



WORLD HEALTH ORGANIZATION  
INTERNATIONAL AGENCY FOR RESEARCH ON CANCER

IARC Monographs on the Evaluation of Carcinogenic Risks to Humans

**Volume 33**  
**Polynuclear Aromatic Hydrocarbons,**  
**Part 2, Carbon Blacks, Mineral Oils (Lubricant Base Oils**  
**and Derived Products) and Some Nitroarenes**

**Summary of Data Reported and Evaluation**

---

[Mineral oils \(lubricant base oils and derived products\)](#)

Nitroarenes

[9-Nitroanthracene](#)

[3-Nitrofluoranthene](#)

---

Last updated: 20 April 1998

# MINERAL OILS

VOL.: 33 (1984) (p. 87)

## 5. Summary of Data Reported and Evaluation

### 5.1 Experimental data

Experiments involving repeated applications of petroleum-derived base oils and formulated products to the skin of mice have been used to evaluate potential skin carcinogenicity. Certain compounds have also been tested in a feeding study, and by subcutaneous and intraperitoneal injection.

Vacuum distillate fractions [class 1], either naphthenic or paraffinic in nature, produced a significant skin tumour response. Dewaxing of these distillates did not appreciably alter their activity.

Acid-treated oils [class 2] of either naphthenic or paraffinic origin produced a significant skin tumour response, unless severe acid treatment had been applied.

Solvent-refined oils (raffinates) [class 3], either naphthenic or paraffinic in nature, generally did not produce skin tumours, unless the solvent treatment had been only mild; in that case, samples retained some of the skin tumour-inducing activity of the original distillate.

Hydrotreated oils [class 4], principally paraffinic in nature, induced a moderate incidence of skin tumours when treatment of the distillates was mild, while no tumour was induced by severely hydrotreated oils. The combination of mild hydrotreating and solvent extraction appears to reduce or eliminate skin tumorigenicity.

White oils and petrolatums [class 5], which are produced from oils that have undergone the most severe acid and/or hydrogen treatment, showed no activity in the skin tumour assay. Subcutaneous injection of three different grades of medicinal petrolatum [class 5] into mice induced no tumour. Intraperitoneal injection of two food-grade mineral oils [class 5] into certain strains of mice induced plasma-cell neoplasms and reticulum-cell sarcomas. A study in rats involving subcutaneous or intraperitoneal injection of liquid paraffin and yellow petroleum [class 5] could not be evaluated. In two feeding studies in which three different samples of medicinal-grade petrolatum and liquid paraffin [class 5] were fed to rats for two years, no significant increase occurred in tumour incidence.

Solvent extracts (sometimes called aromatic oils), which are by-products of solvent refining [class 6.1], induced a significant incidence of skin tumours. The same response was produced with highly concentrated aromatic extracts of medicinal-grade petrolatums. High-boiling fractions from catalytically cracked oils (also classified as aromatic oils) [class 6.2] produced increasing numbers of skin tumours in mice with increasing boiling-ranges above 370 °C; further fractionation established that the activity is maximal in those boiling at 500-520 °C and is concentrated in the aromatic portion of the oils. Promoting activity was also detected in some portions. High-boiling, catalytically cracked oils also produced skin tumours in rabbits and monkeys.

Three formulated products [class 7.1], consisting of blends of base oils and chemical additives, were tested. One of the products, an unused gasoline-engine oil, gave some indication of skin tumorigenic activity (one squamous-cell carcinoma in 36 treated animals); three further samples of unused gasoline-engine oil gave negative results. One sample of unused diesel-engine oil also failed to induce a significant increase in tumour incidence. Used gasoline-engine oils [class 7.2] have tended to have a greater skin tumour activity than unused products. Four samples of cutting oil [class 7.1] tested in the mouse-skin tumour assay had significant activity (the degree of refining of the base oil is not known). Two samples of used cutting oil [class 7.2] were also tested; one was more active than a comparable unused oil, and the other was inactive.

Mineral oils have been found to be embryotoxic and teratogenic to birds; no study on teratogenicity of mineral oils in mammals was available to the Working Group.

Samples of a white oil [class 5], of a refined steel-hardening oil [class 7.1] and of unused crankcase oils [class 7.1] were not mutagenic to *Salmonella typhimurium* strain TA98 in the presence or absence of an exogenous metabolic system. Samples of vacuum distillates [class 1] of solvent-refined oils [class 3], of hydrotreated oils [class 4], of a used hardening oil [class 7.2] and of used crankcase oils [class 7.2] were mutagenic to *S. typhimurium* in the presence and absence (class 7.2 only) of an exogenous metabolic system.

## 5.2 Human data

Mineral oils (lubricant base oils and derived products) are produced in large quantities and are contained in a wide variety of products which are used primarily for lubricating purposes. The composition of these oils varies depending on the crude oil source, the refining process and the additives present. The degree of human exposure to these products also varies widely: in the case of cutting oils, appreciable skin contact and inhalation can occur, unless adequate care is taken, whereas limited exposure occurs to oils (such as hydraulic, circulating, turbine and engine oils) used in closed systems, with which only incidental contact is likely. There are thus various opportunities for occupational, consumer and environmental exposure to these products from their production, use and disposal.

Inhalation, aspiration or ingestion leading to aspiration of white oils and petrolatums suitable for food and medicinal use [class 5] can lead to lipid pneumonia and lipid granuloma.

Exposure to the mineral oils that have been used in a variety of occupations, including mulespinning, metal machining and jute processing, has been associated strongly and consistently with the occurrence of squamous-cell cancers of the skin, and especially of the scrotum.

The epidemiological studies of metal workers that were available to the Working Group comprised one proportional mortality study, three case-control studies and three cohort studies. No excess of respiratory cancer was reported from any of the studies. Excess gastrointestinal malignancies were seen in each of the three cohort studies (stomach in one study, the sum of stomach plus large intestine in one study, and digestive tract in one study). Four cases of scrotal cancer were detected in one relatively small cohort study of metal industry workers. In a case-control study, a relative risk of 4.9 was reported for the association of scrotal cancer with potential exposure of metal workers to mineral oils. Neither the actual levels of exposure nor the classification of the nature of the mineral oil to which the machine workers were potentially exposed were available in the reports of the epidemiological studies. In another case-control study, an excess of sinonasal cancers was seen in toolsetters, set-up men and toolmakers.

An examination of the incidence of second primary cancer among men with scrotal cancer demonstrated excesses of respiratory, upper alimentary tract and skin cancers; when the occupations were grouped, the excess was largely confined to those with oil exposure.

Excesses of bladder cancer have been reported in case-control studies in several countries among machinists and engineers, who were possibly exposed to cutting oils containing aromatic amines as additives.

With regard to printing pressmen, the Working Group considered the results of three cohort mortality studies (one of these was an extension of another) and two proportional mortality studies. There were three additional proportional mortality studies on manual workers in the newspaper printing industry; one of these included two separately exposed groups at different geographical locations. One of the two cohort studies addressing lung cancer showed an excess (not tested for statistical significance). One of the two proportional mortality studies showed a small, statistically non-significant excess of lung cancer among the newspaper pressmen but no excess among non-newspaper pressmen; the other study did not address lung cancer. One of the three proportional mortality studies on manual workers in the printing industry, not specifically addressing printing pressmen, did not show an increased lung cancer risk, whereas the other two studies (not on independent populations) found a statistically significant excess. One of the two proportional mortality studies of printing pressmen indicated a statistically significant excess of rectal cancers, and the other showed a statistically non-significant increase of colon cancers; the cohort study considering colorectal cancers did not show an increased occurrence. One proportional mortality study among newspaper and other commercial printing

pressmen showed a statistically significant excess of cancers of the buccal cavity and pharynx, whereas no such excess was observed in a cohort study. One proportional mortality study among employees in the printing industry (pressmen were not singled out) showed a slight deficit of cancers of the mouth and throat. One case-control study indicated a statistically significant excess of cancers of the buccal cavity and pharynx. The findings regarding other malignancies were inconsistent; scrotal cancers were not mentioned. The type and amount of exposure were usually not described; exposure to both mineral oils and carbon blacks would probably have been involved.

In mortality statistics from the United Kingdom and from Washington State, USA, lung and skin cancer excesses have been registered for jobs entailing exposure to mineral oils; despite limitations of this kind of source as to a causal interpretation, the consistency of such suggestions is worth noting.

### 5.3 Evaluation

There is *sufficient evidence* for the carcinogenicity in experimental animals of untreated vacuum distillates, acid-treated oils, and aromatic oils, including extracts from solvent treatment of distillates and the high-boiling fraction of catalytically cracked oils [classes 1, 2 and 6].

There is *sufficient evidence* that mildly solvent-refined oils [class 3] are carcinogenic to experimental animals. There is *no evidence* that severely solvent-refined oils [class 3] are carcinogenic to experimental animals.

There is *sufficient evidence* that mildly hydrotreated oils [class 4] are carcinogenic to experimental animals; the available data on severely hydrotreated oils [class 4] are *inadequate* to permit an evaluation of their carcinogenicity to experimental animals.

There is *no evidence* for the carcinogenicity to experimental animals of white oils [class 5] when administered by routes other than intraperitoneal injection; when white oils were given by intraperitoneal injection to mice, plasma-cell tumours were produced in repeated experiments. The significance of the latter findings is difficult to interpret.

The data are *inadequate* to evaluate the carcinogenicity to experimental animals of formulated products [class 7.1] as a class, since the possible carcinogenic activity of individual products is dependent upon the severity of processing of the base oils and the nature and concentration of additives.

The data are *inadequate* to evaluate the carcinogenicity to experimental animals of used formulated products [class 7.2] as a class, since the possible carcinogenic activity of individual products is dependent upon the quality of the base oils used, the nature and concentration of additives and contaminants, and the conditions of use.

There is *sufficient evidence* for the carcinogenicity of one sample of used gasoline-engine oil [class 7.2] and *limited evidence* for the carcinogenicity of some cutting oils [classes 7.1 and 7.2] to experimental animals.

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 are carcinogenic to humans.

N.B. - In the absence of adequate data in humans on individual classes of mineral oils, it is reasonable, for practical purposes, to regard fractions for which there is *sufficient evidence* of carcinogenicity in experimental animals as if they presented a carcinogenic risk to humans.

For definition of the italicized terms, see [Preamble Evaluation](#).

**Subsequent evaluation:** [Suppl. 7 \(1987\)](#)

---

Last updated: 20 April 1998

# 9-NITROANTHRACENE

**VOL.:** 33 (1984) (p. 179)

**CAS No.:** 602-60-8

**Chem. Abstr. Name:** Anthracene, 9-nitro-

## 5. Summary of Data Reported and Evaluation

### 5.1 Experimental data

No study of the carcinogenicity of 9-nitroanthracene to experimental animals was available to the Working Group.

9-Nitroanthracene was mutagenic to *Salmonella typhimurium* in the presence and absence of an exogenous metabolic system.

### 5.2 Human data

Environmental exposure to 9-nitroanthracene occurs, as it is a constituent of particulate matter in diesel exhaust and in urban air and has been found in one type of carbon black that is no longer produced commercially. 9-Nitroanthracene is produced only for research purposes.

No case report or epidemiological study was available to the Working Group.

### 5.3 Evaluation

No data were available to evaluate the carcinogenicity of 9-nitroanthracene to humans or to experimental animals.

No evaluation of the carcinogenicity of 9-nitroanthracene to humans could be made.

**Subsequent evaluation:** Suppl. 7 (1987) (p. 67: **Group 3**)

### Synonym

- 5-Nitroanthracene

# 3-NITROFLUORANTHENE

**VOL.:** 33 (1984) (p. 201)

**CAS No.:** 892-21-7

**Chem. Abstr. Name:** Fluoranthene, 3-nitro-

## 5. Summary of Data Reported and Evaluation

### 5.1 Experimental data

In one limited experiment in which 3-nitrofluoranthene was tested in male rats by subcutaneous injection, sarcomas were observed at the injection site.

3-Nitrofluoranthene was mutagenic to *Salmonella typhimurium* in the presence and absence of an exogenous metabolic system. It induced morphological transformation in Syrian hamster embryo cells.

### 5.2 Human data

Environmental exposure to 3-nitrofluoranthene occurs, as it is a constituent of particulate matter in diesel-engine exhaust and in urban air. It is not produced commercially.

No case report or epidemiological study was available to the Working Group.

### 5.3 Evaluation

The available data are *inadequate* to permit an evaluation of the carcinogenicity of 3-nitrofluoranthene to experimental animals.

No data on humans were available.

No evaluation of the carcinogenicity of 3-nitrofluoranthene to humans could be made.

For definition of the italicized terms, see [Preamble Evaluation](#).

**Subsequent evaluation:** Suppl. 7 (1987) (p. 67: **Group 3**)

### Synonym

- 4-Nitrofluoranthene