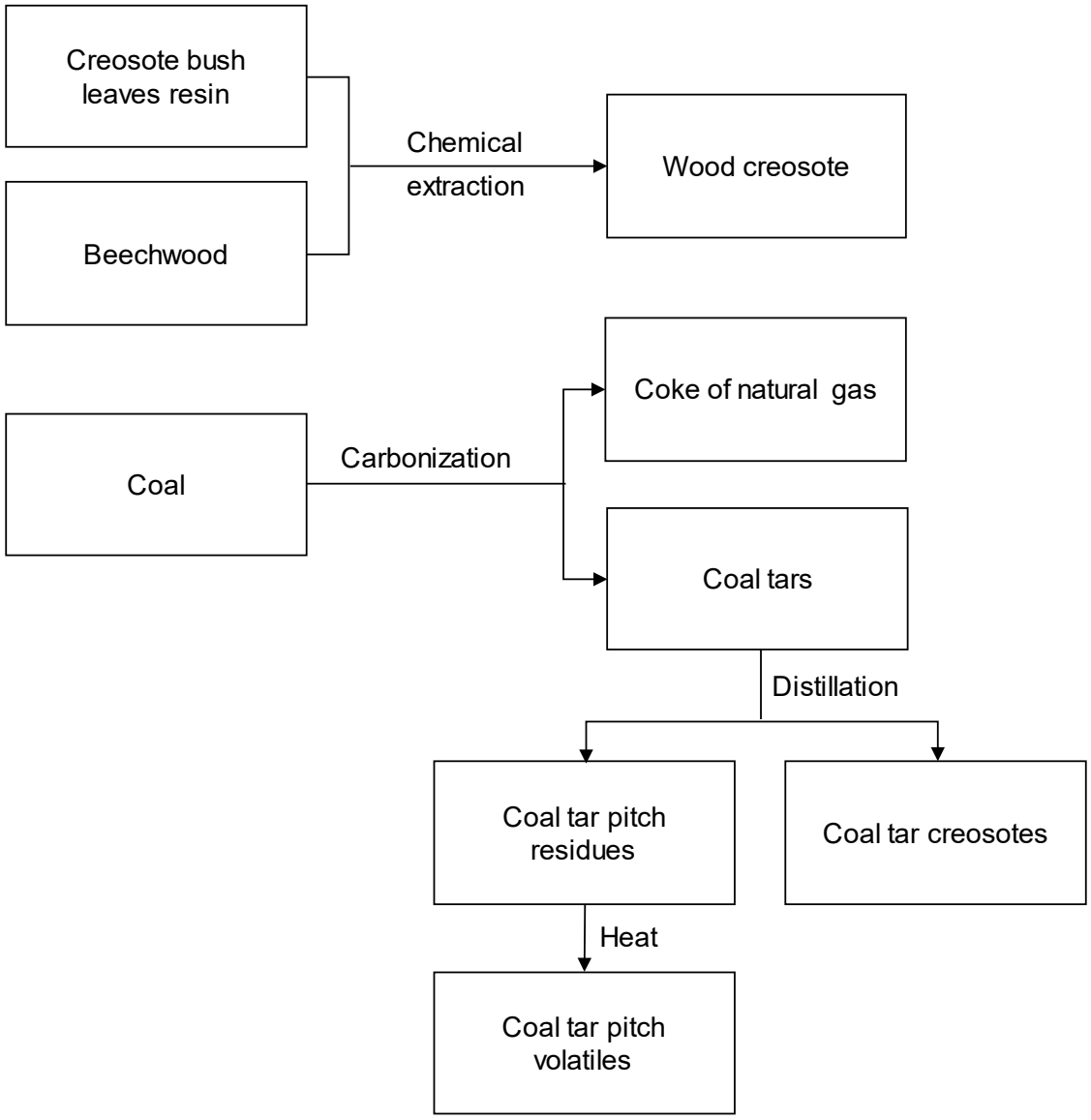
# CHAPTER 1. RELEVANCE TO PUBLIC HEALTH

## 1.1 OVERVIEW AND U.S. EXPOSURES

This profile addresses several substances: wood creosotes, coal tar creosote, coal tar, coal tar pitch, and coal tar pitch volatiles. These substances are complex mixtures of hundreds, if not thousands, of individual chemical components. For this profile, the substances were broadly divided into two categories: wood creosotes and coal tar products, which are very different complex mixtures that can vary greatly, even within the broad categories. Figure 1-1 shows how these substances are produced.

**Figure 1-1. Origin of Wood Creosotes and Coal Tar Products**



## 1. RELEVANCE TO PUBLIC HEALTH

***Wood Creosotes.*** Wood creosote is derived from fractional distillation of either beechwood (*Fagus*, a type of deciduous tree) or the resin from leaves of the creosote bush (*Larrea tridentata*). Wood creosote consists mainly of phenol, cresols, guaiacols, and xylenols. It is a colorless or pale yellowish liquid, and it has a characteristic smoky odor and burnt taste (Miyazato et al. 1981). It had therapeutic applications in the past as a disinfectant, laxative, and a stimulating expectorant, but it is not a major pharmaceutical ingredient today in the United States.

***Coal Tar Products.*** Coal tar products refers to a broad category that includes coal tar, coal tar creosote, coal tar pitch, and coal tar pitch volatiles. Coal tars are byproducts of the carbonization of coal to produce coke and/or natural gas. Coal tar creosotes are distillation products of coal tar, while coal tar pitch is a residue produced during the distillation of coal tar. Coal tar pitch volatiles are compounds given off from coal tar pitch when it is heated. Coal tar creosotes, coal tar, coal tar pitch, and coal tar pitch volatiles are composed of many individual compounds of varying physical and chemical characteristics. In addition, the composition of each, although referred to by specific name (e.g., coal tar creosote), is not consistent, and the components and properties of the mixture depend on the temperature of the destructive distillation (carbonization) and on the nature of the carbon-containing material used as a feedstock for pyrolysis. Usually, coal tars are viscous liquids or semisolids that are black or dark brown with a naphthalene-like odor. Coal tars are complex combinations of polycyclic aromatic hydrocarbons (PAHs), phenols, heterocyclic oxygen, sulfur, and nitrogen compounds. PAH composition of coal tars is variable. Analyses of PAHs in four coal tar samples revealed 2- to 20-fold differences in concentration of selected PAHs among the samples. For example, benzo[a]pyrene ranged from nondetectable levels to 1.7, 3.9, and 6.4 g/kg of coal tar. By comparison, coal tar creosotes have an oily liquid consistency and range in color from yellowish-dark green to brown. The coal tar creosotes consist of PAHs and PAH derivatives. At least 75% of the coal tar creosote mixture is PAHs. Coal tar pitch is a shiny, dark brown-to-black residue that contains PAHs and their methyl and polymethyl derivatives, as well as heteronuclear compounds. There are also over-the-counter medications and shampoos containing low-dose solutions of coal tar to treat certain skin conditions like dandruff, eczema, and seborrheic dermatitis. In the past, wood creosote was used as a disinfectant, laxative, and cough treatment, but is rarely used these in ways today in the United States.

Coal tar creosote has been used as a wood preservative pesticide in the United States for over 100 years. It is used as a fungicide, insecticide, and sporicide for above-ground and below-ground wood protection treatments, as well as for treating wood in marine environments. It is a restricted use pesticide, meaning that it is not available for purchase by the general public in the United States and may only be used by

certified pesticide applicators (EPA 2008, 2015). Since coal tar creosote is not available to the public, most exposures occur for workers employed handling or treating creosote-protected wood products. Dermal and inhalation are the most likely routes of occupational exposures.

People residing near wood treatment facilities or other facilities that produce or use coal tar and coal tar creosote may have exposure to the chemicals in these complex mixtures, particularly PAHs. Inhalation of air, dermal contact with contaminated environmental media (e.g., soil or water), and possibly ingestion of contaminated groundwater are possible exposure routes. PAHs from creosote may also be accumulated in fish and other aquatic species which may be another exposure route for humans. The public may also be exposed via dermal or inhalation routes to PAHs from the use of coal tar-based driveway sealants. There are also over-the-counter medications containing low-dose solutions of coal tar to treat certain skin conditions.

Most environmental releases of coal tar creosote arise from effluents in wood treatment facilities or accidental spills. Since these are complex mixtures of many chemicals, the environmental fate and transport is different for different components in the mixture. In general, many of the chemicals tend to adsorb to soil and sediment, which act as an environmental sink. Some components are volatile and may evaporate into air from water or soil where they can be degraded by atmospheric oxidation reactions or direct photolysis. Biodegradation tends to occur slowly for many of the components of coal tar creosote especially the high molecular weight PAHs. Components of wood creosote tend to be more volatile and less persistent than the components of coal tar and coal tar creosote.

## 1.2 SUMMARY OF HEALTH EFFECTS

The health effects of creosote and creosote compounds have been evaluated in observational occupational and population-based epidemiological studies, case reports, clinical trials, and experimental animal studies. Exposure to wood creosotes occurs mainly through intentional ingestion or dermal application of pharmaceutical products. Coal tar product exposure can occur through inhalation of coal tar aerosols or through dermal contact from industrial uses or from therapeutic applications. Associated health effects are discussed in terms of two major categories: coal tar products (coal tar creosote, coal tar, coal tar pitch, and coal tar pitch volatiles) and wood creosotes.

***Coal Tar Products.*** Exposures to coal tar and coal tar products may take place in industrial and non-industrial settings and can occur through inhalation, oral, and dermal routes of exposure. Information

regarding the adverse human health effects of coal tar creosote, coal tar, coal tar pitch, and coal tar pitch volatiles is available from occupational surveys and retrospective health studies. Unfortunately, the usefulness of many of the occupational studies is hampered by incomplete characterization of worker exposure and the difficulty in ascribing adverse effects to a particular exposure route. Additional health effects information is available from the use of coal tar products in the medical treatment of psoriasis patients. Data are also available from animal studies, although the results are not always consistent across species or sex. The available information suggests that adverse respiratory and developmental effects and increased carcinogenicity risk are the most important health concerns related to exposure to coal tar and coal tar products.

*Respiratory effects (coal tar products).* Coal tar aerosols and volatiles have been linked to adverse respiratory effects. Occupational exposure studies evaluating respiratory effects have been conducted in wood processing and wood preservative workers, electrode manufacturing workers, and aluminum industry workers. An industrial health survey conducted in wood treatment plants reported reduced lung function in 17% of the employees examined, while workers in coal tar plants reported pulmonary deficits in 33% of the workers surveyed (Koppers Company 1979, 1981). Long-term residents near a wood treatment plant reported a significant increase in the prevalence of diagnosed bronchitis and asthma by history, while residents of an area that had been built on land formerly occupied by a coal tar creosote wood treatment facility also showed an increased risk of chronic bronchitis (ATSDR 1994; Dahlgren et al. 2004). Most studies evaluating respiratory effects in animals have focused on changes in lung weight, although a few animal studies have shown histopathological changes following inhalation exposure, including lesions of the olfactory epithelium and lungs (EPA 1995c, 1995d; Springer et al. 1982, 1986b, 1987).

*Developmental effects (coal tar products).* There are no reports of adverse developmental outcomes in humans exposed to coal tar and coal tar products. Women treated with coal tar for psoriasis or dermatitis did not exhibit an increase in spontaneous abortions or congenital disorders in their offspring (Franssen et al. 1999). There were no differences in the number of pregnancies; live, premature, and still births; or spontaneous abortions among women who resided in a housing development built on contaminated land formerly occupied by a coal tar creosote wood treatment plant (ATSDR 1994). On the other hand, multiple animal studies have shown that exposure to coal tar during pregnancy may have adverse effects on the fetus. Increased post-implantation loss and whole litter resorptions, decreased fetal body and lung weight, and increased incidences of malformations including hydrocephaly, dilated ventricles, and cleft palate have been observed in studies evaluating oral and dermal exposure (EPA 1995a, 1995b; Hackett et

al. 1984; Springer et al. 1986a; Zangar et al. 1989). In many of these studies, it is not possible to exclude the potential role of maternal toxicity in the development of adverse fetal effects, but the evidence suggests that fetal effects may result from maternal exposure to coal tar products.

***Wood Creosotes.*** Exposure to wood creosotes appears to be confined to ingestion of plant extracts and dermal contact with the plants. Most of the toxicity data for oral exposure to wood creosotes come from reports of individuals who ingested plant extracts such as chaparral, an herbal extract prepared by grinding leaves of the creosote bush, or "seirogan," a Japanese folk remedy made with wood creosote that is typically taken for stomachaches. Information on adverse health effects associated with wood creosote is very limited. Isolated reports suggest that repeated exposure to chaparral is associated with adverse renal and/or liver effects; however, because of the limited amount of data, it is not possible to attribute the findings to ingestion of chaparral tea. Although the distribution of cancer cases in Japan coincided with "seirogan" production areas, an association between cancer incidence in Japan and the use of "seirogan" cannot be made with the available data. Animal studies evaluating cancer endpoints following oral exposure to wood creosotes have not identified a tumorigenic response. The available data suggest that hepatic effects are the main adverse outcomes that result from exposure to wood creosotes.

*Hepatic effects (wood creosotes).* Acute toxic hepatitis has been attributed to continued ingestion of chaparral. Case reports of intermediate-duration ingestion of chaparral have described patients with a variety of hepatic effects including icterus and jaundice (Alderman et al. 1994; CDC 1992; Gordon et al. 1995; Katz and Saibil 1990). Elevated liver enzymes have been observed, which often return to normal levels 3–6 weeks after exposure to chaparral was discontinued, and biopsies have revealed acute inflammation and other cellular changes (Alderman et al. 1994; CDC 1992; Gordon et al. 1995; Katz and Saibil 1990). In one severe case, the patient's liver biopsy showed severe acute hepatitis with areas of lobular collapse and nodular regeneration, mixed portal inflammation, and marked bile ductular proliferation (Gordon et al. 1995). In animal oral exposure studies, increased liver-to-body weight ratios and serum cholesterol have been observed (Miyazato et al. 1981, 1984a, 1984b), but the lack of associated changes in histopathology makes the toxicological significance of these changes questionable.

***Cancer.*** Studies of workers exposed to coal tar in various industrial environments have found increased cancer risk involving several tissues including the respiratory tract, skin, lung, pancreas, kidney, scrotum, prostate, rectum, bladder, and central nervous system (see Section 2.19). These adverse effects have not been observed in patients undergoing coal tar therapy. Although exposure from inhalation is likely a major factor, significant dermal exposure and possibly oral exposure may also occur in industrial settings,

making the ability to distinguish between routes of exposure difficult. Animal studies have demonstrated the carcinogenic potential of coal tar products through primarily dermal exposure but also following inhalation and oral exposure and have identified cancers of the lungs, liver, forestomach, and skin. In addition, numerous studies provide consistent evidence that exposure to coal tar is genotoxic. Studies evaluating wood creosotes have not identified similar effects in rodents.

The Department of Health and Human Services (HHS) has classified coal tars, coal-tar pitches, and coke-oven emissions to be human carcinogens based on sufficient evidence of carcinogenicity from studies in humans (NTP 2021). The U.S. Environmental Protection Agency (EPA) concluded that coke oven emissions (coal tar pitch volatiles) are a human carcinogen (Group A) based on sufficient evidence in humans and animals (IRIS 1989) and that creosote is a probable human carcinogen (Group B1) based on limited evidence in humans and sufficient evidence in animals (IRIS 1988). The International Agency for Research on Cancer (IARC) classified creosotes as probably carcinogenic to humans (Group 2A) based on limited evidence in humans and sufficient evidence in experimental animals (IARC 2010). In addition, IARC (2012a) classified the carcinogenicity of creosote compounds for specific occupational settings and cancer types. Coke production is carcinogenic to humans (Group 1) based on sufficient evidence in humans for the carcinogenicity of coke production (cancer of the lung) and sufficient evidence in experimental animals for the carcinogenicity of samples of tar taken from coke ovens. Coal gasification is carcinogenic to humans (Group 1) based on sufficient evidence in humans (cancer of the lung) and sufficient evidence in experimental animals for the carcinogenicity of samples of tar taken from coke ovens. Coal gasification is carcinogenic to humans (Group 1) based on sufficient evidence in humans (cancer of the lung) and sufficient evidence in experimental animals for the carcinogenicity of coal tars from gasworks and manufactured gas plant (MGP) residues. Aluminum production is carcinogenic to humans (Group 1) based on sufficient evidence in humans (cancers of bladder and lung) and sufficient evidence in experimental animals for the carcinogenicity of airborne particulate polynuclear organic matter from aluminum-production plants. Coal tar distillation is carcinogenic to humans (Group 1) based on sufficient evidence in humans (cancer of the skin) and sufficient evidence in experimental animals for the carcinogenicity of coal tars. Exposure to coal tar pitch in roofers and pavers is carcinogenic to humans (Group 1) based on sufficient evidence in humans (cancers of the lung and bladder) and sufficient evidence in experimental animals for the carcinogenicity of coal tar pitch (IARC 2012a).

## 1.3 MINIMAL RISK LEVELS (MRLs)

MRLs for creosote compounds have not been derived. Coal tar creosote, coal tar, coal tar pitch, and coal tar pitch volatiles and wood creosotes are extremely complex mixtures containing numerous compounds; furthermore, the compositions of the mixtures are not consistent. Even within a class of creosote compounds, the chemical mixtures vary such that adverse effects profiles and potency may vary within a class of creosote compounds. This is demonstrated by inconsistent results observed in studies evaluating the same class of compounds; a single lowest-observed-adverse-effect level (LOAEL) value may not be representative for a class of compounds. Thus, derivation of an MRL based on single study or group of studies may not be protective for other exposures. The database for creosote compounds was not considered adequate for derivation of inhalation or oral MRLs for any exposure duration (Tables 1-1 and 1-2).

**Table 1-1. Minimal Risk Levels (MRLs) for Coal Tar Products[a]**

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| **No MRLs were derived for any exposure route or duration for coal tar products.** |

[a]See Appendix A for additional information.

**Table 1-2. Minimal Risk Levels (MRLs) for Wood Creosotes[a]**

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|---|
| **No MRLs were derived for any exposure route or duration for wood creosotes.** |

[a]See Appendix A for additional information.