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A prospective study on the role of smoking, environmental tobacco smoke, indoor painting and living in old or new buildings on asthma, rhinitis and respiratory symptoms

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ABSTRACT

We studied associations between tobacco smoke, home environment and respiratory health in a 10 year follow up of a cohort of 11,506 adults in Northern Europe. Multilevel logistic regression models were applied to estimate onset and remission of symptoms. Current smokers at baseline developed more respiratory symptoms (OR = 1.39–4.43) and rhinitis symptoms (OR = 1.35). Starting smoking during follow up increased the risk of new respiratory symptoms (OR = 1.54–1.97) and quitting smoking decreased the risk (OR = 0.34–0.60). ETS at baseline increased the risk of wheeze (OR = 1.26). Combined ETS at baseline or follow up increased the risk of wheeze (OR = 1.27) and nocturnal cough (OR = 1.22). Wood painting at baseline reduced remission of asthma (OR 95%CI: 0.61, 0.38–0.99). Floor painting at home increased productive cough (OR 95%CI: 1.64, 1.15–2.34) and decreased remission of wheeze (OR 95%CI: 0.63, 0.40–0.996). Indoor painting (OR 95%CI: 1.43, 1.16–1.75) and floor painting (OR 95%CI: 1.77, 1.11–2.82) increased remission of allergic rhinitis. Living in the oldest buildings (constructed before 1960) was associated with higher onset of nocturnal cough and doctor diagnosed asthma. Living in the newest buildings (constructed 1986–2001) was associated with higher onset of nocturnal breathlessness (OR = 1.39) and rhinitis (OR = 1.34). In conclusion, smoking, ETS and painting indoor can be risk factors for respiratory symptoms. Wood painting and floor painting can reduce remission of respiratory symptoms. Smoking can increase rhinitis. Living in older buildings can be a risk factor for nocturnal cough and doctor diagnosed asthma. Living in new buildings can increase nocturnal dyspnoea and rhinitis.

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1. Introduction

The prevalence of asthma in different parts of the world can range from 1 to 18% (GINA Report, 2017). Adult asthma is usually more common in developed western countries (6.0%–12.0%) (Lundback et al., 2016). Rhinitis is a common disease in developed countries. In USA and Europe, 10%–20% of the population have allergic rhinitis (Ozdoganoglu and Songu, 2012). However, few studies are available on incidence of asthma or allergic rhinitis among adults (Wang et al., 2019a). Home environment exposure can influence respiratory health in children (WHO, 2009; Quansah et al., 2012; Mendell, 2007; Mendell et al., 2011). One review concluded that chemical emission-related materials or activities in dwellings can impair childhood respiratory health (Mendell, 2007). However, few have studied associations between indoor environment in homes and respiratory health in adults (Mitchell et al., 2007; Jie et al., 2011).

Prospective studies have demonstrated that smoking is a risk factor for onset of asthma (King et al., 2004; Polosa et al., 2008), but we found no prospective studies on smoking and onset of rhinitis. Environmental tobacco smoke (ETS) at home is still an important issue. A multicentre study from Europe found that ETS was more common in the home (13%–40%) than in the workplace (3%–32%) (Heck et al., 2010). ETS exposure at home was less common in Northern Europe as compared to central or Southern Europe (Heck et al., 2010; Olivieri et al., 2019). A recent European multicentre study from 2010 to 2014 reported that 9% were exposed to ETS at home (Olivieri et al., 2019). This study reported that global smoking bans failed to reduce ETS exposure in homes. A number of prospective studies have demonstrated that environmental tobacco smoke (ETS) is associated with asthma onset in adults (Flexeder et al., 2019; Coogan et al., 2015; Lajunen et al., 2013; Thorn et al., 2001; Jaakkola et al., 2003) and that ETS can increase onset of allergic rhinitis in adults (Hur et al., 2014). However, there is a need to study respiratory effects of ETS in Northern Europe where the ETS exposure is relatively low.

Few studies have investigated effects of chemical emissions at home on adult respiratory symptoms, asthma or rhinitis. Two cross-sectional studies from China found that home renovation increased the risk of allergic rhinitis (Dong et al., 2013; Li et al., 2014). One incident case-control study from Finland found that plastic wall materials in the workplace building and floor-levelling plaster at home was related to adult onset asthma (Jaakkola et al., 2006). One study from Sweden reported that formaldehyde and VOCs at home increased the risk of nocturnal breathlessness among adults (Norback et al., 1995). Indoor painting is often a part of the renovation and can cause chemical emissions to the home environment. However, little knowledge is available on domestic indoor painting and adult respiratory health. One recent review (Canova et al., 2013) on domestic painting as a risk factor for asthma found only one study on adult asthma. This study reported that domestic exposure to newly painted surfaces, especially painted wood details and kitchen painting increased adult asthma (Wieslander et al., 1997a). We found no previous cohort studies on respiratory effects of indoor painting.

Building age is an important determinant of the indoor environment, since different construction techniques and building materials have been used in different construction periods. One example is that the energy crisis in 1970' initiated measures to reduce energy consumption in Swedish buildings (Engvall et al., 2000). Buildings constructed after this period in Sweden were more air-tight buildings and had lower ventilation flow.

Few studies are available on the role of construction year for respiratory illnesses in adults. One Swedish study found that living in buildings constructed after 1975 was associated with airway infections (Fu et al., 2015). Another study from Sweden demonstrated that living in buildings constructed 1961–1975 increased asthma exacerbation and living in buildings constructed 1976–1985 was associated with rhinitis (as compared to buildings constructed before 1960) (Wang et al., 2014a, 2017abib_Wang_et_al_2017abib_Wang_et_al_2014a). One study from China reported that adult asthma and allergic rhinitis were more common in older than in newer buildings (Wang et al., 2019b). We found no prospective studies on building age as a risk factor for onset of respiratory symptoms, asthma or rhinitis.

Few prospective studies exist on associations between the home environment and respiratory health in Northern Europe. The main aim of our study is to investigate associations between smoking, environmental tobacco smoke, indoor painting and building construction year and onset and remission of adult respiratory symptoms, asthma and rhinitis.

2. Materials and methods

2.1. Ethics statement

This study was approved by the appropriate ethic board in each centre. All participants gave informed consent prior to participation.

2.2. Study design and target population

The study is based on a 10 year follow up of Respiratory Health in Northern Europe (RHINE). The RHINE II study is a follow up study of subjects from seven Nordic study centres from the European Community Respiratory Health Survey stage I (ECRHS I). The seven centres in northern Europe included Reykjavik in Iceland, Bergen in Norway, Umeå, Uppsala and Gothenburg in Sweden, Aarhus in Denmark and

Tartu in Estonia. The ECRHS I study, conducted in 1989–1992, included randomly selected subjects (aged 20–44 years) from the population register in each centre (3000–4000 subjects per centre). A postal questionnaire was then sent to these subjects.

A total of 21,681 subjects from these seven centres participated in ECRHS I (response rate 86%) (Johannessen et al., 2014). In 1999–2000, they received a follow up postal questionnaire (RHINE II), with questions on respiratory health and the home environment. In 2010–2012, the RHINE II participants were invited for a second follow up (RHINE III), with identical questions on respiratory health as in RHINE II. Totally 11,506 subjects participated in RHINE II and RHINE III (response rate 71%). A flow chart of the study design is shown in Fig. 1. Participation was defined as reporting at least one of five questions on respiratory symptoms (wheeze, nocturnal chest tightness, nocturnal attacks of breathlessness, nocturnal cough or asthma attack, see detailed description below). In this article, RHINE II was defined as the baseline study and the RHINE III as the follow up study.

2.3. Respiratory health

Questions on respiratory symptoms were the same at baseline and at follow up. There were five questions on respiratory symptoms in the last 12 months, including wheeze, nocturnal chest tightness, nocturnal attacks of breathlessness, nocturnal cough and asthma attack. There was one question on productive cough, asking "Do you usually bring up phlegm or do you have phlegm in your lungs which you have difficulty bringing up ?". One additional question asked about current asthma medication (including inhalers, aerosols or tablets). Moreover, there was one question on doctor diagnosed asthma and two questions on rhinitis (allergic rhinitis and rhinitis symptoms). Current asthma was defined as having either asthma attacks or asthma medication, or both (Plaschke et al., 2000). The questions have been described in detail in a previous publication (Wang et al., 2019a).

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

Onset of a particular respiratory symptom (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 it at follow up (Gunnbjornsdottir et al., 2006). Participants with doctor diagnosed asthma at baseline were excluded when calculating onset of these respiratory symptoms.

Remission of a particular respiratory symptom was defined as reporting the particular symptom at baseline but not at follow up. Participants with doctor diagnosed asthma at baseline were not excluded when calculating remission of respiratory symptoms.

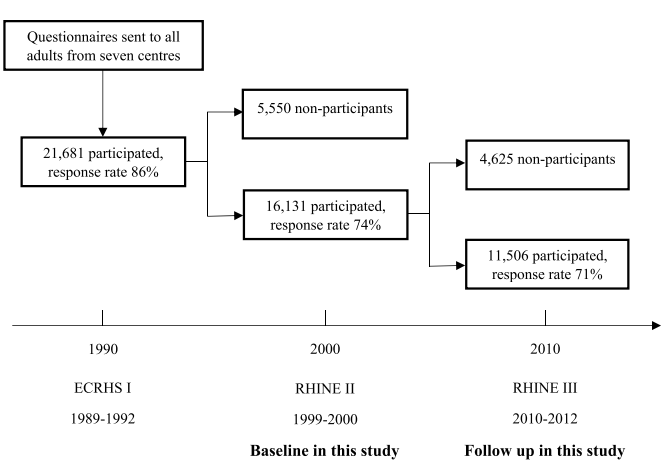

Fig. 1. Flow chart of the study design.

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

2.4. Home environment

Environment tobacco smoke (ETS) was asked about at baseline and at follow up, using the same question: "Does tobacco smoking take place in your present home?". There were four alternatives: yes, daily; yes, often, 1–4 times per week; yes sometimes, 1–3 times per month; no, never. One new variable ("ETS at baseline") was created, coded as 0 if the answer was "no" at baseline, coded as 1 if the answer was "yes, sometimes, 1–3 times per month", and coded as 2 if the answer was yes daily" or "yes, often, 1–4 times per week". Another variable ("ETS at follow up") was defined in the same way. A combined ETS variable was then created, coded as 0 if both ETS at baseline and ETS at follow up equal 0, coded as 2 if any of the two variables equals 2, and coded as 1 if any of the two variables equals 1 but not 2.

The baseline questionnaire included questions on indoor painting, type of dwelling and construction year of the building. Two questions asked on indoor painting: "Have the inside of your dwelling been painted in the past 12 months?" and "What was painted?", giving six alternatives for location of the indoor painting: ceilings, walls, carpentry/wooden details, floors, metal pipes/radiators and other. There was one question on type of dwelling, with four alternatives: detached, semi-detached, apartment and other. Moreover, there was one question on construction year of the building.

2.5. Other covariate variables

Data on gender and age were collected from the baseline data. Height and weight were obtained from baseline and follow up data. Body mass index (BMI) at baseline and follow up were calculated as the ratio of weight in kilograms to height in meters squared (kg/m^2). Change of BMI from baseline to follow up was calculated. Information on education level was only available at follow up. Education level was categorized as primary school, high school or university education. Information on smoking habit (never/ex-smokers/current smokers) was available at baseline and at follow up. A variable on change of smoking from baseline to follow up (no change/start smoking/quit smoking) was created. Smoking at baseline or follow up was defined as: yes, current smokers at baseline or follow up; no, never smokers or ex-smokers at baseline and follow up. Questions on building dampness at baseline asked about four dampness indicators in the past 12 months: water damage (yes/no), floor dampness (yes/no), visible mould (yes/no) and mould odour (yes/no). Questions on building dampness at follow up included only three dampness indicators (in the past 12 months): water damage (yes/no), floor dampness (yes/no) and visible mould (yes/no). A variable called any dampness at baseline (yes/no) was created based on at least one yes answer to the four dampness indicators at baseline. Change of dampness at home from baseline to follow up was calculated, including data on three dampness indicators that available both at baseline and follow up (water damage, floor dampness and visible mould). A detailed description of the dampness questions is available in a previous publication (Wang et al., 2019a).

2.6. Statistical analysis

The statistical analysis was performed by Stata 15.1 (Stata Corporation, College Station, Texas, USA). Initially, two level (centre, individual) logistic regression models were used to estimate associations between smoking habits and onset of respiratory symptoms, doctor diagnosed asthma, allergic rhinitis and rhinitis symptoms. The models included gender, age, smoking at baseline, change of smoking habit from baseline to follow up, BMI at baseline, change of BMI from baseline to follow up, education level at follow up, any dampness at baseline and change of dampness from baseline to follow up (nine covariates).

Subjects with doctor diagnosed asthma at baseline were excluded when analysing onset of respiratory symptoms. Associations between ETS at baseline, the combined ETS variable, painting, construction year and type of dwelling and onset of respiratory symptoms, doctor diagnosed asthma, allergic rhinitis and rhinitis symptoms were analysed using two level logistic regression models, adjusting for the nine covariates.

Stratified analyses (current smokers at baseline or follow up vs. never smokers or ex-smokers at baseline and follow up) were performed, adjusting for the nine covariates. Additional two level logistic regression models were conducted to study the associations between ETS at baseline and onset of symptoms, including only those were current smokers at baseline and adjusting for how much participants smoked at baseline.

Interaction between ETS at baseline and smoking at baseline or follow up (yes/no) in association with onset of respiratory symptoms, doctor diagnosed asthma, allergic rhinitis and rhinitis symptoms were analysed ($p < 0.1$ was considered to be significant for interaction analyses).

Moreover, two level logistic regression models were applied to estimate associations between home environment factors and remission of respiratory symptoms and allergic rhinitis, adjusting for the nine covariates. In order to detect heterogeneity between centres, adjusted ORs were calculated separately for each centre. An average effect estimate was derived, and heterogeneity between centres was examined ($p < 0.1$) using standard methods for random effects meta-analysis. Associations were expressed as odds ratios (OR) with a 95% confidence interval (CI), and 5% significance level.

3. Results

A total of 11,506 subjects participated in RHINE II and RHINE III, 54.3% were females. The mean age at baseline was 40 (SD = 7.3) years. The mean follow up time was 11.3 ± 1.1 years. Current smoking (26.3%) and ex-smoking (26.4%) were common at baseline. During follow up, 2.4% started smoking and 12.1% quitted smoking. Non-participants in RHINE II and RHINE III had slightly higher prevalence of respiratory symptoms as compared to participants in RHINE II and RHINE III (Wang et al., 2019a).

Table 1 shows the prevalence of ETS inside the home, stratified for smoking habits. Totally 37.3% of the homes had ETS at baseline, 13.0% had ETS at follow up, and 39.2% had ETS at baseline or follow up. Smokers were more often exposed to ETS at baseline and at follow up ($p < 0.001$). The prevalence of smokers not exposed to ETS increased from 29.0% at baseline to 67.9% at follow up, suggesting a strong trend of avoiding smoking inside of the home.

Data on the home environment is shown in Table 2. Indoor painting in the last 12 months was common (36.9%), 14.6% had painting on wood and 4.5% had painting on floor. Data on painting on ceilings (21.8%), walls (29.6%), metal pipes/radiators (8.2%) and other places (1.9%) is given in text only. Most of the participants lived in a detached/semidetached house (59.8%) or apartment (38.5%) at baseline. A third (36.3%) lived in buildings constructed before 1960. Exposure to ETS at baseline was most common in Aarhus, any indoor painting was most common in Reykjavik, and wood painting and floor painting were most common in Tartu. ETS, indoor painting, wood painting and floor painting were more common in homes with dampness at baseline ($p < 0.001$). Newer buildings had less dampness at baseline.

Onset and remission rates of respiratory illnesses are shown in Table 3. A total of 9.9% had new onset of wheeze, 9.3% had new onset of productive cough, 4.3% had new onset of doctor diagnosed asthma, and 9.5% had new onset of allergic rhinitis. Onset of rhinitis symptoms was most common (25.9%). The remission rate was high, 47.5%–66.9% of the participants with a particular respiratory symptom at baseline were free from having that symptom at follow up.

In the crude data analyses (Table 3), ETS at baseline was related to onset of wheeze, nocturnal chest tightness, nocturnal attacks of breathlessness, nocturnal cough, productive cough, current asthma, allergic rhinitis and rhinitis symptoms. Any indoor painting and wood painting were related to onset of allergic rhinitis. Floor painting was related to onset of nocturnal chest tightness, nocturnal attacks of breathlessness and productive cough. Moreover, weekly exposure to ETS at baseline was related to less remission of wheeze. Floor painting was related to less remission of nocturnal cough. Weekly exposure to ETS at baseline, any indoor painting and floor painting were all related to remission of allergic rhinitis. Onset and remission rates of respiratory symptoms, asthma, allergic rhinitis and rhinitis symptoms stratifying for construction year are shown in Supplement Table 1. Onset of nocturnal cough was most common in buildings constructed before 1960 ($p = 0.043$). Onset of rhinitis symptoms was most common among those living in buildings constructed during 1986–2001 ($p = 0.001$). No associations were found between type of dwelling (apartment vs. detached/semi-detached house) and onset or remission of respiratory symptoms, asthma or rhinitis (data not shown).

Health associations for smoking habits and ETS are shown in Table 4. Being an ex-smoker at baseline was related to onset of wheeze and rhinitis symptoms. Being a current smoker at baseline was associated with onset of all respiratory health variables except doctor diagnosed asthma and allergic rhinitis. Starting smoking during follow up increased onset of wheeze, nocturnal cough and productive cough. Quitting smoking during follow up decreased onset of wheeze and productive cough. Weekly exposure to ETS at baseline was related to onset of wheeze. Weekly exposure to ETS at baseline or follow up was related to onset of wheeze and nocturnal cough.

Table 5 shows associations between respiratory health and painting and construction year. There was a trend of association between wood painting and onset of allergic rhinitis ($p = 0.05$). Floor painting was associated with onset of productive cough. Participants living in buildings constructed from 1961 to 1975 had less onset of nocturnal cough, and those living in buildings constructed from 1976 to 1985 had less onset nocturnal cough and doctor diagnosed asthma. Living in buildings constructed from 1986 to 2001 increased onset of nocturnal breathlessness and rhinitis symptoms but reduced onset of nocturnal cough. Increased building age was associated with higher onset of nocturnal cough ($p = 0.016$). There was no difference in health associations between apartment and detached/semi-detached house (data not shown). The associations between construction year and onset of respiratory symptoms, asthma and rhinitis did not change with additional adjustment of type of dwelling in the models.

Associations between ETS at baseline and respiratory health are shown in Table 6. Among current smokers at baseline or at follow up, weekly exposure to ETS was associated with onset of wheeze, nocturnal chest tightness and productive cough. The associations between ETS and wheeze and nocturnal chest tightness were stronger among smokers than non-smokers (p value for interaction were 0.083 and 0.021,

Table 1
The prevalence of ETS (%) inside the home (n = 11,506).

		Total	Smoker[a]		
			Yes	No	p[b]
ETS at baseline	Never	62.3	29.0	75.4	
	Sometimes	15.7	16.0	15.7	
	Weekly	22.0	55.0	8.9	**<0.001**
ETS at follow up	Never	87.0	67.9	94.6	
	Sometimes	4.1	8.0	2.6	
	Weekly	8.9	24.2	2.8	**<0.001**
Combined ETS [c]	0	60.8	26.4	74.3	
	1	15.7	15.6	15.9	
	2	23.5	58.0	9.8	**<0.001**

[a] Smoker was defined as: yes, current smokers at baseline or follow up; no, ex/never smokers at baseline and follow up.
[b] Chi-square test.
[c] ETS at baseline or follow up: coded as 0, never; 1, sometimes but not weekly; 2, weekly.

Table 2
Home environment characteristics in seven centres in Northern Europe (%) (n = 11,506).

		Total (%)	Aarhus (%)	Reykjavik (%)	Bergen (%)	Gothenburg (%)	Umeå (%)	Uppsala (%)	Tartu (%)	p[a]	Any dampness[b]		p
											Yes	No	
ETS at baseline	Never	62.3	30.7	49.6	57.6	76.2	83.6	85.4	53.3		58.5	63.4	
	Sometimes	15.7	35.0	17.7	17.2	9.1	6.6	6.5	14.8		16.8	15.3	
	Weekly	22.0	34.3	32.7	25.2	14.7	9.8	8.1	31.9	**<0.001**	24.7	21.3	**<0.001**
Any indoor painting	Yes	36.9	42.3	48.5	39.8	31.4	27.0	29.6	40.6		43.3	35.1	
	No	63.1	57.7	51.5	60.2	68.6	73.0	70.4	59.4	**<0.001**	56.7	64.9	**<0.001**
Wood painting	Yes	14.6[c]	17.7	7.4	12.6	Missing[d]	13.2	16.5	22.2		18.0	13.7	
	No	85.4	82.3	92.6	87.3	Missing[d]	86.8	83.5	77.8	**<0.001**	82.0	86.3	**<0.001**
Floor painting	Yes	4.5[c]	3.5	4.4	2.8	Missing[d]	2.7	2.7	14.5		6.6	3.9	
	No	95.5	96.5	95.6	97.2	Missing[d]	97.3	97.3	85.5	**<0.001**	93.4	96.1	**<0.001**
Construction year	Before 1960	36.3	45.3	27.3	31.7	39.4	38.5	39.5	28.7		41.0	35.1	
	1961–1975	23.3	22.8	22.8	20.6	29.9	21.7	23.5	22.7		25.0	22.7	
	1976–1985	20.5	15.7	21.6	20.5	14.4	27.1	21.1	24.0		19.5	20.9	
	1986–2001	19.9	16.3	28.4	27.1	16.4	12.7	16.0	24.6	**<0.001**	14.6	21.3	**<0.001**

[a] Chi-square test.
[b] Any dampness at baseline was defined as occurrence of any of the four dampness indicators in the past 12 months: water damage, floor dampness, visible mould or mould odour.
[c] The total prevalence was based on data from Aarhus, Reykjavik, Bergen, Umeå, Uppsala and Tartu.
[d] Data for type of painting was missing in Gothenburg.

Table 3
Onset and remission of respiratory symptoms, asthma, allergic rhinitis and rhinitis symptoms (%), stratified for indoor painting and ETS (n = 11,506).

		Total (%)	ETS at baseline (%)				Any indoor painting (%)			Wood painting (%)			Floor painting (%)		
			Never	Sometimes	Weekly	p[a]	Yes	No	p[a]	Yes	No	p[a]	Yes	No	p[a]
Onset	Wheeze	9.9	7.9	9.9	16.4	**<0.001**	10.5	9.5	0.134	10.3	9.9	0.664	12.0	9.8	0.195
	Nocturnal chest tightness	7.2	6.2	7.5	9.9	**<0.001**	7.3	7.2	0.761	8.2	7.1	0.204	9.9	7.2	**0.049**
	Nocturnal attacks of breathlessness	3.7	3.4	3.4	4.6	**0.027**	3.5	3.8	0.387	4.0	3.8	0.705	6.1	3.7	**0.017**
	Nocturnal cough	17.0	16.0	16.4	20.8	**<0.001**	17.1	17.1	0.982	19.0	16.7	0.077	20.8	16.8	0.098
	Productive cough	9.3	8.1	9.3	13.3	**<0.001**	9.1	9.5	0.493	9.0	9.4	0.715	13.7	9.1	**0.005**
	Current asthma [b]	4.1	3.9	3.6	5.1	**0.030**	4.1	4.1	0.973	3.4	4.3	0.126	3.6	4.2	0.543
	Ever doctor diagnosed asthma	4.3	4.2	4.0	4.9	0.299	4.5	4.2	0.458	4.3	4.7	0.560	5.3	4.6	0.519
	Allergic rhinitis	9.5	8.8	10.4	10.7	**0.026**	10.5	8.9	**0.016**	11.7	9.5	**0.022**	13.0	9.7	0.050
	Ever rhinitis symptoms	25.9	24.4	28.2	28.5	**0.004**	25.9	25.7	0.880	27.3	25.9	0.444	29.5	26.2	0.262
Remission	Wheeze	47.5	48.5	52.9	43.9	**0.022**	47.4	47.4	0.995	45.8	47.9	0.517	38.9	48.1	0.062
	Nocturnal chest tightness	66.9	67.9	63.1	67.5	0.482	65.5	68.2	0.338	62.7	66.9	0.298	64.6	66.3	0.782
	Nocturnal attacks of breathlessness	65.8	69.5	65.3	60.6	0.158	65.5	66.9	0.745	62.0	66.6	0.438	60.7	66.1	0.559
	Nocturnal cough	48.2	47.5	50.5	48.2	0.486	49.5	47.3	0.221	47.1	48.4	0.612	37.3	48.8	**0.005**
	Productive cough	54.8	55.7	58.9	51.0	0.072	56.2	54.2	0.424	59.0	55.2	0.259	52.1	56.0	0.524
	Current asthma [b]	38.9	36.9	40.7	43.2	0.368	40.4	37.6	0.472	31.5	39.7	0.111	36.4	38.4	0.817
	Allergic rhinitis	24.7	22.0	26.3	32.3	**<0.001**	28.5	22.5	**0.001**	27.5	24.3	0.204	34.9	24.3	**0.013**

[a] Chi-square test.
[b] Current asthma was defined as either asthma attack, asthma medication or both.

respectively). Extra analyses including only current smokers at baseline, adjusting for how much they smoked, showed that weekly exposure to ETS at baseline was related to onset of wheeze (OR 95%CI: 1.70, 1.29–2.42, p = 0.003).

Associations between ETS at baseline and remission of respiratory symptoms, asthma and rhinitis are shown in Supplement Table 2. There were no associations. Extra analyses for ETS at baseline or follow up and remission of symptoms showed that weekly exposure to ETS (at baseline or follow up) decreased remission of nocturnal breathlessness (OR 95% CI: 0.56, 0.31–0.998, p = 0.049). Wood painting was associated with less remission of current asthma. Floor painting was associated with less remission of wheeze. Any indoor painting and floor painting increased remission of allergic rhinitis. No associations were found between type of dwelling and remission of symptoms. Moreover, we found no associations between construction year and remission of respiratory symptoms, asthma or rhinitis.

Meta-analysis was used to detect heterogeneity between centres (Figs. 2-4). The estimates from the meta-analyses were almost identical to those derived when analysing the pooled data by multilevel logistic regression. There was no centre heterogeneity between ETS at baseline (never vs. sometimes or weekly) and onset of wheeze (Fig. 2). Moreover, there was no centre heterogeneity between floor painting and onset of productive cough (Fig. 3), or between wood painting and onset of allergic rhinitis (Fig. 4).

4. Discussions

Few studies have investigated respiratory effects in adults of other

Table 4
Adjusted odds ratios with 95% confidence intervals (CI) for onset of respiratory symptoms, current asthma, doctor diagnosed asthma and rhinitis in relation to smoking at baseline, change of smoking habit from baseline to follow up, ETS at baseline and combined ETS.

	Smoking at baseline[a,c]		Change of smoking from baseline to follow up[a,d]		ETS at baseline[b,e]		Combined ETS[b,f]	
	Ex-smoker	Current smoker	Start smoking	Quit smoking	Sometimes	Weekly	1	2
Wheeze	1.09(0.88,1.36)	**4.43 (3.61,5.45)** ***	**1.97(1.26,3.09)** **	**0.34 (0.26,0.45)** ***	1.02 (0.82,1.29)	**1.26 (1.02,1.57)***	0.92 (0.72,1.17)	**1.27 (1.03,1.58)** *
Nocturnal chest tightness	1.23(0.98,1.53)	**1.54 (1.20,1.96)** **	1.16(0.70,1.93)	0.88 (0.65,1.19)	1.06 (0.82,1.37)	1.22 (0.95,1.57)	1.03 (0.80,1.34)	1.26 (0.98,1.61)
Nocturnal breathlessness	1.17(0.86,1.60)	1.68 (1.23,2.31) **	0.86(0.39,1.92)	0.75 (0.51,1.13)	0.95 (0.66,1.37)	1.14 (0.82,1.60)	0.90 (0.62,1.32)	1.28 (0.92,1.78)
Nocturnal cough	0.94(0.79,1.11)	**1.69 (1.39,2.05)** ***	**1.54(1.04,2.30)** *	0.78 (0.61,0.99)	1.08 (0.88,1.32)	1.19 (0.97,1.46)	1.09 (0.88,1.34)	**1.25 (1.02,1.52)** *
Productive cough	0.95(0.77,1.18)	**2.52 (2.03,3.12)** ***	**1.67(1.05,2.67)** *	**0.60 (0.46,0.79)** ***	1.13 (0.89,1.43)	1.16 (0.92,1.47)	1.17 (0.92,1.49)	1.20 (0.95,1.51)
Current asthma	0.99(0.74,1.32)	**1.39 (1.02,1.89)** *	1.26(0.64,2.47)	1.11 (0.77,1.61)	0.80 (0.56,1.13)	0.93 (0.68,1.28)	0.84 (0.59,1.19)	1.03 (0.75,1.41)
Doctor diagnosed asthma	1.03(0.79,1.34)	1.14 (0.84,1.56)	0.63(0.27,1.47)	1.05 (0.72,1.53)	0.84 (0.61,1.17)	0.85 (0.62,1.16)	0.80 (0.58,1.12)	0.82 (0.60,1.13)
Allergic rhinitis	0.95(0.77,1.17)	1.18 (0.94,1.48)	0.91(0.52,1.58)	0.97 (0.73,1.29)	1.00 (0.79,1.26)	0.95 (0.75,1.21)	1.07 (0.84,1.35)	1.03 (0.81,1.31)
Rhinitis symptoms	**1.30 (1.10,1.53)** **	**1.35 (1.11,1.64)** **	1.34(0.91,1.97)	1.19 (0.93,1.51)	1.13 (0.93,1.36)	0.97 (0.80,1.19)	1.13 (0.94,1.37)	1.02 (0.84,1.24)

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$.
[a] Two level logistic regression models (centre, individual), including age (follow up), gender (baseline), smoking (baseline), change of smoking habit from baseline to follow up, BMI (baseline), change of BMI from baseline to follow up, education (follow up), any dampness (baseline) and change of dampness from baseline to follow up.
[b] Two level logistic regression models (centre, individual), adjusted for age (follow up), gender (baseline), smoking (baseline), change of smoking habit from baseline to follow up, BMI (baseline), change of BMI from baseline to follow up, education (follow up), any dampness (baseline) and change of dampness from baseline to follow up.
[c] Reference group: never smoker.
[d] Reference group: no change.
[e] Reference group: never.
[f] ETS at baseline or follow up: coded as 0, never; 1, sometimes but not weekly; 2, weekly. Reference group: 0, never.

Table 5
Adjusted odds ratios with 95% confidence intervals (CI) for onset of respiratory symptoms, current asthma, doctor diagnosed asthma and rhinitis in relation to indoor painting and construction year.

	Any indoor painting	Wood painting	Floor painting	Construction year[a]		
				1961–1975	1976–1985	1986–2001
Wheeze	1.13(0.96,1.33)	1.09(0.86,1.37)	0.89(0.58,1.37)	0.86(0.69,1.07)	0.79(0.63,0.99)	0.98(0.78,1.22)
Nocturnal chest tightness	1.03(0.86,1.22)	1.16(0.91,1.48)	1.26(0.85,1.87)	0.86(0.68,1.09)	0.84(0.65,1.08)	1.01(0.79,1.28)
Nocturnal breathlessness	0.88(0.69,1.12)	0.92(0.66,1.30)	1.20(0.73,1.97)	1.23(0.90,1.68)	0.81(0.56,1.17)	**1.39(1.01,1.92)** *
Nocturnal cough	0.99(0.86,1.14)	1.16(0.95,1.41)	1.01(0.70,1.44)	**0.81(0.67,0.97)** *	**0.78(0.64,0.94)** **	**0.74(0.61,0.90)** **
Productive cough	0.97(0.82,1.14)	0.98(0.77,1.25)	**1.64(1.15,2.34)** **	0.98(0.79,1.21)	0.82(0.65,1.03)	0.94(0.75,1.19)
Current asthma	0.98(0.78,1.23)	0.86(0.61,1.22)	0.74(0.39,1.42)	0.77(0.57,1.04)	0.81(0.59,1.10)	0.90(0.67,1.22)
Doctor diagnosed asthma	1.03(0.83,1.28)	0.99(0.73,1.35)	1.24(0.75,2.07)	0.74(0.56,0.99)	**0.67(0.49,0.91)** *	0.83(0.62,1.12)
Allergic rhinitis	1.11(0.94,1.30)	1.24(1.00,1.55)[b]	1.32(0.92,1.90)	0.99(0.80,1.22)	0.80(0.63,1.01)	0.95(0.76,1.19)
Rhinitis symptoms	0.94(0.82,1.08)	1.02(0.84,1.25)	1.01(0.71,1.42)	1.06(0.88,1.26)	0.96(0.80,1.16)	**1.34(1.12,1.61)** **

Two level logistic regression models (centre, individual), adjusted for age (follow up), gender (baseline), smoking (baseline), change of smoking habit from baseline to follow up, BMI (baseline), change of BMI from baseline to follow up, education (follow up), any dampness (baseline) and change of dampness from baseline to follow up.
***$p < 0.001$, **$p < 0.01$, *$p < 0.05$.
[a] Reference group: constructed before 1960.
[b] $p = 0.05$.

home environment factors than dampness and mould. Our study is one of the first prospective studies simultaneously investigating associations between smoking, environmental tobacco smoke, indoor painting, building construction year and onset and remission of adult respiratory symptoms, asthma and rhinitis. We found that risk factors for asthma and rhinitis may differ. Smoking was related to onset of respiratory symptoms and rhinitis symptoms. Starting smoking during follow up increased onset of respiratory symptoms and quitting smoking at follow up decreased the risk. ETS was associated with increased onset of wheeze. Wood painting decreased remission of current asthma. Floor painting increased onset of productive cough and decreased remission of wheeze. Indoor painting, especially floor painting increased remission of allergic rhinitis. Living in older buildings was a risk factor for onset of nocturnal cough and doctor diagnosed asthma. Living in new buildings was a risk factor for nocturnal dyspnoea and rhinitis.

Table 6
Adjusted odds ratios with 95% confidence intervals (CI) for onset of respiratory symptoms, current asthma, doctor diagnosed asthma and rhinitis in relation to ETS at baseline, stratified for smoking at baseline or follow up, and the interaction between ETS at baseline and smoking.

		Smoking at baseline or follow up[a]				
		Yes	p	No	p	p for Interaction[b]
Wheeze	Sometimes	1.18(0.80,1.74)	0.403	0.98(0.75,1.30)	0.913	**0.083**
	Weekly	**1.60(1.20,2.13)**	**0.001**	1.15(0.83,1.60)	0.406	
Nocturnal chest tightness	Sometimes	1.06(0.64,1.76)	0.813	1.10(0.82,1.47)	0.529	**0.021**
	Weekly	**1.74(1.20,2.52)**	**0.003**	0.84(0.58,1.24)	0.387	
Nocturnal breathlessness	Sometimes	1.34(0.73,2.46)	0.351	0.83(0.54,1.28)	0.400	0.332
	Weekly	1.47(0.92,2.36)	0.110	0.96(0.58,1.58)	0.869	
Nocturnal cough	Sometimes	0.95(0.66,1.36)	0.763	1.06(0.83,1.35)	0.641	0.436
	Weekly	1.12(0.85,1.48)	0.430	1.29(0.98,1.69)	0.069	
Productive cough	Sometimes	1.26(0.85,1.87)	0.251	1.04(0.77,1.40)	0.813	0.270
	Weekly	**1.37(1.01,1.86)**	**0.046**	1.09(0.77,1.54)	0.630	
Current asthma	Sometimes	1.06(0.61,1.85)	0.835	0.74(0.49,1.12)	0.159	0.390
	Weekly	1.04(0.68,1.60)	0.841	0.87(0.54,1.42)	0.585	
Doctor diagnosed asthma	Sometimes	0.88(0.48,1.64)	0.693	0.83(0.57,1.20)	0.326	0.277
	Weekly	1.08(0.69,1.69)	0.738	0.73(0.46,1.18)	0.203	
Allergic rhinitis	Sometimes	0.91(0.59,1.42)	0.687	1.14(0.87,1.49)	0.345	0.314
	Weekly	0.93(0.66,1.31)	0.671	1.09(0.78,1.53)	0.615	
Rhinitis symptoms	Sometimes	1.22(0.86,1.73)	0.265	1.16(0.93,1.44)	0.184	0.342
	Weekly	1.00(0.76,1.31)	0.979	1.02(0.77,1.35)	0.908	

Two level logistic regression models (centre, individual), adjusted for age (follow up), gender (baseline), BMI (baseline), change of BMI from baseline to follow up, education (follow up), any dampness (baseline) and change of dampness from baseline to follow up.

[a] Smoking at baseline or follow up: yes, current smokers at baseline or follow up; no, ex/never smokers at baseline and follow up.

[b] Interaction: Interaction between ETS baseline and smoking at baseline or follow up (yes/no) in association with onset of respiratory symptoms, current asthma, doctor diagnosed asthma and rhinitis. Two level logistic regression models (centre, individual), including age (follow up), gender (baseline), BMI (baseline), change of BMI from baseline to follow up, education (follow up), any dampness (baseline) and change of dampness from baseline to follow up in the models.

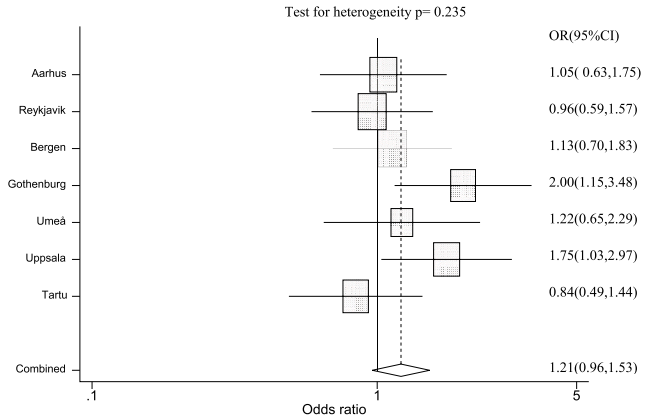

Fig. 2. Adjusted odds ratios and 95% CIs of wheeze in subjects reporting ETS at baseline with a combined odds ratio (diamond indicates 95% CI) from the model with centre as the random effect. The models were adjusted for age (follow up), gender (baseline), smoking (baseline), change of smoking habit from baseline to follow up, BMI (baseline), change of BMI from baseline to follow up, education (follow up), any dampness (baseline) and change of dampness from baseline to follow up.

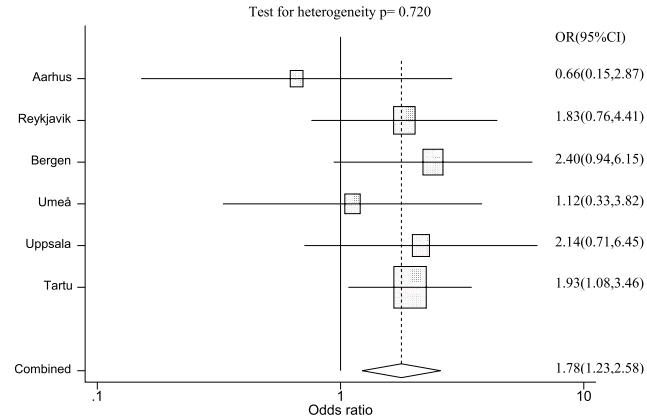

Fig. 3. Adjusted odds ratios and 95% CIs of productive cough in subjects reporting floor painting with a combined odds ratio (diamond indicates 95% CI) from the model with centre as the random effect. The models were adjusted for age (follow up), gender (baseline), smoking (baseline), change of smoking habit from baseline to follow up, BMI (baseline), change of BMI from baseline to follow up, education (follow up), any dampness (baseline) and change of dampness from baseline to follow up.

4.1. Methodological issues

Selection bias should not be a major problem in the initial postal questionnaire study (ECRHS I) since the participation rate was high (Johannessen et al., 2014). Moreover, the response rate from RHINE II to RHINE III was reasonable (71%), and it has been previously demonstrated that exposure-outcome associations were not influenced by loss to follow up (Johannessen et al., 2014). Nonparticipants in RHINE II had slightly higher prevalence of respiratory symptoms in ECRHS I as compared to participants ECRHS I, but the prevalence of allergic rhinitis was lower. Nonparticipants in RHINE III had slightly higher prevalence of respiratory symptoms and asthma in RHINE II as compared to participants in RHINE II, but the prevalence of dampness indicators at baseline (RHINE II) did not differ between participants and nonparticipants in RHINE III. Most exposure data, including ETS, construction year, indoor painting and type of dwelling were collected at baseline to reduce recall bias. Moreover, similar results were obtained in the crude and multivariate data analysis. Thus, our results are unlikely to be influenced by selection or information bias.

4.2. Different risk factors for asthma and rhinitis

Some studies have demonstrated that risk factors for asthma and rhinitis can differ. One study from China found that living close to heavy trafficked roads, redecoration, visible mould and window pane condensation were all risk factors for allergic rhinitis among adults, but only water damage was related to asthma (Wang et al., 2014b). Moreover, the Chinese study found different risk factors for childhood asthma

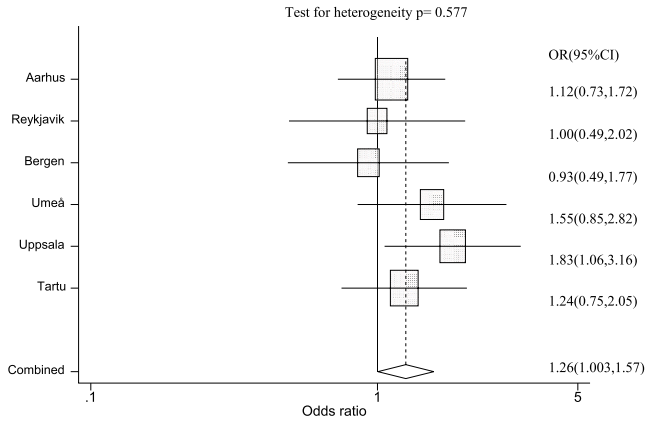

Fig. 4. Adjusted odds ratios and 95% CIs of allergic rhinitis in subjects reporting wood painting with a combined odds ratio (diamond indicates 95% CI) from the model with centre as the random effect. The models were adjusted for age (follow up), gender (baseline), smoking (baseline), change of smoking habit from baseline to follow up, BMI (baseline), change of BMI from baseline to follow up, education (follow up), any dampness (baseline) and change of dampness from baseline to follow up.

and rhinitis (Norback et al., 2018). One study from Sweden reported that ETS, indoor painting and odour were risk factors for adult asthma, while only window pane condensation was related to adult allergic rhinitis (Wang et al., 2014a). Another study from Sweden found that lower air exchange rate increased the risk of adult asthma while higher moisture load was a risk factor for rhinitis in adults (Wang et al., 2017b). The reason that risk factors can differ for asthma and rhinitis is unclear. Different particle size and different degree of water solubility can influence exposure in upper and lower airways. For instance, big particles as well as water soluble compounds are more likely to be deposited in the nose, but smaller particles and compounds with lower degree of water solubility are more likely to be deposited in the lower airways (Shusterman, 2003).

4.3. Smoking in relation to onset of respiratory symptoms and rhinitis symptoms

Our study found that smoking increased onset of respiratory symptoms and rhinitis symptoms. Moreover, we found that starting smoking increased onset of respiratory symptoms and quitting smoking decreased onset of respiratory symptoms. Our results on smoking are in agreement with previous studies. Adult smokers were more likely to develop asthma than those who had never smoked, showed in one review including only prospective studies (King et al., 2004). One cohort study from Italy including non-asthmatic adults with allergic rhinitis found that smoking increased the risk of incident asthma (Polosa et al., 2008). However, we found no previous prospective studies on smoking in relation to onset of rhinitis in adults.

4.4. ETS in relation to onset of respiratory symptoms

Despite smoking bans in public places in the Northern countries, 37.7% of the participants in our study were still exposed to ETS in the home at baseline. However, the prevalence of ETS at home was reduced to 13% at follow up. The reduction of indoor ETS occurred among non-smokers as well as among smokers. ETS at baseline was associated with onset of wheeze. This is in agreement with previous studies. One longitudinal study from Europe reported that ETS was related to onset of asthma and asthma symptoms (Flexeder et al., 2019). One longitudinal study from US found that ETS increased adult onset of asthma (Coogan et al., 2015). One population-based case-referent study from Sweden found that ETS was associated with onset of doctor diagnosed asthma (Thorn et al., 2001). Moreover, one population-based case-control study from Finland showed that ETS exposure at work was related to adult onset asthma. The study found a synergistic effect between asthma heredity (parental asthma) and exposure to ETS on adult onset asthma (Lajunen et al., 2013; Jaakkola et al., 2003).

We found that the respiratory effects of ETS was stronger among smokers than non-smokers. This could be because the smokers had a higher exposure to ETS, or because the combination of own smoking and ETS exposure had a stronger respiratory effect. More detailed studies on ETS exposure are needed in future studies.

4.5. Indoor painting in association with onset and remission of adult respiratory symptoms, asthma and rhinitis

Our study found that floor painting at baseline was associated with increased onset of productive cough and decreased remission of wheeze. Moreover, we found that wood painting was associated with less remission of current asthma. Wood painting can be a source of formaldehyde. Higher indoor formaldehyde concentrations were found in Swedish dwellings with wood painting (Wieslander et al., 1997a). There are few epidemiological studies on indoor painting and adult respiratory symptoms (Canova et al., 2013). One Swedish cross-sectional study found that domestic painting of wood details were related to adult asthma (Wieslander et al., 1997a). Elevated chemical emissions in new buildings from indoor painting can last up to six months (SverreBjørnHoløs et al., 2017). Being exposed to paints emissions may trigger later development of respiratory illnesses. Three cohort studies from Sweden (each with a 10-year follow up time) investigated effects of indoor painting on sick building syndrome (SBS) symptoms. The studies found that indoor painting increased SBS symptoms (Sahlberg et al., 2009) and incidence of general symptoms (Sahlberg et al., 2012). Moreover, indoor painting at baseline decreased remission of general symptoms (Sahlberg et al., 2010).

Any indoor painting and floor painting were related to increased remission of allergic rhinitis. One explanation could be that indoor painting and related renovation activities can benefit indoor environment by removing dirt, allergens and other indoor pollutants. One study on allergen levels in Germany day care centres showed that house dust mite allergens decreased significantly when rooms were renovated (Sander et al., 2016). Another intervention study from US found that cockroach and mouse allergen loads were reduced after house renovation (Jacobs et al., 2014).

One third of the homes in our study had been painted indoors in the last 12 months. Newly applied paints can emit volatile organic compounds (VOCs) and the emissions can last for a long time (Sparks et al., 1999). The indoor concentration of VOCs from indoor paints can differ depending on the type of paint used, the nature of the painted surfaces and the ventilation flow (Canova et al., 2013). There are two main types of indoor paints: water-based paints and solvent-based paints. Solvent-based paints produce rapid and high emissions of non-polar organic solvents. VOCs concentrations can be 100 times higher from solvent-based paints as compared to water-based paints (Wieslander et al., 1997b). Water-based paints are commonly used in Sweden nowadays (Sahlberg et al., 2009; Wieslander et al., 1994, 1997bbib_Wieslander_et_al_1997bbib_Wieslander_et_al_1994), but solvent paints can be used for specific applications such as painting on wood or metal surfaces. One Swedish study reported that working in a newly redecorated building painted with water-based paints increase nasal inflammation in office workers (Wieslander et al., 1999). Little is known about types of paints used in different European countries. It is mentioned in one recent review that European manufacturers are demanded by law to produce paint with low concentrations of VOC and in many countries low-VOC paints are mandatory (Canova et al., 2013).

4.6. Building age in association with adult onset of asthma and rhinitis

Living in older buildings (constructed before 1960) was associated with onset of nocturnal cough and doctor diagnosed asthma. Living in newer buildings, constructed in 1986–2001, was related to higher onset of nocturnal breathlessness. To our knowledge, our study is the first incident study investigating building age as a risk factor for adult respiratory health. One cross-sectional study from China reported that adults living in buildings constructed before 2005 had more asthma (as compared to buildings constructed after 2005) (Wang et al., 2019b). Moreover, one study from Sweden found that adults living in newer buildings (constructed 1961–1975) had higher prevalence of day time breathlessness (as compared to buildings constructed before 1960) (Wang et al., 2014a).

Living in newer buildings (constructed 1986–2001) was related to onset of rhinitis symptoms. This is in agreement with one previous study from Sweden, reporting that living in buildings constructed 1976–1985 was associated with more rhinitis among adults in Sweden (as compared to buildings constructed before 1960) (Wang et al., 2014a).

The reason for different associations between building age and upper and lower airway symptoms is not clear. Newer buildings can have more chemical emissions while older buildings can have more dirt containing bioaerosols. One study from Europe found that dampness and mould were more common in older buildings (Norback et al., 2017). Moreover, two recent studies, from China and Malaysia respectively, investigated microbiome richness in buildings and reported that increased building age was associated with richer bacteria/fungi composition variation in the buildings (Fu et al., 2020a, 2020b). In our study, we adjusted for visible dampness and mould in the analyses, but hidden dampness or mould can exist in older buildings. Moreover, older buildings could have more allergen contamination as compared to newer buildings, and dust and dirt can contain microbial compounds. One study from Germany found that concentrations of house dust mite allergens and cat allergen in dust were higher in older buildings (Fahlbusch et al., 1999). Nevertheless, new buildings can have higher chemical emissions. One study from Hong Kong, China found that newly built apartments had higher indoor formaldehyde concentrations (Guo et al., 2009). Formaldehyde is a water soluble compound mainly affecting the upper airways. Another study from Hong Kong concluded that building materials were the major source of indoor VOCs (Guo, 2011). One Korean study concluded that wood panels/vinyl floor coverings and paints were the main sources of indoor VOCs in newly built apartments (Shin and Jo, 2012). Our results imply that building age can be an indicator of certain indoor pollutants. More specific studies on building factors are needed to identify potential indoor hazards related to building age. It can be interesting to study change of microbial organisms in relation to building age in future studies, since indoor microbiome can influence asthma and rhinitis.

4.7. Strengths and limitations

There are several strengths in our study. Firstly, it is the first prospective study from Northern Europe investigating building characteristics other than dampness and mould in relation to onset and remission of adult respiratory symptoms, asthma and rhinitis. Secondly, the number of participants was large, giving high statistical power. Data on respiratory health associations for dampness in the home have been previously published from the study (Wang et al., 2019a). Dampness and mould is the most well documented home environment risk factor. Two reviews concluded that dampness and mould can increase risk of prevalent rhinitis and development of asthma (Quansah et al., 2012; Jaakkola et al., 2013). We have adjusted for dampness and mould in the models when studying health associations for other home environment factors.

There are some limitations. Our study included only questionnaire data, no objective exposure measurements were made. Moreover, we had no information on other indoor renovation activities than painting. Another limitation is related to symptom reporting. Respiratory symptoms were reported for the past 12 months at baseline and at follow up (with no information about respiratory symptoms several years before the follow up survey), which may not reflect the real onset during the study period. Moreover, we did not ask about pet keeping or family history of allergies or respiratory diseases, which could be potential confounders.

5. Conclusions

This prospective study found that smoking can be a risk factor for adult onset of respiratory symptoms and rhinitis symptoms. Starting smoking can increase onset of respiratory symptoms and quitting smoking can decrease the risk. ETS exposure can be a risk factor for onset of respiratory symptoms. Chemical emissions from wood or floor paints can increase onset and decrease remission of respiratory symptoms. However, indoor painting can increase remission of allergic rhinitis. Living in older buildings can increase adult onset of nocturnal cough and asthma. Living in newer buildings can increase onset of nocturnal breathlessness and allergic rhinitis. Our study indicates that smoking, especially smoking inside homes, should be avoided. Furthermore, health effects of building age and indoor painting needs to be investigated in more detailed studies.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envres.2020.110269.

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