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Research Article

Cancer Prevention Research

Interventions to Reduce Future Cancer Incidence from Diesel Engine Exhaust: What Might Work?

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Abstract

Exposure to diesel engine exhaust (DEE) contributes appreciably to the burden of occupational cancer. This study aims to estimate the potential impact of a range of interventions on the future burden of cancer from occupational exposure to DEE in Australia. The future excess fraction method, a novel method based on the lifetime risk approach, was used to model changes in the future burden of cancer among the Australian working age population exposed to DEE at work in 2012 under various intervention strategies. The interventions modeled were based on the widely accepted hierarchy of control model. At baseline, 600 (0.4%) future bladder and 4,450 (0.6%) future lung cancer cases over the lifetime of the cohort were estimated to be attributable to occupational exposure to DEE in those exposed in 2012. Up to 2,000 of these cases were estimated to be avoidable through the use of various interventions. Exhaust hoses (engineering controls) were estimated to be particularly effective. This study provides an indication of which intervention strategies may be most useful in reducing the future burden of cancer associated with occupational DEE exposure. These results show the potential effect of changing current exposure, rather than focusing on past exposures, and thus provide relevant information for policy planning.

Introduction

Diesel engine exhaust (DEE) consists of a complex mixture of gaseous components and particulate matter (1), with a varying composition depending on factors such as the engine type, operating conditions, and the use (or not) of an emissions control system (2). Diesel engines are widely used in both on-road and off-road applications, including in power generators, heavy equipment, and vehicles such as buses and trucks (3–5). The widespread use of diesel engines has led to concerns regarding the potential health effects of exposure (6), with epidemiologic studies providing evidence of a causal association between DEE exposure and cancer, particularly lung cancer (4). Accordingly, the International Agency for Research on Cancer (IARC) classified DEE as probably carcinogenic to humans (Group 2A) in 1989 (7), revising the classification to Group 1 (definitely carcinogenic) in 2012, with exposure linked to cancers of the bladder and lung (4).

While exposure to DEE is likely among the majority of the general population, and in some cases the general population may be exposed through the work activities of others, occupational exposures are generally of a much higher magnitude than environmental exposures (8). A high number of workers across varying occupations are exposed to DEE (5, 9). In Australia, we estimated that 14% of the working population, equivalent to around 1.2 million workers, were exposed to substantial (high and medium) levels of DEE at work in 2012 (3). Those most commonly exposed worked in the agricultural, mining, transport, and construction industries, and exposures were more common among men and those living in remote areas. This estimate was higher than estimates of exposure prevalence from other countries which have generally ranged from 2%–5% of workers (10–13). This is likely to reflect the mix of industry sectors in each country. Higher prevalence of exposure has been estimated among workers in occupations such as drivers, miners, firefighters, and mechanics (2, 9).

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Exposure to DEE contributes substantially to the burden of occupational cancer. The GBD project found that occupational exposure to DEE was responsible for 485,690 disability adjusted life years and 17,500 deaths related to lung cancer globally in 2016 (14), while work in the United Kingdom has estimated that 0.2% of all current cancers are due to past occupational exposure to DEE (15, 16). Using a different method, we found that occupational DEE exposure in 2012 (at any level) could be expected to contribute approximately 5,500 future cancer registrations (4,500 lung cancers and 1,000 bladder cancers) over the lifetime of the 2012 Australian working population, or 0.12% of all future cancers in this population (17). This places DEE as the occupational carcinogen contributing the fifth highest number of future cancer registrations in Australia, behind exposure to asbestos, solar radiation, benzene, and environmental tobacco smoke (indoor and outdoor) in the workplace.

It is therefore important to control and ultimately prevent DEE exposure in workers. The basic approach to exposure control is to use the hierarchy of control measures: elimination as the most preferable option, followed by substitution, engineering, and administrative controls, and as the last resort, personal protective equipment (PPE; ref. 18). The aim is to control exposure to a level as low as reasonably practicable, with the ultimate goal of eliminating exposure (5). As DEE is encountered in a wide range of occupations, the appropriate control strategies will vary depending on the circumstances of exposure, practicality, and current work practices, as well as cost and benefit considerations (1, 5, 19). Where elimination of exposure is not reasonably practicable, measures to control DEE exposure may include the use of lower emission engines, exhaust filters, exhaust extraction systems, sufficient ventilation, and enclosed cabins (1, 20).

While reducing or preventing workplace exposure to DEE among the largest number of workers may be the obvious approach to reducing the future burden of cancer, exposure prevalence is not the only consideration in comparing the effectiveness of intervention strategies. The number of cancers avoidable through any one intervention is a combination of the current or baseline burden of cancer, the current levels of exposure and the levels achievable through intervention, and the relative risks at different levels of exposure, in addition to the current exposure prevalence. Thus, an approach which reduces exposure from a high to a low level may be more or less effective than one which removes low level exposure, depending on these factors. The future excess fraction (FEF) method used here takes all of these factors into consideration (21).

This study therefore aimed to model the potential effect of the use of a range of control measures (or intervention strategies) across the hierarchy of control on the future number of cancers attributable to occupational exposure to DEE in Australia, using the FEF method.

Materials and Methods

The FEF method was used to estimate the future burden of occupational cancer among the Australian working age population who were exposed to high levels of DEE at work in 2012, and to model the changes in this future burden under various exposure scenarios (or intervention strategies). We conducted estimates for cancers of the bladder (listed by IARC as having limited evidence of a causal relationship with DEE exposure) and lung (sufficient evidence; ref. 4). A full description of the methods has been provided elsewhere (17, 21).

Data sources

We defined the cohort for this study as Australian residents who were aged 18–65 in 2012 (described as the Australian working age population in 2012, n = 14,588,700). We first calculated a matrix showing the proportionate survival of an individual at each future age (from 2012 to 2094) using a double decrement life table. The two endpoints were death and first diagnosis of the cancer of interest (bladder or lung). The future person-years-at-risk for the cohort to 2094 was then obtained by multiplying this matrix by the 2012 mid-year population statistics (22). The year 2094 was chosen as this was the year the youngest individuals in the cohort (i.e., those aged 18 in 2012) would turn 100. Four matrices in total were created (bladder and lung cancer for males and females separately).

We used the R-based software "Canproj" (23) to estimate the number of bladder and lung cancers occurring in the cohort to 2094 (separately by sex). "Canproj" conducts the most appropriate projection model (age-period–cohort or log linear regression) on the basis of observed cancer registrations. Three inputs were required: the number of observed cancer registrations by site, sex, 5-year age group, and year of diagnosis (24); the observed population by sex and 5-year age group for the same period (22); and the projected population to 2094 by sex and single year of age (25). Canproj only allows projections of 25 years, and so projections after 2036 used initial projected incidence rates as well as the observed (1986–2011) rates to produce a new prediction base.

Relative risk (RR) estimates and 95% confidence intervals (CI) for high exposure to DEE for both bladder and lung cancer were sought from the literature. We sought risk estimates from pooled or meta-analyses of studies where the definition of exposure corresponded as closely as possible to that used in our prevalence estimate. The risk estimate for bladder cancer (RR = 1.24; 95% CI, 1.10–1.41) was taken from a meta-analysis of 35 studies (26, 27). For lung cancer, we used a smoking-adjusted risk estimate (RR = 1.47; 95% CI, 1.29–1.67) obtained from a meta-analysis of 9 studies (28). No excess risk was assumed for low exposure for either lung or bladder cancer

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Table 1. Prevalence of occupational exposure to DEE in Australia in 2012, by industry and exposure level

	Sample[a]		Population[b]	
Industry	**High, *n* (%)[c]**	**Low, *n* (%)[c]**	**High, *n* (%)[c]**	**Low, *n* (%)[c]**
Agriculture, forestry, and fishing	2 (1.0)	170 (88.1)	3,700 (1.8)	183,100 (86.6)
Mining	8 (7.3)	77 (70.6)	13,900 (8.0)	137,400 (78.8)
Manufacturing	4 (1.4)	53 (18.8)	7,400 (0.8)	85,300 (9.8)
Electricity, gas, water, and waste services	0 (0.0)	14 (43.7)	0 (0.0)	20,900 (18.4)
Construction	21 (4.6)	152 (33.0)	34,000 (4.2)	253,000 (31.5)
Wholesale trade	0 (0.0)	37 (69.8)	0 (0.0)	78,600 (20.1)
Retail trade	0 (0.0)	24 (7.8)	0 (0.0)	43,900 (4.6)
Accommodation and food services	0 (0.0)	2 (1.3)	0 (0.0)	6,100 (1.1)
Transport, postal, and warehousing	11 (4.8)	158 (68.4)	28,200 (6.1)	310,600 (67.2)
Information media and telecommunications	0 (0.0)	4 (7.1)	0 (0.0)	6,100 (3.6)
Financial and insurance services	0 (0.0)	5 (33.3)	0 (0.0)	7,400 (2.0)
Rental, hiring, and real estate services	0 (0.0)	16 (76.2)	0 (0.0)	45,800 (30.8)
Professional, scientific, and technical services	0 (0.0)	67 (35.8)	0 (0.0)	112,400 (15.9)
Administrative and support services	0 (0.0)	24 (1.2)	0 (0.0)	39,400 (12.6)
Public administration and safety	6 (5.6)	55 (51.4)	12,100 (1.8)	111,100 (16.4)
Education and training	0 (0.0)	10 (2.2)	0 (0.0)	18,700 (2.4)
Health care and social assistance	0 (0.0)	19 (3.2)	0 (0.0)	38,100 (3.4)
Arts and recreation services	0 (0.0)	4 (16.0)	0 (0.0)	11,500 (8.2)
Other services	50 (29.4)	32 (18.8)	74,700 (20.8)	49,400 (13.7)
Total	102 (1.9)	923 (16.8)	174,000 (1.9)	1,558,800 (17.4)

[a]Obtained from the AWES (30).
[b]Sample proportion extrapolated to Australian working population using Census 2011 data (38). Extrapolations conducted separately by occupational group and gender. Rounded to nearest 100.
[c]Percentage of individuals in each industry who were classified as exposed.

(i.e., RR = 1) in our main analyses. However, we also conducted sensitivity analyses whereby we assumed a risk estimate for low exposure to DEE of 1.03 (95% CI, 0.84–1.26) for bladder cancer (from a meta-analysis of 6 studies; ref. 26), and 1.04 (95% CI, 0.95–1.14) for lung cancer (from a pooled analysis of 11 studies; ref. 29; see Supplementary Tables S1 and S2).

The prevalence of exposure to DEE at work in 2012 was obtained from our Australian Work Exposures Study (AWES; ref. 30), supplemented by the AWES-Western Australia. AWES was a cross-sectional telephone survey of 4,993 Australian workers, supplemented by an additional 505 workers from the state of Western Australia. Full methods have been described previously (3, 30). These data provided an estimate of exposure prevalence by sex and occupational group, as well as information about the qualitative level of exposure (high, medium, or low). Medium and low exposures were combined into a low exposure category for the purpose of these analyses. Exposure estimates were based on reported tasks completed. Table 1 presents the prevalence of exposure by industry and exposure level, extrapolated to the Australian working population.

Intervention strategies modeled

The impact of intervention strategies was modeled by changing the number of people completing particular tasks and therefore modifying the prevalence and/or level of exposure to DEE. All other data inputs remained constant. The intervention strategies modeled were based on the hierarchy of control model (5, 18) and were chosen based on guidance documents for managing DEE (31, 32) as well as the exposure and task information we had available through AWES.

The strategies modeled were as follows:

1. Elimination: completely removing all sources of exposure to DEE among workers in the mining, agriculture, and construction industries (modeled separately). This was modeled by assuming that all workers in the target industry were unexposed to DEE.
2. Substitution: substituting all diesel forklift engines with gas or electric engines.
3. Isolation/administrative controls: removing all diesel powered vehicles from indoor workspaces.
4. Engineering controls: improving compliance of use of exhaust hoses when performing tune ups on diesel powered vehicles (by 25%, 50%, 75%, and 100% in turn). The use of exhaust hoses was assumed to reduce DEE exposure from a high to a low level.
5. PPE: well-fitted respiratory equipment used during road construction tasks. This was assumed to reduce DEE exposure to a low level.

Statistical analyses

Full details of the statistical methodology have been published previously (21). Briefly, the FEF method first estimates the lifetime risk (LR_P) in the cohort irrespective of exposure, using the estimated person-years-at-risk, cancer incidence rates, and number of people in the cohort. The excess lifetime risk is then calculated by taking into account the LR_P, relative risk estimate, and number of people in the cohort. This excess risk is then multiplied by the number of people exposed to estimate the number of

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cancers attributable to exposure (the future excess number or FEN). All calculations were conducted separately by sex and cancer type (bladder/lung).

We used the FEF method to estimate the future burden of occupational cancer under each intervention strategy, with only the number of exposed workers differing between estimates. The resulting FEN and FEF were then compared with the baseline FEN and FEF to estimate the number of "avoided" cancers.

Results

Our cohort of the Australian working age population in 2012 was estimated to number 14,588,700 in total, comprising 7,297,000 males and 7,291,700 females. An estimated 142,800 bladder cancers and 728,600 lung cancers were predicted to occur over their lifetime, regardless of the source of exposure.

At baseline (assuming no intervention), 0.4% ($n = 600$) of future bladder cancer cases and 0.6% ($n = 4,450$) of future lung cancer cases were estimated to be attributable to occupational exposure to DEE. The majority of these were estimated to occur among males (bladder cancer FEN = 600; lung cancer FEN = 4,350), with less than 100 cases of each cancer type estimated among females.

Tables 2 and 3 show the results of the modeling of the various intervention strategies. For bladder cancer, the most effective strategies were estimated to be isolation and engineering controls (Table 2). Full (100%) compliance with the use of exhaust hoses prevented the most cases.

For lung cancer, isolation and engineering controls were again estimated to be the most effective strategies (Table 3). More than 1,000 future cases of lung cancer were estimated to be avoidable by the removal of diesel powered vehicles from indoor workspaces, while 100% compliance with the use of exhaust hoses was estimated to prevent four times as many lung cancers as partial compliance.

For both cancers, effects were seen at all levels of the hierarchy of control, with engineering controls of particular note.

Discussion

We have used the FEF method to estimate how many future bladder and lung cancer cases could be prevented by using a range of intervention strategies to reduce or remove occupational exposure to DEE. These results are of particular relevance as they show the potential effect of changing current exposure (which is amenable to change), rather than focussing on past exposures (which cannot be modifed). Our results may therefore be of particular use in policy planning (17).

We found that up to 2,000 (or just under half of all) future cases of bladder and lung cancer due to workplace exposure to DEE could be avoided over the lifetime of our cohort by implementing strategies to reduce exposure. Ours is the first study to use the FEF method to model the impact of potential intervention strategies on future cancers over the lifetime of a cohort. A study in the United Kingdom used the attributable fraction approach to model changes in the future burden of cancer resulting from reductions in workplace exposure limits and/or improvements in compliance to these limits, and found that almost all lung cancers due to DEE exposure could be avoided by 2060 (15). This approach models the effect of changing exposure over a number of years (the risk exposure period) on cancers occurring in one target year, whereas the FEF method

Table 2. Estimated avoidable bladder cancers attributable to occupational DEE exposure for a range of intervention strategies among cohort of working age Australians in 2012

	Intervention strategy[a]	FEF (%)	FEN (*n*)[b]	Avoidable registrations (*n*)[b]
	Baseline (*n* exposed = 1,738,300)	0.42	600	—
Elimination	Scenario 1: Removing all DEE exposure among workers in mining industry (baseline *n* exposed = 151,300)	0.39	550	50
	Scenario 2: Removing all DEE exposure among workers in agriculture industry (baseline *n* exposed = 186,800)	0.41	600	<50
	Scenario 3: Removing all DEE exposure among workers in construction industry (baseline *n* exposed = 287,000)	0.34	500	100
Substitution	Scenario 4: Substituting all diesel forklift engines with gas or electric (baseline *n* exposed = 58,100)	0.38	550	50
Isolation	Scenario 5: Removing all diesel powered vehicles from indoor workspaces (baseline *n* exposed = 273,600)	0.32	450	150
Engineering controls	Scenarios to improve the use of exhaust hoses when performing tune ups on diesel powered vehicles (baseline *n* performing tune ups = 85,800; baseline use of exhaust hoses 9%)			
	Scenario 6: Improving use of exhaust hoses to 25% of those performing tune ups	0.37	550	50
	Scenario 7: Improving use of exhaust hoses to 50% of those performing tune ups	0.32	450	150
	Scenario 8: Improving use of exhaust hoses to 75% of those performing tune ups	0.27	400	200
	Scenario 9: Improving use of exhaust hoses to 100% of those performing tune ups	0.22	300	300
PPE	Scenario 10: Using respiratory protective equipment during road construction tasks (baseline *n* exposed = 28,600)	0.35	500	100

[a]Numbers exposed rounded to the nearest 100.
[b]Number of cancer registrations rounded to the nearest 50.

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Table 3. Estimated avoidable lung cancers attributable to occupational DEE exposure for a range of intervention strategies among cohort of working age Australians in 2012

	Intervention strategy[a]	FEF (%)	FEN (*n*)[b]	Avoidable registrations (*n*)[b]
	Baseline (*n* exposed = 1,738,300)	0.61	4,450	—
Elimination	Scenario 1: Removing all DEE exposure among workers in mining industry (baseline *n* exposed = 151,300)	0.56	4,100	350
	Scenario 2: Removing all DEE exposure among workers in agriculture industry (baseline *n* exposed = 186,800)	0.60	4,350	100
	Scenario 3: Removing all DEE exposure among workers in construction industry (baseline *n* exposed = 287,000)	0.49	3,600	850
Substitution	Scenario 4: Substituting all diesel forklift engines with gas or electric (baseline *n* exposed = 58,100)	0.55	4,000	450
Isolation	Scenario 5: Removing all diesel powered vehicles from indoor workspaces (baseline *n* exposed = 273,600)	0.47	3,400	1,050
Engineering controls	Scenarios to improve the use of exhaust hoses when performing tune ups on diesel powered vehicles (baseline *n* performing tune ups = 85,800; baseline use of exhaust hoses 9%)			
	Scenario 6: Improving use of exhaust hoses to 25% of those performing tune ups	0.54	3,950	500
	Scenario 7: Improving use of exhaust hoses to 50% of those performing tune ups	0.47	3,450	1,000
	Scenario 8: Improving use of exhaust hoses to 75% of those performing tune ups	0.40	2,950	1,500
	Scenario 9: Improving use of exhaust hoses to 100% of those performing tune ups	0.33	2,400	2,050
PPE	Scenario 10: Using respiratory protective equipment during road construction tasks (baseline *n* exposed = 28,600)	0.52	3,750	700

[a]Numbers exposed rounded to the nearest 100.
[b]Number of cancer registrations rounded to the nearest 50.

used in this study estimates the effect of changing exposure in a single year on cancers occurring over the lifetime of the cohort. Therefore, due to fundamental differences in the methods used, these results are not directly comparable with ours.

We found that interventions at all levels of the hierarchy of control, from elimination through to PPE, could be effective in reducing the future number of cancers attributable to occupational DEE exposure. Engineering controls, operationalized as the use of exhaust hoses during vehicle maintenance tasks, were estimated to be particularly effective. Previous research in the United Kingdom has found that exhaust hoses reduce personal and background diesel particulate concentrations (1). However, exhaust hoses were not commonly used in the worksites investigated in that study, in line with our finding that only 9% of those performing vehicle tune ups used an exhaust hose. Engineering controls have been shown more generally to be the most often used method of controlling exposure to DEE (1), as well as the most practicable (19).

Our study also found that some intervention strategies were less effective overall. This is likely to be largely a function of the lower numbers exposed at baseline. For example, just over 58,000 workers were exposed to DEE through the use of diesel forklifts, while over 273,000 were exposed via diesel vehicles in indoor workplaces. Therefore, the effect of removing indoor diesel vehicles (isolation) is likely to be greater than the effect of substituting diesel forklifts with gas or electric engines simply as a result of the higher number of exposed workers involved.

The estimated effectiveness of interventions was also influenced by the levels of exposure observed at baseline. As we assumed no excess risk for low-level exposure to DEE, removing or reducing exposure among workers with low exposure prior to any intervention did not have an effect on future cancer numbers. This may explain why elimination was not estimated to be as effective as some of the other intervention strategies in avoiding cancers, as the majority of workers exposed in the construction, agriculture, and mining industries were exposed at a low level and thus eliminating their exposure did not influence the future burden of cancer. Indeed, in our sensitivity analyses where we assumed a small excess risk for low exposure, we observed that elimination was estimated to be relatively more effective than seen in our main analyses.

Overall, interventions appeared to be proportionately and absolutely more effective in avoiding lung rather than bladder cancer cases, due to a combination of the relative risks and the underlying number of cancers (higher for lung cancer). As lung cancer has a higher relative risk for high exposure than bladder cancer, the same prevalence of (high level) exposure will lead to more lung cancers than bladder cancers. In addition, lung cancer is more common meaning that there is more burden to avoid, and therefore the same proportionate decrease in burden will result in a higher avoided number of cases.

Study limitations

Our findings are subject to the assumptions we made for the FEF approach, which we have described in detail previously and outline here (17, 21). Many of these are uniform across the intervention strategies modeled, and so are likely to impact on the absolute numbers only, and not the differences in effectiveness between interventions. In brief, we assumed that the risk estimates used were

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appropriate for the exposures captured in our estimates of exposure prevalence. We used risk estimates from meta-analyses in an attempt to capture a broad range of exposure patterns, but these were based on studies of past exposure and so may not be entirely relevant to current exposures. We also used risk estimates which were adjusted for smoking and other potential confounders wherever possible. In addition, our prevalence estimates only included qualitative levels of exposure, rather than being based on any quantitative measurements, and so it is unclear how these relate to the exposure levels used in the risk estimates.

We also erred on the side of caution by assuming no excess risk for low exposure levels. This may have the effect of underestimating the number of cancers avoidable through particular interventions (especially elimination). To explore this, we conducted a sensitivity analysis whereby we included risk estimates for low exposure to DEE for both bladder and lung cancer (see Supplementary Tables S1 and S2). As expected, assuming an excess risk for low exposure increased the FEF and FEN for both cancer types; however, the relative effectiveness of interventions was, for the most part, unchanged. The exception to this was the effect of eliminating exposure in the construction industry, which was estimated to be more effective when low exposures were included. This is likely due to the high number of construction workers who were exposed at a low level. It should be noted, however, that the risk estimates for low exposure were not significant (i.e., the confidence intervals included the null), and hence our main analyses assumed no excess risk for low exposure.

In addition, the extent of our projections of cancer incidence (over 80 years) and the assumptions made in conducting these are likely to present some uncertainty. While the level of this uncertainty is unclear, we have previously conducted a sensitivity analysis around these projections which produced a similar albeit slightly higher FEF and FEN than the method used here (17).

The interventions that we modeled relied on the task information that we had available, as well as information about the current use of controls, which was generally restricted to some engineering controls. In some cases, we assumed that no controls were currently in place, and the accuracy of this assumption is unknown. We were unable to model other potential interventions for which we did not have the relevant baseline information. Perhaps the clearest example is the use of exhaust filters, which are commonly recognized as effective tools for reducing or removing DEE exposure (33). There is some suggestion in the literature that the uptake and use of exhaust filters may be hampered by concerns about their complexity and expense (34), as well as the durability of diesel engines (35). However, we did not have any information on the baseline use of exhaust filters. We were also limited in our ability to model changes in exposure limits as per the United Kingdom study (15), as the levels of exposure in our data were qualitative rather than quantitative, and there is currently no exposure limit for DEE in Australia (3). Furthermore, it is unknown to what extent these interventions may be relevant in other countries due to the different regulations already in place (for example, regulations for forklifts used indoors already in place in the European Union).

Some of the interventions we modeled may represent "best case scenarios" and not be immediately practicable. For example, there are likely to be significant practical challenges in attempting to eliminate DEE exposure in a particular industry (36). The intervention strategies we modeled are thus intended to be theoretical rather than necessarily practical, and some may not be feasibly achievable, at least in the short term. As suggested by Cherrie, it may be that a change in attitude towards DEE exposure is needed before these strategies are widely accepted, particularly those requiring greater changes (19). The acceptance of intervention strategies may also be hindered by an inadequate understanding of the risks surrounding DEE exposure, although much greater attention has been given by the public and governmental agencies to the health risks associated with exposure to DEE in recent years (37).

The results presented here provide an indication of which intervention strategies may be most useful in reducing the burden of cancer associated with occupational DEE exposure. These interventions may also be useful in avoiding or reducing the occupational DEE-associated burden of other diseases, including respiratory symptoms (8). For example, we estimated that implementation of engineering controls would lead to the biggest reduction in the number of cancer cases, in part, due to the low compliance with controls currently in place. Therefore, attention could be targeted at increasing compliance with currently implemented controls. In any event, the choice of intervention should be appropriate to the circumstances of exposure, with the ultimate aim of reducing exposure as much as reasonably practicable (5, 19). The results of this study have the potential to generate clear policy recommendations around the prevention of occupational DEE exposure, providing information that is readily understood by, and salient to, policy makers (21).

Disclosure of Potential Conflicts of Interest

T.R. Driscoll has provided expert testimony, in 2015, to BHP Billiton regarding information relevant to developing a revised occupational exposure limit for diesel engine exhaust. No potential conflicts of interest were disclosed by other authors.

Authors' Contributions

Conception and design: R.N. Carey, L. Fritschi, D.C. Glass, G. Benke, A. Reid

Development of methodology: R.N. Carey, L. Fritschi, T.R. Driscoll, S. Peters, D.C. Glass, G. Benke

Analysis and interpretation of data (e.g., statistical analysis, biostatistics, computational analysis): R.N. Carey, L. Fritschi, T.R. Driscoll, S. Peters, D.C. Glass, A. Reid

Writing, review, and/or revision of the manuscript: R.N. Carey, L. Fritschi, T.R. Driscoll, S. Peters, D.C. Glass, G. Benke, A. Reid

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