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ORIGINAL ARTICLE

Mould and dampness in dwelling places, and onset of asthma: the population-based cohort ECRHS

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ABSTRACT

Objectives To study new onset of adult asthma in relation to dampness and moulds in dwelling places.

Methods Totally, 7104 young adults from 13 countries who participated in the European Community Respiratory Health Survey (ECRHS I and II) who did not report respiratory symptoms or asthma at baseline were followed prospectively for 9 years. Asthma was assessed by questionnaire data on asthmatic symptoms and a positive metacholine challenge test at follow-up. Data on the current dwelling was collected at the beginning and at the end of the follow-up period by means of an interviewer-led questionnaire, and by inspection. Relative risks (RR) for new onset asthma were calculated with log-binomial models adjusted for age, sex, smoking and study centre.

Results There was an excess of new asthma in subjects in homes with reports on water damage (RR 1.46; 95% CI 1.09 to 1.94) and indoor moulds (RR=1.30; 95% CI 1.00 to 1.68) at baseline. A dose-response effect was observed. The effect was stronger in those with multisensitisation and in those sensitised to moulds. Observed damp spots were related to new asthma (RR=1.49; 95% CI 1.00 to 2.22). The population-attributable risk was 3–10% for reported, and 3–14% for observed dampness/moulds.

Conclusions Dampness and mould are common in dwellings, and contribute to asthma incidence in adults.

Building dampness and indoor mould growth are common in many countries, both in dwellings and other buildings, and reviews have concluded that there is evidence for an association between damp housing conditions and respiratory symptoms in adults.^{1–4} One meta-analysis concluded that building dampness and indoor moulds are associated with approximately 30–50% increases in a variety of respiratory and asthma-related health outcomes.⁴ These conclusions are mainly based on prevalence studies, as only few longitudinal studies on adult onset of asthma in relation to dampness and moulds are available.^{5–6} Moreover, most studies are based only on questionnaire data, few have included clinical data or independent observation of dampness or moulds. The biological mechanisms behind the observed associations remains unclear. There is a complex exposure pattern related to building dampness, including house dust mite allergens,⁷ microbial compounds from moulds⁸ and bacteria,⁹ chemical emissions from degradation of

What this paper adds

- Several studies have shown associations between dampness and indoor moulds in dwellings, and asthma and respiratory symptoms. Most of these are cross-sectional studies.
- This study adds knowledge on onset of asthma in relation to dampness and moulds in dwellings in a large cohort study in adults. It includes self-reported as well as observed signs of dampness and moulds.
- The results implicate a clear need for improvements of housing conditions in Europe in order to reduce the dampness-related exposure.

building materials¹⁰ as well as volatile organic compounds of microbial origin.¹¹

The European Community Respiratory Health Survey (ECRHS) is an international multicentre epidemiological cohort study on asthma, covering 48 centres in 23 countries, started in 1990–1995.^{12–13} A cross-sectional analysis of data from ECRHS showed that mould exposure was associated with increased prevalence of asthma symptoms and bronchial hyperresponsiveness (BHR). The association was homogeneous among centres, and was stronger in subjects sensitised to moulds.⁷ From 1998 to 2002, a follow-up of the cohort was carried out (ECRHS II).¹⁴ We studied lung function decline in the cohort in relation to dampness and moulds in ECRHS II.¹⁵ This study aims to investigate new onset of asthma in the ECRHS II in relation to self-reported as well as observed building dampness and indoor moulds in the dwelling, and estimate attributable risks. Moreover, new onset was studied in relation to some other building characteristics.

METHODS

Study population and procedures

Each centre covers a source population of approximately 150 000 inhabitants within a defined geographical and administrative area. The full research protocol can be found at <http://www.ecrhs.org>. Ethics approval was obtained for each centre from the ethical committee, and written consent was obtained from each participant. We included

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subjects from the random sample who completed the main medical questionnaires both in ECRHS I and ECRHS II (1998–2002). Totally, 15 716 took part in the random sample of the ECRHS I, and 8770 completed both the medical and the indoor questionnaires in ECRHS II. Two centres (Cardiff, Geleen) omitted the indoor environment questions and were excluded, leaving 25 centres located in 11 countries in Europe and two outside Europe (Melbourne in Australia and Portland in USA). New onset asthma at the follow-up was studied, excluding subjects ($n=1666$) having wheeze without a cold, nocturnal attack of shortness of breath in the last 12 months, or a history of asthma at baseline (ECRHS I), leaving 7104 subjects to be followed prospectively. Strong exclusion criteria were chosen since non-differential disease misclassification may bias incidence risk ratios away from the null.¹⁶ New onset asthma was defined as answering 'yes' on at least one of three questions on current attacks of asthma (last 12 months), current asthma medication and nocturnal attack of shortness of breath in the last 12 months in ECRHS II. Additionally, we tested BHR in ECRHS II. BHR was defined as a fall of forced expiratory volume in the first second (FEV_1) of 20% from the highest FEV_1 , associated with a methacholine dose of 1 mg or less. For a more specific definition of asthma, we combined reports of asthma and BHR at ECRHS II.

Information on age, sex and smoking habits was collected from the interviewer-led questionnaire. Current smoker was defined as subject reporting actual smoking in the interview, or ceasing smoking less than a year ago. Smoking was defined according to ECRHS II. Subjects giving inconsistent answers on smoking habits at ECRHS I and II ($n=78$) were included in the statistical models with a separate code, as an extra dummy variable. Specific serum IgE levels against cat and timothy grass, the mould *Cladosporium herbarium* and the house dust mite *Dermatophagoides pteronyssinus* were determined by using the Pharmacia CAP System (Pharmacia Diagnostics, Uppsala, Sweden), both in ECRHS I and II. Sensitisation was defined as a specific IgE level greater than 0.35 kU/l. In addition, skin prick test to the mould *Alternaria alternata* was performed in ECRHS I, only.

Information on housing characteristics of the current dwelling was obtained from interviewer-led questionnaires performed in the beginning and in the end of the follow-up period. The current dwelling was defined as the dwelling where the subject lived when the interview took place. One question asked if the participant had moved during the follow-up period. Building data included building age, type of building, type of heating system, presence of double glazing, and type of floor cover in the bedroom and the living room. The living room was defined as the room used most at home during the days. There were questions on any history of water damage in the dwelling (broken pipes, leaks, flooding), as well as water leakage the last 12 months. Moreover, there was a question on any history of mould or mildew on any surface inside the dwelling, the location of the mould growth, and indoor mould or mildew during the last 12 months. All these questions were asked in the same way in ECRHS I and ECRHS II. ECRHS II contained one additional question on wet or damp spots on indoor surfaces other than the basement (eg, on walls, wall paper, ceilings or carpets) the last 12 months.

Initially, we analysed for four exposure variables reported at baseline for the dwelling they lived in at that time: any water damage during the last 12 months, any mould growth in the last 12 months, any mould growth in the bedroom and any mould growth in the living room. Additionally, the same exposure variables were analysed for combined exposure data from ECRHS I

and ECRHS II (ever exposed vs never exposed). In addition, associations for reports on any damp spots in the last 12 months, available only in ECRHS II, were analysed. In order to detect any dose-response relationships, two exposure scores were created. The dampness score (0–5) included number of yes answers on ever dampness (ECRHS I or II), 12 months dampness (I or II), or damp spots (ECRHS II). The mould score (0–4) included number of yes answers on ever moulds (ECRHS I or II), and 12 months on moulds (I or II). Finally, the number of rooms with reported mould growth either at ECRHS I or II was counted.

Totally, 22 centres participated in an additional indoor environmental study ('The Indoor Protocol'), including a home visit, building inspection and collecting of dust samples from the bedroom mattress in approximately 200 homes in each centre.¹⁷ Data on observed dampness and indoor moulds was obtained from 2602 homes from the random sample free of asthma at baseline. The indoor protocol contained eight questions on dampness: three for dampness, three for moulds and two for window condensation. These were grouped into three categories.

Observed dampness

Are there any damp patches on the walls or ceilings of the living room, bedroom, bathroom? (Three separate questions filled in by the inspector).

Observed mould

Is there any mould or mildew on the walls and ceilings in the living room, bedroom, bathroom? (Three separate questions filled in by the inspector).

Window condensation

Do you get condensation on your living room/bedroom window especially in the morning in the winter? (Two separate questions asked to the subject at the home visit).

Statistical analysis

Adjusted relative risks (RR) for asthma were calculated, using log-binomial models, adjusting for sex, age, smoking status and study centre. RRs were first analysed on pooled data from all 27 centres, adjusting for centre. We did a meta-analysis to test heterogeneity by centre, using standard methods for random-effects meta-analysis.¹⁸ In the meta-analysis, centres with too low a number of subjects were omitted. For estimation of population-attributable fraction (PAF) we first applied Poisson regression with robust estimation of error to establish RR, and then calculated attributable risks.¹⁹ The statistical analysis was performed using Stata V.10.0 (Stata Corporation, College Station, Texas, USA).

RESULTS

Mean follow-up time was 8.7 years (range 5.9–11.7 years). In the cohort, the prevalence of active smoking had decreased from 31.9% to 27.1%. Sensitisation to common allergens, defined as a positive IgE to cat and timothy grass, house dust mites or the mould, *C. herbarium*, was unchanged (26.2% at ECRHS I and 25.0% at ECRHS II). Totally, there were 355 cases of new onset asthma, the most common symptom was new onset of nocturnal attacks of shortness of breath. Symptoms of asthma and shortness of breath increased in a similar way in both sexes. Relatively few cases ($n=56$) had BHR (table 1).

To study possible selection bias, we compared reporting of asthma and exposure at ECRHS I among participants ($n=8770$)

Table 1 Respiratory health characteristics of the study population (n=7104)

| | ECRHS I | | ECRHS II | |
|--|---------|------|----------|------|
| | n | % | n | % |
| Smoking status | | | | |
| Never | 3267 | 46.0 | 3094 | 43.6 |
| Ex | 1569 | 22.1 | 2010 | 28.3 |
| Current | 2265 | 31.9 | 1922 | 27.1 |
| New onset asthma* | 0 | 0.0 | 355 | 5.0 |
| Current asthma attacks | 0 | 0.0 | 98 | 1.4 |
| Current asthma medication | 0 | 0.0 | 81 | 1.2 |
| Nocturnal attacks of shortness of breath | 0 | 0.0 | 254 | 3.6 |
| Parental asthma | 696 | 10.5 | NA | NA |
| Bronchial hyper-responsiveness (BHR) | 431 | 7.8 | 440 | 9.7 |
| New onset asthma combined with positive BHR† | NA | NA | 56 | 1.4 |
| Positive IgE to <i>Cladosporium herbarum</i> (mould) | 171 | 2.9 | 41 | 0.7 |
| Prick test positivity to <i>Alternaria alternata</i> (mould) | 501 | 8.5 | NA | NA |
| Positive IgE to house dust mite (der p1) | 839 | 14.2 | 756 | 13.6 |
| Positive IgE to cat or timothy grass | 409 | 6.9 | 373 | 6.7 |
| No sensitisation‡ | 4349 | 73.8 | 4161 | 75.0 |

*Current asthma attacks (last 12 months), current asthma medication or nocturnal attacks of shortness of breath (last 12 months).

†Having both new onset asthma and BHR.

‡A positive IgE refers to a specific IgE>0.35 kU/ml to any of cat, timothy grass, house dust mites or moulds.

ECRHS, European Community Respiratory Health Survey; NA, not available.

Table 2 Dampness characteristics of the current dwelling of subjects in the cohort, reported at ECRHS I and ECRHS II

| | | ECRHS I | ECRHS II | Mc Nemar's p value |
|---------------|---|----------|----------|--------------------|
| | | prev (%) | prev (%) | |
| Self-reported | (n=7104) | | | |
| Water damage | Ever*, any placet | 26.7 | 27.9 | 0.118 |
| | 12 months, any placet | 11.2 | 10.2 | 0.054 |
| Damp spots | 12 months, any placet | NA | 19.7 | |
| | Ever, any placet | 29.7 | 24.8 | <0.001 |
| Mould | 12 months, any placet | 18.6 | 16.7 | <0.001 |
| | Observed (n=2602) | | | |
| Damp spots | Any placet | NA | 18.2 | |
| | Visible | NA | 13.6 | |
| moulds | Reported | NA | 32.7 | |
| | Window condensation in winter in any room | | | |

*Ever occurring in the current dwelling.

†Found in any room in the current dwelling.

ECRHS, European Community Respiratory Health Survey; NA, not available.

and non-participants (n=6946) in the follow-up. Prevalent asthma (attacks of asthma and/or nocturnal attack of shortness of breath in the last 12 months and/or current asthma medication) in ECRHS I was 7.6% in participants and 9.4% in non-participants (p<0.001). However, the reports on exposure at baseline did not differ. The prevalence of self-reported water damage in the last 12 months at ECRHS I was 11.7% in participants and 12.8% in non-participants in the follow-up (p=0.50). The prevalence of self-reported moulds in the last 12 months at ECRHS I was 19.7% in participants and 20.1% in non-participants (p=0.59). The prevalence of self-reported moulds in the bedroom at baseline was 11.9% in participants and 12.4% in non-participants (p=0.40). The prevalence of self-reported moulds in the living room at ECRHS I was 6.2% in participants and 6.7% in non-participants (p=0.27).

Self-reported water damage, damp spots and indoor mould growth in the current dwelling was common at both surveys. There was a slight but significant decrease of dampness and moulds during the follow-up. Self-reported moulds were most common in the bathroom and the bedroom, and least common in the kitchen. Totally, 49.7% reported water damage either at ECRHS I or II (damp score>0), and 41.7% reported indoor mould growth (damp score>0). The prevalence of observed dampness and moulds were lower, but window condensation in winter was common (table 2).

First, we analysed RR for new onset asthma in relation to exposure at baseline. RR for onset of asthma was increased among those reporting water damage in the last 12 months and moulds in the last 12 months. The population-attributable fractions for self-reported dampness and moulds were between 2% and 5% for asthma, and 3% and 6% for asthma+BHR. Then we analysed associations for those ever exposed (ever vs never). RR for new onset asthma was increased among subjects reporting water damage in the last 12 months, damp spots in the last 12 months,

and indoor moulds in the last 12 months. The associations were most pronounced for moulds in the bedroom and in the living room. New onset asthma combined with BHR was increased for self-reported moulds in the living room. The PAFs for self-reported dampness and moulds were between 3% and 10% for new onset asthma, and 3% and 8% for asthma+BHR (table 3).

As a next step, we stratified for gender. The risk estimates for new onset asthma in relation to being ever exposed to dampness and moulds were similar in men and women, with no significant sex-interaction except for moulds in the living room (p=0.03 for interaction) (see online supplementary table E1). The RR for new onset asthma in relation to self-reported moulds in the living room was 0.89 (95% CI 0.51 to 1.52) for men and 2.02 (95% CI 1.40 to 2.92) for women. When stratifying for smoking, we found no interaction for current smoking, but lower risk estimates for water damage during the last 12 months (p=0.09), and higher risk estimates for mould score >0 (p=0.03) and moulds in the bedroom (p=0.06) in ex-smokers as compared with never smokers (data not shown). Additionally, we stratified the material with respect to sensitisation at baseline, analysing ever exposed versus never exposed. Among subjects with no sensitisation (n=3329) there were associations between new onset asthma and self-reported water damage in the last 12 months (RR=1.07; 95% CI 1.07 to 2.39) and self-reported mould in the last 12 months (RR=1.57; 95% CI 1.09 to 2.27). Among subjects with sensitisation to cat and timothy grass, but not to house dust mites or mould (n=1303), there were associations between new onset asthma and self-reported mould in the living room in the last 12 months (RR=2.05; 95% CI 1.17 to 3.61). Among subject with sensitisation to house dust mites but not to mould (n=1064), there were associations between new onset asthma and self-reported mould in the bedroom in the last 12 months (RR=1.87; 95% CI 1.12 to 3.10). Among subjects sensitised to mould at baseline (*C herbarium* or *A alternata*) (n=339),

Table 3 New onset of asthma in relation to self-reported dampness and moulds at baseline and at baseline or follow-up (ever vs never exposed)

| | Exposed (%) | Asthma (n=6984) | | | Asthma and BHR (n=6615) | | |
|---|-------------|--------------------|----------------|---------|----------------------------|----------------|---------|
| | | Adjusted RR* | 95% CI | PAF (%) | Adjusted RR* | 95% CI | PAF (%) |
| Exposed at baseline (ECRHS I) | | | | | | | |
| Water damage in last 12 months | 11.3 | 1.46 | (1.09 to 1.94) | 4.8 | 1.48 | (0.71 to 3.07) | 5.7 |
| Mould in last 12 months | 18.6 | 1.30 | (1.00 to 1.68) | 5.1 | 1.16 | (0.61 to 2.22) | 3.0 |
| Mould in bedroom (ever) | 11.4 | 1.08 | (0.79 to 1.48) | 1.0 | 0.97 | (0.43 to 2.16) | – |
| Mould in living room (ever) | 6.1 | 1.34 | (0.91 to 1.97) | 2.0 | 1.50 | (0.61 to 3.69) | 2.7 |
| Ever exposed (ECRHS I and/or ECRHS II) versus never exposed | | | | | | | |
| Water damage in last 12 months | 19.1 | 1.29 | (1.00 to 1.66) | 5.1 | 1.22 | (0.63 to 2.36) | 4.3 |
| Mould in the last 12 months | 28.9 | 1.36 | (1.08 to 1.70) | 10.3 | 1.23 | (0.70 to 2.19) | 7.9 |
| Mould in bedroom (ever) | 18.4 | 1.43 | (1.12 to 1.84) | 4.9 | 1.14 | (0.57 to 2.26) | 2.5 |
| Mould in living room (ever) | 10.0 | 1.48 | (1.10 to 2.01) | 3.4 | 2.24 | (1.15 to 4.36) | 8.0 |
| Damp spots, last 12 months† | 19.7 | 1.28 | (1.00 to 1.65) | 5.3 | 0.96 | (0.49 to 1.89) | – |

*Adjusted for age, sex, smoking status and centre.

†Available only at ECRHS II.

BHR, bronchial hyper-responsiveness; ECRHS, European Community Respiratory Health Survey; PAF, population attributable fraction; RR, relative risks

there were associations between new onset asthma and self-reported water damage in the last 12 months (RR=4.57; 95% CI 2.10 to 9.94) and self-reported mould in the living room (RR=4.38; 95% CI 1.80 to 10.62) (see online supplementary table E2).

As a next step, heterogeneity between centres was tested, comparing those reporting being ever exposed with those never exposed. Meta-analysis of new onset asthma in relation to water damage or damp spots in the last 12 months, did not reveal any heterogeneity between centres (p=0.75 and 0.81, respectively), but some heterogeneity was found for indoor moulds in the last 12 months (p=0.09) (see online supplementary figures E1–E3).

In order to detect any dose-response relationship, associations were analysed for the dampness score, the mould score, and number of rooms with reported mould growth. There was a significant dose-response for dampness score and new onset

asthma, and a significant dose-response for the mould score and new onset asthma. Moreover, there was an association between the number of rooms with mould growth and new onset asthma (p=0.011), and a borderline association (p=0.057) for new onset asthma with BHR (table 4). The two scales had a weak correlation (Spearman's r 0.35), and were independent scales (p<0.001) but with some overlap. Totally, 27% had both the mould score >0 and the dampness score >0.

The RR for new onset asthma was increased in homes with any damp spots (in a bedroom, living room or bathroom) observed at the home inspection (table 5). The PAFs for observed dampness and moulds were between 2% and 8% for asthma, and 6% and 4% for asthma+BHR. There were no associations between new onset asthma and observed mould.

Totally, 49.3% had moved to another home during the follow-up period. The proportion of new onset asthma did not

Table 4 New onset of asthma in relation to self-reported dampness and mould scores and number of rooms with mould, ECRHS I and II

| | n % | Asthma | | | Asthma with BHR | | |
|----------------------|--------|--------------|----------------|---------|-----------------|----------------|---------|
| | | Adjusted RR* | 95% CI | p Value | Adjusted RR* | 95% CI | p Value |
| Damp score | | | | | | | |
| | n=5870 | | | | | | |
| 0 | 50.3 | 1 | (ref.) | | 1 | (ref.) | |
| 1–2 | 37.8 | 1.14 | (0.90 to 1.45) | 0.284 | 0.83 | (0.44 to 1.56) | 0.571 |
| 3–5 | 11.9 | 1.28 | (0.91 to 1.81) | 0.150 | 1.26 | (0.55 to 2.87) | 0.580 |
| Trend | | 1.09 | (1.00 to 1.20) | 0.047 | 1.03 | (0.81 to 1.30) | 0.835 |
| Mould score | | | | | | | |
| | n=6698 | | | | | | |
| 0 | 58.4 | 1 | (ref.) | | 1 | (ref.) | |
| 1–2 | 30.8 | 1.05 | (0.82 to 1.33) | 0.722 | 0.62 | (0.31 to 1.25) | 0.181 |
| 3–4 | 10.8 | 1.73 | (1.27 to 2.37) | 0.001 | 1.83 | (0.88 to 3.80) | 0.105 |
| Trend | | 1.12 | (1.03 to 1.22) | 0.007 | 1.08 | (0.87 to 1.34) | 0.477 |
| Rooms with mould (n) | | | | | | | |
| | n=6776 | | | | | | |
| 0 | 76.0 | 1 | (ref.) | | 1 | (ref.) | |
| 1 | 21.2 | 1.30 | (1.02 to 1.66) | 0.035 | 1.24 | (0.66 to 2.32) | 0.511 |
| 2–3 | 2.8 | 1.43 | (0.83 to 2.48) | 0.196 | 2.41 | (0.82 to 7.05) | 0.108 |
| Dose-response | | 1.17 | (1.04 to 1.32) | 0.011 | 1.31 | (0.99 to 1.74) | 0.057 |

*Adjusted for age, sex, smoking status and centre. RR, relative risks.

Table 5 New onset of asthma in relation to observed dampness, moulds and window condensation

| | ECRHS II Exposed (%) | Asthma (n=2484) | | PAF (%) | Asthma and BHR (n=1557) | | PAF (%) |
|--|-------------------------|--------------------|----------------|---------|----------------------------|-----------------|---------|
| | | Adjusted RR* | 95% CI | | Adjusted RR* | 95% CI | |
| Any damp spots | 18.3 | 1.49 | (1.00 to 2.22) | 8.20 | 1.88 | (0.84 to 4.22) | 14.40 |
| Damp spots in bedroom | 7.0 | 1.63 | (0.95 to 2.78) | 4.10 | 2.57 | (0.91 to 7.27) | 8.70 |
| Damp spots in living room | 5.8 | 0.98 | (0.46 to 2.07) | – | NA | NA | – |
| Damp spots in bathroom | 9.8 | 1.21 | (0.72 to 2.03) | 2.21 | 1.62 | (0.61 to 4.30) | 6.93 |
| Any visible mould | 13.6 | 1.15 | (0.71 to 1.85) | 1.90 | 1.74 | (0.68 to 4.45) | 9.50 |
| Visible mould in bedroom | 4.9 | 1.56 | (0.83 to 2.92) | 2.80 | 2.51 | (0.61 to 10.40) | 6.40 |
| Mould spots in living room | 2.9 | 0.99 | (0.37 to 2.65) | – | 0.97 | (0.12 to 7.56) | – |
| Mould spots in bathroom | 9.2 | 0.79 | (0.41 to 1.51) | – | 1.14 | (0.34 to 3.86) | 1.09 |
| Reported window condensation in winter in any room | 32.7 | 1.07 | (0.75 to 1.53) | 2.00 | 1.43 | (0.67 to 3.07) | 13.20 |

*Adjusted for age, sex, smoking status and centre.

BHR, bronchial hyper-responsiveness; ECRHS, European Community Respiratory Health Survey; NA, not available; PAF, population attributable fraction, RR, relative risks; NA, not available because to small numbers for statistical calculations.

differ between those moving and not moving (data not shown). Moreover, there were no differences in reports on dampness and moulds between those participating and not participating in the home inspections (data not shown). Finally, we compared RR for new onset asthma in relation to self-reported dampness and moulds (ever vs never exposed) in those moving and not moving to another home during the follow-up. RRs for new onset asthma were similar in movers and non-movers for all types of exposure with overlapping CIs. Among those living in the same home, there was an association between new onset asthma and self-reported mould in the bedroom (RR=1.61; 95% CI 1.13 to 2.30) and self-reported damp spots in the last 12 months (RR=1.49; 95% CI 1.06 to 2.09). Among those moving to another home, there was an association between new onset asthma and self-reported water damage (RR=1.48; 96% CI 1.05 to 2.09). When analysing associations for new onset asthma combined with BHR, those staying in the same home had always higher RR as compared with those moving. There were significant associations for self-reported mould in the bedroom (RR=2.64; 95% CI 1.10 to 6.33) and self-reported mould in the living room (RR=3.79; 95% CI 1.64 to 8.73) among non-movers, while movers had no significant associations for any type of exposure (see online supplementary table E3).

DISCUSSION

Our longitudinal study indicated that dampness and moulds can increase the risk for adult new onset asthma. Associations were found both for self-reported and observed exposure. Moreover, there were associations between new onset asthma and the dampness score, mould score and number of rooms with indoor moulds, indicating a dose-response effect. Similar associations, but higher population-attributable risk, was found when considering exposure data from both ECRHS I and ECRHS II (ever vs never exposed). The similarities between these different analysis makes recall bias less probable.

There are certain limitations of the study. Exposure to moulds and dampness was assessed by questionnaire as well as independent observation in the current home of a random subsample. Response bias could have influenced the associations for self-reported exposure, but is less likely to influence associations with observed dampness and indoor mould. However, associations were found for water damage and mould at baseline which is a strength, because the exposure is measured before new

onset of asthma. We did not adjust for season, but the recall period for both symptoms and exposure was either 12 months or ever (ever for exposure in specified rooms). Another limitation is that we only ask for dampness and mould in the current home, and about half the subjects had moved to another home during the follow-up period. Moreover, we have no information as to when the new onset asthma occurred, so in some cases, the new onset could have occurred before the exposure. This limitation would most likely lead to non-differential misclassification.

The study covered different climate zones, and the associations were consistent across areas. It is possible that the international variation of asthma prevalence partly could be explained by variation of this exposure. An ecological analysis has reported a higher prevalence of asthma at oceanic climate.²⁰ The prevalence of self-reported dampness and indoor moulds were high in ECRHS I as well as ECRHS II, indicating only minor improvement of the housing conditions during the 1990s in Europe. There were different patterns of associations for mould growth as compared with water leakage, which indicates that there could be different pathways for the effects of building dampness on asthma. Increased water content in building materials or on indoor surfaces can lead to visible mould growth, as well as increase of house dust mites, hidden microbial growth inside the building, and chemical degradation of building materials without microbial growth.¹⁰

In the current follow-up study, we found significant associations between dampness and mould and new onset asthma, which is in agreement with a previous cross-sectional analysis within ECRHS I.⁷ In another cohort study in preschool children in the Swedish DBH study, there were no associations between dampness/moulds in the dwellings and new onset asthma, except for mouldy odour. By contrast, they found strong associations between prevalence of asthma and dampness or mould in the initial cross-sectional analysis at baseline.²¹ These differences indicate further need to analyse dampness/moulds in dwellings as a risk factor for new onset asthma in cohorts of different ages and in different climate zones, and using different asthma definitions.

A higher RR for new onset asthma was observed for those with multiple sensitisation at baseline, especially if they were sensitised to moulds. Totally 11.8% were sensitised to the moulds *C herbarum* and/or *A alternata*, two common moulds which can be

found in the indoor environment.^{22, 23} There are different possible mechanisms for respiratory effects of mould exposure. Fungi can be sources for allergens that can cause allergic asthma. It has been shown that mould allergy is related to severity of asthma,²⁴ but subjects sensitised to mould are usually sensitised to many other allergens, making it difficult to isolate the effect of the mould allergy. Moreover, moulds may contain compounds which have inflammatory effects, for instance, β -1-3-glucans in the cell wall structure,²⁵ and moulds can produce volatile organic compounds¹⁰ and mycotoxins.²⁶

Sex differences with respect to sensitivity to environmental exposure are an important issue. In general, men and women had similar responses to dampness and mould in our study, except for moulds in the living room, where women had a higher risk. The reason for a higher risk in women is unclear, but it could be that women generally spend more time in the home, and might more often do cleaning or other activities in the living room which could stir up settled dust containing fungal contaminants. There are few studies on sex differences in relation to mould and dampness exposure. One study from Canada suggests that women may be more susceptible to effects of dampness than men are.²⁷

CONCLUSION

Dampness and indoor moulds in dwellings are related to an increased incidence of asthma in adults. The effect seems to be stronger in those with multiple sensitivity, and in those sensitised to moulds. About 5–15% of adult onset asthma could be attributed to dampness-related exposure at home. There is a clear need for improvements of housing conditions in order to reduce the dampness-related exposure to counteract the increase of asthma in adults.

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