

Occupational exposure and the risk of new-onset chronic rhinosinusitis – a prospective study 2013-2018*

Ulrika K. E. Clarhed¹, Helena Johansson², Martin Veel Svendsen³, Kjell Torén⁴,
Anne Kristin Møller Fell³, Johan Hellgren¹

Rhinology 58: 0, 000 - 000, 2020

<https://doi.org/10.4193/Rhin20.104>

¹ Department of Otorhinolaryngology, Head & Neck Surgery, Institute of Clinical Sciences, The Sahlgrenska Academy at the University of Gothenburg, Gothenburg, Sweden

² Mary McKillop Institute for Health Research, Australian Catholic University, Melbourne, Australia

³ Department of Occupational and Environmental Medicine, Telemark Hospital, Skien, Norway

⁴ Occupational and Environmental Medicine, Institute of Medicine, The Sahlgrenska Academy at the University of Gothenburg, Gothenburg, Sweden

***Received for publication:**

March 23, 2020

Accepted: May 21, 2020

Abstract

Background: The aetiology behind chronic rhinosinusitis (CRS) is still poorly understood. The aim of this study was to investigate the association between the onset of CRS and several common occupational exposures over time.

Methodology: An adult random population from Telemark, Norway, comprising 7,952 subjects, who answered a comprehensive respiratory questionnaire including questions on CRS and occupational exposure first in 2013 and again in 2018.

Results: New-onset CRS during the five-year follow-up was independently associated with occupational exposure to hair-care products, cleaning agents among women, super glue, strong acids, cooking fumes and wood dust.

Conclusion: In this random population cohort from Norway, exposure to several common occupational agents, such as hair-care products, super glue and wood dust, was associated with the onset of CRS. It is important that physicians who see patients with CRS inquire about workplace exposure.

Key words: epidemiologic measurements, paranasal sinus diseases, respiratory system, rhinitis, sinusitis

Introduction

Chronic rhinosinusitis (CRS) is an inflammation of the nasal mucosa in the nose and the paranasal sinuses⁽¹⁾, affecting around one in 10 of the European population⁽²⁾. CRS substantially affects the health-related quality of life in these patients, with elevated rates of depression, anxiety, sleep disturbances, sexual dysfunction and fatigue⁽³⁾, as well as impaired sleep quality^(4,5).

In epidemiological studies, chronic rhinosinusitis in adults is defined as the presence of two or more symptoms, one of which should be either nasal blockage/obstruction/congestion or nasal discharge (anterior/posterior nasal drip) for 12 weeks or more⁽¹⁾. CRS includes both allergic and non-allergic phenotypes, but the aetiology behind CRS remains unknown in many pa-

tients⁽¹⁾. Occupational exposure has been associated with CRS⁽⁶⁾, but it has not been thoroughly investigated. In a few studies predating the use of the CRS concept, occupational exposure was linked to rhinitis^(7,8). Occupational rhinitis has been studied with regard to exposure and possible mechanisms. Both high molecular weight agents and low molecular weight agents have been associated with occupational rhinitis and may cause mucosal inflammation either through allergic mechanisms or as irritants⁽⁹⁾. CRS comprises a group of patients defined by specific combinations of rhinosinusitis symptoms lasting for at least 12 weeks. Due to the chronic and often therapy-resistant nature of the disease, it is important to study the role of occupational exposure in relation to CRS, as this has previously only been done for rhinitis.

We have previously reported cross-sectional data from a random population of 16,099 from the County of Telemark in Norway from 2013, in which participants answered a questionnaire on respiratory symptoms and occupational exposure. The prevalence of CRS was 9% and occupational exposure to paper dust and cleaning agents was independently associated with an increased risk of having CRS⁽¹⁰⁾. Since the baseline inclusion in 2013, the Telemark cohort has been followed up after five years. Our aim with this prospective study is to investigate the association between new-onset CRS and several common occupational exposures over time. To our knowledge, this is the first large, prospective, population-based study of CRS and occupational exposure over time.

Materials and methods

Study population and design

This is a prospective study of a random population from the County of Telemark, Norway, called the “Telemark Study”. The study has previously been described in detail^(10, 11). The County of Telemark has a population of about 170,000 inhabitants. A random sample of 50,000 subjects of working age, 16-50, was drawn in 2013 from the population registry and a postal questionnaire assessing nasal symptoms including CRS, asthma, smoking, atopy and occupational exposure was sent out. Two reminders were sent after 1½ months and three months. A five-year follow-up of the cohort was made in 2018 using the same questionnaire and reminder intervals.

A total of 16,099 subjects (33%) answered the questionnaire in 2013. Of the 16,099 subjects who answered the questionnaire in 2013, 7,952 (49%) responded in 2018. The inclusion and exclusion from the sample are further illustrated in Figure 1.

CRS was defined according to the European position paper on rhinosinusitis and nasal polyposis (EPOS) criteria, as the presence of two or more symptoms for ≥ 12 weeks, of which one should be either nasal blockage/obstruction/congestion or nasal discharge (anterior/posterior nasal drip), called “major symptoms”, as well as additional symptoms, such as facial pain/pressure and/or a reduction in or the loss of smell, called “minor symptoms”⁽¹⁾.

Occupational exposure

Self-reported occupational exposure to airborne irritants and sensitizers was assessed by a set of questions which are illustrated in Table 1. Atopy, asthma and smoking were assessed with the questions also listed in Table 1. The questions have previously been used in the European Community of Respiratory Health Survey, the West Sweden Asthma Study⁽¹²⁾ and the Nord-Trøndelag Health Study (HUNT Study). Reported occupational exposure and baseline characteristics were based on the

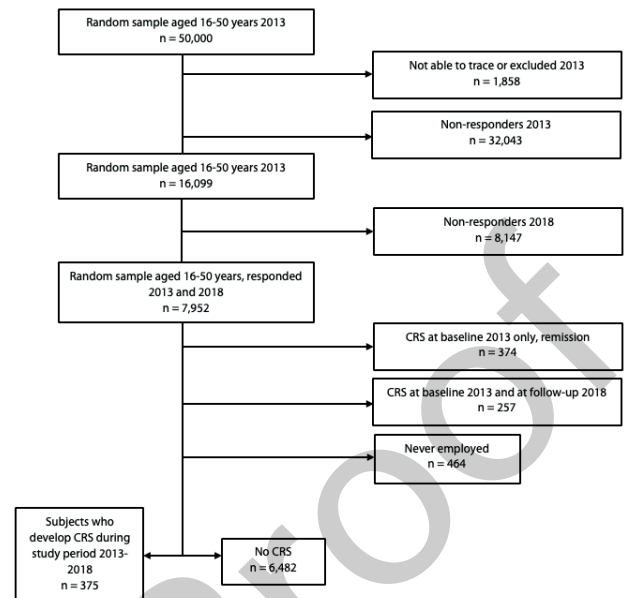


Figure 1. Flowchart of the study population .

2013 survey alone.

Fell et al.⁽¹³⁾ made a principal component analysis (PCA) of the same exposure variables managed in this paper, due to the fact that some of the self-reported occupational exposures are correlated. This analysis yielded six main exposure groups, metals/gases, damp/mould, exercise/cold, cleaning/cooking, organic dust and hair products/animals, which are used in the current paper. The main exposure variables were continuous and were calculated from a linear model with the exposures and their weights coming from the PCA.

Written and spoken information was given to all subjects and informed consent was collected. The study was approved by the Regional Committee for Medical and Health Research Ethics in Norway (2012/1665/REK sør-øst D).

Statistical analyses

As we were only interested in new-onset CRS during the follow-up period, all subjects reporting CRS at baseline in 2013 were excluded. New-onset CRS during the follow-up period was calculated. When describing the study population, comparisons between subjects with new-onset CRS and non-CRS were made using Fisher’s exact test and the chi-square test. $P < 0.05$ was considered significant. Univariable or multiple logistic regression was then applied to data, using CRS as the dependent variable and baseline characteristics or exposures as the independent variable. The odds ratio (OR) with 95% confidence interval (CI) was calculated for the likelihood of CRS. When the influence of six main groups of occupational exposure on CRS was investigated, a multiple logistic regression model was used with all six main

Table 1. Definitions of background factors and occupational exposure.

Definition	Questionnaire
Atopy	'Do you have an allergy that affects your nose, including hay fever?'
Asthma	'Has a physician ever diagnosed you with asthma?'
Smoking	1.'Do you smoke every day?' 2.'Do you only smoke occasionally?' 3.'Did you use to smoke?'
Ever smoking	A positive answer to any of the three questions
Current smoking	A positive answer to 'Do you smoke every day?' or 'Do you only smoke occasionally?'
Gas exposure	
Cooking fumes	'Have you ever, in your work, been exposed to cooking fumes?'
Car/engine exhaust	'Have you ever, in your work, been exposed to car/engine exhaust?'
Strong acids	'Have you ever, in your work, been exposed to strong acids?'
Dust exposure	
Stone dust	'Have you ever, in your work, been exposed to stone dust?'
Flour/grain dust	'Have you ever, in your work, been exposed to flour/grain dust?'
Wood dust	'Have you ever, in your work, been exposed to wood dust?'
Paper dust	'Have you ever, in your work, been exposed to paper dust?'
Textile dust	'Have you ever, in your work, been exposed to textile dust?'
Metal dust	'Have you ever, in your work, been exposed to metal dust?'
Other exposures	
Cleaning agents	'At work, have you worked with cleaning agents?'
Super glue	'At work, have you worked with super glue?'
Painting/varnishing	'At work, have you worked with painting/varnishing?'
Welding/metal smoke	'At work, have you worked with welding/metal smoke?'
Sewage	'At work, have you worked with sewage?'
Hair-care products	'At work, have you worked with hair-care products?'
Animals	'At work, have you worked with animals?'
Moisture exposure	
Moisture damage at work	'Have you worked in workplaces with visible moisture damage?'
Mould at work	'Have you worked in workplaces with visible mould?'
Smell of mildew at work	'Have you worked in workplaces with a smell of mildew?'
Moisture damage at home in the last 10 years	'Have you at any time over the course of the last 10 years seen signs of moisture damage, water leakage or mildew in your home?'
Working conditions	
Cold work	'Have you worked in workplaces with low temperatures (in the cold room or outdoors in the winter)?'
Physically strenuous job	'Have you had a physically strenuous job (so that you have been out of breath and sweaty)?'
Heavy lifting	'Have you had work with repetitive heavy lifting?'

groups, gender, age, allergy, atopy and smoking, in the same model. When the influence of single exposure was investigated, exposures and baseline characteristics that had a p-value of < 0.05 in the univariate analyses were used in a forward multiple logistic regression model with CRS as the dependent variable.

Results

The five-year cumulative incidence of new-onset CRS in the Telemark population between 2013 and 2018 was 5.5% (95% CI 4.9, 6.0). When stratified for gender, the cumulative incidence was 5.7% (95% CI 4.8, 6.6) for men and 5.3% (95% CI 4.6, 6.0) for women (p-value 0.55). When stratified for age, the five-year cumulative incidence was 7.4% (95% CI 6.0, 8.8) for the ages of 16-30, 4.8% (95% CI 3.8, 5.9) for the ages of 31-40 and 5.0%

Table 2. Description of study population (n=6857) with regard to age, gender, smoking habits, asthma and atopy.

	CRS (n=375)	Non-CRS (n=6,482)	Total	P ^a
Age				0.0012
16-30 years, n (%)	106 (7)	1321 (93)	1427	
31-40 years, n (%)	89 (5)	1749 (95)	1838	
41-50 years, n (%)	180 (5)	3412 (95)	3592	
Gender				0.55
Female gender, n (%)	215 (5)	3828 (95)	4043	
Male gender, n (%)	160 (6)	2654 (94)	2814	
Smoking ^b				0.0048
Never, n (%)	187 (5)	3651 (95)	3838	
Past, n (%)	85 (5)	1497 (95)	1582	
Current, n (%)	103 (7)	1334 (93)	1437	
Asthma				<0.001
No, n (%)	310 (5)	5924 (95)	6234	
Yes, n (%)	65 (10)	558 (90)	623	
Atopy				<0.001
No, n (%)	205 (4)	4703 (96)	4908	
Yes, n (%)	170 (9)	1779 (91)	1949	

^a Fisher's exact test and chi-square test; ^b Occasionally is set to current.

(95% CI 4.3, 5.7) for the ages of 41-50 (p-value 0.0012 for the age groups compared to one another).

A description of the study group is given in Table 2. Both asthma and atopy were more common in subjects who developed CRS compared with non-CRS (17% and 9% p-value <0,001, 45% and 27% p-value <0,001, respectively).

In the univariable analysis (Table 3), all the occupational exposures except paper dust and sewage were associated with an increased risk of new-onset CRS. Variables with p < 0.05 in the univariable analysis were then included in the forward multiple logistic regression analysis.

In the multiple logistic regression analysis (stepwise forward), hair-care products, super glue, strong acids, cooking fumes, wood dust, atopy, asthma and current smoking were statistically significantly associated with the risk of developing CRS (Table 4). Other variables in Table 3 were not statistically significantly associated with the risk of developing CRS adjusted for hair-care products, super glue, strong acids, cooking fumes, wood dust, atopy, asthma and current smoking. Females and males were also studied separately (Table 4). New-onset CRS in females was associated with animals, cleaning agents and strong acids. New-

Table 3. OR (univariable logistic regression analysis of CRS and different types of occupational exposure for the whole cohort and stratified by gender).

	All (n=6,857) OR _{uni} (95% CI)	Female (n=4,043) OR _{uni} (95% CI)	Male (n=2,814) OR _{uni} (95% CI)	n*
Cooking fumes	1.59 (1.24-2.03)	1.76 (1.31-2.36)	1.37 (0.85-2.21)	1179
Car/engine exhaust	1.59 (1.23-2.05)	1.82 (1.14-2.91)	1.55 (1.12-2.16)	1066
Strong acids	1.75 (1.34-2.29)	2.02 (1.38-2.97)	1.52 (1.03-2.23)	821
Stone dust	1.45 (1.11-1.90)	0.79 (0.37-1.71)	1.72 (1.24-2.39)	968
Flour/grain dust	1.75 (1.23-2.48)	2.04 (1.32-3.16)	1.37 (0.76-2.47)	431
Wood dust	1.56 (1.19-2.06)	1.71 (0.99-2.96)	1.54 (1.10-2.18)	859
Paper dust	1.25 (0.91-1.71)	1.20 (0.79-1.82)	1.33 (0.81-2.19)	700
Metal dust	1.61 (1.23-2.10)	1.11 (0.54-2.29)	1.83 (1.32-2.55)	880
Cleaning agent	1.51 (1.22-1.86)	1.89 (1.43-2.50)	1.15 (0.82-1.60)	2798
Super glue	1.78 (1.34-2.36)	1.79 (1.07-3.01)	1.80 (1.26-2.58)	723
Painting/varnishing	1.66 (1.27-2.16)	1.31 (0.76-2.26)	1.88 (1.35-2.62)	897
Welding/metal smoke	1.66 (1.27-2.16)	1.77 (0.84-3.70)	1.76 (1.27-2.44)	899
Sewage	1.46 (0.99-2.13)	1.89 (0.86-4.17)	1.33 (0.85-2.08)	409
Hair-care products	2.15 (1.42-3.25)	2.17 (1.38-3.43)	2.59 (0.89-7.52)	253
Animals	1.75 (1.28-2.39)	2.04 (1.35-3.07)	1.42 (0.87-2.32)	563
Moisture/mould/mildew	1.59 (1.25-2.03)	1.46 (1.03-2.06)	1.73 (1.23-2.43)	1247
Cold	1.38 (1.11-1.71)	1.47 (1.08-1.99)	1.29 (0.94-1.78)	2115
Physically strenuous work	1.46 (1.18-1.80)	1.34 (1.01-1.78)	1.70 (1.19-2.41)	3013

* Number of individuals with exposure.

Table 4. OR_{adj} (odds ratio, forward multiple logistic regression analysis of risk of CRS and different types of occupational exposure for the whole cohort and stratified by gender adjusted for the other occupational exposures in this table and age, atopy, asthma and smoking). Variables with p<0.05 in the univariable analysis (Table 3) were included in the forward multiple logistic regression analysis. The remaining variables were not statistically significantly associated with CRS (p>0.05, not shown) when adjusted for the variables in the table below.

Only ever employed (n=6857)	OR (95% CI)	Two-sided p-value
Hair-care products	1.67 (1.09, 2.56)	0.019
Super glue	1.44 (1.06, 1.95)	0.019
Strong acids	1.42 (1.07, 1.89)	0.015
Cooking fumes	1.38 (1.07, 1.77)	0.014
Wood dust	1.36 (1.02, 1.82)	0.039
Female		
Animals	1.60 (1.05, 2.45)	0.030
Cleaning agent	1.57 (1.16, 2.11)	0.0032
Strong acids	1.57 (1.05, 2.34)	0.027
Male		
Metal dust	1.57 (1.11, 2.23)	0.012
Stone dust	1.49 (1.05, 2.11)	0.024

onset CRS in males was associated with metal dust and stone dust. New-onset CRS was also associated with atopy (OR 1.98, 95% CI 1.59, 2.47 p-value <0.001), asthma (OR 1.69, 95% CI 1.26, 2.27 p-value <0.001) and current smoking (OR 1.44, 95% CI 1.13, 1.83 p-value 0.0028) (not shown). Young age was also associated with new-onset CRS (OR per year 0.98, 95% CI 0.973, 0.995 p-value 0.0051) (not shown).

In the multiple logistic regression analysis of the six main groups of occupational exposure, metals/gases, damp/mould, cleaning/cooking, organic dust and hair-products/animals were independently related to new-onset CRS (Table 5).

Discussion

To our knowledge, this is the first large, prospective, population-based cohort of adult subjects where new-onset CRS has been studied during a five-year period in relation to occupational exposure. The overall five-year incidence of new-onset CRS was 5.5% (95% CI 4.9, 6.0) and the most consistent findings were that exposure to hair-care products, cleaning agents among women, super glue, cooking fumes, wood dust and young age were associated with new-onset CRS. When analysing six main exposure groups, metals/gases, damp/mould, cleaning/cooking, organic dust and hair-care products/animals were found to be independently associated with new-onset CRS.

The observed association between the development of CRS and

Table 5. OR_{adj} (multiple logistic regression analysis of CRS and the six main exposure groups, adjusted for age, gender, smoking, asthma, atopy, the other exposure groups included in the model) and stratified by gender.

	OR _{adj} (95% CI) Two-sided p-value		
	Only ever employed n=6,857	Male n=2,814	Female n=4,043
Metal and gases (OR per SD)	1.27 (1.13, 1.42)	1.33 (1.16, 1.53)	1.12 (0.86, 1.47)
Damp and mould (OR per SD)	1.13 (1.02, 1.25)	1.08 (0.94, 1.24)	1.16 (0.99, 1.36)
Exercise and cold (OR per SD)	1.10 (0.97, 1.24)	1.14 (0.95, 1.36)	0.99 (0.82, 1.19)
Cleaning and cooking (OR per SD)	1.15 (1.03, 1.29)	0.98 (0.80, 1.20)	1.28 (1.11, 1.48)
Organic dust (OR per SD)	1.10 (1.0008, 1.2142)	1.15 (0.98, 1.34)	1.06 (0.93, 1.21)
Hair products and animals (OR per SD)	1.14 (1.03, 1.26)	1.03 (0.84, 1.25)	1.18 (1.06, 1.33)

exposures indicates the need for greater focus on the identification and reduction of occupational exposure when making clinical assessments of patients with CRS.

Our data show that working with hair-care products increases the risk of CRS, which is consistent with previous studies showing an increased risk of respiratory problems in this group^(14, 15). Likewise, exposure to glue has previously been reported to contribute to the development of asthma and rhinitis^(16, 17). We can only speculate about the specific occupations of subjects who report exposure to hair-care products and glue. They may include occupations such as hairdressers, dental technicians and nail artists. These working groups are not only exposed to aerosols and vapours but are also in contact with the substances through the skin, which is an additional exposure pathway that needs further investigation. When applying protective measures, both aerosol exposure and dermal contact should be avoided until proven otherwise.

New-onset CRS among females was also associated with cleaning agents in this study. This is in accordance with previous studies^(7, 10, 18) and, to our knowledge, this is the first large, population-based study to confirm the connection between new-onset CRS and cleaning agents. Cleaning agents have also been linked to work-related asthma^(19, 20) and a recent study by Rosenman et al. showed that the percentage of work-related

asthma cases from cleaning products was unchanged over the years⁽²¹⁾. This indicates that further research and preventive measures are needed to avoid a harmful impact on the respiratory system.

Occupational exposure to strong acids, such as hydrochloric acid, sulphuric acid and nitric acid, was also found to increase the risk of developing CRS in our study. To our knowledge, strong acids and their association with CRS have not previously been studied. However, previous studies show that they may have a harmful impact on the lower airways⁽²²⁻²⁴⁾. We hypothesise that the cause of damage is the vapourisation of the acids which are then inhaled. Industrial workers in the Telemark region have worked with these acids and their occupations include bricklaying, industrial cleaning and industrial production.

Cooking fumes were also related to an increased risk of CRS in our study. To our knowledge, occupational exposure to cooking fumes and its relationship to CRS has not been previously studied. However, previous studies have shown that cooking fumes have a negative impact on the lower airways^(25, 26).

In the present study, wood dust was related to an increased risk of new-onset CRS. Wood dust exposure has previously been associated with nasal symptoms and chronic inflammatory rhinitis in nasal cytology^(27, 28). Our findings further strengthen this correlation. Wood dust has also been associated with lower airway disease and a decline in lung function among women⁽²⁹⁾. The inhaled air often passes the nose first before reaching the lungs and the nose also acts as the first line of defence against inhaled particles and fumes. Since the respiratory mucosa of the nose, sinuses and bronchi is similar, the inflammatory response can also be expected to be similar.

As some of the occupational exposures are closely related to each other, we also conducted an analysis of six main exposure groups and their relationship to CRS. We found that metals/gases, damp/mould, cleaning/cooking, organic dust and hair-care products/animals were associated with new-onset CRS. From this, we are not able to discern which specific exposure could be the cause of CRS. However, this illustrates that larger population studies are needed and that additional exposures could be relevant regarding new-onset CRS. Protecting the airway from exposure should be considered and implemented in all occupational environments at risk. In developed countries, such as those in Scandinavia, this should be possible to achieve.

At baseline in 2013, the prevalence of CRS in the Telemark population was 9%, which correlates well with other studies^(2, 30). Population-based studies in general and, in particular, unselected populations regarding the incidence of CRS are few in

number. In a Canadian study by Xu Y et al., the mean incidence of CRS was 2.5 per 1,000 population⁽³¹⁾. This study was, however, based on health-care registers where CRS was diagnosed and coded by a physician after the patient sought medical care and it was not based on self-reported symptoms in a general population. A study by Novis et al. showed that patients diagnosed with CRS by a non-otorhinolaryngologist are unlikely to fulfil the diagnostic criteria for CRS⁽³²⁾ and these data should be used with caution. In this study, the cumulative five-year incidence was 5.5%, which is surprisingly high, given that the subjects in this study were aged between 16 and 55. In younger age groups, CRS without nasal polyps is more common; this includes allergic rhinitis that has a peak onset around 20 years of age⁽¹⁾. CRS with nasal polyps typically starts at a later age⁽³³⁾. In this questionnaire-based study, we are unable to distinguish between the two, as they exhibit similar symptoms. It is possible, however, that the subjects who reported CRS in 2013 and who were excluded from this study represented more allergic rhinitis since the mean age was lower. The fact that the risk of having new-onset CRS decreased with increasing age in this study adds further support. As the cohort is getting older, other aetiological factors could be of importance and occupational exposure is one potential aetiological factor in CRS that is likely to develop after some time. A previous study has shown that the prevalence of CRS increases with age and levels off at around 60⁽³⁴⁾. CRS is a chronic disease which persists over time and its prevalence may thus increase over time, despite a decreasing incidence.

Smoking is a known risk factor for asthma⁽³⁵⁾, as well as CRS, as we have previously demonstrated⁽¹⁰⁾. The present study illustrates that current smokers run a 44% higher risk of developing CRS compared with the non-CRS group (OR 1.44, 95% CI 1.13, 1.83, p-value 0.0028). This study also shows that asthma (OR 1.69, 95% CI 1.26, 2.27, p-value <0.001) and atopy (OR 1.98, 95% CI 1.59, 2.47, p-value <0.001) are associated with new-onset CRS when adjusted for the other variables in Table 4 as well as age, atopy, asthma and smoking. Previous studies have also confirmed the association between asthma and atopy and CRS^(36, 37), but the mechanisms involved are still unclear. A case-control study showed that patients with rhinosinuitis who need surgery, especially repeated surgery, are more likely to have been exposed to inhaled noxious agents at work than controls⁽³⁸⁾.

The study has several strengths and weaknesses. One important strength of this study is the large number of subjects from the same industrialised, geographical area. Studying one geographical area may introduce bias that could reduce the generalisation of the results. The random population in this study had the same prevalence of smoking and asthma as the general population in Norway⁽³⁹⁾. A multicentre study would have reduced any systematic bias related to the Telemark population, but it could

also introduce bias related to different definitions of symptoms or exposures between populations. Another strength is the longitudinal and prospective design of the study which allows us to follow individuals over a period of five years. Self-reported questionnaire data without a clinical validation using a nasal endoscopy and/or a CT scan may, however, lead to misclassification. However, previous work has shown that a symptom-based definition of CRS as stated by the EPOS has a reasonable correlation to endoscopic findings and should be sufficient for use in epidemiological studies⁽⁴⁰⁾. Other studies have also demonstrated an acceptable correlation between symptom-based CRS and CT findings and clinical diagnosis by a trained otorhinolaryngologist respectively^(41,42). It should also be noted that self-reported exposure may both over- and underestimate the actual exposure, especially if there has been a long delay between the exposure and the self-report. Subjects who have developed symptoms may also be more prone to report exposure, but the longitudinal design should reduce the risk of this bias, as exposure is reported prior to the development of CRS. The response rate in this study was comparable to that in other recent population-based studies⁽¹¹⁾. There is a risk that subjects with CRS symptoms who have been exposed to airborne irritants and sensitizers are over-represented among the respondents and that this would thus result in an increased incidence of CRS. The overall prevalence of CRS at inclusion in this study, in 2013, was, however, 9%, which is similar to that in other studies^(2,30). When analysing multiple variables, as has been done in the present study, there is always the risk of mass significance. However, the occupational exposures have not been randomly chosen, but each variable is a well-established risk factor for airway disease. The aim of this study, as stated in the introduction, was

to study new-onset CRS in relation to occupational exposures. It was therefore not possible to study other important aspects, such as the correlation with disease severity or response to treatment.

Conclusion

To our knowledge, this is the first large, prospective, population-based cohort of adult subjects, where new-onset CRS has been studied during a five-year period in relation to occupational exposure. The overall five-year incidence of new-onset CRS was 5.5% (95% CI 5.0, 6.0) and the most consistent findings were that exposure to hair-care products, cleaning agents among women, super glue, cooking fumes, wood dust and young age were associated with new-onset CRS. It is important that physicians treating patients with CRS are aware of this possible relationship and ask patients about workplace exposure.

Acknowledgements

The authors would like to thank Geir Klepaker, Hilde Jernkvist, Cathrine Goberg Olsen, Gølin Finkenhagen Gundersen and Nikola Zivadinovic, who collected the data in Norway. Sources of funding: The Medical Society of Gothenburg (GLS), ALF Regional and governmental funding, Swedish Association for Otorhinolaryngology Head & Neck Surgery, Telemark Hospital, Norway.

Authorship contribution

UKEC, JH, HJ and MVS wrote the paper. All other authors commented and reviewed and gave their final approval.

Conflict of interest

The authors declare that they have no competing interests.

References

- Fokkens WJ, Lund VJ, Hopkins C, et al. European Position Paper on Rhinosinusitis and Nasal Polyps 2020. *Rhinology* 2020;58:1-464.
- Hastan D, Fokkens WJ, Bachert C, et al. Chronic rhinosinusitis in Europe--an underestimated disease. A GA²LEN study. *Allergy* 2011;66:1216-23.
- Rudmik L, Smith TL. Quality of life in patients with chronic rhinosinusitis. *Curr Allergy Asthma Rep* 2011;11:247-52.
- Bengtsson C, Jonsson L, Holmström M, et al. Incident Chronic Rhinosinusitis Is Associated With Impaired Sleep Quality: Results of the RHINE Study. *J Clin Sleep Med* 2019;15:899-905.
- Ylitalo-Heikkilä M, Virkkula P, Sintonen H, Lundberg M, Roine RP, Hytönen M. Different rhinologic diseases cause a similar multidimensional decrease in generic health-related quality of life. *Clin Otolaryngol* 2018;43:1487-93.
- Koh DH, Kim HR, Han SS. The relationship between chronic rhinosinusitis and occupation: the 1998, 2001, and 2005 Korea National health and nutrition examination survey (KNHANES). *Am J Ind Med* 2009;52:179-84.
- Hellgren J, Lillienberg L, Jarlstedt J, Karlsson G, Torén K. Population-based study of non-infectious rhinitis in relation to occupational exposure, age, sex, and smoking. *Am J Ind Med* 2002;42:23-8.
- Radon K, Gerhardinger U, Schulze A, et al. Occupation and adult onset of rhinitis in the general population. *Occup Environ Med* 2008;65:38-43.
- Hox V, Steelant B, Fokkens W, Nemery B, Hellings PW. Occupational upper airway disease: how work affects the nose. *Allergy* 2014;69:282-91.
- Clarhed UKE, Svendsen M, Schiöler L, et al. Chronic Rhinosinusitis Related to Occupational Exposure: The Telemark Population Study. *J Occup Environ Med* 2018;60:656-60.
- Abrahamsen R, Svendsen MV, Henneberger PK, et al. Non-response in a cross-sectional study of respiratory health in Norway. *BMJ open* 2016;6:e009912-e.
- Lötvall J, Ekerljung L, Rönmark EP, et al. West Sweden Asthma Study: prevalence trends over the last 18 years argues no recent increase in asthma. *Respir Res* 2009;10:94.
- Fell AK, Abrahamsen R, Henneberger PK, et al. Breath-taking jobs: a case-control study of respiratory work disability by occupation in Norway. *Occup Environ Med* 2016;73:600-6.
- Moscato G, Galdi E. Asthma and hair-dressers. *Curr Opin Allergy Clin Immunol* 2006;6:91-5.
- Torén K, Hörte LG. Asthma mortality and occupation in Sweden 1981-1992. *Am J Ind Med* 1997;31:678-81.

16. Kopp SK, McKay RT, Moller DR, Cassedy K, Brooks SM. Asthma and rhinitis due to ethylcyanoacrylate instant glue. *Ann Intern Med* 1985;102:613-5.
17. Lindström I, Suojalehto H, Henriks-Eckerman ML, Suuronen K. Occupational asthma and rhinitis caused by cyanoacrylate-based eyelash extension glues. *Occup Med (Lond)* 2013;63:294-7.
18. Folletti I, Zock JP, Moscato G, Siracusa A. Asthma and rhinitis in cleaning workers: a systematic review of epidemiological studies. *J Asthma* 2014;51:18-28.
19. Rosenman KD, Reilly MJ, Schill DP, et al. Cleaning products and work-related asthma. *J Occup Environ Med* 2003;45:556-63.
20. Weinmann T, Forster F, von Mutius E, et al. Association Between Occupational Exposure to disinfectants and asthma in young adults working in cleaning or health services: results from a cross-sectional analysis in Germany. *J Occup Environ Med* 2019;61:754-9.
21. Rosenman K, Reilly MJ, Pechter E, et al. Cleaning Products and Work-Related Asthma, 10 Year Update. *J Occup Environ Med* 2020;62:130-7.
22. Andersson E, Knutsson A, Hagberg S, et al. Incidence of asthma among workers exposed to sulphur dioxide and other irritant gases. *Eur Respir J* 2006;27:720-5.
23. Henneberger PK, Olin AC, Andersson E, Hagberg S, Torén K. The incidence of respiratory symptoms and diseases among pulp mill workers with peak exposures to ozone and other irritant gases. *Chest* 2005;128:3028-37.
24. Vizcaya D, Mirabelli MC, Gimeno D, et al. Cleaning products and short-term respiratory effects among female cleaners with asthma. *Occup Environ Med* 2015;72:757-63.
25. Neghab M, Delikhoon M, Norouziyan Baghani A, Hassanzadeh J. Exposure to cooking fumes and acute reversible decrement in lung functional capacity. *Int J Occup Environ Med* 2017;8:207-16.
26. Svedahl SR, Hilt B, Svendsen K. Work environment factors and respiratory complaints in Norwegian cooks. *Int Arch Occup Environ Health* 2020;93:205-12.
27. Lovato A, Staffieri C, Ottaviano G, et al. Woodworkers and the inflammatory effects of softwood/hardwood dust: evidence from nasal cytology. *Eur Arch Otorhinolaryngol* 2016;273:3195-200.
28. Löfstedt H, Hagström K, Bryngelsson IL, Holmström M, Rask-Andersen A. Respiratory symptoms and lung function in relation to wood dust and monoterpene exposure in the wood pellet industry. *Ups J Med Sci* 2017;122:78-84.
29. Jacobsen G, Schlünssen V, Schaumburg I, Taudorf E, Sigsgaard T. Longitudinal lung function decline and wood dust exposure in the furniture industry. *Eur Respir J* 2008;31:334-42.
30. Shi JB, Fu QL, Zhang H, et al. Epidemiology of chronic rhinosinusitis: results from a cross-sectional survey in seven Chinese cities. *Allergy* 2015;70:533-9.
31. Xu Y, Quan H, Faris P, et al. Prevalence and incidence of diagnosed chronic rhinosinusitis in Alberta, Canada. *JAMA Otolaryngol Head Neck Surg* 2016;142:1063-9.
32. Novis SJ, Akkina SR, Lynn S, Kern HE, Keshavarzi NR, Pynnonen MA. A diagnostic dilemma: chronic sinusitis diagnosed by non-otolaryngologists. *Int Forum Allergy Rhinol* 2016;6:486-90.
33. Klossek JM, Neukirch F, Pribil C, et al. Prevalence of nasal polyposis in France: a cross-sectional, case-control study. *Allergy* 2005;60:233-7.
34. Chen Y, Dales R, Lin M. The epidemiology of chronic rhinosinusitis in Canadians. *Laryngoscope* 2003;113:1199-205.
35. Polosa R, Thomson NC. Smoking and asthma: dangerous liaisons. *Eur Respir J* 2013;41:716-26.
36. Gutman M, Torres A, Keen KJ, Houser SM. Prevalence of allergy in patients with chronic rhinosinusitis. *Otolaryngol Head Neck Surg* 2004;130:545-52.
37. Jarvis D, Newson R, Lotvall J, et al. Asthma in adults and its association with chronic rhinosinusitis: the GA2LEN survey in Europe. *Allergy* 2012;67:91-8.
38. Hox V, Delrue S, Scheers H, et al. Negative impact of occupational exposure on surgical outcome in patients with rhinosinusitis. *Allergy* 2012;67:560-5.
39. Abrahamsen R, Fell AK, Svendsen MV, et al. Association of respiratory symptoms and asthma with occupational exposures: findings from a population-based cross-sectional survey in Telemark, Norway. *BMJ Open* 2017;7:e014018.
40. Tomassen P, Newson RB, Hoffmans R, et al. Reliability of EP30S symptom criteria and nasal endoscopy in the assessment of chronic rhinosinusitis—a GA² LEN study. *Allergy* 2011;66:556-61.
41. Bhattacharyya N, Lee LN. Evaluating the diagnosis of chronic rhinosinusitis based on clinical guidelines and endoscopy. *Otolaryngol Head Neck Surg* 2010;143:147-51.
42. Pilan RR, Pinna FR, Bezerra TF, et al. Prevalence of chronic rhinosinusitis in Sao Paulo. *Rhinology* 2012;50:129-38.

Ulrika K. E. Clarhed
Department of Otorhinolaryngology
Head & Neck Surgery
Institute of Clinical Sciences
University of Gothenburg
Gröna Stråket 9
SE-413 45 Göteborg
Sweden

Tel: +46 31 3421000
Fax: +46 31 825679
E-mail: Ulrika.Clarhed@vgregion.se