

Cancer incidence in an occupational cohort exposed to bitumen fumes

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HANSEN ES. Cancer incidence in an occupational cohort exposed to bitumen fumes. *Scand J Work Environ Health* 1989;15:101-5. This study was conducted to investigate whether bitumen fumes should be considered carcinogenic to human beings. A historical cohort of heavily exposed mastic asphalt workers was followed from 1959 through 1984 (inclusive) with regard to cancer incidence. A total of 679 Danish men were included in the study cohort. Among these, 75 new cases of cancer were observed within the period studied. The cancer incidence observed among the group significantly exceeded that of the total Danish male population, the standardized morbidity ratio (SMR) being 195 (95 % confidence interval (95 % CI) 153-244). Significant increases were seen for cancer of the mouth (SMR 1111, 95 % CI 135-4014), the esophagus (SMR 698, 95 % CI 144-2039), the rectum (SMR 318, 95 % CI 128-656), and the lung (SMR 344, 95 % CI 227-501). It is suggested that exposure to cracking products in the fumes of heated bitumen has contributed to the elevated cancer incidence observed.

Key terms: asphalt, buccal cancer, esophageal cancer, lung cancer, rectal cancer.

Bitumens are produced as residues from the fractional distillation of crude oil. They are widely used because of their adhesive and waterproofing properties. It has been estimated that the current annual world use is over 60 million tons (1). Major applications are in paving for roads and airfields, hydraulic uses (such as dams, water reservoirs, and sea-defense works), roofing, flooring, and the protection of metals against corrosion. Bitumens contain only relatively small amounts of polycyclic aromatic hydrocarbons (PAH) (1). [The reason for these low amounts may be that the refinery process used for the manufacturing of bitumens removes the majority of compounds with lower boiling points, including PAH with three to seven fused rings, which include all of the PAH compounds which have thus far been considered to have carcinogenic effects by the International Agency for Research on Cancer (2)]. The recommended temperatures for handling bitumen-based asphalt mixes range from 65 to 230°C, depending on the type of bitumen (1). The generation of bitumen fumes increases steeply as the temperature increases (3). Besides, the heating may initiate cracking processes which may cause substances not present in the unheated bitumen to form. Several countries, including Denmark, have adopted a time-weighted average (TWA) of 5 mg/m³ as the administrative limit for occupational exposure to bitumen fumes.

Previous epidemiologic studies have shown an increased cancer mortality among roofers, whose occupational exposure includes heated bitumen and bit-

umen fumes. Menck & Henderson (4) used registry data to estimate the lung cancer mortality for a number of occupational groups in Los Angeles County in the United States. For roofers, they found a lung cancer mortality that was about five times that of the population at large. Another study on roofers' mortality was carried out by Hammond and his co-workers (5). They conducted a 12-year follow-up of a cohort of roofers identified from union files in the United States. Among long-term employed roofers, they found an increased mortality from malignant neoplasms in the buccal cavity and pharynx, the digestive organs, the respiratory system, the prostate gland, the bladder, and the skin and from leukemia. However, although the studied groups of roofers (4, 5) had been exposed to bitumen fumes, no clear conclusions could be drawn regarding the carcinogenicity of these fumes because the roofers had been concomitantly exposed to the known carcinogen coal tar.

Only a few experimental studies have been carried out on the potential carcinogenic effects of bitumen fumes. Inhalation studies on rats and guinea pigs (6) and on mice (7) have failed to demonstrate any carcinogenic effect of bitumen fumes. Nor could an effect be demonstrated in a positive control group exposed to coal-tar fumes (6). The employed animal exposure model must thus be rather insensitive, and the apparently negative findings are not informative regarding the potential carcinogenic effects of bitumen fumes. However, a recent animal assay using skin application has shown a strong carcinogenic effect of condensate of bitumen fumes on mice (8). Sixty-six percent of the animals developed malignant neoplasms after skin painting with condensed bitumen fumes. The effect of bitumen fume condensate was almost as strong as that seen in the positive control group, in which the animals were painted with condensed coal-

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tar fumes derived at the same temperatures as the bitumen fumes. The concentration of known carcinogenic PAH was much lower in the condensate of bitumen fumes than in the coal-tar fumes. Chemicals other than the measured PAH must therefore be involved in producing the observed strong carcinogenic effects of bitumen fumes, either as initiators, promoters, or as cocarcinogens.

The present study was set up to investigate further the relationship between human cancer risk and exposure to bitumen fumes. The study considered the cancer incidence in an occupational cohort of Danish mastic asphalt workers heavily exposed to bitumen fumes only.

Subjects and methods

A historical cohort of male mastic asphalt workers was set up from employment lists and union records and followed with regard to cancer incidence during the period 1959–1984.

Exposure

Mastic asphalt is a mixture of fine sand, stone powder, and finely divided limestone with a high content (12–17 %) of hard bitumen. Mastic asphalt is used for surfacing roads and in flooring and roofing. The asphalt mix is manufactured at an asphalt plant, poured into heated, closed tanks, and carried to the place where it is to be applied. The asphalt mixture is kept at a temperature of approximately 250°C until it is filled into buckets. The asphalt workers carry the buckets containing the hot asphalt mixture to the place of application, where it is tipped out and layered by hand floating — a leveling technique similar to that used for smoothing cement. On his knees, the float troweler draws out the hot asphalt mixture with a long wooden trowel. While handling the hot mastic asphalt mixture, the workers may be exposed to high concentrations of bitumen fumes. Hygienic measurements carried out by the Danish National Institute of Occupational Health (Rietz B, internal report of the Danish National Institute of Occupational Health, in Danish) indicate that the TWA of 5 mg/m³ is being considera-

bly exceeded during flooring with mastic asphalt. A total of 35 personal samples was taken during flooring. The concentration of asphalt fume condensate in these samples ranged from 0.5 to 260 mg/m³ with a median of 19.7 mg/m³ and a mean of 41.5 mg/m³. The amount of PAH was determined in nine of the 35 samples for an estimation of the PAH concentration in the workplace air. For total PAH (16 standards), the median equaled 0.183 mg/m³, and the mean was 0.195 mg/m³. For benzo(a)pyrene, the median was 0.004 mg/m³, and the mean 0.0058 mg/m³. Only two samples were taken during manual road paving. The concentration of asphalt fume condensate was estimated to be 4.3 mg/m³ in one and 3.4 mg/m³ in the other. Results similar to these have been reported in several other studies on mastic asphalt workers' exposure to asphalt fumes during flooring and paving. (See reference 1.)

As mentioned previously, mastic asphalt is a mixture of bitumen and mineral aggregates. The mastic asphalt mixture contains an excess of bitumen, and the mineral aggregate used is very fine-grained, which ensures that the technical properties of the mixture are optimal. Therefore, technical auxiliary substances such as coal tar are not added to the mastic asphalt mixture. However, during World War II a shortage of bitumen initiated the use of coal-tar pitch in the production of asphalt mixtures, including that of mastic asphalt.

Identification and follow-up of the subjects

A total of 679 mastic asphalt workers, all men, was entered into the study. The subjects were identified from historical files that covered the period 1959 through 1980. Four hundred subjects were identified from the employment lists of four mastic asphalt plants, 186 subjects from the membership files of an organized group of mastic asphalt workers affiliated with the National Union of General Workers, and 93 subjects from the membership files of a benefit society organized by the workers at one of the mastic asphalt plants. A subject was identified either by name and address, name and date of birth, or by his personal identification number and enrolled into the study by the time when he was first identified in one of the historical files used for establishing the study cohort. The follow-up of a subject was stopped at death, emigration, or on 1 January 1985, whichever occurred first. Information on death and emigration was obtained from the local and central offices of the national register, and by this procedure everyone was traced. By the end of the follow-up, ie, on 1 January 1985, 524 subjects were alive and living in Denmark, 149 had died, and 6 had emigrated. The study comprised 6 692 person-years at risk (table 1). The occurrence of new cases of cancer was ascertained through a search for the cohort members in the Danish Cancer Register.

Table 1. Number of person-years at risk for the 679 mastic asphalt workers by age and subcohort.

Age (years)	Subcohort			Total
	I ^a	II ^b	III ^c	
15–39	·	75	1 995	2 069
40–54	265	742	1 230	2 237
55–64	793	568	·	1 361
65–89	1 025	·	·	1 025
Total	2 083	1 384	3 225	6 692

^a The 194 persons born in 1893–1919.

^b The 129 persons born in 1920–1929.

^c The 356 persons born in 1930–1960.

Table 2. Cancer incidence, standardized morbidity ratio (SMR), and 95 % confidence interval for the 679 mastic asphalt workers, all ages combined.

Cancer site ^a	Observed number of cases	SMR	95 % CI ^b
Buccal cavity and pharynx (140—148)	2	167	20—602
Digestive organs and peritoneum (150—159)	22	227	142—344
Respiratory system (160—164)	31	347	236—493
Breast (170)	1	1 429	36—7 959
Male genital organs (177—179)	5	120	39—280
Urinary system (180—181)	6	133	49—289
Skin (190—191)	4	78	21—199
Other specified sites (192—197)	1	68	2—379
Secondary and unspecified sites (198—199)	1	199	3—663
Lymphatic and hematopoietic tissue (200—205)	2	83	10—299
All malignant neoplasms (140—205)	75	195	153—244

^a Code of the International Classification of Diseases (seventh revision) in parentheses.

^b Poisson-based 95 % confidence interval for the SMR.

Data analysis

The expected cancer incidence in the cohort of mastic asphalt workers was calculated from age-, period-, and site-specific cancer incidence rates for Danish men during the period 1958—1982. Because data on each individual's exposure history were generally not available, induction/latency time from first exposure to the establishment of the cancer diagnosis has not been considered in the analysis. However, the age grouping represents a proxy for an analysis by time from first exposure because new mastic asphalt workers are always young, typically in their early twenties, when they start this job and because the turnover is only moderate in this occupational group. Thus, the analyses of mastic asphalt workers aged 40 years or more may be considered as approximately equal to analyses in which a 15- to 20-year induction/latency time is required. Albeit a crude proxy, analysis by age seemed to be the only alternative to ignoring the induction/latency time totally.

The study cohort included persons born within the years 1893—1960. It is therefore likely to be rather inhomogeneous as regards several risk factors for cancer. In addition, some of the subjects may have been asphalt workers during World War II and may have been exposed to coal-tar pitch. For these reasons, the cohort of mastic asphalt workers was divided into three subcohorts by the subjects' time of birth as follows: subcohort I: persons born in 1893—1919 (coal-tar exposure during World War II likely), subcohort II: persons born in 1920—1929 (some coal-tar exposure during World War II possible), and subcohort III: persons born in 1930—1960 (coal-tar exposure during World War II impossible).

The calculation of the tests and confidence intervals was based on the assumption that the observed numbers were Poisson-distributed.

Table 3. Site-specific cancer incidence, standardized morbidity ratio (SMR), and 95 % confidence interval for the 547 mastic asphalt workers aged 40—89 years.

Cancer site ^a	Observed number of cases	SMR	95 % CI ^b
Mouth (143—144)	2	1 111	135—4 014
Esophagus (150)	3	698	144—2 039
Stomach (151)	4	190	52—488
Colon (153)	5	198	64—463
Rectum (154)	7	318	128—656
Liver (155)	2	476	58—1 720
Larynx (161)	3	435	90—1 271
Lung (162.0—162.1)	27	344	227—501
Prostate (177)	4	119	32—305
Bladder (181)	5	155	50—361
Skin (non-melanoma) (191)	3	67	14—196
Leukemia (204)	0	0	0—401
Other	9	101	46—191
Total (140—205)	74	199	156—250

^a Code of the International Classification of Diseases (seventh revision) in parentheses.

^b Poisson-based 95 % confidence interval for the SMR.

Table 4. Histopathological diagnoses for the 27 cases of primary lung cancer in the study cohort.

Case	Histopathologic diagnosis	Age (years) at diagnosis	Year of birth
1	Squamous cell carcinoma	44	1934
2	Squamous cell carcinoma	49	1920
3	Squamous cell carcinoma	50	1927
4	Squamous cell carcinoma	53	1923
5	Squamous cell carcinoma	58	1923
6	Squamous cell carcinoma	63	1916
7	Squamous cell carcinoma	64	1917
8	Squamous cell carcinoma	67	1900
9	Squamous cell carcinoma	69	1905
10 ^a	Squamous cell carcinoma	72	1906
11	Squamous cell carcinoma	76	1904
12	Anaplastic carcinoma	48	1931
13	Anaplastic carcinoma	55	1918
14	Anaplastic carcinoma	61	1916
15 ^a	Anaplastic carcinoma	65	1915
16	Anaplastic carcinoma	66	1900
17	Anaplastic carcinoma	71	1912
18	Anaplastic carcinoma	71	1904
19	Anaplastic carcinoma	74	1900
20 ^a	Anaplastic carcinoma	77	1902
21	Adenocarcinoma	64	1916
22	Unspecified	50	1930
23	Unspecified	58	1924
24	Unspecified	59	1919
25	Unspecified	61	1921
26	Unspecified	68	1913
27	Unspecified	75	1905

^a Nonsmoker.

Results

The observed cancer incidence among the mastic asphalt workers was almost two times greater than that of the total population. The increased cancer incidence derived mainly from malignancies of the respiratory system and the digestive organs (table 2). For persons aged 40 years or more, statistically significant increases were seen for cancer of the mouth, esophagus, rectum, and lung (table 3). Twenty-seven cases of primary lung cancer were observed among the mastic asphalt workers (table 4). Of the 27 lung cancer cases, 18 occurred

Table 5. Standardized morbidity ratio (SMR) and 95 % confidence interval^a (95 % CI) for primary lung cancer of the mastic asphalt workers by age at diagnosis and subcohort, 27 cases of lung cancer in 1959–1984.

Age (years) at diagnosis	Subcohort							
	I ^b		II ^c		III ^d		Total	
	SMR	95 % CI	SMR	95 % CI	SMR	95 % CI	SMR	95 % CI
40–54	0	0–2 306	682	141–1 993	857	177–2 505	632	232–1 375
55–64	347	127–755	275	57–804	.	.	319	146–606
65–89	295	152–515	.	.	.	.	295	152–515
40–89	302	179–477	392	144–854	857	177–2 505	344	227–501

^a Poisson.

^b The 194 persons born in 1893–1919.

^c The 129 persons born in 1920–1929.

^d The 356 persons born in 1930–1960.

in subcohort I, six in subcohort II, and three in subcohort III. The lung cancer incidence was significantly increased in each of the three subcohorts (table 5). As for other diagnoses, the results for the subcohorts are not shown because almost all of the cases occurred among workers aged 65 years or over, and there were no men of these ages in subcohorts II and III.

Discussion

This study showed an increased cancer incidence in the cohort of mastic asphalt workers. An obvious explanation may be that hot mastic asphalt involves exposure to carcinogenic substances. But before drawing this conclusion, one needs to consider the potential bias in the study.

It was not possible to control for potential differences in life-style (including smoking, drinking, and dietary habits) between the cohort of mastic asphalt workers and the Danish men of the same age in general during the same period. In particular, control for smoking habits would have been advantageous, because smoking is intimately associated with respiratory cancer. Smoking, however, is so prevalent among Danish men that, even if all the asphalt workers had been heavy smokers, smoking could explain no more than a doubling of their lung cancer risk as compared with that of the general population (9). An inquiry in 1976 to mastic asphalt workers in Copenhagen showed that 22 % were nonsmokers, 36 % were medium smokers (ie, daily consumption 1–14 g of tobacco), and 43 % were heavy smokers (ie, daily consumption 15 g of tobacco or more) (10). Similarly, a population survey in 1982 showed that, among men of the same age, 39 % were nonsmokers, 24 % were medium smokers, and 38 % were heavy smokers (11). Smoking differences of this order of magnitude will bring about approximately 20 % more lung cancer cases than “expected” in the group of mastic asphalt workers. The observed lung cancer incidence, however, was more than three times greater than expected. Therefore, though smoking presents an important risk of lung cancer, it is hardly likely to play a major role as a confounding factor in the present study.

The incidence rates for respiratory and digestive cancer among Danish men shows a rural-urban gradient (12). Almost all of the asphalt workers were city dwellers as opposed to only 40 % of the comparison population (13). Therefore, the expected incidence of respiratory cancer may have been underestimated by 35 %, whereas the expected incidence of digestive cancer may have been underestimated by 10 %.

After adjustment for the estimated confounding effect of urbanization and smoking habits, the observed incidence of both respiratory and digestive cancer was about two times greater than expected (adjusted standardized morbidity ratio for respiratory cancer 214 and for digestive cancer 206). All of the 679 mastic asphalt workers considered in this study had been exposed to asphalt fumes, but only some of them had been exposed long-term. Data on individual exposure history are lacking, but a previous questionnaire study among the mastic asphalt workers showed that half of them had been employed for less than 10 years and one-third for less than six years (10). Furthermore, it is necessary to consider the fact that mastic asphalt workers have been casual laborers and typically out of work during the winter months. As regards exposure intensity, the concentration of asphalt fumes has undoubtedly been much higher during flooring operations than during road paving. Roughly estimated, the concentration has been about 4 mg/m³ during road paving and about 20 mg/m³ during flooring operations (Rietz B, internal report of the Danish National Institute of Occupational Health, in Danish). Road paving has made up approximately two-thirds and flooring operations one-third of the workhours of the cohort in question. Thus, weighted over a 12-month period, the mastic asphalt workers’ average exposure has been almost identical to that of continuous exposure to asphalt fumes at the current Danish TWA of 5 mg/m³. The subcohort analysis revealed that the increased lung cancer risk had not been restricted to the mastic asphalt workers who had possibly been exposed to coal-tar pitch during World War II. As a matter of fact, the increased lung cancer risk tended to be manifest earlier (ie, at a younger age) in the subsequent birth cohorts. The numbers are too small, however, to justify

fy any conclusions as to a possible trend of increasing risk in the subsequent birth cohorts.

In conclusion, the results of this study indicate that the occupational exposure of mastic asphalt workers is associated with an increased risk of malignant neoplasms in the buccal cavity, the respiratory system, and the digestive organs. These results are in agreement with those of studies on roofers' mortality (4, 5). The occupational exposure of roofers resembles that of mastic asphalt workers, as both groups are exposed to fumes of heated bitumen. Bitumen fume is a disperse aerosol, the particles of which contain many different hydrocarbons. If this aerosol is inhaled, the small particles are deposited mainly in the deep, non-ciliated airways, whereas the larger particles are deposited on the ciliated epithelium of the conducting airways. The latter particles will be cleared from the airways by the mucociliary escalator mechanism and subsequently swallowed. This way, even the digestive organs may be exposed to the substances contained in bitumen fumes. Soiling of hands, face, and work-clothes also presents a risk of exposure via the mouth. The fumes from hot mastic asphalt contain the most volatile substances of the actual bitumen in addition to potential cracking products. The content of carcinogenic PAH is low in bitumen fume, as compared with that of heated coal tar (1, 8). Despite this difference, bitumen fumes have been shown to produce almost as many experimental cancers as have coal-tar fumes (8). These findings, combined with the fact that unheated bitumen is a rather ineffective carcinogen (1), point to the possibility that carcinogenic substances in bitumen fume may originate from cracking processes initiated by the heating of bitumen. Thus, in reference to the frame for causal inference which has been proposed by Hill (14), there seems to be reason to believe the hypothesis that bitumen fumes are carcinogenic.

The observed association is too strong to be explained by confounding or by random variation, and it has been demonstrated in different populations. Furthermore, the epidemiologic findings are biologically plausible, and the hypothesis is supported by experimental evidence. As to possible weak points in this reasoning, it cannot be totally excluded that the epidemiologic findings may have been brought about by another carcinogenic agent in the work environment. However, it is not likely that the effects demonstrated in the present study should be due solely to an unnoticed occupational risk factor.

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