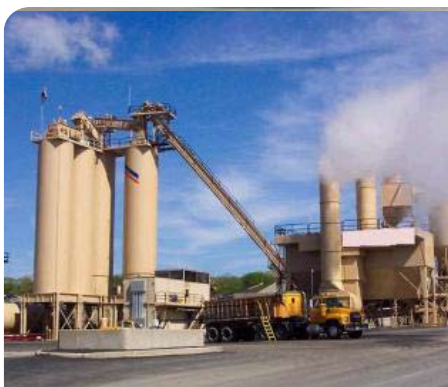
Asphalt Plants

FactPack – P131



Center for Health, Environment & Justice
P.O. Box 6806, Falls Church, VA 22040-6806
703-237-2249 chej@chej.org www.chej.org

Asphalt Plants

Center for Health, Environment & Justice
FactPack - PUB 131

August 2016

P.O. Box 6806 Falls Church, VA 22040-6806 703-237-2249 chej@chej.org www.chej.org

Center for Health, Environment & Justice
P.O. Box 6806 • Falls Church, VA 22040 • Phone: 703.237.2249 • Fax: 703.237.8389 • www.chej.org

Mentoring a Movement

Empowering People

Preventing Harm

About the Center for Health, Environment & Justice

CHEJ mentors a movement building healthier communities by empowering people to prevent harm caused by chemical and toxic threats. We accomplish our work through programs focusing on different types of environmental health threats. CHEJ also works with communities to empower groups by providing the tools, direction, and encouragement they need to advocate for human health, to prevent harm and to work towards environmental integrity.

Following her successful effort to prevent further harm for families living in contaminated Love Canal, Lois Gibbs founded CHEJ in 1981 to continue the journey. To date, CHEJ has assisted over 15,000 groups nationwide. Details on CHEJ's efforts to help families and communities prevent harm can be found on www.chej.org.

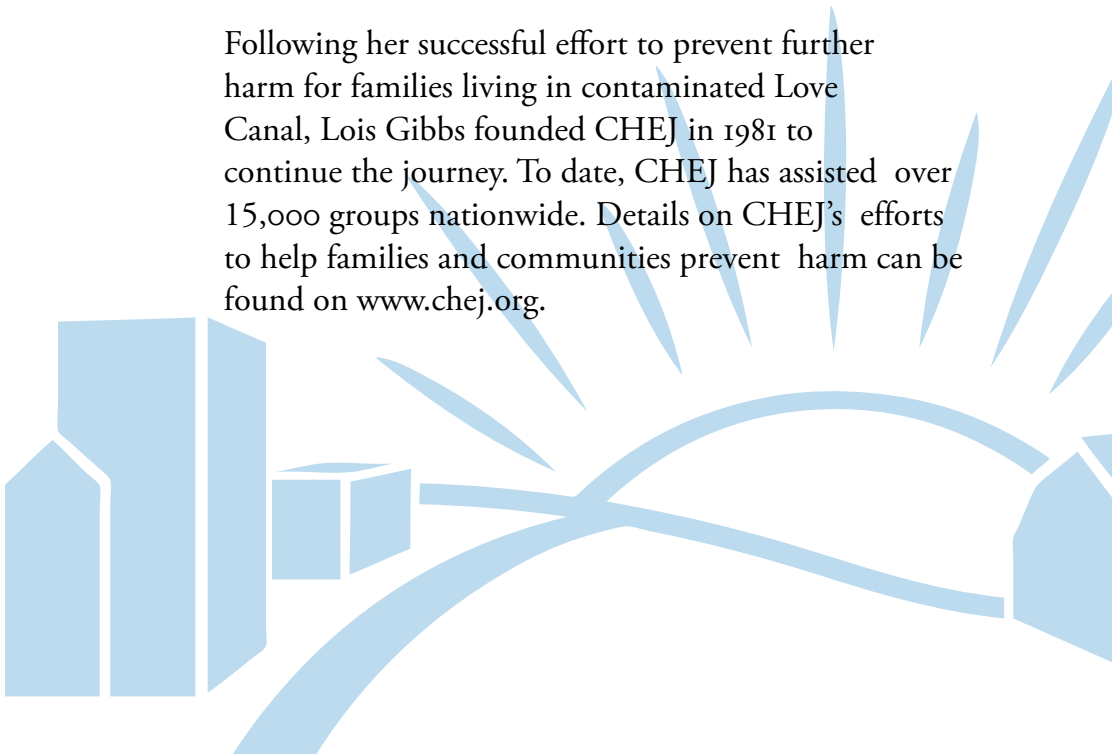


Table of Contents

(page numbers are listed in upper right hand corner)

Introduction to Asphalt Production

Asphalt Fumes

Asphalt Plant Pollution

Asphalt plants mix gravel and sand with crude oil derivatives to make the asphalt used to pave roads, highways, and parking lots across the U.S. These plants release millions of pounds of chemicals to the air during production each year, including many cancer-causing toxic air pollutants such as arsenic, benzene, formaldehyde, and cadmium. Other toxic chemicals are released into the air as the asphalt is loaded into trucks and hauled from the plant site, including volatile organic compounds, polycyclic aromatic hydrocarbons (PAHs), and very fine condensed particulates.[EPA]

■ **Asphalt Fumes are Known Toxins.** The federal Environmental Protection Agency (EPA) states "Asphalt processing and asphalt roofing manufacturing facilities are major sources of hazardous air pollutants such as formaldehyde, hexane, phenol, polycyclic organic matter, and toluene. Exposure to these air toxics may cause cancer, central nervous system problems, liver damage, respiratory problems and skin irritation." [EPA]. According to one health agency, asphalt fumes contain substances known to cause cancer, can cause coughing, wheezing or shortness of breath, severe irritation of the skin, headaches, dizziness, and nausea. [NJDHSS] Animal studies show PAHs affect reproduction, cause birth defects and are harmful to the immune system. [NJDHSS] The US Department of Health and Human Services has determined that PAHs may be carcinogenic to humans. [DHHS]

■ **Health Impacts & Loss of Property Value.** The Blue Ridge Environmental Defense League (BREDL), a regional environmental organization, has done two studies on the adverse impacts on property values and health for residents living near asphalt plants. A property value study documented losses of up to 56% because of the presence of a nearby asphalt plant. In another study, nearly half of the residents reported negative impacts on their health from a new asphalt plant. The door-to-door health survey found 45% of residents living within a half mile of the plant reported a deterioration of their health, which began after the plant opened. The most frequent health problems cited were high blood pressure (18% of people surveyed), sinus problems (18%), headaches (14%), and shortness of breath (9%). [BREDL]

■ **Flawed Tests Underestimate Health Risks.** In addition to smokestack emissions, large amounts of harmful "fugitive emissions" are released as the asphalt is moved around in trucks and conveyor belts, and is stored in stockpiles. A small asphalt plant producing 100 thousand tons of asphalt a year may release up to 50 tons of toxic fugitive emissions into the air. [Dr. R. Nadkarni] Stagnant air and local weather patterns often increase the level of exposure to local communities. In fact, most asphalt plants are not even tested for toxic emissions. The amounts of these pollutants that are released from a facility are estimated by computers and mathematical formulas rather than by actual stack testing, estimates that experts agree do not accurately predict the amount of toxic fugitive emissions released and the risks they pose. According to Dr. Luanne Williams, a North Carolina state toxicologist, 40% of the toxins from asphalt plant smokestacks even meet air quality standards—and for the other 60% of these emissions, the state lacks sufficient data to determine safe levels.

BE SAFE: Take Precautionary Action to Protect Our Communities from Asphalt Plant Air Pollution

BE SAFE's FOUR PRINCIPLES

1. HEED EARLY WARNING SIGNS

There is documented evidence from health experts and federal and state regulators of the serious health effects of asphalt plant emissions. We must heed these early warning signs and take action to prevent communities from further exposure to cancer-causing substances released by asphalt plants. The following actions are needed:

Moratoriums on asphalt plant construction and operation in communities where people live and go to school;

Stricter testing and enforcement of air quality standards at asphalt plants; and

Improved air standards that address all toxic contaminants—including fugitive emissions.

2. PUT SAFETY FIRST

Even if an asphalt plant meets all state and federal air pollution standards, people living nearby are still exposed to cancer-causing substances that can cause long-term damage. These standards are based on the principle of "acceptable risk", and assume each state will enforce the standards, the plants will operate perfectly, and the owners can be trusted to operate on an honor system where they are expected to follow all the laws and regulations that apply to their facility without any government oversight. In the majority of cases, it is unknown whether the 'theoretical' air emissions predicted by computer models and used by plant owners accurately reflect air emissions from a plant's daily operations. We must put safety first and shut down or overhaul the current system that fails to protect communities from the daily health hazards of asphalt plant pollution.

3. EXERCISE DEMOCRACY

Federal regulations based on the "acceptable risk" model and self-regulating honor systems are inadequate to protect public health. Many states rely on inadequate federal standards that do not take into account local factors such as how close an industrial facility is to homes and schools, local weather patterns, and additional 'nuisance' factors such as the effect acrid and nauseating smells have on the quality of life in these communities.

Organizations are working to improve federal and state standards and add asphalt plant fumes to the hazardous air pollutant (HAP) list under the federal Clean Air Act. Communities can take advantage of any state laws aimed at protecting local values that allow counties to determine where new industrial facilities will be located. These communities can band together to work with their county governments to prevent new asphalt plants from being located in their neighborhoods and prevent existing plants from renewing their permits until further evaluation of public health risks are conducted.

BE SAFE is coordinated by the Center for Health, Environment & Justice. To sign the platform or for more information, contact us at CHEJ, P.O. Box 6806, Falls Church, VA 22040, 703-237-2249, or 518-732-4538, or visit **www.besafenet.com**

4. CHOOSE THE SAFEST SOLUTIONS

Communities faced with an asphalt plant proposal should push for setbacks from residences and community buildings, site specific health-based air pollution modeling and monitoring, enclosures for loading zones, and preferably a zero emissions asphalt plant, with total containment of air pollutants.

■ Investigate Pollution in Your Area.

To find out more about asphalt plant pollution in your area, go to **www.scorecard.org**

■ Join the Clean Air Campaign.

Support the campaign on asphalt plant pollution. To find out more, contact the Blue Ridge Environmental Defense League at **www.bredl.org.**

■ BE SAFE.

Take precautionary action to prevent asphalt plant pollution. Sign on to the BE SAFE Platform on the next page. Be counted when we deliver this national Platform to the White House in 2005. Endorse the BE SAFE Platform today at **www.besafenet.com.**

■ Your Vote Counts.

The next election will set the country's course on asphalt plant regulations. For information on environmental voting records, contact **www.sierraclub.org** and **www.lcv.org.** To register to vote, contact **www.earthday.net**

Clean Air Campaign Halts Asphalt Pollution & Improves Air Policies

"Nothing could have prepared us for the horrors of that plant; we cannot be outside when it operates, we are prisoners."

Jerry Starr, Macon County, NC

Blue Ridge Environmental Defense League (BREDL) has been leading a Clean Air Campaign to reduce toxic pollution from asphalt plants in North Carolina. In partnership with many community groups, BREDL defeated numerous asphalt plant proposals, spearheaded a trend of countywide moratoriums on asphalt plant construction and operation, and mounted plant permit challenges. The campaigns included radio ads, posted yard signs, newspaper display ads, and stories in local newspapers.

BREDL and the Clean Air Campaign have succeeded in reducing asphalt pollution and improving air quality policies. North Carolina and Tennessee signed an agreement to protect air quality in the Great Smoky Mountain National Park and other wilderness areas. North Carolina has improved methods to analyze fugitive toxic air emissions and expanded the Toxic Air Pollutant program to include all operating and proposed asphalt plants.

References:

US EPA Office of Air Quality Planning & Standards, AP-42, Fifth Edition, Volume I, Chapter 11: Mineral Products Industry, [EPA] **http://www.epa.gov/ttn/chief/ap42/ch11/final/c11s01.pdf**. *Final Rule to Reduce Toxic Air Emissions From Asphalt Processing & Asphalt Roofing Manufacturing Facilities,* Environmental Protection Agency, June 2000 [EPA]. *Hazardous Substance Fact Sheet, Asphalt Fumes*. New Jersey Department of Health and Senior Services, January 2001 [NJDHSS]. Agency for Toxic Substances and Disease Registry (ATSDR). 1995. *Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs)*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service[DHHS]. Blue Ridge Environmental Defense League Asphalt Health Survey, [BREDL]. Dr. R. Nadkarni developed mass balance equation to estimate total fugitive emissions and his comments to Virginia Dept. of Environmental Quality are at **www.bredl.org/pdf/DEQ072503.pdf.** [Dr. R. Nadkarni].

Primary Contributor: Lou Zeller, Blue Ridge Environmental Defense League.

BE SAFE Platform

In the 21st century, we envision a world in which our food, water and air are clean, and our children grow up healthy and thrive. Everyone needs a protected, safe community and workplace, and natural environment to enjoy. We can make this world vision a reality. The tools we bring to this work are prevention, safety, responsibility and democracy.

Our goal is to prevent pollution and environmental destruction before it happens. We support this precautionary approach because it is preventive medicine for our environment and health. It makes sense to:

- *Prevent pollution and make polluters, not taxpayers, pay and assume responsibility for the damage they cause;*
- *Protect our children from chemical and radioactive exposures to avoid illness and suffering;*
- *Promote use of safe, renewable, non-toxic technologies;*
- *Provide a natural environment we can all enjoy with clean air, swimmable, fishable water and stewardship for our national forests.*

We choose a "better safe than sorry" approach motivated by caution and prevention.
We endorse the common-sense approach outlined in the BE SAFE's four principles listed below.

Platform Principles

HEED EARLY WARNINGS

Government and industry have a duty to prevent harm, when there is credible evidence that harm is occurring or is likely to occur—even when the exact nature and full magnitude of harm is not yet proven.

PUT SAFETY FIRST

Industry and government have a responsibility to thoroughly study the potential for harm from a new chemical or technology before it is used—rather than assume it is harmless until proven otherwise. We need to ensure it is safe now, or we will be sorry later. Research on impacts to workers and the public needs to be confirmed by independent third parties.

EXERCISE DEMOCRACY

Precautionary decisions place the highest priority on protecting health and the environment, and help develop cleaner technologies and industries with effective safeguards and enforcement. Government and industry decisions should be based on meaningful citizen input and mutual respect (the golden rule), with the highest regard for those whose health may be affected and for our irreplaceable natural resources—not for those with financial interests. Uncompromised science should inform public policy.

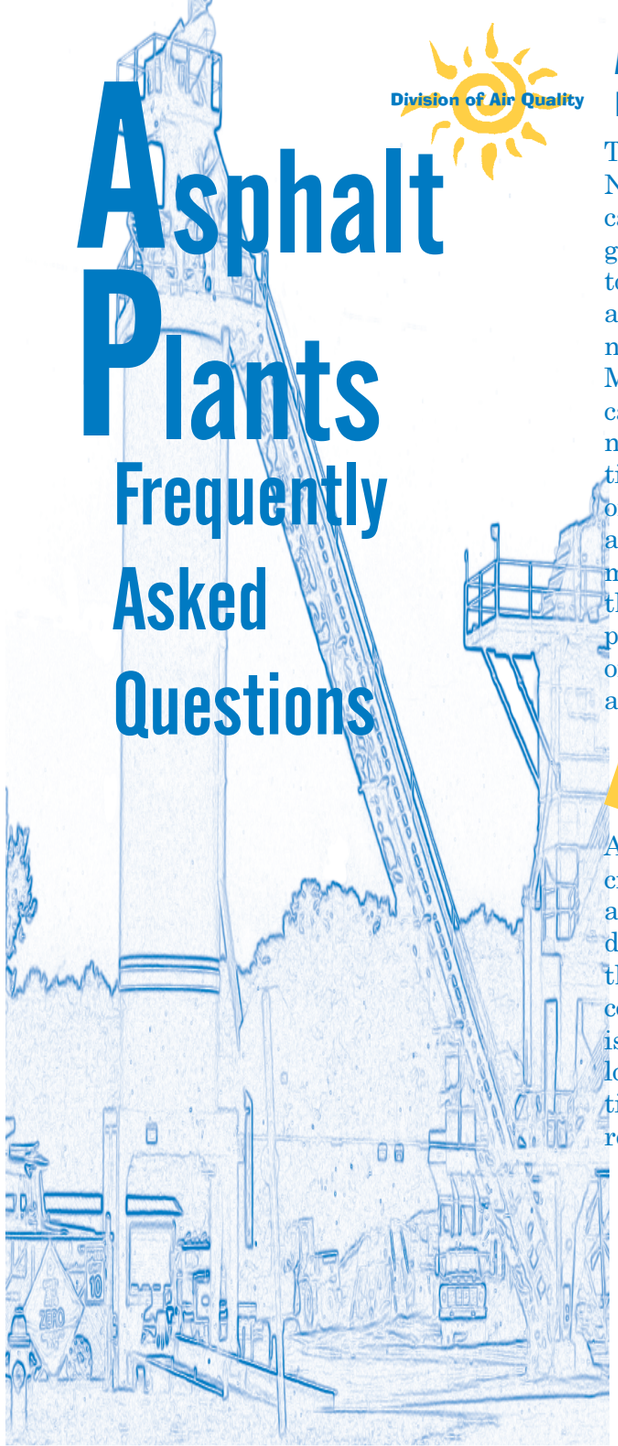
CHOOSE THE SAFEST SOLUTION

Decision-making by government, industry and individuals must include an evaluation of alternatives, and the choice of the safest, technically feasible solutions. We support innovation and promotion of technologies and solutions that create a healthy environment and economy, and protect our natural resources.

Take precautionary action to prevent asphalt plant pollution.
Sign onto the BE SAFE Platform.

Be counted when we deliver this national platform to the White House in 2005.
Endorse the platform today at **www.besafenet.com**

BE SAFE is coordinated by the Center for Health, Environment & Justice. To sign the platform or for more information, contact us at CHEJ, P.O. Box 6806, Falls Church, VA 22040, 703-237-2249, or 518-732-4538, or visit **www.besafenet.com**



Asphalt Plants Frequently Asked Questions

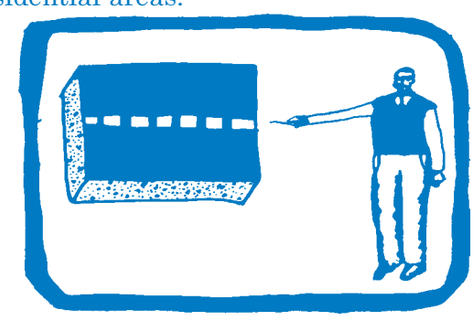
Division of Air Quality

Asphalt Plants: Frequently Asked Questions

The N.C. Department of Environment and Natural Resources (DENR) receives many calls and letters about asphalt plants, generally when companies apply for permits to build new facilities. North Carolina has about 150 asphalt plants, and about five new facilities are permitted each year. Many asphalt plants are portable, so they can be moved to different locations based on needs for new highways and other construction projects. Before a company can build or operate an asphalt plant, it must obtain an air quality permit and in some cases may need water quality permits. In DENR, the Division of Air Quality handles air permits for asphalt plants, and the Division of Water Quality handles water permits (if applicable).

What is asphalt and how is it made?

Asphalt is a paving material made from crushed rock and asphalt cement, which is a mixture of petroleum compounds produced by oil refineries. Asphalt plants heat the asphalt cement in enclosed tanks then combine it with crushed rock. The asphalt is then conveyed to storage silos, where it is loaded onto trucks for delivery to construction sites for highways, parking lots and residential areas.



How do asphalt plants affect air quality?

Air emissions are created at several stages during asphalt production. Most of the emissions come from an asphalt plant's main stack. Fumes from asphalt storage and loading areas account for the remaining air emissions, collectively referred to as fugitive emissions.

Asphalt production, like any process in which materials are heated or burned, can produce a range of air emissions. Many of these same compounds are emitted by cars and trucks, fireplaces and wood stoves, wildfires, and other industries. While some of these emissions potentially can be unhealthy to breathe, such problems can be prevented by requiring asphalt plants to install controls or take other measures that reduce their emissions of harmful air pollutants. That is the guiding principle behind state air quality rules, which set stringent limits for a range of pollutants based on their known health effects. In addition, the Division of Air Quality (DAQ) plans to re-examine its permitting procedures pending the results of a nationwide study of asphalt plant emissions being conducted by the U.S. Environmental Protection Agency (EPA). If changes are warranted based on the EPA study, the DAQ can reopen asphalt plant permits issued since April 1998.

How does the Division of Air Quality control asphalt plants?

All asphalt plants must obtain an air permit from the Division of Air Quality. The DAQ reviews all air permit applications for compliance with state and federal air quality regulations. All asphalt plants must meet air quality limits for particulates, which include dust and soot. In addition, all new, modified or relocated asphalt plants must comply with the state air toxics rules, including emissions from stacks and fugitive sources. To meet air quality limits, all asphalt plants have emissions control equipment such as bagfilters or scrubbers. Other options for curbing their emissions include:

- Limiting production rates or hours of operation.
- Constructing taller emissions stacks.
- Increasing the distance between facilities and property lines.
- Using higher grades of fuel for asphalt heaters.

In reviewing permit applications, the DAQ uses computer models to determine whether emissions will exceed state or federal air quality standards. These computer models, which are approved by the EPA, factor in such information as plant emissions rates, production levels, property lines, local terrain, winds and temperatures. The models assume worst-case meteorological conditions - that is, weather conditions that are most likely to cause air pollution problems.

Is it safe to live near an asphalt plant?

North Carolina's air quality regulations are designed to protect public health. In addition, North Carolina has one of the more stringent state programs for regulating emissions of air toxics. The N.C. Environmental Management Commission adopted the state's air toxics rules in 1990, based on the recommendations of a panel of scientists and health experts who spent more than five years developing a list of air pollutants most likely to pose health risks. The air toxics rules set limits for 105 pollutants that are known to pose either short or long-term hazards for people who breathe them. Under these rules, facilities are not allowed to emit pollutants that exceed any of the air toxics limits at or beyond their property lines. Thus, citizens living near plants that meet the air toxics rules should not be exposed to unhealthy levels of air pollution.

How does the Division of Water Quality control asphalt plants?

Asphalt plants may need a stormwater discharge permit from the Division of Water Quality, depending on where they are located and how they operate. Generally, a facility needs a stormwater permit if it collects rainwater from its site and discharges that runoff into a stream or lake. Many asphalt plants, however, do not discharge their runoff into water bodies and do not need storm water permits. Asphalt plants also may need to obtain sedimentation control permits, which are required if plant construction disturbs more than one acre of land. Most asphalt plants do not require sedimentation control permits. There are no state rules that restrict asphalt plants from locating in floodplains, although some local governments may not allow it.

Why are there are so many asphalt plants?

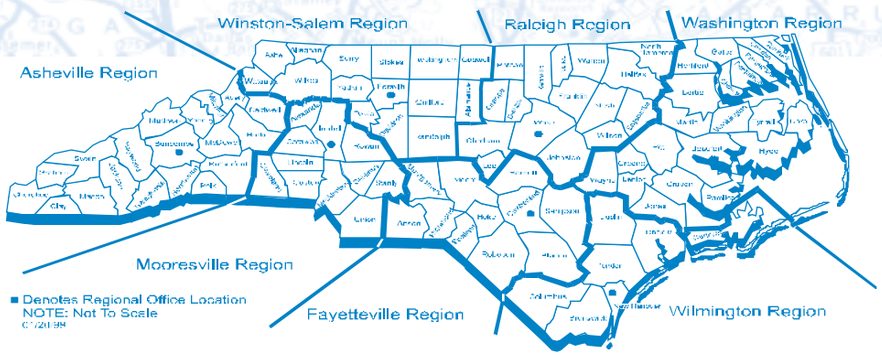
North Carolina has the second-largest state-maintained highway system in the United States, and it takes a lot of asphalt to pave those roads. The state has about 78,000 miles of roads, with more under construction every year. In addition, roads generally need resurfacing every 12 to 15 years, so about 4,400 miles of roads are repaved each year. Another factor contributing to the number of asphalt plants is the nature of the material. Paving is difficult at lower temperatures, and highway contractors must reject asphalt that is not hot enough (at least 250 degrees). That means asphalt plants must be located fairly close to road construction sites.

Who controls where asphalt plants are located?

In reviewing air quality permits for asphalt plants, the Division of Air Quality must ensure that applicants comply with local zoning, and each permit contains a condition stating that the facility must meet these requirements. But the DAQ has no authority over zoning, land use, floodplain development, or where a company decides to build a plant. In North Carolina, local governments are responsible for regulating such land use matters, and they have the final authority over the construction of new facilities through the issuance of building permits. However, many counties and municipalities, particularly in rural areas, have not adopted zoning or land-use controls. The DAQ cannot deny a permit simply because local residents are opposed to a facility; it must base its permitting decisions on whether facilities can meet air quality regulations.

How can I find out more about asphalt plants?

The Division of Air Quality lists applications for air quality permits on its web site, *http://daq.state.nc.us/*. The division's web site also contains news releases with information about asphalt plants, such as changes in regulations or notifications about upcoming public hearings. For more information about asphalt plants or permit applications, contact the division's applicable regional office:



Asheville Regional Office
59 Woodfin Place
Asheville, NC 28801
(828) 251-6208

Fayetteville Regional Office
225 Green St. Suite 714
Fayetteville, NC 28301
(910) 486-1541

Mooresville Regional Office
919 North Main St.
Mooresville, NC 28202
(704) 663-1699

Raleigh Regional Office
3800 Barrett Dr.
Raleigh, NC 27611
(919) 571-4700

Washington Regional Office
943 Washington Square Mall
Washington, NC 27889
(252) 946-6481

Wilmington Regional Office
127 Cardinal Dr. Extension
Wilmington, NC 28405
(910) 395-3900

Winston-Salem Regional Office
585 Waughtown St.
Winston-Salem, NC 27107
(336) 771-4600

NCDENR

ASPARAGINE. See Amino acids.

ASPARTIC ACID. See Amino acids.

ASPHALT

Asphalt is a dark brown to black cementitious material in which the predominating constituents are bitumens that occur in nature or are obtained in petroleum processing. Asphalts characteristically contain very high molecular weight hydrocarbons called asphaltenes and are essentially soluble in carbon disulfide, and aromatic and chlorinated hydrocarbons. Bitumen is a generic term for a class of black or dark-colored (solid, semisolid, or viscous) cementitious substances, natural or manufactured, composed principally of high molecular weight hydrocarbons, of which asphalts, tars, pitches, and asphaltites are typical.

Prior to 1907, most of the asphalt used occurred naturally and included native asphalts, rock asphalts, and lake asphalts. Since the early 1900s, however, most asphalts have been produced from the refining of petroleum and used primarily in paving and roofing applications. Unlike native asphalts, petroleum asphalts are organic with only trace amounts of inorganic materials.

At normal service temperatures, asphalt is viscoelastic; at higher temperatures, it becomes viscous. The disperse phase is a micelle of asphaltenes and the higher molecular weight aromatic components of the petrolenes. Determination of the components of asphalts has always presented a challenge because of the complexity and high molecular weights of the hydrocarbons present. The component of highest carbon content is the fraction termed carboids, which is insoluble in carbon disulfide. This fraction, although organic, is nonasphaltic. The so-called carbenes are insoluble in carbon tetrachloride and soluble in carbon disulfide. Both carboids and carbenes, if present, occur in small amounts. Asphaltenes have a great influence on the viscosity of asphalt. They seem to be relatively constant in composition in residual asphalts, despite the source, as determined by carbon–hydrogen analysis. The nonasphaltene components of asphalt are called maltenes or petrolenes. Properties of asphalts appear in Table 1.

Asphalts are used as protective films, adhesives, and binders because of their waterproof and weather-resistant properties. Some movement without fracture can occur because of their viscous (sol) nature. They have long and continuous satisfactory service because of their slow rate of hardening from heat, oxidation, fatigue, and weathering. Exposed asphalt films harden partially from a loss of volatile oils and to a greater extent from the formation of additional asphaltene fractions and loss of maltenes through oxidation. Such chemical change undoubtedly is catalyzed by uv irradiation. Recent studies have indicated that asphalt stiffness can be used in optimizing performance, although fundamental measures of mechanical properties are preferable. A stiffer asphalt, under uniform loading conditions, could reduce pavement deflection, extend fatigue life, and allow less flow deformation. A softer material would normally allow a longer weathering life before the maltene–asphaltene composition becomes critical in service. Usually, the softest material allowed by initial service needs is selected.

The water resistance of asphalt films is also a manifestation of durability. Asphalts that have a low content of soluble salts show a low water absorption. The pickup of water is primarily a surface manifestation; it softens the film and can cause blistering. Even with a high rate of absorption, asphalt films show little loss of bond to surfaces on continued immersion in water and continue to protect metals from corrosion for long periods of time. Bacteria and fungi can attack the very low molecular weight portion of bituminous materials.

Mineral fillers often are added to asphalts to influence their flow properties and reduce costs. They are used commonly as stabilizers in roofing coatings at concentrations up to 60 wt%. Mineral-filled films show improved resistance to flow at elevated temperatures, improved impact resistance, and better flame-spread resistance. Fillers may increase the water absorption of asphalts. Mineral fillers commonly used are ground limestone, slate flours, finely divided silicas, trap rocks, and mica; they often are produced as by-products in rock-crushing operations. Opaque fillers offer protection from weathering. Asbestos filler has special properties because of its fibrous structure, high resistance to flow, and toughness. It has been used in asphalt paving mixes to increase the

Table 1. Properties of Asphalts

Property	Straight-reduced, residual	Thermal	Air-blown residual
softening point (ring and ball), °C	46	113	93
penetration of 100 g at 25°C for 5 s, mm/10	90	0	20
ductility at 25°C, 5 cm/min, cm	≥ 150	too hard	3.2
specific gravity, 15.6/15.6°C	1.03	1.12	1.05
mean coefficient of cubical expansion/°C			
15.6–65.6°C	0.00063	0.00058	0.00063
15.6–232°C	0.00068	0.00063	0.00068
specific heat, J/(kg·K)[a]			
4.4°C	1675	1549	1633
93.3°C	1968	1842	1926
204.4°C	2345	2177	2303
thermal conductivity at 26.7°C, W/(m·K)	0.16	0.16	0.16
permeability constant at 25°C, kg·m/(m²·s·Pa)[b]			
water vapor	$0.62–1.93 \times 10^{-15}$	1.1×10^{-15}	$1.25–2.4 \times 10^{-15}$
oxygen			0.08×10^{-15}
water absorption of 10-mil films on aluminum panels, wt%			
50 weeks			1.5–10
100 weeks			2.5–16.5
surface tension, mN/m (= dyn/cm)			
25°C	34		32
100°C	27		28
dielectric strength, spherical electrodes, V/m	$11–45 \times 10^6$	36×10^6	$30–35 \times 10^6$
dielectric constant, 50 Hz at 20°C	2.7	3.0	2.7

[a] To convert J to cal, divide by 4.184.
[b] To convert Pa to mm Hg, multiply by 0.0075.

resistance to movement under traffic and in roofing materials for fire-retardant purposes (see Fillers).

Petroleum-derived asphalt, which represents > 99% of total asphalt and asphalt products sold in the United States, is manufactured by the following methods:

Straight reduction. Crude oil at 340–400°C is injected into a fractionating column. The lighter fractions are separated as overhead products, and the residuum is straightreduced asphalt. Crude oil containing ca 30% or more of asphalt can be refined completely in an atmospheric unit to an asphalt cement product. However, most crude oil cannot be distilled at atmospheric pressure because of high percentages of high boiling fractions. As a supplement to the atmospheric process, a second fractionating tower (a vacuum tower) is added. This two-stage process is particularly applicable to crude oils containing 18–30% asphalt. Straight-reduced asphalts are used mainly in pavements, where they serve primarily as binders in paving mixes. The most important recent technical innovation in asphalt paving has been to use asphalt throughout the entire pavement structure (termed total asphalt) to provide more efficient and economical distribution of traffic stresses to the subgrade and provide better protection of the base from intrusion of outside materials, eg, water, soil, etc.

Air-blowing. Asphalt stock (flux) is converted to a harder product by air contact at 200–275°C. Air-blown asphalts are generally more resistant to weather and changes in temperature than straight-reduced asphalts and are produced by batch and continuous methods. Air-blown asphalts of diverse viscosities and flow properties with added fillers, polymers, solvents, and in water emulsions provide products for many applications in roofing and other industries. Air-blowing is also used to produce the harder paving-asphalt grades when the crudes available have a low asphalt content and cannot be reduced directly to grade.

Propane deasphalting involves the precipitation of asphalt from a residuum stock by treatment with propane under controlled conditions. The petroleum stock is usually atmospheric-reduced residue from a primary distillation tower. Propane usually is used in this process although propane–butane mixtures and pentane have been used with some variation in process conditions and hardness of the product. Propane deasphalting is used primarily for crude oils of relatively low asphalt content, generally ≤ 12%. Asphalt produced from this process is blended with other asphaltic residua for making paving asphalt.

Thermal asphalts differ from other asphalts in that they are products of a cracking process. They have relatively high specific gravity, low viscosity, and high temperature susceptibility, and they contain cokelike bodies (carbenes) as indicated by the spot test. Thermal asphalts are used principally as saturants for cellulosic building products such as insulation boards, brick-finish siding, and fiber soil pipes. Currently, their use in road asphalts is rare. Thermal asphalt actually is in very short supply because of changes in cracking methods, and there is little likelihood that it will ever become commonly available.

Blended asphalts may be produced when a refinery stocks two grades of asphalt, one at each end of the viscosity spectrum of the entire product grade requirements. Intermediate grades are prepared by blending (proportioning) the extremes.

Emulsions are immiscible liquids dispersed in one another in the form of very fine droplets from ca 1–25 μm and an average of 5 μm dia. In the most common asphalt emulsion, ie, the oil-in-water type, the asphalt is the dispersed (internal) phase, and water is the continuous (external) phase (see Emulsions). Colloid mills are most commonly used for the manufacture of road emulsions in the United States. A colloid mill usually consists of a rapidly revolving conical disk (rotor). The asphalt, water, and emulsifying agent are forced through the narrow clearance between the rotor and the stator (stationary section).

Industrial emulsions have applications outside the road-building industry. They are made with harder grades of asphalt and contain clays, casein, gelatin, or blood albumin as peptizing agents. Certain clays, such as bentonite, are good emulsion dispersants and impart a buttery consistency to the emulsion. These emulsions have a wide variety of applications, such as in surface coating of asphalt pavements, for built-up roofs, and for other weather coverings.

The large demands for asphalt as a building material were created primarily by mass production of the automobile and the development of asphalt roofing materials (qv) for home construction. The use of asphalt in pavement base-course construction (instead of untreated aggregates), hydraulics, rapid growth in home construction, and the interstate road system have greatly increased its use.

In recent years, the paving market has consumed ca 80% of the product. Asphalt has been used to surface 94% of the United States' highways. The roofing industry typically has accounted for ca 15% of the asphalt market, and miscellaneous industrial asphalts make up the remaining 5%. These products usually are classified only by types (Bureau of Mines), eg, liquid, solid, emulsion; or by use, eg, laminates, pipe coating, automotive asphalts, etc.

The petroleum industry can produce larger quantities of asphalt by adjusting the use of the residual product from refining processes. The integrated refineries have alternative uses for crude-oil residua, ie, coke and residual fuel oil. Very recently, technology improvements have allowed the use of crude residua in existing catalytic cracking units which, in the absence of sufficient distillates normally used for this purpose, places a high alternative value on residua without the attendant need for capital to expand coking facilities. The value of residua for coking and residual fuel establishes a basis for asphalt prices. Asphalt is the preferred product from high sulfur crude stocks because it is a construction material and does not require desulfurization for use as a fuel. Asphalt yields from three crudes are shown in Figure 1.

No significant air-pollution problems are associated with emissions from hot paving operations using several asphalt cements. Concentrations of gaseous substances and emissions from paving-asphalt cement have been found to be very low and within existing EPA and OSHA standards, even when the ambient air sampling was done under confined conditions. Asphalt's very minor content of high molecular weight polynuclear aromatic constituents, however, has been studied as a possible health hazard. The conclusion from these studies suggests that unlike tars, asphalt can be classified in the same manner as particulate dust, but surveillance should be continued although asphalt has not been shown to be a material of significant hazard.

Steps to minimize potential safety hazards in the handling of asphalt are set forth by the American Petroleum Institute and the Asphalt Institute. Hazards include sudden pressure increases from hot asphalt in contact with moisture in enclosed tanks or transports, exposure to air at

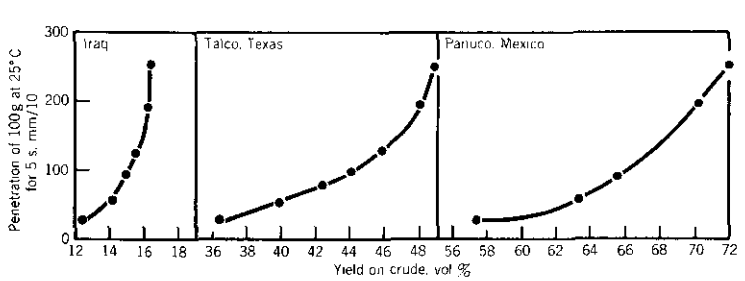


Figure 1. Asphalt yield from three crudes.

$\geq$ 150°C, local overheating above heating coils, flashing of asphalt volatiles in the presence of an ignition source or possible auto-ignition, and hydrogen sulfide from high temperature operations.

JAMES V. EVANS
Amoco Oil Co.

H. Abraham, *Asphalts and Allied Substances*, 6th ed., Vol. 1, D. Van Nostrand Co., Inc., Princeton, N.J., 1960, Chapt. 2.

R.N. Traxler, *Asphalt*, Reinhold Publishing Corp., New York, 1961.

A Brief Introduction to Asphalt and Some of Its Uses, *Manual Series No. 5*, The Asphalt Institute, College Park, Md., 1975.

A.J. Hoiberg, *Bituminous Materials: Asphalts, Tars, and Pitches*, Vols. 1, 2, and 3, Interscience Publishers, a division of John Wiley & Sons, Inc., New York, 1964–1966.

Asphalt Plant Emissions: What are the issues during Site Assignment?

by Ravi Nadkarni

Air Emissions: Emissions of pollutants to the air come from two types of sources: the various stacks in the plant and from other miscellaneous sources, which are called fugitive emissions. At an asphalt plant, incoming stone and gravel are dried in a fuel-fired dryer (which uses natural gas and/or fuel oil) and then mixed with heated liquid asphalt. The gases from the dryer, the mixer, and from various pieces of enclosed equipment are cleaned in a fabric filter (like a large vacuum cleaner bag) and sent to the main stack. The emissions from this stack include gases such as nitrogen oxides, carbon dioxide and sulfur dioxide which result from fuel combustion. In addition, they contain various hydrocarbons which are either unburnt fuel or volatilized organic compounds which come from the hot asphalt. Asphalt is a residue from petroleum refining. While it is a solid at room temperature, it is a watery, low-viscosity liquid at the operating temperature of around 300 to 350 degrees F. This asphalt is stored in a heated tank which has its own stack for the heater (which burns fuel to heat the asphalt) and a vent to allow asphalt vapors to escape when the tank is being filled. Stack emissions contain various "criteria" pollutants which are regulated under the Clean Air Act. These include sulfur dioxide, nitrogen oxides and particulates. Our sense is that stack emissions are analyzed properly by Massachusetts Department of Environmental Protection during the review of the Air Permit Application. Nationally, the same is more or less true since all states operate in a similar fashion with technical guidance from the EPA.

Fugitive emissions: These are uncontrolled emissions, often at ground level, from equipment such as hot asphalt storage silos, conveyors, etc, or from miscellaneous openings. Fugitive emissions also occur during routine operations such as the process of loading hot mix asphalt into trucks. Asphalt contains a wide variety of light and heavy hydrocarbons derived from the processing of petroleum crudes. At the operating temperature, the lighter components evaporate from the liquid and become fugitive emissions of hydrocarbons. There are two major categories: The invisible emissions are mainly non-condensible Volatile Organic Compounds (VOCs) which contribute to smog. EPA research has shown that these VOCs are about 30% of the total hydrocarbons that volatilize. The second category contains heavier hydrocarbons that evaporate at the operating temperature of around 300 degrees F but then condense in the cooler ambient air. These condensed organic compounds are visible and are known in the trade as "blue smoke". Because these condensed organics are in the form of sub-micron particles, they don't settle out like dust particles and will travel great distances before their concentration becomes too low for them to be visible. However, the characteristic odor of asphalt will reveal their presence. The City of Boston Board of Health and Hospitals has collected information from many cities and towns about asphalt plants. They find that complaints about odor, indicating the presence of these volatilized organics, have been recorded at distances as far as a mile from asphalt plants indicating the distance that these emissions travel. The concern about these organic emissions is two fold. The lighter VOCs lead to smog. More important, the heavier emissions contain significant concentrations of polynuclear aromatic compounds which cause cancer and mutations. Unfortunately, the EPA and DEP have totally ignored fugitive emissions until now. This is unfortunate since these fugitives contain hydrocarbon VOCs, regulated as ozone precursors, and particulate condensed organics which contain cancer-causing chemicals, which should be regulated but are not. There has been a significant effort devoted to quantification of these fugitive emissions by concerned citizens. The EPA and DEP are being reluctantly dragged into accepting the fact that these emissions can't and should not be ignored.

An asphalt plant is often associated with a quarry and a screening plant for crushed stone and gravel. These plants generate a lot of dust. Even when an adjacent quarry is absent, the crushed stone and gravel is stored in the open and generates wind-borne dust. While dust control is easy in principle, with the use of water sprays to suppress its generation, it is rarely implemented properly. First, there is an economic incentive not to be generous with the water sprays; after all this water has to be purchased and then it has to be evaporated in the dryer using purchased fossil fuel. Second, a lot of the dust is generated by the truck traffic associated with the asphalt plant and/or quarry. Much of this dust can be deposited on public roads and streets and is no longer on the plant property, although it originated on the plant property. Although inhaling large amounts of such dust leads to silicosis, plant workers and neighbors are generally not exposed to dust loadings where this becomes a serious problem. Rather, it is a nuisance and a major source of complaints.

Emissions to Water: While hot mix asphalt is produced by a totally dry process, each truck is normally coated with a "release agent" to prevent the asphalt from sticking to the truck body. While distillate fuels such as kerosene have been used as release agents in the past, the current practice is to use a water-based release agent called Polyslip. This Polyslip foam is applied

generously to the truck body by the truck driver and the excess drips to the ground as is carried into ground water by rain or runoff.

Polyslip contains a mixture of synthetic detergents such as sodium metasilicate and sodium tripolyphosphate. The other components are butoxyethanol and dodecyl benzene sulfonate. These chemicals are poisons. The toxic dose based on oral ingestion by mice, rats and rabbits, is about 1 gram per kilogram of body weight. For humans, this acute toxic dose translates to about 1 ounce for an adult. Generally, acute toxicity will not be an issue. However, at much lower concentrations and chronic exposures, butoxyethanol is a teratogen (causes birth defects) and a mutagen in its ester form. We have seen such facilities sited near supplies of drinking water or near a cranberry bog, in one instance. The asphalt plants generally call these materials, "a soap solution approved by DEP" and dismiss the issue of health effects because these chemicals are supposedly biodegradable. Boards of Health need to pay more attention to this area, especially when there is potential for contamination of drinking water supplies.

Solid Wastes: A hot mix plant does not produce solid wastes but it often can consume solid and hazardous wastes such as Recycled Asphalt Pavement (RAP) and soil contaminated with oil. RAP is generally much higher in volatile components than is the case with new hot mix asphalt so the air emissions are higher when RAP is being used. A real concern is how these materials are stored on site and whether the toxic chemicals in these solids can be leached into ground water by rainfall.

Noise and other nuisance issues: Asphalt plants are noisy because of the truck traffic, because of associated quarry operations and because the burner on the dryer is also noisy. In addition, if the plant has a contract with Mass Department of Highways, it is required to operate around the clock, regardless of local ordinances. Complaints from neighbors during such periods indicate that the noise from backup alarms on trucks and other mobile equipment is particularly annoying at night.

Overall, the concerns about asphalt plants are a result of their emissions to the air and water and their propensity to be sited near residential areas with minimal buffer zones and in areas such as gravel pits which are also aquifers and sources of drinking water. Some states like South Dakota only allow mobile asphalt plants so that a particular neighborhood does not have to breathe asphalt fumes for scores of years. Even in a petroleum-friendly state like Texas, a half mile buffer zone is required so that an asphalt plant can't be sited closer than half mile from any residence without special state environmental review.

MAHB QUARTERLY
MASSACHUSETTS ASSOCIATION OF HEALTH BOARDS, INC.
VOLUME 14 NUMBER 4 FALL 1996

United States Environmental Protection Agency | Office Of Air Quality Planning And Standards Research Triangle Park, NC 27711 | EPA-454/R-00-019 December 2000

Air

HOT MIX ASPHALT PLANTS

EMISSION ASSESSMENT REPORT

LIST OF ACRONYMS

ASTM	American Society of Testing and Materials
Btu	British thermal unit
CH_4	methane
CO	carbon monoxide (as measured by EPA Method 10)
CO_2	carbon dioxide (as measured by EPA Method 3)
EPA	Environmental Protection Agency
HAP	hazardous air pollutant (listed in or pursuant to section 112(b) of the 1990 Clean Air Act Amendments)
HMA	hot mix asphalt
NO_x	nitrogen oxides (as measured by EPA Method 7)
PAH	polycyclic aromatic hydrocarbon (a class of HAPs)
PM	particulate matter (as measured by EPA Methods 5 or 17)
PM-10	particulate matter less than 10 microns in diameter
PM-2.5	particulate matter less than 2.5 microns in diameter
RAP	reclaimed asphalt pavement
RTFOT	rolling thin film oven test (ASTM Method D2872-88)
SCC	source classification code
SO_2	sulfur dioxide (as measured by EPA Methods 6 or 8)
SO_x	sulfur oxides
TOC	total organic compounds (as measured by EPA Method 25A)
VOC	volatile organic compound (refer to 40 CFR 51.100); VOC is TOC plus formaldehyde, less methane, ethane, acetone, and other chemicals listed as negligibly photochemically reactive.

1. EXECUTIVE SUMMARY

1.1 INTRODUCTION

This report presents an assessment of emissions from hot mix asphalt (HMA) manufacturing facilities. Included in the report is a description of the manufacturing process and the emissions associated with HMA production; the procedures for developing emission factors and emission inventories for the HMA industry; and estimated annual emissions for typical HMA facilities.

1.2 OVERVIEW OF HMA INDUSTRY

Hot mix asphalt is used primarily as paving material and consists of a mixture of aggregate and liquid asphalt cement, which are heated and mixed in measured quantities. Hot mix asphalt facilities can be broadly classified as either drum mix plants or batch mix plants, according to the process by which the raw materials are mixed. In a batch mix plant, the aggregate is dried first, then transferred to a mixer where it is mixed with the liquid asphalt. In a drum mix plant, a rotary dryer serves to dry the aggregate and mix it with the liquid asphalt cement. After mixing, the HMA generally is transferred to a storage bin or silo, where it is stored temporarily. From the silo, the HMA is emptied into haul trucks, which transport the material to the job site. Figure 1 presents a diagram of a typical batch mix HMA plant; a typical drum mix HMA plant is depicted in Figure 2.

In 1996, approximately 500 million tons of HMA were produced at the 3,600 (estimated) active asphalt plants in the United States. Of these 3,600 plants, approximately 2,300 are batch plants, and 1,300 are drum mix plants. The total 1996 HMA production from batch and drum mix plants is estimated at about 240 million tons and 260 million tons, respectively. Based on these figures, an average batch mix plant produces approximately 100,000 tons of HMA annually, and an average drum mix plant produces about 200,000 tons of HMA per year. Natural gas fuel is used to produce 70 to 90 percent of the HMA. The remainder of the HMA is produced using oil, propane, waste oil, or other fuels.

The primary emission sources associated with HMA production are the dryers, hot bins, and mixers, which emit particulate matter (PM) and a variety of gaseous pollutants. Other emission sources found at HMA plants include storage silos, which temporarily hold the HMA; truck load-out operations, in which the HMA is loaded into trucks for hauling to the job site; liquid asphalt storage tanks; hot oil heaters, which are used to heat the asphalt storage tanks; and yard emissions, which consist of fugitive emissions from the HMA in truck beds. Emissions also result from vehicular traffic on paved and unpaved roads, aggregate storage and handling operations, and vehicle exhaust.

The PM emissions associated with HMA production include the criteria pollutants PM-10 (PM less than 10 micrometers in aerodynamic diameter) and PM-2.5, hazardous air pollutant (HAP) metals, and HAP organic compounds. The gaseous emissions associated with HMA production include the criteria pollutants sulfur dioxide (SO_2), nitrogen oxides (NO_x), carbon monoxide (CO), and volatile organic compounds (VOC), as well as volatile HAP organic compounds.

1.3 DEVELOPMENT AND USE OF EMISSION FACTORS FOR HMA FACILITIES

An emission factor relates the quantity (weight) of pollutants emitted to a unit of activity of the source. Emission factors for the HMA industry are generally determined in units of pounds of pollutant emitted per ton of HMA produced. These emission factors typically are used to estimate area-wide

emissions for a large number of facilities and emissions for specific facilities where source-specific emissions data are not available or where source testing is cost prohibitive.

To develop emission factors for the HMA industry, data from more than 390 emission test reports and other documents on the industry were compiled and reviewed. Through a careful screening process, the documents that were determined to be unusable for emission factor development were excluded from further evaluation. The remaining reports were compiled by plant type, emission source, pollutant, and emission control. For each emission test, emission factors were calculated by dividing the measured emission rates by the HMA production rate measured at the time of the emission test. These emission factors were then grouped by source, pollutant, and control device, and an average emission factor was calculated for each group.

Emission factors can be used to estimate emissions from one or more HMA facilities by multiplying the emission factor by the HMA production rate. For example, the emission factor for CO emissions from a natural gas-fired drum mix dryer is 0.13 pounds per ton (lb/ton). If the dryer produces 200,000 tons per year (ton/yr), the estimated CO emissions during that period would be: 200,000 ton/yr × 0.13 lb/ton = 26,000 lb/yr or 13 tons/yr.

1.4 ESTIMATED ANNUAL EMISSIONS FROM TYPICAL HMA FACILITIES

Annual emissions for a facility can be estimated by summing up the emissions from each emission source over the course of a year. Annual emissions for a specific source can be estimated by multiplying the annual throughput or production rate for that source by its corresponding emission factors. For an HMA facility, annual emissions can be estimated by multiplying the annual HMA production rate by the emission factors for each type of source at the facility. Table 1 summarizes annual emissions for a typical HMA batch mix plant, and Table 2 summarizes annual emissions for a typical drum mix HMA plant. The estimates presented in these tables account for all of the identified emission sources at each type of facility. For both batch mix plants (Table 1) and drum mix plants (Table 2), the estimate includes emissions from the dryer/mixer, load-out operations, asphalt storage, yard (fugitive emissions from loaded trucks), diesel exhaust, paved and unpaved road dust, and aggregate processing (screening, conveyor transfer, and reclaimed asphalt pavement [RAP] crushing). Additionally, for the drum mix plant (Table 2), the estimate includes emissions from silo filling operations. Estimates are presented for criteria pollutants (pollutants for which national ambient air quality standards have been developed) and hazardous air pollutants (HAPs, as defined in section 112(b) of the 1990 Clean Air Act Amendments). Criteria pollutants include PM-10, VOC, CO, SO_2, and NO_X. Emissions for three classes of HAPs are presented in Tables 1 and 2: polycyclic aromatic hydrocarbons (PAHs), volatile organic HAPs, and metal HAPs. The emissions were estimated using the emission factors developed for the HMA industry and the following assumptions:

- Dryers are fueled with natural gas or No. 2 fuel oil (estimates are presented for both types). It is estimated that between 70 and 90 percent of HMA plants use natural gas, although some HMA plants use fuel oil as an alternative to natural gas.
- Dryer emissions are controlled with fabric filters.
- PM emissions from load-out and silo filling are entirely PM-10.
- Annual HMA production rate for a typical batch mix plant is 100,000 ton/yr.
- Annual HMA production rate for a typical drum mix plant is 200,000 ton/yr.
- The typical HMA plant has two 18,000-gallon asphalt storage tanks.

As indicated in Table 1, a typical batch mix plant using a No. 2 fuel oil-fired dryer emits over 74,000 lb/yr of criteria pollutants, and a typical batch mix plant using a natural gas-fired dryer emits over

56,000 lb/yr of criteria pollutants, of which approximately 41,000 lb/yr are CO and approximately 10,700 lb/yr are PM-10; emissions of other criteria pollutants range from about 500 to about 12,000 lb/yr. The same plant would emit about 770 lb/yr of HAPs. A typical drum mix plant using a No. 2 fuel oil-fired dryer emits about 83,000 lb/yr of criteria pollutants, and a typical drum mix plant using a natural gas-fired dryer emits around 75,000 lb/yr of criteria pollutants, of which approximately 28,000 lb/yr are CO, about 10,000 lb/yr are VOC, and around 31,000 lb/yr are PM-10. A typical drum mix plant emits from 1,300 to 2,000 lb/yr of HAPs, depending on the fuel used in the dryer.

TABLE 1. ESTIMATED ANNUAL EMISSIONS FOR A TYPICAL BATCH MIX HMA FACILITY[a]

Pollutant	Annual emissions by source, pounds per year								
	Mobile sources (diesel exhaust)	Material handling and road dust	No. 2 fuel oil-fired dryer, hot screens, and mixer[b]	Natural gas-fired dryer, hot screens, and mixer[c]	Load-out[d]	Asphalt Storage[e]	Yard[f]	Total[g] (oil-fired)	Total[g] (gas-fired)
Criteria air pollutants									
Particulate matter less than 10 micrometers (PM-10)	46	7,900	2,700	2,700	52			10,700	10,700
Volatile organic compounds (VOC)	100		820	820	391	32	110	1,500	1,500
Carbon monoxide (CO)	700		40,000	40,000	135	3	35	41,000	41,000
Sulfur dioxide (SO_2)	22		8,800	460				8,800	480
Nitrogen oxides (NO_x)	380		12,000	2,500				12,400	2,900
Hazardous air pollutants (HAPs)									
Polycyclic aromatic hydrocarbons (PAHs)	0.035		11	11	2.0	0.12		13	13
Phenol					0.40			0.40	0.40
Volatile HAPs	1.9		751	751	6.2	140	1.6	760	760
Metal HAPs			1.4	1.4				1.4	1.4
Total HAPs[g]	1.9		760	760	8.6	140	1.6	770	770

[a] Based on an annual HMA production rate of 100,000 tons per year.
[b] Between 10 and 30 percent of the HMA is produced using fuel oil.
[c] Between 70 and 90 percent of the HMA is produced using natural gas.
[d] Loading of HMA into haul trucks.
[e] Includes emissions from oil-fired hot oil heaters.
[f] Fugitive emissions from loaded trucks prior to departure to the job site.
[g] Total expressed using two significant figures.

TABLE 2. ESTIMATED ANNUAL EMISSIONS FOR A TYPICAL DRUM MIX HMA FACILITY[a]

Pollutant	Annual emissions by source, pounds per year									
	Mobile sources (diesel exhaust)	Material handling and road dust	No. 2 fuel oil-fired dryer[b]	Natural gas-fired dryer[c]	Load-out[d]	Silo filling[e]	Asphalt storage[f]	Yard[g]	Total[h] (oil-fired)	Total[h] (gas-fired)
Criteria air pollutants										
Particulate matter less than 10 micrometers (PM-10)	220	26,000	4,600	4,600	104	117			31,000	31,000
Volatile organic compounds (VOC)	190		6,400	6,400	782	2,440	64	220	10,000	10,000
Carbon monoxide (CO)	1,200		26,000	26,000	270	236	6	72	28,000	28,000
Sulfur dioxide (SO_2)	26		2,200	680					2,200	710
Nitrogen oxides (NO_x)	560		11,000	5,200					12,000	5,800
Hazardous air pollutants (HAPs)										
Polycyclic aromatic hydrocarbons (PAHs)	0.13		176	37	4.0	5.8	0.12		190	50
Phenol					0.80				0.80	0.80
Volatile HAPs	6.6		1,560	1,020	12.4	31	140	3.3	1,800	1,200
Metal HAPs			19	16					19	16
Total HAPs[h]	6.7		1,800	1,100	17	37	140	3.3	2,000	1,300

[a] Based on an annual HMA production rate of 200,000 tons per year.
[b] Between 10 and 30 percent of the HMA is produced using fuel oil.
[c] Between 70 and 90 percent of the HMA is produced using natural gas.
[d] Loading of HMA into haul trucks
[e] Filling of temporary storage silo prior to load-out.
[f] Includes emissions from oil-fired hot oil heaters.
[g] Fugitive emissions from loaded trucks prior to departure to the job site.
[h] Total expressed using two significant figures.

The emissions estimates presented in Tables 5 through 12 are based on the emission factors developed for the HMA industry and the following assumptions:

- Batch mix plant and drum mix plant dryers are fueled with either natural gas or fuel oil. It is estimated that between 70 and 90 percent of HMA plants use natural gas, although some HMA plants use fuel oil as an alternative to natural gas. As shown in Tables 5 and 8, fuel oil-fired mixers and dryers have higher emissions of SO_2, NO_x, and some HAPs.
- Batch mix plant dryer, hot screens, and mixer and drum mix plant dryer emissions are controlled with fabric filters.
- PM emissions from load-out and silo filling are entirely PM-10. (However, the organic portion of these emissions also can be assumed to be PM-2.5. Information is available in AP-42 Appendix B.1, Particle Size Distribution Data and Sized Emission Factors for Selected Sources, for categorizing the inorganic or filterable PM into PM-10 and PM-2.5 fractions.)
- Average asphalt loss on heating is -0.5 percent (asphalt volatility).
- Average HMA load-out temperature is 325°F.
- The typical HMA plant has two asphalt storage tanks that are 50 feet long and 8 feet in diameter. It is estimated that these storage tanks require a total heating capacity of about 200,000 Btu/hr, based on a heat loss of 60 Btu/ft^2 of tank surface area. The asphalt storage tanks are kept at 325°F continuously for the five months the HMA plant operates. As a result, 720 million Btu are used to maintain the temperature of the asphalt in the storage tank. For a gas-fired hot oil heater, 720,000 ft^3 of gas is combusted. For an oil-fired hot oil heater, 5,100 gallons of fuel oil are combusted. It should be noted that this fuel usage is about 3 percent of the fuel used in a typical batch mix plant and 1.6 percent of the fuel used in a typical drum mix plant.

TABLE 3. MATRIX OF EMISSION FACTORS DEVELOPED FOR HMA SOURCES

Plant type	Source	Criteria pollutants	HAPs	Other pollutants
Batch mix	Dryer, hot screens, and mixer	PM-10, NO_x, CO, SO_2, VOC	24 organic HAPs 9 metal HAPs	CO_2 4 other organics 3 other metals
	Hot oil heaters		22 organic HAPs	
	Load-out	PM, CO, VOC,	41 organic HAPs	3 other organics
	Yard emissions	VOC	19 organic HAPs	
Drum mix	Dryer	PM-10, NO_x, CO, SO_2, VOC	58 organic HAPs 11 metal HAPs	CO_2 15 other organics, 6 other metals
	Hot oil heaters		22 organic HAPs	
	Load-out	PM, CO, VOC	41 organic HAPs	3 other organics
	Silo filling	PM, CO, VOC	28 organic HAPs	3 other organics
	Yard emissions	VOC	19 organic HAPs	

TABLE 5. ESTIMATED ANNUAL EMISSIONS FOR A TYPICAL BATCH MIX PLANT DRYER, HOT SCREENS, AND MIXER[a]

Pollutant	Oil-fired dryer	Natural gas-fired dryer
	Emissions, lb/yr	
Criteria Pollutants		
PM-10	2,700	2,700
VOC	820	820
CO	40,000	40,000
SO_2	8,800	460
NO_x	12,000	2,500
PAHs (semi-volatile HAPs)		
Naphthalene	3.6	3.6
2-Methylnaphthalene	7.1	7.1
Acenaphthene	0.090	0.090
Acenaphthylene	0.058	0.058
Anthracene	0.021	0.021
Benzo(a)anthracene	0.00046	0.00046
Benzo(a)pyrene	0.000031	0.000031
Benzo(b)fluoranthene	0.00094	0.00094
Benzo(g,h,i)perylene	0.00005	0.00005
Benzo(k)fluoranthene	0.0013	0.0013
Chrysene	0.00038	0.00038
Dibenz(a,h)anthracene	0.0000095	0.0000095
Fluoranthene	0.016	0.016
Fluorene	0.16	0.16
Indendo(1,2,3-cd)pyrene	0.00003	0.00003
Phenanthrene	0.26	0.26
Pyrene	0.0062	0.0062
Total PAHs	11	11
Volatile HAPs		
Acetaldehyde	32	32
Benzene	28	28
Ethylbenzene	220	220
Formaldehyde	74	74
Quinone	27	27
Toluene	100	100
Xylene	270	270
Total Volatile HAPs	751	751
Metal HAPs		
Arsenic	0.046	0.046
Beryllium	0.015	0.015
Cadmium	0.061	0.061
Chromium	0.057	0.057
Lead	0.089	0.089
Manganese	0.69	0.69
Mercury	0.041	0.041
Nickel	0.3	0.3
Selenium	0.049	0.049
Total metal HAPs	1.35	1.35

[a] Dryer, hot screens, and mixer controlled by fabric filter producing 100,000 tons of hot mix asphalt per year. Between 70 and 90 percent of HMA is produced using natural gas; most of the remaining HMA is produced using fuel oil.

TABLE 6. ESTIMATED ANNUAL EMISSIONS FOR TYPICAL BATCH MIX PLANT LOAD-OUT OPERATIONS[a]

Pollutant	Emissions, lb/yr
Criteria Pollutants	
PM-10	52
VOC	391
CO	135
PAHs (semi-volatile HAPs)	
Acenaphthene	0.089
Acenaphthylene	0.0095
Anthracene	0.0239
Benzo(a)anthracene	0.0065
Benzo(b)fluoranthene	0.0026
Benzo(k)fluoranthene	0.00075
Benzo(g,h,i)perylene	0.00065
Benzo(a)pyrene	0.00078
Benzo(e)pyrene	0.0027
Chrysene	0.035
Dibenz(a,h)anthracene	0.00013
Fluoranthene	0.017
Fluorene	0.26
Indeno(1,2,3-cd)pyrene	0.00016
2-Methylnaphthalene	0.81
Naphthalene	0.43
Perylene	0.0075
Phenanthrene	0.28
Pyrene	0.051
Total PAHs	2.02
Other semi-volatile HAPs	
Phenol	0.40
Volatile HAPs	
Benzene	0.22
Bromomethane	0.040
2-Butanone	0.20
Carbon disulfide	0.054
Chloroethane	0.00087
Chloromethane	0.062
Cumene	0.46
Ethylbenzene	1.16
Formaldehyde	0.37
n-Hexane	0.62
Isooctane	0.0075
Methylene chloride	0.00
Methyl tert-butyl ether	0.00
Styrene	0.030
Tetrachloroethene	0.032
Toluene	0.87
1,1,1-Trichloroethane	0.00
Trichloroethene	0.00
Trichlorofluoromethane	0.0054
m-/p-Xylene	1.70
o-Xylene	0.33
Total volatile HAPs	6.18

[a] Uncontrolled emissions from 100,000 tons of hot mix asphalt per year.

TABLE 7. ESTIMATED ANNUAL EMISSIONS FOR TYPICAL BATCH MIX PLANT ASPHALT STORAGE TANK[a]

Pollutant	Emissions, lb/yr
Criteria Pollutants	
PM-10	ND
VOC	32
CO	3
PAHs (semi-volatile HAPs)	
Acenaphthene	0.0027
Acenaphthylene	0.0010
Anthracene	0.00092
Benzo(b)fluoranthene	0.00051
Fluoranthene	0.00022
Fluorene	0.00016
Naphthalene	0.087
Phenanthrene	0.025
Pyrene	0.00016
Total PAHs	0.12
Volatile HAPs	
Benzene	0.010
Bromomethane	0.0016
2-Butanone	0.012
Carbon disulfide	0.0051
Chloroethane	0.0012
Chloromethane	0.0074
Ethylbenzene	0.012
Formaldehyde	140
n-Hexane	0.032
Isooctane	0.000099
Methylene chloride	0.000086
Phenol	0.00
Styrene	0.0017
Toluene	0.020
m-/p-Xylene	0.061
o-Xylene	0.018
Total volatile HAPs	140

[a] Uncontrolled emissions from plant producing 100,000 tons of hot mix asphalt per year. Includes emissions from oil-fired hot oil heaters. All calculated PAH emissions and almost all of the formaldehyde emissions are from the oil-fired hot oil heater.

TABLE 8. ESTIMATED ANNUAL EMISSIONS FOR A TYPICAL DRUM MIX DRYER[a]

Pollutant	No. 2 fuel oil-fired dryer	Natural gas-fired dryer
	Emissions, lb/yr	
Criteria Pollutants		
PM-10	4,600	4,600
VOC	6,400	6,400
CO	26,000	26,000
SO_2	2,200	680
NO_x	11,000	5,200
PAHs (semi-volatile HAPs)		
2-Methylnaphthalene	34	15
Acenaphthene	0.28	0.28
Acenaphthylene	4.4	1.7
Anthracene	0.62	0.044
Benzo(a)anthracene	0.042	0.042
Benzo(a)pyrene	0.0020	0.0020
Benzo(b)fluoranthene	0.020	0.020
Benzo(e)pyrene	0.022	0.022
Benzo(g,h,i)perylene	0.0080	0.0080
Benzo(k)fluoranthene	0.0082	0.0082
Chrysene	0.036	0.036
Fluoranthene	0.12	0.12
Fluorene	2.2	0.76
Indeno(1,2,3-cd)pyrene	0.0014	0.0014
Naphthalene	130	18
Perylene	0.0018	0.0018
Phenanthrene	4.6	1.5
Pyrene	0.60	0.11
Total PAHs	180	37
Volatile HAPs		
Isooctane	8.0	8.0
Hexane	184	180
Benzene	78	78
Ethylbenzene	48	48
Formaldehyde	620	620
Methyl chloroform	9.6	9.6
Toluene	580	30
Xylene	40	40
Total volatile HAPs	1,568	1,020
Metal HAPs		
Lead	3	0.12
Mercury	0.52	0.048
Antimony	0.036	0.036
Arsenic	0.11	0.11
Beryllium	0.000	0.000
Cadmium	0.082	0.082
Chromium	1.1	1.1
Manganese	1.5	1.5
Nickel	12.6	12.6
Selenium	0.070	0.070
Total metal HAPs	19	16

[a] Dryer controlled by fabric filter producing 200,000 tons of hot mix asphalt per year. Between 70 and 90 percent of HMA is produced using natural gas; most of the remaining HMA is produced using fuel oil.

TABLE 9. ESTIMATED ANNUAL EMISSIONS FOR TYPICAL DRUM MIX PLANT LOAD-OUT OPERATIONS[a]

Pollutant	Emissions, lb/yr
Criteria Pollutants	
PM-10	104
VOC	780
CO	270
PAHs (semi-volatile HAPs)	
Acenaphthene	0.177
Acenaphthylene	0.0191
Anthracene	0.0477
Benzo(a)anthracene	0.013
Benzo(b)fluoranthene	0.0052
Benzo(k)fluoranthene	0.0015
Benzo(g,h,i)perylene	0.0013
Benzo(a)pyrene	0.00157
Benzo(e)pyrene	0.0053
Chrysene	0.070
Dibenz(a,h)anthracene	0.00025
Fluoranthene	0.034
Fluorene	0.53
Indeno(1,2,3-cd)pyrene	0.00032
2-Methylnaphthalene	1.62
Naphthalene	0.85
Perylene	0.015
Phenanthrene	0.55
Pyrene	0.10
Total PAHs	4.05
Other semi-volatile HAPs	
Phenol	0.80
Volatile HAPs	
Benzene	0.43
Bromomethane	0.080
2-Butanone	0.41
Carbon disulfide	0.11
Chloroethane	0.0017
Chloromethane	0.12
Cumene	0.91
Ethylbenzene	2.3
Formaldehyde	0.73
n-Hexane	1.25
Isooctane	0.015
Methylene chloride	0.00
Methyl tert-butyl ether	0.00
Styrene	0.06
Tetrachloroethene	0.064
Toluene	1.74
1,1,1-Trichloroethane	0.00
Trichloroethene	0.00
Trichlorofluoromethane	0.011
m-/p-Xylene	3.40
o-Xylene	0.66
Total volatile HAPs	12.35

[a] Uncontrolled emissions from 200,000 tons of hot mix asphalt per year.

TABLE 10. ESTIMATED ANNUAL EMISSIONS FOR TYPICAL DRUM MIX PLANT SILO FILLING OPERATIONS[a]

Pollutant	Emissions, lb/yr
Criteria Pollutants	
PM-10	120
VOC	2,400
CO	240
PAHs (semi-volatile HAPs)	
Acenaphthene	0.24
Acenaphthylene	0.0071
Anthracene	0.066
Benzo(a)anthracene	0.028
Benzo(e)pyrene	0.0048
Chrysene	0.11
Fluoranthene	0.076
Fluorene	0.51
2-Methylnaphthalene	2.7
Naphthalene	0.92
Perylene	0.015
Phenanthrene	0.91
Pyrene	0.22
Total PAHs	5.8
Other semi-volatile HAPs	
Phenol	0.00
Volatile HAPs	
Benzene	0.78
Bromomethane	0.12
2-Butanone	0.95
Carbon disulfide	0.39
Chloroethane	0.095
Chloromethane	0.56
Ethylbenzene	0.93
Formaldehyde	17
n-Hexane	2.4
Isooctane	0.0076
Methylene chloride	0.0066
Styrene	0.13
Toluene	1.5
m-/p-Xylene	4.6
o-Xylene	1.4
Total volatile HAPs	31

[a] Uncontrolled emissions from 200,000 tons of hot mix asphalt per year.

TABLE 11. ESTIMATED ANNUAL EMISSIONS FOR TYPICAL DRUM MIX PLANT ASPHALT STORAGE TANK[a]

Pollutant	Emissions, lb/yr
Criteria Pollutants	
PM-10	ND
VOC	64
CO	6
PAHs (semi-volatile HAPs)	
Acenaphthene	0.0027
Acenaphthylene	0.0010
Anthracene	0.00092
Benzo(b)fluoranthene	0.00051
Fluoranthene	0.00022
Fluorene	0.00016
Naphthalene	0.087
Phenanthrene	0.025
Pyrene	0.00016
Total PAHs	0.12
Volatile HAPs	
Benzene	0.020
Bromomethane	0.0031
2-Butanone	0.025
Carbon disulfide	0.010
Chloroethane	0.0025
Chloromethane	0.015
Ethylbenzene	0.024
Formaldehyde	140
n-Hexane	0.064
Isooctane	0.00020
Methylene chloride	0.00017
Phenol	0.00
Styrene	0.0035
Toluene	0.040
m-/p-Xylene	0.12
o-Xylene	0.036
Total volatile HAPs	140

[a] Uncontrolled emissions from plant producing 200,000 tons of hot mix asphalt per year. Includes emissions from an oil-fired hot oil heater. All of the calculated PAH emissions and almost all of the formaldehyde emissions are from the oil-fired hot oil heater.

TABLE 12. ESTIMATED ANNUAL YARD VOC EMISSIONS FOR TYPICAL BATCH MIX AND DRUM MIX HMA PLANTS[a]

	Batch mix[b]	Drum mix[c]
Pollutant	Emissions, lb/yr	
Criteria Pollutants		
PM-10	ND	ND
VOC	110	220
CO	36	72
PAHs (semi-volatile HAPs)	ND	ND
Other semi-volatile HAPs		
Phenol	0.00	0.00
Volatile HAPs		
Benzene	0.057	0.11
Bromomethane	0.011	0.021
2-Butanone	0.054	0.11
Carbon disulfide	0.014	0.029
Chloroethane	0.00023	0.0046
Chloromethane	0.017	0.033
Cumene	0.12	0.24
Ethylbenzene	0.31	0.62
Formaldehyde	0.10	0.19
n-Hexane	0.17	0.33
Isooctane	0.0020	0.0040
Methylene chloride	0.00	0.00
Styrene	0.0080	0.016
Tetrachloroethene	0.0085	0.017
Toluene	0.23	0.46
Trichlorofluoromethane	0.0014	0.0029
m-/p-Xylene	0.45	0.90
o-Xylene	0.088	0.18
Total volatile HAPs	1.6	3.3

[a] Fugitive VOC emissions from loaded haul truck for eight minutes after completion of load-out.
[b] Uncontrolled emissions from plant producing 100,000 tons of hot mix asphalt per year.
[c] Uncontrolled emissions from plant producing 200,000 tons of hot mix asphalt per year.

AP-42
FIFTH EDITION
JANUARY 1995

COMPILATION OF AIR POLLUTANT EMISSION FACTORS

VOLUME I: STATIONARY POINT AND AREA SOURCES

Office Of Air Quality Planning And Standards
Office Of Air And Radiation
U. S. Environmental Protection Agency
Research Triangle Park, NC 27711

January 1995

11.1 Hot Mix Asphalt Plants

11.1.1 General[1-3,23, 392-394]

Hot mix asphalt (HMA) paving materials are a mixture of size-graded, high quality aggregate (which can include reclaimed asphalt pavement [RAP]), and liquid asphalt cement, which is heated and mixed in measured quantities to produce HMA. Aggregate and RAP (if used) constitute over 92 percent by weight of the total mixture. Aside from the amount and grade of asphalt cement used, mix characteristics are determined by the relative amounts and types of aggregate and RAP used. A certain percentage of fine aggregate (less than 74 micrometers [μm] in physical diameter) is required for the production of good quality HMA.

Hot mix asphalt paving materials can be manufactured by: (1) batch mix plants, (2) continuous mix (mix outside dryer drum) plants, (3) parallel flow drum mix plants, and (4) counterflow drum mix plants. This order of listing generally reflects the chronological order of development and use within the HMA industry.

In 1996, approximately 500 million tons of HMA were produced at the 3,600 (estimated) active asphalt plants in the United States. Of these 3,600 plants, approximately 2,300 are batch plants, 1,000 are parallel flow drum mix plants, and 300 are counterflow drum mix plants. The total 1996 HMA production from batch and drum mix plants is estimated at about 240 million tons and 260 million tons, respectively. About 85 percent of plants being manufactured today are of the counterflow drum mix design, while batch plants and parallel flow drum mix plants account for 10 percent and 5 percent respectively. Continuous mix plants represent a very small fraction of the plants in use ($\leq$0.5 percent) and, therefore, are not discussed further.

An HMA plant can be constructed as a permanent plant, a skid-mounted (easily relocated) plant, or a portable plant. All plants can have RAP processing capabilities. Virtually all plants being manufactured today have RAP processing capability. Most plants have the capability to use either gaseous fuels (natural gas) or fuel oil. However, based upon Department of Energy and limited State inventory information, between 70 and 90 percent of the HMA is produced using natural gas as the fuel to dry and heat the aggregate.

11.1.1.1 Batch Mix Plants –

Figure 11.1-1 shows the batch mix HMA production process. Raw aggregate normally is stockpiled near the production unit. The bulk aggregate moisture content typically stabilizes between 3 to 5 percent by weight.

Processing begins as the aggregate is hauled from the storage piles and is placed in the appropriate hoppers of the cold feed unit. The material is metered from the hoppers onto a conveyer belt and is transported into a rotary dryer (typically gas- or oil-fired). Dryers are equipped with flights designed to shower the aggregate inside the drum to promote drying efficiency.

As the hot aggregate leaves the dryer, it drops into a bucket elevator and is transferred to a set of vibrating screens, where it is classified into as many as four different grades (sizes) and is dropped into individual “hot” bins according to size. At newer facilities, RAP also may be transferred to a separate heated storage bin. To control aggregate size distribution in the final batch mix, the operator opens various hot bins over a weigh hopper until the desired mix and weight are obtained. Concurrent with the aggregate being weighed, liquid asphalt cement is pumped from a heated storage tank to an asphalt bucket, where it is weighed to achieve the desired aggregate-to-asphalt cement ratio in the final mix.

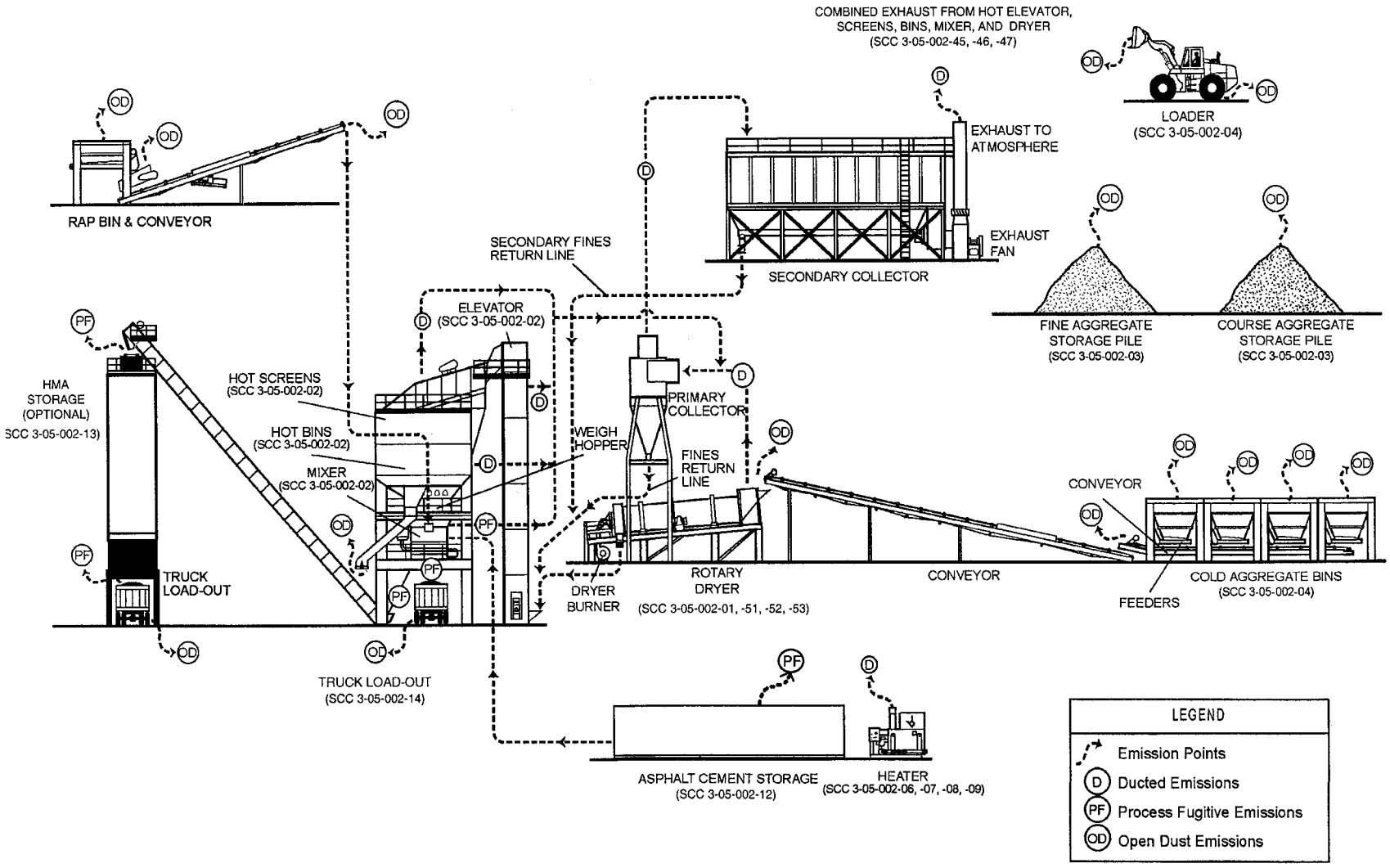


Figure 11.1-1. General process flow diagram for batch mix asphalt plants (source classification codes in parentheses).[3]

The aggregate from the weigh hopper is dropped into the mixer (pug mill) and dry-mixed for 6 to 10 seconds. The liquid asphalt is then dropped into the pug mill where it is mixed for an additional period of time. At older plants, RAP typically is conveyed directly to the pug mill from storage hoppers and combined with the hot aggregate. Total mixing time usually is less than 60 seconds. Then the hot mix is conveyed to a hot storage silo or is dropped directly into a truck and hauled to the job site.

11.1.1.2 Parallel Flow Drum Mix Plants –

Figure 11.1-2 shows the parallel flow drum mix process. This process is a continuous mixing type process, using proportioning cold feed controls for the process materials. The major difference between this process and the batch process is that the dryer is used not only to dry the material but also to mix the heated and dried aggregates with the liquid asphalt cement. Aggregate, which has been proportioned by size gradations, is introduced to the drum at the burner end. As the drum rotates, the aggregates, as well as the combustion products, move toward the other end of the drum in parallel. Liquid asphalt cement flow is controlled by a variable flow pump electronically linked to the new (virgin) aggregate and RAP weigh scales. The asphalt cement is introduced in the mixing zone midway down the drum in a lower temperature zone, along with any RAP and particulate matter (PM) from collectors.

The mixture is discharged at the end of the drum and is conveyed to either a surge bin or HMA storage silos, where it is loaded into transport trucks. The exhaust gases also exit the end of the drum and pass on to the collection system.

Parallel flow drum mixers have an advantage, in that mixing in the discharge end of the drum captures a substantial portion of the aggregate dust, therefore lowering the load on the downstream PM collection equipment. For this reason, most parallel flow drum mixers are followed only by primary collection equipment (usually a baghouse or venturi scrubber). However, because the mixing of aggregate and liquid asphalt cement occurs in the hot combustion product flow, organic emissions (gaseous and liquid aerosol) may be greater than in other asphalt mixing processes. Because data are not available to distinguish significant emissions differences between the two process designs, this effect on emissions cannot be verified.

11.1.1.3 Counterflow Drum Mix Plants –

Figure 11.1-3 shows a counterflow drum mix plant. In this type of plant, the material flow in the drum is opposite or counterflow to the direction of exhaust gases. In addition, the liquid asphalt cement mixing zone is located behind the burner flame zone so as to remove the materials from direct contact with hot exhaust gases.

Liquid asphalt cement flow is controlled by a variable flow pump which is electronically linked to the virgin aggregate and RAP weigh scales. It is injected into the mixing zone along with any RAP and particulate matter from primary and secondary collectors.

Because the liquid asphalt cement, virgin aggregate, and RAP are mixed in a zone removed from the exhaust gas stream, counterflow drum mix plants will likely have organic emissions (gaseous and liquid aerosol) that are lower than parallel flow drum mix plants. However, the available data are insufficient to discern any differences in emissions that result from differences in the two processes. A counterflow drum mix plant can normally process RAP at ratios up to 50 percent with little or no observed effect upon emissions.

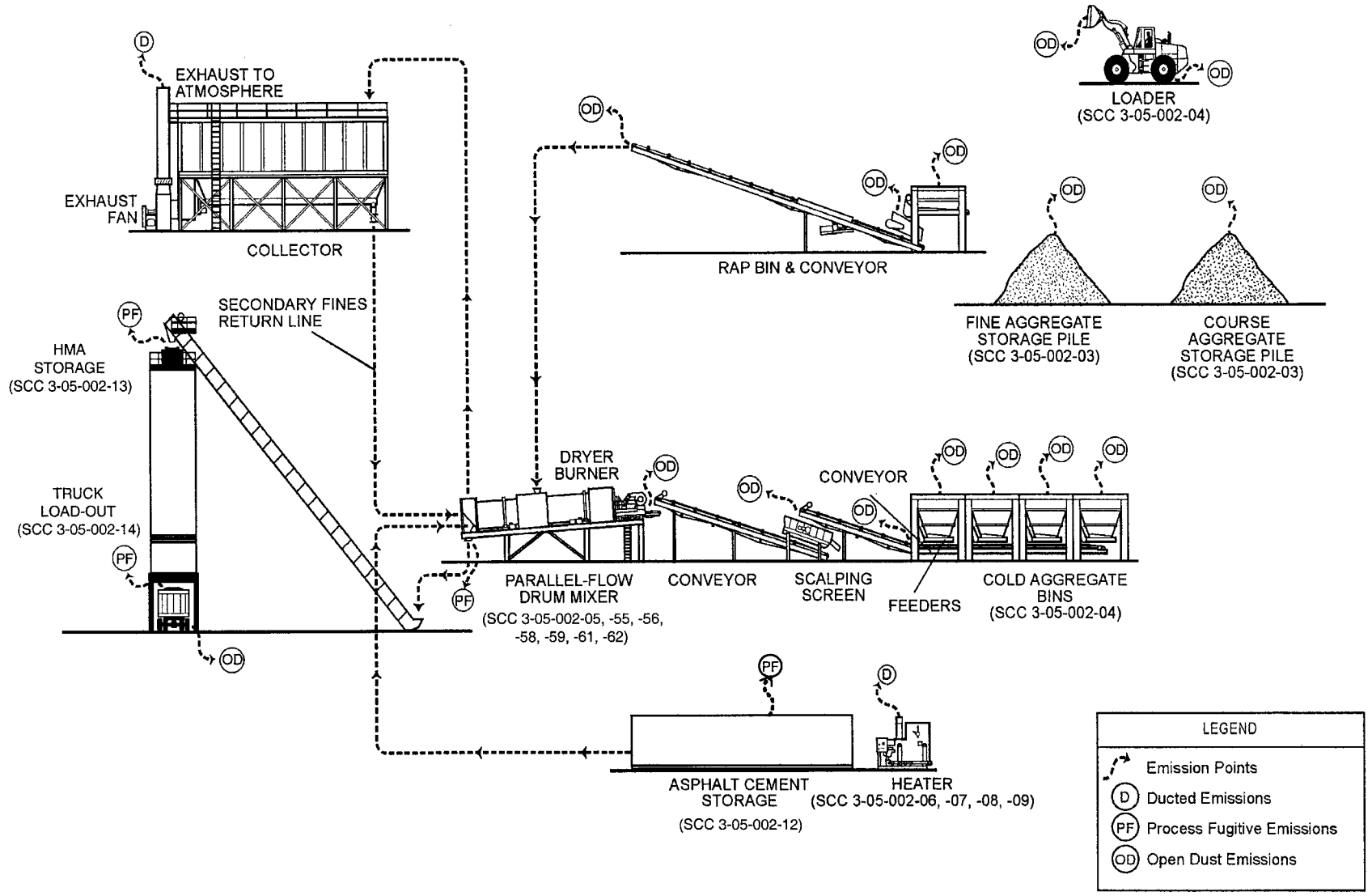


Figure 11.1-2. General process flow diagram for parallel-flow drum mix asphalt plants (source classification codes in parentheses).[3]

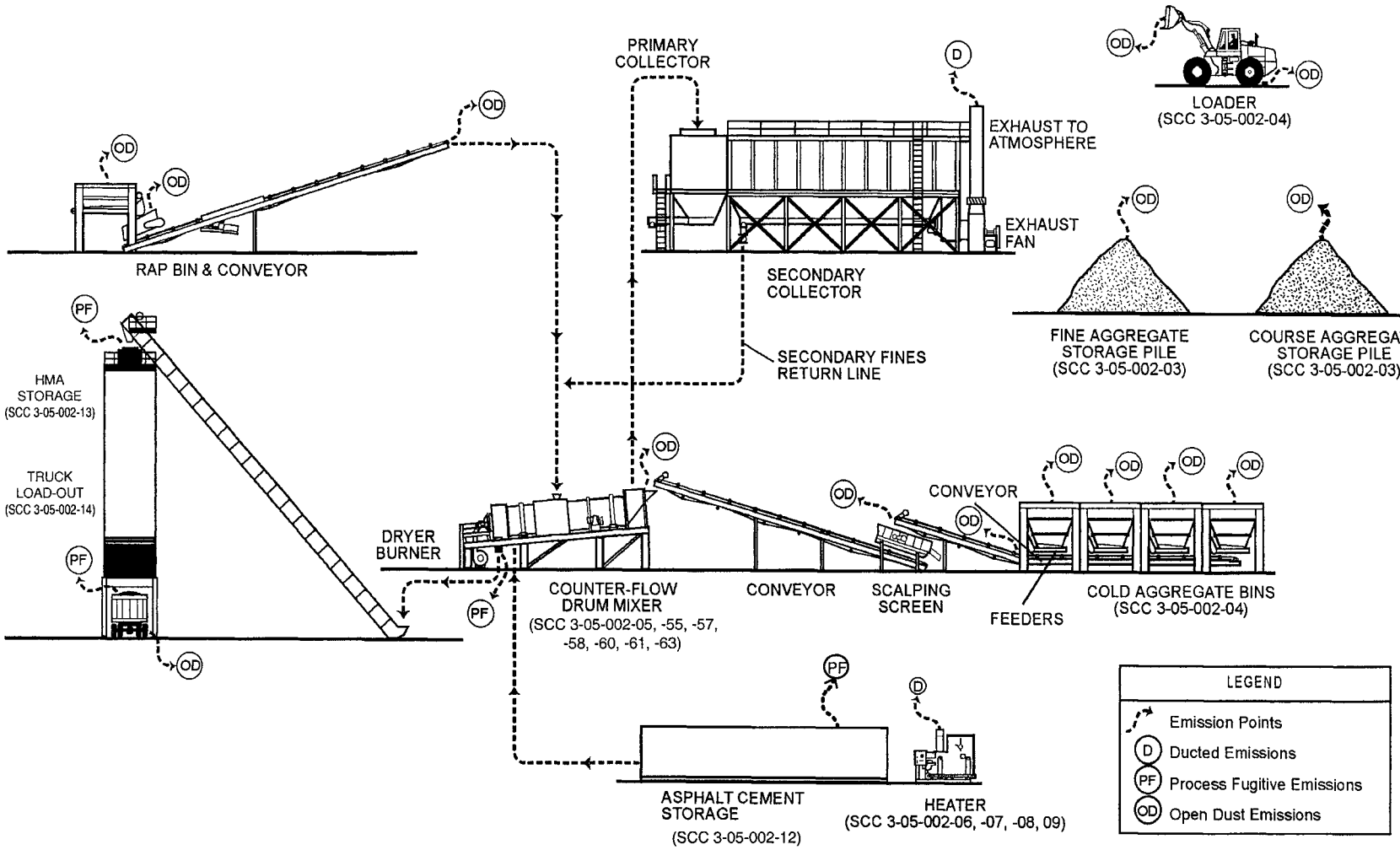


Figure 11.1-3. General process flow diagram for counter-flow drum mix asphalt plants (source classification codes in parentheses).[3]

11.1.1.4 Recycle Processes[393] –

In recent years, the use of RAP has been initiated in the HMA industry. Reclaimed asphalt pavement significantly reduces the amount of virgin rock and asphalt cement needed to produce HMA.

In the reclamation process, old asphalt pavement is removed from the road base. This material is then transported to the plant, and is crushed and screened to the appropriate size for further processing. The paving material is then heated and mixed with new aggregate (if applicable), and the proper amount of new asphalt cement is added to produce HMA that meets the required quality specifications.

11.1.2 Emissions And Controls[2-3,23]

Emissions from HMA plants may be divided into ducted production emissions, pre-production fugitive dust emissions, and other production-related fugitive emissions. Pre-production fugitive dust sources associated with HMA plants include vehicular traffic generating fugitive dust on paved and unpaved roads, aggregate material handling, and other aggregate processing operations. Fugitive dust may range from 0.1 μm to more than 300 μm in aerodynamic diameter. On average, 5 percent of cold aggregate feed is less than 74 μm (minus 200 mesh). Fugitive dust that may escape collection before primary control generally consists of PM with 50 to 70 percent of the total mass less than 74 μm. Uncontrolled PM emission factors for various types of fugitive sources in HMA plants are addressed in Sections 11.19.2, "Crushed Stone Processing", 13.2.1, "Paved Roads", 13.2.2, "Unpaved Roads", 13.2.3, "Heavy Construction Operations", and 13.2.4, "Aggregate Handling and Storage Piles." Production-related fugitive emissions and emissions from ducted production operations are discussed below. Emission points discussed below refer to Figure 11.1-1 for batch mix asphalt plants and to Figures 11.1-2 and 11.1-3 for drum mix plants.

11.1.2.1 Batch Mix Plants –

As with most facilities in the mineral products industry, batch mix HMA plants have two major categories of emissions: ducted sources (those vented to the atmosphere through some type of stack, vent, or pipe), and fugitive sources (those not confined to ducts and vents but emitted directly from the source to the ambient air). Ducted emissions are usually collected and transported by an industrial ventilation system having one or more fans or air movers, eventually to be emitted to the atmosphere through some type of stack. Fugitive emissions result from process and open sources and consist of a combination of gaseous pollutants and PM.

The most significant ducted source of emissions of most pollutants from batch mix HMA plants is the rotary drum dryer. The dryer emissions consist of water (as steam evaporated from the aggregate); PM; products of combustion (carbon dioxide [CO_2], nitrogen oxides [NO_x], and sulfur oxides [SO_x]); carbon monoxide (CO); and small amounts of organic compounds of various species (including volatile organic compounds [VOC], methane [CH_4], and hazardous air pollutants [HAP]). The CO and organic compound emissions result from incomplete combustion of the fuel. It is estimated that between 70 and 90 percent of the energy used at HMA plants is from the combustion of natural gas.

Other potential process sources include the hot-side conveying, classifying, and mixing equipment, which are vented either to the primary dust collector (along with the dryer gas) or to a separate dust collection system. The vents and enclosures that collect emissions from these sources are commonly called "fugitive air" or "scavenger" systems. The scavenger system may or may not have its own separate air mover device, depending on the particular facility. The emissions captured and transported by the scavenger system are mostly aggregate dust, but they may also contain gaseous organic compounds and a fine aerosol of condensed organic particles. This organic aerosol is created by the condensation of vapor into particles during cooling of organic vapors volatilized from the asphalt cement in the mixer (pug mill). The amount of organic aerosol produced depends to a large extent on the temperature of the asphalt cement and aggregate entering the pug mill. Organic vapor and its associated

aerosol also are emitted directly to the atmosphere as process fugitives during truck load-out, from the bed of the truck itself during transport to the job site, and from the asphalt storage tank. Both the low molecular weight organic compounds and the higher weight organic aerosol contain small amounts of HAP. The ducted emissions from the heated asphalt storage tanks include gaseous and aerosol organic compounds and combustion products from the tank heater.

The choice of applicable emission controls for PM emissions from the dryer and vent line includes dry mechanical collectors, scrubbers, and fabric filters. Attempts to apply electrostatic precipitators have met with little success. Practically all plants use primary dust collection equipment such as large diameter cyclones, skimmers, or settling chambers. These chambers often are used as classifiers to return collected material to the hot elevator and to combine it with the drier aggregate. To capture remaining PM, the primary collector effluent is ducted to a secondary collection device. Most plants use either a fabric filter or a venturi scrubber for secondary emissions control. As with any combustion process, the design, operation, and maintenance of the burner provides opportunities to minimize emissions of NO_x, CO, and organic compounds.

11.1.2.2 Parallel Flow Drum Mix Plants –

The most significant ducted source of emissions from parallel-flow drum mix plants is the rotary drum dryer. Emissions from the drum consist of water (as steam evaporated from the aggregate); PM; products of combustion; CO; and small amounts of organic compounds of various species (including VOC, CH_4, and HAP). The organic compound and CO emissions result from incomplete combustion of the fuel and from heating and mixing of the liquid asphalt cement inside the drum. Although it has been suggested that the processing of RAP materials at these type plants may increase organic compound emissions because of an increase in mixing zone temperature during processing, the data supporting this hypothesis are very weak. Specifically, although the data show a relationship only between RAP content and condensible organic particulate emissions, 89 percent of the variations in the data were the result of other unknown process variables.

Once the organic compounds cool after discharge from the process stack, some condense to form a fine organic aerosol or “blue smoke” plume. A number of process modifications or restrictions have been introduced to reduce blue smoke, including installation of flame shields, rearrangement of flights inside the drum, adjustments of the asphalt injection point, and other design changes.

11.1.2.3 Counterflow Drum Mix Plants –

The most significant ducted source of emissions from counterflow drum mix plants is the rotary drum dryer. Emissions from the drum consist of water (as steam evaporated from the aggregate); PM; products of combustion; CO; and small amounts of organic compounds of various species (including VOC, CH_4, and HAP). The CO and organic compound emissions result primarily from incomplete combustion of the fuel, and can also be released from the heated asphalt. Liquid asphalt cement, aggregate, and sometimes RAP, are mixed in a zone not in contact with the hot exhaust gas stream. As a result, kiln stack emissions of organic compounds from counterflow drum mix plants may be lower than parallel flow drum mix plants. However, variations in the emissions due to other unknown process variables are more significant. As a result, the emission factors for parallel flow and counterflow drum mix plants are the same.

11.1.2.4 Parallel and Counterflow Drum Mix Plants –

Process fugitive emissions associated with batch plant hot screens, elevators, and the mixer (pug mill) are not present in the drum mix processes. However, there are fugitive PM and VOC emissions from transport and handling of the HMA from the drum mixer to the storage silo and also from the load-out operations to the delivery trucks. Since the drum process is continuous, these plants have surge

bins or storage silos. The fugitive dust sources associated with drum mix plants are similar to those of batch mix plants with regard to truck traffic and to aggregate material feed and handling operations.

Table 11.1-1 presents emission factors for filterable PM and PM-10, condensable PM, and total PM for batch mix HMA plants. Particle size data for batch mix HMA plants, based on the control technology used, are shown in Table 11.1-2. Table 11.1-3 presents filterable PM and PM-10, condensable PM, and total PM emission factors for drum mix HMA plants. Particle size data for drum mix HMA plants, based on the control technology used, are shown in Table 11.1-4. Tables 11.1-5 and -6 present emission factors for CO, CO_2, NO_x, sulfur dioxide (SO_2), total organic compounds (TOC), formaldehyde, CH_4, and VOC from batch mix plants. Tables 11.1-7 and -8 present emission factors for CO, CO_2, NO_x, SO_2, TOC, CH_4, VOC, and hydrochloric acid (HCl) from drum mix plants. The emission factors for CO, NO_x, and organic compounds represent normal plant operations without scrutiny of the burner design, operation, and maintenance. Information provided in Reference 390 indicates that attention to burner design, periodic evaluation of burner operation, and appropriate maintenance can reduce these emissions. Table 11.1-9 presents organic pollutant emission factors for batch mix plants. Table 11.1-10 presents organic pollutant emission factors for drum mix plants. Tables 11.1-11 and -12 present metals emission factors for batch and drum mix plants, respectively. Table 11.1-13 presents organic pollutant emission factors for hot (asphalt) oil systems.

11.1.2.5 Fugitive Emissions from Production Operations –

Emission factors for HMA load-out and silo filling operations can be estimated using the data in Tables 11.1-14, -15, and -16. Table 11.1-14 presents predictive emission factor equations for HMA load-out and silo filling operations. Separate equations are presented for total PM, extractable organic PM (as measured by EPA Method 315), TOC, and CO. For example, to estimate total PM emissions from drum mix or batch mix plant load-out operations using an asphalt loss-on-heating of 0.41 percent and temperature of 290°F, the following calculation is made:

$$
\begin{aligned}
EF &= 0.000181 + 0.00141(-V)e^{((0.0251)(290 + 460) - 20.43)} \\
&= 0.000181 + 0.00141(-(-0.41))e^{((0.0251)(290 + 460) - 20.43)} \\
&= 0.000181 + 0.00141(0.41)e^{(-1.605)} \\
&= 0.000181 + 0.00141(0.41)(0.2009) \\
&= 0.000181 + 0.000116 \\
&= 0.00030 \text{ lb total PM/ton of asphalt loaded}
\end{aligned}
$$

Tables 11.1-15 and -16 present speciation profiles for organic particulate-based and volatile particulate-based compounds, respectively. The speciation profile shown in Table 11.1-15 can be applied to the extractable organic PM emission factors estimated by the equations in Table 11.1-14 to estimate emission factors for specific organic PM compounds. The speciation profile presented in Table 11.1-16 can be applied to the TOC emission factors estimated by the equations in Table 11.1-14 to estimate emission factors for specific volatile organic compounds. The derivations of the predictive emission factor equations and the speciation profiles can be found in Reference 1.

For example, to estimate TOC emissions from drum mix plant load-out operations using an asphalt loss-on-heating of 0.41 percent and temperature of 290°F, the following calculation is made:

$$
\begin{aligned}
EF &= 0.0172(-V)e^{((0.0251)(290 + 460) - 20.43)} \\
&= 0.0172(-(-0.41))e^{((0.0251)(290 + 460) - 20.43)} \\
&= 0.0172(0.41)e^{(-1.605)} \\
&= 0.0172(0.41)(0.2009) \\
&= 0.0014 \text{ lb TOC/ton of asphalt loaded}
\end{aligned}
$$

To estimate the benzene emissions from the same operation, use the TOC emission factor calculated above and apply the benzene fraction for load-out emissions from Table 11.1-16:

EF = 0.0014 (0.00052)
= 7.3 x 10^{-7} lb benzene/ton of asphalt loaded

Emissions from asphalt storage tanks can be estimated using the procedures described in AP-42 Section 7.1, Organic Liquid Storage Tanks, and the TANKS software. Site-specific data should be used for storage tank specifications and operating parameters, such as temperature. If site-specific data for Antoine's constants for an average asphalt binder used by the facility are unavailable, the following values for an average liquid asphalt binder can be used:

A = 75,350.06
B = 9.00346

These values should be inserted into the Antoine's equation in the following form:

$$\log_{10} P = \frac{-0.05223A}{T} + B$$

where:

P = vapor pressure, mm Hg
T = absolute temperature, Kelvin

The assumed average liquid molecular weight associated with these Antoine's constants is 1,000 atomic mass units and the average vapor molecular weight is 105. Emission factors estimated using these default values should be assigned a rating of E. Carbon monoxide emissions can be estimated by multiplying the THC emissions calculated by the TANKS program by 0.097 (the ratio of silo filling CO emissions to silo filling TOC emissions).

Vapors from the HMA loaded into transport trucks continue following load-out operations. The TOC emissions for the 8-minute period immediately following load-out (yard emissions) can be estimated using an emission factor of 0.00055 kg/Mg (0.0011 lb/ton) of asphalt loaded. This factor is assigned a rating of E. The derivation of this emission factor is described in Reference 1. Carbon monoxide emissions can be estimated by multiplying the TOC emissions by 0.32 (the ratio of truck load-out CO emissions to truck load-out THC emissions).

11.2.3 Updates Since the Fifth Edition

The Fifth Edition was released in January 1995. Revisions to this section since that date are summarized below. For further detail, consult the background report for this section. This and other documents can be found on the CHIEF Web Site at http://www.epa.gov/ttn/chief/, or by calling the Info CHIEF Help Desk at (919)541-1000.

December 2000

- All emission factors were revised and new factors were added. For selected pollutant emissions, separate factors were developed for distilate oil, No. 6 oil and waste oil fired dryers. Dioxin and Furan emission factors were developed for oil fired drum mix plants. Particulate, VOC and CO factors were developed for silo filling, truck load out and post truck load out operations at batch plants and drum mix plants. Organic species profiles were developed for silo filling, truck load out and post truck load out operations.

March 2004

- The emission factor for formaldehyde for oil fired hot oil heaters was revised. An emission factor for formaldehyde for gas fired hot oil heaters and emission factors for CO and CO_2 for gas and oil fired hot oil heaters were developed. (Table 11.1-13)

Table 11.1-9. EMISSION FACTORS FOR ORGANIC POLLUTANT EMISSIONS FROM BATCH MIX HOT MIX ASPHALT PLANTS[a]

Process	Pollutant		Emission Factor, lb/ton	Emission Factor Rating	Ref. Nos.
	CASRN	Name			
Natural gas- or No. 2 fuel oil-fired dryer, hot screens, and mixer with fabric filter (SCC 3-05-002-45,-46)	Non-PAH Hazardous Air Pollutants[b]				
	75-07-0	Acetaldehyde	0.00032	E	24,34
	71-43-2	Benzene	0.00028	D	24,34,46, 382
	100-41-4	Ethylbenzene	0.0022	D	24,46,47,49
	50-00-0	Formaldehyde	0.00074	D	24,34,46,47,49,226,382
	106-51-4	Quinone	0.00027	E	24
	108-88-3	Toluene	0.0010	D	24,34,46,47
	1330-20-7	Xylene	0.0027	D	24,46,47,49
		Total non-PAH HAPs	0.0075		
	PAH HAPs				
	91-57-6	2-Methylnaphthalene[c]	7.1×10^{-5}	D	24,47,49
	83-32-9	Acenaphthene[c]	9.0×10^{-7}	D	34,46,226
	208-96-8	Acenaphthylene[c]	5.8×10^{-7}	D	34,46,226
	120-12-7	Anthracene[c]	2.1×10^{-7}	D	34,46,226
	56-55-3	Benzo(a)anthracene[c]	4.6×10^{-9}	E	46,226
	50-32-8	Benzo(a)pyrene[c]	3.1×10^{-10}	E	226
	205-99-2	Benzo(b)fluoranthene[c]	9.4×10^{-9}	D	34,46,226
	191-24-2	Benzo(g,h,i)perylene[c]	5.0×10^{-10}	E	226
	207-08-9	Benzo(k)fluoranthene[c]	1.3×10^{-8}	E	34,226
	218-01-9	Chrysene[c]	3.8×10^{-9}	E	46,226
	53-70-3	Dibenz(a,h)anthracene[c]	9.5×10^{-11}	E	226
	206-44-0	Fluoranthene[c]	1.6×10^{-7}	D	34,46,47,226
	86-73-7	Fluorene[c]	1.6×10^{-6}	D	34,46,47,226
	193-39-5	Indeno(1,2,3-cd)pyrene[c]	3.0×10^{-10}	E	226
	91-20-3	Naphthalene	3.6×10^{-5}	D	34,46,47,49,226
	85-01-8	Phenanthrene[c]	2.6×10^{-6}	D	34,46,47,226
	129-00-0	Pyrene[c]	6.2×10^{-8}	D	34,46,226
		Total PAH HAPs	0.00011		
	Total HAPs		0.0076		
	Non-HAP organic compounds				
	100-52-7	Benzaldehyde	0.00013	E	24
	78-84-2	Butyraldehyde/ isobutyraldehyde	3.0×10^{-5}	E	24
	4170-30-3	Crotonaldehyde	2.9×10^{-5}	E	24
	66-25-1	Hexanal	2.4×10^{-5}	E	24
		Total non-HAPs	0.00019		

Table 11.1-9 (cont.)

Process	Pollutant CASRN	Pollutant Name	Emission Factor, lb/ton	Emission Factor Rating	Ref. Nos.
Waste oil-, drain oil-, or No. 6 fuel oil-fired dryer, hot screens, and mixer with fabric filter (SCC 3-05-002-47)	Non-PAH Hazardous Air Pollutants[b]				
	75-07-0	Acetaldehyde	0.00032	E	24,34
	71-43-2	Benzene	0.00028	D	24,34,46, 382
	100-41-4	Ethylbenzene	0.0022	D	24,46,47,49
	50-00-0	Formaldehyde	0.00074	D	24,34,46,47,49,226, 382
	106-51-4	Quinone	0.00027	E	24
	108-88-3	Toluene	0.0010	D	24,34,46,47
	1330-20-7	Xylene	0.0027	D	24,46,47,49
		Total non-PAH HAPs	0.0075		
	PAH HAPs[b]				
	91-57-6	2-Methylnaphthalene[c]	7.1×10^{-5}	D	24,47,49
	83-32-9	Acenaphthene[c]	9.0×10^{-7}	D	34,46,226
	208-96-8	Acenaphthylene[c]	5.8×10^{-7}	D	34,46,226
	120-12-7	Anthracene[c]	2.1×10^{-7}	D	34,46,226
	56-55-3	Benzo(a)anthracene[c]	4.6×10^{-9}	E	46,226
	50-32-8	Benzo(a)pyrene[c]	3.1×10^{-10}	E	226
	205-99-2	Benzo(b)fluoranthene[c]	9.4×10^{-9}	D	34,46,226
	191-24-2	Benzo(g,h,i)perylene[c]	5.0×10^{-10}	E	226
	207-08-9	Benzo(k)fluoranthene[c]	1.3×10^{-8}	E	34,226
	218-01-9	Chrysene[c]	3.8×10^{-9}	E	46,226
	53-70-3	Dibenz(a,h)anthracene[c]	9.5×10^{-11}	E	226
	206-44-0	Fluoranthene[c]	2.4×10^{-5}	E	49
	86-73-7	Fluorene[c]	1.6×10^{-6}	D	34,46,47,226
	193-39-5	Indeno(1,2,3-cd)pyrene[c]	3.0×10^{-10}	E	226
	91-20-3	Naphthalene	3.6×10^{-5}	D	34,46,47,49, 226
	85-01-8	Phenanthrene[c]	3.7×10^{-5}	E	49
	129-00-0	Pyrene[c]	5.5×10^{-5}	E	49
		Total PAH HAPs	0.00023		
	Total HAPs		0.0077		
	Non-HAP organic compounds				
	100-52-7	Benzaldehyde	0.00013	E	24
	78-84-2	Butyraldehyde/ isobutyraldehyde	3.0×10^{-5}	E	24
	4170-30-3	Crotonaldehyde	2.9×10^{-5}	E	24
	66-25-1	Hexanal	2.4×10^{-5}	E	24
		Total non-HAPs	0.00019		

[a] Emission factor units are lb/ton of hot mix asphalt produced. Factors represent uncontrolled emissions, unless noted. CASRN = Chemical Abstracts Service Registry Number. SCC = Source Classification Code. To convert from lb/ton to kg/Mg, multiply by 0.5.

[b] Hazardous air pollutants (HAP) as defined in the 1990 Clean Air Act Amendments (CAAA).

[c] Compound is classified as polycyclic organic matter, as defined in the 1990 CAAA.

Table 11.1-10. EMISSION FACTORS FOR ORGANIC POLLUTANT EMISSIONS FROM DRUM MIX HOT MIX ASPHALT PLANTS[a]

Process	Pollutant		Emission Factor, lb/ton	Emission Factor Rating	Ref. No.
	CASRN	Name			
Natural gas-fired dryer with fabric filter[b] (SCC 3-05-002-55, -56,-57)	Non-PAH hazardous air pollutants[c]				
	71-43-2	Benzene[d]	0.00039	A	25,44,45,50, 341, 342, 344-351, 373, 376, 377, 383, 384
	100-41-4	Ethylbenzene	0.00024	D	25,44,45
	50-00-0	Formaldehyde[e]	0.0031	A	25,35,44,45,50, 339-344, 347-349, 371-373, 384, 388
	110-54-3	Hexane	0.00092	E	339-340
	540-84-1	Isooctane (2,2,4-trimethylpentane)	4.0×10^{-5}	E	339-340
	71-55-6	Methyl chloroform[f]	4.8×10^{-5}	E	35
	108-88-3	Toluene	0.00015	D	35,44,45
	1330-20-7	Xylene	0.00020	D	25,44,45
		Total non-PAH HAPs	0.0051		
	PAH HAPs				
	91-57-6	2-Methylnaphthalene[g]	7.4×10^{-5}	D	44,45,48
	83-32-9	Acenaphthene[g]	1.4×10^{-6}	E	48
	208-96-8	Acenaphthylene[g]	8.6×10^{-6}	D	35,45,48
	120-12-7	Anthracene[g]	2.2×10^{-7}	E	35,48
	56-55-3	Benzo(a)anthracene[g]	2.1×10^{-7}	E	48
	50-32-8	Benzo(a)pyrene[g]	9.8×10^{-9}	E	48
	205-99-2	Benzo(b)fluoranthene[g]	1.0×10^{-7}	E	35,48
	192-97-2	Benzo(e)pyrene[g]	1.1×10^{-7}	E	48
	191-24-2	Benzo(g,h,i)perylene[g]	4.0×10^{-8}	E	48
	207-08-9	Benzo(k)fluoranthene[g]	4.1×10^{-8}	E	35,48
	218-01-9	Chrysene[g]	1.8×10^{-7}	E	35,48
	206-44-0	Fluoranthene[g]	6.1×10^{-7}	D	35,45,48
	86-73-7	Fluorene[g]	3.8×10^{-6}	D	35,45,48,163
	193-39-5	Indeno(1,2,3-cd)pyrene[g]	7.0×10^{-9}	E	48
	91-20-3	Naphthalene[g]	9.0×10^{-5}	D	35,44,45,48,163
	198-55-0	Perylene[g]	8.8×10^{-9}	E	48
	85-01-8	Phenanthrene[g]	7.6×10^{-6}	D	35,44,45,48,163
	129-00-0	Pyrene[g]	5.4×10^{-7}	D	45,48
		Total PAH HAPs	0.00019		

Table 11.1-10 (cont.)

Process	Pollutant CASRN	Pollutant Name	Emission Factor, lb/ton	Emission Factor Rating	Ref. No.
Natural gas-fired dryer with fabric filter[b] (SCC 3-05-002-55, -56,-57) (cont.)	Total HAPs		0.0053		
	Non-HAP organic compounds				
	106-97-8	Butane	0.00067	E	339
	74-85-1	Ethylene	0.0070	E	339-340
	142-82-5	Heptane	0.0094	E	339-340
	763-29-1	2-Methyl-1-pentene	0.0040	E	339,340
	513-35-9	2-Methyl-2-butene	0.00058	E	339,340
	96-14-0	3-Methylpentane	0.00019	D	339,340
	109-67-1	1-Pentene	0.0022	E	339-340
	109-66-0	n-Pentane	0.00021	E	339-340
		Total non-HAP organics	0.024		
No. 2 fuel oil-fired dryer with fabric filter (SCC 3-05-002-58, -59,-60)	Non-PAH HAPs[c]				
	71-43-2	Benzene[d]	0.00039	A	25,44,45,50, 341, 342, 344-351, 373, 376, 377, 383, 384
	100-41-4	Ethylbenzene	0.00024	D	25,44,45
	50-00-0	Formaldehyde[e]	0.0031	A	25,35,44,45,50, 339-344, 347-349, 371-373, 384, 388
	110-54-3	Hexane	0.00092	E	339-340
	540-84-1	Isooctane (2,2,4-trimethylpentane)	4.0×10^{-5}	E	339-340
	71-55-6	Methyl chloroform[f]	4.8×10^{-5}	E	35
	108-88-3	Toluene	0.0029	E	25, 50, 339-340
	1330-20-7	Xylene	0.00020	D	25,44,45
		Total non-PAH HAPs	0.0078		
	PAH HAPs				
	91-57-6	2-Methylnaphthalene[g]	0.00017	E	50
	83-32-9	Acenaphthene[g]	1.4×10^{-6}	E	48
	208-96-8	Acenaphthylene[g]	2.2×10^{-5}	E	50
	120-12-7	Anthracene[g]	3.1×10^{-6}	E	50,162
	56-55-3	Benzo(a)anthracene[g]	2.1×10^{-7}	E	48
	50-32-8	Benzo(a)pyrene[g]	9.8×10^{-9}	E	48
	205-99-2	Benzo(b)fluoranthene[g]	1.0×10^{-7}	E	35,48
	192-97-2	Benzo(e)pyrene[g]	1.1×10^{-7}	E	48

Table 11.1-10 (cont.)

Process	Pollutant CASRN	Pollutant Name	Emission Factor, lb/ton	Emission Factor Rating	Ref. No.
No. 2 fuel oil-fired dryer with fabric filter (SCC 3-05-002-58, -59,-60) (cont.)	191-24-2	Benzo(g,h,i)perylene[g]	4.0×10^{-8}	E	48
	207-08-9	Benzo(k)fluoranthene[g]	4.1×10^{-8}	E	35,48
	218-01-9	Chrysene[g]	1.8×10^{-7}	E	35,48
	206-44-0	Fluoranthene[g]	6.1×10^{-7}	D	35,45,48
	86-73-7	Fluorene[g]	1.1×10^{-5}	E	50,164
	193-39-5	Indeno(1,2,3-cd)pyrene[g]	7.0×10^{-9}	E	48
	91-20-3	Naphthalene[g]	0.00065	D	25,50,162,164
	198-55-0	Perylene[g]	8.8×10^{-9}	E	48
	85-01-8	Phenanthrene[g]	2.3×10^{-5}	D	50,162,164
	129-00-0	Pyrene[g]	3.0×10^{-6}	E	50
		Total PAH HAPs	0.00088		
		Total HAPs	0.0087		
	Non-HAP organic compounds				
	106-97-8	Butane	0.00067	E	339
	74-85-1	Ethylene	0.0070	E	339-340
	142-82-5	Heptane	0.0094	E	339-340
	763-29-1	2-Methyl-1-pentene	0.0040	E	339,340
	513-35-9	2-Methyl-2-butene	0.00058	E	339,340
	96-14-0	3-Methylpentane	0.00019	D	339,340
	109-67-1	1-Pentene	0.0022	E	339-340
	109-66-0	n-Pentane	0.00021	E	339-340
		Total non-HAP organics	0.024		

Table 11.1-10 (cont.)

Process	Pollutant		Emission Factor, lb/ton	Emission Factor Rating	Ref. No.
	CASRN	Name			
Fuel oil- or waste oil-fired dryer with fabric filter (SCC 3-05-002-58, -59,-60,-61,-62, -63)	Dioxins				
	1746-01-6	2,3,7,8-TCDD[g]	$2.1x10^{-13}$	E	339
		Total TCDD[g]	$9.3x10^{-13}$	E	339
	40321-76-4	1,2,3,7,8-PeCDD[g]	$3.1x10^{-13}$	E	339
		Total PeCDD[g]	$2.2x10^{-11}$	E	339-340
	39227-28-6	1,2,3,4,7,8-HxCDD[g]	$4.2x10^{-13}$	E	339
	57653-85-7	1,2,3,6,7,8-HxCDD[g]	$1.3x10^{-12}$	E	339
	19408-24-3	1,2,3,7,8,9-HxCDD[g]	$9.8x10^{-13}$	E	339
		Total HxCDD[g]	$1.2x10^{-11}$	E	339-340
	35822-46-9	1,2,3,4,6,7,8-HpCDD[g]	$4.8x10^{-12}$	E	339
		Total HpCDD[g]	$1.9x10^{-11}$	E	339-340
	3268-87-9	Octa CDD[g]	$2.5x10^{-11}$	E	339
		Total PCDD[g]	$7.9x10^{-11}$	E	339-340
	Furans				
	51207-31-9	2,3,7,8-TCDF[g]	$9.7x10^{-13}$	E	339
		Total TCDF[g]	$3.7x10^{-12}$	E	339-340
		1,2,3,7,8-PeCDF[g]	$4.3x10^{-12}$	E	339-340
		2,3,4,7,8-PeCDF[g]	$8.4x10^{-13}$	E	339
		Total PeCDF[g]	$8.4x10^{-11}$	E	339-340
		1,2,3,4,7,8-HxCDF[g]	$4.0x10^{-12}$	E	339
		1,2,3,6,7,8-HxCDF[g]	$1.2x10^{-12}$	E	339
		2,3,4,6,7,8-HxCDF[g]	$1.9x10^{-12}$	E	339
		1,2,3,7,8,9-HxCDF[g]	$8.4x10^{-12}$	E	340
		Total HxCDF[g]	$1.3x10^{-11}$	E	339-340
		1,2,3,4,6,7,8-HpCDF[g]	$6.5x10^{-12}$	E	339
		1,2,3,4,7,8,9-HpCDF[g]	$2.7x10^{-12}$	E	339
		Total HpCDF[g]	$1.0x10^{-11}$	E	339-340
	39001-02-0	Octa CDF[g]	$4.8x10^{-12}$	E	339
		Total PCDF[g]	$4.0x10^{-11}$	E	339-340
		Total PCDD/PCDF[g]	$1.2x10^{-10}$	E	339-340

Table 11.1-10 (cont.)

Process	Pollutant CASRN	Pollutant Name	Emission Factor, lb/ton	Emission Factor Rating	Ref. No.
Fuel oil- or waste oil-fired dryer (uncontrolled) (SCC 3-05-002-58, -59,-60,-61,-62, -63)	Hazardous air pollutants[c]				
	Dioxins				
		Total HxCDD[g]	5.4×10^{-12}	E	340
	35822-46-9	1,2,3,4,6,7,8-HpCDD[g]	3.4×10^{-11}	E	340
		Total HpCDD[g]	7.1×10^{-11}	E	340
	3268-87-9	Octa CDD[g]	2.7×10^{-9}	E	340
		Total PCDD[g]	2.8×10^{-9}	E	340
	Furans				
		Total TCDF[g]	3.3×10^{-11}	E	340
		Total PeCDF[g]	7.4×10^{-11}	E	340
		1,2,3,4,7,8-HxCDF[g]	5.4×10^{-12}	E	340
		2,3,4,6,7,8-HxCDF[g]	1.6×10^{-12}	E	340
		Total HxCDF[g]	8.1×10^{-12}	E	340
Fuel oil- or waste oil-fired dryer (uncontrolled) (SCC 3-05-002-58, -59,-60,-61,-62, -63) (cont.)		1,2,3,4,6,7,8-HpCDF[g]	1.1×10^{-11}	E	340
		Total HpCDF[g]	3.8×10^{-11}	E	340
		Total PCDF[g]	1.5×10^{-10}	E	340
		Total PCDD/PCDF[g]	3.0×10^{-9}	E	340

Table 11.1-10 (cont.)

Process	Pollutant		Emission Factor, lb/ton	Emission Factor Rating	Ref. No.
	CASRN	Name			
Waste oil-fired dryer with fabric filter (SCC 3-05-002-61, -62,-63)	Non-PAH HAPs[c]				
	75-07-0	Acetaldehyde	0.0013	E	25
	107-02-8	Acrolein	$2.6x10^{-5}$	E	25
	71-43-2	Benzene[d]	0.00039	A	25,44,45,50,341,342, 344-351, 373, 376, 377, 383, 384
	100-41-4	Ethylbenzene	0.00024	D	25,44,45
	50-00-0	Formaldehyde[e]	0.0031	A	25,35,44,45,50,339-344,347-349,371-373, 384, 388
	110-54-3	Hexane	0.00092	E	339-340
	540-84-1	Isooctane (2,2,4-trimethylpentane)	$4.0x10^{-5}$	E	339-340
	78-93-3	Methyl Ethyl Ketone	$2.0x10^{-5}$	E	25
	123-38-6	Propionaldehyde	0.00013	E	25
	106-51-4	Quinone	0.00016	E	25
	71-55-6	Methyl chloroform[f]	$4.8x10^{-5}$	E	35
	108-88-3	Toluene	0.0029	E	25, 50, 339-340
	1330-20-7	Xylene	0.00020	D	25,44,45
		Total non-PAH HAPs	0.0095		
	PAH HAPs				
	91-57-6	2-Methylnaphthalene[g]	0.00017	E	50
	83-32-9	Acenaphthene[g]	$1.4x10^{-6}$	E	48
	208-96-8	Acenaphthylene[g]	$2.2x10^{-5}$	E	50
	120-12-7	Anthracene[g]	$3.1x10^{-6}$	E	50,162
	56-55-3	Benzo(a)anthracene[g]	$2.1x10^{-7}$	E	48
	50-32-8	Benzo(a)pyrene[g]	$9.8x10^{-9}$	E	48
	205-99-2	Benzo(b)fluoranthene[g]	$1.0x10^{-7}$	E	35,48
	192-97-2	Benzo(e)pyrene[g]	$1.1x10^{-7}$	E	48
	191-24-2	Benzo(g,h,i)perylene[g]	$4.0x10^{-8}$	E	48

Table 11.1-10 (cont.)

Process	Pollutant CASRN	Pollutant Name	Emission Factor, lb/ton	Emission Factor Rating	Ref. No.
Waste oil-fired dryer with fabric filter (SCC 3-05-002-61, -62,-63) (cont.)	207-08-9	Benzo(k)fluoranthene[g]	$4.1x10^{-8}$	E	35,48
	218-01-9	Chrysene[g]	$1.8x10^{-7}$	E	35,48
	206-44-0	Fluoranthene[g]	$6.1x10^{-7}$	D	35,45,48
	86-73-7	Fluorene[g]	$1.1x10^{-5}$	E	50,164
	193-39-5	Indeno(1,2,3-cd)pyrene[g]	$7.0x10^{-9}$	E	48
	91-20-3	Naphthalene[g]	0.00065	D	25,50,162,164
	198-55-0	Perylene[g]	$8.8x10^{-9}$	E	48
	85-01-8	Phenanthrene[g]	$2.3x10^{-5}$	D	50,162,164
	129-00-0	Pyrene[g]	$3.0x10^{-6}$	E	50
		Total PAH HAPs	0.00088		
		Total HAPs	0.010		
	Non-HAP organic compounds				
	67-64-1	Acetone[f]	0.00083	E	25
	100-52-7	Benzaldehyde	0.00011	E	25
	106-97-8	Butane	0.00067	E	339
	78-84-2	Butyraldehyde	0.00016	E	25
	4170-30-3	Crotonaldehyde	$8.6x10^{-5}$	E	25
	74-85-1	Ethylene	0.0070	E	339, 340
	142-82-5	Heptane	0.0094	E	339, 340
	66-25-1	Hexanal	0.00011	E	25
	590-86-3	Isovaleraldehyde	$3.2x10^{-5}$	E	25
	763-29-1	2-Methyl-1-pentene	0.0040	E	339, 340
	513-35-9	2-Methyl-2-butene	0.00058	E	339, 340
	96-14-0	3-Methylpentane	0.00019	D	339, 340
	109-67-1	1-Pentene	0.0022	E	339, 340
	109-66-0	n-Pentane	0.00021	E	339, 340
	110-62-3	Valeraldehyde	$6.7x10^{-5}$	E	25
		Total non-HAP organics	0.026		

[a] Emission factor units are lb/ton of hot mix asphalt produced. Table includes data from both parallel flow and counterflow drum mix dryers. Organic compound emissions from counterflow systems are expected to be less than from parallel flow systems, but the available data are insufficient to quantify

Table 11.1-10 (cont.)

accurately the difference in these emissions. CASRN = Chemical Abstracts Service Registry Number. SCC = Source Classification Code. To convert from lb/ton to kg/Mg, multiply by 0.5.

[b] Tests included dryers that were processing reclaimed asphalt pavement. Because of limited data, the effect of RAP processing on emissions could not be determined.

[c] Hazardous air pollutants (HAP) as defined in the 1990 Clean Air Act Amendments (CAAA).

[d] Based on data from 19 tests. Range: 0.000063 to 0.0012 lb/ton; median: 0.00030; Standard deviation: 0.00031.

[e] Based on data from 21 tests. Range: 0.0030 to 0.014 lb/ton; median: 0.0020; Standard deviation: 0.0036.

[f] Compound has negligible photochemical reactivity.

[g] Compound is classified as polycyclic organic matter, as defined in the 1990 CAAA. Total PCDD is the sum of the total tetra through octa dioxins; total PCDF is sum of the total tetra through octa furans; and total PCDD/PCDF is the sum of total PCDD and total PCDF.

Table 11.1-11. EMISSION FACTORS FOR METAL EMISSIONS FROM BATCH MIX HOT MIX ASPHALT PLANTS[a]

Process	Pollutant	Emission Factor, lb/ton	Emission Factor Rating	Reference Numbers
Dryer, hot screens, and mixer[b] (SCC 3-05-002-45,-46,-47)	Arsenic[c]	4.6×10^{-7}	D	34, 40, 226
	Barium	1.5×10^{-6}	E	24
	Beryllium[c]	1.5×10^{-7}	E	34, 226
	Cadmium[c]	6.1×10^{-7}	D	24, 34, 226
	Chromium[c]	5.7×10^{-7}	D	24, 34, 226
	Hexavalent chromium[c]	4.8×10^{-8}	E	34, 226
	Copper	2.8×10^{-6}	D	24, 34, 226
	Lead[c]	8.9×10^{-7}	D	24, 34, 226
	Manganese[c]	6.9×10^{-6}	D	24, 34, 226
	Mercury[c]	4.1×10^{-7}	E	34, 226
	Nickel[c]	3.0×10^{-6}	D	24, 34, 226
	Selenium[c]	4.9×10^{-7}	E	34, 226
	Zinc	6.8×10^{-6}	D	24, 34, 226

[a] Emission factor units are lb/ton of HMA produced. Emissions controlled by a fabric filter. SCC = Source Classification Code. To convert from lb/ton to kg/Mg, multiply by 0.5.

[b] Natural gas-, propane-, No. 2 fuel oil-, or waste oil-/drain oil-/No. 6 fuel oil-fired dryer. For waste oil-/drain oil-/No. 6 fuel oil-fired dryer, use a lead emission factor of 1.0×10^{-5} lb/ton (References 177 and 321, Emission factor rating: E) in lieu of the emission factor shown.

[c] Arsenic, beryllium, cadmium, chromium, hexavalent chromium, lead, manganese, mercury, nickel, and selenium are HAPs as defined in the 1990 CAAA.

Table 11.1-12. EMISSION FACTORS FOR METAL EMISSIONS FROM DRUM MIX HOT MIX ASPHALT PLANTS[a]

Process	Pollutant	Emission Factor, lb/ton	Emission Factor Rating	Reference Numbers
Fuel oil-fired dryer, uncontrolled (SCC 3-05-002-58, -59,-60)	Arsenic[b]	1.3×10^{-6}	E	340
	Barium	0.00025	E	340
	Beryllium[b]	0.0	E	340
	Cadmium[b]	4.2×10^{-6}	E	340
	Chromium[b]	2.4×10^{-5}	E	340
	Cobalt[b]	1.5×10^{-5}	E	340
	Copper	0.00017	E	340
	Lead[b]	0.00054	E	340
	Manganese[b]	0.00065	E	340
	Nickel[b]	0.0013	E	340
	Phosphorus[b]	0.0012	E	340
	Selenium[b]	2.4×10^{-6}	E	340
	Thallium	2.2×10^{-6}	E	340
	Zinc	0.00018	E	340
Natural gas- or propane-fired dryer, with fabric filter (SCC 3-05-002-55, -56,-57))	Antimony	1.8×10^{-7}	E	339
	Arsenic[b]	5.6×10^{-7}	D	25, 35, 339-340
	Barium	5.8×10^{-6}	E	25, 339-340
	Beryllium[b]	0.0	E	339-340
	Cadmium[b]	4.1×10^{-7}	D	25, 35, 162, 301, 339-340
	Chromium[b]	5.5×10^{-6}	C	25, 162-164, 301, 339-340
	Cobalt[b]	2.6×10^{-8}	E	339-340
	Copper	3.1×10^{-6}	D	25, 162-164, 339-340
	Hexavalent chromium[b]	4.5×10^{-7}	E	163
	Lead[b]	6.2×10^{-7}	E	35
	Manganese[b]	7.7×10^{-6}	D	25, 162-164, 339-340
	Mercury[b]	2.4×10^{-7}	E	35, 163
	Nickel[b]	6.3×10^{-5}	D	25, 163-164, 339-340
	Phosphorus[b]	2.8×10^{-5}	E	25, 339-340
	Silver	4.8×10^{-7}	E	25, 339-340
	Selenium[b]	3.5×10^{-7}	E	339-340
	Thallium	4.1×10^{-9}	E	339-340
	Zinc	6.1×10^{-5}	C	25, 35, 162-164, 339-340

Table 11.1-12 (cont.)

Process	Pollutant	Emission Factor, lb/ton	Emission Factor Rating	Reference Numbers
No. 2 fuel oil-fired dryer or waste oil/drain oil/No. 6 fuel oil-fired dryer, with fabric filter (SCC 3-05-002-58, -59,-60,-61,-62,-63)	Antimony	$1.8x10^{-7}$	E	339
	Arsenic[b]	$5.6x10^{-7}$	D	25, 35, 339-340
	Barium	$5.8x10^{-6}$	E	25, 339-340
	Beryllium[b]	0.0	E	339-340
	Cadmium[b]	$4.1x10^{-7}$	D	25, 35, 162, 301, 339-340
	Chromium[b]	$5.5x10^{-6}$	C	25, 162-164, 301, 339-340
	Cobalt[b]	$2.6x10^{-8}$	E	339-340
	Copper	$3.1x10^{-6}$	D	25, 162-164, 339-340
	Hexavalent chromium[b]	$4.5x10^{-7}$	E	163
	Lead[b]	$1.5x10^{-5}$	C	25, 162, 164, 178-179, 183, 301, 315, 339-340
	Manganese[b]	$7.7x10^{-6}$	D	25, 162-164, 339-340
	Mercury[b]	$2.6x10^{-6}$	D	162, 164, 339-340
	Nickel[b]	$6.3x10^{-5}$	D	25, 163-164, 339-340
	Phosphorus[b]	$2.8x10^{-5}$	E	25, 339-340
	Silver	$4.8x10^{-7}$	E	25, 339-340
	Selenium[b]	$3.5x10^{-7}$	E	339-340
	Thallium	$4.1x10^{-9}$	E	339-340
	Zinc	$6.1x10^{-5}$	C	25, 35, 162-164, 339-340

[a] Emission factor units are lb/ton of HMA produced. SCC = Source Classification Code. To convert from lb/ton to kg/Mg, multiply by 0.5. Emission factors apply to facilities processing virgin aggregate or a combination of virgin aggregate and RAP.

[b] Arsenic, beryllium, cadmium, chromium, hexavalent chromium, cobalt, lead, manganese, mercury, nickel, and selenium compounds are HAPs as defined in the 1990 CAAA. Elemental phosphorus also is a listed HAP, but the phosphorus measured by Method 29 is not elemental phosphorus.

Table 11.1-13. EMISSION FACTORS FOR HOT MIX ASPHALT HOT OIL SYSTEMS[a]

Process	Pollutant		Emission factor	Emission factor units	EMISSION FACTOR RATING	Reference
	CASRN	Name				
Hot oil system fired with natural gas (SCC 3-05-002-06)	630-08-0	Carbon monoxide	8.9×10^{-6}	lb/ft^3	C	395
	124-38-9	Carbon dioxide	0.20	lb/ft^3	C	395
	50-00-0	Formaldehyde	2.6×10^{-8}	lb/ft^3	C	395
Hot oil system fired with No. 2 fuel oil (SCC 3-05-002-08)	630-08-0	Carbon monoxide	0.0012	lb/gal	C	395
	124-38-9	Carbon dioxide	28	lb/gal	C	395
	50-00-0	Formaldehyde	3.5×10^{-6}	lb/gal	C	395
	83-32-9	Acenaphthene[b]	5.3×10^{-7}	lb/gal	E	35
	208-96-8	Acenaphthylene[b]	2.0×10^{-7}	lb/gal	E	35
	120-12-7	Anthracene[b]	1.8×10^{-7}	lb/gal	E	35
	205-99-2	Benzo(b)fluoranthene[b]	1.0×10^{-7}	lb/gal	E	35
	206-44-0	Fluoranthene[b]	4.4×10^{-8}	lb/gal	E	35
	86-73-7	Fluorene[b]	3.2×10^{-8}	lb/gal	E	35
	91-20-3	Naphthalene[b]	1.7×10^{-5}	lb/gal	E	35
	85-01-8	Phenanthrene[b]	4.9×10^{-6}	lb/gal	E	35
	129-00-0	Pyrene[b]	3.2×10^{-8}	lb/gal	E	35
	Dioxins					
	19408-74-3	1,2,3,7,8,9-HxCDD[b]	7.6×10^{-13}	lb/gal	E	35
	39227-28-6	1,2,3,4,7,8-HxCDD[b]	6.9×10^{-13}	lb/gal	E	35
		HxCDD[b]	6.2×10^{-12}	lb/gal	E	35
	35822-46-9	1,2,3,4,6,7,8-HpCDD[b]	1.5×10^{-11}	lb/gal	E	35
		HpCDD[b]	2.0×10^{-11}	lb/gal	E	35
	3268-87-9	OCDD[b]	1.6×10^{-10}	lb/gal	E	35
		Total PCDD	2.0×10^{-10}	lb/gal	E	35
	Furans					
		TCDF[b]	3.3×10^{-12}	lb/gal	E	35
		PeCDF[b]	4.8×10^{-13}	lb/gal	E	35
		HxCDF[b]	2.0×10^{-12}	lb/gal	E	35
		HpCDF[b]	9.7×10^{-12}	lb/gal	E	35
	67562-39-4	1,2,3,4,6,7,8-HpCDF[b]	3.5×10^{-12}	lb/gal	E	35
	39001-02-0	OCDF[b]	1.2×10^{-11}	lb/gal	E	35
		Total PCDF	3.1×10^{-11}	lb/gal	E	35
		Total PCDD/PCDF	2.3×10^{-10}	lb/gal	E	35

[a] Emission factor units are lb/gal of fuel consumed. To convert from pounds per standard cubic foot (lb/ft^3) to kilograms per standard cubic meter (kg/m^3), multiply by 16. To convert from lb/gal to kilograms per liter (kg/l), multiply by 0.12. CASRN = Chemical Abstracts Service Registry Number. SCC = Source Classification Code.

[b] Compound is classified as polycyclic organic matter, as defined in the 1990 Clean Air Act Amendments (CAAA). Total PCDD is the sum of the total tetra through octa dioxins; total PCDF is sum of the total tetra through octa furans; and total PCDD/PCDF is the sum of total PCDD and total PCDF.

Table 11.1-14. PREDICTIVE EMISSION FACTOR EQUATIONS FOR LOAD-OUT AND SILO FILLING OPERATIONS[a]

EMISSION FACTOR RATING: C

Source	Pollutant	Equation
Drum mix or batch mix plant load-out (SCC 3-05-002-14)	Total PM[b]	$EF = 0.000181 + 0.00141(-V)e^{((0.0251)(T + 460) - 20.43)}$
	Organic PM[c]	$EF = 0.00141(-V)e^{((0.0251)(T + 460) - 20.43)}$
	TOC[d]	$EF = 0.0172(-V)e^{((0.0251)(T + 460) - 20.43)}$
	CO	$EF = 0.00558(-V)e^{((0.0251)(T + 460) - 20.43)}$
Silo filling (SCC 3-05-002-13)	Total PM[b]	$EF = 0.000332 + 0.00105(-V)e^{((0.0251)(T + 460) - 20.43)}$
	Organic PM[c]	$EF = 0.00105(-V)e^{((0.0251)(T + 460) - 20.43)}$
	TOC[d]	$EF = 0.0504(-V)e^{((0.0251)(T + 460) - 20.43)}$
	CO	$EF = 0.00488(-V)e^{((0.0251)(T + 460) - 20.43)}$

[a] Emission factor units are lb/ton of HMA produced. SCC = Source Classification Code. To convert from lb/ton to kg/Mg, multiply by 0.5. EF = emission factor; V = asphalt volatility, as determined by ASTM Method D2872-88 "Effects of Heat and Air on a Moving Film of Asphalt (Rolling Thin Film Oven Test - RTFOT)," where a 0.5 percent loss-on-heating is expressed as "-0.5." Regional- or site-specific data for asphalt volatility should be used, whenever possible; otherwise, a default value of -0.5 should be used for V in these equations. T = HMA mix temperature in °F. Site-specific temperature data should be used, whenever possible; otherwise a default temperature of 325°F can be used. Reference 1, Tables 4-27 through 4-31, 4-34 through 4-36, and 4-38 through 4-41.

[b] Total PM, as measured by EPA Method 315 (EPA Method 5 plus the extractable organic particulate from the impingers). Total PM is assumed to be predominantly PM-2.5 since emissions consist of condensed vapors.

[c] Extractable organic PM, as measured by EPA Method 315 (methylene chloride extract of EPA Method 5 particulate plus methylene chloride extract of impinger particulate).

[d] TOC as propane, as measured with an EPA Method 25A sampling train or equivalent sampling train.

Table 11.1-15. SPECIATION PROFILES FOR LOAD-OUT, SILO FILLING, AND ASPHALT STORAGE EMISSIONS–ORGANIC PARTICULATE-BASED COMPOUNDS

EMISSION FACTOR RATING: C

Pollutant	CASRN[a]	Speciation Profile for Load-out and Yard Emissions[b] Compound/Organic PM[c]	Speciation Profile for Silo Filling and Asphalt Storage Tank Emissions Compound/Organic PM[c]
PAH HAPs			
Acenaphthene	83-32-9	0.26%	0.47%
Acenaphthylene	208-96-8	0.028%	0.014%
Anthracene	120-1207	0.070%	0.13%
Benzo(a)anthracene	56-55-3	0.019%	0.056%
Benzo(b)fluoranthene	205-99-2	0.0076%	ND[d]
Benzo(k)fluoranthene	207-08-9	0.0022%	ND[d]
Benzo(g,h,i)perylene	191-24-2	0.0019%	ND[d]
Benzo(a)pyrene	50-32-8	0.0023%	ND[d]
Benzo(e)pyrene	192-97-2	0.0078%	0.0095%
Chrysene	218-01-9	0.103%	0.21%
Dibenz(a,h)anthracene	53-70-3	0.00037%	ND[d]
Fluoranthene	206-44-0	0.050%	0.15%
Fluorene	86-73-7	0.77%	1.01%
Indeno(1,2,3-cd)pyrene	193-39-5	0.00047%	ND[d]
2-Methylnaphthalene	91-57-6	2.38%	5.27%
Naphthalene	91-20-3	1.25%	1.82%
Perylene	198-55-0	0.022%	0.030%
Phenanthrene	85-01-8	0.81%	1.80%
Pyrene	129-00-0	0.15%	0.44%
Total PAH HAPs		5.93%	11.40%
Other semi-volatile HAPs			
Phenol		1.18%	ND[d]

[a] Chemical Abstract Service Registry Number.
[b] Emissions from loaded trucks during the period between load-out and the time the truck departs the plant.
[c] Emission factor for compound is determined by multiplying the percentage presented for the compound by the emission factor for extractable organic particulate (organic PM) as determined from Table 11.1-14.
[d] ND = Measured data below detection limits.

Table 11.1-16. SPECIATION PROFILES FOR LOAD-OUT, SILO FILLING, AND ASPHALT STORAGE EMISSIONS–ORGANIC VOLATILE-BASED COMPOUNDS

EMISSION FACTOR RATING: C

Pollutant	CASRN	Speciation Profile for Load-Out and Yard Emissions Compound/TOC[a]	Speciation Profile for Silo Filling and Asphalt Storage Tank Emissions Compound/TOC (%)[a]
VOC[b]		94%[b]	100%
<u>Non-VOC/non-HAPs</u>			
Methane	74-82-8	6.5%	0.26%
Acetone	67-64-1	0.046%	0.055%
Ethylene	74-85-1	0.71%	1.1%
Total non-VOC/non-HAPS		7.3%	1.4%
<u>Volatile organic HAPS</u>			
Benzene	71-43-2	0.052%	0.032%
Bromomethane	74-83-9	0.0096%	0.0049%
2-Butanone	78-93-3	0.049%	0.039%
Carbon Disulfide	75-15-0	0.013%	0.016%
Chloroethane	75-00-3	0.00021%	0.0040%
Chloromethane	74-87-3	0.015%	0.023%
Cumene	92-82-8	0.11%	ND[c]
Ethylbenzene	100-41-4	0.28%	0.038%
Formaldehyde	50-00-0	0.088%	0.69%
n-Hexane	100-54-3	0.15%	0.10%
Isooctane	540-84-1	0.0018%	0.00031%
Methylene Chloride	75-09-2	0.0%[d]	0.00027%
MTBE	596899	0.0%[d]	ND[c]
Styrene	100-42-5	0.0073%	0.0054%
Tetrachloroethene	127-18-4	0.0077%	ND[c]
Toluene	100-88-3	0.21%	0.062%
1,1,1-Trichloroethane	71-55-6	0.0%[d]	ND[c]
Trichloroethene	79-01-6	0.0%[d]	ND[c]
Trichlorofluoromethane	75-69-4	0.0013%	ND[c]
m-/p-Xylene	1330-20-7	0.41%	0.2%
o-Xylene	95-47-6	0.08%	0.057%
Total volatile organic HAPs		1.5%	1.3%

Table 11.1-16 (cont.)

[a] Emission factor for compound is determined by multiplying the percentage presented for the compound by the emission factor for total organic compounds (TOC) as determined from Table 11.1-14.

[b] The VOC percentages are equal to 100 percent of TOC minus the methane, acetone, methylene chloride, and 1,1,1-trichloroethane percentages.

[c] ND = Measured data below detection limits. Additional compounds that were not detected are: acrylonitrile, allyl chloride, bromodichloromethane, bromoform, 1,3-butadiene, carbon tetrachloride, chlorobenzene, chloroform, dibromochloromethane, 1,2-dibromoethane, 1,1-dichloroethane, 1,2-dichloroethane, 1,1-dichloroethene, cis-1,2-dichloroethene, trans-1,2-dichloroethene, 1,2-dichloropropane, cis-1,3-dichloropropene, trans-1,3-dichloropropene, 1,2-epoxybutane, ethyl acrylate, 2-hexanone, iodomethane, methyl methacrylate, 1,1,2,2-tetrachloroethane, 1,1,2-trichloroethane, vinyl acetate, vinyl bromide, and vinyl chloride

[d] Values presented as 0.0% had background concentrations higher than the capture efficiency-corrected measured concentration.

U.S. Environmental Protection Agency

Technology Transfer Network Clearinghouse for Inventories & Emission Factors

Proposed Revision to AP-42, 11.1 Hot Mix Asphalt Plants

December 2, 2003

The National Asphalt Pavement Association (NAPA) has proposed a revision of the emission factors for hot oil heaters at hot mix asphalt plants. A preliminary review of their proposal was performed by EPA to verify that the published procedures for developing emission factors (Procedures for Preparing Emission Factor Documents , EPA-454/R-95-015, November 1997, PDF 477K) were followed. As a result, the proposed revisions are being posted on the CHIEF web site as draft for comment. EPA will accept comments on the draft revisions until **January 16, 2004**. Comments concerning the proposed revisions should be e-mailed to Ron Myers at myers.ron@epa.gov with copies to Una Connolly of NAPA at uconnolly@hotmix.org.

To assist reviewers in commenting on the revisions, the following documents are being made available on the CHIEF web site:

Memo from Bryan Shrager and Rick Marenshaw of RTI International to Ron Myers of EPA proposing Hot Mix Asphalt AP-42 Revision.(PDF 57K)

Quality Assurance Project Plan & Site-specific Test Plan, Formaldehyde Emissions Testing from Asphalt Heaters - March 19,2003. (PDF 2.2M)

Test Report - Formaldehyde Emissions Testing from Asphalt Heaters - October 2003.(PDF 1.8M)

Comprehensive Emission Inventory Report As Required Under The Air Toxics Hot Spots Information & Assessment Act Of 1987, September 1990, Reference 35 for Hot Mix Asphalt Plants (PDF 2.75M)

At the conclusion of the comment period, EPA will provide the comments to NAPA for evaluation, preparation of responses and revision of the portions of the Section and Background report. EPA will evaluate the responses and revisions prepared by NAPA and determine whether they are scientifically and technically sound and consistent with established EPA procedures. While the information included in this draft concerns only formaldehyde, CO and CO2 emissions from hot oil heaters, the final AP-42 Section and Background Report will be published as a single set of documents which incorporate these revisions.

AP-42 Emission Factors by Chapter

| Office of Air Quality Planning & Standards | Technology Transfer Network |
| Clearinghouse for Inventories & Emission Factors |

STUDIES DOCUMENT NEGATIVE IMPACTS FROM ASPHALT PLANTS
PROPERTY VALUES AND PUBLIC HEALTH SUFFER

The Blue Ridge Environmental Defense League has released two studies showing the adverse impacts on property values and public health for residents living near operating asphalt plants in Avery and Macon counties. The property value study shows losses of up to 56% around a plant in Pineola, and in Cullasaja nearly half of the residents report negative impacts on their health since asphalt plant operations began in 1999.

In Avery County tax office officials used distance from Maymead Materials, Inc. asphalt plant and noxious odor emissions as the bases for property devaluation in Pineola. The largest percentage drop was recorded on property located directly across the road from the plant. The largest dollar loss of $45,300 was at a church adjacent to the plant. The study documents property value losses up to 3,200 feet from the plant.

Pineola resident Dale Thompson and many of his neighbors sought tax relief when the asphalt plant effectively reduced their use and enjoyment of their homes and land. Mr. Thompson cited smoke and vile odors as reasons why he and his family can no longer spend time outdoors at either recreation or work.

In a second study, the Blue Ridge Environmental Defense League conducted a survey in response to health concerns of residents in the mountain community of Bethel in Macon County. The door-to-door survey shows that 45% of the residents living within a half mile of the two year old Rhodes Brothers asphalt plant report a deterioration of their health which began after the plant opened. The most frequent problems include high blood pressure (18% of people surveyed), sinus problems (18%), headaches (14%), and shortness of breath (9%).

Pineola's experience with property devaluation gives us only a part of the picture. The effect on the health of residents in these two communities is devastating. People who have only a passing acquaintance with asphalt fumes know little about the true dangers of this pollution. Good health is priceless—It's simply absurd to say that asphalt plants have no impact.

Louis Zeller
January 6, 2004

BLUE RIDGE ENVIRONMENTAL DEFENSE LEAGUE

PO Box 88 Glendale Springs, North Carolina 28629 Phone 336-982-2691 Fax 336-982-2954 Email bredl@skybest.com

www.BREDL.org

Asphalt Plant Fugitive Air Emissions: A Public Health Hazard

The effect of fugitive emissions on local pollution levels may exceed the effects of pollutants emitted from the smokestack.

Fugitive emissions are air pollution from a source close to ground level. Hot mix asphalt contains gravel and sand mixed with asphalt cement obtained from crude oil. Hydrocarbons released into the air by the hot mix asphalt as it is loaded into trucks and hauled from the plant site include volatile organic compounds, polycyclic aromatic hydrocarbons, and condensed particulates. Because fugitive emissions occur close to ground level, wind velocity is reduced and air pollution is not subject to the dispersion which occurs at smokestack levels. Stagnant air conditions and inversions increase the level of exposure to the local community.

Asphalt cement is a mixture of hydrocarbons including naphtha which contribute to the vaporization of organic compounds at operating temperatures of 300-350 degrees F. Condensation of particulates occurs at ambient temperatures of 70 degrees F. These very fine particles carry polynuclear aromatic hydrocarbons which are a danger to public health.

If you know the annual consumption of asphalt cement, you can calculate the asphalt vapor emissions from any plant. Asphalt cement comprises 5% (0.05) of the total hot mix plant production. Fugitive air emissions equal 1.07% (0.0107) of the consumed asphalt cement (data from Dr. R.M. Nadkarni).

For an asphalt plant producing 100,000 tons of hot mix asphalt per year:

100,000 tons hot mix x 0.05 = 5,000 tons/year of asphalt cement consumed.

Fugitive air emissions equal 1.07% (0.0107) of the consumed asphalt.

5,000 x 0.0107 = 53.5 tons per year of asphalt vapor fugitive emissions

The bulk of these fugitive emissions are condensed particulates. Volatile organic compounds (VOC's) emissions are about 29% of the this total. Therefore, about 15 tons of VOC's and 38 tons of particulates may be emitted by a 100,000 ton/year asphalt plant as fugitive emissions. To this must be added the total emitted from the smokestack itself.

The US Department of Health and Human Services has determined that PAHs (Polycyclic aromatic hydrocarbons) may be carcinogenic to humans. Animal studies show that PAHs affect reproduction, cause birth defects, and cause harmful effects on skin, body fluids, and the immune system. Similar effects could occur in humans. September 3, 2002 Louis Zeller

BLUE RIDGE ENVIRONMENTAL DEFENSE LEAGUE

www.BREDL.org ~ PO Box 88 Glendale Springs, North Carolina 28629 ~ Phone 336-982-2691 ~ Fax 336-982-2954 ~ Email bredl@skybest.com

BLUE RIDGE ENVIRONMENTAL DEFENSE LEAGUE

www.BREDL.org ~ PO Box 88 Glendale Springs, North Carolina 28629 ~ Phone (336) 982-2691 ~ Fax (336) 982-2954 ~ Email: BREDL@skybest.com

Asphalt Plant versus Wood Stove Pollution

Comparing Apples and Hedgehogs

Asphalt plants and household wood stoves burn fuel to produce heat. Although they burn different types of fuel, both emit some of the same chemical compounds into the air as a result of the combustion process. But a household wood stove emits but a tiny fraction of the pollution emitted by an asphalt plant. This is why one expert said that this is like comparing apples to hedgehogs, because apples-to-oranges simply does not convey the huge disparity in pollution emitted by these two sources. The table below evaluates air pollution from a conventional household wood heater stove and an average size asphalt plant. In every category, the asphalt plant emits from hundreds to thousands or even millions of times more air pollution.

Air pollutant	Wood stove emissions lb/y	Asphalt plant emissions lb/y	Comparison % Wood to asphalt
Carbon monoxide	249.7	39,000	0.64 %
Nitrogen oxides	3.02	16,500	0.02 %
PM-10	33.05	6,900	0.48 %
Sulfur dioxide	0.43	3,300	0.01 %
Total organic compounds	89.6	13,200	0.68 %
Methane	32.4	3,600	0.9 %
Benzene	2.09	117	1.79 %
Toluene	0.79	70	1.13 %
Polycyclic aromatic hydrocarbons	0.79	264	0.30 %
Cadmium	0.00002	1.26	0.0016 %
Chromium	<0.000001	7.2	0.000014 %
Nickel	0.000015	390	0.000004 %

Emission data from US Environmental Protection Agency Air Pollution Emission Factors
Hot Mix Asphalt Plants, AP-42, 11.1 3/04 at http://www.epa.gov/ttn/chief/ap42/ch11/final/c11s01.pdf
Residential Wood Stoves, AP-42, 1.10 10/96 at http://www.epa.gov/ttn/chief/ap42/ch01/final/c01s10.pdf

The US Environmental Protection Agency's most recently available air pollution emission factors for both wood stoves and asphalt plants were used to compile the data in this report.

The combustion of wood produces atmospheric emissions which are highly variable. For

example, when wood is added to a wood-burning heater stove, emissions increase for a short period because of a high burn rate. Then a longer period at lower burn rate follows during which time charcoal is burned, resulting in reduced emissions. Many wood stoves manufactured after 1986 have pollution reduction features.

The manufacture of asphalt paving produces high levels of atmospheric emissions. Some of these pollutants are emitted through the smokestack after passing through a fabric filter, some are released at ground level without any pollution controls. The ground-level emissions are poorly estimated by state and federal air quality guidance.

To calculate the annual pollution totals for this report, we compared a drum-mix asphalt plant burning number 2 fuel oil producing 300 thousand tons of asphalt per year with a residential wood stove burning three cords of oak wood per year.

The Carolina Asphalt Paving Association claims that ten residential wood stoves emit as much polycyclic aromatic hydrocarbons as an asphalt plant. (http://www.carolinaasphalt.org/about_research.asp). Plainly, this assertion is not supported by EPA data. In fact, an average sized asphalt plant can emit more PAH than 300 wood stoves, more sulfur dioxide than 7,000 wood stoves, and more cadmium than 63,000 wood stoves.

Air pollutant	**Fireplace emissions lb/y**	**Asphalt plant Emissions lb/y**	**Comparison % Wood to asphalt**
Volatile organic compounds (VOC)	247.3	9,600	2.6 %

Emission data from US Environmental Protection Agency Air Pollution Emission Factors Residential Fireplaces, AP-42, 1.9, 10/96 at http://www.epa.gov/ttn/chief/ap42/ch11/final/c01s09.pdf

Further, CAPA states that "during the course of a year, an asphalt plant gave off the VOCs of two residential fireplaces." Again, the EPA emission factors tell a different story; a single medium sized asphalt plant produces as much VOC as 39 fireplaces burning three cords of oak. Other categories of pollutants emitted by fireplaces are similar to those of wood-burning stoves. Open fireplaces are inefficient sources of heat because combustion is poorly regulated. Uncontrolled air and a lack of secondary combustion results in relatively high quantities of unburnt compounds going up the chimney. Nevertheless, comparing the air pollution from a modern type asphalt plant with an old-fashioned fireplace is like comparing apples to hedgehogs.

Louis Zeller
October 20, 2005

HAZARD REVIEW

HEALTH EFFECTS OF OCCUPATIONAL EXPOSURE TO ASPHALT

Contributors:
Mary Ann Butler, Ph.D.
Gregory Burr, C.I.H.
David Dankovic, Ph.D.
R. Alan Lunsford, Ph.D.
Aubrey Miller, M.D.
Mimi Nguyen, M.P.H.
Larry Olsen, Ph.D.
Douglas Sharpnack, Ph.D.
John Snawder, Ph.D.
Leslie Stayner, Ph.D.
Marie Haring Sweeney, Ph.D.
Alexander Teass, Ph.D.
Joann Wess, M.S.
Ralph Zumwalde, M.S.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control and Prevention
National Institute for Occupational Safety and Health

December 2000

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).

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
$µg/m^3$	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 (electropositively 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].

ASPHALT FUMES

Description: Asphalt fumes have been defined by NIOSH (1) as the nimbose effusion of small, solid particles created by condensation from the vapor state after volatilization of asphalt. In addition to particles, a cloud of fume may contain materials still in the vapor state.

The major constituent groups of asphalt are asphaltenes, resins, and oils made up of saturated and unsaturated hydrocarbons. The asphaltenes have molecular weights in the range of 1,000 to 2,600, those of the resins fall in the range of 370 to 500, and those of the oils in the range of 290 to 630.

Asphalt has often been confused with tar because the two are similar in appearance and have sometimes been used interchangeably as construction materials. Tars are, however, produced by destructive distillation of coal, oil or wood whereas asphalt is a residue from fractional distillation of crude oil.

The amounts of benzo(a)pyrene found in fumes collected from two different plants that prepared hot mix asphalt ranged from 3 to 22 ng/m^3; this is approximately 0.03% of the amount in coke oven emissions and 0.01% of that emitted from coal-burning home furnaces.

Code Numbers: (Petroleum asphalt fumes) CA 8052-42-4

DOT Designation: —

Synonyms: None.

Potential Exposure: Occupational exposure to asphalt fumes can occur during the transport, storage, production, handling, or use of asphalt. The composition of the asphalt that is produced is dependent on the refining process applied to the crude oil, the source of the crude oil, and the penetration grade (viscosity) and other physical characteristics of the asphalt required by the consumer.

The process for production of asphalt is essentially a closed-system distillation. Refinery workers are therefore potentially exposed to the fumes during loading of the asphalt for transport from the refinery during routine maintenance, such as cleaning of the asphalt storage tanks, or during accidental spills. Most asphalt is used out of doors, in paving and roofing, and the workers' exposure to the fumes is dependent on environmental conditions, work practices, and other factors. These exposures are stated to be generally intermittent and at low concentrations. Workers are potentially exposed also to skin and eye contacts with hot, cut-back, or emulsified asphalts. Spray application of cut-back, or emulsified asphalts may involve respiratory exposure also.

Because of the nature of the major uses of asphalt and asphalt products, it is not possible to determine accurately the number of workers potentially exposed to asphalt fumes in the United States, but an estimate of 500,000 can be derived from estimates of the number of workers in various occupations involved.

Permissible Exposure Limits in Air: Occupational exposure to asphalt fumes shall be controlled so that employees are not exposed to the airborne particulates at a concentration greater than 5 mg/m^3 of air, determined during any 15-minute period. ACGIH gives a tentative STEL of 10 mg/m^3 as of 1983/84.

Occupational exposure to asphalt fumes is defined as exposure in the workplace at a concentration of one-half or more of the recommended occupational exposure limit. If exposure to other chemicals also occurs, as is the case when asphalt is mixed with a solvent, emulsified, or used concurrently with

other materials such as tar or pitch, provisions of any applicable standard for the other chemicals shall also be followed.

Determination in Air: A gravimetric method is recommended for estimation of the air concentration of asphalt fumes (A-1). When large amounts of dust are present in the same atmosphere in which the asphalt fume is present, which may occur in road-building operations, the gravimetric method may lead to erroneously high estimates for asphalt fumes, and to possibly undeserved sanctions and citations for ostensibly exceeding the environmental limit for asphalt fumes or nuisance particulates.

NIOSH recommends (1) that where the resolution of such problems becomes necessary, a more specific procedure which involves solvent extraction and gravimetric analysis, be employed for the determination of asphalt fumes. The best procedure now available seems to be ultrasonic agitation of the filter in benzene and weighing of the dried residue from an aliquot on the clear benzene extract. NIOSH is attempting to devise an even more specific method for asphalt fumes for use under such conditions.

Permissible Concentration in Water: No criteria set.

Routes of Entry: Inhalation of dusts and fumes. Skin exposure can cause thermal burns from hot asphalt.

Harmful Effects and Symptoms: The principal adverse effects on health from exposure to asphalt fumes are irritation of the serous membranes of the conjunctivae and the mucous membranes of the respiratory tract. Hot asphalt can cause burns of the skin. In animals, there is evidence that asphalt left on the skin for long periods of time may result in local carcinomas, but there have been no reports of such effects on human skin that can be attributed to asphalt alone. No reliable reports of malignant tumors of parenchymatous organs due to exposure to asphalt fumes have been found, but there has been no extensive study of this possible consequence of occupational exposure in the asphalt industry.

Points of Attack: Skin, respiratory system.

Medical Surveillance: Details of recommended preplacement and periodic physical examinations and record-keeping have been set forth by NIOSH (1).

Personal Protective Methods: Employees shall wear appropriate protective clothing, including gloves, suits, boots, face shields (8-inch minimum), or other clothing as needed, to prevent eye and skin contact with asphalt.

Respirator Selection: (1) Engineering controls shall be used when needed to keep concentrations of asphalt fumes below the recommended exposure limit. The only conditions under which compliance with the recommended exposure limit may be achieved by the use of respirators are:

(a) During the time required to install or test the necessary engineering controls.
(b) For operations such as nonroutine maintenance or repair activities causing brief exposure at concentrations above the environmental limit.
(c) During emergencies when concentrations of asphalt fumes may exceed the environmental limit.

(2) When a respirator is permitted by (1) above, it shall be selected from a list of respirators approved by NIOSH.

Disposal Method Suggested: Incineration.

Karaman A, Pirim I
Exposure to bitumen fumes and genotoxic effects on Turkish asphalt workers.
Clin Toxicol (Phila) 2009, Apr 14 :1-6.

Objective:

Bitumen fumes consist essentially of polycyclic aromatic hydrocarbons (PAHs) and their derivatives, some of which are known to be carcinogenic or cocarcinogenic in humans. The aim of this study was to investigate exposure to asphalt fumes among Turkish asphalt workers and determine whether any effects could be detected with genotoxic tests. Study Design. The study included 26 asphalt workers and 24 control subjects. Sister chromatid exchange (SCE) and micronucleus (MN) were determined in peripheral lymphocytes. Urinary 1-hydroxypyrene (1-OHP) excretion was used as a biomarker of occupational exposure to PAHs.

Results:

The asphalt workers had a significant increase in SCEs and MN (for each, $p < 0.001$). A positive correlation existed between the duration of exposure and rates of SCE or MN frequencies ($r = 0.49$, $p < 0.05$; $r = 0.53$, $p < 0.05$, respectively). The concentration of 1-OHP in urine was higher for the asphalt workers than for the controls ($p < 0.001$). However, we found that there was no statistically significant correlation between the urinary 1-OHP concentration and SCEs or MN frequencies ($r = 0.25$, $p > 0.5$; $r = 0.17$, $p > 0.5$, respectively).

Conclusions:

This study shows that Turkish asphalt workers have an increased exposure to PAHs from bitumen fumes, and genotoxic effects could be detected by SCEs and MN tests.

http://www.informaworld.com/smpp/content~content=a910409985~db=all~jumptype=rss

Studies of carcinogenicity of bitumen fume in humans

American Journal of Industrial Medicine
Volume 43, Issue 1, Date: January 2003, Pages:1-2

Paolo Boffetta, Igor Burstyn

Abstract

Since antiquity humans have used bitumen, either naturally occurring or derived from crude oil [Broome and Hobson, 1973], and it may have been the binding material described for bricks used in the construction of the Tower of Babel (Genesis 11, 3).Chemically, bitumen is a complex mixture of hydrocarbons consisting of both aliphatic and aromatic compounds, some of which bear nitrogen, oxygen, or sulfur functional groups [Broome and Hobson, 1973]. This material has been in widespread use since the industrial revolution. The first bituminous road was built in 1810 in Lyon, France. Large-scale industrial use of bitumen began with the exploitation of natural bitumen deposits in Trinidad, with the first commercial shipment arriving in England in the 1840s. Bitumen's main use, in terms of volume, has been in paving, as a binder for inorganic fillers in asphalt mixes. According to conservative estimates, there are at present approximately 4000 asphalt mixing plants in western Europe. A typical mixing plant employs five to ten individuals. These plants produce approximately 275 million tons of hot and 10 million tons of cold asphalt annually. Asphalt mixes are applied to road surfaces by approximately 100,000 paving crewmen across western Europe. Other important uses of bitumen are in waterproofing and roofing. Thus, assessment of the health hazards of bitumen fumes may have far-reaching industrial, economic, and public health implications.

Of specific concern is the potential carcinogenicity of bitumen fume inhalation. In 1985 and 1987, the International Agency for Research on Cancer [IARC, 1985, 1987] evaluated extracts of steam-refined and air-refined bitumen carcinogenicity in experimental animals and classified them as possible human carcinogens (IARC Group 2B), while for undiluted bitumen, the evidence of carcinogenicity in humans was inadequate (IARC Group 3).Meta-analysis identified and reveiwed the epidemiological studies informative of cancer hazard in asphalt workers [Partanen and Boffetta, 1994]. However, the aggregated data could not explicitly address effects of bitumen fumes. Agent-specific exposure data were lacking, conjectured, or controversial, leaving open a number of questions with regard to the interpretation of the results. The aggregated results suggested an increased risk of cancers of the lung, (relative risk 1.8; 95% confidence interval 0.8-1.0).The main uncertainty in the assessment of previous epidemiological data arises from the inability to exclude the possibility of confounding by concurrent use of both coal tar a recognized carcinogen, and bitumen by pavers, roofers, and waterproofers [IARC, 1985, 1987]. The voluntary discontinuation of coal tar use by the asphalt industry in western Europe during the past few decades presented an opportunity to discover whether it is likely that bitumen exposure per se is carcinogenic [Partanen et al., 1995].

To address this question, a historical cohort of asphalt workers was assembled by IARC in eight countries (Denmark, Finland, France, Germany, the Netherlands, Norway, Sweden, and Israel) in order to obtain diverse exposure profiles and a sufficient number of cases for the main health outcome of interest: lung cancer. Detailed results on the mortality of the workers included in the international study have been published in an IARC Internal Technical Report [Boffetta et al., 2001]. In this issue of the Journal, several papers report the key findings on cancer mortality [Boffetta et al., 2003a,b], which provide the most complete assessment of cancer hazards among workers exposed to bitumen fumes. It is a complex task to organize and conduct international occupational cohort studies. This project was a successful example of collaboration between academic research groups, public bodies, and industrial associations. Among other challenges, it overcame the Babel of multiple languages.

Acute symptoms associated with asphalt fume exposure among road pavers

American Journal of Industrial Medicine
Volume 49, Issue 9, Date: September 2006, Pages: 728-739

Allison L. Tepper, Gregory A. Burr, H. Amy Feng, Mitchell Singal, Aubrey K. Miller, Kevin W. Hanley, Larry D. Olsen

Background: Although asphalt fume is a recognized irritant, previous studies of acute symptoms during asphalt paving have produced inconsistent results. Between 1994 and 1997, the National Institute for Occupational Safety and Health (NIOSH) evaluated workers at seven sites in six states.

Methods: NIOSH (a) measured exposures of asphalt paving workers to total (TP) and benzene-soluble particulate (BSP), polycyclic aromatic compounds, and other substances; (b) administered symptom questionnaires pre-shift, every 2 hr during the shift, and post-shift to asphalt exposed and nonexposed workers; and (c) measured peak expiratory flow rate (PEFR) of asphalt paving workers when they completed a symptom questionnaire.

Results: Full-shift time-weighted average exposures to TP and BSP ranged from 0.01 to 1.30 mg/m^3 and 0.01 to 0.82 mg/m^3, respectively. Most BSP concentrations were <0.50 mg/m^3. Asphalt workers had a higher occurrence rate of throat irritation than nonexposed workers [13% vs. 4%, odds ratio (OR) = 4.0, 95% confidence interval (CI): 1.2-13]. TP, as a continuous variable, was associated with eye (OR = 1.34, 95% CI: 1.12-1.60) and throat (OR = 1.40, 95% CI: 1.06-1.85) symptoms. With TP dichotomous at 0.5 mg/m^3, the ORs and 95% CIs for eye and throat symptoms were 7.5 (1.1-50) and 15 (2.3-103), respectively. BSP, dichotomous at 0.3 mg/m^3, was associated with irritant (eye, nose, or throat) symptoms (OR = 11, 95% CI: 1.5-84). One worker, a smoker, had PEFR-defined bronchial lability, which did not coincide with respiratory symptoms.

Conclusions: Irritant symptoms were associated with TP and BSP concentrations at or below 0.5 mg/m^3.

http://www.ncbi.nlm.nih.gov/pubmed/16917829

American Journal of Industrial Medicine 25:279–289 (1994)

Toxic Health Effects Including Reversible Macrothrombocytosis in Workers Exposed to Asphalt Fumes

Robert M. Chase, MD, FRCP(C), Gary M. Liss, MD, MS, FRCP(C), Donald C. Cole, MD, FRCP(C), and Bonnie Heath, MHSc

We investigated an outbreak of irritative and neurotoxic symptoms associated with exposure to asphalt fumes in a commercial lighting factory; 27 symptomatic female workers were clinically assessed including hematologic testing. When compared with a laboratory reference group (n = 107), the workers' mean platelet volume (MPV) was significantly higher and mean platelet count was lower (p = 0.013 and p = 0.048, respectively). Five months later, the factory's ventilation system was substantially modified. Follow-up assessments 6 months postmodification on 15 of the original workers documented a significant decline in acute symptoms and a lowering of the subjects' mean MPV towards normal (p = 0.0007 by paired t-test). The findings suggest that reversible macrothrombocytosis (enlarged platelets) can occur among symptomatic workers exposed to asphalt fumes. © 1994 Wiley-Liss, Inc.

Key words: blood platelet disorders, hematological parameters, occupational exposure, asphalt, benzene, biological effect, follow-up studies

INTRODUCTION

Lakeshore Area Multiservice Project (LAMP) Occupational Health Program (LOHP) is a community-based occupational health service in Toronto, Ontario, Canada funded by the provincial government.

In March 1988, several employees from a local plant came to LOHP with complaints of nausea, headache, fatigue, skin rashes, and eye, nose and throat irritation. The onset of these symptoms coincided with the introduction of a new asphalt formulation in the manufacturing process in November 1987. The plant employs approximately 200 production employees, mostly female, manufacturing fluorescent ballast boxes and coils for fluorescent and high intensity lighting. The production area is open, without partitions (approximately 250 × 200 ft with 20 ft ceilings). In 1987, general ventilation (fan) was used to disperse fumes from the soldering stations and

Occupational Health Program, Lakeshore Area Multi-service Project (R.M.C., D.C.C.); Health and Safety Studies Unit, Ontario Ministry of Labour (G.M.L.); Community Health Branch, Ontario Ministry of Health (B.H.), Ontario, Canada.
Address reprint requests to Robert M. Chase, MD, L.A.M.P. Occupational Health Program, 185 Fifth Street, Etobicoke, Ontario, Canada M8V 2Z5.
Accepted for publication March 15, 1993.

http://ntp-server.niehs.nih.gov/index.cfm?objectid=0DA9C8CD-F1F6-975E-7631B117EEDF8C3D

LITERATURE REVIEW OF HEALTH EFFECTS CAUSED BY OCCUPATIONAL EXPOSURE TO ASPHALT FUMES

This Interim Review Produced by NIOSH in Support of Nomination to the National Toxicology Program

6/23/97

CHEMICAL AND PHYSICAL PROPERTIES*

*Information obtained from Sax and Lewis [1987].

_____

Chemical name	. . . . .	Asphalt
CAS number	. . . . .	8052-42-4
Synonyms	Asphaltum; asphalt cement; asphalt emulsion; bitumen; blown asphalt; cutback asphalt; oxidized asphalt; petroleum asphalt; petroleum bitumen; road asphalt	

Physical state at room temperature Black or dark-brown solid or viscous liquid

Solubility in water at 20°C Insoluble

Solubility in organ solvents Carbon disulfide

Definition of asphalt -

Asphalt production is dictated by performance specifications rather than by a specific chemical composition. To meet those specifications, the residual product of petroleum distillation may be further processed, usually by air-blowing or solvent precipitation. The precise chemical composition and physical properties of the resulting products are influenced by the composition of the original crude petroleum oil and the manufacturing processes. The basic chemical components of crude petroleum oil include paraffinic, naphthenic, and aromatic hydrocarbons as well as heterocyclic molecules containing sulfur, oxygen, and nitrogen [AI 1990a]. The proportions of these chemical components may vary significantly because sources of crude petroleum oil occur in various locations throughout the world involving different geologic formations. As a result of these variations, crude oils from different fields may vary in their chemical composition and sometimes variations in chemical composition of crude oils can be found among different locations in the same oil field [Puzinauskas and Corbett 1978]. Therefore, no two asphalts are chemically identical, and chemical analysis defining the precise structure and size of the individual molecules found in asphalt is almost impossible.

Asphalt fumes are defined as the cloud of small particles created by condensation from the gaseous state after volatilization of asphalt. Fumes from some asphalts have been analyzed and their chemical compositions are presented in Table 1 [AI 1975] and Table 2 [Reinke and Swanson 1993].

PRODUCTION, USE, AND POTENTIAL FOR OCCUPATIONAL EXPOSURE

Paving asphalts are manufactured principally by simple atmospheric distillation or by atmospheric distillation followed by fractionation under vacuum. They may also be manufactured by solvent precipitation and mild partial air-blowing. Roofing asphalts are generally produced by atmospheric or vacuum distillation followed by air-blowing [NAPA 1994].

Most of the asphalt produced in the United States is used in paving and roofing. Only about 1% is used for waterproofing, dampproofing, insulation, paints, and other activities [AI 1990a]. The National Occupational

Exposure Survey (NOES) [NIOSH 1983] estimates that during the period 1981-83, more than 473,000 U.S. employees were potentially exposed to asphalt. Table 3 presents the 10 industries and the 10 occupations (excluding janitors) with the most employees potentially exposed to asphalt.

Paving Asphalt

Of the three types of asphalt products used in the construction of paved surfaces in the United States: asphalt paving cements (hot-mix asphalt or HMA), cutback asphalts, and asphalt emulsions, HMA (asphalt mixed with mineral aggregate) accounts for 85% of the total used. Cutback asphalts and asphalt emulsions are used for road sealing and maintenance, and account for 4% and 11% respectively, of the total used. Currently, about 4,000 HMA facilities and 7,000 paving contractors employ nearly 300,000 employees in the United States [AI 1990a].

Roofing Asphalt

Four types of asphalt (I through IV) are used in roofing products in the United States. The type of asphalt used is determined by the grade or slope of the roof. For example, Type I roofing asphalt, often referred to as "dead level," has a low softening point and is used on surfaces with a grade of 0.5 inch per foot or less. Types II and III roofing asphalt are typically used on roofs with slopes of 0.5 to 1.5 and 1 to 3 inches per foot, respectively. Type IV roofing asphalt (a hard asphalt with a high softening point) is used on roofs with a grade of 2 to 6 inches per foot [ASTM 1992]. In 1990, an estimated 46,000 on-roof employees were exposed to asphalt fumes in the United States, and about 6,000 to 12,000 employees were exposed in approximately 120 plants manufacturing asphalt roofing shingles and rolls and modified bitumen[2] roofing products [AI 1990a].

General Exposure

The major route of occupational exposure to asphalt fumes (e.g., paving, roofing, and asphalt-based paints) is by inhalation; they may also be absorbed through the skin. A summary of representative information on the occurrence of asphalt fumes in the workplace is presented in Table 3 and Table 4.

Dermal exposure to asphalt fumes has been examined using skin wipes (see Table 5). Skin wipe samples were collected at various worksites (e.g., refineries, HMA facilities, paving and roofing sites, and roofing manufacturers) and analyzed for PAHs [AI 1991]. The PAH concentrations determined from postshift samples ranged from 2.2 to 520 ng/cm^2 (see Appendix A).

Exposure Limits

The Occupational Safety and Health Administration (OSHA) currently has no permissible exposure limit (PEL) for asphalt fumes. In 1989, OSHA announced that it would delay a final decision to establish a PEL for asphalt fumes because of complex and conflicting issues submitted to the record [54 Fed. Reg. *2641]. The PEL originally proposed to reduce the potential carcinogenic risk of occupational exposure to asphalt fumes was 5 mg/m^3 as an 8-hr TWA. In 1992, OSHA published another proposed rule for asphalt fumes that included a PEL of 5 mg/m^3 (total particulates) for general industry and for the maritime, construction, and agricultural industry [57 Fed. Reg. 26182]. Comments are still being received by OSHA and a final decision is pending.

In a 1977 criteria document, NIOSH established a recommended exposure limit (REL) of 5 mg/m^3 as a 15 min ceiling for up to a 10-hr work shift, during a 40-hr workweek, to protect against irritation of the serous membranes of the conjunctivae and the mucous membranes of the respiratory tract [NIOSH 1977a]. In 1988, NIOSH testimony to the Department of Labor and OSHA recommended that asphalt fumes be considered a potential occupational carcinogen [NIOSH 1988]. This recommendation was based on information presented in the 1977 criteria document [NIOSH 1977a] and a study by Niemeier et al. [1988] showing that exposure to condensates of asphalt fumes caused skin tumors in two strains of mice.

The American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value (TLV®) is 5 mg/m^3 as an 8-hr TWA and was recommended to reduce the risk of possible carcinogenicity [ACGIH 1991]. Australia, Belgium, Denmark, and the United Kingdom have also limited occupational exposures to asphalt fumes to 5 mg/m^3 as an 8-hr TWA. Additionally, the United Kingdom has established a short-term exposure limit (STEL) of 10 mg/m^3.

Germany currently rates asphalt fumes as "suspected of having a carcinogenic potential [ILO 1991].

STUDIES OF GENOTOXICITY AND CARCINOGENICITY (ANIMALS)

Mutagenic Effects

The five fractions of laboratory-generated roofing asphalt fume condensates and unfractionated asphalt fumes used by Sivak et al [1989] (see description of Sivak study under Carcinogenic Effects) were examined for their mutagenic potential in Salmonella. Fractions A through E combined, and fractions B and C were positive; fractions, A, D, and neat asphalt fumes were weakly positive; and fraction E was negative [NTP 1990]. Positive responses required exogenous metabolic activation. The same fractionated asphalt fume condensates from the Sivak et al.study [1989] were also tested using a modified Ames assay [Blackburn and Kriech 1990] and the results were comparable to those of the NTP [1990] study.

Eight asphalt fume samples collected on teflon filters at HMA plants as part of an Interagency Agreement with the Federal Highway Administration (FHA) were tested for mutagenic activity in a Salmonella mutagenicity assay. Preliminary results indicate that there was no mutagenic activity in the whole fume fraction; however, results of 2 of the 8 samples were inconclusive [Olsen, personal communication].

Two Type III roofing asphalts representing different crude oil sources , one of which was similar to the asphalt air-blown using a ferric chloride catalyst and used by Niemeier et al.[1988] and Sivak et al. [1989]; 18 paving asphalts (representing 14 crude oil sources and various process conditions); and Type I coal tar pitch; and their fume condensates were examined not only for mutagenic activity in a modified Ames assay, but also for PAH content [Machado et al. 1993]. The fume generation temperature of all roofing materials was either 232 or 316°C and that of all paving materials was 163°C (one sample was heated to 221°C). The results of the modified Ames assay are presented in Table 6.

The data indicate that all samples tested exerted mutagenic activity; however, the mutagenic responses of the asphalt fume condensates were approximately 100-fold less than the coal tar pitch samples and weak to moderate in potency [Machado et al. 1993]. Responses for the positive control group were all within the expected ranges.

Results of analyses for PAH content, measured by HPLC fluorescence, of the roofing and paving asphalts, coal tar pitch, and their fume condensates were as follows [Machado et al. 1993]. Concentrations of individual PAHs in samples of asphalt and asphalt fume condensates were less than 50 parts per million by weight (ppm), while most concentrations of individual PAHs in roofing (232°C or 316°C) and all concentrations in paving (163°C, except for one sample at 221°C) asphalts, whole or fumes, were less than 10 ppm and 2 ppm, respectively. Concentrations of individual PAHs in the coal tar pitch samples were 100- to 1000- fold higher than in the roofing and paving samples. Benzo[a]pyrene (BP) was detected in all samples examined; the maximum concentrations of BP in whole asphalt, whole coal tar pitch, asphalt and coal tar pitch fume condensates were approximately 6 ppm, 18,000 ppm, 0.1 - 2.8 ppm, and 250-480 ppm, respectively.

Although PAH content correlated with mutagenicity indices for some samples, for others it did not. The investigators concluded that the data suggest that crude oil source along with processing conditions had some influence on the PAH content of the various materials tested [Machado et al.1993].

Reinke and Swanson [unpublished data 1993] examined the relationship between field-, 146-157°C (295-314°F), and laboratory-generated, 149°C (300°F) and 316°C (600°F), asphalt fume condensates by comparing their chemical content (i.e., PAHs and sulfur heterocyclics) and mutagenic potential. The asphalt tested was a straight run, vacuum distilled 85/100 penetration grade asphalt derived from a blend of Canadian heavy, sour crudes. The field asphalt fume condensates were collected from the head space above an asphalt storage tank, stored between 146-157°C (295-314°F), at a HMA production plant into a cold trap system for about 36 continuous hours. The results of the chemical analyses (GC-MS) for PAHs and sulfur heterocyclics and the modified Ames assay are provided in Table 7 and summarized in Table 8.

The data indicate that field-generated asphalt fume condensates exerted a MI of >0 and < 1, while fumes generated in the laboratory at 149°C (300°F) and 316°C (600°F), exerted MIs of 5.3 and 8.3, respectively.

Chromosomal Aberrations

Condensates of Type I and Type III roofing asphalt fumes generated in the laboratory (same methodology as Sivak et al. 1989) at temperatures (316 ± 10C) similar to actual roofing operations caused a dose-related increase in micronucleus (MN) formation in exponentially growing Chinese hamster lung fibroblasts (V79 cells) [Qian et al. 1995]. The results of immunofluorescent antibody staining showed that both roofing asphalt fume condensates induced mainly kinetochore-positive MN (68-70%). The authors suggested that Type I and Type III roofing asphalt fume condensates are aneuploidogens and possess some clastogenic activities.

Reinke and Swanson [1993] also tested 3 asphalt fume condensates (field and lab-generated) in a chromosomal aberration assay and the results were negative. The authors reported that the absence of positive findings may be explained by the fact that this assay has not as yet been optimized for petroleum asphalt fumes.

Intercellular Communication

The five asphalt roofing fume fractions used by Sivak et al. [1989] were tested for inhibition of intercellular communication, i.e., one of several proposed mechanisms of tumor promotion. 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 in Toraason et al. [1991]. The greatest activity was in fraction 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 concentrations in a concentration dependent fashion.

Carcinogenic Effects

Since publication of the NIOSH criteria document [NIOSH 1977a], there have been reports of carcinogenicity following dermal applications of laboratory-generated asphalt roofing fume condensates [Niemeier et al. 1988; Sivak et al. 1989] and raw roofing asphalt [Sivak et al. 1989]. Additional data from these studies are summarized in detail in Appendix B.

Niemeier et al. [1988] investigated the tumorigenicity of fume condensates generated at 232°C (450°F) and 316°C (601°F) from Types I and III roofing asphalt and Types I and III coal-tar pitch through topical applications to the skin of male CD-1 and C3H/HeJ mice. A total of 48 groups of 50 mice each (1 strain) received applications of cryogenically collected fume condensates singly and in combination (Type III asphalt and Type I coal-tar pitch, both generated at 316°C [601°F]) biweekly for 78 weeks (18 months). Half of each group was exposed to simulated sunlight to determine whether photochemical reactions might alter the carcinogenic activity. Analysis of the skin painting solutions by GC/MS revealed that the solutions containing coal-tar pitch fume condensates had higher concentrations of select PAHs than the solutions containing asphalt fume condensates. The authors report that analysis by nuclear magnetic resonance (NMR) indicated that the asphalt fume condensate was <1% aromatic and >99% aliphatic, whereas the coal-tar pitch condensate was >90% aromatic. BaP was selected as a marker compound based on correlations of BaP concentrations and carcinogenicity.

Tumors were produced by fume condensates of both types of asphalt (see Tables 9 and 10) and both types of coal-tar pitch. The majority of benign tumors were papillomas; the majority of malignant tumors were squamous cell carcinomas. The fume condensates from the coal-tar pitches had slightly greater carcinogenic activity than the fume condensates from the asphalts, but the total amount of select PAHs or BaP needed to produce a 50% tumor incidence was much smaller for the asphalt fume condensates (PAHs, 0.58 to 2.63 mg; BaP, less than or equal to 13.6 mg) than for the coal-tar pitch fume condensates (PAHs, >24.5 to >57.4 mg; BaP, 354 to 405 mg). Tumor response to the coal-tar pitch fume condensates was comparable with that of the BaP controls, based on the total dosage of BaP administered. Both strains of mice exposed to asphalt fumes had significantly (P=0.01) more tumors than the control groups, although the C3H/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 C3H/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 those generated at 232°C (450°F); a similar increase was noted only for Type III coal-tar

pitch fumes. Overall, simulated sunlight inhibited tumorigenic responses. The authors speculated that this inhibition may have resulted from the photo-oxidation or photodestruction of the carcinogenic components of the test materials. Niemeier et al. [1988] concluded that the enhanced carcinogenic activity of the asphalt fume condensates may have been due to their high concentration of aliphatic hydrocarbons, which have cocarcinogenic effects. They also concluded that higher generation temperatures may have further increased that hazard. Finally, Niemeier et al. [1988] concluded that the carcinogenic activity of the coal-tar pitch fume condensates (but not that of the asphalt fume condensates) could be explained by their BaP (or PAH) contents.

Sivak et al. [1989] heated Type III roofing asphalt at 316°C, generated fume condensates, and separated them by high-performance liquid chromatography [Belinky et al.]. The chemical composition of the fractions (A through E) is provided in Table 11. Raw asphalt, neat asphalt (whole or unfractionated condensate) fume, the reconstituted asphalt fume, and the asphalt 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 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 2 groups of Sencar mice (30 male mice per group) for 104 weeks (2 years).

A single initial treatment of BaP followed by individual treatments with fractions A, D, and E was used to test the tumor-promoting activity of the asphalt fume condensate. The cocarcinogenicity of fractions A, D, and E was tested with three different doses of BaP. Fractions A, D, and E were used because they were the fractions Sivak et al. [1989] deemed most likely to exhibit cocarcinogenic or tumor-promoting activity based on their chemical compositions, i.e., primarily long chain alkanes and phenol compounds. Two groups of male Sencar mice were included to allow for possible genetic variation and sensitivity to tumor promotion. One of the two groups of Sencar mice was treated with neat asphalt fume (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 BaP.
Table 12 presents only the treatment groups which induced histopathologically confirmed carcinomas (malignant tumors), the number of carcinomas per group, the number of mice with histologically confirmed carcinomas, and the average time (in weeks) to carcinoma development. The raw asphalt and neat asphalt fume induced carcinomas (local skin cancers) in 3 of 30 and 20 of 30 C_3H/HeJ mice, respectively. 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 singly. All the combinations of the fractions induced tumors only if they included B or C; combinations A and D; A and E; and A, D, and E failed to induce any tumors. Furthermore, fractions A, D, and E failed to act as either tumor promoters or cocarcinogens. Fourteen of the 30 Sencar mice treated with the asphalt fume condensate developed carcinomas.

As noted previously, only fractions B and C applied singly and in combinations elicited tumor responses. Fractions containing B and C PACs including 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 4-ring PACs (refer to previous Table). Sivak et al. [1989] stated the need for additional cocarcinogenesis 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 10 lists the positive tumor responses among the groups of mice studied. The raw asphalt (diluted with a 1:1 solution of cyclohexane and acetone to a final concentration of 0.5 g/ml) produced carcinomas in 3 of 30 C3H/HeJ mice. The neat asphalt fume (diluted with a 1:1 solution of cyclohexane and acetone to a final concentration of 0.5 g/ml) produced carcinomas in 20 of 30 C3H/HeJ mice. Fraction B produced local skin cancers (carcinomas) in 10 of 30 male C3H/HeJ mice, and fraction C produced local skin cancers (carcinomas) in 17 of 30 male C3H/HeJ mice. Fractions A, D, and E failed to produce any carcinomas when applied singly. Of the other combinations of fractions, all produced tumors except the following: A and D; A and E; and A, D, and E. None of the groups of mice with the initiating dose of 200 mg of BaP developed tumors, but 7 of the 9 groups tested for cocarcinogenicity developed carcinomas (see Table 10). Fourteen of the 30 Sencar mice treated with neat asphalt fumes (whole condensate) produced carcinomas, and 1 mouse in the Sencar solvent control group produced 1 tumor (sarcoma). Mice in the

C3H/HeJ solvent control group failed to develop tumors, whereas the C3H/HeJ mice in two BaP control groups developed skin tumors (see 0.01% and 0.001% BaP groups in Table 10).

Sivak et al. [1989] observed no tumor responses with the three roofing asphalt fractions (A, D, and E) they considered most likely to exhibit cocarcinogenic or tumor-promoting activities based on their aliphatic hydrocarbon, alcohol, and phenol contents. Treatment with the combined fractions did not produce any synergistic effects. However, tumor responses were elicited by other fractions (B and C) that contained PACs including PAHs, S-PACs, N-PACs, and O-PACs such as alkylated aryl thiophenes, alkylated phenanthrenes, alkylated phenylethanones, and alkylated dihydrofuranones. Fraction B contained most of the S-PACs, and only a few were carried over to fraction C, which contained mainly O-PACs. Because the O-PACs may result from the air-blowing/oxidation refining process common among roofing asphalts, they may be present only in roofing asphalt. If such is the case, the refining process could be altered to eliminate the O-PACs and possibly the carcinogenicity of fraction C. Sivak et al. [1989] stated the need for additional cocarcinogenesis 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.

HUMAN HEALTH EFFECTS

ACUTE

Asphalt fumes are irritants to the mucous membranes of the eyes and respiratory tract; hot asphalt can also cause burns of the skin [NIOSH 1977]. It has been reported that irritant effects on the respiratory tract can possibly progress to such nonmalignant lung diseases as bronchitis, emphysema, and asthma [Hansen, 1991; Maizlish et al. 1988]. Workers engaged in road repair and construction reported symptoms of abnormal fatigue, reduced appetite, eye irritation, and laryngeal/pharyngeal irritation [Norseth et al. 1991].

CHRONIC

Considerable data from epidemiological studies on workers exposed to asphalt fumes during paving and roofing operations, and during the production of asphalt, have become available since the publication of the NIOSH criteria document on asphalt [NIOSH 1977]. The mortality experience of Danish mastic asphalt workers [Hansen 1989a; Hansen 1991] and Swedish asphalt road pavers [Engholm 1991] was investigated (see Table 13). Hansen [1989a] reported that the mastic asphalt workers, when compared with the total male Danish population , experienced significantly increased mortality from cancers of the digestive and respiratory systems, with standardized incidence rates (SIR) of 227 (95% confidence interval of 142-344) and 195 (95% confidence interval of 236-493), respectively. The SIR for all malignant neoplasms was 195 (95% confidence interval of 153-244). Overall, Hansen [1989a] reported that she observed a three-fold increase in the expected number of lung cancers in the mastic asphalt workers compared with the general Danish population. For an assessment of the induction of primary lung cancer Hansen divided the cohort into subcohorts based on birth year because it was necessary to determine the number of employees potentially exposed to coal tar pitch, which had been added to mastic asphalt during World War II. The SIRs for primary lung cancer were then determined to range from 632 (for employees aged 40 to 54) to about 300(for employees aged 64 to 89). Although smoking histories of the cohort were unknown, an inquiry was made in 1976 into the smoking habits of mastic asphalt workers and a pattern emerged. Based on the approximate rates that were calculated, Hansen suggested that smoking could not account for the three-fold increase she had observed.

When Hansen [1991] updated her cohort and adjusted for smoking and urbanization, she reported that the statistically significant ($P<0.01$) increase in cancer mortality among mastic asphalt workers remained. The SIR for lung cancer mortality was 224 (95% CI, 145-330). Criticisms by Wong et al. [1992] and Kreich et al.] 1991] of the Hansen studies [1989a; 1991] are provided in the comments section of Table 13 and include the following: possible exposure to coal tar pitch and inadequate adjustment for smoking and urbanization.

Engholm et al. reported [1991] the occurrence of lung (SIR of 207) and stomach cancers (SIR of 207) in Swedish asphalt road pavers (see Appendix C). Data on previous and current smoking histories had been collected and were used in determining the relative risk (RR) for lung cancer. The RR for lung cancer was on the order of 2 before adjustment for smoking, and it was on the order of 3 after adjustment for smoking. Despite the short follow-up period (an average of 11.5 years) and the very young age (42 years) of the cohort, the authors concluded that this

cohort exhibited a slight excess of lung cancer. However, in a later submission to NIOSH [Engholm and Englund 1993], the results of an update based on the inclusion of three additional years of follow-up were reported. Engholm [1993] indicated that: 1) with the additional follow-up, all measures of any cancer risk were not statistically signigicant; 2) the study results may reflect some selection bias; 3) exposure of the cohort is in doubt.

Results of a proportionate mortality study of California highway maintenance workers [Maizlish et al. 1988] and a long-term mortality study of Minnesota highway maintenance workers [Bender et al. 1989] are also presented in Table 13. Maizlish et al.[1988] determined that the increased mortality from all malignant neoplasms for their cohort was not statistically significant. Additionally, exposure measurements and data on tobacco or alcohol consumption of the cohort were unavailable.

Bender et al. [1989] reported that workers with 30 to 39 years of work experience had a statistically significant ($P<0.01$) SMR of 425 (95% CI, 170-870) for leukemia deaths. The authors concluded, however, that they were unable to relate these findings to asphalt exposure. After additional study of this cohort (case-control studies, cytogenetic studies, updated chhort mortality, and personal air monitoring effort, the Minnesota Department of Health [1993] concluded that it was unlikely that the excess leukemia mortality observed among the highway maintenance workers was job-related.

Only one study is available regarding the mortality experience of roofers [Engholm et al. 1991].

During their investigation of asphalt road pavers in Sweden, these investigators also examined a cohort of roofers (see Appendix C). After adjustment for smoking, the RR for lung cancer in roofers was on the order of 6. The data indicated that though the number of cases was small, there was a lung cancer excess among roofers [Engholm et al. 1991]. Even though the authors acknowledged that the short follow-up period (11.5 years) and the young age (42 years) of the cohort were too short for the normal latency period of a potential carcinogen, they concluded that an excess of lung cancer existed among roofers. In 1993 Engholm and Englund presented to NIOSH information based on their three-year follow-up of Enghom et al. 1991. They concluded that results of the follow-up study " did not permit any final conclusions" regarding health risks of the respective cohorts.

Partanen and Boffetta [1994] conducted a review and meta-analysis of the epidemiologic studies regarding cancer risk in asphalt workers and roofers. They concluded that existing data are insufficient to make a judgment with regard to asphalt. Most epidemiologic studies for lung carcinogenicity (as well as other cancer sites) are either too non-specific for exposure (e.g., highway maintenance workers , census occupational data), or confounded by coal tar exposure.

In 1987, the International Agency for Research on Cancer (IARC) evaluated the available studies involving asphalt fumes [IARC 1987] and concluded that the carcinogenicity of bitumens (shich include asphalt) is unclassifiable in humans.

NIOSH investigators (Kyle Steenland) agree with the review of Partanen and Bofetta [1994]. In addition to the studies' deficiencies already enumerated, insufficient latency for workers exposed to asphalt is also noted. Deficiencies of the Hansen [1989, 1991] studies include the unresolved controversy concerning possible exposure to coal tar, possible selection biases, and the appropriate beginning of person-time at risk.

REFERENCES

ACGIH [1991]. Asphalt fumes. In: Documentation of the threshold limit values and biological exposure indices, 6th ed. Vol. I. Cincinnati, OH: American Conference of Governmental Industrial Hygienists, pp. 95-96.

AI [1990a]. Report to OSHA and NIOSH: Status of Asphalt Industry Steering Committee research program on the health effects of asphalt fumes and recommendation for a worker health standard. Lexington, KY: Asphalt Institute.

AI [1991]. Final report. Asphalt industry cross sectional exposure assessment study. Text and Appendix A. Lexington, KY: Asphalt Institute, July 12, 1991 (prepared by Radian Corporation).

ASTM [1992]. 1992 Annual book of ASTM standards. Part 15-road, paving, bituminous materials; traveled surface characteristics. Philadelphia, PA: American Society for Testing and Materials.

Belanger PL, Elesh E [1979]. Health hazard evaluation report: Kentile Floors, Inc., Chicago, Illinois. Cincinnati, OH: U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, NIOSH Report No. HHE 78-73-612, NTIS No. PB-80-195-308.

Bender AP, Parker DL, Johnson RA, Anderson WK, Crozier MA, Williams AN, et al. [1987]. The Minnesota highway maintenance worker mortality study: 1945-1984. Preliminary report. Minneapolis, MN: Minnesota Department of Health, Section of Chronic Disease and Environmental Epidemiology, No Report No.

Bender AP, Parker DL, Johnson RA, Scharber WK, Williams AN, Marbury MC, Mandel JS [1989]. Minnesota highway maintenance worker study: cancer mortality. Am J Ind Med 15:545- 556.

Burgaz S, Bayhan A, Karakaya AE [1988]. Thioether excretion of workers exposed to bitumen fumes. Int Arch Occup Environ Health 60(5):347-349.

Engholm G, Englund A, Linder B [1991]. Mortality and cancer incidence in Swedish road paving asphalt workers and roofers. Health Environ 1:62-68.

Hansen ES [1989a]. Cancer incidence in an occupational cohort exposed to bitumen fumes. Scand J Work Envir Health 15(2):101-105.

Hansen ES [1989b]. Cancer mortality in the asphalt industry: a 10-year follow-up of an occupational cohort. Br J Ind Med 46(8):582-585.

Hansen ES [1991]. Mortality of mastic asphalt workers. Scand J Work Environ Health 17:20- 24.

Hansen ES [1992]. Cancer mortality and incidence in mastic asphalt workers. Author's reply [letters]. Scand J Work Environ Health 18:135-141.

Hatjian BA, Edwards, JW, Williams FM, Harrison J, Blain PG [1995a]. Risk assessment of occupational expousre to bitumen fumes in the road paving industries. Paper presented at the 1995 Pacific Rim Conference on Occupational and Environmental Health, Sydney, Australia, October4-6, 1995.

Hatjian BA, Edwards, JW, Harrison J, Williams FM, Blain PG [1995b]. Ambient, biological and biological effects monitoring of exposure to polycyclic aromatic hydrocarbons (PAHs). Toxicol letters 77:271-279.

Herbert R, Marcus M, Wolff MS, Perera FP, Andrews L, Godbold JH, et al. [1990]. Detection of adducts of deoxyribonucleic acid in white blood cells of roofers by ^{32}P-postlabeling. Scand J Work Environ Health 16:135-143.

ILO [1991]. Occupational Exposure Limits for Airborne Toxic Substances, 3rd edition. Values of Selected Countries Prepared from the ILO-CIS Data Base of Exposure Limits. Geneva: International Labour Organization, pp. 34-35.

Lafuente A, Mallol J [1987]. Urinary thioethers in workers exposed to asphalt: an impairment of glutathione S-transferase activity? J Tox Envir Health 21(4):533-534.

Lee BM, Baoyun Y, Herbert R, Hemminki K, Perera FP, Santella RM [1991]. Immunologic measurement of polycyclic aromatic hydrocarbon-albumin adducts in foundry workers and roofers. Scand J Work Environ Health 17:190-194.

Lunsford RA, Cooper CV [1989]. Characterization of petroleum asphalt fume fractions by gas chromatography/mass spectrometry. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health.

Machado ML, Beatty PW, Fetzer JC, Glickman AH, McGinnis T [1993]. Evaluation of the relationship between PAH content and mutagenic activity of fumes from roofing and paving asphalts and coal tar pitch. Fund Appl Toxicol 21:492-499.

Maizlish N, Beaumont J, Singleton J [1988]. Mortality among California highway workers. Am J Ind Med 13(3):363-379.

Minnesota Department of Health [1993]. The Minnesota highway maintenance worker mortality study 1945-1989. Final report, May 1, 1993. Minneapolis, MN: Minnesota Department of Health, Chronic Disease and Environmental Epidemiology.

Monarca S, Pasquini R, Scassellati Sforzolini G, Savino A, Bauleo FA, Angeli G [1987]. Environmental monitoring of mutagenic/carcinogenic hazards during road paving operations with bitumens. Intl Arch Occup Envir Health 59(4):393-402.

NAPA [1994]. Information presented during a meeting on February 7, 1994, between M. Acott, T. Brumagin, L. Miller, N. Beckthalt representing the National Asphalt Pavement Association and J. Wess, R. Niemeier, and C. Ellison from the Divisions of Standards Development and Technology Transfer, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Public Health Service, U.S. Department of Health and Human Services.

Niemeier RW, Thayer PS, Menzies KT, Von Thuna P, Moss CE, Burg J [1988]. A comparison of the skin carcinogenicity of condensed roofing asphalt and coal tar pitch fumes. In: Cooke M, Dennis AJ, eds. Polynuclear Aromatic Hydrocarbons: A Decade of Progress. Tenth International Symposium. Columbus, OH: Battelle Press, pp. 609-647.

NIOSH [1977a]. Criteria for a recommended standard: occupational exposure to asphalt fumes. Cincinnati, OH: U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, DHEW (NIOSH) Publication No. 78-106, NTIS Publication No. PB-277-333.

NIOSH [1983b]. National occupational exposure survey (NOES) 1981-1983: estimated total and female employees, actual

observation and trade-name exposure to asphalt and asphalt fumes. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Surveillance, Hazard Evaluation and Field Studies, Surveillance Branch. Unpublished data base; provisional data as of 7/1/90.

NIOSH [1988]. NIOSH testimony to the Department of Labor: Statement of the National Institute for Occupational Safety and Health, the public hearing on occupational exposure to asphalt fumes. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health.

NTP [1990]. NTP Results Report. Results and status information on all NTP chemicals produced from NTP Chemtrack System. Washington, DC: National Toxicological Program.

Puzinauskas VP, Corbett LW [1978]. Differences between petroleum asphalt, coal-tar asphalt, and road tar. College Park, MD: Asphalt Institute. Research Report No. 78-1, 31 pp.

Qian HW, Ong T, Whong WZ [1996]. Induction of micronuclei in cultured mammalian cells by fume condensates of roofing asphalt (submitted for publication in Am J Ind Med).

Sax NI, Lewis RJ, eds. [1987]. Hawley's condensed chemical dictionary. 11th ed. New York, NY: Van Nostrand Reinhold Co., pp. 102-103; 290; 320.

Sivak A, Menzies K, Beltis K, Worthington J, Ross A, Latta R [1989]. Assessment of the cocarcinogenic/promoting activity of asphalt fumes. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Biomedical and Behavioral Science. NIOSH Contract No. 200-83-2612, NTIS Publication No. PB-91-110-213.

Tharr DG [1982a]. Health Hazard Evaluation Report: Roofing Sites, Rochester and Buffalo, NY. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, NIOSH Report No. HETA 81-432-1105, NTIS No. PB-84-141-860/A02.

Tharr DG [1982b]. Health hazard evaluation report: McAlpin's Department Store, Cincinnati, Ohio. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, NIOSH Report No. HETA 82-034-1121.

Toraason M, Bohrman JS, Elmore E, Wyatt G, McGregor D, Willington SE, et al. [1991]. Inhibition of intercellular communication in Chinese hamster V79 cells by fractionated asphalt fume condensates. J Toxicol Environ Health 34:95-102.

Wey HE, Breitenstein MJ, Toraason MA [1992]. Inhibition of intercellular communication in human keratinocytes by fractionated asphalt fume condensates. Carcinogenesis 13(6):1047-1050.

Wolff MS, Herbert R, Marcus M, Rivera M, Landrigan PJ, Andrews LR [1989].

Polycyclic aromatic hydrocarbon (PAH) residues on skin in relation to air levels among roofers. Arch Envir Health 44(3):157-163.

Wong O, Bailey WJ, Amsel J [1992]. Cancer mortality and incidence in mastic asphalt workers [letters]. Scand J Work Environ Health 18:133-135.

Zey JN [1992a]. Letter of July 10, 1992, from J.N. Zey, Division of Surveillance, Hazard Evaluations, and Field Studies, to Forrest L. Cash, Quality Control Department, Barrett Paving Materials, Inc., Cincinnati, Ohio.

Zey JN [1992b]. Letter of July 27, 1992, from J.N. Zey, Division of Surveillance, Hazard Evaluations, and Field Studies, to Blair B. Bury, Vice President of Construction, Midwest Asphalt Corporation, Hopkins, Minnesota.

Zey JN [1992c]. Letter of August 14, 1992, from J.N. Zey, Division of Surveillance, Hazard Evaluations, and Field Studies, to Gerald D. Jordan, Branch Manager, APAC Mississippi, Inc., Greenville, Mississippi.

BRIEF REPORT

Carcinoma of the Pharynx and Tonsils in an Occupational Cohort of Asphalt Workers

Francesca Zanardi,[a] *Renata Salvarani,*[b] *Robin M.T. Cooke,*[a] *Roberta Pirastu,*[c] *Michela Baccini,*[d] *David Christiani,*[e] *Stefania Curti,*[a] *Alessandro Risi,*[f] *Anna Barbieri,*[a] *Giuseppe Barbieri,*[b] *Stefano Mattioli,*[a] *and Francesco Saverio Violante*[a]

Background: We investigated a possible association between pharyngeal/tonsillar carcinoma and mixed carcinogen exposures in an asphalt roll company in Italy that used asbestos until 1979, when a new factory was built using a different production process.

Methods: We evaluated all workers involved in the entire production history of the company, divided into two subcohorts based on exposure status (workers in the original factory, 1964–1979, and those who worked only in the new factory, 1980–1997). We ascertained the vital status of the study population in February 2001.

Results: Among the subset of workers in the earlier subcohort, there were five deaths from pharyngeal/tonsillar carcinoma for a standardized mortality ratio of 21 (95% confidence interval = 8.8–51). No cases were recorded among workers hired after 1979.

Conclusion: The increased standardized mortality ratio for this relatively rare cancer among workers exposed before 1979 may have been due to carcinogenic exposures at the plant.

(*Epidemiology* 2013;24: 100–103)

Submitted 10 June 2011; accepted 28 September 2012.

From the [a]Section of Occupational Medicine, Department of Internal Medicine, Geriatrics and Nephrology, Alma Mater Studiorum-University of Bologna, Bologna, Italy; [b]Occupational Health Unit, Bologna Local Health Authority, San Giorgio di Piano, Bologna, Italy; [c]Department of Biology and Biotechnology "Charles Darwin," La Sapienza University of Rome, Rome, Italy; [d]Department of Statistic, University of Florence, Florence, Italy; [e]Harvard University, Environmental Health and Epidemiology Departments, Boston, MA; and [f]Occupational Medicine Service, Alma Mater Studiorum-University of Bologna, Bologna, Italy.

Supported by INAIL (Istituto Nazionale per l'Assicurazione contro gli Infortuni sul Lavoro) Direzione Regionale Emilia-Romagna, Bologna, Italy; Regione Emilia-Romagna (Emilia-Romagna Regional Administration), Bologna, Italy; and the University of Bologna. The work of F.Z. was partially supported by the Epidemiology Master's course at the University of Turin.

The authors report no conflict of interests.

SDC Supplemental digital content is available through direct URL citations in the HTML and PDF versions of this article (www.epidem.com). This content is not peer-reviewed or copy-edited; it is the sole responsibility of the author.

Correspondence: Stefano Mattioli, Unità Operativa di Medicina del Lavoro, Policlinico S. Orsola-Malpighi, via Pelagio Palagi 9, I-40138 Bologna, Italy. E-mail: s.mattioli@unibo.it.

ISSN: 1044-3983/13/2401-0100
DOI: 10.1097/EDE.0b013e318276cc95

Pharyngeal carcinoma is relatively uncommon, most often present in men in the 6th or 7th decade of life.[1] Recently, its incidence has increased in developing countries.[2] Tobacco and alcohol are important risk factors, along with environmental, genetic, and viral factors.[3–7] Occupational associations between exposure to well-defined carcinogens, such as asbestos or polycyclic aromatic hydrocarbons (PAHs), and cancer of the pharynx have been suggested by several studies.[8–11] We investigated a possible association of pharyngeal/tonsillar carcinoma and mixed carcinogen exposures in a factory that produced asphalt rolls containing asbestos.

METHODS

Study Design

We conducted an occupational cohort study in which we evaluated all the workers—including subcontracted warehouse workers—involved in the 33-year production history of the company (based in the Emilia-Romagna region of Italy) from its first day of production on 7 January 1964 until the closure of the plant on 7 February 1997. We ascertained the vital status of the study population on 7 February 2001. Information on the workplace and available exposure and health monitoring data is summarized in the eAppendix, http://links.lww.com/EDE/A627 (part 1). In 1979, the original factory was closed because of a fire, and the management agreed to eliminate asbestos from all production processes. Production of asphalt roofing rolls resumed in 1980 in a new factory constructed nearby and broadly in line with modern occupational health and safety standards. These changes allowed us to evaluate two subcohorts of workers based on exposure status: (1) those who started work between January 1964 and June 1979, who were exposed to the old factory in which asbestos was used; (2) those who worked only in the new factory (ie, after June 1979). We assessed cause-specific mortality experienced by these two subcohorts, calculating standardized mortality ratios (SMRs) for carcinoma of the lip, oral cavity, and pharynx (international classification of disease ICD-9, 140–149).

Data Collection and Statistical Analysis

We used company records to extract job history information of all workers. For each cohort member, we sent survey cards to local government offices to ascertain vital status and current residence or, for those who had died, date, place, and

cause of death. We requested copies of death certificates for the deceased and embarkation details for workers who had left the country. In each subcohort, subgroups of workers were categorized by occupational exposure characteristics, based on job descriptions contained in factory inspection reports: blue-collar production sector workers (including subcontracted warehouse workers); other blue-collar workers; white-collar workers. This assessment was made without knowledge of case status.

We used Student's *t* test to compare the mean age at initial employment and the mean duration of employment between the two subcohorts. We tabulated observed cause-specific deaths alongside expected numbers specific for sex, age, and calendar period (5-year classification); we then calculated the SMR in the entire cohort, based on mortality rates in the resident population of the Emilia-Romagna region.[11] For cause-specific categories of interest (death from all causes; all cancers; cancer of the lip, oral cavity, and pharynx; gastric carcinoma; lung cancer; cardiovascular diseases), we also calculated SMR by subcohort (before or after 1979), occupational exposure (production-line, other blue-collar, white-collar), and latency. For all estimates, we used the Poisson distribution to calculate 95% confidence intervals (CIs).[12] All analyses were conducted using Stata 11.0 (Stata Corporation, Texas, TX).

RESULTS

Study Cohort

The study cohort comprised 415 workers, including 52 subcontracted warehouse workers who were occupationally exposed to the production sector. The majority of workers (71%, $n = 295$) were in the subcohort who worked in the old factory.

Table 1 shows the occupational characteristics of the subcohorts. Mean age at initial employment was 29.3 years in both subcohorts. Mean number of working years was greater in the earlier subcohort (overall, 10.6 vs. 3.6 years; among production-line workers, 11.6 vs. 4.5 years).

Outcome

Vital status at the end of follow-up is reported in Table 2; loss to follow-up was 1.2% (5/415). Forty deaths were recorded during a total of 9948 person-years of follow-up. Cancer was the most common underlying cause of death (63%, 25/40). Carcinoma of the pharynx/tonsils caused 5 (25%) of the cancer-related deaths (eTable 1, http://links.lww.com/EDE/A627); although health officials were aware of three cases, an additional two cases were uncovered during survival data collection. Cause-specific SMRs for the entire cohort are presented in eTable 2 (http://links.lww.com/EDE/A627). Among the group of workers exposed to the production sector where asbestos was used, the SMR for carcinoma of the lip, oral cavity, and pharynx was 21 (95% CI = 8.8–51; Table 3).

We also recorded an approximately threefold excess mortality rate for gastric cancer, but no evidence of increased mortality from lung cancer or cardiovascular diseases. Mantel-Haenszel estimates of rate ratios adjusted for latency and cause-specific SMRs according to latency are presented in eTables 3 and 4 (http://links.lww.com/EDE/A627).

TABLE 1. Occupational Characteristics of the Entire Cohort and the Two Main Subcohorts

	Entered Employment Before June 1979			Employed Only After June 1979		
	Men No.	Women No.	Total No.	Men No.	Women No.	Total No.
Blue-collar workers exposed to the production line	104	0	104	41	0	41
Production-line workers	67	0	67	26	0	26
Smelters	17	0	17	5	0	5
Pasters	11	0	11	0	0	0
Rollers	37	0	37	21	0	21
Shredding machine operators	2	0	2	0	0	0
Subcontracted warehouse workers (handling raw materials)	37	0	37	15	0	15
Nonproduction-line blue-collar workers	60	3	63	17	1	18
End-product warehouse workers (not handling raw materials)	9	0	9	6	0	6
Machinery operators	11	0	11	0	0	0
Factory maintenance staff	21	0	21	1	0	1
Other[a]	19	3	22	10	1	11
White-collar workers	75	53	128	46	15	61
Office clerks	21	35	56	13	3	16
Representatives	54	18	72	33	12	45
Total	239	56	295	104	16	120

[a]Supervisors, technicians, maintenance workers, canteen workers, office cleaning staff, resident porter/night watchman.

TABLE 2. Vital Status on 7 February 2001 and Person-Years of Follow-Up

	Men (n = 343)	Women (n = 72)	Overall (n = 415)
Alive	298	72	370
Dead	40	—	40
From known causes	39	—	39
From unknown cause	1	—	1
Lost to follow-up	5	—	5
Person-years	8,156	1,792	9,948
Up to 40 years of age	4,612	1,543	6,155
After 40 years of age	3,544	249	3,793

DISCUSSION

Cohort analysis of a small cluster of cases of a rare cancer in a single factory can be hypothesis-generating. The SMR was so likely increased (a 21-fold excess) that we have grounds to believe that the associations observed are unlikely to be due to chance, even if it is not clear which of the various occupational exposures could be implicated.

The study had been initiated after specific concerns about widespread chronic inflammatory diseases of the upper airways among the production-line workers and a cluster of cases of cancer of the pharynx/tonsils. All five subjects affected by cancer of the pharynx/tonsils were among the well-defined production-line workers who experienced, in the old factory, heavy industrial coexposure to a mixture of chrysotile asbestos fibers, bitumen fumes, solvents, PAH, and other dusts, all of which can be carcinogenic.[13–18]

Multiple chemical factors found in bitumen fumes could combine to influence the onset of neoplastic disease.[19–21] As in most historical cohort studies, we were unable to distinguish the work-related etiologic contribution from individual factors[1,8,22] such as smoking and low socioeconomic status. All five workers shared a relatively young age of onset, within 10

TABLE 3. Cause-Specific SMR in the Two Subcohorts Stratified by Occupational Category (Reference Population, Regione Emilia-Romagna)

	Entered Employment Before June 1979			Employed Only After June 1979		
	Observed	Expected	SMR (95% CI)	Observed	Expected	SMR (95% CI)
All causes						
Overall	34	40.8	0.8 (0.6–1.2)	6	5.5	1.1 (0.5–2.4)
Blue-collar workers on the production line	26	22.5	1.2 (0.8–1.7)	3	2.0	1.5 (0.5–4.6)
Nonproduction-line blue-collar workers	6	11.5	0.5 (0.2–1.2)	1	0.6	1.7 (0.2–11.9)
White-collar workers	2	6.8	0.3 (0.1–1.2)	2	2.9	0.7 (0.2–2.8)
All cancers						
Overall	23	15.7	1.5 (1.0–2.2)	2	1.9	1.1 (0.3–4.2)
Blue-collar workers on the production line	22	8.6	2.6 (1.7–3.9)	1	0.7	1.4 (0.2–9.9)
Nonproduction-line blue-collar workers	1	4.5	0.2 (0.03–1.57)	0	0.2	—
White-collar workers	0	2.6	—	1	1.0	1.0 (0.1–7.2)
Lip, oral cavity, and pharynx cancer						
Overall	5	0.4	11.4 (4.8–27.4)	0	0.06	—
Blue-collar workers on the production line	5	0.2	21.1 (8.8–50.7)	0	0.02	—
Nonproduction-line blue-collar workers	0	0.1	—	0	0.01	—
White-collar workers	0	0.1	—	0	0.03	—
Stomach cancer						
Overall	5	1.6	3.0 (1.3–7.3)	0	0.2	—
Blue-collar workers on the production line	5	0.9	5.3 (2.2–12.8)	0	0.06	—
Nonproduction-line blue-collar workers	0	0.5	—	0	0.01	—
White-collar workers	0	0.2	—	0	0.09	—
Trachea, bronchus, and lung cancer						
Overall	5	5.0	1.0 (0.4–2.4)	0	0.6	—
Blue-collar workers on the production line	4	2.9	1.4 (0.5–3.7)	0	0.2	—
Nonproduction-line blue-collar workers	1	1.5	0.7 (0.1–4.8)	0	0.1	—
White-collar workers	0	0.7	—	0	0.3	—
Circulatory system diseases						
Overall	7	13.0	0.5 (0.3–1.1)	0	1.3	—
Blue-collar workers on the production line	3	7.8	0.4 (0.1–1.2)	0	0.5	—
Nonproduction-line blue-collar workers	4	3.7	1.1 (0.4–2.9)	0	0.1	—
White-collar workers	0	1.5	—	0	0.7	—

years of first being hired at the plant. By contrast, no further case of cancer of the pharynx or oral cavity emerged among the subcohort of workers exposed only to the new factory, in which there was little or no use of asbestos and presumably much lower levels to bitumen fumes, solvents, PAH, and dusts.

Absence of excess rates of lung cancer, especially in the "exposed" subcohort in which the follow-up was longer, is in line with a large International Agency for Research on Cancer study, which showed no evidence of bitumen causing lung cancer.[23] Similarly, the apparent excess in gastric cancer among the production-line workers in the exposed subcohort could either reflect the increased risks reported for asphalt workers in general[24–26] or be related to a particular coexposure. A healthy worker effect was observable in both subcohorts. However, the hypothesis that the particular conditions found in the original factory constituted a risk factor for pharyngeal/oral cancer (and probably also gastric cancer) is reinforced by the observation that within the highly exposed production-line workers of the earlier subcohort, cause-specific SMRs were remarkably increased for these particular cancers, but not for other causes of death such as cardiovascular disease.

Study Limitations

The limited exposure information did not allow dose-response analysis and hampered formulation of more detailed etiologic hypotheses. The small number of person-years and the relatively short follow-up impeded assessment of tumors with a long latency, such as mesothelioma.

In summary, we observed a cluster of pharyngeal/tonsillar carcinoma among workers exposed to the production sector of a factory producing asphalt rolls when asbestos was being used as a binding agent. The magnitude of the SMR for this rare type of cancer suggests that the events may not have been due to chance. Although it is unclear which mixed carcinogen exposures were implicated, these observations may be relevant to the etiology of pharyngeal/tonsillar carcinoma. Industrial cohort studies in settings in which asbestos was used in asphalt production could provide further information regarding the cancer-related risks associated with similar industrial mixed carcinogen exposures.

ACKNOWLEDGMENTS

We thank Chiara Scardoni for translation work.

REFERENCES

1. Parkin DM, Whelan S, Ferlay J, Teppo L, Thomas DB. *Cancer Incidence in Five Continents. Vol. VIII*. Lyon, France: International Agency for Research on Cancer. Sci Publ. No l55. 2002:364–365.
2. Franceschi S, Bidoli E, Herrero R, Muñoz N. Comparison of cancers of the oral cavity and pharynx worldwide: etiological clues. *Oral Oncol*. 2000;36:106–115.
3. IARC. *Tobacco Smoking. Monographs on the Evaluation of Carcinogenic Risks to Human*. Vol. 38. Lyon, France: International Agency for Research on Cancer; 1986.
4. IARC. *Alcohol Drinking. Monographs on the Evaluation of Carcinogenic Risks to Humans*. Vol. 44. Lyon, France: International Agency for Research on Cancer; 1988.
5. IARC. *Tobacco Smoking and Involuntary Smoking. Monographs on the Evaluation of Carcinogenic Risks to Human*. Vol. 83. Lyon, France: International Agency for Research on Cancer; 2003.
6. IARC. *Formaldehyde, 2-Butoxyethanol and 1-tert-Butoxy-2-propanol. Monographs on the Evaluation of Carcinogenic Risks to Humans*. Vol. 88. Lyon, France: International Agency for Research on Cancer; 2004.
7. Mucci L, Adami HO. Oral and pharyngeal cancer. In: Trichopoulos D, Adami HO, Hunter D, eds. *Textbook of Cancer Epidemiology*. New York, NY: Oxford University Press; 2002:115–128.
8. Berrino F, Richiardi L, Boffetta P, et al. Milan JEM Working Group. Occupation and larynx and hypopharynx cancer: a job-exposure matrix approach in an international case-control study in France, Italy, Spain and Switzerland. *Cancer Causes Control*. 2003;14:213–223.
9. Gustavsson P, Jakobsson R, Johansson H, Lewin F, Norell S, Rutkvist LE. Occupational exposures and squamous cell carcinoma of the oral cavity, pharynx, larynx, and oesophagus: a case-control study in Sweden. *Occup Environ Med*. 1998;55:393–400.
10. Marchand JL, Luce D, Leclerc A, et al. Laryngeal and hypopharyngeal cancer and occupational exposure to asbestos and man-made vitreous fibers: results of a case-control study. *Am J Ind Med*. 2000;37:581–589.
11. Straif K, Benbrahim-Tallaa L, Baan R, et al. WHO International Agency for Research on Cancer Monograph Working Group. A review of human carcinogens–part C: metals, arsenic, dusts, and fibres. *Lancet Oncol*. 2009;10:453–454.
12. Checkoway H, Pearce N, Kriebel D. *Research Methods in Occupational Epidemiology*. 2nd ed. Oxford, UK: Oxford University Press; 2004.
13. Binet S, Pfohl-Leszkowicz A, Brandt H, Lafontaine M, Castegnaro M. Bitumen fumes: review of work on the potential risk to workers and the present knowledge on its origin. *Sci Total Environ*. 2002;300:37–49.
14. Boffetta P, Burstyn I. Studies of carcinogenicity of bitumen fume in humans. *Am J Ind Med*. 2003;43:1–2.
15. Boffetta P, Burstyn I, Partanen T, et al. Cancer mortality among European asphalt workers: an international epidemiological study. I. Results of the analysis based on job titles. *Am J Ind Med*. 2003;43:18–27.
16. Burstyn I, Randem B, Lien JE, Langård S, Kromhout H. Bitumen, polycyclic aromatic hydrocarbons and vehicle exhaust: exposure levels and controls among Norwegian asphalt workers. *Ann Occup Hyg*. 2002;46:79–87.
17. Herrick RF, McClean MD, Meeker JD, Zwack L, Hanley K. Physical and chemical characterization of asphalt (bitumen) paving exposures. *J Occup Environ Hyg*. 2007;4(suppl 1):209–216.
18. Wang J, Lewis DM, Castranova V, et al. Characterization of asphalt fume composition under simulated road paving conditions by GC/MS and microflow LC/quadrupole time-of-flight MS. *Anal Chem*. 2001;73:3691–3700.
19. Finkelstein MM. Asbestos-associated cancers in the Ontario refinery and petrochemical sector. *Am J Ind Med*. 1996;30:610–615.
20. Melius J. Asphalt–a continuing challenge. *Am J Ind Med*. 2003;43:235–236.
21. Schulte PA. Gaps in scientific knowledge about the carcinogenic potential of asphalt/bitumen fumes. *J Occup Environ Hyg*. 2007;4(suppl 1):3–5.
22. Friborg JT, Yuan JM, Wang R, Koh WP, Lee HP, Yu MC. A prospective study of tobacco and alcohol use as risk factors for pharyngeal carcinomas in Singapore Chinese. *Cancer*. 2007;109:1183–1191.
23. Agostini M, Ferro G, Olsson A, et al. Exposure assessment for a nested case-control study of lung cancer among European asphalt workers. *Ann Occup Hyg*. 2010;54:813–823.
24. Hansen ES. Cancer incidence in an occupational cohort exposed to bitumen fumes. *Scand J Work Environ Health*. 1989;15:101–105.
25. Stücker I, Meguellati D, Boffetta P, Cénée S, Margelin D, Hémon D. Cohort mortality study among French asphalt workers. *Am J Ind Med*. 2003;43:58–68.
26. Cocco P, Ward MH, Dosemeci M. Risk of stomach cancer associated with 12 workplace hazards: analysis of death certificates from 24 states of the United States with the aid of job exposure matrices. *Occup Environ Med*. 1999;56:781–787.

American Journal of Industrial Medicine 26:721–740 (1994)

ARTICLES

Cancer Risk In Asphalt Workers and Roofers: Review and Meta-Analysis of Epidemiologic Studies

Timo Partanen, PhD, MSc, and Paolo Boffetta, MD, MPH

Twenty epidemiologic studies have described cancer risk in asphalt workers and roofers in various countries. A current concern for these workers is the potential carcinogenicity posed by inhalation of bitumen fumes or dermal exposure to bitumens. Bitumens are chemically different from many carcinogenic coal-tar based materials. Both have been employed in road paving and waterproofing. We examined and combined the results of the epidemiologic studies conducted on asphalt workers and roofers. We examined the cancer risk separately in three broad job categories: 1) roofers (exposed to bitumen fumes and previously often to coal-tar fumes); 2) highway maintenance workers (HMWs) and road pavers (exposed to bitumen fumes as well as possibly coal-tar fumes previously); and 3) miscellaneous and unspecified bitumen/asphalt workers. In roofers, an increased risk was suggested for cancers of the lung (aggregated relative risk 1.8, 95% confidence interval 1.5–2.1), stomach (1.7, 1.1–2.5), nonmelanoma skin (4.0, 0.8–12), and leukemia (1.7, 0.9–2.9). Some of the excesses may be attributable to polycyclic aromatic hydrocarbons (PAH) from coal-tar products. The aggregated relative risks in road pavers and HMWs were consistently lower than in roofers for cancers of the lung (0.9, 0.8–1.0), stomach (1.1, 0.8–1.5), bladder (1.2, 0.7–1.8), skin (2.2, 1.2–3.7), and leukemias (1.3, 0.9–1.8). Their risk of skin cancer was significantly increased, based on one study. Miscellaneous and unspecified workers had a significant excess (1.5, 1.2–1.8) of lung cancer. The data were poorly focused to address the carcinogenicity of bitumen fumes, as contrasted with tar-derived exposures. For the prospect of shedding more light on the bitumen-cancer controversy, the feasibility of a powerful multicenter cohort is currently being studied by the International Agency for Research on Cancer (IARC). © 1994 Wiley-Liss, Inc.

Key words: asphalt, bitumen, tar, roofers, pavers, epidemiology, cancer, review, meta-analysis

Unit of Analytical Epidemiology, International Agency for Research on Cancer, Lyon, France (T.P., P.B.).
Department of Epidemiology and Biostatistics, Institute of Occupational Health, Helsinki, Finland (T.P.).
Address reprint requests to Paolo Boffetta, International Agency for Research on Cancer, 150 Cours Albert-Thomas, F-69372 Lyon Cedex 08, France.

Abbreviations: BaP, Benzo (a) pyrene; CI, confidence interval; CONCAWE, Oil Companies' European Organisation for Environmental and Health Protection; DF, degrees of freedom; E, expected number of exposed cases (i.e., cases falling in an occupational category); EAPA, European Asphalt Pavement Association; EUROBITUME, European Bitumen Association; HMW, highway maintenance worker; IARC, International Agency for Research on Cancer; O, observed number of exposed cases (i.e., cases falling in an occupational category); OR, odds ratio; PAH, polycyclic aromatic hydrocarbon; PMR, proportional mortality ratio; RR, relative risk; SIR, standardized incidence ratio; SMR, standardized mortality ratio; TWA, time-weighted average.

Accepted for publication February 27, 1994.

Risk of cancer/ illness from asphalt

CHEJ Asphalt Fact Pack 92

Link back to "Health"

Science says cancers and illness are linked to asphalt and hot asphalt fumes:

****

A powerpoint presentation on health and asphalt is available as an attachment at the bottom on this page.
****

There is a growing list of scientific papers that conclude exposure to hot asphalt emissions can significantly increase the incidence of many types of cancers above background.

There are no studies on children near asphalt plants, although there are various reasons (age, metabolic rate, body proportions, activity levels, growth rates) why children can be more susceptible to carcinogens than adults.

Like many long term studies on cancer, it may take a generation or so before we are aware of the full consequences and dangers of exposure to certain chemicals from asphalt plants. We expect the evidence for asphalt to lag several decades behind that for cigarette smoking (only recently did "big tobacco" stop refuting the health risks from smoking). Many new scientific papers now find a connection between cancer and asphalt, but some of the early and some current studies do not detect associations or strong associations.

It is universally agreed that exposure to chemicals in asphalt causes cancer in animals. Components of asphalt fumes, including benzene are highly carinogenic (we even know how these chemicals biologically induce cancer). Overall, the fact that we are now seeing evidence of increased cancer risk in humans from a number of studies on asphalt is troubling and points to even stronger associations as populations age and we get more data.

So if some papers say yes, and some no, about asphalt causing cancer in humans, which do we go with? Some studies say several fold increase, some say 10-50% increase in cancer rates. What do we do? Toss a coin, take a bet about our children's future? If we bet no effect, and find out that these negative papers were funded by asphalt industries, then we've failed to take a precautionary approach for our children. If we bet on low levels of cancer rates, then how many collateral deaths are worth it for the sake of having an asphalt plant close by?

Overall, all agree that asphalt fumes contain strongly carcinogenic material (Polycyclic aromatic hydrocarbons or PAHs). The disagreements seem to be over the level of exposure that causes a specific and public health risk. This is why the issue of still air over Kunda Park especially in winter is such an important issue, as the still air and temperature inversions will greatly magnify the concentration of carcinogens, and their risk.

Not safe at any level?

Agencies set "safe levels" such as 5 parts per billion in drinking water for benzene (benzene is a carcenogen produced by asphalt plants). However, biological researches are starting to understand there is no such thing as safe levels for some carcinogens like benzene because some carcinogens at any level can damage cells.

Considering that the consequence could be very high (i.e. cancers and health), it is surprising that our government authorities have not undertaken a comprehensive health assessment of this proposal, including an up to date review of the literature, including epidemiological, biochemical, pharmacological, and molecular biology data, and have not sought expert advice from current leaders/scientists in the area (eg, the Boston Health Department and Boston Researchers like Prof. Mike McClean). This sort of review probably should be required under council's "duty of care".

Indeed, with this awareness of impact to health, some residents have asked Sunshine Coast Regional Council to exercise their duty of care to the residents and children and protect them from avoidable exposure to harmful carcinogens, in particular those emanating from the proposed Kunda Park Asphalt Plant. They have asked that council undertake a **site specific health risk assessment** for the community, for food shops, and commercial workers, and include the issue of temperature inversions in the study.

The Pottstown Mercury (pottsmerc.com), Serving Pottstown, PA

Print Page

News

Asphalt plant, and its pollution potential, part of quarry deal

Sunday, April 5, 2009

By Evan Brandt
ebrandt@pottsmerc.com

NEW HANOVER — Signing a proposed settlement with Gibraltar Rock to end litigation over the quarry the company wants to build off Route 73 may obligate the township to support an application for an asphalt plant likely to spew dangerous chemicals into the air.

According to the proposed agreement made public by the supervisors, if the settlement is approved, "the township shall support Gibraltar Rock's applications for air quality permits for a hot mix bituminous concrete plant," among other applications.

In the proposed settlement, the township would also "agree that it will not object to or oppose any permit applications or permit modification applications by Gibraltar Rock related to the activities contemplated by this agreement."

Robert Brant, the attorney who has been negotiating on behalf of the township with Gibraltar Rock's lawyers for more than a year, said he is not sure that language would prevent the supervisors from objecting to increased air pollution from an asphalt plant.

"I don't know if the agreement gives (Gibraltar Rock) carte blanche to any permits at all times," he said.

Brant said in the past, when discussing pollution from the blasting and rock crushing operation neces

sary to a quarry, the township used a consultant who specializes in air pollution.

But Christopher Mullaney, the lawyer representing the Ban the Quarry group, thinks differently.

What will govern what the township government can say and do is not what a consultant says

about the air pollution that comes with an asphalt plant, but what the settlement language says the township must do, Mullaney suggested.

"The proposed settlement would mandate the township supervisors to support that application," Mullaney said. "It says 'shall' right there in the language. That doesn't give you a lot of options. I think the township wouldn't be allowed to object."

The substances to which township officials might not be able to object include an alphabet soup of chemicals classified by the federal government as hazardous air pollutants, or HAPs, and volatile organic compounds, also known as VOCs.

Lynda Rebarchak, a spokeswoman for the Pennsylvania Department of Environmental Protection's Southeast Regional Office in Norristown, said no application related to the asphalt plant has been filed yet by Gibraltar, but one is expected.

The permit her office oversees is an air quality permit, one of two the company had to obtain from the DEP for the mining portion of the project. A separate permit, complete with public hearings, would be required for the asphalt plant.

Hot-mix asphalt plants come in several varieties, with several types of fuel to provide the heat — some are even mobile — and as such their emission profiles differ.

Rebarchak was reluctant to characterize what sort of emissions might be regulated under a permit for which no application has been made, but said generally, every asphalt plant emits carbon monoxide, nitrogen oxide, sulfur dioxide — known as "NOX and SOX" — and volatile organic compounds.

The U.S. Environmental Protection Agency is a little more specific, issuing in 2000 a report assessing the most common emissions from hot-mix asphalt plants.

The primary variables that dictate emissions are the type of mixer, "drum mix or batch mix," the type of fuel used for the dryer, oil or natural gas and the size of the plant or how much material it processes, according to the EPA study.

About 70 to 90 percent of the plants surveyed in 1996 use natural gas as a dryer fuel, the EPA found.

According to the EPA, "a typical batch mix plant using a No. 2 fuel oil-fired dryer emits over 74,000 pounds per year of criteria pollutants and a typical batch mix plant using a natural gas fired dryer emits over 56,000 pounds per year od criteria pollutants of which approximately 41,000 pounds per year are (carbon monoxide) and approximately 10,700 pounds per year are PM-10 emissions."

PM-10 emissions are particulate matter, or dust, of 10 micrometers or less.

]

In addition to the larger volume pollutants mentioned above, the EPA also found that a typical batch mix plant emits 1,500 pounds per year of volatile organic compounds, whereas the average drum mix plant emits 10,000 pounds of VOCs per year.

For example, in terms of sulfur dioxide, the average drum mix plant emits 2,200 pounds if its dryer is oil fired and 710 pounds if it uses gas. The average batch mix plant emits 8,600 pounds of sulfur dioxide a year if its dryer is oil-fired and 480 pounds per year if it uses gas, the EPA concluded.

As for volatile hazardous air pollutants, the typical drum plant emits 1,800 pounds into the air per year if its dryer is oil-fired and 1,200 pounds per year if it uses gas.

The average batch mix plant emits 760 pounds of volatile hazardous air pollutants into the air, no matter what fuel is used in its dryer, the EPA reported.

The effects of these pollutants on human health is not entirely explored or known, although many of them are believed by the federal government to exacerbate breathing problems and asthma, and some are even considered likely carcinogens.

For example, at high concentrations sulfur dioxide "is considered immediately dangerous to life and health," according to the federal Agency for Toxic Substance and Disease Registry.

According to data collected from the Occupancy Health and Safety Administration "long term exposure to persistent levels of sulfur dioxide can also affect your health."

Tests using low-level exposure on guinea pigs found changes in their ability to breath deeply or as much air per breath, reports ATSDR, which also noted "children may be exposed to more sulfur dioxide than adults because they breath more air for their body weight than adults do."

Also, "it is known that exercising asthmatics are sensitive to low concentrations of sulfur dioxide," the agency reported.

Concerns about the effect of quarry operations on asthmatic children at New Hanover/Upper Frederick Elementary School and the Perkiomen Academy, both of which are within a mile of the site of the proposed quarry and asphalt plant, have been raised by residents at several public meetings.

In addition to small amounts of lead, mercury and arsenic, the EPA study also found most hot mix asphalt plants also emit a long list of other volatile hazardous air pollutants in varying amounts.

Among the highest are ethylbenzene, xylene and formaldehyde.

According to ATSDR "health statements" on ethylbenzene, long-term exposure in the air caused kidney damage in animals and "potentially irreversible damage to the inner ear in the

hearing of animals."

More worrisome is the fact that the International Agency for Research on Cancer "has determined long-term exposure to ethylbenzene may cause cancer in humans," according to the ATSDR.

According to the same agency, the effects of long-term exposure to xylene at low levels is not well-studied, but there is some information if can cause damage to the nervous system if inhaled.

"Animal studies showed that xylene absorbed by the mother can cross the placenta and reach the fetus" and some studies found the offspring of those mothers sometimes have reduced body weight and trouble with motor coordination.

The ATSDR also reports that the U.S. Department of Health and Human Services "has determined that formaldehyde may reasonably be anticipated to be a numan carcinogen." Similarly, the International Agency for Research on Cancer "has determined that formaldehyde is probably carcinogenic to humans," a conclusion also reached by the EPA.

"Some studies of humans exposed to lower amounts of formaldehyde in workplace air found more cases of the cancer of the nose and throat than expected," but other studies did not find the same results, the ATSDR health statement says.

While all of the information provided here is in the public domain, some question whether the New Hanover Township Supervisors would be permitted to raise objections based on these or other factors if the proposed settlement is ultimately approved.

There are several asphalt plants in the region, including in Upper Frederick and in Bechtelsville.

Ron Comisky, executive director of the Pennsylvania Asphalt Pavement Association, said that there are about 137 asphalt plants in the state and that there has been no significant increase or decrease in their number in the past 10 years.

URL: http://www.pottsmerc.com/articles/2009/04/05/news/srv0000005039118.prt

]

BLUE RIDGE ENVIRONMENTAL DEFENSE LEAGUE
Central office: PO Box 88 Glendale Springs, NC 28629 (910) 982-2691 fax: (910) 982-2954

FOR IMMEDIATE RELEASE
APRIL 9, 1997

CONTACTS:
Louis Zeller (910) 982-2691
Janet M. Zeller (910) 982-2691
Michelle Kilborne (704) 262-3245
Dale Thompson (704) 733-2478

GROUPS CHARGE MAYMEAD WITH INTENTIONAL VIOLATIONS

At a press conference today in Boone, representatives of three citizens' groups charged that Maymead Materials, Inc. has a history of noncompliance with Tennessee and Virginia regulations in their operation of asphalt plants. Representatives of the Blue Ridge Environmental Defense League (BREDL), Pineola Concerned Citizens (PCC), and Citizens Against Pollution (CAP) released Notices Of Violations, citizens' complaint records, and legal enforcement actions from state files in Johnson City, Tennessee and Abingdon, Virginia.

Records show violations of permits including excess hourly and total annual asphalt production at plants in Russell County, Virginia and Mountain City, Tennessee. Other Notices Of Violations included burning #4 waste oil instead of #2 Diesel and visible emissions of 52%. Complaint files included nearby residents reporting frequent odors and dust.

Of particular concern to BREDL, PCC, and CAP is Maymead's long history of what appears to be intentional violations. "How can any operator burn or buy waste oil without knowing it?" said Lou Zeller, community organizer for BREDL. "In Pineola, NC and now in Mountain City, TN Maymead has been caught burning #4 oil which emits many

-more-

more toxic air pollutants than their permitted fuel."

In a letter dated April 9 to Alan Klimek, head of the NC Division of Air Quality, Zeller wrote, "Maymead cannot be trusted to operate asphalt plants in residential communities in North Carolina; their history of violations in Tennessee, Virginia, and North Carolina reveal a disturbing pattern of willful noncompliance."

Zeller's letter concluded: "On behalf of the Board of Directors of the Blue Ridge Environmental Defense League and on behalf of the Pineola Concerned Citizens and Citizens Against Pollution, I request that the NC Division of Air Quality deny Maymead's permit application for the Roby Green site and re-open the permit for the Pineola plant. Nothing short of an on-site inspector is adequate to enforce compliance by a company of Maymead's character."

Representatives of Pineola Concerned Citizens are gathering data about the adverse health impacts suffered by neighbors of the Maymead plant. Today CAP launched a health survey which their members will carry door-to-door in the residential communities near Roby Green Road, the proposed site for a new Maymead plant. This survey is designed to identify high-risk citizens who have respiratory diseases, pregnancy, etc.

Dale Thompson of PCC reported that neighbors of the recently re-started Pineola plant have experienced odors and witnessed smoke. He also reported that he and his wife Nancy have had a reduced property value because of the asphalt plant. Other Pineola property owners have also been assessed reduced property taxes.

-end-

Significant Issues Pertaining to Poor Air Quality in the Calgary Area...

1 - NW Asphalt Plant

Prime Minister Harper has campaigned on the devotion to real, result oriented, environmental policies and against race-biased policies. The question needs to be asked are we violating both of those principles right? in his own riding, allowing this plant to behave in a way that is damaging the environment and making people in the surrounding communities ill.

"I had noticed I was breathing a little better and had asked a neighbor if they had also noticed it and their comment was yes –since the plant has closed for the season"

"How much longer do we have to wait with this plant, when it would have been shut down if located in any other location?"

What to do if you Smell a Petroleum/Diesel-like Odour?

Environment Alberta cannot keep up with volume of calls, and will only be taking down a count of the number of calls. Please call their Hotline at:

1-800-222-6514.

Breaking News

3/30/2007 - Calgary Clean Air's review of the Final Screening Report for Sarcee Asphalt Plant. " Ultimately, while there is little debate among residents that the major contributing factor to offensive odour and other emissions from this particular facility is its use of "used/waste oil" as its fuel and that a switch to a cleaner fuel such as natural gas or propane would virtually eliminate the odour problem at this facility, and while ***INAC does not compel the Sarcee Asphalt Plant to switch to a cleaner burning fuel***, we must view INAC's requirement that this plant not "cause an offensive odour", with healthy skepticism that the sources of the toxic fumes in our neighbourhoods will actually be mitigated. " Read it here

---

3/16/2007 - CEAA Environmental Screening Report for Sarcee Asphalt Plant is completed - "*It is critical that all recommended mitigation measures be implemented.*" Including... "*Install and properly operate necessary commercially available pollution control technologies to successfully mitigate offensive odours and emissions. Odours from the plant should not be offensive.*" read the entire report

---

3/1/2007 - Federal Clean Air Act - Interestingly enough, Jim Prentice's (Minister for Indian Affairs) official web site mentions his interest on Canada's Clean Air Act (read more about the Act here).

1/08/2007 - Here is the letter we sent to Hon. John Baird, the new Minister of the Environment. download Word version here

previous news below

Why Does this Smell Occur in the SW?

There is an unlicensed asphalt plant located on the Tsuu T'ina Nation, which is owned and operated by the Tsuu T'ina Council and CARMACKS Enterprises. This plant has been operating without any required permits for over 2 1/2 years.

According to the Prime Ministers office "To remain competitive with other asphalt plants in Calgary, the Tsuu T'ina Nation (Council's & Carmacks) plant is using ***waste oil***, whereas ***other producers now use only propane or natural gas***, which considerably diminishes the odour emanating from their plants."

Are Asphalt Fumes Dangerous to our Health?

Asphalt Fumes are Known Toxins. "Asphalt processing and asphalt roofing manufacturing facilities are major sources of hazardous air pollutants such as formaldehyde, hexane, phenol, polycyclic organic matter, and toluene. Exposure to these air toxics may *cause cancer, central nervous system problems, liver damage, respiratory problems and skin irritation.*" [EPA]

more on Health Issues

Previous News on the Asphalt Plant

10/23/06 - Sleepless in South Calgary - Calgary Herald - ....the plant burns dirty, used oil rather than cleaner natural gas. The federal government is now keeping the plant open while it assesses emissions. Federal Indian Affairs Minister Jim Prentice wants to wait for the results of stack tests. If I built an asphalt plant, without a permit that burned dirty fuel upwind from a residential area and 500 Calgarians complained it was making their children sick, would I be granted that leniency? Download

10/19/06 - CBC Radio 1 Eye Opener - "I've been thinking about how societies encourage good behaviour. Most start with the golden rule: treat others as you would like to be treated. The Sarcee Asphalt plant, started up in 2004, upwind of Oakridge community, burns dirty bunker oil instead of cleaner natural gas or propane. The Tsuu T'ina just never bothered to get the required federal permits and have run the plant illegally for two and a half years. After over 500 Calgarians complained that the plant is making their kids ill, Indian and Northern Affairs Minister Jim Prentice said he wouldn't shut down the illegal facility, Concerned Calgarians should write to Stephen Harper, whose riding includes all the communities affected by the Tsuu T'ina actions, and Jim Prentice, who's responsible for Indian Affairs. Tell them you're mad as hell and you're not going to take it anymore." read the transcript

10/14/06 - CTV News - Residents experiencing nauseating, burning sensation throughout several communities. Complaints received by residents both on and off reserve. Several hundred elementary and pre-school children are be subjected to the Tsuu T'ina and Carmarcks un-permitted asphalt plant emissions. Over 600 health complaints received regarding emission of the asphalt plant, however Indian and Northern Affairs Canada (INAC) says results so far are normal and acceptable. *Note:* None of the tests completed so far deal with the odour and its affects, stack tests will be done sometime in the next 2 weeks according to INAC.

10/05/06 - Calgary Clean presents the public input into the Permitting, EA and Screening process. Download document here

9/26/06 Calgary Herald - Asphalt Plant owner and City of Calgary Supplier Carmacks Enterprises *Summoned to Court* to Face 8 Charges for Failing to Ensure the Health and Safety of Workers - Two local companies could be facing up to $500,000 in fines after a Calgary worker was seriously hurt by an electric shock two years ago. The province has laid eight charges under the Occupational Health and Safety Act against Carmacks Enterprises Ltd. and D&D Enterprises. The companies are accused of failing to ensure the health and safety of workers, along with other charges. Both companies have been summoned to appear in Calgary Provincial Court on November 10. Carmacks is currently providing to Alberta Transportation the highway maintenance services on the Deerfoot Trail in the City of Calgary

Indian and Northern Affairs Commitment - "The Department will encourage the Nation to operate within the terms and conditions of the permit, but if it is determined that the plant poses a health risk to Nation members or the public, the Department will do everything within its power and authority to have the plant shut down until remedial action is taken." **read the entire letter**

9/21/06 Numerous Health Concerns have been Reported. If you live in one of the SW Calgary neigbourhoods affected by the Tsuu T'ina Council & CARMACKS asphalt plant (Oakridge, Palliser, Braeside, Lakview, Bayview, CedarBrae, Woodbine) and have experienced unusual health concerns over the summer, you are not alone. Common ailments associated with asphalt plant emissions include: asthma, coughing, wheezing or shortness of breath, severe irritation of the skin, headaches, dizziness, and nausea. Please send an e-mail to **info@calgarycleanair.com** with your health issues and contact information.

9/13/06 READ our Responses and Questions (including satellite photos of the site). After careful review of the Tsuu T'ina's Council submitted EA and Screening report, over 8 pages of inconsistencies where uncovered, including: Incorrect distances (***the plant is actually 2.5kms from the City, not 4kms as the Tsuu T'ina state*** in the report); the Tsuu T'ina Assessment is silent on the description of the oil products that are being used at the asphalt plant; ***Contamination of groundwater is a serious matter and has significant adverse environmental effects*** if not properly mitigated; the Assessment states that trucks leaving the asphalt plant need to travel across a one-lane bridge that crosses the Elbow River. ***The Elbow River crossing site is immediately upstream of the Glenmore Reservoir and this is the water body used by the City of Calgary as its major source for drinking water*** for the city's population. Should a malfunction or accident occur due to either spillage or an unfortunate accident with a truck carrying asphalt leaving the bridge and entering the river, this could have ***significant impact on the water quality***; A critical concern lies with the volume and type of oil stored on the plant site and the significant adverse effect that could occur should these tanks rupture or ignite. There is no mention of secondary containment or fire extinguishing equipment or procedures in the Assessment. The resulting ***potential safety hazard*** as well as adverse environmental effects would be experienced by both the residents of the Reserve and the City of Calgary (possibly similar to the experience relating to the ***Hub Oil facility explosion and fire*** in Calgary, AB). **Download Document**

9/11/06 Download the Tsuu T'ina's Environmental Assessment and Screening Report

9/11/06 Calgary Herald article in City Section - An environmental report completed by the plant operators (**Tsuu T'ina** and **Carmacks Enterprises**) were advised to use cleaner fuels and introduce odour neutralizers to mitigate fume problems. ***However, the Tsuu T'ina and Carmacks Enterprises are clearly ignoring those recommendations by continuing to operate the illegal asphalt plant***. Due to jurisdiction issues, the province hasn't been testing on Tsuu T'ina land, but has offered to do so. So far, the proposal hasn't been accepted.

9/10/06 - Asphalt Plant is back in illegal Operation!! - After 8 days of clean air, Calgary and Tsuu T'ina residents were once again woken by the toxic smell of petroleum/diesel early Sunday morning. It appears not having a permit, or following the Provincial standards outlined for the operation of an asphalt plant has not stopped the **Tsuu T'ina Council** and **Carmacks Enterprises** from operating this plant.

9/8/06 Calgary Herald article in City Section - Alberta Environment admits due to jurisdiction issues they have not been testing at the plant site. This is contrary to what Tsuu T'ina Nation 's Peter Mannywounds said on the Rutherford show on August 31st, where he indicated that any *approved* testing agency can come on to the reserve. **Read the article**

9/6/06 QR77 Radio RUTHERFORD SHOW Interview - Department of Indian and Northern Affairs Minister Jim Prentice confirms that if plant doesn't meet rules it will be shut down. Minister also mentions the asphalt plant could be using waste oil instead of properly scrubbed oil. Caller also says "if this plant was built on City property so close to the Elbow River is would be shut down in a minute."

8/31/06 CTV News 5pm - Headaches, fatigue, throat and eye irritation plague City residents from fumes coming from an un-permitted asphalt plant on Tsuu T'ina. Department of Indian and Northern Affairs Canada spokesperson says funding to the reserve could be affected if this plant does not meet the environmental regulations. Tsuu T'ina Nations comment to CTV News: no comment, they are hoping the problem will just go away.

8/31/06 City of Calgary - City Director of Roads says "the City is not purchasing at this time and is awaiting the Environmental report findings."

8/31/06 QR77 Radio RUTHERFORD SHOW Interview - Department of Indian and Northern Affairs clearly states a land use permit and environment impact assessment were required from the band before an asphalt plant can be built, they also confirmed this was not done until after the plant was operating. Carmacks Enterprises has partnered with the Tsuu T'ina Council to run this un-permitted asphalt plant.

8/30/06 CBC News Canada - Paulson told CBC News that while he is concerned about the complaints, there isn't much that can be done right now, adding that the plant is just one of the inconveniences that comes with industrial development.read the entire story. Listen here

8/29/06 CBC News Canada - some people in southwest Calgary say an asphalt plant on the nearby Tsuu T'ina First Nation has been harming their health, complaining that the aboriginal operators aren't operating under the usual environmental regulations. Alberta Environment officials had been taking air samples but stopped after they were told the reserve falls under federal jurisdiction. read the entire story. Listen here

---

PAHs Underfoot: Contaminated Dust from Coal-Tar Sealcoated Pavement is Widespread in the United States

Peter C. Van Metre*, Barbara J. Mahler and Jennifer T. Wilson
U.S. Geological Survey, Austin, Texas
Environ. Sci. Technol., 2009, 43 (1), pp 20–25

We reported in 2005 that runoff from parking lots treated with coal-tar-based sealcoat was a major source of polycyclic aromatic hydrocarbons (PAHs) to streams in Austin, Texas. Here we present new data from nine U.S. cities that show nationwide patterns in concentrations of PAHs associated with sealcoat. Dust was swept from parking lots in six cities in the central and eastern U.S., where coal-tar-based sealcoat dominates use, and three cities in the western U.S., where asphalt-based sealcoat dominates use. For six central and eastern cities, median ΣPAH concentrations in dust from sealcoated and unsealcoated pavement are 2200 and 27 mg/kg, respectively. For three western cities, median ΣPAH concentrations in dust from sealcoated and unsealcoated pavement are similar and very low (2.1 and 0.8 mg/kg, respectively). Lakes in the central and eastern cities where pavement was sampled have bottom sediments with higher PAH concentrations than do those in the western cities relative to degree of urbanization. Bottom-sediment PAH assemblages are similar to those of sealcoated pavement dust regionally, implicating coal-tar-based sealcoat as a PAH source to the central and eastern lakes. Concentrations of benzo[*a*]pyrene in dust from coal-tar sealcoated pavement and adjacent soils greatly exceed generic soil screening levels, suggesting that research on human-health risk is warranted.

http://www.ncbi.nlm.nih.gov/pubmed/19209579

THE UNIVERSITY of NORTH CAROLINA at CHAPEL HILL

NEWS SERVICES

210 Pittsboro Street
Campus Box 6210
Chapel Hill, NC 27599-6210

T 919-962-2091
F 919-962-2279
www.unc.edu/news/
news@unc.edu

News Release

For immediate use

Dec. 16, 2004 -- No. 591

Increased suicide rate is possibly linked to chemicals released from nearby asphalt plants, study suggests

By LESLIE H. LANG
UNC School of Medicine

CHAPEL HILL -- Exposure to low levels of hydrogen sulfide and possibly other airborne chemicals from nearby asphalt plants may have contributed to an increased suicide rate in a North Carolina community, a study suggests for the first time.

In 2003, the suicide rate in two Salisbury, N.C., neighborhoods was found to be 128 per 100,000 individuals a year, roughly 10 times the statewide average, as stated in community reports confirmed by death certificates for that year by the Blue Ridge Environmental Defense League (BREDL).

The study's lead author is Dr. Richard H. Weisler, adjunct professor of psychiatry at the University of North Carolina at Chapel Hill School of Medicine, adjunct assistant professor of psychiatry at Duke University Medical Center and BREDL volunteer.

Other collaborators in this research were Dr. Jonathan R.T. Davidson, professor of psychiatry at Duke University Medical Center; Dr. Lynn Crosby, a toxicologist with BREDL; Lou Zeller, BREDL director; Hope Taylor-Guevera, director of Clean Water for North Carolina; Sheila Singleton, executive director of the N.C. Depression and Bipolar Support Alliance; and Melissa Fiffer and Stacy Tsougas, undergraduates at Duke University's Nicholas School of the Environment and BREDL summer interns.

The neighborhoods comprising two U.S. census tract block groups contained a total of 1,561 residents who were living immediately downwind from a liquid asphalt terminal; an asphalt hot-mix plant, which also contained a former N. C. Department of Transportation solvent-contaminated cleanup site where the DOT had previously dumped solvents used for testing asphalt; and a contaminated former petroleum tank farm.

Between 1994 and 2003, death certificate evaluations for the two Salisbury neighborhoods showed a three-fold statistically significant increase in the suicide rate, the study found. Four deaths by suicide in adults were reported from the 687 residents in the census tract block group 1. Two deaths by suicide in adults were reported among the 874 residents of census tract block group 2. Only two deaths by suicide would be expected for this population over a 10-year period, but six suicides were observed.

"For example, here in the block group 1 neighborhood in the mid-90s, we found one death by suicide for about every 230 people during the worst 12-month period, versus an average of one death by suicide for every 8,621 people in the rest of North Carolina," Weisler said. "When we saw this data it gave us pause."

Weisler said of hydrogen sulfide, "The odor was frequently apparent when I lived there as a child and later when I visited my mother, who lived in the neighborhood from 1962 until her death in 2001."

That year (2001), the N.C. Department of Environment and Natural Resources (NCDENR) estimated the average maximum hydrogen sulfide level in a large part of the affected area at 215 parts per billion (pbb), while some sections of the neighborhoods were reported as low as 30 ppb. Moreover, based on their own air modeling study, the NCDENR estimated that historical releases of hydrogen sulfide reached average maximum levels of 860 ppb in a few residences very near the asphalt facilities.

By comparison, the World Health Organization has a 10-minute exposure standard of five ppb. The California one-hour standard is 30 ppb. The newly revised, but not yet implemented, North Carolina 24-hour hydrogen sulfide standard is 86.2 ppb.

These exposures accompanied 574 formal complaints to the City of Salisbury from March 11, 1999, to Oct. 15, 2004, for noxious odors and associated respiratory problems, which are still occurring – though at a reduced rate – said Weisler.

In addition to suggestions of an increased suicide rate, the incidence rate of primary brain cancers in these neighborhoods from 1995 to 2000 showed an increase about 6.4 times greater than expected for the population, possibly due to benzene and other solvent exposures, Weisler said.

Several studies have shown increased rates of lung and brain cancer among workers with long-term exposure to asphalt emissions, the researchers said.

Weisler and his study team made a hypothetical link between hydrogen sulfide and suicides due to biological plausibility. They noted that hydrogen sulfide affects brain neurochemistry as a direct gaseous neuromodulator that potentially affects mood states and the psychological stress response. In animal studies, it has been shown to alter the neurotransmitters serotonin, norepinephrine, dopamine, aspartate and glutamate levels.

Hydrogen sulfide also affects the hypothalamic pituitary adrenal axis and corticotropin releasing factor in animal studies, the report said.

"This is the part of the brain involved in the stress response, and we think it’s also involved in psychological resiliency, how people deal with stressors," Weisler said. "It’s frequently associated with mood disorders, and there are suggestions that resiliency is impaired when people are suicidal."

The study team reported that additional neurotoxic compounds such as benzene, chlorinated solvents and carbon disulfide, among others, were released in unknown quantities by the asphalt terminal and hot-mix asphalt plant. Carbon disulfide, also a neurotoxin, has been linked to personality changes, mood disorders and suicides in occupational settings, the researchers said.

In addition, "Some research suggests that highway workers exposed to asphalt-solvent fumes show an increase of suicide rates and brain cancers."

A full characterization of the types of chemicals and the levels of releases at the liquid asphalt terminal is needed, said Weisler.

Also needed, he added, is the retrospective ground water contamination modeling study called for in 2002 by the N. C. Department of Health and Human

Services to more completely understand the possible causes of health problems in the affected neighborhoods.

"I do not know if ground water modeling would help us understand the suicides, but since there were exposures it would be quite useful to have that modeling information. The same modeling would certainly help with interpreting the cancer data as people with brain, lung, blood, pancreatic, breast, and colon cancers had been or may have been using solvent contaminated well water for extended periods," Weisler said.

Davidson said the most important point for people to remember is that effective treatments exist for suicidal depression.

"Given that suicide can be a tragic consequence to depression, people who are experiencing persistent symptoms of depression should contact their health-care provider for a professional evaluation," he said. "The findings of this study may suggest another potential risk factor for suicide, but this needs to be confirmed in future studies."

The most common symptoms of depression include loss of interest in activities once considered pleasurable, social withdrawal, changes in appetite, low mood, inability to function effectively in work or family situations and, often, a feeling of hopelessness and despair. "It is the hopelessness that can lead to suicidal thoughts or actions," Davidson added.

A person with a family history of suicide attempts or substance abuse may be at greater risk than others, he said, adding that the study findings may eventually suggest yet another risk factor for suicide – making further study all the more important.

Weisler and Davidson both emphasized the need to educate residents of the affected areas about mood and anxiety disorders as well as substance use disorders and their treatments.

Formal health studies of the two neighborhoods and other potential sites with chemical exposures are being planned at UNC's School of Public Health.

The health status of residents who died by suicide will be investigated further in a study involving Dr. Steven B. Wing, associate professor of epidemiology, and others at UNC's School of Public Health.

Significant steps have already been taken, said Weisler, but reducing potentially toxic exposures from the industrial plants and safe cleanup of the solvent and petroleum contaminated area sites will be crucial.

"We do not know with scientific certainty that the area suicides are linked to hazardous chemical exposures, but we know enough to recommend that it is not worth taking any more chances on the potential association."

Weisler presented the findings Nov. 19 to the 17th Annual U.S. Psychiatric and Mental Health Congress in San Diego.

- 30 -

Note: For media inquiries about this story and to speak with Weisler, contact Crystal Hinson Miller at (919) 966-9115.

UNC School of Medicine contact: Les Lang, (919) 843-9687 or llang@med.unc.edu

Duke University Medical Center contact: Tracey Koepke, (919) 660-1301 or koepk002@mc.duke.edu

Asphalt Pollution Probe Extends

By
7/6/2004
URL: http://www.recyclingtoday.com/news/news.asp?ID=6040

A U.S. Environmental Protection Agency pollution investigation of asphalt companies, which mix crude oil and gravel to make road-building materials, has expanded from Ohio to Indiana and other states.

The agency has ordered pollution tests and company records from two asphalt plants in Illinois and one each in Indiana, Michigan, Minnesota and in Fairborn, Ohio, near Dayton, The *Columbus Dispatch* reported. Two Columbus, Ohio, asphalt companies got similar orders earlier in the year.

While the agency hasn't discussed the investigation publicly, the EPA said earlier that demands for internal records are the first official steps taken when it suspects Clean Air Act violations.

Ohio has more than 300 asphalt plants. There are more than 1,100 asphalt companies nationwide, many with several operating plants.

Plant owners and the industry's trade association said they didn't know the reason for the EPA directives and questioned the cost.

"I'm one of 200 or so plants in Illinois," said Stephen Kennedy, vice president of Rock Road Cos., which runs a plant in Rockford. "I'm wondering why I'm doing this and my competitors are not."

Bill Omohundro, a U.S. EPA spokesman, would not comment about the expanded investigation, the newspaper said. Omohundro could not be reached for comment July 5th.

The letters sought new air-emission tests for soot, carbon monoxide, nitrogen oxides, sulfur dioxide and volatile organic compounds. Companies must provide information about modifiers put into liquid asphalt and about the fuel burned to keep asphalt from hardening.

Alvin Evans, chief operating officer for J.H. Rudolph & Company Inc. in Evansville, Ind., said he was asked to test for things the state doesn't require. He said Indiana requires estimates of volatile organic chemicals, nitrogen oxides and sulfur emissions.

"We've asked for an extension of the time period we've been given," Evans said.

The other companies facing directives from the EPA were Central Specialties Inc. of Alexandria, Minn., Ajax Materials Corp. of Warren, Mich., Barrett Paving Materials Inc. of Fairborn and Chester Bross Construction of Loraine, Ill., the newspaper said. None could be reached for comment July 5th.

Gary Fore, vice president of environment and safety for the National Asphalt Pavement Association, called the letters unprecedented and said his group would discuss them with the EPA. Fore said the industry has worked for 10 years with the EPA on studies that show asphalt plants are not major sources of pollution.

Theresa Mills, director of the Buckeye Environmental Network, and Simona Vaclavikova, a program director for Ohio Citizen Action, say the tests might not cover all the hazardous substances asphalt plants emit into the air.

Both said neighbors began to complain about asphalt plants in Ohio around 2000. Mills suspects complaints might be linked to the used oil some plants burn as fuel or new modifiers put in the asphalt.

The industry-supported Asphalt Institute said asphalt is the environmental choice of highway builders because, in part,it can be colored to match the surrounding environment and, without seams like concrete section amid heavy truck traffic. Associated Press

BLUE RIDGE ENVIRONMENTAL DEFENSE LEAGUE
Central office: PO Box 88 Glendale Springs, NC 28629 (910) 982-2691 fax: (910) 982-2954

FOR IMMEDIATE RELEASE
JULY 24, 1997

CONTACTS:
Dr. Richard Maas (704) 251-6441
Lou and Janet Zeller (910) 982-2691
Dale and Nancy Thompson (704) 733-2478
Donna Autrey (704) 733-5653
Ron Chivers (704) 262-0070

CARCINOGENS DISCOVERED NEAR MAYMEAD PLANT

Today at a press conference in Pineola in Avery County, citizens' organizations released laboratory test results showing both drinking water well contamination and surface water pollution. The Blue Ridge Environmental Defense League, Pineola Concerned Citizens, and Citizens Against Pollution pointed to the large Maymead asphalt plant as a likely source of the contamination.

Test results of water samples taken from the well of Dale and Nancy Thompson and from area streams show high levels of formaldehyde. The Thompsons live next to the 325 ton per hour Maymead plant. Asphalt plants are a major source of formaldehyde, which is highly soluble in water. The laboratory test results were reviewed by Dr. Richard Maas, a leading water quality expert and head of the Environmental Quality Institute at UNC-Asheville. Dr. Maas concluded, "The analysis shows a severe level of contamination by formaldehyde which is a known carcinogen."

The Thompsons' well water test results revealed .22 mg/L of formaldehyde. Water samples gathered from a pond and streams which drain the asphalt plant area were also highly contaminated with .23 mg/L of formaldehyde.

-more-

The citizens' groups called for an immediate shut down of the Maymead plant, for the installation of groundwater test wells around the facility, and for testing of all drinking water wells within one-half mile of the plant. Lou Zeller, community organizer for BREDL, said, "The state of North Carolina has permitted this plant and the state must take action now to assess the damage to the people of Pineola." Zeller continued, "State air and water permits must mean that the environment and public health are protected."

In the next two weeks, the groups expect the North Carolina Division of Air Quality to release its decision about a proposed Maymead asphalt plant east of Boone. According to the draft permit, that 150 ton per hour plant could release over 3,000 pounds of formaldehyde annually.

Ron Chivers of the Watauga-based Citizens Against Pollution, called on the state to deny the new Maymead permit. Chivers said, "We implore you to remember your own mission goals, to preserve first and foremost the health and welfare of the people you serve." The Avery County Board of Commissioners has requested a full toxic inventory of pollutants coming from the Pineola plant. The NC Division of Air Quality has not yet issued that report. State officials are also conducting a health risk assessment for the Pineola community.

-end-

Childhood Brain Cancers Near Asphalt Industry in Salisbury, North Carolina

Adapted from a presentation by
Dr. Richard Weisler
Blue Ridge Environmental Defense League
January 2003

Health Problems

- Increased Cancer Rate
    - Over the last five years, very rare childhood brain cancers have been occurring at rates 11 times that expected in three census tracts near asphalt plants and untreated groundwater contaminated sites in the Milford Hills community of Salisbury.
    - The four cases were identified by a lone physician and may underestimate the actual cancer rate.
    - The ages of the children diagnosed with brain cancers were 3, 9, 11, and 16.
    - The rate of morbidity may be increasing: 14 of the 19 brain cancers in this survey were diagnosed between 1995 and 2002. Just 5 of the cancers were diagnosed in the previous seven years, between 1988 and 1995.
- Other Health Problems
    - Rates of cancers of the lung, pancreas, and blood system appear to be increased.
    - Because of the types of contamination, other health problems including heart disease, stroke, asthma, diabetes, and thyroid disease are of great concern.
- Citizens' Plan of Action
    - The State of North Carolina failed to protect the health of residents of Milford Hills. Citizens do not believe that the NC Department of Environment and Natural Resources will honestly assess the true risks from the chemical poisons that have contaminated this community.
    - The asphalt industry and the NC Department of Transportation have failed to clean up this environmental disaster and make the area safe for residents.
    - Ultimately, public health officials will need the help of residents past and present to do a proper health study.
    - Our plan is to assist in the identification of residents and workers who have been exposed.

The Polluters

- Chevron Asphalt
    - Poisoned the air, water, and soil for about 45 years with benzene, solvents, asbestos, lead, cadmium, dangerous fine particles, hydrogen sulfide, and other cancer-causing chemicals by the manufacture of "cutback asphalt."
    - Illegally delayed reporting dangerous contamination nine years after finding it, never reported friable asbestos, and never notified workers or neighbors who were exposed.
    - Never controlled or treated toxic plant emissions.

- Exxon Mobile
    - Poisoned the underground water supply and soil with acres of oil products for many decades.
    - Contaminated the air with dangerous chemicals from old storage tanks, spills, and leaks.
    - Emitted benzene at levels 1,233 times about maximum acceptable levels next to a Little League park.
    - Never informed residents or elected officials about the exposure and associated health risks.

- NC Department of Transportation
    - Contaminated shallow and deep groundwater supply for decades. For example, trichloroethylene levels in well water reached 7,600 ppb (the acceptable maximum is 2.8 ppb).
    - Failed to properly limit or advise monitoring for dangerous solvent exposure of workers or residents.
    - Never told residents or city officials of the risks.
    - Delayed cleanup, which remains inadequate.

- Associated Asphalt
    - Released immediately life threatening hydrogen sulfide into the air measured at 2,400 ppm on-site, triggering asthmas at great distances from the plant.
    - Emitted sickening odors that permeated homes and drove people indoors.
    - Poisoned the air with dozens of toxic chemicals.
    - Unlawfully removed asbestos-insulated storage tanks and pipes in 1998 without permits.
    - Scrapped carbon air filters and replaced with ineffective Ecosorb system to save money.
    - Grossly under-reported toxic releases.

CHEJ Asphalt Fact Pack 113

Article

pubs.acs.org/est

Cancer Risk from Incidental Ingestion Exposures to PAHs Associated with Coal-Tar-Sealed Pavement

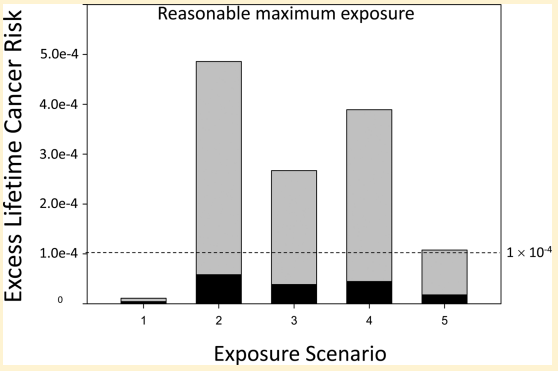
E. Spencer Williams,*[,†] Barbara J. Mahler,[‡] and Peter C. Van Metre[‡]

[†]Baylor University, Center for Reservoir and Aquatic Systems Research, One Bear Place #97178, Waco, Texas 76798-7178, United States

[‡]U.S. Geological Survey, 1505 Ferguson Lane, Austin, Texas 78754, United States

Supporting Information

ABSTRACT: Recent (2009−10) studies documented significantly higher concentrations of polycyclic aromatic hydrocarbons (PAHs) in settled house dust in living spaces and soil adjacent to parking lots sealed with coal-tar-based products. To date, no studies have examined the potential human health effects of PAHs from these products in dust and soil. Here we present the results of an analysis of potential cancer risk associated with incidental ingestion exposures to PAHs in settings near coal-tar-sealed pavement. Exposures to benzo[*a*]pyrene equivalents were characterized across five scenarios. The central tendency estimate of excess cancer risk resulting from lifetime exposures to soil and dust from nondietary ingestion in these settings exceeded 1×10^{-4}, as determined using deterministic and probabilistic methods. Soil was the primary driver of risk, but according to probabilistic calculations, reasonable maximum exposure to affected house dust in the first 6 years of life was sufficient to generate an estimated excess lifetime cancer risk of 6×10^{-5}. Our results indicate that the presence of coal-tar-based pavement sealants is associated with significant increases in estimated excess lifetime cancer risk for nearby residents. Much of this calculated excess risk arises from exposures to PAHs in early childhood (i.e., 0−6 years of age).



INTRODUCTION

The presence of coal-tar-based sealants on asphalt parking lots is associated with elevated concentrations of polycyclic aromatic hydrocarbons (PAHs) in the surrounding environment.[1−6] Sealcoat is a black, shiny substance sprayed or painted on the asphalt pavement of parking lots, driveways, and playgrounds to improve appearance and protect the underlying asphalt. An estimated 85 million gallons (320 million liters) of coal-tar-based sealant are applied to pavement each year,[7] primarily east of the Continental Divide in the U.S. and parts of Canada.[4,8] Coal-tar-based pavement sealants are 15−35% coal-tar pitch, which has been classified as a human carcinogen (IARC Group 1).[9] PAHs are the major constituents of coal-tar pitch,[10] and commercially available coal-tar-based sealants contain on the order of 50 000−100 000 mg/kg PAHs [sum of the 16 U.S. Environmental Protection Agency (USEPA) Priority Pollutant PAHs (ΣPAH_{16})].[7,11] Over time, the dried sealant is abraded from pavement surfaces, and the resulting mobile particles can be transported into nearby environmental compartments.[7,12]

Coal-tar-based pavement sealants are the predominant source of PAHs in the sediment of many urban and suburban lakes, especially areas where population is rapidly growing.[3,13] Coal-tar-based sealants are associated with deleterious effects on local ecosystems, including decreases in species richness and abundance among benthic invertebrates,[14,15] slower growth and impaired swimming behaviors in salamanders,[16] and impaired growth and development of frogs.[17] PAHs from coal-tar-based pavement sealants also contaminate environmental media that are relevant to human exposures. In a study of 23 apartments in Austin, Texas, the median concentration of ΣPAH_{16} in settled house dust (SHD) in residences adjacent to coal-tar-sealed asphalt (CSA) parking lots was 31 times higher than in SHD in apartments adjacent to unsealed asphalt (UA) lots.[18] The presence or absence of coal-tar-based sealants on the adjacent lot explained 48% of the variance in PAH concentrations measured in SHD.[18] Elevated PAH concentrations also have been reported for soil adjacent to CSA lots relative to soil adjacent to UA lots.[2,4] Hereinafter, soil and SHD near CSA or UA parking lots are described as "CSA-affected" or "UA-affected", respectively.

Exposure to PAHs is linked to increased risk for multiple cancer types, including lung, skin, bladder, respiratory, and urinary tract.[19] These studies have mostly examined inhalation exposure at sintering plants, foundries, and similar industrial settings. The carcinogenic properties of tobacco smoke are attributed, in part, to the presence of PAHs.[20] Aside from

Received: August 27, 2012
Revised: November 20, 2012
Accepted: November 23, 2012
Published: November 23, 2012

smoking, nonoccupational exposures to PAHs are believed to occur primarily through dietary ingestion.[21] In the interest of understanding aggregate doses, several studies have characterized the presence of PAHs in a wide array of foodstuffs in different countries, including the U.S., as reviewed in Ramesh et al. (2004).[21] Seven PAHs—benz[*a*]anthracene, benzo[*k*]-fluoranthene, benzo[*b*]fluoranthene, benzo[*a*]pyrene (BaP), chrysene, dibenz[*a*,*h*]anthracene (diBahA), and indeno[123-*cd*]pyrene—have been classified by the USEPA as probable human carcinogens (B2 PAHs).

Nondietary ingestion (incidental ingestion of soil and SHD) is a pathway for exposure to numerous chemicals, including lead, pesticides, polychlorinated dioxins and furans, polybrominated diphenyl ethers, and PAHs, especially in children.[22,23] Many sources and activities are hypothesized to contribute PAHs to SHD, including cooking, smoking, vehicle exhaust, and indoor heating.[24,25] These exposures have been characterized as minor relative to those associated with dietary ingestion;[26,27] however, recent research indicates that in CSA-affected residences, nondietary ingestion of PAHs likely exceeds dietary ingestion.[28]

To date (November 2012), the authors are not aware of any published studies that have assessed the potential risks to human health associated with the elevated concentrations of PAHs measured in CSA-affected environments. The objective of the current study was to examine and compare exposure to and risk arising from ingestion of B2 PAHs in SHD and soil in settings adjacent to CSA and UA parking lots. Standard deterministic risk-assessment techniques were used to estimate B2 PAH doses and associated excess lifetime cancer risk (ELCR) for five exposure scenarios spanning childhood, adolescence, and adulthood, and probabilistic risk calculations were conducted for three of these scenarios.[29]

METHODS

This risk assessment focuses on the B2 PAHs. Each of these compounds has been assigned a potency factor (RPF) relative to the potency of BaP, ranging from 0.001 for chrysene to 1 for diBahA and BaP.[30] Ingestion dose estimates are presented for BaP equivalents (BaPEQ), computed as the sum of the product of the concentration of each B2 PAH and its RPF. Bioavailability is assumed to be 100%.

As noted in ref 18, analytical difficulties with diBahA resulted in nondetections in all but one SHD sample collected for that study. Thus, diBahA is not included here in any computations of BaPEQ in SHD or soil. Estimates of dose including diBahA at the limit of detection divided by two (not shown) indicate that it likely accounted for no more than 5−7% of the total dose of BaPEQ. By comparison, BaP accounted for 72−73% of BaPEQ in SHD samples, and 76−77% in soil samples.

Concentrations of BaPEQ in Dust and Soil. Data on PAHs in SHD used for this analysis were published previously.[18] In that study, SHD and parking lot dust were sampled for 23 ground-floor apartments in Austin, Texas. The parking lot surface adjacent to the apartment complexes was CSA (n = 11), UA (n = 7), asphalt-based sealant over asphalt pavement (n = 3), or unsealed concrete (n = 2). For this analysis, doses and risk associated with residences adjacent to UA parking lots were considered relative to those adjacent to CSA parking lots. BaP concentrations in CSA-affected SHD were high (median and maximum of 4.5 and 24.2 μg/g, respectively) relative to those reported in most parts of the U.S. where coal-tar-based sealcoat is not used (e.g., California: median and maximum of 0.04 and 1.0 μg/g, respectively; Arizona: median and maximum of 0.06 and 0.07 μg/g, respectively[25]). We computed BaPEQ for data presented in;[18] concentrations of BaPEQ in SHD in apartments adjacent to CSA parking lots (8.1 μg/g, geometric mean) were significantly higher than those in apartments adjacent to UA lots (0.61 μg/g, geometric mean) (p = 0.002, Mann−Whitney−Wilcoxon). Risk-assessment guidance recommends the use of the 95% upper confidence limit of the arithmetic mean,[29] but high standard deviations in the data sets, normality testing in log-transformed data, and an emphasis on conservatism in dose and risk estimates dictated the decision to use geometric means of these data to represent the BaPEQ exposure concentration in deterministic calculations.

Dust loading was computed for each location sampled in ref. 18 (Supporting Information Table S1). Loading of BaPEQ in the dust is significantly higher in residences adjacent to CSA pavement (medians of 15.7 μg/m^2 CSA vs 0.63 μg/m^2 UA; p = 0.01, Mann−Whitney−Wilcoxon). Total dust loading is higher in the CSA group relative to the UA group (medians of 346 and 72.3 μg/cm^2, respectively), but the difference was not significant (p = 0.365, Mann−Whitney−Wilcoxon). However, one data point in the UA SHD data set is an outlier (884 μg/cm^2) more than 4 times larger than all other data points and after removal of this data point, CSA settings have significantly higher dust loadings than UA settings (p = 0.043, Student's t test; data passed normality testing after elimination of the outlier). One issue that could not be resolved in this analysis is the relative importance of flooring type, because some samples were collected in combinations of bare and carpeted flooring.

Data for PAHs in CSA- and UA-affected soils are available for samples from New Hampshire (UA n = 1, CSA n = 5)[2] and suburban Chicago (UA n = 2, CSA n = 2).[4] Concentrations of BaP in UA-affected soils ranged from below detection limit to 0.7 μg/g. These are consistent with background concentrations reported for U.S. soils of up to 1.3 μg/g,[19] and somewhat higher than those reported for soil samples collected in remote areas around the world (range <0.0001 to 0.386 μg/g).[31] Concentrations of BaP in CSA-affected soils were substantially higher, ranging from 2.98 to 29.2 μg/g.[2,4] Concentrations of BaP in dust on pavement with coal-tar-based sealant are typically in the 100s of μg/g.[2,18] Concentrations of BaP in the 100s of μg/g in soil are typical of those in soils at manufactured gas sites and wood preservative sites,[32,33] some of which have been classified as Superfund sites (http://www.epa.gov/region5/cleanup/mgp.htm). Geometric mean BaPEQ soil concentrations for CSA-affected settings were 12.4 μg BaPEQ/g soil, and for UA-affected settings were 0.19 μg BaPEQ/g soil.

Deterministic and Probabilistic Estimates of Dose and Excess Lifetime Cancer Risk. Doses of BaPEQ were estimated using the standard equation (eq 1) included in the Risk Assessment Guidance for Superfund, Part A.[29] Exposure assumptions for both deterministic and probabilistic risk calculations are given in Supporting Information Table S2.

$$\text{dose} = \frac{\text{Cm} \times \text{CF} \times \text{IR} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}} \quad (1)$$

where Cm is the concentration of BaPEQ in the dust, soil, or both, CF is the conversion factor, IR is ingestion rate, EF is exposure frequency, ED is exposure duration, BW is body weight, and AT is averaging time.

Table 1. Excess Lifetime Cancer Risk (ELCR) Estimates for Central Tendency (CTE) and Reasonable Maximum (RME) Exposures in Five Scenarios for Carcinogenic Polycyclic Aromatic Hydrocarbons by Ingestion of Settled House Dust, Soil, And Both Media[a]

	age of exposure (years of age)		settled house dust only		soil only		dust and soil	
scenario	UA	CSA	CTE	RME	CTE	RME	CTE	RME
1	0−70	N/A	1.5×10^{-6}	4.4×10^{-6}	1.4×10^{-6}	6.7×10^{-6}	2.9×10^{-6}	1.1×10^{-5}
2	N/A	0−70	2.0×10^{-5}	5.8×10^{-5}	8.9×10^{-5}	4.3×10^{-4}	1.1×10^{-4}	4.9×10^{-4}
3	6−70	0−<6	1.1×10^{-5}	3.8×10^{-5}	2.9×10^{-5}	2.3×10^{-4}	4.0×10^{-5}	2.7×10^{-4}
4	18−70	0−<18	1.4×10^{-5}	4.4×10^{-5}	4.7×10^{-5}	3.4×10^{-4}	6.1×10^{-5}	3.9×10^{-4}
5	0−<18	18−70	8.2×10^{-6}	1.8×10^{-5}	4.3×10^{-5}	9.0×10^{-5}	5.1×10^{-5}	1.1×10^{-4}

[a]UA, unsealed asphalt pavement; CSA, coal-tar-sealed asphalt pavement; N/A, not applicable.

The geometric mean BaPEQ for SHD and soil were used as point estimates for deterministic dose and risk calculations. Lognormal distributions based on data from refs 2,4,18 were developed for probabilistic calculations [UA soil: mean 0.423 μg/g (standard deviation (sd) = 0.523), CSA soil: mean 15.8 μg/g (sd =11.9); UA SHD: mean 1.10 μg/g (sd =1.08), CSA SHD: mean 11.4 μg/g (sd = 9.41)]. Lognormal distributions and corresponding geometric means were chosen to reflect the frequent observation of distributions of this type in environmental contaminant concentrations.

For deterministic calculations of SHD ingestion, we used recently published SHD intake rates for children determined using the Stochastic Human Exposure and Dose Simulation (SHEDS) model for multimedia pollutants.[34] The SHEDS model addresses two pathways of exposure to dust: direct ingestion of SHD from hand-to-mouth contact, and indirect ingestion resulting from mouth contact with inanimate objects such as toys (especially relevant for preschool children). The model takes into account the importance of SHD loading, a strong predictor of blood lead levels related to dust-mediated exposure. The model relies on the Consolidated Human Activity Database, which has activity diaries for over 22 000 individuals.[35] We employed the mean SHD IR estimate from ref. 34 of 27 mg/day (rounded to two significant figures to account for the inherent uncertainty of the model) for children 3−<6 years of age as a central tendency estimate (CTE) of exposure for children 0−6 years of age, and the 95th percentile values from [34] as a reasonable maximum estimate (RME) of exposure. For individuals older than 6 years of age, who are expected to be away from the home for much of the day, we used one-half of the early childhood CTE dust IR (13 mg/day), and 27 mg/day as the RME dust IR. Few data are available for SHD IRs for adults, but previous risk assessments have employed adult SHD IRs of 20 and 50 mg/day,[22,36] higher than the IRs used in this analysis. The distribution of child IRs for SHD was adapted from ref. 34 (mean = 27 mg/day, sd = 40, log-normal) for probabilistic dose and risk calculations, and a similarly shaped distribution was postulated for SHD IR for 6−70 years of age (mean = 13.3 mg/day, sd = 19.6, log-normal).[34]

For deterministic calculations of soil ingestion, default IRs from the Exposure Factors Handbooks and the Child Specific Exposure Factors Handbook,[37,38] with some minor modifications, were used. For persons of all ages, 50 mg/day was used for the CTE soil IR, and the RME IRs used were 400 mg/day from 1−13 years of age and 100 mg/day from 13−70 years of age.

For a distribution for soil IRs for children 0−<13 years of age, we used data generated by the SHEDS model that indicated an arithmetic mean of 60.6 mg/day, sd of 80.5 mg/day.[39] These values are similar to those from a recent review of all published tracer studies on soil ingestion by children, in which the arithmetic mean was estimated at 63 mg/day, with a median of 27 mg/day and a 95th percentile of 195 mg/day.[39] The SHEDS model result was used as the basis for probabilistic calculations of dose and risk in children. For children and adults 13−70 years of age, the arithmetic mean of all available soil ingestion rates from tracer studies was 46 mg/day (rounded to 50 mg/day in deterministic calculations).[39] A distribution similar to that for soil ingestion in children was postulated, and an appropriate standard deviation was calculated for use in a Monte Carlo analysis (http://www.epa.gov/oswer/riskassessment/rags3adt/index.htm). Adult IRs have been updated in the most recent (2011) version of the Exposure Factors Handbook to indicate a central tendency for adults of 20 mg/day for the soil IR and 30 mg/day for the dust IR.[40] These values rely on relative proportions of soil and dust ingestion for children, and thus we have chosen to retain the value of 50 mg/day (i.e., 46 mg/day, rounded to one significant digit) from the previous Handbook, which also is the value indicated in the current Handbook for adults 18−21 years of age.[40] Recalculation of risk estimates using soil and dust ingestion rates in the 2011 version of the Handbook do not change the overall conclusions of this assessment.

Body weight distributions were obtained from a recent (2007) analysis of the National Health and Nutrition Examination Survey (NHANES) data set.[41] Exposure frequency was set at 365 days/year in both deterministic and probabilistic calculations.

Exposure Scenarios. Five scenarios that describe exposures to combinations of UA- and CSA-affected SHD and soil were used (Table 1): exposures in UA-adjacent spaces (UA exposures) during a 70-year lifetime (scenario 1); exposure in CSA-adjacent spaces (CSA exposures) during a 70-year lifetime (scenario 2); CSA exposures during 0−<6 years of age followed by UA exposures during 6−70 years of age (scenario 3); CSA exposures during childhood (0−<18 years of age) followed by UA exposures during adulthood (18−70 years of age, scenario 4); and UA exposures during 0−<18 years of age followed by CSA exposures during adulthood (18−70 years of age, scenario 5). Incremental ELCR values for timeframes of 1 year from 0 to 18 years of age and of 1 year from 18 to 70 years of age were summed to arrive at a lifetime ELCR value for each scenario. Exposure to UA-affected environments during a 70-year lifetime (Scenario 1) was assumed to represent urban background for the purpose of evaluating the potential differences in risks associated with exposure to CSA-affected media. Scenario 1 considers lifetime exposures to SHD and soil not affected by PAHs associated with CSA pavement, and thus represents a reasonable measure of urban background.

dx.doi.org/10.1021/es303371t | Environ. Sci. Technol. 2013, 47, 1101−1109

For the probabilistic calculations, Monte Carlo simulations were performed for 10 000 trials. These simulations were conducted only for scenarios covering lifetime exposures to UA environments (scenario 1), lifetime exposures to CSA environments (scenario 2), and exposures to CSA-affected media in the first 6 years of life (scenario 3).

Estimation of Excess Lifetime Cancer Risk. The ELCR from exposure to a chemical is described in terms of the probability that an exposed individual will develop cancer by age 70 because of that exposure.[42] Estimates of BaPEQ dose were multiplied by the oral cancer slope factor for BaP of 7.3 per mg/kg/day.[43] For single-year calculations of risk (0−18 years of age), the slope factor was divided by 70, and for calculation of risk for adulthood (18−70 years of age), it was divided by (70/52); risk estimates were generated by summing yearly risks from 0−18 years of age and during adulthood (i.e., 18−70 years of age). In general, the USEPA considers excess cancer risks less than 1×10^{-6} so small as to be negligible (i.e., de minimus), and those greater than 1×10^{-4} to be sufficiently large that some sort of remediation is desirable.[42] Excess cancer risks between 1×10^{-6} and 1×10^{-4} generally are considered to be acceptable, although this is evaluated on a case-by-case basis and the USEPA may determine that risks lower than 1×10^{-4} are not sufficiently protective and warrant remedial action.[42]

RESULTS

Deterministic Dose Estimates. Estimated lifetime CTE BaPEQ dose from ingestion of SHD and soil in CSA-affected settings was 38 times greater than that estimated for UA-affected settings (Supporting Information Table S3). Maximum doses occur at young ages (Figure 1), when body weights are lower and ingestion rates are higher than later in life (Supporting Information Table S3). About 50% of the total estimated RME lifetime dose occurs during 0−<6 years of age, and about 80% occurs during 0−<18 years of age. Doses of BaPEQ for ingestion of CSA-affected soil were greater than those for CSA-affected SHD (Figure 1), comprising about 80% of the aggregate (soil + SHD) lifetime dose. The difference arises because BaPEQ concentrations and IRs are higher for CSA-affected soil than for CSA-affected SHD (Supporting Information Table S2). The CTE lifetime dose from CSA-affected SHD alone, however, is not insubstantial, exceeding the lifetime aggregate dose in UA-affected settings by a factor of 7. The RME lifetime aggregate dose estimate for CSA-affected settings is about 4.5 times higher than the CTE lifetime aggregate dose estimate.

Risk Estimates. Deterministic estimates of ELCR were calculated for the five exposure scenarios (Table 1, Figure 2). Under scenario 1 conditions (urban background), soil is estimated to contribute about one-half (48%) of the aggregate (SHD + soil) CTE estimate of ELCR of 2.9×10^{-6} and the majority (61%) of the RME estimate of 1.1×10^{-5}.

Estimated aggregate CTE ELCR for lifetime exposure to CSA-affected settings (1.1×10^{-4}; scenario 2) was 38 times higher than urban background (scenario 1) (Figure 2). About 36% of the increased ELCR attributable to ingestion of CSA-affected SHD and soil occurs during exposures during the first 6 years of life (scenario 3), when IRs are highest and body weights are lowest, and 56% occurs during the first 18 years of life (scenario 4). The RME ELCRs were from 2.2 to 6.8 times higher than CTE ELCRs across all CSA-affected scenarios (2−5), and the difference was greatest for exposure to CSA-affected environments from 0−6 years of age (scenario 3) (Figure 2).

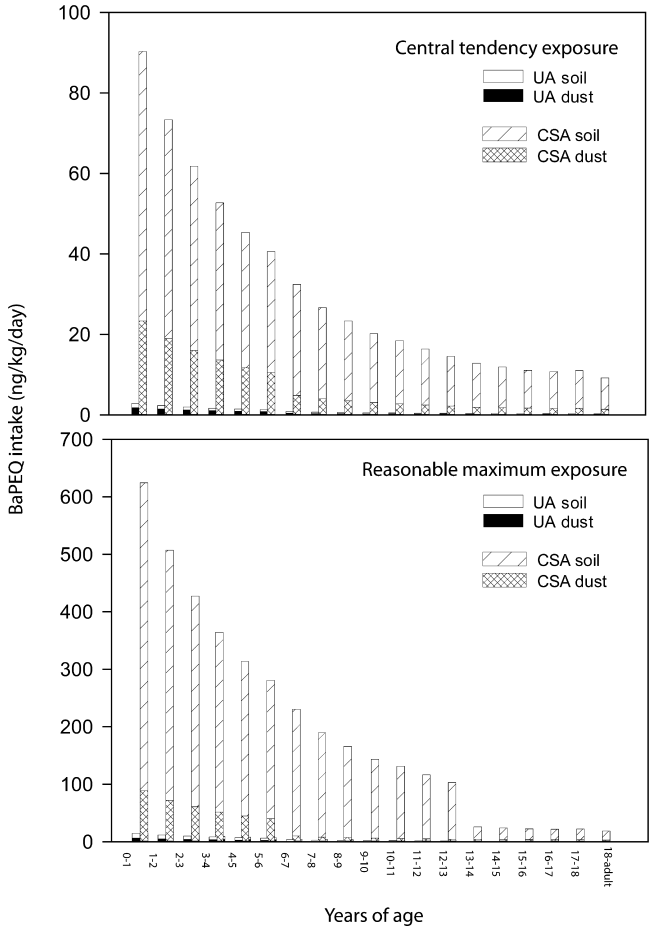


Figure 1. Aggregate doses of benzo[*a*]pyrene equivalents (BaPEQ) (ng/kg/day) from settled house dust and soil in settings adjacent to unsealed asphalt and coal tar-sealed asphalt pavement (UA and CSA, respectively) by year for central tendency and reasonable maximum exposures. Adult years (i.e., 18−70 years of age) are noted as "18-adult.".

In this analysis, ingestion of CSA-affected soil is a more important driver of risk than ingestion of CSA-affected SHD. Ingestion of soil made up about one-half (48%) of ELCR in urban background settings, but made up 72 to 84% of ELCR in CSA-affected settings (Figure 2). Over a lifetime of exposure (scenario 2, CTE), ELCR is estimated to be about 64 times greater for persons who ingest CSA-affected soil relative to their counterparts who are exposed to background concentrations; the comparable difference for CSA-affected and unaffected SHD is a factor of 13. The CTE ELCR for soil alone approaches 1×10^{-4}, and the RME ELCR was estimated at 4.3×10^{-4} (Table 1). Much of the lifetime risk occurs during early childhood (0−<6 years of age, scenario 3) and all childhood (0−<18 years of age, scenario 4) exposures (33 and 53%, respectively). All RME scenarios in CSA-affected environments involving childhood exposure (scenarios 2−4) had ELCR values associated with ingestion of soil exceeding 1×10^{-4}.

Although SHD-mediated exposure to BaPEQ in CSA settings results in less risk compared to soil-mediated exposure, it nonetheless represents a substantial increase in risk over urban background exposure. This is a particularly important pathway of exposure for children. Even more of the lifetime risk

dx.doi.org/10.1021/es303371t | *Environ. Sci. Technol.* 2013, 47, 1101−1109

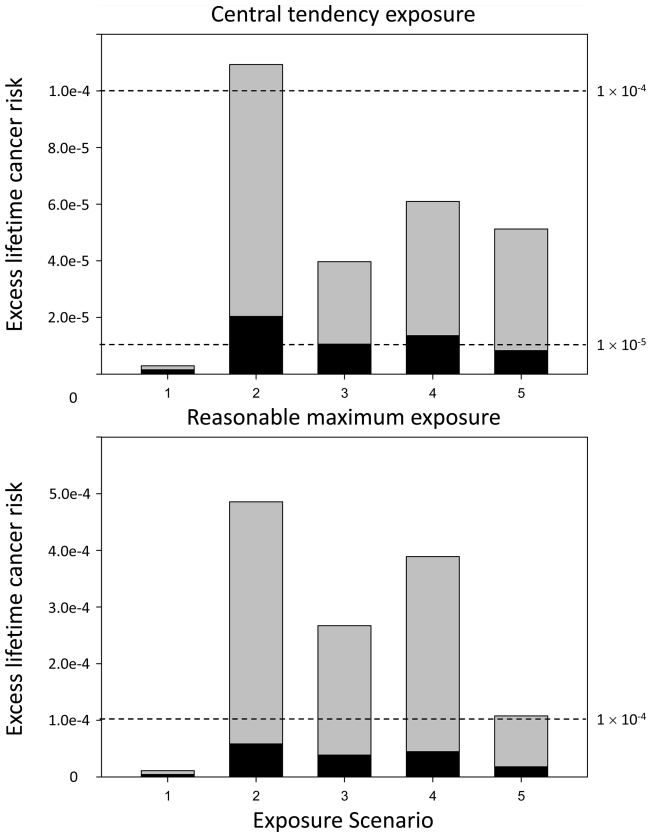


Figure 2. Deterministic excess lifetime cancer risk estimates for the five exposure scenarios described in Table 1 under central tendency and reasonable maximum exposure conditions. Risk attributable to dust is shown in black, and risk attributable to soil is shown in gray.

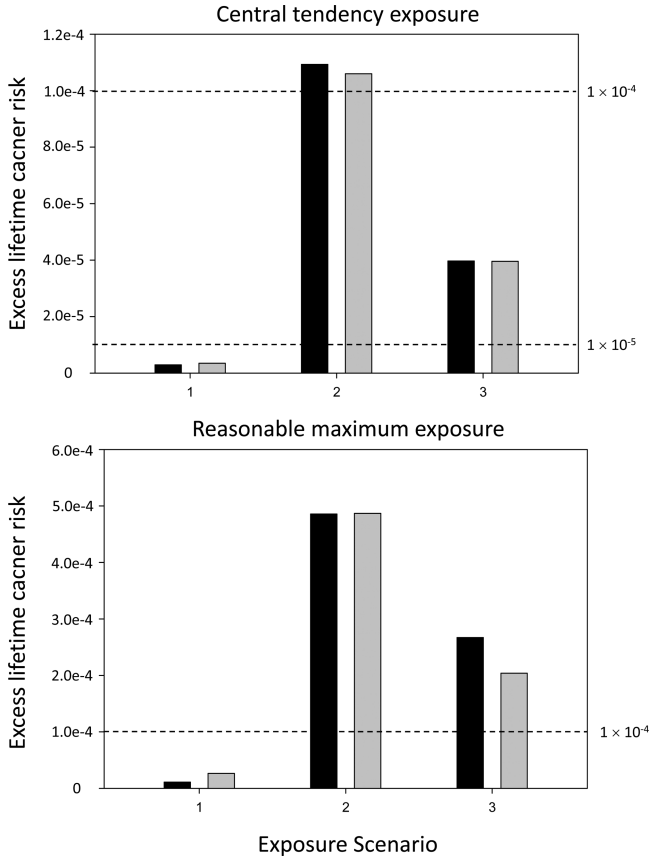


Figure 3. Comparison of deterministic and probabilistic estimates of excess lifetime cancer risk for three exposure scenarios for central tendency exposures (CTE) and reasonable maximum exposures (RME). Deterministic CTE estimates are analogous to 50th percentile probabilistic values, and deterministic RME estimates are analogous to 95th percentile probabilistic values. Black and gray bars depict deterministic and probabilistic risk estimates, respectively.

occurs during early childhood than it does for soil-mediated exposure, with 48 and 64% of the SHD-mediated risk occurring during the first 6 and 18 years of life, respectively. This difference results because the CTE IR for SHD is decreased to one-half its value at age 6 but the CTE IR for soil remains constant from 0−70 years of age (Supporting Information Table S2). All RME scenarios in CSA-affected environments (scenarios 2−5) had ELCR values for ingestion of SHD alone exceeding 1×10^{-5} but none exceeding 1×10^{-4}.

A probabilistic analysis (Monte Carlo) for scenarios 1, 2, and 3 yielded ELCR estimates in a range similar to those estimated deterministically (Table 2, Figure 3), where the 50th percentile statistic is treated as analogous to the CTE and the 95th percentile statistic is treated as analogous to the RME. As with deterministic estimates, probabilistic estimates for ELCR in CSA-affected settings for soil exposures (scenarios 2 and 3) were markedly higher than those for urban background settings (scenario 1) (Table 2). Probabilistic CTE ELCR estimates were very similar to deterministic estimates (Table 1), within 21% for urban background (scenario 1) and identical for 70-year lifespan and the first 6 years of life (scenarios 2 and 3). Probabilistic 95th percentile ELCR estimates differed more from the deterministic estimates, exceeding the deterministic RME for urban background (scenario 1) by a factor of more than 2 and being less than it for the first 6 years of life (scenario 3) by 26%, but the probabilistic and deterministic RME estimates for a 70-year lifespan (scenario 2) were identical.

Sensitivity analyses for the probabilistic ELCR estimates indicate that the proportion of the variability in ELCR contributed by contaminant concentration and IR was different for each scenario (Table 3). For environments where ingestion of UA-affected media only was considered (scenario 1), BaPEQ concentration contributed most of the variability and IR

Table 2. Summary of Probabilistic Estimates (Monte Carlo Simulations, 10 000 runs, 50th Percentile Represents the Central Tendency Exposure and 95th Percentile Represents the Reasonable Maximum Exposure) of Excess Lifetime Cancer Risk for Exposure Scenarios 1−3

	settled house dust only		soil only		dust and soil	
scenario	50th	95th	50th	95th	50th	95th
1	1.2×10^{-6}	1.4×10^{-5}	1.1×10^{-6}	1.6×10^{-5}	3.5×10^{-6}	2.6×10^{-5}
2	1.8×10^{-5}	1.2×10^{-4}	7.3×10^{-5}	4.3×10^{-4}	1.1×10^{-4}	4.9×10^{-4}
3	8.3×10^{-6}	6.1×10^{-5}	2.4×10^{-5}	1.7×10^{-4}	4.0×10^{-5}	2.0×10^{-4}

dx.doi.org/10.1021/es303371t | Environ. Sci. Technol. 2013, 47, 1101−1109

Table 3. Proportion of the Variability in Estimates of Excess Lifetime Cancer Risk Contributed by Parameters Considered[ab]

	scenario 1			scenario 2			scenario 3		
	dust alone	soil alone	dust and soil	dust alone	soil alone[c]	dust and soil[c]	dust alone	soil alone	dust and soil
$[BaPEQ]_{UA\ dust}$	0.71	--	0.33	--	--	--	0.03	--	
$[BaPEQ]_{CSA\ dust}$	--	--	--	0.55		0.07	0.35	--	0.07
$[BaPEQ]_{UA\ soil}$	--	0.80	0.42	--	--	--	--	0.01	0.01
$[BaPEQ]_{CSA\ soil}$	--	--	--	--	0.50	0.44	--	0.32	0.25
$IR_{dust,\ 0-6\ years}$	0.13	--	0.06	0.19	--	0.02	0.59	--	0.12
$IR_{dust,\ 6-70\ years}$	0.16	--	0.08	0.24	--	0.04	0.03	--	0.01
$IR_{soil,\ 0-18\ years}$	--	0.13	0.07	--	0.30	0.26	--	0.66	0.53
$IR_{soil,\ 18-70\ years}$	--	0.06	0.03	--	0.18	0.15	--		

[a][BaPEQ, benzo[a]pyrene equivalents; UA, unsealed asphalt pavement; CSA, coal-tar-sealed pavement; IR, ingestion rate]. [b]-- No contribution to variability is expected from this parameter. [c]Body weight 18−70 years of age contributed ∼1% to variability of estimates.

contributed relatively little. When lifetime exposure or exposure only during the first 6 years of life to CSA environments was considered (scenarios 2 and 3), IR contributed a greater proportion of the variability in estimated ELCR.

DISCUSSION

Four exposure scenarios for nondietary ingestion of CSA-affected soil and SHD resulted in estimated BaPEQ doses that are substantially elevated over the dose for urban background (Table 1). BaPEQ doses from nondietary ingestion of CSA-affected soil and dust range from 91 ng/kg/day during the first year of life to 9.1 ng/kg/day for adults. For comparison, Chuang et al. (1999)[26] reported dietary intake for the sum of B2 PAHs for children (2−4 years of age) in North Carolina as 24.8 ng/kg/day. Dietary intakes among adults of B2 PAHs have been estimated at between 1 and 5 μg/day on average (about 12.5−62.5 ng/kg/day).[44] We recently demonstrated that exposures to B2 PAHs in CSA-affected SHD are expected to exceed dietary intakes in children.[28]

ELCRs associated with CSA-affected settings (scenarios 2−5) greatly exceed those for the urban background (scenario 1). To put CSA-associated ELCRs into context, estimated CTE ELCR for lifetime exposure to CSA-affected soils (8.9×10^{-5}) exceeds that for urban soils in Beijing, China (1.77×10^{-6}),[45] and CTE ELCR for lifetime exposure to CSA-affected SHD (2.0×10^{-5}) exceeds that for exposure to urban surface dust (pavement and road dust) in an industrial area in China (1.05×10^{-6}).[46] However, estimated RME ELCR for lifetime exposure to CSA-affected SHD (5.82×10^{-5}) was less than that reported by Maertens et al. (2008)[47] for children in those residences in Ottawa, Canada, with SHD PAH in the top 10th percentile ($>1 \times 10^{-4}$), although the IR and SHD PAH concentrations were comparable to those used here. The difference likely arises because Maertens et al. included an adjustment factor in their risk analysis to account for exposures taking place during early life stages. ELCRs estimated here for CSA-affected settings exceed those for some other types of exposure to PAHs. For example, estimated CTE ELCRs for CSA-affected settings are much greater than those estimated for ingestion of grilled and smoked meat (2.63×10^{-7})[48] and for inhalation of granulates associated with intense 30-year activity on artificial turf (1×10^{-6} for presumed worst case conditions).[49]

The increased cancer risk associated with CSA-affecting settings likely affects a large number of people in the U.S. Use of the product is widespread in the U.S. east of the Continental Divide,[4] and it also is used in some parts of Canada.[8] Sealed parking lots constituted 1−2% of the area of four mixed commercial and residential neighborhoods mapped in Texas; in a suburb of Chicago, IL, sealcoated pavement constituted 4% of the area, and 89% of driveway area was sealcoated.[18]

Uncertainty. The analysis presented here contains several sources of uncertainty, and many of the choices made for the analysis result in conservative (lower) estimates of ELCR. Concentrations of one of the B2 PAH, diBahA, were not included in computation of BaPEQ because analytical difficulties resulted in nondetections in all but one of the SHD samples.[18] The cancer slope factor used was 7.3; Schneider et al., (2002)[50] on the basis of oral carcinogenicity studies with BaP and coal-tar mixtures, recommend use of a slope factor of 11.5, which would increase ELCR reported here by about 50%. No adjustment factor was used to account for increased risk associated with exposure during early life stages, when children are more susceptible to the effects of chemical exposures.[51]

Although seven carcinogenic PAHs, all of which have a RPF ≤ 1, were considered here, the USEPA recently has proposed that 24 PAHs, with RPFs ranging from 0.1 to 60, be used to determine the relative potency of PAH mixtures.[52] At least three of the PAHs with proposed RPFs exceeding 1—benzo[c]fluorene, proposed RPF of 30; dibenz[a,h]anthracene, proposed RPF of 10; and dibenzo[a,l]pyrene, proposed RPF of 30 [52]—are components of coal tar,[53,54] and BaPEQs associated with coal tar are estimated to increase by almost a factor of 10 if the proposed RPFs are adopted.[55]

Other elements of the analysis also contributed to conservative ELCRs estimates. Most importantly, the risk analysis presented here did not consider nondietary ingestion of outdoor dust on parking lots, driveways, and playgrounds with coal-tar-based sealcoat, as no data are available that quantify IR for these settings. PAH concentrations in dust from coal-tar-sealcoated pavement, however, are 10 or more times higher than those measured in CSA-affected SHD and soil: median BaPEQ concentrations reported range from 60 [2] to 392 μg/g.[18] Ingestion of 4−8 mg of dust from CSA parking lots per day in children less than 6 years of age would add 100 ng BaPEQ/kg/day to the overall dose (data not shown). By comparison, the maximum calculated dose in the CTE scenarios is 91 ng/kg/day.

Further, the BaPEQ concentrations for CSA SHD in the analysis presented here might underrepresent typical BaPEQ associated with CSA-affected environments, because the samples used as representative were collected in Austin in 2008, about 2 1/2 years after use of coal-tar-based pavement sealant was banned in that city.[56] It is not known if or how rapidly concentrations of PAH in SHD decrease as sealant on

the adjacent pavement ages. Inhalation of gas-phase PAHs also was not considered here, and recent measurements of air concentrations of PAHs indicate relatively high concentrations above old (3.6−8 yr) coal-tar-based sealant[58] and very high concentrations above pavement within hours to weeks following sealant application.[57]

Other sources of uncertainty in this risk analysis include choice of IRs, assumption of 100% bioavailability, sample size, and dust loading. Ingestion rate contributed a large proportion of the variability in estimated ELCR associated with CSA-affected settings. For this analysis we used IRs from.[37,39] Dust IRs recently recommended by the USEPA are higher than those used here, but soil IRs are lower.[40] Recalculation of risk estimates using those in the 2011 updated version of the Handbook slightly changes risk estimates but does not change the overall conclusions of our assessment. The assumption of 100% bioavailability likely causes moderate overstatement of risks from ingestion of CSA-affected SHD and soil. The bioavailability of PAHs in abraded particles of coal tar-based sealant has not been investigated, and thus the relevance of studies of the bioavailability of BaP and other B2 PAHs in soil may or may not be robustly applicable to these calculations. Our calculations indicate that bioavailability on the order of 20% would still be associated with risk in excess of 1×10^{-4} in some exposure scenarios (RME, scenario 2). Bioavailability of PAHs in soil has been observed to range as high as 90%.[21]

The data set available for PAHs specifically associated with CSA- and UA-affected settings was relatively small. In particular, data from only three soil samples were available for soil adjacent to unsealed asphalt. However, these concentrations are consistent with upper ranges of concentrations reported in the literature as "background." Sensitivity analysis indicates that the much of the variability in risk estimates arises from concentrations of BaPEQ in SHD and soil (Table 3).

Finally, the data on dust loading adds some uncertainty to the risk estimates. Recall that one data point in the UA SHD data set is an outlier (883 $\mu g/cm^2$, compared to a mean of 85 $\mu g/cm^2$ for the remaining 6 data points). Reanalysis of the set without this data point shows that CSA settings had a significantly higher dust loading than the UA settings (p = 0.043, Student's t test). The source of this difference between the sampled settings is unclear.

In this analysis, lifetime estimated ELCRs for deterministic and probabilistic approaches were virtually identical (Tables 1 and 2, Figure 3). This indicates that point estimates for these parameters, as applied here, reasonably represent values in the center and upper reaches of the distributions of these data. Several of the factors contributing to uncertainty associated with the ELCRs presented here could be more fully accounted for with additional data, resulting in less uncertainty. Because the recognition of coal-tar-based pavement sealants as a source of PAHs to the environment is relatively recent (the first study was published in 2004), there are data gaps for such information as bioavailability of PAHs associated with dried sealant particles, IRs for pavement dust, and change in PAH concentrations in CSA-affected soils and SHD with time since sealant application. Additional data on PAH concentrations in CSA-affected soils and SHD will result in more robust ELCR estimates.

Estimates of excess cancer risk arising from exposure to carcinogenic PAHs in settled house dust and soil near coal tar-sealed parking lots exceeded 1×10^{-4} for the central tendency estimate for lifetime exposure, and for reasonable maximum estimates for all exposure scenarios considered. Exposure to these compounds in settled house dust is a particularly important source of risk for children younger than 6 years of age, as they are expected to ingest this material at higher rates. This indicates that the use of coal-tar-based pavement sealants magnifies aggregate exposures to B2 PAHs in children and adults in residences adjacent to where these products are used, and is associated with human health risks in excess of widely accepted standards. Although the analysis presented here is based on a limited data set, the results indicate that biomonitoring might be warranted to characterize the exposure of children and adults to PAHs associated with coal-tar-based pavement sealant.

■ ASSOCIATED CONTENT

Ⓢ Supporting Information

Additional information on dose and exposure assumptions, estimated doses, and dust loading. Table S1. Mass of house dust (<0.5 mm) collected, area sampled, surface dust loading, and benzo[a]pyrene equivalent (BaPEQ) loading for 18 apartments in the Austin, Tex., area. Table S2. Exposure assumptions for deterministic and probabilistic risk calculations. Table S3. Theoretical yearly doses of benzo[a]pyrene equivalents under central tendency and reasonable maximum exposure conditions. This material is available free of charge via the Internet at http://pubs.acs.org.

■ AUTHOR INFORMATION

Corresponding Author

*Phone: (254) 710-2468; fax: (254) 710-2580; E-mail: sp_williams@baylor.edu, spencer.williams.phd@gmail.com.

Notes

The authors declare no competing financial interest.

■ ACKNOWLEDGMENTS

This work was conducted without the benefit of external funding. Any use of trade, product, or firm names is for descriptive purposes only and does not imply endorsement by the U.S. Government.

■ ABBREVIATIONS:

AT	averaging time
B2 PAH	carcinogenic polycyclic aromatic hydrocarbons (classified B2 by EPA)
BaP	benzo[a]pyrene
CSA	coal-tar-sealed asphalt
CTE	central tendency exposure
BaPEQ	benzo[a]pyrene equivalents
BW	body weight
ED	exposure duration
EF	exposure frequency
ELCR	excess lifetime cancer risk
IR	ingestion rate
PAH	polycyclic aromatic hydrocarbon
RME	reasonable maximum exposure
RPF	relative potency factor
SHD	settled house dust
SHEDS	Stochastic Human Exposure and Dose Simulation
UA	unsealed asphalt

REFERENCES

(1) Mahler, B. J.; Van Metre, P. C.; Bashara, T. J.; Wilson, J. T.; Johns, D. A. Parking lot sealcoat: An unrecognized source of urban polycyclic aromatic hydrocarbons. *Environ. Sci. Technol.* **2005**, *39* (15), 5560−5566.

(2) *Polycyclic Aromatic Hydrocarbons Released from Sealcoated Parking Lots—a Controlled Field Experiment to Determine if Sealcoat Is a Significant Source of PAHs in the Environment*; University of New Hampshire Stormwater Center. Final Report; University of New Hampshire Stormwater Center: Durham, NH, 2010.

(3) Van Metre, P. C.; Mahler, B. J. Contribution of PAHs from coal-tar pavement sealcoat and other sources to 40 U.S. lakes. *Sci. Total Environ.* **2010**, *409* (2), 334−344, DOI: S0048-9697(10)00847-8[pii] 10.1016/j.scitotenv.2010.08.014.

(4) Van Metre, P. C.; Mahler, B. J.; Wilson, J. T. PAHs underfoot: contaminated dust from coal-tar sealcoated pavement is widespread in the United States. *Environ. Sci. Technol.* **2009**, *43* (1), 20−25.

(5) Yang, Y.; Van Metre, P. C.; Mahler, B. J.; Wilson, J. T.; Ligouis, B.; Razzaque, M. D.; Schaeffer, D. J.; Werth, C. J. Influence of coal-tar sealcoat and other carbonaceous materials on polycyclic aromatic hydrocarbon loading in an urban watershed. *Environ. Sci. Technol.* **2010**, *44* (4), 1217−1223, DOI: 10.1021/es902657h.

(6) Mahler, B. J.; Van Metre, P. C.; Wilson, J. T.Concentrations of polycyclic aromatic hydrocarbons (PAHs) and major and trace elements in simulated rainfall runoff from parking lots, Austin, Texas, 2003 (version 3); U.S. Geological Survey Open-File Report: 2004−1208, p. 87

(7) Scoggins, M.; Ennis, T.; Parker, N.; Herrington, C. A photographic method for estimating wear of coal tar sealcoat from parking lots. *Environ. Sci. Technol.* **2009**, *43* (13), 4909−4914.

(8) Diamond Environmental Group. Reconnaissance study of coal tar sealcoat application in Toronto and an eastimate of related PAH emissions. Departments of Geography and Chemical Engineering, University of Toronto, 2011.

(9) National Institutes of Health. Coal tars and coal-tar pitches. National Institute of Environmental Health Science, National Institutes of Health, Department of Health and Human Services, 2011.

(10) Kaushik, S.; Rainal, R. K.; Bhatiaz, G.; Verma, G.; Khandal, R. K. Modification of coal tar pitch by chemical method to reduce benzo(a)pyrene. *Curr. Sci.* **2007**, *93* (4), 540−544.

(11) *Water: CWA Methods, Priority Pollutants*; http://water.epa.gov/scitech/methods/cwa/pollutants.cfm.

(12) Mahler, B. J.; Metre, P. C.; Crane, J. L.; Watts, A. W.; Scoggins, M.; Williams, E. S. Coal-tar-based pavement sealcoat and PAHs: implications for the environment, human health, and stormwater management. *Environ. Sci. Technol.* **2012**, *46* (6), 3039−3045.

(13) Van Metre, P. C.; Mahler, B. J.; Furlong, E. T. Urban sprawl leaves its PAH signature. *Environ. Sci. Technol.* **2000**, *34* (19), 4064−4070, DOI: 10.1021/es991007n.

(14) Bryer, P. J.; Scoggins, M.; McClintock, N. L. Coal-tar based pavement sealant toxicity to freshwater macroinvertebrates. *Environ. Pollut.* **2010**, *158* (5), 1932−1937, DOI: 10.1016/j.envpol.2009.10.038.

(15) Scoggins, M.; McClintock, N. L.; Gosselink, L.; Bryer, P. Occurrence of polycyclic aromatic hydrocarbons below coal-tar-sealed parking lots and effects on stream benthic macroinvertebrate communities. *J. North Am. Benthol. Soc.* **2007**, *26* (4), 694−707.

(16) Bommarito, T.; Sparling, D. W.; Halbrook, R. S. Toxicity of coal-tar pavement sealants and ultraviolet radiation to *Ambystoma maculatum*. *Ecotoxicology* **2010**, *19* (6), 1147−1156, DOI: 10.1007/s10646-010-0498-8.

(17) Bryer, P. J.; Elliott, J. N.; Willingham, E. J. The effects of coal tar based pavement sealer on amphibian development and metamorphosis. *Ecotoxicology* **2006**, *15* (3), 241−247, DOI: 10.1007/s10646-005-0055-z.

(18) Mahler, B. J.; Metre, P. C.; Wilson, J. T.; Musgrove, M.; Burbank, T. L.; Ennis, T. E.; Bashara, T. J. Coal-tar-based parking lot sealcoat: An unrecognized source of PAH to settled house dust. *Environ. Sci. Technol.* **2010**, *44* (3), 894−900, DOI: 10.1021/es902533r.

(19) Agency for Toxic Substances and Disease Registry. Toxicological profile for polycyclic aromatic hydrocarbons. Atlanta, GA, U.S. Department of Health and Human Services, Public Health Service, 1995.

(20) Hoffmann, D.; Hoffmann, I. The changing cigarette, 1950−1995. *J. Toxicol. Environ. Health.* **1997**, *50* (4), 307−364, DOI: 10.1080/009841097160393.

(21) Ramesh, A.; Walker, S. A.; Hood, D. B.; Guillen, M. D.; Schneider, K.; Weyand, E. H. Bioavailability and risk assessment of orally ingested polycyclic aromatic hydrocarbons. *Int. J. Toxicol.* **2004**, *23* (5), 301−333, DOI: WGMX5TX4L3U8CJF7 [pii]10.1080/10915810490517063.

(22) Jones-Otazo, H. A.; Clarke, J. P.; Diamond, M. L.; Archbold, J. A.; Ferguson, G.; Harner, T.; Richardson, G. M.; Ryan, J. J.; Wilford, B. Is house dust the missing exposure pathway for PBDEs? An analysis of the urban fate and human exposure to PBDEs. *Environ. Sci. Technol.* **2005**, *39* (14), 5121−5130.

(23) Lioy, P. J.; Freeman, N. C.; Millette, J. R. Dust: A metric for use in residential and building exposure assessment and source characterization. *Environ. Health Perspect.* **2002**, *110* (10), 969−983, DOI: sc271_5_1835 [pii].

(24) Maertens, R. M.; Bailey, J.; White, P. A. The mutagenic hazards of settled house dust: A review. *Mutat. Res.* **2004**, *567* (2−3), 401−425, DOI: S1383-5742(04)00061-4 [pii]10.1016/j.mrrev.2004.08.004.

(25) Whitehead, T.; Metayer, C.; Gunier, R. B.; Ward, M. H.; Nishioka, M. G.; Buffler, P.; Rappaport, S. M. Determinants of polycyclic aromatic hydrocarbon levels in house dust. *J. Expo. Sci. Environ. Epidemiol.* **2011**, *21* (2), 123−132, DOI: jes200968 [pii] 10.1038/jes.2009.68.

(26) Chuang, J. C.; Callahan, P. J.; Lyu, C. W.; Wilson, N. K. Polycyclic aromatic hydrocarbon exposures of children in low-income families. *J. Expo. Anal. Environ. Epidemiol.* **1999**, *9* (2), 85−98.

(27) Wilson, N. K.; Chuang, J. C.; Lyu, C.; Menton, R.; Morgan, M. K. Aggregate exposures of nine preschool children to persistent organic pollutants at day care and at home. *J. Expo. Anal. Environ. Epidemiol.* **2003**, *13* (3), 187−202, DOI: 10.1038/sj.jea.7500270 [pii].

(28) Williams, E. S.; Mahler, B. J.; Van Metre, P. C. Coal-tar pavement sealants might substantially increase children's PAH exposures. *Environ. Pollut.* **2012**, *164*, 40−41 DOI:.

(29) U.S. Environmental Protection Agency. Risk Assessment Guidance for Superfund Volume I, Human Health Evaluation Manual (Part A). EPA/540/1-89/002; Office of Research and Development: Washington, DC, 1989.

(30) U.S. Environmental Protection Agency. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. EPA/600/R-93/089; Office of Research and Development: Washington, DC, 1993.

(31) Nam, J. J.; Sweetman, A. J.; Jones, K. C. Polynuclear aromatic hydrocarbons (PAHs) in global background soils. *J. Environ. Monit.* **2009**, *11* (1), 45−48, DOI: 10.1039/b813841a.

(32) Lemieux, C. L.; Lambert, I. B.; Lundstedt, S.; Tysklind, M.; White, P. A. Mutagenic hazards of complex polycyclic aromatic hydrocarbon mixtures in contaminated soil. *Environ. Toxicol. Chem.* **2008**, *27* (4), 978−990, DOI: 07-157 [pii]10.1897/07-157.1.

(33) Turczynowicz, L.; Fitzgerald, D. J.; Nitschke, M.; Mangas, S.; McLean, A. Site contamination health risk assessment case study involving tenant relocation from a former gasworks site. *J. Toxicol. Environ. Health A* **2007**, *70* (19), 1638−1653, DOI: 781628196 [pii] 10.1080/15287390701434737.

(34) Ozkaynak, H.; Xue, J.; Zartarian, V. G.; Glen, G.; Smith, L. Modeled estimates of soil and dust ingestion rates for children. *Risk Anal.* **2010**, *31* (4), 592−608, DOI: 10.1111/j.1539-6924.2010.01524.x.

(35) *Consolidated Human Activity Database*; (http://www.epa.gov/chadnet1/). .

(36) Harrad, S.; Ibarra, C.; Diamond, M.; Melymuk, L.; Robson, M.; Douwes, J.; Roosens, L.; Dirtu, A. C.; Covaci, A. Polybrominated

diphenyl ethers in domestic indoor dust from Canada, New Zealand, United Kingdom and United States. *Environ. Int.* **2008**, *34* (2), 232−238, DOI: S0160-4120(07)00160-2 [pii]10.1016/j.envint.2007.08.008.
(37) *Exposure Factors Handbook (Final Report)*; U.S. Environmental Protection Agency: Washington, D.C., 1997.
(38) U.S. Environmental Protection Agency. *Child-Specific Exposure Factors Handbook*. Washington, DC, USEPA, 2008.
(39) Van Holderbeke, M.; Cornelis, C.; Bierkens, J.; Torfs, R. Review of the soil ingestion pathway in human exposure assessment. VITO/RIVM. Flanders, Belgium, VITO/RIVM, 2008.
(40) U.S. Environmental Protection Agency. *Exposure Factors Handbook*, 2011 ed.; Washington, DC, USEPA, 2011.
(41) Portier, K.; Tolson, J. K.; Roberts, S. M. Body weight distributions for risk assessment. *Risk Anal.* **2007**, *27* (1), 11−26, DOI: 10.1111/j.1539-6924.2006.00856.x.
(42) *HH: Risk Characterization, Region 8*; http://ehp03.niehs.nih.gov/static/instructions.action#type.
(43) *Integrated Risk Information System, Benzo[a]pyrene (BaP) (CASRN 50−32−8)*; http://www.epa.gov/iris/subst/0136.htm.
(44) Menzie, C. A.; Potocki, B. B.; Santodonato, J. Exposure to Carcinogenic PAHs in the Environment. *Environ. Sci. Technol.* **1992**, *26* (7), 1278−1284.
(45) Peng, C.; Chen, W.; Liao, X.; Wang, M.; Ouyang, Z.; Jiao, W.; Bai, Y. Polycyclic aromatic hydrocarbons in urban soils of Beijing: Status, sources, distribution and potential risk. *Environ. Pollut.* **2011**, *159* (3), 802−808, DOI: S0269-7491(10)00511-7 [pii]10.1016/j.envpol.2010.11.003.
(46) Wang, W.; Huang, M. J.; Kang, Y.; Wang, H. S.; Leung, A. O.; Cheung, K. C.; Wong, M. H. Polycyclic aromatic hydrocarbons (PAHs) in urban surface dust of Guangzhou, China: Status, sources and human health risk assessment. *Sci. Total Environ.* **2011**, *409* (21), 4519−4527, DOI: S0048-9697(11)00748-0 [pii]10.1016/j.scitotenv.2011.07.030.
(47) Maertens, R. M.; Yang, X.; Zhu, J.; Gagne, R. W.; Douglas, G. R.; White, P. A. Mutagenic and carcinogenic hazards of settled house dust. I: Polycyclic aromatic hydrocarbon content and excess lifetime cancer risk from preschool exposure. *Environ. Sci. Technol.* **2008**, *42* (5), 1747−1753.
(48) Alomirah, H.; Al-Zenki, S.; Husain, A.; Sawaya, W.; Ahmed, N.; Gevao, B.; Kannan, K. Benzo[a]pyrene and total polycyclic aromatic hydrocarbons (PAH) levels in vegetable oils and fats do not reflect the occurrence of the eight genotoxic PAHs. *Food Addit. Contam. Part A: Chem. Anal. Control Expo. Risk Assess* **2010**, *27* (6), 869−878.
(49) Menichini, E.; Abate, V.; Attias, L.; De Luca, S.; di Domenico, A.; Fochi, I.; Forte, G.; Iacovella, N.; Iamiceli, A. L.; Izzo, P.; Merli, F.; Bocca, B. Artificial-turf playing fields: Contents of metals, PAHs, PCBs, PCDDs and PCDFs, inhalation exposure to PAHs and related preliminary risk assessment. *Sci. Total Environ.* **2011**, *409* (23), 4950−4957, DOI: S0048-9697(11)00760-1[pii]10.1016/j.scitotenv.2011.07.042.
(50) Schneider , K.; Roller, M.; Kalberlah, F.; Schuhmacher-Wolz , U. Cancer risk assessment for oral exposures to PAH mixtures. *J. Appl. Toxicol.* **2002**, *22* (1), 73−83.
(51) World Health Organization. Principles for evaluating health risks in children associated with exposure to chemicals. Geneva, Switzerland, 2006.
(52) U.S. Environmental Protection Agency. Development of a relative potency factor (RPF) approach for polycyclic aromatic hydrocarbon (PAH) mixtures. Washington, DC, 2010.
(53) Agency for Toxic Substances and Disease Registry. Toxicological profile for creosote. Atlanta, GA, U.S. Department of Health and Human Services, Public Health Service, 2002.
(54) Wise, S. A.; Poster, D. L.; Leigh, S. D.; Rimmer, C. A.; Mossner, S.; Schubert, P.; Sander, L. C.; Schantz, M. M. Polycyclic aromatic hydrocarbons (PAHs) in a coal tar standard reference material–SRM 1597a updated. *Anal. Bioanal. Chem.* **2010**, *398* (2), 717−728, DOI: 10.1007/s00216-010-4008-x.
(55) Rohr, A. C. Comments on development of a relative potency factor (RPF) approach for polycyclic aromatic hydrocarbon (PAH) mixtures, external review draft. Electric Power Research Institute: Palo Alto, CA, 2010.
(56) City of Austin. An ordinance amending the city code to add a new chapter 6−6 relating to coal tar pavement products, creating offenses, and providing penalties. 2051117−070. Austin, Texas, 2005.
(57) Van Metre, P. C.; Majewski, M. S.; Mahler, B. J.; Foreman, W. T.; Braun, C. L.; Wilson, J. T.; Burbank, T. L. PAH volatilization following application of coal-tar-sealed pavement. *Atmos. Environ.* **2012**, *51*, 108−115.
(58) Van Metre, P. C.; Majewski, M. S.; Mahler, B. J.; Foreman, W. T.; Braun, C. L.; Wilson, J. T.; Burbank, T. L. Volatilization of polycyclic aromatic hydrocarbons from coal-tar-sealed pavement. *Chemosphere* **2012**, *88* (1), 1−7, DOI: S0045-6535(11)01466-4[pii] 10.1016/j.chemosphere.2011.12.072.

Respiratory Symptoms and Lung Functional Impairments Associated with Occupational Exposure to Asphalt Fumes

M Neghab1, F Zare Derisi2, J Hassanzadeh3

1. Department of Occupational Health and Research Center for Health Sciences, Shiraz University of Medical Sciences, Shiraz, Iran
2. Student Research Committee, School of Health, Shiraz University of Medical Sciences, Shiraz, Iran
3. 3Department of Clinical Epidemiology, School of Health, Shiraz University of Medical Sciences, Shiraz, Iran

Correspondence to

Forough Zare Derisi, MSc Student of Occupational Health, Student Research Committee, School of Health, Shiraz University of Medical Sciences, Shiraz, Iran Tel/Fax: +98-71-3627-2045

E-mail: zarederis@sums.ac.ir Received: Jun 27, 2014 Accepted: Aug 27, 2014

Abstract

Background: Controversy exists as to the potential of asphalt fumes to induce respiratory symptoms and lung functional impairments.

Objective: To examine the respiratory effects, if any, of occupational inhalation exposure to asphalt fumes.

Methods: In this cross-sectional study, 74 asphalt workers and 110 unexposed employees were investigated. The prevalence of respiratory symptoms among subjects was investigated by a standard questionnaire. Additionally, the parameters of pulmonary function were measured both, prior to exposure and at the end of work-shift. Furthermore, to assess the extent to which workers were exposed to asphalt fumes, total particulate and the benzene-soluble fraction were measured in different worksites.

Results: The mean levels of exposure to total particulate and benzene-soluble fraction in asphalt fumes were estimated to be 0.9 (SD 0.2) and 0.3 (SD 0.1) mg/m3, respectively. Mean values of FEV1, both prior to the exposure (89.58% [SD 18.69%] predicted value) and at the end of shift (85.38% [SD 19.4%]), were significantly ($p<0.05$) smaller than those of the comparison subjects (93.88% [SD 13.93%]). Similarly, pre-shift (87.05 [SD 8.57]) and post-exposure (89.95 [SD 6.85]) FEV1/FVC ratio were both significantly ($p<0.01$) lower than those of the unexposed employees (107.56 [SD 9.64]). Moreover, the prevalence of respiratory symptoms such as cough and wheezing in exposed employees were 41% and 42%, respectively. The corresponding values for comparison subjects were 10.0% and 3.6%, respectively ($p<0.001$). The pattern of changes in parameters of lung function in asphalt workers was consistent with that of chronic obstructive lung disease.

Conclusion: Significant decrements in the parameters of pulmonary function as well as, a significant increase in the prevalence of respiratory symptoms in asphalt paving workers compared to their

unexposed counterparts provided evidence in favor of a significant association between exposure to asphalt fumes and lung function impairments.

Keywords: Asphalt; Respiratory function tests; Signs and symptoms, respiratory; Occupational exposure; Mastic asphalt; Questionnaires; Benzene; Worksites; Threshold limit values; Air borne disease

Introduction

Asphalt is produced by heating and drying gravel and mixing it with 4%–5% of hot bitumen. Bitumen is the residue of the distillation of selected petroleum crude oils. Fillers and fibers are also added to modify the properties of the asphalt, and small amounts of aliphatic amines are used to improve the binding between the bitumen and the stone materials.1

Asphalt workers are exposed to a wide variety of modulators and modifiers added to the asphalt, such as antioxidants, anti-corrosive agents, fillers, fibers, oxidants, plastics, rubber, waste materials and other volatile products that are released from the asphalt.2,3

A major risk associated with exposure to asphalt is being exposed to polycyclic aromatic hydrocarbons and alkyl derivatives, which are byproducts of petroleum processing or combustion. These are highly carcinogenic at relatively low concentrations.1,4-6 Additionally, asphalt workers are exposed to emissions from the exhaust of passing vehicles.4,7-9 Respiratory effects of exposure to asphalt fume have been evaluated in a few studies.5 However, conclusive results have not been obtained yet.

For instance, many researchers have shown a significant reduction in some parameters of pulmonary function as well as upper respiratory tract irritation and shortness of breath in asphalt workers.5,8,10-13

Some studies showed that exposure to asphalt fumes is associated with the incidence of respiratory symptoms.4,10,14,15 Moreover, bronchitis and emphysema have been reported in asphalt workers.16,17 On the other hand, some studies have not found a consistent relationship between exposure to asphalt fumes and decline in the parameters of lung function or increase in the prevalence of respiratory symptoms in asphalt workers.18 Similarly, Butler, et al,19 in a study on asphalt workers did not find an increased risk for obstructive pulmonary diseases.

The issue of asphalt fumes-induced respiratory disorders is subject to debate and controversy and requires further investigation.5 The respiratory effects of asphalt fumes have so far been studied in a few countries such as USA,10,12,16, Germany,7 and Norway.1,4 However, for differences in the chemical structure and composition of asphalt concrete mixtures, the concentration of asphalt fumes to which workers were exposed, air temperature, mechanical rather than manual processing of asphalt, good occupational health practices in these countries and many other differences, the results of these studies are not necessarily comparable with those of other studies. To the best of our knowledge, no study has been conducted on this issue in Iran. We therefore, conducted this study to examine the possible respiratory effects of exposure to asphalt fumes in asphalt workers.

Materials and Methods

This cross-sectional study was carried out to evaluate the respiratory effects of occupational exposure to asphalt fumes in paving workers of Shiraz, southern Iran. The sample size was calculated based on the expected prevalence of respiratory illness of 5% in unexposed employees, and 20% in asphalt workers, a study power of 80%, and an α of 0.05.4

A total of 74 asphalt paving workers (exposed group) and 110 unexposed employees from governmental departments were randomly selected and served as the comparison group.

The study was conducted in accordance with the Helsinki Declaration of 1964 as revised in 2007.20 Both the exposed and unexposed participants were volunteers. No subject refused to participate in the study. All participants signed an informed consent form before commencement of the study. The protocol of the study was approved by Shiraz University of Medical Sciences Ethics Committee.

None of the exposed subjects had past medical or family history of respiratory illnesses or any other chest operations or injuries. Similarly, none of the subjects in the comparison group had been exposed to asphalt fumes or other chemicals known to cause respiratory symptoms or pulmonary diseases during the course of their employment or prior to it. Only two exposed employees did not meet the criteria to enter the study and were excluded due to pre-existing medical conditions and chest operation.

Measurement of the Study Variables

Respiratory illness

Subjects were interviewed by one of the authors (FZD). A respiratory symptom questionnaire, as suggested by the American Thoracic Society,21 with a few modifications, was administrated to the participants.22 This standardized questionnaire included questions regarding respiratory symptoms (presence or absence of regular dry and/or productive cough, wheezing, shortness of breath, etc), nasal and eye symptoms and smoking habits, as well as occupational, medical and family history of each subject. Symptoms of chronic respiratory disorders included cough with sputum at any time during the day or night for at least three months of the year and for at least two consecutive years. Information extracted from the questionnaires were then used to determine the prevalence of symptoms among the exposed and unexposed groups.

Pulmonary function tests

Pulmonary function tests (PFTs) were performed using a portable calibrated Vitalograph spirometer (Model ST-150, manufactured by a joint Japanese-Philippinian company, Fukuda Sangyo Co, Ltd) on-site. The parameters of pulmonary function were measured twice for the exposed group (pre-shift after a 72-hour exposure-free period and post-shift) and once for the comparison group according to the protocol the details of which are described elsewhere.23,24 The measured parameters included mean percentage predicted vital capacity (VC), forced vital capacity (FVC), forced expiratory volume during the first second (FEV1), and peak expiratory flow (PEF).

Measurement of atmospheric concentrations of asphalt fumes

To assess the extent of subjects' exposure to airborne contaminants, atmospheric concentrations of total particulate (TP) and benzene-soluble fraction (BSF) were measured in different work areas according to the NIOSH analytical method 5042.25 Samples were collected by a personal air sampling pump (Scientific Kit Corporation) equipped with a poly-tetra-fluoro-ethylene (PTFE) membrane filter (2-µm pore size) in a 37-mm cassette filter holder. The samples were re-weighed after sampling; the concentration of TP was calculated based on the weight difference, and the total air volume sampled. After determination of TP, each filter was extracted with benzene for the determination of BSF, which is the gravimetric amount of the TP that is benzene soluble. Filters were submerged in benzene; the soluble parts were weighed to determine the amount of BSF.

Statistical Analysis

The data were analyzed by SPSS® ver 16.0 for Windows®. Student's t test for independent samples, $\chi 2$ or Fisher's exact test, Mann-Whitney U test and logistic and multiple linear regression analysis, were used. A p value <0.05 was considered statistically significant. Continuous variables with normal distribution were presented as mean (SD). Variables such as age, weight, height, smoking habits, education, and marital status were considered as potential confounders and their effects on the prevalence of respiratory symptoms and changes in pulmonary function indices were controlled. The initial model was constructed based on the exposure variable as well as all potential confounding variables. Using the backward elimination method and keeping the main exposure variable, asphalt fume, in the model, the final model was obtained.

Results

Demographic characteristics of the studied groups are presented in Table 1. No significant differences were noted for weight, height, length of employment, number of smokers, duration and intensity of smoking between the two studied groups. Nonetheless, the exposed group, on average, was about 3.5 years older than the comparison group ($p=0.016$). The mean atmospheric concentration of asphalt fumes did not exceed the current threshold limit value (TLV) of 0.5 mg/m3 set by the American Conference of Governmental Industrial Hygienists (ACGIH).26 Pulmonary function test parameters measured in the exposed and unexposed groups are presented in Table 2. VC, FVC, FEV1, and FEV1/FVC declined significantly after a working day in asphalt workers compared to pre-exposure values ($p<0.05$). Moreover, FEV1/FVC and FEV1/VC measured pre-shift in asphalt workers were significantly ($p<0.001$) lower than those in the comparison group.

Table 1: Demographic characteristics of the studied groups. Values are mean (SD), median [IQR], or n (%).

Variable	Exposed (n=74)	Comparison (n=110)	p value
Age (yr)	37.4 (10.9)	33.8 (8.1)	0.016
Height (cm)	174.0 (7.6)	173.3 (6.9)	0.538
Weight (kg)	73.5 (12.1)	70.2 (11.9)	0.074

Length of exposure/employment (yr)	10 [15.5]	8 [6.5]	0.239
Level of education			
Illiterate	9 (12%)	0 (0%)	
Diploma	34 (46%)	19 (17.3%)	0.001
Higher education	31 (42%)	91 (82.7%)	
Marital status			
Single	8 (11%)	12 (10.9%)	0.983
Married	66 (89%)	98 (89.1%)	
Body mass index (kg/m2)	24.3 (3.7)	23.4 (3.8)	0.122
Smokers	19 (26%)	22 (20%)	0.364
Length of smoking (yr)	8.3 (5.7)	9.8 (7.5)	0.465
Number of cigarettes smoked per day	7.8 (6.6)	4.7 (3.0)	0.058
Air-bone concentration of TP (mg/m3)	0.9 (0.2)	—	—
Air-bone concentration of BSF (mg/m3)	0.3 (0.1)	—	—

Table 2: Pulmonary function indices of asphalt workers and comparison group (before and after exposure). Values are mean (SD) percent predicted value.

Variable	Exposed		Comparison (n=110)	p value	
	Pre-shift (n=74)	Post-shift (n=74)		Pre-shift vs post-shift exposed group	Pre-shift exposed vs comparison group
VC	91.23 (4.98)	83.34 (15.28)	93.69 (13.67)	<0.001	0.251
FVC	85.91 (18.81)	78.92 (18.57)	87.58 (13.15)	0.004	0.508
FEV1	89.58 (18.69)	85.38 (19.4)	93.88 (13.93)	0.021	0.094
PEF	81.9 (22.8)	78.79 (23.64)	86.25 (18.85)	0.096	0.160
FEV1/VC	79.5 (15.35)	81.35 (16.76)	100.8 (11.91)	0.385	<0.001
FEV1/FVC	87.05 (8.57)	89.95 (6.85)	107.56 (9.64)	0.008	<0.001

Table 3 shows the prevalence of respiratory symptoms among asphalt workers and comparison group. The prevalence of all respiratory symptoms studied was significantly ($p<0.001$) higher in the exposed group than in the comparison group. Binary logistic regression analysis of data, where age, length of exposure, weight, height, education level, and smoking were considered independent variables, significant ($p<0.001$) association was found between exposure to asphalt fumes and the prevalence of all respiratory symptoms but chest tightness (Table 4).

Table 3: Frequency (%; 95% CI) of respiratory symptoms among asphalt workers and comparison group. All symptoms were significantly ($p<0.001$) more prevalent in exposed than in the comparison group.

Variable	Exposed (n=74)	Comparison (n=110)
Cough	30 (41; 29 to 52)	11 (10.0; 4.3 to 15.7)
Phlegm	28 (38; 27 to 49)	10 (9.1; 3.7 to 14.5)
Productive cough	27 (36; 25 to 48)	7 (6.4; 1.7 to 11.0)
Wheezing	31 (42; 30 to 53)	4 (3.6; 0.1 to 7.2)
Shortness of breath	22 (30; 19 to 40)	6 (5.5; 1.2 to 9.8)
Chest tightness	13 (18; 9 to 26)	0 (0; 0 to 0)

Table 4: Association between exposure to asphalt fumes and development of respiratory symptoms (binary logistic regression analysis).

Outcome	OR (95% CI)
Cough	6.9 (3.1 to 15.4)
Phlegm	6.7 (2.9 to 15.5)
Productive cough	8.5 (3.4 to 21.1)
Wheezing	18.1 (5.9 to 56.0)
Shortness of breath	6.9 (2.6 to 18.9)

After adjusting for age, length of exposure, weight, height, education level, and smoking, multiple linear regression analysis revealed a significant ($p<0.001$) negative correlation between exposure to asphalt fumes and FEV1/VC and FEV1/FVC ratios. Exposure to asphalt fume reduced the FEV1/VC, and FEV1/FVC by 20.4% and 20.3%, respectively (Table 5).

Table 5: Association between exposure to asphalt fumes and changes in pulmonary function test indices (multiple linear regression analysis).

Dependent variable	β (95% CI)
VC	–3.1 (–6.9 to 0.7)
VC	–1.7 (–6.3 to 2.9)
FEV1	–3.6 (–8.2 to 1.1)
PEF	–4.4 (–10.4 to 1.6)
FEV1/VC	–20.4 (–24.3 to –16.5)
FEV1/FVC	–20.3 (–23.0 to –17.6)

Discussion

Apart from age and level of education, there was no significant difference between the two studied groups in terms of other variables. There was also no significant difference in the number of smokers and smoking intensity between the two groups. Therefore, it is unlikely that smoking accounted for the differences observed in spirometry results. The significant reduction in FVC, FEV1, and FEV1/FVC, and the significant increase in the respiratory symptoms are therefore, likely to be the result of exposure to asphalt fumes. This conclusion is also supported by the results of the logistic regression analysis (Table 4).

After adjusting for the important confounders, a significant association was found between exposure to asphalt fumes and prevalence of respiratory symptoms; exposure to asphalt fumes increased the prevalence of cough and wheezing by 6.9 and 18.1 fold, respectively.

These observations are in agreement with the results of the Randem's study on 64 asphalt workers.4 They showed that the risk of wheezing increased by 2.6 times as a result of exposure to asphalt fumes.27 Similarly, findings from other cross-sectional studies have shown a significant increase in the prevalence of respiratory symptoms following exposure to asphalt fumes.28-32

Measurements of lung capacities before and after the exposure were the basis of assessment for acute and chronic effects of exposure to asphalt fumes. To differentiate the acute and chronic effects of exposure to asphalt fumes in this study, pulmonary function parameters were measured at the beginning (after a 72-hour exposure-free period) and at the end of shift. The average FEV1/VC and FEV1/FVC ratios in the exposed group (pre-exposure) were significantly lower than those of the comparison group that showed the chronic effect of the exposure. Furthermore, cross-shift changes in all measured pulmonary function parameters reflected the acute effect of the exposure. These changes could not be attributed to the circadian rhythms considering the circadian rhythms lead to changes in opposite direction.33 The observed changes could be attributed to asphalt fumes-induced acute partially reversible decrements in pulmonary function tests. This conclusion is further confirmed by the results of multiple linear regression analysis (Table 5) and is consistent with the findings of other studies.1,4,5,8,10,14,15

The findings of the current study are not consistent with the findings of some other studies.12,18,19 While the exact reasons for these discrepancies are not clear, factors such as difference in the air concentration of asphalt fumes in different studies, asphalt temperature, the season when the study was conducted, air velocity,26 direction of wind,34 the method asphalt was scattered (manual or mechanical), the emission model of asphalt vapors and fumes, study sample size, how confounding variables were controlled, type of statistical analysis, workload, and the personal protective equipment used may explain in part, this issue.

The nature of respiratory disorder associated with occupational exposure to asphalt fumes is consistent with the pattern of obstructive lung disease. In patients with obstructive lung disease, FVC is either normal or increased. The hallmark of this type of disorder is a significant reduction in FEV1, hence, significant decrease in FEV1/FVC.35 This conclusion is in keeping with the results of some other studies where an increased incidence of airway obstruction among asphalt workers has been reported.4,36 Other cross-sectional studies have also reported that chronic bronchitis and respiratory symptoms are associated with exposure to asphalt fumes.28,30-32 However, many confounding variables such as smoking, were poorly controlled and the study power was mostly poor.

The significant association between exposure to sub-TLV levels of asphalt fumes and increased prevalence of respiratory symptoms with diminished pulmonary function indices might be interpreted with certain level of skepticism. However, it has to be reiterated that these findings are not only found in this study. Other studies have already shown that exposure of dentists to sub-TLV levels of mercury was associated with sub-clinical symptoms of intoxication.37 Additionally, it is worth noting that the study was conducted in winter when, due to cold temperature, the concentrations of asphalt fumes were minimal.34 Therefore, it would plausible to assume that subjects in summer and hot seasons are exposed to higher concentrations of asphalt fumes,34 and thus, their cumulative exposure is likely to exceed the existing TLV values.

This study had some limitations. Cross-sectional studies cannot establish any cause and effect relationship. For this inherent limitation, one might argue that the significant increase in the prevalence of respiratory symptoms and deteriorated lung function in asphalt workers cannot necessarily be attributed to the exposure to asphalt fumes. While true, a few lines of evidence indicate that these are very likely to be the direct consequences of exposure to asphalt fumes: 1) The exposed group had no medical or family history of chronic lung disease, injuries and surgeries on the chest during the course of their employment, or before it. 2) The exposed workers did not have any exposure to other chemicals causing respiratory disorders. 3) While the pulmonary function in the exposed group partially improved after the exposure ceased, they were still significantly different from those in comparison subjects. 4) There were no significant differences in the number of smokers and smoking intensity in two studied groups. 5) Significant association between exposure to asphalt fumes and reduction in lung function parameters was observed. And, 6) after adjusting for confounding variables, significant associations were observed between exposure to asphalt fumes and respiratory disorders in asphalt workers.

Additional longitudinal studies with larger sample size, sufficient follow-up and longer duration of exposure are clearly required to further substantiate our findings.

In conclusion, we found that occupational exposure to sub-TLV levels of asphalt fumes is associated with increased prevalence of respiratory symptoms as well as acute, partially reversible and chronic irreversible changes in some parameters of pulmonary function.

Acknowledgements

The authors sincerely thank Mr. Vahid Dirin and Mr. Sassan Heydari for their technical assistance.

Conflicts of Interest: None declared.

Funding Source

Funding through the Shiraz University of Medical Sciences, Vice Chancellor for Research Affairs, contract #92-6743, partially supported these investigations.

References

Ulvestad B, Randem BG, Hetland S, et al. Exposure, lung function decline and systemic inflammatory response in asphalt workers. Scand J Work Environ Health 2007;33:114-21.

Roberts F, Kandhal P, Brown E, et al. Hot Mix Asphalt Materials, Mixture Design, and Construction. 2nd ed. Lanham, Maryland: NAPA Education Foundation, 1996.

Othmer K. Asphalt Encyclopedia of Chemical Technology. vol 3. Antibiotics (Phenazines) to Bleaching Agents. John Wiley & Sons, US, 1992, pp 299-302.

Randem BG. Respiratory symptoms and airflow limitation in asphalt workers. Occup Environ Med 2004;61:367-9.

Raulf-Heimsoth M, Pesch B, Kendzia B, et al. Irritative effects of vapours and aerosols of bitumen on the airways assessed by non-invasive methods (suppl 1). Arch Toxicol 2011;85:S41-52.

Karimi H, Mohamadzadeh H. Environmental contamination of a polycyclic aromatic hydrocarbons, (PAHs) in asphalt and bitumen. Fourth Conference and Exhibition of Environmental Engineering, 2010.

Marczynski B, Raulf-Heimsoth M, Preuss R, et al. Assessment of DNA damage in WBCs of workers occupationally exposed to fumes and aerosols of bitumen. Cancer Epidemiol Biomarkers Prevention 2006;15:645-51.

Raulf-Heimsoth M, Pesch B, Schott K, et al. Irritative effects of fumes and aerosols of bitumen on the airways: results of a cross-shift study. Arch Toxicol 2007;81:35-44.

Raulf-Heimsoth M, Pesch B, Spickenheuer A, et al. Assessment of irritative effects of fumes of bitumen on the airways by using non-invasive methods–results of a cross-shift study in mastic asphalt workers. J Occup Environ Hygiene 2007;4(suppl 1):223-7.

Kinnes G, Miller A, Burr G. Health Hazard Evaluation Report: The Sim J. Harris Company, San Diego, California, 1996.

Sylvain D, Miller A. Health Hazard Evaluation Report HETA 94-0219-2620, Walsh Construction Company, Boston, Massachusetts, 1997.

Ma JYC, Barger MW, Kriech AJ, Castranova V. Effects of asphalt fume condensate exposure on acute pulmonary responses. Arch Toxicol 2000;74:452-9.

Hansen ES. Cancer mortality in the asphalt industry: a ten year follow up of an occupational cohort. British J Indust Med 1989;46:582-5.

Ekstrom L-G, Kriech A, Bowen C, et al. International studies to compare methods for personal sampling of bitumen fumes. J Environ Monitoring 2001;3:439-45.

Norseth T, Waage J, Dale I. Acute effects and exposure to organic compounds in road maintenance workers exposed to asphalt. Am J Industrial Med 1991;20:737-44.

Maizlish N, Beaumont J, Singleton J. Mortality among California highway workers. Am J Industrial Med 1988;13:363-79.

Hansen ES. Mortality of mastic asphalt workers. Scand J Work Environ Health 1991;17:20-4.

Gamble JF, Nicolich MJ, Barone NJ, Vincent WJ. Exposure-response of asphalt fumes with changes in pulmonary function and symptoms. Scand J work Environ Health 1999;25:186-206.

US Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, Butler MA, Burr G, Dankovic D, et al. Hazard review: health effects of occupational exposure to asphalt, 2000.

Goodyear MD, Krleza-Jeric K, Lemmens T. The declaration of Helsinki. BMJ 2007;335:624-5.

Ferris BG. Epidemiology Standardization Project (American Thoracic Society). Am Rev Respir Dis 1978;118:1-120.

Jahangiri M, Neghab M, Nasiri G, et al. Respiratory disorders associated with occupational inhalational exposure to bioaerosols among wastewater treatment workers of petrochemical complexes. Int J Occup Environ Med 2015;6:41-9.

Neghab M, Mohraz MH, Hassanzadeh J. Symptoms of respiratory disease and lung functional impairment associated with occupational inhalation exposure to carbon black dust. J Occup Health 2011;53:432-8.

Neghab M, Choobineh A. Work-related respiratory symptoms and ventilatory disorders among employees of a cement industry in Shiraz, Iran. J Occup Health 2007;49:273.

Eller P, Cassinelli M. Method 5042, Benzene-soluble fraction and total particulate (asphalt fume). NIOSH Cincinnati, Ohio; 1998, pp 98-119.

ACGIH. TLVs and BEIs: Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Cincinnati, Ohio ACGIH, 2012.

Tompa A, Jakab MG, Biró A, et al. Health, genotoxicology, and immune status of road pavers in Hungary. J Occup Environ Hygiene 2007;4:154-62.

Baylor C, Weaver N. A health survey of petroleum asphalt workers. Arch Environ Health 1968;17:210-4.

Burr G, Tepper A, Feng A, et al. NIOSH Healht Hazard Evaluation Report: HETA 2001-0536-2864 Crumb-Rubber Modified Asphalt Paving: Occupational Exposures and Acute Health Effects. National Institute for Occupational Safety and Health, Cincinnati Ohio, 2001:42.

Krasniuk E, Cherniuk V, Rossinskaia L, Chuĭ T. [The effect of manufacturing factors in asphalt-bitumen plants on the health of the workers]. Lik Sprava. 2000:106-12. [in Russian]

Maintz G, Schneider W, Maczek P. [Chronic obstructive airway diseases caused by long-term occupational exposure to asphalt pyrolysis products]. Z Erkr Atmungsorgane 1987;168:71-6. [in German]

Nyqvist B. [Respiratory tract symptoms in asphalt workers--another occupational bronchitis?] Lakartidningen 1978;75:1173-5. [in Swedish]

Guberan E, Williams MK, Walford J, Smith MM. Circadian variation of F.E.V. in shift workers. British J Indust Med 1969;26:121-5.

Spickenheuer A, Rühl R, Höber D, et al. Levels and determinants of exposure to vapours and aerosols of bitumen. Arch Toxicol 2011;85:21-8.

Kumar V CR, Robbin S. Basic Pathology. 6th ed. Philadelphia, WB Saunders Company, 1997, pp 393-425.

Surange N, Hoyle J. S4 Occupational asthma: is this the cause of excess respiratory symptoms and COPD described in bitumen exposed workers? Thorax 2011;66:A5-A6.

Rachel's Environment & Health News
#559 - Childhood Cancer and Pollution
August 13, 1997

A new peer-reviewed study in England shows that children have an increased danger of getting cancer if they live within three to five kilometers (2 to 3 miles) of certain kinds of industrial facilities.[1] The study, by E.G. Knox and E.A. Gilman, finds that the danger is greatest within a few hundred yards of pollution sources and tapers off with distance. The incidence of childhood cancers per 100,000 children in England and the U.S. has been rising steadily for at least 20 years.

The new study examined data for 22,458 children who died of leukemia (cancer of the blood-forming cells) or of other cancers during the years 1953 to 1980 in England. The study looked at home address at time of birth and home address at time of death, then measured the physical distance from these addresses to nearby industrial facilities.

Excesses of leukemias and other cancers among children were found near the following kinds of industries:

** oil refineries, major oil storage installations, railside oil distribution terminals, and factories making bitumen (a British term for asphalt, crude petroleum and tar).

** automobile factories, auto body construction factories, and auto body repair shops;

** major users of petroleum products including paint sprayers, fiber glass fabricators, paint and varnish makers, manufacturers of solvents, plastics and detergents, and galvanizers (zinc metal platers).

** users of kilns and furnaces, including steel mills, power plants, cement manufacturers, brick makers, crematoria, and foundries for iron and steel, aluminum, and zinc.

** airfields, railways, highways, and harbors.

This study was also interesting for what it did NOT find:

** Rubber manufacturers showed slight increases in childhood cancers nearby, but tire manufacturing plants did not. Likewise, brake manufacturing showed no excessive childhood cancers nearby.

** Despite the use of solvent-based cleaning, electroplating plants showed no childhood cancer increases nearby.

** Twenty-two factories making halogenated hydrocarbons (chlorinated and fluorinated) had no apparent effect but 32 other solvent manufacturers showed cancer effects up to 5 kilometers (3 miles) away.

** Metal casting (aluminum and zinc), metal forming, and welding probably account for the effects seen near automobile manufacturing plants, the authors say. However, casting and refining of lead showed no childhood cancer effects. The manufacture of automobile batteries, on the other hand, exhibited strong effects. The authors speculate that it may be the manufacture of battery casings (plastics forming, and use of solvents) that create the childhood cancer effect, rather than the lead itself.

** Other industries that did not seem to be associated with childhood cancers included agricultural fertilizer rail terminals; TV transmitters; cake and biscuit bakers; dry cell battery manufacturers; magnetic tape makers; nuclear power plants; PVC manufacturers; and the makers of wood preservatives.

** Benzene manufacturing plants were not associated with nearby clusters of childhood cancers. The known leukemia hazard from benzene may have led to special containment measures.

The findings for leukemias and for other cancers were the same. Among children who had changed addresses between birth and death, the cancer hazard could only be seen near the birth address, implying that exposure to pollutants shortly before or after birth caused the cancers.

Knox and Gilman, the authors of this study, have spent several years developing analytic techniques for identifying small-scale cancer clusters, usually cancers occurring within 150 to 300 meters (roughly 150 to 300 yards) of each other.[2] The authors say they are sure their techniques can now identify cancer clusters at the neighborhood level. "First, our recent analyses have effectively dispelled caveats about the reality of short range case clustering and the existence of geographically localised hazards is not now in doubt. Proximity studies are no longer concerned with this issue and can be directed solely at asking what those hazards might be," they say.[1]

This latest study takes these techniques the next step and links the cancer clusters to nearby sources of pollution, particularly those involving large quantities of petroleum.

The weakness of this latest study, the authors say, is that it cannot rule out the possibility that there are excessively large numbers of children living near industrialized facilities, which could create the false impression of high cancer rates. The authors examine this question as best they can, and they show that, in general, there are few residences within short distances (a few hundred yards) of major factories because associated facilities (roads, parking lots, garages, etc.) compete for space with residential buildings.

The authors conclude that childhood cancers cluster around two general kinds of facilities:

** producers, refiners, distributors, and industrial users of petroleum fuels and volatile petroleum products; and

** manufacturing processes using high temperature furnaces, kilns, and combustion chambers.

Some operations, notably internal combustion engines and oil fired furnaces, meet both criteria.

The authors of the study say there may be three mechanisms by which childhood cancers are caused:

** Gases and volatile organic compounds reaching children or their pregnant mothers directly;

** Parents' germ cells being harmed during occupational exposures, giving rise to children who are predisposed to cancers;

** Occupational contamination carried home on clothing, skin, or breath.

Of the three mechanisms, the authors say they believe direct exposure of children or their pregnant mothers is the most likely.

The authors say their study may have missed many local sources of petroleum exposure of children, such as domestic and commercial heating systems, oil storage bunkers, oil delivery spills, small machine shops, bus stations, school or hospital chimneys, municipal incinerators, gasoline stations, etc.

Childhood cancers could be caused by at least 3 mechanisms:

** Pollutants damaging the inherited genetic material (DNA) in cells;

** Pollutants damaging the immune system which would otherwise prevent cancer cells from surviving;

** Pollutants damaging mechanisms of cell division. (Cancer is

uncontrolled cell division.)

These latest findings, that childhood cancers are clustered near industrial facilities, contradict the official view of childhood cancer, at least in the U.S. The National Cancer Institute (NCI) wrote in 1993, "Time trends in childhood cancer are not likely to be affected by environmental agents because very few are known that cause cancer within the pediatric age-span, and exposures have been rare or limited." And: "Clusters of childhood cancer occur very often by chance and almost never because of environmental agents."[3] Nevertheless, the NCI does say that children exposed to radiation (as at Hiroshima and Nagasaki) can develop cancers. Exposure to benzene could cause childhood leukemia, says NCI, because benzene affects chromosomes the same way radiation does. The children of mothers treated with diethylstilbestrol (DES) --a drug given to women in the 1950s to prevent miscarriage --can develop childhood cancers, NCI acknowledges.

NCI reports that the incidence (per 100,000 children) of many childhood cancers have increased steadily during the period 1973-1990. All childhood cancers combined have increased at the rate of 0.9% per year (0.9% per year among whites, and 1.0% per year among African- Americans). Cancer of the brain and central nervous system have increased at 1.8% per year. Leukemias have increased at 1.8% per year. Non-Hodgkin's lymphomas have increased at 1.4% per year. Kidney cancer has increased at 1% per year. However, thanks to surgery, radiation treatments, and chemotherapy, death rates for all these childhood cancers have declined steadily since 1973 at an average rate of 2.9% per year even as the incidence rates have increased.[3]

U.S. environmental officials discourage the kind of study reported here. Each year U.S. Environmental Protection Agency (EPA) collects data on toxic releases as self-reported by industrial polluters, thus creating the annual Toxics Release Inventory, or TRI database, which is authorized by federal law. However, EPA has never assigned any staff to check the quality of the self-reported data, thus making any studies based on the TRI data suspect. Furthermore, when John R. Stockwell, a physician employed by the U.S. Environmental Protection Agency (EPA), developed a technique for linking data from the TRI database with disease rates near pollution sources in Chattanooga, Tennessee, EPA officials immediately tried to fire Stockwell. (See REHW #366, #392.) Because of citizen protests, Stockwell managed to keep his job, but he has not undertaken any similar studies since then, and neither has anyone else within EPA. EPA chief Carol Browner has issued a memo specifically ordering EPA staff to "stay away from linking human health effects and the TRI data." (REHW #392)

Another EPA official who tried to link industrial toxic releases to human health has also found himself in serious trouble. Brian Holtzclaw, an environmental engineer employed by EPA but "on loan" to the state of Kentucky, urged the study of massive toxic releases from an Ashland Oil refinery to see if they correlated with disease rates in neighboring communities. He tried to bring in John Stockwell to study Ashland's toxic discharges, and he himself released some pollution data to local citizens. Holtzclaw was immediately terminated from his Kentucky projects and reassigned to Atlanta, Georgia. Holtzclaw fought the reassignment. Hundreds of environmental groups and individuals all across the country have signed letters and petitions on Holtzclaw's behalf. After a legal battle, EPA --without admitting any wrongdoing -- settled with Holtzclaw for $20,000 and a written promise that he could continue to work on environmental justice issues. However, Holtzclaw's court battle against the U.S. Department of Labor and the state of Kentucky continues. He wants his job back in Kentucky and he wants his court costs reimbursed.[4]

The Stockwell and Holtzclaw cases send an unmistakable message from EPA chief Carol Browner to all EPA employees: Beware. The relationship of pollution to human disease is a forbidden topic of study.

--Peter Montague (National Writers Union, UAW Local 1981/AFL-CIO)

=====

[1] E.G. Knox and E.A. Gilman, "Hazard proximities of childhood cancers in Great Britain from 1953-80," JOURNAL OF EPIDEMIOLOGY AND COMMUNITY HEALTH Vol. 51 (1997), pgs. 151-159.

[2] See E.G. Knox, "Spatial clustering of childhood cancers in Great Britain," JOURNAL OF EPIDEMIOLOGY AND COMMUNITY HEALTH Vol. 50, No. 3 (June 1996), pgs. 313-319. And: E.G. Knox, "Leukaemia clusters in childhood: geographical analysis in Britain," JOURNAL OF EPIDEMIOLOGY AND COMMUNITY HEALTH Vol. 48, No. 4 (August 1994), pgs. 369-376. And: E.G. Knox, "Leukaemia clusters in Great Britain. 1. Space-Time interactions," JOURNAL OF EPIDEMIOLOGY AND COMMUNITY HEALTH Vol. 46, No. 6 (December 1992), pgs. 566-572. And: E.G. Knox, "Leukaemia clusters in Great Britain. 2. Geographical concentrations," JOURNAL OF EPIDEMIOLOGY AND COMMUNITY HEALTH Vol. 46, No. 6 (December 1992), pgs. 573-576. And: E.A. Gilman, "Childhood Cancers: space-time distribution in Britain," JOURNAL OF EPIDEMIOLOGY AND COMMUNITY HEALTH Vol. 49, No. 2 (April 1995), pgs. 158-163.

[3] Barry A. Miller, and others, editors, SEER CANCER STATISTICS REVIEW 1973-1990 [NIH Publication No. 93-2789] (Bethesda, Maryland, 1993), pgs. XXVII.1 to XXVII.15.

[4] Scott Learn, "Project director says EPA won't let doctor participate," LEXINGTON [KENTUCKY] HERALD-LEADER March 20, 1994, pg. A15. And see: Andrew Melnykovych, "EPA to Pay $20,000 settlement to employee," THE [LOUISVILLE, KENTUCKY] COURIER JOURNAL November 3, 1995, pg. 1. And see: Southern Organizing Committee for Economic and Social Justice (SOC), "Hundreds of Citizen Groups Call on Department of Labor to Uphold Rights of Environmental Whistleblower," press release dated October 11, 1996; for further information, contact SOC at (502) 776- 7874, or (404) 755-2855, or Mr. Holtzclaw himself at (404) 562-8868.

Descriptor terms: oil industry; petroleum; cancer; childhood cancers; leukemia; brain cancer; kidney cancer; studies; england; e.g. knox; cancer clusters; automobile manufacture; automobile repair; paint; fiber glass; solvents; plastics; detergents; metal plating and finishing; boilers and industrial furnaces; bifs; crematoria; iron; steel; zinc; aluminum; cement kilns; airports; railroads harbors; rubber manufacturers; metal casting; welding; automobile batteries; emf; benzene; pvc; high-temperature combustion; diesel exhaust; internal combustion engines;

Rachel's Environment & Health News is a publication of the Environmental Research Foundation, P.O. Box 160, New Brunswick, NJ 08903-0160; Phone: (732) 828-9995; Fax (732) 791-4603; E-mail: erf@rachel.org; http://www.rachel.org. Unless otherwise indicated, Rachel's is written by Peter Montague.

www.BREDL.org PO BOX 88 Glendale Springs, North Carolina 28629 BREDL@skybest.com (336) 982-2691 office (336) 977-0852 cell

ASPHALT PLANTS
CONTAMINANTS OF CONCERN:

An overview of 7 toxic substances released from asphalt processing facilities and their known effects on human health

Asphalt plants are sources of air pollution that may emit significant levels of both particulate matter and gaseous volatile organic compounds (VOCs). These pollutants are considered to be dangerous to human health. Some VOCs are also suspected carcinogens or cancer-causing agents (*Fact Sheet: Information Regarding Asphalt Concrete Plants,* number 5, November 1996, Ohio EPA, Division of Air Pollution Control Small Business Assistance Program).

No two asphalts are chemically alike. The chemical makeup of asphalt depends on the chemical content of the original crude petroleum from which it is made. Other manufacturing methods which alter the chemical makeup of asphalt include asphalt cement additives, higher operating temperatures, and the use of recycled asphalt paving cause increases in toxic emissions (Letter to Dr. Ernest Fuller, Division of Air Quality, Raleigh Regional Office, from Louis Zeller, BREDL, re: Tar Heel Paving DRAFT permit #08977R00, March 12, 2001).

The following are examples of seven pollutants typically found at various levels in emissions from asphalt plants - hydrogen sulfide, benzene, chromium, formaldehyde, polycyclic aromatic hydrocarbons (PAHS), cadmium and arsenic – and the known effects of these substances on human health:

Hydrogen sulfide (H_2S). Hydrogen sulfide is a poisonous, colorless gas that is associated with the characteristic smell of rotten eggs. Exposure tends to be a problem in communities located near certain types of industrial sites that release hydrogen sulfide. People who live near an industrial facility that emits hydrogen sulfide may be exposed to higher levels of hydrogen sulfide. Exposure to hydrogen sulfide occurs from breathing contaminated air or drinking contaminated water. Hydrogen sulfide remains in the air for about 18 hrs. after which it changes into sulfur dioxide and sulfuric acid. Hydrogen sulfide may also be released as a liquid waste from an industrial facility. It is not known whether children are more sensitive to hydrogen sulfide than adults nor is it known if hydrogen sulfide causes birth defects (*ToxFAQs for Hydrogen Sulfide*, Agency for Toxic Substances and Disease Registry, July 2006, CAS #7783-06-04.)

Exposures to high concentrations of hydrogen sulfide may result in respiratory distress, pulmonary edema, nervous system depression, neurobehavioral effects, tissue hypoxia, cardiovascular effects, unconsciousness and death. Exposure to lower concentrations of hydrogen sulfide can result in less severe neurological and respiratory effects such as incoordination, loss of smell, nasal symptoms, sore throat, cough, and dyspnea. Some evidence suggests that people with asthma may be overly sensitive to hydrogen sulfide, and impaired function has been observed in people with asthma who were exposed to low levels of hydrogen sulfide.

One community exposure study found an increased prevalence of eye irritations in residents exposed to low levels of hydrogen sulfide. Numerous case reports suggest that high exposures to respiratory arrest and pulmonary edema can occur after a brief exposure to hydrogen sulfide. Although most people recover after exposure to hydrogen sulfide many individuals report permanent or persistent neurological effects including headache, poor concentration ability and attention span, impaired short memory and motor function (*Toxicological Profile for Hydrogen Sulfide*, US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, 2006).

Benzene. Benzene, also known as benzol, is a colorless liquid with a sweet odor. Benzene is a known carcinogen or cancer-causing agent. Benzene enters the body through the lungs, gastrointestinal tract, and across the skin. Brief exposure (5-10 minutes) to very high levels of benzene in air can result in death. Lower levels of exposure can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness. Continuous exposure to benzene can lead to anemia and excessive bleeding, and may be harmful to the immune system by increasing the chance for infection and perhaps lowering the body's defense against cancer.

Exposure to benzene has been associated with development of a particular type of leukemia called acute myeloid leukemia (AML). The Department of Health and Human Services, the International Agency for Cancer Research and the EPA has determined that benzene causes cancer.

Exposure to benzene may be harmful to human reproductive organs. Benzene can pass from the mother's blood to a fetus, but it is not known what effects exposure to benzene might have on the developing fetus in pregnant women or on fertility in men. However, studies with pregnant animals show that breathing benzene has harmful effects on the developing fetus. These effects include low birth weight, delayed bone formation, and bone marrow damage.

Children can be affected by benzene exposure in the same ways as adults, and is not known if children are more susceptible to benzene poisoning than adults (*Public Health Statement for Benzene, Draft for Public Comment,* Agency for Toxic Substances and Disease Registry**,** September 2005, CAS#: 71-43-2).

Chromium. Chromium is a naturally occurring element found in rocks, animals, plants, soil, and in volcanic dust and gases. Chromium cannot be tasted and has no odor. Chromium is present in the environment in several different forms. The most common forms are chromium(0), chromium(III), and chromium(VI), also known as hexavalent chromium.

Chromium(VI) and chromium(0) are usually produced by industrial processes. Breathing high levels of chromium(VI) can cause irritation to the nose, such as runny nose, nosebleeds, and ulcers and holes in the nasal septum. Chromium(VI) at high levels can damage the nose and can cause cancer. Ingesting large amounts of chromium(VI) can cause stomach upsets and ulcers, convulsions, kidney and liver damage, and even death. Skin contact with certain chromium(VI) compounds can cause skin ulcers. Some people are extremely sensitive to chromium(VI) or chromium(III). Allergic reactions consisting of severe redness and swelling of the skin have been noted (*ToxFAQs for Chromium*, Agency for Toxic Substances and Disease Registry**,** February 2001, CAS#: 7440-47-3).

Formaldehyde. Formaldehyde is a nearly colorless gas with a pungent, irritating odor even at very low concentrations (below 1 ppm). Formaldehyde is a potent sensitizer and a probable human carcinogen or cancer-causing agent. Formaldehyde is an eye, skin, and respiratory tract irritant; inhalation of vapors can produce narrowing of the bronchi and accumulation of fluid in the lungs.

Children may be more susceptible than adults to the respiratory effects of formaldehyde. Even fairly low concentrations of formaldehyde can produce rapid onset of nose and throat irritation, causing cough, chest pain, shortness of breath, and wheezing. Higher exposures can cause significant inflammation of the lower respiratory tract, resulting in swelling of the throat, inflammation of the windpipe and bronchi, narrowing of the bronchi, inflammation of the lungs, and accumulation of fluid in the lungs (*Medical Management Guidelines for Formaldehyde,* Agency for Toxic Substances and Disease Registry, CAS#: 50-00-0, updated 11/02/06).

Polycyclic aromatic hydrocarbons (PAHS). Polycyclic aromatic hydrocarbons (PAHs) are a group of over 100 different chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances and found in coal tar, crude oil, creosote, and roofing tar. The Department of Health and Human Services has determined that some PAHs may reasonably be expected to cause cancer. Some people who have breathed or touched mixtures of PAHs and other chemicals for long periods of time have developed cancer.

Certain PAHs have caused cancer in laboratory animals when they breathed air containing them (lung cancer), ingested them in food (stomach cancer) or had them applied to their skin (skin cancer). PAHs are found in air attached to dust particles, and can enter water through discharges can enter water from industrial and wastewater treatment plants where they can move through soil to contaminate groundwater. The PAH contents of plants and animals may be much higher than PAH contents of soil or water in which they live (*ToxFAQs for Polycyclic Aromatic Hydrocarbons (PAHs)*, Agency for Toxic Substances and Disease Registry, September 1996).

Cadmium. Cadmium is an element that occurs naturally in the earth's crust. Pure cadmium is a soft, silver-white metal that attaches to small particles in the air. People who live near hazardous waste sites or factories that release cadmium into the air have the potential for exposure to cadmium in air.

Breathing air with very high levels of cadmium can severely damage the lungs and may cause death. Breathing air with lower levels of cadmium over long periods of time (for years) may result kidney disease, lung damage and fragile bones. Data on human exposure to cadmium is limited, but studies show that rats that breathed in cadmium developed lung cancer, liver damage and changes in the immune system. Female rats and mice that breathed high levels of cadmium had fewer litters, babies with more birth defects than usual, reduced body weight, babies born with behavioral problems and learning disabilities.

As a conservative approach, and based on the limited human data and the studies in rats, the United States Department of Health and Human Services (DHHS) has determined that cadmium and cadmium compounds may reasonably be anticipated to be carcinogens. The International Agency for Research on Cancer (IARC) has determined that cadmium is carcinogenic to humans. The EPA has determined that cadmium is a probable human carcinogen by inhalation (*Public Health Statement for Cadmium*, Agency for Toxic Substances and Disease Registry, July, 1999, CAS # 1306-19-0).

Arsenic. Arsenic occurs naturally in soil and minerals and it therefore may enter the air, water, and land from wind-blown dust and may get into water from runoff and leaching. Arsenic released from power plants and other combustion processes is usually attached to very small dust particles. These dust particles settle to the ground or are washed out of the air by rain. Arsenic attached to dust may stay in the air for many days and travel long distances. Ultimately, most arsenic ends up in the soil or sediment. Children may also be exposed to arsenic by eating dirt, skin contact with soil or water that contains arsenic, or through inhalation. If you breathe air that contains arsenic dust, particles of arsenic-contaminated dust may settle onto the lining of the lungs.

Inorganic arsenic is usually found in the environment combined with other elements such as oxygen, chlorine, and sulfur. Arsenic combined with carbon and hydrogen is referred to as organic arsenic. Long-term oral exposure to inorganic arsenic can results in a pattern of skin changes called "corns" or "warts" on the palms, soles, and torso that may develop into skin cancer. Swallowing arsenic has also been reported to increase the risk of cancer in the liver, bladder, kidneys, prostate, and lungs. The Department of Health and Human Services (DHHS) has determined that inorganic arsenic is known to cause cancer. The International Agency for Research on Cancer (IARC) has determined that inorganic arsenic is carcinogenic to humans. The EPA also has classified inorganic arsenic as a known human carcinogen.

Breathing high levels of inorganic arsenic will result in a sore throat, irritated lungs and the potential to develop lung cancer. This has been seen mostly in workers exposed to arsenic at smelters, mines, and chemical factories, but also in residents living near smelters and chemical factories. People who live near waste sites with arsenic may have an increased risk of lung cancer as well. High doses of an organic arsenic compound may result in nerve injury, stomach irritation or other effects.

All health effects observed in adults are of potential concern in children. Children may be more susceptible to health effects from inorganic arsenic than adults, and there is evidence that suggests that long-term exposure to arsenic in children may result in lower IQ scores (*Public Health Statement for Arsenic, Draft for Public Comment,* Agency for Toxic Substances and Disease Registry, September 2005, CAS#: 7440-38-2).

Asphalt plant would expose children to toxins

By CATHERINE CANTO
Published: Thursday, October 7, 2010 at 3:00 a.m.

For children, the consequences of exposure to toxins are serious and life-long. Recent medical studies show toxins make children more prone to asthma, allergies, cancer, heart disease and developmental delays. Petaluma children are already highly exposed to toxins with many of our schools and parks near freeways. In Sonoma County, one in five children ages 5 to 17 have asthma. The average cost of a childhood asthma hospitalization in Sonoma County is $6,148, and our schools lose money each day a child is absent from school. Dutra's asphalt plant will expose 4,560 students, who attend school within two miles of the proposed plant, to toxins. Can we trust Dutra Materials — a corporation with a history of environmental mistakes, fines and bankruptcy — to do what is necessary to keep our children safe and healthy?

The Community Health Forum, sponsored by the Petaluma Health Care District on Sept. 30, was eye-opening in the conflict over the proposed Dutra asphalt plant. For the first time in the history of this conflict, medical experts had a chance to inform the public of the potential health impacts of the proposed project. With the health care district providing a neutral platform, both the Dutra Corporation and community groups opposed to the project were allowed to invite speakers to address the health concerns of the public.

The most compelling segment was presented by a Marin pediatrician and UCSF Medical Center pediatric environmental health specialist, Dr. Alice Brock-Utne. Dr. Brock-Utne discussed the cutting-edge scientific research that explains how and why children and fetuses are disproportionately susceptible to health problems caused by exposure to airborne toxins.

She stated that children are more active, have greater air intake per minute and poorer toxin-clearing capability than adults. She then discussed how particulate matter (referred to as either PM 10 or PM 2.5, depending on size) works its way into the bodies of children to disrupt normal development. Furthermore, toxic chemicals, such as carcinogenic polyaromatic hydrocarbons (PAHs), can attach to the particulate matter and enter the body.

The particulate matter and accompanying toxins weaken the structure and function of childrens' developing lungs and weaken the lungs' ability to self-repair. Further, because these materials penetrate to the cellular level, they also disrupt the normal development of the immune system. They change the way children's DNA is read, making children more prone to asthma and allergies.

One major source of PM 2.5 is diesel emissions from trucks. Recently updated regulations require PM 2.5 be assessed for industrial projects. Unfortunately, despite public outcry, to date both Dutra and Sonoma County have refused to assess the asphalt plant project for PM 2.5 and its health and environmental impacts, particularly for children, infants and fetuses.

Dutra invited Dr. David Weill, a pulmonary critical care specialist from Stanford University, to present at the forum. Dr. Weill said he had reviewed the health risk assessments prepared by the Bay Area Air Quality Management District (BAAQMD) and by Environ Corporation, Dutra's hired consultants. He said that based on those documents, he saw no evidence of health risks from the project. He also said pollution does not cause any health risks, which goes against current medical knowledge. He did not address the fact that PM 2.5 was not included in BAAQMD's report. Dr. Brock-Utne stated the health-risk assessments done do not include the type of developmental immunotoxins she discussed. Consequently, she noted, protecting children from environmental threats requires a preventive, collaborative effort of government, science, clinician, community and family.

It's our duty to provide our children with schools and parks that are safe and healthy. The Board of Supervisors will be voting on this asphalt plant on Oct. 12. It is now up to us to contact the Board of Supervisors and let them know Dutra's plan for an asphalt factory near Shollenberger Park, schools, offices, homes, visitors and wildlife is absolutely, "the wrong plant, in the wrong place."

Outtakes of the community health forum on the potential health impacts of the proposed Dutra asphalt plant will soon be posted on YouTube and the complete event will be broadcast soon on Petaluma Community Access.

(Catherine Canto has a bachelor's of science in nursing and has worked in many health-care settings. She is a Petaluma Parks, Music and Recreation commissioner, a Youth commissioner, and a board member for Moms for Clean Air.)

Indians Appeal Asphalt Permit

Clean air advocates were dismayed but not surprised last month when the county air pollution control district gave its go-ahead to a new asphalt plant just north of Ukiah. Two appeals of the permit have been filed, however, and a hearing date is to be announced. Meanwhile, new questions arose over who had the authority to grant the project a land use permit, the Pinoleville tribal council or the County of Mendocino.

On December 14, the MCAPCD issued a permit to Shamrock Materials for construction and operation of a 300-ton per hour asphalt plant located at Ross Mayfield Jr.'s Arrow Paving site in the Pinoleville Rancheria just north of Ukiah. Mayfield holds use permits for the project from both the County and the tribal council, while Shamrock is apparently providing the funding. There is currently only one other producer of asphalt in the county, and the Forsythe Creek project on Highway 101 has just opened a tremendous new demand for the product.

Citing health concerns over the impacts that the plant would certainly have on rancheria residents, a group of neighbors opposed to the project appealed the air pollution permit. The Sierra Club filed a separate appeal at the same time. The APCD is now considering when to hold a public hearing on the appeals.

The neighbors have organized themselves as United Tribal Members and Non-Tribal Residents of Pinoleville. The core committee is made up of Margene McGee, Arvada Pacheco, and Nancy and Leona Williams, but the group probably also includes at least twenty more who have expressed concerns about the plant since last summer. At an emotional public meeting on the project in November, the United Tribal women asked county air pollution control officer Craig McMillan, a doctor and director of public health for the County, to deny the permit out of concern for the health of rancheria residents.

McMillan said at that time that it was possible, but not likely, that the district would deny the permit based simply on health considerations. He issued the permit last month with no mention of public health concerns.

Air Rules are Inadequate:

McMillan, along with APCD inspector Philip Towle, has said that current regulations oblige them to issue the air pollution permit because the project meets the requirements of air quality law. This is not surprising, because the current laws are not designed to keep clean air in rural areas clean, but only to prevent dirty air in industrial areas from getting dirtier.

This is all about "Prevention of Significant Deterioration" regulations, or PSD for short. Current air quality law projects a maximum level of permissible pollution (National Ambient Air Quality Standards) and seeks to prevent air pollution in local districts from exceeding these limits. These NAAQS standards reflect the level of pollution in industrialized areas when the laws were written. But now, most large cities exceed the standards. The law also sets minimum thresholds, and defines "significant increase in pollution" as any change which is above the thresholds but below the maximums.

In areas such as ours, where the existing air pollution is below the federal standard, the difference between the current level and the federal maximum for each pollutant is called an increment — it is clean air that can legally be sacrificed to development.

The permit issued by Dr. McMillan shows that the plant would operate 11 hours per day for about nine months of the year. Projected air emissions from the plant itself are: 2.4 lb./hr. of PM10, 7.2 lb./hr. of Nitrogen Oxides, 17.7 lb./hr. of Sulfur Oxides, 30.3 lb./hr. of Carbon Monoxide, and 7.7 lb./hr. of volatile organic compounds. Additional emissions of PM10 — tiny dust particles that lodge in the lungs — will result from traffic and wind erosion from gravel piles, exceeding plant emissions. Projected emissions are apparently taken from manufacturer's data.

The permit is conditioned on the applicant paving traffic areas in the existing concrete and gravel yard, installing some sprinklers, and applying lignin to exposed soil areas.

The permit also sets a stack emissions limit of 0.4 grains per dry cubic foot of exhaust gas to be achieved by installation and operation of a 60,000 cubic-foot per minute baghouse as best available control technology. The baghouse and the parts for the asphalt plant are already on the site awaiting hookup, but there is no construction underway at presstime. The stack emissions limit is the federal standard for new asphalt plants.

The APCD considered how the proposed plant would be viewed under state and federal air quality rules. The district decided that the project was not a major source, and that it would not emit more CO, NOx, SOx, PM10 or VOC than the permitted federal threshold levels. The district also said that the plant's projected emissions were less than those that would require a review of existing sources to see if federal ambient air quality standards are being violated.

Further, the district said that the project met a county air pollution rule that the project will not cause a deterioration of existing air quality in excess of 50% of remaining available PSD incre-

DR. CRAIG McMILLAN, County air pollution control officer at November meeting on the project. RJ photo.

CONTINUED, p. 6

Pinoleville Asphalt Permit

CONT. from p.n 8

ments. That is, the Shamrock/Mayfield plant will not use up half of the remaining clean air, so it can be built.

In granting the permit, the district said that the existing gravel and concrete plant on the site generated 90.7 lbs. of PM10 per day. Now, the site will be paved and oiled, presumably eliminating these emissions. And the new asphalt plant stack would emit 26.4 lb. of PM10 per day and that traffic, wind erosion and dumping would cause another 38.3 lb. of PM10 per day even with the pavement and lignin. Thus the district can show a net reduction of 26 lb. of PM10 per day.

Based on this projected reduction of dust emissions, the district said the project did not require a detailed air quality impact analysis.

The district also required the applicant to perform source tests for PM10 and SO2 within sixty days of startup, and to notify the district in writing of the results.

Standing Up for Health:

Working against a tight deadline, a group of Native Americans living in the rancheria raised the required fee of $276 in order to file their appeal to the air pollution permit just before Christmas. Their attorney, Joanne Moore of Willits, said the appeal was based on the need to protect the health of local citizens, especially children and the elderly.

"We asked the district to revoke the permit for lack of any monitoring by the APCD itself," Moore told MCE. "The applicant has already shown a disregard for self-regulation by installing the baghouse on the site before the permit was granted. We also pointed out that the permit failed to support the claim of net reduction in source emissions. Paving isn't enough. And the district also failed to consider cumulative impact of this source together with other sources in the area like Masonite and LP.

"And the district also gave no consideration to the likely increase of truck traffic at the site. We also questioned the allowance of 60 days of operation before requiring the source test. The district has not properly assessed the environmental problems raised by this project," she added. Moore, who is opening a new law practice in the area, is working on this case pro bono. She was previously active with Willits Citizens for a Safe Environment in their successful challenge of the defeated biomass plant in that town.

Use Permit Questioned:

A recent development calls into question the status of the use permit for the project. Last summer, the elected Tribal Counsel of the Pinoleville Indian Community granted Ross Mayfield a use permit for the asphalt plant because he is a low income Native American. Mayfield, who is half Cherokee, is involved in an undisclosed business relationship with Shamrock Materials which is based in Marin County.

Mayfield and the tribal council proudly point to this project as an example of Indian sovereignty. Mayfield now pays taxes on the gravel and concrete operation to the tribe, which is sorely in need of funds to carry on its activities. These tax payments would increase should the asphalt plant go on line, and Mayfield also offers to employ tribal members in the project. As part of the permit, the tribal council will delegate a representative to monitor air emissions at the plant.

The United Tribal Members, however, criticize this use permit as a usurpation of power by the tribal council. "They never asked people in the rancheria if they wanted this plant which will jeopardize the health of the children," charges McGee. She also points out that the tribal council will never shut down the plant, even if it violates air standards, because it is dependent on Mayfield's tax payments.

Recently, however, county counsel has notified the Mendocino County board of supervisors that the County in fact has jurisdiction over that particular site. This arises from a recent US supreme court decision in the Yakima case that county governments prevail in cases where uses within the boundaries of a reservation have been alienated to non-tribal ownership, as in this case. The County gave Mayfield a permit for the asphalt plant back in 1987.

The tribal council and the County have a dormant court case disputing jurisdiction over the entire rancheria. In this suit, the county has stipulated that the tribal council can decide uses on its land if it can come up with a comprehensive plan. To answer this need, the tribal council has hired a consultant to draft a tentative plan, due to be released later this spring. But McGee says no one in the rancheria has ever seen this land use plan. "How can it represent what we want for the rancheria if we don't even know what's in it?" she asks.

But if the county does indeed have legal jurisdiction over the Shamrock/Mayfield site, opponents of the plant may take heart. If the County supervisors would reconsider the 1987 use permit, they may decide to revoke it based on health considerations. This seems much more likely than hoping that the tribal council not to let the plant be built.

WHAT YOU CAN DO:

1) Write to the Mendocino County Air Pollution Appeals Board asking that the Shamrock asphalt plant air pollution permit be revoked for health reasons. County Courthouse, Ukiah, CA 95482.

2) Write to your County supervisor asking that the county reconsider and revoke the Mayfield use permit for the asphalt plant at Pinoleville. County Courthouse, Ukiah, CA 95482.

3) Send a donation to United Tribal Members to cover administrative and legal fees in the case. Make checks to Margene McGee, 680 Pinoleville Drive, Ukiah, CA 95482. Memo the checks: Clean Air.

4) For the date and place of the appeal hearing, stay in touch with the Mendocino Environmental Center, 468-1660.

1 of 1 DOCUMENT

News & Record (Greensboro, NC)

January 24, 2004 Saturday GREENSBORO/HIGH POINT EDITION

GROUP OPPOSES ASPHALT PLANT;
THE PLANT WOULD BE ABOUT HALF A MILE FROM COLFAX ELEMENTARY SCHOOL.

BYLINE: BY JENNIFER FERNANDEZ Staff Writer

SECTION: TRIAD; Pg. B1

LENGTH: 643 words

DATELINE: COLFAX

A strong whiff of perfume is enough to set off Ashley Carter's asthma, which is so severe that she goes to Duke Children's Hospital to see a specialist and undergoes treatments at home twice a day.

So Ashley's mother, Beth Carter, worries about what will happen to her 8-year-old if the smell of hot asphalt churning in a plant half a mile from Ashley's school comes wafting in.

"No one would want a small child around that, whether they have a problem or not," said Carter, 35, an office manager for a small construction management firm.

Carter is one of several Colfax Elementary School parents who are concerned that a proposed asphalt plant just off West Market Street might get approved by the Guilford County Planning Board.

Parents and community members, who have formed "Neighbors For a Cleaner Colfax Tomorrow," will meet Tuesday at Shady Grove Wesleyan Church to prepare for the Feb. 11 planning board meeting. The board will consider at that meeting a proposal by Vecellio & Grogan, based in Beckley, W.Va., to build a $7 million asphalt plant on 17 acres next to Carolina Steel Corp. across from Colfax Elementary.

Vecellio & Grogan officials could not be reached for comment. In the past, they have said the Colfax plant might one day produce enough asphalt to fill hundreds of dump trucks per day. They've also said there will be few emissions and that asphalt plants are much cleaner today than they used to be.

Vecellio & Grogan runs five asphalt plants in Florida. Three of them are in Florida's Department of Environmental Protection Southeast Division, which has only one violation on record for the company, said division spokesman Willie Puz. In 2000, the company was fined for having too much sulfur in its fuel at its West Palm Beach plant, he said.

The company has won several awards for its practices, including one from a Florida environmental group for its recycling efforts and several from an industry association.

The Asphalt Education Partnership states on its Web site beyondRoads.com that there is "no scientific evidence that the very low levels of emissions from an asphalt facility pose health risks to humans."

GROUP OPPOSES ASPHALT PLANT;THE PLANT WOULD BE ABOUT HALF A MILE FROM COLFAX ELEMENTARY SCHOOL. News & Record (Greensboro, NC) January 24, 2004 Saturday GREENSBORO/HIGH POINT EDITION

The local health department and regional EPA office in Winston-Salem did not have information on health risks associated with asphalt plants.

"I don't care what they say," said Bill Shular, whose home is 1,000 feet from the proposed plant. "The picture they print and what they talk about is not what it's going to be."

Shular, who is part of an organized opposition to the plant, said he visited other asphalt plants in the area. He talked to several residents and said they had the same complaints: smoke, dust, fumes from the trucks, heavy traffic, road deterioration, and health problems, such as sinus conditions.

Parents have told him they will leave if the Colfax plant is approved, Shular said.

Colfax Elementary's PTA has been calling parents to advise them of the situation.

"From what we've heard, we're going to smell this," said Courtney Spencer, 35, co-president of the PTA and the mother of a first-grader at the school. She also has a 4-year-old who will soon be attending Colfax. Spencer said she doesn't want her children exposed to the smells, dust and unseen pollutants of an asphalt plant.

County planners have recommended the board reject the plant based on concerns over the proposed entrance, which would have heavy trucks traveling small side roads to access the plant.

Spencer said that parents are optimistic because of the recommendation, but are nevertheless "afraid to take it for granted." That is why they are continuing to gather support against the plant.

"I think there's a lot of concern that it might slip through if they come up with an alternate entrance," she said.

Contact Jennifer Fernandez at 373-7064 or jfernandez@news-record.com

LOAD-DATE: January 27, 2004

LANGUAGE: ENGLISH

GRAPHIC: Map Graphic, TIM RICKARD; News & Record; PROPOSED ASPHALT PLANT

ASPHALT PLANTS

January 15, 2001

Mountain Air Action Project
Asphalt Plant Campaign Report

This report gives an update on BREDL's campaign to reduce toxic pollution from asphalt plants in western North Carolina. It includes information about six communities which oppose existing or proposed asphalt plants.

After defeating an attempt by industry to eliminate North Carolina's regulations for toxic air pollution control in 1997, BREDL continued to organize communities with actual or proposed toxic air pollution industries and to oppose the deregulation of air pollution. Major accomplishments include:

1-North Carolina signed an agreement with Tennessee to protect air quality in the Great Smoky Mountain National Park, Linville Gorge and other Class I wilderness areas.

2-North Carolina developed a new and better analysis of fugitive toxic air emissions

3-We forced North Carolina to expand the Toxic Air Pollutant program to all operating and proposed asphalt plants

The path to clean air is includes program and policy changes at the state and national levels. But without an actual reduction of pollution in communities where industrial plants are located, we cannot claim success. BREDL community organizers spend 90% of their time working with groups of citizens, most of them new to the environmental movement.

In 1997 BREDL organized local opposition to an asphalt plant proposal in an unzoned community in Watauga County. The permit was denied on the basis of public health protection, a first in North Carolina, and resulted in an eight month statewide moratorium on all new asphalt plant permits.

An asphalt plant proposed for Flat Creek in an unzoned area of Buncombe County was also defeated in 1997. North Buncombe Association of Concerned Citizens and BREDL joined forces to uncover and publicize the track record of the asphalt company. The campaign raised serious doubts about the company's ability to operate a plant within state regulations and the proposal was dropped.

In 1998 we organized four new groups in Polk, Rutherford, Macon, and Ashe counties in western North Carolina. These rural groups which have become BREDL chapters include Foothills Action Committee for the Environment (FACE), Rutherford Environmentalists Against Pollution (REAP), Neighbors Against the Cullasaja Asphalt Plant (NACAP), and Ashe Citizens Against Pollution (ACAP). BREDL staff services included campaign strategy sessions, technical assistance with permits, media training, assistance with fiscal management, and limitation of liability through joint incorporation.

In 1998 our accomplishments included getting North Carolina to develop a new and better analysis of fugitive toxic air emissions and to expand the Toxic Air Pollutant program to all operating and proposed asphalt plants. In July 1998 BREDL challenged the state with a request for a ruling on toxic emissions from asphalt plants. In September BREDL staffer Lou Zeller presented new information about asphalt plant emissions to the state Environmental Management Commission. In 1999 the Blue Ridge Environmental Defense League received the Governor's Conservation Achievement Award for Air Conservationist of the Year.

In 1998 we began to do our own SCREEN3 computer modeling of stationary air pollution sources. This is the principle method used by the NC Division of Air Quality's permitting experts to estimate toxic air pollution emissions. We are expanding this capability to include ISCST3 and more advanced methods. We are compiling background data and computer models which will enable BREDL and our community groups to assess the effects of mobile and stationary air pollution sources on ground level ozone, acid rain, and toxic air pollution. With these tools at the disposal of community activists our campaigns will acquire a new technical sophistication and be better able to combat air pollution of all types.

In 1999 we mounted three simultaneous asphalt plant permit challenges, all in the mountain region. Also, working with a national network of activists to oversee asphalt plant testing by the EPA, we monitored asphalt plant emissions tests in California and Massachusetts. These tests form the basis for EPA guidance for estimation of fugitive, or ground-level, emissions of volatile organic compounds and other asphalt plant toxins.

In 2000 we experienced victories and setbacks. A third asphalt plant proposal was defeated in Watauga County. Citizens remain vigilant in Ashe County since the defeat of an asphalt plant proposal near Jefferson. Residents along the Cullasaja River won a stunning reversal of a judge's decision which upheld an asphalt plant permit, but the company has re-applied for a new permit.

In 2001 we continue our drive for reduction of asphalt plant pollution. Recently, three asphalt plant operators have submitted requests for new pollution permits in western NC, and three new grassroots community organizations have been formed. We have active asphalt plant campaigns in five communities.

Pineola

In Avery County, Christmas Tree Capital of NC, citizens of Pineola tolerated asphalt plant pollution for years. Residents called us complaining of nausea and asthma associated with plant operation. After joining BREDL in early 1997, Pineola Concerned Citizens waged a campaign to stop the poisoning of their village. Our efforts were rewarded on July 6, 1998 when the Avery County Commission voted unanimously for the asphalt plant's immediate closure. But the NC Division of Air Quality has not responded to the county's requests.

However, Pineola Concerned Citizens made significant progress in the campaign to build public opposition to Maymead Materials' asphalt plant pollution in Pineola. In 1998 the plant operated at less than 10% of permitted annual capacity, a reduction of one-third from the previous year. Residents attribute this drop to negative publicity about the plant and heightened environmental

concerns at the county level.

In 1999 Maymead's permit expired, requiring the company to submit a permit application for continued operations. BREDL and Pineola Concerned Citizens presented a report on the new draft permit to the Avery County Commission on April 5th. The report contained an independent SCREEN3 computer model run by BREDL. Our results indicated the plant would not be in compliance with state regulations if permitted at the present location on Highway 181. At issue were benzene and formaldehyde emissions, pollution loopholes, and public health impacts. In response to Pineola Concerned Citizens' request, the Avery County Commission passed a resolution citing our findings and opposing the new permit.

At the April 12th public hearing in Newland everyone but Maymead's attorney testified against the permit. We informed officials of our findings and proved that the map used by the state indicated a false location for the plant. Pineola citizens told the state officials of odor problems and asthma which coincide with plant operations and the negative impact on water quality in the Pineola Bog and the nearby Linville River. Dale Thompson lives next to the Maymead plant and is a member of PCC. He testified about the effects of the asphalt plant on the nearby church cemetery saying, "Dead people can't be bothered by pollution, but it even turns the gravestones black." Donna Autrey said, "Little people are not listened to, but big industry and DOT are."

On June 10, 1999 the Division of Air Quality approved the permit but recommended that Maymead Materials work with Avery County officials to find a more suitable location for the Pineola plant. Our plans are to work with the Pineola Concerned Citizens and to use the Pineola example in an appeal to EPA regarding the misuse of the synthetic minor rule under Title V of the Clean Air Act.

In 2000 residents of Pineola continued the campaign by assisting other communities to learn about asphalt plant pollution impacts. Pineola Concerned Citizens are compiling data which document the loss of property values near the plant. Completion of this study in 2001 will complement statements at public hearings which revealed a $400,000 loss in assessed valuation by the Avery County Board of Adjustment. Property value losses were greater at sites closer to the operating asphalt plant in Pineola.

UPDATE: March 8, 2002: The Blue Ridge Environmental Defense released a study showing the adverse impacts on property values and quality of life around an asphalt plant in Pineola, North Carolina.

Boone

In 1997 our successful campaign against an asphalt plant on Roby Green Road, an unzoned residential area outside of Boone, NC resulted in a de facto statewide moratorium on new asphalt plant permits. But the asphalt and highway industries used their considerable clout in Raleigh and in March 1998 the moratorium was lifted. Soon after, the asphalt company re-applied for an identical permit on the same site.

Citizens Against Pollution (CAP), which led the victorious 1997 campaign against Maymead Materials, again rallied people and other resources. Monthly events kept the issue in the public

eye and meetings with state officials pressed them to justify the lifting of the moratorium. For example, CAP held a Clean Air Walk in downtown Boone in May 1998. In June, public hearings in Boone were packed with citizens demanding extra-territorial zoning of the proposed plant site which lies outside the city limits.

In July 1998 BREDL challenged the state with a request for a ruling on toxic emissions from asphalt plants. Citizens pushed for New River to be named the premier American Heritage River, and President Clinton made the designation in person in July. On August 8 Dr. Ravindra Nadkarni, author of two parts of the national Clean Air Act, testified against the air permit at the NC DAQ public hearing in Boone. In September BREDL staffer Lou Zeller presented new information about asphalt plant emissions to the state Environmental Management Commission.

Ultimately, the state issued a permit which required a year of meteorological studies before the plant could open. CAP prompted the Town of Boone to invoke its extra territorial jurisdiction and the site is now zoned to prohibit heavy industry. Two years of grassroots organizing by Citizens Against Pollution (CAP) and BREDL prevented an asphalt plant in an unzoned community in Watauga County, on the banks of the New River.

The detailed study of weather conditions at the proposed plant site may be the final battle for CAP, or it may spark the beginning of new smokestacks across western NC. Our concerns are that the study, if done poorly, would form the basis for DAQ's approval of new and larger air pollution sources in the mountains.

In April 1999 our staff and volunteers prompted DAQ to produce a written protocol for the meteorological study. In May of that year the Director of DAQ, responding to our questions, said the study would not address the duration of air inversions. This was a change from DAQ's previous statements. These data are essential for a comprehensive study of unique mountain air pollution characteristics.

The shortcomings of the study are that the methodology and equipment will detail only temperature, wind direction and velocity, and relative humidity. And the study does not address air inversions, fogs and other observable events, and pollution trapping. In fact, the study does nothing to address the issues of concern in the state's record of decision about the plant. We suspect that the study is designed to provide data for a new air toxins model which will allow far more pollution without taking into account the special conditions of mountain valleys. Meteorological data collection was completed in 2000.

Jefferson

In October we began working with citizens in Ashe County who were concerned about a proposal for a new asphalt plant near Jefferson, NC. Organizing proceeded quickly and on November 18, 1998 Ashe County Board of Commissioners approved an Ordinance to Place a Moratorium on the Construction and Erection of Asphalt Plants in Ashe County. Ashe Citizens Against Pollution (ACAP), with assistance from BREDL staff, sponsored radio ads, mailed out newsletters, posted yard signs, published newspaper display ads, and generated many news stories in local newspapers, all in a campaign to prevent Tri-County Paving, Inc. from building an asphalt plant at the foot of Mount Jefferson State Park and 6/10 mile from the county high

school.

In January 1999 NC DAQ issued a draft air pollution permit for the proposed plant with a production total of 450,000 tons of asphalt per year. But no plant was ever constructed. During the intervening months BREDL and ACAP members documented the blasting and earth-moving done by the owners at the proposed site. This activity altered the topography and invalidated the computer modeling data submitted to DAQ by Tri-County Paving. We brought this to the attention of state officials who delayed the public hearing and required Tri-County Paving to submit corrected information. We also documented illegal dumping by Tri-County which prompted a notice of violation from the Solid Waste Section of DENR.

Hundreds of people attended the public hearing at the Ashe County courthouse on March 22, 1999. Thirty-eight people spoke in opposition to the plant. Only one spoke in favor: the attorney for Tri-County Paving. Dr. Bill Horn, a pediatrician, noted the asphalt plant's proximity to the high school and said, "There is no justification for putting polluting industries near places where children are playing." Eileen Hartzog presented a petition with 100 student signatures opposing the plant. Kim Faw, whose home is across the road from the proposed plant, addressed the loophole in the state law for toxic pollution saying, "Count all the pollution!"

On May 10 DAQ issued a final permit for a reduced output of 204,000 tons per year. On June 2, 1999 ACAP and BREDL filed a legal challenge against the permit. It stated that DAQ failed to consider on-site meteorological conditions at a mountain site, used erroneous data about the plant, and failed to adequately consider impacts on the high school and the state park.

In July 1999 Tri-County Paving sued Ashe County over the imposition of the moratorium. ACAP and BREDL attempted to intervene on the side of the county, but the judge refused our request. We maintained close contact with county attorney and filed an amicus curiae, friend of the court, brief.

A few months later, the state air pollution permit was rendered useless. On November 4, 1999, near the end of the one year moratorium, the county passed a Polluting Industries Development Ordinance which applied to "all areas of unincorporated Ashe County." The ordinance established county permit requirements and fees for "any polluting industry determined to be so" in unzoned, unincorporated areas outside of town limits. The ordinance names specific industries: mines, quarries, and asphalt plants. It prohibits asphalt plants within a thousand feet of residential and commercial buildings, and within a quarter-mile of schools, day care centers, churches, hospitals, or nursing homes.

In 2001 ACAP continues to watchdog the situation and remains a chapter of the Blue Ridge Environmental Defense League. Members attend county meetings and spread their anti-pollution message whenever possible. Beginning in 2000 they sponsored a superhero cartoon series in the local newspaper which pits Captain Acap against his nemesis, Joe Polluta. Tune in next week for the continuing adventures....

Cullasaja

On October 14, 1998 the NC Division of Air Quality issued an air pollution permit to Rhodes

Brothers Paving in Macon County, NC. The permit allowed production of 180 tons/hour and 100,000 tons/year. Three weeks later BREDL community organizers attended the first community meeting called by residents of the Bethel community in a small church on the Cullasaja River. The state misjudged the level of interest in this rural mountain community and neither notified the people nor held a public hearing. Citizens formed Neighbors Against the Cullasaja Asphalt Plant (NACAP), a chapter of the Blue Ridge Environmental Defense League.

NACAP and BREDL filed suit on December 14, 1998 against the permit issued by the DAQ. Rhodes Brothers Paving, Inc. installed asphalt equipment on property next to the pristine Cullasaja River near Franklin. We challenged the permit on the basis of public health protection in a mountain valley and the failures of public notice and opportunity for public hearing. We later learned that the permit was issued over objections of regional DAQ staff in Asheville.

On May 4, 1999 the asphalt plant commenced operations. Residents were immediately driven from their homes by the smoke and stench. One 90 year old woman confronted the asphalt plant operator on his property and said she would not leave unless the plant shut down. The Secretary of DENR ordered the plant to close and called for an investigation. On May 26th the NC Division of Epidemiology sent Dr. Luanne Williams to meet with the residents and visit the plant site. Residents who attended the meeting were appalled when she said that, based on the data provided to the state by the asphalt company, the emissions were within safe limits.

On September 21, 1999 at the contested case hearing, Administrative Law Judge Beecher Gray ruled that the state acted properly in issuing an air pollution permit to Rhodes Brothers Paving.

Dr. Ravindra Nadkarni, a national expert on asphalt plant pollution, testified on behalf of the citizens' group. He raised many technical issues which showed that North Carolina's asphalt plant permits cannot ensure public health protection as required by law. For example, the state uses the same computer model to predict air pollution levels from both the smokestack and truck loading operations. Dr. Nadkarni said, "The state's model is a quick and dirty approach to pollution prediction, but it is inappropriate for ground-level conditions."

Macon county resident Jerry Starr, testified about the effects of the Rhodes Brothers Paving plant near his home. He said, "Nothing could have prepared us for the horrors of that plant; we cannot be outside when it operates, we are prisoners."

Despite the loss, NACAP and BREDL continued the campaign. Lou Zeller, who testified on behalf of BREDL, said after the hearing, "Someone must listen to the people who are being poisoned by toxic pollution. We appeal to the EMC and Governor Hunt to rein in a state agency which is out of control." The next step was a hearing of the issue before the NC Environmental Management Commission.

On March 9, 2000 the NC EMC voted unanimously to overturn Judge Gray's decision and revoked the Rhodes Brothers permit. The EMC based the decision on the state's failure to hold a public hearing and the failure to notify the Macon County Board of Commissioners. The EMC's final decision was issued May 29 but on May 30 Rhodes Brothers filed for a temporary restraining order against the EMC, allowing the company to continue operations.

In April 2000 Rhodes Brothers Paving, assisted by Kilpatrick Stockton LLP, its Raleigh law firm, submitted a new application for the plant it had operated since May 1999. On May 30, 2000 the DAQ held a public hearing in Franklin which was attended by 300 people; the majority of speakers and 57 of 68 written comments opposed the new permit. Also, the US EPA sent a letter to the Director of NC DAQ which outlined five problems with the Rhodes Brothers permit, signaling a new level of federal scrutiny of North Carolina's permitting process. During this time there were numerous complaints of noise and odor from residents near the plant and a considerable
number of recurring and uncorrected violations of permit conditions.

On August 8, 2000 the DAQ issued an air pollution permit which was virtually identical to the first. NACAP and BREDL have filed a petition for a contested case against this permit, too. The conditions which made the plant intolerable to the people of the Cullasaja are unchanged by legal maneuvering and bureaucratic manipulation. In 2001 BREDL and NACAP will continue this campaign because, as Martin Luther King Jr. said, "the arc of the moral universe is long, but it bends towards justice."

UPDATE: March 8, 2002: The Blue Ridge Environmental Defense League Bethel-Cullasaja community health survey report. The Bethel-Cullasaja community is the site of an asphalt plant permitted to produce 180 tons/hour and 100,000 tons/year of paving asphalt.

White Oak Creek

In December 1997 Polk County adopted an ordinance regulating asphalt plants and five other polluting industries. In 1998 D&S Asphalt Materials submitted an application to build an asphalt plant on White Oak Creek near Tryon. D&S planned to bring in a plant manufactured in 1965 which could emit double the visible pollution of modern plants. On July 6, 1998 BREDL organizers attended the first meeting of local residents opposed to the company's plans. Together we outlined a plan of action which included public meetings, letters and ads in local newspapers, and requests for protection by the county government. At the request of Foothills Action Committee for the Environment, Polk County strengthened the polluting industries ordinance in August.

On November 16, 1998 North Carolina DAQ held a public hearing in Polk County High School attended by hundreds of citizens, the majority opposing the pollution permit. This region of Polk County is in the heart of the Isothermal Belt and standard computer models do not account for air pollution impacts in this mountain region. We submitted these data to the state. But in January the DAQ approved the permit.

Foothills Action Committee for the Environment and BREDL opposed the permit granted and filed against it on February 12, 1999. Our challenge was based on the state's failure to protect public health. Also, FACE is challenging the county's lack of enforcement of its moratorium on new asphalt plants and other polluting industries and the issuance of a building permit to D&S in the flood plain of White Oak Creek.

On September 21, 1999 the Administrative Law Judge heard the combined cases for White Oak

Creek and Cullasaja. This was an example of being right on the issues and losing the decision. Those who attended agreed that our attorney, John Runkle, and our witnesses, technical expert Dr. Nadkarni and the citizens from NACAP and FACE, did an excellent job of presenting our case. But the judge did not even take a day to review the information presented. He made a decision from the bench within ten minutes of the last witness' testimony.

BREDL and Foothills Action Committee for the Environment continue to oppose this permit. On September 20, 2000 D&S Asphalt plant stack tests were reported in the Tryon Daily Bulletin. The tests were performed by a consultant hired by the asphalt company and measure how many pounds of particles are emitted from the smokestack per hour. D&S Operations Manager, Matt Davis, reportedly expressed relief at the result of these tests and said neighbors of the plant used "scare tactics." But the state requires asphalt plants to obtain permits to do tests because they emit thousands of pounds of toxic pollution.

The legal limits for D&S Asphalt allow the plant to emit a total of 14,600 pounds of arsenic, benzene, formaldehyde, and other toxins every year. But D&S Asphalt has never been tested for emissions of toxic air pollutants. In fact, no asphalt plant in NC is tested for toxic emissions. These pollutants are estimated by computers and mathematical formulas rather than by actual stack testing, formulas which experts agree do not accurately predict the effect of toxic fugitive emissions.

The question for is: do pollution limits protect public health and community values? To protect local community values, the state allows counties to determine where new industrial facilities shall be located. The county can determine whether an asphalt plant may operate within, say, a mile or a quarter-mile of an existing elementary school or residential neighborhood. But the state has been derelict in its obligation to protect public health from air pollution. The DAQ and other agencies issue new permits as if no other pollution exists, as if an asphalt plant was located on the Moon. But here on Earth, pollution from thousands of legally permitted smokestacks adds up to a general threat to human and environmental health. The toll in human misery is intolerable and it is up to all of us to put an end to business as usual. We are committed to a continuing campaign of clean air for children's health!

APAC:

POLLUTING WITHOUT BOUNDARIES

A Report On APAC-Atlantic Asphalt Plants in North Carolina

September 23, 2005

by the

BLUE RIDGE ENVIRONMENTAL DEFENSE LEAGUE

www.BREDL.org PO Box 88 Glendale Springs, North Carolina 28629 Phone (336) 982-2691 Fax (336) 982-2954 BREDL@skybest.com

Esse quam videre

Overview

APAC-Atlantic, Inc. operates thirteen asphalt plants in North Carolina. This report will cover violations of environmental laws and citizens' complaints of the five APAC-Atlantic plants which are recorded in state files of the Asheville Regional Office of the NC Department of Environment and Natural Resources Division of Air Quality. The five operating APAC-Atlantic asphalt plants in the Asheville Region are located in Rutherfordton in Rutherford County, Burnsville in Yancey County, Hendersonville in Henderson County, Morganton in Burke County and Penrose in Transylvania County. About a year ago APAC-Atlantic changed its corporate name, so most of the documents in this report will refer to APAC-Carolina; it is the same company.

The APAC-Atlantic plants in this region range from small to large:

Location	Permitted Annual Production Limit (tons)
Penrose	145,500
Henderson	400,000
Burnsville	730,000
Morganton	815,000
Rutherfordton	2,600,000

APAC is one of the nation's largest transportation construction contractors. It possesses huge resources of financial capital, business acumen and engineering skill. But the company's track record in western North Carolina reveals a corporate behemoth with its eye on the bottom line; it seems to care little for the health and well-being of the people who live nearest the asphalt factories it operates.

Help in gathering the information for this report was provided by the able research team of the Rutherford County Citizens Against Pollution.

Louis Zeller, Campaign Coordinator
Blue Ridge Environmental Defense League
September 23, 2005

APAC-Atlantic, Inc.

Penrose
Transylvania County

April 12, 1983 Complaint filed with NC DEM-ARO by local resident who reported that the operation of was much worse this year, citing excessive noise, dust, smoke, and a heavy odor. State inspector noted earlier malfunction which should have required the plant to shut-down during repair. Inspector also noted that noise and odor was consistent with those encountered at other asphalt plants.

April 27, 1983 Resident complained of excessive dust and flames 30 feet high from burning tar. NC DEM inspector arrived May 4, asphalt plant dryer was not operating.

May 19, 1983 Letter from resident to APAC plant operator complaining of "nauseating" exhaust gases which cause headaches. The resident had measured "loud, rumbling" noise levels of 83-86 decibels from his front porch, 240 feet from the plant. He added, "Even when the plant is not operating, the smell from asphalt lingers over our property and in our house."

May 26, 1983 NC DEM inspector finds plant to be in compliance with regulations.

June 3, 1983 NC DEM investigator met with APAC to discuss complaints and possible actions to reduce noise, smoke and dust. Measures completed by July 14, 1983.

September 16, 1983 NC DEM inspection done when plant not in operation.

October 19, 1983 Complaint from resident to NC DEM prompts site visit same day. Visible emissions of 20-30% noted from pollution control device (baghouse filter), indicated non-compliance with state particulate regulations, and large hole in dryer emitting dust.

April 26, 1984 NC DEM inspector noted 5-20% opacity from baghouse, several holes in plant emitting dust; observed "marginal" compliance with particulate regulations but no other violations.

June 5, 1984 Resident called NC DEM complaining of dust worse than ever. No violations noted by NC DEM during inspection 9 days later.

August 6, 1984 Resident again complained on excessive dust from asphalt plant. NC DEM inspected plant two days later, observed excessive dust, recommended repairs. Inspection in October noted no violations.

May 31, 1985 Resident complained of excess dust from plant. NC DEM recommends that plant operator spread oil on roadways.

September 5, 1985 Following a series of complaints from local residents, NC DEM inspectors observe visible emissions of 15-60% opacity. Many holes found in pollution control device (baghouse). Problem corrected.

April 10, 1986 Resident called to complain of dust and grit. NC DEM inspection the following day.

August 4, 1986 A letter from a local resident to NC DEM pleading for help states, "More than three years have passed since we first contacted your department and there is still an excessive amount of dust ejected almost every day the plant is operating. This dust is ejected through the stack, through various leaks in the system and dust kicked up by the loader and truck traffic." He enclosed a series of photographs depicting a "blow out" of pollutants from the asphalt plant. He also said that a "black, oily" substance coated plants along the branch and oil was in the creek. Finally, he noted that the adjacent rock quarry generated large amounts of dust which could be seen rising "above the rim of the quarry." One week later the state inspector replied promising increased surveillance.

August 10, 1987 NC DEM inspector reports plant in compliance with permit.

August 17, 1988 NC DEM reports plant in compliance.

May 1, 1989 In a letter of complaint to NC DEM a resident states that the pollution problems continue to get worse and provides recent photographs of dust clouds at the plant; he recites the history of failure by the state to properly regulate APAC's Penrose asphalt plant, adding, "Do I have to call your department every time there is a hole in one of the dust collector bags or every time the sprinklers are not turned on?" He received no reply.

August 22, 1989 NC DEM reports plant in compliance.

October 20, 1989 Another letter from resident to NC DEM states problem still worse and encloses 18 more photographs taken June through October.

September 6, 1990 NC DEM reports plant in compliance.

August 12, 1991 Yet another letter from resident to NC DEM states problem continues at APAC's asphalt plant. More photographs provided.

March 10, 1992 NC DEM/DENR issues Notice of Violation to APAC-Carolina (now APAC-Atlantic) for "excessive fugitive particulate emissions coming from aggregate dryer and sifting screens at the plant."

Spring 2000 New drum mix plant installed

September 26, 2000 Resident calls NC DAQ complaining that the APAC Penrose plant is operating at night, producing more odor, more emissions and more noise. State inspectors arrived at the plant site on October 4 and noted no violations but recommended further surveillance to determine if an odor problem exists.

November 16, 2000 Resident calls NC DAQ to complain about plant "spewing" pollution which hangs in the air and an "awful smell" which got worse after the new plant was installed. State inspectors arrived later that day after the plant had ceased operation and noted no problems.

July 17 and 18, 2001 Resident phoned in to NC DAQ complaining of odors driving her out of her house. State inspectors arrived at plant to find faint odor on site only.

August 6, 2001 Resident calls NC DAQ with odor complaint. Inspectors arrived next day, found no objectionable odor, gave resident odor log to record occurrences.

September 30, 2002 NC DAQ inspection finds no problems.

APAC-Atlantic, Inc.
Hendersonville
Henderson County

May 29, 1992 NC DEM issued a Notice of Violation for improper operation of the baghouse filter.

May 18, 1995 NC DEM issued a Notice of Violation issued for improper operation of the baghouse filter and a fugitive dust problem.

June 26, 1997 NC DAQ inspection notes visible dust emissions from baghouse filter. On July 1st NC DAQ Issued a Notice of Violation for improper operation of facility resulting in excess air pollution.

October 9, 1997 NC DAQ inspectors again noted visible emissions from baghouse filters. A new Notice of Violation was issued on October 16th. On May 13, 1998 NC DAQ fines APAC-Carolina (now APAC-Atlantic) $1,433 for the October 16, 1997 violation.

October 16, 2001 During stack testing, NC DAQ discovered "strong asphalt odors" in the trailer park across the road from the APAC plant. The inspectors notified the company.

December 5, 2001 NC DAQ inspection prompted by citizens complaints about another asphalt plant in the area finds "strong asphalt odors" in the in the Kingswood Hill Subdivision located near the APAC Hendersonville asphalt plant.

March 25, 2002 DAQ Air Toxics Analytical Support Team performs tests of asphalt plant recycled fuel oil for chemical contamination. Hendersonville plant fails test; elevated halogen levels were found to be from 26% to 123% over maximum limit.

March 27, 2002 NC DAQ issues Notice of Violation to APAC-Carolina (now APAC-Atlantic) for burning No. 4 recycled fuel oil with a total halogen concentration of 2230 parts per million (maximum in 1000 ppm).

May 16, 2002 NC DAQ issued a Notification of Objectionable Odors and Requirement to Implement Maximum Feasible Controls. The notice was based on many citizens complaints which resulted in a determination by NC DAQ that there were "very strong odors beyond the APAC facility's boundaries." APAC-Carolina (now APAC-Atlantic) objected but was required to submit compliance schedule by the Director of DAQ.

April 7, 2003 NC DAQ issued Notice of Violation to APAC-Carolina (now APAC-Atlantic) failure to comply with arsenic emission limitations. The company was cited for failing to meet fuel oil requirements for recycled oil delivered between April and June 2002. The maximum arsenic level for fuel oil is 1 part per million, but APAC's Annual Report submitted to NC DAQ stated that No. 4 fuel oil delivered during that time had an arsenic level of "less than 1.94 ppm." APAC then contracted with another laboratory, Precision Petroleum Labs in Houston, to re-test the fuel oil to show no excess arsenic.

May 13, 2003 NC DAQ issued Recision of Notice of Violation based on re-testing done by APAC-Carolina (now APAC-Atlantic).

July 21, 2004 NC DAQ issues Notice of Violation for failing to submit quarterly reports. APAC is required to report its monthly asphalt production, annual asphalt production, NOx, SO2 and CO emissions, and recycled fuel oil tests. APAC-Atlantic requested to remove the quarterly reporting requirement in its next permit for the Hendersonville plant.

APAC-Atlantic, Inc.
Burnsville
Yancey County

September 25, 1986 Resident complains of excessive dust, said, "he had never before seen an asphalt plant emit that much dust." Pollution control device (baghouse filter) found to have two broken bags.

October 8, 1986 Dust from asphalt plant causes complaints from residents who report the problem has persisted for several weeks. The plume was visible four miles away from asphalt plant, according to state official.

June 10, 1987 Notice of Noncompliance issued by NC DEM for failing to control visible emissions (15A NCAC 2d .0521) exceeding 20% opacity. On June 19 APAC responds to DEM saying problems with baghouse filters fixed.

June 11, 1987 Four residents call NC DEM to complain of excessive dust from asphalt plant.

September 15, 1987 NC DEM inspection again cites failure of operator to control visible emissions, opacity as high as 65%. Photographs of smokestack taken by DEM.

December 1, 1987 DEM issues Notice of Noncompliance for "large quantities" of emissions from several holes in plant observed by state inspectors on November 19th.

February 5, 1988 APAC-Carolina (now APAC-Atlantic) fined $1,722.70 for failure to control emissions. Previous notices of noncompliance by company noted in legal documents: June 6, 1975 and April 19, 1977.

August 22, 1996 NC DEM inspectors note visible emissions from baghouse filter and no method of dust control. Plant operator said bag filters had been changed two weeks earlier. Inspectors recommended Notice Of Violation be issued for excess particulate emissions (15A NCAC 2D .0506c).

August 23, 1996 A different NC DEM inspector noted visible emissions in excess of 20% opacity, recommended Notice of Violation for improper operation of the plant. The NOV for the two occurrences was issued on April 30th.

August 7, 1997 DEM issues two Notice of Violation for repeat offense of excess particulate emissions caused by lack of dust control (15A NCAC 2D .0506c) and fugitive emissions from the baghouse filter.

June 3, 1999 NC DEM inspection notes excess visible emissions up to 40% opacity from the baghouse filter. A Notice of Violation was recommended. On June 8th DEM inspectors again visited the plant following reports of persistent excess emissions up to 30% opacity. On June 10, 1999 NC DEM issued a Notice of Violation for the two events.

September 2, 1999 NC DEM inspection notes plant operator exceeded maximum hourly production limit. Permit stipulated 150 tons/hour while operator was running at 165 tons/hour. Operator reported to have said he thought the plant could run as high as 180 tons per hour. No violation recommended by NC DEM because annual limit not exceeded and state air toxic limit had not been "triggered."

July 17, 2002 NC DEM inspector recommended Notice of Violation for failure to operate plant properly causing excess visible emissions of up to 50% opacity. APAC-Atlantic contested the NOV and no record of a violation being issued was in the file.

September 17, 2003 Resident calls NC DAQ to complain about plant, said that cloths hung on line and outside furniture get covered with soot and black film and that they have to keep their windows closed.

APAC-Atlantic, Inc.
Morganton
Burke County

April 14, 1992 Visible emission of 30-40%, violation for failure to effectively maintain pollution control equipment (baghouse filter).

June 20, 1996 Warning letter for visible emission of 40-50%, violation of (15A NCAC 2D .0521). APAC operated two units at this site, and older batch mix plant and a newer drum mix plant. The violation occurred at the newer plant which was using an "anti-strip" additive made of animal fat by products. The anti-strip was required by NC DOT.

June 26, 1996 Visible emissions of 40-50% again noted by NC DENR inspectors, again from NC DOT required additive.

July 22, 1999 At this inspection NC DENR noted that the offending plant had been relocated to Mecklenburg County.

APAC-Atlantic, Inc.
Rutherfordton
Rutherford County

April 23, 2003 Odor complaint phoned to ARO-DENR by man living about one mile from the Rutherfordton plant. Resident said, “the odor bad, burning eyeseven inside house.”

May 14, 2003 NC DENR Inspector issued NOV for violation of (General Condition #6) excess dust emissions from pollution control device.

POSTED ON FEBRUARY 20, 2002:

Paving the Way

How behind-the-scenes lobbying allows big-money interests like the asphalt industry to steamroll citizens

By Jennifer Strom

For six years, Doug Robins has paved driveways and parking lots with asphalt he buys from other suppliers. About a year ago, the owner of Asphalt Experts decided to open his own plant. But he couldn't find a place to build it, thanks to a law that puts 1,500 feet between the smoky blue plumes of asphalt plants and the places people live. So he did what other Durham business people do when they need help building things. He went to City Hall.

Carolina Sunrock Corporation on Camden Avenue dumps a load.
Alex Maness

Greg Payne, then the city's economic development director, and members of the city/county planning staff got together to work on Robins' problem. After studying their laws and their maps, the staffers suggested Robins request a change to the law to reduce the 1,500 feet to 600 feet.

The plan seemed headed for success as it slid routinely through planning and zoning hearings last summer. It squeaked through Durham's one environmental review board, and the City Council planned to vote on it in September.

But one citizen watchdog sounded an alarm after learning of the proposal by accident. Days before the vote, neighborhood activist and NAACP leader John Schelp roused enough outcry to convince the council to delay the decision. Since then, Schelp has mobilized a diverse coalition of neighborhood activists, civil rights advocates, students and environmentalists.

The proposal would create 10 new sites for potential asphalt plants, which mix gravel, sand and rock with petroleum-based cement, emitting pollutants such as benzene and formaldehyde. Of those, eight are in East Durham, meaning less affluent, inner-city minority communities would bear the brunt of unsightly, odorous and potentially hazardous pollution from a smokestack industry feeding growth in the wealthy white suburbs.

A public debate is now raging over whether it's a good idea to put asphalt plants within two football fields of houses. But until Schelp rallied an unexpected but efficient revolt, the industry's unimpeded progress toward its goal was business as usual in Durham's planning process, where business interests influence the decisions that shape the landscape and affect citizens' everyday lives.

"Industry reps have insinuated themselves into every corner of policy-making, with a deleterious effect for citizens," says Lou Zeller, the research director and Clean Air Campaign organizer for the Blue Ridge Environmental Defense League. His group battles asphalt plants and other controversial industries all over the state. "If the law is inconvenient for the industry to do business, they change the law."

Those who stand to profit from policy decisions have several tactical advantages that begin long before residents have a chance to speak at a public hearing. They originate on the ground floor of City Hall, where the city/county planning staff jokes congenially with developers who come in to study the wall maps and chat about their plans. They continue upstairs, in the economic development office, where the staff's mission includes helping local businesses grow.

Developers and business owners pay consultants to spend hours lobbying and clearing the way for their plans--consultants who often have long-standing relationships with staffers. They provide reams of background information to the planning department, data that often becomes the basis for staff recommendations in their favor.

On the other side, citizens with less technical knowledge and familiarity with the planning process rely on the public process to alert them to what's happening in their neighborhoods. In the asphalt case, the hearing notice was 10 words long, buried in a page-long, inch-wide classified ad, and no letters went out to neighborhood groups.

And even when they do participate in the public process, citizens--and their elected leaders--often find themselves at a disadvantage, says County Commissioner Ellen Reckhow.

"We are not experts on these topics. But the industries come in with all this information and they can tell their side of the story really well," says Reckhow, who chairs the Joint City-County Planning Committee where the asphalt proposal first surfaced in March. A planner by

training, Reckhow calls the overall process "lopsided."

City and county staffers are quick to say their job is to serve the entire public. Anyone can walk into the planning office, ask for a change to the zoning law and launch the same approval process the asphalt industry is now pursuing. Interim Planning Director Dick Hails remembers an example from the 1980s, when a group of neighborhood leaders sought to change the zoning of adjacent properties to preserve open space near their homes.

But most of the time, few people without a financial stake work the system.

"The person that's going to be the most aggressive, and is more likely to spend the time in the planning office pushing projects, are the people who are proposing development," says Mayor Bill Bell. "And they have the time to do it, as opposed to John Q."

In the asphalt case, the financial stakes are high: the N.C. Department of Transportation awarded $1.2 billion in state road contracts last year alone. David Rifenburg, of Rifenburg Construction, who eventually joined Robins' effort to change the law so he, too, could build a Durham asphalt plant, has won bids for major road projects across the state. Rifenburg built the Martin Luther King Jr. Parkway in Durham, a $7.6 million project, and two years ago resurfaced the city's roads with 22,000 tons of asphalt supplied by Carolina Sunrock Corporation. Carolina Sunrock, one of the two existing asphalt plants in Durham, just won a bid to provide asphalt to the city this year, a $147,028 contract.

The Carolina Asphalt Pavement Association (CAPA), the industry lobby in Raleigh, estimates that public road contracts make up only about half the asphalt market. Private projects such as shopping malls and housing developments make up the other half. Rifenburg's private clients include Duke University (football stadium improvements and hospital complex) and Beechwood subdivision in Chapel Hill.

Like other developers with profits at stake, both asphalt companies hired consultants to help work the deal. Asphalt Experts hired Durham attorney Will Anderson, and Rifenburg hired local land planner George Stanziale. Stanziale's friendly relationship with the Durham city/county planning staff dates back to 1984, when he successfully advocated for controversial rezonings that led to the development of tony Treyburn.

"From my point of view, they always try to be helpful," Stanziale says of the planning staff. "Their job is to protect and legislate the [zoning] ordinance. But when someone wants to get something done on a piece of property, and it doesn't meet the ordinance or a particular plan, say, a small-area plan, they're going to always work with us to see how we can get it done."

Stanziale does frequent business with the planning department, wearing several hats on behalf of his clients and his firm, Haden-Stanziale. He represented developers of car dealerships as part of the controversial Streets at Southpoint mall. He co-authored the Downtown Master Plan and is often tapped to serve on advisory committees, such as a 1998 group that recommended changes to Durham's natural resources protections. And he profits from city business, winning publicly funded contracts such as the $78,000 job designing West Chapel Hill Street Park in 2000. He is currently consulting on a rewrite of the city's development ordinance.

Stanziale has never lost a Durham rezoning case. He says that's partly because he chooses his projects carefully but also because he has "great relationships" with the planning staff and involves them early in his plans.

Those relationships are one factor that contribute to citizens feeling disenfranchised by the planning system, says one critic.

"They've been doing this so long, some of them don't see how they bend over backwards for the developers. Then they don't understand why people like me think this is outrageous," says Steve Bocckino, a citizen activist in southwest Durham who got involved in politics when he led his neighborhood's battle against the Streets at Southpoint mall at Fayetteville Road and Interstate 40. Bocckino now serves on the planning commission, where he is frequently outraged by staff recommendations that side with developers, often based on what he calls "tortured logic." "They see the developers all the time and the developers are their friends. And they see residents as contentious naysayers."

But while the planning staff plotted to help Doug Robins and David Rifenburg move asphalt plants closer to homes, there weren't any naysayers to contend with, because no one knew about the plan as it gathered steam behind the scenes.

After cooperating with the planning department to develop language that was acceptable to both the staff and the industry, Stanziale and Anderson took their proposal to the planning committee for review in April.

Zoning text amendments affect property across the county, rather than altering the rules for one particular piece of land the way a rezoning does. Text amendments also differ from rezonings in another crucial way: They require very little public notice. If a resident wants to change a house into an office, the city/county planning department sends a letter to nearby neighbors, with details of the request, an explanatory map, contact numbers for questions and dates and places of public hearings.

But if the asphalt industry--or anyone else--wants to amend the law, the government puts an

advertisement in the local newspaper, back in the classified section, lumped in with a long list of other issues described in technical language.

In presenting the proposal to the planning committee, the industry reps argued several key reasons for the change: The 1,500-foot setback rendered it impossible to build a new plant; changes in asphalt technology and "tougher EPA air quality standards" have cut down dust and noise affecting surrounding properties; and "there is a need for more asphalt plants in Durham to meet local needs."

The planning staff backed them up, recommending approval based on the applicants' arguments and a few of their own. One point staffers cited was that the 1,500-foot setback had arisen out of a 1980s rewrite of zoning laws in the wake of a chemical explosion and a chemical fire in East Durham. During the revisions, asphalt plants were lumped into the same category as hazardous materials and explosives.

"We looked at the actual impacts, and reported to the commission that asphalt plants fall somewhere between concrete plants and hazardous waste," says Hails. Concrete plants require only 100 feet of buffer space, while hazardous waste facilities require the full 1,500 feet.

The planning staff also mentioned that Raleigh, Greensboro and Charlotte do not require any distance between asphalt plants and homes, a point that struck Lou Zeller of the Blue Ridge Environmental Defense League as ironic.

"If Durham is even considering changing their setbacks, that's just incredible to me," Zeller says. "They would be undoing a good measure that protects public health in order to 'dumb-down' to what other communities have."

The citizen coalition has since pointed out several examples of other cities, including Boston, that are expanding the buffers for asphalt plants based on environmental and health concerns.

Another reason the staff cited in its recommendation was Durham's growth, saying "major road improvements and development projects maintain a high demand, with much asphalt being trucked in."

"The rule of thumb is, if we are demanding it, we should take some responsibility for providing it," says Hails. "If there's a demand for something in the community, then you look at whether there's an appropriate site."

The planning committee recommended approval, but also asked the Environmental Affairs Board to review the plan before it went any further.

The EAB, a joint city-county advisory panel, is made up of local residents with professional credentials in a variety of fields. Elected officials created the board a decade ago to provide them with expertise when faced with decisions just like this one.

"The EAB is there because [Durham County Commissioner] Becky Heron thought if we're giving the Chamber of Commerce money, we should do something to balance the other side," says Bocckino. "It's sort of the environmental chamber of commerce."

The 11-member board has specific slots for individual disciplines such as solid waste, water quality, air quality and public health, with the city and county each appointing five members and one seat for a representative of the Soil and Water Conservation District Board.

The EAB discussed the asphalt measure several times, eventually voting 4-3 to approve it with some changes. The board recommended expanding the setback from 600 feet to 750 feet, and requiring a solid 8-foot perimeter wall. An eighth member who had to leave the meeting early has indicated he would have voted against the plan, meaning it narrowly missed a 4-4 tie. One of the points that swayed the supporters was the staff's argument that Durham's setback was so much larger than other North Carolina cities, says one EAB member, who has since been chagrined to hear--via the citizen effort-- that buffers in other states are much larger, and in some cases, expanding.

One of the three no votes was member Marian Johnson-Thompson, who holds the EAB's public health seat.

"It's a situation--again--where money and big business come in and, I shouldn't say have no regard for poor people, but they're not as sensitive as they should be," says Johnson-Thompson, whose profession is studying health disparities among different ethnicities. "As a member of the board, I was embarrassed about how this vote went down."

The EAB discussions focused primarily on noise and dust, despite Johnson-Thompson's concerns that the health and quality of life for Durham's less affluent communities of color were the real target. The industry reps produced a voluminous stack of supporting paperwork, including a report arguing that asphalt plant emissions were no more dangerous than those of an average bakery.

"We did a lot of homework for them," says Stanziale. "We felt like we answered all of their questions."

With approval from the planning committee and the EAB, the proposal went next to a public

hearing at the zoning committee.

John Schelp was sitting in the City Council chambers that night, waiting for his turn to speak. As the president of the Old West Durham Neighborhood Association, Schelp was there to urge the zoning committee to require more open space in "mixed-use" developments.

When the asphalt industry representatives stepped forward, Schelp listened in disbelief as they outlined their proposal.

The industry reps repeated their arguments: the lack of eligible land under the current zoning law and the growing market demand for asphalt in Durham. They said recent technological advancements reduce the environmental impact and health hazards. Missing from the discussion, Schelp says, was any recognition that the residents most likely to be subjected to new asphalt plants within 600 feet of their houses were low-income minorities.

"I thought to myself, we can debate health effects all night, but the asphalt industry says they are only interested in the sites in East Durham," says Schelp. "There's no gray area there."

It was clear to him the proposal had coasted pretty far toward City Council approval with zero public debate, thanks in part to a 10-word-long public hearing notice "the size of a blade of grass."

"The asphalt industry had eight months to whisper 'facts' into the ears of officials while the neighborhoods sat in the dark," Schelp says.

Shocked by the environmental justice issue going unchallenged, and the one-sided nature of the discussion, Schelp began collecting a citizen coalition to shine a public spotlight on the plan.

He started with his position within the Durham NAACP. The only white member of its executive committee, Schelp had been working to build racial bridges for months. A former Peace Corps worker with a master's degree in public administration, six years in the Congo and a long record of neighborhood activism, Schelp had recently been asked to organize the NAACP's "community committee" to bring diverse Durhamites together to work on common initiatives. The five-member group agreed to take on the asphalt project, and the NAACP advanced the committee $50 for publicity.

When the measure was scheduled for a Sept. 16 vote before the City Council, Schelp mobilized letter-writers through an e-mail list-serv that began with the five people on his community committee. It now contains 300 members.

Schelp, two other NAACP leaders and the minister of Morehead Avenue Baptist Church co-authored a letter to the editor published in *The Herald-Sun* on Sept. 16, the day before the scheduled vote.

"It's bad enough that developers are forcing limited local resources to be shifted from less affluent in-town neighborhoods to newly-paved suburban developments," they wrote. "Must we now face the threat of hazardous exposures from the asphalt industry?"

Council members, beginning to receive protest e-mails and facing voters in an election just six weeks away, voted unanimously to table the proposal.

"No one was real excited about this issue once it turned into a hot potato," says Anderson, the attorney for Asphalt Experts.

The neophyte coalition celebrated the delayed vote and began in earnest to outline their next attack. They targeted three issues: the lack of public notice, the potential health hazards, and the disproportionate effect on low-income minority neighborhoods.

Neighborhood associations across the county and churches in East Durham began to spread the word through their memberships. Schelp called Duke University officials to alert them their rare books storage is next door to one of the potential asphalt plant sites. He asked the Eno River Association to look at water quality issues, since one of the potential sites sits along the Eno.

Local environmentalists compiled data about the effects of asphalt plant pollution, including the dangers of toxic emissions from benzene and other byproducts of hot asphalt mixing. Prompted by the protesters, leaders of Environmental Defense, a national advocacy group, wrote a letter to the mayor and city manager citing Durham's ranking among the "dirtiest counties" in the United States based on an average individual's added cancer risk from air pollution. In a letter signed by Director Jane Preyer, the group urged the council to kill the measure, saying more asphalt plants in Durham would raise the county's already dangerous levels of acrolein, a chemical emitted by asphalt plants.

According to the federal Occupational Safety and Health Administration, exposure to asphalt fumes can cause headaches, skin rashes, fatigue, reduced appetite, throat and eye irritation, and coughing. Asphalt paving workers, for example, have reported breathing problems, asthma, bronchitis, and skin irritation, according to OSHA, and studies have reported lung, stomach, and skin cancers following chronic exposures to asphalt fumes.

N.C. Central University professor Yolanda Anderson lent the group an intern and the campus

student group, Central Environmental Action Student Effort (CEASE) got involved. CEASE printed a fact sheet and launched a public education campaign to spread the word to residents in the predominantly black neighborhoods around campus, where four of the potential sites lie. As they walked door-to-door, nine out of 10 residents they talked to had no idea they were living adjacent to a potential asphalt factory, with its burning petroleum smell and dump trucks trailing fumes down the street, says intern Melissa Lewis.

One Morehead Hills resident on the e-mail list eventually sent Schelp a $50 check, saying he was glad to double the group's budget.

When the industry reps said publicly they were seeking "independent data" from the state Division of Air Quality to counteract their critics, Schelp asked DAQ Director Alan Klimek to address the citizens' concerns as well. In a stock response the DAQ has given in similar controversies across the state, Klimek replied that the location of new asphalt plants is strictly "a local issue." Klimek wrote an op-ed piece last year echoing the same sentiments. CAPA, the industry lobby group, likes Klimek's argument so much it now includes his essay in the organization's public information packet.

The industry's influence is even more ingrained at the state level than it is locally, says Zeller, who attends DAQ advisory committee meetings where "The environmental groups don't show up but the industry shows up in droves."

In Durham, as letters from across town poured in to newspapers and City Hall, the local industry reps turned to their state association for backup. CAPA Executive Director Christie Barbee shot back at critics, writing a letter to local newspapers countering Schelp's first attack. She called the environmental injustice issue "baseless" and touted the industry's safety record and Durham's need for asphalt.

"I got a copy of [Schelp's] letter and there was a lot of misinformation about our industry," says Barbee. "No one in our industry strives to be controversial or to stir up trouble. But as city limits grow, you've got more and more area that needs pavement."

Having only two plants in Durham raises the cost of asphalt, says Barbee, citing cheaper prices in areas like Charlotte-Mecklenburg, where there are more than a dozen plants. "Durham is a good market," she says. "And Durham certainly could use the jobs that a plant would bring."

Asked how many employees a plant needs, Barbee estimated "six to eight," but added that the plants also generate related jobs for truck drivers and pavement workers.

As the grassroots organizers mounted their campaign, it was the accusations of environmental

injustice that surprised the city staffers the most, including the interim planning director.

"I've heard of it in other places, but it's new for here," Hails says.

Actually, environmental justice issues in East Durham date back to the 1980s. A chemical company called Armageddon Recycling Co. on Peabody Street was cited repeatedly for leaking barrels that eventually exploded on March 10, 1983. A community group that formed to protest, Citizens for a Safer East Durham, pointed out in its flyers that the situation wouldn't have dragged on so long if the plant were "on the other side of town." Another chemical plant in the same vicinity, SouthChem Inc. on East Pettigrew Street, was the scene of a massive chemical fire on Sept. 3, 1986. Today, SouthChem is still in business and ranks as Durham County's third-largest polluter, according to Environmental Defense.

Overall, Durham's minority residents are four times more likely than whites to live near facilities emitting air pollutants, according to the Environmental Defense scorecard, while families below poverty are also four times more likely to live near polluting facilities than those above poverty.

"If you're poor and black, that's what you get," says Schelp.

The future of the asphalt proposal may be decided within the month. The Joint City/County Planning Committee discussed it again on Feb. 6 and asked for more input from the Environmental Affairs Board. This time around, the industry may have even one more vote, thanks to another behind-the-scenes move. On Oct. 15, the City Council appointed Asphalt Experts attorney Will Anderson to the attorney slot on the EAB. Anderson replaced outgoing member Jim Conner, an environmental activist and lawyer.

The council has scheduled a discussion for Feb. 21. Whether the plan will then be scheduled for a March 4 vote depends on the council, says City Manager Marcia Conner, while complaining about how much time her staff has had to spend responding to the "overwhelming number of e-mails."

Mayor Bell, whose inbox has also been flooded with protests, predicts the grassroots effort has accomplished its mission.

"I don't think this has a chance of going anywhere," he says. "Let me put it this way, I haven't had anybody write me saying we ought to be supporting this."

If they succeed in changing the zoning law, Asphalt Experts and Rifenburg Construction may build a plant together, according to Stanziale.

Schelp and his coalition want the setbacks for asphalt plants to stay at 1,500 feet. They want the planning staff to learn about environmental justice issues. And they want the rules for public notice of text amendments rewritten so they trigger letters to affected neighbors like rezonings do. On the last point, they already have support from the mayor.

"Given the amount of attention that's been given to that issue, I think the staff will look at a little different process," Bell says, though the staff is not so sure. Assistant planning director Bonnie Estes says with text amendments, "It's difficult to know who's impacted, so it would be difficult to know who to notify."

Bocckino and a fellow planning commission member have proposed another system reform: a new ethics policy requiring planning staff and commission members to disclose their personal financial interests.

County Commissioner Reckhow supports improvements to the public notice process and the ethics proposal. But in the bigger picture, she wants the government to balance the interests of people who live here and the people who profit.

"We recognize there's a gap and we're looking for ways to level the playing field," she says. "We need to do a much better job empowering our citizens." ■

URL for this story: http://www.indyweek.com/gyrobase/Content?oid=17208

Coal Tar-Containing Asphalt Resource or Hazardous Waste?

Journal of Industrial Ecology
Volume 11, Issue 4, Date: October 2007, Pages: 99-116
Yvonne Andersson-Sköld, Karin Andersson, Bo Lind, Anna (Nyström) Claesson, Lennart Larsson, Pascal Suer, Torbjörn Jacobson

Abstract:

Coal tar was used in Sweden for the production of asphalt and for the drenching of stabilization gravel until 1973. The tar has high concentrations of polycyclic aromatic hydrocarbons (PAH), some of which may be strongly carcinogenic. Approximately 20 million tonnes of tar-containing asphalt is present in the public roads in Sweden. Used asphalt from rebuilding can be classified as hazardous waste according to the Swedish Waste Act. The cost of treating the material removed as hazardous waste can be very high due to the large amount that has to be treated, and the total environmental benefit is unclear. The transport of used asphalt to landfill or combustion will affect other environmental targets. The present project, based on three case studies of road projects in Sweden, evaluates the consequences of four scenarios for handling the material: reuse, landfill, biological treatment, and incineration. The results show that reuse of the coal tar-containing materials in new road construction is the most favorable alternative in terms of cost, material use, land use, energy consumption, and air emissions.

http://www.energystorm.us/Coal_Tar_containing_Asphalt_Resource_Or_Hazardous_Waste_-r268391.html

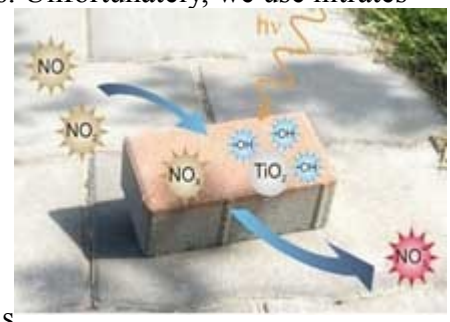
Green Asphalt and Concrete: Eco Friendly Streets

By Nick August 14, 2008

There have been several developments as of late relating to the greening of the world's streets and highways with either asphalt and concrete. Lots of forgotten energy and materials goes into our roads and highways, which are just a bridge for more pollution, making recent developments with greener, more environmentally friendly asphalt and concrete more exciting.

Air Pollution Free in Hengelo -Dutch Concrete paving stones

The recent green developments started in the Netherlands when the Dutch's University of Twente developed a green concrete paving stone that converted the smog, acid rain causing nitrogen oxide into a nitrate, which is not harmful for the environment in small quantities. Unfortunately, we use nitrates all the time in fertilizer, so it could potentially be toxic to humans or animals. The Dutch will be testing it with a grant in Hengelo and scientists will be evaluating it for widespread use.



Air Pollution Reducing Asphalt in Madrid

Engadget reports that Madrid has a similar plan in place with new asphalt that comparable to the Dutch's concrete paving stones. Madrid's asphalt, too, can convert nitrogen oxide from exhausts into less harmful oxides which can just be washed away with rainfall. This asphalt is suppose to be able to get rid of 90% of the nitrogen oxides in the air on a sunny day since it relies on sunlight to work at capacity. Ironic that global warming induced radiation is helpful in combating air pollution. But this is a great development and has a lot of potential.

Laying Cool Green Asphalt is Cool

Earlier this year, the Matter Network discusses how laying asphalt while it is cool instead of hot can save 7 times as much energy during the process. With thousands of miles of US roads getting paved each year, that is some serious energy savings. The problem lies in the fact that US engineers, unlike those overseas, are only familiar with heated laying techniques and measures to predict how well a road will perform. With no cool laying guidelines, it's not realistic for cool laying techniques to be used, yet. So after some control tests headed by University of Wisconsin civil engineering professor Bahia, cool asphalt may become cooler in the civil engineering world thus allowing us to green our roads and environments.

http://www.ecofuss.com/green-asphalt-and-concrete-eco-friendly-streets/

References

Anderson-Sköld, Yvonne; Anderson, Karin; Bo, Lind; Claesson, Anna; Larsson, Lennart; Suer, Pascal; Jacobson, Torbjörn. (2007, October). Coal Tar-Containing Asphalt Resource or Hazardous Waste? *Journal of Industrial Ecology*, Vol. 11, No. 4, pp. 99-116. (abstract only).

Asphalt Pollution Probe Extends. (2004, July 7th). *Recycling Today*. Available at http://www.recyclingtoday.com/news/news.asp?ID=6040

August, Nick. (2008, August 14th). Green Asphalt and Concrete: Eco Friendly Solutions. *EcoFuss*. Available at http://www.ecofuss.com/green-asphalt-and-concrete-eco-friendly-streets/

BE SAFE Campaign. (undated) Asphalt Plant Pollution [Brochure]. Falls Church. VA: Lou Zeller. Availible at http://www.besafe.net.com/Asphalt.pdf

Blue Ridge Environmental Defense League. (1997). Carcinogens Discovered Near Maymead Plant [Press Release]. Glendale Springs, NC.

Blue Ridge Environmental Defense League. (1997). Groups Charge Maymead with Intentional Violations [Press Release]. Glendale Springs, NC. Available at: http://web.archive.org/web/20010303020834/www.boonenc.org/CAP/press_release.html

Blue Ridge Environmental Defense Fund. (2001). Mountain Air Action Project Asphalt Plant Campaign Report. Glendale Springs, NC. Available at http://www.bredl.org/pdf/factsheet-asphaltplants.pdf

Blue Ridge Environmental Defense Fund. (2005). APAC: Polluting Without Boundaries. Glendale Springs, NC. Available at: http://www.bredl.org/pdf/050923_APAC-Atlantic_Report.pdf

Blue Ridge Environmental Defense Fund. (2007). Asphalt Plants: Contaminants of Concern [Fact Sheet]. Glendale Springs, NC. Available at: http://www.bredl.org/pdf/factsheet-asphaltplants.pdf

Boffetta, Paolo; Burstyn, Igor. (2003). Studies of carcinogenicity of bitumen fume in humans. *American Journal of Industrial Medicine*, Vol. 43, pp 1-2.

Brandt, Evan. (2009, April 5th). Asphalt plant, and its pollution potential, part of quarry deal. *The Pottstown Mercury*. Available at http://www.pottsmerc.com/articles/2009/04/05/news/srv0000005039118.prt

Calgary for Clean Air. (2007). http://calgarycleanair.com. Accessed July 2009.

Chase, Robert M.; Liss, Gary M.; Cole, Donarld C.; Heath, Bonnie. (1994). Toxic Health Effects Including Reversible Macrothrombocytosis in Workers Exposed to Asphalt Fumes. *American Journal of Industrial Medicine*, 25, pp. 279

Citizens Against Pollution. (undated). Health Survey Impact of Asphalt at Roby Green Road and US 421. Watauga County, NC: Ronald C. Chivers.

Evans, James V. (1985). Asphalt. In Martin Grayson and David Eckroths (Eds.). *Concise Encyclopedia of Chemical Technology*, pp. 137-139. New York: John Wiley and Sons.

Fernandez, Jennifer. (2001, February 23). Group Opposes Asphalt Plant: The Plant Would be about Half Mile from Colfax Elementary School. *News and Record*

Indians Appeal Asphalt Plant. (undated). *Mendocino Country Environmentalist*, pp. 6, 8.

Karaman, Ali; Pirim, Ibrahim. (2009). Exposure to bitumen fumes and genotoxic effects on Turkish asphalt workers. *Clinical Toxicology*, Vol. 47, No. 4, pp 321-326. (abstract only)

Lang, Leslie H. (2004, December). Increased suicide rate is possibly linked to chemicals released from nearby asphalt plants, study suggests. *UNC School of Medicine*. Available at http://www.unc.edu/news/archives/dec04/weisler121004.html

Montague, Peter. (1997). Childhood Cancer and Pollution. *Rachel's Environmental and Health News*, #559. Available at http://www.rachel.org/bulletin/pdf/Rachels_Environment_Health_News_564.pdf

Nadkami, Ravi. (1996). Asphalt plant emissions: What are the issues during site selection? *Massachusetts Association of Health Boards Quarterly*, 14, pp 9-10.

National Toxicology Program, Department of Health and Human Services: NIOSH(1997, June 23). Literature Review of Health Effects Caused by Occupational Exposure to Asphalt Fumes. Available at http://ntp-server.niehs.nih.gov/index.cfm?objectid=0DA9C8CD-F1F6-975E-7631B117EEDF8C3D

North Carolina Department of Environment and Natural Resources, Division of Air Quality. (undated). Asphalt Plants: Frequently Asked Questions [Brochure]. Raleigh, NC. Available at: http://www.ncair.org/news/brochures/asphalt.pdf

North Carolina Department of Environment and Natural Resources, Division of Air Quality. (1997). Letter to Maymead Materials, Inc. Raleigh, NC: Alan. W. Klimek.

Partanen, Timo; Boffetta, Paolo. (1994). Cancer Risk in Asphalt Workers and Roofers: Review and Meta-Anaysis of Epidemiologic Studies. *American Journal of Industrial Medicine*, 26, pp 721

Sittig, Marshall. (1985). Asphalt Fumes. Pp 97-99 in *Handbook of Taxis and Hazardous Chemicals and Carcinogens* (2nd ed.). Park Ridges, NJ: Noyes

Storm, Jennifer. (2002, January 20). Paving the Way: Behind-the-Scenes Lobbying Allows Big-Money Interests like the Asphalt Industry to Steamroll Citizens. *The Independent Weekly*. Available at http:www.indyweek.com/gyrobase/Content?oid=17208

Tepper, Allison L.; Burr, Gregory A.; Feng, Amy; Singal, Mitchell; Miller, Aubrey K.; Hanley, Kevin W.; Olsen, Larry D. (2006). Acute symptoms associated with asphalt fume exposure among road pavers. *American Journal of Industrial Medicine,* Vol. 49, No. 9, pp. 728-739. (abstract only).

U.S. Department of Health and Human Services, Center for Disease Control and Prevention, National Institute for Occupational Safety and Health. (2000). Health Effects of Occupational Exposure to Asphalt. Pp. viii-xi, xiv-xviii. Available at http://www.cdc.gov/niosh/pdfs/01-110.pdf

U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Office of Air and Radiation. (1995). Mineral Products Industry: Hot Mix Asphalt Plants. Pp. 1-10, 19-36 in *Compilation of Air Pollutant Emission Factors Volume I: Stationary Point and Area Source* (5th ed.). Available at http://www.epa.gov/ttn/chief/ap42/ch11/related/ea-report.pdf

Van Metre, Peter C.; Mahler, Barbara J.; Wilson, Jennifer T. (2009). PAHs Underfoot: Contaminated Dust from Coal-Tar Sealcoated Pavement is Widespread in the United States. *Environmental Science and Technology*, 43 (1), pp. 20-25. (abstract only)

Weisler, Richard. (2003). Childhood Brain Cancers Near Asphalt Industry in Salisbury, NC [Presentation]. Available at http://www.bredl.org/air/ssimages/Childhoodcancers_asphalt.pdf

"CHEJ is the strongest environmental organization today – the one that is making the greatest impact on changing the way our society does business."

Ralph Nader

"CHEJ has been a pioneer nationally in alerting parents to the environmental hazards that can affect the health of their children."

New York, New York

"Again, thank you for all that you do for us out here. I would have given up a long time ago if I had not connected with CHEJ!"

Claremont, New Hampshire

Center for Health, Environment & Justice
P.O. Box 6806, Falls Church, VA 22040-6806
703-237-2249 chej@chej.org www.chej.org