Scand J Work Environ Health 1991;17(1):20-24

<https://doi.org/10.5271/sjweh.1739>

Issue date: Feb 1991

Mortality of mastic asphalt workers.

by [Hansen ES](#)

Affiliation: Institute of Community Health, University of Odense, Denmark.

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/2047802

This work is licensed under a [Creative Commons Attribution 4.0 International License](https://creativecommons.org/licenses/by/4.0/).

Mortality of mastic asphalt workers

by Eva S Hansen, MD¹

HANSEN ES. Mortality of mastic asphalt workers. *Scand J Work Environ Health* 1991;17:20-4. This study was conducted to investigate the adverse health effects of exposure to bitumen fumes. A cohort of 679 mastic asphalt workers was followed from 1959 to 10 June 1986, during this period 169 deaths occurred. The overall standardized mortality ratio (SMR) was 163 (95 % confidence interval (95 % CI) 141-190), the SMR was 225 (95 % CI 172-288) for cancer and 223 (95 % CI 130-358) for external causes. Among persons aged 40 to 89 years, significant increases were seen for lung cancer (SMR 290, 95 % CI 188-429), nonpulmonary cancer (SMR 200, 95 % CI 141-276), and liver cirrhosis (SMR 467, 95 % CI 188-962). Bronchitis, emphysema, and asthma also occurred in excess (SMR 207, 95 % CI 95-393). In conclusion, the inhalation of bitumen fumes may have contributed to the elevated mortality from cancer and respiratory diseases among mastic asphalt workers.

Key terms: bitumen fume, cancer, chronic bronchitis, lung cancer, respiratory disease.

Previous studies have shown an increased morbidity and mortality among persons occupationally exposed to bitumen fumes. Health surveys of asphalt workers have indicated an increased frequency of chronic respiratory disease (1-2). A study on roofers' mortality has revealed an increased mortality from respiratory diseases, cancer, and accidents (3). A Danish mortality study of unskilled asphalt plant employees has indicated an increased mortality from ischemic heart disease and from cancer involving the digestive organs, the respiratory system, the bladder, and the brain (4).

The actual cohort of mastic asphalt workers has previously been followed through 1984 with respect to registered cancer incidence. Compared with the population at large, the mastic asphalt workers displayed an increased incidence of cancer of the mouth, the esophagus, the rectum, and the lung (5).

Increased mutagenic activity has been found in the urine of asphalt workers exposed to rather low concentrations of bitumen fume (6).

Only a few experimental studies have been carried out on the potential carcinogenic effect of bitumen fumes. An animal assay using skin application has shown a strong carcinogenic effect of condensate of bitumen fumes on mice (7).

In summary, there is growing evidence that substances in bitumen fumes present a genotoxic hazard. Furthermore, an association has been reported between chronic bronchitis and the inhalation of bitumen fumes.

The present study was set up to investigate further the chronic health effects of exposure to bitumen fumes. The study considered cause-specific mortality in a cohort of mastic asphalt workers.

Subjects and methods

A historical cohort of male mastic asphalt application workers was set up from employment lists and union records; the cohort was followed with regard to mortality through 10 June 1986.

Mastic asphalt workers have been casual laborers and have typically been out of work during the winter months. Nevertheless, this occupational group has displayed a relatively low turnover. The reasons for this low turnover may be that they have been well paid and that they have typically worked in self-managed groups of 8-10 persons. Furthermore, it has been advantageous to the employer to keep these gangs of workmen intact, because the handling of mastic asphalt requires both experience and cooperation among the workers.

Exposures

Hygienic measurements carried out by the Danish National Institute of Occupational Health (8) indicate that the time-weighted average of 5 mg/m³ has been considerably exceeded during flooring with mastic asphalt. Reportedly, the concentration of asphalt fume condensate in 35 personal samples taken during flooring ranged from 0.5 to 260 mg/m³ with a median of 19.7 mg/m³. Only two samples were taken during manual road paving. In these, the concentration of asphalt fume condensate was 4.3 and 3.4 mg/m³. Similar results have been reported in several other studies on mastic asphalt workers' exposure to asphalt fumes during flooring and paving. (See reference 9.)

Mastic asphalt contains bitumen and mineral aggregates. Unlike asphalt mixes, mastic asphalt has not contained coal tar. There is no technical advantage of adding coal tar because the mastic asphalt contains an excess of bitumen, and the mineral aggregate used is

¹ Institute of Community Health, University of Odense, Odense, Denmark.

very fine-grained granite, which ensures that the adhesive and other technical properties are optimal. However, during World War II a shortage of bitumen led to the use of coal-tar pitch in the production of mastic asphalt.

Identification and follow-up

A total of 679 mastic asphalt workers, all men, was entered into the study from historical files covering the period 1959–1980. The establishment of the cohort has been described in detail previously (5). The follow-up of a subject was stopped at death, emigration, or on 10 June 1986, whichever occurred first. Information on death and emigration was obtained from the local and central offices of the national register, and by this procedure all of the subjects were traced. By the end of the follow-up (ie, on 10 June 1986) 504 subjects were alive and living in Denmark; 169 had died, and 6 had emigrated. For the decedents, information on underlying cause of death was obtained from the death certificate. The study comprised 7434 person-years at risk (table 1).

Data analysis

The expected numbers of death among the mastic asphalt workers were calculated from the national death rates for Danish men in 1960–1985 and were standardized for age and time period. Lung cancer was the only type of cancer for which site-specific death rates were available.

Because data on each individual's exposure history were not available, induction or latency time was not considered in the analysis. However, the age grouping represents a proxy to 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. Thus the analyses of mastic asphalt workers aged 40 years or more may be considered as approximately equal to analyses in which a 15–20 years' induction or latency time is required. Albeit a crude proxy, analysis by age was the only alternative to ignoring induction or latency time totally.

The study cohort included persons born in 1893–1960. It was therefore likely to be rather inhomogeneous as regards risk factors for diseases such as cancer, ischemic heart disease, and chronic bronchitis. In addition, some of the subjects may have been asphalt workers during World War II and may thus have been exposed to coal-tar pitch. For these reasons, the cohort of mastic asphalt workers was divided into the following three subcohorts by year of birth: 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). Because of the age structure of the subcohorts

(table 1) the analyses using the subcohorts were confined to the ages 40 to 64 years.

For the statistical evaluation, observed numbers of deaths beyond 100 were assumed to follow a normal distribution (Yate's correction employed), whereas, for smaller numbers, a Poisson distribution was assumed. A confidence interval for the standardized mortality ratio (SMR) was calculated by the method proposed by Miettinen (10) whenever the observed number of deaths exceeded 100. For smaller numbers "exact" Poisson limits were calculated (11).

Results

During the study period, the cohort of mastic asphalt workers experienced a mortality that was 1.63 times that of the general population (table 2).

Most of the asphalt workers' excess mortality was ascribed to cancer. For 62 decedents, cancer was notified as the underlying cause of death, 25 being lung cancers (expected number 8.65, $P < 0.01$) and 37 being nonpulmonary cancers (expected number 18.93, $P < 0.01$). Regarding the latter, two deaths were due to buccal cancer, three to nonpulmonary respiratory cancer, 19 to gastrointestinal cancer, seven to urogenital cancer, and six to malignant neoplasms with other or unspecified locations.

Regarding the 17 deaths from external causes, two were due to occupational accidents, and five were due to other types of accidents, nine to suicides, and one to homicide.

Besides the nine decedents for whom bronchitis, emphysema, or asthma was notified as the underlying cause of death, 11 decedents had bronchitis, emphysema or asthma notified as a contributory cause of death. Pulmonary heart disease or symptomatic heart disease was notified as a (underlying or contributory) cause of death in nine cases.

The mortality pattern for the men aged 40 years or more revealed an increased risk of lung cancer, nonpulmonary cancer, liver cirrhosis, and bronchitis, emphysema and asthma. Other natural causes of death, exclusive of circulatory diseases, also occurred in ex-

Table 1. Number of person-years at risk by age and subcohort.

Age (years)	Subcohort			Total
	I ^a	II ^b	III ^c	
15–39	—	75	2152	2227
40–54	265	742	1525	2532
55–64	793	703	9	1505
65–74	918	14	—	932
75–89	238	—	—	238
Total	2214	1534	3686	7434

^a 194 persons born in 1893–1919.

^b 129 persons born in 1920–1929.

^c 356 persons born in 1930–1960.

Table 2. Mortality of mastic asphalt workers in 1959–1986 by age and cause of death. (SMR = standardized mortality ratio, 95 % CI = 95 % confidence interval for the SMR)

Underlying cause of death	Age at death (years)							
	15–39		40–64		65–89		Total	
	SMR	95 % CI	SMR	95 % CI	SMR	95 % CI	SMR	95 % CI
All natural deaths	240	65–613	224	176–280	120	94–151	159	135–186
Cancer ^a	0	0–802	304	209–426	178	119–256	225	172–288
Circulatory diseases ^b	882 ^c	182–2579	139	80–210	81	53–118	105	79–139
Other natural deaths	115	3–640	290	179–443	145	84–232	196	139–258
External causes ^d	461	185–949	194	84–382	101	12–365	223	130–358
Total	345	172–617	220	176–273	119	93–149	163	141–190

^a International Classification of Diseases codes 140–205 in the seventh revision and codes 140–209 in the eighth revision.

^b International Classification of Diseases codes 330–334 and 400–468 in the seventh revision and codes 390–458 in the eighth revision.

^c Three deaths including the following diagnoses: heart block, acute myocardial infarction, cerebral hemorrhage.

^d International Classification of Diseases codes 800–999 in the seventh and the eighth revision.

Table 3. Natural deaths among mastic asphalt workers aged 40 to 89 years, 1959–1986. (O = observed number of deaths, SMR = standardized mortality ratio, 95 % CI = 95 % confidence interval for the SMR)

Underlying cause of death	O	SMR	95 % CI
Cancer ^a	62	229	175–293
Lung cancer ^b	25	290	188–429
Nonpulmonary cancer	37	200	141–276
Circulatory diseases ^c	48	100	74–132
Ischemic heart disease ^d	34	100	69–140
Other circulatory diseases	14	99	54–167
Other diseases	38	200	142–275
Bronchitis, emphysema and asthma ^e	9	207	95–393
Liver cirrhosis ^f	7	467	188–962
Other diseases	22	168	105–254
All natural deaths	148	157	134–185

^a International Classification of Diseases codes 140–205 in the seventh revision and codes 140–209 in the eighth revision.

^b International Classification of Diseases code 162 in the seventh and eighth revision.

^c International Classification of Diseases codes 330–334 and 400–468 in the seventh revision and codes 390–458 in the eighth revision.

^d International Classification of Diseases codes 420–422 in the seventh revision, and codes 410–414 in the eighth revision.

^e International Classification of Diseases codes 501–502, 241, and 527 in the seventh revision, and codes 490–493 in the eighth revision.

^f International Classification of Diseases code 581 in the seventh revision, and code 571 in the eighth revision.

cess (table 3). Analysis by year of birth revealed no consistent trend.

Discussion

This cohort study on mastic asphalt workers' mortality revealed an increased risk of lung cancer; non-pulmonary cancer; bronchitis, emphysema and asth-

ma; liver cirrhosis; and death due to external causes including occupational accidents. An increased mortality from natural causes, including cancer and bronchitis, was manifest for the workers aged 40 years or more, that is, after the lapse of approximately 20 years from first exposure.

The tasks carried out by the mastic asphalt workers comprised flooring and road paving. During the latter type of work, the risk of being run over by motor vehicles is high, and strict safety regulations are necessary. The chemical hazards associated with the handling of hot mastic asphalt mainly relate to the asphalt fumes. These fumes are locally irritating and possibly toxic to the respiratory epithelium. In addition, the fumes seem to possess carcinogenic properties. The observed high mortality from cancer, and from bronchitis, emphysema and asthma, may be explained by the mastic asphalt workers' inhalation of huge doses of asphalt fumes. Before drawing any such conclusion, however, one needs to consider the potential bias in the study.

First of all, mastic asphalt workers are subject to health-based selection. The physical work load is heavy, and only healthy, strong, and fit persons are capable of fulfilling the physical requirements for the handling of mastic asphalt. This group of workers constitutes a typical "healthy worker" group in which one would expect a very low prevalence of disabling diseases. Using the general population as a standard of reference for a healthy worker group leads to an underestimation of the possible adverse health effects of the exposure of interest (12). In particular, this situation applies to such diseases as chronic bronchitis, emphysema, and asthma, and to most circulatory diseases. Therefore, the calculated SMR values for these diseases are likely to be biased in a negative direction. Against this background it is remarkable that, for asphalt workers aged 40 years or more, the SMR values for circulatory diseases and for chronic bronchitis, emphysema, and asthma were observed to equal 100 and 207, respectively.

It has not been possible to control for potential differences in life-style (including smoking, drinking, and dietary habits) between the cohort of mastic asphalt workers and Danish men of the same age. In particular, control for smoking habits would have been advantageous because smoking is intimately associated with the risk of lung cancer, bronchitis, and emphysema. 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 (13). An inquiry in 1976 among mastic asphalt workers showed that 22 % were nonsmokers, 36 % medium smokers (ie, daily consumption 1–14 g of tobacco), and 43 % heavy smokers (ie, daily consumption ≥ 15 g of tobacco or more) (14). Similarly, a population survey in 1982 showed that, among men of the same age, 39 % were nonsmokers, 24 % medium smokers, and 38 % heavy smokers (15). With the use of data on mortality in relation to smoking among male British doctors (16), the reported smoking differences would approximately bring about an 18 % excess of lung cancer deaths, and a 21 % excess of deaths from chronic bronchitis in the group of mastic asphalt workers. However, the observed mortality from lung cancer and for bronchitis, emphysema, and asthma was two to three times greater than expected, a finding indicating that smoking was hardly likely to have been of major importance as a confounding factor in the present study.

Almost all of the mastic asphalt workers were city dwellers as opposed to only 40 % of the comparison population. For several causes of death, the mortality varies with the degree of urbanization. In Danish urban municipalities, a 5 % excess mortality from natural causes has been reported for men between the ages of 45 to 74 years, the excess mortality mainly being due to cancer (4 % increase), lung cancer in particular (10 % increase), circulatory diseases (3 % increase), and other diseases (11 % increase), including liver cirrhosis (137 % increase), and bronchitis, emphysema and asthma (20 % increase) (17).

The estimated confounding effect of urbanization and smoking habits may be used to adjust the calculated SMR values. (See the appendix.) When corrected for urbanization, the following SMR values were obtained for the age group ≥ 40 years: all cancer 220; lung cancer 264; nonpulmonary cancer 198; circulatory diseases 97; other diseases 180; bronchitis, emphysema, and asthma 172; liver cirrhosis 197; and all natural causes of death combined 150. When corrected independently for urbanization and smoking habits — a procedure that certainly represents an overcorrection because the two factors are positively correlated — the SMR for lung cancer is reduced to 224, whereas that for bronchitis, emphysema, and asthma is reduced to 142. Still, there remains a statistically significant ($P < 0.01$) excess in the mortality from cancer, lung cancer, nonpulmonary cancer, diseases other than can-

cer and circulatory diseases, and all natural causes of death combined.

Unfortunately, no data were available on the drinking habits of the mastic asphalt workers. The increased mortality from liver cirrhosis might indicate an above-average alcohol consumption. It cannot be excluded, however, that certain occupational factors have played an additional role.

Regarding cancer, the mortality data are consistent with the previously reported incidence data for the cohort in question (5). The observed cancer mortality pattern also corresponds well with that which has previously been reported for roofers (3). The occupational exposure of roofers resembles that of mastic asphalt workers, as both groups are exposed to fumes from heated bitumen. Furthermore, studies on urine mutagenicity (6), as well as animal assays (7), have indicated that bitumen fumes may exert a genotoxic effect.

The observation of an increased risk of bronchitis, emphysema, and asthma is in agreement with previous findings on roofers' mortality (3) and health surveys of asphalt workers (1–2). The workers' inhalation of toxic substances in the bitumen fume seems to present a natural explanation of the high risk of chronic obstructive respiratory disease among mastic asphalt workers.

Acknowledgments

This work was supported by grant number 71.18.3/84 from the Research Fund of the Danish Medical Society.

I wish to thank Dr FB Behrendt for kindly providing identification data from the employment lists of three mastic asphalt plants, and Ms MF Poulsen for her assistance in the preparation of the manuscript.

References

1. Baylor CH, Weaver NK. A health survey of petroleum asphalt workers. *Arch Environ Health* 1968;17:210–4.
2. Nyqvist B. Luftvägssymptom hos asfaltarbetare — ytterligare en yrkesbronkit? [Respiratory tract symptoms in asphalt workers — another occupational bronchitis?]. *Läkartidningen* 1978;12:1173–5.
3. Hammond EC, Selikoff IJ, Lawther PL, Seidman H. Inhalation of benzopyrene and cancer in man. *Ann NY Acad Sci* 1976;271:116–24.
4. Hansen ES. Cancer mortality in the asphalt industry — a ten-year follow-up of an occupational cohort. *Br J Ind Med* 1989;46:582–5.
5. Hansen ES. Cancer incidence in an occupational cohort exposed to bitumen fumes. *Scand J Work Environ Health* 1989;15:101–5.
6. Pasquini R, Monarca S, Sforzolini GS, Savino A, Bauleo FA, Angeli G. Urinary excretion of mutagens, thioethers and D-glucaric acid in workers exposed to bitumen fumes. *Int Arch Occup Environ Health* 1989;61:335–40.
7. Thayer PS, Menzies KT, von Thuna PC. Roofing asphalts, pitch and UVL carcinogenesis — report. US Department of Health and Human Services. Springfield, VA: National Technical Information Service, 1983.

- (PB83—134767).
8. Rietz B. Luft forurening ved asfaltarbejde [Air pollution by handling of asphalt]. Copenhagen: Danish Institute of Occupational Health, 1979.
 9. International Agency for Research on Cancer. Polynuclear aromatic compounds; part 4, bitumens, coaltars and derived products, shale-oils and soots. Lyon: International Agency for Research on Cancer, 1985. (IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans; vol 35.)
 10. Miettinen OS. Simple interval-estimation of risk ratio. *Am J Epidemiol* 1974;100:515—6.
 11. Ciba-Geigy. Documenta Geigy: mathematics and statistics. Copenhagen: Ciba-Geigy, 1975:107.
 12. McMichael AJ. Standardized mortality ratios and the "healthy worker effect": scratching beneath the surface. *J Occup Med* 1976;18(3):165—8.
 13. Axelson O. Aspects on confounding in occupational health epidemiology. *Scand J Work Environ Health* 1978;4:85—9.
 14. Hasle P, Hegelund G, Høgh K et al. Asfaltarbejde — en arbejdsmiljøundersøgelse [Handling of asphalt — a study on working environment]. Roskilde: Roskilde Universitetsforlag, 1977.
 15. Madsen F, Hedegaard L, Sjø A. Lugesymptomer og lungefunktioner [Respiratory symptoms and lung functions]. In: Hagerup L, ed. Sygdom og sundhed — befolkningsundersøgelserne i Glostrup [Illness and health — the population surveys in Glostrup]. Glostrup: Glostrupgruppen, 1987:57—75.
 16. Doll R, Peto R. Mortality in relation to smoking: 20 years' observations on male British doctors. *Br Med J* 1976;2:1525—36.
 17. Danmarks Statistik. Regionale dødelighedsforskelle i Danmark 1971—79 [Regional variation in mortality in Denmark 1971—79]. Copenhagen: Danmarks Statistik, 1985. (Statistiske undersøgelser nr 39.)

Appendix

Correcting for confounding

The study group of mastic asphalt workers differed from the total population with respect to smoking habits and geographic distribution. These differences are likely to have brought about mortality differences unrelated to the occupational exposure in question. The magnitude of the resulting confounding effect (from smoking and urbanization) has been estimated from previously published data. The way in which this confounding effect may be removed from the actual data is illustrated for lung cancer mortality among mastic asphalt workers aged 40 to 89 years in table A1. The correction factor for smoking has been derived from the data shown in table A2.

Table A1. Observed and expected lung cancer mortality among mastic asphalt workers aged 40—89 years, inclusive of correction for confounding from smoking and urbanization. (SMR = standardized mortality ratio, 95 % CI = 95 % confidence interval for the SMR)

	Observed numbers	Correction factor for expected numbers	SMR	95 % CI
No correction	25	1.00	290	188—429
Correction for urbanization	25	1.10 ^a	264	171—390
Correction for smoking	25	1.18 ^b	246	159—363
Correction for urbanization and smoking	25	1.10 × 1.18	224	145—330

^a From reference 17.

^b See table A2.

Table A2. Estimation of smoking-related differences in lung cancer mortality between mastic asphalt workers and the comparison population.

	Lung cancer mortality rate ^a	Distribution of population ^b		Expected lung cancer mortality due to smoking ^c	
		Asphalt workers	Comparison population	Asphalt workers	Comparison population
Nonsmokers	R_0	0.22	0.39	0.22 R_0	0.39 R_0
Medium smokers	7.8 R_0	0.36	0.24	2.81 R_0	1.87 R_0
Heavy smokers	18.9 R_0	0.43	0.38	8.13 R_0	7.18 R_0
Total		1.00	1.00	11.16 R_0	9.44 R_0

^a From reference 16.

^b From references 14 and 15.

^c Estimated rate ratio due to smoking habit differences: $RR_{\text{smoking}} = 11.16 R_0 / 9.44 R_0 = 1.18$.

Received for publication: 3 May 1990