HEALTH EFFECTS - HEAVY DUTY DIESEL ENGINES

LITERATURE STUDY

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ABSTRACT

As a continuation of the report ”Comparing Exhaust Emissions From Heavy Duty Diesel Engines using EN 590 vs. MK1 Diesel Fuel” (28) and as a request from that projects reference group a study on health effects caused by diesel engine emissions have been performed. The goal was to determine if the emission difference that a change of the diesel fuel quality in Sweden would bring would give any measurable health effects.

AVL in Sweden has great knowledge concerning combustion engines, there function, there emissions and the way to measure and quantify improvements, but there is a clear lack of knowledge when it comes to health effects on humans. So to be able to perform the task asked to us from the Swedish Transport Administration (STA) we needed to seek expert help.

Prof. Ulf Rannug, from Stockholm University, has written chapters two and three. AVL has written chapters one and four, plus merging the different chapters together to form a complete report.

With the data that has been available to Prof. Rannug a fuel quality change could theoretically result in an increase of cancer cases in Sweden due to diesel emissions with 25 to 60 cases / year.

This number should just be seen as an indication of possible effects if fuel quality would be changed. Also the estimate is built on data gathered from earlier engine technologies.

To be able to determine the current health benefits and, in the future, possible / reduced / non existing, health benefits from continuing use of Mk1 diesel fuel compared to European standard diesel additional emission data needs to be gathered from modern diesel engines.

It is our recommendation to gather more data through emission tests using modern engine technologies to see there sensitivity to fuel quality.

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1 BACKGROUND

All human activities affect the environment. Those activities can have impact on the local, regional and/or global scale. Today the main discussions in the society are focused on the global influence – effects on greenhouse gas emissions. However, historically, the focus has mainly been on local and regional effects. In this report the focus will be on local and regional emissions and there health effects but in any evaluation of environmental effects both issues must be considered.

Air quality in Swedish cities has improved considerably over the last decades. Reason being reduced emissions from transport, industrial activity and households. Still measurements performed by Swedish Environmental Research Institute (IVL) have shown that many Swedish cities have particulate matter and nitrogen dioxide levels at, or above, the specified air quality requirements in the European Union air quality directive, 2008/50/EC. Several cities also experience difficulties to comply with the environmental quality standards for ground-level ozone.

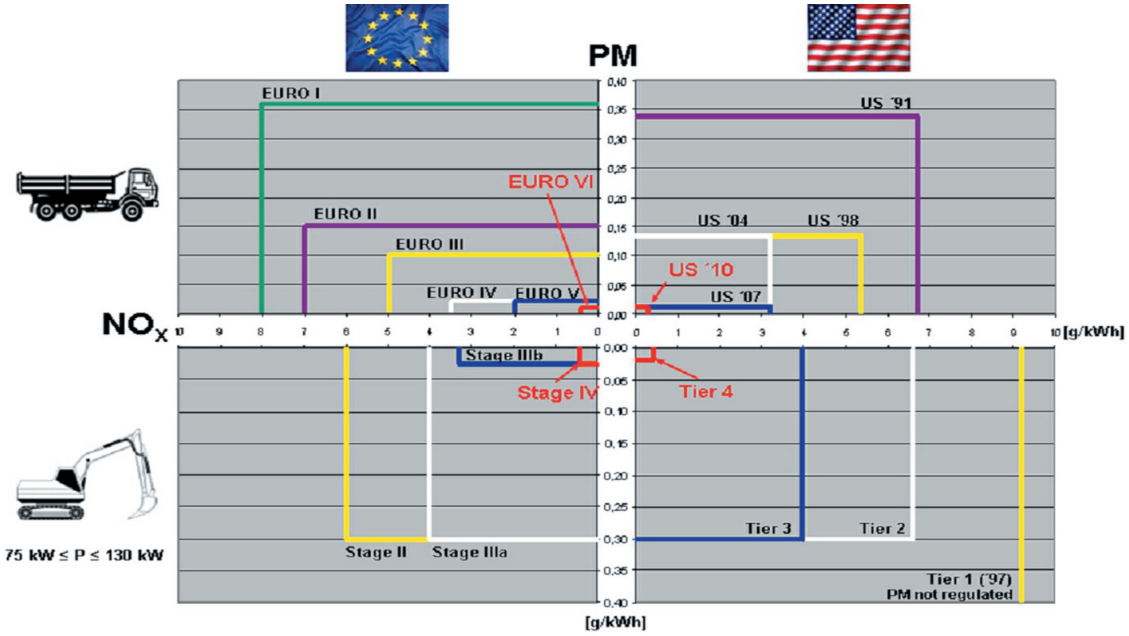
There are a number of different sources for emissions in our society. Combustion engines contribute to a large part of what we today consider as hazardous air pollutants, but there are also often other sources for the same type of pollutants.

This report will address components that can be found in heavy duty on-road and non-road diesel engine emissions and how they affect ambient air quality and the human health. The diesel engine contribution to each component will also be discussed.

The development of engine technology for heavy duty diesel engines over the last 10 to 20 years has mainly been driven by more and more stringent emission regulations and the effort from manufacturers to reduce fuel consumption by making engines more fuel efficient. The huge development can be illustrated by Graph 1, below where the changes of limit values for NO_x and PM emissions in Europe and the US that has been implemented from 1989 and onwards can be seen.

To render some engine technologies possible there has been a need for cleaner fuels. The most obvious improvement is the massive reduction in diesel fuel sulphur, from maximum 2000 ppm in 1998 to maximum 10 ppm (Europe) today.

Graph 1
European and North American NO_x and PM Emission history



Regulated emission components are carbon monoxide (CO), total hydrocarbon (THC), Nitrogen oxides (NO_x) and particulates (PM) and lately, for Euro VI emission levels, also the particulate number (PN). Many other pollutants in the exhaust are of equal or higher interest in health effects evaluation and are, so far, not covered by emission legislations. Since those emissions are not regularly measured it is unclear if those emissions have gone through the same improvements over the years.

During the last 20 years the Swedish traffic work for trucks and buses, expressed as vehicle kilometres, have increased with almost 30%. During the same time period the regulated emissions for those vehicles have been reduced considerably, more that the increased traffic work. A possible conclusion could be that the traffic work increase has been more than compensated for by the emission regulation development resulting in improved air quality.

There are, however, uncertainties in that assumption.

- Are the emissions measurements carried out according to the European test cycles for certification purposes representative for normal driving or are there large differences when the engines are used outside of the regulated areas (off-cycle)?
- How have the unregulated emissions been affected by new engine and emission aftertreatment technologies? It is not clear that emission levels for those components have been reduced to the same extent as the regulated ones.

In an investigation initiated by The Swedish Environmental Protection Agency it was concluded that engine technology and driving conditions had large effects on emissions, but that much could also be done by improving the fuel quality. As an outcome from this work Sweden introduced environmental classification of diesel fuels and from early 1990s the cleanest of those diesel fuels, Mk1 – according to SS 15 54 35, have dominated the Swedish market.

From the time of that investigation the European diesel quality, according to EN 590, has improved immensely. A study, financed by Swedish Transport Administration (STA) was conducted in 2010 where available literature comparing emissions from heavy duty diesel engines operating on Swedish Mk1 diesel fuel and European standard diesel fuel according to EN 590 was summarized into a report. From the literature study it could be confirmed that Mk1 still have emission benefits in earlier engine technologies (up to Euro III and Stage I), even compared to the improved EN 590 of today. Concerning newer engine technologies there was not enough data to make the same conclusion. The report can be found on Swedish Transport Administration (STA) homepage[1].

One of the findings of the literature study was that there is a lack of data regarding fuel sensitivity for the latest engine technologies. In conjunction with the final literature study report a presentation was held for the reference group[2]. It was recommended that further investigations should be conducted, this time using real engine tests to gather data to fill in the gap in information verified by the literature study. During the discussion that followed the presentation the reference group discussed “what is a relevant emission difference”? It was then decided that an initial investigation into the health effects of emission components from diesel engines was to be conducted.

This report is an attempt to compare the contribution of exhaust gas pollutants from diesel engines compared to other sources. The effects on health that this contribution has and to evaluate what effects a reduction / increase of the diesel engine contribution will have on human health.

[1] http://fudinfo.trafikverket.se/fudinfoexternwebb/pages/PublikationVisa.aspx?PublikationId=1100

[2] The reference group consisted of members from the main stakeholders in the Swedish oil industry and heavy-duty vehicle industry

2 HEALTH EFFECTS OF DIESEL EXHAUST

2.1 Introduction

In 1989 the International Agency for Research on Cancer (IARC) evaluated diesel engine exhaust as probable carcinogenic to humans, i.e. group 2A, Monograph 46 (1). The overall evaluation was based on the conclusion that there was limited evidence for carcinogenicity in humans of diesel engine exhaust, while there was inadequate evidence for the carcinogenicity in humans of gasoline engine exhaust. In 1998 the California Environmental Protection Agency's Office of Environmental Health Hazard Assessment (OEHHA) completed an assessment of diesel exhaust based on 30 studies of people working around diesel equipment. Based on the information from the OEHHA document the California Air Resources Board (ARB) concluded that the level of diesel particles measured in California in 2000 could cause 540 "excess" cancers, (compared to a situation with no diesel particles in the air) in a population of 1 million people over a 70-year lifetime.

2.2 Air pollution and diesel exhaust

Air pollution can give rise to both short-term and long-term health effects. Although there is an individual difference in sensitivity to air toxicants, young children and elderly people generally suffer more from such exposures. How severe the effects will be is of course dependent on the total exposure, i.e. the duration of the exposure as well as the concentration of toxicants. The short-term effects are for instance bronchitis and pneumonia, but also allergic reactions can occur. The long-term effects most often discussed are lung cancer and heart disease. Of the different components of air pollution the particulate part has caused most concern and has therefore been extensively studied. The particulate matter (PM) has also been a significant part of diesel exhaust although the particles emitted from diesel powered engines has decreased over the years owing to engine and aftertreatment developments as well as introduction of new types of diesel fuels. Since most data are related to health effects of particles and the possible health effects of nanoparticles has become a matter of concern the present report is focused on the particulate fraction and associated polycyclic aromatic hydrocarbons (PAH). A literature review on health risk of exposure to traffic related aerosols with focus on particles and chemical composition has also been published 2007 within the Swedish emission research program, EMFO (2).

2.3 Particulate air pollution

Particulate air pollution is a complex mixture normally containing material from several sources although in urban areas traffic is one of the major sources of particulates. Its impact on human health has been studied with growing concern since the first published results by Pope and colleagues demonstrating an association between fine particulate matter and mortality in six eastern U.S. cities (3-

7). It should be mentioned that the majority of the particles emitted by diesel engines are in the 20-500 nanometer range. The proportion of diesel engines and diesel emission derived particles varies in different regions and over time. For instance, in 1997 it was estimated that 75.7% of trucks, 98 % of busses and 12.1% of passenger cars in Japan were diesel powered. Furthermore, in 1998 it was estimated that 18 % of all motor vehicles were diesel powered and accounted for almost all of the motor vehicle generated particulate emissions (8). In a more recent report from Finland by Jalava and coworkers it has been estimated that the proportion of traffic derived emission of total fine particulate emission is 19 % and that 65% of that is caused by diesel vehicles (9). In Sweden the number of light duty diesel vehicles among new registrations increased from 41% to 51% between 2009 and 2010 (10).

2.4 Diesel particulate exposures and non-carcinogenic effects

In 2002, US Environmental Protection Agency (EPA) published a health assessment document for diesel engine exhaust based on exposure data from engines build before mid-1990s, (11). Although the data are based on older engines it is stated in the report that it is applicable to the engines in use. Both short-term as well as long-term exposures were evaluated. From the data included in the assessment it was concluded that short-term exposures could cause transient irritation and inflammatory symptoms, but that the effects were highly variable in the population. The data also indicated that existing allergies and asthma symptoms could be worsened by this type of exposure.

Recently, Hesterberg and coworkers evaluated over 100 published articles on non-cancer health effects of diesel exhaust, all published after the EPA's Health Assessment Document, the "Diesel HAD" (12) and references therein. The articles covered both human controlled-exposure studies and studies on laboratory animals. The human studies were focused on lung inflammatory responses and functional effects and cardiovascular health effects. Most of the studies employed exposure concentrations in the 100 -300 µg/m^3 range, which is 1-2 orders of magnitude over diesel-exhaust particulate (DEP) concentrations representative of general population DEP exposure. At these elevated exposure levels an inflammatory role of diesel exhaust in humans can be found. It has been assumed that certain subpopulations, such as asthmatics and people with chronic obstructive pulmonary disease (COPD) would be more sensitive to diesel emissions (DE) but according to Hesterberg et al. the few studies of these subgroups did not seem to provide consistent support for the assumption that they are at greater risk (12). However, the question is controversial and final conclusions have to wait further analyses. Both the human exposure studies and laboratory animal experiments evaluated by Hesterberg et al. indicate that short-term inhalation of DE at elevated concentration causes cardiovascular health effects (12). One factor associated with several of these symptoms is oxidative stress, which was also demonstrated on the cellular or molecular level in a number of the articles. However the human studies provided little dose-response information since most studies were only applying one exposure concentration. The few studies on lower exposure concentrations have generally given equivocal results.

In a recent and comprehensive update Brook et al. review the new evidence linking PM exposure with cardiovascular disease (13 and references therein). On the basis of their findings in that study, they conclude that exposure to PM less than 2.5 µm in diameter (PM2.5) over a few weeks can cause cardiovascular disease-related death and nonfatal events. Furthermore, longer-term exposure increases the risk for cardiovascular mortality even more and reduces life expectancy, at least for the more highly exposed part of the population with several months to a few years. Accordingly, reductions in PM levels reduce cardiovascular mortality within a few years. The authors conclude that the overall evidence is consistence with a causal relationship between PM2.5 exposure and cardiovascular morbidity and mortality (13). A further conclusion drawn by the authors is that the PM2.5 concentration-cardiovascular risk relationships for both long- and short–term exposures appear to extend below 15µg/m^3 (the 2006 annual National Ambient Air Quality Standards ,NAAQS, level) in a linear fashion, i.e. without a safe threshold. The data from most recent studies reviewed in this update by Brook and colleagues also support the conclusion that the overall risk for mortality as a result of exposure (both short- and long-term) to PM is greater for cardiovascular than pulmonary diseases.

2.5 Diesel exhaust and carcinogenic effects

A large amount of studies on the carcinogenic potential of diesel exhaust have been published, for recent reviews see (14,15) and references therein. Epidemiologic studies have shown small increases in lung cancer incidence resulting in relative risks (RRs) of or below 1.5. If the RR is 1.5 or close to that it means that the probability to attract lung cancer is increased up to 50% compared to the non-exposed control population, which would indicate a probable association between DE and lung cancer. However, in both these reviews the same type of limitations in the epidemiologic studies is discussed. One problem, especially with the early studies is the gradual introduction and increase in diesel powered vehicles making it difficult to assess the exposure level. In many of the studies the resulting latency period has been too short as well. These circumstances will result in different exposure doses for the individuals in the cohort, although normally one exposure level is assumed in the study. The outcome of that can result in a lower RR but also make it difficult to establish an exposure-response relationship. In the animal tests only the high exposure dose normally shows an effect, again making it difficult to establish a dose-response relationship. Another argument used in these two reviews is that most of the data are based on exposure to traditional diesel exhaust, i.e. studies published prior to 1988 or from the transition diesel exhaust period (1988-2006) and what is missing is studies based on new-technology diesel exhaust (NTDE).

In a recent article by Olsson et al. (16) the association between occupational diesel motor exhaust exposure and lung cancer was analyzed. The study was based on pooled information from Europe and Canada comprising 13304 cases and 16282 controls. Their results showed a consistent association between occupational exposure to diesel exhaust and increased risk of lung cancer. The authors also found a significant exposure-response relationship and concluded that it was unlikely that

the association could be explained by bias or confounding since these issues were addressed by adjusting the models and subgroup analyses.

In United States a large collaborative research program has started coordinated by Coordinating Research Council (CRC) (17). One major part of that study is a chronic inhalation toxicity study applying multiple exposure levels of NTDE and two species of animals. The inhalation study will use the National Toxicology Program protocol. No data from the animal tests are yet available.

2.6 Polycyclic aromatic hydrocarbons (PAH), diesel particulate extracts and genotoxic effects

In the original 1989 IARC evaluation (1) of diesel engine exhaust it was stated that there was sufficient evidence for the carcinogenicity in experimental animals of particulate extract from diesel exhaust

Particulate extracts were similarly analyzed in a number of mutagenicity tests in the 1980s. In addition to particulate extracts, volatile or semi-volatile components, collected by condensation or absorption after dilution and filtration of the particles, were also applied to different organisms. The majority of tests were based on different types of genetic effects such as, DNA damage or gene mutations in bacteria or mammalian cells in vitro. Sister chromatid exchanges were also induced by diesel particulate extracts in animal and human cells in vitro. When these early test were performed the motor vehicle exhausts contained a larger amount of particulate matter and also a higher concentration of genotoxic substances as can be seen when comparing the bacterial mutagenicity and chemical analyses of polycyclic aromatic hydrocarbons (PAHs) from the early period (18) with a more recent report (19). It can therefore be concluded that diesel exhaust contain mutagenic and carcinogenic compounds and PAHs are contributing to these health effects.

Chemically PAHs consist of fused aromatic rings, the simplest being naphthalene with only two fused benzene rings. One of the heavier PAH in vehicle exhaust particulate matter (PM) is coronene comprising six peri-fused benzene rings. PAHs are generally produced in incomplete combustion processes, and their occurrence and emissions have therefore been substantial during the past centuries. IARC has evaluated several situations in which exposure to PAHs occur and the overall evaluation is that PAH-containing material is carcinogenic to humans (20-24). IARC has also evaluated the carcinogenicity of several individual PAHs (20,24,25). These evaluations are based on animal data since no human data exist for individual PAHs. Cyclopenta[c,d]pyrene, dibenz[a,h]anthracene are two PAHs found in diesel exhaust that are classified as probably carcinogenic to humans (2A) by IARC, while benzo[a]pyrene (B[a]P), which is the main indicator of carcinogenic PAHs recently was classified as a group 1 by IARC (24).

The Carcinogenesis is a multistep, process involving genotoxic events (mutations), altered gene expression at the transcriptional, translational, and posttranslational levels (epigenetic events), and altered cell survival (proliferation and apoptosis). Thus

the cancer process may therefore be divided in three main steps, initiation, promotion and progression. Many PAHs are considered to be complete carcinogens, i.e. can act on all these steps. The first initiation step involves a mutation and the substance is thus mutagenic or genotoxic. The promotion step in carcinogenesis is an epigenetic step that alters the gene expression profile, reprogramming the cell to proliferate. In the last step, the progression, further changes occur in the cells leading to an increased genomic instability and a further development toward malignancy.

Several hundred PAHs have been detected in air although generally only few PAHs are analyzed, as indicators of PAHs or more often just B[a]P is analyzed as representative for the whole PAH group (cf. above). In Stockholm the sum of 14 different PAHs has been reported to be between 100 and 200 ng/m^3 at the street level (26 and references therein). The corresponding concentration for B[a]P was 1-2 ng/m^3. Vehicle exhaust is the largest contributor to PAH emissions in central parts of large cities. However there are several factors affecting the content in vehicle exhaust, e.g. fuel, driving conditions and exhaust aftertreatment device (see (19) and references therein).

2.7 Comparing the emission of PAHs and the genotoxic effects of two classified diesel fuels

In a report by Westerholm and colleagues the exhaust emissions from two diesel fuels were analyzed both chemically and biologically with focus on cancer risk (19). One of the fuels was a Swedish environmental classified diesel fuel (Mk1). It is known that the total PAH emission from Mk1 is at least a factor of 5 lower compared to a non-environmentally classified fuel, e.g.Mk3 – EN 590, see (26). The emissions from Mk1 were compared to the emissions from one references fuel used in the “European Program on Emissions Fuels and Engine Technologies” (EPEFE) program. A Volvo FH12 truck with a D12A 420 diesel engine (Euro II) equipped with a turbo, intercooler and electronic fuel-injection system generated the exhaust emissions that were analyzed. Extracts of the particulate phase and the semi-volatile phase were analyzed chemically for the quantification of polycyclic aromatic compounds. Fourteen different PAHs were quantified in the two types of extracts. The same extracts were also tested for genotoxicity in Ames Salmonella mutagenicity assay and for aryl hydrocarbon receptor activity. The results showed that the particulate and semi-volatile associated PAH level in the Mk1 exhaust was 11% and 23%, respectively, of the EPEFE level. Mk1 exhausts also showed a 75-90 % reduction of the particulate mutagenicity compared to EPEFE fuel. Also the semi-volatile phase showed a reduced mutagenicity of between 40 and 60%. Similarly, the aryl hydrocarbon receptor binding activity was also much lower for Mk1.

From these results it could be concluded that the level of genotoxic effects and bioactivity followed the PAH emissions, which strongly indicate that the health effects would be much less using MK1 compared to the EPEFE fuel. It should be noted that of the three IARC class 2A PAHs mentioned above the difference was negligible for dibenz[a,h]anthracene and B[a]P, while for benz[a]anthracene the emissions from the

EPEFE fuel was 40 times higher. This comparison was made using a diesel engine that was representative for heavy duty diesel vehicles fulfilling the Euro II requirements. A heavy duty diesel complying with Euro V would produce lower emissions and would hypothetically give lower health effects, such as e.g. genotoxicity. However, comparable studies with diesel engines meeting the Euro V exhaust requirements have not been done and such data are thus missing.

2.8 Impact on health – Variable exposure levels

As a way to estimate possible health effects from a change of fuel quality a hypothetic question is asked. “What impact on health effects would a decrease to 50% or a doubling of diesel exhaust exposure levels have?” The following chapter is an attempt to answer that question.

Even though there is a risk for mortality in cardiovascular disease as a result of exposure (both short- and long-term) to particulate matter this type of effects may be more associated with particles as such, rather than diesel particles specifically. Carcinogenicity, on the other hand, may be regarded as a health effect more clearly associated with diesel exhaust and PAHs.

Total PAH levels of ambient air from different measurements are difficult to compare, since different individual PAHs have been analyzed. In Europe, B[a]P concentrations are often below 1 ng/m^3 at background stations, whereas at locations close to traffic, concentrations range between 1 and 5 ng/m^3. At the street-level site in the center of Stockholm (Hornsgatan), Sweden, the sum of 14 PAHs ranged from 100 to 200 ng/m^3. The B[a]P levels varied between 1 and 2 ng/m^3 (26). According to the same report data from Sweden indicated that the emissions of PAHs were reduced by 35% between 1980 and 1987 and by 15% between 1987 and 1995.

In reference 32, chapter 5.2, the reduction of PAH emissions in the 1980s is explained by the introduction of particulate regulations in the industrial sector and that during the same time there was a massive move away from fossil fuel usage in industrial applications.

Our opinion is that the same factors also contributes to the PAH reduction during the years 1987 to 1995, but that a considerable part of the reduction also can be contributed to the introduction of catalytic converters on gasoline cars (1989 onwards) and the introduction of Mk1 diesel quality in Sweden (introduced in 1990, and market dominant from 1993)

To simplify the following discussion a few assumptions are made. It might be assumed that the general PAH levels are somewhat lower today, with lower PAH levels in the cities and assuming a genotoxic mechanism for a carcinogenic risk a linear dose-response relationship for a risk increment is applicable. Also in the situation in which a low level of exposure to a genotoxic mixture occurs simultaneous with an exposure to a promoter above its no-effect threshold linearity in the dose-response relationship would be expected.

Thus assuming that carcinogenicity is the critical health effect and PAH in diesel exhaust is of major concern, at least in urban areas, the following development could be expected. Based on a similar discussion as given in (19) a doubling of the diesel exhaust would then roughly give rise to a risk increment of the same order of magnitude under the same circumstances. This means that only a quantitative change in PAH emissions would takes place without any changes in PAH profile of the exhaust, neither in the particulate fraction nor in the semi-volatile phase. Also no changes in particle size distribution should occur that would change the bioavailability of PAHs. It must be emphasized that any fuel or engine developments as well as developments of new aftertreatment equipment that would alter these parameters would make a new risk evaluation necessary.

As mentioned above, Jalava and coworkers reported that in Finland the proportion of traffic derived emission of total fine particulate emission is 19 % and that 65% of that is caused by diesel vehicles (9). Assuming similar percentages in Sweden, a rough estimate of the number of annually cancer cases caused by a doubling of diesel exhaust emissions might be indicative. If air pollution is causing 200-300 cancer cases annually in Sweden (27), between 25 and 60 cases would be associated with diesel particulate emission and a doubling would than increase the number of cancer cases to 50 - 120. It should be kept in mind that this estimate is tentative and based on several assumptions that may not be applicable in a real situation.

3 CONCLUSION

In 2002, US EPA published a health assessment document for diesel engine exhaust. From the data it was concluded that short-term exposures could cause transient irritation and inflammatory symptoms, but that the effects were highly variable in the population. Furthermore, both the human exposure studies and laboratory animal experiments indicate that short-term inhalation of diesel exhaust at least at elevated concentration cause cardiovascular health effects. It should be mentioned that particles by themselves seem to evoke this type of effects, which make it more difficult to evaluate the contribution by diesel particles in a common exposure situation.

In 1989 IARC evaluated diesel engine exhaust and classified it in group 2A, that is as probable carcinogenic to humans. This evaluation was mainly based on carcinogenicity in experimental animals. Several different epidemiological analyses have also been carried out, regarding carcinogenic as well as non-carcinogenic health effects. The particulate matter has been a significant part of diesel exhaust and most analyses have therefore been focusing on particles and particle associated PAH. Most of them have shown a small increase in lung cancer incidence. However, some limitations in the studies, such as too short latency periods and uncertain exposures levels, have made it problematic to unequivocally ascribe the effects to diesel exhaust.

Even though there is a risk for mortality in cardiovascular disease as a result of exposure (both short- and long-term) to particulate matter this type of effects may be more associated with particles as such, rather than diesel particles specifically. Carcinogenicity, on the other hand, may be regarded as a health effect more clearly associated with diesel exhaust and PAHs.

Numerous genotoxicity tests have been carried out on particulate extracts from diesel exhaust, but also volatile and semi-volatile components have been tested. Most of these tests have employed a combination of chemical analyses and mutagenicity tests. It can therefore be concluded that diesel exhaust contain mutagenic and carcinogenic compounds and PAHs are contributing to these health effects.

Exhaust emissions from different diesel fuels have also been analyzed both chemically and biologically with focus on cancer risk. In one Swedish study the emissions from a Swedish environmental classified diesel fuel (Mk1) were compared to one references fuel used in the “European Program on Emissions Fuels and Engine Technologies” (EPEFE) program. From these results it could be concluded that the level of genotoxic effects and bioactivity followed the PAH emissions, which strongly indicated that the health effects would be much less using Mk1 compared to the EPEFE. Though it should be mentioned that this comparison was made using a diesel engine that was representative for heavy duty diesel vehicles fulfilling the Euro II requirements. A heavy duty diesel complying with Euro V would produce lower emissions and would hypothetically give lower health effects, such as e.g. genotoxicity. However, comparable studies with diesel engines meeting the Euro V exhaust requirements have not been done and such data are thus missing.

4 RECOMMENDATIONS

In this chapter we compile information from chapters 2 and 3 together with conclusions from the report “COMPARING EXHAUST EMISSIONS FROM HEAVY DUTY DIESEL ENGINES USING EN 590 VS. MK1 DIESEL FUEL“ (25). Particulates are known to have both non-carcinogenic and carcinogenic effects. PAH´s have also been shown to have carcinogenic effects. Nitrogen oxides have likewise been discussed as a health issue. All three of those components can be found in diesel engine emissions and, furthermore, the emission levels of those components have been shown to be affected by the fuel quality.

Carcinogenic effects seem to be without “threshold levels” meaning that there is no completely “safe limit” to be set on air quality regarding particulate matters and PAHs.

It has been concluded in (25) that emission data support emission benefit from using Swedish Environmentally Classified diesel fuel (according to SS 15 54 35 – Mk1), compared to European standard diesel according to EN 590, when used in heavy duty engines up to emission class Euro III. On heavy duty diesel engines meeting more stringent emission legislations there are indications on, but not enough data to prove, emission benefits with Mk1 diesel fuel.

To be able to determine the current health benefits and, in the future, possible / reduced / non existing, health benefits from continuing use of Mk1 diesel fuel compared to European standard diesel additional emission data needs to be gathered from modern diesel engines.
Our recommendation is to gather more data through emission tests within the following areas:

- Comprehensive test programs have to be developed for NRMM engines meeting Stage IIIA and IIIB and later emissions requirements.
- Comprehensive test programs have to be developed for engines meeting Euro V and later emission requirements, making sure to cover differences in aftertreatment equipment
- Added test programs for series produced engines meeting Euro IV emission requirements
- Emission data covering PAH, particle number and bio reactivity on emission classes Euro IV and V and Stage IIIA and IIIB are needed to understand how those emissions from the Swedish heavy duty vehicle/engine fleet will evolve as fleets are renewed.

5 ABBREVIATIONS

ARB	California Air Resources Board
B[a]P	benzo[a]pyrene
CO	Carbon monoxide
COPD	chronic obstructive pulmonary disease
CRC	Coordinating Research Council
DE	diesel exhaust
DEP	diesel-exhaust particulate
EMFO	Swedish emission research program
EPA	US Environmental Protection Agency
EPEFE	European Program on Emissions Fuels and Engine Technologies
HAD	Health Assessment Document
IARC	International Agency for Research on Cancer
IVL	Swedish Environmental Research Institute
NAAQS	National Ambient Air Quality Standards
NOx	Nitrogen oxides
NTDE	new-technology diesel exhaust
OEHHA	Office of Environmental Health Hazard Assessment
PAH	polycyclic aromatic hydrocarbons
PM	Particulate Matter
PN	Particle Number
RR	relative risk
STA	Swedish Transport Administration
THC	Total Hydrocarbon

6 REFERENCES

1. IARC. (1989) *IARC Monogr Eval Carcinog Risks Hum* **46**, 1-458
2. Sehlstedt, M., Forsberg, B., Westerholm, R., Boman, C., Cabecadas, J., and Sandström, T. (2007) *EMFO report*, 1-85
3. Dockery, D. W., Pope, C. A., 3rd, Xu, X., Spengler, J. D., Ware, J. H., Fay, M. E., Ferris, B. G., Jr., and Speizer, F. E. (1993) *N Engl J Med* **329**, 1753-1759
4. Pope, C. A., 3rd, Burnett, R. T., Thurston, G. D., Thun, M. J., Calle, E. E., Krewski, D., and Godleski, J. J. (2004) *Circulation* **109**, 71-77
5. Hoek, G., Brunekreef, B., Goldbohm, S., Fischer, P., and van den Brandt, P. A. (2002) *Lancet* **360**, 1203
6. Schwartz, J., Laden, F., and Zanobetti, A. (2002) *Environmental Health Perspectives* **110**, 1025
7. Laden, F., Neas, L. M., Dockery, D. W., and Schwartz, J. (2000) *Environ Health Perspect* **108**, 941-947
8. Kagawa, J. (2002) *Toxicology* **181-182**, 349-353
9. Jalava, P. I., Tapanainen, M., Kuuspalo, K., Markkanen, A., Hakulinen, P., Happo, M. S., Pennanen, A. S., Ihalainen, M., Yli-Pirila, P., Makkonen, U., Teinila, K., Maki-Paakkanen, J., Salonen, R. O., Jokiniemi, J., and Hirvonen, M. R. (2010) *Inhal Toxicol* **22 Suppl 2**, 48-58
10. Trafikverket. (2010), 55
11. National Center for Environmental Assessment (Washington D.C.). (2002) Health assessment document for diesel engine exhaust. National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency, Washington, DC
12. Hesterberg, T. W., Long, C. M., Bunn, W. B., Sax, S. N., Lapin, C. A., and Valberg, P. A. (2009) *Critical Reviews in Toxicology* **39**, 195-227
13. Brook, R. D., Rajagopalan, S., Pope, C. A., 3rd, Brook, J. R., Bhatnagar, A., Diez-Roux, A. V., Holguin, F., Hong, Y., Luepker, R. V., Mittleman, M. A., Peters, A., Siscovick, D., Smith, S. C., Jr., Whitsel, L., and Kaufman, J. D. (2010) *Circulation* **121**, 2331-2378
14. Gamble, J. (2010) *Crit Rev Toxicol* **40**, 189-244
15. Hesterberg, T. W., Bunn, W. B., 3rd, Chase, G. R., Valberg, P. A., Slavin, T. J., Lapin, C. A., and Hart, G. A. (2006) *Crit Rev Toxicol* **36**, 727-776
16. Olsson, A. C., Gustavsson, P., Kromhout, H., Peters, S., Vermeulen, R., Bruske, I., Pesch, B., Siemiatycki, J., Pintos, J., Bruning, T., Cassidy, A., Wichmann, H. E., Consonni, D., Landi, M. T., Caporaso, N., Plato, N., Merletti, F., Mirabelli, D., Richiardi, L., Jockel, K. H., Ahrens, W., Pohlabeln, H., Lissowska, J., Szeszenia-Dabrowska, N., Zaridze, D., Stucker, I., Benhamou, S., Bencko, V., Foretova, L., Janout, V., Rudnai, P., Fabianova, E., Dumitru, R. S., Gross, I. M., Kendzia, B., Forastiere, F., Bueno-de-Mesquita, B., Brennan, P., Boffetta, P., and Straif, K. (2011) *Am J Respir Crit Care Med* **183**, 941-948

17. Khalek, I. A., Bougher, T. L., and Merritt, P. M. (2009)
18. Rannug, U. (1983) *Environ Health Perspect* **47**, 161-169
19. Westerholm, R., Christensen, A., Tornqvist, M., Ehrenberg, L., Rannug, U., Sjogren, M., Rafter, J., Soontjens, C., Almen, J., and Gragg, K. (2001) *Environ Sci Technol* **35**, 1748-1754
20. IARC. (1983) *IARC Monogr Eval Carcinog Risk Chem Hum* **32**, 1-453
21. IARC. (1984) *IARC Monogr Eval Carcinog Risk Chem Hum* **34:2**, 1-222
22. IARC. (1984) *IARC Monogr Eval Carcinog Risk Chem Hum* **34:3**, 1-219
23. IARC. (1985) *IARC Monogr Eval Carcinog Risk Chem Hum* **35**, 1-247
24. IARC. (2010) *IARC Monogr Eval Carcinog Risk Chem Hum* **92**, 1-868
25. IARC. (1987) *IARC Monogr Eval Carcinog Risk Chem Hum* **Supplement No. 7**, 1-440
26. Bostrom, C. E., Gerde, P., Hanberg, A., Jernstrom, B., Johansson, C., Kyrklund, T., Rannug, A., Tornqvist, M., Victorin, K., and Westerholm, R. (2002) *Environ Health Perspect* **110 Suppl 3**, 451-488
27. Nordling, E., and Bellander, T. (2009) Luftföroreningar utomhus. in *Socialstyrelsen-Miljöhälsorapport*, Stockholm
28. AVL MTC - 0015. (2010) Comparing Exhaust Emissions From Heavy Duty Diesel Engines using EN 590 vs. MK1 Diesel Fuel.
29. Sjöberg K, et al. (2004) Air quality in urban areas (Luftkvalitet i tätorter) IVL report B1553, Swedish Environmental Research Institute
30. Sjöberg K, et al. (2006) Air quality in urban areas 2005 (Luftkvalitet i tätorter 2005) IVL report B1667, Swedish Environmental Research Institute
31. Sjöberg K, et al. Luftkvalitet i Sverige 2002 – 2010, Results from the URBAN-measurement-net; IVL reports B1535, B1593, B1636, B1690, B1744, B1801, B1868 and B1940, Swedish Environmental Research Institute
32. Förslag till miljökvalitetsnormer för arsenik, kadmium, nickel och bens(a)pyren, Konsekvensanalys för införande i svensk lagstiftning av direktivet 2004/107/EG, Naturvårdsverket, ISBN 91-620-, ISSN 0282-7298