

CAREX methodology. Exposure for the indoor working population was estimated using province-specific radon measurements from the Canadian federal building survey (n=12 870 samples). The proportion of workers exposed to specific ranges of radon (50–100, 100–150, 150–200, 200–400, 400–800, >800 Bq/m<sup>3</sup>) were calculated and we assigned the midpoint of the range as the average radon concentration for each exposure group. For the >800 Bq/m<sup>3</sup> category, the province-specific mean of measurements >800 Bq/m<sup>3</sup> was assigned. The above exposure assessment was applied to a population model of the historical Canadian labour force and exposures between 1961 and 2001 (the risk exposure period) were considered as contributing to cancer cases in 2011. The BEIR VI exposure-age-concentration model was used to assign relative risks by exposure category. The population attributable fraction was calculated using Levin's equation.

**Results** There were an estimated 4.4 million indoor workers and 26 000 highly exposed workers exposed to radon during the risk exposure period. Nearly 80% of these workers were exposed below 50 Bq/m<sup>3</sup> (half the WHO reference level). Combining the indoor and highly exposed workers, we calculated that 0.80% of lung cancers are attributable to occupational radon exposure; this equates to 188 lung cancer cases per year.

**Conclusions** Ours was the first study to use a data-driven approach to estimate radon exposure and lung cancer burden for indoor workers. Some of the attributable cases can be prevented by reducing workers' exposure at workplace.

#### 08A.2 BREAST CANCER AND PERSISTENT NIGHT SHIFT WORK STARTING AT AN EARLY AGE THAT CAUSES CIRCADIAN DISRUPTION

<sup>1</sup>Pamela Schwingl, <sup>2</sup>Suril Mehta, <sup>2</sup>Ruth Lunn\*. <sup>1</sup>Integrated Laboratory Systems, Inc., Morrisville, USA; <sup>2</sup>National Toxicology Program, National Institute of Environmental Health Sciences, Report on Carcinogens, Research Triangle Park, USA

10.1136/OEM-2019-EPI.188

Over 10 million U.S. adults and ~15%–20% worldwide work night shifts. Shift work, a complex exposure scenario, can cause circadian disruption (CD) and possible adverse health effects such as breast cancer. Although there have been a plethora of meta-analysis on shift work and breast cancer, these are not very informative because of inconsistent definition of shift work across studies.

The U.S. National Toxicology Program (NTP) conducted a systematic review (SR) to determine whether night shift work should be listed in the Report on Carcinogens (RoC). The SR included a review of cancer epidemiology studies and mechanistic studies of CD and cancer. NTP developed a protocol, based on scientific input gathered during a public workshop, which identified key issues for conducting the SR: definition of surrogates of night work related to CD, inclusion of multiple metrics of night work, information on early age at exposure when breast tissue is most susceptible, cancer subtypes, effect modifiers and confounders, and cohort truncation. Up to three reviewers evaluated the potential for bias and study sensitivity of each of the 26 cohort and case-control studies considered in the assessment, with five excluded due to exposure assessment concerns. To reach an overall conclusion, findings were integrated across studies,

considering factors listed above and confidence in the evidence from each study.

11 of 13 most informative studies and 6 of 8 less informative night shift work studies found increased risks of breast cancer related to night shift work. Excess risks were found mainly among women working frequent nights for long durations starting at an early age (e.g., persistent night shift work). Mechanistic data provided evidence that night shift work causes CD, which plays a major role in its carcinogenicity.

In conclusion, NTP recommends that persistent night shift work that causes CD be listed in the RoC.

#### 08A.3 MINING EXPOSURES AND LUNG CANCER IN CONTEMPORARY WESTERN AUSTRALIAN MINERS

<sup>1</sup>Nita Sodhi-Berry\*, <sup>1,2</sup>Nicholas de Klerk, <sup>1</sup>Peter Franklin, <sup>3</sup>Alison Reid, <sup>3</sup>Lin Fritsch, <sup>4</sup>Bill Musk, <sup>5</sup>Roel Vermeulen, <sup>5</sup>Susan Peters. <sup>1</sup>Occupational Respiratory Epidemiology, School of Population and Global Health, The University of Western Australia, Perth, Australia; <sup>2</sup>Telethon Kids Institute, Nedlands, Australia; <sup>3</sup>School of Public Health, Curtin University, Bentley, Australia; <sup>4</sup>Department of Respiratory Medicine, Sir Charles Gairdner Hospital, Nedlands, Australia; <sup>5</sup>Environmental Epidemiology Division, Institute for Risk Assessment Sciences, Utrecht University, Utrecht, The Netherlands

10.1136/OEM-2019-EPI.189

**Objectives** Mining is associated with exposures to various lung carcinogens such as diesel engine exhaust (DEE) and respirable crystalline silica (RCS). We aimed to determine if lung cancer incidence was higher in Western Australian (WA) miners than the general population and if risk varied within the cohort according to exposures and work or job types.

**Methods** Exposure data for 1 72 398 miners living and working in WA between 1996 and 2013 was combined with administrative WA cancer and death data until June-2017. Causal Incidence Ratios (CIRs) were calculated for general population comparisons. Hazard Ratios (HRs) were derived from multivariable Cox regression models including sex, only-underground work, ore-type (gold, iron-ore, other metal, non-metal, unknown or multi-ore mines) and quantitative estimates of DEE (measured as elemental carbon) and RCS, after adjusting for ever-smoker status and entry-age. Additional analyses were done after lagging exposures by 15 years.

**Results** Mean DEE and RCS cumulative exposures were estimated as 0.15 mg/m<sup>3</sup>-years (std:0.37) and 0.09 mg/m<sup>3</sup>-years (std:0.18), respectively. Miners had lower lung cancer incidence than the general population (observed=382; expected=538.11; CIR:0.71, 95% CI:0.64–0.78). Within the mining cohort, higher lung cancer risks were observed for: females vs. males (HR:1.44, 95% CI:0.97–2.03); ever-smokers vs. never-smokers (HR:10.1, 95% CI:6.37–16.1); only-underground vs. only-surface miners (HR:1.72, 95% CI:1.02–2.90); only gold vs. multi-ore miners (HR:1.44, 95% CI:1.01–2.05); and only iron ore vs. multi-ore miners (HR:1.47, 95% CI:1.07–2.04). Neither DEE (HR:1.01, 95% CI:0.89–1.14) nor RCS (HR:0.89, 95% CI:0.61–1.3) was significantly associated with incidence. There was no significant difference in estimates after lagging exposures.

**Conclusion** Miners had lower risk of lung cancer than the general population. Workers mining exclusively in underground, iron ore or gold mines had higher lung cancer risks than their peers, as did ever-smokers and females. Low levels of DEE and RCS exposures in WA mines may explain the lack of association between these exposures and lung cancer.