health aspects of worker exposure to oil mists

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1. INTRODUCTION

The current Threshold Limit Value (TLV) for worker exposure to oil mist recommended by the American Conference of Governmental Industrial Hygienists (ACGIH) is an 8-hour time-weighted average (TWA) level of 5 mg/m^3 (1). This figure has also been adopted by many countries in national lists of permissible exposure standards. The documentation supporting the ACGIH TLV (2) comments "The 5 mg/m^3 limit would appear to provide a considerable margin of safety against even relatively minor changes in the lungs, especially for the types of cutting and lubricating oils most frequently encountered, and where there has been no substantial altering of composition by heat and/or oxidation". It continues with the proviso: - "Certain oils, however, may contain toxic and even carcinogenic components either natural or added, and in such cases there may be little or no safety factor in the 5 mg/m^3 TLV".

Despite this warning it has become quite common practice to apply the 5 mg/m^3 TLV to all varieties of oil mist without regard to the make up of the oil from which the mist is generated. Therefore, as part of its continuing reviews of the health aspects of petroleum products, CONCAWE has considered it appropriate to assess, from available published information, the broad classes of oils for which the ACGIH oil mist TLV is applicable. The oil mists considered are those aerosols derived from the manufacture or use, excluding combustion, of petroleum-based products.

2. MANUFACTURE AND COMPOSITION OF OIL PRODUCTS

2.1 OILS USED FOR APPLICATIONS ASSOCIATED WITH MIST GENERATION

Oil mists can arise in a variety of mineral oil applications. Table 1 lists the principal applications associated with potential generation of oil mists together with an indication of the changes which have been made in the types of base oils used in the last 10 to 20 years. It should be noted that oils used in the applications listed in Table 1 may be formulated with additives of various types and at various levels (see Section 2.3). The trend to use more severely refined base oils has resulted partly from the need to provide improved performance characteristics and increased lubricant life, and partly from recognition that lightly refined mineral base oils may present a carcinogenic risk in applications where a high degree of skin contact may be difficult to avoid. Those mineral base oils used in metal working applications are good examples of this trend. However, technical aspects require the continued use of less severely refined base oils for some applications.

2.2 MANUFACTURE AND COMPOSITION OF MINERAL BASE OILS

The following is a brief outline of the refining of mineral base oils and the effects which different refining processes have on composition and characteristics. More detailed information can be found in Chapter 1 (Chemical and Physical Data) and Chapter 2 (Production, Use, Occurrence and Analysis) of the IARC Monograph on Mineral Oils (3) and in the CONCAWE report on "Health Aspects of Lubricants" (4).

Mineral base oils are prepared from naturally occurring crude petroleum oils. The crude oil is distilled, normally at atmospheric pressure, and then under high vacuum, to give vacuum distillates and residual fractions which can be further refined to mineral base oils. Historically, the additional refining processes included treatment with sulphuric acid and special earths, but these have largely been superseded by selective solvent extraction (to reduce aromatic compounds, waxes or asphaltic materials) and, more recently, by hydrogenation processes (hydrotreatment).

The chemical composition of the mineral base oil produced depends both on the original crude and on the processes used during refining. Crude oils are usually described as naphthenic or paraffinic depending on the preponderance of hydrocarbon types present. With the simpler refining techniques the composition of the finished base oil reflects that of the crude. After severe refining, variations due to crude are less apparent.

Table 1: Oils for applications associated with mist generation

Application	Oils used	
	Historical	Current
Metal Working	Acid-treated, Vacuum distillates. Some coal and shale oils.	Mildly refined. Severely refined. Synthetics.
Textile machinery	Vacuum distillates, acid treated coal and shale oils, moving to technical white oils.	Mildly or severely refined.
Rock drills	Acid-treated. Mildly refined.	Severely refined.
Mist lubrication	Mildly or severly refined.	Severely refined.
Agricultural sprays	Mildly or severly refined.	Mild or severely refined.
Concrete moulds	Acid-treated, vacuum distillates, middle distillates, mildly refined, re-refined.	Acid-treated vacuum distillates, middle distillates mildly refined, re-refined.
Corrosion preventives	Mildly refined, cut-back bitumen, hydrocarbon solvents.	Greater use of severely refined oils.
Printing inks	Aromatic extracts.	Aromatic extracts, Vacuum distillates, Severely refined.
Rubber extenders	Aromatic extracts.	Aromatic extracts and other oils.

Finished base oils are predominantly hydrocarbons but also contain some sulphur, oxygen and nitrogen compounds and traces of a number of metals. The hydrocarbons are normally a complex mixture of aromatics, naphthenes and paraffins. The proportions of the different species are responsible for the different characteristics of the base oils. The higher the molecular weights, the more viscous are the oils.

Oils containing predominantly aromatic and naphthenic hydrocarbons are normally termed Low Viscosity Index (LVI) implying a comparatively rapid change of viscosity with temperature. Oils predominantly paraffinic in character have High Viscosity Indices (HVI) and change viscosity less rapidly with temperature. Medium Viscosity Index (MVI) oils are intermediate in hydrocarbon type and may be obtained from either naphthenic or paraffinic crudes. In addition to the effect on viscosity index, hydrocarbon type differences also alter other physical and chemical properties of base oils. For example, LVI oils tend to be better solvents for some additives and for waste or degradation products whilst HVI oils are often more chemically stable during use.

All crude oils contain some polycyclic aromatic hydrocarbons compounds (PAH) some of which (mainly 4 to 6 condensed-ring compounds) are known to be carcinogenic. Although the contents vary with crude sources, the proportions and types of such compounds in finished base oils are mainly determined by the refining processes. Mild processing, such as acid/earth, reduces the total aromatics content slightly but does not significantly reduce the amount of PAH. Mild hydrotreatment reduces the PAH content but has little effect on the total aromatic content. Severe solvent refining (extraction) or hydrotreatment reduces substantially both the PAH and the total aromatic content. Sufficiently severe treatment with fuming sulphuric acid (oleum) can remove aromatics including PAH almost entirely, for example to produce white oils of medicinal quality. A similar effect can also be achieved by deep extraction and hydrofinishing.

Aromatic extracts, obtained as by-products in the solvent refining of mineral base oils, have relatively high contents of total aromatics and PAHs. Their good solvent characteristics have been found valuable in special applications such as rubber extenders and printing inks (although in the latter case there has been a trend to replacement by well-refined mineral base oils).

Vacuum distillates which have not been further refined have also found some use in formulated products. As indicated above, their chemical composition will reflect that of the original crude. They are likely to contain more aromatics and PAHs then well-refined mineral oils. As shown in Table 1, vacuum distillates have been replaced in most of the products for applications associated with oil mist by more highly refined base oils.

2.3 COMPOSITION OF FORMULATED LUBRICANTS

A wide range of additives is used at concentrations ranging from a few parts per million to about 20 per cent to modify the physical and/or chemical characteristics of mineral base oils in order to provide the performance requirements of specific applications.

Additives are usually proprietary materials and composition details will therefore vary between individual suppliers. The general principles of additive functions and the groups of compounds used have been previously described by IARC (3) and CONCAWE (4).

An important group of formulated products is that known as soluble or emulsifiable oils consisting of mineral base oils plus significant amounts of emulsifying agents and other additives. In use, these are added to water to form emulsions which contain typically 1 to 10 per cent of the neat soluble oil.

3. INDUSTRIAL HYGIENE ASPECTS

As shown in Table 1 exposure to oil mist and vapour may occur in a wide variety of situations. Consideration of the physical form, composition and concentration is required if exposure is to be assessed and controlled.

3.1 GENERATION

Application of energy to an oil may result in the generation of liquid droplets dispersed in air, namely oil mist. The physico-chemical properties and concentrations of such mist are related to the properties and conditions of use of the oil concerned. The term "fume" generally refers to airborne solid particulate resulting from condensation of vapour or from gaseous chemical reactions and hence should not be used to describe aerosols of liquids such as oil.

In practice, oil mists may be generated by several routes, such as aeration, contact with a fast moving surface, or by heating. The particle size is likely to differ for each of these processes. Thermally derived oil mists are produced by condensation subsequent to the complete or partial vaporisation of the oil and the particle size is typically very small and relatively monodisperse in distribution terms. Mist generated by mechanical means may vary widely in terms of particle size and distribution depending upon the specific conditions at source.

The composition of oil mist may differ from that of the original oil, for example, because of preferential vaporisation of lower boiling components (12) or because of thermal degradation reactions (19). A mixture of both oil mist and vapour may be formed from products containing lower boiling components. In addition oil droplets formed from such products may be unstable as further vaporisation occurs (12). These possible variations in composition of oil mists must be considered in assessing both exposure measurements and health risks.

3.2 PARTICLE SIZE

Once an oil mist has been generated, particles may be inhaled. The location and likelihood of deposition of oil mist in the respiratory tract depends primarily on the particle size. In general terms, particles above around 50 microns in diameter are unlikely to remain airborne sufficiently long to be inhaled. This is in contrast to particles of below about 5 microns in diameter which are likely to remain airborne for relatively long periods. As a further generalisation, particles above 5 microns will be largely deposited in the upper respiratory tract and nasal passages whereas the majority of smaller ones may penetrate to the deeper recesses of the lungs.

Once deposited, oil particles may be removed by clearance mechanisms or absorbed into body fluids, and therefore are capable of causing both local and systemic toxic effects.

Relatively little information has been published in regard to either the particle size or concentrations of oil mists occurring in industry. Particle sizes during steel rolling (5) are reported as 70% in the range 0.8-1.0 microns and in hot forging (6) 40-60% below 10 microns diameter. In metal working machine shops, where oil mist may be generated both thermally and mechanically during grinding, turning, drilling etc., particle sizes have been reported in the range 2.4-5.6 microns (7) and around 2 microns (8). With newspaper printing inks containing high concentrations of mineral oil, mechanically generated oil mist has been reported to have diameters of 14 microns (9) and 15 microns (10). A study of eight press rooms (11) reported that 4-57% of the ink mist particles were below 11.4 microns in diameter.

3.3 VOLATILITY

The base oils used in the manufacture of lubricants typically contain hydrocarbons with more than 20 carbon atoms and have final boiling points in the range 350 -550°C. At room temperature these oils have a very low volatility; any mist formed is relatively stable and not normally associated with significant levels of hydrocarbon vapour. Even when the oil mist is thermally generated there is unlikely to be any major increase in the level of oil vapour associated with the aerosol.

However, there are some products which contain more volatile components such as kerosines. Products of this type include some metal working oils (rolling oils and grinding fluids) and corrosion preventives. Oil mist generated from such products may be accompanied by the presence of significant quantities of oil vapour. The mist is also likely to have a relatively short lifetime (12) as the micron size particles will tend to evaporate quite rapidly. This instability has implications for the sampling and analytical methods as well as for exposure evaluation and the subsequent health risk assessment. Many published studies have failed to take these factors into consideration with the result that oil mist exposures have been overestimated where both mist and vapour have been collected and underestimated in cases where droplets have vaporised during the sampling procedure (6, 13, 14).

3.4 EXPOSURE MEASUREMENT

As has already been indicated, the likely physical form of the airborne contamination must first be considered before a sampling technique is selected. It is essential to distinguish between particulate and vapour and to take account of the instability of airborne particles from lighter products as well as the possibility of vaporisation from filters during sampling. In view of the possible local effects of oil mist on the upper respiratory system, including the nasal passages, it is necessary to measure the total inhalable particulate in all cases.

CONCAWE Report No. 1/81, "Guidelines for the Determination of atmospheric concentrations of oil mist" (15), based on an earlier method published by the UK Institute of Petroleum (16), provides an adequate discussion of exposure measurement recommendations. Similar methodologies have been described by NIOSH (17).

Emulsifiable or soluble oils also present a specific problem which is worth highlighting. Such oils are very commonly encountered in metal working operations. In-use, emulsions consist primarily of water with less than 10% of mineral oils and additives. Use of the IP/CONCAWE standard methodology for mists from such oils presents assessment difficulties. Prior to weighing the filters, the aqueous component will have evaporated and the remaining material will thus be mineral oil and non-volatile additives in addition to metal particles, etc. Therefore, results obtained from sampling mists of emulsions may not reflect the actual mist and vapour complex to which the workers were exposed and comparison with the ACGIH TLV for oil mist of 5 mg/m^3 may not be appropriate. The high additive contents of some soluble oils may also make this limit inappropriate.

3.5 CHEMICAL ADDITIVES

A variety of types of additives may be incorporated at various concentrations into oils used in applications associated with mist generation. For many additives, little actual data on potential respiratory system effects will be available. However, informed judgements may be feasible from a knowledge of the concentrations present, the chemical nature, practical experience and other toxicological data such as skin or eye irritancy. The potential effects of additives should be borne in mind when measuring and assessing mist exposures.

3.6 POLYCYCLIC AROMATIC HYDROCARBONS

When organic materials are heated or burned, small quantities of a wide range of polycyclic aromatic hydrocarbons (PAH) may be formed (18). Increases in the PAH content of used oils were reported (19) to be usually less than 10-fold for cutting oil sump and mist samples but up to 100-fold for quench oils. Certain mineral oil fractions, particularly those containing relatively high levels of 4-6 ring PAHs, have been shown to be skin carcinogens. It is therefore reasonable to question the applicability of a 5 mg/m^3 exposure limit for oil mists which may contain significant levels of PAHs. This will also be the case where the fresh oil contains relatively high PAH levels, for example, distillate aromatic extract oils.

3.7 INDUSTRIAL HYGIENE ASPECTS OF EPIDEMIOLOGICAL STUDIES

Most epidemiological studies have been conducted retrospectively and consequently the exposure levels have almost invariably been evaluated after the study period. Thus, the data may not correctly represent past exposures. In studies where past exposure data are available, the type of sampling and analysis used and the nature of the oils involved also need careful scrutiny in view of the assessment difficulties described under Sections 3.3-3.5.

3.8 INDUSTRIAL HYGIENE CONCLUSIONS

Oil mist may exist in a wide range of particle sizes, compositions and concentrations in industry, depending on the nature of the oil, the process involved and control measures employed.

In the case of mist originating from lighter products, there is likely to be oil vapour associated with the particulate. In addition, mist particles from such products may also have a fairly short lifetime, evaporating in seconds.

With mists from emulsions, the water content evaporates rapidly. With standard particulate sampling techniques, only the mineral base oil and non-volatile additives are collected and these may not be representative of actual exposures. In addition, the high amounts of additives often present in soluble oils further complicate the assessment of exposures.

Oils subject to very heavy duty and a high thermal insult, may generate potentially carcinogenic PAHs and these may in turn be present in any oil mist. Other oils may, as supplied, contain relatively high levels of such PAHs and mists from such products may be regarded as presenting a different type and level of risk to the majority of mineral oil products in general use.

Exposure data associated with published epidemiological studies are sparse, often are of dubious quality, or have been collected after the study period and hence may not reflect historical exposures. Improved characterisation of exposures is essential for future investigations.

The combination of difficulties presented by considerations of particle size, volatility, additives and the possible presence of certain PAHs, suggests that one single exposure limit for all mineral oil mists may not be appropriate.

4. ANIMAL TOXICITY STUDIES OF MINERAL OIL MISTS

Animal toxicity studies on mineral oil mists have involved exposure of mice, rats, guinea pigs, rabbits, hamsters, dogs and rhesus monkeys for periods varying from one hour up to as long as two years.

4.1 ACUTE TOXICITY STUDIES ON MINERAL OIL MISTS

In short-term experiments carried out by Shoshkes et al (20), male and female mice were exposed for 2 hours to liquid petrolatum and motor oil and also to edible oils. Groups of six mice were exposed to very high concentrations (4500 mg/m^3 and 4300 mg/m^3 for liquid petrolatum and SAE No. 10 respectively). The average droplet size of the oil mists was 2.5 microns, and approximately 80% of the particles retained in all areas of the lungs were 2.5 microns, or less in diameter. Phagocytosis of the droplets was completed within 48 hours. The initial concentration of retained oil droplets was similar for all types of oil mists. During a 90 hours follow-up period, however, the lung concentrations of edible oil droplets decreased progressively whereas mineral oil droplets remained practically unchanged.

In studies carried out by Costa and Admur (21), respiratory function was monitored while guinea pigs were exposed for periods up to one hour to a variety of mineral oils. Below 200 mg/m^3 there were no significant effects on respiratory function for any of the following oils: Medicinal grade mineral oil, laboratory grade paraffin, light lubricating oil, unused motor oil and used motor oil. At concentrations above 200 mg/m^3 there was evidence of significantly reduced lung compliance. Further studies carried out by Costa and Amdur (22) indicated similar results and also that very high exposures to certain mineral oils conferred protection against the irritant effects of sulphur dioxide.

In studies carried out by Keen (23), Wistar rats were exposed to aerosols of diesel fuel and kerosine at concentrations up to 10,000 mg/m^3. Droplet size averaged less than 2 microns in diameter. There was no evidence of damage to the pulmonary surfactant layer. (It is likely that, certainly in the case of kerosine, considerable droplet vaporisation will have occurred resulting in exposure to vapour as well as mist).

Wagner et al (24) exposed albino mice to mineral oil aerosols at concentrations of about 200 mg/m^3 for four hours. The oils used were "SAE No. 10-20" automotive lubricating oils and "mineral oil". A mild inflammatory reaction was observed in the lungs of animals killed both immediately after the four hour exposure and at periods up to 144 hours post exposure, with a peak in the response being reached at 96 hours. It was also found in these studies that inhalation of the mineral oil particles reduced the acute lethal effects of respired oxidants such as ozone and nitrogen dioxide. This effect was demonstrable only after a latent period of up to 8-9 days following oil mist exposure and was thought to result from the formation of a thin film of oil on the alveolar surfaces.

It is noted that the nature and composition of oils used in these short-term studies have generally not been clearly defined. However, the observations of only mild effects, even at high exposures, indicate that a range of oils (including highly refined mineral base oils and formulated products) have low acute toxicity. No acute toxicity studies have been published on soluble oils, unrefined distillates or aromatic extracts.

4.2 SUB-ACUTE TOXICITY STUDIES ON MINERAL OIL MISTS

Sub-acute studies carried out by Shoshkes et al (20) involved exposure of mice for periods varying from 4 to 8 hours a day, 5 days a week for periods of 2 weeks for one series of experiments to approximately one month for another series. Concentrations used were 4500 and 4330 mg/m^3 for liquid petrolatum and SAE No. 10 motor oil respectively. There was heavy retention of oil particles of all size ranges in all sub-divisions of the respiratory tree. A large number of giant droplets (30 microns and larger in diameter) were seen, probably formed by the coalescence of many smaller droplets. This accumulation of droplets resulted in localised foreign body reactions of moderate severity as well as frequently occurring patches of lipoid pneumonia.

Studies carried out by Wagner et al (24) involving exposure of mice to 200 mg/m^3 of SAE No. 10-20 automotive lubricating oil or "mineral oil", for 7 hours/day for 4 consecutive days, resulted in no evidence of any pathological lesions in the animals' lungs at either 24 or 96 hours following the last exposures.

Eckert & Kandt (25) exposed 20 male Wistar rats to a mineral oil aerosol (described as "ura-mol F25 Hydrierwerk Zeitz") for 6 hours a day for up to 3 weeks. An extremely high aerosol concentration of 30,000 mg/m^3 was reported, with droplet diameters of approximately 0.3-0.5 microns. Changes consistent with classic lipoid pneumonia and oil granuloma formation followed this exposure. Further studies by Eckert et al (26) showed similar effects in female mice exposed 6 hours a day for 6 days.

Carpenter et al (27) have reported investigations into the sub-acute inhalation toxicity of aerosols of deodorised kerosine. Two groups of 25 rats were exposed to an aerosol concentration of 100 mg/m^3 for 6 hours a day, 5 days per week for 13 weeks. It was concluded that there were no treatment related adverse effects. It should be noted that in view of the volatility of kerosine it is very likely that animals in these studies were exposed to vapour as well as mist. 100 mg/m^3 is approximately equivalent to 10 to 15 ppm vapour; 20 mg/m^3 is approximately equivalent to 2 to 3 ppm. Calculations have shown that a 10 micron kerosine droplet will completely evaporate in under one second at 20°C (12). Because kerosine droplet vaporisation will decrease as the surrounding air becomes saturated with vapour, both animal and human exposures are likely to be to a mixture of mist and vapour. Droplets larger than 10 microns will have a longer life and therefore any pathological effects associated with kerosine mist may be more likely to occur in the upper respiratory tract.

Beagle dogs exposed to 20 mg/m^3 of deodorised kerosine aerosol 6 hours per day, 5 days a week for 13 weeks showed a borderline but significant mean weight increase at the end of the exposure period. There were no treatment related histopathological findings (Carpenter et al (27)). As noted above it is very likely that these animals were exposed to kerosine vapour as well as aerosol.

According to studies carried out by Lutov (28), rats were exposed to aerosols of lubricating oils (axle and machine) at concentrations of 60, 30 and 13 mg/m^3, 5 hours per day for 6 months. Reversible effects on immune reactivity, electrocardiograms, respiratory function, and arterial pressure were found. These effects were reported to occur even at the lowest exposure concentration. Some evidence of liver, kidney, adrenal and myocardial degeneration was reported in this study. Based on this and other similar studies a maximum admissible workplace concentration of 5 mg/m^3 was recommended as the official U.S.S.R. exposure limit.

In summary, sub-acute toxicity studies have been reported for liquid petrolatum, 4 formulated oils, 1 undefined mineral oil and deodorised kerosine. Results from these studies indicate that although most of the materials examined were not well defined, they were of a low order of sub-acute toxicity. Inflammatory reactions of the lung and lipoid pneumonia were observed following repeated exposures to extremely high concentrations. Studies at concentrations more relevant to the workplace (less than 50 mg/m^3) have failed to elicit toxicological effects.

4.3 CHRONIC TOXICITY STUDIES ON MINERAL OIL MISTS

In studies by Lushbaugh et al (29) of the effects of long-term inhalation of oil mists, animals were exposed to mists of automobile lubricating oil (Penn oil, SAE No. 10) and diesel engine lubricating oil (SGF No. 1) at concentrations of 132 mg/m^3 (average particle diameter 0.58 microns) and 63 mg/m^3 (average particle diameter of 0.45 microns) respectively. Rats and rabbits were exposed for one year, with no significant adverse effects being reported. No significant increase in tumour incidence or reduction in the latent period for spontaneous tumours were found in mice.

Monkeys exposed for 100 days accumulated relatively large amounts of oil in the lungs compared to mice. Although there was no evidence of lipoid pneumonia, the incidence of infective pneumonia was greatly increased. The authors noted the known susceptibility of these monkeys to pneumonia but concluded that the inhaled oil may have played a role in the increase in infective pneumonia.

Wagner et al (30) reported studies involving 5 species of laboratory animals (dog, rat rabbit, hamster, mouse). Animals were exposed daily for periods of from one year to 26 months to petroleum based mineral oil mist (described as a white mineral oil, naphthenic, molecular weight 350 to 410, 25 to 30 carbon atoms) at closely controlled concentrations of 5 mg/m^3 and 100 mg/m^3. The results suggest that the 5 mg/m^3 concentration would present no toxic hazard upon prolonged exposure. Exposure of both dogs and rats for 12 months at 100 mg/m^3 resulted in pulmonary deposition of oil and/or lipoid granuloma formation.

A parallel, but unpublished, study (31) of sulphurised solvent-extracted naphthenic base oil to which animals were exposed daily at 50 mg/m^3 for 18 months, failed to reveal a single animal with deleterious effects from oil mist inhalation.

The above studies form the main basis of the ACGIH TLV of 5 mg/m^3.

In a NIOSH (32) sponsored study, monkeys and rats were exposed to "oil mist" at a concentration of 50 mg/m^3. Monkeys were exposed for a period of 27 weeks to this concentration and rats for 13 months. In summary, no consistent, significant treatment related effects were evident in either rats or monkeys. Details of the oil used in these tests are not available.

Stula and Kwon (33) exposed dogs, rats, gerbils and two strains of mice for 6 hours a day, 5 days a week for up to 24 months to 5 and 100 mg/m^3 of a finishing oil with 1000 ppm acetone vapour. The finishing oil was described as a 70% paraffinic oil with 30% surfactant buffers and additives. Average particle diameters were 0.93 microns at 5 mg/m^3 and 1.15 microns at 100 mg/m^3. Oil particles were detectable within the lung macrophages of all species tested and at both concentrations. However, only at the higher concentration (100 mg/m^3) and in dogs and rats was the retention of oil of such a magnitude as to result in the development of oil-granuloma. Rats given a 10 month recovery period following the 12 months of exposure still showed the presence of the oil-granuloma. In the adenoma-sensitive strain of mice (CAF/JAX) which was studied, there was no significant difference in the lung adenoma incidence in the test or control animals. The results of this study are similar to those of Wagner et al (30) using a comparable concentration of pure white mineral oil of similar droplet size and also indicate that acetone has no effect on the toxicity of oil mist.

Summarising these aerosol studies, the limited range of materials investigated shows a low chronic toxicity and a lack of carcinogenic potential. Repeated prolonged exposure to relatively high levels (100 mg/m^3) has been reported to result in accumulation of oil in the lungs with lipoid granuloma formation. Primate studies have shown that, compared with other species, primates may accumulate greater quantities of oil in the lungs following mist exposures, and that repeated prolonged exposure to high levels of oil mist may exacerbate an existing predisposition to infective pneumonia. Long-term inhalation exposures at lower oil mist concentrations more similar to actual workplace levels have generally not shown adverse effects. No significant carcinogenic effects have been reported in any species studied.

4.4 THE TOXICITY OF OIL ADDITIVES

In view of the large variation in types, levels and toxicity of additives which can be present in industrial oils, each formulated product should be the subject of a toxicological review. It should be noted that for many products it is likely that the levels and toxicity of the additives are such that the ACGIH TLV of 5 mg/m^3 may still be applicable to the product. Nevertheless, this should be checked by an appropriate review of the toxicity and composition of each product.

4.5 COMMENT ON RELEVANCE OF ANIMAL MODELS FOR MAN

Several factors make the extrapolation of animal aerosol inhalation studies to man very difficult. These include: respiratory rate, diameter of the airways (particularly the upper airway), pulmonary defence mechanisms, background incidence of the normally occurring diseases, species susceptibility to specific diseases etc. For example, differences in deposition rates in the airways have been demonstrated for different species for small particles (Young et al (34, Hatch and Gross (35)). For larger particles it has been demonstrated that they also enter the gastro-intestinal tract (Goldberg and Leif (36), Berteau and Bermann (37)). It is probable that the fate of inhaled particles may depend on the size of the glottis (Dimmick et al (38), Pappagianis (39)).

Extreme care must, therefore, be taken when interpreting the results of animal studies with oil mist.

4.6 IARC EVALUATION OF THE CARCINOGENICITY OF MINERAL OILS

IARC (3) has reviewed the evidence in the available published literature for carcinogenicity of mineral oils to animals and man. The IARC conclusions, based on experimental animal skin painting studies, are shown in Table 2.

Table 2: IARC evaluation of carcinogenic risk of mineral oils

Type of oil	Carcinogenicity to experimental animals
Vacuum distillates	Sufficient evidence
Severely solvent refined	No evidence
Mildly solvent refined	Sufficient evidence
Severely hydrotreated	Inadequate evidence
Mildly hydrotreated	Sufficient evidence
Severely (oleum) acid treated	No evidence
Mildly acid treated	Sufficient evidence
Aromatic distillate extracts	Sufficient evidence
White oils	No evidence

For formulated products IARC concluded that data were inadequate to evaluate the carcinogenicity as a class to experimental animals since the possible carcinogenic activity of individual products is dependent upon the severity of processing of the base oils and the nature and concentrations of additives. A similar conclusion was reached for used formulated products; in this case activity is also dependent on the nature and concentration of contaminants, and the conditions of use. IARC also concluded that there is sufficient evidence from studies in humans that mineral oils (containing various additives and impurities) that have been used in occupations such as mulespinning, metal machining and jute processing have caused skin cancer in humans.

In assessing the applicability of exposure limits to various types of oil mists, the IARC evaluation should be taken into account since it is the most recent thorough published evaluation of mineral oil carcinogenicity available. However, the results of skin painting tests may not necessarily be directly translatable into inhalation effects.

4.7 CONCLUSIONS FROM ANIMAL STUDIES ON TOXICITY OF MINERAL OIL MISTS

The majority of the published oil mist toxicology studies which have been reviewed above have been deficient in one or more aspects. Typical deficiencies identified have been imprecise definitions of the oil evaluated, incomplete characterisation of the test atmospheres, use of small numbers of animals, and inadequate description of the experimental design and results. Furthermore, many of these studies were carried out at a time when techniques and understanding were not as well developed as at present. A number of other published studies which were reviewed have not been referred to in this document because they were considered to have more serious deficiencies.

There are two overriding concerns over the studies which have been reviewed. Firstly, only a narrow range of oils has been evaluated in animal studies and, secondly, due to the differences in respiratory physiology and anatomy, the most commonly studied species may not be the most appropriate models for assessing human effects.

Despite these reservations and the possible criticisms of individual studies, consideration of the range of published studies as a whole suggests the following useful conclusions on the toxicity of oil mists.

Mineral oil mists derived from highly refined oils and several formulated products appear to have a low acute and low sub-acute toxicity in animals. Single and short-term repeated exposures (up to 6 months) to relatively high concentrations (well in excess of 100 mg/m^3) have resulted in lung inflammatory reactions, lipoid granuloma formation, and lipoid pneumonia. In acute and sub-acute studies involving exposures more similar to actual workplace levels, there was, in general, no evidence of toxicological effects of significance to man. Only in two studies carried out by Russian investigators have reversible systemic toxic effects been observed, and from these it was concluded that 5 mg/m^3 would be an appropriate exposure limit.

Long-term inhalation studies indicate that those oils within a limited range which has actually been tested have a low chronic toxicity. Repeated prolonged exposures up to 2 years to very high concentrations, (100 mg/m^3 and above) have resulted in lung inflammatory reactions and lipoid granuloma formation. No carcinogenic effects have been reported in any species studied, including susceptible strains of mice. Adverse effects have not been found in long-term inhalation studies at lower oil mist concentrations more similar to actual workplace levels.

5. HUMAN HEALTH ASPECTS OF OIL MISTS

5.1 SHORT-TERM STUDIES

5.1.1 Skin

Various publications (4, 40, 41, 42, 43, 44) have shown that workers repeatedly and heavily exposed to mineral oil have developed skin lesions in the form of dermatitis and oil acne, but there is no published study indicating that lesions of this kind can be induced by skin exposure to oil mists alone.

5.1.2 Respiratory tract

The effects of aspirating mineral oil into the lung are well documented (40, 45, 46, 47, 48, 49). Large amounts of oil entering the lung cause a chemical pneumonitis which may occasionally be fatal or lead to complications such as pulmonary fibrosis. None of these complications have been reasonably substantiated from exposure to oil mist except where exposure has been massive; lipoid pneumonia has been reported following heavy exposure to oil mist in the absence of adequate ventilation (50, 51, 52). A German study (53) was unable to correlate complaints of respiratory irritation with exposure to concentrations of oil mist as high as 80 mg/m^3. However, this exposure and concentration may also have included some oil vapour.

Workplaces where oil mist was known to occur were investigated by NIOSH (54, 55, 56) in response to complaints from workers. None of the studies identified evidence of skin or respiratory tract irritation from exposures to oil mist which were at levels below the 5 mg/m^3 TWA-TLV.

A study on 19 volunteers from workers in a steel rolling mill (5) reported 12 with radiological evidence of increased linear striations in the lungs, but the changes were not marked and the causation uncertain. In a Norwegian study (14) of workers in a cable plant, slight basal lung fibrosis was reported. Although oil mist levels were reported in the range 0.15 to 0.30 mg/m^3, the authors suggested that the sampling methodology underestimated the actual exposures. Furthermore, the contribution of the substantial short-term vapour exposures (reported as up to 4000 mg/m^3) to lung fibrosis is uncertain. In a series of studies from Sweden (8, 57, 58), a sample of 164 workers making bearing rings were examined for the prevalence of respiratory symptoms (using a mailed questionnaire) and given extensive lung function tests. No lung function or X-ray abnormalities were detected though the exposed workers were assessed as complaining of more respiratory symptoms, consistent with irritation and chronic bronchitis, than an unexposed control group. In a follow-up of the same workers three years later, there was no substantial change in the author's conclusions.

Two other extensive reviews have not identified any serious health hazards in workers exposed to oil mists (59, 40).

5.2 LONG-TERM EPIDEMIOLOGY STUDIES

The principal concern about mineral oil mists stems from the knowledge that some oils (such as cutting oils developed from lightly refined base oils), when in regular and frequent contact with the skin (especially the scrotum), can cause skin cancer. Many investigators have thus tried to determine if there is an increased cancer risk associated with exposure to mist generated from the same types of oil.

5.2.1 Lung cancer

A number of investigations on metal machinists (7, 60, 61, 62, 63) have uniformly failed to confirm a potential risk of lung cancer.

Two studies (64, 65) have analysed the deaths of news print workers and both reported an excess of deaths from lung cancer over the numbers to be expected in the general population. However, because of confounding factors and limitations in the statistical methods used, neither study was able to conclusively associate occupation with the excess of lung cancer observed.

Other studies on print workers have been performed along similar lines (10, 66, 67, 68, 69) but have not found an increased incidence of lung cancer.

Parkes (70) reported a small excess of lung cancer among workers in the UK rubber industry in which aromatic extracts are used as extenders. However, because of the diversity and multiplicity of chemicals used or formed, and the confounding factors such as smoking and diet which have not been taken into consideration, the effect cannot be attributed to any specific agent.

5.2.2 Gastro-intestinal cancer

Two studies in the United States on workers employed in the printing industry (66, 67) have examined mortality data which suggested that a variety of gastro-intestinal cancers (rectum, colon, pancreas, buccal cavity and pharynx) may be more prevalent in these workers than in the general population, but neither study was able to conclude that the increased incidence of gastro-intestinal cancers reported could be directly linked with occupation.

In a survey of cause specific mortality rates of 5189 workers exposed to oil mist and employed for at least one year on metal machining in a heavy industry plant in the United States (71), small excesses of digestive tract and respiratory tract cancers were reported but these excess levels did not reach statistical significance or show a dose response relationship.

A second survey by the same author (7) on 2485 men who had been employed for 5 years or more in the same plant included a sub-group of 1137 workers with heavy exposure to oil mist. While accepting certain confounding variables had not been allowed for, the author concluded that occupational exposure to soluble and non-soluble cutting oil mists may be associated with certain forms of gastro-intestinal cancer.

Other studies (10, 64, 65, 68, 69) involving printers have not demonstrated an increased incidence of gastro-intestinal cancer. Similarly, other groups of machinists who have been studied (60, 61) have not shown an increased incidence of such cancer.

5.2.3 Kidney and bladder cancers

An analysis of 344 death certificates from a cohort of Los Angeles newspaper pressmen (68) revealed a statistically significant excess of deaths from kidney cancer but the number of cases was small and additional studies are required to verify an association with kidney cancer.

By contrast, several studies (10, 64, 65, 67, 72) do not indicate that printers are at particular risk of developing kidney and bladder cancers. Studies amongst machinists (7, 60, 61) also report negative findings.

5.2.4 Lymphatic and haematopoetic cancers

Excess deaths from multiple myeloma, Hodgkins disease and leukaemia were reported amongst workers employed in a U.S. Government Printing Office (67) but these excesses occurred primarily among compositors and binders rather than the pressmen who tend to be more exposed to oil mists. Other studies of printers (65, 66) have not identified a potential problem of these cancers; nor have studies among machinists (7, 61).

5.2.5 Skin cancer

It is well recognised that poorly refined mineral oil derived from petroleum can induce skin and scrotal cancers after prolonged, repeated and heavy direct contact with the skin; many studies confirm this association (73, 74, 75, 76, 77, 78). In one of these studies (73), on workers suffering from scrotal cancer, a statistically significant excess of cases of second primary tumours of the lung and skin was reported. The reason for these excesses could not be clearly explained though the cause was suspected to be exposure to oil in the course of their occupation. In a later review (80) of these data for the same population but covering an extra 5 years of employment, the number of second primary tumours did not appear to differ significantly between the groups with exposure to occupational carcinogens and those without.

Other reviews of the status of lubricating oils and the occurrence of skin cancer have been published (2, 3, 43, 76, 79). However, it should be noted that exposure to oil mist alone has not been shown to cause skin cancers.

5.3 CONCLUSION ON HUMAN HEALTH ASPECTS

Each of the studies reviewed suffers from one or more methodological weaknesses and none is considered strong enough to support a cause and effect relationship between exposure to lower levels of oil mists and adverse health effects. Attempts to demonstrate an association have, on balance, not identified any harmful biological effects. Thus on the basis of available studies, exposure to oil mist alone has not been demonstrated to cause adverse human health effects except at levels greatly above 5 mg/m^3. However, it is not possible to conclude from these limited human studies that 5 mg/m^3 is an acceptable exposure limit for all types of oils.

6. DISCUSSION

Central to the question of whether the ACGIH TLV of 5 mg/m^3 is applicable to all classes of oil mist, is the fact that lubricating oils usually consist of a base mineral oil to which are added varying percentages of additives. The presence of additives, some of which may not have been adequately tested for their biological effect, introduces uncertainties. Thus it is not advisable to apply the 5 mg/m^3 oil mist TLV to oils containing unknown concentrations and types of additive. If a manufacturer of lubricating oils wishes to adopt the 5 mg/m^3 exposure limit for his products, he must first satisfy himself, by appropriate review of the amounts, nature and toxicological properties of the additives present that oil mist exposures up to this level will not present any unacceptable risk to human health. In the absence of such a conclusion, a lower exposure limit should be recommended. The advice that the ACGIH TLV cannot be universally applied to all classes of oil mist is hinted at in the ACGIH documentation which states "Certain oils, however, may contain toxic or even carcinogenic components either natural or added, and in such cases there may be little or no safety factor in the 5 mg/m^3 TLV" (2).

Severely solvent refined base oils are not carcinogenic in mouse painting studies (3). By contrast, poorly refined base oils and their solvent extracts containing a higher proportion of PAHs are carcinogenic in skin painting studies. Although animal studies and human epidemiological investigations provide no convincing evidence to show that mists of mineral oils formulated from poorly refined base oils or aromatic extracts have induced cancers of the skin, respiratory system or gastro-intestinal tract, it seems prudent to advise that the 5 mg/m^3 oil mist TLV should not apply to oils of these types.

7. GENERAL SUMMARY AND CONCLUSIONS

There are no date to suggest that any serious adverse effects have been observed in animals at oil mist levels of up to about 5 mg/m^3. However, it is recognised that only a limited range of product types has been tested and that many of the reported studies can be considered deficient in one or more aspects.

Excessive skin contact of workers with certain mineral oils has been shown to cause oil acne and dermatitis as well as skin cancer. A few human studies have reported adverse lung effects at oil mist exposure levels below the ACGIH TLV of 5 mg/m^3. However, in all of these studies the quality of the exposure assessment can be seriously questioned on the grounds that the exposure data have been estimated retrospectively, because inappropriate sampling methodology has been used which would have grossly underestimated the actual exposures, or high concentrations of vapour have been present. Although no definitive study has demonstrated that exposure to oil mist alone has caused any adverse human health effects except at levels greatly above 5 mg/m^3, it cannot be concluded that this is an acceptable exposure limit for all types of product.

An association between exposure of human populations to oil mist and cancer of any particular organ system has never been regularly and predictably demonstrated. Although this suggests that there is unlikely to be a human risk of developing cancer from oil mist exposure, it must be recognised that the quality of epidemiological data can be questioned in terms of definition of exposures and the power to detect effects. In particular, populations exposed to airborne mists from products containing recognised carcinogens, such as aromatic extracts, have not been adequately studied. Furthermore, the small number of workers and the confounding factors involved, such as mixed exposures, are such that an authoritative study would be extremely difficult to conduct. Therefore, it would be prudent not to apply the ACGIH TLV of 5 mg/m^3 to oil mists from products containing components which are recognised as potential carcinogens unless objective assessment of the specific formulation indicates this would be acceptable.

Some products contain lighter oils such as kerosine and in such cases the rapid vaporisation and short life of mist droplets may present exposure assessment difficulties. The ACGIH oil mist TLV of 5 mg/m^3 may not be appropriate for these types of oils and it may also be necessary to assess exposure to oil vapour.

Some oils may contain a larger proportion of additives. In these cases, it may be more appropriate to monitor and control exposure to the additives rather than to oil mist. The current ACGIH TLV of 5 mg/m^3 may not, therefore, be appropriate for such oils.

With soluble oil emulsions, the water component of mist droplets vaporises rapidly. Standard exposure sampling and measurement techniques therefore only determine the non-volatile mineral oil and additive components of the mist which may not be indicative of the actual exposures. For this reason, as well as because of the high level of additives normally present, the ACGIH TLV of 5 mg/m^3 is unlikely to be appropriate for soluble oil emulsions.

The evidence reviewed therefore indicates the following conclusions:

1. A single generic exposure limit is not applicable to all types of oil mists;

2. The ACGIH TLV of 5 mg/m^3 provides an adequate safety margin for:

   a) a broad range of oils recognised as non-carcinogenic, i.e.:

      - severely solvent refined oils;
      - severly hydrotreated oils;
      - oils sequentially processed by mild hydrotreating and mild solvent refining;
      - white oils and petrolatums of medicinal or food grade quality.

   b) products formulated from the above base oils and containing additives which, by virtue of their nature or the small amounts present, are of no toxicological concern.

3. The ACGIH of 5 mg/m^3 may not be applicable to:

   - products containing lighter oils such as kerosine;
   - products for which exposures to additive components need to be controlled because of their nature or amounts present;
   - products used as emulsions.

   For these types of products, acceptable exposure limits can be based on review of specific formulations.

4. For products containing potentially carcinogenic oils, an exposure limit below 5 mg/m^3 would be prudent. Guidance provided in Appendix to CONCAWE Report No. 1/81 (15) is still considered applicable and is repeated here:

   "Where exposure to mists from oil containing significant concentrations of polycyclic aromatic hydrocarbons, such as aromatic extract oils, is likely to occur, some guidance can be gained from the ACGIH TLV-TWA of 0.2 mg/m^3 for particulate polycyclic aromatic hydrocarbons (as benzene soluble material). This TLV was the basis for the standard for employee exposure to coal tar pitch volatiles (0.2 mg/m^3 as benzene solubles) adopted by the USA Occupational Safety and Health Administration (OSHA) in 1971. Coal tar pitch volatiles are defined by OSHA as including the fused polycylic hydrocarbons which volatilise from the distillation residues of coal, petroleum, wood and other organic matter. In the case of aromatic extract oils, the fact that a major part of the benzene soluble material consists of non-polycyclic aromatics should be taken into account, i.e. a 0.2 mg/m^3 exposure limit may be unnecessarily restrictive".

The preface to the ACGIH publication (1) "Threshold limit values for chemical substances in the work environment", in which is quoted the 5 mg/m^3 oil mist limit, includes the following statement:

"In spite of the fact that serious injury is not believed likely as a result of exposure to the threshold limit concentrations, the best practice is to maintain concentrations of all atmospheric contaminants as low as is practical".

This established general principle of good industrial hygiene practice of reducing exposures to the lowest reasonably achievable level should be applied to oil mist as much as it is to any other airborne contaminant.

8. REFERENCES

1. ACGIH. Threshold limit values for chemical substances in work environment adopted by the ACGIH for 1985-86. Cincinnati, OH: American Conference of Governmental Industrial Hygienists

2. ACGIH (1980) Documentation of TLVs for substances in workroom air. Fourth edition. Cincinnati, OH: American Conference of Governmental Industrial Hygienists

3. IARC (1984) IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans, Vol. 33, Mineral oils (Lubricant base oils and derived products). Lyon: International Agency for Research on Cancer. p. 87-168

4. CONCAWE (1983) Health aspects of lubricants. Report No. 1/83. The Hague: CONCAWE

5. Jones, J.G. (1961) An investigation into the effects of exposure to an oil mist on workers in a mill for the cold reduction of steel strip. Annals of Occupational Hygiene 3, 264-271

6. Goldsmith, A.H. et al (1976) Health hazards from oil, soot and metals at a hot forging operation. American Industrial Hygiene Association Journal 37, 217-226

7. Decoufle, P. (1978) Further analysis of cancer mortality patterns among workers exposed to cutting oil mists. Journal of the National Cancer Institute 61, 4, 1025-1030

8. Järvholm, B. et al (1982) Respiratory symptoms and lung function in oil mist-exposed workers. Journal of Occupational Medicine 24, 6, 473-479

9. Lipmann, M. and Goldstein, D.H. (1970) Oil-mist studies, environmental evaluation and control. Archives of Environmental Health 21, 591-599

10. Goldstein, D.H. et al (1970) An epidemiologic study of an oil mist exposure. Archives of Environmental Health 21, 600-603

11. Lawther, P.J. (1979) A report on ink mist in newspaper press rooms. IFRA symposium on health safety and ecology in newspaper plant, Copenhagen

12. Davies, C.N. (1977) Atmospheric concentrations of oil mists. Annals of Occupational Hygiene 20, 91-92

13. Cullen, M.R. et (1981) Lipoid pneumonia caused by oil mist exposure from a steel rolling tandem mill. American Journal of Industrial Medicine 2, 51-58

14. Skyberg, K. et al (1986) Pulmonary fibrosis in cable plant workers exposed to mist and vapour of petroleum distillates. Environmental Research 40, 261-273

15. CONCAWE (1981) Guidelines for the determination of atmospheric concentrations of oil mists. Report No. 1/81. The Hague: CONCAWE

16. Turner, D. et al (1975) Methods for the determination of atmospheric concentration of oil mist. Annals of Occupational Hygiene 18, 293-297

17. NIOSH (1978) NIOSH manual of analytical methods. 2nd edition, Vol. 4. Cincinnati, OH: Department Health and Human Services

18. Deutsche Forschungsgemeinschaft (DFG) (1984) Maximum allowable concentrations at the workplace and biological tolerance values for working materials 1985. Report No. XXI, Weinheim: Verlag Chemie

19. Eyres, A.R. (1981) Polycyclic aromatic hydrocarbon contents of used metal working oils. Report IP 81-002. London: Institute of Petroleum

20. Shoshkes, M. et al (1950) Distribution, effect and fate of oil aerosol particles retained in the lungs of mice. Archives of Industrial Hygiene and Occupational Medicine 1, 20-35

21. Costa, D.L. and Amdur, M.O. (1979) Respiratory response of guinea pigs to oil mists. American Industrial Hygiene Association Journal 40, 8, 673-679

22. Costa, D.L. and Amdur, M.O. (1979) Effect of oil mists on the irritancy of sulfur dioxide. Mineral oils and light lubricating oil. American Industrial Hygiene Association Journal 40, 8, 680-685

23. Keen, T.E.B. (1968) The effects of petroleum distillates on lung surfactant. Australian Pediatric Journal 4, 229-235

24. Wagner, W.M.D. et al (1961) Antagonistic action of oil mist on air pollution: effects on oxidants, ozone and nitrogen dioxide. Archives of Environmental Health 2, 523

25. Eckert, H. and Kandt, D. (1975) Morphologische Veranderungen bei experimenteller Lipoidpneumonie. Zeitschrift für Erkränkungen der Atmungsorgane 142, 59-67

26 Eckert, H. et al (1979) Ultrastrukturelle und autoradiographische Untersuchungen in den Frühstadien einer experimentellen Lungenfibrose. Zeitschrift für Erkränkungen der Atmungsorgane 152, 37-41

27. Carpenter, C.P. et al (1979) Petroleum hydrocarbon toxicity studies XI. Animal and human response to vapours of deodorized kerosine. Toxicology and Applied Pharmacology 36, 443-456

28. Lutov, V.A. (1974) Data on the substantiation of maximum allowable concentrations of petroleum - derived additive - free oil aerosols used as lubricant-coolant fluids. Gigiena Truda I Professional 'nije Zabolevanija 10, 49-52

29. Lushbaugh, C.C. et al (1950) Effects of prolonged inhalation of oil fogs on experimental animals. Archives of Industrial Hygiene and Occupational Medicine, 237-247

30. Wagner, W.D. et al (1964) Inhalation toxicology of oil mists. Chronic effects of white mineral oil. American Industrial Hygiene Association Journal 25, 158-168

31. Wagner, W.D. et al Unpublished results, US Public Health Service, 4676 Columbia Parkway, Cincinnati, OH 45226

32. NIOSH (1972) Histopathology report of oil mist inhalation study in rats. Cincinnati, OH: National Institute for Occupational Safety and Health

33. Stula, E.F. and Kwon, B.K. (1978) Pulmonary pathology from inhalation of a complex mineral oil mist in dogs, rats, mice and gerbils. American Industrial Hygiene Association Journal 39, 5, 393-399

34. Young, H.W. et al (1984) Modified spinning top homogenous spray apparatus for use in experimental respiratory disease studies. Applied Microbiology 28, 929-934

35. Hatch, T.F. and Gross, P. (1964) Pulmonary deposition and retention of inhaled aerosols. New York: Academic Press, p. 64

36. Goldberg, L.J. and Leif, W.R. (1950) The use of radioactive isotope in determining the retention and initial distribution of airborne bacteria in the mouse. Science 112, 299-300

37. Berteau, P.E. and Biermann, A.H. (1977) Respiratory deposition and early distribution of inhaled vegetable oil aerosols in mice and rats. Toxicology and Applied Pharmacology 39, 177-183

38. Dimmick, R.L. et al (1975) A simple method for estimation of coagulation efficiency in mixed aerosols. Journal of Aerosol Science 6, 375-377

39. Pappagianis, D. (1969) Some characteristics of respiratory infection in man. In: An Introduction to Experimental Aerobiology. (Dimmick R.L. and Akers, A.B. eds) New York: Wiley-Interscience. p. 390-406

40. Liss-Suter, D. and Villaume, J.E. (1978) Occupational health and safety aspects of the fog oils SGF No. 1 and SGF No. 2 and smoke screens generated from them.
Report prepared by the Science Information Services Department of the Franklin Institute Research Laboratories for the US Army Medical Research and Development Command under contract DAMD-17-77-C-7020

41. Cottini, G.B. and Mazzone, G.B. (1939) The effects of 3:4 benz-pyrene on human skin. American Journal of Cancer 37, 186-195

42. Hodgson, G. (1973) Health problems arising from contact and exposure of workers to metal working fluids. Journal of the Institute of Petroleum 59, 1-8

43. Swedish Health Authority (1982) Consensus report for oil mist. Arbete och Halsa 9, 69-83

44. Finnie, J.S. (1960) Oil folliculitis. A study of 200 men employed in an engineering factory. British Journal of Industrial Medicine 17, 130-140

45. Weill, H. et al (1964) Early lipoid pneumonia. Roentgenologic, anatomic and physiologic characteristics. American Journal of Medicine 36, 370-376

46. Miller, A. et al (1962) Mineral oil pneumonia. Annals of Internal Medicine 57, 627-634

47. Keshishian, J.M. et al (1969) Lipoid pneumonia. Review with a report of a case of carcinoma occurring within an area of lipoid pneumonia. Annals of Thoracic Surgery 7, 3, 231-234

48. Volk, B.W. (1964) Lipoid pneumonia - clinical pathological and chemical aspects. Biochem. Clin. 4, 187-194.

49. Laughlen, G.F. (1925) Studies on pneumonia following naso-pharyngeal injections of oil. American Journal of Pathology 1, 407-415

50. Foe, R.B. and Bingham, R.S. (1954) Lipoid pneumonia following occupational exposure to oil spray. Journal of the American Medical Association 155, 1, 33-34

51. Proudfit, J.P. et al (1950) Chronic lipoid pneumonia following occupational exposure. Archives of Industrial Hygiene and Occupational Medicine 1, 105-111

52. Weissman, H. Lipoid pneumonia: a report of two cases. American Review of Tuberculosis 64, 572-576

53. Drasche, H. et al (1974) A survey of some occupational medical aspects of exposure to oil mists. (Arbeitsmedizinische Erhebungen bei ölnebelexponierten personen) Zentralblatt für Arbeitsmedizin und Arbeitsschutz 10, 1-15

54. Butler, G.J. (1974) Health hazard evaluation/toxicity determination report 73-193-133. Rockville, Maryland: National Institute for Occupational Safety and Health

55. Gunter, B. (1979) Health hazard evaluation determination report no. 79-62-606. Cincinnati, OH: National Institute for Occupational Safety and Health

56. Chrostek, W.J. (1978) Health hazard evaluation determination report no. 78-88-533. Cincinnati, OH: National Institute for Occupational Safety and Health

57. Järvholm, B. and Thiringer, G. (1981) Screening for lung diseases in men exposed to oil mists. European Journal of Respiratory Diseases. 62, 47

58. Järvholm, B. (1982) Cutting oil mist and bronchitis. European Journal of Respiratory Diseases. 63, 118, 79-83

59. Hendricks, N.V. et al (1962) A review of exposures to oil mist. Archives of Environmental Health 4, 264-271

60. Järvholm, B. et al (1981) Cancer morbidity among men exposed to oil mist in the metal industry. Journal of Occupational Medicine 23, 333-335

61. Ely, T.S. et al (1970) A study of mortality, symptoms and respiratory function in humans occupationally exposed to oil mist. Journal of Occupational Medicine 12, 7, 253-261

62. Decoufle, P. (1976) Cancer mortality among workers exposed to cutting-oil mist. Annals New York Academy of Sciences 271, 94-101

63. Nickol, K.H. (1970) Hazard of mineral oil mist? Lancet 1184-1185

64. Moss, E. et al (1972) Mortality of newspaper workers from lung cancer and bronchitis 1952-66. British Journal of Industrial Medicine 29, 1-14

65. Greenberg, M. (1972) A proportional mortality study of a group of newspaper workers. British Journal of Industrial Medicine 29, 15-20

66. Lloyd, J.W. et al (1977) Unusual mortality experience of printing pressmen. Journal of Occupational Medicine 19, 8, 543-550

67. Greene, M.H. et al (1979) Cancer mortality among printing plant workers. Environmental Research 20, 66-73

68. Paganini-Hill, A., et al (1980) Cause-specific mortality among newspaper webs pressmen. Journal of Occupational Medicine 22, 8, 542-544

69. Pasternack, B. and Ehrlich, L. (1972) Occupational exposure to an oil mist atmosphere - a 12-year mortality study. Archives of Environmental Health 25, 286-294

70. Parkes, H.G.A. (1984) Follow-up study of cancer mortality in the British rubber industry. Conference on Health and Safety in the Plastics and Rubber Industries, York, England

71 Decoufle, P. (1976) Cancer mortality among workers exposed to cutting oil mist. Annals New York Academy of Sciences, 271, 94-101

72. Cole, P. et al (1972) Occupation and cancer of the lower urinary tract. Cancer 29, 1250-1260

73. Brown, A.J. et al (1975) Report on a study of occupational skin cancer with special reference to scrotal cancer. University of Birmingham

74. Waldron, H.A. (1977) Health care of people at work - exposure to oil mist in industry. Journal Society of Occupational Medicine 27, 45-49

75. Holmes, J.G. et al (1970) Subsequent malignancies in man with scrotal epithelioma. Lancet, 214-215

76. Kipling, M.D. (1971) Oil cancer in the Savoy Alps and Birmingham region: a comparison. Transactions of the Society of Occupational Medicine 21, 73-78

77. Waterhouse, J.A.H. (1972) Lung cancer and gastro-intestinal cancer in mineral oil workers. Annals of Occupational Hygiene 15, 43-44

78. Waldron, H.A. (1975) The carcinogenicity of oil mist. British Journal of Cancer 32, 256-257

79. Tordoir, W.F. (1974) Oliemist en longafwijkingen. Tijdschrift voor Sociale Geneeskunde 52, 149-151

80. Waldron, H.A. et al (1984) Scrotal cancer in the West Midlands 1939-76. British Journal of Industrial Medicine 41, 437-444.