Chronic rhinosinusitis

Epidemiological studies on occupational exposure, obesity and sick leave

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Gothenburg 2025

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Figures 1, 2, 4, 5, 6, 7 and 8 were created with BioRender.com.

ISBN 978-91-8069-991-4 (PRINT)
ISBN 978-91-8069-992-1 (PDF)

Printed in Borås, Sweden 2025
Printed by Stema Specialtryck AB

To Daniel, Judith, Esther and Arthur

"You don't realize how much you take breathing for granted until you have a stuffed nose"

Chronic rhinosinusitis

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ABSTRACT

Chronic rhinosinusitis (CRS) is a disease characterised by chronic inflammation of the mucosa lining the nose and paranasal sinuses, with a significant negative impact on patients' quality of life. CRS affects around 11% of the population of Europe, and currently there is no curative, but only symptomatic, treatment. For the majority of patients, the cause of CRS is unknown. Risk factors include asthma, smoking and allergy, and in recent years occupational exposure has been acknowledged as a contributing cause. However, there is a scarcity of studies on CRS that use the current definition stipulated by the European Position Paper on Rhinosinusitis and Nasal Polyps (EPOS) and deal with occupational exposures. The papers appended to this thesis relate to the Telemark population study in the south of Norway. The aim of this thesis was to investigate further the epidemiology of CRS, with the focus on occupational exposures, obesity and sick leave.

Paper I comprises a cross-sectional study in which the prevalence of CRS in the Telemark population was 9% in 2013. Occupational exposure to paper dust, cleaning agents, metal dust, animals, mould/moisture/mildew and physically strenuous work was associated with an increased odds of having CRS.

Paper II describes a prospective study in which occupational exposures to hair-care products, cleaning agents (among women), super glue, strong acids, cooking fumes, and wood dust were associated with an increased odds of new-onset CRS in a 5-year period (2013–2018). The cumulative, 5-year incidence of new-onset CRS in the Telemark population was found to be 5.5%.

Paper III represents a prospective study for the period of 2013–2018 in which the odds of new-onset CRS was found to be 53% higher in obese subjects (BMI≥30) than in subjects with normal weight (18.5≤BMI<25).

Paper IV describes an investigation of the frequency of sick leave among subjects in the previous 12 months, cross-sectionally in 2013 and 2018. Subjects with CRS had around a 60% increased odds of taking sick leave, as compared with subjects without CRS, in both 2013 and 2018. When stratifying for sex in 2013, women with CRS had an almost two-fold higher odds of taking sick leave compared to men with CRS. Sick leave was found to be more common for subjects with CRS than for subjects without CRS in some occupational groups.

The **main conclusions** from this thesis work are that: 1) occupational exposure is a risk factor for CRS, and particular consideration should be given to cleaning agents, hair-care products, paper dust, metal dust, animals, mould/moisture/mildew, super glue, strong acids, cooking fumes and wood dust; 2) high BMI is a risk factor for CRS and should be taken into consideration when assessing patients with CRS; and 3) subjects with CRS in the Telemark population have a higher frequency of sick leave in the previous year compared to subjects without CRS and, in this context, women appear to be more affected than men.

Keywords: sinusitis, occupation, epidemiology, sick leave, obesity

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SAMMANFATTNING PÅ SVENSKA

Kronisk rinosinuit (CRS) är en sjukdom som innebär långvarig inflammation i slemhinnan som täcker ytan i näsa och bihålor. Studier har visat att ungefär 11% av befolkningen i Europa har CRS. Besvären består av nästäppa, rinnsnuva, ansiktsvärk och nedsatt, eller avsaknad av, luktsinne. Patienter med CRS har ofta sänkt livskvalitet med ökad risk för bland annat depression, ångest och sömnsvårigheter. I dagsläget finns det endast lindrande, ej botande, behandling för CRS. Orsaken till CRS anses vara multifaktoriell med riskfaktorer som astma, rökning och allergi. I botten anser man att det rör sig om en skada i slemhinnan som orsakats av en kombination av inre faktorer (hos individen, en benägenhet för att utveckla inflammation vid vissa stimuli) och yttre faktorer (från omgivningen). Yttre faktorer kan vara till exempel rökning och ämnen i luften som uppkommer i samband med olika yrken.

Denna avhandling syftar till att studera olika yrkesexponeringars koppling till utveckling av CRS, Body Mass Index (BMI) koppling till CRS samt hur CRS påverkar sjukskrivning. Arbetena i denna avhandling är utförda som en del av Telemarksstudien, en norsk populationsstudie på personer mellan 16-50 år från södra Norge som utfördes år 2013 samt 2018.

I delarbete I studerades 16,099 personer år 2013 och där var yrkesexponering för pappersdamm, rengöringsmedel, metalldamm, djur, fukt och mögel samt fysiskt ansträngande arbete korrelerat till ökad risk för att ha CRS. Förekomsten av CRS i befolkningen i Telemark var 9%.

I delarbete II studerades 7,952 personer mellan år 2013 och 2018 och då var yrkesexponering för hårvårdsprodukter, rengöringsmedel hos kvinnor, superlim, starka syror, matlagningsångor och trädamm kopplat till en ökad risk för att insjukna i CRS.

I delarbete III studerades 5,769 personer under åren 2013-2018 och resultaten visade att personer med fetma (BMI≥30) har 53% ökad odds att insjukna i CRS jämfört med normalviktiga personer (18,5≤BMI<25).

I delarbete IV studerades 15,484 personer år 2013 samt 13,966 personer år 2018. Resultaten visade att personer med CRS är ca 60% mer sjukskrivna generellt under det gångna året jämfört med personer utan CRS. Skillnaden verkar vara extra uttalad vad gäller kvinnor med CRS jämfört med män med CRS.

Sammanfattningsvis visar denna avhandling att yrkesexponering är en riskfaktor för att utveckla CRS. Särskilt uppmärksam bör man vara vid yrkesexponering för rengöringsmedel, hårvårdsprodukter, pappersdamm, metalldamm, trädamm, djur, fukt och mögel, superlim, matlagningsångor och starka syror. Högt BMI ökar risken för att insjukna i CRS. Personer med CRS är generellt mer sjukskrivna under det gångna året jämfört med personer utan CRS, och detta verkar vara särskilt uttalat hos kvinnor med CRS.

LIST OF PAPERS

This thesis is based on the following studies, which are referred to in the text by their Roman numerals.

I. **Clarhed UKE**, Svendsen M, Schiöler L, Kongerud J, Torén K, Hellgren J, Fell AK (2018) Chronic rhinosinusitis related to occupational exposure: The Telemark population study. J Occup Environ Med 60: 656-660.

II. **Clarhed UKE**, Johansson H, Svendsen MV, Torén K, Fell AKM, Hellgren J (2020) Occupational exposure and the risk of new-onset chronic rhinosinusitis: a prospective study 2013-2018. Rhinology 58: 597-604.

III. **Clarhed UKE**, Schiöler L, Torén K, Fell AKM, Hellgren J (2022) BMI as a risk factor for the development of chronic rhinosinusitis: a prospective, population-based study. Eur Arch Otorhinolaryngol 279: 4953-4959.

IV. **Clarhed UKE**, Schiöler L, Torén K, Fell AKM, Hellgren J (2024) Women suffering from chronic rhinosinusitis in Norway are more likely to take sick leave. PLoS One Nov 1;19(11):e0313122

CONTENTS

ABBREVIATIONS

BMI	Body Mass Index
CI	Confidence interval
CRS	Chronic rhinosinusitis
CRSsNP	CRS without nasal polyps
CRSwNP	CRS with nasal polyps
CT	Computed tomography
EPOS	European Position Paper on Rhinosinusitis and Nasal Polyps
ESS	Endoscopic sinus surgery
FESS	Functional endoscopic sinus surgery
HMW	High molecular weight
IgA	Immunoglobulin A
IgD	Immunoglobulin D
IgE	Immunoglobulin E
IgG	Immunoglobulin G
IgM	Immunoglobulin M
IL-2	Interleukin 2
IL-4	Interleukin 4
IL-5	Interleukin 5
IFN-γ	Interferon gamma

ISCO-88	International standard classification of occupations
JEM	Job-exposure matrix
LMW	Low molecular weight
OcR	Occupational rhinitis
OR	Odds Ratio
Th1-cell	T-helper cell type 1
Th2-cell	T-helper cell type 2

1 INTRODUCTION

The nose and sinuses make up a crucial component of the global airways. By heating, humidifying and cleaning the air we breathe, the nose acts as a gatekeeper for the lower airways. Sino-nasal disease results in a loss of this function, which can have detrimental effects on the upper and lower airways, as well as human well-being (Hens & Hellings 2006).

Historians believe that the ancient Egyptians had knowledge of the paranasal sinuses. As part of the process of mummifying a body, they used special tools to remove the brain through the nasal cavity, probably through the ethmoid cells. Hippocrates, in all probability, also had knowledge of the paranasal sinuses, although the sinuses haven't been detailed in his work. Leonardo da Vinci later created anatomical drawings of the human body that portrayed the frontal and maxillary sinuses. Around 1650, Nathaniel Highmore described and drew the maxillary sinus, which is sometimes still referred to as *Highmore's antrum*. Since da Vinci's drawings weren't found until 1901, it was believed for many years that Nathaniel Highmore was the first to draw and describe the maxillary sinus (Mavrodi & Paraskevas 2013). In the second half of the 20th century, Walter Messerklinger conducted studies of muco-ciliary clearance and its role in the pathogenesis of sinusitis. Subsequently, Heinz Stammberger developed functional endoscopic sinus surgery (FESS) based on these findings (Govindaraj et al 2010). This has contributed to our modern understanding of sino-nasal anatomy and function.

Today, rhinosinusitis is the term used to describe inflammation in the nose and paranasal sinuses. It depicts the inter-connection between the nose and sinuses with regards to both physiology and pathophysiology, and shows that the two are closely connected (Fokkens et al 2020).

1.1 CHRONIC RHINOSINUSITIS

1.1.1. DEFINITIONS

The classical symptoms of chronic rhinosinusitis include nasal congestion, discoloured nasal secretions, facial pain/pressure, and absence of or reduced sense of smell. Similar definitions exist today (Fokkens et al 2020, Marple et al 2009), although the most widely used definition in clinical practice and in academic research (and the one that we apply) is that stated in the European

Position Paper on Rhinosinusitis and Nasal Polyps (EPOS). In EPOS, nasal congestion and nasal secretion are considered major symptoms, whereas facial pain/pressure and absence of/reduced sense of smell are deemed to be minor symptoms. According to EPOS, the definition of CRS is as follows: 'Presence of two or more symptoms, one of which should be either *nasal blockage/obstruction/congestion* or *nasal discharge (anterior/posterior nasal drip)* ± *facial pain/pressure* ± *reduction or loss of smell.* The symptoms should be present for 12 weeks or more. For epidemiological studies, the definition is based on symptomatology usually without an ear-nose-and-throat examination or radiology. A clinical definition includes endoscopic signs of *nasal polyps and/or mucopurulent discharge primarily from middle meatus and/or oedema/mucosal obstruction primarily in middle meatus and/or CT-changes (showing mucosal changes within the ostiomeatal complex and/or sinuses)'* (Fokkens et al 2020).

1.1.2 INFLAMMATORY MECHANISMS

CRS is considered to be a multi-factorial inflammatory disease and different underlying mechanisms have been proposed, including colonisation by bacteria, such as *Staphylococcus aureus* and biofilms thereof, and viral and fungal infections (Fokkens et al 2020, Ponikau et al 1999). Since the aetiology of CRS remains largely unknown, it is important to investigate possible external factors and their roles in the development of CRS. These external factors include infections, air pollution, and respiratory irritants in the occupational environment.

The inflammatory reactions that occur in the respiratory mucosa in cases of CRS involve different components of the immune system. To better understand where occupational exposure may interact with the inflammatory mechanisms of CRS, an understanding of the immune system and key inflammatory reactions is necessary. There follows a schematic and abridged summary of the inflammatory mechanisms related to CRS in this thesis.

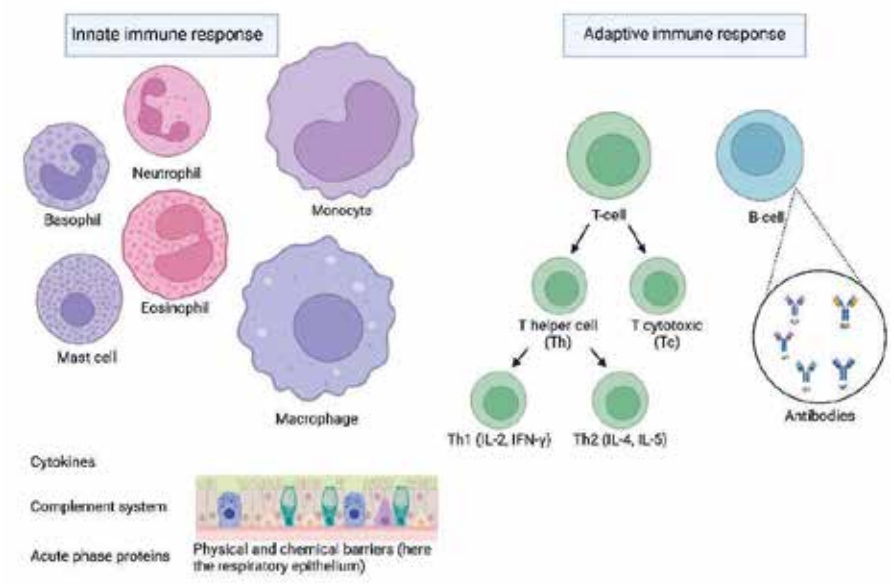

Figure 1. Some of the main components of the innate and the adaptive immune system. The figure is not to scale.
Figure adapted from Parkin J & Cohen B (2001) An overview of the immune system. Lancet 357: 1777-1789 and Gohy S et al (2020) Key role of the epithelium in chronic upper airways diseases. Clin Exp Allergy 50: 135-146.

Our complex and multi-faceted immune system is divided into two parts: the *innate immune response* and the *adaptive immune response*. The innate immune system is comprised of neutrophils, monocytes, macrophages, the complement system, cytokines and acute-phase proteins (Parkin & Cohen 2001). It also includes physical and chemical barriers such as the respiratory and the gastrointestinal mucosa. The normal respiratory mucosa forms a physical barrier of muco-ciliated pseudostratified epithelium, which is comprised mainly of ciliated cells and goblet cells that work together to produce and continuously remove mucus. The sinonasal epithelium also produces several defence proteins, such as mucins, cytokines and chemokines (Gohy et al 2020). The gastrointestinal mucosa acts as both a physical barrier (the intestinal epithelial cells) and a chemical barrier (multiple defence molecules, such as mucins and immunoglobulins, are secreted by the epithelial cells into the mucosal fluid) (Sperandio et al 2015, Sansonetti 2004). While the innate immune system responds immediately to invading pathogens and is of vital importance to even the simplest animals, it lacks specificity. In contrast, the adaptive immune system takes days or weeks to react but is specifically

targeted. The adaptive immune system encompasses B lymphocytes and T lymphocytes, both of which develop from progenitor cells in the bone marrow. B cells remain in the bone marrow throughout their development, whereas T cells migrate to the thymus to continue their development. The T cells subsequently migrate to the secondary lymphoid tissues, such as the lymph nodes, spleen, tonsils, and mucosa-associated lymphoid tissue. Around 95% of the T lymphocytes are found within the lymphoid tissue. However, they do not remain stationary but instead move around from one lymphoid tissue to the next. In fact, it takes 1–2 days for a T lymphocyte to travel around the whole body via the blood or lymph. T cells can proliferate and differentiate into three types of cells: CD4 Th1-cells; CD4 Th2-cells; and CD8 cytotoxic cells. CD4 Th-cells are categorised according to the cytokines that they produce. Th1-cells produce interleukin 2 (IL-2) and interferon-gamma (IFN-γ), amongst other immune mediators, and mainly induce a cell-mediated inflammatory response. A Th1 response is fundamental to enabling the host to defeat intracellular pathogens, although it may contribute to the pathogenesis of autoimmune diseases such as rheumatoid arthritis (RA) and multiple sclerosis. Th2 cells, on the other hand, produce IL-4 (stimulating B cells to produce IgE) and IL-5 (promoting the growth of eosinophils). As such, a Th2 response is linked to allergic disease (Parkin & Cohen 2001).

According to Jutel, Agache and colleagues: '*Hypersensitivity refers to an undesirable, uncomfortable or damaging response that arises from a tissue cell dysfunction or immune system overreaction. Allergy is an abnormal or exaggerated reaction to exogenous stimuli which involves various types of hypersensitivity reactions engaging antibodies, immune cell-mediated, tissue-driven or metabolic mechanisms resulting in the development of respiratory, skin, eye, gastrointestinal and other symptoms, including anaphylaxis*' (Jutel, Agache et al 2023). According to Gell and Coombs (1963), hypersensitivity reactions have traditionally been divided into four different types of reactions: Type 1 reactions are immediate and IgE-mediated; Type 2 are cytotoxic reactions; Type 3 are mediated by immune complexes; and Type 4 represent a delayed reaction mediated by T cells (Jutel, Agache et al 2023).

Today, the definition has been expanded and the following classifications are used: Types 1–3 are antibody-mediated reactions; Type 4 are cell-mediated reactions; Types 5 and 6 are tissue-driven mechanisms; and Type 7 represent direct response to chemicals.

Type 1 reactions are IgE-mediated responses that commonly occurs in patients with asthma, allergic rhinitis, urticaria or food allergy. Classic allergens include pollens, animal dander, certain foods (e.g., shellfish), and latex. *Type II* reactions are antibody-mediated cellular cytotoxicity reactions. They are involved in several autoimmune diseases, for example myasthenia gravis and transfusion reactions involving mismatched blood types. *Type III* reactions are immune complex-mediated. They involve the binding of IgG and IgM to soluble antigens, leading to the formation of antigen-antibody complexes, and they are involved in various autoimmune diseases such as systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA). *Type IV* reactions are cell-mediated and are divided into three different types. A *Type IVa* response is considered to be mediated mainly by Th1 cells; examples include allergic contact dermatitis and celiac disease. *Type IVb* reactions are mediated by Th2 cells and include CRS, allergic rhinitis, and eosinophilic oesophagitis. *Type IVc* reactions are driven by neutrophilic inflammation and occur in neutrophilic asthma and atopic dermatitis. A *Type V* reaction is an epithelial barrier defect. Here, it seems as if the inflammation is caused by a defect in the barrier function of, for example, the skin or the mucosa, rather than a dysfunctional immune system. Examples include inflammatory diseases such as CRS, asthma and eosinophilic oesophagitis. A *Type VI* reaction involves metabolic-induced immune dysregulation. Clinical examples include obesity and asthma. A *Type VII* reaction entails direct cellular and inflammatory responses to chemical substances. These reactions can occur in patients with, for example, asthma and acute urticaria (Jutel, Agache et al 2023).

The prevailing hypothesis regarding the pathophysiology of CRS is that multiple inflammatory pathways operating at different sites in the same individual contribute to and cause the broad disease spectrum of CRS. At the root of these inflammatory pathways is damage to the mucosal barrier, which involves both host mechanisms (such as genetic and epigenetic variation of the mucosal immune system) and exogenous factors (such as cigarette smoke, fungi, viruses, bacteria, allergens, airborne pollutants and occupational exposure) (Fokkens et al 2020). '*These host and environmental factors interact over time to trigger one or more mechanistic pathways (endotypes) of chronic tissue inflammation that lead to the clinical presentation (phenotype)*' (Fokkens et al 2020).

1.1.3 EPIDEMIOLOGY

CRS is a common disease, the prevalence of which has been estimated in a large, multi-centre study as around 11% in Europe (Hastan et al 2011). The prevalence rates of CRS around the world are roughly similar, with 9% in Telemark, Norway (**Paper I**), around 6% in Brazil (Pilan et al 2012), 12% in the US (Hirsch et al 2017), and 8% in China (Shi et al 2015).

Some studies have shown that CRS as a single entity of disease is slightly more common in women than in men (Hastan et al 2011, Chen et al 2003). However, CRS may be further divided into chronic rhinosinusitis with nasal polyps (CRSwNP) and chronic rhinosinusitis without nasal polyps (CRSsNP). CRSsNP is diagnosed more frequently in women, whereas CRSwNP tends to be seen more commonly in men (Busaba et al 2008, Behnke et al 2024, Johansson et al 2003, Won et al 2018). Some studies show that CRSsNP is more common than CRSwNP in the general population (Fokkens et al 2020, Tan et al 2013, Won et al 2018). In general, the prevalence of CRS increases with age and tends to have peak incidence in midlife (Chen et al 2003, Hirsch et al 2017).

CRS can also be categorised as primary and secondary CRS, and localised (unilateral) and diffuse (bilateral) CRS. Furthermore, the terms *eosinophilic CRS* and *non-eosinophilic CRS* are increasingly used. The different phenotypes of CRS also include allergic fungal rhinosinusitis and central compartment allergic disease (Fokkens et al 2020).

There are several predisposing factors for CRS. CRS is firmly associated with *asthma* (Jarvis et al 2012), and in a study from Georgia, USA, more than 20% of patients with CRS were found to also suffer from asthma, as compared with 5% of the general adult population (Seybt et al 2007). The prevalence of asthma in Norway is somewhat higher at around 12% (Abrahamsen et al 2017), while in Sweden it is in the range of 9%–11% (Molarius & Hasselgren 2023, Borna et al 2019). CRSwNP appears to be associated with adult-onset asthma (i.e., onset after 18 years of age), whereas CRSsNP is more linked to childhood-onset asthma (onset before 18 years of age) (Won et al 2018). *Allergy* is a risk factor for CRS, although it is more prevalent in some forms of CRS, for example central compartment allergic disease (Marcus et al 2020). *Smoking* is a known risk factor for CRS (Hastan et al 2011), causing increased inflammation and mucosal changes, and may also alter the sinonasal microbiome and increase the risk of biofilm formation. Even passive smoking seems to increase the risk of CRS (Christensen et al 2018). *Obstructive sleep*

apnea is common in patients with CRS and there is an overlap between these two diseases (Cha et al 2024, Ylitalo-Heikkilä et al 2018). CRS has also previously been associated with *obesity* (Nam et al 2021), and studies have shown an association between low serum *vitamin D* levels and chronic rhinosinusitis (Li et al 2021, Zand et al 2020).

1.1.4 CRS AND OCCUPATION

Since CRS is a chronic disease that affects both women and men, particularly during their working years, there is an urgent need to identify the contributing factors. Known risk factors include the above-mentioned asthma (Jarvis et al 2012), allergy (Marcus et al 2020) and smoking (Hastan et al 2011), and in recent years the working environment has been identified as a possible contributor to CRS.

Working adults in mid-life spend a large part of the day at their workplace. As such, occupational exposure is a highly relevant risk factor to investigate in relation to CRS. In a study carried out by Weakley and co-workers in 2016, 9,848 male firefighters from the Fire Department of New York City who were present at the disaster site shortly after the terrorist attacks on the World Trade Centers on September 11th 2001 were examined. The risk of new-onset CRS for these subjects was increased by exposure to the World Trade Center disaster site. Subjects with high-level exposure (present at the disaster site on the morning of September 11th) had almost twice as high a risk of acquiring CRS as those with low-level exposure (present at the disaster site between September 13th and 24th) (Weakley et al 2016).

Occupational rhinitis (OcR) is defined as: *'An inflammatory disease of the nose, which is characterized by intermittent or persistent symptoms (ie, nasal congestion, rhinorrhea, sneezing and itching) and/or variable nasal airflow limitation due to causes and conditions attributable to a particular work environment and not to stimuli encountered outside the workplace'* (Vandenplas et al 2020). OcR may be divided into two categories; **sensitiser-induced OcR** (which is caused by immunological sensitisation to a certain agent) and **irritant-induced OcR** (caused by occupational exposure to high levels of irritants). Sensitiser-induced OcR can be caused by high molecular weight (HMW) agents such as proteins of animal or plant origin which act through an IgE-mediated mechanism. But sensitiser-induced OcR can also be caused by low molecular weight (LMW) agents such as reactive chemicals, metals and wood dusts. Irritants causing irritant-induced OcR include compounds such as chlorine, sulfur dioxide, ozone, and thermal degradation

products. The mechanisms underlying the reactions induced by irritants are not completely understood but are thought to involve both the innate immune system and the nasal sensory nervous system, resulting in epithelial injury (Vandenplas et al 2020).

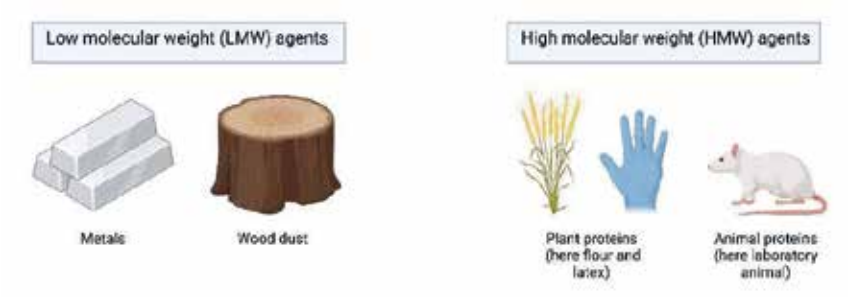

Figure 2. Examples of sources of low molecular weight (LMW) agents and high molecular weight (HMW) agents. Figure adapted from Vandenplas O et al (2020) Occupational rhinitis. J Allergy Clin Immunol Pract 8: 3311-3321.

High-molecular-weight (HMW) agents are biological substances, for example proteins from laboratory animals or seafood, as well as plant proteins such as flour and latex. These agents cause an IgE-mediated immune sensitisation of the individual. **Low-molecular-weight** (LMW) agents include reactive chemicals, such as isocyanates and persulfates, and metals, such as platinum salts. For most LMW agents the pathophysiological mechanism is less well understood. However, some LMW agents (platinum salts) have been shown to induce an IgE-mediated immune response. Moreover, LMW agents with sensitising potential may function as irritants when present in high concentrations (Vandenplas et al 2020).

The concepts of sensitisers, irritants, HMW agents and LMW agents are important to understand due to their different pathophysiological mechanisms. However, in reality, a single occupational exposure may encompass several different substances, including HMW and LMW agents acting as both sensitisers and irritants. For example, wood dust is a composite of many substances, such as cellulose, terpenes and resin acids, including both HMW and LMW agents. Monoterpenes and resin acids are thought to act through both sensitisation and irritant pathways (Baatjies et al 2023, Straumfors et al 2018).

Workplace exposure can be assessed using different techniques. Self-reporting of occupational exposures using questionnaires is one option. It is also possible to use a job-exposure matrix (JEM). A JEM is a table in which job titles are linked to categories of exposure. Using the subject's job title, the JEM is able to provide exposure estimates for that individual. Direct on-site measurement of workplace exposure is another option (Descatha et al 2022).

Since the concept of the global airways was proposed in the mid-1980s, the nose, the epi-, oro- and hypopharynx, the trachea, and the lungs have been considered parts of the same entity. Diseases often occur simultaneously, e.g., asthma and CRS, and the current hypothesis is that the same type of pathological mechanism causes disease in different parts of the global airways at the same time (Bachert et al 2023, Passalacqua et al 2001). Much of what is known about the effects of occupational exposures on the respiratory mucosa was first uncovered in relation to the lower airways, and in later years this knowledge was expanded to include the upper airways.

Previous studies have revealed connections between paper dust and non-infectious rhinitis (Hellgren et al 2001, Hellgren et al 2002). Several other occupational exposures have also been linked to rhinitis, for example cleaning agents (Folletti et al 2014), wood dust (Wilhelmsson & Drettner 1984, Holmström & Wilhelmsson 1988, Holmström et al 1995), working with laboratory animals (Gross 1980, Slovak & Hill 1981), occupational exposure to flour, buckwheat and psyllium (Jungewelter et al 2020, Jungewelter et al 2021), and chemicals such as dimethylbenzylamine (Lindahl et al 2004).

Since the body of evidence regarding the pathophysiology and clinical presentation of patients with CRS based on computed tomography imaging and immunological characterisation has expanded in recent years, it is timely and warranted to explore further occupational exposures in relation to CRS, in order to understand more clearly the disease and its epidemiology.

1.1.5 WHY THIS THESIS?

CRS is ultimately the most serious form of rhinitis and presently lacks a curative treatment. Prior to the work of this thesis, there was a scarcity of studies regarding CRS and occupational exposure (Sundaresan et al 2015), despite the severity of the disease and the fact that it mainly affects people of working age. The previous lack of a unanimous definition for CRS in epidemiological studies has hindered progress and made studying CRS and

occupational exposure difficult. The publication of EPOS in 2012 (Fokkens et al 2012), with a precise and clear definition of CRS, has resolved this issue.

We were fortunate to be invited to take part in the Telemark Population study in the south of Norway. It is a large, prospective study in which residents in the age range of 16–50 years were invited to complete a questionnaire regarding their occupation and their experiences of airway symptoms, such as CRS and asthma, in 2013 and 2018. The questions were phrased in accordance with the EPOS definition of CRS used in epidemiological studies.

To bridge the knowledge gap in relation to CRS and occupational exposure, we conducted one cross-sectional and one prospective study on CRS and occupational exposure (**Papers I** and **II**). Studies looking at the connections between obesity and new-onset CRS and between recent sick leave and CRS were also carried out (**Papers III** and **IV**).

2 AIMS

The *overall aim* of this thesis is to contribute to increasing knowledge of CRS, in particular regarding occupational exposures, connections to obesity and associations with sick leave.

Specific aims:

Paper I To identify occupational exposures associated with an increased odds of CRS in a cross-sectional study. To determine the prevalence of CRS in the Telemark population.

Paper 2 To identify occupational exposures associated with an increased odds of new-onset CRS in a prospective study. To determine the cumulative 5-year incidence of new-onset CRS during the period of 2013–2018 in the Telemark population.

Paper 3 To evaluate the linkage between obesity and new-onset CRS in the Telemark population.

Paper 4 To determine whether subjects with CRS have a higher frequency of sick leave in the past year than subjects without CRS, and if there is a difference between women with CRS and men with CRS. To determine whether the frequency of sick leave is higher in subjects with CRS in some occupational groups.

3 SUBJECTS AND METHODS

Norway is a country in northern Europe with a population of approximately 5.5 million (Statistisk sentralbyrå, Norway, www.ssb.no Accessed: 2024-12-09). Telemark Fylke (Telemark County) is a region in the south of Norway with approximately 177,000 inhabitants. The county has many industries and has high rates of export of, for example, chemical products, raw materials, fuels and machines (www.telemarkfylke.no Accessed: 2024-09-10). Thus, it is suitable for studies of occupational exposures. The age and gender distributions in Telemark in 2013 were similar to those in the rest of Norway. The prevalence rates of daily and occasional smokers per year were slightly higher in Telemark (at 16% and 10%, respectively) than in Norway as a whole (12% and 9%) during the period of 2013–2017. In general, the use of healthcare services in Telemark is somewhat higher than in the rest of Norway, and the residents of Telemark County have slightly lower socio-economic status (Statistisk sentralbyrå, Norway, www.ssb.no Accessed: 2024-09-19).

The Telemark population study began in 2013 when 50,000 randomly selected individuals residing in Telemark were invited to participate. The subjects were in the age range of 16–50 years and possessed a Norwegian social security number. Subjects received a written invitation to participate and received a questionnaire by mail, followed by two written reminders around 1 month apart. The questionnaires were in the Norwegian language only. Subjects who decided to participate received written information and informed consent (in writing) was obtained.

Of the 50,000 subjects who were invited to participate in the study, 48,142 were eligible. In contrast, 1,858 could not be traced or were excluded, 1,793 had moved, 4 were deceased, 13 could not answer due to illness/functional impairment, 23 could not answer due to language problems, and 25 did not answer for other reasons. In total, 16,099 subjects answered the questionnaire in 2013, resulting in a response rate of 33%.

The study was repeated in 2018, subjects received a follow-up questionnaire where the questions regarding the studied variables were the same. The participants were the same 16,099 subjects who answered in 2013, as well as 23,840 new participants who fulfilled the inclusion criteria (see above). In total, 39,939 participants were invited to join the 2018 study. Overall, 1,834 persons could not be traced or were excluded, 36 did not wish to participate, 4 could not answer due to language difficulties, 1 had died, 9 could not answer

due to illness/functional impairment, 2 had moved abroad, and 1,782 questionnaires were returned due to unknown address. In the end, 14,509 participated in the 2018 study, and the response rate was 36%.

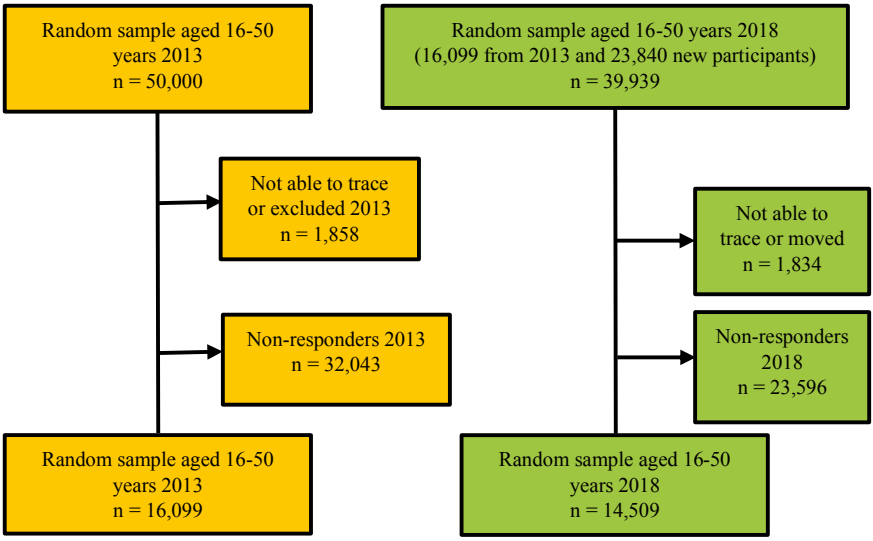

Figure 3. Flow chart of the study populations in 2013 and 2018. Figure adapted from Paper IV.

Financial incentives to participate in the 2013 study were offered: two participants were randomly drawn and offered either an iPad or 5,000 Norwegian kroner in the form of a travel gift card. No financial incentive was provided in the 2018 study.

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).

3.1 STATISTICS

Paper I

Paper I comprises a cross-sectional study of the Telemark population in 2013, with a study population of 16,099 participants. Subjects who reported that they had never been employed were removed from the analysis (n=1,193). Comparisons between the two groups (CRS and non-CRS) were made using Fisher's exact and Chi^2-tests. Missing answers were regarded as not having been exposed or not having the symptom. A sensitivity analysis was performed in which all missing answers were excluded, and it yielded similar results. Odds ratios (ORs) with 95% confidence intervals (95% CI) were calculated using logistic regression to estimate the effects of smoking, asthma, atopy and occupational exposure on the probability of CRS. Calculations were made both without adjustment and with adjustments for confounders such as age, gender, smoking, asthma, atopy and the other types of occupational exposures. P-values <0.05 were considered statistically significant.

Paper II

Paper II describes a prospective study conducted between 2013 and 2018 with 6,857 study participants. Since the objective was to study new-onset CRS between 2013 and 2018, all subjects who reported CRS at baseline in 2013 were excluded from the study (n=631). As in **Paper I**, subjects who reported that they had never been employed were removed from the analysis (n=464). The CRS group and the group without CRS were compared regarding age, gender, smoking, asthma and atopy using Fisher's exact test and the Chi^2 test. The 5-year cumulative incidence of new-onset CRS in the Telemark population between 2013 and 2018 was calculated. For the main analysis, the ORs and 95% CI were calculated using univariable and multivariable logistic regression analysis with CRS as the dependent variable and the different occupational exposures as independent variables. When the influence of a single exposure was investigated, exposures and baseline characteristics that had a p-value <0.05 in the univariate analyses were used in a forward multiple logistic regression model with CRS as the dependent variable.

A multiple logistic regression analysis was conducted using six main groups of exposures (metals/gases, damp/mould, exercise/cold, cleaning/cooking, organic dust, hair products/animals) as independent variables and CRS as the dependent variable. P-values <0.05 were considered statistically significant.

Paper III

Paper III provides the results of a prospective study that was carried out between 2013 and 2018 with a study population of 5,769. Since the objective was to study new-onset CRS in relation to BMI, all of the subjects who reported CRS at baseline in 2013 were excluded from the analysis (n=622). Likewise, subjects who had not responded to the CRS-questions in either 2013 or 2018 were omitted (n=334). Overall, 1,227 subjects had missing answers with respect to either height or weight so the BMI could not be calculated; they were therefore also removed from the analysis. A total of 92 subjects had missing data on asthma and smoking and they were removed from the regression analyses. ORs for new-onset CRS in relation to BMI were calculated using logistic regression analysis and adjusting for the confounders gender, smoking, asthma and age. Restricted cubic splines were used to model the continuous variables age and BMI (when analysed as a continuous variable). P-values <0.05 were considered statistically significant.

Paper IV

This study was conducted as two cross-sectional studies, in 2013 (n=15,484) and 2018 (n=13,966), respectively. Subjects who lacked responses to the CRS-related questions were excluded (n=476 in 2013, n=318 in 2018), as were those subjects with missing answers regarding sick leave (n=139 in 2013, n=225 in 2018). Multivariable logistic regression models were used to investigate how CRS/CRS-symptoms were related to differences in the frequency of sick leave in the past 12 months. The models were adjusted for age, sex, asthma and smoking. Age was included as a continuous variable using cubic restricted splines. The Cochran-Armitage trend test was used to investigate trends in the association between number of sinus symptoms and sick leave. P-values <0.05 were considered statistically significant.

4 RESULTS

4.1 PAPER I

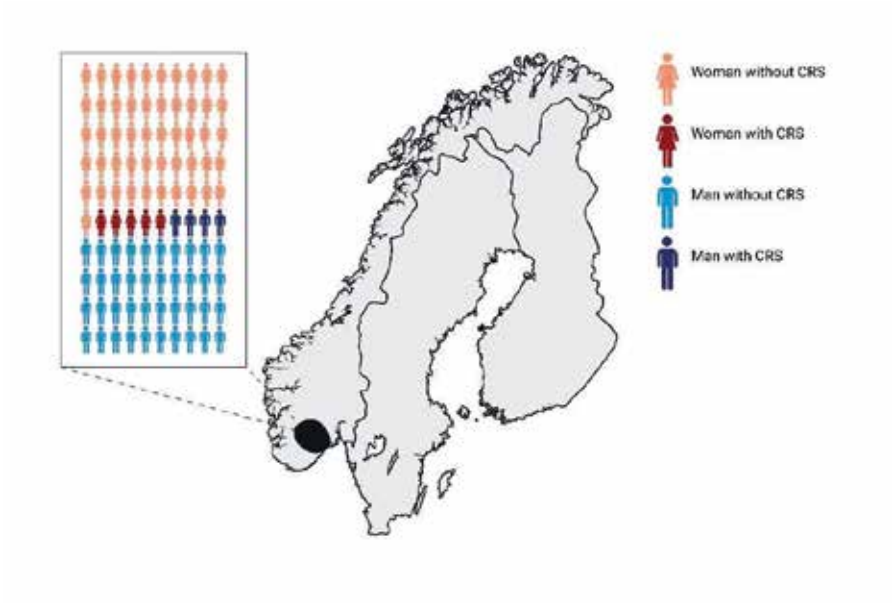

Figure 4. Prevalence of CRS (9%) in the Telemark population in 2013.

The prevalence of CRS was found to be 9%. CRS was slightly more common in women than in men (55% vs 45%). Asthma was more common in the CRS group than in subjects without CRS (28% vs 10%). Atopy was more common in the CRS group than in the non-CRS group (58% vs 28%).

Figure 5. Self-reported occupational exposures with increased odds of having CRS in the Telemark population in 2013.

The odds of having CRS was significantly increased in subjects who had previously been exposed to paper dust (OR 1.3, 95% CI 1.1–1.5), metal dust (OR 1.3, 95% CI 1.1–1.6), cleaning agents (OR 1.2, 95% CI 1.0–1.3), animals (OR 1.2, 95% CI 1.0–1.5), moisture/mould/mildew (OR 1.3, 95% CI 1.1–1.5) or engaged in physically strenuous work (OR 1.4, 95% CI 1.2–1.7), as compared to subjects without that particular workplace exposure. When stratified for sex, only paper dust (OR 1.4, 95% CI 1.1–1.7), moisture/mould/mildew (OR 1.3, 95% CI 1.0–1.5), and physically strenuous work (OR 1.4, 95 % CI 1.2–1.7) imparted a significantly increased OR for CRS in women. For men, metal dust (OR 1.5, 95% CI 1.2–1.9), moisture/mould/mildew (OR 1.3, 95% CI 1.1–1.6), and physically strenuous work (OR 1.4, 95% CI 1.1–1.8) remained significantly linked to an increased OR for CRS.

The most common CRS symptom was a stuffed nose, followed by, in declining order, facial pain/pressure, discoloured secretions and a reduced sense of smell.

4.2 PAPER II

Asthma was more common in the CRS group than in the non-CRS group (17% vs 9%). Atopy was found to be more common in the CRS group than in the non-CRS group (45% vs 27%).

The cumulative 5-year incidence of new-onset CRS between 2013 and 2018 was 5.5% (95% CI 4.9–6.0).

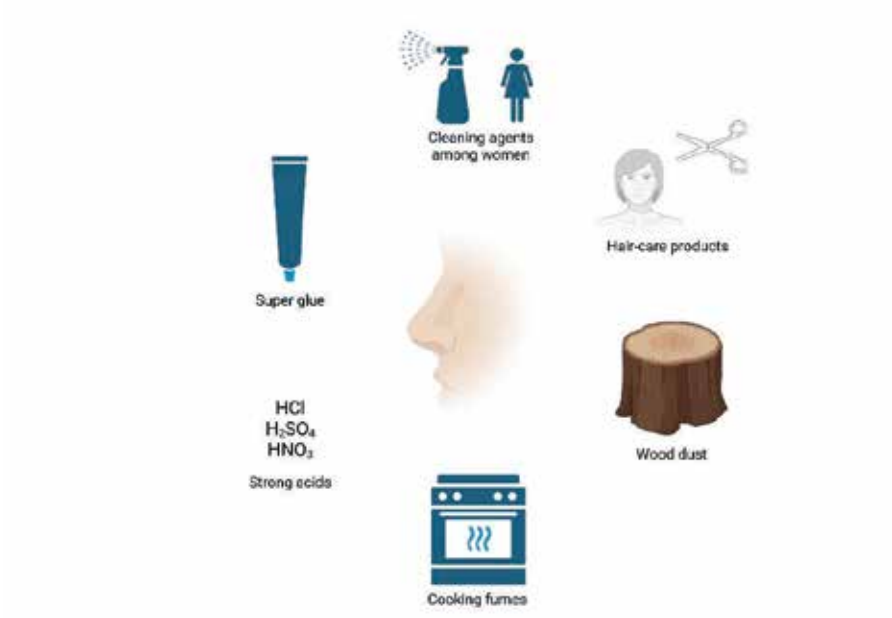

Figure 6. Self-reported occupational exposures with increased odds of new-onset CRS in the Telemark population 2013-2018.

An increased odds of acquiring CRS during the observation period was independently and significantly associated with occupational exposure to hair-care products (OR 1.7, 95% CI 1.1–2.6), super glue (OR 1.4, 95% CI 1.1–2.0), strong acids (OR 1.4 (95% CI 1.1–1.9), cooking fumes (OR 1.4, 95% CI 1.1–1.8) and wood dust (OR 1.4, 95% CI 1.0–1.8).

With stratification for sex, the following occupational exposures remained significantly associated with increased odds for CRS in women: animals (OR 1.6, 95% CI 1.1–2.5); cleaning agents (OR 1.6, 95% CI 1.2–2.1); and strong acids (OR 1.6, 95% CI 1.1–2.3). For men, the following occupational

exposures were significantly associated with increased odds for CRS: metal dust (OR 1.6, 95% CI 1.1–2.2); and stone dust (OR 1.5, 95% CI 1.1–2.1).

Six main groups of occupational exposures (metals/gases, damp/mould, exercise/cold, cleaning/cooking, organic dust, and hair products/animals) were analysed in a multiple logistic regression analysis. All of the groups, except for exercise/cold, were significantly correlated with increased odds of acquiring CRS during the observation period, as compared with unexposed subjects. When stratifying for gender, only cleaning/cooking and hair products/animals remained significantly linked to CRS in women. For men, only metal/gases remained significantly linked to CRS.

4.3 PAPER III

Overall, 1% of the study population was under-weight (BMI<18.5), 50% had normal weight (BMI 18.5 to <25), 35% were over-weight (BMI 25.0 to <30), and 14% were obese (BMI≥30).

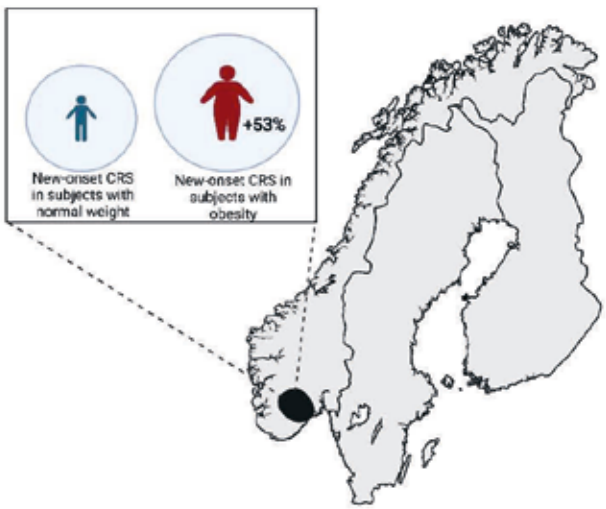

Figure 7. Odds of new-onset CRS in 2018 based on BMI in 2013 when comparing subjects with normal weight (18.5≤BMI<25) and obesity (BMI≥30).

The odds of new-onset CRS in 2018 based on BMI in 2013 was 53% higher (OR 1.53, 95% CI 1.1–2.1) for subjects who were obese than for subjects of normal weight.

A sensitivity analysis was conducted amongst subjects with missing answers regarding height or weight, for whom the BMI could not be calculated (n=1,227). This group of subjects had similar distributions in terms of gender, asthma, smoking and new-onset CRS as the study group.

4.4 PAPER IV

As in the above-mentioned papers, asthma was found to be more common in the CRS group compared to the non-CRS group in both 2013 (29% vs 10%) and 2018 (28% vs 11%).

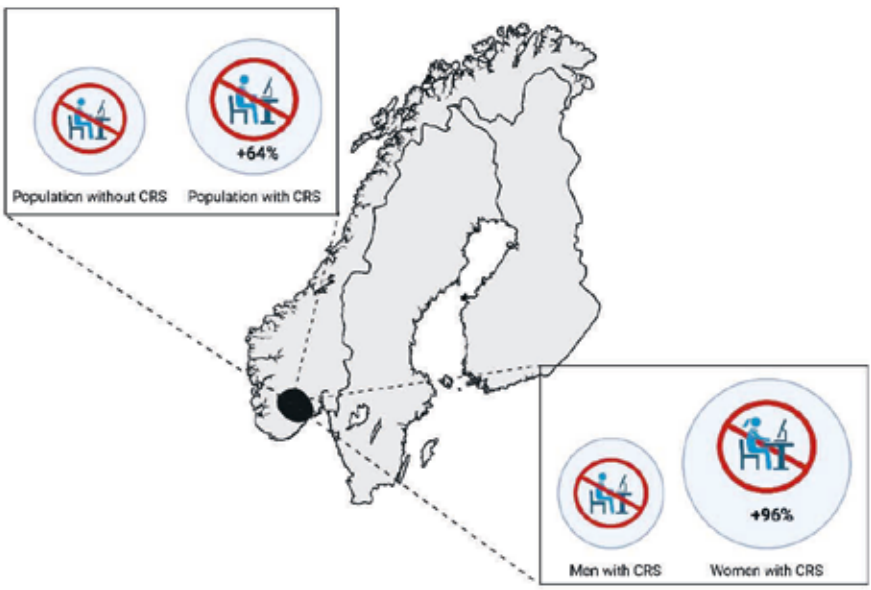

Figure 8. Odds of self-reported sick leave in the last year in subjects with CRS compared to subjects without CRS in the Telemark population in 2013 (LEFT). Odds of self-reported sick leave in the last year in women with CRS compared to men with CRS in 2013 (RIGHT).

Self-reported sick leave in the past year was reported more frequently in the CRS group than in the non-CRS group in both 2013 (OR 1.64, 95% CI 1.5–

1.9) and 2018 (OR 1.60, 95% CI 1.4–1.8), when adjusted for age, sex, asthma and smoking.

Comparing the group with CRS and asthma to the group with CRS only, there was no significant difference in the frequency of self-reported sick leave in 2013, both without adjustment (OR 1.2, 95% CI 0.9–1.5) and with adjustments for age, sex and smoking (OR 1.1, 95% CI 0.9–1.5). The results were similar in 2018, both without adjustment (OR 1.2, 95% CI 0.9–1.6) and with adjustments for age, sex and smoking (OR 1.1, 95% CI 0.9–1.5).

Also compared were women with CRS and men with CRS. In 2013, women with CRS reported sick leave during the past year almost twice as frequently as men with CRS (OR 1.96, 95% CI 1.6–2.5), when adjustments were made for age, smoking and asthma. In 2018, women with CRS still reported sick leave in the past year more frequently than men with CRS, although the difference was smaller (OR 1.28, 95% CI 1.0–1.6), when adjustments were made for age, smoking and asthma.

5 DISCUSSION

Chronic rhinosinusitis is a common disease, having a prevalence of 9% in Norway **(Paper I**) and 11% in Europe as a whole (Hastan et al 2011). As it mainly affects working subjects in mid-life (Chen et al 2003, Hirsch et al 2017), it is important to consider how the workplace affects CRS and what synergistic effects occur.

To the best of our knowledge, there are no previous, large, population-based studies regarding CRS using the current epidemiological definition and information on workplace exposure. Before the consensus in 2012 on how to define CRS in epidemiological studies (Fokkens et al 2012), comparing studies was difficult because there existed many different definitions of chronic rhinosinusitis, and not all CRS diagnoses are made by otorhinolaryngologists (Novis et al 2016). Since the EPOS definition emerged in 2012, it has become remarkably easier to compare studies. Our studies are unique in that we examine how CRS in the general population may be linked to different occupational exposures, not only patients with CRS who are selected for surgery and already within the health-care system.

5.1 CRS AND OCCUPATIONAL EXPOSURES

Our results suggest that previous, self-reported occupational exposures to paper dust, metal dust, cleaning agents, animals, moisture/mould/mildew and physically strenuous work are a risk factor for CRS (**Paper I**). Furthermore, occupational exposures to hair-care products, cleaning agents among women, super glue, strong acids, cooking fumes and wood dust are found to be associated with an increased risk of new-onset CRS during a 5-year period (**Paper II**).

The occupational exposures associated with CRS differ between the two studies. This could be due to several reasons. **Paper I** is a cross-sectional study that investigates the prevalence of CRS in association with occupational exposures (i.e., what you have been exposed to when you have CRS). **Paper II** is a prospective study that explores new-onset CRS during a 5-year period in relation to occupational exposures (i.e., what you have been exposed to when you develop CRS). The individuals investigated are not the same in the two studies and the sample sizes are different. Some occupational exposures may require a longer period of exposure in order to induce CRS. There could

also be individual differences in sensitivity to a specific exposure, and this sensitivity may vary with age.

Exposure to **paper dust** has previously been linked to rhinitis (Hellgren et al 2001, Hellgren et al 2002, Theander & Bende 1989). Paper dust exposure may lead to increased nasal congestion and nasal crusts (Hellgren et al 2001). Nasal blockage, in turn, may promote a congested ostiomeatal complex and decreased drainage from the sinuses, which may facilitate the development of CRS.

Metal dust has been associated with a decline in lung function and possible airway inflammation (Rehfisch et al 2012, Assenhöj et al 2023). One hypothesis is that metal dust causes inflammation of the nasal mucosa and elicits a response that, together with other co-factors, ultimately leads to chronic inflammation and CRS.

In our studies, we show that **cleaning agents** are risk factors for CRS cross-sectionally, as well as for new-onset CRS during a 5-year period (**Papers I** and **II**). The subjects in the Telemark population who reported exposure to cleaning agents worked primarily in health-care services and office/shop/domestic settings, and to a lesser extent with industrial cleaning. Although we are not able to discern the specific cleaning agents used, this further strengthens the connection between cleaning agents and their harmful effects on the airways, which have previously been shown in relation to rhinitis (Hellgren et al 2002) and asthma (Siracusa et al 2013, Folletti et al 2014). Cleaning products such as bleach and ammonia can worsen already existing asthma and can also induce new-onset asthma. While the exact pathophysiological mechanisms are not known, irritants appear to be the most common causative agents, although sensitisation also occurs. It is more common for women to work in cleaning occupations and women also seem to be more pre-disposed to occupational asthma caused by cleaning. Cleaning product-linked asthma is commonly seen in health-care occupations (Folletti et al 2017). This finding is similar to our results in **Papers I** and **II**, in that the subjects who reported exposure to cleaning products often worked in health-care services. A study conducted by Rava and colleagues has illustrated how occupational exposure to low-molecular-weight agents and irritants (such as some cleaning products) may interact with genes linked to a pre-disposition to adult-onset asthma (Rava et al 2017, Folletti et al 2017). Another recent study by Tjalvin and co-workers has shown that children whose mothers were occupationally exposed to cleaning agents pre-conception, around the time of conception, and during

pregnancy had an increased odds for childhood asthma and wheezing (Tjalvin et al 2022). This further illustrates and underlines the importance of cleaning agents as occupational hazards that are relevant to airway inflammation. The results of our studies contribute to this field of research, as we show that occupational exposure to cleaning agents plays a part in diseases of the upper airways, such as CRS.

Allergy to laboratory **animals** is a known occupational risk factor. In laboratory animal allergy, exposure to animal proteins elicits an IgE-mediated immune response, resulting in nasal congestion and nasal discharge amongst other symptoms (Bush 2001). Daily workplace exposure to an animal may, therefore, contribute to the development of CRS. Allergic rhinitis is also more frequently seen in Western Europe (Bauchau & Durham 2004), and allergy is a known risk factor for CRS (Marcus et al 2020). Therefore, several risk factors for CRS are present in the Telemark population, and our results show that a significant percentage experience CRS symptoms (**Papers I** and **II**).

Allergy to **mould** is believed to activate mechanisms involving both the innate and adaptive immune systems (Williams et al 2016). Moreover, occupational exposure to mould is a known risk factor for both asthma and allergic rhinitis (Caillaud et al 2018). Our results show that occupational exposure to moisture/mould/mildew is, likewise, a risk factor for CRS.

In our study, occupational exposure to **hair-care products** was significantly correlated with an increased risk of new-onset CRS. Working with hair-care products is known to have a negative effect on the lower airways, particularly in relation to the development of asthma (Moscato & Galdi 2006, Torén & Hörte 1997). Hair-care products are also known to cause allergic rhinitis (Schwartz et al 1990, Moscato & Galdi 2006). Therefore, it is not surprising that hair-care products can also facilitate the development of CRS. Persulphate salts are the most common causative agents of occupational asthma and occupational rhinitis among hairdressers (Moscato & Galdi 2006, Pignatti et al 2013). In our study, we lack information regarding which specific substances the subjects were exposed to, as we have only established that they worked with hair-care products.

Allergy is a known risk factor for CRS (Marcus et al 2020). This fact is also relevant with regard to CRS and occupational exposure to hair-care products. When working with hair-care products the subject is exposed to potentially disease-causing irritants and sensitisers in two ways: via the air and,

consequently, the respiratory mucosa; and via the skin. Hair-care products may cause disease through a pathway involving IgE when the nasal mucosa is exposed to persulphate salts (Moscato & Galdi 2006). This response may not only occur in the respiratory mucosa but may also occur in the skin, in the form of contact urticaria, which has been described in subjects who handle hair dyes (Haltia et al 2021).

There have also been cases of occupational rhinitis and contact urticaria in subjects who are handling pork meat (Jungewelter et al 2019), suggesting the possibility of dual sensitisation of the airways and skin. Hair-care products contain diverse chemicals, and hypothetically these products could initiate another type of immune response via the skin. Contact dermatitis, for example, is thought to be mediated by complex signalling pathways that involve T cells (Brar 2021). Could these two different exposure pathways (nose and skin), as well as the different pathophysiological mechanisms (Type 1: urticaria, and Type 4: contact dermatitis, hypersensitivity reactions), have a synergistic effect in patients with occupational exposure to hair-care products who are developing CRS? This idea is interesting and warrants further research.

Super glue, with a base of cyanoacrylate glue, is known to cause rhinitis and asthma (Kopp et al 1985, Lindström et al 2013). Just as in the case of hair-care products, subjects who are working with super glue are not only exposed to the vapours and fumes, but are also in contact with the substance via the skin. Subjects who work with super glue are repeatedly exposed and the nose, in particular, acts as a first-line barrier.

Cooking fumes are known to cause problems in the lower airways (Svedahl et al 2020, Fell et al 2016, Neghab et al 2017). Harmful by-products may be generated when food is cooked at high temperatures. These by-products include aldehydes, such as acrolein and formaldehyde, which are known to cause airway irritation when inhaled (Svendsen et al 2003). It is reasonable to assume that these fumes also have similar, detrimental effects on the sino-nasal mucosa.

Wood dust is a known risk factor for occupational asthma, and subjects who are working in the construction, furniture building, carpentry, joinery, sawmilling and forestry sectors are among those with the highest levels of exposure (Baatjies et al 2023). While the pathophysiological mechanisms for wood dust's detrimental effects on the airways are not fully understood, cytological examinations of nasal smears obtained from woodworkers show

increased numbers of inflammatory cells, indicating chronic inflammation (Lovato et al 2016). Large amounts of both small and large inhalable products were released into the environment in connection with the terrorist attacks on the World Trade Centers in New York City in 2001, and it is estimated that around 70% of the buildings were pulverised. The risk of new-onset CRS appeared to increase in the years following the attack for employees of the Fire Department of the City of New York (FDNY) who were exposed to the debris. A correlation was found between the level of exposure and the incidence of disease: CRS was more common in individuals who experienced a higher exposure (Weakley et al 2016).

In **Paper I**, we show an association between **physically strenuous work** and increased odds for CRS. This finding should be interpreted with caution because exercise is known to have a positive effect on asthma (Freeman et al 2020), as well as on general health (Warburton & Bredin 2017, Pucci et al 2012). The prevalence of asthma is high among elite athletes, indicating a somewhat complex relationship (Pedersen et al 2011). One hypothesis is that the subjects in our study who reported physically strenuous work have jobs such as asphalt paver and/or brick-laying or other dusty jobs. Thus, they may be simultaneously exposed to additional occupational risks, such as hazardous gases and/or dust, perhaps accounting for their increased odds of CRS. Self-reported physically strenuous work may also indicate that a subject has worked in cold storage rooms, repeatedly moving from cold to warm areas throughout the workday. Airway reactions to cold air exposure can be categorised as: 1) cold air-provoked short-term responses, which include rhinorrhea, congestion and sneezing; 2) cold air-provoked long-term responses, which cause damage to the airway epithelium and changes to the airway wall structure and function; 3) cold airway-provoked reflex lower airway responses, which involve slight bronchoconstriction. The cold air is unlikely to be a causal factor for disease, although it may well trigger symptoms and act in concert with another factor to cause disease. It is also likely that individual susceptibility has a part in the impact of occupational cold exposure (Koskela 2007).

Understanding, and reflecting upon, the pathophysiological mechanisms behind the different occupational exposures generates a more comprehensive picture of CRS. As mentioned above, CRS is a multifactorial disease with many different phenotypes (Fokkens et al 2020). We hypothesise that CRS has a phenotype that is predominantly connected to allergy, and a hypersensitivity reaction of type IVb (a response with Th2-cells and eosinophils). Let us imagine that an individual with this genetic constitution works with laboratory

animals and, as a consequence, is exposed to animal dander, which we know consists of HMW agents that induce mainly an IgE-mediated immune response. Is it possible that the combination of these two factors synergistically affects the individual, contributing to the development and progression of CRS? This is an interesting question to consider from the perspective of Rothman's sufficient-component cause model (Rothman 1995). This model postulates that the occurrence of disease is dependent upon many, different, smaller potential causes, and that these combine in a certain way to cause disease in an individual. Perhaps occupational exposure is one such causal component which could cause disease in some individuals with CRS. Occupational exposure may also be a causal component causing exacerbations of nasal symptoms and/or CRS. The difficulty lies in identifying these subjects, and adequately preventing or decreasing exposure before disease occurs.

5.2 CRS AND OBESITY

Obesity is currently one of the worlds' most challenging health problems (Afshin et al 2017, Ng et al 2014). Numerous diseases are associated with obesity, such as hypertension, coronary heart disease, stroke, type 2 diabetes mellitus, and sleep apnea (Jensen et al 2014). Obesity is the result of excessive energy intake (diet) and simultaneous low energy expenditure (physical exercise). However, the etiology of obesity is multi-faceted and complex, with both environmental and genetic contributions amongst others (Wright & Aronne 2012).

CRS has been linked to obesity in several cross-sectional studies (Bhattacharyya 2013, Kim et al 2015, Nam et al 2021), and in **Paper III** we demonstrate that there is a significant effect of obesity on new-onset CRS during a 5-year observational period. A high BMI should, therefore, be considered an independent risk factor for CRS and taken into consideration when encountering patients with CRS in the clinic.

There may be multiple reasons for this connection between obesity and CRS. Obesity seems to cause a low-grade, chronic inflammation (Gregor & Hotamisligil 2011), which in theory could also affect the sino-nasal mucosa. Although the multi-factorial pathogenesis of CRS is not fully understood, there is a general consensus that chronic inflammation plays an important role in the development and persistence of CRS (Fokkens et al 2020). Thus, it is possible that one way in which obesity negatively affects CRS is through a low-grade,

chronic inflammation. Also likely is that individual susceptibilities influence the effect of obesity on CRS.

Obesity is a well-established risk factor for asthma in adults, as well as in children (Pite et al 2020, Xie et al 2024). Subjects with asthma and concomitant obesity exhibit poorer responses to treatment and a higher symptom burden (Klepaker et al 2019, Pite et al 2020). It appears that even other parts of the metabolic syndrome (metabolic conditions such as increased blood pressure, high blood sugar levels, and abnormal cholesterol/triglyceride levels) are also independently related to asthma (Pite et al 2020).

One aspect of the negative effect of obesity on CRS is presumably the effect that obesity has on **sleep apnea**. Obesity is a major risk factor for obstructive sleep apnoea (Jordan et al 2014). Obstructive sleep apnea is known to decrease the health-related quality of life of patients with CRS and other rhinologic diseases, such as allergic rhinitis and non-allergic rhinitis. However, subjects with CRS who do not suffer from obstructive sleep apnea also have a decreased health-related quality of life compared with the general population (Ylitalo-Heikkilä et al 2018). Therefore, it appears that CRS alone, even in the absence of a concomitant obstructive sleep apnea diagnosis, causes poor-quality sleep. This is important as it highlights that the symptoms of CRS, mainly nasal congestion, are in themselves sufficient to cause sub-standard sleep and, consequently, have a significant negative impact on the daily life of sufferers. Obesity accompanied by obstructive sleep apnea may also lower the tolerance level for CRS symptoms in some individuals, in that it may be easier to tolerate a stuffed and runny nose when you are well rested than when you are exhausted from lack of sleep.

5.3 CRS AND SICK LEAVE

CRS is a common disease in the general population, affecting around 11% of Europeans (Hastan et al 2011). It is also a costly disease, entailing substantial direct costs (hospital visits, medicine) and indirect costs (workdays missed, loss of productivity) (Fokkens et al 2020). Studies of the economic impact of CRS in Europe are scarce. Lourijsen et al conducted a cross-sectional study in 2020 and found that the total annual cost of patients with CRSwNP in The Netherlands was €1.9 billion per year (Lourijsen et al 2020). Wahid et al found that patients with CRS in general missed 18.7 days of work per year for both CRS-related reasons and non-CRS-related reasons, as compared to 2.9 workdays per year for subjects without CRS. When accounting for only CRS-

related reasons, the number of missed workdays in a year for subjects with CRS was 7.8 (Wahid et al 2020). Subjects with CRS report a decrease in their health-related quality of life, in similarity to patients with other chronic diseases (Ylitalo-Heikkilä 2018, Gliklich & Metson 1995). Despite this there are, to our knowledge, no population-based studies that have investigated the frequency of sick leave among subjects with CRS in the general population. Therefore, we conducted the study that resulted in **Paper IV** where we analysed the frequency of self-reported sick leave in the last year in subjects who reported CRS in Telemark, Norway.

In our study, CRS seems to have a strong impact on sick leave, although we have not investigated if there is a causal link. This seems especially true for women with CRS, as they were almost twice as likely to take sick leave in the last year compared to men with CRS in 2013. There could be several reasons for this outcome. Males in general appear to have a larger nasal surface area and a larger nasal volume compared with females (Russel & Frank-Ito 2023). Women may also be affected by pregnancy rhinitis (Ellegård 2003). Interestingly, there is also a difference between the sexes with regard to the immune system. The female immune system elicits a stronger response to infections and vaccinations, and women also have higher incidences of certain autoimmune diseases (Moulton 2018). The importance of these phenomena in this context are uncertain, although they illustrate the complexity of studying the differences between women and men with CRS. These factors combined may contribute to the difference in frequency of sick leave observed in our study between women with CRS and men with CRS.

Women with CRS also tend to report a heavier burden of symptoms (compared to men with CRS) when it comes to quality of life (Behnke et al 2024, Adams et al 2023). Some studies have investigated a potential difference in the pathophysiology of CRS between the sexes, proposing for example that sex hormones may be involved in nasal polyposis (Espersen et al 2020). Furthermore, women who are undergoing endoscopic sinus surgery (ESS) may have higher serum levels of IgE and they show evidence of more fungal tissue components in histopathological examinations (Azar et al 2017). However, since the studies are few and are poorly supported in the literature, further studies are needed in this area (Behnke et al 2024). A recent study has shown that 5 years after undergoing ESS women reported more severe symptoms compared to their male counterparts (Adams et al 2023). In summary, the current literature, this thesis included, appears to indicate that there are differences between the sexes regarding CRS. However, it is not currently

known for certain in what respects these differences exist and if, and how, the pathophysiology differs. Further investigation is warranted.

One purpose of my PhD project was to reach a better understanding of CRS in relation to known and hitherto unknown occupational exposures. The work environment is an important part of public health, and research within this field can emphasise the need for further regulations to protect vulnerable groups in society. One new approach to studying CRS and sick leave, particularly in women, is to use the concept of vulnerability, which is complex and multi-faceted. Diderichsen and colleagues describe a functional definition of vulnerability that encompasses the following three dimensions: 1) Exposure to hazard; 2) Susceptibility; and 3) Capacity of response through coping and adaptability (Diderichsen et al 2019).

Differential exposure (1) means that some causes of disease are unevenly distributed across socio-economic groups. Furthermore, Diderichsen et al report that susceptibility (2) is closely linked to Rothman's sufficient-component cause model. This means that a person's susceptibility to disease is dependent upon many, different, smaller potential causes and that these combine in a certain way to cause disease in an individual. Lastly, capacity of response (3) denotes the individual's capacity to handle exposure or to balance exposure. It may also refer to the capacity of an individual to handle the adverse outcomes from an exposure (Diderichsen et al 2019).

It may be beneficial to consider these concepts and theories in order to interpret and understand our results in **Paper IV**. In general, there is a lack of application of public health theories, such as those mentioned above, in the CRS research field. This may be valuable for future studies to broaden and better understand CRS and all of its complexity.

5.4 GENERAL AND METHODOLOGICAL ASPECTS

There are some methodological aspects in this thesis that need to be considered. The way in which we *define chronic rhinosinusitis* is important. In this context, it is essential to include everyone who should be included (sensitivity), and there is a risk that subjects who do not have CRS are also included (1-specificity). Previously, it has been difficult to compare studies on CRS due to the lack of a unanimous definition. The publication of EPOS in

2012, with its epidemiological definition of CRS, has rendered epidemiological research on CRS significantly more comparable. One difficulty encountered in epidemiological research on CRS is the absence of a clinical examination. When interpreting the results one should be aware that the prevalence of CRS, based solely on the epidemiological definition, may be overestimated (Bhattacharyya & Lee 2010). Bhattacharyya & Lee studied 202 patients and compared a symptom-based diagnosis only of CRS with symptoms and endoscopic examination, using computed tomography (CT) as the gold standard. They found a sensitivity of 88.7% and a specificity of 12.3% when the diagnosis of CRS was based on symptoms alone. Another study of 80 subjects has shown a reasonable correlation between use of the epidemiological definition and clinical diagnosis by a trained otorhinolaryngologist (Pilan et al 2012). In this thesis, the epidemiological definition of CRS is used, which given the reasoning above, may result in an overestimation of CRS. The existence of an overestimation per se does not necessarily pose a problem as long as it is taken into account when interpreting and discussing the results.

The *healthy worker effect* is a special form of selection bias (Chowdhury et al 2017). This means that there is a risk that subjects who develop symptoms when working in a certain trade/occupation then leave that occupation. Consequently, when studying this group of workers there is a risk that only the healthy, non-symptomatic subjects remain. We are able to circumvent this effect in **Papers I** and **II** because the subjects reported previous as well as present occupations and symptoms of CRS. Thus, subjects with a specific self-reported exposure were included in the study, regardless of whether they had left that occupation. Another form of selection bias is that individuals with symptoms of CRS may be more likely or motivated to participate in a study because they have symptoms. This would result in the prevalence of CRS being overestimated. However, the prevalence of CRS in the Telemark population was 9% (**Paper I**) which is in accordance with previous prevalence rates for Europe (Hastan et al 2011).

When a subject is asked to determine an occupational exposure retrospectively there is a risk for *recall bias* (Coughlin 1990). Thus, there is a risk that subjects with symptoms recall their occupational exposure better than those without symptoms. The data on occupational exposure in our study are self-reported. As such, there is always a risk that there will be overestimation of the factual exposure and the effect of the exposure on CRS. Therefore, the use of occupational titles is also important.

When studying the effect of an exposure (self-reported occupational exposure) on an outcome (CRS) there is a risk of *confounding*. A confounder is a variable that has an association with the disease and is associated with the exposure, but may not be an effect of the exposure. Different strategies can be used to try to minimise the risk of confounding, such as randomisation, stratification, and using multivariable logistic regression, which is what we have done in our studies (Jager et al 2008).

When conducting epidemiological studies and examining multiple occupational exposures as we have done, there is always the risk of *mass significance* to consider. When conducting multiple statistical tests one needs to be aware that chance may render a few statistically significant results even though all the null hypotheses are true. Bearing this in mind, the Telemark population study has some beneficial characteristics. The sample size is large and the queried occupational exposures are not randomly chosen but instead each represent an exposure that has previously been linked to negative effects on the airways (Moscato & Galdi 2006, Baatjies et al 2023, Siracusa et al 2013). It is always crucial to interpret carefully the statistical results, as well as to evaluate whether the results are sound and if there are other studies that support the findings.

5.5 STRENGTHS AND LIMITATIONS

A strength of the present study is that it is population-based, encompassing subjects from the general population. Many previous studies on CRS have included subjects who are already within the health-care system, often subjects who are already selected for surgery (Sahlstrand-Johnson et al 2011, Azar et al 2017, Adams et al 2023). Our approach allows us to access data on CRS and occupational exposures in the general population, which have not previously been studied to any great extent. The characteristics of the Telemark population (age and gender) are similar to those of Norway in general. This makes it easier to deduce sound generalisations from our results. Nevertheless, it is important to recognize that the study only observes persons aged between 16 and 55 years.

In this thesis, we have assessed occupational exposures using self-reported data. This is a common method to appraise occupational exposures, and it is easy to use but it is connected to a risk of overestimation of the factual exposure. Another method that is used to evaluate occupational exposure is the job exposure matrix (JEM). '*A JEM provides a cross-tabulation of job titles*

(sometimes combined with industry) and estimated exposures to workers carrying out these jobs during different time periods' (Descatha et al 2022). The advantages of using a JEM are that it is an objective method with low cost, and that it allows one to assess occupational exposure when there are no data available other than job titles. The disadvantage is that there may be an underestimation of the factual exposure and effect due to misclassification, and the JEM does not account for exposures varying within an occupation (Descatha et al 2022). The optimal way to evaluate occupational exposure is to measure the factual exposure at a workplace. However, this method is both time-consuming and costly.

The questions listed in the Telemark population study questionnaire have previously been used in several studies, for example the European Community of Respiratory Health Survey and the West Sweden Asthma Study. The questionnaire was only slightly altered to include/exclude occupational exposures that exist/do not exist in the region of Telemark. The questions included regarding respiratory symptoms and disease have been validated in several previous studies (Burney et al 1989, Kongerud et al 1989, Torén et al 1993). The questions regarding CRS have been phrased in accordance with EPOS (Fokkens et al 2020).

The *response rate* in the Telemark population study was relatively low (33% in 2013 and 38% in 2018). Response rates in epidemiological studies have steadily been decreasing over the past four decades. This is thought to be due to more subjects actively choosing not to participate in epidemiological studies, and possibly also due to the increasing numbers of epidemiologic studies in which subjects are asked to participate. Subjects also prefer to participate in studies that relate to issues important in their own lives, and are less likely to take part in studies that involve issues that are un-related to them. The surveys themselves have also become increasingly complex, including for example multiple responses, participation in blood sampling, and entailing a significant time commitment from the participants. The question then arises as to who participates in epidemiologic studies today. The evidence is clear that women are more likely than men to participate in scientific studies. In addition, subjects with higher socioeconomic status, higher education, and subjects with employment and married subjects are more likely to participate in such studies. The evidence regarding age and ethnicity and study participation appears to be inconsistent (Galea & Tracy 2007). This of course also applies to the Telemark population study. However, whilst we are well aware of possible *selection bias*, there are some factors that strengthen the validity of the Telemark

population. The study population in Telemark has similar prevalence rates of smoking and similar age-distribution as the rest of Norway (Statistisk sentralbyrå, Norway, www.ssb.no Accessed: 2024-09-19). Despite the relatively low response rate, the total number of participants is high (around 16,000 in 2013, around 14,500 in 2018 and around 7,900 for responders in both 2013 and 2018). Moreover, a non-responder study conducted in 2016 showed similar prevalence rates for physician-diagnosed asthma and several respiratory symptoms, comparing responders and non-responders (Abrahamsen et al 2016). Risk factors linked to not responding in the 5-year follow-up appear to be younger age, male gender, current smoking, lower educational level, and higher symptom prevalence and morbidity. However, the results from this follow-up study suggest that this loss does not affect occupational exposures as risk factors for respiratory symptoms (Zivadinovic et al 2023).

6 CONCLUSIONS

In the first, cross-sectional study, CRS was significantly and independently correlated with occupational exposure to paper dust, cleaning agents, metal dust, animals, moisture/mould/mildew and physically strenuous work. The prevalence of CRS in the Telemark population in Norway was 9% in 2013.

Occupational exposure to hair-care products, cleaning agents (among women), super glue, strong acids, cooking fumes and wood dust were associated with new-onset CRS. The cumulative 5-year incidence of new-onset CRS between 2013 and 2018 in the Telemark population was 5.5%.

There was a link between BMI and new-onset CRS, with the odds of new-onset CRS increasing with increasing BMI.

Subjects with CRS in the Telemark population reported a higher frequency of sick leave in the past 12 months in both 2013 and 2018, as compared with subjects without CRS. Women with CRS appeared to be more affected than men with CRS. Sick leave in the last year was found to be more common in some occupational groups in subjects with CRS, as compared with subjects without CRS.

7 FUTURE PERSPECTIVES

CRS is a multifactorial disease with many pieces to its jigsaw puzzle. One such piece is occupational exposure, so questioning patients about their occupational exposures is important. My hope is that our research will help to highlight this.

It would be very interesting to repeat the prospective study in this thesis in 2025, which is when the 12-year data from the Telemark population study will become available. It would also be valuable to conduct a clinical validation of the Telemark population, to further corroborate our results. One way in which this could be done would be to select a sample of subjects with the epidemiological definition of CRS in 2025. These subjects could then be invited to participate in a clinical examination, which would include a nasal exam and/or CT of the nose and paranasal sinuses.

Another interesting finding in this thesis that warrants further research is the difference between women with CRS and men with CRS regarding sick leave in the last year. In **Paper IV**, we touch upon possible reasons for this, and further research into the gender differences in CRS would be of great interest and might improve both the efficacy of treatments and prognoses.

There are different ways to assess the relationship between CRS and workplace exposure. In this thesis, we have used self-reporting of occupational exposures. It would be ideal to evaluate workplace exposure using both self-reported exposures and a JEM to correlate with CRS and compare the results. While measuring exposures at the workplace is resource- and time-consuming, it would fill some gaps in the knowledge.

The genetics of CRS are complex and multi-faceted. Nonetheless, it would be very interesting to study the role of occupational exposure in relation to the genetics and epigenetics of CRS. While this would be a difficult and complex study to conduct, it could be worthwhile to identify subjects who have a genetic risk profile and who, therefore, might be informed regarding the risks associated with certain occupations.

8 ACKNOWLEDGEMENTS

First and foremost I would like to extend my sincere gratitude to my main supervisor, Johan Hellgren, for his invaluable guidance and profound knowledge of research and otorhinolaryngology. Thank you for your generous support, expert advice and patience during this thesis.

To Anne Kristin Møller Fell, my co-supervisor, for giving me encouraging and constructive support along the way, for your extensive knowledge of research and fantastic interpersonal skills.

To Kjell Torén, my co-supervisor, for all enriching discussions and whose knowledge and long experience of research has been of great benefit to me.

To Anne Kristin Møller Fell, Martin Veel Svendsen, Geir Klepaker, Regine Abrahamsen and the whole research team at Telemark Sykehus in Norway for allowing me to participate in the Telemark Population Study. I am truly grateful for your enthusiasm, unpretentious sharing of knowledge and for always taking the time to answer my questions.

To Linus Schiöler, Helena Johansson and Martin Veel Svendsen for helping me to understand the world of statistics.

To current and past clinical heads at Capio Lundby Hospital, Therese Westgren, Johan Ellegård, Ulrika Rosell and Agnetha Folestad, for allowing me time off from clinical work to complete this thesis. To past and present heads at the Department of Otorhinolaryngology at Sahlgrenska University Hospital, Radi Jönsson, Erik Renvall and Björn Torén Krusell, for creating a research friendly work environment during the early years of this thesis.

To all of my colleagues and co-workers at the Ear-Nose-and-Throat clinic at Capio Lundby Hospital and at Sahlgrenska for taking care of our patients in my absence.

To all of the respondents of the Telemark population study, thank you for taking the time to participate in the study and consequently making this thesis possible.

Above all, I am forever indebted and especially grateful to my family for without who's assiduous, 24-hour-a-day support for many years this thesis would not have been possible:

My mother, Carina Svensson, for endless love and support, and for introducing me to the world of otorhinolaryngology.

My father, Björn Svensson, for endless love and support, and whose genuine enthusiasm and curiosity for research, and life, is a constant source of inspiration.

My siblings, Hedvig Örneholm and Hjalmar Svensson, and their respective families for always being there. Being your little sister is my great fortune.

Last and always first, to my husband and my co-worker in life, Daniel Clarhed, for whose love and support no formal words could ever convey my gratitude. To our children, Judith, Esther and Arthur, for being our everything.

This thesis and the studies included were financed by grants from the Swedish state under the agreement between the Swedish government and the county councils, the ALF-agreement, and grants from the Gothenburg Society of Medicine, the Foundation Acta Oto-Laryngologica and the Swedish Association for Otorhinolaryngology Head and Neck Surgery.

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10 APPENDIX

Sykehuset Telemark

Påminnelse

Dersom du allerede har svart kan du se bort i fra denne henvendelsen

Astma i Telemark

Ditt svar teller!

Spørreskjema for alle telemarkinger - også for deg som ikke har astma

Dine svar lagres ikke sammen med informasjon om hvem du er

- **ENKELT**
- **VIKTIG**
- **TREKNING AV iPAD/REISE**

Riv av omslaget

We address to you to ask if you want to participate in a research project whose goal is to find out which factors inside and outside of work that affects the respiratory system. Such knowledge can help to prevent the disease of the respiratory tract occurs and to provide better guidance to those who have been sick. The survey will give us more knowledge, the more people who respond. Your response is just as important, whether you are healthy or sick. We ask that you answer as best you can, even if some of the questions may be a bit difficult. It takes about 20 minutes to fill out the form. The questionnaire will be sent to 50,000 randomly selected residents in Telemark county, Norway. The project is carried out of the hospital, Norway in cooperation with Oslo University Hospital.

Tear off the cover (this sheet) and return the questionnaire filled in in the attached envelope frankerte. Thank you in advance for your help!

All who respond to the questionnaire have the chance to win an iPad or a travel gift certificate. The two winners will be drawn and both can choose one of the two Prize options. Premium choice for the one Grand Prize winner does not affect the options to the other. That is, for example, two iPad can be handed out.

For more complete information about the study, see the information type page 15 and 16 as well as our Web site www.sthf.no/astma. You can also scan the QR code below with your Smartphone.

If you have questions about the survey, you can call, send message or email to one of the team members by the Division of the work medicine, Hospital, Norway, Tel: 953 69 315 e-mail: astma@sthf.no

Slik fyller du ut skjemaet.

- Skjemaet vil bli lest maskinelt.
- Det er derfor viktig at du krysser av riktig: Rett ☒ Galt ☑ ☒
- Dersom du krysser feil sted, retter du ved å fylle boksen slik: ■
- Skriv tydelige tall: 0 1 2 3 4 5 6 7 8 9
- Skriv ikke utenfor oppmerket område. Dette vil ikke bli lest av maskinen.
- Bruk svart eller blå penn. Ikke bruk blyant eller tusj.

Personopplysninger

Today's date (ddmmåå):

Gender:

☐ Woman

☐ Man

Height: | | | *cm*

Weight: | | | , *kg*

What is your marital status?

☐ Single

☐ Married

☐ Living With Someone

☐ Divorced/separated

☐ Widow/widowed

Hvor mange års skolegang har du?

(Fra og med første klasse på barneskolen til og med siste fullførte skoleår/studieår).

år

*Hva er din høyeste **fullførte** utdanning?*

(Går du på videregående/fagskole/høyskole/universitet kryss av for siste fullførte utdanning).

☐ Grunnskole/folkeskole

☐ Grunnkurs/1-2 årig utdannelse etter grunnskole

☐ Videregående/gymnas/yrkesskole (3-årig)

☐ Fagbrev

☐ Universitet/høyskole på 4 år eller mindre

☐ Universitet/høyskole på mer enn 4 år

☐ Annet: _____

Vi antar at din arbeidsevne, da den var best, vurderes med 10 poeng. Hvilket poengtall ville du gitt din nåværende arbeidsevne?

(0 betyr at du ikke kan arbeide og 10 at din arbeidsevne er som aller best akkurat nå).

0	1	2	3	4	5	6	7	8	9	10
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Arbeidsforhold

1. *Har du noen gang vært i arbeid?*

☐ Nei (gå til spørsmål 10)

☐ Ja (gå til spørsmål 2)

2. ***Oppgi dine ulike arbeidsforhold (ansettelser) med arbeidsoppgaver og tidsperiode.*** *Arbeidsforhold kortere enn tre måneder behøver du ikke oppgi.*

Hvis du har hatt svært mange arbeidsgivere, men har hatt liknende arbeidsoppgaver, kan du slå sammen periodene. (Eksempel: Bygg og anlegg, gravemaskinfører hos Selmer/Veidekke/Kruse-Smith, 1993-2009). Med ansettelse menes også arbeid som selvstendig næringsdrivende.

Eksempler:

Yara/Fullgjødselfabrikken	*Prosessoperatør*	*2008*	*2010*
Undervisning	*Lærer på yrkesskole*	*2010*	*2011*
Rådgivning	*Konsulent eget firma*	*2011*	*d.d.*

Bransje/industri	***Yrke(tittel)/arbeidsoppgaver***	***Begynt årstall***	***Sluttet årstall***

3. *Har du vært i arbeid* ***de siste 12 måneder****?*

☐ Nei

☐ Ja

Utfyllende spørsmål om dine arbeidsoppgaver ved ulike ansettelsesforhold: Mange av disse spørsmålene er spesielle for visse yrkesgrupper. Hvis spørsmålet ikke gjelder deg; svar nei og gå videre til neste spørsmål.

4. *Har du* ***noen gang*** *i ditt arbeid vært utsatt for:*
Gass, røyk eller støv?

☐ Nei

☐ Ja

5. *Hvis JA, hvor ofte var du utsatt for gass, røyk eller støv i løpet av de **siste fem årene**? (Ta et gjennomsnitt)*

- ☐ Daglig, store deler av arbeidsdagen
- ☐ Daglig, men kortvarig
- ☐ Ukentlig
- ☐ Sjeldnere

6. *Har du **noen gang** i ditt **arbeid** vært utsatt (eksponert) for:*

	Nei	Ja	Siste år utsatt (eksponert)
Stekeos	☐	☐	
Bileksos/motoreksos	☐	☐	
Sterke syrer, ammoniakk eller formalin	☐	☐	
Steinstøv	☐	☐	
Melstøv	☐	☐	
Trestøv	☐	☐	
Papirstøv	☐	☐	
Tekstilstøv	☐	☐	
Metallstøv	☐	☐	

7. *Har du på arbeidsplassen arbeidet med:*

	Nei	Ja	Siste år utsatt (eksponert)
Rengjøring/desinfeksjonsmidler	☐	☐	
Hvis JA, bruker/brukte du spray?	☐	☐	
Superlim eller lynlim	☐	☐	
Malings- eller lakkeringsarbeid	☐	☐	
Sveising eller annen metallrøyk	☐	☐	
Kloakk- eller renseanlegg	☐	☐	
Hårpleieprodukter	☐	☐	
Dyr	☐	☐	

Hvis JA, hvilke dyr? _____

Gass, støv eller damp som ikke er nevnt over

_____

_____

8.

Har du arbeidet i lokaler med:	Nei	Ja	Siste år utsatt (eksponert)
Synlige fuktskader	☐	☐	
Synlig mugg	☐	☐	
Lukt av mugg (kjellerlukt)	☐	☐	
Kulde (i kjølerom eller utendørs på vinteren)	☐	☐	
Har du hatt fysisk anstrengende arbeid (slik at du har blitt andpusten og svett)	☐	☐	
Har du hatt arbeid med gjentakende tunge løft?	☐	☐	

9. *Har du benyttet åndedrettsvern (verne-/støvmaske) på jobb siste 10 år?*

☐ Alltid/nesten alltid

☐ Av og til

☐ Aldri/nesten aldri

Har du bare brukt verne-/støvmaske ved høy eksponering?

☐ Nei

☐ Ja

10. *Har du hatt uhell hjemme eller på jobb hvor du har blitt utsatt for høye nivåer av gass, røyk eller støv?*

☐ Nei

☐ Ja

Hvis JA, fikk du plager fra luftveiene (hoste, tungpust, piping/hvesing) da uhellet skjedde eller like etterpå?

☐ Nei

☐ Ja

Plager fra luftveiene

11.

		Nei	Ja
11.1	Har du hatt piping eller hvesing i brystet på noe tidspunkt i løpet av **de siste 12 månedene**? Hvis NEI, gå til spørsmål 11.2, hvis JA:	☐	☐
	a Har du i det hele tatt vært andpusten når du har hatt piping eller hvesing i brystet?	☐	☐
	b Har du hatt piping eller hvesing i brystet uten at du har vært forkjølet?	☐	☐
11.2	Har du våknet med en følelse av tetthet i brystet på noe tidspunkt i løpet av **de siste 12 månedene**?	☐	☐
11.3	Har du våknet av anfall med tungpust på noe tidspunkt i løpet av **de siste 12 månedene**?	☐	☐

		Nei	Ja
11.4	Har du våknet av hosteanfall på noe tidspunkt i løpet av **de siste 12 månedene**?	☐	☐
11.5	Har du hatt astmaanfall i løpet av **de siste 12 månedene**?	☐	☐
11.6	Bruker du for tiden medisin (spray, inhalasjonspulver eller tabletter) mot astma?	☐	☐
11.7	Har du allergi som gir symptomer fra nesen, inkludert høysnue?	☐	☐
11.8	Har du i løpet av de siste årene hatt langvarig hoste?	☐	☐
11.9	Pleier du å hoste opp slim eller har du slim i lungene som er vanskelig å få opp? Hvis NEI gå til spørsmål 11.10, hvis JA:	☐	☐
	a Hoster du opp, eller får du opp slim på denne måten, nesten hver dag i minst tre måneder hvert år?	☐	☐
	b Har du hatt perioder med slike symptomer i minst to år etter hverandre?	☐	☐
	c Hvor gammel var du da disse problemene begynte? ☐☐ år		
11.10	Har du noen gang hatt piping eller hvesing i brystet?	☐	☐
	Hvis JA, hvor gammel var du da du opplevde piping eller hvesing i brystet første gang? ☐☐ år		
11.11	Har du, eller har du noen gang hatt astma? Hvis NEI gå til spørsmål 11.12, hvis JA:	☐	☐
	a Har du noen gang fått diagnosen astma av lege?	☐	☐
	b Hvor gammel var du da du opplevde astmasymptomer første gang? ☐☐ år		
	c Hvilket år opplevde du sist astmasymptomer? ☐☐☐☐ (åååå)		
11.12	Har en lege noen gang fortalt deg at du har kronisk obstruktiv lungesykdom (KOLS)?	☐	☐
	Hvis JA, hvor gammel var du da du opplevde symptomer på KOLS første gang? ☐☐ år		
11.13	Har du noen gang opplevd nesesymptomer som tett nese, rennende nese eller nyseanfall uten å være forkjølet? Hvis NEI gå til spørsmål 11.14, hvis JA:	☐	☐
	a Hvor gammel var du da du opplevde slike nesesymptomer første gang? ☐☐ år		
	b Har du hatt nesesymptomer **de siste 12 måneder**?	☐	☐
	c Hvilken årstid er dine plager verst? (velg kun ett alternativ) ☐Vår ☐Sommer ☐Høst ☐Vinter ☐Alltid ☐Vet ikke		

		Nei	Ja
11.14	Har du vært tett i nesen **i mer enn 12 uker i løpet av de siste 12 månedene**?	☐	☐
11.15	Har du hatt smerter eller trykk rundt pannen, nese eller øynene **i mer enn 12 uker i løpet av de siste 12 månedene**?	☐	☐
11.16	Har du hatt misfarget nesesekret (snørr) eller misfarget slim i halsen **i mer enn 12 uker i løpet av de siste 12 måneder**?	☐	☐
11.17	Har din luktesans vært nedsatt eller borte **mer enn 12 uker i løpet av de siste 12 månedene**?	☐	☐

Luftveisplager og arbeid

12. *Har du hatt gjentakende luftveisplager (hoste, tungpust, hvesing, piping)* ***på jobb****?*

☐ Nei (gå til spørsmål 15)

☐ Ja

Hvis JA, hvor alvorlige var luftveisplagene?

(0 betyr at du ikke hadde plager og 10 at du hadde svært alvorlige plager).

0	1	2	3	4	5	6	7	8	9	10
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

13. *Ble plagene bedre:*

	Nei	Ja
- i helgene?	☐	☐
- i feriene?	☐	☐
- ved annet fravær fra jobb?	☐	☐
- ved bytte av jobb/omplassering?	☐	☐

14. *Hvis du bruker/har brukt medisin mot luftveisplager; kan/kunne du redusere bruken/dosen?*

	Nei	Ja
- i helgene?	☐	☐
- i feriene?	☐	☐
- ved annet fravær fra jobb?	☐	☐
- ved bytte av jobb/omplassering?	☐	☐

15. *Har du noen gang byttet jobb fordi jobben har påvirket pusten din?*

☐ Nei

☐ Ja

Hvis JA, når var det (hvilket eller hvilke år)?

Årstall ☐☐☐☐

Årstall ☐☐☐☐

Hvis JA, hvilken arbeidsplass (arbeidsoppgaver) hadde du da?

_____

_____

16. *Har du noen gang byttet jobb på grunn av: Høysnue eller andre neseproblemer?*

☐ Nei

☐ Ja

Hvis JA, når var det (hvilket eller hvilke år)?

Årstall ☐☐☐☐

Årstall ☐☐☐☐

Hvis JA, hvilken arbeidsplass (arbeidsoppgaver) hadde du da?

_____

_____

17. *Har du noen gang byttet jobb på grunn av andre helseproblemer/sykdommer?*

☐ Nei

☐ Ja

18. *Har du vært sykemeldt **i løpet av de siste 12 månedene**?*

☐ Nei

☐ Ja

Hvis JA, i hvor mange dager?

Velg kun ett alternativ

☐ 1-7 dager ☐ 8 -14 dager ☐ 15 dager - 12 uker ☐ Mer enn 12 uker

19. *Har du vært sykemeldt **i løpet av de siste 12 månedene** på grunn av pusteproblemer?*

☐ Nei

☐ Ja

Røyke- og snusevaner

20.

	Nei	Ja
Røyker du daglig (gjelder selv om du kun røyker noen få sigaretter, sigarer eller pipe daglig)?	☐	☐
Røyker du bare av og til (ikke daglig, men helger, festrøyking eller liknende)?	☐	☐
Har du røykt tidligere?	☐	☐

Hvis bare NEI-svar på spørsmål 20, gå til spørsmål 25.

21. *Hvor mye røyker/røkte du? (Ta et gjennomsnitt)*

☐ Sigaretter pr **dag** eller ☐ sigaretter pr **uke**

☐ Sigarer pr uke

☐ Pakker rulle-/pipetobakk pr uke

22. *Hvor gammel var du da du begynte å røyke?*

☐ år

23. *Hvor lenge har du røykt (gjelder både nåværende og tidligere røyking)?*

☐ år

24. *Hvis du har røykt tidligere, når sluttet du?*

☐ årstall

25. *Bruker du, eller har du brukt snus?*

Nei, aldri ☐ Ja, av og til ☐

Ja, men jeg har sluttet ☐ Ja, daglig ☐

*Hvis du **aldri** har brukt snus, gå til spørsmål 26.*

Hvis JA:

Hvor gammel var du da du begynte med snus? ☐ *år gammel*

Hvor mange bokser snus bruker/brukte du pr måned? ☐ *bokser snus pr måned*

Dersom du har sluttet å snuse, hvor gammel var du da du sluttet? ☐ *år*

Boligforhold

26. *I hvilken type bolig bor du*

☐ Enebolig

☐ Rekkehus/tomannsbolig

☐ Leilighet/hybel

☐ Annet

27. *Når flyttet du inn i din nåværende bolig?*

☐☐☐☐ årstall

28. *Hvor mange timer pr døgn tilbringer du vanligvis i boligen din?*

Hverdager ☐☐ timer Helg ☐☐ timer

29. *Forekommer det tobakksrøyking inne i din nåværende bolig?* Velg kun ett alternativ.

☐ Nesten daglig ☐ 1-4 ganger/uken ☐ 1-3 ganger/mnd ☐ Aldri

30. *Har du hatt noe av følgende i din bolig?*

	Nei	Ja	Antall år	Siste år utsatt (eksponert)
Vannskader/fuktskader innvendig i boligen på vegger, gulv eller tak?	☐	☐	☐☐	☐☐☐☐
"Bulkete" plastmatter, gulnede plastbelegg eller parkett som har blitt mørk av fukt?	☐	☐	☐☐	☐☐☐☐
Synlig mugg på vegger, gulv eller tak?	☐	☐	☐☐	☐☐☐☐
Har du noen gang i løpet av de siste 10 årene sett tegn på fuktskader, vannlekkasje eller mugg i din bolig?	☐	☐	☐☐	☐☐☐☐

31. *Ligger ditt soveromsvindu nær en gate (mindre enn 20 m)? Velg kun ett alternativ*

☐ Nei
☐ Ja, med lite trafikk
☐ Ja, med moderat trafikk
☐ Ja, med mye trafikk

32. *Hvor mye tid tilbringer du vanligvis på å gå eller ferdes langs en moderat-mye trafikkert vei i løpet av en vanlig hverdag?*

Ca ☐☐☐ min pr dag

33. *Hvilke(n) av følgende oppvarmingsmåter ble mye brukt i ditt hjem da du var fem år gammel?* Flere enn ett alternativ kan være aktuelt.

☐ Vedfyring
☐ Kull
☐ Parafin
☐ Elektrisitet
☐ Gass
☐ Olje
☐ Vannbåren-/ fjernvarme

34. *Hvilket ord beskriver best det stedet du bodde størstedelen av tiden da du var under fem år gammel?* Velg kun ett alternativ

- ☐ Bondegård med dyr
- ☐ Bondegård uten dyr
- ☐ Bygd/tettsted
- ☐ Småby/bynært
- ☐ Storby

35. *Har du (siste 12 måneder) brukt sprayprodukter regelmessig ved rengjøring hjemme?*

- ☐ Nei
- ☐ Ja

Barndom og familie

36.

	Nei	Ja	Vet ikke
Hadde du som barn alvorlig luftveisinfeksjon før 5-års alder?	☐	☐	☐
Røkte din mor regelmessig da du var barn?	☐	☐	☐
Røkte din far regelmessig da du var barn?	☐	☐	☐
Røkte noen annen i ditt hjem regelmessig da du var barn?	☐	☐	☐

37. *Har du foreldre som har, eller har hatt, følgende sykdommer (oppgi også for evt. avdøde foreldre)?* Sett kryss hvis JA

	Mor	Far
Astma	☐	☐
Kronisk bronkitt, emfysem eller KOLS	☐	☐
Hjertesykdom	☐	☐
Høyt blodtrykk	☐	☐
Hjerneblødning/hjerneslag	☐	☐
Diabetes (sukkersyke)	☐	☐
Kreft	☐	☐

Fysisk aktivitet og kosthold

38. *Hvor ofte mosjonerer/trener du? (Ta et gjennomsnitt)*

- ☐ Aldri
- ☐ Mindre enn 1 gang pr uke
- ☐ 1 gang pr uke
- ☐ 2-3 ganger pr uke
- ☐ Omtrent daglig (4-7 ganger pr uke)

39. *Hvis du trener 1 gang pr uke eller mer:*
Hvor hardt mosjonerer/trener du?

- ☐ Tar det rolig uten å bli andpusten eller svett
- ☐ Tar det så hardt at jeg blir andpusten og/eller svett
- ☐ Tar meg nesten helt ut

40. *Hvor lenge pleier du å trene? (Ta et gjennomsnitt)*

- ☐ Mindre enn 15 minutter
- ☐ 15-29 minutter
- ☐ 30 minutter til 1 time
- ☐ Mer enn 1 time

41. *Har du vanligvis minst 30 minutter fysisk aktivitet daglig?*

☐ Nei ☐ Ja

42. *Hvor ofte spiser du vanligvis disse matvarene? (Sett kun ett kryss pr linje)*

	0-3 ganger pr mnd	1-3 ganger pr uke	4-6 ganger pr uke	1 gang pr dag	2 ganger eller mer pr dag
Frukt/bær	☐	☐	☐	☐	☐
Grønnsaker	☐	☐	☐	☐	☐
Sjokolade/smågodt	☐	☐	☐	☐	☐
Kokte poteter	☐	☐	☐	☐	☐
Pasta/ris	☐	☐	☐	☐	☐
Pølser/hamburgere	☐	☐	☐	☐	☐
Fet fisk (laks, ørret, sild, makrell, uer som pålegg/middag)	☐	☐	☐	☐	☐

43. *Bruker du følgende kosttilskudd? (Sett kun ett kryss pr linje)*

	Ja, daglig	Av og til	Nei
Tran	☐	☐	☐
Omega-3-kapsler	☐	☐	☐
Vitamin- og/eller mineraltilskudd	☐	☐	☐

Andre sykdommer og plager

44. *Hvis JA på spørsmålene under, ber vi deg om å fylle inn alder lengst til høyre.*

(Kryss enten nei eller ja på alle spørsmålene)

	Nei	Ja	Hvis JA, hvor gammel var du **første** gang?
Har du fått beskjed av lege om at du har høyt blodtrykk?	☐	☐	☐☐ år
Bruker du medisiner mot høyt blodtrykk?	☐	☐	☐☐ år
Har lege sagt at du har sukkersyke (diabetes)?	☐	☐	☐☐ år
Bruker du medisiner mot diabetes?	☐	☐	☐☐ år
Har du vært innlagt på sykehus med hjerteinfarkt eller hjertekrampe (angina)?	☐	☐	☐☐ år
Har lege noen gang sagt at du har hjertesvikt (svakt hjerte, vann i lungene, hovne ben)?	☐	☐	☐☐ år

45. *Har du, eller har du noen gang hatt noen av disse sykdommene/plagene?*

(Kryss enten nei eller ja på alle spørsmålene)

	Nei	Ja	Hvis JA, hvor gammel var du **første** gang?
Hjerneslag/hjerneblødning?	☐	☐	☐☐ år
Hjerteflimmer?	☐	☐	☐☐ år
Eksem på hendene (med unntak av psoriasis)?	☐	☐	☐☐ år
Annen kronisk lungesykdom enn astma eller KOLS?	☐	☐	☐☐ år
Har du noen gang hatt psykiske plager som du har søkt hjelp for?	☐	☐	☐☐ år

For å sikre din anonymitet blir opplysningene nedenfor ikke skannet eller lagret i databasen.

Samtykke til deltakelse i studien
(Vi ønsker dine svar selv om du ikke velger å skrive under her.)

Jeg er villig til å delta i studien.

(Signatur, dato)

Noen få ganger kan det være aktuelt å kontakte den som har fylt ut skjemaet for å avklare et eller flere spørsmål. **Hvis** du synes det er i orden, ber vi deg om å fylle ut feltene under:

Mobil:_____

Annen tlf. dagtid:_____

Annen tlf. kveldstid:_____

Takk for hjelpen!

Sykehuset Telemark

Astma i Telemark

Astma i Telemark

Bakgrunn og hensikt
Vi henvender oss til deg for å spørre om du vil delta i et forskningsprosjekt som har som mål å finne ut hvilke faktorer i og utenfor arbeid som påvirker luftveiene. Slik kunnskap kan bidra til å hindre at sykdom i luftveiene oppstår og til å gi bedre veiledning til de som er blitt syke. I denne type undersøkelse er hver deltaker viktig, enten du er frisk eller syk. Prosjektet gjennomføres av Sykehuset Telemark i samarbeid med Oslo Universitets-sykehus.

Hva innebærer studien?
I prosjektet sender vi ut et spørreskjema til 50 000 tilfeldig utvalgte innbyggere i alderen 16-50 år i Telemark fylke. Vi spør deg og de andre mottakerne om luftveisplager og hva dere utsettes for i arbeid og på fritiden. Vi planlegger å kontakte noen av dem som har astma og noen friske deltagere på et senere tidspunkt, for å spørre om de kan tenke seg å være med i en oppfølgende studie. Deltagelse i senere studier er frivillig.

Mulige fordeler og ulemper
Fordeler ved deltakelse i studien: Det er viktig å vite noe om hvor mange som har plager fra luftveiene og som utvikler astma. Enda viktigere er det å vite mer om hvilke faktorer i og utenfor arbeid som kan gi plager fra luftveiene for å kunne forebygge sykdom i fremtiden. Dette kan du bidra til ved å delta i studien. I tillegg vil studien også kunne være til hjelp for politikere og helsepersonell til bedre å kunne planlegge og gjennomføre nødvendige tiltak i Telemark og landet for øvrig, for å møte de behov og kostnader som følger av astma.

Mulige ulemper ved deltakelse i studien: Det hender at noen deltagere blir bekymret av å fylle ut et spørreskjema. Hvis dette gjelder deg, er vi tilgjengelige på telefon for å kunne svare på spørsmål og gi råd.

Hva skjer med informasjonen om deg?
Opplysningene som registreres skal kun brukes slik som beskrevet i hensikten med studien. Informasjonen om deg, vil bli viderebehandlet uten navn, fødselsnummer eller andre direkte gjenkjennende opplysninger. Dine opplysninger vil bli knyttet til en kode gjennom en løpenummerliste som kun autorisert personell som er tilknyttet prosjektet har adgang til. Det vil ikke være mulig å identifisere deg i resultatene av studien når disse publiseres.

Frivillig deltakelse
Deltakelse i prosjektet er frivillig og du kan trekke deg når som helst uten å oppgi noen grunn. Dersom du senere ønsker å trekke deg vil alle opplysninger om deg bli slettet. Dersom du ønsker å delta, undertegner du samtykkeerklæringen, besvarer spørre-skjemaet og returnerer det i vedlagte ferdig frankerte svarkonvolutt. Dersom du senere ønsker å trekke deg eller har spørsmål til studien, kan du kontakte en av prosjektmedarbeiderne på Sykehuset Telemark, tlf: 953 69 315 (kl. 08.00-16.00). Mer informasjon om personvern og forsikring finnes under tilsvarende avsnitt nedenfor.

Personvern, økonomi og forsikring

Personvern

Opplysninger som registreres om deg vil bli oppbevart i låste arkiver og i datasystem som er beskyttet av Sykehuset Telemarks IT-rutiner. Ved publisering av resultatene vil alle opplysninger være anonymisert. Opplysningene fra spørreskjemaet vil bli slettet når prosjektet avsluttes, og senest i 2035. Sykehuset Telemark HF ved administrerende direktør er databehandlingsansvarlig.

Utlevering av materiale og opplysninger til andre

Hvis du sier ja til å delta i studien, gir du også ditt samtykke til at avidentifiserte opplysninger utleveres til prosjektmedarbeidere tilknyttet prosjektet ved Sykehuset Telemark HF og Oslo Universitetssykehus HF.

Rett til innsyn og sletting av opplysninger om deg og sletting av prøver

Hvis du sier ja til å delta i studien, har du rett til å få innsyn i hvilke opplysninger som er registrert om deg. Du har videre rett til å få korrigert eventuelle feil i de opplysningene vi har registrert. Dersom du trekker deg fra studien, kan du kreve å få slettet innsamlede opplysninger, med mindre opplysningene allerede er inngått i analyser eller brukt i vitenskapelige publikasjoner.

Økonomi

Studien er finansiert gjennom forskningsmidler fra Sykehuset Telemark HF.

Forsikring

Alle deltakerne i studien er forsikret gjennom Norsk pasientskadeerstatning.

Informasjon om utfallet av studien

Resultatene av prosjektet vil bli formidlet gjennom offentlige medier lokalt og nasjonalt og gjennom vitenskapelige artikler internasjonalt. Du vil finne oppdatert informasjon om prosjektet på prosjektets hjemmeside: www.sthf.no/astma

Med vennlig hilsen

Regine Abrahamsen
Lege, Sykehuset Telemark
Seksjon for arbeidsmedisin

Anne Kristin Møller Fell
Overlege, Sykehuset Telemark
Seksjon for arbeidsmedisin

Dersom du har spørsmål, kan du ringe, sende melding eller e-post til en av prosjektmedarbeiderne ved Seksjon for arbeidsmedisin, Sykehuset Telemark tlf: 953 69 315
e-post: astma@sthf.no

SYKEHUSET TELEMARK

TELEMARKSTUDIEN

Ola Nordmann
Adresse
0000 Poststed

Siste påminnelse

Dersom du allerede har svart kan du se bort i fra denne henvendelsen

Forespørsel til deg

Dette er en forespørsel til deg som bor eller har bodd i Telemark om å delta i en befolkningsundersøkelse, et helseforskningsprosjekt. Vi vil be deg om å fylle ut dette spørreskjemaet. Det tar ca. 20 minutter. Du kan velge om du vil svare på nett (elektronisk spørreskjema) eller returnere papirskjema i vedlagte frankerte konvolutt. Undersøkelsen gjennomføres av Sykehuset Telemark i samarbeid med Universitetet i Oslo. Opplysningene blir brukt til forskning og forebyggende helsearbeid. Forskere vil kun ha tilgang til anonymiserte data, det vil si at resultatene ikke kan spores tilbake til en enkeltperson.

Spørreskjemaet har tidligere vært sendt ut i 2013, men noen får det nå for første gang. Denne oppfølgingen vil gi ny oppdatert kunnskap.

Riv av forsiden (denne siden) før du returnerer spørreskjemaet. Porto er betalt.

For forebyggende helsearbeid og forskning

Ved elektronisk besvarelse

Gå inn på: **resp.nsd.no**

BrukerID: **XXXXXXXX**
Passord: **XXXX**

Slik fyller du ut skjemaet

- Skjemaet vil bli lest maskinelt
- Det er derfor viktig at du krysser av korrekt: **Rett** ☒ **Galt** ☒ ☑
- Dersom du krysser feil sted, retter du ved å fylle boksen slik: ■
- Skriv tydelige tall: 0 1 2 3 4 5 6 7 8 9
- Skriv ikke utenfor oppmerket område. Dette vil ikke bli lest av maskinen
- Bruk svart eller blå penn. Ikke bruk blyant eller tusj.

Vennligst fyll ut skjemaet, og post det snarest mulig dersom du ønsker å delta.

Ved spørsmål kan du ringe eller sende melding til tlf: 953 69 315,
eller sende e-post til: astma@sthf.no

TELEMARK STUDIEN

Resultater fra første runde av Telemarkstudien

Telemarkstudien har pågått siden 2013 og har gitt ny kunnskap og mange interessante funn. Nedenfor finner du noen av dem. Vi takker deg for at du er med. Dine svar bidrar til mer effektiv forebygging av helseplager i regionen vår.

Færre røyker

Telemarkstudien viser at det er færre som røyker daglig i Telemark sammenliknet med tidligere. I 2013 oppga 15 prosent i aldersgruppen 16 til 50 år at de røykte. Vi mangler ferske tall for Telemark, men landsgjennomsnittet er nå redusert til 12 prosent. Likevel er det mange som utsettes for passiv røyking. Seks prosent av voksne i Telemark som aldri har røykt, har vært utsatt for passiv røyking hjemme. Disse hadde økt risiko for kronisk hoste og oppvåkning om natten på grunn av tung pust.

Link: http://erj.ersjournals.com/content/48/suppl_60/PA4290

Fukt og mugg

I 2013 svarte hver femte telemarking at de har hatt fukt- eller muggskader i egen bolig. Å være utsatt for fukt og mugg hjemme gir økt risiko for luftveisplager og astma.
Det var også 15 prosent som hadde observert slike skader på arbeidsplassen. Hvis du har mugg og fukt i din bolig, bør du så snart det er praktisk mulig, fjerne de skadede materialene og lufte godt i mange uker.

Kronisk bihulebetennelse

I Telemark rapporterte nesten hver tiende deltager å ha kronisk bihulebetennelse. Vi vet fra andre studier at bihuleplager går utover livskvaliteten. Telemarkstudien har vist at de som blir utsatt for metallstøv, rengjøringsmidler og stekeos på jobben har økt risiko for kronisk bihulebetennelse. I tillegg fant vi at astmatikere og røykere hadde mer bihulebetennelse enn andre. Denne kunnskapen er viktig i arbeidet med å forebygge denne hyppige og plagsomme tilstanden.

Må bytte jobb på grunn av pusten

De som svarte på Telemarkstudiens spørreskjema i 2013 har deltatt i nybrottsarbeid: For første gang vet vi nå hvilke yrker som kan påvirke pusten så mye at ansatte må bytte jobb. To prosent av telemarkingene oppga at de hadde byttet jobb fordi jobben påvirket pusten.
Yrkene som gav økt risiko for jobb-bytte var kokk, platearbeider, sveiser, gartner, frisør, renholder og ansatt på større bondegårder. Tiltak som gjør at de fleste kan fortsette i disse yrkene er at støvnivåene reduseres, at andre arbeidsoppgaver tilbys de som får plager og at bruken av verneutstyr blir bedre.

Link: http://oem.bmj.com/content/oemed/73/9/600.full.pdf

Yrker og luftveisplager

Elleve prosent av voksne i Telemark har astma som de oppgir er diagnostisert av lege. Det er ikke flere enn i resten av landet, men når hver tiende innbygger har denne kroniske lungesykdommen bør mer gjøres. For å hindre at flere blir syke, og for at flere skal få behandling, er det behov for mer kunnskap. Telemarksstudien viser at de næringene som gav økt risiko for å få luftveisplager var jordbruk, fiskeindustri, håndverk og handel. Å være utsatt for blant annet melstøv, isocyanater, sveiserøyk og eksos gir også økt risiko for enkelte luftveisplager. Disse funnene gjør at vi nå vet mer om hvor tiltak bør settes inn.

Link: http://bmjopen.bmj.com/content/bmjopen/7/3/e014018

Dato for utfylling: ☐ / ☐ / 2018
Dag Mnd År

PERSONOPPLYSNINGER

Kjønn:

☐ Kvinne

☐ Mann

Høyde ☐ cm
Vekt ☐ kg

Hva er din sivilstand?

☐ Enslig

☐ Gift

☐ Samboer

☐ Skilt/separert

☐ Enke/-mann

Hvor mange års skolegang/utdannelse har du?
(Fra og med første klasse på barneskolen til og med siste fullførte skoleår/studieår). ☐ år

Hva er din høyeste **fullførte** utdanning?
(Går du på videregående/fagskole/høyskole/universitet kryss av for siste fullførte utdanning).

☐ Grunnskole/folkeskole

☐ Grunnkurs/1-2 årig utdannelse etter grunnskole

☐ Videregående/gymnas/yrkesskole (3-årig)

☐ Fagbrev

☐ Universitet/høyskole på 4 år eller mindre

☐ Universitet/høyskole på mer enn 4 år

☐ Annet: _ _ _ _ _ _ _ _ _ _ _ _ _ _ _ _

Vi antar at din arbeidsevne, da den var best, vurderes med 10 poeng. Hvilket poengtall ville du gitt din nåværende arbeidsevne? (0 betyr at du ikke kan arbeide og 10 at din arbeidsevne er som aller best akkurat nå).

0	1	2	3	4	5	6	7	8	9	10
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Total samlet bruttoinntekt i husstanden

☐ Under 500 000

☐ Over 500 000

☐ Over 1 000 000

ARBEIDSFORHOLD

1. Har du noen gang vært i arbeid?

☐ Nei (gå til spørsmål 11)

☐ Ja (gå til spørsmål 2)

2. **Oppgi dine ulike arbeidsforhold (ansettelser) med arbeidsoppgaver og tidsperiode.** Arbeidsforhold kortere enn tre måneder behøver du ikke oppgi.

Hvis du har hatt svært mange arbeidsgivere, men har hatt liknende arbeidsoppgaver, kan du slå sammen periodene. (Eksempel: Bygg og anlegg, gravemaskinfører hos Selmer/Veidekke/Kruse-Smith, 1993-2009). Med ansettelse menes også arbeid som selvstendig næringsdrivende.

Eksempel:
Bransje/industri: Yara/fullgjødselfabrikken
Yrke(tittel)/arbeidsoppgaver: Prosessoperatør
Begynt årstall 2008 *Sluttet årstall* d. d.

Bransje/industri _ _ _ _ _ _ _ _ _ _ _ _ _ _ _
Yrke(tittel)/arbeidsoppgaver _ _ _ _ _ _ _ _ _ _ _
Begynt årstall ☐ Sluttet årstall ☐

Bransje/industri _ _ _ _ _ _ _ _ _ _ _ _ _ _ _
Yrke(tittel)/arbeidsoppgaver _ _ _ _ _ _ _ _ _ _ _
Begynt årstall ☐ Sluttet årstall ☐

Bransje/industri _ _ _ _ _ _ _ _ _ _ _ _ _ _ _
Yrke(tittel)/arbeidsoppgaver _ _ _ _ _ _ _ _ _ _ _
Begynt årstall ☐ Sluttet årstall ☐

Bransje/industri _ _ _ _ _ _ _ _ _ _ _ _ _ _ _
Yrke(tittel)/arbeidsoppgaver _ _ _ _ _ _ _ _ _ _ _
Begynt årstall ☐ Sluttet årstall ☐

Bransje/industri _ _ _ _ _ _ _ _ _ _ _ _ _ _ _
Yrke(tittel)/arbeidsoppgaver _ _ _ _ _ _ _ _ _ _ _
Begynt årstall ☐ Sluttet årstall ☐

3. Har du vært i arbeid **de siste 12 måneder?**

☐ Nei ☐ Ja

Utfyllende spørsmål om dine arbeidsoppgaver ved ulike ansettelsesforhold: mange av disse spørsmålene er spesielle for visse yrkesgrupper. Hvis spørsmålet ikke gjelder deg; svar nei og gå videre til neste spørsmål.

4 Har du noen gang i ditt arbeid vært utsatt for: gass, røyk eller støv? *Hvis NEI gå til spørsmål 6*

☐ Nei ☐ Ja ☐ Ja, de siste 12 mnd

5 Hvis du har vært utsatt for gass, røyk eller støv i løpet av **de siste 12 mnd**, hvor ofte? (Ta et gjennomsnitt)

☐ Daglig, store deler av arbeidsdagen
☐ Daglig, men kortvarig
☐ Ukentlig
☐ Sjeldnere

6 Har du i ditt arbeid vært utsatt (eksponert) for:

	Nei	Ja	Ja, siste 12 mnd
Stekeos	☐	☐	☐
Bileksos/motoreksos	☐	☐	☐
Sterke syrer, ammoniakk eller formalin	☐	☐	☐
Steinstøv	☐	☐	☐
Melstøv (baker, konditor, kokk, møller)	☐	☐	☐
Trestøv	☐	☐	☐
Støv fra papirproduksjon	☐	☐	☐
Metallstøv	☐	☐	☐

7 Har du på arbeidsplassen arbeidet regelmessig med:

	Nei	Ja	Ja, siste 12 mnd (eksponert)
Rengjøring/desinfeksjonsmidler	☐	☐	☐
Hvis JA, bruker/brukte du spray?	☐	☐	☐
Lim (super-lynlim, lim til negler/vipper)	☐	☐	☐
Malings- eller lakkeringsarbeid	☐	☐	☐
Sveising eller annen metallrøyk	☐	☐	☐
Kloakk- eller renseanlegg	☐	☐	☐
Hårpleieprodukter	☐	☐	☐
Dyr	☐	☐	☐
Biologisk støv (planter, organisk materiale, o.l.)	☐	☐	☐
Gass, støv eller damp som ikke er nevnt over	☐	☐	☐

8 Har du arbeidet i lokaler med:

	Nei	Ja	Ja, siste 12 mnd (eksponert)
Synlige fuktskader	☐	☐	☐
Synlig mugg	☐	☐	☐
Lukt av mugg (kjellerlukt)	☐	☐	☐
Kulde (kjølerom/utendørs på vinteren)	☐	☐	☐
Har du hatt fysisk anstrengende arbeid (slik at du har blitt andpusten og svett)	☐	☐	☐
Har du hatt arbeid med gjentakende tunge løft?	☐	☐	☐

9 Har du benyttet åndedrettsvern (verne-/støvmaske) på jobb **de siste 12 mnd**?

☐ Alltid/nesten alltid ☐ Av og til ☐ Aldri/nesten aldri

10 Har du hatt uhell på jobb eller i fritid hvor du har blitt utsatt for høye nivåer av gass, røyk eller støv?

Jobb: ☐ Nei ☐ Ja ☐ Ja, siste 12 mnd

Fritid: ☐ Nei ☐ Ja ☐ Ja, siste 12 mnd

Hvis JA, Hvilken type gass, røyk eller støv var du utsatt for?

Hvis JA, Fikk du luftveisplager (hoste, tungpust, piping/hvesing) da uhellet skjedde eller like etterpå?

Jobb: ☐ Nei ☐ Ja ☐ Ja, siste 12 mnd

Fritid: ☐ Nei ☐ Ja ☐ Ja, siste 12 mnd

PLAGER FRA LUFTVEIENE

11 Har du noen gang hatt piping eller hvesing i brystet?

☐ Nei ☐ Ja

Hvis JA, hvor gammel var du da du opplevde piping eller hvesing i brystet første gang? ☐☐ år

12 Har du hatt piping eller hvesing i brystet på noe tidspunkt i løpet av **de siste 12 mnd**? Nei ☐ Ja ☐

Hvis NEI, gå til spørsmål 13

Hvis JA:

a) Har du vært andpusten når du har hatt piping eller hvesing i brystet? ☐ ☐

b) Har du hatt piping eller hvesing i brystet uten at du har vært forkjølet? ☐ ☐

13 Har du hatt tungpust på noe tidspunkt i løpet av **de siste 12 månedene**?

☐ Nei ☐ Ja

Hvis NEI, gå til spørsmål 15. **Hvis JA:**

a) Har du på noe tidspunkt **de siste 12 månedene** hatt anfall med tungpust når du har vært i ro? Nei ☐ Ja ☐

b) Har du på noe tidspunkt **de siste 12 månedene** hatt anfall med tungpust etter å ha vært eksponert for kulde? ☐ ☐

14 Har du våknet av anfall med tungpust på noe tidspunkt i løpet av **de siste 12 månedene**?

☐ Nei ☐ Ja

15 Har du våknet med en følelse av tetthet i brystet på noe tidspunkt i løpet av **de siste 12 månedene**?

☐ Nei ☐ Ja

16 Har du våknet av hosteanfall på noe tidspunkt i løpet av **de siste 12 månedene**?

☐ Nei ☐ Ja

17 Har du vært tungpustet uten at du har vært forkjølet?

☐ Nei ☐ Ja

18 Har du i løpet av **de siste årene** hatt langvarig hoste?

☐ Nei ☐ Ja

---

19 Pleier du å hoste opp slim eller har du slim i lungene som er vanskelig å få opp?

☐ Nei ☐ Ja

Hvis NEI gå til spørsmål 20

Hvis JA:

	Nei	Ja
a) Hoster du opp, eller får du opp slim på denne måten, nesten hver dag i minst tre måneder hvert år?	☐	☐
b) Har du hatt perioder med slike symptomer i minst to år etter hverandre?	☐	☐

c) Hvor gammel var du da disse problemene begynte?

☐☐ år

---

20 Har du, eller har du noen gang hatt astma?

☐ Nei ☐ Ja

Hvis NEI gå til spørsmål 21

Hvis JA:

	Nei	Ja
a) Har du noen gang fått diagnosen astma av lege?	☐	☐

b) Hvor gammel var du da du opplevde astmasymptomer første gang? ☐☐ år

c) Har du hatt astmaanfall i løpet av **de siste 12 månedene**? ☐ ☐

d) Hvilket år opplevde du sist astmasymptomer?

☐☐☐☐ (åååå)

---

21 Har en lege noen gang fortalt deg at du har kronisk obstruktiv lungesykdom (KOLS)?

☐ Nei ☐ Ja

Hvis JA, hvor gammel var du da du opplevde symptomer på KOLS første gang? ☐☐ år

---

22 Har du allergi som gir symptomer fra nesen, inkludert høysnue?

☐ Nei ☐ Ja

---

23 Har du noen gang opplevd nesesymptomer som tett nese, rennende nese eller nyseanfall **uten å være forkjølet**?

☐ Nei ☐ Ja

Hvis NEI gå til spørsmål 24

Hvis JA:

a) Hvor gammel var du da du opplevde slike nesesymptomer første gang? ☐☐ år

b) Har du hatt nesesymptomer **de siste 12 måneder**?

☐ Nei ☐ Ja

c) Hvilken årstid er dine plager verst? (velg kun ett alternativ)

☐ Vår ☐ Sommer ☐ Høst

☐ Vinter ☐ Alltid ☐ Vet ikke

---

24 Har du vært tett i nesen i **mer enn 12 uker i løpet av de siste 12 månedene**?

☐ Nei ☐ Ja

---

25 Har du hatt smerter eller trykk rundt pannen, nese eller øynene i **mer enn 12 uker i løpet av de siste 12 månedene**?

☐ Nei ☐ Ja

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26 Har du hatt misfarget nesesekret (snørr) eller misfarget slim i halsen i **mer enn 12 uker i løpet av de siste 12 måneder**?

☐ Nei ☐ Ja

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27 Har din luktesans vært nedsatt eller borte **mer enn 12 uker i løpet av de siste 12 månedene**?

☐ Nei ☐ Ja

---

28 Har du operert bort mandler eller nesepolypper?

☐ Nei ☐ Ja

Hvis JA, omtrent hvor gammel var du **første gang**?

☐☐ år

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29 Har du på noe tidspunkt **de siste 12 månedene** hatt anfall med tungpust etter anstrengelse?

☐ Nei ☐ Ja ☐☐ Antall ganger

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30 Har du på noe tidspunkt **de siste 12 månedene** oppsøkt fastlege, spesialist, legevakt eller sykehus på grunn av akutt pustebesvær?

☐ Nei ☐ Ja ☐☐ Antall ganger

Hvis JA

☐ Fastlege ☐ Spesialist

☐ Legevakt ☐ Sykehus

Hvis sykehus:

Har du på noe tidspunkt **de siste 12 månedene** vært innlagt på sykehus på grunn av pustebesvær?

☐ Nei ☐ Ja ☐☐ Antall ganger

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31 Har du økt bruken eller fått nye medisiner på grunn av lunge- eller luftveisplager på noe tidspunkt **de siste 12 månedene**?

☐ Nei ☐ Ja

Hvis JA	Antall ganger
Bare antibiotika	☐☐
Bare kortison (f.eks prednisolon)	☐☐
Antibiotika og kortison	☐☐

LUFTVEISPLAGER OG ARBEID

32 Har du hatt gjentakende luftveisplager (hoste, tungpust, hvesing, piping) **på jobb**?

☐ Nei (gå til spørsmål 35)

☐ Ja

☐ Ja, de siste 12 mnd

Hvor alvorlige var luftveisplagene?
(0 betyr at du ikke hadde plager og 10 at du hadde svært alvorlige plager).

0	1	2	3	4	5	6	7	8	9	10
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

33 Ble plagene bedre:

	Nei	Ja
- i helgene?	☐	☐
- i feriene?	☐	☐
- ved annet fravær fra jobb?	☐	☐
- ved bytte av jobb/omplassering?	☐	☐

34 Hvis du bruker/har brukt medisin mot luftveisplager; kan/kunne du redusere bruken/dosen

	Nei	Ja
- i helgene?	☐	☐
- i feriene?	☐	☐
- ved annet fravær fra jobb?	☐	☐
- ved bytte av jobb/omplassering?	☐	☐

35 Har du noen gang byttet jobb fordi jobben har påvirket pusten din?

☐ Nei ☐ Ja

Hvis JA, når var det (hvilket eller hvilke år)?

____ Årstall ____ Årstall

Hvis JA, hvilken arbeidsplass (arbeidsoppgaver) hadde du da?

36 Har du noen gang byttet jobb på grunn av: Høysnue eller andre neseproblemer?

☐ Nei ☐ Ja

Hvis JA, når var det (hvilket eller hvilke år)?

____ Årstall ____ Årstall

Hvis JA,
hvilken arbeidsplass (arbeidsoppgaver) hadde du da?

37 Har du vært sykemeldt **i løpet av de siste 12 månedene**?

☐ Nei ☐ Ja

Hvis JA, i hvor mange dager? (Velg kun ett alternativ)

☐ 1-7 dager ☐ 8 -14 dager

☐ 15 dager - 12 uker ☐ Mer enn 12 uker

38 Har du vært sykemeldt i løpet av **de siste 12 månedene** på grunn av pusteproblemer?

☐ Nei ☐ Ja

MEDISINER

39 Har du i løpet av **de siste 12 månedene** brukt

	Nei	Ja
Kortisontabletter	☐	☐
Avsvellende nesespray (Rhinox, Dexyl, Nazaren, Otrivin, Zymelin, Zycomb)	☐	☐
Nesespray med kortison (Budesonid, Rhinocort, Flutide, Mometasone, Nasonex, Hasacort, Dymista)	☐	☐
Allergitabletter (antihistaminer) f. eks Zyrtec, Aerius, Cetirizine	☐	☐
Medisiner mot høyt blodtrykk	☐	☐
Medisiner mot diabetes	☐	☐
Medisiner mot høyt kolesterol	☐	☐

40 Bruker du for tiden medisin (spray, inhalasjonspulver eller tabletter) mot astma?

☐ Nei (gå til spørsmål 43)

☐ Ja

41 Har du i løpet av **de siste 12 månedene** brukt en eller flere av disse lungemedisinene:

	Nei	Ja, fast	Ja, ved behov
Airomir, Buventol, Ventoline,Bricanyl, Seritide, Oxis, Onbrez, Striverdi	☐	☐	☐
Airflusal, Seretide, Salmeterol/ fluticasone, Duoresp, Symbicort, Inuxair, Relvar, Flutiform	☐	☐	☐
Anoro, Ultibro, Duaklir, Spiolto	☐	☐	☐
Airobec, Giona, Pulmicort, Flutide, Flutikason, Asmanex, Alvesco	☐	☐	☐
Seebri, Incruse	☐	☐	☐

42 Har du **økt** din kortisoninhalasjon på noe tidspunkt **de siste 12 månedene**?

☐ Nei

☐ Ja

RØYKE- OG SNUSEVANER

	Nei	Ja
43 Røyker du daglig (gjelder selv om du kun røyker noen få sigaretter, sigarer eller pipe daglig)?	☐	☐
Røyker du bare av og til (ikke daglig, men helger, festrøyking eller liknende)?	☐	☐
Har du røykt tidligere?	☐	☐

Hvis bare NEI-svar på spørsmål 43, gå til spørsmål 48

44 Hvor mye røyker/røkte du? (Ta et gjennomsnitt)

☐☐ Sigaretter pr dag eller ☐☐ sigaretter pr uke

☐☐ Sigarer pr uke

☐☐ Pakker rulle-/pipetobakk pr uke

45 Hvor gammel var du da du begynte å røyke?

☐☐ år

46 Hvor lenge har du røykt (gjelder både nåværende og tidligere røyking)?

☐☐ år

47 Hvis du har røykt tidligere, når sluttet du?

☐☐☐☐ årstall

48 Bruker du, eller har du brukt snus?

☐ Nei, aldri (gå til spørsmål 50) ☐ Ja, av og til

☐ Ja, men jeg har sluttet ☐ Ja, daglig

49 Hvor lenge har du brukt snus (gjelder både nåværende og tidligere snusing)?

☐☐ år

BOLIGFORHOLD OG EIERFORM

50 I hvilken type bolig bor du? **(Velg to alternativer)**

☐ Enebolig ☐ Leilighet/hybel

☐ Rekkehus/tomannsbolig ☐ Annet

OG

☐ Selveier/Sameie ☐ Leier i privat eid bolig

☐ Leier i offentlig eid bolig

51 Når flyttet du inn i din nåværende bolig?

☐☐☐☐ årstall

52 Forekommer det tobakksrøyking inne i din nåværende bolig? (Velg kun ett alternativ)

☐ Daglig/nesten daglig ☐ 1-4 ganger/uken

☐ 1-3 ganger/mnd ☐ Aldri

53 Har du hatt noe av følgende i din bolig?

	Siste år utsatt (eksponert) (åååå)
Vannskader/fuktskader innvendig i boligen på vegger, gulv eller tak? ☐ Nei ☐ Ja ☐☐ Antall år	☐☐☐☐
"Bulkete" plastmatter, gulnede plastbelegg eller parkett som har blitt mørk av fukt? ☐ Nei ☐ Ja ☐☐ Antall år	☐☐☐☐
Synlig mugg på vegger, gulv eller tak? ☐ Nei ☐ Ja ☐☐ Antall år	☐☐☐☐
Har du noen gang i løpet av de siste 10 årene sett tegn på fuktskader, vannlekkasje eller mugg i din bolig? ☐ Nei ☐ Ja ☐☐ Antall år	☐☐☐☐

54 Ligger ditt soveromsvindu nær en gate (mindre enn 20 m)? Velg kun ett alternativ

☐ Nei ☐ Ja, med moderat trafikk

☐ Ja, med lite trafikk ☐ Ja, med mye trafikk

55 Hvor mye tid tilbringer du vanligvis på å gå eller ferdes (sykkel e.l.) langs en moderat-mye trafikkert vei i løpet av en vanlig hverdag?

Ca ☐☐☐ min pr dag

57 Har du **de siste 12 måneder** brukt sprayprodukter regelmessig ved rengjøring hjemme?

☐ Nei ☐ Ja

58 Har du vedfyring i din bolig?

☐ Nei ☐ Ja

Hvis JA, hvilken type:

☐ Ny eller rentbrennende vedovn

☐ Gammel vedovn

☐ Åpen peis

Hvis JA, hvor ofte fyrer du med ved i fyringssesongen?

☐ Daglig ☐ 2-3 ganger per uke ☐ Sjeldnere

59 Når du sover, har du vanligvis vinduet på soverommet åpent eller lukket? (Sett ett kryss for hver linje.)

Sommer:	☐ Åpent	☐ Lukket
Vinter:	☐ Åpent	☐ Lukket

BARNDOM OG FAMILIE

61 Har du foreldre som har, eller har hatt, følgende sykdommer (oppgi også for evt. avdøde foreldre)?

	Mor	Far
Astma	☐	☐
Kronisk bronkitt, emfysem eller KOLS	☐	☐
Hjertesykdom	☐	☐
Høyt blodtrykk	☐	☐
Hjerneblødning/hjerneslag	☐	☐
Diabetes (sukkersyke)	☐	☐
Kreft	☐	☐

FYSISK AKTIVITET OG KOSTHOLD

62 Hvor ofte mosjonerer/trener du?

- ☐ Aldri
- ☐ Mindre enn 1 gang pr uke
- ☐ 1 gang pr uke
- ☐ 2-3 ganger pr uke
- ☐ Omtrent daglig (4-7 ganger pr uke)

63 Hvis du trener 1 gang pr uke eller mer: Hvor hardt mosjonerer/trener du?

- ☐ Tar det rolig uten å bli andpusten eller svett
- ☐ Tar det så hardt at jeg blir andpusten og/eller svett
- ☐ Tar meg nesten helt ut

64 Hvor lenge pleier du å trene? (Ta et gjennomsnitt)

- ☐ Mindre enn 15 minutter
- ☐ 15-29 minutter
- ☐ 30 minutter til 1 time
- ☐ Mer enn 1 time

65 Har du vanligvis minst 30 minutter fysisk aktivitet daglig slik at du blir svett eller andpusten?

- ☐ Nei
- ☐ Ja

66 Hvor ofte spiser du vanligvis disse matvarene? (Sett kun ett kryss pr linje)

	0-3 pr mnd	1-3 pr uke	4-6 pr uke	1 pr dag	2 eller fler pr dag
Grovbrød eller andre grove kornprodukter	☐	☐	☐	☐	☐
Sjokolade/smågodt	☐	☐	☐	☐	☐
Sukkerholdig brus eller annen leskedrikk med sukker	☐	☐	☐	☐	☐
Pølser/hamburgere	☐	☐	☐	☐	☐
Fet fisk (laks, ørret, makrell, sild, uer som pålegg/middag)	☐	☐	☐	☐	☐

67 Hvor mange porsjoner grønnsaker eller frukt/bær spiser du i løpet av dagen? En porsjon kan f.eks. være 1 middels stor frukt eller 1 gulrot, 1 skive kålrot eller en porsjonsbolle salat.

Antall posjoner: (sett kryss nedenfor)

	0	½	1	2	3	4	5 eller fler
Grønnsaker (uten poteter)	☐	☐	☐	☐	☐	☐	☐
Frukt eller bær (inkludert juice, max 1 glass)	☐	☐	☐	☐	☐	☐	☐

68 Hvor ofte spiser du frokost?

- ☐ Sjelden/aldri
- ☐ 1-2 ganger i uka
- ☐ 3-4 ganger i uka
- ☐ 5-6 ganger i uka
- ☐ Hver dag

ANDRE SYKDOMMER OG PLAGER

69 **Hvis JA på spørsmålene under**, ber vi deg om å fylle inn alder lengst til høyre. (Kryss enten nei eller ja på alle spørsmålene)

	Nei	Ja	**Hvis JA**, alder første gang?
Har du fått beskjed av lege om at du har høyt blodtrykk?	☐	☐	☐☐ år
Har lege sagt at du har sukkersyke (diabetes)?	☐	☐	☐☐ år
Har du vært innlagt på sykehus med hjerteinfarkt eller hjertekrampe (angina)?	☐	☐	☐☐ år
Har lege sagt at du har hjertesvikt (svakt hjerte, vann i lungene, hovne ben)?	☐	☐	☐☐ år

70 Har du, eller har du noen gang hatt noen av disse sykdommene/plagene? (Kryss enten nei eller ja på alle spørsmålene)

	Nei	Ja	**Hvis JA**, alder første gang?
Hjerneslag/ hjerneblødning?	☐	☐	☐☐ år
Hjerteflimmer?	☐	☐	☐☐ år
Eksem på hendene (med unntak av psoriasis)?	☐	☐	☐☐ år
Annen kronisk lungesykdom enn astma eller KOLS?	☐	☐	☐☐ år
Psykiske plager som du har søkt hjelp for?	☐	☐	☐☐ år
Sure oppstøt	☐	☐	☐☐ år

Tusen takk for ditt bidrag!

MER INFORMASJON OM STUDIEN OG FORESPØRSEL OM DELTAKELSE I HELSE-FORSKNINGSPROSJEKTET TELEMARKSTUDIEN

Dette er et en forespørsel til deg om å delta i en stor befolkningsundersøkelse i Telemark. Telemarkstudien er et helseforskningsprosjekt. Tidligere het studien Astma i Telemark, men har nå byttet navn til Telemarkstudien. Vi spør om forskjellige sykdommer og plager, men har et særlig fokus på allergi, astma og KOLS. Målet er å undersøke hvordan det vi utsettes for i arbeid og miljø påvirker helse og livskvalitet. Denne kunnskapen bidrar til å hindre at sykdom oppstår og gir bedre veiledning og behandling til de som er syke. Spørreundersøkelsen vil gi mer kunnskap jo flere som svarer. Ditt svar er like viktig, enten du er frisk eller syk. Vi ber deg om å svare så godt du kan, selv om noen av spørsmålene kan være litt vanskelige. Det tar ca. 20 minutter å fylle ut skjemaet. Du kan velge om du vil svare på nett (elektronisk spørreskjema) eller returnere papirskjema i vedlagte frankerte konvolutt.

Spørreskjemaet ble også sendt ut i 2013 til 50.000 tilfeldig utvalgte innbyggere i Telemark i alderen 16-50 år. **Du var en av de som svarte.** Vi følger nå opp med en ny spørreundersøkelse, blant annet for å se om det er noen endringer i helsen din eller i det du påvirkes av i arbeidet eller i miljøet. Vi vil denne gangen også sende spørreskjemaet til en del av de innbyggerne i Telemark som ikke fikk forespørsel om deltakelse i 2013. Noen av resultatene fra forrige undersøkelse er omtalt foran i spørreskjemaet.

Prosjektet gjennomføres av Sykehuset Telemark i samarbeid med Universitetet i Oslo.

For mer utfyllende informasjon om undersøkelsen, se vår nettside www.sthf.no/Telemarkstudien.
Dersom du har spørsmål til spørreundersøkelsen kan du ringe, sende melding eller e-post til en av prosjektmedarbeiderne ved avdeling for arbeidsmedisin, Sykehuset Telemark, tlf: 953 69 315
e-post: astma@sthf.no

HVA INNEBÆRER PROSJEKTET?
Denne delen av Telemarkstudien bygger på svar på spørreskjemaene som returneres. Vi bruker internasjonalt anerkjente spørsmål for å kartlegge forekomst av sykdom og symptomer og eksponeringer i og utenfor arbeid.

I prosjektet vil vi innhente og registrere opplysninger om deg som så avidentifiseres (se mer informasjon nedenfor). Ved å samtykke i studien samtykker du også til at opplysningene fra spørreskjemaet kan kobles med opplysninger fra Folkeregisteret, Reseptregisteret, de nasjonale kvalitetsregistrene, Dødsårsaksregisteret, FD-trygd og KUHR (Kontroll og Utbetaling av HelseRefusjoner fra behandlere og helseinstitusjoner til staten).

MULIGE FORDELER OG ULEMPER
Fordeler ved deltakelse i studien: Det er viktig å vite noe om hvor mange som har plager fra luftveiene og som utvikler luftveissykdom. Enda viktigere er det å vite hvilke faktorer i og utenfor arbeid som kan gi plager fra luftveiene for å kunne forebygge sykdom i fremtiden. Mer kunnskap kan også være med å hindre forverring hos de som allerede har plager. Dette kan du bidra til ved å delta i studien. I tillegg vil studien også kunne være til hjelp for politikere og helsepersonell bedre å kunne planlegge og gjennomføre nødvendige tiltak i Telemark og landet for øvrig, for å møte de behov og kostnader som følger av luftveissykdommer.
Mulige ulemper ved deltakelse i studien: Det hender at noen deltagere blir bekymret av å fylle ut et spørreskjema. Hvis dette gjelder deg, er vi tilgjengelige på telefon for å kunne svare på spørsmål og gi råd.

FRIVILLIG DELTAKELSE OG MULIGHET FOR Å TREKKE SITT SAMTYKKE
Deltakelse i prosjektet er frivillig og du kan trekke deg når som helst uten å oppgi noen grunn. Dersom du senere ønsker å trekke deg, vil alle opplysninger om deg bli slettet. Dersom du ønsker å delta, besvarer du spørreskjemaet og returnerer det i vedlagte ferdig frankerte svarkonvolutt. Ved å sende tilbake spørreskjemaet samtykker du til å delta i studien.

Dersom du skulle forandre mening og ønske å trekke deg fra prosjektet, kan du kreve å få slettet innsamlede prøver og opplysninger, med mindre opplysningene allerede er inngått i analyser eller brukt i vitenskapelige publikasjoner. Dersom du senere ønsker å trekke deg eller har spørsmål til prosjektet, kan du kontakte en av prosjektmedarbeiderne på Sykehuset Telemark, tlf: 953 69 315 (kl. 08.00-15.00).

HVA SKJER MED INFORMASJONEN OM DEG?
Informasjonen som registreres om deg skal kun brukes slik som beskrevet i hensikten med studien. Du har rett til innsyn i hvilke opplysninger som er registrert om deg og rett til å få korrigert eventuelle feil i de opplysningene som er registrert.

Alle opplysningene vil bli behandlet uten navn og fødselsnummer eller andre direkte gjenkjennende opplysninger. Opplysningene er da avidentifiserte. En kode knytter deg til dine opplysninger gjennom en navneliste som kun et lite antall prosjektmedarbeidere ved Sykehuset Telemark HF har tilgang til. Ved publisering av resultatene vil alle opplysninger være anonymisert.

Opplysningene behandles konfidensielt. Den tekniske gjennomføringen av nettskjema undersøkelsen foretas av Norsk Senter for Forskningsdata (NSD). Forskerne får utlevert data fra NSD uten tilknytning til e-post/IP-adresse.

Prosjektleder har ansvar for den daglige driften av forskningsprosjektet og at opplysninger om deg blir behandlet på en sikker måte. Opplysninger som registreres om deg vil bli oppbevart i låste arkiver og i datasystem som er beskyttet av Sykehuset Telemarks IT-rutiner. Sykehuset Telemark HF ved administrerende direktør er databehandlingsansvarlig. Informasjon om deg vil bli anonymisert eller slettet senest fem år etter prosjektslutt i 2035.

FORSIKRING
Alle deltakerne i studien er forsikret gjennom Norsk pasientskadeerstatning.

UTLEVERING AV OPPLYSNINGER TIL ANDRE
Ved å delta i prosjektet, samtykker du også til at avidentifiserte opplysninger kan utleveres til prosjektmedarbeidere tilknyttet prosjektet ved Sykehuset Telemark HF og Oslo Universitetssykehus HF. Du samtykker også til at avidentifiserte opplysninger kan sendes til våre samarbeidspartnere i Sverige og USA. Dette kan være land med lover som ikke tilfredsstiller europeisk personvernlovgivning. Koden som knytter deg til dine personidentifiserende opplysninger, vil ikke bli utlevert.

OPPFØLGINGSPROSJEKT
Vi vil gjennomføre en non-responder studie der vi kontakter noen av de som ikke har svart på spørreskjemaet med noen få enkle spørsmål blant annet for å se om vi får en skjevfordeling av svarene. Det er viktig å være sikre på at de svarene vi får er representative.

I 2018 og 2019 vil vi invitere en gruppe utvalgte personer som har svart på spørreundersøkelsen til en forskningspoliklinikk der vil vi gjøre ytterligere undersøkelser for eksempel blodprøver og pusteprøver. De som blir innkalt vil få mer opplysninger om undersøkelsen.

Informasjon om utfallet av studien: Resultatene av prosjektet vil bli formidlet gjennom offentlige medier lokalt og nasjonalt og gjennom vitenskapelige artikler internasjonalt. Du vil finne oppdatert informasjon om prosjektet på prosjektets hjemmeside: www.sthf.no/Telemarkstudien

ØKONOMI
Studien er finansiert gjennom forskningsmidler fra Sykehuset Telemark HF.

GODKJENNING
Prosjektet er godkjent av Regional komite for medisinsk og helsefaglig forskningsetikk, saksnr. hos REK (2012/1665).