Results Methodologically simple research papers identified targets for disease prevention early on in both industries: from 1918 for silicon carbide, and from 1936 for primary aluminium. Later and more complicated studies of disease mechanisms, and studies involving detailed exposure characterisations, do not seem to have served preventive practice to any great extent. The scientific community tends to support stakeholders request for more research before lowering of TLVs or reducing exposure. Disagreement about what constitutes evidence has delayed prevention and stimulated research, but the research questions were not always relevant for prevention. The Norwegian regulatory model, with environmental standards based on tripartite consensus, may have discouraged technological innovation.
Conclusions Regulatory authorities must accept documentation of harmful exposure as sufficient evidence, long before the scientific community is ready to reject the null hypothesis of no risk. Quasi-experimental prevention can eradicate disease earlier than prevention based on too much evidence. But we may never know exactly why our efforts seemed to work.

Session: 7. Occupational asthma

262 SHORT-TERM LUNG FUNCTION EFFECTS AFTER OCCUPATIONAL EXPOSURE TO CLEANING PRODUCTS

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Objective To evaluate the acute effects of exposure to cleaning products on lung function of female cleaning workers.
Methods A panel study including 21 female cleaners with persistent asthma symptoms was nested within a case-control study. Participants recorded the use of cleaning products in 2-week diaries resulting in 312 person-days. All participants were trained to perform lung function testing using a PIKO-1® device to measure FEV_1 (mL) and PEF (L/min) three times per day (in the morning after waking-up, at midday and in the evening before going to sleep). Associations between cleaning products and FEV_1 and PEF in the evening of the same day of exposure, in the morning next day and FEV_1 and PEF's diurnal variation (amplitude over daily mean) were evaluated using linear mixed regression analysis. All models included a random term for individual and were adjusted for age, height, number of cigarettes smoked, respiratory infection, and respiratory medication. The reference category for all comparisons was "No use of cleaning products".
Results Evening FEV_1 and PEF were 8.7 ml (95% confidence interval (CI) 1.7–15.7) and 36.9 l/min (CI 4.3–69.5), lower on days when three or more cleaning sprays were used, respectively (p-values for trend: 0.054 for FEV_1 and 0.053 for PEF). Evening FEV_1 significantly decreased after exposure to hydrochloric acid (30.8 ml) and solvents (37.6 ml). Diurnal variation in FEV_1 increased on days using ammonia (12.7%), lime-scale removers (9.3%), air-fresheners (7.2%) and multiuse products (6.8%). Diurnal variation in PEF increased on days using ammonia (17.0%), lime-scale removers (13.0%), powder detergents (11.4%), and air-fresheners (8.6%). Morning FEV_1 decreased on days following the use of solvents (53.0; 36.3–69.6), hydrochloric acid (26.3 ml; CI: 14.7–37.9), powder detergents (26.1; 16.7–35.6), and degreasers (19.1; 12.6–25.7).
Conclusions Acute changes in lung function suggest that the use of specific cleaning products may exacerbate pre-existing asthma.

263 THE RISK OF REDEMPTION OF ASTHMA PHARMACEUTICALS AMONG WELDERS: A NATIONWIDE FOLLOW-UP STUDY

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Objectives The purpose was to examine if stainless steel and mild steel welding confers an increased risk of bronchial asthma.
Methods A Danish national company-based historical cohort of 5,499 ever-welders and 1,514 never-welders was with the Danish Medicinal Product Registry followed from 1995 through 2011 to identify the first-time redemption of asthma pharmaceuticals including Beta-2-adrenoreceptor agonists, Adrenergic and other drugs for obstructive airway diseases, Inhaled Glucocorticoids. Lifetime exposure to welding fume particulates was estimated by combining questionnaire data on welding work with a welding exposure matrix based on more than 1000 personal measurements of ambient air concentrations of welding fume particulates. The estimated exposure accounted for calendar-time, welding intermittence, type of steel, welding methods, local exhaustion and welding in confined spaces. Hazard ratios (HR) with 95% confidence intervals (CI) were calculated using a Cox proportional hazards model adjusting for potential confounders.
Results The average incidence of redemption of asthma pharmaceuticals in the cohort was 17/1000 years (95% CI 0.01–0.02). Asthma pharmaceuticals were not redeemed more often among stainless steel (n = 3874) and mild steel welders (n = 1625) than among never-welders. Among ever-welders redemption of asthma pharmaceuticals was not related to life-long exposure to welding fume. Analyses of specific subgroups of asthma pharmaceuticals did not reveal consistent associations with welding exposure. However, among non-smoking stainless steel welders the risk increased with cumulative welding dust exposure (HR for high- vs. low level exposed 1.41, 95% CI 1.06–1.89).
Conclusions The results showed no consistent association between lifetime exposure to welding fume and use of asthma pharmaceuticals. However, an increased risk of asthma pharmaceuticals among non-smoking stainless welders may indicate that stainless welding does confer an increased risk of asthma, which escapes detection among smokers having a high prevalence of obstructive airway disease.

264 OCCUPATIONAL ASTHMA IN NEW ZEALAND SAWMILL WORKERS: A LONGITUDINAL STUDY

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Objectives Wood dust is known to be associated with a range of respiratory effects including reduced lung function, increased

bronchial responsiveness and occupational asthma. Cross-sectional studies have suggested an excess of asthma symptoms and lung function decline in sawmill workers. The study aims were to measure the incidence of new-onset asthma, to examine longitudinal changes in lung function, and associations with dust exposures.

Methods Associations between dust exposures, asthma symptoms and lung function were studied over three years in a prospective cohort of sawmill workers. Respiratory morbidity was assessed using spirometry and symptom questionnaires, and personal dust exposure was measured, initially on recruitment into the study and then annually for at least two years.

Results We recruited 281 sawmill workers from seven sawmills. Wood dust sampling (n = 300) showed an overall average of GM = 0.6 mg/m^3 (GSD 3.41), range <0.1 to 16.9 mg/m^3. Asthma symptoms were more common in workers with high exposure compared to the reference group with low exposures (OR = 1.31, 95% CI = 0.45–3.83). The incidence of new-onset asthma was 4.6% overall, and 8% among those with high "dry"-dust exposure. In the longitudinal follow-up of workers a clear decline in forced expiratory volume in 1 second (FEV_1) and maximal mid expiratory flow (MMEF) was evident in the high exposure group, when compared with the non/low-exposed. Although the numbers were small, the decline over the 3 years in both FEV_1 and MMEF was statistically significant in the high exposure group.

Conclusions This study confirms that New Zealand sawmill workers generally experience levels of dust exposure below most exposure standards. Notwithstanding this, there is evidence of an increased risk of developing asthma symptoms and lung function decline (of an obstructive nature) over time even at low dust levels, with a suggestion of a dose-response relationship between dust levels and the development of symptoms.

265 INCIDENCE OF SELF REPORTED ASTHMA OR WHEEZE AMONG WOMEN IN WELDING AND ELECTRICAL TRADES IN THE WHAT-ME STUDY IN CANADA

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Objectives The WHAT-ME study (Women's Health in Apprenticeship Trades-Metalworkers and Electricians) was established because of concerns about risk to the fetus of women welding during pregnancy. Prospective data on work-related health outcomes are also collected. The potential of the study is investigated using 'new-onset asthma or wheeze' as an example.

Methods Women in registered apprenticeships since 2005 in welding, boiler-making, steam fitting/pipefitting ('welders') or electrical trades are invited to join the study. They complete questionnaires on health and exposure at baseline and subsequently every six months. Exposure data are based on the last day at work at each contact, and include information on hazards encountered for each task performed. For the analyses presented here a woman was considered currently working within her trade if working as a welder or electrician at the time of the interview or in the preceding month.

Results To date 496 women have signed up, and recruitment is underway across Canada. The results here are from 385 early recruits (mean age 31.6 years) with completed baseline questionnaires: 220 women, analysed here, have also completed the first (6 month) follow-up questionnaire. At baseline more welders (28%) than electricians (18%) were current smokers ($p < 0.01$). Amongst those working since baseline, and who did not initially report asthma/wheeze, 13/95 welders and 3/65 electricians reported asthma/wheeze at 6 months. In a logistic regression analysis, adjusting for smoking, months working in their trade since baseline, and current employment in the trade, welders had an odds ratio of 3.23 (95%CI 0.85–12.18) for new-onset asthma or wheeze.

Conclusions Female welders appear at higher risk than female electricians of reporting new-onset asthma or wheeze. The continuing recruitment and follow-up of the cohort will provide increasing power for this analysis and information on tasks within trade will help identify recommendations for prevention.

266 ENDOTOXIN AND GLUCAN EXPOSURE PROTECTS AGAINST ATOPY AND HAY FEVER: A LONGITUDINAL STUDY

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Objectives We previously showed that occupational endotoxin exposure in agricultural workers was associated with wheeze and negatively associated with atopy. We recently completed a 5"year follow" up of the initial study population. This study aimed to analyse change and persistence in status of atopic sensitizaton, (self reported) allergy, hay fever and wheeze in relation to baseline endotoxin and glucan exposure.

Methods We studied an occupational cohort of 259 Dutch farmers and agricultural workers recruited in 2006 and followed up in 2011. Endotoxin and glucan exposure were assigned based on measurements in a subset of the population and exposure modelling. Allergic sensitisation to common allergens (house dust mite, grass, cat, and dog) was based on serum IgE. Atopy was defined as sensitisation against ≥one common allergens. Self-reported wheeze, allergy and hay fever were determined by questionnaire. Associations between exposure and health outcomes were analysed by multinomial logistic regression using four categories based on presence or absence of the outcome at baseline and at follow-up. Analyses were adjusted for possible confounders age, gender, smoking and farm childhood.

Results Baseline glucan and endotoxin exposure levels were highly correlated ($r > 0.9$). Glucan and endotoxin exposure were negatively associated with persistent reporting of hay fever symptoms (OR 0.58, 95% CI [0.41-0.82] and 0.69 [0.48-0.98] respectively) or atopy (OR 0.7 [0.56-0.90] and 0.74 [0.56-0.98] respectively). Higher endotoxin exposure tended to be negatively associated with persistent self reported allergy and sensitisation against grasspollen (OR 0.84 [0.68-1.02] and 0.79 [0.62-1.02]). Presence of house dust mite-specific IgE or wheeze was not associated with glucan or endotoxin exposure. Changes in outcome status were rare and not clearly associated to exposure.

Conclusions Exposure to both endotoxin and glucan protects from persistent atopic disease. These results could be suggestive of a healthy worker selection. However, a previously performed healthy worker survivor analysis showed no such selection.