ORIGINAL ARTICLE

Burden of lung cancer attributable to occupational diesel engine exhaust exposure in Canada

Joanne Kim,[1,2] Cheryl E Peters,[3,4] Victoria H Arrandale,[1,5] France Labrèche,[6,7] Calvin B Ge,[3,8] Christopher B McLeod,[9] Chaojie Song,[1] Jérôme Lavoué,[7] Hugh W Davies,[3,9] Anne-Marie Nicol,[3,10] Manisha Pahwa,[1] Paul A Demers[1,3,5,9]

[1]Occupational Cancer Research Centre, Cancer Care Ontario, Toronto, Ontario, Canada
[2]Department of Epidemiology, Biostatistics and Occupational Health, McGill University, Montreal, Quebec, Canada
[3]CAREX Canada, Simon Fraser University, Burnaby, British Columbia, Canada
[4]Department of Cancer Epidemiology and Prevention Research, Alberta Health Services, Calgary, Alberta, Canada
[5]Dalla Lana School of Public Health, University of Toronto, Toronto, Ontario, Canada
[6]Chemical and Biological Hazards Prevention, Institut de recherche Robert-Sauvé en santé et en sécurité du travail du Québec (IRSST), Montreal, Quebec, Canada
[7]Département de santé environnementale et santé au travail, Université de Montréal, Montreal, Quebec, Canada
[8]Institute for Risk Assessment Sciences, Universiteit Utrecht, Utrecht, The Netherlands
[9]School of Population and Public Health, University of British Columbia, Vancouver, British Columbia, Canada
[10]Faculty of Health Sciences, Simon Fraser University, Burnaby, British Columbia, Canada

Correspondence to
Dr Paul A Demers;
Paul.Demers@cancercare.on.ca

Received 8 December 2017
Revised 28 March 2018
Accepted 9 April 2018
Published Online First
28 April 2018

Check for updates

To cite: Kim J, Peters CE, Arrandale VH, *et al*. *Occup Environ Med* 2018;**75**:617–622.

ABSTRACT

Objective To estimate the population attributable fraction (PAF) and number of incident and fatal lung cancers in Canada from occupational exposure to diesel engine exhaust (DEE).

Methods DEE exposure prevalence and level estimates were used with Canadian Census and Labour Force Survey data to model the exposed population across the risk exposure period (REP, 1961–2001). Relative risks of lung cancer were calculated based on a meta-regression selected from the literature. PAFs were calculated using Levin's equation and applied to the 2011 lung cancer statistics obtained from the Canadian Cancer Registry.

Results We estimated that 2.4% (95% CI 1.6% to 6.6%) of lung cancers in Canada are attributable to occupational DEE exposure, corresponding to approximately 560 (95% CI 380 to 1570) incident and 460 (95% CI 310 to 1270) fatal lung cancers in 2011. Overall, 1.6 million individuals alive in 2011 were occupationally exposed to DEE during the REP, 97% of whom were male. Occupations with the highest burden were underground miners, truck drivers and mechanics. Half of the attributable lung cancers occurred among workers with low exposure.

Conclusions This is the first study to quantify the burden of lung cancer attributable to occupational DEE exposure in Canada. Our results underscore a large potential for prevention, and a large public health impact from occupational exposure to low levels of DEE.

Key messages

What is already known about this subject?

- Diesel engine exhaust (DEE) is a known human lung carcinogen, and is the most prevalent occupational lung carcinogen in Canada.

What are the new findings?

- We estimated that occupational exposure to diesel exhaust causes 2.4% of lung cancers in Canada, resulting in 560 incident cases and 460 deaths in 2011.
- The majority of the burden was attributable to low concentrations of DEE.

How might this impact on policy or clinical practice in the foreseeable future?

- Our findings may be used to support policies to reduce exposure to DEE in workplaces, such as the implementation of occupational exposure limits, and to promote recognition of work-related cancers by clinicians.

INTRODUCTION

After decades of mounting evidence, diesel engine exhaust (DEE), a complex mixture of gases and particulates produced from the combustion of diesel fuel, was classified in 2012 by the International Agency for Research on Cancer (IARC) as a group 1 definite human carcinogen.[1] This evidence came mainly from workers in the railroad, trucking and mining industries, where the use of diesel engines is widespread. Millions of workers around the world are exposed to diesel exhaust,[2] and in Canada, it is the most prevalent occupational lung carcinogen.[3] Lung cancer is the most commonly diagnosed cancer as well as the leading cause of death from cancer in Canada,[4 5] therefore the public health impact of occupational DEE exposure may be considerable.

The public health impact of specific exposures on disease burden can be calculated using the population attributable fraction (PAF), which is the proportion of disease cases in a population which are attributed to that exposure. The burden of lung cancer caused by occupational DEE exposure has been estimated in other countries.[6–9] For example, Brown *et al*[10] estimated that 1.8% of lung cancers in the UK were caused by DEE exposure in the workplace, making DEE the third highest contributor of occupational lung cancer burden after asbestos and silica.

Since these earlier burden estimates were produced, new epidemiologic data on the quantitative dose–response relationship between DEE exposure and lung cancer mortality have been published.[11–13] Building on the burden estimation methods developed in the UK Burden of Occupational Cancer Study,[14 15] we have incorporated quantitative exposure estimates and the latest dose–response information to estimate the current burden of lung cancer in Canada due to occupational exposure to DEE. This work is part of a larger effort to estimate the burden of occupational cancer in Canada from known and suspected carcinogens.

METHODS

Calculation of the PAF for DEE-attributable occupational lung cancer using Levin's equation[16] required an estimate of the relative risk (RR) of developing cancer due to DEE exposure, as well as an estimate of the proportion of the population exposed (PrE) to DEE. Following the methodological framework developed by the UK Burden of Occupational Cancer study,[14] we assumed a latency of 10–50 years before diagnosis for lung cancer. This established a risk exposure period (REP) of 1961–2001 during which exposures could contribute to cancers diagnosed in 2011, the most recent year for concurrent cancer and census statistics. Briefly, the methodology may be presented in four general steps:

1. Select the most appropriate risk estimates from a review of the recent epidemiologic literature.
2. Assess the prevalence and level of exposure.
3. Calculate the PrE by estimating the number of workers ever exposed during the REP (N_{eREP}) and dividing it by the population alive in 2011 that were of working age at any point during the REP (N_{pREP}).
4. Calculate the PAF using Levin's equation.

Selection of risk estimates

Epidemiologic studies investigating the risk of lung cancer from occupational DEE exposure were identified from the latest IARC evaluation of DEE[17] and from studies indexed in PubMed between January 2012 and January 2016. To select the most relevant risk estimates, we sought studies that: (A) applied to the Canadian exposure context (ie, from countries with a similar degree of industrial development); (B) were of high study quality (ie, reporting quantitative dose–response relationships, ideally through pooled or meta-analysis); and (C) controlled for relevant potential confounders such as smoking and occupational coexposures, where possible.[14]

As the source of our RR estimates, we selected a meta-regression by Vermeulen *et al*[7] which combined the results from three studies that had reported quantitative exposure-response estimates between cumulative exposure to DEE measured as elemental carbon (EC) and the risk of lung cancer mortality: a nested case–control study within a cohort of 11 000 US trucking industry workers,[18] a cohort study of 31 000 US trucking industry workers[11] and the nested case–control study of the Diesel Exhaust in Miners Study (DEMS), within a cohort of 12 000 US non-metal miners.[13] The risk of lung cancer mortality was modelled as a function of cumulative DEE exposure (μg/m^3-years EC): $\ln(RR)=0.000982(DEE)+0.08813$, with a 95% CI of the slope estimate: (0.00055 to 0.00141).[7] Due to the log-linear shape, a maximum RR was set to prevent calculated RRs from exceeding the range of observed RRs in epidemiological studies. Exposure above the median of the highest quartile (EC ≥1005 μg/m^3-years) in the DEMS nested case–control study was associated with an OR of 3.20,[13] which corresponds to a cumulative exposure of 1095 μg/m^3-years EC in the meta-regression model.

Exposure assessment

A DEE job-exposure matrix (JEM) was constructed according to the method developed by CAREX Canada,[3] with prevalence and level of exposure assigned to each job group defined by detailed occupation (four-digit National Occupational Classification for Statistics 2006) and industry (four-digit North American Industrial Classification System 2002). Prevalence estimates were assigned by expert assessment and agreed on by two independent industrial hygienists (CEP, CBG). A category of low, medium or high level of exposure was also assigned based on expert assessment, with consideration of the findings from Pronk *et al*[19] regarding occupational DEE exposure levels reported in the literature, as well as an updated search for diesel exposure measurement data in the peer-reviewed and Canadian grey literature. Generally, jobs in outdoor work sites or in enclosed areas separated from the diesel engine (eg, truck drivers) were categorised as low-exposed jobs involving work with or near smaller diesel-powered equipment in semienclosed areas (eg, mechanics) were categorised as moderately exposed, and since underground mining is a distinct exposure scenario with much greater levels of EC compared with other occupational groups,[19] only underground miners were categorised as highly exposed.

For each qualitative exposure level category (low, moderate and high), we assigned a quantitative exposure concentration to represent the average concentration for the group over the REP. To do this, we first assumed ranges of 0–10 μg/m^3 EC and 10–20 μg/m^3 EC for the low and moderate categories, respectively, and estimated the average concentration from the midpoint of the range. Therefore, in our model, low exposure corresponds to an average concentration of 5 μg/m^3 EC, and moderate exposure corresponds to 15 μg/m^3 EC, respectively. The threshold values of 10 and 20 μg/m^3 EC for the ranges were selected based on the threshold limit value (TLV) and action limit (half of the TLV) which were proposed and subsequently withdrawn by American Conference of Governmental Industrial Hygienists,[20] and supported by exposures observed during the REP in the occupational diesel measurement literature. The high exposure category consisted solely of underground miners, and we directly estimated their average exposure to be 200 μg/m^3 EC, a level supported by measurements collected in North American mines. The oldest DEE measurement records from Canadian mines reported mean concentrations equivalent to 166–429 μg/m^3 EC by job type, during 1977–1981, around the midpoint of our REP.[21] The mean concentration in a survey of 183 US mines in 2002–2003 was equivalent to 170 μg/m^3 EC.[22]

To estimate a worker's cumulative DEE exposure, the average exposure concentration was multiplied by the average job duration for that occupation, sex and age groups from the lifetime work histories of control subjects in the Canadian National Enhanced Cancer Surveillance System.[23] These cumulative exposure estimates were then applied to the meta-regression dose–response model by Vermeulen *et al* in order to calculate the corresponding RR of lung cancer.

PrE during the REP

The PrE was calculated by dividing the number of workers ever exposed during the REP (N_{eREP}) by the population alive in 2011 that were of working age at any point during the REP (N_{pREP}). To quantify the N_{eREP}, our strategy was to count all exposed workers in the first REP year (1961) and only new hires in each subsequent REP year (1962–2000). We first constructed a model of the historical Canadian labour force using census data from 1961, 1971, 1981, 1991 and 2001, interpolating between years, to which we applied the CAREX JEM exposure prevalence estimates. This resulted in estimates of the number of exposed workers in each year of the REP, by industry, occupation, province, sex and exposure level. We additionally modelled the age distribution of workers (in 5-year groups, by sex, province and industry) and the proportion of new hires (by sex, industry and age group) using data from the Labour Force Survey.[24] Only new hires who had worked for at least a year, and were between the ages of 15 and 44 inclusively, were included in the calculation of the N_{eREP}. Finally, the probability of survival to the target year (2011) was calculated using Canadian life tables.[25] Therefore, the N_{eREP} included all exposed workers in the initial REP year (1961) and the exposed new hires from each subsequent

REP year (1962–2000) who were alive in 2011. Allowing a minimum latency of 10 years, the population aged 25 and over in the 2011 Canadian census was used to estimate the N_{pREP}.

Population attributable fraction

From the estimates of RR and PrE, we calculated PAFs using Levin's equation[16]:

$$PAF = \frac{PrE \times (RR-1)}{1+PrE \times (RR-1)}, \text{ where } PrE = \frac{NeREP}{NpREP}$$

The 95% CI of the PAF was constructed using Monte Carlo simulation of the PrE and the RR. For the PrE, we assumed a lognormal distribution with a geometric SD of 2.7. For the RR, we assumed a lognormal distribution with the mean and SD based on the point estimate and variance from the metaregression. Ten thousand samples were drawn from the RR and PrE distributions, and the 2.5th and 97.5th percentiles of the resulting PAFs were used to define the 95% CI. The number of attributable cancers was calculated by applying the PAFs (and their 95% CIs) to the total number of incident lung cancer cases and lung cancer deaths in 2011 that occurred among Canadians 25 years of age or older, obtained from the Canadian Cancer Registry.

Sensitivity analyses

The calculated 95% CI captures random error from the two parameters of Levin's equation: the RR and the PrE. In addition, we conducted sensitivity analyses to determine the independent and combined impact of the following assumptions: the average EC concentration for each exposure level group (±20%, ie, 4–6, 12–18 and 160–240 µg/m³ EC for low, medium and high exposures, respectively); the exposure prevalence of each job group in the JEM (±20%, setting a maximum value of 100% exposure prevalence); and the definition of new hires in the population model to include all working ages versus 15–44 year-olds (primary model), versus restricting further to 15–24 year-olds.

RESULTS

Risk estimates

The risks of lung cancer estimated for our modelled population, by exposure-level group, are shown in table 1.

Exposure assessment

The CAREX Canada DEE JEM captured 3894 unique industry and occupation combinations that were exposed to diesel exhaust in the workplace, accounting for 897 000 Canadians in 2006, representing an exposure prevalence of 5.3%. Of these exposed workers, the majority (87%) were exposed to low levels of DEE (mean: 5 µg/m³ EC); this group included motor vehicle drivers, railroad workers, firefighters and construction labourers. Approximately 11% were exposed to moderate levels (mean: 15 µg/m³ EC), including automotive and heavy duty equipment mechanics, dockworkers and supervisors in mining. The high exposure group (mean: 200 µg/m³ EC) constituted 2% of the exposed population and included only underground miners.

Table 1 Mean relative risks (RR) by exposure group using the primary estimate and the 95% confidence limits (CL) risk estimates from the *Vermeulen et al*'s[7] meta-regression

Exposure group	Primary risk estimate		Lower 95% CL risk estimate		Upper 95% CL risk estimate	
	Mean RR	Range	Mean RR	Range	Mean RR	Range
Low	1.14	1.10–1.20	1.12	1.10–1.15	1.16	1.10–1.26
Medium	1.24	1.11–1.47	1.17	1.10–1.29	1.31	1.12–1.67
High	3.13	1.47–3.20	2.78	1.29–3.20	3.18	1.68–3.20

Table 2 Average (µg/m³ EC) and cumulative (µg/m³ EC-years) DEE exposure and NeREP, by exposure-level group

Exposure level	Range of exposure (µg/m³ EC)	Average exposure (µg/m³ EC)	Cumulative exposure (µg/m³-years)		N_{eREP} (% of N_{eREP})
			Mean	Range	
Low	>0 to <10	5	41.3	5.6–99.8	1 356 910 (84)
Medium	10–20	15	131.3	17.5–299.4	199 445 (12)
High	>20	200	1575.5	225.6–2957.2*	55 856 (4)

*Due to the maximum RR of 3.20, this effectively sets a maximum cumulative exposure of 1095 µg/m³-years (see the Methods section: Selection of risk estimates).
DEE, diesel engine exhaust; EC, elemental carbon; N_{eREP}, number of workers ever exposed during the risk exposure period (REP).

PrE during the REP

Approximately 1.61 million Canadians (6.9%) were exposed to DEE at work for some time during the REP (1961–2001) (table 2). The majority of exposed workers were male (1.48 million, 92%). Overall, the top three industries in terms of numbers of exposed workers were transportation and warehousing, trade, and manufacturing. The occupational groups with the largest numbers of exposed workers were motor transport operating occupations, mechanics of diesel machinery and material handlers. Of all the workers exposed during the REP, 84% were exposed to low levels of DEE, 12% to intermediate and 4% to high levels.

Population attributable fraction

We estimated that the percentage of lung cancers in Canada attributable to occupational DEE exposure was 2.4% (95% CI 1.6% to 6.6%) overall, 4.3% (95% CI 2.8% to 12.2%) for men and 0.21% (95% CI 0.13% to 0.75%) for women. This translates into a total of 562 (95% CI 380 to 1567) incident lung cancers and 456 (95% CI 309 to 1271) lung cancer deaths in 2011. Half of the estimated burden occurred among those exposed at low levels, 14% among moderately exposed workers and 35% among highly exposed workers. Most lung cancers occurred among workers in the broad industries of mining, transportation and diesel engine maintenance (table 3).

Sensitivity analyses

The average RRs by exposure level group, resulting from the primary exposure response model and the 95% CI of the slope, are shown in table 4. Changes to the risk estimates, exposure assessment and population model independently led to PAFs ranging from 1.87% to 2.73%. When all lower or higher estimates were combined in the calculation, this led to PAFs of 1.21% and 3.66% (table 4).

DISCUSSION

Our study presents the first comprehensive estimate of DEE-attributable lung cancer burden in Canada. We estimated that the percentage of lung cancers in Canada attributable to occupational DEE exposure was 2.4% (95% CI 1.6% to 6.6%) overall, 4.3% (95% CI 2.8% to 12.2%) for men and 0.21% (95% CI 0.13% to 0.75%) for women. Therefore, approximately 560 Canadians were diagnosed with, and 460 Canadians died from, lung cancer caused by occupational exposure to DEE in 2011. Of these, almost all occurred among men (96%), which reflects the historical gender composition of the workforce in the industries

Table 3 Estimated DEE-attributable lung cancers by sex, exposure level, industry and occupation

	Attributable number of lung cancers		Proportion of estimated attributable cancers (%)
	Incident cancers, 2011	Cancer deaths, 2011	
Total	562	456	100
Sex			
Male	540	439	96
Female	23	17	4
Exposure level			
Low	286	232	51
Medium	79	64	14
High	198	160	35
Industry			
Mining and oil and gas extraction	224	181	40
Transportation and warehousing	128	104	23
Trade	55	45	10
Manufacturing	42	34	7
Construction	28	23	5
Other	85	69	15
Occupation			
Occupations unique to primary industry	240	194	43
Transport and equipment operators	191	155	34
Other trade occupations	58	47	10
Trade helpers, construction and transportation labourers	43	35	8
Contractors and supervisors in trades and transportation	13	11	2
Other	17	14	3

DEE, diesel engine exhaust.

where elevated DEE exposure occurs, such as in mining, transportation and construction.

Comparison of findings

Our estimated overall PAF of 2.4% (95% CI 2.0% to 2.7%) is slightly higher than those reported for other countries, which range from 1.2% to 1.8%.[6 7 10] Direct comparison of DEE lung cancer PAFs is challenging due to differences in exposure context and study methodology. Many PAFs reported in the literature for lung cancer attributable to occupational DEE are based on the application of a single summary risk estimate to a single exposure prevalence estimate. For example, assuming an RR of 1.31 and 1.35 million exposed workers in the early 1980s, Steenland *et al*[6] estimated a PAF of 1.20% in the USA. As an alternative to using Levin's equation, some studies used population-based case–control studies to calculate PAFs. Using this method, no lung cancers were attributed to occupational DEE in Italy based on no association found in the Environment and Genetics in Lung cancer Etiology (EAGLE) study.[26] In one of the few studies that included adjustment for confounders, Matrat *et al*[9] estimated a PAF in France of 7.2% (95% CI 1.8% to 12.3%) for male lung cancers, after adjustment for smoking and exposure to asbestos. This is higher than the male-only PAF estimate of 2.5% for Finland,[8] as well as our male-specific PAF estimate of 4.3% (95% CI 3.7% to 4.9%) for Canada. The most comparable methodology to our own is from the UK Burden of Occupational Cancer study, which used two exposure-level groups and a 10–50 year latency period.[14 15 27] Our estimates fall within the upper range of the estimated PAFs for the UK: 1.8% (0.0%–3.4%) of lung cancers overall, 2.9% (1.4%–4.8%) among men and 0.4% (0.0%–1.4%) among women.

Table 4 Results of sensitivity analyses: NeREP, attributable lung cancers and attributable fraction

	N_eREP	Attributable incident lung cancers	Attributable fraction (%)
Primary estimate			
Exposure-response slope: 0.000982 Prevalence of exposure using the JEM Age of new hires: 15–44 years	1 611 897	562	2.37
Using lower estimates			
Exposure-response slope, lower 95% confidence limit: 0.00055	No change	480	2.02
Population exposed (–20%)	1 289 768	455	1.92
Quantitative exposure estimates (–20%)	No change	527	2.22
Age of new hires: 15–24 years	1 331 650	444	1.87
All lower estimates, combined	1 065 320	287	1.21
Using upper estimates			
Exposure-response slope, upper 95% confidence limit: 0.00141	No change	641	2.70
Population exposed (+20%)	1 744 437	614	2.59
Quantitative exposure estimates (+20%)	No change	600	2.53
Age of new hires: 15–64 years	1 756 144	648	2.73
All upper estimates, combined	1 899 498	869	3.66

JEM, job-exposure matrix; N_{eREP}, number of workers ever exposed during the risk exposure period.

Methodological considerations

The key strengths of our study are the detailed approach to exposure assessment and the incorporation of a continuous dose–response model. This allowed us to match risk estimates quantitatively, rather than qualitatively, to the estimated cumulative exposures of the modelled population. Furthermore, we leveraged rich sources of Canadian data to account for sex, province and industry-specific trends in our model of the historical Canadian labour force.

However, burden estimation is based on a large number of assumptions. Though we calculated CIs based on variance in both the RR and exposure estimation, they do not account for the uncertainty in the assumptions made. To address this, we investigated the impact of varying the assumptions made for three key components in sensitivity analyses, and found that our overall estimate of the PAF for DEE remained around 2%–3%. The two most influential assumptions investigated were the age distribution of new hires and the job-specific exposure prevalence estimates.

The latency period of 10–50 years between exposure and cancer outcome remains a key assumption. In the absence of a systematic review of the epidemiologic data to address this, we have adhered to the model of the UK burden study, allowing for comparability between our two studies.

We assumed generalisability of the dose–response curve from which we derived our risk estimates across all occupational settings where DEE exposure occurs. The composition of DEE exhaust is known to vary in different contexts and this may alter its carcinogenic potential. However, the metaregression combines results from two heterogeneous groups: truck drivers and miners. Thus, both on-road and off-road applications of diesel engines are represented, as well as the full range of occupational DEE exposure levels (very low among truckers, very high among underground miners).

Although estimates from one of the three studies included in the metaregression were adjusted for smoking, we treated the resulting RRs as unadjusted estimates in our calculation of the PAF. This may have introduced some bias in our estimates, and the magnitude of such bias would be related to the magnitude of confounding.[28] Other occupational exposures such as radon and silica have been found not to confound the association between DEE and lung cancer,[9 13] though confounding by exposure to asbestos has been observed in a population-based case–control study in France.[9]

We independently arrived at a similar distribution and level of exposure (see table 2) as Cherrie *et al*,[29] who estimated that 80% of DEE exposure occurs at 3 µg/m^3 and 20% occurs at 13 µg/m^3. Due to the sparsity of DEE measurement data, especially for the earlier decades of the REP, we were unable to quantify and incorporate time trends in our exposure estimates, though we know that occupational exposures were higher in the past.[19 30] However, we considered the concentrations of 5, 15 and 200 µg/m^3 EC to represent exposure levels, on average, over the years of the REP, on average. Therefore, we do not expect that the incorporation of time trends would greatly affect the PAF estimate.

We were able to estimate lifetime exposure duration from the lifetime occupational histories of a national case–control study.[23] This is an improvement over duration estimated from cross-sectional surveys, such as the Labour Force Survey, which captures only in-progress job duration. For the underground miners, however, lifetime employment duration as a miner likely overestimates time spent underground, potentially overestimating cumulative exposure and therefore the RRs in this group. Our decision to set a maximum RR of 3.20 mitigated this potential overestimation.

Significance

Recent epidemiologic data allowed us to account for underground miners as a distinct risk group, which likely had a significant impact on our burden estimates. Over a third of the estimated DEE-related lung cancers were attributed to miners, a highly exposed group constituting 4% of the ever exposed population. The majority of the estimated cancers, however, were caused by low (<10 µg/m^3) levels of DEE. This finding shows the importance of burden studies such as this one. Focusing intervention efforts on reducing exposure only for the moderate or highly exposed workers would eliminate fewer than half of the DEE-attributable lung cancers.

The assessment by the Health Effects Institute's Diesel Epidemiology Panel[31] that the data from the DEMS[12 13] and the Trucker[11] studies are useful for quantitative risk assessment introduces the possibility of an occupational exposure limit for DEE. Based on the meta-regression results by Vermeulen *et al*,[7] preliminary estimates of health-based limit values (at which one excess lung cancer would occur for every 1000 exposed workers) range from 0.85 to 1.67 µg/m^3 EC.[32] Such exposure levels may not be achievable without a combination of several exposure control strategies, including biodiesel fuel substitution and new engine technology, short of eliminating the use of diesel fuel altogether.

CONCLUSIONS

DEE exposure in the workplace causes approximately 2.4% of lung cancers in Canada, resulting in 560 incident cases and 460 deaths in 2011. Given continuity of occupational exposure to DEE, hundreds of Canadians will continue to be diagnosed and die from preventable lung cancers each year. Our findings highlight that excess lung cancers occur across the exposure spectrum. Miners are particularly in need of protection from the cancer risks arising from their exposure to DEE, and further exposure reductions to those with low DEE exposures must be achieved. Achieving an acceptable level of exposure to DEE is likely to require significant policy changes and prompt turnover of the existing fleet of old diesel-powered machinery and vehicles. Such exposure reductions will take decades to affect cancer burden. Burden estimates such as these may be used to support efforts to preserve the health and safety of workers.

Acknowledgements We gratefully acknowledge the invaluable input from Dr Lesley Rushton and Sally Hutchings of Imperial College London on the overall design of our study methodology. We also acknowledge Jill Hardt and Zoey Cochran for their helpful feedback on manuscript drafts. This project used data collected through the National Enhanced Cancer Surveillance System, a collaboration of the Science Integration Division, Centre for Chronic Disease Prevention and Control, Public Health Agency of Canada and the Canadian Cancer Registries Epidemiology Research Group.

Contributors The project was initiated and conceptualised by PAD, in collaboration with CEP, FL, CBM, JL, HWD, AMN and MP. CEP and CBG created the DEE CAREX JEM, with input from VHA and PAD. JK designed and conducted the analysis, assisted by CS, with input from VHA, CBM and PAD. All authors provided input on the study methods. JK drafted the manuscript under the guidance of PAD and FL and all authors participated in its revision.

Funding This study was funded by the Canadian Cancer Society Research Institute Multi-sector Team Grant in Prevention Research (Grant No 701285).

Competing interests None declared.

Patient consent Not required.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Benbrahim-Tallaa L, Baan RA, Grosse Y, *et al*. Carcinogenicity of diesel-engine and gasoline-engine exhausts and some nitroarenes. *Lancet Oncol* 2012;13:663–4.
2 Kauppinen T, Toikkanen J, Pedersen D, *et al*. Occupational exposure to carcinogens in the European Union. *Occup Environ Med* 2000;57:10–18.
3 Peters CE, Ge CB, Hall AL, *et al*. CAREX Canada: an enhanced model for assessing occupational carcinogen exposure. *Occup Environ Med* 2015;72:64–71.
4 Statistics Canada. *Table 103-0554 - New cases and 2011 age-standardized rate for primary cancer (based on the July 2016 CCR tabulation file), by cancer type and sex, Canada, provinces and territories, annual*. Ottawa, ON, Canada: CANSIM (database), 2016.
5 Statistics Canada. *Table 102-0551 - Deaths and mortality rate, by selected grouped causes, age group and sex, Canada, annual*. Ottawa, Canada: CANSIM (database), 2016.
6 Steenland K, Loomis D, Shy C, *et al*. Review of occupational lung carcinogens. *Am J Ind Med* 1996;29:474–90.
7 Vermeulen R, Silverman DT, Garshick E, *et al*. Exposure-response estimates for diesel engine exhaust and lung cancer mortality based on data from three occupational cohorts. *Environ Health Perspect* 2014;122:172–7.
8 Nurminen M, Karjalainen A. Epidemiologic estimate of the proportion of fatalities related to occupational factors in Finland. *Scand J Work Environ Health* 2001;27:161–213.
9 Matrat M, Guida F, Cénée S, *et al*. Occupational Exposure to Diesel Motor Exhaust and Lung Cancer: A Dose-Response Relationship Hidden by Asbestos Exposure Adjustment? The ICARE Study. *J Cancer Epidemiol* 2015;2015:1–10.
10 Brown T, Darnton A, Fortunato L, *et al*. Occupational cancer in Britain. Respiratory cancer sites: larynx, lung and mesothelioma. *Br J Cancer* 2012;107(Suppl 1):S56–70.
11 Garshick E, Laden F, Hart JE, *et al*. Lung cancer and elemental carbon exposure in trucking industry workers. *Environ Health Perspect* 2012;120:1301–6.
12 Attfield MD, Schleiff PL, Lubin JH, *et al*. The Diesel Exhaust in Miners study: a cohort mortality study with emphasis on lung cancer. *J Natl Cancer Inst* 2012;104:869–83.

13 Silverman DT, Samanic CM, Lubin JH, *et al*. The Diesel Exhaust in Miners study: a nested case-control study of lung cancer and diesel exhaust. *J Natl Cancer Inst* 2012;104:855–68.
14 Hutchings SJ, Rushton L. Occupational cancer in Britain. Statistical methodology. *Br J Cancer* 2012;107(Suppl 1):S8–17.
15 Rushton L, Hutchings SJ, Fortunato L, *et al*. Occupational cancer burden in Great Britain. *Br J Cancer* 2012;107(Suppl 1):S3–S7.
16 Levin ML. The occurrence of lung cancer in man. *Acta Unio Int Contra Cancrum* 1953;9:531–41.
17 IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Diesel and gasoline engine exhausts and some nitroarenes. Iarc monographs on the evaluation of carcinogenic risks to humans. *IARC Monogr Eval Carcinog Risks Hum* 2014;105:9-699.
18 Steenland K, Deddens J, Stayner L. Diesel exhaust and lung cancer in the trucking industry: exposure-response analyses and risk assessment. *Am J Ind Med* 1998;34:220–8.
19 Pronk A, Coble J, Stewart PA. Occupational exposure to diesel engine exhaust: a literature review. *J Expo Sci Environ Epidemiol* 2009;19:443–57.
20 American Conference of Governmental Industrial Hygienists. *Documentation of the threshold limit values and biological exposure indices*. Cincinnati, OH: American Conference of Governmental Industrial Hygienists, 2001.
21 Grenier M, d'Aguiar D. *DPM Exposure in Canadian Mines. Mining Diesel Emissions Conference*. Markham, Canada, 1999.
22 Mine Safety and Health Administration. *MSHA's report on data collected during a joint MSHA/industry study of DPM levels in underground metal and nonmetal mines*. Arlington: Mine Safety and Health Administration, 2003.
23 Johnson KC, Mao Y, Argo J, *et al*. The National Enhanced Cancer Surveillance System: a case-control approach to environment-related cancer surveillance in Canada. *Environmetrics* 1998;9:495–504.
24 Statistics Canada. *Labour Force Survey (microdata files)*. Ottawa, Canada: IDLS, 1976–2003.
25 Statistics Canada. Division D, ed. *Life Tables, Canada, Provinces and Territories*, 1960–2002.
26 De Matteis S, Consonni D, Lubin JH, *et al*. Impact of occupational carcinogens on lung cancer risk in a general population. *Int J Epidemiol* 2012;41:711–21.
27 Van Tongeren M, Jimenez AS, Hutchings SJ, *et al*. Occupational cancer in Britain. Exposure assessment methodology. *Br J Cancer* 2012;107(Suppl 1):S18–26.
28 Greenland S. Bias in methods for deriving standardized morbidity ratio and attributable fraction estimates. *Stat Med* 1984;3:131–41.
29 Cherrie JW, Gorman Ng M, Shafrir A, *et al*; *Health, Socio-economic and Environmental Aspects of Possible Amendments to the EU Directive on the Protection of Workers from the Risks Related to Exposure to Carcinogens and Mutagens at Work*. Edinburgh: Institute of Occupational Medicine, 2011.
30 Symanski E, Kupper LL, Hertz-Picciotto I, *et al*. Comprehensive evaluation of long-term trends in occupational exposure: Part 2. Predictive models for declining exposures. *Occup Environ Med* 1998;55:310–6.
31 Health Effects Institute. *Diesel Emissions and Lung Cancer: An Evaluation of Recent Epidemiological Evidence for Quantitative Risk Assessment*. Boston, MA: Health Effects Institute, 2015.
32 Vermeulen R, Portengen L. Is diesel equipment in the workplace safe or not? *Occup Environ Med* 2016;73:846–8.