BMJ Open

Occupational exposure and new-onset asthma in the population-based Telemark study: a 5-year follow-up

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To cite: Zivadinovic N, Jaoiun K, Klepaker G, *et al*. Occupational exposure and new-onset asthma in the population-based Telemark study: a 5-year follow-up. *BMJ Open* 2024;**14**:e090131. doi:10.1136/bmjopen-2024-090131

► Prepublication history for this paper is available online. To view these files, please visit the journal online (https://doi.org/10.1136/bmjopen-2024-090131).

Received 18 June 2024
Accepted 27 August 2024

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ABSTRACT

Objectives This study aimed to estimate the incidence of asthma and assess the association between job exposure matrix (N-JEM) assigned occupational exposure, self-reported occupational exposure to vapour, gas, dust and fumes (VGDF), mould, damages from moisture and cold, and new-onset asthma. We also aimed to assess the corresponding population attributable fraction (PAF) for ever exposure to VGDF.

Design Longitudinal population-based respiratory health study.

Setting Responders from the baseline Telemark Study in south-eastern Norway were followed up from 2013 to 2018.

Participants 7120 participants, aged 16–55, were followed during a 5-year period.

Main outcome measures New-onset asthma and its association with self-reported occupational exposure to VGDF, data from the N-JEM and self-reported workplace conditions were assessed using logistic regression adjusted for gender, age, smoking and body mass index. The PAF was calculated using the PUNAF command in STATA.

Results There were 266 (3.7%) cases of new-onset asthma and an incidence density of 7.5 cases per 1000 person-years. A statistically significant association was found for ever exposed to VGDF with an OR of 1.49 (95% CI 1.15 to 1.94), weekly OR 2.00 (95% CI 1.29 to 3.11) and daily OR 2.46 (95% CI 1.39 to 4.35) exposure to VGDF. The corresponding PAF for ever exposed to VGDF was 17% (95% CI 5.4% to 27.8%) and the risk of asthma onset increased with frequent VGDF exposure, indicating a possible exposure–response relationship (p=0.002 for trend). The N-JEM exposure group, accidental peak exposure to irritants had an increased risk of new-onset asthma, OR 2.43 (95% CI 1.21 to 4.90). A significant association was also found for self-reported exposure to visible damages due to moisture 1.51 (95% CI 1.08 to 2.11), visible and smell of mould 1.88 (95% CI 1.32 to 2.68), 1.55 (95% CI 1.12 to 2.16) and cold environment 1.41 (95% CI 1.07 to 1.86).

Conclusion Participants had elevated ORs for asthma associated with self-reported and N-JEM-assigned exposures. A PAF of 17% indicates that work-related asthma is still common. The possible exposure–response relationship suggests that reducing occupational VGDF exposure frequency could prevent the onset of asthma.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ This study is a general population-based study with a prospective study design.
⇒ The large sample size is a strength of this study.
⇒ The study uses self-reported occupations combined with job-exposure matrix-assigned exposure to reduce recall bias.
⇒ Even though the analyses of the impact of loss to follow-up have been assessed in a previous study, the limitation that 49% of the participants were lost to follow-up must be considered when interpreting the results.
⇒ Self-report of occupational exposure may also be a limitation of this study.

INTRODUCTION

Asthma affects approximately 1%–18% of the population worldwide.[1] Several risk factors have been identified for asthma development in adulthood.[2 3] Workplace exposure is a well-recognised risk factor. This type of asthma is known as work-related asthma (WRA) and includes both new-onset occupational asthma (OA) and work-exacerbated asthma (WEA).[4] Cross-sectional studies using measures of prevalence have shown that up to 15% of adult-onset asthma is related to occupational exposure.[5] WEA is a more common condition, accounting for 25% of employed patients with asthma.[5] Asthma in these patients is pre-existing asthma that is exacerbated by exposure at work.

Exposure to sensitisers or irritating agents can lead to the onset of sensitiser-induced OA (SI-OA) or irritant-induced non-sensitising OA, respectively.[6] More than 400 sensitising agents with high and low molecular weights (LMWs) have been identified as potential causes of OA, and new sensitisers are identified every year.[7–9] Pooling data from longitudinal studies over the past 20 years have shown that the occupational contribution to the burden of asthma incidence is approximately

16%,[10] which is similar to the results from cross-sectional studies.

Studies over the last decade have shown a decrease in the incidence of new-onset asthma.[11 12] Interestingly, recent studies indicated that this trend may change.[13]

In Norway, there are no overall national estimates of asthma prevalence; however, according to the national prescription register, there has been a growing trend in the use of medication for obstructive lung diseases in the last 5 years.[14] Studies from other parts of Norway (eg, Hordaland and Northern Trøndelag) have estimated the occurrence of respiratory diseases and have drawn attention to some risk factors such as anxiety, depression and working as a farmer or forester.[15 16] These studies used crude measures of self-reported exposure, however, do not combine self-reported occupation with a job-exposure matrix (JEM). Thus, it is necessary to investigate these issues in a larger prospective study with more objective data on occupational exposure.

This study aimed to estimate asthma incidence, assess the association between JEM-assigned occupational exposure, self-reported occupational exposure to vapour, gas, dust, and fumes (VGDF), mould, damage due to moisture, and cold, and new-onset asthma and to determine the corresponding population-attributable fraction (PAF).

METHODS

Study population

Telemark is a county with both urban and rural parts and contains one of the leading industrial centres in Norway. In 2013, a sample of 50 000 people, randomly chosen, were invited to participate in a large population study on respiratory health. The first data collection included 16 099 participants and found a prevalence of physician-diagnosed asthma of 11.5%.[17]

In 2018, a 5-year follow-up of the Telemark Study baseline cohort was conducted. Among the 16 099 baseline participants, 7958 (51%) answered the follow-up questionnaire while 7723 (49%) did not. The characteristics of these two groups and the analyses of the possible risk factors associated with loss of follow-up have been described in a previous study.[18] Out of 7958 participants who answered the questionnaire in 2018, 6 did not complete the questionnaire, and 832 reported physician-diagnosed asthma at baseline in 2013. Therefore, these patients were excluded from the present study. This left us with a study population of 7120 participants who were at risk for asthma onset after 2013 (figure 1).

Study design

At the 5-year follow-up in 2018, the participants were asked to complete a postal questionnaire and return it in a prepaid envelope. Participants were also given the option to complete the questionnaire by logging onto a secure internet webpage. The names, addresses and ID numbers of all the participants were obtained from the National Population Register. We sent two reminders with the purpose to increase response rates. In addition, we performed a telephone survey in 2023, to assess new asthma cases in the period from 2013 to 2018. We asked those who reported new-onset asthma the following question: 'In what year was your asthma diagnosed by a physician?' 196 (74%) participants agreed to answer the question and 70 (26%) did not consent to the telephone survey or were not possible to trace.

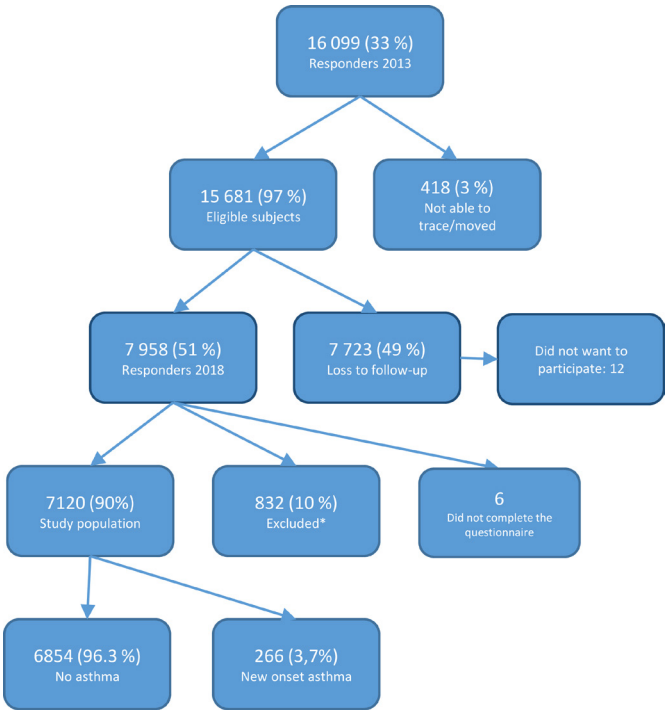

Figure 1 Flow chart of the Telemark Study showing new-onset asthma at follow-up in 2018. *Reported asthma in 2013.

Questionnaire

The questionnaire used in both the 2013 baseline and 2018 follow-up surveys was based on the European Community Respiratory Health Survey questionnaire. This questionnaire was designed to assess respiratory symptoms and diseases among adults and to compare the occurrence in different European countries. The questionnaire included questions regarding obstructive lung disease, respiratory symptoms, respiratory symptoms at work, occupational history, exposures and comorbidities.[19]

Outcomes and occupational exposures

Physician-diagnosed asthma was defined as a positive response to the question, 'Has a physician ever diagnosed you with asthma?' The questionnaire asked the participants to list their occupational history: 'Describe your employment and work tasks with their associated time frames'. Self-reported occupations at the follow-up in 2018 were classified by trained personnel according to the 1988 International Standard Classification of Occupations (ISCO-88). The ISCO codes were then connected to an asthma-specific JEM developed for five Northern

European countries (Sweden, Iceland, Estonia, Denmark and Norway) (N-JEM). The N-JEM consists of six main exposure groups: exposed to high-molecular-weight (HMW) agents such as animal-derived antigens, plant-associated antigens, latex protein and pharmaceutical product antigens; exposed to LMW agents such as reactive chemicals, acrylates, epoxy chemicals and diisocyanates; exposed to irritating agents such as cleaning agents, wood and paper dust, inorganic dust and fumes, textile dusts, metal working fluids, vehicle/motor exhaust, and environmental tobacco smoke; exposed to accidental peak exposure to irritants; uncertain or low exposed to respiratory allergens and irritants mentioned above and non-exposed reference group. More details on the most common occupations associated with exposures are provided in two studies by Lillienberg *et al*.[20 21] Participants were also asked questions concerning specific occupational exposures, such as exposure to VGDF and exposure to visible mould, mould odour, water damage or dampness and cold. Exposure to VGDF was also categorised into four frequency levels (daily large parts of working days, daily but for a short period, weekly and less often). In cases where data on ever being exposed to VGDF were missing, we designated them as 'yes' if the participants responded to the question regarding the frequency of VGDF exposure. There were 121 (1.7%) participants with missing data for both questions, and these were excluded from the analysis. For cases where data on other occupational exposures were missing, the missing values were assigned to 'no' for that specific occupational risk factor.

Covariates

Self-reported weight and height values at baseline and follow-up were used to calculate the body mass index (BMI). There were 1276 (17.8%) missing BMI values at baseline and another 161 (2.3%) with BMI missing at follow-up. To address missing BMI data at baseline, we started with the BMI at follow-up and subtracted the average change observed in the study population's BMI from baseline to follow-up. For missing BMI data at follow-up, we adjusted the baseline BMI by adding the average change observed in the study population's BMI over the same period. Age was stratified into three groups: 16–30 years, 31–40 years and 41–50 years. Smoking at baseline was categorised based on whether the participants were never, past, occasional or daily smokers. The missing data for smoking (41 participants, 0.6%) were found to be completely random. For the regression models, all cases with missing data on any variable were excluded from the analyses.

Statistical analyses

BMI was summarised using the median and IQR because it was not normally distributed, as indicated by the Shapiro-Wilk test. Binary logistic regression models were used, to analyse the association between occupational exposure and the development of new-onset asthma, with the entire study population. Models were adjusted for age (in three categories), gender and smoking as categorical variables and BMI as a continuous variable at baseline. In addition, logistic regression analyses adding the covariates nasal allergy, family history of asthma, domestic exposure to moulds and non-occupational exposure to exercise together with the original covariates mentioned above were performed in a separate regression model. The results of regression analyses were calculated and presented as ORs with 95% CIs to investigate the risk factor of new-onset asthma. Based on the answers to the telephone survey, Cox regression analyses were performed to analyse asthma as a time-to-event variable. Furthermore, we tested the trend of the frequency of exposure to VGDF after fitting the logistic model adjusted for all covariates using postestimation contrast commands in STATA. PAF was estimated using Miettinen's formula[22]:

$$PAF = \frac{p_c(RR_{adj}-1)}{RR_{adj}}$$

Where p_c means proportion of population exposed to risk factors and RR is the relative risk of incidence of the case of the exposed over the non-exposed. In the study, the adjusted OR (OR_{adj}) was used in place of RR_{adj}. Moreover, PUNAF command in STATA was also used to calculate PAF and its lower and upper confidence limits after fitting the logistic model adjusted for all covariates. All statistical tests were performed by using STATA V.17.0 (StataCorp). Statistical significance was defined as $p<0.05$.

Patient and public involvement

Written information regarding participation at baseline in 2013 was sent to all participants together with the questionnaire. All participants at baseline returned a signed consent that they want to participate in the study. At follow-up in 2018, the participants were informed that returning the completed questionnaire would be considered giving consent (according to approval from the REC). The participants were also informed that they had the right to withdraw from the study at any time, without providing a reason. User representatives were involved in the planning of the study and designing of the questionnaire. A representative from the Norwegian Asthma and Allergy Association who is a member of the steering committee contributed to the questionnaire development.

RESULTS

In the 5 year follow-up of the Telemark Study cohort, there were 266 new cases of physician-diagnosed asthma, for a 5-year cumulative incidence of 3.7% and an incidence density of 7.5 cases per 1000 person-years. The characteristics of the study population are summarised in table 1.

In the analyses of exposure to self-reported VGDF and new-onset asthma, the regression models adjusted for four potential confounders showed several significantly elevated ORs (table 2). For participants ever exposed to VGDF, the OR was 1.49 (95% CI 1.15 to 1.94) with a related PAF of 17% (95% CI 5.4% to 27.8%). The results for frequency of VGDF exposure in the past 12 months were as follows: an OR of 2.46 (95% CI

Table 1 The follow-up study population and their characteristics reported at baseline

	Study population 7120 (100%)	No asthma 6854 (96.3%)	New-onset asthma 266 (3.7%)
Gender			
Female	4128 (58.0%)	3958 (57.8%)	170 (63.9%)
Male	2992 (42.0%)	2896 (42.2%)	96 (36.1%)
Area of residence			
Urban	4641 (65.2%)	4473 (65.2%)	168 (63.2%)
Rural	2479 (34.8%)	2381 (34.8%)	98 (36.8%)
Age category			
16–30	1663 (23.4%)	1593 (23.2%)	70 (26.3%)
31–40	1834 (25.8%)	1766 (25.8%)	68 (25.6%)
41–50	3623 (50.8%)	3495 (51.0%)	128 (48.1%)
Education			
Elementary+1–2	853 (12.0%)	813 (11.9%)	40 (15.0%)
Upper secondary and certificant	2552 (35.8%)	2456 (35.8%)	96 (36.1%)
University/college	3466 (48.7%)	3346 (48.8%)	120 (45.1%)
Other/missing	249 (3.5%)	239 (3.5%)	10 (3.8%)
Smoking habits			
Never	4021 (56.5%)	3885 (57.0%)	136 (51.1%)
Past	1590 (22.3%)	1524 (22.3%)	66 (24.8%)
Occasional	611 (8.6%)	583 (8.5%)	28 (10.5%)
Daily	857 (12.0%)	827 (12.1%)	30 (11.3%)
BMI, median (IQR), n=7089			
	24.9 (22.5–27.8)	24.8 (22.5–27.8)	25.6 (23.2–28.8)

BMI, body mass index.

1.39 to 4.35) for exposure daily large parts of the working day and an OR of 2.00 (95% CI 1.29 to 3.11) for weekly exposure. In addition, there was a borderline statistically significant OR of 1.62 (95% CI 0.98 to 2.70, p=0.059) for those exposed to VGDF daily for a short period. The risk of asthma onset increased with frequent VGDF exposure (p=0.002 for trend).

The analyses of the association between occupational exposure assigned by the N-JEM (2018) and new-onset asthma showed a statistically significant association with accidental peak exposure to irritants with an OR of 2.43 (95% CI 1.21 to 4.90) when adjusting for four potential confounders (table 3). A reduced odd of new-onset asthma was observed in workers with uncertain or low exposure, with an OR of 0.39 (95% CI 0.18 to 0.82). The ORs were not statistically significant for HMW agents, LMW agents and irritants.

Table 2 Self-reported exposure to VGDF at follow-up in 2018 and new-onset asthma

	Asthma cases N=266	OR crude (95% CI)*	OR adj. (95% CI)†
Ever exposed to VGDF	132 (50.0%)	**1.32 (1.03 to 1.69)**	**1.49 (1.15 to 1.94)**
Frequency of exposure to VGDF in the past 12 months			
Daily, large parts of the working day	15 (6.3%)	**1.97 (1.13 to 3.42)**	**2.46 (1.39 to 4.35)**
Daily, but for a short period	19 (7.9%)	1.44 (0.88 to 2.37)	1.62 (0.98 to 2.70)
Weekly	29 (12.1%)	**1.72 (1.13 to 2.60)**	**2.00 (1.29 to 3.11)**
Less often	53 (22.2%)	1.07 (0.76 to 1.49)	1.24 (0.88 to 1.74)

Bold typeface represents p<0.05.
*Reference categories in each separate model were not exposed to VGDF.
†Adjusted for age, gender, smoking status and BMI.
BMI, body mass index; VGDF, vapour, gas, dust and fumes.

Table 3 Occupational exposures assigned by the N-JEM and new-onset asthma

N-JEM	Asthma cases N=266	OR crude (95% CI)*	OR adj. (95% CI)†
High-molecular-weight (HMW) agents	36 (13.5%)	1.15 (0.80 to 1.65)	1.04 (0.71 to 1.51)
Low-molecular-weight (LMW) agents	16 (6.0%)	1.23 (0.73 to 2.07)	1.40 (0.82 to 2.36)
Irritants	32 (12.0%)	0.94 (0.64 to 1.37)	0.98 (0.66 to 1.45)
Accidental peak exposure to irritants	9 (3.4%)	**2.08 (1.04 to 4.16)**	**2.43 (1.21 to 4.90)**
Uncertain or low exposure to HMW, LMW and irritants	8 (3.0%)	**0.41 (0.20 to 0.84)**	**0.39 (0.18 to 0.82)**

Bold typeface represents p<0.05.
*Reference category in each separate model was the unexposed group based on the N-JEM.
†Adjusted for age, gender, smoking status and BMI.
BMI, body mass index; N-JEM, job exposure matrix.

Table 4 shows the results of the multivariable analyses on self-reported exposure to dampness, mould and cold in the workplace and new-onset asthma. The analyses that adjusted for all four potential confounders showed significant associations for each of the exposures: visible damages due to moisture (OR 1.51, 95% CI 1.08 to 2.11), visible mould (OR 1.88, 95% CI 1.32 to 2.68), smell of mould (OR 1.55, 95% CI 1.12 to 2.16) and cold (OR 1.41, 95% CI 1.07 to 1.86).

The results of the multivariable analyses using covariates nasal allergy, family history of asthma, domestic exposure to moulds and non-occupational exposure to exercise together with the original covariates age, gender, smoking status and BMI were as follows: ever exposed to VGDF OR 1.46 (95% CI 1.12 to 1.91), exposed to VGDF daily, large parts of working day: 2.26 (95% CI 1.27 to 4.04), weekly exposed to VGDF: 1.83 (95% CI 1.17 to 2.86), accidental peak exposure to irritants: 2.29 (95% CI 1.12 to 4.67), exposure to visible damages due to moisture: 1.37 (95% CI 0.98 to 1.92), exposure to visible mould: 1.75 (95% CI 1.22 to 2.52), exposure to smell of mould: 1.45 (95% CI 1.04 to 2.02) and cold: 1.36 (95% CI 1.02 to 1.80).

DISCUSSION

From our follow-up study of new-onset asthma, the 5-year cumulative incidence was 3.7%, the incidence density was 7.5 cases per 1000 person-years and the PAF was 17%. The results showed increased ORs for asthma associated with ever exposed to VGDF, exposure that occurred weekly and daily in large parts of the working day. Our results showed that self-reported exposure to visible mould and mould odours, damages due to moisture and cold environments were also associated with the onset of asthma.

The calculated PAF in our study is consistent with a recent study summary estimate of 16%.[10] A self-reported single VGDF variable can be useful for assessing occupational exposure.[23] It has been used in other population-based studies to investigate the association between VGDF exposure and the risk of asthma.[24 25] These studies have shown an increased risk of asthma associated with exposure to VGDF. This finding is consistent with the results of the present study. In addition, we asked the participants about the frequency of VGDF exposure. To the best of our knowledge, the frequency of such exposures, in relation to asthma, has not been extensively studied previously. The test for trends in the frequency of exposure to VGDF showed a positive correlation with more frequent exposure, indicating a possible exposure–response relationship.

Using JEMs to study the association between exposure and disease development provides the advantage of more objective estimates than self-reports and reduces the risk of recall bias.[20] Our study did not find any association between exposure to HMW and LMW agents assigned by the N-JEM and new-onset asthma. These two groups include more than 400 known causative agents that can

Table 4 Self-reported exposure to dampness and mould in 2018 and new-onset asthma

Have you worked on premises with	Asthma cases N=266	OR crude (95% CI)*	OR adj. (95% CI)†
Visible damages due to moisture	45 (16.9%)	**1.44 (1.04 to 2.00)**	**1.51 (1.08 to 2.11)**
Visible mould	40 (15.0%)	**1.76 (1.24 to 2.48)**	**1.88 (1.32 to 2.68)**
Smell of mould	47 (17.7%)	**1.47 (1.06 to 2.03)**	**1.55 (1.12 to 2.16)**
Cold (cooling room/outdoors at winter)	82 (30.8%)	**1.38 (1.06 to 1.80)**	**1.41 (1.07 to 1.86)**

Bold typeface represents p<0.05.
*Reference category in each separate model was the unexposed group.
†Adjusted for age, gender, smoking status and BMI.
BMI, body mass index.

induce asthma via an immune response after exposure. Identifying causative agents is an important step towards the application of preventive measures. Common sensitisers such as isocyanates or latex were recognised as risk factors for OA 20–30 years ago. This evidence has led to the application of surveillance systems and preventive measures, which may have contributed to the reduction of the incidence of OA caused by sensitiser exposure.[26] Irritants can cause airway inflammation and induce asthma development. Unfortunately, studies on irritant exposure and OA are difficult to perform because of the uncertain timing of symptom onset relative to exposure, exposure complexity and different levels of exposure.[27] Most of these studies included case reports, case series and population-based cohort studies using specific JEMs.[28] The results of our longitudinal study showed an increased risk of new-onset asthma owing to accidental peak exposure to irritants. This is in line with the findings of a previous large population-based study follow-up that reported an OR of 2.4 (95% CI 1.3 to 4.7).[20] The groups exposed to accidental peak exposure in that study included occupations such as welders and flame cutters, sheet metal workers, ore and metal furnace operators, police officers, and firefighters. The nine new asthma cases with this exposure involved five police officers, two participants employed in fertiliser production plants, one firefighter and one welder. Awareness of irritant exposure and asthma development is still important, as these patients have more exacerbations, use more medications and may have a poorer prognosis than patients with SI-OA.[29]

Even though N-JEM includes exposure to various groups of allergens and irritants, there are no groups that include exposure to mould, dampness and cold. The results of our study showed an increased risk of asthma development in workers exposed to visible mould and damage due to moisture. In addition, we found that exposure to mould odours in the workplace could be a risk factor for new-onset asthma.

Various outdoor and indoor conditions can contribute to exposure to dampness and mould. Floods, damage to outdoor construction, leakage from pipe systems and production processes, and inadequate ventilation are sources of moisture and damage to buildings. Damage to both organic and inorganic materials (wood and medium-density fiberboard) may result in conditions that are susceptible to mould growth. Although there are few estimates of the prevalence of exposure to damp and mould in the workplace in Norway, a 2016 report from the National Health Institute summarised that the prevalence of moisture problems in Norwegian buildings is between 10% and 20%.[30] Results from a large population-based follow-up study (RHINE II) showed a prevalence of 19.4% for exposure to dampness or mould in workplace buildings in seven centres in the five Nordic countries.[31]

Over the past two decades, there has been growing evidence that exposure to indoor dampness and mould is associated with respiratory health outcomes. A review of scientific evidence from 2004 and the WHO guidelines from 2009 concluded that there is sufficient evidence of an association between exposure to indoor dampness and asthma development.[32 33] However, evidence for mould exposure was insufficient to confirm such an association. Caillaud *et al* published in 2018 a review of papers published from 2006 to 2017, focusing on the effects of indoor mould exposure on asthma and rhinitis.[34] The review concluded that there is sufficient evidence of an association between exposure to mould at work and new-onset asthma as well as exacerbation of asthma. The RHINE II longitudinal study found that exposure to water damage, floor dampness and visible mould at work could be risk factors for the onset of asthma.[31] The results of this prospective study are consistent with ours.

Our study found that self-reported exposure to cold air in the workplace (indoors or outdoors) may be a risk factor for new-onset asthma. Previous studies have shown that outdoor exposure to low temperatures can lead to the exacerbation of asthma and more hospitalisations due to the worsening of pre-existing asthma in adults.[35 36] Some studies have also proposed possible pathophysiological mechanisms that cause asthma exacerbation under cold conditions.[37] However, evidence of the association between exposure to cold air and new-onset asthma remains limited. A case-crossover study from Finland concluded that a cold winter season could increase the risk of asthma development within 1–2 years.[38] We found no other studies that used a prospective design to investigate exposure to cold air at work or asthma development.

Strengths and limitations

One strength of this study was that it was a population-based survey with a prospective design. The large sample size included in this study provided more statistical power and better quality of our results. However, 49% of participants from baseline were lost to follow-up. Possible reasons for non-response could be the comprehensive questionnaire (10 pages with 68 questions), and the strict requirements from REC, regarding how many times, and in what way the participants could be contacted. Loss to follow-up may be a limitation of our study and challenge our risk estimates as non-response at follow-up can cause selection bias and affect estimates of exposure and outcome. Our loss to follow-up study showed that male gender, young age, low education level and current smoking may be risk factors for non-response at follow-up.[18] These results are in accordance with other similar studies assessing non-participation.[39 40] We also found that physician-diagnosed asthma, exposure to VGDF, LMW and irritants may be a risk factor for not participating at follow-up. A higher non-response of participants with physician-diagnosed asthma and those exposed to VGDF, LMW and irritants may have contributed to an underestimation of our results in this study. Nevertheless, we performed an analysis, in which we compared the baseline group sample, the loss to follow-up group and the responders in 2018 regarding

occupational exposure to LMW and its relationship with wheezing. The results showed no statistically significant differences between groups.[18]

There are numerous well-documented risk factors that may cause or affect asthma. In our questionnaire, we obtained answers to questions regarding family history of asthma, nasal allergies, domestic exposure to moulds and non-occupational exposure to exercise. The sensitivity analyses that included these variables did not substantially change the estimates except for visible damages due to moisture which was then statistically non-significant with an OR of 1.37 (95% CI 0.98 to 1.92).

Although Telemark County has a heterogeneous population, it has lower work participation and level of education than the country average. Therefore, the results may not be entirely representative of the general population of Norway. The use of self-reported questionnaires may lead to recall bias, which may be another limitation. The question on self-reported physician diagnosis of asthma has shown high specificity for confirmation of asthma.[41] However, there are limitations as subjects with less symptoms and milder asthma seem to be more likely not to report their diagnosis.[42] Some participants could have not reported asthma diagnosed by a physician at baseline in 2013 but then reported asthma at follow-up due to more symptoms in 2018. This likely represents a small number of cases. We modelled asthma rather than OA which is an accepted approach to investigate occupational causes of asthma in a population-based study. Further, it is known that self-reported exposure can be affected by recall bias. Nonetheless, observational methods for assessing dampness and mould may be effective in assessing exposure in the workplace. An observational tool, called the Dampness and Mould Assessment Tool, was developed by the National Institute for Occupational Safety and Health and is used to assess indoor exposure to dampness and mould with self-reported perceptions of visible water damage or dampness, visible mould and mould odour. Exposure assessed by the DMAT has been shown to be associated with objective measures based on environmental samples.[43] The Telemark study used self-reported occupation which was coded by the ISCO-88 system and then combined with an asthma-specific JEM. The use of a JEM was intended to reduce recall bias. For VGDF exposure, we used self-reported exposure at follow-up in 2018. This could also be a limitation of our study, as there could be participants who were diagnosed with asthma in the 5-year period, and therefore, changed jobs. One reason for this is that we did not have the exact time of asthma onset for our participants in the original questionnaire. Nevertheless, only 7 of the 266 new asthma cases reported work changes because of respiratory symptoms. In our telephone survey of the new asthma cases reported in 2018, we asked about the year in which they were diagnosed with asthma by a physician. The resulting Cox regression analyses using this variable yielded minor differences in outcomes for the risk factors (data are not shown).

In conclusion, this 5-year prospective study of new-onset asthma found a 5-year cumulative incidence of 3.7% and an incidence density of 7.5 cases per 1000 person-years. Asthma onset was associated with self-reported and JEM-assigned occupational exposures. The results showed an elevated OR for participants ever exposed to VGDF and increasing OR values with more frequent VGDF exposure. A PAF of 17% for ever VGDF exposure indicates that WRA is still common, and the possible exposure–response relationship suggests that reducing occupational VGDF exposure frequency could prevent the onset of asthma cases.

We found that occupational exposure to accidental peaks of irritants, mould, dampness and cold may be risk factors for asthma development. These findings underline that awareness of occupational risk factors in addition to protective measures is important for the prevention of new-onset asthma.

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Acknowledgements The authors thank our helping statistician Martin Veel Svendsen and medical technician Gølin Gundersen for helping with data collection. We would also like to thank Editage (www.editage.com) for their English language editing services.

Contributors NZ was involved in writing the manuscript, the study design, data collection, data analysis, data management and data interpretation. He is the guarantor for this study. KJ was involved in statistical analyses, data management and the writing of the manuscript. GK participated in the study design, planning and formulating the study questionnaire, data collection, data management, data interpretation and critical revision of the manuscript. AW participated in the data interpretation and critical revision of the manuscript. KT and PKH were involved in the data analysis, data interpretation, analytical strategies and critical revision of the manuscript. JK was involved in the study design, data interpretation, analytical strategies and critical revision of the manuscript. RA and AKMF were involved in the study design, planning and formulating the study questionnaire, data collection, data analysis, data management, data interpretation and critical revision of the manuscript. The guarantor of the study (NZ) accepted full responsibility for the finished work and/or the conduct of the study, had access to the data and controlled the decision to publish.

Funding This study was supported by the Department of Research at Telemark Hospital (grant number 5690.20).

Disclaimer The findings and conclusions of this report are those of the authors and do not necessarily represent the official position of the National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention.

Competing interests None declared.

Patient and public involvement Patients and/or the public were involved in the design, or conduct, or reporting, or dissemination plans of this research. Refer to the Methods section for further details.

Patient consent for publication Not applicable.

Ethics approval The Regional Committee for Medical and Health Research Ethics (REC) restricted further contact with the participants. Ethics approval was obtained from the REC in 2012 (2012/1665/REK Sør-Øst D).

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available on reasonable request. Data are available on reasonable request to the Telemark Study Steering Board leader, after approval from the Regional Committee for Medical and Health Research Ethics in Norway (REC).

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