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Asphalt and risk of cancer in man

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Abstract

Epidemiological publications regarding the carcinogenic potential of asphalt (bitumen) are reviewed. In 1984 the International Agency for Research on Cancer (IARC) stated that there is "inadequate evidence that bitumens alone are carcinogenic to humans." They did, however, conclude that animal data provided sufficient evidence for the carcinogenicity of certain extracts of steam refined and air refined bitumens. In the absence of data on man, IARC considered it reasonable to regard chemicals with sufficient evidence of carcinogenicity in animals as if they presented a carcinogenic risk to man.

Epidemiological data for man accumulated since the IARC report do not fulfil the criteria for showing a causal association between exposure to asphalt and development of cancer. The studies cited all suffer from a lack of data on exposure or potential confounders, which are necessary to establish whether or not such an association may or may not exist. In view of the evidence (or lack thereof) regarding asphalt today, an appropriate public health attitude suggests at least that action be taken to protect those working with asphalt by monitoring the workplace, taking whatever steps are possible to minimise exposures and to inform workers of potential hazards. At the same time, a need exists for well designed analytical epidemiological studies to determine whether a risk of cancer in man exists from exposure to asphalt.

The carcinogenic potential of asphalt (bitumen) and asphalt fumes continues to be of concern. This derives from the potential presence of polycyclic aromatic hydrocarbons (PAHs) in asphalt (although the precise chemical composition may be dependent on the source of the crude oil and the manufacturing process) and persists despite the suggestion that the manufacturing process is likely to remove essentially all of those aromatic hydrocarbons that are known or suspected to be carcinogens.[1]

Asphalts are manufactured by vacuum distillation of crude petroleum oils. Steam may be entered into the distillation process and, depending on the particular crude material and the requirements of end users, the vacuum residue may be further processed by air blowing or by solvent precipitation. Asphalt products are grouped into four categories, asphalt cements, air blown asphalts, cutback asphalts, and emulsified asphalts. Major applications of asphalts include road paving, roofing, and flooring with over 80% used in road construction and maintenance. Much of the remainder is used in the manufacture and installation of roofing products. A complete description of the manufacturing process, chemical composition, and applications are available from King *et al*[1] and IARC.[2]

Several reviews have appeared regarding the carcinogenic potential of petroleum hydrocarbons but these deal only briefly with the issue of asphalt. In 1980, Bingham *et al* reviewed the carcinogenic potential of petroleum hydrocarbons.[3] The animal data suggested a potential for inducing cancer by petroleum derived asphalts that appeared to be dependent on the source of the asphalt and the processing of the final mixtures. These reviewers indicated that it was difficult to interpret the available information on the possible role of petroleum derived bitumens in the production of cancer in man. Epidemiological evidence was scarce and study of the effect of asphalts was complicated by the fact that "the working histories of most individuals in the paving and roofing industries include previous or combined exposure to coal tar, which is considered by the US National Institute for Occupational Safety and Health to be a known carcinogen."[3]

In 1982, the World Health Organisation task group on environmental health criteria for selected petroleum products, after reviewing the then current evidence, concluded that "although some bitumens applied to the skin of mice exhibit carcinogenic activity, it is low compared with that of coal tar, and it is generally accepted that the toxicity of bitumens is low."[4] Review of the data for man resulted in the conclusion that "petroleum based bitumens do not present a significant health hazard." The reviewers

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also concluded, however, that despite the lack of substantiating data, the possibility has to be considered that bitumen and bitumen vapours might contribute to the overall incidence of cancer of the skin and respiratory tract in view of their content of polynuclear aromatic compounds.

In 1984, the IARC working group on the evaluation of carcinogenic risk of chemicals to humans reviewed the evidence regarding carcinogenicity of bitumens.[2] The working group cautioned that bitumens must be distinguished from coal tars, which are the products of the destructive distillation of coals, and also from coal tar pitches, which are residues from the distillation of coal tars because such coal tar products are sometimes used in applications for which bitumens are employed. They did, however, state that animal data provided sufficient evidence for the carcinogenicity of certain extracts of steam refined and air refined bitumens. It was concluded by IARC that there is "inadequate evidence that bitumens alone are carcinogenic to humans." In the absence of data on man, IARC considered it reasonable to regard chemicals for which sufficient evidence of carcinogenicity exists in animals as if they presented a carcinogenic risk to man.

Rothman and Emmett (1988) considered the carcinogenic potential for a broad range of petroleum derived products and dealt briefly with asphalt.[5] Based primarily on the toxicological publications, they concluded that "although petroleum derived asphalts have been consistently shown to be markedly less carcinogenic than coal derived products, their carcinogenic potential is still of some concern." Further, they suggested that exposure to volatile components of asphalt produced by heating may prove to be more hazardous than exposure to the complete compound. In view of the toxicological evidence, they judged that highway and roofing workers need to be evaluated for excess cancer risk presumably because over 80% of asphalts are used in these occupations.

Invariably, as indicated above, the data for man have been limited and have not supported a cause and effect relation between exposure to asphalt and cancer. In fact, they may confuse rather than illuminate the issue. Often cited are two publications appearing in 1976; (1) an excess of lung cancer (not statistically significant) among roofers in a population based death certificate study by Menck and Henderson[6] and (2) raised death rates from lung cancer and cancer of several other sites among roofers studied by Hammond *et al.*[7] Neither study, however, directly considered the issue of the result of exposures to asphalt. The first study had no assessment of exposure and utilised occupation as recorded on the death certificate only as a surrogate for exposure to asphalt. Not only did the second study have no quantitative measures of exposure to asphalt or its constituents for the studied cohort, it was also confounded both by exposure of workers to coal tar products and lack of information on cigarette smoking.

The reviews dealing with asphalt are unanimous in concluding that the carcinogenicity of extracts of bitumens has been shown in experimental animals. They also agree that data for man are lacking and further evidence is needed to assess whether there is a potential threat to producers and users of asphalt products. The purpose of the current paper is to review the most recent (peer reviewed) epidemiological publications on the carcinogenic potential of asphalt in man and to determine what further evidence, if any, has been made available since the IARC review.

Studies in man

Although the epidemiological publications considering man remain limited, several studies have appeared in the last few years with mixed results regarding any potential association between exposures to asphalt and cancer. Momsen and Aagard (1984) performed a case-control study of 212 bladder cancer patients and 259 controls.[8] Cases were admitted to the Department of Oncology and Radiotherapy, University of Aarhus, Denmark. Lifetime occupational histories plus socioeconomic state and some demographic factors were obtained by self administered questionnaire with telephone follow up of both cases and controls. Categorisation within a particular industry required employment for a minimum of one year and analyses were carried out using the job held for the longest period. They report an increased though not statistically significant relative risk of developing bladder cancer for several employment categories including "work with kerosine or asphalt" (odds ratio = 3·12; 95% confidence interval 0·88–11·00). Unfortunately, not only were no exposure data presented, the main occupation on which exposure was inferred was not limited to asphalt so this study can be viewed as mildly suggestive at best.

Hansen (1989) reports on a cohort of 679 male mastic asphalt workers identified from 1959 to 1980 with follow up to 1 January 1985.[9] Mastic asphalt is described as a mixture of fine sand, stone powder, and finely divided limestone with 12–17% of hard bitumen and is used for surfacing roads and in flooring and roofing. Cases of newly diagnosed cancers (incidence) were identified through the Danish Cancer Register. Expected cancer incidence was calculated from age, period, and site specific cancer incidence rates for Danish men for the period 1958–82. Statistically significant increases were noted for cancers of the mouth (observed = 2), oesophagus (observed = 3), rectum (observed = 7), and lung (observed = 27). No exposure data were available

and a large proportion of the workers had worked with asphalt for less than six years. Also no possibility existed for the control of potential confounders such as smoking and urbanisation. Substantially more of the mastic asphalt workers were smokers when compared with the general population and almost all of the workers were city dwellers compared with 40% of the comparison population. Also, it was possible that some of the cohort had been exposed to coal tar pitch during the second world war.

Hansen suggests that the association is too strong to be explained by confounding or random variation and postulates that exposure to cracking products in the fumes of heated bitumen has contributed to the raised incidence of cancer found. Because of the lack of data on exposure to asphalt and potential confounders, however, the data are too weak to stand on their own in implicating asphalt fumes as a potential carcinogen. They do, however, suggest the need for further investigation taking into account total employment history along with data on exposure to asphalt fumes and potential confounders.

In a separate report, Hansen (1989) gives the results of a historical cohort study of subjects identified from the Danish national census of November 1970.[10] Self reported occupation, trade industry, and employment on the day of the census were recorded for Danish inhabitants over the age of 14. A total of 1320 asphalt workers and 43 024 other unskilled workers were identified and the cohort was traced for 10 years through national record systems available in Denmark. Mortality among the asphalt workers was compared with the mortality experience of the other unskilled workers. Standardised mortality ratios (SMRs) were raised for respiratory and bladder cancers but the differences were not significant. A number of potential errors are discussed by the author. The most serious deficiency is the classification of "exposure" by employment on a single day. As pointed out by the author, the exposed category may include those with short term, low, or even no exposure and the unexposed category may include those previously exposed and comparisons in mortality between the two groups may not reflect differences in asphalt experience. Furthermore, the possibility exists for differences in lifestyle between the exposed and unexposed groups, which can have an impact on the mortality experience. Thus this study fails to advance our understanding as to whether any association exists between exposure to asphalt and cancer in man.

Studies of mortality among highway workers in California and Minnesota have been reported. Maizlish *et al* (1988) presented a proportionate mortality ratio (PMR) analysis of California Department of Transportation Employees who left employment for any reason between 1970 and 1983 and died in California between those dates.[11] Although PMR analyses are useful for mortality surveillance and hypothesis generation, exposure data were not sufficient to associate any increased PMR with specific job hazards, job classifications, job duration, or time since first exposure. Lack of exposure data, work histories, knowledge of previous or concomitant non-highway department employment, and data on non-occupational risk factors preclude focused hypotheses. As asphalt is only one of many potential exposures, further study is needed to confirm that excess risks exist among these employees and if so to determine whether asphalt or any other factors are associated.

Bender *et al* (1989) conducted a historical cohort mortality study of highway maintenance workers in Minnesota.[12] The cohort consisted of 4849 workers who had one or more years of experience as a highway maintenance worker with the Minnesota Department of Transportation and had worked at least one day after 1 January 1945. Follow up was through 31 December 1984 and the Minnesota mortality experience for white men was used to calculate the expected number of deaths. Although no precise exposure data were available, it was stated that these highway maintenance workers were potentially exposed to a variety of substances including asphalts. Further, the authors assert that highway maintenance work in Minnesota has not employed coal tar products for 50 years.

Overall mortality, all cancer mortality, and lung cancer mortality were significantly low. These deficits were not explained on the basis of differential smoking as such an interpretation was inconsistent with the SMRs of other smoking related causes of death, which were nominal. Increases were noted for leukaemia and urological cancers and the paper states that further investigations are underway to determine if these increases are occupationally related.

Epidemiological evidence for causation in man

The epidemiological data accumulated for man since the IARC report do not fulfil the criteria for showing a causal association between exposure to asphalt and development of cancer.[13] The studies cited all suffer from a lack of data on exposure or potential confounders, which are necessary to establish whether or not such an association may or may not exist. In carrying out a study of the feasibility of conducting an epidemiological investigation among asphalt roofing manufacturers in the United States, the authors have determined that historical exposure data for this industry are extremely limited and any association found might be confounded by smoking or the presence of asbestos before the introduction of fibreglass felts for roofing shingles. Such exposure data as may be available could provide only a qualitative assessment of exposure to asphalt fumes, silica,

limestone, asbestos, and talc, which must be assigned on the basis of limited job histories. This would suggest that historical cohort studies could go back only as far as adequate industrial hygiene data were available, perhaps to the early 1980s for the roofing manufacturing industry and even more recently for paving and roofers. Possibly the only definitive studies might be forward cohort studies and given the long latent period for lung cancer, an answer would not be forthcoming for many years. Nevertheless, such studies need to be undertaken if the question of the carcinogenicity of asphalt in man is to be resolved. We would agree with Rothman and Emmett[5] that the carcinogenic potential of petroleum derived asphalts is of concern and highway and roofing workers working with these substances need to be evaluated for evidence of excess cancer risk.

Summary

Even though the epidemiological data to date are inadequate to suggest, with any degree of scientific certainty, whether asphalt does or does not present a cancer risk to man, general agreement exists that, on the basis of animal studies, asphalt may pose such a risk.

Well designed analytical epidemiological studies are needed to determine if there is a cancer risk to man from exposure to asphalt. Nothing is to be gained by further descriptive studies that suffer from the same deficiencies as those cited. Little progress has been made in a decade and a half in resolving the issue and different approaches need to be taken. Future studies must include information on exposure as well as on potential confounders. Such studies would be time consuming and costly but both the time and effort can be justified considering the large number of workers with potential exposure.

There remains the question of what action, if any, should be taken at this time. There need not be conflict between "a public health attitude which accepts the risk of having excess concern for a still uncertain problem in contrast to the more conservative assumption that there is no risk until it is scientifically proven."[14] Concurrent actions can be taken while awaiting the "scientific certainty". Reports by Silverstein *et al*[15] and Knecht and Woitowitz[16] illustrate the potential for the "public health attitude" by identifying potential problems and suggesting appropriate corrective action before establishment of risk with "scientific certainty". For example, the second study reports that in Germany, tar bitumen is used as a binder for asphalt mixes used in road works or the laying of load bearing binding and surface layers. Tar bitumen is a standardised product containing about 70% road bitumen and 25–30% special coal tar pitch. Whereas it is unclear how extensive the practice is world wide for combining coal tar pitch with asphalt in road paving, their investigation serves to illustrate the point here. It targeted the PAH profile in road works utilising tar bitumen and concluded that it was possible to determine quantitatively several PAHs with carcinogenic potential including benzo(a)pyrene. Because factors such as varying temperatures of the asphalt mix and the variable pitch content of the binder lead to considerable variation in the PAH content of the daily profile, the PAH content presents as identifiable potential carcinogenic risk. They suggest that even with no further investigation, the continued use of tar bitumen in road works should be questioned and consideration given as to whether an alternative product can replace coal tar as the binder. Taking steps to do so would be consistent with the "public health attitude" described above.

Changes in the production process in the asphalt roofing manufacturing industry in recent years clearly already have resulted in reduction of exposure to asphalt fumes. At one time, roofing felt manufacture was based on immersion in asphalt of a dry felt made from waste paper or rags. The immersion process posed the possibility of heavy exposure, especially when repairing a "break" in the production line. Today many roofing felts are made of fibreglass for which no immersion is required, thus lessening the possibility of exposure on the production line.

Chong *et al*[17] suggest going even further and taking a pragmatic approach to standard setting with regard to asphalt. The issue of risk assessment and standard setting for asphalt is beyond the scope of this paper. We believe, however, that in view of the evidence (or lack thereof) regarding asphalt today, an appropriate public health attitude requires at least that action be taken to protect those working with asphalt by monitoring the workplace, taking whatever steps are possible to minimise exposures and to inform workers of potential hazards. At the same time, well designed analytical epidemiological studies are needed to determine whether there is a cancer risk to man from exposure to asphalt.

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References should be numbered consecutively in the order in which they are first mentioned in the text by Arabic numerals above the line on each occasion the reference is cited (Manson[1] confirmed other reports[2–5] . . .). In future references to papers submitted to the *Br J Ind Med* should include: the names of all authors if there are six or less or, if there are more, the first three followed by *et al*; the title of journal articles or book chapters; the titles of journals abbreviated according to the style of *Index Medicus*; and the first and final page numbers of the article or chapter.

Examples of common forms of references are:

1 International Steering Committee of Medical Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1979;1:532–5.
2 Soter NA, Wasserman SI, Austen KF. Cold urticaria: release into the circulation of histamine and eosino-phil chemotactic factor of anaphylaxis during cold challenge. *N Engl J Med* 1976;**294**:687–90.
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