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Experimental studies of exposure to cooking fumes, and epidemiological investigations in a cohort

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Doctoral thesis

NTNU
Norwegian University of Science and Technology

Thesis for the Degree of
Philosophiae Doctor

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Kokker, arbeidsmiljø og helse

-Eksperimentelle studier av eksponering for stekeos, og epidemiologiske undersøkelser i en kohort

Allerede i de Hippokratiske tekstene fra omtrent 400 år f. kr., fremholdes det at frisk luft og en sunn diett er blant de viktigste forutsetningene for god helse. Nå, over 2 400 år senere kan vi lese på nettsidene til verdens helseorganisasjon (WHO) at omtrent 7 millioner for tidlige dødsfall årlig kan knyttes til luftforurensning, og at luftforurensning er den miljøfaktoren som har størst betydning for sykdomsbelastningen på verdensbasis. WHO sine beregninger har også vist at innendørs luftforurensning har omtrent like store negative konsekvenser for folkehelsen som luftforurensning i ytre miljø. Samtidig som sunn mat er viktig for god helse, så er tilbereding av mat anerkjent som en av de viktigste kildene til innendørs forurensning. En relativt ny studie av et utvalg av den urbane befolkningen i storbyer i Europa og Australia, viste at stekeos sannsynligvis er den viktigste kilden til forurensninger som påvirker luftveiene. Stekeos består blant annet av fett og fettsyrer i dråper som er så små at de kan pustes helt ned i lungene. I de siste 10-20 årene har det vært økende interesse knyttet til spesifikke helseskadelige og kreftfremkallende stoffer som kan finnes i stekeos, slik som blant annet aldehyder, polysykliske aromatiske hydrokarboner (PAH) og heterosykliske aminer.

Tidligere studier har vist en økt forekomst av ulike typer luftveisplager, for eksempel rhinitt, astma og emfysem, blant personer som daglig utsettes for stekeos. Det er derfor grunn til å tro at innendørs luftforurensning fra matlaging kan bidra til å forårsake og/eller forverre luftveissykdommer, særlig hos kokker som daglig arbeider med steking av mat. Flere europeiske undersøkelser har vist økt dødelighet blant kokker, sammenholdt med befolkningen forøvrig. Tall fra statistisk sentralbyrå har vist at kokker er en av yrkesgruppene i Norge som har lavest forventet levealder.

I dette prosjektet har vi både undersøkt mulige effekter av kortvarig eksponering for stekeos hos 24 frivillige forsøkspersoner, og samlet informasjon omkring arbeidsmiljøforhold og helse hos kokker i Midt-Norge. Informasjonen vi fikk fra kokkene som deltok i våre epidemiologiske undersøkelser, viste at ulike arbeidsmiljøfaktorer som kan gi økt eksponering for stekeos, er assosiert med en økt forekomst av luftveisplager. Det viste seg også at ugunstig arbeidstidsordninger var den vanligste angitte grunnen til å slutte i kokkeyrket. I de eksperimentelle undersøkelsene var det også enkelte tendenser i resultatene som kunne tyde på en reaksjon i luftveiene etter eksponering for stekeos, men dette var basert på få deltakere og er beheftet med en del usikkerhet, slik at disse mønstrene kan også ha oppstått som følge av tilfeldigheter.

Samlet sett tyder våre undersøkelser på at yrkeseksponering for stekeos medfører økt risiko for luftveisplager, samtidig som organisatoriske arbeidsmiljøforhold ser ut til å ha større innvirkning på hvor lenge en kokk velger å forbli i yrket.

Våre funn støtter opp under at det å redusere eksponeringen for stekeos så mye som mulig vil kunne forebygge luftveisplager hos kokker. Sett i sammenheng med eksisterende kunnskap, vil vi hevde at å forebygge eksponering for stekeos er viktig for å oppnå sunne arbeidsmiljø i profesjonelle kjøkken, noe som på lang sikt kan bidra til å motvirke den økte sykelighet og dødelighet som har vært observert blant kokker.

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List of papers

The original articles included in this thesis are listed below, and referred to by their roman numbers throughout the thesis.

Paper I

Svedahl S, Svendsen K, Qvenild T, Sjaastad AK, Hilt B. **Short term exposure to cooking fumes and pulmonary function.** *J Occup Med Toxicol*. 2009;4:9.

Paper II

Svedahl SR, Svendsen K, Tufvesson E, Romundstad PR, Sjaastad AK, Qvenild T, Hilt B. **Inflammatory Markers in Blood and Exhaled Air after Short-Term Exposure to Cooking Fumes.** *Ann Occup Hyg* (2013) 57 (2): 230-239

Paper III

Svedahl SR, Svendsen K, Romundstad PR, Qvenild T, Strømholm T, Aas O, Hilt B. **Work environment factors and work sustainability in Norwegian cooks.** *Int J Occup Med Environ Health* 2016;29(1):41–53.

Paper IV

Svedahl, SR, Hilt, B, Svendsen K. **Work environment factors and respiratory complaints in Norwegian Cooks.** *Submitted.*

Abbreviations

95% CI	95% confidence intervals
CO	Carbon monoxide
CO2	Carbon dioxide
COPD	Chronic obstructive pulmonary disease
EBC	Exhaled breath condensate
Exhaled NO	Exhaled Nitrogen Oxide
FET	Forced expiratory time
FEV1	Forced expiratory volume in 1 second
FEF_{25-75}	Forced expiratory flow from 25-75% of FVC
FVC	Forced vital capacity
IARC	International Agency for Research on Cancer
IL-1β,	Interleukin-1beta
IL-6	Interleukin-6
LTB4	Leukotriene B4
NO	Nitrogen monoxide
NO2	Nitrogen dioxide
NTNU	Norwegian University of Science and Technology
OR	Odds ratio
PAH	Polycyclic aromatic hydrocarbons
PEF	Peak expiratory flow
PM2.5	Fine particulate matter (diameter < 2.5 micrometres)
ppb	Parts per billion
SHR	Standard hospitalization ratio
SIR	Standardized incidence ratio
SMR	Standardized mortality ratio
TVOC	Total volatile compounds
WHO	World Health Organization

Contents

1 Introduction

"*Eating is a necessity, but cooking is an art*" – unknown.
Providing and consuming food has been an essential part of human living everywhere and throughout time. Activities related to this have presumably engaged people from the origin of the first civilizations. Little is known about the work environment and work techniques used by the first professional cooks. Ancient texts from Mesopotamia, dating approximately 4000 years ago, describe different recipes for advanced food preparation, including cooking all sorts of meats and adding different types of herbs (1). Although many other occupational groups with a shorter history have been described and investigated quite thoroughly, it seems that up until recently, little attention has been given to the work environment and occupational health of professional cooks.

Decennial cross-sectional studies of occupational mortality in England and Wales have been published since the mid-1880s and up until 1990. These and similar reports from other regions might have inspired some of the early studies on the mortality of cooks. In a publication on mortality of British Army cooks from 1993, the author refers to four reports of occupational mortality based on statistics from censuses in Great Britain and Denmark performed between 1971 and 1985, that showed indications of a high rate of lung cancer in cooks (2). Similar results were also reported from concurrent statistics in Norway and Sweden, and gave rise to the question of whether cooks might be exposed to occupational carcinogen(s) (3). A study published in 1986 that explored the occupational and smoking history of cancer patients in three English counties also found an excess of lung cancers in cooks. The authors then stated that "theoretic explanations for the risk include occupational exposure to polycyclic aromatic hydrocarbons or nitrosamines in fumes" (4). An increasing interest in cooks´ chemical work environment emerged in the following years, and, carcinogenic and mutagenic substances, such as polycyclic aromatic hydrocarbons (PAHs), heterocyclic amines and mutagenic aldehydes are some of the ingredients that have been characterized in cooking fumes (5-12). In the early 1990´s, it was hypothesized that occupational

exposure to volatile carcinogenic compounds formed during the cooking process could contribute to the excess mortality of some cancers among cooks (13). The focus on the harmful contents of cooking fumes was probably further stimulated in 1999 and thereafter by the publication of Asian investigations that showed indications of high rates of lung cancer in non-smoking women exposed to cooking fumes (14-19).

Airway irritants and toxic substances found in private kitchens and restaurant kitchens have also been shown to lead to other respiratory complaints such as lipid pneumonia, allergic rhinitis and increased incidence of productive cough (20-24). There are also research findings indicating that cooking fumes can affect the lung function of exposed subjects (21, 22, 25). In addition to these effects on the respiratory system, an association between cooks' working environment and cardiovascular disease has also been reported (26, 27).

Concurrent with the expanding insight into the potential health hazards from exposure to cooking fumes, the burden of disease caused by exposure to air pollution in general has also received increasing attention. Exposure to inhalable air pollution has been known as an important cause of respiratory diseases, but it is increasingly also recognized to significantly contribute to morbidity from other diseases, such as cardiovascular diseases and cancer, and is currently considered to be the world’s largest single environmental health risk (28, 29). The World Health Organization (WHO) estimates published in 2014, showed that indoor air pollution contributes more to the global burden of disease than ambient air pollution, mainly due to indoor air pollution in households using solid fuels for cooking in low and middle income countries (28). Cooking activities have also been linked to negative health effects without the use of solid fuels, and in 2010, emissions from high temperature frying was categorized as probably carcinogenic to humans by the International Agency for Research on Cancer (IARC) (30). The impact of indoor air pollution on human health and well-being in developed countries has also gained more attention in recent years, probably due to the fact that urban inhabitants in developed countries spend typically 90% of their time

indoors. Further, cooking fumes are considered to be one of the most important sources of indoor air pollution in developed countries (31, 32), and is readily present in the cooks' work environment. As mentioned above, the chemical content of cooking fumes have been characterized in many studies, revealing good reasons for anticipating negative health effects for exposed groups.

A Norwegian report on "The workday of the cook" [Kokkens Hverdag], published in 2006, describes a profession with a high pace, shift work with unfavourable working hours, competition and somehow unhealthy lifestyles (33). The report presents estimates suggesting that there is a high turnover in the profession and that on average, a cook works for only about 6.5 years after completing a professional exam. They also found that more than half of the cooks who were asked would prefer another job. Moreover, high work load and shift work are factors that can be associated with increased turnover and health problems compared to other professions. Other possible causes of dropout are muscle and skeletal disorders that may be associated with factors in the physical environment (34, 35) and increased morbidity and mortality.

The new insight from occupational mortality studies in cooks based on national censuses and other epidemiologic studies from Europe and the USA published during the 1990´s, together with early publications on morbidity in cooks and air pollution in the cooks' work environment, might be important reasons why increasing interest has been given to the work environment and occupational health of cooks over recent decades. There are still a lot of unanswered questions regarding both morbidity and mortality in cooks, and probably also important questions that have not yet been raised. Although the extent of related literature is still limited, there have been some interesting publications during recent years. A selection of the more recent literature, as well as some of the relevant early publications, will be presented in more detail in the following paragraphs. Characterization of cooking fumes was the main topic in another PhD-thesis from our research group: "Exposure to cooking fumes during the pan frying of beefsteak under domestic and occupational conditions" (36); thus, this topic will only be briefly referred to.

1.1 Epidemiological studies of mortality among cooks

The above mentioned study on British Army cooks were performed on a cohort of 1,798 cooks who had retired from the British Army Catering Corps (2). The authors calculated standardized mortality ratios (SMR) that were significantly increased for the cooks compared to the national population for lung cancer (SMR 1.82), large intestine cancer (SMR 3.03), ischemic heart disease (SMR 1.42), cerebrovascular disease (SMR 2.05), and digestive disease (SMR 2.27). The authors concluded that "The high rate of lung cancer among the cooks supports the hypothesis of an occupational hazard, although at least part of the excess was probably due to smoking." The year before, an excess mortality due to oral, pharyngeal and laryngeal cancer was reported among cooks in a study based on Swiss mortality data from 1979-1987 (13). The authors acknowledge combined tobacco and alcohol consumption as dominant risk factors of such cancers, but also suggest that occupational exposure to volatile carcinogenic compounds formed during the cooking process may contribute to the excess mortality.

Increased mortality among cooks from cancers of the oral cavity (SMR 5.57) and pharynx (SMR 2.66) was also found in a study on the "Mortality of butchers and cooks identified from the 1961 census of England and Wales," which was published in 1995. However, in contrast to previous studies, this study found no increase in mortality from lung cancers among cooks, and the mortality from all causes was significantly lower than in the national population (SMR 0.89, 95% CI 0.84-0.95).

A high general mortality among cooks was found by a study published in 1999, comparing the mortality differences for specific and general categories of occupations using a national cohort of approximately 380,000 persons, aged 25-64, from the U.S. National Longitudinal Mortality Study (37). Some of their important findings were that specific occupational exposures are more important than the social status grouping in describing the effects of occupation on mortality, and that cooks and other specific occupations had an increased mortality beyond what was explained by social status, income, and education.

A study by Statistics Norway that included all persons 25 to 65 years of age and investigated mortality by occupation between 1960-2000 showed that cooks had one of the lowest life expectancies of all occupations in Norway for both males and females (38). Disease-specific mortality was analysed for groups of occupations. Cooks and kitchen managers accounted for 54 - 66% of the subjects in the occupational group "male hotel and restaurant workers and waiters" who had the second highest mortality rate of all occupational groups, next to male seamen. The mortality was high for both cardiovascular disease and cancer, and in particular for respiratory diseases and diseases of the digestive system. It was shown that female cooks had a lower increase in mortality than male cooks. The distribution of the type of workplaces was also different; the female cooks worked mostly in kitchens for institutions, while the male cooks, to a larger extent, worked at restaurants.

An increased mortality among cooks in England and Wales from cancers of the oral cavity and pharynx was reported in 2010 (39). This study focused on the occupational differences in mortality from some selected diseases that are considered to be related to alcohol, drugs and sexual habits. Data from all deaths at ages 16-74 years during 1991-2000 was retrieved from national registers, and deaths from the selected diseases were linked to the last full-time occupation of the decedent. The analysis showed that cooks had an increased mortality from liver cancer, and some other alcohol related diseases. As such, the authors' hypothesized that the cooks might have had high alcohol consumption due to easy access.

1.2 Studies of morbidity among cooks

There are several studies that have investigated different aspects of occupational morbidity among cooks. The following paragraphs are focused on the existing literature on cancer and respiratory health, which are health areas where inhalable air pollution is acknowledged as a significant risk factor. The literature regarding musculoskeletal complaints, mental health and work-place injuries are omitted, since it is not within the primary scope of this thesis. The literature regarding cardiovascular diseases is also left

out, but some of it is briefly referred to towards the end of Chapter 1.4.1, “Measuring inflammatory markers.”

1.2.1 Occurrence of cancer in cooks

The studies that have investigated cancer risk in cooks, show conflicting results. Some authors have claimed that an observed excess risk is mainly due to lifestyle factors like smoking and alcohol consumption. Others have interpreted their findings as indications of an occupational exposure to carcinogens in cooks. Due to such interesting variations, it does not seem appropriate to try to summarize a main tendency, or overall interpretation of the results from these different studies. The reported occurrence of specific cancers can, however, help to shed some light on exposure and health outcomes in the occupation, and are important source of information, especially since there is a limited amount of other publications on morbidity in cooks. Therefore, a brief review of studies that have addressed cancer in cooks is included in the following paragraphs, even though this thesis does not address cancer as an outcome.

As previously mentioned, the retrospective investigation published in 1986 that compared the occupational and smoking history of cancer patients from three countiesin England, showed indications of an excess risk of lung cancer in cooks (4). The study population was men, aged 18-54, diagnosed with cancer between 1975-1980, and were identified by hospitals and cancer registries. The information on occupational and smoking history was retrieved from the patient or the next of kin. From a total of 2,942 cancer records, the overall response rate was 52.1 %. When each specific cancer diagnosis was compared to all other cancers combined, lung cancer was associated with having worked as a cook, with an estimated relative risk of 2.5, (95 % C.I.: 1.2-2.9).

In 1993, a study of cancer incidence in two Norwegian cohorts of 1,463 male waiters and 2,582 male cooks was published (40). The cohorts were followed from 1956 through 1991, and the standardized incidence ratio (SIR) for all causes of cancers was

1.4 for waiters and 1.1 for cooks. An excess risk of cancers of the upper aero-gastric tract and the liver, which has alcohol consumption as a common risk factor, was found for both cooks (SIR: 4.2) and waiters (SIR: 5.1). The study did not have data on the smoking habits or alcohol consumption, but such information was obtained subsequently by a questionnaire sent to cooks and waiters organized in the Hotel and Restaurant Workers' Union. The data indicated a higher average alcohol and tobacco consumption among waiters than in the general population. For cooks, the data showed indications of lower than average alcohol consumption for the oldest age group, and marginally higher levels for the younger age groups. Thus, the authors state that "for cooks, the data indicates a much smaller excess of alcohol consumption compared to the general population, as well as more similar smoking habits." An excess of lung cancer was found among waiters (SIR: 2.0) and not cooks. The study presents further analyses on the data stratified both by region and by time in the occupation, and for some specific cancer sites. Some of the analyses of the subgroups included a low number of observations, and the cooks of the cohort were relatively young. The authors recognize that this affects the stability of some of the estimates, and that further follow-up may be needed to verify the lack of an increase in lung cancer among cooks.

A study investigating cancer incidence by occupation from 1970-1990 in the Nordic population was published in 1999 (41). For the occupational group of cooks and stewards, it showed an increased incidence of lung cancer (SIR 1.43 in men, 1.17 in women), and bladder cancer (SIR 1.31 in men, 1.18 in women). The men in this group also showed an increased incidence for cancers of the mouth, pharynx, oesophagus, liver, pancreas, larynx, breast, prostate, and unknown sites. The women showed an increase in cancers of the cervix and ovary. In a later study, it was attempted to adjust the lung cancer incidence of men for smoking (42). This was done by retrieving information on smoking habits by occupation from national surveys. Some of the occupations were combined into "smoking groups" due to the small numbers on smoking habits for each occupation. For the estimation of smoking habits, cooks and stewards were grouped together with "home helpers" and waiters. This adjustment for smoking in cooks and stewards changed the SIR for lung cancer from 1.48 to 1.00, when assuming that cooks and stewards smoked as much as waiters and home helpers.

Surveys on smoking habits presented in the study on cancers in waiters and cooks mentioned above indicated, however, that waiters smoked more than the general population, while cooks did not (40).

In a Swiss study that was published in 2002 (43), the data was collected from cancer registers that covered approximately 40 % of the population, with information on cancer site and last occupation. The analysis of cancer incidence in the different occupations between 1980-1993 was performed by using all other cancers as a control group for the investigated cancer type. An increased incidence of cancers of the oral cavity and oropharynx, oesophagus and liver was found in cooks.

A case-control study from Sweden that was published in 2006 investigated the association between occupational exposure to inhalable air pollution and risk of cancer of the oesophagus and gastric cardia based on the hypothesis that airborne chemicals or particles might be captured in the airways and swallowed to act as carcinogens directly on the oesophageal or cardia mucosa (44). A total of 558 cases and 820 matched controls were interviewed, and their occupational exposures assessed. Variations by occupational groups were shown for both cardia adenocarcinomas, and for oesophageal squamous cell carcinoma. A tendency of an increased risk of oesophageal squamous cell carcinoma was found in the workers that were assessed to have been highly exposed to particular agents. Furthermore, increased risk of oesophageal squamous cell carcinoma was found in the occupational groups of “concrete and construction work” (odds ratio (OR): 2.2, based on 13 cases), “food and tobacco processing work” (OR: 5.1, based on 5 cases) and “hotel and restaurant work” (OR: 3.9, based on 2 cases).

A study with up to 45 years of cancer incidence data by occupation category for the Nordic population, including 15 million subjects, was published in 2009 (45). For the occupational category of cooks and stewards, the study showed an increased incidence of cancer of all sites combined, with SIR (presented as 95% confidence interval) for men (1.16 – 1.23) and for women (1.01 – 1.04). Specific cancer sites with increased

incidence for both men and women were larynx (SIR for men: 1.84 – 2.77, women: 1.09 – 2.24), lung (SIR for men: 1.44 – 1.68, women: 1.05 – 1.21) and bladder/ureter/urethra (SIR for men: 1.08 – 1.35, women: 1.00 – 1.22). The men in this group also showed increased incidences for cancers of the tongue (SIR: 1.83 – 3.49), oral cavity (SIR: 2.34 – 3.88), pharynx (SIR: 2.07 – 3.29), oesophagus (SIR: 1.72 – 2.60), liver (SIR: 2.09 – 3.19), gallbladder (SIR: 1.03 – 2.27), pancreas (SIR: 1.13 – 1.57), kidney (SIR: 1.06 – 1.45) and unknown sites (SIR: 1.16 – 1.23). The women showed increased incidences in cancers of the cervix (SIR: 1.16 – 1.37) and the ovaries (SIR: 1.06 – 1.20). Much of these results were quite similar to the findings from previously published 20 year follow up studies on cancer incidence in the same population, referred to above (41). Alcohol and smoking habits are important but unfortunately, were unmeasured confounders in these studies. In an additional publication based on the same data, the authors underline that alcohol and smoking are the most important risk factors for some cancer sites, such as oral/pharyngeal, oesophageal, and laryngeal cancer. Further, they stated that "most of the variation in risk at these cancer sites across occupations can probably be explained by smoking and drinking habits" (46).

In 2015, no increased risk of lung cancer overall was found in cooks after adjusting for smoking in a pooled analysis of 16 case-control studies that addressed lung cancer, and included information on lifetime work histories and smoking habits (47). A total of 1242 cooks, 554 of the 19,370 cases and 588 of the 23,674 controls, were detected from the pooled data of 16 case-control studies conducted in Europe, Canada, New Zealand, and China. An increased risk of lung cancer was found in male cooks before, but not after adjusting for smoking. The smoking habits were found to be similar in cooks, and "noncooks". In the control group, 63 % of the cooks were current or former smokers, compared to 68 % of the "noncooks", while the number of pack years for ever-smokers was 29 years in the cooks, and 26 years in the "noncooks". After adjusting for smoking, no overall increased risk was found, except from in a sub analysis that indicated an excess risk of some types of lung cancer in female cooks. Among other explanations, the authors suggested the observed increased risk of squamous cell carcinoma, and a possible increased risk of small cell carcinoma, indicated "the possibility of a joint effect with smoking, as well as residual confounding from smoking or confounding

because of environmental tobacco smoke." Further, the authors speculated that "women may be more sensitive than men to carcinogens in cooking fumes or that the exposure situation in the kitchen may differ between men and women." They emphasized the need for cautious interpretation of these subgroup analyses, and the main message was that cancer risk in cooks may be confounded by smoking.

The above given examples of the literature on cancer by occupation, shows some of the variation in the estimates and interpretations of cancer risk in cooks. An excess of cancers is reported by several of the studies, while highlighting the lack of data on, and possible confounding from lifestyle factors. Another important limitation for most of the existing literature seems, however, to be the lack of information on specific work environment factors.

1.2.2 Respiratory health in cooks

Although not in abundance, there are some interesting publications on different aspects on the respiratory health of cooks other than cancers, ranging from epidemiological investigations to case reports. Some of the available literature is presented chronologically in the following paragraphs.

In a Danish study published in 2000, an increased risk of hospitalization due to COPD was found in those employed in the hotel and restaurant industry, and their standard hospitalization ratio were 140 for men, and 141 for women (48). All employed persons in the industry were included in this group, and there was no presentation of separate data for waiters, cooks or other occupations. A similar study was published in 2002, this time analysing the rate of hospitalization for various diseases among employees in the Danish hotel and restaurant industry compared to this rate among all gainfully employed people in Denmark. The authors state that "standard hospitalization ratios with respect to diseases in practically all systems and organs of the body were higher

among employees in the hotel and restaurant industry than in the working population of Denmark at large."

In a cross-sectional study published in 2003, kitchen workers from 67 kitchens in central Norway exhibited increased prevalence of dyspnoea and respiratory symptoms at work, compared to a control group of 1,500 persons from the same area (49).

Further, a cross sectional investigation of lung function in 37 cooks in four hospital kitchens in Brazil was published in 2007. It showed indications that working as a cook and nitrogen dioxide (NO_2) concentration in the kitchen was correlated with decreased forced expiratory volume in the first second (FEV_1) and forced expiratory flow in 25-75% of forced vital capacity (FEF_{25-75}) compared to expected values (21). The authors further claim that it can be estimated from their results that "ten years of work as a cook may result in 20 and 30 % decreases in the predicted values of these two lung function indicators, respectively."

A link between air pollutants in the work environment and reduction in FEV_1 was also found in a study of respiratory health and lung function in 393 restaurant workers in 53 Chinese restaurants, published in 2011 (50). Respiratory symptoms were surveyed and lung function was tested among 115 workers from restaurants using only electric stoves and compared to 278 workers from restaurants using only gas stoves. Measured levels of air pollutants were highest in the kitchens using gas stoves, and the workers in these kitchens had lower mean FEV_1 and forced vital capacity (FVC) than those working in kitchens using electric stoves. The only statistically significant differences in the occurrence of respiratory symptoms between the two groups were an increased odds ratio of 2.7 for having regular phlegm in those working at kitchens using gas. Some of the reported prevalence of respiratory symptoms in that group was: wheeze 3.6 %, phlegm 9.9 %, and regular cough 6.5 %. Compared to the kitchens using electric stoves, the average concentration of total volatile organic compounds (TVOC) and particulate matter (PM2.5) were 81% and 78 % higher, in the kitchens using gas, while the median concentration of nitrogen monoxide (NO), NO2 and carbon dioxide (CO2) were 7.4, 1.5 and 1.6 times higher.

From Nigeria a cross sectional study of workers exposed to both wood smoke and cooking fumes was published in 2013 (51). The study surveyed respiratory symptoms and measured lung function in 48 males that worked with roasting fresh meat over open fire, and in a control group of 32; age and sex matched controls without any work-related exposure to wood-smoke and/or cooking fumes. Comparisons between the groups were then made for the non-smokers. Compared to the control group, the exposed workers had a lower mean FEV1, and increased occurrence of chest tightness: 59%, nasal congestion: 37%, and wheeze: 12%, (95 % confidence intervals of the odds ratios ranged from 1.0 – 5.8).

An increased risk of respiratory work disability was shown for cooks and six other occupations in a Norwegian case control study published in 2016 (52). A total of 16,099 responders from a random sample of inhabitants aged 15-50 in Telemark County in Norway, were asked about occupational exposures and respiratory complaints. Respiratory work disability was defined as responding that they ever had to change or leave their job because it affected their breathing. Based on a subset of 125 responders that had provided year and occupation at such job change, and 8,352 controls with "occupation code," the odds ratio for respiratory work disability in cooks was estimated to 3.6 (95% C.I. 1.6 – 8.0) after adjusting for age, gender and smoking.

Increased prevalence of respiratory symptoms in kitchen and restaurant workers was also found in recent cross-sectional studies from Thailand and Iran. The study from Thailand used questionnaires to obtain data on exposure to kitchen air pollutants and respiratory symptoms in 224 workers from 142 restaurants and 395 controls (53). Compared to the control group, restaurant workers had a higher risk of several respiratory symptoms within the past 30 days, as well as chronic symptoms. The prevalence of chronic respiratory symptoms in the restaurant workers were: dyspnoea (males 30.7 %, females 52.3 %), stuffy nose (males 48.0 %, females 45.8 %), cough (males 16.3 %, females 32.5 %), wheeze (males 38.7 %, females 25.5 %), and phlegm (males 25.3 %, females 14.4 %). Hours working in the kitchen, the number of fried

dishes prepared per week, and experiencing tears while cooking where factors found to predict most of the respiratory symptoms.

The study from Iran showed cross-shift decrease in lung function in a group of 60 kitchen workers employed at two universities (54). Compared to 60 controls selected by "simple random sampling" among office-workers from one of the universities, these kitchen workers also exhibited increased occurrence of respiratory symptoms with odds ratio for the different specific symptoms ranging from 3 - 27. This comparison was, however, hampered by that the controls had higher levels of education. The prevalence of respiratory symptoms in the kitchen workers were: wheezing 20 %, shortness of breath 63 %, chest tightness 32 %, cough 28 %, chronic cough 15 %, phlegm 33 %, and chronic bronchitis 20 %.

A wide range of approaches towards investigating respiratory health of cooks and kitchen workers in various settings are shown in the literature referred to above. The characterization of exposure and outcomes varies substantially, and furthermore, some of the studies have quite small numbers. Several studies relied mainly on self-reported information on work environment factors, confounders, and outcomes, while some also included measured information, such as measured air pollutants in the kitchens, and measured lung function. In addition, the referred studies have been conducted in diverse regions, under varying circumstances. An overall interpretation of the existing evidence on the respiratory health of cooks is limited by these differences between the studies, as well as the moderate amount of available literature. It seems, however, that it would be fair to say that the literature so far show that occupational exposures can influence the respiratory health of cooks.

1.3 Exposure to cooking fumes

Cooks work in various types of restaurants and institutions, such as canteens, which are often combined with kitchens at hospitals and homes for elderly, along with fast food kitchens, food factories, catering businesses, and grocery stores. This entails many different tasks and work related exposures. Albeit the many differences, all the various

ways of working as a cook share the main purpose of processing and preparing foodstuffs to make them more valuable and suitable for consumption.

In contrast to the pragmatic description above, there are numerous quotations describing the essence of cooking in more poetic terms. The following was written in a cookbook originally published in 1984: “Whenever we cook, we become practical chemists, drawing on the accumulated knowledge of generations, and transforming what the Earth offers us into more concentrated forms of pleasure and nourishment.” – Harold McGee 1984.

This thesis focuses on the impact of specific work environment factors on the health and work sustainability of cooks, with a main focus on the effects of exposure to cooking fumes. As the exposure to cooking fumes has been described in another thesis from our research group (36), the work related exposures in professional kitchens are only referred to in an arbitrary manner in these thesis. The following section refers briefly to some of the literature on the chemical components of cooking fumes, while some of the other relevant work environment factors are mentioned in the general introduction.

When food is cooked at temperatures up to 300^{o}C, carbohydrates, proteins, and fat are reduced to toxic products, such as aldehydes and alkanoic acids (7, 55-57), which can cause irritation of the airway mucosa (58-61). Cooking fumes also contain carcinogenic and mutagenic compounds, such as polycyclic aromatic hydrocarbons and heterocyclic compounds (5, 7, 55, 56, 62-65). Frying at high temperatures also produces aerosols of fat with small aerodynamic diameters of 20-500 nm, which disperse in the air of the kitchen and nearby facilities. Such aerosols, containing fatty acids, irritate the airway mucosa and can cause pneumonia (66-68). The content, concentration and dispersion of cooking fumes is difficult to predict, and depends on several factors, such as the type of cooking oil, type of food, energy source, cooking temperature and ventilation, and measurements of exposure levels in kitchens have shown large day to day variations (69-73). Time spent frying has recently been identified as a determinant increasing the

total personal exposure to airborne particles in kitchen workers at “large-scale-kitchens” and European restaurants (73).

1.4 Occupational health research

- Background, context and introduction to applied methods

“The secret of being miserable is to have leisure to bother about whether you are happy or not. The cure for it is occupation.” - George Bernard Shaw

It is commonly acknowledged that occupational activity entails an extensive impact on personal and public health. In the contemporary society, some of the essential connections between occupation and health become increasingly apparent to many of us. However, when looking back a few generations, the conception of work related health was probably quite different, at least in academic life. One of the very first known academic publications describing health issues specific for different occupations, “De Morbis Artificum Diatriba”, was written by the Italian physician and professor named Bernardino Ramazzini, and published in 1700 (74). The book addresses the work environment and health of 42 groups of workers and contains suggestions of preventive measures. Some of the wording used in this book indicates that Ramazzini’s interest and insight into the field of occupational health probably was uncommon in academia at that time. Some of the phrases in the section on the conditions for sewage workers, reveals that he even expected some of his colleges to be embarrassed by reading that he approached and described such work environments.

By observing the occurrence of different manifestations of health complaints and diseases in specific occupations, Ramazzini discovered many important associations between work exposures and the development of disease, and suggested possible explanations and interventions. At that time, essential research instruments, such as the microscope, was under development but not readily available, and obtaining more detailed knowledge on the bio-physiological aetiology behind some of the observed associations would presumably be impossible.

1.4.1 Measuring inflammatory markers

With later advances in technology and science, many of the mechanisms of how work related exposures afflict human health have now been investigated and are better understood. New knowledge and new technologies have also provided the possibility of observing and investigating possible preclinical physiological changes caused by different exposures. Physiological changes leading to a disease is often termed 'pathophysiology of disease.' There has, for many years, been an increasing focus on the role of inflammation in the pathophysiology of many different diseases, such as asthma, COPD, cancer, diabetes, and cardiovascular disease. The following section describes some of the more recent methods for investigating inflammation as a possible response to exposure to air pollutants.

When commencing the research project, "Cooking fumes and health," methods for the quantification of the inflammation of lung diseases was mainly based on analyses of samples collected by invasive methods, such as bronchoalveolar lavage and bronchial biopsy (75), the semi-invasive method induced sputum (76), and measurement of inflammatory markers in the blood (77) and urine (78), of which the latter two are usually more likely to reflect systemic effects rather than inflammation of the lungs (79).

Analysis of inflammatory markers in exhaled air was, at the start of this project, a relatively new way of studying airway conditions based on completely non-invasive methods, which can be repeated several times with short intervals between the samplings (80).

Exhaled air consists of a gas phase that contains volatile substances like nitrogen oxide (NO), carbon monoxide (CO) and hydrocarbons, as well as a vapour phase that can be captured by the cooling of exhaled air to condense it (international abbreviation exhaled breath condensate, EBC) (79, 81).

Measuring exhaled NO is a well-known, non-invasive method for assessing respiratory tract inflammation (82-85). The concentration of exhaled NO is increased in asthma and positively related to the degree of eosinophilic inflammation (84, 85). Oxidative stress and inflammation of the lungs also causes an increase in CO concentration that can be detected in exhaled air; however, the values for CO are also heavily influenced by tobacco smoke and contaminated ambient air, and are therefore less used than NO as an inflammatory marker (86).

Measurement of the aliphatic hydrocarbon ethane has been recommended as a non-invasive marker for an induced lipid peroxidation of free radicals in humans (87). Ethane in exhaled air has been used as a marker for oxidative stress in smokers (88), and in interstitial lung disease (89). However, a later study claimed that ethane in respiratory air is a biomarker of exposure and not an effect as such (90).

Exhaled breath condensate (EBC) can contain many different inflammatory products, but due to limited volume of samples and relatively low concentrations, only a few markers have usually been analysed for each sample. Methodological improvements have been achieved for the measurement of interleukin (IL) -1β, leukotriene B4 (LTB4) and 8-isoprostane (91), which are among the markers estimated to reflect respiratory tract inflammation (81). LTB4 is formed from arachidonic acid as a result of enzymatic hydrolysis of leukotriene A4, which is a proinflammatory mediator and a potent neutrophil granulocytic activator (92). 8-isoprostane is a stable prostaglandin-like product formed from arachidonic acid by non-enzymatic activity of reactive oxygen radicals, and is therefore suggested to be a marker of oxidative stress (93). The concentration of LTB4 and 8-isoprostan in EBC has been found to be elevated in asthmatics and in patients with COPD. The levels in COPD patients were further increased during COPD exacerbations and decreased after treatment with antibiotics (94). It has been suggested that interleukin-1beta (IL-1β) in the respiratory tract contributes to non-eosinophilic inflammation and in pathogenesis in difficult to treat asthma (95).

The research group at the Department of Occupational Medicine, St. Olav's hospital and NTNU where this PhD-project was conducted, has previously investigated possible relationships between respiratory exposures and ischemic heart disease (96, 97). This was based on a hypothesis that the inhalation of fine dust and other air pollution induces inflammatory reactions in the lungs with the release of mediators that can affect the blood's coagulability (98). It has been suggested that a pathophysiological mechanism for this may be that the release of interleukin-6 (IL-6) from cells of the bronchial mucosa stimulates the production of fibrinogen from hepatocytes (99). In 1998, a meta-analysis of more than 18 studies concluded that there was an association between fibrinogen concentration and the occurrence of ischemic heart disease, which is probably causal (100). Increased blood concentration of fibrinogen increases blood coagulability, and this may be part of the pathophysiological explanation of why workers in different occupations exposed to airborne particles and other respiratory irritants have been found to have an increased risk of ischemic heart disease (97, 99, 101). Based on this, we found it interesting to explore the possible effects on inflammatory markers from exposure to cooking fumes, as the air pollutant is readily found in the cooks' work environment.

1.4.1 Lung function measurements

Spirometry is the most common lung function test. It has been used for a long time in many surveys to detect chronic work-related pulmonary function impairment in general, and has also made possible the study of short-term cross-shift changes in different situations (102, 103). The traditional spirometric time-volume curve describes the functional volume of the lungs, while flow-volume curves visualizes the airflow. The commonly used specific parameters in clinical settings, such as forced expiratory volume in the first second (FEV1), are mostly affected by the status of the proximal airways. Moreover, it has been suggested that other specific flow-parameters, such as FEF_{25-75}, might provide some additional information regarding the function and air flow of the smaller and more peripheral airways (104).

1.5 Epidemiology

As mentioned above, Ramazzini's research was based on observing differences in health outcomes between different groups of people. This methodology might be regarded as the essential concept of epidemiology. The progress of consecutive epidemiological research constitutes major and possibly decisive elements in the evolving understanding of specific "risk factors," other determinants of health outcomes in general, as well as work related health issues. The following section is included with the intention of briefly considering some of the relevant and interesting aspects of epidemiological research methods, with a main focus on validity. With the purpose of establishing contemporary content of the term "epidemiology," the following definition from WHO is embedded:

"Epidemiology is the study of the distribution and determinants of health-related states or events (including disease), and the application of this study to the control of diseases and other health problems. Various methods can be used to carry out epidemiological investigations: surveillance and descriptive studies can be used to study distribution; analytical studies are used to study determinants" (105).

One of the main differences between experimental research and epidemiological investigations are that in the latter, information is retrieved from people in real life settings. This entails both ethical and methodological challenges. In research on laboratory animals, the premises can be controlled and accounted for. In contrast, it is not possible to control all conditions accompanying human behaviour and everyday life. For research purposes, this entails challenges in ensuring that the information obtained is as accurate as possible, in order to enable the analyses made on the basis of this information to provide results that are as valid as possible. The term validity exhibits different definitions depending on the context. The following comprehensible definition is provided by Wikipedia: "**Validity** is the extent to which a concept, conclusion or measurement is well-founded and corresponds accurately to the real world" (106). In scientific research, it is common to differentiate between internal and external validity.

In this context, “internal validity” is the extent to which a causal conclusion based on a study is warranted, which is determined by the degree to which a study minimizes systematic error. In relation to this, the external validity is the extent to which the results from a study can be generalized to other populations and/or to other situations.

Experimental research may often show a high degree of internal validity due to the capability of avoiding systematic errors by keeping close control of all the premises. On the other hand, it can often be difficult to ensure that the experiments resemble real life situations and exposures to such a degree that the results are relevant and generalizable, and significant uncertainties regarding the external validity of the results are often implicated. Furthermore, some real life situations and exposures are difficult to reconstruct in experimental settings. For instance, it would obviously be considered unethical to perform experimental research on humans with the intention to evaluate exposures at levels suspected to induce significant and persistent negative effects on health. Developing knowledge on the effects of such exposures might, to a large extent, depend on epidemiological approaches.

The validity of epidemiological investigations is threatened by several types of systematic errors that can lead to skewness or so-called bias. Such systematic errors can be divided into the following three main groups: Selection bias, information bias, and confounding (107). In the following, a brief introduction of these terms is presented.

1.5.1 Selection bias

Conducting research on humans implies constructing a sample. This is done by selecting specific people for the study based on defined criteria. Both weaknesses of the inclusion criteria and difficulty reaching out to and retrieving the people who fit the study may cause the selection of subjects that is successfully included to not be representative of the population of interest. Selection is particularly prominent in cross-sectional studies, where usually, there is no information on what has happened in the

population prior to the establishment of the research groups. For instance, some working environments may require the workers to be of good health in order to endure, which might implicate a tendency of recruiting and retaining workers of exceptional good health into such work-places. This type of selection bias is often referred to as the "healthy worker effect." It is also comprehensible that for different work-places, those workers who develop a severe disease are eventually not able to continue their job and thus, might already have quit and become unavailable for inclusion before the establishment of the study groups. Selection can also be particularly prominent in investigations of serious illness, where some people who should have been included in the sample will not have survived long enough to be part of the study.

Most surveys are also dependent on that the persons in the sample make themselves available for research by for instance by providing consents to participate, answering questions or making other types of efforts. The people who decline to participate fall out of the study and without any further information from these people, it is difficult to know how this affects the representativeness of the sample. Generally, people, who for various reasons, do not participate in surveys, usually have poorer health than those who participate. This phenomenon is often called the "non-responder bias." Avoiding selection bias in epidemiological investigations is difficult, but should be strived for by designing the studies in such a way that they take into account, as much as possible, various selection mechanisms.

1.5.2 Information bias

Information bias is a term that includes the errors that arise in obtaining and classifying information from the sample of a study. One of the main concerns are errors related to the misclassification of the data. These errors can be systematic (differential misclassification) or unsystematic (non-differential misclassification.) Differential misclassification of the determinant occurs when factors related to the outcome also determine who are considered as exposed or not and vice versa, as to the outcome. The "determinant" is the factor that the study attempts to investigate the effect of, while an outcome is the event(s) that one may think is influenced by the determinant. For

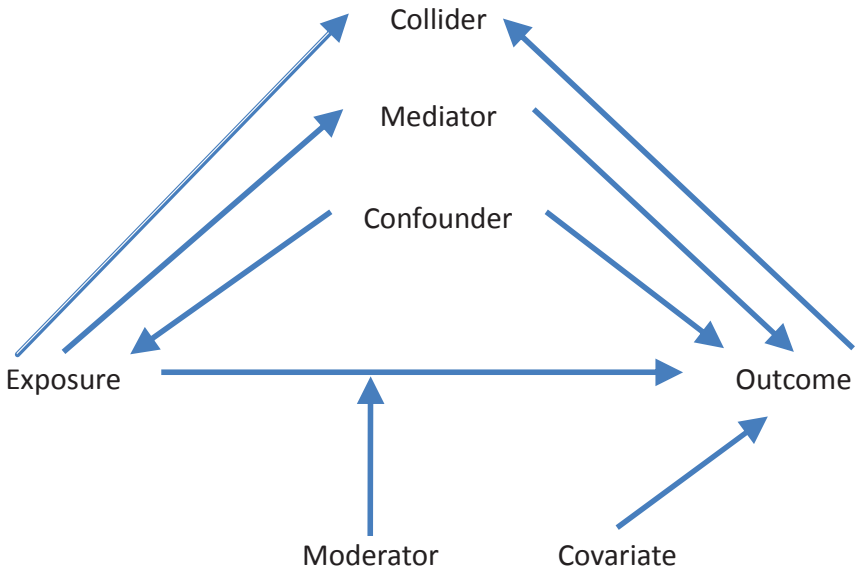
instance, the “determinant” can be specific exposures or risk factors, while “the outcome” is often a particular disease or health condition. Non-differential misclassification is a matter of sensitivity and specificity in the characterization of both the determinant and the outcome, and will usually weaken any estimated effects of the determinant, thus increasing the risk of missing differences between the groups. This effect is often called ‘bias’ toward the null hypothesis. Differential misclassification may affect the results towards a particular direction, compromising the validity of the result. This may be due to individual perceptions of possible causes of disease, affecting the individual memory in such ways that a person who has a disease or health condition has an increased chance of remembering the factors he or she believes may be related to the disease / health damage (recall bias) (108, 109).

Efforts to avoid bias due to misclassification needs to be incorporated into the design of epidemiological studies. In order to limit bias as from non-differential misclassification, one can choose whether to emphasize sensitivity or specificity in the characterization of the determinant and the outcome, depending on how frequently these occur in the study population.

1.5.3 Confounding

The term “confounding” includes known and unknown causal factors other than the studied exposure being more or less prominent in the exposed group than in the control group, and which may affect the outcome rate. This may lead to a situation where the effect of the exposure is mixed with the effects of other causal factors. In other words, confounding in epidemiological investigations is any phenomena, except from the studied exposure, that make the exposure group less comparable to the control group. When present, confounding may cause the estimated effect of the exposure on the outcome to be biased in any direction, leading to wrong conclusions. Thus, the validity of the results depends on the study’s ability to adequately address the issue of confounding. A challenging, yet crucial step when addressing the issue of confounding, is the effort of identifying possible “confounders,” which must be evaluated under

scrutiny. A “confounder” is a factor that to some degree, is predictive of both the exposure and the outcome. It is necessary to maintain control of which potential confounders are present and to take these into account in the design of the study or in the analysis. Otherwise, there is no way of telling whether the estimated effect is truly caused by the exposure or if it is actually caused by confounders. It may be useful to outline the relationship between different factors in a diagram or flow chart. The illustration on the next page provides an example of how such a figure might look. Confounding can for instance be handled both in the design of a survey through so-called "matching" and/or in the analysis through stratification or adjustment (standardization) for the occurrence of potential confounders in the population. However, identifying and handling potential confounders requires expert knowledge in the field, and that information about the distribution of such underlying factors is available.



2 Summary of the introduction and aims

Work-related health challenges and an increased morbidity and mortality have been reported among cooks. It has also been indicated that Norwegian cooks have high turnover rates. The kitchen as a work-place entails multiple occupational hazards, including exposure to cooking fumes. Several studies have shown possible connections between exposure to cooking fumes and adverse effects on the airways, as well as various other negative health effects. Based on this, the aims of the studies were:

- to investigate if short term exposure to moderate levels of cooking fumes in an indoor environment causes changes in pulmonary function
- to investigate whether the inhalation of cooking fumes alters the expression of inflammatory reactions in the bronchial mucosa and its subsequent systemic inflammatory response in blood biomarkers
- to elucidate work sustainability in cooks and to analyse if there is a connection between work environment factors and work sustainability
- to evaluate determinants for the presence of work related respiratory complaints among Norwegian cooks

3 Research design, methods and analyses

The research design in this doctoral project is two-fold, with an initial laboratorial research experiment focusing on exposure to cooking fumes, followed by epidemiological investigations in a cohort of real life cooks. The main results and applied methods are presented and discussed in the enclosed papers, Paper I-IV.

3.1 Experimental research (Paper I - II).

3.1.1 Subjects

Twenty-four non-smoking students without any current respiratory disease were recruited for the experimental study. The first twelve subjects recruited were assigned to Group A, and consisted of 8 males and 4 females. Consecutively, twelve subjects, 6 males and 6 females, were recruited for Group B. The two groups were examined on two different occasions under slightly different conditions.

3.1.2 Exposure model

On the same weekday of two consecutive weeks, the study subjects spent time in a model kitchen and had samples taken, and lung function was measured. For all the subjects, the first week was spent without any exposure other than to normal air, while during the second week, they were exposed to generated cooking fumes. In their second stay in the model kitchen, all the subjects were exposed to controlled levels of cooking fumes while pan-frying beef; Group A used an electric hob, while Group B used a gas hob. The concentration of cooking fumes in the kitchen was monitored with an instrument and regulated by adjusting the quantity of the beef in the pan, the extraction rate of the kitchen ventilator, and the effect level of the hotplate or the gas burner. The levels were kept at 8-10 mg/m^3 for Group A and at 10-14 mg/m^3 for group B. Group A was exposed to cooking fumes in the kitchen for 2 hours, with each person performing the frying on 3 occasions for approximately 15 minutes each time, while Group B was exposed for 4 hours, with each person doing the frying on 3 occasions for approximately 25 minutes each time. Each person was equipped with sampling devices

for total particulate matter, with filters placed on the right shoulder, close to the breathing zone. The filters were analysed gravimetrically.

3.1.3 Sampling of biomarkers and lung function measurements

Three samples of blood and exhaled air were collected and lung function was tested four times during two consecutive 24 hour periods, with one week in between. The first sampling period was without any exposure other than normal air, while during the second period, the participants were exposed to controlled levels of cooking fumes. Besides the differences in exposure to cooking fumes, the program in the two sampling periods were exactly the same in regards to location, activities, testing of lung function and sampling of blood and exhaled air. Thus, the subjects were their own controls. This made it possible to compare each subject's change in lung function and concentration of biological markers over a certain period of time with short term exposure to cooking fumes and with the corresponding changes over a certain period of time without such exposure. In both periods, the three sampling points in time were: 1) in the morning before entering the kitchen, 2) when leaving the kitchen after two hours (Group A) or four hours (Group B), and 3) 24 hours after entering the kitchen. In addition to these time points, lung function was also tested 6 hours after entering the kitchen.

3.1.4 Statistical analysis

Data was registered and some of the analyses were performed using IBM® SPSS® Statistics program for Windows (version 14). A Spearmen-Rank test was used to compare the intra-individual change in pulmonary function during the day with exposure, as well as the intra-individual change during the day without exposure.

We used mixed effects modelling with a random intercept to study the association between exposure to cooking fumes and changes in the different inflammatory markers. This analysis was performed using Stata® Statistics program for Windows (version 11). To account for the tendency of log normality, the data was log-transformed and the

results presented as geometric means with a 95% confidence interval. In the model, exposure and baseline measurements were included as covariates together with a time variable, and measurements at Occasions 1 and 2 were included as the outcome variable. The analysis was performed both separately for the Groups A and B and combined for the two groups. Further, the chosen significance level was 5%.

3.2 Epidemiological investigations (Paper III-IV)

3.2.1 Study population

For the purpose of elucidating work environment factors, work sustainability and occupational health in cooks, we founded a historic prospective cohort of skilled cooks in 2010. In order to make the project feasible, the material was limited to cooks in the middle part of Norway. The educational authorities provided us with the names and the 11 digit personal identification number of 2082 subjects who had qualified as a cook during the years 1988-2008. Invitations to join the cohort were sent out by mail. We were unable to find the postal addresses for 155 subjects, and while 11 had emigrated and 4 were deceased, we ended up with 1912 eligible subjects, of whom 894 gave consent to participate. There were 540 female and 354 male respondents. The mean age when answering the questionnaire was 33.7 years and 33.1 years for female and male respondents, respectively. The mean age when graduating as a cook was 22.3 years (females) and 21.4 years (males). A summary of some characteristics of the study subjects in Papers III – IV is presented in Table 1, (in Paper III).

3.2.2 Methods

The data used in this thesis was collected in 2010 by a mailed questionnaire, inquiring about some health related issues, including respiratory symptoms, what types of kitchens they were or had been working at, and various work environment factors. Those who had left the profession before the survey were asked to relate their answers to the last place they had been working as a cook. They were also asked about smoking

habits and alcohol consumption (the whole questionnaire is enclosed; see Appendix 5 in Norwegian).

3.2.3 Statistical analysis

All data were stored and analysed with the IBM® SPSS® Statistics program for windows (version 20 and 23). Work sustainability was analysed with Kaplan-Meier survival analysis in relation to the type of education, last or present place of work and type of kitchen with the longest held job. Differences between groups were estimated by the Log Rank (Mantel Cox) test. In addition, we performed Cox regression analysis with adjustment for sex by the enter method in relation to the last or present place of work, type of kitchen with the longest held job, shift work, working hours, types of food prepared, and various ergonomic factors. Other differences between groups were tested for statistical significance with a two-tailed Pearson's Chi Square test. Determinants for respiratory symptoms were investigated with logistic regression adjusting for age, sex, and smoking.

4 Ethical considerations

4.1 Paper I-II

As the experimental intervention was short and the exposure level was kept within fixed limits, the total accumulated exposure to cooking fumes experienced by the participants were quite low, and considered to not constitute any risk of inducing persisting complaints or negative health effects. The experimental study was approved by the Ethical Committee for Medical Research in central Norway. Participation was voluntary and all persons gave their informed consent prior to their inclusion in the study. Written information about the project was given to every participant, also stating that he/she could withdraw from the study at any time. All participants received an allowance for their participation.

4.2 Paper III-IV

Responding to questionnaires might always induce reflections related to the questions that are asked, but no adverse effects were expected from participating in the prospective cohort of cooks. The study protocol was approved by the Regional Committee for Medical Research Ethics in central Norway (approval No. 4.2008/2527), and signed informed consents were obtained from each participant.

5 Results and summaries of papers

The main results in the thesis are summarized in the following section. For closer details, please consult the results sections of the Papers I-IV.

5.1 Paper I-II

In our experimental study, 24 healthy volunteers were exposed to normal air and to cooking fumes for 2 or 4 hours in a model kitchen on two different occasions. The purpose was to explore whether short term exposure to these cooking fumes alters lung function (Paper I) and the expression of inflammatory reactions in the bronchial mucosa, as well as its subsequent systemic inflammatory response in the blood (Paper II). The changes in spirometric values during the day with exposure to cooking fumes were not significantly different from the changes during the day without exposure, with the exception of forced expiratory time (FET). The change in FET from entering the kitchen until six hours later was significantly prolonged between the exposed and the unexposed day, with a 15.7 % increase on the exposed day, compared to a 3.2 % decrease during the unexposed day (p-value = 0.03). The same tendency could be seen for FET measurements done immediately after the exposure and on the next morning, but this was not statistically significant. As well, the measured levels of inflammatory markers showed minor variations during the days of the experiment. Thus, the variations showed some different patterns, but did not amount to any conclusive evidence of a biological effect from the exposure. When comparing the development in levels of inflammatory markers during the days with and without exposure, the only difference that reached statistical significance was a slight increase in the concentration of d-dimer in blood from 0.27 mg ml^{-1} on the morning before exposure to cooking fumes to 0.28 mg ml^{-1} on the morning after, compared to a slight decrease from the morning before exposure to normal air to the morning after (P = 0.004). There was also a trend of an increase in interleukin (IL)-6 in the blood, ethane in exhaled air, and IL-1β in EBC after exposure to cooking fumes. The separate analysis for group A and B, seemed less robust than the combined analysis, with some uncertain further variation in

measured levels in group A that should be interpreted with caution. Group A, showed an increase in the levels of ethane, from 2.83 parts per billion (ppb) on the morning before exposure to cooking fumes to 3.53 ppb on the morning after exposure (P = 0.013) and IL-1β, from 1.04 on the morning before exposure to cooking fumes to 1.39 pg ml^{-1} immediately after (P = 0.024), and a decrease in levels of 8-isoprostane and LTB_4 (P- values = 0.003–0.022) both immediately after exposure to cooking fumes and on the morning of the day after.

5.2 Paper III-IV

Work environment factors, work sustainability and its potential determinants was surveyed in a cohort of 2,082 subjects, who, from 1988 through 2008, had qualified as skilled cooks in the middle of three counties in Norway. Prevalence of respiratory complaints and associated work environment factors were investigated in the same cohort (Paper IV). The median time at work was 16.6 years. Substantial differences in sustainability between types of kitchens was observed for both sexes (p = 0.00). The lowest work-sustainability was found among the cooks in restaurants, with an estimated median time in the profession of 9.2 years, while the cooks in institutions and canteens showed a substantially higher sustainability, with 75.4 % still at work after 10 years, and 57 % still at work after 20 years in the profession. Of those still at work as a cook, 91.4 % reported a good or very good contentment. Excitement of cooking, the social working environment, and the creative features of cooking were frequently reported as reasons to continue among the 67.4 % who expected to stay in the profession over the next 5 years. Musculoskeletal complaints were the most common health-related reason for leaving work as a cook, while working hours was the most common non-health-related reason. Of those still working as a cook at the time of the survey, 17.2 % reported respiratory complaints at work and among those, close to 27.7 % stated daily or almost daily symptoms.

In those still working as a cook in restaurants and reporting respiratory symptoms at work, as many as 72.7 % experienced improvements during vacations and weekends, which gives strong reasons to suspect work environment factors to be contributing to their morbidity. The cooks who spent more than half of their workday frying food by the use of a plate, frying pan or grill, showed an increased odds ratio of 2.5 (95 % C.I.: 1.2 – 5.3) for having chronic bronchitis and an odds ratio for improvement when off work of 1.9 (95 % C.I.: 1.0-3.7), compared to those who spent less than half the workday with such activities.

6 Discussion

6.1 Methodological considerations

Most previous investigations on the effects of cooking fumes have looked at manifest diseases and chronic effects in the airways of cooks and other vulnerable groups. In the experimental study, we aimed to detect short-term, small changes in inflammatory markers and in the lung function in healthy volunteers as a result of exposure to controlled levels of cooking fumes. This is different from normal clinical practices. When applying diagnostic tests in the examination of patients in search for manifest diseases, there is usually a predetermined cut-off level for when a test should be interpreted as "positive." When attempting to detect transient changes from low exposure, the possible changes in biomarkers are not expected to reach any such predetermined pathological levels. This complicates the interpretation of the results. In addition, changes in lung function and level of inflammatory markers that are expected to occur in connection with short-term exposure may be so modest that they are difficult to detect with existing methods. Nevertheless, when applied in an experimental setting, we found that it might be possible to display interesting contrasts even at low levels and small changes precisely because each subject is used as its own control and thus, the changes seen do not necessarily have to reach a level that is diagnostic of manifest disease to be interesting. An important challenge in this context is to think broader than in a clinical practice. For example, when applying spirometric testing of patients for diagnostic purposes, the parameters used in the diagnostic criteria of asthma or COPD is the gross measures of lung function, such as FEV1 and FVC. In our experimental setting exploring possible changes in lung function after short term exposure that is so low that it is predicted not to cause any chronic respiratory effects, we would not expect to find dramatic changes in spirometric parameters such as FVC, FEV1 or peak expiratory flow (PEF). In this setting, other parameters, such as FEF_{25-75}, which reflects the function of the small respiratory tract, might be more interesting.

The main advances of applied experimental methods are the possibility to control the premises and to control the level of exposure, as well as to observe and measure

possible outcomes of the exposure. The limitations are however many. The main disadvantage might be that it is impossible to study the effects of long-term exposure to cooking fumes in an experimental setting, since it would be too time consuming, resource demanding, and quite unethical. Thus, conducting epidemiological studies gives the best opportunity when trying to further elucidate on this topic. Gathering information from subjects in their natural habits entails the opportunity to achieve realistic exposures, and any findings might potentially be expected to have a higher external validity than findings from experimental investigations. The downsides are the many sources of potential errors threatening the internal validity, and thus, the validity as such when conducting epidemiological investigations. Thus, both experimental and epidemiological research methods have several potential sources of error that may threaten both the internal and the external validity of the results. The context of the research methods applied in this thesis and their ability to account for potential error are further discussed in the following sections, with a focus on random errors, and the systematic errors of selection bias, information bias and confounding.

6.1.1. Random errors

6.1.1.1 Experimental studies (Paper I and II).

In the experimental studies (Paper I and II), we were able to keep a close control of the premises and aimed at attaining a high internal validity. However, we could not keep the subjects under surveillance for the whole period. We tried to be careful by instructing the participants to act normally and without excesses of any kind at the weeks of the experiments. Even so, it cannot be ruled out that some of them had variations in their day-to-day physical activities and/or experienced incidental exposures not related to the experiments. Any such events may have influenced the results; however, since this would not be related to any specific part of the experimental setting, it would most likely not cause systematic errors. The limitations related to the measurement uncertainty of the applied tests are also expected to be stable during the whole experiment. To minimize random errors in our experimental investigations, all

measurements were performed by trained personnel using a specific procedure, and the instruments used were calibrated as recommended.

6.1.1.2 Epidemiological studies (Paper III and IV).

In our epidemiological investigations, random errors might have occurred during registration of the data. The subjects responded on printed questionnaires, which were scanned and read by a computer. Some of the data might have been lost or misinterpreted by the computer. To reduce the chances of such errors, a manual check by trained personnel was applied to all responses that the computer found difficult to read, any mistakes were corrected, and approximately every tenth computer reading was also checked manually.

6.1.2 Systematic errors

6.1.2.1 Selection bias

6.1.2.1.1 Experimental studies (Paper I and II).

For the experimental studies (Paper I and II), the twenty-four volunteers were recruited conveniently among students on the campus and selection bias was thus heavily present. Apart from being non-smokers and free of respiratory disease, we applied no other inclusion criteria for the study subjects. They were all quite young, and there were more males than females. No randomization was performed, and all subjects were non-smokers with no current respiratory disease. As well, they were all likely to complete higher education. In other words, the sample was not representative of the general population. We would neither claim that our sample was representative for the workforce of cooks in Norway, although the age distribution might be somewhat comparable. It turned out that eight out of twenty-four subjects reported to have had an allergy at some time. We lacked information about what kinds of allergies, but none of the subjects had allergic symptoms when the investigations were done. However, two of them used antihistamine medication at the time of the experiment. When looking separately at groups with reported allergies and use of medication, they did not differ

from the whole group with regards to any outcome, neither did excluding them from the statistical analysis change the results.

Since the experiment aimed to elucidate transient changes in biomarkers related to short term exposure to cooking fumes and the subjects were used as their own controls, the selection bias introduced when recruiting volunteers to these experiments were considered to be of minor importance. However, the small and somewhat homogenous sample of only healthy subjects makes it difficult to assess the possible impacts of individual vulnerability because possibly none of the subjects included had any susceptibilities making them more prone to get reactions from the exposure. This is unfortunate, as individual susceptibilities might be of importance for what effect similar exposure in a long lasting occupational setting might have on health for individual workers. Also, the small sample size naturally hampers the power of the experimental studies. However, it was not feasible to include more subjects at the time, and we were anyway not able to calculate what would be a pertinent sample size in advance due to a lack of previous similar studies. There are, however, some quite similar studies of EBC in different occupational settings that have unveiled effects from specific exposures to airway irritants on exhaled markers (110-113). Changes in markers in the blood have been shown in other studies with a comparable sample size (96, 110, 114-117).

6.1.2.1.2 Epidemiological studies (Paper III and IV).

Since the recruitment to our epidemiological investigations was based on the registers made at the time of qualifying as cooks (1988–2008) and information was collected retrospectively, we consider it to have a kind of a historic prospective approach. When initially including all persons who had qualified as cooks for this period regardless of later events, we were able to reduce the "healthy worker effect," which is often a challenge in cross-sectional studies. However, the low response rate of just below 50% in our study could have introduced another source of selection bias. We did, therefore, perform a non-responder inquiry by telephone call to non-responders, asking them only if they were still active as a cook. Interestingly, 50% of the 46 non-responders that were

reached were still in the profession, while the corresponding figure for the original responders was 61.9%. With this small difference, there is little reason to assume that the low participation rate decisively biased our estimates on work sustainability. One might, however, wonder if subjects who were content with their work as a cook would be more prone to respond to the survey. It is also quite possible that any subjects having developed any dementia or other cognitive problems would choose not to respond to the survey. Nevertheless, we did not find strong reasons to assume that the respondents would have more respiratory complaints than the non-respondents, and thus, we do not think that possible selection mechanisms have decisively biased the estimated prevalence of respiratory complaints in our study.

Self-selection into different types of work places related to personal preferences, health issues or psychosocial factors are likely and have reduced our ability to identify determinants of work sustainability and respiratory complaints. In our investigations of possible determinants of respiratory complaints in the current work environment of still active cooks, any "healthy worker effect" is not so much accounted for, even though the inclusion is based on historical registers because those who have developed health issues related to one work environment might have quit the profession, migrated into different work tasks or work places within the profession. However, it seems that relatively few cooks in our cohort had left the profession due to respiratory symptoms.

6.1.2.2 Information bias

6.1.2.2.1 Experimental studies (Paper I and II).

In our experimental investigations, most of the potential errors related to the measurements would be expected to occur randomly. Since the experiments were not blinded, it is possible that any expectations from the researcher or the subjects might have influenced the effort when performing spirometry. The quality indicator of the spirometry might, to some extent, prevent such potential bias, since it is generally difficult to perform three similar spirometric manoeuvres without giving full effort each time.

6.1.2.2.2 Epidemiological studies (Paper III and IV).

The observations in our epidemiological investigations needs to be interpreted with caution due to the possibility of information bias associated with the use of questionnaires to obtain data, in particular when inquiring on both independent and dependent variables simultaneously (118). The subjects' own perceptions of possible association of work environment factors and experienced health issues might influence what they remember and report regarding their earlier work environment, a mechanism often referred to as 'recall bias.' Since those included in the analysis of determinants of respiratory symptoms were still working as cooks, and the questions regarding the work environment factors investigated was quite categorical and descriptive, we expect that subjects were able to answer these questions correctly without substantial error from recall bias. However, since such biases cannot be excluded, our findings regarding associations between some specific work environment factors and respiratory complaints needs to be confirmed by further investigations on the topic.

In our analysis of potential determinants of work sustainability, those subjects who had quit the profession up to twenty years before they received the questionnaire were required to retain a good memory in order to answer correctly. Regardless of this we would expect that most of the respondents remember some main aspects of their work as a cook, such as their last work place and main work tasks, and therefore, we assume that the data used in our analysis are fairly trustable.

6.1.2.3 Confounding

6.1.2.3.1 Experimental studies (Paper I and II).

It is hard to imagine any confounder that would influence both the determinant and the outcome in the experimental investigations, since the determinant "exposure to cooking fumes" was monitored and adjusted to a predetermined level during the experiments. However, it is possible that we missed unknown covariates or moderators that may have

influenced the outcome differently throughout the various parts of the experiments. When looking at the three morning-samples taken without any prior experimental exposure, it turns out that the day-to-day variation in measured levels of the inflammatory markers in EBC was high. This shows the importance of including situations without exposure in similar studies in order to explore and account for diurnal variability and other systematic effects.

Variation in outdoor temperatures during the days of the experiments is another factor that is beyond our control. On the days of the experiments, the mean temperatures ranged from –7.3 to + 16.8 °C for Group A and from +5.4 to + 19.4 °C for Group B. The influence from the outdoor temperatures, if any, would potentially be different during the various settings with and without exposure, but we did not find it possible to adjust for it in the statistical model. Since all subjects were exposed to normal air on the first occasion and to controlled levels of cooking fumes on the second occasion, any potential moderating effects related to uncontrollable factors in the surroundings on the different days of the experiments may have led to systematic errors and thus, decreasing or increasing the estimated effect of the determinant. However, we could not be certain that any effect from the exposure of cooking fumes during the experiment would not still have an influence on the test results one week later, as it seemed highly unlikely that the exposure to normal air would influence the test results on the day with exposure to cooking fumes one week later. Thus, in this setting, we did not find it reasonable to randomize the order of the two different exposures.

6.1.2.3.2 Epidemiological studies (Paper III and IV).

In our epidemiological investigations, there were several possibilities of confounding. It is possible that lifestyle factors varied between the groups with different work places and work-related exposures. Our statistical analyses were adjusted for smoking, and we did not find any decisive reasons to believe that other lifestyle factors would have confounded the observed associations between the specific work environment factors, such as the amount of frying, and the occurrence of respiratory complaints in our study.

6.2 Discussion of the results

In the experimental setting, the short-term exposure to cooking fumes showed small but quite uncertain effects on lung function and markers of inflammation, while in the epidemiological investigations, exposure to cooking fumes in the work environment of actual cooks showed more clear associations with the occurrence of respiratory symptoms. The overall work sustainability of cooks was far better than what has been previously reported. The following section includes a further discussion of the results from our experimental investigations, (Paper I and II), followed by a discussion of the results from our epidemiological investigations (Paper III and IV).

6.2.1 Experimental investigations, Paper I and II

That our experimental exposure to cooking fumes showed little effect on spirometric parameters apart from a possible effect on FET and some modest changes in inflammatory markers, is to some extent in line with a previous experimental study, where 19 subjects were exposed to ultrafine particles for 2 hours and an increase in d-dimer was observed in the blood after exposure, but no change in fibrinogen or spirometric parameters (116). Increase in d-dimer after exposure to ultrafine particles have been suggested to reflect a systemic change in haemostasis towards the activation of fibrin production, which probably contributes to the occlusive effect such exposure has on the coronary arteries (116).

The increase in d-dimer after exposure to cooking fumes found in our studies, was however very modest, and thus of uncertain relevance. Another study, that measured levels of inflammatory markers in workers exposed to airborne particles, found no significant changes when comparing the levels measured after two days away from work, with levels after two days at work (119). The subjects worked in the Stockholm underground system, and were classified into three different groups, with low, modest or high exposure to airborne particles. Those who were highly exposed, tended to have elevated levels of some measured markers, compared to workers with low exposure,

which indicated a more long term effect on inflammatory markers. This exemplifies that it might be difficult to detect changes in such markers after relatively short periods of exposure to air pollutants, while it also support the possibility of long term adverse health effects from such exposure.

The increase in ethane in exhaled air after exposure to cooking fumes found in group A, might indicate that such exposure also affects the respiratory tract, but these findings are uncertain, since a similar increase was not found in the combined analysis. Variations in exhaled ethane have mainly been regarded as an acute and transient reaction in the airway epithelium as a result of oxidative stress (87, 89, 90). Thus, the results from group A might indicate that exposure to cooking fumes induces oxidative stress in the respiratory tract.

A possible interpretation of the lack of statistically significant changes in parameters other than d-dimer, ethane and FET in the experimental setting may be that twenty-four subjects were too few to provide sufficient statistical strength when studying small changes in inflammatory markers and lung function; however, this was difficult to predict in advance. In turn, we cannot conclude that none of the other parameters were affected, although we could not detect any further statistically significant differences between the days with and without exposure.

We believe that the chosen short-term exposure of cooking fumes was quite realistic. For both Group A and B, the exposure was at a level that led to subjective discomfort, so we found it incorrect to set it any higher. Nevertheless, it might be that the exposure to cooking fumes would need to be higher in order to irritate the lungs enough to provide a short-term reaction that can be measured by further spirometry parameters. The duration of exposure, respectively, two hours (Group A) and four hours (Group B) may also have been too short to provide a response that can be measured with change in multiple inflammatory markers and spirometry parameters. On the other hand, similar studies with other exposures have been able to reveal spirometric changes over relatively short periods of time (103, 120). There are also some comparable studies of EBC in different occupational settings that have unveiled effects from specific

exposures to airway irritants on exhaled markers (110-113) and changes in markers in the blood have been shown in other studies with a comparable sample size (96, 110, 114-117).

The somewhat higher share of non-positive results in our experiments could indicate that short-term exposure to cooking fumes have less or different effects than the exposures to other airway irritants such as ozone, welding fumes, swine dust, ship engine work, tunnel work and chromium, which have been investigated in similar, previous studies. Alternative explanations could be that the methodology applied in our study was less sensitive, or that controlling for variation during a period without exposure made our study more conservative than some of the previous studies.

Although including measurements also from a period without exposure is time-consuming and somewhat less expedient, it might be a crucial feature entailing the possibility to control for various possible biases. In some experimental settings, such efforts may also be regarded as mandatory in order to achieve an acceptable validity to the results, and in particular, when diurnal variations are likely or possible for the chosen parameters measured.

6.2.2 Epidemiological investigations, Paper III and IV:

In our epidemiological investigations, we showed that the specific work environment factors that entail an increase in occupational exposure to cooking fumes were associated with an increased occurrence of respiratory complaints. Together with the large proportion who experienced improvements when off work, our findings provide reasons to assume that work environment factors contribute to the morbidity of the cooks in our cohort.

We also showed that the work sustainability of the cooks in our cohort was better than what has been previously assumed, with a median time working as a cook of 16.5 years for all participants, compared to the previous estimate of 6.5 years. The variation

between the different type of kitchens, ranging from 9.2 years for the cooks in restaurants to more than 20 years for the cooks in institutions and cantinas, seemed to be mainly explained by differences in organizational work environment factors between the different types of kitchens. When exploring the potential impact on work sustainability from different work environment factors, we found that working mostly late shifts was the strongest determinant for leaving the profession. Working in other kitchen types than institutions or cantinas was also a determinant for leaving the profession, which might be much related to differences in working hours between the kitchen types.

When considering that quite a few reported to have daily or almost daily respiratory symptoms at work, and symptoms that improved during week-ends and vacations, it seems somewhat puzzling that respiratory complaints were not a more common reason for leaving the profession. Furthermore, we found that the determinants for respiratory symptoms indicated that exposure to cooking fumes was the most plausible cause for these symptoms. Considered together, this implies that some cooks continue their work despite incurring respiratory complaints, and thus, possibly experiencing long term exposure to cooking fumes at a level that seems to aggravate and potentially be the cause of their complaints. In the study of respiratory work disability by occupations conducted in Telemark county, Norway, cooks were shown to have an increased risk of quitting the profession because it affected their respiratory health (52). While changing jobs might not always be negative for the individual, leaving a profession due to health complaints should be viewed as a more severe outcome. Thus, this study demonstrates important negative consequences from work related respiratory complaints in cooks. When considered together with our findings, it seems plausible that work related respiratory complaints might also be experienced by a substantial share of the cooks that had not left their jobs. In a recent investigation of respiratory symptoms and asthma in the same population, it seems that, compared to a control group of unexposed occupations, there might be a tendency of an increased occurrence of several respiratory complaints in cooks (121). The cooks exhibited increased odds ratios of wheezing 1.4, woken with dyspnea 1.2, asthma attack 1.5, use of asthma medication 1.2, but not for

for having “physician diagnosed asthma” (odds ratio 0.91). There were, however, only 168 cooks in the study, and the odds ratio were not statistically significant.

In light of the known adverse health effects from inhaling air pollutions such as cooking fumes, we find that our investigations, and the mentioned existing literature, warrants further attention to the question of whether exposure to cooking fumes contributes to the observed increased morbidity and mortality in cooks. As the specific work environment factors vary substantially between individual cooks and workplaces, it seems crucial to retrieve detailed information on such factors on an individual level when attempting to elucidate occupational health issues in cooks. Missing information on specific work environment factors might dilute and obscure the effects from specific occupational exposures, while missing information on relevant confounders, such as lifestyle factors, hampers the opportunity to distinguish between potential effects from occupation and from lifestyle factors on the observed outcome. It seems, however, that a majority of the previous studies on morbidity and mortality in cooks in the western part of the world obtained little or no information on lifestyle and specific work environment factors. As such, incurring both confusion between the effects from occupational exposures and lifestyle factors, as well as substantial dilution of the effects of specific occupational exposures, seems likely, and might be important reasons for the conflicting results of previous studies.

In our epidemiological investigations, (Paper III and IV,) we found that knowledge on specific exposures is crucial to find any effects in a group of cooks with many different tasks. Appropriate characterisation of the exposure is important both in regard to occupational factors and lifestyle factors when attempting to elucidate occupational health issues. The lack of due characterisation of relevant exposures in epidemiological studies cannot be easily mended. Retrospectively, adjusting the observed standardised incidence ratio for lung cancer in cooks was attempted in the previously mentioned study from 2004, where smoking habits were assumed on the basis of pooled information on smoking habits in cooks, waiters and home helpers retrieved from

unrelated national surveys (42). By using such an approach, the standardised incidence ratio (SIR) for lung cancer in cooks and stewards was adjusted down to unity, from the quite high unadjusted SIR of 1.48 that was reported in the original publication. If the smoking habits of cooks, waiters and home helpers were similar, this type of adjusting according to their pooled information might have been appropriate. However, if the cooks smoked less than waiters and home helpers, the use of such a pooled estimate of smoking habits would introduce a bias by over-adjusting for smoking in cooks, and in turn, obscuring the effect of occupation on the SIR of lung cancer. Others have referred to surveys indicating that Norwegian waiters smoked more than the general population, while cooks had more similar smoking habits (40). This raises the question of whether it is likely that the increased SIR of 1.48 for lung cancer in these cooks and stewards that were originally published without any data on smoking (41) could be fully explained as a consequence of excess smoking. In our view, the effects from occupational exposures should still be considered as an alternative and plausible explanation.

Some previous studies from Europe have suggested that in addition to smoking, a high alcohol intake represents alternative explanations for the observed increase in the morbidity of cooks, although lacking data on these factors (46, 122). The rationale for this seems to be a combination of a lack of other plausible explanations, as well as specific disease distributions with high rates of diseases where smoking and alcohol consumption are known risk factors, such as oral/pharyngeal, oesophageal, and laryngeal cancer. Occupational exposures have, however, been suggested to contribute to a similar disease distribution in chimney sweeps (46). Furthermore, two studies from Norway have indicated that alcohol intake among cooks is similar to the average population (40).

It has been suggested that the oesophageal mucosa might be exposed to inhaled air pollutants by retrograde ciliar transport in the bronchial tree and subsequent swallowing (44, 123). If such a mechanism is plausible, it is relevant not only in chimney sweeps, but also, in other occupational groups that are exposed to airborne carcinogens. It has become well established that cooks are exposed to airborne occupational carcinogens in cooking fumes. As such, it seems appropriate to hypothesize that deposit of airborne

occupational carcinogens in the airways and consecutive transport to the digestive tract might also contribute to the reported increased incidence of cancers in the digestive tract in cooks. Therefore, the observed disease distribution with high incidence of cancers of both the respiratory tract and the digestive tract in cooks does not necessarily implicate that their increased morbidity and mortality are solely or mainly caused by lifestyle factors.

While it remains somewhat uncertain whether western cooks might have an unhealthier lifestyle than the general population, the insight into the occupational hazards in the profession has increased over recent decades. The exposure to cooking fumes in professional kitchens has been well documented, and the harmful contents of cooking fumes have also been comprehensibly characterized. There is also increasing documentation on the negative health effects from exposure to cooking fumes, mainly from studies on domestic exposure. Although IARC have classified emissions from high temperature frying as probably carcinogenic to humans, it is not established whether work as a cook is associated with increased risk of cancers. Working as a cook does not always entail occupational exposure to cooking fumes, since cooks perform so many different tasks and are employed in various and diverse workplaces. Epidemiological investigations that emphasize the characterization of both specific work environment factors, and lifestyle factors are warranted in order to conclude on the effects from occupational exposures on the morbidity of cooks.

By linking the extent of exposure to cooking fumes with the occurrence of respiratory complaints, our epidemiological investigation substantiates a plausible connection between the current insight regarding the hazards of cooking fume exposure and the reported respiratory morbidity in cooks. Although there are probably several other factors that determine the health of professional cooks and many questions remain unanswered, it is well founded to recommend interventions aimed at the reduction of exposure to cooking fumes. Our findings support that this would be appropriate when attempting to prevent respiratory complaints in cooks. However, when considering our

findings together with the existing evidence, we would further suggest that reducing exposure to cooking fumes should be viewed as a central step towards providing healthy work environments in professional kitchens, which in the long term, might alleviate excess morbidity and mortality in the occupation.

7 Future research

Occupations related to cooking and preparing food for consumption will probably employ many people all over the world throughout the years to come. A significant share of the cooks in our cohort report a high contentment with their work and points to the social work environment and creative features of the work as reasons to continue in the profession. Research on the occupational health of the profession continues to be provident. Both the previously reported increased morbidity and mortality in the profession and the increased occurrence of work related respiratory complaints in cooks shown in our studies, warrant further investigations. An increased insight into the causes of morbidity and mortality in cooks might be achieved by conducting prospective cohort studies, retrieving data on both specific work environment factors and lifestyle throughout their occupational active years, as well as consecutively evaluating health outcomes from national registries. Such an approach was also one of the main intentions when establishing the cohort of cooks that we have used in our epidemiological investigations. We hope that further insight might be achieved by evaluating this cohort in the future, providing the opportunity for evidence based preventive measures. It is an ethical paradox, however, that the members of the cohort will most likely not benefit from any such preventive measures derived from future data on severe disease or mortality that they might supply. Thus, we find that, albeit various limitations in the existing literature, the current knowledge on the occupational exposures faced by cooks, the reported excess morbidity and mortality in cooks, and our recent findings warrants efforts into preventive measures in the profession without awaiting the results of further longitudinal data. The preventive measures should be accompanied with research on the efficiency of the measures, as well as monitoring of the occupational health of the cooks, either concurrent with, or preferably as part of, longitudinal prospective cohort studies in cooks.

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Paper I

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Short term exposure to cooking fumes and pulmonary function

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Abstract

Background: Exposure to cooking fumes may have different deleterious effects on the respiratory system. The aim of this study was to look at possible effects from inhalation of cooking fumes on pulmonary function.

Methods: Two groups of 12 healthy volunteers (A and B) stayed in a model kitchen for two and four hours respectively, and were monitored with spirometry four times during twenty four hours, on one occasion without any exposure, and on another with exposure to controlled levels of cooking fumes.

Results: The change in spirometric values during the day with exposure to cooking fumes, were not statistically significantly different from the changes during the day without exposure, with the exception of forced expiratory time (FET). The change in FET from entering the kitchen until six hours later, was significantly prolonged between the exposed and the unexposed day with a 15.7% increase on the exposed day, compared to a 3.2% decrease during the unexposed day (p-value = 0.03). The same tendency could be seen for FET measurements done immediately after the exposure and on the next morning, but this was not statistically significant.

Conclusion: In our experimental setting, there seems to be minor short term spirometric effects, mainly affecting FET, from short term exposure to cooking fumes.

Background

Exposure to cooking fumes is abundant both in domestic homes and in professional cooks and entails a possible risk of deleterious health effects. When food is cooked at temperatures up to 300°C, carbohydrates, proteins, and fat are reduced to toxic products, such as aldehydes and alkanoic acids[1-4] which can cause irritation of the airway mucosa[5-8]. Cooking fumes also contains carcinogenic and mutagenic compounds, such as polycyclic aromatic hydrocarbons and heterocyclic compounds[1-3,9-13]. Exposure to cooking fumes has also been associated in several studies with an increased risk of respiratory cancer[14-18]. Recently, the International Agency for Research on Cancer has classified emissions from high temperature frying as probably carcinogenic to humans[19].

Frying at high temperatures also produces aerosols of fat with small aerodynamic diameters of 20–500 nm which disperse in the air of the kitchen and nearby facilities. Such aerosols, containing fatty acids, irritate the airway mucosa, and can cause pneumonia[20-22]. It has also been shown that the inhalation of aerosols of oil mist from other kinds of oils can cause small airway obstruction[23-25]. Chinese investigations have shown that exposure to cooking fumes at work can be associated with rhinitis[26], respiratory disorders, and impaired pulmonary function[27]. In two Norwegian studies, it has been shown that cooks and kitchen workers had an increased occurrence of respiratory distress associated with work[28] and increased mortality from airway disease[29]. Few other studies have addressed the biological effects of exposure to cooking fumes in western domestic and professional kitchens.

Spirometry is the most common, and also a quite sensitive pulmonary function test. It has been used for a long time in many investigations, for detecting chronic work-related impaired lung function in general, but it has also been possible to study short term cross-shift changes in different settings[30,31]. The traditional spirometric time-volume curve measures the bowl function of the lungs, while flow-volume curves and other measures also give indications of the function of the smaller and more peripheral airways.

The aim of this study was to see if short term exposure to moderate levels of cooking fumes in an indoor environment causes changes in pulmonary function.

Methods

Twenty four voluntary non-smoking students without any chronic or current respiratory disease were recruited for the study. They were split into group A which consisted of 8 males and 4 females, and group B with 7 males and 5 females. For both groups, measurements of pulmonary function were made under the same setting on two consecutive days during one week without exposure to cooking fumes, and then on the same weekdays during one subsequent week with exposure in an experimental setting.

The subjects were exposed to controlled levels of cooking fumes during the pan-frying of beef in a model kitchen of 56 m^3 (2.5 × 4 × 5.6 m) by use of an electric hob for group A and a gas hob for group B. The door and the window were kept closed, and the only ventilation was a kitchen ventilator which exhaled air at a rate of up to 600 m^3/h. The level of cooking fumes in the kitchen was regulated by adjusting the quantity of beef in the pan, the extraction rate of the kitchen ventilator, and the effect level of the hotplate or the gas burner. The concentration of cooking fumes was monitored with a MIE pDR-1200 optical aerosol monitor (Thermo Andersen Inc., Smyrna, USA) located on a table 1.5 m from the cooking device and set to register the concentration of PM5 aerosols. The level was kept between 8–10 mg/m^3 for group A, and 10–14 mg/m^3 for group B. Group A was exposed to cooking fumes in the kitchen for 2 hours, with each person performing the frying 3 times for approximately 15 minutes each time, while group B was exposed for 4 hours with each person frying 3 times for approximately 25 minutes each time.

The sampling of total particles was performed using preweighed, double Gelman AE glassfiber filters (37 mm). The filters were placed in a closed face, clear styrene, acrylonitrile (SAN) cassette connected to a pump (Casella Vortex standard 2 personal air sampling pump, Casella CEL, Bedford, England) with an air flow of 2 l/min. The filters were placed on the right shoulder of the participant. Before and after sampling, the filters were conditioned in an exicator for 24 hours. The filters were analyzed gravimetrically, using a Mettler weight (0.01 mg dissolution). An inner calibration was performed on the weight before every weighing. Blank filters were included in the analysis in order to control for deviations caused by temperature or humidity.

The pulmonary function of the participants was measured with standard spirometry (Spirare sensor model SPS 310 based on tachopneumographic principles) and data were registered and analysed by the Spirare 3 software (Diagnostica corp., Norway). Spirometric parameters were measured with the subject in a sitting position, wearing a nose-clip, and breathing through the mouthpiece. Standardised instructions were given according to the criteria of American Thoracic Society[32]. We measured forced vital capacity (FVC), forced expiratory volume in one second (FEV1), peak expiratory flow (PEF), forced expiratory flows at 25, 50, and 75% of the vital capacity (FEF25, FEF50, FEF75), and forced expiratory time (FET), defined as the time from the start of the expiratory manoeuvre until the beginning of the end-expiratory plateau. The values used in the analysis were from the best curve out of three qualified performances. The best measurement was defined as that with the greatest sum of FEV1 and FVC. Measurements were done at four occasions for each person both during the week without exposure ("blind") and during the week with exposure to cooking fumes: 1) in the morning before entering the kitchen (between 8 and 9 am), 2) when leaving the kitchen after two hours (between 10 and 11 am (group A)), or four hours (between 12 am and 1 pm (group B)), 3) six hours after entering the kitchen (between 2 and 3 pm), and 4) twenty-four hours after entering the kitchen (between 8 and 9 am). The programme on the "blind day" was exactly

the same as on the day with exposure in regard to location and activities, except that the subjects did not fry any beef, and were not exposed to any cooking fumes. In this way, the subjects were their own controls, making it possible to compare each subject's change in pulmonary function on a day with short term exposure to cooking fumes, with the change in pulmonary function on a day without exposure. Predicted values were based on a European reference material [33].

Results were registered and analyzed using SPSS for Windows version 14. Spearmen-Rank test was used to compare the intra-individual change in pulmonary function during the day with exposure, to the intra-individual change during the day without exposure. A significance level of 5% was chosen, and all statistical test results were two-sided.

The study was approved by the ethical committee for medical research in Central Norway. The participation was entirely voluntary, and written information was given to every participant about the project, also stating that he/she at any time could withdraw from the study. All participants received a symbolic allowance for their participation. There were no known conflicts of interest for any of the authors.

Results

Table 1 shows the individual levels of exposure to cooking fumes, and some background variables for group A (par-

Table 1: Personal exposure to particles from cooking fumes and personal characteristics of the twenty-four volunteers who participated in the study.

Group and subject number		Personal Exposure mg/m3	Sex*	Age (Years)	Height (cm)	Weight (Kg)	Current cold	Known allergy	Current medication
A	1	13.8	F	24	173	65	No	No	No
	2	14.4	M	25	193	105	No	No	No
	3	14.9	F	24	163	61	No	No	Yes
	4	13.9	F	21	152	45	No	No	No
	5	18.9	M	24	183	90	Yes	No	No
	6	20.8	F	22	166	66	No	Yes	Yes
	7	24.1	M	26	193	95	No	No	No
	8	24.4	M	28	177	75	No	No	No
	9	15.4	M	26	184	76	No	Yes	No
	10	32.9	M	25	172	67	Yes	No	No
	11	23.7	M	25	187	74	No	No	No
	12	16.7	M	24	187	84	Yes	Yes	No
All group A mean (SD)		19,5 (5,9)	50% female	24.5 (1.8)	177.5 (12.7)	75.3 (16.4)	25%	25%	17%
B									
	13	33.1	F	24	172	65	No	No	Yes
	14	43.2	M	23	185	73	Yes	Yes	No
	15	50.1	F	21	165	65	Yes	No	No
	16	32.8	F	21	162	49	No	Yes	Yes
	17	53.2	F	24	166	85	No	No	Yes
	18	31.9	M	23	187	86	No	Yes	No
	19	38.6	F	19	170	58	No	No	No
	20	31.2	M	31	176	78	No	No	No
	21	52.5	M	21	165	68	No	Yes	Yes
	22	44.8	M	25	171	63	No	No	No
	23	47.3	M	22	169	63	Yes	Yes	Yes
	24	54.9	M	23	180	95	No	No	Yes
All group B mean (SD)		42,8 (9,0)	42% female	23.1 (3.0)	172.3 (8.1)	70.7 (13.2)	25%	42%	50%
All 24 mean (SD)		31,1 (14,0)	46% female	23.8 (2.5)	174.9 (10.8)	73.0 (14.7)	25%	33%	33%

F = female, m = male

ticipants 1–12) and group B (participants 13–24). The individual level of exposure measured by gravimetric analysis ranged from 13.8 to 32.9 mg/m^3 for group A, and from 31.2 to 54.9 mg/m^3 for group B. The mean spirometric performance of the participants on the first unexposed morning and the mean percent of their predicted values are shown in Table 2. Group A had a higher mean forced vital capacity (FVC) and forced expiratory volume in one second (FEV1), but the groups have about the same results relative to the percent of predicted values. Table 3 shows the changes in spirometric performance during the course of the days with and without exposure, while figure 1 shows the courses of some selected spirometric values as such.

The forced expiratory time (FET) on entering the kitchen compared to the FET six hours later was significantly altered, with a 15.7% increase on the exposed day, compared to a 3.2% decrease during the "blind day" (p-value = 0.03).

The same tendency can be seen for FET measurements done immediately after the exposure and on the next morning, but this was not statistically significant. For the forced expiratory flow when 50% is exhaled (FEF50), group B showed a statistically significant increase between both the first and the second (2-1) and the first and the third (3-1) measurements.

For FEF25 (when 25% is exhaled), a similar difference was found between the first and the third measurement (3-1). We found no statistically significant differences between the changes in other spirometric measurements during the day of exposure, compared to the changes during the "blind day".

Discussion

Most previous studies of effects from cooking fumes have looked at manifest diseases and chronic respiratory effects in cooks and other exposed groups[14-18,26-29]. In this study we aimed to determine early, short term changes in lung function in healthy subjects subsequent to exposure to cooking fumes in an experimental setting. In such a setting we did not expect to find dramatic changes in crude spirometric measures such as FVC, FEV1 or PEF, but rather hypothesised that there might be changes in measures that reflected more the function of the small airways, such as FEF 75 and FET.

In our paired analysis it was shown that FET developed differently during the day of exposure, compared to the "blind day". Prolonged FET has been associated with obstructive disorders[34], and abnormalities in FET have been found in symptomatic smokers with normal FEV1[35]. FET has been suggested as a measure of small airways obstruction[36]. It has been found to have an important discriminatory ability[37], but also a rather low repeatability[37-39]. A recent population study found that FET had a high coefficient of variation (CoV) of 11.3% compared to FVC, FEV1, and PEF which had CoV of 1.38%, 1.44% and 3.0% respectively [38]. It has also been shown that airflow limitation tends to prolong FET, even in healthy subjects [40]. The increase in FET during the day of exposure in our study might thus be explained by inflammatory responses and an obstruction in the distant peribronchiolar tissue caused by the inhalation of cooking fumes. It has, however, been claimed that there is an association between improved spirometric performance and the FET, and that repeated measurements can lead to a training effect[41]. The increase in FET during the day of exposure, which was subsequent to the "blind day", could therefore alternatively be explained by better spirometric performance resulting from a training effect. However, if a learning response was the explanation for the prolonged FET in our study, one would expect to have an increase in FET during the blind day as well, but instead, a decrement in FET appeared. Moreover, if a prolonged FET should be seen as a result of a training effect, the change would probably have gone along with an increase in the FVC and other parameters as well. The lack of such

Table 2: Spirometric values measured in the two groups and % of predicted values.

Spirometric measure	Group A		Group B		All	
	Mean (SD)	% of pred	Mean (SD)	% of pred	Mean (SD)	% of pred.
FVC, litres	5.2 (1.3)	105	4.6 (1.0)	101	4.9 (1.2)	103
FEV1, litres	4.0 (0.8)	95	3.9 (0.8)	102	4.0 (0.8)	99
FEV%	79.0 (8.8)	n.a.	86.2 (3.6)	n.a	82.6 (7.5)	n.a.
PEF litres/min	570 (112)	103	550 (126)	106	560 (117)	104
FEF25 litres/sec.	7.0 (1.6)	88	7.7 (1.7)	104	7.4 (1.7)	96
FEF50 litres/sec	4.3 (0.8)	78	5.2 (1.1)	102	4.8 (1.0)	91
FEF75 litres/sec	1.9 (0.3)	73	2.2 (0.5)	92	2.1 (0.5)	84
FET seconds	5.1 (1.1)	n.a.	3.9 (1.2)	n.a.	4.5 (1.3)	n.a.

n.a. = not applicable

Table 3: Percentual changes in spirometric values at different points in time in the groups and during periods with (E) and without (B) exposure to cooking fumes.

Spirometric measure		Group A (n = 12)			Group B (n = 12)			All (n = 24)		
		2-1#	3-1	4-1	2-1	3-1	4-1	2-1	3-1	4-1
FVC	B	-1.1	-0.6	+0.1	-1.7	-1.3	-2.3	-1.4	-0.9	-1.1
	E	+0.2	-0.5	-0.8	-1.3	-0.9	+0.1	-0.6	-0.7	-0.4
FEV1	B	+1.1	+1.3	+0.6	-0.8	-0.6	-1.6	+0.2	+0.4	-0.5
	E	+0.5	-0.5	-1.2	-0.8	-0.5	-0.5	-0.2	-0.5	-0.9
FEV%	B	+2.3	+1.9	+0.6	+0.9	+0.8	+0.7	+1.6	+1.4	+0.7
	E	+0.3	+0.0	-0.3	+0.5	+0.5	-0.6	+0.4	+0.2	-0.5
PEF	B	+2.4	-0.5	-1.7	-1.7	-1.7	-3.0	+0.4	-1.1	-2.3
	E	-0.8	-0.2	-0.6	+0.9	+2.6	+1.4	+0.1	+1.2	+0.4
FEF25	B	+3.8	+5.9	+3.6	-5.0	-5.4	-4.2	-0.6	+0.3	-0.3
	E	-0.9	+0.5	-0.4	-1.4	+1.9*	+0.7	-1.2	+1.2	+0.1
FEF50	B	+0.6	+3.4	-0.2	-2.6	-4.5	-4.6	-1.0	-0.6	-2.4
	E	-0.6	+0.7	-2.5	+6.5*	+6.1*	+0.6	+2.9	+3.4	-1.0
FEF75	B	-0.7	+3.7	-0.9	+3.8	+3.8	+0.1	+1.6	+3.7	-0.4
	E	+2.3	+0.6	+0.9	+1.3	-1.0	-0.6	+1.8	-0.2	+0.1
FET	B	+1.0	+0.2	-4.5	-0.7	-6.7	+8.7	+0.1	-3.2	+2.1
	E	+1.0	+16.9	+1.0	+12.8	+14.6	+7.3	+6.9	+15.7*	+4.2

* $p < 0.05$

2-1 is the difference between the first measurement and the measurement at the time of leaving the kitchen after 2 or 4 hours. 3-1 is the difference between the first measurement and the measurement taken 6 hours after entering the kitchen. 4-1 is the difference between the first measurement and the measurement taken 24 hours after entering the kitchen during the day with exposure compared to the day without exposure.

an improvement in our study makes the possibility of a learning effect in regard to the observed increase in FET less probable, in our view.

Although the other spirometric parameters did not develop significantly differently on the "blind" day and the day with exposure, there might have been a tendency. We find it interesting that the mean FEV1 increased by 0.4% from the morning until 2 – 3 pm on the "blind" day, while it decreased by 0.5% during the same period of time on the day with exposure (Table 3 and Figure 1). The increase of FEV1 during the blind day could reflect diurnal variation. In a recent study FEV1 in young adults was shown to increase by 120 ml from 9.00 A.M. until noon, and decreased a little in the afternoon[42]. The diurnal variation of FEV1 was, however, shown to be less pronounced in those who were without symptoms and non-smokers. As our subjects were young, a certain increase in FEV1 from the morning till noon could be expected. On the other hand, all of our subjects were both symptom-free and non-smokers, which might explain the low observed diurnal variation of FEV1 in our study. Also, in the statistical analysis the diurnal variation was controlled for since the change in spirometry was compared between weeks with measurements at the same points of time. The observation of some statistical improvement in FEF25 and FEF50 in group B on the day with exposure compared to the day without was unexpected. When exploring the data, three subjects from group B had unusual, and unexplainably high, starting values for these variables solely on the day without exposure (point 1, dotted line in figure 1). Thus, the difference could as much be due to an unexplainable fall in these measurements on the blind day as due to the slight increase on the exposed day. When the three subjects with the unusual starting values were taken out of the analysis, there were no statistically significant differences.

One possible interpretation of the lack of statistically significant changes in other spirometric measures than the FET could be that the twenty-four subjects that we had access to might be too few to render enough statistical power when studying small changes in the airways. Thus, we cannot conclude that some other parameters of the pulmonary function were not affected, even though we could not detect any significant differences between the "blind" day, and the exposed day.

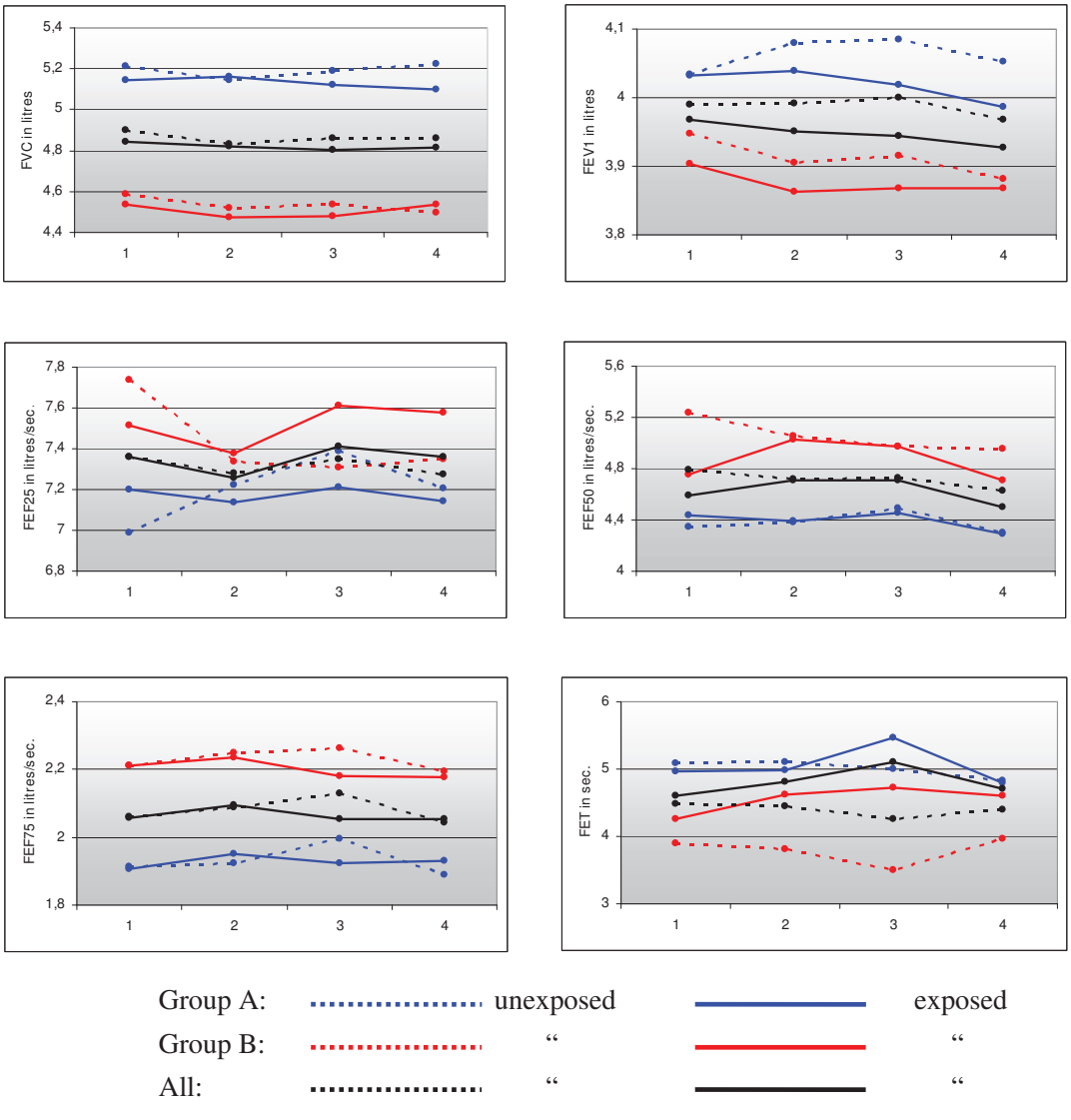

Figure 1
Development of selected spirometric varaiables from 1) Just before entering the model kitchen, 2) When leaving it after 2 (group A) or 4 (group B) hours, 3) Six hours after entering, and 4) 24 hours after entering (next morning).

We think that the chosen short term exposure of the groups to cooking fumes was quite realistic. Both for group A and B, the exposure was at a level that led to subjective annoyance; thus we did not find it right to make it any higher. Even so, it might still have been too low in both groups to irritate the lungs enough to give a short term response that can be measured by more spirometric parameters. By gravimetrical analyses of the personal filters carried by the participants, the exposure seemed to be higher than the levels measured on a stationary basis by

the MIE instrument in the model kitchen. The reason for this was most likely that the MIE instrument was placed 1.5 meters away from the hob, while the filters were mounted near the breathing zone of the subjects, and thus came closer to the hob when the subjects were actually frying beef.

With regard to the duration of the exposure, both two hours (group A) and four hours (group B) might have been too short to give a short term response that can be measured by more spirometric parameters. On the other hand, other studies have been able to unveil spirometric changes over relatively short time spans[30,31]. It should also be recognised that there were no differences in changes in lung function between group A and B, even though group B had a mean cumulative exposure (degree × time) that was more than four times as high as for group A. Thus, the study did not unveil any relationship between cumulative exposure and lung function changes. One should also be aware that there were other differences in exposure between the groups in that group A worked with an electrical hob, while B had a gas hob without observed differences in spirometric changes.

Conclusion

In conclusion, there seems, in our experimental setting, to be minor short term spirometric effects from exposure to cooking fumes, mainly affecting FET.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

SS participated in the design of the study, drafting the manuscript and in performing the statistical analyses. BH participated in the design of the study, drafting the manuscript and in performing the statistical analyses. TQ participated in the design of the study. AKS contributed to the manuscript and was responsible for the exposure conditions. KS participated in the design of the study, contributed to the manuscript and in performing the statistical analyses. All authors participated during the execution of the experimental. All authors read and approved the final manuscript.

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Paper II

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Inflammatory Markers in Blood and Exhaled Air after Short-Term Exposure to Cooking Fumes

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Objectives: Cooking fumes contain aldehydes, alkanoic acids, polycyclic aromatic hydrocarbons, and heterocyclic compounds. The inhalation of cooking fumes entails a risk of deleterious health effects. The aim of this study was to see if the inhalation of cooking fumes alters the expression of inflammatory reactions in the bronchial mucosa and its subsequent systemic inflammatory response in blood biomarkers.

Methods: Twenty-four healthy volunteers stayed in a model kitchen on two different occasions for 2 or 4 h. On the first occasion, there was only exposure to normal air, and on the second, there was exposure to controlled levels of cooking fumes. On each occasion, samples of blood, exhaled air, and exhaled breath condensate (EBC) were taken three times in 24 h and inflammatory markers were measured from all samples.

Results: There was an increase in the concentration of the *d*-dimer in blood from 0.27 to 0.28 mg ml^{-1} on the morning after exposure to cooking fumes compared with the levels the morning before (*P*-value = 0.004). There was also a trend of an increase in interleukin (IL)-6 in blood, ethane in exhaled air, and IL-1β in EBC after exposure to cooking fumes. In a subanalysis of 12 subjects, there was also an increase in the levels of ethane—from 2.83 parts per billion (ppb) on the morning before exposure to cooking fumes to 3.53 ppb on the morning after exposure (*P* = 0.013)—and IL-1β—from 1.04 on the morning before exposure to cooking fumes to 1.39 pg ml^{-1} immediately after (*P* = 0.024).

Conclusion: In our experimental setting, we were able to unveil only small changes in the levels of inflammatory markers in exhaled air and in blood after short-term exposure to moderate concentrations of cooking fumes.

Keywords: cooking fume; exposure; human experiment; inflammation; inhalation

INTRODUCTION

When food is prepared, often at temperatures up to 300 °C, carbohydrates, proteins, and fat are degraded into potentially harmful substances, such as aldehydes and alkanoic acids (Vainiotalo and Matveinen 1993; Robinson *et al*. 2006).

Cooking fumes, especially from frying, contain fine and ultrafine particles (UFP) and several specific agents (Svendsen *et al*. 2002; Wallace, Emmerich, and Howard-Reed 2004; Afshari, Matson, and Ekberg 2005; Sjaastad and Svendsen 2008; Sjaastad *et al*. 2010). Earlier studies have

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shown that frying mainly produces particles of sizes <0.5 μm (Abt *et al*. 2000; Sjaastad and Svendsen 2008) and it seems as if the level of UFP is higher during frying on a gas stove compared to frying over an electric stove (Sjaastad *et al*. 2010). The different chemical substances identified in cooking fumes include aldehydes, polycyclic aromatic hydrocarbons (PAHs), heterocyclic amines, aromatic amines, and alkanoic acids (Vainiotalo and Matveinen 1993; Svendsen *et al*. 2002; Robinson *et al*. 2006; Sjaastad and Svendsen 2008; Sjaastad *et al*. 2010). Personal measurements performed during the frying of beefsteak on both gas and electric stoves have shown that cooks are exposed to PAHs, though in low concentrations (Sjaastad and Svendsen 2009; Sjaastad *et al*. 2010). Cooking with oils rich in polyunsaturated fatty acids at high temperatures also generates various higher aldehyde species, i.e. aldehydes with a higher number of carbon atoms, such as *trans*-2-alkenals, *trans*,*trans*-alka-2,4-dienals and *n*-alkanals, arising from the fragmentation of conjugated hydroperoxy diene precursors (Gertz 2000). Both short-chain aldehydes and higher aldehydes have been detected in the breathing zone of cooks, both in restaurants and during the normal domestic frying of beefsteak (Svendsen *et al*. 2002; Sjaastad and Svendsen 2008, 2009; Sjaastad *et al*. 2010). The levels of the specific agents and particles, however, do not correlate well when measurements are done during the preparation of different types of food (Sjaastad and Svendsen 2009; Sjaastad *et al*. 2010). Both the brand and the age of the cooking oil may also have implications for the composition of the emission (Sjaastad *et al*. 2010). This implies that measuring "total particles" is not a good indicator of exposure to potentially harmful components in cooking fumes. It is still unknown which specific components in cooking fumes may contribute to an increased risk of specific diseases like asthma and cancer.

Previous studies have shown that cooks show an increased prevalence of respiratory distress, increased mortality from airway disease (Svendsen, Sjaastad, and Sivertsen 2003; Borgan 2009), and increased risk of ischemic heart disease (Sjögren, Barlow, and Weiner 2009). A link between the inhalation of respiratory irritants and increased blood coagulability was suggested >15 years ago. Several subsequent studies have found increments in inflammatory markers after various inhalation exposures (Sjögren *et al*. 1999; Corradi *et al*. 2002; Hilt *et al*. 2002; Barregard *et al*. 2006; Barreto *et al*. 2006; Boyce *et al*. 2006; Caglieri *et al*. 2006; Barregard *et al*. 2008; Samet *et al*. 2009). Exposure to cooking fumes has also been associated with an increased risk of respiratory cancer (Coggon *et al*. 1986; Zhong *et al*. 1999; Yang *et al*. 2000; Zhou *et al*. 2000). Based on 'limited evidence in humans, and sufficient evidence in experimental animals', the International Agency for Research on Cancer (IARC) recently classified 'emissions from high temperature frying' to be probably carcinogenic to humans (IARC 2010).

Oxidative stress and inflammatory processes have been suggested to be central parts of the mechanism behind the deleterious effects of cooking fumes (Tung *et al*. 2001; Chang, Lo, and Lin 2005; Wang *et al*. 2010). Such reactions can be assessed both in blood and in exhaled air. The measurement of inflammatory markers in exhaled air provides a possibility of examining the status of the respiratory tract with non-invasive procedures that can be repeated at short intervals (Gergelova *et al*. 2008). Exhaled air consists of a gaseous phase that contains volatile substances, such as nitric oxide (NO) and hydrocarbons, and vapour with epithelial lining fluid that can be captured in an exhaled breath condensate (EBC; Mutlu *et al*. 2001).

The measurement of exhaled nitric oxide (eNO) is a method of assessing airway inflammation, which has proved useful for monitoring patients with asthma [American Thoracic Society and the European Respiratory Society (ATS/ERS) 2005; Corradi and Mutti 2005]. The level of the aliphatic hydrocarbon ethane in exhaled air is seen as a marker of free radical-induced lipid peroxidation (Kneepkens, Lepage, and Roy 1994). Clinically, this has been used to monitor airway reactions in smokers (Puri *et al*. 2008) and to follow patients with interstitial lung disease (Kanoh, Kobayashi, and Motoyoshi 2005).

The levels of interleukin-1β (IL-1β), leukotriene B_4 (LTB_4) and 8-isoprostane in EBC are considered to reflect inflammatory reactions in the airways (Horvath et al. 2005). IL-1β is an important early-response cytokine and was, therefore, chosen as an interesting inflammatory biomarker for analysis. LTB_4 is linked to neutrophil-driven inflammation and 8-isoprostane is a marker of oxidative stress, which is a central part of the airway reaction to cooking fumes. EBC has been previously used for not only disease surveillance mainly but also evaluation of the effects of exposure to xenobiotics in a few studies (Corradi *et al*. 2002; Barreto *et al*. 2006; Boyce *et al*. 2006; Caglieri *et al*. 2006; Barregard *et al*. 2008)

For preventive purposes, we find it worthwhile to try to refine non-invasive methods for the early detection of adverse respiratory and systemic effects in subjects exposed to xenobiotics. The aim of this

study was to see whether the inhalation of cooking fumes alters the expression of inflammatory reactions in the bronchial mucosa and its subsequent systemic inflammatory response in blood biomarkers.

SUBJECTS AND METHODS

Subjects

There were 24 non-smoking students without any current respiratory diseases who volunteered to participate in the study. They were divided into two groups (A and B) of 12 subjects each, and they were examined on two different occasions under slightly different conditions. Lung function measurements in the same groups have been reported earlier (Svedahl *et al*. 2009).

Exposure model

On the same weekday of two consecutive weeks, the study subjects spent time in a model kitchen and had samples taken. For all the subjects, the first week was spent without any exposure other than to normal air, and the second week, with exposure to generated cooking fumes. The cubic content of the kitchen was 56 m^3 (2.5 × 4 × 5.6 m). The door and the window were kept closed, and the only ventilation was a kitchen hood with a capacity to extract up to 600 m^3 h^{-1}. In their second period in the model kitchen, all subjects were exposed to controlled levels of cooking fumes during the pan frying of beef on an electric hob for the first group of 12(Group A) and on a gas hob for the second group of 12(Group B). The concentration of cooking fumes was monitored with an MIE pDR-1200 optical aerosol monitor (Thermo Andersen, Inc., Smyrna, GA, USA) located on a table ~1.5 m from the cooking device and set to register the concentration of PM5 particulate matter in which 50% of particles have an aerodynamic diameter of less than 5 µm aerosols. The levels were kept at 8–10 mg m^{-3} for Group A and at 10–14 mg m^{-3} for Group B by adjusting the quantity of beef in the pan, the extraction level of the kitchen hood, and the effect level of the hotplate or the gas burner. Four subjects participated each time on the days of the experiment. The subjects in Group A stayed in the kitchen for 2 h, and on the day with exposure to cooking fumes, each person performed the frying on three occasions for approximately 15 min each time. The subjects in Group B stayed in the kitchen for 4 h, and on the day with exposure to cooking fumes, each person performed the frying on three occasions for approximately 25 min each time. Table 1 summarizes the time frame of the occasions when subjects from Groups A and B stayed in the kitchen and when samples were taken.

During the stay in the kitchen on the exposure day, each person was equipped with a sampling device for the measurement of personal exposure to total particulate matter. For the sampling, pre-weighed, double Gelman AE glass fibre filters (37 mm) were placed in a closed face, clear styrene, acrylonitrile cassette connected to a pump (Casella Vortex Standard 2 personal air-sampling pump, Casella CEL, Bedford, UK) and set at an air flow of 2 l min^{-1}. The filters were placed on the right shoulder of the participant. Before and after sampling, the filters were conditioned in an exicator for 24 h. The filters were analysed gravimetrically, using a Mettler balance (0.01 mg resolution). An inner calibration was performed on the balance before every weighing. Blank filters were included in the analysis in order to control for deviations caused by temperature or humidity.

Sampling of biomarkers

Three samples of blood and exhaled air were taken from the participants during two consecutive 24-h periods with one week in between. The first period with sampling was without any other exposure than normal air, and during the second period, the participants were exposed to controlled levels of cooking fumes. On both occasions, the three sampling points in time were (i) 0: the morning before entering the kitchen, (ii) 1: when leaving the kitchen after 2 (Group A) or 4 h (Group B), and (iii) 2: 24 h after entering the kitchen. The programme of the participants on the unexposed days was exactly the same as on the days with exposure in regard to location and activities, except for the cooking activities. This facilitated an evaluation of the subjects as their own controls, making it possible to compare each subject's development in terms of the levels of biological markers in exhaled air and blood during a period with short-term exposure to cooking fumes with the development in levels of biological markers from a period without such exposure.

EBC was collected using a breath condenser (ECoScreen; Jaeger, Wurzburg, Germany). The subjects rinsed their mouth with water. In a sitting position, wearing a noseclip, they breathed tidally for 15 min through a two-way non-rebreathing valve, which also served as a saliva trap. In order to avoid loss of molecules from inflammatory markers due to adhesion to the walls, the tubes were coated with 1% bovine serum albumin and 0.01% Tween 20 for 30 min according to previous optimization procedures (Tufvesson and Bjermer 2006). The EBC samples were immediately frozen at –70 °C. Owing to low concentrations in EBC, samples were concentrated [5 times (IL-1β) and 10 times (LTB_4 and

Table 1. Timing of the exposure and the sampling of exhaled breath, exhaled breath condensate (EBC), and blood in the subjects who first entered the exposure chamber on each of the days of the experiments and a comparison of the timing for Groups A and B.

Action time	Sample number	Sampled	Subjectnumber	Time of the day	
				Group A(3 × 4 persons)	Group B(3 × 4 persons)
Morning before entering the kitchen	0	Exhaled breath, EBC, and blood	1	8:30	8:30
			2	8:45	8:45
			3	9:00	9:00
			4	9:15	9:15
Period in the kitchen (same day)			1	9:00–11:00,	9:00–13:00
			2	9:15–11:15,	9:15–13:15
			3	9:30–11:30,	9:30–13:30
			4	9:45–11:45	9:45–13:45
Immediately after leaving the kitchen(same day)	1	Exhaled breath, EBC, and blood	1	11:05	13:05
			2	11:20	13:20
			3	11:35	13:35
			4	11:50	13:50
Next morning	2	Exhaled breath, EBC, and blood	1	8:30	8:30
			2	8:45	8:45
			3	9:00	9:00
			4	9:15	9:15

8-isoprostane)] by freeze-drying and resolved in the respective assay buffer [as previously described (Tufvesson *et al*. 2010)]. The final concentrations were calculated from the specific freeze-dried volumes. LTB_4 and 8-isoprostane were analysed using the EIA kit from Cayman Chemical (Ann Arbor, MI) with a detection limit of 6 and 2.7 pg ml^{-1} respectively. IL-1β was measured using Quantikine HS from R&D Systems (Minneapolis, MN) with a detection limit of 0.05 pg ml^{-1}.

Measurements of eNO were performed by using a Logan LR 2000 chemiluminescence analyser (Logan Research Ltd, UK) in accordance with recommendations by the ATS/ERS (2005). The subjects were in a sitting position, exhaling from total lung capacity to residual volume against a resistance of 4–5 cm water, aiming at a flow rate of 250 ml s^{-1}, aided by a biofeedback monitor. For each subject, the mean of three plateau levels from acceptable eNO curves was registered in parts per billion (ppb).

Ethane in exhaled air was sampled in a 50-ml polypropylene syringe (Terumo) graded to contain 60 ml. A bacteria/virus filter (Icor, leda, Norway) and a non-rebreathing valve were connected to an exhaled air reservoir. The subjects were asked to perform three deep breaths through the mouthpiece mounted on the reservoir. Ethane samples were taken from the reservoir in three parallels. The samples were kept in a refrigerator and analysed within 24 h. Samples of background air were taken at each sampling time. The samples were analysed using a gas chromatograph (HRGC Mega 2 Model 8530, Fisons Instruments S.p.A. Milan, Italy) with a flame ionization detector and two capillary columns [GC-Q, 30 m, internal diameter 0.53 mm (J&W Scientific, Folsom, CA] using a cryofocusing technique described elsewhere (Dale *et al*. 2003).

IL-6 level in serum was measured with the commercial ELISA kit Human Interleukin-6 UltraSensitive (Biosource, Belgium). After standard incubations, the optical density was measured at 450 nm (Wallac Victor^{3}™ 1420 Multilabel Counter, Perkin Elmer, Shelton, CT, USA). Fibrinogen and *d*-dimer in blood were analysed at the Department of Clinical Chemistry at the St Olavs University Hospital of Trondheim. The fibrinogen concentration in plasma was measured by a Fibri-Prest automated by the clotting method of Clauss (Clauss 1957). *D*-dimer was measured by a latex-enhanced immunoturbidimetric method with an automated chemical analysis system (Roche modular-P, Mannheim, Germany) according to the protocols of the manufacturer.

Statistical analysis

We used mixed effects modelling with a random intercept to study the association between exposure

to cooking fumes and the various outcomes. To account for the tendency of log normality, the data was log-transformed and the results presented as geometric means with a 95% confidence interval. In the model, exposure and baseline measurements were included as covariates together with a time variable, and measurements at Occasions 1 and 2 were included as the outcome variable. The data analysis was performed using Stata for Windows version 11. The analysis was performed both separately for the Groups A and B and combined for the two groups, and the chosen significance level was 5%.

Ethical considerations

The study was approved by the ethical committee for medical research in Central Norway. Participation was voluntary and all persons gave their informed consent prior to their inclusion in the study. Written information about the project was given to every participant, also stating that he/she could withdraw from the study at any time. All participants received an allowance for their participation.

RESULTS

Some background variables and mean measured concentrations of cooking fumes on the day with exposure for the participants are given in Table 2. The levels of personal exposure to particles measured by gravimetric analysis on the exposure days ranged from 13.8 to 32.9 mg m^{-3} for Group A and from 31.2 to 54.9 mg m^{-3} for Group B. In regard to current medication, one subject in Group A used contraceptives, and in Group B, two subjects were on contraceptives, two used antihistamines, and one used insulin. No other medication was reported.

Table 3 shows the geometric mean concentrations for inflammatory markers in blood, in exhaled air, and in EBC on the three points of measurements during the week without any exposure other than normal air and during the week with exposure to cooking fumes. When comparing the development in levels of inflammatory markers for all 24 subjects during the 2 weeks, the only difference that reached statistical significance on the 5% level was a slight increase in the concentration of *d*-dimer in blood from the morning before exposure to cooking fumes to the morning after, compared to a slight decrease from the morning before exposure to normal air to the morning after ($P = 0.004$). There also seemed to be a trend of an increase in *d*-dimer and IL-6 in blood and IL-1β in EBC immediately after exposure to cooking fumes. The concentration of ethane in exhaled air showed a trend to increase both immediately after exposure to cooking fumes and on the morning after. The levels of 8-isoprostane and LTB_4 in EBC showed a trend to decrease more after exposure to cooking fumes than after exposure to normal air, whereas the levels of fibrinogen in blood and NO in exhaled air showed only minor variations.

A separate analysis of Groups A and B was performed, but because evidence of heterogeneity was only found for the results from the data of 8-isoprostane, the combined analysis was preferred for the interpretation of the data. The results from the separate analysis may, however, be viewed in the supplementary data (available at *Annals of Occupational Hygiene* online), but the estimates in this analysis should be interpreted with more caution because they seem less robust than those in the combined analysis. Apart from the development of *d*-dimer, post-exposure increases were found only for Group A, which showed an increase in ethane from 2.83 ppb (in the morning before exposure to cooking fumes) to 3.53 ppb (in the morning after; $P = 0.013$) and an increase in levels of IL-1β from 1.04 to 1.39 pg ml^{-1} from the morning before exposure to cooking fumes to immediately after ($P = 0.024$). Group A showed a decrease in levels of 8-isoprostane and LTB_4 (*P*-values = 0.003–0.022) both immediately after exposure to cooking fumes and on the morning of the day after (Time points 1 and 2).

The estimates from the combined analysis on the development of the levels of inflammatory markers are presented in Fig. 1 as 'net changes', which were calculated from the differences in levels from before

Table 2. Mean exposure to particles from cooking fumes and the characteristics for all the 24 volunteers who participated in the study and separate for the two groups A (2-h exposure) and B (4-h exposure).

Group	Personalexposure (mg m–3)	Sex	Age, years	Height, cm	Weight, kg	Known allergy	Current medication
Group A, mean (SD)	19.5 (5.9)	33% female	24.5 (1.8)	177.5 (12.7)	75.3 (16.4)	25%	8.3%
Group B, mean (SD)	42.8 (9.0)	50% female	23.1 (3.0)	172.3 (8.1)	70.7 (13.2)	42%	41.7%
All 24, mean (SD)	31.1 (14.0)	42% female	23.8 (2.5)	174.9 (10.8)	73.0 (14.7)	33%	25.0%

Table 3. Inflammatory markers before and after the stay in the kitchen with exposure to normal air and cooking fumes for all 24 subjects are given as the geometric mean (95% confidence interval).

		Exposure to normal air			Exposure to cooking fumes		
Timing of sample		0 = Before exposure[a]	1 = Immediately after exposure[b]	2 = 24 h after start of exposure[c]	0 = Before exposure[a]	1 = Immediately after exposure[b]	2 = 24 h after start of exposure[c]
Exhaled air	Ethane (ppb)	2.22 (1.97–2.51)	2.31 (2.03–2.63)	2.32 (2.04–2.63)	2.39 (2.12–2.70)	2.59 (2.30–2.93)	2.77 (2.44–3.14)
	eNO (ppb)	4.96 (4.22–5.83)	5.12 (4.35–6.01)	5.29 (4.50–6.22)	5.11 (4.35–6.01)	5.22 (4.44–6.13)	5.25 (4.47–6.17)
EBC	IL-1β (pg ml^{-1})	0.84 (0.64–1.10)	0.87 (0.66–1.15)	0.80 (0.61–1.06)	0.90 (0.70–1.15)	1.12 (0.87–1.44)	0.95 (0.74–1.23)
	8-isoprostane (pg ml^{-1})	4.54 (3.31–6.24)	3.83 (2.78–5.26)	3.15 (2.29–4.33)	4.87 (3.66–6.47)	3.33 (2.49–4.45)	3.25 (2.45–4.32)
	LTB_4 (pg ml^{-1})	5.60 (4.28–7.33)	5.70 (4.35–7.46)	5.05 (3.86–6.61)	7.20 (5.62–9.24)	5.28 (4.12–6.78)	5.22 (4.07–6.70)
Serum	Fibrinogen (g dl^{-1})	2.66 (2.46–2.88)	2.65 (2.45–2.87)	2.63 (2.43–2.85)	2.68 (2.47–2.90)	2.60 (2.40–2.81)	2.71 (2.50–2.93)
	IL-6 (pg ml^{-1})	0.43 (0.31–0.59)	0.41 (0.30–0.57)	0.33 (0.24–0.46)	0.34 (0.25–0.47)	0.41 (0.30–0.56)	0.33 (0.24–0.45)
	d-dimer (mg ml^{-1})	0.29 (0.24–0.35)	0.28 (0.23–0.33)	0.25 (0.21–0.30)	0.27 (0.23–0.32)	0.28 (0.23–0.33)	0.28 (0.24–0.34)

[a]In the morning before entering the kitchen.
[b]When leaving the kitchen after 2 h (Group A) or 4 h (Group B).
[c]Twenty-two hours after leaving the kitchen (Group A) or 20 h after leaving the kitchen (Group B).

the exposure to after the exposure and by subtracting any change that occurred from before staying in the kitchen without exposure to the level after. In addition to the statistically significant development for *d*-dimer, Fig. 1 shows a trend of an increase in levels of ethane, IL-1β, and IL-6 both immediately after exposure to cooking fumes (Time 1) and the morning after (Time 2).

DISCUSSION

The levels of inflammatory markers showed a different development during the period with exposure to cooking fumes compared to the period with exposure to normal air. For some of the markers, the differences reached statistical significance, whereas for most of them, there were only trends. The measured levels of *d*-dimer in plasma developed statistically significantly differently during the two periods of the experiment. In the separate analysis for the Groups A and B, there seemed to be a statistically significant increase in ethane in exhaled air and IL-1β in EBC after exposure to cooking fumes for Group A, but not for Group B. The slight increase in *d*-dimer in blood, IL-1β in EBC, and ethane in exhaled air could reflect an inflammatory response subsequent to the exposure to cooking fumes. It is, however, not entirely clear whether such a response is to be considered pathological or physiological. An increase in *d*-dimer after short-term experimental exposure to UFP has been reported earlier. It has been hypothesized that this increase in *d*-dimer after exposure to UFP reflects a systemic stimulation of fibrin formation, leading to a change in blood coagulability and probably contributing to adverse effects imposed on the arterial endothelium (Samet *et al*. 2009). The lack of a simultaneous increase in the levels of fibrinogen in plasma could be due to different mechanisms as the manner of even normal catabolism is still uncertain, although an endothelial catabolic pathway is suggested as the main pathway (Aliberti *et al*. 2005). Changes in fibrinogen metabolism caused by inflammation are characterized by increased synthesis (Mansoor *et al*. 1997), but this may also be followed by an increased decomposition that can be measured as an increase in levels of *d*-dimer.

IL-1β is one of the most important early-response cytokines, and high levels have been found in fluid obtained by bronchoalveolar lavage of patients with sustained acute respiratory distress syndrome (Park *et al*. 2001). The observed trend towards an increase in IL-1β in EBC immediately after exposure to cooking fumes (although statistically significant only for Group A) seems coherent with the early role of this cytokine in the inflammatory response.

Variations in exhaled ethane have mainly been regarded as an acute and transient reaction in the airway epithelium as a result of oxidative stress

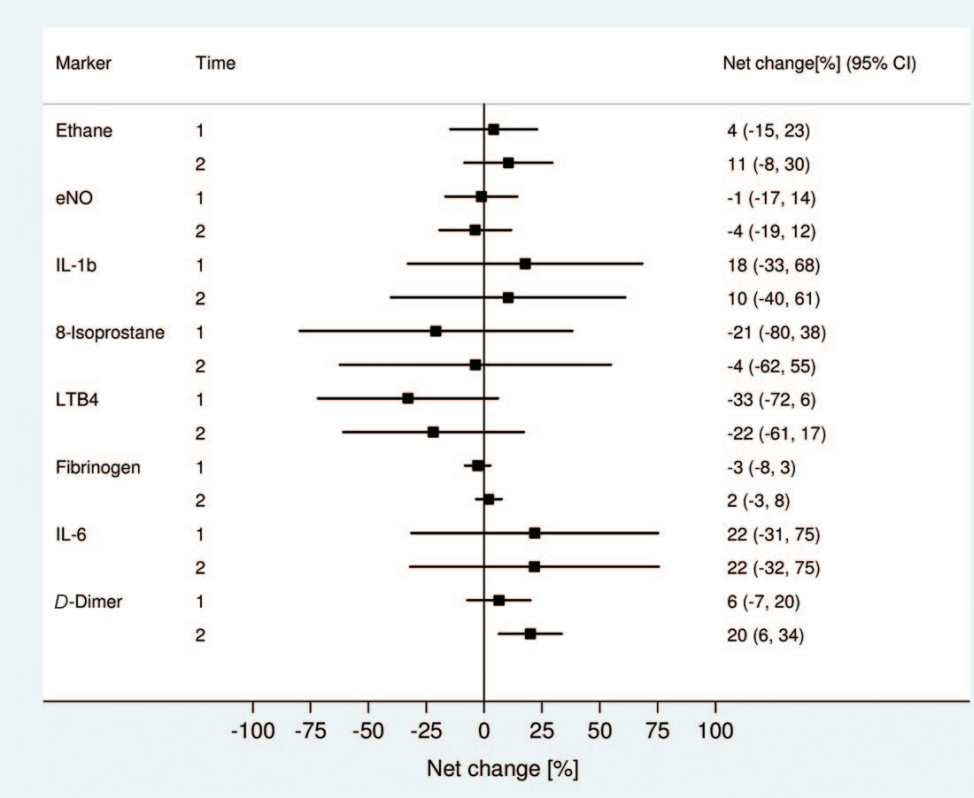

Fig. 1. Net change in levels of inflammatory markers from exposure to cooking fumes for all 24 subjects, according to time since exposure. The estimates are presented as geometric mean changes in percent (95% CI). Time 1 is the net change in levels of inflammatory markers from before exposure to immediately after the exposure. Time 2 is the net change in levels from before entering the kitchen to 24 h after entering the kitchen.

(Kneepkens, Lepage, and Roy 1994; Kanoh, Kobayashi, and Motoyoshi 2005; Gorham *et al*. 2009). However, because the possible increase in our study was found 24 h after the exposure, one could perhaps also regard this as a sign of a more persistent reaction.

In the separate analysis, 8-isoprostane showed a somewhat unexpected development for Group A, with a relatively high starting level the morning before exposure to cooking fumes, followed by a decrease both immediately after the exposure to cooking fumes and the morning after. A similar pattern can also be seen for LTB_4. When looking at the three morning samples taken without any prior experimental exposure, which are presented in Table 3 (Samples 0 and 2 in the week without exposure, and Sample 0 in the week with exposure), it turned out that the day-to-day variation in measured levels of the inflammatory markers in EBC was high. When viewed in this context, the slightly paradoxical post-exposure decrease in LTB_4 and 8-isoprostane seems more likely to be a result of unexplained variability in the estimates rather than an effect related to the exposure. This shows the importance of including situations without exposure in similar studies in order to explore and account for diurnal variability and other systemic effects. In the present study, all the subjects went through a session with exposure to normal air first and then a session with exposure to cooking fumes a week later. As has also been argued in other studies (Barregard *et al*. 2008), we chose not to randomize the order of these sessions because possible long-lasting inflammatory effects from the exposure to cooking fumes could then have affected a control session 1 week later, whereas the opposite seemed less likely.

The low number of participants and the low statistical power of this study might have led to bias with

more non-positive than negative results. *A priori* power estimates were not attempted because there were scarce data on how large the effects could be expected to be from the chosen short-term exposure to cooking fumes on the levels of the inflammatory markers. There are, however, some quite similar studies of EBC in different occupational settings, which have unveiled effects from specific exposures to airway irritants on exhaled markers (Barreto *et al*. 2006; Boyce *et al*. 2006; Caglieri *et al*. 2006; Barregard *et al*. 2008). Changes in markers in blood have been shown in other studies with a comparable sample size (Sjögren *et al*. 1999; Corradi *et al*. 2002; Hilt *et al*. 2002; Barregard *et al*. 2006; Barregard *et al*. 2008; Samet *et al*. 2009). The higher share of non-positive results in our study could indicate that short-term exposure to cooking fumes has less or a different effect than the exposures to other airway irritants, such as ozone, welding fumes, swine dust, ship engine work, tunnel work, and chromium, which have been investigated in similar studies previously. Alternative explanations could be that the methodology applied in our study was less sensitive or that controlling for variation during a period without exposure made our study more conservative than some of the previous studies. Some of the studies mentioned herein were performed in workers at their worksites and some on voluntary subjects as in our study. A possible difference in reaction between subjects with previous exposure and subjects without, due to sensitization, would be interesting to elucidate in future studies.

The two groups A and B had some differences in the exposure, with Group B having both a longer exposure time and higher concentrations of cooking fumes. Nevertheless, the difference in exposure for Groups A and B should probably be regarded as minor and low compared to the levels of exposures that might be needed to induce easily detectable inflammatory changes. Thus, clear differences in the measured effects on inflammatory markers between the two groups may not have been expected. This may explain why the test of heterogeneity shows mostly no difference in the results between the two groups and supports the decision of interpreting the data for all 24 subjects together. However, when exploring the data separately for the two groups, one might get the impression that the exposure to cooking fumes had more effect on the subjects in Group A than the subjects in Group B, even though the subjects in Group B experienced a somewhat higher exposure. There might be unknown factors within the subjects making some of them more susceptible to being affected by the exposure. In the present study, with few subjects, there is a chance that such factors may be distributed unequally between the two groups A and B, contributing to such slightly paradoxical observations.

Apart from being non-smokers and free of respiratory disease, we applied no other inclusion criteria for the study subjects. It turned out that eight of the 24 subjects reported having had allergy. We lacked information about what kind of allergies, but none of the subjects had allergic symptoms when the investigations were done. However, two of them used antihistamine medication at the time of the experiment. When looking separately at the groups with reported allergy and the use of medication, they did not differ from the whole group with regard to any outcome, neither did excluding them from the statistical analysis change the results. At the time of the experiments, we tried to be careful to instruct the participants to act normally and without excesses of any kind during the 2 weeks. Even so, it cannot be ruled out that some of them have had variations in their day-to-day physical activities that may have influenced the results. Variation in outdoor temperatures during the days of the experiments is another factor that is beyond control. On the days of the present study, the mean temperatures ranged from –7.3 to + 16.8 °C for Group A and from +5.4 to + 19.4 °C for Group B. As this might have influenced the results, we would with hindsight, recommend performing such experimental studies under more stable climatic conditions.

It is conceivable that the levels of exposures to cooking fumes applied in our study were representative for conditions that can be found in both professional and domestic settings (Svendsen *et al*. 2002; Sjaastad and Svendsen 2009). In any case, the applied exposures must be regarded as moderate. As the exposures for both groups, and in particular for Group B, were at levels that led to some subjective discomfort, we did not find it appropriate to go any higher. Yet, the exposures may still have been too low and/or too short to cause the expected effect. However, in a similar study with exposure to UFP for 2 h, cardiac effects and mild inflammatory and prothrombic responses were shown (Samet *et al*. 2009).

Most previous studies on the effects from exposure to cooking fumes have looked at manifest diseases and chronic respiratory effects following chronic exposures (Coggon *et al*. 1986; Ng, Hui, and Tan 1993; Ng and Tan 1994; Zhong *et al*. 1999; Zhou *et al*. 2000; Svendsen, Sjaastad, and Sivertsen 2003). In the present study, we focused on the possible acute responses to short-term exposure to cooking fumes. To our knowledge, this is the first study to look at inflammatory markers in that context.

CONCLUSION

In our experimental setting, we were able to unveil only small changes in the levels of inflammatory markers in exhaled air and in blood after short-term exposure to moderate concentrations of cooking fumes. The applied methods can be of relevance for identifying reactions in the airway mucosa and for the prevention of chronic respiratory diseases.

SUPPLEMENTARY DATA

Supplementary data can be found at http://annhyg.oxfordjournals.org/.

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Paper III

ORIGINAL PAPER

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WORK ENVIRONMENT FACTORS AND WORK SUSTAINABILITY IN NORWEGIAN COOKS

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Abstract
Objectives: Cooks have increased morbidity and mortality. A high turnover has also been reported. We aimed to elucidate work environment and work sustainability in Norwegian cooks. **Material and Methods:** A questionnaire inquiring about working conditions and work participation was sent to 2082 cooks who had qualified from 1988 onwards. Of these, 894 responded. Time at work was analyzed with Kaplan-Meier plots and possible determinants for quitting work as a cook was analyzed with Cox regression. **Results:** The median time at work was 16.6 years. There were differences in sustainability between types of kitchens for both sexes ($p = 0.00$). The median time in the profession was 9.2 years for the cooks in restaurants, while the cooks in institutions and canteens showed a substantially higher sustainability with 75.4% still at work after 10 years, and 57% still at work after 20 years in the profession. Of those still at work as a cook, 91.4% reported a good or very good contentment, and the 67.4% who expected to stay in the profession the next 5 years frequently answered that excitement of cooking, the social working environment, and the creative features of cooking were reasons to continue. Musculoskeletal complaints were the most common health-related reason for leaving work as a cook, while working hours was the most common non-health-related reason. **Conclusions:** There are significant differences in work sustainability between the cooks in the different types of kitchens. The identified determinants for length of time in the occupation can be used for preventive purposes.

Key words:
Occupational health, Occupational exposure, Cooks, Work sustainability, Working hours, Musculoskeletal complaints

INTRODUCTION

Work-related health challenges have been reported among cooks [1–8]. Cooks face different physical risk factors including heavy lifting and repetitive movements, slippery floors, sharp objects, and hot equipment and substances [9,10]. An increased occurrence of musculoskeletal problems has been observed among cooks [3,11–13]. In accord with other studies we have previously shown that cooks are exposed to chemicals in cooking fumes such as polycyclic aromatic hydrocarbons (PAH), heterocyclic amines, and

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aldehydes [14–18]. Such exposures may be associated with rhinitis, respiratory disorders, and impaired lung function [19,20]. In a previous study we found an increased occurrence of respiratory symptoms in kitchen workers [4].

Forty percent of Norwegian cooks work irregular hours (6:00 p.m. – 6:00 a.m.) [21]. Shift-work has been found to reduce both length and quality of sleep, and is associated with several negative effects including mental, cardiovascular and reproductive health [22,23]. Long working hours are associated with sleep problems, mental disorders and coronary heart disease [24]. Psychosocial strain in the professional kitchen includes pace pressure [9,25] a high priority of customer service [9,26,27] strict hierarchical systems and instances of workplace bullying [28].

It has been a common assumption that Norwegian cooks have a high occupational turnover with a mean duration of work as a cook of only 6–7 years after qualifying. Work-related health problems have been seen as an important reason for leaving the profession. In a previous study on Danish employees 35–59 years of age, work environment factors and self-rated health were independent predictors for remaining in work 5 years later [29]. In a recent cross sectional study on Swedish employees, fair organizational climate, high control, and low physical demands were associated with remaining in work, with < 14 days sick leave the current year [30]. The aim of our study was to elucidate work sustainability in cooks and to analyse if there is a connection between work environment factors and work sustainability.

MATERIAL AND METHODS

In Norway, education as a cook can follow two different paths, one as a restaurant cook and one as a cook for institutions. The administrations in 3 counties in the middle part of Norway provided us with names and personal identification number of 2082 subjects who had qualified as either of these in the years from 1988 through 2008. Early in 2010 they were asked to respond to a mailed questionnaire inquiring about working conditions at the place where they were working at present or had worked before quitting if they had ceased working as a cook. Of these subjects, 894 responded. Of the non-respondents, 155 had wrong and untraceable postal addresses, 11 had emigrated, 4 were deceased, and 4 gave other reasons for not participating. Consequently, the response rate was 49.6%.

Table 1 shows background data for the cohort in relation to type of education, last or present place of work, and the type of kitchen with the longest held job for those who had worked ≥ 2 years. In this context, the workplaces were divided into "Institution/canteen," "À la carte/ethnic," "Hotel," and "Others" which included all other types of kitchens such as pizza restaurants and takeaways.

The time of inclusion into the cohort was the date given by the authorities when each participant had qualified as a skilled cook, and the date of quitting was the month and year given by the participants who had ceased working as a cook. The year of inclusion in the cohort was near to evenly distributed between the 2 types of training. Those with training for institutions had a peak recruitment in the 5 years from 1997 to 2001 with 34.9%, while the restaurant cooks had a peak recruitment a little later with 28.6% in the years 2001–2005 (Figure 1). For those who had not ceased, observation time was set to be through February 28, 2010.

As a basis for calculations of their longest held job, the participants were also inquired about the number of months they had previously worked in different types of kitchens. For those who had ceased working as a cook we asked for the reason, with the possibility to tag ≥ 1 of the following options: musculoskeletal complaints, respiratory complaints, skin complaints, income, stress, inconvenient working hours, tight job market, or other reasons.

All data were stored and analysed with the IBM® SPSS® Statistics program version 20. Work sustainability was analysed with Kaplan-Meier survival analysis in relation to type of education, last or present place of work and type of kitchen with the longest held job. Differences between

Table 1. Total respondents, proportion of those still working as a cook and respondents' characteristics at inclusion and when answering the questionnaire, by type of training, last or present place of work, and longest place of work for those who had worked for ≥ 2 years

Variable	Females				Males				Total			
	n	still at work [%]	age [years] (M±SD)		n	still at work [%]	age [years] (M±SD)		n	still at work [%]	age [years] (M±SD)	
			at inclusion	when answering			at inclusion	when answering			at inclusion	when answering
Respondents (total)	540	61.1	22.3±4.3	33.7±7.8	354	63.0	21.4±3.5	33.1±7.0	894	61.9	21.9±4.1	33.5±7.5
Type of training												
institution cook	299	60.2	23.2±5.0	34.6±8.2	102	59.9	22.3±5.0	34.3±6.8	401	59.1	23.0±5.0	34.5±7.9
restaurant cook	237	61.6	20.9±2.4	32.6±6.9	249	65.5	21.1±2.6	32.7±7.1	486	63.6	21.0±2.5	32.7±7.0
unknown	4				3				7			
Last or present place of work												
institution/canteen	303	73.3	22.8±4.9	34.7±7.7	124	71.0	21.8±4.0	34.7±6.8	427	72.6	22.5±4.6	34.7±7.4
à la carte/ethnic	112	42.0	21.6±4.0	31.8±8.2	121	57.9	20.9±3.0	31.1±7.2	233	50.2	21.3±3.5	31.4±7.7
hotel	75	48.0	21.1±2.1	31.7±6.4	88	65.9	21.2±2.9	33.0±6.5	163	57.7	21.1±2.5	32.4±6.5
other	50	50.0	22.2±3.9	35.4±7.8	21	33.3	22.4±5.1	35.7±7.4	71	45.1	22.3±4.3	35.5±7.7
Longest place of work (> 2 years of work in total)												
institution/canteen	108	74.3	23.2±4.9	35.9±7.5	103	69.0	22.5±5.0	36.3±6.8	211	72.8	23.0±4.9	36.0±7.3
à la carte/ethnic	94	53.7	22.0±4.0	34.6±7.7	100	64.1	21.1±2.7	33.0±6.0	194	58.8	21.6±3.5	33.8±6.9
hotel	218	60.6	20.8±2.0	32.7±6.5	84	65.0	21.3±3.4	33.7±6.4	302	62.9	21.1±2.8	33.2±6.4
other	18	66.7	23.2±5.2	36.6±7.7	8	75.0	21.9±2.1	38.8±4.2	26	69.2	22.8±4.5	37.2±6.8

M – mean; SD – standard deviation.

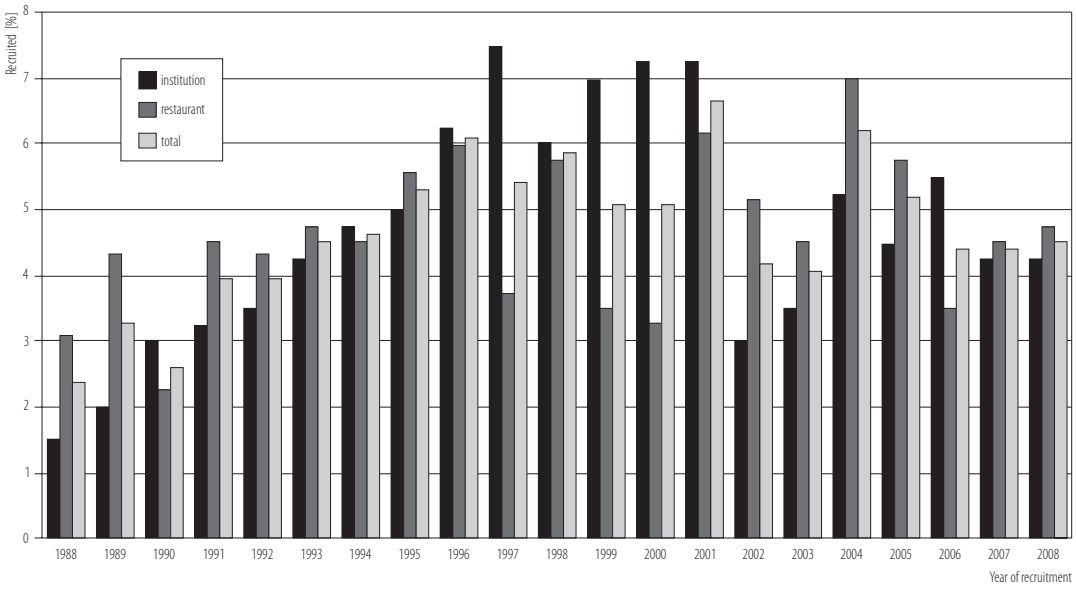

Fig. 1. Recruitment to the cohort 1988–2008 by specific type of training as a cook

groups were estimated by the Log Rank (Mantel Cox) test. In addition, we performed Cox regression analysis with adjustment for sex by the enter method in relation to last or present place of work, type of kitchen with the longest held job, shift work, working hours, type of food prepared, and some ergonomic factors. Other differences between groups were tested for statistical significance with a 2-tailed Pearson's Chi^2 test.

We also asked the participants how pleased they were with their job as a cook in general, and from those who were still working we asked if they anticipated that they would still be working as a cook after 5 years. We examined this in relation to the type of kitchen where they were working at present. Reasons for anticipating quitting or staying were also explored with the possibility to tag ≥ 1 options.

The study protocol was reviewed and approved by the regional committee for medical research ethics in central Norway (approval No. 2008/2527). Signed informed consent was obtained from each subject.

RESULTS

There was a certain migration between the 2 educational groups in the study. Of 401 who were originally trained as a cook for institutions, 130 (32.4%) had migrated and reported à la carte/ethnic, hotels or others as their last or present place of work. Of 486 originally trained for restaurants, 154 (31.7%) reported institutions/canteens as their last or present place of work.

The median time in the profession for all participants was 16.5 years. Figure 2 shows a Kaplan-Meier plot of sustainability for men and women separately in relation to last or present place of work. There were statistically significant differences in sustainability between the groups for both males and females ($p = 0.00$). For both sexes together the median time in the profession was 9.2 years for the cooks in restaurants and 11.5 years for the cooks in hotels. The cooks in institutions and canteens showed a substantially higher sustainability with 75.4% still at work after 10 years, and 57% still at work in the profession after 20 years.

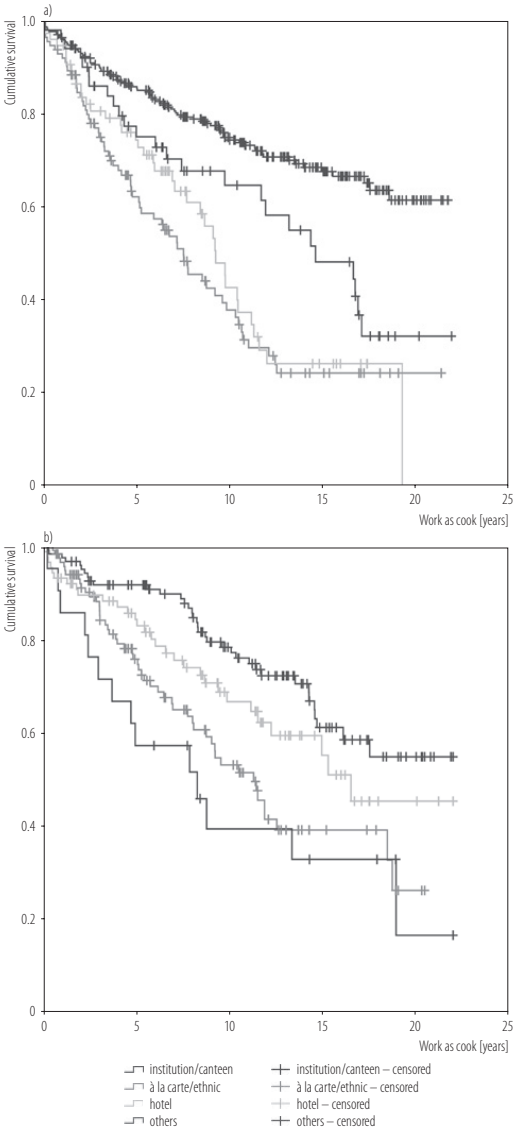

Fig. 2. Kaplan-Meier plots for sustainability as a cook for a) females and b) males separately in relation to last or present place of work as a cook

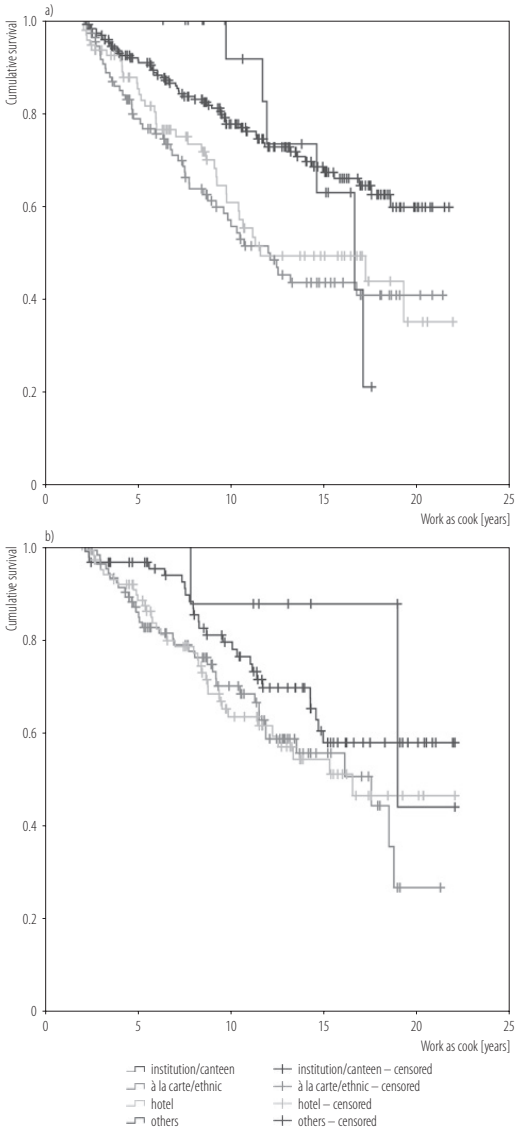

Fig. 3. Kaplan-Meier plots for sustainability as a cook for a) females and b) males in relation to type of kitchen with the longest held job as a cook

We also performed Kaplan-Meier analysis in relation to type of kitchen with "longest held job," (decided by calculating in what type of kitchen each subject had worked for the longest time). This analysis showed statistically significant differences in the sustainability between the different types of kitchens for females ($p = 0.00$), but not for males ($p = 0.142$) (Figure 3).

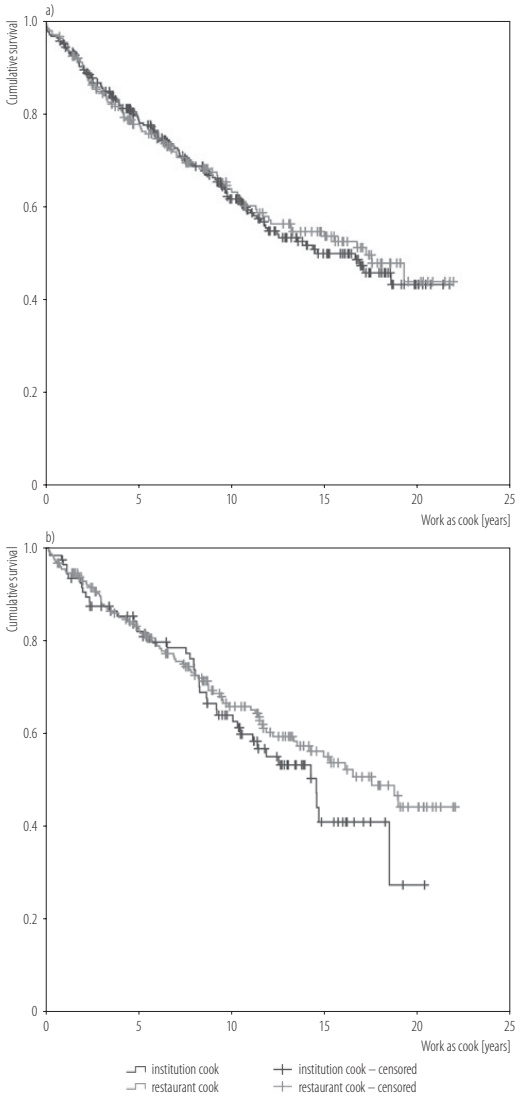

Fig. 4. Kaplan-Meier plots for sustainability as a cook for a) females and b) males in relation to type of training as a cook

In regard to type of education, the Kaplan-Meier plots were identical for females, while for the males the plot showed a better sustainability for restaurant cooks, without the difference being statistically significant (p = 0.3) (Figure 4).

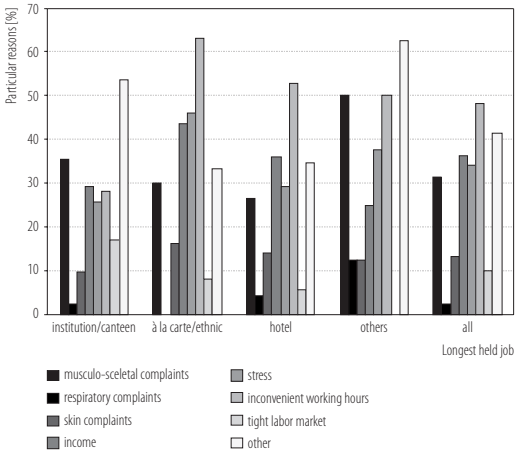

Fig. 5. Reasons given for quitting in relation to the longest held job in 249 who no longer worked as a cook

Seven subjects with unknown type of education were excluded from that analysis.

When looking at the reasons for having left work as a cook in relation to the longest held job in the 249 who had left the occupation, musculoskeletal complaints was the most common health-related reason with up to 50% in "other kitchens," while inconvenient working hours was the most common non-health-related reason with up to 63.2% in à la carte/ethnic and 52.8% in hotels (Figure 5). Institutions made an exception here with income as the most frequent non-health-related specific reason for having ceased working as a cook. When comparing cooks with their longest held job in institutions and canteens with all the others, the only reasons given that differed in a statistically significant manner were fewer complaints of inconvenient working hours (p = 0.0001) in the 1st group and that they more frequently reported anticipation of a tight job market (p = 0.001).

In search for determinants for having quitted work as a cook (survival), a Cox regression analysis adjusting for sex and some other factors was performed. Table 2 presents hazard ratios for quitting as a cook determined

by the regression analysis with the enter method and adjustment for sex in relation to last or present place of work, type of kitchen with the longest held job, shift work, working hours, type of food prepared, and some ergonomic factors. When comparing to institutions/canteens, those working in hotels or à la carte/ethnic restaurants showed increased hazard ratios in relation to both "last or present place of work" and "longest held type of kitchen."

Other determinants that showed statistically significant differences were related to shift work and weekly working hours. For the latter, those with working hours of > 50 h a week were shown to be less prone to quitting. Ergonomic factors like physically heavy work and monotonous and repetitive work did not turn out to be a determinant for quitting.

The 561 respondents who answered that they were still working as a cook, were asked about their

Table 2. Cox regression analysis in relation to last or present place of work, longest place of work for those with > 2 years at work in total, and some specific work characteristics

Variable	Respondents (N = 894) [n]	Hazard ratio
Last or present workplace		
institution/canteen	233	1.000
à la carte/ethnic	163	2.970**
hotel	427	2.200**
other	71	2.200**
Longest place of work (> 2 years of work in total)		
institution/canteen	211	1.000
à la carte/ethnic	194	1.940**
hotel	302	1.780**
other	26	0.928
Shift work (info from 874 respondents)		
only daytime	388	1.000
mostly late	56	3.340**
2 shift	321	1.810**
other schedules	109	1.120
Weekly working time (info from 870 respondents)		
< 20 h	36	1.050
20–39 h ("normal")	522	1.000
40–49 h	214	1.690**
> 50 h	98	0.600*
What kind of food is/was usually prepared		
about same amount of warm and cold	596	1.000
mostly cold	43	1.580**
mostly warm	229	0.726

Table 2. Cox regression analysis in relation to last or present place of work, longest place of work for those with > 2 years at work in total, and some specific work characteristics – cont.

Variable	Respondents (N = 894) [n]	Hazard ratio
To what extent is/was your work heavy		
little	62	1.000
< 1/2 of the time	408	1.020
> 1/2 of the time	402	0.880
To what extent is/was your work monotonous and repetitive		
small	85	1.000
moderate	297	1.370
large	491	1.280

* $p < 0.05$; ** $p < 0.000$.

general contentment with their work as a cook, and asked if they anticipated that they would still be working as a cook in 5 years time. Of those still working as a cook, 91.5% reported a very good or good contentment with their work as a cook. When stratifying the answers in regard to present work place, there were only small differences between the particular groups in regard to the general contentment, while there was a statistically significantly greater proportion of cooks in institutions/canteens (75.1%) compared to cooks in other workplaces (57.4%) who thought that they would continue as a cook after 5 years ($p = 0.0001$) (Table 3).

Out of those still working as a cook, 180 subjects (32.1%) stated that they thought they would not be working as a cook in 5 years. Among them, the reasons most frequently given for this were: musculoskeletal complaints (46.1%), income (52.2%), stress (52.2%) and their working hours (50.6%). In Figure 6 we have presented these reasons stratified by current workplace. Here the only statistically significant finding was that, compared to cooks in all other workplaces, the cooks in institutions/canteens less frequently reported "working hours" and "stress" as reasons to anticipating quitting, ($p = 0.0001$).

Table 3. Work contentment and anticipations among the respondents still working as cooks

Present place of work	Respondents (N = 551)				
	share anticipation to be at work in 5 years [n (%)]	general contentment with work [%]			
		very good	good	not too good	poor
Institution/canteen	232 (75.1)	40.3	52.6	6.2	1.0
À la carte/ethnic	61 (52.1)	37.1	48.3	13.8	0.9
Hotel	60 (63.8)	35.5	58.1	6.5	0.0
Other	18 (58.1)	45.2	48.4	3.2	3.2
All	371 (67.3)	39.1	52.4	7.7	0.9

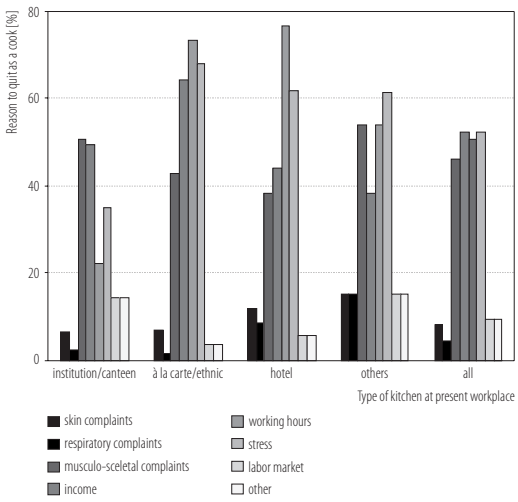

Fig. 6. Reasons given for possibly quitting as a cook during the next 5 years by 180 cooks who anticipated that they would not be working as a cook in 5 years time

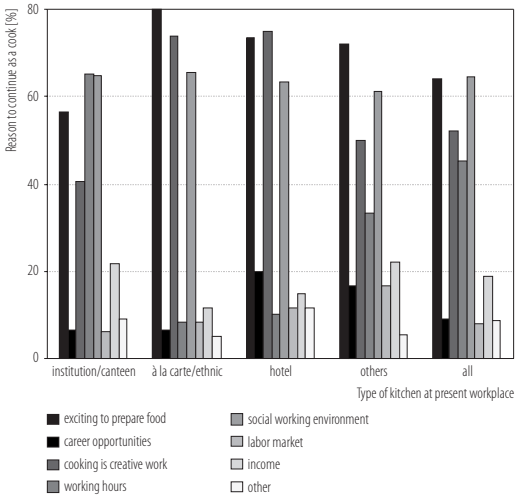

Fig. 7. Reasons given for wanting to stay as a cook by 371 cooks who anticipated that they would still be working as a cook in 5 years time

For the 371 who thought they would stay working as a cook, the most common reasons for staying were that it is exciting to prepare food (63.9%), that cooking is creative work (52%), and a good social working environment (64.4%) (Figure 7). Compared to cooks in institutions/canteens, a greater proportion of cooks in other workplaces found it more exciting to prepare food ($p = 0.004$) and, in particular for cooks working in à la carte restaurants and hotels, that cooking is a creative job ($p = 0.0001$). Cooks in institutions/canteens more frequently found that their working hours was a reason to continue as a cook ($p = 0.0001$).

DISCUSSION

This study shows by Kaplan-Meier plots that there are differences in work sustainability in cooks between the different types of kitchens, both in terms of last and present workplace for both sexes, and in terms of longest place of work for females. From the accessible literature, it seems well established that work environment factors in general have impact on health, work-wellbeing, and sickness absence, but there have been few publications on determinants of work sustainability apart from the previous study on Danish workers showing that work environment factors and self-rated health were independent predictors of remaining in work 5 years later [29]. Though not directly comparable, we find it reasonable to assume that the shown difference in work sustainability between the different types of kitchens in our cohort is determined by differences in work environment factors.

In à la carte restaurants the activities are directed by the current customer orders, usually with a fixed maximum delivery time, which may implicate an extremely high workload during the busiest periods. This may obstruct ergonomic work methods as well as hamper the possibility to take breaks when needed. À la carte restaurants usually have their busiest hours during late evenings, which also implicate a deviation from normal working hours. In canteens and institutions, it is often possible to plan the work in advance with a more even workload throughout the day, including the possibility to take breaks when needed.

It is known that shift work, long or irregular working hours can have negative impact on health [22–24]. In our Cox regression analysis, specific work-related factors like shift work and working hours showed a significant impact on work sustainability. Working mostly late shifts was the specific work environment factor that implicated the highest risk of quitting, while those working in a "two-shift" schedule also showed higher risk of quitting than those working only daytime.

Since 55.6% of the cooks in our cohort declared working shift work, and earlier studies have shown that 40% of the cooks in Norway work irregular hours [21], this may well be a major determinant of both health and sustainability in cooks. Such implications on the cooks' work sustainability are also supported by the reasons given by the respondents for anticipating to stay or to quit the profession. Convenient working hours was frequently stated as a reason to continue as a cook in institutions and canteens, while inconvenient working hours was frequently stated as a reason for quitting among those working in other types of kitchens. And, even though the cooks who were working in institutions and canteens less frequently stated that creative features of cooking was a reason to stay in the profession, a greater proportion of them (75.1%), compared to cooks in other workplaces (57.4%), thought that they would continue as a cook after 5 years.

In our study, the most frequently stated health-related reason for leaving the occupation was musculoskeletal complaints. Previous studies have shown that known risk factors for musculoskeletal disorders such as repetitive movements, lifting, and stress are common amongst professional cooks [9,10]. Previous studies have also reported an increased occurrence of musculoskeletal disorders in kitchen workers [3,11–13], but apart from one cross-sectional study in restaurant workers from Taiwan, the social consequences have mostly not been investigated. In the study from Taiwan it was found that the increased prevalence of work-related musculoskeletal disorders probably did not affect job performance or daily living [13]. This is in contrast to our finding with a significant effect on work sustainability due to musculoskeletal complaints. This may, however, be explained by methodological differences between the 2 studies, or reflect cultural and socioeconomic differences between Norway and Taiwan. As the recruitment to our study was based on the registers made at the time of qualifying as cooks (1988–2008), and information was collected retrospectively, we consider it to have a historic prospective approach. In contrast to a cross-sectional study, the relatively long follow up in our study might have reduced selection bias and strengthened the possibility to elucidate health-related social consequences. Having to end a professional career due to work-related health complaints is a serious problem both for the individual and the society. The fact that data was collected at the end of the observation period with a mailed questionnaire can, however, have introduced information bias, in particular when inquiring about complaints and consequences simultaneously [31].

As cooking fumes contain known airway irritants [14–18], the low rate of respiratory complaints as a reason for quitting was unexpected. We have previously shown that cooks have an increased prevalence of both dyspnea and other respiratory symptoms [4]. This may still be the case in the present cohort, but apparently the cooks did not quit because of such complaints. Another explanation could also be the relatively young age of the cohort, and the picture might have looked differently in a group of older cooks.

The low response rate of just below 50% in our study could have introduced another source of selection bias. We did, therefore, perform a non-responder inquiry by telephone calls to non-responders asking them only if they were still active as a cook. There were 50% of the 46 non-responders that were reached who were still in the profession, while the corresponding figure for the original responders was 61.9%. With this small difference, there is little

reason to assume that the low participation rate biased the results decisively.

There were no statistically significant differences in sustainability between cooks originally trained for restaurants and cooks originally trained for institutions. We did, however, observe that there had been a considerable migration from type of education to current place of work, and this was, to our surprise, similar in both directions. We expected that those originally trained for restaurants would show a greater migration towards work in institutions and canteens in order to seek more favourable working hours and less stress. There might, however, still be different reasons for migrating, such as to seek excitement in the restaurants and to seek more quiet work, and more favourable working hours in the institutions. Thus, the migration pattern may have diluted possible differences in sustainability between the different types of educations. Anyway, it seems that place of work is more important than type of education as a determinant of work sustainability.

Work environment factors may influence migration and work sustainability differently in males and females. Females are often expected to take responsibility for family and children and might therefore tend to leave work places with shift work, and long working hours earlier in their career than males. Such differences in migration between jobs may also explain why differences in sustainability in relation to the longest held job was statistically significant for female cooks, but not for male.

With a median sustainability in the profession 14–18 years, the results showed that Norwegian cooks stay longer in their profession than the 6–7 years previously assumed. We do, however, not know the significance of this compared to other occupations. In our view, having a median time in the occupation of 16.5 years in relation to a maximum observation time of 21 years can be considered as acceptable. It should also be recognised that the relatively young cooks who participated in our study, independent of their place of work, seem to have a high contentment with their work as a cook, and that the majority of those who anticipate to stay in the occupation think of their work as both exciting and creative. An advice to employers of cooks who want to stabilize their workforce should be to care for the working environment in general, to intervene with work organization and working hours, and sustain the features that have been shown to be appreciated by cooks.

CONCLUSIONS

There are significant differences in work sustainability between the cooks in the different types of kitchens, and the work sustainability is determined both by work- and health-related factors, the most important being working hours and musculoskeletal complaints.

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Paper IV

Is not included due to copyright

Supplementary material Paper II

Online resource 1 – Inflammatory markers before and after the stay in the kitchen with exposure to normal air and with exposure to cooking fumes for each of the two groups A and B. Data are given as the geometric mean (95% confidensinterval).

			Exposure to normal air			Exposure to cooking fumes		
	Timing of sample:		Before exposure[0]	Immediately after exposure[1]	24 hours after start of exposure[2]	Before exposure[0]	Immediately after exposure[1]	24 hours after start of exposure[2]
		Groups						
Exhaled air:	Ethan (ppb)	A	2.91 (2.58–3.28)	2.85 (2.53–3.21)	2.65 (2.29–3.07)	2.83 (2.51–3.19)	3.09 (2.74–3.48)	3.53 (3.13–3.98)
		B	1.70 (1.49–1.93)	1.84 (1.57–2.15)	2.03 (1.78–2.31)	2.02 (1.77–2.30)	2.18 (1.91–2.48)	2.13 (1.85–2.45)
	eNO (ppb)	A	5.52 (4.49–6.82)	6.25 (5.08–7.70)	6.01 (4.88–7.40)	5.13 (4.16–6.32)	5.51 (4.47–6.78)	5.78 (4.70–7.12)
		B	4.44 (3.50–5.63)	4.19 (3.30–5.31)	4.66 (3.67–5.91)	5.10 (4.02–6.47)	4.94 (3.90–6.27)	4.77 (3.76–6.05)
EBC:	IL-1β (pg/ml)	A	1.39 (0.94–2.07)	0.87 (0.58–1.29)	1.01 (0.68–1.50)	1.04 (0.79–1.38)	1.39 (1.03–1.88)	1.07 (0.79–1.44)
		B	0.70 (0.49–0.99)	0.87 (0.61–1.23)	0.74 (0.52–1.04)	0.81 (0.57–1.15)	0.98 (0.69–1.38)	0.89 (0.63–1.25)
	8-isopr. (pg/ml)	A	2.98 (1.82–4.89)	5.15 (3.14–8.44)	2.95 (1.80–4.84)	10.26 (7.19–14.63)	5.30 (3.64–7.74)	3.36 (2.35–4.79)
		B	5.06 (3.61–7.09)	3.36 (2.39–4.70)	3.12 (2.23–4.37)	2.96 (2.11–4.15)	2.51 (1.79–3.52)	3.18 (2.27–4.46)
	LTB_4 (pg/ml)	A	2.44 (1.59–3.73)	3.57 (2.33–5.47)	2.63 (1.72–4.03)	5.69 (4.12–7.86)	4.05 (2.94–5.60)	2.82 (2.04–3.89)
		B	8.13 (6.38–10.35)	7.33 (5.75–9.34)	6.91 (5.42–8.80)	8.43 (6.61–10.74)	6.30 (4.94–8.03)	7.89 (6.19–10.05)
Serum:	Fibrinogen(g/dl)	A	2.61 (2.38–2.86)	2.63 (2.40–2.88)	2.67 (2.44–2.92)	2.70 (2.46–2.95)	2.74 (2.50–3.00)	2.80 (2.56–3.07)
		B	2.72 (2.38–3.12)	2.68 (2.34–3.07)	2.60 (2.27–2.98)	2.66 (2.32v3.04)	2.46 (2.15–2.81)	2.61 (2.28–2.99)
	IL-6 (pg/ml)	A	0.52 (0.30–0.90)	0.49 (0.28–0.84)	0.34 (0.19–0.58)	0.39 (0.22–0.67)	0.55 (0.32–0.95)	0.37 (0.21–0.66)
		B	0.35 (0.26–0.48)	0.35 (0.26–0.48)	0.33 (0.24–0.45)	0.30 (0.22–0.40)	0.30 (0.22–0.40)	0.28 (0.21–0.39)
	d-dimer (mg/ml)	A	0.31 (0.23–0.43)	0.31 (0.23–0.43)	0.25 (0.18–0.34)	0.28 (0.21–0.39)	0.30 (0.22–0.40)	0.31 (0.23–0.43)
		B	0.26 (0.22–0.32)	0.25 (0.20–0.29)	0.25 (0.21–0.30)	0.26 (0.22–0.31)	0.26 (0.22–0.32)	0.26 (0.21–0.31)

0) In the morning before entering the kitchen

1) When leaving the kitchen after two hours (group A) or four hours (group B)

2) Twenty-two hours after leaving the kitchen (group A) or 20 hours after leaving the kitchen (group B).

Appendix I

Spørreskjema om arbeidsmiljø og helse blant kokker i Midt-Norge

1. Arbeider du som kokk?

☐ Ja → *Gå videre til spørsmål 4*

☐ Nei

2. Vennligst angi måned og årstall du sluttet som kokk: ☐☐ Måned ☐☐☐☐ Årstall

3. Hva er grunnen til at du ikke jobber i kokkeyrket nå?
(Sett ett eller flere kryss)

☐ Muskel- og leddplager
☐ Luftveisplager (for eksempel astma)
☐ Hudplager
☐ Lønn
☐ Stress
☐ Ugunstige arbeidstider
☐ Dårlig arbeidsmarked (nedlegging / innskrenking)

☐ Annet I tilfelle hva? _____

4. Ved hvilken type kjøkken har du arbeidet tidligere?
Fyll ut samlet varighet (antall måneder) på de forskjellige typer kjøkken

Skriv 5 som: ☐☐5 Skriv 10 som: ☐10 Skriv 120 som: 120

☐ A la carte restaurant	☐☐☐	antall måneder
☐ Fastfood/gatekjøkken	☐☐☐	antall måneder
☐ Pizzarestaurant	☐☐☐	antall måneder
☐ Institusjonskjøkken	☐☐☐	antall måneder
☐ Hotellkjøkken	☐☐☐	antall måneder
☐ Kantine	☐☐☐	antall måneder
☐ Etnisk restaurant (kinesisk, indisk etc)	☐☐☐	antall måneder

5. Hvis du har jobbet ved etnisk restaurant, hvilken type?
(Sett ett eller flere kryss)

☐ Italiensk ☐ Kinesisk ☐ Indisk ☐ Tyrkisk ☐ Gresk ☐ Annet

Spørsmålene nedenfor gjelder nåværende eller siste arbeidssted som kokk. Dersom du har sluttet i yrket, fyll ut for siste arbeidssted der du arbeidet som kokk.

6. Hvilken type kjøkken jobber du ved nå?

- ☐ A la carte
- ☐ Fastfood/gatekjøkken
- ☐ Pizzarestaurant
- ☐ Institusjonskjøkken
- ☐ Hotellkjøkken
- ☐ Kantine
- ☐ Etnisk restaurant (kinesisk, indisk etc)

7. Hvis du jobber ved etnisk restaurant, hvilken type?

☐ Italiensk ☐ Kinesisk ☐ Indisk ☐ Tyrkisk ☐ Gresk ☐ Annet

8. Når begynte du ved din nåværende arbeidsplass? ☐☐ Måned ☐☐☐☐ Årstall

9. Hva slags skift jobber du vanligvis?
(Sett ett kryss)

☐ Dag ☐ Kveld ☐ To-skiftordning ☐ Annet

10. Hvor mange timer arbeider du i gjennomsnitt pr uke?
(Sett ett kryss)

☐ Mindre enn 20 timer ☐ 20-39 timer ☐ 40-50 timer ☐ Mer enn 50 timer

11. Lages det hovedsakelig varm mat eller kald mat ved ditt arbeidssted?
(Sett ett kryss)

☐ Varm mat ☐ Kald mat ☐ Omtrent like mye varm og kald mat

12. Hvor stor del av din arbeidsdag består av steking på plate/grill/stekepanne?
(I gjennomsnitt gjennom en måneds arbeid)

- ☐ Mer enn halvparten av tiden
- ☐ Mindre enn halvparten av tiden
- ☐ Jeg steker ikke på plate/grill/stekepanne → *Gå videre til spørsmål 19*

13. Hvilken stekeinnretning benyttes mest?
(Sett ett eller flere kryss)

☐ Stor stekeplate ☐ Stekepanne ☐ Grill

14. Hva stekes det mest av på plate/grill/i stekepanne?
(Sett inntil 3 kryss)

- ☐ Hamburgere/pølser
- ☐ Biffkjøtt
- ☐ Fisk
- ☐ Kylling/andre typer fjærfe
- ☐ Svinekjøtt/bacon
- ☐ Annet

15. Hva slags stekefett benyttes mest?
(Sett ett kryss)

☐ Vegetabilsk olje ☐ Smør ☐ Margarin ☐ Kombinasjon av smør og olje

16. Brukes gass til steking? ☐ Ja ☐ Nei

17. Hvordan synes du at avtrekkshetta over stekesonen fungerer?

☐ Svært bra ☐ Bra ☐ Dårlig ☐ Svært dårlig ☐ Vet ikke

18. Brukes vifta i avtrekkshetta over stekesonen for det meste på full effekt?

☐ Ja ☐ Nei ☐ Vet ikke

19. Brukes gass som energikilde til annen matlaging enn steking? ☐ Ja ☐ Nei

20. Brukes det frityrgryte i kjøkkenet? ☐ Ja ☐ Nei

21. Hvor stor del av din arbeidsdag består av stående/gående arbeid?

- ☐ Mer enn halvparten av tiden
- ☐ Mindre enn halvparten av tiden
- ☐ Svært liten del av tiden

22. Hvor stor del av din arbeidsdag vil du si innebærer fysisk tungt arbeid?

- ☐ Mer enn halvparten av tiden
- ☐ Mindre enn halvparten av tiden
- ☐ Min arbeidsdag innebærer svært lite fysisk tungt arbeid

23. I hvilken grad innebærer dine arbeidsoppgaver ensidig gjentakende bevegelser?

☐ Svært stor grad ☐ Stor grad ☐ Moderat grad ☐ Liten grad ☐ Svært liten grad

24. Har du noen gang mens du har arbeidet som kokk hatt smerter/stivhet i muskler eller ledd som har vart i 3 måneder eller mer?

☐ Ja
☐ Nei → *Gå videre til spørsmål 29*
☐ Vet ikke → *Gå videre til spørsmål 29*

25. Hvilke kroppsdeler har du i tilfelle merket disse plagene fra?
(Sett ett eller flere kryss)

☐ Nakke
☐ Skuldre/aksler
☐ Øvre del av ryggen
☐ Albuer
☐ Korsryggen
☐ Handledd/hender
☐ Hofter
☐ Knær
☐ Ankler/føtter

26. Har du hatt disse plagene i 3 måneder eller mer i løpet av det siste året?

☐ Ja ☐ Nei ☐ Vet ikke

27. Har disse plagene i løpet av den siste måneden hindret deg i å utføre daglige aktiviteter?

I arbeidet ☐ Ja ☐ Nei ☐ Vet ikke

I fritida ☐ Ja ☐ Nei ☐ Vet ikke

28. Tror du disse plagene har sammenheng med belastninger i arbeidet som kokk?

☐ Ja ☐ Nei ☐ Vet ikke

29. Er arbeidet ditt så fysisk anstrengende at du ofte er sliten i kroppen etter en arbeidsdag?

☐ Ja, nesten alltid ☐ Ganske ofte ☐ Ganske sjelden ☐ Aldri eller nesten aldri

30. Er det mye stress og mas i arbeidet ditt?

☐ Ja, nesten hele tida ☐ Ja, en god del ☐ Sjelden ☐ Nei, ikke i det hele tatt

31. I hvilken grad har du selv innflytelse på din arbeids<u>mengde</u>?

☐ Svært stor grad ☐ Stor grad ☐ Moderat grad ☐ Liten grad ☐ Svært liten grad

32. I hvilken grad har du selv innflytelse på ditt arbeids<u>tempo</u>?

☐ Svært stor grad ☐ Stor grad ☐ Moderat grad ☐ Liten grad ☐ Svært liten grad

33. Hvordan er mulighetene i jobben din til å utnytte ferdigheter, kunnskaper og erfaring du har fått gjennom utdanning og arbeid?

☐ Svært gode ☐ Gode ☐ Dårlige ☐ Svært dårlige

34. Hvordan er mulighetene i jobben din til å videreutvikle deg faglig på de områdene du ønsker?

☐ Svært gode ☐ Gode ☐ Dårlige ☐ Svært dårlige

35. Har du merket luftveisplager i forbindelse med jobb?

☐ Ja
☐ Nei → *Gå videre til spørsmål 39*
☐ Vet ikke → *Gå videre til spørsmål 39*

36. I tilfelle ja, hva slags luftveisplager?
(Sett ett eller flere kryss)

☐ Hoste med oppspytt
☐ Tørrhoste
☐ Tungpust
☐ Pipende pust

☐ Annet _____

37. Hvor ofte har du slike plager?

☐ Daglig/nesten daglig ☐ Hver uke ☐ 1 – 2 ganger per måned ☐ Mer sjelden

38. Blir du bedre av dine luftveisplager i helger/ferier?

☐ Ja ☐ Nei ☐ Vet ikke

39. Har du hatt daglig hoste og oppspytt i mer enn tre måneder per år i minst to år på rad?

☐ Ja ☐ Nei

40. Hadde du fått påvist sykdom i luftveiene av lege før du begynte å arbeide som kokk?

☐ Ja
☐ Nei → *Gå videre til spørsmål 43*
☐ Vet ikke → *Gå videre til spørsmål 43*

41. I tilfelle hva slags sykdom?
(Sett ett eller flere kryss)

☐ Astma
☐ Kronisk bronkitt
☐ KOLS

☐ Annet _____

42. Har du merket forverring av luftveissykdommen(e) i forbindelse med arbeid som kokk?

☐ Ja ☐ Nei ☐ Vet ikke

43. Har du <u>som barn</u> hatt noen av de følgende sykdommer?
(Sett ett eller flere kryss)

☐ Ja, eksem
☐ Ja, astma
☐ Ja, allergi
☐ Nei, jeg har ikke hatt noen av disse sykdommene som barn

44. Har du <u>nå</u> noen av de følgende sykdommer
(Sett ett eller flere kryss)

☐ Ja, eksem
☐ Ja, astma
☐ Ja, allergi
☐ Nei, jeg har ikke noen av disse sykdommene nå → *Gå videre til spørsmål 47*

45. Hvis du er allergisk, hva er du i tilfelle allergisk mot

_____, _____, _____

46. Har du merket forverring av eksem/astma/allergi i forbindelse med arbeid?
(Sett ett eller flere kryss)

☐ Ja, eksem
☐ Ja, astma
☐ Ja, allergi
☐ Nei, jeg har ikke merket forverring av noen av disse i forbindelse med arbeid

47. Røyker du daglig for tiden?

☐ Ja
☐ Nei → *Gå videre til spørsmål 50*

48. Hvor mange sigaretter røyker du i tilfelle om dagen? ☐☐

49. Hvor mange år har du til sammen røykt? ☐☐

Viss du har svart på spørsmål 48 og 49, kan du gå videre til spørsmål 54.

50. Har du røykt tidligere?

☐ Ja
☐ Nei → *Gå videre til spørsmål 54*

51. Hvor lenge er det siden du sluttet å røyke?

☐ Mindre enn 3 måneder ☐ 3 måneder - 1 år ☐ 1-5 år ☐ Mer enn 5 år

52. I hvor mange år røykte du? ☐☐

53. Hvor mange sigaretter røykte du pr dag? ☐☐

54. Hvor mange enheter alkohol drikker du i løpet av en vanlig uke?
(En enhet alkohol tilsvarer 1 glass vin, 1 liten flaske øl eller 1 drink)

☐ 0 (Totalavholdende) ☐ Mindre enn 1 enhet i uka ☐ 1-4 ☐ 5-10 ☐ Mer enn 10

Hvis du har sluttet i kokkeyrket kan du avslutte her. Hvis du fortsatt arbeider som kokk vær vennlig og besvar resten av spørsmålene.

55. Har du i løpet av de siste 12 månedene hatt sykefravær?
(Sett ett eller to kryss)

☐ Ja, med egenmelding
☐ Ja, med sykmelding fra lege
☐ Nei → *Gå videre til spørsmål 57*

56. Hvor lenge har du hatt sykefravær til sammen i løpet av de siste 12 månedene?

☐ 2 uker eller mindre ☐ 2-8 uker ☐ Mer enn 8 uker

57. Har du i løpet av de siste 12 månedene vurdert å skifte yrke eller arbeidsplass?

☐ Ja ☐ Nei

58. Hvordan trives du alt i alt med arbeidet som kokk?

☐ Veldig godt ☐ Godt ☐ Ikke særlig godt ☐ Dårlig

59. Tror du at du er i arbeid som kokk om 5 år?

☐ Ja → *Gå videre til spørsmål 61*
☐ Nei

60. Hvis nei på spørsmål 59, hva tror du i tilfelle ville være årsak til at du ikke er i arbeid som kokk om 5 år? (Sett ett eller flere kryss)

☐ Hudplager
☐ Luftveisplager (som astma, KOLS eller lignende)
☐ Lønn
☐ Ugunstige arbeidstider
☐ Stress
☐ Dårlig arbeidsmarked (nedlegging / innskrenking)
☐ Muskel- og leddplager

☐ Annet_____

61. Hvilke sider ved arbeidet du har nå taler mest for at du kommer til å fortsette i kokkeyrket? (Sett ett eller flere kryss)

☐ Spennende å lage mat
☐ Gode karrieremuligheter
☐ Kreativt arbeid
☐ Gunstige arbeidstider
☐ Godt sosialt arbeidsmiljø
☐ Bra arbeidsmarked
☐ Lønn

☐ Annet_____

Takk for at du tok deg tid til å svare på spørsmålene.
Vennligst husk å skrive under samtykkeerklæringen på neste side før du sender inn skjemaet!