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September 17, 2013

Associate Director
Office of Science Quality and Integrity
U.S. Geological Survey
108 National Center
Reston, VA 20192

Via E-Mail (InfoQual@usgs.gov) and Overnight Mail

Re: Request for Correction of Information Submitted Under U.S. Geological Survey Information Quality Guidelines

Publications: Williams, E.S., B.J. Mahler, and P.C. Van Metre, "Cancer Risk from Incidental Ingestion Exposures to PAHs Associated with Coal-Tar-Sealed Pavement," *Environmental Science & Technology*. 47:1101-1109 (2013). Publication Date (Web): November 23, 2012 [hereinafter referred to as the "Risk Assessment"]

USGS Newsroom, "Proximity to Coal-Tar-Sealed Pavement Raises Risk of Cancer, Study Finds," March 28, 2013 [hereinafter "Press Release"] http://www.usgs.gov/newsroom/article.asp?ID=3538

USGS Science Feature – Top Story, "You're Standing on It! Health Risks of Coal-Tar Pavement Sealcoat," March 28, 2013 [hereinafter "Top Story" feature] http://www.usgs.gov/blogs/features/usgs_top_story/youre-standing-on-it-health-risks-of-coal-tar-pavement-sealcoat/

Dear Sir or Madam:

On behalf of the Pavement Coatings Technology Council (PCTC), which represents numerous companies throughout the country that are part of the sealcoat industry, I write to submit a request for correction of information disseminated by the U.S. Geological Survey (USGS). This request is made pursuant to the USGS Information Quality Guidelines and the U.S. Department of the Interior and the Office of Management and Budget (67 F.R. 8452) in accordance with Section 515 of the Treasury and General Government Appropriations Act for Fiscal Year 2001 (Public Law 106-554).

INFORMATION REQUIRING CORRECTION – OVERVIEW

As part of a long standing campaign, certain individuals within the USGS continue to use their government positions to influence and elicit emotional responses from consumers,

legislators, the press and even other government agencies with the goal of banning the use of coal tar sealants across the country. All too often, this campaign has side stepped sound scientific methodology, upon which the vast majority of USGS scientists pride themselves, and has relied instead upon a collection of questionably executed "studies" and press releases in which contrary scientific views are ignored, data are cherry picked or withheld, methodology flaws are overlooked, and perhaps most disturbing, hypotheses are presented essentially as undisputed facts.[1] Unfortunately, most people, including the press, do not have the necessary time, training and tools to recognize when findings and conclusions are being exaggerated in terms of their scientific significance, especially when phrases such as "increased cancer risks" are thrown about and other tactics[2] - designed to create unjustified fear - are being used to promote the agenda of a few.

The type of scenario described above has been identified in the scientific community as "White Hat Bias." This phenomenon was first recognized in a 2010 article by two NIH-funded researchers that was published in the International Journal of Obesity and posted on the NIH Public Access website.[3] While this initial article focused on the impact of White Hat Bias in the field of obesity research, the lessons to be learned are equally applicable to the USGS' coal tar sealant research:

> 'White hat bias' (WHB) (bias leading to distortion of information in the service of what may be perceived to be righteous ends) is documented via quantitative data and anecdotal evidence from the research record regarding the postulated predisposing and protective effects respectively of nutritively-sweetened beverages and breastfeeding on obesity. Evidence of an apparent WHB is found in a degree sufficient to mislead readers. ***WHB bias may be conjectured to be fueled by feelings of righteous zeal, indignation toward certain aspects of industry, or other factors. Readers should beware of WHB and our field should seek methods to minimize it.*** (emphasis added).[4]

The manner in which White Hat Bias can creep into government studies and specifically into the Risk Assessment that is the focus of this DQA challenge will be explored in the sections below. Dr. Brian Magee, a board certified toxicologist with special expertise in the fields of PAH toxicity and risk assessment, prepared a well documented Report entitled "Peer Review of Coal-Tar-Sealed Pavement Risk Assessment" that identifies the many ways in which the

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[1] This is the 3rd DQA challenge that has been filed against the USGS and its coal tar sealant publications over the past 4 months. The first challenge, filed on May 15, 2013, focused on the USGS' flawed modeling in its 40 Lakes Paper in which the USGS claimed, mistakenly, that coal tar sealants had been shown to be the primary source of PAH contamination in lakes east of the Continental Divide. The 2nd challenge, filed on May 31, 2013, focused on the USGS' inappropriate use of catfish tumor photos to frighten the public into considering coal tar sealant bans. Thus far, the USGS has requested two extensions of time up to mid-November, 2013, to respond to both challenges. See http://www.usgs.gov/info_qual/coal_tar_sealants.html.

[2] *See e.g.,* catfish photos that are the subject of the 2nd DQA challenged referenced in fn.1, *supra.*

[3] Cope,M and Allison, D, "White Hat Bias: Examples of Its Presence in Obesity research and a Call for Renewed Commitment to Faithfulness in Research Reporting," Int J Obes (Lond); 34 (1): 84-88; January, 2010.; http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2815336/

[4] *Id.* at 1.

Williams/USGS Risk Assessment has run afoul of sound scientific methodology. The Report is attached hereto as Exhibit A and was summarized by Dr. Magee as follows:

> [The Williams/USGS Risk Assessment] 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.

USGS Guidelines, of course, dictate that all reasonable efforts be made to guard against bias and advocacy in USGS publications and statements, and when these efforts fail, corrections must be made. Since serious assertions once again have been made that certain USGS scientists have breached these Guidelines in the context of coal tar sealant research, it is prudent to begin this analysis with yet another review of the relevant Guidelines.

USGS GUIDELINES

The USGS Guidelines require that USGS data collection and research activities be "carried out in a consistent, objective, and replicable manner" aimed at ensuring the objectivity, utility, and integrity of information disseminated to the public. *See* USGS Guidelines, Section III; Office of Management and Budget ("OMB") Guidelines, 67 F.R. 8452 (February 22, 2002) (incorporated by reference in the USGS Guidelines). To be "objective," information published by the USGS must be presented in an "accurate, clear, complete, and unbiased manner." *Id.* at 8459. "Objectivity" also requires that original and supporting data be generated, and analytic results developed, using sound statistical and research methods. *Id.*

The USGS Manual also refers to "impartiality and non-advocacy" as terms that build upon the concepts of "objectivity" raised by the OMB. Specifically, the USGS Manual emphasizes the importance of presenting facts and interpretations impartially for others to use for their own purposes: "Alternatives are evaluated rather than solutions recommended. Advocacy positions are avoided. There is no implied adverse criticism of the private sector." USGS Manual 500.9 § 5.C. It goes without saying that a refusal to acknowledge or cite peer reviewed articles that take a position contrary to the USGS' own research is a form of advocacy that clearly lacks objectivity.

Another way to determine if any bias or advocacy exists within the USGS on the issue of coal tar sealants is for the USGS to produce all related data, correspondence and emails concerning its coal tar sealant research and any internal agenda that it or certain of its scientists may have regarding this product. A FOIA request asking for such materials was sent off more than two years ago and, incredibly, remained "open" until August 22, 2013.[5] As will be

[5] Details regarding the failure of the USGS to respond in a timely manner to the above mentioned FOIA request were set forth in a March 15, 2013 letter to the USGS FOIA liaison. That letter is an exhibit within the 40 Lakes DQA filed on May 15, 2013, referenced in fn. 1, *supra*.

demonstrated in greater detail below, the USGS has sought to minimize evidence of advocacy within its ranks by withholding and redacting large volumes of correspondence and email between the USGS staff and other individuals who have made it their goal to ban coal tar sealants across the country.[6] These efforts by the USGS to withhold certain documents are not only at odds with the above mentioned USGS Guidelines, but also contrary to the need for transparency that is emphasized throughout the Guidelines and by the President and the U.S. Attorney General.[7]

WHITE HAT BIAS – WARNING SIGNS

> Whether WHB is intentional or unintentional, stems from a bias toward anti-industry results, significant findings, feelings of righteous indignation, results that may justify public health actions, or yet other factors is unclear. Future research should study approaches to minimize such distortions in the research record.[8]

The quote above comes from Drs. Mark Cope and David Allison of the University of Alabama at Birmingham. They identify four categories in which White Hat Bias can affect studies and papers that, on their face, appear to be premised on solid scientific reasoning. The four categories are: (1) Citation Bias; (2) Publication Bias; (3) Miscommunications in Press Releases; and (4) Inappropriate or Questionable Inclusion of Information.

Citation bias is described as a tendency to cite other research or papers in a way that exaggerates or misrepresents the strength of evidence for a given proposition, thereby creating the potential for readers to be deceived. Publication bias focuses on the tendency of studies with "significant" results to be published in favor of studies that statistically have "non-significant" results. Both sets of biases are apparent in the present Risk Assessment. For example, the Risk Assessment asserts early on that "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."[9] This statement is made as though it is an undisputed fact in the scientific literature and is designed to sensitize readers to the alleged pervasiveness of coal tar sealant contamination. In reality, this "fact" is nothing more than a hypothesis, and a much contested one at that.

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[6] Over the last three months, three separate FOIA appeals have been filed that seek to compel the USGS to produce various emails, calculations, data and draft reports that have been redacted or withheld by the USGS pursuant to a claimed "deliberative process" privilege. While the appeals remain pending, the public is left to speculate as to why the USGS wishes to hide the content of these specific documents. Thus far, there has been no response from the USGS regarding the appeals or their status.

[7] On President Obama's first full day in office on January 21, 2009, he declared "a new era of open government" and ordered that FOIA "should be administered with a clear presumption: in the face of doubt, openness prevails." Pursuant to President Obama's directive, Attorney General Holder issued FOIA Guidelines on March 19, 2009, to the heads of executive departments and agencies "reaffirming the commitment to accountability and transparency." U.S. DOJ FOIA Post, *"Creating a New Era of Open Government,"* 2009; http://www.justice.gov/oip/foiapost/2009foiapost8.htm.

[8] *See* fn. 3, *supra*, p. 5.

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

Specifically, whenever the Risk Assessment discusses coal tar sealants and PAHs in lake sediment, it is referring to the USGS' 40 Lakes Paper that was authored by Dr. Barbara Mahler (USGS Geologist and Research Hydrologist) and her husband, Dr. Peter Van Metre (USGS Hydrologist), who also happen to be two of the three Risk Assessment authors. The 40 Lakes Paper is the subject of a separate DQA challenge in which numerous flaws have been identified, including the failure of USGS to acknowledge or cite peer reviewed studies that found no significant correlation between sediment contamination and coal tar sealants. The arguments set forth in the 40 Lakes DQA will not be repeated here. Suffice it to say that the same types of citation and publication biases associated with the 40 Lakes Paper are perpetuated in the present Risk Assessment. Indeed, the volume of self conducted "research" that was favorably cited in the Risk Assessment, compared to the conspicuous absence of any citations to industry funded research, should be a clear warning sign that some sort of bias might be at play.

As for the third category – miscommunications in press releases – evidence obtained and cited by Drs. Cope and Allison suggests that "press releases from academic medical centers often promote research that has uncertain relevance to human health and do not provide key facts or acknowledge important limitations."[10] Certainly, if academic medical centers are at risk for issuing misleading press releases, government agencies are not immune to this same problem. The fourth category – inappropriate or questionable inclusion of information – raises similar concerns.[11] Once again, both types of bias are apparent in the Risk Assessment itself and in the manner in which the Risk Assessment was handled and promoted by the USGS.

Consider, for example, the Risk Assessment as published. It was first made available online on November 23, 2012 with little press attention or fanfare. In retrospect, this was not particularly surprising since the article was not available on a government website. Instead, it had to be purchased through a privately owned website for $35, and that is still the case today. The fact that government research must be purchased raises an ancillary, but important concern. Will the public and press actually bother to read the underlying article if they have to pay $35 for a copy of it? Probably not.

The USGS apparently decided that their coal tar sealant risk assessment needed to get more attention. Thus, four months later on March 28, 2013, the USGS took on the role of advocate and posted on its website a "Science Features: Top Story" entitled "You're Standing on It! Health Risks of Coal Tar Pavement Sealcoat." This Top Story feature warned the public that "living adjacent to a coal tar sealed pavement is associated with ***significant*** [emphasis added] increases in estimated excess lifetime cancer risks, and that much of the increased risk occurs during early childhood." It further claimed that "the average estimated lifetime BAPEQ dose for someone living adjacent to a coal tar sealcoated pavement was 38 times greater than for someone living adjacent to unsealed asphalt pavement." To further ensure that this message was received loud and clear, the USGS issued a Press Release that covered the same topics in the same dramatic manner. Like the Risk Assessment, both the subsequent USGS Top Story feature and Press Release are the subject of this DQA challenge.

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[10] *See* fn.3, *supra*, p.3.

[11] A misleading USGS press release related to the 40 Lakes Paper has already been addressed in an earlier DQA challenge and, as indicated above, will not be repeated here.

The USGS' Press Release and Top Story feature were picked up by the media shortly after they hit the USGS website. By combining allegations of increased cancer risks with references to children, it was a virtual certainty that these communications would generate attention. Indeed, U.S. News and World Report ran an article which repeated the USGS' alarmist assertions just a few days later on April 4, 2013.[12] The title of the article was "Common Asphalt Sealant May Raise Cancer Risks."

Many other news organizations and television stations followed suit.[13] Yet, as mentioned above, it seems highly unlikely that any of these media outlets and reporters actually purchased the Risk Assessment for $35 and reviewed the underlying data and findings. In essence, the manner in which the USGS characterized the Risk Assessment in its Press Release and Top Story feature became the newsworthy item, more so than the study itself. Drs. Van Metre and Mahler, of course, were also contacted by the media and continued to self-promote their findings. The problems with this type of blatant advocacy should be obvious. Limitations of the Risk Assessment were not properly conveyed in these abbreviated USGS communications and more importantly, numerous problems with the Risk Assessment's underlying methodology and data were ignored, thereby breaching the USGS Guidelines. However, before one can fully appreciate the magnitude of the defects within the Risk Assessment, and the misleading nature of the subsequent Press Release and Top Story postings, it is necessary to have a basic understanding of PAHs and what a risk assessment is and isn't.

PAH BACKGROUND FACTS

Various USGS scientists, including Drs. Van Metre and Mahler, claim that contamination of urban lakes and streams by polycyclic aromatic hydrocarbons (hereinafter PAHs) is widespread in the U.S. This assertion is not particularly surprising since there is a consensus in the scientific community that PAHs have many potential sources, including vehicle emissions, tire particles, motor oil, crude oil, power plant emissions and industrial releases. Indeed, almost any type of combustion with organic matter will produce PAHs as a by-product, including natural sources such as forest fires and volcanoes down to something as basic as grilling on the backyard barbecue. Thus, one would expect PAHs to be ubiquitous in our environment and, in fact, they are. Just as important, PAHs have been around since the dawn of man. If there was a fire that offered our ancestors warmth or light, or cooked their food, PAHs were present.

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[12] http://health.usnews.com/health-news/news/articles/2013/04/04/common-asphalt-sealant-may-raise-cancer-risks

[13] *E.g.*, Huffington Post, http://www.huffingtonpost.com/2013/08/15/coal-tar-sealant-cancer-asphalt-carcinogen_n_3762033.html; USA Today, http://www.usatoday.com/story/money/business/2013/06/16/toxic-driveways-cities-states-ban-coal-tar-pavement-sealants/2028661/; UK Daily Mail, http://www.dailymail.co.uk/news/article-2342854/Cities-ban-coal-tar-driveway-sealants-amid-accusations-cause-cancer.html; Cleveland TV station KSDK, http://www.ksdk.com/news/article/390342/3/Some-communities-ban-certain-asphalt-sealant

The near universal presence of PAHs in sediment, dust, soil, food and air creates some uncomfortable truths for individuals who wish to use theoretical cancer risks as a reason for banning coal tar sealants. If one assumes - as does the USGS - that any level of exposure to PAHs, no matter how small, is theoretically capable of causing cancer (i.e. a linear cancer slope factor), then one comes away with the inevitable conclusion that one's risk of cancer increases whenever we eat or drink food, breathe air, or touch soil and dust, regardless of the presence of coal tar sealants. Thus, it is essentially meaningless and misleading for someone to warn the public that exposure to coal tar sealants increases one's risk of cancer. Given the assumptions described above, so will eating a hamburger, drinking coffee, breathing city air, or picking up a handful of dirt virtually anywhere in the country.

From a policy perspective, the key question is not whether there is a theoretical increased cancer risk associated with the use of coal tar sealants, but whether the risk is *significant*. By characterizing the coal tar sealant risk as being *significant* in its Press Release and Top Story feature, the USGS quickly got the attention that ban advocates within the agency had sought. The analysis, of course, must be taken a couple of steps further. How exactly was the crucial finding of "significance" reached in the Risk Assessment and did the process comply with USGS Guidelines?

RISK ASSESSMENTS AS POLICY TOOLS

Generally speaking, quantitative risk assessments are calculated by using a formula that incorporates a cancer slope factor, numerous exposure assumptions and sampling data. The cancer slope factor reflects, to some degree, real world observations (usually in laboratory animals exposed at high doses) in which greater exposures lead to greater risk.[14] Exposure assumptions focus on factors such as the amount of soil or dust that is accidentally ingested by a child or an adult in a given day, the number of days during the year in which exposure is likely, whether the exposure is residential or commercial, and the amount of the chemical that is bio-available or actually metabolized by the body once ingested or inhaled. Sampling data seek to establish the concentration of chemicals in the soil, dust or air to which a person has been exposed.

As one can see, there are many different variables in a cancer risk assessment. A mistaken assumption or bad piece of sampling data can have a profound impact on the overall calculation. Since governmental agencies prefer to err on the side of caution, their assumptions tend to overestimate, not underestimate risk. For these reasons, toxicologists do not consider risk assessments to be reflections of actual cancer risk.[15] Instead, risk assessments are tools that can be useful in making policy decisions about whether certain exposures are "significant." State

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[14] Cancer slope factors are many times premised upon chemicals that are similar to, but not identical to the chemical or product being considered. It is also common for animals to be subjected to chemical exposures at levels much greater than what would be expected in humans.

[15] As noted by Dr. Magee at pages 23-24 of his Report marked as Exhibit A, despite what theoretical risk assessments may say, there is no evidence that low level or intermittent exposure to coal tar or coal tar pitch has caused cancer in humans. The long history of coal tar use as a therapeutic agent further supports this conclusion. 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.

legislatures or environmental agencies are then tasked with deciding if those types of exposures, from a policy perspective, are acceptable. What is deemed to be an "acceptable" level of exposure can differ significantly from state to state, from agency to agency, and even from one USEPA Region to another.[16]

In his Report attached hereto as Exhibit A, Dr. Magee takes the time to explain how "significant" cancer risks are frequently determined from a policy perspective. This type of explanation can sound a bit technical, but is crucial to understand.

> 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)

Essentially, an increased cancer risk of 1×10^{-4} refers to 1 additional case of cancer in 10,000 people over the course of a lifetime, which is assumed to be 70 years. Statistically, if one assumes a male's lifetime cancer risk is 40%, then an exposure which leads to an incremental lifetime cancer risk of 1×10^{-4} increases that risk, over 70 years, to 40.01%. Clearly, the incremental (and theoretical) risk of cancer from such an exposure is quite small compared to the male's overall cancer risk, but as mentioned above, government agencies tend to err on the side of caution when setting policy.

In the Risk Assessment that is being challenged, the USGS considers any incremental cancer risk greater than 1×10^{-4} to be *significant.* This fact is confirmed in the USGS' own Top Story posting, which reads in relevant part as follows:

> For the average individual who lives adjacent to coal-tar sealed pavement for either their entire life or just the first 6 years, the excess lifetime cancer risk is estimated to be greater than 1 in 10,000. Estimated cancer risk associated with coal tar sealcoat is even higher for children that consume larger than average amounts of soil and dust. In general, the U.S. Environmental Protection Agency considers excess cancer risks greater than 1 in 10,000 to be sufficiently large that some sort of remediation is desirable.

Unfortunately, given the number of assumptions and variables that go into a cancer risk assessment, the stage is set for White Hat Bias to play a prominent role in determining whether coal tar sealant exposure can truly be characterized as a "significant" cancer risk. If one simply

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[16] Risk assessments conducted on exposures to complex mixtures of PAHs, such as coal tar sealants, using EPA's "Relative Potency Factor" (RPF) guidance must be qualified as policy tools even more so than risk assessments premised upon single chemical exposures. Large uncertainties surround the RPF assumption that the exposure of rodents to a pure PAH compound adequately represents human exposures to a complex mixture that contains many different types of PAH compounds. There is a well founded concern that these RFP assumptions may overestimate actual risk. See eg., "Advisers Try to Clarify EPA's Risk Assessment Approach for PAH Mixtures," INSIDEEPA.COM, October 7, 2010, attached hereto as Exhibit B.

changes a couple of variables, an exposure risk that was initially deemed to be insignificant (i.e. a lifetime incremental cancer risk of less than 1×10^{-4}) can suddenly become significant. The only way for someone to determine if the Williams/USGS Risk Assessment conclusions are scientifically sound and consistent with USGS Guidelines is to actually purchase the Risk Assessment for $35 and go through the process of analyzing all of the citations, data and assumptions upon which the Risk Assessment relies. While one might not expect a reporter from U.S. News and World Report to conduct such an analysis, or city councilmen to do so, Dr. Brian Magee did, and what he discovered was disturbing.

SPECIFIC EXAMPLES OF RISK ASSESSMENT GUIDELINE BREACHES

Overview of Dr. Magee's Report

The scope of Dr. Magee's Report is quite extensive. His report, alone, clearly demonstrates the many ways in which the Williams/USGS Risk Assessment is flawed and thereby fails to prove - through the use of sound scientific methodologies, data collection and assumptions - that coal tar sealant exposures are a "significant" cancer risk for children or adults. Nevertheless, the USGS continues to post on its website the related Press Release and "Top Story" feature that warn the public about "significant" cancer risks allegedly caused by residing near coal tar sealed pavement. This conclusion is presented as a scientific fact with no reservations, with no indication of uncertainties and with no references to the specific concerns raised by Dr. Magee. Such conduct is not only misleading and biased toward the encouragement of coal tar sealants bans, it is also a clear violation of the USGS Guidelines.

By attaching a copy of Dr. Magee's Report as Exhibit A, the details of his analysis are available for everyone to review and will not be repeated here. For the sake of convenience, Dr. Magee also prepared a summary of his findings, set forth below as Table 1, which lists the many flaws that have been ignored and overlooked by the Risk Assessment authors as they promote their internal agenda of banning coal tar sealants.

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. (2009) 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.

SURDYK & BAKER

As one might have expected, when proper assumptions are inserted into the risk assessment formula, the lifetime incremental cancer risk for coal tar sealant exposure drops dramatically, well below the risk of 1×10^{-4} which has been used by many agencies and the Risk Assessment authors as a threshold for "significant" risk. Dr. Magee also summarized the extent to which cancer risks drop after inserting different assumptions – assumptions supported by the scientific literature and more accurate sets of data. That summary is set forth in Table 8, below.

Table 8. Comparison of Risk Estimates

Scenario		Estimated Lifetime Risk
0	Williams et al. (2012) 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.

As stated at the beginning of this DQA challenge, Dr. Magee has demonstrated that no increases in estimated cancer risks above regulatory levels of concern have been established.[17] Dr. Magee's conclusions are further supported by comparisons made at the end of his Report to other types of PAH exposures commonly experienced by people today.[18] In essence, the theoretical risks generated by coal tar sealant exposures are comparable to those generated by ordinary food consumption, and less than those generated by certain consumer products, such as psoriasis treatments that have coal tar as an ingredient.[19] As pointed out by Dr. Magee, the FDA concluded more than a decade ago that "[t]here 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." Similarly, there is no evidence that coal-tar-based sealants adversely affect people's health.[20]

According to its own Guidelines, the USGS can no longer continue to let the public believe that living next to coal tar sealed parking lots do, in fact, create significant cancer risks and that this issue has now been settled, scientifically, by the USGS. The Risk Assessment only proves one thing with any certainty; namely, that White Hat Bias can distort findings in many different ways.

Whereas Dr. Magee premised his findings on data, citations and information set forth within the Risk Assessment, other information gradually has been made available as a result of

[17] This is true even when assuming, as did the Risk Assessment authors, that the EPA's Relative Potency Factor (RPF) approach is reasonable and does not overestimate risks associated with complex mixtures of PAHs, such as those found in sealants. As indicated in fn. 16, *supra*, this assumption is questionable.
[18] *See* Dr. Magee's Report, Exhibit A, pp. 20-23.
[19] Once again, allowing for the sake of argument that the RPF approach adequately allows a comparison of fundamentally different complex mixtures of PAHs.
[20] *See* Dr. Magee's Report, Exhibit A at pp. 23-24.

FOIA requests sent to the USGS, the Minnesota Department of Pollution Control (MPCA) and the USEPA. The FOIA requests sent to the USGS and the EPA are still being pursued since thousands of pages of emails and calculations have been withheld, purportedly for the reason that they reflect the agencies' deliberative process. The USGS' and EPA's position on this issue is being contested since it is clearly contrary to President Obama's and the U.S. Attorney General's call for transparency with regard to FOIA requests.[21] Once produced, it is highly likely that the documents presently withheld will give the public further insights into the thought processes of Dr. Van Metre, Dr. Mahler and others within the USGS as far as their coal tar sealant agenda is concerned. In this regard, it is not particularly surprising that the USGS and EPA are making every effort to keep these documents hidden from view. Indeed, the USGS has been stalling and fighting against complete disclosure for nearly 2 ½ years.[22]

Nevertheless, some correspondence has been produced by the EPA and USGS with respect to its coal tar sealant research, and more emails have been generated by the MPCA, with which the USGS has worked closely. Although these disclosures are incomplete, they provide additional evidence of White Hat Bias as it pertains to the Williams/USGS Risk Assessment. Since neither Dr. Magee, nor any other peer reviewer, could be expected to have access to this information, highlights are presented below as additional support for what, ultimately, must result in a retraction by the USGS of the Risk Assessment, the Press Release and subsequent Top Story posting.

Dust and Dr. Mahler

The Williams/USGS Risk Assessment uses two types of sampling data to perform the cancer risk calculations: dust and soil. Thus, the cancer risks in the Risk Assessment are premised upon the incidental ingestion of dust and soil from one's hands. Theoretically, the risks are greater for small children because it is assumed that they put their fingers into their mouths more than adults and teenagers. In this respect, when it comes to dust or soil contamination, it does not matter what the contaminant is – the risk to children will always be greater because of these assumptions. In other words, the alleged increased risk to children is not specific to coal tar sealants, even though the wording of the USGS Press Release and Top Story feature make it appear that way.

The Risk Assessment obtained all of its house dust data from one source: a 2010 article written by Dr. Mahler, Dr. Van Metre, Tom Ennis and several others entitled "Coal Tar Based Parking Lot Sealcoat: An Unrecognized Source of PAH to Settled House Dust" [hereinafter "House Dust Study"].[23] It should be noted that Mr. Ennis, in addition to working for the City of Austin and being a major proponent of the coal tar sealant ban in that city, is also the founder and primary contributor to an anti coal tar sealant blog called "Coal Tar Free America." While none of the authors of the House Dust Study appear to have been trained in the fields of toxicology, risk assessment or exposure assessment, they did not hesitate to speculate at the end of their article about alleged cancer risks caused by children's exposure to house dust near sealed parking lots.

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[21] *See* fn. 7, *supra.*
[22] *See* fn. 5 & 6, *supra.*
[23] Environ Sci Technol., 2010, Vol. 44, p. 894-900.

> Maertens et. al. calculated the excess cancer risk resulting from nondietary ingestion of carcinogenic (B2) PAHs in SHD [Settled House Dust] during preschool years. They reported that 10% of the households they sampled had a concentration of B2 PAHs that exceeded 40 ug/g, resulting in an excess cancer risk of greater than 1×10^{-4} for a "high" dust ingestion scenario of .1g/day. Of the 11 apartments with CT parking lots sample for this study, six (55%) had a concentration of B2 PAHs that exceeded 40 ug/g, indicating that use of coal tar sealcoat on parking lots and driveways is related to elevated concentrations of carcinogenic PAHs in SHD.[24]

The reference to an alleged incremental lifetime cancer risk greater than 1×10^{-4} is telling. Drs. Van Metre and Mahler undoubtedly believed that this type of residential exposure represented a "significant" cancer risk that needed to be recognized and addressed. Ultimately, the authors concluded that house dust near coal tar sealed parking lots contained concentrations of PAHs that would be unacceptable under German environmental standards. German standards were consulted because, according to the authors, there were no applicable U.S. dust standards. Although not explicitly stated, it was evident that the solution, in the minds of the authors, was to expand the coal tar sealant ban that had been adopted in Austin. No other inference can be drawn.

Given these conclusions, and the fact that no toxicologist was actually involved in developing or publishing the House Dust Study, efforts were made to have the article reviewed by a board certified toxicologist with experience dealing with PAHs. For this particular project, Dr. Rosalind Schoof was retained to evaluate the House Dust Study. Her complete Report, dated April 19, 2010, is attached hereto as Exhibit C. Dr. Schoof summarized her conclusions as follows, and they are still valid today.

> Short-comings in the study design introduced uncertainty in data quality and in data evaluation, including uncertain identification of coal-tar sealed and non-coal-tar sealed parking lots; absence of characterization of other PAH sources; absence of consideration of ages of apartment complex, parking lot and sealant, and carpeting; collection of composite samples that may not accurately represent exposure potential; and potential for cross-contamination between samples.
>
> Both concentration and dust loading are important factors in evaluating exposure to chemicals in dust. Mahler et al. (2010) did not evaluate dust loading, which is critical in understanding *how much* dust is available for contact by residents.
>
> Mahler et al. (2010) did not compare PAH results to a health-based standard to determine the potential risk associated with the levels measured in house dust. Use of the screening level developed for cleanup of residences near the World Trade Center in New York City indicates that cancer-causing PAHs in dust measured by Mahler et al. (2010) are below

[24] Id. at p. 899.

> levels of concern. In fact, the highest level measured by Mahler et al. (2010) in indoor dust is less than half of the World Trade Center screening level, even though PAH concentrations in dust may be overestimated due to the selected sampling method.
>
> Intake of cancer-causing PAHs in dust occurs every day through the air we breathe and food we eat. The levels measured by Mahler et al. (2010) that could be taken in via house dust are consistent with background intake levels via food, air, and water.[25]

In a nutshell, Dr. Schoof expressed a number of concerns about the manner in which the house dust data were collected. Nevertheless, she assumed for the sake of argument that the data were sound. She then performed a screening level risk assessment that was premised upon health-based standards adopted by the EPA for the cleanup of residences near the World Trade Center in New York City after 9/11. Dr. Schoof concluded that house dust exposures in apartments next to coal tar treated parking lots did **not** create a significant cancer risk for children or anyone else. She further noted that several studies have demonstrated that the presence of chemicals in house dust, including PAHs and dioxins, have little or no correlation with chemicals measured in residents exposed to that house dust.[26]

After completing her initial analysis, Dr. Schoof asked the USGS for certain underlying data from the House Dust Study that had not been produced. This information would have allowed Dr. Schoof and others to assess more fully the modeling that allegedly connected the house dust PAHs to coal tar sealed parking lots. Her request was also accompanied by a number of written questions that sought clarification on the manner in which the House Dust Study was conducted. This is what scientists do in order to check and double check the credibility of new "findings."

Although Dr. Mahler never formally responded to Dr. Schoof, an internal USGS document was discovered in a FOIA response which appears to provide insights as to how she and Dr. Van Metre viewed Dr. Schoof's risk assessment and requests.[27] A copy of that document has been attached hereto as Exhibit D. Assuming that it was, in fact, drafted by Dr. Mahler, it would be an understatement to say that she was not receptive to Dr. Schoof's inquiries, which were repeatedly characterized as being nothing more than "obfuscation." With respect to Dr. Schoof's risk assessment, Dr. Mahler seemed to take offense and wrote: "So, they are accusing us of doing a health-risk analysis when we did not do so, and then they go ahead and do a health risk

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[25] See Exhibit B, p. 16

[26] Id., p. 6. The studies that fail to show any correlation between house dust exposures and body burden were not only ignored by Drs. Van Metre and Mahler in 2010, but later as well. The Williams/USGS Risk Assessment makes no mention whatsoever of these studies which, of course, would be inconsistent with the warnings expressed in the subsequent Press Release and Top Story feature.

[27] Exhibit D presumably was drafted by Dr. Mahler and /or Dr. Van Metre. By refusing to respond completely to the FOIA request, it is not presently possible to know with 100% certainty if Dr. Mahler was the author as opposed to her husband, Dr. Van Metre. Regardless of the author (Dr. Mahler or Dr. Van Metre), the points to be made are the same.

analysis on nonexistent data." What's especially curious about this assertion is that the Risk Assessment authors presumably relied upon this same "non-existent" data when they later decided to perform their own incremental cancer risk calculations for house dust. Why Dr. Mahler would be annoyed at Dr. Schoof performing a risk assessment that used this same information is unclear.[28]

On January 24, 2012, another coal tar sealant article written by Drs. Mahler and Van Metre was published. This one was titled "Coal Tar Based Pavement Sealcoat and PAHs: Implications for the Environment, Human Health, and Stormwater Management" [hereinafter referred to as the "Sealcoat Implications" article].[29] Dr. Mahler was listed as the "corresponding author" and the co-authors included Dr. Allison Watts and a new recruit, Dr. E. Spencer Williams, who is an assistant research scientist at the Center for Reservoir and Aquatic Systems Research in Baylor's College of Arts & Sciences. Unlike the other authors, Dr. Williams has a PhD in Toxicology. Near the end of this article and in a section captioned "Human-Health Concerns," an observation was made that "non-dietary ingestion of PAH-contaminated house dust and soil likely are the most important routes of exposure, but a complete human risk analysis is required before the cancer risk associated with ingestion of these media can be quantified."[30] It would appear that Dr. Williams was brought on board to perform this "human risk analysis."[31] Ten months later this analysis was available on line as the Williams/USGS Risk Assessment which is the focus of this DQA challenge.[32]

Furthermore, what previously had been a somewhat hidden agenda suddenly burst into the open in this particular article. In essence, the Sealcoat Implications article represented the formal coming out party for the USGS' agenda to ban coal tar sealants throughout the country. Indeed, the last section of this article was captioned "Regulatory and Retail Actions" and described in detail a "patchwork of actions [that] has been taken to either ban or restrict the use of coal tar based sealcoat in the United States." Several paragraphs were used to mention bans that had been implemented in Austin, Texas, Washington D.C., several cities in Minnesota, Washington State and elsewhere.[33] By contrast, not one sentence was written about states and municipalities where Drs. Mahler or Van Metre had given presentations and proposed bans were rejected. The article further claimed that "research to date, as documented here, provides a compelling weight-of-

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[28] At roughly this same time in 2010, Dr. Mahler was also displeased with other scientists who questioned her findings and conclusions with respect to coal tar sealants. For example, in an email dated 2/24/2010, Dr. Mahler suggested that PCTC Executive Director, Dr. Anne LeHuray, had been providing the Springfield City Council with a "few outright lies." Said email is attached hereto as Exhibit E. Clearly, by 2010, battle lines had been drawn, at least from the perspective of Dr. Mahler, which almost certainly would have increased the risk of White Hat Bias taking hold. It should be noted that Dr. Mahler chose to share her thoughts with Dr. Allison Watts of the University of New Hampshire, who was copied on this particular email. The significance of Dr. Watts' involvement will become more apparent in the following section that addresses the flaws associated with the soil data.

[29] Environ Sci Technol, 2012, Vol. 46, pp. 3039-3045.

[30] *Id.* at 3043.

[31] Exactly how and why Dr. Williams was selected for this task is unclear. According to his CV and bio, Dr. Williams's background is in cardiovascular toxicology and until being contacted by Drs. Mahler and Van Metre, he had no publications or research projects related to human PAH exposures.

[32] As indicated in the caption of this DQA challenge, the online version was also published in a subsequent issue of *Environmental Science & Technology*.

[33] *See* fn. 29, *supra*, p. 3043.

evidence [argument] that coal tar based sealcoat products are an important source of PAHs to our environment."[34] However, when the "research to date" ignores conflicting peer reviewed articles and cites instead the authors' own findings from earlier studies, it becomes clear that this "weight-of-evidence" argument was essentially derived by putting a thumb on the USGS side of the scale. All of these claims are the hallmarks of White Hat Bias and advocacy.

Time has been spent analyzing the Sealcoat Implications article because it truly gives context to the importance of the Williams/USGS Risk Assessment from the perspective of Drs. Mahler and Van Metre. Beginning in the early 2000s, they had literally spent years collecting different types of data, writing articles, talking to the press and giving various presentations all devoted to demonstrating the alleged hazards of coal tar sealants to the environment. By 2010, they expanded the scope of their warnings and used their House Dust article to assert that humans, especially children, were also at significant risk, not just for minor aliments such as skin irritation, but for cancer. They were vested personally and professionally to these propositions, and there was no turning back. Scientists retained by industry who challenged their findings, such as Dr. Schoof, could be dismissed as obstructionists and industry "hired guns."[35]

Given the complexity of the science, especially chemical fingerprinting, it was easier for the press and certain legislators simply to trust the USGS. And why not? The USGS has a good reputation. Indeed, it would be almost unthinkable to suggest that the USGS as a whole would not only allow, but actively support (through continued funding, Press Releases and other website postings) two scientists in their pursuit of a nationwide coal tar sealant ban unless sound scientific methodology had undeniably proven that Drs. Mahler and Van Metre were correct.

Despite the confidence exuded by Drs. Mahler and Van Metre, by January of 2012 it became evident that a crucial piece of the puzzle was still missing. As indicated above, the authors of the Sealcoat Implications article recognized that so long as they continued to assert that living next to coal tar sealed lots created a significant cancer risk, especially for children, they would need a formal risk assessment to support this assertion. That was the role for which Dr. Williams was selected. All that remained to be done during the rest of 2012 was for Dr. Williams to confirm that the cancer risks described in the 2010 House Dust Study were indeed "significant." Drs. Mahler and Van Metre, however, never received that confirmation. It turned out that the risk assessment did not support the dire warnings set forth in the 2010 House Dust Study, at least not with respect to house dust. In essence, Dr. Schoof had been right all along.

According to the Risk Assessment, the lifetime excess cancer risk for house dust exposure, using reasonable *maximum* exposure estimates (otherwise known as RME - a term of art defined in EPA guidance) was only 5.8×10^{-5}. And if one used average or central tendency exposures, the

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[34] *Id.*

[35] Dr. Schoof and Dr. LeHuray were not the only ones who were summarily dismissed by Drs. Mahler and Van Metre. For example, Dr. Robert DeMott received similar treatment. At pages 3 and 4 of Exhibit D, Dr. Mahler and/or Dr. Van Metre attacked efforts by Dr. DeMott to publish findings that were critical of earlier USGS sealcoat conclusions. In doing so, they appeared to possess and convey to colleagues presumably confidential information regarding the ongoing peer review process. Exactly how Drs. Mahler and Van Metre obtained this information is unclear. Regardless, Dr. DeMott's findings were later published in a peer reviewed journal. When this fact was brought to the attention of Dr. Van Metre, he simply chose to reject Dr. DeMott's article in a conclusory manner without any explanation. *See e.g.*, email marked as Exhibit F.

risk dropped by more than half to 2.0×10^{-5}. In terms of policy making, these risks are less than 1×10^{-4} and are not considered to be significant. From the perspective of Drs. Mahler and Van Metre, this news must have been devastating. Yet, there still remained one possible way of using risk assessment to argue that living next to a coal tar sealed lot created a *significant* cancer risk, and that was to rely instead on soil exposure, not household dust. The House Dust Study, however, did not include soil samples. Thus, the soil data would have to be obtained from an entirely different set of studies, and as it turned out, the choices were not only extremely limited, they were also extremely flawed. Nevertheless, that did not prevent Drs. Mahler, Van Metre and Williams from moving forward. There was too much at stake.

Soil and Dr. Van Metre

The impact of soil data on the overall conclusions of the Risk Assessment cannot be overstated. According to the calculations set forth within the Risk Assessment, the ingestion of soil affected by coal tar sealant "is a more important driver of risk" than the ingestion of house dust affected by coal tar sealant. In these types of settings, soil accounts for 72 to 84% of the alleged excess lifetime cancer risk.[36] Drs. Mahler, Van Metre and Williams then go onto to note that excess lifetime cancer risks allegedly caused by soil near coal tar sealed lots can be as high as 4.3×10^{-4} when RME assumptions are used. Since that risk is greater than the 1×10^{-4} policy threshold discussed above, the authors and USGS apparently concluded that they were justified in warning the media on March 28, 2013 that people who reside near coal tar sealed parking lots face "significant" cancer risks.

Soil then, is the fundamental driver of the alleged cancer risk that caught the attention of media across the country beginning in March of 2013, not house dust. Did the USGS Press Release and Top Story make this subtle fact known? To the contrary, those specific communications carefully lumped dust and soil together, so it would appear that both created a significant cancer risk. Since Dr. Mahler and Van Metre had already issued a stern warning in their 2010 House Dust study about the alleged health hazards of house dust contaminated by coal tar sealants - and were committed to this proposition - they presumably were not anxious to point out this subtle distinction in press releases, or acknowledge to their peers inside the USGS and out that Dr. Schoof may have been correct. Of course, since soil was the driver of risk, combining soil exposure with any other exposure, such as eating a hamburger, would have made the combined exposures a "significant" cancer risk. This tactic is another prime example of White Hat Bias influencing the manner in which information was disseminated.

Putting aside that particular example of White Hat Bias, the Press Release and Top Story feature might still have been partially credible if it could be shown by Dr. Mahler, Dr. Van Metre and Dr. Williams that living next to soil affected by coal tar sealants really did create a "significant" cancer risk, and that the soil data used by them could be confidently and consistently applied to all other people who lived next to coal tar sealed parking lots and driveways across the country. Indeed, the purpose of the Press Release and Top Story clearly was to generate a degree of apprehension and concern amongst what certainly must be millions of people who fall into that category. There was no attempt to limit the scope of the Risk Assessment findings to the small

[36] *See* fn. 9, *supra*, p. 1104.

group of people who had been studied, or warn that the findings were incomplete, preliminary, potentially flawed or subject to any debate. Such qualifications tend to lessen media interest. Instead, the increased cancer risk, especially for children, was presented as a scientific fact to the media and nation. What remains a mystery is why it became necessary for Dr. Magee to point out the many defects in the Risk Assessment's soil gathering techniques, methodology, related assumptions and risk calculations. All of these defects should have been pointed out long ago to supervisors and directors not only within the USGS, but also in the Department of Interior. The Press Release and Top Story feature never should have been issued.

As mentioned by Dr. Magee, one of the most obvious flaws in the Risk Assessment analysis is that the tested soil had little relevance to the alleged cancer risks that were being described in the Press Release and Top Story. In the Press Release, it is asserted that "the use of coal tar based pavement sealants magnifies aggregate exposures to PAHs in children and in residences *adjacent* to where the products are used and is associated with human health risks in excess of widely accepted standards." In the Top Story feature, and as indicated in the beginning of this DQA challenge, it's claimed "that living *adjacent* to a coal tar sealed pavement is associated with significant increases in estimated excess lifetime cancer risk." Thus, in both the Press Release and Top Story feature, the public was led to believe that the soil sampling must have come from areas such as homes with front yards next to seal coated driveways. That was not the case. The soil samples actually came from areas located directly next to or actually *within* large commercial parking lots with no homes or apartments nearby. Unless someone lives on a median in the middle of large parking lot, played there as a child 7 days a week and continued to live there for 70 years, the soils samples used for the Risk Assessment are irrelevant.

To understand fully how this bit of misdirection took place, it is important to recall that dust, of course, was the focus of 2010 House Dust Study along with the alleged health risks that it might create. Soil apparently was not a concern. Indeed, if it had been, one can only assume that soil *adjacent* to the apartments would have been tested along with the interior house dust samples. That was not done. Given the fact that soil samples were now needed if there was going to be any chance of demonstrating the existence of a "significant" cancer risk via risk assessment, other soil sampling options had to be considered, and fast. Admitting that house dust did not create a significant cancer risk never appeared to be an option that was entertained by Drs. Williams, Mahler and Van Metre.

In terms of methodology, one can suppose that the next best alternative would have been for the Risk Assessment authors to locate another study in which a large number of soil samples had been taken from residences adjacent to seal coated parking lots and driveways. Obviously, as pointed out by Dr. Magee, even this alternative would have been flawed since the testing would have been on homes and residences that were not the same as house dust residences, thereby allowing for many different variables to tarnish the significance of any findings that might have been generated.[37] Unfortunately, even this questionable alternative was not available to the authors of the Risk Assessment. Yet, Drs. Mahler, Van Metre and Williams decided to press forward.

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[37] See Dr. Magee's Report, Exhibit A at p. 9.

Ultimately, it turned out that the Risk Assessment authors could only find two studies that had collected soil samples that were "adjacent" to both coal tar sealed parking lots and unsealed parking lots, the latter of which were to be used for the purpose of comparison. By this time, it apparently did not matter to Drs. Mahler, Van Metre or Williams that neither study actually evaluated the impact of parking lots or driveways that were adjacent to residences. They needed soil data for their Risk Assessment, and this is what was available. Dr. Magee summarized at page 3 of his Report the soil data that were actually used in the Risk Assessment:

> 4 soil samples from Lake in the Hills, Illinois[38] (2 samples next to CSA[39] parking lots and 2 soil samples next to UA[40] parking lots);
>
> 6 soil samples from Durham, New Hampshire[41] (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 [Risk Assessment] relies on a total of 10 soil samples to represent the entire U.S.

Actually, the data can be broken down even further. Dr. Magee noted that only 7 soil samples actually dealt with soil allegedly contaminated by coal tar sealants, and those were cherry picked from a larger data set.[42] The Project Leader of the unpublished New Hampshire Study, Dr. Allison Watts, further acknowledged that her soil data likely was impacted by snow plow shavings.[43] Not very impressive science, but it would appear that such defects were not going to get in the way of certain alarmist conclusions that the Risk Assessment authors felt the media and public needed to hear.

What Dr. Magee did not realize, but Dr. Van Metre did, is that the problems associated with the New Hampshire study even go beyond what Dr. Magee outlined in his Report. For

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[38] The Lake in the Hills data is reported in Van Metre, P., et.al., "PAHs Underfoot: Contaminated Dust from Coal Tar Sealcoated Pavement Is Widespread in the United States," Environ. Sci. Technol., 2009, Vol. 43(1), pp. 20-25.
[39] CSA = Coal-tar Sealed Asphalt
[40] UA = Unsealed Asphalt
[41] The New Hampshire soil data are reported in an *unpublished* study entitled "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, 2010. The Project Leader was Dr. Allison Watts.
[42] See Dr. Magee's Report, Exhibit A at p. 5.
[43] The Risk Assessment notes that the highest level of BaP detected in a "contaminated" soil sample was 29.2 ug/g, which was from the New Hampshire Study. Exhibit G is a graphic display of all the unpublished soil and dust data generated by the New Hampshire Study. Exhibit G can be found at p. 6 of said Study. The data within Circle A of Exhibit G reflects all of the sampling that was conducted at the specific site which generated the BaP reading of 29.2 ug/g. BaP is a subset of Total PAHs, and the total PAHs reported for this sample was 411 ug/g in the Spring of 2009, which would have been after the winter melt off. If Dr. Watts' concerns were correct and certain samples had been affected by snow plow shavings, this sample presumably was one of them. What the Risk Assessment authors neglected to point out was that this same soil sampling site was tested 6 months later in the Fall of 2009, and once again in the Fall of 2010. By that time, the total PAHs detected were only 19.9 ug/g., a dramatic decrease that is consistent with background levels. The Risk Assessment authors chose to ignore this fact and assume that people would be exposed to the much higher levels (i.e. BaP – 29.2 ug/g; Total PAH – 411 ug/g) for 70 years. It's an assumption that is contrary to the facts and another example of White Hat Bias at work.

example, the New Hampshire study was initially designed so that the sampling would be conducted next to three different types of parking lots: one covered with a coal tar sealant; one covered with an asphalt sealant; and a third left untreated. According to Dr. Watts, this plan was not followed:

> The contractor was requested to apply a coal tar based sealant to Lot A and an asphalt based product to Lot B using standard industry practices. . . . However, after the sealant was applied, it was found that coal tar based sealant had been applied to both lots and the study was adjusted to accommodate two coal tar sealed lots, rather than one.[44]

Technically, it's true that a problem was suspected "after the sealant was applied." As it turned out, it was nearly two years afterwards. The precise manner in which this problem was "found" is enlightening, especially since it reflects the manner in which Dr. Van Metre exerted influence on one of the few coal tar sealant studies that was not published under his or his wife's name. This is a fact that would not have been discovered but for the FOIA request that was filed.

When the sealant was being applied in 2007, Dr. Watts had assumed that the dust and soil samples associated with the coal tar sealed lot would be much higher than what was found next to the asphalt sealed lot. This is what she had been led to believe according to earlier studies conducted by Drs. Mahler and Van Metre. It turned out, however, that the second highest soil B(a)P and Total PAH reading came from a 2009 sample that was located next to Lot B,[45] which according to the contractor was asphalt sealed. To explain this discrepancy, Dr. Watts assumed that coal tar sealant was applied to both Lots A & B and, as indicated above, she retroactively "adjusted" her study and subsequent Report to accommodate this new "fact."

Although Dr. Watts chose not to publish her dust and soil data (a fact that apparently caused no concern to anyone within the USGS), she did publish data and findings regarding sediment and stormwater run-off.[46] After this aspect of her Study had been published, it was brought to Dr. Watts' attention that the contractor never backed down from his contention that Lot B was treated with asphalt sealant and that her assumptions regarding the use of coal tar sealants on both Lots A & B might be flawed. It was further suggested that Dr. Watts might wish to considered retracting or correcting her article and subsequent Report. Dr. Watts reached out to Dr. Van Metre and Tom Ennis for advice.[47] Using a classic example of circular reasoning, Dr. Van Metre and Mr. Ennis both argued that the contractor must have been mistaken and that he must have put down coal tar sealant on Lot B, not asphalt sealant, because Dr. Watts' data (see Exhibits G and I) would otherwise be inconsistent with Dr. Van Metre's and Mr. Ennis' pre-existing belief that coal tar sealed parking lots must cause much greater soil contamination than asphalt sealed parking lots. Dr. Van Metre and Mr. Ennis ultimately convinced Dr. Watts not to

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[44] Watts, A., et. al., "Polycyclic Aromatic Hydrocarbons in Stormwater Runoff from Sealcoated Pavements," Environ. Sci. Technol., 2010, Vol. 44(23), p. 8850.
[45] See Exhibit G, Circle B.
[46] *See* fn 44 *supra.*
[47] See emails marked as Exhibit H. Also recall that Mr. Ennis operates the blog known as "Coal Tar Free America."

retract her article and welcomed her "to the club."[48] This is yet another example of White Hat Bias at play.

Despite all of the shortcomings that plague Dr. Watts' New Hampshire study, it nevertheless provides 5 of the 7 data points used by the Risk Assessment authors to argue that soil next to sealed pavement can cause "significant" cancer risks, not just in New Hampshire, but all across the country wherever coal tar sealants are used. The inadequate and extremely limited nature of the data being used to support this proposition should now be evident to all except those with a pre-existing agenda to ban coal tar sealants. To make matters worse, in order to calculate RME risk, it would appear that the Risk Assessment used at least one soil sample located next to Lot B (the asphalt sealed lot?) with the highest B(a)P level while rejecting two Lot B soil samples with the lowest B(a)P levels - levels that were entirely consistent with normal background levels.[49] In other words, it would seem that Lot B was considered to be a coal tar sealed lot when soil B(a)P levels were relatively elevated and then was treated as an asphalt sealed lot that could be ignored when soil B(a)P soil levels were low and consistent with background sources. If there is any truth to this impression, such conduct would represent an egregious form of data cherry picking that is inexcusable. Presently, no one can be certain of the motivation behind the Lot B data selection process because the Risk Assessment authors apparently decided that there was no need for anyone to know why certain data were included and rejected.

Once the New Hampshire Study is put aside, the Lake in the Hills study is the only one left that has been cited as support for the proposition that living next to a coal tar sealed driveway or parking lot creates a significant cancer risk as far as soil exposure is concerned. Even a superficial review of the Lake in the Hills study quickly demonstrates that no such conclusion can be rationally or scientifically extrapolated from this study.

First, no one can seriously suggest that four data points make up a well conceived scientific study that can somehow be applied to the rest of the country. Second, the two "contaminated" soil samples were taken from sites next to or within large commercial parking lots and not near any residences.[50] Third, one of the two soil samples reportedly affected by coal tar sealant had B(a)P measured at 2.98 ug/g which is actually lower than background levels for the City of Chicago according to an entirely separate USGS soil study conducted in 2001 and 2002.[51] The Risk Assessment authors chose to ignore the USGS Chicago study in its entirety,

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[48] *Id.*

[49] At p. 1102 of the Risk Assessment, it is asserted that the "coal tar sealant" soil samples used in the risk calculations ranged from 2.98 to 29.2 ug/g. We also know from the same page of the Risk Assessment that 5 of the 7 soil samples came from the New Hampshire study. Raw data for the New Hampshire soil sampling can be found at the end of Dr. Watts' Report and is attached hereto as Exhibit I. In looking at the 5 soil samples associated with Lot A, one has a B(a)P level less than 2.98 ug/g, so we know that at the most, only 4 of the 5 Lot A samples were used and at least one sample had to come from Lot B. We also know that at least two of the 4 Lot B samples had B(a)P levels below 2.98 ug/g, which means that they were rejected.

[50] See Van Metre, et. al., "Collection and Analysis of Samples for Polycyclic Aromatic Hydrocarbons in Dust and Other Solids Related to Sealed and Unsealed Pavement from 10 Cities Across the United States, 2005-07," Data Series 361, USGS, 2008, p. 2.

[51] Kay, R., et. al.., "Concentrations of Polycyclic Aromatic Hydrocarbons and Inorganic Constituents in Ambient Surface Soils, Chicago, Illinois: 2001-02," Water Resources Investigation Report 03-4105, USGS, 2003; *see also*

which is not surprising since that particular study also describes in detail the proper method for conducting statistically appropriate soil sampling grids, both in terms of locations and quantity. Needless to say, no such methodology was implemented in the Lake in the Hills or New Hampshire studies.

Ultimately, what remains clear is that Drs. Williams, Mahler and Van Metre had an obligation to alert peer reviewers, USGS management and the media about the many limitations and flaws associated with the New Hampshire and Lake in the Hill studies and the contorted manner in which a "significant" cancer risk was found to exist. Unfortunately, all three did just the opposite by instead promoting themselves and their agenda in the Press Release and Top Story feature posted on the USGS website.

REQUESTED CORRECTIVE ACTION

The many examples of White Hat Bias described above, regrettably, are not limited to the USGS or the topic of coal tar sealants. For example, on July 24, 2013, the Energy and Environment Subcommittees of the U.S. House of Representatives' Committee on Science, Space and Technology held a joint hearing that focused on the EPA's scientific processes for studying the potential impacts of hydraulic fracturing on drinking water. Specific concerns were raised about prior EPA studies that explored alleged groundwater contamination at three different locations: Pavillion WY; Parker County, TX; and Dimock, PA. Significantly, after certain sampling and data deficiencies were brought to light, the EPA retreated from its original conclusions that hydraulic fracturing might have caused groundwater contamination at those sites. Environment Subcommittee Chair Chris Stewart (R-Utah) offered the following observations:

> EPA's recent announcement that it is walking away from its attempt to link hydraulic fracturing to groundwater issues in Pavillion, Wyoming is the most recent example of the agency employing a 'shoot first, ask questions later' policy toward unconventional oil and gas production. This marks the third case in which EPA has made sweeping allegations of fracking-caused contamination, only to have to recant these claims later due to errors, omissions and breaches of protocol. At a time when so many Americans are learning to distrust our federal government, this is another blow for the credibility of our federal agencies.[52]

The same House Committee expressed similar concerns regarding questionable comments made by NIEHS/NTP Director Linda Birnbaum on the alleged health effects of certain environmental chemicals. The Committee criticized Dr. Birnbaum for failing to clarify whether her statements were NIEHS/NTP policy or merely personal opinion.[53] The point to be

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Magee Report, Exhibit A at p. 9. The significance of the USGS background soil sampling in Chicago is further addressed in the 40 Lakes DQA challenge that was filed on May 15, 2013.

[52] http://science.house.gov/press-release/members-question-scientific-integrity-epa-hydraulic-fracturing-studies; *see also.*, Kish, D., "Lisa Jackson's EPA Goes Rogue," US New & World Report, web edition, April 6, 2012, http://www.usnews.com/opinion/blogs/on-energy/2012/04/06/lisa-jacksons-epa-has-gone-rogue

[53] http://science.house.gov/sites/republicans.science.house.gov/files/documents/06-13-2013%20Letter%20to%20Dr.%20Collins.pdf

made is that the problem of White Hat Bias is a perpetual one that must not only be constantly addressed by the EPA and NTP, but by the USGS as well, regardless of how embarrassing or difficult it may be for the USGS to do so.

If the USGS wishes to maintain its reputation for high level scientific inquiry and integrity, it must acknowledge the problems that now exist and act upon them. Circling the wagons and hoping the problems will disappear through delays and withholding crucial documents is not a viable option. Indeed, more DQA challenges are coming. As the USGS might have suspected, the findings set forth within the coal tar sealant vapor emission studies published by Drs. Mahler and Van Metre are next. Why the USGS would allow two hydrologists to spearhead not one, but two vapor emission studies is one of the issues that will be explored.

As far as the Risk Assessment is concerned, the first set of corrective actions should start with the Press Release and Top Story feature. Both of these communications reflect an agenda to ban coal tar sealants by grossly distorting research findings in order to scare the public into thinking that living next to coal tar sealed pavement significantly increases cancer risks. This alleged "fact" clearly has not been established by sound scientific methodology and most likely never will be. Thus, the Press Release and Top Story as presently drafted must be removed from the USGS website.

Second, for the reasons set forth in Dr. Magee's Report, the actual Risk Assessment must also be retracted or withdrawn. The USGS Guidelines mandate that such action be taken. It has been demonstrated repeatedly how the Risk Assessment is not only filled with examples of White Hat Bias, but is premised upon data that no one can seriously suggest were obtained in an "objective" way by using sound statistical methods and research techniques. This type of corrective action is entirely consistent with the solution adopted by the EPA with respect to its flawed hydraulic fracturing studies. There is no acceptable alternative. Ignoring the problem only compounds past mistakes and makes matters worse.

Third, with respect to future USGS presentations on coal tar sealants, one can only assume that Drs. Mahler and Van Metre will continue to cite their Risk Assessment for the propositions set forth in the now discredited Press Release and Top Story feature. This must stop. Furthermore, given the many examples of White Hat Bias that have been found to apply to Drs. Mahler and Van Metre, in this DQA Challenge and earlier ones, the time has come to restrict them from participating in future coal tar sealant presentations sponsored by the USGS. Any additional coal tar sealant research performed under the auspices of the USGS should be conducted by a new group of scientists who are not personally and professionally committed to a certain agenda and to each other.

Respectfully Submitted,

Leonard S. Kurfirst

LSK/lk (w/enclosures)

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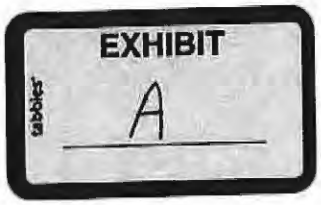

ARCADIS

Peer Review of Coal-Tar-Sealed Pavement Risk Assessment

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

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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.

5. References

Alderson, M.R. and J.A. Clarke. 1983. Cancer incidence in patients with psoriasis. *Br J Cancer* 47:857-859.

Aquilina, N.J., J.M. Delgado-Saborit, C. Meddings, S. Baker, R.M. Harrison, P. Jacob III, M. Wilson, L. Yu, M. Duan, and N.L. Benowitz. 2010. Environmental and biological monitoring of exposures to PAHs and ETS in the general population. *Environment International* 36:763-771.

Bhate, S.M., G.R. Sharpe, J.M. Marks, S. Shuster, and W.M. Ross. 1993. Prevalence of skin and other cancers in patients with psoriasis. *Clinical and. Experimental Dermatology* 18:401-4.

Bickers, D.R. 1981. The carcinogenicity and mutagenicity of therapeutic coal tar - a perspective. *J Invest Dermatol* 77:173-174.

Bordelon, N.R., K.C. Donnelly, L.C. King, D.C. Wolf, W.R. Reeves, and S.E. George. 2000. Bioavailability of the genotoxic components in coal tar contaminated soils in Fischer 344 rats. *Toxicological Sciences*. 56: 37-48.

Bradley, L.J.N., B.H. Magee, and S.L. Allen. Background Levels of Polycyclic Aromatic Hydrocarbons (PAH) and Selected Metals in New England Urban Soils. *Journal of Soil Contamination*. 1994.

Brune, H., R.P. Deutsch-Wenzel, M. Habs, S. Ivankovic, and D. Schmahl. 1981. Investigation of the tumorigenic response to benzo(a)pyrene in aqueous caffeine solution applied orally to Sprague-Dawley rats. *J Cancer Res Clin Oncol* 102(2):153-157.

Butler, J.P., G.B. Post, P.J. Lioy, J.M. Waldman, and A. Greenberg. 1993. Assessment of carcinogenic risk from personal exposure to benzo(a)pyrene in the Total Human Environmental Exposure Study (THEES). *Air & Waste* 43(7):970-977.

California Air Resources Board (CARB). 1994. *Research Note 94-10 – Indoor Concentrations of Phthalates and PAHs*. California Environmental Protection Agency. June.

Chuang, J.C., G.A. Mack, M.R. Kuhlman, and N.K. Wilson. 1991. Polycyclic aromatic hydrocarbons and their derivatives in indoor and outdoor air in an eight-home study. *Atmospheric Environment* 25B(3):369-380.

Chuang J.C., P.J. Callahan, R.G. Menton, S.M. Gordon, R.G. Lewis, and N.K. Wilson. 1995. Monitoring methods for polycyclic aromatic hydrocarbons and their distribution in house dust and track-in soil. *Environ Sci Technol* 29:494-500.

Chuang J.C., P.J. Callahan, C.W. Lyu, and N.K. Wilson. 1999. Polycyclic aromatic hydrocarbon exposures of children in low-income families. *J Expo Anal Environ Epidemiol* 9(2):85-98. [PubMed:10321348]

Culp, S.J., D.W. Gaylor, W.G. Sheldon, L.S. Goldstein, and F.A. Beland. 1998. A comparison of the tumors induced by coal tar and benzo[a]pyrene in a 2-year bioassay. *Carcinogenesis* 19:117-124.

Dennis, M.J., R.C. Massey, D.J. McWeeny, and M.E. Knowles. 1983. Analysis of polycyclic aromatic hydrocarbons in UK total diets. *Food and Chemical Toxicology* 21(5):569-574.

De Vos, R.H., W. van Dokkum, A. Schouten, and P. de Jong-Berkhout. 1990. Polycyclic aromatic hydrocarbons in Dutch total diet samples (1984-1986). *Food and Chemical Toxicology* 28(4)263-268.

Electric Power Research Institute (EPRI). 2003. Polycyclic Aromatic Hydrocarbons (PAHs) in Surface Soils in Western New York. Electric Power Research Institute. 1005296. October.

EPRI. 2008. Examination of the Sources of Polycyclic Aromatic Hydrocarbon (PAH) in Urban Soil. Electric Power Research Institute. 1015558. December.

European Commission. 2002 . *Opinion of the Scientific Committee on Food on the Risks to Human Health of Polycyclic Aromatic Hydrocarbons in Food.* European Commission, Health and Consumer Protection Directorate – General Directorate C – Scientific Opinions. C2 – Management of Scientific Committees; scientific co-operation and networks. Scientific Committee on Food. Brussels, Belgium. December. http://europa.eu.int/comm/food/fs/sc/scf/index_en.html

European Food Safety Authority (EFSA). 2008. Scientific opinion of the panel on contaminants in the food chain on a request from the European Commission on Polycyclic Aromatic Hydrocarbons in Food. *The EFSA Journal* 724:1-114.

Epstein, J.H. 1979. Risks and benefits of the treatment of psoriasis. *New England J Med* 300(15):852-853.

Falcó, G., J.L. Domingo, J.M. Llobet, A. Teixidó, C. Casas, and L. Müller. 2003. Polycyclic aromatic hydrocarbons in foods: human exposure through the diet in Catalonia, Spain. *Journal of Food Protection* 66(12):2325-2331.

FDA (U.S. Food and Drug Administration). 2001. Letter from Dennis E. Baker, Associate Commissioner for Regulatory Affairs, FDA to Perry M. Gottesfeld, Occupational Knowledge International. Docket No. 00P-1210. February.

Forsberg, N.D., D. Stone, A. Harding, B. Harper, S. Harris, M.M. Matzke, A. Cardenas, K.M. Waters, and K.A. Anderson. 2012. Effect of Native American fish smoking methods on dietary exposure to polycyclic aromatic hydrocarbons and possible risks to human health. *Journal of Agricultural and Food Chemistry* 60:6899-6906.

Goon, D., N.S. Hatoum, M.J. Klan, J.D. Jerniganm and R.G. Farmer. 1991. Oral bioavailability of "aged" soil-adsorbed benzo[a]pyrene (BaP) in rats. *Toxicologist* 11:1356.

Gron, C., A. Oomen, E. Weyand, and J. Wittsiepe. 2007. Bioaccessibility of PAH from Danish soils. *Journal of Environmental Science and Health, Part A.* 42:1233-1239.

Hannukesela-Svahn, A., E. Pukkala, E. Läärä, K. Poikolainen, and J. Karvonen. 2000. Psoriasis, its treatment, and cencer in a cohort of Finnish patients. *The Journal of Investigative Dermatology*114(3):587-590.

Ibáñez, R., A. Agudo, A. Berenguer, P. Jakszyn, M. Tormo, M. Sanchez, J. Quiros, G. Pera, C. Navarro, C. Martinez, N. Larranaga, M. Dorronsoro, M. Chirlaque, A. Barricarte, E. Ardanaz, P. Amiano, and C. Gonzalez. 2005. Dietary Intake of Polycyclic Aromatic Hydrocarbons in a Spanish Population. *Journal of Food Protection*, Vol. 68, No. 10, 2005, Pages 2190–2195.

ICF Consulting. 2000. *Estimation of Lifetime Skin Cancer Risk from the Use of Coal Tar-Containing Shampoos.* Prepared by The K.S. Crump Group, Inc. and ICF Consulting. 602 East Georgia Street, Ruston, Louisiana 71270. Reviewed by TERA Peer Review Panel. September.

IADN (Integrated Atmospheric Deposition Network). 2007. *Temporal and Spatial Trends of Atmospheric Toxic Substances near the Great Lakes: IADN Results through 2003.* Environmental Canada and the U.S. Environmental Protection Agency. ISBN: 978-0-662-45561-5. Available at: http://publications.gc.ca/site/eng/308345/publication.html.

International Agency for Research on Cancer (IARC). 2010. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 92. Some Non-heterocyclic Polycyclic Aromatic Hydrocarbons and Some Related Exposures.* World Health Organization (WHO), Available at: http://monographs.iarc.fr/ENG/Monographs/vol92/mono92.pdf

IEPA. 2007. Appendix A, Table H renumbered to Appendix A, Table I and new Appendix A, Table H added at 31 Ill. Reg. 4063, effective February 23, 2007.

Interstate Technology & Regulatory Council (ITRC). 2008. *Use of Risk Assessment in Management of Contaminated Sites. RISK-2.* Washington, D.C. Risk Assessment Resources Team. www.itrcweb.org.

Jemec G.B.E. and A. Østerlind. 1994. Cancer in patients treated with coal tar: a long-term follow up study. *J Eur Acad Dermatol Venereol* 3:153-156.

Jones, S.K., R.M. Mackie, D.J. Hole, and C.R. Gillis. 1985. Further evidence of the safety of tar in the management of psoriasis. *British Journal of Dermatology* 113:97-101.

Kazerouni, N., R. Sinha, C. Hsu, A. Greenberg, and N. Rothman. 2001. Analysis of 200 food items for benzo[a]pyrene and estimation of its intake in an epidemiologic study. *Food and Chemical Toxicology* 39:423-436.

Knize, M.G., C.P. Salmon, P. Pais, and J.S. Felton. 1999. Food Heating and the Formation of Heterocyclic Aromatic Amine and Polycyclic Aromatic Hydrocarbon Mutagens/Carcinogens. In: *Impact of Processing on Food Safety*, edited by Jackson et al. Kluwer Academic / Plenum Publishers, New York.

Koganti, A., D.A. Spina, K. Rozett, B-L Ma, E.H. Weyand, B.B. Taylor, and D.M. Mauro. 1998. Studies on the applicability of biomarkers in estimating the systemic bioavailability of polynuclear aromatic hydrocarbons from manufactured gas plant tar-contaminated soils. *Environmental Science and Technology* 32:3104-3112.

Kroese, E.D., J.J.A. Muller, G.R. Mohn, P.M. Dortant, and P.W. Wester. 2001. Tumorigenic effects in Wistar rats orally administered benzo[a]pyrene for two years (gavage studies): implications for human cancer risks associated with oral exposure to polycyclic aromatic hydrocarbons. Rijksinstituut voor Volksgezondheid en Milieu. Report nr. 658603010. November.

Larko, O. and G. Swanbeck. 1982. Is UVB treatment of psoriasis safe? A study of extensively UVB-treated psoriasis patients compared with a matched control group. *Acta Derm Venereol* 62:507-12.

Larsson, B.K., G.P. Sahlberg, A.T. Eriksson, and L.A. Busk. 1983. Polycyclic aromatic hydrocarbons in grilled goods. *Journal of Agriculture and Food Chemistry* 31:867-873.

Lewis, R.G., C.R. Fortune, R.D. Willis, D.E. Camann, and J.T. Antley. 1999. Distribution of pesticides and polycyclic aromatic hydrocarbons in house dust as a function of particle size. *Environ. Health Perspect.* 107: 721-726.

Li, A., T.M. Schoonover, Q. Zou, F. Norlock, P.A. Scheff, and R.A. Wadden. 2005. *Research Report: Polycyclic Aromatic Hydrocarbons in the Air of Ten Chicago Area Homes*. NUATRC Research Report No. 6:1-104. National Urban Air Toxics Research Center.

Lin, A.N. and K. Moses. 1985. Tar revisited, *Int J Dermatol* 24:216-218.

Lindelof, B. and B. Sigurgeirsson. 1993. PUVA and cancer: A case-control study. *Br J Dermatol* 129:39-41.

Lodovici, M., P. Dolara, C. Casalini, S. Ciappellano, and G. Testolin. 1995. Polycyclic aromatic hydrocarbon contamination in the Italian diet. *Food Addit. Contam.* 12(5):703-713.

Mackenna, R.M.B. 1959. Uncomplicated psoriasis. *Br Med J* December 1959:244-1247.

Maertens, R.M., J. Bailey, and P.A. White. 2004. The mutagenic hazards of settled house dust: A review. *Mutat Res* 567(2-3):401-425. [PubMed: 15572288]

Magee, B.H., Dolan, D.G., Paley, D.A., and Weyand, E. 1999. Benzo[a]pyrene bioavailability from residential soils. *The Toxicologist* (48):11.

Mahler, B.J., P.C. Van Metre, J.T. Wilson, M. Musgrove, T.L. Burbank, T.E. Ennis, and T.J. Bashara. 2010. Coal-tar-based parking lot sealcoat: An unrecognized source of PAH to settled house dust. *Environ Sci Technol* 44(9):894-900.

Massachusetts Department of Environmental Protection (MADEP). 2002. *Technical Update: Background Levels of Polycyclic Aromatic Hydrocarbons and Metals in Soil.*

Maughan, W.Z., S.A. Muller, H.O. Perry, M.R. Pittelkow, and P.C. O'Brien. 1980. Incidence of skin cancers in patients with atopic dermatitis treated with coal tar. *Am Acad Dermatol* 3(6):612-615.

Menter, A. and D.L. Cram. 1983. The goeckerman regimen in two psoriasis day care centers. *J Am Acad Dermatol* 9:59-65.

Mitra, S. and B. Ray. 1995. Patterns and sources of polycyclic aromatic hydrocarbons and their derivatives in indoor air. *Atmospheric Environment* 29(22):3345-3356.

Muller, S.A. and R.R. Kierland. 1964. Crude coal tar in dermatologic therapy. *Mayo Clin Proc* 39:275-280.

Muller, S.A., H.O. Perry, M.R. Pittelkow, W.Z. Maughhan, and P.C. O'Brien. 1981. Coal tar, ultraviolet light, and cancer. *J Am Acad Dermatol* 4(2):234-235.

Muller, S.A. and H.O. Perry. 1984. The Goeckerman treatment in psoriasis: six decades of experience at Mayo Clinic. *Cutis* 34:265-269.

Murkerjee, S., W.D. Ellenson, R.G. Lewis, R.K. Stevens, M.C. Sommerville, D.S. Shadwick, and R.D. Willis. 1997. An environmental scoping study in the lower Rio Grande valley of Texas – III. Residential microenvironmental monitoring for air, house dust, and soil. *Environ Int* 23(5):657-673.

Neal, J. and R.H. Rigdon. 1967. Gastric tumors in mice fed benzo(a)pyrene: A quantitative study. *Texas Rep Biol Med* 25:553.

Northcross, A.L., M. Trinh, J. Kim, I.A. Jones, M.J. Myers, D.D. Dempsey, N.L. Benowitz, and S.K. Hammond. 2012. Research Report: Particulate mass and polycyclic aromatic hydrocarbons exposure from secondhand smoke in the back seat of a vehicle. *Tobacco Control* 0:1-7.

Perry, H.O., C.W. Soderstrom, and R.W. Schulze. 1968. The Goeckerman treatment of psoriasis. *Arch Dermatol* 98:178-182.

Phillips, D.H. 1999. Polycyclic aromatic hydrocarbons in the diet. *Mutation Research* 443:139–147.

Pittelkow, M.R., H.O. Perry, S.A. Muller, W.Z. Maughan, and P.C. O'Brien. 1981. Skin cancer in patients with psoriasis treated with coal tar. *Arch Dermatol* 117:465-468.

Ramesh, A., S.A. Walker, D.B. Hood, M.D. Guillen, K. Schneider, and E.H. Weyand. 2004. Bioavailability and risk assessment of orally ingested polycyclic aromatic hydrocarbons. *Int J of Toxicol* 23:301-333.

Roelofzen, J., K. Aben, U. Oldenhof, P. Coenraads, H. Alkemade, P. van de Kerkhof, P. van der Valk, and L. Kiemeney. 2010. No increased risk of cancer after coal tar treatment in patients with psoriasis or eczema. *Journal of Investigative Dermatology* 130: 953.

Sawicki, E., T.R. Hauser, W.C. Elbert, F.T. Fox, and J.E. Meeker. 1962. Polynuclear aromatic hydrocarbon composition of the atmosphere in some large American cities. *American Industrial Hygiene Association Journal* 23(2):137-144.

Slezakova, K., D. Castro, M.C. Pereira, S. Morais, C. Delerue-Matos, and M.C. Alvim-Ferraz. 2009. Influence of tobacco smoke on carcinogenic PAH composition in indoor PM_{10} and $PM_{2.5}$. *Atmospheric Environment* 43:6376-6382.

Steuer, J., W. Selbig, N. Hornewer, and J. Prey. 1997. *Sources of Contamination in an Urban Basin in Marquette, Michigan, and an Analysis of Concentrations, Loads, and Data Quality*. U.S. Geological Survey Water Resources Investigations Report 97-4242, Middleton, Wisconsin, 25 pp.

Torinuki, W. and H. Tagami. 1988. Incidence of skin cancer in Japanese psoriatic patients treated with either Methoxsalen Phototherapy, Goeckerman Regimen, or both therapies. *J Am Acad Dermatol* 18:1278-1281.

University of New Hampshire Stormwater Center (UNHSC). 2010. *Final Report. 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.* December.

U.S. Environmental Protection Agency (USEPA). 1982. *An Exposure and Risk Assessment for Benzo[a]pyrene and Other Polycyclic Aromatic Hydrocarbons*. Office of Water Regulations and Standards (WH-553). EPA 440/4-85-020. Washington, DC. July.

USEPA. 1989. *Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual, Part A.* Office of Emergency and Remedial Response, Washington, D.C. EPA/540/1-89-002. December.

USEPA. 1991a. *Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual, Part A, Supplemental Guidance: "Standard Default Exposure Factors".* Office of Solid Waste and Emergency Response. Washington, D.C. OSWER Directive 9285.6-03. March.

USEPA. 1991b. *Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions. Office of Solid Waste and Emergency Response.* Washington, D.C. OSWER Directive 9355.0-30. April.

USEPA. 1992. *Supplemental Guidance to RAGS: Calculating the Concentration Term.* Office of Solid Waste and Emergency Response, Washington, D.C. May.

USEPA. 1994a. *Evaluation of the HVS3 Sampler for Sampling Polycyclic Aromatic Hydrocarbons and Polychlorinated Biphenyls.* EPA 600/R-94/188. Research Triangle Park, NC.

USEPA. 1994b. *Development and Evaluation of Monitoring Methods for Polycyclic Aromatic Hydrocarbons in House Dust and Track-in Soil.* EPA 600-R-94/189. Research Triangle Park, NC.

USEPA. 1996. *Soil Screening Guidance: User's Guide.* Office of Solid Waste and Emergency Response, Washington, D.C. OSWER 9355.4-23. July.

USEPA. 1997. *Exposure Factors Handbook.* Office of Research and Development, National Center for Environmental Assessment, Washington, D.C. EPA/600/P95/002Ba. August.

USEPA. 2003. *National Contingency Plan*, 40 CFR Part 300, §300.430. 7-1-03 Edition.

USEPA. 2007. *Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Final Revision.* October. http://www.epa.gov/oswer/riskassessment/ragse/pdf/part_e_final_revision_10-03-07.pdf

USEPA. 2008a. Triad Investigations: New Approaches and Innovative Strategies. Module 1: *Best Practices for Efficient Soil Sampling Designs.* Deana Crumbling, Office of Superfund Remediation & Technology Innovation, and Robert Johnson, Argonne National Laboratory, Environmental Science Division. Workshop June 10, 2008.

USEPA. 2008b. *Polycyclic Aromatic Hydrocarbons (PAHs).* Office of Solid Waste, Washington, D.C. January. Accessed from http://www.epa.gov/osw/hazard/wastemin/minimize/factshts/pahs.pdf

USEPA. 2011. *Exposure Factors Handbook: 2011 Edition.* National Center for Environmental Assessment, Office of Research and Development Washington, D.C. EPA/600/R-09/052F. September.

USEPA. 2012. *Regional Screening Levels. Users Guide.* November. Available at http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/

USEPA. 2013. *IRIS Summary/Toxicological Review for benzo[a]pyrene. Integrated Risk Information System (IRIS).* U.S. Environmental Protection Agency. www.epa.gov/iris

USEPA. undated. *National Primary Drinking Water Regulations. Technical Factsheet on: Polycyclic Aromatic Hydrocarbons (PAHs).* Accessed 7/25/2013 from http://www.epa.gov/ogwdw/pdfs/factsheets/soc/tech/pahs.pdf

USGS. 2003. *Concentrations of Polynuclear Aromatic Hydrobcarbons and Inorganic Constituents in Ambient Surface Soils, Chicago, Illinois: 2001-02. Water-Resources Investigations Report 03-4105.* In cooperation with the Chicago Department of Environment.

Van Metre, P., B.J. Mahler, J.T. Wilson, and T.L. Burbank. 2008. *Collection and Analysis of Samples for Polycyclic Aromatic Hydrocarbons in Dust and Other Solids Related to Sealed and Unsealed Pavement from 10 Cities Across the United States, 2005-07.* U.S. Department of the Interior, U.S. Geological Survey Data Series 361.

Van Schooten, F. and R. Godschalk. 1996. Coal tar therapy: Is it carcinogenic? *Drug Safety* 15(6):374-377.

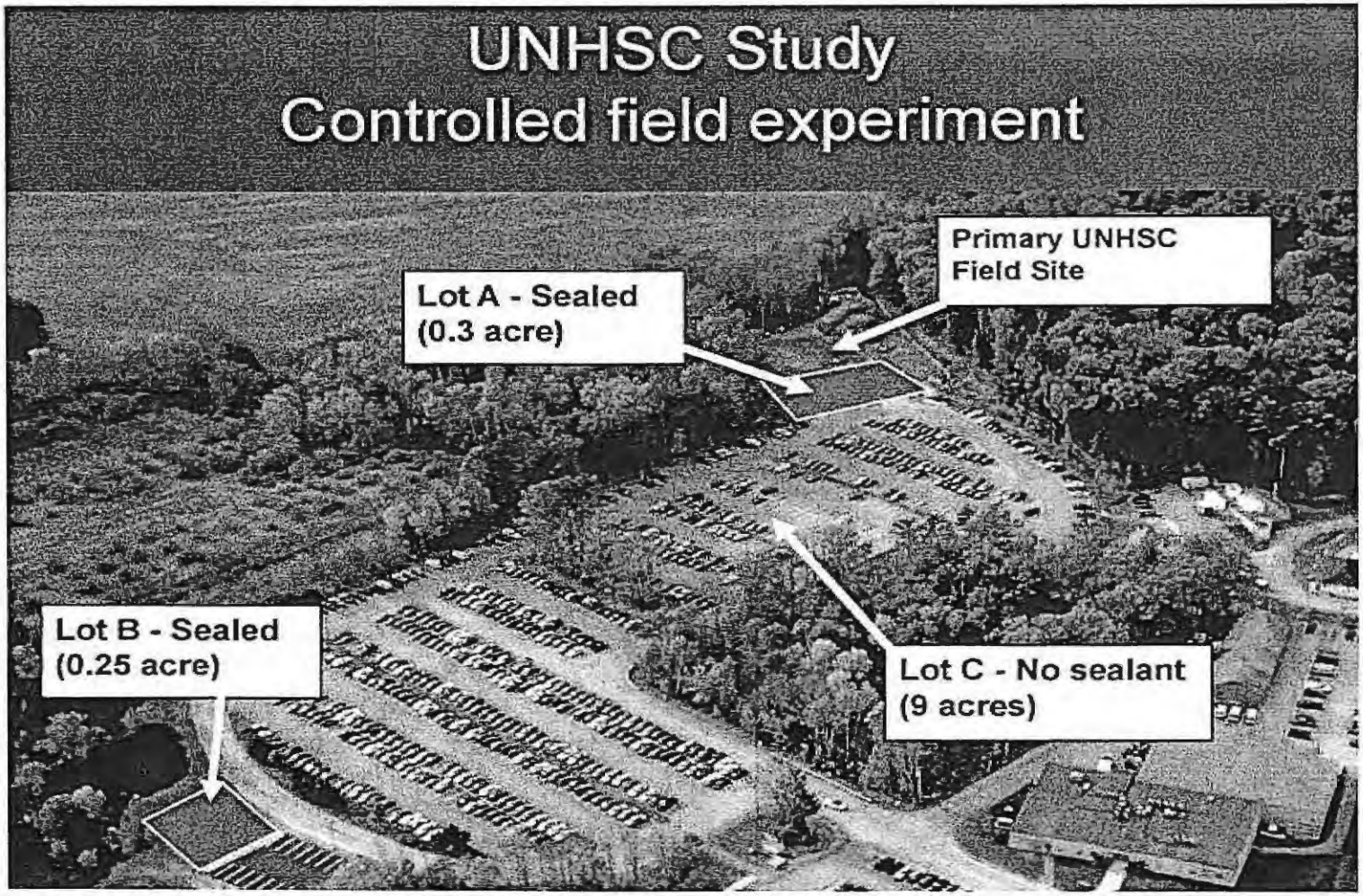
Van Winkle, M.R. and P.A. Scheff. 2001. Volatile organic compounds, polycyclic aromatic hydrocarbons and elements in the air of ten urban homes. *Indoor Air* 11:49-64.

Weyand, E.H., K. Rozett, A. Koganti, and R. Singh. 1996. Effect of Soil on the Genotoxicity of Manufactured Gas Plant Residue. *Fundamental and Applied Toxicology* 30:1, Part 2.

Whitehead, T., C. Metayer, R.B. Gunier, M.H. Ward, M.G. Nishioka, P. Buffler, and S.M. Rappaport. 2011. Determinants of polycyclic aromatic hydrocarbon levels in house dust. *J Expo Sci Environ Epidemiol* 21(2): 123-132.

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. *Environ Sci Technol* 47:1101-1109.

Figures



Note:

Photo from UNHSC 2010

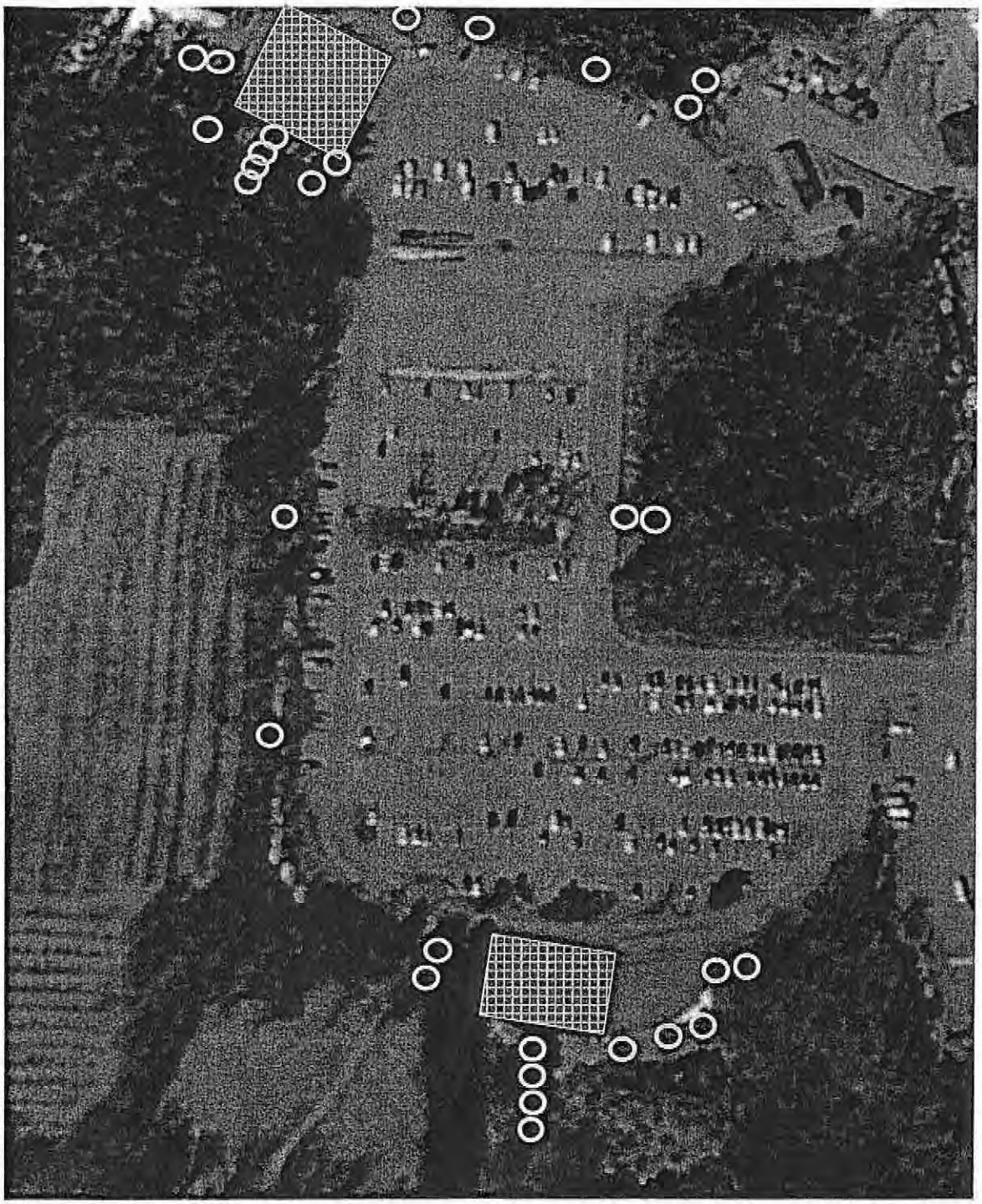
PAVEMENT COATINGS TECHNOLOGY COUNCIL

PEER REVIEW OF COAL-TAR-SEALED PAVEMENT RISK ASSESSMENT

UNHSC Study Parking Lots

ARCADIS

FIGURE 1



Note:

UNH sealed lots shown as

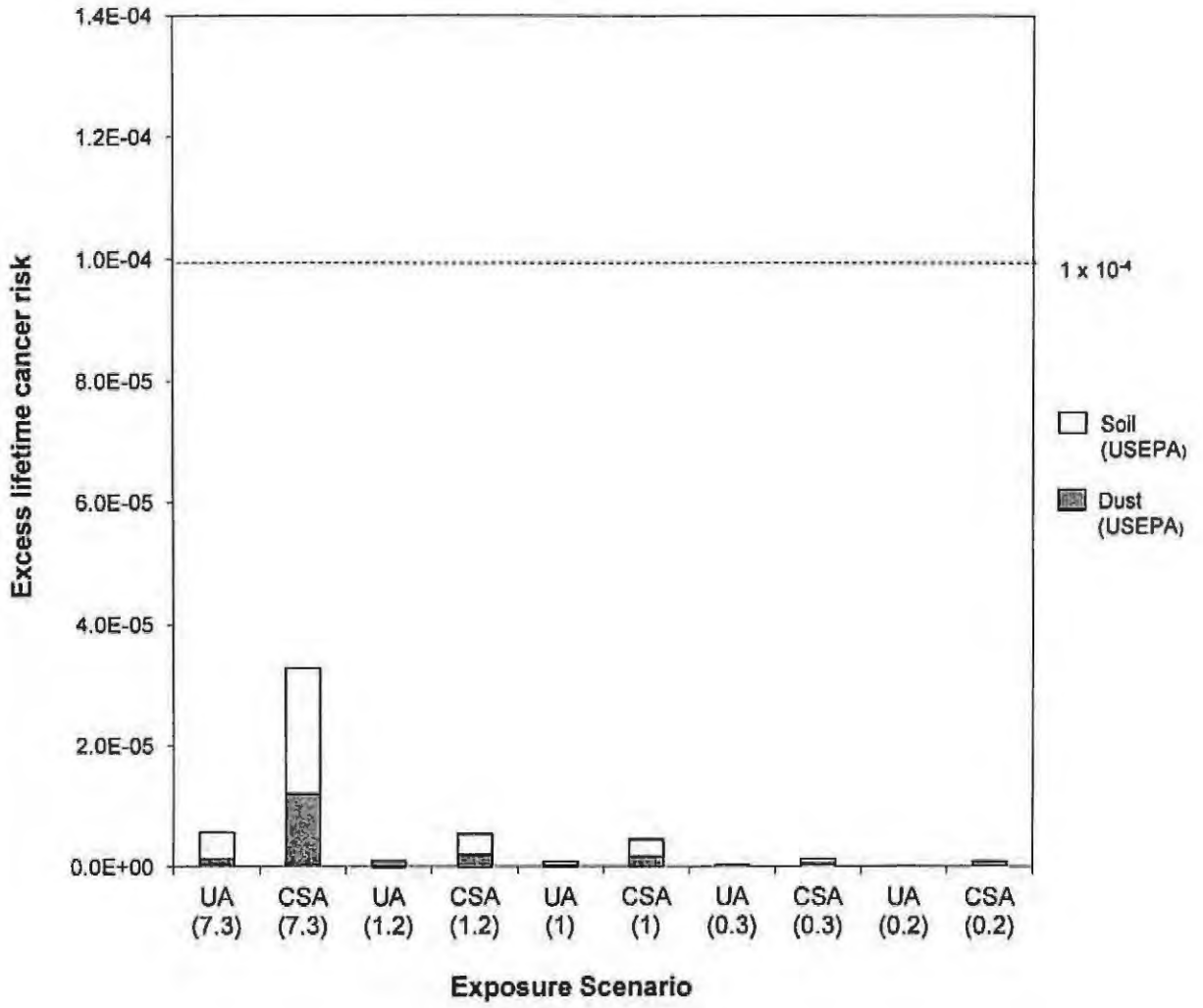
PAVEMENT COATINGS TECHNOLOGY COUNCIL

PEER REVIEW OF COAL-TAR-SEALED PAVEMENT RISK ASSESSMENT

UNHSC (2010) Soil Sample Locations

ARCADIS

FIGURE 2



Note:

Value in parentheses denotes OSF used.

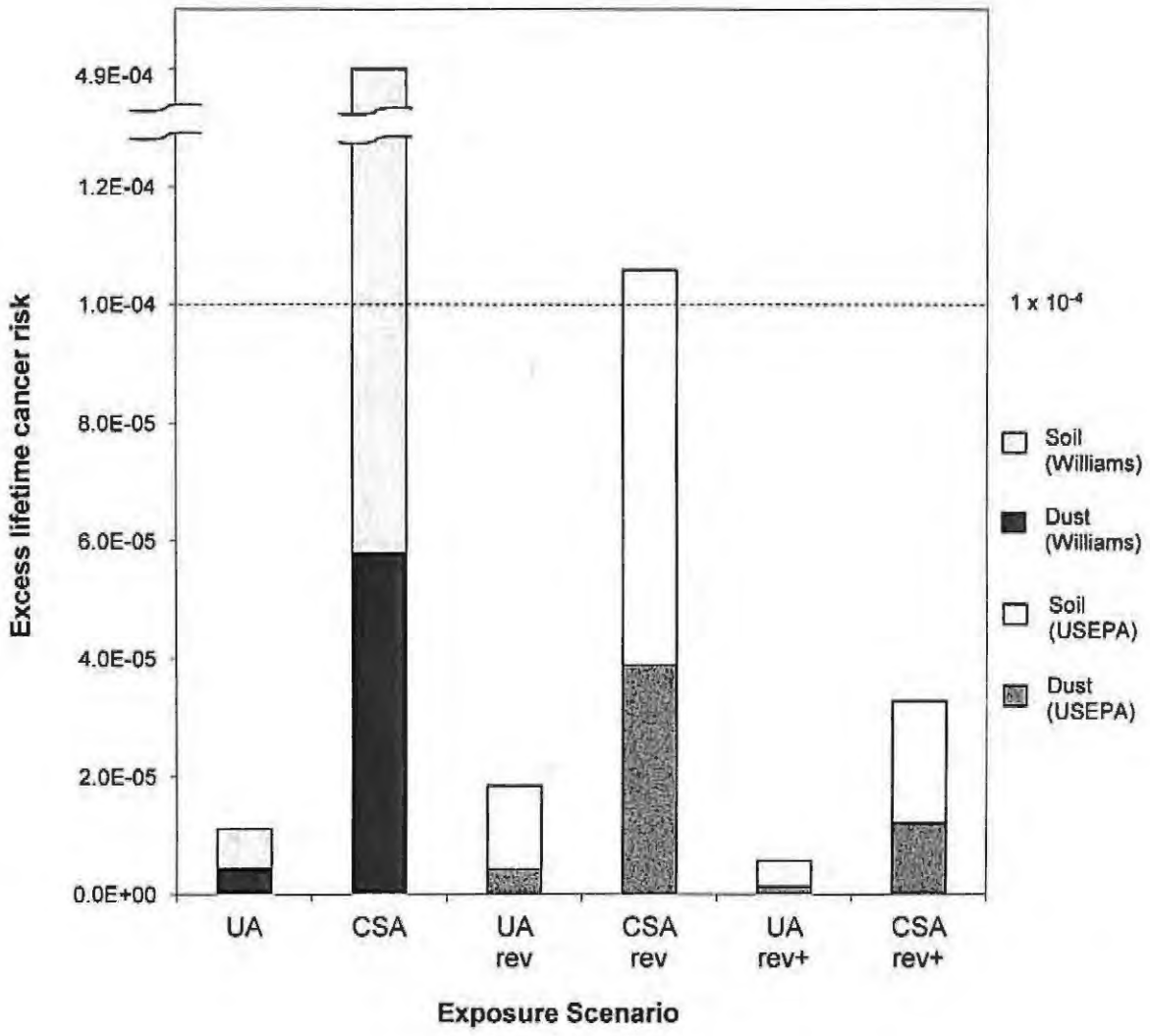
PAVEMENT COATINGS TECHNOLOGY COUNCIL

PEER REVIEW OF COAL-TAR-SEALED PAVEMENT RISK ASSESSMENT

Risk Estimates Over the Range of BaP OSFs

ARCADIS

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

ARCADIS

FIGURE 4

ARCADIS

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

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Advisers Try To Clarify EPA's Risk Assessment Approach For PAH Mixtures

Posted: October 6, 2010

EPA's science advisers continue to raise concerns about the agency's planned approach for assessing the cancer risk from complex mixtures of polycyclic aromatic hydrocarbons (PAHs), such as coal tar, and are seeking clarifications on how the agency will calculate the cancer values in the approach, saying the proposed methodology may overestimate risk.

A panel of EPA's Science Advisory Board (SAB) are raising concerns about applying the approach to complex PAH mixtures, calling on the agency to develop a more robust approach within the next 10 years in a Sept. 8 draft report of their recommendations. The report follows a critical peer review meeting the panel held in June.

In the interim, the draft report recommends that EPA make significant revisions to the methodology they used to calculate relative potency factor (RPF) values for assessing carcinogenicity of PAH compounds, saying the approach the agency is taking may overestimate cancer risks.

PAHs are an ubiquitous class of chemicals found across a wide range of natural and industrial sources, including crude oil, asphalt, and vehicle emissions. EPA's National Center for Environmental Assessment (NCEA) in February published its draft approach for how to assess the risks of PAH mixtures, titled "Development of a Relative Potency Factor (RPF) Approach for PAH Mixtures."

The RPF approach --similar to the approach used to assess dioxins -- is used to calculate a cancer potency factor for each PAH relative to one index chemical, benzo(a)pyrene (BaP). NCEA selected BaP as the reference chemical because it is among the most studied of the PAHs. Once the formula is finalized, regulators will use it to calculate limits for a range of media exposures, including water contamination from runoff from roads, railroads. In the Gulf region, for example, EPA in May set health-based screening levels for PAHs, including BaP, concerned that the chemicals could represent potential inhalation exposure risks as weathered oil from the BP spill reaches the coastline and evaporates.

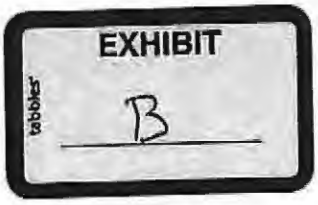
Though the panel had strong criticism for EPA's draft approach, it agreed with EPA that the limited data available did not make a whole mixtures-based approach feasible at this time. Still, the draft report suggests that the agency set the goal of developing such an approach within the next five to 10 years, "as a strategic initiative, with a specific timeline and benchmarks, that lays the foundation for an underlying concerted research program to achieve this goal. The Panel recommends that the Agency seek support from the National Toxicology Program (NTP) and or other entities to test a portfolio of 12-15 different complex PAH mixtures, using in vivo tumor studies. These complex PAH mixtures should represent a diverse array of mixtures, but also represent the most important PAH mixture classes of concern to EPA."

Weighting Data Quality

The panel discussed their draft report during a Sept. 30 conference call, focusing on what types of data should be factored into the derivation and how to weight data quality. The panel also discussed whether single dose studies, or single experiments should be used in the calculations -- one part of NCEA's proposal which some panel members said could result in high cancer potency values. "The panel is also concerned with extraordinarily high RPF values that were calculated from only a limited number of bioassays," the draft report said.

EPA's planned approach for deriving RPFs for individual PAHs by averaging together tumor incidence data with tumor multiplicity data also met with criticism from the panel. The panelists' draft report indicates tumor multiplicity data should only be used in calculating RPF values when bolstered by additional studies showing dose-response information, allowing for a comparison of relative differences between the compounds. Tumor multiplicity refers to the number of induced tumors in a study.

"You have to have a dose-response to be able to accurately use the tumor multiplicity data," panel member John DiGiovanni, of the University of Texas at Austin, said on the call.



Lynn Flowers, NCEA's acting associate director for health, told the panelists that NCEA needed additional clarification on what the panel wants in terms of weighting the quality of data used to select the PAHs.

"If it's not high quality, you don't include it," panel member Marile Gammon, of University of North Carolina at Chapel Hill, said during the call. Gammon asked for an a prior set of rules that NCEA would use in terms of data quality information, including sample size, dosing, and whether or not the data are derived from tumor multiplicity. During the SAB panel peer review in July, some members expressed concerns that EPA's planned approach might exclude some potentially carcinogenic PAHs if the data didn't fit into statistical models.

Another panel member, Joshua Hamilton, of the Marine Biological Laboratory, voiced concern during the call about the feasibility of an pre-determined list of rules because there was no way to predict what types of studies might be available for the various PAHs.

Another concern about the draft approach raised in the draft report is that NCEA is proposing to calculate RPFs with only those studies where BaP was tested concurrently with the PAH under consideration. The panel is recommending a "daisy-chain" approach that would require EPA to use studies where BaP had been tested as a "surrogate" for studies where BaP was not tested concurrently. "This may allow for additional quality studies to be included. The panel recommends that this be examined especially in those instances where limited tumor data were used to establish an RPF value," according to the draft report.

Nancy Kim, who chairs the SAB panel, tentatively set a deadline of December for the completed draft report. -- *Bridget DiCosmo*

Related News: Toxics

2340820

Review of "Coal-Tar-Based Parking Lot Sealcoat: An Unrecognized Source of PAH to Settled House Dust" by Mahler et al., published in *Environmental Science and Technology*, January 2010

Prepared by:
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April 19, 2010

Review of Mahler et al. (2010) Study Design

The Mahler et al. (2010) study describes an evaluation of polycyclic aromatic hydrocarbons (PAHs) in indoor and outdoor dust collected from apartments and their associated parking lots. Of 23 apartments tested, Mahler et al. (2010) determined that 11 had asphalt parking lots with coal-tar-based sealant and the remaining 12 had concrete or asphalt parking lots that were unsealed or coated with an asphalt-based sealant. Coal-tar-based sealcoated parking lots are referred to as "CT" while non-coal-tar-sealcoated parking lots are referred to as "NCT."

Mahler et al. (2010) found median total PAH concentrations of 4,760 µg/g and 9.0 µg/g in dust collected from CT and NCT parking lots, respectively. Median total PAH concentrations of 129 µg/g and 5.1 µg/g are reported for indoor dust collected from CT and NCT apartments, respectively. The presence or absence of CT parking lots was reported to explain 48 percent of the variance in log-transformed total PAH concentrations in indoor dust. Other factors including land use, frequency of vacuuming, indoor burning, and more also were evaluated. Of these other factors, Mahler et al. (2010) report that only urban land use intensity near the sampled apartment has a significant relationship with total PAH concentrations.

General comments regarding study design are listed here, followed by additional discussion in subsequent sections:

- Lack of precision in selection of sample locations contributes to variability between the sampled areas and consequently, uncertainty regarding external influences when evaluating the results
- Small sample size (especially given lack of precision in sample location selection)
- Particle size fraction evaluated not appropriate for dermal and ingestion exposures
- Dust loading not evaluated
- Incomplete evaluation of independent variables

Analytical Methods

The analytical method used is considered appropriate for analysis of PAH concentrations; although, some analytical difficulties were encountered, preventing quantitation of dibenz[a,h]anthracene in all but one sample. A summary of quality assurance/quality control (QA/QC) data is provided in the Supplementary Information; however, the raw QA/QC data are not presented. This information would be required for a proper evaluation of data quality. For example, the authors report that individual PAH compounds were detected in blank samples more than 20% of the time, but no information is presented to identify which samples were associated with contaminated blank samples.

Sampling Methodology

Mahler et al. (2010) collected 23 indoor and outdoor dust samples between April and July 2008. No detailed information is provided regarding how sample locations were selected other than presence or absence of CT parking lots. Due to a lack of site selection or exclusion criteria other than presence or absence of CT parking lots, other potential factors affecting PAH concentrations in parking lot dust may have been overlooked or unaccounted for. For example, little to no information is presented to support the classification of CT parking lots, parking lot

selection criteria, or sample location selection criteria – all of which can affect the variability of the data. Site selection appears to have been based solely on "coffee/tea" field screening tests. The "coffee/tea" test is not a standard, validated method so its accuracy in identifying CT parking lots is uncertain.

There is no indication that interviews with apartment maintenance staff and owners and/or review of maintenance records were conducted. Such interviews and records review could not only confirm the use of coal-tar-based sealant, but also provide useful information on lapsed time since sealcoat application, frequency of application, application formulation, and other maintenance history of potential relevance. This information would help to confirm the presence or absence of coal-tar-based sealant as well as optimize uniformity of the sample locations by selecting those with the most similar application and maintenance history.

Also, no criteria are provided for selection of specific sample locations within each parking lot other than avoidance of painted areas and drip lines. The number of days since last rainfall or washing event, traffic and runoff patterns, number of parking lot stalls/cars, and the location of stains, cracks, and debris in each parking lot would help guide when and where to collect samples at each lot. Again, use of this information to inform site selection and timing of sample collection would reduce uncertainties associated with comparability of CT and NCT data. The timing of sample collection with respect to rainfall and washing events is particularly important given the extended, three-month duration of the sample collection period. More precise timing of the sampling event would reduce bias introduced due to sampling during variable weather conditions. For example, little to no rainfall was recorded between February and April of the year the sampling was conducted. This could have resulted in an accumulation of dust at the beginning of the sampling program. In April, heavier and more frequent rainfall was experienced which then decreased in volume and frequency each successive month over the course of the sampling event until its completion in July. Again, depending on the timing of the sample collection, the changing weather could have introduced variability in the dust data.

Similarly, no criteria are provided for selection of apartments other than presence or absence of CT parking lots. Additional criteria such as apartment age, flooring type and age, and period of time occupied by current owner could be used to obtain as uniform a sample population as possible and thereby improve comparability between samples. This is especially useful for small sample sizes where the influence of variable apartment and flooring characteristics as well as influence of previous owners (if newly occupied) will have a greater effect than in larger sample sizes.

The Supplementary Information indicates that the apartment complex build dates range from 1961 to 2007 with a median date of 1978. This indicates that about half of the apartments were more than 30 years old. Although no information is presented to determine the relative age of CT apartments compared to NCT apartments; certain statements in the paper suggest that NCT apartments are much newer. For example, in the second paragraph of the discussion section, the authors explain differences in dust concentrations from parking lots with asphalt-based sealcoat measured in this study with levels detected in a previous study by implying the lots in this study were newer.

> "The difference likely is because the asphalt-based sealcoat on the lots tested by [another study] had been applied over worn coal-tar sealcoat, whereas the asphalt-based sealcoat on the parking lots tested for this study had been applied over new asphalt pavement."

The presence of new asphalt pavement suggests that the associated apartments are also newer. Also, later in the paper when the authors are discussing potential contributions from coal-tar based flooring adhesives[1], the following statement is made:

> "Of the four other NCT apartments in this study with wood laminate or linoleum flooring, only one was built prior to 2000 and it did not have elevated T-PAH concentrations."

Thus, it appears that the NCT apartments represent newer housing stock compared to CT apartments. To the extent that older apartments (and carpets) reflect longer-term accumulation of PAHs, if for example the apartment is located nearby a heavily travelled roadway, then apartment age may be a significant variable that has not been evaluated.

Field replicate samples were collected at two indoor locations and one outdoor location. These samples were used as measures of sample variability. However, it appears that no field rinsate samples were collected as part of QA/QC procedures. Given the elevated levels of PAHs observed at CT parking lots, it would be useful to evaluate the decontamination process by collecting equipment rinsate samples to verify that the HVS3 was adequately decontaminated between samples. This is a legitimate concern, particularly given the frequency of detecting PAHs in the laboratory method blanks. Further, it is always prudent to first collect samples assumed to have lower levels of contamination and then collect samples assumed to have higher levels of contamination, to minimize cross-contamination of field equipment. Dedicated indoor and outdoor vacuums could be used to first sample CT locations then sample NCT locations, thereby reducing the potential for contamination between CT and NCT locations as well as indoor and outdoor locations. Because a standard operating procedure was not provided in the Supplementary Information, it is not known whether or not measures were employed to reduce cross-contamination of samples. At a minimum, it would be advisable to collect NCT samples first, along with an equipment rinsate sample, followed by collection of CT samples and another equipment rinsate sample to minimize the potential for cross-contamination of samples and determine if cross-contamination was an issue.

The high-efficiency vacuum sampler recommended in ASTM Method D 5438 (2005) was stated to have been utilized according to the manufacturer operation manual (CS_3 Inc. 2004). Mahler et al. (2010) state that a sample was collected from an entry way and adjacent living room floors. In the absence of child residents, sample locations recommended by EPA (2008) for assessing lead in indoor dust include the 1) entryway, 2) bed room, and 3) other room most often occupied by the residents. While Mahler et al. (2010) collected a composite sample, EPA (2008) guidance recommends collection of discrete samples within the targeted areas of the residence so that a weighted average dust concentration can be calculated based on the fraction of the day that the resident spends in each area. In this way, areas with low dust loading are not combined with those with higher dust loading and as discussed below, exposure is related to dust loading.

Dust load is expected to be highest at the entryway and in carpets; however, PAH concentrations are expected to be highest at the entryway and lowest in carpets. Since most time will be spent in the living areas rather than at the entryway, composite samples that combine both areas do not represent average exposure concentrations. The composite

[1] The use of tar-based flooring adhesives in Germany is reported by Heudorf and Angerer (2001); however, it is not clear that these products were or are available for use in the United States.

concentrations will overestimate the average exposure concentration both due to higher concentration and due to higher loading.

Mahler et al. (2010) note the range in area sampled among apartments (1.6 to 13 square meters indoors, 2.0 to 7.5 square meters outdoors) but the rationale for this variability is not provided. Presumably, the range in sampled area is due to differences in floor type and loading, i.e., larger areas were sampled as necessary to obtain adequate sample size from bare floors or relatively cleaner floors. However, the relative contribution from different areas within apartments could bias the PAH concentrations high or low, depending on the sampled location and loading at that location. Of particular concern is that there may have been consistent over-representation of high concentration dust from the entryway due to heavy dust loads in that area. A discussion of the dust loading levels at sampled locations would be helpful in understanding the concentration data and perhaps explaining the need for the range in sampled area.

Prior to analysis, Mahler et al. (2010) sieved samples to obtain the size fraction less than 500 µm diameter. For the purpose of estimating potential exposures to dusts, EPA (2004, 2008), CS_3 Inc (2004), and ASTM Method D 5438 (2005) recommend obtaining the size fraction that is most likely to adhere to skin surfaces. EPA (2004, 2008) recommends sieving dust samples and analyzing the portion smaller than 250 µm, while CS_3 Inc. (2004) and ASTM (2005) recommend analyzing the size fraction less than 150 µm in diameter. The size fraction obtained by Mahler et al. (2010) does not represent the particle size most likely to adhere to skin surface and may not provide a realistic estimate of exposure material for dermal absorption and ingestion of dust via hand-to-mouth activity. Typically, contaminant concentrations become enriched as particle size decreases (Lewis et al. 1999). Depending on the particle sizes that best represent sloughed parking lot sealant, the influence of particle size in this dataset is uncertain.

Statistical Approach

Given only the PAH analytical data supplied in the Supplementary Information, ENVIRON was able to verify the summary statistics, but could not verify the influence of independent variables reported in the paper. If made available, the additional information obtained from the study participants and characteristics of the sample locations could be used to confirm the significance of parking lot surface type and land use intensity in explaining the variability of PAH concentrations in dust samples, as well as the lack of significance associated with smoking and distance to the nearest roadway.

Additional variables that should be considered but were not reported in Mahler et al. (2010) include apartment and carpet age and degree of sealcoat wear. The age of the apartment and carpet could be important variables explaining differences in indoor dust concentrations. As previously indicated, there is summary information on the apartment age variable provided in the Supplementary Information; however, this variable is not included in Table 1 of the paper, indicating that it was not evaluated as an independent variable.

In contrast, the degree of sealcoat wear is listed in Table 1 of Mahler et al. (2010) as an independent variable potentially related to the levels of PAH detected in indoor dust and parking lot dust samples. However, there is no information presented as to how this wear was evaluated, nor is there any information in the supporting material that summarizes the range of wear levels for the parking lots examined in the study. If parking lot surface type is believed to be a significant factor in explaining indoor and parking lot dust PAH levels, then one might expect that the degree of sealcoat wear should also be a factor.

Other factors such as the size of the apartment complex or size of the associated parking lot might also be expected to be factor in determining PAH levels in indoor dust, but these data are not presented.

ENVIRON evaluated PAH analytical data available from the Supplementary Information in an attempt to identify patterns in PAHs detected in the CT and NCT samples. No obvious distinction between CT and NCT samples could be discerned from the available data; however, the analytical method used for this study does not allow for a more comprehensive evaluation that could be used to identify unique patterns in the dataset that are specific to the source of the PAHs. From ENVIRON's limited review, observations appear to most closely resemble what would be considered an "urban background" profile as described by Stout et al. (2004).

Metrics for Evaluating Dust Exposure

The variable "days since sampling area last vacuumed" is listed in Table 1 of Mahler et al. (2010) as a variable potentially associated with the level of PAHs in indoor dust. Presumably, this metric is meant to provide a measure of cleanliness in the home. However, a better metric is the actual dust loading in the areas sampled. The information required to calculate dust loadings was collected (i.e. summary information for mass of dust and area sampled is provided); however, these data are not reported and not evaluated.

Both PAH concentration and dust loading from each living area are needed to assess exposures. While PAH concentration is useful in providing the amount of PAH in dust, it does not provide information about the amount of dust that is available on an exposure area or surface. For example, a high concentration of a contaminant in house dust may present a low risk if dust loading is low, or conversely, a low contaminant dust concentration may present a high risk if dust loading is quite high (WTC 2005). Concentration of chemicals in dust, alone, is not adequate for predicting risk.

In most house dust studies involving lead, blood lead levels have been shown to correlate either most closely with lead loading or equally with both lead concentration and lead loading (see review by Adgate et al. 1995). The different results are influenced by a number of factors, including study design, additional sources of lead aside from dust, behavior patterns, bioavailability of the lead in the exposure matrix, and more. For this reason, EPA (2008), ASTM (2005), and CS_3 Inc (2004) recommend evaluating both concentration and loading metrics when evaluating exposures to dust. In this way, the data can be evaluated to obtain the best possible understanding of the chemicals present in dust and potential for exposure of residents.

There also are studies demonstrating that the presence of chemicals in dust have little or no correlation with chemicals measured in humans. For example, Heudorf and Angerer (2001) found no correlation between PAHs measured in house dust and urinary metabolites of PAHs.

University of Michigan researchers studying dioxins in people living in an area contaminated by a manufacturing facility did not find an association between dioxins in house dust / soil and blood dioxin levels (Garabrant et al. 2009). Even though greater concentrations of dioxins were measured in the soil and house dust of homes within the contaminated area compared to a reference area, the primary factors associated with blood dioxin levels were age, history of working at the facility, and fishing and hunting in waterways within the contaminated area (Garabrant et al. 2009). The results of this study demonstrate that many factors must be evaluated to understand exposure to environmental contaminants and no one factor is likely to be responsible for total body burden, given multiple sources and pathways of exposure.

Similar results were obtained in an exposure study of a population in Calcasieu Parish, Louisiana. The Agency for Toxic Substances and Disease Registry (ATSDR) found elevated blood dioxin levels in older residents only and these levels were found to be associated with historical exposures rather than levels measured in their homes (ATSDR 2006). Although these results relate to dioxins rather than PAHs, they remind us that collection of exposure information is important in helping us to understand potential exposure scenarios. In some cases, measured chemical concentrations in our homes are not always correlated with levels of chemicals measured in our bodies. This is particularly likely for chemicals such as PAHs and dioxins which have other pervasive sources of exposure such as diet.

Given the range in indoor areas sampled by Mahler et al. (2010), it could be assumed that dust loading varied substantially among the residences sampled. Evaluation of loading by residence then may provide insight into potential exposures to PAHs in house dust or potential for tracking parking lot dust into homes. However, the presence of PAHs in indoor dust measured via concentration and/or loading does not necessarily equate to risks to residents.

PAH Toxicity

PAHs are a class of compounds consisting of two or more bonded aromatic rings, excluding those compounds with anything other than hydrogen or carbon atoms. PAHs are formed during incomplete combustion of organic materials such as gas, wood, oil, garbage, cigarettes, and grilled or charbroiled foods. Although there are over 100 PAHs, a subset of 16 are routinely evaluated using standard analytical methods. Of these PAHs, seven have been classified as probable human carcinogens (Group B2) (1993; see EPA's Integrated Risk Information System 2010): benzo(a)pyrene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, dibenz(a,h)anthracene, indeno(1,2,3-cd)pyrene, and chrysene. The seven carcinogenic PAHs are referred to here as cPAHs.

Although some chemical mixtures (e.g., tobacco smoke, chimney soot, others) that include PAHs have been shown to be carcinogenic by inhalation or dermal contact in humans, the relative potency of the individual PAH compounds has not been established in humans. The EPA toxicity assessment for benzo(a)pyrene and the other cPAHs has been based on the results of studies in rodents; however, these chemicals have only been shown to be rodent carcinogens at portal of entry sites such as skin or lung.

To quantify the carcinogenicity of the cPAHs, a relative potency of carcinogenicity was assigned to each cPAH with benzo(a)pyrene used as the reference compound (EPA 1993). Benzo(a)pyrene is the most well-studied of the cPAHs (EPA 1993). The carcinogenic potency of each cPAH was estimated relative to benzo(a)pyrene based primarily on comparison of mouse skin tumor data. While skin tumor data from mice for multiple PAHs may allow a comparison across PAHs, the relevance and predictiveness of this test system for oral cancer risk in humans is questionable. Consequently, there is a high degree of uncertainty associated with cancer risk estimates for the cPAHs.

EPA (1993) recommends the following relative potencies for cPAHs[2]:

[2] EPA is currently accepting public comments on revised potency factors provided in an external review draft report published in February 2010. This revised guidance includes additional cPAHs and includes revised potency factors for many of the seven currently listed cPAHs; however, the limitations described

cPAH	Relative potency factors
benzo(a)pyrene	1.0
benzo(a)anthracene	0.1
benzo(b)fluoranthene	0.1
benzo(k)fluoranthene	0.01
dibenz(a,h)anthracene	1
indeno(1,2,3-cd)pyrene	0.1
Chrysene	0.001

Table 2 in Mahler et al. (2010) lists analytical results separately as the sum of total PAHs (16 PAH compounds), benzo(a)pyrene, and sum of cPAHs. However, the cPAH concentrations have not been modified by their relative potencies to benzo(a)pyrene. This means that the cPAH concentrations provided in Table 2 are not equivalencies of benzo(a)pyrene and should not be compared to health- or risk-based values that are based on equivalent concentrations of benzo(a)pyrene. Evaluating the sum of bulk cPAHs without adjustment for relative potency to benzo(a)pyrene results in an assumption that all cPAHs are as potent as benzo(a)pyrene when in fact, that is not the case (EPA 1993).

When evaluating the indoor and outdoor dust concentrations, it is useful to adjust the cPAH concentrations by their relative potencies to better understand the potential risks associated with cPAHs in dust. Concentrations of cPAHs provided by Mahler et al. (2010; see Supplementary Information) were modified by their respective potency factors to obtain total cPAH concentrations presented as a benzo(a)pyrene equivalent concentrations, or benzo(a)pyrene equivalents, for each sample using the following equation:

$$BaPE = \sum_{i=cPAH}^{n=7} C_i \times RPF_i$$

Where:

BaPE = Concentration of cPAHs as benzo(a)pyrene equivalent

C_i = Concentration of individual cPAH

RPF_i = Relative potency factor for each respective cPAH

i = Each of 7 individual cPAHs

in the text related to uncertainty associated with extrapolating from rodent portal of entry data to human oral cancer risk have not been addressed by EPA.

The benzo(a)pyrene equivalent concentrations calculated for samples collected by Mahler et al. (2010) are provided in the following table.

cPAH concentrations as benzo(a)pyrene equivalents (BaPE) in indoor dust.

Parking Lot Status	Sum of bulk cPAHs (µg/g)	BaPE (µg/g)
CT	22	4.5
CT	94	20
CT	70	15
CT	29	5.7
CT	104	20
CT	8.6	1.7
CT	18	2.3
CT	54	10
CT	30	6.1
CT	47	6.8
CT	156	32
NCT	0.99	0.21
NCT	9.4	1.9
NCT	22	5.0
NCT	3.2	0.75
NCT	8.5	2.0
NCT	12	2.8
NCT	86	17
NCT	0.47	0.085
NCT	1.8	0.36
NCT	1.7	0.31
NCT	2.3	0.63
NCT	1.6	0.34

Regulatory risk assessments rely on the use of benzo(a)pyrene equivalent concentrations to estimate potential risk from exposure to cPAHs. Although it is preferable to evaluate whole mixtures (refined coal-tar-based sealant) as opposed to individual components (cPAHs as benzo(a)pyrene equivalents), the lack of toxicity information specific to whole mixtures led to the development of relative potency methods. This approach does not take into account interactions between individual components of chemical mixtures and also necessitates the use of assumptions about the toxicity of individual compounds relative to a reference compound. In the absence of an alternative method, use of benzo(a)pyrene equivalent concentrations is the accepted method for evaluating risks associated with exposure to cPAHs.

Risk assessment is a tool used to evaluate and manage potential risks from exposure to chemicals. This tool combines assumptions about reasonable maximum exposures of a population (e.g., contact rates, bioaccessibility, duration of exposure, body weight, etc) with measured or modeled data for exposure media (e.g., chemical concentrations in soil, dust, air, water, food, etc) to obtain an estimate of a daily or lifetime intake level for a population. Next,

this intake level is combined with a quantitative estimate of a chemical's toxicity to obtain an estimated cancer risk or non-cancer hazard. To be protective of more sensitive members of the population, this risk assessment model is intended to overestimate risks rather than underestimate risks.

EPA's National Oil and Hazardous Substances Pollution Contingency Plan guidance (March 8,1990; 40 CFR 300) identifies estimated cancer risks falling between 1×10^{-6} and 1×10^{-4} (or, one additional cancer case per million people and one additional cancer case per ten-thousand people) as within an acceptable risk management range. At sites where cancer risks exceed 1×10^{-4}, a remedial action is considered. In some cases, remedial action may be determined to be unnecessary when risks are slightly greater than 1×10^{-4} or considered necessary when risks are less than 1×10^{-4} (EPA 1991). In this way, risk assessment informs the remedial investigation process and can also be used to evaluate the effectiveness of remediation alternatives if cleanup is warranted.

Risk assessment is not, however, a tool used to predict the incidence of cancer or non-cancer health effects. Exceedance of a risk management guideline, including risk-based screening levels, does not indicate that exposure-related illness will occur. Instead, exceedance of the guidelines indicates that further investigation may be necessary to confirm that appropriate assumptions were incorporated into the risk analysis and/or indicates that cleanup may be recommended.

Comparison to Health-based Standards

As noted by Mahler et al. (2010), there is no regulatory standard for PAHs in indoor or outdoor dust. For lack of a criterion to evaluate the dust data, Mahler et al. (2010) relied on a German Federal Environmental Agency (FEA) value of 10 µg/g for benzo(a)pyrene, established by their Commission for Indoor Air Quality. As discussed below, the FEA value is not a health-based criterion. Consequently, ENVIRON evaluated the applicability of a health-based criterion developed by the World Trade Center (WTC) Indoor Air Task Force Working Group (2003) for cPAHs. The criteria established by the Germany FEA and WTC working group are discussed below.

German FEA Standard

Mahler et al. (2010) rely on a German FEA action level of 10 µg/g for benzo(a)pyrene in household dust. This level was developed in response to concerns regarding coal-tar parquet glues commonly used in homes built in the 1950's and 1960's. In Heudorf and Angerer (2001), 1,213 residents of 511 homes were recruited to evaluate coal-tar in flooring glue. Following analysis of PAH metabolites in urine and benzo(a)pyrene in indoor dust and parquet floor glue, no relationship was observed between levels of PAHs in urine and dust or glue. Also, no difference in PAH levels was observed between homes with and without the suspect parquet flooring. Based on these results, it was not possible for the German FEA to develop an exposure-based limit for PAH contamination in parquet flue and house dust. Heudorf and Angerer (2001) states that the German FEA could not define a threshold limit value below which there would be no risk to residents contacting PAHs in coal-tar-based parquet glue. Instead, the FEA selected the value of 10 µg/g as the maximum limit of benzo(a)pyrene in house dust in an attempt to minimize exposure of residents. Heudorf and Angerer (2001) state this limit applies to benzo(a)pyrene but do not discuss comparison of all cPAHs to this limit. In Mahler et al. (2010), benzo(a)pyrene levels in indoor dust at 4 of 11 CT locations and 1 of 12 NCT locations exceed the German FEA action level of 10 µg/g.

This action level is not a health-based value and is not useful in gaining an understanding of potential health risks associated with exposure to the PAH concentrations measured by Mahler et al. (2010). Based on the results presented by Heudorf and Angerer (2001), exceedance of the FEA value does not provide information about residential exposure or risk levels. For this reason, ENVIRON considered an alternate screening value based on standard risk and exposure assessment assumptions.

World Trade Center Criterion

Multiple federal, state, and local agencies collaborated on development of indoor air and dust screening criteria for chemicals of potential concern, including cPAHs, in an effort to assess environmental health conditions of residences in the vicinity of the collapsed World Trade Center buildings (see WTC 2003). The EPA-led effort resulted in development of peer-reviewed, health-based criteria which were used to support cleanup efforts at residences and other buildings where occupants were assumed to have long-term exposure to pollutants generated during the collapse of the World Trade Center towers.

For development of the cPAH loading criterion for settled dust, the WTC working group relied largely on EPA's *Policy Number 12 on Recommended Revisions to the Standard Operating Procedures (SOPs) for Residential Exposure Assessments* (2001) and EPA's *Risk Assessment Guidance for Superfund Volume 1: Human Health Evaluation Manual, Part E, Supplemental Guidance for Dermal Risk Assessment* (2004). The health-based criterion is based on the toxicity of cPAHs relative to benzo(a)pyrene and assumes exposure via both ingestion and dermal exposure pathways for an individual from age 1 through 31 years.

The WTC criterion for cPAHs was calculated using the following equation,

$$CancerRisk = LADD \times CSF$$

Where,

Cancer risk = target cancer risk of 1×10^{-4}

LADD = lifetime average daily dose (g/kg-day)

CSF = cancer slope factor for benzo(a)pyrene (7.3 kg-day/g)

The LADD was calculated as the sum of the potential dose rates (PDRs) for the dermal and ingestion exposure pathways averaged over 70 years. The health-based loading criterion was developed by adjusting the LADD iteratively until the target cancer risk level of 1×10^{-4} was reached.

For dermal contact with settled dust, the WTC criterion includes a number of factors to estimate the PDR, including a measure of the contaminant surface load (CSL) and fraction transferred from surface to skin (FTSS), transferrable residue on indoor surfaces (CSL * FTSS), a transfer coefficient (TC), exposure time (ET), and body weight (BW), as shown in the following equation:

$$PDR = \frac{\left(TC \times ET_{hard} \times FTSS_{hard} \times CSL_{hard}\right) + \left(TC \times ET_{soft} \times FTSS_{soft} \times CSL_{soft}\right)}{BW}$$

Where,

PDR = potential dose rate (g/kg-day)

TC = transfer coefficient (cm^2/hr)

ET = exposure time for hard and soft surfaces (hr/d)

FTSS = fraction transferred from hard or soft surfaces to skin (unitless fraction)

CSL = contaminant surface load on hard or soft surfaces (g/cm^2)

BW = body weight (kg)

The transfer coefficient (TC) represents the rate of skin contact with a surface and is based on several assumptions including a contaminant surface load (CSL) value of 50 g/cm^2 for dust loading on typical indoor residential surfaces (obtained from Rodes et al. 2001), as well as skin surface area of 5,000 cm^2 for children and 9,000 cm^2 for adults. Exposure time (ET) was assumed to be 8 hrs/day for carpets (i.e., soft surfaces) and 4 hrs/day for hard floors (i.e., hard surfaces) for children age 0 to 6 years and adults over the age of 18 years. For adolescents age 6 to 18 years, ET was assumed to be lower, 6 hrs/day for carpets and 2 hrs/day for hard surfaces, due to time spent away from home while at school.

The fraction of dust that can be transferred from hard or soft surfaces to skin (FTSS) is based on hand press experiments using lipophilic compounds conducted by Rodes et al. (2001). The values of 0.10 for soft surfaces and 0.50 for hard surfaces from Rodes et al. (2001) were modified to account for body parts that have less intensive contact with indoor surfaces than hands (e.g., arms, legs, face), resulting in FTSS values of 0.05 and 0.25 for soft and hard surfaces, respectively.

The body weight assumed for children (15 kg) and adults (71.8 kg) are based on a compilation of national data provided by EPA (1997).

Once the dermal PDR was calculated, the product then was multiplied by a factor of 0.13 to account for the dermal absorbed fraction of cPAHs.

The WTC criterion also takes into account ingestion of dust via hand-to-mouth contact. Several assumptions for the ingestion PDR are similar to those used to estimate the dermal PDR, as seen in the following equation:

$$PDR = \frac{\left[\left(ET_{hard} \times FTSS_{hard} \times CSL_{hard}\right) + \left(ET_{soft} \times FTSS_{soft} \times CSL_{soft}\right)\right] \times SA \times FQ \times SE}{BW}$$

Where the unique input parameters are,

SA = skin surface area (cm^2/event)

FQ = frequency of hand-to-mouth events (events/hr)

SE = saliva extraction factor (unitless fraction)

Skin surface area (SA) is based on the area of three fingers only, and was assumed to be 15 cm^2 for children and 45 cm^2 for adults. These values were extrapolated from data provided by EPA (1997). The frequency of hand-to-mouth events (FQ) was extrapolated from a study by Michaud et al. (1994) for four age groups as follows: 1 to 6 yrs, 9.5 events/hr; 7 to 12 yrs, 5 events/hr; 8 to 18 yrs, 2 events/hr; and 19 – 31 yrs, 1 event/hr. A default value of 0.50 from EPA's Office of Pesticide Protection (2001) was selected to represent the fraction of dust transferred from the skin to the mouth (SE).

The values for FTSS for the ingestion exposure pathway were obtained directly from Rodes et al. (2001), 0.10 and 0.50 for soft and hard surfaces, respectively. These values were measured during hand press experiments using lipophilic compounds and dry skin.

One unique aspect of the WTC working group health-based criterion is the assumption that the source of contaminants present as a result of the collapse of the WTC towers is not an infinite source. In other words, it was assumed that regular cleaning of the residences and other occupied buildings would diminish WTC-related contaminants over the assumed 30-year exposure time-frame. Following a review of a number of studies on dissipation of contaminants in indoor dust, it was assumed that the half-life of WTC-related contaminants would be 22 months (resulting in a decay rate constant of 0.38 per year). To account for this, the CSL variable was modified according to:

$$CSL = CSL_{initial} \times e^{-kt}$$

Where "k" is the dissipation rate constant of 0.38 per year and "t" is the time (years) over which the exposure is expected to occur. Assuming a finite source, the WTC criterion for cPAHs is 150 $\mu g/m^2$.

EPA was consulted on adjustment of the WTC criterion for cPAHs to eliminate the dissipation rate constant. When assuming an infinite source (i.e., coal-tar sealant is continuously maintained on parking lots throughout exposure duration), the criterion for cPAHs is adjusted downward to 34 $\mu g/m^2$ (Maddaloni, personal communication 2010).

Comparison of Dust Data to WTC Criterion

The WTC health-based criterion of 34 $\mu g/m^2$ (modified for an infinite source) is considered relevant to residential indoor dust evaluations in other areas because it was intended for residential settings, takes into account both dermal and ingestion exposure pathways, assumes a 30-year exposure time-frame spanning child, adolescent, and adult life stages, is based on standard EPA exposure and risk assessment methodology, utilizes the current recommended cancer slope factor for benzo(a)pyrene, and assumes exposure to all seven cPAHs.

Mahler et al. (2010) indicate that the mass of dust was weighed both before and after sieving but these data were not provided. With dust loading data, a residence-specific dust loading value for cPAHs could be derived according to:

$$\text{Residence} - \text{specific cPAH Loading Level}\left(\frac{\mu g}{m^2}\right) = Loading\left(\frac{g}{m^2}\right) \times Concentration\left(\frac{\mu g}{g}\right)$$

In the absence of the residence-specific dust loading data from Mahler et al. (2010), it can be assumed that the dust loading level in the sampled apartments is 0.5 g/m^2 (WTC 2003). This level of dust loading selected for development of WTC dust screening criteria is consistent with the geometric mean loading of 0.42 g/m^2 for bare floors reported by Adgate et al. (1995), 1.3 g/m^2 for carpets before cleaning and 0.1 g/m^2 for carpets after cleaning reported by Roberts et al. (1999), and range of 0.05 to 7 g/m^2 for bare floors reported by Lioy et al. (2002). A thorough discussion of dust loading levels is provided in the WTC criteria development document (2003).

The WTC criterion of 34 µg/m^2 is derived using the cancer slope factor for benzo(a)pyrene. As discussed previously, when comparing cPAH concentrations to health-based standards, cPAH concentrations must be adjusted by their relative potency factor. Concentrations of cPAHs as benzo(a)pyrene equivalents were used in the equation listed above to calculate residence-specific cPAH loading levels. The cPAH loading levels then can be compared to the WTC criterion of 34 µg/m^2.

cPAH dust loading levels (as benzo(a)pyrene equivalent concentrations) assuming dust loading level of 0.5 g/m^2.		
Parking Lot Status	**Sum of cPAH as BaPE (µg/g)**	**cPAH (as BaPE) Loading (µg/m^2)**
CT	4.5	2.3
CT	20	10
CT	15	7.3
CT	5.7	2.8
CT	20	9.9
CT	1.7	0.84
CT	2.3	1.2
CT	10	5.0
CT	6.1	3.0
CT	6.8	3.4
CT	32	16
NCT	0.21	0.10
NCT	1.9	0.93
NCT	5.0	2.5
NCT	0.75	0.38
NCT	2.0	1.0
NCT	2.8	1.4
NCT	17	8.4
NCT	0.085	0.042
NCT	0.36	0.18
NCT	0.31	0.16
NCT	0.63	0.32
NCT	0.34	0.17

As shown above, the maximum cPAH loading level of 16 µg/m^2 is less than half the WTC health-based criterion of 34 µg/m^2. The median cPAH indoor dust loading level for an apartment with a coal-tar sealed parking lot is 3.4 µg/m^2, which is an order of magnitude lower than the WTC criterion. Although indoor dust cPAH concentrations are greater in CT apartments, the

levels measured by Mahler et al. (2010) are well below health-based standards derived in accordance with WTC methodology.

It should be noted that exceedance of the WTC criterion would not suggest that adverse health effects would be experienced by the resident. Instead, exceedance would indicate that further study of the home may be necessary to better understand PAH sources in the home, exposure pathways, and perhaps biomonitoring to determine whether an exposure is occurring followed by abatement if further investigation indicates that a potential for risk to the resident is apparent.

Mahler et al. (2010) provide a comparison of indoor dust cPAH concentrations from CT sample locations to a concentration of 40 µg/g cPAHs (bulk concentration) provided by Maertens et al. (2008) that is equivalent to a 1×10^{-4} cancer risk level. Maertens et al. (2008) found that ingestion of 0.1 g/day of house dust by children ages 0 to 5 years results in less than 1×10^{-4} cancer risk for cPAH dust concentrations less than 40 µg/g. While seven indoor dust samples exceed this value (six CT locations, one NCT location), it is important to note that the exposure model described by Maertens et al. (2008) is not as sophisticated as that developed for the WTC criterion. For example, Maertens assumes a child will consume 0.1 g of dust per day without considering the dust loading level, the frequency of hand-to-mouth movements, the hand's skin surface area that transfers the dust to the mouth, the amount of dust transferred during the hand-to-mouth movements, or that the 0.1 g/day ingestion rate is based on a combined soil and house dust ingestion rate. In addition, Maertens et al. (2008) do not take into account exposures beyond childhood. Because the WTC criterion is based on a more robust evaluation of exposures from childhood through adulthood, the health-based WTC criterion of 34 $\mu g/m^2$ is more appropriate.

Dietary PAH Intakes

Ingestion of PAHs in food and inhalation of PAHs in tobacco smoke, wood smoke, and ambient air are the primary sources of PAH exposure for most people who are not exposed to PAHs in the workplace (ATSDR 1995). The highest levels of PAHs in food are found in foods that are grilled or smoked. On average, the Agency for Toxic Substances and Disease Registry (ATSDR; 1995) estimates that a total daily intake of PAHs includes 0.16 to 1.6 µg from food, 0.207 µg from air, and 0.027 µg from water. The World Health Organization (WHO; 1998) provides a daily intake estimate from food of 0.1 to 8 µg. The WHO (1998) notes that while PAHs may be found on fruits and vegetables due to atmospheric deposition and/or due to food processing such as frying and roasting, the highest levels of PAHs have been found in smoked meat (over 100 µg/kg) and fish (up to 86 µg/kg).

Assuming exposure to cPAHs in dust at the highest detected concentration for a CT location (16 $\mu g/m^2$) reported by Mahler et al. (2010), the total daily intake of cPAHs would be 0.28 µg. This intake is based on exposure parameters identical to those used to derive the WTC screening criterion of 34 $\mu g/m^2$. This intake for cPAHs not only is shown to be below an acceptable risk management level through comparison with the WTC criterion, but also is consistent with other background exposures via food and air.

Conclusions

ENVIRON performed a technical review of the study, "Coal-Tar-Based Parking Lot Sealcoat: An Unrecognized Source of PAH to Settled House Dust" by Mahler et al. published in *Environmental Science & Technology* (2010). The review was limited to information published in the study itself and Supplementary Information provided by the publisher.

ENVIRON notes the following points regarding the study by Mahler et al. (2010):

- Short-comings in the study design introduced uncertainty in data quality and in data evaluation, including uncertain identification of coal-tar sealed and non-coal-tar sealed parking lots; absence of characterization of other PAH sources; absence of consideration of ages of apartment complex, parking lot and sealant, and carpeting; collection of composite samples that may not accurately represent exposure potential; and potential for cross-contamination between samples.

- Both concentration and dust loading are important factors in evaluating exposure to chemicals in dust. Mahler et al. (2010) did not evaluate dust loading, which is critical in understanding *how much* dust is available for contact by residents.

- Mahler et al. (2010) did not compare PAH results to a health-based standard to determine the potential risk associated with the levels measured in house dust. Use of the screening level developed for cleanup of residences near the World Trade Center in New York City indicates that cancer-causing PAHs in dust measured by Mahler et al. (2010) are below levels of concern. In fact, the highest level measured by Mahler et al. (2010) in indoor dust is less than half of the World Trade Center screening level, even though PAH concentrations in dust may be overestimated due to the selected sampling method.

- Intake of cancer-causing PAHs in dust occurs every day through the air we breathe and food we eat. The levels measured by Mahler et al. (2010) that could be taken in via house dust are consistent with background intake levels via food, air, and water.

References

Adgate J.L., C. Weisel, Y. Wang, G.G. Rhoads, and P.J. Lioy. 1995. Lead in house dust: Relationships between exposure metrics. Environmental Research 70:134-147.

ASTM (American Society of Testing and Materials). 2005. Method D 5438-05. Practice for Collection of Floor Dust for Chemical Analysis. Philadelphia, PA.

ATSDR (Agency for Toxic Substances and Disease Registry). 1995. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. U.S. Department of Health and Human Services, Public Health Service, Atlanta.

ATSDR. 2006. Health consultation, Follow-up exposure investigation, Calcasieu Estuary (a/k/a Mossville), Lake Charles, Calcasieu Parish, Louisiana. EPA Facility ID LA0002368173.

CS_3 Inc. 2004. High volume small surface sampler HVS3 operation manual.

Garabrant D.H., A. Franzblau, J. Lepkowski, B.W. Gillespie, P. Adriaens, A. Demond, E. Hedgeman, K. Knutson, L. Zwica, K. Olson, T. Towey, Q. Chen, B. Hong, C. Chang, S. Lee, B. Ward, K. LaDronka, W. Luksemburg, M. Maier. 2009. The University of Michigan Dioxin Exposure Study: Predictors of human serum dioxin concentrations in Midland and Saginaw, Michigan. Environmental Health Perspectives 117:818-824.

Heudorf U. and J. Angerer. 2001. Internal exposure to PAHs of children and adults living in homes with parquet flooring containing high levels of PAHs in the parquet glue. Int Arch Occup Environ Health 74:91-101.

Lewis R.G., C.R. Fortune, R.D. Willis, D.E. Camann, and J.T. Antley. 1999. Distribution of pesticides and Polycyclic aromatic hydrocarbons in house dust as a function of particle size. Environmental Health Perspectives 107(9):721-726.

Lioy P.J., N.C.G. Freeman, and J.R. Millette. 2002. Dust: A metric for use in residential and building exposure assessment and source characterization. Environmental Health Perspectives 110(10):969-983.

Maddaloni, Mark. 2010. Personal communication between Dr. Rosalind Schoof, ENVIRON International Corporation and Dr. Mark Maddaloni, U.S. Environmental Protection Agency, Region II Toxicologist, Regional Risk Coordinator, via electronic mail correspondence on April 5, 2010.

Maertens R.M., X. Yang, J. Zhu, R.W. Gagne, G.R. Douglas, and P.A. White. 2008. Mutagenic and carcinogenic hazards of settled house dust I: polycyclic aromatic hydrocarbon content and excess lifetime cancer risk from preschool exposure. Environmental Science and Technology 42:1747-1753.

Mahler B. J., P.C. Van Metre, J.T. Wilson, and M. Musgrove. 2010. Coal-Tar-Based Parking Lot Sealcoat: An Unrecognized Source of PAH to Settled House Dust. Environmental Science and Technology 44(3):894-900.

Michaud J.M., S.L. Huntley, R.A. Sherer, M.N. Gray, and D.J. Paustenbach. 1994. PCB and dioxin re-entry criteria for building surfaces and air. Journal of Exposure Analysis and Environmental Epidemiology 4(2):197-227.

Rodes C., R. Newsome, R. Vanderpool, J. Antley, R. Lewis. 2001. Experimental methodologies and preliminary transfer factor data for estimation of dermal exposure to particles. Journal of Exposure Analysis and Environmental Epidemiology 11:123-139.

Stout S.A., A.D. Uhler, S.D. Emsbo-Mattingly. 2004. Comparative evaluation of background anthropogenic hydrocarbons in surficial sediments from nine urban waterways. Environmental Science and Technology 38(11):2987-2994.

U.S. Environmental Protection Agency (EPA). 1991. Role of baseline risk assessment in Superfund remedy selection decisions. Office of Solid Waste and Emergency Response Directive 9355.0-30.

U.S. EPA. 1993. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. Office of Research and Development, Washington DC. EPA/600/R-93/089.

U.S. EPA. 1997. Exposure Factors Handbook. National Center for Environmental Assessment, Office of Research and Development, Washington DC. EPA/600/P-95/002Fa.

U.S. EPA. 2001. Science Advisory Council for Exposure, Policy Number 12 on Recommended Revisions to the Standard Operating Procedures (SOPs) for Residential Exposure Assessments.

U.S. EPA. 2004. Risk Assessment Guidance for Superfund, Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Office of Superfund Remediation and Technology Innovation, Washington DC. EPA/540/R/99/05.

U.S. EPA. 2008. Guidance For the Sampling and Analysis of Lead in Indoor Residential Dust for Use in the Integrated Exposure Uptake Biokinetic (IEUBK) Model. Prepared by the Lead Committee of the Technical Review Workgroup for Metals and Asbestos, Office of Superfund Remediation and Technology Innovation. OSWER 9285.7-80.

U.S. EPA. 2010. Integrated Risk Information System (IRIS). Accessed online at: http://www.epa.gov/ncea/iris/

WHO (World Health Organization). 1998. Selected non-hetrocyclic polycyclic aromatic hydrocarbons. Environmental Health Criteria 202. United Nations Environment Programme, International Labour Organisation, World Health Organization, International Programme on Chemical Safety.

WTC (World Trade Center Indoor Air Task Force Work Group). 2003. World Trade Center Indoor Environment Assessment: Selecting Contaminants of Potential Concern and Setting Health-based Benchmarks. Prepared by the Contaminants of Potential Concern Committee.

WTC. 2005. World Trade Center Indoor Dust Test and Clean Program Plan. Final.

Dust Study Questions

1. Describe the approach used to collect samples at each property, including specifying the order in which samples were collected. The sampling period in the Mahler et al. 2010 study is described as between April and July 2008. The weather in Austin, Texas varied considerably during that period. From the beginning of 2008 through mid-April, there was little rain in Austin which would be expected to result in considerable dust build up. From late April through July there were periods of relatively frequent rain. For this reason as we well as others, it's important to understand which samples were collected at which times. Which was sampled first, outdoors or indoors? Was the order the same at every property? Do you have a sampling SOP (standard operating procedure) that you could share with us?

Indoor samples were collected from Mar 28 to May 23. Outdoor samples were collected from Mar 28 to July 23. In all cases the indoor sample was collected prior to the outdoor sample. Seasonal build-up of dust is not relevant to PAH concentrations.

2. Describe the approach used to collect the indoor dust samples. The paper says that dust was collected from the entryways and adjacent living room floors, and that the areas sampled ranged from 1.6 to 13 m^2. Why wasn't a standard area vacuumed? What was the relative area of the entry vs. living area that was vacuumed? Was there any difference between the concentrations of PAHs at the entryway as opposed to the living room?
[USEPA guidance for sampling lead in indoor dust specifies that discrete samples should be collected from a bedroom, most frequently used living space and the most frequently used entrance. It is further recommended that these samples be used to calculate an average concentration based on time-weighted activity patterns for residents.]

A sufficient area was vacuumed to obtain a sufficient amount of dust for analysis. Indoor dust was analyzed as a single composite. This is obfuscating the fact that indoor dust had elevated concentrations of PAHs if the parking lot had a sealcoated parking lot.

3. Were PAH loads calculated? The mass of dust collected indoors was stated to vary from 0.36 to 80 g (median of 28 g). Did you evaluate the influence of dust load on PAH concentrations?
[Both PAH concentration and PAH load from each living area are needed to assess exposures because exposure will be a function of transfer of PAH to hands, which will be a function of both concentration and load. Dust load is expected to be highest at the entryway and in carpets; however, PAH concentrations are expected to be highest at the entryway and lowest in carpets. Since most time will be spent in the living areas rather than at the entryway, composite samples that combine both areas do not represent average exposure concentrations. The composite concentrations will overestimate the average exposure concentration both due to higher concentration and due to higher loading.]

EXHIBIT
D

Yes, they were. PAH loads from apartments with CT lots were 16 times higher than those from apartments with NCT lots (based on median values). Loads for CT apartments ranged from 9 to 480 ug/m^2 with a mean value of 160 ug/m^2.

This publication was not a health risk analysis. The paper demonstrated that PAH concentrations are 25 times in residences with sealcoated parking lots. We did compute PAH loads, but it's not relevant to this discussion.

4. Describe the process of removing samples from the HVS3 and preparing it for collection of the next sample. Describe if/how the HVS3 was cleaned, and the frequency with which the cleaning was performed. Was this done between every sample? [On average, parking lot dust concentrations are 37 times higher than indoor dust concentrations. Consequently, if parking lots were sampled first, and if the HSV3 was not decontaminated according to recommended procedures, residual parking lot dust could account for the elevated indoor dust concentrations. Even if the indoor dust was sampled first, failure to decontaminate between properties could result in high values due to residual contamination from the prior property.]

The HVS3 was cleaned between collection of every sample.

5. Did you evaluate the possible influence of apartment or carpet age on dust PAH concentrations?

Yes. There is no relation.

6. Will you share with us the individual data for the independent variables listed in Table 1 of the paper? How can the alleged impact of these variables be checked if the data for each apartment are not produced? Were data collected for any other parameters not listed in Table 1? When will the USGS produce this data?

Obfuscation.

7. The degree of sealcoat wear is listed in Table 1 as an independent variable potentially related to the levels of PAH detected in SHD and parking lots dust samples. However, there is no information presented as to how this wear was evaluated, nor is there any information in the supporting material that summarizes the range of wear levels for the parking lots examined in the study. If parking lot surface type is believed to be a significant factor in explaining indoor and parking lot dust PAH levels, why wasn't the degree of sealcoat wear estimated? Were such estimates made in earlier USGS "parking lot" articles? If so, why were those USGS techniques for estimating wear abandoned this time around? Is the USGS saying that the "untreated asphalt" parking lots in their study had never been treated with any type of sealant? Did the USGS assume that a parking lot which had no obvious "sealant chips" for the "coffee/tea" screening test was a parking lot that had never been sealed in the past? If so, did the USGS conduct any research to demonstrate that such an assumption was valid?

Obfuscation. The enormous difference between NCT lots and CT lots indicates that wear was not an issue, and unsealcoated lots were not misidentified.

8. Describe the relevance of the German dust PAH guideline of 10 µg/g.
[U.S. EPA (2003) derived a dust PAH guideline of 145 µg/m^2 for total PAH for the World Trade Center residential studies. This value is based on an incremental risk of 1 x 10^{-4} for ingestion of and dermal contact with dust for 30 years (daily dust intake of 13 mg/d for children and 6 mg/d for adults). The target risk is justified due to limits of analytical methods for PAH in dust and to background concentrations of other COPCs such as dioxins. We can estimate an approximately equivalent T-PAH concentration based on literature reports of typical dust loads in homes. For example, Adgate (1995) report a geometric mean dust load of 38 mg/ft^2 for 216 homes, which is 0.409 g/m^2. At this loading, the U.S. EPA guideline of 145 µg/m^2 is equivalent to a concentration of 355 µg/g. This compares to a range of coal tar sealcoat indoor dust T-PAH concentrations of 20 to 335 µg/g in the Mahler et al. study. Consequently, the reported indoor dust concentrations do not exceed the health-based benchmarks used in the World Trade Center Indoor Environmental Assessment.]

So, they are accusing us of doing a health-risk analysis when we did not do so, and then they go ahead and do a health-risk analysis on nonexistent data.

The WTC daily dust ingestion value of 13 mg/d for toddlers is very small relative to that presented in other EPA documents: 1275 mg/d from one study (Calabrese), 55 mg/d from EPA's own summary of estimates, and that's for the median child. Upper percentile (top 10%) estimate is 432 mg/d. EPA Child-Specific Exposure Factors Handbook. "EPA (1997) recommends central estimates of total soil ingestion rates of 100 mg/d for children and 50 mg/d for adults. It is logical that lower ingestion rates would apply to dust only, however, it is uncertain how much less. This uncertainty appears to have more potential for leading to under than over estimates of risk." So they admit that they are probably underestimating the risk.

Further, the total exposure is based on the assumption that the residue level will dissipate according to first-order kinetics, i.e., that in the case of the WTC the contaminant was delivered only once and from then on it continually decreased as a result of dissipation from cleaning, chemical breakdown, etc. This is clearly not the case with CT sealcoat dust, which is constantly replenished. (Appendix D5, Section 3.3, World Trade Center Indoor Environmental Assessment.)

"Dose rates were estimated based on a number of assumptions—for example, the fraction of dust residues that can be transferred to the skin, daily skin loads, mouthing behaviors for different age groups, and dissipation of surface loading over time."

First of all the content of these papers is not germane as they have not been published. The two included are still in review; to the best of my knowledge, the Demott paper was rejected by ES&T and is now in review at Environmental Forensics; they have been

citing it as "in review" for 18 months now. The one that is listed as "in press" is not included in the .pdf, and is in a trade journal, not a peer-reviewed scientific journal.

Because of this, let's stick to just one stopper for each paper.

Demott – Analytical error

O'Reilly – misleading use of data

There are substantial problems with the two papers submitted.

The Demott paper has so much analytical error that the data cannot be reasonably evaluated. They only collected two sets of duplicates for all samples, and the relative percent difference was 42% and 87%. Now, the amount of decrease in PAHs that we might expect to see of the course of 2 years (assuming a half-life of 15 years) is about 9%. How can we possibly see a decrease of 9% if the experimental error is 40% or greater? And in fact, these weren't even field duplicates, they were splits. They collected a bunch of sediment, mixed it in a bowl, divided it in half, and analyzed each half. There are a lot of other very serious issues, such as the fact that collection of streambed sediment is a very poor choice of approach for determining trends, but this one overwhelms them.

The O'Reilly paper has four major flaws. First, they don't specify which data they used for their "forensic" plots, but the data for "coal tar sealed lot" shown on their "forensic" plots don't include any of the data from any of our publications. If you plot our parking lot data on there, it very nicely overlies the stormwater pond data. Second, these are pretty weak "forensic" methods anyway – they only used three methods, as methods 1 and 2 and methods 4 and 5 are really the same thing. They're fine for screening, but cannot be considered advanced forensic tools. The science has moved on, and the CMB model approach we've taken is much more sophisticated and preferable to any of these approaches. Third, they exclude any consideration of PAH concentrations. How can atmospheric deposition be the principal source of PAHs to stormwater ponds in the Minneapolis area if there is a thousand-fold difference in the PAH concentrations of those ponds? And finally, none of the sources that they consider, with the exception of dust from lots with coal-tar-based sealcoat (and apparently they don't even use that) are actually sources – atmospheric particles, urban soils, and rooftop particles are all themselves receivers of PAHs from different sources, and themselves may (and likely do) include PAHs from coal-tar-based sealcoat.

And note, the atmospheric particle standards used by O'Reilly are either from Simcik, 1999, which were for atmospheric particles in the Chicago area (2 of the 3 points); the other is the NIST atmospheric dust standard, for which O'Reilly does not provide a reference – which NIST standard is this? If it's 8785 (Air particulate matter on filter media), it consists of the fine fraction of the "urban dust" standard SRM 1649a. Urban dust is simply "an atmospheric particulate material collected in an urban area." We have no idea what urban area.

Crane, Judy (MPCA)

From: Barbara J Mahler [bjmahler@usgs.gov]
Sent: Wednesday, February 24, 2010 2:26 PM
To: Crane, Judy (MPCA)
Cc: Alison.Watts@unh.edu
Subject: RE: meeting w/ Anne LeHuray in Minnesota on March 12

Judy,

Just had a long talk with Dan Chiles, mayor pro-tem of Springfield. Apparently LeHuray and DeMott were in Springfield this afternoon (their third visit), and in his words, they "took apart" our research. He's sending me a transcript and they're going to post the video on their website. But from talking to him, it sounds like what they did was put up a smokescreen, bringing up a bunch of non-relevant points and mixing it in with a few outright lies.

I think it will be important for us to take a look at this and come up with a point-by-point refutation of what they're saying, as well as some powerful points of our own. It sounds like they're not pulling any punches.

Barbara

****

Barbara J. Mahler, Ph.D., P.G.
Research Hydrologist
U.S. Geological Survey
1505 Ferguson Ln., Austin, TX 78754
(512) 927-3566

From: "Crane, Judy (MPCA)" <Judy.Crane@state.mn.us>
To: "Mike Kromrey (mike@watershedcommittee.org)" <mike@watershedcommittee.org>, "Peter Van Metre (pcvanmet@usgs.gov)" <pcvanmet@usgs.gov>, "Barbara Mahler (bjmahler@usgs.gov)" <bjmahler@usgs.gov>, "Tom Ennis (Tom.Ennis@ci.austin.tx.us)" <Tom.Ennis@ci.austin.tx.us>
Cc: "Alison Watts (alison.watts@unh.edu)" <alison.watts@unh.edu>
Date: 02/24/2010 02:11 PM
Subject: RE: meeting w/ Anne LeHuray in Minnesota on March 12

Hi—

As a follow-up to the below email, Don Berger of our staff provided me with some additional info about our upcoming meeting with White Bear Lake city officials and Anne LeHurray & PCTC members. Don has been doing some work on the coal tar sealcoat issue from the policy side of things. He's trying to get the city to put together an agenda for this meeting. Of particular note are the sentences I have highlighted below (for which publication of these results in the peer-reviewed literature will be necessary before we give it much attention).

Purpose:
I believe there are two purposes and I have asked the PCTC to put theirs in writing. I expect the PCTC's purpose to include convincing the City of White Bear Lake that they should not take action to ban coal tar sealcoat within their jurisdiction. I believe our purpose to be much different. I believe our purpose is to support the City in moving toward a coal tar sealer restriction in their jurisdiction, gather as much information from the industry and the legislative lobbyists as possible, and represent the health and welfare of the environment, our stakeholders, and the public well being as best we can. I want you to know that it is not my intention to debate scientific studies, research, or sampling results with the PCTC. My discussions with the City of White Bear Lake

Crane, Judy (MPCA)

From: Peter C VanMetre [pcvanmet@usgs.gov]
Sent: Thursday, December 09, 2010 12:59 PM
To: Crane, Judy (MPCA)
Cc: Barbara J Mahler
Subject: Re: FW: [npsinfo] Pavement Sealer Study-Product Ban Fails to Lower or Change Sources of PAHs in Watershed

We've seen much of what's in this paper in various presentations by Bob DeMott. The approaches used are not technically defensible.
Pete

Peter Van Metre
Research Hydrologist
USGS
1505 Ferguson Lane
Austin, TX 78754
512-927-3506

From: "Crane, Judy (MPCA)" <Judy.Crane@state.mn.us>
To: "Tom Ennis (Tom.Ennis@ci.austin.tx.us)" <Tom.Ennis@ci.austin.tx.us>, "Peter Van Metre (pcvanmet@usgs.gov)" <pcvanmet@usgs.gov>, "Barbara Mahler (blmahler@usgs.gov)" <blmahler@usgs.gov>
Date: 12/09/2010 12:50 PM
Subject: FW: [npsinfo] Pavement Sealer Study-Product Ban Fails to Lower or Change Sources of PAHs in Watershed

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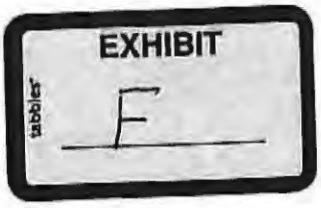
I'm sure you've already seen the below article....

Judy

From: Gelbmann, Anne (MPCA)
Sent: Thursday, December 09, 2010 10:45 AM
To: Berger, Donald (MPCA)
Cc: Crane, Judy (MPCA); Thompson, Dale (MPCA)
Subject: FW: [npsinfo] Pavement Sealer Study-Product Ban Fails to Lower or Change Sources of PAHs in Watershed

Don/Judy/Dale-are you on this list serve? Lots of e-mails today about the PAH ban.



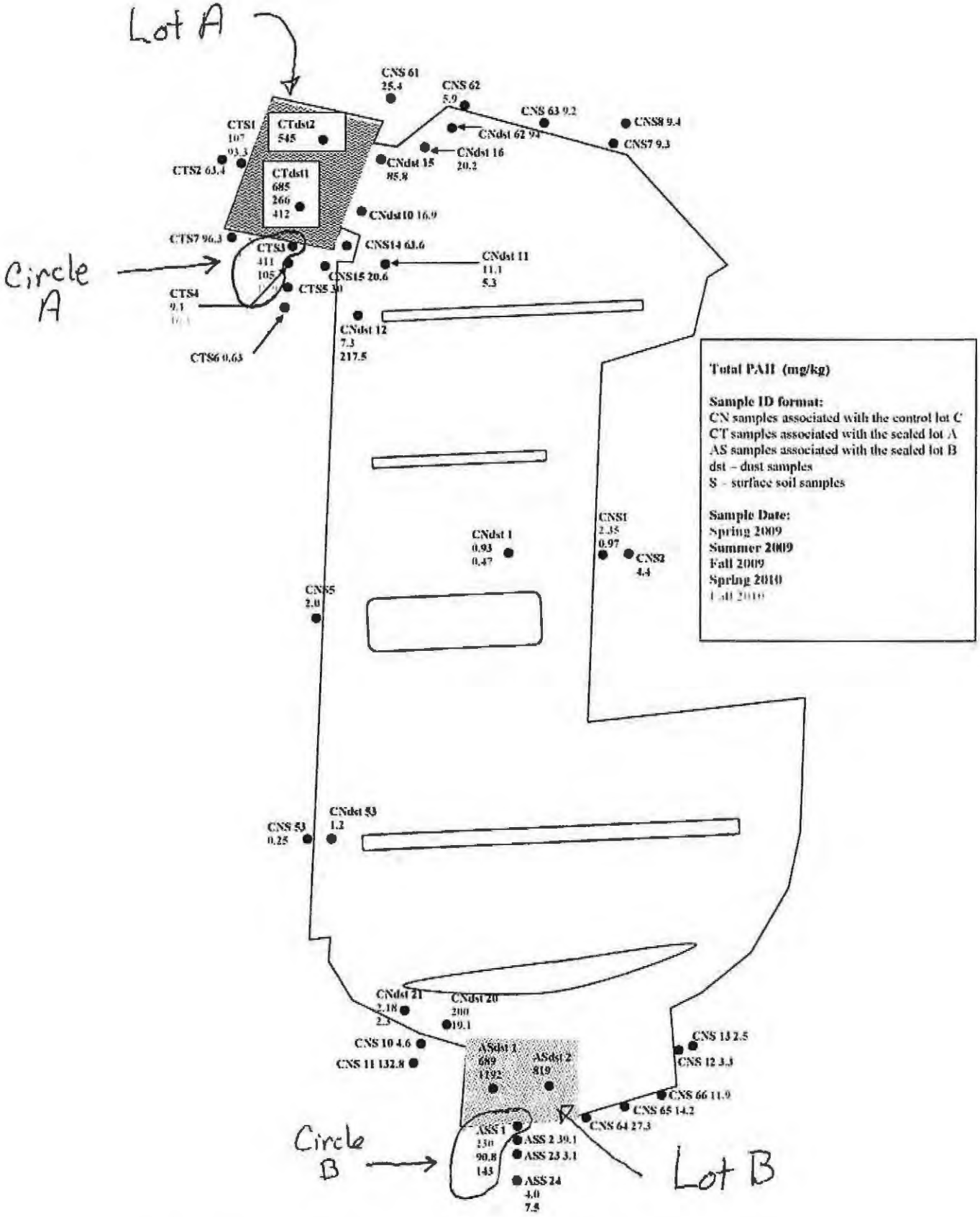


Figure 2. Surface soil and dust sampling locations and concentrations (total PAH mg/kg).

EXHIBIT
G

My thought is that there is absolutely no reason to even consider withdrawing the paper! The high concentrations confirm that it's coal tar sealant and regardless of what the sealant is made from, the conclusion that high PAHs in a sealant product lead to high PAHs in runoff (and dust, air, etc.) are still completely valid. The main thing your experience says to me is that applicators are sometimes not clear on
the contents of the products they use.

In fact, if I were writing regulations on products, I would base them on maximum PAH concentrations and not just named contents, especially considering the range of names used for "refined tar" and the frequent uncertainty in contents.

(welcome to the club)

Peter Van Metre
Research Hydrologist
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1505 Ferguson Lane
Austin, TX 78754
512-927-3506

From: "Watts, Alison" <Alison.Watts@unh.edu>
To: "'Tom.Ennis@ci.austin.tx.us'" <Tom.Ennis@ci.austin.tx.us>
Cc: "'Peter C VanMetre'" <pcvanmet@usgs.gov>
Date: 03/04/2011 02:42 PM
Subject: As vs CT

Tom - Do you have any documented cases where sealant applicators genuinely intended to apply asphalt sealant, but put down CT instead? The PCTC has suggested that we would like to withdraw our ES&T paper based on the fact that the applicator's records show that they put down asphalt sealer (as we requested), but based on analyses we thought that they applied CT. My take is that a) the applicator probably made a genuine mistake, and b) it really doesn't change our conclusions which were that high PAHs in sealant lead to high PAHs in runoff.

Thoughts?

Alison W. Watts, Ph.D.
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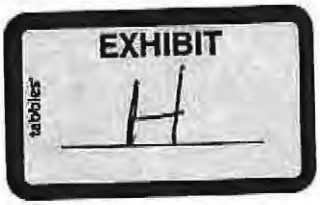
alison.watts@unh.edu



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$Abstract: Alison:
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Alison:

I agree with Pete.

Interestingly we have seen confusion among the applicators about what they are actually using. Early in our ban we had a citizen complaint against an applicator of a fast food restaurant. The applicator confessed to using coal tar, but the labs showed that he was actually using AE.

In another instance we had a suspicious lot based upon the field test. The applicator said he didn't know what he had. Whether that is truthful
or not remains to be seen, but the labs showed that it was mostly likely a weak blend of asphalt sealant with a small amount of coal tar.

We also have had oddities with the suppliers of commercial products. We had one that tested as asphalt sealant, but was labeled as coal tar.

All this to say that attention to product type and quality (and dilution!) has been lax in this industry. Because of this some manufacturer's have wanted to only allow "certified applicators" of their
product, but this remains a wish as far as I know.

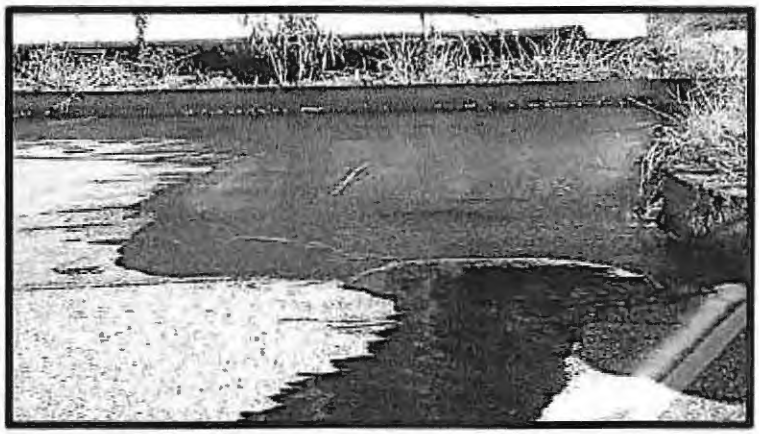
Hope this helps....

Tom

From: Peter C VanMetre [mailto:pcvanmet@usgs.gov]
Sent: Friday, March 04, 2011 3:00 PM
To: Watts, Alison
Cc: Ennis, Tom; Barbara J Mahler
Subject: Re: As vs CT

UNIVERSITY OF NEW HAMPSHIRE
STORMWATER CENTER

Final Report
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



Prepared by
The University of New Hampshire Stormwater Center

Prepared under Support from U.S. Environmental Protection Agency GLNPO-4-17

December 2010

Lot A

Soil and Sediment Analytic Data (mg/kg)
Meta Environmental Laboratory

Sample ID	CT- S1a	CT- S1a	CT- S3a	CTS3Cd	CTS4C
Date Sampled:	05/08/09	05/08/09	05/08/09	11/7/2009	11/7/2009
mg/kg dry wt					
Naphthalene	0.055	0.044	0.207	0.036	0.008
2-Methylnaphthalene	0.052	0.038	0.192	0.025	0.008
1-Methylnaphthalene	0.047	0.036	0.174	0.022	0.007
C1 - Naphthalene	0.063	0.048	0.235	0.027	0.009
C2 - Naphthalene	0.201	0.147	0.695	0.074	0.018
C3- Naphthalene	0.345	0.266	1.2	0.150	0.027
C4- Naphthalene	0.263	0.212	0.925	0.120	0.018
Biphenyl	0.037	0.028	0.155		
Acenaphthylene	0.256	0.217	0.932	0.257	0.106
Acenaphthene	0.763	0.517	2.63	0.382	0.052
Dibenzofuran	0.458	0.288	1.6	0.239	0.026
Fluorene	1.15	0.731	3.81	0.625	0.068
C1 - Fluorene	0.385	0.281	1.35	0.245	0.034
C2 - Fluorene	0.391	0.321	1.51	0.298	0.041
C3 - Fluorene	0.263	0.195	0.947	0.174	0.021
Phenanthrene	13.6	11.6	48.2	8.87	0.982
Anthracene	3.25	2.6	11.6	2.09	0.226
C1 - Phenanthrene/Anthracene	3.68	2.83	12.1	2.51	0.279
C2 - Phenanthrene/Anthracene	1.82	1.48	6.29	1.24	0.142
C3 - Phenanthrene/Anthracene	0.67	0.539	2.52	0.503	0.058
C4 - Phenanthrene/Anthracene	0.187	0.16	0.79	0.183	0.024
Dibenzothiophene	0.811	0.594	2.73	0.521	0.050
C1 - Dibenzothiophene	0.3	0.239	0.971	0.160	0.026
C2 - Dibenzothiophene	0.236	0.195	0.75	0.152	0.033
C3 - Dibenzothiophene	0.15	0.129	0.485	0.109	0.022
C4 - Dibenzothiophene	0.076	0.068	0.237	0.062	0.013
Benzo(b)naphtho(2,1-d)thiophene	1.58	1.28	5.7	1.51	0.129
Fluoranthene	22.2	21.4	82.1	24.1	1.78
Pyrene	17.1	16.3	64.1	19.0	1.41
C1 - Fluoranthene/Pyrene	6.28	5.17	22.1	5.26	0.492
C2 - Fluoranthene/Pyrene	2.54	2.09	9.19	2.47	0.271
C3 - Fluoranthene/Pyrene	0.755	0.648	3.02	0.803	0.098
Benz[a]anthracene	7.02	5.5	28.6	6.56	0.591
Chrysene*	8.04	6.6	32.9	7.99	0.797
C1 - Benz(a)anthracene/Chrysene	2.16	1.74	7.98	2.03	0.206
C2 - Benz(a)anthracene/Chrysene	0.758	0.652	2.89	0.853	0.089
C3 - Benz(a)anthracene/Chrysene	0.315	0.235	1.03	0.336	0.035
C4 - Benz(a)anthracene/Chrysene	0.23	0.196	0.875	0.440	
Benzo[b]fluoranthene	7.66	6.5	32.6	8.15	0.699
Benzo[j/k]fluoranthene	6.38	5.24	27.2	6.73	0.625
Benzo(e)pyrene	5.31	4.45	22.6	5.97	0.520
Benzo[a]pyrene	7.29	5.97	29.2	7.49	0.666
Perylene	2.23	1.79	8.21	2.23	0.175
Indeno[1,2,3-cd]pyrene	4.99	4.22	20.8	5.48	0.467
Dibenz[a,h]anthracene	1.73	1.45	6.68	1.36	0.111
Benzo[g,h,i]perylene	5.26	4.43	19.4	5.72	0.496
Coronene	1.62	1.33	6.18	1.84	0.150
Total PAH (16)	107	93.3	411	105	9.08
Total PAH (42)	138	118	524	132	11.8

Lot B

Soil and Sediment Analytic Data (mg/kg)
Meta Environmental Laboratory

Sample ID	ASS1a	ASS1B	ASS23B	ASS24B
Date Sampled:	05/08/09	8/17/2009	8/17/2009	8/17/2009
mg/kg dry wt				
Naphthalene	0.066			
2-Methylnaphthalene	0.02	0.026	0.002	0.002
1-Methylnaphthalene	0.013			
C1 - Naphthalene	0.023			
C2 - Naphthalene	0.046			
C3- Naphthalene	0.045			
C4- Naphthalene	0.023			
Biphenyl	0.021			
Acenaphthylene	0.322	0.073	0.007	0.007
Acenaphthene	0.26	0.056	0.002	0.003
Dibenzofuran	0.241	0.073	0.003	0.004
Fluorene	0.541	0.131	0.005	0.007
C1 - Fluorene	0.147			
C2 - Fluorene	0.161			
C3 - Fluorene	0.135			
Phenanthrene	12.6	4.46	0.156	0.224
Anthracene	2.88	0.647	0.023	0.027
C1 - Phenanthrene/Anthracene	2.91			
C2 - Phenanthrene/Anthracene	1.08			
C3 - Phenanthrene/Anthracene	0.41			
C4 - Phenanthrene/Anthracene	0.129			
Dibenzothiophene	0.801			
C1 - Dibenzothiophene	0.302			
C2 - Dibenzothiophene	0.179			
C3 - Dibenzothiophene	0.116			
C4 - Dibenzothiophene	0.074			
Benzo(b)naphtho(2,1-d)thiophene	3.54			
Fluoranthene	46.5	15.8	0.567	0.752
Pyrene	35	12.1	0.426	0.556
C1 - Fluoranthene/Pyrene	10.6			
C2 - Fluoranthene/Pyrene	4.88			
C3 - Fluoranthene/Pyrene	1.26			
Benz[a]anthracene	16.7	6.03	0.193	0.241
Chrysene*	23.5	9.38	0.326	0.407
C1 - Benz(a)anthracene/Chrysene	3.84			
C2 - Benz(a)anthracene/Chrysene	1.03			
C3 - Benz(a)anthracene/Chrysene				
C4 - Benz(a)anthracene/Chrysene				
Benzo[b]fluoranthene	23.5	9.38	0.333	0.407
Benzo[j/k]fluoranthene	20.1	8.14	0.260	0.318
Benzo(e)pyrene	12.4	6.76	0.230	0.285
Benzo[a]pyrene	19.2	8.57	0.279	0.341
Perylene	5.24	2.28	0.071	0.091
Indeno[1,2,3-cd]pyrene	12.2	7.36	0.245	0.305
Dibenz[a,h]anthracene	4.44	2.0	0.067	0.084
Benzo[g,h,i]perylene	12.7	6.68	0.222	0.276
Coronene	3.92	1.89	0.063	0.078
Total PAH (16)	**230**	**90.8**	**3.11**	**3.96**
Total PAH (42)	276	99.9	3.42	4.34