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

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