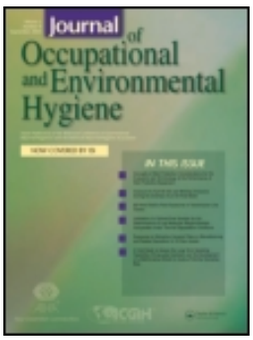

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Gaps in Scientific Knowledge About the Carcinogenic Potential of Asphalt/Bitumen Fumes

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Despite a relatively large body of published research, the potential carcinogenicity of asphalt/bitumen fumes is still a vexing question. Various uncertainties and gaps in scientific knowledge need to be addressed. These include uncertainties in chemistry, animal studies, and human studies. The chemistry of asphalt/bitumen fumes is complex and varies according to the source of the crude oil and the application parameters. The epidemiological studies, while showing weak evidence of lung cancer, are inconsistent and many confounding factors have not been addressed. Studies of animal exposure are also inconsistent regarding laboratory and field-generated fumes. There is a need for further human studies that address potential confounding factors such as smoking, diet, coal tar, and diesel exposures. Animal inhalation studies need to be conducted with asphalt/bitumen fumes that are chemically representative of roofing and paving fumes. Underlying all of this is the need for continued characterization of fumes so their use in animal and field studies can be properly assessed. Nonetheless, uncertainties such as these should not preclude appropriate public health actions to protect workers in the even that asphalt fumes are found to be a carcinogenic hazard.

Keywords asphalt, bitumen, fume, inhalation, cancer

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INTRODUCTION

It has been very difficult to determine the human carcinogenic potential of asphalt/bitumen fumes and vapor. This is because of the complex and variable nature of the chemical composition of the fumes, the heterogeneity of the workplaces, the high turnover rates of the workforce, and the fact that cancers of most concern are associated with various other risk factors. Consequently, despite the existence of a body of scientific evidence and review reports, there are still major unanswered scientific questions that leave gaps in any assessment of carcinogenic potential.[1–3]

Uncertainty, however, is not a contraindication for taking public health action. In fact, in the absence of a clear picture of a potential hazard, it is prudent to take preventive action such as was taken in the United States to reduce exposure to asphalt fumes by design criteria for new highway class paving machines, and for the development of new types of low fuming asphalt roofing compounds.[4,5] Nonetheless, these are interim measures that will not resolve the questions about hazard potential. Such questions need to be examined and addressed and that is the purpose of this meeting.

Uncertainties in the Evidence

The best evidence for determining whether a substance is a human carcinogen is in well conducted epidemiological studies. The criteria for experimental design quality include, among other factors, adequate sample size and follow-up latency, sufficient exposure information, minimal bias and measurement error, and control of confounding factors.

For asphalt, the largest and best conducted study involved 29820 workers from eight different countries, engaged in road paving, roofing, asphalt mixing, waterproofing, or other specified jobs where exposure to asphalt fumes was possible.[6,7] The study was retrospective in design and involved 481,089 person-years of observation for asphalt workers. For comparison purposes, the mortality rates from similar populations in the ground and building construction industry were used. Although the overall mortality for the entire exposed cohort was below 1.0 (SMR = 0.96 [95% C.I. = 0.93–0.99)), the study showed that mortality from lung cancer was increased among asphalt workers, SMR = 1.17 (95% C.I. 1.04–1.30) and from head and neck cancer SMR = 1.27 (95% C.I. 1.02–1.56). Moreover, an exposure-response relationship for lung cancer, based on 63 deaths, was found using the metric of cumulative exposure: RR = 1.43 (95% C.I., 0.87–2.33), 1.77 (95% C.I., 0.99–3.19), and 3.53 (95% C.I. 1.58–7.89), respectively (p-value test for trend, 0.01). Similarly, results were found for unlagged average exposures.

An earlier study involving a meta-analysis of 20 epidemiological studies did not find overall evidence for lung cancer risk among paving workers exposed to asphalt (RR = 0.87, 95%

C.I., 0.76–1.08); however, a statistically significant excess for lung cancer in roofers was determined (RR = 1.78, 95% C.I., 1.5–2.1).[8]

Do these findings indicate that asphalt exposure is a causal factor in cancer (specifically lung cancer, but also head and neck and other cancers)?

The strength of an association in an epidemiological study, such as those found in the studies by Boffetta et al. and Partanen and Boffetta,[6,7] increases the credibility of the findings; however, the fact that an association is weak does not rule out a causal connection.[9,10] The strength of an association is not a biologically consistent feature, but rather a characteristic that depends on the relative prevalence of other causes. A cause and effect hypothesis is further strengthened when results of a study demonstrate that an incremental or decremental change in the exposure factors was accompanied by a corresponding change in a risk indicator.[9,10]

The associations in the study by Boffetta et al.[6,7] are SMR = 1.17 for lung cancer and SMR = 1.27 for head and neck cancer. Traditionally associations between 1.2 and 1.6 in epidemiological studies imply a weak hazard.[10] Weak associations are more likely to be explained by undetected biases.

The finding of an exposure response relationship for two metrics of exposure (cumulative and average) contributes to the evidence base for a causal connection between asphalt exposure and lung cancer. In contrast, the meta-analysis of 20 studies does not show any excess of lung cancer in paving workers.[8] Whereas, for roofers, the excess RR was 1.78, which is typically considered to imply a moderate hazard and at a level to provide more confidence in the findings although the heterogeneity of conditions in the various studies, is not a guard against the influence of various types of biases. In epidemiological studies, the evaluation of associations between exposure and risk factors will be tempered by the extent to which there has been control for known or suspected confounding factors. For studies of roofers and pavers, there had been little control for confounding factors.

The biological plausibility of the two studies is integral to the acceptance of the association as causal. For asphalt fumes, biological plausibility of the associations found in the epidemiological studies is bolstered by the composition of the fume as described in the World Health Organization (WHO) assessment that: "Under various performance specifications, it is likely that asphalt fumes and paints contain carcinogenic substances."[11] This finding was based upon a review conducted by NIOSH in 2000.

Is this conclusion the same as saying that asphalt fume is carcinogenic? At issue is whether the conditions of use are such that they result in the formation and emission of carcinogenic compounds and whether exposure to such a mixture results in an excess of lung cancers. The chemical composition of asphalt fumes is broadly variable and dependent on the chemical composition of the original crude petroleum, the manufacturing process, and the temperature of use/application.

Ideally, for determining biological plausibility and ultimately, causality, human studies of a potential hazard are corroborated by animal and in vitro studies. In the case of asphalt fumes, it has been difficult to conduct studies with asphalt fumes that are similar in composition to what is encountered in the workplace. In vitro analysis of mutagenicity correlated with generation temperature and collection method.[3]

Generally all laboratory-generated roofing and paving asphalts were positive for mutagenicity in the Ames salmonella assay, fumes collected above the head space of an asphalt storage tank during paving operations were not.

Moreover, no animal inhalation studies have been reported that utilized a fume representative of what would occur in human occupational exposures. Consequently, to date there are no lung cancer findings in experimental animals that are sufficient to provide support for the hypothesis that asphalt fumes cause human lung carcinogen.

There are, however, various mechanistically relevant human and animal studies that can be further used to investigate the link between exposure to asphalt fumes and cancer. These include DNA strand breaks in roofers but not pavers or painters,[12] and DNA strand breaks in roofers.[12,13] Although the findings have not been entirely consistent, they do lend mechanistic support to the link between asphalt fume exposure and cancer. The major uncertainties in hazard characterizations of asphalt fumes are summarized in the Table I. Research is needed to address these uncertainties. More research is needed to characterize the fume and the hazard.

Research Needs

NIOSH identified the following research needs pertinent to lung cancer[1]:

- Human studies
  - If suitable population and methods are found, design and conduct epidemiologic studies that address potential confounders such as smoking, diet, coal-tar, and diesel.
  - Conduct studies of biomarkers associated with cancer.
- Animal studies
  - Generate asphalt fumes that are chemically representative of roofing and paving fumes and test their carcinogenic potential with lifetime inhalation bioassays in rodents.

TABLE I. Major Uncertainties in Hazard Characterization of Asphalt/Bitumen

	Description
Chemistry	Complex mixture that varies according to crude source and manufacturing process
Animal studies	Inconsistent findings in studies using laboratory- and field-generated fumes
Human studies	Inconsistent epidemiologic findings[11]

- Generate asphalt fumes that are chemically representative of roofing and paving fumes to conduct skin-painting studies to determine tumor genicity.

- Fume characterization
  - Continue to assess the chemical differences between laboratory-generated and field-generated (during paving and roofing) fumes and continue the research on constituents of fumes that correlate with health effects.
  - Conduct additional studies of genotoxicity and mutagenicity of fumes collected during paving, roofing, using the modified Ames assay.
  - Develop methods for determining the total three-ring and higher PAC content, and for determining individual PACs and other chemical analyses so that correlations can be made between concentrations and biomarkers that indicate exposures to known carcinogens and genotoxins.
- Characterize the particle size of fumes (which is related to the hazard potential of inhaled chemical).

The hope of the organizers of this meeting is that scientific research currently underway or recently completed can be presented in a timely fashion for review and comment. Thus various uncertainties in the carcinogenic hazard classification of asphalt/bitumen can be addressed.

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