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Exposure, lung function decline and systemic inflammatory response in asphalt workers

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Objectives The aim of this study was to determine the association between exposures in asphalt work and changes in lung function, blood concentrations of interleukin-6 (IL-6), micro-C-reactive protein, and fibrinogen among asphalt workers during a work season.

Methods Blood samples from all asphalt workers (N=140) in Norway's largest road construction and maintenance company were taken in April–May 2005 and again in September–October 2005. Spirometric tests of the asphalt workers and a reference group (heavy construction workers, N=126) were carried out before the asphalt season, and the asphalt workers were tested again at the end of the season. Exposure to total dust, oil mist, polycyclic aromatic hydrocarbons, and gases was measured by personal samplers during the asphalt season.

Results The asphalt workers had a significantly a lower forced expiratory volume in 1 second (FEV₁) and forced expiratory flow rate of 50% of the forced vital capacity than the reference group at the beginning of the season. The asphalt workers were divided according to their exposure into two groups, asphalt pavers (N=81) and asphalt plant operators and truck drivers (N=54). The screedmen, a group of the asphalt pavers, had a statistically significant lower FVC and FEV₁ after one season of asphalt work than all of the other asphalt workers (P<0.05). The mean plasma concentration of IL-6 increased among the asphalt pavers from 1.55 pg/ml before the season to 2.67 pg/ml at the season's end (P=0.04, adjusted for current smoking).

Conclusions Exposure in asphalt paving may enhance the risk of lung function decline.

Key terms interleukin-6, oil mist.

Asphalt paving is used for road construction and road maintenance and is carried out all over the world. In Norway, 5 million tons of asphalt is laid every year, and about 3000 workers are involved in the production and application of various asphalt mixes. The asphalt season in Norway is short, lasting from April to October in most of the country.

Air pollution is a public health issue that constitutes a health problem for workers in the heavy construction industry and is also a problem for workers involved in asphalt paving. Air pollution may affect lung function and has also been shown to affect the immune system (1–4). Asphalt workers are exposed to bitumen fumes, oil mist and vapor, polycyclic aromatic hydrocarbons (PAH), and vehicle exhaust (nitrogen dioxide, carbon monoxide) (5). Studies have reported increased mortality from respiratory diseases among asphalt workers. A

standardized mortality ratio (SMR) of 207 [95% confidence interval (95% CI) 95–393] for bronchitis, asthma, and emphysema was reported in 1991 for mastic asphalt workers (6). The asphalt worker mortality study involving 29 820 European asphalt workers reported that, relative to construction workers, asphalt workers had a risk ratio (RR) of 1.36 (95% CI 1.06–1.74) of dying from a nonmalignant respiratory disease (7). Cross-sectional studies have shown a greater risk for respiratory symptoms among asphalt workers than for road maintenance workers (8). Adverse respiratory effects and systemic inflammatory responses in workers exposed to dusts and fumes have recently been reported for several different occupations, for example, welders (9), firefighters (10, 11) salt miners (12), and tunnel workers (13–15).

In an earlier cross-sectional survey, we found that asphalt workers had more respiratory symptoms and

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airflow limitation than heavy construction workers (16). However, exposure measurements were not carried out, and we were not able to point out which exposures or worktasks were of importance. Neither did we investigate signs of systemic inflammation. Our present report presents the results of a parallel and larger cohort. We hypothesized (i) that workers exposed to air pollutants during asphalt work would have a higher risk of accelerated decline in lung function than other road construction workers, (ii) that exposure to bitumen fumes would be associated with signs of inflammation as indicated by inflammatory markers, and (iii) that these findings would be related to job tasks and differences in exposure to particles or gases during the asphalt season. We also hypothesized that smoking status would modify the association (17).

Study population and methods

Study population

All of the asphalt workers (paver operators, screedmen, roller drivers, asphalt strippers, asphalt plant operators, and asphalt lorry drivers) (N=140 males) employed in Norway's largest road construction and road maintenance company were studied with lung function tests and blood samples in April–May 2005, before the asphalt season started. As a reference group, 126 male nonasphalt heavy construction workers, mainly machine operators (hereafter referred to as the reference group) belonging to the same company were included in the before-season lung function study. Because of economic constraints, we did not collect blood samples from the reference group. Demographic data on the study participants are given in table 1.

The group of asphalt workers was studied again with lung function tests and blood samples just before the asphalt season ended, September–October 2005. The asphalt workers were divided according to their exposure into two groups, asphalt pavers (paver operator, screedman, roller driver, and asphalt stripper) (N=81) and asphalt plant operators and asphalt truck drivers (N=54), who then served as an internal comparison group for changes in the inflammatory markers. The

study was approved by the National Data Inspectorate and the Regional Medical Board of Ethics.

Description of asphalt work

Asphalt consists of bitumen, as a binder, mixed with crushed stone. In road paving the bitumen content is usually 4–5%. Bitumen is the residue of the distillation of selected petroleum crude oils. The type of bitumen and the size of the gravel vary with the properties requested for the road surface. Filler or fibers can also be added to modify the properties of the asphalt, and small amounts of aliphatic amines are used to improve the binding between the bitumen and the stone material.

Asphalt is produced by heating and drying the gravel and mixing the hot bitumen with it. The asphalt is transported to the paving site by trucks and emptied onto the front of the paving machine. It passes underneath the machine and is spread to the desired width by the screed. Two screedmen control the asphalt discharge through the screed, and they fix the edges of the asphalt on the road manually. Subsequently, a roller compresses the asphalt.

Exposure assessment

Sampling strategy. Measurements were carried out between April and October 2005 to assess exposure in modern asphalt work. A random sample of workers representing different worktasks was asked to participate in the exposure assessment. Participation was voluntary, but all of the selected workers decided to participate.

Exposure to dust and gases was determined by means of personal sampling, and two or more agents were measured simultaneously for each person for at least 2 days. The sampling duration was 7–8 hours because of the limited battery capacity of the sampling equipment. The sampling time was considered representative for the whole workshift because the same tasks were repeated all the time.

Sampling methods and analyses. Total dust and particulate PAH were collected on glass fiber filters (Whatman International Ltd, Maistone, UK), fitted in 37-mm closed-faced aerosol filter cassettes (Millipore Corporation, Bedford, MA, USA). Gaseous PAH were collected on tubes filled with the adsorbent XAD-2 (SKC,

Table 1. Characteristics of the study population stratified by job category. (AM = arithmetic mean, SD = standard deviation, BMI = body mass index)

Group	Age (years)		Height (cm)		BMI		Current smoking		Pack-years		Self-reported allergy	
	AM	SD	AM	SD	AM	SD	N	%	AM	SD	N	%
Asphalt workers (N=140)	44.6	9.2	179	6.0	27.2	3.6	35	25	10.9	12.0	34	23
Reference group (N=126)	46.0	9.5	179	6.2	26.6	3.4	29	23	9.9	11.9	34	24

Blandford Forum, Dorset, UK). Filters and tubes for the collection of particulate and gaseous PAH were mounted in series during the sampling. The sampling flow rate was 2 l/min. The filter and adsorbent were extracted by the addition of dichloromethane mixed with internal standards [naphthalene-D8, phenanthrene-D10, fluoranthene-D10, benzo(a)pyrene-D12].

The particle mass was measured by a microbalance (Mettler Toledo AT261, Columbus, OH, USA), with a detection limit of 0.031 mg/m³ based on 8-hour sampling. Total PAH (16 EPA) were measured by gas chromatography with a mass selective detector. The detection limits of the PAH were 0.005–0.010 µg/m³, depending on the specific component, based on 8-hour sampling at a flow rate of 2 l/min.

Oil mist was collected on glass fiber filters (Whatman), and oil vapor was collected on tubes containing XAD-2 (SKC). Filters and tubes for the collection of oil mist and oil vapor were mounted in series during the sampling. The sampling flow rate was 2 l/min. Oil mist was measured with a Fourier transform (FT-IR) spectrophotometer (PE-1600, Perkin-Elmer, MA, USA) and oil vapor was measured by chromatography with a flame ionization detector. The detection limit was 10 µg and 5 µg for the oil mist and oil vapor, respectively.

Respirable dust was collected on 37-mm cellulose acetate filters with a pore size of 0.8 µm using a cyclone separator (SKC) at a sampling flow rate of 2.2 l/min. The particle mass was measured gravimetrically (with a detection limit of 0.03 mg), and the alpha-quartz content in the respirable dust sample was measured with the FT-IR PE-1600 spectrophotometer. The detection limit was 1.7 µg/m³ if it is assumed that the sampling volume was 1 m³.

The carbon monoxide and nitrogen dioxide concentrations were measured with direct-reading electrochemical sensors with a data-logging facility built into the instrument (type PAC III, Dräger Aktiengesellschaft, Lübeck, Germany). An averaging period of one reading every 2 minutes was selected. The detection limit of the carbon monoxide and nitrogen dioxide measurements was 2 ppm and 0.2 ppm, respectively.

Assessment of respiratory health effects

Spirometric tests. The lung function tests were performed for both the asphalt workers and the referents between 0700 and 1000 in the morning shortly before the asphalt season started. Shortly before the asphalt season ended, the lung function tests were performed again for the asphalt workers at the same time of day. The reference group was not reexamined due to study economic constraints. The participants who reported physician-diagnosed asthma were tested, but they were excluded from the statistical analyses. All of the participants had

to be free of respiratory infections for 3 weeks prior to the testing.

Spirometric measurements were performed in the sitting position with a Spirare SPS310 spirometer (Diagnostica, Oslo, Norway), using bi-directional ultrasound transit-time measurements. The spirometer was operated by the same three trained technicians. The spirometer was controlled by a 3-liter syringe. The participant wore a nose clip. The measurements were performed in accordance with the guidelines recommended by the American Thoracic Society (18). Each person performed at least three preferably identical forced vital capacity curves (ie, within a variation of 50 ml or a maximum of 3%). The best independent values were selected for the statistical analysis.

The following variables were recorded: forced vital capacity (FVC), forced expiratory volume in 1 second (FEV₁), and forced expiratory flow rate of 50% (FEF₅₀) of the FVC. The lung function variables were expressed in absolute values and as the percentage of the predicted value according to the reference values of the European Coal and Steel Community (19).

Questionnaire. Information on age and smoking status was obtained from a general questionnaire used in an earlier cross-sectional study (16). The questions included whether the person had allergies or had been diagnosed by a physician as having asthma. The workers answered the questionnaire before the first examination. They were classified as never smokers, former smokers, and current smokers. Former smokers were those who had stopped smoking more than 12 months earlier. For the current and former smokers, the quantitative effect of smoking was measured in pack-years. Pack-years were calculated by multiplying the duration of smoking (in years) by the average number of cigarettes smoked daily divided by 20 (table 1).

A questionnaire was also distributed at the point of follow up. The asphalt workers were asked about job tasks they had had during the season and were divided into groups according to the tasks they had most often executed. The job tasks were asphalt plant operator, asphalt truck driver, paver operator, screedman, roller driver, and asphalt stripper.

Assessment of inflammatory responses

Blood samples were taken for the analysis of interleukin-6 (IL-6), fibrinogen, C-reactive protein (micro-CRP), and cholesterol (total and high-density lipoprotein). The workers had been told not to smoke, drink, or eat after midnight before the examination. The blood samples were taken between 0700 and 1000 in the morning shortly before the asphalt season started. Shortly before the asphalt season ended, new blood samples were taken

at the same time of day. The blood plasma samples, anticoagulated with citrate, ethylenediaminetetraacetic acid (EDTA), and heparin, respectively, were kept on ice until 60 minutes before they were centrifuged at 2000 *g* for 15 minutes. The blood serum samples were kept at room temperature for coagulation from 60–120 minutes before they were centrifuged at 1300 *g* for 15 minutes. Plasma and serum were then frozen in cryotubes on dry ice and transported to the Ulleval University Hospital, where they were stored at -70°C .

EDTA plasma samples were analyzed for the concentration of IL-6 2 months after the asphalt season ended. The before- and after-season samples were analyzed at the same time at the Centre for Clinical Research, Ulleval University Hospital of Oslo, Norway. The IL-6 was measured with the commercial enzyme-linked immunosorbent assay (ELISA) kit Quantikine HS from R & D systems, Abingdon, UK. The sensitivity of the assay is 0.039 pg/ml. The other blood analyses were performed at the Department of Clinical Chemistry at the Ulleval University Hospital. The fibrinogen concentration in citrate plasma was measured according to Clauss (20) using reagents from bio Mericux (Durham, NC, USA) as adapted to their Stago analyzer. High-sensitivity micro-CRP was determined by an immunoturbidometric method (Roche, Basel, Switzerland) using a Hitachi 917 analyzer (Roche). The sensitivity of the assay is 0.03 mg/ml.

Data analysis

With the use of cumulative probability plots, the exposure data were found to be best described by log-normal distributions and were ln-transformed before further statistical analyses were undertaken. The measured exposure values were used without further adjustment as they were regarded as representative of the whole workshift. Standard measures of central tendency and distributions [arithmetic (AM) and geometric (GM) means and geometric standard deviations (GSD)] were calculated. Kruskal-Wallis tests were used to evaluate

the differences in the exposure levels between the job task categories. Mann-Whitney tests were used for two-group comparisons for total dust and total PAH and IL-6. The relationship between job category and the changes in IL-6, fibrinogen, and micro-CRP was tested with a one-way analysis of variance (ANOVA).

The lung function variables were presented as percentages of the predicted values. The relationship between the lung function variables and the covariates occupational group, smoking status (pack-years), and body mass index (BMI) was investigated with an ANOVA.

The change in lung function among the asphalt workers over the asphalt season (change in FVC, change in FEV₁, and change in FEF₅₀), was defined as the difference between lung function at the start of the asphalt season and the lung function at the end of the season. Individual lung function changes were evaluated by comparing the lung function before and at the end of the period using paired t-tests. The relationship between the changes in lung function and job task category was tested using a one-way ANOVA with Bonferroni posthoc tests. Potential confounders (age, height, smoking status, and allergy) were evaluated for their effect using a multiple linear regression.

The statistical analyses were carried out in SPSS (version 13.0, SPSS Inc, Chicago, IL, USA).

Results

Exposure

A total of 42 asphalt workers carried personal samplers in the exposure study, and most of the workers (90%) were monitored on more than one occasion.

The asphalt strippers had a significantly higher exposure to total dust than the other asphalt workers ($P<0.001$) (table 2). The paver operators, screedmen, and roller drivers had a significantly higher exposure to total PAH than the truck drivers, asphalt strippers, and plant operators (table 2).

Table 2. Personal exposure levels for each job category by agent (PAH = polycyclic aromatic hydrocarbons, N = number of measurements, GM=geometric mean, GSD=geometric standard deviation, NM=not measured)

Job task	Total dust (mg/m ³)			Total PAH (µg/m ³)			Oil mist (mg/m ³)		
	N	GM	GSD	N	GM	GSD	N	GM	GSD
Paver operator	16	0.3	1.9	12	1.8 ^a	1.9	7	0.23	3.4
Screedman	32	0.3	2.5	29	1.6 ^a	2.2	9	0.09	2.3
Roller driver	8	0.4	2.7	7	1.3 ^a	4.3		NM	
Asphalt stripper	9	2.4 ^b	1.5	9	0.5	1.8	4	0.19	2.6
Plant operator	9	0.9	1.8	12	0.5	1.7		NM	
Lorry driver	10	0.1	2.4	6	0.3	1.4		NM	

^a Paver operators, screedmen, and roller drivers versus other workers, Mann-Whitney test, $P<0.001$; see the text for details.

^b Asphalt strippers versus other workers, Mann-Whitney test, $P<0.001$; see the text for details.

Although the geometric mean exposure to nitrogen dioxide was less than 1 ppm for all of the groups, the paving group may have been exposed to higher levels when paving asphalt in tunnels (maximum 3.4 ppm measured). Although the geometric mean exposure to oil mist was $<0.3 \text{ mg/m}^3$, the exposure to oil mist can also be significant on some occasions. Oil mist was measured on one occasion as 1.7 mg/m^3 in an asphalt paving machine without a cabin. Exposure to oil mist correlated with the exposure to total PAH (Spearman's rho 0.62, $P=0.04$).

Only a few analyses of respirable dust, volatile organic compounds, and carbon monoxide were done, due to previous analyses that showed low concentrations.

Clinical and laboratory findings at the start of the season

The asphalt workers and the reference group were comparable with respect to age, height, BMI, smoking habits, and the occurrence of self-reported allergy (table 1). Eight (7.5%) of the asphalt workers and nine (5.7%) of the reference group reported physician-diagnosed asthma.

FEV_1 and FEF_{50} were significantly lower among the asphalt workers than in the reference group (table 3). There appeared to be no statistically significant dif-

Table 3. Mean (SE) lung function of the asphalt workers and a reference group before the asphalt paving season (SE = standard error of the mean, FVC = forced vital capacity, FEV_1 = forced expiratory volume in 1 second, FEF_{50} = forced expired flow of 50% of FVC)

Group	FVC (% of predicted)		FEV_1 (% of predicted)		FEF_{50} (% of predicted)	
	Mean	SE	Mean	SE	Mean	SE
Asphalt workers (N=140)	97.4	1.0	92.6 ^a	1.1	84.8 ^b	2.6
Reference group (N=126)	100.4	1.1	96.9	1.2	92.9	3.3

^a $P=0.01$.

^b $P=0.03$, with adjustment for pack-years and body mass index.

ferences in FVC between the asphalt workers and the referents, even though the asphalt workers tended to have lower lung function parameters. The lung function decline did not correlate with the duration of employment in asphalt work.

Within the asphalt worker group, the roller drivers had a statistically significant lower FVC and FEV_1 than the rest of the group [FVC (% of predicted) 92.2 (SE 2.9) versus 98.9 (SE 1.2), $P=0.048$, and FEV_1 (% of predicted) 89.1 (SE 2.3) versus 95.0 (SE 1.3), $P=0.04$]. Eight of the twelve roller drivers were former screedmen.

The results of the IL-6 measurements are shown in table 4. According to their exposure, the asphalt workers were divided into two groups, one comprised of the asphalt pavers (N=81) and the other forming a control group of asphalt plant operators and asphalt truck drivers (N=54). There were no statistically significant differences between the asphalt worker groups with respect to the before-season values. For the entire study group, the smokers had statistically significant higher IL-6 values than the nonsmokers before the season began [GM 2.11 (GSD 1.89) ng/l versus GM 1.39 (GSD 2.06) ng/l, $P=0.026$].

Clinical and laboratory findings at the end of the season

Eight asphalt workers were lost to the follow-up, three of whom were deer hunting during the follow-up period and five of whom had left the job. In addition, eight asphalt workers used asthma medicine and were excluded from the analyses, leaving 124 to be followed up.

The results of the lung function changes among the asphalt workers are shown in table 5. For the entire follow-up group, the mean values of the FVC, FEV_1 , and FEF_{50} at the start of the survey were 98%, 94%, and 87% of the predicted values, respectively. There were no statistically significant differences between the job task groups with respect to the before-season values. The screedmen had a statistically significant decrease in FVC and FEV_1 after a season of asphalt work when compared with all of the other asphalt workers ($P<0.05$).

Table 4. interleukin-6 (IL-6), fibrinogen, and C-reactive protein (micro-CRP) of the asphalt pavers and a control group of plant operators and truck drivers and changes after the asphalt season. (N = number of measurements, GM = geometric mean, GSD = geometric standard deviation)

Job task	IL-6 ng/l before season		Change in IL-6 ng/l		Fibrinogen before season		Change in Fibrinogen		Micro-CRP before season		Change in Micro-CRP	
	GM	GSD	GM	GSD	GM	GSD	GM	GSD	GM	GSD	GM	GSD
Asphalt pavers (N=76)	1.55	2.0	1.12 ^a	1.75	3.29	1.2	-0.04	0.4	0.90	2.5	0.12	2.1
Plant operators and truck drivers (N=48)	1.76	2.1	-0.89	1.91	3.32	1.2	-0.06	0.6	1.32	2.4	-0.29	0.9

^a Analysis of variance, $P=0.04$, adjusted for daily smoking during the season.

Table 5. Lung function of the asphalt workers of each job task category and the lung function changes after the asphalt season. (FVC = forced vital capacity, FEV₁ = forced expiratory volume in one second, FEF₅₀ = forced expired flow of 50% of FVC)

Job task	FVC (l) before season		Change in FVC (l)		FEV ₁ (l) before season		Change in FEV ₁ (l)		FEF ₅₀ (l/s) before season		Change in FEF ₅₀ (l/s)	
	AM	SD	AM	SD	AM	SD	AM	SD	AM	SD	AM	SD
Paver operator (N=16)	4.9	0.5	-0.05	0.3	3.9	0.5	-0.04	0.2	4.9	1.9	-0.13	0.7
Screedman (N=42)	4.8	0.8	0.13 ^a	0.4	3.7	0.6	0.09 ^{a,b}	0.3	4.6	1.5	0.34	1.3
Roller driver (N=12)	4.3	0.7	-0.05	0.3	3.4	0.4	0.04	0.1	4.4	1.4	0.28	0.6
Asphalt stripper (N=6)	4.9	1.4	-0.21	0.3	3.9	1.5	0.04	0.2	4.9	2.7	0.87	1.1
Plant operator (N=30)	4.6	0.8	0.01	0.3	3.5	0.6	0.02	0.2	4.2	1.6	0.21	0.8
Lorry driver (N=18)	4.5	0.8	0.10	0.3	3.5	0.6	0.04	0.2	4.2	1.5	-0.11	1.2

^a Paired sample t-test, P<0.05.^b ANOVA, P<0.05, screedmen versus all other asphalt workers, adjusted for smoking.

There were no significant differences between the other job task groups (table 5).

Among the screedmen the nonsmokers showed an average decrease in FEV₁ of 51 ml versus the smokers, who had a decrease of 146 ml during the asphalt season. The difference between the nonsmokers and smokers was not significant.

The asphalt pavers had a statistically significant increase in IL-6 during the asphalt season when they were compared with the asphalt plant operators and truck drivers after adjustment for current smoking (table 4).

Only nonsmokers showed a significant increase in IL-6 during the asphalt season [change in IL-6 1.18 (GSD 1.76) ng/l]. The increase in IL-6 was not associated with a decrease in lung function. The plasma fibrinogen and serum micro-CRP levels did not increase significantly among the workers over the season, but were correlated with the increase in the IL-6 levels [Spearman's rho 0.47 (P=0.0001) for micro-CRP and 0.42 (P=0.001) for fibrinogen].

Discussion

The group of asphalt workers in this study had a significantly lower FEV₁ (% of predicted) and FEF₅₀ (% of predicted) than the reference group at the start of the asphalt season. The FVC and FEV₁ of the screedmen decreased an average of 2.7% and 2.4%, respectively, during the asphalt season. This result supports the suggestion from other studies (6, 7, 16) that asphalt paving work may be a contributing factor to the development of obstructive lung diseases. Accelerated loss of lung function may be linked to the risk of developing lung disease, including chronic obstructive pulmonary disease (21). There is also an association between a rapid rate of decline in lung function and coronary heart disease (22). Our findings provide new evidence that employment in modern asphalt paving may lead to a decrease in lung function among screedmen during the asphalt paving season.

It is not clear, however, which agent(s) cause the observed lung function changes because the exposure of the paving team, also that of the screedmen, was low to moderate as compared with that of the Norwegian occupational exposure limits (6–48% of the limits for total dust). There was a significant difference in the exposure to total PAH between the asphalt paving team (screedmen, paver operators and roller drivers) and the other asphalt workers (truck drivers and asphalt plant operators). The personal measurements of the total PAH exposure of the study participants showed, however, low levels when compared with the occupational exposure limits. Oil mist was only measured among the screedmen, paver operators, and asphalt strippers, but its level correlated with that of total PAH. Oil mist was the only exposure that, in one situation, was measured above the occupational exposure limit, and otherwise one-fourth of the measurements showed levels above one-third of the occupational exposure limit. The method employed to measure oil mist, 37-mm, closed-faced, three-part Millipore cassettes, has some limitations. The Millipore cassettes are known to somewhat overestimate the coarse fraction (aerodynamic diameter >20 µm) of the thoracic fraction. However, for sampling the thoracic fraction of particles, Millipore cassettes are efficient, even though some coarse particles not belonging to the thoracic fraction are also sampled (23). Oil mist has been reported to cause airway obstruction (24).

The exposure of the screedmen did not differ from that of the rest of the asphalt paving team for total dust and total PAH. For oil mist we did not carry out sufficient measurements to determine whether or not the exposure differed. Screedmen's work is known to be physically demanding, and increased respiration (time volume), inhaling more air pollutants per time unit, may be a possible explanation for these findings. The paver operators and the roller drivers were seated within a cabin during almost all of the paving operations. The company we studied was one of few that provided cabins on paving machines and rollers for their operators. This

use may offer another explanation for the difference in lung function between the screedmen and the machine operators.

The asphalt strippers had significantly higher exposure to total dust than the rest of the asphalt workers. We found that the strippers had a nonsignificant fall in FEV₁ through the season. However, there were only six asphalt strippers, and we cannot draw any specific conclusions from this finding. Of the six, five were nonsmokers.

Smoking is an important factor in the development of airway obstruction, and the screedmen who smoked showed a greater decrease in FEV₁ (146 ml) during the asphalt season of 6 months than the screedmen who did not smoke (51 ml). The annual decrease determined for an unexposed nonsmoker has been estimated to be 27 ml (25). Smokers have been shown to have an excess yearly decrease of 10 ml (26). The excess decline that occurred among the nonsmoking screedmen was probably related to work exposure. The much accelerated decline that occurred among the smoking screedmen showed that smoking combined with the screedmen's exposure is especially problematic. With rapid rates of annual decline in FEV₁, some degree of respiratory impairment could occur within 10 to 15 years.

The decline of 90 ml in FEV₁ in the group of screedmen during the asphalt season was one-third of the decrease found in a study of tunnel workers (270 ml) after 2 weeks of exposure to nitrogen dioxide and dust from blasting (14). Among the tunnel workers, the lung function values had returned to the baseline levels for FVC, FEV₁, and FEF₂₅₋₇₅ after 10 days off work. This may also be the situation for the screedmen. Asphalt workers from this company usually change worktasks, doing snow removal during the winter season, and have very little harmful exposure during the winter. However, the fact that we found reduced lung function among the asphalt workers at the start of the asphalt season may mean that repeated exposures in asphalt work may make lung function loss chronic.

An increase in IL-6 occurred among the asphalt pavers during the season, and this change may have been due to exposure in asphalt paving work. IL-6 is a pro-inflammatory mediator produced locally in the lung, but it may also be produced in muscle and increase during exercise (27, 28).

The observed relationship between exposure in asphalt work and airway dysfunction may have been biased by several factors. People who choose to enter dusty professions have better lung function than those who do not (primary health selection). To evade bias from healthy worker selection, we chose a reference group that was comparable with the study group in respect to education, socioeconomic status, and selection for employment. Thus, if primary health selection had occurred, it would have occurred in both the study

and reference groups. Therefore, we believe that the observed differences in airflow limitation between the asphalt workers and the reference group may reflect differences in occupational exposure.

A drawback of the study design was, of course, that the reference group was only examined once due to study constraints; the asphalt workers were examined both before and after the asphalt season. The reference group was, as the asphalt worker group, spread all over Norway. It was decided that the expense of seeking out the reference group for another spirometric test was too high.

The lower lung function among the asphalt workers at the start of the season did not correlate with the duration of employment in asphalt work. We think, however, that the cross-sectional design of the study before the asphalt paving season may have led to an underestimation of the effects of exposure because sensitive employees may have left the occupation or changed tasks within the occupation. The workforce studied in this cross-sectional survey can therefore be regarded as the "survivors" of the trade. The fact that the roller drivers had the lowest FVC and FEV₁ points in this direction. Eight of the twelve roller drivers were former screedmen. Screedmen and paver operators often rake the asphalt or do hand paving, which may be physically very demanding. Working as a roller driver is less demanding.

The exposure levels measured in the personal sampling in our study highly agreed with the estimates made in previous studies of asphalt workers (5, 26). Oil mist, however, which may be a major problem in asphalt paving, has seldom been measured or mentioned in previous studies. In our study, exposure to oil mist was only measured among the pavers. The oil mist values correlated, however, with the exposure to total PAH, which was significantly higher among the pavers than among the controls. More should be done to reduce exposure in asphalt paving. Possible technological solutions to reduce exposure are dependent on more exact knowledge of which agent(s) cause the lung function decline. On the basis of our results, we suspect oil mist and are planning to measure oil mist in different phases of asphalt paving during the coming asphalt season. We also want to investigate the workers' exposure to ultrafine particles. Epidemiologic studies have shown a strong association between ultrafine particles in air pollution and adverse pulmonary and cardiovascular health effects (28). Ultrafine particles have also been reported to induce an increase in inflammatory mediators, among them IL-6, in alveolar macrophage-epithelial cell cultures (30).

In conclusion, exposure during asphalt paving may enhance the risk of lung function decline among screedmen. This finding suggests that technological solutions are necessary to reduce exposure during asphalt paving.

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