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Peer Review of Coal-Tar-Sealed Pavement Risk Assessment

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Tables

Figures

Attachments

Acronyms and Abbreviations

AAF	adjusted absorption factor
BaP-TE	benzo(a)pyrene toxic equivalents
CSA	coal-tar-sealed asphalt
CTE	central tendency exposure
ED	exposure duration
EFH	Exposure Factors Handbook
EPC	exposure point concentration
EPRI	Electric Power Research Institute
FDA	U.S. Food and Drug Administration
GLP	Good Laboratory Practice
g/day	grams per day
HHRA	human health risk assessment
IRIS	Integrated Risk Information System
kg	kilogram
m	meter
mg	milligram
mg/day	milligrams per day
mg/kg	milligrams per kilogram
mg/kg-day	milligrams per kilogram of body weight per day
ng	nanogram
ng/kg-day	nanograms per kilogram of body weight per day
NHANES	National Health and Nutrition Examination Survey
OSF	oral slope factor
OSWER	Office of Solid Waste and Emergency Response
OTC	over the counter
PAH	polycyclic aromatic hydrocarbon
RME	reasonable maximum exposure
SHD	settled house dust
UA	unsealed asphalt
UCL	upper confidence limit
ug	micrograms
ug/day	micrograms per day
ug/g	micrograms per gram
ug/L	micrograms per liter

UK	United Kingdom
UNH	University of New Hampshire
UNHSC	University of New Hampshire Stormwater Center
USEPA	U.S. Environmental Protection Agency
USGS	U.S. Geological Survey

1. Introduction

ARCADIS has prepared this report to summarize a peer review of the following publication related to human health risk assessment of coal-tar-sealed pavement:

> Williams, E.S., B.J. Mahler, and P.C. Van Metre. 2013. Cancer Risk from Incidental Ingestion Exposures to PAHs Associated with Coal-Tar-Sealed Pavement. *Environmental Science & Technology*. 47:1101-1109.

The Williams et al. (2013) paper asserts that the presence of coal-tar-based pavement sealants is associated with significant increases in estimated cancer risks for residents living adjacent to coal-tar-sealed paved surfaces. Our evaluation finds that no such association has been established between residents living adjacent to sealed paved surfaces, and no increases in estimated cancer risks above regulatory levels of concern have been established.

1.1 Objectives and Approach

ARCADIS' peer review, as described in detail in this report, critically evaluates the data and risk assessment methods described in the Williams et al. (2013) paper. This report also presents dosimetry information to describe multiple sources of exposure to PAHs in the environment and to provide context for the Williams et al. (2013) dose estimates.

The Williams et al. (2013) paper relies on previously-published data associated with settled house dust (SHD) in living spaces and soil adjacent to large parking lots, but not from the same locations. Samples of polycyclic aromatic hydrocarbons (PAH) in SHD were collected from 23 ground floor apartments in Austin, Texas and summarized in Mahler et al. (2010). Samples of PAHs in surface materials adjacent to parking lots were collected from 2 locations in suburban Chicago (Van Metre et al., 2008) and from 1 location in Durham, New Hampshire (UNHSC, 2010). Mean concentrations of benzo(a)pyrene toxic equivalents (BaP-TE) were calculated and used in deterministic and probabilistic dose calculations with the U.S. Environmental Protection Agency (USEPA) current oral slope factor (OSF) of 7.3 $(mg/kg/day)^{-1}$ to examine the potential human health effects of PAHs from coal-tar-based products in SHD and soil. The paper presents central tendency estimates of excess cancer risk resulting from lifetime exposures ranging from 4.0×10^{-5} to 1.1×10^{-4} and reasonable maximum risk estimates greater than 1×10^{-4} for all exposure scenarios evaluated. The paper concludes that "the use of coal-tar-based pavement sealants magnified 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".

The Williams et al. paper is a human health risk assessment (HHRA). This paper is also referred to in this peer review report as the USGS Team HHRA because Williams' co-authors work for the U.S. Geological Survey and work published by those co-authors is the source of much of the data and information about coal tar-based pavement sealers relied on in the HHRA paper. The approach used to conduct the peer review considers the USEPA (1989) paradigm for HHRA: Hazard Identification, Exposure Assessment, Toxicity

Assessment, Risk Characterization and Uncertainty Analysis, and guidance for completing HHRAs (USEPA 1989, 2011, 2012). The degree to which the methods and assumptions used in the Williams et al. HHRA conforms to the USEPA standard risk assessment methods was reviewed. The use of standardized approaches, or departures from USEPA standard approaches, was considered. Also, the effect of alternative assumptions was considered. Table 1 below highlights the factors that were critically reviewed as part of the peer review.

Table 1. Scope and Findings of Peer Review

HHRA Component	Critical Review Item	Flaws in Williams et al. (USGS Team HHRA)
Hazard Identification	• Evaluate data set from Mahler et al. (2010), Van Metre et al. (2008) and UNHSC (2010)	• Not enough data • Samples not collected properly • Selective use of data not explained • PAHs concentrations not attributable to coal-tar-sealant
Exposure Assessment	• Evaluate adequacy of data by exposure units • Evaluate exposure assumptions for deterministic risk calculations • Describe alternate risk estimates based on USEPA exposure assumptions • Describe alternate risk estimates including effect of PAH bioavailability	• Data not representative of exposure areas • Combined data from TX, IL and NH to describe exposure for an exposure point that does not exist • Did not use standard risk assessment assumptions • Did not consider bioavailability
Toxicity Assessment	• Compare risk estimates based on a range of values for benzo(a)pyrene toxicity	• Did not consider best available toxicity information
Risk Characterization	• Compare risk estimates using USEPA standard assumptions to Williams et al. risk estimates using non-standard assumptions	• Risk estimates do not characterize real exposure • Risk estimates are exaggerated
Uncertainty Analysis	• Describe the sensitive parameters and the effect on risk estimates • Describe conservative nature of HHRA and the direction of the impact of uncertainty on the risk estimates	• Uncertainty analysis describes only how risk estimates could be higher than presented in paper.

2. Critique of Williams et al. HHRA

2.1 Hazard Identification

The objective of the hazard identification component of a standard HHRA is to evaluate the adequacy and quality of available data to describe the constituents of concern related to identified sources of environmental exposures.

2.1.1 Not Enough Data

Three data sources were reportedly relied upon in the Williams et al. HHRA including:

1. A study of PAHs in house dust samples collected in Austin, Texas (Mahler et al., 2010);

2. Soil samples collected from grass-covered medians or islands about 0.5 meters from the edge of the curb at the edge of parking lots in Lake in the Hills, Illinois (suburban Chicago) by USGS (Van Metre et al., 2008); and

3. Soil samples collected near a large institutional parking lot area on the University of New Hampshire (UNH) campus in Durham, New Hampshire (UNHSC, 2010).

Williams et al. make assertions about the broad applicability of these highly localized data to populations throughout the U.S. based on very small data sets from these studies in which data were collected with varied methodologies. The data set used to estimate intakes and risks in the Williams et al. HHRA includes a small number of samples from three distinct and physically separate locations:

- 18 SHD samples from Austin, Texas [11 samples from apartments adjacent to coal-tar-sealed asphalt (CSA) parking lots and 7 samples from apartments adjacent to unsealed asphalt (UA) parking lots];

- 4 soil samples from Lake in the Hills, Illinois (2 samples next to CSA parking lots and 2 soil samples next to UA parking lots);

- 6 soil samples from Durham, New Hampshire (5 samples next to large institutional CSA parking lots and 1 sample next to a co-located unsealed parking lot).

Thus, the Williams et al. HHRA relies on a total of 10 soil samples to represent the entire U.S.

The inadequacy of the size of the data set is confirmed by consideration of USEPA guidance on how many data points are needed for risk assessment.

> "Sampling data from Superfund sites have shown that data sets with fewer than 10 samples per exposure area provide poor estimates of the mean concentration . . . while data sets with 10 to 20 samples per exposure area provide somewhat better estimates of the mean, and data sets with 20 to 30 samples provide fairly consistent estimates of the mean." (USEPA 1992)

USEPA has provided guidance on the minimum number of soil samples required per exposure area for HHRA in at least three HHRA guidance documents. In *Supplemental Guidance to Risk Assessment Guidance for Superfund (RAGS): Calculating the Concentration Term*, USEPA (1992) recommended 20 to 30 samples per exposure area. In the *Soil Screening Guidance: User's Guide*, USEPA (1996) recommended six composite samples, for each 0.5-acre exposure area, with each composite sample

made up of four individual samples. In *Best Practices for Efficient Soil Sampling Designs*, USEPA (2008a) recommended 10 to 20 samples per exposure unit. In addition to federal guidance, in a survey of state regulators conducted by the Interstate Technology and Regulatory Council (ITRC, 2008), regulators stated that between 14 and 34 samples are the minimum number of soil samples required for evaluating conditions at a residential lot.

In the Williams et al. HHRA, the greatest number of soil samples collected from any one location is 5 samples collected adjacent to a large institutional parking lot on the UNH campus, which is not a residential lot or adjacent to residences.

2.1.2 Samples Not Collected Properly

Soil samples collected by USGS were not sieved. It is unclear from the UNHSC report if those soil samples were sieved, but given that results were not reported by fraction size, it is assumed that none of the soil samples used in the Williams et al. HHRA was sieved. Soil samples collected adjacent to parking lots likely contained large pieces of sealer or sealer/pavement, according to sample descriptions, photos provided in various reports, and the lack of sieved soil samples. If the parking lot surface particles were deposited onto the surface soil that was sampled, then the materials in the soil samples were large particles that are not representative of soil exposure. USEPA (2007) has concluded that people contact soil particles less than 250 microns in size.

Soil samples collected by UNH cannot be attributed to coal tar sealed pavement. Sealed and unsealed lots are attached to each other (Figure 1). Sampling locations overlap with snow plow disposal adjacent to abutting parking lots. Mixing of snow and suspended particles in snow from multiple adjacent lots makes it impossible to link surface soil results to any one section of the large parking lot area. Soil samples collected by USGS may also subject to the same limitation in that at least one of the surface soil samples was collected in a curbed island within the parking lot boundary which was reportedly subject to snow and surface particulate mixing and disposal on the sampled surface soil. Other samples were reportedly collected less than 1 meter from the parking lot edge, a location that was likely also used for snow pile storage.

2.1.3 Selective Use of Data Not Explained

Williams et al. used 18 out of 23 SHD samples from Mahler et al. (2010), choosing to exclude 5 out of 12 samples of SHD near unsealed surfaces. The individual sample results from Mahler et al. (2010) are presented in Table A.1 in Appendix A to this report. Four of the excluded samples had Total PAH concentrations that were within the range of Total PAH concentrations for apartments near a large coal-tar-sealed asphalt parking lot. One of the excluded samples, collected from an apartment residence adjacent to a large unsealed concrete parking lot, had the highest reported Total PAH concentration for the non-coal tar sealant data set identified in Mahler et al. (2010). Excluding these higher concentration samples from the evaluation of unsealed surfaces serves to inappropriately increase the apparent difference between CSA and UA settings.

A subset of available soil samples was also selected for use in the Williams et al. HHRA but without explanation. In the UNHSC (2010) study, a total of 29 locations around a large institutional parking lot area on campus were sampled between 2009 and 2010 (Figure 2) with 21 samples analyzed by the UNH laboratory and a subset of 14 samples analyzed by a fully accredited commercial laboratory (META Environmental). The primary investigator (Alison Watts, personal communication, 2013) stated that for reliable measurements of individual PAH concentrations, only the META Environmental data should be used. Williams et al. reportedly used a total of 6 soil samples from the UNH data set (5 CSA samples and 1 UA sample). However, the Williams et al. paper fails to provide details on the samples that were selected for use in their HHRA, so it is not known if only the accredited laboratory data were used or why more than half of the available soil samples were excluded from their HHRA. It is known that among the 5 CSA soil samples included in the Williams et al. HHRA from the available 9 CSA soil samples in the UNH data set, the sample with the maximum detected BaP concentration (29.2 ug/g) and maximum BaP-TE concentration (44.4 ug/g) was used to calculate a geometric mean concentration for CSA soils. It is also known that the 1 UA soil sample included in the Williams et al. HHRA, out of a total of 5 available UA soil samples in the UNH data set, had the lowest reported BaP (0.17 ug/g) and BaP-TE concentration (0.26 ug/g). Differences in concentrations of BaP-TE between the CSA soil and UA soil are inappropriately magnified when a subset of available sample results are used. The individual sample results for CSA soil and UA soil are presented in Table A.2 and Table A.3, respectively, in Appendix A to this report.

For the Williams et al. HHRA, geometric mean concentrations of BaP-TE were calculated based on 7 CSA soil samples and 3 UA soil samples. Table 2 compares the geometric mean BaP-TE concentrations reported by Williams et al. (2013) to the geometric mean BaP-TE concentrations calculated using data from all of the soil samples from the sources cited in the Williams et al. paper [UNHSC, 2010 and Van Metre et al., 2008, a co-author on the Williams et al. paper]. A similar comparison is made using data from all of the SHD samples from the source cited in the Williams et al. paper (Mahler et al., 2010, also a co-author on the Williams et al. paper). The Williams et al. HHRA relied on geometric mean concentrations as point estimates for deterministic dose and risk calculations. The presented geometric mean concentration for CSA soil of 12.4 mg/kg is approximately double what the geometric mean concentration for CSA soil would be (5.86 mg/kg) if all identified sample results were used to calculate the geometric mean. The resulting risk estimates based on the higher geometric mean concentration are also approximately doubled.

Table 2. Comparison of Geometric Mean BaP-TE Concentrations from Selected Data Sets				
	BaP-TE (ug/g) Coal Tar Sealed		**BaP-TE (ug/g) Unsealed**	
Soil concentration reported by Williams et al. (2013)	12.4	(*n* = 7)	0.19	(*n* = 3)
Soil concentration calculated using all soil samples from Van Metre et al., 2008 and UNHSC, 2010	5.86	(*n* = 11)	1.24	(*n* = 7)
SHD concentration reported by Williams et al. (2013)	8.1	(*n* = 11)	0.61	(*n* = 7)
SHD concentration calculated using all SHD samples from Mahler et al., 2010	7.9	(*n* = 11)	0.87	(*n* = 12)
Notes: BaP-TE = benzo(a)pyrene toxic equivalent concentration ug/g = microgram per gram *n* = number of samples SHD = settled house dust				

2.1.4 PAH Concentrations Not Attributable to Coal-Tar-Sealant

While PAHs are constituents in coal tar products, the Williams et al. HHRA did not convincingly make the case that the PAHs measured in SHD and/or soil was in any way caused by the release of PAHs from coal tar pavement sealants. The soil samples used in the HHRA were not co-located with residences where SHD samples were collected. In the UNHSC study, the concentrations of PAHs in parking lot sweepings samples were higher on the unsealed area of the parking lot than the sealed area of the parking lot and given the UNHSC-postulated movement of sweepings onto adjacent surface soils via snow plows and snow disposal on the edge of abutting sealed and unsealed parking lot areas, no attribution of measured soil PAH concentrations to a particular sealed or unsealed portion of the large parking lot can be made. The sealed and unsealed areas of pavement were also vastly different. As noted in Figure 1, the coal tar sealed portion of the large co-located institutional parking lot was only 6% of the total surface area of the parking lot. Also, no background PAH soil sampling was performed before the test area of the parking lot was sealed with coal tar sealant products.

The proper design of a study with a goal to differentiate the PAH concentrations in soils adjacent to sealed or unsealed parking lots would require the location of the two parking lots with enough distance between them that wind erosion, surface water runoff, tracking, sweeping, or snow plow action on the test parking lot would not affect the soil adjacent to the control parking lot. In addition, soil would be tested before and after pavement sealing at identical locations.

2.2 Exposure Assessment

The objective of the exposure assessment component of a standard HHRA is to identify potential pathways of human exposure to constituents in the environment and to estimate the magnitude of that exposure to an individual at a specific location.

2.2.1 Data Not Representative of Exposure Areas

Soil samples were collected adjacent to large commercial or institutional parking lots. Children routinely play in residential yards and playgrounds, not at the edge of commercial or institutional parking lots. USGS and UNHSC soil samples were not taken from locations that are exposure points. This point is well recognized by USEPA and state regulators. In their guidance for background soil sampling for PAHs, regulatory agencies disallow PAH soil sampling anywhere near pavement because: (a) such locations are not exposure points and (b) high levels of PAHs are known to be present adjacent to pavement because of runoff of oils, vehicle exhaust, tire wear, etc. Thus, soil samples collected near pavement are not relevant for risk assessment. In fact, USGS research shows that PAH levels are much lower adjacent to residential driveways than adjacent to commercial parking lots. In a USGS report, Steuer et al. (1997) show that PAHs are highest from commercial parking lots compared to other sources. Residential driveways are much lower and residential lawns are lower still (Table 3).

Table 3. PAH Concentrations from Commercial Parking Lots Higher than from Residential Land Uses

Locations	Total PAH in runoff (ug/L)	Benzo(a)Pyrene in runoff (ug/L)
Parking lots	76	4
Residential driveways	2	0.3
Residential lawns	Not detected (<0.002)	Not detected (<0.002)

2.2.1.1 *PAHs in Settled House Dust*

Concentrations of total potentially carcinogenic PAHs in house dust from sources other than coal-tar-based sealants were obtained for locations throughout the U.S. (Table 4) and compared to the indoor dust dataset from Mahler et al. (2010), which includes only 23 samples, and those PAH concentrations are 10-fold higher than the more comprehensive dataset of Whitehead et al. (2011), who summarized PAH levels in house dust from 583 households in California.

Table 4. Total Potentially Carcinogenic PAH Concentrations in Settled House Dust (ug/g)			
Research Study	**Total Potentially Carcinogenic PAH Concentration**		**Number of Samples**
Whitehead et al., 2011 San Francisco Bay and California Central Valley	0.304	median total (range 0.003-2.45)	583
Mukerjee et al.,1997 Lower Rio Grande Valley, TX	0.674	median total (summer sample period)	6
Mukerjee et al., 1997 Lower Rio Grande Valley, TX	0.866	median total (spring sample period)	9
Chuang et al., 1999 Durham, NC	1.73	average total	Unknown
Lewis et al., 1999 Research Triangle, NC (USEPA)	2.21	average total	Composite sample separated into 7 fractions
Lewis et al., 1999 (USEPA) NC, MD, OH, NJ	7.63	average total; SRM 2583 (NIST indoor dust standard reference material)	Composite sample separated into 7 fractions
USEPA, 1994a Seattle, WA	11	average total	Unknown
Maertens et al., 2004 Varies (analysis of 18 published studies; primarily NC based locations)	11.67	average total (range 0.14-268)	126
Mahler et al., 2010 Austin, TX	12.5	average total (range 0.98-85.8); Unsealed Lot	12
Mahler et al., 2010 Austin, TX	57.5	average total (range 8.62-156); Sealed Lot	11
Chuang et al., 1995; USEPA, 1994b Columbus, OH	72	average total	Unknown

2.2.1.2 *PAHs in Soil*

Concentrations of BaP-TE in background soil samples were obtained from studies performed in the eastern U.S. (Table 5) and compared to the soil dataset from USGS (Van Metre et al., 2008) and UNHSC (2010). The concentration of BaP-TE for soil samples collected adjacent to an unsealed parking lot in New Hampshire is the highest value tabulated. If the sample locations were truly reflective of background conditions, the resulting concentrations would be equal to or less than the other sampled locations listed below. The BaP-TE concentration for soil samples collected adjacent to sealed parking lots in New Hampshire (UNHSC, 2010) is also the highest value tabulated and was almost double the BaP-TE concentration for the CSA soil samples collected in Illinois (Van Metre et al., 2008). The results from the UNHSC study are clearly skewed toward higher concentrations and are not representative of either background conditions or conditions presumed to be affected by coal tar sealant use.

Table 5. Typical Urban Background Levels of PAHs in Soil as BaP-TE (ug/g)		
Published Study		**Type of Statistic**
Bradley et al. (1994) New England	3.3	95% UCL on the mean
MADEP (2002) Massachusetts	3	Concentration in "natural" soil; no statistic given
Chicago (USGS 2003 individual data) Used by IEPA (2007)	4.3	95% UCL on the mean
Illinois Metro Areas (EPRI 2004 individual data) Used by IEPA (2007)	1.7	95% UCL on the mean
EPRI (2003) Western New York	1.78	Sum of 95% UCLs of individual PAHs converted to BaP-TE
EPRI (2003) Western New York	1.82	95% UCL on the mean of individual sample BaP-TE values
EPRI (2008) Urban Soil	0.9	Sum of 95% UCLs of individual PAHs converted to BaP-TE (individual data not available)
USGS (Van Metre et al., 2008) Lake in the Hills, IL	1	BaP-TE for individual sample near unsealed lot
USGS (Van Metre et al., 2008) Lake in the Hills, IL	3.9	BaP-TE for individual sample near sealed lot
USGS (Van Metre et al., 2008) Lake in the Hills, IL	18	BaP-TE for individual sample near sealed lot
UNHSC (2010) Durham, NH	5.2	95% UCL on the mean of individual sample BaP-TE values for unsealed lots
UNHSC (2010) Durham, NH	35	95% UCL on the mean of individual sample BaP-TE values for sealed lots

2.2.2 No Such Exposure Point Exists

Williams et al. take soil data adjacent to two parking lots in Illinois and combine it with soil data adjacent to a parking area at one location in New Hampshire to estimate exposure to PAHs in soil for a hypothetical U.S. resident. Standard risk assessment practice requires samples to be collected for each location of potential exposure ("exposure point"). Data from multiple non-contiguous sampling locations cannot be mixed to describe an exposure point for an individual. Combining data on SHD in Texas, soil in Illinois and soil in New Hampshire does not represent actual exposure for any one person nor does it represent exposure to the U.S. population.

2.2.3 Did Not Use Standard Risk Assessment Assumptions

The exposure assumptions used in the Williams et al. HHRA (Table 6) were compared to the standard default assumptions recommended by USEPA (1991a, 1997, 2011, 2012). The distribution of exposure assumptions used in the risk calculations were evaluated for representativeness and sources of bias in the selected range of exposure assumption values.

Table 6. Comparison of Williams et al. (2013) Exposure Assumptions to Standard USEPA HHRA Assumptions

	USGS Team HHRA	Standard USEPA HHRA
Soil ingestion rate for children (0-6 years)	400 mg/day	200 mg/day* EFH(USEPA, 2011)
Soil + Dust ingestion rate for children (0-6 years)	500 mg/day	200 mg/day EFH (USEPA, 2011)
Soil ingestion rate for children (7-13 years)	400 mg/day	100 mg/day EFH (USEPA, 2011)
Soil exposure frequency	365 days/year	350 days/year (USEPA, 1991a, 2012)
Exposure duration for residents	70 years	30 years (USEPA, 1991a, 2012)

Notes:
EFH = Exposure Factors Handbook
*Accounts for ingestion of both outdoor soil and indoor dust and is an upper bound value.

Risk assessments are supposed to evaluate "reasonable" exposures. Soil ingestion was evaluated. The intake of constituents from incidental soil ingestion is related to the amount of material ingested. Children may ingest soil that adheres to their hands during play. Adults may also ingest soil particles that adhere to food or their hands during normal activities. As a result, individuals may incidentally ingest surface soil that they contact.

The child soil ingestion rate for the "reasonable maximum exposure (RME)" case in the Williams et al. HHRA is double what USEPA risk assessments use. The recommended 200 mg/day soil ingestion rate for a child age 0 to 6 years has been in place for more than 20 years (USEPA, 1991a) and continues to be used in recent USEPA publications (2011, 2012). The USEPA (2011) Exposure Factors Handbook (EFH) identifies the 200 mg/day rate for soil plus dust ingestion. Williams et al. assumes an additional 100 mg/day dust ingestion for children in their RME case, effectively using a soil plus dust ingestion rate of 500 mg/day which is 2.5 times higher than the USEPA-recommended value for this exposure parameter. In another departure from USEPA-recommended values, the Williams et al. HHRA assumes the 400 mg/day soil ingestion rate for 0 to 6 year olds applies to older children from age 7 to 13 years. The USEPA-recommended soil ingestion rate for 7 to 13 year olds is 100 mg/day for the RME case. The Williams et al. risk estimates for this age range are overstated by a factor of 4.

Assuming an exposure frequency of either 350 or 365 days per year for the RME case is an intentional overestimate. Children and adults are not expected to play at the edge of commercial parking lots 365 days

per year. While standard USEPA risk assessments may use upper bound values for the RME case, often the exposure frequency is reduced for the central tendency exposure (CTE) case. The RME scenario is intended to represent the "highest exposure that is reasonably expected to occur at a site" (USEPA, 1989) and the CTE scenario is intended to represent more likely exposures associated with more common or typical rates of contact. In most cases, the values chosen for the CTE scenario represent an average exposure level, while the RME value represents the 90th, 95th or other higher end measure of exposure. Williams et al. assumed exposure would occur every day of a 70-year lifetime for both the RME and CTE cases. The USEPA standard exposure duration for evaluating residential exposures is 30 years, not 70 years. The standard USEPA approach relies on an exposure duration (ED) of nine years (CTE) or 24 years (RME) to represent hypothetical residential exposure for the adult in conjunction with an ED of six years for hypothetical child residents, which together represents the 50th and 90th percentile values of residential tenure for the U.S. population.

2.2.4 Did Not Consider Bioavailability

An additional factor to consider in the calculation of theoretical exposure doses of PAHs is bioavailability. The Williams et al. HHRA assumed that the bioavailability of BaP-TE was 100%. Williams et al. (2013) assume 100% bioavailability of PAHs in soil and SHD based on a citation of bioavailability percentages for PAHs published by Ramesh et al. (2004). The values summarized by Ramesh et al. (2004) are absolute bioavailability percentages for a variety dosing media including emulsified suspensions, oil suspensions, diet, and spiked soil samples. The range of absolute bioavailability percentages ranged from 5.5% to 102%. Williams et al. (2013) assume that 100% bioavailability reflects the availability of PAHs incidentally ingested in soil and dust matrices. PAH from ingested soil are not 100% absorbed. PAHs are bound to soil and other matrices, such as pieces of asphalt pavement.

Ramesh et al. (2004) also discusses the concept of relative bioavailability using the terminology of adjusted absorption factor (AAF). The authors stated that in a case of evaluating the relative bioavailability of a PAH mixture in soil from manufactured gas plant site resulted in a relative bioavailability percentage (AAF) of 29% when comparing the absolute availability of PAHs in site soil to that of a reference medium of PAHs in diet. Williams et al. (2013) made no attempt to determine the relevance of relative bioavailability of PAHs in an exposure medium that includes a coal tar matrix and supporting soil/dust structure that would reduce the absorption of PAHs in the human digestive tract. The literature for absolute absorption of PAHs from coal tar media, as well as coal tar media combined with soil and dust, supports the use of a 31% bioavailability factor.

Magee et al. (1999) tested PAH bioavailability in animals fed coal tar in soil and the resulting bioavailability was 18%. Others have found similar results. The average of 27 values from six bioavailability studies is 31% (Bordelon et al., 2000; Goon et al., 1991; Gron et al., 2007; Koganti et al., 1998; Magee et al., 1999; Weyand et al., 1996).

2.3 Toxicity Assessment

To assess potential carcinogenic effects, USEPA has derived oral slope factors for chemicals that are regulated as carcinogens. OSFs are derived from dose-related, statistically significant increases in tumor incidence in an exposed population relative to the incidence of tumors observed in an unexposed population. These dose-related incidence rates are usually determined in a laboratory study using rats and/or mice. OSFs are typically developed based on oral toxicity studies and are expressed in terms of a risk per a measure of oral dose, in units of $(mg/kg\text{-}day)^{-1}$. The OSFs are used to quantify an incremental cancer risk associated with ingestion exposures.

The OSF of 7.3 $(mg/kg\text{-}day)^{-1}$ is based on the geometric mean of four oral slope factors obtained from the following two rodent studies: Neal and Rigdon (1967) and Brune et al*.* (1981). The utility of rodent forestomach data for quantitative human cancer risk assessment has been questioned because humans have no forestomach. While rodent forestomach and human esophagus tissues are related, there are substantial physiological differences in these tissues (e.g., protection from mucus secretions, pH, retention and contact with food). Because the rodent forestomach does not represent any human tissue, tumor data from other sites should be given greater weight in dose-response modeling. An alternative oral slope factor for BaP from the more recent, guideline-compliant study on BaP (Culp et al*.,* 1998) based on esophagus tumors in addition to forestomach tumors is 1.2 $(mg/kg\text{-}day)^{-1}$.

Issues with the Neal and Rigdon (1967) study include:

- Not done using Good Laboratory Practices (GLP);
- Animals varied from 18 to 101 days old;
- Exposure duration varied from 70-197 days;
- Age at sacrifice varied from 88 -219 days; and
- Study not appropriate for dose-response assessment.

In the 1990's, the Electric Power Research Institute (EPRI) sponsored a study of two manufactured gas plant coal tar samples plus BaP as a positive control. USEPA co-designed and approved the study plan which was a two-year cancer bioassay in the sensitive B6C3F1 mouse. The study was GLP-compliant and was performed at the U.S. Food and Drug Administration's (FDA's) National Center for Toxicological Research and completed in 1998. USEPA is aware that the study is a Gold Standard study and that the current OSF in USEPA's Integrated Risk Information System (IRIS) is outdated.

2.3.1 Alternative Risk Estimates Based on Updated Toxicity Information

The current USEPA IRIS OSF for BaP is 7.3 $(mg/kg\text{-}day)^{-1}$ (USEPA, 2013). Recently, USEPA has reported that the OSF for BaP will be lower than the current value. The expected new OSF may drop to 1.2 or even 0.2 $(mg/kg\text{-}day)^{-1}$. A range of alternate OSF values (Table 7), including 0.2 and 1.2 $(mg/kg\text{-}day)^{-1}$, were used to present updated cancer risk calculations for comparison to the Williams et al. (2013) risk estimates (Figure 3).

Table 7. Range of Oral Slope Factors for BaP

Oral Slope Factors $(mg/kg\text{-}day)^{-1}$	Sources
7.3 (outdated)	Neal and Rigdon (1967); Brune et al. (1981)
0.2	Culp et al. (1998)
1.2	Culp et al. (1998)
0.3	Kroese et al. (2001)
0.2	Kroese et al. (2001)

2.4 Risk Characterization

The risk characterization integrates the results of the hazard identification, exposure assessment and toxicity assessment to evaluate potential risks associated with presumed exposure to PAHs in SHD and soil.

2.4.1 Risk Estimates Do Not Characterize Real Exposure

Adding risks from dust in Texas, soil in Illinois and soil in New Hampshire does not represent actual exposure for anyone. It also does not describe risk to the U.S. population. Given that data were not collected at each exposure unit, it is inappropriate to sum risks by adding risks for dust in Texas, soil in Illinois and soil in New Hampshire.

2.4.2 Risk Estimates Are Exaggerated

- The Williams et al. risk estimates are dominated by soil exposures (approximately 80% for scenarios adjacent to CSA lots) and soil data are flawed and not representative of residential exposures.

- Selected data overstates the risk estimates for soil ingestion near sealed surfaces and use of the full data set would decrease soil risk estimates by approximately 53% if all sealed UNH and USGS samples were used.

- The soil ingestion rate used in the Williams et al. HHRA is double the standard USEPA rate.

- Williams et al. incorrectly double-counted dust ingestion exposures when summing soil and dust risks by failing to account for the ingestion rate that already includes dust exposure.

- The Williams et al. HHRA assumed 100% bioavailability.

Table 8 presents a comparison of the highest reported risk estimate from the Williams et al. HHRA (Scenario 2) to the risk estimates that would be calculated using: (1) corrected BaP-TE concentrations with standard USEPA exposure assumptions; (2) corrected BaP-TE concentrations with standard USEPA exposure assumptions plus 31% bioavailability; (3) corrected BaP-TE concentrations with standard USEPA exposure assumptions plus 31% bioavailability over the range of OSF values for BaP-TE. A chart comparing the results of these calculations is provided in Figure 4.

Table 8. Comparison of Risk Estimates

Scenario		Estimated Lifetime Risk
0	Williams et al. (2013) Scenario 2	$5x10^{-4}$
1	Revised estimates (EPC + USEPA exposure assumptions)	$1x10^{-4}$
2	Revised estimates (EPC + USEPA exposure assumptions + Bioavailability)	$3x10^{-5}$
3	Revised estimates (EPC + USEPA exposure assumptions + Bioavailability + Updated Toxicity OSF)	$9x10^{-7}$ to $5x10^{-6}$

Notes:
EPC = exposure point concentration
EPC for BaP-TE of 5.8 ug/g for CSA soils and 1.24 ug/g in SHD used in Scenarios 1, 2 and 3.
Risk estimates rounded to one significant figure.

The USEPA has established a range of incremental cancer risks of 1×10^{-4} to 1×10^{-6} as a "target range within which the Agency strives to manage risks as part of a Superfund cleanup" (USEPA 1991b). The National Contingency Plan states that "for known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper-bound lifetime cancer risk to an individual of between 1×10^{-4} to 1×10^{-6} (USEPA 2003).

Only the Williams et al. risk estimates exceed the upper end of the USEPA target risk of 1×10^{-4}. Risk estimates based on inclusion of all available data and use of USEPA standard assumptions do not exceed the USEPA target risk range.

2.5 Uncertainty Analysis

All risk assessments are subject to uncertainty in data, exposure, and toxicity. However, Williams et al. describe in their paper, how the risk estimates could be higher than presented, not lower.

As demonstrated in this report, there are many parameter values in the calculations that should be changed to comply with USEPA recommendations. Use of these recommended values would produce lower risk estimates than presented in Williams et al. (2013).

Assumptions about body weight used in the Williams et al. HHRA were consistent with the EFH (USEPA 2011). The corrected risk estimates calculated in this report could be even lower than listed in Table 8 if the

higher adult body weight used by Williams et al. (79.7 kg) was also used in the calculations rather than the USEPA default value of 70 kg.

3. Typical Exposures to PAHs

The study variables described above should be considered in a broader context than that expressed by Williams et al. (2013), to recognize the multiple sources of exposure to PAHs in the environment. To put the Williams et al. (2013) results into context, ARCADIS gathered dosimetry information from other sources, such as food, background ambient air, indoor air, cigarette smoke, coal tar shampoo, and coal tar pharmaceuticals.

PAHs are measurable in air from power plant emissions, vehicle emissions, fireplaces, wood burning stoves, industrial emissions, cigarettes, and all combustion sources. PAHs are also present in food from deposition onto farms, cooking of food, and smoking of food. Other sources of exposures to PAHs include use of consumer products including shampoos, ointments, medications, protective paints, protective coatings, fuels, and lubricating oils. USEPA states that the major exposure to PAHs is from consumption of food, especially broiled or smoked food. In comparison, exposure to PAHs in soil and dust are less significant.

3.1 PAHs in Food

It is well known that PAHs are in foods, and that ingestion of food is a major source of PAH exposure to the general population. For instance, the World Health Organization (IARC, 2010) reports: “Food is a major source of intake of PAHs for the general population. Estimates of PAH intake from food vary widely, ranging from a few nanograms to a few micrograms per person per day. Sources of PAHs in the diet include barbecued/grilled/broiled and smoke-cured meats; roasted, baked and fried foods (high temperature heat processing); breads, cereals and grains (at least in part from gas/flame drying of grains); and vegetables grown in contaminated soils or with surface contamination from atmospheric fall-out of PAHs…”

IARC (2010) also states: “…it is clear that dietary intake is the major route of exposure to PAHs for a large proportion of the nonsmoking, non-occupationally exposed population…”

Phillips et al. (1999) also state: “It is clear that diet contributes substantially to nonoccupational exposure to PAHs. For nonsmokers, more than 70% of exposure is attributable to diet.”

Ramesh et al. (2004) concluded that “dietary intake of PAHs constitutes a major source of exposure in humans.”

Butler et al. (1993) concluded from their study that “…food ingestion was clearly the predominant exposure pathway” for BaP.

USEPA (2008b) in a fact sheet entitled *Polycyclic Aromatic Hydrocarbons (PAHs)* also concludes that the diet is a major exposure route for PAH exposures. They state: “Most exposures to PAHs happen every day

at very low levels in the air we breathe and the foods we eat." In another fact sheet entitled *Technical Factsheet on: Polycyclic Aromatic Hydrocarbons (PAHs)*, USEPA (undated) states: "Human exposure will be from inhalation of contaminated air and consumption of contaminated food and water. Especially high exposure will occur through the smoking of cigarettes and the ingestion of certain foods (e.g. smoked and charcoal broiled meats and fish)."

The European Commission (2002) in their *Opinion of the Scientific Committee on Food on the risks to human health of Polycyclic Aromatic Hydrocarbons in food* has also concluded that the diet is the major source of exposure to PAHs in nonsmoking individuals. Specifically, they state: "For non-smoking humans, food is the main source of exposure to PAH. In cigarette smokers, the contributions from smoking and food may be of a similar magnitude."

All regulatory authorities acknowledge that the diet is a major if not *the* major source of exposure to PAHs. Despite this fact, Williams et al. (2013) state that soil exposure is more important than dietary exposure when assessing the total risks of PAH exposures in the general population. ARCADIS has compiled (Table 8) the daily intake of BaP-TE. In many cases, scientific studies have reported the BaP daily intake of the studied population but do not report the individual PAHs that USEPA considers potentially carcinogenic. In these cases, the intake for BaP alone is reported. Obviously, the true intake posed by ingestion of BaP-TE is underestimated in such cases.

Table 9. Daily Intake of BaP and BaP-TE from Diet

Source	Daily Intake (ug/day)	Notes
Daily Intake BaP Only		
Kazerouni et al. (2001)	0.05 (BaP only) average 0.09 (BaP only) 95th percentile	228 subjects in Washington, D.C. 2000
Butler et al. (1993)	0.14 (BaP only)	15 subjects from Phillipsburg, NY 1988
Ibáñez et al. (2005)	0.14 (BaP only)	40,690 subjects from 5 regions of Spain
Daily Intake BaP-TE		
De Vos et al. (1990)	0.41	Market basket, Netherlands, 1984-1986
Falcó et al. (2003) 1.	0.248	Market basket study in seven sites in Catalonia, Spain, 2000-2002. Intake for male adults
EFSA (2008)	0.374 average 0.620 high end	Typical intake for 16 EU countries using ratio from Table 7 of 1.72/1.08 to pro-rate BaP to BaP-TE
Dennis et al. (1983)	0.321	Total diet samples from 5 colleges in the UK, 1979
Lodovici et al. (1995)	0.196	Market basket study in Milan, Italy 1985-1988, 560 adults.
Forsberg et al. (2012)	0.087 low consumption 5.199 high consumption	Average of 4 types of native American smoked salmon (5 g/day or 300 g/day)
Single Item Intake, BaP Only		
Kazerouni et al. (2001)	0.215 (BaP only)	Consumption of 1 well done grilled/barbequed hamburger (85 g) per day
Kazerouni et al. (2001)	0.024 (BaP only)	Average consumption of 1 well done grilled/barbequed hamburger (85 g) assuming it is eaten once per week for 55 years

Table 9. Daily Intake of BaP and BaP-TE from Diet		
Source	**Daily Intake (ug/day)**	**Notes**
Kazerouni et al. (2001)	0.091 (BaP only)	Consumption of 1 well done grilled/barbequed steak (112g) per day
Kazerouni et al. (2001)	0.010 (BaP only)	Average consumption of 1 well done grilled/barbequed steak (112g) assuming it is eaten once per week for 55 years
Single Item Intake, BaP-TE		
Knize et al. (1999)	0.812	Consumption of 1 propane grilled hamburger (85 g) per day
Knize et al. (1999)	0.091	Average consumption of 1 propane grilled hamburger (85 g) assuming it is eaten once per week for 55 years
Knize et al. (1999)	0.112	Consumption of 1 charcoal grilled hamburger (85 g) per day
Knize et al. (1999)	0.0125	Average consumption of 1 charcoal grilled hamburger (85 g) assuming it is eaten once per week for 55 years
Larsson et al. (1983)	5.9	Consumption of 1 log grilled hot dog (85 g) per day
Larsson et al. (1983)	0.66	Average consumption of 1 log grilled hot dog (85 g) assuming it is eaten once per week for 55 years
Larsson et al. (1983)	0.89	Consumption of 1 log ember grilled hot dog (85 g) per day
Larsson et al. (1983)	0.100	Average consumption of 1 log ember grilled hot dog (85 g) assuming it is eaten once per week for 55 years

The literature summarized in Table 9 is discussed below.

3.1.1 BaP Intake from Food

Daily intake is reported for BaP alone for three studies. The BaP daily intake ranges from 0.05 ug/day to 0.14 ug/day.

Kazerouni et al. (2001) studied the intake rates of various food items of 228 subjects in the Washington, D.C. area. BaP levels in various foods were determined from the Second National Health and Nutrition Examination Survey (NHANES II). The most common foods consumed by the general population were obtained and analyzed. Meat samples were prepared by different methods. Other food items were purchased at supermarkets.

Butler et al. (1993) studied the food intake patterns of 15 subjects from Phillipsburg, NY. Food was analyzed by the researchers.

Ibáñez et al. (2005) studied the food intake of 40,690 subjects from five regions of Spain. These data were linked to BaP content of different foods and food groups. BaP concentrations in food were taken from the

''Food Content of Potential Carcinogens'' database. This database included information on the BaP content for 313 food items reported in 26 publications from 13 different countries.

3.1.2 BaP-TE Intake from Food

Daily intake and excess lifetime cancer risk are reported for BaP-TE for six studies. The BaP-TE daily intake ranges from 0.1 ug/day to 5.9 ug/day.

De Vos et al. (1990) performed a PAH sampling study of 221 different food items from a typical market basket of 18-year-old males in the Netherlands. The sampling was performed every three months over a period of 2.5 years, resulting in ten sample sets.

Falcó et al. (2003) evaluated the PAH intake rates for children, adolescents, male adults, female adults, and seniors living in Catalonia. The PAH concentrations were analyzed for food samples randomly obtained from local markets, big supermarkets, and grocery stores in seven cities in the year 2000.

The European Food Safety Authority (EFSA 2008) analyzed PAHs in 9,714 samples of food in 33 food categories/subcategories. PAH intake rates were calculated based on the median value of the mean consumption rates for each food category as reported by the Member States. The authors note that high consumption of certain home barbecued foods would cause the typical PAH intake rate to exceed the values presented in the report.

Dennis et al. (1983) analyzed total UK diet samples from five colleges in the UK for PAH in 1979. The BaP-TE intake rate was calculated from the weight of each food group consumed per person in the UK.

Lodovici et al. (1995) measured the PAH content in Italian foods from many different foods collected from a market basket study in Milan. During the period 1985-1988, a food consumption survey was performed for 560 adults. The BaP-TE daily intake was calculated from the BaP-TE content of various foods and the consumption rate for each food.

Forsberg et al. (2012) collected smoked salmon samples from four Native American traditional smoking methods. Two methods each with two wood types were studies. PAHs were analyzed. CUTIR members reported that fish consumption rated from low (<100 g/day), moderate (100-454 g/day) to high (454-1000 g/day). The fraction of consumed fish that was smoked ranged from 5 to 50%. Accordingly, the authors estimated daily intake of BaP-TE assuming 5 g/day and 300 g/day of smoked fish.

Data from specific high PAH food items were reported from three studies summarized on Table 9. Using the data from Kazerouni et al. (2001), the daily intake of BaP from ingesting one well done grilled/barbequed hamburger was 0.22 ug/day and the daily intake risk from ingesting one well done grilled/barbequed steak was 0.09 ug/day. A more realistic average daily intake estimate would result from assuming that a person ingests one hamburger or steak a week for 55 years from age 15 to age 70. The estimated lifetime average daily dose for this scenario is 0.02 ug/day for the hamburger, and 0.01 ug/day for the steak.

Similar daily intake measurements are available from studies by Knize et al. (1999) and Larsson et al. (1983). The daily dose from a single propane grilled hamburger is 0.81 ug/day and from a charcoal grilled hamburger is 0.11 ug/day. If it is assumed that a person eats one hamburger a week for 55 years, the average daily intake is 0.09 and 0.01 ug/day, respectively. The daily intake from a single log grilled hot dog is 5.9 ug/day and a log ember grilled hot is 0.89 ug/day. If it is assumed that a person eats one hot dog a week for 55 years, the average daily intake is 0.66 and 0.10 ug/day, respectively.

Most of the BaP-TE daily intake rates from eating a full diet are in the range of 0.2 to 0.6 ug/day. The EFSA (2008) concluded that the average BaP-TE daily intake for all Europeans is 0.4 ug/day. Many people, however, consume BaP-TE at daily levels of 0.6 ug/day.

3.2 PAHs in Air

While food is a major source of PAH exposure and risk to the general population, indoor and outdoor air is also a significant source of exposure. Table 10 shows the BaP or BaP-TE concentrations in indoor or outdoor air from a variety of published studies. Estimated daily intakes were calculated assuming USEPA's standard inhalation rate of 20 m^3/day. Daily intake of BaP-TE ranges from 0.003 ug/day to 2 ug/day for indoor and outdoor air studies. Most of the daily intakes are in the range of 0.006 to 0.02 which indicates that dietary exposures are far higher than air exposures to indoor or outdoor air.

Table 10. Daily Intake of BaP-TE from Air

Sources	BaP or BaP-TE Concentration (ug/m^3)	Daily BaP or BaP-TE Intake (ug/day)	Notes
USEPA (1982)	0.001 to 0.100	0.02 to 2.0	Data from 1980 report
Butler et al. (1993)	0.0060	0.12	15 subjects from Phillipsburg, NY 1988
Sawicki et al. (1962)	0.002 to 0.03	0.040 to 0.60	1958-1959, 10 US cities
IADN (2007)	0.00131	0.026	1996-2003, Chicago
CARB (1994)	0.0007 (BaP only)	0.014	Indoor, Riverside CA, 125 homes, 1990
CARB (1994)	0.0003 (BaP only)	0.0060	Outdoor, Riverside CA, 125 homes, 1990
Chuang et al. (1991)	0.00064 (kitchen) 0.00118 (living room)	0.013 0.024	Indoor, Columbus, OH, 8 homes, 1986-7
Chuang et al. (1991)	0.00031	0.0062	Outdoor, Columbus, OH, 8 homes, 1986-7
Li et al. (2005) (NUATRC)	0.00029	0.0058	Indoor, Chicago, IL, 10 homes, 2000-1
Li et al. (2005) (NUATRC)	0.00061	0.012	Outdoor, Chicago, IL, 10 homes, 2000-1
Aquilina et al. (2010)	0.00026 (all) 0.00034 (smoking) 0.00024 (nonsmoking)	0.0052 0.0048 0.0068	Indoor air, 100 adults in UK, 2005-7
Mitra and Ray	0.00135 (smokers)	0.027	Indoor, Columbus, OH, 8 homes,

Table 10. Daily Intake of BaP-TE from Air			
Sources	**BaP or BaP-TE Concentration (ug/m^3)**	**Daily BaP or BaP-TE Intake (ug/day)**	**Notes**
(1995)	0.00068 (nonsmokers)	0.014	1986-7
Mitra and Ray (1995)	0.00031 (smokers) 0.00050 (nonsmokers)	0.0062 0.010	Outdoor, Columbus, OH, 8 homes, 1986-7
Northcross et al. (2012)	0.00767	0.006	Inside car with smoker for 1 hour (3 cigarettes smoked over 1 hour)
Slezakova et al. (2009)	0.0130	0.26	Indoor air, smoking, Portugal, 2008
Slezakova et al. (2009)	0.0041	0.082	Indoor air, nonsmoking, Portugal, 2008
Van Winkle and Scheff (2001)	0.00015	0.0031	Indoor air, 10 homes in Chicago from 1994-5
Van Winkle and Scheff (2001)	0.00029	0.0057	Outdoor air, 4 locations in Chicago, 1994-5

Risks from breathing indoor air in areas where smokers' second hand smoke is present are clearly higher than risks in areas without smokers.

Smokers, themselves have higher risk because they inhale the mainstream smoke in addition to the sidestream smoke. EFSA (2008) states that smokers who smoke 20 cigarettes per day obtain an exposure dose of BaP of 0.131 ug/day. People exposed to passive smoking would be exposed to 0.010 ug/m^3 for 5 hours per day resulting in a dose of 0.040 ug/day.

People exposed to second hand smoke were also shown by Northcross et al. (2012) to have high intake rates of PAHs. The authors measured the BaP-TE in the air of a car in which a smoker smoked 3 cigarettes over a period of one hour. The BaP-TE concentration was 7.67 ng/m^3. Over the course of that one hour, the daily dose of BaP-TE is 0.006 ug/day, which is similar to the BaP-TE daily dose the population gets in some locations over the entire day. Aquilina et al. (2010) found PAH in indoor air of homes with smoking. The daily dose is 0.005 ug/day. Mitra and Ray (1995) found a similar result and the daily dose of BaP-TE is 0.027 ug/day. Slezakova et al. (2009) found higher levels in locations where smoking occurred and the daily dose is 0.26 ug/day.

3.3 PAHs in Coal Tar Pharmaceuticals

Coal tar ointments, creams, and liquid pharmaceuticals have been used for over 100 years to treat psoriasis, eczema and atopic dermatitis. Many studies have been performed over the years to see if the patients who intentionally expose themselves to high level doses of coal tar for long periods of time have increased risks of cancer. All of the studies performed have been negative. Selected studies are summarized below.

Roelofzen et al. (2010) performed an epidemiological study on a cohort of 13,200 patients with psoriasis and eczema. 8,062 of these patients received coal tar treatments. There was no statistically significant increase

in overall cancer, skin cancer, internal cancer, or cancer of specific sites, including hematological, breast, lung, gastrointestinal, bladder and urinary tract, prostate or female reproductive organs observed in this study.

Hannukesela-Svahn et al. (2000) performed an epidemiology study of 5,687 Finnish patients with psoriasis. Coal tar with ultraviolet light treatment was studied (Goeckerman regimen) and there was no statistically significant increase in squamous cell carcinoma or non-Hodgkin's lymphoma in this study.

Pittelkow et al. (1981) performed a 25-year follow-up on 280 patients with psoriasis who received coal tar treatments. There was no increase in skin cancer of the coal tar treated individuals compared to expected cancer incidences. The authors stated: "The results of this study suggest that the incidence of skin cancer is not appreciably increased above the expected incidence for the general population when patients are treated with coal tar ointments."

Maughan et al. (1980) performed a 25-year follow-up study on 426 patients who received coal tar ointments clinically. The incidence of skin cancer was not increased above the expected incidence for unexposed populations. The authors' conclusion was: "Our study provides some assurance that the clinical use of coal tar products has not significantly altered the frequency of neoplasms from the natural course." "Those patients in whom skin cancers developed did not receive tar products any longer while hospitalized than did those without skin cancers; nor were they hospitalized more frequently. They did not receive any more coal tar than did the others, and many had received less."

Other papers that conclude that the use of coal tar pharmaceuticals does not increase the risk of cancer include:

- Mackenna (1959)
- Muller and Kierland (1964)
- Perry et al. (1968)
- Epstein (1979)
- Muller et al. (1981)
- Bickers (1981)
- Larko and Swanbeck (1982)
- Menter and Cram (1983)
- Alderson and Clarke (1983)
- Muller and Perry (1984)
- Lin and Moses (1985)
- Jones et al. (1985)
- Torinuki and Tagami (1988)
- Lindelof and Sigurgeirsson (1993)

- Bhate et al. (1993)
- Jemec and Østerlind (1994)
- Van Schooten and Godschalk (1996)

In an externally peer reviewed risk assessment report, ICF (2000) estimated that the average total lifetime exposure to patients in the Pittelkow et al. (1981) study was 254 grams of absorbed PAHs from coal tar. The average daily dose over the lifetime is 254 grams/ (70 years * 365 days/year) = 9.9 mg coal tar per day. The BaP-TE content of coal tar can be taken from Culp et al. (1998). The BaP-TE for two coal tar samples was 2,696 ppm and 3,965 ppm. The average is 3,331 ppm or 0.003331. The BaP-TE content of the average daily dose of the coal tar pharmaceutical users can be estimated as (9.9 mg coal tar) x (0.003331 BaP-TE/coal tar) = 0.033 mg BaP-TE per day (33 ug BaP-TE per day).

ICF (2000) also derived a dose of 5 ug of coal tar absorbed per day from coal tar shampoo use. Assuming the average BaP-TE content of coal tar from above, 3331 ppm, the dose of BaP-TE from coal tar shampoo use can be estimated as 5 ug/day x 0.003331 = 0.0167 ug/day.

3.4 Comparison of BaP-TE Intakes from Typical Exposures

Table S3 of Williams et al. provides intakes (i.e., average daily doses) for ingestion of soil and dust, expressed in nanograms per kilogram of body weight per day (ng/kg/day). These lifetime doses were converted to intakes in units of micrograms per day (ug/day) and compared to intakes from typical exposures to PAHs.

The average daily intake rate of BaP-TE for the general population ranges from 0.2 to 0.6 ug/day (Table 11). For tobacco smokers, this rate would range from 0.2 to 1 ug/day. For coal tar shampoo users or coal tar pharmaceutical users, the total daily intake would range from 0.017 to 33 ug/day.

The inflated BaP-TE intake rates assumed by the Williams et al. (2013) risk assessment report are, indeed, higher than the typical intake rates for the general population, which are dominated by dietary intake as noted by many summary documents on PAHs. However, when the errors and unconventional assumptions are corrected in this report, the average daily intake rate drops by more than an order of magnitude and are less than the typical intake rate for the general population.

This peer review report has made the observation that the study authors have no data whatsoever to characterize the levels of BaP-TE in soils at locations that are true exposure points. Thus, if the soil intake is excluded, the daily intake from Williams et al. (2013) for dust only is 0.27 ug/day of BaP-TE. This is about the same as the daily intake from other sources for the general population (0.2 to 0.6 ug/day). However, when errors in the risk assessment are corrected, as noted elsewhere in this report, the daily intake from dust using the dust data used by Williams et al. (2013) would be approximately 0.04 ug/day. This is about ten times lower than the daily intake from other sources for the general population.

Table 11. Summary of Daily Intakes of Benzo(a)pyrene Toxic Equivalents (BaP-TE)	
Source of Exposure	**Average Daily Intake (ug/day)**
Soil and dust near coal tar sealed commercial parking lots from Williams et al. (2013)	2.2
Soil and dust near coal tar sealed commercial parking lots with errors corrected per this report	0.13
Dust near coal tar sealed commercial parking lots from Williams et al. (2013)	0.27
Dust near coal tar sealed commercial parking lots with errors corrected per this report	0.042
Ambient air and indoor air	0.006 to 0.02
Diet	0.2 to 0.6
Smoking	0.2 to 1
Second hand smoke	0.005 to 0.26
Coal tar pharmaceuticals	33
Coal tar shampoo	0.017

4. Summary and Conclusions

Williams et al. (2013) is a regulatory risk assessment performed by USGS and Baylor University that attempted to link presence of PAHs in coal tar sealants to significant health risk. Risk assessments do not predict actual risks nor do they find associations between chemicals in the environment and health outcomes. HHRA is a structured procedure for answering questions about the risks of chemicals and physical agents on health but does not predict actual risk to people because of the many conservative approaches and safety factors used. Although PAHs are present in coal-tar-based sealants, there is no evidence that coal-tar-based sealants affect people's health. Furthermore, there is no evidence in people who intentionally put pure coal tar on their skin that the coal tar causes health problems. In fact, there is good evidence that it does not.

The flaws of the Williams et al. HHRA have been described in detail in this peer review report. Risks to people living near coal tar sealed pavement have not been established by the HHRA. Soil exposures to coal tar constituents in areas near sealed pavement where people might actually be exposed have not been characterized. For these reasons, the HHRA cannot be used to make any decisions about the risk of coal tar sealants.

The long history of use of coal tar as a therapeutic agent demonstrates that coal tar exposures do not increase people's risks of cancer. There is no evidence that low level or intermittent exposure to coal tar or coal tar pitch has caused cancer in humans. There is little evidence that high level repeated exposures have caused cancer in humans. There are some studies about high temperature industrial processes such as

aluminum smelting or coke oven gases that show some adverse effects but these studies have no relevance to coal tar sealants. Coal tar has a long history of use as a medicinal agent and in dandruff shampoo. People with psoriasis and other skin disorders apply coal tar ointments to large portions of their bodies for long periods of time. There is human evidence that coal tar pharmaceuticals do not cause cancer in humans. Numerous robust epidemiological studies have shown no increase in cancer risk in users of coal tar pharmaceuticals. In 2001, the FDA performed a formal review of the safety of coal tar as an over-the-counter (OTC) pharmaceutical and found that coal tar products are safe (FDA, 2001). “There is no evidence that topical treatment of dermatological disorders with OTC coal tar shampoo, soap, or ointment drug products increases the risk of skin cancers.” Coal tar pharmaceuticals are FDA-approved.

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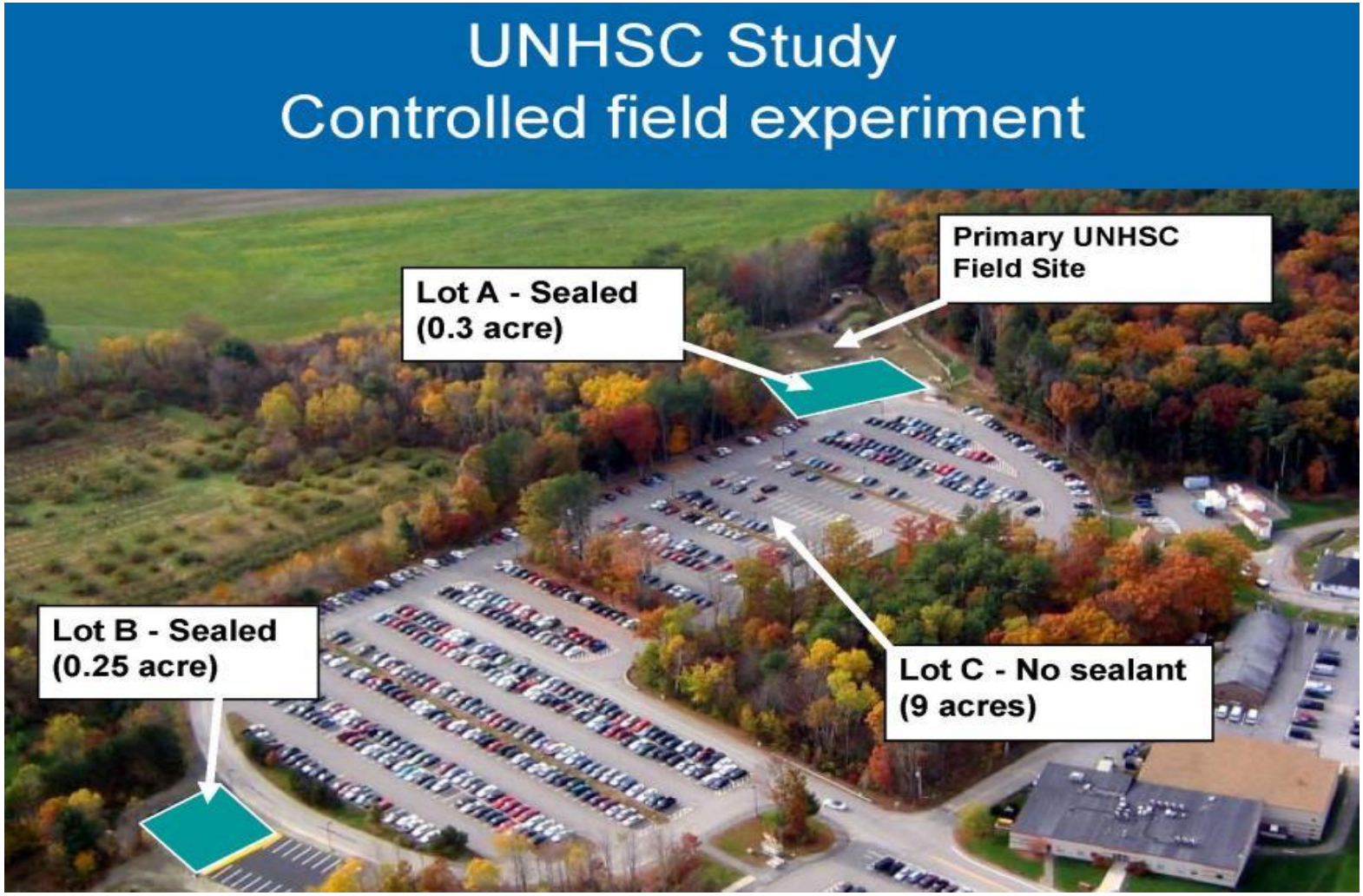
ARCADIS

Figures



Note:

Photo from UNHSC 2010

PAVEMENT COATINGS TECHNOLOGY COUNCIL

PEER REVIEW OF COAL-TAR-SEALED PAVEMENT RISK ASSESSMENT

UNHSC Study Parking Lots

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FIGURE

1

Note:

UNH sealed lots shown as

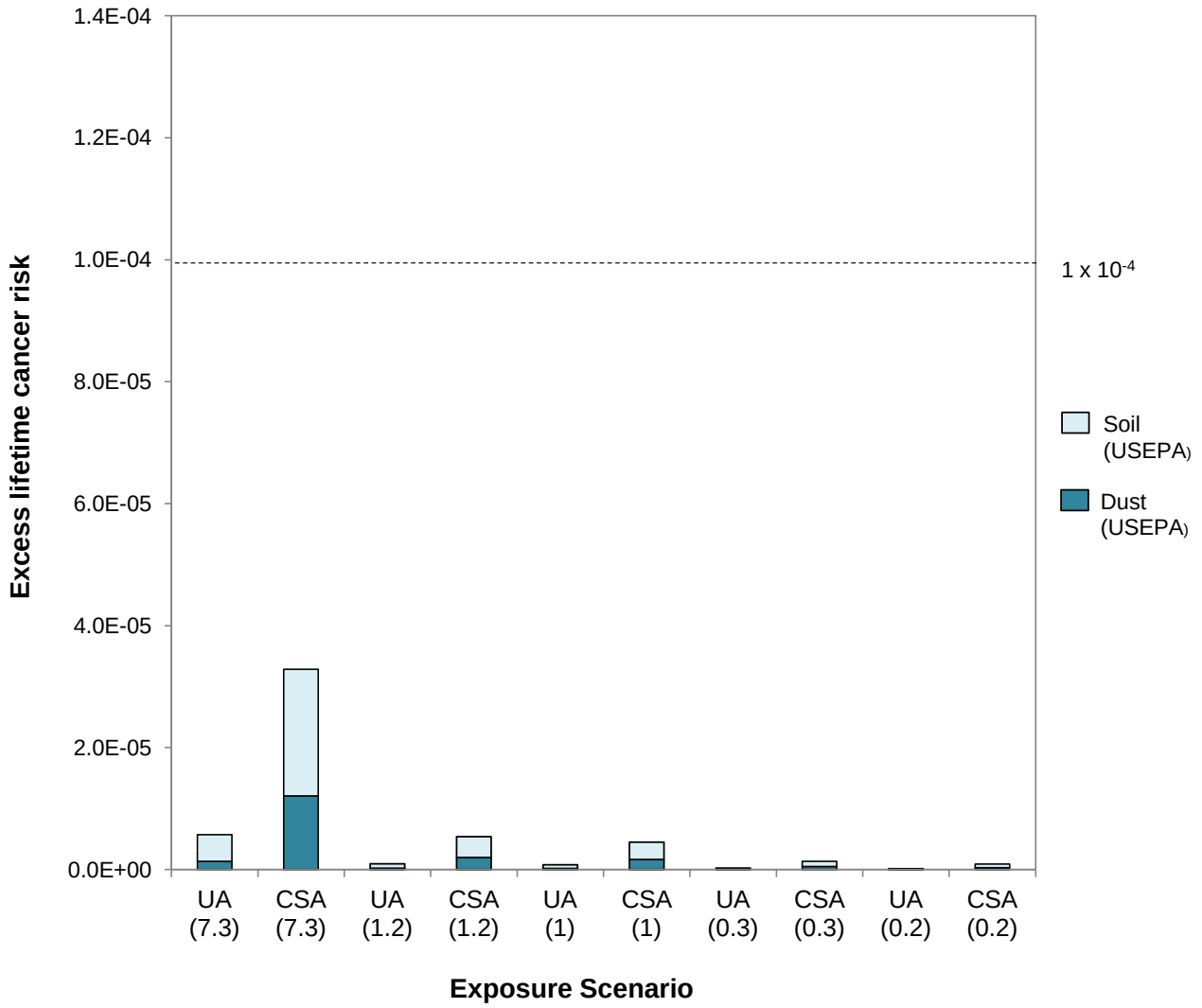
PAVEMENT COATINGS TECHNOLOGY COUNCIL

PEER REVIEW OF COAL-TAR-SEALED PAVEMENT RISK ASSESSMENT

UNHSC (2010) Soil Sample Locations

ARCADIS

FIGURE 2



Note:

Value in parentheses denotes OSF used.

PAVEMENT COATINGS TECHNOLOGY COUNCIL

PEER REVIEW OF COAL-TAR-SEALED PAVEMENT RISK ASSESSMENT

Risk Estimates Over the Range of BaP OSFs

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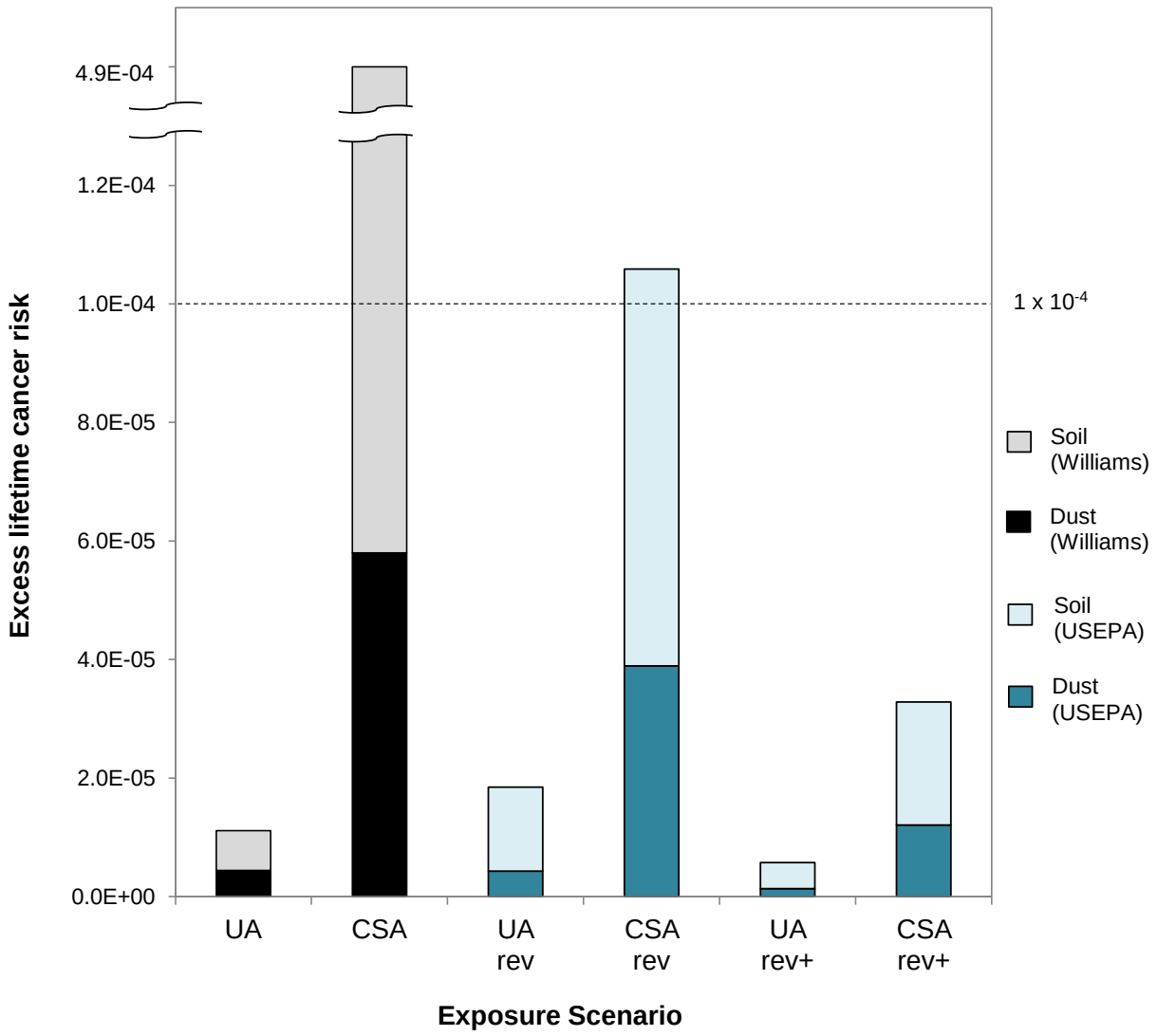
FIGURE 3



Notes:

rev = Revised EPCs and exposure assumptions

rev+ = Revised EPCs, exposure assumptions and 31% RBA

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PEER REVIEW OF COAL-TAR-SEALED PAVEMENT RISK ASSESSMENT

Comparison of Williams et al. 2013 and Corrected Risk Estimates

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FIGURE 4

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Attachment A

Analytical Data Tables

Table A.1
Concentrations of Potentially Carcinogenic PAHs in Settled House Dust from Mahler et al. (2010)

Pavement surface type	Parking lot designation	Benzo(a)pyrene	Benzo(a)Anthracene	Benzo(b)fluoranthene	Benzo(k)Fluoranthene	Chrysene	Dibenzo(a,h) Anthracene	Indeno(1,2,3-cd)Pyrene	**Sum of PAHs**	**BAP-TE**
		1	*0.1*	*0.1*	*0.01*	*0.001*	*1*	*0.1*		*<--TEF*
Coal-tar-based sealcoat	CT	3.42	3.17	5.66	2.24	5.21	<0.57	2.13	21.8	4.5
Coal-tar-based sealcoat	CT	15.2	13.2	25.5	9.47	20.6	<2.44	10.1	94.1	20.2
Coal-tar-based sealcoat	CT	10.9	7.7	20.8	7.07	15.6	<2.12	8.27	70.3	14.7
Coal-tar-based sealcoat	CT	4.04	4.01	8.51	2.73	6.75	<0.84	3.40	29.4	5.7
Coal-tar-based sealcoat	CT	14.3	14.7	28.5	10.4	24.7	<2.54	11.3	103.9	19.9
Coal-tar-based sealcoat	CT	1.21	0.93	2.70	0.80	2.00	<0.28	0.98	8.6	1.7
Coal-tar-based sealcoat	CT	1.41	3.99	2.73	1.14	6.87	<0.50	2.05	18.2	2.3
Coal-tar-based sealcoat	CT	7.33	6.24	14.7	5.04	15.2	<1.26	5.33	53.8	10.0
Coal-tar-based sealcoat	CT	4.50	4.15	8.33	3.09	6.94	<0.84	3.11	30.1	6.1
Coal-tar-based sealcoat	CT	4.44	4.07	15.9	3.43	15.7	<0.91	3.38	46.9	6.8
Coal-tar-based sealcoat	CT	24.2	20.8	38.4	15.2	38.3	<5.27	18.7	155.6	32.2
Unsealed concrete	NCT	0.15	0.10	0.26	0.10	0.18	<0.08	0.20	1.0	0.2
Unsealed asphalt	NCT	1.36	0.95	2.91	1.10	1.98	<0.31	1.05	9.4	1.9
Asphalt-based sealcoat	NCT	3.91	1.8	6.48	2.80	5.02	<0.75	2.22	22.2	5.0
Asphalt-based sealcoat	NCT	0.58	0.35	0.89	0.40	0.51	<1.25	0.45	3.2	0.8
Unsealed asphalt	NCT	1.50	1.19	2.33	0.95	1.61	<0.30	0.96	8.5	2.0
Unsealed asphalt	NCT	2.05	1.86	4.00	1.38	1.09	<0.42	1.36	11.7	2.8
Unsealed concrete	NCT	12.4	9.42	25.0	8.38	21.1	<2.55	9.52	85.8	16.9
Unsealed asphalt	NCT	0.06	0.05	0.14	0.06	0.11	<0.01	0.05	0.5	0.1
Unsealed asphalt	NCT	0.26	0.18	0.55	0.23	0.38	<0.05	0.20	1.8	0.4
Unsealed asphalt	NCT	0.23	0.20	0.48	0.18	0.45	<0.04	0.14	1.7	0.3
Asphalt-based sealcoat	NCT	0.30	<0.2	0.69	0.25	0.50	0.23	0.30	2.3	0.4
Unsealed asphalt	NCT	0.25	0.17	0.48	0.18	0.32	<0.05	0.19	1.6	0.3

Notes:
All concentrations in ug/g.
BaP-TE = benzo(a)pyrene toxic equivalents
CT = coal-tar-sealcoated parking lot
NCT = parking lot not coal-tar-sealcoated
SHD = settled house dust
TEF = toxicity equivalency factor

Table A.2
Concentrations of Potentially Carcinogenic PAHs in Soil Samples Collected Adjacent to Coal-Tar-Sealed Asphalt

			Site ID	421049088201301	421045088200001	Lot A	Lot A	Lot A	Lot A	Lot A	Lot B	Lot B	Lot B	Lot B
			Sample name	LKH.SC2	LKH.SC4	CT S1a	CT S1a	CT S3a	CTS3Cd	CTS4C	ASS1a	ASS1B	ASS23B	ASS24B
			Location	Lake in the Hills, IL	Lake in the Hills, IL	UNH	UNH	UNH	UNH	UNH	UNH	UNH	UNH	UNH
			Sample Type	CSA	CSA	CSA	CSA	CSA	CSA	CSA	CSA	CSA	CSA	CSA
			Sampling Date	7/31/2007	7/31/2007	5/8/2009	5/8/2009	5/8/2009	11/7/2009	11/7/2009	5/8/2009	8/17/2009	8/17/2009	8/17/2009
	TEF	Units												
Benz[a]anthracene	0.1	mg/kg		2.22	10.6	7.02	5.5	28.6	6.56	0.591	16.7	6.03	0.193	0.241
Benzo[a]pyrene	1	mg/kg		2.98	13.6	7.29	5.97	29.2	7.49	0.666	19.2	8.57	0.279	0.341
Benzo[b]fluoranthene	0.1	mg/kg		5.14	22.6	7.66	6.5	32.6	8.15	0.699	23.5	9.38	0.333	0.407
Benzo[j/k]fluoranthene	0.01	mg/kg		1.77	8.68	6.38	5.24	27.2	6.73	0.625	20.1	8.14	0.260	0.318
Chrysene	0	mg/kg		3.55	15.6	8.04	6.6	32.9	7.99	0.797	23.5	9.38	0.326	0.407
Dibenz[a,h]anthracene	1	mg/kg		<2.1	<2.6	1.73	1.45	6.68	1.36	0.111	4.44	2.0	0.067	0.084
Indeno[1,2-cd]pyrene	0.1	mg/kg		1.77	9.56	4.99	4.22	20.8	5.48	0.467	12.2	7.36	0.245	0.305
BaP-TE		mg/kg		3.91	17.98	11.06	9.10	44.38	10.94	0.96	29.10	12.94	0.43	0.52
													geometric mean BaP-TE	5.86

Notes:
All concentrations in mg/kg.
BaP-TE = benzo(a)pyrene toxic equivalents
CSA = coal-tar-sealed asphalt
TEF = toxicity equivalency factor
UNH = University of New Hampshire

Data sources:
Van Metre et al. (USGS) 2008
UNHSC 2010

Table A.3
Concentrations of Potentially Carcinogenic PAHs in Soil Samples Collected Adjacent to Unsealed Asphalt

			Site ID	421017088201401	420843088205601	Lot C	Lot C	Lot C	Lot C	Lot C
			Sample name	LKH.SC1	LKH.SC3	CN S1a	CNS7C	CNS8C	CSN14B	CNS15B
			Location	Chicago, Ill.	Chicago, Ill.	UNH	UNH	UNH	UNH	UNH
			Sample Type	UA	UA	UA	UA	UA	UA	UA
			Sampling Date	7/31/2007	7/31/2007	5/8/2009	11/7/2009	11/7/2009	8/17/2009	8/17/2009
	TEF	Units								
Benz[a]anthracene	0.1	mg/kg		0.666	<0.050	0.137	0.647	0.647	4.25	1.36
Benzo[a]pyrene	1	mg/kg		0.749	<0.050	0.17	0.647	0.63	4.95	1.54
Benzo[b]fluoranthene	0.1	mg/kg		1.58	<0.050	0.204	0.654	0.593	5.37	1.62
Benzo[j/k]fluoranthene	0.01	mg/kg		0.514	<0.050	0.182	0.582	0.53	4.16	1.48
Chrysene	0	mg/kg		1.3	<0.050	0.215	0.771	0.71	5.48	1.79
Dibenz[a,h]anthracene	1	mg/kg		<0.760	<0.050	0.04	0.111	0.102	1.07	0.348
Indeno[1,2-cd]pyrene	0.1	mg/kg		<0.760	<0.050	0.129	0.415	0.386	3.91	1.27
BaP-TE		mg/kg		0.98	ND	0.26	0.94	0.90	7.42	2.33
									geometric mean BaP-TE	1.24

Notes:
All concentrations in mg/kg.
BaP-TE = benzo(a)pyrene toxic equivalents
TEF = toxicity equivalency factor
UA = unsealed asphalt
UNH = University of New Hampshire

Data sources:
Van Metre et al. (USGS) 2008
UNHSC 2010