Industrial Injuries Advisory Council Information Note

Lung and Bladder Cancer and Diesel Exhaust Emissions

September 2015

1. The possible carcinogenicity of diesel engine exhaust is one of the topics that the Industrial Injuries Advisory Council (IIAC) has had under review.

2. In diesel engines, air is introduced into the engine and heated by compression to a temperature in excess of 425°C. The fuel is introduced into the combustion chamber by a high-pressure injection system and is mixed with the hot air until the jet of fuel becomes sufficiently hot for auto-ignition to occur. The centre of this burning jet is very rich in fuel, which leads to the formation of elemental carbon, partially burned fuel, polycyclic aromatic hydrocarbons and carbon monoxide. At the outer edges of the burning jet of fuel, excess air leads to high temperatures and the formation of nitrogen oxides (International Agency for Research on Cancer (IARC), 2013). Rudolf Diesel patented the diesel engine in 1898. In the early part of the twentieth century, diesel engines were used mainly in marine applications and then installed in heavy goods vehicles (HGVs) in Europe in the 1920s. In the 1930s, manufacturers in the USA started to install diesel engines in commercial HGVs, buses and tracked vehicles. The first mass-produced diesel passenger car was introduced in Europe in 1936. Diesel engines had largely replaced steam power in railway (railroad) locomotives by the early 1950s, and had replaced petroleum (gasoline) engines in most HGVs by the 1960s. Today, diesel engines power all types of automotive vehicles: passenger cars (up to 50% of car sales in some European countries), commercial vehicles, buses and coaches, industrial, agricultural and construction equipment, mine vehicles, locomotives, shops and many stationary power applications (IARC, 2013). Until the mid-1980s, a wide variety of diesel engine designs and technologies were available. However, with increasingly stringent regulations on emissions, the industry converged on a common diesel engine architecture. Other technological changes have ensued. Fuel technology has also changed, with the reduction of the sulphur content. After-combustion treatment techniques such as selective catalytic reduction or nitrogen oxide adsorber-based systems and particle filtration in exhaust systems have been introduced to further reduce emissions (IARC, 2013).

3. Diesel engine exhaust comprises a combination of particles, chemical molecules and gases, some of which (such as polycyclic aromatic hydrocarbons (PAHs) adsorbed on particles) are hazardous and potentially carcinogenic.

4. IARC has classified diesel engine exhaust as a 'definite' carcinogen in humans, based on epidemiological, experimental and other evidence (Benbrahim-Tallaa *et al.*, 2012; IARC, 2013). It was recognised that many occupations potentially entail exposure both to diesel exhaust and to petroleum exhaust, and to distinguish their effects, IARC concentrated on studies involving exposure to diesel in the absence of petroleum exhaust (IARC, 2013), and therefore on investigations of railway workers, professional drivers (of heavy good vehicles, buses and trams) and miners. The Council has adopted the same approach in this report. Reported increases in risk in IARC's monographs appeared to be most evident for two types of cancer, lung cancer and to some extent bladder cancer, so this report focuses specifically on these two tumours.

5. To supplement the studies included in the IARC monograph, the Council's Research Working Group conducted a literature review in relation to the occupational groups and cancers mentioned above, but found no additional material meriting inclusion in the evidence tables. The studies considered by the Council, and also by IARC, are summarised in a concluding set of tables.

6. As explained in previous reports, before recommending prescription for diseases that 1) are not specific to occupation, and 2) in which cases caused by occupation cannot reliably be distinguished in the individual case from those that would arise irrespective of occupation, the Council normally seeks research evidence that the risks are more than doubled (relative risk (RR)>2) in workers with a given exposure relative to a suitable comparator. The aim is to identify circumstances in which attribution to work can be established on the balance of probabilities.

7. As lung cancer and bladder cancer are not specific to occupation, and occupational instances of these diseases are not distinguishable reliably from non-occupational instances by clinical means, the literature was considered against this threshold of evidence.

Miners

Lung cancer

8. Interpretation of the evidence on lung cancer and diesel emissions in underground miners is complicated by the potential for co-exposure to other carcinogens found in mines (e.g. silica, radon). The Council has decided, therefore, to collect further evidence on the topic, which will be the subject of a separate later report.

Bladder cancer

9. Of the four studies considered by IARC in relation to bladder cancer in miners (Table 1), none demonstrated a doubling of risk (Kogevinas *et al.*, 2003; Guo *et al.*, 2004 (a); Neumeyer-Gromen *et al.*, 2009; Attfield *et al.* 2012; RR<1.4).

10. Overall, therefore, the Council has concluded there is insufficient evidence to recommend prescription of bladder cancer in miners exposed to diesel engine exhaust.

Railway workers

Lung cancer

11. Table 2 summarises seven studies concerning lung cancer in railway workers. Most of these studies did not indicate a doubling of risk. In a large cancer incidence study by Guo *et al.*, 2004 (b), involving population data on more than one million Finns, summary risk estimates for locomotive drivers were not elevated, a finding echoed in a report concerning over 8,000 members of a Finnish trade association of locomotive drivers (Nosko-Koivisto and Pukkala, 1994). Similarly, a case-control study involving almost 600 lung cancer cases in Turin (Richiardi *et al.*, 2006) did not find any increase in risk (among railroad workers). A cohort study of some 43,000 pensioners of the Canadian railways found little increase overall in mortality from lung cancer (RR 1.06), but somewhat higher risks in ex-conductors and yard helpers, and a doubling of risk (RR 2.13) in ex-porters (Howe *et al.*, 1983). Some workers involved in the maintenance of steam locomotives were potentially exposed to high levels of asbestos, although it is unclear how far this contributed to the reported excess risks in these trades. Garshick *et al.*, 2004 and Boffetta *et al.*, 1988 reported risks that were elevated some 40% and 60% respectively. In the first of these studies, lung cancer mortality was inversely related to total years worked, rather than increasing with increasing exposure as might be typical for a causal association. The analysis

by Boffetta allowed for workers' smoking habits. A report, by Swanson *et al.*, 1993, of a population-based case-control study of residents of the Detroit metropolitan area, found a statistically significant doubling of risks among white men employed for 10 or more years (Odds Ratio (OR) 2.4, 95% Confidence Interval (CI) 1.1 to 5.1), and a non-significant doubling among black men with more than 1 year of exposure (OR for 1-10 years, 2.6, Probability (P)>0.05; OR for more than 10 years 2.7, P>0.05).

12. Although these findings are somewhat mixed, the Council has concluded that there is insufficient evidence to recommend prescription of lung cancer in railway workers.

Bladder cancer

13. As for lung cancer, the majority of the five studies that considered bladder cancer in railway workers (Table 3) did not indicate that risks could be as much as doubled (Howe *et al.*, 1983; Nosko-Koivisto and Pukkala, 1994; Kogevinas *et al.*, 2003; Guo *et al.*, 2004 (a)), with summary estimates of RR ranging from below one to about 1.4. An exception was a British mortality study by Dolin and Cook-Mozaffari, 1992, in which mortality risks were raised more than two-fold in railway guards, although findings were not statistically significant and based on only 4 exposed cases (standardised mortality ratio (SMR) 2.6, 95% CI 0.7 to 6.6). In the same study SMRs for railway engine drivers, railway shunters and railway signalmen were also elevated by about 50-60% (P>0.05).

14. On balance the Council has concluded that there is insufficient evidence to recommend prescription of bladder cancer in railway workers.

Bus drivers

Lung cancer

15. Table 4 summarises the five reports identified by IARC on lung cancer in bus drivers. A mortality study in more than 3,000 London bus drivers (including 18 deaths among them from lung cancer) (Balarajan and McDowell, 1988) found a 40% increase in mortality risk, although findings were not significant at the 5% level. However, in a larger study of cancer incidence among bus drivers and tramway employees of the Copenhagen Traffic Company, risks were generally not elevated in most analyses and such risks as were described were not related to duration of employment (Soll-Johanning *et al.*, 2003), nor in a cohort study of over 2,000 bus drivers from

Denmark's three largest cities (Petersen *et al.*, 2010). A survey of cancer incidence in more than one million Finns found no clear relationship with estimates of diesel exhaust exposure and lung cancer (RR 0.99 in men and 1.22 in women, P>0.05) and no relationship with being a male bus driver; risks were raised almost four-fold in female bus drivers, but with only one driver with lung cancer, this estimate was very uncertain statistically (Guo *et al.*, 2004 (b)). Finally, an analysis which pooled data from individual case-control studies in the US (Hayes *et al.*, 1989) reported a 60% increase in risk in those who had driven buses for 10 years or more, a finding that was not statistically significant. In summary, none of the studies provided evidence of a doubling of the risks.

16. Overall, therefore, the Council has concluded that there is insufficient evidence to recommend prescription of lung cancer in bus drivers**.**

Bladder cancer

17. A similar situation exists for bladder cancer in bus drivers (Table 5). Two British studies, one of London bus drivers (Balarajan and McDowell, 1988), a second involving a population sample of men from England and Wales (Dolin and Cook-Mozaffari, 1992), reported lower risks in those with exposure, whereas that by Guo *et al.*, 2004 (a), found a 29% increase in risk of incident bladder cancer in Finnish bus drivers. Two studies of bus drivers in Denmark (Soll-Johanning *et al.*, 2003; Petersen *et al.*, 2010) found increased risks in subgroups, depending on duration of employment and analytic choice. In the study by Soll-Johanning *et al.*, 2003, risks were doubled only in those employed for a brief period (<3 months relative to 3-24 months), with substantial statistical uncertainty; in those with longer employment, risks were less elevated (13% to 61%) with no pattern of increasing risk with increasing service. In the second Danish study (Petersen *et al.*, 2010), risks were increased overall by about 60%, but no trend was found with length of employment. Thus, none of the studies that IARC identified offer convincing evidence of a doubled risk of bladder cancer in this occupation.

18. Overall, therefore, there is insufficient evidence to recommend prescription of bladder cancer in bus drivers.

Lorry and HGV drivers

Lung cancer

19. In all, 13 studies have been reviewed of lung cancer in lorry drivers (Table 6). Four of these studies found an elevation in risk of about 50% to 60% (Menck and Henderson, 1976; Balarajan and McDowell, 1988; Hayes *et al.*, 1989; Guberan *et al.*, 1992) and six found a small (4% to 24%) elevation in risk, or none at all (Boffetta *et al.*, 1988; Hansen, 1993; Jarvholm and Silverman 2002; Guo *et al,*, 2004; Laden *et al.*, 2007; Birdsey *et al.*, 2010). In an American case-control study based on a truck drivers' union membership, RRs tended to increase with years of employment as a driver, up to 1.6- to 1.8-fold among short and long-haul drivers with service of 18 years or more (Steenland *et al.*, 1990). The remaining two studies provided some evidence of a doubled risk. Among a cohort of truck drivers from Reykjavik, Iceland who were members of a drivers' union, the overall SMR for lung cancer was doubled (SMR 2.1, 95% CI 1.4 to 3.2, P<0.05) (Rafnsson and Gunnarsdottir, 1991). Risk estimates varied by duration of follow-up, but the pattern was uneven and sub-analyses were based on fairly small numbers with large statistical uncertainty. In the previously-mentioned population-based case-control study from Detroit (Swanson *et al.*, 1993), ORs were more than doubled for white truck drivers employed for 20 years or more years (2.5, P<0.05) and mostly doubled for black drivers with more than a year of service (with ORs of 2.7, 1.9, and 2.1 respectively for 1-9 years, 10-19 years, and 20 or more years of service; P>0.05).

20. Although findings on lung cancer and lorry driving tend, therefore, to indicate an increase in risk, only limited evidence has been found that risks can be as much as doubled. The Council considers that present evidence is insufficient to recommend prescription of lung cancer in heavy goods vehicle drivers.

Bladder cancer

21. Risks of bladder cancer in HGV drivers have been evaluated in eight studies (Table 7), in most of which findings have been compatible with no effect or a comparatively small one (Balarajan and McDowell, 1988; Rafnsson and Gunnarsdottir, 1991; Hansen, 1993; Siemiatycki *et al.*, 1994; Guo *et al.*, 2004 (a); Laden *et al.*, 2007; Birdsey *et al.*, 2010). In a Swiss study of licensed HGV, taxi, bus, coach and Public Service Vehicle drivers, the SMR was raised 1.4-fold under certain assumptions (Guberan *et al.*, 1992) but all other risk estimates were smaller than this and so far fall short of a doubling of risks.

22. The Council has concluded that on present evidence no case exists for recommending prescription of bladder cancer in HGV drivers.

Summary

23. IARC has classified diesel engine exhaust as a 'definite' carcinogen in humans, as judged by a range of evidence, including animal experiments and mechanistic studies (Benbrahim-Tallaa *et al.*, 2012; IARC, 2013). The epidemiological reports contributing to this view, and which relate to cancers of the lung and bladder in railway workers, bus drivers, and heavy good vehicle drivers, and cancer of the bladder in miners, have been considered in this report.

24. Elevated risks of these cancers have been reported in many of the studies, in keeping with IARC's assessment of the hazard. However, risks have seldom been as much as doubled – the threshold usually required before the Council recommends an exposure-outcome circumstance should be eligible for prescription. IIAC has, therefore, concluded that none of these can be recommended for prescription at this time, although these topics will remain under review.

25. It should be stressed that a disease can still be occupationally-caused and an important focus for preventive activity at lower levels of relative risk than the threshold for prescription. The latter is used to allow attribution to work on the balance of probabilities in individual claimants, in line with the legislative requirements of the Industrial Injuries Disablement Benefit Scheme. This note should not be construed as indicating that the Council disagrees with IARC's classification of diesel engine exhaust as a human carcinogen.

This information note contains technical terms which are explained in an accompanying glossary.

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Glossary

Types of study

Case-control study: A study which compares people who have a given disease (cases) with people who do not (non-cases, also known as controls) in terms of exposure to one or more risk factors of interest. Have cases been exposed more than non-cases? The outcome is expressed as an **Odds Ratio**, a form of **Relative Risk**.

Cohort study: A study which follows up a population of individuals (usually defined by a workplace) over time and compared the rate of disease or mortality among those within the cohort or with an external comparison population. The outcome is expressed as a Rate Ratio or **Relative Risk**, **Standardised Incidence Ratio** or **Standardised Mortality Ratio**, depending on the type of analysis and the disease outcome being studied.

Measures of association

Statistical significance and P values: Statistical significance refers to the probability that a result as large as that observed, or more extreme still, could have arisen simply by chance. The smaller the probability, the less likely it is that the findings arise by chance alone and the more likely they are to be 'true'. A 'statistically significant' result is one for which the chance alone probability is suitably small, as judged by reference to a pre-defined cut-point. (Conventionally, this is often less than 5% ($P<0.05$)).

Relative Risk (RR): A measure of the strength of association between exposure and disease. RR is the ratio of the risk of disease in one group to that in another. Often the first group is exposed and the second unexposed or less exposed. *A value greater than 1.0 indicates a positive association between exposure and disease*. (This may be causal, or have other explanations, such as bias, chance or **confounding**.)

Odds Ratio (OR): A measure of the strength of association between exposure and disease. It is the odds of exposure in those with disease relative to the odds of exposure in those without disease, expressed as a ratio. For rare exposures, odds and risks are numerically very similar, so the OR can be thought of as a **Relative Risk**. *A value greater than 1.0 indicates a positive association between exposure and disease*. (This may be causal, or have other explanations, such as bias, chance or **confounding**.)

Standardised Mortality Ratio (SMR): A measure of the strength of association between exposure and mortality; a form of **Relative Risk (RR)** in which the outcome is death. The

SMR is the ratio of the number of deaths (due to a given disease arising from exposure to a specific risk factor) that occurs within the study population to the number of deaths that would be expected if the study population had the same rate of mortality as the general population (the standard).

By convention, SMRs (and standardised incidence rates (SIR) as described below) are usually multiplied by 100. Thus, an SMR (or SIR) of 200 corresponds to a RR of 2.0. For ease of understanding in this report, SMRs (or SIRs) are quoted as if RRs, and are not multiplied by 100. Thus, *a value greater than 1.0 indicates a positive association between exposure and disease.* (This may be causal, or have other explanations, such as bias, chance or **confounding**.)

Standardised incidence ratio (SIR): An SIR is the ratio of the observed number of cases of disease (e.g. cancer) to the expected number of cases, multiplied by 100. The ratio is usually adjusted to take account of differences in the population evaluated with the comparison or "normal population", due to age, gender, calendar year, and sometimes geographical region or socioeconomic status.

Incidence Rate Ratio (IRR): A form of **Relative Risk**. The ratio of the **incidence rate** of a new event in an exposed population relative to an unexposed one.

Other epidemiological terms

Incidence rate: The rate of occurrence of a new event of interest (e.g. cancer) in a given population over a given time period. (The rate is often expressed in terms of cases per year of 'person-time', and so incorporates the numbers at risk of the event, the time for which they are at risk and the numbers that go on to develop that event.)

Confidence Interval (CI): The **Relative Risk** reported in a study is only an *estimate* of the true value of relative risk in the underlying population; a different sample may give a somewhat different estimate. The CI defines a plausible range in which the true population value lies, given the extent of statistical uncertainty in the data. The commonly chosen 95% CIs give a range in which there is a 95% chance that the true value will be found (in the absence of bias and confounding). *Small studies generate much uncertainty and a wide range, whereas very large studies provide a narrower band of compatible values*.

Confounding: Arises when the association between exposure and disease is explained in whole or part by a third factor (confounder), itself a cause of the disease, that occurs to a

different extent in the groups being compared.

For example, smoking is a cause of lung cancer and tends to be more common in blue-collar jobs. An apparent association between work in the job and lung cancer could arise because of differences in smoking habit, rather than a noxious work agent.

Studies often try to mitigate the effects of ('control for') confounding in various ways such as: restriction (e.g. only studying smokers); matching (analyzing groups with similar smoking habits); stratification (considering the findings separately for smokers and non-smokers); and mathematical modelling (statistical adjustment).

Lagged analysis: This is a method of analysis in which it is assumed that exposure that occurs at a certain interval of time immediately before the occurrence of disease is not relevant to causation and should be excluded in the assessment of the relationship between exposure and disease.

*For example, in calculating the **incidence rates** of cancer in exposed and unexposed populations, a 10 year lagged analysis would discard in its calculations the last 10 years both of person-time and of cancer events*.

Job exposure matrix: This is a tool used to assess exposure to potential health hazards in epidemiological studies in which jobs are assigned their typical exposures and individuals are assumed, by virtue of their job title, to have incurred these exposures.

Appendix – Tables of Epidemiological Evidence

Table 1 – Diesel Exhaust and Bladder Cancer in Miners

Reference	Study Type	Follow-up/Case Ascertainment	Exposure Assessment	Relative Risk (RR) (95% Confidence Interval (CI), Number of exposed cases)
Kogevinas *et al.*, 2003	Pooled analysis of population-based case-control studies from Denmark, France, Germany, Greece, Italy and Spain comprising 3,346 cases, aged 30-79, interviewed within 2 years of diagnosis, and 6,840 age-matched controls.	1976-1996.	Occupational code for the longest-held job. A Finnish national job exposure matrix (FINJEM) was used to estimate exposure to PAHs and diesel engine exhaust exposures and additional specific exposures.	Odds Ratio (OR) (adjusted for age, smoking and study centre) miners, quarrymen, wet drillers and related workers 1.26 (1.00 to 1.58, 147)
Guo *et al.*, 2004 (a)	All economically active Finns born 1906-45 who participated in the national population census in 1970.	1971-1995. Follow-up virtually complete.	Occupation held for the longest period in 1970. Classified by expert review (FINJEM) as having been exposed to diesel but not gasoline exhaust.	Standards Incidence Ratio (SIR) in men in mine and quarry work: Involving metal ore, 1.38 (0.29 to 4.04, 3) Involving non-metal ore, 1.16 (0.73 to 1.76, 22) Involving other work, 0.82 (0.22 to 2.09, 4). Figures were not adjusted for smoking. The study contained a further analysis of diesel exhaust adjusted for smoking, but this was not specific to miners.
Neumeyer-Gromen *et al.*, 2009	Cohort of 5,862 German underground potash miners who worked for at least 1 year in a mine.	1970-2001. Completeness of follow-up not stated.	Estimates of diesel exposure, expressed as total carbon in respirable dust were obtained in 1992, for exposures between 1969 and 1991 when the mine closed.	Overall Standardised Mortality Ratio (SMR) 0.80 (0.40 to 1.60, 8) Smoking data were available, but there were insufficient bladder cancer cases to justify an exposure-response analysis.

Appendix – Tables of Epidemiological Evidence

Reference	Study Type	Follow-up/Case Ascertainment	Exposure Assessment	Relative Risk (RR) (95% Confidence Interval (CI), Number of exposed cases)
Attfield *et al.*, 2012	Cohort of 12,315 blue-collar workers who were employed in one of eight non-metal mines in the USA for at least 1 year after diesel equipment had been introduced. The mines were chosen because there was minimal exposure to silica, radon and asbestos.	1960-1997. Completeness of follow-up not stated.	Exposure to diesel exhaust was assessed blind to mortality data based on respirable elemental carbon for all surface and underground jobs by year and facility using measurements taken 1998-2001.	SMR 1.09 (0.58 to 1.86, 13). No smoking data were available for the cohort study.

Appendix – Tables of Epidemiological Evidence

Table 2 - Diesel Exhaust and Lung Cancer in Railway Workers

Reference	Study Type	Follow-up / Case Ascertainment	Exposure Assessment	RR (95% CI, Number of exposed cases)
Howe *et al.*, 1983	Cohort study of 43,826 male pensioners (retired before 1965) of the Canadian national railway company.	1965-1977. 94% of known deaths were successfully linked to national mortality data.	Occupation retirement was used to classify the cohort as never, possibly or probably exposed to diesel.	SMR: Overall, 1.06 (0.99-1.13, 933). Conductor 1.52 (1.18-2.17, 50) Porter 2.13 (1.19-3.81, 11) Foreman 1.62 (1.17-2.30, 31) Yard helper 1.93 (1.17-6.06, 14) SMR vs. non-exposed: Possibly exposed, 1.20 ($p = 0.013$) Probably exposed 1.35 ($p < 0.001$). (This trend was statistically significant, $p<0.001$). Smoking data were not available for individuals. However, indirect assessment of the possible effect of smoking suggests the possibility of some confounding of the lung cancer associations by smoking.
Boffetta *et al.*, 1988	Sub-cohort of 2,973 railroad workers from a cohort study of over 1,200,000 Americans and Puerto Ricans whose vital status was known at end of 1984. Analysis restricted to men aged 40-79 years in 1982.	1982-1984. 0.8% of subjects lost to follow-up.	Questionnaire, to ascertain current occupation, last job if retired and longest held job. 21% of the cohort did not have data on diesel exhaust exposure.	RR (railroad vs. not) 1.59 (0.94 to 2.69, 14) Analysis adjusted for smoking status.

Appendix – Tables of Epidemiological Evidence

Reference	Study Type	Follow-up / Case Ascertainment	Exposure Assessment	RR (95% CI, Number of exposed cases)
Swanson *et al.*, 1993	Population-based case-comparison study of residents, aged 40-84, of the metropolitan Detroit area. Incident cases from the Detroit cancer surveillance system. Comparison group of cases with cancer of colon or rectum.	1984-87. Completeness of follow-up not stated.	Lifetime work history collected by telephone interview. Occupation and industry codes were assigned using the 1980 US Census Bureau classification codes.	RR, railroad industry vs never: *White workers* 1-9 years, 1.2 (0.5 to 2.7, 27) 10+ years 2.4 (1.1 to 5.1, 40) *Black workers* 0 years 1.0 1-9 years, 2.6 (0.8 to 7.9, 22) 10+ years 2.7 (0.6 to 21.1, 9) RRs adjusted for pack-years of smoking.
Nosko-Koivisto and Pukkala, 1994	Cohort study of cancer incidence in 8391 members of the Finnish Locomotive Drivers' Association	1953-91. Probably a complete follow-up, although not stated.	Based on a reconstruction of conditions related to workshops for steam trains and personal air sampling. Benzo[a]pyrene concentration measured in refurbished cabins. Lack of specific information linking job titles to diesel locomotive use.	SIR (exposed vs. not): Overall, 0.86 (0.75 to 0.97, 236) 0 to 14 yrs since 1st employed, 1.02 (0.37 to 2.22, 6) 15-29 years since 1st employed, 0.73 (0.55 to 0.97 ≥30 years since 1st employed 0.89 (0.77 to 1.02, 187). No adjustment for smoking although it was suggested that the prevalence of smoking in the cohort is low.
Garshick *et al.*, 2004	Cohort mortality study of 54,973 US white male railroad workers aged 50-64 with 10-20 years of service.	1959-1996. Cause of death ascertained for >98% of deaths.	Occupations defined as exposed were engineers and conductors. Clerks and signal maintainers were defined as unexposed.	RR (exposed vs not exposed): Overall, 1.40 (1.30 to 1.51) (The same result was found when a 5-year exposure lag was applied.) Individual data on smoking were not available, socioeconomic status was used as a proxy for this in analysis. Also an indirect adjustment for smoking was also carried out, based on data from an earlier case-control study.

Appendix – Tables of Epidemiological Evidence

Reference	Study Type	Follow-up / Case Ascertainment	Exposure Assessment	RR (95% CI, Number of exposed cases)
Guo *et al.*, 2004 (b)	Cancer incidence cohort study of economically active Finns (667,121 men; 513,110 women) born between 1906 and 1945 who participated in the 1970 census.	1971-1995. Follow up was virtually complete.	Exposure to diesel exhaust was assessed using the job exposure matrix FINJEM.	SIR (male locomotive drivers vs. not) 0.63 (0.51 to 0.78, 85) Analysis allowed for smoking habits.
Richiardi *et al.*, 2006	595 incident lung cancer cases aged <76 and 845 population controls matched for age and sex in Turin, Italy.	1991-1992.	By administered questionnaire - diesel job-specific modules identified pre-defined high exposure jobs.	OR (railroad workers vs not), 0.44 (0.12 to 2.52, 4) Analysis allowed for smoking habits and potential exposure to other lung carcinogens.

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Table 3 - Evidence Table Diesel Exhaust and Bladder Cancer in Railroad Workers

Reference	Study Type	Follow-up/Case Ascertainment	Exposure Assessment	RR (95% CI, Number of exposed cases)
Howe *et al.*, 1983	Cohort study of 43,826 male pensioners of the Canadian national railway company.	1965-1977. 94% of known deaths successfully linked to national mortality data.	Occupation at time of retirement was used to classify the cohort as never, possibly or probably exposed to diesel.	SMR overall, 1.03 (n 175). Smoking data were not available for individuals. However, indirect assessment of the possible effect of smoking suggests the possibility of some confounding for bladder cancer from smoking.
Dolin and Cook-Mozaffari, 1992	Population-based mortality study of 2,457 men aged 25-64 from coastal and estuarine regions of England and Wales.	1965-1980.	Information on occupation and industry was extracted from death certificates. Exposures were assessed using a job exposure matrix.	SMR: Engine driver, 1.61 (0.85 to 2.75, 13) Shunter ,1.49 (0.40 to 3.82, 4) Signalman, 1.46 (0.47 to 3.39, 5) Guard, 2.58 (0.70 to 6.61, 4) Degree of urbanisation was used as a proxy for smoking in analysis
Nosko-Koivisto and Pukkala, 1994	Cohort study of cancer incidence in 8391 members of the Finnish Locomotive Drivers' Association.	1953-91.	Based on a reconstruction of conditions related to workshops for steam trains and personal air sampling. Benzo[a]pyrene concentration measured in refurbished cabins. Lack of specific information linking job titles to diesel locomotive use.	SIR: Overall, 1.08 (0.80 to 1.43, 48) 15-29 years since 1st employed, 1.35 (0.62 to 2.57, 9) $\geq$30 y since 1st employed, 1.05 (0.75 to 1.43, 39). No adjustment for smoking although it was suggested that the prevalence of smoking in the cohort is low.

Kogevinas *et al.*, 2003	Pooled analysis of population-based case-control studies from Denmark, France, Germany, Greece, Italy and Spain consisting of 3,346 cases, aged 30-79, interviewed within 2 years of diagnosis and age and region matched 6,840 controls.	1976-1996.	Occupational code for the longest-held job. A Finnish national job exposure matrix (FINJEM) was used to estimate diesel engine exhaust exposures.	OR (adjusted for age, smoking and study centre): Railway engine drivers and firemen, 1.41 (0.87 to 2.28, 34) Brakemen, signalmen and shunters, 1.43 (0.77 to 2.63, 18)
Guo *et al.*, 2004 (a)	Cancer incidence cohort study of economically active Finns (667,121 men; 513,110 women) born between 1906 and 1945 who participated in the 1970 census.	1971-1995.	Exposure to diesel exhaust was assessed using the job exposure matrix FINJEM.	RR for locomotive drivers), 0.85 (0.53 to 1.28, 22) Analysis allowed for smoking habits.

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Table 4 - Evidence Table Diesel Exhaust and Lung Cancer in Bus Drivers

Reference	Study Type	Follow-up/Case Ascertainment	Exposure Assessment	RR (95% CI, Number of exposed cases)
Balarajan and McDowell, 1988	Cohort mortality study of 3,392 professional drivers (in 1939) from London, identified via the NHS central register.	1950-1984. 100% follow-up.	Analysis based on job title (no estimation of exposures to diesel exhaust).	SMR, bus and coach drivers 142 (0.84 to 2.24, 18) Analysis did not allow for smoking habits.
Hayes *et al.*, 1989	Three pooled US case-control studies from Florida, New Jersey and Louisiana.	1976-83.	Schemes for coding occupations varied by study.	OR, Bus drivers vs not: <10 years, 1.1 (0.6 to 1.7, 38) ≥10 years, 1.6 (0.9 to 2.8, 38) Analysis allowed for smoking habits
Soll-Johanning *et al.*, 2003	Case control analysis nested with a cohort cancer incidence study of 18,174 bus drivers and tramway employees from the Copenhagen Traffic Company. One to four randomly sampled controls were matched to cases on year of birth and vital status with various applied exclusions. Only cases and controls aged <85 years were approached.	1900-1994. Response rate 75%.	Occupational histories determined by Interview with cases, wives, or ex-wives. An air pollution index was constructed using measurements of NO_x, NO_2, CO and benzene and data on traffic congestion.	OR (vs employed 3 months - 2 years): Employed < 3 months, 0.74 (0.23 to 2.39, 5/14 cases/controls) Employed 2 –10 years, 1.26 (0.69 to 2.28, 54/94) Employed 10-20 years, 1.39 (0.69 to 2.81, 22/35) Employed ≥20 years, 0.63 (0.32 to 1.14, 43/144) Findings were similar when analysed with a 10 year lag period. Analyses were adjusted for pack-years of smoking and use of snuff and chewing tobacco.
Petersen *et al.* 2010	Retrospective cohort study 2,037 male bus drivers from the three largest cities in Denmark.	1979-2003 82.6% responded to survey and were not otherwise excluded.	Duration of employment was used as a proxy for exposure to traffic exhaust.	SIR overall, 1.2 (1.0 to 1.4, 100) Incidence Rate Ratio (IRR) (vs <15 years employed): 15-24 years, 0.89 (0.59 to 1.48, 24) ≥25 years, 0.95 (0.55 to 1.63, 25) Analysis allowed for smoking habits

Appendix – Tables of Epidemiological Evidence

Table 5 - Evidence Table for Diesel Exhaust and Bladder Cancer in Bus Drivers

Reference	Study Type	Follow-up/Case Ascertainment	Exposure Assessment	RR (95% CI, Number of exposed cases)
Balarajan and McDowell, 1988	Cohort mortality study of 3,392 professional drivers (in 1939) from London identified via the NHS central register.	1950-1984. 100% follow-up.	Analysis based on job title (no estimation of exposures to diesel exhaust).	SMR bladder cancer bus and coach drivers 0.58 (0.015 to 3.23, 1) Analysis did not allow for smoking habits.
Dolin and Cook-Mozaffari, 1992	Population-based mortality study of 2,457 men aged 25-64 from coastal and estuarine regions of England and Wales.	1965-1980.	Information on occupation and industry was extracted from death certificates. Exposures were assessed using a job exposure matrix.	SMR bus driver 0.81 (0.44 to 1.36, 14) Degree of urbanisation was used as a proxy for smoking in analysis.
Soll-Johanning *et al.*, 2003	Case control analysis nested with a cohort cancer incidence study of 18,174 bus drivers and tramway employees from the Copenhagen Traffic Company. One to four randomly sampled controls were matched to cases on year of birth and vital status with various applied exclusions. Only cases and controls aged <85 years were approached.	1900-1994. Overall response rate 75%.	Occupational histories determined by Interview with cases, wives, or ex-wives. An air pollution index was constructed using measurements of NO_x, NO_2, CO and benzene and data on traffic congestion.	OR (vs employed 3 months - 2 years): Employed < 3 months, 2.00 (0.37 to 10.92, 3/13 cases/controls) Employed 2 – 10 years, 1.18 (0.47 to 2.96, 17/66) Employed 10-20 years, 1.24 (0.46 to 3.33, 15/41) Employed $\geq$20 years, 1.13 (0.47 to 2.68, 33/93) When the data were analysed with a 10 year lag period, the respective ORs were 1.21, 1.23, 1.61 and 1.28. No finding was statistically significant (P>0.05). Analyses were adjusted for pack-years of smoking and use of snuff and chewing tobacco.

Guo *et al.*, 2004 (a)	Cancer incidence cohort study of economically active Finns (667,121 men; 513,110 women) born between 1906 and 1945 who participated in the 1970 census.	1971-1995. Follow-up virtually complete.	Exposure to diesel exhaust was assessed using the job exposure matrix FINJEM.	RR in bus drivers, 1.29 (1.02 to 1.62, 75). Analysis allowed for smoking habits.
Petersen *et al.*, 2010	Retrospective cohort study 2,037 male bus drivers from the three largest cities in Denmark.	1979-2003. 82.6% responded to survey and were not otherwise excluded.	Duration of employment used as a proxy for exposure to traffic exhaust.	SIR overall, 1.6 (1.2 to 2.0, 69) IRR (vs <15 years employed): 15-24 years,1.11 (0.60 to 2.03, 24) ≥25 years, 1.31 (0.70 to 2.48, 25) Analysis allowed for smoking habits.

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Table 6 - Diesel Exhaust and Lung Cancer in Lorry Drivers

Reference	Study Type	Follow-up / Case Ascertainment	Exposure Assessment	RR (95% CI, Number of exposed cases)
Menck and Henderson, 1976	Mortality (2,161) and cancer incidence (1,777) study of lung cancer among white males aged 20-64 in Los Angeles.	1968-70 for deaths and 1972-73 for incident cases.	Occupation on death certificate and in cancer surveillance programme.	SMR truck driver 1.65 (1.35 to 1.99, 58 deaths, 51 incident cases). There was an absence of data on smoking.
Boffetta *et al.*, 1988	Cohort study of 1,200,000 men and women aged over 30 from the US and Puerto Rico. Analysis reported was restricted to men aged 40-79 at enrolment whose status was known at 20-year follow-up.	1982-1984. 0.8% of subjects lost to follow-up.	Questionnaire, to ascertain current occupation, last job if retired longest held job and exposures to any of 12 substances, including diesel exhaust.	RR: Truck driver, 1.24 (0.93 to 1.66, 48) Truck driver ever vs. never exposed to diesel, 1.03 Exposed 1-15 years, 0.87 (0.33 to 2.25, 6) Exposed ≥16 years, 1.33 (0.64 to 2.75, 12) Analysis adjusted for smoking and other occupational exposures.
Balarajan and McDowell, 1988	Cohort mortality study of 3,392 professional drivers (in 1939) from London identified via the NHS central register.	1950-1984. 100% follow-up.	Analysis based on job title (no estimation of exposures to diesel exhaust).	SMR lorry driver, 1.59 (1.41 to 1.79, 280). Analysis did not allow for smoking habits.
Hayes *et al.*, 1989	Three pooled US case-control studies from Florida, New Jersey and Louisiana.	1976-83.	Schemes for coding occupations varied by study.	Truck driver: Overall, 1.5 (1.1 to 1.9, 147) <10 years) 0.9 (0.8 to 1.2, 196) ≥10 years) 1.5 (1.1 to 1.9, 147) Analysis allowed for smoking habits

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Steenland *et al.*, 1990	Case-control study of deaths in Teamsters Union pensioners with 20 or more years of membership vs. deaths from other cancers or motor vehicle accidents.	1982-83. 81% response rate.	Next of kin provided data on work histories (after 1959), but no particulars on diesel exposure.	Long haul drivers: 1-11 years, 1.08 (0.68 to 1.70, 152) 12-17 years, 1.41 (0.90 to 2.21, 228) ≥18 years, 1.55 (0.97 to 2.47, 213) Short haul drivers: 1-11 years, 1.11 (0.61 to 203, 36) 12-17 years, 1.15 (0.63 to 2.43, 37) ≥18 years, 1.79 (0.94 to 3.42, 40) Smoking and other confounders were controlled for.
Rafnsson and Gunnarsdottir, 1991	Cohort study of 1,075 truck drivers (members of truck drivers' union) alive in 1951 in Reykjavik, Iceland.	1951-1988. Probably a complete follow-up.	Analysis based on job title and duration of employment.	SMR, overall, 2.14 (1.37 to 3.18, 24) Follow-up of: 0-9 years, 4.76 (0.12 to 26.53) 10-19 years, 4.17 (0.86 to 12.18) 20-29 years, 1.00 (0.12 to 3.61) 30-39 years, 3.38 (1.89 to 5.57) 40-49 years, 0.63 (0.08 to 2.27) ≥50 years, 1.49 (0.04 to 8.28) Smoking not directly adjusted for, but some smoking data on truck drivers used to assess possible confounding by smoking.
Guberan *et al.*, 1992	Cohort mortality and incidence study in Geneva, Switzerland of 6,630 men with a driver's licence for HGVs (1278), taxis (128), buses and coaches (320), or light duty road passenger vehicles (4,904).	1949-1986 (deaths) and 1970-1986 (cancer registrations). Follow-up 97% complete.	Analysis based on driving qualification as a professional driver (supplied by a licensing bureau).	SMR (professional driver vs. not), 1.50 (1.23 to 1.81, 77) SIR (professional driver vs. not), 1.61 (1.28 to 1.98, 64) Analyses assumed a 15-year latency. Smoking was not controlled for, but smoking data external to the study was used to assess its possible impact as a confounder.

Hansen, 1993	Historical cohort mortality study of 14,225 truck drivers (exposed) and 43,024 other unskilled labourers (unexposed), identified from the Danish 1970 census.	1970-1980. Probably a complete follow-up.	Self-report of work as a truck driver at census.	SMR, cancer respiratory organs, 1.09 (0.79 to 1.46, 44) Smoking was not adjusted for.
Swanson *et al.*, 1993	Population-based case-comparison study among residents, aged 40-84, of the metropolitan Detroit area. Incident cases from the Detroit cancer surveillance system. Comparison group of cases with cancer of colon or rectum.	1984-87. Completeness of follow-up not stated.	Lifetime work history collected by telephone interview. Occupation and industry codes were assigned using the 1980 US Census Bureau classification codes.	Heavy truck drivers (white) 1-9 years, 1.4 (0.8 to 2.4, 78) 10-19 years, 1.6 (0.8 to 3.5, 38) 20+ years, 2.5 (1.4 to 4.4, 121) Heavy truck drivers (black) 1-9 years, 2.7 (0.8 to 9.2, 27) 10-19 years, 1.9 (0.5 to 7.2, 16) 20+ years, 2.1 (0.5 to 9.2, 16) RRs adjusted for pack-years of smoking.
Jarvholm and Silverman, 2002	A mortality and cancer incidence cohort study based on a Swedish register of 389,000 construction workers undergoing health examinations between 1971 and 1992. Male truck drivers (n = 6,364); male drivers of heavy construction vehicles (14,364) and a reference group of male carpenters/electricians (119,984).	1971-1995. The register was estimated to be 80% complete. Loss to follow-up 0.01%.	Occupation as defined on the register.	Standardised Incidence Ratio: vs. carpenters/electricians, 1.29 (0.99 to 1.65, 61) vs. general population, 1.14 (0.87 to 1.46, 61) SMR: vs. carpenters/electricians, 1.37 (1.04 to 1.78, 57) vs. general population, 1.18 (0.89 to 1.53, 57) Analysis allowed for smoking status.

Guo *et al.*, 2004 (b)	All economically active Finns born 1906-45 who participated in the national population census in 1970.	1971-1995. Virtually complete follow-up.	Occupation held for the longest period in 1970. Classified by expert review as having been exposed to diesel exhaust (using NO_2 in air as an indicator) but not gasoline exhaust (FINJEM).	SIR (men) 1.13 (1.04 to 1.22, 620) There were no cases in women. Analysis did allowed for smoking status.
Laden *et al.*, 2007	Retrospective cohort mortality study of 54,319 male truck drivers employed in 1985 in four companies.	1985-2000. Completeness of follow-up not stated.	Cohort members were categorised as drivers or non-drivers.	SMR overall, 1.04 (0.97 to 1.12, 769) No direct adjustment for smoking, but an indirect assessment for confounding was made using survey data.
Birdsey *et al.*, 2010	Cohort study of 156,241 members of a trade association that provided services to independent truck drivers who were regular members between 1989 and 2004.	1989-2004. Completeness of follow-up not stated.	Member of a trade association.	SMR respiratory cancers, 1.00 (0.92 to 1.09, 557). No adjustment for smoking was made.

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Table 7 - Diesel Exhaust and Bladder Cancer in Lorry Drivers

Reference	Study Type	Follow-up/Case Ascertainment	Exposure Assessment	RR (95% CI, Number of exposed cases)
Balarajan and McDowell, 1988	Cohort mortality study of 3,392 professional drivers (in 1939) from London identified via the NHS central register.	1950-1984. 100% follow-up.	Analysis based on job title (no estimation of exposures to diesel exhaust).	SMR lorry drivers, 1.06 (19). Analysis did not allow for smoking habits.
Rafnsson and Gunnarsdottir, 1991	Cohort study of 1,075 truck drivers (members of truck drivers' union) alive in 1951 in Reykjavik, Iceland.	1951-1988. Probably a complete follow-up.	Analysis based on job title and duration of employment.	SMR, bladder and other urinary organs, 1.02 (0.21 to 2.97, 3). Smoking not directly adjusted for, but some smoking data on truck drivers used to assess possible confounding by smoking.
Guberan *et al.*, 1992	Cohort mortality and incidence study in Geneva, Switzerland of 6,630 with a driving licence for: HGVs (1278), taxis (128), buses and coaches (320), and light duty road passenger vehicles (4,904).	1949-1986 (deaths) and 1970-1986 (cancer registrations). Follow-up 97% complete.	Analysis based on driving qualification as a professional driver (supplied by a licensing bureau).	SMR (professional driver vs. not),1.43 (0.80 to 2.36, 11) SIR (professional driver vs. not),1.25 (0.74 to 1.99, 13) Analyses assumed a15-year latency. Smoking was not controlled for, but smoking data external to the study was used to assess its possible impact as a confounder.
Hansen, 1993	Historical cohort mortality study of 14,225 truck drivers (exposed) and 43,024 other unskilled labourers (unexposed) identified from the Danish 1970 census.	1970-1980. Probably a complete follow-up.	Self-report of work as a truck driver at census.	SMR, cancer of urinary organs, 0.98 (0.49 to 1.75, 11). Smoking was not adjusted for.

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Siemiatycki *et al.*, 1994	Population-based case-control study of 484 residents aged 35-70 in Montreal, Canada and 2,412 controls.	1979-1986. Response rates, 84% for cases; 72% for controls.	Detailed job histories were obtained from face-to-face interviews. Hygienists assessed the probability and frequency of exposure and concentration level in the environment.	OR truck drivers vs not: < 10 years 1.1 (0.8 to 1.8, 25) ≥10 years 1.2 (0.8 to 1.9, 26). Analysis allowed for smoking habits and other potential confounders. Controls were a pool of population (533) and cancer (1,879, not lung or kidney) controls
Guo *et al.*, 2004 (a)	Cancer incidence cohort study of economically active Finns (667,121 men; 513,110 women) born between 1906 and 1945 who participated in the 1970 census.	1971-1995. Follow-up virtually complete.	Exposure to diesel exhaust was assessed using the job exposure matrix FINJEM.	RR lorry drivers, 1.01 (0.85 to 1.19, 144) Analysis allowed for smoking habits
Laden *et al.*, 2007	Retrospective cohort mortality study of 54,319 male truck drivers employed in 1985 in four companies.	1985-2000. Completeness of follow-up not stated.	Cohort members were categorised as drivers or non-drivers	SMR overall, 0.80 (0.56 to 1.13, 29). No direct adjustment for smoking, but an indirect assessment for confounding was made using survey data.
Birdsey *et al.*, 2010	Cohort study of 156,241 current and former members of a trade association that provides services to independent truck drivers who were regular members between Sept. 1989 and Dec. 2004.	1989-2004. Completeness of follow-up not stated.	Member of a trade association.	SMR urinary cancers, 0.93 (0.62 to 1.34, 29). No adjustment for smoking was made.