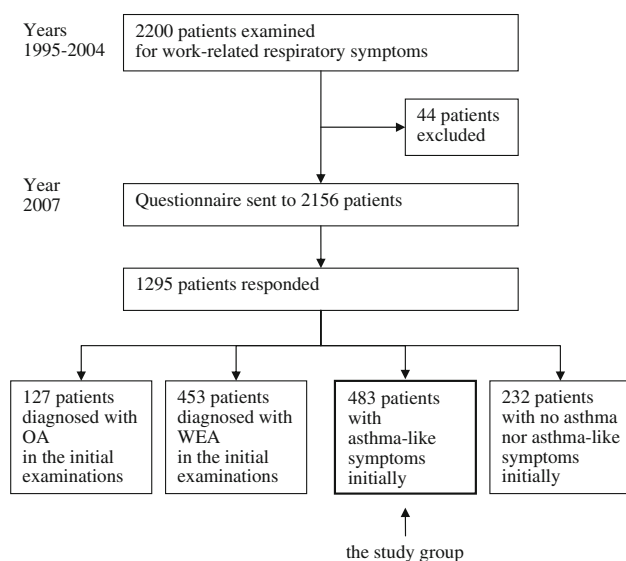


**Fig. 1** Distribution of the examined patients. OA occupational asthma, WEA work-exacerbated asthma

asthma-like symptoms (cough, dyspnea, and wheeze) without objective evidence of asthma in the initial examinations, (2) exposure to indoor dampness or molds or both at work before the initial examinations, (3) absence of indoor dampness at home (as reported by the patients in the initial examinations), and (4) asthma having never been diagnosed earlier. “Work-related symptoms” signified symptoms repeatedly appearing or worsening at work or improving away from work. Patients whose asthma had been diagnosed prior to the investigations at FIOH were likewise excluded. The absence of clinical asthma was ascertained from the medical records of all the patients finally included in the study group.

In order to validate the self-reported asthma diagnoses of the questionnaire study, all those who had reported having received a physician’s diagnosis of asthma were contacted. The patients were interviewed by phone and asked for written consent to study their clinical files, which were acquired from respective hospitals and physicians. The same aforementioned diagnostic criteria for asthma were used in the assessment of the medical records.

### Exposure assessment

In the initial examinations, building-related dampness or mold or both in the work environment had been described in all of the patient referrals by the physicians. The information had been corroborated by initial interviews with the patients. The exposure had taken place in various office environments, schools, hospitals, day care centers, etc. Alternative confounding exposures at work were excluded. All the reports on technical inspections, quality

measurements of the indoor air, and structural measurements of the workplace had been assembled in the patient records. In the reports, the moisture damage and microbial growth had been described by indoor air researchers with a background in construction engineering and occupational safety or health service personnel. In most (about 80%) cases, microbial analyses (material, settled dust, and air samples) and technical reports on building structure damage had verified the exposure to dampness and molds.

In conjunction with another study on source population, a retrospective assessment of the exposure to dampness and molds was undertaken. An experienced indoor air researcher evaluated the exposure to molds for a sample of 649 of the 2,200 patients (i.e., 30% of the source population). She assessed all of the available exposure data in the patient medical records by criteria reported in our earlier study (Karvala et al. 2010). For 83%, exposure to indoor molds could be verified by microbial measurements and technical reports, whereas the evidence of mold exposure was insufficient for 17% of the patients. If the exposure assessment was based on observations like mold odor without proper investigations into dampness and mold at work, the evidence considered insufficient.

The assessment of continued exposure was based on a questionnaire inquiry into the measures taken at the workplace and, in cases of relocation, the presence of building-related indoor dampness in the new work environment.

### Statistical methods

Multiple logistic regression analyses were used to examine the associations between asthma development and workplace exposure situations. The analyses included age, gender, atopic history, and smoking habits as confounders. The odds ratios (OR) with 95% confidential intervals (95% CI) are presented in the tables. The symptom prevalences for those who had developed asthma and those who had not were compared with the use of a  $\chi^2$ -test, the significance level being set at 0.05. The severity of symptoms at the follow-up displayed a non-normal distribution. Therefore, when comparing the symptom severities among those still exposed and the unexposed, we used the Wilcoxon two-sample test. All of the analyses were carried out using SAS 9.1 (SAS Institute Inc., Cary, NC, USA). The attributable risk was calculated using the equation  $(RR-1)/RR$ , where RR stands for the risk ratio for those exposed.

### Results

Most of the 483 persons studied were women (88%), the mean age being 45.3 (SD 8.6) years. The mean time from

the onset of symptoms to the initial examinations at FIOH was 4.8 (SD 3.3) years. The mean time from the examinations at FIOH to the point of the questionnaire study was 8.3 (SD 2.6) years.

A total of 62 patients (12.8%) claimed to have developed clinical asthma as diagnosed by a physician during the study period. The mean period from the examinations at FIOH to the asthma diagnosis was 4.8 (SD 3.1) years. During the follow-up, the incidence rate of asthma was 267 per 10,000 person-years. The mean time elapsed from the onset of symptoms to the time of receiving a diagnosis of asthma was 9.2 (SD 5.2) years.

There were no differences in the age or gender distribution between the patients who developed asthma and those who did not (Table 1). The mean age of the groups was 44.8 (SD 8.8) years and 45.2 (SD 8.6) years, respectively. Most of the patients (78%) worked in office-like surroundings, schools, hospitals, or children's day care centers (Table 1).

A variety of work-related symptoms were reported prior to the initial examinations at FIOH (Table 2). The prevalence of symptoms of the upper respiratory tract and the eyes had been high. A high prevalence of general symptoms, such as fever or chills, myalgia, arthralgia, headache, and fatigue, had also been reported. The prevalence of none

of these irritant or systemic symptoms differed between the persons who developed asthma and those who did not. At the follow-up, the current nasal and eye symptoms were significantly ( $p < 0.01$ ) less severe among those no longer exposed to indoor dampness at work than among those still exposed. In addition, the symptom scores for fatigue, fever or chills, myalgia or arthralgia, and headaches were lower for the unexposed patients ( $p < 0.0004$ ). Of the three asthma symptoms, the prevalence of dyspnea was higher for those who developed asthma, whereas cough and wheeze did not differ between the groups (Table 2).

An atopic history, either allergic rhino-conjunctivitis or atopic dermatitis, was a moderate risk factor for asthma (Table 3).

Among the patients who were working at the time of the follow-up, a larger proportion of those who developed asthma, than patients who did not, reported that they were still working in the same non-remediated environment (6.5% vs. 2.4%). A significantly lower proportion of those who had developed asthma had been relocated within the same company or organization than those who had not developed asthma (12.9% vs. 28.9%). Continued exposure to indoor dampness in the same or the new work environment was reported by 18.8% of the asthmatics and 11.4% of the non-asthmatics. The proportion of those currently unemployed was higher among those who had developed asthma (48.4% vs. 31.9%) (Table 4).

Among the currently employed (i.e., working at the time of the questionnaire study), the risk of new-onset asthma in relation to continued workplace exposure was increased more than fourfold (adjusted OR 4.6, 95% CI 1.8–11.6). Continued work in the former, non-remediated work environment was associated with a further increase in the risk (adjusted OR 6.4, 95% CI 1.5–27.7). Working for a new employer but still exposed to building-related dampness at work was associated with a similarly increased risk (OR 5.7, 95% CI 1.6–20.7) (Table 5). For the group still exposed, the proportion of asthma attributable to exposure to indoor dampness and mold at work was calculated to be 78%. For the group of workers continuing to work in the former unremediated environment, the attributable fraction was 85%.

When stratified by atopic history, the risk of asthma in the continuing exposure situation was only slightly higher among atopics (OR 5.1, 95% CI 1.4–19.1) than among non-atopic subjects (OR 4.68, 95% CI 1.2–18.6).

We were able to obtain some medical records for 46 of the 62 patients who reported having developed physician-diagnosed asthma. In 31 cases, the records fulfilled our diagnostic criteria for asthma which were the same as in the initial examinations, whereas the available clinical records of 15 cases did not. According to the questionnaire study, 48 of the 62 patients were using inhaled steroids regularly. When the excess risk of developing asthma in relation to

**Table 1** Characteristics of the study population with asthma-like symptoms at the time of the baseline examinations

|                                    | Development of new asthma |      |                      |      |
|------------------------------------|---------------------------|------|----------------------|------|
|                                    | Yes<br><i>n</i> = 62      |      | No<br><i>n</i> = 421 |      |
|                                    | <i>n</i>                  | %    | <i>n</i>             | %    |
| Gender                             |                           |      |                      |      |
| Male                               | 5                         | 8.1  | 53                   | 12.6 |
| Female                             | 57                        | 91.9 | 368                  | 87.4 |
| Age (years)                        |                           |      |                      |      |
| <35                                | 9                         | 14.5 | 52                   | 12.3 |
| 35–44                              | 19                        | 30.7 | 138                  | 32.8 |
| 45–54                              | 22                        | 35.5 | 163                  | 38.8 |
| 55–64                              | 12                        | 19.3 | 67                   | 15.9 |
| >64                                | 0                         | 0    | 1                    | 0.2  |
| Workplace                          |                           |      |                      |      |
| Office                             | 17                        | 27.4 | 150                  | 35.6 |
| School, college, university        | 10                        | 16.1 | 64                   | 15.2 |
| Day care center, kindergarten      | 10                        | 16.1 | 32                   | 7.6  |
| Hospital, health clinic or similar | 9                         | 14.5 | 84                   | 20.0 |
| Military forces                    | 3                         | 4.8  | 10                   | 2.3  |
| Production plant                   | 2                         | 3.2  | 14                   | 3.3  |
| Kitchen                            | 2                         | 3.2  | 14                   | 3.3  |
| Laboratory                         | 2                         | 3.2  | 8                    | 1.9  |
| Others                             | 7                         | 11.3 | 45                   | 10.7 |

**Table 2** Prevalence of reported work-related symptoms prior to the initial examinations

|                                                  | Development of new asthma |       |                |       | $\chi^2$ -test |
|--------------------------------------------------|---------------------------|-------|----------------|-------|----------------|
|                                                  | Yes                       |       | No             |       |                |
|                                                  | <i>n</i> = 62             |       | <i>n</i> = 421 |       | <i>p</i>       |
|                                                  | <i>n</i>                  | %     | <i>n</i>       | %     |                |
| Asthma-like symptoms                             |                           |       |                |       |                |
| Cough                                            | 54                        | 87.1  | 333            | 79.1  | 0.141          |
| Dyspnea                                          | 46                        | 74.2  | 253            | 60.1  | 0.033          |
| Wheezing                                         | 19                        | 30.6  | 110            | 26.2  | 0.460          |
| Cough, dyspnea, or wheeze                        | 62                        | 100.0 | 421            | 100.0 | –              |
| Symptoms of the upper respiratory tract and eyes |                           |       |                |       |                |
| Eye irritation                                   | 50                        | 80.6  | 315            | 74.8  | 0.319          |
| Nasal congestion                                 | 53                        | 85.5  | 336            | 80.2  | 0.323          |
| Rhinorrhea                                       | 36                        | 58.1  | 212            | 50.5  | 0.264          |
| Sneezing                                         | 32                        | 51.6  | 206            | 49.2  | 0.719          |
| Nasal congestion, rhinorrhea, or sneezing        | 55                        | 88.7  | 375            | 89.1  | 0.932          |
| Nasal congestion, rhinorrhea, and sneezing       | 26                        | 41.9  | 129            | 30.6  | 0.075          |
| Hoarseness                                       | 46                        | 74.2  | 312            | 74.1  | 0.989          |
| Sinus symptoms                                   | 40                        | 64.5  | 248            | 59.0  | 0.412          |
| Other symptoms                                   |                           |       |                |       |                |
| Fatigue                                          | 49                        | 79.0  | 354            | 84.3  | 0.297          |
| Headaches                                        | 39                        | 62.9  | 249            | 59.3  | 0.588          |
| Fever or chills                                  | 3                         | 49.2  | 194            | 46.4  | 0.686          |
| Myalgia or arthralgia                            | 27                        | 43.6  | 169            | 40.1  | 0.610          |
| Dermal symptoms                                  | 25                        | 40.3  | 162            | 38.6  | 0.792          |

**Table 3** Risk of asthma development in relation to atopic history and smoking

|                               | Development of new asthma |      |                      |      | Crude odds ratio |         | Adjusted odds ratio <sup>a</sup> |         |
|-------------------------------|---------------------------|------|----------------------|------|------------------|---------|----------------------------------|---------|
|                               | Yes<br><i>n</i> = 62      |      | No<br><i>n</i> = 421 |      |                  |         |                                  |         |
|                               | <i>n</i>                  | %    | <i>n</i>             | %    | OR               | 95% CI  | OR                               | 95% CI  |
| Atopic history                |                           |      |                      |      |                  |         |                                  |         |
| None (reference)              | 24                        | 38.7 | 263                  | 62.5 | 1.00             |         | 1.00                             |         |
| Allergic rhino-conjunctivitis | 32                        | 51.6 | 117                  | 28.8 | 2.6              | 1.5–4.6 | 2.6                              | 1.5–4.5 |
| Atopic dermatitis             | 23                        | 37.7 | 93                   | 22.9 | 2.0              | 1.2–3.6 | 2.1                              | 1.2–3.6 |
| Any atopic history            | 38                        | 61.3 | 158                  | 37.5 | 2.6              | 1.5–4.6 | 2.6                              | 1.5–4.6 |
| Smoking                       |                           |      |                      |      |                  |         |                                  |         |
| Never smoker (reference)      | 46                        | 74.2 | 241                  | 58.1 | 1.00             |         | 1.00                             |         |
| Ex-smoker                     | 6                         | 9.7  | 115                  | 27.7 | 0.3              | 0.1–0.7 | 0.3                              | 0.1–0.7 |
| Current smoker                | 10                        | 16.1 | 59                   | 14.2 | 0.9              | 0.4–1.9 | 0.9                              | 0.4–1.9 |

OR odds ratio

<sup>a</sup> Adjustment for age and gender

continued exposure was analyzed for only 18 persons who had physician-diagnosed asthma and who were working at the time of the questionnaire study, the statistical significance remained (adjusted OR 4.9, 95% CI 1.3–18.2).

## Discussion

When the study was being planned, sufficient epidemiological evidence already indicated an association between

indoor dampness and respiratory symptoms, including cough, dyspnea, wheeze, and chest tightness (IOM 2004). The evidence was only suggestive of an association with asthma development (Thorn et al. 2001; Jaakkola et al. 2002; IOM 2004). We tested the hypothesis that exposure to indoor dampness and mold at work increases the risk of developing new-onset asthma among workers suffering from work-related asthma-like symptoms (cough, dyspnea, and wheeze) but lacking the lung function changes required for a diagnosis of asthma. The study population had been

**Table 4** Exposure situation at the workplace among those who developed asthma and those who did not at the follow-up

| Workplace exposure situation              | Development of new asthma         |      |                                   |      |
|-------------------------------------------|-----------------------------------|------|-----------------------------------|------|
|                                           | Yes<br><i>n</i> = 62 <sup>a</sup> |      | No<br><i>n</i> = 421 <sup>b</sup> |      |
|                                           | <i>n</i>                          | %    | <i>n</i>                          | %    |
| Working in the same environment           |                                   |      |                                   |      |
| Workplace remediated                      | 8                                 | 13.3 | 63                                | 15.3 |
| Workplace not remediated                  | 4                                 | 6.7  | 10                                | 2.4  |
| Relocated in the same organization        |                                   |      |                                   |      |
| No moisture damage in the new environment | 5                                 | 8.3  | 95                                | 23.1 |
| Moisture damage in the new environment    | 3                                 | 5.0  | 24                                | 5.8  |
| Working for a new employer                |                                   |      |                                   |      |
| No moisture damage in the new environment | 7                                 | 11.7 | 75                                | 18.2 |
| Moisture damage in the new environment    | 5                                 | 8.3  | 13                                | 3.2  |
| Not currently employed                    | 28                                | 46.7 | 131                               | 31.9 |

<sup>a</sup> Information was unavailable for two patients<sup>b</sup> Information was unavailable for ten patients**Table 5** Risk of asthma development in relation to the continuation of workplace exposure

|                                                     | Development of new asthma         |                                   | Crude odds ratio |          | Adjusted odds ratio <sup>c</sup> |          |
|-----------------------------------------------------|-----------------------------------|-----------------------------------|------------------|----------|----------------------------------|----------|
|                                                     | Yes<br><i>n</i> = 34 <sup>a</sup> | No<br><i>n</i> = 290 <sup>b</sup> |                  |          |                                  |          |
|                                                     | <i>n</i>                          | <i>n</i>                          | OR               | 95% CI   | OR                               | 95% CI   |
| Cessation of exposure <sup>d</sup> (reference)      | 12                                | 170                               | 1.00             |          | 1.00                             |          |
| Continuation of exposure                            |                                   |                                   |                  |          |                                  |          |
| Working in the same, not remediated environment     | 4                                 | 10                                | 5.7              | 1.5–20.8 | 6.4                              | 1.5–27.7 |
| Relocated in the same organization, moisture damage | 3                                 | 24                                | 1.8              | 0.5–6.7  | 2.9                              | 0.7–12.4 |
| Working for a new employer, moisture damage         | 5                                 | 13                                | 5.4              | 1.7–17.8 | 5.7                              | 1.6–20.7 |
| Any of the above-mentioned exposure situations      | 12                                | 47                                | 3.6              | 1.5–8.6  | 4.6                              | 1.8–11.6 |
| Working in the same, remediated environment         | 8                                 | 63                                | 1.8              | 0.7–4.6  | 2.0                              | 0.7–5.4  |

Only currently employed persons included

OR odds ratio

<sup>a</sup> Information was unavailable for two patients<sup>b</sup> Information was unavailable for ten patients<sup>c</sup> Adjustment for age, gender, atopic history, and smoking<sup>d</sup> Relocated in the same organization or working for a new employer, and no moisture damage in the workplace

exposed to indoor dampness and molds at their workplaces. A significant proportion of the patients reported having developed asthma during the follow-up period. The incidence of new-onset asthma in this group was high, 267 per 10,000 person-years. By comparison, the incidence of asthma in a Finnish birth cohort born in 1966 was 18.5 per 10,000 person-years (Kujala et al. 2005). Our results suggest that asthma-like symptoms in relation to exposure to a damp and moldy work environment are associated with a marked increase in the risk of developing asthma. To our knowledge, such a relationship has not earlier been demonstrated for occupational environments.

The finding that asthma-like symptoms may increase the risk of developing asthma is supported by the results of

some earlier studies. A follow-up study (mean time 4.4 years) on adults suffering from chronic cough without having clinical asthma revealed that 16% (29/182) were diagnosed with asthma (Puolijoki and Lahdensuo 1987). In another Finnish study on children aged 7–12 years, who had lower airway symptoms but did not fulfill the criteria for asthma, one-third (11/33) developed asthma during a 2-year follow-up period (Remes et al. 1998). In our study, the proportion of those who developed asthma was lower (13%). However, the 62 patients having developed asthma at the time of the questionnaire study had developed asthma slowly. They reported having suffered from work-related asthma-like symptoms for a mean of 4.8 years earlier and a further mean of 4.8 years after the

examinations at FIOH before the diagnosis of asthma was established. The percentage would probably have been higher if the study had had a prospective study design in which also those with a shorter symptomatic period prior to developing asthma would have been included.

The most important risk factor for developing clinical asthma was continuing exposure to indoor dampness and molds in the work environment. The continuation of any exposure to dampness was associated with a more than fourfold increase in the risk of asthma (OR 4.6, 95% CI 1.8–11.6). Continued work in the former, non-remediated work environment was associated with a further increase in the risk (OR 6.4, 95% CI 1.5–27.7). The results suggest a clear association between factors related to indoor dampness and the development of asthma. The finding is supported by the most recent literature. Research groups and an international WHO expert group have concluded that the epidemiological evidence for an association between indoor dampness and mold and the development of new-onset asthma is sufficient (Fisk et al. 2007; WHO 2009). One study has reported an exposure–response relationship between indoor dampness and mold in the living area of homes and the development of asthma (Pekkanen et al. 2007).

Working in a remediated work environment was associated with a decrease in the risk of developing asthma, although the excess risk remained statistically insignificant (OR 2.0, 95% CI 0.7–5.4). The statistical insignificance was likely to be due to the small number of workers in this category (Table 5). The findings agree with the results of some recent studies. Successful remediation of moldy buildings has significantly reduced asthma morbidity (Jarvis and Morey 2001; Patovirta et al. 2004). In the intervention study by Jarvis and Morey (2001), a high rate of asthma was found for the 488 occupants of a mold-contaminated office building. After thorough repairs, no excess building-related illness occurred in the 5-year follow-up. In another study, a cluster of asthma cases was identified in a school with indoor dampness and molds, and after building repairs, no new cases of asthma appeared among the 31 teachers in a 3-year follow-up (Patovirta et al. 2004). Similarly, a well-designed intervention study by Kercsmar et al. (2006) showed that the removal of sources of dampness and molds was followed by a significant decrease in the exacerbation of asthma. The remaining excess risk for repaired work environments found in our study may indicate that the remediation of the buildings had been insufficient.

The outcome of the study (i.e., the development of clinical asthma) was established using questionnaire information on physician-diagnosed asthma. The question is widely used in epidemiological investigations and has been considered to be highly specific (99%) with respect

to asthma (Toren et al. 1993). The sensitivity is moderate (Toren et al. 1993) and selective for the severity of disease (Toren et al. 2006). The high specificity makes the question suitable for comparisons of risk estimates, whereas the moderate sensitivity should lead to an underestimation of incidence, especially for mild disease (Toren et al. 1993, 2006). A source of uncertainty with respect to the validity of the question is the fact that the patients who were followed had been examined at FIOH because of suspected mold-induced asthma. They were, therefore, aware of the suspected associations and may consequently have been more observant with respect to their symptoms and the diagnosis of asthma when answering the questionnaire.

Variations in the diagnostic procedures of physicians are a potential source of bias. However, in Finland, the diagnostics for asthma are rather uniform due, to a large extent, to the remedy reimbursement policy in force in Finland. The National Social Insurance Institution reimburses 72% of the costs of asthma medication to patients, provided that the specified diagnostic criteria of asthma are met. Thus, financial incentives encourage patients to obtain a physician's diagnosis of asthma. Furthermore, the clinical guidelines on asthma therapy and diagnostics, published in 2000 by the Finnish Medical Society Duodecim in the "Current Care Guidelines" program, are widely disseminated and available on the web (Duodecim 2000), and they have been adopted by most physicians (Ketola et al. 2007).

The attempt to retrospectively assess the validity of the patients' allegations of having developed asthma diagnosed by a physician proved to be difficult. Despite repeated efforts, only 46 patients could be reached. Moreover, medical records ascertaining the asthma diagnoses in accordance with diagnostic criteria were obtained for 31 cases. For 15 patients, the diagnosis could not be verified. Thus, it remains conjectural exactly how the asthma has been diagnosed. As the diagnoses for 31 patients could not be verified, there is a possibility of misclassification among those. For instance, asthma may have been mentioned by the physician as a rationale for asthma treatment. It was possible to analyze the association between continued exposure and the development of asthma among those 18 verified cases of asthma who still were working and thus could still be occupationally exposed to molds. Despite the small number included, an association with over fourfold risk remained.

The assessment of exposure is a common difficulty in studies on building-related dampness (IOM 2004; WHO 2009). There is a lack of valid, quantitative sampling methods for assessing exposure (IOM 2004; WHO 2009). No single measurement showing exposure is available, but several exposure indicators are used instead. In most

epidemiological studies, the exposure assessment is based on self-reports. It is the most cost-effective way to gather information on large numbers of persons (IOM 2004). In the source population, several methods to estimate the presence of moisture and mold damage were used: visual inspection of buildings, a more specific investigation into the building construction, and microbial sampling focusing on fungi and bacteria. The retrospective assessment of a sample comprising more than 600 (30%) of the source population showed that reliable data on dampness and molds at work were available for 83%. Although only about one-third of our study group was included in the sample, there is little reason to believe that our group would differ to any important extent in this respect.

The questionnaire study on exposure conditions may have been influenced by the participants' awareness of the exposure of interest (i.e., indoor dampness and mold growth at their workplace). Naturally, the persistence of symptoms at work and a physician's diagnosis of asthma at follow-up may have made them more inclined to observe and report dampness and mold at work as compared with those whose symptoms had ceased or attenuated. However, the highest risk of asthma was associated with continued work in the same non-remediated environment. The information on the measures used to repair these workplaces should be reliable. Some studies concerning housing environments have reported that occupants tend to underestimate the occurrence of dampness in comparisons with professional inspectors (Williamson et al. 1997; Koskinen et al. 1999; Engman et al. 2007). In summary, it is unlikely that information bias would explain the associations we found between continued exposure to indoor dampness and the development of asthma.

The study group of 483 patients was derived from a source population of 2,200 patients who had been investigated because of various work-related symptoms and a suspicion of exposure to dampness and moldy indoor environments as a cause of the symptoms. Of the source population invited to participate in the questionnaire study, 60% responded. The long follow-time of 3–12 years is likely to have decreased the interest in participating. As about 60% of the patients had not received a diagnosis of asthma, and a further large proportion had not even suffered from asthma-like symptoms, their interest in participating has presumably been low, as can partly be seen in Fig. 1. On the other hand, having a diagnosis of asthma did not cause any large selection as a total of 64% of those having received a diagnosis of asthma participated. In light of this, the participation rate was not considered unexpectedly low and should not have caused any serious bias.

The prevalence of respiratory, eye, and general symptoms leading to referral for examinations had been high.

In addition to symptoms from the lower airways, irritant symptoms of the upper airways and eyes, as well as general symptoms such as fever, headache, and fatigue, were very common. These symptoms have been found to be strongly associated with exposure to indoor dampness and mold (Bornehag et al. 2001; IOM 2004; Fisk et al. 2007; WHO 2009). The prevalence of these symptoms at the time of the baseline examinations did not differ between those who developed asthma and those who did not. At the follow-up, the score for reported eye, upper respiratory, and general symptoms was significantly lower among those no longer exposed to dampness and molds at their current workplace than among those still exposed. In accordance with the literature, our results indicate that irritant respiratory and general symptoms are associated with exposure to dampness and mold at work. Such high prevalences are invariably associated with poor indoor climate.

Atopic history, allergic rhino-conjunctivitis, or atopic eczema were, to some extent, related to an increase in the risk of asthma. With continued exposure, atopic history had a slight enhancing effect on the development of asthma.

In conclusion, our findings add to the knowledge on the association between indoor dampness and new-onset adult asthma. Our results suggest that for workers with work-related asthma-like symptoms occurring in relation to indoor dampness and molds at work, continuing work in such environments is a risk for developing asthma. The association appeared strongest if the worker continued in his or her former unremediated workplace, or in any work with continued exposure to indoor dampness. The avoidance of further exposure seemed to decrease the risk significantly. Continued work in the former remediated workplace was also associated with a reduced risk. These are novel findings that may apply to other occupational environments with irritant and sensitizing exposures as well. However, the study is based on a patient series and a later questionnaire. Therefore, selection may have modified the outcomes to some extent. In addition, a certain degree of diagnostic misclassification cannot be ruled out. Considering the study design, it was unfeasible to show a causal relationship. It is important that further studies on this topic are conducted to corroborate the associations found.

**Acknowledgments** The authors thank Dr Tuula Lindholm for her comments on the lung function measurements, Mrs Tuula Suomela for searching the patient information, Mrs Eeva-Liisa Ruohonen for contacting the persons who reported having developed asthma, and the Nursing and Support Services Team of FIOH for coding the questionnaire data. This study was supported by the Finnish Work Environment Fund.

**Conflict of interest** The authors declare that they have no conflict of interest.

## References

- Bornehag CG, Blomquist G, Gyntelberg F, Jarvholm B, Malmberg P, Nordvall L et al (2001) Dampness in buildings and health. Nordic interdisciplinary review of the scientific evidence on associations between exposure to “dampness” in buildings and health effects (NORDDAMP). *Indoor Air* 11:72–86. doi: [10.1034/j.1600-0668.2001.110202.x](https://doi.org/10.1034/j.1600-0668.2001.110202.x)
- Bornehag CG, Sundell J, Bonini S, Custovic A, Malmberg P, Skerfving S et al (2004) Dampness in buildings as a risk factor for health effects, EUROEXPO: a multidisciplinary review of the literature (1998–2000) on dampness and mite exposure in buildings and health effects. *Indoor Air* 14:243–257. doi: [10.1111/j.1600-0668.2004.00240.x](https://doi.org/10.1111/j.1600-0668.2004.00240.x)
- Burge PS (1993) Use of serial measurements of peak flow in the diagnosis of occupational asthma. *Occup Med* 8:279–294
- Chiry S, Boulet LP, Lepage J, Forget A, Begin D, Chaboillez S et al (2009) Frequency of work-related respiratory symptoms in workers without asthma. *Am J Ind Med* 52:447–454. doi: [10.1002/ajim.20695](https://doi.org/10.1002/ajim.20695)
- Dicpinigaitis PV (2006) Chronic cough due to asthma: ACCP evidence-based clinical practice guidelines. *Chest* 129(1 Suppl): 75–79. doi: [10.1378/chest.129.1\\_suppl.75S](https://doi.org/10.1378/chest.129.1_suppl.75S)
- Duodecim (2000) Astman diagnostiikka ja hoito [Diagnostics and therapy of asthma]. Duodecim; laaketieteellinen aikakauskirja, 116:2568–2584. <http://www.kaypahoito.fi>
- Engman LH, Bornehag CG, Sundell J (2007) How valid are parents' questionnaire responses regarding building characteristics, mouldy odour, and signs of moisture problems in Swedish homes? *Scand J Public Health* 35:125–132. doi: [10.1080/14034940600975658](https://doi.org/10.1080/14034940600975658)
- Fishwick D, Bradshaw L, Davies J, Henson M, Stenton C, Burge S et al (2007) Are we failing workers with symptoms suggestive of occupational asthma? *Prim Care Respir J* 16:304–310. doi: [10.3132/pcrj.2007.00064](https://doi.org/10.3132/pcrj.2007.00064)
- Fisk WJ, Lei-Gomez Q, Mendell MJ (2007) Meta-analyses of the associations of respiratory health effects with dampness and mold in homes. *Indoor Air* 17:284–296. doi: [10.1111/j.1600-0668.2007.00475.x](https://doi.org/10.1111/j.1600-0668.2007.00475.x)
- Haahtela T (1999) Early treatment of asthma. *Allergy* 54(Suppl 49):74–81. doi: [10.1111/j.1398-9995.1999.tb04392.x](https://doi.org/10.1111/j.1398-9995.1999.tb04392.x)
- IOM (Institute of Medicine of the National Academies of Science) (2004) Damp indoor spaces and health. National Academies Press, Washington DC
- Jaakkola MS, Nordman H, Piipari R, Uitti J, Laitinen J, Karjalainen A et al (2002) Indoor dampness and molds and development of adult-onset asthma: a population-based incident case-control study. *Environ Health Perspect* 110:543–547
- Jarvis JQ, Morey PR (2001) Allergic respiratory disease and fungal remediation in a building in a subtropical climate. *Appl Occup Environ Hyg* 16:380–388. doi: [10.1080/10473220117482](https://doi.org/10.1080/10473220117482)
- Karvala K, Toskala E, Luukkonen R, Lappalainen S, Uitti J, Nordman H (2010) New-onset adult asthma in relation to damp and moldy workplaces. *Int Arch Occup Environ Health* 83:855–865. doi: [10.1007/s00420-010-0507-5](https://doi.org/10.1007/s00420-010-0507-5)
- Kercsmar CM, Dearborn DG, Schluchter M, Xue L, Kirchner HL, Sobolewski J et al (2006) Reduction in asthma morbidity in children as a result of home remediation aimed at moisture sources. *Environ Health Perspect* 114:1574–1580. doi: [10.1289/ehp.8742](https://doi.org/10.1289/ehp.8742)
- Ketola E, Kaila M, Honkanen M (2007) Guidelines in context of evidence. *Qual Saf Health Care* 16:308–312. doi: [10.1136/qshc.2006.019752](https://doi.org/10.1136/qshc.2006.019752)
- Koskinen OM, Husman TM, Meklin TM, Nevalainen AI (1999) The relationship between moisture or mould observations in houses and the state of health of their occupants. *Eur Respir J* 14:1363–1367
- Kujala V, Remes J, Latvala J, Jarvelin MR (2005) Incidence of asthma in twelve thousand Finnish adults born in 1966. *Int J Occup Med Environ Health*. 18:255–258
- Nevalainen A, Partanen P, Jääskeläinen E, Hyvärinen A, Koskinen O, Meklin T et al (1998) Prevalence of moisture problems in Finnish houses. *Indoor Air* 8(Suppl 4):45–49. doi: [10.1111/j.1600-0668.1998.tb00007.x](https://doi.org/10.1111/j.1600-0668.1998.tb00007.x)
- Patovirta RL, Husman T, Haverinen U, Vahteristo M, Uitti JA, Tukiainen H et al (2004) The remediation of mold damaged school—a three-year follow-up study on teachers' health. *Cent Eur J Public Health* 12:36–42
- Pekkanen J, Hyvärinen A, Haverinen-Shaughnessy U, Korppi M, Putus T, Nevalainen A (2007) Moisture damage and childhood asthma: a population-based incident case-control study. *Eur Respir J* 29:509–515. doi: [10.1183/09031936.00040806](https://doi.org/10.1183/09031936.00040806)
- Piipari R, Keskinen H (2005) Agents causing occupational asthma in Finland in 1986–2002: cow epithelium bypassed by moulds from moisture-damaged buildings. *Clin Exp Allergy* 35:1632–1637. doi: [10.1111/j.1365-2222.2005.02386.x](https://doi.org/10.1111/j.1365-2222.2005.02386.x)
- Puolijoki H, Lahdensuo A (1987) Chronic cough as a risk indicator of broncho-pulmonary disease. *Eur J Respir Dis* 71:77–85
- Quanjer PH, Tammeling GJ, Cotes JE, Pedersen OF, Peslin R, Yernault JC (1993) Lung volumes and forced ventilatory flows. Report Working Party Standardization of Lung Function Tests, European Community for Steel and Coal. Official statement of the European Respiratory Society. *Eur Respir J Suppl* 16:5–40
- Remes ST, Korppi M, Remes K (1998) Outcome of children with respiratory symptoms without objective evidence of asthma: a two-year, prospective, follow-up study. *Acta Paediatr* 87:165–168. doi: [10.1111/j.1651-2227.1998.tb00969.x](https://doi.org/10.1111/j.1651-2227.1998.tb00969.x)
- Ryttilä P, Ghaly L, Varghese S, Chung W, Selroos O, Haahtela T (2008) Treatment with inhaled steroids in patients with symptoms suggestive of asthma but with normal lung function. *Eur Respir J* 32:989–996. doi: [10.1183/09031936.00062307](https://doi.org/10.1183/09031936.00062307)
- Sovijarvi AR, Malmberg LP, Reinikainen K, Ryttilä P, Poppius H (1993) A rapid dosimetric method with controlled tidal breathing for histamine challenge. Repeatability and distribution of bronchial reactivity in a clinical material. *Chest* 104:164–170
- Thorn J, Brisman J, Toren K (2001) Adult-onset asthma is associated with self-reported mold or environmental tobacco smoke exposures in the home. *Allergy* 56:287–292. doi: [10.1034/j.1398-9995.2001.00805.x](https://doi.org/10.1034/j.1398-9995.2001.00805.x)
- Toren K, Brisman J, Jarvholm B (1993) Asthma and asthma-like symptoms in adults assessed by questionnaires. A literature review. *Chest* 104:600–608
- Toren K, Palmqvist M, Lowhagen O, Balder B, Tunsater A (2006) Self-reported asthma was biased in relation to disease severity while reported year of asthma onset was accurate. *J Clin Epidemiol* 59:90–93. doi: [10.1016/j.jclinepi.2005.03.019](https://doi.org/10.1016/j.jclinepi.2005.03.019)
- WHO (2009) WHO guidelines for indoor air quality: dampness and mould. WHO Regional Office for Europe, Copenhagen. <http://www.euro.who.int/document/E92645.pdf>
- Williamson IJ, Martin CJ, McGill G, Monie RD, Fennerty AG (1997) Damp housing and asthma: a case-control study. *Thorax* 52:229–234. doi: [10.1136/thx.52.3.229](https://doi.org/10.1136/thx.52.3.229)