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

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 use diaries during their working hours. 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 workers (n = 3874) and mild steel welders (n = 1625) than among never-welders. Among ever-welders, asthma pharmaceuticals were not related consistent associations with welding exposure. However, among non-smoking stainless steel welders the risk increased with cumulative welding dust exposure (HR = 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 diseases.

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

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Objective: 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 linked to the Danish Medication Product Registry followed from 1995 through 2011 to identify the first-time redemption of asthma pharmaceuticals including beta-2-adrenoceptor agonists, anti-asthmatic 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 100 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 welders (n = 3874) and mild steel welders (n = 1625) than among never-welders. Among ever-welders, asthma pharmaceuticals were not related consistent associations with welding exposure. However, among non-smoking stainless steel welders the risk increased with cumulative welding dust exposure (HR = 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 diseases.

OCCUPATIONAL ASTHMA IN NEW ZEALAND SAWMILL WORKERS: A LONGITUDINAL STUDY

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Objective: Wood dust is known to be associated with a range of respiratory effects including reduced lung function, increased...
Abstracts

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.

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 sensitization, (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 allergen. 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 grass pollen (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.