Int Arch Occup Environ Health (2017) 90:63–71
DOI 10.1007/s00420-016-1173-z

ORIGINAL ARTICLE

Lung function in asphalt pavers: a longitudinal study

Bente Ulvestad[1] · Britt Grethe Randem[2] · Øivind Skare[1] · Trond Mogens Aaløkken[3] · Georg Karl Myranek[3] · Karine Elihn[4] · May Brit Lund[5,6]

Received: 7 March 2016 / Accepted: 2 October 2016 / Published online: 8 October 2016

Abstract
Purpose To study longitudinal changes in lung function in asphalt pavers and a reference group of road maintenance workers, and to detect possible signs of lung disease by high-resolution computed tomography (HRCT) scans.
Methods Seventy-five asphalt pavers and 71 road maintenance workers were followed up with questionnaires and measurements of lung function. Not every worker was tested every year, but most of them had four or more measurement points. The 75 asphalt pavers were also invited to have HRCT scans of the lungs at the end of the follow-up period.
Results Mean annual decline in forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV_1) of the asphalt pavers was 58 and 35 ml, respectively. Adjusted for age at baseline, packyears of smoking and BMI, the asphalt pavers had a significant excess annual decline in FVC and FEV_1 compared to the references. The screedmen, the most exposed group of the asphalt pavers, showed a significantly larger decline in FVC than the other asphalt pavers ($P = 0.029$). Fine intralobular fibrosis without evident cysts was identified with HRCT in three subjects (4 %).
Conclusion We conclude that our findings may indicate an excess annual decline in FVC and FEV_1 related to exposure to asphalt fumes. The screedmen, who carry out their work behind and close to the paving machine, had the largest decline in lung function. The finding of adverse pulmonary effects in asphalt pavers calls for better technological solutions to prevent exposure.

Keywords Asphalt fumes · Oil mist · Spirometry · HRCT

Introduction

Asphalt pavers are exposed to air pollution from asphalt fumes, diesel exhaust and road dust. Such exposure may adversely affect lung function. Asphalt fumes generate from the hot asphalt mix. Petroleum hydrocarbons in the crude oil/bitumen, used as a binder in asphalt, evaporate as a gas that condenses into fine particles as it cools. The chemical composition of the aerosol varies with the type of crude oil and the type of asphalt and is dependent on temperature. The aerosol contains volatile organic compounds, polycyclic aromatic compounds and naphthalene, particles, nitrogen oxides and sulfur-containing compounds. Naphthalene is a bicyclic aromatic hydrocarbon. Asphalt fumes consist partly of oil mist. In a previous study, exposure to oil mist in asphalt paving on occasion exceeded Scandinavian occupational exposure limits (Elihn et al. 2008). Exposure to oil mist may cause pulmonary fibrosis (Skyberg et al. 1986, 1992). A fraction of the particles in

✉ Bente Ulvestad
bente.ulvestad@stami.no

1 National Institute of Occupational Health, Oslo, Norway
2 Environmental and Occupational Medicine, Oslo University Hospital, Oslo, Norway
3 Department of Radiology and Nuclear Medicine, Oslo University Hospital, Rikshospitalet, University of Oslo, Oslo, Norway
4 Department of Applied Environmental Science, Stockholm University, Stockholm, Sweden
5 Department of Respiratory Medicine, Oslo University Hospital, Oslo, Norway
6 Institute of Clinical Medicine, University of Oslo, Oslo, Norway

asphalt fumes are ultrafine (UFPs), and asphalt pavers are exposed to relatively high concentrations of these particles throughout their working day (Elihn et al. 2008; Freud et al. 2012). Exposure to total dust and nitrogen dioxide is low to moderate in asphalt paving, compared with Scandinavian occupational exposure limits. In addition, exposure to diesel particulate matter, measured as elemental carbon (EC), is reportedly low, i.e., around 3 μg/m^3 (Elihn et al. 2008).

Studies have reported increased mortality from lung diseases in asphalt workers (Randem et al. 2003; Burstyn et al. 2003; Hansen 1991). In a previous study, we found that a group of asphalt pavers who perform their work behind the paving machine (hereafter called screedmen) declined significantly in both forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV_1) during the asphalt paving season (April to October in Norway) (Ulvestad et al. 2007). The screedmen control asphalt mass discharge through the screed and fix the edges of the asphalt manually. They walk behind the paving machine and are not protected by a cabin, unlike the rest of the team.

Studies on respiratory effects of exposure to oil mist or oil vapor are few. One animal study suggests that high exposure to mineral oil-based oil mist may result in pathological alterations of the alveoli of the lungs, while the bronchi appear to be less vulnerable (Dalbey 2001).

The aims of the present investigation was first to assess whether the reduction in ventilatory lung function over time was greater in asphalt workers than in unexposed subjects, and second to decide whether asphalt pavers may have signs of lung disease detectable by high-resolution computed tomography (HRCT).

Subjects and methods

Study population and design

The study population comprised all male asphalt workers employed in one of Norway's major road maintenance companies. In 2005, all 100 asphalt workers handling hot mix asphalt (16 paver operators, 42 screedmen, 12 roller drivers and 30 plant operators) were examined at the start of the asphalt paving season (April). Prior to the 2005 season, the company had modernized the engines. Both the paving machines and the rollers had been fitted with ventilated cabins, and also the asphalt plants had been fitted with ventilated operators' cabins.

In 2006, it was decided that the occupational health team should carry out annual follow-up of lung function in the asphalt pavers working with hot mix asphalt (16 paver operators, 42 screedmen, 12 roller drivers). Work rotation was introduced in 2006, but 35 (of the 42) screedmen continued with screed work as their main task. Twenty-five plant operators were not included because they were considered non-exposed to hot mix asphalt. Five plant operators had changed work to paving and were included. Thus, a total of 75 asphalt pavers, all men, were included. A reference group of 71 road maintenance workers (hereafter called referents) was also included. The flowchart shown in Fig. 1 gives a summary of the design and the number of participants at baseline and the different follow-ups. All participants were tested at least twice. Ten of the exposed workers were tested twice, 21 three times, 28 four times, 12 five times, and 4 six times. Of the referents, 55 were tested twice, 15 thrice, and one four times.

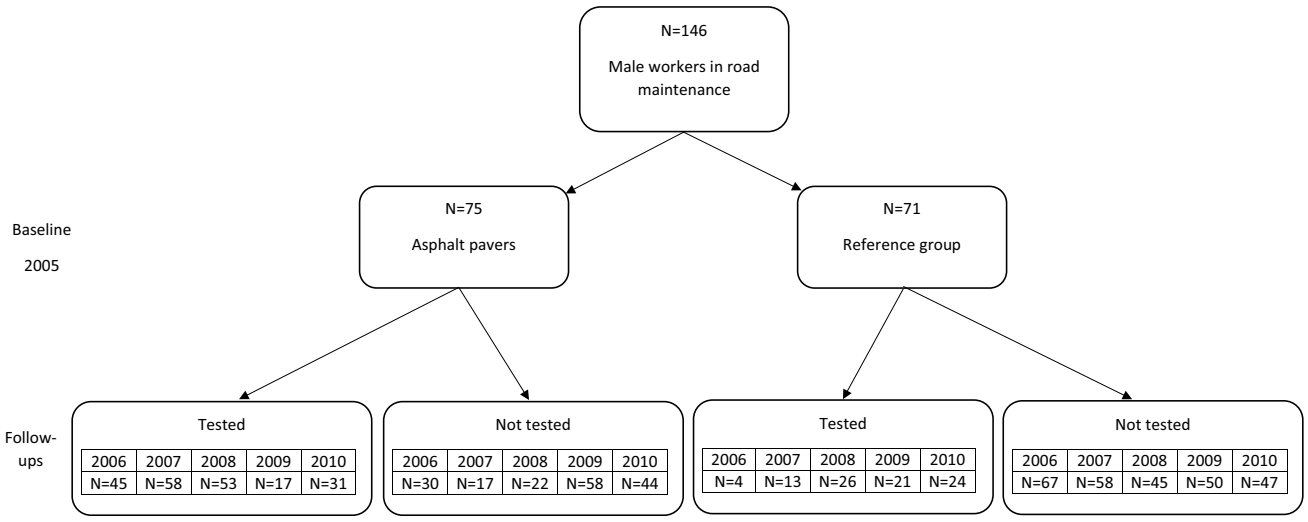

Fig. 1 Flowchart of the follow-up study

Also all referents were men, and they were recruited from three different road maintenance departments of the same company. The road maintenance workers perform different tasks such as assembling road signs, repairing damaged guardrails, cutting vegetation along roads, plowing snow, and cleaning and inspecting roads. Much of the time during a typical working day is spent driving a car or a truck.

The plan was that the study subjects should perform spirometry annually (before the start of the asphalt season) until retirement or transfer to other work. However, in 2011, the management of the company decided to close down the asphalt division, and the remaining crews and equipment were transferred to other contractors. Measurements of lung function had then been taken in April 2005, April 2006, April 2007, April 2008, April 2009 and April 2010. Due to study constraints, not every worker was tested every year, but most of the pavers ($n = 44$) had four or more measurement points and none had fewer than two. In 2011, before the transfer of the workers to other contractors, all the asphalt pavers ($n = 75$) were invited to have HRCT scans of the lungs as a part of closing the follow-up study. The workers were contacted by telephone and then received a formal written invitation. All ($n = 75$) accepted to participate in the HRCT study, and the scans were done after the transfer to other contractors. Controls were not invited in order to avoid unnecessary radiation exposure. Written consent was obtained from all participants.

The study was approved by the Regional Ethical Committee for Medical Research, Norway (REK 2011/170a).

Questionnaire

Information on age, allergy and smoking habits was collected in 2005. Annual follow-up questionnaires included information on development of respiratory symptoms, or whether the subjects had been diagnosed by a physician as having asthma or chronic obstructive pulmonary disease (COPD). Information on current and former smoking was also collected.

Spirometry

Spirometry was performed according to the American Thoracic Society (ATS)/European Respiratory Society (ERS) guidelines (Miller et al. 2005). The following variables were recorded: forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV_1), and the value of FEV_1/FVC was calculated. The lung function variables were expressed in absolute values and as the percentage of predicted values (Quanjer et al. 1993). The tests were carried out in connection with start-up meetings held at different hotels before the start of the asphalt paving season each year. The same well-trained technician performed the spirometric measurements, using the same equipment (Spirare SPS330 Sensor, Diagnostica AS).

Height and weight of the participants were recorded, and body mass index (BMI) was calculated.

Acquisition and review of HRCT images

CT scanners at thirteen institutions in different parts of the country were used in this study. Similar scanning protocol was used in all institutions.

Low-dose thin-section CT images were obtained in the supine position during breath-holding and deep inspiration. Supplementary expiratory scans were obtained in each subject except thirteen. In two subjects, supplementary images were obtained in the prone position. The images were obtained at 1–1.25 mm section thickness and inspiratory scans at 10-mm intervals and expiratory scans at 20-mm intervals. Supplementary low-dose volume scan was obtained in twelve subjects. Images were reconstructed with a high-spatial-frequency (bone) algorithm. Tube current settings were adjusted to each patient's weight, but with low-dose references.

Electronic files with the HRCT images were transferred to our institution and reviewed on a PACS (picture archiving and communication system) screen in consensus and in random order by two chest radiologists (TMA and GKM, with 20 and 14 years of experience, respectively). The observers were blinded to lung function tests. The observers evaluated the presence and extent of airways disease and interstitial lung disease. These findings included ground-glass opacity, airspace consolidation, reticular pattern, bronchiectasis, nodules and air trapping. The severity of the HRCT abnormalities was evaluated by criteria previously reported (Aaløkken et al. 2012). Sub-segmental air trapping comprising <5 % of the lung parenchyma was considered normal (Tanaka et al. 2003).

Exposure measurements

The air sampling methods and results of exposure assessments have been described elsewhere (Elihn et al. 2008). Exposure to total dust, oil mist, oil vapor, polycyclic aromatic hydrocarbons and NO_2 was determined by personal sampling. We measured exposure to ultrafine particles by means of particle counters and scanning mobility particle sizer mounted on a van following the paving machine. The fractions of organic and elemental carbon were determined.

A sample of workers, representing different work tasks, was asked to participate in the exposure assessment. Participation was voluntary, but all the selected workers agreed to participate. Description of asphalt work has been reported elsewhere (Elihn et al. 2008; Freud et al. 2012).

Statistical analysis

Linear mixed models were used to analyze changes in lung function (FEV_1, FVC and FEV_1/FVC) (Fitzmaurice et al. 2011). Prior to analysis, FEV_1 and FVC were normalized by dividing its values by standing height squared (h^2). The analyses were adjusted for baseline values of age, BMI and packyears of smoking in smokers and ex-smokers. Packyears were calculated by multiplying the duration of smoking (in years) by the average number of cigarettes smoked daily divided by 20. The packyears variable was allowed to interact with baseline age and with years since baseline, in order to take into account that smokers may have a more pronounced decline in lung function. In a first analysis, we compared the decline in lung function between workers and references. This was done by including an interaction term between group and years since baseline. In a second analysis, we divided the workers in three groups, references, non-screed and screedmen, and did pairwise group comparisons of the decline in lung function.

To account for the dependency due to repeated observations, a subject-specific random intercept was included in the model. In mathematical terms, the first model may be formulated as follows.

$$\begin{aligned} Y_{\mathrm{it}} = {} & \mu + \beta^{\mathrm{G}}\mathrm{group}_i + \beta^{\mathrm{T}}T_{\mathrm{it}} + \beta^{\mathrm{G\times T}}\mathrm{group}_i \times T_{\mathrm{it}} \\ & + \alpha^{\mathrm{P}}\mathrm{packyears}_i + \beta^{\mathrm{B}}\mathrm{bmi}_i + \beta^{\mathrm{A}}\mathrm{age}_{\mathrm{it}} \\ & + \beta^{\mathrm{A\times P}}\mathrm{age}_{\mathrm{it}} \times \mathrm{packyears}_i + \beta T^{\mathrm{T\times P}}T_{\mathrm{it}} \\ & \times \mathrm{packyears}_i + u_i + \varepsilon_{\mathrm{it}} \end{aligned}$$

where Y_{it} is the observed lung function (FEV_1/h^2, FVC/h^2 or FEV_1/FVC) for worker i at years since baseline T_{t} (0, 0.5, 1, 2, 3, 4, 5). Furthermore, μ, β^{G}, β^{T}, $\beta^{\mathrm{G\times T}}$, β^{P}, β^{B}, β^{A}, $\beta^{\mathrm{A\times P}}$ and $\beta^{\mathrm{T\times P}}$ are fixed effects, where μ is the intercept value, β^{G} the effect of group (exposed vs referents), β^{P} the effect of packyears, β^{B} the effect of BMI, while β^{A} gives the effect of baseline age and β^{T} the effect of year on lung function. The effects of the interaction terms are given by $\beta^{\mathrm{G\times T}}$ (group × years since baseline), $\beta^{\mathrm{A\times P}}$ (age × packyears) and $\beta^{\mathrm{T\times P}}$ (years since baseline × packyears). The random intercepts u_{i} and residuals $\varepsilon_{\mathrm{it}}$ are normally distributed random variables

$$u_i \overset{iid}{} N\left(0, \sigma_u^2\right),\ \varepsilon_{\mathrm{it}} \overset{iid}{} N\left(0, \sigma^2\right).$$

To assess the decline in lung function in absolute values, for a worker of average height (1.80 m), the estimates and confidence intervals for the decline in FEV_1/h^2 and FVC/h^2 were multiplied by 1.80 m squared. *T* tests were used to compare baseline values for referents and asphalt pavers. Linear mixed models were analyzed with the nlme package in *R*, and all other statistical analyses were carried out with SPSS 21.0 (SPSS Inc, Chicago, IL, USA).

Results

Subjects

Table 1 describes the general characteristics of the study population at baseline. The asphalt pavers were somewhat older than the control group (mean (SD) age 45.6 (8.5) versus 42.5 (8.5) years, respectively). FEV_1 % of predicted and FEV_1/FVC were significantly lower in asphalt pavers than in the reference group. At baseline in 2005, there were 25 % smokers among both references and pavers.

In 2011, only 19 % of the pavers remained smokers, while the fraction of smokers was unchanged (25 %) in the references. Mean numbers of asphalt seasons was 17.4 (SD 8.8).

There were no differences between the screedmen and the rest of the asphalt paving team with respect to number of asphalt seasons, age, BMI or smoking habits.

Exposure

The exposure measurements were taken during two asphalt seasons (2005 and 2006). A total of 51 asphalt workers carried personal samplers in the exposure study, and many workers were monitored on more than one occasion. Altogether 65 measurements of total dust, 58 of total PAHs and 22 of oil mist and oil vapor were taken. Although the geometric mean concentration of oil mist was less than 0.3 mg/m^3, the exposure to oil mist was high on some occasions; for example, on one occasion a level of 1.7 mg/m^3 was measured (the Norwegian occupational exposure limit

Table 1 Characteristics at baseline for 146 workers included in the follow-up study

Variable	Referents ($N = 71$)		Asphalt workers ($N = 75$)	
	Mean	SD	Mean	SD
Age (years)	42.5	8.5	45.6*	8.5
Height (cm)	180.2	5.7	179.3	6.1
BMI (kg/m^2)	27.9	9.6	27.3	4.1
Smoking				
Current smoker (%)	25		25	
Ever smoker (%)	55		57	
Lung function				
FEV_1 (L)	4.0	0.5	3.7*	0.6
FEV_1 (% of predicted)	99.9	10.5	95.8*	10.5
FVC (L)	4.9	0.7	4.8	0.7
FVC (% of predicted)	100.7	10.8	99.1	10.9
$FEV_1/FVC \times 100$	81.2	4.9	78.6*	6.1

The means are unadjusted. * Independent sample *t* test, $p < 0.05$

FVC forced vital capacity, *FEV_1* forced expiratory volume in one second

(NOEL) of oil mist is 1.0 mg/m^3). This peak air concentration was measured by personal sampling and a screedman carried the sampler.

The asphalt paving workers were exposed to ultrafine particles with medium concentration of about 3.4 × 10^4/cm^3. Ultrafine particles at the paving sites mainly originate from asphalt fumes during asphalt paving activities and partly from traffic exhaust. Diesel particulate matter was measured as elemental carbon, which was low, around 3 µg/m^3. Nitrogen dioxide (NO_2) and total dust did not exceed occupational exposure limits. The pavers may, however, have been exposed to higher levels of NO_2 when paving asphalt in tunnels (max level measured was 3.4 ppm (NOEL of NO_2 = 0.6 ppm).

Lung function

Table 2 shows the annual individual changes in lung function for references and pavers during the 5-year follow-up. In Table 3, the differences in annual change between references and pavers are described. Adjusted for baseline age, BMI and packyears of smoking, the pavers lost significantly more lung volumes (FVC/h^2 and FEV_1/h^2) than the references. In Table 4, the asphalt pavers have been split into two groups: screed and non-screed pavers. The screedmen had the largest decline in lung function (FVC/h^2 and FEV_1/h^2) followed by the other pavers, while the references had a smaller decline. Table 5 describes the differences in annual individual changes between references, screedmen and non-screed pavers. The screedmen declined more in FVC/h^2

Table 2 Annual individual change in lung function for referents and asphalt pavers during the 5-year follow-up

Outcome	Group	Estimate	*P*	95 % CI
FEV_1/h^2	Referents	−0.0024	0.35	−0.0075 to 0.0026
FEV_1/h^2	Paver	−0.011	<0.0001	−0.016 to −0.006
FVC/h^2	Referents	−0.0074	0.054	−0.015 to 0.0001
FVC/h^2	Paver	−0.018	<0.0001	−0.025 to −0.011
FEV_1/FVC	Referents	0.11	0.39	−0.14 to 0.37
FEV_1/FVC	Paver	0.28	0.032	0.026–0.53

Adjustment for baseline age, BMI and packyears in linear mixed models

FVC forced vital capacity, *FEV_1* forced expiratory volume in one second, *CI* confidence interval

Table 3 Difference in annual individual change between asphalt pavers and referents

Outcome	Group	Change	*P*	95 % CI
FEV_1/h^2	Pavers versus referents	−0.0085	0.0077	−0.015 to −0.0023
FVC/h^2	Pavers versus referents	−0.011	0.025	−0.02 to −0.0014
FEV_1/FVC	Pavers versus referents	0.16	0.31	−0.15 to 0.48

Adjustment for baseline age, BMI and packyears in linear mixed models

FVC forced vital capacity, *FEV_1* forced expiratory volume in one second, *CI* confidence interval

Table 4 Annual individual change in lung function for the reference group and two groups of asphalt pavers (non-screed and screed) during the 5-year follow-up

Outcome	Group	Change	*P*	95 % CI
FEV_1/h^2	Referents	−0.0024	0.36	−0.0075 to 0.0027
FEV_1/h^2	Non-screed	−0.0095	0.004	−0.016 to −0.0031
FEV_1/h^2	Screed	−0.012	0.0003	−0.019 to −0.0059
FVC/h^2	Referents	−0.0072	0.059	−0.015 to 0.0002
FVC/h^2	Non-screed	−0.011	0.022	−0.021 to −0.0017
FVC/h^2	Screed	−0.025	<0.0001	−0.035 to −0.015
FEV_1/FVC	Referents	0.11	0.41	−0.15 to 0.36
FEV_1/FVC	Non-screed	0.014	0.93	−0.31 to 0.34
FEV_1/FVC	Screed	0.55	0.0012	0.22 to 0.88

Adjustment for baseline age, BMI and packyears in linear mixed models

FVC forced vital capacity, *FEV_1* forced expiratory volume in one second, *CI* confidence interval

Table 5 Difference in annual individual change between exposed groups and reference group

Outcome	Group	Change	*P*	95 % CI
FEV_1/h^2	Non-screed versus referents	−0.0071	0.06	−0.015 to 0.0002
FEV_1/h^2	Screed versus referents	−0.01	0.011	−0.018 to −0.0025
FEV_1/h^2	Screed versus non-screed	−0.0029	0.51	−0.011 to 0.0055
FVC/h^2	Non-screed versus referents	−0.0039	0.48	−0.015 to 0.0068
FVC/h^2	Screed versus referents	−0.018	0.002	−0.029 to −0.0067
FVC/h^2	Screed versus non-screed	−0.014	0.029	−0.026 to −0.0016
FEV_1/FVC	Non-screed versus referents	−0.092	0.63	−0.46 to 0.27
FEV_1/FVC	Screed versus referents	0.44	0.023	0.067–0.82
FEV_1/FVC	Screed versus non-screed	0.54	0.013	0.12–0.95

Adjustment for baseline age, BMI and packyears in linear mixed models

FVC forced vital capacity, *FEV_1* forced expiratory volume in one second, *CI* confidence interval

than the non-screed pavers. Adjusted for baseline values for age, BMI and packyears of smoking, the annual decline in asphalt pavers of average height of 180 cm was calculated to 58 ml (95 % CI [−82, −34]) in FVC and 35 ml (95 % CI [−52, −19]) in FEV_1, while the referents of average height of 180 cm had an average decline of 24 ml (95 % CI [−48, 0]) in FVC and 8 ml (95 % CI [−24, +8]) in FEV_1.

HRCT findings

The HRCT findings are shown in Table 6. Fine intralobular fibrosis without evident cysts was identified in three subjects (4 %), all of them with <5 % overall extent of fibrosis.

All three had earlier exposure to quartz in earlier dusty occupations apart from asphalt work. Coarse fibrosis with cystic reticular pattern and honeycombing was not observed in any subject. Moderate centrilobular emphysema was detected in three subjects (4 %), two of them had physician-diagnosed COPD, and all were former smokers. Airways disease (air trapping, bronchiectasis or centrilobular micronodules) was detected in 8 subjects (11 %). Other benign findings (perifissural nodules, hamartomas, small atelectasis, lung cysts) considered to be incidental and not related to occupational exposure were detected in 14 subjects (19 %). No differences in HRCT findings between the screed workers and the non-screed workers were observed.

Discussion

In this study, we used data from medical examinations in one of Norway's major construction companies in order to study longitudinal changes in lung function in asphalt pavers handling hot mixture asphalt. In addition, all the asphalt pavers had HRCT scans of the lungs at the end of the 5-year follow-up period.

We found that the asphalt pavers had an accelerated decline in both FVC and FEV_1 during the 5-year follow-up compared to a reference group of road maintenance workers. The annual average total declines in FVC and FEV_1 for the pavers were 58 and 35 ml, respectively. The annual average decline in FEV_1 determined for an unexposed non-smoker has been estimated to 27 ml (Burrows et al. 1986). Excess declines found in heavily exposed subjects working in tunneling, mining and cement production have been reported to be 7–38 ml (Ulvestad et al. 2001; Cowie and Mabena 1991; Marine et al. 1988; Nordby et al. 2011). Among the asphalt pavers, the screedmen had larger annual declines in FVC and FEV_1 than the other pavers (roller drivers and paver operators) did. The screedmen are the most heavily exposed workers among the asphalt pavers.

The reference group of road maintenance workers was exposed to road dust, but not to asphalt fumes.

Asphalt pavers are exposed to asphalt fumes including organic substances with particle sizes in the respirable fraction. High exposure levels to organic carbon (OC) and a low EC/total carbon ratio have been demonstrated in asphalt paving (Elihn et al. 2008). Exposure to OC may be an expression of oil mist exposure. Asphalt pavers' exposure to oil mist, especially among screedmen, has been shown to exceed Scandinavian occupational exposure limits on some occasions (Elihn et al. 2008).

Few published studies deal with exposure to oil mist and lung function.

In a study of machine workers exposed to an average of 0.33 mg/m^3 of oil mist (total aerosol) in car production, no restrictive or obstructive lung function impairments were found, but the exposed subjects reported more lung symptoms than the references (Sprince et al. 1997). In another study from the car industry, self-reported respiratory symptoms increased, related to the use of mineral oil-based products (Greaves et al. 1997). Eisen et al. (2001) described lung function measurements from that study. They found no effects on lung function by ongoing exposure, but an association between past exposures to mineral oil-based oil mist and reduced FVC (Eisen et al. 2001). Cable workers, exposed to oil mist with a time-weighted

Table 6 HRCT findings in 75 asphalt pavers

Normal HRCT	44 (59 %)
Lung fibrosis: fine intralobular without evident cysts	3 (4 %)
Lung fibrosis: microcystic reticular pattern	0
Lung fibrosis: macrocystic reticular pattern with honeycombing	0
Emphysema	3 (4 %)
Airways disease (air trapping[a], bronchiectasis, centrilobular micronodules)	8 (11 %)
Coronary calcification	4 (5 %)
Other incidental benign findings (perifissural nodules, hamartomas, small atelectasis, lung cysts)	14 (19 %)

Some of the subjects had more than one finding

[a] Air trapping was evaluated in 62 subjects

average of 0.15–0.30 mg/m^3, but with occasionally much higher peak exposures, have been shown to have a high prevalence of lung fibrosis (Skyberg et al. 1992). Thus, based on the studies by Skyberg et al. (1986) and Eisen et al. (2001), it would be expected that exposure to oil mists may cause a restrictive pattern in lung function. In an animal study, exposure to oil mists caused toxic effects in the lungs of rats after 2 weeks of inhalation in exposure chambers (Skyberg et al. 1990). Toxic effects included lower whole-body weights compared to those of controls, and high-level exposure was lethal to three out of four animals, due to pulmonary edema.

Asphalt pavers are exposed to a complex mixture of airborne contaminants, including particles generated from the hot asphalt mix, and emissions from diesel engines. These contaminants form in high concentrations and have a size distribution that allows penetration into the alveolar region of the lung (Elihn et al. 2008). A large fraction of the inhaled UFPs deposits in the alveoli, and even more during hard exercise. UFPs clear less efficiently from the alveoli than larger particles (Oberdörster et al. 2005). This is one of the reasons why UFPs are considered more harmful than fine particles, but high concentrations of adsorbed volatile pollutants on large total particle surface area may also contribute to potential negative health effects. Our instrument measuring UFPs and fine particles could not differentiate between types of particles, i.e., no chemical information was gained. Furthermore, UFPs are considered more reactive than larger particles of the same material. This size dependence has been shown in many in vitro experiments (Midander et al. 2009; Soenen et al. 2013). Several studies suggest harmful effects of UFP on the cardiovascular and respiratory systems (Delfino et al. 2005; Shannahan et al. 2012; Weichenthal. 2012).

In a former study of asphalt workers (Ulvestad et al. 2007), only the screedmen declined significantly in FVC and FEV_1 during the asphalt paving season. After the first study, work rotation was introduced among the asphalt pavers (paver operators, screedmen, roller drivers). A rotation system reduces exposure for the entire group of asphalt pavers since both the paving machines and the rollers are fitted with ventilated cabins. Although work rotation had been introduced, 35 workers reported work behind the screed as their main job task. The effect of working as a screedman found in the present study may indicate that exposure to oil mist and UFPs occurs predominantly in this group of asphalt pavers. In the present study, also the non-screed pavers showed statistically significant loss in both FVC and FEV_1, compared to the controls. The difference in annual individual change between screedmen and non-screedmen was significant for FVC only.

One strength of the present study is the longitudinal design, in which the subjects serve as their own controls at follow-up. Another strength is a reference group of road maintenance workers comparable to the index group of asphalt pavers with respect to socioeconomic status, working schedule and smoking habits.

More participants would have been desirable, but asphalt workers are, at any time, dispersed throughout the country and are not easily accessible for lung function testing. All health examinations were performed at the same time of the year, which reduces the effect of seasonal variation known to affect lung function (Senthilselvan et al. 2000). The spirometric measurements were taken by the same well-trained technician, using the same equipment, thereby reducing the within-worker variability of multiple measurements. The follow-up time of 5 years is acceptable (Bakke et al. 2011). A potential limitation of the present study is a possible "healthy worker effect." It is probable that workers susceptible to adverse effects from exposure to asphalt fumes leave the industry, leaving only more robust subjects to follow up. If so, the lung function decline found in the present study is underestimated. The statistical analyses performed, including mixed models, should be adequate for handling confounders like smoking, aging and BMI.

CT scanners at 13 institutions were used in this study; however, the same scanning protocol was used. All electronic files were transferred to our tertiary university hospital and reviewed in random order by two highly experienced chest radiologists who were blinded to the results of lung function testing.

Despite the fact that HRCT scanning is a sensitive method for diagnosing possible work-related conditions, like pneumoconiosis, pleural plaques and emphysema, the majority of the pavers with long-term exposure in asphalt paving had normal findings.

A reticular pattern with sub-pleural distribution was detected in three workers. This feature is strongly associated with interstitial fibrosis and implies distortion of the lung architecture. Two of these workers had worked in an asphalt plant that shared site with a stone crushing plant, for 20 and 25 years, respectively, and the third worker had worked with rock drilling for 3 years and with paving for eight seasons. Consequently, a causative relationship between the lung fibrosis and α-quartz exposure cannot be excluded even if nodules are the most common radiographic finding of silicosis (Sirajuddin and Kanne 2009).

Emphysema and the other reported HRCT findings in our study are not specific for work-related conditions.

The possible health hazards of exposure to asphalt fumes are still open for discussion. In a cross-shift study of 320 mastic asphalt workers and a reference group of 118 construction workers, slight cross-shift declines in FVC and FEV_1 were found in the bitumen-exposed workers, but not in the references (Raulf-Heimsoth et al. 2011a). In induced sputum examinations, levels of the biomarkers

interleukin-8, total protein and matrix metalloproteinase-9 have been found to be significantly higher in bitumen-exposed workers than in the referent workers, indicating potentially (sub-) chronic inflammatory effects in the lower airways of bitumen-exposed workers (Raulf-Heimsoth et al. 2011a). Exposure in flooring or paving with mastic asphalt is known to be substantial (Burstyn et al. 2003; Raulf-Heimsoth et al. 2011b) compared with exposure in road paving.

We conclude from the present study that our findings may indicate an excess annual decline in FVC and FEV_1 related to exposure to asphalt fumes. The screedmen, who carry out their work behind and close to the paving machine, had the largest decline in lung function. The finding of adverse pulmonary effects in asphalt pavers calls for better technological solutions to prevent exposure.

Acknowledgments We thank the workers participating in the study. The project received financial support from the Norwegian Heart and Lung Association.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

References

Aaløkken TM, Naalsund A, Mynarek G, Berstad A, Solberg S, Strøm EH, Scott H, Kolbenstvedt A, Søyseth V (2012) Diagnostic accuracy of computed tomography and histopathology in the diagnosis of usual interstitial pneumonia. Acta Radiol 53:296–302

Bakke PS, Rönmark E, Eagan T, Pistelli F, Annesi-Maesano I, Maly M, Meren M, Vermeire Dagger P, Vestbo J, Viegi G, Zielinski J, Lundbäck B, European Respiratory Society Task Force (2011) Recommendations for epidemiological studies on COPD. Eur Respir J 38:1261–1267

Burrows B, Lebowitz MD, Camili AE, Knudson RJ (1986) Longitudinal changes in forced expiratory volume in one second in adults: methodologic considerations and findings in healthy nonsmokers. Am Rev Respir Dis 133:974–980

Burstyn I, Boffetta P, Heederik D, Partanen T, Kromhout H, Svane O, Langård S, Frentzel-Beyme R, Kauppinen T, Stücker I, Shaham J, Ahrens W, Cenée S, Ferro G, Heikkilä P, Hooiveld M, Johansen C, Randem BG, Schill W (2003) Mortality from obstructive lung diseases and exposure to polycyclic aromatic hydrocarbons among asphalt workers. Am J Epidemiol 158:468–478

Cowie RL, Mabena SK (1991) Silicosis, chronic airflow limitation, and chronic bronchitis in South African gold miners. Am Rev Respir Dis 143:80–84

Dalbey WE (2001) Subchronic inhalation exposures to aerosols of three petroleum lubricants. AIHAJ 62:49–56

Delfino RJ, Sioutas C, Malik S (2005) Potential role of ultrafine particles in associations between airborne particle mass and cardiovascular health. Environ Health Perspect 113(8):934–946

Eisen EA, Smith TJ, Kriebel D, Woskie SR, Myers DJ, Kennedy SM, Shalat S, Monson RR (2001) Respiratory health of automobile workers and exposures to metal-working fluid aerosols: lung spirometry. Am J Ind Med 39:443–453

Elihn K, Ulvestad B, Hetland S, Wallen A, Randem BG (2008) Exposure to ultrafine particles in asphalt work. J Occup Environ Hyg 5:771–779

Fitzmaurice GM, Laird NM, Ware JH (2011) Applied longitudinal analysis, 2nd edn. Wiley, Hoboken

Freud A, Zuckerman N, Baum L, Milek D (2012) Submicron particle monitoring of paving and related road construction operators. J Occup Environ Hyg 9:298–307

Greaves IA, Eisen EA, Smith TJ, Pothier LJ, Kriebel D, Woskie SR, Kennedy SM, Shalat S, Monson RR (1997) Respiratory health of automobile workers exposed to metal-working fluid aerosols: respiratory symptoms. Am J Ind Med 32:450–459

Hansen ES (1991) Mortality of mastic asphalt workers. Scand J Work Environ Health 17(1):20–22

Marine WM, Gurr D, Jacobsen M (1988) Clinically important respiratory effects of dust exposure and smoking in British coal miners. Am Rev Respir Dis 137:106–112

Midander K, Cronholm P, Karlsson H, Elihn K, Möller L, Leygraf C, Odnevall Wallinder I (2009) Surface characteristics, copper release, and toxicity of nano- and micrometer-sized copper and copper (II) oxide particles: a cross-disciplinary study. Small 5(3):389–399

Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, Crapo R, Enright P, van der Grinten CP, Gustafsson P, Jensen R, Johnson DC, MacIntyre N, McKay R, Navajas D, Pedersen OF, Pellegrino R, Viegi G, Wanger J (2005) Standardisation of spirometry. Eur Respir J 26:319–338

Nordby KC, Fell AK, Notø H, Eduard W, Skogstad M, Thomassen Y, Bergamaschi A, Kongerud J, Kjuus H (2011) Exposure to thoracic dust, airway symptoms and lung function in cement production workers. Eur Respir J 38:1278–1286

Oberdörster G, Oberdörster E, Oberdörster J (2005) Nanotoxicology: an emerging discipline evolving from studies of ultrafine particles. Environ Health Perspect 113:823–839

Quanjer PH, Tammeling GJ, Cotes JE, Fabbri LM, Matthys H, Pedersen OF, Peslin R, Roca J, Sterk PJ, Ulmer WT, Yernault JC (1993) Lung volumes and forced ventilatory flows. Report working party standardization of lung function tests, European community for steel and coal. Official statement of the European respiratory society. Eur Respir J 6:S5–S40

Randem BG, Langård S, Kongerud J, Dale J, Burstyn I, Martinsen JI, Aa Andersen (2003) Mortality from non-malignant diseases among male Norwegian asphalt workers. Am J Ind Med 43:96–103

Raulf-Heimsoth M, Marczynski B, Spickenheuer A, Pesch B, Welge P, Rühl R, Bramer R, Kendzia B, Heinze E, Angerer J, Brüning T (2011a) Bitumen workers handling mastic versus rolled asphalt in a tunnel: assessment of exposure and biomarkers of irritation and genotoxicity. Arch Toxicol 85:81–87

Raulf-Heimsoth M, Pesch B, Kendzia B, Spickenheuer A, Bramer R, Marczynski B, Merget R, Brüning T (2011b) Irritative effects of vapours and aerosols of bitumen on the airways assessed by non-invasive methods. Arch Toxicol 85:41–52

Senthilselvan A, Dosman JA, Semchuk KM, McDuffie HH, Cessna AJ, Irvine DG, Crossley MF, Rosenberg A (2000) Seasonal changes in lung function in a farming population. Can Respir J 7:320–325

Shannahan JH, Urmila PK, Brown JM (2012) Manufactured and airborne nanoparticle cardiopulmonary interactions: a review of mechanisms and the possible contribution of mast cells. Inhal Toxicol 24(5):320–339

Sirajuddin A, Kanne JP (2009) Occupational lung disease. J Thorac Imaging 24(4):310–320

Skyberg K, Rønneberg A, Kamøy JI, Dale K, Borgersen A (1986) Pulmonary fibrosis in cable plant workers exposed to mist and vapour of petroleum distillates. Environ Res 40:261–273

Skyberg K, Skaug V, Gylseth B, Pedersen JR, Iversen OH (1990) Subacute inhalation toxicity of mineral oils, C 15–C 20

alkylbenzenes, and polybutene in male rats. Environ Res 53:48–61

Skyberg K, Rønneberg A, Christensen CC, Næss-Andersen CF, Borgersen A, Refsum HE (1992) Lung function and radiographic signs of pulmonary fibrosis in oil exposed workers in a cable manufacturing company: a follow up study. Br J Ind Med 49:309–315

Soenen SJ, Manshian B, Doak SH, Smedt SC, Braeckmans K (2013) Fluorescent non-porous silica nanoparticles for long-term cell monitoring: cytotoxicity and particle functionality. Acta Biomater 9:9183–9193

Sprince NL, Thorne PS, Popendorf W, Zwerling C, Miller ER, DeKoster JA (1997) Respiratory symptoms and lung function abnormalities among machine operators in automobile production. Am J Ind Med 31:403–413

Tanaka N, Matsumoto T, Miura G, Emoto T, Matsunaga N, Ueda K, Lynch DA (2003) Air trapping at CT: high prevalence in asymptomatic subjects with normal pulmonary function. Radiology 227:776–785

Ulvestad B, Bakke B, Eduard W, Kongerud J, Lund MB (2001) Cumulative exposure to dust causes accelerated decline in lung function in tunnel workers. Occup Environ Med 58:663–669

Ulvestad B, Randem BG, Hetland S, Sigurdardottir G, Johannessen E, Lyberg T (2007) Exposure, lung function decline and systemic inflammatory response in asphalt workers. Scand J Work Environ Health 33:114–121

Weichenthal S (2012) Selected physiological effects of ultrafine particles in acute cardiovascular morbidity. Environ Res 115:26–36