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Hazard Review

Health Effects of Occupational Exposure to Asphalt

U.S. Department of Health and Human Services
Public Health Service
Centers for Disease Control and Prevention
National Institute for Occupational Safety and Health

HAZARD REVIEW

HEALTH EFFECTS OF OCCUPATIONAL EXPOSURE TO ASPHALT

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FOREWORD

As part of its mandate to ”provide a safe and healthful workplace for working women and men,” the National Institute for Occupational Safety and Health (NIOSH) critically evaluates the scientific data on potentially hazardous occupational exposures or work conditions and makes recommendations that address measures for minimizing the risk from the hazard. This document, *Hazard Review: Health Effects of Occupational Exposures to Asphalt*, is an evaluation of the health effects and other relevant data that have become available since publication of the 1977 NIOSH document *Criteria for a Recommended Standard: Occupational Exposure to Asphalt Fumes*. It includes an assessment of chemistry, health, and exposure data from studies in animals and humans exposed to raw asphalt, paving and roofing asphalt fume condensates, and asphalt-based paints. Most important, the document serves as a basis for identifying future research to reduce occupational exposures to asphalt.

The complex chemical composition of asphalt makes it difficult to identify the specific component(s) responsible for adverse health effects observed in exposed workers. Known carcinogens have been found in asphalt fumes generated at worksites. Observations of acute irritation in workers from airborne and dermal exposures to asphalt fumes and aerosols and the potential for chronic health effects, including cancer, warrant continued diligence in the control of exposures.

NIOSH and its labor and industry partners are making great strides in reducing worker exposures to paving and roofing asphalt fumes. The partnership has succeeded because the partners set aside key differences to focus on the development of engineering and other control measures to reduce workplace exposures. A major success occurred when 100 percent of the asphalt paving industry voluntarily agreed to install new controls on all new highway pavers produced after July 1997—effectively reducing asphalt fume exposure. Other aspects of the partnership have encouraged collaborative laboratory and field research and the development of communication materials for workers and contractors on methods for reducing workplace exposures. Representatives of industry, labor, government, and academia met in Cincinnati, OH, on September 11 and 12, 2000, and identified research needed to assess completely the health risks associated with exposure to asphalt. Through these and other efforts of the partnership, effective workplace measures can be implemented to reduce worker exposure to asphalt fumes.

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EXECUTIVE SUMMARY

In 1977, the National Institute for Occupational Safety and Health (NIOSH) reviewed the available data on the health effects of occupational exposure to asphalt and asphalt fumes. NIOSH determined the principal adverse health effects to be irritation of the serous membranes of the conjunctivae and mucous membranes of the respiratory tract. NIOSH also acknowledged that evidence from animal studies indicated that asphalt left on the skin for long periods of time could result in local carcinomas but that no comparable reports of these effects existed for humans. On the basis of this evidence, NIOSH recommended an exposure limit (REL) for asphalt fumes of 5 milligrams per cubic meter of air (5 mg/m^3) measured as total particulates during any 15-minute period. In testimony to the Department of Labor in 1988, NIOSH recommended that asphalt fumes also be considered a potential occupational carcinogen. Since then, additional data have become available from studies of animals and humans exposed to asphalt, paving and roofing asphalt fume condensates, and asphalt-based paints. This document evaluates the health effects data that have become available since publication of the 1977 NIOSH criteria document; it also assesses exposures associated with occupations that involve the use of roofing and paving asphalts and asphalt-based paints.

Asphalt is a dark brown to black, cementlike semisolid or solid produced by the nondestructive distillation of crude oil during petroleum refining. The three major types of asphalt products are paving asphalts, roofing asphalts, and asphalt-based paints. Performance specifications—not chemical composition—direct the type of asphalt produced. Most of the asphalt produced in the United States is used in paving and roofing operations. Only about 1% is used for waterproofing, damp-proofing, insulation, paints, or other activities and products. Approximately 300,000 workers are employed at hot-mix asphalt facilities and paving sites; an estimated 50,000 workers are employed in asphalt roofing operations; and about 1,500 to 2,000 workers are exposed to asphalt fumes in approximately 100 roofing manufacturing plants.

The exact chemical composition of asphalt depends on the chemical complexity of the original crude petroleum and the manufacturing processes. The proportions of the chemicals that constitute asphalt (mainly aliphatic compounds, cyclic alkanes, aromatic hydrocarbons, and heterocyclic compounds containing nitrogen, oxygen, and sulfur atoms) can vary because of significant differences in crude petroleum from various oil fields and even from various locations within the same oil field. Further analysis of the chemical data indicates that paving and roofing asphalts are qualitatively and quantitatively different; therefore, the vapors and fumes from these asphalt products may also be different. Other factors that increase the variability of asphalt vapors and fumes include temperature and mixing during the manufacturing process, and temperature and extent of mixing during laboratory generation or field operations. Studies indicate that the composition of asphalt fumes generated in the laboratory may differ qualitatively and quantitatively from asphalt fumes generated during field operations. However, one study showed that it is possible to generate asphalt fumes in the laboratory that are representative of field fumes.

Data are limited regarding the presence of carcinogens in asphalt fumes generated at U.S. worksites. The occasional detection of benzo(a)pyrene, B(a)P, in asphalt fumes generated at worksites as well as the more frequent detection of B(a)P and other carcinogenic polycyclic aromatic compounds in laboratory-generated asphalt fumes indicate that under some conditions, known carcinogens are

likely to be present. Moreover, asphalt fumes generated at high temperatures are probably more likely to generate carcinogenic polycyclic aromatic hydrocarbons (PAHs) than fumes generated at lower temperatures.

Studies of the acute toxic effects of asphalt fume exposures in workers have repeatedly reported irritant symptoms of the serous membranes of the conjunctivae (eye irritation) and the mucous membranes of the upper respiratory tract (nasal and throat irritation). These health effects are best described in asphalt road pavers and typically appear to be mild in severity and transient in nature. Similar symptoms were also reported in workers exposed to asphalt fumes during the manufacture of asphalt roofing shingles and fluorescent lights, the insulation of cables, and exposure to a malfunctioning light fixture in an office environment. Workers employed in five segments of the asphalt industry (hot-mix plants, terminals, roofing, paving, and roofing manufacturing) experienced mild transient symptoms of nasal and throat irritation, headache, and coughing. In addition to mucosal irritation, workers with differing occupational exposures to asphalt fumes (e.g., paving operations, insulation of cables, and manufacturing of fluorescent light fixtures) also reported skin irritation, pruritus, rashes, nausea, stomach pain, decreased appetite, headaches, and fatigue. Such nonspecific symptoms require further investigation to clarify and establish the nature of causal relationships with asphalt fume exposure.

Results from recent studies indicated that some workers involved in asphalt paving operations experienced lower respiratory tract symptoms (e.g., coughing, wheezing, and shortness of breath) and pulmonary function changes. Irritant symptoms were noted in workers involved in open-air paving operations whose average personal exposures were generally below 1.0 mg/m^3 total particulates and 0.3 mg/m^3 benzene-soluble particulates calculated as a full-shift time-weighted average (TWA). Although an exposure-response relationship has not yet been established in these studies, the identification of health effects related to higher mean personal exposures during underground asphalt paving[*] indicates that such a relationship may exist. Bronchitis that is possibly related to lower respiratory tract irritation has also been reported among asphalt workers and highway maintenance workers; however, the data are insufficient to conclude that the bronchitis was caused by occupational exposure to asphalt fumes.

A recent meta-analysis of epidemiologic studies of roofers indicates an excess of lung cancer among roofers, but it is uncertain whether this excess is related to asphalt and/or to carcinogens such as coal tar or asbestos. Data from studies in animals and *in vitro* assays indicate that laboratory-generated roofing asphalt fume condensates are genotoxic and produce skin tumors in mice. Known carcinogenic PAHs have been identified in roofing asphalt fumes.

In contrast to the studies of roofers, epidemiologic studies of pavers exposed to asphalt fumes have yielded contradictory results regarding lung cancer. Although some of the studies reported an elevated risk for lung cancer among pavers exposed to asphalt, design limitations of these studies precluded any strong conclusions. Confounders included smoking and coexposure to coal tar and other potential lung carcinogens (e.g., diesel exhaust, silica, and asbestos). Furthermore, a recently

[*]Total particulate or benzene-soluble particulate measurements were up to 10 times higher than measurements taken during open-air paving, but they were still below 2.2 mg/m^3.

conducted meta-analysis of these studies failed to find overall evidence for a lung cancer risk among pavers exposed to asphalt. However, carcinogenic PAHs have been detected in asphalt paving fumes—although at lower concentrations than those found in fumes from roofing asphalt. No published data examine the carcinogenic potential of paving asphalt fumes or fume condensates in animals.

A few studies reported an association between cancer at sites other than the lungs (e.g., bladder, kidneys, brain, and liver) with occupations having potential exposure to asphalt. Since the interpretation of these findings is limited by the study designs and the lack of good exposure data and consistent findings, no association can be made at this time. Further confirmation is needed by studies with better control of confounding variables and better identification of asphalt exposures.

Conflicting results were obtained when raw roofing asphalts were applied dermally to mice. In one study, the raw roofing asphalt was weakly carcinogenic and caused malignant skin tumors in mice. In the other study, the raw roofing asphalt was not carcinogenic. Available data also indicate that several formulations of asphalt-based paints cause benign and malignant skin tumors in mice. However, these paints were not mutagenic in the Ames *Salmonella* mutagenicity assay, either with or without metabolic activation. Several other asphalt-based paints caused the formation of DNA adducts in the skin and lungs of treated mice and in fetal and adult human skin cultures.

Conclusions

In this hazard review, NIOSH has evaluated the scientific evidence concerning the potential health effects of occupational exposure to asphalt. On the basis of available data from studies in animals and humans, as well as in *in vitro* studies, NIOSH concludes the following about the acute health effects of asphalt exposure:

- The findings of this hazard review continue to support the assessment of the 1977 NIOSH criteria document on asphalt fumes, which associated exposure to asphalt fumes from roofing, paving, and other uses of asphalt with irritation of the eyes, nose, and throat. Furthermore, in studies conducted since the publication of the 1977 criteria document, these symptoms have also been noted among workers exposed to asphalt fumes at geometric mean concentrations generally below 1 mg/m^3 total particulates and 0.3 mg/m^3 benzene-soluble or carbon disulfide-soluble particulates, calculated as a full-shift TWA. Recent studies also report evidence of acute lower respiratory tract symptoms among workers exposed to asphalt fumes. These data are currently being further analyzed to assess the relationship between lower respiratory tract symptoms and asphalt fume exposure. The available data on chronic pulmonary effects (such as bronchitis) are insufficient to support an association with asphalt fume exposures.

In 1988, NIOSH recommended to OSHA that asphalt fumes be considered a potential occupational carcinogen based on the results of an animal study in which laboratory-generated roofing asphalt fume condensates induced malignant skin tumors in mice. Since then, investigators have described differences in chemical composition, physical characteristics, and biological activity between asphalt fumes collected in the field and those generated in the laboratory. The relevance of these differences in ascribing adverse health effects in humans is unknown. Data from studies in humans indicate that

some workers exposed to asphalt fumes are at an elevated risk of lung cancer; however, it is uncertain whether this excess is related to asphalt and/or other carcinogens in the workplace. Although carcinogenic PAHs have been identified in asphalt fumes at various worksites, the measured concentrations and the frequency of their occurrence have been low.

Based on evaluation of these data, the following conclusions were drawn regarding the carcinogenicity of asphalt under several conditions of use:

- Data regarding the potential carcinogenicity of paving asphalt fumes in humans are limited. Only one study identified B(a)P in field fumes, but it was unclear whether paving asphalt fumes were the source of the B(a)P. Chrysene has been identified only in laboratory-generated paving asphalt fumes. The available data from studies in humans have not provided consistent evidence of carcinogenic effects in workers exposed to asphalt fumes during paving operations. No animal studies have examined the carcinogenic potential of either field- or laboratory-generated samples of paving asphalt fume condensates. Although genotoxicity assays (but no carcinogenicity assays) using laboratory-generated and field-generated (storage tank paving asphalt) fumes have been conducted, only the laboratory-generated fumes were genotoxic. Therefore, NIOSH concludes that the collective data currently available from studies on paving asphalt provide insufficient evidence for an association between lung cancer and exposure to asphalt fumes during paving. The available data, however, do not preclude a carcinogenic risk from asphalt fumes generated during paving operations.

- The results from epidemiologic studies indicate that roofers are at an increased risk of lung cancer, but it is uncertain whether this increase can be attributed to asphalt and/or to other exposures such as coal tar or asbestos. Data from experimental studies in animals and cultured mammalian cells indicate that laboratory-generated roofing asphalt fume condensates are genotoxic and cause skin tumors in mice when applied dermally. Furthermore, a known carcinogen, B(a)P, was detected in field-generated roofing fumes. The collective health and exposure data provide sufficient evidence for NIOSH to conclude that roofing asphalt fumes are a potential occupational carcinogen.

- The available data indicate that although not all asphalt-based paint formulations may exert genotoxicity, some are genotoxic and carcinogenic in animals. No published data examine the carcinogenic potential of asphalt-based paints in humans, but NIOSH concludes that asphalt-based paints are potential occupational carcinogens.

Current data are considered insufficient for quantifying the acute and chronic health risks of exposure to asphalt, asphalt-based paint, or asphalt fumes and vapors. However, data from at least two studies of acute effects are currently being evaluated to determine their usefulness in deriving an REL. Additional studies of workers exposed to asphalt fumes, vapors, and aerosols (e.g., during paving, roofing, and painting operations) are needed to better characterize exposures and to evaluate the risk of chronic disease, including lung cancer. Also required are experimental animal studies that use laboratory generation methods to produce fumes and vapors representative of asphalt roofing and paving operations. Until the results of these studies become available, NIOSH recommends minimizing possible acute or chronic health effects from exposure to asphalt, asphalt fumes and

vapors, and asphalt-based paints by adhering to the current NIOSH REL of 5 mg/m^3 during any 15-min period and by implementing the following practices:

- Prevent dermal exposure.
- Keep the application temperature of heated asphalt as low as possible.
- Use engineering controls and good work practices at all work sites to minimize worker exposure to asphalt fumes and asphalt-based paint aerosols.
- Use appropriate respiratory protection (see Appendix C).

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SELECTED ABBREVIATIONS

AC	asphalt cement
AI	Asphalt Institute
AREC	Asphalt Roofing Environmental Council
ARMA	Asphalt Roofing Manufacturers' Association
ASTM	American Society for Testing and Materials
B(a)P	benzo(a)pyrene
CAS	Chemical Abstracts Service
CI	confidence interval
DNA	deoxyribonucleic acid
FHWA	Federal Highway Administration
GC/FID	gas chromatography with flame ionization detector
GC/MS	gas chromatography/mass spectrometry
GM	geometric mean
HMA	hot-mix asphalt
HMW	highway maintenance workers
HPLC	high-performance liquid chromatography
IARC	International Agency for Research on Cancer
LC	liquid chromatography
NAPA	National Asphalt Pavement Association
NMR	nuclear magnetic resonance
NMRD	nonmalignant respiratory disease
OR	odds ratio
PAC	polycyclic aromatic compound
PAH	polycyclic aromatic hydrocarbon
PEFR	peak expiratory flow rate
PMR	proportional mortality ratio
REL	recommended exposure limit
RR	relative risk
RTECS	Registry of Toxic Effects of Chemical Substances
SCE	sister chromatid exchange
SEM	standard error of mean
SIR	standardized incidence ratio
SMR	standardized mortality ratio
STEL	short-term exposure limit
TLV®	threshold limit value
TPA	12-*O*-tetradecanoylphorbol-13-acetate
TWA	time-weighted average
VOC	volatile organic compound
cm	centimeter
g	gram
g/mL	grams per milliliter
hr	hour
in/ft	inches per foot
L/min	liters per minute
mg	milligram
mg/m^3	milligrams per cubic meter
min	minute
mL	milliliter
mV	millivolt
ng/cm^2	nanograms per square centimeter
nm	nanometer
sec	second
°C	degrees Celsius
°F	degrees Fahrenheit
%	percent
wt %	weight percent
µg	microgram
$\mu g/m^3$	micrograms per cubic meter
µL	microliter

GLOSSARY OF TERMS

Aggregate: Graduated fragments of hard, inert mineral material that are mixed with asphalt. Aggregate includes sand, gravel, crushed stone, and slag [Stein 1980].

Asphalt (CAS number 8052-42-4): The product of the nondestructive distillation of crude oil in petroleum refining; it is a dark brown to black cement-like semisolid or solid. Depending on the crude oil used as a feedstock, the distillation residuum may be further processed, typically by air blowing (sometimes with a catalyst) or solvent precipitation, to meet performance specifications for individual applications [AI 1990b]. It is a mixture of paraffinic and aromatic hydrocarbons and heterocyclic compounds containing sulfur, nitrogen, and oxygen [Sax and Lewis 1987].

Asphalt cement: Asphalt that is refined to meet specifications for paving, roofing, industrial, and special purposes [AI 1990b].

Asphalt, cutback: An asphalt liquefied by the addition of diluents (typically petroleum solvents). Cutback asphalts are used in both paving and roofing operations depending on whether a paving or roofing asphalt is liquefied [AI 1990b; Roberts et al. 1996; Speight 1992a].

Asphalt, emulsified: A mixture of two normally immiscible components (asphalt and water) and an emulsifying agent (usually soap, but may be starch, glue, gum, colloidal clay, or other materials with similar properties) that allows the asphalt and water to mix. Emulsified asphalts are either cationic (electro-positively charged micelles containing asphalt molecules or anionic (electro-negatively charged micelles containing asphalt molecules) depending on the emulsifying agent. Emulsified asphalts are used for seal coats on asphalt pavements, built-up roofs, and for other waterproof coverings. Emulsified asphalts are also called asphalt emulsions [AI 1990b; Roberts et al. 1996; Speight 1992a; Stein 1980].

Asphalt fumes: The cloud of small particles created by condensation from the gaseous state after volatilization of asphalt [NIOSH 1977a].

Asphalt-based paints: A specialized cutback asphalt product that can contain small amounts of other materials such as lampblack, aluminum flakes, or mineral pigments. They are used as a protective coating in waterproofing operations and other similar applications [AI 1990b].

Asphalt, hot mix (HMA): Paving material that contains mineral aggregate coated and cemented together with asphalt cement [AI 1990b].

Asphalts, liquids: These are asphalts that are liquids at ambient temperatures. Liquid asphalts include cutback and emulsified asphalts [Roberts et al. 1996; Speight 1992a].

Asphalt, mastic: A mixture of asphalt and fine mineral material in such proportions that it may be poured hot into place and compacted by hand-troweling to a smooth surface [AI 1990b]. It is similar to hot-mix asphalt, but it is a finer aggregate.

Asphalt, oxidized (blown or air-refined) [CAS number 64742-93-4]: Asphalt treated by blowing air through it at elevated temperatures to produce physical properties required for the industrial use of the final product. Oxidized asphalts are typically used in roofing operations, pipe coating, undersealing for Portland cement concrete pavements, hydraulic applications, membrane envelopes [AI 1990b], and the manufacture of paints [Speight 1992a].

Asphalt, roofing: Asphalt that is refined or processed to meet specifications for roofing.

Asphalt, paving: Asphalt that is refined to meet specifications for paving.

Bitumen: The term more commonly used in Europe to refer to asphalt.

Coal tar: A tar that contains polycyclic aromatic compounds and is produced by the destructive distillation of bituminous coal [Bingham et al. 1980]. Distillation of coal-tar produces a variety of compounds such as coal tar pitch, creosote, and other chemicals or oils [NIOSH 1977b]. It is used in roofing, roads, waterproofing, paints, pipe coatings, sealants, insulation, and pesticides [Sax and Lewis 1987].

Coal tar pitch (CTP): A black or dark brown cementitious solid that is obtained as a residue in the partial evaporation or fractional distillation of coal tar [Bingham et al. 1980]. CTP is used in coatings, paints, roads, roofing, coal briquettes, and sealants [Sax and Lewis 1987].

Coal tar pitch volatiles (CTPV): Volatile matter emitted into the air when coal tar, coal tar pitch, or their products are heated [NIOSH 1977b].

Fog coat: Light application of slow-setting asphalt emulsion diluted with water. Fog coats are used to renew old asphalt surfaces and seal small cracks and surface voids [Stein 1980].

***International Agency for Research on Cancer (IARC)* categorization of agents as to their carcinogenicity:**

Group 1—The agent is carcinogenic to humans.

Group 2A—The agent is probably carcinogenic to humans.

Group 2B—The agent is possibly carcinogenic to humans.

Group 3—The agent is not classifiable as to its carcinogenicity to humans.

Group 4—The agent is probably not carcinogenic to humans.

Penetration macadam: Roadway consisting of a liquid asphalt sprayed onto a coarse aggregate (usually crushed gravel, slag, or stone) of uniform size [Stein 1980].

Polycyclic aromatic compound (PAC): A class of chemical compounds that contains two or more fused benzenoid rings. This class of compounds includes polycyclic aromatic hydrocarbons (PAHs) and heterocyclic derivatives where one or more of the carbon atoms in the benzenoid rings have been replaced by a heteroatom of nitrogen (N-PAC), oxygen (O-PAC), or sulfur (S-PAC) [Vo-Dinh 1989].

Polycyclic aromatic hydrocarbons (PAH): A class of chemical compounds that only contain carbon and hydrogen in two or more fused benzenoid rings [Vo-Dinh 1989].

Prime coat: Application of a viscous liquid asphalt by spraying onto an absorbent surface. It is used to prepare an untreated base for an asphalt overlay. The prime penetrates the base, filling voids, and hardens the top so that the asphalt overlay will bond [Stein 1980].

Seal coat: A liquid asphalt treatment used to waterproof and improve the texture of an asphalt wearing surface. Many seal coats are covered with an aggregate [Stein 1980].

Slurry seal: A mixture of a slow-setting emulsified asphalt, fine aggregate, and mineral filler with enough water added to form a slurry [Stein 1980].

Surface treatments: The addition of an asphaltic material to any road surface, with or without a covering of aggregate, that increases the thickness of the surface by less than 1 inch [Stein 1980].

Tack coat: A light application (usually by spraying) of a liquid asphalt cement to an existing pavement so that a bond can form with the new asphalt pavement [FAA 1991].

1 Introduction

1.1 Purpose

In 1977, the National Institute for Occupational Safety and Health (NIOSH) reviewed the available health effects data on occupational exposure to asphalt and asphalt fumes. NIOSH determined that the principal adverse health effects were irritation of the serous membranes of the conjunctivae and the mucous membranes of the respiratory tract [NIOSH 1977a]. Evidence from animal studies indicated that asphalt left on the skin for long periods of time could result in local carcinomas, but no comparable reports of such effects had been reported for humans exposed to asphalt or asphalt fumes. At that time, NIOSH recommended that occupational exposure to asphalt fumes be controlled so that employees were not exposed to airborne particulates at a concentration greater than 5 milligrams per cubic meter of air (5 mg/m^3) determined during any 15-min period.[1] Since then, additional data from studies on both animals and humans exposed to asphalt, paving and roofing asphalt fume condensates, and asphalt-based paints have become available. This report provides a review and evaluation of these new data, as well as other information. It is expected that this report will serve as a basis for identifying future research needs.

[1]When large amounts of dust are present in the work environment, use of the gravimetric method may lead to erroneously high estimates for asphalt fumes. Where resolution of such problems becomes necessary, a more specific procedure involving solvent extraction and gravimetric analysis should be employed for the determination of asphalt fumes [NIOSH 1977a].

1.2 Scope

The information in this document assesses the health hazards associated with occupational exposure to asphalt. Chapter 2 presents information about the uses of asphalt and the number of workers potentially exposed to asphalt during paving and roofing operations and during the manufacturing of asphalt roofing products. Chapter 3 describes the chemical and physical properties of asphalt, production methods, similarities and differences between paving and roofing asphalts and asphalt-based paints, and the representativeness of field- and laboratory-generated asphalt fumes. Chapter 4 discusses exposure monitoring methods, extent of worker exposure, and estimates of biological responses in asphalt-exposed workers. Subsequent chapters describe the effects of exposure to asphalt and asphalt fumes on humans and animals, conclusions, recommendations, and needed research.

2 Background

2.1 Number of Workers Potentially Exposed

About 3,600 hot-mix asphalt facilities and 7,000 paving contractors employ nearly 300,000 workers in the United States [Asphalt Paving Environmental Council (APEC) 1999]. Currently, the industry estimates that about 50,000 on-roof workers are exposed to asphalt fumes during, on the average, 40% of their working hours. Approximately 1,500 to 2,000 employees are exposed to asphalt fumes in approximately 100 roofing manufacturing plants [Asphalt Roofing Environmental Council (AREC) 1999].

2.2 Uses

Asphalt is commercially valuable because of its adhesive properties, flexibility, durability, water resistance, and ability to form strong cohesive mixtures with mineral aggregates. Most of the asphalt produced in the United States is used in paving and roofing. Only about 1% is used for other purposes, such as waterproofing, damp-proofing, insulation, and paints [Asphalt Institute (AI) 1990a]. Of the three types of asphalt products (asphalt paving cements, cutback asphalts, and asphalt emulsions) applied in U.S. paving operations, asphalt paving cements account for 85% of the total. Cutback asphalts and asphalt emulsions for road sealing and maintenance account for 4% and 11%, respectively, of the total [AI 1990a].

There are three basic grades of roofing asphalt.

- Saturant grade asphalt, a nonoxidized "straight reduced" asphalt or asphalt flux (typically an AC–10 or AC–20 grade material) used to manufacture saturated organic felt plies for built-up-roof (BUR) systems, organic felt shingles, and other roofing materials such as roll roofing.

- Coating grade asphalt, an oxidized asphalt used to manufacture roofing materials for a variety of roofing systems, such as asphalt shingles, polymer-modified bitumen roofing, felts, and roll roofing products.

- Mopping grade asphalt, an end-product that is melted and used in the construction of BUR systems and some modified bitumen systems. In the United States, mopping-grade roofing asphalts are classified into four types on the basis of their softening point temperature and resistance to flow (Table 2–1). The specific type applied to a roof is determined by roof grade or slope. Type I roofing asphalt, often referred to as "dead level," has a low softening point and is applied on surfaces having a grade of 0.5 inches per foot (in/ft) or less. Types II and III are typically applied on roofs having slopes of 0.5 to 1.5 and 1 to 3 in/ft, respectively. Type IV (a hard asphalt with a high softening point) is applied on roofs with a grade of 2 to 6 in/ft [American Society

for Testing Materials (ASTM) 1997]. For further information, see section 3.2.2.2.

Each of these three grades of asphalt—saturant, coating, or mopping—is also used in the manufacture of a variety of miscellaneous asphalt roof coating and sealant products. Asphalt roofing shingles, roll goods, underlayment felts, and roof coatings, cements, and mastics do not require heating during installation, and therefore workers are not exposed to asphalt fumes.

Information on asphalt-based paints can be found in section 3.2.3. Additional uses and applications of asphalt are provided in Appendix A.

Table 2–1. Types of mopping-grade roofing asphalts

Roofing asphalts	Description
Type I or dead level	Relatively susceptible to flow at roof temperatures. Can be used on slopes up to 2%. Softening point 57 to 66 ˚C (135 to 151 °F).
Type II or flat	Moderately susceptible to flow at roof temperatures. Can be used on slopes up to 4%. Softening point 70 to 80 ˚C (158 to 176 °F).
Type III or steep	Relatively nonsusceptible to flow at roof temperatures. Can be used on slopes up to 25%. Softening point 85 to 96 ˚C (185 to 205 °F).
Type IV or special steep	Relatively nonsusceptible to flow at roof temperatures. Can be used on slopes up to 50%. Softening point 99 to 107 ˚C (210 to 225 °F).

Source: Asphalt Roofing Manufacturers Association (ARMA) [1996].

3 Physical and Chemical Properties

This chapter describes the physical and chemical properties of asphalt products. Section 3.1 discusses physical and chemical properties and how manufacturing processes influence the chemical composition of asphalt products and how chemical composition, in turn, influences physical properties. Section 3.2 describes the different types and uses of asphalt products. Section 3.3 notes the use of asphalt modifiers and additives. Section 3.4 examines vapors and fumes and the differences in their chemical composition, as well as the difficulties involved in producing asphalt fumes in the laboratory that are representative of fumes produced in the field. Section 3.5 discusses the usefulness of various analytical sampling and analysis methods used to characterize asphalt exposures, and section 3.6 is a brief summary.

3.1 Properties

Table 3–1 is a summary of the physical properties, chemical names and synonyms, and numbers from the Chemical Abstract Service (CAS) and the Registry of Toxic Effects of Chemical Substances (RTECS) for asphalt, asphalt fumes, and asphalt-based paints.

Asphalt is the residuum produced by the distillation of crude petroleum at "atmospheric and under reduced pressures in the presence or absence of steam" [Puzinauskas and Corbett 1978]. Performance specifications (physical properties) and not chemical composition direct asphalt production. To meet performance specifications, asphalt may be air blown or further processed by solvent precipitation or propane deasphalting. In addition, the products of other refining processes may be blended with asphalt to achieve the desired performance specifications. Therefore, the exact chemical composition of asphalt depends on the chemical complexity of the original crude petroleum plus the manufacturing processes involved in creating the product.

Crude petroleum consists mainly of aliphatic compounds; cyclic alkanes; aromatic hydrocarbons; heterocyclic compounds containing nitrogen, oxygen, and sulfur atoms; and metals, e.g., iron, nickel, and vanadium. The proportions of these chemicals can vary greatly because of significant differences in crude petroleum from oil field to oil field or even from different locations in the same oil field [AI 1990a]. Consequently, because of their complexity, no two asphalts are chemically identical, and chemical analysis cannot be used to define the exact chemical structure or chemical composition of asphalt.

Elemental analyses indicate that most asphalts contain 79 to 88 weight percent (wt %) carbon, 7 to 13 wt % hydrogen, traces to 8 wt % sulfur, 2 to 8 wt % oxygen, and traces to 3 wt % nitrogen (see the examples in Table 3–2) [Speight 1992a]. Although heteroatoms (i.e., nitrogen, oxygen, and sulfur) make up only a minor component of most asphalts, they profoundly influence the differences in the physical properties of asphalts from different crude petroleum sources. The heteroatoms cause differences in physical properties by forming functional groups and imparting polarity to the asphalts; in turn, these functional groups and differences in polarity cause a variety of chemical interactions among asphalt molecules [Roberts et al. 1996; Speight 1992a].

Table 3–1. Physical properties and other information regarding asphalt [Sax and Lewis 1987], asphalt fumes, and asphalt-based paints

General information	Asphalt	Asphalt fumes	Asphalt paints
CAS number	8052-42-4	None	None
RTECS number	CI99000	None	None
Synonyms	Asphaltum, asphalt cement, asphalt emulsion, bitumen, air-blown asphalt, cutback asphalt, oxidized asphalt,* paving asphalt, petroleum asphalt, petroleum bitumen, road asphalt, roofing asphalt	Bitumen fume	Bitumen paint
Physical state at room temperature	Black or dark-brown solid or viscous liquid	Varies from light straw- or amber-colored low viscosity liquid to black or dark-brown solid or viscous liquid	Black or dark-brown viscous liquid
Solubility in water at 20 °C	Insoluble	Insoluble	Insoluble
Solubility in organic solvent	Partially soluble in aliphatic organic solvents; soluble in carbon disulfide	Same as asphalt	Same as asphalt

CAS=Chemical Abstract Service.
RTECS=Registry of Toxic Effects of Chemical Substances.
*CAS number 64742-93-4.

Table 3–2. Elemental analysis of asphalts from different crude petroleum sources (adapted from Speight 1992a)

Crude sources	Carbon, wt %	Hydrogen, wt %	Nitrogen, wt %	Sulfur, wt %	Oxygen, wt %	Vanadium, ppm	Nickel, ppm
Mexican blend	83.77	9.91	0.28	5.25	0.77	180	22
Arkansas-Louisiana	85.78	10.19	0.26	3.41	0.36	7	0.4
Boscan	82.90	10.45	0.78	5.43	0.29	1380	109
California	86.77	10.94	1.10	0.99	0.20	4	6

3.2 Types and Uses of Asphalt

3.2.1 Paving Asphalts

Paving asphalts are manufactured to meet performance specifications that are based on the physical properties of the asphalt product and not on chemical properties.

3.2.1.1 Manufacturing Processes

To produce a paving asphalt, crude petroleum is heated from 340 to 400 °C (644 to 752 °F) and introduced at atmospheric pressures into a distillation tower in which the most volatile components will vaporize. The volatile components rise in the distillation tower and

slowly cool. More volatile components will rise higher in the tower than less volatile components. When temperatures drop below the boiling point of a specific component, that component will condense and be collected in a tray. The remaining residuum is called "straight-reduced asphalt" [Federal Aviation Administration (FAA) 1991; Roberts et al. 1996; Speight 1992a].

However, because distillation is an inefficient separation process, considerable amounts of volatile components may remain in the residuum. Components with higher boiling points may need to be removed to meet the desired physical specifications. The residuum may be transferred to a vacuum distillation tower in which the distillation process is repeated at a reduced pressure. As pressure is reduced, the less-volatile components can vaporize at lower temperatures, and cracking (thermally breaking apart the asphalt molecules) is less likely to occur. The resulting residuum may be used to produce a "vacuum-processed asphalt." If steam is used during distillation, the resulting residuum is called a "vacuum-processed, steam-refined asphalt" [FAA 1991; Roberts et al. 1996; Speight 1992a].

While physical properties may change dramatically during the manufacturing process, the chemical nature of an asphalt does not change unless thermal cracking occurs. Raising the temperature to 400 to 565 °C (752 to 1049°F) will increase the likelihood of cracking and cause the more-volatile components (and even the components with higher boiling points) to be released from the residuum [Roberts et al. 1996; Speight 1992a].

Other common manufacturing processes include solvent precipitation, air blowing, and blending of asphalts or crude petroleum from different sources. Solvent precipitation (usually using propane or butane) removes high-boiling-point components from vacuum-processed asphalt; these components are then used to make other products. Solvent precipitation results in a harder asphalt that is less resistant to temperature changes. This asphalt is often blended with straight-reduced or vacuum-processed asphalts. Paving asphalt is not usually air blown, but air can be introduced to a vacuum-processed asphalt to form a more viscous product that is more resistant to weather and temperature changes. The air-blowing process can be a continuous or a batch operation. Because a continuous operation is faster and results in a softer asphalt, it is preferred for processing paving asphalts. Crude petroleum from different sources can be blended before refining so that the resulting asphalt meets required specifications; similarly, a higher viscosity asphalt can be blended with a lower viscosity asphalt to produce an asphalt of intermediate viscosity [Roberts et al. 1996; Speight 1992a].

3.2.1.2 Types, Uses, and Grades

Three types of asphalt are used in paving: asphalt cements, cutback asphalts, and emulsified asphalts. Cutback and emulsified asphalts also are called liquid asphalts because they are liquid at ambient temperatures [Roberts et al. 1996; Speight 1992a].

Asphalt cement refers to a straight-reduced or vacuum-processed asphalt manufactured according to paving specifications. Asphalt cements are used mainly as binders (4% to 10% of the mixture) in hot-mix asphalts and serve to hold the aggregate together [Roberts et al. 1996; Speight 1992a]. The asphalt cement is heated to about 149 to 177°C (300 to 350 °F) and mixed with mineral aggregate heated from 143 to 163 °C (290 to 325 °F). Once transported to the worksite, the hot-mix asphalt is applied to the roadway. The temperature at application is generally between 112 and 162 °C (235 and 325 °F) [AI 1990a; FAA 1991; Roberts et al. 1996; Speight 1992a].

The grade of asphalt cement is measured by either penetration or viscosity and depends on the amount of the higher boiling-point components that have been removed from the residuum. Penetration grade is determined by the depth a standard needle will sink in a

sample of asphalt cement at a given temperature, for a given time, and under a given load. There are five penetration grades: 40–50, 60–70, 85–100, 120–150, and 200–300 dmm (0.1 mm). The hardest asphalt cement (40–50 dmm) will allow the least penetration, while the softest (200-300 dmm) will allow the most penetration.

Viscosity grade can be based on the original asphalt cement (AC-2.5, AC-5, AC-10, AC-20, AC-30, and AC-40) or on the asphalt residue (AR-4000, AR-8000, and AR-16000). Asphalt residue is asphalt cement aged in a rolling-thin-film oven. Both the AC number and the AR number indicate viscosity in hundreds of poises (gram per centimeter second) at 60 °C (140 °F) [Roberts et al. 1996; Speight 1992a]. Performance grades as defined by the Strategic Highway Research Program [Roberts et al. 1996] are not included here because this information adds little to understanding the health effects of asphalt exposures.

To achieve the same density of the final pavement, a harder asphalt cement requires more compaction by a roller than does a softer (i.e., less-viscous) asphalt cement or it must be laid at a higher temperature [FAA 1991; Roberts et al. 1996; Speight 1992a]. Even if two asphalt cements have the same penetration or viscosity grades at one temperature, they may not have the same grade at a different temperature when the underlying chemistries of the two are different [Roberts et al. 1996; Speight 1992a].

A cutback asphalt is made by adding a diluent (typically a petroleum distillate) to an asphalt cement. Because cutback asphalts are liquids at or near ambient temperatures, they are often applied by spraying them on a surface. Cutback asphalts are graded depending on their viscosity at 60 °C (140 °F). Cutback asphalts are further classified according to the type of solvent used to liquefy the asphalt cement. Rapid-curing cutback asphalts are made by adding gasoline or naphtha and are mainly used as surface treatments, seal coats, and tack coats. Kerosene is added to produce medium-curing cutback asphalts, and diesel or other gas oils are added to produce slow-curing cutback asphalts. Medium- and slow-curing cutback asphalts are used mainly as surface treatments, primer coats, tack coats, mix-in-place road mixtures, and patching mixtures [Roberts et al. 1996; Speight 1992a].

Emulsified asphalt is a mixture of two normally immiscible components (asphalt cement and water) and an emulsifying agent (soap is an example). Since emulsified asphalts are liquids, they are often applied at ambient temperatures up to 150 °C (300 °F) simply by spraying them on a surface. Emulsified asphalts are graded as either cationic (electro-positively charged micelles containing asphalt molecules) or anionic (electro-negatively charged micelles containing asphalt molecules), depending on the emulsifying agent. Emulsified asphalts are further graded on the basis of their setting rate, i.e., rapid, medium, or slow. Rapid-setting grades are used for surface treatments, seal coats, and penetration macadams; medium-setting grades are used for patching mixtures; and slow-setting grades are used in mix-in-place road mixtures, patching mixtures, tack coats, fog coats, slurry sealants, and soil stabilizers [Roberts et al. 1996; Speight 1992a].

3.2.2 Roofing Asphalts

Roofing asphalts are manufactured to meet roofing performance specifications on the basis of the physical properties and not the chemical properties of the asphalt product.

3.2.2.1 Manufacturing Processes

Although straight-reduced or vacuum-processed asphalts are used to make roofing products, much of the asphalt used in roofing operations is made by air blowing these asphalts. Air-blown asphalts are called oxidized asphalts, air-refined asphalts, or roofing asphalts. In the

air-blowing process, asphalt hardens as it comes into contact with air at 204 to 288 °C (400 to 550 °F [Corbett 1979]). Once the asphalt has the desired specifications, it is either held in storage tanks at elevated temperatures or is cooled before it is pumped into storage containers, where it solidifies. The air-blowing process can be a continuous or a batch operation. A batch operation is slower and produces a harder asphalt, and is preferred for processing roofing asphalts [Puzinauskas and Corbett 1978; Corbett 1979; Roberts et al. 1996; Speight 1992a].

Air blowing combines oxygen with the hydrogen in the asphalt to produce water vapor. This decreases saturation and increases cross-linkages within and among different asphalt molecules. The process is exothermic (produces heat) and may include a series of chemical reactions, such as oxidation, condensation, dehydration, dehydrogenation, and polymerization. These reactions cause the amount of asphaltenes (hexane-insoluble materials) in the asphalt to increase, the amounts of polar aromatics (hard resins), cycloalkanes, and nonpolar aromatics to decrease (soft resins), while the amount of aliphatic compounds (oils and waxes) remains about the same (Table 3–3); the oxygen content of the asphalt increases (Moschopedis and Speight 1973; Corbett 1975; Puzinauskas and Corbett 1978; Boduszynski 1981; Roberts et al. 1996; Speight 1992a].

The effect of air blowing also can be facilitated with chemical compounds. Ferric chloride, aluminum chloride, zinc chloride, phosphorus pentoxide, copper sulfate, or boric acid have been used to produce catalytic asphalts. Moreover, sulfur or chlorine can be added to the asphalt to react with hydrogen, yielding hydrogen sulfide or hydrogen chloride, respectively [Puzinauskas and Corbett 1978; Corbett 1979; Roberts et al. 1996; Speight 1992a].

Roofing asphalt specifications also can be influenced by blending crude petroleum from various sources or asphalts. Crude petroleum can be blended before refining and air blowing to meet needed specifications; similarly, a high-viscosity roofing asphalt can be blended with a low-viscosity roofing asphalt to produce an intermediate-viscosity roofing asphalt [Speight 1992a].

Table 3–3. Changes in physical properties and chemical classes in a straight-reduced Arkansas asphalt with increasingly longer air-blowing times ($T_0<T_1<T_2<T_3$) (adapted from Speight 1992a)

Ashphalt properties	T_0	T_1	T_2	T_3
Physical properties:				
Softening point, °C	54.4	85	96.1	173.3
Penetration, mm/10 (0.1 mm)	36	13	9	1
Chemical class, wt %:				
Asphaltenes	14.8	26.9	31.4	51.3
Hard resins	45.5	36.6	36.1	19.6
Soft resins	25.0	22.3	20.9	16.9
Oils	12.3	11.9	10.0	11.1
Waxes	2.5	2.0	1.8	1.6
Total	100.0	99.7	100.2	100.5

NOTE: T_0 is equivalent to nonoxidized asphalt (no air-blowing time).

3.2.2.2 Types, Uses, and Grades

Although the focus in this section is on BUR products, a variety of asphalts are used in roofing products. The asphalts used to produce shingles, roll goods, felts, and underlayments may or may not be air blown and are shipped hot and kept hot until they are used in a manufacturing process [AREC 1999].

Most of the air-blown asphalt used for roofing is shipped as solid kegs in cardboard cartons and heated in a kettle at the worksite until it becomes a liquid. Asphalt also may be delivered as a hot liquid in a tanker truck, but this practice is becoming less common because of higher costs, regulatory constraints, and product supply considerations. Asphalt delivered by tanker may be also heated to the desired temperature in the tanker or transferred to a kettle for heating, after which it is pumped to the roof [AREC 1999].

Some cutback and emulsified asphalts are also used in roofing operations [Speight 1992a]. Although these asphalts represent only a small amount of the asphalt used in roofing, a recent study suggests their use may be increasing [Herbert et al. 1995].

Mopping-grade roofing asphalts in the United States are classified into four types—I, II, III, and IV—based on increasing hardness. The type of asphalt to be used is determined by the grade or slope of the roof. Type I roofing asphalt, often called "dead level," has the lowest softening point and is used on surfaces with a grade of 0.5 in/ft or less. Types II and III roofing asphalts are typically used on roofs with grades of 0.5 to 1.5 and 1 to 3 in/ft, respectively. Type IV roofing asphalt has the highest softening point and is used on roofs with a grade of 2 to 6 in/ft [ASTM 1997].

A mopping-grade roofing asphalt is best applied when the asphalt is at its equiviscous temperature, or the temperature at which the viscosity of the asphalt is either 125±25 centistokes for hand mopping or 75±25 centistokes for mechanical spreaders [Intec 1998]. Table 3–4 lists recommended application temperatures for mopping-grade roofing asphalts [AI 1990a, Appendix C]. However, to achieve these application temperatures, the asphalt must be heated to even higher temperatures in the kettle. During recent surveys of roofing operations in which Type III roofing asphalts were used, the kettles were often maintained at 288 °C (550 °F). Several kettlemen stated that if the demand on the roof for asphalt is high, they will heat the asphalt to 316 °C (600 °F) [Hayden 1998; Mead 1998].

Table 3–4. Recommended application and maximum heating temperatures used with mopping-grade roofing asphalts

	Recommended application temperatures*		Recommended maximum heating temperature†	
Type	**°F**	**°C**	**°F**	**°C**
I	330–355	166–179	475	246
II	365–390	185–199	500	260
III	395–420	202–216	525	274
IV	430–445	221–229	525	274

*Adapted from AI [1990a, Appendix C] and AREC [1999].
†Adapted from AREC [1999].

Care must be exercised when operating kettles at high temperatures. Temperatures in excess

of the flash point of the asphalt can result in fires that cause serious burns. If not quickly extinguished, kettle fires can spread rapidly to the exterior of the kettle and engulf equipment, including propane tanks (and gasoline tanks on some models), with catastrophic results. An explosion hazard may be created when the kettle lid is closed and the atmosphere in the headspace of the kettle is within explosive limits [AREC 1999].

3.2.3 Asphalt-Based Paints

Asphalt-based paints are specialized cutback asphalt products that may contain a small amount of lampblack, aluminum flakes, or mineral pigments. Asphalt-based paints are used as protective coatings in waterproofing operations and in other similar applications [AI 1990b]. The asphalt used to make an asphalt-based paint may or may not be air blown [Speight 1992a,b]; the only requirement is that the final product has the flow and drying characteristics of a paint. Basically, asphalt-based paints may be applied at or near ambient temperatures by spraying or brushing. Once the asphalt-based paint is applied to a surface, it should not flow, and it should harden quickly. This is achieved either by manipulating the manufacturing process or by the addition of diluents.

3.3 Asphalt Modifiers and Additives

Although the subject of asphalt modifiers and additives is beyond the scope of this document, it would be remiss not to mention their use because a worker may be exposed to a modifier or an additive or even to their decomposition products, and their presence in asphalt may affect the composition of asphalt fumes. Asphalt modifiers and additives are used to enable asphalt products to meet desired performance specifications and serve a variety of functions, as described in Table 3–5 [Roberts et al. 1996; Speight 1992a].

3.4 Asphalt Vapors and Fumes

When asphalt products are heated, vapors are released; as these vapors cool, they condense. By definition, the condensate is asphalt fume; however, because the components in the vapor do not condense all at once, workers are exposed not only to asphalt fumes, but also to vapors. The physical nature of fumes and vapors has not been well characterized, but fumes should be fairly viscous. When liquid asphalt products are used at ambient temperatures, workers are exposed to the liquid product and to vapors, but not to fumes. Fume particles may collide and stick together, making it difficult to characterize fume particle size. Some of the vapors may condense only to the liquid phase, thus forming a viscous liquid containing some solids.

3.4.1 Cutback Asphalts, Emulsified Asphalts, and Asphalt-Based Paints

Cutback asphalts, emulsified asphalts, and asphalt-based paints are liquids and are applied at or near ambient temperatures [Roberts et al. 1996; Speight 1992a]. Because these products are liquids, workers may be exposed through both respiratory and dermal contact. These products are applied in a variety of ways, including by spraying, and if the spray nozzle becomes clogged, a worker may face increased dermal exposure and clothing contamination when cleaning the nozzle. Petroleum distillates added to asphalt products can dry the skin, weakening the protective barrier skin provides and facilitating the entry of various compounds into the body. Furthermore, petroleum distillates introduce the hazard of exposure to volatile organic compounds (VOCs).

Table 3–5. Asphalt modifiers and additives (adapted from Roberts et al. 1996 with information from Speight 1992a)

Type	Examples
1. Antioxidants	Calcium salts
	Carbon
	Lead compounds
2. Antistripping agents	Amines
	Lime
3. Combinations	Blends of plastics (9) and rubbers (10)
4. Extenders	Lignin
	Sulfur
5. Fibers	Manufactured:
	Cellulose
	Fiberglass
	Mineral
	Polyester
	Polypropylene
	Natural:
	Asbestos
	Rock wool
6. Fillers	Carbon black
	Mineral filler:
	Crusher fines
	Fly ash
	Lime
	Portland cement
7. Hydrocarbons	Hard and natural asphalts
	Recycling and rejuvenating oils
8. Oxidants	Manganese salts
9. Plastics	Ethylene acrylate copolymers
	Ethylene propylene
	Ethyl-vinyl-acetate
	Polyethylene/polypropylene
	Polyolefins
	Polyvinyl chloride
10. Rubbers:	Natural rubber
Block copolymers	Styrene-butadiene
Natural latex	Polychloroprene latex
Reclaimed	Styrene-butadiene-styrene, styrene-isoprene-styrene
Synthetic latex	Crumb-rubber-modifier
11. Waste materials	Glass
	Recycled tires
	Roofing shingles
12. Miscellaneous	Coal liquefaction products
	Components of shale oil
	Deicing calcium chloride granules
	Petroleum distillates:
	Diesel and other gas oils
	Gasoline
	Kerosene
	Naphthas
	Stoddard solvent
	Shale oil residues
	Silicones

The composition of the vapors released from these asphalt products during drying can be explained with Raoult's Law: The composition of the vapor phase above a solution is directly proportional to the mole fraction and vapor pressure of each component in the solution. Other factors influencing the composition of the vapor phase are that (1) as temperature increases, vapor pressure increases, which may allow appreciable quantities of certain compounds to exist in the vapor phase, and (2) the chemical composition of a chemical class will generally increase in complexity in the vapor phase. Generally, the smaller compounds in a given chemical class will have higher vapor pressures.

As liquid asphalt products harden from the outside surface in, the added diluents slowly evaporate from the outside surface, thus trapping part of the diluent inside the asphalt layer. However, if these asphalt products are heated even slightly, not only will the same compounds vaporize faster, but higher concentrations of the same compounds will vaporize along with other compounds that do not vaporize appreciably at ambient temperatures. In the absence of significant increases in temperature, these asphalt products are expected to release primarily vapors from the evaporating solvent.

No reports discussing the chemical analysis of cutback asphalts or emulsified asphalts were found. However, Robinson et al. [1984] reported on the analysis of select polycyclic aromatic compounds (PACs) in several asphalt-based paints using gas chromatography-mass spectroscopy (GC/MS). Benz[a]anthracene, benzo[a]pyrene (B(a)P), benzo[e]pyrene, chrysene, and phenanthrene were measured, but only trace amounts of phenanthrene (<0.01%) were detected.

3.4.2 Comparison of Vapors and Fumes from Paving and Roofing Asphalts

Information presented in the previous section indicates that (1) higher temperatures increase the chemical complexity of paving and roofing asphalts, (2) paving and roofing vapors and fumes are chemically more complex than liquid asphalt vapors and fumes, (3) vapors and fumes from paving asphalts are different from those of roofing asphalts because of differences in application temperatures, i.e., roofing asphalts are applied at higher temperatures (166 to 229 °C [340 to 455 °F]) than paving asphalts (112 to 162 °C [235 to 325 °F]), and (4) differences in manufacturing processes affect the composition of asphalt and consequently the composition of fumes. Compared to air-blown roofing asphalts, nonoxidized asphalts generally contain more aliphatic compounds, about the same amount of cycloalkanes and nonpolar aromatics, and smaller amounts of polar aromatic compounds and asphaltenes (Table 3–3). This does not mean, however, that air-blown roofing asphalts contain more aromatics than nonoxidized asphalts.

Differences in the way paving and roofing asphalts are handled also probably influence the composition of vapors and fumes. For example, a hot-mix asphalt begins to cool from the moment it leaves the plant and may not be used immediately when it arrives at a worksite, whereas roofing asphalts are heated continuously and stirred occasionally at a worksite until they are needed.

Using GC/MS, several investigators reported on chemical analyses of paving and roofing asphalt fumes [Reinke and Swanson 1993; Niemeier et al. 1988; Lunsford and Cooper

1989; Hatjian et al. 1995a, 1997]. Others have used liquid chromatography (LC) methods to analyze for polycyclic aromatic hydrocarbons (PAHs) in asphalt vapors and fumes. Because of methodology limitations, LC methods should not have been used to analyze PAHs in asphalt fumes; therefore, results from these studies are not discussed here. However, LC methods and their limitations are discussed in section 3.5.

Reinke and Swanson [1993] collected paving asphalt fumes from a straight-reduced, vacuum-processed 85-100 grade asphalt cement. Using the NIOSH protocol [Thayer et al. 1981; Sivak et al. 1989], the authors collected fumes from a storage tank at a hot-mix plant at a temperature of 149 °C (300 °F) and from laboratory generation at temperatures of 149 and 316 °C (300 and 601 °F). The fume samples were then analyzed for selected PACs (Table 3–6). The results indicate that only two- and three-ring PACs were present in the fumes from the storage tank, but that the chemical classes identified in the laboratory-generated fumes were mostly two- and three-ring PAHs and a few three-ring sulfur-PACs (S-PACs) and four-ring PAHs. Several of the four-ring PAHs are carcinogenic, i.e., benz[a]anthracene and chrysene. Methylated chrysenes, pyrenes, and fluoranthenes were also detected and may be of concern because of their structural relationship to known carcinogens. The concentration of four-ring PAHs was highest in fumes generated in the laboratory at the highest temperature. However, the concentration of two-ring PAHs was lowest in laboratory fumes generated at the highest temperature, most abundant in fumes from the storage tank, and lower in the fumes generated in the laboratory at the ambient temperature of the storage tank.

In the laboratory tests, once the asphalt melted, the mixture was stirred constantly until it reached the desired temperature. The higher the generation temperature, the longer the mixture had to be stirred before the desired temperature was reached, which caused more of the two-ring PAHs to reach the surface of the liquid and escape before collection of the fumes began. This stirring procedure might explain why some three-ring PACs were found in the laboratory-generated fumes, but not in the fumes from the storage tank. In the storage tank, not enough of the three-ring PACs were brought to the surface to escape in sufficient concentrations to be detected. Of interest is that a summation of the measured PAHs (Table 3–6) only accounts for 0.8% to 1.3% of the total asphalt fumes, assuming the density of asphalt to be 1 gram per milliliter (g/mL) [Speight 1992a]. This is not surprising since aliphatic compounds compose the majority of the compounds present in asphalt fumes.

Thayer et al. [1981] and Niemeier et al. [1988] collected asphalt fumes generated in the laboratory from Type I and Type III roofing asphalts and Type I and Type III roofing coal-tar pitches. The fumes were generated at temperatures of 232 and 316 °C (450 and 601 °F) and analyzed for PAHs by GC/MS. The results of the analysis and information regarding which PAHs are considered carcinogenic are given in Table 3–7. The results indicate that asphalt fumes had much lower concentrations of PAHs than the coal-tar-pitch fumes and consisted mainly of two- to four-ring PAHs with small amounts of five-ring PAHs. Concentrations of two-, three-, and some four-ring PAHs were generally lowest in the laboratory fumes generated at the highest temperatures. Concentrations of the two-, three- and some four-ring PAHs decreased before collection began, because the laboratory-generated fumes were allowed to escape until the entire mixture reached the desired temperature. Moreover, once the asphalt melted, the mixture was stirred constantly, causing more of the two-, three-, and four-ring PAHs to reach the surface of the liquid and escape before collection began.

Table 3–6. Chemical analysis by GC/MS of storage-tank and laboratory-generated paving asphalt fume condensates, mg/mL per sample (adapted from Reinke and Swanson 1993)

Chemical analyte	Tank fumes at 149 °C (300 °F)	Laboratory fumes	
		149 °C (300 °F)	316 °C (601 °F)
Naphthalene	2.1	1.6	0.1
Acenaphthene	0.12	0.03	—
Fluorene	0.12	0.22	0.09
Phenanthrene	0.15	0.47	0.27
Anthracene	0.13	0.46	0.03
Fluoranthene	—	0.02	—
Pyrene	—	0.03	0.07
Chrysene	—	0.02	—
Benz[a]anthracene and chrysene	—	—	0.11
Methyl naphthalenes	4.90	5.2	0.4
Methyl fluorenes	0.17	0.36	0.16
Methyl phenanthrenes and anthracenes	0.22	1	1.4
Methyl pyrenes or fluoranthenes	—	—	0.15
Methyl chrysenes	—	—	0.11
Dibenzothiophene	0.09	0.57	0.24
Methyl dibenzothiophenes	0.15	1.1	0.72
“C2" alkyl dibenzothiophenes	0.17	1.3	1.1
“C3" alkyl dibenzothiophenes	0.1	0.88	0.85
Benzo[a]naphthothiophenes	—	0.03	0.12
Methyl benzo[b]naphthothiophenes	—	0.06	0.33
“C2" alkyl benzo[b]naphthothiophenes	—	0.04	0.35
“C3" alkyl benzo[b]naphthothiophenes	—	0.03	0.37

—Not reported.
NOTE: Asphalts were straight-reduced, vacuum-processed, 85-100 grade.

Also, the higher the generation temperature, the longer the mixture would be stirred before the desired temperature was reached.

Because concentrations of the remaining four- and five-ring PAHs were low and similar in amount at the two generation temperatures, similar trends in concentration were not observable. Air blowing appears to have had little effect on concentrations of the higher molecular weight PAHs, but decreased concentrations of the lower molecular weight PAHs (Table 3–7).

Niemeier et al. [1988] reported that analysis by nuclear magnetic resonance spectroscopy (NMR) indicated that asphalt fume condensates were less than 1% aromatic and more than 99% aliphatic, whereas the coal-tar-pitch condensates were more than 90% aromatic. Assuming ^{13}C NMR was used, these percentages are indicative of the carbon atom character. These results also indicate that in asphalt fumes, most of the carbon atoms are contained in aliphatic groups, while in the coal-tar-pitch condensates, most of the carbons are contained in aromatic groups.

Sivak et al. [1989, 1997] heated Type III roofing asphalt from the same lot used by Niemeier et al. [1988] at 316 °C (601 °F), generated fume condensates, and separated them into fractions by high-performance liquid chromatography (HPLC) [Belinky et al. 1988]. Using GC/MS, Lunsford and Cooper [1989] characterized the chemical classes present in these asphalt fume fractions. Results (Table 3–8) indicate the relative

Table 3–7. Mean concentration of 18 PAHs determined by GC/MS in skin painting solutions, µg/mL (adapted from Niemeier et al. 1988 and Thayer et al. 1981)

	Solutions containing roofing asphalt fume condensates				Solutions containing roofing coal-tar-pitch fume condensates			
	Type I		Type III		Type I		Type III	
PAH	232 °C	316 °C	232 °C	316 °C	232 °C	316 °C	232 °C	316 °C
Naphthalene	22	4	17	49	>1800	1770	288	620
Fluorene	36	22	39	28	ND	740	ND	ND
Carbazole	20	1	6	ND	1980	1450	540	1400
Anthracene/phenanthrene	180	53	300	69	>960	2960	>2580	>5200
Fluoranthene	86	10	97	7	>2940	2350	>960	>2800
Pyrene	70	9	63	8	>2070	1790	>720	>2300
Benz[a]anthracene*	11	10	8	6	570	330	330	800
Chrysene/triphenylene†	25	19	13	14	460	300	290	710
Benzofluoranthenes‡	2	4	5	ND	230	230	250	250
Benzo[e]pyrene	6	8	4	1	42	51	45	46
B(a)P*	2	2	3	ND	96	85	102	90
Indeno[cd]pyrene§	3	3	2	ND	33	2	11	7
Benzo[ghi]perylene	1	2	1	ND	28	2	7	1
Dibenzanthracenes**	2	ND	2	ND	12	ND	4	ND
Coronene	ND	ND	ND	ND	ND	ND	ND	ND
Dibenzopyrenes††	ND	ND	ND	ND	ND	ND	ND	ND

ND=not detected.
*Included in IARC Cancer Review Group 2A. (Probably carcinogenic to humans.)
†Classified by NIOSH as a potential occupational carcinogen; included in IARC Cancer Review Group 3. (Not classifiable as to its carcinogenicity to humans.)
‡Benz[e]acephenanthrylene (benzo[b]fluoranthene), benzo[j]fluoranthene, and benzo[k]fluoranthene are specific compounds included in IARC Cancer Review Group 2B. (Possibly carcinogenic to humans.)
§Included in IARC Cancer Review Group 2B.
**Dibenz[a,h]anthracene included in IARC Cancer Review Group 2A.
††Benzo[rst]pentaphene (dibenzo[a,i]pyrene); dibenz[b,def]chrysene (dibenzo[a,h]pyrene); dibenzo[def,p] chrysene (dibenzo[a,l]pyrene); and naphtho[1,2,3,4-def] chrysene (dibenzo[a,e]pyrene) are specific compounds included in IARC Cancer Review Group 2B.
NOTE: 232 °C=450 °F; 316 °C=601 °F.

abundance of each compound class in the fractions, but not in the classes. The results also indicate that many of the compounds are aliphatic and many compound classes contain alkylated isomers. Fraction A constituted 64.1% of the asphalt fume condensate, while fractions B, C, D, and E constituted 8.3%, 10.5%, 11.5%, and 5.6%, respectively. Most compound classes were found in more than one fraction, indicating the complexity created by the addition of alkyl groups and the presence of many different isomers for each compound class. Despite this complexity, only fractions B and C caused tumors in a mouse-skin painting study (see section 6.2.1). Fraction B contained mainly alkylated two- and three-ring PAHs, oxygen-PACs (O-PACs), and S-PACs; a few alkylated four-ring PAHs (pyrenes and fluoranthenes); and a variety of ketones. Little is known about the toxicity of most of these compounds except that some O-PACs may cause cancer and some S-PACs may cause mutations [Tennant and Ashby 1991; Pelroy et al. 1983; McFall et al. 1984]. The two- and three-ring O-PACs were not detected in fraction C, but the other PACs and ketones found in fraction B were also found in fraction C. Fraction C also contained alkylated and unalkylated isomers of chrysene and benz[a]anthracene, a few four-ring S-PACs with and without alkyl groups attached, a wide variety of ketones, and alkanoic acids.

Table 3–8. Analysis by GC/MS of chemical composition of asphalt fume fractions A-E from Type III roofing asphalt fumes collected during laboratory generation at 316 °C (601 °F) (adapted from Lunsford and Cooper 1989)

	Fraction†				
Compound class*	A	C	B	D	E
Hydrocarbons:					
Alkanes, C_9 - C_{27}	++	+	+	−−	−−
Alkenes/cycloalkanes	++	+	+	−−	−−
Benzenes, C_2 - C_8	++	+	−−	−−	−−
Indanes, C_0 - C_4	++	+	−−	−−	−−
Indenes, C_0 - C_3	++	+	−−	−−	−−
Naphthalenes, C_0 - C_5	++	+	−−	−−	−−
Biphenyls, C_0 - C_2	++	+	−−	−−	−−
Fluorenes, C_0 - C_3	++	+	−−	−−	−−
Anthracenes/phenanthrenes, C_0 - C_4	++	+++	+	−−	−−
Pyrenes/fluoranthenes, C_0 - C_2	−−	++	+	−−	−−
Chrysenes/benz[a]anthracenes, C_0 - C_2	−−	−−	+	−−	−−
Sulfur-containing compounds:					
Benzothiophenes, C_0 - C_9	++	+	−−	−−	−−
Dibenzo-/naphthothiophenes, C_0 - C_4	++	+++	+	−−	−−
Tricarbocyclic fused-ring thiophenes, C_0 - C_1	−−	−−	+	−−	−−
Hydroxybenzenethiols, C_0 - C_4	−−	−−	+	−−	−−
Oxygen-containing compounds:					
Benzofurans, C_0 - C_2	−−	+	−−	−−	−−
Dibenzofurans, C_0 - C_2	−−	+	−−	−−	−−
Acetophenones, C_0 - C_3	−−	+++	++	+	+
Fluorenones, C_0 - C_3	−−	+	++	−−	−−
Dihydroindenones, C_0 - C_4	−−	++	+++	+	+
Cycloalkenones, C_6 - C_{11}	−−	+	+++	++	+
Dihydrofuranones	−−	−−	+	++	−−
Isobenzofuranones, C_0 - C_3	−−	−−	+	++	−−
Phenols, C_0 - C_4	−−	−−	−−	+	−−
Naphthols, C_0 - C_2	−−	−−	−−	+	−−
Furanones, C_1 - C_3	−−	−−	−−	+	−−
Alkanones, C_8 - C_{22}	−−	−−	−−	++	+
Alkanoic acids, C_5 - C_{14}	−−	−−	+	++	+++
Benzoic acids, C_0 - C_4	−−	−−	−−	−−	+
Nitrogen-containing compounds:					
Carbazoles, C_0 - C_4	−−	−−	−−	+	−−

−− Not observed.
*Degree of alkyl substitution given by C_n, where subscript=number of substituent carbon atoms.
†Relative abundance across fractions, but not classes, indicated by +++ > ++ > +.

Chrysene and benz[a]anthracene are known carcinogens, but little is known about the toxicity of the other observed PACs except that some S-PACs may cause mutations [Pelroy et al. 1983; McFall et al. 1984]. Many alkylated isomers of PACs have been identified in asphalt fumes, but although little is known about their carcinogenic and genotoxic activity, these PACs are a cause for concern because of their structural similarity to known carcinogens and genotoxins.

Probably because an air-blown roofing asphalt was used, numerous oxidized compounds were found; however, if paving fumes had been studied, it is possible that not as many

oxidized compounds would have been detected. The presence of alkanoic acids probably indicates that the starting crude petroleum source contained aldehydes because aldehydes are easily oxidized to carboxylic acids. Because of the process (addition of ferric chloride and air blowing) used to prepare the roofing asphalt, it is unlikely that very many aldehydes survived the manufacturing process. However, if any of the aldehydes did survive, it is unlikely that workers would have been exposed to them in the field, because aldehydes are easily oxidized at ambient temperatures, and the elevated temperatures needed at a field site would only hasten the oxidation process. While it is more likely that aldehydes would not oxidize as easily during manufacturing of paving asphalts, it is also likely most of the aldehydes would oxidize before asphalt pavers could be exposed. Because only a few nitrogen-containing compounds were found, the asphalt may have been manufactured from a crude petroleum containing low amounts of nitrogen; therefore, other asphalts with a higher nitrogen content may yield more nitrogen-containing compounds.

Hatjian et al. [1995a, 1997] reported on the GC/MS analysis of nine PAHs in personal-breathing-zone air samples. The samples were collected from two sets of asphalt pavers (P1 and P2), two sets of asphalt roofers (R1 and R2), manual laborers (M) who had no apparent occupational exposure to PAHs, and office workers (C) who were used as controls. The median percentage for each PAH determined, the number of rings in each PAH, and the number of samples for each group are given in Table 3–9. No results for the control group are included in Table 3–9 because none of the samples contained detectable amounts of any of the measured PAHs.

Naphthalene accounted for 80% to 90% of the measured PAH exposure for each group, except in group R2 (60%). Naphthalene, acenaphthene, and phenanthrene accounted for 98% to 99% of the measured PAH exposure for each group, with the exception of groups R1 and R2 (94% and 84%, respectively). The four- and five-ring PAHs each accounted for less than 1% of the measured PAH exposure for all groups except R1 and R2. For group R1, the four- and five-ring PAHs (except pyrene) each accounted for less than 1% of the measured PAH exposure; pyrene accounted for 1%. For group R2, benz[a]anthracene and pyrene accounted for 2% and 7% of the measured PAH exposure, respectively, while the five-ring PAHs accounted for less than 1% each. Because roofing asphalts are heated to hotter temperatures and applied at higher temperatures than paving asphalts, which increases the amount of the larger PAHs in the fumes, the median percentage values for two- and three-ring PAHs were lower (Table 3–10).

Table 3–10 contains a summary of the PAH data and smoking habits for each group. For each PAH, a mean concentration was calculated on the basis of a 3-day geometric mean of the air samples expressed as an 8-hour time-weighted average (TWA). These mean concentrations were summed for different groups of PAHs: all PAHs ($\sum$ nine PAHs), all PAHs except naphthalene ($\sum$ eight PAHs), and four- and five-ring PAHs ($\sum$ four- and five-ring PAHs). Summations of four- and five-ring PAHs were not calculated for groups other than roofers, nor were summations of mean concentrations calculated for the control group because in both instances there were not enough data recorded at detectable levels to allow these calculations.

B(a)P was detected in personal-breathing-zone samples as follows: manual laborers (5.9% of

Table 3–9. Median percentage of TWA concentrations of nine PAHs determined in personal-breathing-zone samples (adapted from Hatjian et al. 1995a, 1997)

PAH and number of rings	Group M (n=34)	Group P1 (n=18)	Group P2 (n=30)	Group R1 (n=27)	Group R2 (n=12)
Naphthalene (2)	90	84	88	80	60
Acenaphthene (3)	5	8	4	5	4
Phenanthrene (3)	3	7	7	9	20
Pyrene (4)	<1	<1	<1	1	7
Benz[a]anthracene (4)	<1	<1	<1	<1	2
Benzo[b and k]fluoranthene (5)	<1	<1	<1	<1	<1
B(a)P (5)	<1	<1	<1	<1	<1
Dibenz[a,h]anthracene (5)	<1	<1	<1	<1	<1

Abbreviations: M=manual laborer group; n=number of samples; P1 and P2=paver groups 1 and 2; R1 and R2=roofer groups 1 and 2.
NOTE: Group C (control group) not included since none of the samples contained detectable amounts of the measured PAHs.

Table 3–10. Summary of smoking habits, asphalt temperatures, and summed individual geometric mean ±SEM concentrations for different groups of PAHs, ng/m^3 (adapted from Hatjian et al. 1995a, 1997)

	Smokers		Asphalt temperature				
Group	No.	%	°C	°F	∑ 9 PAHs	∑ 8 PAHs*	∑ 4- and 5-ring PAHs
Controls (office workers)†	3	50	NA	NA	NC	NC	NC
Manual laborers	4	27	NA	NA	448 ±71.2	72.2 ±16.1	NC
Asphalt pavers:							
P1	2	33	NL	NL	1584 ±454	285 ±79.0	NC
P2	5	50	150–180	302–356	2100 ±501	223 ±44.6	NC
Asphalt roofers:							
R1	2	22	300	572	442 ±109	107 ±24.7	35.2 ±7.0
R2	3	75	190	374	2120 ±680	55 ±255	69.1 ±24.3

Abbreviations: NA=not applicable; NC=not calculated, "not enough with detectable levels;" NL=not listed.
*All measured PAHs excluding naphthalene.
†Only static air samples were collected.

the samples), group P1 (5.6%), group P2 (3.3%), group R1 (28%), group R2 (25%), and the control group (<1%). The highest B(a)P concentrations were 0.17 [Hatjian 1995], 0.02 and 0.20 μg/m^3 for manual laborers, pavers, and roofers, respectively. Hatjian et al. [1995a, 1997] stated that the manual laborers had no apparent occupational exposure to B(a)P. Their reported exposures probably resulted from the environment, an unidentified source, or a sampling and analytical error. The authors did not provide background concentrations of PAHs downstream of the paving or roofing operations. However, in his review of the NIOSH draft hazard review, Hatjian [1999] stated that "background concentrations of PAHs downstream of the roofing operations in Hatjian's study showed non-detectable levels of B(a)P on the day of monitoring the roofing operation."

When reviewing these data, consideration must be given to B(a)P concentration and sampling variability. Since the highest B(a)P concentration for a paver was only twice the reported detection limit for B(a)P, this determination is not reliable. Among all the groups, only one roofer had more than one of three personal-breathing-zone samples with detectable concentrations of B(a)P [Hatjian 1995].

Environmental and personal factors as well as work practices could contribute to pavers' and roofers' exposures to B(a)P. For example, 22% to 75% of the workers in the paving and roofing groups were smokers, and at least one paving group was exposed to diesel exhaust. In addition, the highest B(a)P concentration for a roofer may be atypically high because of work practices at both roofing sites (workers knelt while spreading roofing asphalt with a trowel or brush) and the high kettle temperature (300 °C [572 °F] at the R1 site). This temperature is about 70 °C (158 °F) higher than the highest recommended temperature for roofing application (Table 3–4).

3.4.3 Field-Generated Versus Laboratory-Generated Asphalt Fumes

When a large quantity of an asphalt fume is needed, collecting the fumes in a laboratory setting is more practical than collecting fumes at worksites. Niemeier et al. [1988], Lunsford and Cooper [1989], and Reinke and Swanson [1993] studied asphalt fumes generated in the laboratory using the procedure described in section 3.4.2. This procedure involves placing asphalt in a vessel, heating it, and stirring it at least 200 revolutions per minute once it had melted sufficiently. The fumes were allowed to escape until the desired temperature was reached, at which point collection began by pulling air at 10 liters per minute (L/min) through a series of cold traps. (This procedure could account for the differences in chemical composition often noted between asphalt fumes collected in the field and those generated in the laboratory.) The fumes were collected for at least 8 hours.

The following studies evaluated under what conditions laboratory-generated asphalt fumes could mimic asphalt fumes generated in the field.

Kriech and Kurek [1993] showed how generation conditions can affect the composition of fumes. Using a variety of analytical techniques (gas chromatography with flame ionization detection [GC/FID], gas chromatography with flame photometric detection [GC/FPD], gas chromatography with atomic emission detection [GC/AED], and GC/MS), they compared asphalt fumes generated in the laboratory with fume samples collected from the headspace in a storage tank at a hot-mix plant (paving asphalt), from the headspace in kettles (roofing asphalt), and from personal-breathing-zone samples. Both the field and laboratory fumes were collected with a series of cold traps, while the personal-breathing-zone samples were collected at the field sites on a membrane filter backed up with a sorbent tube.

Kriech and Kurek concluded that temperature, rate of stirring, and pulling versus pushing the collection air all affected the chemical composition of the fumes. Based on simulated distillation data and analyses of high-molecular-weight S-PACs, they also concluded that the storage-tank samples resembled the personal-breathing-zone samples more closely than did the laboratory-generated samples. However, the S-PAC data also indicated that the storage-tank samples contained more S-PACs than the personal-breathing-zone samples.

Similarly, Reinke and Swanson [1993] considered asphalt fumes collected from a storage tank at a hot-mix plant to be representative of asphalt fumes from a field paving site. However, Reinke and Swanson [1993] did not give a detailed analysis of the storage-tank samples or the personal-breathing-zone samples. Therefore, these questions remain: Are storage-tank fumes truly representative of the asphalt fumes to

which workers are exposed in the field? Are storage-tank fumes more representative of field fumes than of fumes generated in the laboratory using the NIOSH protocol [Thayer et al. 1981; Sivak et al. 1989]?

In another study, Brandt et al. [1985] collected field and laboratory asphalt fume samples and analyzed them for total particulates, the benzene-soluble fractions of total particulates, and PAHs. In the field, point-emission and personal-breathing-zone (using personal-type samplers) samples were collected at both roofing and paving operations, while the laboratory samples were collected under a variety of conditions. The intent was to identify the conditions under which asphalt fumes generated in the laboratory would be similar to those collected at actual worksites.

Results indicated that temperature and heating time affected chemical composition of the fumes. Higher temperatures and longer heating times resulted in higher exposures to total and benzene-soluble particulates and changed the chromatographic elution profiles of the PAHs. Comparing analyses of the field samples and the laboratory-generated samples led Brandt et al. to conclude that their "laboratory rig" could produce laboratory fumes representative of field fumes. In this study, fumes were collected when the center of the sampler was placed 13 cm above the level of the asphalt. Brandt et al. felt this distance prevented the sampler from influencing the chemical equilibrium above the asphalt surface when the sampler flow rate was 2 L/min. Collection times had to be short (15 to 60 minutes), and generation temperature had to be close to that used in the field.

3.5 Analytical Sampling and Analysis Methods

This section is not intended to be an all-inclusive list of the analytical sampling and analysis methods available for characterizing asphalt vapor and fume exposures. Most of the methods are nonspecific, and none can be used to characterize total asphalt fume exposure.

3.5.1 Total and Respirable Particulates

NIOSH Method 0500 can be used to determine total particulates, and NIOSH Method 0600 can be used to determine respirable particulates [NIOSH 1994]. The only difference between these two methods is that NIOSH Method 0600 uses a size-selective inlet. Both methods are nonspecific; consequently, whatever is deposited on the sampling medium and remains until the sample is analyzed is included in the determination. Moreover, when matrices such as an asphalt fume are sampled, air stripping can cause volatile fume components to be lost from the sampling medium. Because both methods use a membrane filter as the sampling medium, these methods are not useful for collecting vapors.

3.5.2 Benzene-Soluble Fraction of Total Particulates

NIOSH Method 5042 can be used to determine both total particulates and the benzene-soluble fraction of total particulates employing a single sampler [NIOSH 1998]. Previously, benzene solubles and total particulates were determined using different samplers, thus making a comparison of results questionable. Also, the methods used to determine benzene solubles were originally developed for coal-tar-pitch volatiles, and the results were correlated to adverse health effects [Occupational

Safety and Health Administration (OSHA) Method 58, 1986].

These methods of determining benzene solubles have commonly been used with other matrices where the results were only suspected of relating to an adverse health effect. These methods are nonspecific because most organic compounds are soluble in benzene and because asphalt fumes contain many organic compounds and compound classes not found in coal-tar-pitch volatiles. Anything in addition to asphalt fumes that is deposited on the sampling medium and is benzene soluble will interfere with the determination. Air stripping can cause volatile fume components to be lost from the sampling medium. Because NIOSH Method 5042 uses a membrane filter for the sampling medium, it is not useful for collecting vapors.

3.5.3 Polycyclic Aromatic Hydrocarbons

NIOSH Method 5506 uses liquid chromatography with ultraviolet and fluorescence detection (LC/UV/Fl) to determine selected PAHs, and NIOSH Method 5515 uses gas chromatography with a flame ionization detector (GC/FID) to determine selected PAHs [NIOSH 1998; NIOSH 1994]. In NIOSH Method 5506, some of the PAHs (acenaphthene, acenaphthylene, anthracene, chrysene, fluorene, naphthalene, and phenanthrene) are determined by UV detection, and the other PAHs (benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[ghi]perylene, B(a)P, benzo[e]pyrene, dibenz[a,h]anthracene, fluoranthene, indeno[1,2,3-cd]pyrene, and pyrene) are determined by fluorescence. NIOSH Methods 5506 and 5515 have been used to determine selected PAHs in matrices that contain only a few alkylated PAHs in relatively low concentrations compared with the unalkylated PAHs of interest, and possibly a few alkylated and unalkylated N-, O-, and S-PACs. These matrices would include most coal derived products and combustion by-products. However, because asphalt fumes are composed of many alkylated isomers (e.g., mono, di, tri, and tetra-methyl) of PAHs, along with O-PACs and S-PACs, with the exception of naphthalene and some 3-ring PAHs, they are so chemically complex that they cannot be separated into discrete compounds (see section 3.4.2). The greater the lack of resolution between compounds, the less reliable are the quantification results. Because of the poor resolution obtained with asphalt fume samples, quantification is unreliable when these or other HPLC or GC/FID methods are used.

Also, the limitations of NIOSH Methods 5506 and 5515 require that an alternative method (such as GC/MS) be used to confirm the identity of any suspected PAHs, including naphthalene and other possible baseline resolved PAHs. Any compounds reported using NIOSH Methods 5506 and 5515, or similar methods, are tentative identifications at best, and the more complex the matrix, the more unreliable these identifications become. Furthermore, since chromatographic software programs assign peak identification based on the largest peak in a given time window and not on retention time, the wrong peak may be assigned and analyzed. Since these methods use a gradient elution (e.g., the mobile-phase composition varies during the chromatographic run), retention times may vary, thus, further complicating the selection of the correct peak for identification and analysis. For reasons stated above, these problems can be overcome for matrices consisting of coal-derived products or combustion by-products. However, for asphalt fumes, these are formidable problems, because the alkylated PAHs are more abundant and are in higher concentrations than the PAHs of interest.

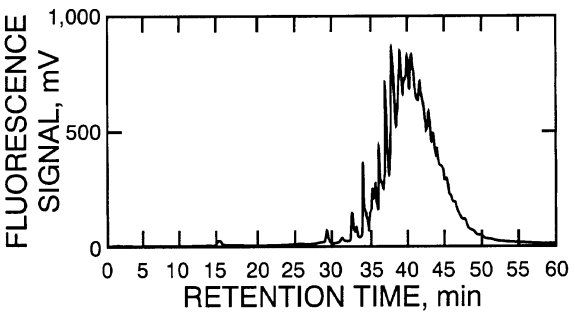

Figure 3–1. Typical asphalt fume chromatogram obtained using liquid chromatography with fluorescene detection.

Figure 3–1 shows a typical asphalt fume chromatogram obtained using liquid chromatography and a fluorescence detector. Figure 3–1 indicates base line resolution is not achieved; hence, this analytical technique should not be used for determining the concentration of PAHs in asphalt fume samples. Therefore, excluding results for naphthalene and some 3-ring PAHs, the analytical results for PAHs previously reported using NIOSH Method 5506 or similar HPLC methods are unreliable. Moreover, since many previously reported studies do not include chromatograms or sufficient experimental details, the methodology and data cannot be critically reviewed; therefore, results for naphthalene and 3-ring PAHs should also be considered suspect. Furthermore, tangent skimming along the oscillations in the chromatogram for resetting the baseline would not be meaningful, because the remaining peak is too small and the peak widths are too wide to represent a single compound.

While NIOSH Method 5506 does not allow for varying the fluorescence excitation and emission wavelengths, these wavelengths can be varied to improve sensitivity; however, varying these wavelengths will introduce added concerns. If the fluorescence response at the new wavelength is not roughly zero, the accompanying autozero that occurs will distort the chromatogram, and the data. For these reasons, PAH analyses in asphalt fumes by HPLC/fluorescence techniques are considered unreliable.

The UV chromatogram obtained by using NIOSH Method 5506 is even more complex because all the PACs and other chemical classes in the asphalt fume sample absorb UV light at the wavelength being monitored (254 nanometers [nm]). NIOSH Method 5515 would produce an even more complex chromatogram since the FID responds to everything passing through it.

Because of these limitations and a growing concern that all PACs in asphalt fume may play a role in adverse health effects, a method is needed to monitor all PAC material. A NIOSH investigation used a modification of NIOSH Method 5506 (i.e., Method 5800) to monitor all PAC material in asphalt fumes [Hanley and Miller 1996a,b; Almaguer et al. 1996; Miller and Burr 1996a,b, 1998; Kinnes et al. 1996; NIOSH 1998]. Basically, the same analytical equipment is used, except the LC column has been removed and the UV detector has been replaced with a second fluorescence detector. Because no LC column is used, the entire sample reaches the flow cell at once, resulting in a rapid and sensitive analysis of the sample. The two fluorescence detectors monitor different excitation and emission wavelengths. One set of wavelengths is more sensitive to two- and three-ring PACs, and the second set of wavelengths is more sensitive to four-ring and higher ring PACs.

3.5.4 Selected Solvent Methods

NIOSH Method 1550 can be used to determine exposure to naphthas [NIOSH 1994]. The term naphthas includes petroleum ether, rubber solvent, petroleum naphtha, VM&P naphtha, mineral spirits, Stoddard solvent, kerosene, and coal tar naphtha. This method may be useful because some liquid asphalt products contain petroleum distillates for which exposure limits have been established. The samples are collected on a sorbent tube and analyzed using GC/FID. Because these solvent mixtures are chemically complex and the components elute over a wide

temperature range, interferences from other substances are possible.

Other NIOSH methods can be used to determine selected solvents that may be present in asphalt vapors and fumes [NIOSH 1994]. NIOSH Methods 1300 and 1301 have been used to determine ketones, and NIOSH Method 1501 has been used to determine total PAHs [Hanley and Miller 1996a,b; Almaguer et al. 1996].

3.6 Conclusions

An analysis of the chemical data indicates that paving and roofing asphalts are qualitatively and quantitatively different; therefore, the vapors and fumes from these asphalt products may also be presumed to be different. Chemical composition of asphalt vapors and fumes varies and depends on crude petroleum sources, type of asphalt, temperature and mixing during the manufacturing process, and temperature and extent of mixing during either laboratory generation or field operations. Although asphalt vapors and fumes have not been well characterized, the analysis of selected PAHs in asphalt vapors and fumes from asphalt products has been of interest. Many studies have been directed to the identification of PAHs in asphalt fume samples. The most meaningful of these studies used GC/MS for the analysis. PAH data obtained by HPLC/ fluorescence techniques are not included, because the PAH identifications are uncertain and the results unreliable, see section 3.5.3.

Robinson et al. [1984] used GC/MS to analyze several asphalt-based paints for chrysene, benz[a]anthracene, B(a)P, benzo[e]pyrene, and phenanthrene; they detected only phenanthrene (0.01%). Several other investigators have reported on the chemical analysis of paving and roofing asphalt fumes [Niemeier et al. 1988; Lunsford and Cooper 1989; Reinke and Swanson 1993; Hatjian et al. 1995a, 1997]. Low levels of carcinogenic PAHs have been detected in laboratory-generated asphalt fumes. Reinke and Swanson [1993] detected 0.02 µg/m^3 chrysene in fumes generated in the laboratory at 149 °C (300 °F). Niemeier et al. [1988] measured low concentrations of several carcinogenic PAHs in roofing asphalt fumes generated in the laboratory at both 232 and 316 °C (450 and 651 °F). Most of the PAHs in the Niemeier et al. study were two-, three-, and four-ring PAHs. Lunsford and Cooper [1989] reported finding two- to four-ring PAHs along with many alkylated PAHs, O-PACs with and without alkyl groups, and S-PACs with and without alkyl groups in laboratory-generated asphalt fume fractions that caused tumors in a mouse-skin-painting study. The presence of O-PACs and S-PACs is a cause for concern, since some O-PACs may cause cancer, and some S-PACs may cause mutations [Tennant and Ashby 1991; Pelroy et al. 1983; McFall et al. 1984]. Also, because little is known about the carcinogenic and genotoxic activity of most of the alkylated PACs, these PACs are a cause for concern because of their structural similarity to known carcinogens and genotoxins.

Few studies have been directed at the identification and measurement of PAHs in asphalt fumes generated at U.S. worksites. Reinke and Swanson [1993] collected paving asphalt fumes from a storage tank at 149 °C (300 °F) at a hot-mix plant (Table 3-6). Although they had detected chrysene in laboratory-generated asphalt fumes, they did not detect chrysene in asphalt fumes collected from the storage tank, and although two- and three-ring PAHs were found in the storage tank fumes, four-ring PAHs were not.

Hatjian et al. [1995a, 1997] reported on a GC/MS analysis for selected PAHs in asphalt paving and roofing fumes collected at several worksites. Two- and three-ring PAHs accounted for 99% of PAH exposures in the two paving asphalt groups. In the two roofing asphalt

groups, two- and three-ring PAHs accounted for 84% and 94% of PAH exposures, respectively. Naphthalene accounted for 60% to 90% of PAH exposures for all work groups. B(a)P was detected in less than 6% of the personal-breathing-zone air samples from asphalt road pavers and manual laborers who had no occupational exposure to PAHs and in 28% or 25% of the personal-breathing-zone air samples obtained from asphalt roofers, R1 and R2, respectively.

In a NIOSH study, environmental samples from paving operations were analyzed for PACs as a class, but no individual PAHs were determined [Hanley and Miller 1996a,b; Almaguer et al. 1996; Miller and Burr 1996a,b, 1998; Kinnes et al. 1996].

While data regarding the presence of carcinogens in asphalt fumes generated at U.S. worksites are limited, the occasional detection of B(a)P at these sites [Hatjian et al. 1995a, 1997] and more frequent detection of B(a)P and other carcinogenic PAHs in laboratory-generated asphalt fumes indicate that under some conditions, known carcinogens are likely to be present [Niemeier et al. 1988; Lunsford and Cooper 1989; Reinke and Swanson 1993]. Moreover, asphalt fumes generated at high temperatures are probably more hazardous than fumes generated at lower temperatures. Because asphalt fume samples collected in the field have not been well characterized, additional research is needed to better characterize them. Also, laboratory generation methods need to be evaluated to identify those that produce asphalt fume samples representative of fumes to which workers are exposed.

The presence of numerous alkylated PAHs, O-PACs, and S-PACs in asphalt fumes is cause for concern. Although little is known about their toxicologic activity, their structural similarity to known carcinogens and genotoxins is troublesome. If these compounds are a health concern, new sampling and analytical methods specifically for these compounds would need to be developed. Given the chemical complexity of asphalt fume samples, the most likely methods would utilize GC/MS techniques.

Various NIOSH methods have been used for characterizing asphalt vapor and fume exposures. However, most of the methods are nonspecific, and none are useful for characterizing total asphalt fume exposure. New or improved analytical sampling methods need to be developed.

4 Exposure

The first part of this chapter discusses three air sampling methods—total particulate, benzene-soluble particulate fraction, and PAHs, all of which have been used in recent NIOSH investigations to evaluate occupational exposures to asphalt fumes [Almaguer et al. 1996; Hanley and Miller 1996a,b; Kinnes et al. 1996; Miller and Burr 1996a,b, 1998]. Table 4–1 provides a summary of these sampling and analytical methods. In addition, worker exposure data from studies evaluating asphalt refining, hot-mix asphalt plants, road paving, roofing (both manufacturing and installation), flooring, and waterproofing are reviewed and summarized in Appendix B.

4.1 Methods for Analyzing Workplace Air and Dermal Exposures

A variety of sample collection and analytical methods are available for evaluating exposures to asphalt fumes in the workplace. Two methods frequently employed measure either total particulates or the benzene-soluble fraction of total particulates. Unfortunately, neither of these methods measures exposure to distinct chemical components or even a distinct class of chemicals, making it difficult to relate specific components to possible health effects. For example, many organic compounds are soluble in benzene, and any dust or aerosols may contribute to total particulate concentrations. In an attempt to characterize asphalt fumes more accurately, investigators have developed methods to measure individual unsubstituted PAHs, such as acenaphthylene, anthracene, and naphthalene; total PACs; or other potentially irritating substances, such as sulfur-containing compounds. These methods are described below.

4.1.1 Total Particulates as an Indicator of Asphalt Fumes

Total particulates are a measure of all airborne particulates that can be collected on a tared (weighed) sample filter. Several current occupational exposure limits for asphalt fumes are expressed as total particulates. In a study at an asphalt hot-mix plant, the size distribution of approximately 95% to 98% of the asphalt particles was shown to be between 1 and 5 µm in diameter, while at an asphalt paving site, where samples were collected above the screed auger of the paver vehicle, approximately 76% of the particles were between 1 and 5 µm in diameter [Hicks 1995]. These data indicate that asphalt fumes are composed of relatively small particles and may be collected equally well using the more traditional sampling method (closed-face, 37-mm sampling cassettes) or inhalable samplers. However, further research is warranted to define the various size fractions of asphalt fumes at paving and other worksites where asphalt is used.

In the 1977 criteria document, NIOSH established a recommended exposure limit (REL) of 5 mg/m^3 as a 15-min ceiling limit[2] for asphalt fumes measured as total particulates. The NIOSH REL was intended to protect workers against acute effects of exposure to asphalt fumes, including irritation of the serous membranes of the conjunctivae and the mucous membranes of the respiratory tract. In 1988, NIOSH (in testimony to the Department of Labor) recommended that asphalt fumes should be considered a potential occupational carcinogen [NIOSH 1988].

[2]See footnote 1 in chapter 1.

Table 4-1. Examples of sampling and analytical methods for characterizing occupational exposure to asphalt fumes [NIOSH 1994]

Substance	Sample media	Analytical method	Additional information
Total particulates	Tared PVC filter (37-mm diam, 0.8-µm pore size) *or* tared Zefluor filter (37-mm diam, 1-µm pore size)	Tared filter (either PVC or PTFE) is gravimetrically analyzed. Note: filters should be allowed to equilibrate in an environmentally controlled weighing area or chamber. The LOD and LOQ for total particulates were 0.04 and 0.13 mg per sample, respectively.	NIOSH Sampling and Analytical Method No. 5042 for TP and benzene-soluble fraction (asphalt fumes) recommends using a tared PTFE filter. This allows simultaneous measurement of both TP and BSP.
Benzene-soluble particulates	Tared PTFE filter (3-mm diam, 1-µm pore size)	The PTFE filters are rinsed with benzene, leachate collected and evaporated, and residue weighed to report benzene-soluble fraction. The LOD and LOQ for benzene solubles were 0.04 and 0.14 mg per sample, respectively.	Organic compounds are generally soluble in benzene. Sampling for BSP (or TP) assumes that the process producing the asphalt fumes is the predominant contributor to air pollution at the worksite.
Polycyclic aromatic compounds and sulfur compounds	PTFE filter (37-mm diam, 2-µm pore size), followed by an ORBO 42 sorbent tube	After collection, asphalt fume samples are extracted with hexane and then eluted through a solid-phase extraction column to separate aliphatic and aromatic compounds from compounds with polar functional groups. PACs are quantitated using reversed-phase liquid chromatography with fluorescence detection. Since excitation and emission wavelengths are not the same for all PACs, two sets of excitation and emission wavelengths are used. Sulfur compounds are subsequently analyzed by GC with sulfur chemiluminescence detection.	NIOSH Sampling and Analytical Method No. 5800 contains more details on collection and analysis of PACs. This method is similar to NIOSH Sampling and Analytical Method No. 5506, Polynuclear Aromatic Hydrocarbons. Opaque filter cassettes and sorbent tube holders are recommended to prevent degradation of PACs by UV. For more information, refer to section 3.5.3.

Abbreviations: BSP=benzene-soluble particulates; GC=gas chromatography; HPLC=high-performance liquid chromatography; LOD=limit of detection; LOQ=limit of quantitation; PTFE=polytetra-fluoroethylene (Teflon®); PVC=polyvinyl chloride; TP=total particulates; UV=ultraviolet radiation.

Currently, no OSHA standard exists for asphalt fumes. In a 1988 proposed rule on Air Contaminants, OSHA proposed a PEL of 5 mg/m^3 as an 8-hr time-weighted average (TWA) for asphalt fume exposures in general industry. This proposal was based on a preliminary finding that asphalt fumes should be considered a potential carcinogen [53 Fed. Reg. 21193 (1988)]. In 1989, OSHA announced that it would delay a final decision on the 1988 proposal because of complex and conflicting issues submitted to the record [54 Fed. Reg. 2679 (1989)]. In 1992, OSHA published another proposed rule for asphalt fumes that included a PEL of 5 mg/m^3 (total particulates) for general industry, construction, maritime, and agriculture [57 Fed. Reg. 26182 (1992)]. Although OSHA invited comment on all of the alternatives, its proposed standard for asphalt fumes would establish a PEL of 5 mg/m^3 (total particulates) based on avoidance of adverse respiratory effects. The OSHA docket is closed, and OSHA has not scheduled any further action.

The current American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value (TLV®) for asphalt fumes is 0.5 mg/m^3 (8-hr TWA) as a benzene-soluble aerosol (inhalable fraction) or equivalent method with an A4 designation, indicating that it is not classifiable as a human carcinogen [ACGIH 2000]. Irritation is the critical effect.

4.1.2 Benzene-Soluble Particulate Fraction

The benzene-soluble particulate fraction is that portion of total particulates that is soluble in benzene. Organic compounds are generally soluble in benzene, whereas inorganic compounds are not. Historically, this particulate fraction has been used to differentiate between asphalt fumes and other nonasphalt particulates present, such as road dust, at paving sites. Of course, sampling for benzene solubles (or total particulates) assumes that asphalt fumes (as opposed to diesel engine exhaust, for example) are the predominant or sole contributor to air pollution at a worksite. NIOSH Sampling and Analytical Method 5042 contains further details on the collection and analysis of total particulates and benzene solubles.

In the past, because of concerns with the carcinogenicity of benzene, other solvents (such as cyclohexane, acetonitrile, and methylene chloride) have been used in place of benzene to measure the soluble fraction of a particular matrix. When sampling asphalt fumes, however, it is difficult to compare the results because the extraction capability of these solvents varies. For example, carbon disulfide may not be as effective as benzene for extracting the polar compounds in the fumes. NIOSH researchers believe that benzene provides the best overall solubility for asphalt fumes.

4.1.3 Polycyclic Aromatic Hydrocarbons and Polycyclic Aromatic Compounds

In many asphalt fume studies, researchers have attempted to analyze individual PAHs using either LC/UV/FID or GC/FID. Although this approach has been successful in many matrices containing PAHs, studies of asphalt fumes have shown that these fumes contain a complex mixture of PACs, a class of chemical compounds that contain two or more fused aromatic rings. NIOSH researchers believe that, on an individual basis, these PACs cannot be easily separated or quantified (see section 3.5.3).

In response to this analytical dilemma, NIOSH researchers developed a flow-injection method (NIOSH Method 5800) to measure the total PAC content of asphalt fumes [Miller and Burr 1998, Appendix A]. After it is collected, the asphalt fume sample is extracted from the sampling filter with hexane. This extract is then eluted through a solid-phase extraction column to separate the aliphatic and aromatic compounds. The aromatic compounds are then extracted from the aliphatic compounds using a liquid-liquid extraction procedure.

Because the excitation and emission wavelengths are not the same for all PACs, two sets of excitation and emission wavelengths were used in seven asphalt paving studies conducted by NIOSH [Almaguer et al. 1996; Hanley and Miller 1996a,b; Kinnes et al. 1996; Miller and Burr 1996a,b, 1998]. One set of wavelengths (254-nm excitation, 370-nm emission) is more sensitive for two-ring and three-ring compounds (the lower molecular weight PACs); the second set of wavelengths (254-nm excitation, 400-nm emission) is more sensitive for four-ring and higher compounds (the higher molecular weight PACs). It should be noted that other researchers [Kriech et al. 1999; Kurek et al. 1999] are using similar techniques for measuring the presence or absence of four- to six-ring PACs in asphalt fumes. No occupational exposure limits have been established for total PACs associated with asphalt fumes. NIOSH Sampling and Analytical Method 5800 contains further details on the collection and analysis of PACs.

4.2 Occupational Exposure Data, Air and Dermal Wipe Sampling

Comparing historical occupational exposure data from asphalt fume studies can be complicated by many factors, including the complex and variable nature of the asphalt itself, the lack of a single chemical substance accepted as representative of asphalt fume exposure, and the use of different sampling and analytical methods. This last factor is important in terms of assessing exposures because such differences can affect what markers are measured for asphalt fume exposure and how comparable the results of different studies are. For example, studies of asphalt fumes may report individual PAHs or total PAHs, but the analytical methods used to obtain results may vary in accuracy and PAH identifications are unreliable (see section 3.5). Also, when solvents other than benzene, such as cyclohexane or acetonitrile, are used to obtain the soluble fraction of total particulates, the results cannot be compared easily because the extraction ability of these solvents varies.

Because of the potential problems encountered when combining results from studies in which sampling and analytical methods differ, environmental data obtained from studies of asphalt refining, hot-mix asphalt plants, road paving, roofing, flooring, and waterproofing are summarized by topic in Appendix B. Analysis of these data indicated that the highest personal total particulate exposures were measured during asphalt flooring and waterproofing activities (1.1 to 42 mg/m^3), followed by roofing products manufacturing (0.07 to 15 mg/m^3), asphalt refining (0.3 to 14 mg/m^3), roofing application (0.04 to 13 mg/m^3), activities at hot-mix asphalt plants (0.1 to 7.2 mg/m^3), and road paving (0.1 to 5.6 mg/m^3). Personal exposures to benzene-soluble particulates followed a similar pattern, with the highest exposures once again being measured during asphalt flooring and waterproofing activities (0.8 to 14 mg/m^3), followed by asphalt refining (0.03 to 13 mg/m^3), roofing application (0.04 to 6.9 mg/m^3), road paving (0.03 to 4.4 mg/m^3), and roofing products manufacturing (0.01 to 3.7 mg/m^3). The following section discusses several recent asphalt exposure studies in greater detail.

4.2.1 NIOSH/FHWA Evaluation of Asphalt Paving Workers

Between 1994 and 1997, seven surveys [Almaguer et al. 1996; Hanley and Miller 1996a,b; Kinnes et al. 1996; Miller and Burr 1996a,b, 1998] were completed as part of an interagency agreement between NIOSH and the Federal Highway Administration (FHWA) of the U.S. Department of Transportation. The objectives were to (1) develop and field test new methods of characterizing asphalt fume exposures and (2) identify potential health effects associated with asphalt exposures (health effects are discussed in section 5).

At each NIOSH survey site, full-shift personal-breathing-zone samples were collected from the

paving crew, which typically consisted of six to 10 workers, for total particulates and the benzene-soluble particulate fraction. Table 4–2 shows that average personal-breathing-zone air concentrations for both total particulates and benzene solubles were below 0.5 mg/m^3, TWA. Table 4–3 shows personal-breathing-zone air concentrations for PACs that were collected and analyzed using a method similar to NIOSH Sampling and Analytical Method No. 5506 (see Table 4–1 and section 3.5.3 for more information). Two spectrofluorometric emission wavelengths were used in the PAC analyses. These were 370 nm, which is more sensitive to the lower molecular weight, two–ring and three–ring PAC compounds (termed PAC_{370}); and 400 nm, which is more sensitive for the higher molecular weight, four–ring and larger compounds (referred to as PAC_{400}). In these studies, concentrations of PAC_{370} always exceeded concentrations of PAC_{400}, implying that the lower molecular weight, two- and three-ring PACs (postulated by NIOSH investigators to be more responsible for irritant effects) may be more abundant in asphalt fumes.

In addition to the personal-breathing-zone samples, area air samples were collected over the screed auger section of the paver vehicle and analyzed for total particulates, respirable particulates, benzene solubles, and total hydrocarbons. Area air samples were also collected for VOCs, carbon monoxide, hydrogen sulfide, sulfur dioxide, and ozone, substances which NIOSH investigators theorized could also be present during road paving.

Area air sampling results for respirable and total particulates, benzene-soluble particulates, and total hydrocarbons are summarized in Table 4–4. Across the seven paving sites, area concentrations of respirable particulates at the screed auger ranged from 0.055 to 0.97 mg/m^3, total VOCs (measured as either n-hexane or Stoddard solvent) ranged from 0.5 to 30 mg/m^3, and concentrations of selected individual VOCs (benzene, toluene, xylene, and methyl isobutyl ketone) were generally less than 1 part per million (ppm). At some sites, area concentrations of carbon monoxide ranged up to 1,000 ppm where gasoline–powered equipment, such as vibrating tampers or portable generators, was in use. At all survey locations, concentrations of hydrogen sulfide and sulfur dioxide were not detected.

4.2.2 NIOSH Evaluation of Asphalt Paving among Tunnel Workers

In 1996, NIOSH evaluated exposures of paving crews working within the Third Harbor Tunnel in Boston, MA [Sylvain and Miller 1996]. The work included the collection of full-shift personal-breathing-zone and area air samples for total particulates and benzene solubles, questionnaires administered to obtain information on symptoms, and tests of peak lung flow (see section 5.1 for details on the medical results). As shown in Table 4–5, personal exposures to total particulates and benzene solubles averaged 1.6 and 0.76 mg/m^3, respectively. These concentrations were up to three times higher than exposures measured during the seven NIOSH/FHWA surveys at open-air roadway paving sites (see section 4.2.1). Poorer ventilation in the tunnel (as compared to open-air paving sites) likely contributed to these higher personal-breathing-zone exposures.

4.2.3 Cross-Sectional Occupational Exposure Assessment Study

In a cross-sectional occupational exposure assessment [Hicks 1995] covering road paving sites, hot-mix plants, refineries and terminals, roofing manufacturing plants, and roofing application sites, 219 full-shift personal-breathing-zone air samples were collected and analyzed (most sampling periods ranged from 7 to 9 hours) [Hicks 1995]. In addition to air samples, 131 dermal wipe samples for benzene solubles were collected (see section 4.3). The objective of this study was to characterize worker exposures to asphalt fumes via both airborne and dermal routes.

Table 4–2. Full-shift personal-breathing-zone sample results for total particulates* and benzene-soluble particulates obtained from NIOSH paving surveys, mg/m³

Occupation	Arizona			California 1			California 2			Florida			Indiana			Massachusetts			Michigan		
	n	GM	Max.	*n*	GM	Max.	*n*	GM	Max.	*n*	GM	Max.	*n*	GM	Max.	*n*	GM	Max.	*n*	GM	Max.
Paver operators:																					
Total particulates	2	0.8	1.0	2	0.85	1.3	2	0.62	0.55	2	0.39	0.5	1	0.0087	0.0087	4	0.34	0.52	2	0.17	0.2
Benzene-soluble particulates	2	0.59	0.82	NC	—	—		0.33	0.46	NC	—	—	NC	—	—	4	0.22	0.4	NC	—	—
Screed auger operators:																					
Total particulates	2	0.43	0.47	2	0.31	0.31	4	0.70	1.0	8	0.1	0.17	1	0.78	0.3	2	0.22	0.27	4	0.12	0.17
Benzene-soluble particulates	2	0.29	0.37	NC	—	—	4	0.19	0.21	NC	—	—	NC	—	—	2	0.082	0.099	NC	—	—
Roller operators:																					
Total particulates	5	0.053	0.17	4	0.21	0.35	2	0.18	0.22	4	0.057	0.14	6	0.04	0.1	4	0.055	0.1	6	0.10	0.15
Benzene-soluble particulates	5	0.022	0.06	NC	—	—	2	0.014	0.02	NC	—	—	NC	—	—	4	0.03	0.045	NC	—	—
Laborers/other:†																					
Total particulates	5	0.33	0.68	7	0.27	0.38	8	0.48	0.89	4	0.077	0.13	4	0.031	0.09	4	0.16	0.19	10	0.22	1.2
Benzene-soluble particulates	5	0.17	0.49	NC	—	—	8	0.13	0.32	NC	—	—	NC	—	—	4	0.055	0.8	NC	—	—
Overall (all paving job titles combined):																					
Total particulates	14	0.2	1.0	15	0.30	1.3	16	0.48	1.0	18	0.075	0.17	15	0.041	0.1	14	0.15	0.52	22	0.16	1.2
Benzene-soluble particulates	14	0.11	0.82	NC	—	—	16	0.12	0.46	NC	—	—	NC	—	—	14	0.073	0.4	NC	—	—

Abbreviations: GM=geometric mean; Max.=maximum concentrations for sample set; *n*=number of samples in sample set; NC=not collected.

*Total particulate samples were collected on either tared PVC or PTFE filters.

†This category includes laborers, rakers, haulage truck drivers, traffic personnel, site foremen, and tack men.

Table 4–3. Full-shift personal-breathing-zone sample results for PACs* obtained from NIOSH paving surveys, µg/m³

Occupation		Arizona			California 1			California 2			Florida			Indiana			Massachusetts			Michigan		
		n	GM	Max.	*n*	GM	Max.	*n*	GM	Max.	*n*	GM	Max.	*n*	GM	Max.	*n*	GM	Max.	*n*	GM	Max.
Paver operators:																						
PAH	C_{370}	2	30	49	NC	—	—	2	18	24	1	2.7	—	2	1.8	2.8	2	60	84	NC	—	—
PAH	C_{400}	2	4.3	6.5	NC	—	—		2.4	3.3	1	0.43	—	2	0.27	0.47	2	8.5	12	NC	—	—
Screed auger operators:																						
PAH	C_{370}	2	9.1	23	NC	—	—	4	17	26	4	1.5	3.4	4	0.87	1.2	2	72	191	1	3.9	—
PAH	C_{400}	2	1.3	3.5	NC	—	—		2.3	3.5	4	0.20	0.36	4	0.15	0.28	2	9.8	25	1	1.2	—
Roller operators:																						
PAH	C_{370}	5	0.18	1.2	2	1.4	2.4	2	1.1	1.3	1	1.1	—	6	0.07	0.8	4	5.3	17	NC	—	—
PAH	C_{400}	5	0.04	0.18	2	0.25	0.43	2	0.15	0.16	1	0.17	—	6	0.01	0.042	4	0.67	3.4	NC	—	—
Laborers/other:†																						
PAH	C_{370}	5	7.9	20	NC	—	—	8	8.1	12	NC	—	—	3	0.39	0.54	2	16	22	2	11	—
PAH	C_{400}	5	1.2	2.7	NC	—		8	1.1	1.6	NC	—		3	0.09	0.11	2	2.4	2.9	2	3.0	4.2
Overall (all paving job titles combined):																						
PAH	C_{370}	14	2.5	49	2	1.4	2.4	16	8.4	26	6	1.5	3.4	15	0.3	2.8	10	22	191	3	7.6	16
PAH	C_{400}	14	0.43	6.5	2	0.25	0.43	16	1.1	3.5	6	0.21	0.43	15	0.05	0.47	10	2.9	25	3	2.2	4.2

Abbreviations: GM=geometric mean; Max.=maximum concentrations for sample set; n=number of samples in sample set; NC=not collected.

*Total particulate samples were collected on either tared PVC or PTFE filters.

†This category includes laborers, rakers, haulage truck drivers, traffic personnel, site foremen, and tack men.

Table 4-4. Summary of results from NIOSH paving surveys of full-shift area air sampling at screed auger, mg/m^3

Substance	Arizona			California 1			California 2			Florida			Indiana			Massachusetts			Michigan		
	n	GM	Max.	*n*	GM	Max.	*n*	GM	Max.	*n*	GM	Max.	*n*	GM	Max.	*n*	GM	Max.	*n*	GM	Max.
Total particulates*	8	1.3	5.5	2	2.0	3.2	8	2.4	3.0	4	0.25	0.45	4	0.14	0.25	8	1.6	1.9	2	0.29	0.45
Respirable particulates	4	0.86	1.4	2	1.5	3.1	4	0.43	1.2	4	0.11	0.22	4	0.07	0.17	4	0.58	0.77	2	0.11	0.16
Benzene-soluble particulates	8	1.1	5.1	2	2.0	3.0	8	1.7	2.4	4	Trace	—	4	0.19	0.26	4	1.2	1.2	2	0.14	0.22
Total VOCs (as n–hexane)†	4	0.73	1.5	2	1.2	2.1	4	0.56	1.1	4	0.19	0.31	4	0.06	0.17	4	1.9	2.3		NAn	NAn
Total VOCs (as Stoddard solvent)‡	4	22	74	2	7.7	9.0	4	13	25	4	3.8	8.2	4	2.5	2.9	4	21	24	2	0.57	0.57

Abbreviations: Max.=maximum concentrations for sample set; n=number of samples in sample set; NAn=not analyzed; VOCs=volatile organic carbons.

*Total particulate samples were collected on either tared PVC or PTFE filters.

†Represents total hydrocarbons having a retention time less than toluene.

‡Represent total hydrocarbons having a retention time greater than toluene.

As shown in Table 4–6, concentrations of total particulates (0.18 to 1.4 mg/m^3) and benzene solubles (0.15 to 0.27 mg/m^3) varied across all industry types. Geometric mean exposures in all sectors were comparable when measured as benzene solubles. The highest concentration for total particulates was at roofing manufacturing plants, but this result may be attributable to nonasphalt-related particles in these plants. The air samples with the most abundant PAH compounds were obtained from workers at construction sites (roofers and paving crews) (Table 4–7). Hicks reported that lower molecular weight PAHs, such as naphthalene, were more frequently detected than the higher molecular weight compounds, such as B(a)P. Carcinogenic PAHs (chrysene, B(a)P, and benzo(b) fluoranthene) were detected in personal samples collected from employees working in the industry categories of refineries and terminals, roofing manufacturing, roofing contractors, and paving operations. Fluorene, naphthalene, and phenanthrene were detected in all of the industry categories. It should be noted that HPLC with an ultraviolet/fluorescence detector (the method used in the Hicks study) may not be able to distinguish discrete PAHs present in asphalt fumes. See section 3.5 for a more complete discussion of the analysis of asphalt fumes.

4.2.4 Exxon Cross-Sectional Evaluation of Asphalt Workers

Personal exposures and health outcomes of 170 workers in five segments of the asphalt industry (hot-mix plants, terminals, roofing

Table 4–5. Personal-breathing-zone sample results for total particulates and benzene-soluble particulates, mg/m^3 (adapted from Sylvain and Miller 1996)

		Total particulates		Benzene-soluble particulates	
Job	No. of samples	Geometric mean	Maximum	Geometric mean	Maximum
Paver operator	1	1.9		1.1	
Screed operator	1	1.5		0.91	
Roller operator	1	2.1		0.87	
Laborers*	6	1.5	2.2	0.44	1.3
Overall	9	1.6	2.2	0.76	1.3

*Group included 4 rakers and 2 laborers.
NOTE: Maximum=maximum concentrations for sample set.

Table 4–6. Personal-breathing-zone sample results for total particulates and benzene-soluble particulates, mg/m^3 (adapted from Hicks 1995)

		Total particulates		Benzene-soluble particulates	
Type of industry	No. of samples	Geometric mean	Maximum	Geometric mean	Maximum
Refineries/terminals	44	0.18	14	0.16	13
Hot-mix asphalt facilities	33	0.78	15	0.15	1.7
Paving operations	37	0.37	0.85	0.24	4.4
Roofing manufacturers	34	1.4	13	0.27	3.7
Roofing contractors	38	0.55	2.5	0.25	2.4

NOTE: Maximum=maximum concentrations for sample set.

Table 4–7. Geometric mean of personal-breathing-zone air samples* for PAHs, µg/m³ (adapted from Hicks 1995)

Type of industry	ACN†			ACY†			ANT†			BAP‡			BBF‡			BEP‡		
	No. of samples§	GM	Max.	No. of samples	GM	Max.	No. of samples	GM	Max.	No. of samples	GM	Max.	No. of samples	GM	Max.	No. of samples	GM	Max.
Refineries/terminals	9/1	0.99	3.4	9/2	0.54	4.9	9/1	0.047	2	44/1	0.15	1.3	9/1	0.12	4.2	9/1	0.29	3.2
Hot-mix asphalt facilities	8/2	0.9	2.1	8/0	ND	<0.57	8/0	ND	<0.036	33/0	ND	<0.14	8/0	ND	<0.09	8/0	ND	<0.16
Paving operations	9/4	1.3	2.7	9/3	0.69	4.5	9/3	0.05	1.5	37/0	ND	<0.14	9/0	ND	<0.09	9/1	0.16	1.2
Roofing manufacturers	7/1	0.9	2.4	7/3	0.95	5.1	7/0	ND	<0.036	34/1	0.16	1.1	7/0	ND	<0.09	7/0	ND	<0.16
Roofing contractors	11/1	0.74	1.6	11/7	2.0	2	11/1	0.04	1.2	38/0	ND	<0.14	11/3	0.11	1.8	11/4	0.34	2.8

	CHR‡			FLA‡			FLE†			NAP†			PHN†			PYR‡		
Refineries/terminals	9/0	ND	<0.11	9/1	0.18	0.18	9/1	0.25	2.5	9/8	4.1	3.0	9/2	0.13	4.5	9/0	ND	<0.14
Hot-mix asphalt facilities	8/0	ND	<0.11	8/2	0.15	1.8	8/1	0.25	2.2	8/2	1.5	2.8	8/4	0.16	2.4	8/0	ND	<0.14
Paving operations	9/2	0.13	1.3	9/8	0.24	1.5	9/8	0.51	1.8	9/8	5.4	2	9/8	0.43	2.3	9/2	0.17	1.3
Roofing manufacturers	7/0	ND	<0.11	7/0	ND	<0.11	7/3	0.35	2.1	7/5	5.1	2.6	7/6	0.26	2.40	7/2	0.21	1.8
Roofing contractors	11/0	ND	<0.11	11/5	0.29	3.1	11/6	0.77	4.9	11/7	3.2	3.9	11/6	0.28	3.4	11/3	0.34	3.4

Abbreviations: ACN=acenaphtlene; ACY=acenaphthylene; ANT=anthracene; BAP=benzo(a)pyrene; BBF=benzo(b)fluoranthene; BEP=benzo(e)pyrene; CHR=chrysene; FLA=fluoranthene; FLE=fluorene; GM=geometric mean; Max.=maximum concentration for a sample set; NAP=naphthalene; ND=not detected (below the practical quantitation limit); PHN=phenanthrene; PYR=pyrene.

*These personal breathing-zone samples were analyzed using high-performance liquid chromatography with an ultraviolet/fluorescence detector. We have included these data for completeness; however, because of the asphalt fume matrix and the analytical technique used to evaluate the PAHs, the PAH identifications and the concentration data are considered to be unreliable. See section 3.5.3 for more information.

†PAHs with 2–3 rings.

‡PAHs with 4–rings.

§Total number of samples analyzed per industry category, followed by the number of samples above the practical quantitation limit (defined as the upper confidence limit).

manufacturing plants, roofing application sites, and paving sites) were evaluated by Exxon Biomedical Sciences [Exxon 1997]. Gamble et al. [1999] have published a summary of these data. Personal samples were collected across two work days for total and respirable particulates and the benzene-soluble fraction of total particulates. Samples were also collected for VOCs, nitrous oxide, hydrogen sulfide, sulfur dioxide, and ozone. The health outcomes measured included changes in lung function between shifts and the administration of a questionnaire on symptoms (see section 5.1.3).

Full-shift personal-breathing-zone concentrations ranged up to 6.2 mg/m^3; respirable particulates up to 1.4 mg/m^3; benzene solubles up to 1.3 mg/m^3; and VOCs up to 20 mg/m^3. Table 4–8 summarizes the geometric means and maximum exposures to total particulates, respirable particulates, benzene-soluble particulates, and VOCs by industry. Concentrations of nitrous oxide, hydrogen sulfide, and sulfur dioxide were typically near or below detection limits of the analytical methods used. Ozone concentrations were below 100 parts per billion.

4.2.5 Occupational Dermal Exposures

Dermal exposure to asphalt fumes has been examined using skin wipes, which represent the potential contribution of dermal exposure to total body burden. Wolff et al. [1989] collected 10 skin-wipe and nine personal-breathing-zone samples from 10 roofers who had removed an old coal-tar-pitch roof and replaced it with an asphalt roof. PAHs were detected in samples from the breathing zones of employees involved with applying asphalt on two separate days (5.8 and 22 µg/m^3, mean) and removing coal-tar pitch (9.6 and 23 µg/m^3, mean). Because NIOSH Sampling and Analytical Method No. 5506 was used, the PAH identifications and the concentration data are considered to be unreliable; however, these data are included for completeness. PAH residues per square centimeter of skin were higher in postshift samples (6.1 to 31 nanograms per square centimeter [ng/cm^2]) than in preshift samples (0.44 to 2.2 ng/cm^2). Eight of nine cases showed a significant correlation (r=0.97) between PAHs found in personal air samples and in postshift skin wipe residues. However, employees monitored during the entire roofing application were potentially exposed to PAHs during both the removal of the old coal-tar-pitch roof and the application of hot asphalt for the new roof.

One-hundred thirty-one postshift dermal wipe samples were collected from workers at refineries, hot-mix facilities, paving sites, roofing manufacturing plants, and roofing sites and analyzed for PAHs in an exposure assessment study sponsored by the Asphalt Institute [Hicks 1995]. These samples were obtained by wiping the foreheads or backs of hands of selected workers with premoistened smear tabs and then analyzing the wipes for the PAH species listed in Table 4–9.

Table 4–8. Geometric mean of personal exposures by industry, mg/m^3 (adapted from Exxon 1997)

Substance	HMA manufacturing	Asphalt distribution	Roofing manufacturing	Roofing application	HMA paving
No. of samples	20	47	77	60	80
Total particulates	0.45 (1.3)	0.19 (2.5)	0.60 (6.2)	0.34 (2.7)	0.33 (1.7)
Respirable particulates	0.10 (0.60)	0.06 (0.16)	0.08 (0.56)	0.14 (1.4)	0.1 (1.1)
Benzene-soluble particulates	0.06 (0.14)	0.05 (1.3)	0.08 (1.3)	0.12 (1.2)	0.09 (0.65)
Total volatile organic compounds	1.1 (6.3)	1.6 (20)	0.70 (8.7)	0.30 (6.7)	0.38 (7.7)

HMA=hot-mix asphalt.
NOTE: Number in parentheses indicates maximum concentrations for sample set.

The PAH concentrations determined from these postshift samples ranged from 2.2 to 520 ng/cm^2. Employees in paving operations produced the largest number of PAHs detected (12), while refinery and roofing installation workers had the fewest (2). Naphthalene was detected at all sites. Table 4–10 shows the six PAHs (of the 17 PAHs analyzed within this sample set) that were above the detection limit.

NIOSH investigators have collected preshift and postshift skin wipe samples during paving operations at three separate locations [Zey 1992a,b,c]. The samples were analyzed as described in Wolff et al. [1989]. No PAHs were detected in any of the skin wipe samples, which may have been because of low concentrations of asphalt fumes during paving operations and because the PAH concentrations were below the detection limit of the analytical method.

In Zhou [1997], pre-and postshift hand wipes were collected from a group of 17 asphalt-exposed road pavers and 16 controls to evaluate dermal PAH exposure. These hand-wipe samples were analyzed by HPLC fluorescence for the following nine PAHs: anthracene, fluoranthene, pyrene, benzo(b)fluoranthene, benzo (k)fluoranthene, dibenz(a,h)anthracene, benz(a) anthracene, chrysene, and B(a)P. Zhou reported that among the group exposed to asphalt, total PAH, carcinogenic PAH, and pyrene concentrations increased when pre- and postshift hand-wipe samples were compared (Table 4–11.)

Table 4–9. PAH species analyzed from 131 skin wipe samples (adapted from Hicks 1995)

Acenaphthene
Anthracene
Benz(a) anthracene
Benzo(a)pyrene*
Benzo(b)fluoranthene
Benzo(e)pyrene
Benzo(ghi)perylene
Benzo(k)fluoranthene
Chrysene*
Dibenz(a,h)anthracene*
Fluoranthene
Fluorene
Indo(1,2,3-cd)pyrene
Naphthalene
Phenanthrene*
Pyrene*

*All samples were analyzed for these PAH species; 20% of these samples were also analyzed for the remaining PAHs.

NOTE: These skin wipe samples were analyzed using HPLC fluorescence. We have included these data for completeness; however, because of the asphalt fume matrix and the analytical technique used to evaluate the PAHs, the PAH identifications and the concentration data are considered to be unreliable. See section 3.5.3 for more information.

4.2.6 Summary

Based on results of studies of open-air paving sites, refineries, asphalt distribution terminals, and hot-mix asphalt plants, mean personal airborne exposures to asphalt fumes were generally below 1.0 mg/m^3 for total particulates and 0.3 mg/m^3 for benzene solubles, calculated as a full-shift TWA (Table 4–12). Full-shift TWA personal exposures measured during some activities, such as underground paving, roofing manufacturing, and roofing application, however, were higher, ranging up to 1.6 mg/m^3 for total particulates and up to 0.76 mg/m^3 for benzene solubles.

While PAH data were included for completeness, the results are not provided in this summary because PAH identifications and concentration data are considered to be unreliable. See section 3.5.3 for more information.

4.3 Biomarkers

In addition to measures of ambient exposures to occupational chemicals, various studies have used readily accessible body fluids and/or physiological functions as biomarkers for exposure to asphalt fumes. Urinary thioether excretion, glucaric acid metabolites in urine, detection of mutagens in urine, sister chromatid exchange and primary DNA damage in lymphocytes, urinary 1-hydroxypyrene, and DNA or protein adducts have been described as indicators of exposure to or effects of asphalt fumes.

Table 4–10. Skin wipe results for PAHs, ng/cm^2 (adapted from Hicks 1995)

Type of industry	No. of samples	CHR	DBA	FLA	NAP	IDP	PHN
Refineries/terminals	26	ND (<3.3)	ND (<0.35)	ND (<3.3)	5.5-290	ND (<4.5)	ND (<2.2)
HMA facilities	25	ND (<3.3)	ND (<0.35)	ND (<3.3)	390*	19*	3.2*
Paving operations	30	6.2*	<350	4.7*	430*	320*	<2.2-13
Roofing manufacturers	29	ND (<3.3)	ND (<0.35)	ND (<3.3)	<5.5-160	<4.5-25	2.4*
Roofing contractors	21	ND (<3.3)	ND (<0.35)	ND (<3.3)	510-520	ND (<4.5)	ND (<2.2)

Abbreviations: CHR=chrysene; DBA=dibenz(a,h)anthracene; FLA=fluoranthene; HMA=hot-mix asphalt; IDP=indol(1,2,3-cd) pyrene; NAP=naphthalene; ND=not detected; PHN=phenanthrene.
*Single sample revealed detectable results.
NOTE: Numbers in parentheses indicate minimum detectable concentrations.
NOTE: These skin wipe samples were analyzed using HPLC fluorescence. We have included these data for completeness; however, because of the asphalt fume matrix and the analytical technique used to evaluate the PAHs, the PAH identifications and the concentration data are considered to be unreliable. See section 3.5.3 for more information.

Table 4–11. Postshift skin wipe results, ng/cm^2 (adapted from Zhou 1997)

Employee group	Total PAH	Carcinogenic PAH*	Pyrene
Postshift	0.63 to 6.9	0.067 to 1.4	0.25 to 5.5
Preshift	0.10 to 3.3	0.014 to 0.86	ND (<0.37) to 0.73

ND=not detected (below the limit of detection).
*Carcinogenic PAH was calculated by summing benzo(b)fluoranthene, benzo(k)fluoranthene, dibenz(a,h)anthracene, benz(a)anthracene, chrysene, and benzo(a)pyrene.
NOTE: These skin wipe samples were analyzed using HPLC fluorescence. We have included these data for completeness; however, there is concern about the use of this analytical method for evaluating individual PAHs. See chapter 3.5.3 for more information.

4.3.1 Urinary Thioethers

Urinary thioethers have been proposed as potential biomarkers of internal exposure to electrophilic compounds [Van Doorn et al. 1981]. The glutathione-S-transferase (GST) enzyme system facilitates the conjugation of glutathione with electrophilic agents. This conjugation step usually results in detoxification of the agent and enhances its elimination in bile or urine. In addition to xenobiotic agents, numerous endobiotic materials are also conjugated to glutathione by GST. When compounds are conjugated with glutathione, mercapturic acids and other thioethers appear in the urine as nonspecific indicators of exposure to electrophilic agents.

Numerous researchers have attempted to correlate asphalt exposure (both road paving and roofing operations) to increased urinary thioether excretion [Lafuente and Mallol 1987; Burgaz et al. 1988, 1992; Pasquini et al. 1989; Hatjian et al. 1995a, 1997]. These efforts have been unsuccessful. Even in limited cases where potential correlations may have existed, values were within normal human ranges.

4.3.2 Urinary Glucaric Acid

Glucaric acid excretion is another indirect measure of exposure to materials eliminated by conjugation. Like thioethers, many endobiotic agents are conjugated with glucuronic acid for transport and elimination by organisms. In theory, increased exposure to agents that are made less toxic via glucuronidation should result in increased elimination that could be estimated by glucaric acid excretion.

Table 4–12. Summary of full-shift personal-breathing-zone samples for total particulates and benzene-soluble particulates, mg/m^3

Studies	No. of samples	No. of sites sampled	Total particulates: Geometric mean	Total particulates: Maximum	Benzene-soluble particulates: Geometric mean	Benzene-soluble particulates: Maximum
Open-air paving:						
Exxon [1997]	80	4	0.33	1.7	0.09	0.65
Norseth et al. [1991]	51	10	NC		0.28*	0.88
Hicks [1995]	37	6	0.37	0.85	0.24	4.4
NIOSH/FHWA - MI	22	1	0.16	1.2	NC	
NIOSH/FHWA - CA1	15	2	0.3	1.3	NC	
NIOSH/FHWA - FL	18	1	0.075	0.17	NC	
NIOSH/FHWA - IN	14	1	0.041	0.1	NC	
NIOSH/FHWA - AZ	14	1	0.2	1.0	0.11	0.82
NIOSH/FHWA - CA2	16	2	0.48	1.0	0.12	0.46
NIOSH/FHWA - MA	14	2	0.15	0.52	0.073	0.4
Underground paving:						
Norseth et al. [1991]	20	4	NC		0.56*	1.3
Sylvain and Miller [1996]	9	1	1.6	2.2	0.76	1.2
Roofing application:						
Hicks [1995]	38	6	1.4	2.5	0.25	2.4
Exxon [1997]	60	4	0.34	2.7	0.12	1.2
Roofing manufacturing:						
Hicks [1995]	34	6	1.4	13	0.27	3.7
Exxon [1997]	77	3	0.6	6.2	0.08	1.3
Refineries/asphalt distribution terminals:						
Hicks [1995]	44	7	0.18	14	0.16	13
Exxon [1997]	47	3	0.19	2.5	0.05	1.3
Hot-mix asphalt plants:						
Hicks [1995]	33	6	0.78	15	0.15	1.7
Exxon [1997]	20	2	0.45	1.3	0.06	0.14

NC=not collected.
*Sample results were reported as "asphalt fume" and were the carbon disulfide extractable fraction of total particulates.
NOTE: Maximum=maximum concentrations for sample set.

Pasquini et al. [1989] and Hatjian et al. [1995a, 1997] measured D-glucaric acid in workers exposed to asphalt fumes. They reported no differences in concentrations of urinary D-glucaric acid in exposed workers compared to unexposed workers.

4.3.3 Mutagenic Activity in Urine

Mutagens excreted in the urine are thought to be indicative of exposure to mutagenic agents. The presence of mutagens excreted in urine of asphalt-exposed workers involved in road

paving was examined by Pasquini et al. [1989]. Results were based on the Ames *Salmonella* mutagenicity assay with TA98 strain and metabolic activation by rat S9. In nonsmoking individuals, asphalt-exposed workers had a significant increase in mutagenic activity in urine when compared to unexposed workers. However, among smokers, there was no significant difference in mutagenic activity in urine between exposed and unexposed workers, hence Pasquini et al. could not attribute this activity to asphalt exposure with confidence.

4.3.4 Sister Chromatid Exchange

Sister chromatid exchange (SCE) is a sensitive, indirect measure of genetic damage. However, SCE provides no information as to the identity of the genotoxic agent. SCE in white blood cells has been used as a biomarker to estimate genotoxicity of asphalt exposure. Hatjian et al. [1995b] reported SCE frequencies for a combined group of road pavers and roofers and concluded that the mean SCE frequency was increased ($P<0.05$) in the paver-roofer group compared to unexposed office workers. However, these mean SCE frequency levels did not differ from those of a group of manual workers with no asphalt exposure. The office workers were all nonsmokers, while other groups included 20% or fewer smokers.

In a second report, Hatjian et al. [1995a, 1997] combined individuals from the first study [Hatjian et al. 1995b] with new groups of workers. They were divided into four occupational groups: office workers, manual laborers, two groups of road pavers, and two groups of roofers. One group of pavers and both groups of roofers had significantly higher SCE frequencies than either manual laborers or office workers.

4.3.5 Urinary 1-Hydroxypyrene

Urinary 1-hydroxypyrene is often used as a biomarker of exposure to pyrene and, by extrapolation, to PAHs from any source [Lauwerys and Hoet 1993]. Table 4–13 summarizes the reported use of urinary 1-hydroxypyrene as a biomarker of exposure to asphalt and asphalt fumes. The logic behind this use is that asphalt and asphalt fumes are complex mixtures containing PAHs, including pyrene. Pyrene in exposed humans is metabolized to 1-hydroxypyrene and excreted in urine, mainly as glucuronide. For biological monitoring, postshift urine specimens are collected and analyzed; the analytical methods call for hydrolysis of glucuronide and other conjugates of 1-hydroxypyrene before the liberated 1-hydroxypyrene is quantified. The concentration of 1-hydroxypyrene frequently is normalized to the concentration of creatinine to correct for urine dilution.

The data in Table 4–13 are grouped by occupation and, within the road paver group, by decreasing mean level of urinary 1-hydroxypyrene. The ranges of concentrations found for the occupationally exposed and reference populations overlapped, and in many cases, the differences between mean concentrations for the two populations were not statistically significant. This result most likely reflects the contributions by nonoccupational exposures to PAH, such as ambient air pollution, tobacco smoke, and fried, roasted, and charbroiled food. The influence of nonoccupational exposures is demonstrated by mean urinary 1-hydroxypyrene concentrations in the populations of road pavers, which varied over 60-fold and overlapped the 50-fold range of mean concentrations of the reference populations. The data in Table 4–13 are more easily compared after nonoccupational exposures are adjusted by dividing the means of the exposed populations by the means of the reference populations. For road

pavers, these ratios were 2.2 to 3.7; for roofers, 1.3 and 2.7; and for workers with asphalt, 1.4 and 1.8. In comparison, road pavers working with surfacing material containing a mixture of asphalt and coal tar had mean concentrations 3.3 to 12 times the mean for the reference population [Jongeneelen et al. 1988].

Table 4–13. Urinary 1-hydroxypyrene in workers exposed to asphalt or asphalt fumes and in reference populations

Occupationally exposed population	Urinary 1-hydroxypyrene* (μmol/mol creatinine)		Reference nonoccupationally exposed population	Ratio of exposed to nonexposed population†	References
	Occupationally exposed	Nonoccupationally exposed			
Road pavers:					
P_1, 0.28 μg/m^3 PAH‡	4.2 (?? - 14)§	0.9 (?? - 3.2)§	Office workers, < 0.07 μg/m^3 PAH‡	3.7	Hatjian et al.1995a,b, 1997
P_2, 0.22 μg/m^3 PAH	2.6 (?? - 6.9)	1.4 (?? - 4.9)	Manual workers, 0.07 μg/m^3 PAH	2.7	
Road pavers	0.61 (0.16 - 1.8)**	0.28 (?? - 1.4)§	University staff and students	2.2	Burgaz et al.1992
Road pavers (asphalt-only group)	0.6	0.26 (0.02 - 0.66)††	Nonsmokers	2.2	Jongeneelen et al. 1988
		0.28 (0.09 - 1.31)	Smokers		
Road pavers	0.19‡‡ (<0.12 - 1.4)	<0.08‡‡ (<0.08 - 1.4)	Paving site preparers	3.3	Zhou 1997
Road pavers, all non-smokers, < 1 μg/m^3 PAH§§	0.07***	0.04***	Construction workers, nonsmokers	2.3	Levin et al. 1995
		0.02	Office workers, non-smokers		
Roofers:					
R_2, 0.76 μg/m^3 PAH‡	2.4 (?? - 6.9)§	0.9 (?? - 3.2)§	Office workers, < 0.07 μg/m^3 PAH‡	2.7	Hatjian et al. 1995a,b, 1997
R_1, 0.11 μg/m^3 PAH	1.5 (?? - 4.4)	1.4 (?? - 4.9)	Manual workers, 0.07 μg/m^3 PAH	1.3	
Asphalt road-tanker loaders	0.22 (0.05 - 0.41)	0.12 (0.10 - 0.21)†††	Loaders not exposed for 2-3 days	1.8	Boogaard and van Sittert 1994, 1995‡‡‡
Asphalt production	0.17 (< 0.05 - 0.72)	0.12 (<0.05 - 0.67)†††	Workers not exposed for at least 1 week	1.4	Boogaard and van Sittert 1995‡‡‡

*Data are reported as mean (range) unless otherwise indicated and are for conjugated plus free 1-hydroxypyrene in postshift urine specimens.
†When there are two reference populations, average of the two was divided into the exposed population.
‡Average total concentration of eight 3-5-ring PAH in personal air samples.
§Upper end of range computed as mean plus 2 times standard deviation.
**Range from Table 4 of Boogaard and van Sittert [1994].
††Median and 90% confidence interval.
‡‡Median. "<" data based on lowest reported measurement for group.
§§Average total concentration of seven 3-5-ring PAHs in personal air samples as determined by a method using HPLC fluorescence [Andersson et al. 1983].
***Median for data converted from nanograms per milliliter using conversion suggested by authors (1 μmol/mol creatinine ≈ 3 ng/mL).
†††Data converted from micrograms per gram creatinine.
‡‡‡Asphalt called bitumen in referenced report.

These observations suggest that the utility of urinary 1-hydroxypyrene as a biomarker of exposure to asphalt and asphalt fumes is limited. This biomarker may prove useful for revealing relatively high exposures to asphalt and for demonstrating reduction of exposures resulting from implementation of engineering controls and improved work practices. However, there are several weaknesses in the use of 1-hydroxypyrene as an indicator of PAH exposure. For instance, nonoccupational exposures to PAH may complicate determination of the contributions of low-level occupational exposures. Moreover, in the case of exposure to fumes, the fraction of pyrene in the fumes will vary with the concentration of pyrene in the asphalt and the temperature of the bulk asphalt, both factors that decrease how accurately urinary 1-hydroxypyrene represents overall exposure.

The proportional relationships between pyrene and other PAHs are changed also by the differences between the rates of skin absorption for pyrene and other PAHs. Such differences were demonstrated in an animal model by Van Rooij et al. [1995], who found that B(a)P and other PAHs of similar size or larger were absorbed more slowly than pyrene.

4.3.6 DNA Adducts

One of the primary hypotheses of chemical carcinogenesis is that the interaction between specific chemicals (or their metabolites) and DNA can result in damage to DNA that may lead to neoplastic cells or cancer [Randerath et al. 1983; Slaga 1984]. The chemically modified DNA is referred to as adducted DNA or more simply, DNA adducts. Reactive chemicals may also bind to cellular proteins, thus forming protein adducts. DNA and protein adducts in readily accessible tissues have been used as biomarkers of biological effects in workers exposed to asphalt.

Herbert et al. [1990] used the ^{32}P postlabeling methodology to examine DNA adducts in the white blood cells of 12 roofers with asphalt exposure and 12 unexposed individuals matched for age, sex, and smoking status. They also performed tests for PAH on personal-breathing-zone samples and skin wipes using HPLC fluorescence (NIOSH Method 5506). Eighty-three percent of the roofers, compared to 17% of the unexposed individuals, had detectable concentrations of aromatic DNA adducts. DNA-adduct concentrations were not correlated with PAH content of personal-breathing-zone samples, but were positively associated with postshift skin concentrations of PAHs. The contribution of exposure to PAH from removing an old pitch roof (type of pitch not specified) could not be separated from exposures while applying a new asphalt roof. It must also be noted that in the two roofers evaluated for adduct type, the adducts did not appear to be the major one normally associated with B(a)P exposure. These data indicate that B(a)P is not the major source of DNA-adduct formation, but that a yet-to-be-identified compound(s) contributes to DNA-adduct formation.

Lee et al. [1991] used an immunoassay to measure protein adducts in serum albumen in the same group studied by Herbert et al. [1990] (12 roofers and 12 unexposed individuals). These researchers reported significantly greater borderline numbers of adducts in exposed workers compared to unexposed individuals ($P \leq 0.10$). The antibody used in these studies reacts with adducts of B(a)P and, to different degrees, cross-reacts with several other PAHs. However, because the identity of the adducts being measured was unknown, this immunologic assay may not provide an accurate estimate of adduct concentrations.

Fuchs et al. [1996] measured primary DNA damage (strand breaks) and DNA adducts in mononuclear cells of workers exposed to asphalt.[3] These workers included roofers (n=7), pavers (n=18), and asphalt painters (n=9). The control group (n=34) consisted of students and office workers. All roofers and 10 members of the control group smoked. The roofers studied had significantly greater ($P<0.002$) numbers of DNA strand breaks, and these were found to increase during the work week. Because the type of roofing work and materials used were not defined, exposure to coal tar could not be excluded. Pavers and asphalt painters did not differ statistically from controls in the incidence of DNA strand breaks; however, the numbers of strand breaks were found to increase during the work week in the group of pavers. DNA adducts were found in 10 of 14 samples obtained from pavers and asphalt painters, and DNA adduct concentrations were positively correlated with age and years of exposure. Technical problems prohibited analysis of DNA adducts in other subjects.

Zhou [1997] measured DNA adducts in exfoliated uroepithelial cells in 12 road pavers and 13 road construction workers who had no exposure to asphalt fumes. No correlation was found between exposure to asphalt fumes and DNA adducts.

4.3.7 Conclusions

Biomarker studies conducted in workers exposed to asphalt are summarized in Table 4–14. Until a chemical component specific to asphalt fumes is identified, a biomarker specific and unique to asphalt exposure cannot be developed. Many of the studies of biomarkers conducted in workers exposed to asphalt were designed to determine if exposure to PAHs had occurred [Burgaz et al. 1992; Hatjian et al. 1995a,b, 1997; Herbert et al. 1990; Lee et al. 1991; Levin et al. 1995; Boogaard and van Sittert 1994, 1995; Zhou 1997]. Other studies utilized endpoints that were not specific to PAH exposure [Lafuente and Mallol 1987; Burgaz et al. 1988, 1992; Pasquini et al. 1989; Hatjian et al. 1995a,b, 1997].

Exposure to potentially genotoxic compounds may occur during work with asphalt. Evidence of such exposures is indicated by—

- The observed concentrations of mutagens in the urine of asphalt workers who were nonsmokers [Pasquini et al. 1989],
- Observed concentrations of SCE [Hatjian et al. 1995a,b, 1997] and DNA adducts [Herbert et al. 1990] in white blood cells, and
- Exposure to PAHs as detected by the presence of the sentinel urinary metabolite, 1-hydroxypyrene [Burgaz et al. 1992; Levin et al. 1995; Zhou 1997].

However, in the studies reported, smoking, environmental factors, and diet frequently confounded study interpretations. It is difficult to categorize exposure based solely on *occupational* classification, because exposures may have been misclassified. In every case, the significance of the relatively small differences in biomarkers observed in exposed workers compared to controls was not clear.

[3]Called bitumen by these authors.

Table 4–14. Summary of human biomarker studies

References	Worker population	Country	Biomarker	Measurements of exposure	Author's conclusions	Comments
Lafuente and Mallol 1987	4 asphalt road pavers, 2 asphalt production workers	Spain	Thioether excretion	Occupational classification	Thioether excretion is biphasic, increasing in first days of work period and decreasing in last days of work period.	Lack of exposure data, limited number of subjects, and inadequate study design prohibit critical analysis of the significance of these results.
Burgaz et al. 1988	12 men in asphalt-mixing plant, 32 male road pavers, 37 office clerks	Turkey	Thioether excretion	Occupational classification	No significant effect on urinary thioether excretion attributable to bitumen exposure. Smoking responsible for majority of thioether excretion. No significant differences observed in thioether excretion following bitumen exposure in nonsmokers.	Asphalt exposure caused no significant differences in thioether excretion.
Pasquini et al. 1989	16 male road pavers, 27 male office clerks	Italy	Thioether and D-glucuronic acid excretion	Environmental monitoring	No significant differences in thioether and D-glucuronic acid excretion noted between exposed and unexposed workers, or between smokers and nonsmokers.	Asphalt exposure caused no significant differences in thioether excretion.
			Urinary mutagenicity		Among nonsmokers, exposed workers had significantly higher urinary mutagenicity than unexposed workers. Smoking is a major confounding factor in urinary mutagenicity determinations.	Asphalt exposure increases urinary mutagenicity in nonsmokers.
Burgaz et al. 1992	39 male road pavers, 29 university staff and students	Turkey	Thioether excretion	Occupational exposure	In nonsmokers, thioether excretion significantly higher in exposed workers. Effects of smoking stronger than effects of occupational exposure.	The range of values in workers unexposed and exposed to asphalt overlap and fall within the range of normal human values [Van Doorn et al. 1981].
			Urinary 1-hydroxypyrene excretion		Concentrations of 1-hydroxypyrene were significantly higher in pavers than in controls (P=0.004). The effect of occupational exposure on 1-hydroxypyrene levels was stronger than that of smoking (P=0.057).	

(Continued)

Table 4–14 (Continued). Summary of human biomarker studies

References	Worker population	Country	Biomarker	Measurements of exposure	Author's conclusions	Comments
Levin et al. 1995	57 road pavers and 34 construction workers (all non-smokers)	Sweden	Urinary 1-hydroxypyrene excretion	Breathing zone air sample measurements (phenanthrene, anthracene, fluoranthene, pyrene, benzo-(a)anthracene, chrysene, and B(a)P	Pavers had higher median urinary postshift concentrations of 0.21 ng/mL compared with 0.11 ng/mL for controls (P<0.05). Pavers' Monday morning urine had significantly lower 1-hydroxypyrene (0.15 ng/mL) than on other weekday mornings (0.3 ng/mL).	Since coal tar is known not to be used in road paving in Sweden, the low concentrations of 1-hydroxypyrene in road pavers compared to controls may be a reflection of PAH exposure in road paving.
Zhou 1997	17 road pavers, 16 road construction workers	U.S.	Urinary 1-hydroxypyrene uroepithelial cell DNA adducts	Breathing zone air samples and dermal exposure measurements	Increase in urinary 1-hydroxypyrene in asphalt paving workers is related to PAH exposure during road paving. Asphalt exposure may have no or only marginal effect on formation of DNA adducts in exfoliated uroepithelial cells under current U.S. asphalt formulations.	Author did not control for dietary or other recent exposure to PAHs.
Lee et al. 1991	12 roofers, 12 without occupational exposure to PACs	U.S.	PAH-albumin adducts	Breathing zone air sample and skin wipe measurements	Roofers showed elevated adduct levels compared with the workers with no occupational exposure to PAH, but the difference was of borderline significance (P<0.1).	The antibody used in this work recognizes protein adducts of B(a)PDE-1 and a number of B(a)P metabolites, and cross-reacts with several other PAHs. Since adducts being measured are unknown, an immunologic assay such as this may over- or under-estimate actual adducts.
Boogaard and van Sittert 1994	4 workers involved in loading bitumen road tankers	Netherlands	Urinary 1-hydroxypyrene excretion	Static and personal air monitoring for 15 PAHs	Workers loading bitumen road tankers were found to excrete 0.43 µg 1-hydroxypyrene/g creatinine compared to 121 workers with pre-exposure values of 0.32 µg/g creatinine (means).	Few details, including results of air monitoring, provided on this limited number of workers analyzed in unreferenced 1989 study.

(Continued)

Table 4–14 (Continued). Summary of human biomarker studies

References	Worker population	Country	Biomarker	Measurements of exposure	Author's conclusions	Comments
Herbert et al. 1990	12 roofers, 12 without occupational exposure to PAC	U.S.	DNA adducts in white blood cells	Breathing zone air sample and skin wipe measurements	83% of 12 roofers and 17% of 12 nonroofers had detectable concentrations of aromatic DNA adducts. Adduct concentrations in the roofers were not correlated with total PAH or benzo(a)pyrene concentrations in personal air samples, but were correlated with postshift skin concentrations. Smoking and dietary PAH consumption were not associated with elevated adduct concentrations in roofers. In two samples evaluated, adducts did not appear to be major B(a)PDE-1-guanine N2 adduct.	The ^{32}P postlabeling assay may be useful for monitoring internal exposures to complex mixtures of aromatic hydrocarbons. The contribution of PAH exposure from removal of old pitch roof and that from application of new asphalt roof is not known.
Fuchs et al. 1996	7 roofers, 18 pavers, 9 bitumen painters, 34 office employees and students	W. Germany	Primary DNA damage (DNA strand breaks and cross-links) DNA adducts in peripheral mononuclear white blood cells	Occupational classification	Roofers showed significant increase in DNA strand breaks and significantly elevated levels of DNA strand breaks at end of work week compared to controls. For road pavers and bitumen painters, there were no significantly altered levels of DNA strand breaks compared to controls. DNA adducts were analyzed in 12 pavers and 2 bitumen painters, and found to be present in 10 of these workers. Adduct concentrations were correlated with number of years of employment.	Type of roofing work was not described. Thus, it is impossible to evaluate if there might be a potential for exposure to coal-tar products during roofing repair.
Boogaard and van Sittert 1995	4 workers involved in loading bitumen tankers, 59 workers involved in bitumen manufacture, 121 people with no known PAC exposure from 6 studies	Netherlands	Urinary 1-hydroxypyrene excretion	Occupational classification	Manufacture (mean 0.22 µmol/mol creatinine) and handling (0.17 µmol/mol) of bitumen did not cause a significant change in urinary excretion of 1-hydroxypyrene, compared to controls (0.16 µmol/mol creatinine).	Details are not provided on time (pre- or postshift or day of working week) for workers handling bitumen.

(Continued)

Table 4–14 (Continued). Summary of human biomarker studies

References	Worker population	Country	Biomarker	Measurements of exposure	Author's conclusions	Comments
Hatjian et al. 1995a,b, 1997	6 pavers, 10 roofers, 15 manual workers with no known occupational exposure to PACs 8 university staff (sister chromatid exchange analyses only)	England	Thioether and D-glucaric acid excretion Sister chromatid exchange Urinary 1-hydroxypyrene excretion	Breathing-zone air sample measurements	No significant differences found in thioether and D-glucuronic acid excretion between exposed and unexposed workers. No statistically significant difference found between roofers/pavers and manual workers with no known occupational exposure to PACs. SCE levels in both groups were significantly greater than levels in office workers. Rate of increase in urinary 1-hydroxypyrene concentrations over the 3-day work period greatest for pavers, less for manual workers, and minimal for roofers. Authors assumed this finding related to degree of health risk suggested by the biomarker and compared this observation to different order of risk suggested by exposures to 8 PAHs in workplace air. They proposed that the difference in risk ranking could be due to the different routes of exposure represented, differences in the PAH composition of the fume, and interindividual differences in absorption and metabolism. Using multiple linear regression analysis of day 3 postshift urinary 1-hydroxypyrene against day 1 preshift urinary 1-hydroxypyrene and the 3-day average personal air concentration of 8 PAHs, authors found significant modest correlation for the group "pavers plus manual workers" (r^2=0.43, *P*=0.001), but not for group "roofers plus manual workers" (r^2=0.23, *P*=0.067). Authors concluded that route of exposure for roofers may have been mainly pulmonary.	The range of values in workers unexposed and exposed to asphalt overlap and fall within the range of normal human values [Van Doorn et al. 1981].

5 Human Health Effects

5.1 Acute Health Effects

In 1977, based on a review of the available scientific literature, NIOSH reported that the acute toxic effects of exposure to asphalt fumes were irritation of the serous membranes of the conjunctivae and the mucous membranes of the respiratory tract [NIOSH 1977a]. Subsequently, a number of noncarcinogenic health effects continued to be reported among workers exposed to asphalt fumes. These effects include eye, nose, throat, skin, and respiratory tissue irritation; fatigue; headaches; dizziness; nausea; stomach discomfort; and insomnia. Hansen [1991] and Maizlish et al. [1988] indicated that nonmalignant lung diseases, such as bronchitis, emphysema, and asthma, were also among the toxic effects of exposure to asphalt fumes. Following is a review of the more pertinent studies concerning the noncarcinogenic health effects (excluding thermal burns) associated with exposure, including studies reported prior to the 1977 NIOSH criteria document. The review is divided into three sections—asphalt paving exposures, roofing industry exposures, and other asphalt exposures—because these categories involve differences in both worker exposures and potential health effects.

5.1.1 Health Effects Associated with Asphalt Exposures during Paving

! Norseth et al. [1991]

In a cross-sectional study performed in Norway, Norseth et al. [1991] evaluated the incidence of self-reported symptoms among 333 workers exposed to asphalt and 247 controls. Workers were divided into three groups. Group I consisted of 79 asphalt pavers who underwent personal exposure monitoring during 5 days of paving, group II consisted of 254 asphalt pavers who did not undergo personal exposure monitoring, and group III consisted of 247 maintenance workers with no reported exposure to asphalt. Subjective symptoms for a 1-week period were determined by standardized questionnaires administered to all workers at the end of the week. Asphalt exposure data, weather conditions, and traffic density were monitored for employees in the three groups. Results were calculated separately for (1) smokers and nonsmokers and (2) other background variables, such as age, number of hours worked the previous week, and work experience. Analysis of reported symptoms was based on a symptom sum score that showed significantly increased frequency in the asphalt groups. Calculation of the symptom sum score accounted for the frequency and number of days a symptom was reported.

The response rates for groups I, II, and III were 100%, 57%, and 70%, respectively. Symptoms of fatigue, reduced appetite, eye irritation, and laryngeal-pharyngeal irritation were reported more frequently among workers exposed to asphalt fumes than among unexposed workers. No differences were found for symptoms of headache, dizziness, nausea, abdominal pain, disturbed sleep, skin reactions, or a “smell of sweetness.”

Asphalt-exposed workers were found to have a significantly higher symptom sum score than unexposed maintenance workers ($P<0.001$). These differences could not be explained by smoking, hours worked during the previous week, work experience, traffic density, or

weather conditions. In addition, asphalt workers in underground facilities (garages or tunnels) had significantly higher symptom sum scores than other asphalt workers ($P<0.05$). Even with underground workers excluded, the difference between asphalt workers and unexposed road maintenance workers was still statistically significant ($P<0.001$).

Symptom sum scores correlated significantly with asphalt temperatures ($P<0.01$). The most marked increase in scores was recorded when asphalt temperatures reached 146 °C (295 °F) and continued to increase to 175 °C (347 °F). Symptom sum scores also significantly correlated with increasing asphalt fume concentration, which were apparently measured as total organic compounds, but the analytical methods were not clearly defined. The average symptom sum score was 1.3 for employees exposed to asphalt fume concentrations <0.40 mg/m^3 and 3.0 ($P<0.05$) for employees exposed to concentrations >0.40 mg/m^3.

Limitations and potential biases of this study include (1) the use of self-administered questionnaires, which may be inaccurate because of recall bias, (2) variations in the response rate among the different groups participating in the study (there may have been a response bias), and (3) lack of control for smoking in all the analyses, although evidence is presented that the unexposed group may have smoked more than the exposed group.

! NIOSH-Federal Highway Administration Interagency Agreement

Seven health hazard evaluations (HHEs) were completed as part of an interagency agreement between NIOSH and the Federal Highway Administration (FHWA) of the U.S. Department of Transportation. The evaluations were conducted during open-air highway paving operations in Michigan [Hanley and Miller 1996a], Florida [Almaguer et al. 1996], Indiana [Miller and Burr 1996a], Arizona [Miller and Burr 1996b], Massachusetts [Miller and Burr 1998], and two in California—in Sacramento [Hanley and Miller 1996b] and San Diego [Kinnes et al. 1996]. The purpose of the agreement was to evaluate occupational exposures and health effects among workers paving with crumb-rubber-modified (CRM) and conventional (noncrumb-rubber-containing) asphalt. Subjects for each evaluation were the 6 to 10 workers ("pavers") whose various tasks involved direct exposure to asphalt during paving operations. Job titles included the paver, screed, and roller operators; rakers; laborers; and dumpmen. A control group of nonpavers consisted of road workers (i.e., foreman, heavy equipment operators, traffic controllers, road surveyors) employed in the same area who were not exposed to paving operations. Pavers were evaluated during 2 days of paving with asphalt containing CRM and 2 days of paving with conventional asphalt, and nonpavers were evaluated during the corresponding 4-day period. Because of the confounding of health effects associated with CRM asphalt, the only findings presented here involve pavers using conventional asphalt and the nonpavers over the same 2 days.

Asphalt laydown temperatures varied somewhat from day to day and site to site. At five sites, temperatures ranged from 138 to 147 °C (280 to 296 °F). In Indiana, temperatures ranged from 121 to 132 °C (250 to 270 °F), and in Florida, temperatures ranged from 99 to 104 °C (211 to 219 °F). Worker exposures were evaluated during the workshift at each study site and included personal-breathing-zone and area measurements (Table 4–2). Each study participant received a one-time general health questionnaire, serial symptom surveys (administered up to five times per day), and serial peak expiratory flow rate (PEFR) tests at the same time as the short symptom surveys. PEFR testing was conducted to evaluate acute changes in lung function.

Forty-four pavers and forty-five nonpavers completed 376 and 389 symptom questionnaires, respectively, over the course of the seven surveys (Table 5–1). While the number of current smokers varied between pavers and nonpavers at individual sites, there was little difference in the overall percentage of current smokers among the groups (i.e., pavers 41%, nonpavers 40%). The number of symptoms reported per completed questionnaire for pavers, as compared to nonpavers, was higher in six of the seven surveys. In Florida, this finding was reversed, and the number of symptoms per completed questionnaire was higher among nonpavers. The number of symptoms for all causes at all seven sites combined was 0.47 among pavers and 0.21 among controls. Pavers reported a higher number of symptoms per completed questionnaire for eye, nose, throat, and skin irritation; shortness of breath; and wheezing as compared to the nonpavers (Table 5–2). The most frequently reported symptoms among pavers, in descending order, were throat irritation, nasal irritation, eye irritation, and coughing. Given the choices of mild, moderate, and severe, over 90% of the symptoms reported by pavers were classified as mild.

One of the 44 pavers (2%) reported symptoms accompanied by increased bronchial lability (i.e., the difference between the minimum and maximum PEFR on at least one survey day exceeded 20% of that day's maximum PEFR [Scanlon and Hankinson 1996]). This worker was a former smoker with a history of physician-confirmed asthma that had developed after starting work on a road crew. None

Table 5–1. Number of symptoms per completed questionnaire among pavers and nonpavers participating in seven NIOSH paving surveys

	Arizona	California 1	California 2	Florida	Indiana	Massachusetts	Michigan
Pavers (n=44):							
Total number of workers	6	7	7	4	7	6	7
No. of smokers	2	0	1	3	3	4	5
No. of symptoms reported	37	24	70	17	6	16	7
No. of completed questionnaires	60	58	63	35	56	55	49
Rate[*,†]	0.62	0.41	1.1	0.49	0.11	0.29	0.14
Nonpavers (n=45):							
Total number of workers	6	7	8	4	7	8	5
No. of smokers	2	2	3	3	2	5	1
No. of symptoms reported	19	13	21	24	2	3	0
No. of completed questionnaires	55	63	78	33	68	60	32
Rate[†]	0.35	0.21	0.27	0.73	0.03	0.05	0

[*]Rates shown are only for periods when pavers were performing conventional asphalt paving and reflect all symptom causes.
[†]Rate=number of symptoms reported divided by the number of completed questionnaires.

Table 5–2. Number of symptoms per completed questionnaire among pavers and nonpavers participating in seven NIOSH paving surveys

	Eye irritation	Nasal irritation	Throat irritation	Skin irritation	Cough	Shortness of breath	Wheezing
Pavers (n=44):							
No. of symptoms reported	32	54	45	6	20	13	7
No. of completed questionnaires	376	376	376	376	376	376	376
Rate*,†	0.09	0.14	0.12	0.02	0.05	0.04	0.02
Nonpavers (n=45):							
No. of symptoms reported	10	31	15	1	24	1	0
No. of completed questionnaires	389	389	389	389	389	389	389
Rate†	0.03	0.08	0.04	0.003	0.06	0.003	0

*Rates shown are only for periods when pavers were performing conventional asphalt paving and reflect all symptom causes.
†Rate=number of symptoms reported divided by the number of completed questionnaires.

of the 45 nonpavers experienced increased bronchial lability on any of the survey days.

While mean personal exposures to asphalt paving fumes were generally below 1 mg/m^3 total particulates calculated as a full-shift TWA (Table 4–2), pavers experienced increased symptom rates for irritation of the eyes, nose, throat, and skin; shortness of breath; and wheezing compared to unexposed road maintenance workers. Pavers did not appear to be at an increased risk for bronchial lability compared to nonpavers under the conventional paving conditions evaluated during these seven surveys (i.e., outdoor paving with highway class pavers).

However, these findings are inconclusive given the small, and possibly unrepresentative, sample groups and the lack of pre- and postshift spirometry with which to evaluate the effects of asphalt exposures on lung function more fully. Also, a possible response bias stemming from differences in worker concerns about the safety of CRM versus conventional asphalt might have influenced symptom reporting. Available sampling technology did not permit continuous short-term, task-based sampling to determine if workers were experiencing peak exposures in association with symptoms or in excess of the NIOSH REL during work. Various exposure-response relationships continue to be analyzed.

! Sylvain and Miller [1996]

During a NIOSH HHE, industrial hygiene and medical assessments were performed during a single overnight workshift on two separate paving crews (crew 1 and crew 2) working within the Third Harbor Tunnel (Ted Williams Tunnel) in Boston, MA [Sylvain and Miller 1996]. Conventional asphalt was applied at 154 °C (310 °F). Worker exposures were evaluated during the workshift and included personal breathing zone and area measurements (Table 4–4). Nine workers participated in the health assessment, which included a short general health and occupational history questionnaire, serial symptom surveys, and serial peak PEFR testing to evaluate acute changes in lung function. Participants were considered to have increased bronchial lability if the difference between minimum and maximum PEFR on at least one survey day exceeded 20% of that day's maximum PEFR [Scanlon and Hankinson 1996].

The five workers on crew 1 reported a number of acute health symptoms in association with their work exposures during the survey. However, no acute health symptoms were reported by the four workers on crew 2. The most frequently reported symptoms were eye irritation, coughing, nasal irritation, and shortness of breath. Eighty-four percent of the reported symptoms were rated as mild in severity given choices of mild, moderate, or severe. PEFR measurements indicated three workers (one from crew 1 and two from crew 2) experienced increased bronchial lability during the survey. Only one of the three workers with bronchial lability had a history of smoking.

NIOSH investigators concluded that (1) underground personal exposures (total particulates or benzene solubles) were up to 10 times higher than those found during recent open-air asphalt paving evaluations, but were still below 2.2 mg/m^3 calculated as a full-shift TWA (Table 4–5); (2) some workers experienced eye and nasal irritation, coughing, and shortness of breath in association with asphalt paving; and (3) under certain conditions, such as during indoor paving, workers with exposure to asphalt may be at increased risk for bronchial reactivity.

Limitations and potential biases associated with this study are that (1) results are based on a very small, and possibly unrepresentative, sample of pavers, (2) results reflect production and environmental conditions specific to underground paving at this site, and (3) no control group was included.

5.1.2 Health Effects Associated with Asphalt Exposures in the Roofing Industry

Only a few studies are available concerning acute health effects among workers exposed to asphalt fumes within the roofing industry. Further complicating this review is the fact that these studies are limited by their small sample sizes, lack of control groups, and the presence of possible confounding factors, such as coal tar or fiberglass.

! Hervin and Emmett [1976]

NIOSH researchers evaluated the health of 34 roofers exposed to asphalt, coal-tar pitch, and fiberglass insulation during roofing operations [Hervin and Emmett 1976]. Work involved laying down layers of asphalt applied at approximately 249 °C (480 °F) and fiberglass insulation, and then layers of coal-tar pitch applied at approximately 191 to 204 °C (376 to 399 °F), and felt. During this HHE, workers underwent medical interviews and limited physical exams that focused primarily on the skin and eyes.

Twenty-three (68%) workers complained of skin problems (burning, irritation, blistering), primarily on the face and neck, that were exacerbated by sun exposure. Nineteen roofers (56%) complained of eye irritation, and six (18%) had evidence of conjunctivitis during the survey. Conjunctivitis was significantly correlated with coal-tar-pitch exposures measured as the cyclohexane-soluble portion of total particulate concentrations above 0.2 mg/m^3. All air sampling results were less than 40% of the recommended ACGIH TLV® for asphalt fumes (5 mg/m^3) and fiberglass (10 mg/m^3). For the most part, workers reported that their eye and skin problems were caused by exposures to coal-tar pitch. Aside from thermal burns, none of the workers described eye or skin problems in association with exposure to asphalt fumes. No information was provided to allow researchers to determine if problems other than those related to eyes and skin were occurring in

association with asphalt or other exposures. The authors concluded that there was an increase in acute eye and skin disorders that appeared to be related to coal-tar-pitch exposures.

While the findings suggest that exposure to asphalt fumes during roofing does not cause appreciable skin and eye problems, the asphalt fume exposures were quite low and were not evaluated independently of other exposures, such as coal-tar pitch. In addition, these results reflect working conditions specific to this site and are based on a small, and possibly unrepresentative, sample of workers having no comparison group.

! Emmett [1986]

Emmett [1986] summarized the results from on-site surveys of roofing crews (which included the study by Hervin and Emmett [1976]) during both installation of new roof and tear-off operations. Worker exposures during the roofing operations included coal-tar pitch, asphalt, and fiberglass insulation. The surveys involved over 50 workers and included histories of medical complaints and limited physical examinations in which eyes and skin were emphasized. The largest number of skin and eye complaints were associated with coal-tar-pitch exposures; no complaints were associated with exposure to asphalt fumes. Findings from a survey of 15 roofers asked to rate the environmental causes of their eye and skin problems suggested that asphalt fume exposures were not as irritating as other types of exposures, such as sunlight, summer weather, humidity, and coal-tar pitch. While this suggests that eye and skin problems among roofers are not appreciably related to asphalt fumes, these exposures were not evaluated independently of coal-tar-pitch exposures, nor were exposure-response comparisons described. In addition, no information was given to determine if problems other than those related to eyes and skin were occurring in association with asphalt or other exposures.

! Maintz et al. [1987]

Maintz et al. [1987] evaluated six roofers who had specialized in the production of asphalt insulating roofs and asphalt insulation of wet rooms for more than 20 years. The insulation process involved laying down a cold coat (asphalt and solvent) followed by several layers of hot asphalt (180 to 200 °C [356 to 392 °F]) or tar paper. All six workers were diagnosed with chronic bronchitis, and five of the workers had a history of obstructive pulmonary function. Five of the six workers had a long-standing history (>20 years) of cigarette smoking. While interesting, this report is of limited value with respect to the pulmonary morbidity of asphalt fume exposure because of the small group size, lack of controls, and the confounding factor of smoking.

5.1.3 Health Effects Associated with Asphalt Exposure among Roofers and Pavers

! Nyqvist [1978]

Nyqvist [1978] performed a cross-sectional study of 231 asphalt workers (194 road pavers and 37 roofers) and a control group matched by age and smoking habits of workers employed in the building trades who were not normally exposed to smoke, dust, or gas. Participants filled out simple questionnaires concerning symptoms of bronchitis and underwent a one-time spirometry evaluation. The frequency of subjective symptoms of bronchitis increased with increasing time of asphalt exposure, suggesting a dose-related pattern. Compared with controls, asphalt-exposed workers reporting *slight symptoms* of bronchitis had increased relative risks of 0.67 at <3 years exposure, 1.5 at 3 to 8 years, and 5 at >8 years.

Workers reporting *severe symptoms* of bronchitis had increased relative risks of 0.33 at <3 years exposure, 1.5 at 3 to 8 years, and 5 at >8 years. Thus relative risks were significant only for symptomatic workers with exposures >8 years as compared to controls. No significant differences in spirometric values were found between exposed workers and controls. While smoking was controlled for between groups, there appeared to be some correlation among bronchitis, smoking, and long-term asphalt exposures.

The author did not provide information concerning group demographics, response rates, and risks by occupation (i.e., pavers versus roofers). The use of self-administered questionnaires for identifying symptoms could result in inaccurate results or recall bias, and the reported years of asphalt exposure might not have accurately reflected actual exposures.

5.1.4 Health Effects Associated with Asphalt Exposures in Other Occupations

! Zeglio [1950]

Zeglio [1950] published observations on 22 workers who insulated electrical cables and telegraph and telephone lines for a large Italian company. Although only asphalt was reportedly used in the process, the possibility of adulteration of the asphalt with residual coal-tar pitch was raised by the author. Workers exposed to fumes from tanks heated to 120 °C (248 °F) complained of coughing and burning in their throats and chests and frequent hoarseness. Headaches and nasal discharge were also reportedly associated with exposure. Typically, effects were reported to diminish rapidly after workers left work. However, workers with longer lengths of employment tended to experience more instances of chronic nasal, pharyngeal, and pulmonary symptoms. Among the 22 workers evaluated, physical examinations revealed 10 cases of rhinitis, 13 cases of oropharyngitis, 4 cases of laryngitis, and 19 cases of bronchitis.

Limitations and potential biases of the Zeglio study include (1) small and possibly unrepresentative sample group, (2) lack of a comparison group, (3) source and composition of the bitumens not elucidated and the potential for confounding exposure to coal tar, and (4) no measurements of worker exposures.

! Baylor and Weaver [1968]

Baylor and Weaver [1968] reviewed 841 questionnaires from 462 asphalt workers and 379 controls. The questionnaires were obtained from medical personnel employed by seven petroleum companies that produced asphalt and included information on each worker's medical history (including a brief physical examination), occupational history, and smoking history. No workers with less than 5 years of work with asphalt were included in the survey; the average duration of employment for both workers and controls was 15.1 years.

Results of the survey indicated no significant differences in cancer, lung disease, and skin disease between asphalt workers and controls. The number of cases of miscellaneous lung disease (such as bronchitis and asthma) were more frequent among asphalt workers (8.6%) compared to controls (4.3%), although excess cigarette use (20 cigarettes a day for >20 years) was similar between asphalt workers (26%) and controls (24%). The vast majority of the cases of miscellaneous lung disease were for chronic bronchitis, while a few cases of asthma and emphysema were noted.

Based on undefined information provided to them by representatives from 31 paving companies, 15 state highway commissions, three roofing manufacturers, and six insurance

carriers, the authors also reported that asphalt-exposed workers were not experiencing any notable adverse health effects. While this study is frequently cited in the literature, it is of limited value to the current assessment of health effects because of its lack of information regarding study methods and results (i.e., response rates, selection of participants, content of questionnaires, description of exposures, etc.).

! Apol and Okawa [1977]

A NIOSH HHE conducted in October 1976 studied 15 workers involved in the production of fibrous glass asphalt roofing shingles [Apol and Okawa 1977]. During the period of the survey, workers had intermittent and variable exposures to mineral dust (slate granules, talc, sand), felt, glue, and asphalt fumes (asphalt heated to approximately 204 °C [399 °F]), depending upon job tasks. No fiberglass was being used at the facility during the portion of the survey discussed here. Participating workers underwent medical interviews and limited physical exams. All were male and had a mean of 7 years employment at the plant.

The most frequently reported problems were nasal irritation (47%), throat irritation (47%), and eye irritation (40%). One worker, a smoker, complained of shortness of breath; otherwise, all the interviewed workers denied past or current breathing problems. Eight workers were noted to have apparent work-related eye irritation during a postshift physical examination. The authors noted that exposure measurements indicated fairly high worker exposures to asphalt fumes and dust during the workshift (one of the 19 asphalt fume samples and seven of the 35 total dust samples exceeded 5.0 mg/m^3 of total particulates).

Possible limitations of the study include (1) small and possibly unrepresentative study group, (2) lack of a control group for comparison, and (3) lack of an evaluation of the relationship between specific work exposures and reported health symptoms. No information concerning the asphalt formulation was provided to help determine if other additives or contaminants might have been present.

! Chase et al. [1994]

Chase et al. [1994] reported complaints of nausea, headache, fatigue, skin rash, and eye, nose, and throat irritation among 27 of 200 employees manufacturing ballast boxes and coils for fluorescent and high-intensity lighting. Symptoms were associated with exposure to fumes from a new asphalt formulation (heated to 270 °C [518 °F]) used to embed and insulate electronic components inside the ballast boxes.

Personal-breathing-zone samples were collected from six of the symptomatic workers and showed asphalt fume levels ranging from 0.50 to 1.30 mg/m^3 (mean of 0.83 mg/m^3). Headspace analysis of bulk asphalt samples at 180 °C (356 °F) identified volatile thermal decomposition products that included acetaldehyde, acetone, carbon monoxide, and carbonyl sulfide. At 260°C (500 °F), the headspace analysis identified an ether, 1-butanol, butyl Cellosolve®, methanol, carbon disulfide, isobutylene, and ethylene.

Medical assessments of the 27 symptomatic employees included personal interviews, questionnaires, physical examinations, spirometry, and blood tests to screen for hepatic, renal, and hematologic functions. During the initial interviews, all 27 employees reported symptoms relating to the central nervous system, ears, nose, and throat. Other symptoms reported were related to eyes (93%), gastrointestinal (89%), and respiratory (59%) systems, and skin (41%). Physical examinations revealed conjunctivitis (11%),

evidence of nose bleeds (52%), throat irritation (59%), and skin rash (15%). Medical tests showed no significant effects on liver, kidneys, or lungs (spirometry results). Hematologic tests showed increased erythrocyte sedimentation rates (48%), increased mean platelet volume values ($P = 0.013$) (41%), and decreased red blood cell numbers (41%) in comparison with the standard laboratory reference range. Follow-up medical assessments among 15 of the 27 symptomatic employees conducted after workplace modifications had been made (i.e., installation of local exhaust ventilation) showed a significant decline in workers' acute symptoms and a decrease in mean platelet volume toward normal.

While the findings described in this study are interesting, particularly the hematologic testing, the results are difficult to interpret because of (1) the small and possibly unrepresentative sample group, (2) lack of a comparison group, and (3) confounding exposures to additives likely to have been present in the asphalt formulation.

! Tavris et al. [1984]

Tavris et al. [1984] investigated an outbreak of health problems related to volatilized asphalt fumes among office workers. Interviews were conducted with 15 of the 19 workers employed in the problem office area. The most frequently reported problems included headaches, eye irritation, sore throat, nasal congestion, nausea, lightheadedness, and itchy skin.

Laboratory analyses of blood specimens (SGOT, BUN, CBCs) of nine of the 15 workers were normal except for a slight eosinophilia (4% to 5%) in five of the workers. Worker symptoms were attributed to volatilized asphalt fumes from a malfunctioning fluorescent light fixture covered with melted asphalt from an overheated light ballast. Workplace measurements for formaldehyde, carbon monoxide, and carbon dioxide were normal; however, no specific measurements for asphalt fumes were made.

Limitations of the study include a small study group and lack of a comparison group. Also, no specific measurements for asphalt fumes were made, and no product information or analysis of the asphalt was provided to help determine if other additives or contaminants might have been present.

5.1.5 Health Effects among Asphalt Workers Reported in Other Studies

A 1990 review of the scientific literature [Fries and Knudson 1990] on asphalt fumes conducted by the European Asphalt Producers Association cited three European studies that have not been independently reviewed by NIOSH researchers. Short reviews of these studies from the Association report are presented below, without comment, to provide a comprehensive presentation of available literature regarding effects on human health associated with exposure to asphalt fumes. No exposure values or dose-response information were provided in these studies.

(1) Hasle et al. [1977] evaluated 166 Danish pavers. Chronic bronchitis and difficulty in breathing were reported in 25% and 40% of the paving workers, respectively.

(2) Schaffer et al. [1985] conducted clinical, x-ray, and biochemical analyses of 50 bitumen-exposed workers and 15 controls. Among exposed workers, there were increased symptoms of bronchitis, stomach pain, and skin irritation, but no statistical evaluations or conclusions regarding health hazards were reported.

(3) Waage and Nielson [1986] reported significantly higher prevalences of smarting eyes, stomach pains, and skin irritation among Norwegian asphalt pavers. Also, an increased incidence of headaches, dizziness, sleepiness, nausea, reduced appetite, and markedly reduced lung function (PEFR values) was reported.

! Exxon [1997]

A study by Exxon Biomedical Sciences entitled *Shift Study of Pulmonary Function and Symptoms in Workers Exposed to Asphalt Fume* [Exxon 1997] was recently completed. In this study, 170 asphalt-exposed workers employed in five segments of the asphalt industry (hot-mix plants [n=11], terminals [n=24], roofing manufacturers [n=43], roofers [n=37], pavers [n=55]) were evaluated to determine whether there was an association between the incidence of symptoms and changes in pulmonary function related to workshift asphalt exposures. Researchers evaluated personal exposures over 2 days for each participant at each worksite. About 288 person-days of observation were completed in different segments of industry as follows: paving, 82 person-days (32.4%); roofing manufacturing, 77 person-days (25.3%); roofing, 62 person-days (21.8%); terminals, 47 person-days (14.1%); and hot-mix plants, 20 person-days (6.5%). Only small differences were noted in mean age, height, and weight among participants from the different industry segments. Smoking varied somewhat by industry segment, i.e., paving, 38%; roofing, 43%; roofing manufacturing, 44%; hot-mix asphalt plants, 54%; and terminals, 17%.

As part of the health assessment, each participant received a standardized respiratory health questionnaire, serial symptom surveys (administered up to five times during a workshift), and serial PEFR tests performed at the same time as the symptom surveys. Pre- and postshift pulmonary function tests were conducted to evaluate changes in lung function over the workshift and included measures of forced expiratory volume in 1 sec, forced vital capacity, PEFR, and mid-expiratory flow volume (FEF25-75).

All personal-breathing-zone sample results for total particulates and benzene-soluble particulates were measured as 8-hr TWAs (Table 4–12). Results indicated that workers exposed to asphalt were typically symptomatic less than 5% of the time and that most symptoms were reported to be mild. The most commonly reported problems were breathing difficulty, nose irritation, headache, throat irritation, and coughing. Various analyses of individual symptom responses and a developed symptom score (derived from a score for each of the 15 symptoms assessed for each subject) did not reveal any significant associations with workers' personal-breathing-zone measurements of asphalt exposure. In addition, different analyses (i.e., logistic regression, nonparametric regression, factor analysis) of pulmonary function tests regarding workers' smoking frequency, reported symptoms, developed symptom score, or measured asphalt exposures did not show any significant associations.

Limitations and potential biases associated with this study include (1) a relatively small and possibly unrepresentative sample from each industry segment, (2) narrow exposure ranges and very little data on higher concentrations, which reduces researchers' ability to detect significant exposure-response associations and levels of adverse effects (i.e., lowest observable adverse effect level [LOAEL] or no observable adverse effect level [NOAEL]), (3) possible lack of correlation between 8-hr average exposure measurements and assessed acute health effects (which were evaluated every few hours; shortterm peak exposure measurements may be

necessary to determine an exposure-response association), and (4) lack of inclusion of an unexposed comparison group (although workers at most study sites who had lesser amounts of exposure were included in the exposure-response analysis).

5.1.6 Conclusions

Studies concerning the acute toxic effects of exposure to asphalt fumes have repeatedly found symptoms of irritation of the serous membranes of the conjunctivae (eye irritation) and the mucous membranes of the upper respiratory tract (nasal and throat irritation) among workers. These health effects have been best described in asphalt road pavers [Norseth et al. 1991; Hanley and Miller 1996a,b; Almaguer et al. 1996; Miller and Burr 1996a,b, 1998; Kinnes et al. 1996; Sylvain and Miller 1996]. They typically appear to be of mild severity and transitory in nature [Hanley and Miller 1996a,b; Almaguer et al. 1996; Miller and Burr 1996a,b, 1998; Kinnes et al. 1996; Exxon 1997]. Similar symptoms have also been reported in workers exposed to asphalt fumes during the manufacture of asphalt roofing shingles [Apol and Okawa 1977] and fluorescent lights [Chase et al. 1994], cable insulating activities [Zeglio 1950], and from a malfunctioning light fixture in an office [Tavris et al. 1984]. The occurrence of mild transitory symptoms (i.e., nasal and throat irritation, headaches, and coughing) was recently reported among workers employed in five segments of the asphalt industry (hot-mix plants, terminals, roofing, roofing manufacturing, and paving), although no significant dose-response associations were found between measured exposures and symptoms [Exxon 1997]. While these acute health effects have been reported in a number of work settings, the specific association, if any, between symptoms and asphalt fume exposure has been difficult to establish because of a lack of research on this topic and various limitations of those studies that have been conducted.

In addition to mucosal irritation, skin irritation, pruritus, and occasionally rashes have been reported [Hanley and Miller 1996a,b; Almaguer et al. 1996; Miller and Burr 1996a,b; Kinnes et al. 1996; Chase et al. 1994; Tavris et al. 1984; Schaffer et al. 1985; Waage and Nielson 1986]. Given the presence of confounding co-exposures (i.e., diesel fuel, coal tar, fiberglass) and environmental conditions (wind, heat and humidity, UV radiation), the extent to which asphalt fumes may be associated with these skin problems is unclear. If asphalt-related dermal photosensitization is occurring, such as seen with coal tar, it has not been described in the literature and so needs to be further investigated, as do the other reported skin problems.

Symptoms of nausea, stomach pain, decreased appetite, headaches, and fatigue have also been reported among workers exposed to asphalt [Norseth et al 1991; Chase et al. 1994; Tavris et al. 1984; Schaffer et al. 1985; Waage and Nielson 1986; Exxon 1997], although no significant dose-response associations were found between measured exposures and symptoms [Exxon 1997]. These nonspecific types of symptoms require further investigation to help clarify and establish the nature of any causal relationships with asphalt fume exposure.

Lower respiratory tract symptoms (coughing, wheezing, shortness of breath) [Hanley and Miller 1996a,b; Almaguer et al. 1996; Miller and Burr 1996a,b; Kinnes et al. 1996; Sylvain and Miller 1996; Nyqvist 1978; Zeglio 1950] and changes in pulmonary function (e.g., bronchial lability) [Sylvain and Miller 1996; Waage and Nielson 1986] have been described among workers exposed to

asphalt fumes. Results from recent studies [Exxon 1997; Hanley and Miller 1996a,b; Almaguer et al. 1996; Miller and Burr 1996a,b; Kinnes et al. 1996] showed that some workers experienced lower respiratory tract problems or changes in pulmonary function when exposure to asphalt fumes was relatively low, such as during open-air highway paving. The NIOSH studies [Kinnes et al. 1996; Sylvain and Miller 1996] indicated significant changes in pulmonary function in one of 44 workers engaged in open-air asphalt paving and three of nine workers engaged in underground asphalt paving. The Exxon study [1997] found no significant association between pulmonary function measurements and asphalt exposures among workers employed in five segments of the asphalt industry. Some limited evidence suggests that personal health factors (i.e., pre-existing asthma) or exposures to greater amounts of asphalt fumes, such as those found during underground paving, may increase workers risk for lower respiratory tract symptoms or changes in pulmonary function [Norseth et al. 1991; Sylvain and Miller 1996]. However, the current data are insufficient to determine the relationship between asphalt fume exposures and these health effects.

While asphalt fume concentrations associated with the health effects noted above have not been well characterized, symptoms of irritation were noted during open-air paving among workers whose average personal exposures were generally below 1.0 mg/m^3 total particulates and 0.3 mg/m^3 benzene- or carbon disulfide-soluble particulates calculated as a full-shift TWA [Norseth et al. 1991; Hanley and Miller 1996a,b; Almaguer et al. 1996; Miller and Burr 1996a,b, 1998; Kinnes et al. 1996; Sylvain and Miller 1996; Exxon 1997]. Presently, none of these studies have established a clear exposure-response relationship between exposures and health effects. However, health effect findings from research on underground asphalt paving, where exposures are greater, suggest that a dose-response relationship may exist [Norseth et al. 1991; Sylvain and Miller 1996]. Improved research studies, such as those involving larger groups of participants and controls, evaluation of workers with higher levels of exposure, and enhanced measurement of exposures (i.e., real-time peak concentrations) in relation to health responses may be necessary to elucidate any exposure-response relationships, if present.

In addition, bronchitis possibly related to chronic lower respiratory tract irritation has been reported among asphalt workers in several studies [Hansen 1991; Maizlish et al. 1988; Maintz et al. 1987; Nyqvist 1978; Zeglio 1950; Baylor and Weaver 1968; Hasle et al. 1977]. Unfortunately, the limited data preclude making any determinations concerning asphalt-exposure-related chronic pulmonary morbidity. Until additional data have been gathered to clarify the health risks associated with occupational exposure to asphalt, it would be prudent to be cautious when working with these materials and to limit worker exposures to the extent feasible.

5.2 Chronic Health Effects

The 1977 NIOSH *Criteria for a Recommended Standard: Occupational Exposure to Asphalt Fumes* contains discussions of the pertinent epidemiologic data published through 1976 on workers exposed to roofing or paving asphalt fumes. These earlier studies relating asphalt fume exposure to cancer mortality were judged to be inconclusive because of methodological problems, such as incomplete exposure data, discrepancies in terminology, insufficient latency periods, and confounding variables (e.g., smoking and exposure to other potential carcinogens, such as coal-tar products) [NIOSH 1977a]. These problems made it impossible to determine the

cause of observed excesses of cancer incidence in employees exposed to asphalt fumes during roofing and paving operations.

Since the release of the NIOSH criteria document, additional epidemiologic studies have been conducted to evaluate the possible association between asphalt fume exposure and cancer risk [Hansen 1989 a,b, 1991; Engholm et al. 1991; Wilson 1984; Maizlish et al. 1988; Bender et al. 1989; Mommsen et al. 1983; Risch et al. 1988; Bonassi et al. 1989]. These studies are reviewed below under the headings of "Road Workers," "Pavers," "Roofers," and "Others" because the nature of exposures to asphalt fumes is different and because exposures to other carcinogenic hazards may confound interpretations of the data.

The results of the studies are summarized in Tables 5–3 through 5–8.

5.2.1 Pavers

5.2.1.1 Cohort Studies

! Hansen [1989a, 1991]

In a retrospective cancer incidence study by Hansen [1989a], the causes of death of 679 male Danish mastic asphalt employees were compared with causes of death in the total Danish male population over the same period. Mastic asphalt, which is a mixture of fine sand, stone powder, finely divided limestone, and 12% to 17% asphalt, is used in paving and flooring operations in Denmark. The mix is emptied into buckets at worksites, and workers apply the mastic asphalt by pouring out a given amount and leveling or smoothing it with a wooden trowel.

The same workers perform both flooring and paving activities. To determine asphalt fume concentrations, the Danish National Institute of Occupational Health collected 35 personal-breathing-zone samples during flooring operations, representing a third of the total work hours of the cohort, and two samples during paving operations, representing the remaining two-thirds of the total work hours. The 35 samples collected during flooring operations ranged from 0.5 to 260 mg/m^3 of asphalt fume condensate, with a median of 19.7 mg/m^3, and the two samples collected during paving operations were 3.5 and 4.3 mg/m^3 of asphalt fume condensate. On the basis of these results, occupational exposure to asphalt fumes was estimated to be almost the equivalent of a continuous work-time exposure at the current Danish TWA standard of 5 mg/m^3.

Hansen identified the employees through files covering the time period 1959-1980 and followed them through January 1, 1985. As of January 1, 1985, 524 employees were living, 149 were deceased, and 6 had emigrated. The standardized incidence ratio (SIR) for all malignant neoplasms was 1.95 (75 cases, with a confidence interval [CI] of 95%=1.53-2.44). As a group, mastic asphalt employees 40 years or older when diagnosed (n=547) had statistically significant increases in SIRs for cancers of the lung (SIR=3.44; 95% CI=2.27-5.01), mouth (SIR=11.11; 95% CI=1.35-40.14), esophagus (SIR=6.98; 95% CI=1.44-20.39), and rectum (SIR=3.18; 95% CI=1.28-6.56). Hansen divided the cohort into three birth-year subcohorts (1893-1919, 1920-1929, and 1930-1960). Lung cancer was elevated in all three subcohorts, but was highest among workers born in the period between 1930 and 1960 (SIR=8.57, 95% CI=1.77-25.05). These workers were believed to be the least likely to have been exposed to coal-tar pitch.

Histories for tobacco consumption were not available for the cohort. However, Hansen cited a 1976 survey of smoking habits of Danish mastic asphalt employees that found that 22% were nonsmokers and 78% were smokers. Hansen also cited another survey of the Danish male population done in 1982 that found fewer smokers among men the same

Table 5-3. Epidemiologic studies on asphalt exposure: cohort studies, pavers

Author, country, and occupation	No. of study subjects	Dates of case ascertainment	Type or site of condition	No. of deaths or cases	Risk ratio	95% CI or *P* value
Hansen 1989a, Denmark, mastic asphalt workers	679	1959-1986	All cancers	74	SIR 1.95[*]	1.53–2.44
			Lung cancer	27	SIR 3.44[†]	2.27–5.01
			Mouth	2	SIR 11.11[†]	1.35–40.14
			Esophagus	3	SIR 6.98[†]	1.44–20.39
			Rectum	7	SIR 3.18[†]	1.28–6.56
Hansen 1991, Denmark, mastic asphalt workers	679	1959-1986	All causes	148	SMR 1.57[†]	1.34–1.85
			All cancers	62	SMR 2.29[†]	1.75–2.93
			Lung cancer	25	SMR 2.90[†]	1.88–4.29
			Nonlung cancer	37	SMR 2.00[†]	1.41–2.76
			Bronchitis, emphysema, asthma	9	SMR 2.07[†]	0.95–3.93
			Liver cirrhosis	7	SMR 4.67[†]	1.88–9.62
Engholm et al. 1991, Sweden, pavers	2,572	1971-1985	All causes	96	SMR 0.69	NR
			All cancers	47	SIR 0.86	NR
			Stomach cancer	5	SMR 2.01	NR
			Stomach cancer	6	SIR 2.07	NR
			Lung cancer	7	SMR 1.10	NR
			Lung cancer	8	SIR 1.24	NR
Maizlish et al. 1988, United States, highway maintenance workers (n=307)	1,570	1970-1983	Emphysema	8	PMR 2.50	1.80–4.92
			Digestive system cancer	25	PMR 1.51	0.97–2.23
			Stomach cancer	6	PMR 2.27	0.83–4.95
			Skin cancer	2	PMR 1.22	0.12–4.93
			Prostate cancer	7	PMR 2.26	0.91–4.66
			Brain cancer	4	PMR 1.60	0.40–4.10
			Lymphopoietic cancer	8	PMR 1.15	0.50–2.26
Bender et al. 1989, United States, highway maintenance workers	4,849	1945-1984	All causes	1,530	SMR 0.9	0.86–0.96
			All cancers	274	SMR 0.83	0.73–0.94
			Lung cancer	57	SMR 0.69	0.52–0.90
			Mouth, pharyngeal cancer	2[‡]	SMR 11.10	1.30–40.10
			Gastrointestinal cancer	3[§]	SMR 5.82	1.20–17.00
			Prostate cancer	11[**]	SMR 2.98	*P*<0.01

See footnotes at end of table.

(Continued)

Table 5-3 (Continued). Epidemiologic studies on asphalt exposure: cohort studies, pavers

Author, country, and occupation	No. of study subjects	Dates of case ascertainment	Type or site of condition	No. of deaths or cases	Risk ratio	95% CI or *P* value
			Kidney, bladder, other urinary organ cancers	7[††]	SMR 2.92	1.17–6.02
			Leukemia	8[‡‡]	SMR 4.49	1.94–8.84
Partanen et al. 1997, Finland, road pavers (males only)			Lung cancer	NR	SMR 1.5	1.2–1.9
				NR	SIR 1.4[§§]	0.9–1.9
Milham 1997, United States, road graders, pavers, machine operators, excavators, operating engineers	7,266	1950-1989				
			Respiratory system	614	PMR 1.1	*P*<0.01
			Bronchus, trachea, lung cancer (ICD 162)	558	PMR 1.20	*P*<0.01
operating engineers, only			Respiratory system cancer	136	PMR 1.21	*P*<0.05
			Bronchus, trachea, lung (ICD 162)	122	PMR 1.21	*P*<0.05
			Bronchus, lung (ICD 162.1, 163)	76	PMR 1.42	*P*<0.01
			Asthma	5	PMR 1.60	NS
			Lymphatic, hematopoietic cancer	43	PMR 1.42	*P*<0.05
			Reticulosarcoma	7	PMR 1.37	NS
			Lymphosarcoma	6	PMR 1.88	NS
			Hodgkins disease	4	PMR 1.58	NS
			Other lymphomas	10	PMR 2.00	*P*<0.05
			Motor vehicle accidents	47	PMR 1.59	*P*<0.01
road graders, pavers, machine operators, and excavators.			Bronchus, lung cancer (ICD 162.1, 163)	288	PMR 1.24	*P*<0.01
			Motor vehicle accidents	249	PMR 1.39	*P*<0.01

Abbreviations: CI=confidence interval; ICD=*International Classification of Diseases*; NR=not reported; NS=not statistically significant; PMR=proportionate mortality ratio; SIR=standardized incidence ratio; SMR=standardized mortality ratio.

[*]All mastic asphalt workers (n=679).

[†]Mastic asphalt workers aged 40-89 years (n=547).

[‡]Employed ≥40 years.

[§]Urban workers with 40-49 years of latency.

[**]Started working 1955-1964.

[††]Workers with 40-49 years of latency.

[‡‡]Employed 30-39 years.

[§§]Asphalt exposure.

Table 5-4. Epidemiologic studies on asphalt exposure: cohort studies, roofers

Author, country, and occupation	No. of study subjects	Dates of case ascertainment	Type or site of condition	No. of deaths or cases	Risk ratio	95% CI or *P* value
Hammond et al. 1976, United States,						
Roofer, waterproofer	5,939	1960-1971	Lung cancer	99	SMR 1.58*	1.29–1.94
			Lung cancer	24	SMR 2.0†	1.28–4.32
			Respiratory disease‡	71	SMR 1.67	
Menck and Henderson 1976, United States,						
Roofer	2,000	1968-1970	Lung cancer	3	SMR 8.78	*P*>0.01
				2 incident cases		
Engholm et al. 1991, Sweden,						
Roofer	704	1971-1985	Lung cancer	3 deaths	SMR 2.79	
				4 cases	SIR 3.62	
				3 cases	OR 6.0§	
			Stomach cancer	5 deaths	SMR 2.01	
				1 case	SIR 1.98	
			Lymphatic, hematopoietic cancer	2 deaths	SMR 2.68	
			Leukemia	1 case	SIR 2.26	
Hrubec et al. 1992, United States,						
Roofer, slater	52	1954-1980	Lung cancer	4 deaths	RR 3.0	1.30–6.75**
Pukkala 1995, Finland,						
Asphalt roofer	47,000	1971-1985	Lung cancer	18 cases	SIR 3.25 ††	1.92–5.13
Milham 1997, United States, Washington State,						
Roofers and slaters	1,057	1950-1989	Buccal cavity, pharynx cancer	9	PMR 1.67	NS
			Respiratory cancer	105	PMR 1.53	*P*<0.01
			Larynx cancer	6	PMR 2.59	*P*<0.05
			Bronchus, trachea, lung cancer (ICD 162)	86	PMR 1.44	*P*<0.01
			Bronchus, lung cancer (ICD 162.1, 163)	53	PMR 1.60	*P*<0.01
			Asthma	7	PMR 2.86	*P*<0.01
			Diseases of circulatory system	364	PMR 0.88	*P*<0.05
			Diseases of respiratory system	76	PMR 1.20	NS
			Chronic bronchitis	4	PMR 1.99	NS
			Bronchitis with emphysema	4	PMR 2.16	NS
			Other diseases of respiratory system (ICD 510-527)	52	PMR 1.43	*P*<0.05

See footnotes at end of table.

(Continued)

Table 5-4 (Continued). Epidemiologic studies on asphalt exposure: cohort studies, roofers

Author, country, and occupation	No. of study subjects	Dates of case ascertainment	Type or site of condition	No. of deaths or cases	Risk ratio	95% CI or *P* value
			Other diseases of lung, pleural cavity	46	PMR 1.49	*P*<0.01
			Emphysema without bronchitis	28	PMR 1.63	*P*<0.01
			Cirrhosis of liver with alcoholism	17	PMR 2.84	*P*<0.01
			Cirrhosis of liver without alcoholism	23	PMR 1.49	NS
			Falls from elevation	17	PMR 4.00	*P*<0.01
			Psychosis	5	PMR 2.23	NS

Abbreviations: CI=confidence interval; ICD=*International Classification of Diseases*; NS=not statistically significant; OR=odds ratio; PMR=proportionate mortality ratio; RR=relative risk; SIR=standardized incidence ratio; SMR=standardized mortality ratio.

[*]> 20 years since joining union.

[†]>40 years since joining union.

[‡]Pneumonia, TB, influenza excluded.

[§]Adjusted for smoking, relative risk.

[**]90% confidence interval.

[††]Adjusted for age, calendar time, and soclal class.

Table 5–5. Epidemiologic studies on asphalt exposure: case-control studies of roofers

Author, country, and occupation	Dates of case ascertainment	Number of study subjects		Number of subjects with lung cancer		Odds ratio*	95% CI
		Cases	Controls	Cases	Controls		
Zahm et al. 1989, United States							
Roofer	1980-1985	4,431	11,326	6	7	2.1	0.6-8.2
Schoenberg et al. 1987, United States							
Roofer, slater	1967-1976	763	900	13	8	1.7	0.7-4.4
Morabia et al. 1992, United States							
Roofer, slater	1980-1985	1,793	3,228	7	6	2.1	0.7-6.2

Abbreviations: CI=confidence interval.
*Adjusted for smoking.

Table 5–6. Epidemiologic studies of asphalt exposure: case control studies of bladder, pelvis, and ureter cancer

Author, country, and exposure or occupations	Dates of case ascertainment	Site	Number of study subjects		Number of exposed subjects		Risk ratio	95% CI
			Cases	Controls	Cases	Controls		
Mommsen et al. 1983, Denmark								
Petroleum or asphalt	Not given	Bladder	212	259	2	3	RR 2.36	NS
Risch et al. 1988, Canada								
Asphalt or tar	1979-1982	Bladder	739	781	739	781	OR 1.44*	0.78- 2.74
							OR 3.11†	1.19- 9.68
							OR 2.02‡	1.08-4.97
Bonassi et al. 1989, United States								
Road menders	Not given	Bladder	121	342	2	6	OR 1.40	0.27-7.28
Jensen et al. 1988, Denmark								
Asphalt or tar	1979-1982	Renal pelvis, ureter	96	294	9	6	RR 5.5	1.6-19.6

Abbreviations: CI=confidence interval; NS=not statistically significant; OR=odds ratio; RR=relative risk.
*Ever exposed to "tar and asphalts" (n = 46).
†Exposed during full-time job of at least 6 months 8 to 28 years before diagnosis (n = 23).
‡Trend with duration. Odds ratio for trend at 10 years duration.

Table 5–7. Epidemiologic cohort study of asphalt exposure during manufacture of asphalt products, Denmark [Hansen 1989b]

Number of study subjects	Type of condition	No. of deaths or cases	SMR	95% CI
1,320 exposed	All cancers	29	1.59*	1.06-2.28
43,024 unexposed	Digestive cancer	6	1.57	0.58-3.43
	Respiratory cancer	11	1.52	0.76-2.71
	Bladder cancer	3	2.91	0.60-8.51
	Brain cancer	3	5.00	1.03-14.61
	Ischemic heart disease	29	1.31	0.88-1.89

Abbreviations: CI=confidence interval; SMR=standardized mortality ratio.
*Workers ≥45 years of age between 1975-1980.
NOTE: Case ascertainment was for 1970-1980.

Table 5–8. Epidemiologic studies on asphalt exposure: case control studies of respiratory cancer and other diseases

Author, country, and occupation	Dates of case ascertainment	Site	Number of study subjects		Number of exposed subjects		Odds ratio	95% CI
			Cases	Controls	Cases	Controls		
Vineis et al. 1988, United States								
Roofers, asphalt workers	1974-1981	Lung cancer	2,973	3,210	45	37	1.4	0.9-2.3
Zahm et al. 1989, United States								
Pavers, surfacers, materials-moving equipment operators.	1980-1985	Lung cancer	4,431	11,326	32	64	0.9	0.6-1.5
Roofers					6	7	2.1	0.6-8.2
Chiazze et al. 1993, United States*	Not given	Lung cancer	144	260	111	251	0.96	0.65-1.42
		Nonmalignant respiratory diseases	101	183	79	171	1.34	0.82-2.2
Austin et al. 1987, United States†	Not given	Hepatocellular carcinoma	80	146	7	5	3.2	0.9-11
Siemiatycki 1991, Canada	Not given	Colon cancer	3,730	533‡	22	‡	1.6	1.1-2.5

Abbreviations: CI=confidence interval.
*Exposed to asphalt fumes of >0.01 mg/m^3 cumulative exposure concentration.
†Exposed to asphalt.
‡Number of controls exposed not available.

ages as the cohort (39% nonsmokers and 61% smokers).

Hansen estimated that differences in smoking rates between mastic asphalt workers and the general population would increase the incidence of lung cancer in mastic asphalt workers by about 20%, which would not account for the threefold increase in cancer in the mastic asphalt cohort.

Hansen also considered the potential for confounding by urbanization. Urban areas of Denmark were reported to have higher incidences of cancer than rural areas. Hansen suggested that nearly all of the asphalt workers in her study were urban dwellers, as compared to only 40% of the referent population, and that this potential bias may have underestimated the expected cancer incidence in the study population by 35%. However, again, this would not account for the observed threefold excess of respiratory cancer among workers exposed to asphalt fumes.

In 1991, Hansen conducted a retrospective mortality study of the original cohort in whichthe study population was followed to June 10, 1986 [Hansen 1991]. As of that date, 504 employees were living, 169 were deceased, and the vital status of six workers could not be determined. The overall mortality of the cohort was significantly elevated compared to the general population (standardized mortality ratio [SMR]=1.63; 95% CI=1.41-1.90). SMRs for all cancers (SMR=2.29; 95% CI=1.75-2.93), lung cancer (SMR=2.90; 95% CI=1.88-4.29), and all nonlung cancers (SMR=2.00; 95% CI=1.41-2.76) were significantly elevated among workers aged 40 to 89. Increased mortality was also reported for nonmalignant respiratory diseases (emphysema, bronchitis, and asthma) (SMR=2.07; 95% CI=0.95-3.93) and liver cirrhosis (SMR=4.67; 95% CI=1.88-9.62). As she did in the 1989 cancer incidence study, Hansen considered the potentially confounding effects of smoking and urbanization on lung cancer. For urbanization, Hansen used an adjustment factor of 10% to increase the expected number of lung cancers, whereas in the 1989 study, she used an adjustment factor of 35%. The 10% adjustment factor appears to be based on reported differences in urban and rural lung cancer mortality rates. For smoking, Hansen used an upward adjustment factor of 18% for the expected number of lung cancers, which is similar to the 20% factor she used in her 1989 study [Hansen 1989a]. Based on these adjustment factors, Hansen estimated that among mastic asphalt employees 40 years or older, the SMR for lung cancer mortality was 2.46 (95% CI=1.59-3.63) when adjusted for smoking, 2.64 (95% CI=1.71-3.90) when adjusted for urbanization, and 2.24 (95% CI=1.45-3.30) when adjusted for both smoking and urbanization. Hansen then concluded that the increase in lung cancer mortality observed in the 1991 study could not be explained by differences in either smoking habits or degree of urbanization.

The validity of several aspects of the design and analysis of Hansen's incidence and mortality studies has been debated. Wong et al. [1992] critiqued both studies, and Hansen [1992] subsequently published a reply. The major criticisms were the lack of control for confounding by smoking and urbanization, possible confounding by coal tar, biases in the selection of the cohort, and inadequate data on work and exposure histories. The issue of confounding by coal tar and other materials remains unresolved and limits and complicates overall interpretation of the studies.

! Engholm et al. [1991]

The Swedish Construction Industry's Organization for Working Environment, Safety and

Health conducted a study of cancer mortality and incidence among Swedish construction workers [Engholm et al. 1991]. Male workers (n=226,000) who received medical examinations between 1971 and 1979 were followed for mortality to 1985 and for cancer incidence until December 1984. National mortality and incidence rates were used as reference rates to estimate age and calendar-year-adjusted SMRs and SIRs. Of the original cohort, 2,572 construction workers were road pavers exposed to asphalt. The results for 704 roofers in this cohort are described in section 5.2.2. The average length of the follow-up period for mortality was 11.5 years, and median age during the follow-up was 42 years. This implies that the study cohort was very young at the beginning of the study. The long latency period required for most cancers to become detectable would make it difficult to find any increased risk of occupationally related cancer during such a short follow-up period.

The overall SMR for all causes of death was 0.69 (96 cases), and the SIR for all cancer sites was 0.86 (47 cases). Excess mortality from and incidences of stomach cancer were observed among pavers (SMR=2.01; SIR=2.07), although the number of cases was small and was reported by the authors not to be statistically significant. Lung cancer among pavers was not statistically significantly elevated in the analyses of mortality (SMR=1.10; 95% CI=0.44-2.23) or incidence (SIR=1.24; 95% CI=0.53-2.44). A case-control study of lung cancer was conducted to control for cigarette smoking using data collected during examinations of the employees. Among pavers, there were seven incident cases of lung cancer. The odds ratio (OR) for lung cancer was approximately 2 before adjusting for smoking and population density and approximately 3 after adjusting for smoking.

Significant limitations of this study include a short latency period (11.5 years) and lack of quantitative information about exposures.

! Maizlish et al. [1988]

Maizlish et al. [1988] conducted a proportional mortality study of 27,162 employees who left employment with the California Department of Transportation between 1970 and 1983. Of the 1,570 deaths during that time, 307 occurred among highway maintenance employees considered most likely to have been exposed to asphalt fumes. The authors found that these employees had a statistically significant increase in mortality from emphysema (proportional mortality ratio [PMR]=2.50; 95% CI=1.08-4.92) and a statistically nonsignificant excess of deaths from cancer of the lymphopoietic system, digestive organs, skin, stomach, prostate, and brain. However, the study did not find an excess of deaths from lung cancer.

As the authors clearly state, proportionate mortality studies are inherently limited by the lack of independence among causes of death. Furthermore, no exposure measurements were available for asphalt fumes or other chemicals used by highway maintenance employees, and no data were available on tobacco consumption.

! Bender et al. [1989]

Bender et al. [1989] conducted a retrospective mortality study of a cohort of 4,849 men who each had at least 1 year of experience as a Minnesota highway maintenance employee and who had worked at least 1 day between January 1, 1945, and December 31, 1984. During the study period, 1,530 deaths occurred among these 4,849 men with 96,596 person years at risk. The male population of Minnesota was used as the reference group. The

highway maintenance employee cohort and the reference group were divided into urban and rural categories to evaluate the effects of differences in mortality rates between urban and rural populations.

Mortality from all causes (SMR=0.91; 95% CI=0.86-0.96) and all cancers (overall SMR= 0.83; 95% CI=0.73-0.94; $P<0.01$) was statistically significantly lower than expected. Mortality from lung and respiratory cancers was also significantly less than expected (overall SMR=0.69; 95% CI=0.52-0.90; $P<0.05$) based on 57 deaths (82.6 were expected) regardless of latency or whether the employee worked in an urban or a rural environment. No deaths resulted from melanoma or soft tissue cancers (2.9 and 1.4, respectively, were expected). Bender et al. attributed these decreases in mortality to the healthy worker effect, which may be particularly applicable to highway maintenance employees who have physically demanding jobs.

Statistically significant excesses of cancer mortality rates at industrial sites were reported in the investigation of subgroups, particularly among workers employed for a long time or with long latency (time since first exposure). Statistically significant ($P<0.05$) excesses of mortality were reported for (1) cancer of the mouth and pharynx (SMR=11.10; 95% CI=1.30-40.10) among men who were employed for 40 or more years (two deaths), (2) gastrointestinal cancer among urban workers with 40 to 49 years of latency (SMR=5.82; 95% CI=1.20-17.00) (three deaths), (3) prostatic cancer among workers who started work between 1955 and 1964 (SMR=2.98, $P<0.01$), (4) cancers of the kidneys, bladder and other urinary organs among workers employed for 40 to 49 years (overall SMR=2.92; 95% CI=1.17-6.02), and (5) leukemia among workers employed for 30 to 39 years (overall SMR=4.25; 95% CI=1.71-8.76). The Minnesota Department of Health [1993] concluded that it was unlikely that the excess leukemia mortality observed among the highway maintenance employees was job related.

Interpretation of the study is limited by two considerations. (1) Employees could have been exposed to a variety of confounding factors. (2) The category of "highway maintenance employee" covers a wide range of jobs, including paving, sign painting, mowing, landscaping, and garage and office work.

Analysis of personal-breathing-zone samples and bulk samples of asphalts, oils, and tack coats failed to detect pyrene, B(a)P, or chrysene in any of the substances in use at the time of this study. It is also important to note that highway maintenance work in Minnesota has not involved application of coal-tar products in highway repairs for more than 50 years, thus minimizing risks of exposure to this potential confounder.

! Partanen et al. [1997]

Partanen et al. [1997] recently reported findings from a retrospective cohort study of 9,643 Finnish workers employed for at least 3 months between 1969 and 1984 by one of six companies involved in road paving. This study is a part of a larger ongoing study by the International Agency for Research on Cancer (IARC) on cancer risk in European asphalt workers in seven countries. The cohort was followed for both mortality and cancer incidence through 1994. Relative to the general Finnish population, a statistically significant excess of deaths from lung cancer (SMR=1.5; 95% CI=1.4-1.7) was observed in the entire cohort. However, the excess of lung cancer was evident in both workers exposed to asphalt (SMR=1.4) and workers not exposed to asphalt (construction, SMR=1.4; excavation, SMR=1.8). Associations were also observed for incidences of lung cancer and exposure to asphalt (SIR=1.4; 95% CI=0.9-1.9), as well as exposure to silica, diesel

exhaust, gasoline exhaust, and inorganic dusts. The authors did not attempt to separate the possible effects of asphalt, diesel exhaust, and silica. This report was an abstract from conference proceedings, and thus a thorough evaluation of the study is not possible at this time.

! Milham [1997]
Milham [1997] analyzed occupational and cause-of-death information on 588,090 Washington State males between 1950 and 1989 and 88,071 females between 1974 and 1989 compared to deaths in the general population of the state using an age and year-of-death standardized PMR program. Occupation was abstracted from the "Usual Occupation" field on each death certificate. Ninety-seven percent of all death certificates of males contained information on usual occupation. Based on interviews with next-of-kin, the accuracy of the Usual Occupation field was greater than 75%. Occupations reported included 7,266 deaths among "road graders, pavers, machine operators, and excavators" and 1,437 deaths among "operating engineers." These two groups are believed to have had the greatest likelihood of being exposed to asphalt.

Among workers classified as road graders, pavers, machine operators, and excavators, mortality was statistically significantly increased ($P<0.05$) for cancers of the respiratory system (PMR=1.17, based on 614 deaths) and bronchus, trachea, and lungs (PMR=1.20, based on 558 deaths). Mortality was also increased because of motor vehicle accidents (PMR=1.39, based on 249 deaths).

Among individuals classified as operating engineers, mortality was statistically significantly increased for cancers of the respiratory system (PMR=1.21, based on 136 deaths) and bronchus, trachea, and lungs (PMR=1.21, based on 76 deaths). The PMRs for the categories of all malignancies of the lymphatic and hematopoietic system (PMR=1.42, based on 42 deaths) and other lymphomas (PMR=2.00, based on 10 deaths) were statistically significantly increased ($P<0.05$). Deaths from motor vehicle accidents were also significantly higher (PMR=1.59, based on 47 deaths).

The results of this study are limited by the limitations of proportionate mortality studies, e.g., interdependence of cause-specific PMRs, inaccuracies resulting from obtaining usual occupations from death certificates, and lack of detailed information on exposures and confounders, particularly smoking.

5.2.1.2 Case-Control Studies

! Zahm et al. [1989] compiled information from the Missouri Cancer Registry on 4,431 histologically confirmed lung cancer cases and 11,326 cancer controls diagnosed in white males between 1980 and 1985. Occupational history was obtained from medical records and coded according to the U.S. Bureau of Census' *1980 Alphabetical Index of Industries and Occupation*. Sufficient occupational data to perform an analysis were contained in the medical records of only 52% of the cases and 45% of the controls. After adjusting the analyses for age and cigarette smoking, 32 cases and 64 controls were identified among workers classified as pavers, surfacers, and materials-moving-equipment operators. The OR for working in these occupations was below 1.00 (OR=0.9; 95% CI=0.6-1.5).

5.2.2 Roofers

5.2.2.1 Cohort Studies

There have been several cohort mortality studies of roofers exposed to asphalt fumes. These studies share a common limitation, i.e., the potential for confounding because of

exposure to coal tar and asbestos. Coal tar is a well-recognized human lung carcinogen.

! Hammond et al. [1976]

Many studies use the "Usual Occupation" field from death certificates or interviews as surrogates for exposure information. The earliest epidemiologic study was done by Hammond et al. in 1976 in a retrospective mortality study of members of the United Slate, Tile and Composition Roofers, Damp and Waterproof Workers' Association. Local unions involved only in the tile and slate industries were excluded. Workers included were those involved primarily in applying hot pitch or asphalt to roofs or waterproofing materials to basements. According to the authors, "In former years pitch was used more frequently than asphalt, but today asphalt is more commonly used." Hence workers in this study were exposed to both asphalt and coal tar. The study included 5,939 active, probational, and retired workers who had been in the union for at least 9 years when the studybegan on January 1, 1960. Vital status, primarily from union life insurance records, was established as of December 31, 1970, for 97.5% of this cohort.

Lung cancer mortality was observed to increase with time since a worker first joined the union (a surrogate for length of exposure). A statistically significant excess of lung cancer was observed among workers who had first joined the union more than 20 years earlier (SMR=1.58; 95% CI=1.29-1.94) and also among workers with more than 40 years of work (SMR=2.0; 95% CI=1.28-4.32). A statistically significant excess of upper respiratory cancers (buccal cavity, pharynx, larynx, and esophagus) was also reported (SMR=1.59; 95% CI=1.32-2.76). Although the authors suggested their findings might be explained by exposure to B(a)P, it is impossible to rule out exposure to asphalt or other substances, or smoking, as contributing to the observed excesses of respiratory cancer.

! Menck and Henderson [1976]

Menck and Henderson [1976] conducted an investigation of lung cancer mortality and incidence rates in Los Angeles. A total of 2,161 deaths and 1,777 lung cancer incidents were identified among white males aged 20 to 64 during 1968 and 1970. The subjects' last known occupations and industry affiliations were coded from death certificates or from medical records. Age-, industry-, and occupation-specific estimates of the population at risk were derived from the 1970 census for Los Angeles. A statistically significant excess of lung cancer (mortality and incidence combined) was observed for the occupational category of roofers (SMR=8.78; $P<0.01$).

In addition to possible confounding by coal tar, this study is limited in that the analysis was based on the "Usual Occupation" field on the death certificate, which may not accurately reflect lifetime work histories of individuals. Moreover, the authors were unable to analyze the data by level of exposure, duration of exposure, or latency.

! Engholm et al. [1991]

The Swedish Construction Industry's Organization for Working Environment, Safety and Health [Engholm et al. 1991] conducted a study of cancer mortality and incidence among Swedish construction workers (described earlier in section 5.2.1), who included 704 roofers. Increased mortality and/or incidence were observed among roofers for cancers of the lung (SMR=2.79, three deaths; SIR=3.62, four cases) and stomach (SMR=2.01, five deaths; SIR=1.98, one case); lymphatic and hematopoietic tumors (SMR=2.68, two deaths); and leukemia (SIR=2.26, one case).

As noted, these findings were all based on a relatively small number of cases and are therefore statistically unstable. After controlling for smoking, an OR of 6, based on three cases, was derived for lung cancer among roofers in a nested case-control analysis of incidents.

! Hrubec et al. [1992]

Hrubec et al. [1992] conducted a mortality study of approximately 300,000 veterans who served in the U.S. Armed Forces between 1917 and 1940. Information on occupation, industry of employment, and smoking history was obtained from a questionnaire mailed to veterans in 1954 and a follow-up questionnaire mailed to nonrespondents in 1957. The 284,046 respondents to this questionnaire were followed for vital status ascertainment as of 1980. Poisson regression models were used to estimate rate ratios for each occupation and industry while controlling for smoking habits. An elevated risk of respiratory cancer was observed among the job category "roofers and slaters" (rate ratio=3.0; 90% CI=1.30-6.75).

One strength of this study is that the analyses were adjusted for smoking. Weaknesses are that information on work histories was obtained at one point in time only and that it was obtained from a self-administered questionnaire.

! Pukkala [1995]

Pukkala [1995] reported findings from a study in which the entire 1970 population of Finland was followed from 1971 to 1985 to track cancer incidence. An analysis of the relationship between cancer incidence and occupation was performed among individuals between the ages of 25 to 64 in 1970. The analysis controlled for possible confounding by social class, age, and calendar time; smoking was not considered in the analysis. The SIR for cancers of the lung, bronchus, and trachea among men coded as "asphalt roofers" in the 1970 census was 3.25 (95% CI=1.92-5.13).

! Milham [1997]

In Washington State, 1,057 deaths among male residents classified as "roofers and slaters" were recorded for the years 1950 through 1989 [Milham 1997]. For this occupational classification, statistically significant ($P<0.01$) PMRs were observed for cancers of the respiratory system (PMR=1.53, based on 105 deaths); larynx (PMR=2.59, based on six deaths, $P<0.05$); and bronchus, trachea, and lungs (PMR=1.44, based on 86 deaths); as well as asthma (PMR=2.86, based on seven deaths); emphysema without bronchitis (PMR=1.63, based on 28 deaths); and cirrhosis of the liver with alcoholism (PMR=1.49, based on 17 deaths). Of note is that PMRs from these causes were elevated despite a significant increase in deaths from falls from heights (PMR=4.00, based on 17 deaths). Not statistically significant increased PMRs were noted for cancers of the buccal cavity and pharynx (PMR=1.67, based on nine deaths), diseases of the respiratory system, chronic bronchitis with and without emphysema, cirrhosis of the liver without alcoholism, and psychosis. In contrast, mortality from diseases of the circulatory system was statistically significantly decreased (PMR=0.88, based on 364 deaths).

The results of this study are limited by the interdependence of cause-specific PMRs, the accuracy of the "Usual Occupation" field on death certificates, and lack of detailed information on exposures and confounders, particularly smoking.

5.2.2.2 Case-Control Studies

Three case-control studies examined the relationship between lung cancer and occupation as a roofer [Zahm et al. 1989] or as a roofer

and slater [Schoenberg et al. 1987; Morabia et al. 1992]. Each of the studies included only histologically confirmed lung cancer cases among males. All studies adjusted the risk measures for smoking and found elevated ORs that were not statistically significant. Only Zahm et al. evaluated roofers as a separate occupation, while Morabia et al. and Schoenberg et al. analyzed roofers and slaters as a single occupational group. Inclusion of slaters in these analyses could dilute the effects of relationships between exposure to asphalt roofing products and lung cancer.

! In the study by Zahm et al. [1989], six cases and seven controls were coded as roofers (Bureau of the Census code 595). There was a twofold increase in lung cancer among roofers that was not statistically significant (OR=2.1; 95% CI=0.6-8.2).

! Schoenberg et al. [1987] conducted a hospital-based study of 763 lung cancer cases and 900 controls among white males in New Jersey. New cases diagnosed between September 1980 and October 1981 and reported to the New Jersey State Department of Health were included in the study. Occupational history was obtained by interviewing the patient or the patient's next-of-kin. Thirteen cases and 8 controls were identified as roofers or slaters. The OR was not statistically significant, although it was greater than 1 (OR=1.7; 95% CI=0.7-4.4).

! Morabia et al. [1992] interviewed 1,793 cases and 3,228 controls (one cancer control and one noncancer control per case), matched for age, race, geographical area, questionnaire version, and history of smoking, from 24 hospitals in nine metropolitan areas in the United States between 1980 and 1989. Seven cases and six controls were identified as roofers and slaters. The ORs for roofers and slaters were not statistically significant (OR=2.1; 95% CI=0.7-6.2).

Neither Morabia et al. nor Schoenberg et al. identified the number of individuals who were exposed to hot asphalt roofing products. Indeed, it is not clear that either the cases or the controls had significant occupational exposure.

5.2.3 Meta-analysis of Asphalt Workers and Roofers

! Partanen and Boffetta [1994]

Partanen and Boffetta [1994] conducted a comprehensive review and meta-analysis of 20 epidemiologic studies of asphalt workers and roofers. The "most relevant" relative risk (RR) estimates (OR, SMR or SIR) were extracted from the reports for the meta-analysis. The authors defined the most relevant RR estimates to be those that (1) best approximated exposure to asphalt, (2) were adjusted to the extent possible for potential confounders, and (3) used an appropriate induction-latency period. Summary RRs were estimated from the individual study findings for (1) all asphalt workers and roofers, (2) road pavers and highway maintenance workers, (3) roofers, and (4) "miscellaneous or unspecified" asphalt and bitumen workers. The summary RR for lung cancer was increased among roofers (RR=1.78; 95% CI=1.50-2.10) and miscellaneous or unspecified workers (RR=1.49; 95% CI=1.22-1.80), but not among pavers and highway maintenance workers (RR=0.9; 95% CI=0.8-1.0). Statistically significant increased RRs were also observed for stomach cancer among roofers (RR=1.7; 95% CI=1.1-2.5) and for nonmelanotic skin cancer among pavers and highway maintenance workers (RR=2.2; 95% CI=1.2-3.7). Similar results for lung cancer were found when the analysis was restricted to studies that controlled for cigarette smoking. The authors appropriately suggest that the available studies (and hence their meta-analysis) were "poorly focused" to address the question of whether asphalt exposure is carcinogenic because of numerous limitations in the design of these studies, many of which are

discussed in previous sections.

5.2.4 Other Studies of Asphalt Exposure

5.2.4.1 Cohort Studies

! Hansen [1989b]

In a retrospective cohort mortality study, Hansen [1989b] compared the mortality rates of 1,320 Danish workers employed in the asphalt industry at asphalt plants, roofing felt plants, and one tar plant with those of 43,024 unskilled Danish employees employed in other industries. The cohort was selected from the November 9, 1970, Danish census conducted by the Danish National Bureau of Statistics and was traced until November 9, 1980. There were 113 deaths among the 1,320 asphalt employees and 3,811 deaths among the 43,024 unskilled employees.

During the last 5 years of the 10-year follow-up study, asphalt employees aged 45 or older had a statistically significant SMR for all malignant neoplasms (SMR=1.59; 95% CI=1.06-2.28) and brain cancers (SMR=5.00; 95% CI=1.03-14.61) and elevated SMRs for digestive cancer (SMR=1.57; 95% CI=0.58-3.43), respiratory cancer (SMR=1.52; 95% CI=0.76-2.71), and bladder cancer (SMR=2.91; 95% CI=0.60-8.51). Limitations of the study include lack of information about the length of employment in the asphalt industry and the extent of exposure.

5.2.4.2 Case-Control Studies

Three case-control studies [Mommsen et al. 1983; Risch et al. 1988; Bonassi et al. 1989] examined the relationship between bladder cancer and a variety of occupational exposures, including exposures to asphalt. Broad categories of jobs or industries were included that were not specific to asphalt, limiting the use of these studies in assessing risks of asphalt exposures.

! Mommsen et al. [1983]

Mommsen et al. [1983] reported findings of a study of 212 bladder cancer cases (165 men and 47 women) admitted to the Department of Oncology and Radiotherapy in Aarhas, Denmark. Information on male cases was collected from 1977 to 1979 and on female cases from 1977 to 1980. The 259 controls were matched for age, sex, geographic area, and degree of urbanization. Information on smoking and occupational history was also collected from each study participant by questionnaire. An approximately 2½-fold increase in risk of bladder cancer was reported for occupational exposure to “petroleum or asphalt.”

! Risch et al. [1988]

Risch et al. [1988] studied 781 controls and 739 patients with bladder cancer diagnosed between 1979 and 1982 in Edmonton and Calgary, AB, and Toronto and Kingston, ON, Canada. Information on occupation, tobacco use, and other factors likely to be related to bladder cancer was collected during interviews. Histologic verification of all tumors was obtained. However, only 67% of the eligible cases and 53% of the eligible controls participated in the study. A statistically significant association (OR=3.11; 95% CI=1.19-9.68) was observed among workers exposed to “tar and asphalts” during a full-time job of at least 6 months duration and at least 8 years of latency.

! Bonassi et al. [1989]

Bonassi et al. [1989] examined the relationship between bladder cancer and potential lifetime occupational exposure to PAHs in 121 cases and 342 controls. Cases were histologically confirmed, and smoking history was obtained. Eleven occupational categories were selected on the basis of their potential for exposing workers to PAHs, and subjects were classified into these categories if they had worked in one for a year or more. A statistically nonsignificant association (OR=1.4; 95% CI=0.27-7.28) was observed between bladder cancer and employment as a

"road mender" based on two cases and six controls.

! Jensen et al. [1988]

Jensen et al. [1988] investigated the relationship between occupational exposure and incidence of renal pelvis and ureter cancer among Danish residents. Occupational history and demographic data were obtained by interviewing 96 cases and 294 hospital controls; cases and controls were matched by age (within 5 years), hospital, and sex. Of the male study subjects, nine cases and six controls reported exposure to asphalt or tar. Smoking-adjusted RRs for exposure to asphalt or tar were statistically significantly elevated (RR=5.5; 95% CI=1.6-19.6).

! Vineis et al. [1988]

One case-control study evaluated the relationship between lung cancer and occupation or occupational exposure to well-known and suspected lung carcinogens. Vineis et al. [1988] combined 2,973 male cases and 3,210 controls from five studies conducted throughout the United States and adjusted them for age, birth cohort, and cigarette use. Forty-five cases and 37 controls were classified into the category of "roofers and asphalt workers." Risk of lung cancer for the combined group of "roofers and asphalt workers" was not statistically significant [OR=1.4; 95% CI=0.9-2.3)].

! Chiazze et al. [1993]

Chiazze et al. [1993] conducted a case-control study of malignant and nonmalignant respiratory disease in workers employed in a fiberglass insulation production facility. The maximum number available for a matched analysis of interview data was 144 lung cancer cases with 260 controls and 101 nonmalignant respiratory disease cases with 183 controls. Quantitative estimates of lifetime exposure to asbestos, talc, asphalt fumes, formaldehyde, and silica were calculated. Asphalt exposure was dichotomized in "never-exposed" versus "exposures $\geq$0.01-mg/m^3" days. ORs were calculated using conditional logistic regression adjusted for age, smoking, and occupational exposure to respirable fibers, asbestos, talc, formaldehyde, silica, and total particulates. Asphalt exposure was not related to either lung cancer (OR=0.96; 95% CI=0.65-1.4) or nonmalignant respiratory disease (OR=1.3; 95% CI=0.82-2.2) for workers in the higher exposure group.

! Siemiatycki [1991]

Siemiatycki [1991] obtained occupational history and exposure information from interviews with 3,730 hospital-based cancer cases and 533 population-based controls residing in the province of Quebec, Canada. ORs were calculated for 23 cancer sites adjusted for confounding variables, including smoking. Statistically significant increased ORs for any exposure to asphalt were observed for colon cancer (OR=1.6; 95% CI=1.1-2.5, based on 22 cases), while ORs for cancers of the esophagus, stomach, pancreas, lungs, prostate, and non-Hodgkins lymphoma were approximately 1 and not statistically significant.

! Austin et al. [1987]

Austin et al. [1987] conducted a hospital-based case-control study to examine the role of occupation and other factors in the etiology of hepatocellular carcinoma. Cases and controls were patients at one of five participating hospitals. Each case was matched to two controls by age, sex, race, and study hospital. Occupational or recreational exposures to 26 substances, including asphalt, were obtained from each of the 80 cases and 146 matched controls. Seven cases and five controls reported exposure to asphalt for at least 3 hr/wk for at least 6 months at some time during their lives (RR=3.2; 95% CI=0.9-11). Of these cases and controls, one case and one control had worked for more than 10 years in road building, and one other case had worked as a laborer in an asphalt manufacturing company.

5.2.5 Conclusions

5.2.5.1 Lung Cancer among Pavers

Epidemiologic studies of lung cancer among pavers exposed to asphalt fumes have yielded contradictory results. That is, while some studies have reported an elevated risk of lung cancer, design limitations of these studies preclude drawing any strong conclusions [Hansen 1989a; Engholm et al. 1991; Partanen et al. 1997; Milham 1997]. Of particular concern is the possibility of confounding from co-exposures to coal tar and other potential lung carcinogens (e.g., diesel exhaust, silica, and asbestos) [Hansen 1989a]. Failure to control adequately for smoking is also an issue in several studies [Engholm et al. 1991; Milham 1997]. Several studies of pavers or highway workers have failed to demonstrate an excess of lung cancer [Maizlish et al. 1988; Bender et al. 1989]. A meta-analysis of all these studies failed to find overall evidence for a lung cancer risk among pavers exposed to asphalt [Partanen and Bofetta 1994]. Hence, the epidemiologic evidence for an association between lung cancer and exposure to asphalt in paving is inconclusive at this time.

5.2.5.2 Lung Cancer among Roofers

In contrast to pavers, epidemiologic studies of roofers have generally demonstrated an excess number of lung cancer cases [Hammond et al. 1976; Menck and Henderson 1976; Engholm et al. 1991; Hrubec et al. 1992; Pukkala 1995; Milham 1997; Zahm et al. 1989; Schoenberg et al. 1987; Morabia et al. 1992]. The metaanalysis by Partanen and Boffetta [1994] has also revealed an overall excess of lung cancer among roofers. However, it is uncertain to what extent these findings may be attributable to asphalt exposures. In the past, roofers have been exposed to coal tar and asbestos, which are known human lung carcinogens, as well as asphalt. Hence, while strong epidemiologic evidence of an association between lung cancer and working as a roofer exists, it is uncertain whether asphalt or other substances are responsible for these findings.

5.2.5.3 Cancers at Other Sites

A few studies have reported an association between cancers at sites other than lungs and occupations having the potential for exposures to asphalt [Mommsen et al. 1983; Risch et al. 1988; Bonassi et al. 1989; Jensen et al. 1988]. Of particular interest is an association reported in several case-control studies between bladder and renal cancers and occupations having exposures to asphalt. Isolated studies have reported associations between occupations with asphalt exposure and cancers of the brain, liver, and other digestive organs [Hansen 1989b; Austin et al. 1987; Siemiatycki 1991]. Interpretation of the findings of these studies is limited by a lack of consistency among studies and issues of the confounding effects of other substances. Furthermore, many of these findings are from population-based, case-control studies organized by broad job classifications that are prone to errors in defining asphalt exposures [Mommsen et al. 1983; Risch et al. 1988; Bonassi et al. 1989; Jensen et al. 1988; Siemiatycki 1991]. Thus, the evidence for an association between exposure to asphalt and nonrespiratory cancers is weak and requires further confirmation by studies with better control of confounding variables and better identification of asphalt exposures.

6 Experimental Studies

This chapter provides a review of the *in vitro* and *in vivo* animal studies completed since the publication of the NIOSH criteria document on asphalt fumes in 1977. Ideally, these studies should provide definitive data regarding the genotoxicity, carcinogenicity, and other toxic effects of asphalt-based paints and asphalt fumes generated during paving and roofing operations. Because of the difficulty in obtaining a sufficient quantity of paving and roofing asphalt fumes in the field, however, many of the studies reviewed used laboratory-generated asphalt fumes.

6.1 Genotoxicity

Since publication of the NIOSH criteria document [1977a], genotoxic effects were described in the following studies: National Toxicology Program (NTP) [NTP 1990], Blackburn and Kriech [in AI 1990a], Machado et al. [1993], Reinke and Swanson (laboratory-generated asphalt fumes) [1993], Qian et al. [1996], Schoket et al. [1988a,b], Toraason et al. [1991], and Wey et al. [1992]. However, genotoxic effects were not observed by Reinke and Swanson in a study of fumes collected from a hot-mix asphalt storage tank [1993].

6.1.1 Mutagenic Effects

The NTP evaluated the mutagenic potential of roofing asphalt fume condensate fractions and neat (unfractionated) asphalt fumes from the study by Sivak et al. [1989]. Sivak et al. heated Type III roofing asphalt to 316 °C (601 °F) to generate fume condensates and then separated the condensates using HPLC. Five fractions, designated A through E, of the condensates and unfractionated asphalt fumes were examined for mutagenic potential using the Ames *Salmonella* mutagenicity assay. The chemical composition of each fraction is provided in section 6.2.1. Fractions B and C and recombined fractions A through E were reported as positive, fractions A and D and the unfractionated fumes were weakly positive, and fraction E was negative. Positive responses were observed only with metabolic activation (S9) [NTP 1990; Zeiger 1990].

The same fractionated asphalt fume condensates from the study by Sivak et al. [1989] were tested by Blackburn and Kriech [in AI 1990a] using the modified Ames *Salmonella* mutagenicity assay. The results were consistent with those of the NTP study. Mutagenicity indices of 21 asphalt fume samples collected under a variety of conditions ranged from 0 to 8.8, with an average of 4.7. These indices were approximately 150-fold less than the indices for coal-tar-pitch fumes.

Machado et al. [1993] evaluated the mutagenic activity and PAH content of laboratory-generated fumes from a variety of asphalts. Materials examined included two Type III roofing asphalts representing different crude petroleum sources. One of the roofing asphalts was similar to the asphalt (air-blown using a ferric chloride catalyst) examined by Niemeier et al. [1988] and Sivak et al. [1989]. Machado et al. also evaluated 18 paving asphalts representing 14 crude petroleum sources and various processing conditions and a Type I coal-tar-pitch fume. Fume condensates were examined for mutagenic activity with a modified Ames *Salmonella* mutagenicity assay

(Table 6-1) and for PAH content. The fume generation temperature of all roofing materials was either 232 ˚C or 316 ˚C (450 °F or 601 °F) and that of all paving materials was 163 ˚C (325 °F). One sample of paving material was heated to 221 ˚C (430 °F). Machado et al. reported that all samples tested showed weak-to-moderate mutagenic activity (Table 6–1). Moreover, the mutagenic responses to the asphalt fume condensates were approximately 100-fold less than mutagenic responses to the coal-tar-pitch samples.

Results of the analyses for PAH content, as measured by HPLC fluorescence, of the roofing and paving asphalts, coal-tar pitch, and their fume condensates were as follows. Concentrations of individual PAHs in samples of roofing and paving asphalt and asphalt fume condensates were less than 50 ppm by weight. Most concentrations of individual PAHs in roofing asphalt or fumes were less than10 ppm, and all concentrations in paving asphalts or fumes were less than 2 ppm. Concentrations of individual PAHs in the coal-tar-pitch samples were 100- to 1,000-fold higher than in the roofing and paving samples. For example, B(a)P was detected in all samples examined; maximum concentrations in asphalt, coal-tar pitch, and asphalt and coal-tar-pitch fume condensates were approximately 6 ppm, 18,000 ppm, 2.8 ppm, and 480 ppm, respectively.

Machado et al. attempted to correlate mutagenicity indices with PAH content. The correlation coefficient of the pooled data varied from 0.17 to 0.86, depending upon which samples were included in the analysis. The investigators also suggested that the crude petroleum source, along with processing conditions, had some influence on the PAH content of the various materials tested.

Reinke and Swanson [1993] compared the chemistry of PAHs and S-PACs and mutagenic potential of field- and laboratory-generated asphalt fumes from an asphalt cement. Temperatures ranged from 146 to 157 ˚C (295 to 315 °F) for the field samples and from 149

Table 6–1. Summary of *Salmonella* mutagenicity data for asphalt and coal-tar pitch fume condensates (adapted from Machado et al. 1993)

Source of fume condensates	Sample no.	Fume generation temperature, °C (°F)				Mutagenicity index*
		163 (235)	221 (430)	232 (450)	316 (601)	
Roofing asphalt†	2	−	−	+	−	12 (1)
	2	−	−	−	+	10 (1)
Roofing asphalt†	3	−	−	+	−	12 (2)
	3	−	−	−	+	10 (1)
Paving asphalt‡		+	−	−	−	14§
		−	+**	−	−	18†† (13)
Type I coal-tar pitch		−	−	+	−	725 (35)
		−	−	−	+	1555 (5)

− No fumes were generated at this temperature. +Fumes were generated at this temperature.
*Slope of dose-response curve, i.e., revertants per microliter of dosing solution (± asymptotic standard error).
†Two Type III roofing asphalts used representing different crude sources. One of the roofing asphalts was air blown using ferric chloride as a catalyst (no. 3), and the other was air blown without the use of a catalyst (no. 2).
‡Eighteen samples of paving asphalt fume condensates used representing 14 crude oil sources and various process conditions.
§Mean of 37 experiments on 17 paving asphalt samples. One to three experiments were run for each sample. Mutagenicity indices ranged from 5 to 49, and the mutagenicity indices for the pooled data ranged from 6 to 29.
**One paving asphalt was generated and tested at 221 °C (430 °F).
††Mean of three experiments.

to 316 °C (300 to 601 °F) for the laboratory samples. The field asphalt fume condensates were collected from the headspace of an asphalt storage tank at a hot-mix asphalt production plant. Fumes were collected into a cold trap for approximately 36 continuous hours.

A summary of the chemical analyses (GC/MS) for PAHs and S-PACs and the modified Ames *Salmonella* mutagenicity assay is provided in Table 6–2. The mutagenicity index of the storage tank headspace asphalt fumes was between 0 and 1, while the mutagenicity indices of the asphalt fumes generated in the laboratory at 149 and 316 °C (300 and 601°F) were 5.3 and 8.3, respectively. The authors noted positive trends between mutagenicity indices and the percentage of ≥three-ring PAHs and S-PACs. They suggested that the increased mutagenicity of the fumes generated at 316 °C (601 °F) could be attributed to its increased content of four-ring S-PACs (Table 3–7).

A study was undertaken in Europe by De Méo et al. [1996a] to compare the mutagenic potential of fume condensates generated at 160 and 200 °C (320 and 392 °F) from coal tar and two paving asphalts (45/60 pen and 160/210 pen) in a modified Ames *Salmonella* mutagenicity assay [De Méo et al. 1996b]. Modifying the procedure of Brandt et al. [1985], the authors generated coal tar and asphalt fume condensates (a mix of the vapor phase and the particulate phase) that they considered to be representative of fumes produced in the field [1996a]. The condensates were tested for mutagenic activity in the presence and absence of metabolic activation (S9) using the *Salmonella* tester strains TA98, TA100, YG1041, and YG1042. All fume condensates were mutagenic to all bacterial tester strains only in the presence of metabolic activation. The mutagenic potencies of the coal-tar fume condensates were 15- to 600-fold higher than those of the asphalt fume condensates. The authors further investigated the effect of these fume condensates on *in vitro* DNA-adduct formation; these results are presented in section 6.1.3.

Robinson et al. [1984] examined the mutagenic potential of several asphalt-based paints using the Ames *Salmonella* mutagenicity assay. None of the asphalt-based paints demonstrated mutagenic activity in either the presence or absence of metabolic activation (S9).

6.1.2 Chromosomal Aberrations

Condensates of Type I and Type III roofing asphalt fumes generated in the laboratory at 316±10 °C (601 °F) using the same methodology as in Sivak et al. [1989] caused a dose-related increase in micronucleus formation in exponentially growing Chinese hamster lung fibroblasts (V79 cells) [Qian et al. 1996]. The results of immunofluorescent antibody staining

Table 6–2. Summary of data from Reinke and Swanson [1993], mg/mL

	PAHs		S-PACs	Mutagenicity
Asphalt fume condensates	**<3-ring**	**≥3-ring**	**≥3-ring**	**index***
Storage tank headspace	7.0 (84)†	0.8 (9)	0.5 (7)	>0 and <1
Lab-generated at 149 °C	6.9 (51)	2.6 (19)	4.0 (30)	5.3
Lab-generated at 316 °C	0.5 (7)	2.8 (38)	4.1 (55)	8.3

*Raw data not provided. Positive control had an index of 4.6.
†Percentage of total PAHs and S-PACs.

showed that both roofing asphalt fume condensates induced mainly kinetochore-positive micronuclei (68 to 70%). The authors suggested that Type I and Type III roofing asphalt fume condensates are aneuploidogens and possess some clastogenic activities. The asphalt fume condensates cause mainly cytogenetic damage by spindle apparatus alterations in cultured mammalian cells.

Reinke and Swanson [1993] tested three paving asphalt fume condensates generated in the field and in the laboratory in an unspecified chromosomal aberration assay. Results were negative. The authors suggested that "the absence of positive findings" may be explained by the fact that this assay was not "optimized for petroleum asphalt fumes."

6.1.3 DNA-Adduct Formation

De Méo et al. [1996a] also tested coal tar and paving (45/60 pen and 160/210 pen) asphalt fume condensates generated at 160 and 200 °C (320 and 392 °F) for their ability to produce DNA adducts *in vitro* when added to calf thymus DNA. DNA-adduct formation was assessed by ^{32}P-postlabeling; the fume condensates were diluted in acetone to a final concentration of 20 µg in 1 mL. B(a)P was used as the positive control. The authors reported that all of the fume condensates induced DNA-adduct formation. No specific DNA adducts were identified. The authors further noted that the patterns of the autoradiograms of the DNA adducts demonstrated qualitative differences, indicating qualitative differences in the nature of the compounds in the coal tar and asphalt fume condensates responsible for the formation of these adducts.

As a continuation of the De Méo et al. study [1996a], Genevois et al. [1996] tested the same coal tar and paving asphalt fume condensates for their ability to induce DNA-adduct formation *in vivo*. They applied 100 µL (about 100 mg) of the undiluted fume condensates to the shaved dorsal skin of BD4 rats (three rats per group) twice 2 days apart; three untreated rats served as the control group. Twenty-four hours after the last treatment, all of the rats were sacrificed, and skin, lungs, and lymphocytes were collected. DNA-adduct formation was assessed by ^{32}P-postlabeling. DNA adducts were found in skin, lungs, and lymphocytes of all the treated rats, but no specific DNA adducts were identified. These *in vivo* data are in agreement with De Méo et al.'s *in vitro* data and indicate qualitative differences in the nature of the compounds in the coal tar and asphalt fume condensates responsible for the induction of the DNA adducts.

De Méo et al. and Genevois et al. also analyzed the fume condensates for PAH content using HPLC-fluorescence. The data indicated that while large amounts of unsubstituted PAHs are present in the coal-tar fumes, these compounds are only minor constituents of the asphalt fume condensates.

In two studies, Schoket et al. reported upon the formation of DNA adducts in (1) mice [1988a] and (2) adult and fetal human skin samples maintained in short-term tissue culture [1988b]. In both studies, asphalt- or creosote-based paints or pharmaceutical-grade coal tar were applied topically, and DNA-adduct formation was assessed by ^{32}P-postlabeling. The results in both studies suggested that a variety of adducts were formed from the three materials; however, no specific DNA adducts were identified.

The DNA-adduct concentrations found in mouse skin 24 hours after a single application of the test materials are listed in Table 6–3. Mice that received multiple applications of the three agents showed accumulations of DNA adducts in both skin and lung tissue. The DNA-adduct concentrations observed in the multiple- treatment studies were reported in

Table 6–3. Concentration of DNA adducts in mouse skin 24 hr after a single application of asphalt- or creosote-based paints or pharmaceutical-grade coal-tar solution (adapted from Schoket et al. 1988a)

Treatment agent*	Dose per mouse	Femtomole adducts, µg of DNA†
Asphalt‡	3 mg	0.00
Asphalt	15 mg	0.09
Creosote	5 µL	0.19
Creosote	25 µL	0.40
Coal tar	6 mg	0.14
Coal tar	30 mg	0.38

*Control values not reported.
†One femtomole adduct per microgram of DNA = 33 adducts per 10^8 nucleotides [Schoket et al. 1988b].
‡Referred to as bitumen by Schoket et al. [1988a].

graphic form only; the adduct concentrations found in the lungs were consistently lower than those in the skin. The authors concluded that the detection of DNA adducts in the lungs demonstrated that PAHs in the three agents were absorbed from the skin and metabolically activated in organs distant from the site of application.

DNA-adduct concentrations found in adult human skin 24 hours after a single application of the test materials are listed in Table 6–4. The authors concluded that similar amounts of DNA adducts are formed from these materials in both mouse and human skin.

6.1.4 Intercellular Communication

The five asphalt roofing fume fractions used by Sivak et al. [1989] were tested for inhibition of intercellular communication. The inhibition of intercellular communication by a tumor promoter is believed to isolate an initiated or preneoplastic cell from the growth regulatory signals of surrounding cells, leading to the development of neoplasia. All fractions inhibited intercellular communication in Chinese hamster lung fibroblasts (V79 cells) [Toraason et al. 1991]. The greatest activity occurred in fractions D and E, and the least activity in fraction A.

Similarly, Wey et al. [1992] examined the effect of these fractions on intercellular communication in human epidermal keratinocytes. All asphalt roofing fume fractions inhibited intercellular communication in a concentration-dependent fashion.

6.2 Carcinogenicity

Since publication of the NIOSH criteria document [1977a], there have been several reports of carcinogenicity in mice following applications of laboratory-generated asphalt roofing fume condensates [Thayer et al. 1981; Niemeier et al. 1988; Sivak et al. 1989, 1997], raw roofing asphalt [Sivak et al. 1989, 1997], and asphalt-based paints [Robinson et al. 1984; Bull et al. 1985] to the skin of mice. However, in another study [Emmett et al. 1981], raw roofing asphalt applied dermally to mice was not carcinogenic.

6.2.1 Roofing Asphalt Fume Condensates and Raw Asphalt

! Niemeier et al. [1988]

Niemeier et al. [1988] investigated the tumorigenicity of fume condensates generated in the laboratory at 232 and 316 °C (450 and 601 °F) from Type I and III roofing asphalt and

Type I and III coal-tar pitch. All fume samples were cryogenically collected. Fume condensates were applied biweekly to the skin of male CD-1 (nonpigmented) and C_3H/HeJ (pigmented) mice for 78 weeks. Eighteen groups of 50 mice per strain received these applications singly or in combination. Half of each group was exposed to simulated sunlight. Tumors were produced by fume condensates of both types of asphalt (Tables 6–5 and 6–6) and both types of coal-tar pitch. The majority of benign tumors were papillomas; the majority of malignant tumors were squamous cell carcinomas. Both strains of mice exposed to asphalt fumes had significantly (P=0.01) more tumors than the control groups, although the

Table 6–4. Concentration of DNA adducts in adult human skin 24 hr after a single application of asphalt- or creosote-based paints or pharmaceutical-grade coal-tar solution (adapted from Schoket et al. 1988b)

Treatment agent	Dose per skin patch	Femtomole adducts, µg of DNA*
Solvent†	150 µL	0.10
Asphalt‡	15 mg	0.22
Creosote	25 µL	0.31
Coal tar	30 mg	0.35

*One femtomole adduct per microgram of DNA=33 adducts per 10^8 nucleotides [Schoket et al. 1988b].
†Ethanol or tetrahydrofuran.
‡Referred to as bitumen by Schoket et al. [1988a].

Table 6–5. Final histopathology of tumors induced in CD-1 mice treated dermally with roofing asphalt fume condensates (adapted from Thayer et al. 1981)

		Tumor-bearing animals		Tumors		
Material tested	Sunlight	Benign	Malignant	Papilloma	Squamous cell carcinoma	Total*
Type I asphalt @ 232 °C†	−	6	0	12	0	12
	+	2	0	3	0	3
Type I asphalt @ 316 °C†	−	13	1	18	0	19
	+	3	0	3	0	3
Type III asphalt @ 232 °C†	−	9	1	11	1	13
	+	5	2	5	1	7
Type III asphalt @ 316 °C†	−	13	3	17	1	20
	+	4	1	5	1	6
B(a)P‡	−	24	11	43	10	58
	+	9	3	11	1	18
Cyclohexane/acetone§	−					0
	+					0

*Other tumor types observed included fibrosarcomas, kerato-acanthomas, fibromas, and unclassified benign epitheliomas.
†25 mg of total solid per application.
‡5 µg per application.
§50 µL of a 1:1 solution.

Table 6–6. Final histopathology of tumors induced in C_3H/HeJ mice treated dermally with roofing asphalt fume condensates (adapted from Thayer et al. 1981)

	Tumor-bearing animals	Tumors

Material tested	Sunlight	Benign	Malignant	Papilloma	Squamous cell carcinoma	Total*
Type I asphalt @ 232 °C†	−	24	22	34	26	76
	+	14	27	22	25	62
Type I asphalt @ 316 °C†	−	13	31	27	31	78
	+	18	26	36	26	73
Type III asphalt @ 232 °C†	−	15	25	32	19	66
	+	11	20	14	19	54
Type III asphalt @ 316 °C†	−	12	28	24	36	82
	+	20	18	34	20	65
B(a)P‡	−	11	27	12	29	53
	+	7	27	11	22	43
Cyclohexane/acetone§	−	0	0	0	0	0
	+	1	0	2	2	4

*Other tumor types observed included fibrosarcomas, kerato-acanthomas, fibromas, and unclassified benign epitheliomas.
†25 mg of total solid per application.
‡5 µg per application.
§50 µL of a 1:1 solution.

C_3H/HeJ mice demonstrated a greater tumorigenic and carcinogenic response to both asphalt and coal-tar-pitch fume solutions than did the CD-1 mice. The C_3H/HeJ mice showed a significant increase (P=0.01; Fisher-Irwin exact test) in tumorigenic response for both types of condensed asphalt fumes generated at 316 °C (601 °F) compared with tumors generated at 232 °C (450 °F). The mean time to tumor appearance was longer for all groups of CD-1 mice compared with the corresponding C_3H/HeJ groups. The mean latency period ranged from 39.5 to 56.1 weeks among the C_3H/HeJ groups and from 47 to 76.5 weeks among the CD-1 groups treated with roofing asphalt fume condensates. Mean latency time increased with simulated sunlight, which generally inhibited tumorigenic response, possibly because of photo oxidation or photodestruction of the carcinogenic components of the test materials. Niemeier et al. [1988] concluded the following:

- Unlike the carcinogenic activity of coal-tar pitch, that of asphalt fume condensates could not be explained by B(a)P (or PAH) content.

- The carcinogenic activity of the asphalt fume condensates may have been due to the high concentrations of aliphatic hydrocarbons, which have co-carcinogenic effects.

- Higher generation temperatures may have increased carcinogenic effects.

! Sivak et al. [1989, 1997]

Sivak et al. [1989, 1997] heated Type III roofing asphalt from the same lot used by Niemeier et al. [1988] at 316 °C (601 °F), generated fume condensates, and separated them by HPLC (see Belinky et al. 1988 for a description of this procedure). The chemical composition of fractions A through E, as analyzed by GC/MS, is provided in Table 3–8.

Raw roofing asphalt, neat asphalt fumes, asphalt heated to 316°C (601°F) with fumes allowed to escape, reconstituted asphalt fumes, and the asphalt fume fractions—individually and in various combinations, were then tested for their carcinogenic and tumor-promoting activity. Fractions A through E were dissolved in a 1:1 solution of cyclohexane and acetone to yield concentrations proportional to their presence in the unfractionated (neat) asphalt fume condensate, i.e., 64.1%, 8.3%, 10.5%, 11.5% and 5.6%, respectively, and were applied biweekly to 40 groups of male C_3H/HeJ mice and two groups of male Sencar mice (30 mice per group) for 104 weeks (2 years). The Sencar mice were included to allow for possible genetic variation and susceptibility to tumor development.

A single initial treatment of B(a)P followed by individual treatments with fractions A, D, and E were used to test the tumor-promoting activity of the asphalt fume condensate. The co-carcinogenicity of fractions A, D, and E was tested with three different doses of B(a)P. Fractions A, D, and E were used because they were the fractions Sivak et al. [1989, 1997] deemed most likely to exhibit co-carcinogenic or tumor-promoting activity based on their chemical compositions, i.e., primarily long chain alkanes and phenolic compounds. One of the two groups of Sencar mice was treated with neat asphalt fumes (whole condensate), and the other was used as an unexposed solvent control. The negative control group was treated with cyclohexane and acetone, and the positive control groups were treated with three different concentrations of B(a)P.

Table 6–7 shows all the treatment groups, the number of papillomas and carcinomas per group, the number of tumor-bearing mice, and the average time (in weeks) to carcinoma development. The raw roofing asphalt and neat asphalt fumes induced carcinomas (local skin cancers) in three of 30 and 20 of 30 C_3H/HeJ mice, respectively. However, the heated asphalt with fumes allowed to escape did not induce any tumors. Fractions B and C induced carcinomas in 10 of 30 and 17 of 30 C_3H/HeJ mice, respectively, while fractions A, D, and E failed to induce any carcinomas when applied alone. All the combinations of the fractions induced tumors only if they included B or C. The A and D combination, the A and E combination, and the A, D, and E combination failed to induce any tumors. Furthermore, fractions A, D, and E failed to act as either tumor promoters or co-carcinogens. Fourteen of the 30 Sencar mice treated with the asphalt fume condensate developed carcinomas.

As noted in the preceding paragraph, only fractions B and C, whether applied alone or in combination, elicited tumor responses. Fractions B and C contained PACs that included PAHs, S-PACs, and O-PACs, such as alkylated aryl thiophenes, alkylated phenanthrenes, alkylated acetophenones, and alkylated dihydrofuranones. Fraction B contained most of the S-PACs, and only a few were carried over to fraction C. Fraction C contained a small amount of four-ring PACs (refer to Table 6–7). Sivak et al. [1989, 1997] stated the need for additional co-carcinogenesis and tumor-promotion experiments using a wider range of experimental variables, further chemical separation of fractions B and C, more short-term genotoxicity assays, and additional carcinogenicity assays to identify biologically active materials in the roofing asphalt fume condensates.

Table 6–7. Tumorigenic response in all treatment groups (adapted from Sivak et al. 1989; 1997)

Group number	Treatment	Asphalt dose, mg*	Total no. of tumors per group†		No. of tumor-bearing mice	Average time to carcinoma, weeks‡
			Papilloma	Carcinoma		
1	Raw asphalt	25	1	3	4	101
2	Heated asphalt (less fume)	25				
3	Heated asphalt (plus fume)	25				
4	Neat asphalt fume	25	12§	25§	21	74
5	Solvent control	0				
6	Fraction A	16				
7	Fraction B	2.3	2	10§	11	98
8	Fraction C	2.6	4	18§	20	86
9	Fraction D	2.3				
10	Fraction E	1.6				
11	Fractions A+B+C+D+E	24.8	30§	23§	25	75
12	Fractions A+B	18.3	10§	8§	13	97
13	Fractions A+C	18.6	12§	16§	15	90
14	Fractions A+D	18.3				
15	Fractions A+E	17.6				
16	Fractions B+C+D+E	8.8	9§	18§	19	81
17	Fractions A+B+C+D	23.2	17§	22§	24	80
18	Fractions A+B+C+E	22.5	26§	30§	27	77
19	Fractions B+C+D	7.2	15§	22§	21	86
20	Fractions B+C	4.9	12§	26§	26	73
21	Fractions A+C+D+E	22.5	5§	14§	17	89
22	Fractions A+B+D+E	22.2	5	7§	9	97
23	Fractions A+D+E	19.9	2			2
24	0.01% B(a)P	0**	1	28§	27	56
25	0.001% B(a)P	0**	2	3	5	103
26	0.0001% B(a)P	0**				
27	Fraction A+.01% B(a)P	16	7§	28§	24	70
28	Fraction A+.001% B(a)P	16	1	1	2	106
29	Fraction A+.0001%	16		1	1	106
30	Fraction D+.01% B(a)P	2.3	14§	34§	29	64
31	Fraction D+.001% B(a)P	2.3	2		2	
32	Fraction D+.0001%	2.3		1	1	106
33	Fraction E+.01% B(a)P	1.6	1	23§	24	61
34	Fraction E+.001% B(a)P	1.6		2	2	106
35	Fraction E+.0001%	1.6				
36	B(a)P then fraction A	16††				
37	B(a)P then fraction D	2.3††				
38	B(a)P then fraction E	1.6††				
39	B(a)P alone	0				
40	Sentinel mice‡‡	0				
41	Sencar fume	25	21§	18§	20	83
42	Sencar control	0				

*Asphalt, asphalt plus fume, or asphalt fume alone per 50 µL per application.
†Only histologically confirmed skin tumors are given.
‡Based on gross observation.
§There were significantly more tumors, earlier onset of tumors, or both in these groups compared to controls.
**5, 0.5, 0.05 µg B(a)P/50 µL application per group, respectively.
††Mice were initiated with a single application of 200 µg B(a)P/50 µL followed by twice-weekly applications of indicated fractions.
‡‡Five mice were sacrificed prior to the initiation of the study and after 6, 12, 18, and 24 months.

! Emmett et al. [1981]

In an earlier study, Emmett et al. [1981] examined the carcinogenic potential of a standard roofing asphalt (asphalt type not provided) dissolved in redistilled toluene at a 1:1 ratio by weight. Fifty milligrams of this solution was applied twice a week to the shaved intrascapular region of the back of 50 male C3H/HeJ mice. The vehicle control group of 50 mice received 50 mg of toluene twice a week, and the positive control group received 50 mg of 0.1% B(a)P in toluene twice a week. The dosing regimen continued for 80 weeks or until a skin lesion was diagnosed as a papilloma; when a papilloma progressed and was diagnosed grossly as a carcinoma, the tumor-bearing mouse was sacrificed and autopsied. Selected histopathological examination of the tumors confirmed the gross diagnosis.

No tumors were observed in the mice treated either with roofing asphalt or toluene. Twenty-six of the first group and 37 of the second group survived 60 or more weeks. Seventy-nine percent of the mice treated with B(a)P developed tumors. Chemical analysis by gas chromatography (with an electron capture detector) of the raw roofing asphalt indicated that B(a)P concentrations were below the level of detection, i.e., <0.0004%.

6.2.2 Asphalt-Based Paints

Robinson et al. [1984] examined the effects of four formulations of asphalt-based paints (labeled A through D) and three formulations of coal-tar-based paints (labeled E, F, and G) using female Sencar mice in mouse skin bioassays. All formulations except G had been used to prevent corrosion in drinking water distribution systems [Alben 1980; Miller et al. 1982]. The asphalt-based paints were formulations containing xylene, or xylene and mineral spirits with between 89% and 98% cutback asphalt.

The asphalt- and coal-tar-based paints were evaluated not only for their potential tumor-initiating ability, but also for their ability to function as complete carcinogens. Both the coal-tar and asphalt paint formulation groups initiated tumor development in mouse skin. The activity exerted by the coal-tar paints (data not presented) was approximately 100-fold greater than the activity exerted by the asphalt-based paints. Table 6–8 presents data demonstrating the tumor-initiating activity of the asphalt-based paints, provides gross tumor observations, and classifies tumors examined histologically [Robinson et al. 1984; Bull et al. 1985]. Animals receiving the initial 200-µL dose of the asphalt solutions showed a statistically significant increase ($P<0.05$; multiple-cell chi square analysis) in both the number of tumor-bearing animals and number of tumors per animal compared with animals treated with mineral spirits [Bull et al. 1985]. However, the tumor response induced by the coal-tar paints was greater than that induced by the asphalt-based paints, even though the volume of the coal-tar paints (0.2-20 µL) was less than the volume of the asphalt-based paints (200 µL).

Only coal-tar paint formulation E and asphalt paint D were analyzed for their ability to act as complete carcinogens. Two microliters of coal tar and 200 µL of asphalt D were applied to 40 female Sencar mice once a week for 30 weeks; the mice were sacrificed after 52 weeks. Under the experimental conditions provided, only coal-tar formulation E acted as a complete carcinogen. It induced the development of 171 tumors (papillomas) in 83%, or 33 of 40, of the mice; 10% of the mice had carcinomas (four animals had five carcinomas). Of the mice that had been treated with asphalt D, only one in 40 (3%) developed a tumor (papilloma), while three of 40 mice in the group treated with mineral spirits developed papillomas.

Table 6–8. Tumor-initiating activity of asphalt-based paints (adapted from Robinson et al. 1984 and Bull et al. 1985)

		Gross observations		No. of animals showing squamous cell abnormalities after histopathological examination at 52 weeks			
Asphalt-based paints	Dose, µL unless otherwise indicated*	Animals with tumors†	Total no. of tumors	No. examined/ initiated‡	Papillomas	Carci-nomas§	Tumors§
Asphalt A	200	18/40 (45)	25	36	4	2	6
	600	21/40 (53)	31	38	8	2	10
Asphalt B	200	17/40 (43)	23	31	5	0	5
	600	20/40 (50)	34	35	4	2	6
Asphalt C	200	19/40 (48)	28	31	4	5	8
	600	23/40 (58)	51	36	11	4	13
Asphalt D	200	21/40 (53)	33	33	9	6	9
	600	15/40 (38)	22	35	2	3	4
Mineral spirits	600	5/40 (13)	6	37	1	0	1
Acetone	200	6/30 (20)	6	23	4	0	4
B(a)P	10.0 µg	22/30 (73)	99	27	11	9	15
DMBA	2.65 µg	Not given	Not given	8	3	6	8

DMBA=Dimethyl benzanthraceme.
*The 200-µL dose was administered in one dose, while the 600-µL dose was administered as three weekly 200-µL doses. All animals were treated with 1 µg TPA in 200 µL of acetone three times weekly for 20 weeks beginning 2 weeks after the last initiating dose.
†Data represent cumulative tumor counts through 40 weeks. Number in parentheses indicates percentage.
‡Each treatment group except the DMBA-treatment group contained 40 female Sencar mice. Only 20 were in the DMBA-treatment group.
§The asphalt D group also had one animal with a fibrosarcoma and one with a basal cell carcinoma. Total number of animals having squamous cell papillomas and/or carcinomas does not agree with number of animals with squamous cell tumors because some animals had both types.

Chemical analyses of coal-tar formulations E, F, G, and H and asphalt-based paints A and D, which are also used to prevent corrosion in drinking water systems, were conducted using GC/MS. Results of the analyses indicated that PAH concentrations were high in coal-tar formulations E, F, G, and H, and very low in asphalt-based paints A and D. This observation is based on the five biologically active PAHs (chrysene, benz[a]anthracene, B(a)P, benzo[e]pyrene, and phenanthrene) found in coal-tar-based paints. The concentrations of these five compounds as a percentage of total PAHs were 44% in coal tar E, 42% in coal tar F, and 33% in coal tar G. Of these five PAHs, only trace amounts of phenanthrene (<0.01%) were found in both asphalt-based paints.

Robinson et al. [1984] concluded that the four asphalt-based paints tested contained chemicals capable of initiating tumors in mice, and that a number of these tumors were carcinomas. However, they were not found to be complete carcinogens.

6.3 Conclusions

The following conclusions concerning the adverse effects of asphalt fumes, raw asphalt, and asphalt-based paints are based on the results of the preceding experimental studies.

6.3.1 Asphalt Fumes

Asphalt fumes are comprised of complex chemical mixtures generated by the

volatilization of asphalt. In attempts to simulate occupational exposure to asphalt fumes in experimental animals, investigators developed several methods for generating asphalt fumes in the laboratory. It has yet to be determined whether these fumes are representative of the fumes to which workers are exposed during the manufacture and application of asphalt products (see section 3.4.3). Currently, available data generated using asphalt fume condensates (fumes were collected above the asphalt surface inside a hot-mix asphalt storage tank) are limited. A comparison of the biologic activity of these storage tank fumes and asphalt fume condensates generated in the laboratory at typical paving temperatures indicated that (1) fumes collected from a hot-mix asphalt storage tank *were not* mutagenic (Table 6–2) and (2) the laboratory-generated fumes *were* mutagenic [Reinke and Swanson 1993]. In other studies, paving and roofing asphalt fumes generated in the laboratory under a variety of conditions were also mutagenic [Machado et al. 1993; NTP 1990; AI 1990a; De Méo et al. 1996a]. These results indicate that asphalt fumes collected above the asphalt surface inside a hot-mix asphalt storage tank and laboratory-generated asphalt fume condensates may not induce similar biologic activity. In addition, fumes generated in the laboratory from two paving asphalts at 160 and 200 °C (320 and 392 °F) induced DNA-adduct formation *in vitro* and *in vivo* [De Méo et al. 1996a; Genevois et al. 1996].

Two studies examined the carcinogenic potential of roofing asphalt fume condensates generated in the laboratory at temperatures approximating those observed in typical and worst-case roofing operations [Niemeier et al. 1988; Sivak et al. 1989, 1997]. The data indicated that roofing asphalt fume condensates generated in the laboratory and applied dermally cause benign and malignant skin tumors in several strains of mice. Furthermore, additional data supportive of carcinogenicity demonstrated that these and similarly derived laboratory roofing asphalt fume condensates are mutagenic in the Ames *Salmonella* mutagenicity assay [NTP 1990; AI 1990a; Machado et al. 1993], induce micronuclei formation [Qian et al. 1996], and inhibit intracellular communication in mammalian cells [Toraason et al. 1991; Wey et al. 1992]. Differences in chemical composition and physical characteristics have been noted between roofing asphalt fumes collected in the field and those generated in the laboratory (see chapter 3, Kriech and Kurek [1993]). However, it is not known if these differences are responsible for the genotoxic and carcinogenic effects reported in the preceding experimental studies. Although no animal studies have examined the carcinogenic potential of asphalt fumes collected during roofing operations, the carcinogenic response using laboratory-generated asphalt fumes suggests a potential hazard to roofers.

Since no animal studies have examined the carcinogenic potential of either field or laboratory-generated samples of paving asphalt fume condensates, no definitive determination can be made about the carcinogenic potential of paving asphalt fume condensates in experimental animals. However, the positive mutagenic responses obtained using laboratory-generated paving asphalt fumes are a cause for concern and support the need for further research.

6.3.2 Raw Asphalt and Asphalt Paints

Conflicting results from two separate studies [Sivak et al. 1989,1997; Emmett et al. 1981] were obtained when raw roofing asphalts were applied to the skin of mice. The raw roofing asphalt used by Sivak et al. [1989, 1997] was weakly carcinogenic and caused malignant skin tumors, while the raw roofing asphalt used by Emmett et al. [1981] did not. Available data also indicate that several formulations of asphalt-based paints caused benign and malignant skin tumors in mice [Robinson et al. 1984; Bull et al. 1985]. However, these paints were not mutagenic in the Ames *Salmonella* mutagenicity assay either with or without

metabolic activation (S-9). Several other asphalt-based paints were positive in another type of genotoxicity assay, i.e., DNA-adduct formation, which is postulated to be one of the steps responsible for mutagenesis and carcinogenesis [Schoket et al. 1988a]. These asphalt-based paints also caused the formation of DNA adducts in the skin and lungs of treated mice and in fetal and adult human skin cultures [Schoket et al. 1988a,b].

The results are conflicting as to the carcinogenicity of raw roofing asphalt. One study reported a weak carcinogenic response [Sivak et al. 1989, 1997], while another study reported no carcinogenic response [Emmett et al. 1981]. However, the data indicate that the asphalt paint formulations used in the preceding studies [Robinson et al. 1984; Bull et al. 1985; Schoket et al. 1988a,b] are carcinogenic and exert some genotoxicity. Although no published data exist that examine the carcinogenic potential of asphalt-based paints in humans, NIOSH concludes that asphalt-based paints are potential occupational carcinogens.

7 Conclusions

The following conclusions concerning possible health effects caused by exposure to paving and roofing asphalts, raw asphalt, and asphalt-based paint were derived from an evaluation of data from studies in humans and experimental animals that have become available since the 1977 criteria document on asphalt [NIOSH 1977a].

In the 1977 criteria document, NIOSH established a REL of 5 mg/m^3 as a 15-min ceiling limit[4] for asphalt fumes measured as total particulates. The NIOSH REL was intended to protect workers against acute effects of exposure to asphalt fumes, including irritation of the serous membranes of the conjunctivae and the mucous membranes of the respiratory tract. In 1988, NIOSH (in testimony to the Department of Labor) recommended that asphalt fumes should be considered a potential occupational carcinogen [NIOSH 1988].

7.1 Cancer Issues

7.1.1 Characterization of Asphalt Fumes

An analysis of the chemical data indicates that paving and roofing asphalts are qualitatively and quantitatively different; therefore, the vapors and fumes from these asphalt products also may be different. The chemical composition of vapors and fumes from asphalt products is variable and depends on the crude petroleum source, type of asphalt, temperature and extent of mixing during the manufacturing process, and temperature and extent of mixing during laboratory generation or field operation, e.g., paving or roofing. For these reasons, it is possible that asphalt fumes generated in some laboratory experiments may be qualitatively and quantitatively different from fumes workers are exposed to in the workplace.

Although asphalt vapors and fumes are not well characterized, the determination of selected PAHs in asphalt products, vapors, and fumes is of interest. Many studies have been directed to identification of PAHs in asphalt fume samples. PAH identification by HPLC/fluorescence techniques are unreliable and results for asphalt fume samples are considered unreliable. For more information see section 3.5.3. However, asphalt fume PAH data obtained by GC/MS are considered reliable. The most meaningful of these studies used GC/MS for the analysis. Robinson et al. [1984] used GC/MS to analyze several asphalt-based paints for chrysene, benz[a]anthracene, B(a)P, benzo[e]pyrene, and phenanthrene; only trace amounts of phenanthrene (<0.01%) were detected. Also using GC/MS, several other investigators reported on the chemical analysis of paving and roofing asphalt fumes [Niemeier et al. 1988; Lunsford and Cooper 1989; Reinke and Swanson 1993; Hatjian et al. 1995a, 1997].

Low concentrations of carcinogenic PAHs have been detected in laboratory-generated asphalt fumes. Niemeier et al. [1988] measured low concentrations of several carcinogenic PAHs in roofing asphalt fumes generated at both 232 and 316 °C (450 and 601 °F); most were two and three ring. Lunsford and Cooper [1989] reported results similar to those of Niemeier et al. [1988]. Reinke and Swanson [1993] detected chrysene (0.02 µg/m^3) in paving asphalt fumes generated in the laboratory at 149 °C (300 °F). Lunsford and Cooper [1989] and Reinke and Swanson

[4]See footnote 1 in chapter 1.

[1993] also reported the presence of alkylated PACs in roofing and paving asphalt fumes, which is of concern because these PAHs are structurally similar to known carcinogens.

Few studies have been directed to the identification and measurement of PAHs in asphalt fumes generated at U.S. worksites. Reinke and Swanson [1993] collected paving asphalt fumes at 149 °C (300 °F) from a storage tank at a hot-mix plant, as well as laboratory-generated paving asphalt fumes at 149 and 316 °C (300 and 601 °F) (Table 3–6). Although they detected chrysene in the laboratory-generated asphalt fumes, they did not detect chrysene in the fumes collected from the storage tank. Two- and three-ring PAHs were found in the storage tank fumes, but not four-ring PAHs.

Hatjian et al. [1995a, 1997] reported on a GC/MS analysis for selected PAHs in asphalt paving and roofing fumes collected at several worksites. These investigators found that naphthalene accounted for 60% to 90% of the measured PAH exposure for the asphalt workers studied. Also, two- and three-ring PAHs accounted for 99% of the measured PAH exposure for asphalt pavers and 84% to 94% for asphalt roofers. B(a)P was detected in less than 6% of the personal-breathing-zone air samples of asphalt road pavers and manual laborers who had no occupational exposure to PAHs; B(a)P was detected in 28% and 25% of the personal-breathing zone samples from the two roofing groups R1 and R2, respectively.

In a NIOSH study, researchers detected PAHs in environmental samples from paving operations, but made no attempt to determine individual carcinogenic PAHs [Hanley and Miller 1996 a,b; Miller and Burr 1996 a,b, 1998; Kinnes et al. 1996; Almaguer et al. 1996].

While the data regarding the presence of carcinogens in asphalt fumes generated at U.S. worksites are limited, the occasional detection of B(a)P at worksites and the more frequent detection of B(a)P and other carcinogenic PACs in laboratory-generated asphalt fumes indicate that, under some conditions, known carcinogens are likely to be present. Moreover, asphalt fumes generated at high temperatures are more likely to generate carcinogenic PAHs and therefore are potentially more hazardous than fumes generated at lower temperatures.

7.1.2 Paving Asphalt

7.1.2.1 Short-Term Assays

Several laboratory-generated paving asphalt fume condensates were mutagenic in an Ames *Salmonella* mutagenicity assay [Machado et al. 1993; Reinke and Swanson 1993; De Méo et al. 1996a], while samples of field-generated fumes were nonmutagenic [Reinke and Swanson 1993]. Fumes generated in the laboratory from two paving asphalts induced *in vitro* and *in vivo* DNA-adduct formation [De Méo et al. 1996a; Genevois et al. 1996]. No other experimental animal studies have been conducted to determine the carcinogenic potential of either field- or laboratory-generated paving asphalt fume condensates.

7.1.2.2 Human Studies

! Epidemiology

Epidemiologic studies of pavers and highway workers exposed to asphalt were examined to determine the carcinogenic potential of paving asphalt fumes. An analysis of these studies indicates that although some studies reported an elevated risk for lung cancer among pavers [Hansen 1989a; Engholm et al. 1991; Partanen et al. 1997; Milham 1997], others did not [Maizlish et al. 1988; Bender et al. 1989]. Design limitations of both positive and negative studies restrict their interpretation. Partanen and Boffetta [1994] recently conducted a meta-analysis of studies involving pavers and highway workers exposed to asphalt. Their assessment did not find overall evidence for lung cancer risk among pavers. Overall, the epidemiologic evidence for an association be-

tween lung cancer and exposure to asphalt in paving is inconclusive at this time.

! Biomarkers

Reported urinary 1-hydroxypyrene data suggest that asphalt road pavers are at increased risk of exposure to PAHs in asphalt fume. Of the seven reports [Jongeneelen et al. 1988; Burgaz et al. 1992; Levin et al. 1995; Hatjian et al. 1995 a,b, 1997; Zhou 1997] in which the use of urinary 1-hydroxypyrene was described, three [Burgaz et al. 1992; Levin et al. 1995; Zhou 1997] noted statistically significant, increased postshift levels compared with reference populations, and one [Hatjian et al. 1997] noted a significant increase in one of two paving groups over a 3-day period. Four studies in which urinary thioethers were analyzed [Lafuente and Mallol 1987; Burgaz et al. 1988, 1992; Pasquini et al. 1989; Hatjian et al. 1995b] and two studies in which urinary D-glucaric acid was analyzed [Pasquini et al. 1989; Hatjian et al. 1995b] found no significant elevations in these biomarkers for asphalt-exposed workers relative to the reference populations. However, Burgaz et al. [1992] found significantly elevated urinary thioethers when only nonsmokers were evaluated.

Of three studies of possible genetic damage, none reported that exposure to asphalt fumes led to a substantial elevation of biomarkers of genetic damage. Pasquini et al. [1989] found significantly increased urinary mutagenicity, but only for nonsmokers; therefore, smoking status was a more important factor than was asphalt fume exposure. Fuchs et al. [1996] found no significant end-of-workweek elevation of DNA strand breaks. An observed elevation in sister chromatid exchange [Hatjian et al. 1995b] was confounded by smoking status and other exposures to PAHs.

7.1.3 Roofing Asphalt

7.1.3.1 Animal Studies

Data from experimental studies in animals and cultured mammalian cells indicate that laboratory-generated roofing asphalt fume condensates are genotoxic [NTP 1990; AI 1990a; Machado et al. 1993; Qian et al. 1996; Toraason et al. 1991; Wey et al. 1992] and cause skin tumors in mice when applied dermally [Niemeier et al. 1988; Sivak et al. 1989, 1997]. The absence of data to indicate that laboratory-generated roofing asphalt fume condensates are representative of field-generated fumes limits the usefulness of these data for determining the genotoxicity and potential carcinogenicity of field-generated roofing asphalt fume condensates.

7.1.3.2 Human Studies

! Epidemiology

Epidemiologic studies of roofers have generally demonstrated an excess of lung cancer in these workers [Hammond et al. 1976; Menck and Henderson 1976; Engholm et al. 1991; Hrubec et al. 1992; Pukkala 1995; Milham 1997; Zahm et al. 1989; Schoenberg et al. 1987; Morabia et al. 1992] (see sections 5.2.3 and 5.2.4). A meta-analysis of these studies conducted by Partanen and Boffetta [1994] also indicated an overall excess of lung cancer among roofers. However, it is unclear to what extent these cancers may be attributable to asphalt exposures during roofing operations, since in the past, roofers have been exposed to coal tar and asbestos, which are known human lung carcinogens. Although strong epidemiologic evidence exists of an association between lung cancer and working as a roofer, it is uncertain whether exposure to asphalt is related to this association.

! Biomarkers

Biomarker concentrations provided no clear insight about hazards from exposure to asphalt fumes. In a study by Hatjian et al. [1995a, 1997], one of two groups of roofers may have had elevated urinary 1-hydroxypyrene. Both groups had significantly elevated levels of

SCE but no elevated level of urinary thioethers or urinary D-glucaric acid. In two other studies, Herbert et al. [1990] found slightly elevated levels of DNA adducts, and Fuchs et al. [1996] found elevated levels of DNA strand breaks in end-of-workweek samples. However, in these studies, prior exposure to aged coal-tar pitch from roofing tear-off operations might have occurred. Currently, the available data indicate that roofers may be at risk for carcinogenic and genotoxic effects. However, it is uncertain whether exposure to roofing asphalt fumes is associated with this risk.

7.1.4 Exposures to Other Asphalt Products

7.1.4.1 Animal Studies—Raw Asphalt and Asphalt-Based Paint

The results are conflicting as to the carcinogenicity of raw roofing asphalt considering one experimental study reported a weak carcinogenic response in mice [Sivak et al. 1989, 1997], while another study reported no carcinogenic response [Emmett et al. 1981]. Similarly, the available data indicate that while not all asphalt-based paint formulations may exert genotoxicity and carcinogenicity, some are genotoxic [Robinson et al. 1984; Schoket et al. 1988 a,b] and carcinogenic [Robinson et al. 1984; Bull et al. 1985]. Although no published data exist that examine the carcinogenic potential of asphalt-based paints in humans, NIOSH concludes that asphalt-based paints are potential occupational carcinogens.

7.1.4.2 Human Studies

! *Epidemiology*

No epidemiologic studies of worker exposure to asphalt or asphalt-based paints have been reported.

! *Biomarkers*

Three reported studies provide no evidence for an exposure risk. In one study, a group of workers who loaded asphalt tankers and another group involved in the manufacture of asphalt showed no elevation of urinary 1-hydroxypyrene [Boogard and van Sittert 1995]. Burgaz et al. [1988] reported that workers in an asphalt hot-mix plant showed no elevation in urinary thioethers, and Fuchs et al. [1996] reported that asphalt painters showed no elevated levels of DNA strand breaks.

7.2 Noncarcinogenic Health Effects

A relatively small number of studies on the acute health effects associated with exposure to asphalt fumes have been published since the NIOSH criteria document on asphalt was published in 1977. Although the results from these studies are of limited value because of limitations in design and inadequate characterization of workers' exposures, certain acute health effects can reasonably be ascribed to asphalt fume exposure on the basis of the consistency of findings among studies.

Studies of workers exposed to asphalt fumes have repeatedly found irritation of the serous membranes of the conjunctivae (eye irritation)

and the mucous membranes of the upper respiratory tract (nasal and throat irritation). These health effects, which have been best described in asphalt road pavers [Norseth et al. 1991; Hanley and Miller 1996 a,b; Almaguer et al. 1996; Miller and Burr 1996 a,b, 1998; Kinnes et al. 1996; Sylvain and Miller 1996], typically appear to be mild and transitory. In studies of open-air paving, irritant symptoms occurred among workers exposed to asphalt fumes at geometric mean concentrations generally below 1.0 mg/m^3 total particulates and 0.3 mg/m^3 benzene- or carbon disulfide-soluble particulates calculated as a full-shift TWA [Almaguer et al. 1996; Hanley and Miller 1996 a,b; Miller and Burr 1996 a,b, 1998; Kinnes et al. 1996; Exxon 1997; Norseth et al. 1991]. Similar symptoms were reported in workers exposed to asphalt fumes during the manufacture of asphalt roofing shingles [Apol and Okawa 1977] and fluorescent lights [Chase et al. 1994], during insulation of cable [Zeglio 1950], and from a malfunctioning light fixture in an office [Tavris et al. 1984]. Exxon [1997] reported the occurrence of mild transitory symptoms of nasal and throat irritation, headaches, and coughing among workers employed in five segments of the asphalt industry (hot-mix plants, terminals, roofing application, roofing product manufacturing, and paving), although no significant dose-response associations were found between measured exposures and symptoms.

In addition to mucosal irritation, reports of skin irritation, pruritus, and occasionally rashes have been described in workers with occupational exposures to asphalt fumes [Hanley and Miller 1996 a,b; Almaguer et al. 1996; Miller and Burr 1996 a,b, 1998; Kinnes et al. 1996; Chase et al. 1994; Tavris et al. 1984; Schaffer et al. 1985; Waage and Nielson 1986]. In a recent survey of 50 roofers and 101 road pavers [Riala et al. 1998], work-induced skin irritation was reported in 44% of the roofers and 31% of the pavers. Dermatitis, predominately of the face, hands and arms, and lower extremities, occurred more often among road pavers (22%) than among roofers (15%). It was not determined if the dermatitis was irritant or allergic in nature, or if dermal photosensitization similar to that seen with coal tar, was occurring. Given the presence of confounding co-exposures (i.e., diesel fuel, coal tar, fiberglass) and environmental conditions (wind, heat and humidity, UV radiation), the extent to which asphalt fumes may be associated with these skin problems is unclear and should be studied further.

Symptoms of nausea, stomach pain, decreased appetite, headaches, and fatigue have been commonly reported among workers exposed to asphalt [Norseth et al. 1991; Chase et al. 1994; Tavris et al. 1984; Schaffer et al. 1985; Waage and Nielson 1986; Exxon 1997]. These nonspecific symptoms also require further investigation to clarify and establish the nature of any causal relationships with asphalt fume exposure.

Reports of acute lower respiratory tract symptoms (i.e., coughing, wheezing, shortness of breath) [Hanley and Miller 1996 a,b; Almaguer et al. 1996; Miller and Burr 1996 a,b, 1998; Kinnes et al. 1996; Sylvain and Miller 1996; Nyqvist 1978; Zeglio 1950]) and changes in pulmonary function (e.g., bronchial lability) [Sylvain and Miller 1996; Waage and Nielson 1986] among exposed workers are of particular concern. Results from recent studies [Exxon 1997; Hanley and Miller 1996 a,b; Almaguer et al. 1996; Miller and Burr 1996 a,b, 1998; Kinnes et al. 1996] indicated that some workers experienced lower respiratory tract symptoms (and in one case, significant changes in pulmonary function) during relatively low exposures to asphalt fumes, such as those found during open-air highway paving (0.075 to 0.48 mg/m^3 total particulates and 0.07 to 0.24 mg/m^3 benzene-soluble particulates, mean range exposures). Present data are insufficient to determine the causal relationship between asphalt fume exposures and lower respiratory symptoms or changes in pulmonary function; however, personal health factors (i.e., pre-existing asthma) or higher

exposures to asphalt fumes, such as those found during underground paving, might increase risks to workers [Norseth et al. 1991; Sylvain and Miller 1996].

Bronchitis, possibly related to chronic lower respiratory tract irritation, was reported among workers exposed to asphalt in several studies [Hansen 1991; Maizlish et al. 1988; Nyqvist 1978; Zeglio 1950; Baylor and Weaver 1968; Hasle et al. 1977]. Reports from Hueper and Payne [1960] and Simmers [1964] in IARC [1985] indicated that patchy distributions of emphysema, bronchiolar dilatation, pneumonitis, and severe localized bronchitis were observed in guinea pigs, rats, and mice chronically exposed to bitumens during inhalation studies. Findings of measurable decrements in pulmonary function and reports of bronchitis among asphalt-exposed workers suggest that chronic exposure to asphalt fumes may pose similar risks for humans. Unfortunately, the limited data preclude making any determinations concerning asphalt exposure-related chronic pulmonary morbidity at this time.

7.3 Overall Conclusions

In this hazard review, NIOSH has evaluated the scientific evidence concerning the potential health effects of occupational exposure to asphalt. On the basis of available data from studies in animals and humans, NIOSH concludes the following about the acute health effects of asphalt exposure:

- The findings of this hazard review continue to support the assessment of the 1977 NIOSH criteria document on asphalt fumes, which associated exposure to asphalt fumes from roofing, paving, and other uses of asphalt with irritation of the eyes, nose, and throat. Furthermore, in studies conducted since the publication of the 1977 criteria document, these symptoms have also been noted among workers exposed to asphalt fumes at geometric mean concentrations generally below 1 mg/m^3 total particulates and 0.3 mg/m^3 benzene-soluble or carbon disulfide-soluble particulates, calculated as a full-shift TWA. Recent studies also report evidence of acute lower respiratory tract symptoms among workers exposed to asphalt fumes. These data are currently being further analyzed to assess the relationship between lower respiratory tract symptoms and asphalt fume exposure. The available data on chronic pulmonary effects (such as bronchitis) are insufficient to support an association with asphalt fume exposures.

In 1988, NIOSH recommended to OSHA that asphalt fumes be considered a potential occupational carcinogen based on the results of an animal study in which laboratory-generated roofing asphalt fume condensates induced malignant skin tumors in mice. Since then, investigators have described differences in chemical composition, physical characteristics, and biological activity between asphalt fumes collected in the field and those generated in the laboratory. The relevance of these differences in ascribing adverse health effects in humans is unknown. Data from studies in humans indicate that some workers exposed to asphalt fumes are at an elevated risk of lung cancer; however, it is uncertain whether this excess is related to asphalt and/or other carcinogens in the workplace. Although carcinogenic PAHs

have been identified in asphalt fumes at various work sites, the measured concentrations and the frequency of their occurrence have been low. In addition, data from studies using HPLC analysis to identify PAHs were not considered because of the limitations of this method (e.g., compound resolution).

Based on evaluation of these data, the following conclusions were drawn regarding the carcinogenicity of asphalt under several conditions of use:

- Data regarding the potential carcinogenicity of paving asphalt fumes in humans are limited. Only one study identified B(a)P in field fumes, but it was unclear whether paving asphalt fumes were the source of the B(a)P. Chrysene has been identified only in laboratory-generated paving asphalt fumes. The available data from studies in humans have not provided consistent evidence of carcinogenic effects in workers exposed to asphalt fumes during paving operations. No animal studies have examined the carcinogenic potential of either field- or laboratory-generated samples of paving asphalt fume condensates. Although genotoxicity assays (but no carcinogenicity assays) using laboratory-generated and field-generated (storage tank paving asphalt) fumes have been conducted, only the laboratory-generated fumes were genotoxic. Therefore, NIOSH concludes that the collective data currently available from studies on paving asphalt provide insufficient evidence for an association between lung cancer and exposure to asphalt fumes during paving. The available data, however, do not preclude a carcinogenic risk from asphalt fumes generated during paving operations.

- The results from epidemiologic studies indicate that roofers are at an increased risk of lung cancer, but it is uncertain whether this increase can be attributed to asphalt and/or to other exposures such as coal tar or asbestos. Data from experimental studies in animals and cultured mammalian cells indicate that laboratory-generated roofing asphalt fume condensates are genotoxic and cause skin tumors in mice when applied dermally. Furthermore, a known carcinogen, B(a)P, was detected in field-generated roofing fumes. The collective health and exposure data provide sufficient evidence for NIOSH to conclude that roofing asphalt fumes are a potential occupational carcinogen.

- The available data indicate that although not all asphalt-based paint formulations may exert genotoxicity, some are genotoxic and carcinogenic in animals. No published data examine the carcinogenic potential of asphalt-based paints in humans, but NIOSH concludes that asphalt-based paints are potential occupational carcinogens.

7.4 Recommendations

In 1977, NIOSH recommended a REL of 5 mg/m^3 (15-min sample[5]) for asphalt fumes to minimize the risk of acute respiratory and eye irritation. Because there were insufficient data to ascribe the chemical fume components responsible for the irritant effects, the REL was based on a total particulate sample, which was determined to be an appropriate surrogate for exposure.

Recent epidemiologic studies of workers exposed to asphalt fumes indicate that irritant effects and acute respiratory symptoms (e.g., coughing, shortness of breath) are still occurring. In addition, exposure assessment studies demonstrate the complexity of

[5]See footnote 1 in chapter 1.

monitoring and evaluating the many components that may be present in asphalt, asphalt-based paints, and asphalt fumes and vapors. The complexity occurs because the quantities of these components vary depending on use conditions.

Current data are considered insufficient for quantifying the acute and chronic health risks of exposure to asphalt, asphalt-based paint, or asphalt fumes and vapors. However, data from at least two studies of acute effects are currently being evaluated to determine their usefulness in deriving an REL. Additional studies of workers exposed to asphalt fumes, vapors, and aerosols (e.g., during paving, roofing, and painting operations) are needed to better characterize exposures and to evaluate the risk of chronic disease, including lung cancer. Also required are experimental animal studies that use laboratory generation methods to produce fumes and vapors representative of asphalt roofing and paving operations. Until the results of these studies become available, NIOSH recommends minimizing possible acute or chronic health effects from exposure to asphalt, asphalt fumes and vapors, and asphalt-based paints by adhering to the current NIOSH REL of 5 mg/m^3 (15-min sample) and by implementing the following practices:

- Prevent dermal exposure.
- Keep the application temperature of heated asphalt as low as possible.
- Use engineering controls and good work practices at all work sites to minimize worker exposure to asphalt fumes and asphalt-based paint aerosols.
- Use appropriate respiratory protection (see Appendix C).

8 Research Needs

The research identified below represents the many facets of information needed to assess completely the health risks associated with dermal and respiratory exposures to asphalt and asphalt fumes and aerosols. The principal goal of the proposed research should be to answer the following questions. (1) What health effects are associated with exposure to asphalt? (2) What constituent(s) of asphalt fumes is responsible for acute and possibly chronic adverse health effects? (3) What constituent(s) of asphalt fumes should be used as the metric for monitoring workplace exposures? (4) What types of control technology (e.g., engineering, work-practice controls) are feasible to prevent worker exposure to asphalt? (5) What is an appropriate "health-based" exposure concentration of asphalt fumes (or its constituents) that will prevent acute and possibly chronic adverse health effects?

8.1 Human Studies

8.1.1 Cancer Studies

! Determine the availability of U.S. worker cohorts suitable for evaluating cancer risk from exposure to asphalt fumes during paving and roofing operations; the manufacture of asphalt products; and the use of asphalt-based paints, cutback asphalts, and asphalt emulsions. This study should be coordinated with the IARC study to facilitate comparison of results and perhaps integrate U.S. data with IARC data in a pooled analysis.

! If suitable populations and methods of study are found, design and conduct epidemiologic studies that include—

- Careful characterization of past and current worker exposures,
- Consideration of potential confounders, such as smoking, diet, medication, coal tar, and diesel exhaust, and
- Use of suitable biological monitoring methods.

! Perform quantitative risk estimates for any identified cancers.

! Conduct biomarker studies (see section 8.3).

! Conduct studies in which representative samples of exposure levels are evaluated in workers exposed to asphalt products, such as roofers, applicators, etc.

8.1.2 Noncancer Studies

! Using a population of U.S. workers exposed to asphalt during paving and roofing operations; the manufacture of asphalt products; and the use of asphalt-based paints, cutback asphalts, and asphalt emulsions—

- Test for the occurrence of acute effects,

- Characterize the nature of the exposures, and if possible,
- Establish exposure-response relationships.

! Develop and validate biomarkers of acute effects.

! Conduct a morbidity study of U.S. workers.

8.2 Animal Studies

! Generate asphalt fumes that are chemically representative of roofing and paving fumes in the field and test their carcinogenic potential with lifetime inhalation bioassays in rodents.

! Use these fumes to conduct limited skin-painting studies to determine tumorigenicity.

! Use these fumes in range-finding toxicity studies to characterize acute and subchronic effects and to develop novel techniques for biological monitoring and biomarkers.

! Use toxicity testing to determine dose-response relationships for both acute and chronic endpoints.

8.3 Biomarker Studies

! Study biomarkers associated with cancer risk as surrogates for cancer mortality or morbidity. Also study biomarkers that indicate exposure to known carcinogens (e.g., DNA adducts) and genotoxins.

! Develop an alternative to urinary 1-hydroxypyrene, which seems only weakly useful as a biomarker of exposure to asphalt fumes. Urinary biomarkers derived from these more abundant compounds, such as methylnaphthalenes, naphthalene, C3-alkylbenzothiophenes, C2-alkylbenzothiophenes, and methylphenanthrenes or methylanthracenes, might better represent exposure to asphalt fumes than urinary 1-hydroxypyrene.

! Develop biomarker methods that allow PAH exposure from asphalt to be differentiated from other sources of PAH exposure, such as coal tar, tobacco smoke, and ambient pollution.

8.4 Fume Characterization, Sampling, and Analytical Methods

! Conduct studies to identify and evaluate properties of crude source, asphalt type and use, and manufacturing processes that contribute to the qualitative and quantitative chemical composition of asphalt fumes and vapors.

! Continue to assess the differences between laboratory-generated and field-generated fumes and continue the search for the constituents of asphalt fumes that correlate closely with health effects. Once relevant compounds or compound classes are identified, develop analytical sampling and analysis methods.

! One approach to identifying a marker chemical or chemical class for carcinogenicity is to determine which components of the fumes used by Sivak et al. [1989, 1997] caused cancer in mouse skin-painting models. Continue research to identify these chemicals or to measure them without identification. Once they are identified, use these marker compounds to determine how the positive animal studies relate to the risks posed by workplace fumes.

- Develop or improve analytical sampling and analysis methods.

- Develop a method for determining the total three-ring and higher PAC content, since asphalt fumes contain many alkylated PACs.

- Develop an analysis method for determining individual PACs or other chemical analytes so that correlations can be made between air concentrations and biomarkers.

- Conduct additional studies of the genotoxicity and mutagenicity of fumes collected during paving, roofing, and manufacturing using modified Ames assays, SCE assays, and DNA adduct assays.

- Characterize the molecular weight distribution of the chemical classes and different chemical functional groups in the vapors and fumes to which workers are exposed. Collect vapors and fumes from worksites where paving asphalt, roofing asphalt, cutback asphalts, asphalt emulsions, or asphalt-based paints are being used. Use increasingly sophisticated techniques to characterize further the chemistry of these exposures.

- Develop and validate a dermal method for assessing asphalt fume exposure, because exposure to asphalt fumes may also occur by dermal contact.

- Characterize particle sizes of fumes. Because the hazard potential of chemicals present in inhaled air depends both on particle size and concentration, additional research is needed to define the size fractions present in asphalt fumes more clearly. This research would help determine the most effective sampling devices (inhalable, thoracic, or respirable) to use when evaluating asphalt fume exposures.

- Determine the effect of modifiers on asphalt fume composition.

8.5 Control Technology

8.5.1 Paving Operations

- Evaluate new formulations (e.g., introduction of additional polymers) to determine whether the constituents and amount of fumes generated change.

- Evaluate the effectiveness of the engineering controls recently incorporated into highway-class pavers. Identify the type and frequency of maintenance required to maintain optimum effectiveness of these controls.

- Determine the feasibility of engineering controls on commercial-class pavers (smaller than 16,000 pounds) to reduce asphalt fume exposures.

- Investigate the feasibility of incorporating engineering controls on nonpaver equipment used in laying hot-mix asphalt during road construction and repair. Such equipment includes surface grinders, materials-transfer vehicles, windrow machines, truck-mounted patching pavers, and crack sealers.

- Evaluate the design and use of receiving hoppers operated in conjunction with materials-transfer vehicles to determine whether changes can be made to reduce worker exposure to asphalt fumes.

8.5.2 Roofing Operations

- Continue to evaluate the various types of asphalt kettles and determine what types of engineering controls and design configurations provide optimal reductions in asphalt fume exposure. Investigate alternative methods for feeding asphalt into the kettle that will reduce the need for and frequency of lifting the kettle lid. The efficacy of new “low-fuming” asphalts should be evaluated.

- Investigate all sources of asphalt fume exposure during the application of hot asphalt to roofs and determine what types of engineering control methods and work practice changes can be instituted to reduce such exposures.

- Evaluate procedures and equipment used during roof tear-off to determine the most effective means of reducing worker exposures.

8.5.3 Waterproofing Operations

- Investigate methods (e.g., engineering controls, work practices) that are effective in minimizing airborne and dermal exposures during the application of asphalt waterproofing materials.

8.6 Training and Education Effectiveness

- Ascertain the effectiveness of current training and educational efforts to inform workers of the potential hazards associated with working with asphalt. Develop intervention strategies where warranted.

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Appendix A

Uses and Applications of Asphalt*

Agriculture

(See also Buildings, Hydraulics and Erosion Control, and Paving)
Cattle sprays
Damp-proofing and waterproofing buildings, structures
Disinfectants
Fence post coating
Mulches
Mulching paper
Paved barn floors, barnyards, feed platforms, etc.
Protecting tanks, vats, etc.
Protection for concrete structures
Tree paints
Water and moisture barriers (above and below ground)
Wind and water erosion control
Weather modification areas

Buildings

(See also Industrial, Paving)

Floors

Damp-proofing and waterproofing
Floor composition, tiles, coverings
Insulating fabrics, papers
Step treads

Roofing

Building papers
Built-up roof adhesives, felts, primes
Caulking compounds
Cement waterproofing compounds
Cleats for roofing
Glass wool compositions
Insulating fabrics, felts, papers
Joint filler compounds
Laminated roofing, shingles
Liquid roof coatings
Plastic cements
Shingles

Walls, Siding, Ceilings

Acoustical blocks, compositions, felts
Architectural decoration
Bricks
Brick siding
Building blocks, papers
Damp-proofing coatings, compositions
Insulating board, fabrics, felts, paper
Joint filler compounds
Masonry coatings
Plaster boards
Putty, asphalt
Siding compositions
Soundproofing
Stucco base
Wallboard

Miscellaneous

Air-drying paints, varnishes
Artificial lumber
Ebonized lumber
Insulating paints
Plumbing, pipes
Treated awnings

Hydraulics and Erosion Control

Canal linings, sealants
Catchment areas, basins
Dam grouts
Dam linings, protection
Dike protection
Ditch linings

*Adapted from AI [1990b].

Drainage gutters, structures
Embankment protection
Groins
Jetties
Levee protection
Mattresses for levee and bank protection
Membrane linings, waterproofing
Ore leaching pads
Reservoir linings
Revetments
Sand dune stabilization
Sewage lagoons, oxidation ponds
Swimming pools
Waste ponds
Water barriers

Industrial

Aluminum Foil Compositions Using Asphalt

Backed felts
Conduit insulation, lamination
Insulating boards
Paint compositions
Papers
Pipe wrapping
Roofing, shingles

Automotive

Acoustical compositions, felts
Brake linings
Clutch facings
Floor sound deadeners
Friction elements
Insulating felts
Panel boards
Shim strips
Tacking strips
Underseals

Electrical

Armature carbons, windings
Battery boxes, carbons
Electrical insulating compounds, papers, tapes, wire coatings
Junction box compound
Molded conduits

Composition Materials

Black grease
Buffing compounds
Cable splicing compound
Embalming
Etching compositions
Extenders, rubber, other
Explosives
Fire extinguisher compounds
Joint fillers
Lap cement
Lubricating grease
Pipe coatings, dips, joint seals
Plastic cements
Plasticizers
Preservatives
Printing inks
Well drilling fluid
Wooden cask liners

Impregnated, Treated Materials

Armored bituminized fabrics
Burlap impregnation
Canvas treating
Carpeting medium
Deck cloth impregnation
Fabrics, felts
Mildew prevention
Packing papers
Pipes and pipe wrapping
Planks
Rugs, asphalt base
Saw dust, cork, asphalt composition
Textiles, waterproofing
Tiles
Treated leather
Wrapping papers

Paints, Varnishes, etc.

Acid-proof enamels, mastics, varnishes
Acid-resistant coatings
Air-drying paints, varnishes
Anticorrosive and antifouling paints
Antioxidants and solvents
Base for solvent compositions
Baking and heat resistant enamels
Boat deck sealing compound

Lacquers, japans
Marine enamels

Miscellaneous

Belting
Blasting fuses
Briquette binders
Burial vaults
Casting molds
Clay articles
Clay pigeons
Depilatories
Expansion joints
Flower pots
Foundry cores
Friction tape
Gaskets
Imitation leather
Mirror backing
Phonograph records
Rubber, molded compositions
Shoe fillers, soles
Table tops

Paving

(See also Agriculture, Hydraulics and Erosion Control, Railroad, Recreation)
Airport runways, taxiways, aprons, etc.
Asphalt blocks
Brick fillers
Bridge deck surfacing
Crack fillers
Curbs, gutters, drainage ditches
Floors for buildings, warehouses, garages, etc.
Highways, roads, streets, shoulders
Parking lots, driveways
Portland concrete cement (PCC)
Underseals
Roof-deck parking
Sidewalk, footpaths
Soil stabilization

Railroads

Ballast treatment
Curve lubricant
Dust laying
Paved ballast, subballast
Paved crossings, freight yards, station platforms
Rail fillers
Railroad ties
Tie impregnating, stabilization

Recreation

Paved Surfaces

Dance pavilions
Drive-in movies
Gymnasiums, sports arenas
Playgrounds, school yards
Race tracks
Running tracks
Skating rinks
Swimming and wading pools
Tennis courts, handball courts

Appendix B

Summary of Occupational Exposure Data

Table B-1. Exposures to general asphalt refinery workers during asphalt refinery operations

Occupation	Source of exposure	Samples		Concentration (mg/m^3)			References
		Number	Type	Range	Geo. mean	Ari. mean	
General asphalt refinery workers	Vacuum distillation	4*	Total PAHs†	0.0047-0.016	0.0082	0.0095	NIOSH 1980
General asphalt refinery workers	Asphalt processing (6 refineries)	14*	Total PAHs†	0.0015-0.031	0.067	0.010	NIOSH 1983
General asphalt refinery workers	Deasphalting (1 refinery)	4*	Total PAHs†	0.0014-0.041	0.12	0.021	
Operator		2	Total PAHs†	0.0034-0.021	0.0084	0.012	
Assistant operator		3	Total PAHs†	0.0025-0.050	0.14	0.025	
Bitumen loaders	Outdoor bitumen refinery unit (bitumen at 170 to 210°C [338 to 410°F])	‡	Benzene solubles	0.3-1.0	§	§	Claydon et al. 1984
		‡	Total particulates	0.1-1.4	§	§	
Package fillers	Indoor area (bitumen at 220°C [428 °F])	2	Total particulates	0.20-0.32	0.25	0.23	Brandt et al. 1985
Bitumen loaders	Outdoor bitumen refinery unit (bitumen at 170 to 210°C [338 to 410°F])	4	Total particulates	0.7-2.9	§	1.4	
		4	Benzene solubles	<0.1-1.0	§	0.4	
		4	Total PAHs†	3.8-95	§	33	
Operator		11	Total particulates	<0.03-8.2	0.17	0.88	Hicks 1995
		11	Benzene solubles	0.034-1.9	0.14	0.42	
Assistant operator	Asphalt refinery/terminal (temperature of product at fume source ranged from 160 to 375°C [320 to 707°F])	9	Total particulates	<0.03-0.49	0.18	0.25	
		9	Benzene solubles	<0.066-0.32	0.11	0.13	
		4	Total particulates	0.17-0.26	0.22	0.23	
Lab technician		4	Benzene solubles	<0.062-0.43	0.15	0.21	
Loader/pumper operator		10	Total particulates	<0.026-14	0.29	1.6	
		10	Benzene solubles	0.038-13	0.29	1.6	
Maintenance/administration	Asphalt refinery/terminal (temperature of produce at fume source ranged from 160 to 375°C [320 to 707°F])	6	Total particulates	<0.032-0.77	0.17	0.29	
		6	Benzene solubles	0.011-0.22	0.076	0.1	
Utility worker		4	Total particulates	<0.024-0.062	0.039	0.043	
		4	Benzene solubles	<0.054-1.1	0.3	0.49	

Abbreviations: Ari. mean=arithmetic mean; Geo. mean=geometric mean.
*Area air samples. All remaining samples were personal-breathing-zone air samples.
†The sampling and analytical methods used for measuring PAH concentrations may vary between studies, and results may not be directly comparable.
‡Number of samples collected not available.
§Information not provided.
NOTE: Sampling periods ranged from 6 to 8 hours. Results shown are time-weighted averages.
NOTE: Solvents such as cyclohexane and acetonitrile have been used in place of benzene to measure the soluble fraction of a particular matrix. Because the extraction ability of these solvents varies, results are not comparable.

Table B-2. Exposures during road paving operations

Occupation	Source of exposure	Samples: Number	Samples: Type	Exposure concentration (mg/m³): Range	Geo. mean	Ari. mean	References
Screedman, rakerman, foreman, roller operator	Road paving using an asphalt mix	215	Total particulates	*	*	0.3-0.7	Byrd and Mikkelsen 1979
Paver operator		72	Total particulates	*	*	1.1	
Surface dressing worker	Hot asphalt cutback surface (cutback at 135°C [275 °F])	9	Total particulates	*	*	0.8	
Asphalt cement pavers		17	Total particulates	<0.1-1.2	*	0.3	Virtamo et al. 1979
		1	Cyclohexane solubles	*	0.3	0.3	
Course asphalt base worker		10	Total particulates	0.4-1.1	*	0.6	
		1	Cyclohexane solubles	*	0.6	0.6	
Mastic asphalt worker	Road paving (asphalt temperatures ranged from 145 to 195°C [293 to 383 °F])	16	Total particulates	0.2-4.2	*	1.7	
		11	Cyclohexane solubles	<0.1-2.7	*	1.2	
Unknown		18	Total particulates	0.1-3.0	*	1.0	
		6	Cyclohexane solubles	<0.1-1.3	*	0.7	
Surface dressing worker		4	Total particulates	0.8-2.5	*	1.7	
		4	Cyclohexane solubles	<0.1-0.4	*	0.2	
Oil gravel worker		8	Total particulates	0.1-1.4	*	0.6	
		2	Cyclohexane solubles	<0.1-0.5	0.22	0.3	
Paver operator	Road paving (asphalt mix temperature ranged from 130 to 170°C [266 to 338 °F])	14	Total particulates	0.15-5.6	0.80	1.3	Puzinauskas 1980
Rakerman		7	Total particulates	0.25-3.5	0.61	0.93	
Screedman		7	Total particulates	0.33-1.5	0.62	0.83	
Paver operator, screedman	Road paving	4†	Total PAHs‡	0.0043-0.013	0.0075	0.0083	Malajyandi et al. 1982
Screed operator	Sulfur-extended asphalt road construction	1	Total particulates	*	4.5	4.5	Daniels and Kramkoski 1983
Laborer		2	Total particulates	2.4-3.6	2.9	3	
Various paving workers	Hot bitumen cutback (temperature ranged from 120 to 158 °C [248 to 316 °F])	8	Total particulates	0.2-15	*	2.6	Brandt et al. 1985
		7	Benzene solubles	0.1-0.3	*	0.2	
Surface dressing worker		7	Total particulates	0.2-1.5	*	0.6	
		4	Benzene solubles	0.1-0.5	*	0.2	
Pavers (smokers)	Road paving with asphalt	4	Total PAHs‡	0.00075-0.0031	0.0015	0.0017	Pozzoli et al. 1985
Paver operator	Road paving using bitumens	5	Total particulates	*	*	0.58	Monarca et al. 1987
		5	Cyclohexane solubles	*	*	0.17	

See footnotes at end of table.

(Continued)

Table B-2 (Continued). Exposures during road paving operations

Occupation	Source of exposure	Samples		Exposure concentration (mg/m³)			Reference
		Number	Type	Range	Geo. mean	Ari. mean	
Screedman		12	Total particulates	*	*	0.83	
		12	Cyclohexane solubles	*	*	0.16	
Screedman 1 (smoker)	Asphalt paving crew in Florida (using AC-30 grade)	1	Total particulates	*	0.22	0.22	Gunkel 1989
		1	Benzene solubles	*	0.11	0.11	
Screedman 2 (nonsmoker)		1	Total particulates	*	0.22	0.22	
		1	Benzene solubles	*	0.13	0.13	
Luteman (nonsmoker)		1	Total particulates	*	0.16	0.16	
		1	Benzene solubles	*	*	ND	
Roller operator (nonsmoker)		1	Total particulates	*	0.48	0.48	
		1	Benzene solubles	*	*	ND	
Screedman 1 (smoker)	Asphalt paving crew in Maryland (using an asphalt cement, Grade 20)	1	Total particulates	*	0.14	0.14	
		1	Benzene solubles	*	0.20	0.20	
Screedman 2 (nonsmoker)		1	Total particulates	*	0.44	0.44	
		1	Benzene solubles	*	0.41	0.41	
Luteman (nonsmoker)		1	Total particulates	*	0.32	0.32	
		1	Benzene solubles	*	0.38	0.38	
Roller operator (nonsmoker)		1	Total particulates	*	0.33	0.33	
		1	Benzene solubles	*	0.098	0.098	
Paver operator (nonsmoker)		1	Total particulates	*	0.90	0.90	
		1	Benzene solubles	*	0.76	0.76	
Foreman	Paving operations temperature of product at fume source ranged from 123 to 152 °C [253 to 306 °F])	2	Total particulates	0.32-0.53	0.41	0.43	Hicks 1995
		2	Benzene solubles	0.12-0.22	0.16	0.17	
Laborer		7	Total particulates	0.22-0.43	0.34	0.34	
		7	Benzene solubles	0.049-0.5	0.15	0.2	
Screedman		10	Total particulates	0.21-0.86	0.48	0.54	
		10	Benzene solubles	0.03-4.4	0.28	0.73	
Luteman		5	Total particulates	0.12-0.35	0.21	0.23	
		5	Benzene solubles	<0.078-0.33	0.14	0.16	
Roller operator		5	Total particulates	0.11-0.82	0.3	0.4	
		5	Benzene solubles	<0.071-2.5	0.42	0.77	
Spreader operator (screedman)		8	Total particulates	0.14-0.86	0.46	0.54	
		8	Benzene solubles	0.038-1.2	0.33	0.47	

See footnotes at end of table.

(Continued)

Table B-2 (Continued). Exposures during road paving operations

Occupation	Source of exposure	Samples		Exposure concentration (mg/m^3)			Reference
		Number	Type	Range	Geo. mean	Ari. mean	
Foreman	Paving operation (temperature of product at fume source ranged from 135 to 151 °C [275 to 304 °F])	3	Total particulates	0.19-0.24	0.22	0.22	Zey 1992a
		1	Benzene solubles	*	*	ND	
Screedman		5	Total particulates	0.05-0.26	0.18	0.15	
		3	Benzene solubles	ND-0.20	0.015	0.047	
		2	Acetonitrile solubles	*	*	ND	
Raker		6	Total particulates	0.01-0.47	0.14	0.23	
		4	Benzene solubles	ND-0.35	0.008	0.2	
		2	Acetonitrile solubles	*	*	ND	
Roller		6	Total particulates	0.10-1.21	0.28	0.388	
		4	Benzene solubles	ND-0.12	0.0039	0.03	
		2	Acetonitrile solubles	*	*	ND	
Paver operator		3	Total particulates	0.09-0.29	0.19	0.21	
		2	Benzene solubles	0.08-0.1	0.089	0.09	
Foreman	Paving operations (temperature of product at fume source ranged from 135 to 158 °C [275 to 316 °F])	3	Total particulates	ND-0.44	0.047	0.22	Zey 1992b
		2	Benzene solubles	*	*	ND	
		1	Acetonitrile solubles	*	*	ND	
Screedman		6	Total particulates	0.20-1.14	0.45	0.54	
		4	Benzene solubles	0.07-0.13	0.09	0.098	
		2	Acetonitrile solubles	ND-0.13	0.0089	0.015	
Raker/Laborer		6	Total particulates	0.24-4.17	0.69	1.2	
		3	Benzene solubles	0.08-0.18	0.14	0.14	
		3	Acetonitrile solubles	*	*	ND	
Roller operator		4	Total particulates	0.32-0.84	0.46	0.49	
		3	Benzene solubles	0.07-0.07	0.07	0.07	
		1	Acetonitrile solubles	*	*	ND	
Paver operator		3	Total particulates	0.24-0.74	0.50	0.50	
		2	Benzene solubles	0.07-0.18	0.11	0.13	
		1	Acetonitrile solubles	*	*	ND	

See footnotes at end of table.

(Continued)

Table B-2 (Continued). Exposures during road paving operations

Occupation	Source of exposure	Samples		Exposure concentration (mg/m^3)			Reference
		Number	Type	Range	Geo. mean	Ari. mean	
Screedman/supervisor	Paving operations (temperature of product at fume source ranged from 123 to 198°C [253 to 388 °F])	2	Total particulates	0.07-0.19	0.12	0.13	Zey 1992c
		1	Benzene solubles	*	*	ND	
		1	Acetonitrile solubles	*	*	ND	
Laborer/supervisor		1	Total particulates	*	0.15	0.15	
		1	Benzene solubles	*	*	ND	
Raker/laborer		8	Total particulates	0.09-0.48	0.22	0.28	
		5	Benzene solubles	0.07	0.004	0.026	
		3	Acetonitrile solubles	0.05	0.031	0.033	
Roller		4	Total particulates	0.06-0.16	0.11	0.11	
		3	Benzene solubles	*	*	ND	
		1	Acetonitrile solubles	*	*	ND	
Paver operator		3	Total particulates	0.13-0.21	0.16	0.17	
		2	Benezene solubles	0.07-0.07	0.07	0.07	
		1	Acetonitrile solubles	*	0.05	0.05	

Abbreviations: Ari.mean=arithmetic mean; Geo. mean=geometric mean; ND=not detected.
*Information not provided.
†Area air samples. All remaining samples were personal-breathing-zone air samples.
‡The sampling and analytical methods used for measuring PAH concentrations may vary between studies, and results may not be directly comparable.
NOTE: Sampling periods ranged from 6 to 8 hours. Results shown are time-weighted averages.
NOTE: Solvents such as cyclohexane and acetonitrile have been used in place of benzene to measure the soluble fraction of a particular matrix. Because the extraction ability of these solvents varies, results are not comparable.

Table B-3. Exposures during hot-mix asphalt preparation

Occupation	Source of exposure	Samples		Exposure concentration (mg/m^3)			References
		Number	Type	Range	Geo. mean	Ari. mean	
Not specified	Preparation of hot-mix asphalt	8	Total particulates	0.5-7.2	*	*	Puzinauskas and Corbett 1975
		8†	Benzene solubles	0.2-5.4	*	*	
Man in control cabin and operator on hot-mix asphalt storage bins	Asphalt plants (2)	‡	Total particulates	ND-1.7	*	0.7	Byrd and Mikkelsen 1979
Loader/operator	Hot-mix asphalt preparation (temperature of product at fume source ranged from 129 to 170°C [264 to 338 °F])	9	Total particulates	0.16-6.0	1.2	1.8	Hicks 1995
		9	Benzene solubles	0.033-1.7	0.16	0.33	
Groundman		9	Total particulates	0.17-1.4	0.53	0.61	
		9	Benzene solubles	0.062-0.43	0.19	0.23	
Plant operator		6	Total particulates	0.1-1.5	0.58	0.79	
		6	Benzene solubles	0.026-0.28	0.13	0.16	
Laborer		5	Total particulates	0.2-15	1.5	4.0	
		5	Benzene solubles	0.011-0.22	0.069	0.1	
Foreman/supervisor		4	Total particulates	0.27-1.1	0.53	0.61	
		4	Benzene solubles	<0.065-1.4	0.28	0.5	

Abbreviations: Ari. mean=arithmetic mean; Geo. mean=geometric mean; ND=not detected.
*Information not provided.
†Area air samples. All remaining samples were personal-breathing-zone air samples.
‡Number of samples collected not available.
NOTE: Sampling periods ranged from 6 to 8 hours. Results shown are time-weighted averages.
NOTE: Solvents such as cyclohexane and acetonitrile have been used in place of benzene to measure the soluble fraction of a particular matrix. Because the extraction ability of these solvents varies, results are not comparable.

Table B-4. Exposures during roofing operations

Occupation	Source of exposure	Samples		Exposure concentration (mg/m^3)			References
		Number	Type	Range	Geo. mean	Ari. mean	
Hot asphalt machine operator/carrier	Roofing operation	6	Cyclohexane solubles	<0.02-0.19	0.050	0.082	Hervin and Emmett 1976
Kettleman, felt layer	Asphalt heating and mopping	1	Benzene solubles	*	0.75 (f) 0.04 (s)	0.75 (f) <0.04 (s)	Brown and Fajen 1977a
Felt layer		2	Benzene solubles	0.35-1.3 (f) <0.04-0.23 (s)	0.67 (f) 0.096 (s)	0.825 (f) 0.14 (s)	
Foreman, mopper		1	Benzene solubles	*	4.9 (f) 0.12 (s)	0.49 (f) 0.12 (s)	
Felt tacker		1	Benzene solubles	*	1.1 (f) 0.16 (s)	1.1 (f) 0.16 (s)	
Kettleman		4†	Benzene solubles	170-420 (f) <0.17-1.1 (s)	245 (f) 0.34 (s)	260.(f) 0.47 (s)	
Felt layer	Asphalt heating and mopping	2	Benzene solubles	0.08-0.78 (f) <0.04-0.35 (s)	0.25 (f) 0.12 (s)	0.43 (f) 0.20 (s)	Brown and Fajen 1977b
Foreman		1	Benzene solubles	*	2.1 (f) 0.15 (s)	2.1 (f) 0.15 (s)	
Felt machine operator, mopper	Application of hot roofing asphalt	2	Benzene solubles	0.17-2.5 (f) 0.22-0.47 (s)	0.65 (f) 0.32 (s)	1.3 (f) 0.35 (s)	Brown and Fajen 1977c
Hot asphalt carrier		1	Benzene solubles	*	0.57 (f) 0.16 (s)	0.57 (f) 0.16 (s)	
Mopper		1	Benzene solubles	*	0.38 (f) <0.04 (s)	0.38 (f) <0.04 (s)	
Felt layer		1	Benzene solubles	*	0.17 (f) 0.08 (s)	0.17(f) 0.08 (s)	
Asphalt tank operator		1	Benzene solubles	*	<0.11 (f & s)	<0.11 (f & s)	
Kettleman	Type III roofing asphalt	6	Total particulates	1.3-12	2.6	3.7	Merz and Weisgerber 1977
		6	Benzene solubles	0.91-6.9	1.8	2.3	
Mopper		6	Total particulates	1.1-8.4	2.9	3.6	
		6	Benzene solubles	0.68-6.5	2.1	2.6	
Paperman		6	Total particulates	0.35-3.3	1.2	1.5	
		6	Benzene solubles	0.50-2.4	0.8	1.1	

See footnotes at end of table.

(Continued)

Table B-4 (Continued). Exposures during roofing operations

Occupation	Source of exposure	Samples		Exposure concentration (mg/m^3)			References
		Number	Type	Range	Geo. mean	Ari. mean	
Kettleman	Type III roofing asphalt a) low volatility	3	Total particulates	1.28-2.5	1.7	1.7	Puzinauskas 1979
		3	Benzene solubles	0.91-1.9	1.3	1.4	
Mopper		3	Total particulates	1.7-8.4	3.2	4.2	
		3	Benzene solubles	6.6	2.5	3.1	
Paperman		3	Total particulates	0.35-2.0	0.77	1.0	
		3	Benzene solubles	0.25-1.7	0.60	0.81	
Kettleman	b) high volatility	3	Total particulates	1.8-13	4.0	5.7	
		3	Benzene solubles	1.4-6.9	2.5	3.3	
Mopper		3	Total particulates	1.1-4.5	2.6	3.1	
		3	Benzene solubles	0.68-3.4	1.7	2.0	
Paperman		3	Total particulates	0.88-3.3	1.8	2.1	
		3	Benzene solubles	0.57-2.4	1.2	1.4	
Mopper	Spreading hot bitumen	8	Total particulates	0.2-3.4	*	*	Priha et al. 1980
		8	Cyclohexane solubles	0.29-2.9	*	*	
Mopper and kettleman	Asphalt roofing	6*	Total PAHs‡	0.015-0.11	0.023	0.033	Malaiyandi et al. 1982
Kettleman	Asphalt heating and mopping	1	Cyclohexane solubles	*	0.28	0.28	Tharr 1982a
Mopper		2	Cyclohexane solubles	0.16-0.17	0.17	0.17	
Laborer	Tear-off operations of an old asphalt roof (petroleum pitch)	9	Cyclohexane solubles	0.09-2.3	0.22	0.47	Tharr 1982b
Paper roller	Asphalt roofing	2	Benzene solubles	1.0-1.1	1.1	1.1	Reed 1983
Mopper		1	Benzene solubles	*	0.9	0.9	
Kettleman		2	Benzene solubles	1.2-1.2	1.2	1.2	
Kettleman	Kettle emissions and bitumen spreading	2	Total particulates	4.1-6.4	5.1	5.3	Brandt et al. 1985
		2	Benzene solubles	3.5-5.4	4.3	4.5	
		7	Total particulates	0.5-1.7	*	1.3	
		7	Benzene solubles	0.2-1.1	*	0.7	

See footnotes at end of table.

(Continued)

Table B-4 (Continued). Exposures during roofing operations

Occupation	Source of exposure	Samples		Exposure concentration (mg/m^3)			References
		Number	Type	Range	Geo. mean	Ari. mean	
Roof-level workers (laborer, mopper, carrier, etc.)	Application of an asphalt built-up roof	16	Acetonitrile solubles	0.04-2.7	0.16	0.34	Carson 1986
Ground-level workers (kettleman)		3	Acetonitrile solubles	0.04-0.83	0.27	0.49	
Laborer	Tear-off operations of an old asphalt roof (petroleum pitch)	6	Total particulates	0.76-2.8	1.72	1.9	Reed 1986
		6	Benzene solubles	ND-0.32	0.0096	0.11	
Various (kettleman, laborer, etc.)	Application of asphalt roof	28	Benzene solubles	ND-1.4	0.18	0.39	Zey et al. 1988
Various (kettleman, laborer, etc.)	Application of Koppers roof	10	Benzene solubles	ND-1.9	0.013	1.9	
Roofer	Roofing operation (temperature of product at fume source from 163 to 316 °C [325 to 600 °F])	12	Total particulates	0.04-2.2	0.36	0.58	Hicks 1995
		12	Benzene solubles	0.011-1.7	0.19	0.45	
Laborer		5	Total particulates	0.21-0.91	0.38	0.47	
		5	Benzene solubles	0.17-0.62	0.3	0.34	
Mechanic		7	Total particulates	0.24-1.2	0.54	0.65	
		7	Benzene solubles	<0.078-1.8	0.26	0.49	
Felt machine operator		7	Total particulates	0.57-2.5	1.0	1.3	
		7	Benzene solubles	0.046-2.4	0.21	0.53	
Kettleman		4	Total particulates	0.36-1.6	1.0	1.2	
		4	Benzene solubles	0.14-1.2	0.67	0.89	
Mopper		3	Total particulates	0.27-1.2	0.51	0.63	
		3	Benzene solubles	<0.085-0.75	0.21	0.33	

Abbreviations: Ari.mean=arithmetic mean; f=filter; Geo. mean=geometric mean; ND=not detected; s=sorbent tube.
* Information not provided.
† Area air samples. All remaining samples were personal-breathing-zone air samples.
‡Sampling and analytical methods used for measuring PAH concentrations may vary among studies, and these results may not be directly comparable.
NOTE: Sampling periods ranged from 6 to 8 hours. Results shown are time-weighted averages.
NOTE: Solvents such as cyclohexane and acetonitrile have been used in place of benzene to measure the soluble fraction of a particular matrix. Because the extraction ability of these solvents varies, results are not comparable.

Table B-5. Exposures during manufacturing of roofing products

Occupation	Source of exposure	Samples		Exposure concentration (mg/m^3)			References
		Number	Type	Range	Geo. mean	Ari. mean	
Saturator operator	Production of asphalt shingles and rolled roofing materials	3	Total particulates	2.2-7.3	4.9	5.6	Apol and Okawa 1977
		2	Cyclohexane solubles	0.81-2.6	1.4	1.7	
Coater operator		2	Total particulates	14-15	14	14	
		2	Cyclohexane solubles	0.87-6.8	2.4	3.9	
Coater trainee		2	Total particulates	11-15	13	13	
		2	Cyclohexane solubles	0.30-2.3	0.84	1.3	
Press operator		2	Total particulates	6.1-32	14	19	
		2	Cyclohexane solubles	1.4-1.5	1.4	1.42	
Seal down operator		2	Total particulates	0.99-1.4	1.2	1.21	
		2	Cyclohexane solubles	0.43-1.2	0.71	0.81	
Foreman		2	Total particulates	5.3-6.3	5.8	5.8	
		2	Cyclohexane solubles	0.47-0.53	0.50	0.5	
Hallmark operation worker		7	Total particulates	0.96-2.16	1.6	1.6	
		5	Cyclohexane solubles	0.23-0.76	0.46	0.50	
Hallmark worker	Production of asphalt shingles	8	Total particulates	0.38-5.2	1.0	1.4	Okawa and Apol 1977
		8	Cyclohexane solubles	0.24-3.1	0.76	1.05	
Saturator/Coater		6	Total particulates	5.3-29	8.03	12	
		6	Cyclohexane solubles	0.66-1.3	0.88	0.91	
Saturator operator	Production of asphalt shingles and rolled roofing materials	1	Total particulates	*	2.4	2.4	Apol and Okawa 1978
		1	Cyclohexane solubles	*	0.76	0.76	
Coater		2	Total particulates	12-16	14	14	
		2	Cyclohexane solubles	0.21-1.1	0.47	0.64	
Wrapping machine operator		1	Total particulates	*	1.4	1.40	
		1	Cyclohexane solubles	*	0.77	0.77	
Lead man		1	Total particulates	*	5.3	5.29	
		1	Cyclohexane solubles	*	0.76	0.76	
Presser		2	Total particulates	8.3-22	13	15	
		2	Cyclohexane solubles	0.30-0.54	0.402	0.42	
Coater	Production of asphalt shingles and rolled roofing materials	2	Total particulates	4.2-5.1	4.6	4.7	Okawa and Apol 1978a
		2	Cyclohexane solubles	3.3-3.9	3.6	3.6	
Cooling section operator		2	Total particulates	0.3-1.0	0.55	0.65	
		2	Cyclohexane solubles	0.7-1.6	1.06	1.2	
Machine tender		2	Total particulates	0.4-2.8	1.06	1.6	
		2	Cyclohexane solubles	0.6-1.8	1.039	1.2	

See footnotes at end of table.

(Continued)

Table B-5 (Continued). Exposures during manufacturing of roofing products

Occupation	Source of exposure	Samples		Exposure concentration (mg/m^3)			References
		Number	Type	Range	Geo. mean	Ari. mean	
Saturator operator	Production of asphalt shingles and rolled roofing materials	1	Total particulates	*	1.0	1.0	Okawa and Apol 1978b
		1	Cyclohexane solubles	*	2.1	2.1	
Coaterman		1	Total particulates	*	2.1	2.1	
		1	Cyclohexane solubles	*	0.22	0.22	
Slateman		1	Total particulates	*	3.4	3.4	
		1	Cyclohexane solubles	*	0.21	0.21	
Paper production workers (smokers)	Asphalted paper production	4	Total PAHs†	0.0048-0.036	0.009	0.014	Pozzoli et al. 1985
Paper production workers (nonsmokers)		2	Total PAHs†	0.0034-0.311	0.010	0.017	
Loader/asphalt handler	Roofing manufacturing (temperature of product at fume source from 149 to 377 °C [300 to 710 °F])	6	Total particulates	0.068-0.94	0.51	0.66	Hicks 1995
		6	Benzene solubles	0.041-0.71	0.31	0.41	
Slate blend operator		4	Total particulates	1.1-13	3.9	5.6	
		4	Benzene solubles	0.013-0.94	0.077	0.27	
Coater operator		8	Total particulates	0.42-2.5	1.0	1.3	
		8	Benzene solubles	0.049-1.6	0.2	0.37	
Machine operator	Roofing manufacturing (temperature of product at fume source from 149 to 377 °C [300 to 710°F])	8	Total particulates	0.8-4.4	1.6	1.9	
		8	Benzene solubles	<0.071-3.6	0.55	1.5	
Press operator/cooling operator		7	Total particulates	0.55-11	2.6	4.8	
		7	Benzene solubles	<0.085-3.7	0.32	1.0	
Supervisor		1	Total particulates	*	1.4	1.4	
		1	Benzene solubles	*	0.32	0.32	

Abbreviations: Ari. mean=arithmetic mean; Geo. mean=geometric mean.
*Information not provided.
†The sampling and analytical methods used for measuring PAH concentrations may vary between studies, and results may not be directly comparable.
NOTE: Sampling periods ranged from 6 to 8 hours. Results shown are time-weighted averages.
NOTE: Solvents such as cyclohexane and acetonitrile have been used in place of benzene to measure the soluble fraction of a particular matrix. Because the extraction ability of these solvents varies, results are not comparable.

Table B-6. Exposures during flooring and waterproofing operations

Occupation	Source of exposure	Samples		Exposure concentration (mg/m³)			References
		Number	Type	Range	Geo. mean	Ari. mean	
Brusher of felt interply adhesive	Waterproofing of basements, kitchens, bathrooms, and corridors	9	Total particulates	1.1-32	*	10.4	Ahmen et al. 1977
		5	Cyclohexane solubles	0.8-28	*	7.7	
Mixing operator (other possible exposures)	Manufacturer of vinyl asbestos and asphalt asbestos floor covering	6	Benzene solubles	0.08-0.66	0.30	0.37	Belanger and Elesh 1979
Spreader of hot bitumen and felt seaming	Waterproofing of kitchens, bathrooms, and underground spaces	15	Total particulates	1.9-42	*	18	Priha et al. 1980
		†	Cyclohexane solubles	0.3-39	*	*	
Spreader of hot bitumen and felt seaming	Waterproofing of basements	6	Total particulates	1.6-13	*	6.1	
		†	Cyclohexane solubles	1.6-11	*	5.6	
Spreader	Flooring-industrial asphalt mixture of bitumen, lime, sand, gravel, and coloring agent	10	Carbon tetrachloride extracts	8.2-237	*	8.3	Haydon et al. 1984
Tipper		10	Carbon tetrachloride extracts	3.1-260	*	7.3	
Float finisher		8	Carbon tetrachloride extracts	1.7-214	*	6.3	
Bucket carrier		4	Carbon tetrachloride extracts	0.5-5.5	*	*	
Bucket filler		2	Carbon tetrachloride extracts	3.1-4.2	3.6	*	
Troweler	Bitumen mastic on gymnasium floor	2	Total particulates	5.5-12	8.1	8.7	
Troweler		2‡	Total particulates	1.2-6.3	2.7	3.8	
		4‡	Total particulates	1.7-30	*	*	
Troweler	Hand laying mastic containing 11% bitumen at 265-280°C [509-536 °F]	1	Benzene solubles	*	10	10	
Tipper		1	Benzene solubles	*	7.1	7.1	
Carrier		1	Benzene solubles	*	11	11	
Kettleman		1	Benzene solubles	*	2.9	2.9	
Bucket carrier, pourer (indoors)	Mastic asphalt (265 to 280°C [509 to 536 °F])	5	Total particulates	11-18	*	13.3	Brandt et al. 1985
		5	Benzene solubles	6.0-14	*	8.8	
Kettleman (outside)		4	Total particulates	2.9-7.7	*	4.4	
		4	Benzene solubles	2.0-5.0	*	*	
Troweler		3	Total particulates	11-18	*	14	
		3	Benzene solubles	7.3-13	*	10	

Abbreviations: Ari. mean=arithmetic mean; Geo. mean=geometric mean.

*Information not provided.

†Number of samples collected not available.

‡Area air samples. All remaining samples were personal-breathing-zone air samples.

NOTE: Sampling periods ranged from 6 to 8 hours. Results shown are time-weighted averages.

NOTE: Solvents such as cyclohexane and acetonitrile have been used in place of benzene to measure the soluble fraction of a particular matrix. Because the extraction ability of these solvents varies, results are not comparable.

Appendix C

Respirators

At most worksites where hot asphalt or asphalt-based paints are used, concentrations of asphalt fumes or aerosols are generally below the NIOSH REL of 5 mg/m^3. However, constantly changing environmental and worksite conditions may result in fluctuating airborne concentrations of asphalt fumes and asphalt-based paint aerosols. Such fluctuations may result in exposures exceeding the NIOSH REL and warrant the use of respiratory protection. If respirators are required at the worksite, the employer is responsible for ensuring that respirators are NIOSH-approved and that all OSHA regulations pertaining to the implementation of a respirator program are followed. Important elements of these OSHA regulations [29 CFR 1910.134] are—

- An evaluation or the worker's ability to perform the work while wearing a respirator,
- Regular training of workers,
- Periodic environmental monitoring,
- Respirator fit-testing, maintenance, inspection, cleaning, and storage,
- Periodic changes of cartridges, and
- Cartridge testing for service life.

No NIOSH-approved respirator filter cartridge or canister exists specifically for asphalt fumes or aerosols. But the respirators listed below will reduce exposure.

- Any half-facepiece, air-purifying respirator equipped with a combination R100 or P100 filter and an organic vapor cartridge, or
- Any powered, air-purifying respirator with a hood, helmet, or loose-fitting facepiece equipped with a combination HEPA and organic vapor cartridge.

The appropriate respirator filters are R100, P100, or HEPA as listed under 42 CFR 84 [NIOSH 1996]. The appropriate organic vapor cartridge or canister should contain a charcoal sorbent. This type of protection may also be used when there is a potential exposure to dusts containing coal tar or asbestos.

A comprehensive assessment of workplace exposures should always be performed to ensure that the proper respiratory protection is used. Other types of respirators can provide a higher level of protection and may be required under certain conditions (e.g., work in confined spaces) [NIOSH 1987].