Diesel Exhaust: Human Health and the Workplace

Mining Diesel Emissions Council:
20th Annual Conference

October 9, 2014, Toronto, Ontario

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UNIFOR

Rudolf Diesel

Rudolf Diesel was born in Paris in 1858 to a German couple from Bavaria. His father was a craftsman and his mother a governess and a language teacher.

After graduation from the Munich Polytechnical, Diesel spent several years working with ice making machines.

During this time he continued his work on improving the efficiency of the then used steam engines. Steam engines where only about 15-18% efficient.

His new design was an internal combustion machine that could generate up to 75% efficiency.

By 1898 he had a working model and a patent.

His original dream was to give small craftsmen and farmers an affordable engine run by fuels that were readily available – what we would call today biofuels. **The first diesels were run on peanut oil.**

By 1904, a Russian oil company started using heavy oil distillates in the diesel engine.

By the 1920's the engine was used on trains, tractors, trucks and later automobiles. The first Cummins engine was produced in 1919.

By 1930 diesel engines were used in mines and construction sites. For example, a potash mine in New Mexico dieselized in 1947.

Rudolf Diesel once said **"The use of vegetable oils for engine fuels may seem insignificant today. But such oils may become in the course of time as important as the petroleum and coal tar products of the present time."**

What makes diesel exhaust different from gas powered internal combustion engines?

The diesel engine uses compression to create the high temperature needed to cause combustion.

It uses the same compression to mix the oxygen from the air and the fuel.

This creates a non-uniform combustion with small amounts of un-burnt fuels and lubricants.

There is also small amounts of incomplete combustion products.

In general a diesel engine can emit from 10 to 100 times more mass of particulates than gas powered spark ignition engines.

What is diesel exhaust?

The exhaust from a diesel engine creates a mixture of gasses, fumes and particles.

There may be as many as 20,000 chemicals in diesel exhaust (DE). Over 700 have been identified.

Many of the gasses and fumes then condense to form more particles.

The gasses include carbon dioxide, oxygen, nitrogen, water vapor, carbon monoxide, nitrogen compounds, sulfur compounds, and low weight hydrocarbons.

Diesel particulate matter (DPM) found in the exhaust includes elemental carbon, absorbed organic carbon compounds, sulfate, nitrates, hydrocarbon particles and some trace metals.

What is diesel exhaust?

Many of the gasses and fumes then condense to form more particles.

These elemental carbon particles have two phases:

- The initial nucleation phase,
- And than an accumulation phase.

The particles in the nucleation phase combine to form larger particles in the accumulation phase.

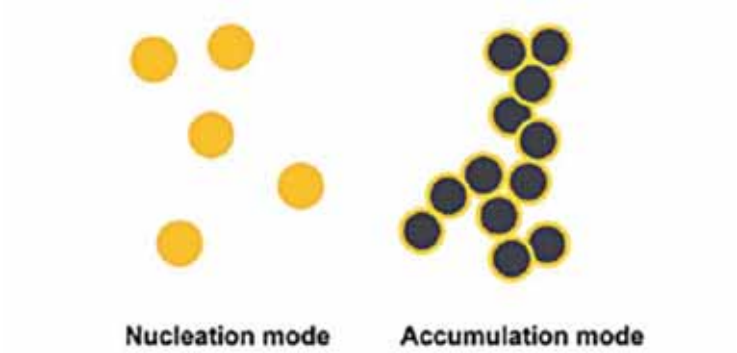


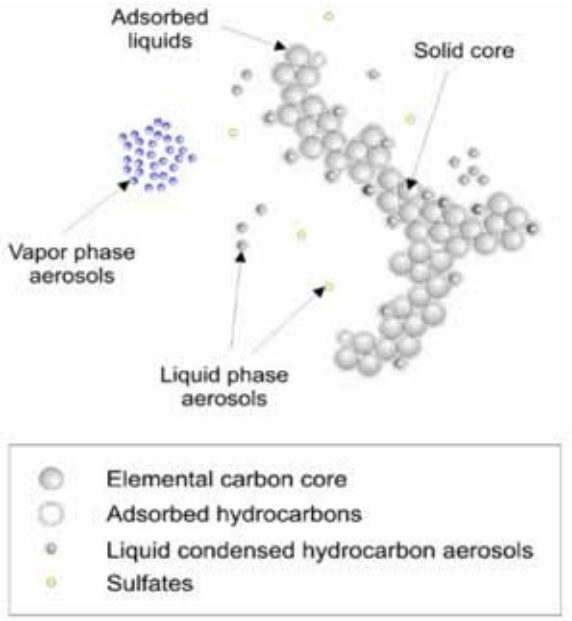
Figure 3 Diesel particulate matter composition and structure for assessing its respiratory health effects. (●) primary carbon particle; (○) adsorbed organics; (●) organic particle.

A graphic of the two types of particles

The components of DE that may cause cancer are:

- particles or carbon core;
- the organic substances and metals that are absorbed to the carbon core;
- and the organic compounds present in the gas/liquid phase.

Elemental carbon particles absorb hydrocarbons and then these smaller particles can merge to form larger particles.



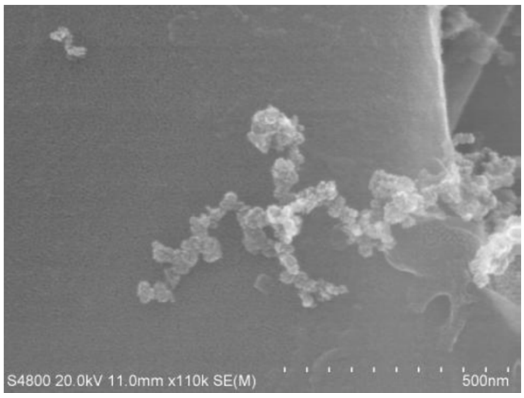
How large and small the particles are important from a human health point of view.

- Generally larger particles deposit in the nose/throat and upper airways of the lung.
- Smaller particles get farther in and are deposited on lower bronchi and in the alveoli.
- The smaller size particles have a higher surface area to mass ratio. It is the surface area where the organic hydrocarbons /metals are absorbed.
- A smaller particular carries correspondingly more organic carbon (OC) per mass than larger particles. It is the OC portion of DPM that many researchers believe is biologically active and dangerous. Others believe that the carbon core itself can cause biological responses.

Approximately 92% of the particles emitted from diesel engines are less than 1.0 micrometer (µm) in diameter. (NTP 12th Report).

Because of their small size they easily reach the bronchi and the alveoli.

This is an electron microscope picture of a DE particle.



Lung deposition by particle size, mass and number.

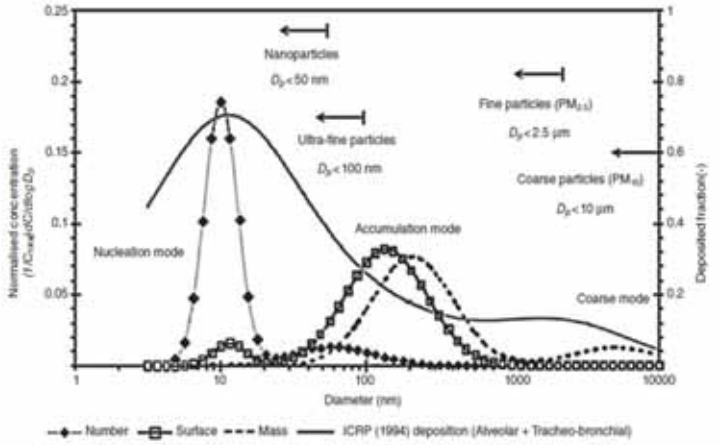


Figure 4 A graph showing the relationship between various diesel particulate matter particle metrics (number, surface and mass) and the deposition of particles in the tracheo-bronchial and alveolar regions of the human lung (adapted from Kittelson *et al.*[36] with permission).

Why we are concerned about organic carbon?

The second largest mass of carbon from diesel exhaust is organic carbon (OC).

These chemicals are formed from the incomplete burning of the diesel fuel (or lubricating oils).

The fuels and/or lubricating oils can be heated enough in the engine to turn into a gas phase, or they may be incompletely burned and turn into various other types of organic carbon compounds such as Polycyclic aromatic hydrocarbons (PAHs).

These OC compounds can then be absorbed onto the elemental carbon, or stay in the gas phase. Either way they can get access to the deepest parts of the lung.

Why we are concerned about organic carbon?

(continued)

These OC compounds include formaldehyde, acrolien, benzene, 1,3 butadiene, Polycyclic aromatic hydrocarbons (PAHs), nitro-PAHs, alkanes, alkanes, quinones.

"Many of these hydrocarbons are known to have mutagenic and carcinogenic properties." (HEI, 1995)

OC can also form particles without being absorbed onto the elemental carbon (EC).

These particles are in the nanometer size range.(NIOSH)

Again, they act like a gas and get deep into the lung.

Two other particulate compounds found in DPM are ash and sulfuric compounds.

Ash is mostly traces of metal that are introduced as additives to the fuel or lubricating oils. Metal from engine wear is also a contributor to ash, which forms a much smaller portion of the overall DPM.

Sulfuric compounds also can be absorbed onto the carbon cores. New ultra low sulfur content fuels and lubricating oils should greatly reduce this fraction of DPM.

This is a good example of control at the source- we should eliminate sulfur in the fuel.

Poison Tipped Arrows

DPM include:

- a large number of particles that can lodge deeply in the lungs;
- many of the particles are in the nanometer size range;
- and the particles are coated with biologically active chemicals.

What about other gasses?

Carbon Monoxide is one of the gasses found in DE.

When you breathe in carbon monoxide, it builds up quickly and combines with the blood to produce "carboxyhemoglobin" (COHb), which reduces the ability of blood to carry oxygen.

*At **low levels**, symptoms include headaches, tiredness, shortness of breath and impaired motor functions. These symptoms sometimes feel like the flu.*

*At **high levels**, or if people are exposed to **low levels for long periods of time**, people can experience dizziness, chest pain, tiredness, poor vision and difficulty thinking.*

*At very **high levels**, carbon monoxide can cause convulsions, coma and even death.*

It is regulated in most workplaces. The ACGIH TLV 25 ppm
[American Conference of Governmental Industrial Hygienists]

Sulfur Dioxide

Sulfur Dioxide is in diesel exhaust is created through oxidation of the sulfur in the fuel or lubrication oils.

It will be greatly reduced with ultra-low sulfur diesel fuels (ULSDF) and low sulfur lubrication oil.

High exposures can cause life-threatening throat and lung damage and it implicated as a cause of Asthma and Reactive Lung Disease.

It is regulated in most workplaces.

The ACGIH TLV (as a ceiling and short term exposure limit) is 0.25 ppm.

Nitrogen Dioxide

Nitrogen Dioxide is an irritant to the lower respiratory tract. If it reaches the bronchi and alveoli, the gas has time to react and cause extensive damage.

It often takes several hours for a worker to feel the effect of the gas. At higher levels it can cause fatal pulmonary damage.

At lower levels (5 -20 ppm), nitrogen dioxide may result in eye irritation, and throat irritation. Exposure at this level over time may lead to chronic bronchitis and damaged red blood cells.

NO2 is regulated in most workplaces. The ACGIH TLV is 0.2 ppm.

Nitrogen Oxide (NO)

Nitrogen oxide (NO) is not considered as toxic as nitrogen dioxide. However, because it can easily be oxidized it can be more dangerous NO2.

It causes severe lung damage and even death at high concentrations.

NO is regulated in most workplaces. The ACGIH TLV is 25 ppm.

Diesel exhaust is made up of a mixture of gasses and particles.

Most of the major gasses emitted are regulated.

Acute Exposure to Diesel Exhaust

Higher exposures to DE causes eye, throat and nose irritation including watering, irritation, reddening and pain.

At higher exposure levels, both the sulfur oxides and the nitrogen oxides are known irritants as demonstrated by individual case reports and clinical human studies.

Along with the irritation, exposed workers may develop non-ordinary coughs and phlegm.

The irritation effects and cough and phlegm effects have been replicated in several animal studies.

There are scientific articles documenting DE causing/exacerbating asthma.

Acute Exposure to Diesel Exhaust

Exposure to DE also increases the severity of reactions to allergens.

" Coexposure to DPM and ragweed pollen was reported to significantly enhance the immunologlobin E antibody response relative to ragweed pollen alone" (Ris). DPM increasing allergic reactions have been replicated in animal studies.

Human trials found that DPM exposure increases antibody production and inflammation of nasal tissue.

These biochemical markers are associated with asthma.

Air pollution health: Cardiovascular disease is associated with particulate exposure.

"It is the opinion of the writing group that the overall evidence is consistent with a causal relationship between PM(2.5) exposure and cardiovascular morbidity and mortality. This body of evidence has grown and been strengthened substantially since the first American Heart Association scientific statement was published."

(Brook 2010)

DPM, a type of PM, is a significant contributor to air pollution.

PM (2.5) describes particles less than 2.5 um.

"Brief exposure to dilute diesel exhaust increases myocardial ischemia and impairs endogenous fibrinolytic capacity in men with stable coronary heart disease"

(Mills)

Myocardial ischemia is reduced blood flow to the heart.

Endogenous fibrinolytic capacity is the enzyme that clears plaque buildup from blood vessels.

These specific effects occur during exposure and up to six hours afterwards.

These same authors also stated that it was the particulate matter in DE, not the gasses, that "mediates" this effect.

The lesson learned is that acute exposure to Diesel Exhaust can cause:

- Eye, nose, throat irritation;
- coughs and phlegm;
- allergic reactions;
- asthma attacks;
- short term exposure is also linked to heart disease in already compromised people.

Chronic Exposure to Diesel Exhaust

There are two categories of diseases associated with long term exposures to DE:

- non-cancerous lung diseases:
  - chronic cough and phlegm
  - potentially leading to Chronic Obstructive Pulmonary diseases (COPD) and asthma.
- cancers

A small but measurable risk

A cohort study of railroad workers found a 2.5% increase risk for COPD in workers exposed diesel exhaust from locomotives

A recent study at the Kaiser Permanente Northwest Health Maintenance Organization found that those exposed to diesel exhaust had a 90% increase of COPD compared to those never exposed.

COPD was even more severe in non-smokers.

Lung Toxicity

Lung toxicity is related to the particles of elemental carbon and the associated OC, ash and metals found on the surface of the particle.

The greater the size of the particle surface area, the greater its potential toxic effects on the lung.

Smaller particles have a higher effective surface area.

Lung Toxicity

DPM causes inflammation through a series of steps involving the body's defense mechanisms.

A particle gets into our body. This generates a chemical, immunological and cellular (macrophage) response.

These chemical, immunological and cellular responses increase local inflammation.

With chronic exposure there is a continued response that leads to chronic inflammation.

The chronically inflamed cellular structures can be damaged which, in turn, can lead to cellular death and fibrosis.

Lung Toxicity

DPM also increases free-radicals within cells, which is related to oxidative stress. Free-radicals are chemicals that are more reactive than oxygen. They can damage cell tissue.

Imbalances between free-radicals and anti-oxidants is called oxidative stress.

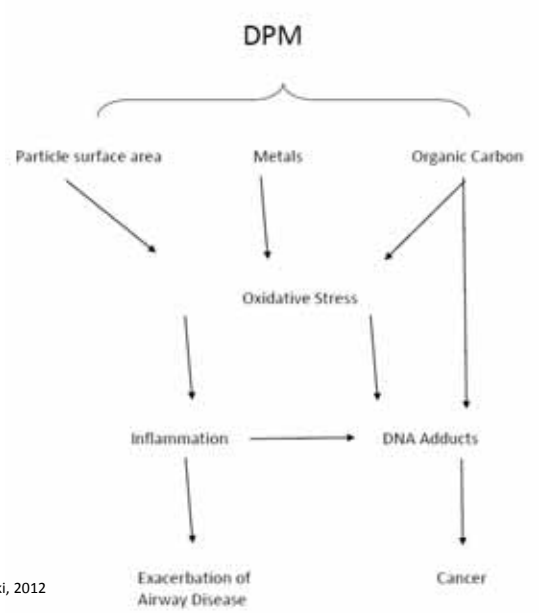
Healthy cells are balanced between the two.

Chemicals that increase free radicals are found on the surface of the carbon particle, or are produced by the defense systems of lung cells as they react to the surface of the carbon particles.

Lung Toxicity

Excess free radicals, whether caused by DPM or by the body's response to exposure to DPM, can cause molecular interactions such as:

- tissue damage;
- Inflammation;
- and generate damage to DNA.



Derived from Ristowski, 2012

Cancer and Diesel Exhaust Exposures

That diesel exhaust may cause lung cancer was first reported in 1955.

Lab experiments identified that PAHs , a known mutagen, was associated with the particulate fraction.

Throughout the 1980 various animal studies found a relationship between lung cancers and exposure to DE.

IARC, in its 1988 review of studies about the connection between DE and lung cancer, found that the animal experiments were convincing, but that the human epidemiological studies to date were not.

[International Agency for Research on Cancer]

No Dose – Response

In 1995 the Health Effects Institute (HEI) argued that "exposure data" for humans was inadequate.

The U.S. EPA summery of health effects of diesel exhaust came to a similar conclusion in 2007,

" Given the equivocal evidence for the presence or absence of an exposure-response for the studies of railroad workers and exposure uncertainties for the study of truck drivers, it is judged that available data are too uncertain at this time …"

The National Toxicology Program in it's twelfth report echoed the EPA Summery and 1995 HEI.

"Exposure to diesel exhaust particulates is reasonably anticipated to be a human carcinogen, based on limited evidence of carcinogenicity from studies in humans and supporting evidence from studies in experimental animals and mechanistic studies." (NTP, 2011).

New studies find a clear dose-response

In 2011 a large study by Olsson, which pooled eleven smaller studies from Canada and Europe, found a small but consistent association with lung cancer.

Workers exposed at Low Levels for 30 years have a small but significant increased risk of 1.17% when adjusted for age, sex, and smoking.

New studies find a clear dose-response

For the workers assigned to the **High Levels** the study found a clear dose-response with the number of years exposed:

Duration (years)	Odds Ratio
O	**1.00**
1-10	**1.28**
11-20	**1.21**
21-30	**1.52**
>30	**1.45**

[An odds ratio of 1.5 means that the workers have a fifty % extra risk compared to the background lung cancer rate in the general population. For the US population that background rate is about 5% for developing lung cancer in a lifetime.]

DEMS Studies 2012

Eight US non-metal mining facilities with over 12,000 workers were chosen for these studies. Underground and surface miners were included.

These were strong studies because of extensive occupational hygiene monitoring in the mines and because past exposures were modeled by correlating diesel horsepower in the mine, ventilation in the mines and existing historical carbon monoxide CO data.

These studies also were adjusted for smoking, and other lung carcinogens caused by radon and asbestos exposures.

Attfield: a cohort study

In this study, the workers' health outcomes are then compared to the general population.

The unadjusted complete cohort of underground and surface miners found an elevated and statistically significant increase in lung cancer as compared to the general population of 1.26.

Also for the complete cohort the study found elevated esophageal cancer and elevated death due to pneumoconiosis.

The researchers also found increased, but not statistically significant, bladder cancers in workers who worked only above ground.

Attfield: a cohort study

For ever-underground workers the risk of lung cancer increased with level of exposure to respirable elemental carbon (REC) exposure. The more dose of accumulated EC, the higher the risk or Hazard Ratio (HR) to die from lung cancer.

The hazard ratios rose to a maximum for 15-year lagged cumulative REC exposures in the 640 to <1280 µg/m3-y category excluding workers with less than 5 years tenure (**HR = 5.01**, 95% CI = 1.97 to 12.76, P = .001),"

That is a 500% increase in risk over the general population.

Attfield: a cohort study

Cumulative exposure is adding up each workers exposure over the years of employment to get their total exposure over their work life. This is expressed as ug/m3-yrs.

A worker exposed to 10 ug/m3 over ten years has a cumulative dose of 100 ug/m3-yrs. A worker exposed at 10 ug/m3 for twenty years has a dose assigned of 200 ug/m3-yr.

Lagging is an analytical tool that excludes the last years of exposure. The idea is that lung cancer takes years to develop and the last years of exposure are not that relevant to disease outcomes.

Attfield: a cohort study

In the above study a worker exposed to 22 ug/m3 for a life time of work (45 years). With his last 15 years of exposure excluded, he has a HR of 5.01, or 500 times the background rate.

This study also looked at hazard ratios at low chronic exposures and what they found was similar to other epidemiological studies.

"These hazard ratios, possibly applicable to reported arithmetic mean exposures of 3–5 µg/m3 for highway and local drivers , are similar to what we found for surface-only workers at equivalent levels of exposure (HR of 2.33 at the average REC level for the 2 to <4 µg/m3 category."

Garshick: Transportation Workers in US

The Garshick paper (Garshick, 2012) analyzed the records of over 50,000 unionized trucking industry workers.

The authors found that their risk (Hazard Ratio) was similar to what was found in the Attfield study, after converting their findings into the cumulative metric used by Attfield:

"This calculation resulted in an HR of 2.77 (95% CI: 0.85, 9.00) per 1,000 µg/m3-years, indicating overlap between the relative risks estimated for the two study cohorts." (Garshick, 2012)

Garshick concluded that lung cancer mortality increased with the cumulative exposure to EC.

Silverman: Case-Control Study

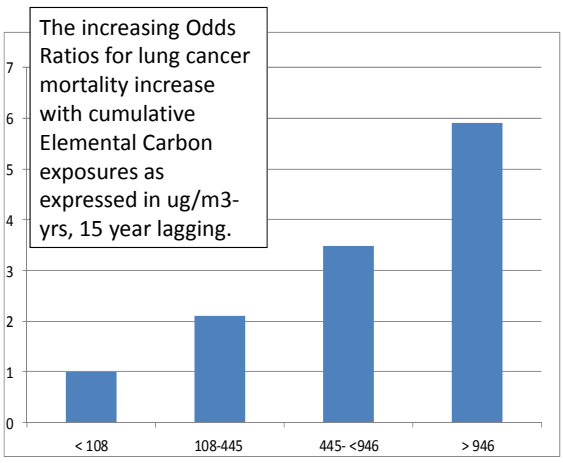
The other DEMS study is a case-control study and was published in 2012. The authors found;

"This case–control study nested within a cohort of miners showed a strong and consistent relation between quantitative exposure to diesel exhaust and increased risk of dying of lung cancer."

"To our knowledge, this is the first report of a statistically significant exposure–response relationship for diesel exposure and lung cancer based on quantitative estimates of historical diesel exposure with adjustment for smoking and other potential confounder." (Silverman, 2012)

Silverman: Case-Control Study

OR



Silverman: Case-Control Study

"Environmental exposure to average elemental carbon levels in the 2-6 µg/m3 range over a lifetime as would be experienced in highly polluted cities approximates cumulative exposures experienced by underground miners with low exposures in our study.

Because such workers had at least a 50% increased lung cancer risk, our results suggest that the high air concentrations of elemental carbon reported in some urban areas may confer increased risk of lung cancer. "

IARC

Lyon, France, June 12, 2012 -- After a week-long meeting of international experts, the International Agency for Research on Cancer (IARC), which is part of the World Health Organization (WHO), today classified diesel engine exhaust as carcinogenic to humans (Group 1), based on sufficient evidence that exposure is associated with an increased risk for lung cancer.

Dose-Response and Risk Estimates

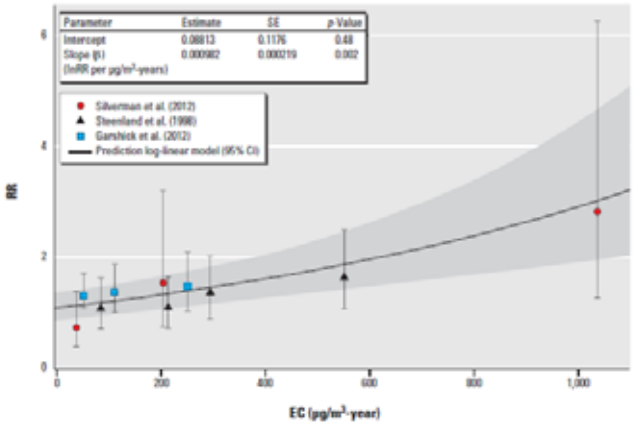


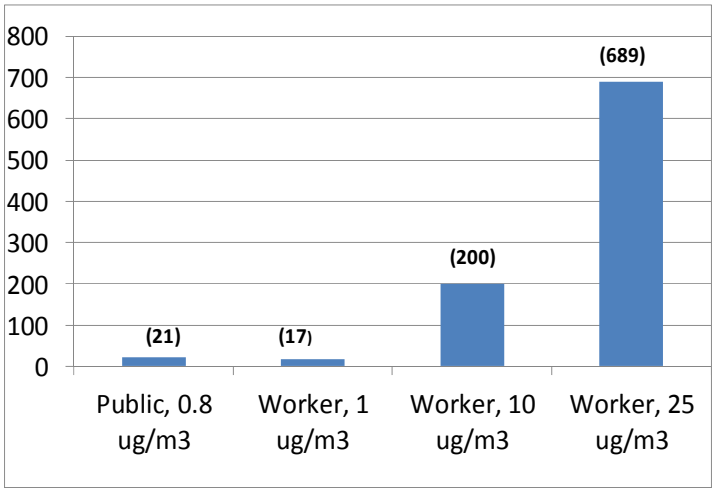
Figure 1. Predicted exposure–response curve based on a log-linear regression model using RR estimates from three cohort studies of DEE and lung cancer mortality. Individual RR estimates [based on HRs reported by Garshick et al. (2012) or ORs reported by Silverman et al. (2012) and Steenland et al. (1998)] are plotted with their 95% CI bounds indicated by the whiskers. The shaded area indicates the 95% CI estimated based on the log-linear model. The insert presents the estimates of the intercept and beta slope factor, the SE of these estimates, and the associated *p*-values.

Dose-Response and Risk Estimates

The curve predicts that the a cumulative exposure of approximately 640 ug/m3-yrs carries a Relative Risk (RR) of 2, or a two hundred % increase risk of lung cancer compared to the background rate of lung cancer.

CAREX Canada is a Canadian organization funded by the Canadian Partnership Against Cancer. Their researchers identify populations exposed to carcinogens in the workplace and the environment. They calculated that at 120 ug/m3-yrs cumulative exposure there will be an RR of 1.27, or a 27% increase in risk.

Excess Lung Cancer Deaths per 10,000 Due to Diesel Exhaust



Vermeulen, et al . 2014, Environ Health Perspect

NIOSH researcher risk assessment using DEMS

Dr. Robert M. Park, Risk Analysis Branch - NIOSH presented his research at the HEI workshop held March 6, 2014. This is his analysis, not NIOSH's.

Dr. Park used the DEMS study to find what exposure to DE would not increase the risk of lung cancer higher that 1/1000.

This is based upon 45 years of employment, 40 hours worked per week.

0.42 ug/m3.

NIOSH Recommended Exposure Limits are not regulatory limits. Regulatory limits take into account technical and economic feasibility.

Exposures in the workplace

CAREX Canada estimates there are 800,000 workers in Canada exposed to diesel exhaust.

The two largest groups of workers are truck drivers and heavy equipment operators. Other transportation workers are also exposed such as taxi drivers, auto/truck repair and maintenance, and school bus drivers.

The mining sector, while smaller than transportation, has some of the highest exposures to DE.

In Alberta there is estimated to be over 100,000 workers exposed to diesel exhaust, and up to 190,000 workers exposed in Quebec.

Exposure Profile of Various Occupations to Elemental Carbon (EC)

Pronk, et al. , Journal of Exposure Science and Environmental Epidemiology, (2009) 443-457.
•National Toxicology Program, Department of Health and Human Services, Report on Carcinogens, Twelfth Edition (2011).

Occupation	Lower exposure range, ug/m3.	Upper exposure range, ug/m3.
Underground mining- #	27	658
Tunnel construction -#	132	314
Underground maintenance - #	53	144
On-road maintenance -#	0	50
Surface mining -*	13	23
Enclosed space mining -*	0	1280
Bus mechanics -*	20	40
Fire fighters -*	10	40
Highway construction -*	4	13
Dock/forklift drivers -*	4	122
Loading ships -*	6	49
On-road driver -#	0	25

Elemental Carbon Exposure Profiles

The exposure ranges reported here may not be similar to a particular workplace in Canada.

Some of these reported exposure levels will be lower or higher depending on the workplaces circumstances.

But these reported exposures can help give you a perspective on potential exposures in a workplace.

Occupational Exposure Levels (OELs)

DE is a complex mixture of gasses and particulates.

Most of the gaseous portion of the exhaust are regulated by all jurisdictions.

Diesel exhaust is now measures by the surrogate of elemental carbon (EC), and in some cases total carbon (TC).

ACGIH does not have a TLV at this time but it did propose a TLV in 2001 and 2002 of **20 ug/m3**.

The proposed change was withdrawn in 2003.

A short list of regulatory limits is found below.

Jurisdiction **Diesel Exhaust Particulate Regulatory Limits/Recommended limits**	RCD ug/m³	TC ug/m³	EC ug/m³
MSHA (USA)		160	124
Ontario (underground non-coal mines)		400	308
Quebec (underground non-coal mines	600		
ACGIH (2002, 2003 withdrawn)			20
Germany (TRK) all workplaces			100
EPA (USA) RfC		5	3-4
Australia (proposal AIOH)			100
Finland - Mining			20
Finland - General Workplaces			5
Canada-Federal	None	None	None
Canada Non-Federal	None	None	None

1 RCD: Respirable Combustible Dust
2 TC: Total Carbon
3 EC: Elemental Carbon
4 TRK: Technically Achievable Concentration
5 RfC: Non-carcinogenic effects – general public
6 AIOH: Australian Institute of Occupational Health

The Ontario Regulation 854 also has requirements on the type of engines, the **testing** of engines and the type of fuel that can be used.

Specifically low sulfur fuels.

There is also a restriction of carbon monoxide emission from engines.

The regulations also set required workplace DE exposure monitoring.

All the monitoring and testing must be done in consultation and the review of the Joint Health and Safety Committees.

Except for the mining sectors in Ontario and Quebec, there are no specific regulations for workplace exposures to DE.

NEW Diesel

"New Diesel" is a term for the emissions in newer diesel engines that were mandated in Canada, the US and Europe.

The newer engine designs and associated technologies were driven by stricter air emission standards. The new engines and the new low sulfur fuels, and biofuels reduce emissions and increase engine efficiency.

This is great news for workers, the general population and the environment!

NEW Diesel

"In meeting the U.S. EPA 2007–2010 emission standards for heavy-duty diesel engines, the exhaust composition of new engines will be markedly changed because of the reduced mass of particulate emissions.

The consequence of this reduced mass is that health hazards, as benchmarked by the mass of DPM emitted, are expected to be significantly reduced." (Ris, 2007)

NEW Diesel

Fortunately, increasingly stringent on-road emission standards for diesel engines were introduced in the United States, the European Union (U.S. 2010 and Euro 6 standards) and other countries (e.g., China, India, Brazil) following with a delay of about 5–10 years (Scheepers and Vermeulen 2012).

These regulations spurred the development of new diesel engine technologies (integration of the wall-flow diesel particulate filter and diesel oxidation catalyst) that on a per-kilometer basis achieve a > 95% reduction of particulate mass and nitrogen oxides emissions (Scheepers and Vermeulen 2012).

Next steps - Stakeholders?

- Regulate Exposures to DE in all Canadian Workplaces.
- Enforce Strict Limits to Exposure. IRS only works in a sea of enforcement.
- Increase Workers Rights.

Lesley Rushton, A widely respected expert in public health field in an editorial of the Journal of National Cancer Institute (JNCI) :

"The continuous analyses and particularly the lagged categorical analyses in the case – control study by Silverman et al., indicate a sharp rise in risk at lower levels and show that background levels of 1 – 2 µ g/m 3 are still likely to carry a small excess risk; substantial proportions of the population exposed at these low levels of exposure would thus continue to contribute to the burden of cancer from DEE.

These results indicate that ***stringent occupational and particularly environmental standards*** for Diesel Exhaust should be set and ***compliance ensured*** to have an impact on health outcomes."