



# Dampness, mould, onset and remission of adult respiratory symptoms, asthma and rhinitis

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**Dampness and mould at home and in the workplace can increase onset and decrease remission of respiratory symptoms, doctor-diagnosed asthma and rhinitis among adults. Our study adds new evidence on the health significance of dampness and mould.** <http://ow.ly/Ek1330o3IBn>

**Cite this article as:** Wang J, Pindus M, Janson C, *et al.* Dampness, mould, onset and remission of adult respiratory symptoms, asthma and rhinitis. *Eur Respir J* 2019; 53: 1801921 [<https://doi.org/10.1183/13993003.01921-2018>].

## ABSTRACT

**Study question:** Is dampness and indoor mould associated with onset and remission of respiratory symptoms, asthma and rhinitis among adults?

**Materials and methods:** Associations between dampness, mould and mould odour at home and at work and respiratory health were investigated in a cohort of 11 506 adults from Iceland, Norway, Sweden, Denmark and Estonia. They answered a questionnaire at baseline and 10 years later, with questions on respiratory health, home and work environment.

**Results:** Baseline water damage, floor dampness, mould and mould odour at home were associated with onset of respiratory symptoms and asthma (OR 1.23–2.24). Dampness at home during follow-up was associated with onset of respiratory symptoms, asthma and rhinitis (OR 1.21–1.52). Dampness at work during follow-up was associated with onset of respiratory symptoms, asthma and rhinitis (OR 1.31–1.50). Combined dampness at home and at work increased the risk of onset of respiratory symptoms and rhinitis. Dampness and mould at home and at work decreased remission of respiratory symptoms and rhinitis.

**The answer to the question:** Dampness and mould at home and at work can increase onset of respiratory symptoms, asthma and rhinitis, and decrease remission.

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Received: Oct 07 2018 | Accepted after revision: March 07 2019

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## Introduction

Respiratory illnesses can be affected by the home environment [1–4]. Indoor dampness and mould are associated with respiratory illnesses, but most studies are cross-sectional studies which limits conclusions on causality [1–4]. One review included 16 studies on associations between residential dampness and mould and incidence of asthma [1], concluding that dampness and mould at home are determinants of incident asthma. However, only two studies investigated adult asthma [5, 6]. Later, a longitudinal multicentre study in Europe demonstrated that water damage and indoor moulds in homes were related to asthma onset [7].

Asthma and rhinitis coexist [8, 9]. An association between adult asthma and allergic rhinitis has been documented in a population study in Sweden [10]. A systematic review concluded that dampness and mould was associated with rhinitis [2], but only two out of 31 studies were on adult rhinitis [11, 12]. None were prospective studies.

Few studies exist on asthma or rhinitis in relation to dampness at work. Working in a damp office building was associated with work-related asthma [13]. An incident case–control study found that mould and mould odour in the workplace were related to adult-onset asthma [6, 14]. One hospital study showed that water damage was associated with onset of asthma [15]. An intervention study found that remediation of mould in an office building decreased asthmatic symptoms [16].

Mould odour at home has been shown to be associated with incident asthma [1]. As compared to other dampness indicators, mould odour had the strongest association with asthma onset [1] and rhinitis [2].

More longitudinal studies are needed on respiratory effects of dampness and mould. The Respiratory Health in Northern Europe (RHINE) study is a large population-based cohort study among adults [5]. Our aim was to study associations between onset and remission of respiratory symptoms, asthma and rhinitis among adults and indoor dampness, mould and mould odour at home and in the workplace building, in a RHINE follow-up from 1999–2000 (baseline) to 2010–2012 (follow-up).

## Materials and methods

### Study design and target population

The RHINE II study is a follow-up study of subjects from seven Nordic study centres from the European Community Respiratory Health Survey stage (ECRHS) I in 1989–1992. In ECRHS I, 3000–4000 subjects (aged 20–44 years) were randomly selected from the population register in each centre. A postal questionnaire was then sent to those subjects.

The RHINE II study included all subjects from seven centres in northern Europe: Reykjavik in Iceland, Bergen in Norway, Umeå, Uppsala and Göteborg in Sweden, Aarhus in Denmark and Tartu in Estonia. In total, 21 681 subjects participated in ECRHS I (response rate 86%) [17]. In RHINE II, participants received a follow-up postal questionnaire in 1999–2000. The RHINE II questionnaire included questions on respiratory health and the indoor environment at home and at work. The RHINE II participants were invited for a second follow-up (RHINE III) in 2010–2012, with identical questions on respiratory health as in RHINE II. In total, 11 506 subjects participated in RHINE II and RHINE III (response rate 71%) (figure 1). Participation was defined as answering at least one of five questions on respiratory symptoms (wheeze, nocturnal chest tightness, nocturnal attacks of breathlessness, nocturnal cough or asthma attack; detailed description is given later). In the present study, we define RHINE II as the baseline study and the RHINE III survey as the follow-up.

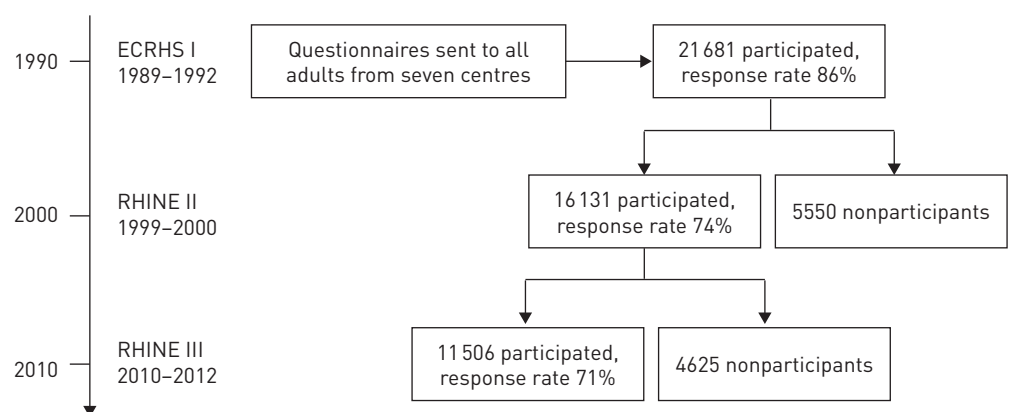


FIGURE 1 The flow chart of the study design. ECRHS: European Community Respiratory Health Survey; RHINE: Respiratory Health in Northern Europe study.

**Assessment of health and demographic data**

Questions regarding respiratory symptoms (same at baseline and follow-up) included the following:

- 1) Wheezing or whistling in the chest in the last 12 months (“wheeze”)
- 2) Woken up with a feeling of tightness in the chest at any time in the last 12 months (“nocturnal chest tightness”)
- 3) Woken up by an attack of shortness of breath in the last 12 months (“nocturnal attacks of breathlessness”)
- 4) Woken up by an attack of coughing in the last 12 months (“nocturnal cough”)
- 5) An asthma attack in the last 12 months (“asthma attack”)
- 6) Usually bring up phlegm or have any problem with bringing up phlegm in the past 12 months (“productive cough”)
- 7) Currently taking any medication (including inhalers, aerosols or tablets) for asthma

Current asthma was defined as having either asthma attacks in the past 12 months or current asthma medication, or both [18].

There was one question regarding doctor-diagnosed asthma:

- 1) Ever had asthma diagnosed by a doctor (“ever doctor-diagnosed asthma”). This question measured cumulative incidence.

Questions regarding allergic rhinitis and rhinitis symptoms included the following:

- 1) Any nasal allergies including hay fever (“allergic rhinitis”)
- 2) Ever had nose symptoms such as stuffy nose, runny nose and/or sneezing when not having a cold (“ever rhinitis symptoms”). This question measured cumulative incidence of rhinitis

Onset of doctor-diagnosed asthma, allergic rhinitis and rhinitis symptoms were defined as not reporting the particular respiratory illness at baseline, but reporting it at follow-up.

Onset of a particular respiratory symptom such as wheeze, nocturnal chest tightness, nocturnal breathlessness, nocturnal cough, productive cough or current asthma was defined as not reporting the particular symptom at baseline, but reporting the particular symptom at follow-up [5]. Participants with doctor-diagnosed asthma at baseline were excluded when calculating onset of these respiratory symptoms.

Remission of a particular respiratory symptom (wheeze, nocturnal chest tightness, nocturnal breathlessness, nocturnal cough, productive cough and current asthma) was defined as reporting the particular symptom at baseline, but not at follow-up, including those with doctor-diagnosed asthma at baseline.

Remission of allergic rhinitis was defined as reporting allergic rhinitis at baseline, but not at follow-up.

Data on sex, age, height and weight were obtained from the baseline data, while education level was only available at follow-up. Smoking habit (never-/ever-/current smokers) were available both at baseline and follow-up. Body mass index (BMI) was calculated as the ratio of weight in kilograms to height in metres squared ( $\text{kg}\cdot\text{m}^{-2}$ ). Change of smoking habit from baseline to follow-up (no change/start smoking/stop smoking) was calculated. Education level was categorised as primary school, high school or university education at follow-up.

**Assessment of indoor dampness, mould and mould odour**

Four questions about the home environment were asked about the current home in the past 12 months, as follows:

- 1) Water leakage or water damage indoors on walls, floors or ceilings (“water damage”)
- 2) Bubbles or yellow discolouration on plastic floor covering or black discolouration of parquet floor (“floor dampness”)
- 3) Visible mould growth indoors on walls, floors or ceilings (“visible mould”)
- 4) Mould odour in one or several rooms (other than the cellar)

The variable “any dampness” was defined as answering yes to question 1), 2) or 3) above.

Moreover, two questions were asked about home and workplace exposure during the follow-up period:

- 1) Any dampness damage, water leakage or visible mould at home during the past 10 years (“dampness or mould at home during follow-up”)
- 2) Any dampness damage, water leakage or visible mould in the workplace building during the past 10 years (“dampness or mould at work during follow-up”)

A categorised variable with four alternatives was created: no dampness/mould; dampness/mould at home only; dampness/mould at work only; and dampness/mould at home and at work.

### Statistical analysis

We used Stata 15.1 (Stata Corporation, College Station, TX, USA). Two-level (centre, individual) logistic regression models were performed to estimate associations between dampness indicators at baseline or during follow-up and onset of respiratory symptoms (wheeze, nocturnal chest tightness, nocturnal breathlessness, nocturnal cough, productive cough and current asthma), doctor-diagnosed asthma, allergic rhinitis and rhinitis symptoms, adjusting for sex, age, smoking habit at baseline, change of smoking habit from baseline to follow-up, BMI at baseline and education level at follow-up. Subjects with doctor-diagnosed asthma at baseline were excluded when analysing onset of respiratory symptoms. Similar two-level logistic regression models were then applied to estimate associations between dampness indicators and remission of respiratory symptoms and allergic rhinitis. Additionally, associations between combination of dampness and mould at home and at work during follow-up (categorised as none, at home, at work or both) and onset of respiratory symptoms and allergic rhinitis were estimated using similar logistic regression models. Associations were expressed as OR (95% CI), with 5% significance.

### Ethics statement

This study was conducted with the approval of the appropriate ethics board at each centre. All participants gave informed consent prior to participation.

### Results

In total, 11 506 respondents were included; 54.3% were female, 26.3% were current smokers and 26.4% were ex-smokers at baseline. During follow-up, 2.4% started smoking and 12.1% stopped smoking. There were no associations between smoking habit and dampness indicators at baseline (RHINE II). The mean $\pm$ SD age at baseline was 40 $\pm$ 7.3 years. The mean follow-up time was 11.3 $\pm$ 1.1 years. Nonparticipants in RHINE II had slightly higher prevalence of wheeze, nocturnal chest tightness and nocturnal attacks of breathlessness, but lower prevalence of allergic rhinitis in ECRHS I as compared to participants (data not shown). Moreover, nonparticipants in RHINE III had slightly higher prevalence of respiratory symptoms and asthma in RHINE II as compared to participants (table 1). However, prevalence of dampness indicators at home at baseline (RHINE II) did not differ between participants and nonparticipants in RHINE III (table 1).

TABLE 1 Respiratory health and dampness indicators in the Respiratory Health in Northern Europe (RHINE) II study among participants and nonparticipants in RHINE III

	Participants in RHINE III	Nonparticipants in RHINE III	p-value
<b>Subjects n</b>	11 506	4625	
<b>Health RHINE II</b>			
Wheeze	19.4	23.6	<0.001
Nocturnal chest tightness	10.6	12.8	<0.001
Nocturnal attacks of breathlessness	4.7	6.7	<0.001
Nocturnal cough	28.3	30.8	0.002
Productive cough	16.6	21.1	<0.001
Current asthma <sup>#</sup>	6.5	7.5	0.022
Ever doctor-diagnosed asthma	7.9	9.1	0.010
Allergic rhinitis	23.3	22.9	0.525
Ever rhinitis symptoms	47.9	48.9	0.265
<b>Dampness RHINE II</b>			
Water damage	13.4	13.6	0.713
Floor dampness	3.8	3.8	0.862
Visible mould	6.7	6.7	0.963
Mould odour	3.5	3.8	0.349
Any dampness <sup>¶</sup>	17.9	18.1	0.685

Data are presented as (%), unless otherwise stated. <sup>#</sup>: defined as an asthma attack in the past 12 months, current asthma medication or both; <sup>¶</sup>: defined as water damage, floor dampness or visible mould in the past 12 months at baseline.

TABLE 2 Onset and remission over the study period of respiratory symptoms, asthma, allergic rhinitis and rhinitis symptoms

	Aarhus	Reykjavik	Bergen	Göteborg	Umeå	Uppsala	Tartu	Total
<b>Onset</b>								
Wheeze	8.7	13.0	9.4	9.5	7.7	8.8	13.7	9.9
Nocturnal chest tightness	8.5	8.4	6.3	6.9	4.1	4.6	13.9	7.2
Nocturnal attacks of breathlessness	2.4	2.2	3.8	2.9	3.1	2.9	11.0	3.7
Nocturnal cough	13.4	16.6	12.3	17.5	18.4	16.7	31.5	17.0
Productive cough	6.6	12.0	9.0	9.8	9.7	7.5	12.6	9.3
Current asthma <sup>#</sup>	2.6	6.3	4.8	3.7	3.3	4.7	3.6	4.1
Ever doctor-diagnosed asthma	3.4	7.0	5.9	2.4	3.9	3.7	3.8	4.3
Allergic rhinitis	11.2	12.7	7.8	7.3	7.1	7.5	14.4	9.5
Ever rhinitis symptoms	25.2	27.6	24.8	24.4	22.0	22.1	40.9	25.9
<b>Remission</b>								
Wheeze	54.8	44.9	45.7	46.2	47.4	51.0	41.0	47.5
Nocturnal chest tightness	72.4	73.4	63.0	71.3	66.7	69.0	56.1	66.9
Nocturnal attacks of breathlessness	70.9	76.7	66.2	66.3	71.3	61.9	57.3	65.8
Nocturnal cough	61.5	49.8	52.6	47.2	44.3	46.7	32.2	48.2
Productive cough	62.7	56.1	55.5	49.5	51.7	54.5	57.8	54.8
Current asthma <sup>#</sup>	41.4	42.6	42.1	42.6	32.3	35.9	45.5	38.9
Allergic rhinitis	24.2	33.6	19.9	24.1	22.0	19.9	36.0	24.7

Data are presented as (%). <sup>#</sup>: defined as an asthma attack in the past 12 months, current asthma medication or both.

The highest onset rate over the study period was for rhinitis symptoms (25.9%). Onset rates of wheeze (9.9%), productive cough (9.3%) and allergic rhinitis (9.5%) were similar. The onset rate for doctor-diagnosed asthma was 4.3%. Among all the centres, Tartu had the highest onset rates for most respiratory symptoms. Approximately half of the participants with wheeze, nocturnal cough or productive cough at baseline were free from having those particular symptoms at follow-up. The highest remission rate was for nocturnal chest tightness (66.9%) (table 2).

Among environmental factors reported at baseline, water damage (13.4%) and visible mould (6.7%) were most common (table 3). A quarter (25.2%) of the participants in follow-up reported dampness at home in their current or previous home during the past 10 years, and 19.4% reported dampness in their current or previous workplace building in the past 10 years. All signs of indoor dampness, except floor dampness, were most common in Tartu. Floor dampness was most common in Reykjavik. Mould odour was most common in Tartu. In general, the signs of indoor dampness and mould were less common in Bergen, Göteborg and Umeå.

Dampness indicators at baseline and during follow-up were risk factors for onset of wheeze, nocturnal chest tightness, nocturnal breathlessness, nocturnal cough, productive cough, current asthma or doctor-diagnosed

TABLE 3 Prevalence of signs of indoor dampness and mould at home and in the workplace building in seven centres

	Aarhus	Reykjavik	Bergen	Göteborg	Umeå	Uppsala	Tartu	Total
<b>Baseline (at home)</b>								
Water damage	13.3	20.0	13.3	8.1	10.0	8.9	23.3	13.4
Floor dampness	1.6	6.7	2.1	4.2	5.7	3.9	2.9	3.8
Visible mould	9.8	6.1	4.7	4.6	3.9	6.4	13.0	6.7
Mould odour	4.2	4.8	2.3	2.4	2.5	3.2	5.9	3.6
Any dampness <sup>#</sup>	17.8	22.7	16.2	12.4	14.0	14.9	31.7	17.9
<b>Follow-up</b>								
Dampness or mould at home during follow-up	23.8	32.6	24.5	20.9	20.9	23.2	34.2	25.2
Dampness or mould in the workplace building during follow-up	11.6	22.1	17.1	19.4	21.0	23.5	23.7	19.4

Data are presented as (%). <sup>#</sup>: defined as water damage, floor dampness or visible mould in the past 12 months at baseline.

asthma (table 4). Mould odour at baseline was associated with onset of wheeze, nocturnal chest tightness, nocturnal breathlessness, nocturnal cough and doctor-diagnosed asthma (table 4).

There were few associations between dampness indicators and rhinitis. Dampness or mould at home during follow-up was the only risk factor for onset of allergic rhinitis. Dampness or mould at home during follow-up and dampness or mould at work during follow-up were both associated with onset of rhinitis symptoms (table 4).

Dampness and indoor mould decreased the remission rate. Water damage at baseline was related to less remission of nocturnal chest tightness and nocturnal cough at follow-up. Visible mould at baseline was associated with less remission of nocturnal chest tightness and nocturnal breathlessness. Any dampness at baseline decreased the likelihood for remission of nocturnal chest tightness, nocturnal breathlessness and productive cough. Dampness or mould at home during follow-up was related to decreased remission of nocturnal cough and allergic rhinitis. Dampness or mould at work during follow-up was associated with decreased remission of wheeze (table 5).

Participants exposed to dampness both at home and in workplace buildings had the strongest associations with onset of wheeze, nocturnal chest tightness, nocturnal breathlessness, nocturnal cough, productive cough, doctor-diagnosed asthma and rhinitis symptoms (table 6).

## Discussion

In this prospective study we found associations between dampness and mould at home at baseline and onset of respiratory symptoms and doctor-diagnosed asthma. Moreover, dampness and mould decreased the remission of respiratory symptoms and allergic rhinitis. Moreover, mould odour at home at baseline was associated with onset of respiratory symptoms and doctor-diagnosed asthma. Dampness or mould at home and at work during follow-up were related to onset of respiratory symptoms and doctor-diagnosed asthma as well as allergic rhinitis and rhinitis symptoms. Exposure to dampness during follow-up both at home and at work increased the risk of onset of respiratory symptoms and rhinitis symptoms.

In total, 17.9% of the homes had dampness at baseline (in the past 12 months). This prevalence is similar to that reported in a previous review (16.5%) including 31 studies on dampness and mould in the European housing stock [19]. Water damage (in the past 12 months) at baseline was 13.4% in our study. This is higher than the mean prevalence of water damage (10.0%) in Europe reported from the ECRHS study [20]. In our study, the prevalence of water damage differed from 7.7% in Göteborg to 23.4% in Tartu, possibly due to different building technologies in different Nordic countries. It has been reported that dampness in homes in Europe is associated with annual precipitation and higher ambient temperature [20]. Thus, differences in climate between the centres may also play role for the differences in prevalence of dampness. However, the prevalence of visible mould in our study (6.7%) was much lower as compared to the multicentre study (16%) [20].

Participants with doctor-diagnosed asthma at baseline were excluded from the calculation of onset of respiratory symptoms, since the aim was to study onset of symptoms, and not worsening of existing asthma. The associations between dampness at home and onset of respiratory symptoms and asthma in our study is consistent with two previous studies [5, 7]. Dampness at home increased onset of respiratory symptoms in the RHINE II study [5], with slightly lower odds ratios as compared to our study. The other multicentre study in Europe found stronger associations between water damage (OR 1.46), visible mould (OR 1.30) and onset of asthma than our study. Dampness problems on floors have been reported to cause higher emission of ammonia [21] and 2-ethyl-1-hexanol to indoor air [22]. Previous prevalence studies reported that dampness in the floor construction can be associated with reduced lung function (forced expiratory volume in 1 s) [23, 24], asthma symptoms [5, 23–25] and asthma [26].

Our study suggested that mould odour at baseline can increase onset of respiratory symptoms and doctor-diagnosed asthma. This is in agreement with a previous review on incident asthma [1]. However, this review included mainly childhood studies [1]. Previous prevalence studies have shown associations between mould odour at home and asthma or asthmatic symptoms among adults [11, 27]. Mould odour can be enhanced by poor ventilation. One Swedish study found that mould odour at home was associated with asthma symptoms in the initial model, but the association disappeared when adjusting for measured air exchange ventilation in the home [25]. An experimental study found that increased ventilation was associated with fewer reports of odour in university classrooms [28]. Unfortunately, we have no information on ventilation flow in the RHINE study.

We found that dampness and mould in the workplace building during follow-up was associated with onset of respiratory symptoms and doctor-diagnosed asthma. This is in agreement with previous studies on respiratory effects of dampness in workplace buildings. One recent review concluded that exposure to mould

TABLE 4 Adjusted odds ratios with 95% confidence intervals for onset of respiratory symptoms, doctor-diagnosed asthma and rhinitis

	Water damage	Floor dampness	Visible mould	Mould odour	Any dampness <sup>#</sup>	Dampness or mould at home during follow-up	Dampness or mould in the workplace building during follow-up
<b>Wheeze</b>	1.60 (1.31–1.97)	2.24 (1.61–3.13)	1.49 (1.12–1.98)	1.56 (1.07–2.29)	1.61 (1.33–1.94)	1.38 (1.17–1.64)	1.46 (1.21–1.75)
p-value	<b>&lt;0.001</b>	<b>&lt;0.001</b>	<b>0.006</b>	<b>0.022</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>
<b>Nocturnal chest tightness</b>	1.28 (1.02–1.60)	1.61 (1.08–22.39)	1.21 (0.88–1.66)	1.81 (1.25–2.63)	1.28 (1.04–1.58)	1.34 (1.11–1.61)	1.50 (1.22–1.83)
p-value	<b>0.035</b>	<b>0.018</b>	0.247	<b>0.002</b>	<b>0.018</b>	<b>0.002</b>	<b>&lt;0.001</b>
<b>Nocturnal breathlessness</b>	1.32 (0.98–1.79)	2.11 (1.32–3.38)	1.43 (0.96–2.12)	2.01 (1.26–3.22)	1.48 (1.13–1.93)	1.24 (0.97–1.60)	1.41 (1.08–1.84)
p-value	0.069	<b>0.002</b>	0.075	<b>0.004</b>	<b>0.004</b>	0.088	<b>0.012</b>
<b>Nocturnal cough</b>	1.23 (1.02–1.49)	1.51 (1.08–2.11)	1.34 (1.03–1.73)	1.46 (1.04–2.06)	1.31 (1.11–1.55)	1.21 (1.04–1.40)	1.47 (1.25–1.72)
p-value	<b>0.032</b>	<b>0.015</b>	<b>0.029</b>	<b>0.030</b>	<b>0.002</b>	<b>0.013</b>	<b>&lt;0.001</b>
<b>Productive cough</b>	1.21 (0.97–1.51)	1.48 (1.02–2.14)	1.46 (1.10–1.95)	1.37 (0.92–2.04)	1.35 (1.12–1.65)	1.52 (1.29–1.81)	1.33 (1.11–1.61)
p-value	0.088	<b>0.038</b>	<b>0.010</b>	0.116	<b>0.002</b>	<b>&lt;0.001</b>	<b>0.003</b>
<b>Current asthma</b>	1.34 (1.01–1.77)	1.44 (0.90–2.31)	1.25 (0.84–1.86)	1.46 (0.89–2.39)	1.36 (1.05–1.76)	1.09 (0.86–1.38)	1.25 (0.97–1.61)
p-value	<b>0.044</b>	0.129	0.279	0.136	<b>0.018</b>	0.482	0.084
<b>Doctor-diagnosed asthma</b>	1.36 (1.04–1.78)	1.96 (1.30–2.96)	1.36 (0.93–1.97)	2.23 (1.48–3.37)	1.43 (1.12–1.83)	1.32 (1.06–1.65)	1.40 (1.10–1.79)
p-value	<b>0.027</b>	<b>0.001</b>	0.109	<b>&lt;0.001</b>	<b>0.004</b>	<b>0.014</b>	<b>0.006</b>
<b>Allergic rhinitis</b>	1.14 (0.92–1.41)	0.99 (0.65–1.49)	1.05 (0.77–1.43)	0.89 (0.57–1.40)	1.10 (0.90–1.34)	1.28 (1.08–1.52)	1.21 (1.00–1.47)
p-value	0.239	0.948	0.752	0.613	0.360	<b>0.005</b>	0.050
<b>Ever rhinitis symptoms</b>	1.01 (0.84–1.23)	1.03 (0.70–1.52)	0.98 (0.74–1.30)	1.23 (0.84–1.79)	1.06 (0.89–1.26)	1.36 (1.18–1.58)	1.31 (1.11–1.54)
p-value	0.905	0.867	0.909	0.286	0.509	<b>&lt;0.001</b>	<b>0.001</b>

Two-level logistic regression models (centre, individual), adjusted for age (baseline), sex (baseline), smoking (baseline), change of smoking habit from baseline to follow-up, body mass index (baseline) and education (follow-up). Bold type represents statistical significance. #: defined as water damage, floor dampness or visible mould in the past 12 months at baseline.

TABLE 5 Adjusted odds ratios with 95% confidence intervals for remission of respiratory symptoms and allergic rhinitis

	Water damage	Floor dampness	Visible mould	Mould odour	Any dampness <sup>#</sup>	Dampness or mould at home during follow-up	Dampness or mould in the workplace building during follow-up
<b>Wheeze</b>	0.89 (0.70–1.15)	1.01 (0.68–1.49)	1.21 (0.88–1.67)	1.02 (0.69–1.53)	0.93 (0.75–1.16)	0.87 (0.71–1.06)	0.71 (0.57–0.89)
p-value	0.376	0.973	0.242	0.906	0.531	0.162	<b>0.003</b>
<b>Nocturnal chest tightness</b>	0.70 (0.51–0.97)	0.69 (0.42–1.14)	0.64 (0.42–0.96)	0.71 (0.42–1.20)	0.68 (0.51–0.92)	0.84 (0.64–1.12)	0.76 (0.56–1.02)
p-value	<b>0.032</b>	0.150	<b>0.031</b>	0.196	<b>0.011</b>	0.235	0.070
<b>Nocturnal breathlessness</b>	0.63 (0.39–1.02)	0.55 (0.27–1.09)	0.52 (0.29–0.95)	0.70 (0.35–1.42)	0.59 (0.38–0.91)	0.83 (0.54–1.27)	0.68 (0.43–1.09)
p-value	0.059	0.085	<b>0.035</b>	0.325	<b>0.018</b>	0.389	0.108
<b>Nocturnal cough</b>	0.79 (0.64–0.98)	0.82 (0.59–1.15)	0.82 (0.63–1.08)	0.96 (0.67–1.38)	0.84 (0.70–1.01)	0.81 (0.68–0.96)	0.88 (0.74–1.06)
p-value	<b>0.030</b>	0.257	0.163	0.828	0.057	<b>0.013</b>	0.194
<b>Productive cough</b>	0.77 (0.59–1.01)	0.82 (0.54–1.26)	0.73 (0.51–1.03)	0.87 (0.55–1.36)	0.76 (0.60–0.97)	1.01 (0.82–1.26)	0.90 (0.70–1.15)
p-value	0.055	0.368	0.077	0.541	<b>0.026</b>	0.909	0.387
<b>Current asthma</b>	1.36 (0.86–2.17)	0.99 (0.51–1.91)	1.03 (0.60–1.78)	1.08 (0.52–2.22)	0.97 (0.65–1.44)	1.07 (0.74–1.56)	1.05 (0.71–1.55)
p-value	0.192	0.970	0.912	0.843	0.885	0.705	0.816
<b>Allergic rhinitis</b>	1.00 (0.76–1.32)	1.10 (0.70–1.75)	1.10 (0.78–1.56)	1.36 (0.90–2.06)	1.07 (0.84–1.37)	0.77 (0.61–0.96)	0.93 (0.73–1.18)
p-value	0.991	0.676	0.576	0.148	0.581	<b>0.021</b>	0.535

Two-level logistic regression models (centre, individual), adjusted for age (baseline), sex (baseline), smoking (baseline), change of smoking habit from baseline to follow-up, body mass index (baseline) and education (follow-up). Bold type represents statistical significance. #: defined as water damage, floor dampness or visible mould in the past 12 months at baseline.

TABLE 6 Associations between onset of respiratory symptoms and allergic rhinitis and dampness or mould during follow-up (four categories: none, at home, at work and both)

	None	Only at home	Only at work	Both
<b>Wheeze</b>	1.00	1.34 (1.09–1.65)	1.45 (1.14–1.84)	1.75 (1.34–2.28)
p-value		<b>0.005</b>	<b>0.003</b>	<b>&lt;0.001</b>
<b>Nocturnal chest tightness</b>	1.00	1.26 (1.003–1.58)	1.49 (1.15–1.94)	1.75 (1.32–2.33)
p-value		<b>0.047</b>	<b>0.003</b>	<b>&lt;0.001</b>
<b>Nocturnal breathlessness</b>	1.00	1.23 (0.90–1.67)	1.47 (1.05–2.07)	1.54 (1.04–2.28)
p-value		0.191	<b>0.026</b>	<b>0.031</b>
<b>Nocturnal cough</b>	1.00	1.16 (0.97–1.38)	1.44 (1.18–1.76)	1.61 (1.28–2.04)
p-value		0.115	<b>&lt;0.001</b>	<b>&lt;0.001</b>
<b>Productive cough</b>	1.00	1.51 (1.23–1.85)	1.24 (0.96–1.59)	1.80 (1.38–2.33)
p-value		<b>&lt;0.001</b>	0.096	<b>&lt;0.001</b>
<b>Current asthma</b>	1.00	1.11 (0.83–1.48)	1.42 (1.04–1.94)	1.07 (0.72–1.60)
p-value		0.487	<b>0.027</b>	0.741
<b>Doctor-diagnosed asthma</b>	1.00	1.29 (0.98–1.69)	1.47 (1.08–2.01)	1.51 (1.06–2.15)
p-value		0.068	<b>0.016</b>	<b>0.021</b>
<b>Allergic rhinitis</b>	1.00	1.31 (1.07–1.60)	1.29 (1.01–1.65)	1.32 (0.99–1.75)
p-value		<b>0.010</b>	<b>0.044</b>	0.055
<b>Ever rhinitis symptoms</b>	1.00	1.28 (1.07–1.52)	1.16 (0.94–1.43)	1.75 (1.37–2.25)
p-value		<b>0.006</b>	0.167	<b>&lt;0.001</b>

Data are presented as adjusted OR (95% CI), unless otherwise stated. Two-level logistic regression models (centre, individual), adjusted for age (baseline), sex (baseline), smoking (baseline), change of smoking habit from baseline to follow-up, body mass index (baseline) and education (follow-up). Bold type represents statistical significance.

in workplace buildings is associated with incidence of occupational asthma [29]. A population-based incident case-control study in Finland reported that visible mould and mould odour in the workplace were associated with adult-onset asthma [6, 14]. Water damage was related to new-onset asthma among employees in two hospitals in the USA [15]. Higher level of fungal exposure in dust from a water-damaged office building was found to be associated with a higher prevalence of respiratory symptoms among the office workers in USA [30]. A prospective study from Sweden found that dampness and mould in the workplace building were associated with increased incidence and decreased remission of work-related symptoms [31].

Dampness at home and in the workplace building were related to onset of allergic rhinitis and rhinitis symptoms in our study. We have not found any other incident study on rhinitis among adults in relation to indoor dampness and mould. However, one prevalence study from China found that water damage at home was associated with current rhinitis, and visible indoor mould was associated with allergic rhinitis [27]. One Swedish population study found that mould odour was associated with pollen allergy (hay fever) [32]. Another Swedish study found that measured moisture load in the home, a marker of the excess water vapour, was associated with the prevalence of rhinitis [25].

#### Strengths and limitations

Initial selection bias is unlikely, since the participation rate for the initial ECRHS I postal questionnaire was high (86%) [17], and the response rate from RHINE II to RHINE III was reasonable (71%). Nonparticipants in RHINE II had slightly higher prevalence of respiratory symptoms, but lower prevalence of allergic rhinitis in ECRHS I. Nonparticipants in RHINE III (n=4625) had slightly higher prevalence of respiratory symptoms and asthma in RHINE II as compared to participants. However, the prevalence of dampness indicators at baseline (RHINE II) did not differ between participants and non-respondents in RHINE III. Most of the exposure data were assessed at baseline to avoid recall bias. Similar results were obtained both in the crude and multivariate analysis with adjusting of potential confounders. Thus, our results are unlikely to be influenced by selection or information bias. One limitation is that we did not ask about family history of allergies or respiratory diseases.

#### Conclusions

Dampness and mould at home and in the workplace building can be risk factors for onset of respiratory symptoms, doctor-diagnosed asthma and rhinitis. Mould odour can be a risk factor for onset of respiratory symptoms and asthma. Dampness and indoor mould can decrease remission of respiratory symptoms and allergic rhinitis. There is a need to reduce indoor dampness and mould, as they may increase the risk of respiratory illnesses.



Conflict of interest: None declared.

Support statement: The study was funded by the Icelandic Research Council, the Swedish Heart and Lung Foundation, the Vårdal Foundation for Health Care and Allergic Research, the Swedish Association Against Asthma and Allergy, the Swedish Council for Working Life and Social Research, the Swedish AFA Insurance (No. 467801100), the Bror Hjerpstedt Foundation, the Norwegian Research Council, the Norwegian Asthma and Allergy Association, the Danish Lung Association and the Estonian Science Foundation. Funding information for this article has been deposited with the Crossref Funder Registry.

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