on current and life time measures of depression, anxiety, alcohol misuse, post-traumatic stress disorder, suicide and trauma will be presented. The findings will be discussed in the light of current policies and strategies and recommendations for further practice will be outlined.

O3B.6 THE ECONOMIC BURDEN OF WORK-RELATED ASBESTOS EXPOSURE

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The objective of this study was to estimate the economic burden of lung cancer and mesothelioma due to occupational and paraoccupational asbestos exposure in Canada.

We estimated the lifetime cost of newly diagnosed lung cancer and mesothelioma cases associated with occupational and para-occupational asbestos exposure for calendar year 2011 based on the societal perspective. The key cost components considered were healthcare costs, productivity and output costs, and quality of life costs.

There were 427 cases of newly diagnosed mesothelioma cases and 1904 lung cancer cases attributable to asbestos exposure in our reference year—calendar year 2011—for a total of 2331 cases. Our estimate of the economic burden is \$C831 million in direct and indirect costs for newly identified cases of mesothelioma and lung cancer and \$C1.5 billion in quality of life costs based on a value of \$C100,000 per quality-adjusted life year. This amounts to \$C356,429 and \$C652,369 per case, respectively.

The economic burden of lung cancer and mesothelioma associated with occupational and para-occupational asbestos exposure is substantial. The estimate identified is for 2331 newly diagnosed, occupational and para-occupational exposure cases in 2011, so it is only a portion of the burden of existing cases in that year. Our findings provide important information for policy decision makers for priority setting, in particular the merits of banning the mining of asbestos and use of products containing asbestos in countries where they are still allowed and also the merits of asbestos removal in older buildings with asbestos insulation.

Silica Exposure and Health Effects

O3C.1 SILICA EXPOSURE IN SWEDISH IRON FOUNDRIES AND BIOLOGICAL MARKERS OF INFLAMMATION AND COAGULATION IN BLOOD

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Background Work-related exposure to silica is a health hazard worldwide causing i.e. silicosis. Inflammation is known to be a cause of cardiovascular diseases and some studies have presented elevated cardiovascular disease mortality in relation to silica exposure. The aim of this study was to investigate the relationship between inhalation of exposure to silica in Swedish iron foundries and markers of inflammation and coagulation in blood.

Methods Personal sampling of respirable dust and silica was performed for 85 subjects in three Swedish iron foundries. Stationary measurements were used to study concentrations of respirable dust and silica, inhalable and total dust, PM10 and PM2.5, the particle surface area and the particle number concentrations. The markers of inflammation analyzed were, interleukins (IL-1 β , IL-6, IL-8, IL-10 and IL-12), C-reactive protein, serum amyloid A (SAA), and markers of coagulation fibrinogen, factor VIII (FVIII), von Willebrand factor, and ddimer were measured in plasma or serum. The sampling was performed on the second or third day of a working week following a work free weekend, and additional sampling on the fourth or fifth day.

The personal and stationary measurement data were categorized into three classes to introduce high contrast with a minimum of 10 workers in each group. A mixed model analysis adjusted for sex, age, smoking, infections, blood group, sampling day and BMI was used.

Results For personal sampling the average 8 hour timeweighted average air concentration of respirable dust were 0.85 mg/m3 and respirable silica 0.052 mg/m3. For the high exposure group, statistically significant increased levels of SAA, fibrinogen and FVIII were determined for some exposure measures.

Conclusions This study may indicate an increased risk of cardiovascular disease when observing relations between particle exposure and inflammatory markers.

O3C.2 A 30-YEAR IMPACT ANALYSIS OF BEST PRACTICES FOR SILICA DUST EXPOSURE REDUCTION IN CONSTRUCTION: COSTS, BENEFITS AND HRQL

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Crystalline silica dust exposure is common in the construction sector. In fact, it is the industry with the largest number of people exposed in Canada. Inhalation of silica dust is known to cause lung cancer, silicosis, chronic obstructive pulmonary diseases, rheumatoid arthritis, and tuberculosis. The hierarchy of controls provide a range of prevention options for reducing or eliminating exposure. In this study we undertake an impact analysis of the implementation of two exposure reduction approaches—use of personal protective equipment by all exposed individuals and use of engineering controls wherever and whenever feasible.

We estimated the intervention costs over a 30 year time period (2020–2050) of each exposure reduction approach implemented across the entire sector in Ontario, Canada, and considered equipment, maintenance, training, and productivity costs of each approach over the time period. The economic impacts from lung cancer cases averted due to exposure reduction included three broad categories of impacts—direct, indirect and intangible. We took a societallevel perspective in the analysis. All costs and benefits were discounted to the beginning of the exposure reduction period. Various sensitivity analyses were undertaken with key parameters.

Findings suggest the net benefit is positive from 2055 onward for both reduction approaches, i.e., there is a positive return on investment at the societal level. The largest component of benefits is from health-related quality of life gains, with productivity/output gains accounting for the second largest component. Healthcare savings is the smallest component, primarily because cancer cases incur relatively modest health costs due to their short life-expectancy following diagnosis.

Promoting the use of personal protective equipment and engineering controls across the construction sector can substantially reduce exposures to silica dust and give rise to net benefits at the societal level in terms of improvements in health-related quality of life, increased productivity/output, and reduce healthcare costs.

03C.3 LONGITUDINAL ASSESSMENT OF SMOKING CESSATION AND MORTALITY FROM ALL-CAUSE AND ALL-CANCER AMONG SILICOTICS IN HONG KONG, 1981–2014

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Background Occupational epidemiological studies among silicotics showed that long-term smoking cessation had lowered lung cancer risk by over 50%, but the beneficial effect for reducing risks of other diseases remains unknown. We aimed to evaluate the impact of smoking cessation on the mortality from all-cause and all-cancer using a large historical cohort of 3185 Chinese silicotics since 1981 and followed-up till 2014.

Methods Each silicotic's baseline information was collected including socio-demographics, occupational history and medical history. Smoking habits were assessed at the baseline and reassessed during the follow-up. Multiple Cox proportional hazards model was performed to evaluate the impact of smoking cessation on all-cause and all-cancer mortality using adjusted hazard ratio (AHR) and 95% confidence interval (95% CI).

Results By the end of 2014, a total of 1942 deaths occurred and 360 silicotics died from cancer. Compared with never smokers, silicotics who were new quitters had 30% and 65% higher risk of all-cause of death [AHR=1.30, 95% CI: 1.06–1.58] and all-cancer (AHR=1.65, 95% CI: 1.04–2.62), while persistent quitters had a 52% and 49% excess risk of all-cause of death (AHR=1.52, 95% CI: 1.25–1.84) and allcancer (AHR=1.49, 95% CI: 0.94–2.36), respectively. AHR for all-cause mortality among never quitters was 1.40 (95% CI: 1.14–1.73) while the HR for all-cancer was 2.08 (95% CI: 1.30–3.32). Both all-cause mortality and all-cause mortality decreased sharply after 5 years of smoking cessation and their risks almost equaled to those of the never smokers if the quitters could have kept abstained for more than 20 years. **Conclusions** Smoking cessation sharply decreased all-cause and all-cancer mortality among workers with silicosis, and the beneficial effect was prominent for the long-term quitters. **Acknowledgement** Pneumoconiosis Compensation Fund Board

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03C.4 INCREASES THE RISK OF SARCOIDOSIS BY SILICA EXPOSURE? A CASE-CONTROL STUDY

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Introduction Sarcoidosis is an inflammatory disease with unknown etiology that involves the formation of granulomas, mainly in the lungs and/or intrathoracic lymph nodes. Hypothesis about the etiology of sarcoidosis are combination of genetic and environmental factors. Previous studies have linked exposure to silica dust with increased risk of sarcoidosis.

Object A case-control study to investigate the silica exposure among Swedish sarcoidosis cases.

Methods The data was collected from the National non-primary outpatient care register kept by the Swedish National Board of Health and Welfare. All cases between the age of 20 and 65 with the diagnosis Sarkoidos-D86 according to the International Classification of Diseases, 10th Edition (ICD-10) was included in the study (11 772 cases). The information was matched towards the register for cause of death and the register for emigration.

For each case two controls were included matched for age, sex, was resident in the same county, should not be first degree relatives to cases and not have been diagnosed with the investigated disease using the Swedish Central Bureau of Statistics (SCB) multigeneration register. Cases and controls was matched against SCBs occupational registry for work profession. The levels of silica dust exposure were estimated using NOCCA-JEM (Nordic Occupational Cancer study jobexposure matrix) a modified version of the Finnish Information System on Occupational Exposure job-exposure matrix (FINJEM) which is a well-established method of estimating exposure.

Result Cases of sarcoidosis have an increased exposure to silica before diagnosis (1.19; 95% CI 1.1 to 1.30).

Conclusion The increased exposure to silica among sarcoidosis cases suggest that silica can be an environmental factor that contribute to development of sarcoidosis.

03C.5 REDUCED SERUM CLARA CELL PROTEIN (CC16) AS AN EARLY PULMONARY INJURY MARKER FOR FINE PARTICULATE MATTER EXPOSURE IN OCCUPATIONAL POPULATION

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Background Particulate matter is the key component of air pollutants, mainly produced by emissions of coal-fired plants and road traffic. Exposure to fine particulate matter (PM_{2.5}) pollution is associated with increased morbidity and mortality