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HEALTH EFFECTS ASSESSMENT FOR COAL TARS

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U.S. Environmental Protection Agency
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Office of Solid Waste and Emergency Response
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PREFACE

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with coal tars. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. Pertinent toxicologic and environmental data were located through on-line literature searches of the Chemical Abstracts, TOXLINE, CANCERLINE and the CHEMFATE/DATALOG data bases. The basic literature searched supporting this document is current up to September, 1984. Secondary sources of information have also been relied upon in the preparation of this report and represent large-scale health assessment efforts that entail extensive peer and Agency review. The following Office of Health and Environmental Assessment (OHEA) sources have been extensively utilized:

U.S. EPA. 1982a. Carcinogen Assessment of Coke Oven Emissions. Office of Health and Environmental Assessment, Office of Research and Development, Washington, DC. EPA 600/6-82-003. NTIS PB 83-129551.

U.S. EPA. 1982b. Coal Tars Health Effects Assessment. Environmental Criteria and Assessment Office, Cincinnati, OH. p. 10. Internal draft.

The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data were available. Values were not derived or larger uncertainty factors were employed when the variable data were limited in scope tending to generate conservative (i.e., protective) estimates. Nevertheless, the interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed.

Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, the AIS or acceptable intake subchronic, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval (i.e., for an interval that does not constitute a significant portion of the lifespan). This type of exposure estimate has not been extensively used or rigorously defined, as previous risk assessment efforts have been primarily directed towards exposures from toxicants in ambient air or water where lifetime exposure is assumed. Animal data used for AIS estimates generally include exposures with durations of 30-90 days. Subchronic human data are rarely available. Reported exposures are usually from chronic occupational exposure situations or from reports of acute accidental exposure.

The AIC, acceptable intake chronic, is similar in concept to the ADI (acceptable daily intake). It is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan [see U.S. EPA (1980) for a discussion of this concept]. The AIC is route specific and estimates acceptable exposure for a given route with the implicit assumption that exposure by other routes is insignificant.

Composite scores (CSs) for noncarcinogens have also been calculated where data permitted. These values are used for ranking reportable quantities; the methodology for their development is explained in U.S. EPA (1983).

For compounds for which there is sufficient evidence of carcinogenicity, AIS and AIC values are not derived. For a discussion of risk assessment methodology for carcinogens refer to U.S. EPA (1980). Since cancer is a process that is not characterized by a threshold, <u>any</u> exposure contributes an increment of risk. Consequently, derivation of AIS and AIC values would be inappropriate. For carcinogens, q_1*s have been computed based on oral and inhalation data if available.

ABSTRACT

In order to place the risk assessment in proper context, the reader is referred to the preface of this document. The preface outlines limitations applicable to all documents of this serves as well as the appropriate interpretation and use of the quantitative numbers.

There are extensive data which indicated that coal tars and coal tar components are carcinogenic in experimental animals. Human data indicate that industrial exposure mixtures containing coal tar volatiles are associated with excess cancer risk.

U.S. EPA (1982a) has used epidemiological data to develop a quantitative risk assessment for coke oven workers, expressed as an incremental risk of 3.2 $(mg/kg/day)^{-1}$. Although coal tar volatiles are present as major constituents in coke oven emissions it is probable that differences exist between the composition of the oven emissions and potential exposure mixtures from coal tar products following the distillation process. It is felt that despite these reservations, exposures should be substantially similar and that the coke oven data can be used to estimate risk associated with coal tar exposure.

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Scientists from the following U.S. EPA offices provided review comments for this document series:

Environmental Criteria and Assessment Office, Cincinnati, OH
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Editorial review for the document series was provided by:

Judith Olsen and Erma Durden
Environmental Criteria and Assessment Office
Cincinnati, OH

Technical support services for the document series was provided by:

Bette Zwayer, Pat Daunt, Karen Mann and Jacky Bohanon
Environmental Criteria and Assessment Office
Cincinnati, OH

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LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
AIC	Acceptable intake chronic
AIS	Acceptable intake subchronic
BaP	Benzo(a)pyrene
CTPV	Coal tar pitch volatiles
CS	Composite score
DNA	Deoxyribonucleic acid
PAH	Polycyclic aromatic hydrocarbons
TWA	Time-weighted average

1. ENVIRONMENTAL CHEMISTRY AND FATE

Coal tar is a black, viscous liquid or semisolid substance obtained by the destructive distillation of bituminonus coal as in the production of coke. Typically, one ton of coal in a coke oven may yield ~27.5-34 ℓ of coal tar (U.S. EPA, 1982a). The physical properties of coal tar vary substantially. The specific gravity of coal tar may vary from 1.18-1.23 (Hawley, 1981). Coal tar is soluble in ether, benzene and chloroform, and is partially soluble in ethanol, acetone and methanol and only slightly soluble in water (Hawley, 1981). The typical constituents of United States coke oven-derived coal tar are the following: benzene, 0.12%; toluene, 0.25%; o-xylene, 0.04%; m-xylene, 0.07%; p-xylene, 0.03%; ethylbenzene, 0.02%; styrene, 0.02%; phenol, 0.61%; o-cresol, 0.25%; m-cresol, 0.45%; p-cresol, 0.27%; xylenols, 0.36%; high boiling tar acids, 0.83%; naphtha, 0.97%, naphthalene, 8.8%; 1-methylnaphthalene, 0.65%; 2-methylnaphthalene, 1.23%; acenaphthene, 1.05%; fluorene, 0.64%; anthracene, 0.75%; phenanthrene, 2.66%; carbazole, 0.6%; tar bases, 2.08%; and medium soft pitch, 63.5% (McNeil, 1983). The pitch contains four, five, six and seven-ring PAHs and their methylated derivatives (McNeil, 1983). The coal tar obtained through destructive distillation of coal is usually subjected to further distillation. The distillation of coke oven tar produces the following fractions: light oil, phenolic oil, naphthalene oil, wash oil, light anthracene oil, heavy anthracene oil and medium soft pitch (McNeil, 1983).

The exposure of undistilled coal tar to the ambient atmosphere is likely to contaminate the atmosphere primarily with volatile compounds such as monocyclic and PAHs, phenolic compounds and heterocyclic compounds. The fate and transport of these compounds in the atmosphere may be determined by

their chemical and photochemical reactivity and physical removal mechanisms (dry and wet deposition) in the atmosphere. In general, the fate of the most relevant compounds will be very similar to the fate of PAHs in the atmosphere.

In aquatic systems, coal tar constituents will partition into two fractions: one very small fraction will be solubilized in the water and stay as the mobile phase, and the other very large fraction will precipitate as sediment in water. The solubilized fraction containing different chemical constituents may undergo chemical and microbiological reactions, and evaporative and sorptive processes in aquatic media. The insoluble part in the sediment is expected to have a very long lifetime.

In soils, coal tar may partition itself into three fractions. Volatile constituents in coal tar may enter the atmosphere as a vapor or partially in the particulate phase. A second fraction of coal tar constituents may be solubilized by rainwater. This solubilized part, consisting of several chemicals, may undergo physical and chemical processes in soil. The third and very large fraction that may consist of the nonvolatile and insoluble part of coal tar may persist in soil for a long period of time.

2. ABSORPTION FACTORS IN HUMANS AND EXPERIMENTAL MAMMALS

2.1. ORAL

Pertinent data regarding the quantitative absorption of coal tars following exposure by the oral route were not located in the available literature.

2.2. INHALATION

Pertinent data regarding the quantitative absorption of coal tars following exposure by the inhalation route were not located in the available literature.

3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS

3.1. SUBCHRONIC

3.1.1. Oral. Pertinent data regarding the subchronic oral toxicity of coal tars were not located in the available literature.

3.1.2. Inhalation. Kinkead (1973) prepared an aerosol of coal tar in which the solids previously had been removed by centrifugation. He exposed female Sprague-Dawley yearling rats (64), male (32) and female (32) Sprague-Dawley weanling rats, male ICR mice (50), and male CAF-1 mice (50) continuously for 90 days at concentrations of 0.2, 2.0 and 10 mg/m^3. In addition, 80 yearling female Sprague-Dawley rats, 9 weanling rats of each sex, 25 male CAF-1 mice, 25 male ICR mice, 24 female New Zealand white rabbits and 100 male Syrian golden hamsters were exposed continuously for 90 days at 20 mg/m^3. Greater than 95% of the aerosol droplets were $\leq$5 μm in diameter.

Considerable mortality among exposed animals was encountered in this study, which the authors attributed to debilitation causing greater susceptibility to infections. However, cumulative animal mortality was proportional to exposure concentration. In all species tested, there was a marked effect of exposure on body weight growth curves. Animals either lost weight (mice, hamsters, rabbits) or grew at a slower rate than nonexposed controls (rats). Even the lowest exposure concentration, 0.2 mg/m^3, produced some adverse effects on body weight gain, with the most striking effect observed in male CAF-1 mice whose body weight decreased from 30 g to 25 g during the 90 days in the 0.2 mg/m^3 group. Following the termination of exposure, the inhibitory effect of coal tar aerosol on growth was still evident for at least 7 months in most species.

Kinkead (1973) conducted a subsequent study, described by McConnell and Specht (1973), employing a coal tar in which the solid particles and light oil fractions were retained in the experimental aerosol. Sprague-Dawley rats, New Zealand white rabbits, JAX mice, and Syrian golden hamsters (numbers not specified) were exposed continuously for 90 days to the coal tar aerosol at a concentration of 10 mg/m³. In addition, 150 CF-1 mice were exposed to the aerosol and serially sacrificed for histopathologic analysis. Among exposed rats and hamsters, McConnell and Specht (1973) described three significant lesions occurring at the termination of exposure. These were phagocytized coal tar pigment in alveolar macrophages and in the peribronchial lymphoid tissue; hepatic and renal hemosiderosis, which disappeared by 100 days postexposure; and mild central lobular necrosis in the liver. Among mice sacrificed 99 days postexposure, moderate pigmentation of alveolar macrophages was observed in 14/15 CF-1 mice, but in only 1/13 exposed JAX mice.

3.2. CHRONIC

3.2.1. Oral. Pertinent data regarding the chronic oral toxicity of coal tars were not located in the available literature.

3.2.2. Inhalation. MacEwen et al. (1976) prepared a composite coal tar mixture from multiple coking ovens around the greater Pittsburg area. Coal tar samples were blended together with a 20% by volume amount of BTX (benzene, toluene, xylene) fraction of coke oven distillate. This material was believed to be more representative of that inhaled by workers on top of coke ovens. Female (75) ICR-KF-1 mice, female (50) CAF-1-JAX mice, male (40) and female (40) weanling Sprague-Dawley rats, New Zealand white rabbits (18), and male (5) and female (9) Macaca mulatta monkeys were exposed to a coal tar aerosol at 10 mg/m³, 6 hours daily, 5 days/week, for 18 months.

Animals were held for an additional 6-month observation period following termination of exposure. Development of skin tumors in mice precluded assessment of other systemic effects. A significant inhibition of body growth rate was observed for both male and female rats after 4 months and for rabbits by the end of the 1st month. Monkeys showed no significant inhibition of growth rate. In this study, 16/18 test rabbits and 6 control rabbits died during the test period.

3.3. TERATOGENICITY AND OTHER REPRODUCTIVE EFFECTS

3.3.1. Oral. Pertinent data regarding the teratogenicity of coal tars following oral exposure were not located in the available literature.

3.3.2. Inhalation. Pertinent data regarding the teratogenicity of coal tars following inhalation exposure were not located in the available literature.

3.4. TOXICANT INTERACTIONS

Pertinent quantative data regarding the interactions of coal tars with other compounds were not located in the available literature.

4. CARCINOGENICITY

4.1. HUMAN DATA

4.1.1. Oral. Pertinent data regarding the carcinogenicity of orally administered coal tars were not located in the available literature.

4.1.2. Inhalation. Studies of workers exposed to "tarry fumes" in the coal gas (Doll et al. 1972, Kawai et al., 1967), steel (Redmond et al., 1972) and aluminum reduction industries (Gibbs and Horowitz, 1979; Milham, 1979) have supported an occupational association with lung, skin and hematopoietic cancers. Tarry fumes, however, were not coal tar per se. Only in the studies of roofers (Hammond et al., 1976) and coke oven workers (Redmond et al., 1979) was exposure specifically to coal tars or coal tar volatiles.

Hammond et al. (1976) observed increases in both lung and skin cancer in roofers with >20 years of work experience, and increases in skin cancer in a group with 9-19 years of work experience. A single measurement reported that workers inhaled BaP, a polyaromatic hydrocarbon in coal tar, at levels ranging from undetectable to 153 μg during a 7-hour work shift. The corresponding levels of coal tar were not reported.

Redmond et al. (1979), in a final report of an extensive epidemiologic study of coke oven workers, summarized the results of interim reports by Lloyd (1971), Redmond et al. (1972, 1976) and Mazumdar et al. (1975). Coke oven workers (2552 employed in Allegheny County) with >5 years of exposure had an increased relative risk of lung cancer (2.63) and kidney cancer (3.55). Mazumdar et al. (1975) calculated cumulative exposures to CTPV for the subgroups of the cohort and suggested that exposures <200 mg/m³/month would not result in an increased risk of cancer. Using CTPV estimates, there was a dose-related increase in lung cancer among nonwhite workers, while for white workers, the increased risk appeared not to be related to

CTPV. The increased rate/1000 of lung cancer in nonwhite males was 4.0, 12.9, 24.9 and 54.6 for groups exposed to <199, 200-499, 500-699 and >700 mg/m³-month. In addition, Redmond et al. (1976) reported that side oven workers also had an increase in risk of cancer, although these workers are exposed to much lower levels of CTPV. In 10 plants studied outside Allegheny county, there was no difference between the races, although similar excesses in risk of lung cancer were reported. The excess risk of kidney cancer was not apparent in the non-Allegheny county plants.

4.2. BIOASSAYS

4.2.1. Oral. Pertinent data regarding the carcinogenicity of orally administered coal tar to experimental animals were not located in the available literature.

4.2.2. Inhalation. The first experimental demonstration of cancer produced by inhalation of coke oven-derived material was by Horton et al. (1963). They used the same coal tar sample, taken from a coke oven, that had previously been reported to produce tumors on the skin of mice (Horton, 1961). The tar was characterized as having a BaP content of 0.71%, a high "tar acid" content (5.1%), and a low content of benzene insolubles (2.74%) in comparison with other coal tars. Among 33 C3H mice inhaling a coal tar aerosol at 300 mg/m³ for a total of 40-100 hours over a 13- to 35-week period, 5 developed squamous cell tumors in the periphery of the lung during the 36-week postexposure observation period. One of the tumors was an invasive squamous cell carcinoma. The control mice were killed at 82 weeks of age and none of the 30 animals had tumors of the lung.

Since the development of cancers by the inhalation of xenobiotics was, at that time, a rarely observed phenomenon in experimental animals, further inhalation studies were pursued. Tye and Stemmer (1967) employed two coke

oven-derived tars in their study. The first was a tar sample of the same composition as that used in previous studies, as described by Horton et al. (1963). The second coal tar was high in toluene-insoluble material (17.8%), low in tar acids (1.4%) and had a BaP content of 1.1%. Both tars were separated into phenolic and nonphenolic fractions, and various combinations were used to produce aerosols for inhalation exposure to male C3H/HeJ mice. Groups of 50 mice were exposed for 2 hours, three times weekly, for 55 weeks. The concentration of coal tar aerosol was 200 mg/m³ for the first 8 weeks and 120 mg/m³ for the remaining 47 weeks. Animals were examined after spontaneous death or after scheduled intervals. During the study, three mice from each group were killed at 4 weeks, five mice from each group were killed at 31 weeks and all surviving animals were killed at 55 weeks. Their findings included the observation of increases in squamous metaplasia, intrabronchial and alveolar adenomas, and adenocarcinomas. The most prominent lesions in exposed animals were intrabronchial adenoma and adenocarcinoma. None of the control animals developed lung tumors. The authors concluded that the presence of phenols in the coal tar exerted a cocarcinogenic effect together with polynuclear aromatic hydrocarbons, possibly caused by an irritant effect. No direct evidence was provided, however, to explain the role of the phenolic fraction in tumor development, and the tumorigenic potential of the phenolic fraction alone was not assessed.

In later studies, Kinkead (1973) exposed mice, rats, hamsters and rabbits to a coke oven-derived coal tar aerosol at 20, 10, 2 and 0.2 mg/m³ continuously for 90 days and observed them until death. The aerosol consisted of a benzene extract of coal tar from which the solids were removed by centrifugation, and before generating the aerosol, the added benzene and the light oil fractions were also removed. Several types of tumors developed in the exposed animals, as described by McConnell and Specht (1973).

An independent analysis of the pathologic data from this study was conducted by the National Institute for Occupational Safety and Health (NIOSH, 1978), and the findings are summarized in Table 4-1. No data were available for the 20 mg/m³ concentration. From the NIOSH (1978) analysis of the Kinkead (1973) data, it was concluded that the coal tar aerosol produced a dose-related incidence of lung tumors in mice. In addition, the increased incidence of splenic lymphosarcomas in mice and lung tumors in rats was also felt to be related to the coal tar exposure. There is some disagreement between the exposure data of Kinkead (1973) and the pathologic report in NIOSH (1978). Kinkead (1973) stated that rabbits and hamsters were exposed only at 20 mg/m³, while the NIOSH (1978) report gives data for these species at lower exposure concentrations. These conflicting points make it unclear as to whether the animals examined in NIOSH (1978) were the same animals exposed by Kinkead (1973) and examined by McConnell and Specht (1973).

A follow-up long-term study reported by MacEwen and Vernot (1976) and MacEwen et al. (1976) involved the exposure of female ICR CF-1 (75) and CAF-1 JAX (50) mice, male (40) and female (40) weanling Sprague-Dawley rats, 18 New Zealand albino female rabbits, and male (5) and female (9) Macaca mulatta monkeys to a coke oven-derived coal tar aerosol. Animals were exposed to a chamber concentration of 10 mg/m³, 6 hours daily, for up to 18 months. Histopathologic data for mice and rats have been reported. The rabbits and monkeys were not sacrificed and examined in this study, but were maintained for long-term, postexposure observation.

Various tumors were found in mice, although principally alveolargenic carcinomas of the lung were observed (26/61 in ICR CF-1 mice and 27/50 in CAF-1 JAX mice as compared with 3/68 and 8/48 in the respective controls).

TABLE 4-1

Tumors from Exposure to Airborne Coal Tar in Four Animal Species[a]

Species[b]	Tumor	Tumor Incidence (%)			
		0.00 mg/m³	0.20 mg/m³	2.00 mg/m³	10.00 mg/m³
Mice	skin	3	1	1	6
	lung	30	39	58	77
	spleen	5	20	5	14
	kidney	1	3	0	0
	liver	9	4	11	0
	urocyst	0	0	3	0
Rats	skin	10	6	3	0
	lung	4	3	10	18
	spleen	8	4	4	8
	kidney	1	0	0	6
	liver	1	0	0	3
Hamsters	skin	0.7	0	0	4
	lung	0.6	3	0	4
	spleen	0.7	0	0	4
	kidney	2	1	0	0
	liver	0.6	0	0	2
	adrenals	27	0	57	17
Rabbits	skin	0	0	0	12
	lung	0	0	0	0
	spleen	0	0	0	0
	kidney	0	0	0	0
	liver	0	0	0	0
	urocyst	0	0	0	0

[a]Source: NIOSH, 1978

[b]Results are based on data from a total of 63 rabbits, 376 hamsters, 498 rats and 563 mice.

The incidence of squamous cell carcinomas in the lungs of exposed rats was 100% in males and 82% in females. None of the control rats developed lung tumors.

4.3. OTHER RELEVANT DATA

Horton (1961) demonstrated that twice weekly dermal application of coal tar to the shaved skin of mice resulted in increases in dermal tumors. The latency period ranged from 7-15 months depending on the source of the tar. Human case reports dating back to 1885 (NIOSH, 1978) have also supported an association between occupational exposure to coal tars and the development of skin cancer.

Although coal tar contains individual components, primarily PAHs, which have been extensively evaluated and found to be mutagenic (Fishbein, 1976; U.S. EPA, 1982a), few data are available concerning the mutagenicity of coal tar mixtures as a whole. Brat et al. (1982) tested roofing tar pot emissions for genotoxicity in a number of systems using mammalian cells. No details were given concerning sample collection methods or sample composition. The tar emission samples gave positive results in a HPC/DNA repair assay using primary hepatocyte cultures, but not the ARL/HGPRT mutagenesis assay. Coke oven emissions were also tested in the DNA repair assay and yielded greater DNA repair activity than the tar sample (106 grains/nucleus vs. 37.7 grains/nucleus) when both were tested at doses of 10^{-1} mg/mℓ.

More extensive data are available concerning the mutagenicity of coke oven emission; these are reviewed in U.S. EPA (1982a). Extracts of coke oven door emissions have been shown to be mutagenic in bacteria. Extracts of samples from coke oven collecting mains have been shown to cause mutations in bacteria and mammalian cells in culture. These studies are

relevant to coal tars in that a large proportion of the identified mutagenic compounds in coke oven emissions are PAHs, presumably from coal tar volatilization.

BaP is the marker PAH which has been most extensively monitored. BaP was detected in "air contaminated with coal tar pitch fumes" at a level of 400 μg/1000 m^3 (1800 μg/g particulates) (Sawicki et al., 1965). Worker-exposure concentrations of BaP in coal and pitch coking plants have been measured as 0.3-35 mg/1000 m^3 (Fishbein, 1976). Emissions from coke ovens in the USSR have been reported to contain 120-1700 μg BaP/g particulates (von Lehmden et al., 1965).

4.4. WEIGHT OF EVIDENCE

Studies in rats and mice indicate that inhalation exposure to coal tars results in increased incidence of lung tumors (Tye and Stemmer, 1967; Kinkead, 1973; McConnell and Specht, 1973; MacEwen et al., 1976). In epidemiologic studies of roofers exposed to coal tars, there was an elevated risk of both skin and lung cancer (Hammond et al., 1976), while coke oven workers exposed to coal tars had an elevated risk of lung and kidney cancers (Redmond et al., 1979). The evidence for carcinogenicity for coal tars in animals is "sufficient," and the evidence for carcinogenicity in humans is "limited," bordering on "sufficient," using the criteria for weight of evidence proposed by the Carcinogen Assessment Group of the U.S. EPA (Federal Register, 1984). Coal tars as a chemical class are, therefore, most appropriately classified as a Group B1 chemical.

5. REGULATORY STANDARDS AND CRITERIA

NIOSH (1978) has recommended a 10-hour TWA concentration for coal tars (cyclohexane-extractable fraction) of 0.1 mg/m^3. The ACGIH (1980) considers coal tar pitch volatiles to be a recognized carcinogen and suggests a TWA of 0.2 mg/m^3.

6. RISK ASSESSMENT

6.1. ACCEPTABLE INTAKE SUBCHRONIC (AIS)

Coal tars are a class of chemicals that are known to be animal carcinogens and for which a carcinogenic potency factor has been estimated by the Carcinogen Assessment Group from human epidemiologic data. It is, therefore, inappropriate to calculate an oral or inhalation AIS for coal tars.

6.2. ACCEPTABLE INTAKE CHRONIC (AIC)

Coal tars are a class of chemicals that are known to be animal carcinogens and for which a carcinogenic potency factor has been estimated by the Carcinogen Assessment Group from human epidemiologic data. It is, therefore, inappropriate to calculate an oral or inhalation AIC for coal tars.

6.3. CARCINOGENIC POTENCY (q_1*)

6.3.1. Oral. Pertinent data regarding the carcinogenicity of coal tars following oral exposure were not available for calculating a carcinogenicity potency factor, q_1*.

6.3.2. Inhalation. The animal studies available also do not provide sufficient data to derive a q_1* using the linearized multistage model. The subchronic study by Kinkead (1973) reports dose-related increases in lung tumors in mice exposed to coal tar aerosols of between 0.2 and 10 mg/m³. The number of animals per group and number of tumor-bearing animals were not provided in this report, which precludes the derivation of a q_1*. The chronic study of MacEwen et al. (1976) employed only one exposure level and male rats at this level had 100% tumor incidence. Since male rats appear to be the most sensitive species, this group should be used in risk assessment; however, a q_1* cannot be derived from data when only a single exposure is used and 100% tumor incidence is reported.

The U.S. EPA (1982a) Cancer Assessment Group (CAG) has used the epidemiologic data from the study of Redmond et al. (1979) along with the exposure data developed by Mazumdar et al. (1975) to calculate a carcinogenic potency factor for coke oven emissions. In the analysis of the study of Redmond et al. (1979), the CAG grouped the nonwhite coke oven workers into four age and exposure groups as summarized in Table 6-1. Using these data, CAG calculated a unit risk of 0.9×10^{-3} for lifetime exposure to 1 $\mu g/m^3$ of coal tars. This incremental risk can also be expressed as 3.2 $(mg/kg/day)^{-1}$ by assuming a 70 kg man breathes 20 m^3 of air per day and that complete absorption occurs.

Coal tar is a by-product of bituminous coal distillation in coke production. Many components of coke oven emissions are present in coal tar, especially a wide variety of PAHs and their methylated derivatives. However, it might be suspected that the exposure mixture would be somewhat different during exposure to oven emissions as compared to exposure to the coal tar product after the coke oven distillation process is completed.

Good epidemiological data for exposure to coal tars *per se* are not available for risk assessment purposes. Despite uncertainties concerning potential differences in exposure mixture composition, it is felt that exposures should be substantially similar and that the coke oven data can be used to estimate risk associated with coal tar exposure.

TABLE 6-1

Average Dose Levels, Lung Cancer Deaths, Person-Years Observation for Risk, and Rates Used to Estimate Dose-Response Relationships, by Model Used to Define Dose and by Age at Entry to Study[a]

Nonwhite Workers Only

Dose Model	Age at Entry	Dose Range[b]	Average Dose[b]	Lung Cancer Deaths	Person-Years	Yearly Rate per 100,000
Zero lag	25-34	nonoven	0	3	22,405	13.4
		0-99	36.6	1	3,202	31.2
		100-199	149.0	0	2,685	0
		200-299	249.5	3	3,030	99.0
		300+	386.8	4	3,062	130.6
	35-44	nonoven	0	4	16,227	34.7
		0-149	67.8	0	2,388	0
		150-299	226.8	2	2,976	67.2
		300-499	366.4	3	2,727	110.0
		450+	590.9	5	2,027	246.7
	45-54	nonoven	0	17	11,306	150.4
		0-249	147.7	1	1,527	65.5
		250-449	353.2	4	1,706	234.5
		450-699	564.5	4	1,545	258.9
		700+	885.5	8	1,330	601.5
	55-69	nonoven	0	4	5,820	68.7
		0-249	153.3	1	491	203.7
		250-449	333.4	1	596	167.8
		450-749	600.9	4	716	558.7
		750+	972.4	10	450	2222.2
5-year lag	25-34	nonoven	0	3	22,405	13.4
		0-49	19.0	1	2,567	39.0
		50-149	102.3	0	3,985	0
		150-199	172.2	4	2,822	141.7
		200+	265.0	3	2,605	115.2
	35-44	nonoven	0	4	16,227	24.7
		0-99	44.6	0	2,254	0
		100-199	149.0	2	2,952	67.8
		200-349	259.3	4	3,072	130.2
		350+	462.1	4	1,840	217.4

TABLE 6-1 (cont.)

Dose Model	Age at Entry	Dose Range[b]	Average Dose[b]	Lung Cancer Deaths	Person-Years	Yearly Rate per 100,000
		Nonwhite Workers Only				
5-year lag (cont.)	45-54	nonoven	0	17	11,306	150.4
		0-199	112.4	1	1,828	54.7
		200-349	268.9	4	1,397	286.3
		350-599	460.3	2	1,573	127.2
		600+	763.0	10	1,310	763.4
	55-69	nonoven	0	4	5,820	68.7
		0-199	115.4	1	516	193.8
		200-399	293.0	2	665	300.8
		400-649	535.5	4	567	705.5
		650+	851.7	9	503	1789.3
10-year lag	25-34	nonoven	0	3	22,405	13.4
		0-49	18.8	1	3,964	25.2
		50-99	76.8	3	3,371	89.0
		100-149	120.6	1	3,226	31.0
		150+	193.1	3	1,418	211.6
	35-44	nonoven	0	4	16,227	24.7
		0-99	46.7	1	3,956	25.3
		100-149	123.5	1	2,283	43.8
		150-249	191.5	4	2,360	169.5
		250+	353.0	4	1,519	263.3
	45-54	nonoven	0	17	11,306	150.4
		0-149	76.4	2	2,308	98.5
		150-249	193.0	3	1,308	229.4
		250-499	362.4	2	1,607	124.5
		500+	641.8	10	1,162	860.6
	55-69	nonoven	0	4	5,820	68.7
		0-149	95.3	1	660	151.5
		150-299	299.8	1	576	173.6
		300-549	439.4	5	505	990.1
		550+	729.7	9	512	1757.8

TABLE 6-1 (cont.)

Dose Model	Age at Entry	Dose Range[b]	Average Dose[b]	Lung Cancer Deaths	Person-Years	Yearly Rate per 100,000
		Nonwhite Workers Only				
15-year lag	25-34	nonoven	0	3	22,405	13.4
		0-9	1.5	2	4,457	44.9
		10-39	27.1	2	2,303	86.8
		40-69	55.3	0	3,098	0
		70+	100.3	4	2,121	188.6
	35-44	nonoven	0	4	16,227	24.7
		0-9	1.6	1	2,374	42.1
		10-69	43.3	2	3,014	66.4
		70-129	97.1	2	2,601	76.9
		130+	197.7	5	2,129	234.9
	45-54	nonoven	0	17	11,306	150.4
		0-69	24.4	2	1,623	123.2
		70-149	104.7	3	1,612	186.1
		150-349	222.7	2	1,512	132.3
		350+	486.6	10	1,361	734.8
	55-69	nonoven	0	4	5,820	68.7
		0-69	48.7	1	602	166.1
		90-199	141.3	2	609	328.4
		200-429	331.4	6	579	1036.3
		430+	604.9	7	461	1518.4

[a]Source: U.S. EPA, 1982a

[b]mg/m^3-months exposure to CTPV

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APPENDIX

Summary Table for Coal Tars[a]

	Species	Experimental Dose/Exposure	Effect	q_1*
Inhalation				
AIS				ND
AIC				ND
Carcinogenic potency	humans	0-700+ mg/m³-month	lung tumors	3.2[b] $(mg/kg/day)^{-1}$
Oral				
AIS				ND
AIC				ND
Carcinogenic potency				ND

[a]Source: U.S. EPA, 1982a

[b]This value is not a q_1*, but instead an incremental risk value expressed in equivalent units and based on human epidemiological data from coke oven workers.

ND = Not derived