



WORLD HEALTH ORGANIZATION
INTERNATIONAL AGENCY FOR RESEARCH ON CANCER

IARC Monographs on the Evaluation of Carcinogenic Risks to Humans

Volume 35
Polynuclear Aromatic Compounds, Part 4,
Bitumens, Coal-tars and Derived Products,
Shale-oils and Soots

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Last updated: 20 April 1998

BITUMENS

VOL.: 35 (1985) (p. 39)

5. Summary of Data Reported and Evaluation

5.1 Exposures

World usage of bitumens is estimated to be more than 60 million tonnes per year; exposures occur in a variety of applications. A total of 90-95% of bitumen is used hot (> 100 °C) in road construction, roofing and flooring. Fumes from these operations contain polynuclear aromatic compounds, although almost all of the mass of material in the fumes remains uncharacterized. Cutback bitumens, blended grades and bitumen emulsions are usually used at ambient or warm temperatures, where the potential for direct skin contact may be greater.

Blended or fluxed bitumens, which represent a relatively small percentage of the total usage, may contain aromatic oils, thermally-cracked petroleum residues or coal-tar products, which contain polynuclear aromatic compounds.

5.2 Experimental data

Steam-refined petroleum bitumens were tested by application to the skin of mice. Skin tumours were produced with undiluted bitumens, with dilutions in benzene and with a fraction of steam-refined bitumen.

When air-refined (oxidized) bitumens were applied to the skin of mice, no tumour was found with undiluted bitumens; but, in one experiment, an air-refined bitumen in solvent (toluene) produced topical skin tumours.

Two cracking-residue bitumens produced skin tumours when applied to the skin of mice.

A pooled mixture of steam- and air-blown petroleum bitumens in benzene produced tumours at the site of application on the skin of mice.

One sample of heated, air-refined bitumen injected subcutaneously into mice produced a few sarcomas at the injection sites.

A pooled mixture of steam- and air-blown petroleum bitumens produced sarcomas at the site of subcutaneous injection in mice. Steam-distilled bitumens injected intramuscularly produced local sarcomas in one experiment in rats.

Both an extract of road-surfacing bitumen and its emissions were mutagenic to *Salmonella typhimurium*.

Subsequent to the meeting of the Working Group, the Secretariat became aware of a study showing that solutions of the fumes from two types of roofing bitumens, generated at 232 °C or 316 °C, produced skin tumours when applied topically to mice (Thayer *et al.*, 1983).

5.3 Human data

No epidemiological study of workers exposed only to bitumens is available. A cohort study of US roofers indicates an increased risk for cancer of the lung and suggests increased risks for cancers of the oral cavity, larynx, oesophagus, stomach, skin and bladder and for leukaemia. Some support for excess risks of lung, oral cavity and laryngeal cancers is provided by other epidemiological studies of roofers. As roofers may be exposed not only to bitumens but also to coal-tar pitches and other materials, the excess cancer risk cannot be attributed specifically to bitumens. Several case reports of skin cancer among workers exposed to bitumens

are available; however, exposure to coal-tars or products derived from them cannot be ruled out.

5.4 Evaluation

There is *sufficient evidence* for the carcinogenicity of extracts of steam-refined bitumens, air-refined bitumens and pooled mixtures of steam- and air-refined bitumens in experimental animals.

There is *limited evidence* for the carcinogenicity of undiluted steam-refined bitumens and for cracking-residue bitumens in experimental animals.

There is *inadequate evidence* for the carcinogenicity of undiluted air-refined bitumens in experimental animals.

There is *inadequate evidence* that bitumens alone are carcinogenic to humans.

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

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

Last updated: 20 April 1998

COAL-TARS AND DERIVED PRODUCTS

VOL.: 35 (1985) (p. 83)

5. Summary of Data Reported and Evaluation

5.1 Exposures

Large quantities of crude coal-tars, which contain polynuclear aromatic compounds as major components, are formed as by-products of the destructive distillation of coal, coke-ovens being the major source. The distillate fractions of crude coal-tars alone or in combination with coal-tar pitch, both of which contain polynuclear aromatic compounds, are mixed to produce various products, such as creosote. Most human exposure to crude coal-tars and derived products takes place during the destructive distillation of coal in foundries and in aluminium production, on which monographs have already been prepared. Members of the general population use topical pharmaceutical coal-tar preparations.

Exposure to polynuclear aromatic compounds in other occupations is especially heavy among workers applying hot coal-tar pitch, such as in roofing, paving, surface coatings and in the production of refractory bricks. There is also potential exposure in coal-tar distillation plants.

Creosote has world-wide use as a wood preservative. In addition to possible inhalation exposures, cutaneous exposure may be considerable.

5.2 Experimental data

Three high-temperature tars, one undiluted and two as benzene extracts, all produced skin tumours, including carcinomas, when applied to the skin of mice. Each of five blast-furnace tars and two extracts of blast-furnace tars produced skin tumours, including carcinomas, after topical application to mice.

Each of five pharmaceutical coal-tar preparations caused skin tumours, including carcinomas, when applied to the skin of mice.

Two unspecified coal-tars both caused skin tumours, including carcinomas, after application to the skin of mice. Lung tumours but no skin tumour were produced in rats after application of a coal-tar to the skin. A fourth, unspecified coal-tar produced tumours when applied to the ears of rabbits.

Intramuscular administration of a coal-tar fume condensate to mice in one experiment gave evidence of sarcoma formation.

Five creosotes or creosote oils all produced skin tumours, including carcinomas, when applied to the skin of mice. One of the creosotes also produced lung tumours in mice after skin application. In two limited studies, a basic fraction of creosote oil was not carcinogenic for the skin of mice.

In two experiments, anthracene oils produced skin tumours (and, in one experiment, carcinomas) when applied to the skin of mice.

Six coal-tar pitches and three extracts of coal-tar pitches all produced skin tumours, including carcinomas, when applied to the skin of mice. An extract of roofing-tar pitch had both initiating and promoting activity in separate experiments.

In one experiment, mice developed skin tumours, including carcinomas, after whole-body exposure to pitch powder.

No data were available to the Working Group on the carcinogenicity of distillation fractions of low-temperature tars or of products derived from them.

Samples of therapeutic coal-tar and extracts of coal-tar shampoos were mutagenic in *Salmonella typhimurium* in the presence of an exogenous metabolic system. Extracts of urine from patients undergoing combined treatment with coal-tar preparations and ultraviolet light were mutagenic in *S. typhimurium*.

Extracts of coal-tar pitch were mutagenic in *S. typhimurium* in the presence of an exogenous metabolic system.

Extracts of roofing-tar emissions were mutagenic in *S. typhimurium* in the presence of an exogenous metabolic system and were mutagenic in two mutation assays and induced sister chromatid exchanges in cultured mammalian cells, both in the presence and absence of an exogenous metabolic system. Viral transformation was enhanced in Syrian hamster embryo cells. A statistically non-significant increase in the number of morphologically transformed foci was observed in BALB/c 3T3 cells. The material did not cause DNA fragmentation in cultured Syrian hamster embryo cells.

Creosote and a coal-tar-creosote mixture were mutagenic in *S. typhimurium* and were positive in the mouse lymphoma L5178Y system, in the presence of an exogenous metabolic system. The urine from rats administered creosote was mutagenic in *S. typhimurium* in the presence of an exogenous metabolic system.

5.3 Human data

A mortality analysis in the UK from 1946 showed a greatly increased scrotal cancer risk for patent-fuel workers exposed to pitch. Furthermore, a large number of case reports describing the development of skin (including the scrotum) cancer in workers occupationally exposed to coal-tar and/or to pitch have been published.

A cohort study of US roofers indicated an increased risk for cancer of the lung and suggested increased risks for cancers of the oral cavity, larynx, oesophagus, stomach, skin and bladder and for leukaemia. Some support for excess risks of lung, laryngeal and oral-cavity cancer is provided by other epidemiological studies of roofers. Roofers may be exposed not only to a mixture of pitches but also to bitumens and other materials.

One study showed a small excess of bladder cancer in tar distillers and in patent-fuel workers. An elevated risk of cancer of the renal pelvis was also seen in workers exposed to 'petroleum or tar or pitch'.

There have been a number of case reports of skin cancer developing in patients using tar ointments to treat a variety of skin diseases.

A mortality analysis of many occupations indicated an increased risk of mortality from scrotal cancer for creosote-exposed brickmakers. Malignant epitheliomas, about a third of which were of the scrotum, have been reported in several case reports of workers exposed to creosote. The only available cohort study suffered from limitations of size.

5.4 Evaluation

There is *sufficient evidence* for the carcinogenicity in experimental animals of coal-tars, creosotes, creosote oils, anthracene oils and coal-tar pitches.

There is *sufficient evidence* that occupational exposure to coal-tars as it occurs during the destructive distillation of coal is causally associated with the occurrence of skin cancer in humans (IARC, 1984a). The findings of the few studies available on other occupational exposures to coal-tars are consistent with that evaluation.

There is *sufficient evidence* that coal-tar pitches are carcinogenic in humans.

There is *limited evidence* that coal-tar-derived creosotes are carcinogenic in humans.

Taken together, the data indicate that coal-tars and coal-tar pitches are causally associated with cancer in humans and that creosotes derived from coal-tars are probably carcinogenic in humans.

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

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

Last updated: 20 April 1998

SHALE-OILS

VOL.: 35 (1985) (p. 161)

5. Summary of Data Reported and Evaluation

5.1 Exposures

Commercial oil-shale processing industries presently exist only in the USSR and in China (a commercial facility is expected to begin operation in the USA in 1984), producing crude shale-oils that are used as fuels or chemical-plant feedstocks. Crude shale-oils were produced in the past in several countries; an industry existed in the UK for more than 100 years.

Workers involved in oil-shale mining and processing may be exposed to complex mixtures of dusts, gases and vapours. The dusts may contain significant levels of crystalline silica. Inorganic gases and vapours to which workers may be exposed include carbon monoxide and hydrogen sulphide. Workers may also be exposed to gases and vapours containing organic compounds, including low levels of polynuclear aromatic compounds. The composition of spent oil shale can vary widely in respect to the remaining tar material, depending on the variation of retorting methods. Skin contact with crude shale-oil may occur, but is limited primarily to maintenance workers in modern oil-shale processing facilities.

Contact with shale-oil liquids occurred extensively in the past in the Scottish shale-oil industry, and in the British cotton-textile industry where lubricants derived from shale-oils were used.

5.2 Experimental data

Extracts of raw oil shale from Scotland (UK) and the Green River Formation (USA) were tested by skin application in mice, and no skin tumour was observed. A suspension of raw oil-shale dust from the Green River Formation was also tested by intratracheal instillation in rats and hamsters, and no local tumour was observed; exposure of rats by inhalation to raw oil-shale dust resulted in the induction of lung tumours.

Crude shale-oils from low-temperature retorting from different sources were tested for carcinogenicity in various experiments by skin application in different strains of mice. Samples from Jurassic Chuvash (USSR), Estonia (USSR), the Green River Formation (USA) and Fushun (China) all resulted in the induction of benign and malignant skin tumours. A sample from Estonia was also tested by skin application in rabbits and induced benign and malignant skin tumours. Lung tumours were produced in mice following intratracheal administration of a crude shale-oil from the Green River Formation.

Three samples of crude shale-oils from high-temperature retorting from Estonia were tested for carcinogenicity by skin application in mice, and one sample was also tested in rabbits. All the samples resulted in the induction of benign and malignant skin tumours; these samples were more carcinogenic than crude shale-oils from low-temperature retorting from the same source.

An extract of spent oil shale from the Green River Formation resulted in the induction of skin tumours in mice after topical application. Dusts prepared from this sample induced lung tumours in rats after inhalation exposure. No lung tumour occurred in rats or hamsters exposed by intratracheal administration to a suspension of the spent oil-shale dusts.

Various fractions of low- and high-temperature shale-oils were tested by skin application in mice and rabbits and by intramuscular application in mice; their carcinogenic activities did not necessarily parallel the benzo[a]pyrene content of the fractions.

Various crude shale-oil distillation fractions from Scotland were tested by skin application in mice; the less

refined shale-oils were more highly carcinogenic to the skin than the more refined products. Heavy fractions of shale-oil from Estonia were more carcinogenic than the light fractions or the total oil when tested in mice by skin application; in contrast, in a study from Scotland, light distillation fractions of a lubricating oil induced more tumours than heavier fractions.

Comparative carcinogenicity studies in mice by skin application indicate that residual shale-oil bitumen (Estonia) was more active in inducing skin tumours than blown (oxidized) bitumen.

Commercial samples representing blends of shale-oils from Estonia induced skin tumours in mice after topical application; the carcinogenic effect increased with increasing content of crude shale-oil from high-temperature retorting. In similar experiments, commercial products containing low-temperature retorting oils did not induce skin tumours.

A pot residue of shale-oil distillation ('shale-oil coke') from the Green River Formation was carcinogenic to mouse skin after topical application in benzene; however, the same sample did not induce respiratory tumours in hamsters after intratracheal instillation.

No relevant data were available to the Working Group on the carcinogenicity in experimental animals of oil-shale retort process-waters.

All the shale-derived materials tested in short-term tests came from sources in the USA, and were therefore all from low-temperature processes.

Chromosomal aberrations were induced in bone-marrow cells of rats following administration by gavage of a suspension of raw oil shale. In-vitro tests of extracts of raw oil shale in bacteria, yeast and cultured mammalian cells gave negative results.

Preparations of spent oil shale yielded contradictory results in bacterial mutation assays and were negative in mutation assays with eukaryotic cells *in vitro* and in a chromosomal assay *in vivo*.

Preparations of shale-derived crude oils from various sources and retort processes were mutagenic in bacteria, yeast and cultured mammalian cells following metabolic or photo-induced activation. Three crude shale-oil preparations did not induce mitotic gene conversion in yeast; two others induced sister chromatid exchanges in cultured mammalian cells. Both positive and negative results were obtained in mammalian *in vivo* assays for chromosomal effects.

As compared with the corresponding crude shale-oils, preparations of hydrotreated oils showed decreased activity or were negative in various short-term tests.

A preparation of refined shale-oil was not mutagenic in bacteria.

Oil-shale retort process-waters elicited DNA damage and mutations in bacteria and in cultured mammalian cells following metabolic activation or photoactivation. They induced chromosomal aberrations in cultured mammalian cells; and induced chromosomal aberrations but not sister chromatid exchanges in mouse cells *in vivo*.

Extracts of oil-shale ash were mutagenic in bacteria both in reversion and forward mutation assays in the absence of a metabolic system. Tests with eukaryotic systems gave negative results.

5.3 Human data

The association between shale-oils and skin cancers, particularly of the scrotum, was demonstrated by analyses of 65 cases of skin cancer, including 31 of the scrotum, from the Scottish shale-oil industry. In the

UK, over 2000 cases of skin cancer ('mule-spinners' cancer) were recorded among cotton-textile workers and others exposed to lubricating oils (many of which are believed to have been shale-derived). The occupational etiology of these cases is supported by occupational mortality statistics for the UK and by an occupational comparison with fatal cases of penile cancer. In contrast, one study reported very few scrotal cancers among US cotton-textile workers employed in mills where shale-derived lubricants were not used. A cohort study of shale-oil workers in western USA found statistically significant excesses of total cancer and of colon cancer, although data on duration and time since first exposure were not available. A cohort study of shale-oil workers in Estonia found significant excesses of skin cancer, but not of cancers at other sites.

5.4 Evaluation

There is *sufficient evidence* for the carcinogenicity in experimental animals of high-temperature crude shale-oils, low-temperature crude shale-oils, fractions of high-temperature shale-oil, crude shale-oil distillation fractions, shale-oil bitumens and commercial blends of shale-oils.

There is *limited evidence* for the carcinogenicity in experimental animals of raw oil shale, spent oil shale and a residue of shale-oil distillation.

There is *sufficient evidence* that shale-oils are carcinogenic in humans.

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

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

Last updated: 20 April 1998

SOOTS

VOL.: 35 (1985) (p. 219)

5. Summary of Data Reported and Evaluation

5.1 Exposures

Humans (primarily chimney-sweeps) are exposed to chimney soots in the course of chimney maintenance. Exposures occur to a limited extent in horticultural uses and in other occupations. The general public may be exposed to the particulates emitted from chimneys when domestic heating fuels are burned.

5.2 Experimental data

Coal soot was tested in two experiments in mice by whole-body exposure, but the studies were inadequate for evaluation.

Coal-soot extracts applied to the skin of mice produced skin tumours in two studies.

A wood-soot extract applied to the skin of mice was inadequately tested. In limited studies, subcutaneous implants of wood soot in female rats produced a few local sarcomas; similar implants in the scrotal sac of rats did not produce local tumours.

An extract of fuel-oil soot was inadequately tested by application to the skin of mice.

Extracts of soot from the combustion of oil shale produced skin tumours in mice after dermal application and lung tumours in rats after intratracheal instillation. Extracts of soot from the combustion of a heating oil produced from shale-oil produced skin tumours in mice in two experiments when applied to the skin.

In one study, extracts of soot samples from domestic sources were mutagenic in *Salmonella typhimurium*. Extracts of an experimentally-derived soot were mutagenic in forward mutation assays in *S. typhimurium* and in cultured human lymphoblasts.

5.3 Human data

The carcinogenicity of soot is demonstrated by numerous case reports, dating back over 200 years, of skin cancer, particularly of the scrotum, among chimney-sweeps.

Cohort studies of mortality among chimney-sweeps in Sweden and Denmark have shown a significantly increased risk of lung cancer. Supporting evidence for an association with lung cancer was provided by two earlier epidemiological studies in the German Democratic Republic and the UK. The potentially confounding and interactive effects of smoking could not be evaluated; however, cigarette smoking is not believed to have seriously biased these estimates.

In addition to lung cancer, statistically significant excess mortality from oesophageal cancer, primary liver cancer and leukaemia was found among chimney-sweeps in one study.

5.4 Evaluation

There is *sufficient evidence* for the carcinogenicity of coal-soot extract and of oil-shale soot extract in experimental animals.

There is *limited evidence* for the carcinogenicity of implanted wood soot and for that of an extract of a soot from heating oil produced from oil shale in experimental animals.

There is *inadequate evidence* for the carcinogenicity of coal soot, of an extract of wood soot and of an extract of a fuel-oil soot in experimental animals.

There is *sufficient evidence* that soot is carcinogenic to humans.

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

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

Last updated: 20 April 1998